Teratogenic Effects of Radiofrequency Radiation

Joseph M. Lary
David L. Conover
Physical Agents Effects Branch
Division of Biomedical and Behavioral Science
National Institute for Occupational Safety and Health

OVER THE PAST four decades, the use of radiofrequency (RF) radiation sources has increased dramatically. Because of the increasing prevalence of occupational, medical, and environmental exposure to RF radiation, there is growing concern about the potential effects of such radiation on the unborn child. This article will assess the current state of knowledge of the effects of RF radiation on prenatal development. Although some experimental RF teratology work has been carried out using birds and insects as subjects, this review will be limited to the extensive work performed with mammals. The term RF radiation will be used to designate the portion of the electromagnetic spectrum below 3000 GHz, including the microwave spectrum from 300 MHz to 3000 GHz.

In a 1980 review of the RF teratology literature, O'Connor [1] concluded that only intense fields that result in significant heating are associated with reliable induction of teratogenesis. In a review that covered the RF teratology literature through 1981, Berman [2] concluded that teratogenesis is associated with high-intensity RF fields and that maternal temperature is the only available indicator of a threshold for teratogenesis in mammals. Since 1981, the RF teratology literature base has expanded considerably and has strengthened the hypothesis that RF-induced teratogenic and embryolethal effects result from hyperthermia produced by the RF fields and not from field-specific effects unrelated to heating. The RF teratology literature is generally consistent with the hyperthermia teratology literature. Recent reviews indicate that, regardless of the species, heat causes birth defects and prenatal mortality when the temperature of the body reaches 40° to 44°C [3, 4]. Birth defects appear to result from the direct effect of heat on the embryo, rather than from maternal toxicity [3, 4]. Because of the apparent relationship between RF-induced hyperthermia and teratogenic/embryotoxic effects in animals, the studies covered in this review have been arranged in order of the maternal colonic or rectal temperature induced by RF exposure.

Most of the health effects of exposure to RF radiation appear to be dependent upon the rate of energy absorption by the body. The rate of energy absorption is defined as the specific absorption rate (SAR) and is measured in watts per kilogram of body weight (W/kg). In several of the RF teratology studies covered in this review, the SAR was not measured or estimated. Often, studies reported only the power density of the incident radiation (mW/cm²) or the electric and magnetic field strength (V/m and A/m, respectively.) To permit a comparison of developmental effects across studies based on the rate of energy absorption, we have (when possible) estimated SARs for those papers in which it is not reported. The estimates are based on absorption curves in the *Radiofrequency Radiation Dosimetry Handbook* [5].

RF EFFECTS IN ANIMALS AT BODY TEMPERATURES OF 41°C OR HIGHER

RF fields which raise the core temperature of pregnant animals to 41°C or higher usually produce teratogenic and embryolethal effects. In a series of reports on virtually the same experiments [6–10], Hofmann, Dietzel, and their associates irradiated rats with a 27.12-MHz diathermy applicator, exposing the abdomen for 5 to 10 minutes or longer on one of gestation days 1 through 16. The electric and magnetic field strengths and the SAR were not determined. Rats were irradiated until their rectal temperatures reached 1°, 3°, or 4°C above their normal baseline temperature of 38°C. Final rectal temperatures were about 39°, 41°, or 42°C. The incidence of fetal malformations increased with temperature and was as high as 46 percent in animals heated to 42°C on gestation days 13 or 14. Exposures which resulted in temperatures of 41° or 42°C produced malformations of the tail, limbs, eyes, and head, and caused a high incidence of prenatal mortality. No significant effects occurred in the 39°C exposure group, compared to untreated controls.

In addition to rats, Hofmann and Dietzel [6] exposed the abdomens of rabbits to 27.12 MHz fields for 15 minutes on days 7, 8, and 9; days 10, 11, and 12; or days 17, 18, and 19 of gestation. Rectal temperatures increased by 1.3° to 4.0°C. The exposures produced a high incidence of abortions but no gross malformations.

In an earlier study of rabbits exposed to high-intensity 10-MHz radiation for one to two hours, two to five days/week before and during gestation [11], rectal temperatures rose to 41° to 42°C and litter size was reduced compared to controls. The offspring were morphologically normal.

Laskey, et al. [12] exposed rats to 2450 MHz microwaves at 100 mW/cm² for 8 to 13 minutes on day 2, days 2 and 5, day 8, or day 13 of gestation. At this intensity, the SAR is estimated to have been 21 W/kg [5]. Rectal temperatures averaged 41.4° to 42.8°C. Prenatal mortality was significantly increased in the day 8 group as compared to sham-irradiated or untreated controls. Mean body weight and mean brain weight were reduced in fetuses irradiated on gestation days 8 or 13.

In a study in which appreciable hyperthermia occurred but colonic temperatures were not measured, Moayer [13] irradiated rats for 2 to 4 minutes on gestation days 5 or 14 with a high-intensity 27.12 MHz shortwave applicator. The electric and magnetic field strengths and the SAR were not determined. The RF exposure damaged the placentas and decreased their vascularity. It also increased the incidence of aborted embryos.

