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MICROWAVE RADIATION AND THERMOREGULATION

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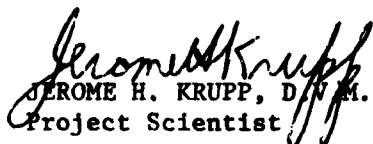
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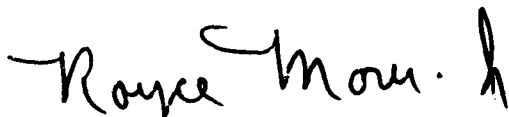
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The Office of Public Affairs has reviewed this report, and it is releasable to the National Technical Information Service, where it will be available to the general public, including foreign nationals.

This report has been reviewed and is approved for publication.

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Adult male squirrel monkeys (Saimiri sciureus) were equilibrated to one of three ambient temperatures (T_a=20, 26 and 32 °C) and then re-equilibrated for a 90-min period in the presence of a 2450-MHz CW microwave field, (unilateral planewave). Power densities of 10, 15, 20, and 25 mW/cm² were explored (SAR=1.5, 2.25, 3.0, and 3.75 W/kg, respectively) at each T_a, except for 25 mW/cm² at T_a=32 °C. In accordance with the method of partition-al calorimetry, the following autonomic responses of heat production and heat loss were measured during the experiments: metabolic heat production, respiratory evaporative heat loss, total evaporative heat loss, skin and deep body temperatures that allowed determina-tion of heat flows within the body, and from the body to the environment. At all power densities in all T_a, the monkeys achieved thermal balance by the mobilization of appropri-ate thermoregulatory responses. These included a reduction of metabolic heat production in cool T_a, vasodilation of tail and foot in thermoneutral T_a, and initiation of thermo-regulatory sweating in warm T_a. The coefficient of heat transfer to the environment, ⌐

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19. ABSTRACT (continued)

derived from these measures, was the same as that determined directly in the test environment. Changes in skin temperature during microwave exposure were primarily responsible for the initiation of changes in metabolic heat production, sweating rate and regulated internal body temperature, indicating the predominant role of peripheral thermal sensors in thermoregulation at this frequency. The major conclusion derived from these studies is that the thermoregulatory system deals with energy absorbed from microwave fields in exactly the same way as energy produced in the body by normal metabolic processes or absorbed during exposure to conventional radiant or convective heat sources.

TABLE OF CONTENTS

FIGURES

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OOPY
INSPECTED
1

A-1

v

MICROWAVE RADIATION AND THERMOREGULATION

INTRODUCTION

Temperature Changes and Thermoregulation

Endotherms are organisms capable of maintaining a stable internal body temperature in the face of rather wide fluctuations in the thermal characteristics of the environment. Thermoregulation in endotherms is accomplished by fine adjustments in appropriate autonomic response systems, by which the body gains or loses heat, acting in concert with a wide range of behavioral maneuvers that provide a hospitable microclimate for the animal. Whenever possible, the behaviorally generated microclimate is thermally neutral, a situation that maximizes the economy of water and energy stores in the body and minimizes the involvement of autonomic mechanisms. Thus the description of thermoregulation in any endotherm involves detailed knowledge of thermoregulatory behavior, both instinctive and learned, and of individual autonomic processes of heat production and heat loss. The particular autonomic response that may be operative at any given time is dictated by the prevailing environmental temperature; i.e., endotherms shiver in the cold and sweat or pant in the heat, but not the reverse, and they will avoid doing either one if an efficient behavioral maneuver is available to them.

In intact animals, thermal stimuli will elicit both behavioral and autonomic thermoregulatory responses. These stimuli include not only variations in the microclimate (i.e., ambient temperature, ambient vapor pressure, air movement, and insulation), but also internal temperature changes due to, e.g., circadian variation, febrile disease, and exercise. 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 of these sites in normal thermoregulation. Since microwave radiation can be absorbed in extremely complex configurations by biological entities, thereby generating heat in the tissues of the body, it must be regarded as a significant thermal stimulus to thermoregulation.

Summary of Earlier Findings

The thermoregulatory consequences of exposure to radio frequency electromagnetic radiation (2450-MHz CW microwaves) have been under intense study in the microwave laboratories at the John B. Pierce Foundation Laboratory since 1977. The goal of this research has been to quantify, using the squirrel monkey as an animal model, the minimal incident microwave energy (in mW/cm^2) that reliably influences the normal responses, both autonomic and behavioral, that regulate the body temperature. The nature of the thermoregulatory alteration has always been characterized in terms of both absorbed energy and local body temperature perturbations. Other goals of this research project have involved exploration of other parameters of microwave exposure such as the intensity and duration of the exposure, the part of the body exposed, and the effects of concomitant thermal stimulation of the hypothalamic thermoregulatory center by means of implanted thermodes. Controls for surface heating,

1

provided by infrared radiation or convective heating, have been an integral part of many experimental designs.

The animals are chair restrained in the far field of a horn antenna inside an electromagnetically anechoic chamber of interior dimensions 1.83 m x 1.83 m x 2.45 m. A valve system allows air from one of two closely regulated (±0.5 °C) sources to circulate through the anechoic space. Each monkey is trained to pull a response cord to operate the valves, thereby selecting the environmental temperature the animal prefers. The use of a single air source provides an environment of constant temperature for the assessment of autonomic thermoregulatory responses. Usually, in order to achieve more precise control over the 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 Model S2.5W generator and fed to the antenna via standard waveguide components. Calibration measurements to determine field uniformity at the animal's location, made with a Narda Model 8316B broadband isotropic radiation detector fitted with a model 8323 probe, 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 and Vitek probes (18) for measurement of body temperatures, tubing for the circulation of temperature-controlled silicone oil to thermodes implanted in the monkey's brainstem, or a Lucite boot and hose connections for measurement of thermoregulatory sweating from the foot of the animal.

An assessment of whole-body energy absorption over the power density range from 5 to 40 mW/cm^2 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 per mW/cm^2, with the higher values corresponding to the smaller masses. Rectal temperature increments in conscious squirrel monkeys, during 10-min microwave exposures in thermoneutral environments, yielded a comparable SAR of 0.15 W/kg per mW/cm^2.

Brief (5-10 min) unilateral exposure of the monkey's whole body to 2450-MHz CW microwaves (E polarization) allowed us to determine the minimal power density (mW/cm^2) that reliably alters thermoregulatory responses (thresholds). These thresholds are very similar: 6-8 mW/cm^2 (SAR=1.1 W/kg) stimulates the animal to select a cooler environment behaviorally, induces peripheral vasodilation of the tail vessels, and initiates thermoregulatory sweating from the foot, while a slightly lower power density, 4-6 mW/cm^2 (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 2½ h) produces little or no adaptation of behavioral thermoregulatory adjustments, but does produce a gradual adaptation of metabolic heat production such that, in the steady state, the SAR (W/kg) of the exposure 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 mi

exposure in warm environments, but it is not sufficient to prevent a rise in body temperature in this species.

When only the head is exposed to the microwave field, the trunk and extremities being screened, the power density must be nearly 10 times greater than when the whole body is irradiated to produce a given reduction of metabolic heat production in a cold environment. It is clear that heat generated when energy is deposited directly in the head may be efficiently carried to other parts of the body by the circulatory system. Indeed, when the power density is averaged over the total exposed silhouette in accordance with localized measurements of field strength, the response change is found to depend on the integrated energy absorption by the whole body, not on energy deposited in some particular body locus. Other experiments, conducted when the head was screened and the remainder of the body was exposed to the microwave field, confirmed this analysis: the thresholds for the reduction of metabolic heat production in the cold or the alteration of thermoregulatory behavior when the head alone was screened were only slightly higher than the thresholds measured during whole-body exposure.

The depth to which microwaves at a frequency of 2450 MHz may penetrate, roughly 2.0 to 2.5 cm, indicates that this frequency is resonant to the head of the squirrel monkey. Thus, there is the potential for an enhanced rate of local energy deposition, and therefore selective local heating, in the center of the brain. Such heating may occur in the medial preoptic/anterior hypothalamic area (PO/AH), the region of the anterior brainstem that has been shown to harbor the "central thermostat" for the regulation of the body temperature. We have developed a chronic brain implant that allows both the measurement and control of the temperature of this brainstem area in squirrel monkeys exposed to microwave fields. Brain temperature measurements in several squirrel monkeys exposed (unilateral exposure of the whole body) to 2450-MHz CW microwaves reveal that a PO/AH temperature rise of 0.2-0.3 °C is associated with the threshold power density that stimulates selection of a cooler environment by the animal. A PO/AH temperature rise of the same magnitude, produced by perfusing an implanted thermode with warm silicone oil, will also stimulate the selection of a cooler environment, lower metabolic heat production of an animal in the cold, and initiate thermoregulatory sweating of an animal in a warm environment. Experiments of complex design have demonstrated that a small temperature rise in the PO/AH, that may occur during microwave exposure, must be supplemented by temperature changes elsewhere in the body to produce given thermoregulatory response changes. Indeed, the data show that the PO/AH area probably plays a rather limited role in the thermoregulatory response to microwaves and that, at this frequency, the thermal receptors in the skin are of equal importance in the mobilization of efficient autonomic and behavioral responses.

Our experience over the past several years, in light of the current research into the biological effects of exposure to radiofrequency radiation, points clearly to an important fact: in order to characterize completely the autonomic thermoregulatory response of an animal to an imposed radiofrequency field of any frequency, all efferent response systems that influence thermal balance should be quantified. Only by so doing, will it be possible to pinpoint the particular system that may be under alteration at any given time and to predict, on the basis of differences in "thermoregulatory profiles," how human beings might respond in the presence of comparable radiofrequency

fields. The research described has been conducted with this important fact in mind. An augmented set of response measures describes the total thermoregulatory response of the squirrel monkey when it is in equilibrium with a range of microwave power densities and ambient temperatures. Only autonomic response systems are considered here, not thermoregulatory behavior. The method of partitional calorimetry, a standardized technique in thermal physiology (29), has been employed throughout. A description of this method and the basic data on thermoregulation in the squirrel monkey, derived from partitional calorimetry, appear in the following sections of this report.

Method of Partitional Calorimetry

The thermalization of tissues in the body that accompanies the exposure of an organism to microwaves presents a unique challenge to the thermoregulatory system. The heat generated in the body tissues during such exposures may be considered to be comparable to that produced during physical exercise, except that no increase in metabolic heat production would be anticipated. In other words, during microwave exposure, tissue heating is passive rather than active. The simplest way of determining the impact of any microwave exposure on the thermoregulatory system is to apply the methods of partitional calorimetry to the organism in question.

In general, the thermoregulatory system mobilizes responses to imposed thermal disturbances in such a way as to minimize or negate the disturbance. The result of this activity is that the internal body temperature, the regulated variable, is maintained at a constant or set level. The disturbance may be a change in the rate of heat production (as during exercise) or a change in the rate of heat exchange between the organism and the environment (as during a change in ambient conditions). In the steady state, the heat produced in the body of an endotherm is balanced by the heat lost to the environment such that storage of heat within the body is minimal. The concept can be expressed by a generalized heat balance equation of the form (17):

$$\underline{M} - \underline{W} = \underline{R} + \underline{C} + \underline{E} \pm \underline{S} \tag{1}$$

The thermal energy produced in the body by metabolic processes $\underline{M}$ will be lified by any work $\underline{W}$ produced by the animal on the environment. While $\underline{W}$ may be a significant factor for humans or beasts of burden, it may be considered negligible for other endothermic mammals. Thus, for practical purposes, $\underline{M}$ represents the metabolic heat production of the body.

The first three terms on the right side of the equation represent the different avenues by which heat is exchanged between the body and the environment: $\underline{R}$ represents radiation, $\underline{C}$ represents convection, and $\underline{E}$ is the heat lost through the evaporation of water from the skin surface and respiratory tract. No term appears in the equation for heat exchange by conduction since it is usually insignificant in most species. If the environmental temperature is higher than that of the body, the direction of heat transfer may be into the body and $\underline{R}$ and $\underline{C}$ may have a negative sign. Under such conditions, evaporation of fluid from the body surface (and the lungs) is the only available avenue of heat loss. Man and certain nonhuman primates (e.g., Erythrocebus patas) sweat profusely and thus are able to thermoregulate efficiently, even during exercise in hot environments (21). The squirrel monkey does not

4

capability, possessing eccrine sweat glands only on the friction surfaces of the palms and soles (35,41). Thus, this species must rely to a large extent on behavioral responses (e.g., spreading urine, creating a cool microclimate) to ensure maintenance of a stable body temperature in the heat. It is important to note that these behavioral responses are not available to a squirrel monkey under restraint in a laboratory setting such as the one described.

The last term in the heat balance equation, S, represents the rate of heat storage in the body. If S is positive, body temperature rises; if S is negative, body temperature falls. Clearly, the goal of normal thermoregulation is the minimization of S and thus the achievement of a stable internal body temperature. The methods of partitional calorimetry are particularly useful to determine the values of individual terms in the heat balance equation at any given environmental temperature for a particular species.

When the method of partitional calorimetry is used, the experimental animal is brought into thermal equilibrium (i.e., $S=0$) with a particular environmental temperature and the steady-state heat production and heat loss responses are measured. A wide range of environmental temperatures is studied, which encompasses the range to which the animal is normally exposed, a design that yields a thermoregulatory profile of the species in question.

Metabolic heat production M is calculated from oxygen consumption and carbon dioxide production from which the respiratory quotient (RQ) may also be calculated. Oxygen consumption alone will suffice with the assumption of a constant RQ (0.83 for the squirrel monkey). Total evaporative water loss, which includes water lost through respiration, passive diffusion through the skin, and that evaporated as sweat, is determined from the total reduction in body weight during the experiment, urine and feces being trapped under oil. Evaporative heat loss E is then calculated assuming the latent heat of evaporation of water to be 0.72 W·h/g. Dry heat exchanged with the environment through convection C and radiation R must be expressed in terms of the surface area of the body. For the squirrel monkey, Stitt et al. (43) have determined that a body mass of 1 kg is equivalent to a surface area of 0.108 m^2.

