NTP TECHNICAL REPORT

ON THE

TOXICOLOGY AND CARCINOGENESIS STUDIES IN B6C3F1/N MICE EXPOSED TO WHOLE-BODY RADIO FREQUENCY RADIATION AT A FREQUENCY (1,900 MHz) AND MODULATIONS (GSM AND CDMA) USED BY CELL PHONES

Scheduled Peer Review Date: March 26-28, 2018

NTP TR 596

National Toxicology Program

National Institutes of Health
Public Health Service
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

FOREWORD

The National Toxicology Program (NTP) is an interagency program within the Public Health Service (PHS) of the Department of Health and Human Services (HHS) and is headquartered at the National Institute of Environmental Health Sciences of the National Institutes of Health (NIEHS/NIH). Three agencies contribute resources to the program: NIEHS/NIH, the National Institute for Occupational Safety and Health of the Centers for Disease Control and Prevention (NIOSH/CDC), and the National Center for Toxicological Research of the Food and Drug Administration (NCTR/FDA). Established in 1978, the NTP is charged with coordinating toxicological testing activities, strengthening the science base in toxicology, developing and validating improved testing methods, and providing information about potentially toxic substances to health regulatory and research agencies, scientific and medical communities, and the public.

The Technical Report series began in 1976 with carcinogenesis studies conducted by the National Cancer Institute. In 1981, this bioassay program was transferred to the NTP. The studies described in the Technical Report series are designed and conducted to characterize and evaluate the toxicologic potential, including carcinogenic activity, of selected substances in laboratory animals (usually two species, rats and mice). Substances selected for NTP toxicity and carcinogenicity studies are chosen primarily on the basis of human exposure, level of production, and chemical structure. The interpretive conclusions presented in NTP Technical Reports are based only on the results of these NTP studies. Extrapolation of these results to other species, including characterization of hazards and risks to humans, requires analyses beyond the intent of these reports. Selection *per se* is not an indicator of a substance's carcinogenic potential.

The NTP conducts its studies in compliance with its laboratory health and safety guidelines and FDA Good Laboratory Practice Regulations and must meet or exceed all applicable federal, state, and local health and safety regulations. Animal care and use are in accordance with the Public Health Service Policy on Humane Care and Use of Animals. Studies are subjected to retrospective quality assurance audits before being presented for public review.

NTP Technical Reports are indexed in the NIH/NLM PubMed database and are available free of charge electronically on the NTP website (http://ntp.niehs.nih.gov). Additional information regarding this study may be requested through Central Data Management (CDM) at cdm@niehs.nih.gov. Toxicity data are available through NTP's Chemical Effects in Biological Systems (CEBS) database: https://www.niehs.nih.gov/research/resources/databases/cebs/index.cfm.

NTP TECHNICAL REPORT

ON THE

TOXICOLOGY AND CARCINOGENESIS STUDIES IN B6C3F1/N MICE EXPOSED TO WHOLE-BODY RADIO FREQUENCY RADIATION AT A FREQUENCY (1,900 MHz) AND MODULATIONS (GSM AND CDMA) USED BY CELL PHONES

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CONTENTS

ABSTRACT

GSM- AND CDMA-MODULATED CELL PHONE RADIO FREQUENCY RADIATION

Synonyms: Cell phone radio frequency radiation; mobile phone radio frequency radiation

The predominant source of human exposure to radio frequency radiation (RFR) occurs through usage of cellular phone handsets. The Food and Drug Administration nominated cell phone RFR emission for toxicology and carcinogenicity testing in 1999. At that time, animal experiments were deemed crucial because meaningful human exposure data from epidemiological studies were not available. Male and female B6C3F1/N mice were exposed to time-averaged whole-body specific absorption rates of 0 (sham control), 5, 10, or 15 W/kg Global System for Mobile Communications (GSM)- or Code Division Multiple Access (CDMA)-modulated cell phone RFR at 1,900 MHz for 28 days or 0, 2.5, 5, or 10 W/kg GSM- or CDMA-modulated cell phone RFR for up to 2 years. Genetic toxicology studies were conducted in mouse peripheral blood erythrocytes and leukocytes, brain cells, and liver cells.

GSM

28-DAY STUDY

Groups of 10 male and 10 female core study mice and groups of 20 male and 20 female special study mice were housed in specially designed reverberation chambers and received whole-body exposures to GSM-modulated cell phone RFR at power levels of 0 (sham control), 5, 10, or 15 W/kg, for up to 18 hours and 20 minutes per day, 5 or 7 (last week of study) days per week for at least 28 days with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. The sham control animals were housed in reverberation chambers identical to those

used for the exposed groups, but were not exposed to cell phone RFR; a shared group of unexposed mice of each sex served as sham controls for both cell phone RFR modulations. All mice survived to the end of the study. Mean body weights of exposed groups of males and females were similar to controls. There were no exposure-related clinical signs, differences in organ weights, or histopathologic findings. Differences in body temperatures between the exposed groups and the control group were not considered to be related to cell phone RFR exposure.

2-YEAR STUDY

Groups of 105 male and 105 female mice were housed in reverberation chambers and received whole-body exposures to GSM-modulated cell phone RFR at power levels of 0 (sham control), 2.5, 5, or 10 W/kg, 9 hours and 10 minutes per day, 7 days per week for 106 (males) or 108 (females) weeks with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; shared groups of unexposed mice of each sex served as sham controls for both cell phone RFR modulations. Fifteen mice per group were randomly selected from the core group after 10 weeks of study; ten of those 15 mice per group were used for interim evaluation at 14 weeks, and five mice per group were used for genetic toxicity testing at 14 weeks. The remaining 90 animals per group were exposed up to 2 years.

At the 14-week interim evaluation in the 2-year study, mean body weights of exposed groups of males and females were similar to those of the sham controls. There were no changes to the hematology variables attributable to GSM cell phone RFR exposure. Differences in organ weights were not associated with histopathologic findings and were not considered related to exposure. In males, there were no exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility. In females, there were no exposure related effects on estrous cycle length, number of cycling females, or relative amount of time spent in the estrous stages. The only histopathologic finding at the 14-week interim evaluation was an increased incidence of minimal focal inflammation in the liver of the 5 W/kg males.

In the 2-year study, percent survival was significantly higher for the 5 W/kg males than the sham control group. Survival of the other exposed groups of males and females was generally similar to that of the sham controls. Mean

body weights of exposed groups of males and females were similar to those of the sham controls throughout the study.

The combined incidences of fibrosarcoma, sarcoma, or malignant fibrous histiocytoma of the skin were increased in 5 and 10 W/kg males, although not significantly or in an exposure concentration-related manner; however, the incidences exceeded the overall historical control ranges for malignant fibrous histiocytoma. In the lung, there was a significant positive trend in the incidences of alveolar/bronchiolar adenoma or carcinoma (combined) in males. Compared to the sham controls, all exposed groups of females had increased incidences of malignant lymphoma and the incidences in the 2.5 and 5 W/kg groups were significantly increased. The sham control group had a low incidence of malignant lymphoma compared to the range seen in historical controls.

There were no nonneoplastic lesions that were considered related to exposure to GSM-modulated cell phone RFR.

CDMA

28-DAY STUDY

Groups of 10 male and 10 female core study mice and groups of 20 male and 20 female special study mice were housed in reverberation chambers and received whole-body exposures to CDMA-modulated cell phone RFR at power levels of 0 (sham control), 5, 10, or 15 W/kg, for up to 18 hours and 20 minutes per day, 5 or 7 (last week of study) days per week for at least 28 days with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; a shared group of unexposed mice of each sex served as sham controls for both cell phone RFR modulations. All mice survived to the end of the study. Mean body weights of exposed groups of males and females were similar to controls. There were no exposure-related clinical signs, differences in organ weights, or histopathologic findings. Differences in body temperatures between the exposed groups and the control group were not considered to be related to cell phone RFR exposure.

2-YEAR STUDY

Groups of 105 male and 105 female mice were housed in reverberation chambers and received whole-body exposures to CDMA-modulated cell phone RFR at power levels of 0 (sham control), 2.5, 5, or 10 W/kg, 9 hours and 10 minutes per day, 7 days per week for 106 (males) or 108 (females) weeks with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; shared groups of unexposed mice of each sex served as sham controls for both cell phone RFR modulations. Fifteen mice per group were randomly selected from the core group after 10 weeks of study; ten of those 15 mice per group were used for interim evaluation at 14 weeks, and five mice per group were used for genetic toxicity testing at 14 weeks. The remaining 90 animals per group were exposed up to 2 years.

At the 14-week interim evaluation of the 2-year study, mean body weights of exposed groups of males and females were similar to those of the sham controls. There were no changes to the hematology variables attributable to CDMA cell phone RFR exposure. Differences in organ weights in male mice were not associated with histopathologic findings and were not considered related to exposure; there were no significant changes in organ weights in females. In males, there were no exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility. In females, there were no exposure related effects on estrous cyclicity. Compared to the sham controls, there were statistically significant differences for extended estrous in the 2.5 W/kg group and extended diestrus in the 5 W/kg group; however, these changes were considered sporadic due to the lack of an exposure-related response. In the kidney of 10 W/kg females, there was a significantly increased incidence of minimal to mild interstitial lymphocytic cellular infiltration.

Percent survival was significantly higher in 2.5 W/kg males compared to that in the sham controls in the 2-year study. Survival of males and females in all other exposed groups was generally similar to that of the sham controls. Mean body weights of exposed groups of males and females were similar to those of the sham controls throughout the study.

There was a significantly increased incidence of hepatoblastoma in 5 W/kg males. Compared to the sham controls, the incidences of malignant lymphoma were increased in all exposed groups of females, and the increase was significant in the 2.5 W/kg group. As noted for the GSM study, the shared sham control group had a low incidence of malignant lymphoma compared to the range observed in historical controls.

There were no nonneoplastic lesions that were considered related to exposure to CDMA-modulated cell phone RFR.

GENETIC TOXICOLOGY

Comet Assay

As part of the 14-week interim evaluation, samples of frontal cortex, hippocampus, cerebellum, liver, and blood leukocytes were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, and five tissues per animal). Samples of peripheral blood were also evaluated for chromosome damage in the micronucleus assay. Results are based on the 100-cell scoring approach that was standard at the time of the study; data obtained using a second 150-cell scoring approach, recommended in a recently adopted international guideline for the *in vivo* comet assay, are noted for the few instances where results differed between the two methods. Significant increases in DNA damage were observed in cells of the frontal cortex of male mice exposed to both modulations, GSM and CDMA. No other tissues showed evidence of a treatment-related effect in male mice. In female mice exposed to the CDMA modulation, significant increases in DNA damage were seen in blood leukocytes at all three exposure levels using both scoring approaches. No statistically significant increases in percent comet tail DNA were observed in any of the samples from female mice exposed to the GSM modulation with the 100-cell scoring method. Scoring 150 cells resulted in an equivocal response in liver of female mice exposed to CDMA; a similar pattern of response was seen with the 100-cell scoring method, but none of the increases were significant.

Micronucleus Assay

No significant increases in micronucleated red blood cells or changes in the percentage of immature erythrocytes among total erythrocytes were observed in the peripheral blood of mice of either sex exposed to either modulation of cell phone RFR.

CONCLUSIONS

Under the conditions of these 2-year studies, there was *equivocal evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 1,900 MHz in male B6C3F1/N mice based on the combined incidences of fibrosarcoma, sarcoma, or malignant fibrous histiocytoma in the skin and the incidences of alveolar/bronchiolar adenoma or carcinoma (combined) in the lung. There was *equivocal evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 1,900 MHz in female B6C3F1/N mice based on the incidences of malignant lymphoma (all organs). There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 1,900 MHz in male B6C3F1/N mice based on the incidences of hepatoblastoma of the liver. There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 1,900 MHz in female B6C3F1/N mice based on the incidences of malignant lymphoma (all organs).

Exposure to GSM- or CDMA-modulated cell phone RFR at 1,900 MHz did not increase the incidence of any nonneoplastic lesions in male or female B6C3F1/N mice.

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* Explanation of Levels of Evidence of Carcinogenic Activity is on page 14.

Summary of the 2-Year Carcinogenesis and Genetic Toxicology Studies of GSM- and CDMA-Modulated Cell Phone RFR Exposure in Mice

	GSM-Modulated Cell Phone RFR Male Mice	GSM-Modulated Cell Phone RFR Female Mice	CDMA-Modulated Cell Phone RFR Male Mice	CDMA-Modulated Cell Phone RFR Female Mice
Whole-body GSM- or CDMA-modulated cell phone RFR exposure	0, 2.5, 5, or 10 W/kg	0, 2.5, 5, or 10 W/kg	0, 2.5, 5, or 10 W/kg	0, 2.5, 5, or 10 W/kg
Survival rates	66/90, 63/90, 80/90, 72/90	67/90, 74/90, 70/90, 73/90	66/90, 83/91, 71/90, 71/90	67/90, 75/89, 70/90, 72/90
Body weights	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group
Nonneoplastic effects	None	None	None	None
Neoplastic effects	None	None	None	None
Equivocal findings	Skin: fibrosarcoma, sarcoma, or malignant fibrous histiocytoma (1/90, 1/89, 5/90, 4/90) Lung: alveolar/bronchiolar adenoma or carcinoma (23/90, 24/89, 32/90, 34/90)	All organs: malignant lymphoma (2/90, 13/90, 9/90, 6/90)	Liver: hepatoblastoma (6/90, 6/89, 16/90, 7/90)	All organs: malignant lymphoma (2/90, 9/89, 6/90, 7/90)
Level of evidence of carcinogenic activity	Equivocal evidence	Equivocal evidence	Equivocal evidence	Equivocal evidence
Genetic toxicology				
DNA damage:				
GSM-modulated		Positive in frontal cortex (males); negative in frontal cortex (females); negative in hippocampus, cerebellum, liver, and leukocytes (males and females)		
CDMA-modulated		Positive in frontal cortex (males) and leukocytes (females); negative in hippocampus, cerebellum, and liver (males and females); negative in leukocytes (males) and frontal cortex (females)		
Micronucleated erythrocytes in peripheral blood *in vivo*:				
GSM-modulated		Negative in males and females		
CDMA-modulated		Negative in males and females		

EXPLANATION OF LEVELS OF EVIDENCE OF CARCINOGENIC ACTIVITY

The National Toxicology Program describes the results of individual experiments on a test agent and notes the strength of the evidence for conclusions regarding each study. Negative results, in which the study animals do not have a greater incidence of neoplasia than control animals, do not necessarily mean that a test agent is not a carcinogen, inasmuch as the experiments are conducted under a limited set of conditions. Positive results demonstrate that a test agent is carcinogenic for laboratory animals under the conditions of the study and indicate that exposure to the test agent has the potential for hazard to humans. Other organizations, such as the International Agency for Research on Cancer, assign a strength of evidence for conclusions based on an examination of all available evidence, including animal studies such as those conducted by the NTP, epidemiologic studies, and estimates of exposure. Thus, the actual determination of risk to humans from test agents found to be carcinogenic in laboratory animals requires a wider analysis that extends beyond the purview of these studies.

Five categories of evidence of carcinogenic activity are used in the Technical Report series to summarize the strength of evidence observed in each experiment: two categories for positive results **(clear evidence and some evidence)**; one category for uncertain findings **(equivocal evidence)**; one category for no observable effects **(no evidence)**; and one category for experiments that cannot be evaluated because of major flaws **(inadequate study)**. These categories of interpretative conclusions were first adopted in June 1983 and then revised on March 1986 for use in the Technical Report series to incorporate more specifically the concept of actual weight of evidence of carcinogenic activity. For each separate experiment (male rats, female rats, male mice, female mice), one of the following five categories is selected to describe the findings. These categories refer to the strength of the experimental evidence and not to potency or mechanism.

- **Clear evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.
- **Some evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a test agent-related increased incidence of neoplasms (malignant, benign, or combined) in which the strength of the response is less than that required for clear evidence.
- **Equivocal evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be test agent related.
- **No evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing no test agent-related increases in malignant or benign neoplasms
- **Inadequate study** of carcinogenic activity is demonstrated by studies that, because of major qualitative or quantitative limitations, cannot be interpreted as valid for showing either the presence or absence of carcinogenic activity.

For studies showing multiple test agent-related neoplastic effects that if considered individually would be assigned to different levels of evidence categories, the following convention has been adopted to convey completely the study results. In a study with clear evidence of carcinogenic activity at some tissue sites, other responses that alone might be deemed some evidence are indicated as “were also related” to test agent exposure. In studies with clear or some evidence of carcinogenic activity, other responses that alone might be termed equivocal evidence are indicated as “may have been” related to test agent exposure.

When a conclusion statement for a particular experiment is selected, consideration must be given to key factors that would extend the actual boundary of an individual category of evidence. Such consideration should allow for incorporation of scientific experience and current understanding of long-term carcinogenesis studies in laboratory animals, especially for those evaluations that may be on the borderline between two adjacent levels. These considerations should include:

- adequacy of the experimental design and conduct;
- occurrence of common versus uncommon neoplasia;
- progression (or lack thereof) from benign to malignant neoplasia as well as from preneoplastic to neoplastic lesions;
- some benign neoplasms have the capacity to regress but others (of the same morphologic type) progress. At present, it is impossible to identify the difference. Therefore, where progression is known to be a possibility, the most prudent course is to assume that benign neoplasms of those types have the potential to become malignant;
- combining benign and malignant tumor incidence known or thought to represent stages of progression in the same organ or tissue;
- latency in tumor induction;
- multiplicity in site-specific neoplasia;
- metastases;
- supporting information from proliferative lesions (hyperplasia) in the same site of neoplasia or other experiments (same lesion in another sex or species);
- presence or absence of dose relationships;
- statistical significance of the observed tumor increase;
- concurrent control tumor incidence as well as the historical control rate and variability for a specific neoplasm;
- survival-adjusted analyses and false positive or false negative concerns;
- structure-activity correlations; and
- in some cases, genetic toxicology.

NATIONAL TOXICOLOGY PROGRAM TECHNICAL REPORTS
PEER REVIEW PANEL

The members of the Peer Review Panel who evaluated the draft NTP Technical Report on GSM- and CDMA-modulated cell phone RFR in mice on March 26-28, 2018, are listed below. Panel members serve as independent scientists, not as representatives of any institution, company, or governmental agency. In this capacity, panel members have five major responsibilities in reviewing the NTP studies:

- to ascertain that all relevant literature data have been adequately cited and interpreted,
- to determine if the design and conditions of the NTP studies were appropriate,
- to ensure that the Technical Report presents the experimental results and conclusions fully and clearly,
- to judge the significance of the experimental results by scientific criteria, and
- to assess the evaluation of the evidence of carcinogenic activity and other observed toxic responses.

SUMMARY OF PEER REVIEW PANEL COMMENTS

NOTE: A summary of the Peer Review Panel's remarks will appear in a future draft of this report.

INTRODUCTION

GSM- AND CDMA-MODULATED CELL PHONE RADIO FREQUENCY RADIATION

Synonyms: Cell phone radio frequency radiation; mobile phone radio frequency radiation

OVERVIEW

All consumer cell phone devices function through the transmission of radio waves on a cellular network. The cellular network itself is composed of a collection of individual "cells" that include a fixed-location transceiver (a device that transmits and receives radio signals), also referred to as a cell tower. The collection of adjacent smaller "cells" in the cellular network enables cell phones and towers to use low-power transmitters, thereby allowing for the same frequencies to be reused in non-adjacent cells without interference. Together the individual "cells" comprise the cellular network that provides coverage over a large geographical area. In the United States, there are two major nation-wide cellular networks: CDMA (Code Division Multiple Access) and GSM (Global System for Mobile Communications). With technologies rapidly evolving to meet consumers' increased demand for better coverage, increased call quality, faster data transfer rates, and increased accessibility, the terms CDMA and GSM tend to group together multiple, sometimes successive, technologies that are implemented by the service providers that maintain the two networks. In the United States, Sprint® and Verizon® use and maintain the CDMA network; AT&T® and T-Mobile® use and maintain the GSM network.

For both the GSM and CDMA networks, transmissions occur at specific radio wave frequencies, which are allocated and regulated by the Federal Communications Commission (FCC). While the transmission of radio signals occurs at the same frequencies for both networks, the networks differ in the method by which their signal is modulated. In telecommunications, modulation is a process of conveying a signal, like a cell phone user's voice during a call,

inside another signal that can be physically transmitted. This process involves modulation of the signal prior to transmission at one end, and then demodulation at the other end. Since this process requires different technologies for CDMA and GSM, many cell phones are not interchangeable between the two networks and will only function on one or the other of the networks, not both.

The constantly evolving cellular technologies are commonly referred to by their successive generations (G). The first generation (1G) devices were analogue phones, as opposed to the digital phones of today. Digital voice systems of the second generation (2G) replaced the analogue system of 1G. At the time that these studies were being designed, 2G technology was the primary technology in use and 3G technologies were emerging. Therefore, the current studies were conducted using modulated signals that replicated the 2G and 3G technology in use at the time. Over the course of the studies, however, more advanced 4G technologies were developed. Currently, all of these technologies (2G, 3G, and 4G) are still actively in use for mobile communication applications. 2G and 3G are still the basis for voice calling applications, while 3G and 4G technologies were primarily developed to offer faster access to the internet. Some of the 3G technology is based on 2G technology. While 2G technology is being phased out in the United States, this technology will remain in use in other places throughout the world. More advanced and efficient technologies that are currently in development, such as 5G, will utilize higher frequencies than existing technologies.

RADIO FREQUENCY RADIATION (RFR)

In the context of this report, radio frequency (RF) radiation refers to the broad range of electromagnetic fields from 3 kilohertz (3 kHz) to 300 gigahertz (300 GHz). Different applications utilize different frequency bands within the RF portion of the electromagnetic spectrum. The range of frequencies for radio and television are in the 145 kHz to 850 MHz range. These include long, medium, shortwave, and very high frequency (VHF) radio transmissions and VHF and ultra-high frequency (UHF) over-the-air television transmissions. Wireless communications and networking typically utilize frequencies between 800 MHz and 6 GHz. Cell phone networks (2G, 3G, and 4G) utilize frequencies in the range of 600 MHz to 5.7 GHz. In the United States, wireless telecommunications networks and devices operate in bands at frequencies of nominally 800 MHz, 850 MHz, or 1,900 MHz for 2G; 850 MHz, 1,700 MHz, 1,900 MHz, or 2,100 MHz for 3G; and 600 MHz, 700 MHz, 800 MHz, 850MHz, 1,700 MHz,

1,900 MHz, 2,100 MHz, 2,300 MHz, 2,500 MHz, 5,200 MHz, or 5,700 MHz for 4G. The next generation, i.e., the 5th generations of wireless communications, will also utilize the RFR spectrum above 6 GHz. Other terms are also used in the literature for part of the RFR spectrum, e.g., microwaves for frequencies above 1 GHz, millimeter waves for frequencies above 30 GHz.

CELL PHONES AND RFR

Cell phones and other commonly used wireless communication devices transmit their signals via RFR to enable voice calls and data transfer, including communication through the internet. Wireless phones are two-way radios that contain both a receiver and a transmitter. When a user makes a call, voice sound is converted into digital information. The information is imposed on to RFR and transmitted to the nearest base station. Base stations, commonly referred to as cell towers, have antennas placed on towers that are free standing or mounted on existing structures such as trees, water tanks, or tall buildings and contain electronic equipment and antennas that receive and transmit RF signals and form a bridge to the rest of the communications infrastructure. The base station receives and transmits radio signals in its area or "cell." As the user moves around, the radio signal can be relayed within the communications network from one "cell" of coverage to another, maintaining call connection. The call is routed through the communications network either through a land line phone or another wireless phone again using radio signals. To conserve energy and minimize interference, mobile phones automatically regulate the RFR signal strength, and hence the emitted field, to the lowest power level possible for a connection to be made. However, in a poor transmission environment (caused by, e.g., a distant base station, presence of obstacles between the base station and the mobile phone, or interferences from adjacent calls) there is a higher output power and emission from the mobile phone in order to make a connection. Therefore, the better the connection, the lower the power output of the wireless device.

PROPERTIES OF CELL PHONE RFR

Cell phone RFR is a form of nonionizing electromagnetic energy that consists of propagating electromagnetic waves of oscillating electric (E-) and magnetic (H-) fields that move together through space at the speed of light. As opposed to ionizing radiation, which contains enough energy when passing through matter to break chemical bonds or remove an electron from an atom or molecule to produce charged ions, nonionizing radiation refers to electromagnetic energy that at most only has sufficient energy for excitation of an electron to a higher energy state. Nonionizing radiation includes a broad range of the electromagnetic spectrum from extremely low frequency (ELF) radiation to radio and microwaves, infrared, visible light, and near ultraviolet radiation. It has a lower frequency and longer wavelength than ionizing radiation (Figure 1).

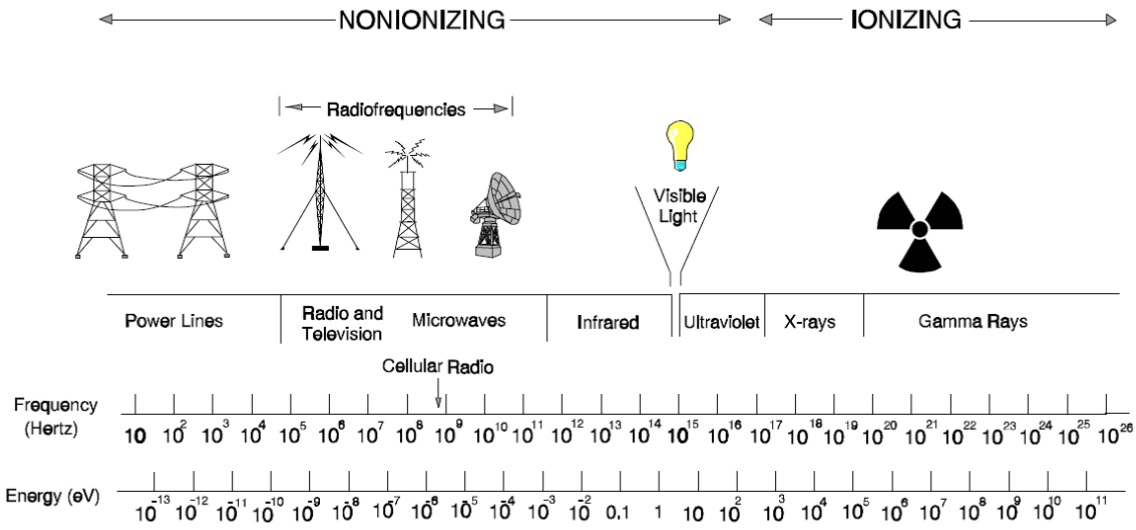


FIGURE 1
Electromagnetic Spectrum (OET, 1999)

Cell phone RFR fields transport large amounts of data at a very fast rate over long distances. RF waves are characterized by their wavelength (the distance covered by one complete cycle of the electromagnetic wave) and their frequency (the number of electromagnetic waves passing a given point in 1 second). The frequency of an RF signal is expressed in terms of Hertz (Hz), where one Hz is equivalent to one cycle per second. The RF segment of the electromagnetic spectrum is generally defined as the frequencies between approximately 3 kHz to 300 GHz. The intensity of an RF field can be expressed by its electric and magnetic components and is measured in volts per meter (V/m) for electric fields and amperes per meter (A/m) for magnetic fields. Another measure of RFR is the power density, which is defined as the power per unit area and is expressed in watts per square meter (W/m^2) in the far-field of sources. The quantity used to describe the amount of RFR energy absorbed by the body is referred to as the specific absorption rate (SAR), which is expressed in watts per kilogram (W/kg). SAR is a function of the geometry and the dielectric properties of biological tissues absorbing the energy (which result from the interaction of electromagnetic radiation with constituents at the cellular and molecular level), the square of the strength of the induced E-field, and the mass density of the exposed tissue. The SAR value is derived by averaging the absorbed energy over a specific volume (typically 1 gram, 10 grams, or the whole body for regulatory purposes).

Cell Phone RFR Signal Modulation

In wireless telecommunications, modulation is the process of conveying digital or analog signals or information (the message) by varying one or more parameters of another signal (the carrier), typically at a much higher frequency, that can be transmitted over a distance. The modulated carrier contains complete information about the message signal and the original message can be recovered by suitable signal processing of the signal when received at a remote location (base station). One of the main goals of the modulation used in mass wireless communication systems is to transfer as much data as possible in the least amount of spectrum. Over the years, multiple modulation techniques have emerged to achieve and improve spectral efficiency, either when considering a single user in isolation or multiple users simultaneously using the same spectrum.

Cell phone technology is typically referred to in “generations.” The first generation (1G) of wireless technology was an analog system that used analog frequency modulation for voice calls. The 1G networks were introduced in the 1980s and continued until they were replaced by networks of the second-generation (2G) networks. These

networks differed from the 1G networks in that they were digital, provided encryption, were significantly more efficient, and introduced data services [i.e., text messages, picture messages, and Multimedia Message Service (MMS)] in addition to voice calls. The 2G networks became commercially available in 1992 and used three common multiple access technologies for accommodating multiple simultaneous users:

- Frequency Division Multiple Access (FDMA): the available spectrum is split into a number of distinct parts (channels) each large enough to accommodate a single user or call without overlap; all users utilize their channel 100% of the time for the duration of the call or message. The channels are normally of equal bandwidth;
- Time Division Multiple Access (TDMA): the available spectrum is allocated to a single channel; each user or call is assigned a certain portion of time;
- Code Division Multiple Access (CDMA): the available spectrum is allocated to a single channel; each user or call is assigned a unique sequence code to spread the message over the available spectrum. All users use the whole of the spectrum all of the time. At the receiver, the same unique sequence code is used to recover the desired signal from the sum of all the user calls.

2G systems used a combination of FDMA/TDMA or CDMA for, for example, GSM and cdmaOne (IS-95), respectively. While the 2G technology continues to operate, subsequent third and fourth generations of network technologies were introduced in 1998 (3G), 2006 (4G), and 2011 (4G-LTE). These technologies were developed to support increased data needs for multimedia access with increased bandwidth and transfer rates to accommodate internet-based broadband applications, including video conferencing, streaming video, sending and receiving faxes, and instantly downloading e-mail messages with attachments. With the introduction of 3G technology, "smartphones" were developed. With these devices, the newer technologies were overlaid with 2G to support multiple access modes (2G, 3G, and 4G) (Buddhikot *et al.*, 2009). Although the 2G technologies will be phased out over time and replaced by newer technologies, the current wireless communication networks continue to utilize 2G for voice and text.

All 3G systems utilize CDMA/WCDMA technology and fall into two groups complying with the 3rd Generation Partnership Project (3GPP) or 3GPP2 family of standards. Universal Mobile Telecommunications Service (UMTS), Wideband Code Division Multiple Access (WCDMA), and Time Division-Synchronous Code Division Multiple Access (TD-SCDMA) are 3GPP variants; CDMA2000 (which is based on 2G cdmaOne) is 3GPP2. 4G systems use Orthogonal Frequency Division Multiplexing (OFDM) within the E-UTRAS (LTE-Advanced) or Worldwide Interoperability for Microwave Access (WiMAX) standards.

Modulation Schemes (GSM and CDMA)

The Global System for Mobile Communications (originally *Groupe Spécial Mobile*; GSM) was developed to establish a digital standard for compatibility throughout Europe. GSM is a circuit-switched system that uses both FDMA and TDMA technologies. The frequency division mechanism divides the GSM band into 200 kHz-wide channels. The time division mechanism enables up to eight time slots (voice channels) per frequency channel wherein a single cell phone transmits in only one out of eight available time slots during a voice communication. This introduces a pulsed signal shape with a pulse repetition rate of 217 Hz. Such a TDMA frame has a length of 4.6 milliseconds (ms), and 26 TDMA frames make up a multiframe with a 120 ms duration. During a multiframe, a mobile phone transmits in 25 out of 26 possible time slots. This TDMA frame structure causes significant low frequency amplitude modulation components to be superimposed on the RF carrier at 8.3 and 217 Hz.

With GSM, the duplexing between uplink (when the handset transmits to the base station) and downlink (when the base station transmits to the handset) is implemented in the frequency and time domain. Constant frequency spacing is maintained between up and downlink frequencies: in the United States, the uplink is 1,850 to 1,910 MHz, and the downlink is 1,930 to 1,990 MHz. The uplink and downlink frequencies are chosen according to the cell (area that is covered by a base station) into which the mobile is registered. In order to minimize interference between neighboring cells, a frequency reuse policy is applied. In this approach, when a mobile phone moves from one cell into an adjacent cell, frequencies used for data uplink and downlink change in association with this movement (i.e., transmission frequencies change at handover from one cell to another).

GSM technology implements a power control in order to increase the battery life of mobile handsets. The power control has a dynamic range of 30 decibels (dB) subdivided into 2 dB power-level steps. The power control is typically implemented using the Slow Associated Control Channel (SACCH), which facilitates a power control update rate no faster than every four multiframes (480 ms). Once a target power level is received, the mobile station is able to regulate its power in 2 dB steps every 60 ms. This means that power regulation over 15 steps (full dynamic range) takes 900 ms. GSM base stations typically average the received signal strength from a mobile phone over 1 second, such that the actual power regulation usually takes place after multiples of 480 ms.

The GSM supports data transfer speeds up to 9.6 kilobits/second, allowing the transmission of basic data services such as Short Message Service (SMS), but not large packets of data such as internet access and streaming video.

CDMA technology uses a form of coded transmission known as Direct Sequence Spread Spectrum (DSSS) in which data multiplies by a much faster pseudo random code before being modulated on to the carrier. The effect of the multiplication is to spread the message across all frequency bands available for use at any time but with very specific characteristics. CDMA signal access technology is based on code division separation of mobile stations as well as base stations. This implies strong differences of the signal structure compared to GSM. For example, in Interim Standard 95 (IS-95), in the forwardlink (downlink), a set of 64 Walsh codes (which are deterministic and orthogonal) are applied to spread/separate the individual channels in the downlink of a cell. After the orthogonal spreading, a short (16-bit) Pseudo Noise (PN) code is applied to further spread the signal and identify the cell. Hence, a separation of neighboring cells in the frequency domain is no longer necessary. Eventually, there is no need for the mobile station to change its transmission frequency during the transition from one cell into another. As with GSM systems, the duplexing between the forward and reverse links is implemented in the frequency domain. In CDMA systems, an efficient power control is crucial. Because all mobile stations transmit and interfere in the same frequency channel, each mobile device decreases the signal to noise ratio of all the other mobile devices. Hence, the output power of a mobile phone should be kept at a minimum that guarantees good transmission quality. On the other hand, when moving around, the mobile device is subject to slow and fast fading, shadowing, external interference, etc. In order to keep the signal received at the base station constant and compensate for effects on the communication channel, a fast power control is necessary. Therefore, when a CDMA mobile station is active

(communicating), a closed-loop power control is applied. The base station monitors the signal quality in the reverse link and inserts power-control bits in the communication channel. For example, in IS-95, the power control over a dynamic range of 48 dB in 1 dB steps with an update rate of 800 MHz is implemented. The power control is implemented by sending a binary value of "1" to regulate the transmit power 1 dB down, and "0" to regulate the transmit power 1 dB up. A quasistatic power level is therefore implemented by an alternating 0101 power-control pattern.

IS-95 (also known as cdmaOne) was developed by Qualcomm (San Diego, CA) as the first 2G CDMA-based digital cellular technology. The term IS-95 generally applies to a protocol revision (P_REV=1) that was adopted as a standard (TIA-EIA-95) by the Telecommunications Industry Association (TIA) in 1995. Over time, subsequent iterations of the IS-95 protocol such as IS-95A, TSB-74, and IS-95B were developed, each with incremental improvements over the previous protocols. Later, more advanced versions of the CDMA technology evolved to include IS-2000, which incorporated much higher transfer rates than the previous 2G versions.

SOURCES, USE, AND HUMAN EXPOSURE

The predominant source of RFR for the majority of the population is in telecommunications and mobile internet access applications for wireless devices. Aside from telecommunications, there are other man-made applications of RFR, which include microwave ovens, radar, industrial heating and sealing, medical diagnostics [Magnetic Resonance Imaging (MRI)] and therapy (surgical diathermy and ablation), and remote tracking or detection of objects [anti-theft, Radio Frequency Identification (RFID)]. However, there are also natural sources of RFR such as atmospheric electrical discharges (lightning) and solar and cosmic radiation. RFR exposures from natural sources are much smaller and tend to be spread over a much wider range of frequencies compared to man-made fields (IARC, 2013).

Highest human exposure to cell phone RFR primarily occurs through the use of cellular phone handsets and other wireless devices held in closest proximity to the human body such as tablets and laptop computers. The use of cell phones has become widespread over the last two decades amongst adults and children, thereby increasing the level of RFR the population is exposed to. Concern has been expressed regarding the potential health risks associated

with use of cell phones. Particularly, there has been a great deal of focus on the possibility of increased risk of brain cancer because traditionally, these devices were used in close proximity (0 to 2 cm) to the head, yet the advent of smart phones has altered dramatically the usage scenarios for such devices away from a simple phone call. The RFR exposure of a person is defined in terms of SAR, the power absorbed in the body, because the body has complex geometry and tissue distributions, and even exposure to uniform RFR electromagnetic fields (EMF) will result in nonuniform SAR distributions. In general (apart from the case when very close to the antenna), the level of RFR exposure by a cell phone is inversely proportional to the square of the distance of the body from the device's antenna, and the highest SAR levels occur in the parts of the body nearest to the antenna. Accordingly, there is a very nonuniform exposure to cell phone RFR across the whole body of cell phone users and even of bystanders.

Accurate and detailed estimation of cell phone RFR exposure in humans is difficult to obtain because the output power of wireless devices constantly varies depending on several factors. Overall, the network carrier adjusts the output power of each connected device to the lowest level that is still compatible with a good quality signal. This adaptive power control occurs continuously and is achieved by a logarithmic downscaling of the time-averaged power from the maximum of 0.125 and 0.25 W to a level as low as 1 mW. When a device is in use, the output power (and subsequent exposure to cell phone RFR) from the device is increased compared to the output from that same device in "standby" mode. Therefore, levels of exposures are related to the amount of active time a user spends on the device. The output power of a device changes based on the signal received at the base station. Decreases in signal strength result in higher output powers. Therefore, there are increases in the output power as the distance between the device and the base station increases, if there are physical obstacles between the device and the base station, multipath reflections, and during handovers in the case of GSM (handover is the passing of a call from one base station to another when the user moves across the borders of cells or by network request to optimize communication traffic). The proximity of the device to the body and the type, number, and position of antennas in the device are other important factors affecting the amount of exposure to cell phone RFR.

Potential exposure to cell phone RFR also occurs from the cell phone towers (or base stations) that form the network. While modern towers emit substantially more power than devices, exposures from base station antennas are considerably lower in users than from the hand-held device. Typically, base station antennas are placed at

heights of 50 to 200 feet, in order to adequately cover an area (or cell). The antennas direct RF energy toward the horizon, with some downward tilt. As with all forms of radiation (ionizing and nonionizing), the RF energy level decreases rapidly as the distance from the antenna increases. As a result, the level of exposure to cell phone RFR at ground level is very low compared to the level close to the antenna. Overall, the exposure level from base stations is very small compared to exposure from the handheld devices.

Some base station antennas are installed on rooftops and at the top of lamp poles that are in close proximity or adjacent to office space and residential buildings. Levels of exposure from these sources can approach or exceed Federal Communications Commission (FCC) safety guidelines. Occupational exposure occurs during maintenance on base stations. As a result, the FCC established guidelines for occupational exposures. Safety guidelines and regulatory compliance are discussed below.

The levels of cell phone RFR inside buildings with base station antennas mounted on the roof or on the side of the building are typically much lower than the levels outside, depending on the construction materials of the building. Wood or cement block reduces the exposure to cell phone RFR by a factor of about 10. Due to the directional nature of the signals, the energy level behind an antenna is orders of magnitude lower than in front of the antenna.

According to a Pew Research poll (Pew, 2017), approximately 95% of adult Americans own a cell phone. As of December 2015, the number of active wireless subscriber connections was 377.9 million, which exceeded the population of the United States (CTIA, 2017). According to the same survey, 49.3% of households in the United States utilize only a wireless phone, and not a landline.

Safety Guidelines for Exposure

The Federal Communications Commission (FCC) and U.S. Food and Drug Administration (FDA) are jointly responsible for the regulation of wireless communication devices.

Federal Communications Commission

The FCC is required by its responsibilities under the National Environmental Policy Act of 1969 to evaluate the impact of emissions from FCC-regulated transmitters on the quality of the human environment (42 USC §4321 *et seq.*). As a result, the FCC regulates both the wireless devices as well as the base stations that form the cells of the network. Since 1996, the FCC has required that all wireless communications devices (transmitting in the 100 kHz to 6 GHz frequency range) sold in the United States comply with its minimum guidelines for safety and maximum RFR absorption standards based on SAR. The FCC requires a formal approval process for all devices sold in the United States. FCC approval is contingent on the demonstration that the device does not exceed the maximum allowable SAR level when the device is operating at its maximum power. The SAR limit adopted by the FCC for exposure in the general population is 0.08 W/kg, as averaged over the whole body, and a peak spatial-average SAR of 1.6 W/kg, averaged over any 1 gram of tissue (47 CFR §1.1310) when averaged over 6 minutes. Exceptions are made for the extremities (hands, wrists, feet, ankles, and pinnae), where the peak spatial-average SAR limit is 4 W/kg, averaged over any 10 grams of tissue for an exposure period of no longer than 30 minutes. For occupational exposures, the whole-body SAR limit is 0.4 W/kg, and the limit for the peak spatial-average SAR is 8 W/kg, averaged over any 1 gram of tissue. For the hands, wrists, feet, ankles, and pinnae, the peak spatial-average SAR limit for occupational exposure is 20 W/kg, averaged over any 10 grams of tissue for an exposure period not to exceed 6 minutes.

The FCC rules and guidelines for cell phone RFR exposure are based upon standards initially developed by the Institute of Electrical and Electronics Engineers (IEEE) and the National Council on Radiation Protection and Measurements (NCRP). These standards for RF exposure in workers and the general population are based on protection against adverse effects that might occur due to increases in tissue or body temperature in excess of 1° C (wbSAR, approximately 4 W/kg) or less (after applying safety factors). Because RF-energy absorption and any induced effects are dependent on the frequency of incident-field parameters and the composition of exposed tissues, it has been suggested that quantifying SARs in small averaging regions is more relevant for evaluations of human health effects.

Food and Drug Administration

The FDA does not currently regulate the use of wireless communications devices or the devices themselves. The FDA also does not require safety evaluations for radiation-emitting wireless communication devices. It does maintain the authority to take regulatory action if it is demonstrated that exposure to the emitted cell phone RFR from these devices is hazardous to the user.

ABSORPTION OF CELL PHONE RFR

RFR interacts with the human body via inductive or capacitive coupling or a combination of both. The absorption of the coupled RFR is dependent on the frequency of the signal and the dielectric properties of the exposed tissue. It generates oscillating currents in the tissue, which in turn give rise to induced E-fields. The energy is transferred into molecular motion of polar molecules like water, a strongly dipolar molecule and major component of biological tissues. Resonant oscillations in polar subgroups of cellular macromolecules are damped by collisions with surrounding water molecules that disperse the energy of the RF signal into random molecular motion. Tissue heating occurs as the energy is transferred to the surrounding aqueous environment as heat (IARC, 2013).

The SAR (W/kg) is a measure of the absorption of RF energy by biological tissues. It is a function of several main factors: the electrical conductivity (Siemens/meter) of the tissue, the square of the strength (Volts/meter) of the induced E-field, and the geometry and mass density (kg/meter^3) of the tissue absorbing the energy. The SAR is calculated as the average of the absorbed power over a specific volume of tissue (typically 1 or 10 gram volume of tissue or the whole body).

TOXICITY

A comprehensive review of the toxicity of cell phone RFR in *in vitro* models, laboratory animals, and humans was recently conducted and published in the IARC Monograph series (IARC, 2013).

Thermal Effects

Given the ability of cell phone RFR to heat tissues, the toxic effects of cell phone RFR are often classified as thermal or nonthermal effects, based on whether the observed effect was a result of a significant temperature change (thermal effects) or independent of any change in temperature considered in excess of thermal noise (nonthermal effects). The most well-established and biologically plausible mechanism for cell phone RFR-induced effects in biological systems is through tissue heating resulting in damage. It has been well established that excessive heating causes significant damage to cells, tissues, and organs. At high enough levels of cell phone RFR exposure, the absorption of energy could lead to increased heating to the point that it overwhelms an organism's ability to thermoregulate and maintain an acceptable body temperature. Because human exposures to cell phone RFR occur at intensities that are not expected to cause significant thermal effects, the nonthermal effects are more appropriate to the evaluation of potential effects in humans.

Nonthermal effects refer to biological changes that occur with body temperature increases that are below 1° C. Changes of temperature up to 1° C are considered in the range of thermal noise (IARC, 2013). There is an ongoing debate regarding whether nonthermal biological effects can occur as a result of exposures to low-intensity cell phone RFR. It has been suggested that there is no plausible nonthermal mechanism by which exposure to low-intensity RFR could induce significant biological effects (Adair, 2003; Prohofsky, 2004; Sheppard *et al.*, 2008). However, there are numerous reports of specific biological effects associated with cell phone RFR exposures at levels considered below those expected to result in a measurable amount of tissue heating. Other than tissue heating, the mechanisms of interaction between cell phone RFR and biological systems have not been well characterized, but several mechanisms have been proposed for these nonthermal effects in biological systems, including the generation of reactive oxygen species, induction of ferromagnetic resonance, demodulation of pulsed RF signals, and the alteration of ligand binding to hydrophobic sites in receptor proteins (IARC, 2013). Additionally, low levels of exposure to cell phone RFR may result in small temperature changes in localized areas of exposed tissues that cause conformational changes in temperature-sensitive proteins and induce the expression of heat-shock proteins.

Experimental Animals

Toxic effects have been reported in various types of studies in cell phone RFR-exposed laboratory animals and *in vitro* systems. Most studies investigating the potential toxicity of cell phone RFR have focused primarily on genotoxicity and related effects; these findings are summarized in the Genetic Toxicity section. However, several studies have been conducted to evaluate other aspects of toxicity, including specific studies on gene and protein expression, immunotoxicity, and permeability of the blood-brain barrier. The results of these studies have been mixed. It is important to note that these studies were conducted with cell phone RFR of differing parameters (frequency, power density, continuous wave versus amplitude-modulated signals, etc.). Because there may be differences in cell phone RFR-induced responses depending on the frequency, modulation, and power density, it is not surprising that the results reported in the literature can be somewhat inconsistent.

Several effects on the humoral and cell-mediated responses of the immune system have been reported at various frequencies of cell phone RFR in rats and mice. These include effects on the activity of NK cells, plaque-forming cell response to sheep erythrocytes, production of tumor necrosis factor (TNF) in peritoneal macrophages and splenic T-cells, mitogenic response in T lymphocytes, phagocytic activity of neutrophils, leukocyte profile, and thymic and splenic cellularity (Smialowicz *et al.*, 1983; Guy *et al.*, 1985; Veyret *et al.*, 1991; Novoselova *et al.*, 1999; Lushnikov *et al.*, 2001; Kolomytseva *et al.*, 2002). However, many of these effects were observed in studies conducted with cell phone RFR at frequencies greater than 10 GHz. Other studies have demonstrated no exposure-related effects on the immune system (Elekes *et al.*, 1996; Chagnaud and Veyret, 1999; Lushnikov *et al.*, 2001; Gatta *et al.*, 2003; Nasta *et al.*, 2006).

A few studies have investigated the impact of cell phone RFR at frequencies between 800 and 1,900 MHz on gene and protein expression. Several studies have demonstrated that cell phone RFR can alter the expression of certain genes in the brain (Fritze *et al.*, 1997; Belyaev *et al.*, 2006; Nittby *et al.*, 2008), while others have failed to associate cell phone RFR exposure with changes in gene expression (Stagg *et al.*, 2001; Paparini *et al.*, 2008). The expression of various proteins has also been investigated in rats and mice. These studies have primarily yielded negative results for the specific proteins being evaluated in the rat brain (Fritze *et al.*, 1997; Belyaev *et al.*, 2006; Ammari *et al.*, 2008, 2010; Dasdag *et al.*, 2009). Similarly, no effects of cell phone RFR on protein expression have been reported

in the testis (Lee *et al.*, 2010) or in the skin (Masuda *et al.*, 2006; Sanchez *et al.*, 2006, 2008). Changes in the expression of bone morphogenic protein and bone morphogenic protein receptors have been reported in the kidney of newborn rats (Pyrpasopoulou *et al.*, 2004). A study by Eşmekaya *et al.* (2010) also demonstrated increased expression and activity for caspase 3 and caspase 9 in the thyroid gland of Wistar rats.

Exposure to cell phone RFR induces changes in markers for oxidative stress in multiple tissues, including the brain (Ilhan *et al.*, 2004; Meral *et al.*, 2007; Ammari *et al.*, 2008; Sokolovic *et al.*, 2008; Imge *et al.*, 2010), heart (Ozguner *et al.*, 2005a), kidney (Oktem *et al.*, 2005; Ozguner *et al.*, 2005b), eye (Ozguner *et al.*, 2006), liver (Ozgur *et al.*, 2010; Tomruk *et al.*, 2010), endometrium (Oral *et al.*, 2006; Guney *et al.*, 2007), and testis and epididymis (Mailankot *et al.*, 2009). A few studies have also demonstrated cell phone RFR-mediated effects on differentiation and apoptosis in the endometrium (Oral *et al.*, 2006; Guney *et al.*, 2007) and brain (Dasdag *et al.*, 2009; Sonmez *et al.*, 2010). Changes have also been noted in the permeability of the blood-brain barrier in some studies (Eberhardt *et al.*, 2008; Nittby *et al.*, 2009, 2011). However, other studies conducted under similar experimental conditions failed to demonstrate any effect of cell phone RFR exposure on the permeability of the blood-brain barrier (Grafström *et al.*, 2008; de Gannes *et al.*, 2009; McQuade *et al.*, 2009; Masuda *et al.*, 2009).

Humans

Numerous epidemiology studies have been conducted to investigate the association between exposure to cell phone RFR and health effects in humans. However, many of these studies were conducted in small groups exposed to cell phone RFR signals with different characteristics (frequencies, modulations, intensities, etc.) than the specific frequency bands and modulated cell phone RFR signals used in wireless communication. Many of these studies evaluate microwaves, ELF fields, and radar, which are all different forms of RFR. While these studies may provide additional data for the evaluation of the toxicity of RFR in general, a smaller subset of these studies, which specifically evaluated cell phone RFR at the frequencies and modulations used in wireless communications is more critical to evaluating the potential toxicity of cell phone RFR from mobile communication devices.

There is a very limited set of research investigating the general toxicity of cell phone RFR in humans because most of the focus for research has been on the potential for carcinogenic effects. Studies in humans have failed to

demonstrate any consistent adverse health effects in cell phone RFR-exposed populations. There are reports of some exposed individuals that describe acute, subjective effects following exposure to cell phone RFR, including headaches, fatigue, skin itching, and sensations of heat (Frey, 1998; Chia *et al.*, 2000; Hocking and Westerman, 2000; Sandström *et al.*, 2001; Santini *et al.*, 2002a,b). However, these have primarily been reported in people that consider themselves electrosensitive, and not in the general population. It has been suggested that there are likely other causes, not cell phone RFR, for these subjective symptoms (Kwon and Hämäläinen, 2011). In fact, the validity of electrosensitivity as an actual phenomenon has been questioned and debated. Variable results have been observed in the electroencephalogram (EEG) of volunteers exposed to RFR during sleep. Some studies indicate that exposure to cell phone RFR induces changes in sleep latency and sleep EEG (Mann and Röschke, 1996; Wagner *et al.*, 1998, 2000; Borbély *et al.*,1999; Huber *et al.*, 2000, 2002, 2003; Loughran *et al.*, 2005; Hung *et al.*, 2007; Regel *et al.*, 2007; Lowden *et al.*, 2011). Glucose metabolism in the brain, a marker for brain activity, is increased in the region of the brain closest to the antenna (Volkow *et al.*, 2011). While these results demonstrate exposure-related effects, the toxicologic significance of these findings is unclear.

No effects of cell phone RFR on the neuroendocrine system, auditory and vestibular systems, or consistent effects on cognitive performance have been reported in humans. There is also no clear evidence of effects on heart rate or blood pressure.

CARCINOGENICITY

The carcinogenic potential of cell phone RFR in animals and humans is widely debated. A comprehensive review of the carcinogenicity of cell phone RFR in laboratory animals and humans was recently conducted and published in the International Agency for Research on Cancer (IARC) Monograph series (IARC, 2013).

Experimental Animals

Studies published to date have not demonstrated consistently increased incidences of tumors at any site associated with exposure to cell phone RFR in rats or mice. No increases in tumor incidences were observed in B6C3F1 mice exposed to GSM-modulated cell phone RFR for 24 months (Tillmann *et al.*, 2007), F344 rats exposed to CDMA-modulated cell phone RFR for 24 months (La Regina *et al.*, 2003), or Wistar rats exposed to

GSM-modulated cell phone RFR for 24 months (Smith *et al.*, 2007). In studies conducted in transgenic and tumor-prone mouse strains, exposure to cell phone RFR has not been consistently associated with an increased incidence of tumors at any site (Utteridge *et al.*, 2002; Sommer *et al.*, 2004, 2007; Oberto *et al.*, 2007; Lee *et al.*, 2011). While these studies have advanced the knowledge of the potential toxicity of cell phone RFR, critical limitations in the design of many of these studies severely limit the utility of the information to adequately evaluate the carcinogenicity of cell phone RFR. These limitations include studies with very short daily exposure durations ($\leq$ 2 hours per day) in restrained animals or with levels of cell phone RFR exposures too low to adequately assess carcinogenic potential. The focus of many of these studies conducted in genetically-altered and tumor-susceptible mice was not to evaluate the overall carcinogenicity of cell phone RFR, but to investigate the effects in the specific predisposed tissues in that model.

Based on the constraints in the designs of the existing studies, it is difficult to definitively conclude that these negative results clearly indicate that cell phone RFR is not carcinogenic. To adequately evaluate the potential chronic toxicity and carcinogenicity of cell phone RFR, further studies with enhanced study designs and improved exposure paradigms were needed.

Humans

As a result of the IARC review conducted in 2011, RF electromagnetic fields were classified as possibly carcinogenic to humans (Group 2B). This classification was based on limited evidence of carcinogenicity in humans based on positive associations between exposure to RFR from wireless phones and increased risk for gliomas and acoustic neuromas, specifically in users with the greatest amount of cell phone usage. The IARC Working Group acknowledged that the findings were affected by potential selection and information bias, weakness of associations, and inconsistencies between study results (IARC, 2011).

While several other studies were considered, the IARC evaluation was based primarily on reports from the INTERPHONE Study, the largest research effort conducted to date examining the potential association between exposure to cell phone RFR and cancer in humans. INTERPHONE was an IARC-coordinated research effort that included a series of studies conducted with a common core protocol at 16 study centers in 13 countries: Australia,

Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, and the United Kingdom (Cardis *et al.*, 2007). The studies were specifically designed to investigate the association between cell phone RFR and tumors of the brain (glioma and meningioma), acoustic nerve (schwannoma), and parotid gland. The final report for the INTERPHONE studies was published in 2011 (IARC, 2011).

The results of these studies seemingly demonstrated an elevated risk of glioma and acoustic neuroma in the group in the highest decile for exposure (cumulative phone call time). However, the INTERPHONE study group concluded that recall and selection biases and implausible values for usage reported by the participants in the study may explain the increased risk (INTERPHONE Study Group, 2010, 2011). Further, the INTERPHONE studies and other published epidemiological studies may have been concluded prior to the potential lag time (the interval between the time of the onset of exposure and the subsequent development of a tumor) for the development of slow-growing brain tumors. Overall, the authors of these studies concluded that there was no significant increase in risk of glioma, meningioma, or acoustic neuroma associated with the use of cell phones.

Other studies have compared time trends in cell phone usage and the incidences of different types of cancers to investigate indirect evidence of an association between cell phone RFR and cancer. These studies were conducted across several different countries (Saika and Katanoda, 2011), and in a group of European countries (Lönn *et al.*, 2004; Nelson *et al.*, 2006; Röösli *et al.*, 2007; Deltour *et al.*, 2009; de Vocht *et al.*, 2011), the United States (Muscat *et al.*, 2006; Propp *et al.*, 2006; Inskip *et al.*, 2010), Japan (Nomura *et al.*, 2011), New Zealand (Cook *et al.*, 2003), and Israel (Czerninski *et al.*, 2011). Overall, the evaluations suggest that there were no significant changes in the trends of cancer incidences. Any minor increases in cancer rates that were observed in these studies were attributed to enhanced detection capabilities for cancer that were the result of advances in diagnostic medical equipment, like computerized tomography (CT) scans and MRI.

Several cohort studies have been conducted, but also failed to establish a clear association between cell phone RFR and the development of any of the investigated cancer types (Johansen *et al.*, 2001; Schüz *et al.*, 2006, 2011). Additional studies have demonstrated that there was no association between cell phone usage and pituitary gland tumors (Takebayashi *et al.*, 2008; Schoemaker and Swerdlow, 2009), testicular tumors (Schüz *et al.*, 2006; Hardell

et al., 2007), parotid gland tumors (Hardell *et al.*, 2004; Lönn *et al.*, 2006), uveal melanoma in the eye (Schüz *et al.*, 2006; Stang *et al.*, 2009), and cutaneous melanoma (Hardell *et al.*, 2011). Some studies have demonstrated that there was no association between cell phone usage and leukemia (Johansen *et al.*, 2001; Schüz *et al.*, 2006) and non-Hodgkin's lymphoma (Hardell *et al.*, 2005), whereas others have reported increased risk of non-Hodgkin's lymphoma (Linet *et al.*, 2006) and leukemia (Kaufman *et al.*, 2009).

Many of the epidemiological studies that have been published are limited in their ability to definitively establish a causal association between cell phone usage and increased cancer incidences due to recall and selection bias, confounding factors, and low study participation.

As mentioned previously, the utility of human studies with regard to evaluation of the carcinogenic potential of cell phone RFR is dependent upon the length of time the subjects in the studies were exposed to cell phone RFR. Given the long latency period between the initiation of exposures and the development of tumors, a sufficient duration of exposure must be reached in order to evaluate the association between exposure and cancer outcome. Because widespread usage did not occur until the 1990s in some countries, these populations may not have been exposed long enough to expect any changes in cancer incidences compared to studies in populations where widespread use occurred five or more years earlier in the late 1980s.

GENETIC TOXICITY

Extensive reviews of the literature on the genotoxicity of various frequencies and modulations of cell phone RFR, covering experimental systems ranging broadly from cell-free DNA preparations to cells of exposed animals and humans, have concluded that evidence for cell phone RFR-associated genotoxicity is inconsistent and weak (Brusick *et al.*, 1998; Verschaeve *et al.*, 2010; Repacholi *et al.*, 2012; Vijayalaxmi and Prihoda, 2012). Interpretations of the genotoxicity studies and the ability to draw definitive conclusions based on weight-of-evidence from the large number of studies that have been reported have been hampered by inadequacies in experimental design, especially related to exposure standards and radiation-measuring procedures (Brusick *et al.*, 1998). Although the majority of studies report a lack of effect, the several reports of a positive response are concentrated among experiments assessing chromosomal or DNA damage in mammalian cell systems *in vitro* and *in vivo*. Some key studies

reporting cell phone RFR-associated genotoxicity in human cell lines, including DNA damage and chromosomal effects, could not be replicated (Speit *et al.*, 2007, 2013). A critical complicating factor in the study of the genotoxic effects of cell phone RFR is that under certain conditions, cell phone RFR is sufficiently energetic to heat cells and tissues, and not all studies have considered this factor in their design. Exposure to heat *in vivo* and *in vitro* has produced positive results in tests for genotoxicity, such as the comet assay and micronucleus assay (Asanami and Shimono, 1997; Komae *et al.*, 1999; Speit and Schütz, 2013). The mode of action whereby heat induces these effects appears to be through induction of protein denaturation and aggregation, which can interfere with chromatin structure and slow the kinetics of DNA repair (Kampinga and Dikomey, 2001; Hunt *et al.*, 2007). Thus, heat-induced increases in DNA migration seen in the comet assay may reflect slowed repair of endogenous lesions, and similarly, activity in the micronucleus assay may be due to aneugenic rather than clastogenic events (Asanami and Shimono, 1997; Komae *et al.*, 1999; Speit and Schütz, 2013). Therefore, it is important to distinguish between nonthermal and thermal conditions when studying measures of genotoxicity following exposure to cell phone RFR.

STUDY RATIONALE

The FDA nominated cell phone RFR emissions of wireless communication devices for toxicology and carcinogenicity testing. Current exposure guidelines are based on protection from acute injury from thermal effects and little is known about the potential for health effects from long-term exposure to RFR below the thermal hazard threshold. Epidemiology studies that have been conducted to date have not demonstrated a causal link between cell phone RFR and any health problems in humans, however the results of these studies are complicated by confounding factors and potential biases. Additionally, exposures in the general population may not have occurred for a long enough period to account for the long latency period of some types of cancers in humans. Similar to the challenges faced in epidemiological studies, studies in laboratory animals have been complicated by limitations that researchers have faced in conducting robust studies designed to characterize the toxicity and carcinogenicity of cell phone RFR.

For years, the primary concern regarding the potential health risk of chronic exposure to cell phone RFR was brain cancer based on the proximity of wireless devices near the head during use. While the brain is an organ of concern, understanding the potential toxicity and carcinogenicity of whole-body exposure is critical. Cell phone RFR is

constantly emitted from wireless devices to communicate with base stations, regardless of whether the user is on a call or not. As the public has become more aware of the uncertainty regarding the potential effects of cell phone RFR on the brain, more emphasis has been placed on the use of wired or wireless headsets (like Bluetooth), which minimize cell phone RFR exposure to the head. In recent years, the density of cell towers has increased to cope with the increasing demand for capacity, resulting in installations closer to residential neighborhoods and schools. Additional cell phone RFR technologies, like SmartMeters used by power companies, transmit data in real time using cell phone-type RFR. These existing and emerging technologies may potentially increase the levels of exposures in human populations. These and other additional sources also expose different parts of the body, not only the head.

In 2013, cell phone RFR was classified by the IARC as possibly carcinogenic to humans based on limited evidence of an association between exposure to cell phone RFR from heavy wireless phone use and glioma and vestibular schwannoma (acoustic neuroma) in human epidemiology studies and limited evidence for the carcinogenicity of cell phone RFR in experimental animals (IARC, 2013). While ionizing radiation is a well-accepted human carcinogen, theoretical arguments have been raised against the possibility that nonionizing radiation could induce tumors (discussed in IARC, 2013). Given the extremely large number of people who use wireless communication devices, even a very small increase in the incidence of disease resulting from exposure to cell phone RFR generated by those devices would translate to a large number of affected individuals, which would have broad implications for public health.

MATERIALS AND METHODS

OVERVIEW

The establishment of the National Toxicology Program (NTP) research program on radio frequency radiation (RFR) has required the coordination of expertise from multiple scientific and engineering disciplines. At the initiation of the RFR research program, a collaboration was established with technical experts from the Radio-Frequency Fields Group in the Radio Frequency (RF) Technology Division, which is part of the Communications Technology Laboratory (CTL) at the National Institute of Standards and Technology (NIST, Boulder, CO). NIST evaluated the existing exposure systems and identified the types of improvements that would be required to provide a system of sufficient size and power to conduct robust toxicology and carcinogenicity studies with uniform RFR exposures in unrestrained, individually housed animals for a minimum of 6 hours a day at frequencies and modulations that reflected those in use at the time. The design of the chambers and toxicology studies required special consideration of logistical, financial, and engineering limitations.

NIST tested the feasibility of a reverberation chamber-type exposure system by conducting a series of studies on field strengths, field uniformity, and power requirements under various conditions of RFR exposure in such chambers. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements.

NTP also worked with the Foundation for Research on Information Technologies in Society (IT'IS, Zurich, Switzerland), which conducted studies using computational models that simulated RFR dosimetry to provide estimates of whole-body and organ-specific internal field strengths and specific absorption rates (SARs) during exposure. Based on information and parameters obtained during the NIST feasibility studies, IT'IS built a prototype reverberation chamber as the basis for an exposure system to study health effects of long-term exposure of

laboratory animals. Following completion, NIST evaluated the prototype exposure chamber to determine if it met the requirements specified by the NTP.

After prototype-testing by IT'IS Foundation and NIST, the IT'IS Foundation built the reverberation chambers required for the NTP RFR exposure facility. Chambers were installed at the Illinois Institute of Technology (IIT) Research Institute (IITRI, Chicago, IL). Following the installation and initial testing of the exposure system by IT'IS and IITRI, technical experts from NIST conducted an independent validation of the system. NIST confirmed that the probe readings in the system were consistent, that field uniformity was within expected specifications, and that the signal quality was acceptable. NIST performed additional evaluations prior to initiation of the 2-year studies and after completion of the studies to determine if any changes occurred in the signal quality, field uniformity, or consistency of in-chamber field measurements. All studies were conducted at IITRI with real-time monitoring of the system performance at IT'IS Foundation.

Institution	Role
National Institute of Standards and Technology (NIST) (Boulder, CO)	Suggested reverberation chamber exposure system Conducted feasibility studies for reverberation chambers Established various technical parameters for chambers Evaluated the prototype chamber built by IT'IS Foundation Validated the system prior to the conduct of studies at IITRI Reevaluated RFR exposures prior to and after 2-year studies
IT'IS Foundation (Zurich, Switzerland)	Constructed and tested prototype chamber Refined technical parameters Built the chambers for the NTP exposure facility Installed chambers at IITRI Monitored system performance throughout all phases of the studies Conducted maintenance on exposure system hardware and software
IIT Research Institute (IITRI) (Chicago, IL)	Tested exposure system after installation Conducted maintenance of exposure system hardware Conducted all toxicology and carcinogenicity studies Conducted day-to-day operations

REVERBERATION CHAMBER METHOD OF EXPOSURE

The use of the reverberation exposure chamber as a method for exposing rats and mice to cell phone RFR was conceptualized by NIST and further designed and tested by NIST and the IT'IS Foundation. A reverberation chamber is a resonant box where the resonances and field structure are continuously modified under the influence of metallic stirrers, introduced to change the effective geometry, such that when averaged over time, the field strength is uniform over the entire exposure volume. A reverberation chamber exposure system was selected by the NTP for the primary benefit that controlled exposures can be achieved in unrestrained animals (rats and mice) with extended daily RFR exposure periods compared to other methods of exposure for up to 2 years.

Preliminary studies were first conducted at NIST to test the concept of reverberation chambers. In these studies, field strengths and field uniformity were measured under various conditions of cell phone RFR exposure, including an empty chamber and a chamber loaded with water bottles (simulating animals) at different locations in the chamber. Power requirements were evaluated to achieve desired SAR levels. The effects of proximity between water bottles were also investigated to avoid electromagnetic coupling. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements. The results of these investigations demonstrated that while variations occurred over time and space the average cell phone RFR field was uniform over the large volume of the chamber. These studies also demonstrated that cell phone RFR field exposure occurred from all directions and all polarizations, and that there was uniformity of SAR in reverberation chambers. Based on the information and parameters obtained during the NIST feasibility studies, a custom-built prototype reverberation chamber was constructed and tested by the IT'IS Foundation. The development of the prototype chamber involved the design of amplifiers and antennas for signal generation, the design of vertical and horizontal stirrers to improve the homogeneity of experimentally generated RF fields, the development of both hardware and software for the control and monitoring of experimentally generated RF signals, and testing of chamber performance. During the design of the prototype exposure chamber, engineering studies were performed to optimize the following prior to construction:

- The uniform field volume within each chamber to minimize spatial variability in the characteristics of generated RF fields within a chamber such that all animals housed within the chamber space were exposed to comparable RF field strengths
- The design and placement of stirrers in each chamber in order to maximize homogeneity of experimentally-generated RF fields
- The design and location of RF antennas in each chamber
- The location of cage racks within the exposure chamber in order to provide appropriate separation of individual animal cages and cage racks from all reflective surfaces (chamber walls, chamber floor and ceiling, antennas, and stirrers) in the reverberation chamber
- Chamber volume to provide adequate space for staff to observe animals, collect data, and perform routine animal husbandry operations, while minimizing overall chamber volume to minimize the chamber size/footprint and the RF power required to maintain target SARs

The final reverberation chamber design for use in these studies was a fully-shielded room constructed of stainless steel, equipped with a shielded room door to eliminate leakage of RFR signals, two rotating stirrers (one horizontal and one vertical), ventilation structures, and RFR excitation antennas. A detailed rationale for the selection of reverberation chambers for exposure to RFR and a full description of the exposure system are provided in Capstick *et al.* (2017) and Gong *et al.* (2017).

