

AUTHORS: Mayers CP, Habeshaw JA:

DATE: 1973

TITLE: Depression of phagocytosis: a non-thermal effect of microwave radiation as a potential hazard to health.

SOURCE: Int J Radiat Biol 24(5):449-61, Nov 1973

MAIN SUBJECT HEADING:

AN
ANALYTICS

HU
HUMAN
EFFECTS

AT
ANIMAL
TOXICITY

IH
WORKPLACE PRACTICES-
ENGINEERING CONTROLS

M
MISCELLANEOUS

SECONDARY SUBJECT HEADINGS:

AN

HU

AT

IH

M

Physical/Chemical Properties

Review

Animal Toxicology

Non-occupational Human
Exposure

Occupational Exposure

Epidemiology

Standards

Manufacturing

Uses

Reactions

Sampling/Analytical Methods

Reported Ambient Levels

Measured Methods

Work Practices

Engineering Controls

Biological Monitoring

Methods of Analysis

Treatment

Transportation/Handling/
Storage/Labelling

MR 33

Depression of phagocytosis: a non-thermal effect of microwave radiation as a potential hazard to health

C. P. MAYERS and J. A. HABESHAW

Department of Pathology, University of Edinburgh, Scotland

(Received 5 September 1972; accepted 26 March 1973)

The widespread use of microwave equipment presents potential hazards to health from inadvertent exposure. The hazards due to the heating effect of microwaves are well recognized. Recent evidence suggests that microwave radiation produces other biological effects that are not in any way due to heating. Experiments were performed to investigate the non-thermal effects of microwaves on the process of phagocytosis. Monolayer cultures of mouse macrophages on cover-slips were perfused with suspensions of human red blood cells while being exposed to microwave radiation of 2450 MHz frequency, at a power level of 50 mW/cm², under strict temperature control. Comparison of the phagocytic indices of the irradiated cultures with unirradiated control cultures demonstrated a marked depression of phagocytic activity. Further investigation showed that macrophage phagocytic activity was restored to normal if the radiation was discontinued. The potential hazards to health of this phenomenon are briefly discussed.

1. Introduction

Microwave equipment is used extensively in radar, communications, medical diathermy, microwave ovens, glue-setting in laminated wood fabrications, plastic setting, rubber curing, grain drying, concrete splitting (Puschner 1966), and even in the eradication of malarial mosquitoes (*New Scientist*, 1972). Increasing numbers of people are being exposed to potential health hazards due to the biological effects of microwaves (Michaelson 1971, Powell and Rose 1970, Swanson, Rose and Powell 1970).

The most obvious biological effect of microwaves is heating. The quantity and distribution of heat production depends on such factors as the wave-length, duration and power of the applied energy, and on the absorption coefficient and internal reflection at interfaces in the exposed material (Schwan and Piersol 1954, Schwan 1971). The human health hazards include cataract formation and testicular damage. Many countries now have 'safety' limits for microwave exposure (Swanson *et al.* 1970). These are based largely on the heating effects of microwaves in animal experiments. The most commonly accepted upper level of safety is 10 mW/cm².

In recent years, evidence has accumulated that microwave energy may produce other and entirely different biological effects, which are not in any way due to heating. For instance, whole mice exposed to microwaves of 10 cm wave-length produced an initial rise in phagocytic activity, as measured by the carbon clearance technique, which was followed by a fall (Plurien, Sentenac-Roumanov, Jolly and Drovot 1966). Similar phasic changes in the phagocytic and bactericidal activity of the blood have been demonstrated in whole mice exposed to other frequencies of microwaves (Smurova 1967).

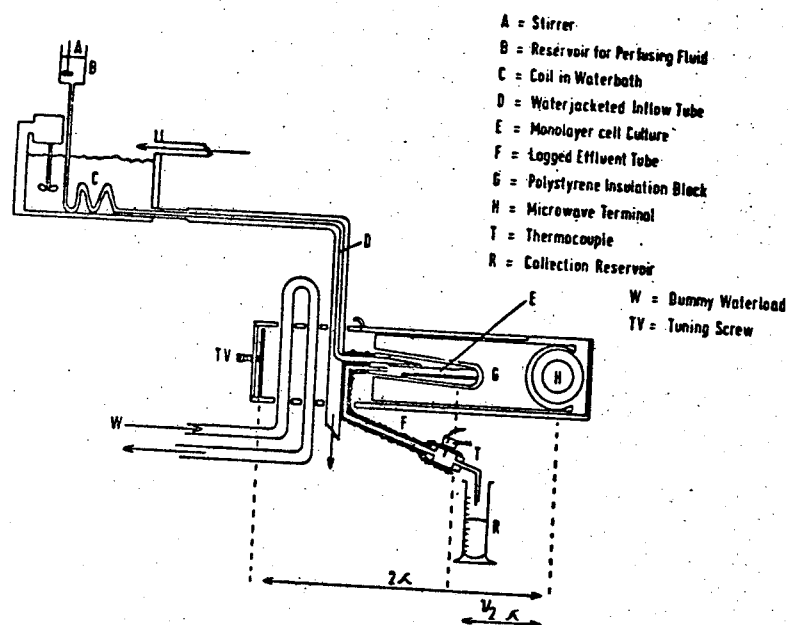


Figure 1. General arrangement of apparatus.

