THE EFFECT OF MICROWAVE RADIATION ON THE EMBRYONIC DEVELOPMENT AND POST EMBRYONIC GROWTH IN CHICKENS AND TURKEYS

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ABSTRACT

A Preliminary and five other low density experiments were conducted in which chick embryos were subjected to a treatment level of 0.4 mw/cm.^{2} and other chick embryos to a trace level of 0.005 mw/cm.^{2}. A sixth experiment exposed chicks of a meat type strain and an egg type strain to 407 picowatts/cm.^{2} or 0.022 picowatts/cm.^{2} during the growing stage.

Three high density experiments were conducted in which chick embryos were subjected to densities of 0.051 w/cm.^{2} to 1.02 w/cm.^{2} at various times of exposure during the first two days of embryonic development.

In the experiments involving treating during the embryonic stage, hatch weight, feed efficiency, weekly body weight and mortality records were kept. Eggs which failed to hatch were broken out and infertility or time of death determined. In the experiments involving treatment during growing period the birds were weighted at 1 day of age, 4 weeks, 8 weeks, 12 weeks, 16 weeks, 20 weeks and mortality feed conversion data was kept.

In the low density experiments the data indicated that exposure to microwaves at the above low levels had no significant effect on embryonic development or post embryonic growth to 2 weeks of age in chickens and turkeys.

The same was true with regard to the 2 breeds of chickens exposed during the entire growing period to 20 weeks of age.

In the high density experiments there was a definite detrimental effect on hatchability and possibly on hatch weight with irradiation with 0.246 - 1.02 w/cm.^{2}. The magnitude of this effect being affected by (1) the day of incubation on which irradiation took place; (2) the density of irradiation given; (3) the time of exposure. There was evidence that this effect was not entirely thermal.

INTRODUCTION

Since the development of radar during World War II the use of microwaves for industrial, medical, and domestic purposes has increased greatly. This was accompanied by more refined and improved technology as well as increased power in some of the microwave equipment. For example, radar sets increased from 10 W to over 1 million W by 1960 (De Minco 1961). This increased power and utilization of microwaves has inevitably lead to an increased exposure of biological material, human, animal and vegetable to radiations. Numerous research projects attempted to study the effect of microwaves on biological material as indicated in the review of literature. In order to determine the effect of microwaves on poultry, located within a microwave field of the type found in the proximity of a microwave tower, the Departments of Animal Science and Engineering, University of Manitoba, undertook a joint research project (Kondra et al. 1969).

The Kondra project was in part repeated using not only the egg laying type of bird as had been done previously, but also a meat type strain. Simultaneously, a project was undertaken to evaluate the effect of microwave exposure at different stages of embryonic development and on post embryonic growth of chickens and turkeys. Also a portion of this investigation was an attempt to determine the effect of microwave radiations, at higher frequencies and densities than those prevailing in the vicinity of a microwave tower on embryonic development and post embryonic growth.

selves. If a stream of electrons is passed across the mouths of the 'C' shaped cavities, an oscillating current is set up by means of electromagnetic induction. The current involved will be small because of the smallness of the cavities and the current electrons. By connecting several cavities in parallel it is possible to build up current and power of these resonant oscillations.

The principle is analogous to blowing across the top of a bottle, the air travelling across the top of the bottle creates oscillations of air in the bottle. The frequency of this oscillation is governed by the size and shape of the cavity of the bottle. If one uses multiples of the same sized bottle one achieves the same frequency but the volume or power is increased.

The flow of electrons from the cathode to the anode is produced by applying a circular magnetic field between them. The electrons leave the cathode and then describe a circular course under the influence of this field and thereby pass across the mouths of the cavities in the anode before actually reaching it. This produces the frequency of oscillation and the power required.

The microwave output is usually conducted along a wave-guide which terminates in a microwave anntenna (e.g. horn) when it is desirable to emit the incident energy into the surrounding space. Microwaves do not require a medium of transmission as do ultra-sound waves.

In television broadcasting it is usually desired to distribute the radiation over a very large area so that a non-focusing antenna is required. However, in communications relay and radar it is necessary to use a reflecting or parabolic antenna to focus the waves into a narrow beam for more effective transmission. This ability to be focused, much like visible light into intense beams, is one of the properties which makes microwaves a hazard (Kalant 1959). The beam rays are not absolutely parallel so that the power density decreases as the square of the distance from the source point in the manner of visible light. When the radiation reaches an object it may be reflected, diffracted, absorbed or refracted through the object. The percentage of the incident energy which will pass through, be absorbed or reflected, depends on many parameters including the microwave wavelength, the texture and orientation of the target surface, the composition of the target and its thickness (Kalant 1959). Kalant also states that the problem in biological materials is complicated by the inhomogeneity of the media, e.g. the presence or absence of fur, hair or feathers, the water content and thickness and orientation of subcutaneus fat. Biqu Del Blanco (1969) when developing his "theory of electromagnetic interaction with living tissues in the microwave range" felt that the following parameters must be considered:

a. The geometry of the object under study, upon which radiation is incident.

b. Wavelength (centimeters) and frequency (cycles or megacycles/second of radiation).

c. Amplitude of oscillations associated with the electromagnetic fields.

d. Degree and type of polarization.

e. Spatial orientation of the field vectors.

f. Angle of incidence between the wave propagation vector and the incidence surface of the object under study.

g. Power density of incident wave (usually mw/cm.^{2}).

h. Irradiation mode, i.e. whether continuous (c.w.) or discontinuous (pulse). ("The second requires knowledge of the duty cycle - i.e. the time between the commencement of one burst of power and the next burst of power").

Thus he feels the amount of energy absorbed is not only dependent upon the "characteristics of the incident radiation but also on the geometry, physical dimensions and the electrical properties of the target". Thus Bigu Del Blanco (1969) suggests that changes in biological systems depend upon how the microwaves interact with the chemical as well as the physical structure of the target media. He feels that the reaction is most dependent upon the reaction of the chemical structure as "two media of similar electromagnetic characteristics may differ in chemical structure and therefore radiation might affect them both differently."

The energy which is absorbed by any object or body must, by the law of conservation of energy, be converted into some other form of energy. The primary resultant energy of microwave radiation is heat energy which causes a local increase in the temperature of the tissues (Kalant 1959). The effect of temperature elevation of biological tissues will be discussed later.

Bigu Del Blanco (1969) and Murray (1963) both suggest that where atoms, molecules, ions and radicals are placed under the influence of electromagnetic fields (microwaves) the charges are redistributed according to the intensity and orientation of the field vectors. This process being the condition of a "dipole movement indicating an unequal charge distribution about one or more bonds in the molecule which is due to the unequal sharing of the bond electrons by the atoms involved in the bond". More simply this induced dipole is produced by distortion of the electron cloud by the applied fields (Ander and Sonnessa 1965).

In biological systems when molecules are subjected to an alternating electromagnetic (microwave) field, the different elements constituting the molecular system interact, thereby converting electrical energy into heat energy. The heat energy thus produced causes a rise in cellular temperature which may reach a point at which living cell components are damaged. Of the various biological effects of microwaves

heat production or thermal phenomenon has been the most extensively studied.

Salisbury et al (1949) treated the eyes of anaesthetized rabbits with ten minute exposures at a field strength of 3 $W/cm.^2$ and 12 centimeters wavelength. Three to ten days after exposure cataracts appeared in the exposed eyes. Herrich and Krusen (1953), using a cavity magnetron tube producing continuous waves with a frequency of 2400-2500 megacycles/second at 125 watts power and a wavelength of 12.2 cm. were able to produce two types of cataracts in the eyes of rabbits. One type was seen only when grossly visible damage to the other occular structures were produced in corneal clouding, congestion and haemorrhages. The other type was similar to those produced by Salisbury (1949) and was confined to the posterior cortex but could, with repeated exposure, be made to involve the whole eye.

Because of the lack of basic information on radiation dosage employed in these studies, Williams et al (1955) designed an experiment to establish the time and power requirements for lenticular opacities by single doses of 12.3 cm. wavelength radiation with a power output of 100 watts continuous at 2450 megacycles/second. The threshold required to produce lenticular opacities ranged from 5 minutes to 0.59 $watts/cm.^2$ to 90 minutes at 0.29 $watts/cm.^2$ Vitreous temperatures within the eye were measured with some difficulty and ranged between 49° C and 53° C, representing a 10° C to

14° C elevation above normal and agrees with the range of 51° C to 55° C found by Richardson et al (1948). The opacities did not appear until 1 to 14 days after exposure. Threshold exposures less than 25 minutes produced less than 2° C rise in body temperature, but 50 to 70 minute threshold exposures, at considerably lower power densities, caused a 3° C to 5° C increase in body temperature. It appeared to the above authors that the rise in body temperature is a direct function of the duration of threshold irradiation. A study was also undertaken at this time on an already irretrievably lost human eye. The production of sensible discomfort before injury at 0.24 watts/cm.^{2} indicated that injury from 12 cm. waves, before the warning of the few heat receptors in the lens can be activated, is unlikely. This is contrary to the suggestion of Salisbury et al (1949).

The eye is not the only area of the body with limited thermo-regulatory capacity. Ely et al (1957) investigated the testes as a factor in microwave hazard. The generator used in this case had a frequency of 2880 megacycles/second and a 10.4 cm. wavelength. The normal testicular temperature of a dog in the above experiment ranged from 30.3° C to 36.25° C. In this report the 10 cm. waves were used to increase testicular temperatures to from 36° C to 44° C and maintain it there for sixty minutes in all but one case. Below 39° C the tubule lining had a finistrated appearance and the cells appeared to be grouped in columns. The more centrally located tubules

were indistinguishable from the controls. Others were characterized by more marked disassociation of the tubule lining cells, associated with focal pykinosis, and giant cells in the lumen. The more central cells either appeared unaltered or gave the first appearance. No clear cut relationship between temperature or exposure time was encountered with this latter type of lesion. Still another effect was encountered at temperatures between 41^{o} - $44^{o}C$ treated for 60 minutes. It involved necrosis desquamation of cells, haemorrhage, lesser pykinosis and cellular disintegration. The above authors felt that testicular reaction to microwaves was similar to other forms of heat injury. Even at a temperature of $44^{o}C$ for one hour "the damage did not appear irrepairable and permanent sterility did not appear likely".