In a series of related studies, Rugh and coworkers [14–17] exposed pregnant mice to 2450 MHz microwaves at an SAR of 93 to 114 W/kg (estimated power density of 123 mW/cm²). The exposures were acutely hyperthermic and just sublethal to the dam, but colonic temperatures were not monitored. Exposure for 2 to 5 minutes on gestation day 8 1/2 produced a high incidence of prenatal death and fetal malformations, predominately exencephaly and stunting [14, 15]. Other mice were exposed for 4 minutes on gestation days 9, 12, or 16 [16]. Male offspring irradiated on gestation day 12 and female offspring irradiated on gestation day 16 had significantly lower body weights than controls at two months of age.

In another study, mice were irradiated on one of gestation days 0 to 11 [17]. About 9 percent of the fetuses exposed on gestation days four or eight were malformed, while 42 percent exposed on gestation day eight died in utero. The observed malformations included stunting, deformed arms and heads, curly tails, and exencephaly.

Chernovetz, et al. [18] exposed rats for 20 minutes to 2450 MHz microwaves at 31 W/kg or to infrared radiation in

an incubator at an air temperature of about 47°C. The microwave exposures were made in a multimode cavity in which power density could not be measured. Rats were exposed on one of gestation days 10 through 16. In both groups, the mean colonic temperature of the animals increased to 42.0°C. Exposures were lethal to 27 percent of the microwave-treated dams and 12 percent of the infrared-treated dams. The incidence of prenatal mortality was higher in microwave-treated rats than in infrared-treated or sham-irradiated rats. No structural malformations occurred, but hemorrhages were common in both microwave- and infrared-treated fetuses. Fetal weight was reduced in both irradiated groups compared with controls. Brain norepinephrine levels were lower in microwave-treated fetuses than in infrared-treated or control fetuses, but brain dopamine levels were unchanged. The authors concluded that mortality and resorptions (early prenatal death) are probably a function of peak body temperature, irrespective of the radiation source.

Chernovetz, et al. [19] exposed rats to 2450 MHz microwaves for 20 minutes on one of gestation days 8, 10, 12, or 14 at SARs of 14 or 28 W/kg (power density could not be measured). Rectal temperatures increased from a preexposure temperature of about 38°C to about 40°C at 14 W/kg and 42°C at 28 W/kg. Controls were sham-irradiated or untreated. No structural malformations were observed at either temperature, nor was prenatal mortality increased. However, irradiation at 28 W/kg on gestation day 8 resulted in reduced fetal weight, while fetuses irradiated on gestation days 12 or 14 were heavier than controls. Fetuses irradiated at 14 W/kg on gestation days 12 or 14 were smaller than controls.

Lary, et al. [20] irradiated pregnant rats at 55 A/m and 300 V/m in a 27.12 MHz RF field, with a resultant SAR of 11.1 to 12.5 W/kg. Dams were irradiated for about 20 to 40 minutes on one of gestation days 1, 3, 5, 7, 9, 11, 13, or 15 until their colonic temperature reached 43.0°C. Controls were sham-irradiated or untreated. RF exposure during the postimplantation period (gestation days 7–15) produced a significant incidence of fetal malformations (up to 67 percent). Prenatal mortality was also high (up to 49 percent) after exposure on

gestation days 7 or 9. Fetal weight and crown-rump length were reduced in all of the postimplantation exposure groups. More than 200 different types of abnormalities were noted.

In a follow-up on their 1982 study, Lary, et al. [21] irradiated rats on gestation day 9 in a 27.12 MHz field at 55 A/m and 300 V/m with a resultant SAR of about 11 W/kg. Rats were exposed for different durations at colonic temperatures of 41.0° or 42.0°C. At both temperatures, the teratogenic and embryotoxic effects of RF-induced hyperthermia increased as the duration of exposure increased, but the increase was especially pronounced at 42.0°C.

Since the literature indicates that RF-induced teratogenesis is correlated with maternal hyperthermia, Lary, et al. [22] investigated the dose-response relationship between maternal core temperature during RF exposure and the incidence of congenital malformations and prenatal death. Rats were irradiated on gestation day 9 in a 27.12 MHz field at 55 A/m and 300 V/m until their colonic temperature reached 41.0°, 41.5°, 42.0°, 42.5°, or 43.0°C. The SAR was 10.8 W/kg. Control rats were sham irradiated or untreated. The incidences of birth defects and prenatal mortality increased directly with maternal colonic temperature after a threshold of approximately 41.5°C was exceeded (Fig. 1). About 15 percent of the dams heated to 43.0°C died; higher temperatures were nearly always lethal.

