Title: Microwave Radiation and Thermoregulation

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- Phantom models

Abstract:
Low intensity microwave fields alter normal responses, both autonomic and behavioral, that regulate the body temperature. Using the squirrel monkey as an animal model, we have quantified the minimal intensity of 2450 MHz CW microwaves that will lower metabolic heat production in the cold, initiate thermoregulatory sweating in the heat, alter peripheral vasomotor tone in thermoneutral environments, and stimulate a behaving animal to select a cooler...
Abstract (Box 20) cont'd.

Environment. The threshold intensities for all responses were remarkably similar (4-8 mW/cm²), representing 15-20% of the monkey's resting metabolic rate. This finding suggests a common thermal basis for the response changes. Autonomic responses that generate or dissipate body heat showed some adaptation during prolonged microwave exposure whereas behavioral thermoregulatory responses persisted unchanged as long as the microwave field was present. Partial body microwave exposure produced appropriate adjustments in thermoregulatory responses to a degree nominally proportional to the fraction of the body so exposed. In general, whether the environment is cold or warm, endotherms detect and respond immediately to low intensity microwave fields as they do to other environmental thermal stimuli with the result that internal body temperature is regulated with precision at the normal level.
MICROWAVE RADIATION AND THERMOREGULATION

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SUMMARY

The goal of this research project has been to quantify, using the squirrel monkey as an animal model, the minimal incident energy (in mW/cm²) derived from 2450 MHz CW microwaves that reliably influences the normal responses, both physiological and behavioral, that regulate the body temperature. The nature of the thermoregulatory disturbance is characterized in terms of both absorbed energy and resulting local body temperature perturbations. Additional goals have involved exploration of other parameters of microwave exposure (e.g., intensity, duration, partial vs whole body exposure) for their impact upon thermoregulatory functioning. Controls for surface heating, provided by infrared radiation or convective heating, have been an integral part of most experimental designs.

The animals are chair restrained in the far field of a horn antenna inside a 1.83 m x 1.83 m x 2.45 m electromagnetically anechoic chamber. A valve system allows air from one of two closely-regulated (+ 0.5°C) air sources to circulate through the anechoic space. The monkey is trained to pull a response cord to operate the valves, thereby selecting the environmental temperature he prefers. Use of a single air source provides an environment of constant temperature for the assessment of physiological thermoregulatory responses. Sometimes, in order to achieve more precise control over environmental temperature in his immediate vicinity, the monkey is confined within an air-conditioned styrofoam box.

Continuous microwaves of a single frequency, 2450 ± 25 MHz, are generated by a Cober 52.5 W generator and fed to the antenna through standard waveguide components. Calibrations to determine field uniformity at the animal's location, using a Narda 8316B broadband isotropic radiation detector, show a maximum nonuniformity of 8%. With the restraining chair absent and an additional 5% with chair present. Insignificant changes occur with the introduction of a hood and hose connections for measurement of oxygen consumption, fine thermocouples for measurement of body temperatures, or a lucite boot and hose connections for measurement of thermoregulatory sweating from the foot of the animal.

Assessment of whole-body energy absorption over the power density range 5-40 mW/cm² has been based upon temperature increments produced at 4 depths in 3 sizes of saline-filled cylindrical styrofoam models by 10-min microwave exposures. The mean temperature rise in the liquid above an equilibrated 35°C was used to calculate the specific absorption rate (SAR). This ranged from 0.135 to 0.153 W/kg.mW-l.cm-2, with the higher values corresponding to the smaller mass. Rectal temperature increments in conscious squirrel monkeys undergoing 10-minute microwave exposures in thermo-neutral environments yielded a comparable SAR of 0.150 W/kg.mW-l.cm-2.

Brief (5-10 minute) whole-body exposure to 2450 MHz CW microwaves (E polarization) has allowed us to determine the minimal power density (mW/cm²) that reliably alters thermoregulatory responses (thresholds). These thresholds are very similar: 6-8 mW/cm² (SAR = 1.1 W/kg) stimulates the animal to lower the chamber air temperature behaviorally, induces peripheral vasodilation of the tail vessels, and initiates thermoregulatory sweating from the foot, while 4-6 mW/cm² (SAR = 0.8 W/kg) reliably lowers metabolic heat production in cold environments.

Microwave intensities above threshold stimulate proportionally greater response changes. Extending the exposure duration (up to 2 1/2 hours) produces little or no adaptation of behavioral thermoregulatory adjustments, but does produce a gradual adjustment in metabolic heat production such that, in the steady state, the SAR (W/kg)
is exactly balanced by the reduction in metabolic heat production (W/kg). This result confirms our dosimetry studies of temperature increments in saline-filled styrofoam models. Adaptation also occurs in thermoregulatory sweating during prolonged microwave exposure in warm environments, but it is not sufficient to prevent a rise in body temperature in this species.

When only the head (side or back) is exposed to the microwave field, the trunk and extremities being screened, the power density must be nearly 10 times stronger than for whole-body exposure to produce a given reduction of metabolic heat production in a cold environment. Evidently heat deposited directly in the head is efficiently carried away by the circulatory system. However, under these same conditions, power densities as high as 60 mW/cm$^2$ fail to influence the environmental temperature selected by a behaving animal. On the other hand, when the head is screened but the rest of the body is exposed to the microwave field, the thresholds for reduction of heat production or alteration of thermoregulatory behavior are only slightly higher than those measured during whole-body exposure. Results indicate that whatever portion of the body is exposed, the response depends upon the absorbed energy integrated over the total body mass.

The ultimate goal of research such as that described above is to evaluate the impact of microwave exposure on the human thermoregulatory system with reference to current maximum permissible exposure standards. This must necessarily involve careful power and frequency extrapolations as well as considerations of body heat balance in man vs the animal model. It would appear that, with the possible exception of exposure to moderate microwave intensities in warm environments, the squirrel monkey model will be extremely useful in this regard. Our most important single finding over the four years of this research project is that, no matter in what thermal environment the organism finds itself, it detects and responds immediately to low intensity microwave fields as it does to other environmental thermal stimuli. The net result is that internal body temperature is regulated with precision at the normal level.
BACKGROUND

A. Temperature changes and thermoregulation

The ability to maintain a constant internal body temperature is essential to the survival and optimal functioning of every organism. Every species exhibits a characteristic strategy for dealing with the thermal aspects of its environment in order to maintain a characteristic (neutral) body temperature. These strategies range from simple locomotion (thermotropisms) of unicellular organisms to the combined behavioral-autonomic mechanisms exhibited by primates including man. Ectotherms exhibit strategies that are largely behavioral because autonomic mechanisms of heat loss and heat production are limited. On the other hand, the higher vertebrates (endotherms), although quite capable of autonomic regulation, nevertheless utilize behavioral strategies whenever possible. Behavioral responses can exert a powerful control over the environment, thus providing a narrow range of temperatures at the air-skin interface. Peripheral vasomotor responses function within this narrow zone to exert additional control over the body temperature. Endotherms thus try to avoid shivering or sweating, with constant economy of body energy stores and body water.

In intact animals, thermal stimuli will elicit both behavioral and autonomic thermoregulatory responses. These stimuli include not only variations in the microclimate (ambient temperature, ambient vapor pressure, insulation, etc.) but also internal temperature changes due to circadian variation, febrile disease, exercise, etc. In experimental animals, highly localized temperature changes in specific central nervous system (CNS) sites can be brought about by implanted devices called thermodes, so as to study the role played by these sites in normal thermoregulation (Hardy, 1961; Hammel, 1968). Extensive neurophysiological research, beginning with the studies of lesioned animals nearly a century ago (Aronsohn and Sachs, 1885; Ott, 1887), has shown that certain CNS tissue is essential for, or at least involved in, the control of autonomic mechanisms of heat loss and heat production in endotherms. As early as 1912, Barbour showed that a primitive thermode implanted in the rostral brainstem could be used to trigger many thermoregulatory responses. Beginning with the work in Hardy's laboratory in 1961, many CNS sites have been explored with microelectrodes revealing the existence of single units whose firing rates depend upon the local temperature as controlled by a thermode (Guieu and Hardy, 1971). Tissue sites that have been shown to exhibit such thermosensitivity include medial preoptic area, lateral hypothalamus, posterior hypothalamus, midbrain reticular formation, motor cortex, thalamus, spinal cord, medulla and deep viscera. Temperature changes localized to several of these areas can trigger behavioral thermoregulatory responses as well as autonomic responses (Satinoff, 1964; Adair, 1974; Lipton, 1971; Cabanac, 1972; Carlisle and Ingram, 1973). Thus, any means by which such central nervous system structures may be thermally stimulated has the potential for altering the normal thermoregulatory response strategy of an endotherm. Clearly microwave radiation, known to be absorbed in extremely complex configurations by biological entities, is such a potential stimulus.

Of course, the primary source of an organism's information about the thermal characteristics of the environment resides in the activity of the thermal receptors in the skin. A sensitive and efficient biological entity will utilize such information to generate anticipatory thermoregulatory adjustments before any change in central temperature occurs, indeed to prevent such a change. Studies by Vendrik and Vos (1958), Hendler, Hardy, and Murgatroyd (1963), and Eijkman and Vendrik (1961) demonstrated plainly that both 3 and 10 cm microwaves could provide clear sensations of warmth at or near the skin surface. In addition, other attributes of the sensory experience became evident, such as a slower appearance than sensations aroused by infrared irradiation (Justesen, et al., in press) and a tendency to persist (i.e., not to
adapt-out as quickly as sensations produced by infrared). It is essential, therefore, that the effects of a control infrared irradiation be examined whenever possible before drawing any conclusion that the effects of microwave exposure in an animal subject are due to absorption in deep tissues rather than at the skin surface.

B. Basic data on thermoregulation in the squirrel monkey

Since 1968, investigators at the John B. Pierce Foundation Laboratory have made intensive study of the thermoregulatory responses of the squirrel monkey (Saimiri sciureus). Quantitative measurements have shown how this animal uses autonomic mechanisms of heat production (shivering and vasoconstriction) and heat loss (vasodilation and sweating) to maintain thermal balance when exposed to environmental temperatures within the range 10° to 40°C (Stitt and Hardy, 1971). This study illuminated the importance of vasomotion in the tail and extremities to the fine control of internal body temperature when the animals were in 25-35°C environments. A study of the control of heat loss through sweating (Nadel and Stitt, 1970) indicated that this mechanism is inadequate to prevent hyperthermia in environments warmer than 39°C. Intensive study of 6 monkeys detailed the complex interaction between peripheral and central temperatures in the control of autonomic thermoregulatory responses and emphasized the shift in vasomotor thresholds that can occur with only slight alterations in hypothalamic temperature (Lynch, Adair, and Adams, 1980).

The control of thermoregulatory behavior came under intense scrutiny in this program. Monkeys were trained to regulate the temperature of their immediate environment through a method of air temperature selection (Adair, Casby, and Stolwijk, 1970). Many trained monkeys then underwent a chronic surgical procedure during which sealed tubes (re-entrant tubes) were stereotaxically implanted in the medial preoptic/anterior hypothalamic area and often other areas of the brainstem. These tubes served as thermodomes (devices to alter local tissue temperature) when perfused with water of controlled temperature. They also served as convenient orifices for repeated insertion of thermocouples to measure brainstem temperature.

A variety of experimental procedures produced a great body of evidence pinpointing the medial preoptic area of the hypothalamus (PO/AH) as the major CNS thermosensitive site that controls behavioral as well as autonomic thermoregulatory responses, confirming many researches with a variety of other species. For this reason, this region of the brainstem is often referred to as the "central thermostat."

As a general rule, when the PO/AH is warmed, endotherms select cooler environments and core and skin temperatures fall. Conversely when the PO/AH is cooled, the animals select warmer environments and core and skin temperatures rise. The latency of changes in behavioral thermoregulatory responses to sudden PO/AH temperature change was found to be very short (often less than 15 sec). This behavioral measure was also found to be very sensitive; data from many squirrel monkeys showed that a highly localized PO/AH temperature change of the order of 0.2-0.3°C was sufficient to trigger an immediate and appropriate change in thermoregulatory behavior.

Behavioral responses to prolonged PO/AH heating and cooling differ, the latter showing some adaptation while the former does not. Responses to PO/AH cooling, while initially strong, tend to decrease over time. Presumably this occurs as a protection against hyperthermia, since the normal response to such stimulation is a rise in body temperature. On the other hand, local PO/AH heating is a persistent and powerful stimulus to any behavioral response that produces a decrease in environmental (skin) temperature. In our research to investigate the effects of microwaves on thermoregulation (see below), we have found that prolonged exposure to low intensity microwave fields results in the persistent selection of a cooler environment (Adair and Adams, 1980c). Heating of the PO/AH (as well as other brainstem loci) was strongly suspected as a concomitant of microwave exposure in this case.
THE PROBLEM

Electromagnetic radiation of the microwave frequency range is an environmental energy source that can, under appropriate conditions, produce heating of both peripheral and deep body tissues. Unlike infrared radiation, which does not penetrate the skin, microwaves can be absorbed below the skin surface in complex patterns that depend on many factors, including the physical characteristics of the radiation and the size and complexity of the biological medium. Thus, microwaves have the potential to alter both behavioral and autonomic thermoregulatory responses through direct stimulation of thermosensitive sites deep in the body, as well as those in the skin.

An elevated deep body temperature has often been recorded in experimental animals exposed to microwaves, particularly if the absorbed energy is high (e.g., Michaelson et al., 1961; Chernovetz et al., 1977; Phillips et al., 1975; Michaelson, 1974; de Lorge, 1976). Prior to the start of the present project, certain compensatory thermoregulatory adjustments had been measured, including thermal adaptation (Michaelson et al., 1961), reduced metabolic heat production (Phillips et al., 1975; Ho and Edwards, 1977), and microwave avoidance (Frey et al., 1975). Few investigations into the consequences of microwave exposure on thermoregulatory behavior had appeared in the literature. D'Andrea et al. (1978) reported that whiptail lizards placed in a 2450 MHz CW microwave gradient preferentially selected maximum microwave exposure and thereby achieved substantial body temperature regulation. However, systematic investigation of how microwaves may modify thermoregulatory behavior as well as the full range of autonomic thermoregulatory responses had not been undertaken for any single species. Such investigation for the squirrel monkey was undertaken in the research project described in the present report.

Individual experiments were designed to determine 1) the minimal power density of 2450 MHz CW microwaves (far field exposure, both whole-body and partial-body exposure) that will reliably alter thermoregulatory responses (heat production and heat loss, ongoing thermoregulatory behavior) of squirrel monkeys, 2) the nature of the alteration, and 3) the degree to which the observed effects may be attributed to heating of the body surface.
METHODS

A. Subjects

The subjects were adult male squirrel monkeys (Saimiri sciureus). They were housed individually in a colony room maintained at 24 ± 2°C. All animals were well-adapted to the restraining chair and each was highly trained to regulate environmental temperature behaviorally. The basic training procedures have been described by Adair et al. (1970).

B. Test Chamber and Response Measures

Figure 1 is a diagram of the basic elements of the behavioral test system. During the experiments the monkey was chair-restrained in the far field of a 15 dB standard gain horn antenna inside an anechoic chamber of interior dimensions 1.83 m x 1.83 m x 2.45 m. The distance from the front edge of the antenna to the monkey's center line was 1.85 m. The interior chamber walls were covered with 20 cm pyramidal microwave absorber (Advanced Absorber Products, Type AAP-8) to minimize reflections (<40 dB).