As part of the validation of the reverberation chamber exposure system design, a team of engineers from NIST conducted an independent evaluation of chamber design and exposure system operation in order to evaluate the suitability of the reverberation chamber model for use in the program. NIST engineers evaluated the design and operation of the prototype chamber and performed an extensive series of RF measurements to support an evaluation of system performance.

CELL PHONE RFR EXPOSURE FACILITY

The exposure facility was specifically designed to expose mice in reverberation chambers to three different power levels of modulated cell phone RFR [Global System for Mobile Communications (GSM) or Code Division Multiple

Access (CDMA)] at 1,900 MHz for up to 2 years to evaluate toxicity and carcinogenicity. The completed exposure facility consisted of a total of 21 RFR reverberation exposure chambers (seven designated for mice); the RFR signal generation, amplification, and monitoring systems; software for chamber operation; and hardware and software for monitoring of environmental and exposure conditions within each chamber. All system hardware and software were installed by the IT'IS Foundation.

During exposures, modulated (GSM or CDMA) cell phone RFR signals were generated by a signal generator, amplifiers amplified the signals, and the signals were delivered by antennas in the reverberation chambers. RFR field strengths were monitored in real time and were adjusted throughout the studies to achieve specific exposure levels [based on SARs quantitated in watts (W) per kg body weight]. Environmental conditions were also monitored and controlled in real time throughout the study. RFR exposures and environmental conditions were monitored and controlled by a computer in a control room at the study laboratory at IITRI; the IT'IS Foundation was also capable of remote system monitoring and control.

Facility Design and Reverberation Chambers

Each reverberation chamber was permanently programmed for a specified modulation (GSM or CDMA) of the 1,900 MHz cell phone RFR specified for the mouse studies. SARs for each chamber were adjustable and selected prior to exposures. The field strength required to achieve a given target SAR (W/kg) exposure level is a function of animal body weight (kg); however, separate chambers were not required for male and female B6C3F1/N mice because their body weights and growth curves are sufficiently similar to yield similar SARs. To conduct robust toxicology studies with three exposure groups (low, medium, and high), three chambers were required for different levels of exposures for GSM modulation and three for CDMA modulation. A sham exposure chamber without any cell phone RFR signal provided shared control groups for the parallel studies of the two modulations. As per these requirements, the cell phone RFR exposure facility consisted of seven reverberation chambers for exposures in mice including:

- Three power levels for mice exposed to GSM-modulated cell phone RFR at 1,900 MHz
- Three power levels for mice exposed to CDMA-modulated cell phone RFR at 1,900 MHz
- One sham control chamber for mice with no RFR exposure.

The chamber size was designed to accommodate the RF field stirring paddles (described below), approximately 220 individually housed mice, and a minimum distance (3/4 of a wavelength) between the cages and the walls, floor, ceiling and stirrers, respectively. The interior of the chamber was suitable for cleaning using high-pressure water (after the RF antennas were protected). The internal dimensions of the chambers were 2.2 m (width) × 3.7 m (length) × 2.6 m (height); the exterior dimensions were 2.3 m (width) × 3.8 m (length) × 2.85 m (height). A floorplan for the exposure facility and images of the interior and exterior of the chambers are presented in Figures 2 and 3.

Each chamber contained two motor-controlled stirring paddles (one vertical and one horizontal) with adjustable speed control (1 to 50 rpm) and large asymmetrical reflecting surfaces. Stirring paddles were placed off center in the chamber for maximum scattering of the RFR fields to generate a statistically homogeneous field distribution when averaged over time. The horizontal stirrer was mounted on the ceiling of the chamber. The vertical stirrer was at the rear of the chamber, and was protected by rack guides that prevented contact with the animal cage racks.

Cage Racks and Watering System

Cages, cage racks, and watering systems for standard laboratory use contain elements that have the ability to alter the exposure of the animals or introduce potential confounding factors. Because cage racks and the drinking water delivery system were contained inside the chambers during exposure periods, it was required that these components be constructed of durable materials that had essentially no impact on the RF fields generated in the chamber. Metallic cage rack components, cage lids, feed dispensers, and cage grommets all needed to be eliminated. Hence, custom engineering was required to overcome the challenges regarding potential RFR exposure-altering aspects of the caging and cage racks used to house the animals during the studies. The safe provision of drinking water provided the largest challenge for the studies.

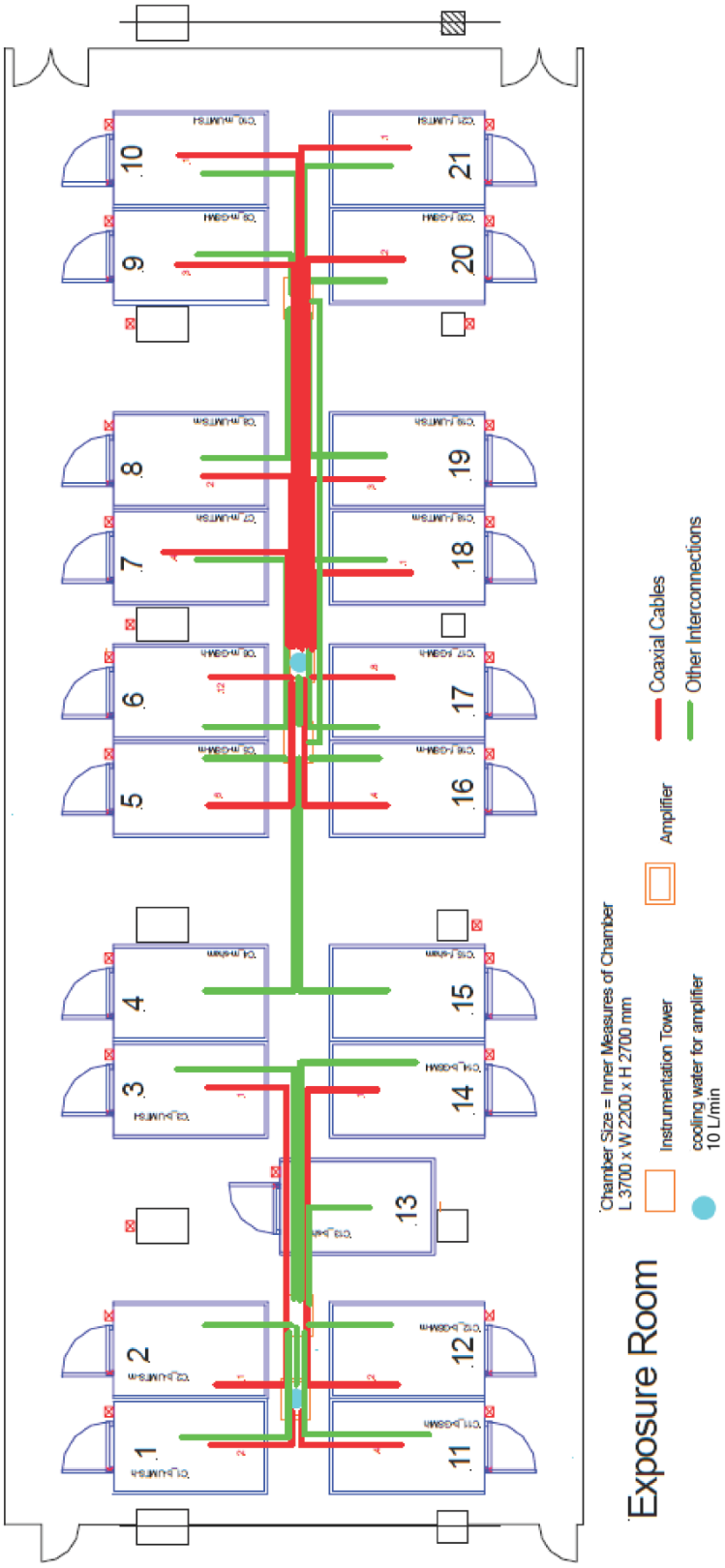


FIGURE 2
Exposure Facility Floor Plan for the Cell Phone RFR Studies (Not shown are the Ethernet connections to computers in the control room.) Mouse chamber designations: low GSM=14; medium GSM=12; high GSM=11; low CDMA=3; medium CDMA=2; high CDMA=1; sham control=13. The 14 other chambers (including 12 for cell phone RFR exposure and two for sham control) were designated for concurrent rat studies.

FIGURE 3
Exterior view of chambers, empty chamber showing the vertical and horizontal stirrers, and chamber with cage racks in place

The absorption of RFR energy by water, if supplied by nonmetallic sipper tubes and distribution systems or bottles, could lead to dose-dependent elevated water temperatures. As the same time, the potential for enhanced exposure fields by metallic sipper tubes or lixits precluded the use of water bottles or a standard automatic watering system in the reverberation chambers. The absorption of RFR energy by water could result in significant heating of the drinking water, thereby decreasing water palatability and increasing the required RFR power to achieve the desired exposure field strength, potentially to the extent that the exposure levels could not be met. To overcome these challenges, adaptations were made to an automatic watering system so that the delivery of drinking water to the animals would not interfere with cell phone RFR dosimetry. The water system was constructed from stainless steel ensuring no dose-dependent energy absorption in the water (avoiding exposure-dependent water temperature) and in structures around the lixits to ensure no enhanced fields that could lead to excessive SAR in the animals while drinking.

Customized, nonmetallic animal cage racks for the reverberation chambers were designed by IITRI to minimize any absorption of RFR or disruption of RF field homogeneity. Cage racks were constructed primarily of box beam fiberglass (with some angle beam fiberglass used in nonweight-bearing areas of the rack). The shelves/cage lids were constructed of a clear polycarbonate sheet with slots for increased airflow. The potential impact of the racks on RF fields was evaluated in the prototype reverberation chamber by the IT'IS Foundation. Cage racks were designed to accommodate the automatic watering system and position the perimeter of each animal cage at least one-half wavelength from any reflecting surface. The specific considerations for design and further details of the custom-designed cage racks and adapted automated watering system are provided in Capstick *et al.* (2017).

Cell Phone RFR Exposure System Control

The hardware and chambers designated for mice (using an exposure frequency of 1,900 MHz) were connected to a dedicated computer control system using an Ethernet protocol. The computerized control system managed and monitored the cell phone RFR exposures and environmental conditions in the chambers. A more detailed description of the computer control of cell phone RFR exposure is provided in Capstick *et al.* (2017).

The control computer managed the exposure schedule, stirrer rotation speeds, exposure signal and level, and monitored air flow, temperature, humidity, light, and the electric and magnetic fields (E- and H-fields, respectively) in each chamber. The hardware for the exposure system consisted of the control computer and a rack containing communications interfaces and instrumentation for signal generation, data acquisition, signal monitoring, signal amplifiers, and the chamber hardware (which included the stirrer motors and environmental and RFR sensors). The instrumentation rack contained the equipment that generated the cell phone RFR signal, acquired cell phone RFR field strengths and environmental data, and provided an interface between the components and the control computer.

The mouse system hardware included an Ethernet to general purpose interface bus, a cell phone RFR signal generator, three data acquisition units, four RF field measurement units, a power supply unit, and an Ethernet hub. The amplifier array housed signal amplifiers, an amplifier cooling system, and two real-time digital control units that directly controlled the six amplifiers in the mouse system. Each amplifier produced 400 W peak power and in excess of 200 W average power. A closed-circuit cooling system ran cool water through the amplifiers to keep them from overheating. The real-time digital control units controlled which chamber the amplifier output was routed to and the level of amplifier output power while it was routed to that particular chamber.

CELL PHONE RFR SIGNAL GENERATION

GSM-modulated and CDMA-modulated cell phone RFR signals were generated experimentally via a SMIQ02B vector signal generator with options SMIQB11 and SMIQB20 and software options 100421 – 100423 (Rohde and Schwarz, Munich, Germany). Signals were amplified using six LSE™ amplifiers (LSE, Spanga, Sweden) in the exposure system. The outputs of each individual amplifier were set by real-time controllers on a slot-by-slot basis for GSM or CDMA modulation to control the E-field strength in each chamber. Each chamber contained at least one standard gain antenna (two half-wave dipoles) that was mounted a quarter of a wavelength in front of a reflector plate. Antennas were directed towards one of the two stirrers to maximize scattering and obtain acceptable E-field homogeneity within the chamber space. The computerized control system managed the exposure schedule, stirrer rotation speeds, and exposure signal type and level.

The RFR power introduced into a given chamber was adjusted to achieve target field strengths; to maintain constant exposure levels (W/kg) in a given chamber, the field strengths [measured in volts (V) per meter] were regularly adjusted to reflect changes in the average mass of the exposed animals. The relationship between animal mass, field strength, and SAR was determined from numerical dosimetry and programmed into the control software, hence the required exposure field strength was computed from the average animal weights entered for each exposure group. The interval at which animal weights were updated was determined on how rapidly the animals were growing, at the start of the exposure period this was once per week, and as long as up to every 4 weeks later in the study.

VERIFICATION OF CELL PHONE RFR EXPOSURE

Prior to initiation of the animal studies, the RF Fields Group in the Communications Technology Laboratory at the NIST performed an independent, detailed evaluation of 18 of the reverberation chambers (excluding the three sham control chambers; Figure 2) to verify the cell phone RFR exposure fields, chamber characteristics (field uniformity), and signal quality to determine the accuracy of field values reported by the developers of the exposure system (IT'IS Foundation). Full reports detailing the procedures for measurements and calculations are available from the NTP.

All E-field measurements agreed within the estimated uncertainty bounds, indicating that the chamber fields measured by the NIST agreed with the measurements provided by the IT'IS Foundation probes. During validation, it was determined that the H-field probes at higher signal levels in the mid- and high-power GSM chambers reported higher fields than indicated by other measurements, potentially leading to a modest overestimation of chamber field strengths. In these chambers, H-field probes were replaced with E-field probes, which provided more accurate measurements of the RF fields. The magnitude of field variation throughout the volume of a fully loaded chamber was consistent with earlier values reported for the prototype chamber. However, it was determined that there may have been up to ± 2.5 dB of variation in the exposure field depending on location in the cage racks. To mitigate this positional variation, cages were routinely rotated to various locations within and between the cage racks. The quality of the modulated signals was found to be acceptable with regard to distortion and harmonic content.

Overall, the NIST confirmed that the cell phone RFR reverberation chamber exposure system was operating correctly and cell phone RFR exposures were within specifications.

CELL PHONE RFR EXPOSURE MONITORING

During all exposure periods, experimentally generated cell phone RFR was continuously monitored by the control system via two RF sensors (E-field and/or H-field probes) in each exposure chamber that measured real-time signal strengths. The use of two probes provided two independent measurements of RF field strengths and ensured that appropriate quantitation of experimentally generated RF fields continued even in the unlikely event that one probe failed. The E-field sensor measured electric field strength (V/m). The H-field sensor measured magnetic field strength [measured in amperes (A) per meter]. All chambers were instrumented with one E-field sensor (ER3DV6) and one H-field sensor (H3DV6) [both from Schmid and Partner Engineering AG (SPEAG), Zurich, Switzerland], except for the medium and high power GSM chambers. These chambers were instrumented with two E-field probes because H-field probes saturated at high field strengths. This change in hardware did not result in the loss of monitoring capability. The measured E- and H-fields were communicated to the control computer in order to maintain exposure to selected levels of RFR. During daily shutdown periods when RFR exposures were not active, RF sensors monitored ambient RF fields in the exposure chambers. RF sensors were calibrated twice by the manufacturer (SPEAG); once prior to initiation of any of the animal studies and once prior to initiation of the 2-year studies. All E-field probes were calibrated in air from 100 MHz to 3.0 GHz, and had an absolute accuracy of ± 6.0% (k=2) with a spherical isotropy of better than ± 0.4 dB. All H-field probes were calibrated in air from 200 MHz to 3.0 GHz and had an absolute accuracy of ± 6.0% (k=2) with a spherical isotropy of better than ± 0.2 dB.

Data collected by the RF sensors were transmitted to the exposure and monitoring system on a real-time basis and were recorded throughout the study. Chamber field strengths are reported as V/m and animal exposure levels (SAR values) are reported as W/kg. The chamber field strength is the average effective E-field strength from both probes. E-field and H-field strengths are related by the impedance of free space which is ~377 Ohms. Where an H-field probe was used, the value in A/m was multiplied by 377 to calculate the equivalent E-field strength in V/m; it is this effective E-field value that was used to report the chamber field strength. Field strength data reported for each day of exposure included mean ± standard deviation, minimum field strength, maximum field strength, total number of readings in range/total number of readings for the period, and percentage of readings in range. After each exposure day, cell phone RFR exposure data were downloaded onto DVDs for long-term archival. Summaries of the 2-year cell phone RFR exposure data from the studies are presented in Appendix I. The SAR and chamber-fields in the

exposure chambers were within the target ranges (defined as ± 2 dB) for >99.85% of recorded measurements over the course of the 2-year study; ≥99.70% of recorded E-field and H-field measurements were within the target ranges for all but one chamber (97.35% within range). All recorded SAR and field measurements were within the target ranges for the sham control chamber. In the 28-day studies, the performance of the sham control and exposure chambers was similar for SAR and field measurements as in the 2-year studies (data not shown).

As previously stated, the performance of the cell phone RFR exposure and monitoring system was independently validated by engineers from the NIST prior to the initiation of the animal studies.

MONITORING AND MAINTENANCE OF ENVIRONMENTAL CONDITIONS

Environmental conditions including temperature, humidity, and airflow in all exposure chambers, as well as in other areas of the IITRI cell phone RFR exposure facility, were maintained by a computer-controlled environmental management system (Siemens Industries, Inc.). Monitoring instrumentation for each chamber was located in the air exhaust duct. Each chamber was fitted by the IT'IS Foundation with a sensor box that contained sensors for temperature and humidity (Type EE06; E + E Elektronik GmbH, Engerwitzdorf, Austria), oxygen level (Pewatron Type FCX-MC25; Zurich, Switzerland), air speed (model EE65A; E + E Elektronik GmbH), light (light-dependent resistor), noise (design based on WL-93 microphone; Shure Brothers, Inc., Evanston, IL), and RFR. Outputs from the sensor box were monitored using Agilent data acquisition units, with the exception of the RF sensor. The RF sensor was directly wired to a warning light as a safety precaution to indicate active RFR exposures and not intended to quantitatively measure RFR field strengths.

Exposure chambers were equipped with incandescent lights located on light bars in each corner of the chamber. All connections were RF-filtered. Chamber lighting was controlled using an adjustable daily cycle of 12 hours on, 12 hours off. In order to minimize the heat load generated by the incandescent lights, low wattage bulbs were used that maintained chamber lighting within a range that was sufficient to support normal *in vivo* operations, while minimally affecting chamber temperature. Given the expected effect of RFR field exposures on chamber temperature during the toxicology studies, it was considered prudent to attempt to minimize heat load generated by local sources in each chamber.

Differences in noise levels in the exposure chambers resulting from the heating, ventilation, and air conditioning system were equalized by the installation of sound baffles in various ducts within the system. An audible signal generated by the high intensity GSM signal was detected and equalized in all chambers by the introduction of a "pink noise" masking sound; this masking noise equalized sound levels in all chambers. As a result of the combination of these efforts, noise levels in all chambers were essentially equivalent and met the NC-35 noise specification. [The noise criterion (NC) is a widely accepted numerical index commonly used to define the maximum allowable noise. It primarily applies to the noise produced by ventilation systems, but is applied to other noise sources, as well. Standards organizations, such as the American National Standards Institute (ANSI), Acoustical Society of America (ASA), and International Standards Organization, provide definitions of various NCs for ambient noise in enclosed spaces. The ANSI/ASA standard (S12.2-2008) recommends NCs for various types of rooms, including private residences (NC 25-40), schools (NC 25-35), offices (NC 25-40), libraries (NC 30-35), and restaurants (NC 40-45)].

ANIMAL SOURCE

Male and female B6C3F1/N mice were obtained from the NTP colony maintained at Taconic Farms, Inc. (Germantown, NY), for the 28-day and 2-year studies.

ANIMAL WELFARE

Animal care and use are in accordance with the Public Health Service Policy on Humane Care and Use of Animals. All animal studies were conducted in an animal facility accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International. Studies were approved by the IITRI Animal Care and Use Committee and conducted in accordance with all relevant NIH and NTP animal care and use policies and applicable federal, state, and local regulations and guidelines.

28-DAY STUDIES

The 28-day studies were conducted to evaluate the cumulative effects of repeated GSM- or CDMA-modulated cell phone RFR exposure and to determine the appropriate cell phone RFR power levels to be used in the 2-year studies.

Groups of 10 male and 10 female core study mice and groups of 20 male and 20 female special study mice were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 5, 10, or 15 W/kg, for 9 hours and 10 minutes per day for 5 or 7 (last week of study) days per week for at least 28 days with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in a reverberation chamber identical to those used for the exposed groups, but were not exposed to cell phone RFR; a shared group of unexposed mice of each sex served as sham controls for both cell phone RFR modulations.

Animals were observed twice daily and were weighed once during quarantine, initially, and weekly thereafter. Clinical signs were recorded once during quarantine and then weekly. In core study mice, subcutaneously implanted temperature microchips and monitoring equipment (Bio Medic Data Systems, Seaford, DE) were used to monitor individual animal body temperatures. Body temperature measurements were taken prior to initial exposure at the beginning of the study, on days 7 and 14 during inactive shutdown periods with no exposure, and on days 2, 4, 17, 20, and 27 within 5 minutes of exposure pauses at the end of the second to the last "on" cycle at the same time each day.

Mice were quarantined for 9 or 3 days (first and second shipment, respectively) before the beginning of the studies. Ten mice (two males and eight females) that were not assigned during randomization were selected for parasite evaluation and gross observation of disease. Mice were approximately 5 to 6 weeks old at the beginning of the studies. The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Mice were housed individually. Feed and water were available *ad libitum*. To avoid interference with cell phone RFR dosimetry, feed was provided in ceramic (nonmetallic) bowls and water was delivered in an adapted automatic watering system (Capstick *et al.*, 2017). Cages were changed weekly and rotated within the racks weekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Necropsies were performed on all core study mice on day 29 or 30. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus. Tissues for microscopic examination were fixed and preserved in 10% neutral buffered formalin (except eyes, testis with epididymis, and vaginal tunics were first fixed in Davidson's solution or modified Davidson's solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 µm, and stained with hematoxylin and eosin. Complete histopathologic examinations were performed by the study laboratory pathologist on all 0 (sham control) and 15 W/kg GSM- and 15 W/kg CDMA-modulated cell phone RFR core study mice. Table 1 lists the tissues and organs routinely examined.

After a review of the laboratory reports and selected histopathology slides by a quality assessment (QA) pathologist, the findings and differences of opinions between the study pathologist (SP) and the QA pathologist were reviewed by the NTP pathologist. Slides containing representative lesions of exposure-related lesions or differences of opinions between pathologists were brought to a Pathology Peer Review (PPR). Final diagnoses for reviewed lesions represent a consensus of the PPR or a consensus between the study laboratory pathologist, NTP pathologist, and the QA pathologist(s). Details of these review procedures have been described, in part, by Maronpot and Boorman (1982) and Boorman *et al.* (1985).

2-YEAR STUDIES

Study Design

Groups of 105 male and 105 female mice were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 2.5, 5, or 10 W/kg, 9 hours and 10 minutes per day, 7 days per week for 106 (males) or 108 (females) weeks with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but were not exposed to cell phone RFR; shared groups of unexposed mice of each sex served as sham controls for both cell phone RFR modulations. Fifteen mice per group were randomly selected from the core group after 10 weeks of study; ten mice per group were randomly selected for interim evaluation at 14 weeks, and five mice per group were used for genetic toxicity testing at 14 weeks.

Mice were quarantined for 9 days before the beginning of the studies. An additional five male and five female mice not assigned during randomization were selected for parasite evaluation and gross observation of disease. Mice were approximately 5 to 6 weeks old at the beginning of the studies. The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Mice were housed individually. Feed and water were available *ad libitum*. To avoid interference with cell phone RFR dosimetry, feed was provided in ceramic (nonmetallic) bowls and water was delivered in an adapted automatic watering system (Capstick *et al.*, 2017). Cages were changed weekly and rotated within the racks biweekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Clinical Examinations and Pathology

Animals were observed twice daily and were weighed initially, weekly for the first 14 weeks, at 4-week intervals during weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical signs were recorded once during quarantine and at least every 4 weeks during the studies.

Hematology evaluations were performed on 10 male and 10 female interim evaluation mice from each group at 14 weeks. Mice were anesthetized with 70% CO_2/30% O_2 and blood was collected from the retroorbital sinus and placed into tubes containing EDTA as an anticoagulant. Hematology parameters were determined on an ADVIA™ 120 automated hematology analyzer (Bayer Diagnostic Division, Tarrytown, NY). The parameters measured are listed in Table 1. Wright Giemsa stained peripheral blood smears were prepared and evaluated for any blood cell abnormalities. Blood was collected from the remaining five male and five female interim evaluation mice per exposure group at 14 weeks for use in the comet and micronucleus assays; methods for these assays are presented in Appendix E.

At 14 weeks, samples were collected for sperm motility and count and vaginal cytology evaluations on 10 male and 10 female interim evaluation mice from each group. The parameters evaluated are listed in Table 1. For 15 or

16 consecutive days prior to scheduled euthanasia, the vaginal vaults of the females were moistened with saline, if necessary, and samples of vaginal fluid and cells were stained. Relative numbers of leukocytes, nucleated epithelial cells, and large squamous epithelial cells were determined and used to ascertain estrous cycle stage (i.e., diestrus, proestrus, estrus, and metestrus). Male animals were evaluated for sperm count and motility. The left testis and left epididymis were isolated and weighed. The tail of the epididymis (cauda epididymis) was then removed from the epididymal body (corpus epididymis) and weighed. Modified Tyrode's buffer was applied to slides and a small incision was made at the distal border of the cauda epididymis. The sperm effluxing from the incision were dispersed in the buffer on the slides, and the numbers of motile and nonmotile spermatozoa were counted for five fields per slide by two observers. Following completion of sperm motility estimates, each left cauda epididymis was placed in buffered saline solution. Caudae were finely minced, and the tissue was incubated in the saline solution and then heat fixed at 65° C. Sperm density was then determined microscopically with the aid of a hemacytometer. To quantify spermatogenesis, the testicular spermatid head count was determined by removing the tunica albuginea and homogenizing the left testis in phosphate-buffered saline containing 10% dimethyl sulfoxide. Homogenization-resistant spermatid nuclei were counted with a hemacytometer.

All mice were necropsied. The cerebrum, frontal cortex, hippocampus, and liver were collected from five male and five female interim sacrifice animals per exposure group at 14 weeks for use in the comet assay; methods for this assay are presented in Appendix E. Microscopic examinations were performed on 10 male and 10 female interim evaluation mice in each group at 14 weeks and all core study mice, including those found dead or euthanized moribund. At the interim evaluation, the brain, right and left epididymides, heart, right and left kidneys, liver, lung, right and left ovaries, right and left testes, and thymus were weighed. At necropsy, all organs and tissues were examined for grossly visible lesions, and all major tissues were fixed and preserved in 10% neutral buffered formalin (except eyes were first fixed in Davidson's solution, and testes, vaginal tunics, and epididymides were first fixed in modified Davidson's solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 µm, and stained with hematoxylin and eosin for microscopic examination. For all paired organs (e.g., adrenal gland, kidney, ovary), samples from each organ were examined. Tissues examined microscopically are listed in Table 1.

Microscopic evaluations were completed by the study laboratory pathologist, and the pathology data were entered into the Toxicology Data Management System. The report, slides, paraffin blocks, residual wet tissues, and pathology data were sent to the NTP Archives for inventory, slide/block match, wet tissue audit, and storage. The slides, individual animal data records, and pathology tables were evaluated by an independent quality assurance (QA) laboratory. The individual animal records and tables were compared for accuracy, the slide and tissue counts were verified, and the histotechnique was evaluated. For the 2-year studies, a QA pathologist evaluated slides from all tumors and all potential target organs, which included the brain, spinal cord, heart, and kidney. In addition, the liver, large intestine (cecum and colon), small intestine (duodenum, jejunum, and ileum), lung, testis, urinary bladder, and Harderian gland were reviewed from all male mice for specific lesions; and the bronchial and mesenteric lymph nodes, spleen, ovary, urinary bladder, Harderian gland, and thyroid gland were reviewed from all female mice for specific lesions.

The QA report and the reviewed slides were submitted to the NTP pathologist, who reviewed and addressed any inconsistencies in the diagnoses made by the laboratory and QA pathologists. The QA pathologist, who served as the coordinator of the Pathology Working Group (PWG) presented representative histopathology slides containing examples of lesions related to test agent administration, examples of disagreements in diagnoses between the laboratory and QA pathologists, or lesions of general interest to the PWG for review. The PWG consisted of the NTP pathologist and other pathologists experienced in rodent toxicologic pathology. This group examined the tissues without any knowledge of exposure groups. When the PWG consensus differed from the opinion of the laboratory pathologist, the diagnosis was changed. Final diagnoses for reviewed lesions represent a consensus between the laboratory pathologist, QA pathologist(s), and the PWG. Details of these review procedures have been described, in part, by Maronpot and Boorman (1982) and Boorman *et al.* (1985). For subsequent analyses of the pathology data, the decision of whether to evaluate the diagnosed lesions for each tissue type separately or combined was generally based on the guidelines of Brix *et al.* (2010).

TABLE 1
Experimental Design and Materials and Methods in the Whole-Body Exposure Studies of GSM- and CDMA-Modulated Cell Phone RFR

28-Day Studies	2-Year Studies
Study Laboratory IIT Research Institute (Chicago, IL)	IIT Research Institute (Chicago, IL)
Strain and Species B6C3F1/N mice	B6C3F1/N mice
Animal Source Taconic Farms, Inc. (Germantown, NY)	Taconic Farms, Inc. (Germantown, NY)
Time Held Before Studies 9 and 3 days (first and second shipment, respectively)	9 days
Average Age When Studies Began Approximately 5 to 6 weeks	5 to 6 weeks
Date of First Exposure September 6, 2010	June 18, 2012
Duration of Exposure 9 hours and 10 minutes per day over an 18 hour and 20 minute period as exposures cycled between modulations every 10 minutes, 5 or 7 (last week of study) days per week for at least 28 days.	9 hours and 10 minutes per day over and 18 hour and 20 minute period as exposures cycled between modulations every 10 minutes, 7 days per week for 14 weeks (interim evaluation) or 106 (males) or 108 (females) weeks (2-year studies).
Date of Last Exposure October 3 or 4, 2010	Males: June 26, 2014 Females: July 9, 2014
Necropsy Dates October 4 or 5, 2010	Males: June 16 to 26, 2014 Females: June 26 to July 9, 2014
Age at Necropsy Approximately 9 to 10 weeks	Males: 110 to 112 weeks Females: 111 to 114 weeks
Size of Study Groups 10 males and 10 females	Core study: 90 males and 90 females Interim evaluation: 10 male and 10 females Genetic toxicity: Five male and five females
Method of Distribution Animals were distributed randomly into groups of approximately equal initial mean body weights.	Animals were distributed randomly into groups of approximately equal initial mean body weights.
Animals per Cage 1	1
Method of Animal Identification Tail tattoo	Tail tattoo
Diet Certified, irradiated NTP-2000 rodent diet wafer (Zeigler Brothers, Inc., Gardners, PA), available *ad libitum*, ceramic feed bowls changed weekly	Same as 28-day studies

TABLE 1
Experimental Design and Materials and Methods in the Whole-Body Exposure Studies of GSM- and CDMA-Modulated Cell Phone RFR

28-Day Studies	2-Year Studies
Water Tap water (Chicago municipal supply) via an adapted automatic watering system (SE Lab Group, Cincinnati, OH), available *ad libitum*	Same as 28-day studies
Cages Polycarbonate, solid bottom "shoebox" cages (Allentown Caging, Allentown, NJ), changed and rotated within the rack weekly	Same as 28-day studies
Bedding Certified, irradiated hardwood bedding (P.J. Murphy Forest Products Corp., Montville, NJ), changed weekly	Same as 28-day studies
Racks Custom-designed fiberglass cage racks (Ultra, Inc., Milwaukee, WI), changed biweekly	Same as 28-day studies
Reverberation Chambers Fully-shielded, stainless steel room equipped with a stainless steel door to eliminate leakage of cell phone RFR signals, cell phone RFR excitation antennas, and two rotating stirrers; chambers were cleaned at least once weekly.	Same as 28-day studies
Reverberation Chamber Environment Temperature: 72° ± 3° F Relative humidity: 50% ± 15% Room incandescent light: 12 hours/day Chamber air changes: at least 10/hour	Temperature: 72° ± 3° F Relative humidity: 50% ± 15% Room incandescent light: 12 hours/day Chamber air changes: at least 10/hour
Exposure Concentrations Time-averaged whole-body SARs of 0 (sham control), 5, 10, and 15 W/kg GSM- or CDMA-modulated cell phone RFR	Time-averaged whole-body SARs of 0 (sham control), 2.5, 5, and 10 W/kg GSM- or CDMA-modulated cell phone RFR
Type and Frequency of Observation Observed twice daily; animals were weighed once during quarantine, initially, and weekly thereafter. Clinical signs were recorded once during quarantine and then weekly. Body temperature measurements were taken on core study mice prior to initial exposure at the beginning of the study, on days 7 and 14 during inactive exposures, and on days 2, 4, 17, 20, and 27 within 5 minutes of exposure pauses at the end of the second to the last "on" cycle.	Observed twice daily; animals were weighed initially, weekly for the first 14 weeks, at 4-week intervals during weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical signs were recorded once during quarantine and at least once every 4 weeks during the studies.
Method of Euthanasia Carbon dioxide asphyxiation	Carbon dioxide asphyxiation
Necropsy Necropsies were performed on all core study mice on day 29 or 30. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus.	Necropsies were performed on all mice. Organs weighed in 10 mice per exposure group at 14 weeks were the brain, heart, kidneys (right and left), liver, lung, ovaries (right and left), testes (right and left) with epididymides (right and left), and thymus.

TABLE 1
Experimental Design and Materials and Methods in the Whole-Body Exposure Studies of GSM- and CDMA-Modulated Cell Phone RFR

28-Day Studies	2-Year Studies
Clinical Pathology	
None	Blood was collected from the retroorbital sinus of 10 mice per group at 14 weeks for hematology. ***Hematology:*** hematocrit (auto and manual); hemoglobin concentration; erythrocyte, reticulocyte, and platelet counts; erythrocyte, leukocyte, and platelet morphology; mean cell volume; mean cell hemoglobin; mean cell hemoglobin concentration; and leukocyte count and differentials
Histopathology	
Complete histopathology was performed on all 0 (sham control) and 15 W/kg groups. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, esophagus, eyes, gallbladder, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, larynx, liver, lung, lymph nodes (mandibular and mesenteric), mammary gland, muscle, nerve (sciatic), nose, oral cavity, ovary, pancreas, pharynx, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spinal cord, spleen, stomach (forestomach and glandular), testis with epididymis, thymus, thyroid gland, tongue, trachea, urinary bladder, uterus, vagina, and Zymbal's gland.	Complete histopathology was performed on 10 mice from each group at 14 weeks, on all mice that died early, and on all mice surviving to the end of the studies. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, esophagus, eyes, gallbladder, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, liver, lung with bronchi, lymph nodes (mandibular and mesenteric), mammary gland, muscle, nerve (sciatic, trigeminal, and ganglion), nose, ovary, pancreas, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spinal cord, spleen, stomach (forestomach and glandular), testis with epididymis, thymus, thyroid gland, trachea, urinary bladder, and uterus.
Sperm Motility and Count and Vaginal Cytology	
None	Spermatid and sperm samples were collected from 10 male mice in each group at 14 weeks. The following parameters were evaluated: spermatid heads per testis and per gram testis, sperm motility, and sperm per cauda epididymis and per gram cauda epididymis. The left cauda, left epididymis, and left testis were weighed. Vaginal samples were collected from 10 females in each group for 15 or 16 days prior to the 14-week interim evaluation.

STATISTICAL METHODS

For all analyses, P values less than 0.05 were considered statistically significant.

Survival Analyses

The probability of survival was estimated by the product-limit procedure of Kaplan and Meier (1958) and is presented in the form of graphs. Animals found dead of other than natural causes or missing were censored; animals dying from natural causes were not censored. Statistical analyses for possible dose-related effects on survival used Cox's (1972) method for testing two groups for equality and Tarone's (1975) life table test to identify dose-related trends. All reported P values for the survival analyses are two sided.

Calculation of Incidence

The incidences of neoplasms or nonneoplastic lesions are presented in Tables A1, A4, B1, B4, C1, C4, D1, and D4 as the numbers of animals bearing such lesions at a specific anatomic site and the numbers of animals with that site examined microscopically. For calculation of statistical significance, the incidences of most neoplasms (Tables A2, B2, C2, and D2) and all nonneoplastic lesions are given as the numbers of animals affected at each site examined microscopically. However, when macroscopic examination was required to detect neoplasms in certain tissues (e.g., mesentery, pleura, peripheral nerve, skeletal muscle, tongue, tooth, and Zymbal's gland) before microscopic evaluation, the denominators consist of the number of animals that had a gross abnormality. When neoplasms had multiple potential sites of occurrence (e.g., leukemia or lymphoma), the denominators consist of the number of animals on which a necropsy was performed. Tables A2, B2, C2, and D2 also give the survival-adjusted neoplasm rate for each group and each site-specific neoplasm. This survival-adjusted rate (based on the Poly-3 method described below) accounts for differential mortality by assigning a reduced risk of neoplasm, proportional to the third power of the fraction of time on study, only to site-specific, lesion-free animals that do not reach terminal euthanasia.

Analysis of Neoplasm and Nonneoplastic Lesion Incidences

The Poly-k test (Bailer and Portier, 1988; Portier and Bailer, 1989; Piegorsch and Bailer, 1997) was used to assess neoplasm and nonneoplastic lesion prevalence. This test is a survival-adjusted quantal-response procedure that modifies the Cochran-Armitage linear trend test to take survival differences into account. More specifically, this method modifies the denominator in the quantal estimate of lesion incidence to approximate more closely the total number of animal years at risk. For analysis of a given site, each animal is assigned a risk weight. This value is one if the animal had a lesion at that site or if it survived until terminal euthanasia; if the animal died prior to terminal euthanasia and did not have a lesion at that site, its risk weight is the fraction of the entire study time that it survived, raised to the kth power.

This method yields a lesion prevalence rate that depends only upon the choice of a shape parameter for a Weibull hazard function describing cumulative lesion incidence over time (Bailer and Portier, 1988). Unless otherwise specified, a value of k=3 was used in the analysis of site-specific lesions. This value was recommended by Bailer

and Portier (1988) following an evaluation of neoplasm onset time distributions for a variety of site-specific neoplasms in control F344 rats and $B6C3F_1$ mice (Portier *et al.*, 1986). Bailer and Portier (1988) showed that the Poly-3 test gave valid results if the true value of k was anywhere in the range from 1 to 5. A further advantage of the Poly-3 method is that it does not require lesion lethality assumptions. Variation introduced by the use of risk weights, which reflect differential mortality, was accommodated by adjusting the variance of the Poly-3 statistic as recommended by Bieler and Williams (1993).

Tests of significance included pairwise comparisons of each dosed group with controls and a test for an overall dose-related trend. Continuity-corrected Poly-3 tests were used in the analysis of lesion incidence, and reported P values are one sided. The significance of lower incidences or decreasing trends in lesions is represented as 1–P with the letter N added (e.g., P=0.99 is presented as P=0.01N). For neoplasms and nonneoplastic lesions detected at the interim evaluation, the Fisher exact test (Gart *et al.*, 1979), a procedure based on the overall proportion of affected animals, was used.

Analysis of Continuous Variables

Two approaches were employed to assess the significance of pairwise comparisons between dosed and control groups in the analysis of continuous variables. Organ and body weight data, which historically have approximately normal distributions and body temperatures, were analyzed with the parametric multiple comparison procedures of Dunnett (1955) and Williams (1971, 1972). Hematology, spermatid, and epididymal spermatozoal data, which have typically skewed distributions, were analyzed using the nonparametric multiple comparison methods of Shirley (1977) (as modified by Williams, 1986) and Dunn (1964). Jonckheere's test (Jonckheere, 1954) was used to assess the significance of the dose-related trends and to determine whether a trend-sensitive test (Williams' or Shirley's test) was more appropriate for pairwise comparisons than a test that does not assume a monotonic dose-related trend (Dunnett's or Dunn's test). Prior to statistical analysis, extreme values identified by the outlier test of Dixon and Massey (1957) were examined by NTP personnel, and implausible values were eliminated from the analysis. Tests for extended periods of estrus, diestrus, metestrus, and proestrus, as well as skipped estrus and skipped diestrus, were constructed based on a Markov chain model proposed by Girard and Sager (1987). For each dose group, a transition probability matrix was estimated for transitions among the proestrus, estrus, metestrus, and diestrus stages,

with provision for extended stays within each stage as well as for skipping estrus or diestrus within a cycle. Equality of transition matrices among dose groups and between the control group and each dosed group was tested using chi-square statistics. P values for these analyses are two-sided.

Historical Control Data

The concurrent control group represents the most valid comparison to the treated groups and is the only control group analyzed statistically in NTP bioassays. However, historical control data are often helpful in interpreting potential treatment-related effects, particularly for uncommon or rare neoplasm types. For meaningful comparisons, the conditions for studies in the historical control database must be generally similar. Significant factors affecting the background incidences of neoplasms at a variety of sites are diet, sex, strain/stock, and route of exposure. The NTP historical control database contains all 2-year studies for each species, sex, and strain/stock with histopathology findings in control animals completed within the most recent 5-year period (Haseman, 1992, 1995; Haseman and Rao, 1992). In general, the historical control database for a given study includes studies using the same route of administration, and the overall incidences of neoplasms in controls for all routes of administration are included for comparison. Because the two mouse studies presented in this report are the only two using this whole-body exposure method, only the overall incidences for all routes are included.

QUALITY ASSURANCE METHODS

The 28-day and 2-year studies were conducted in compliance with Food and Drug Administration Good Laboratory Practice Regulations (21 CFR, Part 58). In addition, the 28-day and 2-year study reports were audited retrospectively by an independent QA contractor against study records submitted to the NTP Archives. Separate audits covered completeness and accuracy of the pathology data, pathology specimens, final pathology tables, and a draft of this NTP Technical Report. Audit procedures and findings are presented in the reports and are on file at NIEHS. The audit findings were reviewed and assessed by NTP staff, and all comments were resolved or otherwise addressed during the preparation of this Technical Report.

GENETIC TOXICOLOGY

The genetic toxicity of GSM- and CDMA-modulated cell phone RFR was assessed by measuring the frequency of micronucleated erythrocytes in peripheral blood and DNA damage in five different tissues of male and female mice following 14 weeks of exposure. Micronuclei (literally "small nuclei" or Howell-Jolly bodies) are biomarkers of induced structural or numerical chromosomal alterations and are formed when acentric fragments or whole chromosomes fail to incorporate into either of two daughter nuclei during cell division (Schmid, 1975; Heddle *et al.*, 1983). The alkaline (pH>13) comet assay (OECD, 2014) (also known as the single cell gel electrophoresis assay) detects DNA damage in any of a variety of eukaryotic cell types (Tice *et al.*, 2000; Collins, 2004; Brendler-Schwaab *et al.*, 2005; Burlinson *et al.*, 2007); cell division is not required. The type of DNA damage detected includes nicks, adducts, strand breaks, and abasic sites that are converted to DNA strand breaks after treatment of cells in an alkaline (pH>13) solution. Transient DNA strand breaks generated by the process of DNA excision repair may also be detected. DNA damage caused by crosslinking agents has been detected as a reduction of DNA migration (Pfuhler and Wolf, 1996; Hartmann *et al.*, 2003). The fate of the DNA damage detected by the comet assay is varied; most of the damage is rapidly repaired resulting in no sustained impact on the tissue but some may result in cell death or may be incorrectly processed by the repair proteins and result in a fixed mutation or chromosomal alteration. The detailed protocols for these studies and the results are given in Appendix E.

The genetic toxicity studies have grown out of an earlier effort by the NTP to develop a comprehensive database permitting a critical anticipation of a test article's carcinogenicity in experimental animals based on the results from a number of *in vitro* and *in vivo* short-term tests measuring functionally distinct genotoxicity endpoints. The short-term tests were originally developed to clarify proposed mechanisms of chemical-induced DNA damage based on the relationship between electrophilicity and mutagenicity (Miller and Miller, 1977) and the somatic mutation theory of cancer (Straus, 1981; Crawford, 1985). However, it should be noted that not all cancers arise through genotoxic mechanisms, and in these studies, the test article is not a chemical. Many studies have established the genotoxicity of some forms of radiation including, for example, UV light radiation and X-ray radiation, which are both forms of ionizing radiation. Because exposure to cell phone RFR requires specialized and highly technical exposure protocols, only *in vivo* biomarkers associated with genotoxicity could be investigated.

Clearly positive results in long-term peripheral blood micronucleus tests have high predictivity for rodent carcinogenicity; a weak response in one sex only or negative results in both sexes in this assay do not correlate well with either negative or positive results in rodent carcinogenicity studies (Witt *et al.*, 2000). The relationship between comet assay results and rodent carcinogenicity was investigated previously and a close association was observed (Sasaki *et al.*, 2000); however, this assay is best employed as a hazard identification assay. Because of the theoretical and observed associations between induced genetic damage and adverse effects in somatic and germ cells, the determination of *in vivo* genetic effects is important to the overall understanding of the risks associated with exposure to a particular test article.

RESULTS

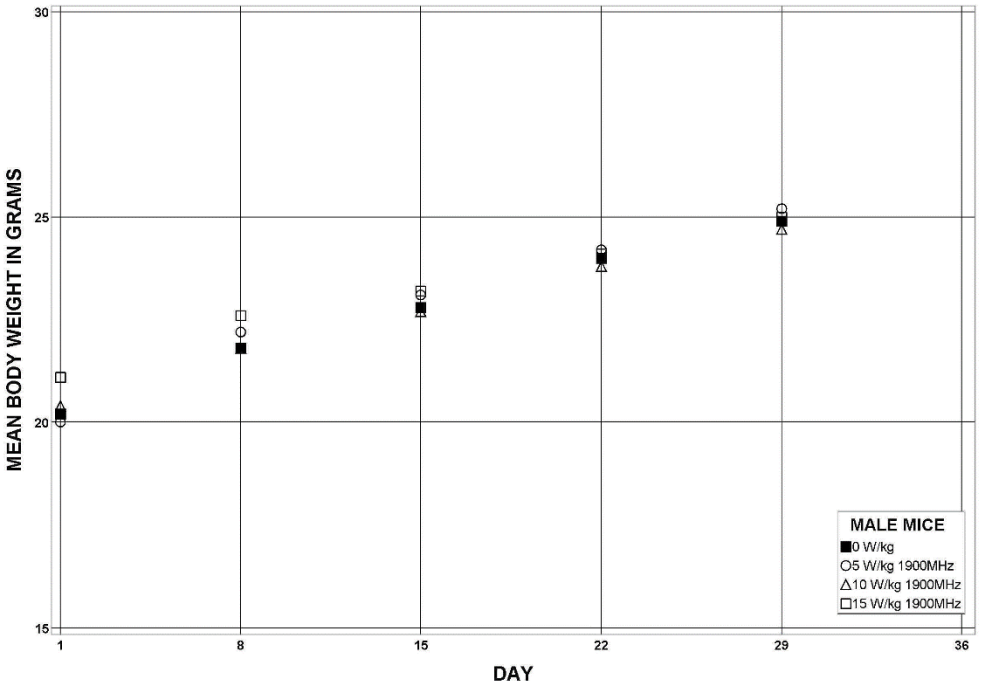
GSM

28-DAY STUDY

All mice survived to the end of the study (Table 2). Weekly mean body weights of exposed groups of males and females were similar to those of the sham controls at all time points (Table 2 and Figure 4). There were no clinical signs related to exposure to GSM cell phone RFR.

TABLE 2
Mean Body Weights and Survival of Mice Exposed to GSM-Modulated Cell Phone RFR for 28 Days

	Sham Control		5 W/kg			10 W/kg			15 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	20.2	10	20.0	98.9	10	20.4	100.8	10	21.1	104.7	10
8	21.8	10	22.2	101.5	10	21.8	99.8	10	22.6	103.3	10
15	22.8	10	23.1	101.4	10	22.7	99.4	10	23.2	101.7	10
22	24.0	10	24.2	101.0	10	23.8	99.4	10	24.1	100.5	10
29	24.9	10	25.2	101.2	10	24.7	99.5	10	25.0	100.5	10
Female											
1	18.1	10	17.8	98.3	10	17.4	96.1	10	17.9	98.9	10
8	18.9	10	19.0	100.7	10	18.4	97.3	10	18.5	98.0	10
15	20.1	10	20.1	100.0	10	19.5	97.0	10	19.6	97.3	10
22	21.0	10	21.1	100.4	10	20.4	97.1	10	20.3	96.8	10
30	21.7	10	21.9	100.9	10	21.2	97.5	10	21.0	96.6	10



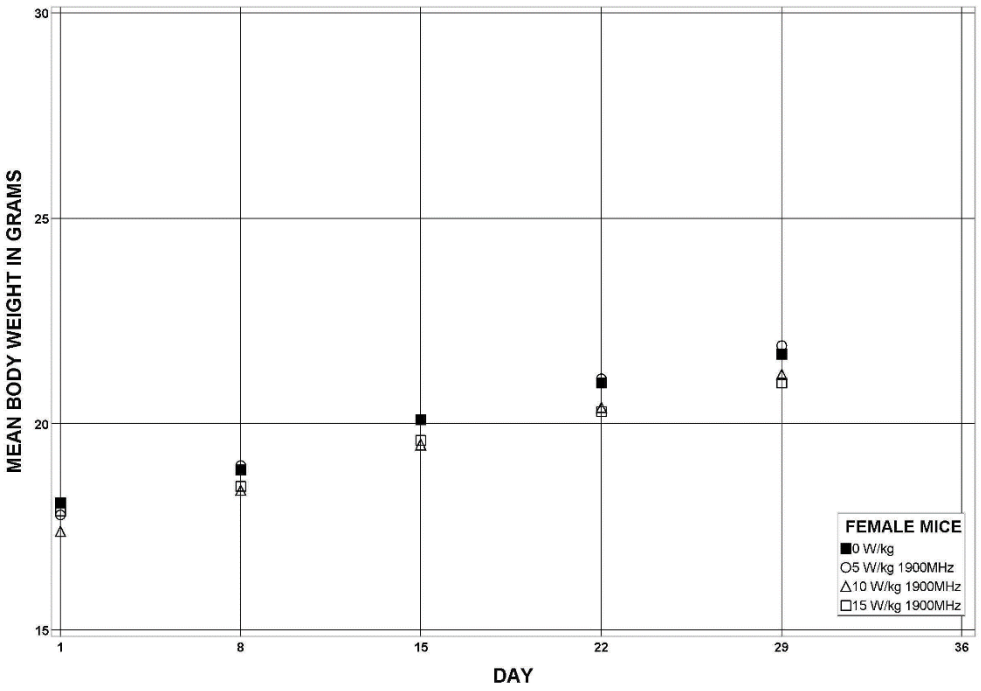


FIGURE 4
Growth Curves for Mice Exposed to GSM-Modulated Cell Phone RFR for 28 Days

Body temperatures were significantly higher in RFR-exposed male mice at several time points (Table 3). In female mice, there were a few occurrences of significantly lower body temperatures in the exposed groups, but no significantly higher body temperatures. These changes in body temperature were inconsistent and not SAR-related.

TABLE 3
Mean Body Temperatures of Mice Exposed to GSM-Modulated Cell Phone RFR for 28 Days[a]

Day	Sham Control		5 W/kg		10 W/kg		15 W/kg	
	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured
Male								
0	37.0 ± 0.2	10	38.5 ± 0.2	10	37.3 ± 0.3	10	36.2 ± 0.2*	10
2	35.7 ± 0.1	10	37.2 ± 0.3**	10	37.1 ± 0.3**	10	37.0 ± 0.3**	10
4	36.2 ± 0.2	10	37.0 ± 0.2	10	37.1 ± 0.3*	10	37.1 ± 0.2*	10
7[b]	36.6 ± 0.2	9	37.4 ± 0.2	10	37.7 ± 0.4*	10	36.8 ± 0.1	10
14[b]	35.5 ± 0.3	10	36.0 ± 0.1	10	36.1 ± 0.4	10	35.9 ± 0.1	10
17	36.0 ± 0.3	10	37.2 ± 0.3*	10	36.7 ± 0.3	10	36.8 ± 0.4	10
20	36.5 ± 0.3	10	37.0 ± 0.3	10	37.6 ± 0.3*	10	37.2 ± 0.2	10
27	35.8 ± 0.4	9	37.6 ± 0.3**	10	37.4 ± 0.2**	10	37.2 ± 0.3**	10
2-27[c]	36.0 ± 0.2	10	37.1 ± 0.1**	10	37.1 ± 0.2**	10	36.9 ± 0.2**	10
Female								
0	38.1 ± 0.1	10	37.9 ± 0.1	9	37.2 ± 0.3**	9	37.2 ± 0.1**	10
2	37.5 ± 0.2	10	37.4 ± 0.2	9	37.3 ± 0.3	9	37.5 ± 0.1	10
4	37.0 ± 0.2	10	37.5 ± 0.2	10	37.1 ± 0.5	10	37.6 ± 0.1	10
7[b]	38.6 ± 0.1	10	38.1 ± 0.2	10	37.8 ± 0.5	10	38.5 ± 0.1	10
14[b]	36.9 ± 0.1	10	36.4 ± 0.1	10	36.6 ± 0.2	9	37.0 ± 0.2	10
17	37.9 ± 0.1	10	37.3 ± 0.2**	10	37.7 ± 0.1	9	37.6 ± 0.1	10
20	37.7 ± 0.2	10	37.6 ± 0.2	10	37.6 ± 0.1	9	37.8 ± 0.1	10
27	37.8 ± 0.1	10	38.2 ± 0.1	10	37.2 ± 0.2*	9	37.5 ± 0.2	10
2-27	37.6 ± 0.1	10	37.5 ± 0.1	10	37.3 ± 0.2	10	37.6 ± 0.1	10

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test
** $P \leq 0.01$
[a] Temperatures are given as mean ± standard error.
[b] All temperatures were recorded within 5 minutes of the exposure cessation, except for the measurements on days 7 and 14, which were recorded at least 1 hour after exposure.
[c] Average of days 2 to 27, excluding days 7 and 14

There were no exposure-related effects on the organ weights of males exposed to GSM cell phone RFR (Table G1). The absolute heart weight of 15 W/kg females was significantly less than that of the sham controls, and there were negative trends in the absolute weights of the brain, right kidney, and liver, all of which were considered to be due to minor reductions in body weight. There were no significantly lower relative organ weights and no associated histopathologic findings, therefore, these organ weight changes were considered sporadic and not related to GSM cell phone RFR exposure.

There were no histopathologic lesions related to the effects of exposure to GSM cell phone RFR.

Exposure Level Selection Rationale: In male and female mice exposed for 5 days to cell phone RFR up to 12 W/kg, only sporadic increases were observed in body temperature, regardless of the sex or age of the animals (Wyde *et al.*, 2018). Because no significant effects of cell phone RFR were observed in body temperature at 12 W/kg, a higher upper exposure level was selected for the 28-day studies. Due to limits on the maximum capacity of the exposure system to generate high RF fields, the maximum achievable exposure level capacity was 15 W/kg, which was selected as the highest exposure level for the 28-day studies. Selection of the highest exposure level for the 2-year studies was also limited by the power capacity of the exposure system to generate maximum RF fields. Based on the technical limitations and increased body temperature at various time points that were similarly observed at 10 and 15 W/kg in the 28-day studies, the exposure levels selected for the 2-year studies were 2.5, 5, and 10 W/kg.

2-YEAR STUDY

Survival

Estimates of 2-year survival probabilities for male and female mice are shown in Table 4 and in the Kaplan-Meier survival curves (Figure 5). Survival was significantly higher for the 5 W/kg males than the sham control group. Survival of the rest of the exposed groups of males and females was generally similar to that of the sham controls.

TABLE 4
Survival of Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Male				
Animals initially in study	105	105	105	105
14-week interim evaluation[a]	15	15	15	15
Accidental death[b]	0	1	0	0
Missing[b]	0	1	0	0
Moribund	8	6	2	6
Natural deaths	16	19	8	12
Animals surviving to study termination	66	63	80[f]	72[g]
Percent probability of survival at end of study[c]	73	72	89	80
Mean survival (days)[d]	687	693	717	707
Survival analysis[e]	P=0.135N	P=0.959	P=0.013N	P=0.360N
Female				
Animals initially in study	105	105	105	105
14-week interim evaluation[a]	15	15	15	15
Moribund	9	9	9	6
Natural deaths	14	7	11	11
Animals surviving to study termination	67[f]	74[h]	70[i]	73[j]
Percent probability of survival at end of study	74	80	77	80
Mean survival (days)	704	715	711	712
Survival analysis	P=0.476N	P=0.420	P=0.709N	P=0.405N

[a] Excluded from survival analysis
[b] Censored in the survival analysis
[c] Kaplan-Meier determinations
[d] Mean of all deaths (uncensored, censored, and terminal euthanasia)
[e] The result of the life table trend test (Tarone, 1975) is in the sham control column, and the results of the life table pairwise comparisons (Cox, 1972) with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by **N.**
[f] Includes one animal that died during the last week of the study
[g] Includes four animals that died during the last week of the study
[h] Includes four animals that died during the last week of the study; two of these were censored in the survival analysis
[i] Includes two animals that died during the last week of the study; one of these was censored in the survival analysis
[j] Includes one animal that died during the last week of the study; this animal was censored in the survival analysis

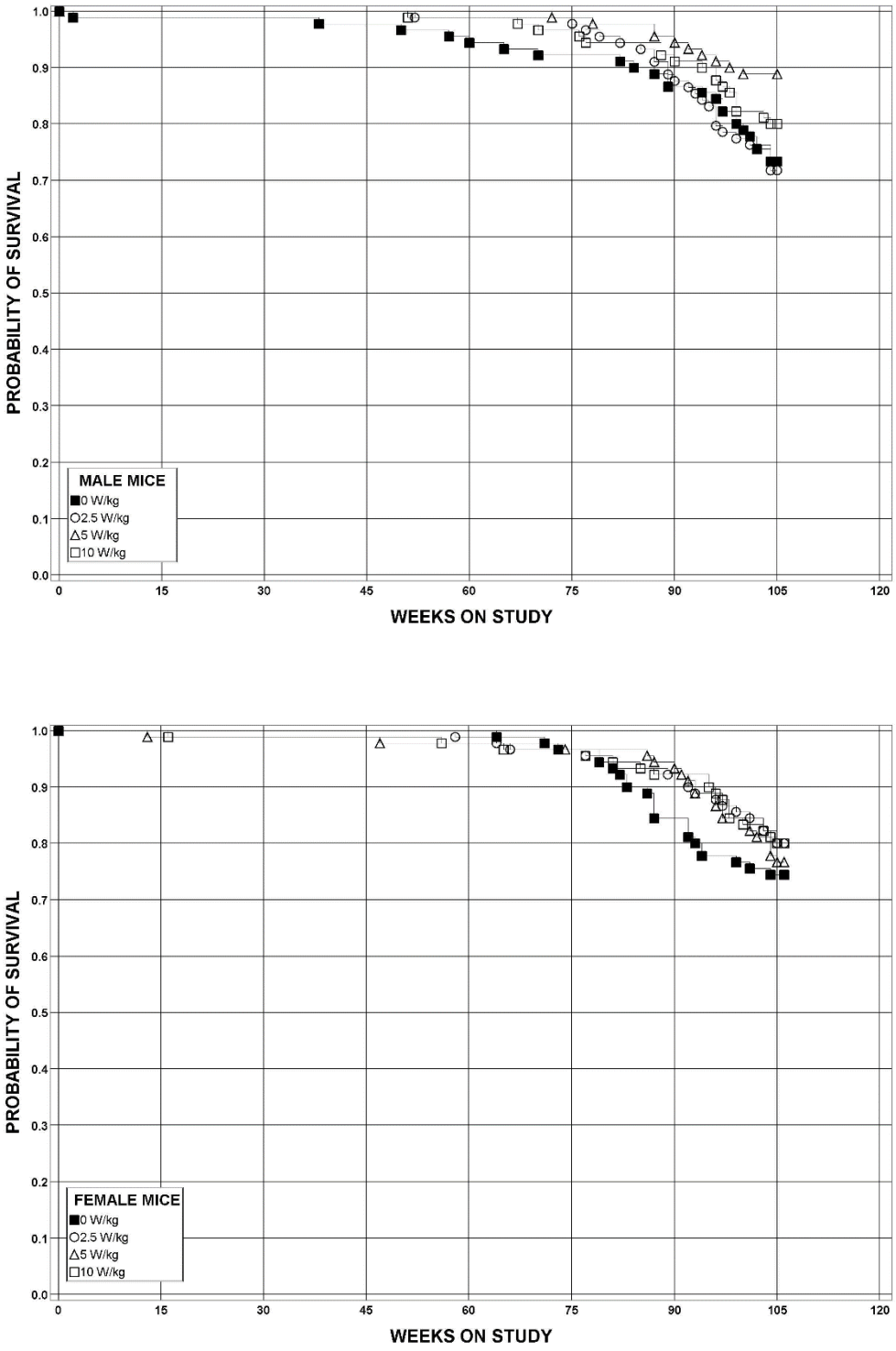


FIGURE 5
Kaplan-Meier Survival Curves for Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

Body Weights and Clinical Observations

Mean body weights of exposed groups of males and females were similar to those of the sham controls throughout the study (Tables 5 and 6; Figure 6). Clinical signs included more occurrences of thin and ruffled fur in 10 W/kg males and thin, ruffled fur, and mass-torso/ventral in 5 and 10 W/kg females. These findings were not correlated with differences in body weights or incidences of neoplasms in exposed animals.