The effect of microwaves on hematopoetic tissue has been studied extensively in North America and the U.S.S.R. Deichman et al (1963) carried out four experiments on rats, the first three at a power density of 20 $mw/cm.^{2}$ and using exposure times ranging from 7 minutes to 7.5 hours. He observed that the longer exposure resulted in a significant decrease in leucocyte concentration, a significant increase in erythrocyte count and a slight increase in hemoglobin. The short exposure time within the above range was not a factor. The same workers quote Schwann (1958 by personal communication) as recommending 10 $mw/cm.^{2}$ as the tolerance level for humans.

His last experiment at this density of treatment for 3 hours every second day for a total of eleven days showed that each exposure induced significant levels of leucocytosis, lymphacytosis and neutrophilia followed by complete recovery within the 45 hours before the next subsequent treatment.

In the U.S.S.R. Gorodelskaya (Dodge 1965) reports that white mice, exposed for five minutes to a density of 0.4w/cm.^{2} pulsed at 577 cycles/second, showed a decrease in erythrocytes, hemoglobin and leucocyte counts immediately after exposure and until the tenth day after exposure when recovery began. Convectional heat produced less distinct changes and more rapid recovery.

Kitsovskaya (Dodge 1965) subjected rats to 3000 megacycles as follows: (1) 60 minutes exposure at a density of 10 mw/cm.^{2} for 216 days. (2) 15 minutes exposure at a density of 40 mw/cm.^{2} for 20 days and (3) 5 minutes exposure at a density of 100 mw/cm.^{2} for 6 days. In the first treatment total white blood cells and absolute lymphocytes decreased and granulocytes increased. At the two higher densities total erythrocyte and leucocyte and absolute lymphacyte count decreased while granulocytes and reticulocytes increased.

Michaelson et al (1967), working with dogs, found a marked decrease in lymphocytes and eosinophils after six hours exposure at 100 mw/cm.^{2} and 2800 megacycles/second. After two hours exposure to 165 mw/cm.^{2} there was a slight decrease in all white cells which was less evident twenty-four

hours later. When exposure time was increased to three hours an immediate increase in leucocytes was observed which was more pronounced at twenty-four hours. Clumping of the leucocytes in some of these dogs was noted several months after irradiation.

Other organs of the body may be affected as shown by Michaelson et al (1967) who reports altered thyroid function in dogs with the use of I^{131} studies. Nikoyosyan (Dodge 1965), studying cholinesterase activity in rabbits subjected to 10 cm. waves, found that with an intensity of 40 mw/cm.^{2} a decrease in cholinesterase activity of the blood serum was produced. Also cholinesterase activity of the internal organs of rats and rabbits decreased. This lead to the assumption of increased acetyl choline in the organism. Michaelson et al (1967) quotes Nieset as having found that young animals (species not given) exposed repeatedly to 3000 megacycles/second (no density given) had a 4-6% increase in growth rate. He also quotes Richardson as having found that rats, given repeated doses of 2450 megacycles/second gained weight faster than the controls.

Numerous workers, particularly in the U.S.S.R., have reported that microwaves may have an effect on the cardiovascular system. Presman and Levitina (Dodge 1965), exposed rabbits to non pulsed 12.5 cm. wavelength microwaves with a field intensity of 7-12 mw/cm.^{2}. Each animal received radiation 12-13 times for twenty minutes. Dorsal radiation had

a positive chronotrophic effect while ventral stimulation produced a negative influence. All of these cardiac rhythm effects lasted only thirty to forty minutes after irradiation ceased. The authors felt that these rhythmic cariations reflected the action of microwaves on the skin and vascular receptors or on cells of the cerebral cortex. These authors repeated a similar experiment using 10 cm. waves of 3-5 mw/cm.^{2} intensity pulsed at 700 impulses per second. The chronotrophic effects were the same as with non-pulsed waves except more pronounced. Levitina (Dodge 1965) locally irradiated rabbits on different portions of the body locally with two types of high intensity microwave radiations: (1) continuous pulse, wavelength 12.5 cm., pulse duration of 100 m sec., pulse frequency of 2 pulses/second, density of 740-1250 mw/cm.^{2}, and (2) wavelength 10 cm., pulse duration of 1000 m sec., and density of 350-385 mw/cm.^{2}

This local irradiation reduced cardiac rhythm regardless of intensity. Dorsal irradiation with low intensity produced a positive effect and high intensity a negative effect. However, if the skin was anesthetized no effect was produced. This tended to support the hypothesis of Presman that reduced cardiac rhythm is a result of microwaves acting on thermal receptors in the skin.

Kaplan et al (1970) found no change in heart rate during or after twenty minutes of radiation of the dorsal aspect of the head of albino rabbits with 24 GHz CW microwaves at 10 mw/cm.^{2} before any change in heart rate could be effected.

The most recent interest in the biological effects of microwaves has been in their effect on the function and morphology of the central nervous system. According to Dodge (1966) Tolyskaya (1959) compared the thermal effects and non-thermal effects on various organs when the whole body of rats was irradiated with 10 cm. waves. Exposure to fields of 40-110 mw/cm.^{2} resulted in pericellular and perivascular edema in the nervous system, both massive and minute cerebral haemorrhaging, vascularization, and protoplasmic swelling of the brain cells. In the slightly thermal case at 19-31 mw/cm.^{2} level, the same results were obtained as well as severe protoplasmic swelling of parenchymatores nerve cells and significant cerebral microglial activity. In the non-thermal case of 10 cm. waves for thirty minutes (7.0-9.5 mw/cm.^{2}) even more pronounced vascularization in neural structures than in other organs was found. The authors concluded that the thalamus and hypothalamus are the most sensitive structures to microwaves.

It should be noted that Murray (1963) quotes Carpenter (1962) as having produced lenticular opacities by repeated subthreshold exposures of rabbits to a density capable of producing opacities in one exposure by thermal effect. By subthreshold he indicates the exposures were each not long enough and had sufficient interval between them to prevent the rise in temperature necessary to give opacities by thermal effect. This would indicate that both thermal and non-thermal

effects can take place at the same density depending upon the exposure time to the microwaves.

Lobanova (Dodge 1966) investigated the effects of non-thermal 10 cm. waves on the cytomorphology of interneuron connections. She found the fine projections of the dendrites were in the process of disappearing and in some cases thickening and swelling. Increasing the number of exposures caused the dendrite formation to extend deeper into the cortex toward the nerve cell itself. She concluded that the changes in higher nervous activity of the animals induced by microwaves was a function of interneuron disruption.

Kholodov (Dodge 1966) studied radiations of 1000 v/m on neuronally isolated sections of the cerebrum and mid brain of rabbits. With an exposure duration of 2-3 minutes, he found the latent period of the reaction of a neuronally isolated section was reduced (from 53 to 27 seconds) as opposed to the intact brain.

Kamenskiy (Dodge 1966) studied the functional state of the frog nerve exposed to pulsed and continuous waves 10-125 cm. When exposure was to continuous waves (wavelength 12.5 cm., density of 11 $\mu w/cm.^2$, and exposure time of 20 minutes) no evident changes in threshold activity were noted. However there was increased conduction rate, abbreviated absolute and relative refractory phases and altered current amplitude. With pulsed waves there was a definite increase in neural conduction and excitability resulting from non-thermal effects.

Goroditskaya (Dodge 1966) studied the effect of 3 cm. wavelength, pulsed at frequencies of 577 c.p.s. and at a density of 0.4 w/cm.^{2}, on the behaviour of mice and their progeny, 10 cm. away from a generator. The animals exhibited more pronounced conditioned reflex reactions in the form of negative responses than did the controls.

Faytel'berg-Blank (Dodge 1966) found a 10 minute exposure of the epigastral region to a 70 V, 12.6 cm. wave field did not change the intestinal glucose absorption in a dog whose solar plexis had been eliminated. He concluded that sympathetic nerves participate in the transmission of microwave effect from the epigastral region to the intestinal region. When vagosympathetic nerves were blocked at the neck, the stimulating effect of microwaves or ultra high frequency (U.H.F.) waves was impeded. When spinal ganglia were blocked, U.H.F. slightly elevated the level of reabsorption. Since these shifts in reabsorption were observed even with denervation he suggests humoral effects by U.H.F.

Yatrenko (Dodge 1966) investigated the effects of a twenty minute exposure to 12.6 cm. waves at a power level of 40 W on the absorption of radioactive phosphorus by knee-joint synovial membranes in normal animals and those with severed spinal columns. Absorption activity increased during U.H.F. radiation, spinal chord alterations retarded absorption but it again increased under U.H.F. radiation. The above author felt that this indicated a direct action of microwaves on

synovial receptors.

Semenov (Dodge 1966), investigating the thermodynamics of enervated and denervated femoral tissue in rabbits exposed to 12.6 cm. waves at a power density of 15^{o} mw/cm.^{2} at three, four and twelve hour intervals. He attributed a thermal cumulation or summation to neuroreflectory processes in the central nervous system. Testing this hypothesis he anesthetized animals and then increased the power density to 300 mw/cm.^{2} and found that the cumulative thermal effect was almost entirely precluded.

Malokhov (Dodge 1966) attempted to develop conditioned reflexes to a 13.7 cm. field (20 mw/cm.^{2}) in mice by using a conditioning signal of 20 seconds and 15 second unconditioned signals. While reflexes to U.H.F. were demonstrable, they were characterized by a long period of development, instability, short residual effect and rapid extinction.

Baldwin, Bach and Lewis (1960) when exposing the heads of young Macaca monkeys to exposures of 100 watts for ten to thirty minutes, observed that the clinical signs developed in sequence. The sequence began with agitation, followed by a startled reaction which was in turn followed by akinesis. This was followed by either drowsiness or agitation. Thereafter a seizure might follow. The majority of these signs disappeared without a trace when exposure was discontinued. The nature and severity of clinical signs varied with the head position. Elevation of the chin accelerated the effects.

Finally there was some histological evidence of an intraneural disturbance. The Nissl substance was washed out of many neurons. This may have been the result of molecular change subsequent to the electronic activation.

Throughout the literature we are reminded that although microwaves produce many effects in experimental animals, human beings, for example, have a larger capacity for dissipating heat (Kalant 1959). Most of the information on human subjects is the result of people coming in contact with microwaves in the course of their employment. Thus the distances from sources of radiation and times of exposure were not controlled as they would be in a planned experiment.