To determine if microwave-induced teratogenic effects are due to hyperthermia or to field-specific effects unrelated to heating, Rugh and McManaway [23] used anesthesia to lower the preexposure temperature of pregnant mice by 4° to 5°C. Hypothermic mice and normal-temperature mice were then exposed to 2450 MHz radiation for four minutes on gestation day 8. Based on information in a previous study [14], the power density was about 123 mW/cm^2, with a resultant SAR of 93 to 113 W/kg. The rectal temperatures of the normal-temperature mice climbed to an average of 40.8°C, with several exceeding 42°C. The rectal temperatures of the hypothermic mice climbed to the approximate normal baseline temperature of 37°C. The microwave exposure caused a high incidence of prenatal death and some malformations in the normal-temperature mice, but did not affect the hypothermic mice. These results strongly suggested that the teratogenic and embryolethal effects of RF radiation are due to hyperthermia rather than field-specific effects of the high-intensity exposure.

In a similar study, Lacy [24] found that lowering the body temperature with anesthesia before exposure protected pregnant rats against the teratogenic and embryolethal effects of high-intensity 27.12 MHz radiation. In the same report, Lacy compared the teratogenic effects of hyperthermia induced by exposure to RF radiation with hyperthermia induced by immersion in a 42.5°C hot water bath. In both treatment groups, rats were exposed for 20 to 30 minutes on gestation day nine until their colonic temperatures reached 43°C. The RF exposures occurred in a 27.12 MHz field at 55 A/m and 300 V/m, with a resultant SAR of 13.6 W/kg. Both hyperthermic exposures caused a high incidence of malformations and prenatal mortality. The incidence and types of malformations were nearly identical in both groups, as was the incidence of prenatal death. A light and electron microscopic study of the exposed embryos revealed that the type and extent of cellular damage caused by the two sources of hyperthermic exposure were identical. For both RF and conventional heating, selective cell death and inhibition of mitosis during embryogenesis appeared to be the most likely cause of the congenital malformations produced. Lacy concluded that the teratogenic and embryotoxic effects of exposure to high-intensity RF radiation results from hyperthermia, not from field-specific effects unrelated to heating.
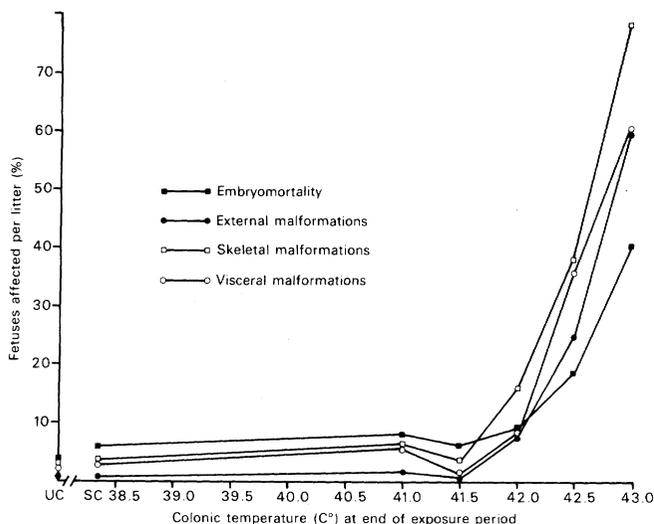

Figure 1. Incidence of embryonic death and fetal malformations in rats made hyperthermic by exposure to 27.12-MHz RF radiation for 10 to 40 minutes on gestation day 9. Colonic temperatures of untreated controls (UC) were not measured. Mean colonic temperature of sham-irradiated controls (SC) was 38.3°C at the end of the 45-minute sham exposure period. (From Lary, et al. [22]).

RF EFFECTS IN ANIMALS AT BODY TEMPERATURES OF 39° TO 41°C

RF exposures that increase the core temperature of the pregnant mother to 39° to 41.0°C generally do not result in gross structural malformations. However, the resultant thermal stress may significantly increase the incidence of prenatal mortality, result in lower body weight, cause histological or physiological changes, or alter the behavior of the exposed offspring.

Michaelson, et al. [25] exposed rats to 2450 MHz microwaves for one hour at 10 mW/cm^2 on gestation days nine and 16, or at 40 mW/cm^2 on gestation days 9, 13, 16, or 20. The estimated SARs were about 2.5 W/kg and 9 to 10 W/kg, respectively. Colonic temperatures increased by up to 0.5°C (38.1°C) at 10 mW/cm^2, and up to 3°C (39.5°C) at 40 mW/cm^2. Pups exposed to 40 mW/cm^2 fields had increased plasma cortisone and thyroxin levels and increased adrenal gland weight compared to sham-irradiated controls. Irradiated pups had a greater increase in metabolic rate during exposure to cold than did the control pups. However, no change was noted in the gestation length, litter size, or growth rate of the offspring at either exposure level.