Figure 1. Schematic diagram (as viewed from above) of the basic elements in the test system. The receiving end of a 1.83 m x 1.83 m x 2.45 m anechoic chamber is ventilated by air of controlled temperature supplied by two air source chambers. Valves (V1 and V2) operated by animal's response cord (c) determine air flow. Air-source temperatures, sensed by thermistors (T1 and T2) are controlled by heat exchangers (E1 and E2) and coil heaters (H1 and H2). The monkey is located in the far field of the horn antenna. Use of a single air source provides an environment of constant temperature for the study of physiological thermoregulatory responses. See text for details of microwave source and other features.
A valve system ($V_1$ and $V_2$ in Fig. 1) allowed air from one of two closely-regulated sources to circulate through the anechoic space. The temperature-controlled space was restricted by a nylon sailcloth partition transparent to microwaves. The monkeys were highly trained to pull a response cord (c) to operate the valves, thereby selecting a preferred $T_a$ from two preset air temperatures 15° and 55°C. During behavioral experiments the animal was exposed to one air temperature and each response was reinforced by a 15-sec presentation of the other. The original incoming air temperature then automatically returned until the animal responded again. Under long-term exposure, all animals responded at a frequency that produced a preferred average air temperature of 35-36°C. During physiological experiments, the response cord was retracted and an environment of constant temperature (+ 0.5°C) was achieved through use of a single air source. Ambient temperature ($T_a$) was measured by a copper-constantan thermocouple suspended in the air outlet from the chamber and recorded continuously on a strip chart. This tracing afforded an accurate temporal record of the animal's behavioral responses as well as the actual airstream temperature.

In later stages of the project, the monkey was enclosed by a 30 cm x 51 cm x 33 cm styrofoam box through which the temperature-controlled airstream was directed. This arrangement improved the control of ambient temperature in the immediate vicinity of the animal, particularly during experiments involving partial-body exposures at high power densities, and during measurement of thermoregulatory sweating produced by microwave exposure.

During all experimental sessions, except the initial series to determine the microwave threshold for alteration of thermoregulatory behavior, rectal temperature ($T_{re}$) and four representative skin temperatures ($T_{sk}$) taken from the abdomen, tail, leg, and foot were sampled at 1-min intervals by an on-line data acquisition system. All temperatures were measured with 36-ga copper-constantan thermocouples having a reference junction in a bath of melting ice and water (Adair, 1976; Stitt and Hardy, 1971). All thermocouple junctions were extremely small and constructed in special configurations. Whenever they were used in the microwave field, the lead wires were shielded and held out of alignment with the E-vector insofar as was physically possible. Any thermocouple voltage showing evidence of electrical artifacts (i.e., abrupt voltage changes greater than 4 μV, equivalent to 0.1°C, correlated with microwave onset or offset) was discarded as an inadmissible datum. Ambient temperature at the monkey's location was assessed by a thermocouple affixed to the restraining chair support. A weighted mean skin temperature was calculated from the four skin temperatures by the relation (Stitt et al., 1971)

$$T_{sk} = 0.07 \ T_{foot} + 0.37 \ T_{leg} + 0.45 \ T_{abdomen} + 0.11 \ T_{tail}.$$  

This calculated value (together with individual skin temperatures, calculated metabolic heat production, rectal temperature, and other experimental parameters as desired) were displayed during the experiments on a Calcomp Model 565 Plotter.

All experimental sessions were conducted in the presence of a 73 dB SPL masking noise to guard against auditory cues to the presence or absence of microwaves. A closed-circuit TV monitoring system provided continuous surveillance of the monkey throughout each experiment.
Continuous microwaves of a single frequency, 2450 ± 25 MHz, were generated by a commercial source (Cober Model S2.5W) and fed to the antenna via standard waveguide components. The operating forward power range of the generator, 200 W to 2.5 KW, could be reduced to as little as 2 W by one of two directional couplers that diverted a portion of the forward power into a water-cooled dummy load.

Calibrations of the far field to determine uniformity, presence of standing waves, field perturbations produced by the restraining chair, hood, thermocouples, sweat boot, etc. were made using a Narda Model 8316B broadband isotropic radiation detector fitted with a Model 8323 field probe. At any far field location, the incident power density, measured in mW/cm² was a monotonically increasing function of generator forward power. In addition, at a constant forward power, the field was mapped at 5 cm intervals across a 70 cm x 70 cm plane that passed through the center of the restraining chair location orthogonal to the direction of propagation of the microwaves. This field map is shown in Figure 2 for a forward power of 250 W and indicates the location of the experimental animal in the field center. The microwave field in the central portion (40 cm x 40 cm) of this plane, encompassing the restraining chair location, showed a maximum nonuniformity of ~8% with chair absent. The Plexiglas restraining chair increased nonuniformity in the center of the field by an additional 5% and lowered overall field intensity by 30%. Addition of a 7.5 cm diameter Plexiglas hood to the neck plate of the chair (to collect expired air for analysis of oxygen consumption) added no significant nonuniformity to the field as measured at

**Figure 2.** Contour plot of field strength across a 70 cm x 70 cm plane orthogonal to the K vector centered on the antenna boresight 1.42 meters from the front edge of the antenna. Dashed outline shows location of 900 g monkey in the field. Forward power = 250 W.
the location of the monkey's torso or adjacent to the location of the monkey's head. Similarly, the presence of the shielded thermocouples (propped into place with styrofoam spacers, monkey absent) provided no significant field perturbations at the location of the monkey head, abdomen, or feet. A recent theoretical analysis by Ho (1978) concluded that large field perturbations produced by such devices may not be as important as other variables (e.g., uniformity of the incident microwave field, polarization, configuration and size of the animal) in their effect upon the energy absorbed by the animal.

The power densities specified in the whole-body experiments reported here (1 to 30 mW/cm²) were determined with the Narda field probe positioned in the center of the restraining chair at the location of the monkey's head. An assessment of the whole body energy absorption over this range was based upon measured temperature increments in three sizes of saline-filled cylindrical styrofoam phantom models (.75, 1.1, and 1.5 liter volumes). Tests on each model were conducted as follows: the saline-filled cylinder was placed at the animal's position inside the anechoic chamber and equilibrated for 16 hours to a circulating Ta of 35°C. As shown in Figure 3, four copper-constantan thermocouples were positioned on the central axis of the cylinder at different depths within the liquid but equidistant from the antenna. The emerging wire leads were aligned with the direction of propagation of the electromagnetic field (K-vector) and carefully shielded. A series of 10-min microwave exposures to 5, 10, 20, 30, and 40 mW/cm² were separated by minimum 2-hour re-stabilization periods. Sample temperature tracings (upper right in the figure) for the 40 mW/cm² exposure show that the temperature rise was related directly to the thermocouple depth within the unstirred liquid, reflecting thermal stratification. Also shown in Fig. 3 are mean temperature increments (and the range of measurements) in the 1.1 liter model as a function of incident power density. From these data, absorbed power or specific absorption rate (SAR) was calculated using the following relationship:

![Figure 3. Mean and range of temperature increments recorded by four thermocouples (TC) positioned as shown in a 1.1 liter saline-filled model as a function of power density for 10-min exposures to 2450 MHz CW microwaves. Insert (upper right) shows temperature tracings during 40 mW/cm² exposure. Ambient temperature (Ta) = 35°C.](image-url)
\[
\text{SAR (W/kg)} = \frac{\Delta T(°C)}{t(s)} \cdot 4.186 \left( \frac{\text{J.g}^{-1} \cdot \text{°C}^{-1}}{} \right) \cdot \frac{M(\text{g})}{M(\text{kg})}
\]

where \( \Delta T \) = temperature increment

\( t \) = duration of exposure

4.186 = specific heat of water

\( M \) = mass of the model.

The SARs calculated in this manner are shown in Figure 4 for the 3 sizes of saline-filled cylindrical models. Temperature increments were determined twice in the 0.75 liter model, once standing alone and once inserted into the restraining chair, to assess the effect of the chair on energy absorption. No differences were evident between the SARs determined from the two sets of measurements. The rate of energy absorption per mW/cm\(^2\) was nearly identical for all the models, as would be expected since they vary very little in mass.

Comparable dosimetric procedures can be performed using conscious squirrel monkeys, exposed to controlled microwave fields, in environments that are thermally neutral for the animal. Figure 5 summarizes the measured rectal temperature increase in several monkeys during 10-minute whole-body exposures to 2450 MHz CW microwaves as a function of power density. At ambient temperatures (Ta) of 34-35.5°C, the animal was fully vasodilated but not sweating and metabolic heat production was at the resting

![Figure 4. Specific absorption rate (W/kg) as a function of power density (mW/cm\(^2\)) calculated on the basis of mean temperature increments produced in 3 sizes of saline-filled cylindrical models by 10-min exposures to 2450 MHz CW microwaves. Models were equilibrated to an ambient temperature (Ta) of 35°C.](image)
Figure 5. Mean rectal temperature increase in conscious squirrel monkeys during 10-minute whole-body exposure to 2450 MHz CW microwaves at different power densities. Ambient temperature ($T_a$) was 34-35.5°C. The number of observations is noted above each point. Average body mass of the animals was 850 g. The point for 20 mW/cm$^2$ was not used in calculation of SAR since animals were becoming hyperthermic at this power density.

(lowest) level. The linear increase of rectal temperature with increasing power density (up to 16 mW/cm$^2$ only) was used to calculate SAR by the above method, assuming a tissue specific heat of 0.9 J·g$^{-1}$·°C$^{-1}$ and an average body mass of 850 g. A summary of these calculated SARs appears in Table I. The agreement between values is quite good. The SAR value of 0.145 W/kg·mW$^{-1}$·cm$^{-2}$ has been adopted for our whole-body exposure conditions as most representative of all the dosimetric determinations. It is this value that determines the function drawn in Figure 4.

### Table I

<table>
<thead>
<tr>
<th>Model</th>
<th>Mass (kg)</th>
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<th>SAR (W/kg·mW$^{-1}$·cm$^{-2}$)</th>
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<tr>
<td>Cylinder</td>
<td>0.75</td>
<td>35</td>
<td>0.153</td>
</tr>
<tr>
<td>Cylinder</td>
<td>1.1</td>
<td>35</td>
<td>0.135</td>
</tr>
<tr>
<td>Cylinder</td>
<td>1.5</td>
<td>35</td>
<td>0.151</td>
</tr>
<tr>
<td>Monkey</td>
<td>0.85</td>
<td>34-35.5</td>
<td>0.150</td>
</tr>
</tbody>
</table>
D. Infrared Source and Calibrations

In order to evaluate the contribution of skin heating alone, control exposures to infrared radiation were included in the experimental design of most of our experiments. Two T-3 infrared quartz lamps, 41 cm long, .64 cm in diameter, were located at the focus of 8 cm x 43 cm parabolic reflectors on individual metal bases. These were positioned in the anechoic chamber on the animal's side of the cloth partition 60 cm from the restraining chair location. Thus, the infrared radiation had the same directionality as the microwaves and impinged upon the same central portion of the anechoic space.

The energy (irradiance) from the lamps incident on a plane passing through the center of the monkeys' location was measured with a wide-angle radiometer (Hardy et al., 1952, p. 73-79) calibrated by a Bureau of Standards radiation lamp. A survey of the central portion (50 cm x 50 cm) of this plane encompassing the restraining chair location, showed insignificant (< 1%) nonuniformity for a constant lamp voltage. With the radiometer positioned at the location of the monkeys' head, the lamp voltage was determined (range = 35-65 V A.C.) that yielded incident intensities of 1-25 mW/cm², the same as the range of microwave intensities explored. Thus, the two types of radiation were equated on the basis of energy incident from the same direction upon a specific locus of a plane (orthogonal to the direction of propagation of the radiation) that passed through the center of the animal subject.

Two additional tests for the equality of infrared and microwave radiation were conducted. The first determined their effects upon a constant circulating air temperature. Temperature increments produced by 10-min exposures to equal intensities of the two types of electromagnetic radiation were measured in the chamber air outlet. The data in Table II show that these air temperature increments were nearly identical for a given intensity, ranging from a few tenths of a degree at 1 mW/cm² to 3.1°C at 25 mW/cm². Circulation air temperature was 35°C at the beginning of all exposures. Of course, during these tests, infrared heated the air directly whereas the microwaves were absorbed by the receiving (load) wall of the chamber which then heated the air through conduction and convection.

**TABLE II**

<table>
<thead>
<tr>
<th>Power Density - mW/cm²</th>
<th>1</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>20</th>
<th>25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infrared (T-3 quartz lamps)</td>
<td>0.2°</td>
<td>0.8°</td>
<td>1.4°</td>
<td>1.9°</td>
<td>2.6°</td>
<td>3.1°</td>
</tr>
<tr>
<td>2450 MHz CW Microwaves</td>
<td>0.1°</td>
<td>0.6°</td>
<td>1.3°</td>
<td>1.9°</td>
<td>2.5°</td>
<td>3.1°</td>
</tr>
</tbody>
</table>

The second test confirmed that rectal temperature increments in a conscious monkey equilibrated to an ambient temperature of 33°C were the same during 10-minute exposures to equal infrared and microwave intensities, although skin temperatures were elevated more under infrared than under microwaves. Table III shows that the rectal temperature increments were nearly identical for a given intensity of either type of radiation.
TABLE III

Rectal temperature increments in a conscious monkey, equilibrated to 33°C air temperature, produced by 10-min exposures to infrared or microwave radiation of equal intensity.

<table>
<thead>
<tr>
<th>Power Density - mW/cm²</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infrared (T-3 quartz lamps)</td>
<td>0.1°</td>
<td>0.3°</td>
<td>0.5°</td>
<td>0.6°</td>
</tr>
<tr>
<td>2450 MHz CW Microwaves</td>
<td>0.0°</td>
<td>0.3°</td>
<td>0.5°</td>
<td>0.7°</td>
</tr>
</tbody>
</table>

In some of the experiments described below (e.g., those in which the test animal was enclosed by a styrofoam box) it was difficult or impossible to utilize infrared irradiation as a control stimulus. In many of these cases, gradual increases in the temperature of the circulating air, comparable to that produced by microwave exposure, served as control stimuli. These were produced manually by the experimenter.
EFFECTS OF WHOLE-BODY MICROWAVE EXPOSURE

A. Thermoregulatory Behavior

The primary purpose of these experiments was to determine the minimal power density of 2450 MHz CW microwaves during brief exposures that will reliably alter normal ongoing thermoregulatory behavior, the nature of the alteration, and the degree to which this effect may be attributed to skin heating. Secondary objectives were 1) to assess the impact of suprathreshold intensities on the ability of the animals to thermoregulate behaviorally, and 2) to explore the effects of longer exposure durations.