Heat exchange via radiation and convection is determined indirectly in the method of partitional calorimetry and requires the measurement of the animal's mean skin temperature ($\bar{T}_{sk}$). Under steady-state conditions of rest at a constant air temperature T_a, Equation (1) can be simplified to

$$M = R + C + E \tag{2}$$

In the steady state, when the walls of the environmental test chamber are at the same temperature as T_a, the heat losses from the body due to radiation and convection $R + C$ are a function of the thermal gradient between skin and air; thus

$$R + C = h\,(\bar{T}_{sk} - T_a) \tag{3}$$

where h is the coefficient of heat transfer to the environment. Substituting in Equation (2) yields

$$M = h\,(\bar{T}_{sk} - T_a) + E \tag{4}$$

5

in which $\underline{h}$ is the only unknown. This coefficient can be determined at any given T_a from the relation

$$\underline{h} = (\underline{M} - \underline{E})/\overline{T}_{sk} - T_a)$$ (5)

and a plot of $(\underline{M} - \underline{E})$ versus $(\overline{T}_{sk} - T_a)$ yields a straight line of slope $\underline{h}$ that passes through the origin, where $\overline{T}_{sk} \stackrel{\sim}{=} T_a$. The units in which $\underline{h}$ is expressed are $(W/m^2)/°C$.

The total evaporative heat losses from the body $(\underline{E}_{tot})$ may be partitioned into that which is lost from the respiratory tract $(\underline{E}_{res})$, and that which leaves the skin in the form of sweat $(\underline{E}_{sw})$; thus

$$\underline{E}_{tot} = \underline{E}_{res} + \underline{E}_{sw}$$ (6)

An assessment of the vasomotor tonus of the peripheral circulation can be made by calculating conductance $\underline{K}$, a measure of the core-to-skin heat flow. With the exception of the respiratory evaporative heat loss, $\underline{E}_{res}$, all heat leaving the body must pass from the deep body core to the skin, from which it is transferred to the environment by radiation, convection, and evaporation. Under steady-state conditions when heat storage in the body is zero, the heat leaving the body must be equal to the metabolic heat production (Eq. 2). In this case, $\underline{K}$ is the total amount of heat leaving the surface of the body divided by the temperature gradient between the core (T_{co}) and the skin $(\overline{T}_{sk})$. $\underline{E}_{res}$ is not included in this heat flow because it leaves the body directly through the respiratory tract. Thus

$$\underline{K} = (\underline{M} - \underline{E}_{res})/(T_{co} - \overline{T}_{sk})$$ (7)

When an organism is exposed to a radiofrequency field, the energy absorbed from the field $(\underline{A}_{rfr})$ must be added to the metabolic heat produced by the body. Neglecting the work factor, Equation 1 would then become

$$(\underline{M} + \underline{A}_{rfr}) = \underline{C} + \underline{R} + \underline{E} \pm \underline{S}$$ (8)

and all other expressions that involve heat production $\underline{M}$ would be similarly modified. Thus, the heat transfer coefficient for an organism exposed to a radiofrequency field would become

$$\underline{h} = [(\underline{M} + \underline{A}_{rfr}) - \underline{E}]/(\overline{T}_{sk} - T_a)$$ (9)

and conductance would become

$$\underline{K} = ([\underline{M} + \underline{A}_{rfr}] - \underline{E}_{res})/(T_{co} - \overline{T}_{sk})$$ (10)

Basic Thermal Physiology of the Squirrel Monkey

The autonomic thermoregulatory responses of five adult male squirrel monkeys to T_a that ranged from 10 to 39 °C were measured by Stitt and Hardy (41). During the experimental tests, individual animals were restrained in a Plexiglas chair (1) inside an environmental test chamber. The T_a was closely regulated and air movement within the chamber was < 8 m/min (still air conditions). A Plexiglas hood over the animal's head collected the expired air.

6

which was drawn outside the chamber at 7-10 L/min for analysis of oxygen content. The increase in relative humidity of the expired air was also measured so that respiratory evaporative heat loss, E_{res}, could be calculated. The restraining chair was mounted on a platform which was suspended from a sensitive balance. In this way the reduction in body mass could be monitored continuously during the experiment. Deep colonic temperature, 10-cm beyond the anal sphincter, was measured with a polyethylene-encased copper-constantan thermocouple with a reference junction in melting ice and water. Four representative skin temperatures, taken from shaved areas on the abdomen, tail, leg and foot were also measured with copper-constantan thermocouples constructed in special configurations. These temperatures were used to calculate a weighted $\bar{T}_{sk}$ as suggested by Stitt et al. (43):

$$\bar{T}_{sk} = 0.45T_{abd} + 0.37T_{leg} + 0.11T_{tail} + 0.07T_{foot} \qquad (11)$$

Each animal underwent at least three experimental tests at each of the following T_a: 10, 15, 20, 25, 30, and 35 °C. A few tests were conducted at T_a=39 °C, but a steady state could not ordinarily be achieved since this T_a is very close to the animal's normal regulated internal body temperature. Figure 1 shows the thermoregulatory profile of the squirrel monkey derived from the data of Stitt and Hardy (41). The functions drawn in the figure are the lines of best fit to the data and show the mean steady-state levels of metabolic heat production ($\underline{M}$), respiratory evaporative heat loss (E_{res}), total evaporative heat loss ($\underline{E_{tot}}$), and thermal conductance ($\underline{K}$) as a function of the ambient temperature to which the animals were exposed. Steady-state colonic temperature, weighted mean skin temperature, and the temperatures of tail and foot skin, measured in the same experiments, are shown in Figure 2. The data in Figures 1 and 2 represent the fundamentals of autonomic thermoregulation in the squirrel monkey. The individual responses are discussed in some detail in the following paragraphs.

When squirrel monkeys are restrained in cool environments, the body temperature is regulated by an increase in metabolic heat production ($\underline{M}$). All other responses are at low ebb (Fig. 1). The figure shows that as $\bar{T}_a$ falls below about 26-27 °C, $\underline{M}$ increases linearly at a rate of about 0.35 (W/kg)/°C. Active shivering may be observed at T_a=20 °C and below. At T_a = 26-27 °C, designated the lower critical temperature (LCT), resting heat production reaches its nadir and remains at this low level throughout the range of T_a that is designated the thermoneutral zone (TNZ) (T_a=27-35 °C). Stitt and Hardy (41) found that the resting heat production of chaired animals ranged from 4.5 to 7.0 W/kg in the TNZ. Other data (2,9,36) confirm this general range although the particular values obtained will depend on the air movement in the test chamber as well as the method used to measure oxygen consumption. Excessively warm environments, e.g., T_a above 35 °C, threaten effective thermoregulation in this species. An increase in $\underline{M}$ has been reported under such conditions that is presumably related to increased behavioral activity. The upturn in the $\underline{M}$ function in Figure 1 is likely the result of struggling against the restraining chair as the animal attempts to escape from the warm environment. Exposure of these animals to microwave fields at T_a=35 °C and above can be threatening (3).

Figure 1 displays two functions for evaporative heat loss, that from the body as a whole, $\underline{E_{tot}}$, and that from the respiratory tract, $\underline{E_{res}}$. The difference between the two represents evaporative heat loss through sweating, $\underline{E_{sw}}$,

Figure 1. Thermoregulatory profile for the restrained squirrel monkey equili-
brated to ambient temperatures ranging from 10 to 39 °C. Individual
functions show metabolic heat production ($\underline{M}$), total evaporative heat
loss ($\underline{E}_{tot}$), respiratory evaporative heat loss ($\underline{E}_{res}$), and tissue
conductance ($\underline{K}$). The thermoneutral zone of vasomotor control (TNZ)
encompasses ambient temperatures between the lower critical tempera-
ture (LCT) of 26 °C and the upper critical temperature (UCT) of 35
°C. Figure constructed from data in Stitt and Hardy (41).

and a minimal amount of passive diffusion of water through the skin (commonly
designated "insensible perspiration"). The latter tends to be constant and is
insignificant for thermoregulation. Both $\underline{E}_{tot}$ and $\underline{E}_{res}$ are low and constant
at T_a below about 30 °C. At T_a above 35 °C (UCT), $\underline{E}_{tot}$ becomes significantly
elevated above the basal level, whereas $\underline{E}_{res}$ changes hardly at all. This fact
implies that the major avenue of evaporative heat loss in this species is
sweating, not panting, an implication confirmed by Nadel and Stitt (38) who
recorded changes in sweat rate from the foot of squirrel monkeys restrained in
warm environments. The sweating is truly thermoregulatory because its rate
can be altered by changing the local tissue temperature of the PO/AH thermo-
regulatory center (42). Because sweating in this species can be emotional as

well as thermoregulatory, it is important to measure concomitant changes in $\underline{M}$ during experiments in which sweating is anticipated.
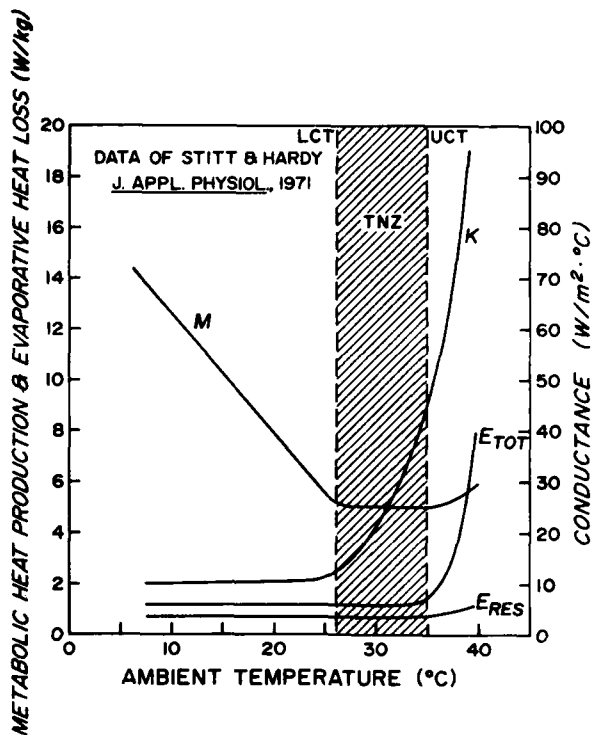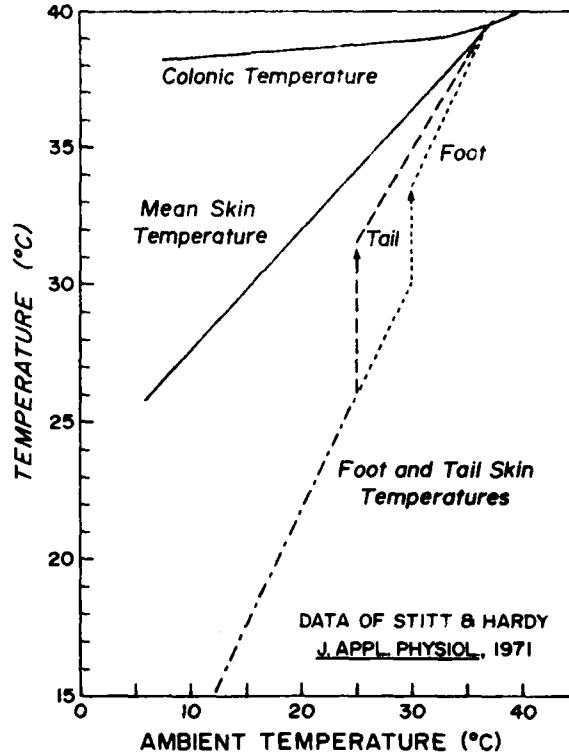

Figure 2. Body temperatures of the restrained squirrel monkey equilibrated to ambient temperatures ranging from 10 to 39 °C. Individual functions show deep colonic temperature, mean skin temperature based on four skin sites, foot, and tail skin temperatures. Arrows indicate vasodilation of vessels in tail and foot skin. Figure constructed from data in Stitt and Hardy (41).

Figure 2 illustrates the relationship between various body temperatures and T_a as measured by Stitt and Hardy (41). Mean colonic temperature, T_{co}, is regulated between 38.5 and 39.8 °C over the range of T_a from 10 to 39 °C. There is a slightly higher rate of increase above the TNZ than below. The weighted $\bar{T}_{sk}$ increases linearly with T_a; the slope of the function will be higher when the air in the test compartment is moving, rather than still. The figure also shows dramatic discontinuities in the temperature functions for foot and tail skin. These discontinuities occur at discrete T_a and reflect vasodilation of the peripheral blood vessels of the tail and foot. Warm blood from deep in the body is brought close to the skin surface when these vessels vasodilate, aiding the transfer of metabolic heat to the environment. Changes in vasomotor tonus provide the means for efficient thermoregulation in the

9

squirrel monkey across the entire TNZ. The precise T_a that produce vasodilation of the tail and foot were pinpointed by Lynch et al. (34), who measured steady-state $\bar{T}_{sk}$ of monkeys restrained in many discrete T_a. At T_a of 26.5 °C for the tail and 32 °C for the foot, these local T_{sk} varied widely, indicative of wide variation in local tissue blood flow. Similar results have recently been reported by Adair and Adams (8).

The vasomotor response is truly thermoregulatory in nature; experimental warming of the PO/AH thermoregulatory center in animals equilibrated to sub-threshold T_a produces prompt vasodilation of both tail and foot vessels (33,34,41). [a] However, the tail appears to exhibit a much greater degree of vasodilation than does the foot; the range of T_{t1} during dilation is about 7 °C, while the corresponding range for T_{ft} is about 3 °C. Since the surface area of the tail represents 11% of the total surface area of the body (cf. Eq. 11), this organ has the capability of dissipating significant amounts of metabolic heat to the environment and will play a major role in thermoregulation when animals are exposed to microwave fields at thermoneutral T_a.