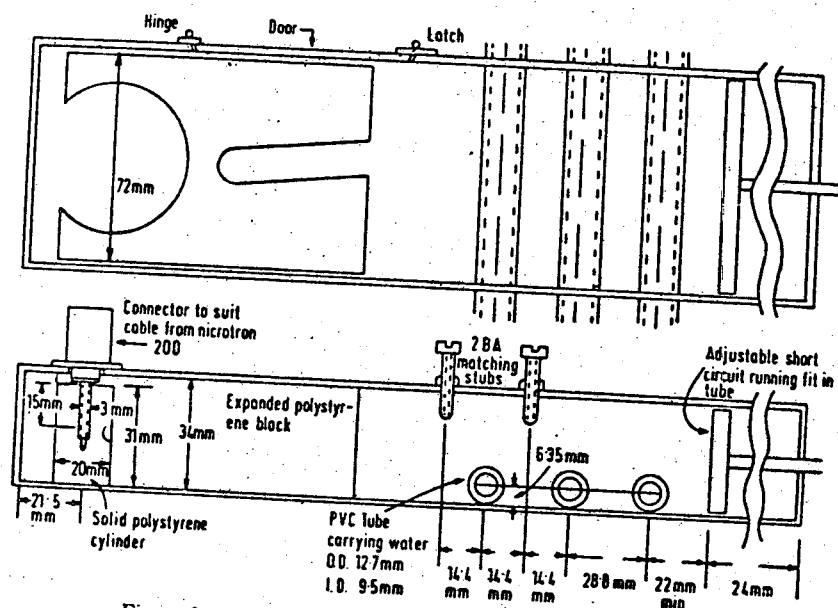


Figure 2. Elevation (above) and plan of microwave chamber.

Nuclear abnormalities have been produced in the lymphocytes of whole animals exposed to microwaves (Baranski 1971) and lymphoblastoid transformation has been demonstrated in lymphocytes irradiated *in vitro* (Stodolnik-Baranska 1967). Microwave radiation has also been shown to induce chromosomal aberrations in the cells of garlic root-tips (Heller and Texeira-Pinto 1959), and has been shown to affect the motility of unicellular organisms and mammalian cells (Furedi and Valentine 1962, Furedi and Ohad 1964, Hubner 1960, Texeira-Pinto, Nejelski, Cutler and Heller 1960) and to induce abnormal development of chick embryos (Van Ummersen 1963) and of insect pupae (Carpenter and Livstone 1971). In Soviet Russia there has been considerable experimental interest in the non-heating effects of microwaves (Subbota 1972), although as Dodge (1969) has pointed out, some of the Russian reports lack details of microwave energy absorbed or of experimental controls.

The experiments here described were designed to investigate the biological effects of microwaves at a cellular level, and in particular to investigate:

- (a) whether or not a non-heating effect of microwaves could be demonstrated, and
- (b) if so, what effect, if any, microwave radiation might have on intercellular reactions.

The process of phagocytosis was selected for study as an example of an intercellular reaction. The experiments involved the exposure of red blood cells to the phagocytic activity of macrophages, with and without microwave radiation. An advantage of this system is the reliability with which phagocytosis can be quantitated (Habeshaw 1970). The experimental set-up consisted of cultures of macrophages perfused with suspensions of erythrocytes in a microwave chamber, with stringent controls to maintain a constant 37°C temperature.

2. Materials and methods

The general arrangement of the apparatus is shown in figure 1. It consisted of a microwave chamber in which a culture of macrophages on a glass coverslip in a small test-tube was maintained at constant temperature and perfused with a continuous flow of erythrocytes suspended in culture medium.

2.1. Microwave resonance chamber and generator

The copper microwave chamber was 26 cm long and 74 × 32 mm in cross-section (figure 2). The chamber carried three paired tubes to allow for the circulation of a dummy load of running water (W) (figure 1). A tight-fitting door allowed access to the chamber, which contained a carved polystyrene-foam block (G), in which the test-tubes containing the macrophage monolayers (E) were placed. The block located the cultures in the same position in each experiment, at approximately $\frac{1}{4}\lambda$ from the microwave input terminal (H).