The first report of radar hazard is by Daily (1943) involving low power radar sets of that era in which case there was no evidence of radar induced pathology. However, McLauglin (1957) reports an individual proportedly killed by a high power radar set but this was disputed by other workers (Kalant 1959). In any case, the above claim of harmful effects, together with pathological effects in animals referred to earlier, have kept the area of clinical study open.

Barron and Barroff (1958) of the Locheed Aircraft Corporation studying two hundred and twenty-six employees with between two to five years exposure to radar found no pathology or adverse physical effects unequivocally attributable to radar.

Kevork'yon (Dodge 1965) investigating eighty-seven people exposed to microwaves for from two to three years found in a

proportion of cases complaints of headache, sleepiness, heartburn, weight loss, etc. Only fifteen people had no complaints. Objective investigation revealed hypotonia, bradycardia, dermographism, extremity tremors, pupil dilation, low blood pressure and loss of cutaneous sensitivity in a proportion of the cases studied.

Osipov (Dodge 1965), investigating people working in a microwave field of intensity range 300.3 - 43.2 V/m., found moderate body temperature increase, slowed pulse, and lowered blood pressure.

Korsun and Mikhaylov (Dodge 1965), examining personnel working around radar generators, tended to agree with the American work and to attribute the differences observed in cardiovascular function to other factors in the workers environment besides the microwaves. Drogechina (Dodge 1965), in a survey of people whose work exposed them to U.H.F. for lengthy periods of time, observed hypotension, bradycardia, form shifts in the parasympathetic nervous system, auriculoventricular conduction was lengthened, extrasytole was intensified and cardiac rhythm slowed. He also found that the thyroid gland is extremely sensitive to microwave emanations. In many cases exposed persons had hyper-thyroid activity. In some cases the actual volume of the gland increased, which however rarely caused alteration in function. Changes in humoral regulation were reflected in an almost two-fold increase in blood histamine and disassociation of individual

protein fractions of the blood.

Kapitanenko (Dodge 1965) also found that chronic microwave exposure lead to cardiac shifts, alterations in the nervous system and leukopenia. Revuts'kyy and Eydel'man (Dodge 1965) applied local fields of 13.56 mv. and 23.75 mv., and wavelengths of 22.12 M. and 12.6 cm., to patients with chronic diseases of the intestine and stomach. The effect on the blood histamine content decreased with the 22.12 M. treatment while the activity of the specific cholinesterase activity was unchanged. When a 12.6 cm. field was used, the blood histamine content remained unchanged, but the activity of the specific cholinesterase of the erythracytes decreased. This would indicate that these two wavelengths differ in biologic effect.

Not all of the effects of microwaves on biological systems are adverse. The use of microwaves in connection with therapy was first mentioned by Krusen et al (1947). Since that time the method has gradually gained a certain amount of acceptance in treating specific conditions. Krusen has shown that the depth of tissue heating at medically used frequencies extends to about 3 cm. Most generators used for therapeutic purposes operate between 2000 and 3000 megacycles per second on wavelengths of 10-15 cm. (Scott 1965). Time of patient exposure varies from a low of ten minutes to a high of thirty minutes. According to Scott (1965) the majority of treatments in the United Kingdom take about twenty minutes. Microwave diathermy does not have the penetration depth of short waves

but deeper than infra-red or radiant heating.

Scott (1965) reports treatment of many different types of lesions with microwaves. These include recent sprains, arthritis, tension headaches, disc syndromes and whiplash. In spite of the danger of lens opacities as reported in animals (Michaelson et al 1967, Herrich and Krusen 1952 and Clark 1950),Scott (1965) indicates eye conditions including iritis, cyclites, keratitis, blypharites etc. have been treated. Pelvic conditions have also been treated, but Scott (1965) could not see the value of this treatment because of a limited depth of penetration. He also reports that dental conditions of a superficial nature such as gum abcesses and opicil abcess have been treated. Mastitis furunculitis, sweat gland abcess, sinusitis and sciatica have also been treated.

The effect of microwave treatment on the behaviour pattern of birds has been studied in the hope of altering or controlling flight. Deichmann et al (1959) subjected ten day old bantam chicks to microwave power densities of 0.17 $w/cm.^2$ and 0.27 $w/cm.^2$ for five hours a day on each of ten days over a twelve day period. During the first and second periods of exposure to the central area of the radar beam, a chick would stagger and demonstrate muscular weakness or collapse. Occasionally, tonic or clonic spasms of the legs and wings were noticed. Recovery occurred between five and ten minutes later while exposure continued. During subsequent exposures

the birds avoided the central area of the beam.

Tanner (1966) subjected fourteen week old English Game hens to power levels in the range of 10 - 30 mw/cm.^{2} at a frequency of 16,000 Mc/S and 8000 pulses per second. With an antenna mounted above the cage, in a few seconds after the onset of radiation, sustained extensor activity of the wing and leg occurred. The author felt this to be due to the possible induced electrical activity in the spinal column. Shielding first the head and then the body did not change the manifested extensor activity. Tanner (1967) repeated these experiments with seagulls and pigeons. The pigeons registered distress and unsteadiness of gait but not the extensor reaction. The seagulls registered distress and unsteadiness but were able to shrug off the muscular effects by flapping their wings. Tanner (1966) suggested that the short exposure time (less than 60 seconds) precluded thermal effects as the cause of the extensor reaction.

Tanner et al (1967) conducted experiments on three species of birds using 9.3 Mc/S pulsed at 416 p.p.s. with a pulse width of 2.3 U sec. The average field intensity in the test cage was 46 mw/cm.^{2}. At the onset of treatment the wing outside the field of radiation collapsed and the opposite wing was extended. The legs showed a similar reaction. The chickens inclined their heads so that the eye closest to the field was oriented to the field and the sagital axis of the head was kept in line with the appropriate axis of the body.

The outer side of the body was paralyzed and when the bird reached the floor of the cage the reaction was manifested by increased extensor reaction of the field side of the head. Occasionally animals turned a hyperactive side to the field before the above reaction took place. The above worker noted that different reactions could be elicited by radiating different portions of the bird.

Subsequently, Tanner et al (1969) performed variations in this treatment using similar equipment. Twelve birds were treated to determine the relationship of time of escape reaction and field intensity. Starting at 45 mw/cm.^{2} and taking a single reading at each step down to 5 mw/cm.^{2}, it was found that as the field intensity decreased the time to cause a reaction increased. A further test attempted to measure reaction time when repeated exposures were made to a field of constant intensity. In each case the response time decreased over the first three or four trials, then increased to a value twice the original. When X band (9.3 GHz) microwaves were used in a repeat of the original experiment the reaction was identical, however exposure time was twice that of K_u band to produce the same reaction.

Dorsal stimulation at 60 mw/cm.^{2} caused birds to ruffle feathers and move around the cage. Dorsal stimulation with an infra-red generator at 100 mw/cm.^{2} produced neither agitation nor distress after three minutes exposure. When the head and body were selectively shielded before application of

a microwave field, a slightly significant (5 seconds less) time to give the flanking reaction was noted with the head exposed.

Other tests were performed using pulsed K_u band with the horn antenna in the horizontal position. The radiated bird could orient itself so that the radiation to the head would vary between 50 mw/cm.^{2} and 20 mw/cm.^{2} depending on whether or not it faced the horn. A test was also conducted with the horn beneath the cage giving a density of 40 mw/cm.^{2} Both birds showed a startled reaction at the onset of radiation but only in the first case did this continue into agitation, flanking or initiation of flight. From these experiments Tanner concluded the escape reaction may be attributed not only to the heating effect of microwaves but also to parameters such as electromagnetic interaction with nerve structures, molecular resonance, or chemical excitation. He hypothesized that disturbance of the heat balance of the animal by microwaves was a contributary factor but one which may be secondary to the main effect. He has suggested that if the range of paralysis can be increased this effect may be useful in controlling birds around airports. The above work has dealt with radiations in the range of 3 to 1250 mw/cm.^{2}.

Kondra et al (1970) investigated the effect of microwave radiations such as those found in the vicinity of microwave communication towers by exposing birds in cages in the laboratory. The generator power in one copper cage was

0.02 picowatts/cm.^{2} This level simulated the level prevailing in the vicinity of a tower. In another cage, power was 400 picowatts/cm.^{2} or 20,000 times the level around a tower. Each of five groups of birds was treated for a fifty-six day period at a different age; another group was treated continuously from day one to 476 days of age at each of the high and low densities, plus one untreated control. The difference in weight at 8 weeks and 20 weeks as well as the feed conversion for these periods showed no significant treatment effect. Similarly, no significant treatment effect was found in age at sexual maturity or percent lay. When only the continuously radiated groups were considered, that is from day one to 476 days, both the high and the low density treatments showed significantly higher egg production than the untreated birds. The difference between high and low levels was not significant. When egg weights were compared, they were lower in the irradiated group. However, on the basis of grams of egg/hen/day no significant treatment effect was found. Thus only the frequency of ovulation was affected which the authors hypothesized is likely due to pituitary stimulation by microwaves. Significant differences in fertility between the high and low levels were attributed to chance differences in male fertility. Hatchability was non-significant while chick weight followed egg weight. Mortality was normal. It was concluded that microwaves radiation at up to 20,000 times the level found in the area of microwave towers had little if

any, effect on laying birds.

Romero-Sierra and Tanner (1970) also found egg production increased (13.7% over the control birds) when the birds were subjected to microwave intensities ranging from 0.18mw/cm.^{2} to 360mw/cm.^{2} at a controlled temperature of 70°F. to 80°F. This increased egg production did not result in reduced egg weight.

In 1960 Van Ummersen irradiated 48 hour chicken embryos with 2450 megacycles/second frequency at a density of 400 mw/cm.^{2} for periods between one minute and five and one-half minutes. No deviation from the controls was noticed up to four minutes, while four and one-half to five minutes caused abnormalities and five and one-half minutes proved fatal.

In the cases showing abnormalities the wing bud development was abnormally small, the posterior limb buds, tail and allantois which normally develop in the region posterior to the wing bud existed at the forty-eight hour stage only as potential areas. The posterior most portion of the embryo consisted of only three germ layer although the neural folds were present and there were differentiated extra embryonic blood vessels. The effect is to present differentiation of new structures. She reported from these observations that "microwave radiation appeared to inhibit cellular differentiation in the chick embryo". In structures which had already begun to differentiate, cellular proliferation continued, but no further degree of differentiation occurred.

Structures which had not yet begun to differentiate failed to do so.