It was previously stated that tissue conductance, $\underline{K}$, is an indicant of peripheral vasomotor state and represents the flow of heat from the body core to the skin. Figure 1 shows that there is a sharp increase in $\underline{K}$ at or near the lower end of the TNZ, increasing dramatically as the T_a increases. At T_a below the LCT, $\underline{K}$ is minimal and constant; in other words, the peripheral vasculature of the squirrel monkey is maximally vasoconstricted in T_a below 26 °C. It has been demonstrated that under these conditions, vasodilation of the tail can be provoked by whole-body exposure of the animal to a microwave field (8).

10

THE PROBLEM

The method of partitional calorimetry was used to generate the thermo-regulatory profile of the squirrel monkey shown in Figure 1. Steady-state temperatures to be expected in many parts of the body were also determined by this method (Fig. 2). Of particular utility is the determination, from changes in local skin temperatures, of the environmental conditions that will initiate vasodilation of the peripheral vasculature, a significant mechanism of heat loss in this species. It is important to remember, however, that the data presented in Figures 1 and 2 were collected from animals equilibrated to environments of constant temperature. When an additional source of thermal-izing energy, such as a source of radiofrequency radiation, is added to such an environment, the thermoregulatory profile and body temperatures of the subject animal may be drastically altered. Investigation of these alterations when squirrel monkeys are exposed to 2450-MHz CW microwaves at different power densities in selected thermal environments is the subject of this report.

Use of the method of partitional calorimetry to study autonomic thermo-regulatory responses of an animal exposed to microwaves permits a complete accounting of all the sources of thermal energy that influence the individual thermoregulatory mechanisms of the body. It also permits the specification of the exact means by which that thermal energy is eliminated by the body to the environment. It is possible, if sufficient T_a are explored in such an inves-tigation, that specific rates of microwave energy (SAR) may be characterized in terms of an equivalent T_a that does not contain a source of microwaves; comparable autonomic response changes under the two environmental conditions would serve as the basis for this equivalence.

The ultimate goal of research into the biological effects of exposure to radiofrequency radiation is to evaluate the impact of comparable exposure on the health and functioning of human beings. Since it is considered morally indefensible to deliberately expose humans to radiofrequency fields, it is necessary to use other means to predict potential consequences. Data derived from animal experiments have been useful in the past and will continue to be an important predictive source. For example, a major consideration in the formulation of the current American National Standards Institute (ANSI) standard (15) was the phenomenon of "work stoppage" (e.g., ref. 19). Ongoing, food-motivated behavior is reliably disrupted by microwave exposure at an SAR (4 W/kg) that elevates the internal body temperature of the behaving animal by about 1 °C. Since work stoppage has been demonstrated in several animal species, it appears to have good predictive value for the human condition.

A recent attempt by Gordon (24,25) to extrapolate to man certain thermo-physiological data derived from the laboratory mouse solely on the basis of differences in body mass was not so successful. This approach ignored the dependence of thermoregulatory processes on the prevailing T_a and the unique thermophysiological profiles of the individual species involved (13,14). Furthermore, it assigned paramount importance to possible differences between radiofrequency radiation and conventional forms of heat stress such as exer-cise or elevated T_a. It now appears highly unlikely that any SAR-vs-body mass

11

function can be given so much precision that extrapolation over several orders of magnitude will become the method of choice for predicting human thermoregulatory responses to radiofrequency radiation. The use of sophisticated simulation models of the human thermoregulatory system (44) coupled to a block model of RFR-energy deposition (22), together with the assumption that radio-frequency radiation is equivalent to other forms of thermal energy, would seem to be one alternative that is far more precise. The determination of T_a equivalents to SAR and the extrapolation of data derived from animals to man on the basis of thermoregulatory profiles would seem to be another interesting alternative. The latter approach has been explored in the research reported here.

METHODS

Subjects

Adult male squirrel monkeys (<u>Saimiri sciureus</u>) served as subjects in this study. Their estimated ages ranged from 6 to 8 yr, and their body masses ranged from 900 to 1050 g at the time of testing. They were housed individually in a colony room maintained at 24 ± 2 °C and 40 ± 10% relative humidity. All animals were well adapted to the restraining chair and most had previously participated in a variety of experiments to assess behavioral and physiological thermoregulatory capability. Some of these experiments involved brief exposures to 2450-MHz CW microwave fields at power densities at or below 15 mW/cm^2. The basic procedures for adaptation and chair training have been described by Adair et al. (12).

Test Chamber and Response Measures

During the experiments, the monkey was chair-restrained in the far field of a 15-dB standard gain horn antenna inside an electromagnetically anechoic chamber of interior dimensions 1.83 x 1.83 x 2.45 m. The interior walls were covered with 20-cm pyramidal microwave absorber (Advanced Absorber Products, Type AAP-8) to minimize reflections (<40 dB). The restraining chair was enclosed by a 30 x 33 x 78-cm box constructed of 5-cm thick closed-cell Styrofoam. Air from a temperature-controlled (±0.5 °C) source circulated at 0.36 m/s through the box in the direction shown in Figure 3. This arrangement provided a closely regulated thermal environment for the test animal. The temperature of the air inside the box (T_a) was sensed by a copper-constantan thermocouple located in the air outlet from the anechoic chamber (cf. Fig. 3) and recorded continuously on a strip chart. The temperature of the interior wall of the Styrofoam box was also recorded for comparison purposes. The monkey was under constant video surveillance during the 4- to 5-h test sessions. The test sessions were conducted in the presence of a 73-dB sound-pressure level (SPL) (re 0.0002 dyne/cm^2) masking noise to prevent auditory cues when the microwave generator was turned on and off (27).

The restraining chair was suspended from the platform of a sensitive (± 1g) Potter triple-beam balance (Potter Mfg. Co., Model No. 82) which was mounted on the roof of the anechoic chamber directly above the location of the Styrofoam test box. A Plexiglas yoke attached to the neck plate of the restraining chair accepted the suspension rod. A 10-cm length of nylon line incorporated in the suspension reduced the torque on the balance platform. A Teflon pin screwed into the base of the chair rode freely within an adjustable cardboard bushing when the chair was in place; this prevented the chair from swinging laterally when the monkey moved. A notched card attached to the base of the chair engaged a small Styrofoam block cemented to the box wall; this arrangement prevented rotational movement of the chair. These minimal-friction impediments to chair movement (diagrammed in Fig. 4) permitted very accurate recordings of total body weight loss during the experimental sessions, an example of which is shown in Figure 5.
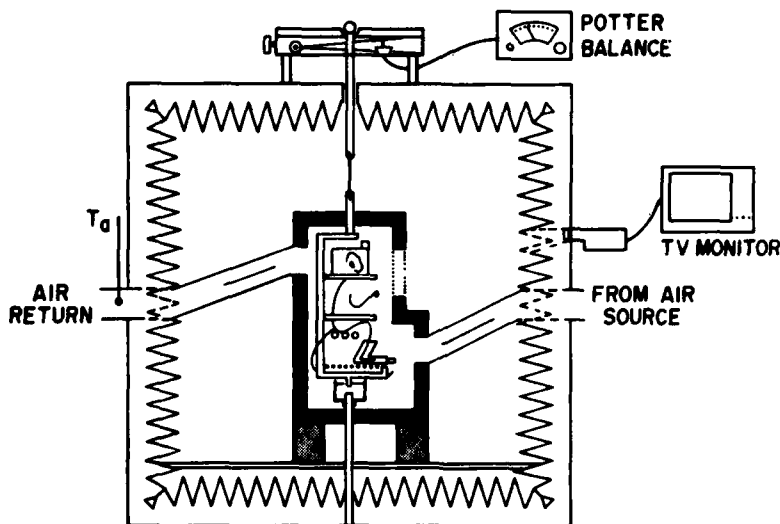
13

Figure 3. Schematic diagram (as viewed from the horn antenna) of the convective system that provides climate conditioning of the animal's test box inside the anechoic chamber. The system for suspending the animal's restraining chair from a sensitive Potter balance is also shown. Constant video surveillance of the animal is possible through a window in the wall of the test box.

During the experimental test sessions, the following body temperatures were sampled at 1-min intervals by an on-line computer: T_{co} and four representative $\bar{T}_{sk}$ taken from the abdomen, tail, leg, and foot (34). All were measured with 36-gauge copper-constantan thermocouples having a reference junction in a bath of melting ice and water (2,41). All thermocouple junctions were very small and constructed in configurations appropriate to the locus of application. All $\bar{T}_{sk}$ were air-skin interface temperatures: the thermal junction was in contact with the skin on one side and open to the air on the other. To minimize perturbation of the microwave field, the lead wires were shielded and held out of alignment with the electric vector of the incident planewave. Any thermocouple electromotive force (emf) that showed evidence of electrical artifacts (i.e., abrupt voltage changes greater than 4μV, equivalent to 0.1 °C, correlated with microwave onset or termination) was discarded as an inadmissible datum. A weighted $\bar{T}_{sk}$ was calculated from the four skin temperatures by the relation given in Equation 11 (43).

Oxygen consumption ($\dot{V}_{O2}$) was measured using an open-flow draw system. Chamber air was drawn at a constant rate of 7 L/min through a Plexiglas hood over the monkey's head and thence outside the chamber through Teflon tubing. The arrangement was similar to that depicted in Adair (2, Fig. 1). The oxygen

partial pressure (P_{O_2}) deficit was measured downstream by a Beckman Model 755 paramagnetic oxygen analyzer that sampled the passing airstream at a rate of 0.3 L/min. Metabolic heat production (M) was calculated from $\dot{V}_{O_2}$ assuming a constant RQ of 0.83 (31,40). This value of the RQ was checked by simultaneous measurement of carbon dioxide production with a Beckman Model 149148-LB2 medical gas analyzer during a few isolated tests. However, the latter instrument was not used routinely during experimental test sessions because it was difficult and time consuming to calibrate.
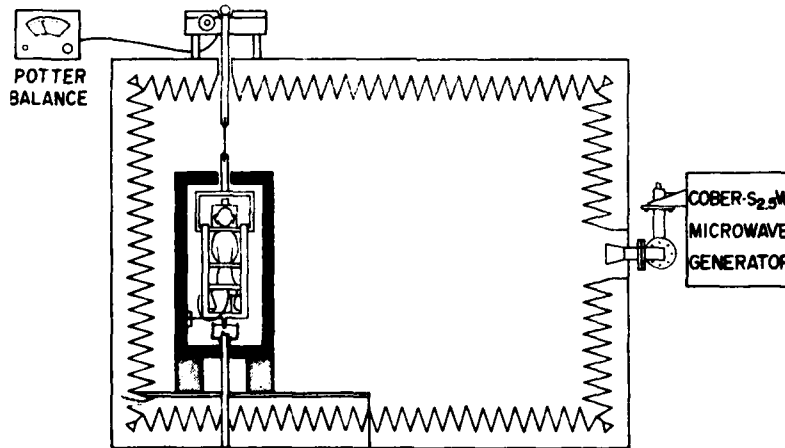
Figure 4. Schematic diagram (left elevation) of the location of the animal's test box inside the anechoic chamber. Box is located in the far field of the horn antenna. Additional details of the system for suspending the animal's restraining chair from the Potter balance are also shown.

To measure minute-to-minute E_{res}, a portion of the expired air was drawn through a dewpoint temperature sensing device developed at the Pierce Foundation Laboratory (26). From the dewpoint temperature (T_{dp}), which was measured and recorded continuously, the vapor pressure of water in the sampled air was calculated by means of Antoine's equation (46):

$$P_{H_2O} = e^{a - b\,(T_{dp} + c)^{-1}} \tag{12}$$

where: P_{H_2O} = water vapor pressure (mm Hg)

a = 18.67
b = 4030.18   empirical constants for water
c = 235

The water evaporated from the lungs, or E_{res}, was then calculated from a modified gas equation:

$$E_{res} = \frac{(M.W.)\ (\Delta P_{H_2O})\ AF}{R \cdot T_a} \quad g/min \qquad (13)$$

where: M.W. = molecular weight of water
P_{H_2O} = the difference in water vapor pressure in air before and after evaporation of water
AF = air flow (L/min)
R = gas constant 62.396
T_a = air temperature (°K)
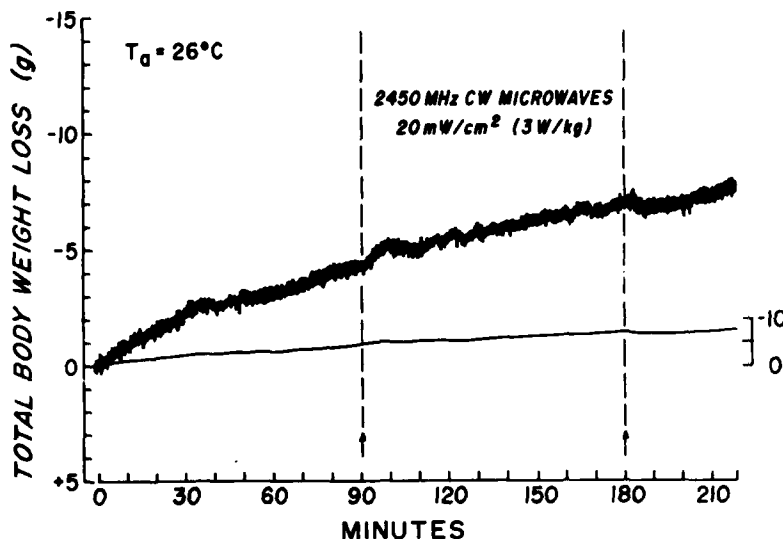
Figure 5. Representative strip chart record of total body weight loss measured by the Potter balance during a single 220-min experiment on one monkey. After a 90-min equilibration to an ambient temperature (T_a) of 26 °C, monkey was exposed to 2450-MHz CW microwaves at a power density of 20 mW/cm^2 (SAR=3 W/kg) for 90 min. Two tracings are shown, representative of two recorder sensitivities.

Thermoregulatory sweating from the right foot was also measured with the same kind of dewpoint temperature sensing device. The monkey wore an L-shaped Plexiglas boot with the sole of the foot resting on a nylon support. Chamber air was drawn through the boot, at the rate of 1.9 L/min, and thence outside the chamber through Tygon tubing where the T_{dp} was measured and recorded continuously. Sweating rate ($\dot{m}_{sw}$) was calculated in the same manner as E_{res}.