The microwave generator was a Microtron 200 microwave diathermy unit made by Electro-Medical Supplies (Greenham) Ltd, London. The operating frequency was 2450 ± 25 MHz. The microwave source in this instrument was a Mullard magnetron, type JP2-02.

2.2. Cultures of macrophages

Macrophages were obtained from 12 to 20 g mice of a closed colony strain by the method of Stuart (1967). The mice were killed and after partial dissection, 6 mm of culture medium was injected into the peritoneal cavity, and circulated by gentle prodding. The medium used was Medium 199 (Wellcome Reagents, England) and contained penicillin 200 iu/ml, streptomycin 100 µg/ml and heparin 5 iu/ml. The fluid was aspirated from the peritoneal cavity through a wide-bore needle, and the aspirates from batches of six mice were pooled. 1 ml volumes of the cell suspension at a concentration of 1×10^6 cells per ml were seeded onto flying cover-slips measuring 10×6 cm in 40×10 mm Pyrex culture tubes. The medium was supplemented by the addition of 10 per cent volume of serum-free medium supplement (Habeshaw 1972) containing lactalbumin hydrolysate 4.7 g, oyster glycogen 1 g, sucrose 0.5 g, polyvinyl pyrrolidone 0.75 g, calcium pantothenate 20 mg, L glutamine 10 mg and water to 100 ml. The tubes were stoppered with Fesco white rubber stoppers and incubated at 37°C for 24 hours before use.

2.3. Erythrocyte suspensions

Fresh human red blood cells were obtained from volunteers by venipuncture, anticoagulated with heparin and sedimented by centrifugation for 15 min at 1500 g. The supernatant plasma was removed and the cells were washed four times in a large excess of 0.85 per cent NaCl solution. They were reconstituted as a suspension in medium 199, with 10 per cent serum-free medium supplement, to a concentration of 8×10^6 red cells/ml. The suspension was placed in the reservoir (B) (figure 1), and the red cells were kept in suspension by a gentle mechanical stirrer (A). From the reservoir (B) the perfusion fluid was led through the coil (C) in a 37°C water-bath, and through a water-jacketed inflow tube (D) in the microwave chamber to the culture tube (E). The outflow ran in an insulated effluent tube (F), past a thermocouple probe (T), and was collected in the measuring cylinder (R). The flow-rate of the red cell suspension through the macrophage culture chamber was kept constant at 3 ml/min by adjusting the height of the reservoir (B).

2.4. Temperature Control

To control the temperature of the cells in the culture tubes, the temperature of the inflow to the culture tube was held constant at 37°C, the temperature of the outflow was continuously monitored, the temperature within the culture chamber was measured at the end of each experiment, and calibrations were performed to establish the heating and cooling characteristics of the system.

2.4.1. Inflow temperature

The perfusion inflow was warmed from room temperature (usually 20–22°C, in the reservoir, B in figure 1) to 37°C by passage through the coil (C), in the water-bath. The inflow tube (D) to the culture tube was encased in a water-jacket, through which water from the water-bath was constantly circulated by means of a roller-impeller pump. The temperature of the water-bath was thermostatically controlled at 37°C. During each experiment, the temperature

of the water-bath and the temperature of the return flow from the water-jacket were frequently checked by in-dwelling mercury thermometers. It was found that with the ambient room temperatures experienced, there was very little variation in the water-bath or water-jacket temperatures, and at no time did the temperature drop below 36.5°C or rise above 37.5°C.

2.4.2. Outflow temperature

The temperature of the outflow from the culture chamber was continuously monitored by thermocouple. The outflow passed out of the microwave chamber along a short (6 cm) heavily-lagged tube into a small chamber of 1 ml volume into which the needle-probe of a thermocouple (T) was inserted. The temperature of the outflow was continuously monitored throughout each experiment, by a thermocouple connected to a Pye 'Scalamp' galvanometer. During most experimental runs, the outflow temperature remained constant. During a few runs, the mean outflow temperature was calculated as the mean of thermocouple temperature readings taken through the experimental run.

At the end of each experimental run, the culture tube was removed quickly from the microwave chamber, and its temperature was taken using the same thermocouple probe. This procedure served to check that the outflow temperatures did in fact closely reflect the temperatures obtained in the culture tube.

2.4.3. Calibration of thermocouple

A series of readings was made of water temperatures from 0° to 100°C using mercury thermometers and the thermocouple connected to a galvanometer. A linear relationship between the mercury thermometer and the thermocouple readings was established. The accuracy and reproducibility of the thermocouple and mercury thermometer were investigated by taking repeated readings of melting ice and of boiling distilled water. The most important part of the temperature scale was the 30° to 45°C range and thermocouple readings of the melting points of o Cresol (m.p. 30.9°C), p Cresol (m.p. 34.7°C) and phenol (carbolic acid, m.p. 41.0°C) were used to determine the accuracy and reproducibility of the readings in this range. The drift of the thermocouple readings was investigated by taking repeated readings of melting ice and boiling water over a period of two hours with no correction of the zero setting.