Nawrot, et al. [26] exposed mice to 2450 MHz microwaves for eight hours/day at 5 mW/cm^2 (SAR = 6.7 W/kg) on gestation days 1 through 15, at 21 mW/cm^2 (SAR = 18.1 W/kg) on gestation days 1 through 6 or 6 through 15, or to 30 mW/cm^2 (SAR = 40.2 W/kg) on gestation days 1 through 6 or 6 through 15. Exposures at 21 and 30 mW/cm^2 caused increases in colonic temperature of 1°C (38.5°C) and 2.3°C (39.8°C), respectively. Exposure at 5 mW/cm^2 caused no increase in temperature. Groups of mice were also exposed to elevated ambient temperatures sufficient to raise mean colonic temperatures to the level of the groups exposed to microwaves at 21 and 30 mW/cm^2. Both the number of implantation sites and mean fetal weight were significantly reduced following exposure to 30 mW/cm^2 on days 1 through 6. A slight but significant increase in the percentage of malformed fetuses occurred following exposure to 30 mW/cm^2 on days 6 through 15. Conventional heat exposure causing a 2.3°C colonic temperature increase also resulted in a higher incidence of malformed fetuses than in controls, but the incidence was not statistically significant.

Berman, et al. [27] reported significantly increased prenatal mortality, decreased body weight, and decreased skeletal maturity in fetuses of Syrian hamsters exposed to 2450 MHz microwaves at 30 mW/cm^2 (SAR = 9 W/kg) for 100 minutes/day on gestation days 6 through 14. The mean colonic temperature increased to 39.8°C, 1.6°C higher than the temperature of sham-irradiated controls. Exposures at 20 mW/cm^2 (6 W/kg) neither increased colonic temperature nor produced any fetotoxic effects.

Berman and Carter [28] reported significantly lower fetal body weight and delayed skeletal development in rats exposed to 2450 MHz microwaves at 40 mW/cm^2 (SAR = 6.0 W/kg) for 100 minutes daily on gestation days 6 through 15. The exposure elevated the mean colonic temperature by 2°C (39.6°C).

Chernovetz, et al. [29] and Berman, et al. [30] found no significant alterations in prenatal development of mice or rats following exposures to 2450-MHz microwaves sufficient to increase mean maternal rectal temperatures to 40.3° and 40.6°C, respectively. The resultant SARs were 38 W/kg (mice) and 4.2 W/kg (rats).

RF EFFECTS IN ANIMALS AT BODY TEMPERATURES LESS THAN 39°C

Several investigators have reported significant physiological and behavioral changes in animals prenatally exposed to RF fields at levels which caused or were expected to cause little or no increase in body temperature. Other investigators found no significant effects at these levels. None of these exposures produced gross structural malformations in the offspring, but in several studies there were indications of an embryotoxic response. In the majority of the positive reports, pregnant rats were exposed to 100, 918, 2450, or 6000 MHz-fields at SARs ranging from 0.3 to 7.3 W/kg [31–39]. Decreased fetal weight or postnatal body weight was the most common effect reported. Other effects noted in the offspring included a decreased number of Purkinje cells in the cerebellum; changes in the levels of adenosine tri-, di-, and monophosphate in the brain; lower brain weight; earlier eye opening; greater postnatal activity; poorer performance on behavioral tests; less tolerance to cold stress; and an increase in mitogen-stimulated response of T- and B-lymphocytes in the blood.

In other studies, pregnant mice were exposed to 2450 or 3000 MHz fields at SARs of 11 to 16.5 W/kg [40–43]. The predominant effect was lower fetal weight or birth weight. Increased prenatal and postnatal mortality was also reported, as well as increased minor fetal anomalies which appeared to be caused by delayed embryonic development. Other effects reported included lower brain weight, delayed skeletal development, and delayed behavioral development.

Kaplan, et al. [44] reported significantly higher prenatal and postnatal mortality in squirrel monkeys irradiated with 2450 MHz microwaves at an SAR of 3.4 W/kg. However, they were unable to replicate these findings in a more extensive follow-up study [45].

No significant effects were reported in rats prenatally exposed to 100, 915, 1700, or 2450 MHz fields at SARs of 0.4 to 5 W/kg [46–54]. No effects were reported in mice prenatally exposed to 2450 MHz microwaves at 2.0 to 22 W/kg [55, 56]. Albert, et al. [57] found no significant effects on the brains of squirrel monkeys prenatally exposed to 2450 MHz microwaves at 3.4 W/kg compared to sham-irradiated controls.

Because of the highly nonuniform absorption of RF energy in exposed animals, it is possible that appreciable heating of the uterus or other organs of the body sometimes occurs in the absence of a significant increase in maternal rectal or colonic temperature. Therefore, significant developmental effects that occur in the absence of a measurable increase in rectal or colonic temperature cannot necessarily be attributed to a nonthermal causative mechanism. While the possibility of significant nonthermal RF effects on prenatal development cannot be excluded, the literature to date indicates that heat is the probable cause of RF-induced teratogenic and embryotoxic effects.