Microwave Source, Field Measurements, and Dosimetry

Continuous microwaves of a single frequency, 2450±25 MHz, were generated by a commercial source (Cober Electronics Model S2.5W) and fed to the antenna through standard waveguide components. The electric vector of the incident planewave was aligned with the long axis of the animal's body (E-polarization). Generator forward power capability ranged from 190 W to 2.5 kW, which yielded a range of incident power densities from 2.5 to >100 mW/cm^2 as measured in the far field at the location of the monkey's head.

Calibration measurements of the far field were made with a Narda model 8316B broadband isotropic radiation detector fitted with a model 8323 probe. At a constant 1.0-kW forward power, the microwave field was mapped at 12-cm intervals across a 1.0 x 1.5-m plane that passed through the center of the restraining chair location (1.85 m from the antenna's leading edge) orthogonal to the direction of propagation of the incident planewave. The maximum field nonuniformity of the central 50 x 50 cm of this plane, encompassing the monkey location, was 8% with the chair absent and 13% with the chair present. Insignificant perturbations of the field were introduced by the hood, sweat boot, fine thermocouples, and the suspension rod for the restraining chair. In this regard, Ho (30) has demonstrated that field perturbations produced by such devices may not be as important as other variables (e.g., animal size, configuration and movement, polarization and uniformity of the incident field).

A rough assessment of the whole-body SAR produced by power densities from 5 to 40 mW/cm^2 was based on temperature increments measured in three sizes of saline-filled cylindrical Styrofoam models (0.75-, 1.1-, and 1.5-L volumes). The inside height of all cylinders was 33 cm, and the inside diameters were approximately 5, 6.5, and 8 cm. Tests on each model were conducted as follows: A cylinder filled with physiological saline was placed at the animal's position inside the anechoic chamber and equilibrated for 16 h to a circulating T_a of 35 °C. The hot junctions of four 36-gauge copper-constantan thermocouples were positioned on the vertical axis of the cylinder (equidistant from the antenna) at different depths within the liquid. The wire leads were led through the cylinder wall in alignment with the direction of propagation of the electromagnetic wave (K vector) and carefully shielded. Reference junctions were placed in a bath of melting ice and water located outside the anechoic chamber. The four temperatures were recorded continuously during a series of 10-min microwave exposures to 5, 10, 20, 30, and 40 mW/cm^2. Individual exposures were separated by at least 2-h restabilization periods. For the 1.1-L model, the mean temperature increment in the liquid ranged from 0.1 °C at 5 mW/cm^2 to 0.6 °C at 40 mW/cm^2, yielding calculated SAR from 0.5 to 5.8 W/kg. For all three models, the average SAR was determined to be 0.15 W/kg per mW/cm^2. Temperature increments were determined twice in the 0.75-L model, once standing alone and once inserted into the restraining chair, to assess the effects of the chair on whole-body energy absorption. No differences were found between the SARs determined from the two sets of measurements.

This rough assessment of SAR, yielding a value comparable to that predicted for the squirrel monkey by Durney et al. (20), has been confirmed by three different independent procedures. First, as reported by Adair and Adams (9), the steady-state reduction in M of cold-exposed squirrel monkeys, exposed for 90 min to a controlled microwave field is exactly equal (in W/kg) to the

SAR of the imposed field. Second, as reported by Adair (4), conscious squir-
rel monkeys equilibrated to a T_a just below the UCT (e.g., 33 °C) can be used
as adjunctive dosimeters. The SAR can be determined from T_{co} increments
during 10-min microwave exposures in animals that are fully vasodilated but
not sweating. If the body mass of the monkey is 0.9 to 1.0 kg, the SAR so
determined will be 0.15 W/kg per mW/cm^2. Third, the whole-body SAR was
determined in our exposure facility by J.B. Kinn (32), who used the method of
twin-well calorimetry. A model of a seated squirrel monkey, filled with 0.76
kg of tissue-equivalent material (28), was exposed, at the monkey's location,
for 10 min at a power density of 20 mW/cm^2. Three separate determinations
under these conditions yielded a whole-body SAR of 0.18 W/kg per mW/cm^2, a not
unreasonable value considering that the mass of the model was somewhat less
than that of most of our animal subjects.

Experimental Design

Three T_a were selected for study: 20, 26, and 32 °C. These T_a were
determined on the basis of pilot experiments to be representative of particu-
lar portions of the thermoregulatory profile of the squirrel monkey (cf. Fig.
1) that would exhibit maximal thermoregulatory alteration in the presence of
an imposed microwave field. A T_a of 20 °C is well below the TNZ so that
microwave exposure should provoke a reduction of $\underline{M}$ and perhaps, at higher
power densities, initiate vasodilation of the tail vessels (cf. Fig. 2). A T_a
of 26 °C is just below the LCT so that microwave exposure should initiate
vasodilation of the tail vessels and perhaps, at higher power densities,
initiate vasodilation of the vessels in the foot. A T_a of 32 °C is close to
the UCT so that microwave exposure should initiate vasodilation of the foot
(if it had not already occurred) and initiate thermoregulatory sweating at
higher power densities.

At each T_a, the thermoregulatory consequences of exposure to 4 discrete
power densities (10, 15, 20, and 25 mW/cm^2) were explored. These power
densities represent whole-body SARs of 1.5, 2.25, 3.0, and 3.75 W/kg, respec-
tively. At a T_a of 32 °C, it was not possible to expose the animals to the
highest power density because they tended to develop hyperthermia over the
course of the 90-min microwave exposure; it will be remembered that a thermo-
regulatory steady state is required in order to employ the method of parti-
tional calorimetry. At all T_a, data were collected on three or four monkeys,
a single replication at $T_a=20$ °C and two replications at the other two T_a.

Each experimental test session on a single monkey involved equilibration
of the animal for a minimum of 90 min to the prevailing T_a, followed by a
90-min exposure to 2450-MHz CW microwaves at a particular power density. A 20
- 30 min reequilibration period terminated the test session. Strict criteria
for the acceptability of all data were established prior to the conduct of any
experiments: any test session in which a thermoregulatory steady state was
not achieved, in either the initial equilibration period or during microwave
exposure, was discarded. Malfunctions of test equipment, on-line computer,
etc. were also grounds for discarding entire test sessions. Over the course
of the research project, approximately 40% of the conducted tests were dis-
carded on these grounds.

In summary, the following independent variables were measured in each experimental test session:

colonic temperature	T_{co}
4 skin temperatures	T_{sk}
abdomen	T_{abd}
tail	T_{tl}
leg	T_{lg}
foot	T_{ft}
mean skin temperature	$\bar{T}_{sk}$
oxygen consumption	$\bar{M}$
T_{dp} of expired air	$\bar{E}_{res}$
body mass	$\bar{E}_{tot}$
T_{dp} of foot capsule air	$\dot{m}_{sw}$

All experimental test sessions were conducted in the morning so as to avoid possible circadian shifts in resting levels of thermoregulatory processes. At the start of the test, approximately 18 h had elapsed since the monkey's last meal. Prior to introduction of the animal into the test environment, all equipment was checked and/or calibrated and a stable T_a was established in the test chamber. The chaired monkey was weighed on an electronic analytical platform balance before being instrumented with thermocouples, boot, hood, etc.; a post-test weight served to check the accuracy of the total body weight loss measured during the course of the test. After the chaired animal had been suspended from the Potter balance inside the test compartment and all connections made to the measuring equipment, 80 ml of mineral oil was introduced into a trap in the base of the restraining chair; this prevented evaporation of urine and feces during the experiment. The weight loss was tared to zero immediately before the start of the 90-min equilibration period. At specific times during the test session, 5-min baseline checks of the T_{dp} and O_2 content of the chamber air were made to track any possible drifts in baseline levels. Throughout the test session, all data were sampled at 1-min intervals by an on-line computer. In addition, continuous strip-chart records were made of total body weight loss, T_{dp} of the expired air, T_{dp} of foot capsule air, and T_a. At the end of the test session, after the animal had been removed from the test compartment and returned to the home cage, a 10 to 20-min baseline check of the physical characteristics of chamber air completed the test data.

Representative Data from Individual Experiments

The data from individual test sessions were analyzed in 5-min time bins, i.e., means and standard errors of each dependent variable were calculated at 5-min intervals across the duration of the test session. Sample experiments for each of the three test T_a at a microwave power density of 15 mW/cm^2 (SAR=2.25 W/kg) are shown in Figures 6, 7, and 8. Data from three different monkeys are represented in these three figures.

Figure 6. Representative experiment on one monkey equilibrated to an ambient temperature (T$_a$) of 20 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 15 mW/cm^2 (SAR=2.25 W/kg). Individual plotted points represent means of preceding 5 min.
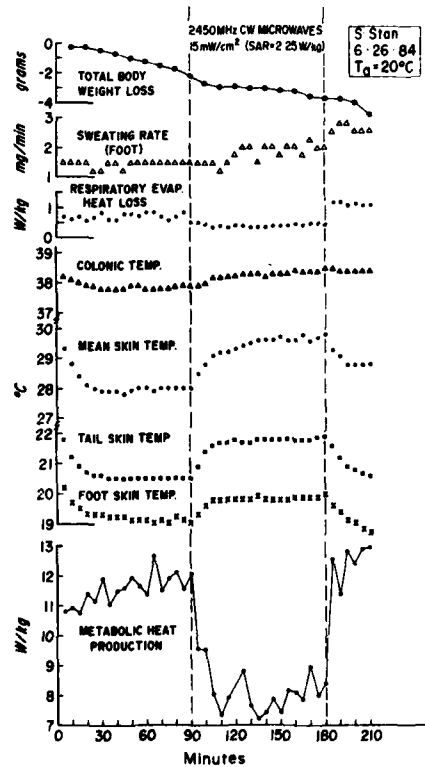
Figure 7. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 26 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 15 mW/cm² (SAR=2.25 W/kg). Individual plotted points represent means of preceding 5 min.

Figure 6 presents data from one monkey equilibrated to a T_a of 20 °C and then exposed for 90 min to microwaves at a power density of 15 mW/cm². During the equilibration period, metabolic heat production (M) was elevated to 11.7 W/kg, skin temperatures stabilized at low levels indicative of vasoconstriction, T_{co} stabilized at just under 38 °C, and evaporative heat loss was minimal. At the initiation of microwave exposure, a substantial reduction in M occurred together with passive increases in the temperatures of skin and body core. The rate of total body weight loss appeared to decrease during the period of microwave exposure, increasing again when the microwave field was extinguished. The primary consequence of the microwave exposure at this T_a was a reduction in the animal's heat production.
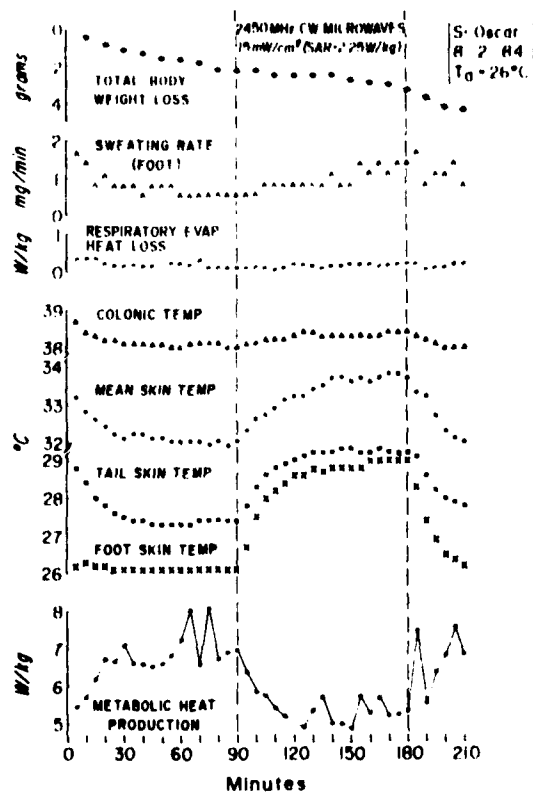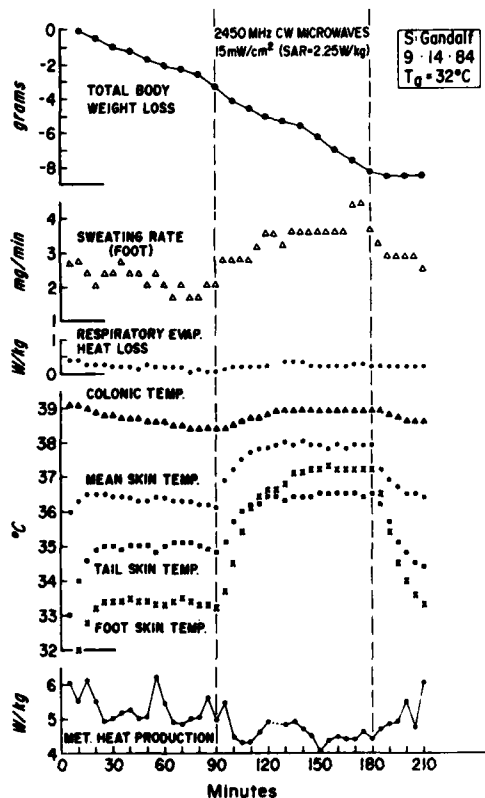
21

Figure 8. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 32 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 15 mW/cm^2 (SAR=2.25 W/kg). Individual plotted points represent means of preceding 5 min.

Figure 7 presents data for another monkey equilibrated to a T_a of 26 °C and then exposed for 90 min to microwaves at the same power density, 15 mW/cm^2. During the equilibration period, $\underline{M}$ was only slightly elevated above the resting level, skin temperatures stabilized at low levels indicative of vasoconstriction, T_{co} stabilized at 38 °C, and evaporative heat loss was minimal. At the initiation of microwave exposure, $\underline{M}$ fell slightly to the resting level, significant increases occurred in the temperature of foot and tail skin, and other variables showed little or no change. The primary consequence of microwave exposure at this T_a was an increase in T_{sk} of the extremities, indicative of vasodilation, and a slight decrease in $\underline{M}$.