1. Ten-minute Microwave Exposures

a. Microwave Threshold Experiments

Three adult male squirrel monkeys served as subjects. Prior to microwave exposure, five four-hour experimental sessions of behavioral thermoregulation were conducted on each monkey. The mean ambient temperature selected by one monkey (taken in 10-minute time blocks) is plotted in Figure 6 against experimental time (squares connected by dashed lines). This animal was exposed to 55° and each response produced a 15-sec reinforcement of 15°C. After an initial "warm-up" period the ambient temperature he selected was 34-36°C, falling off only slightly as the 4-hour session progressed. Also shown in the figure (circles - solid lines) are the mean results from 5 experimental sessions when the same monkey was exposed to microwaves of increasing power density (1, 2, 4, 6, 8, and 10 mW/cm²). Each 10-minute period of microwave exposure was followed by a 10-min period of no microwaves. The data show that the ambient temperature selected by the animal decreased in orderly fashion beginning at about 6 mW/cm². Statistical analysis revealed that the ambient temperature selected under 6 and 8 mW/cm² was significantly lower than the control condition at the .05 level of confidence; the difference at 10 mW/cm² (2° below the ambient temperature the monkey normally selected) was highly significant.

Figure 6. Mean ambient temperature selected by one monkey during five 4-hour control experiments (---■--) and five 4-hour experiments in which the animal was exposed to 10-min periods of 2450 MHz CW microwaves of increasing power density (——○——).
The change in the behavior of a second animal under microwave exposure is illustrated in Figure 7, which presents a segment of the strip-chart record of temperature changes in the air outlet from the anechoic chamber taken during one experiment. This monkey worked in 15°C air for 55°C reinforcements. No microwaves were present during minutes 213-220, when the animal worked for 2.1 ambient temperature changes per minute, and thereby produced an average air temperature of 34.6°C. At 220 minutes, the microwaves were turned on at a power density of 10 mW/cm². Within the first minutes of the exposure, response rate decreased as the animal lowered the average ambient temperature to 31.5°C.

A summary of results for the third monkey appears in Figure 8, which depicts the mean ambient temperature selected as a function of microwave power density together with data from control runs. This animal always worked in an ambient temperature of 15°C for 15-sec reinforcements of 55°C, the reverse conditions to those for the monkey whose data were shown in Figure 6. The slightly higher threshold for a change in normal thermoregulatory behavior under microwave exposure seen here probably reflects individual differences between animals. It may also partially reflect the relative sensitivity of the behavioral measure, that is an animal working in the cold for warmth vs an animal working in the heat for cooling. Thus, despite some slight variability in threshold, the major finding is that brief exposures to 2450 MHz CW microwaves at a power density of 6-8 mW/cm² can stimulate squirrel monkeys to select a lower environmental temperature. Based on our dosimetry, the 8 mW/cm² level represents a SAR of 1.2 W/kg, a value comparable to about 20% of the resting metabolic heat production of the squirrel monkey (Stitt and Hardy, 1971; Lynch, 1976).

In an effort to determine how much of the response change could be attributed to skin heating, the experimental protocol was varied so that infrared radiation of equal incident energy was substituted for microwaves. Two T-3 quartz lamps were
Figure 8. Mean ambient temperature (+ 1 SEM) selected by one monkey as a function of incident power density for control (---) and experimental (---) sessions.

Figure 9. Mean ambient temperature (+ 1 SEM) selected by one monkey as a function of incident energy for control, infrared, and 2450 MHz microwave experiments.
positioned inside the chamber so that the radiation would have the same directionality as the microwaves had. The energy from the lamps was calibrated with a Hardy radiometer as detailed above. The voltage to the lamps was determined that would yield incident energies of 1, 2, 4, 6, 8, and 10 mW/cm². Five experimental sessions were then conducted on each animal using the same temporal protocol as before.

Figure 9 summarizes the results of the infrared experiments for one monkey and compares them to the results reported above for the control and microwave experiments. Once again, the mean ambient temperature selected over 10-minute periods is plotted as a function of incident energy (or the comparable control period) for the control experiments, infrared exposure, and microwave exposure. The significant reduction in ambient temperature selected at the higher microwave energies is evident as before, but there is no significant reduction from the control level when the monkey was exposed to infrared. Identical results were obtained for all animals.

Both microwaves and infrared radiation raised the temperature of the circulating chamber air (as well as the monkey) during the 10-minute exposure periods. Infrared heated the air directly; microwave energy was absorbed by the receiving wall which then transferred heat to the air by conduction and convection. Table II (above) showed that the air temperature increments were nearly the same for the two types of radiation. During infrared exposure, the monkey adjusted his response rate to compensate for this air temperature increment so that he selected the same environment as he would if there were no infrared stimulus present. However, the air temperature reduction seen under microwaves was in addition to this compensatory reduction and represented a deliberate response of the animal to microwaves per se. These results suggest strongly that some thermosensitive structure (or structures) other than those in the skin were being stimulated by the microwaves and were triggering changes in the effector response system that regulates the body temperature.

**b. Effects of Higher Power Densities**

The protocol was identical to that of the microwave threshold series except that higher power densities were explored (8, 10, 12, 15, 18, and 22 mW/cm²). Tₐ was measured in the chamber air outlet; body temperatures were monitored continuously using carefully shielded 36 ga copper-constantan thermocouples. Five experimental sessions were conducted on each of two animals.

The range of power densities explored in this second (suprathreshold) microwave series overlapped that of the first series and extended upward to nearly three times the previously determined threshold power density. The mean ambient temperature selected by one monkey (taken in 10-minute time blocks) during the five sessions of the suprathreshold series is plotted in Figure 10 (circles - solid lines) as a function of experimental time. Also shown are the rectal (squares) and weighted mean skin temperatures (triangles) measured during these experiments. This animal, whose data for the earlier series appeared in Figure 6, again worked in warm air for cool air reinforcements. The effects of brief exposure to 2450 MHz microwaves of higher power density were even more pronounced than at near-threshold levels. By working vigorously, the monkey selected a lower and lower ambient temperature as power density increased from 8 to 22 mW/cm². At 22 mW/cm², the selected temperature was a full 4°C below the normally-preferred 35°C. During each 10-minute interval between microwave exposures, the monkey allowed the environment to warm up by greatly reducing his response rate.
Figure 10. Rectal temperature ($T_{rc}$), mean skin temperature ($T_{sk}$), and ambient temperature ($T_a$) selected by one monkey (taken in 10-min time blocks) as a function of experimental time. Points represent means of 5 experiments in which animal was exposed to 10-min periods of 2450 MHz CW microwaves of increasing power density (8, 10, 12, 15, 18, and 22 mW/cm$^2$). Monkey worked in 55°C air for 15°C reinforcement.

Figure 11. Mean ambient temperature selected by one monkey as a function of power density and SAR (W/kg) derived from dosimetric studies on saline-filled models. Threshold series (Part 1) and suprathreshold series (Part 2) are combined in this graph.
Figure 10 demonstrates further that the changes in the monkey's thermoregulatory behavior were geared toward the maintenance of stable skin and deep body temperatures. The initial "warm-up" behavior in the first hour raised the mean skin temperature to about 38° so that all skin areas were vasodilated but sweating was not initiated (Stitt and Hardy, 1971; Lynch and Adair, 1978; Lynch, Adair, and Adams, 1980). Deep body temperature, assessed rectally, stabilized at about 39°. There was no evidence of significant changes in either skin or rectal temperature during the 10-min periods of exposure to microwaves, even at a power density of 22 mW/cm². By lowering environmental temperature behaviorally, the monkey regulated his body temperatures precisely at the preferred stabilized levels. Results from a second monkey replicated those shown in Figure 10.

Figure 11 combines the data from the two microwave series (i.e., Figs. 6 and 10) in terms of the ambient temperature selected by the animal as a function of microwave intensity. Although the baseline control data are not presented here, this monkey always selected a Ta of 35-36°C when no microwave stimulus was present. The point marked "THRESHOLD" represents the power density at which the animal selected a significantly lower Ta than during the control sessions (cf. Fig. 6). The plotted points for both microwave series are seen to fall upon the same function; indeed the mean values for 8 and 10 mW/cm² from the two series are nearly identical. As power density increased, mean selected Ta continued to fall with only a slight indication of leveling off. Included in the figure is a second abscissa, indicating SAR in W/kg, based upon our dosimetry. The SAR at 22 mW/cm² power density is ~3.2 W/kg, roughly 2/3 of the resting metabolic heat production of the squirrel monkey (Stitt and Hardy, 1971).

c. Implications of the Findings

Three major results are evident from the experiments reported above:

1) Brief whole-body exposures to 2450 MHz CW microwaves at a power density of 6-8 mW/cm² and above will stimulate squirrel monkeys to select a lower ambient temperature.

2) Exposure to infrared radiation of equivalent power density does not produce this effect.

3) Exposure to 2450 MHz CW microwaves at power densities up to 2.5 times the threshold level provokes an increasingly lower selected ambient temperature. Body temperatures are thereby regulated at stable levels.

Rapid regulated changes in thermoregulatory effector responses of the type measured in these experiments are normally triggered by a thermal event occurring in one or more thermosensitive tissue sites. Therefore we are led to conclude that the microwave exposures caused some thermal disturbance, however small, somewhere in the body. Although it is impossible to characterize explicitly how the two types of electromagnetic energy (microwaves and infrared) are absorbed and dealt with by the body, some tentative generalizations should be offered.

Both microwaves and infrared radiation raised the temperature of the circulating chamber air in the animal's vicinity (Table II). The temperature increments measured in the air outlet were essentially the same for the two types of radiation at a given power density. During infrared exposure the monkey adjusted his response rate to compensate for the air temperature increment so that he selected the same environment
as he would if there had been no infrared stimulus present (Figure 9). However, the air temperature reduction seen during microwave exposure was in addition to this compensatory reduction and, as noted above, represented a deliberate response of the animal to microwaves per se. We know that microwaves are directly absorbed by deep body tissues and that 12.4 cm microwaves (2450 MHz) can be expected to penetrate ∼2 cm below the skin surface (Durney et al., 1978). In a 1 kg squirrel monkey, this energy could be deposited in a variety of internal sites known to be thermosensitive (e.g., anterior hypothalamus, cortex, thalamus, medulla, midbrain reticular formation, spinal cord, and deep abdominal structures); the same would not be true in humans. The experimental results described above suggest that changes in thermoregulatory behavior triggered by absorbed microwaves resulted from stimulation of internal thermoreceptors, whereas responses to infrared radiation resulted primarily from stimulation of receptors in the skin. Since the whole body was exposed to the microwave field, it is reasonable to suspect involvement of multiple thermosensitive sites (rather than, e.g., the anterior hypothalamic thermoregulatory center alone) in the handling of body heating by microwaves.

2. Effects of Exposure Duration

All of the experiments described above involved 10-minute microwave exposures. Additional experiments were conducted that were designed to answer two questions: 1) Will a subthreshold microwave intensity eventually alter thermoregulatory behavior if exposure duration is increased? 2) Will the environmental temperature reduction the animal produces when exposed to a suprathreshold microwave intensity for 10 minutes adapt or moderate if exposure duration is increased (even up to 2 1/2 hours)?

a. Protocols and Results

Two squirrel monkeys served as subjects; both had served in the threshold experiments detailed above. During all experiments we measured rectal temperature, four representative skin temperatures (abdomen, tail, leg, foot) and the air temperature selected by the animal. The power densities used were 4, 10, and 20 mW/cm², representing whole-body SARs of 0.6, 1.5, and 3.0 W/kg respectively. The 4 mW/cm² intensity was subthreshold, while the other two were well above threshold for both animals.

Two series of experiments were conducted. In the first series, microwave exposure duration during experimental sessions ranged from 5 to 25 minutes. The temporal protocol was always the same. Each session began with a 2-hour baseline period of behavioral thermoregulation (during which the microwave field was absent), followed by 5 microwave exposures of increasing duration, 5, 10, 15, 20, and 25 minutes. Each microwave exposure was followed by a microwave-free period of like duration: thus, 5-min on, 5-min off, 10-min on, 10-min off, etc. Six experimental sessions were conducted on one monkey at 4 mW/cm², and five experimental sessions on each of the two monkeys at 10 mW/cm². The lower power density was below the threshold for alteration of thermoregulatory behavior, whereas the higher was well above threshold for both animals (Adair and Adams, 1980b). Five 4-hour sessions of behavioral thermoregulation in which no microwaves were present constituted control data for each monkey.

In the second series, all experiments began with a 90-minute period of behavioral thermoregulation to allow stabilization of behavior and all body temperatures. This was followed by a 2.5-hour microwave exposure to a given power density, 10 or 20 mW/cm². A 10-min post-exposure period of behavioral thermoregulation terminated the session. Five experimental sessions were conducted on each monkey at each power density.
For the first series, the data for each animal were analyzed separately. Means of rectal and weighted mean skin temperatures together with the air temperature selected by the animal were calculated across sessions in 10-min time bins for the first 120 minutes and in 5-minute time bins thereafter. Figure 12 shows the results of experiments in which one monkey was exposed to 4 mW/cm² for different durations.

Figure 12. Mean ambient temperature selected by one monkey exposed to 4 mW/cm² (SAR = 0.6 W/kg) for different durations and compared with control data. Skin (---■---) and rectal (---▲---) temperatures show minor fluctuations unrelated to microwave exposure.

The solid circles-solid lines delineate the mean air temperature selected by the animal over the course of the 4 1/2-hour sessions. Except for the initial "warm-up" overshoot, this function is little different from the control condition (open circles - dashed lines) in which no microwaves were present. The minor fluctuations of rectal (triangles) and skin (squares) temperatures (measured during the microwave experiments) appear to be correlated with fluctuations in the air temperature selected by the animal rather than with the presence or absence of the microwave field. On the basis of these data, it would appear that a subthreshold microwave intensity will fail to alter thermoregulatory behavior no matter how long it lasts. In other words, microwaves exhibit no temporal summation.

This picture changes dramatically when the microwave intensity is well above threshold, as shown in Figure 13. With the exception of the initial 5-min exposure, each microwave exposure stimulates the animal to lower the chamber temperature 2-3°C. The normally-preferred 35°C environment is reinstated during each ensuing period of no microwaves. By these dramatic behavioral alterations, skin and deep body temperatures sustain only minor perturbations. There is some tendency for this animal to select an increasingly cooler environment the longer the microwave exposure lasts, although the second monkey did not show this effect. Of more interest perhaps is the fact that the initial 5-min microwave presentation provoked little behavioral change, whereas the initial 5 minutes of subsequent (longer) exposures often provoked a dramatic reduction in chamber air temperature.
Figure 13. Mean ambient temperature selected by one monkey exposed to 10 mW/cm$^2$ (SAR = 1.5 W/kg) for different durations. Skin (---) and rectal (---) temperatures remain relatively constant despite dramatic air temperature adjustments made by the animal.

Figure 14. Mean air temperature (+ 1 SEM) selected by two monkeys exposed to 2450 MHz CW microwaves at 10 mW/cm$^2$ as a function of exposure duration. Rectal and mean skin temperatures achieved are also shown in the figure. For meaning of , see text.
These results are clearer in Figure 14 which summarizes, for both animals, the mean air temperature selected (+1 SEM) during the 10 mW/cm² exposures as a function of exposure duration. The points for 10-25 minutes each differ significantly from the control condition at the .01 level of confidence (Student's t-test) but do not differ statistically from each other. The downward trend of selected air temperature with increased exposure duration, while not significant statistically, suggests a slightly increased thermal burden in the body over time that is countered by selection of an ever-cooler environment, although this result was attributable primarily to the behavior of one monkey. The two upper functions in Figure 14 demonstrate that the skin and deep body temperatures remained stable at the normal level as a result of efficient behavioral responding.