TABLE 5
Mean Body Weights and Survival of Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control		2.5 W/kg			5 W/kg			10 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
0	20.6	105	20.4	99.1	105	20.4	99.4	105	20.4	99.0	105
8	21.9	104	22.0	100.4	104	21.8	99.5	105	21.9	100.2	105
15	22.9	104	23.1	100.7	104	22.8	99.4	105	23.1	100.6	105
22	24.1	104	24.4	101.4	104	24.0	99.7	105	23.9	99.1	105
29	25.1	104	25.4	101.2	104	25.0	99.6	105	24.8	98.5	105
36	26.3	104	26.2	99.9	104	26.1	99.5	105	25.7	98.0	105
43	27.3	104	27.1	99.3	104	27.2	99.5	105	26.4	96.7	105
50	28.1	104	27.8	99.1	104	28.1	100.2	105	27.5	98.1	105
57	29.3	104	28.9	98.5	104	29.3	99.9	105	28.7	97.8	105
64	30.5	104	29.6	97.2	104	30.3	99.4	105	29.7	97.2	105
71	31.7	104	30.8	97.3	104	31.7	100.1	105	30.9	97.5	105
79	32.9	104	31.8	96.6	104	32.9	100.1	105	32.1	97.5	105
86	33.6	104	33.0	98.3	104	33.8	100.5	105	32.7	97.2	105
93	34.3	94	34.0	99.0	94	34.7	101.2	95	33.6	98.0	95
121	38.6	89	38.5	99.5	89	39.4	101.9	90	38.5	99.7	90
149	42.0	89	42.2	100.6	89	42.1	100.2	90	42.8	101.9	90
177	44.3	89	44.4	100.3	89	44.7	100.9	90	44.8	101.0	90
205	46.0	89	46.3	100.7	89	46.4	100.8	90	46.1	100.3	90
233	47.3	89	47.1	99.7	89	47.3	100.1	90	46.7	98.8	90
261	47.6	89	47.9	100.8	89	47.9	100.6	90	47.1	99.0	90
289	48.2	88	48.6	100.7	89	48.5	100.6	90	47.7	98.9	90
317	48.8	88	49.1	100.7	89	49.1	100.6	90	48.2	98.8	90
345	49.4	88	49.7	100.7	89	49.7	100.6	90	48.6	98.4	90
373	50.0	87	50.3	100.6	88	50.2	100.4	90	49.2	98.3	89
401	50.4	86	51.0	101.0	88	51.0	101.0	90	49.7	98.4	89
429	50.7	85	51.2	100.9	88	51.5	101.4	90	50.1	98.8	89
457	51.1	84	51.7	101.1	88	51.9	101.5	90	50.3	98.4	89
485	51.5	84	52.1	101.3	88	52.3	101.5	90	51.1	99.3	88
513	50.5	83	51.4	101.8	88	52.0	103.0	89	50.6	100.2	87
541	49.7	83	50.7	102.0	86	51.2	103.0	89	50.6	101.7	85
569	50.4	82	51.1	101.5	85	51.8	102.8	88	51.2	101.8	85
597	50.9	81	51.8	101.7	83	52.1	102.3	88	51.5	101.3	85
625	50.7	78	51.6	101.7	79	52.1	102.7	86	51.5	101.5	82
639	49.8	78	51.5	103.6	78	51.9	104.3	85	51.1	102.7	82
653	49.0	78	51.1	104.3	75	51.9	105.8	84	50.8	103.6	82
667	49.0	76	51.0	104.0	71	52.0	106.1	82	50.5	103.0	81
681	49.2	74	51.1	103.9	69	51.7	105.0	82	49.8	101.2	77
695	48.7	71	50.6	104.0	68	51.1	104.9	81	50.0	102.7	74
709	48.5	69	50.3	103.6	67	50.6	104.3	80	49.1	101.3	74
723	48.4	67	50.6	104.6	65	50.0	103.4	80	48.9	101.1	73
Mean for Weeks											
1-13	27.3		27.0	99.2		27.2	99.8		26.8	98.3	
14-52	44.7		44.8	100.3		45.0	100.8		44.4	99.5	
53-105	49.9		51.1	102.4		51.5	103.1		50.4	100.9	

TABLE 6
Mean Body Weights and Survival of Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control		2.5 W/kg			5 W/kg			10 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
0	17.4	105	17.2	99.1	105	17.5[a]	100.3	104	17.3	99.6	105
8	18.4	105	18.3	99.5	105	18.5	100.9	105	18.4	100.3	105
15	19.4	105	19.4	99.6	105	19.4	99.6	105	19.3	99.3	105
22	20.2	105	20.3	100.4	105	20.2	99.8	105	20.2	99.8	105
29	20.8	105	20.9	100.5	105	20.9	100.4	105	20.8	99.8	105
36	21.5	105	21.5	99.9	105	21.7	100.9	105	21.5	99.9	105
43	22.0	105	21.9	99.4	105	21.9	99.8	105	21.8	99.4	105
50	22.5	105	22.3	98.8	105	22.5	99.9	105	22.5	100.1	105
57	22.8	105	22.6	99.0	105	22.9	100.6	105	22.7	99.8	105
64	23.3	105	23.3	99.7	105	23.7	101.4	105	23.5	100.7	105
71	23.4	105	23.6	101.0	105	24.1	102.9	105	24.1	102.9	105
79	23.9	105	24.2	101.0	105	24.4	102.1	105	24.6	102.9	105
86	24.0	105	24.3	101.4	105	24.5	101.9	104	24.7	102.8	105
93	24.3	95	24.4	100.3	95	25.2	103.6	94	25.0	103.0	95
121	26.3	90	26.8	101.7	90	28.2	107.2	89	27.3	103.6	89
149	28.8	90	29.3	101.6	90	30.6	106.2	89	30.4	105.5	89
177	30.8	90	31.7	103.1	90	33.6	109.3	89	33.4	108.5	89
205	33.4	90	34.9	104.6	90	36.7	109.9	89	36.1	108.2	89
233	36.8	90	37.3	101.5	90	39.7	108.0	89	39.0	106.0	89
261	38.4	90	38.9	101.3	90	41.5	107.9	89	41.6	108.2	89
289	40.3	90	40.3	100.1	90	43.2	107.1	89	42.7	106.0	89
317	42.3	90	42.8	101.4	90	45.4	107.6	89	45.3	107.2	89
345	45.0	90	45.3	100.7	90	47.7	106.0	88	47.2	104.8	89
373	47.6	90	47.7	100.1	90	49.6	104.2	88	49.1	103.0	89
401	49.9	90	49.3	98.7	90	51.9	103.9	88	51.2	102.5	88
429	51.4	90	51.3	99.9	89	53.4	103.9	88	52.4	102.1	88
457	53.3	89	52.6	98.7	88	54.5	102.3	88	53.7	100.8	87
485	55.0	89	53.9	98.1	87	55.6	101.1	88	55.4	100.7	87
513	54.5	87	53.3	97.8	87	54.1	99.3	88	54.1	99.2	87
541	51.9	87	51.4	99.1	86	52.6	101.4	87	52.2	100.7	86
569	52.2	83	52.0	99.7	84	53.6	102.7	87	53.0	101.5	85
597	55.3	80	54.4	98.4	84	55.0	99.5	87	54.8	99.2	84
625	56.3	76	55.0	97.8	83	55.5	98.6	85	56.0	99.6	83
639	54.8	75	54.0	98.6	82	54.6	99.6	83	54.5	99.6	83
653	54.5	71	53.4	97.9	80	54.9	100.8	80	53.8	98.7	83
667	55.1	70	53.2	96.6	79	54.8	99.6	80	53.5	97.2	81
681	54.6	70	52.7	96.5	78	54.2	99.2	76	53.4	97.8	77
695	54.0	69	52.1	96.3	77	53.1	98.2	76	52.8	97.7	76
709	53.7	68	52.1	96.9	76	52.0	96.8	74	51.8	96.5	75
723	53.0	68	51.7	97.6	74	51.8	97.7	71	52.0	98.1	74
737	52.2	67	51.2	98.1	72	51.1	97.8	69	51.6	98.9	72
Mean for Weeks											
1-13	22.4		22.5	100.1		21.7	101.0		22.6	100.8	
14-52	34.6		35.2	101.6		37.2	107.3		36.8	106.1	
53-107	53.3		52.3	98.2		53.5	100.4		53.1	99.7	

[a] One animal not weighed

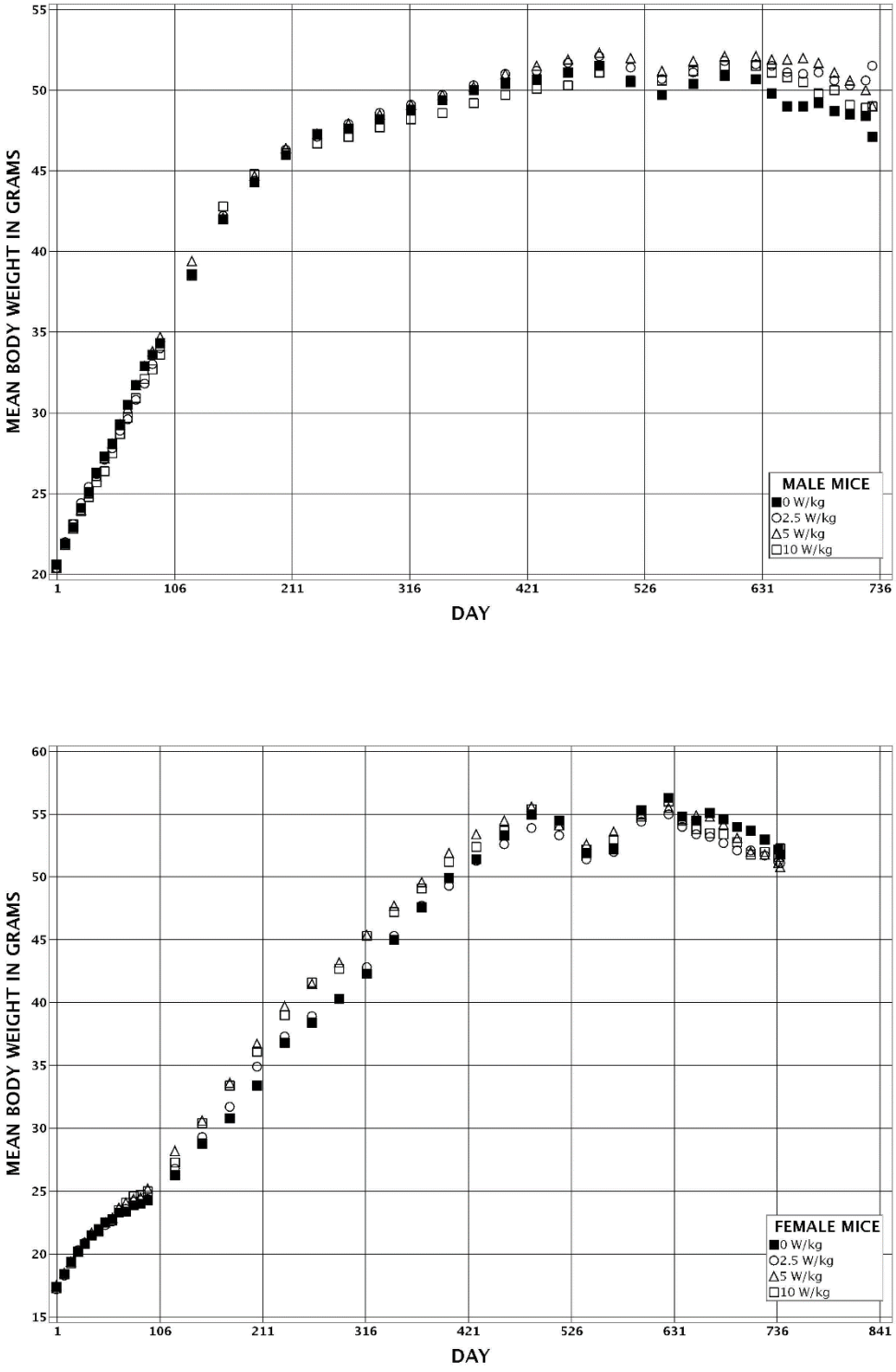


FIGURE 6
Growth Curves for Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

14-Week Interim Evaluation

There were no changes to the hematology variables attributable to GSM cell phone RFR exposure (Table F1).

At the 14-week interim evaluation, mean body weights of exposed groups of males and females were similar to those of the sham controls (Table G2). In males, the absolute right kidney weights were significantly lower (7%) in the 5 and 10 W/kg groups compared to the sham controls, and the absolute left kidney weight was significantly lower (12%) in the 10 W/kg group (Table G2). The absolute liver weights of 5 and 10 W/kg males were significantly lower (10%) and the relative liver weight was significantly lower in 5 W/kg males. These organ weight changes were considered small changes and were not accompanied by exposure-related histopathologic lesions. In 10 W/kg females, there were significantly lower relative weights in the brain and right kidney (Table G2); these changes were not accompanied by significant changes in absolute weights and were not considered toxicologically important. The absolute thymus weight of 10 W/kg females was 20% higher compared to the sham controls, but this was not correlated with any histopathologic lesions in the thymus.

In males, there were no exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility (Table H1). In females, there were no exposure-related effects on estrous cycle length, number of cycling females, or relative amount of time spent in the estrous stages (Tables H2 and H3; Figure H1).

In the liver, a significantly higher incidence of focal inflammation occurred in 5 W/kg males (sham control, 0/10; 2.5 W/kg, 2/10; 5 W/kg, 4/10; 10 W/kg, 0/10; Table A4). Focal inflammation is commonly seen in B6C3F1/N mice, and consisted of small clusters of mixed inflammatory cells, predominantly lymphocytes with fewer macrophages and an occasional neutrophil. There was no zonal pattern to this finding and the inflammation was randomly scattered within the hepatic parenchyma. All of the lesions were of minimal severity that typically consisted of one to three small areas of inflammation, and they were not considered biologically relevant.

Pathology and Statistical Analyses

This section describes the statistically significant or potentially biologically noteworthy changes in the incidences of malignant lymphoma and neoplasms and/or nonneoplastic lesions of the skin, lung, mediastinum, and ovary in the 2-year study. Summaries of the incidences of neoplasms and nonneoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix A for male mice and Appendix B for female mice.

Skin (Subcutaneous Tissue): The incidences of malignant fibrous histiocytoma were higher in 5 and 10 W/kg males, although not significantly or in an exposure concentration-related manner (Tables 7, A1, and A2); however, the incidences exceeded the overall historical control ranges for malignant fibrous histiocytoma (Tables 7 and A3a). The combined incidences of fibrosarcoma, sarcoma, or malignant fibrous histiocytoma were also increased in the 5 and 10 W/kg males, although still not in a statistically significant or exposure concentration-dependent manner. In males, all but one of the malignant fibrous histiocytomas occurred on the tail; the remaining neoplasm (in a 5 W/kg animal) was located on the pinna of the ear. Malignant fibrous histiocytomas can have a variable appearance. In general, all the malignant fibrous histiocytomas had a portion of the neoplasm that was composed of spindle-shaped cells arranged in interlacing or irregular bundles or whorls amongst a background of varying amounts of collagen and a sizable population of cells resembling histiocytes – large cells with abundant eosinophilic cytoplasm and small basophilic nuclei. Multinucleated cells were present in most of the tumors, but were more abundant in the neoplasm on the ear. Several of the neoplasms on the tail had areas of pigment in the section – possibly from the tail tattoo. The single malignant fibrous histiocytoma that occurred in a sham control male metastasized throughout the abdominal cavity, involving the liver, stomach, mesentery, adrenal gland, and seminal vesicle, as well as being found in the mesenteric lymph nodes and skeletal muscle. None of the other neoplasms had distant metastases.

The single occurrences of sarcoma in a 2.5 W/kg male (sham control, 0/90; 2.5 W/kg, 1/89; 5 W/kg, 0/90; 10 W/kg, 0/90) and fibrosarcoma in a 10 W/kg male (0/90, 0/89, 0/90, 1/90) were histologically much different from the malignant fibrous histiocytomas (Table A1). They were much larger neoplasms, with large areas of necrosis. They were poorly circumscribed and consisted of interlacing bundles of elongated cells in a background of varying

TABLE 7
Incidences of Neoplasms of the Skin (Subcutaneous Tissue) in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Number Examined Microscopically	90	89	90	90
Malignant Fibrous Histiocytoma, Multiple[a]	0	0	1 (1%)	0
Malignant Fibrous Histiocytoma (includes multiple)[b]				
Overall rate[c]	1/90 (1%)	0/89 (0%)	5/90 (6%)	3/90 (3%)
Adjusted rate[d]	1.2%	0.0%	5.8%	3.6%
Terminal rate[e]	0/66 (0%)	0/63 (0%)	4/80 (5%)	3/72 (4%)
First incidence (days)	674	—[g]	654	729 (T)
Poly-3 test[f]	P=0.127	P=0.499N	P=0.124	P=0.321
Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma[h]				
Overall rate	1/90 (1%)	1/89 (1%)	5/90 (6%)	4/90 (4%)
Adjusted rate	1.2%	1.2%	5.8%	4.7%
Terminal rate	0/66 (0%)	0/63 (0%)	4/80 (5%)	3/72 (4%)
First incidence (days)	674	523	654	488
Poly-3 test	P=0.093	P=0.758N	P=0.124	P=0.197

(T) Terminal euthanasia
[a] Number of animals with neoplasm
[b] Historical control incidence for 2-year studies (all routes) (mean ± standard deviation): 2/589 (0.3% ± 0.7%), range 0%-2%
[c] Number of animals with neoplasm per number of animals necropsied
[d] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
[e] Observed incidence at terminal euthanasia
[f] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A lower incidence in an exposure group is indicated by **N**.
[g] Not applicable; no neoplasms in animal group
[h] Historical control incidence: 5/589 (0.8% ± 1.0%), range 0%-2%

amounts of collagen or myxomatous material. Nuclei were long and oval and typically vesicular, in comparison to the small, often round, densely basophilic nuclei found in the malignant fibrous histiocytomas. There was no population of histiocyte-like cells in the sarcoma or the fibrosarcoma. Neither of these neoplasms occurred on the tail. Fibrosarcoma, sarcoma, and malignant fibrous histiocytomas are all neoplasms of mesenchymal origin.

Lung: There was a significant positive trend in the incidences of alveolar/bronchiolar adenoma or carcinoma (combined) in males (Tables 8 and A2). The incidences of focal alveolar epithelial hyperplasia were similar in all groups of males (6/90, 8/89, 8/90, 7/90; Table A4). Alveolar/bronchiolar adenomas were discrete, expansile proliferations of cuboidal to columnar cells supported by a fine fibrovascular stroma arranged in solid nests or papillary fronds projecting into alveolar spaces and causing compression of the surrounding parenchyma.

TABLE 8
Incidences of Alveolar/bronchiolar Neoplasms of the Lung in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Number Examined Microscopically	90	89	90	90
Alveolar/bronchiolar Adenoma, Multiple[a]	2	0	2	1
Alveolar/bronchiolar Adenoma (includes multiple)[b]				
Overall rate[c]	13/90 (14%)	13/89 (15%)	18/90 (20%)	16/90 (18%)
Adjusted rate[d]	16.0%	16.0%	20.7%	19.0%
Terminal rate[e]	9/66 (14%)	10/63 (16%)	16/80 (20%)	14/72 (19%)
First incidence (days)	488	663	604	658
Poly-3 test[f]	P=0.297	P=0.583	P=0.279	P=0.380
Alveolar/bronchiolar Carcinoma, Multiple	2	0	1	1
Alveolar/bronchiolar Carcinoma (includes multiple)[g]				
Overall rate	13/90 (14%)	12/89 (13%)	16/90 (18%)	18/90 (20%)
Adjusted rate	16.1%	14.7%	18.5%	21.2%
Terminal rate	12/66 (18%)	8/63 (13%)	16/80 (20%)	14/72 (19%)
First incidence (days)	568	594	729 (T)	614
Poly-3 test	P=0.165	P=0.488N	P=0.418	P=0.259
Alveolar/bronchiolar Adenoma or Carcinoma[h]				
Overall rate	23/90 (26%)	24/89 (27%)	32/90 (36%)	34/90 (38%)
Adjusted rate	28.1%	29.2%	36.8%	39.9%
Terminal rate	18/66 (27%)	17/63 (27%)	30/80 (38%)	28/72 (39%)
First incidence (days)	488	594	604	614
Poly-3 test	P=0.040	P=0.506	P=0.149	P=0.074

(T) Terminal euthanasia

[a] Number of animals with neoplasm

[b] Historical control incidence for 2-year studies (all routes) (mean ± standard deviation): 84/589 (14.3% ± 5.4%), range 8%-24%

[c] Number of animals with neoplasm per number of animals with lung examined microscopically

[d] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

[e] Observed incidence at terminal euthanasia

[f] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A lower incidence in an exposure group is indicated by **N**.

[g] Historical control incidence: 66/589 (11.0% ± 4.4%), range 4%-20%

[h] Historical control incidence: 142/589 (24.0% ± 5.3%), range 16%-34%

Alveolar/bronchiolar carcinomas were usually larger than adenomas and tended to be poorly demarcated and locally invasive. They were composed of cuboidal to columnar epithelial cells that displayed moderate to marked pleomorphism and lacked a normal orderly arrangement, with multiple layers and piling up of cells. The neoplastic cells were arranged in papillary arrangements or solid sheets of cells; most carcinomas contained both growth patterns. Occasional mitoses were present.

Malignant Lymphoma: Compared to the sham controls, all exposed groups of females had higher incidences of malignant lymphoma and the incidences in the 2.5 and 5 W/kg groups were significantly higher (Tables 9, B1, and B2). The sham control group had a low incidence of malignant lymphoma compared to the range seen in historical controls (Tables 9 and B3). All of the incidences in the exposed groups fell within the overall historical control range. Malignant lymphoma involved many organs, most frequently the spleen, lymph nodes, thymus, lung, kidney, liver, and bone marrow, and was characterized by the effacement of normal architecture by a monomorphic population of neoplastic lymphocytes, which tended to be larger than normal lymphocytes. In spleens with malignant lymphoma, there was a loss of individual follicles and periarteriolar lymphoid sheaths, as the enlarged white pulp became one solid sheet of neoplastic cells sometimes leading to the gross enlargement of the organ. In the lymph nodes and thymus, malignant lymphoma led to the loss of distinguishable cortical and medullary regions, with the entire node appearing to contain only a single type of cell. Involved lymph nodes were typically grossly enlarged. In the liver and kidney, aggregates of neoplastic lymphocytes disrupted the normal arrangement of the parenchyma, and in the lungs, neoplastic lymphocytes were often found expanding the bronchial-associated lymphoid tissue. Malignant lymphoma in the bone marrow resulted in a hypercellular marrow cavity with a monotonous population of malignant lymphocytes rather than the typical mix of erythrocytes and leukocytes in various stages of maturity.

TABLE 9
Incidences of Malignant Lymphoma in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Malignant Lymphoma[a]				
Overall rate[b]	2/90 (2%)	13/90 (14%)	9/90 (10%)	6/90 (7%)
Adjusted rate[c]	2.5%	15.6%	10.7%	7.1%
Terminal rate[d]	1/67 (1%)	12/72 (17%)	5/69 (7%)	3/72 (4%)
First incidence (days)	604	731	516	590
Poly-3 test[e]	P=0.474	P=0.004	P=0.035	P=0.153

[a] Historical control incidence for 2-year studies (all routes) (mean ± standard deviation): 89/590 (16.0% ± 8.3%), range 2%-36%
[b] Number of animals with neoplasm per number of animals necropsied
[c] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
[d] Observed incidence at terminal euthanasia
[e] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia.

Other Tissues: Several tissues had significantly increased incidences of lesions in one, or even two exposed groups of males or females. Many of them, such as lymphocytic infiltration or inflammation in various tissues, are common findings in aged mice and the incidences and severities recorded in this study were not considered exposure related. The incidence of other lesions lacked an exposure concentration response and were considered sporadic occurrences or of unknown importance.

Two hibernomas of the mediastinum occurred in 5 W/kg males (sham control, 0/90; 2.5 W/kg, 0/89; 5 W/kg, 2/90; 10 W/kg, 0/90; Table A1). These are unusual neoplasms of brown adipose tissue. Hibernomas were composed of round cells with moderate amounts of cytoplasm filled with tiny vacuoles, and small, round nuclei. Two benign ovarian teratomas occurred in 5 W/kg females, and one in 10 W/kg females (0/75, 0/86, 2/82, 1/80; Table B1). Neither of these neoplasms occurred in the sham controls, nor have they occurred in the overall historical control populations [males: mediastinum, hibernoma (0/589); females: ovary, benign teratoma (0/590)]. However, benign teratomas have been reported in the literature to occur in B6C3F1 mice (Alison *et al.*, 1987). Both the hibernomas and the teratomas were considered sporadic occurrences of rare neoplasms, and while unusual, were not considered exposure related.

CDMA

28-DAY STUDY

All mice survived to the end of the study (Table 10). Weekly mean body weights of exposed groups of males and females were similar to those of the sham controls at all time points (Table 10 and Figure 7). There were no clinical signs related to exposure to CDMA cell phone RFR.

TABLE 10
Mean Body Weights and Survival of Mice Exposed to CDMA-Modulated Cell Phone RFR for 28 Days

	Sham Control		5 W/kg			10 W/kg			15 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	20.2	10	20.4	100.7	10	20.4	101.0	10	20.9	103.2	10
8	21.8	10	21.8	100.0	10	22.2	101.6	10	22.4	102.4	10
15	22.8	10	22.4	98.3	10	23.0	100.9	10	23.3	102.3	10
22	24.0	10	23.5	98.0	10	23.9	99.6	10	24.2	101.0	10
29	24.9	10	24.3	97.6	10	25.2	101.2	10	25.1	101.1	10
Female											
1	18.1	10	18.2	100.5	10	17.9	99.2	10	17.6	97.5	10
8	18.9	10	19.0	100.8	10	18.7	99.3	10	18.7	99.0	10
15	20.1	10	20.1	99.6	10	20.0	99.4	10	19.8	98.2	10
22	21.0	10	21.0	99.9	10	20.8	99.1	10	20.5	97.4	10
30	21.7	10	21.7	99.7	10	21.6	99.4	10	21.2	97.5	10

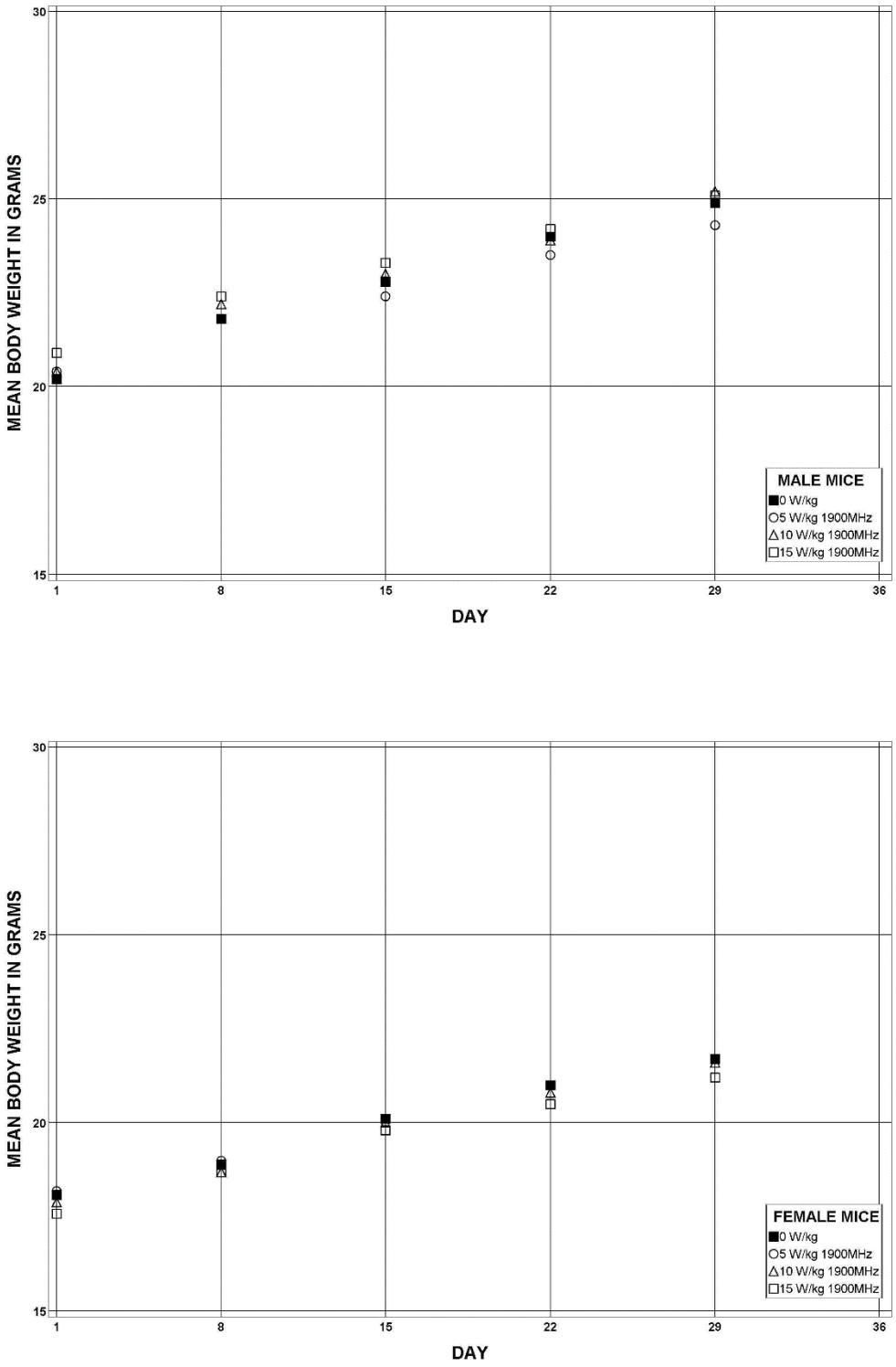


FIGURE 7
Growth Curves for Mice Exposed to CDMA-Modulated Cell Phone RFR for 28 Days

Similar to what was seen in mice exposed to GSM cell phone RFR, body temperatures were significantly higher in males and significantly lower in females at several time points (Table 11).

TABLE 11
Mean Body Temperatures of Mice Exposed to CDMA-Modulated Cell Phone RFR for 28 Days[a]

Day	Sham Control		5 W/kg		10 W/kg		15 W/kg	
	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured	Temperature (° C)	No. Measured
Male								
0	37.0 ± 0.2	10	37.0 ± 0.1	10	38.0 ± 0.2**	10	37.8 ± 0.2**	10
2	35.7 ± 0.1	10	36.1 ± 0.1	10	37.0 ± 0.3**	10	36.5 ± 0.2**	10
4	36.2 ± 0.2	10	36.7 ± 0.2	10	37.0 ± 0.2**	10	37.1 ± 0.2**	10
7[b]	36.6 ± 0.2	9	36.4 ± 0.2	10	37.3 ± 0.3	10	37.3 ± 0.2	10
14[b]	35.5 ± 0.3	10	35.8 ± 0.1	10	36.1 ± 0.2	10	36.0 ± 0.1	10
17	36.0 ± 0.3	10	36.2 ± 0.3	10	36.8 ± 0.4	10	37.2 ± 0.3*	10
20	36.5 ± 0.3	10	36.4 ± 0.2	10	37.3 ± 0.3*	10	37.6 ± 0.2**	10
27	35.8 ± 0.4	9	36.5 ± 0.3	10	37.4 ± 0.3**	10	36.8 ± 0.3	10
2-27[c]	36.0 ± 0.2	10	36.3 ± 0.1	10	37.1 ± 0.1**	10	36.9 ± 0.1**	10
Female								
0	38.1 ± 0.1	10	37.5 ± 0.1*	9	38.3 ± 0.1	10	38.0 ± 0.2	10
2	37.5 ± 0.2	10	37.0 ± 0.2	9	38.1 ± 0.2	10	37.5 ± 0.2	10
4	37.0 ± 0.2	10	37.2 ± 0.2	10	37.7 ± 0.2	10	37.5 ± 0.2	10
7[b]	38.6 ± 0.1	10	37.9 ± 0.2**	9	38.0 ± 0.1*	10	38.3 ± 0.1	10
14[b]	36.9 ± 0.1	10	36.5 ± 0.2	9	37.0 ± 0.1	10	37.0 ± 0.2	10
17	37.9 ± 0.1	10	37.1 ± 0.2**	10	37.6 ± 0.1	10	37.4 ± 0.2	10
20	37.7 ± 0.2	10	37.2 ± 0.1	10	37.5 ± 0.2	10	37.9 ± 0.1	10
27	37.8 ± 0.1	10	37.4 ± 0.3	10	37.9 ± 0.2	10	38.0 ± 0.3	10
2-27	37.6 ± 0.1	10	37.2 ± 0.1**	10	37.7 ± 0.1	10	37.7 ± 0.1	10

* Significantly different (P≤0.05) from the sham control group by Williams' or Dunnett's test
** P≤0.01
[a] Temperatures are given as mean ± standard error.
[b] All temperatures were recorded within 5 minutes of the exposure cessation, except for the measurements on days 7 and 14, which were recorded at least 1 hour after exposure.
[c] Average of days 2 to 27, excluding days 7 and 14

There were no exposure-related effects on organ weights of males exposed to CDMA cell phone RFR (Table G3). The absolute kidney weight of 15 W/kg females was significantly less (12%) than that of the sham controls (Table G3); however, because there was no similar effect on relative kidney weight and no associated histopathologic findings, the biological significance of this finding was unknown.

There were no histopathologic lesions related to the effects of exposure to CDMA cell phone RFR.

Exposure Level Selection Rationale: In male and female mice exposed for 5 days to cell phone RFR up to 12 W/kg, only sporadic increases were observed in body temperature, regardless of the sex or age of the animals (Wyde *et al.*, 2018). Because no significant effects of cell phone RFR were observed in body temperature at 12 W/kg, a higher upper exposure level was selected for the 28-day studies. Due to limits on the maximum capacity of the exposure system to generate high RF fields, the maximum achievable exposure level capacity was 15 W/kg, which was selected as the highest exposure level for the 28-day studies. Selection of the highest exposure level for the 2-year studies was also limited by the power capacity of the exposure system to generate maximum RF fields. Based on the technical limitations and increased body temperatures at various time points that were similarly observed at 10 and 15 W/kg in the 28-day studies, the exposure levels selected for the 2-year studies were 2.5, 5, and 10 W/kg.

2-YEAR STUDY

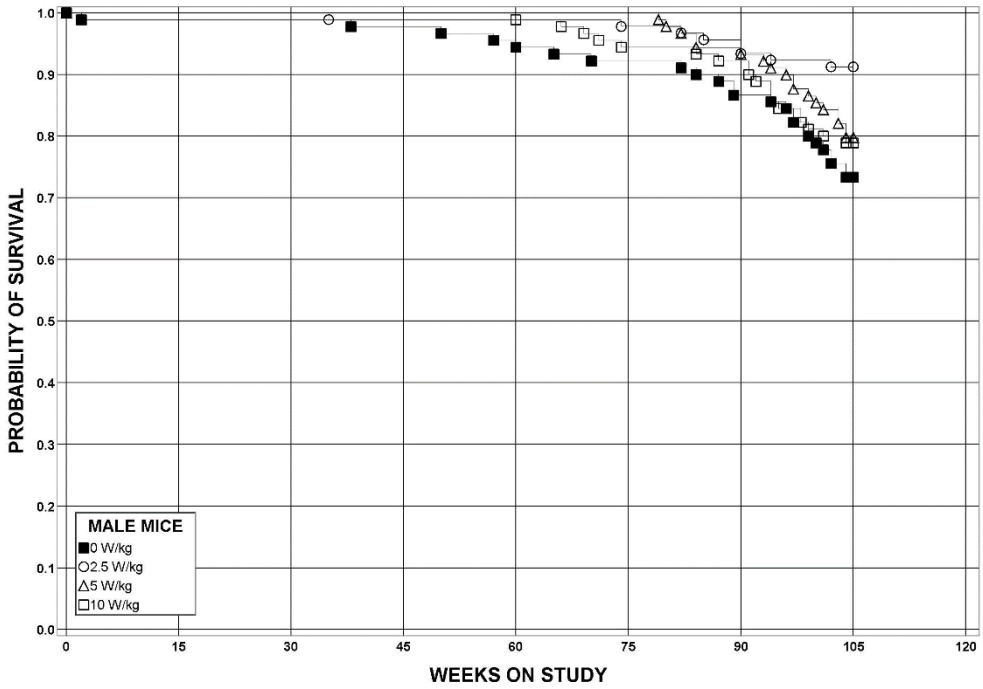
Survival

Estimates of 2-year survival probabilities for male and female mice are shown in Table 12 and in the Kaplan-Meier survival curves (Figure 8). Survival was significantly higher in 2.5 W/kg males compared to that in the sham controls. Survival of males and females in all other exposed groups was generally similar to that of the sham controls.

TABLE 12
Survival of Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Male				
Animals initially in study	105	106	105	105
14-week interim evaluation[a]	15	15	15	15
Accidental death[b]	0	0	1	0
Moribund	8	2	5	3
Natural deaths	16	6	13	16
Animals surviving to study termination	66	83	71	71
Percent probability of survival at end of study[c]	73	91	80	79
Mean survival (days)[d]	687	715	706	704
Survival analysis[e]	P=1.000	P=0.003N	P=0.343N	P=0.482N
Female				
Animals initially in study	105	104	105	105
14-week interim evaluation	15	15	15	15
Moribund	9	5	4	4
Natural deaths	14	9	16	14
Animals surviving to study termination	67[f]	75[g]	70[h]	72[h]
Percent probability of survival at end of study	74	84	77	79
Mean survival (days)	704	715	715	712
Survival analysis	P=0.758N	P=0.168N	P=0.702N	P=0.517N

[a] Excluded from survival analysis
[b] Censored in the survival analysis
[c] Kaplan-Meier determinations
[d] Mean of all deaths (uncensored, censored, and terminal euthanasia)
[e] The result of the life table trend test (Tarone, 1975) is in the sham control column, and the results of the life table pairwise comparisons (Cox, 1972) with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by **N**.
[f] Includes one animal that died during the last week of the study
[g] Includes three animals that died during the last week of the study; one of these was censored in the survival analysis
[h] Includes one animal that died during the last week of the study; this animal was censored in the survival analysis



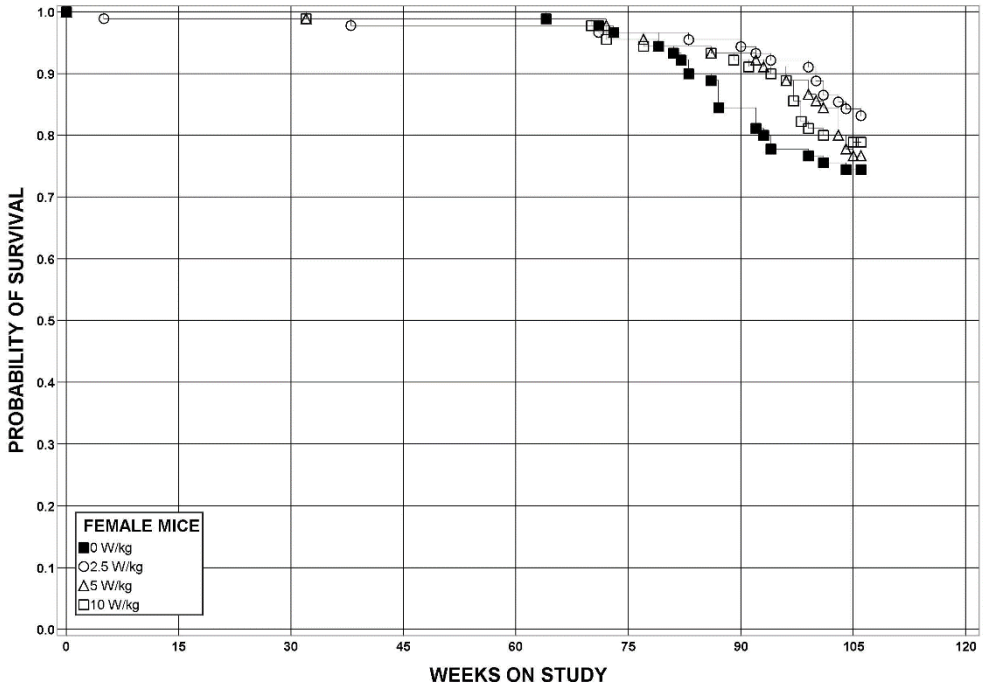


FIGURE 8
Kaplan-Meier Survival Curves for Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

Body Weights and Clinical Observations

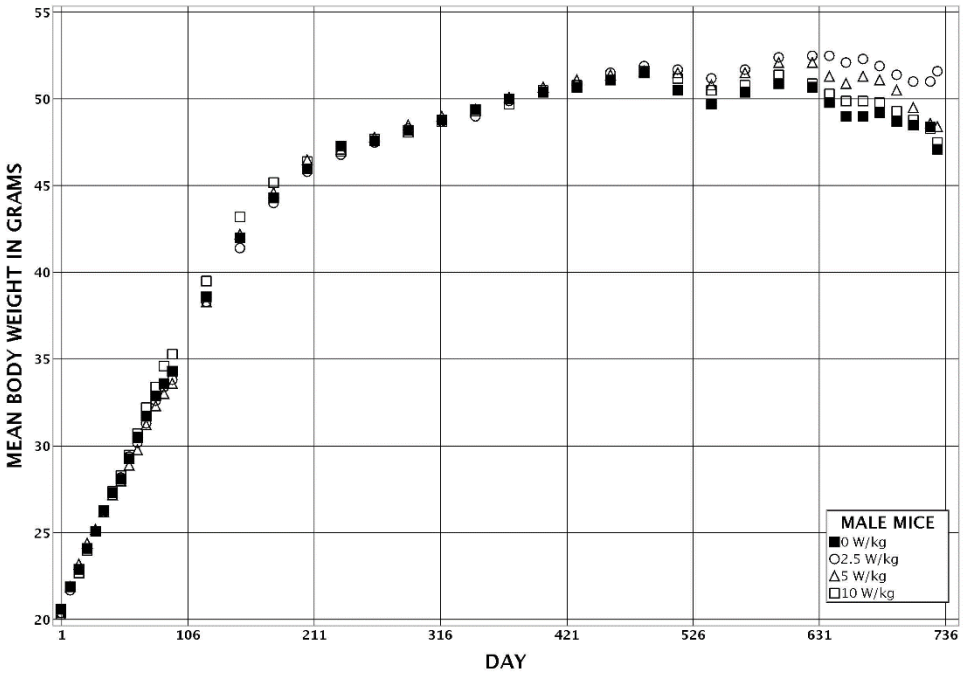
Mean body weights of exposed groups of males and females were similar to those of the sham controls throughout the study (Tables 13 and 14; Figure 9). In males, there were higher occurrences of the clinical signs mass-torso/lateral and mass-torso/ventral in the 10 W/kg group. In females, more occurrences of ruffled fur were recorded in the 5 and 10 W/kg groups and more occurrences of thin were recorded in all exposed groups. These findings were not correlated with differences in body weights or incidences of neoplasms in exposed animals.

TABLE 13
Mean Body Weights and Survival of Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control		2.5 W/kg			5 W/kg			10 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
0	20.6	105	20.4	99.1	106	20.3	99.0	105	20.4	99.3	105
8	21.9	104	21.7	99.0	106	21.9	100.0	105	21.9	100.2	105
15	22.9	104	22.9	99.9	106	23.2	101.3	105	22.7	99.2	105
22	24.1	104	24.1	100.0	106	24.4	101.2	105	24.0	99.6	105
29	25.1	104	25.1	99.9	106	25.2	100.3	105	25.1	99.8	105
36	26.3	104	26.2	99.9	106	26.2	100.0	104	26.3	100.1	105
43	27.3	104	27.3	99.8	106	27.2	99.7	104	27.4	100.3	105
50	28.1	104	28.2	100.5	106	28.0	99.8	104	28.3	100.8	105
57	29.3	104	29.4	100.2	106	28.9	98.5	104	29.5	100.5	105
64	30.5	104	30.2	99.1	106	29.8	97.8	104	30.7	100.5	105
71	31.7	104	31.3	98.9	106	31.2	98.3	104	32.2	101.7	105
79	32.9	104	32.6	99.0	106	32.3	98.1	104	33.4	101.6	105
86	33.6	104	33.4	99.2	106	33.0	98.0	104	34.6	102.8	105
93	34.3	94	33.8	98.5	96	33.6	98.0	94	35.3	102.8	95
121	38.6	89	38.3	99.0	91	38.3	99.1	89	39.5	102.4	90
149	42.0	89	41.4	98.6	91	42.2	100.6	89	43.2	103.0	90
177	44.3	89	44.0	99.4	91	44.6	100.7	89	45.2	102.0	90
205	46.0	89	45.8	99.6	91	46.5	101.0	89	46.4	101.0	90
233	47.3	89	46.8	99.1	91	47.1	99.6	89	47.0	99.4	90
261	47.6	89	47.5	99.8	90	47.8	100.5	89	47.7	100.3	90
289	48.2	88	48.3	100.2	90	48.5	100.5	89	48.1	99.8	90
317	48.8	88	48.7	99.7	90	49.0	100.4	89	48.7	99.8	90
345	49.4	88	49.0	99.1	90	49.4	100.1	89	49.3	99.7	90
373	50.0	87	49.9	99.9	90	50.1	100.1	89	49.7	99.4	90
401	50.4	86	50.4	99.8	90	50.7	100.5	89	50.5	100.2	90
429	50.7	85	50.8	100.1	90	51.1	100.8	89	50.8	100.1	89
457	51.1	84	51.5	100.7	90	51.4	100.5	89	51.1	100.1	89
485	51.5	84	51.9	100.8	90	51.6	100.3	89	51.6	100.3	87
513	50.5	83	51.7	102.4	90	51.5	102.0	89	51.2	101.3	86
541	49.7	83	51.2	103.0	89	50.8	102.3	89	50.5	101.7	85
569	50.4	82	51.7	102.7	88	51.5	102.3	87	50.8	101.0	85
597	50.9	81	52.4	103.0	87	52.1	102.4	84	51.4	101.1	84
625	50.7	78	52.5	103.4	86	52.1	102.7	84	50.9	100.3	83
639	49.8	78	52.5	105.5	85	51.3	103.1	83	50.3	101.0	80
653	49.0	78	52.1	106.4	85	50.9	103.8	82	49.9	101.8	79
667	49.0	76	52.3	106.6	84	51.3	104.6	80	49.9	101.7	76
681	49.2	74	51.9	105.6	84	51.1	104.0	78	49.8	101.3	75
695	48.7	71	51.4	105.5	84	50.5	103.8	76	49.3	101.2	73
709	48.5	69	51.0	105.2	84	49.5	102.0	75	48.8	100.5	72
723	48.4	67	51.0	105.5	83	48.6	100.5	73	48.3	99.9	71
Mean for Weeks											
1-13	27.3		27.1	99.6		27.0	99.4		27.4	100.5	
14-52	44.7		44.4	99.3		44.7	100.1		45.0	101.0	
53-105	49.9		51.5	103.3		50.9	102.1		50.3	100.8	

TABLE 14
Mean Body Weights and Survival of Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control		2.5 W/kg			5 W/kg			10 W/kg		
Day	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
0	17.4	105	17.4	99.7	104	17.4	100.2	105	17.5	100.4	105
8	18.4	105	18.4	100.0	104	18.5	100.5	105	18.4	100.2	105
15	19.4	105	19.6	100.7	104	19.5	100.3	105	19.3	99.0	105
22	20.2	105	20.3	100.5	104	20.2	99.7	105	20.1	99.6	105
29	20.8	105	21.0	100.8	104	20.8	99.8	105	20.7	99.6	105
36	21.5	105	21.6	100.4	103	21.5	99.9	105	21.5	99.8	105
43	22.0	105	22.0	100.0	103	21.9	99.7	105	21.8	99.4	105
50	22.5	105	22.4	99.4	103	22.3	99.0	105	22.0	97.8	105
57	22.8	105	22.7	99.8	103	22.9	100.3	105	22.6	99.1	105
64	23.3	105	23.4	100.2	103	23.4	100.4	105	23.2	99.4	105
71	23.4	105	23.7	101.2	103	23.9	102.2	105	23.8	101.6	105
79	23.9	105	24.4	101.8	103	24.7	103.1	105	24.5	102.4	105
86	24.0	105	24.1	100.5	103	24.6	102.6	105	24.5	102.2	105
93	24.3	95	24.1	99.2	93	24.9	102.4	95	24.8	102.2	95
121	26.3	90	26.4	100.2	88	26.9	102.2	90	27.0	102.5	90
149	28.8	90	29.5	102.3	88	29.9	103.9	90	30.2	104.8	90
177	30.8	90	32.2	104.6	88	32.3	105.0	90	33.0	107.1	90
205	33.4	90	35.6	106.5	88	35.1	105.2	90	35.8	107.1	90
233	36.8	90	38.2	103.7	88	38.2	103.9	89	39.1	106.3	89
261	38.4	90	40.4	105.1	88	40.8	106.1	89	42.1	109.5	89
289	40.3	90	43.6	108.1	87	43.3	107.4	89	44.1	109.4	89
317	42.3	90	45.6	108.0	87	46.0	108.9	89	46.8	110.8	89
345	45.0	90	48.0	106.7	87	48.7	108.3	89	49.0	109.0	89
373	47.6	90	50.4	105.8	87	50.7	106.4	89	51.0	107.0	89
401	49.9	90	52.2	104.5	87	52.6	105.3	89	52.7	105.6	89
429	51.4	90	53.5	104.2	87	54.3	105.8	89	53.6	104.3	89
457	53.3	89	55.2	103.7	87	55.6	104.5	89	54.8	102.9	89
485	55.0	89	56.2	102.3	87	56.8	103.3	89	56.0	102.0	88
513	54.5	87	55.9	102.6	86	56.8	104.3	87	56.7	104.1	86
541	51.9	87	54.0	104.1	86	54.7	105.4	86	54.1	104.4	85
569	52.2	83	54.2	103.8	86	55.2	105.7	85	54.5	104.5	85
597	55.3	80	56.8	102.7	85	57.5	104.0	84	56.7	102.5	84
625	56.3	76	56.9	101.1	85	57.7	102.6	84	57.1	101.5	83
639	54.8	75	55.8	101.9	83	56.2	102.5	84	55.7	101.6	82
653	54.5	71	55.3	101.4	83	55.5	101.8	82	55.7	102.1	81
667	55.1	70	55.3	100.5	82	55.1	100.1	81	55.0	99.9	81
681	54.6	70	54.6	99.9	82	54.7	100.1	80	53.9	98.6	77
695	54.0	69	54.0	99.9	80	53.6	99.1	77	53.7	99.3	73
709	53.7	68	53.2	99.1	77	53.3	99.3	76	53.2	99.0	72
723	53.0	68	52.3	98.7	75	53.4	100.7	72	52.7	99.4	72
737	52.2	67	51.3	98.3	75	53.2	101.9	69	52.2	99.9	71
Mean for Weeks											
1-13	22.4		22.6	100.5		22.6	100.7		22.5	100.1	
14-52	34.6		36.4	104.4		36.6	105.3		37.2	106.9	
53-107	53.3		54.3	101.9		54.8	102.9		54.4	102.1	



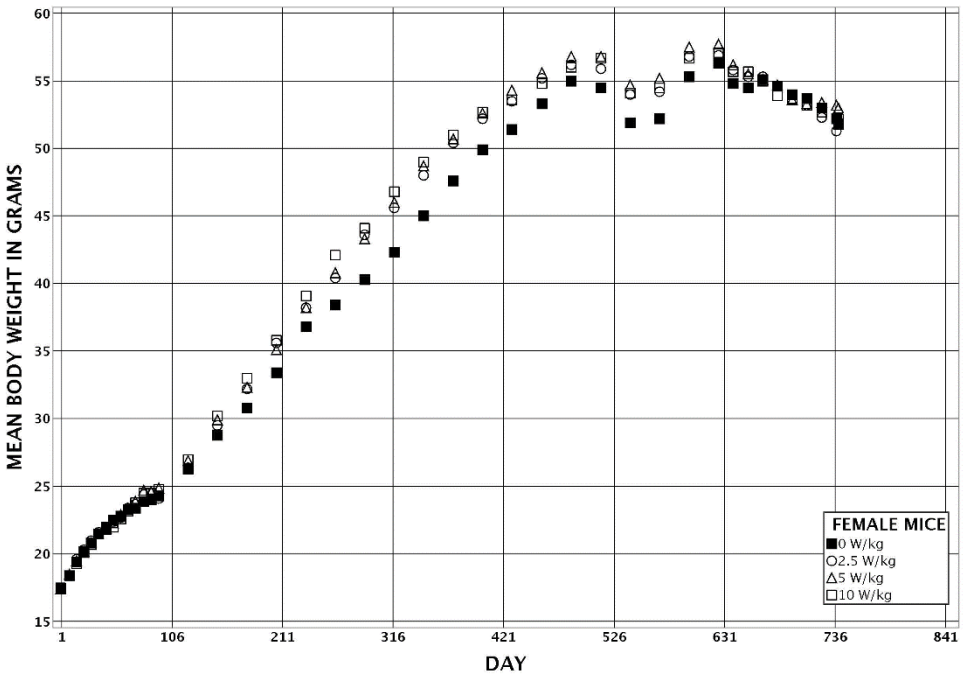


FIGURE 9
Growth Curves for Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

14-Week Interim Evaluation

There were no changes to the hematology variables attributable to CDMA cell phone RFR exposure (Table F2).

At the 14-week interim evaluation, mean body weights of exposed groups of males and females were similar to those of the sham controls (Table G4). The absolute right and left kidney weights were significantly lower (7% and 8%, respectively) in 5 W/kg males, and the absolute left kidney weight was significantly lower (8%) in 10 W/kg males (Table G4). The relative right and left kidney weights were significantly lower in 10 W/kg males. The absolute liver weight was significantly lower (10%) in 5 W/kg males, and the relative liver weight was significantly lower in 10 W/kg males. The changes in the liver weights were considered small and sporadic and therefore not toxicologically relevant; there were no histopathologic lesions that would account for changes in liver weights. Although the absolute thymus weight of 10 W/kg males was 22% higher than that of the sham controls, the relative thymus weight was not higher in the 10 W/kg males, nor were there any histopathologic lesions in the thymus. There were no significant changes in organ weights in females.

In males, there were no exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility (Table H4). In females, there were no exposure-related effects on estrous cyclicity (Tables H5 and H6; Figure H2). Compared to the sham controls, there were statistically significant differences for extended estrous in the 2.5 W/kg group and extended diestrus in the 5 W/kg group; however, these changes were considered sporadic due to the lack of an exposure-related response.

In the kidney of 10 W/kg females, there was a significantly higher incidence of interstitial lymphocytic cellular infiltration (sham control, 0/10; 2.5 W/kg, 1/10, 5 W/kg, 1/10; 10 W/kg, 5/10; Table D4). The lesions were minimal to mild in severity, and consisted of clusters of lymphocytes within the interstitium.

Pathology and Statistical Analyses

This section describes the statistically significant or biologically noteworthy changes in the incidences of malignant lymphoma and neoplasms and/or nonneoplastic lesions of the liver, pituitary gland, and uterus in the 2-year study. Summaries of the incidences of neoplasms and nonneoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix C for male mice and Appendix D for female mice.

Liver: There was a significantly higher incidence of hepatoblastoma in 5 W/kg males (Tables 15, C1, and C2). In 2.5 W/kg males, there was a significantly higher incidence of hepatocellular adenoma and a significantly lower incidence of hepatocellular carcinoma. When these neoplasms were combined (hepatocellular adenoma, hepatocellular carcinoma, or hepatoblastoma), there were no significant differences in the incidences between exposed and sham control groups of males. Hepatocellular adenomas were well-circumscribed lesions that compressed the surrounding liver parenchyma. Most were considerably larger than a hepatic lobule, and when located at the edge of the liver would usually cause an outward protrusion of the liver surface. They were made up of hepatocytes that lacked the normal architectural arrangement; while portal areas might be found near the edge of a hepatocellular adenoma, they were typically lacking within the center of the neoplasm. Most adenomas lacked cellular pleomorphism and contained few, if any, mitotic figures. Hepatocellular carcinomas were usually large lesions, typically larger than hepatocellular adenomas, and frequently contained areas of necrosis. They were often multinodular and compressive, and were composed of trabeculae of neoplastic hepatocytes that were arranged at least three cells wide (in contrast to normal hepatic trabeculae, which are a single hepatocyte wide). Cells within hepatocellular carcinomas had higher mitotic rates and more pleomorphism when compared to hepatocellular adenomas. Hepatoblastomas were composed of small cells with scant cytoplasm and hyperchromatic, oval nuclei, often arranged in nests and whorls. Hepatoblastomas frequently arose from within a hepatocellular adenoma or carcinoma; when this occurred, only the hepatoblastoma was recorded.

TABLE 15
Incidences of Neoplasms of the Liver in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Number Examined Microscopically	90	89	90	90
Hepatocellular Adenoma[a]				
Overall rate[b]	52/90 (58%)	66/89 (74%)	55/90 (61%)	62/90 (69%)
Adjusted rate[c]	62.3%	75.4%	64.9%	72.7%
Terminal rate[d]	45/66 (68%)	64/83 (77%)	51/71 (72%)	54/71 (76%)
First incidence (days)	393	625	656	478
Poly-3 test[e]	P=0.199	P=0.043	P=0.428	P=0.096
Hepatocellular Carcinoma[f]				
Overall rate	28/90 (31%)	18/89 (20%)	25/90 (28%)	31/90 (34%)
Adjusted rate	34.2%	20.6%	29.0%	36.2%
Terminal rate	18/66 (27%)	16/83 (19%)	18/71 (25%)	22/71 (31%)
First incidence (days)	608	629	559	461
Poly-3 test	P=0.177	P=0.033N	P=0.287N	P=0.459
Hepatoblastoma, Multiple[g]	0	0	1	0
Hepatoblastoma (includes multiple)[h]				
Overall rate	6/90 (7%)	6/89 (7%)	16/90 (18%)	7/90 (8%)
Adjusted rate	7.5%	6.9%	18.9%	8.5%
Terminal rate	5/66 (8%)	6/83 (7%)	14/71 (20%)	7/71 (10%)
First incidence (days)	711	729 (T)	679	729 (T)
Poly-3 test	P=0.328	P=0.562N	P=0.026	P=0.523
Hepatocellular Adenoma, Hepatocellular Carcinoma, or Hepatoblastoma[i]				
Overall rate	68/90 (76%)	70/89 (79%)	69/90 (77%)	75/90 (83%)
Adjusted rate	80.3%	79.6%	79.8%	85.6%
Terminal rate	52/66 (79%)	67/83 (81%)	59/71 (83%)	61/71 (86%)
First incidence (days)	393	625	559	461
Poly-3 test	P=0.175	P=0.532N	P=0.548N	P=0.230

(T) Terminal euthanasia
[a] Historical control incidence for 2-year studies (all routes) (mean ± standard deviation): 308/589 (51.9% ± 10.3%), range 34%-70%
[b] Number of animals with neoplasm per number of animals with liver examined microscopically
[c] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
[d] Observed incidence at terminal euthanasia
[e] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A lower incidence in an exposure group is indicated by **N**.
[f] Historical control incidence: 164/589 (27.6% ± 8.3%), range 16%-42%
[g] Number of animals with neoplasm
[h] Historical control incidence: 19/589 (3.0% ± 2.2%), range 0%-7%
[i] Historical control incidence: 408/589 (68.8% ± 8.6%), range 53%-80%

Malignant Lymphoma: Compared to the sham controls, the incidences of malignant lymphoma were higher in all exposed groups of females compared to the controls, and the increase in the 2.5 W/kg group was statistically significant (Tables 16, D1, and D2). This was similar to the pattern seen in females exposed to GSM cell phone RFR in that the incidences of malignant lymphoma in groups exposed to cell phone RFR (either CDMA or GSM)

TABLE 16
Incidences of Malignant Lymphoma in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Malignant Lymphoma[a]				
Overall rate[b]	2/90 (2%)	9/89 (10%)	6/90 (7%)	7/90 (8%)
Adjusted rate[c]	2.5%	10.7%	7.2%	8.4%
Terminal rate[d]	1/67 (2%)	8/74 (11%)	4/69 (6%)	4/71 (6%)
First incidence (days)	604	689	716	635
Poly-3 test[e]	P=0.220	P=0.035	P=0.152	P=0.094

[a] Historical control incidence for 2-year studies (all routes) (mean ± standard deviation): 89/590 (16.0% ± 8.3%), range 2%-36%
[b] Number of animals with neoplasm per number of animals necropsied
[c] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality
[d] Observed incidence at terminal euthanasia
[e] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia.

were similar and increasingly higher exposures did not have increasingly higher incidences. The incidence in the sham control group, shared by the GSM- and CDMA-modulated cell phone RFR studies, was at the low end of the range for malignant lymphoma in historical controls (Tables 16 and D3). All of the incidences in the exposed groups fell within the overall historical control range. Malignant lymphoma in the CDMA cell phone RFR-exposed groups was similar in appearance, and in the organs that were involved, to that observed in the sham controls and the GSM cell phone RFR-exposed groups.

Other Tissues: Several tissues had significantly increased incidences of lesions in one, or even two, exposed groups of males or females. Some of these lesions are common background lesions and were not considered toxicologically important; the incidences of others lacked a dose response and were considered sporadic occurrences and not related to treatment.

In 5 W/kg males, two adenomas (0/86, 0/84, 2/89, 0/83) and one carcinoma (0/86, 0/84, 1/89, 0/83) occurred in the pars distalis of the pituitary gland (Table C1); no neoplasms of the pituitary gland pars distalis occurred in the sham control group or in the other exposed groups of males, including those in the GSM study (Table A1). Only two

adenomas of the pituitary gland (pars distalis) have been recorded in the current (August 2017) historical control database of 576 male mice (all routes), and no carcinomas of the pars distalis have been recorded.

In the uterus of female mice, there were one or two occurrences of adenocarcinoma (sham control, 0/89; 2.5 W/kg, 2/89; 5 W/kg, 0/88; 10 W/kg, 1/90) or leiomyosarcoma (0/89, 1/89, 1/88, 2/90) in most of the exposed groups; these neoplasms did not occur in the sham control group (Table D1). Neither uterine adenocarcinomas nor leiomyosarcomas have been recorded in the current historical control database (0/590). These neoplasms were considered sporadic occurrences, and not related to exposure.

GENETIC TOXICOLOGY

Twenty tissue samples obtained from animals in the 14-week interim evaluation study were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, five tissues). Results are based on the standard 100-cell scoring approach in use at the time these data were collected; data obtained using a 150-cell scoring approach, recommended in a recently adopted international guideline for the *in vivo* comet assay, are noted here for the few instances where results differed between the two methods. The complete 100-cell and 150-cell data are presented in Appendix E data tables. Significant increases in DNA damage (percent tail DNA) were observed in cells of the frontal cortex of male mice exposed to both modulations, CDMA and GSM (Tables E1 and E2). Positive results were also obtained for male mouse frontal cortex (CDMA and GSM) (Table E3) using the 150-cell approach. Of note is the low percent comet tail DNA value in the frontal cortex of sham control mice. There is no appropriate historical control database to provide context for this response, but bonafide changes in DNA damage levels in a treatment group should remain constant relative the control value. No technical aspects of the study that may have influenced this control value independently of the treated group values (e.g., % agarose gel, duration of electrophoresis, electromagnetic field strength, slide position in the electrophoresis tank) were identified. Technical factors that influence control levels have not been shown to alter sensitivity to detect effects in treated groups (Recio *et al.*, 2012). No other tissues showed evidence of a treatment-related effect in male mice. In female mice exposed to the CDMA modulation, significant increases in DNA damage were seen in blood leukocytes using both scoring approaches (Tables E4 and E6). In female mouse liver samples exposed to either modulation, the mean percent comet tail DNA was elevated above the sham control for all exposures when evaluated using either scoring

approach. Results of the 100-cell scoring approach were judged to be negative (Tables E4 and E6); scoring 150 cells resulted in a negative call for GSM-exposed female mice (Table E5) but in CDMA-exposed female mouse liver, a significant increase (P=0.010) in percent comet tail DNA was seen in the 5 W/kg group, resulting in an equivocal call for this dataset (Table E6).

In the micronucleus assay for male mice exposed to CDMA (Table E7), although a significant trend was observed for micronucleated polychromatic erythrocytes (PCEs) (P=0.013), the absolute increase was quite small and fell within the laboratory's historical control range. In addition, no corresponding increase in micronucleated normochromatic erythrocytes was observed; the mature erythrocyte population ought to be in steady state equilibrium after continuous 14 weeks of exposure, such as occurred in this study. Thus, the overall result in the micronucleus assay for male mice exposed to CDMA was judged to be negative. No other significant effects on either micronucleus frequency or % PCEs were seen in male or female mice exposed to either modulation of cell phone RFR.

DISCUSSION AND CONCLUSIONS

The Food and Drug Administration (FDA) nominated the radio frequency radiation (RFR) emissions of wireless communication devices for toxicology and carcinogenicity testing based on several factors. Current exposure guidelines are based on protection from acute injury from thermal effects, and little is known about the potential for health effects of long-term exposure. Epidemiology and toxicology studies have not definitively demonstrated an association between cell phone RFR exposure and any specific health problems in humans; however, the results of these studies are mixed and further complicated by confounding factors (including potential recall biases of the study participants that could impact the assessment of exposure). For epidemiology studies, exposures in the general population may not have occurred for a long enough period of time to accommodate the long latency period for some types of cancers in humans. Studies in laboratory animals have been complicated by limitations that researchers have faced in conducting robust studies designed to characterize the toxicity and carcinogenicity of cell phone RFR.

To improve on the existing methods of exposing laboratory animals to RFR, the NTP worked in collaboration with experts from the Radio-Frequency Fields Group at the National Institute of Standards and Technology (NIST, Boulder, CO) and IT'IS Foundation (Zurich, Switzerland) to design, construct, and validate a novel system of delivering RFR exposure that improved on the designs of previous exposure systems. Together with NIST and the IT'IS Foundation, the NTP identified and constructed an exposure system designed to uniformly expose unrestrained, individually housed animals to a uniform field of cell phone RFR at frequencies and modulations that reflect those currently in use in wireless communication devices (GSM and CDMA). The exposure facility was installed at IIT Research Institute (Chicago, IL) where all animal studies were conducted following system testing and RFR exposure validation.

Studies were designed to evaluate the toxicology and carcinogenicity of whole-body exposure to cell phone RFR in individually housed, unrestrained animals. Studies for both GSM- and CDMA-modulated cell phone RFR were

conducted simultaneously with a common control group in a sham chamber. Exposures were conducted in 10 minute periods, followed by 10 minutes of rest with no cell phone RFR exposure. The exposure system ran continuously, alternating each 10 minute block of active exposure between the GSM- and CDMA-exposed mice over the course of approximately 18 hours a day, 7 days per week. Based on the on/off cycling scheme, the actual daily exposure time to cell phone RFR was approximately 9 hours per day.

Studies were conducted in multiple phases. The first phase comprised a series of short-term toxicity studies conducted in young and aged B6C3F1/N mice and Hsd:Harlan Sprague Dawley SD rats to characterize the effects of cell phone RFR exposure on body temperature and the potential impact of animal size. The impact of cell phone RFR exposure during pregnancy was also evaluated in rats. These studies demonstrated that rats were more sensitive to the heating effects of cell phone RFR than were the mice (Wyde *et al*., 2018). In both young and aged male and female mice, body temperatures were only sporadically increased at exposures to cell phone RFR up to 12 W/kg (GSM and CDMA). These data suggest that exposures of up to 12 W/kg did not markedly alter the thermoregulatory capacity in mice. It must be noted, however, that core body temperature is a general surrogate for the heating effects of RFR and that these results do not address the issue of potential changes in temperature that may occur in localized areas within some tissues.

The findings from these short-term studies were used to guide the selection of cell phone RFR exposure levels for the 28-day and 2-year studies. Because no significant effect of cell phone RFR exposure up to 12 W/kg was observed in the body temperature of mice in these thermal pilot studies, a higher level of cell phone RFR exposure (15 W/kg) was selected for the highest exposure group in the 28-day studies. The selection of 15 W/kg was determined by the technical limitations of the exposure system to deliver higher cell phone RFR fields in the 28-day studies. Results from the 28-day studies demonstrated some increases in core body temperature at various time points at 10 and 15 W/kg. Based on the observed increases in body temperature and the power limitations of the system to generate maximum RFR fields for the large numbers of mice that were required for the 2-year studies, the highest exposure level for the 2-year studies was 10 W/kg.

The effects of whole-body exposure to GSM- or CDMA-modulated cell phone RFR at 1,900 MHz for 14 weeks or 2 years were studied in B6C3F1/N mice at specific absorption rates (SARs) of 2.5, 5, and 10 W/kg, with a common sham control group for both GSM- and CDMA-modulated signals. At SAR exposures up to 10 W/kg, there were no exposure-related effects on survival or mean body weights in either modulation (GSM or CDMA).

In both the GSM and CDMA studies, the incidences of malignant lymphoma in all exposed female groups were higher than that in the sham controls. These incidences were significantly increased only in the GSM groups at 2.5 and 5 W/kg, and in the CDMA group at 2.5 W/kg compared to sham controls. The 2% incidence of lymphoma in the concurrent sham controls was the lowest incidence observed thus far in female B6C3F1/N mice. The incidence is well below the overall historical control mean of 16%, and appreciably lower than the lower end of the range of overall historical control values in other studies (10% to 36%). Additionally, the incidences of malignant lymphoma in all exposed groups were within the range observed in overall historical controls. These considerations reduce the confidence that these increases in incidences were attributable to the RFR exposure, so these were considered equivocal findings. In NTP conclusions, such uncertain responses in the absence of other clearer effects on carcinogenicity would be referred to as equivocal evidence of carcinogenicity (i.e., may have been related to exposure).

In males, there were no common lesions observed between the two modulations. Potential cell phone RFR-mediated effects observed in the lung and the skin of males were specific to the GSM modulation. In the lung, there was a positive trend in the combined incidence of alveolar/bronchiolar adenoma or carcinoma in male mice, but there was no significant effect in any of the individual groups compared to controls. The combined incidences at the upper two exposure levels exceeded the historical control range (16% to 34%). Despite a significant trend in the combined incidence of alveolar/bronchiolar adenoma or carcinoma, the observation that the incidences were only marginally outside the historical range reduce the confidence that the increased incidences were attributable to the RFR exposure. Therefore, these were considered equivocal findings.

The combined incidences of fibrosarcoma, sarcoma, or malignant fibrous histiocytoma in the skin were higher in the 5 and 10 W/kg GSM males but were not statistically different than that of the sham controls. Malignant fibrous

histiocytoma was the predominant neoplasm in this combination. There was also a lack of an increased exposure level response. However, the incidences in both groups were above the historical control range for malignant fibrous histiocytoma. Additionally, there was one occurrence of a sarcoma in the 2.5 W/kg GSM males and one occurrence of a fibrosarcoma in the 10 W/kg GSM males. While the incidences in the 5 and 10 W/kg GSM males were not significant versus the current sham controls, the increases were seen in the top two exposure groups and were outside the historical range. This suggests that the increases in incidences observed may have been attributable to the RFR exposure, so these were considered equivocal findings.

At 2 years in the CDMA study only, there was a significantly increased incidence of hepatoblastoma in males exposed to 5 W/kg. The incidence at 5 W/kg exceeded the historical control; however, no increases were observed in males at 10 W/kg. Additionally, when all liver neoplasms (hepatocellular adenoma, hepatocellular carcinoma, or hepatoblastoma) were combined, there were no significant differences between any of the exposed groups compared to the sham controls. The isolated increase in only the 5 W/kg group and overall lack of exposure response reduces the confidence that the increase in incidence of hepatoblastoma observed was attributable to the RFR exposure, therefore, this was considered an equivocal finding.

Subsets of male and female mice from the 2-year studies were examined at 14 weeks to evaluate biomarkers of genotoxicity. Chromosomal damage was evaluated using the peripheral blood erythrocyte micronucleus (MN) assay, and DNA damage was evaluated in the frontal cortex, hippocampus, cerebellum, liver, and peripheral blood using the comet assay. Results of the MN assays were negative, but significant increases in DNA damage were observed in cells of the frontal cortex of male mice exposed to both modulations (GSM and CDMA) and in blood leukocytes of female mice (CDMA only).

Unlike ionizing radiation or ultraviolet light, cell phone RFR is not sufficiently energetic, by several orders of magnitude, to directly damage macromolecules (IARC, 2013), and little is known about the mechanisms by which RFR could induce DNA damage in the absence of thermal effects. Proposed mechanisms include, for example, induction of oxygen radicals and interference with DNA repair mechanisms (Ruediger, 2009; Yakymenko *et al.*, 2016).