Figure 8 presents data for a third monkey equilibrated to a T_a of 32 °C and then exposed for 90 min to microwaves at the same power density, 15 mW/cm^2. During the equilibration period, $\underline{M}$ stabilized at the resting level

22

($\sim$5 W/kg), the local skin temperatures indicated that the tail skin was vasodilated but that the foot skin was not, T_{co} stabilized at 38.4 °C, and evaporative heat loss was minimal. At the initiation of microwave exposure, a substantial increase in T_{ft} occurred that was indicative of vasodilation, and sweating was initiated from the foot. These increases in heat loss were mirrored by an increase in the rate of total body weight loss. All of these trends were curtailed or reversed when the microwave field was extinguished. A remarkable feature of each of these experiments was the stability of T_{co} during the period of microwave exposure. It is of note that the SAR of 2.25 W/kg (at 15 mW/cm^2) was equivalent to approximately 50% of the resting heat production of the squirrel monkey at thermoneutrality.

A second series of sample experiments is shown in Figures 9, 10, and 11. All of these experiments were conducted at the same T_a (26 °C), as was the experiment shown in Figure 7, which completes the series, but the power density in each case was different. Inspection of these four figures indicates how the magnitude of individual thermoregulatory responses is directly influenced by the intensity of the microwave exposure when that exposure occurs at the same T_a.

Even though the four figures present data collected on three different monkeys, the equilibrated level of $\underline{M}$, before the onset of microwave exposure, was quite similar from animal to animal, 6.5 to 8.5 W/kg. Since this level is only 2 to 3 W/kg above the resting level (Fig. 1), subsequent microwave exposure, at even the lowest power density (10 mW/cm^2) was sufficient to reduce $\underline{M}$ to the resting level, higher power densities provoking dramatic changes in other thermoregulatory responses.

Of the remaining thermoregulatory responses, the greatest alterations occurred in the temperature of certain skin areas, notably the tail and foot. It is clear, from the steady-state levels of T_{tl}, that full vasodilation of this area occurred at a power density of 20 mW/cm^2 and above, while, in most cases, partial vasodilation of the foot, as indexed by T_{ft}, occurred at these same power densities. Little or no change was evident in the regulated level of T_{co} across the range of power densities explored. Foot sweating was initiated in the monkey exposed at 25 mW/cm^2 (Fig. 11) but not at lower power densities, and no change occurred in respiratory evaporative heat loss during any experiment. There is a suggestion of a reduction in the rate of total body weight loss during the 90-min periods of microwave exposure at this T_a, an effect that may result from the reduced $\underline{M}$. During the periods when the microwave field was present, the animals were observed to sit much more quietly than during the equilibration period, often with eyes closed as though asleep.

To summarize, the individual experiments depicted in Figures 7, 9 10, and 11 show that when squirrel monkeys are equilibrated to a T_a just below the LCT (26 °C), microwave exposure will first reduce $\underline{M}$ to the resting level and then, at higher power densities, will initiate vasodilation of first the tail and then the foot. Foot sweating may also be initiated if the power density is sufficiently high, although this response may only occur in certain animals that may be classified as "efficient" sweaters (cf. ref. 3).
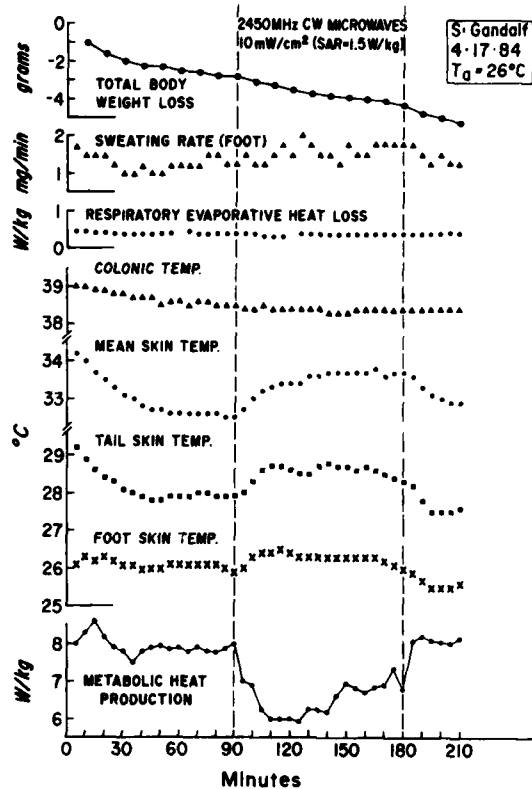
Figure 9. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 26 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 10 mW/cm² (SAR=1.5 W/kg). Individual plotted points represent means of preceding 5 min.

Steady-state Thermoregulatory Responses
Measured During Microwave Exposure at Three T_a

Each experiment yielded two values of each dependent variable that could be used for further analysis: the first value represented the steady-state level just prior to the onset of microwave exposure when the animal was fully equilibrated to the prevailing T_a, and the second represented the steady-state level when the microwave field was present. To obtain these values, means and standard errors were calculated across the final 20 min of the initial equilibration period and across the final 20 min of the 90-min period of microwave exposure. All of the means, for all animals at all T_a, are summarized in Figures 12 through 18. In each figure, particular dependent variables (e.g.,

$\underline{M}$, $\underline{T}_{co}$) or calculated variables (e.g., $\underline{h}$, $\underline{K}$) are plotted as a function of both power density (mW/cm^2) and SAR (W/kg).
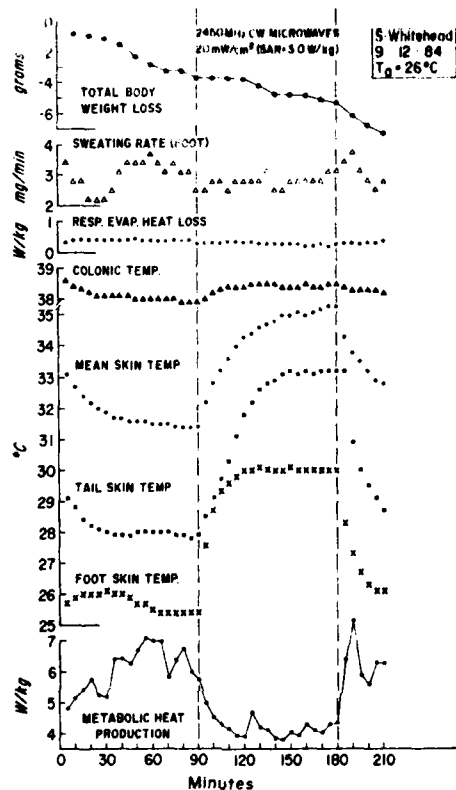

Figure 10. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 26 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 20 mW/cm^2 (SAR=3.0 W/kg). Individual plotted points represent means of preceding 5 min.

Figures 12 and 13 summarize all data collected at a T_a of 20 °C. Each point plotted at zero power density represents the initial equilibrated value for one monkey, coded by symbol, in a single experiment; the steady-state value of that variable during microwave exposure is plotted at the appropriate power density. In Figure 12, we see that little change occurs in T_{co} or $\overline{T}_{sk}$ when the microwave field is present; the major change is a reduction in $\underline{M}$ that is a linear function of the field strength. That the $\underline{M}$ reduction is of the same magnitude as the SAR is clear because the open symbols (representing $\underline{M}$ + $\underline{A}_{rfr}$) fall along a horizontal line that lies at the initial equilibrated level. This finding confirms our earlier report of the equivalence between $\underline{M}$ reduction and SAR (9) and further supports the validity of our dosimetric measures. Figure 13 shows that the heat transfer coefficient ($\underline{h}$) does not

25

change as a function of power density and, indeed, has a value that is nearly identical to that calculated by Berglund (16) on the basis of physical measurements made in our test facility. Similarly, tissue conductance, indicative of changes in vasomotor tonus, changes little as power density increases. There is only a slight rise at the 25 mW/cm^2 power density when the absorbed energy (A_{rfr}) is added to M during the calculation of K (represented by open symbols).
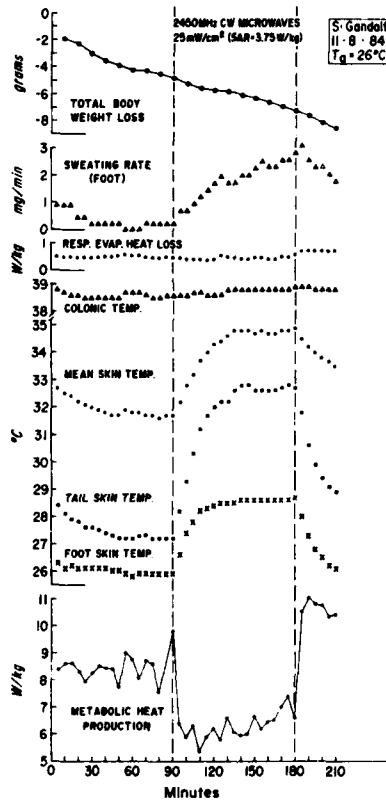

Figure 11. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 26 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 25 mW/cm^2 (SAR=3.75 W/kg). Individual plotted points represent means of preceding 5 min.

Figures 14 and 15 summarize the data collected at T_a=26 °C for all animals. Once again, great stability of T_{co} is evident during microwave exposure, but a slight increase of $\overline{T}_{sk}$ occurs as the power density increases. A reduction in M is associated with microwave exposure, but it is of small magnitude because the initial equilibrated level, at 0 power density, is only 2 to 3 W/kg above the resting level. When A_{rfr} is added to M, the sum increases slightly at the higher power densities. It is this augmented rate of ener-

26

production/absorption that is responsible for the increase in tissue conductance seen in Figure 15. Once again, the heat transfer coefficient is constant at all power densities and is nearly identical to Berglund's value of 12.3 $(W/m^2)/°C$. At a T_a of 26 °C, the principal effect of microwave exposure is an increase in thermal conductance.
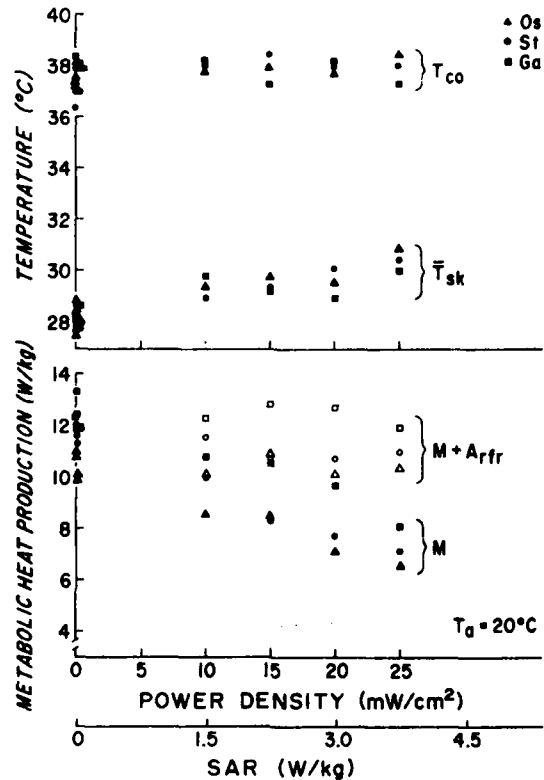

Figure 12. Mean steady-state colonic (T_{co}) and weighted mean skin ($\bar{T}_{sk}$) temperatures and metabolic heat production ($\underline{M}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 20 °C. $\underline{A}_{rfr}$ = absorbed radiofrequency radiation (W/kg). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

Figures 16, 17, and 18 summarize the data collected at T_a=32 °C for all animals. At this T_a, we begin to see a slight increase in T_{co} as the power density of the microwave field increases. However, at no time was any animal unable to achieve thermal equilibrium in the presence of the microwave field as long as the power density was 20 mW/cm^2 or below. It was previously stated (cf. Experimental design) that the 25 mW/cm^2 power density was not presented at this T_a because the animals tended to develop hyperthermia. The basis for this result is evident in Figure 16; the $\bar{T}_{sk}$, increasing regularly with power density, approaches the T_{co} at this power density. Figure 2 shows that, under

normal conditions, $\bar{T}_{sk} = T_{co}$ at $T_a = 37$-38 °C; thus, we may state that for the squirrel monkey, a microwave exposure at 25 mW/cm² in a T_a of 32 °C may be equivalent, in terms of changes in body temperatures, to an exposure to a T_a of 37-38 °C alone.