Van Ummersen (1963) quoted by Carpenter and Livstone (1971) irradiated chick embryos within a terminal section of waveguide maintained under incubation conditions of temperature and humidity. At the waveguide powers employed (not stated) abnormalities of development resulted from a 5 to 12 minute irradiation or at a lower power (not stated) from 14 to 16 minutes irradiation. Yolk temperature reached 56° to 57°C. during the period of exposure.

Paff et al (1962) tested the effect of 24 GHz radiation in isolated hearts from 72 hour chick embryos. At a density of 75 mw/cm.^{2}, the thermal effect was so slight that cardiac rate did not deviate from normal limits. Nevertheless changes in electrocardiagram were noted in all cases, and persisted after irradiation ceased.

MATERIALS AND METHODS

A series of experiments were conducted on the effect of microwave radiation on embryonic development and post embryonic growth. These experiments will be denoted as follows:

A preliminary experiment, experiments 1 to 5 involving low density irradiation, and experiments A, B and C involving high density irradiation, all treatment irradiations were applied during embryonic development, whereas experiment 6 involved two levels of treatment during the growth period.

For exposure of eggs to microwave radiation during incubation, a special experimental incubator was used, consisting of two compartments each measuring 60 cm. wide x 81 cm. long x 72 cm. high. One compartment was lined with copper screening to confine the microwaves to this unit and to prevent their leakage to other areas. In this compartment the turning device was made of wood and the eggs were held in plastic filler trays to minimize reflection of the microwaves. The adjacent plywood compartment was not lined with copper screen and the turning device was of chromed steel. Microwave radiation was introduced into the screened compartment of the incubator by a coaxial lead, from a Varian LD 807 generator, connected to an antenna which was mounted on the sidewall 40 cm. from the bottom of this incubator compartment. The field density within the lined side of the incubator averaged 0.2 mw/cm.^{2} with a range of 0.1 mw/cm.^{2} to

0.4 mw/cm.^{2}. The unlined side of the incubator had a trace field density below 0.005 mw/cm.^{2}. When the eggs were not being subjected to microwave radiation, their incubation was continued in a Robbins Model 11 H incubator in a building located remotely from the treatment unit. All treated as well as untreated eggs were placed at 19 to 21 days incubation in the hatcher portion of the Robbins machine.

Preliminary Experiment:

In the preliminary experiment a total of 90 Cobb broiler eggs were treated as follows: 30 eggs were placed in the 0.2 mw/cm.^{2} treatment incubator; 30 eggs were placed in the trace density incubator for the first week of incubation, and 30 eggs were kept in the control machine for the duration of their incubation period. After hatching, the chicks were wing banded and grown in chick batteries to five weeks of age. The following records were kept: hatch weight, feed efficiency, weekly body weight and mortality. Eggs which failed to hatch were broken out and infertility or time of death was estimated.

Experiments 1 and 2:

Experiments 1 and 2 involved two identical series of treatments and will be considered together. In each of these experiments, 30 eggs per treatment were placed in the treatment incubator and, simultaneously, 30 eggs per treatment were placed in the trace incubator as shown in Table 1. Also two groups of 30 eggs each were incubated in the control in-

cubator for the entire period of incubation. The same data were recorded in the preliminary experiment.

Experiments 3 and 4:

As these two experiments are very similar, they will be considered together. In both of these experiments, 30 eggs per treatment group were exposed to microwaves during the periods of incubation as shown in Table 1. This also included two groups of 30 eggs each incubated in the control machine for the entire incubation period. In experiments 3 and 4, the chicks were grown to five weeks of age as in the previous two experiments except that in experiment 4 feed conversion data could not be obtained because of space problems.

Experiment 5: (Turkeys)

This test involved treatment of turkey eggs in the previously described incubation equipment. However the plastic trays were removed and replaced with paper mache egg trays with the bottom removed from each cup for improved air circulation. The use of turkey trays necessitated reducing the number of eggs per treatment to 25. The eggs used, of the Wrolstad strain, were treated as shown in Table 1. Also one group, the control, remained for all four weeks of incubation in the control incubator. After hatching, the birds were maintained for two weeks in batteries and then they were moved to floor pens. Data recorded consisted of hatch weight, weekly weight to 4 weeks, 12 and 20 week weights and mortality.

Eggs which did not hatch were broken out and infertility or time of death determined.

Experiment A, High Density:

Twenty-five eggs, of the Dekalb laying stock mating from the University of Manitoba poultry flock, were exposed to microwaves for 10 seconds on days 0, 1 and 2 of incubation. These eggs were placed at zero distance from a Philips DX 260 magnetron producing microwaves at a frequency of 2450 MGHz with a power input of 1200 watts. All of the eggs were incubated in the control incubator before and after treatment. Each day a different control group was removed from the control incubator along with the eggs to be treated and carried to the magnetron and back without being subjected to microwave radiation. One group remained in the control incubator for the full incubation period without being removed. The hatched chicks were maintained in batteries until 5 weeks of age while the following data was collected: hatch weight, weekly weight and mortality. The unhatched eggs were broken out to determine time of death or infertility.

Experiments B and C:

In the second and third high density experiments, the equipment used was identical to experiment A, High Density and the eggs were from the same source. The treatments were arranged as shown in Tables 2 and 3, and the density of irradiation at the distance from the source used is given in Table 3.

Experiment 6: Exposure During Growing Period.

Chicks of one egg type hybred and a meat (broiler) type hybred were subjected to microwaves, continuously at a frequency of 6 GHz and at two densities, during the growing period. The birds were housed in duplicate copper screened cages each 6.96 m wide x 3.65 m long x 2.28 m high, located in a well insulated force ventilated windowless poultry house. Each cage was divided into four pens, each 1.74 m x 3.65 m, so as to accomodate two replications of each of the two types of stock. Two waveguide radiators were suspended in each cage, one for each pair of adjacent pens, to provide a uniform field intensity at ground level. The power level was adjustable in each cage. Periodically the field intensity in each cage and within the poultry house was measured by a Stoddard NW-62 B field intensity meter using a calibrated parabolic antenna probe.

The field within the cages at high density varied from 400-420 picowatts (average 407 picowatts) and in the lower density cage from 0.021 - 0.022 picowatts (average 0.022 picowatts). Thus four low density pens were available. At one day of age, groups of 30 Cobb broiler hens and 4 cockerels were placed in each of the two high density pens and each of the two low density pens. Simultaneously, groups of 30 Dekalb laying stock chicks and 4 cockerels were placed in each of two of the high density pens and each of two of the low density pens. As controls two groups of broilers and two groups of

laying stock were housed in the same size pens outside of the microwave irradiated area in the same building. The birds were weighed at one day of age, 4 weeks, 8 weeks, 12 weeks, 16 weeks and 20 weeks and mortality records were kept. On weigh days the treatments were rotated from one pen to another within the same density treatment, i.e. the four pens at high density were rotated among themselves, the four pens at lower density and the four controls in an attempt to remove any pen effect.

RESULTS AND DISCUSSION

LOW DENSITY EXPERIMENTS

EFFECT OF TREATMENT ON HATCHING BODY WEIGHT

A one way analysis of variance of the preliminary experiment showed that hatching weights of chicks were significantly different at the 5% level but not at the 1% level. Duncan's test revealed that the chick hatching weights from eggs exposed to trace level of radiation were significantly less (P .05) than the chick hatching weights from the 0.2 mw/cm.^{2} density treatments or the control chick hatching weights. However the chick hatching weights of the two treatment groups were not significantly different from the control chick weights (Tables 4 and 5).

In experiments 1 - 4 the hatching weights were analyzed by a two way analysis of variance. The mean of both the treatment and trace densities was compared by a one way analysis to give an estimate of the effect of treatment at either level at different stages of embryonic development. The treatment and trace levels were considered as one of the variables to be measured (A) while the stage of incubation during which treatment was applied was considered as the other variable (B). In experiments 1, 2 and 3 there were no significant differences in hatching weights due to variables A or B (Tables 4, 6 and 7). In experiment 2 there was a significant effect (at 1%) due to the interaction between

variables A and B (Tables 4 and 6). The cause of this interaction is unknown. In experiment 4 there was a significant difference at the 1% level due to variable A while variable B and the interaction showed no significant difference (Tables 4 and 7). In this experiment (Table 4) the treatment level of microwaves gives a consistently lower hatching weight than the trace level of microwave radiation for all of the periods of exposure used. Only in experiment 4, where Duncan's test showed that those treated at either density for the 1st, and 2nd week were heavier than those treated for all 3 weeks, 2nd week only, 3rd week only, and the control was any significant difference in hatch weight due to stage of incubation at time of exposure found. However as the majority of the data (experiments 1, 2 and 3) gives no significant difference in hatch weight it is felt that this slightly lower hatch weight was probably due to slight differences in incubation conditions rather than the microwave radiation.

Experiment 5 (Turkeys) was analyzed by a one way analysis of variance as only one control group was available. A significant difference (5% level) in hatching weights was found (Tables 4 and 8). Duncan's test revealed that only the birds in the treatment and trace incubators during the third and fourth weeks of incubation showed a significantly lower hatching weight than the control group (Table 4). As both the treatment and the trace levels appeared to be equally affected while those which spent the entire period of in-

cubation subjected to the microwaves were not affected it is more likely that the disturbance involved in moving the eggs, or some other factor involved in changing incubation conditions at that time affected the hatching weight rather than the exposure to microwave radiation.

TREATMENT EFFECT ON GROWTH TO 2 WEEKS IN CHICKS, 6 WEEKS AND 20 WEEKS IN TURKEYS

In the preliminary experiment there was no significant difference in body weight at 2 weeks of age (Tables 9 and 10). In experiment 1 the density of microwaves (Factor A) significantly affected 2 week weight while the stage of incubation at the time of treatment (Factor B) also had a significant effect (5%) on 2 week weight. In experiment 1 birds given the treatment level of microwaves were consistently heavier than those given the trace level. In experiments 2, 3 and 4, however, variable A was not significant (Tables 11 and 12). Therefore treatment with these two levels of microwave radiation did not have a consistently detrimental effect on 2 week weight. Variable B was not significant in experiments 2, 3 and 4. The overall mean weight of both treatment and trace groups was calculated for each time of exposure. These means were compared, including the control, by a one way analysis of variance. They were significant in all 4 experiments (Tables 11 and 12). However, Duncan's test showed that throughout the 4 experiments there is no treatment or group of treatments which were consistently heavier or lighter than any other treatment or group of treatments.