The most striking aspect of Figure 14 is the minimal behavioral effect during the initial 5-min exposure period exhibited by both animals; the mean air temperature selected does not differ significantly from the control (no microwave) condition. For comparison purposes, the mean air temperature selected in the initial 5 minutes of the four subsequent (longer) microwave exposures was calculated across the 10 experimental sessions. The value is indicated in Figure 14 by an asterisk. This point (*) falls on the extrapolated linear function determined by the points for the 10-25 minute durations, implying that a 5-minute exposure to a 10 mW/cm² will stimulate a vigorous and appropriate change in the behavior that regulates the environmental temperature.

In these experiments, the order in which the different durations was presented was always the same, i.e., the 5-min duration was always presented first. Order and duration effects were thus confounded. However, other observations in our laboratory tend to confirm a minimal impact on thermoregulatory behavior of the initial microwave presentation within a series. Figure 15 presents the averaged results of 10 experiments on the same 2 monkeys when they were exposed repeatedly to the same microwave field, 10 mW/cm². These experimental sessions were conducted in identical fashion.
to those described above except that exposure duration was always the same, 10 minutes. The figure shows that the air temperature selected during the initial microwave presentation fell within the range of air temperatures preferred during most of the pre-exposure baseline period and was at least 1°C warmer than that selected during the 4 subsequent microwave periods. The minimal behavioral change when the microwave field was first presented resulted in elevated skin and deep body temperatures, ameliorated somewhat during the following microwave exposures by vigorous behavioral action.

In the second experimental series, that involving exposure durations of 2 1/2 hours, the data for each animal were again analyzed separately. Means of rectal and weighted mean skin temperatures, together with the ambient temperature selected, were calculated in 10-min time bins across the 5 experimental sessions. Representative results for both animals are presented in panels A and B of Figure 16. In general, the initial stabilization of air and body temperatures was more rapid in these experiments than in those described above because the animals were enclosed by a styrofoam box and yielded more precise control over a much smaller volume of air.

The 35.5°C ambient temperature preferred by one monkey during the baseline period was lowered to about 34°C during the 10 minutes following the onset of the 10 mW/cm² microwave field (Panel A). This adjusted ambient temperature was maintained without significant alteration for the duration of the microwave exposure. When the field was extinguished, the animal worked hard to warm the environment up again. These deliberate adjustments in thermoregulatory behavior served to maintain cutaneous and deep body temperatures at the normal (baseline) level.

Analogous behavioral changes appear in more exaggerated form in Panel B. In this case, the second monkey underwent 2 1/2 hour exposures to a field strength of 20 mW/cm². This level represents an absorbed energy dose rate of 3.0 W/kg or approximately 60% of the resting metabolic heat production of the squirrel monkey. At microwave onset the animal quickly lowered the air temperature by increasing his response rate for cool air reinforcement. Within the first half hour, a preferred air temperature 3°C below the baseline level had been achieved. This level was maintained, without significant alteration, for the duration of the microwave exposure. Throughout this period, slight fluctuations occurred in the skin temperature that were correlated primarily with oscillations in selected air temperature. However, deep body temperature remained unchanged. Once again, extinction of the microwave field provoked a dramatic behavioral restoration of the normally-preferred thermal environment.

The data presented in Figure 16 are representative of the responses of both animals to both 10 and 20 mW/cm² power densities during 2 1/2 hour exposures. Under 10 mW/cm², air temperature was reduced by about 1.5°C; under 20 mW/cm², it was reduced by about 3°C. In all experiments, skin temperature variation was insignificant and rectal temperature remained stable at the normal level.

b. Implications of the Findings: the "Warm-up" Effect

In two series of experiments, we investigated the potential for adaptation of thermoregulatory behavior during prolonged (up to 2 1/2 hours) exposure to a microwave field. Three major results are evident from these experiments:

1) Whole-body exposure to 2450 MHz CW microwaves, at a power density (4 mW/cm²) below that which will normally stimulate squirrel monkeys to select a cooler environment, did not alter thermoregulatory behavior no matter how long it lasted;
Figure 16. Mean ambient temperature ($T_a$) selected by each of two monkeys exposed to 2450 MHz CW microwaves for 2 1/2 hours at a power density of 10 mW/cm$^2$ (panel A) or 20 mW/cm$^2$ (panel B). Skin (- - - - - -) and rectal (⋯ ⋯ ⋯ ⋯ ⋯ ) temperatures are also shown. Monkey in panel A worked in cool air for warm air reinforcement; monkey in panel B the reverse.
2) Exposure to higher power densities, 10 and 20 mW/cm², stimulated the monkeys to select ambient temperatures 1.5 and 3.0°C cooler than control levels, thereby insuring stability of the body temperatures; but

3) Except for the first microwave presentation of a series or the early minutes of a single long exposure, the length of time the field was on had no significant bearing upon the air temperature selected or the resulting body temperatures achieved thereby.

A power density of 5 mW/cm² and above for the rat (Stern et al., 1979) and 6-8 mW/cm² and above for the squirrel monkey (Adair and Adams, 1980b) will provoke reliable alterations in thermoregulatory behavior, i.e., selection of a cooler environment by the irradiated animal. These measured thresholds for both species represent a whole body energy absorption (SAR) of 1.0-1.5 W/kg. Presumably, these levels deposit sufficient thermalizing energy in the animal's body to heat specific CNS thermoregulatory detectors whose altered output provides for the initiation of cooling responses. Thus, behavioral selection of cooler surroundings serves to increase the thermal gradient from body core to skin, thereby facilitating heat loss to the environment. Microwave intensities below these threshold levels also deposit thermalizing energy in the body but of insufficient quantity to influence thermoregulatory behavior. Even prolonged exposure to such a subthreshold microwave field (Figure 12) had no behavioral consequence; the insignificant additional energy must be within the noise level of the thermoregulatory system.

Microwave fields of suprathreshold intensity (e.g., 10 mW/cm²) provoke changes in thermoregulatory behavior whose purpose is the efficient regulation of deep body temperature. Rapidity of response adjustment at microwave onset together with behavioral consistency thereafter are crucial for achievement of this regulation. The rectal temperatures of the animals undergoing repetitive 10 mW/cm² exposures for different durations (Figure 13) varied by a maximum of +0.3°C from the stabilized level, little more than the variation seen in Figure 12. Considering the coarse nature of the control afforded the animals by the contingencies of the experiment, these variations in core temperature during microwave exposure seem rather modest. In the steady state (Figure 16), rectal temperature varied almost not at all.

However, it is clear that a core temperature rise always occurs on those occasions when the animal fails to counter microwave onset with rapid environmental cooling. Increases in both rectal and skin temperatures were observed during the first of a series of 10 mW/cm² microwave exposures (Figure 13) accompanying a minimal behavioral change. Analysis of this result (Figure 14) and supporting data (Figure 15) suggest that the initial brief exposure of a series may serve to prime or sensitize the system in some way so that the impact of subsequent exposures may be optimized. It should be emphasized that this is not a learning phenomenon; both experimental animals had had extensive experience with microwave exposure prior to the present study. A similar "warm up" in the efficiency of escape responding by rats from a visually-cued microwave field occurred at the beginning of daily test sessions following training in a recent study by Levinson et al. (in press). This phenomenon may be akin to the "evoked thermocolonic response" (Justesen, 1974) that is characterized as the metabolic arousal of an animal by a benign or noxious stimulus change in the environment (Justesen and Bruce-Wolfe, 1977). Stimuli need not be thermogenic in order to initiate such arousal; its occurrence has been demonstrated in rats given 0.5 sec mild foot shock or undergoing daily gentling procedures (Justesen et al., 1974).
In the present case, however, the thermogenic property of the microwave stimulus may have been useful to the animals since 2450 MHz microwaves may be absorbed deeper in the body than at the location of cutaneous thermodetectors. Given that a sensation of warmth is the normal precursor of effective changes in thermoregulatory behavior, what could be the sequence of underlying physiological events that energized the system? Two alternatives come to mind. First, thermalization occurring in deep tissues could be dissipated by increased regional blood flow (Kritikos et al., 1980; Stolwijk, 1980), thereby bringing warmer blood to peripheral tissues and stimulating cutaneous thermodetectors. In addition, localized SARs higher than the whole-body average will occur in the limbs and tail of monkeys undergoing whole-body exposure (Guy et al., 1978), enhancing the possibility of direct but delayed thermal stimulation of cutaneous thermodetectors.

On the other hand, thermalization occurring in deep tissues could build up over time, supersede a threshold increment, and alter the firing rate of deep body thermodetectors such as those in the hypothalamus, thereby initiating a change in thermoregulatory effector response (Adair and Adams, 1980). A rise in hypothalamic temperature ($\Delta T_{hy}$) of 0.65°C has been measured in rats exposed to 2800 MHz pulsed microwaves at 10 mW/cm² (Brainard et al., 1978). Recent observations in our laboratory have demonstrated a $\Delta T_{hy}$ of 0.45°C in anaesthetized squirrel monkeys exposed to 2450 MHz CW microwaves for 10 minutes to a power density of 10 mW/cm² (see below). A $\Delta T_{hy}$ of only 0.3°C, produced by an implanted thermode, will stimulate a squirrel monkey to cool its environment behaviorally (Adair, Casby and Stolwijk, 1970).

Sufficient data are not yet available, however, for more than speculation on which of these two alternatives (or both) may be correct.

B. Autonomic Thermoregulatory Responses

1. Introduction: Thermoregulatory Profile of the Squirrel Monkey

Warm-blooded organisms, called endotherms, are able to maintain a stable internal body temperature in the face of rather wide fluctuations in the temperature of their environment. This is accomplished by fine adjustments in appropriate autonomic response systems, through which the body gains or loses heat, acting in concert with behavioral responses that provide a hospitable microclimate whenever possible. The particular autonomic response that may be operative at a given time is dictated primarily by the environmental temperature: thus, animals shiver in the cold and pant or sweat in the heat, but not the reverse.

Using the standard heat balance equation and methods of partitional calorimetry (Hardy and Stolwijk, 1966), it is possible to quantify the steady-state contribution of each autonomic response system to the total thermoregulatory response across a selected range of environmental temperatures. The result is a thermoregulatory profile for the species in question. Figure 17 shows an example of such a profile for the squirrel monkey, Saimiri sciureus (Stitt and Hardy, 1971), the experimental animal we use to investigate the effects of microwave exposure on thermoregulatory function. In cold environments, up to an ambient temperature ($T_a$) of 26.5°C, thermoregulation is accomplished by adjustments in metabolic heat production ($M$). In general, the lower the $T_a$, the higher the $M$, with the response systems for heat loss remaining at constant low levels. At $T_a = 26.5°C$, designated the lower critical temperature (LCT), resting heat production is minimal and fine control of the body temperature is taken over by the peripheral vasomotor system (Lynch and Adair, 1978; Lynch, Adair, and Adams, 1980). Peripheral vessels in first the tail and then the extremities vasodilate, bringing warm blood from the body core to the surface, greatly increasing
conductance (K). Peripheral vasodilation is complete and thermoregulatory sweating is initiated at $T_a = 36^\circ C$, designated the upper critical temperature (UCT). In the squirrel monkey, however, heat loss through evaporation is limited and, since $M$ tends to increase in warm environments, the animal may quickly become hyperthermic at $T_a > 39^\circ C$.

It is clear from inspection of Fig. 17 that if the animal is exposed to a microwave field, the particular response system that may be altered by absorbed thermalizing energy will be directly related to the particular $T_a$ at which the exposure is made. Our strategy, then, was to isolate each response system by controlling the environmental temperature, and examine each to quantify the effects on thermoregulatory function of both brief and prolonged exposure to 2450 MHz CW microwaves. We have measured the reduction of metabolic heat production in cold environments, the stimulation of peripheral vasodilation of the tail in thermoneutral environments, and the initiation of thermoregulatory sweating from the foot in warm environments. In all cases, the environmental temperature was held constant; the animal had no control over it as in the behavioral thermoregulation experiments described above.

2. Heat Loss via the Peripheral Vasomotor System

In thermally neutral environments, the peripheral vasomotor response of endothermic species functions continuously to provide fine control of the body temperature. Physiological regulation of heat flow into or out of the body depends to a large extent upon autonomically controlled changes in the volume, rate, and distribution of blood supplied to the skin. The stimulus to constriction or dilation of cutaneous vessels is often peripheral, as when localized or whole-body changes occur in the temperature of the skin. The stimulus can also originate centrally, in the absence of peripheral thermal events, when the deep body temperature rises, as during
exercise. Rapid changes in peripheral vasomotor state have been produced in a variety of experimental animals by altering the temperature of the anterior hypothalamus via stereotaxically-implanted thermode devices. In such experiments, dilation or constriction in highly vasoactive skin areas such as ears, tail, or extremities is often indexed by abrupt increases or decreases in local skin temperature. In the experiments described here, vasomotor activity in cutaneous tail veins was mirrored by abrupt changes in tail skin temperature during brief exposures to 2450 MHz CW microwaves.

Three squirrel monkeys served as subjects. They were chair restrained in the anechoic chamber in environments of constant temperature. Rectal and four skin temperatures (abdomen, tail, leg, foot) were monitored continuously. After minimum two-hour equilibration to a cool environment of constant temperature (range 22 to 26.5°C) at which tail and extremities were fully vasoconstricted, the monkeys underwent 5-minute exposures to 2450 ± 25 MHz CW microwaves. Microwave power density, initially at a low level of 2.5 - 4 mW/cm², was increased at each successive exposure until a criterion tail vasodilation occurred. This criterion was earmarked by an abrupt and rapid rise in tail skin temperature that persisted after termination of the microwave exposure. Figure 18A shows an example of criterion tail vasodilation in one monkey equilibrated to an ambient temperature of 25°C. The microwave power density producing the response in this case was 10 mW/cm², which represents a whole-body energy absorption rate of 1.5 W/kg or roughly 25% of the resting metabolic heat production of the animal.

Microwave power densities below that which initiated tail vasodilation often produced increases in the temperature of the air or of skin areas other than the tail. Control experiments (example in Fig. 18B) demonstrated that tail vasodilation was not initiated passively as a result of air temperature elevations. This monkey, also equilibrated to a 25°C environment, failed to exhibit any change in peripheral vasomotor state when exposed to infrared radiation of equivalent power density to the microwaves. Taken together, these results suggest that thermosensitive structures other than those residing in the skin may be responsible for altered thermoregulatory responses during microwave exposure.

The microwave power density required to stimulate criterion tail vasodilation was directly related to the environmental temperature in which the monkey was restrained. Figure 19A summarizes the data from 16 experiments on 3 animals at discrete ambient temperatures that range downward from 26.5°C, the temperature at which the tail vessels of a sedentary monkey may dilate spontaneously (Stitt and Hardy, 1971; Lynch, Adair and Adams, 1980). The plotted points describe a linear relationship which reveals that to initiate a criterion response, an increase of 3 to 4 mW/cm² in microwave power density is required for every 1°C reduction of ambient temperature. A second abscissa relates the data to absorbed microwave energy based upon our dosimetry. Thus, in a 23°C environment, when the animal's metabolic heat production is elevated 2.5 - 3 W/kg above the resting level, microwave energy deposited at a rate equal to this metabolic elevation will vasodilate the tail. Stability of the internal body temperature is thereby assured within the limits possible through changes in vasomotor state (uppermost tracing in Figure 18A).

