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(57) **Abstract**

[00001] A hazard warning system radiates pulses of microwave energy in the frequency range of 1 GHz to about 40 GHz to alert and warn target flying birds of the presence of wind turbine electrical generators, power distribution systems, aircraft, and other protected areas from hazardous intrusion. The warning system includes a control unit governing pulse control circuitry that outputs pulses ranging from about 5  $\mu$ s to about 25  $\mu$ s in duration. These pulses trigger a pulsed source of microwave energy that is coupled to a microwave antenna that emanates the warning radiation. The radiation is sensed by the birds auditory system, attaining their attention to the presence of the protected area. The sensed radiation itself may cause the birds to veer from a collision course, or supplemental hazard-warning radiation including ultraviolet light and infrasound may also be employed. A proximity detector can enhance operating efficiency by steering the antenna toward a detected target. Further, the warning system can remain in a standby mode until alerted by the proximity detector to the presence of target birds, whereupon the warning system begins to output pulsed microwave energy. The pulse control circuitry may be caused to generate complex pulse trains that can preferably evoke a biologically significant response within recipient birds. The warning system operates at the speed of light, and can transmit a benign warning, transparently to humans. Not only is an area protected by the system, but the birds themselves can be protected from the area.

**1 Claim, 3 Drawing Sheets, and 4 Figures**

[00002] This is a continuation of application Ser. No. 08/598,093 filed Feb. 7, 1996, now abandoned which is a continuation of application Ser. No. 08/280,287 filed Jul. 26, 1994 now abandoned.

**FIELD OF THE INVENTION**

[00003] The present invention relates generally to systems that seek to ward off flying birds, and more specifically to systems for protecting wind turbine electrical power generators, electrical power distribution equipment, flying aircraft, and other objects from danger from flying birds.

**BACKGROUND OF THE INVENTION**

[00004] Flying birds can represent a safety hazard to many objects, and conversely such objects represent a hazard to the birds. Birds colliding with wind turbine electrical generators can not only damage the generators, but generally are injured or die as a result. For example, in northern California, the collision-death of eagles and other protected bird species has resulted in environmental litigation threatening to curtail operation of such generators. Further, birds colliding with high voltage power lines can break or short-circuit the lines, interrupting electrical power distribution. The resultant power outage can also cause damage to the equipment generating and distributing the high voltage. In addition, sparks from broken power lines can start fires.

[00005] Birds also present a serious danger to flying aircraft. A medium sized bird striking the windshield or engine an aircraft in flight can damage the aircraft, endangering the safety of those on board. Even modern jet aircraft are susceptible to damage from birds. Not only can they break the windshield, but birds can be sucked into the air intake of a jet engine. The resultant engine damage can require substantial maintenance to repair.

[00006] Birds that fly into skyscrapers, monuments and the like can also create serious danger. A bird flying into the window of a skyscraper may cause serious injury to persons on the ground cut by falling broken window glass. Although birds that fly into other large objects may not present as serious a hazard to humans, repairing any resultant damage can be risky and expensive.

[00007] It is known in the art to use flashing lights to try to ward birds away from an object. For example, U.S. Pat. No. 5,270,707 to Schute et al. discloses using flashing aircraft lights to ward off in-flight impact with birds. Apparently birds viewing the flashing lights may tend to change their course to avoid collision.

[00008] It is generally assumed that flying birds will not knowingly collide with an object. However, the efficiency of Schute et al.'s system is diminished unless the presence of the aircraft is indeed communicated to the birds by the lights. The birds may not be looking at the aircraft (and thus at the lights), perhaps because they are searching for food, are viewing predators including other birds. Alternatively, the lights may not be seen by the birds because visibility is diminished due to inclement weather.

[00009] It is also known to use reflector strips that are intended to deter birds in scarecrow-like fashion from a protected area such as a garden. For example, power utility companies often mount passive strips of reflecting material near high voltage towers and lines in an attempt to ward off birds. However, such passive strips are of little use during darkness or other periods of diminished visibility. Further, such passive strips do not actively communicate their presence to the birds, whose attention may in fact be directed elsewhere.

[00010] Further, because reflector strips tend to present a substantially constant stimulus pattern to the birds, whatever warning effect they initially provide soon diminishes. This effect is termed "adaptation", and is analogous to the ability of humans to disregard a continuous loud noise (or other stimulus) after hearing the noise for a few minutes.

[00011] It is also known to use audible sound waves to try to ward off impact by flying birds. However, if the environment to be protected from avian collision is noisy, the efficiency of such sound waves is diminished. Further, because acoustic waves propagate at only about 1,100 feet/second (335 m/second), any benefit they might provide can literally occur too slowly to be of use.

[00012] Unfortunately, the use of light energy or acoustic energy is conspicuous and thus not transparent to humans in the vicinity. What is meant by "not transparent" is that humans not intended to be the target of the hazard communication system can sense the object-announcing light or sound radiation, and become distracted or otherwise annoyed.