No histopathologic assessments of cytotoxicity (apoptosis and necrosis) were conducted in the male mouse brain tissues that were examined for DNA damage, which leaves open the possibility that apoptosis or necrosis may have confounded the comet assay results. However, this seems unlikely as brain sections from other groups of mice in this interim 14-week study and in the 2-year study did undergo histopathologic assessment and no significant evidence of cytotoxicity was observed.

Although increases in DNA damage were observed in the frontal cortex of male mice, there were no increases observed in the incidences of any type of neoplasm in the brain of males in the 2 year study. Similarly, while increased DNA damage was observed in blood leukocytes of female mice exposed to CDMA-modulated cell phone RFR, there were no increased incidences of related neoplasms. Therefore, no association was established between DNA damage appearing early in the studies and neoplasm development in these tissues.

CONCLUSIONS

Under the conditions of these 2-year studies, there was *equivocal evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 1,900 MHz in male B6C3F1/N mice based on the combined incidences of fibrosarcoma, sarcoma, or malignant fibrous histiocytoma in the skin and the incidences of alveolar/bronchiolar adenoma or carcinoma (combined) in the lung. There was *equivocal evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 1,900 MHz in female B6C3F1/N mice based on the incidences of malignant lymphoma (all organs). There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 1,900 MHz in male B6C3F1/N mice based on the incidences of hepatoblastoma of the liver. There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 1,900 MHz in female B6C3F1/N mice based on the incidences of malignant lymphoma (all organs).

Exposure to GSM- or CDMA-modulated cell phone RFR at 1,900 MHz did not increase the incidence of any nonneoplastic lesions in male or female B6C3F1/N mice.

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* Explanation of Levels of Evidence of Carcinogenic Activity is on page 14.

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APPENDIX A
SUMMARY OF LESIONS IN MALE MICE EXPOSED TO GSM-MODULATED CELL PHONE RFR FOR 2 YEARS

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death		1		
Moribund	8	6	2	6
Natural deaths	16	19	8	12
Survivors				
Died last week of study			1	4
Terminal euthanasia	66	63	79	68
Missing		1		
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Nervous System				
Brain	(10)	(10)	(10)	(10)
Hamartoma, lipomatous				1 (10%)
Systems Examined with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Respiratory System				
Special Senses System				
Urinary System				
2-Year Study				
Alimentary System				
Esophagus	(88)	(87)	(88)	(90)
Gallbladder	(73)	(66)	(74)	(79)
Intestine large, cecum	(81)	(77)	(84)	(78)
Leiomyoma			1 (1%)	
Intestine large, colon	(84)	(83)	(85)	(84)
Intestine large, rectum	(84)	(85)	(86)	(84)
Intestine small, duodenum	(77)	(77)	(83)	(79)
Adenocarcinoma	1 (1%)	1 (1%)		
Adenoma			1 (1%)	
Intestine small, ileum	(81)	(79)	(85)	(80)
Intestine small, jejunum	(79)	(79)	(82)	(79)
Adenocarcinoma	2 (3%)			1 (1%)
Hepatocellular carcinoma, metastatic, liver	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver		1 (1%)		

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Liver	(90)	(89)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Carcinoma, metastatic, islets, pancreatic				1 (1%)
Hemangioma			1 (1%)	
Hemangiosarcoma	1 (1%)	3 (3%)	2 (2%)	2 (2%)
Hepatoblastoma	6 (7%)	3 (3%)	8 (9%)	1 (1%)
Hepatoblastoma, multiple			1 (1%)	
Hepatocellular adenoma	25 (28%)	28 (31%)	20 (22%)	26 (29%)
Hepatocellular adenoma, multiple	27 (30%)	33 (37%)	46 (51%)	29 (32%)
Hepatocellular carcinoma	26 (29%)	23 (26%)	28 (31%)	19 (21%)
Hepatocellular carcinoma, multiple	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Hepatocholangiocarcinoma	1 (1%)	4 (4%)		
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Mesentery	(12)	(14)	(13)	(17)
Hemangiosarcoma	1 (8%)		1 (8%)	
Hepatocholangiocarcinoma, metastatic, liver		1 (7%)		
Malignant fibrous histiocytoma, metastatic, skin	1 (8%)			
Fat, hepatocholangiocarcinoma, metastatic, liver	1 (8%)	1 (7%)		
Fat, lipoma	1 (8%)			
Pancreas	(87)	(88)	(88)	(86)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)	2 (2%)		
Salivary glands	(90)	(89)	(89)	(89)
Stomach, forestomach	(88)	(87)	(89)	(87)
Squamous cell papilloma		1 (1%)	2 (2%)	
Stomach, glandular	(87)	(86)	(88)	(85)
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Tooth	(27)	(26)	(16)	(20)
Cardiovascular System				
Aorta	(89)	(89)	(89)	(87)
Alveolar/bronchiolar carcinoma, metastatic, lung	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver		1 (1%)		
Blood vessel	(1)	(0)	(0)	(0)
Heart	(90)	(89)	(90)	(90)
Alveolar/bronchiolar carcinoma, metastatic, lung	1 (1%)	1 (1%)		2 (2%)
Hemangiosarcoma		1 (1%)		1 (1%)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)	2 (2%)		

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(89)	(89)	(88)
Bilateral, malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Subcapsular, adenoma		3 (3%)	3 (3%)	
Adrenal medulla	(90)	(88)	(88)	(86)
Islets, pancreatic	(88)	(88)	(90)	(89)
Adenoma				2 (2%)
Adenoma, multiple		1 (1%)		
Carcinoma			1 (1%)	1 (1%)
Parathyroid gland	(68)	(68)	(67)	(66)
Pituitary gland	(86)	(85)	(87)	(85)
Thyroid gland	(89)	(88)	(88)	(88)
General Body System				
Peritoneum	(1)	(0)	(0)	(0)
Hepatocholangiocarcinoma, metastatic, liver	1 (100%)			
Tissue NOS	(0)	(0)	(0)	(1)
Genital System				
Coagulating gland	(2)	(2)	(0)	(4)
Epididymis	(90)	(89)	(90)	(90)
Hemangioma		1 (1%)		
Hepatocholangiocarcinoma, metastatic, liver		1 (1%)		
Preputial gland	(89)	(88)	(90)	(89)
Prostate	(90)	(87)	(90)	(87)
Seminal vesicle	(90)	(88)	(90)	(90)
Fibroma	1 (1%)			
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Testis	(90)	(88)	(90)	(90)
Hemangioma		1 (1%)		
Interstitial cell, adenoma	2 (2%)			
Hematopoietic System				
Bone marrow	(90)	(88)	(90)	(90)
Hemangiosarcoma			1 (1%)	1 (1%)
Lymph node	(6)	(8)	(7)	(9)
Sarcoma, metastatic, skin		1 (13%)		
Axillary, hepatocholangiocarcinoma, metastatic, liver	1 (17%)			
Lymph node, mandibular	(72)	(61)	(63)	(60)
Lymph node, mesenteric	(85)	(82)	(88)	(83)
Hemangioma	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver		1 (1%)		
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Spleen	(87)	(88)	(89)	(88)
Hemangiosarcoma		4 (5%)	1 (1%)	1 (1%)

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Thymus	(75)	(83)	(81)	(72)
Hepatocellular carcinoma, metastatic, liver		1 (1%)		
Hepatocholangiocarcinoma, metastatic, liver		3 (4%)		
Thymoma benign			1 (1%)	
Integumentary System				
Mammary gland	(2)	(5)	(2)	(8)
Skin	(90)	(89)	(90)	(90)
Keratoacanthoma			1 (1%)	
Pilomatrixoma	1 (1%)			
Sebaceous gland, adenoma			1 (1%)	
Subcutaneous tissue, fibrosarcoma				1 (1%)
Subcutaneous tissue, hemangioma				1 (1%)
Subcutaneous tissue, hemangiosarcoma	1 (1%)		2 (2%)	
Subcutaneous tissue, lipoma	1 (1%)			
Subcutaneous tissue, liposarcoma		1 (1%)		
Subcutaneous tissue, malignant fibrous histiocytoma	1 (1%)		4 (4%)	3 (3%)
Subcutaneous tissue, malignant fibrous histiocytoma, multiple			1 (1%)	
Subcutaneous tissue, sarcoma		1 (1%)		
Musculoskeletal System				
Bone	(90)	(88)	(90)	(90)
Hepatocholangiocarcinoma, metastatic, liver		1 (1%)		
Skeletal muscle	(90)	(89)	(90)	(90)
Hepatocellular carcinoma, metastatic, liver	1 (1%)		1 (1%)	
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)	2 (2%)		
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Sarcoma	1 (1%)			
Nervous System				
Brain	(90)	(89)	(90)	(90)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			
Brain trigeminal ganglion	(69)	(79)	(72)	(79)
Nerve trigeminal	(67)	(53)	(66)	(63)
Peripheral nerve, sciatic	(89)	(89)	(90)	(89)
Spinal cord	(90)	(89)	(90)	(90)

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(89)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Alveolar/bronchiolar adenoma	11 (12%)	13 (15%)	16 (18%)	15 (17%)
Alveolar/bronchiolar adenoma, multiple	2 (2%)		2 (2%)	1 (1%)
Alveolar/bronchiolar carcinoma	11 (12%)	12 (13%)	15 (17%)	17 (19%)
Alveolar/bronchiolar carcinoma, multiple	2 (2%)		1 (1%)	1 (1%)
Carcinoma, metastatic, islets, pancreatic				1 (1%)
Hepatoblastoma, metastatic, liver	1 (1%)	1 (1%)		1 (1%)
Hepatocellular carcinoma, metastatic, liver	11 (12%)	8 (9%)	6 (7%)	5 (6%)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)	3 (3%)		
Sarcoma, metastatic, skin		1 (1%)		
Mediastinum	(0)	(0)	(2)	(1)
Alveolar/bronchiolar carcinoma, metastatic, lung				1 (100%)
Hibernoma			2 (100%)	
Nose	(90)	(89)	(90)	(89)
Trachea	(90)	(89)	(89)	(90)
Special Senses System				
Eye	(90)	(89)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Harderian gland	(88)	(89)	(90)	(90)
Adenocarcinoma	3 (3%)	2 (2%)	1 (1%)	
Adenoma	6 (7%)	7 (8%)	11 (12%)	5 (6%)
Urinary System				
Kidney	(90)	(89)	(90)	(89)
Alveolar/bronchiolar carcinoma, metastatic, lung				1 (1%)
Hepatocellular carcinoma, metastatic, liver	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)	2 (2%)		
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Renal tubule, adenoma		1 (1%)	1 (1%)	
Urinary bladder	(87)	(88)	(90)	(89)
Hemangioma		2 (2%)		
Urothelium, papilloma				2 (2%)
Systemic Lesions				
Multiple organs[b]	(90)	(89)	(90)	(90)
Histiocytic sarcoma			1 (1%)	2 (2%)
Leukemia granulocytic				1 (1%)
Lymphoma malignant	6 (7%)	4 (4%)	3 (3%)	4 (4%)
Mast cell tumor	1 (1%)			

TABLE A1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms[c]				
14-Week interim evaluation				1
2-Year study	79	82	82	77
Total primary neoplasms				
14-Week interim evaluation				1
2-Year study	144	152	182	140
Total animals with benign neoplasms				
14-Week interim evaluation				1
2-Year study	61	67	77	61
Total benign neoplasms				
14-Week interim evaluation				1
2-Year study	77	91	109	81
Total animals with malignant neoplasms				
2-Year study	49	47	53	45
Total malignant neoplasms				
2-Year study	66	61	73	59
Total animals with metastatic neoplasms				
2-Year study	14	15	6	10
Total metastatic neoplasms				
2-Year study	34	37	7	12
Total animals with uncertain neoplasms-benign or malignant				
2-Year study	1			
Total uncertain neoplasms				
2-Year study	1			

[a] Number of animals examined microscopically at the site and the number of animals with neoplasm
[b] Number of animals with any tissue examined microscopically
[c] Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE A2
Statistical Analysis of Primary Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Harderian Gland: Adenoma				
Overall rate[a]	6/90 (7%)	7/89 (8%)	11/90 (12%)	5/90 (6%)
Adjusted rate[b]	7.5%	8.7%	12.7%	6.0%
Terminal rate[c]	6/66 (9%)	5/63 (8%)	11/80 (14%)	4/72 (6%)
First incidence (days)	729 (T)	672	729 (T)	689
Poly-3 test[d]	P=0.415N	P=0.506	P=0.194	P=0.470N
Harderian Gland: Adenoma or Carcinoma				
Overall rate	9/90 (10%)	9/89 (10%)	12/90 (13%)	5/90 (6%)
Adjusted rate	11.2%	11.1%	13.9%	6.0%
Terminal rate	8/66 (12%)	5/63 (8%)	12/80 (15%)	4/72 (6%)
First incidence (days)	690	651	729 (T)	689
Poly-3 test	P=0.160N	P=0.588N	P=0.386	P=0.179N
Liver: Hepatocellular Adenoma				
Overall rate	52/90 (58%)	61/89 (69%)	66/90 (73%)	55/90 (61%)
Adjusted rate	62.3%	73.8%	75.3%	64.7%
Terminal rate	45/66 (68%)	52/63 (83%)	61/80 (76%)	49/72 (68%)
First incidence (days)	393	533	605	614
Poly-3 test	P=0.526N	P=0.072	P=0.044	P=0.437
Liver: Hepatocellular Carcinoma				
Overall rate	28/90 (31%)	25/89 (28%)	30/90 (33%)	22/90 (24%)
Adjusted rate	34.2%	30.0%	34.1%	25.9%
Terminal rate	18/66 (27%)	15/63 (24%)	25/80 (31%)	17/72 (24%)
First incidence (days)	608	547	604	538
Poly-3 test	P=0.169N	P=0.340N	P=0.556N	P=0.157N
Liver: Hepatocellular Adenoma or Carcinoma				
Overall rate	67/90 (74%)	68/89 (76%)	74/90 (82%)	64/90 (71%)
Adjusted rate	79.1%	79.9%	83.4%	74.3%
Terminal rate	51/66 (77%)	52/63 (83%)	66/80 (83%)	54/72 (75%)
First incidence (days)	393	533	604	538
Poly-3 test	P=0.232N	P=0.526	P=0.296	P=0.281N
Liver: Hepatoblastoma				
Overall rate	6/90 (7%)	3/89 (3%)	9/90 (10%)	1/90 (1%)
Adjusted rate	7.5%	3.7%	10.4%	1.2%
Terminal rate	5/66 (8%)	3/63 (5%)	8/80 (10%)	1/72 (1%)
First incidence (days)	711	729 (T)	667	729 (T)
Poly-3 test	P=0.105N	P=0.244N	P=0.350	P=0.054N
Liver: Hepatocellular Carcinoma or Hepatoblastoma				
Overall rate	32/90 (36%)	27/89 (30%)	35/90 (39%)	23/90 (26%)
Adjusted rate	39.1%	32.4%	39.7%	27.1%
Terminal rate	22/66 (33%)	17/63 (27%)	29/80 (36%)	18/72 (25%)
First incidence (days)	608	547	604	538
Poly-3 test	P=0.089N	P=0.230N	P=0.534	P=0.067N
Liver: Hepatocellular Adenoma, Hepatocellular Carcinoma, or Hepatoblastoma				
Overall rate	68/90 (76%)	68/89 (76%)	74/90 (82%)	65/90 (72%)
Adjusted rate	80.3%	79.9%	83.4%	75.4%
Terminal rate	52/66 (79%)	52/63 (83%)	66/80 (83%)	55/72 (76%)
First incidence (days)	393	533	604	538
Poly-3 test	P=0.243N	P=0.553N	P=0.367	P=0.276N

TABLE A2
Statistical Analysis of Primary Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Lung: Alveolar/bronchiolar Adenoma				
Overall rate	13/90 (14%)	13/89 (15%)	18/90 (20%)	16/90 (18%)
Adjusted rate	16.0%	16.0%	20.7%	19.0%
Terminal rate	9/66 (14%)	10/63 (16%)	16/80 (20%)	14/72 (19%)
First incidence (days)	488	663	604	658
Poly-3 test	P=0.297	P=0.583	P=0.279	P=0.380
Lung: Alveolar/bronchiolar Carcinoma				
Overall rate	13/90 (14%)	12/89 (13%)	16/90 (18%)	18/90 (20%)
Adjusted rate	16.1%	14.7%	18.5%	21.2%
Terminal rate	12/66 (18%)	8/63 (13%)	16/80 (20%)	14/72 (19%)
First incidence (days)	568	594	729 (T)	614
Poly-3 test	P=0.165	P=0.488N	P=0.418	P=0.259
Lung: Alveolar/bronchiolar Adenoma or Carcinoma				
Overall rate	23/90 (26%)	24/89 (27%)	32/90 (36%)	34/90 (38%)
Adjusted rate	28.1%	29.2%	36.8%	39.9%
Terminal rate	18/66 (27%)	17/63 (27%)	30/80 (38%)	28/72 (39%)
First incidence (days)	488	594	604	614
Poly-3 test	P=0.040	P=0.506	P=0.149	P=0.074
Skin (Subcutaneous Tissue): Malignant Fibrous Histiocytoma				
Overall rate	1/90 (1%)	0/89 (0%)	5/90 (6%)	3/90 (3%)
Adjusted rate	1.2%	0.0%	5.8%	3.6%
Terminal rate	0/66 (0%)	0/63 (0%)	4/80 (5%)	3/72 (4%)
First incidence (days)	674	—[e]	654	729 (T)
Poly-3 test	P=0.127	P=0.499N	P=0.124	P=0.321
Skin (Subcutaneous Tissue): Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	1/90 (1%)	1/89 (1%)	5/90 (6%)	4/90 (4%)
Adjusted rate	1.2%	1.2%	5.8%	4.7%
Terminal rate	0/66 (0%)	0/63 (0%)	4/80 (5%)	3/72 (4%)
First incidence (days)	674	523	654	488
Poly-3 test	P=0.093	P=0.758N	P=0.124	P=0.197
Spleen: Hemangiosarcoma				
Overall rate	0/87 (0%)	4/88 (5%)	1/89 (1%)	1/88 (1%)
Adjusted rate	0.0%	5.0%	1.2%	1.2%
Terminal rate	0/66 (0%)	3/63 (5%)	1/80 (1%)	0/72 (0%)
First incidence (days)	—	672	729 (T)	681
Poly-3 test	P=0.538N	P=0.065	P=0.515	P=0.507
All Organs: Hemangiosarcoma				
Overall rate	2/90 (2%)	6/89 (7%)	6/90 (7%)	2/90 (2%)
Adjusted rate	2.5%	7.4%	6.9%	2.4%
Terminal rate	0/66 (0%)	4/63 (6%)	6/80 (8%)	1/72 (1%)
First incidence (days)	702	667	729 (T)	681
Poly-3 test	P=0.394N	P=0.141	P=0.163	P=0.677N
All Organs: Hemangioma or Hemangiosarcoma				
Overall rate	3/90 (3%)	10/89 (11%)	7/90 (8%)	3/90 (3%)
Adjusted rate	3.7%	12.3%	8.1%	3.6%
Terminal rate	1/66 (2%)	8/63 (13%)	7/80 (9%)	2/72 (3%)
First incidence (days)	702	667	729 (T)	681
Poly-3 test	P=0.277N	P=0.042	P=0.195	P=0.641N

TABLE A2
Statistical Analysis of Primary Neoplasms in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
All Organs: Malignant Lymphoma				
Overall rate	6/90 (7%)	4/89 (4%)	3/90 (3%)	4/90 (4%)
Adjusted rate	7.3%	4.9%	3.5%	4.8%
Terminal rate	4/66 (6%)	1/63 (2%)	3/80 (4%)	3/72 (4%)
First incidence (days)	263	609	729 (T)	690
Poly-3 test	P=0.307N	P=0.375N	P=0.222N	P=0.359N

(T) Terminal euthanasia

[a] Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for liver, lung, and spleen; for other tissues, denominator is number of animals necropsied.

[b] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

[c] Observed incidence at terminal euthanasia

[d] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A negative trend or a lower incidence in an exposure group is indicated by **N**.

[e] Not applicable; no neoplasms in animal group

TABLE A3a
Historical Incidence of Skin Neoplasms in Control Male B6C3F1/N Mice[a]

	Fibrous Histiocytoma	Malignant Fibrous Histiocytoma	Fibrosarcoma, Sarcoma, or All Fibrous Histiocytoma
Overall Historical Incidence: All Routes			
Total (%)	1/589 (0.2%)	1/589 (0.2%)	5/589 (0.9%)
Mean ± standard deviation	0.2% ± 0.6%	0.1% ± 0.3%	0.8% ± 1.0%
Range	0%-2%	0%-1%	0%-2%

[a] Data as of August 2017

TABLE A3b
Historical Incidence of Alveolar/bronchiolar Neoplasms in Control Male B6C3F1/N Mice[a]

	Adenoma	Carcinoma	Adenoma or Carcinoma
Overall Historical Incidence: All Routes			
Total (%)	84/589 (14.3%)	66/589 (11.2%)	142/589 (24.1%)
Mean ± standard deviation	14.3% ± 5.4%	11.0% ± 4.4%	24.0% ± 5.3%
Range	8%-24%	4%-20%	16%-34%

[a] Data as of August 2017

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death		1		
Moribund	8	6	2	6
Natural deaths	16	19	8	12
Survivors				
Died last week of study			1	4
Terminal euthanasia	66	63	79	68
Missing		1		
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Liver	(10)	(10)	(10)	(10)
Inflammation, focal		2 (20%)	4 (40%)	
Pancreas	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte		1 (10%)		
Inflammation, chronic		1 (10%)		
Genital System				
Prostate	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte			2 (20%)	1 (10%)
Hematopoietic System				
Lymph node, mandibular	(5)	(7)	(10)	(8)
Hemorrhage			2 (20%)	
Nervous System				
Brain	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)	1 (10%)		
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	1 (10%)			1 (10%)
Hemorrhage	2 (20%)	3 (30%)	2 (20%)	2 (20%)
Nose	(10)	(10)	(10)	(10)
Respiratory epithelium, hyperplasia		2 (20%)	1 (10%)	
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive	1 (10%)	2 (20%)	1 (10%)	
Interstitium, infiltration cellular, lymphocyte	2 (20%)	1 (10%)	1 (10%)	1 (10%)

[a] Number of animals examined microscopically at the site and the number of animals with lesion

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
14-Week Interim Evaluation (continued)				
Systems Examined with No Lesions Observed				
Cardiovascular System				
Endocrine System				
General Body System				
Integumentary System				
Musculoskeletal System				
Special Senses System				
2-Year Study				
Alimentary System				
Esophagus	(88)	(87)	(88)	(90)
Gallbladder	(73)	(66)	(74)	(79)
Inflammation, acute		1 (2%)		
Intestine large, cecum	(81)	(77)	(84)	(78)
Intestine large, colon	(84)	(83)	(85)	(84)
Intestine large, rectum	(84)	(85)	(86)	(84)
Intestine small, duodenum	(77)	(77)	(83)	(79)
Intestine small, ileum	(81)	(79)	(85)	(80)
Peyer's patch, hyperplasia, lymphoid	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Peyer's patch, infiltration cellular, plasma cell	1 (1%)			
Intestine small, jejunum	(79)	(79)	(82)	(79)
Inflammation, granulomatous	1 (1%)			
Epithelium, cyst	1 (1%)			
Peyer's patch, hyperplasia, lymphoid			1 (1%)	1 (1%)
Liver	(90)	(89)	(90)	(90)
Angiectasis			2 (2%)	
Basophilic focus	1 (1%)	2 (2%)	4 (4%)	3 (3%)
Clear cell focus	28 (31%)	34 (38%)	41 (46%)	31 (34%)
Eosinophilic focus	4 (4%)	4 (4%)	8 (9%)	1 (1%)
Extramedullary hematopoiesis	2 (2%)	2 (2%)	2 (2%)	
Fatty change	37 (41%)	31 (35%)	35 (39%)	35 (39%)
Fibrosis	1 (1%)			
Hemorrhage	1 (1%)			
Infiltration cellular, lymphocyte	2 (2%)			2 (2%)
Infiltration cellular, mixed cell	1 (1%)			
Inflammation, focal	1 (1%)	1 (1%)	3 (3%)	
Inflammation, chronic				2 (2%)
Inflammation, chronic active	2 (2%)		1 (1%)	1 (1%)
Mixed cell focus	2 (2%)	3 (3%)	7 (8%)	4 (4%)
Necrosis	6 (7%)	6 (7%)	4 (4%)	3 (3%)
Bile duct, cyst		2 (2%)	1 (1%)	
Hepatocyte, fatty change, focal		1 (1%)		2 (2%)
Mesentery	(12)	(14)	(13)	(17)
Artery, inflammation, chronic active		1 (7%)		2 (12%)
Fat, hemorrhage		1 (7%)		
Fat, inflammation, granulomatous				1 (6%)
Fat, mineral			1 (8%)	
Fat, necrosis	8 (67%)	11 (79%)	12 (92%)	13 (76%)

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Pancreas	(87)	(88)	(88)	(86)
Hemorrhage	1 (1%)			
Infiltration cellular, lymphocyte	3 (3%)	5 (6%)	3 (3%)	1 (1%)
Infiltration cellular, mixed cell				1 (1%)
Inflammation, granulomatous	1 (1%)			
Inflammation, acute				1 (1%)
Inflammation, chronic active				1 (1%)
Acinus, atrophy		1 (1%)		
Duct, cyst	1 (1%)	2 (2%)		
Duct, fibrosis	1 (1%)			
Salivary glands	(90)	(89)	(89)	(89)
Infiltration cellular, lymphocyte	58 (64%)	59 (66%)	65 (73%)	65 (73%)
Stomach, forestomach	(88)	(87)	(89)	(87)
Cyst, squamous			1 (1%)	3 (3%)
Hyperkeratosis		1 (1%)		2 (2%)
Infiltration cellular, lymphocyte			1 (1%)	
Inflammation, chronic			1 (1%)	
Epithelium, hyperplasia, focal	3 (3%)	2 (2%)	2 (2%)	
Epithelium, hyperplasia, diffuse		1 (1%)		1 (1%)
Stomach, glandular	(87)	(86)	(88)	(85)
Accumulation, hyaline droplet		2 (2%)		
Cyst				1 (1%)
Hemorrhage	1 (1%)			
Ulcer				1 (1%)
Epithelium, hyperplasia, focal	1 (1%)			
Tooth	(27)	(26)	(16)	(20)
Dysplasia	26 (96%)	26 (100%)	14 (88%)	20 (100%)
Inflammation, suppurative	2 (7%)		2 (13%)	
Inflammation, chronic active				1 (5%)
Cardiovascular System				
Aorta	(89)	(89)	(89)	(87)
Blood vessel	(1)	(0)	(0)	(0)
Inflammation, chronic	1 (100%)			
Heart	(90)	(89)	(90)	(90)
Bacteria	1 (1%)	2 (2%)		
Cardiomyopathy	10 (11%)	2 (2%)	1 (1%)	2 (2%)
Inflammation, acute	1 (1%)			
Inflammation, chronic active	2 (2%)	2 (2%)		1 (1%)
Thrombus	1 (1%)	2 (2%)		1 (1%)
Artery, inflammation, chronic active	1 (1%)	2 (2%)		3 (3%)
Endocardium, mineral	1 (1%)			
Endothelium, hyperplasia		1 (1%)		1 (1%)
Epicardium, inflammation, chronic	1 (1%)			
Epicardium, mineral	1 (1%)			
Myocardium, hemorrhage		1 (1%)		
Myocardium, mineral	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Myocardium, necrosis	1 (1%)	2 (2%)		

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(89)	(89)	(88)
Accessory adrenal cortical nodule	1 (1%)			
Angiectasis	1 (1%)			
Hyperplasia, focal	3 (3%)	1 (1%)	6 (7%)	6 (7%)
Hypertrophy, focal	2 (2%)	8 (9%)	9 (10%)	1 (1%)
Infiltration cellular, mononuclear cell				1 (1%)
Bilateral, hyperplasia, focal			1 (1%)	
Bilateral, hypertrophy, focal	1 (1%)	5 (6%)	4 (4%)	1 (1%)
Subcapsular, hyperplasia	69 (77%)	72 (81%)	80 (90%)	72 (82%)
Adrenal medulla	(90)	(88)	(88)	(86)
Islets, pancreatic	(88)	(88)	(90)	(89)
Atrophy				1 (1%)
Hyperplasia	18 (20%)	20 (23%)	16 (18%)	10 (11%)
Infiltration cellular, lymphocyte	2 (2%)	1 (1%)	2 (2%)	
Parathyroid gland	(68)	(68)	(67)	(66)
Cyst		2 (3%)	4 (6%)	1 (2%)
Pituitary gland	(86)	(85)	(87)	(85)
Pars distalis, angiectasis	1 (1%)			
Pars distalis, cyst	3 (3%)	4 (5%)	3 (3%)	4 (5%)
Pars distalis, hyperplasia, focal	1 (1%)	2 (2%)	1 (1%)	
Thyroid gland	(89)	(88)	(88)	(88)
Infiltration cellular, lymphocyte			1 (1%)	1 (1%)
General Body System				
Peritoneum	(1)	(0)	(0)	(0)
Tissues NOS	(0)	(0)	(0)	(1)
Genital System				
Coagulating gland	(2)	(2)	(0)	(4)
Cyst	2 (100%)	1 (50%)		3 (75%)
Bilateral, inflammation, chronic active		1 (50%)		
Epididymis	(90)	(89)	(90)	(90)
Granuloma sperm	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Infiltration cellular, lymphocyte	29 (32%)	17 (19%)	22 (24%)	28 (31%)
Spermatocele				1 (1%)
Bilateral, duct, atrophy				1 (1%)
Preputial gland	(89)	(88)	(90)	(89)
Infiltration cellular, lymphocyte	43 (48%)	32 (36%)	38 (42%)	33 (37%)
Inflammation, suppurative	1 (1%)			
Inflammation, chronic active	1 (1%)		1 (1%)	
Bilateral, hyperplasia			1 (1%)	
Bilateral, duct, dilation	6 (7%)	2 (2%)	9 (10%)	2 (2%)
Duct, dilation	10 (11%)	6 (7%)	11 (12%)	4 (4%)
Duct, inflammation, chronic active			1 (1%)	
Duct, necrosis	1 (1%)			
Prostate	(90)	(87)	(90)	(87)
Hyperplasia, focal				1 (1%)
Infiltration cellular, lymphocyte	4 (4%)	3 (3%)	6 (7%)	9 (10%)
Inflammation, acute		1 (1%)		5 (6%)
Inflammation, chronic active	1 (1%)		1 (1%)	

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Genital System (continued)				
Seminal vesicle	(90)	(88)	(90)	(90)
Dilation	4 (4%)	4 (5%)	5 (6%)	4 (4%)
Hyperplasia				1 (1%)
Inflammation, chronic active	1 (1%)	1 (1%)		
Bilateral, atrophy				1 (1%)
Bilateral, dilation	27 (30%)	26 (30%)	23 (26%)	29 (32%)
Bilateral, fibrosis				1 (1%)
Bilateral, inflammation, acute				1 (1%)
Bilateral, inflammation, chronic		1 (1%)	1 (1%)	
Bilateral, inflammation, chronic active				1 (1%)
Testis	(90)	(88)	(90)	(90)
Bilateral, germ cell, degeneration				1 (1%)
Germ cell, degeneration	2 (2%)	1 (1%)	1 (1%)	
Hematopoietic System				
Bone marrow	(90)	(88)	(90)	(90)
Hypercellularity	3 (3%)		2 (2%)	3 (3%)
Lymph node	(6)	(8)	(7)	(9)
Bronchial, infiltration cellular, mixed cell				1 (11%)
Iliac, erythrophagocytosis		1 (13%)		
Iliac, hemorrhage		1 (13%)		
Iliac, hyperplasia, lymphoid		1 (13%)		2 (22%)
Iliac, infiltration cellular, histiocyte		2 (25%)	2 (29%)	
Iliac, infiltration cellular, plasma cell				1 (11%)
Iliac, pigment			2 (29%)	
Lumbar, hemorrhage				1 (11%)
Mediastinal, hyperplasia, lymphoid				1 (11%)
Mediastinal, infiltration cellular, plasma cell		1 (13%)		
Pancreatic, hyperplasia, lymphoid			2 (29%)	
Renal, hemorrhage	1 (17%)		1 (14%)	
Renal, hyperplasia, lymphoid			1 (14%)	
Renal, infiltration cellular, mixed cell				1 (11%)
Lymph node, mandibular	(72)	(61)	(63)	(60)
Hemorrhage		1 (2%)		
Hyperplasia, lymphoid	2 (3%)			
Infiltration cellular, histiocyte	1 (1%)			1 (2%)
Lymph node, mesenteric	(85)	(82)	(88)	(83)
Erythrophagocytosis	1 (1%)	5 (6%)	4 (5%)	1 (1%)
Hemorrhage	10 (12%)	11 (13%)	7 (8%)	13 (16%)
Hyperplasia, lymphoid	4 (5%)	2 (2%)	2 (2%)	5 (6%)
Infiltration cellular, histiocyte	8 (9%)	7 (9%)	5 (6%)	4 (5%)
Infiltration cellular, mixed cell		2 (2%)		
Infiltration cellular, plasma cell	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Spleen	(87)	(88)	(89)	(88)
Extramedullary hematopoiesis	15 (17%)	15 (17%)	13 (15%)	12 (14%)
Hyperplasia, lymphoid	5 (6%)	2 (2%)	5 (6%)	3 (3%)
White pulp, atrophy			1 (1%)	
Thymus	(75)	(83)	(81)	(72)
Atrophy	11 (15%)	16 (19%)	4 (5%)	14 (19%)
Cyst	11 (15%)	16 (19%)	26 (32%)	15 (21%)
Hemorrhage	1 (1%)	1 (1%)	1 (1%)	
Infiltration cellular, histiocyte				1 (1%)

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Integumentary System				
Mammary gland	(2)	(5)	(2)	(8)
Skin	(90)	(89)	(90)	(90)
Cyst, squamous				1 (1%)
Hyperkeratosis		1 (1%)		
Infiltration cellular, mixed cell	1 (1%)			
Inflammation, chronic		1 (1%)		
Ulcer	2 (2%)	2 (2%)	2 (2%)	1 (1%)
Epidermis, hyperplasia, focal		1 (1%)		
Hair follicle, atrophy				2 (2%)
Subcutaneous tissue, inflammation, granulomatous			1 (1%)	
Musculoskeletal System				
Bone	(90)	(88)	(90)	(90)
Callus				1 (1%)
Increased bone			1 (1%)	
Skeletal muscle	(90)	(89)	(90)	(90)
Degeneration	1 (1%)			1 (1%)
Infiltration cellular, lymphocyte	3 (3%)		5 (6%)	5 (6%)
Inflammation, acute		1 (1%)		1 (1%)
Inflammation, chronic active				1 (1%)
Necrosis		1 (1%)		
Nervous System				
Brain	(90)	(89)	(90)	(90)
Hemorrhage	2 (2%)	2 (2%)		
Infiltration cellular, lymphocyte	1 (1%)			
Inflammation, acute		1 (1%)		
Mineral	79 (88%)	81 (91%)	80 (89%)	76 (84%)
Squamous cyst			1 (1%)	
Artery, meninges, inflammation, chronic active	1 (1%)			1 (1%)
Brain trigeminal ganglion	(69)	(79)	(72)	(79)
Nerve trigeminal	(67)	(53)	(66)	(63)
Peripheral nerve, sciatic	(89)	(89)	(90)	(89)
Axon, degeneration	9 (10%)	9 (10%)	9 (10)	4 (4%)
Spinal cord	(90)	(89)	(90)	(90)
Cyst, squamous			1 (1%)	
Degeneration		1 (1%)		
Hemorrhage		1 (1%)		
Necrosis	1 (1%)	1 (1%)		1 (1%)
Artery, meninges, inflammation, chronic active	1 (1%)		2 (2%)	1 (1%)

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(89)	(90)	(90)
Congestion	2 (2%)	2 (2%)	3 (3%)	3 (3%)
Hemorrhage	3 (3%)	5 (6%)	4 (4%)	3 (3%)
Infiltration cellular, histiocyte	6 (7%)	1 (1%)	1 (1%)	2 (2%)
Infiltration cellular, lymphocyte	1 (1%)		1 (1%)	
Infiltration, chronic active		1 (1%)		
Alveolar epithelium, hyperplasia, focal	6 (7%)	8 (9%)	8 (9%)	7 (8%)
Bronchiole, foreign body	1 (1%)			
Bronchiole, inflammation, suppurative	1 (1%)			
Mediastinum	(0)	(0)	(2)	(1)
Nose	(90)	(89)	(90)	(89)
Inflammation, acute	1 (1%)			
Respiratory epithelium, accumulation, hyaline droplet	1 (1%)			
Respiratory epithelium, hyperplasia	5 (6%)			
Vomeronasal organ, fibrosis	1 (1%)			
Trachea	(90)	(89)	(89)	(90)
Special Senses System				
Eye	(90)	(89)	(90)	(90)
Phthisis bulbi		1 (1%)	3 (3%)	1 (1%)
Cornea, fibrosis	1 (1%)			
Cornea, inflammation, chronic active				1 (1%)
Optic nerve, degeneration			1 (1%)	
Retina, atrophy				1 (1%)
Retina, degeneration			1 (1%)	
Harderian gland	(88)	(89)	(90)	(90)
Hemorrhage	1 (1%)			
Hyperplasia, focal	2 (2%)	1 (1%)	2 (2%)	1 (1%)
Infiltration cellular, lymphocyte	36 (41%)	36 (40%)	32 (36%)	40 (44%)
Urinary System				
Kidney	(90)	(89)	(90)	(89)
Bacteria		1 (1%)		
Infarct	7 (8%)	4 (4%)	4 (4%)	8 (9%)
Infiltration cellular, histiocyte		1 (1%)		
Infiltration cellular, mixed cell		1 (1%)		
Inflammation, suppurative	1 (1%)			
Inflammation, granulomatous	1 (1%)			
Inflammation, acute		1 (1%)		
Metaplasia, osseous	3 (3%)	6 (7%)	5 (6%)	1 (1%)
Mineral		2 (2%)		
Nephropathy, chronic progressive	74 (82%)	66 (74%)	76 (84%)	74 (83%)
Bilateral, bacteria		1 (1%)		
Bilateral, inflammation, acute		1 (1%)		1 (1%)
Bilateral, renal tubule, bacteria				1 (1%)
Bilateral, renal tubule, pigment		1 (1%)		1 (1%)
Glomerulus, cyst	1 (1%)			1 (1%)
Interstitium, infiltration cellular, lymphocyte	41 (46%)	50 (56%)	56 (62%)	44 (49%)
Pelvis, dilation	1 (1%)			1 (1%)

TABLE A4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Urinary System (continued)				
Kidney (continued)	(90)	(89)	(90)	(89)
Renal tubule, accumulation, hyaline droplet				1 (1%)
Renal tubule, bacteria		1 (1%)		
Renal tubule, cyst	8 (9%)	3 (3%)	4 (4%)	5 (6%)
Renal tubule, dilation				1 (1%)
Renal tubule, mineral	1 (1%)			4 (4%)
Urothelium, inflammation, chronic active	1 (1%)			
Urinary bladder	(87)	(88)	(90)	(89)
Hemorrhage	3 (3%)			
Infiltration cellular, lymphocyte	26 (30%)	20 (23%)	24 (27%)	21 (24%)
Inflammation, acute		1 (1%)		
Inflammation, chronic active		1 (1%)		
Transitional epithelium, hyperplasia, diffuse		1 (1%)		
Transitional epithelium, hyperplasia, multifocal		2 (2%)		

APPENDIX B
SUMMARY OF LESIONS IN FEMALE MICE EXPOSED TO GSM-MODULATED CELL PHONE RFR FOR 2 YEARS

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Moribund	9	9	9	6
Natural deaths	14	7	11	11
Survivors				
Died last week of study	1	4	2	1
Terminal euthanasia	66	70	68	72
Animals examined microscopically	100	100	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
2-Year Study				
Alimentary System				
Esophagus	(87)	(90)	(87)	(90)
Gallbladder	(79)	(75)	(74)	(72)
Intestine large, cecum	(84)	(82)	(83)	(82)
Fibrosarcoma, metastatic, skin	1 (1%)			
Leiomyosarcoma				1 (1%)
Intestine large, colon	(84)	(84)	(86)	(85)
Intestine large, rectum	(88)	(86)	(88)	(86)
Fibrosarcoma, metastatic, skin	1 (1%)			
Osteosarcoma, metastatic, skin	1 (1%)			
Intestine small, duodenum	(82)	(83)	(84)	(81)
Fibrosarcoma, metastatic, skin	1 (1%)			
Intestine small, ileum	(83)	(82)	(82)	(80)
Intestine small, jejunum	(84)	(81)	(81)	(80)
Adenoma		1 (1%)		
Liver	(89)	(90)	(90)	(89)
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangiosarcoma		1 (1%)		1 (1%)
Hepatoblastoma	1 (1%)			
Hepatocellular adenoma	14 (16%)	16 (18%)	11 (12%)	8 (9%)
Hepatocellular adenoma, multiple	5 (6%)	2 (2%)	2 (2%)	2 (2%)
Hepatocellular carcinoma	6 (7%)	6 (7%)	5 (6%)	5 (6%)
Hepatocellular carcinoma, multiple	2 (2%)		1 (1%)	1 (1%)
Hepatocholangiocarcinoma			1 (1%)	
Osteosarcoma, metastatic, bone	1 (1%)			
Osteosarcoma, metastatic, skin	1 (1%)			

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Mesentery	(29)	(24)	(32)	(30)
Fibrosarcoma, metastatic, skin	1 (3%)			
Renal mesenchymal tumor, metastatic, kidney	1 (3%)			
Fat, hemangioma	1 (3%)			
Fat, lipoma		1 (4%)		1 (3%)
Oral mucosa	(0)	(0)	(2)	(0)
Pancreas	(87)	(88)	(89)	(86)
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangioma				1 (1%)
Hepatocellular carcinoma, metastatic, liver			1 (1%)	
Renal mesenchymal tumor, metastatic, kidney	1 (1%)			
Acinus, carcinoma			1 (1%)	
Salivary glands	(89)	(89)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Stomach, forestomach	(86)	(89)	(90)	(85)
Fibrosarcoma, metastatic, skin	1 (1%)			
Squamous cell papilloma	1 (1%)			
Stomach, glandular	(85)	(87)	(85)	(85)
Fibrosarcoma, metastatic, skin	1 (1%)			
Tongue	(0)	(0)	(1)	(0)
Tooth	(0)	(0)	(1)	(0)
Cardiovascular System				
Aorta	(84)	(88)	(90)	(89)
Blood vessel	(0)	(0)	(2)	(0)
Heart	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Hemangioma			1 (1%)	
Osteosarcoma, metastatic, skin	1 (1%)			
Endocrine System				
Adrenal cortex	(84)	(88)	(90)	(90)
Adenoma	1 (1%)			
Adrenal medulla	(83)	(84)	(86)	(87)
Pheochromocytoma benign			1 (1%)	
Pheochromocytoma malignant	2 (2%)			
Islets, pancreatic	(87)	(88)	(90)	(86)
Adenoma			1 (1%)	1 (1%)
Carcinoma	1 (1%)	1 (1%)		
Parathyroid gland	(60)	(57)	(64)	(62)
Pituitary gland	(80)	(80)	(84)	(84)
Pars distalis, adenoma	6 (8%)	5 (6%)	7 (8%)	5 (6%)
Pars distalis, carcinoma			2 (2%)	1 (1%)
Thyroid gland	(86)	(89)	(86)	(86)
C-cell, carcinoma	1 (1%)			
Follicular cell, carcinoma			1 (1%)	

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
General Body System				
Peritoneum	(0)	(0)	(1)	(0)
Tissue NOS	(1)	(1)	(1)	(2)
Hemangiosarcoma				1 (50%)
Abdominal, osteosarcoma, metastatic, skin	1 (100%)			
Genital System				
Clitoral gland	(82)	(84)	(80)	(86)
Ovary	(75)	(86)	(82)	(80)
Cystadenoma	2 (3%)	2 (2%)	3 (4%)	6 (8%)
Granulosa cell tumor benign	1 (1%)		1 (1%)	1 (1%)
Hemangioma	2 (3%)	2 (2%)		
Hemangiosarcoma		1 (1%)		
Luteoma		1 (1%)		
Teratoma benign			2 (2%)	1 (1%)
Thecoma malignant	1 (1%)			
Oviduct	(0)	(0)	(1)	(0)
Uterus	(89)	(90)	(90)	(89)
Adenocarcinoma				1 (1%)
Fibroma	1 (1%)			
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangiosarcoma			1 (1%)	
Leiomyoma	1 (1%)			
Polyp stromal		3 (3%)	2 (2%)	2 (2%)
Vagina	(0)	(0)	(1)	(0)
Hematopoietic System				
Bone marrow	(90)	(89)	(89)	(90)
Hemangiosarcoma			2 (2%)	1 (1%)
Lymph node	(18)	(20)	(16)	(14)
Bronchial, alveolar/bronchiolar carcinoma, metastatic, lung	1 (6%)			
Bronchial, fibrosarcoma, metastatic, skin	1 (6%)			
Lymph node, mandibular	(76)	(77)	(81)	(83)
Lymph node, mesenteric	(71)	(84)	(80)	(83)
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangiosarcoma				1 (1%)
Renal mesenchymal tumor, metastatic, kidney	1 (1%)			
Spleen	(86)	(87)	(89)	(87)
Hemangiosarcoma		2 (2%)	2 (2%)	
Thymus	(85)	(80)	(84)	(86)

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Integumentary System				
Mammary gland	(85)	(88)	(88)	(84)
Adenocarcinoma			1 (1%)	1 (1%)
Skin	(90)	(90)	(90)	(90)
Sebaceous gland, adenoma		1 (1%)		
Subcutaneous tissue, fibroma	1 (1%)			
Subcutaneous tissue, fibrosarcoma	3 (3%)		3 (3%)	
Subcutaneous tissue, hemangiosarcoma	2 (2%)			
Subcutaneous tissue, lipoma	1 (1%)			
Subcutaneous tissue, malignant fibrous histiocytoma		1 (1%)	3 (3%)	
Subcutaneous tissue, osteosarcoma	1 (1%)			
Subcutaneous tissue, sarcoma	2 (2%)		1 (1%)	1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(89)	(90)
Hemangioma	1 (1%)			
Hemangiosarcoma		1 (1%)		
Osteosarcoma	1 (1%)			
Skeletal muscle	(89)	(90)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Osteosarcoma	1 (1%)			1 (1%)
Rhabdomyosarcoma				1 (1%)
Sarcoma, metastatic, skin			1 (1%)	
Nervous System				
Brain	(87)	(90)	(90)	(90)
Carcinoma, metastatic, pituitary gland			2 (2%)	1 (1%)
Meningioma benign		1 (1%)		
Osteosarcoma, metastatic, skeletal muscle	1 (1%)			
Brain trigeminal ganglion	(75)	(74)	(80)	(79)
Nerve trigeminal	(56)	(58)	(53)	(35)
Carcinoma, metastatic, pituitary gland				1 (3%)
Peripheral nerve	(0)	(0)	(1)	(0)
Peripheral nerve, sciatic	(88)	(87)	(88)	(88)
Spinal cord	(90)	(90)	(90)	(90)

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Larynx	(0)	(0)	(2)	(0)
Lung	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Alveolar/bronchiolar adenoma	3 (3%)	5 (6%)	7 (8%)	1 (1%)
Alveolar/bronchiolar adenoma, multiple		1 (1%)		
Alveolar/bronchiolar carcinoma	3 (3%)	1 (1%)		1 (1%)
Carcinoma, metastatic, pancreas			1 (1%)	
Carcinoma, metastatic, thyroid gland	1 (1%)			
Fibrosarcoma, metastatic, skin	1 (1%)		2 (2%)	
Hepatocellular carcinoma, metastatic, liver	2 (2%)		2 (2%)	1 (1%)
Hepatocholangiocarcinoma, metastatic, liver			1 (1%)	
Osteosarcoma, metastatic, bone	1 (1%)			
Osteosarcoma, metastatic, skeletal muscle	1 (1%)			
Osteosarcoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skin			1 (1%)	
Mediastinum	(2)	(0)	(0)	(0)
Hepatocellular carcinoma, metastatic, liver	1 (50%)			
Nose	(89)	(90)	(90)	(90)
Adenocarcinoma, metastatic, Harderian gland		1 (1%)		
Pleura	(0)	(0)	(1)	(0)
Trachea	(90)	(89)	(90)	(90)
Special Senses System				
Ear	(0)	(0)	(1)	(0)
Eye	(89)	(88)	(90)	(90)
Harderian gland	(89)	(90)	(90)	(87)
Adenocarcinoma		1 (1%)	1 (1%)	
Adenoma	4 (4%)	7 (8%)	5 (6%)	6 (7%)
Lacrimal gland	(0)	(1)	(2)	(0)
Zymbal's gland	(0)	(0)	(1)	(0)
Urinary System				
Kidney	(89)	(87)	(89)	(88)
Renal mesenchymal tumor	1 (1%)			
Renal tubule, adenoma	2 (2%)			
Ureter	(0)	(0)	(1)	(0)
Urethra	(0)	(0)	(1)	(0)
Urinary bladder	(86)	(87)	(86)	(86)
Systemic Lesions				
Multiple organs[b]	(90)	(90)	(90)	(90)
Histiocytic sarcoma	8 (9%)	2 (2%)	8 (9%)	5 (6%)
Leukemia erythrocytic		1 (1%)		
Lymphoma malignant	2 (2%)	13 (14%)	9 (10%)	6 (7%)

TABLE B1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms[c]				
2-Year study	59	55	57	44
Total primary neoplasms				
2-Year study	85	79	85	64
Total animals with benign neoplasms				
2-Year study	36	37	35	29
Total benign neoplasms				
2-Year study	47	48	43	35
Total animals with malignant neoplasms				
2-Year study	33	27	35	24
Total malignant neoplasms				
2-Year study	38	31	42	29
Total animals with metastatic neoplasms				
2-Year study	9	1	9	2
Total metastatic neoplasms				
2-Year study	29	5	11	3

[a] Number of animals examined microscopically at the site and the number of animals with neoplasm
[b] Number of animals with any tissue examined microscopically
[c] Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE B2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Harderian Gland: Adenoma				
Overall rate[a]	4/90 (4%)	7/90 (8%)	5/90 (6%)	6/90 (7%)
Adjusted rate[b]	5.0%	8.3%	6.0%	7.2%
Terminal rate[c]	4/67 (6%)	4/72 (6%)	5/69 (7%)	6/72 (8%)
First incidence (days)	739 (T)	562	739 (T)	739 (T)
Poly-3 test[d]	P=0.436	P=0.299	P=0.524	P=0.398
Harderian Gland: Adenoma or Carcinoma				
Overall rate	4/90 (4%)	8/90 (9%)	6/90 (7%)	6/90 (7%)
Adjusted rate	5.0%	9.4%	7.2%	7.2%
Terminal rate	4/67 (6%)	4/72 (6%)	6/69 (9%)	6/72 (8%)
First incidence (days)	739 (T)	562	739 (T)	739 (T)
Poly-3 test	P=0.471	P=0.214	P=0.397	P=0.398
Liver: Hepatocellular Adenoma				
Overall rate	19/89 (21%)[e]	18/90 (20%)	13/90 (14%)	10/89 (11%)
Adjusted rate	23.6%	21.5%	15.6%	12.0%
Terminal rate	17/67 (25%)	17/72 (24%)	11/69 (16%)	9/72 (13%)
First incidence (days)	511	638	674	700
Poly-3 test	P=0.022N	P=0.448N	P=0.134N	P=0.041N
Liver: Hepatocellular Carcinoma				
Overall rate	8/89 (9%)	6/90 (7%)	6/90 (7%)	6/89 (7%)
Adjusted rate	10.0%	7.2%	7.2%	7.2%
Terminal rate	7/67 (10%)	4/72 (6%)	5/69 (7%)	4/72 (6%)
First incidence (days)	656	650	701	720
Poly-3 test	P=0.348N	P=0.354N	P=0.358N	P=0.361N
Liver: Hepatocellular Adenoma or Carcinoma				
Overall rate	25/89 (28%)	24/90 (27%)	17/90 (19%)	15/89 (17%)
Adjusted rate	30.9%	28.5%	20.3%	18.0%
Terminal rate	22/67 (33%)	21.72 (29%)	14/69 (20%)	12/72 (17%)
First incidence (days)	511	638	674	700
Poly-3 test	P=0.020N	P=0.436N	P=0.082N	P=0.040N
Liver: Hepatocellular Carcinoma or Hepatoblastoma				
Overall rate	9/89 (10%)	6/90 (7%)	6/90 (7%)	6/89 (7%)
Adjusted rate	11.3%	7.2%	7.2%	7.2%
Terminal rate	8/67 (12%)	4/72 (6%)	5/69 (7%)	4/72 (6%)
First incidence (days)	656	650	701	720
Poly-3 test	P=0.268N	P=0.261N	P=0.265N	P=0.267N
Lung: Alveolar/bronchiolar Adenoma				
Overall rate	3/90 (3%)	6/90 (7%)	7/90 (8%)	1/90 (1%)
Adjusted rate	3.8%	7.2%	8.4%	1.2%
Terminal rate	3/67 (5%)	6/72 (8%)	7/69 (10%)	1/72 (1%)
First incidence (days)	739 (T)	739 (T)	739 (T)	739 (T)
Poly-3 test	P=0.190N	P=0.268	P=0.180	P=0.292N
Lung: Alveolar/bronchiolar Adenoma or Carcinoma				
Overall rate	6/90 (7%)	6/90 (7%)	7/90 (8%)	2/90 (2%)
Adjusted rate	7.5%	7.2%	8.4%	2.4%
Terminal rate	5/67 (8%)	6/72 (8%)	7/69 (10%)	2/72 (3%)
First incidence (days)	607	739 (T)	739 (T)	739 (T)
Poly-3 test	P=0.108N	P=0.592N	P=0.526	P=0.127N

TABLE B2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Ovary: Cystadenoma				
Overall rate	2/75 (3%)	2/86 (2%)	3/82 (4%)	6/80 (8%)
Adjusted rate	3.0%	2.5%	3.9%	7.9%
Terminal rate	2/56 (4%)	2/69 (3%)	3/65 (5%)	6/67 (9%)
First incidence (days)	739 (T)	739 (T)	739 (T)	739 (T)
Poly-3 test	P=0.067	P=0.623N	P=0.564	P=0.186
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	6/80 (8%)	5/80 (6%)	7/84 (8%)	5/84 (6%)
Adjusted rate	8.4%	6.8%	9.0%	6.4%
Terminal rate	5/60 (8%)	5/65 (8%)	4/64 (6%)	5/68 (7%)
First incidence (days)	703	739 (T)	712	739 (T)
Poly-3 test	P=0.417N	P=0.475N	P=0.563	P=0.435N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	6/80 (8%)	5/80 (6%)	9/84 (11%)	6/84 (7%)
Adjusted rate	8.4%	6.8%	11.5%	7.6%
Terminal rate	5/60 (8%)	5/65 (8%)	4/64 (6%)	5/68 (7%)
First incidence (days)	703	739 (T)	606	676
Poly-3 test	P=0.553	P=0.475N	P=0.362	P=0.549N
Skin (Subcutaneous Tissue): Fibrosarcoma or Sarcoma				
Overall rate	5/90 (6%)	0/90 (0%)	4/90 (4%)	1/90 (1%)
Adjusted rate	6.2%	0.0%	4.8%	1.2%
Terminal rate	1/67 (2%)	0/72 (0%)	1/69 (1%)	0/72 (0%)
First incidence (days)	607	—[f]	646	607
Poly-3 test	P=0.159N	P=0.031N	P=0.478N	P=0.098N
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, or Sarcoma				
Overall rate	6/90 (7%)	0/90 (0%)	4/90 (4%)	1/90 (1%)
Adjusted rate	7.4%	0.0%	4.8%	1.2%
Terminal rate	2/67 (3%)	0/72 (0%)	1/69 (1%)	0/72 (0%)
First incidence (days)	607	—	646	607
Poly-3 test	P=0.094N	P=0.016N	P=0.351N	P=0.055N
Skin (Subcutaneous Tissue): Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	1/90 (1%)	7/90 (8%)	1/90 (1%)
Adjusted rate	6.2%	1.2%	8.4%	1.2%
Terminal rate	1/67 (2%)	0/72 (0%)	3/69 (4%)	0/72 (0%)
First incidence (days)	607	562	646	607
Poly-3 test	P=0.193N	P=0.097N	P=0.407	P=0.098N
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	6/90 (7%)	1/90 (1%)	7/90 (8%)	1/90 (1%)
Adjusted rate	7.4%	1.2%	8.4%	1.2%
Terminal rate	2/67 (3%)	0/72 (0%)	3/69 (4%)	0/72 (0%)
First incidence (days)	607	562	646	607
Poly-3 test	P=0.125N	P=0.054N	P=0.527	P=0.055N
All Organs: Hemangiosarcoma				
Overall rate	2/90 (2%)	5/90 (6%)	3/90 (3%)	3/90 (3%)
Adjusted rate	2.5%	6.0%	3.6%	3.6%
Terminal rate	1/67 (2%)	4/72 (6%)	1/69 (1%)	2/72 (3%)
First incidence (days)	703	638	629	720
Poly-3 test	P=0.572N	P=0.238	P=0.521	P=0.518

TABLE B2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
All Organs: Hemangioma or Hemangiosarcoma				
Overall rate	6/90 (7%)	7/90 (8%)	4/90 (4%)	4/90 (4%)
Adjusted rate	7.5%	8.4%	4.8%	4.8%
Terminal rate	5/97 (8%)	6/72 (8%)	1/69 (1%)	3/72 (4%)
First incidence (days)	703	638	629	720
Poly-3 test	P=0.218N	P=0.533	P=0.343N	P=0.348N
All Organs: Histiocytic Sarcoma				
Overall rate	8/90 (9%)	2/90 (2%)	8/90 (9%)	5/90 (6%)
Adjusted rate	9.7%	2.4%	9.5%	5.9%
Terminal rate	2/67 (3%)	1/72 (1%)	3/69 (4%)	1/72 (1%)
First incidence (days)	562	458	629	660
Poly-3 test	P=0.419N	P=0.048N	P=0.587N	P=0.270N
All Organs: Malignant Lymphoma				
Overall rate	2/90 (2%)	13/90 (14%)	9/90 (10%)	6/90 (7%)
Adjusted rate	2.5%	15.6%	10.7%	7.1%
Terminal rate	1/67 (1%)	12/72 (17%)	5/69 (7%)	3/72 (4%)
First incidence (days)	604	731	516	590
Poly-3 test	P=0.474	P=0.004	P=0.035	P=0.153

(T) Terminal euthanasia

[a] Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for liver, lung, ovary, and pituitary gland; for other tissues, denominator is number of animals necropsied.

[b] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

[c] Observed incidence at terminal euthanasia

[d] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A negative trend or a lower incidence in an exposure group is indicated by **N**.

[e] A single incidence of hepatoblastoma occurred in an animal that also had an adenoma.