Thresholds for initiation of other thermoregulatory effector processes, such as shivering and panting, have been demonstrated to vary with both the ambient (skin) temperature and the local temperature either of the preoptic hypothalamus (Brück and Wunnenberg, 1970; Cabanac et al., 1965) or of other thermosensitive sites such as the
Figure 18. Representative experiments showing effects of 5-min exposures (hatched bars) to 2450 MHz CW microwaves (A) or infrared radiation (B) on skin and deep body temperatures of squirrel monkeys equilibrated to a 25°C environment. (A) Exposure to microwaves of increasing power density (4, 6, 8, 10 mW/cm²) were separated by 15-min recovery periods. Criterion vasodilation of tail skin, evidenced by rapid and persistent rise in tail temperature, occurred at 10 mW/cm², preventing a rise in deep body temperature. (B) Control exposures to infrared radiation of increasing incident power density (4, 6, 8, 10, 12 mW/cm²) failed to induce tail vasodilation despite significant elevations in ambient temperature.
spinal cord (Jessen, 1977) as controlled by implanted thermodes. The form of such functions often resembles the relation presented in Figure 19A. Earlier research in our laboratory determined how tail and foot vasodilation can be triggered by heating thermodes implanted in the hypothalamus of squirrel monkeys restrained in cool environments (Lynch, Adair, and Adams, 1980). Some of these results appear in Figure 19B in a form that facilitates direct comparison with the adjacent microwave data. The striking resemblance lends further support to the hypothesis that low-intensity microwaves, absorbed in the vicinity of thermosensitive neural tissue in the hypothalamus and elsewhere (for example, posterior hypothalamus, midbrain, spinal cord, deep viscera), can provoke immediate and dramatic changes in thermoregulatory response systems.

3. Metabolic Heat Production

When behavioral alternatives are unavailable or inefficient, endotherms respond to cold environments by elevating their metabolic heat production (M). In general, below some critical ambient temperature that is characteristic for each species (cf. Figure 17), the colder the environment the higher the rate of body heat production (Hardy, 1953-54). The net effect of such metabolic adjustments is the maintenance of a stable internal body temperature.

As an aid to the conservation of endogenous energy stores, cold-exposed organisms avail themselves of exogenous thermal energy whenever possible. Radiant, conductive, or convective sources that transfer heat to the body surface are conventional modes by which endogenous heat production may be minimized. Due to its potential for heating not only superficial but deep body tissues, microwave radiation could be expected to reduce the endogenous heat production of cold-exposed endotherms.
The basic fact that microwave exposure can lower M has been previously demonstrated for the mouse by Ho and Edwards (1977) and for the rat by Phillips et al. (1975). In neither study were the thermoregulatory consequences of the absorbed microwave energy evaluated while the exposures were in progress. We designed several different experiments to determine how whole-body exposure to 2450 MHz CW microwaves can influence M and body temperatures of squirrel monkeys restrained in selected cold environments. A particular goal was to assess both the latency and the efficiency of thermoregulatory response changes to microwaves and determine to what extent they may be attributed to variations in environmental temperature alone.

a. Metabolic adjustments during brief microwave exposure

Three squirrel monkeys served as subjects in the experiments. They were chair restrained in the anechoic chamber in environments of constant temperature. These were 15°, 20°, and 25°C (1 monkey only at this temperature). Rectal and four representative skin temperatures were monitored continuously. A Plexiglas hood over the monkey's head, attached to the neck plate of the restraining chair, collected the expired air which was drawn outside the chamber through teflon tubing. Oxygen deficit was measured downstream by a Beckman F-3 Paramagnetic Oxygen Analyzer, which sampled the passing airstream at the rate of 0.3 L/min. Metabolic heat production was calculated from oxygen consumption assuming a constant RQ of 0.83.

After 90-minute equilibration (see Figure 20), the monkeys underwent 10-minute exposures to 2450 MHz CW microwaves of increasing power density (2.5, 4, 6, 8, and 10 mW/cm²), separated by sufficient time to allow re-stabilization of all measured parameters. Three such experiments were conducted on each animal at each environmental temperature. Control experiments, in which 10-minute air temperature increments (comparable to those produced during the microwave exposures) were substituted for the microwaves, probed the effects of brief periods of skin warming on metabolic heat production.

![Figure 20. Representative experiment on one monkey equilibrated to an ambient temperature of 20°C to determine effects on metabolic heat production (M) of 10-minute exposures to 2450 MHz CW microwaves of increasing power density. Also shown are rectal, mean skin, and tail skin temperatures.](image-url)
Figure 21. Metabolic heat production of 1 monkey during 10-minute exposures to 2450 MHz CW microwaves. Also shown are the 5-minute periods immediately preceding and following microwave exposure. The parameter is microwave power density.

Figure 22. Metabolic heat production of 1 monkey during 10-minute ambient temperature excursions identical to those that occur during microwave exposure. Also shown are the 5-minute periods immediately preceding and following the ambient temperature transients. The parameter is the ambient temperature elevation ($\Delta T_a$).
The experiment depicted in Figure 20 shows many features that were common to all conducted. In both 15° and 20°C environments, M was reliably reduced (p < .05) below the stabilized baseline level by 10-minute microwave exposures to a power density of 4 mW/cm² (2 monkeys) or 6 mW/cm² (1 monkey) and above. On some occasions, for some animals, a power density of 2.5 mW/cm² also stimulated reliable M reductions. These M reductions serve to regulate the internal body temperature at a characteristic stable level.

The magnitude of the M reduction was directly related to microwave intensity. This is shown in Figure 21 which superimposes the mean responses of one monkey to different power densities upon the same time scale. It includes the 5-minute time segment immediately preceding the microwave exposure as well as the first 5 minutes following the exposure. The reduction in heat production with microwave onset was always rapid in this animal, more gradual with the other two. However, extinction of the microwave field always provoked an abrupt recovery of M, sometimes resulting in an overshoot that was also related to microwave intensity.

In contrast to these results, no M adjustments occurred during the control experiments that were related in any direct way to the magnitude of the ambient (or skin) temperature increases produced experimentally. Sample data for one monkey, displayed similarly to Figure 21, are shown in Figure 22. The figure shows that even a gradual 1.1°C ambient temperature rise over a 10-minute period is insufficient to influence the M level reliably. These results, comparable to the infrared control experiments reported earlier (Figures 9 and 18), provide additional evidence that some thermosensitive structure (or structures) other than those in the skin are being stimulated by absorbed microwaves to trigger changes in the autonomic effector response system that regulates the body temperature.

The responses to brief microwave exposure in both 15° and 20°C environments tended to be of similar magnitude for a given power density, although they were superimposed upon a higher stabilized level of heat production in the colder environment. Metabolic adjustments in the 25°C environment were somewhat smaller, primarily because heat production is only slightly elevated above the resting level in this nearly-neutral ambient temperature.

Figure 23 summarizes the data for all animals at the two cooler environmental temperatures. It depicts the mean reduction in metabolic heat production during the second 5 minutes of the 10-minute microwave exposure relative to the last 5 minutes of stabilization immediately preceding that exposure. The points are plotted against SAR. A linear relationship between the magnitude of the M response change and absorbed microwave energy is clearly evident, as is the primary dependence of that response change upon intensity rather than upon environmental temperature.

However, close inspection of Figure 23 reveals that the M reduction (in W/kg) is always greater than the energy absorbed (in W/kg). The ratio between the two is approximately 2.5:1. Assuming the validity of our dosimetry, one might expect to see a sharp reduction in rectal temperature under these circumstances, as the animal appears to be losing 2.5 times as much energy as he is absorbing. Of course, 10-minute microwave exposures provide only transient response shifts that may be expected to moderate in the steady state. Furthermore, the time constant for deep body temperature change is very long (Nadel and Horvath, 1970); it would be surprising to see significant changes in rectal temperature within a 10-minute period.
Figure 23. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures to 2450 MHz CW microwaves as a function of SAR (W/kg). The parameter is environmental temperature ($T_a$).

Figure 24. Stripchart tracings showing changes in metabolic heat production during 4 10-minute exposures to 10 mW/cm$^2$. Response latency to microwave onset is 60-90 seconds; that to microwave termination is 45 seconds.
b. Response latencies to microwave onset and termination

The metabolic adjustments occurring in the presence of brief microwave exposure appeared to be closely bound to the onset and termination of the microwave stimulus. (See Figure 20) This remarkable temporal contiguity contrasts, for example, with the behavioral thermoregulatory adjustment whose onset may often be delayed many minutes after microwave onset and which may often develop gradually as the microwave exposure continues (cf. Adair and Adams, 1980b, Figure 5). We attempted to measure the M response latencies to microwave onset and termination in one monkey exposed repeatedly to 10-minute periods of 10 mW/cm² while restrained in a 20°C environment. The output voltage from the Beckman F-3 oxygen analyzer was recorded continuously on a strip chart.

Figure 24 displays a sample of four individual tracings from one experiment superimposed on the same time scale (drawn to compensate for the response time of the O₂ analyzer). The individual tracings are remarkably similar, indicating the high reproducibility of this response to a specific microwave exposure. The M onset latency, the more difficult of the two to quantify, appears to fall between 60 and 90 seconds. However, the M reaction to microwave termination is well underway by 45 seconds. These results suggest that if a reliable and highly sensitive physiological indicator of microwave effects is needed, a very good candidate would be metabolic heat production in cold or cool environments.

c. Metabolic adjustments under 90-minute microwave exposures

The results displayed in Figure 23 provided an input-output discrepancy in need of resolution. We thus undertook an additional series of experiments to examine the effects of longer duration (i.e., 90-minute) microwave exposure on heat production and body temperature in the cold. A 90-minute period is sufficiently long for the animal to reach a thermoregulatory steady state. A typical example of the results obtained in this type of experiment is given in Figure 25. It contrasts mean data
from three control experiments (open symbols) in which no microwaves were present
with three microwave experiments (closed symbols) in which the same monkey was ex-
posed, (following a 90-minute stabilization period) for 90 minutes to 2450 MHz CW
radiation at 8 mW/cm² (SAR = 1.2 W/kg). Ambient temperature was always 20°C. Each
plotted point represents the mean for the preceding 5 minutes of the animal’s rectal
temperature (triangles), weighted mean skin temperature (squares), and metabolic
heat production (circles). The strong metabolic reduction at microwave onset gradu-
ally moderated over the 90-minute exposure period to insure continual stability
of the internal body temperature. For the final 30 minutes of the microwave exposure,
average heat production was 1.2 W/kg below the average level at the same time period
in the control experiments. Table IV summarizes the results of comparable experi-
ments on three animals at 4 different power densities. In nearly every case, the
metabolic reduction (in W/kg), when the animal achieves a thermoregulatory steady
state during prolonged microwave exposure, is the same as the SAR (in W/kg) derived
from other dosimetric methods (cf. Section C above). The results of the present ex-
periments suggest an important new approach for determining whole-body SAR, at least
for low-to-moderate power densities that can be effectively handled by the thermo-
regulatory system.

### TABLE IV

Mean steady-state levels of metabolic heat production (M) during minutes 150-180
of control experiments and of experiments in which animals were exposed for 90 minutes
to 2450 MHz CW microwaves at the power densities indicated. Specific absorption
rate (SAR) is derived from dosimetry on saline-filled models.

<table>
<thead>
<tr>
<th>Power Density (mW/cm²)</th>
<th>Subject</th>
<th>Mass (g)</th>
<th>M(W/kg)</th>
<th>M(W/kg)</th>
<th>ΔM (W/kg)</th>
<th>SAR (W/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Lancelot</td>
<td>1050</td>
<td>10.9</td>
<td>9.9</td>
<td>-1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>8</td>
<td>Paul</td>
<td>1105</td>
<td>11.0</td>
<td>9.8</td>
<td>-1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>10</td>
<td>Gandalf</td>
<td>950</td>
<td>12.5</td>
<td>10.5</td>
<td>-2.0</td>
<td>1.5</td>
</tr>
<tr>
<td>12</td>
<td>Gandalf</td>
<td>950</td>
<td>11.7</td>
<td>9.8</td>
<td>-1.9</td>
<td>1.8</td>
</tr>
</tbody>
</table>

When squirrel monkeys are restrained in cold environments, body temperature is
regulated by an increase in metabolic heat production. These experiments have demon-
strated that either brief or prolonged whole-body exposure to a homogeneous 2450 MHz
microwave field, at a power density of 4-6 mW/cm² and above, will cause a reduction
of this elevated heat production by an amount directly related to the microwave energy
absorbed. This response can occur within 60-90 seconds of microwave onset and per-
sists, with some adaptation, as long as the microwave field is present. Termination
of the microwave exposure stimulates an immediate (i.e., within 45 sec) rebound of
heat production, often to a level higher than the pre-exposure baseline. These dra-
matic alterations in metabolic heat production serve to regulate the internal body
temperature with precision at the normal level. As in other experiments described in this report, the absence of response change during control conditions (convective heating in this case) provides additional evidence for a non-cutaneous thermosensitive site as the major location of microwave action on the thermoregulatory system.

4. Heat Loss via Thermoregulatory Sweating

In warm environments, above the upper critical temperature (cf. Figure 17), endotherms must depend upon specialized heat loss mechanisms to achieve regulation of the body temperature. Some species, such as rodents, salivate copiously and spread the saliva over the body surface to increase evaporative heat loss from the skin. Others (e.g., dogs and cats) increase respiratory frequency dramatically (panting) thereby eliminating large body heat loads from the respiratory tract. Still others (e.g., horses, certain non-human primates, and man) activate sweat glands over some or all of the body surface, thereby increasing heat lost from the skin by evaporation. In man, the sweating rate has been shown to depend not only upon the temperature of the environment (skin) but also upon the internal body temperature (Nadel et al., 1971).

Since microwaves have the potential to heat tissues deep in the body, they would be expected to trigger the characteristic heat loss mechanism of any endotherm in a warm environment whether that mechanism be salivation, panting, or sweating.

Sweating is the primary heat loss response of the squirrel monkey in warm environments (Nadel and Stitt, 1970). However, in this species, sweat glands are restricted to the palms and soles, a distribution which severely limits the evaporative capability of these animals. In the natural environment, they rely upon behavioral adjustments (seeking shade, draping themselves over tree limbs, copious drinking) to supplement their limited autonomic mechanisms of heat loss. Complicating the picture is a tendency for heat production to increase in warm environments (Figure 17). Thus, squirrel monkeys may quickly become hyperthermic when the environmental temperature reaches 39°C (normal body temperature) and above.

We designed a series of experiments to determine the minimal power density of brief (10-min) exposures to 2450 MHz CW microwaves that will reliably initiate thermoregulatory sweating from the foot of monkeys restrained in environmental temperatures just below the upper critical temperature (36°C) at which sweating occurs naturally. Microwave exposures of longer duration (90-min) were employed to investigate the efficiency of the sweating response in regulating the body temperature. Since sweating in this species has an emotional as well as thermoregulatory component, it is essential to assess changes in metabolic heat production that may accompany experimentally-induced changes in sweating. Therefore, oxygen consumption was always measured during these experiments.

a. Sweating initiated by brief microwave exposures

Four squirrel monkeys served as subjects. A 30 cm x 51 cm x 33 cm styrofoam box enclosed the restrained monkey inside the anechoic chamber, as shown in Figure 26, thus affording precise control over the air temperature in the animal's immediate vicinity. Rectal and skin temperatures were monitored continuously. Oxygen consumption was measured by methods described above.