RF EFFECTS ON HUMAN DEVELOPMENT

Reports of possible adverse effects of RF exposure on human development are sparse and anecdotal. Most of the published reports have dealt with embryos exposed to RF diathermy during early development, before pregnancy had been diagnosed. Hofmann and Dietzel [6], in a review of the literature on RF diathermy treatment during pregnancy, discussed the physiological consequences of shortwave heating of the uterus and concluded that shortwave diathermy is absolutely contraindicated during pregnancy.

Marchese [58] reported on a woman inadvertently given a series of shortwave treatments of the abdomen (presumably at 27.12 MHz) of 10 to 30 minutes during the first month of pregnancy. She gave birth to a child that was one month premature and had multiple congenital malformations. Cocozza, et al. [59] and Minecki [60] described a grossly malformed infant born to a woman inadvertently given

shortwave (presumably 27.12 MHz) treatments to the abdomen for 20 minutes daily or on alternate days during the first and second months of pregnancy. The baby was born two months premature and weighed only 2.2 kg.

Rubin and Erdman [61] reported on two women given 2450-MHz diathermy treatment of the pelvic region just prior to and during early pregnancy. One aborted the embryo on gestation day 67, while the other delivered a normal infant at term. Imrie [62] related that one of three pregnant women aborted following shortwave (presumably 27.12 MHz) diathermy treatment of the pelvis during the first trimester of pregnancy. The other two delivered normal infants at term. Imrie measured the temperatures of 25 women exposed to pelvic diathermy and found an average uterine temperature increase of 0.5°C in premenopausal women and 1.0°C in postmenopausal women. He concluded that the danger to the human embryo from accidental exposure to diathermy radiation during early pregnancy is not as great as suspected by Hofmann and Dietzel [6].

In a cohort epidemiology study of 2043 babies born to 2018 females registered as physiotherapists at the time of pregnancy, Kallen, et al. [63] found a slightly higher incidence of shortwave equipment use among females who delivered dead or malformed infants than among control females. However, the authors reported that the statistical significance was borderline ($p = 0.03$), and that this could be a random finding.

At present, there is little evidence to suggest that exposure to common sources of RF radiation is teratogenic or embryotoxic in human beings. People do not often encounter RF fields that exceed the consensus standard recommended by the American National Standards Institute (ANSI) [64]. The ANSI limits are designed to prevent average whole-body SARs in excess of 0.4 W/kg. In contrast, the lowest SAR reported to cause fetal malformations in animals is 10.8 W/kg [22], while the lowest SAR reported to cause prenatal death is 9 W/kg [27]. While some developmental effects, including reduced body weight and changes in postnatal behavior, have been reported in animals at SARs between 0.3 and 11 W/kg, there is presently no indication that RF exposures below the ANSI limits will have any effect on the unborn child. The teratology literature indicates that only exposures that result in appreciable heating of the uterus (i.e., greater than 39°C) are likely to adversely affect the human embryo. Only RF sources that emit radiation substantially in excess of the ANSI standard are likely to cause significant heating of the human body. Significant heating may occur from medical exposure to RF diathermy or magnetic resonance imaging equipment and from close-proximity exposure to high intensity radar antennas, radio and TV broadcast antennas, or unshielded RF heaters and sealers. Except for medical treatment under the care of a physician, it is advisable that women avoid exposure to such high-intensity RF sources when they are pregnant or pregnancy is suspected.