2.4.4. Calibrations of power level, heat generation and cooling rate

The rate of heating in the microwave chamber of unperfused 1 ml volumes of water was investigated under a range of input levels to determine the heat energy absorbed by the specimens, and also the subsequent cooling rate of the unperfused specimens. From these observations, nomograms were constructed to relate the heating rate to the power input, and the rate of cooling to the difference of temperature between the specimen and the surrounding air.

2.5. Experimental method

The thermocouple galvanometer was switched on for 40 min, and the water-bath was turned on to reach 37°C before each experiment commenced. The macrophage cultures in test-tubes were placed in the polystyrene block in the microwave chamber. A bung carrying the inflow and outflow tubes of the

perfusing system was inserted into the end of the culture tube, and perfusion was performed for 30 min. During this time the microwave generator was either switched on at 25 W output level, or was off for a control run. Each 30 min test run in which a culture was irradiated was followed by a control run in which the generator was turned off. During each 30 min run, the temperatures of the water-bath, the water-jacket return flow, and culture tube outflow were continuously monitored, and the perfusion flow-rate checked and adjusted if necessary.

At the end of each run, the temperature of the fluid in the culture tube was measured directly by thermocouple. The macrophage monolayer on a cover-slip was removed from the culture tube, rinsed in physiological saline, fixed in a mixture of equal parts of methanol and acetone and stained by the Giemsa method. The phagocytic index was calculated as:

$$\frac{\text{Number of macrophages containing one or more ingested red cells}}{\text{Total number of macrophages counted}} \times 100.$$

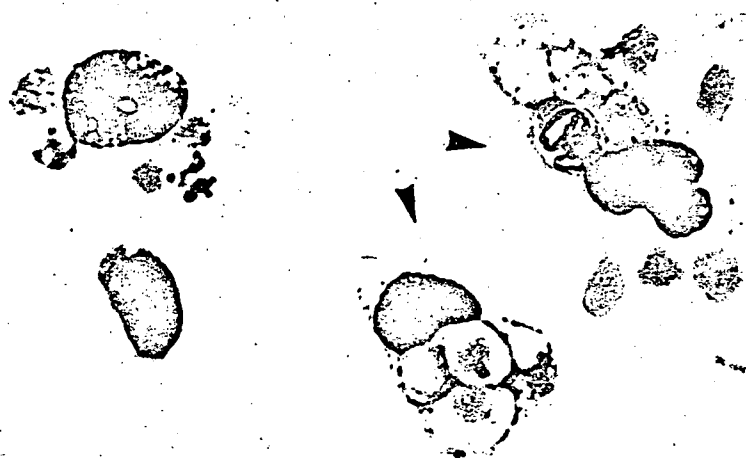


Figure 3. Mouse macrophages after exposure to suspensions of human red blood cells ($\times 800$, Giemsa). The cells arrowed show ingestion of red blood cells.

Figure 3 shows the microscopic appearance of mouse macrophages after exposure to human red blood cells. Some of the macrophages show ingestion of red cells. The percentage of macrophages showing erythrophagocytosis gives the phagocytic index.

2.6. Controls

The following six groups of control experiments were performed:

Group 1: Twenty control experiments in which monolayer cultures of macrophages were incubated at 37°C for 30 min with a suspension of red cells. No perfusion was performed on these cultures. The phagocytic indices from these controls provided the basic data with which the data from the experimental series were compared.

Group 2: Twenty-six control experiments in which macrophage cultures were perfused with red cell suspensions for 30 min, and in which neither the red cells nor the macrophages were irradiated. The phagocytic indices of these cultures were compared with those of the non-perfused controls (Group 1) to determine whether or not the physical movement produced by perfusion affected the phagocytic index.

Group 3: Five macrophage cultures were perfused with tissue culture medium devoid of red cells and exposed to microwaves for 30 min. These cultures were examined for morphological changes and compared with unirradiated cultures to determine whether or not any morphological change was produced in macrophages by irradiation.

Group 4: Five macrophage cultures were perfused with tissue-culture medium devoid of red cells and exposed to microwaves for 30-minute periods. These cultures were subsequently exposed to perfusion by red cell suspensions for 30 min in the absence of microwaves. The phagocytic indices from these cultures were compared with those of the unirradiated perfused cultures to determine whether or not macrophage phagocytic activity was restored to normal after cessation of microwave exposure.

Group 5: Five suspensions of red cells were each allowed to flow for one hour through a continuously-irradiated chamber containing no macrophages. These suspensions were subsequently perfused for 30 min, through unirradiated macrophage cultures. The phagocytic indices of these cultures were compared with those of the non-irradiated perfused cultures of Group 2 to investigate whether previously irradiated red cells were phagocytosed as readily as unirradiated red cells.