[f] Not applicable; no neoplasms in animal group

TABLE B3
Historical Incidence of Malignant Lymphoma in Control Female B6C3F1/N Mice[a]

	Incidence in Controls
Overall Historical Incidence: All Routes	
Total (%)	89/590 (15.1%)
Mean ± standard deviation	16.0% ± 8.3%
Range	2%-36%

[a] Data as of August 2017; includes data for histiocytic, lymphocytic, mixed, unspecified, or undifferentiated cell types

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Moribund	9	9	9	6
Natural deaths	14	7	11	11
Survivors				
Died last week of study	1	4	2	1
Terminal euthanasia	66	70	68	72
Animals examined microscopically	100	100	100	100
14-Week Interim Evaluation				
Alimentary System				
Liver	(10)	(10)	(10)	(9)
Inflammation, focal	1 (10%)	2 (20%)	4 (40%)	3 (33%)
Necrosis		1 (10%)		
Salivary glands	(10)	(10)	(10)	(9)
Infiltration cellular, lymphocyte			3 (30%)	
Stomach, glandular	(10)	(10)	(10)	(9)
Infiltration cellular, mixed cell		1 (10)%		
Endocrine System				
Thyroid gland	(10)	(10)	(10)	(9)
Infiltration cellular, lymphocyte	1 (10%)			
Hematopoietic System				
Thymus	(10)	(10)	(10)	(9)
Hemorrhage		2 (20%)	3 (30%)	
Integumentary System				
Skin	(10)	(10)	(10)	(9)
Hair follicle, inflammation, chronic active				1 (11%)
Nervous System				
Spinal cord	(10)	(10)	(10)	(10)
Cyst, squamous, multiple				1 (10%)
Respiratory System				
Lung	(10)	(10)	(10)	(9)
Hemorrhage		1 (10%)		
Special Senses System				
Harderian gland	(10)	(10)	(10)	(9)
Infiltration cellular, lymphocyte				1 (11%)

[a] Number of animals examined microscopically at the site and the number of animals with lesion

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
14-Week Interim Evaluation (continued)				
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive		1 (10%)		
Interstitium, infiltration cellular, lymphocyte			3 (30%)	
Urinary bladder	(10)	(10)	(10)	(9)
Infiltration cellular, lymphocyte				1 (11%)
Systems Examined with No Lesions Observed				
Cardiovascular System				
General Body System				
Genital System				
Musculoskeletal System				
2-Year Study				
Alimentary System				
Esophagus	(87)	(90)	(87)	(90)
Gallbladder	(79)	(75)	(74)	(72)
Cyst		1 (1%)	1 (1%)	
Infiltration cellular, lymphocyte	2 (3%)	5 (7%)	2 (3%)	4 (6%)
Intestine large, cecum	(84)	(82)	(83)	(82)
Intestine large, colon	(84)	(84)	(86)	(85)
Intestine large, rectum	(88)	(86)	(88)	(86)
Intestine small, duodenum	(82)	(83)	(84)	(81)
Inflammation, acute	1 (1%)			
Intestine small, ileum	(83)	(82)	(82)	(80)
Peyer's patch, hyperplasia, lymphoid			1 (1%)	
Intestine small, jejunum	(84)	(81)	(81)	(80)
Peyer's patch, hyperplasia, lymphoid	1 (1%)	1 (1%)		
Liver	(89)	(90)	(90)	(89)
Basophilic focus	4 (4%)	2 (2%)	5 (6%)	5 (6%)
Clear cell focus	1 (1%)			
Eosinophilic focus	2 (2%)	1 (1%)	1 (1%)	
Extramedullary hematopoiesis	1 (1%)		1 (1%)	
Fatty change	7 (8%)	1 (1%)		2 (2%)
Hemorrhage	1 (1%)	1 (1%)	1 (1%)	
Infiltration cellular, lymphocyte	33 (37%)	25 (28%)	21 (23%)	32 (36%)
Infiltration cellular, mononuclear cell	1 (1%)	2 (2%)	1 (1%)	
Infiltration cellular, polymorphonuclear		1 (1%)		
Inflammation, focal	4 (4%)	2 (2%)		
Inflammation, acute			1 (1%)	
Inflammation, chronic active	1 (1%)			
Mixed cell focus	5 (6%)	1 (1%)		1 (1%)
Necrosis	6 (7%)	5 (6%)	6 (7%)	3 (3%)
Artery, inflammation, chronic			1 (1%)	
Bile duct, cyst		1 (1%)	1 (1%)	
Centrilobular, hepatocyte, hypertrophy				1 (1%)
Hepatocyte, fatty change, focal	3 (3%)		1 (1%)	
Hepatocyte, hyperplasia			1 (1%)	
Hepatocyte, hypertrophy			1 (1%)	
Hepatocyte, inclusion body intracytoplasmic				1 (1%)
Hepatocyte, vacuolization cytoplasmic			4 (4%)	1 (1%)
Kupffer cell, hyperplasia	1 (1%)			

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Mesentery	(29)	(24)	(32)	(30)
Artery, inflammation, chronic		2 (8%)		
Fat, infiltration cellular, lymphocyte	2 (7%)	1 (4%)		
Fat, inflammation, granulomatous		1 (4%)		
Fat, inflammation, chronic active	1 (3%)			
Fat, mineral		1 (4%)	1 (3%)	
Fat, necrosis	25 (86%)	19 (79%)	27 (84%)	27 (90%)
Oral mucosa	(0)	(0)	(2)	(0)
Pancreas	(87)	(88)	(89)	(86)
Degeneration				1 (1%)
Infiltration cellular, lipocyte		1 (1%)		1 (1%)
Infiltration cellular, histiocyte			1 (1%)	
Infiltration cellular, lymphocyte	27 (31%)	26 (30%)	30 (34%)	24 (28%)
Inflammation, suppurative				1 (1%)
Inflammation, chronic active	1 (1%)			
Necrosis				1 (1%)
Acinus, atrophy			2 (2%)	1 (1%)
Duct, cyst	1 (1%)	1 (1%)	2 (2%)	3 (3%)
Duct, inflammation, chronic active				1 (1%)
Salivary glands	(89)	(89)	(90)	(90)
Atrophy	1 (1%)			
Infiltration cellular, lymphocyte	59 (66%)	54 (61%)	55 (61%)	62 (69%)
Inflammation, acute		1 (1%)		
Mineral			1 (1%)	
Arteriole, inflammation, chronic		1 (1%)		
Stomach, forestomach	(86)	(89)	(90)	(85)
Cyst			1 (1%)	
Hyperkeratosis			1 (1%)	
Ulcer		1 (1%)		
Epithelium, hyperplasia, focal			2 (2%)	
Stomach, glandular	(85)	(87)	(85)	(85)
Cyst	3 (4%)	3 (3%)	4 (5%)	
Infiltration cellular, lymphocyte			1 (1%)	2 (2%)
Ulcer			1 (1%)	
Tongue	(0)	(0)	(1)	(0)
Tooth	(0)	(0)	(1)	(0)
Cardiovascular System				
Aorta	(84)	(88)	(90)	(89)
Degeneration				1 (1%)
Inflammation, chronic active	1 (1%)			
Blood vessel	(0)	(0)	(2)	(0)
Heart	(90)	(90)	(90)	(90)
Bacteria	1 (1%)	1 (1%)	2 (2%)	
Cardiomyopathy	3 (3%)	1 (1%)	3 (3%)	3 (3%)
Thrombus	3 (3%)	1 (1%)	2 (2%)	
Artery, inflammation, chronic active		4 (4%)		1 (1%)
Endocardium, hyperplasia			1 (1%)	
Epicardium, infiltration cellular, mixed cell	1 (1%)			
Epicardium, infiltration cellular, mononuclear cell	1 (1%)			

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Cardiovascular System (continued)				
Heart (continued)	(90)	(90)	(90)	(90)
Myocardium, fibrosis	1 (1%)			
Myocardium, infiltration cellular, lymphocyte			1 (1%)	2 (2%)
Myocardium, inflammation, acute		1 (1%)	2 (2%)	
Myocardium, inflammation, chronic active	1 (1%)			
Myocardium, mineral	4 (4%)			1 (1%)
Valve, hemorrhage	1 (1%)			
Valve, infiltration cellular, lymphocyte	1 (1%)			
Valve, inflammation, chronic			1 (1%)	
Valve, thrombus	1 (1%)			
Endocrine System				
Adrenal cortex	(84)	(88)	(90)	(90)
Accessory adrenal cortical nodule		1 (1%)	1 (1%)	1 (1%)
Angiectasis	1 (1%)			
Hemorrhage	1 (1%)		3 (3%)	
Mineral	1 (1%)			
Vacuolization cytoplasmic		2 (2%)		
Bilateral, extramedullary hematopoiesis	1 (1%)			
Bilateral, hyperplasia, focal		1 (1%)		
Bilateral, vacuolization cytoplasmic		3 (3%)	1 (1%)	1 (1%)
Subcapsular, hyperplasia	81 (96%)	85 (97%)	88 (98%)	86 (96%)
Adrenal medulla	(83)	(84)	(86)	(87)
Hemorrhage	2 (2%)		1 (1%)	
Hyperplasia			2 (2%)	
Mineral	1 (1%)			
Islets, pancreatic	(87)	(88)	(90)	(86)
Hyperplasia	1 (1%)	3 (3%)	1 (1%)	
Infiltration cellular, lymphocyte	3 (3%)	2 (2%)	2 (2%)	3 (3%)
Parathyroid gland	(60)	(57)	(64)	(62)
Cyst	1 (2%)			
Infiltration cellular, lymphocyte			1 (2%)	1 (2%)
Pituitary gland	(80)	(80)	(84)	(84)
Pars distalis, angiectasis	2 (3%)	7 (9%)	6 (7%)	5 (6%)
Pars distalis, cyst	1 (1%)	3 (4%)		1 (1%)
Pars distalis, cytoplasmic alteration				1 (1%)
Pars distalis, hyperplasia, focal	2 (3%)	4 (5%)	5 (6%)	4 (5%)
Thyroid gland	(86)	(89)	(86)	(86)
Infiltration cellular, lymphocyte	1 (1%)	6 (7%)	6 (7%)	3 (3%)
Ultimobranchial cyst			2 (2%)	
Follicle, cyst	1 (1%)	1 (1%)		
Follicular cell, hyperplasia, focal		4 (4%)		
General Body System				
Peritoneum	(0)	(0)	(1)	(0)
Tissue NOS	(1)	(1)	(1)	(2)

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Genital System				
Clitoral gland	(82)	(84)	(80)	(86)
Infiltration cellular, lymphocyte	3 (4%)			1 (1%)
Duct, cyst	1 (1%)		1 (1%)	
Ovary	(75)	(86)	(82)	(80)
Angiectasis		1 (1%)	1 (1%)	2 (3%)
Cyst	9 (12%)	13 (15%)	8 (10%)	7 (9%)
Cyst, squamous			1 (1%)	
Hemorrhage	1 (1%)	2 (2%)	1 (1%)	
Hyperplasia, cystic, papillary		1 (1%)	1 (1%)	
Hyperplasia, tubulostromal				1 (1%)
Infiltration cellular, lymphocyte				1 (1%)
Mineral	1 (1%)			
Thrombus			1 (1%)	1 (1%)
Bursa, cyst				1 (1%)
Follicle, cyst	9 (12%)	11 (13%)	6 (7%)	7 (9%)
Granulosa cell, hyperplasia		2 (2%)		
Paraovarian tissue, cyst				1 (1%)
Oviduct	(0)	(0)	(1)	(0)
Uterus	(89)	(90)	(90)	(89)
Angiectasis	1 (1%)	6 (7%)	5 (6%)	3 (3%)
Congestion		1 (1%)		
Dilation	35 (39%)	29 (32%)	30 (33%)	26 (29%)
Hemorrhage	1 (1%)	1 (1%)	1 (1%)	
Infiltration cellular, lymphocyte			1 (1%)	
Inflammation, acute		1 (1%)		
Thrombus	1 (1%)			
Endometrium, cyst	3 (3%)			1 (1%)
Endometrium, hyperplasia		1 (1%)		1 (1%)
Endometrium, hyperplasia, cystic	68 (76%)	75 (83%)	72 (80%)	68 (76%)
Endometrium, metaplasia, squamous	1 (1%)			
Vagina	(0)	(0)	(1)	(0)
Hematopoietic System				
Bone marrow	(90)	(89)	(89)	(90)
Hypercellularity	7 (8%)	8 (9%)	4 (4%)	4 (4%)
Hypocellularity	1 (1%)	1 (1%)		
Myeloid cell, hypercellularity	1 (1%)			
Lymph node	(18)	(20)	(16)	(14)
Hemorrhage			1 (6%)	1 (7%)
Hyperplasia, lymphoid	1 (6%)			
Axillary, infiltration cellular, mixed cell	1 (6%)			
Axillary, pigment	1 (6%)			
Bronchial, hyperplasia, lymphoid	2 (11%)			4 (29%)
Iliac, erythrophagocytosis		1 (5%)		
Iliac, hemorrhage	1 (6%)		2 (13%)	
Iliac, hyperplasia, lymphoid	4 (22%)	2 (10%)	6 (38%)	2 (14%)
Iliac, infiltration cellular, histiocyte		1 (5%)	1 (6%)	
Iliac, infiltration cellular, mixed cell	1 (6%)	1 (5%)	4 (25%)	
Iliac, pigment			4 (25%)	
Lumbar, hyperplasia, lymphoid		1 (5%)		
Lumbar, infiltration cellular, mixed cell	1 (6%)			
Mediastinal, hyperplasia, lymphoid	1 (6%)	2 (10%)		
Mediastinal, infiltration cellular, plasma cell		1 (5%)		

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Lymph node (continued)	(18)	(20)	(16)	(14)
Pancreatic, hyperplasia, lymphoid	1 (6%)			
Renal, erythrophagocytosis			1 (6%)	
Renal, hemorrhage	1 (6%)			
Renal, hyperplasia, lymphoid	3 (17%)	2 (10%)	1 (6%)	
Lymph node, mandibular	(76)	(77)	(81)	(83)
Hemorrhage	3 (4%)	2 (3%)	1 (1%)	1 (1%)
Hyperplasia, lymphoid	1 (1%)	1 (1%)	1 (1%)	4 (5%)
Infiltration cellular, histiocyte			2 (2%)	
Infiltration cellular, mixed cell	1 (1%)			
Lymph node, mesenteric	(71)	(84)	(80)	(83)
Angiectasis		1 (1%)		
Erythrophagocytosis	1 (1%)		1 (1%)	
Hemorrhage	1 (1%)	4 (5%)	2 (3%)	1 (1%)
Hyperplasia, lymphoid	1 (1%)	10 (12%)	3 (4%)	3 (4%)
Infiltration cellular, histiocyte	3 (4%)	8 (10%)	6 (8%)	4 (5%)
Infiltration cellular, plasma cell		2 (2%)		
Spleen	(86)	(87)	(89)	(87)
Atrophy	1 (1%)			
Extramedullary hematopoiesis	20 (23%)	15 (17%)	19 (21%)	11 (13%)
Hyperplasia, lymphoid	11 (13%)	7 (8%)	13 (15%)	10 (11%)
Capsule, fibrosis	1 (1%)			
Capsule, inflammation, chronic active			1 (1%)	
Thymus	(85)	(80)	(84)	(86)
Atrophy	5 (6%)	3 (4%)	8 (10%)	1 (1%)
Cyst	2 (2%)	2 (3%)	7 (8%)	2 (2%)
Hemorrhage			1 (1%)	1 (1%)
Hyperplasia, lymphoid	1 (1%)		2 (2%)	2 (2%)
Infiltration cellular, histiocyte		1 (1%)		
Integumentary System				
Mammary gland	(85)	(88)	(88)	(84)
Hyperplasia, focal	1 (1%)		1 (1%)	
Hyperplasia, diffuse	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Duct, dilation	1 (1%)	1 (1%)	2 (2%)	1 (1%)
Skin	(90)	(90)	(90)	(90)
Inflammation, chronic		1 (1%)		
Ulcer	2 (2%)	2 (2%)	2 (2%)	2 (2%)
Epidermis, hyperplasia, multifocal				1 (1%)
Hair follicle, atrophy	2 (2%)	1 (1%)	4 (4%)	8 (9%)
Subcutaneous tissue, inflammation, chronic			1 (1%)	
Musculoskeletal System				
Bone	(90)	(90)	(89)	(90)
Decreased bone		1 (1%)		
Fibro-osseous lesion	11 (12%)	6 (7%)	4 (4%)	3 (3%)
Increased bone		1 (1%)		1 (1%)
Periosteum, vertebra, inflammation, granulomatous		1 (1%)		

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Musculoskeletal System (continued)				
Skeletal muscle	(89)	(90)	(90)	(90)
Degeneration			2 (2%)	1 (1%)
Infiltration cellular, lymphocyte	16 (18%)	5 (6%)	10 (11%)	16 (18%)
Inflammation, chronic active				1 (1%)
Mineral	1 (1%)			
Arteriole, inflammation, chronic		2 (2%)		
Nervous System				
Brain	(87)	(90)	(90)	(90)
Cyst, squamous			2 (2%)	
Hemorrhage	2 (2%)		3 (3%)	
Hydrocephalus	1 (1%)			
Inflammation, acute	1 (1%)			
Inflammation, chronic		1 (1%)		
Mineral	80 (92%)	78 (87%)	77 (86%)	78 (87%)
Necrosis	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Artery, meninges, inflammation, chronic active		5 (6%)	3 (3%)	2 (2%)
Brain trigeminal ganglion	(75)	(74)	(80)	(79)
Nerve trigeminal	(56)	(58)	(53)	(35)
Peripheral nerve	(0)	(0)	(1)	(0)
Peripheral nerve, sciatic	(88)	(87)	(88)	(88)
Axon, degeneration	12 (14%)	6 (7%)	7 (8%)	6 (7%)
Spinal cord	(90)	(90)	(90)	(90)
Cyst, squamous		1 (1%)		
Necrosis		3 (3%)		
Artery, meninges, inflammation, chronic active		5 (6%)	1 (1%)	2 (2%)
Respiratory System				
Larynx	(0)	(0)	(2)	(0)
Lung	(90)	(90)	(90)	(90)
Congestion		2 (2%)	2 (2%)	1 (1%)
Hemorrhage	4 (4%)	1 (1%)	3 (3%)	3 (3%)
Hyperplasia, lymphoid		1 (1%)	1 (1%)	
Infiltration cellular, histiocyte	1 (1%)	2 (2%)		
Infiltration cellular, lymphocyte	3 (3%)	1 (1%)	2 (2%)	3 (3%)
Infiltration cellular, mononuclear cell		1 (1%)		
Inflammation, acute	1 (1%)			
Inflammation, chronic	1 (1%)			
Inflammation, chronic active				1 (1%)
Alveolar epithelium, hyperplasia, focal	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Serosa, inflammation, chronic		1 (1%)		
Mediastinum	(2)	(0)	(0)	(0)
Nose	(89)	(90)	(90)	(90)
Inflammation, acute	1 (1%)			
Respiratory epithelium, accumulation, hyaline droplet		1 (1%)		
Respiratory epithelium, hyperplasia			1 (1%)	
Vomeronasal organ, cyst				1 (1%)
Pleura	(0)	(0)	(1)	(0)
Trachea	(90)	(89)	(90)	(90)

TABLE B4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Special Senses System				
Ear	(0)	(0)	(1)	(0)
Eye	(89)	(88)	(90)	(90)
Phthisis bulbi			1 (1%)	
Anterior chamber, inflammation, acute				1 (1%)
Bilateral, retina, hemorrhage			1 (1%)	
Cornea, inflammation, acute			1 (1%)	1 (1%)
Cornea, inflammation, chronic			2 (2%)	
Cornea, inflammation, chronic active		1 (1%)		
Cornea, necrosis		1 (1%)	1 (1%)	
Harderian gland	(89)	(90)	(90)	(87)
Hyperplasia, focal			1 (1%)	
Infiltration cellular, lymphocyte	58 (65%)	68 (76%)	69 (77%)	63 (72%)
Inflammation, chronic active				1 (1%)
Lacrimal gland	(0)	(1)	(2)	(0)
Zymbal's gland	(0)	(0)	(1)	(0)
Urinary System				
Kidney	(89)	(87)	(89)	(88)
Cyst	1 (1%)			
Glomerulopathy, hyaline		2 (2%)		
Hemorrhage				1 (1%)
Infarct	14 (16%)	19 (22%)	20 (22%)	14 (16%)
Inflammation, acute			1 (1%)	
Metaplasia, osseous	2 (2%)	2 (2%)	2 (2%)	2 (2%)
Nephropathy, chronic progressive	8 (9%)	15 (17%)	19 (21%)	14 (16%)
Bilateral, infarct	1 (1%)			
Interstitium, infiltration cellular, lymphocyte	63 (71%)	60 (69%)	65 (73%)	56 (64%)
Papilla, mineral		2 (2%)	1 (1%)	1 (1%)
Pelvis, dilatation	1 (1%)			
Pelvis, mineral				1 (1%)
Pelvis, necrosis			1 (1%)	
Renal tubule, dilation	1 (1%)		1 (1%)	1 (1%)
Renal tubule, hyaline droplet		1 (1%)		
Renal tubule, mineral	1 (1%)		1 (1%)	
Renal tubule, vacuolization cytoplasmic		1 (1%)		
Ureter	(0)	(0)	(1)	(0)
Urethra	(0)	(0)	(1)	(0)
Urinary bladder	(86)	(87)	(86)	(86)
Angiectasis				2 (2%)
Infiltration cellular, lymphocyte	62 (72%)	67 (77%)	65 (76%)	68 (79%)
Inflammation, acute			1 (1%)	
Arteriole, inflammation, chronic		1 (1%)		
Urothelium, hyperplasia			1 (1%)	

APPENDIX C
SUMMARY OF LESIONS IN MALE MICE EXPOSED TO CDMA-MODULATED CELL PHONE RFR FOR 2 YEARS

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	106	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death			1	
Moribund	8	2	5	3
Natural deaths	16	6	13	16
Survivors				
Terminal euthanasia	66	83	71	71
Animals examined microscopically	100	101	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
2-Year Study				
Alimentary System				
Esophagus	(88)	(91)	(89)	(88)
Gallbladder	(73)	(80)	(75)	(76)
Intestine large, cecum	(81)	(87)	(81)	(80)
Adenoma			1 (1%)	
Intestine large, colon	(84)	(88)	(84)	(81)
Adenocarcinoma			1 (1%)	
Intestine large, rectum	(84)	(89)	(85)	(85)
Intestine small, duodenum	(77)	(86)	(81)	(80)
Adenocarcinoma	1 (1%)			
Intestine small, ileum	(81)	(88)	(83)	(81)
Adenoma				1 (1%)
Intestine small, jejunum	(79)	(87)	(81)	(82)
Adenocarcinoma	2 (3%)		1 (1%)	2 (2%)
Adenoma				1 (1%)
Hepatocellular carcinoma, metastatic, liver	1 (1%)			

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Liver	(90)	(89)	(90)	(90)
Hemangiosarcoma	1 (1%)	4 (4%)	2 (2%)	1 (1%)
Hepatoblastoma	6 (7%)	6 (7%)	15 (17%)	7 (8%)
Hepatoblastoma, multiple			1 (1%)	
Hepatocellular adenoma	25 (28%)	23 (26%)	22 (24%)	36 (40%)
Hepatocellular adenoma, multiple	27 (30%)	43 (48%)	33 (37%)	26 (29%)
Hepatocellular carcinoma	26 (29%)	13 (15%)	18 (20%)	24 (27%)
Hepatocellular carcinoma, multiple	2 (2%)	5 (6%)	7 (8%)	7 (8%)
Hepatocholangiocarcinoma	1 (1%)			2 (2%)
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skeletal muscle				1 (1%)
Mesentery	(12)	(9)	(18)	(16)
Hemangiosarcoma	1 (8%)			
Hepatoblastoma, metastatic, liver			1 (6%)	
Malignant fibrous histiocytoma, metastatic, skin	1 (8%)			
Fat, hepatocholangiocarcinoma, metastatic, liver	1 (8%)			
Fat, lipoma	1 (8%)		2 (11%)	
Pancreas	(87)	(88)	(88)	(88)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			
Salivary glands	(90)	(90)	(89)	(90)
Stomach, forestomach	(88)	(89)	(86)	(87)
Squamous cell papilloma			1 (1%)	
Stomach, glandular	(87)	(88)	(87)	(87)
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Tooth	(27)	(15)	(17)	(23)
Cardiovascular System				
Aorta	(89)	(88)	(90)	(89)
Alveolar/bronchiolar carcinoma, metastatic, lung	1 (1%)			
Blood vessel	(1)	(1)	(0)	(0)
Heart	(90)	(91)	(90)	(90)
Alveolar/bronchiolar carcinoma, metastatic, lung	1 (1%)	1 (1%)		
Hemangioma		1 (1%)		
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			1 (1%)
Sarcoma, metastatic, skeletal muscle				1 (1%)

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(89)	(90)	(89)
Bilateral, malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Bilateral, subcapsular, adenoma		1 (1%)	1 (1%)	
Subcapsular, adenoma		1 (1%)	3 (3%)	4 (4%)
Subcapsular, carcinoma			1 (1%)	
Adrenal medulla	(90)	(89)	(88)	(89)
Pheochromocytoma benign				1 (1%)
Islets, pancreatic	(88)	(90)	(89)	(89)
Adenoma		1 (1%)		
Carcinoma		1 (1%)		
Parathyroid gland	(68)	(57)	(66)	(65)
Pituitary gland	(86)	(84)	(89)	(83)
Pars distalis, adenoma			2 (2%)	
Pars distalis, carcinoma			1 (1%)	
Thyroid gland	(89)	(89)	(88)	(87)
Follicular cell, adenoma		1 (1%)	1 (1%)	1 (1%)
General Body System				
Peritoneum	(1)	(0)	(0)	(0)
Hepatocholangiocarcinoma, metastatic, liver	1 (100%)			
Tissue NOS	(0)	(1)	(0)	(0)
Fat, hemangiosarcoma		1 (100%)		
Genital System				
Coagulating gland	(2)	(3)	(0)	(1)
Epididymis	(90)	(91)	(90)	(90)
Preputial gland	(89)	(89)	(89)	(89)
Prostate	(90)	(86)	(90)	(88)
Seminal vesicle	(90)	(90)	(90)	(90)
Fibroma	1 (1%)			
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Testis	(90)	(91)	(88)	(90)
Interstitial cell, adenoma	2 (2%)	2 (2%)		
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hemangiosarcoma			1 (1%)	
Lymph node	(6)	(6)	(11)	(10)
Axillary, hepatocholangiocarcinoma, metastatic, liver	1 (17%)			
Axillary, squamous cell carcinoma, metastatic, skin			1 (9%)	
Bronchial, sarcoma, metastatic, skeletal muscle				1 (10%)
Lumbar, squamous cell carcinoma, metastatic, skin			1 (9%)	
Pancreatic, hepatoblastoma, metastatic, liver			1 (9%)	
Lymph node, mandibular	(72)	(70)	(63)	(64)

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Lymph node, mesenteric	(85)	(88)	(86)	(85)
Hemangioma	1 (1%)			
Hepatoblastoma, metastatic, liver			1 (1%)	
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Spleen	(87)	(89)	(87)	(86)
Hemangiosarcoma		2 (2%)	1 (1%)	2 (2%)
Thymus	(75)	(76)	(80)	(81)
Integumentary System				
Mammary gland	(2)	(1)	(0)	(3)
Skin	(90)	(91)	(90)	(90)
Lipoma				1 (1%)
Pilomatrixoma	1 (1%)			
Squamous cell carcinoma			1 (1%)	
Subcutaneous tissue, hemangiosarcoma	1 (1%)			
Subcutaneous tissue, lipoma	1 (1%)		1 (1%)	
Subcutaneous tissue, malignant fibrous histiocytoma	1 (1%)	1 (1%)	2 (2%)	
Musculoskeletal System				
Bone	(90)	(91)	(90)	(90)
Skeletal muscle	(90)	(91)	(90)	(90)
Alveolar/bronchiolar carcinoma, metastatic, lung		1 (1%)	1 (1%)	
Hepatoblastoma, metastatic, liver			1 (1%)	
Hepatocellular carcinoma, metastatic, liver	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			1 (1%)
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Sarcoma	1 (1%)			1 (1%)
Squamous cell carcinoma, metastatic, skin			1 (1%)	
Nervous System				
Brain	(90)	(91)	(90)	(90)
Carcinoma, metastatic, pituitary gland			1 (1%)	
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			
Brain trigeminal ganglion	(69)	(79)	(80)	(80)
Nerve trigeminal	(67)	(57)	(43)	(55)
Peripheral nerve, sciatic	(89)	(91)	(87)	(88)
Spinal cord	(90)	(91)	(90)	(90)

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(91)	(90)	(90)
Alveolar/bronchiolar adenoma	11 (12%)	8 (9%)	14 (16%)	12 (13%)
Alveolar/bronchiolar adenoma, multiple	2 (2%)		2 (2%)	
Alveolar/bronchiolar carcinoma	11 (12%)	13 (14%)	11 (12%)	11 (12%)
Alveolar/bronchiolar carcinoma, multiple	2 (2%)			
Hepatoblastoma, metastatic, liver	1 (1%)		2 (2%)	
Hepatocellular carcinoma, metastatic, liver	11 (12%)	4 (4%)	9 (10%)	11 (12%)
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			1 (1%)
Sarcoma, metastatic, skeletal muscle				1 (1%)
Squamous cell carcinoma, metastatic, skin			1 (1%)	
Nose	(90)	(91)	(90)	(90)
Trachea	(90)	(90)	(90)	(89)
Special Senses System				
Eye	(90)	(91)	(89)	(90)
Harderian gland	(88)	(91)	(90)	(88)
Adenocarcinoma	3 (3%)	3 (3%)	1 (1%)	2 (2%)
Adenoma	6 (7%)	4 (4%)	4 (4%)	4 (5%)
Urinary System				
Kidney	(90)	(89)	(90)	(90)
Hepatocellular carcinoma, metastatic, liver	1 (1%)			
Hepatocholangiocarcinoma, metastatic, liver	1 (1%)			
Malignant fibrous histiocytoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skeletal muscle				1 (1%)
Renal tubule, adenoma				1 (1%)
Ureter	(0)	(0)	(1)	(0)
Urinary bladder	(87)	(90)	(90)	(89)
Systemic Lesions				
Multiple organs[b]	(90)	(91)	(90)	(90)
Histiocytic sarcoma		2 (2%)	1 (1%)	2 (2%)
Leukemia granulocytic				1 (1%)
Lymphoma malignant	6 (7%)	3 (3%)	5 (6%)	4 (4%)
Mast cell tumor	1 (1%)			

TABLE C1
Summary of the Incidence of Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms[c]				
2-Year study	79	80	82	94
Total primary neoplasms				
2-Year study	144	139	157	155
Total animals with benign neoplasms				
2-Year study	61	70	63	70
Total benign neoplasms				
2-Year study	77	85	87	89
Total animals with malignant neoplasms				
2-Year study	49	42	58	50
Total malignant neoplasms				
2-Year study	66	54	70	66
Total animals with metastatic neoplasms				
2-Year study	14	6	14	13
Total metastatic neoplasms				
2-Year study	34	6	21	19
Total animals with uncertain neoplasms-benign or malignant				
2-Year study	1			
Total uncertain neoplasms				
2-Year study	1			

[a] Number of animals examined microscopically at the site and the number of animals with neoplasm
[b] Number of animals with any tissue examined microscopically
[c] Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE C2
Statistical Analysis of Primary Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Harderian Gland: Adenoma				
Overall rate[a]	6/90 (7%)	4/91 (4%)	4/90 (4%)	4/90 (4%)
Adjusted rate[b]	7.5%	4.6%	4.7%	4.8%
Terminal rate[c]	6/66 (9%)	4/83 (5%)	4/71 (6%)	3/71 (4%)
First incidence (days)	729 (T)	729 (T)	729 (T)	707
Poly-3 test[d]	P=0.342N	P=0.322N	P=0.342N	P=0.353N
Harderian Gland: Adenoma or Carcinoma				
Overall rate	9/90 (10%)	7/91 (8%)	5/90 (6%)	6/90 (7%)
Adjusted rate	11.2%	8.0%	5.9%	7.2%
Terminal rate	8/66 (12%)	7/83 (8%)	5/71 (7%)	5/71 (7%)
First incidence (days)	690	729 (T)	729 (T)	707
Poly-3 test	P=0.237N	P=0.331N	P=0.176N	P=0.273N
Liver: Hepatocellular Adenoma				
Overall rate	52/90 (58%)	66/89 (74%)	55/90 (61%)	62/90 (69%)
Adjusted rate	62.3%	75.4%	64.9%	72.7%
Terminal rate	45/66 (68%)	64/83 (77%)	51/71 (72%)	54/71 (76%)
First incidence (days)	393	625	656	478
Poly-3 test	P=0.199	P=0.043	P=0.428	P=0.096
Liver: Hepatocellular Carcinoma				
Overall rate	28/90 (31%)	18/89 (20%)	25/90 (28%)	31/90 (34%)
Adjusted rate	34.2%	20.6%	29.0%	36.2%
Terminal rate	18/66 (27%)	16/83 (19%)	18/71 (25%)	22/71 (31%)
First incidence (days)	608	629	559	461
Poly-3 test	P=0.177	P=0.033N	P=0.287N	P=0.459
Liver: Hepatocellular Adenoma or Carcinoma				
Overall rate	67/90 (74%)	70/89 (79%)	66/90 (73%)	73/90 (81%)
Adjusted rate	79.1%	79.6%	76.6%	83.3%
Terminal rate	51/66 (77%)	67/83 (81%)	58/71 (82%)	59/71 (83%)
First incidence (days)	393	625	559	461
Poly-3 test	P=0.278	P=0.543	P=0.412N	P=0.302
Liver: Hepatoblastoma				
Overall rate	6/90 (7%)	6/89 (7%)	16/90 (18%)	7/90 (8%)
Adjusted rate	7.5%	6.9%	18.9%	8.5%
Terminal rate	5/66 (8%)	6/83 (7%)	14/71 (20%)	7/71 (10%)
First incidence (days)	711	729 (T)	679	729 (T)
Poly-3 test	P=0.328	P=0.562N	P=0.026	P=0.523
Liver: Hepatocellular Carcinoma or Hepatoblastoma				
Overall rate	32/90 (36%)	22/89 (25%)	37/90 (41%)	35/90 (39%)
Adjusted rate	39.1%	25.1%	42.8%	40.9%
Terminal rate	22/66 (33%)	20/83 (24%)	28/71 (39%)	26/71 (37%)
First incidence (days)	608	629	559	461
Poly-3 test	P=0.159	P=0.036N	P=0.370	P=0.472
Liver: Hepatocellular Adenoma, Hepatocellular Carcinoma, or Hepatoblastoma				
Overall rate	68/90 (76%)	70/89 (79%)	69/90 (77%)	75/90 (83%)
Adjusted rate	80.3%	79.6%	79.8%	85.6%
Terminal rate	52/66 (79%)	67/83 (81%)	59/71 (83%)	61/71 (86%)
First incidence (days)	393	625	559	461
Poly-3 test	P=0.175	P=0.532N	P=0.548N	P=0.230

TABLE C2
Statistical Analysis of Primary Neoplasms in Male Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Lung: Alveolar/bronchiolar Adenoma				
Overall rate	13/90 (14%)	8/91 (9%)	16/90 (18%)	12/90 (13%)
Adjusted rate	16.0%	9.1%	19.0%	14.4%
Terminal rate	9/66 (14%)	7/83 (8%)	15/71 (21%)	11/71 (16%)
First incidence (days)	488	594	727	585
Poly-3 test	P=0.441	P=0.131N	P=0.382	P=0.474N
Lung: Alveolar/bronchiolar Carcinoma				
Overall rate	13/90 (14%)	13/91 (14%)	11/90 (12%)	11/90 (12%)
Adjusted rate	16.1%	14.7%	12.9%	13.1%
Terminal rate	12/66 (18%)	10/83 (12%)	9/71 (13%)	8/71 (11%)
First incidence (days)	568	625	588	518
Poly-3 test	P=0.326N	P=0.486N	P=0.360N	P=0.375N
Lung: Alveolar/bronchiolar Adenoma or Carcinoma				
Overall rate	23/90 (26%)	21/91 (23%)	25/90 (28%)	21/90 (23%)
Adjusted rate	28.1%	23.7%	29.4%	24.9%
Terminal rate	18/66 (27%)	17/83 (21%)	22/71 (31%)	17/71 (24%)
First incidence (days)	488	594	588	518
Poly-3 test	P=0.444N	P=0.312N	P=0.496	P=0.385N
All Organs: Hemangiosarcoma				
Overall rate	2/90 (2%)	7/91 (8%)	4/90 (4%)	3/90 (3%)
Adjusted rate	2.5%	8.0%	4.7%	3.6%
Terminal rate	0/66 (0%)	7/83 (8%)	4/71 (6%)	3/71 (4%)
First incidence (days)	702	729 (T)	729 (T)	729 (T)
Poly-3 test	P=0.483N	P=0.107	P=0.362	P=0.513
All Organs: Hemangioma or Hemangiosarcoma				
Overall rate	3/90 (3%)	8/91 (9%)	4/90 (4%)	3/90 (3%)
Adjusted rate	3.7%	9.2%	4.7%	3.6%
Terminal rate	1/66 (2%)	8/83 (10%)	4/71 (6%)	3/71 (4%)
First incidence (days)	702	729 (T)	729 (T)	729 (T)
Poly-3 test	P=0.325N	P=0.135	P=0.526	P=0.647N
All Organs: Malignant Lymphoma				
Overall rate	6/90 (7%)	3/91 (3%)	5/90 (6%)	4/90 (4%)
Adjusted rate	7.3%	3.4%	5.9%	4.8%
Terminal rate	4/66 (6%)	3/83 (4%)	3/71 (4%)	4/71 (6%)
First incidence (days)	263	729 (T)	674	729 (T)
Poly-3 test	P=0.413N	P=0.217N	P=0.478N	P=0.366N

(T) Terminal euthanasia

[a] Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for liver and lung; for other tissues, denominator is number of animals necropsied.

[b] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

[c] Observed incidence at terminal euthanasia

[d] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A negative trend or a lower incidence in an exposure group is indicated by **N**.

TABLE C3
Historical Incidence of Liver Neoplasms in Control Male B6C3F1/N Mice[a]

	Hepatocellular Adenoma	Hepatocellular Carcinoma	Hepatoblastoma	Hepatocellular Adenoma, Hepatocellular Carcinoma, or Hepatoblastoma
Overall Historical Incidence: All Routes				
Total (%)	308/589 (52.3%)	164/589 (27.8%)	19/589 (3.2%)	408/589 (69.3%)
Mean ± standard deviation	51.9% ± 10.3%	27.6% ± 8.3%	3.0% ± 2.2%	68.8% ± 8.6%
Range	34%-70%	16%-42%	0%-7%	53%-80%

[a] Data as of August 2017

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	106	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Accidental death			1	
Moribund	8	2	5	3
Natural deaths	16	6	13	16
Survivors				
Terminal euthanasia	66	83	71	71
Animals examined microscopically	100	101	100	100
14-Week Interim Evaluation				
Alimentary System				
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte			1 (10%)	
Infiltration cellular, mixed cell, multifocal			2 (20%)	
Infiltration cellular, mixed cell		1 (10%)		
Inflammation, focal				1 (10%)
Salivary glands	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte		1 (10%)		
Endocrine System				
Thyroid gland	(10)	(9)	(10)	(10)
Infiltration cellular, lymphocyte				1 (10%)
Hematopoietic System				
Lymph node, mandibular	(5)	(8)	(9)	(10)
Hemorrhage			1 (11%)	2 (20%)
Nervous System				
Brain	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)			
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	1 (10%)			
Hemorrhage	2 (20%)			
Infiltration cellular, mixed cell			1 (10%)	
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive	1 (10%)		1 (10%)	1 (10%)
Interstitium, infiltration cellular, lymphocyte	2 (20%)	2 (20%)	3 (30%)	1 (10%)

[a] Number of animals examined microscopically at the site and the number of animals with lesion

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
14-Week Interim Evaluation (continued)				
Systems Examined with No Lesions Observed				
Cardiovascular System				
General Body System				
Genital System				
Integumentary System				
Musculoskeletal System				
Special Senses System				
2-Year Study				
Alimentary System				
Esophagus	(88)	(91)	(89)	(88)
Infiltration cellular, lymphocyte		1 (1%)		
Gallbladder	(73)	(80)	(75)	(76)
Cyst			1 (1%)	
Intestine large, cecum	(81)	(87)	(81)	(80)
Intestine large, colon	(84)	(88)	(84)	(81)
Intestine large, rectum	(84)	(89)	(85)	(85)
Intestine small, duodenum	(77)	(86)	(81)	(80)
Peyer's patch, hyperplasia, lymphoid				1 (1%)
Intestine small, ileum	(81)	(88)	(83)	(81)
Peyer's patch, hyperplasia, lymphoid	1 (1%)	1 (1%)	1 (1%)	3 (4%)
Peyer's patch, infiltration cellular, plasma cell	1 (1%)			
Intestine small, jejunum	(79)	(87)	(81)	(82)
Inflammation, granulomatous	1 (1%)			
Epithelium, cyst	1 (1%)			
Peyer's patch, hyperplasia, lymphoid		1 (1%)	2 (2%)	3 (4%)
Peyer's patch, infiltration, cellular, polymorphonuclear		1 (1%)		
Serosa, inflammation, granulomatous			1 (1%)	
Liver	(90)	(89)	(90)	(90)
Angiectasis			1 (1%)	
Basophilic focus	1 (1%)	2 (2%)	3 (3%)	2 (2%)
Clear cell focus	28 (31%)	49 (55%)	35 (39%)	31 (34%)
Congestion, chronic		1 (1%)		
Eosinophilic focus	4 (4%)	5 (6%)	2 (2%)	5 (6%)
Extramedullary hematopoiesis	2 (2%)		1 (1%)	2 (2%)
Fatty change	37 (41%)	51 (57%)	26 (29%)	33 (37%)
Fibrosis	1 (1%)			
Hemorrhage	1 (1%)			
Infiltration cellular, lymphocyte	2 (2%)		1 (1%)	3 (3%)
Infiltration cellular, mixed cell	1 (1%)	1 (1%)		
Infiltration cellular, polymorphonuclear				1 (1%)
Inflammation, focal	1 (1%)	1 (1%)		1 (1%)
Inflammation, chronic active	2 (2%)		1 (1%)	
Metaplasia				1 (1%)
Mineral				1 (1%)
Mixed cell focus	2 (2%)	2 (2%)	3 (3%)	1 (1%)
Necrosis	6 (7%)	2 (2%)	9 (10%)	10 (11%)
Bile duct, cyst				1 (1%)
Capsule, fibrosis		1 (1%)		1 (1%)
Hepatocyte, degeneration			1 (1%)	
Vein, inflammation, chronic active		1 (1%)		

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Mesentery	(12)	(9)	(18)	(16)
Artery, inflammation, chronic active		1 (11%)		1 (6%)
Artery, thrombus				1 (6%)
Fat, inflammation, granulomatous			1 (6%)	1 (6%)
Fat, necrosis	8 (67%)	8 (89%)	14 (78%)	12 (75%)
Pancreas	(87)	(88)	(88)	(88)
Hemorrhage	1 (1%)			
Infiltration cellular, lymphocyte	3 (3%)	2 (2%)	2 (2%)	4 (5%)
Inflammation, granulomatous	1 (1%)			
Artery, inflammation, chronic		1 (1%)		
Duct, cyst	1 (1%)			1 (1%)
Duct, fibrosis	1 (1%)			
Salivary glands	(90)	(90)	(89)	(90)
Infiltration cellular, lymphocyte	58 (64%)	67 (74%)	68 (76%)	61 (68%)
Stomach, forestomach	(88)	(89)	(86)	(87)
Cyst, squamous		1 (1%)		3 (3%)
Inflammation, acute			1 (1%)	
Ulcer				1 (1%)
Epithelium, hyperplasia, focal	3 (3%)		3 (3%)	
Epithelium, hyperplasia, diffuse				1 (1%)
Stomach, glandular	(87)	(88)	(87)	(87)
Hemorrhage	1 (1%)			
Mineral			2 (2%)	
Epithelium, hyperplasia, focal	1 (1%)			
Tooth	(27)	(15)	(17)	(23)
Dysplasia	26 (96%)	15 (100%)	16 (94%)	22 (96%)
Inflammation, suppurative	2 (7%)	1 (7%)		
Inflammation, chronic active			1 (6%)	2 (9%)
Cardiovascular System				
Aorta	(89)	(88)	(90)	(89)
Inflammation, chronic		1 (1%)		
Blood vessel	(1)	(1)	(0)	(0)
Inflammation, chronic	1 (100%)	1 (100%)		
Heart	(90)	(91)	(90)	(90)
Bacteria	1 (1%)		1 (1%)	
Cardiomyopathy	10 (11%)	6 (7%)	6 (7%)	10 (11%)
Inflammation, acute	1 (1%)		1 (1%)	1 (1%)
Inflammation, chronic active	2 (2%)		1 (1%)	
Mineral				1 (1%)
Thrombus	1 (1%)	1 (1%)	1 (1%)	
Artery, inflammation, chronic active	1 (1%)	3 (3%)	1 (1%)	
Endocardium, mineral	1 (1%)			
Endothelium, hyperplasia		2 (2%)		1 (1%)
Epicardium, inflammation, chronic	1 (1%)			
Epicardium, mineral	1 (1%)			
Intima, vein, hyperplasia		2 (2%)		
Myocardium, infiltration cellular, lymphocyte		2 (2%)		2 (2%)
Myocarcium, inflammation, chronic active		1 (1%)		
Myocardium, mineral	2 (2%)	2 (2%)		1 (1%)
Myocardium, necrosis	1 (1%)		1 (1%)	
Valve, inflammation, chronic active		1 (1%)		
Vein, inflammation, chronic active		1 (1%)		

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Endocrine System				
Adrenal cortex	(90)	(89)	(90)	(89)
Accessory adrenal cortical nodule	1 (1%)			
Angiectasis	1 (1%)			
Hemorrhage		1 (1%)		
Hyperplasia, focal	3 (3%)	3 (3%)	3 (3%)	1 (1%)
Hypertrophy, focal	2 (2%)	8 (9%)		3 (3%)
Vacuolization cytoplasmic, focal		1 (1%)		
Bilateral, hyperplasia, focal			1 (1%)	
Bilateral, hypertrophy, focal	1 (1%)	1 (1%)		1 (1%)
Subcapsular, hyperplasia	69 (77%)	73 (82%)	74 (82%)	66 (74%)
Adrenal medulla	(90)	(89)	(88)	(89)
Hyperplasia		1 (1%)		
Islets, pancreatic	(88)	(90)	(89)	(89)
Hyperplasia	18 (20%)	13 (14%)	14 (16%)	11 (12%)
Infiltration cellular, lymphocyte	2 (2%)	1 (1%)		4 (4%)
Parathyroid gland	(68)	(57)	(66)	(65)
Cyst		1 (2%)		1 (2%)
Pituitary gland	(86)	(84)	(89)	(83)
Pars distalis, angiectasis	1 (1%)			
Pars distalis, cyst	3 (3%)	1 (1%)	1 (1%)	2 (2%)
Pars distalis, hyperplasia, focal	1 (1%)			
Thyroid gland	(89)	(89)	(88)	(87)
Infiltration cellular, lymphocyte		1 (1%)	1 (1%)	
Arteriole, inflammation, chronic active		1 (1%)		
Epithelium, follicle, hyperplasia, focal		1 (1%)		
General Body System				
Peritoneum	(1)	(0)	(0)	(0)
Tissues NOS	(0)	(1)	(0)	(0)
Genital System				
Coagulating gland	(2)	(3)	(0)	(1)
Cyst	2 (100%)	3 (100%)		1 (100%)
Infiltration cellular, lymphocyte		1 (33%)		
Inflammation, chronic		1 (33%)		
Epididymis	(90)	(91)	(90)	(90)
Granuloma sperm	1 (1%)		2 (2%)	
Infiltration cellular, lymphocyte	29 (32%)	26 (29%)	26 (29%)	32 (36%)
Infiltration cellular, mononuclear cell		1 (1%)		
Artery, inflammation, chronic		1 (1%)	1 (1%)	
Preputial gland	(89)	(89)	(89)	(89)
Atrophy		1 (1%)		
Infiltration cellular, lymphocyte	43 (48%)	40 (45%)	39 (44%)	38 (43%)
Inflammation, suppurative	1 (1%)			
Inflammation, chronic active	1 (1%)	3 (3%)	3 (3%)	1 (1%)
Bilateral, inflammation, chronic active				1 (1%)
Bilateral, duct, dilation	6 (7%)	4 (4%)	5 (6%)	3 (3%)
Duct, dilation	10 (11%)	10 (11%)	5 (6%)	5 (6%)
Duct, inflammation, chronic active				1 (1%)
Duct, necrosis	1 (1%)			

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Genital System (continued)				
Prostate	(90)	(86)	(90)	(88)
Infiltration cellular, lymphocyte	4 (4%)	8 (9%)	10 (11%)	9 (10%)
Inflammation, acute			1 (1%)	
Inflammation, chronic active	1 (1%)			
Arteriole, inflammation, chronic		1 (1%)		
Seminal vesicle	(90)	(90)	(90)	(90)
Dilation	4 (4%)	9 (10%)	4 (4%)	2 (2%)
Inflammation, chronic active	1 (1%)			1 (1%)
Bilateral, dilation	27 (30%)	19 (21%)	26 (29%)	14 (16%)
Testis	(90)	(91)	(88)	(90)
Bilateral, germinal epithelium, atrophy			1 (1%)	
Germ cell, degeneration	2 (2%)	2 (2%)	4 (5%)	7 (8%)
Seminiferous tubule, necrosis				1 (1%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hypercellularity	3 (3%)		2 (2%)	3 (3%)
Myeloid cell, hypercellularity			1 (1%)	
Lymph node	(6)	(6)	(11)	(10)
Bronchial, hyperplasia, lymphoid				2 (20%)
Iliac, erythrophagocytosis		1 (17%)		
Iliac, hemorrhage			1 (9%)	
Iliac, hyperplasia, lymphoid		2 (33%)	4 (36%)	
Iliac, infiltration cellular, histiocyte				1 (10%)
Iliac, pigment			2 (18%)	1 (10%)
Inguinal, hyperplasia, lymphoid				2 (20%)
Mediastinal, hyperplasia, lymphoid		2 (33%)	1 (9%)	2 (20%)
Renal, hemorrhage	1 (17%)			
Renal, pigment			1 (9%)	
Lymph node, mandibular	(72)	(70)	(63)	(64)
Hemorrhage			1 (2%)	1 (2%)
Hyperplasia, lymphoid	2 (3%)		1 (2%)	2 (3%)
Infiltration cellular, histiocyte	1 (1%)	1 (1%)	1 (2%)	2 (3%)
Infiltration cellular, plasma cell		1 (1%)		
Lymph node, mesenteric	(85)	(88)	(86)	(85)
Erythrophagocytosis	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Hemorrhage	10 (12%)	21 (24%)	16 (19%)	14 (16%)
Hyperplasia, lymphoid	4 (5%)	3 (3%)	5 (6%)	6 (7%)
Infiltration cellular, histiocyte	8 (9%)	7 (8%)	5 (6%)	5 (6%)
Infiltration cellular, mixed cell			1 (1%)	
Infiltration cellular, plasma cell	1 (1%)		1 (1%)	
Spleen	(87)	(89)	(87)	(86)
Extramedullary hematopoiesis	15 (17%)	7 (8%)	21 (24%)	16 (19%)
Hyperplasia, lymphoid	5 (6%)	4 (4%)	3 (3%)	9 (10%)
Thymus	(75)	(76)	(80)	(81)
Atrophy	11 (15%)	3 (4%)	8 (10%)	5 (6%)
Cyst	11 (15%)	17 (22%)	19 (24%)	19 (23%)
Hemorrhage	1 (1%)			1 (1%)
Hyperplasia, lymphoid		2 (3%)		

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Integumentary System				
Mammary gland	(2)	(1)	(0)	(3)
Skin	(90)	(91)	(90)	(90)
Infiltration cellular, mixed cell	1 (1%)			
Inflammation, chronic				1 (1%)
Ulcer	2 (2%)	1 (1%)	2 (2%)	4 (4%)
Epidermis, hyperplasia, focal			1 (1%)	2 (2%)
Musculoskeletal System				
Bone	(90)	(91)	(90)	(90)
Fibro-osseous lesion			1 (1%)	
Increased bone				1 (1%)
Skeletal muscle	(90)	(91)	(90)	(90)
Degeneration	1 (1%)	1 (1%)		
Infiltration cellular, lymphocyte	3 (3%)	1 (1%)	1 (1%)	4 (4%)
Inflammation, granulomatous				1 (1%)
Inflammation, acute			1 (1%)	
Nervous System				
Brain	(90)	(91)	(90)	(90)
Hemorrhage	2 (2%)			
Infiltration cellular, lymphocyte	1 (1%)			
Mineral	79 (88%)	80 (88%)	77 (86%)	77 (86%)
Necrosis		1 (1%)		
Artery, meninges, inflammation, chronic active	1 (1%)	2 (2%)	3 (3%)	1 (1%)
Meninges, inflammation, chronic			1 (1%)	
Brain trigeminal ganglion	(69)	(79)	(80)	(80)
Nerve trigeminal	(67)	(57)	(43)	(55)
Peripheral nerve, sciatic	(89)	(91)	(87)	(88)
Inflammation, chronic active				1 (1%)
Axon, degeneration	9 (10%)	5 (5%)	4 (5%)	11 (13%)
Spinal cord	(90)	(91)	(90)	(90)
Degeneration		1 (1%)		
Necrosis	1 (1%)	1 (1%)	1 (1%)	
Squamous cyst				2 (2%)
Artery, meninges, inflammation, chronic active	1 (1%)	2 (2%)	4 (4%)	1 (1%)
Respiratory System				
Lung	(90)	(91)	(90)	(90)
Congestion	2 (2%)		3 (3%)	2 (2%)
Hemorrhage	3 (3%)	2 (2%)	4 (4%)	1 (1%)
Infiltration cellular, histiocyte	6 (7%)	5 (5%)	3 (3%)	5 (6%)
Infiltration cellular, lymphocyte	1 (1%)	1 (1%)	3 (3%)	
Infiltration, mononuclear cell			1 (1%)	3 (3%)
Inflammation, granulomatous				1 (1%)
Alveolar epithelium, hyperplasia, focal	6 (7%)	7 (8%)	8 (9%)	5 (6%)
Bronchiole, foreign body	1 (1%)			
Bronchiole, inflammation, suppurative	1 (1%)			

TABLE C4
Summary of the Incidence of Nonneoplastic Lesions in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System (continued)				
Nose	(90)	(91)	(90)	(90)
Infiltration cellular, lymphocyte			1 (1%)	
Inflammation, acute	1 (1%)			
Respiratory epithelium, accumulation, hyaline droplet	1 (1%)			1 (1%)
Respiratory epithelium, hyperplasia	5 (6%)	1 (1%)		
Vomeronasal organ, fibrosis	1 (1%)			
Trachea	(90)	(90)	(90)	(89)
Special Senses System				
Eye	(90)	(91)	(89)	(90)
Atrophy			1 (1%)	1 (1%)
Bilateral, cornea, inflammation, chronic active				1 (1%)
Bilateral, iris, synechia				1 (1%)
Cornea, edema		1 (1%)		
Cornea, fibrosis	1 (1%)			
Cornea, hyperplasia, squamous, diffuse		1 (1%)		
Sclera, inflammation, acute			1 (1%)	
Harderian gland	(88)	(91)	(90)	(88)
Hemorrhage	1 (1%)			
Hyperplasia, focal	2 (2%)	3 (3%)	1 (1%)	
Infiltration cellular, lymphocyte	36 (41%)	41 (45%)	40 (44%)	38 (43%)
Mineral		1 (1%)		
Urinary System				
Kidney	(90)	(89)	(90)	(90)
Infarct	7 (8%)	6 (7%)	11 (12%)	7 (8%)
Inflammation, suppurative	1 (1%)			
Inflammation, granulomatous	1 (1%)			
Metaplasia, osseous	3 (3%)	8 (9%)	3 (3%)	5 (6%)
Mineral		3 (3%)	1 (1%)	1 (1%)
Nephropathy, chronic progressive	74 (82%)	84 (94%)	81 (90%)	77 (86%)
Artery, inflammation, chronic active		1 (1%)		
Bilateral, bacteria			1 (1%)	
Bilateral, infarct		1 (1%)		2 (2%)
Bilateral, inflammation, acute			1 (1%)	
Bilateral, renal tubule, pigment				1 (1%)
Glomerulus, cyst	1 (1%)	2 (2%)		1 (1%)
Interstitium, infiltration cellular, lymphocyte	41 (46%)	55 (62%)	57 (63%)	40 (44%)
Interstitium, inflammation, acute				1 (1%)
Papilla, bacteria			1 (1%)	
Papilla, inflammation, acute			1 (1%)	
Pelvis, dilation	1 (1%)			2 (2%)
Pelvis, inflammation, acute				1 (1%)
Renal tubule, cyst	8 (9%)	11 (12%)	7 (8%)	10 (11%)
Renal tubule, hyperplasia, focal		1 (1%)		
Renal tubule, mineral	1 (1%)	4 (4%)	4 (4%)	5 (6%)
Urothelium, inflammation, chronic active	1 (1%)			
Ureter	(0)	(0)	(1)	(0)
Inflammation, chronic active			1 (100%)	
Urinary bladder	(87)	(90)	(90)	(89)
Hemorrhage	3 (3%)			
Infiltration cellular, lymphocyte	26 (30%)	33 (37%)	39 (43%)	41 (46%)

APPENDIX D
SUMMARY OF LESIONS IN FEMALE MICE EXPOSED TO CDMA-MODULATED CELL PHONE RFR FOR 2 YEARS

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	104	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Moribund	9	5	4	4
Natural deaths	14	9	16	14
Survivors				
Died last week of study	1	3	1	1
Terminal euthanasia	66	72	69	71
Animals examined microscopically	100	99	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
2-Year Study				
Alimentary System				
Esophagus	(87)	(88)	(87)	(87)
Gallbladder	(79)	(75)	(72)	(73)
Intestine large, cecum	(84)	(82)	(80)	(81)
Fibrosarcoma, metastatic, skin	1 (1%)			
Intestine large, colon	(84)	(85)	(85)	(86)
Intestine large, rectum	(88)	(86)	(84)	(88)
Fibrosarcoma, metastatic, skin	1 (1%)			
Osteosarcoma, metastatic, skin	1 (1%)			
Intestine small, duodenum	(82)	(81)	(80)	(77)
Fibrosarcoma, metastatic, skin	1 (1%)			
Intestine small, ileum	(83)	(82)	(76)	(81)
Intestine small, jejunum	(84)	(81)	(80)	(77)
Liver	(89)	(88)	(90)	(90)
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangiosarcoma		1 (1%)		
Hepatoblastoma	1 (1%)			
Hepatocellular adenoma	14 (16%)	20 (23%)	17 (19%)	13 (14%)
Hepatocellular adenoma, multiple	5 (6%)	4 (5%)	5 (6%)	7 (8%)
Hepatocellular carcinoma	6 (7%)	5 (6%)	3 (3%)	5 (6%)
Hepatocellular carcinoma, multiple	2 (2%)		2 (2%)	
Hepatocholangiocarcinoma			1 (1%)	
Osteosarcoma, metastatic, bone	1 (1%)			1 (1%)
Osteosarcoma, metastatic, brain		1 (1%)		
Osteosarcoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skeletal muscle		1 (1%)		

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Mesentery	(29)	(24)	(34)	(24)
Fibrosarcoma, metastatic, skin	1 (3%)			
Leiomyosarcoma, metastatic, uterus				1 (4%)
Renal mesenchymal tumor, metastatic, kidney	1 (3%)			
Sarcoma, metastatic, skeletal muscle		1 (4%)		
Fat, hemangioma	1 (3%)			
Fat, lipoma		2 (8%)	1 (3%)	
Pancreas	(87)	(86)	(85)	(84)
Fibrosarcoma, metastatic, skin	1 (1%)			
Leiomyosarcoma, metastatic, uterus				1 (1%)
Renal mesenchymal tumor, metastatic, kidney	1 (1%)			
Acinus, adenoma			1 (1%)	
Salivary glands	(89)	(88)	(87)	(89)
Stomach, forestomach	(86)	(88)	(87)	(87)
Fibrosarcoma, metastatic, skin	1 (1%)			
Leiomyosarcoma, metastatic, uterus				1 (1%)
Squamous cell papilloma	1 (1%)	1 (1%)		1 (1%)
Stomach, glandular	(85)	(88)	(85)	(83)
Fibrosarcoma, metastatic, skin	1 (1%)			
Cardiovascular System				
Aorta	(84)	(87)	(89)	(90)
Heart	(90)	(89)	(90)	(90)
Osteosarcoma, metastatic, bone			1 (1%)	1 (1%)
Osteosarcoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skeletal muscle		1 (1%)		
Endocrine System				
Adrenal cortex	(84)	(88)	(87)	(88)
Adenoma	1 (1%)			
Adrenal medulla	(83)	(87)	(84)	(84)
Pheochromocytoma benign				1 (1%)
Pheochromocytoma malignant	2 (2%)			
Bilateral, pheochromocytoma benign			1 (1%)	
Islets, pancreatic	(87)	(88)	(89)	(87)
Adenoma				1 (1%)
Carcinoma	1 (1%)			
Parathyroid gland	(60)	(59)	(65)	(68)
Pituitary gland	(80)	(79)	(88)	(86)
Pars distalis, adenoma	6 (8%)	8 (10%)	8 (9%)	1 (1%)
Pars distalis, carcinoma				1 (1%)
Pars distalis, fibrosarcoma, metastatic, skin				1 (1%)
Thyroid gland	(86)	(87)	(88)	(88)
C-cell carcinoma	1 (1%)			
Follicular cell, adenoma		2 (2%)	1 (1%)	1 (1%)
General Body System				
Tissue NOS	(1)	(1)	(1)	(0)
Hemangiosarcoma		1 (100%)		
Abdominal, osteosarcoma, metastatic, skin	1 (100%)			

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Genital System				
Clitoral gland	(82)	(82)	(81)	(82)
Ovary	(75)	(84)	(84)	(83)
Adenocarcinoma, metastatic, uterus		2 (2%)		
Cystadenoma	2 (3%)	2 (2%)	6 (7%)	6 (7%)
Granulosa cell tumor benign	1 (1%)			
Hemangioma	2 (3%)			1 (1%)
Luteoma			2 (2%)	1 (1%)
Teratoma benign			1 (1%)	
Thecoma malignant	1 (1%)			
Tubulostromal adenoma			1 (1%)	
Uterus	(89)	(89)	(88)	(90)
Adenocarcinoma		2 (2%)		1 (1%)
Adenoma			1 (1%)	
Fibroma	1 (1%)			
Fibrosarcoma, metastatic, skin	1 (1%)			
Granular cell tumor malignant		1 (1%)		
Hemangiosarcoma		1 (1%)		1 (1%)
Leiomyoma	1 (1%)			
Leiomyosarcoma		1 (1%)	1 (1%)	2 (2%)
Polyp stromal				1 (1%)
Hematopoietic System				
Bone marrow	(90)	(89)	(89)	(89)
Hemangiosarcoma			1 (1%)	
Lymph node	(18)	(21)	(18)	(18)
Bronchial, adenocarcinoma, metastatic, uterus		1 (5%)		
Bronchial, alveolar/bronchiolar carcinoma, metastatic, lung	1 (6%)			
Bronchial, fibrosarcoma, metastatic, skin	1 (6%)			
Iliac, hemangiosarcoma		1 (5%)		
Lumbar, leiomyosarcoma, metastatic, uterus				1 (6%)
Pancreatic, adenocarcinoma, metastatic, uterus		1 (5%)		
Lymph node, mandibular	(76)	(79)	(76)	(73)
Lymph node, mesenteric	(71)	(86)	(75)	(81)
Adenocarcinoma, metastatic, uterus		1 (1%)		
Fibrosarcoma, metastatic, skin	1 (1%)			
Hemangiosarcoma		1 (1%)		
Leiomyosarcoma, metastatic, uterus				1 (1%)
Renal mesenchymal tumor, metastatic, kidney	1 (1%)			
Sarcoma, metastatic, skeletal muscle		1 (1%)		
Spleen	(86)	(87)	(86)	(88)
Hemangiosarcoma		3 (3%)	2 (2%)	1 (1%)
Leiomyosarcoma, metastatic, uterus				1 (1%)
Thymus	(85)	(83)	(82)	(82)
Osteosarcoma, metastatic, bone			1 (1%)	

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Integumentary System				
Mammary gland	(85)	(87)	(90)	(88)
Adenocarcinoma				2 (2%)
Adenoma		1 (1%)	2 (2%)	
Skin	(90)	(89)	(90)	(90)
Squamous cell carcinoma				1 (1%)
Subcutaneous tissue, fibroma	1 (1%)			
Subcutaneous tissue, fibroma, multiple				1 (1%)
Subcutaneous tissue, fibrosarcoma	3 (3%)	1 (1%)	3 (3%)	2 (2%)
Subcutaneous tissue, fibrosarcoma, multiple				1 (1%)
Subcutaneous tissue, hemangioma		1 (1%)		
Subcutaneous tissue, hemangiosarcoma	2 (2%)			1 (1%)
Subcutaneous tissue, lipoma	1 (1%)			
Subcutaneous tissue, malignant fibrous histiocytoma		1 (1%)	1 (1%)	
Subcutaneous tissue, osteosarcoma	1 (1%)			
Subcutaneous tissue, sarcoma	2 (2%)			
Musculoskeletal System				
Bone	(90)	(89)	(90)	(89)
Hemangioma	1 (1%)			
Osteosarcoma	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Skeletal muscle	(89)	(89)	(90)	(90)
Adenocarcinoma, metastatic, uterus		1 (1%)		
Hemangiosarcoma		2 (2%)		
Leiomyosarcoma, metastatic, uterus				1 (1%)
Osteosarcoma	1 (1%)			
Sarcoma		1 (1%)		
Nervous System				
Brain	(87)	(88)	(90)	(90)
Carcinoma, metastatic, pituitary gland				1 (1%)
Fibrosarcoma, metastatic, skin				1 (1%)
Osteosarcoma, metastatic, skeletal muscle	1 (1%)			
Osteosarcoma, metastatic, uncertain primary site		1 (1%)		
Brain trigeminal ganglion	(75)	(82)	(75)	(74)
Nerve trigeminal	(56)	(30)	(52)	(51)
Peripheral nerve, sciatic	(88)	(88)	(89)	(88)
Spinal cord	(90)	(89)	(90)	(90)
Osteosarcoma, metastatic, bone				1 (1%)

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(89)	(90)	(90)
Alveolar/bronchiolar adenoma	3 (3%)	6 (7%)	4 (4%)	1 (1%)
Alveolar/bronchiolar adenoma, multiple				1 (1%)
Alveolar/bronchiolar carcinoma	3 (3%)	3 (3%)	3 (3%)	5 (6%)
Carcinoma, metastatic, thyroid gland	1 (1%)			
Fibrosarcoma, metastatic, skin	1 (1%)		1 (1%)	
Granular cell tumor malignant, metastatic, uterus		1 (1%)		
Hepatocellular carcinoma, metastatic, liver	2 (2%)		1 (1%)	
Osteosarcoma, metastatic, bone	1 (1%)	1 (1%)	1 (1%)	2 (2%)
Osteosarcoma, metastatic, brain		1 (1%)		
Osteosarcoma, metastatic, skeletal muscle	1 (1%)			
Osteosarcoma, metastatic, skin	1 (1%)			
Sarcoma, metastatic, skeletal muscle		1 (1%)		
Squamous cell carcinoma, metastatic, skin				1 (1%)
Mediastinum	(2)	(0)	(0)	(0)
Hepatocellular carcinoma, metastatic, liver	1 (50%)			
Nose	(89)	(89)	(90)	(90)
Respiratory epithelium, adenoma				1 (1%)
Trachea	(90)	(87)	(89)	(88)
Special Senses System				
Eye	(89)	(89)	(90)	(89)
Harderian gland	(89)	(88)	(89)	(89)
Adenocarcinoma		1 (1%)	1 (1%)	2 (2%)
Adenoma	4 (4%)	8 (9%)	8 (9%)	4 (4%)
Urinary System				
Kidney	(89)	(89)	(88)	(87)
Adenocarcinoma, metastatic, uterus		1 (1%)		
Renal mesenchymal tumor	1 (1%)			
Renal tubule, adenoma	2 (2%)			
Urinary bladder	(86)	(86)	(83)	(85)
Systemic Lesions				
Multiple organs[b]	(90)	(89)	(90)	(90)
Histiocytic sarcoma	8 (9%)	3 (3%)	2 (2%)	7 (8%)
Leukemia erythrocytic		1 (1%)		
Leukemia granulocytic			2 (2%)	
Lymphoma malignant	2 (2%)	9 (10%)	6 (7%)	7 (8%)

TABLE D1
Summary of the Incidence of Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Neoplasm Summary				
Total animals with primary neoplasms[c]				
2-Year study	59	62	56	56
Total primary neoplasms				
2-Year study	85	96	88	83
Total animals with benign neoplasms				
2-Year study	36	42	43	32
Total benign neoplasms				
2-Year study	47	55	59	42
Total animals with malignant neoplasms				
2-Year study	33	32	26	36
Total malignant neoplasms				
2-Year study	38	41	29	41
Total animals with metastatic neoplasms				
2-Year study	9	6	3	6
Total metastatic neoplasms				
2-Year study	29	17	5	16
Total animals with malignant neoplasms-of uncertain primary site				
2-Year study		1		

[a] Number of animals examined microscopically at the site and the number of animals with neoplasm
[b] Number of animals with any tissue examined microscopically
[c] Primary neoplasms: all neoplasms except metastatic neoplasms

TABLE D2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Harderian Gland: Adenoma				
Overall rate[a]	4/90 (4%)	8/89 (9%)	8/90 (9%)	4/90 (4%)
Adjusted rate[b]	5.0%	9.5%	9.6%	4.8%
Terminal rate[c]	4/67 (6%)	8/74 (11%)	7/69 (10%)	4/71 (6%)
First incidence (days)	739 (T)	739 (T)	704	739 (T)
Poly-3 test[d]	P=0.415N	P=0.208	P=0.208	P=0.621N
Harderian Gland: Adenoma or Carcinoma				
Overall rate	4/90 (4%)	9/89 (10%)	9/90 (10%)	6/90 (7%)
Adjusted rate	5.0%	10.7%	10.7%	7.2%
Terminal rate	4/67 (6%)	9/74 (12%)	8/69 (12%)	5/71 (7%)
First incidence (days)	739 (T)	739 (T)	704	653
Poly-3 test	P=0.482	P=0.143	P=0.143	P=0.397
Liver: Hepatocellular Adenoma				
Overall rate	19/89 (21%)[e]	24/88 (27%)	22/90 (24%)	20/90 (22%)
Adjusted rate	23.6%	28.8%	26.0%	24.1%
Terminal rate	17/67 (25%)	22/73 (30%)	17/69 (25%)	18/71 (25%)
First incidence (days)	511	579	644	679
Poly-3 test	P=0.466N	P=0.282	P=0.429	P=0.543
Liver: Hepatocellular Carcinoma				
Overall rate	8/89 (9%)	5/88 (6%)	5/90 (6%)	5/90 (6%)
Adjusted rate	10.0%	6.0%	6.0%	6.0%
Terminal rate	7/67 (10%)	3/73 (4%)	3/69 (4%)	5/71 (7%)
First incidence (days)	656	639	692	739 (T)
Poly-3 test	P=0.255N	P=0.255N	P=0.251N	P=0.259N
Liver: Hepatocellular Adenoma or Carcinoma				
Overall rate	25/89 (28%)	29/88 (33%)	26/90 (29%)	22/90 (24%)
Adjusted rate	30.9%	34.5%	30.6%	26.5%
Terminal rate	22/67 (33%)	25/73 (34%)	19/69 (28%)	20/71 (28%)
First incidence (days)	511	579	644	679
Poly-3 test	P=0.217N	P=0.371	P=0.552N	P=0.325N
Liver: Hepatocellular Carcinoma or Hepatoblastoma				
Overall rate	9/89 (10%)	5/88 (6%)	5/90 (6%)	5/90 (6%)
Adjusted rate	11.3%	6.0%	6.0%	6.0%
Terminal rate	8/67 (12%)	3/73 (4%)	3/69 (4%)	5/71 (7%)
First incidence (days)	656	639	692	739 (T)
Poly-3 test	P=0.186N	P=0.178N	P=0.174N	P=0.181N
Lung: Alveolar/bronchiolar Adenoma				
Overall rate	3/90 (3%)	6/89 (7%)	4/90 (4%)	2/90 (2%)
Adjusted rate	3.8%	7.2%	4.8%	2.4%
Terminal rate	3/67 (5%)	5/74 (7%)	4/69 (6%)	2/71 (3%)
First incidence (days)	739 (T)	738	739 (T)	739 (T)
Poly-3 test	P=0.262N	P=0.271	P=0.525	P=0.484N
Lung: Alveolar/bronchiolar Carcinoma				
Overall rate	3/90 (3%)	3/89 (3%)	3/90 (3%)	5/90 (6%)
Adjusted rate	3.7%	3.6%	3.6%	6.0%
Terminal rate	2/67 (3%)	3/74 (4%)	1/69 (1%)	4/71 (6%)
First incidence (days)	607	739 (T)	511	684
Poly-3 test	P=0.270	P=0.641N	P=0.638N	P=0.377

TABLE D2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Lung: Alveolar/bronchiolar Adenoma or Carcinoma				
Overall rate	6/90 (7%)	9/89 (10%)	7/90 (8%)	6/90 (7%)
Adjusted rate	7.5%	10.7%	8.3%	7.2%
Terminal rate	5/67 (8%)	8/74 (11%)	5/69 (7%)	5/71 (7%)
First incidence (days)	607	738	511	684
Poly-3 test	P=0.423N	P=0.325	P=0.536	P=0.595N
Ovary: Cystadenoma				
Overall rate	2/75 (3%)	2/84 (2%)	6/84 (7%)	6/83 (7%)
Adjusted rate	3.0%	2.5%	7.6%	7.9%
Terminal rate	2/56 (4%)	2/70 (3%)	6/66 (9%)	5/65 (8%)
First incidence (days)	739 (T)	739 (T)	739 (T)	597
Poly-3 test	P=0.077	P=0.627N	P=0.202	P=0.189
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	6/80 (8%)	8/79 (10%)	8/88 (9%)	1/86 (1%)
Adjusted rate	8.4%	10.7%	9.8%	1.3%
Terminal rate	5/60 (8%)	8/67 (12%)	6/68 (9%)	1/68 (2%)
First incidence (days)	703	739 (T)	704	739 (T)
Poly-3 test	P=0.029N	P=0.430	P=0.499	P=0.043N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	6/80 (8%)	8/79 (10%)	8/88 (9%)	2/86 (2%)
Adjusted rate	8.4%	10.7%	9.8%	2.5%
Terminal rate	5/60 (8%)	8/67 (12%)	6/68 (9%)	2/68 (3%)
First incidence (days)	703	739 (T)	704	739 (T)
Poly-3 test	P=0.063N	P=0.430	P=0.499	P=0.104N
Skin (Subcutaneous Tissue): Fibrosarcoma or Sarcoma				
Overall rate	5/90 (6%)	1/89 (1%)	3/90 (3%)	3/90 (3%)
Adjusted rate	6.2%	1.2%	3.6%	3.6%
Terminal rate	1/67 (2%)	0/74 (0%)	1/69 (1%)	2/71 (3%)
First incidence (days)	607	715	669	731
Poly-3 test	P=0.422N	P=0.097N	P=0.338N	P=0.346N
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, or Sarcoma				
Overall rate	6/90 (7%)	1/89 (1%)	3/90 (3%)	3/90 (3%)
Adjusted rate	7.4%	1.2%	3.6%	3.6%
Terminal rate	2/67 (3%)	0/74 (0%)	1/69 (1%)	2/71 (3%)
First incidence (days)	607	715	669	731
Poly-3 test	P=0.301N	P=0.054N	P=0.228N	P=0.235N
All Organs: Hemangiosarcoma				
Overall rate	2/90 (2%)	7/89 (8%)	2/90 (2%)	3/90 (3%)
Adjusted rate	2.5%	8.3%	2.4%	3.6%
Terminal rate	1/67 (2%)	4/74 (5%)	0/69 (0%)	2/71 (3%)
First incidence (days)	703	626	644	653
Poly-3 test	P=0.436N	P=0.098	P=0.674N	P=0.517
All Organs: Hemangioma or Hemangiosarcoma				
Overall rate	6/90 (7%)	8/89 (9%)	2/90 (2%)	4/90 (4%)
Adjusted rate	7.5%	9.5%	2.4%	4.8%
Terminal rate	5/67 (8%)	5/74 (7%)	0/69 (0%)	3/71 (4%)
First incidence (days)	703	626	644	653
Poly-3 test	P=0.161N	P=0.431	P=0.122N	P=0.349N

TABLE D2
Statistical Analysis of Primary Neoplasms in Female Mice
Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
All Organs: Histiocytic Sarcoma				
Overall rate	8/90 (9%)	3/89 (3%)	2/90 (2%)	7/90 (8%)
Adjusted rate	9.7%	3.5%	2.4%	8.4%
Terminal rate	2/67 (3%)	1/74 (1%)	0/69 (0%)	5/71 (7%)
First incidence (days)	562	493	725	675
Poly-3 test	P=0.558	P=0.098N	P=0.048N	P=0.494N
All Organs: Malignant Lymphoma				
Overall rate	2/90 (2%)	9/89 (10%)	6/90 (7%)	7/90 (8%)
Adjusted rate	2.5%	10.7%	7.2%	8.4%
Terminal rate	1/67 (2%)	8/74 (11%)	4/69 (6%)	4/71 (6%)
First incidence (days)	604	689	716	635
Poly-3 test	P=0.220	P=0.035	P=0.152	P=0.094

(T) Terminal euthanasia

[a] Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for liver, lung, ovary, and pituitary gland; for other tissues, denominator is number of animals necropsied.