REFERENCES

1. **O'Connor ME:** Mammalian teratogenesis and radio-frequency fields. *Proc IEEE*, 68:56, 1980.
2. **Berman E:** Reproductive efffects. In: *Biological Effects of Radiofrequency Radiation*, Elder JA, Cahill DF (Eds.). EPA-600/8-83-026F, Health Effects Research Laboratory, US Environmental Protection Agency, Research Triangle Park, NC, 1984.
3. **Edwards MJ:** Congenital defects due to hyperthermia. *Adv Vet Sci Comp Med*, 22:29, 1978.
4. **Lary JM:** Hyperthermia and teratogenicity. In: *Hyperthermia in Cancer Treatment*, Vol. 1, Anghileri A, Robert D (Eds.). pp. 107–126, Boca Raton, CRC Press, 1986.
5. **Durney C, Johnson C, Barber P, et al.:** *Radiofrequency Radiation Dosimetry Handbook* (2nd edition). Report SAM-TR-78-22, US Air Force School of Aerospace Medicine, Brooks Air Force Base, Texas, 1978.
6. **Hofmann D, Dietzel F:** Aborte und Missbildungen nach Kurzwellendurch-flutung in der Schwangershaft [Abortions and deformities following shortwave diathermy during pregnancy]. *Geburts Frauenheilkunde*, 26:378, 1966.
7. **Dietzel F, Kern W:** Fehlgeburt nach Kurzwellenbehandlung—Tierexperimentelle Untersuchungen [Abortion following ultra-short-wave hyperthermia—animal experiments]. *Arch Gynäek* 209:237, 1970.
8. **Dietzel F, Kern W:** Kann hohes mütterliches Fieber Missbildungen beim Kind auslösen? [Can maternal pyrexia cause fetal deformities?] *Geburtshilfe und Frauenheilkunde*, 31:1074, 1971.
9. **Dietzel F, Kern W, Steckenmesser R:** Missbildungen und intrauterines Absterben nach Kurzwellenbehandlung in der Früschwangerschaft Tierexperimentelle Untersuch ungen. [Deformity and intrauterine death after short wave therapy. Animal experiments]. *Muench Med Wochenshrift*, 114:228, 1972.
10. **Dietzel F:** Effects of electromagnetic radiation on implantation and intrauterine development of the rat. *Annals NY Acad Science*, 247:367, 1975.
11. **Boak RA, Carpenter CM, Warren SL:** Studies on the physiological effects of fever temperatures II. The effect of repeated short wave (30 meter) fevers on growth and fertility in rabbits. *J Exp Med*, 56:725, 1932.
12. **Laskey J, Dawes D, Howes M:** Progress report on 2450 MHz irradiation of pregnant rats and the effect on the fetus. In: *Radiation Bioeffects Summary Report*, PHS, USDHEW, BRH/DBE-70, Rockville, Maryland, pp. 167–173, 1970.
13. **Moayer M:** Die morphologischen Veränderungen der Plazenta unter dem Einfluss der Kurzwellendurchflutung. Tierexperimentelle Untersuchengen [The morphological changes of the placenta under the influence of short-wave irradiation. Experimental investigations on animals]. *Strahlentherapie*, 142:609, 1971.
14. **Rugh R, Ginns E, Ho H, et al.:** Are microwaves teratogenic? In: *Biological Effects and Health Hazards of Microwave Radiation*, Czerski P, Ostrowski K, Shore M, et al. (Eds.). pp. 98–107. Warsaw: Polish Medical Publishers, 1974.
15. **Rugh R, Ginns E, Ho H, et al.:** Responses of the mouse to microwave radiation during estrous cycle and pregnancy. *Radiation Res*, 62:225, 1975.
16. **Rugh R:** Are mouse fetuses which survive microwave radiation permanently affected thereby? *Health Physics*, 31:33, 1976.
17. **Rugh R, McManaway M:** Mouse fetal sensitivity to microwave radiation. *Congenital Anomalies*, 17:39, 1977.
18. **Chernovetz ME, Justesen DR, Oke AF:** A teratological study of the rat: Microwave and infrared radiations compared. *Radio Sci*, 12(6S):191, 1977.
19. **Chernovetz ME, Justesen DR, Levinson DM:** Acceleration and deceleration of fetal growth by 2450-MHz microwave irradiation of rats. In: *Electromagnetic Fields in Biological Systems*, pp. 175–193, Stuchly SS (Ed.). Ottawa, Canada, 1979.
20. **Lary JM, Conover DL, Foley ED, et al.:** Teratogenic effects of 27.12 MHz radiofrequency radiation in rats. *Teratology*, 26:299, 1982.
21. **Lary JM, Conover DL, Johnson PH, et al.:** Teratogenicity of 27.12-MHz radiation in rats is related to duration of hyperthermic exposure. *Bioelectromagnetics*, 4:249, 1983.
22. **Lary JM, Conover DL, Johnson PH, et al.:** Dose-response relationship between body temperature and birth defects in radiofrequency-irradiated rats. *Bioelectromagnetics*, 7:141–149, 1986.
23. **Rugh R, McManaway M:** Anesthesia as an effective agent against the production of congenital anomalies in mouse fetuses exposed to electromagnetic radiation. *J Exp Zool*, 197:363, 1976.
24. **Lacy KK:** *The Role of Hyperthermia in Radiofrequency Induced Teratogenesis*. (Ph.D. Dissertation) Univ. of Cincinnati, Ohio, 1981.
25. **Michaelson SM, Guillet R, Hegeness FW:** Influence of microwave exposure on functional maturation of the rat. In: *Developmental Toxicology of Energy-Related Pollutants*, Mahlum DD, Sikov MR, Hackett PL, et al. (Eds.). pp. 300–316, DOE Symposium Series 47, Washington, D.C., 1978.
26. **Nawrot PS, McRee DI, Staples RE:** Effects of 2.45 GHz CW microwave radiation on embryofetal development in mice. *Teratology*, 24:303, 1981.
27. **Berman E, Carter HB, House D:** Observations of Syrian hamster fetuses after exposure to 2450-MHz microwaves. *J Microwave Power*, 17:107, 1982.
28. **Berman E, Carter HB:** Decreased body weight in fetal rats after irradiation with 2450-MHz (CW) microwaves. *Health Physics*, 46:537, 1984.
29. **Chernovetz M, Justesen D, King N, et al.:** Teratology, survival, and reversal learning after fetal irradiation of mice by 2450-MHz microwave energy. *J Microwave Power*, 10:391, 1975.
30. **Berman E, Carter HB, House D:** Observations of rat fetuses after irradiation with 2450-MHz (CW) microwaves. *J Microwave Power*, 16:9, 1981.
31. **Albert E, Sherif M, Papadopoulos N, et al.:** Effect of nonionizing radiation on the Purkinje cells of the rat cerebellum. *Bioelectromagnetics*, 2:247, 1981.
32. **Jamakosmanovic A, Nakás M, Drecun M, et al.:** The levels of ATP, ADP, and AMP in rat brain during postnatal development following "in utero" 2450 MHz microwave irradiation. *Periodicum Biologorum*, 81:151, 1981.
33. **Jensh RP:** Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. I. Morphologic analysis at term. *Radiation Res*, 97:272, 1984.
34. **Jensh RP:** Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. II. Postnatal psychophysiologic evaluations. *Radiation Res*, 97:282, 1984b.
35. **Jensh RP, Vogel WH, Brent RL:** An evaluation of the teratogenic

potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation. II. Postnatal psychophysiological analysis. *J Toxicol Environ Health*, 11:37, 1983.

36. **Johnson RB, Mizumori S, Lovely RH:** Adult behavioral deficit in rats exposed prenatally to 918-MHz microwaves. In: *Developmental Toxicology of Energy-Related Pollutants*, Mahlum DD, Hackett PL, Andrews FD, (Eds.). pp. 281–299, DOE Symposium Series 47, Washington, D.C., 1978.

37. **Lovely R, Mizumori S, Johnson R, et al.:** Subtle consequences of exposure to weak microwave fields: Are there nonthermal effects? In: *Microwaves and Thermoregulation*, Adair ER (Ed.). pp. 401–430, New York: Academic Press, 1983.

38. **Shore ML, Felten RP, Lamanna A:** The effect of repetitive prenatal low-level microwave exposure on development in the rat. In: *Symposium on Biological Effects and Measurement of Radio Frequency/Microwaves*, Hazard DG, (Ed.). pp. 280–289, HEW Publication (FDA) 77-8026, Rockville, Maryland, 1977.

39. **Smialowicz RJ, Kinn JB, Elder JA:** Perinatal exposure of rats to 2450-MHz CW microwave radiation: Effects on lymphocytes. *Radio Sci*, 14(6S):147, 1979.

40. **Bereznitskaya AN:** [Some indicators of fertility in female mice irradiated with 10-cm long waves.] Gigiena Truda i Professional'nye Zabolevaniya (Moscow), 9:33, 1968.

41. **Bereznitskaya AN, Rysina TZ:** Embryotropic effects of microwaves. In: *Biological Effects of Radiofrequency Electromagnetic Fields*, Gordon ZV (Ed.). pp. 230–236, US Joint Publications Research Service, 1974.

42. **Berman E, Carter HB, House D:** Reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves. *Bioelectromagnetics*, 3:285, 1982.

43. **Berman E, Carter HB, House D:** Growth and development of mice offspring after irradiation in utero with 2,450-MHz microwaves. *Teratology*, 30:393, 1984.

44. **Kaplan J, Polson P, Rebert C, et al.:** Biological and behavioral effects of prenatal and postnatal exposure to 2450-MHz electromagnetic radiation in the squirrel monkey. *Radio Sci*, 17(5S): 135S, 1982.

45. **Kaplan JN:** *Study of the lethal effects of microwaves in the developing squirrel monkey.* Final report for EPA contract no. 68-02-3210. EPA 600/0-81-039. US Environmental Protection Agency, Health Effects Research Laboratory, Research Triangle Park, North Carolina, Sept. 1981.

46. **Rioch EM:** *Effects of microwave irradiation on embryonic brain tissue.* Report on US Army contract no. DAH C04-74-0004, pp. 1–7, 1974.

47. **Jensh R, Ludlow J, Vogel W, et al.:** Studies concerning the effects of non-thermal protracted prenatal 915 MHz microwave radiation on prenatal and postnatal development in the rat. *Resumes of the XIV Symposium International sur les Applications Energetiques des Microondes*, pp. 99–101, IMPI, Paris, France, 1979.

48. **Jensh RP:** Behavioral teratology: Application in low dose chronic microwave irradiation studies. In: *Neural and Behavioral Teratology.* Vol. 4, Advances in the Study of Birth Defects, Persaud TVN, (Ed.). pp. 135–162, Baltimore: University Park Press, 1980.

49. **Jensh RP, Vogel WH, Brent RL:** Postnatal functional analysis of prenatal exposure of rats to 915 MHz microwave radiation. *J Amer College Toxicol*, 1:73, 1982.

50. **Jensh RP, Weinberg I, Brent RL:** Teratologic studies of prenatal exposure of rats to 915-MHz microwave radiation. *Radiation Res*, 92:160, 1982.

51. **Inouye M, Galvin MJ, McRee DI:** Effect of 2,450 MHz microwave radiation on the development of the rat brain. *Teratology*, 28:413, 1983.