There was a significant difference due to interaction between variables A and B at 2 weeks of age in experiment 3 (Tables 11 and 12). The cause of this interaction is not known.

The weights of the turkeys in experiment 5 were analyzed by a one way analysis of variance at 6 and 20 weeks of age. Neither 6 nor 20 weeks of age showed any significant difference (Tables 13 and 14).

The weights of the two strains of birds exposed to microwaves during the growing period were analyzed at 4, 8 and 20 weeks of age as a two way analysis of variance with the strains of birds as one variable and the density of microwaves as the other. The weights were significantly different only between the strains of birds used (Tables 16, 17 & 18). Thus low density microwave exposure during the growing stage (up to 20 weeks) did not affect growth.

TREATMENT EFFECT ON FEED EFFICIENCY

As only one value per group was available for feed efficiency in each experiment, this trait was analyzed between experiments by a two way analysis of variance with the density of microwave exposure as one variable (A) and the stage of incubation during exposure as the other variable (B). In experiments 1, 2 and 3 as only one estimate of feed efficiency for the exposure during the second week of incubation was available this treatment was excluded from the analysis (Table 19). Over the two weeks of growth there was no significant difference in feed efficiency due to variables A, B or the interaction between them (Table 20).

In the experiments involving exposure of two strains of chickens during the entire growing period the feed efficiency was analyzed according to strain of birds (A) and density of microwave radiations (B). Only the two strains of birds showed a significant difference in feed efficiency at 4 weeks (Tables 21, 22 and 23). The meat type strain required fewer grams of feed per gram of gain than did the laying strain.

TREATMENT EFFECT ON HATCHABILITY

Only one estimate of hatchability was available for each treatment combination (Table 24). The arcsin values of the percentage of fertile eggs hatched (Table 25) were compared by a two way analysis of variance combining data from experiments 1 - 4. There was a significant difference in hatchability due to exposure at different periods of incubation (Table 26). Duncan's test on the arcsin values of the percentage hatch revealed the eggs treated for all 3 weeks of incubation have a significantly lower hatchability than the control or those treated for third week only. The eggs treated for all three weeks had a lower over-all average hatchability for the four experiments. However it was the exceptionally low hatchability of 42.3% for this group compared to 88.9% for the control group in experiment 4 which led to the significant difference. This difference could not be attributed to the microwave treatments. Firstly, a difference this large did not appear in experiments 1 - 3. Secondly, when the treatments of first week only, second week only and third week only were examined, no difference

exists. Thus at no given point in this experiment did the microwave radiation appear to be limiting. As a possible explanation it appears from Table 24 that the longer a group of eggs remained in that compartment of the incubator the lower the hatchability became. This trend would tend to indicate that some slight difference in incubation conditions could have been detrimental rather than the microwave radiation. It was also noted in this instance that when the eggs, which did not hatch were broken out 31 out of 49 had died between 19 and 21 days of development. Two out of 49 had died during the first 48 hours of incubation. Very few embryos (7) were found to have died between 3 days and 18 days of incubation. If the microwaves were having a detrimental effect it would be expected that embryonic mortality would likely have occurred during the organization and early development, i.e. 1 to 5 days of the embryos development. This was not the case.

In experiment 5 (turkeys) an analysis of the hatchabilities was not carried out because of the lack of numbers. However both the treatment and trace density had lower hatchability than the control. Also the eggs exposed to the trace density for weeks 2 to 4 of incubation had a lower hatchability than the corresponding treatment density eggs. In the other treatments, including exposure for the entire incubation period, the situation was reversed. Thus no consistent effect on the hatchability of turkey eggs resulted from treatment.

TREATMENT EFFECT ON MORTALITY

The mortalities for the preliminary experiment and experiments 1 - 4 are presented in Table 27. The maximum mortality of 11.5% in the treatment at 3 weeks of incubation in experiment 3 represents 3 birds dying by 2 weeks out of a total of 26 birds hatched. The mortality for birds exposed up to 20 weeks of age to microwave radiation was less than 10% in all groups. The only mortality figures which proved unusual were those for the turkeys in experiment 5. The 5 birds in the group treated for four weeks which starved during the first week of brooding gave a 33.3% mortality. Although this could be a response to treatment it was not borne out by the groups treated for the first 2 weeks of incubation and the group treated for the last 2 weeks of incubation. If it was the microwave radiation which had been responsible for these deaths it would be expected that the mortality would also be high in one of these groups depending on the time of incubation when the microwaves became detrimental. This was not the case. Thus to the stage of life that birds were kept these low levels of microwave exposure did not effect post embryonic mortality.

HIGH DENSITY EXPERIMENTS

TREATMENT EFFECT ON HATCHING WEIGHT

In the high density experiments the analysis of weights was confined to groups in which 4 or more eggs hatched as it was felt that this would be the minimum number required to give an accurate sample. As so many groups in experiments A, B and C did not meet this requirement these experiments were analyzed by a one way analysis of variance with each group of eggs treated at a given time of exposure, density of radiation, and stage of incubation considered as a treatment.

In experiments A and B there was no significant difference due to the above treatments (Tables 28, 29, 30 and 32), whereas in experiment C chick hatching weights show a significant difference (1%) due to these treatments. Duncan's test shows that in most cases all control group displays a significantly higher hatching weight than those treated on either the first or second day of incubation (Table 31). On the other hand from the superscripts (Table 30) it is evident that differences in density of microwave irradiation on the length of exposure do not show any significant effect on chick hatching weights in this experiment. The fact that in most cases chick hatching weights of the treated groups were significantly lower than those of the control would tend to agree with the findings of Van Ummersen (1961) who exposed embryos to 400 mw/cm.^{2} for $4\frac{1}{2}$ to 5 minutes and observed a retardation in embryonic development. It is possible that all of the treatment densities

and times of exposure in our tests were adequate to produce some degree of retardation of embryonic development.

TREATMENT EFFECT ON 2 WEEK WEIGHT

Although treatment differences in 2 week weights of the birds in experiments A, B and C (Tables 33, 34, 35, 36 and 37) approach a significant level, such differences are not significant. Thus if a treatment effect was present, as evidenced by the hatch weights of experiment C it disappeared by 2 weeks of age.

TREATMENT EFFECT ON HATCHABILITY

The percentage of fertile eggs hatching in each group of the 3 high density experiments are presented in Tables 37, 38 and 39. They reflect the thermal effect of microwave radiation. The mean temperature rise of the eggs is given in Figure 1. In experiment B at 0 days of incubation there is no effect on hatchability. Eggs treated at 2 days incubation had almost a complete failure of the eggs, exposed to the two higher densities (1.02 w/cm.^{2} and 0.246 w/cm.^{2}), to hatch. Treated at day one of incubation the two highest densities, (1.02 w/cm.^{2} and 0.246 w/cm.^{2}), show harmful effect at 45 seconds and 150 seconds respectively.

In experiment C there was no difference in the hatchabilities of the control groups (Table 40). In this experiment there was no definite association between embryonic mortality and stage of incubation during exposure. However

as the length of exposure time decreased in the two higher densities the hatchability increased. At a microwave radiation of 0.246 w/cm.^{2} the longest exposure of 150 seconds was lethal on both day 1 and day 2, whereas exposure for 120 seconds was completely lethal to the first day of incubation stage only. If the response was a result of thermal effect of microwaves only, it would be expected that eggs exposed to 0.123 w/cm.^{2} for 210 seconds would have a lower hatchability than those exposed at 0.246 w/cm.^{2} for 150 seconds as a higher mean temperature is reached (Figure 1). However, from Table 39 this is not the case and therefore some other mechanism may be involved.

One of the possible causes of this difference is the coagulation of protein both in the egg constituents as a food source for the embryo and within the embryonic tissue itself. Herrick and Krusen (1952) quote a text book of comparative physiology as follows, "the coagulation temperature of a given protein may vary within wide limits, conditioned by the salt content of the solution, or even more by the hydrogen in concentration of the solution. For example, a 1 percent solution of crystallized egg albumen pH 4.8 precipitates (forms a coagulum) at about 60 degrees centigrade while at pH 4.39 the coagulation forms at about 80 degrees. At pH 4.25 or below, heat coagulation no longer occurs at 95 degrees centigrade the solution becoming opalescent". Although the times of irradiation in these experiments were set so that no

visible coagulation of the albumen or yolk took place, it is possible that some microscopic changes could have occurred. From looking at the data and realizing that the greatest effect on hatchability occurred when irradiation took place on the second day of incubation it is probable that it is the process of organization and development of the embryo rather than its nutrient supply which is being affected. If the effect on hatchability were through an influence on the nutrient supply then this effect should be similar on day 0, 1 or 2 of incubation.

As has been indicated in the literature there is also the possibility of other than thermal effects on the hatchability. Bigu Del Blanco (1969) suggests orientation effects such as Pearl chain formation (Sarto, Schwan and Schwartz, 1966), the lining up of particles in suspension along the direction of the impressed electric field, as a possible mechanism for non-thermal effects. Seth and Michaelson (1964) however feel that non spherical particles shorter than 15 μ will not be orientated by pulsed or non pulsed fields which do not overheat the tissues. It is unlikely in their opinion that any histological structure exists which is superficially sufficiently large and free to be orientated. In spite of this objection Bigu Del Blanco (1969) states "a feature of this effect" (Pearl chain formation) "seems to be that when applied to cells they cannot reproduce under this condition". This may be one of the causes for the reduced hatchability and lower hatching weight

of chicks.

Bigu Del Blanco (1969) also suggests that microwaves have an effect (what the effect is, is not stated) on bio-membranes and the rate of diffusion of bio-aqueous solutions through bio-membranes. If this was to slow down the absorption of nutrient through the cell wall or the dispersion of the waste products of cell metabolism out through the cell wall we would have another possible cause for reduced hatchability, i.e. early embryonic death. Freedenberg (1967) states however "the influence of external electric fields on the bilayer structure" (of cell membranes) "has not been established".

TREATMENT EFFECT ON MORTALITY

The mortality in experiment A consisted of 2 birds. Both of these from the 0 stage treatment, were due to starving during the first 3 days after hatching. In experiment B, 2 birds died in the 30 sec. exposure to density 1.02 w/cm.^{2} at 0 stage of incubation which starved during the first week of brooding. In experiment C one bird starved during the first week in each of the following treatments: 1.02 w/cm.^{2} for 45 seconds on day 1 of incubation, 0.123 w/cm.^{2} for 210 seconds on day 1 of incubation and 0.123 w/cm.^{2} for 210 seconds on day 2 of incubation. This mortality was well within the normal expected proportions and microwave radiation (up to 1.02 w/cm.^{2} for 45 seconds) was not considered to have any effect on post embryonic mortality to 2 weeks of age.