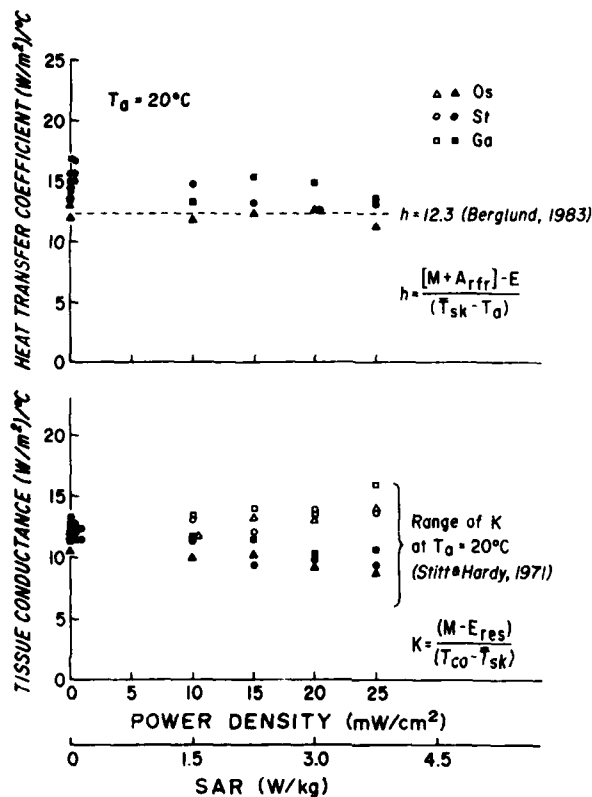Figure 13. Mean steady-state values of the heat transfer coefficient ($\underline{h}$) and tissue conductance ($\underline{K}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 20 °C. Open symbols represent ($\underline{M} + \underline{A}_{rfr}$). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

The heat transfer coefficient during microwave exposure, when $T_a = 32$ °C, remains close to Berglund's value (Fig. 17), but tissue conductance increases dramatically as shown in Figure 18. This is not only due to an increase in heat storage (see data for $\underline{M} + \underline{A}_{rfr}$ in Fig. 16), but also to the narrowing of the difference between T_{co} and $\bar{T}_{sk}$, the denominator of the $\underline{K}$ equation. Values of K as high as 60 (W/m²)/°C were recorded by Stitt and Hardy (41) at $T_a = 37$ - 38 °C, as can be seen in Figure 1. Thus, we have a second basis for the equivalence determined above, that a microwave exposure at 25 mW/cm² in a T_a of 32 °C may be equivalent to an exposure to a T_a of 37-38 °C alone.
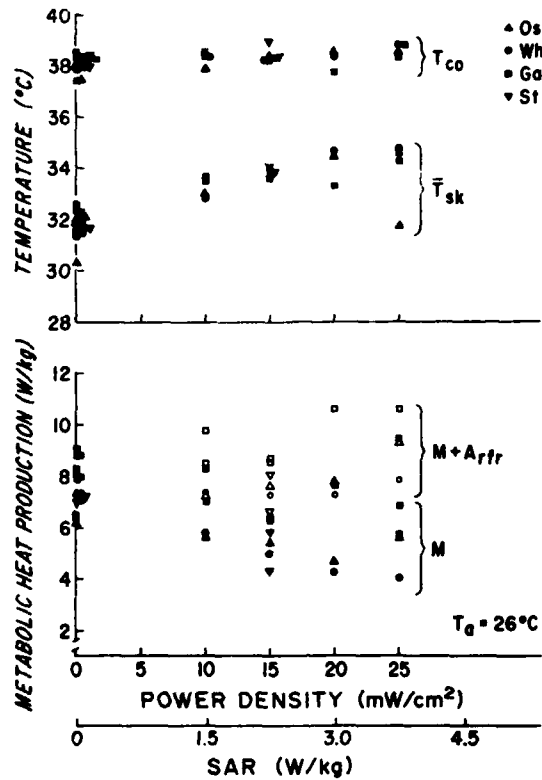
28

Figure 14. Mean steady-state colonic (T_{co}) and weighted mean skin ($\bar{T}_{sk}$) temperatures and metabolic heat production (M) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 26 °C. A_{rfr} = absorbed radiofrequency radiation (W/kg). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

A plot of dry heat losses as a function of the skin-to-ambient temperature gradient for all data collected on all animals appears in Figure 19. In the figure, solid symbols represent steady-state data collected during the initial equilibration period when no microwaves were present and the unfilled symbols, coded in terms of power density, represent steady-state data collected during microwave exposure. The line of best fit, calculated by the method of least squares, is equal to the heat transfer coefficient (h) and has a slope of 12.77 $(W/m^2)/°C$, a value very close to the 12.3 $(W/m^2)/°C$ calculated by Berglund (16). Since there can be no radiant or convective heat flow from the surface of the animal when ($\bar{T}_{sk} - T_a$) = 0, the function should pass through the origin. That this is very nearly the case (y=3.45 when x=0) confirms that there were no systematic errors in the partitional calorimetry employed in this study. Of even greater importance is the fact that a single

29

function describes all the data collected from microwave and non-microwave conditions alike. This fact indicates that the thermoregulatory system deals with energy absorbed from radiofrequency fields in exactly the same way as energy produced by the body during normal metabolic processes. In other words, heat generated in body tissues by absorbed microwaves should be regarded as no different from that deposited by more conventional (radiative or convective) sources or from excess metabolic heat produced during physical exercise.
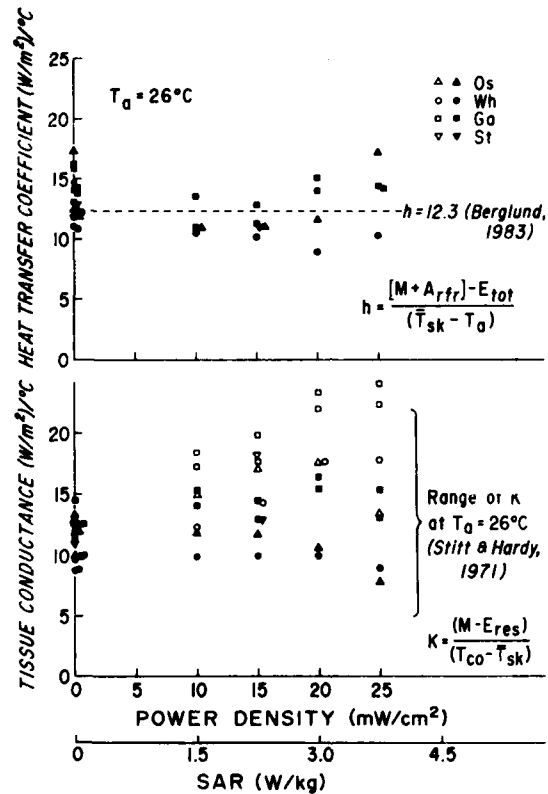

Figure 15. Mean steady-state values of the heat transfer coefficient ($\underline{h}$) and tissue conductance ($\underline{K}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 26 °C. Open symbols represent ($\underline{M} + \underline{A}_{rfr}$). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

Changes in Thermoregulatory Responses
During Microwave Exposure

In Figures 12 through 18, the points plotted at zero power density represent the steady-state level of each dependent variable just prior to the onset of microwave exposure when the animals were fully equilibrated to the prevailing T_a. At the three T_a selected for study, 20, 26, and 3.

30

measured values fall within the range reported by Stitt and Hardy (41), as summarized in Figures 1 and 2, and may thus be considered normal values for the squirrel monkey. It is useful to determine the change in each thermoregulatory response from the normal level that occurs during microwave exposure in order to gain insight into possible thermoregulatory aberrations that may be produced by microwaves.
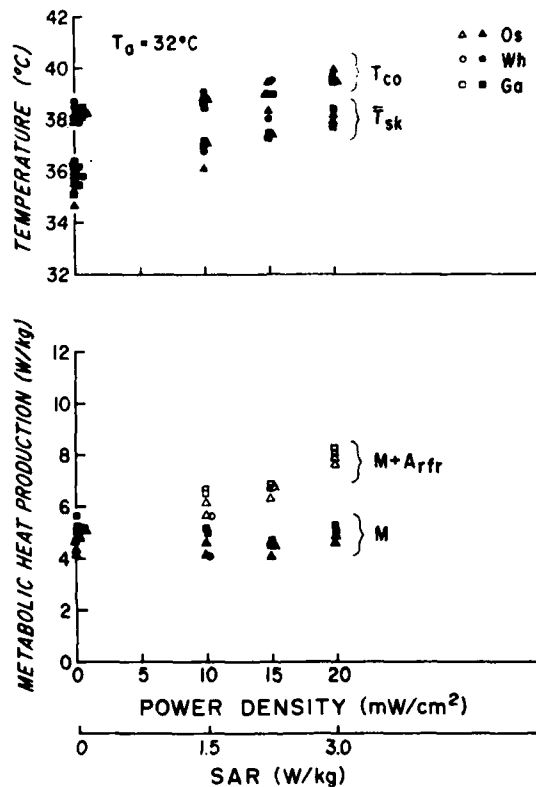

Figure 16. Mean steady-state colonic (T_{co}) and weighted mean skin ($\tilde{T}_{sk}$) temperatures and metabolic heat production ($\underline{M}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 32 °C. $\underline{A}_{rfr}$ = absorbed radiofrequency radiation (W/kg). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

With the exception of the calculated tissue conductance at $T_a=32$ °C (Fig. 18), all of the data derived from individual animals (Figs. 12 through 17) are very similar. Therefore, grand means were calculated across animals as representative values for each exposure condition. Grand means were also calculated across animals for the normal equilibrated responses at each T_a (data plotted at zero power density). The differences between these means represent the change in each measured dependent variable that occurred during microwave exposure. Figures 20, 21, and 22 summarize these calculated mean

31

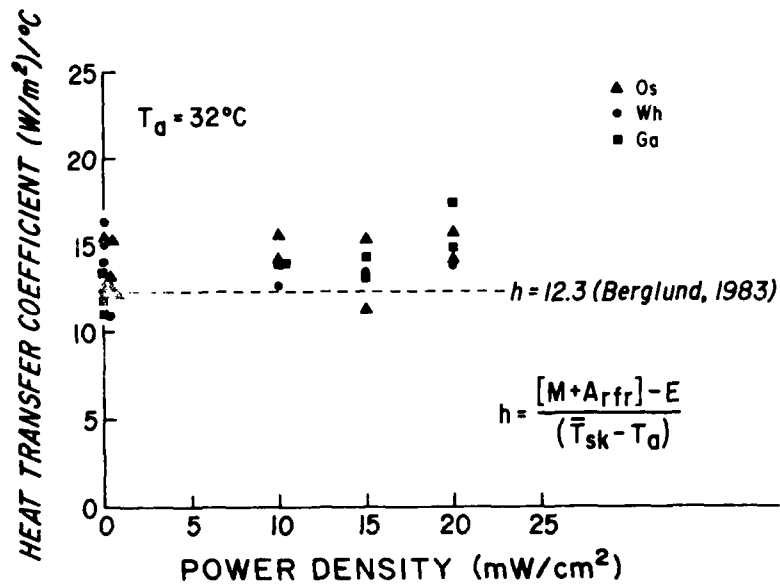changes in thermoregulatory responses as a function of the power density of the imposed microwave field.

Figure 17. Mean steady-state values of the heat transfer coefficient ($\underline{h}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 32 °C. Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

The mean changes in T_{co} and in $\bar{T}_{sk}$ are shown in Figure 20 for all power densities and the three exposure T_a. With the sole exception of the 20 mW/cm² exposure at T_a=32 °C, colonic temperature sustained a slight rise (0 < T_{co} < 0.66 °C) during all microwave exposures. This rise was as likely to occur at T_a=20 °C as at T_a=32 °C. The large increase in T_{co} during microwave exposure at 20 mW/cm² when T_a=32 °C, indicates that thermoregulatory mechanisms were beginning to break down under these conditions, i.e., that the mobilized heat loss responses were insufficient to eliminate the heat stored in the body. This condition has been previously discussed.

The lower panel of Figure 20 shows the change in $\bar{T}_{sk}$ during microwave exposure as a function of power density. The greatest changes occurred when T_a=26 °C, a result that is not unexpected since vasodilation of specific skin areas was most easily initiated at this T_a by the specific power densities employed. Considerable passive heating of the skin occurred at all T_a, however, and the change in $\bar{T}_{sk}$ was a direct function of power density.
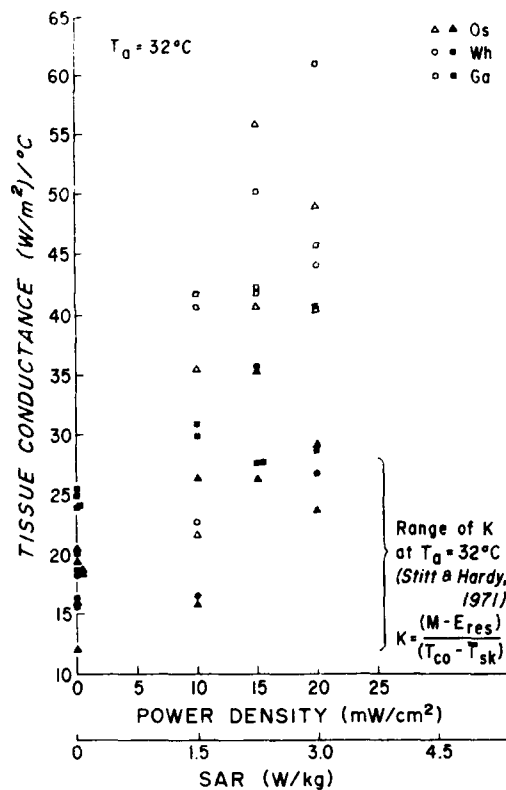
Figure 18. Mean steady-state values of tissue conductance ($\underline{K}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 32 °C. Open symbols represent ($\underline{M} + \underline{A}_{rfr}$). Points plotted at zero power density represent equilibrated levels prior to microwave exposure.

The mean changes in metabolic heat production and in sweating rate are shown in Figure 21 for all power densities and the three exposure T_a. No change in $\underline{M}$ occurred at any power density when $T_a = 32$ °C; this finding was anticipated because at this T_a $\underline{M}$ is at the resting level (cf. Fig. 1) and therefore could not be further reduced. Dramatic reductions in $\underline{M}$ occurred at $T_a = 20$ °C, that were a linear function of power density. This finding confirms that already published by Adair and Adams (9). The intermediate $\underline{M}$ reduction at $T_a = 26$ °C, also a linear function of power density, would not have been predicted from the thermoregulatory profile of the squirrel monkey presented in Figure 1. The $\underline{M}$ function in this profile is depicted as being low and flat across the entire width of the TNZ. A calculated mean reduction in $\underline{M}$ produced by microwave exposure of animals equilibrated to T_a close to the LCT may indicate individual variability in LCT from animal to animal as well as individual variability in metabolic functioning.
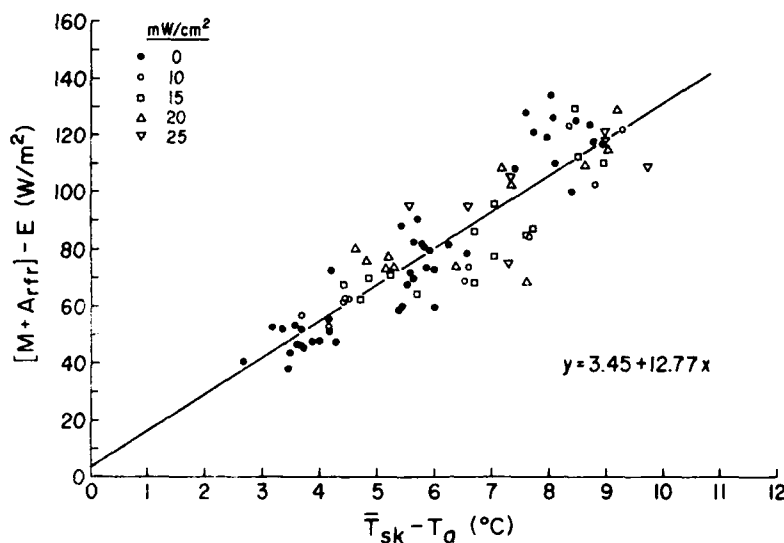
33

Figure 19. A plot of dry heat losses ($[\underline{M} + \underline{A}_{rfr}] - \dot{E}$) for all animals as a
function of the skin-to-ambient temperature gradient ($\bar{T}_{sk} - T_a$).
The function was fitted by the method of least squares.