Group 6: Twenty-four macrophage cultures were incubated with suspensions of red cells for two hours at different temperatures to determine the effect of temperature on phagocytic activity in this experimental system.

2.7. Precautions

(a) Two monolayer cultures of macrophages from each batch were tested before use by incubation for 24 hours with red cells coated with a standard preparation of rabbit haemolysin. Healthy cultures showed a phagocytic index of over 90 per cent and were morphologically normal. A few cultures which showed less phagocytic activity, or showed any morphological abnormalities were discarded. The cultures that had to be discarded were derived from animals which subsequently showed evidence of infection.

(b) All the glassware and other apparatus used for the preparation of the macrophage cultures and red cell suspensions was bacteriologically sterile. The tubing used for each of the 30-min, or 60-min experiments was not bacteriologically sterile, but was repeatedly washed through with sterile normal saline solution between experiments. The subsequent microscopic examination of the cultures showed no evidence of bacteriological or other infection.

(c) No specific precautions were taken to eliminate mechanical injury to the red cells. A slight increase of phagocytic activity was noted towards the end of

each batch, probably due to mechanical injury to the cells during their passage through the perfusing system, but as irradiated cultures and unirradiated control cultures were equally affected, no attempt was made to prevent it.

3. Results

3.1. Accuracy of temperature readings

The temperature of the three melting-points, as recorded by the thermocouple and mercury thermometer, are shown in table 1, together with the standard deviations. The table indicates that 95 per cent of thermocouple readings are accurate to $\pm 0.4^\circ\text{C}$, and that 95 per cent of the mercury thermometer readings are accurate to within $\pm 0.6^\circ\text{C}$. (The effect of barometric pressure on the melting points was calculated and found to be negligible at 450 ft above sea-level and thermocouple drift was also found to be negligible, after the initial warm up period of 40 min).

	o Cresol	p Cresol	Phenol
Theoretical melting point $^\circ\text{C}$	30.9	34.7	41.0
Thermocouple, mean of 10 readings in $^\circ\text{C}$ with standard deviation	30.62 ± 0.34	34.85 ± 0.36	40.6 ± 0.415
Mercury thermometer, mean of 10 readings in $^\circ\text{C}$, with standard deviation	30.56 ± 0.48	34.12 ± 0.61	40.26 ± 0.5

Table 1. The temperatures recorded by thermocouple and mercury thermometer at the melting points of o Cresol, p Cresol and Phenol (Carbolic acid).

3.2. Heating rate, cooling rate and absorbed power levels

The rate of heat loss of a 1 ml unperfused specimen at 37°C (with room temperature 21°C) was 3.6°C per min. The heating rate experiments demonstrated that with a microwave power input reading of 50 W, it was just possible to achieve and maintain the unperfused specimen at 37°C . The energy absorbed as heat by the specimen was $3.6 \text{ cal/min} = 15.0 \text{ J/min}$. This incidentally indicates that only 0.5 per cent of the input energy was absorbed by the specimen.

3.3. The effect of microwave irradiation on the morphology of macrophages

The macrophage monolayers exposed to microwaves for 30 min showed no morphological changes on comparison by light microscopy with unirradiated cultures.

3.4. The effects of microwaves upon erythrophagocytosis

Microwave irradiation produced a significant depression of macrophage activity as indicated by the phagocytic index. The phagocytic indices are shown in table 2, together with the significance of the results as compared with the unperfused unirradiated controls (Group 1).

The mean value of the phagocytic indices of the 26 perfused irradiated cultures was $6.3 (\pm 2.5)$ per cent and for the perfused cultures not exposed to microwaves $20.2 (\pm 5.8)$ per cent. The irradiated cultures differ significantly from the controls ($P < 0.025$), but the unirradiated perfused cultures do not ($P < 0.3$).

Type of culture	Erythrophagocytic index	Significance P
Control (Group 1)	31.5 ± 3.4 per cent	—
Perfused, unirradiated (Group 2)	20.2 ± 5.8 per cent	$P < 0.30$
Perfused irradiated	6.3 ± 2.5 per cent	$P < 0.025$
Irradiated, subsequently exposed to red cell perfusion (Group 4)	23.2 ± 1.0 per cent	$P < 0.50$

Table 2. The effects of microwave radiation on erythrophagocytosis.

3.5. The effect of previous irradiation of macrophages on phagocytosis

Macrophage cultures exposed to microwaves, and subsequently interacted with red cells gave a mean phagocytic index of $23.2 (\pm 1.04)$ per cent, a result that did not differ significantly from the control values ($P < 0.5$).