CONCLUSION

In the low density (0.2 mw/cm.^{2} or less than 0.005 mw/cm.^{2}) experiments data on hatchability, feed efficiency, hatching weight, 2 week weight and mortality supports the previous findings that exposure to microwaves at the above densities have no significant effect on embryonic development of chicks or turkeys. In addition the post embryonic growth to 2 weeks of age of these birds was not influenced by radiation treatments. The same is true with regard to two breeds of chickens exposed during the entire growing period to 20 weeks of age.

In the high density experiments the chicks hatched showed no difference in post embryonic growth as evidenced by non significant differences in 2 week weight and mortality. However, there was a definite detrimental effect on hatchability and possibly on hatch weight resulting from irradiation with higher densities of microwaves, i.e. 0.246 - 1.02 w/cm.^{2} The magnitude of this effect on hatchability was affected by, (1) the day of incubation on which irradiation took place, day 2 being more susceptible than day 0 or day 1, (2) the density of irradiation given,the two higher densities, 1.02 w/cm.^{2} and 0.246 w/cm.^{2} being more detrimental or even lethal than 0.123 w/cm.^{2} or 0.051 w/cm.^{2},(3) the time of exposure; as exposure time increases to 45 seconds for the 1.02 w/cm.^{2} density and to 150 seconds for

0.246 w/cm.^{2} density the hatchability decreases (experiments B and C). Thus exposure for 90 to 120 seconds to between 0.123 w/cm.^{2} and 0.246 w/cm.^{2} density microwave irradiation becomes lethal to chick embryos. There is evidence that this effect is not entirely thermal.

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A P P E N D I X 1

TABLE 1

PLAN OF MICROWAVE TREATMENTS OF CHICKEN AND TURKEY EMBRYOS AT DIFFERENT STAGES OF DEVELOPMENT

Lot	Embryonic Stages of Treatment					
	Experiments 1 & 2		Experiments 3 & 4		Experiment 5	
	(A) 0.2 mw/cm^2	(B) .005 mw/cm^2	(C) 0.2 mw/cm^2	(D) 0.005 mw/cm^2	(E) 0.2 mw/cm^2	(F) 0.005 mw/cm^2
1	1st Week	1st Week	1st Week	1st Week	1&2 Wks.	1&2 Wks.
2	1&2 Wks.	1&2 Wks.	1&2 Wks.	1&2 Wks.	3&4 Wks.	3&4 Wks.
3	1,2&3 Wks.	1,2&3 Wks.	1,2&3 Wks.	1,2&3 Wks.	1-4 Wks.	1-4 Wks.
4	2&3 Wks.	2&3 Wks.	2nd Week	2nd Week		
5	3rd Week	3rd Week	3rd Week	3rd Week		
7	Control	Control	Control	Control	Control	

TABLE 2 - DESIGN OF HIGH DENSITY EXPERIMENT B.

1*	0									1							
2	6.99			13.97			27.94			6.99			13.97			27.94	
3	45	30	20	150	120	90	300	240	210	45	30	20	150	120	90	300	240	210
4	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6
5		6			6			6			6			6			6	
6														6				

1*	2								
2	6.99			13.97			27.94		
3	45	30	20	150	120	90	300	240	210
4	6	6	6	6	6	6	6	6	6
5		6			6			6	
6									

1* - Day of incubation when treated.
2 - Distance of egg from source of microwaves (cm).
3 - Time of exposure to microwaves (seconds).
4 - No. of eggs given each treatment.
5 - No. of eggs in each travelling control group.
6 - No. of eggs in control not taken from control incubator.

TABLE 2 - DESIGN OF HIGH DENSITY EXPERIMENT C.

1*	1												
2*	6.99			13.97			20.96			27.94			
3*	45	30	20	150	120	90	210	180	150	300	240	210	
4*	10	10	10	10	10	10	10	10	10	10	10	10	
5*	10												
6													10

1*	2												
2*	6.99			13.97			20.96			27.94			
3*	45	30	20	150	120	90	210	180	150	300	240	210	
4*	10	10	10	10	10	10	10	10	10	10	10	10	
5*	10												
6													

1* - Day of incubation when treated.
2* - Distance of egg from source of microwaves (cm).
3* - Time of exposure to microwaves (seconds).
4* - No. of eggs given each treatment.
5* - No. of eggs in each travelling control group.
6 - No. of eggs in control not taken from control incubator.

TABLE 3.

Distance From Source	Density
6.99	1.02 w/cm.^{2}
13.97	0.246 w/cm.^{2}
20.96	0.123 w/cm.^{2}
27.94	0.051 w/cm.^{2}

measured by $\frac{\text{power absorbed}}{\text{antenna effective area}}$

TABLE 4 - MEAN HATCHING WEIGHTS OF CHICKS AND TURKEYS IN GRAMS EXPOSED TO LOW DENSITY MICROWAVE RADIATIONS AT VARIOUS STAGES OF INCUBATION.

Week of	Preliminary		Experiment 1			Experiment 2		
Treatment	Treat.	Trace	Treat.	Trace	Average	Treat.	Trace	Average
1	43.21	40.76	39.36	41.21	40.50	43.63	41.95	42.77
1 & 2			41.09	39.61	40.35	44.72	43.66	44.25
1,2 & 3			38.74	39.09	38.89	42.95	43.27	43.10
2 & 3			38.92	40.04	39.46	40.40	41.42	42.28
2			-	-	-	-	-	-
3			39.13	39.76	39.47	41.52	44.00	42.86
Control	43.61		40.86	40.32	41.10	43.43	44.13	43.84

TABLE 4 - (continued)

Week of Treatment	Experiment 3			Experiment 4		
	Treat.	Trace	Average	Treat.	Trace	Average
1	36.34	35.92	36.13	35.68	37.00	36.32
1 & 2	37.00	36.26	36.64	36.08	37.88	37.26
1,2 & 3	36.00	36.56	36.30	34.46	36.17	35.46
2 & 3	-	-	-	-	-	-
2	37.72	37.26	37.53	34.89	36.08	35.43
3	36.66	36.66	36.66	35.38	36.42	35.92
Control	35.85	35.93	35.89	35.50	35.82	35.67

Experiment 5 - Turkeys

	Treat.	Trace
1 & 2	53.53	52.18
3 & 4	49.53	48.50
1 - 4	51.66	54.41
Control	53.27	

TABLE 5 - ANALYSIS OF VARIANCE OF THE HATCHING WEIGHTS OF CHICKS TREATED FOR THE FIRST WEEK OF INCUBATION WITH LOW DENSITY MICROWAVE RADIATION - PRELIMINARY EXPERIMENT.

Source	df	MS
Treatments	2	50.68
Error	59	14.78
Total	61	

TABLE 6 - ANALYSIS OF VARIANCE OF THE HATCHING WEIGHTS OF CHICKS TREATED WITH 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION.

EXPERIMENTS 1 AND 2

	Experiment 1		Experiment 2	
Source	df	MS	df	MS
A	1	23.73	1	0.03
B	4	21.38	4	14.86
A+B	4	15.31	4	49.34
Error	227	13.99	212	11.38

ANALYSIS OF VARIANCE OF THE MEAN OF BOTH TREATMENT AND TRACE LEVELS INCLUDING THE CONTROL.

	Experiment 1		Experiment 2	
Source	df	MS	df	MS
Treatment	5	32.46	5	23.98
Error	279	14.86	262	12.48

TABLE 7 - ANALYSIS OF VARIANCE OF THE HATCHING WEIGHTS OF CHICKS TREATED WITH 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION - EXPERIMENTS 3 AND 4.

	Experiment 3		Experiment 4	
Source	df	MS	df	MS
A	1	2.58	1	108.86*
B	4	14.55	4	22.24
AxB	4	3.72	4	1.47
Error	246	12.58	233	9.81

ANALYSIS OF VARIANCE OF THE MEAN HATCH WEIGHT OF BOTH TREATMENT AND TRACE LEVELS INCLUDING THE CONTROL.

	Experiment 3		Experiment 4	
Source	df	MS	df	MS
Treatment	5	16.69	5	23.69*
Error	305	12.33	292	9.20

TABLE 8 - ANALYSIS OF VARIANCE OF THE HATCHING WEIGHTS OF TURKEYS TREATED WITH 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION - EXPERIMENT 5.

	Experiment 5	
Source	df	MS
Treatment	6	63.19
Error	101	26.30
Total	107	

TABLE 9 - MEAN WEIGHT OF CHICKS AT TWO WEEKS OF AGE GIVEN 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} MICROWAVE TREATMENTS AT VARIOUS STAGES OF INCUBATION.

Week of	Preliminary		Experiment 1			Experiment 2		
Treatment	Treat.	Trace	Treat.	Trace	Average	Treat.	Trace	Average
1	192	193	197	170	183[ac]	177	170	173[a]
1 & 2			200	184	191[bc]	179	187	183[a]
1,2 & 3			192	189	190[bc]	182	183	183[a]
2 & 3			199	203	201[b]	173	177	175[a]
2			-	-	-	-	-	-
3			192	185	188[ac]	173	174	174[a]
Control	196		171	179	175[a]	193	200	197[b]

1. Different superscripts indicate a significant difference at 5%.

TABLE 9 - (continued)

Week of Treatment	Experiment 3			Experiment 4		
	Treat.	Trace	Average	Treat.	Trace	Average
1	180	167	174[a]	196	197	196[ac]
1 & 2	173	184	179[a]	198	197	197[ac]
1,2 & 3	189	163	176[a]	199	193	195[ac]
2 & 3	-	-	-	-	-	-
2	181	173	174[a]	195	192	193[a]
3	178	179	178[a]	202	212	207[b]
Control	155	160	157[b]	210	206	206[bc]

1. Different superscripts indicate a significant difference at 5%.

TABLE 10 - ANALYSIS OF VARIANCE OF THE BODY WEIGHT OF CHICKS AT TWO WEEKS OF AGE, EXPOSED TO 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} MICROWAVE RADIATION DURING THE FIRST WEEK OF INCUBATION - PRELIMINARY EXPERIMENT.