[b] Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality

[c] Observed incidence at terminal euthanasia

[d] Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. A negative trend or a lower incidence in an exposure group is indicated by **N**.

[e] A single incidence of hepatoblastoma occurred in an animal that also had an adenoma.

TABLE D3
Historical Incidence of Malignant Lymphoma in Control Female B6C3F1/N Mice[a]

	Incidence in Controls
Overall Historical Incidence: All Routes	
Total (%)	89/590 (15.1%)
Mean ± standard deviation	16.0% ± 8.3%
Range	2%-36%

[a] Data as of August 2017; includes data for histiocytic, lymphocytic, mixed, unspecified, or undifferentiated cell types

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Disposition Summary				
Animals initially in study	105	104	105	105
14-Week interim evaluation	15	15	15	15
Early deaths				
Moribund	9	5	4	4
Natural deaths	14	9	16	14
Survivors				
Died last week of study	1	3	1	1
Terminal euthanasia	66	72	69	71
Animals examined microscopically	100	99	100	100
14-Week Interim Evaluation				
Alimentary System				
Liver	(10)	(10)	(10)	(10)
Inflammation, focal	1 (10%)		3 (30%)	2 (20%)
Necrosis, focal			1 (10%)	
Endocrine System				
Thyroid gland	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte	1 (10%)			
Hematopoietic System				
Lymph node, mandibular	(8)	(8)	(9)	(10)
Hemorrhage			1 (11%)	
Thymus	(10)	(10)	(10)	(10)
Hemorrhage			1 (10%)	2 (20%)
Nervous System				
Spinal cord	(10)	(10)	(10)	(10)
Cyst, squamous		1 (10%)		
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Hemorrhage			1 (10%)	
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Retina, dysplasia		1 (10%)		
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive		1 (10%)		
Interstitium, infiltration cellular, lymphocyte		1 (10%)	1 (10%)	5 (50%)
Urinary bladder	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte			1 (10%)	2 (20%)

[a] Number of animals examined microscopically at the site and the number of animals with lesion

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
14-Week Interim Evaluation (continued)				
Systems Examined with No Lesions Observed				
Cardiovascular System				
General Body System				
Genital System				
Integumentary System				
Musculoskeletal System				
2-Year Study				
Alimentary System				
Esophagus	(87)	(88)	(87)	(87)
Gallbladder	(79)	(75)	(72)	(73)
Cyst			1 (1%)	
Infiltration cellular, lymphocyte	2 (3%)	4 (5%)	2 (3%)	3 (4%)
Epithelium, hyperplasia, diffuse			1 (1%)	
Intestine large, cecum	(84)	(82)	(80)	(81)
Intestine large, colon	(84)	(85)	(85)	(86)
Intestine large, rectum	(88)	(86)	(84)	(88)
Intestine small, duodenum	(82)	(81)	(80)	(77)
Inflammation, acute	1 (1%)			
Intestine small, ileum	(83)	(82)	(76)	(81)
Inflammation, suppurative		1 (1%)		
Peyer's patch, hyperplasia, lymphoid		2 (2%)		
Intestine small, jejunum	(84)	(81)	(80)	(77)
Peyer's patch, hyperplasia, lymphoid	1 (1%)	2 (2%)		
Liver	(89)	(88)	(90)	(90)
Angiectasis				1 (1%)
Basophilic focus	4 (4%)	4 (5%)	3 (3%)	1 (1%)
Clear cell focus	1 (1%)			
Eosinophilic focus	2 (2%)		2 (2%)	2 (2%)
Extramedullary hematopoiesis	1 (1%)	2 (2%)		2 (2%)
Fatty change	7 (8%)	1 (1%)	2 (2%)	9 (10%)
Hemorrhage	1 (1%)		1 (1%)	1 (1%)
Infiltration cellular, lymphocyte	33 (37%)	22 (25%)	26 (29%)	24 (27%)
Infiltration cellular, mononuclear cell	1 (1%)			
Inflammation, focal	4 (4%)	2 (2%)		
Inflammation, chronic active	1 (1%)			2 (2%)
Mixed cell focus	5 (6%)			
Necrosis	6 (7%)	2 (2%)	3 (3%)	4 (4%)
Centrilobular, necrosis				1 (1%)
Hepatocyte, fatty change, focal	3 (3%)		1 (1%)	
Hepatocyte, hypertrophy			1 (1%)	
Kupffer cell, hyperplasia	1 (1%)			
Oval cell, hyperplasia			1 (1%)	
Mesentery	(29)	(24)	(34)	(24)
Artery, inflammation, chronic			1 (3%)	
Fat, infiltration cellular, lymphocyte	2 (7%)		1 (3%)	1 (4%)
Fat, inflammation, chronic				2 (8%)
Fat, inflammation, chronic active	1 (3%)			2 (8%)
Fat, mineral		1 (4%)	2 (6%)	
Fat, necrosis	25 (86%)	22 (92%)	30 (88%)	19 (79%)

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Alimentary System (continued)				
Pancreas	(87)	(86)	(85)	(84)
Infiltration cellular, lymphocyte	27 (31%)	23 (27%)	21 (25%)	19 (23%)
Inflammation, chronic active	1 (1%)			
Necrosis		1 (1%)		2 (2%)
Acinus, atrophy			1 (1%)	2 (2%)
Duct, cyst	1 (1%)		3 (4%)	1 (1%)
Salivary glands	(89)	(88)	(87)	(89)
Atrophy	1 (1%)			
Infiltration cellular, lymphocyte	59 (66%)	61 (69%)	54 (62%)	60 (67%)
Stomach, forestomach	(86)	(88)	(87)	(87)
Cyst			1 (1%)	
Epithelium, hyperplasia, focal			1 (1%)	1 (1%)
Stomach, glandular	(85)	(88)	(85)	(83)
Cyst	3 (4%)	2 (2%)		
Infiltration cellular, lymphocyte			2 (2%)	
Cardiovascular System				
Aorta	(84)	(87)	(89)	(90)
Degeneration			1 (1%)	
Inflammation, acute			1 (1%)	
Inflammation, chronic active	1 (1%)			
Thrombus				1 (1%)
Heart	(90)	(89)	(90)	(90)
Bacteria	1 (1%)		1 (1%)	2 (2%)
Cardiomyopathy	3 (3%)	4 (4%)	4 (4%)	6 (7%)
Thrombus	3 (3%)			2 (2%)
Artery, inflammation, chronic active		2 (2%)	1 (1%)	
Endothelium, hyperplasia		1 (1%)		
Epicardium, infiltration cellular, mixed cell	1 (1%)			
Epicardium, infiltration cellular, mononuclear cell	1 (1%)			
Myocardium, fibrosis	1 (1%)			1 (1%)
Myocardium, hemorrhage		1 (1%)		
Myocardium, inflammation, acute				2 (2%)
Myocardium, inflammation, chronic active	1 (1%)	1 (1%)	2 (2%)	
Myocardium, mineral	4 (4%)		1 (1%)	2 (2%)
Myocardium, necrosis			1 (1%)	
Valve, hemorrhage	1 (1%)			
Valve, infiltration cellular, lymphocyte	1 (1%)			
Valve, thrombus	1 (1%)		1 (1%)	
Endocrine System				
Adrenal cortex	(84)	(88)	(87)	(88)
Accessory adrenal cortical nodule			2 (2%)	3 (3%)
Angiectasis	1 (1%)			
Hemorrhage	1 (1%)		1 (1%)	1 (1%)
Hyperplasia, focal		1 (1%)		
Infiltration cellular, mixed cell			1 (1%)	
Mineral	1 (1%)			
Vacuolization cytoplasmic		1 (1%)		
Vacuolization cytoplasmic, focal				1 (1%)

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Endocrine System (continued)				
Adrenal cortex (continued)	(84)	(88)	(87)	(88)
Bilateral, extramedullary hematopoiesis	1 (1%)			
Bilateral, infiltration cellular, mixed cell			1 (1%)	
Bilateral, vacuolization cytoplasmic		1 (1%)		2 (2%)
Subcapsular, hyperplasia	81 (96%)	84 (95%)	84 (97%)	86 (98%)
Adrenal medulla	(83)	(87)	(84)	(84)
Hemorrhage	2 (2%)			
Hyperplasia		2 (2%)	1 (1%)	
Mineral	1 (1%)			
Islets, pancreatic	(87)	(88)	(89)	(87)
Hyperplasia	1 (1%)		1 (1%)	2 (2%)
Infiltration cellular, lymphocyte	3 (3%)	3 (3%)	5 (6%)	1 (1%)
Parathyroid gland	(60)	(59)	(65)	(68)
Cyst	1 (2%)			
Infiltration cellular, lymphocyte		1 (2%)		1 (1%)
Pituitary gland	(80)	(79)	(88)	(86)
Pars distalis, angiectasis	2 (3%)	9 (11%)	4 (5%)	2 (2%)
Pars distalis, cyst	1 (1%)	1 (1%)	1 (1%)	2 (2%)
Pars distalis, hyperplasia, focal	2 (3%)	2 (3%)	4 (5%)	6 (7%)
Thyroid gland	(86)	(87)	(88)	(88)
Infiltration cellular, lymphocyte	1 (1%)	1 (1%)	7 (8%)	5 (6%)
Inflammation, chronic active		1 (1%)		
Follicle, cyst	1 (1%)		2 (2%)	3 (3%)
General Body System				
Tissue NOS	(1)	(1)	(1)	(0)
Genital System				
Clitoral gland	(82)	(82)	(81)	(82)
Infiltration cellular, lymphocyte	3 (4%)			1 (1%)
Inflammation, granulomatous				1 (1%)
Duct, cyst	1 (1%)	1 (1%)		
Ovary	(75)	(84)	(84)	(83)
Angiectasis		3 (4%)		
Cyst	9 (12%)	11 (13%)	7 (8%)	4 (5%)
Hemorrhage	1 (1%)	1 (1%)		
Infiltration cellular, histiocyte			1 (1%)	
Inflammation, granulomatous				1 (1%)
Mineral	1 (1%)		1 (1%)	
Follicle, cyst	9 (12%)	7 (8%)	6 (7%)	8 (10%)
Granulosa cell, hyperplasia				1 (1%)
Paraovarian tissue, cyst		2 (2%)		
Uterus	(89)	(89)	(88)	(90)
Adenomyosis			1 (1%)	
Angiectasis	1 (1%)	3 (3%)	3 (3%)	4 (4%)
Dilation	35 (39%)	21 (24%)	44 (50%)	37 (41%)
Hemorrhage	1 (1%)			
Infiltration cellular, lymphocyte				1 (1%)
Inflammation, chronic active			1 (1%)	
Mineral			1 (1%)	
Necrosis			1 (1%)	
Thrombus	1 (1%)		3 (3%)	1 (1%)

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Genital System (continued)				
Uterus (continued)	(89)	(89)	(88)	(90)
Arteriole, degeneration			1 (1%)	
Endometrium, cyst	3 (3%)			2 (2%)
Endometrium, hyperplasia, cystic	68 (76%)	75 (84%)	67 (76%)	68 (76%)
Endometrium, metaplasia, squamous	1 (1%)			
Hematopoietic System				
Bone marrow	(90)	(89)	(89)	(89)
Hypercellularity	7 (8%)	8 (9%)	3 (3%)	4 (4%)
Hypocellularity	1 (1%)		1 (1%)	
Myeloid cell, hypercellularity	1 (1%)	1 (1%)		
Lymph node	(18)	(21)	(18)	(18)
Hemorrhage			1 (6%)	
Hyperplasia, lymphoid	1 (6%)			
Infiltration cellular, mixed cell				1 (6%)
Axillary, infiltration cellular, mixed cell	1 (6%)			
Axillary, pigment	1 (6%)			
Bronchial, hemorrhage			1 (6%)	
Bronchial, hyperplasia, lymphoid	2 (11%)	2 (10%)		1 (6%)
Bronchial, infiltration cellular, histiocyte		1 (5%)		
Bronchial, infiltration cellular, mixed cell		1 (5%)		
Iliac, hemorrhage	1 (6%)		3 (17%)	1 (6%)
Iliac, hyperplasia, lymphoid	4 (22%)	4 (19%)	9 (50%)	4 (22%)
Iliac, infiltration cellular, histiocyte		1 (5%)	1 (6%)	
Iliac, infiltration cellular, mixed cell	1 (6%)	1 (5%)	1 (6%)	1 (6%)
Iliac, infiltration cellular, plasma cell			1 (6%)	
Iliac, pigment		1 (5%)	2 (11%)	
Lumbar, hemorrhage		1 (5%)		
Lumbar, hyperplasia, lymphoid		2 (10%)	1 (6%)	
Lumbar, infiltration cellular, mixed cell	1 (6%)			
Mediastinal, hyperplasia, lymphoid	1 (6%)	4 (19%)	1 (6%)	3 (17%)
Mediastinal, infiltration cellular, histiocyte				1 (6%)
Pancreatic, hyperplasia, lymphoid	1 (6%)			1 (6%)
Renal, ectasia		1 (5%)		
Renal, hemorrhage	1 (6%)			
Renal, hyperplasia, lymphoid	3 (17%)		2 (11%)	2 (11%)
Renal, infiltration cellular, mixed cell		1 (5%)		
Lymph node, mandibular	(76)	(79)	(76)	(73)
Hemorrhage	3 (4%)	1 (1%)	1 (1%)	2 (3%)
Hyperplasia, lymphoid	1 (1%)	1 (1%)	1 (1%)	2 (3%)
Infiltration cellular, histiocyte		1 (1%)	1 (1%)	
Infiltration cellular, mixed cell	1 (1%)			
Lymph node, mesenteric	(71)	(86)	(75)	(81)
Ectasia				1 (1%)
Erythrophagocytosis	1 (1%)			
Hemorrhage	1 (1%)	3 (3%)	6 (8%)	3 (4%)
Hyperplasia, lymphoid	1 (1%)	5 (6%)	4 (5%)	4 (5%)
Infiltration cellular, histiocyte	3 (4%)	4 (5%)	2 (3%)	4 (5%)
Infiltration cellular, plasma cell			1 (1%)	
Spleen	(86)	(87)	(86)	(88)
Atrophy	1 (1%)			
Extramedullary hematopoiesis	20 (23%)	18 (21%)	12 (14%)	16 (18%)
Hemorrhage		1 (1%)		
Hyperplasia, lymphoid	11 (13%)	10 (11%)	12 (14%)	14 (16%)
Capsule, fibrosis	1 (1%)		1 (1%)	

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Hematopoietic System (continued)				
Thymus	(85)	(83)	(82)	(82)
Atrophy	5 (6%)	3 (4%)	4 (5%)	2 (2%)
Cyst	2 (2%)	4 (5%)	4 (5%)	6 (7%)
Hemorrhage		2 (2%)	1 (1%)	2 (2%)
Hyperplasia, lymphoid	1 (1%)		2 (2%)	
Integumentary System				
Mammary gland	(85)	(87)	(90)	(88)
Hyperplasia, focal	1 (1%)		1 (1%)	
Hyperplasia, diffuse	1 (1%)	1 (1%)		
Infiltration cellular, lymphocyte		1 (1%)		
Duct, dilation	1 (1%)	2 (2%)	2 (2%)	2 (2%)
Skin	(90)	(89)	(90)	(90)
Ulcer	2 (2%)	3 (3%)	1 (1%)	1 (1%)
Epidermis, hyperplasia, focal		1 (1%)		1 (1%)
Hair follicle, atrophy	2 (2%)	2 (2%)		5 (6%)
Subcutaneous tissue, fibrosis				1 (1%)
Musculoskeletal System				
Bone	(90)	(89)	(90)	(89)
Fibro-osseous lesion	11 (12%)		1 (1%)	2 (2%)
Increased bone		1 (1%)	1 (1%)	
Skeletal muscle	(89)	(89)	(90)	(90)
Degeneration		1 (1%)		1 (1%)
Infiltration cellular, lymphocyte	16 (18%)	7 (8%)	11 (12%)	9 (10%)
Mineral	1 (1%)			
Nervous System				
Brain	(87)	(88)	(90)	(90)
Hemorrhage	2 (2%)			2 (2%)
Hydrocephalus	1 (1%)			
Inflammation, acute	1 (1%)			
Mineral	80 (92%)	81 (92%)	78 (87%)	74 (82%)
Necrosis	1 (1%)		1 (1%)	
Artery, meninges, inflammation, chronic active		3 (3%)	1 (1%)	1 (1%)
Brain trigeminal ganglion	(75)	(82)	(75)	(74)
Nerve trigeminal	(56)	(30)	(52)	(51)
Peripheral nerve, sciatic	(88)	(88)	(89)	(88)
Axon, degeneration	12 (14%)	4 (5%)	8 (9%)	11 (13%)
Spinal cord	(90)	(89)	(90)	(90)
Cyst, squamous, multiple			1 (1%)	1 (1%)
Degeneration		1 (1%)		
Demyelination		1 (1%)		
Metaplasia, osseous		1 (1%)		
Necrosis		2 (2%)		

TABLE D4
Summary of the Incidence of Nonneoplastic Lesions in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 2 Years

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
2-Year Study (continued)				
Respiratory System				
Lung	(90)	(89)	(90)	(90)
Congestion			2 (2%)	4 (4%)
Hemorrhage	4 (4%)	7 (8%)	4 (4%)	4 (4%)
Infiltration cellular, histiocyte	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Infiltration cellular, lymphocyte	3 (3%)	2 (2%)	4 (4%)	
Infiltration cellular, mixed cell		1 (1%)		
Infiltration cellular, mononuclear cell		1 (1%)		
Inflammation, granulomatous		1 (1%)		
Inflammation, acute	1 (1%)			
Inflammation, chronic	1 (1%)			
Inflammation, chronic active				1 (1%)
Alveolar epithelium, hyperplasia, focal	1 (1%)	3 (3%)	3 (3%)	3 (3%)
Mediastinum	(2)	(0)	(0)	(0)
Nose	(89)	(89)	(90)	(90)
Inflammation, acute	1 (1%)			
Respiratory epithelium, accumulation, hyaline droplet			1 (1%)	
Trachea	(90)	(87)	(89)	(88)
Special Senses System				
Eye	(89)	(89)	(90)	(89)
Phthisis bulbi				1 (1%)
Anterior chamber, inflammation, acute				1 (1%)
Harderian gland	(89)	(88)	(89)	(89)
Hyperplasia, focal		1 (1%)		
Infiltration cellular, lymphocyte	58 (65%)	66 (75%)	61 (69%)	64 (72%)
Urinary System				
Kidney	(89)	(89)	(88)	(87)
Cyst	1 (1%)			
Infarct	14 (16%)	26 (29%)	23 (26%)	17 (20%)
Metaplasia, osseous	2 (2%)	1 (1%)	2 (2%)	2 (2%)
Nephropathy, chronic progressive	8 (9%)	12 (13%)	7 (8%)	7 (8%)
Bilateral, infarct	1 (1%)			
Interstitium, infiltration cellular, lymphocyte	63 (71%)	65 (73%)	50 (57%)	50 (57%)
Medulla, mineral				1 (1%)
Papilla, mineral			2 (2%)	
Papilla, necrosis		1 (1%)		
Pelvis, dilation	1 (1%)			
Renal tubule, cyst				1 (1%)
Renal tubule, dilation	1 (1%)		1 (1%)	1 (1%)
Renal tubule, mineral	1 (1%)			
Renal tubule, vacuolization cytoplasmic			1 (1%)	
Urinary bladder	(86)	(86)	(83)	(85)
Angiectasis			6 (7%)	2 (2%)
Infiltration cellular, lymphocyte	62 (72%)	64 (74%)	70 (84%)	65 (76%)
Transitional epithelium, hyperplasia, diffuse			1 (1%)	

APPENDIX E
GENETIC TOXICOLOGY

GENETIC TOXICOLOGY

COLLECTION OF TISSUE SAMPLES FOR GENOTOXICITY TESTING

Exposures ceased at 7 a.m. on the day of necropsy at 14 weeks. Thirty-five male mice (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 2 to 4 hours after cessation of exposure and 35 female mice (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 5 to 7 hours after cessation of exposure. Animals were necropsied in the following order: one animal from each exposure group starting with the sham control group, moving through each of the exposed groups for each of the radio frequency modulations in turn, then rotating back to the sham control group; animals were necropsied in numerical order within each exposure group. Five different tissues (cerebrum, frontal cortex, hippocampus, liver, and blood leukocytes) were collected from each animal for the comet assay. Because blood was examined in both the micronucleus and the comet assays, a single tube of blood was collected per animal by retroorbital bleeding, and the sample was divided into two aliquots, one that was processed for the comet assay and the other for the micronucleus assay.

COMET ASSAY

For preparation of samples for the comet assay, a 50 µL sample of blood was transferred to a tube containing 1 mL of freshly prepared cold mincing buffer [Mg^{+2}, Ca^{+2}, and phenol free Hank's Balanced Salt Solution (Life Technologies, Carlsbad, CA) with 20 mM ethylenediaminetetraacetic acid (EDTA) pH 7.3 to 7.5 and 10% v/v fresh dimethyl sulfoxide (DMSO)]. The liver and the hippocampus, cerebellum, and frontal cortex sections of the brain were rinsed with cold mincing buffer to remove residual blood and held on ice briefly (≤5 minutes) until processed. Small portions (3 to 4 mm) of the left lobe of the liver and each brain section were placed in tubes containing cold mincing solution and rapidly minced until finely dispersed. All samples prepared for the comet assay were immediately flash frozen in liquid nitrogen (Recio *et al.*, 2010) and subsequently transferred to a –80° C freezer for storage until shipment by overnight courier on dry ice to the analytical laboratory. Upon receipt, all samples were immediately placed in a –80° C freezer for storage until further processing.

Blood and tissue samples were thawed on ice and maintained on ice during slide preparation. Just prior to use, each cell suspension was shaken gently to mix the cells and placed back on ice for 15 to 30 seconds to allow clumps to settle. A portion of the supernatant was empirically diluted with 0.5% low melting point agarose (Lonza, Walkersville, MD) dissolved in Dulbecco's phosphate buffer (Ca^{+2}, Mg^{+2}, and phenol free) at 37° C and layered onto each well of a 2-well CometSlide™ (Trevigen, Gaithersburg, MD). Slides were immersed in cold lysing solution [2.5 M NaCl, 100 mM Na_2EDTA, 10 mM tris(hydroxymethyl)aminomethane (Tris), pH 10, containing freshly added 10% DMSO (Fisher Scientific, Pittsburgh, PA) and 1% Triton X-100] overnight in a refrigerator, protected from light. The following day, the slides were rinsed in 0.4 M Trizma base (pH 7.5), randomly placed onto the platform of a horizontal electrophoresis unit and treated with cold alkali solution (300 mM NaOH, 1 mM Na_2EDTA, pH>13) for 20 minutes to allow DNA unwinding, then electrophoresed at 4º to 9º C for 20 minutes at 25 V (0.7 V/cm), with a current of approximately 300 mA. Following electrophoresis, slides were neutralized with 0.4 M Trizma base (pH 7.5) for 5 minutes and then dehydrated by immersion in absolute ethanol (Pharmco-AAPER, Shelbyville, KY) for at least 5 minutes and allowed to air dry. Slides were prepared in a laboratory with a relative humidity no more than 60% and stored at room temperature in a desiccator with a relative humidity of no more than 60% until stained and scored; stained slides were stored in a desiccator. NaCl, Na_2EDTA, Triton X-100, and Trizma base were purchased from Sigma-Aldrich (St. Louis, MO); NaOH was purchased from Fisher Scientific (Pittsburgh, PA).

After staining with SYBR® Gold (Molecular Probes, Life Technologies, Grand Island, NY), slides, independently coded to mask treatment, were scored using Comet Assay IV Imaging Software, Version 4.3.1 (Perceptive Instruments, Ltd., Suffolk, UK) validated for GLP Part 11 compliance. In the alkaline (pH>13) comet assay, when damaged nuclear DNA fragments, it undergoes unidirectional migration through the agarose gel within an electrical field, forming an image that resembles a comet, and the greater the amount of fragmentation, the greater the amount of DNA migration that will occur. The image analysis software partitions the intensity of the fluorescent signal of the DNA in the entire comet image into the percent that is attributable to the comet head and the percent attributable

to the tail. Manual adjustment of the automated detection of head and tail features is sometimes required. To evaluate DNA damage levels, the extent of DNA migration was characterized for 100 scorable comet figures per animal/tissue as percent tail DNA (intensity of all tail pixels divided by the total intensity of all pixels in the comet, expressed as a percentage).

Comet figures are classified during the scoring process as scorable (evaluated for percent tail DNA), non-scorable (due to inability to evaluate percent tail DNA, e.g. if comets overlapped), and "hedgehog." Hedgehogs either have no defined head, i.e., all DNA appears to be in the tail, or the head and tail appear to be separated. Hedgehogs may represent cells that have sustained high levels of DNA damage and are apoptotic, although certain data suggest they may represent cells with high levels of repairable DNA damage (Rundell *et al.*, 2003; Lorenzo *et al.*, 2013). The frequency of hedgehogs (%HH) was determined by tabulating the number observed in a separate group of 100 cells per animal/tissue.

In Technical Report 595 (NTP, 2018), in which the comet assay results in rats exposed to cell phone radio frequency radiation (RFR) are reported, it was noted that a marked interanimal variation in percent tail DNA and high %HH values were observed in some tissues, yet the range of percent tail DNA values appeared to be truncated at approximately 65%. To better understand these observations, rat slides were reanalyzed by scoring 150 cells/tissue per animal, as recommended by the OECD guideline (OECD, 2014). In this rescoring of the rat samples, all scorable cells were included in the sample of 150 analyzed cells, regardless of the apparent level of DNA damage estimated by the scorer prior to software analysis of the images; highly damaged cells that were unscorable using the software (true HH) were not included. For the 150-cell scoring method, the %HH was not independently determined due to limitations at the time in the comet assay software arising from the added number of cells scored. Therefore, %HH was estimated by dividing the number of comets having more than 90% tail DNA by 150.

Although far less interanimal variation was observed in mouse tissues compared to rat tissues, in an effort to maintain consistency in analyses across species, the mouse tissues that showed a clear response or a suggestion of a treatment-related effect were reevaluated using the same 150-cell approach that was used to reevaluate all of the rat tissues. These tissues included male mouse frontal cortex and female mouse liver and peripheral blood exposed to the CDMA and GSM modulations.

Although there was no concurrent positive control group in these cell phone RFR studies, slides were made with human TK6 cells treated with ethyl methanesulfonate (standard positive control compound for the comet assay) and were included in each electrophoresis run with each slide set as an internal technical positive control.

MICRONUCLEUS ASSAY

For the micronucleus assay, sampling schedules were as described for the comet assay. At 14 weeks, blood samples (approximately 200 µL) obtained by retroorbital bleeding (one sample per mouse) were placed into EDTA tubes and immediately refrigerated. The samples were sent on the day of collection to the analytical laboratory well insulated on cold packs via overnight delivery. Upon arrival, blood samples were diluted in anticoagulant (heparin) and fixed in ice cold methanol (Sigma-Aldrich; St. Louis, MO) according to instructions provided with the MicroFlow^{PLUS} Kit (Litron Laboratories, Rochester, NY). Fixed blood samples were stored in a –80° C freezer for at least 3 days prior to analysis by flow cytometry.

Flow cytometric analysis of red blood cell samples was performed using MicroFlow^{PLUS} Kit reagents and a FACSCalibur™ dual-laser bench top system (Becton Dickinson Biosciences, San Jose, CA) as described by Witt *et al.* (2008). Both mature [normochromatic erythrocytes (NCEs)] and immature [reticulocytes; polychromatic erythrocytes (PCEs)] erythrocytes were analyzed for the presence of micronuclei. Immature erythrocytes are distinguished by the presence of an active transferrin receptor (CD-71) on the cell surface. For each sample, 20,000 (± 2,000) immature CD71-positive erythrocytes were analyzed by flow cytometry to determine the frequency of micronucleated reticulocytes. Aggregates were excluded on the basis of forward and side scatter, platelets were excluded based on staining with an anti-CD61 antibody, and nucleated leukocytes were excluded on the basis of intense propidium iodide staining. Typically, more than one million NCEs (CD-71 negative) were enumerated concurrently during PCE analysis, allowing for calculation of the percentage of PCEs among total erythrocytes as a measure of bone marrow toxicity.

DATA ANALYSIS FOR THE COMET AND MICRONUCLEUS ASSAYS

Data from both the comet and the micronucleus assays were analyzed using the same statistical methods (Kissling *et al.*, 2007). Mean percent tail DNA was calculated for each cell type for each animal; likewise, mean micronucleated PCEs/1,000 PCEs and micronucleated NCEs/1,000 NCEs, as well as % PCEs, were calculated for each animal. These data are summarized in the tables as mean ± standard error of the mean. Levene's test was used to determine if variances among treatment groups were equal at P=0.05. When variances were equal, linear regression analysis was used to test for linear trend and Williams' test was used to evaluate pairwise differences of each exposed group with the sham control group. When variances were unequal, nonparametric methods were used to analyze the data; Jonckheere's test was used to evaluate linear trend and Dunn's test was used to assess the significance of pairwise differences of each exposed group with the sham control group. To maintain the overall significance level at 0.05, the trend as well as the pairwise differences from the sham control group were declared statistically significant if P<0.025. A result was considered positive if the trend test was significant and if at least one exposed group was significantly elevated over the sham control group, or if two or more exposed groups were significantly increased over the corresponding sham control group. A response was considered equivocal if only the trend test was significant or if only a single exposed group was significantly increased over the sham control.

RESULTS

Twenty sets of tissues obtained from animals at the 14-week interim evaluation in the 2-year study were evaluated for DNA damage using the comet assay (two sexes, five tissues, two cell phone RFR modulations). Results are reported based on the 100-cell scoring approach that was the standard method in use at the time of the study. Data for some tissues obtained using a second, 150-cell scoring approach recommended by a recently adopted international guideline for the *in vivo* comet assay, are noted for comparison. Significant increases in DNA damage were observed in cells of the frontal cortex of male mice exposed to both modulations, CDMA and GSM (Tables E1 and E2). Positive results were also obtained for male mouse frontal cortex (CDMA and GSM) (Table E3) using the 150-cell approach. Of note is the low percent tail DNA value in the frontal cortex of sham control mice. There is no appropriate historical control database to provide context for this response, but bonafide changes in DNA damage levels in a treatment group should remain constant relative to the control value. No technical aspects of the study that may have influenced this control value independently of the treated group values (e.g., % agarose gel, duration of electrophoresis, electromagnetic field strength, slide position in the electrophoresis tank) were identified. Technical factors that influence control levels have not been shown to alter sensitivity to detect effects in treated groups (Recio *et al.*, 2012). No other tissues showed evidence of a treatment-related effect in male mice. In female mice exposed to the CDMA modulation, significant increases in DNA damage were seen in blood leukocytes using both scoring approaches (Tables E4 and E6). In female mouse liver samples exposed to either modulation, the mean percent tail DNA was elevated above the sham control for all exposures when evaluated using either scoring approach. Results of the 100-cell scoring approach were judged to be negative (Tables E4 and E6); scoring 150 cells resulted in a negative call for GSM-exposed female mice (Table E5) but in CDMA-exposed female mouse liver, a significant increase (P=0.010) in percent tail DNA was seen in the 5 W/kg group, resulting in an equivocal call for this dataset (Table E6).

In the micronucleus assay for male mice exposed to CDMA (Table E7), although a significant trend was observed for micronucleated PCEs (P=0.013), the absolute increase was quite small and fell within the laboratory's historical control range. In addition, no corresponding increase in micronucleated NCEs was observed; the mature erythrocyte population ought to be in steady state equilibrium after continuous 14 weeks of exposure, such as occurred in this study. Thus, the overall result in the micronucleus assay for male mice exposed to CDMA was judged to be negative. No other significant effects on either micronucleus frequency or % PCEs were seen in male or female mice exposed to either modulation of cell phone RFR.

TABLE E1
DNA Damage in Male Mice Exposed to CDMA-Modulated Cell Phone RFR for 14 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	0.63 ± 0.08		0.40 ± 0.24
CDMA	2.5	3.46 ± 0.65	0.014	0.60 ± 0.40
	5	5.88 ± 1.06	0.001	0.60 ± 0.24
	10	8.85 ± 1.09	0.001	4.40 ± 1.69
		P=0.001[e]		
Hippocampus				
Sham Control	0	7.69 ± 2.00		1.20 ± 0.58
CDMA	2.5	9.59 ± 4.33	0.521	5.40 ± 2.11
	5	6.44 ± 1.21	0.606	2.80 ± 0.97
	10	6.38 ± 0.93	0.641	4.40 ± 2.27
		P=0.740		
Cerebellum				
Sham Control	0	5.48 ± 1.30		1.80 ± 0.80
CDMA	2.5	7.35 ± 2.47	0.339	4.40 ± 2.06
	5	7.87 ± 2.80	0.404	4.60 ± 2.34
	10	5.43 ± 2.43	0.431	1.60 ± 0.93
		P=0.554		
Liver				
Sham Control	0	16.30 ± 2.21		6.80 ± 2.82
CDMA	2.5	17.66 ± 1.89	1.000	21.60 ± 16.88
	5	16.15 ± 1.15	1.000	11.00 ± 3.77
	10	16.43 ± 0.83	1.000	7.20 ± 1.11
		P=0.368		
Peripheral Blood				
Sham Control	0	1.60 ± 0.68		0.40 ± 0.24
CDMA	2.5	2.10 ± 0.50	0.449	1.20 ± 0.58
	5	1.30 ± 0.28	0.527	0.40 ± 0.24
	10	2.86 ± 0.26	0.046	1.40 ± 0.87
		P=0.057		

[a] Study was performed at ILS, Inc. The detailed protocol (100 cell) is presented by Recio *et al.* (2010). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] No exposure to CDMA-modulated cell phone RFR
[e] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E2
DNA Damage in Male Mice Exposed to GSM-Modulated Cell Phone RFR for 14 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	0.63 ± 0.08		0.40 ± 0.24
GSM	2.5	1.71 ± 0.46	0.081	1.80 ± 0.97
	5	1.39 ± 0.15	0.081	1.60 ± 0.81
	10	3.73 ± 0.65	0.001	1.00 ± 0.45
		P=0.001[e]		
Hippocampus				
Sham Control	0	7.69 ± 2.00		1.20 ± 0.58
GSM	2.5	8.74 ± 1.93	0.514	5.40 ± 2.11
	5	7.17 ± 1.08	0.598	2.20 ± 0.97
	10	6.90 ± 1.19	0.633	5.40 ± 2.54
		P=0.720		
Cerebellum				
Sham Control	0	5.48 ± 1.30		1.80 ± 0.80
GSM	2.5	3.66 ± 0.30	0.831	3.00 ± 1.38
	5	3.90 ± 0.59	0.896	1.80 ± 0.92
	10	3.85 ± 1.08	0.919	3.40 ± 1.50
		P=0.838		
Liver				
Sham Control	0	16.30 ± 2.21		6.80 ± 2.82
GSM	2.5	17.66 ± 1.89	0.469	8.20 ± 3.84
	5	15.40 ± 1.20	0.549	6.60 ± 1.96
	10	18.94 ± 2.00	0.213	12.80 ± 4.40
		P=0.198		
Peripheral Blood				
Sham Control	0	1.60 ± 0.68		0.40 ± 0.24
GSM	2.5	1.85 ± 0.96	0.416	1.20 ± 1.20
	5	1.75 ± 0.37	0.491	1.00 ± 0.55
	10	1.85 ± 0.24	0.494	0.80 ± 0.58
		P=0.408		

[a] Study was performed at ILS, Inc. The detailed protocol (100 cell) is presented by Recio *et al.* (2010). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] No exposure to GSM-modulated cell phone RFR
[e] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E3
DNA Damage in the Frontal Cortex of Male Mice Exposed to CDMA- or GSM-Modulated Cell Phone RFR for 14 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Sham Control[e]	0	1.32 ± 0.21		0
CDMA	2.5	4.52 ± 0.57	0.131	0
	5	6.06 ± 0.96	0.018	0
	10	10.04 ± 2.08	0.001	0.53 ± 0.39
		P=0.001[f]		
GSM	2.5	4.25 ± 1.20	0.063	0.13 ± 0.13
	5	3.69 ± 0.53	0.063	0
	10	5.60 ± 1.28	0.006	0.13 ± 0.13
		P=0.004		

[a] Study was performed at ILS, Inc. The detailed protocol (150 cell) is presented by OECD (2014). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] Percent hedgehogs=estimated as the number of comets with >90% tail DNA/150
[e] No exposure to CDMA- or GSM-modulated cell phone RFR
[f] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E4
DNA Damage in Female Mice Exposed to CDMA-Modulated Cell Phone RFR for 14 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	8.11 ± 2.13		3.40 ± 1.47
CDMA	2.5	4.88 ± 0.55	0.911	0.80 ± 0.49
	5	4.89 ± 0.57	0.955	1.20 ± 0.49
	10	4.80 ± 0.90	0.968	0.80 ± 0.58
		P=0.935[e]		
Hippocampus				
Sham Control	0	8.15 ± 1.65		2.60 ± 1.69
CDMA	2.5	5.76 ± 1.00	0.839	1.80 ± 0.80
	5	5.22 ± 1.02	0.903	1.20 ± 0.58
	10	5.34 ± 1.82	0.925	2.20 ± 0.97
		P=0.892		
Cerebellum				
Sham Control	0	5.88 ± 0.85		0.20 ± 0.20
CDMA	2.5	6.56 ± 1.22	0.296	1.75 ± 1.03
	5	8.39 ± 1.13	0.194	0.20 ± 0.20
	10	6.73 ± 0.77	0.207	0.40 ± 0.40
		P=0.298		
Liver				
Sham Control	0	5.48 ± 0.60		0.60 ± 0.40
CDMA	2.5	7.54 ± 0.90	0.034	1.00 ± 0.45
	5	7.36 ± 0.72	0.041	4.40 ± 2.11
	10	7.63 ± 0.59	0.030	2.00 ± 0.77
		P=0.050		
Peripheral Blood				
Sham Control	0	1.03 ± 0.13		0.20 ± 0.20
CDMA	2.5	2.52 ± 0.54	0.020	2.00 ± 1.14
	5	1.71 ± 0.37	0.024	0
	10	2.20 ± 0.19	0.018	0.20 ± 0.20
		P=0.085		

[a] Study was performed at ILS, Inc. The detailed protocol (100 cell) is presented by Recio *et al.* (2010). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] No exposure to CDMA-modulated cell phone RFR
[e] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E5
DNA Damage in Female Mice Exposed to GSM-Modulated Cell Phone RFR for 14 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	8.11 ± 2.13		3.40 ± 1.47
GSM	2.5	7.33 ± 0.90	0.657	1.00 ± 0.45
	5	7.69 ± 1.98	0.744	2.00 ± 0.84
	10	5.74 ± 0.62	0.779	1.00 ± 0.32
		P=0.861[e]		
Hippocampus				
Sham Control	0	8.15 ± 1.65		2.60 ± 1.69
GSM	2.5	6.23 ± 1.00	0.866	0.80 ± 0.58
	5	4.54 ± 1.29	0.923	1.20 ± 0.58
	10	5.22 ± 1.23	0.942	1.60 ± 1.36
		P=0.933		
Cerebellum				
Sham Control	0	5.88 ± 0.85		0.20 ± 0.20
GSM	2.5	6.56 ± 1.22	1.000	1.20 ± 0.73
	5	5.26 ± 0.59	1.000	0.60 ± 0.40
	10	6.54 ± 1.71	1.000	1.80 ± 0.73
		P=0.606		
Liver				
Sham Control	0	5.48 ± 0.60		0.60 ± 0.40
GSM	2.5	7.06 ± 0.61	0.096	3.40 ± 1.17
	5	6.36 ± 0.25	0.117	1.20 ± 0.37
	10	6.47 ± 0.79	0.124	2.60 ± 1.33
		P=0.249		
Peripheral Blood				
Sham Control	0	1.03 ± 0.13		0.20 ± 0.20
GSM	2.5	1.25 ± 0.44	0.335	0.20 ± 0.20
	5	1.17 ± 0.08	0.400	0
	10	1.32 ± 0.34	0.316	0
		P=0.266		

[a] Study was performed at ILS, Inc. The detailed protocol (100 cell) is presented by Recio *et al.* (2010). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] No exposure to GSM-modulated cell phone RFR
[e] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E6
DNA Damage in Female Mice Exposed to CDMA- or GSM-Modulated Cell Phone RFR for 14 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Liver				
Sham Control[e]	0	4.34 ± 0.60		0
CDMA	2.5	6.80 ± 1.17	0.146	0
	5	7.70 ± 0.95	0.010	0
	10	6.14 ± 0.26	0.131	0
		P=0.030[f]		
GSM	2.5	7.44 ± 0.48	0.027	0
	5	5.45 ± 0.96	0.032	0
	10	6.52 ± 0.75	0.030	0
		P=0.133		
Peripheral Blood				
Sham Control	0	2.15 ± 0.08		0
CDMA	2.5	3.62 ± 0.66	0.011	0
	5	3.39 ± 0.45	0.015	0.13 ± 0.13
	10	2.45 ± 0.24	0.428	0
		P=0.173		
GSM	2.5	2.58 ± 0.35	0.504	0
	5	2.23 ± 0.19	1.000	0
	10	2.28 ± 0.51	1.000	0
		P=0.657		

[a] Study was performed at ILS, Inc. The detailed protocol (150 cell) is presented by OECD (2014). Groups of five mice per exposure group were 5 to 6 weeks old when exposure began.
[b] Mean ± standard error
[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.
[d] Percent hedgehogs=estimated as the number of comets with >90% tail DNA/150
[e] No exposure to CDMA- or GSM-modulated cell phone RFR
[f] Dose-related trend; significant at P≤0.025 by linear regression.

TABLE E7
Frequency of Micronuclei in Peripheral Blood Erythrocytes of Mice Following Exposure to CDMA- or GSM-Modulated Cell Phone RFR for 14 Weeks[a]

	Dose (W/kg)	Number of Mice with Erythrocytes Scored	Micronucleated PCEs/ 1,000 PCEs[b]	P Value[c]	Micronucleated NCEs/ 1,000 NCEs[b]	P Value[c]	PCEs[b] (%)	P Value[c]
Male								
Sham Control[d]	0	5	2.55 ± 0.11		1.50 ± 0.04		1.45 ± 0.05	
CDMA	2.5	5	2.44 ± 0.13	0.611	1.45 ± 0.03	0.748	1.47 ± 0.04	0.765
	5	5	2.77 ± 0.13	0.168	1.46 ± 0.04	0.827	1.50 ± 0.04	0.736
	10	5	2.93 ± 0.18	0.044	1.49 ± 0.02	0.736	1.47 ± 0.04	0.778
			P=0.013[e]		P=0.497		P=0.803	
GSM	2.5	5	2.84 ± 0.14	0.384	1.49 ± 0.04	0.695	1.41 ± 0.04	0.667
	5	5	2.47 ± 0.19	0.455	1.45 ± 0.02	0.781	1.40 ± 0.04	0.787
	10	5	2.53 ± 0.13	0.484	1.50 ± 0.02	0.675	1.47 ± 0.08	0.830
			P=0.733		P=0.561		P=0.803	
Female								
Sham Control	0	5	2.72 ± 0.27		1.18 ± 0.02		1.33 ± 0.11	
CDMA	2.5	5	2.16 ± 0.15	0.846	1.06 ± 0.04	0.956	1.33 ± 0.13	1.000
	5	5	2.32 ± 0.22	0.908	1.09 ± 0.03	0.982	1.45 ± 0.11	0.930
	10	5	2.48 ± 0.20	0.883	1.14 ± 0.02	0.929	1.28 ± 0.09	0.935
			P=0.629		P=0.585		P=0.843	
GSM	2.5	5	2.50 ± 0.40	0.774	1.14 ± 0.05	0.827	1.19 ± 0.08	0.671
	5	5	2.35 ± 0.15	0.850	1.09 ± 0.02	0.893	1.17 ± 0.06	0.791
	10	5	2.16 ± 0.15	0.878	1.12 ± 0.04	0.916	1.45 ± 0.09	0.438
			P=0.937		P=0.891		P=0.245	

[a] Study was performed at ILS, Inc. The detailed protocol is presented by Witt *et al*. (2008). Mice were 5 to 6 weeks old when exposure began. NCE=normochromatic erythrocyte; PCE=polychromatic erythrocyte

[b] Mean ± standard error

[c] Pairwise comparison with the sham control group; exposed group values are significant at P≤0.025 by Williams' test.

[d] No exposure to CDMA- or GSM-modulated cell phone RFR

[e] Dose-related trend significant at P≤0.025 by linear regression

APPENDIX F
HEMATOLOGY RESULTS

TABLE F1
Hematology Data for Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Male				
Hematocrit (%)	54.8 ± 0.5	54.1 ± 0.9	54.2 ± 0.3	53.5 ± 0.5
Manual hematocrit (%)	50 ± 0	49 ± 1[b]	49 ± 0	49 ± 0
Hemoglobin (g/dL)	16.1 ± 0.1	16.0 ± 0.2	16.0 ± 0.1	15.9 ± 0.2
Erythrocytes (10^6/µL)	10.87 ± 0.09	10.66 ± 0.15	10.76 ± 0.06	10.61 ± 0.10
Reticulocytes (10^3/µL)	386.3 ± 8.2	363.6 ± 9.3	358.5 ± 7.5	357.9 ± 7.9
Nucleated erythrocytes (/100 leukocytes)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	50.4 ± 0.1	50.8 ± 0.2	50.4 ± 0.2	50.4 ± 0.2
Mean cell hemoglobin (pg)	14.8 ± 0.0	15.0 ± 0.1	14.9 ± 0.1	15.0 ± 0.1
Mean cell hemoglobin concentration (g/dL)	29.4 ± 0.1	29.5 ± 0.1	29.5 ± 0.1	29.7 ± 0.1
Platelets (10^3/µL)	1,115 ± 31	1,065 ± 30	1,111 ± 35	1,116 ± 32
Leukocytes (10^3/µL)	5.80 ± 0.50	5.11 ± 0.53	5.52 ± 0.43	6.30 ± 0.47
Segmented neutrophils (10^3/µL)	0.68 ± 0.06	0.58 ± 0.07	0.62 ± 0.04	0.67 ± 0.05
Lymphocytes (10^3/µL)	4.82 ± 0.41	4.28 ± 0.44	4.63 ± 0.37	5.29 ± 0.40
Monocytes (10^3/µL)	0.09 ± 0.01	0.09 ± 0.01	0.09 ± 0.01	0.11 ± 0.01
Basophils (10^3/µL)	0.04 ± 0.01	0.04 ± 0.01	0.04 ± 0.01	0.05 ± 0.01
Eosinophils (10^3/µL)	0.09 ± 0.01	0.07 ± 0.01	0.08 ± 0.01	0.10 ± 0.01
Large unstained cells (10^3/µL)	0.07 ± 0.01	0.06 ± 0.01	0.07 ± 0.01	0.07 ± 0.01
Female				
Hematocrit (%)	54.9 ± 2.0	55.7 ± 0.9	55.6 ± 0.5	55.2 ± 0.4
Manual hematocrit (%)	50 ± 2	52 ± 1	52 ± 1	51 ± 0
Hemoglobin (g/dL)	16.4 ± 0.5	16.8 ± 0.3	16.8 ± 0.2	16.5 ± 0.1
Erythrocytes (10^6/µL)	10.77 ± 0.34	10.88 ± 0.16	10.90 ± 0.11	10.75 ± 0.07
Reticulocytes (10^3/µL)	346.8 ± 17.4	365.5 ± 20.0	328.6 ± 13.5	378.6 ± 15.5
Nucleated erythrocytes (/100 leukocytes)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	50.9 ± 0.3	51.2 ± 0.2	51.1 ± 0.2	51.4 ± 0.2
Mean cell hemoglobin (pg)	15.2 ± 0.1	15.4 ± 0.1	15.4 ± 0.1	15.3 ± 0.1
Mean cell hemoglobin concentration (g/dL)	29.9 ± 0.3	30.1 ± 0.1	30.2 ± 0.1	29.8 ± 0.1
Platelets (10^3/µL)	758 ± 65	714 ± 37	717 ± 52	782 ± 29
Leukocytes (10^3/µL)	5.15 ± 0.57	5.06 ± 0.60	5.07 ± 0.57	4.88 ± 0.58
Segmented neutrophils (10^3/µL)	0.60 ± 0.08	0.53 ± 0.07	0.44 ± 0.07	0.58 ± 0.06
Lymphocytes (10^3/µL)	4.35 ± 0.49	4.30 ± 0.51	4.41 ± 0.49	4.09 ± 0.52
Monocytes (10^3/µL)	0.07 ± 0.01	0.08 ± 0.01	0.08 ± 0.01	0.08 ± 0.01
Basophils (10^3/µL)	0.03 ± 0.01	0.04 ± 0.00	0.04 ± 0.01	0.03 ± 0.00
Eosinophils (10^3/µL)	0.06 ± 0.01	0.07 ± 0.01	0.06 ± 0.01	0.06 ± 0.01
Large unstained cells (10^3/µL)	0.04 ± 0.01	0.04 ± 0.01	0.04 ± 0.01	0.04 ± 0.00

[a] Data are presented as mean ± standard error. Jonckheere's test for trend and Shirley's and Dunn's tests were performed on unrounded data.
[b] n=9

TABLE F2
Hematology Data for Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Male				
Hematocrit (%)	54.8 ± 0.5	54.6 ± 0.6	54.0 ± 0.6	54.5 ± 0.6
Manual hematocrit (%)	50 ± 0	50 ± 1	49 ± 1	50 ± 1
Hemoglobin (g/dL)	16.1 ± 0.1	16.0 ± 0.2	16.0 ± 0.2	16.1 ± 0.2
Erythrocytes (10^6/µL)	10.87 ± 0.09	10.77 ± 0.09	10.68 ± 0.12	10.76 ± 0.11
Reticulocytes (10^3/µL)	386.3 ± 8.2	367.1 ± 9.0	360.3 ± 8.8	374.6 ± 6.3
Nucleated erythrocytes (/100 leukocytes)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	50.4 ± 0.1	50.7 ± 0.2	50.6 ± 0.1	50.7 ± 0.2
Mean cell hemoglobin (pg)	14.8 ± 0.0	14.9 ± 0.1	14.9 ± 0.1	15.0 ± 0.1
Mean cell hemoglobin concentration (g/dL)	29.4 ± 0.1	29.3 ± 0.1	29.5 ± 0.1	29.6 ± 0.1
Platelets (10^3/µL)	1,115 ± 31	1,087 ± 36	1,128 ± 30	1,104 ± 40
Leukocytes (10^3/µL)	5.80 ± 0.50	5.41 ± 0.35	5.57 ± 0.43	5.45 ± 0.44
Segmented neutrophils (10^3/µL)	0.68 ± 0.06	0.59 ± 0.04	0.62 ± 0.05	0.58 ± 0.05
Lymphocytes (10^3/µL)	4.82 ± 0.41	4.57 ± 0.31	4.67 ± 0.38	4.57 ± 0.36
Monocytes (10^3/µL)	0.09 ± 0.01	0.09 ± 0.01	0.10 ± 0.01	0.09 ± 0.01
Basophils (10^3/µL)	0.04 ± 0.01	0.03 ± 0.01	0.04 ± 0.01	0.05 ± 0.01
Eosinophils (10^3/µL)	0.09 ± 0.01	0.07 ± 0.01	0.09 ± 0.01	0.09 ± 0.02
Large unstained cells (10^3/µL)	0.07 ± 0.01	0.06 ± 0.01	0.07 ± 0.01	0.07 ± 0.01
Female				
Hematocrit (%)	54.9 ± 2.0	55.2 ± 0.8	56.4 ± 0.6	56.1 ± 0.4
Manual hematocrit (%)	50 ± 2	52 ± 1	52 ± 1	52 ± 0
Hemoglobin (g/dL)	16.4 ± 0.5	16.6 ± 0.2	17.0 ± 0.2	16.8 ± 0.2
Erythrocytes (10^6/µL)	10.77 ± 0.34	10.78 ± 0.14	11.10 ± 0.11	10.96 ± 0.06
Reticulocytes (10^3/µL)	346.8 ± 17.4	371.2 ± 14.4	366.7 ± 20.6	374.7 ± 13.8
Nucleated erythrocytes (/100 leukocytes)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	50.9 ± 0.3	51.2 ± 0.2	50.8 ± 0.2	51.2 ± 0.1
Mean cell hemoglobin (pg)	15.2 ± 0.1	15.4 ± 0.1	15.3 ± 0.1	15.3 ± 0.1
Mean cell hemoglobin concentration (g/dL)	29.9 ± 0.3	30.1 ± 0.1	30.1 ± 0.1	29.9 ± 0.2
Platelets (10^3/µL)	758 ± 65	736 ± 53	668 ± 38	685 ± 41
Leukocytes (10^3/µL)	5.15 ± 0.57	5.24 ± 0.45	4.66 ± 0.55	4.53 ± 0.34
Segmented neutrophils (10^3/µL)	0.60 ± 0.08	0.52 ± 0.04	0.51 ± 0.08	0.42 ± 0.05
Lymphocytes (10^3/µL)	4.35 ± 0.49	4.52 ± 0.40	3.95 ± 0.46	3.92 ± 0.31
Monocytes (10^3/µL)	0.07 ± 0.01	0.07 ± 0.01	0.07 ± 0.01	0.07 ± 0.01
Basophils (10^3/µL)	0.03 ± 0.01	0.04 ± 0.00	0.03 ± 0.01	0.03 ± 0.01
Eosinophils (10^3/µL)	0.06 ± 0.01	0.06 ± 0.01	0.06 ± 0.01	0.06 ± 0.01
Large unstained cells (10^3/µL)	0.04 ± 0.01	0.04 ± 0.00	0.04 ± 0.01	0.04 ± 0.00

[a] Data are presented as mean ± standard error. Jonckheere's test for trend and Shirley's and Dunn's tests were performed on unrounded data.

APPENDIX G
ORGAN WEIGHTS
AND ORGAN-WEIGHT-TO-BODY-WEIGHT RATIOS

TABLE G1
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice in the 28-Day GSM-Modulated Cell Phone RFR Study[a]

	Sham Control	5 W/kg	10 W/kg	15 W/kg
n	10	10	10	10
Male				
Necropsy body wt.	24.9 ± 0.4	25.2 ± 0.4	24.7 ± 0.3	25.0 ± 0.4
R. Adrenal gland				
Absolute	0.0032 ± 0.0006	0.0025 ± 0.0003	0.0031 ± 0.0006[b]	0.0030 ± 0.0003
Relative	0.13 ± 0.02	0.10 ± 0.01	0.12 ± 0.02[b]	0.12 ± 0.01
Brain				
Absolute	0.47 ± 0.00	0.47 ± 0.00	0.47 ± 0.00	0.47 ± 0.00
Relative	19.06 ± 0.28	18.63 ± 0.24	18.86 ± 0.25	18.78 ± 0.33
Heart				
Absolute	0.14 ± 0.00	0.14 ± 0.00	0.14 ± 0.00	0.14 ± 0.00
Relative	5.59 ± 0.14	5.41 ± 0.14	5.50 ± 0.10	5.57 ± 0.15
R. Kidney				
Absolute	0.22 ± 0.01	0.23 ± 0.00	0.22 ± 0.00	0.22 ± 0.01
Relative	8.80 ± 0.14	9.07 ± 0.14	8.90 ± 0.12	8.64 ± 0.15
Liver				
Absolute	1.29 ± 0.03	1.29 ± 0.03	1.25 ± 0.03	1.23 ± 0.03
Relative	51.86 ± 0.73	51.24 ± 0.80	50.63 ± 0.94	49.16 ± 0.95
Lung				
Absolute	0.20 ± 0.01	0.19 ± 0.01[b]	0.20 ± 0.01	0.19 ± 0.01
Relative	7.87 ± 0.35	7.44 ± 0.41[b]	8.14 ± 0.45	7.45 ± 0.54
R. Testis				
Absolute	0.094 ± 0.005	0.097 ± 0.002	0.093 ± 0.005	0.097 ± 0.002
Relative	3.79 ± 0.21	3.88 ± 0.09	3.75 ± 0.21	3.87 ± 0.06
Thymus				
Absolute	0.045 ± 0.002	0.046 ± 0.001	0.046 ± 0.001	0.047 ± 0.002
Relative	1.81 ± 0.06	1.84 ± 0.04	1.84 ± 0.05	1.89 ± 0.11

TABLE G1
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice in the 28-Day GSM-Modulated Cell Phone RFR Study

	Sham Control	5 W/kg	10 W/kg	15 W/kg
n	10	10	10	10
Female				
Necropsy body wt.	21.7 ± 0.3	21.9 ± 0.2	21.2 ± 0.2	21.0 ± 0.2*
R. Adrenal gland				
Absolute	0.0037 ± 0.0006	0.0031 ± 0.0006	0.0037 ± 0.0005	0.0036 ± 0.0003
Relative	0.17 ± 0.03	0.14 ± 0.03	0.18 ± 0.02	0.17 ± 0.01
Brain				
Absolute	0.48 ± 0.00	0.49 ± 0.00	0.47 ± 0.00	0.47 ± 0.00
Relative	22.24 ± 0.32	22.15 ± 0.18	22.21 ± 0.24	22.56 ± 0.27
Heart				
Absolute	0.13 ± 0.00	0.13 ± 0.00	0.12 ± 0.00	0.12 ± 0.00*
Relative	6.00 ± 0.19	5.85 ± 0.11	5.77 ± 0.12	5.62 ± 0.17
R. Kidney				
Absolute	0.17 ± 0.00	0.16 ± 0.00	0.15 ± 0.00	0.16 ± 0.00
Relative	7.65 ± 0.18	7.44 ± 0.13	7.27 ± 0.18	7.39 ± 0.11
Liver				
Absolute	1.14 ± 0.03	1.18 ± 0.02	1.10 ± 0.02	1.07 ± 0.03
Relative	52.61 ± 0.66	53.72 ± 0.85	51.77 ± 0.92	50.72 ± 0.95
Lung				
Absolute	0.18 ± 0.01	0.19 ± 0.01	0.17 ± 0.01	0.17 ± 0.00
Relative	8.48 ± 0.45	8.45 ± 0.34	8.18 ± 0.25	8.06 ± 0.19
Thymus				
Absolute	0.056 ± 0.001	0.057 ± 0.001	0.054 ± 0.001	0.055 ± 0.002
Relative	2.57 ± 0.07	2.59 ± 0.04	2.53 ± 0.07	2.62 ± 0.11

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test

[a] Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error).

[b] n=9

TABLE G2
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Male				
Necropsy body wt.	34.8 ± 0.8	33.3 ± 0.8	34.3 ± 0.6	33.0 ± 0.5
Brain				
Absolute	0.48 ± 0.01	0.48 ± 0.00	0.47 ± 0.00	0.47 ± 0.01
Relative	13.74 ± 0.43	14.44 ± 0.39	13.73 ± 0.28	14.18 ± 0.22
R. Epididymis				
Absolute	0.0504 ± 0.0024	0.0466 ± 0.0023	0.0471 ± 0.0029	0.0489 ± 0.0016
Relative	1.45 ± 0.06	1.40 ± 0.05	1.37 ± 0.07	1.49 ± 0.05
L. Epididymis				
Absolute	0.0478 ± 0.0019	0.0499 ± 0.0026	0.0500 ± 0.0026	0.0468 ± 0.0023
Relative	1.38 ± 0.06	1.50 ± 0.07	1.46 ± 0.07	1.43 ± 0.09
Heart				
Absolute	0.16 ± 0.00[b]	0.17 ± 0.01	0.16 ± 0.00[b]	0.16 ± 0.01
Relative	4.52 ± 0.09[b]	5.06 ± 0.18	4.75 ± 0.13[b]	4.87 ± 0.21
R. Kidney				
Absolute	0.27 ± 0.01	0.26 ± 0.01	0.25 ± 0.00*	0.25 ± 0.01**
Relative	7.80 ± 0.17	7.89 ± 0.18	7.44 ± 0.14	7.63 ± 0.19
L. Kidney				
Absolute	0.26 ± 0.01	0.25 ± 0.01	0.25 ± 0.01	0.23 ± 0.00**
Relative	7.54 ± 0.15	7.65 ± 0.15	7.20 ± 0.16	7.08 ± 0.16
Liver				
Absolute	1.54 ± 0.05	1.44 ± 0.05	1.38 ± 0.03*[b]	1.39 ± 0.03*[b]
Relative	44.27 ± 0.73	43.18 ± 0.85	40.73 ± 0.82*[b]	42.31 ± 1.01[b]
Lung				
Absolute	0.28 ± 0.02	0.29 ± 0.02	0.24 ± 0.01	0.27 ± 0.02
Relative	7.84 ± 0.58	8.62 ± 0.58	6.98 ± 0.34	8.24 ± 0.68
R. Testis				
Absolute	0.110 ± 0.002	0.111 ± 0.004	0.102 ± 0.008	0.109 ± 0.002
Relative	3.16 ± 0.10	3.34 ± 0.11	2.98 ± 0.22	3.31 ± 0.09
L. Testis				
Absolute	0.104 ± 0.002	0.107 ± 0.002	0.101 ± 0.007	0.105 ± 0.002
Relative	3.01 ± 0.12	3.22 ± 0.09	2.96 ± 0.21	3.20 ± 0.09
Thymus				
Absolute	0.035 ± 0.002	0.037 ± 0.002	0.037 ± 0.002	0.036 ± 0.002
Relative	1.02 ± 0.07	1.10 ± 0.04	1.07 ± 0.05	1.10 ± 0.04

TABLE G2
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Female				
Necropsy body wt.	24.4 ± 0.4	24.9 ± 0.5	25.0 ± 0.5	26.2 ± 0.7*
Brain				
Absolute	0.49 ± 0.00	0.49 ± 0.00	0.49 ± 0.00	0.49 ± 0.01
Relative	20.21 ± 0.32	19.85 ± 0.37	19.78 ± 0.36	18.86 ± 0.40*
Heart				
Absolute	0.15 ± 0.01	0.16 ± 0.01	0.16 ± 0.01	0.16 ± 0.01
Relative	6.24 ± 0.25	6.34 ± 0.25	6.31 ± 0.22	6.11 ± 0.28
R. Kidney				
Absolute	0.18 ± 0.00	0.18 ± 0.00	0.17 ± 0.00	0.17 ± 0.01
Relative	7.26 ± 0.13	7.08 ± 0.18	6.92 ± 0.23	6.65 ± 0.17*
L. Kidney				
Absolute	0.16 ± 0.00	0.16 ± 0.00	0.15 ± 0.00	0.16 ± 0.01
Relative	6.52 ± 0.16	6.35 ± 0.08	6.19 ± 0.15	6.20 ± 0.16
Liver				
Absolute	1.21 ± 0.03	1.24 ± 0.03	1.22 ± 0.02	1.28 ± 0.05
Relative	49.60 ± 0.66	49.76 ± 0.59	48.96 ± 0.75	48.94 ± 1.28
Lung				
Absolute	0.31 ± 0.02	0.34 ± 0.02	0.32 ± 0.01	0.31 ± 0.01
Relative	12.59 ± 0.60	13.45 ± 0.73	12.75 ± 0.39	11.75 ± 0.60
R. Ovary				
Absolute	0.0077 ± 0.0004	0.0072 ± 0.0006	0.0067 ± 0.0009	0.0070 ± 0.0006
Relative	0.32 ± 0.02	0.29 ± 0.02	0.27 ± 0.04	0.27 ± 0.02
L. Ovary				
Absolute	0.0069 ± 0.0009	0.0058 ± 0.0006	0.0053 ± 0.0008	0.0064 ± 0.0003
Relative	0.28 ± 0.04	0.23 ± 0.02	0.21 ± 0.03	0.25 ± 0.01
Thymus				
Absolute	0.041 ± 0.003	0.044 ± 0.001	0.043 ± 0.002	0.049 ± 0.002*
Relative	1.66 ± 0.10	1.75 ± 0.04	1.73 ± 0.04	1.86 ± 0.06

* Significantly different (P≤0.05) from the sham control group by Williams' or Dunnett's test
** P≤0.01
[a] Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error).
[b] n=9

TABLE G3
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice in the 28-Day CDMA-Modulated Cell Phone RFR Study[a]

	Sham Control	5 W/kg	10 W/kg	15 W/kg
n	10	10	10	10
Male				
Necropsy body wt.	24.9 ± 0.4	24.3 ± 0.3	25.2 ± 0.4	25.1 ± 0.3
R. Adrenal gland				
Absolute	0.0032 ± 0.0006	0.0025 ± 0.0002	0.0026 ± 0.0006	0.0030 ± 0.0006[b]
Relative	0.13 ± 0.02	0.10 ± 0.01	0.10 ± 0.02	0.12 ± 0.02[b]
Brain				
Absolute	0.47 ± 0.00	0.46 ± 0.00	0.48 ± 0.00	0.47 ± 0.00
Relative	19.06 ± 0.28	19.14 ± 0.13	19.11 ± 0.25	18.52 ± 0.24
Heart				
Absolute	0.14 ± 0.00	0.13 ± 0.00	0.14 ± 0.00	0.14 ± 0.00
Relative	5.59 ± 0.14	5.52 ± 0.12	5.40 ± 0.12	5.58 ± 0.14
R. Kidney				
Absolute	0.22 ± 0.01	0.21 ± 0.01	0.22 ± 0.01	0.22 ± 0.01
Relative	8.80 ± 0.14	8.77 ± 0.16	8.86 ± 0.20	8.67 ± 0.20
Liver				
Absolute	1.29 ± 0.03	1.24 ± 0.02	1.29 ± 0.02	1.25 ± 0.02
Relative	51.86 ± 0.73	51.26 ± 0.73	51.20 ± 0.82	49.85 ± 0.77
Lung				
Absolute	0.20 ± 0.01	0.18 ± 0.00	0.19 ± 0.01	0.18 ± 0.00
Relative	7.87 ± 0.35	7.55 ± 0.16	7.45 ± 0.24	7.13 ± 0.16
R. Testis				
Absolute	0.094 ± 0.005	0.094 ± 0.003	0.099 ± 0.001	0.096 ± 0.003
Relative	3.79 ± 0.21	3.88 ± 0.13	3.94 ± 0.09	3.81 ± 0.11
Thymus				
Absolute	0.045 ± 0.002	0.045 ± 0.001	0.046 ± 0.002	0.043 ± 0.001
Relative	1.81 ± 0.06	1.86 ± 0.04	1.85 ± 0.10	1.71 ± 0.04

TABLE G3
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice in the 28-Day CDMA-Modulated Cell Phone RFR Study

	Sham Control	5 W/kg	10 W/kg	15 W/kg
n	10	10	10	10
Female				
Necropsy body wt.	21.7 ± 0.3	21.7 ± 0.3	21.6 ± 0.3	21.2 ± 0.3
R. Adrenal gland				
Absolute	0.0037 ± 0.0006	0.0044 ± 0.0005	0.0037 ± 0.0005	0.0037 ± 0.0005
Relative	0.17 ± 0.03	0.20 ± 0.02	0.17 ± 0.03	0.17 ± 0.02
Brain				
Absolute	0.48 ± 0.00	0.48 ± 0.00	0.48 ± 0.00	0.48 ± 0.00
Relative	22.24 ± 0.32	22.26 ± 0.37	22.29 ± 0.29	22.48 ± 0.29
Heart				
Absolute	0.13 ± 0.00	0.12 ± 0.00	0.13 ± 0.00	0.12 ± 0.00
Relative	6.00 ± 0.19	5.73 ± 0.17	5.89 ± 0.16	5.81 ± 0.13
R. Kidney				
Absolute	0.17 ± 0.00	0.16 ± 0.00	0.16 ± 0.00	0.15 ± 0.00*
Relative	7.65 ± 0.18	7.21 ± 0.14	7.39 ± 0.23	7.24 ± 0.13
Liver				
Absolute	1.14 ± 0.03	1.14 ± 0.02	1.13 ± 0.02	1.09 ± 0.02
Relative	52.61 ± 0.66	52.79 ± 0.74	52.63 ± 0.68	51.27 ± 0.66
Lung				
Absolute	0.18 ± 0.01	0.20 ± 0.01	0.19 ± 0.01	0.17 ± 0.00
Relative	8.48 ± 0.45	9.14 ± 0.34	8.78 ± 0.40	8.18 ± 0.22
Thymus				
Absolute	0.056 ± 0.001	0.055 ± 0.002	0.054 ± 0.002	0.052 ± 0.002
Relative	2.57 ± 0.07	2.53 ± 0.08	2.52 ± 0.08	2.47 ± 0.08

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test

[a] Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error).