Thermoregulatory sweating from the foot was measured by a dewpoint sensing device controlled by a Peltier module, developed at the Pierce Foundation Laboratory. The monkey wore a L-shaped Plexiglas boot with the sole of the foot resting on a nylon mesh support. Chamber air was drawn through the boot and thence outside the chamber.
Figure 26. Schematic diagram (as viewed from above) of anechoic chamber, microwave generator and transmission system, oxygen analyzer, dewpoint sensor, and system for air temperature control. Valves (V) permit source air to circulate through styrofoam box in which monkey sits. Source air temperature, sensed by thermistor (T), is controlled by heat exchanger (E) and coil heaters (H).

where the dewpoint temperature (Tdp) was measured and recorded continuously. From Tdp, the vapor pressure of water in the sampled air was calculated using Antoine's equation (Wood, 1970):

\[ P_{H_2O} = e^{a-b(T_{dp}+c)} \]

where:
- \( P_{H_2O} \) = water vapor pressure (mm Hg)
- \( a = 18.6686 \)
- \( b = 4030.18 \) empirical constants for water.
- \( c = 235 \)

The water evaporated from the sole, or sweating rate (\( \dot{M}_w \)) was then calculated from a modified gas equation:

\[ \dot{M}_w = \frac{(M.W.) \ (\Delta P_{H_2O}) \ AF}{R \cdot T_a} \text{ g/min} \]
where:  
M.W. = molecular weight of water  
$\Delta P_{H2O}$ = the difference in water vapor pressure in air before and after evaporation of foot sweat  
AF = air flow (L/min)  
R = gas constant 62.396  
T$_a$ = air temperature (°K)

The experimental procedure was designed to determine the power density of 10-minute microwave exposures that will reliably initiate thermoregulatory sweating in monkeys restrained in $T_a$'s just below that at which sweating normally occurs. After a minimal 90-minute equilibration period (see Figure 27) for stabilization of all measured parameters, the animal was exposed to 10-minute periods of 2450 MHz CW microwaves of increasing power density (e.g., 4, 6, 8, 10 mW/cm$^2$). These exposures were separated by sufficient time for re-equilibration of the animal's responses. Each animal was tested several times at $T_a$'s ranging from 31.5° to 35°C.

Figure 27 presents a representative experimental session on one monkey conducted at a $T_a$ of 35°C. After a 30-minute calibration period to determine instrument baselines (O2 meter, dewpoint sensor), the animal was introduced into the chamber and instrumented. During the 100-minute equilibration period, vasodilation of the extremities occurred, evidenced by an abrupt rise in foot skin temperature. Mean skin temperature stabilized at $\sim$ 3°C above $T_a$, indicating complete peripheral vasodilation. Internal body temperature was regulated by a reduction of heat production to the resting level. No sweating from the foot was evident. The monkey then underwent 10-minute microwave exposures of increasing power density (in this case 4, 6, 8, and 10 mW/cm$^2$) to determine that power density at which a reliable increase in thermoregulatory sweating occurred. The figure shows a reliable, regulated rise in the dewpoint temperature of foot capsule air at 6 mW/cm$^2$ and above. The sweating responses to 8 and 10 mW/cm$^2$ were even more rapid and dramatic, although the response to the higher power density failed to prevent a slight rise in internal body temperature ($T_{re}$).

The power density required to initiate thermoregulatory sweating from the foot was directly related to the environmental temperature in which the monkey was restrained. Figure 28 summarizes the data from 30 experiments on four animals at discrete ambient temperatures that range downward from 36°C, the temperature at which sweating in a sedentary monkey may occur spontaneously. The plotted points appear to describe two functions that are roughly linear with power density or SAR: two monkeys (open symbols) showing minimal sweating and relative insensitivity to microwaves and two monkeys (closed symbols) showing generous sweating and greater sensitivity to microwaves. We have classified the former as "inefficient sweaters" and the latter as "efficient sweaters" although their ability to regulate internal body temperature during microwave exposure in warm environments appeared to be about equal. "Efficient sweaters" tended to have a somewhat higher level of resting heat production that "inefficient sweaters." In any case, for all animals, at a $T_a$ just below the UCT, thermoregulatory sweating was reliably initiated by a 10-minute microwave exposure at a power density of 6-8 mW/cm$^2$ (SAR 1.1 W/kg). This represents roughly 70 percent of the resting metabolic heat production of the squirrel monkey (Stitt and Hardy, 1971; Lynch, 1976) and is comparable to microwave thresholds we have determined for other thermoregulatory responses (see Figure 35 below).

The magnitude of the change in sweating rate at a given $T_a$ is directly related to the intensity of the microwave field. Clearly evident in Figure 27, this finding is quantified for two of the four monkeys in Figure 28 (an "efficient sweater") and Figure 30 (an "inefficient sweater"). At each $T_a$ (ranging from 30.5° to 35.5°C),
Figure 27. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 35°C to determine effects on metabolic heat production (M) and on dewpoint temperature of foot capsule air (T_dp) of 10-minute exposures to 2450 MHz CW microwaves of increasing power density. Also shown are rectal (T_re), weighted mean skin (T_s) and foot skin (T_foot) temperatures.

Figure 28. Threshold function for initiation of thermoregulatory sweating from the foot of squirrel monkeys by 10-minute whole body exposures to 2450 MHz CW microwaves. Each data point represents the least microwave power density, or absorbed microwave energy, required to induce criterion increase in sweating rate at the ambient temperature indicated. Open symbols (△ ○) indicate "inefficient sweaters"; closed symbols (● ☐) indicate "efficient sweaters".
Figure 29. Change in foot sweating rate from baseline level produced by 10-minute microwave exposures of different power densities. The parameter is the ambient temperature ($T_a$) at which the experiment was conducted. Data from one monkey, an "efficient sweater".

Figure 30. Change in foot sweating rate from baseline level produced by 10-min microwave exposures of different power densities. The parameter is the ambient temperature ($T_a$) at which the experiment was conducted. Data from one monkey, an "inefficient sweater".
plotted point represents the change in sweating rate measured from t=0 to t=10 min of each 10-minute microwave exposure plotted against the power density of that exposure. The points for any given T_a are best described by a straight line the slope of which varies in regular fashion with T_a. Thus, at any T_a, at or below 35.5°C, the higher the power density the greater the increase in sweating rate of the monkey under microwave exposure, but this increase becomes less and less in cooler and cooler environments, i.e., as the skin temperature falls. Indeed, there is a strong suggestion that if T_a were to fall much below 32°C, it would be very difficult to initiate thermoregulatory sweating by microwave exposure. These cooler environments are well within the thermoneutral zone (Figure 17) and the autonomic heat loss mechanism that would be activated would be vasomotor, not sudomotor under these conditions.

The relationships displayed in Figures 29 and 30 are qualitatively similar to human data reported by Nadel, Bullard, and Stolwijk (1971). The local sweat rate of exercising humans increases linearly as body core temperature rises during exercise in a given T_a. But a higher core temperature is necessary to initiate sweating as skin temperature falls and the rate of increase in sweating during exercise is lower and lower in cooler and cooler environments. This suggests that in the squirrel monkey the rate of thermoregulatory sweating initiated by microwave exposure depends not only upon the ambient (skin) temperature but also upon the temperature of the body core as it is directly increased by absorbed microwave energy.

b. Thermoregulatory sweating during prolonged microwave exposures

To probe the efficiency of sweating to regulate the body temperature during microwave exposure in warm environments, another series of experiments was conducted. Three of the four monkeys from the threshold series served as subjects, one "efficient sweater" and the two "inefficient sweaters". The experimental conditions were chosen so as to minimize thermal distress and thus the potential for hyperthermia while ensuring a robust sweating response from all the animals. Following a 90-minute period of equilibration to a 34-34.5°C environment, the animals underwent a 90-minute whole-body microwave exposure to a power density of 10 mW/cm² (SAR = 1.5 W/kg). This intensity was well above threshold for the "efficient sweater" and close to the threshold level for the other two animals (cf. Figure 28). Body temperatures and oxygen consumption were measured in the usual manner together with sweating rate from the foot. Three such sessions were conducted on each animal.

A typical example of the results obtained in this type of experiment, for an animal classified as an "inefficient sweater", is shown in Figure 31. Contrasted are mean data from three control experiments (open symbols) in which no microwaves were present with the three microwave experiments (closed symbols) in which the same monkey was exposed for 90 minutes to microwaves at a power density of 10 mW/cm². Each plotted point represents the mean for the preceding 5 minutes of the animal's rectal temperature (triangles), weighted mean skin temperature (squares), metabolic heat production (circles), and sweating rate (crosses). Within 10-15 minutes after the beginning of the microwave exposure, sweating rate began to rise above the control level, continuing to rise slowly for the first hour and then leveling off. No change occurred in metabolic heat production which remained low and stable. That the sweating response was insufficient to prevent a rise in internal body temperature is evidenced by the 1.1°C rise in T_re over the course of the exposure. The rise in weighted mean skin temperature accompanying the T_re rise brings the temperature of the body surface close to the normal regulated internal body temperature of the animal, a condition conducive to uncontrollable hyperthermia. Results such as these point out the importance of specifying and controlling the environmental temperature in which an experimental
Figure 31. Mean sweating rate, metabolic heat production, skin and rectal temperature (taken in 5-min time bins) of one monkey equilibrated to an ambient temperature ($T_a$) of 34.5°C during 3 control experiments (open symbols) and 3 experiments involving 90-minute exposure to 2450 MHz CW microwaves at a power density of 10 mW/cm² (closed symbols). Animal classified as an "inefficient sweater".

Figure 32. Sweating rate, metabolic heat production, skin and rectal temperatures (taken in 5-min time bins) of one monkey equilibrated to an ambient temperature ($T_a$) of 34°C during a single experiment involving a 90-minute exposure to 2450 MHz CW microwaves at a power density of 4 mW/cm². Animal classified as an "efficient sweater".
Figure 33. Mean sweating rate, metabolic heat production, skin and rectal temperatures (taken in 5-min time bins) of one monkey equilibrated to an ambient temperature ($T_a$) of 34°C during 3 experiments involving 90-minute exposure to 2450 MHz CW microwaves at a power density of 10 mW/cm². Animal is classified as an "inefficient sweater".

Figure 34. Mean sweating rate, metabolic heat production, skin and rectal temperatures (taken in 5-min time bins) of one monkey equilibrated to an ambient temperature ($T_a$) of 34°C during 3 experiments involving 90-minute exposure to 2450 MHz CW microwaves at a power density of 10 mW/cm². Animal classified as an "efficient sweater".
animal undergoes microwave exposure: a microwave intensity that may be thermally benign to an animal in the cold becomes lethal rapidly if administered in a moderately warm environment. Indeed, pilot data (Figure 32) indicate that in warm environments, when the animal has no behavioral options, even power densities that are subthreshold for brief exposures will produce slow but sufficient heating of the body core to ultimately trigger some response change. In this case, the response was emotional resulting in an abrupt increase of metabolic heat production and initiation of emotional sweating. Future studies might well probe deeper into the synergistic effects of warm environments and microwave exposure on autonomic thermoregulatory responses as well as general ceiling effects in a wide range of environmental temperatures. It is as important to know the maximal intensities that can be tolerated as the minimal intensities that will reliably initiate response change.

Figures 33 and 34 present the results of 90-min exposures to 10 mW/cm² on the sweating response of the other two monkeys, one an "inefficient sweater", the other an "efficient sweater". It is clear that even the tremendous increase in sweating rate exhibited by the latter animal (Figure 34) fails to prevent a rise in rectal temperature of 0.7°C during the microwave exposure. Some of the sweating must be classified as emotional in this highly-sensitive animal owing to the concomitant increase in metabolic heat production. Both of these factors, limited sweating capabilities and increased heat production in warm environments, conspire against effective thermoregulation during microwave exposure in the heat in this species, the only possible defect in the predictive capability of the squirrel monkey thermoregulatory model we are developing in this research project.

C. Thresholds for Alteration of Thermoregulatory Responses: A Summary and Possible Mechanisms

Figure 35 summarizes experimentally-determined microwave thresholds (5-10 min exposures) for the alteration of four types of thermoregulatory responses in terms of both power density and specific absorption rate. A power density of 6-8 mW/cm² will reliably stimulate squirrel monkeys to lower the chamber air temperature behaviorally (Adair and Adams, 1980b). The same intensity will initiate criterion vasodilation of the tail veins in monkeys restrained in a 26°C environment, a Ta just below the LCT (Adair and Adams, 1980a). A slightly lower power density (4-6 mW/cm²) will reliably lower the metabolic heat production of monkeys restrained in 15°C and 20°C environments (Adair, 1980), while a power density of 6-8 mW/cm² will initiate thermoregulatory sweating from the foot of monkeys restrained in a 35°C environment, a Ta just below the UCT (present report). The whole-body energy absorption in all cases represents 15-20% of the resting metabolic heat production of the squirrel monkey (Stitt and Hardy, 1971; Fuller, Sulzman, and Moore-Ede, 1979; Lynch, 1976). The remarkable similarity of these thresholds suggests strongly that the same configuration of deep body thermosensitive sites is being stimulated by absorbed microwaves (during whole-body exposure) to provide the neural impetus for response change. Further, the entire thermoregulatory armament, autonomic response systems for heat production and heat loss as well as thermoregulatory behavior, that functions over a wide range of environmental temperatures (Figure 17), appears equally sensitive in coping with exogenous deep body heating.

Over the last 100 years, a variety of experimental procedures in many species has produced a great body of evidence pinpointing the medial preoptic area of the hypothalamus (PO/AH) as the major CNS thermosensitive site that controls behavioral as well as autonomic thermoregulatory responses. For this reason, this region of the brainstem is often referred to as the "central thermostat." Our own researches
Figure 35. Specific absorption rate (W/kg) as a function of power density (mW/cm²) calculated on the basis of mean temperature increments produced in 3 sizes of saline-filled cylindrical styrofoam models. Dashed line at 5 W/kg indicates average resting metabolic heat production of squirrel monkey in thermoneutral zone. Arrows indicate microwave thresholds for alteration of thermoregulatory behavior, initiation of tail vasodilation, reduction of metabolic heat production in the cold, and initiation of thermoregulatory sweating in warm environments.

Figure 36. Measurements of temperature increases in the medial preoptic area (PO/AH) and midbrain reticular formation (MB) using a VITEK probe inserted in stereotactically implanted nylon or teflon re-entrant tubes. Anaesthetized monkeys underwent 10-minute whole-body exposures to 2450 MHz CW microwaves at different power densities. Animals equilibrated to 35-36°C environment had internal body temperature within normal range (39.0-40.0°C).
with the squirrel monkey (see for example, Adair, Casby, and Stolwijk, 1970; Adair, 1971; Adair and Rawson, 1974) have consistently shown that a highly localized PO/AH temperature change of the order of 0.2-0.3°C, produced by an implanted thermode device, is sufficient to trigger an immediate and appropriate change in thermoregulatory responses. We have recently determined that a temperature rise of this magnitude in the PO/AH area of the squirrel monkey brain accompanies a 10-minute microwave exposure at a power density of 6-8 mW/cm².