Source	df	MS
Treatment	2	102.99 NS.
Error	57	769.32
Total	59	

TABLE 11 - ANALYSIS OF VARIANCE OF BODY WEIGHTS OF CHICKS (AT TWO WEEKS OF AGE) EXPOSED TO 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION - EXPERIMENTS 1 AND 2.

	Experiment 1		Experiment 2	
Source	df	MS	df	MS
A	1	5179.11*	1	53.95
B	4	2067.67	4	958.54
AxB	4	1068.70	4	388.92
Error	218	718.89	212	836.82

ANALYSIS OF VARIANCE OF THE MEAN OF TWO WEEK WEIGHT OF BOTH TREATMENT AND TRACE LEVELS INCLUDING THE CONTROL.

Source	df	MS	df	MS
Treatment	5	3481.59*	5	3761.95*
Error	268	814.9	264	837.01

TABLE 12 - ANALYSIS OF VARIANCE OF BODY WEIGHTS OF CHICKS AT TWO WEEKS OF AGE EXPOSED TO 0.2 mw/cm.^{2} OR 0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION - EXPERIMENTS 3 AND 4.

	Experiment 3		Experiment 4	
Source	df	MS	df	MS
A	1	2063.98	1	62.20
B	4	556.12	4	1185.37
AxB	4	2569.75*	4	732.17
Error	238	729.62	230	643.09

ANALYSIS OF VARIANCE OF THE MEAN OF TWO WEEK WEIGHT OF BOTH TREATMENT AND TRACE LEVELS INCLUDING THE CONTROL.

Source	df	MS	df	MS
Treatment	5	3360.46	5	1756.12
Error	295	719.80	286	665.71

TABLE 13 - MEAN WEIGHTS IN GRAMS OF CHICKS AT FOUR, EIGHT AND TWENTY WEEKS TREATED DURING THE GROWING STAGE WITH 0.2 mw/cm.^{2} OR < 0.005 mw/cm.^{2} MICROWAVE RADIATION.

	High Density		Low Density		Control	
	Broilers	Layers	Broilers	Layers	Broilers	Layers
4 Weeks	532	228	531	220	520	227
	517	235	526	227	504	227
8 Weeks	1361	629	1513	605	1500	621
	1522	626	1482	617	1500	617
20 Weeks	3420	1443	3494	1411	3519	1434
	3547	1429	3360	1350	3448	1389

TABLE 14 - ANALYSIS OF VARIANCE OF BODY WEIGHTS OF TURKEYS AT FOUR WEEKS AND TWENTY WEEKS OF AGE IN EXPERIMENT 5.

	4 Weeks			20 Weeks		
Source	df	MS	f	df	MS	f
Treatment	6	53255.80	0.59	6	200	0.80
Error	81	89932.11		78	247	
Total	87			84		

TABLE 15 - MEAN WEIGHTS IN Kgms. OF TURKEY POULTS AT SIX AND TWENTY WEEKS OF AGE IN EXPERIMENT 5.

	6 Weeks		20 Weeks	
Weeks	Treat.	Trace	Treat.	Trace
1 & 2	1.74	1.68	6.39	6.28
3 & 4	1.58	1.56	5.69	5.75
1 - 4	1.67	1.62	6.67	5.69
Control	1.71		6.48	

TABLE 16 - ANALYSIS OF VARIANCE OF THE FOUR WEEK WEIGHT OF CHICKS SUBJECTED TO 4.07 PICOWATTS/cm.^{2} OR 0.022 PICOWATTS/cm.^{2} DURING THE GROWING PERIOD.

Source	df	MS	f
Grand Total	426	-	-
Breeds	1	9,193116	8.18 6688	4.32
Treatments	2	6162.82	4.48
Breed x Treatment	2	699.49	0.50
Between Pens Within Treatment	6	891.82	0.34
Within Pens Within Treatment	415	2563.95	

TABLE 17 - ANALYSIS OF VARIANCE OF EIGHT WEEK WEIGHT OF CHICKS SUBJECTED TO 407 picowatts/cm.^{2} OR 0.002 picowatts/cm.^{2} DURING THE GROWING PERIOD.

Source	df	MS	f
Grand Total	424		
Breeds	1	400.96	2673.06
Treatment	2	0.23	1.53
Breed x Treatment	2	0.14	0.93
Between Pens Within Treatment	6	0.02	
Within Pens Within Treatment	413	0.32	0.06

TABLE 18 - ANALYSIS OF VARIANCE OF THE TWENTY WEEK WEIGHTS OF CHICKS SUBJECTED TO 407 picowatts/cm.^{2} OR 0.022 picowatts/cm.^{2} DURING THE GROWING PERIOD.

Source	df	MS	f
Grand Total	399		
Breeds	1	1959.63	3767.88
Treatments	2	0.94	1.80
Breed x Treatment	2	0.72	1.38
Between Pens Within Treatment	6	0.52	0.59
Within Pens Within Treatment		0.88	

TABLE 19 - FEED EFFICIENCY (gms. feed/gms. gain) 0-2 WEEKS OF AGE OF CHICKS TREATED WITH 0.2 mw/cm.^{2} OR<0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION.

Week of	Preliminary		Experiment 1		Experiment 2		Experiment 3	
Treatment	Treat.	Trace	Treat.	Trace	Treat.	Trace	Treat	Trace
1			2.01	1.99	2.24	2.17	1.93	2.22
1 & 2			2.03	2.14	2.37	2.21	2.08	1.93
1, 2 & 3			1.93	1.79	2.19	2.41	2.04	2.23
2 & 3			1.85	1.77	2.29	2.10	-	-
2							2.23	2.04
3			2.06	1.92	2.48	2.17	2.01	2.22
Control			1.83	1.74	2.66	2.56	2.19	2.03

TABLE 20 - ANALYSIS OF VARIANCE OF THE FEED EFFICIENCIES 0-2 WEEKS OF AGE OF CHICKS EXPOSED TO 0.2 mw/cm.^{2} OR $<$0.005 mw/cm.^{2} AT VARIOUS STAGES OF INCUBATION.

Source	df	MS
A	1	0.010 NS
B	5	0.014 NS
AxB	5	0.002 NS
Treatment	11	
Error	22	0.061
Total	33	

TABLE 21 - FEED EFFICIENCY TO FOUR AND EIGHT WEEKS OF AGE OF CHICKS EXPOSED TO LOW LEVELS 407 picowatts/cm.^{2} OR 0.022 picowatts/cm.^{2} DURING THE GROWING PERIOD.

Meat Type			Laying Type		
4 Weeks					
High	Low	Control	High	Low	Control
1.85	1.83	1.83	2.68	2.52	2.65
1.94	1.87	1.87	2.73	2.88	2.84
8 Weeks					
2.21	2.21	2.21	3.00	3.06	3.05
2.30	2.20	2.21	3.28	3.20	3.15

TABLE 22 - ANALYSIS OF VARIANCE OF THE FEED EFFICIENCIES TO FOUR WEEKS OF AGE OF THE CHICKS EXPOSED TO 407 picowatts/cm.^{2} OR 0.022 picowatts/cm.^{2} DURING THE GROWING PERIOD.

Source	df	SS	MS	f
A	1	447374.07	447374.00	144.82**
B	2	283.50	141.75	0.04
AB	2	1935	967.71	0.31
Treatment	5	449593		
Error	6	1853.5	3089.16	
Total	11	4681.28		

TABLE 23 - ANALYSIS OF VARIANCE OF THE FEED EFFICIENCIES TO EIGHT WEEKS OF AGE OF THE CHICKS EXPOSED TO LOW LEVEL MICROWAVE IRRADIATION DURING THE GROWING PERIOD.

Source	df	SS	MS	f
A	1	503070.74	503070.74	255.70
B	2	717.17	358.58	0.18
AB	2	175.51	87.75	0.04
Treatment	5	503963.42		
Error	6	11801.50	1966.91	
Total	11	515764.92		

TABLE 24 - HATCHABILITY OF CHICK AND POULT EMBRYOS SUBJECTED TO 0.2 mw/cm.^{2} OR <0.005 mw/cm.^{2} AT DIFFERENT STAGES OF DEVELOPMENT.

Week of	Preliminary		Experiment 1		Experiment 2		Experiment 3		Experiment 4		
Treatment	Treat.	Trace	Treat.	Trace	Treat.	Trace	Treat.	Trace	Treat.	Trace	
1	65.5	72.4	67.8	96.7	81.5	82.4	86.7	89.3	86.7	78.6	80.7
2			-	-	-	-	79.3	89.7	96.7	85.7	87.8
3			78.6	89.7	82.1	93.1	93.1	92.3	92.8	93.3	89.3
1 & 2			72.4	70.0	73.3	77.8	90.0	89.7	79.3	93.3	80.7
2 & 3			89.7	82.8	80.0	92.8	-	-	-	-	86.3
1,2 & 3			90.0	75.9	74.1	69.2	86.6	86.7	42.3	78.6	75.4
Control	75.9		85.2	92.6	85.2	96.8	90.0	96.5	88.9	100.0	90.0

Experiment 5 - Turkeys

	Treat.	Trace
1 & 2	60.0	64.0
2 - 4	60.0	40.0
1 - 4	60.0	68.0
Control	78.2	

TABLE 25 - ARCSIN OF THE PERCENTAGE HATCHABILITIES OF EXPERIMENTS ONE TO FOUR.

Week of	Experiment 1		Experiment 2		Experiment 3		Experiment 4		
Treatment	Treat.	Trace	Treat.	Trace	Treat.	Trace	Treat.	Trace	
1	55.43	86.86	64.52	65.20	68.61	70.91	68.61	62.41	67.8
2	-	-	-	-	62.94	71.28	79.13	67.78	70.3
3	62.44	71.23	64.23	74.77	74.77	73.89	74.44	74.88	71.2
1 & 2	58.31	56.79	53.89	61.89	71.56	71.28	62.94	75.00	63.9
2 & 3	71.28	65.50	63.44	74.44	-	-	-	-	68.6
1, 2 & 3	71.56	60.60	59.41	56.29	68.53	68.61	40.57	62.46	60.9
Control	67.37	74.21	67.37	78.17	71.56	78.22	70.51	90.00	74.6

TABLE 26 - ANALYSIS OF VARIANCE OF THE ARCSIN VALUES OF THE PERCENTAGE HATCH OF THE CHICKS TREATED WITH 0.2 mw/cm.^{2} OR<0.005 mw/cm.^{2} AT DIFFERENT STAGES OF INCUBATION.