3.6. The effect of microwave radiation upon red cells

Red cell suspensions exposed to microwave radiation while being perfused through a blank chamber showed no greater susceptibility to phagocytosis than fresh red cells. The phagocytic index with fresh red cells was $23.3 (\pm 2.4)$ per cent and with irradiated red cells $25.5 (\pm 3.3)$ per cent. There is no significant difference between these results. Macrophages readily ingest injured human red cells, and the increased phagocytic activity can be used to detect injury to red cells (Stuart 1967). In this case, as there is no significant increase in phagocytosis, we can assume that no significant degree of injury is present in the red cells as a result of microwave radiation exposure.

3.7. Temperatures recorded during the experiments

At the end of each experiment, the temperature within the culture chamber was measured. It was always found to be within 0.8°C of the outflow temperature. The constantly monitored outflow temperatures thus indicate that the chamber temperatures recorded did not conceal a hidden rise of chamber temperature. The chamber temperatures achieved are shown in table 3.

The temperatures recorded were lower than 37°C , even when the cultures were being irradiated. The principal heat loss occurred in the short length of perfusion tubing between the water-jacket and the culture chamber, and this could not be overcome. It is noteworthy that the temperature of the perfused control cultures were always less than those of the perfused irradiated cultures, but that their phagocytic indices were higher.

Type of culture	Chamber temperature	Phagocytic index
Control (Group 1)	$37.0 \pm 0.5^\circ\text{C}$	31.5 ± 3.4 per cent
Perfused unirradiated (Group 2)	$31.8 \pm 0.8^\circ\text{C}$	20.2 ± 5.8 per cent
Irradiated, perfused	$34.3 \pm 0.7^\circ\text{C}$	6.3 ± 2.5 per cent

Table 3. Microwave chamber temperatures and phagocytic indices.

3.8. The effect of temperature on phagocytic activity

The phagocytic indices obtained at different incubation temperatures (Control Group 6) are shown in the graph (figure 4). Phagocytic activity increases with temperature up to 38.5°C , after which it falls away rapidly.

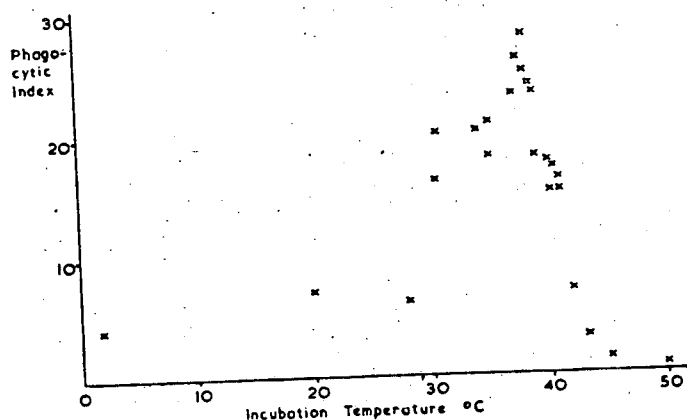


Figure 4. The effect of temperature on phagocytosis. The phagocytic indices were obtained by incubating macrophage cultures with red cell suspensions at different constant temperatures, with no exposure to microwave radiation.

4. Discussion

The health hazards due to the heating effects of microwaves are readily appreciated. The non-thermal effects of microwaves, and their possible ill-effects, are less well established. We believe that our experiments show that a non-thermal effect does occur in biological material.

In these experiments, the interaction between macrophages and red blood cells occurred in a small test-tube exposed to microwaves. The phagocytic index of the irradiated cultures was 6.3 ± 2.5 per cent, compared with the index of 20.2 ± 5.8 per cent for the control unirradiated cultures. This difference was statistically significant ($P < 0.025$). There can be no doubt that microwave radiation depressed phagocytosis. The question then arises whether this was

a thermal or a non-thermal effect, and it is for this reason that we have described in some detail the temperature control and monitoring methods used and the results obtained.

The principal monitoring device was the thermocouple probe placed in the test tube from the microwave chamber, the readings from which were continuously observed during each experiment. It has been shown that these temperature readings were an accurate reflection of the actual temperature within the microwave chamber, and it has also been shown that 95 per cent of the thermocouple readings were accurate to within 0.4°C .