As part of the present project, we have developed an implantable non-metallic re-entrant tube system that can be used as an orifice for inserting a VITEK (Bowman, 1976) probe to measure local brain tissue temperature. Closed tubes, machined from nylon or teflon rod, have an inside diameter of 1.1 mm and a wall thickness of 0.005". An inner rod system holds the tube securely for accurate placement during stereotaxic surgery and is then removed from the preparation. Nylon screws and dental acrylic tie the implant to the skull.

Two animals (acute preparations) have been implanted with several of these tubes in both the PO/AH and the midbrain reticular formation (MB). Post-surgically, the anaesthetized animals were placed in the restraining chair inside the anechoic chamber and exposed to a variety of microwave power densities. Pertinent data appear in Figure 36 which shows the temperature increase in the PO/AH or the MB measured with the VITEK probe (inserted in the implanted tubes) as a function of the power density of 10-minute exposures to 2450 MHz CW microwaves. Also indicated in the figure is the local PO/AH tissue temperature rise (produced via thermode) that is usually necessary to initiate changes in thermoregulatory responses. The data provide strong evidence that a power density of 6-8 mW/cm² produces a PO/AH temperature rise of 0.2-0.3°C in a squirrel monkey restrained in our exposure chamber.

The data in Figure 36 confirm earlier measurements we had made of temperature increases under controlled microwave exposures in tissue-equivalent spheres the approximate size of a squirrel monkey head. Spherical molds were cut from blocks of

![Figure 37. Temperature excursion in 20 seconds along the z-axis of a 3.0 cm radius homogeneous brain-equivalent sphere exposed to 2450 MHz CW microwaves, 70 mW/cm² in the far field. Predicted excursions for a 2.3 cm radius sphere are shown for comparison.](image-url)
closed-cell styrofoam and were filled with either brain- or muscle-equivalent material according to the method of Guy (1971). A 1 mm diameter track to guide the VITEK probe was molded into each sphere along the central axis at the time of construction. The styrofoam blocks were affixed to the Plexiglas restraining chair in the usual position of the monkey's head and the chair was placed in its usual position on the stand in the anechoic chamber. Ambient temperature was held at 25 ± 1°C during microwave exposure.

The VITEK probe was inserted along the axis at selected locations and the temperature was recorded during 20 sec exposures to a power density of 70 mW/cm² (method of Burr and Krupp, 1980). Two exposures were made at each probe position and the results averaged. Enough time was allowed between exposures for temperature equilibration before the next exposure was begun. Some of the results were compared with predictions made for a simulated squirrel monkey head by the thermal-response model developed by Burr et al. (1980).

Experimental values from two series of measurements along the K vector of a 6 cm diameter sphere are compared in Figure 37 with the theoretical prediction. Agreement is generally good, the experimental values being somewhat lower than predicted in the center of the sphere. This could be due to any of three factors: 1) the presence of the restraining chair, not accounted for in the model; 2) the slightly larger size of the sphere over the model; and 3) the finite time (averaging 3 seconds) to bring the generator up to the power required to produce 70 mW/cm².

Theoretical analyses (Kritikos and Schwan, 1979) have also suggested that internal hotspots of this magnitude could occur in the brain under our experimental conditions. Thus, we must conclude that whatever temperature changes may occur elsewhere in the body during whole-body microwave exposure, the local tissue temperature rise in the "central thermostat" of the brainstem is probably sufficient to trigger changes in thermoregulatory effector responses at a threshold power density of 6-8 mW/cm².
EFFECTS OF PARTIAL-BODY MICROWAVE EXPOSURE

It is tempting to try to assess the role of hypothalamic and other brainstem thermosensitive sites vs other deep body thermosensitive sites in the handling of microwave body heating by selectively exposing individual body parts to the microwave field. If it were not for diffraction effects that are characteristic of all electromagnetic radiation, it might be possible to screen one part of the body cleanly and completely while exposing the remainder to the microwaves. In practice, however, any small aperture (of dimensions > λ/2) in a screen interposed between the microwave source and the biological target will itself become a new source of radiation. A target placed close to such an aperture will be effectively in the near field of the new source.

These considerations notwithstanding, we have attempted to expose only part of the squirrel monkey to 2450 MHz microwaves while screening the rest of the body as effectively as possible. Although we have made extensive field measurements at the animal's location and performed dosimetric procedures whenever applicable, the partial body experiments reported here must be regarded as preliminary in nature and the results only suggestive.

Two series of experiments were conducted. In the first series the head only was exposed, the rest of the body being screened; in the second, the head was screened and the remainder of the body (trunk, extremities, and tail) was exposed. In each series, both behavioral thermoregulatory responses and metabolic heat production in cold environments were monitored during 10-minute exposures to the microwave field. The latter response was selected from the several autonomic thermoregulatory responses described above because of its sensitivity, consistency, and ease of assessment.

A. Exposing only the Head to the Microwave Field

A three-panel screen was constructed of plywood and covered with 20 cm pyramidal microwave absorber (Advanced Absorber Products, Type AAP-8). A square aperture, 30 cm x 30 cm, was cut in the center panel of the screen so that when it was interposed between the antenna and the monkey (as diagrammed in Figure 38), only the monkey's head received substantial radiation, the rest of the body being screened fairly successfully, given the limits imposed by diffraction effects.

To determine the extent of field nonuniformity behind the screen, the field was mapped at 5 cm intervals across a 61 cm x 86 cm plane passing through the center of the restraining chair location orthogonal to the direction of propagation of the incident microwave (K vector). Figure 39 represents a contour map of power density in this plane when generator forward power was constant. Although fairly steep power density gradients occur behind the opening in the screen, the region occupied by the monkey's head (dashed circle in the figure) shows relatively low nonuniformity and the trunk (which lies below the opening) is always exposed to at least 10 dB less than the head.

One series of dosimetric measurements determined the difference in temperature rise within a 6-cm diameter brain-equivalent sphere with the 3-panel screen present and absent. The results shown in Figure 40 depict the rate of temperature rise (°C/min) along the Z-axis (K vector) under the two conditions when exposed to 70 mW/cm² for 20 sec. Although the functions have similar shape, the presence of the screen reduces the peak temperature rise by about 16%. The power density was measured at the sphere
Figure 38. Schematic diagram (as viewed from above) of the basic elements of the test system, showing the location of the 3-panel screen interposed between the antenna and the animal to screen part of the body from the microwave field. For other elements, see legend to Figure 1.

Figure 39. Contour map of field intensity across a 61 cm x 86 cm plane orthogonal to the K vector centered on the antenna boresight 1.42 meters from the front edge of the antenna. Three-panel screen with 30 cm x 30 cm aperture in place. Circle shows location of monkey head in the plane of measurement. Forward power = 0.5 kW. Contour interval = 5 mW/cm².
Figure 40. Rate of temperature rise along the Z-axis of a 3.0 cm radius homogeneous brain-equivalent sphere exposed to 2450 MHz CW microwaves, 70 mW/cm² in the far field. Data collected with 3-panel screen for partial body exposure present (---Δ---) and absent (-----). Expose head only to 2450 MHz CW microwaves (mW/cm²)

Figure 41. Representative experiment on one monkey equilibrated to an ambient temperature of 20°C to determine effects on metabolic heat production (M) of 10-minute exposures of the head alone to 2450 MHz CW microwaves. Also shown are rectal and mean skin temperatures.
location, with restraining chair present, using the Narda field probe (an inappropriate instrument for near-field measurements). The reduced effectiveness of the microwave stimulus under 'head only' exposure may contribute to the greatly elevated thresholds we obtained experimentally on live animals under these field conditions (see below).

Pilot experiments demonstrated that the head had to be exposed to quite high power densities relative to levels that will alter thermoregulatory responses under whole-body exposure. Significant air temperature elevations inside the anechoic chamber interfered with air temperature control, particularly by the monkey during behavioral experiments. The series of experiments to measure changes in M (described below) were conducted by continually adjusting the circulating air temperature to counteract microwave-induced T_a increments. Tests to determine the influence of head-only microwave exposure on thermoregulatory behavior were conducted using the ventilated styrofoam box already described.

1. Changes in Metabolic Heat Production

Reductions in the metabolic heat production (M) of monkeys in a 20°C environment were determined during 10-min exposures of the head to 2450 MHz CW microwaves of increasing power density (range = 40-70 mW/cm²). The protocol and response measures were the same as employed in the original threshold experiments to determine the minimal power density that will reliably lower the elevated M of a monkey in the cold (see Figure 20). A representative experiment on one monkey is shown in Figure 41. Three such experiments were conducted on each of three monkeys with the radiation directed toward the right side of the head. A second series of three experiments was conducted on each monkey with the radiation directed to the back of the head, in view of the report by Burr and Krupp (1980) that the peak tissue temperature rise inside the head may be greatest for this head orientation (at least in Macaca mulatta).

Inspection of Figure 41 reveals that exposure of the head alone does indeed initiate reliable reductions in the metabolic heat production of an animal in a 20°C environment but the power densities, measured at the location of the head, must be very high. The data from these experiments were analyzed similarly to those from the original threshold test series in terms of the reduction in heat production from the pre-exposure level (ΔM) produced by each microwave power density. Figure 42 compares the two exposure orientations (right side of head exposed vs back of head exposed) on this M reduction. No reliable differences were found between the two series of experiments that may be attributed to animal orientation.

Figure 43 compares the ΔM results of head-only exposure (side of head exposed) with those of whole-body exposure of the same 3 monkeys (originally presented in Figure 23) at an ambient temperature of 20°C. When only the head is exposed, the power density must be roughly 10 times higher than for whole-body exposure to produce a comparable reduction in metabolic heat production. The head of the squirrel monkey constitutes approximately 9% of the total body mass. Thus, the microwave threshold for alteration of physiological thermoregulatory responses probably depends upon the integrated whole-body energy absorption, not upon energy deposited in some specific location which can be rapidly dispersed by the circulatory system. This interpretation was reinforced by the dosimetric studies of the temperature rise inside homogeneous brain-equivalent spheres described above. It is clear from our results that intense microwave exposure to the head alone, at least for short exposure durations, does not automatically mean that hypothalamic temperature receptors will be thermally stimulated. Greatly increased regional blood flow must quickly dissipate any local heat depots that occur (Stolwijk, 1980). The absence of vigorous heat loss responses in our experimental subjects at lower power densities is confirming evidence for this interpretation.
Figure 42. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures of the head only to 2450 MHz CW microwaves. The parameter is animal orientation. Ambient temperature (T_a) was 20°C in all experiments.

Figure 43. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures to 2450 MHz CW microwaves as a function of power density (mW/cm²). Symbols at low power densities (▲) are for whole-body exposure, those at high power densities (●) are for head-only exposure. Ambient temperature (T_a) was 20°C for all experiments.
2. Changes in Thermoregulatory Behavior

The availability of the ventilated styrofoam enclosure around the monkey allowed us to conduct a series of experiments to determine the effects on thermoregulatory behavior of exposing only the animal's head to the microwave field. As in the metabolic rate experiments described above, the purpose of these experiments was to make preliminary assessment of the role of hypothalamic vs other deep body receptors in the handling of microwave body heating.

Two animals served as experimental subjects. They were chair-restrained inside the ventilated styrofoam box, which was positioned immediately behind the 3-panel screen described above. Each animal regulated the chamber air temperature behaviorally by selecting between two circulating air temperatures, 10°C and 50°C. The experimental protocol, identical to our original series of threshold experiments (Adair and Adams, 1980b), featured a 2-hour baseline period of behavioral thermoregulation followed by 6 10-minute microwave exposures of increasing power density.

The results of one experimental series appear in Figure 44, which shows the skin and rectal temperatures of one monkey and the air temperature he selected during the 2-hour baseline period and subsequent exposures of the head alone to 2450 MHz CW microwaves. Power density in this series ranged from 15 to 40 mW/cm². Although the animal often selected a slightly cooler environment during the 10-minute periods when the microwaves were on compared to the immediately preceding period of microwaves-off, at no power density was this Tₐ reduction statistically significant.

Figure 44. Mean ambient temperature (Tₐ) selected by one monkey as a function of experimental time for three experiments in which the animal's head was exposed to 10-minute periods of 2450 MHz CW microwaves of increasing power density. The monkey worked in 10°C air for 50°C reinforcement. Resulting rectal (••••••) and mean skin (- - --- - - ) temperatures are also shown. Animal enclosed by styrofoam box.
Figure 45. Mean ambient temperature \( (T_a) \) selected by one monkey as a function of experimental time for five experiments in which the animal's head was exposed to 2450 MHz CW microwaves of increasing power density. The monkey worked in 10°C air for 50°C reinforcement. Resulting rectal (\( \cdots \triangle \cdots \)) and mean skin (\( --\bullet-- \)) temperatures are also shown. Animal enclosed by styrofoam box.

Figure 46. Mean ambient temperature \( (T_a) \) selected by one monkey as a function of experimental time for five experiments in which the animal's head was exposed to 2450 MHz CW microwaves of increasing power density. The monkey worked in 50°C air for 10°C reinforcement. Rectal (\( \cdots \triangle \cdots \)) and mean skin (\( --\bullet-- \)) temperatures are also shown. Animal enclosed by styrofoam box.
Figure 45 shows a second experimental series on this animal, in which the power densities ranged up to 60 mW/cm$^2$. This series yielded the same inconclusive results. No reduction in rectal temperature, such as might be expected if hypothalamic temperature sensors were being heated, was ever recorded during microwave exposure of the head. Our single positive, but unquantified, finding involved observations via the closed circuit TV observation system of an increasing agitation correlated with applied power densities of 45 mW/cm$^2$ and above. These results were confirmed in the second animal during a slightly different series of power densities (Figure 46). Once again, although the animal appeared to be lowering the chamber air temperature while the microwaves were on, these temperature excursions were not significantly greater than those occurring during the baseline period. We must conclude, therefore, that while exposure of the head alone may provoke some adjustment in the heat production of an animal in the cold, it carries little significance for behavioral thermoregulatory responses.

B. Screening the Head and Exposing the Remainder of the Body

The three-panel screen used in the experiments described above was modified slightly. The original 30 cm x 30 cm aperture was closed with a plywood insert and covered with 20 cm pyramidal microwave absorber. A new aperture, 30 cm wide and 45 cm high, was cut just below the location of the original one in the center panel of the screen so that when it was interposed between the antenna and the monkey (Figure 38), the monkey's body from the neck down received substantial radiation, the head being screened fairly successfully.

To determine the extent of field nonuniformity behind this new aperture, the field was mapped again at 5 cm intervals across the same 61 cm x 86 cm plane as that mapped previously. This plane passed through the center of the restraining chair location orthogonal to the direction of propagation of the incident field (K vector). Figure 47 represents a contour map of power density in this plane when forward power was constant at the same value as that previously employed (0.5 kW). A very steep power density gradient is evident in the center of the aperture, with the highest field strength measured in the region occupied by the animal's lower trunk and tail (hot seat?). Once again, the body region we hoped to screen (in this case the head) was exposed nominally to 10 dB less than the remainder of the body mass. Power densities specified in the experiments described below were measured, with the restraining chair present, at the location of the monkey's chest. Thus, a power density specified as 5 mW/cm$^2$ would produce $\sim 1.4$ mW/cm$^2$ at the head and $\sim 12$ mW/cm$^2$ at the lower trunk, neglecting field perturbations by the restraining chair and the animal subject.