Sweating rate from the right foot was measured continuously during all
experimental test sessions as an index of evaporative heat loss. In the
determination of heat balance (Eq. 1), heat lost through sweating (E_{sw})
together with respiratory evaporative heat loss (E_{res}) are combined in the
term $\underline{E}$. In the method of partitional calorimetry, E_{sw} is determined in-
directly as ($E_{tot} - E_{res}$). It is often useful, however, to have an indepen-
dent means of assessing the onset and rate of thermoregulatory sweating. As
for the other dependent variables, mean sweating rate was calculated for the
final 20 min of the period of equilibration to the prevailing T_a and the final
20 min of the 90-min period of exposure to microwaves in each experimental
test session. Grand means were then calculated across animals, and the mean
change in sweating rate as a function of power density is shown in the lower
panel of Figure 21. No significant change in sweating occurred at any power
density in the two cooler environments (T_a=20 and 26 °C). A notable increase
in sweating rate was stimulated by microwave exposure at a power density of 15
mW/cm² and above in animals equilibrated to T_a= 32 °C.

A similar pattern of response change is evident in Figure 22 which shows
the mean change in tissue conductance as a function of power density for all
T_a. A slight reduction in conductance as power density increased was evident
at T_a - 20 °C presumably because $\bar{T}_{sk}$ rose and T_a did not. A slight increase
in conductance, moderated at 25 mW/cm², occurred when T_a=26 °C, while a large
increase occurred at all power densities in the 32 °C environment. It should

34

be noted that the value $\underline{M}$, not $(\underline{M} + \underline{A}_{rfr})$, was used for the calculations of $\underline{K}$ that contributed to Figure 22; had the latter values been used, the calculated changes in $\underline{K}$ would have been generally larger.
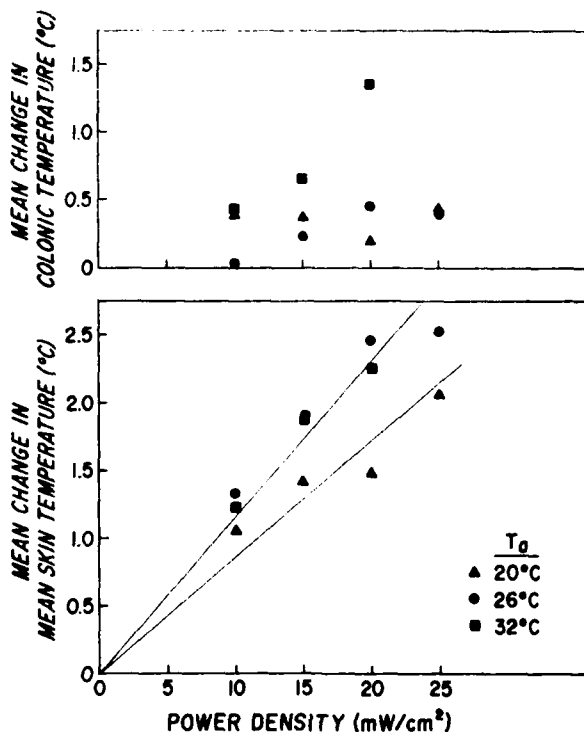

Figure 20. Mean change from equilibrated baseline level in colonic temperature (top panel) and weighted mean skin temperature (bottom panel) as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

Useful information about the interaction between individual variables can often be derived by expressing one as a function of others. For example, it would be of interest to know if the measured changes in thermoregulatory function of squirrel monkeys exposed to 2450-MHz CW microwaves are due primarily to thermal stimulation of receptors in the skin or of those deeper in the body. At this microwave frequency, the incident radiation may be absorbed as deeply as 2.5 - 3.0-cm below the skin surface. There is a strong possibility that thermosensitive sites of the CNS (e.g., the PO/AH, the midbrain, the medulla, the spinal cord) could provide the neural signals for thermoregulatory response change during microwave exposure. Figure 23 shows the mean change in metabolic heat production from the equilibrated baseline level plotted both as a function of the associated change in mean skin temperature (top panel) and change in colonic temperature (bottom panel). It should be noted that since these are steady-state data, the sluggishness of the deep-body temperature is not an important factor in this comparison. There is a clear indication, from inspection of the two panels, of an orderly relationship between delta $\underline{M}$ and delta $\overline{T}_{sk}$ that would implicate the thermal receptors of the skin as the mediators of the metabolic response to microwaves at this frequency. By contrast, no meaningful relationship exists between delta $\underline{M}$ and

delta T_{co}, which provisionally eliminates deep-body thermoreceptors from consideration as the primary mediators of this response.
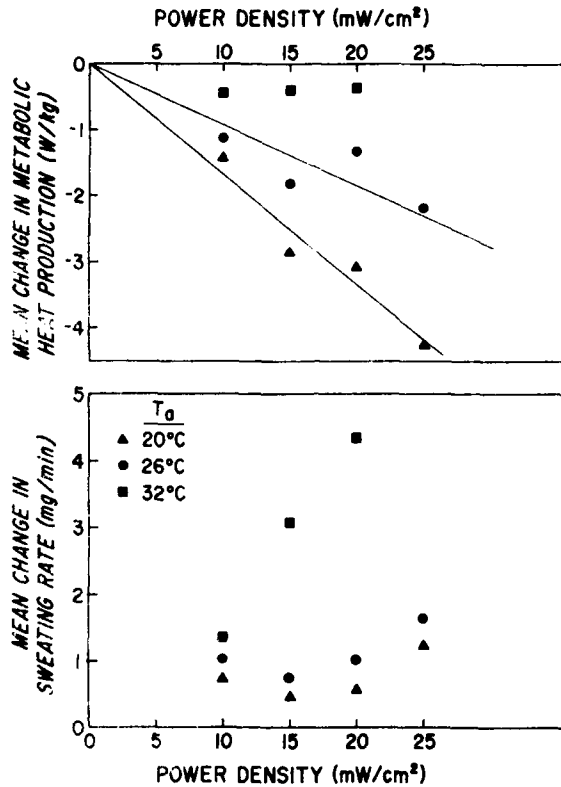

Figure 21. Mean change in equilibrated baseline level in metabolic heat production (top panel) and sweating rate (bottom panel) as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

Figure 24 shows a similar comparison for the change in sweating rate from the foot; the upper panel plots the data as a function of the change in $\overline{T}_{sk}$ and the lower panel plots the data as a function of the change in T_{co}. It is not as simple to decide in this case which relationship is the more meaningful because the sweating response is not normally mobilized in cooler environments (i.e., T_a=20 and 26 °C). Thus, even though $\overline{T}_{sk}$ may rise several degrees Celsius during microwave exposure, sweating may not be initiated. The lower panel shows a clear relationship between delta $\dot{m}_{sw}$ and delta T_{co}, but there are too few data points to determine this relationship with certainty. Thermoregulatory sweating in man and nonhuman primates has been shown to depend on both central and peripheral temperature changes (21,37) and the sweating response of the squirrel monkey in the presence of microwave fields depends both on the rate of energy absorption and the T_a at which the exposure occurs (3). A further complication arises from the fact that some squir-

monkeys appear to be efficient, and others inefficient sweaters (3). More data are required before this question can be resolved.
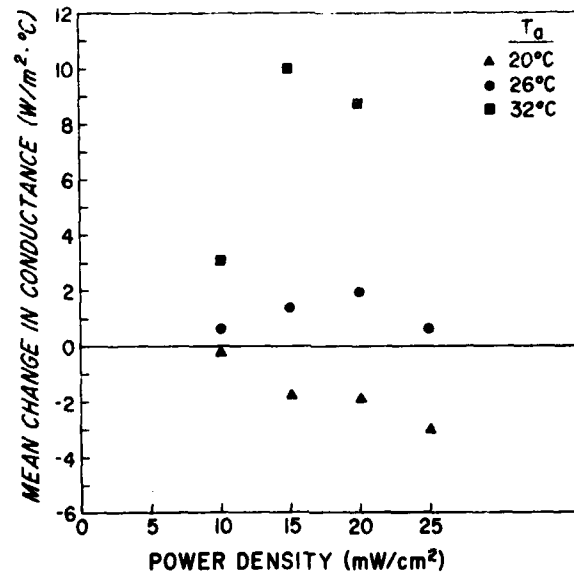

Figure 22. Mean change from equilibrated baseline level in tissue conductance as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

Role of Skin Temperature In The
Thermoregulatory Response To Microwaves

The preceding comparisons have provided strong evidence of the preponderant role of thermal receptors in the skin vs those deep in the body in the mediation of thermoregulatory responses of squirrel monkeys exposed to 2450-MHz CW microwaves. Consideration of this fact permits a second method of determining if absorbed radiofrequency radiation may be somehow unique in its impact upon the thermoregulatory system. The relationship already presented in Figure 19 indicated that the fate of heat generated in the body by absorbed microwaves was no different from any other source of heat. Figures 25, 26, and 27 show sweating rate, colonic temperature, and metabolic heat production (with and without A_{rfr}) as a function of mean skin temperature. In all figures, each plotted point represents the steady-state value from one experimental session; open symbols are data collected during the initial

equilibration period when no microwaves were present and closed symbols, coded in terms of power density, are data collected during microwave exposure.
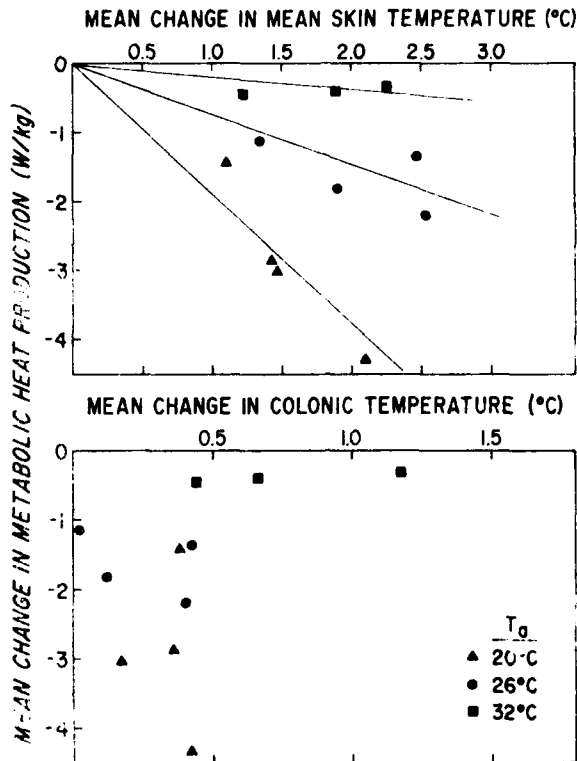

Figure 23. Mean change from equilibrated baseline level in metabolic heat production as a function of the mean change in weighted mean skin temperature during microwave exposure (top panel) and the mean change in colonic temperature (T_{co}) during microwave exposure (bottom panel). The parameter is ambient temperature (T_a).

Figure 25 shows the steady-state sweating rate as a function of $\bar{T}_{sk}$ for all animals at all T_a. The open symbols (no microwaves present) fall naturally into three groups which represent the three T_a under investigation. The solid symbols (microwaves present) fall in orderly fashion between these three groups such that the aggregate describes a continuous functional relationship between the two variables. (The two points off the function were contributed by a single monkey, an extremely efficient sweater at T_a=32 °C). There is no evidence in this figure that the sweating response in the presence of microwaves is any different from the sweating response when the skin is warmed by convection and radiation.