The temperatures recorded for the irradiated cultures were $34.3^{\circ} \pm 0.7^{\circ}\text{C}$, for the unirradiated control cultures $31.9^{\circ} \pm 0.8^{\circ}\text{C}$. It is apparent that microwave irradiation has produced an average of 2.5°C temperature increase in the irradiated cultures. If we allow for inaccuracy of the thermocouple (0.4°C), and discrepancy between the outflow temperature and that actually existing within the chamber at the end of each experiment (maximum 0.8°C), the variation in the temperature of the cultures (0.7°C), then the maximum temperature that could have existed within the culture chamber at any time during the experiment was 36.2°C . We have shown that the rate of phagocytosis in our test system increases with temperature up to 38.5°C (figure 4). Other workers (Harmon, Zarafonitis and Clark 1946) have previously shown similar changes in phagocytic activity with temperature, using a test system of human pig leucocytes versus *Staphylococci*. We conclude that the depression of phagocytosis shown by the irradiated cultures cannot be accounted for on a thermal basis. Indeed the 2.5°C higher temperature of the irradiated cultures could have been expected to enhance phagocytosis rather than depress it, if in fact, the microwaves had no effect on phagocytosis other than heating.

Another possibility which might be considered is that microscopic foci of heat production may have occurred within the cells, or on the cell surfaces, as postulated by Tomburg (1961). If such microscopic foci of heating do occur, their effects appear to be very limited, as:

- (a) Microwave radiation produced no permanent impairment of macrophage function or morphological appearance, and no injury to the red cells.
- (b) Any appreciable quantity of heat produced at a theoretical localized site would have been rapidly dispersed through the medium by conduction, but in our experiments, the medium showed only a 2.5°C temperature increase.
- (c) As the heating effects of microwaves depend on the presence of dipolar molecules, and as water molecules are the most numerous of the dipolar molecules, and as they are ubiquitous and almost uniform in their distribution throughout the cells and the media, it is difficult to conceive of any significant degree of microscopically localized heat production.

As mass-heating has been excluded and as any significant degree of microscopic heating appears improbable, we conclude that the effects demonstrated are non-thermal, but we are not in a position to describe the precise manner of their production.

It may, however, be of interest to speculate to what extent the results obtained in these experiments indicate a potential health hazard. A transient exposure

to microwaves would probably have little effect other than heating and any non-thermal effect would be confined to the superficial tissues as the effective depth of penetration by microwaves of this wavelength is no more than a few centimetres (Michaelson 1971, Schwan and Piersol 1954). However, longer periods of exposure might be expected to affect the mechanism of cell-mediated immunity in superficial tissues, with consequent increased susceptibility to infections, and suppression of the immunological surveillance mechanisms which might allow aberrant cell forms to develop.

It is possible that other cellular interactions might similarly be affected, and that for instance, the phagocytic activity of neutrophil leucocytes, might be depressed. In the case of long term exposure, this might be expected to produce a condition similar to agranulocytosis of the superficial tissues, and infections such as boils might be anticipated. We have shown that a non-thermal effect can be demonstrated at the cellular level. We have noted the energy absorbed, but we have not been able to describe the intensity of the field to which the specimens were exposed. It is suggested that the quantification of the field intensity should be urgently investigated in view of the potential health hazard due to the non-heating effects of microwaves. It is probable that the duration of exposure will have to be taken into account as well as the field intensity.

5. Conclusions

1. Microwave radiation at a frequency of 2450 ± 25 mHz and an absorbed power level of 50 mW/cm^2 depresses phagocytosis.
2. This effect is non-thermal.
3. The depression of phagocytic activity is transient, and ceases when the irradiation is stopped.
4. At this power level and frequency, microwave radiation does not produce red cell injury, nor does it produce any detectable morphological change in macrophages.
5. The precise nature of this non-thermal effect of microwave is not known.
6. The present safety limits of microwave exposure which relate to absorbed power levels may need to be qualified as to permitted duration of exposure.

ACKNOWLEDGMENT

We acknowledge receipt of a grant from the Scottish Home and Health Department for the purchase of the microwave apparatus.

L'usage répandu de matériel à micro-ondes présente des risques pour la santé à cause de la possibilité d'exposition accidentelle. Les dangers que présente l'effet thermique des micro-ondes sont bien connus. Toutefois, des observations récentes donnent à penser que l'irradiation par micro-ondes produit des effets biologiques qui ne sont nullement causés par l'effet thermique. On a effectué des expériences pour étudier les effets non-thermiques des micro-ondes sur le processus de la phagocytose. Des cultures de macrophages de souris, en couche simple sur lamelles couvre-objets, ont été perfusées d'érythrocytes de sang humain en suspension tout en étant exposées à une irradiation par micro-ondes à 2450 MHz de fréquence et à une puissance de 50 mW/cm^2 , sous strict contrôle de température.

La comparaison entre les indices phagocytaires des cultures irradiées et les cultures de contrôle non-irradiées a montré une nette baisse d'activité phagocytaire. Des expériences complémentaires ont montré que l'activité phagocytaire des macrophages redevenait normale après discontinuation de l'irradiation. Les dangers que ce phénomène peut présenter pour la santé sont brièvement examinés.