Source	df	SS	MS	f
A	1	32.47	32.47	0.54
B	6	917.14	152.35	2.55*
A X B	6	488.89	81.48	1.36
Treatments	13	1435.50		
Error	34	2024.34	59.53	
Total	47	3459.84		

TABLE 27 - PERCENT MORTALITY OF CHICKS (TO TWO WEEKS) AND TURKEY POULTS (TO SIX WEEKS) TREATED WITH LOW DENSITY MICROWAVE RADIATION AT VARIOUS STAGES OF INCUBATION.

Week of	Preliminary			Experiment 1			Experiment 2		
Treatment	Treat.	Trace	Control	Treat.	Trace	Control	Treat.	Trace	Control
1	5.2	4.7	0.0	0.0	0.0		0.0	4.3	
1 & 2				9.5	4.7		0.0	4.8	
1, 2 & 3				3.8	0.0	0.0 8.0	0.0	0.0	0.0 0.0
2 & 3				3.8	0.0		0.0	0.0	
2									
3				0.0	0.0		4.34	0.0	

TABLE 27 - (continued)

Week of Treatment	Experiment 3			Experiment 4		
	Treat.	Trace	Control	Treat.	Trace	Control
1	0.0	4.0		0.0	0.0	
1 & 2	3.7	0.0		4.5	0.0	
1, 2 & 3	11.5	3.8	0.0	2.0	0.0	0.0
2 & 3			3.7			6.8
2	0.0	3.8		0.0	4.1	
3	3.7	3.8		0.0	0.0	

Experiment 5 - Turkeys

	Treat.	Trace	Control
1 - 2	15.4	1.87	
3 - 4	7.1	10.0	
1 - 4	33.3	5.9	10.0

TABLE 28 - MEAN HATCH WEIGHTS OF CHICKS TREATED WITH HIGH DENSITY MICROWAVE RADIATIONS AT 0, 1 OR 2 DAYS OF INCUBATION - EXPERIMENT A.

Day of Incubation	Treatment	Travelling Control	Unmoved Control
0	42.52 gms.	41.10 gms.	
1	-	40.26 "	
2	-	- [1]	
			42.11 gms.

TABLE 29 - ANALYSIS OF VARIANCE OF THE HATCH WEIGHTS OF CHICKS RESULTING FROM EXPOSURE OF EGGS TO HIGH DENSITY MICROWAVE RADIATIONS AT 0, 1 OR 2 DAYS OF INCUBATION - EXPERIMENT A.

Source	DF	SS	MS	f
Treatment	3	40.49	13.49	1.28
Error	81	849.94	10.49	
Total	84	890.43		

1. Not recorded as no treatment chicks hatched.

TABLE 30 - MEAN HATCH WEIGHTS IN GRAMS OF CHICKS EXPOSED TO 1.02 w/cm.^{2}, 0.246 w/cm.^{2} OR 0.051 w/cm.^{2} AT DAYS 0, 1 OR 2 OF INCUBATION - EXPERIMENT B.

		Treatment			Control		
Density	Time	Day 0	Day 1	Day 2	0	1	2
1.02 w/cm.^{2}	45	-	-	-	38.20	39.83	37.16
	30	-	39.50	-			
	20	41.20	39.33	-			
0.246 w/cm.^{2}	150	-	-	-	-	41.60	-
	120	38.33	-	-			
	90	41.75	-	-			
0.51 w/cm.^{2}	300	38.83	39.40	-	42.25	-	-
	240	40.25	-	36.80			
	210	-	40.60	39.75			
Unmoved Control - 41.00							

TABLE 31 - MEAN HATCH WEIGHTS IN GRAMS OF CHICKS EXPOSED TO 1.02, 0.246, 0.123 AND 0.05 w/cm.^{2} ON DAYS 1 OR 2 OF INCUBATION - EXPERIMENT C.

Density	Time (Sec.)	Day 1	Day 2
	45	-	-
1.02 w/cm.^{2}	30	34.50^{acegikm}	-
	20	35.11^{acegikm}	37.11^{bdegikm}
	150	-	-
0.246 w/cm.^{2}	120	-	-
	90	34.71^{acegikm}	38.75^{bdfhjkm}
	210	34.57^{acegikm}	35.75^{acegikm}
0.123 w/cm.^{2}	180	37.57^{bdegikm}	36.92^{acegikm}
	150	35.09^{acegikm}	36.22^{acegikm}
	300	32.90^{acegikm}	38.50^{bdfhikm}
0.051 w/cm.^{2}	240	32.75^{acegikm}	35.87^{acegikm}
	210	34.33^{acegikm}	37.77^{bdegikm}
Travelling Control -		38.10^{bdegikm}	39.57^{bdfhjln}
Unmoved Control -			37.20^{bdegikm}

TABLE 32 - ANALYSIS OF VARIONS OF THE EFFECT OF HIGH DENSITY MICROWAVE RADIATION ON THE HATCHING WEIGHT OF CHICKS RESULTING FROM EXPOSURE OF EMBRYOS AT DIFFERENT STAGES OF DEVELOPMENT - EXPERIMENT B AND C.

	Experiment B		Experiment C	
Source	df	MS	df	MS
Treatments	19	12.33	19	29.32*
Error	80	9.39	147	11.23
Total	99		166	

TABLE 33 - MEAN TWO WEEK WEIGHTS OF CHICKS IN GRAMS EXPOSED TO HIGH D ENSITY MICROWAVE RADIATION AT 0, 1 OR 2 DAYS OF INCUBATION - EXPERIMENT A.

Day of Incubation	Treatment	Travelling Control	Unmoved Control
0	110.20	109.75	
1	-	118.00	
2	-		106.83

TABLE 34 - ANALYSIS OF VARIANCE OF THE TWO WEEK WEIGHTS OF CHICKS EXPOSED TO HIGH DENSITY MICROWAVE RADIATIONS ON DAYS 0, 1 OR 2 OF INCUBATION - EXPERIMENT A.

Source	df	MS
Treatments	3	328.25
Error	67	170.52
Total	70	

TABLE 35 - MEAN TWO WEEK WEIGHTS IN GRAMS OF CHICKS EXPOSED TO 1.02, 0.246 or 0.051 w/cm.^{2} ON DAYS 0, 1 OR 2 OF INCUBATION - EXPERIMENT B.

Density of Radiation	Exposure Time(sec.)	Treatment Weight Day 0	Day 1	Day 2	Control Weight Day 0	Day 1	Day 2	Unmoved Control
	45	-	-	-				
1.02 w/cm.^{2}	30	-	107	-	111	116	111	
	20	107	108	-				
	150	112	-	-				
0.246 w/cm.^{2}	120	107	-	-	-	125	-	
	90	108	99	-				
	300	120	117	-				
0.051 w/cm.^{2}	240	126	-	121	106	-	-	
	210	-	100	116				112
								115

TABLE 36 - MEAN TWO WEEK WEIGHTS OF CHICKS IN GRAMS EXPOSED TO 1.02, 0.246, 0.123 OR 0.51 w/cm.^{2} ON DAYS 1 OR 2 OF INCUBATION - EXPERIMENT C.

Density of Radiation	Time of Exposure (seconds)	Treatment Weight Day 1	Day 2
1.02 w/cm.^{2}	45	-	-
	30	126	-
	20	121	118
0.246 w/cm.^{2}	150	-	-
	120	-	-
	90	119	120
0.123 w/cm.^{2}	210	111	138
	180	127	133
	150	124	126
0.051 w/cm.^{2}	300	131	127
	240	130	119
	210	124	121
	Travelling Control	114	118
	Unmoved Control	124	

TABLE 37 - ANALYSIS OF VARIANCE OF THE TWO WEEK WEIGHTS OF CHICKS EXPOSED TO HIGH DENSITY MICROWAVES AT DAY 0, 1 OR 2 OF INCUBATION - EXPERIMENTS B AND C.

	Experiment B		Experiment C	
Source	df	MS	df	MS
Treatment	19	248.94	19	334.82
Error	81	146.89	144	205.22
Total	100		163	

TABLE 38 - PERCENT HATCHABILITY OF FERTILE EGGS EXPOSED TO HIGH DENSITY MICROWAVE RADIATIONS ON DAYS 0, 1 OR 2 OF INCUBATION - EXPERIMENT A.

Day of Incubation	0	1	2	
Treatment	72.4	10.3	0	
Travelling Control	74.1	86.6	83.3	
Unmoved Control				78.3

TABLE 39 - PERCENT HATCHABILITY OF FERTILE EGGS EXPOSED TO 1.02, 0.246 OR 0.051 w/cm.^{2} ON DAY 0, 1 OR 2 OF INCUBATION - EXPERIMENT B.

Density	Time of Exposure	Day of Incubation 0	1	2	Travelling 0	1	Control 2	Unmoved	Control
1.02 w/cm.^{2}	45	60.0	40.0	0	83.3	100	100.0		
	30	66.6	80.0	0					
	20	83.3	100.0	0					
0.246 w/cm.^{2}	150	83.3	16.6	0	100.0	83.3	50.0	84.6	
	120	100.0	80.0	0					
	90	83.3	83.3	16.6					
0.051 w/cm.^{2}	300	100.0	83.3	66.6	80.0	60.0	50.0		
	240	66.3	50.0	100.0					
	210	40.0	100.0	80.0					

TABLE 40 - PERCENT HATCHABILITY OF FERTILE EGGS EXPOSED TO 1.02, 0.246, 0.123 OR 0.051 w/cm.^{2} ON DAY 1 OR 2 OF INCUBATION - EXPERIMENT C.

Dist. from Source	Time of Exposure	Day of Incubation		Travelling Control		Unmoved Control
		1	2	1	2	
1.02 w/cm.^{2}	45	37.5	33.3			
	30	100.0	37.5			
	20	75.0	100.0			
0.246 w/cm.^{2}	150	0	0			
	120	0	40.0	100.0	100.0	100.0
	90	77.7	57.1			
0.123 w/cm.^{2}	210	100.0	88.9			
	180	100.0	87.5			
	150	100.0	100.0			
0.051 w/cm.^{2}	300	100.0	80.0			
	240	100.0	100.0			
	210	100.0	100.0			

A P P E N D I X 2

AT VARIOUS DENSITIES OF MICROWAVE RADIATION