[b] n=9

TABLE G4
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Male				
Necropsy body wt.	34.8 ± 0.8	35.5 ± 0.4	33.2 ± 0.7	36.2 ± 0.7
Brain				
Absolute	0.48 ± 0.01	0.48 ± 0.01	0.47 ± 0.00	0.47 ± 0.00
Relative	13.74 ± 0.43	13.46 ± 0.21	14.15 ± 0.30	13.09 ± 0.26
R. Epididymis				
Absolute	0.0504 ± 0.0024	0.0499 ± 0.0020[b]	0.0472 ± 0.0021[b]	0.0521 ± 0.0036
Relative	1.45 ± 0.06	1.41 ± 0.07[b]	1.43 ± 0.08[b]	1.44 ± 0.10
L. Epididymis				
Absolute	0.0478 ± 0.0019	0.0510 ± 0.0020	0.0467 ± 0.0011	0.0508 ± 0.0033
Relative	1.38 ± 0.06	1.44 ± 0.05	1.41 ± 0.03	1.40 ± 0.09
Heart				
Absolute	0.16 ± 0.00[b]	0.16 ± 0.00[b]	0.16 ± 0.00	0.16 ± 0.01
Relative	4.52 ± 0.09[b]	4.65 ± 0.11[b]	4.70 ± 0.10	4.35 ± 0.09
R. Kidney				
Absolute	0.27 ± 0.01	0.28 ± 0.00	0.25 ± 0.01*	0.25 ± 0.01
Relative	7.80 ± 0.17	7.99 ± 0.16	7.54 ± 0.17	6.99 ± 0.17**
L. Kidney				
Absolute	0.26 ± 0.01	0.27 ± 0.01[b]	0.24 ± 0.00**	0.24 ± 0.00**
Relative	7.54 ± 0.15	7.57 ± 0.23[b]	7.10 ± 0.17	6.57 ± 0.12**
Liver				
Absolute	1.54 ± 0.05	1.58 ± 0.06	1.39 ± 0.04*	1.49 ± 0.04
Relative	44.27 ± 0.73	44.37 ± 1.38	41.76 ± 0.70	41.25 ± 0.85*
Lung				
Absolute	0.28 ± 0.02	0.28 ± 0.02	0.27 ± 0.02	0.31 ± 0.03
Relative	7.84 ± 0.58	7.84 ± 0.55	8.13 ± 0.46	8.62 ± 0.80
R. Testis				
Absolute	0.110 ± 0.002	0.109 ± 0.005	0.110 ± 0.003	0.110 ± 0.003
Relative	3.16 ± 0.10	3.09 ± 0.14	3.31 ± 0.10	3.04 ± 0.09
L. Testis				
Absolute	0.104 ± 0.002	0.106 ± 0.005	0.105 ± 0.001	0.109 ± 0.002
Relative	3.01 ± 0.12	2.99 ± 0.14	3.18 ± 0.07	3.01 ± 0.07
Thymus				
Absolute	0.035 ± 0.002	0.035 ± 0.002	0.033 ± 0.001	0.043 ± 0.002*
Relative	1.02 ± 0.07	1.00 ± 0.06	1.00 ± 0.03	1.18 ± 0.05

TABLE G4
Organ Weights and Organ-Weight-to-Body-Weight Ratios for Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Female				
Necropsy body wt.	24.4 ± 0.4	25.5 ± 0.5	25.7 ± 0.7	24.5 ± 0.3
Brain				
Absolute	0.49 ± 0.00	0.50 ± 0.01	0.49 ± 0.01	0.49 ± 0.00
Relative	20.21 ± 0.32	19.64 ± 0.50	19.28 ± 0.42	20.04 ± 0.27
Heart				
Absolute	0.15 ± 0.01	0.17 ± 0.01	0.16 ± 0.01	0.15 ± 0.00
Relative	6.24 ± 0.25	6.50 ± 0.15	6.30 ± 0.28	6.11 ± 0.16
R. Kidney				
Absolute	0.18 ± 0.00	0.18 ± 0.01	0.18 ± 0.00	0.16 ± 0.00
Relative	7.26 ± 0.13	7.16 ± 0.17	7.13 ± 0.22	6.68 ± 0.17
L. Kidney				
Absolute	0.16 ± 0.00	0.17 ± 0.00	0.17 ± 0.00	0.15 ± 0.00
Relative	6.52 ± 0.16	6.54 ± 0.11	6.47 ± 0.20	6.20 ± 0.16
Liver				
Absolute	1.21 ± 0.03	1.26 ± 0.03	1.25 ± 0.02	1.17 ± 0.04
Relative	49.60 ± 0.66	49.38 ± 0.72	48.97 ± 0.89	47.65 ± 1.15
Lung				
Absolute	0.31 ± 0.02	0.33 ± 0.02	0.33 ± 0.01	0.31 ± 0.01
Relative	12.59 ± 0.60	13.03 ± 0.48	12.87 ± 0.40	12.67 ± 0.52
R. Ovary				
Absolute	0.0077 ± 0.0004	0.0080 ± 0.0007	0.0071 ± 0.0007	0.0068 ± 0.0005
Relative	0.32 ± 0.02	0.32 ± 0.03	0.28 ± 0.03	0.28 ± 0.02
L. Ovary				
Absolute	0.0069 ± 0.0009	0.0068 ± 0.0005	0.0060 ± 0.0006	0.0055 ± 0.0005
Relative	0.28 ± 0.04	0.27 ± 0.02	0.23 ± 0.03	0.22 ± 0.02
Thymus				
Absolute	0.041 ± 0.003	0.043 ± 0.002	0.044 ± 0.002	0.045 ± 0.001
Relative	1.66 ± 0.10	1.70 ± 0.04	1.71 ± 0.05	1.83 ± 0.05

* Significantly different (P≤0.05) from the sham control group by Williams' or Dunnett's test
** P≤0.01
[a] Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error).
[b] n=9

APPENDIX H
REPRODUCTIVE TISSUE EVALUATIONS AND ESTROUS CYCLE CHARACTERIZATION

TABLE H1
Summary of Reproductive Tissue Evaluations for Male Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	34.8 ± 0.8	33.3 ± 0.8	34.3 ± 0.6	33.0 ± 0.5
L. Cauda epididymis	0.020 ± 0.001	0.020 ± 0.001	0.021 ± 0.001	0.019 ± 0.001
L. Epididymis	0.048 ± 0.002	0.050 ± 0.003	0.050 ± 0.003	0.047 ± 0.002
L. Testis	0.104 ± 0.002	0.107 ± 0.002	0.101 ± 0.007	0.105 ± 0.002
Spermatid measurements				
Spermatid heads (10^6/testis)	21.9 ± 1.9	22.2 ± 1.6	20.2 ± 3.0	22.4 ± 1.4
Spermatid heads (10^3/mg testis)	210.6 ± 17.0	208.2 ± 13.9	186.4 ± 26.6	213.0 ± 12.2
Epididymal spermatozoal measurements				
Sperm motility (%)	73.5 ± 5.7	66.8 ± 6.1	66.2 ± 7.9	76.8 ± 5.0
Sperm (10^6/cauda epididymis)	24.2 ± 4.7	18.0 ± 3.2	18.3 ± 2.2	15.9 ± 2.5
Sperm (10^3/mg cauda epididymis)	1,254.1 ± 258.5	921.1 ± 164.5	880.0 ± 122.2	825.1 ± 129.7

[a] Data are presented as mean ± standard error. Differences from the sham control group are not significant by Williams' or Dunnett's test (body and tissue weights) or Dunn's test (spermatid and epididymal spermatozoal measurements).

TABLE H2
Estrous Cycle Characterization for Female Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Number weighed at necropsy	10	10	10	10
Necropsy body wt (g)	24.4 ± 0.4	24.9 ± 0.5	25.0 ± 0.5	26.2 ± 0.7*
Proportion of regular cycling females[b]	10/10	10/10	10/10	10/10
Estrous cycle length (days)	4.0 ± 0.05	4.0 ± 0.03	4.2 ± 0.22	4.2 ± 0.21
Estrous stages (% of cycle)				
Diestrus	33.8	32.5	33.8	42.0
Proestrus	0.6	1.3	0.0	0.0
Estrus	51.0	49.0	49.7	47.8
Metestrus	14.6	15.9	15.9	9.6
Uncertain diagnoses	0.0	1.3	0.6	0.6

* Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test

[a] Necropsy body weights and estrous cycle length data are presented as mean ± standard error. Differences from the sham control group are not significant by Dunn's test (estrous cycle length). Tests for equality of transition probability matrices among all groups and between the sham control group and each exposed group indicated exposed females did not have extended estrus or diestrus.

[b] Number of females with a regular cycle/number of females cycling

Dose (W/kg)																							
0								E	E	D	E	E	D	D	E	E	D	D	E	E	M	D	E
0								E	M	D	E	D	D	D	E	E	M	D	E	E	D	D	
0								E	M	D	E	E	D	E	E	E	D	D	E	E	E	D	E
0								E	M	D	E	E	D	D	E	E	D	D	E	E	M	D	E
0							E	E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
0							E	E	M	D	E	E	M	D	E	E	M	D	E	E	M		
0							E	E	M	D	E	E	D	D	E	E	M	D	E	E	M	D	
0							E	E	M	D	E	E	D	D	E	E	M	D	E	E	M		
0							E	D	D	P	E	D	D	E	E	D	D	E	E	M	D	E	
0						D	E	E	D	D	E	E	D	D	E	E	M	D	E	E	M		
2.5								E	E	I	E	E	M	D	E	E	M	D	E	E	D	D	E
2.5								E	M	D	E	E	M	D	E	E	D	D	E	E	M	D	
2.5								E	M	D	E	E	D	E	E	E	D	D	D	E	M	D	E
2.5							E	E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
2.5							E	M	D	P	E	M	D	E	E	M	D	E	E	M	D		
2.5						E	E	M	D	P	E	D	D	E	E	M	D	E	M	D			
2.5						D	E	E	M	D	E	E	E	D	E	E	D	D	E	E	M		
2.5					M	D	E	E	M	D	E	E	D	D	E	E	M	I	E	E			
2.5					M	D	E	E	M	D	E	E	D	D	E	E	D	D	E	E			
2.5					D	D	E	E	D	D	E	E	D	D	E	E	D	D	E	E			
5								E	E	D	E	M	D	E	E	M	D	E	E	M	D	E	E
5								E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
5								E	D	D	E	E	M	D	E	E	M	D	E	E	M	D	E
5							E	E	D	D	E	E	D	D	E	E	M	D	E	E	D	D	
5							E	I	D	D	E	E	D	D	E	E	M	D	E	E	D		
5						E	E	E	M	D	E	E	M	D	E	E	E	M	D	E	E		
5						D	E	E	M	D	E	E	M	D	E	E	M	D	E	E	M		
5						D	E	E	D	D	E	E	D	D	E	E	M	D	E	E	M		
5					D	D	E	E	D	D	E	E	M	D	E	E	M	D	E				
5	D	E	E	M	D	D	D	D	D	D	E	D	E	E	E	M							
10								E	E	D	E	E	M	D	E	E	D	D	E	E	D	D	E
10								E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
10								E	M	D	E	E	D	D	E	E	D	D	E	E	M	D	
10								E	I	D	E	E	D	D	E	E	D	D	E	E	D	D	E
10						D	E	E	D	D	E	E	D	D	E	E	M	D	E	E	M		
10					M	D	E	E	D	D	E	E	M	D	E	E	M	D	E				
10					D	D	E	E	D	D	E	E	D	D	E	E	E	D	E	E			
10				D	D	E	E	E	D	D	E	E	D	D	E	E	D	D	E				
10				D	D	D	E	E	D	D	E	E	M	D	E	E	M	D	E				
10		E	E	D	D	D	D	D	D	D	E	M	D	E	E	D	D						

FIGURE H1
Vaginal Cytology Plots for Female Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study
I = Insufficient number of cells to determine stage; D = diestrus, P = proestrus, E = estrus, M = metestrus

TABLE H3
Results of Vaginal Cytology Study Using the Transition Matrix Approach in Female Mice at the 14-Week Interim Evaluation in the 2-Year GSM-Modulated Cell Phone RFR Study

Stage	Comparison	P Value	Trend[a]
Overall Tests	Overall	0.649	
Overall Tests	2.5 W/kg vs. Sham Controls	0.999	
Overall Tests	5 W/kg vs. Sham Controls	0.42	
Overall Tests	10 W/kg vs. Sham Controls	0.291	
Extended Estrus	Overall	0.997	
Extended Estrus	2.5 W/kg vs. Sham Controls	0.999	
Extended Estrus	5 W/kg vs. Sham Controls	0.755	
Extended Estrus	10 W/kg vs. Sham Controls	1	
Extended Diestrus	Overall	0.414	
Extended Diestrus	2.5 W/kg vs. Sham Controls	1	
Extended Diestrus	5 W/kg vs. Sham Controls	0.324	
Extended Diestrus	10 W/kg vs. Sham Controls	0.147	
Extended Metestrus	Overall	1	
Extended Metestrus	2.5 W/kg vs. Sham Controls	1	
Extended Metestrus	5 W/kg vs. Sham Controls	1	
Extended Metestrus	10 W/kg vs. Sham Controls	1	
Extended Proestrus	Overall	1	
Extended Proestrus	2.5 W/kg vs. Sham Controls	1	
Extended Proestrus	5 W/kg vs. Sham Controls	1	
Extended Proestrus	10 W/kg vs. Sham Controls	1	
Skipped Estrus	Overall	1	
Skipped Estrus	2.5 W/kg vs. Sham Controls	1	
Skipped Estrus	5 W/kg vs. Sham Controls	1	
Skipped Estrus	10 W/kg vs. Sham Controls	1	
Skipped Diestrus	Overall	1	
Skipped Diestrus	2.5 W/kg vs. Sham Controls	1	
Skipped Diestrus	5 W/kg vs. Sham Controls	1	
Skipped Diestrus	10 W/kg vs. Sham Controls	1	

[a] N means that the treated group had a lower probability of transitioning to the relevant abnormal state (extended estrus, extended diestrus, extended metestrus, extended proestrus, skipped estrus, or skipped diestrus) than did the sham control group.

TABLE H4
Summary of Reproductive Tissue Evaluations for Male Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	34.8 ± 0.8	35.5 ± 0.4	33.2 ± 0.7	36.2 ± 0.7
L. Cauda epididymis	0.020 ± 0.001	0.021 ± 0.001	0.020 ± 0.000	0.021 ± 0.000
L. Epididymis	0.048 ± 0.002	0.051 ± 0.002	0.047 ± 0.001	0.051 ± 0.003
L. Testis	0.104 ± 0.002	0.106 ± 0.005	0.105 ± 0.001	0.109 ± 0.002
Spermatid measurements				
Spermatid heads (10^6/testis)	21.9 ± 1.9	21.2 ± 1.9	23.4 ± 1.6	22.5 ± 1.8
Spermatid heads (10^3/mg testis)	210.6 ± 17.0	196.6 ± 11.1	222.8 ± 15.0	205.4 ± 14.6
Epididymal spermatozoal measurements				
Sperm motility (%)	73.5 ± 5.7	66.3 ± 6.7	67.5 ± 5.9	68.1 ± 8.3
Sperm (10^6/cauda epididymis)	24.2 ± 4.7	18.5 ± 4.8	13.0 ± 2.1	18.4 ± 1.5
Sperm (10^3/mg cauda epididymis)	1,254.1 ± 258.5	851.4 ± 181.3	674.6 ± 118.8	892.2 ± 69.2

[a] Data are presented as mean ± standard error. Differences from the sham control group are not significant by Williams' or Dunnett's test (body and tissue weights) or Dunn's test (spermatid and epididymal spermatozoal measurements).

TABLE H5
Estrous Cycle Characterization for Female Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study[a]

	Sham Control	2.5 W/kg	5 W/kg	10 W/kg
Number weighed at necropsy	10	10	10	10
Necropsy body wt (g)	24.4 ± 0.4	25.5 ± 0.5	25.7 ± 0.7	24.5 ± 0.3
Proportion of regular cycling females[b]	10/10	10/10	10/10	10/10
Estrous cycle length (days)	4.0 ± 0.05	4.8 ± 0.71	4.0 ± 0.07	4.0 ± 0.00
Estrous stages (% of cycle)				
Diestrus	33.8	34.8	42.0	29.9
Proestrus	0.6	1.3	0.6	1.9
Estrus	51.0	47.5	47.8	49.0
Metestrus	14.6	15.2	8.9	19.1
Uncertain diagnoses	0.0	1.3	0.6	0.0

[a] Necropsy body weights and estrous cycle length data are presented as mean ± standard error. Differences from the sham control group are not significant by Jonckheere's, Williams', or Dunnett's test (body weight) or Jonckheere's, Shirley's, or Dunn's test (estrous cycle length). Tests for equality of transition probability matrices among all groups and between the sham control group and each exposed group indicated exposed females did not have extended estrus or diestrus.

[b] Number of females with a regular cycle/number of females cycling

Dose (W/kg)																									
0											E	E	D	E	E	D	D	E	E	D	D	E	E	M	D	E
0											E	M	D	E	D	D	D	E	E	M	D	E	E	D	D	
0											E	M	D	E	E	D	E	E	E	D	D	E	E	E	D	E
0											E	M	D	E	E	D	D	E	E	D	D	E	E	M	D	E
0										E	E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
0										E	E	M	D	E	E	M	D	E	E	M	D	E	E	M		
0										E	E	M	D	E	E	D	D	E	E	M	D	E	E	M	D	
0										E	E	M	D	E	E	D	D	E	E	M	D	E	E	M		
0										E	D	D	P	E	D	D	E	E	D	D	E	E	M	D	E	
0									D	E	E	D	D	E	E	D	D	E	E	M	D	E	E	M		
2.5											E	M	D	E	E	E	M	D	E	E	E	M	D	E	E	E
2.5											E	D	D	E	E	M	D	E	E	M	D	E	E	M	D	E
2.5											E	D	D	E	E	D	D	E	E	M	D	E	M	E	D	E
2.5										E	E	M	D	E	E	I	D	E	E	D	D	E	E	M	D	
2.5										E	E	D	D	E	E	M	D	E	E	D	D	E	E	M	D	
2.5										E	M	D	P	E	D	E	E	D	D	D	E	E	D	D	E	
2.5									D	E	E	M	D	E	E	E	D	E	E	M	D	E	E	M		
2.5								M	D	E	E	D	D	E	E	M	D	E	E	M	D	E				
2.5								D	D	E	E	M	D	E	E	E	M	D	E	E	E	M				
2.5	D	P	E	M	D	D	D	D	D	D	D	I	D	E	M	D										
5											E	M	D	E	E	D	D	E	E	D	D	E	E	D	D	D
5											E	D	D	E	E	D	D	E	E	D	D	E	E	D	D	E
5											E	D	D	E	E	E	D	D	E	E	D	D	D	D	I	E
5									E	E	E	M	D	E	E	E	D	E	E	D	D	E	E	M		
5									E	E	E	D	D	E	E	M	D	E	E	M	D	E	E			
5									E	E	D	D	P	E	D	D	E	E	D	D	E	E	M	D		
5									D	E	E	M	D	E	E	D	D	E	E	M	D	E	E			
5									D	E	E	M	D	E	E	D	D	E	E	M	D	D	D	D		
5								D	D	E	E	M	D	E	E	M	D	E	E	D	D	E	E			
5					D	D	D	D	D	E	E	D	D	E	M	D	E	E	M							
10											E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	E
10										E	E	M	D	E	E	M	D	E	E	M	D	E	E	M	D	
10										E	E	D	D	E	E	M	D	E	E	M	D	E	E	M		
10										E	E	D	D	E	E	D	D	E	E	M	D	E	E	M		
10										E	D	D	P	E	M	D	E	E	M	D	E	E	M	D	E	
10									D	E	E	M	D	E	E	M	D	E	E	M	D	E	E	M		
10								M	D	E	E	M	D	E	E	M	D	E	E	D	D	E	E			
10								M	D	E	E	D	D	E	E	M	D	E	E	M	D	E	E			
10								M	D	E	E	D	D	E	E	D	D	E	E	M	D	E	E			
10								D	P	E	M	D	P	E	D	D	E	E	M	D	E	E				

FIGURE H2
Vaginal Cytology Plots for Female Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study
I = Insufficient number of cells to determine stage; D = diestrus, P = proestrus, E = estrus, M = metestrus

TABLE H6
Results of Vaginal Cytology Study Using the Transition Matrix Approach in Female Mice at the 14-Week Interim Evaluation in the 2-Year CDMA-Modulated Cell Phone RFR Study

Stage	Comparison	P Value	Trend[a]
Overall Tests	Overall	<0.001	
Overall Tests	2.5 W/kg vs. Sham Controls	<0.001	
Overall Tests	5 W/kg vs. Sham Controls	0.003	
Overall Tests	10 W/kg vs. Sham Controls	0.209	N
Extended Estrus	Overall	0.042	
Extended Estrus	2.5 W/kg vs. Sham Controls	0.012	
Extended Estrus	5 W/kg vs. Sham Controls	0.333	
Extended Estrus	10 W/kg vs. Sham Controls	0.358	N
Extended Diestrus	Overall	0.006	
Extended Diestrus	2.5 W/kg vs. Sham Controls	0.113	
Extended Diestrus	5 W/kg vs. Sham Controls	0.002	
Extended Diestrus	10 W/kg vs. Sham Controls	0.602	N
Extended Metestrus	Overall	1	
Extended Metestrus	2.5 W/kg vs. Sham Controls	1	
Extended Metestrus	5 W/kg vs. Sham Controls	1	
Extended Metestrus	10 W/kg vs. Sham Controls	1	
Extended Proestrus	Overall	1	
Extended Proestrus	2.5 W/kg vs. Sham Controls	1	
Extended Proestrus	5 W/kg vs. Sham Controls	1	
Extended Proestrus	10 W/kg vs. Sham Controls	1	
Skipped Estrus	Overall	1	
Skipped Estrus	2.5 W/kg vs. Sham Controls	1	
Skipped Estrus	5 W/kg vs. Sham Controls	1	
Skipped Estrus	10 W/kg vs. Sham Controls	1	
Skipped Diestrus	Overall	1	
Skipped Diestrus	2.5 W/kg vs. Sham Controls	0.934	
Skipped Diestrus	5 W/kg vs. Sham Controls	1	
Skipped Diestrus	10 W/kg vs. Sham Controls	1	
Summary of Significant Groups			
Overall Tests	2.5 W/kg vs. Sham Controls	<0.001	
Overall Tests	5 W/kg vs. Sham Controls	0.003	
Extended Estrus	2.5 W/kg vs. Sham Controls	0.012	
Extended Diestrus	5 W/kg vs. Sham Controls	0.002	

[a] N means that the treated group had a lower probability of transitioning to the relevant abnormal state (extended estrus, extended diestrus, extended metestrus, extended proestrus, skipped estrus, or skipped diestrus) than did the sham control group.

APPENDIX I
GSM- AND CDMA-MODULATED CELL PHONE RFR EXPOSURE DATA

GSM- AND CDMA-MODULATED CELL PHONE RFR EXPOSURE DATA

OVERVIEW

Exposure data include SAR (W/kg) (Tables I1 and I5), chamber field strength (V/m) (Tables I2 and I6), E- and H-field measurements (V/m) (Tables I3, I4, I7, and I8). For the medium and high dose GSM chambers, where a second E-field probe was used, the H-field measurements were converted from E-field measurements (E-field divided by 377). Fields were measured continuously throughout the study and measurements were automatically recorded approximately every 20 seconds. For every 20 second interval, the SAR was calculated based on the average H- and/or E-field data. The data presented for each exposure parameter include the mean and standard deviation [expressed in decibels (dB), W/kg, or V/m]; the total number of measurements recorded during the identified period of exposure (>44,000 calculated SAR per month and more than 1.1 million over the course of the 2-year study); the lowest (min) and highest (max) measurement recorded during the given exposure period; the number of measurements that were within the acceptable range; and the ratio of all measurements within range. The data reported for SAR also include the range of animal body weights (g) over the indicated time period of exposure, as well as the selected target SAR for each group. The data reported for field strengths (chamber, E-field, and H-field) include the target range of the field required to maintain appropriate SAR exposures. The minimum and maximum exposure values reported represent a single recorded measurement over the 2-year exposure period. The SAR and chamber-field in the sham and exposure chambers were within the target ranges (defined as ± 2 dB) for >99.85% of recorded measurements over the course of the 2-year study; ≥99.70% of E-field and H-field exposures in the sham and exposure chambers were within the target ranges for all but one chamber (97.35% within range).

The dB is a mathematical transformation of a number or numerical ratio using base 10 logarithms. Multiplication of ratios is transformed into addition of dBs; raising a number to a power is transformed into multiplication of dBs.

In general, dB(power) = 10 × log(R), and dB(field) = 20 × log(R). The formulas differ by a factor of two because power or SAR varies as the square of the fields. For SAR (in watts/kg), the decibel formula is calculated as:

$SAR(dB) = 10 \times \log(SAR_M/SAR_T)$
where SAR_M is the measured value and SAR_T is the target value, and

$-2\ dB = 10 \times \log(SAR_L/SAR_T)$, where SAR_L (low) $= SAR_T \times 10^{-0.2}$
$+2\ dB = 10 \times \log(SAR_H/SAR_T)$, where SAR_H (high) $= SAR_T \times 10^{0.2}$

On this basis, the ± 2 dB range specified by the NTP translates to the following ranges for each SAR used in the 2-year study:

Target SAR (W/kg)	Acceptable SAR Range (W/kg; ± 2 dB)
2.5	1.58 to 3.96
5	3.15 to 7.92
10	6.31 to 15.85

TABLE I1
Summary of GSM-Modulated Cell Phone RFR Exposure Data – SAR[a]

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
June 18 to 30, 2012								
Ch11 Mouse GSM High	18.9 to 20.2	10.00	10.08	0.23/0.05	3.944	23.576	19472/19475	1.000
Ch12 Mouse GSM Med	18.9 to 20.2	5.00	5.04	0.21/0.05	2.105	11.918	19472/19475	1.000
Ch14 Mouse GSM Low	18.8 to 20.2	2.50	2.51	0.20/0.05	1.948	3.175	19475/19475	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	19475/19475	1.000
July 1 to 31, 2012								
Ch11 Mouse GSM High	20.2 to 24.1	10.00	10.01	0.23/0.05	7.430	13.279	48731/48731	1.000
Ch12 Mouse GSM Med	20.2 to 24.6	5.00	5.01	0.21/0.05	3.349	7.170	48731/48731	1.000
Ch14 Mouse GSM Low	20.2 to 24.5	2.50	2.50	0.18/0.04	2.103	3.135	48731/48731	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48731/48731	1.000
August 1 to 31, 2012								
Ch11 Mouse GSM High	24.1 to 27.5	10.00	10.02	0.20/0.05	6.893	13.910	47488/47488	1.000
Ch12 Mouse GSM Med	24.6 to 27.9	5.00	5.03	0.20/0.05	3.911	6.803	47488/47488	1.000
Ch14 Mouse GSM Low	24.5 to 27.2	2.50	2.50	0.18/0.04	1.900	3.441	47488/47488	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47488/47488	1.000
September 1 to 30, 2012								
Ch11 Mouse GSM High	27.5 to 29.3	10.00	10.01	0.21/0.05	5.187	12.693	47186/47187	1.000
Ch12 Mouse GSM Med	27.9 to 30.0	5.00	5.01	0.19/0.04	2.558	6.280	47184/47185	1.000
Ch14 Mouse GSM Low	27.2 to 29.2	2.50	2.51	0.18/0.04	1.444	3.129	47184/47185	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47185/47185	1.000
October 1 to 31, 2012								
Ch11 Mouse GSM High	29.3 to 32.9	10.00	10.02	0.19/0.05	3.290	12.620	48801/48802	1.000
Ch12 Mouse GSM Med	30.0 to 33.8	5.00	5.02	0.18/0.04	3.828	6.511	48801/48801	1.000
Ch14 Mouse GSM Low	29.2 to 32.7	2.50	2.51	0.17/0.04	2.017	3.080	48801/48801	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48801/48801	1.000
November 1 to 30, 2012								
Ch11 Mouse GSM High	32.9 to 36.6	10.00	10.03	0.19/0.04	7.649	13.824	47314/47314	1.000
Ch12 Mouse GSM Med	33.8 to 36.4	5.00	5.02	0.17/0.04	3.724	6.537	47314/47314	1.000
Ch14 Mouse GSM Low	32.7 to 35.8	2.50	2.51	0.17/0.04	2.054	3.110	47314/47314	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47314/47314	1.000
December 1 to 31, 2012								
Ch11 Mouse GSM High	36.6 to 39.1	10.00	10.07	0.20/0.05	7.992	12.863	48750/48750	1.000
Ch12 Mouse GSM Med	36.4 to 39.2	5.00	5.04	0.18/0.04	4.145	6.028	48748/48748	1.000
Ch14 Mouse GSM Low	35.8 to 38.1	2.50	2.52	0.17/0.04	2.139	3.109	48748/48748	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48748/48748	1.000

[a] Ch=chamber (*e.g.*, Ch11=Chamber 11)

TABLE I1
Summary of GSM-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
January 1 to 31, 2013								
Ch11 Mouse GSM High	39.1 to 41.1	10.00	9.78	0.30/0.07	7.230	13.516	48689/48689	1.000
Ch12 Mouse GSM Med	39.2 to 41.6	5.00	5.02	0.20/0.05	3.121	7.619	48681/48682	1.000
Ch14 Mouse GSM Low	38.1 to 40.6	2.50	2.51	0.17/0.04	2.037	2.987	48682/48682	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48682/48682	1.000
February 1 to 28, 2013								
Ch11 Mouse GSM High	41.1 to 42.9	10.00	8.23	0.22/0.05	6.187	10.364	44057/44058	1.000
Ch12 Mouse GSM Med	41.6 to 43.5	5.00	5.02	0.17/0.04	3.872	5.967	44058/44058	1.000
Ch14 Mouse GSM Low	40.6 to 42.2	2.50	2.52	0.17/0.04	1.851	3.048	44058/44058	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	44058/44058	1.000
March 1 to 31, 2013								
Ch11 Mouse GSM High	42.9 to 44.4	10.00	8.25	0.24/0.06	6.753	10.627	48892/48892	1.000
Ch12 Mouse GSM Med	43.5 to 44.7	5.00	5.04	0.17/0.04	4.276	5.871	48892/48892	1.000
Ch14 Mouse GSM Low	42.2 to 43.4	2.50	2.51	0.17/0.04	2.143	2.943	48892/48892	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48892/48892	1.000
April 1 to 30, 2013								
Ch11 Mouse GSM High	44.4 to 46.8	10.00	7.95	0.20/0.05	6.370	9.872	48130/48130	1.000
Ch12 Mouse GSM Med	44.7 to 47.3	5.00	5.02	0.17/0.04	4.113	5.862	48130/48130	1.000
Ch14 Mouse GSM Low	43.4 to 46.0	2.50	2.52	0.16/0.04	2.214	2.995	48130/48130	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48130/48130	1.000
May 1 to 31, 2013								
Ch11 Mouse GSM High	46.8 to 47.9	10.00	7.94	0.19/0.04	5.057	9.940	48509/48510	1.000
Ch12 Mouse GSM Med	47.3 to 48.7	5.00	5.01	0.17/0.04	3.264	7.176	48510/48510	1.000
Ch14 Mouse GSM Low	46.0 to 47.5	2.50	2.51	0.17/0.04	1.809	2.957	48510/48510	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48510/48510	1.000
June 1 to 30, 2013								
Ch11 Mouse GSM High	47.9 to 49.2	10.00	7.44	0.17/0.04	6.134	9.224	47246/47248	1.000
Ch12 Mouse GSM Med	48.7 to 49.9	5.00	5.01	0.17/0.04	4.185	5.911	47248/47248	1.000
Ch14 Mouse GSM Low	47.5 to 49.0	2.50	2.50	0.16/0.04	1.964	2.923	47248/47248	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47248/47248	1.000
July 1 to 31, 2013								
Ch11 Mouse GSM High	49.2 to 50.5	10.00	8.02	0.26/0.06	5.376	10.141	49496/49573	0.998
Ch12 Mouse GSM Med	49.9 to 51.5	5.00	5.01	0.17/0.04	3.448	5.910	49573/49573	1.000
Ch14 Mouse GSM Low	49.0 to 50.2	2.50	2.51	0.17/0.04	1.876	2.915	49573/49573	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	49573/49573	1.000

TABLE I1
Summary of GSM-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2013								
Ch11 Mouse GSM High	50.5 to 51.3	10.00	8.33	0.22/0.05	6.666	10.172	50850/50850	1.000
Ch12 Mouse GSM Med	51.5 to 52.5	5.00	4.99	0.18/0.04	4.182	6.536	50850/50850	1.000
Ch14 Mouse GSM Low	50.2 to 51.3	2.50	2.50	0.17/0.04	2.065	3.317	50850/50850	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50850/50850	1.000
September 1 to 30, 2013								
Ch11 Mouse GSM High	51.3 to 52.0	10.95	9.88	0.40/0.10	3.473	14.080	46959/46961	1.000
Ch12 Mouse GSM Med	52.5 to 53.2	5.00	5.01	0.18/0.04	4.292	5.988	46960/46960	1.000
Ch14 Mouse GSM Low	51.3 to 52.2	2.50	2.50	0.17/0.04	1.329	2.996	46956/46960	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	46960/46960	1.000
October 1 to 31, 2013								
Ch11 Mouse GSM High	52.0 to 53.3	10.95	10.83	0.22/0.05	6.969	14.792	50408/50408	1.000
Ch12 Mouse GSM Med	53.2 to 54.0	5.00	5.01	0.19/0.04	3.215	5.974	50408/50408	1.000
Ch14 Mouse GSM Low	52.2 to 53.0	2.50	2.50	0.16/0.04	1.669	3.009	50408/50408	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50408/50408	1.000
November 1 to 30, 2013								
Ch11 Mouse GSM High	52.4 to 53.3	10.95	9.54	0.35/0.08	5.677	13.090	46609/46613	1.000
Ch12 Mouse GSM Med	53.1 to 54.0	5.00	4.96	0.24/0.06	2.923	7.086	46612/46613	1.000
Ch14 Mouse GSM Low	52.4 to 53.0	2.50	2.50	0.19/0.05	1.629	2.955	46613/46613	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	46613/46613	1.000
December 1 to 31, 2013								
Ch11 Mouse GSM High	51.4 to 52.4	10.95	10.22	0.36/0.09	7.156	13.073	48423/48423	1.000
Ch12 Mouse GSM Med	51.9 to 53.1	5.00	4.96	0.21/0.05	3.618	7.513	48423/48423	1.000
Ch14 Mouse GSM Low	51.1 to 52.4	2.50	2.50	0.18/0.04	1.475	4.060	48421/48423	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48423/48423	1.000
January 1 to 31, 2014								
Ch11 Mouse GSM High	51.4 to 52.1	10.95	10.08	0.27/0.06	6.308	13.144	48774/48775	1.000
Ch12 Mouse GSM Med	51.9 to 52.7	5.00	5.00	0.20/0.05	3.593	6.330	48775/48775	1.000
Ch14 Mouse GSM Low	51.1 to 51.6	2.50	2.50	0.18/0.04	0.472	3.843	48772/48775	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48775/48775	1.000
February 1 to 28, 2014								
Ch11 Mouse GSM High	52.1 to 53.2	10.95	10.92	0.24/0.06	8.258	15.024	44092/44092	1.000
Ch12 Mouse GSM Med	52.7 to 53.6	5.00	4.59	0.41/0.10	2.273	6.443	43990/44092	0.998
Ch14 Mouse GSM Low	51.6 to 53.1	2.50	2.50	0.18/0.04	1.622	3.807	44092/44092	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	44092/44092	1.000

TABLE I1
Summary of GSM-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
March 1 to 31, 2014								
Ch11 Mouse GSM High	52.8 to 53.8	10.95	10.27	0.43/0.10	6.995	15.483	48571/48571	1.000
Ch12 Mouse GSM Med	53.3 to 53.8	5.00	4.56	0.60/0.15	2.706	6.677	47927/48571	0.987
Ch14 Mouse GSM Low	52.8 to 53.3	2.50	2.51	0.16/0.04	1.877	3.110	48571/48571	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48571/48571	1.000
April 1 to 30, 2014								
Ch11 Mouse GSM High	51.5 to 52.3	10.95	10.73	0.41/0.10	7.050	14.898	47274/47274	1.000
Ch12 Mouse GSM Med	53.0 to 53.4	5.00	4.92	0.66/0.16	2.581	8.007	46546/47274	0.985
Ch14 Mouse GSM Low	51.9 to 52.3	2.50	2.50	0.15/0.03	1.868	2.893	47274/47274	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47274/47274	1.000
May 1 to 31, 2014								
Ch11 Mouse GSM High	50.5 to 51.5	10.95	10.95	0.22/0.05	6.826	14.016	48620/48622	1.000
Ch12 Mouse GSM Med	51.3 to 53.0	5.00	5.00	0.22/0.05	2.622	7.487	48550/48622	0.999
Ch14 Mouse GSM Low	51.2 to 51.9	2.50	2.50	0.15/0.03	2.188	2.921	48622/48622	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48622/48622	1.000
June 1 to 30, 2014								
Ch11 Mouse GSM High	49.4 to 50.5	10.95	10.97	0.20/0.05	8.188	13.942	47144/47144	1.000
Ch12 Mouse GSM Med	50.8 to 51.3	5.00	5.01	0.16/0.04	3.005	5.830	47142/47144	1.000
Ch14 Mouse GSM Low	50.2 to 51.2	2.50	2.51	0.15/0.03	2.153	2.870	47144/47144	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47144/47144	1.000
July 1 to 9, 2014								
Ch11 Mouse GSM High	49.4 to 49.4	10.95	11.08	0.19/0.04	8.839	13.746	12532/12532	1.000
Ch12 Mouse GSM Med	50.8 to 50.8	5.00	5.02	0.15/0.04	4.488	5.844	12532/12532	1.000
Ch14 Mouse GSM Low	50.2 to 50.2	2.50	2.51	0.17/0.04	1.995	3.111	12532/12532	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	12532/12532	1.000
June 18, 2012, to July 9, 2014								
Ch11 Mouse GSM High	18.9 to 53.8	10.95	9.56	0.44/0.11	3.290	23.576	1136126/1136221	1.000
Ch12 Mouse GSM Med	18.9 to 54.0	5.00	4.97	0.29/0.07	2.105	11.918	1134656/1136208	0.999
Ch14 Mouse GSM Low	18.8 to 53.3	2.50	2.51	0.17/0.04	0.472	4.060	1136198/1136208	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1136208/1136208	1.000

TABLE I2
Summary of GSM-Modulated Cell Phone RFR Exposure Data – Chamber Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch11 Mouse GSM High	194.10 to 199.60	197.33	0.23/5.27	121.76	297.71	38944/38950	1.000
Ch12 Mouse GSM Med	137.20 to 141.20	139.51	0.21/3.45	88.97	211.67	38944/38950	1.000
Ch14 Mouse GSM Low	97.00 to 99.80	98.42	0.20/2.26	88.09	109.25	38950/38950	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	38950/38950	1.000
July 1 to 31, 2012							
Ch11 Mouse GSM High	199.60 to 212.30	205.98	0.23/5.60	180.13	237.61	97462/97462	1.000
Ch12 Mouse GSM Med	141.20 to 150.10	145.84	0.21/3.55	119.12	174.30	97462/97462	1.000
Ch14 Mouse GSM Low	99.80 to 106.10	103.40	0.18/2.22	92.84	116.92	97462/97462	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97462/97462	1.000
August 1 to 31, 2012							
Ch11 Mouse GSM High	212.30 to 222.20	216.19	0.20/5.04	181.61	257.98	94976/94976	1.000
Ch12 Mouse GSM Med	150.10 to 157.10	154.16	0.20/3.67	136.80	180.42	94976/94976	1.000
Ch14 Mouse GSM Low	106.10 to 111.10	108.48	0.18/2.26	95.61	130.19	94976/94976	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94976/94976	1.000
September 1 to 30, 2012							
Ch11 Mouse GSM High	222.20 to 228.20	226.30	0.21/5.48	162.27	251.45	94372/94374	1.000
Ch12 Mouse GSM Med	157.10 to 161.40	160.65	0.19/3.49	115.42	179.26	94368/94370	1.000
Ch14 Mouse GSM Low	111.10 to 114.10	112.89	0.18/2.35	85.61	126.49	94368/94370	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94370/94370	1.000
October 1 to 31, 2012							
Ch11 Mouse GSM High	228.20 to 235.60	232.23	0.20/5.31	135.19	264.78	97602/97602	1.000
Ch12 Mouse GSM Med	161.40 to 168.00	165.17	0.18/3.51	141.20	191.80	97602/97602	1.000
Ch14 Mouse GSM Low	114.10 to 117.80	116.22	0.17/2.29	102.50	129.10	97602/97602	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97602/97602	1.000
November 1 to 30, 2012							
Ch11 Mouse GSM High	235.60 to 242.70	240.08	0.19/5.34	212.13	285.16	94628/94628	1.000
Ch12 Mouse GSM Med	168.00 to 171.70	170.49	0.17/3.37	145.05	192.18	94628/94628	1.000
Ch14 Mouse GSM Low	117.80 to 120.60	119.65	0.17/2.35	108.08	134.46	94628/94628	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94628/94628	1.000
December 1 to 31, 2012							
Ch11 Mouse GSM High	242.70 to 247.20	246.27	0.20/5.79	220.75	280.06	97500/97500	1.000
Ch12 Mouse GSM Med	171.70 to 174.80	174.25	0.18/3.56	158.98	191.03	97496/97496	1.000
Ch14 Mouse GSM Low	120.60 to 122.90	122.40	0.17/2.38	111.52	136.85	97496/97496	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch11=Chamber 11)

TABLE I2
Summary of GSM-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch11 Mouse GSM High	247.20 to 249.80	246.34	0.31/8.90	212.57	290.65	97378/97378	1.000
Ch12 Mouse GSM Med	174.80 to 176.70	176.61	0.21/4.30	139.67	218.22	97362/97364	1.000
Ch14 Mouse GSM Low	122.90 to 124.30	124.22	0.17/2.39	112.14	135.78	97364/97364	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97364/97364	1.000
February 1 to 28, 2013							
Ch11 Mouse GSM High	249.80 to 251.10	227.29	0.25/6.60	197.26	254.51	88114/88116	1.000
Ch12 Mouse GSM Med	176.70 to 178.50	178.60	0.17/3.60	157.05	194.95	88116/88116	1.000
Ch14 Mouse GSM Low	124.30 to 125.60	125.67	0.17/2.43	107.89	138.45	88116/88116	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	88116/88116	1.000
March 1 to 31, 2013							
Ch11 Mouse GSM High	251.10 to 253.60	229.49	0.26/7.11	207.98	261.00	97784/97784	1.000
Ch12 Mouse GSM Med	178.50 to 179.30	179.57	0.17/3.62	165.02	193.99	97784/97784	1.000
Ch14 Mouse GSM Low	125.60 to 126.20	126.39	0.17/2.55	116.82	136.92	97784/97784	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97784/97784	1.000
April 1 to 30, 2013							
Ch11 Mouse GSM High	253.60 to 256.00	227.10	0.23/5.99	203.37	253.19	96260/96260	1.000
Ch12 Mouse GSM Med	179.30 to 181.80	180.58	0.18/3.69	163.42	195.38	96260/96260	1.000
Ch14 Mouse GSM Low	126.20 to 127.40	127.12	0.16/2.42	119.14	138.56	96260/96260	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	96260/96260	1.000
May 1 to 31, 2013							
Ch11 Mouse GSM High	256.00 to 257.20	227.85	0.21/5.62	181.80	254.88	97018/97020	1.000
Ch12 Mouse GSM Med	181.80 to 182.60	182.15	0.17/3.65	147.02	218.01	97020/97020	1.000
Ch14 Mouse GSM Low	127.40 to 128.60	127.68	0.17/2.51	108.39	138.57	97020/97020	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97020/97020	1.000
June 1 to 30, 2013							
Ch11 Mouse GSM High	257.20 to 259.40	222.18	0.20/5.26	202.90	247.16	94490/94496	1.000
Ch12 Mouse GSM Med	182.60 to 183.40	182.92	0.17/3.62	167.03	198.51	94496/94496	1.000
Ch14 Mouse GSM Low	128.60 to 129.10	128.74	0.16/2.39	114.06	139.13	94496/94496	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94496/94496	1.000
July 1 to 31, 2013							
Ch11 Mouse GSM High	259.40 to 260.60	232.30	0.29/7.92	189.95	261.63	98976/99146	0.998
Ch12 Mouse GSM Med	183.40 to 185.00	183.88	0.17/3.71	152.12	201.19	99146/99146	1.000
Ch14 Mouse GSM Low	129.10 to 130.30	129.63	0.17/2.54	111.83	140.17	99146/99146	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	99146/99146	1.000

TABLE I2
Summary of GSM-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch11 Mouse GSM High	260.60 to 261.60	238.28	0.24/6.76	212.95	263.95	101700/101700	1.000
Ch12 Mouse GSM Med	185.00 to 185.80	185.04	0.18/3.97	169.82	211.58	101700/101700	1.000
Ch14 Mouse GSM Low	130.30 to 130.80	130.56	0.17/2.57	118.52	150.21	101700/101700	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	101700/101700	1.000
September 1 to 30, 2013							
Ch11 Mouse GSM High	261.60 to 273.80	259.49	0.42/12.99	154.24	310.55	93918/93922	1.000
Ch12 Mouse GSM Med	185.80 to 186.60	186.08	0.19/4.02	172.65	203.93	93920/93920	1.000
Ch14 Mouse GSM Low	130.80 to 131.40	131.14	0.17/2.55	95.42	143.74	93912/93920	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	93920/93920	1.000
October 1 to 31, 2013							
Ch11 Mouse GSM High	273.80 to 276.10	273.26	0.23/7.32	219.98	320.51	100816/100816	1.000
Ch12 Mouse GSM Med	186.60 to 186.60	186.50	0.19/4.08	149.42	203.69	100816/100816	1.000
Ch14 Mouse GSM Low	131.40 to 131.40	131.40	0.16/2.42	107.28	144.05	100816/100816	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	100816/100816	1.000
November 1 to 30, 2013							
Ch11 Mouse GSM High	275.00 to 276.10	256.54	0.37/11.29	197.87	301.50	93218/93226	1.000
Ch12 Mouse GSM Med	186.60 to 186.60	185.47	0.25/5.34	142.46	221.82	93224/93226	1.000
Ch14 Mouse GSM Low	131.40 to 131.40	131.34	0.20/3.01	106.00	142.76	93226/93226	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	93226/93226	1.000
December 1 to 31, 2013							
Ch11 Mouse GSM High	273.80 to 275.00	264.56	0.38/11.98	222.16	299.75	96846/96846	1.000
Ch12 Mouse GSM Med	185.00 to 186.60	184.56	0.21/4.62	157.41	228.42	96846/96846	1.000
Ch14 Mouse GSM Low	130.80 to 131.40	130.98	0.18/2.74	100.51	166.76	96842/96846	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	96846/96846	1.000
January 1 to 31, 2014							
Ch11 Mouse GSM High	273.80 to 275.00	263.34	0.29/8.82	208.57	301.08	97548/97550	1.000
Ch12 Mouse GSM Med	185.00 to 185.80	185.51	0.20/4.28	157.42	208.93	97550/97550	1.000
Ch14 Mouse GSM Low	130.80 to 130.80	130.79	0.18/2.80	56.85	162.24	97544/97550	1.000
Ch13 Mouse Sham	000.00 to 000.00	0.00	–/0.00	0.00	0.00	97550/97550	1.000
February 1 to 28, 2014							
Ch11 Mouse GSM High	275.00 to 276.10	275.23	0.24/7.74	239.47	323.01	88184/88184	1.000
Ch12 Mouse GSM Med	185.80 to 186.60	178.26	0.45/9.39	125.65	211.52	87980/88184	0.998
Ch14 Mouse GSM Low	130.80 to 131.90	131.73	0.18/2.73	106.14	162.60	88184/88184	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	88184/88184	1.000

TABLE I2
Summary of GSM-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch11 Mouse GSM High	275.00 to 276.10	266.21	0.46/14.46	220.40	327.90	97142/97142	1.000
Ch12 Mouse GSM Med	186.60 to 186.60	177.33	0.67/14.20	137.09	215.33	95760/97142	0.986
Ch14 Mouse GSM Low	131.40 to 131.90	131.77	0.16/2.40	114.17	146.97	97142/97142	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97142/97142	1.000
April 1 to 30, 2014							
Ch11 Mouse GSM High	273.80 to 275.00	271.16	0.43/13.88	220.51	319.44	94548/94548	1.000
Ch12 Mouse GSM Med	185.80 to 186.60	184.10	0.70/15.37	133.88	235.80	93074/94548	0.984
Ch14 Mouse GSM Low	130.80 to 131.40	131.27	0.15/2.29	113.50	141.25	94548/94548	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94548/94548	1.000
May 1 to 31, 2014							
Ch11 Mouse GSM High	272.70 to 273.80	273.60	0.23/7.40	216.22	309.84	97240/97244	1.000
Ch12 Mouse GSM Med	185.00 to 185.80	185.60	0.24/5.11	134.48	227.23	97098/97244	0.998
Ch14 Mouse GSM Low	130.80 to 130.80	130.87	0.15/2.24	122.41	141.45	97244/97244	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97244/97244	1.000
June 1 to 30, 2014							
Ch11 Mouse GSM High	271.50 to 272.70	272.64	0.20/6.44	236.01	307.96	94288/94288	1.000
Ch12 Mouse GSM Med	184.20 to 185.00	184.73	0.16/3.49	142.97	199.14	94286/94288	1.000
Ch14 Mouse GSM Low	130.30 to 130.80	130.94	0.15/2.22	121.45	140.21	94288/94288	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94288/94288	1.000
July 1 to 9, 2014							
Ch11 Mouse GSM High	271.50 to 271.50	272.57	0.19/5.96	243.57	303.74	25064/25064	1.000
Ch12 Mouse GSM Med	184.20 to 184.20	184.80	0.15/3.27	174.73	199.39	25064/25064	1.000
Ch14 Mouse GSM Low	130.30 to 130.30	130.69	0.17/2.66	116.49	145.47	25064/25064	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch11 Mouse GSM High	194.10 to 276.10	244.10	0.48/13.78	121.76	327.90	2272234/2272442	1.000
Ch12 Mouse GSM Med	137.20 to 186.60	176.76	0.31/6.35	88.97	235.80	2269198/2272416	0.999
Ch14 Mouse GSM Low	97.00 to 131.90	125.14	0.17/2.48	56.85	166.76	2272396/2272416	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	2272416/2272416	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data – E-Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch11 Mouse GSM High	194.1 to 199.6	220.41	0.36/9.20	134.8	342.3	38908/38950	0.999
Ch12 Mouse GSM Med	137.2 to 141.2	148.22	0.31/5.41	93.1	224.6	38944/38950	1.000
Ch14 Mouse GSM Low	97.0 to 99.8	93.00	0.25/2.72	82.8	106.6	38950/38950	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	38950/38950	1.000
July 1 to 31, 2012							
Ch11 Mouse GSM High	199.6 to 212.3	232.54	0.37/10.25	191.3	275.5	97310/97462	0.998
Ch12 Mouse GSM Med	141.2 to 150.1	154.86	0.31/5.70	121.0	190.6	97460/97462	1.000
Ch14 Mouse GSM Low	99.8 to 106.1	96.90	0.23/2.66	85.6	111.1	97462/97462	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97462/97462	1.000
August 1 to 31, 2012							
Ch11 Mouse GSM High	212.3 to 222.2	249.45	0.37/10.92	204.4	315.0	94710/94976	0.997
Ch12 Mouse GSM Med	150.1 to 157.1	164.75	0.31/5.97	144.8	197.2	94968/94976	1.000
Ch14 Mouse GSM Low	106.1 to 111.1	101.69	0.23/2.69	89.1	120.4	94976/94976	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94976/94976	1.000
September 1 to 30, 2012							
Ch11 Mouse GSM High	222.2 to 228.2	267.26	0.37/11.47	193.8	317.6	93216/94374	0.988
Ch12 Mouse GSM Med	157.1 to 161.4	171.56	0.31/6.17	119.3	197.8	94366/94370	1.000
Ch14 Mouse GSM Low	114.1 to 117.8	110.31	0.25/3.25	94.8	125.7	94962/94962	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94962/94962	1.000
October 1 to 31, 2012							
Ch11 Mouse GSM High	228.2 to 235.6	270.30	0.36/11.39	161.2	317.0	97028/97604	0.994
Ch12 Mouse GSM Med	161.4 to 168.0	172.36	0.29/5.83	149.2	200.7	97602/97602	1.000
Ch14 Mouse GSM Low	114.1 to 117.8	110.32	0.25/3.26	94.8	125.7	97602/97602	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97602/97602	1.000
November 1 to 30, 2012							
Ch11 Mouse GSM High	235.6 to 242.7	280.59	0.36/11.92	236.7	351.9	93952/94628	0.993
Ch12 Mouse GSM Med	168.0 to 171.7	175.49	0.26/5.33	150.0	202.3	94628/94628	1.000
Ch14 Mouse GSM Low	117.8 to 120.6	112.89	0.23/3.09	99.2	127.3	94628/94628	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94628/94628	1.000
December 1 to 31, 2012							
Ch11 Mouse GSM High	242.7 to 247.2	291.82	0.38/13.01	253.5	340.0	95756/97500	0.982
Ch12 Mouse GSM Med	171.7 to 174.8	180.56	0.27/5.72	161.5	204.8	97496/97496	1.000
Ch14 Mouse GSM Low	120.6 to 122.9	117.17	0.24/3.25	104.1	133.5	97496/97496	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch11=Chamber 11)

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch11 Mouse GSM High	247.2 to 249.8	285.35	0.64/21.65	229.0	359.4	96120/97378	0.987
Ch12 Mouse GSM Med	174.8 to 176.7	183.63	0.30/6.49	148.1	233.4	97362/97364	1.000
Ch14 Mouse GSM Low	122.9 to 124.3	119.13	0.24/3.35	104.9	133.0	97364/97364	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97364/97364	1.000
February 1 to 28, 2013							
Ch11 Mouse GSM High	249.8 to 251.1	245.59	0.41/11.75	208.2	292.5	88116/88116	1.000
Ch12 Mouse GSM Med	176.7 to 178.5	187.59	0.28/6.15	161.8	212.4	88116/88116	1.000
Ch14 Mouse GSM Low	124.3 to 125.6	119.66	0.24/3.39	99.0	135.0	88114/88116	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	88116/88116	1.000
March 1 to 31, 2013							
Ch11 Mouse GSM High	251.1 to 253.6	248.20	0.40/11.68	213.4	298.3	97784/97784	1.000
Ch12 Mouse GSM Med	178.5 to 179.3	187.29	0.30/6.68	163.4	215.1	97784/97784	1.000
Ch14 Mouse GSM Low	125.6 to 126.2	121.50	0.25/3.56	109.2	138.8	97784/97784	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97784/97784	1.000
April 1 to 30, 2013							
Ch11 Mouse GSM High	253.6 to 256.0	245.75	0.37/10.67	206.0	283.6	96260/96260	1.000
Ch12 Mouse GSM Med	179.3 to 181.8	188.33	0.28/6.14	169.2	215.0	96260/96260	1.000
Ch14 Mouse GSM Low	126.2 to 127.4	119.94	0.24/3.42	106.9	134.6	96260/96260	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	96260/96260	1.000
May 1 to 31, 2013							
Ch11 Mouse GSM High	256.0 to 257.2	245.46	0.34/9.92	192.9	297.6	97018/97020	1.000
Ch12 Mouse GSM Med	181.8 to 182.6	184.92	0.36/7.72	154.7	219.8	97020/97020	1.000
Ch14 Mouse GSM Low	127.4 to 128.6	123.25	0.25/3.53	101.9	137.4	97020/97020	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97020/97020	1.000
June 1 to 30, 2013							
Ch11 Mouse GSM High	257.2 to 259.4	238.55	0.31/8.53	208.8	277.6	94496/94496	1.000
Ch12 Mouse GSM Med	182.6 to 183.4	180.20	0.27/5.60	160.4	204.7	94496/94496	1.000
Ch14 Mouse GSM Low	128.6 to 129.1	122.13	0.22/3.17	106.8	134.2	94496/94496	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94496/94496	1.000
July 1 to 31, 2013							
Ch11 Mouse GSM High	259.4 to 260.6	252.77	0.48/14.26	209.6	302.8	99146/99146	1.000
Ch12 Mouse GSM Med	183.4 to 185.0	180.18	0.26/5.54	149.9	202.6	99146/99146	1.000
Ch14 Mouse GSM Low	129.1 to 130.3	122.67	0.24/3.49	99.9	137.4	99144/99146	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	99146/99146	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch11 Mouse GSM High	260.6 to 261.6	238.13	0.36/10.20	205.2	287.5	101698/101700	1.000
Ch12 Mouse GSM Med	185.0 to 185.8	180.96	0.29/6.22	160.7	210.2	101700/101700	1.000
Ch14 Mouse GSM Low	130.3 to 130.8	124.66	0.25/3.62	109.9	141.5	101700/101700	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	101700/101700	1.000
September 1 to 30, 2013							
Ch11 Mouse GSM High	261.6 to 273.8	251.71	0.43/12.83	161.3	311.1	93890/93922	1.000
Ch12 Mouse GSM Med	185.8 to 186.6	181.55	0.28/5.84	159.1	205.9	93920/93920	1.000
Ch14 Mouse GSM Low	130.8 to 131.4	124.86	0.25/3.59	89.3	140.0	93912/93920	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	93920/93920	1.000
October 1 to 31, 2013							
Ch11 Mouse GSM High	273.8 to 276.1	264.68	0.29/9.12	209.0	306.5	100814/100816	1.000
Ch12 Mouse GSM Med	186.6 to 186.6	182.15	0.28/5.91	145.4	205.6	100814/100816	1.000
Ch14 Mouse GSM Low	131.4 to 131.4	123.98	0.24/3.55	105.9	138.5	100816/100816	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100816/100816	1.000
November 1 to 30, 2013							
Ch11 Mouse GSM High	275.0 to 276.1	257.28	0.39/11.82	206.1	315.3	93220/93226	1.000
Ch12 Mouse GSM Med	186.6 to 186.6	184.13	0.34/7.34	135.2	228.1	93216/93226	1.000
Ch14 Mouse GSM Low	131.4 to 131.4	126.40	0.28/4.12	100.9	141.5	93198/93226	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	93226/93226	1.000
December 1 to 31, 2013							
Ch11 Mouse GSM High	273.8 to 275.0	265.70	0.39/12.26	214.7	308.3	96844/96846	1.000
Ch12 Mouse GSM Med	185.0 to 186.6	179.76	0.29/6.11	155.1	224.0	96846/96846	1.000
Ch14 Mouse GSM Low	130.8 to 131.4	122.89	0.25/3.62	95.3	152.2	96842/96846	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	96846/96846	1.000
January 1 to 31, 2014							
Ch11 Mouse GSM High	273.8 to 275.0	266.12	0.43/13.48	208.2	324.0	97548/97550	1.000
Ch12 Mouse GSM Med	185.0 to 185.8	180.59	0.27/5.78	156.4	207.5	97550/97550	1.000
Ch14 Mouse GSM Low	130.8 to 130.8	123.43	0.25/3.65	54.4	157.5	97538/97550	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97550/97550	1.000
February 1 to 28, 2014							
Ch11 Mouse GSM High	275.0 to 276.1	285.13	0.43/14.31	235.0	380.5	88182/88184	1.000
Ch12 Mouse GSM Med	185.8 to 186.6	182.81	0.38/8.21	125.8	214.8	88086/88184	0.999
Ch14 Mouse GSM Low	130.8 to 131.9	123.35	0.24/3.44	103.5	152.6	88180/88184	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	88184/88184	1.000

TABLE I3
Summary of GSM-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch11 Mouse GSM High	275.0 to 276.1	261.60	0.57/17.65	199.8	325.6	96810/97142	0.997
Ch12 Mouse GSM Med	186.6 to 186.6	178.89	0.67/14.44	138.6	218.0	96626/97142	0.995
Ch14 Mouse GSM Low	131.4 to 131.9	124.02	0.24/3.40	105.7	138.7	97142/97142	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97142/97142	1.000
April 1 to 30, 2014							
Ch11 Mouse GSM High	273.8 to 275.0	264.65	0.57/18.01	203.2	327.8	94118/94548	0.995
Ch12 Mouse GSM Med	185.8 to 186.6	182.58	0.71/15.58	129.0	246.4	93222/94548	0.986
Ch14 Mouse GSM Low	130.8 to 131.4	122.29	0.23/3.35	106.3	141.5	94548/94548	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94548/94548	1.000
May 1 to 31, 2014							
Ch11 Mouse GSM High	272.7 to 273.8	268.70	0.37/11.81	201.7	319.0	97086/97244	0.998
Ch12 Mouse GSM Med	185.0 to 185.8	184.59	0.34/7.30	131.9	223.2	97154/97244	0.999
Ch14 Mouse GSM Low	130.8 to 130.8	122.38	0.23/3.30	108.2	136.7	97244/97244	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97244/97244	1.000
June 1 to 30, 2014							
Ch11 Mouse GSM High	271.5 to 272.7	275.38	0.41/13.20	222.4	327.6	94288/94288	1.000
Ch12 Mouse GSM Med	184.2 to 185.0	180.65	0.26/5.58	141.8	202.2	94282/94288	1.000
Ch14 Mouse GSM Low	130.3 to 130.8	123.72	0.23/3.37	110.5	137.5	94288/94288	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94288/94288	1.000
July 1 to 9, 2014							
Ch11 Mouse GSM High	271.5 to 271.5	281.98	0.27/8.95	242.7	312.4	25064/25064	1.000
Ch12 Mouse GSM Med	184.2 to 184.2	185.84	0.22/4.78	169.6	201.3	25064/25064	1.000
Ch14 Mouse GSM Low	130.3 to 130.3	125.14	0.23/3.34	113.1	138.1	25064/25064	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch11 Mouse GSM High	194.1 to 276.1	259.29	0.88/27.75	134.8	380.5	2265600/2272442	0.997
Ch12 Mouse GSM Med	137.2 to 186.6	179.13	0.47/9.94	93.1	246.4	2270352/2272416	0.999
Ch14 Mouse GSM Low	97.0 to 131.9	118.53	0.26/3.55	54.4	157.5	2272352/2272416	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2272416/2272416	1.000

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data – H-Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch11 Mouse GSM High	0.52 to 0.53	0.462	0.26/0.014	0.29	0.67	38922/38950	0.999
Ch12 Mouse GSM Med	0.36 to 0.38	0.347	0.27/0.011	0.23	0.53	38944/38950	1.000
Ch14 Mouse GSM Low	0.26 to 0.27	0.275	0.29/0.009	0.25	0.31	38950/38950	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	38950/38950	1.000
July 1 to 31, 2012							
Ch11 Mouse GSM High	0.53 to 0.56	0.476	0.33/0.018	0.40	0.57	96830/97462	0.994
Ch12 Mouse GSM Med	0.38 to 0.40	0.363	0.25/0.011	0.30	0.42	97458/97462	1.000
Ch14 Mouse GSM Low	0.27 to 0.28	0.292	0.27/0.009	0.26	0.33	97462/97462	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97462/97462	1.000
August 1 to 31, 2012							
Ch11 Mouse GSM High	0.56 to 0.59	0.485	0.29/0.017	0.40	0.56	93530/94976	0.985
Ch12 Mouse GSM Med	0.40 to 0.42	0.381	0.25/0.011	0.33	0.45	94976/94976	1.000
Ch14 Mouse GSM Low	0.28 to 0.30	0.306	0.26/0.009	0.27	0.37	94976/94976	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94976/94976	1.000
September 1 to 30, 2012							
Ch11 Mouse GSM High	0.59 to 0.61	0.492	0.26/0.015	0.35	0.55	88558/94374	0.938
Ch12 Mouse GSM Med	0.42 to 0.43	0.397	0.25/0.012	0.30	0.45	94362/94370	1.000
Ch14 Mouse GSM Low	0.30 to 0.30	0.316	0.26/0.009	0.25	0.36	94370/94370	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94370/94370	1.000
October 1 to 31, 2012							
Ch11 Mouse GSM High	0.61 to 0.63	0.515	0.25/0.015	0.29	0.58	95516/97604	0.979
Ch12 Mouse GSM Med	0.43 to 0.45	0.419	0.25/0.012	0.34	0.49	97602/97602	1.000
Ch14 Mouse GSM Low	0.30 to 0.31	0.324	0.26/0.010	0.28	0.36	97602/97602	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97602/97602	1.000
November 1 to 30, 2012							
Ch11 Mouse GSM High	0.63 to 0.64	0.529	0.25/0.015	0.47	0.61	91670/94628	0.969
Ch12 Mouse GSM Med	0.45 to 0.46	0.439	0.24/0.012	0.37	0.49	94628/94628	1.000
Ch14 Mouse GSM Low	0.31 to 0.32	0.335	0.26/0.010	0.29	0.38	94628/94628	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94628/94628	1.000
December 1 to 31, 2012							
Ch11 Mouse GSM High	0.64 to 0.66	0.532	0.24/0.015	0.47	0.60	90788/97500	0.931
Ch12 Mouse GSM Med	0.46 to 0.46	0.445	0.23/0.012	0.40	0.50	97496/97496	1.000
Ch14 Mouse GSM Low	0.32 to 0.33	0.339	0.25/0.010	0.29	0.38	97496/97496	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch11=Chamber 11)