Bei der weitverbreiteten Anwendung von Mikrowellenausrichtungen besteht die Möglichkeit von Gesundheitsschäden aufgrund unvorhergesehener Bestrahlung. Die Schäden, die vom Hitzeeffekt der Mikrowellen herrühren, sind weithin erkannt. Jüngste Ergebnisse lassen darauf schliessen daß Mikrowellenbestrahlung andere Wirkungen hervorruft, die keineswegs auf Hitze zurückzuführen sind. Es wurden Versuche durchgeführt, um die nicht-thermischen Wirkungen von Mikrowellen auf den Prozeß der Phagozytose zu untersuchen. Einschichtige Kulturen von Mausemakrophagen auf Deckgläsern wurden mit Aufschwemmungen von menschlichen Erythrozyten durchtränkt, während sie einer Mikrowellenbestrahlung mit einer Frequenz von 2450 MHz und bei einer Stromstärke von 50 mW/cm² unter strikter Temperaturkontrolle ausgesetzt wurden. Ein Vergleich der Phagozytenindizes der bestrahlten Kulturen mit nicht bestrahlten Kontrollkulturen zeigte eine bemerkenswerte Abnahme des phagozytären Aktivität. Weitere Untersuchungen ergaben, daß bei Unterbrechung der Bestrahlung die normale phagozytäre Aktivität wiederhergestellt wurde. Die potentiellen Gesundheitsschäden dieser Erscheinung werden kurz behandelt.

REFERENCES

- BARANSKI, S., 1971, *Aerospace Med.*, **42**, 1196.
 CARPENTER, R. L., and LIVSTONE, E. M., 1971, *I.E.E.E. Trans.*, MTT **19**, 173-178.
 DODGE, C., 1969, *Symposium on the Biological Effects and Health Implications of Microwave Irradiation*, Richmond, Virginia (quoted by Michaelson).
 FUREDI, A. A., and VALENTINE, R. C., 1962, *Biochim. Biophys. Acta*, **52**, 33.
 FUREDI, A. A., and OHAD, I., 1964, *Biochim. Biophys. Acta*, **79**, 1.
 HABESHAW, J. A., 1970, Ph.D. Thesis, University of Edinburgh; 1972, *J. Path.*, **108**, 95.
 HARMON, D. R., ZARAFONETIS, C., and CLARK, P. F., 1946, *J. Bact.*, **52**, 337.
 HELLER, J. H., and TENEIRA-PINTO, A. A., 1959, *Nature, Lond.*, **183**, 905.
 HUBNER, R., 1960, *Electr. Rundschau*, **6**, 229.
 MICHAELSON, S. M., 1971, *Am. ind. Hyg. Ass. J.*, **32**, 338.
 MILROY, W. C., and MICHAELSON, S. M., 1972, *Aerospace Med.*, **43**, 67.
 NEW SCIENT., 1972, Editorial, 56, 704.
 PLURIEN, G., SENTENAC-ROUMANOV, H., JOLLY, R., and DROVET, J., 1966, *C. r. Séance Soc. Biol.*, **160**, 597.
 POWELL, C. H., and ROSE, V. E., 1970, *Am. ind. Hyg. Ass. J.*, **31**, 358.
 PUSCHNER, H., 1966, *Heating with Microwaves* (London: Phillips Technical Library).
 SCHWAN, H. P., 1971, *I.E.E.E. Trans. microv. Theory Tech.*, **19**, 146-152.
 SCHWAN, H. P., and PIERSOL, G. M., 1954, *Am. J. phys. Med.*, **33**, 371.
 SMUROVA, E. I., 1967, *Gig. Sanit.*, **32**, 37.
 STODOLINIK-BARANSKA, W., 1967, *Nature, Lond.*, **214**, 102.
 STUART, A. E., 1967, *The Handbook of Experimental Immunology*, edited by D. M. Weir (Oxford: Blackwell), p. 1037.
 STUART, A. E., and CUMMING, R. A., 1967, *Vox Sang.*, **13**, 270.
 SUBBOTA, A. G., 1972, *Influence of Microwave Radiation on the Organism of Man and Animals*, edited by I. R. Petrov (Washington: National Aeronautics and Space Administration Technical Translation NASA TT F-708), p. 66.
 SWANSON, J. R., ROSE, V. E., and POWELL, C. H., 1970, *Am. ind. Hyg. Ass. J.*, **31**, 623.
 TENEIRA-PINTO, A. A., NEJELSKI, L. L., CUTLER, J. L., and HELLER, J. H., 1960, *Expl. Cell Res.*, **20**, 548.
 TOMBURG, V. T., 1961, *The Biological Effects of Microwave Radiation*, edited by Mary F. Peyton (New York: Plenum Press), p. 221.
 VAN UNNEMSEN, C. A., 1963, Ph.D. Dissertation, Tufts University, Medford, Massachusetts. Quoted by R. L. Carpenter and E. M. Livstone.