Similar functions for the steady-state levels of T_{co} and M as a function of $\bar{T}_{sk}$ appear in Figure 26. In both panels, the symbols have the same meaning

as in Figure 25. Once again, all points fall in orderly fashion along a single function, with no indication that the response in the presence of microwaves is different from the normal response. An iterative statistical procedure, performed on the $\underline{M}$ data, determined that the best-fitting function was a hyperbola of the form $\overline{y} = a + b/x$. The highest R^2 (0.87) was obtained for the baseline data alone (unfilled symbols), an R^2 nearly as high (0.80) for the baseline and microwave data combined (all symbols), and a somewhat lower R^2 (0.69) for the microwave data alone (filled symbols). An even lower R^2 resulted (0.66) when A_{rfr} was added to the $\underline{M}$ values measured during microwave exposure as shown in Figure 27. Indeed, the $\underline{M}$ vs $\overline{T}_{sk}$ function becomes increasingly distorted at the higher T_a when the absorbed energy is accounted for as part of the total heat to be eliminated from the body. In summary, Figures 26 and 27 provide convincing evidence of the involvement of $\overline{T}_{sk}$ in the thermoregulatory responses of squirrel monkeys to 2450-MHz CW microwaves and of the fact that absorbed microwaves are dealt with in normal fashion by the thermoregulatory system.
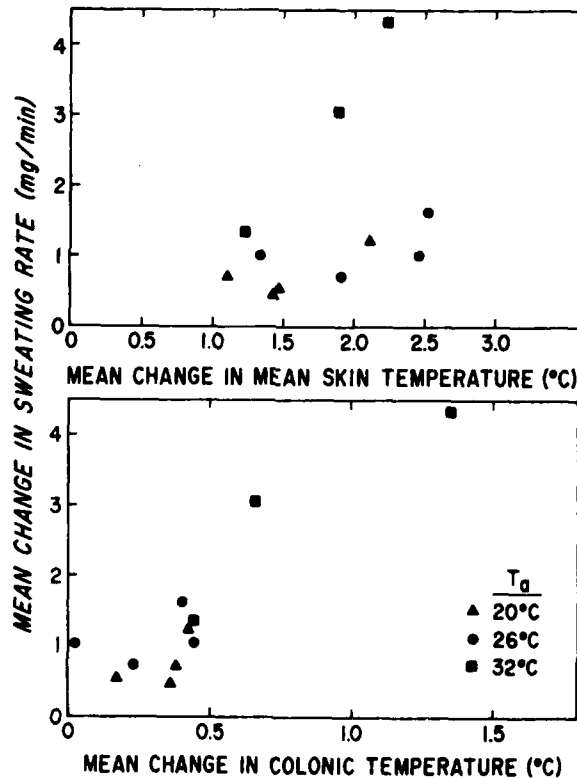
Figure 24. Mean change from equilibrated baseline level in sweating rate as a function of the mean change in weighted mean skin temperature during microwave exposure (top panel) and the mean change in colonic temperature during microwave exposure (bottom panel).
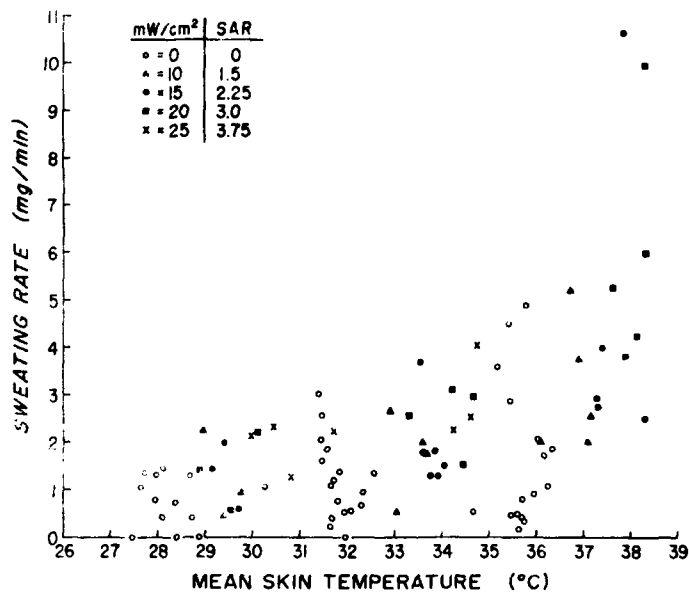
Figure 25. Steady-state sweating rate from the foot as a function of weighted
mean skin temperature. Open symbols (power density and SAR = 0)
represent data from the baseline equilibration period when micro-
waves were absent. Solid symbols, coded by power density and SAR,
represent data from microwave exposure period.
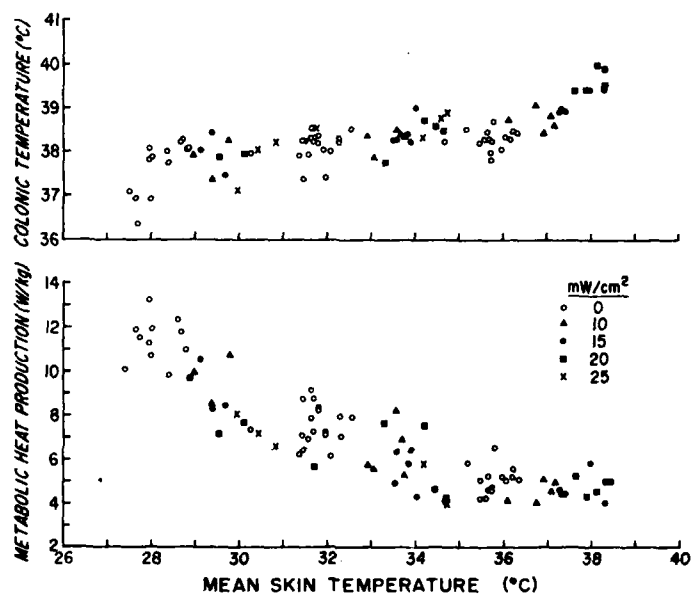
Figure 26. Steady-state colonic temperature (top panel) and metabolic heat
production (bottom panel) as a function of weighted mean skin
temperature. Open symbols (power density and SAR = 0) represent
data from the baseline equilibration period when microwaves were
absent. Solid symbols, coded by power density and SAR, represent
data from microwave exposure period.

Figure 27. Steady-state values of metabolic heat production plus absorbed radiofrequency energy (A_{rfr}) as a function of weighted mean skin temperature. Open symbols (A_{rfr} = 0 W/kg) represent data from the baseline equilibration period when microwaves were absent. Solid symbols, coded by A_{rfr}, represent data from microwave exposure period.
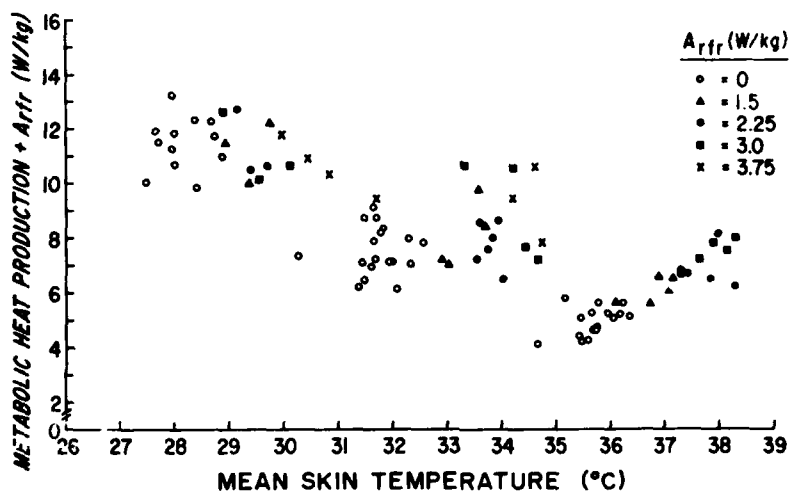
DISCUSSION AND EVALUATION

The method of partitional calorimetry has been used to determine the steady-state heat balance of adult male squirrel monkeys exposed to 2450-MHz CW microwave fields at controlled T_a of 20, 26, and 32 °C. Four microwave power densities (three at T_a=32 °C) were studied at each T_a. The particular T_a explored were chosen, after consideration of the thermoregulatory profile of the experimental animal in question, because they represent certain critical values at which specific effects of microwave exposure would be evident. The data revealed no systematic errors in the calorimetry employed and indicated, without question, that the animal subjects achieved thermal balance by mobilizing normal processes of heat production and heat loss, whether a microwave field was present or not. Skin temperature appeared to play a dominant role over deep body temperature in the control of thermoregulatory effector responses when the microwave field was present; this indicates that the primary neural input to the central thermoregulatory controller was from thermosensors in the skin rather than in central nervous system sites such as the hypothalamus. This finding confirms other results from our laboratory that have been recently reported (10,11). It is important to note, however, that the results of these studies may be frequency-specific and that a greater role might be played by deep body thermosensors if the microwave frequency employed was closer to whole-body resonance.

The major finding of these studies is that the animal subjects, during periods of exposure to a 2450-MHz microwave field, were able to attain thermal equilibrium by an orderly and predictable mobilization of normal thermoregulatory responses. The sole exception, as has been noted previously (3), involves microwave exposure at T_a close to the UCT. The squirrel monkey exhibits limited capacity for evaporative heat loss in warm environments (41) and can rapidly become hyperthermic when the temperature of the skin approaches that of the body core (Figs. 1 and 2). Thus, at T_a=32 °C, absorbed microwave energy that is the equivalent of about 75% of the resting heat production of the monkey cannot be tolerated for periods as long as 90 min. In stark contrast, human beings, with their extraordinary capacity for heat loss through the evaporation of sweat, can sustain increases in metabolic heat production of 6-to-10 times the resting level during prolonged physical exercise (6,37,45), and can adapt with relative ease to excessively warm environments (23). Rates of energy absorption from radiofrequency fields that are the equivalent of man's resting metabolic rate (1.4 W/kg) or less should provide minimal perturbation to the human thermoregulatory system, even in thermally hostile environments.

In the cool (T_a=20 °C) and thermoneutral (T_a=26 °C) environments explored in the present studies, microwave power densities as high as 25 mW/cm² (SAR=3.75 W/kg) were sustained by the mobilization of thermoregulatory responses appropriate to the prevailing T_a. The SAR at this exposure is the equivalent of 75% of the resting metabolic heat production of the squirrel monkey. At T_a=20 °C, the thermoregulatory response to this SAR was roughly the equivalent, in the steady state, to the response that would be observed if these same animals were exposed to a T_a of about 26 °C without microwaves (Figs. 12

43

and 14). It has already been stated (cf. RESULTS 2d section) that the thermo-
regulatory response to the same SAR at a T_a of 32 °C may be the equivalent of
exposure to a T_a of 37-38 °C without microwaves. Additional extrapolations of
this type are risky, however, because insufficient baseline data (i.e., levels
of individual thermoregulatory responses in the absence of microwaves) have
been collected in the particular test environment used for these studies.

The data from which the thermoregulatory profile of the squirrel monkey
was constructed (Figs. 1 and 2) were collected under still air conditions; in
the studies reported here, air moved past the animal's body at 0.36 m/s.
Since the heat transfer coefficient $\underline{h}$ varies directly with air movement, all
measured thermoregulatory responses will assume different values that will be
related to $\underline{h}$. The value of $\underline{h}$ determined from the data in Figure 19 was 12.77
$(W/m^2)/$ °C; the value determined by Stitt and Hardy (41) for still air condi-
tions, across a range of T_a from 10 to 39 °C, was 6.50 (W/m^2) °C, roughly half
our value. Figure 1 shows that $\underline{M}$ assumes the value of about 8 W/kg in animals
exposed to T_a = 20 °C under still air conditions. The comparable value mea-
sured in the present study was 11.75 ± 0.29 W/kg, a level nearly 4 W/kg
higher. Similarly, at T_a =26 °C, Figure 1 shows that $\underline{M}$ has reached a stable,
low level of about 5 W/kg, but in the present study, $\underline{M}$ ranges from 6 to 9 W/kg
at this T_a (Fig. 14). These results confirm observations made by others that
at any given ambient temperature, the metabolic heat production of the squir-
rel monkey will be higher when the animal is in moving air than in still air
conditions (2,9,36,40,41). They also provide an explanation for the finding
that microwave exposure can reduce the $\underline{M}$ of an animal equilibrated to a T_a
that is nominally "thermoneutral".

The influence of air movement on thermoregulatory response measures has
generally been ignored by students of microwave bioeffects, but it is of
considerable importance to the accurate determination of an experimental
animal's thermal state, particularly in cool environments. On the other hand,
relative humidity may play an equally important role in warm environments
because high levels may severely limit evaporative heat loss through sweating.
Thus, the proper assessment of the thermoregulatory consequences of microwave
exposure must involve not only measurement of the experimental animal's
thermoregulatory profile but also careful attention to the physical character-
istics of the particular test environment employed.

One of the most striking consequences of microwave exposure in a cool
environment is the reduction of $\underline{M}$ (cf. Fig. 6). In the steady state, this
reduction (in W/kg) is equal to the SAR (in W/kg) of the imposed microwave
field, a finding reported by Adair and Adams (9) and confirmed in the present
studies. At a frequency that provides significant surface heating, such as
the 2450 MHz used in these experiments, no change in deep-body temperature,
and only small increments in skin temperature, accompany microwave exposure.
The advantages of such a thermal environment have been recognized by Pound
(39), who has proposed that 3-cm microwaves be considered as a source of
comfort heating for human beings in otherwise-cold interior spaces. Pound has
predicted that significant energy savings would result from such an applica-
tion. As evidence mounts for the benign nature of radiofrequency radiation,
the possibilities for advantageous use of these frequencies should be con-
sidered ever more seriously. The results of the present studies establish
that a microwave environment is the thermal equivalent of a radiant or convec-
tive environment in terms of the basic functioning of the autonomic

thermoregulatory system. No unusual physiological responses were detected that would point to the microwave environments studied as being other than benign.

CONCLUSIONS

Partitional calorimetric studies have examined the autonomic thermoregulatory responses of squirrel monkeys exposed to 2450-MHz CW microwaves at selected ambient temperatures, 20, 26, and 32 °C. Heat production and heat loss responses were analyzed in terms of a heat balance equation which incorporated the energy absorbed from the microwave field. The results of these studies allow the following conclusions to be drawn:

1. The animals were able to maintain thermal balance, i.e., regulate their body temperature, under all conditions employed;

2. In a 20 °C environment, thermal balance was maintained by a reduction of metabolic heat production when microwaves were present;

3. In a 26 °C environment, thermal balance was maintained by a further reduction of metabolic heat production and initiation of vasodilation in t e tail and foot when microwaves were present;

4. In a 32 °C environment, thermal balance was maintained by vasodilation of the foot and initiation of thermoregulatory sweating, as long as the microwave power density was 20 mW/cm^2 or below;

5. The animals could not maintain thermal balance in a 32 °C environment if the microwave power density was greater that 20 mW/cm^2;

6. Responses measured with and without microwaves present were described by the same functional relationships, indicating that the thermoregulatory system deals with microwaves in the same manner as other environmental energy sources;

7. Thermal sensors in the skin, rather than those deeper in the body, were probably responsible for most of the response changes observed;

8. Predictions of potential human responses in the presence of comparable microwave fields will not be possible until additional baseline data are collected on the experimental animals and a few sample measurements are made on human volunteers to serve as anchor points.

ACKNOWLEDGMENTS

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