The effects of the steep gradient in field strength were evident during dosimetric procedures. Temperature increments were measured at 4 depths in the 1.1 liter saline-filled model under controlled 10-min exposures to different power densities. The procedure was identical to that described earlier in this report. The physical arrangement of the model relative to the screen is diagrammed in Figure 48 (insert). The SAR calculated from the temperature increments (Figure 48) was 25% higher than that derived for far-field exposure with the screen absent. On the basis of these data, we anticipated that microwave thresholds for the alteration of thermoregulatory responses under these exposure conditions would be little different from, perhaps lower than, those measured under conditions of whole-body far-field exposure.
Figure 47. Contour map of field intensity across a 61 cm x 86 cm plane orthogonal to the K vector centered on the antenna boresight 1.42 meters from the front edge of the antenna. Three-panel screen with 30 cm x 45 cm aperture (for exposing only the animal’s body) in place. Dashed figure shows location of monkey in the plane of measurement. Forward power = 0.5 kW. Contour interval = 10 mW/cm² in the center of the field, 1 mW/cm² in the periphery.

Figure 48. Mean and range of temperature increments recorded by four thermocouples (TC) positioned as shown (insert) in a 1.1 liter saline-filled cylindrical model as a function of power density for 10-minute exposures to 2450 MHz CW microwaves. Screen interposed between antenna and model exposed lower part of model. Ambient temperature ($T_a$) = 35°C.
1. Changes in Metabolic Heat Production

Reductions in the metabolic heat production (M) of monkeys in a 20°C environment were determined during 10-minute exposures of the trunk and extremities (head screened) to 2450 MHz CW microwaves of increasing power density (range = 2-10 mW/cm²). The protocol and response measures were the same as those employed in the original threshold experiments to determine the minimal power density that would reliably lower the elevated heat production (M) of a monkey in the cold (see Figure 20), as well as the analogous experiments in which only the head was exposed (Figure 41). Three such experiments were conducted on each of three monkeys.

It should be noted that the power density range studied here was comparable to that studied during whole-body exposure to the microwave field. Indeed, a typical experiment would nearly duplicate the one presented in Figure 20, conducted under conditions of whole-body exposure. We found that metabolic heat production was reliably reduced below the stabilized baseline level by 10-minute microwave exposure at 6-8 mW/cm² and above. These adjustments in heat production served to regulate the internal body temperature at the characteristic normal level.

Once again, the magnitude of the M adjustment was directly related to microwave intensity. This is shown in Figure 49 which superimposes the mean responses of one monkey to different microwave power densities upon the same time scale. Comparing this figure to Figure 21 (whole-body exposure), we see that the higher the power density, the greater the M reduction. However, there were subtle differences between the responses under the two conditions: the threshold was slightly higher and the latencies to microwave onset and termination somewhat longer when the head was screened than when the whole body was exposed to the microwave field. None of the animals in the partial body experimental series reduced M reliably at a power density of 4 mW/cm²; this threshold was 6 mW/cm² for 2 animals and 8 mW/cm² for the third. Thus, it would appear that screening the head reduces the effectiveness of the microwave field despite the fact that the rate of energy deposition, integrated over the whole body, may be considerably higher than for whole-body exposure of comparable intensity.

![Figure 49](image-url)
EXPOSE TRUNK ONLY TO 2450 MHz CW MICROWAVES

Figure 50. Mean metabolic heat production (M), weighted mean skin (T_{sk}) and rectal (T_{re}) temperatures of two monkeys as a function of power density for 10-min exposure of the trunk and extremities to 2450 MHz CW microwaves. Ambient temperature (T_a) = 20°C.

2. Changes in Thermoregulatory Behavior

One series of experiments was conducted to determine the effects on thermoregulatory behavior of screening the animal's head while exposing the rest of the body to the microwave field. Three animals served as experimental subjects. They were chair-restrained inside the ventilated styrofoam box which was positioned immediately behind the 3-panel screen described above. Each animal regulated the circulating air temperature behaviorally by selecting between two available air temperatures, 10° and
Figure 51. Mean ambient temperature ($T_a$) ± 1 SEM selected by one monkey as a function of experimental time for 5 experiments in which the animal's trunk and extremities were exposed to 2450 MHz CW microwaves of increasing power density. The monkey worked in 10°C air for 50°C reinforcement. Rectal (⋯△⋯) and mean skin (---□---) temperatures are also shown. Animal enclosed by styrofoam box.

50°C. Once again, the experimental protocol was identical to our original series of threshold experiments (Adair and Adams, 1980b), and featured a 2-hour baseline period of behavioral thermoregulation followed by 6 10-minute microwave exposures of increasing power density (2, 4, 6, 8, 10, and 12 mW/cm$^2$). Five such experimental sessions were conducted on each animal.

The results of this experimental series for one monkey appear in Figure 51 which shows his rectal and skin temperatures and the air temperature selected during the 2-hour baseline period and subsequent exposures of the trunk and extremities to 2450 MHz CW microwaves. In general, this animal prefers a somewhat cooler environment (≈ 32°C) than most of the other squirrel monkeys in our colony, but is nevertheless adept at regulating his body temperature behaviorally. He showed a clear and reliable reduction in preferred environmental temperature (threshold) when exposed at a power density of 12 mW/cm$^2$, and a strong tendency toward such a reduction at 8 and 10 mW/cm$^2$ as well.

The threshold of the second animal, whose data appear in Figure 52, was determined to be 10 mW/cm$^2$ with a tendency to reduce the preferred air temperature at 8 mW/cm$^2$ as well. This animal, one of those tested in the original threshold series, showed a clear threshold at 6 mW/cm$^2$ when the whole body was exposed. The threshold of the third animal, also one of those tested in the original series, was essentially indeterminate in the absence of current baseline data. The results for this monkey, shown in Figure 53, are complicated by baseline drift which occurred gradually over the course of the 4-hour sessions. Students t-tests performed between consecutive means yielded 6 mW/cm$^2$ as a reliable threshold for reduction of the preferred air temperature from the preceding 10-min period when no microwaves were present. Whole-body exposure produced the same threshold level for this animal.
Figure 52. Mean ambient temperature ($T_a$) ± 1 SEM selected by one monkey as a function of experimental time for 5 experiments in which the animal's trunk and extremities were exposed to 2450 MHz CW microwaves of increasing power density. The monkey worked in 50°C air for 10°C reinforcement. Rectal (--- Δ---) and mean skin (--□--) temperatures are also shown. Animal enclosed by styrofoam box.

Figure 53. Mean ambient temperature ($T_a$) ± 1 SEM selected by one monkey as a function of experimental time for 5 experiments in which the animal's trunk and extremities were exposed to 2450 MHz CW microwaves of increasing power density. The monkey worked in 10°C air for 50°C reinforcement. Rectal (--- Δ---) and mean skin (--□--) temperatures are also shown. Animal enclosed by styrofoam box.
The results of these preliminary experiments suggest that the threshold power density that will cause a behaving animal to select a cooler environment is slightly higher when the head is screened than when the whole body is exposed to microwaves. The difference is probably of the order of 10-20%. It should be emphasized that this conclusion is tentative because control data are lacking and only one test protocol has so far been performed. A measurement of brainstem temperature during these and the previous experiments in which only the head was exposed would be very useful for interpreting the results. The necessary methodology for making such measurements should be available in the near future.

C. A Summary of Partial Body Exposure Effects: Do Two Halves Make a Whole?

We have attempted, with moderate success, to selectively shield the head or the rest of the squirrel monkey's body from an imposed 2450 MHz CW microwave field. One of our hopes was that we might selectively heat the thermosensitive areas in the anterior brainstem, a hope not realized because of the phenomenal capacity of the circulatory system to dissipate local heat so deposited. Some interesting results emerged from these studies, however. Exposing only the animal's head to microwaves lowered the metabolic heat production of animals in cold environments, but the power density required to produce this effect had to be about 10 times that required during exposure of the whole body. Similar head-only exposures had no reliable effects upon thermoregulatory behavior and appeared only to make the animals agitated.

On the other hand, when the head was screened and the remainder of the body exposed to the field, both autonomic and behavioral thermoregulatory responses were altered in ways we have previously demonstrated for whole-body microwave exposure. There were some small differences, however; thresholds were probably slightly higher and the responses could be characterized as sluggish and of smaller magnitude. Perhaps some, but by no means all, of the cause for these differences may reside in a few changes in the roster of subjects; indeed, many of the same animals have served through the project from start to finish.

An interesting exercise shed some light on these matters. Three separate series of experiments had measured the reduction of metabolic heat production of monkeys in a 20°C environment that was produced by 10-minute microwave exposures. The first series involved whole-body exposure, the next series exposure of the head alone, and the third series exposure of the remainder of the body excluding the head. The basic question asked was whether the sum of the separate responses to partial body microwave exposure was equivalent to the response magnitude produced by whole-body exposure. An affirmative answer would strengthen the tentative conclusions drawn above.

Figure 54 shows the change in metabolic heat production from the preceding baseline level produced by 10-minute microwave exposures at different power densities. Data for whole-body exposure are compared here with data from exposures in which the head was screened. The former produced a substantially greater effect upon the measured response than the latter, by about 27%.

Comparable data from experiments in which only the head was exposed to microwaves appear in Figure 55. In this case, much higher power densities were required to produce reliable reductions in metabolic rate. The best-fitting function to these data has been extrapolated to the origin and values read for the lower power densities (2, 4, 6, 8, and 10). These extrapolated values were added to the appropriate data from the experimental series in which the head was screened and the resulting sum appears
Figure 54. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures to 2450 MHz as a function of power density (mW/cm²). Data for whole-body exposure (□) and exposure of trunk and extremities only (○) are shown. Ambient temperature (Tₐ) = 20°C for all experiments.

Figure 55. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures of the head alone to 2450 MHz CW microwaves as a function of power density (mW/cm²). Best-fitting function is extrapolated to the origin. Ambient temperature (Tₐ) = 20°C.
Figure 56. Mean change in metabolic heat production (W/kg) from stabilized level produced by 10-minute exposures to 2450 MHz CW microwaves as a function of power density (mW/cm^2). Data for whole-body exposure (▲) is compared with summed data for partial-body exposures (●). Ambient temperature (T_a) = 20°C for all experiments.

In Figure 56. For comparison, the values for whole-body exposure are also shown in the figure. The agreement is fairly good, leading to the conclusion that while two halves do not quite make a whole, they probably do within experimental error. This analysis, while providing indirect evidence for the effectiveness of our screening methods, reinforces the conclusion that the thermoregulatory response, when part of the body is exposed to a microwave source, results from an integration of the absorbed energy over the total body mass.
CONCLUSIONS

Low intensity microwave fields have been shown to influence the normal responses, both autonomic and behavioral, that function to regulate the body temperature. Using the squirrel monkey as an animal model, we have determined the minimal incident energy (in mW/cm²) derived from 2450 MHz CW microwaves that is necessary to lower metabolic heat production in the cold, alter peripheral vasomotor tonus in thermoneutral environments, initiate thermoregulatory sweating in the heat, and stimulate a behaving animal to select a cooler environment. The threshold power densities in all cases were remarkably similar (4-8 mW/cm²), suggesting a common thermal basis for the response changes. The whole-body SAR (in W/kg) at threshold represents 15-20% of the resting metabolic heat production of the squirrel monkey. Preliminary experiments on anaesthetized animals have determined that the local tissue temperature rise in the "central thermostat" of the brainstem at a threshold power density of 6-8 mW/cm² is probably sufficient to trigger the observed changes in thermoregulatory effector responses.

Under conditions of prolonged microwave exposure (up to 2 1/2 hours), adaptation of the autonomic responses that generate or dissipate body heat often occurred, enabling precise regulation of the internal body temperature. No such adaptation was evident in behavioral thermoregulatory responses, which persisted as long as the microwave field was present. Partial body exposure to microwave fields was found to produce appropriate changes in both autonomic and behavioral thermoregulatory responses to a degree nominally proportional to the fraction of the body so exposed.

The ultimate goal of research such as that reported here is to evaluate the impact of microwave exposure on the human thermoregulatory system with reference to current maximum permissible exposure standards. This must necessarily involve careful power and frequency extrapolations as well as considerations of body heat balance in man vs the animal model. It would appear that, with the possible exception of exposure to moderate microwave intensities in warm environments, the squirrel monkey model will be extremely useful in this regard. Our most important single finding to date is that, no matter in what environment the organism finds itself, it detects and responds immediately to low intensity microwave fields as it does to other environmental thermal stimuli. The net result is that internal body temperature is regulated with precision at the normal level.
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A. Publications


Bruce-Wolfe, V. and Adair, E. R. Operant control of microwave radiation by squirrel monkeys. (In preparation)


Adair, E. R. Thermoregulation in the squirrel monkey (Saimiri sciureus) during exposure to 2450 MHz CW microwaves (In preparation).


B. Presentations at Scientific Meetings.


9 April, 1979 "Microwaves alter thermoregulatory behavior" FASEB, Dallas, TX.

18 June, 1979 "Microwave modification of thermoregulatory behavior: Threshold and suprathreshold effects" Bioelectromagnetics Society, Seattle, WA.

10 September 1979 "Microwave modification of thermoregulatory behavior: Threshold and suprathreshold effects" International Congress of Biometeorology, Shefayim, Israel.

9 November, 1979 "Microwaves modify thermoregulatory behavior but exposure duration matters little" Psychonomic Society, Phoenix, AZ.


16 September, 1980 "Metabolic adjustments during whole-body 2450 MHz CW microwave exposure" Bioelectromagnetics Society, San Antonio, TX.

14 November, 1980 "Behavioral thermoregulation: Absence of adaptation to prolonged microwave exposure" Psychonomic Society, St. Louis, MO.

14 April, 1981 "Adjustments in metabolic heat production during whole-body 2450 MHz CW microwave exposure" FASEB, Atlanta, GA.


12 August, 1981  "Initiation of thermoregulatory sweating by whole-body 2450 MHz microwave exposure"
                Bioelectromagnetics Society, Washington, DC.

11 August, 1981  "Operant control of microwave radiation by squirrel monkeys"
                Bioelectromagnetics Society, Washington, DC.

27 October, 1981 "Changes in thermoregulatory behavior during microwave irradiation"
               Microwaves and Thermoregulation: A Symposium
               New Haven, CT.

C. Seminars (colloquia)

8 November, 1978 School of Aerospace Medicine, Brooks AFB, TX.
22 January, 1979 John B. Pierce Foundation Laboratory, New Haven, CT.
21 February, 1979 Walter Reed Army Institute of Research, Washington, DC.
19 March, 1979    Wellesley College, Wellesley, MA.
19 April, 1979    VA Medical Center, Kansas City, MO.
19 November, 1979 Armed Forces Radiobiology Research Institute, Bethesda, MD.
18 January, 1980  Environmental Protection Agency, HERL, Research Triangle Park, NC.
26 February, 1981 Mt. Holyoke College, So. Hadley, MA.
15 April, 1981    Naval Medical Research Institute, Bethesda, MD.