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch11 Mouse GSM High	0.66 to 0.66	0.550	0.32/0.021	0.46	0.63	91600/97378	0.941
Ch12 Mouse GSM Med	0.46 to 0.47	0.450	0.28/0.015	0.35	0.54	97286/97364	0.999
Ch14 Mouse GSM Low	0.33 to 0.33	0.343	0.25/0.010	0.31	0.39	97364/97364	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97364/97364	1.000
February 1 to 28, 2013							
Ch11 Mouse GSM High	0.66 to 0.67	0.554	0.23/0.015	0.49	0.62	86086/88116	0.977
Ch12 Mouse GSM Med	0.47 to 0.47	0.450	0.22/0.012	0.40	0.50	88116/88116	1.000
Ch14 Mouse GSM Low	0.33 to 0.33	0.349	0.24/0.010	0.29	0.39	88116/88116	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	88116/88116	1.000
March 1 to 31, 2013							
Ch11 Mouse GSM High	0.67 to 0.67	0.559	0.25/0.016	0.49	0.63	94850/97784	0.970
Ch12 Mouse GSM Med	0.47 to 0.48	0.456	0.25/0.013	0.40	0.52	97784/97784	1.000
Ch14 Mouse GSM Low	0.33 to 0.34	0.348	0.24/0.010	0.31	0.39	97784/97784	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97784/97784	1.000
April 1 to 30, 2013							
Ch11 Mouse GSM High	0.67 to 0.68	0.553	0.21/0.014	0.49	0.62	91198/96260	0.947
Ch12 Mouse GSM Med	0.48 to 0.48	0.458	0.22/0.012	0.40	0.50	96260/96260	1.000
Ch14 Mouse GSM Low	0.34 to 0.34	0.356	0.26/0.011	0.31	0.39	96260/96260	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	96260/96260	1.000
May 1 to 31, 2013							
Ch11 Mouse GSM High	0.68 to 0.68	0.558	0.22/0.014	0.45	0.62	92216/97020	0.950
Ch12 Mouse GSM Med	0.48 to 0.48	0.476	0.33/0.018	0.37	0.58	97018/97020	1.000
Ch14 Mouse GSM Low	0.34 to 0.34	0.350	0.25/0.010	0.31	0.39	97020/97020	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97020/97020	1.000
June 1 to 30, 2013							
Ch11 Mouse GSM High	0.68 to 0.69	0.546	0.23/0.015	0.48	0.60	81918/94496	0.867
Ch12 Mouse GSM Med	0.48 to 0.49	0.492	0.25/0.014	0.45	0.56	94496/94496	1.000
Ch14 Mouse GSM Low	0.34 to 0.34	0.359	0.24/0.010	0.32	0.39	94496/94496	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94496/94496	1.000
July 1 to 31, 2013							
Ch11 Mouse GSM High	0.69 to 0.69	0.562	0.25/0.016	0.45	0.63	92320/99146	0.931
Ch12 Mouse GSM Med	0.49 to 0.49	0.498	0.25/0.014	0.41	0.55	99146/99146	1.000
Ch14 Mouse GSM Low	0.34 to 0.35	0.362	0.26/0.011	0.32	0.40	99146/99146	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	99146/99146	1.000

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch11 Mouse GSM High	0.69 to 0.69	0.632	0.45/0.034	0.51	0.78	101140/101700	0.994
Ch12 Mouse GSM Med	0.49 to 0.49	0.502	0.27/0.016	0.44	0.58	101700/101700	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.362	0.26/0.011	0.32	0.43	101700/101700	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	101700/101700	1.000
September 1 to 30, 2013							
Ch11 Mouse GSM High	0.69 to 0.73	0.709	0.53/0.044	0.39	0.88	93920/93922	1.000
Ch12 Mouse GSM Med	0.49 to 0.50	0.506	0.27/0.016	0.46	0.58	93920/93920	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.365	0.25/0.011	0.27	0.40	93914/93920	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	93920/93920	1.000
October 1 to 31, 2013							
Ch11 Mouse GSM High	0.73 to 0.73	0.748	0.38/0.033	0.61	0.89	100816/100816	1.000
Ch12 Mouse GSM Med	0.50 to 0.50	0.506	0.27/0.016	0.41	0.59	100816/100816	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.368	0.26/0.011	0.29	0.43	100816/100816	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100816/100816	1.000
November 1 to 30, 2013							
Ch11 Mouse GSM High	0.73 to 0.73	0.679	0.44/0.036	0.49	0.83	93208/93226	1.000
Ch12 Mouse GSM Med	0.50 to 0.50	0.496	0.29/0.017	0.40	0.60	93226/93226	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.361	0.28/0.012	0.28	0.41	93226/93226	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	93226/93226	1.000
December 1 to 31, 2013							
Ch11 Mouse GSM High	0.73 to 0.73	0.699	0.48/0.040	0.56	0.82	96800/96846	1.000
Ch12 Mouse GSM Med	0.49 to 0.50	0.502	0.31/0.018	0.41	0.62	96846/96846	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.369	0.26/0.011	0.28	0.48	96844/96846	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	96846/96846	1.000
January 1 to 31, 2014							
Ch11 Mouse GSM High	0.73 to 0.73	0.691	0.32/0.026	0.55	0.82	97548/97550	1.000
Ch12 Mouse GSM Med	0.49 to 0.49	0.505	0.27/0.016	0.41	0.58	97550/97550	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.366	0.26/0.011	0.16	0.44	97538/97550	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97550/97550	1.000
February 1 to 28, 2014							
Ch11 Mouse GSM High	0.73 to 0.73	0.704	0.35/0.029	0.59	0.82	88184/88184	1.000
Ch12 Mouse GSM Med	0.49 to 0.50	0.461	0.68/0.038	0.32	0.57	87662/88184	0.994
Ch14 Mouse GSM Low	0.35 to 0.35	0.372	0.25/0.011	0.29	0.46	88176/88184	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	88184/88184	1.000

TABLE I4
Summary of GSM-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch11 Mouse GSM High	0.73 to 0.73	0.718	0.48/0.041	0.59	0.88	97142/97142	1.000
Ch12 Mouse GSM Med	0.50 to 0.50	0.466	0.74/0.041	0.33	0.59	93996/97142	0.968
Ch14 Mouse GSM Low	0.35 to 0.35	0.370	0.23/0.010	0.33	0.41	97142/97142	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97142/97142	1.000
April 1 to 30, 2014							
Ch11 Mouse GSM High	0.73 to 0.73	0.737	0.46/0.040	0.60	0.88	94548/94548	1.000
Ch12 Mouse GSM Med	0.49 to 0.50	0.492	0.81/0.048	0.34	0.68	92916/94548	0.983
Ch14 Mouse GSM Low	0.35 to 0.35	0.372	0.24/0.010	0.32	0.41	94548/94548	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94548/94548	1.000
May 1 to 31, 2014							
Ch11 Mouse GSM High	0.72 to 0.73	0.739	0.34/0.030	0.60	0.85	97244/97244	1.000
Ch12 Mouse GSM Med	0.49 to 0.49	0.495	0.33/0.019	0.36	0.61	97052/97244	0.998
Ch14 Mouse GSM Low	0.35 to 0.35	0.370	0.22/0.010	0.34	0.41	97244/97244	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97244/97244	1.000
June 1 to 30, 2014							
Ch11 Mouse GSM High	0.72 to 0.72	0.716	0.36/0.031	0.63	0.84	94288/94288	1.000
Ch12 Mouse GSM Med	0.49 to 0.49	0.501	0.25/0.015	0.38	0.55	94284/94288	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.366	0.23/0.010	0.33	0.40	94288/94288	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94288/94288	1.000
July 1 to 9, 2014							
Ch11 Mouse GSM High	0.72 to 0.72	0.698	0.26/0.021	0.63	0.78	25064/25064	1.000
Ch12 Mouse GSM Med	0.49 to 0.49	0.487	0.23/0.013	0.45	0.54	25064/25064	1.000
Ch14 Mouse GSM Low	0.35 to 0.35	0.361	0.26/0.011	0.32	0.41	25064/25064	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch11 Mouse GSM High	0.52 to 0.73	0.607	0.73/0.053	0.29	0.89	2212122/2272442	0.973
Ch12 Mouse GSM Med	0.36 to 0.50	0.463	0.46/0.025	0.23	0.68	2266828/2272416	0.998
Ch14 Mouse GSM Low	0.26 to 0.35	0.349	0.26/0.011	0.16	0.48	2272388/2272416	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2272416/2272416	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – SAR[a]

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
June 18 to 30, 2012								
Ch01 Mouse IS95 High	18.9 to 20.2	10.00	9.98	0.13/0.03	5.483	20.437	19472/19475	1.000
Ch02 Mouse IS95 Med	18.9 to 20.2	5.00	4.94	0.17/0.04	3.641	7.729	19475/19475	1.000
Ch03 Mouse IS95 Low	18.9 to 20.1	2.50	2.49	0.11/0.03	2.134	3.273	19475/19475	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	19475/19475	1.000
July 1 to 31, 2012								
Ch01 Mouse IS95 High	20.2 to 24.6	10.00	9.98	0.12/0.03	6.703	16.201	48730/48731	1.000
Ch02 Mouse IS95 Med	20.2 to 24.6	5.00	4.96	0.15/0.04	3.476	8.609	48730/48731	1.000
Ch03 Mouse IS95 Low	20.1 to 24.7	2.50	2.49	0.11/0.03	1.742	4.180	48730/48731	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48731/48731	1.000
August 1 to 31, 2012								
Ch01 Mouse IS95 High	24.6 to 28.0	10.00	9.98	0.12/0.03	8.349	13.937	47488/47488	1.000
Ch02 Mouse IS95 Med	24.6 to 27.6	5.00	4.96	0.14/0.03	4.115	9.356	47487/47488	1.000
Ch03 Mouse IS95 Low	24.7 to 27.6	2.50	2.49	0.11/0.03	2.031	3.689	47488/47488	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47488/47488	1.000
September 1 to 30, 2012								
Ch01 Mouse IS95 High	28.0 to 30.1	10.00	9.98	0.11/0.03	8.923	11.261	47187/47187	1.000
Ch02 Mouse IS95 Med	27.6 to 29.3	5.00	4.96	0.13/0.03	4.325	6.682	47187/47187	1.000
Ch03 Mouse IS95 Low	27.6 to 29.1	2.50	2.49	0.11/0.03	2.180	2.844	47187/47187	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47185/47185	1.000
October 1 to 31, 2012								
Ch01 Mouse IS95 High	30.1 to 33.3	10.00	9.97	0.11/0.03	8.718	11.420	48802/48802	1.000
Ch02 Mouse IS95 Med	29.3 to 32.6	5.00	4.98	0.13/0.03	4.296	5.798	48802/48802	1.000
Ch03 Mouse IS95 Low	29.1 to 32.4	2.50	2.49	0.10/0.02	2.208	2.766	48802/48802	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48801/48801	1.000
November 1 to 30, 2012								
Ch01 Mouse IS95 High	33.3 to 36.7	10.00	9.98	0.11/0.03	4.470	11.321	47313/47314	1.000
Ch02 Mouse IS95 Med	32.6 to 36.1	5.00	4.98	0.13/0.03	2.462	5.974	47313/47314	1.000
Ch03 Mouse IS95 Low	32.4 to 35.5	2.50	2.49	0.11/0.03	1.174	2.967	47313/47314	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47314/47314	1.000
December 1 to 31, 2012								
Ch01 Mouse IS95 High	36.7 to 39.1	10.00	10.01	0.11/0.03	8.925	11.723	48750/48750	1.000
Ch02 Mouse IS95 Med	36.1 to 38.5	5.00	4.98	0.13/0.03	4.364	6.018	48750/48750	1.000
Ch03 Mouse IS95 Low	35.5 to 38.2	2.50	2.50	0.11/0.03	2.227	2.909	48750/48750	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48748/48748	1.000

[a] Ch=chamber (*e.g.*, Ch01=Chamber 1)

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
January 1 to 31, 2013								
Ch01 Mouse IS95 High	39.1 to 41.1	10.00	9.98	0.12/0.03	8.549	11.885	48689/48689	1.000
Ch02 Mouse IS95 Med	38.5 to 40.8	5.00	4.98	0.13/0.03	4.218	5.822	48689/48689	1.000
Ch03 Mouse IS95 Low	38.2 to 40.8	2.50	2.50	0.11/0.03	2.161	2.957	48689/48689	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48682/48682	1.000
February 1 to 28, 2013								
Ch01 Mouse IS95 High	41.1 to 43.1	10.00	9.98	0.12/0.03	8.591	11.811	44058/44058	1.000
Ch02 Mouse IS95 Med	40.8 to 42.7	5.00	4.99	0.14/0.03	3.890	7.124	44058/44058	1.000
Ch03 Mouse IS95 Low	40.8 to 42.6	2.50	2.50	0.11/0.03	2.117	3.036	44058/44058	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	44058/44058	1.000
March 1 to 31, 2013								
Ch01 Mouse IS95 High	43.1 to 44.9	10.00	10.01	0.12/0.03	8.731	11.338	48892/48892	1.000
Ch02 Mouse IS95 Med	42.7 to 44.3	5.00	5.00	0.14/0.03	4.271	6.209	48892/48892	1.000
Ch03 Mouse IS95 Low	42.6 to 44.0	2.50	2.50	0.11/0.03	2.213	2.833	48892/48892	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48892/48892	1.000
April 1 to 30, 2013								
Ch01 Mouse IS95 High	44.9 to 47.8	10.00	10.00	0.12/0.03	8.512	12.723	48130/48130	1.000
Ch02 Mouse IS95 Med	44.3 to 47.5	5.00	4.98	0.14/0.03	4.329	5.899	48130/48130	1.000
Ch03 Mouse IS95 Low	44.0 to 47.2	2.50	2.49	0.12/0.03	2.196	2.858	48130/48130	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48130/48130	1.000
May 1 to 31, 2013								
Ch01 Mouse IS95 High	47.8 to 49.2	10.00	9.99	0.14/0.03	7.772	12.345	48510/48510	1.000
Ch02 Mouse IS95 Med	47.5 to 49.1	5.00	4.97	0.14/0.03	4.225	6.364	48510/48510	1.000
Ch03 Mouse IS95 Low	47.2 to 48.5	2.50	2.49	0.12/0.03	2.170	3.006	48510/48510	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48510/48510	1.000
June 1 to 30, 2013								
Ch01 Mouse IS95 High	49.2 to 50.4	10.00	10.02	0.15/0.03	7.714	12.935	47257/47257	1.000
Ch02 Mouse IS95 Med	49.1 to 50.4	5.00	4.99	0.14/0.03	4.037	6.047	47257/47257	1.000
Ch03 Mouse IS95 Low	48.5 to 50.2	2.50	2.50	0.25/0.06	1.455	3.737	47253/47257	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47248/47248	1.000
July 1 to 31, 2013								
Ch01 Mouse IS95 High	50.4 to 51.6	10.00	9.99	0.16/0.04	7.808	13.620	49573/49573	1.000
Ch02 Mouse IS95 Med	50.4 to 51.7	5.00	4.97	0.14/0.03	4.158	6.040	49573/49573	1.000
Ch03 Mouse IS95 Low	50.2 to 51.4	2.50	2.50	0.28/0.07	1.626	3.683	49573/49573	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	49573/49573	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
August 1 to 31, 2013								
Ch01 Mouse IS95 High	51.6 to 52.2	10.00	9.99	0.14/0.03	7.168	12.173	50856/50856	1.000
Ch02 Mouse IS95 Med	51.7 to 52.7	5.00	4.99	0.16/0.04	4.279	6.445	50856/50856	1.000
Ch03 Mouse IS95 Low	51.3 to 52.2	2.50	2.50	0.14/0.03	1.851	3.071	50856/50856	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50850/50850	1.000
September 1 to 30, 2013								
Ch01 Mouse IS95 High	52.2 to 53.0	10.95	10.11	0.14/0.03	8.045	13.922	46961/46961	1.000
Ch02 Mouse IS95 Med	52.7 to 53.5	5.00	5.00	0.15/0.03	4.143	6.336	46961/46961	1.000
Ch03 Mouse IS95 Low	52.2 to 53.4	2.50	2.51	0.35/0.08	1.262	4.130	46946/46961	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	46960/46960	1.000
October 1 to 31, 2013								
Ch01 Mouse IS95 High	53.0 to 53.8	10.00	10.03	0.13/0.03	8.027	12.009	50408/50408	1.000
Ch02 Mouse IS95 Med	53.5 to 54.2	5.00	4.99	0.15/0.03	4.224	6.669	50408/50408	1.000
Ch03 Mouse IS95 Low	53.4 to 54.1	2.50	2.50	0.16/0.04	1.718	3.359	50408/50408	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50408/50408	1.000
November 1 to 30, 2013								
Ch01 Mouse IS95 High	53.8 to 54.0	10.00	10.03	0.15/0.04	4.536	12.268	46637/46639	1.000
Ch02 Mouse IS95 Med	54.2 to 54.2	5.00	4.98	0.16/0.04	2.225	6.187	46637/46639	1.000
Ch03 Mouse IS95 Low	53.8 to 54.1	2.50	2.48	0.27/0.06	0.161	4.469	46551/46639	0.998
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	46613/46613	1.000
December 1 to 31, 2013								
Ch01 Mouse IS95 High	52.3 to 54.0	10.00	10.02	0.14/0.03	7.143	14.712	48423/48423	1.000
Ch02 Mouse IS95 Med	52.8 to 54.2	5.00	4.99	0.16/0.04	3.949	7.068	48423/48423	1.000
Ch03 Mouse IS95 Low	52.6 to 53.8	2.50	2.50	0.14/0.03	1.989	3.035	48423/48423	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48423/48423	1.000
January 1 to 31, 2014								
Ch01 Mouse IS95 High	52.3 to 52.7	10.00	9.99	0.14/0.03	6.121	14.242	48776/48777	1.000
Ch02 Mouse IS95 Med	52.8 to 53.4	5.00	5.00	0.15/0.04	1.378	6.481	48775/48777	1.000
Ch03 Mouse IS95 Low	52.6 to 53.0	2.50	2.51	0.31/0.07	0.429	4.110	48768/48776	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48775/48775	1.000
February 1 to 28, 2014								
Ch01 Mouse IS95 High	52.7 to 54.1	10.00	9.95	0.13/0.03	6.403	13.519	44092/44092	1.000
Ch02 Mouse IS95 Med	53.4 to 54.8	5.00	4.98	0.16/0.04	2.326	9.889	44085/44092	1.000
Ch03 Mouse IS95 Low	53.0 to 54.6	2.50	2.48	0.35/0.08	1.358	3.784	44059/44092	0.999
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	44092/44092	1.000

TABLE I5
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/ Total	Ratio
March 1 to 31, 2014								
Ch01 Mouse IS95 High	53.0 to 54.1	10.00	9.45	0.48/0.12	0.124	18.080	48470/48590	0.998
Ch02 Mouse IS95 Med	53.8 to 54.9	5.00	5.00	0.16/0.04	2.146	9.590	48585/48590	1.000
Ch03 Mouse IS95 Low	54.2 to 54.7	2.50	2.47	0.35/0.08	0.607	6.319	48505/48590	0.998
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48571/48571	1.000
April 1 to 30, 2014								
Ch01 Mouse IS95 High	51.8 to 52.8	10.00	9.96	0.21/0.05	0.435	21.444	47241/47275	0.999
Ch02 Mouse IS95 Med	52.9 to 53.2	5.00	5.02	0.21/0.05	0.771	12.768	47238/47275	0.999
Ch03 Mouse IS95 Low	53.3 to 53.8	2.50	2.48	0.27/0.06	0.656	3.495	47261/47275	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47274/47274	1.000
May 1 to 31, 2014								
Ch01 Mouse IS95 High	51.0 to 51.8	10.00	9.98	0.14/0.03	8.159	12.373	48622/48622	1.000
Ch02 Mouse IS95 Med	51.4 to 52.9	5.00	5.00	0.14/0.03	3.701	6.542	48622/48622	1.000
Ch03 Mouse IS95 Low	52.2 to 53.3	2.50	2.49	0.27/0.06	1.281	3.566	48619/48622	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	48622/48622	1.000
June 1 to 30, 2014								
Ch01 Mouse IS95 High	50.5 to 51.2	10.00	9.97	0.13/0.03	7.103	12.077	47144/47144	1.000
Ch02 Mouse IS95 Med	51.0 to 51.4	5.00	5.00	0.14/0.03	3.476	6.143	47144/47144	1.000
Ch03 Mouse IS95 Low	50.6 to 52.2	2.50	2.49	0.28/0.07	1.038	4.032	47132/47144	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	47144/47144	1.000
July 1 to 9, 2014								
Ch01 Mouse IS95 High	51.2 to 51.2	10.00	10.00	0.11/0.03	8.426	11.320	12549/12549	1.000
Ch02 Mouse IS95 Med	51.3 to 51.3	5.00	4.99	0.12/0.03	4.148	5.780	12549/12549	1.000
Ch03 Mouse IS95 Low	50.6 to 50.6	2.50	2.53	0.65/0.16	0.633	4.854	12390/12549	0.987
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	12532/12532	1.000
June 18, 2012, to July 9, 2014								
Ch01 Mouse IS95 High	18.9 to 54.1	10.95	9.97	0.17/0.04	0.124	21.444	1136122/1136284	1.000
Ch02 Mouse IS95 Med	18.9 to 54.9	5.00	4.99	0.15/0.03	0.771	12.768	1136228/1136284	1.000
Ch03 Mouse IS95 Low	18.9 to 54.7	2.50	2.49	0.21/0.05	0.161	6.319	1136030/1136283	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1136208/1136208	1.000

TABLE I6
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – Chamber Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch01 Mouse IS95 High	194.10 to 199.60	196.37	0.13/2.96	143.58	277.18	38944/38950	1.000
Ch02 Mouse IS95 Med	137.20 to 141.20	138.19	0.17/2.76	120.44	170.46	38950/38950	1.000
Ch03 Mouse IS95 Low	97.00 to 99.80	98.08	0.12/1.31	91.19	110.93	38950/38950	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	38950/38950	1.000
July 1 to 31, 2012							
Ch01 Mouse IS95 High	199.60 to 212.30	205.11	0.12/2.82	173.76	270.15	97460/97462	1.000
Ch02 Mouse IS95 Med	141.20 to 150.10	145.03	0.15/2.59	125.14	196.92	97460/97462	1.000
Ch03 Mouse IS95 Low	99.80 to 106.10	103.17	0.11/1.37	88.58	137.22	97460/97462	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97462/97462	1.000
August 1 to 31, 2012							
Ch01 Mouse IS95 High	212.30 to 222.20	217.26	0.12/2.98	199.87	258.23	94976/94976	1.000
Ch02 Mouse IS95 Med	150.10 to 157.10	152.68	0.15/2.58	138.34	208.61	94974/94976	1.000
Ch03 Mouse IS95 Low	106.10 to 111.10	108.44	0.11/1.37	98.59	132.85	94976/94976	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94976/94976	1.000
September 1 to 30, 2012							
Ch01 Mouse IS95 High	222.20 to 230.90	227.67	0.11/2.97	212.83	244.74	94374/94374	1.000
Ch02 Mouse IS95 Med	157.10 to 161.40	159.33	0.13/2.37	148.22	184.17	94374/94374	1.000
Ch03 Mouse IS95 Low	111.10 to 114.10	112.90	0.11/1.46	104.64	121.19	94374/94374	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94370/94370	1.000
October 1 to 31, 2012							
Ch01 Mouse IS95 High	230.90 to 237.60	233.91	0.11/3.07	215.34	250.45	97604/97604	1.000
Ch02 Mouse IS95 Med	161.40 to 166.60	163.67	0.13/2.46	151.48	176.83	97604/97604	1.000
Ch03 Mouse IS95 Low	114.10 to 117.80	115.72	0.11/1.42	108.66	123.97	97604/97604	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97602/97602	1.000
November 1 to 30, 2012							
Ch01 Mouse IS95 High	237.60 to 242.70	240.37	0.11/3.13	158.92	257.10	94626/94628	1.000
Ch02 Mouse IS95 Med	166.60 to 171.70	169.18	0.13/2.58	116.96	185.43	94626/94628	1.000
Ch03 Mouse IS95 Low	117.80 to 120.60	119.29	0.12/1.60	80.77	131.35	94626/94628	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94628/94628	1.000
December 1 to 31, 2012							
Ch01 Mouse IS95 High	242.70 to 247.20	245.58	0.11/3.21	231.33	264.34	97500/97500	1.000
Ch02 Mouse IS95 Med	171.70 to 173.80	172.59	0.13/2.64	162.14	190.41	97500/97500	1.000
Ch03 Mouse IS95 Low	120.60 to 122.90	121.97	0.11/1.55	113.87	132.39	97500/97500	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch01=Chamber 1)

TABLE I6
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch01 Mouse IS95 High	247.20 to 249.80	249.07	0.12/3.50	230.99	272.55	97378/97378	1.000
Ch02 Mouse IS95 Med	173.80 to 175.70	174.88	0.13/2.63	161.36	189.41	97378/97378	1.000
Ch03 Mouse IS95 Low	122.90 to 124.30	123.76	0.11/1.65	115.26	135.11	97378/97378	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97364/97364	1.000
February 1 to 28, 2013							
Ch01 Mouse IS95 High	249.80 to 252.40	251.73	0.12/3.39	233.92	274.28	88116/88116	1.000
Ch02 Mouse IS95 Med	175.70 to 177.60	176.86	0.14/2.85	156.42	211.68	88116/88116	1.000
Ch03 Mouse IS95 Low	124.30 to 125.60	125.26	0.12/1.67	115.38	138.19	88116/88116	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	88116/88116	1.000
March 1 to 31, 2013							
Ch01 Mouse IS95 High	252.40 to 253.60	253.18	0.12/3.50	236.57	269.59	97784/97784	1.000
Ch02 Mouse IS95 Med	177.60 to 179.30	178.79	0.14/2.88	165.47	199.50	97784/97784	1.000
Ch03 Mouse IS95 Low	125.60 to 126.20	125.98	0.11/1.61	118.72	133.49	97784/97784	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97784/97784	1.000
April 1 to 30, 2013							
Ch01 Mouse IS95 High	253.60 to 257.20	255.59	0.12/3.58	235.87	288.37	96260/96260	1.000
Ch02 Mouse IS95 Med	179.30 to 181.80	179.86	0.14/2.87	167.66	195.71	96260/96260	1.000
Ch03 Mouse IS95 Low	126.20 to 128.60	127.23	0.12/1.72	119.08	136.24	96260/96260	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	96260/96260	1.000
May 1 to 31, 2013							
Ch01 Mouse IS95 High	257.20 to 259.40	257.37	0.14/4.29	226.87	285.93	97020/97020	1.000
Ch02 Mouse IS95 Med	181.80 to 183.40	181.64	0.14/2.98	167.27	205.30	97020/97020	1.000
Ch03 Mouse IS95 Low	128.60 to 129.10	128.55	0.12/1.77	119.88	141.09	97020/97020	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97020/97020	1.000
June 1 to 30, 2013							
Ch01 Mouse IS95 High	259.40 to 260.60	259.64	0.15/4.39	227.53	294.63	94514/94514	1.000
Ch02 Mouse IS95 Med	183.40 to 184.20	183.31	0.14/3.05	164.59	201.45	94514/94514	1.000
Ch03 Mouse IS95 Low	129.10 to 130.30	129.22	0.25/3.81	98.48	159.44	94506/94514	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94496/94496	1.000
July 1 to 31, 2013							
Ch01 Mouse IS95 High	260.60 to 261.60	260.92	0.16/4.87	230.46	304.39	99146/99146	1.000
Ch02 Mouse IS95 Med	184.20 to 185.00	184.01	0.14/3.03	168.19	202.70	99146/99146	1.000
Ch03 Mouse IS95 Low	130.30 to 130.80	130.53	0.28/4.24	105.17	158.30	99146/99146	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	99146/99146	1.000

TABLE I6
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch01 Mouse IS95 High	261.60 to 262.80	261.92	0.14/4.30	222.33	288.89	101712/101712	1.000
Ch02 Mouse IS95 Med	185.00 to 185.80	185.08	0.16/3.35	171.22	210.82	101712/101712	1.000
Ch03 Mouse IS95 Low	130.80 to 131.40	130.89	0.14/2.08	113.00	145.03	101712/101712	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	101700/101700	1.000
September 1 to 30, 2013							
Ch01 Mouse IS95 High	262.80 to 275.00	264.04	0.14/4.41	235.55	309.86	93922/93922	1.000
Ch02 Mouse IS95 Med	185.80 to 186.60	186.05	0.15/3.20	169.04	209.04	93922/93922	1.000
Ch03 Mouse IS95 Low	131.40 to 131.90	131.72	0.35/5.41	93.28	169.36	93890/93922	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	93920/93920	1.000
October 1 to 31, 2013							
Ch01 Mouse IS95 High	262.80 to 263.90	263.44	0.13/4.00	236.10	288.79	100816/100816	1.000
Ch02 Mouse IS95 Med	186.60 to 187.40	186.81	0.15/3.17	171.27	216.71	100816/100816	1.000
Ch03 Mouse IS95 Low	131.90 to 132.50	132.12	0.17/2.54	109.99	152.72	100816/100816	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	100816/100816	1.000
November 1 to 30, 2013							
Ch01 Mouse IS95 High	263.90 to 263.90	263.87	0.15/4.69	177.48	291.88	93274/93278	1.000
Ch02 Mouse IS95 Med	187.40 to 187.40	187.21	0.16/3.50	125.18	208.73	93274/93278	1.000
Ch03 Mouse IS95 Low	131.90 to 132.50	131.56	0.33/5.16	33.46	176.17	93098/93278	0.998
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	93226/93226	1.000
December 1 to 31, 2013							
Ch01 Mouse IS95 High	262.80 to 263.90	263.08	0.14/4.35	221.94	318.53	96846/96846	1.000
Ch02 Mouse IS95 Med	185.80 to 187.40	186.01	0.16/3.57	166.56	220.78	96846/96846	1.000
Ch03 Mouse IS95 Low	131.40 to 131.90	131.34	0.14/2.10	117.52	144.93	96846/96846	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	96846/96846	1.000
January 1 to 31, 2014							
Ch01 Mouse IS95 High	262.80 to 262.80	262.41	0.14/4.18	205.46	313.40	97552/97554	1.000
Ch02 Mouse IS95 Med	185.80 to 186.60	186.14	0.16/3.37	97.83	212.15	97550/97554	1.000
Ch03 Mouse IS95 Low	131.40 to 131.40	131.41	0.31/4.82	54.39	168.36	97536/97552	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97550/97550	1.000
February 1 to 28, 2014							
Ch01 Mouse IS95 High	262.80 to 265.00	264.44	0.13/4.06	212.35	308.55	88184/88184	1.000
Ch02 Mouse IS95 Med	186.60 to 187.40	187.06	0.16/3.50	127.99	263.90	88170/88184	1.000
Ch03 Mouse IS95 Low	131.40 to 132.50	131.92	0.36/5.64	97.78	163.25	88120/88184	0.999
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	88184/88184	1.000

TABLE I6
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch01 Mouse IS95 High	262.80 to 265.00	255.18	0.64/19.48	22.44	353.11	96940/97180	0.998
Ch02 Mouse IS95 Med	186.60 to 187.40	187.10	0.16/3.42	122.08	258.06	97170/97180	1.000
Ch03 Mouse IS95 Low	132.50 to 132.50	131.79	0.38/5.91	65.38	210.94	97010/97180	0.998
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97142/97142	1.000
April 1 to 30, 2014							
Ch01 Mouse IS95 High	261.60 to 262.80	261.90	0.27/8.39	22.44	384.56	94478/94550	0.999
Ch02 Mouse IS95 Med	185.80 to 186.60	186.68	0.20/4.33	73.16	297.77	94476/94550	0.999
Ch03 Mouse IS95 Low	131.90 to 131.90	131.23	0.28/4.23	67.49	155.79	94522/94550	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94548/94548	1.000
May 1 to 31, 2014							
Ch01 Mouse IS95 High	260.60 to 261.60	261.26	0.14/4.17	236.40	291.11	97244/97244	1.000
Ch02 Mouse IS95 Med	185.00 to 185.80	185.64	0.14/2.95	159.77	212.41	97244/97244	1.000
Ch03 Mouse IS95 Low	131.40 to 131.90	131.27	0.28/4.25	94.32	156.82	97238/97244	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	97244/97244	1.000
June 1 to 30, 2014							
Ch01 Mouse IS95 High	260.60 to 261.60	260.64	0.13/3.91	219.82	287.61	94288/94288	1.000
Ch02 Mouse IS95 Med	184.20 to 185.00	184.79	0.14/2.97	153.76	204.42	94288/94288	1.000
Ch03 Mouse IS95 Low	130.30 to 131.40	130.58	0.28/4.32	84.05	165.61	94264/94288	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	94288/94288	1.000
July 1 to 9, 2014							
Ch01 Mouse IS95 High	261.60 to 261.60	261.65	0.11/3.45	240.24	278.45	25098/25098	1.000
Ch02 Mouse IS95 Med	185.00 to 185.00	184.91	0.12/2.53	168.56	198.96	25098/25098	1.000
Ch03 Mouse IS95 Low	130.30 to 130.30	130.67	0.68/10.57	65.61	181.71	24784/25098	0.987
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch01 Mouse IS95 High	194.10 to 275.00	250.68	0.20/5.84	22.44	384.56	2272240/2272568	1.000
Ch02 Mouse IS95 Med	137.20 to 187.40	177.23	0.15/3.06	73.16	297.77	2272456/2272568	1.000
Ch03 Mouse IS95 Low	97.00 to 132.50	125.25	0.22/3.20	33.46	210.94	2272056/2272566	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.00	–/0.00	0.00	0.00	2272416/2272416	1.000

TABLE I7
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – E-Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch01 Mouse IS95 High	194.1 to 199.6	182.96	0.16/3.46	132.6	264.8	38944/38950	1.000
Ch02 Mouse IS95 Med	137.2 to 141.2	127.98	0.20/2.95	109.8	152.6	38926/38950	0.999
Ch03 Mouse IS95 Low	97.0 to 99.8	94.73	0.17/1.82	86.4	108.0	38950/38950	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	38950/38950	1.000
July 1 to 31, 2012							
Ch01 Mouse IS95 High	199.6 to 212.3	191.13	0.18/3.93	161.2	251.6	97458/97462	1.000
Ch02 Mouse IS95 Med	141.2 to 150.1	134.04	0.20/3.16	115.7	186.0	97458/97462	1.000
Ch03 Mouse IS95 Low	99.8 to 106.1	99.09	0.17/1.98	83.9	131.8	97460/97462	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97462/97462	1.000
August 1 to 31, 2012							
Ch01 Mouse IS95 High	212.3 to 222.2	204.21	0.16/3.81	186.0	241.5	94976/94976	1.000
Ch02 Mouse IS95 Med	150.1 to 157.1	140.96	0.20/3.34	124.8	194.4	94974/94976	1.000
Ch03 Mouse IS95 Low	106.1 to 111.1	104.20	0.18/2.18	94.2	127.8	94976/94976	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94976/94976	1.000
September 1 to 30, 2012							
Ch01 Mouse IS95 High	222.2 to 230.9	214.97	0.17/4.21	191.7	239.8	94374/94374	1.000
Ch02 Mouse IS95 Med	157.1 to 161.4	147.53	0.18/3.03	133.9	170.0	94374/94374	1.000
Ch03 Mouse IS95 Low	111.1 to 114.1	107.71	0.17/2.09	98.9	118.0	94374/94374	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94370/94370	1.000
October 1 to 31, 2012							
Ch01 Mouse IS95 High	230.9 to 237.6	221.21	0.15/3.87	201.8	239.3	97604/97604	1.000
Ch02 Mouse IS95 Med	161.4 to 166.6	149.17	0.17/2.99	134.5	163.2	97604/97604	1.000
Ch03 Mouse IS95 Low	114.1 to 117.8	112.30	0.17/2.27	101.0	125.3	97604/97604	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97602/97602	1.000
November 1 to 30, 2012							
Ch01 Mouse IS95 High	237.6 to 242.7	227.51	0.17/4.54	151.4	254.3	94626/94628	1.000
Ch02 Mouse IS95 Med	166.6 to 171.7	154.82	0.18/3.32	104.7	176.2	94626/94628	1.000
Ch03 Mouse IS95 Low	117.8 to 120.6	116.59	0.17/2.36	76.7	128.5	94626/94628	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94628/94628	1.000
December 1 to 31, 2012							
Ch01 Mouse IS95 High	242.7 to 247.2	233.09	0.17/4.48	213.6	253.7	97500/97500	1.000
Ch02 Mouse IS95 Med	171.7 to 173.8	159.76	0.19/3.47	144.1	176.0	97500/97500	1.000
Ch03 Mouse IS95 Low	120.6 to 122.9	118.37	0.19/2.59	109.0	131.2	97500/97500	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch01=Chamber 1)

TABLE I7
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch01 Mouse IS95 High	247.2 to 249.8	235.20	0.16/4.42	215.1	260.4	97378/97378	1.000
Ch02 Mouse IS95 Med	173.8 to 175.7	162.37	0.18/3.35	148.0	178.4	97378/97378	1.000
Ch03 Mouse IS95 Low	122.9 to 124.3	118.71	0.18/2.51	108.3	130.2	97378/97378	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97364/97364	1.000
February 1 to 28, 2013							
Ch01 Mouse IS95 High	249.8 to 252.4	238.62	0.16/4.53	217.4	264.4	88116/88116	1.000
Ch02 Mouse IS95 Med	175.7 to 177.6	162.29	0.19/3.61	141.3	193.5	88116/88116	1.000
Ch03 Mouse IS95 Low	124.3 to 125.6	119.75	0.19/2.59	109.5	131.5	88116/88116	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	88116/88116	1.000
March 1 to 31, 2013							
Ch01 Mouse IS95 High	252.4 to 253.6	238.57	0.18/5.13	219.6	265.7	97784/97784	1.000
Ch02 Mouse IS95 Med	177.6 to 179.3	164.78	0.19/3.74	149.7	187.8	97784/97784	1.000
Ch03 Mouse IS95 Low	125.6 to 126.2	122.27	0.20/2.80	111.3	136.0	97784/97784	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97784/97784	1.000
April 1 to 30, 2013							
Ch01 Mouse IS95 High	253.6 to 257.2	242.43	0.19/5.50	219.5	273.9	96260/96260	1.000
Ch02 Mouse IS95 Med	179.3 to 181.8	167.11	0.18/3.58	151.5	185.2	96260/96260	1.000
Ch03 Mouse IS95 Low	126.2 to 128.6	123.32	0.18/2.59	113.4	135.4	96260/96260	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	96260/96260	1.000
May 1 to 31, 2013							
Ch01 Mouse IS95 High	257.2 to 259.4	244.96	0.20/5.75	215.4	275.7	97020/97020	1.000
Ch02 Mouse IS95 Med	181.8 to 183.4	168.02	0.18/3.60	150.5	190.8	97020/97020	1.000
Ch03 Mouse IS95 Low	128.6 to 129.1	124.68	0.18/2.57	113.0	137.8	97020/97020	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97020/97020	1.000
June 1 to 30, 2013							
Ch01 Mouse IS95 High	259.4 to 260.6	246.98	0.21/5.93	212.0	288.6	94514/94514	1.000
Ch02 Mouse IS95 Med	183.4 to 184.2	170.19	0.18/3.64	154.4	190.5	94514/94514	1.000
Ch03 Mouse IS95 Low	129.1 to 130.3	128.47	0.64/9.79	88.7	176.8	94186/94514	0.997
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94496/94496	1.000
July 1 to 31, 2013							
Ch01 Mouse IS95 High	260.6 to 261.6	249.22	0.21/6.06	220.7	300.9	99146/99146	1.000
Ch02 Mouse IS95 Med	184.2 to 185.0	170.91	0.19/3.80	154.7	193.1	99146/99146	1.000
Ch03 Mouse IS95 Low	130.3 to 130.8	141.91	0.62/10.43	96.1	177.5	98928/99146	0.998
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	99146/99146	1.000

TABLE I7
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch01 Mouse IS95 High	261.6 to 262.8	249.57	0.20/5.69	215.3	288.2	101712/101712	1.000
Ch02 Mouse IS95 Med	185.0 to 185.8	170.62	0.20/3.99	153.4	196.1	101712/101712	1.000
Ch03 Mouse IS95 Low	130.8 to 131.4	147.36	0.24/4.17	111.4	161.5	101712/101712	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	101700/101700	1.000
September 1 to 30, 2013							
Ch01 Mouse IS95 High	262.8 to 275.0	254.31	0.20/6.01	220.4	300.7	93922/93922	1.000
Ch02 Mouse IS95 Med	185.8 to 186.6	171.99	0.19/3.87	155.0	194.2	93922/93922	1.000
Ch03 Mouse IS95 Low	131.4 to 131.9	136.81	0.82/13.59	85.5	183.6	93028/93922	0.990
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	93920/93920	1.000
October 1 to 31, 2013							
Ch01 Mouse IS95 High	262.8 to 263.9	250.46	0.20/5.73	221.9	282.9	100816/100816	1.000
Ch02 Mouse IS95 Med	186.6 to 187.4	172.10	0.19/3.78	158.0	197.8	100816/100816	1.000
Ch03 Mouse IS95 Low	131.9 to 132.5	140.01	0.32/5.24	95.8	158.3	100766/100816	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100816/100816	1.000
November 1 to 30, 2013							
Ch01 Mouse IS95 High	263.9 to 263.9	254.87	0.21/6.33	172.3	287.3	93272/93278	1.000
Ch02 Mouse IS95 Med	187.4 to 187.4	173.77	0.22/4.48	116.2	196.2	93242/93278	1.000
Ch03 Mouse IS95 Low	131.9 to 132.5	139.81	0.43/7.11	34.8	193.8	92976/93278	0.997
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	93226/93226	1.000
December 1 to 31, 2013							
Ch01 Mouse IS95 High	262.8 to 263.9	264.86	0.26/8.09	222.6	327.2	96846/96846	1.000
Ch02 Mouse IS95 Med	185.8 to 187.4	174.60	0.22/4.51	152.2	209.3	96846/96846	1.000
Ch03 Mouse IS95 Low	131.4 to 131.9	141.81	0.23/3.79	119.4	161.0	96846/96846	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	96846/96846	1.000
January 1 to 31, 2014							
Ch01 Mouse IS95 High	262.8 to 262.8	259.88	0.24/7.35	197.0	322.5	97552/97554	1.000
Ch02 Mouse IS95 Med	185.8 to 186.6	171.64	0.20/4.08	93.5	198.8	97550/97554	1.000
Ch03 Mouse IS95 Low	131.4 to 131.4	138.63	0.57/9.33	56.5	182.3	96868/97552	0.993
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97550/97550	1.000
February 1 to 28, 2014							
Ch01 Mouse IS95 High	262.8 to 265.0	254.65	0.22/6.40	202.8	293.6	88182/88184	1.000
Ch02 Mouse IS95 Med	186.6 to 187.4	172.87	0.22/4.50	121.5	251.8	88162/88184	1.000
Ch03 Mouse IS95 Low	131.4 to 132.5	130.86	0.88/13.88	85.2	176.6	87104/88184	0.988
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	88184/88184	1.000

TABLE I7
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch01 Mouse IS95 High	262.8 to 265.0	248.73	0.66/19.55	21.5	370.2	96900/97180	0.997
Ch02 Mouse IS95 Med	186.6 to 187.4	178.11	0.21/4.32	114.7	249.5	97168/97180	1.000
Ch03 Mouse IS95 Low	132.5 to 132.5	125.83	0.60/8.93	56.2	202.2	96866/97180	0.997
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97142/97142	1.000
April 1 to 30, 2014							
Ch01 Mouse IS95 High	261.6 to 262.8	251.78	0.30/8.89	20.9	380.7	94432/94550	0.999
Ch02 Mouse IS95 Med	185.8 to 186.6	176.58	0.25/5.24	68.9	283.3	94536/94550	1.000
Ch03 Mouse IS95 Low	131.9 to 131.9	132.52	0.51/7.96	68.5	160.3	94454/94550	0.999
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94548/94548	1.000
May 1 to 31, 2014							
Ch01 Mouse IS95 High	260.6 to 261.6	250.61	0.21/6.02	218.6	280.8	97244/97244	1.000
Ch02 Mouse IS95 Med	185.0 to 185.8	174.90	0.18/3.74	145.3	200.0	97242/97244	1.000
Ch03 Mouse IS95 Low	131.4 to 131.9	129.23	0.53/8.16	89.0	165.6	97218/97244	1.000
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	97244/97244	1.000
June 1 to 30, 2014							
Ch01 Mouse IS95 High	260.6 to 261.6	253.68	0.18/5.36	213.1	282.1	94288/94288	1.000
Ch02 Mouse IS95 Med	184.2 to 185.0	177.67	0.23/4.76	153.6	200.6	94288/94288	1.000
Ch03 Mouse IS95 Low	130.3 to 131.4	125.07	0.47/6.93	79.5	174.0	94236/94288	0.999
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	94288/94288	1.000
July 1 to 9, 2014							
Ch01 Mouse IS95 High	261.6 to 261.6	259.20	0.16/4.71	236.0	277.7	25098/25098	1.000
Ch02 Mouse IS95 Med	185.0 to 185.0	184.69	0.16/3.33	168.3	199.6	25098/25098	1.000
Ch03 Mouse IS95 Low	130.3 to 130.3	127.53	0.87/13.46	60.8	192.7	24686/25098	0.984
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch01 Mouse IS95 High	194.1 to 275.0	239.79	0.28/7.76	20.9	380.7	2272148/2272568	1.000
Ch02 Mouse IS95 Med	137.2 to 187.4	164.41	0.22/4.14	68.9	283.3	2272446/2272568	1.000
Ch03 Mouse IS95 Low	97.0 to 132.5	125.83	0.60/9.02	34.8	202.2	2268550/2272566	0.998
Ch13 Mouse Sham	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2272416/2272416	1.000

TABLE I8
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – H-Field[a]

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
June 18 to 30, 2012							
Ch01 Mouse IS95 High	0.52 to 0.53	0.556	0.17/0.011	0.41	0.77	38948/38950	1.000
Ch02 Mouse IS95 Med	0.36 to 0.38	0.394	0.22/0.010	0.35	0.50	38948/38950	1.000
Ch03 Mouse IS95 Low	0.26 to 0.27	0.269	0.17/0.005	0.25	0.30	38950/38950	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	38950/38950	1.000
July 1 to 31, 2012							
Ch01 Mouse IS95 High	0.53 to 0.56	0.581	0.19/0.013	0.49	0.77	97460/97462	1.000
Ch02 Mouse IS95 Med	0.38 to 0.40	0.414	0.21/0.010	0.36	0.55	97460/97462	1.000
Ch03 Mouse IS95 Low	0.27 to 0.28	0.284	0.17/0.005	0.25	0.38	97460/97462	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97462/97462	1.000
August 1 to 31, 2012							
Ch01 Mouse IS95 High	0.56 to 0.59	0.611	0.17/0.012	0.55	0.73	94976/94976	1.000
Ch02 Mouse IS95 Med	0.40 to 0.42	0.436	0.21/0.011	0.39	0.59	94974/94976	1.000
Ch03 Mouse IS95 Low	0.28 to 0.30	0.299	0.17/0.006	0.27	0.37	94974/94976	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94976/94976	1.000
September 1 to 30, 2012							
Ch01 Mouse IS95 High	0.59 to 0.61	0.638	0.17/0.013	0.58	0.69	94374/94374	1.000
Ch02 Mouse IS95 Med	0.42 to 0.43	0.454	0.19/0.010	0.42	0.53	94374/94374	1.000
Ch03 Mouse IS95 Low	0.30 to 0.30	0.313	0.17/0.006	0.29	0.35	94374/94374	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94370/94370	1.000
October 1 to 31, 2012							
Ch01 Mouse IS95 High	0.61 to 0.63	0.654	0.16/0.012	0.59	0.70	97604/97604	1.000
Ch02 Mouse IS95 Med	0.43 to 0.44	0.473	0.19/0.010	0.43	0.51	97604/97604	1.000
Ch03 Mouse IS95 Low	0.30 to 0.31	0.316	0.17/0.006	0.29	0.34	97604/97604	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97602/97602	1.000
November 1 to 30, 2012							
Ch01 Mouse IS95 High	0.63 to 0.64	0.672	0.17/0.013	0.44	0.72	94626/94628	1.000
Ch02 Mouse IS95 Med	0.44 to 0.46	0.487	0.22/0.012	0.34	0.54	94626/94628	1.000
Ch03 Mouse IS95 Low	0.31 to 0.32	0.324	0.17/0.006	0.23	0.36	94626/94628	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94628/94628	1.000
December 1 to 31, 2012							
Ch01 Mouse IS95 High	0.64 to 0.66	0.685	0.17/0.013	0.62	0.74	97500/97500	1.000
Ch02 Mouse IS95 Med	0.46 to 0.46	0.492	0.20/0.012	0.45	0.54	97500/97500	1.000
Ch03 Mouse IS95 Low	0.32 to 0.33	0.333	0.18/0.007	0.30	0.36	97500/97500	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97496/97496	1.000

[a] Ch=chamber (*e.g.*, Ch01=Chamber 1)

TABLE I8
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
January 1 to 31, 2013							
Ch01 Mouse IS95 High	0.66 to 0.66	0.697	0.17/0.014	0.63	0.77	97378/97378	1.000
Ch02 Mouse IS95 Med	0.46 to 0.47	0.497	0.19/0.011	0.45	0.55	97378/97378	1.000
Ch03 Mouse IS95 Low	0.33 to 0.33	0.342	0.18/0.007	0.31	0.38	97378/97378	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97364/97364	1.000
February 1 to 28, 2013							
Ch01 Mouse IS95 High	0.66 to 0.67	0.702	0.16/0.013	0.65	0.77	88116/88116	1.000
Ch02 Mouse IS95 Med	0.47 to 0.47	0.508	0.21/0.012	0.46	0.61	88114/88116	1.000
Ch03 Mouse IS95 Low	0.33 to 0.33	0.347	0.18/0.007	0.32	0.38	88116/88116	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	88116/88116	1.000
March 1 to 31, 2013							
Ch01 Mouse IS95 High	0.67 to 0.67	0.710	0.19/0.015	0.65	0.77	97784/97784	1.000
Ch02 Mouse IS95 Med	0.47 to 0.48	0.511	0.22/0.013	0.46	0.56	97784/97784	1.000
Ch03 Mouse IS95 Low	0.33 to 0.34	0.344	0.19/0.008	0.32	0.38	97784/97784	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97784/97784	1.000
April 1 to 30, 2013							
Ch01 Mouse IS95 High	0.67 to 0.68	0.713	0.20/0.016	0.65	0.82	96260/96260	1.000
Ch02 Mouse IS95 Med	0.48 to 0.48	0.511	0.19/0.011	0.47	0.56	96260/96260	1.000
Ch03 Mouse IS95 Low	0.34 to 0.34	0.348	0.18/0.007	0.32	0.38	96260/96260	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	96260/96260	1.000
May 1 to 31, 2013							
Ch01 Mouse IS95 High	0.68 to 0.69	0.716	0.19/0.016	0.63	0.79	97020/97020	1.000
Ch02 Mouse IS95 Med	0.48 to 0.49	0.518	0.21/0.013	0.46	0.59	97020/97020	1.000
Ch03 Mouse IS95 Low	0.34 to 0.34	0.351	0.17/0.007	0.32	0.39	97020/97020	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97020/97020	1.000
June 1 to 30, 2013							
Ch01 Mouse IS95 High	0.69 to 0.69	0.722	0.20/0.017	0.64	0.84	94514/94514	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.521	0.21/0.013	0.46	0.57	94514/94514	1.000
Ch03 Mouse IS95 Low	0.34 to 0.35	0.345	0.53/0.022	0.28	0.43	94512/94514	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94496/94496	1.000
July 1 to 31, 2013							
Ch01 Mouse IS95 High	0.69 to 0.69	0.723	0.21/0.018	0.63	0.84	99146/99146	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.523	0.21/0.013	0.48	0.58	99146/99146	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.316	0.45/0.017	0.28	0.41	99146/99146	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	99146/99146	1.000

TABLE I8
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
August 1 to 31, 2013							
Ch01 Mouse IS95 High	0.69 to 0.70	0.727	0.19/0.016	0.60	0.81	101712/101712	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.529	0.22/0.013	0.48	0.62	101710/101712	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.303	0.18/0.006	0.28	0.35	101712/101712	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	101700/101700	1.000
September 1 to 30, 2013							
Ch01 Mouse IS95 High	0.70 to 0.73	0.726	0.19/0.016	0.63	0.85	93922/93922	1.000
Ch02 Mouse IS95 Med	0.49 to 0.50	0.531	0.22/0.013	0.47	0.59	93922/93922	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.336	0.62/0.025	0.22	0.45	93914/93922	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	93920/93920	1.000
October 1 to 31, 2013							
Ch01 Mouse IS95 High	0.70 to 0.70	0.733	0.19/0.016	0.66	0.82	100816/100816	1.000
Ch02 Mouse IS95 Med	0.50 to 0.50	0.535	0.22/0.013	0.49	0.63	100816/100816	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.330	0.22/0.008	0.29	0.41	100816/100816	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100816/100816	1.000
November 1 to 30, 2013							
Ch01 Mouse IS95 High	0.70 to 0.70	0.724	0.20/0.017	0.48	0.81	93274/93278	1.000
Ch02 Mouse IS95 Med	0.50 to 0.50	0.532	0.23/0.014	0.36	0.59	93276/93278	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.327	0.27/0.010	0.19	0.43	93096/93278	0.998
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	93226/93226	1.000
December 1 to 31, 2013							
Ch01 Mouse IS95 High	0.70 to 0.70	0.693	0.25/0.020	0.59	0.82	96846/96846	1.000
Ch02 Mouse IS95 Med	0.49 to 0.50	0.524	0.24/0.015	0.46	0.62	96844/96846	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.321	0.19/0.007	0.28	0.36	96846/96846	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	96846/96846	1.000
January 1 to 31, 2014							
Ch01 Mouse IS95 High	0.70 to 0.70	0.703	0.22/0.018	0.57	0.81	97554/97554	1.000
Ch02 Mouse IS95 Med	0.49 to 0.50	0.532	0.22/0.014	0.27	0.62	97550/97554	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.329	0.39/0.015	0.14	0.41	97542/97552	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97550/97550	1.000
February 1 to 28, 2014							
Ch01 Mouse IS95 High	0.70 to 0.70	0.727	0.21/0.018	0.59	0.86	88184/88184	1.000
Ch02 Mouse IS95 Med	0.50 to 0.50	0.534	0.24/0.015	0.36	0.74	88170/88184	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.353	0.70/0.029	0.27	0.41	88180/88184	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	88184/88184	1.000

TABLE I8
Summary of CDMA-Modulated Cell Phone RFR Exposure Data – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/ Total	Ratio
March 1 to 31, 2014							
Ch01 Mouse IS95 High	0.70 to 0.70	0.694	0.65/0.054	0.10	0.90	96954/97180	0.998
Ch02 Mouse IS95 Med	0.50 to 0.50	0.520	0.21/0.013	0.34	0.71	97170/97180	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.365	0.43/0.019	0.19	0.58	97036/97180	0.999
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97142/97142	1.000
April 1 to 30, 2014							
Ch01 Mouse IS95 High	0.69 to 0.70	0.722	0.31/0.026	0.15	1.03	94494/94550	0.999
Ch02 Mouse IS95 Med	0.49 to 0.50	0.522	0.28/0.017	0.21	0.83	94406/94550	0.998
Ch03 Mouse IS95 Low	0.35 to 0.35	0.345	0.38/0.015	0.18	0.42	94540/94550	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94548/94548	1.000
May 1 to 31, 2014							
Ch01 Mouse IS95 High	0.69 to 0.69	0.721	0.21/0.017	0.64	0.82	97244/97244	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.521	0.20/0.012	0.46	0.60	97244/97244	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.354	0.37/0.016	0.26	0.42	97240/97244	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	97244/97244	1.000
June 1 to 30, 2014							
Ch01 Mouse IS95 High	0.69 to 0.69	0.710	0.17/0.014	0.60	0.79	94288/94288	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.509	0.24/0.014	0.41	0.57	94288/94288	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.361	0.27/0.011	0.24	0.43	94276/94288	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	94288/94288	1.000
July 1 to 9, 2014							
Ch01 Mouse IS95 High	0.69 to 0.69	0.701	0.14/0.011	0.65	0.76	25098/25098	1.000
Ch02 Mouse IS95 Med	0.49 to 0.49	0.491	0.14/0.008	0.44	0.53	25098/25098	1.000
Ch03 Mouse IS95 Low	0.35 to 0.35	0.355	0.68/0.029	0.19	0.49	24740/25098	0.986
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	25064/25064	1.000
June 18, 2012, to July 9, 2014							
Ch01 Mouse IS95 High	0.52 to 0.73	0.694	0.29/0.023	0.10	1.03	2272276/2272568	1.000
Ch02 Mouse IS95 Med	0.36 to 0.50	0.504	0.23/0.013	0.21	0.83	2272380/2272568	1.000
Ch03 Mouse IS95 Low	0.26 to 0.35	0.331	0.53/0.021	0.14	0.58	2272194/2272566	1.000
Ch13 Mouse Sham	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2272416/2272416	1.000

APPENDIX J
INGREDIENTS, NUTRIENT COMPOSITION, AND CONTAMINANT LEVELS IN NTP-2000 RAT AND MOUSE RATION

TABLE J1
Ingredients of NTP-2000 Rat and Mouse Ration

Ingredients	Percent by Weight
Ground hard winter wheat	22.26
Ground #2 yellow shelled corn	22.18
Wheat middlings	15.0
Oat hulls	8.5
Alfalfa meal (dehydrated, 17% protein)	7.5
Purified cellulose	5.5
Soybean meal (49% protein)	5.0
Fish meal (60% protein)	4.0
Corn oil (without preservatives)	3.0
Soy oil (without preservatives)	3.0
Dried brewer's yeast	1.0
Calcium carbonate (USP)	0.9
Vitamin premix[a]	0.5
Mineral premix[b]	0.5
Calcium phosphate, dibasic (USP)	0.4
Sodium chloride	0.3
Choline chloride (70% choline)	0.26
Methionine	0.2

[a] Wheat middlings as carrier
[b] Calcium carbonate as carrier

TABLE J2
Vitamins and Minerals in NTP-2000 Rat and Mouse Ration[a]

	Amount	Source
Vitamins		
A	4,000 IU	Stabilized vitamin A palmitate or acetate
D	1,000 IU	D-activated animal sterol
K	1.0 mg	Menadione sodium bisulfite complex
α-Tocopheryl acetate	100 IU	
Niacin	23 mg	
Folic acid	1.1 mg	
d-Pantothenic acid	10 mg	*d*-Calcium pantothenate
Riboflavin	3.3 mg	
Thiamine	4 mg	Thiamine mononitrate
B_{12}	52 µg	
Pyridoxine	6.3 mg	Pyridoxine hydrochloride
Biotin	0.2 mg	*d*-Biotin
Minerals		
Magnesium	514 mg	Magnesium oxide
Iron	35 mg	Iron sulfate
Zinc	12 mg	Zinc oxide
Manganese	10 mg	Manganese oxide
Copper	2.0 mg	Copper sulfate
Iodine	0.2 mg	Calcium iodate
Chromium	0.2 mg	Chromium acetate

[a] Per kg of finished product

TABLE J3
Nutrient Composition of NTP-2000 Rat and Mouse Ration

Nutrient	Mean ± Standard Deviation	Range	Number of Samples
Protein (% by weight)	14.4 ± 0.38	13.9 – 15.1	17
Crude fat (% by weight)	8.4 ± 0.37	7.7 – 9.2	17
Crude fiber (% by weight)	9.4 ± 0.41	8.6 – 9.9	17
Ash (% by weight)	4.9 ± 0.13	4.7 – 5.1	17
Amino Acids (% of total diet)			
Arginine	0.794 ± 0.070	0.67 – 0.97	26
Cystine	0.220 ± 0.022	0.15 – 0.25	26
Glycine	0.700 ± 0.038	0.62 – 0.80	26
Histidine	0.344 ± 0.074	0.27 – 0.68	26
Isoleucine	0.546 ± 0.041	0.43 – 0.66	26
Leucine	1.092 ± 0.063	0.96 – 1.24	26
Lysine	0.700 ± 0.110	0.31 – 0.86	26
Methionine	0.408 ± 0.043	0.26 – 0.49	26
Phenylalanine	0.621 ± 0.048	0.47 – 0.72	26
Threonine	0.508 ± 0.040	0.43 – 0.61	26
Tryptophan	0.153 ± 0.027	0.11 – 0.20	26
Tyrosine	0.413 ± 0.063	0.28 – 0.54	26
Valine	0.663 ± 0.040	0.55 – 0.73	26
Essential Fatty Acids (% of total diet)			
Linoleic	3.95 ± 0.242	3.49 – 4.55	26
Linolenic	0.31 ± 0.030	0.21 – 0.35	26
Vitamins			
Vitamin A (IU/kg)	3,899 ± 77	2,820 – 5,450	17
Vitamin D (IU/kg)	1,000[a]		
α-Tocopherol (ppm)	79.7 ± 20.42	27.0 – 124.0	26
Thiamine (ppm)[b]	11.8 ± 17.85	6.6 – 81.0	17
Riboflavin (ppm)	8.1 ± 2.91	4.20 – 17.50	26
Niacin (ppm)	78.9 ± 8.52	66.4 – 98.2	26
Pantothenic acid (ppm)	26.7 ± 11.63	17.4 – 81.0	26
Pyridoxine (ppm)[b]	9.7 ± 2.09	6.44 – 14.3	26
Folic acid (ppm)	1.59 ± 0.45	1.15 – 3.27	26
Biotin (ppm)	0.32 ± 0.10	0.20 – 0.704	26
Vitamin B_{12} (ppb)	51.8 ± 36.6	18.3 – 174.0	26
Choline (ppm)[b]	2,665 ± 631	1,160 – 3,790	26
Minerals			
Calcium (%)	0.903 ± 0.070	0.697 – 1.01	17
Phosphorus (%)	0.553 ± 0.026	0.510 – 0.596	17
Potassium (%)	0.669 ± 0.030	0.626 – 0.733	26
Chloride (%)	0.386 ± 0.037	0.300 – 0.474	26
Sodium (%)	0.193 ± 0.024	0.160 – 0.283	26
Magnesium (%)	0.216 ± 0.057	0.185 – 0.490	26
Sulfur (%)	0.170 ± 0.029	0.116 – 0.209	14
Iron (ppm)	190.5 ± 38.0	135 – 311	26
Manganese (ppm)	50.7 ± 9.72	21.0 – 73.1	26
Zinc (ppm)	58.2 ± 26.89	43.3 – 184.0	26
Copper (ppm)	7.44 ± 2.60	3.21 – 16.3	26
Iodine (ppm)	0.514 ± 0.195	0.158 – 0.972	26
Chromium (ppm)	0.674 ± 0.265	0.330 – 1.380	25
Cobalt (ppm)	0.235 ± 0.157	0.094 – 0.864	24

[a] From formulation
[b] As hydrochloride (thiamine and pyridoxine) or chloride (choline)

TABLE J4
Contaminant Levels in NTP-2000 Rat and Mouse Ration[a]

	Mean ± Standard Deviation[b]	Range	Number of Samples
Contaminants			
Arsenic (ppm)	0.20 ± 0.039	0.14 – 0.28	17
Cadmium (ppm)	0.05 ± 0.004	0.04 – 0.06	17
Lead (ppm)	0.21 ± 0.027	0.07 – 1.19	17
Mercury (ppm)	<0.02		17
Selenium (ppm)	0.17 ± 0.024	0.10 – 0.20	17
Aflatoxins (ppb)	<5.00		17
Nitrate nitrogen (ppm)[c]	18.76 ± 9.49	10.0 – 45.9	17
Nitrite nitrogen (ppm)[c]	0.61		17
BHA (ppm)[d]	<1.0		17
BHT (ppm)[d]	<1.0		17
Aerobic plate count (CFU/g)	<10.0		17
Coliform (MPN/g)	3.0		17
Escherichia coli (MPN/g)	<10		17
Salmonella (MPN/g)	Negative		17
Total nitrosoamines (ppb)[e]	9.2 ± 5.55	0.0 – 19.9	17
N-Nitrosodimethylamine (ppb)[e]	1.3 ± 1.04	0.0 – 3.0	17
N-Nitrosopyrrolidine (ppb)[e]	8.0 ± 5.02	0.0 – 18.6	17
Pesticides (ppm)			
α-BHC	<0.01		17
β-BHC	<0.02		17
γ-BHC	<0.01		17
δ-BHC	<0.01		17
Heptachlor	<0.01		17
Aldrin	<0.01		17
Heptachlor epoxide	<0.01		17
DDE	<0.01		17
DDD	<0.01		17
DDT	<0.01		17
HCB	<0.01		17
Mirex	<0.01		17
Methoxychlor	<0.05		17
Dieldrin	<0.01		17
Endrin	<0.01		17
Telodrin	<0.01		17
Chlordane	<0.05		17
Toxaphene	<0.10		17
Estimated PCBs	<0.20		17
Ronnel	<0.01		17
Ethion	<0.02		17
Trithion	<0.05		17
Diazinon	<0.10		17
Methyl chlorpyrifos	0.16 ± 0.179	0.02 – 0.686	17
Methyl parathion	<0.02		17
Ethyl parathion	<0.02		17
Malathion	0.117 ± 0.140	0.02 – 0.585	17
Endosulfan I	<0.01		17
Endosulfan II	<0.01		17
Endosulfan sulfate	<0.03		17

[a] All samples were irradiated. CFU=colony-forming units; MPN=most probable number; BHC=hexachlorocyclohexane or benzene hexachloride

[b] For values less than the limit of detection, the detection limit is given as the mean.

[c] Sources of contamination: alfalfa, grains, and fish meal

[d] Sources of contamination: soy oil and fish meal

[e] All values were corrected for percent recovery.

APPENDIX K
SENTINEL ANIMAL PROGRAM

SENTINEL ANIMAL PROGRAM

METHODS

Rodents used in the National Toxicology Program are produced in optimally clean facilities to eliminate potential pathogens that may affect study results. The Sentinel Animal Program is part of the periodic monitoring of animal health that occurs during the toxicologic evaluation of test agents. Under this program, the disease state of the rodents is monitored via sera or feces from extra (sentinel) or dosed animals in the study rooms. The sentinel animals and the study animals are subject to identical environmental conditions. Furthermore, the sentinel animals come from the same production source and weanling groups as the animals used for the studies of test agents.

Blood samples were collected and allowed to clot, and the serum was separated. All samples were processed appropriately with serology testing performed by IDEXX BioResearch [formerly Research Animal Diagnostic Laboratory (RADIL), University of Missouri, Columbia, MO] for determination of the presence of pathogens. The laboratory methods and agents for which testing was performed are tabulated below; the times at which samples were collected during the studies are also listed.

Blood was collected from five mice per sex per time point except for the following:
28-day studies, study termination collection: Two males and eight females
2-year studies, study termination collection: 10 males and 10 females

Method and Test	Time of Collection
28-Day Studies	
Multiplex Fluorescent Immunoassay	
Ectromelia virus	Study termination
EDIM (epizootic diarrhea of infant mice)	Study termination
LCMV (lymphocytic choriomeningitis virus)	Study termination
Mycoplasma pulmonis	Study termination
MHV (mouse hepatitis virus)	Study termination
MNV (mouse norovirus)	Study termination
MPV (mouse parvovirus)	Study termination
MVM (minute virus of mice)	Study termination
PVM (pneumonia virus of mice)	Study termination
REO3 (reovirus)	Study termination
Sendai	Study termination
TMEV (Theiler's murine encephalomyelitis virus)	Study termination

Method and Test	Time of Collection
2-Year Studies	
Multiplex Fluorescent Immunoassay	
Ectromelia virus	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
EDIM	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
LCMV	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
M. pulmonis	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
MHV	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
MNV	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
MPV	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
MVM	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
PVM	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
REO3	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
Sendai	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
TMEV	End of quarantine, 4 weeks, 6, 12, and 18 months, study termination
Immunofluorescence Assay	
MNV	Study termination
Polymerase Chain Reaction	
Helicobacter species	18 months

RESULTS

All test results were negative.