52. **Jensh RP, Weinberg I, Brent RL:** An evaluation of the teratogenic potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation: I. Morphological analysis at term: *J Toxicol Environ Health*, 11:23, 1983.

53. **Lary JM, Conover DL, Johnson PH:** Absence of embryotoxic effects from low-level (nonthermal) exposure of rats to 100 MHz radiofrequency radiation. *Scand J Work Environ Health*, 9:120, 1983.

54. **Merritt JH, Hardy KA, Chamness AF:** In utero exposure to microwave radiation and rat brain development. *Bioelectromagnetics*, 5:315, 1984.

55. **Berman E, Kinn JB, Carter HB:** Observations of mouse fetuses after irradiation with 2.45 GHz microwaves. *Health Physics*, 35:791, 1978.

56. **Inouye M, Matsumoto M, Galvin M, et al.:** Lack of effect of 2.45-GHz microwave radiation on the development of preimplantation embryos of mice. *Bioelectromagnetics*, 3:275, 1982.

57. **Albert EN, Sherif MF, Papadopoulos NJ:** Effect of nonionizing radiation on the Purkinje cells of the uvula in squirrel monkey cerebellum. *Bioelectromagnetics*, 2:241, 1981.

58. **Marchese GS:** Prematuranza e malformazioni congenite multiple da onde corte? Nota preventiva [Premature birth and multiple congenital malformations due to short waves? Preventive note]. *Minerva nipiol*, 3:100, 1953.

59. **Cocozza G, Blasio A, Nunziata B:** Rilievi sulle embriopatie da onde corte [Remarks on short-wave embryopathy]. *Pediatria rivista d'igiene med e chir dell'infanzia*, 68:7, 1960.

60. **Minecki L:** [Influence of high electromagnetic fields on embryogenesis.] *Medycyna Pracy*, 15:391, 1964.

61. **Rubin A, Erdman WJ:** Microwave exposure of the human female pelvis during early pregnancy and prior to conception. *Amer J Phys Med*, 38:219, 1959.

62. **Imrie AH:** Pelvic short wave diathermy given inadvertently in early pregnancy. *J Obstet Gynaecol Brit Commonwealth*, 78:91, 1971.

63. **Kallen B, Malmquist G, Moritz U:** Delivery outcome among physiotherapists in Sweden: Is non-ionizing radiation a fetal hazard? *Arch Environ Health*, 37:81, 1982.

64. **American National Standards Institute.** *American national standard safety levels with respect to human exposure to radiofrequency electromagnetic fields (300 kHz-100 GHz).* ANSI C95.1-1982. New York, 1982.

Joseph M. Lary was born in Meridian, Mississippi on July 29, 1946. He received the B.S. degree in physics in 1968 and the M.S. and Ph.D. degrees in developmental biology from the University of Alabama in Tuscaloosa in 1973 and 1977, respectively. Dr. Lary is a commander in the U.S. Public Health Service. Since 1977 he has been a research biologist for the Physical Agents Effects Branch of the National Institute for Occupational Safety and Health in Cincinnati, Ohio. His primary research interests are on the reproductive and teratogenic effects of radio-frequency radiation. He has also worked extensively to develop a recommended federal occupational exposure standard for radio-frequency radiation.

Dr. Lary is a member of the Bioelectromagnetics Society, Teratology Society, Sigma Xi, Sigma Pi Sigma, Beta Beta Beta, Psi Chi, and the Creation Research Society. He is also a member of ANSI C95 Subcommittee IV and the WHO Working Group on Nonionizing Radiation. Dr. Lary is a research advisor for the National Research Council of the National Academy of Sciences, past president of the Cincinnati Federal Environmental Chapter of Sigma Xi, and is listed in the second edition of *Who's Who in Frontiers of Science and Technology.*

Dr. Lary's address is Physical Agents Effects Branch, NIOSH, 4676 Columbia Parkway, Cincinnati, OH 45226.

David L. Conover received the B.A. in physics from Taylor University, Upland, Indiana in 1968, and the M.S. in 1969 and the Ph.D. in 1973 in health physics from Purdue University. Dr. Conover is currently a research physicist at NIOSH.

Dr. Conover contributed to the development of unique radiofrequency (RF) survey monitors, RF exposure/calibration facilities for bioeffects and dosimetry research, and RF power absorption analyzer equipment to assist in extrapolation of animal bioeffects data to man. He made major contributions to a NIOSH Control Technology Document to reduce worker exposure from RF dielectric heaters, and he was extensively involved in the development of the NIOSH RF/microwave worker exposure standard. Dr. Conover was project officer for the RF teratology research project and participated in its biological and dosimetry aspects. He was awarded U.S. Public Health Service commendation medals in 1979 and 1983 for contributions to the RF teratology project and the development of unique RF dosimetry equipment for improved worker protection. He is a member of the NCRP Scientific Committee No. 78 (RF/Microwave Exposure Measurements) and ANSI C95.1 (RF Exposure Measurements).