[00013] In general, active hazard warning systems, e.g., flashing lights or radiated sound waves, tend to be active at all times, even if potential danger from flying birds is not present. Such constant activation is not always desirable, especially if the lights or sound will needlessly annoy humans in the area to be protected. In addition, constant activation is undesirable because it causes adaptation, whereby the recipient birds soon pay little or no attention to the stimulus. Further, constant activation of such systems wastes operating electrical power, and shortens the working lifetime of the hazard warning system.

[00014] Thus, there is a need for a hazard communication system to alert and warn birds of the presence of an object or area with which a collision should be avoided. Such system should preferably be transparent to humans in the protected area, and should communicate its warning at the speed of light.

[00015] Further, the efficiency of the warning should not be substantially diminished by poor visibility or ambient acoustic noise, or by the target not looking at the source of the system radiation. Such system should also minimize adaption by the birds receiving the warning. Finally, such system should be capable of activation only when a target is sufficiently close to the protected region to warrant communicating the presence of the protected region to the target.

[00016] The present invention discloses such a system.

#### **SUMMARY OF THE INVENTION**

[00017] The present invention communicates the presence of a protected area to flying birds and other vertebrates by radiating pulses of microwave energy to announce the presence of such area. Applicant has discovered that 5  $\mu$ s to 75  $\mu$ s pulses of microwave radiation in the 1 GHz to 40 GHz range are sensed by the birds, apparently by stimulating their auditory system.

[00018] The pulsed microwave radiation attains the birds' attention in a benign manner, serving to communicate the presence of the protected area to the birds. This attention-grabbing warning is communicated to the birds at the speed of light, without regard to visibility conditions, and without regard to whether the birds happen to be looking toward the protected area.

[00019] The effect of the pulsed microwave radiation upon the birds' auditory system may itself cause the birds to veer off course, to avoid collision with the now-noticed protected area. However, collision avoidance may be further enhanced by providing supplemental hazard-warning radiation including light, ultraviolet, and/or sound, including infrasound. The efficiency of such additional warning radiation may be promoted because the birds' attention will have been attained by the present invention.

[00020] The present invention includes a control unit that governs pulse control circuitry whose output triggers a pulsed source of microwave energy in the L through K.sub.a bands. The microwave energy is coupled to a microwave antenna system that emanates the protective radiation to announce the presence of the protected area. The microwave energy preferably is radiated at average power levels in the approximate range 1 mw/cm.sup.2 to 10 mw/cm.sup.2 for considerations of environmental safety. The present invention can be used to communicate to birds the presence of diverse protected areas or objects, for example wind turbine power generators, power transmission systems, and airborne aircraft.

[00021] To minimize stimulus adaptation by the recipient birds and to promote effective communication of the warning, the pulse control circuitry preferably permits generating complex, pulse-code modulation type waveforms. Suitably complex pulse trains are believed to evoke a biologically relevant response, causing the recipient bird to be more alert to the warning.

[00022] The antenna system preferably is steerable to increase the effective range of the protected area. The present invention may include an optional proximity detector that can detect the presence of nearby targets. The output from the proximity detector may be used to steer the antenna toward the detected target, thus enhancing system operating efficiency.

[00023] Further, the proximity detector allows maintaining the present invention in a standby mode until such time as oncoming targets are detected by the proximity detector. Upon target detection, the present invention enters an active mode, and emits the microwave radiation for as long as the targets remain in proximity to the protected area. Such bimodal system operation both conserves operating power and extends the lifetime of the system. Further, such operation tends to reduce adaption by recipient birds.

[00024] Because the hazard communicating system uses microwave rather than visible light or acoustic energy, the present invention is transparent to humans not within the radiation target range of the antenna system. Further, the system remains transparent if the supplemental hazard-warning radiation is ultraviolet.

[00025] Other features and advantages of the invention will appear from the following description in which the preferred embodiments have been set forth in detail, in conjunction with the accompanying drawings.

#### **BRIEF DESCRIPTION OF THE DRAWINGS**

[00026] FIG. 1 is a block diagram of a hazard communication system, according to the present invention;

[00027] FIG. 2 depicts protection of a wind turbine power generator with a hazard communication system, according to the present invention;

[00028] FIG. 3 depicts protection of a power transmission system with a hazard communication system, according to the present invention;

[00029] FIG. 4 depicts protection of an aircraft in flight with a hazard communication system, according to the present invention.

#### **DETAILED DESCRIPTION OF THE PREFERRED EMBODIMENT**

[00030] FIG. 1 depicts a hazard communication system 10 as emanating a primary microwave radiation 12 that attains the attention of birds 14 (and possibly bats) within the effective range of the radiation. By thus attaining the immediate attention of the birds, the birds may be warned of the presence of a protected region, and a collision by the birds with the protected region may be avoided.

[00031] Alternatively, supplemental secondary emitters may cause the birds to veer off course, avoiding a collision. Such secondary emitters are more likely to be noticed by the birds, whose attention has been attained by the primary pulsed microwave radiation.

[00032] More specifically, the present invention 10 includes a control unit 16 that governs operation of pulse control circuitry 18, whose output triggers a pulsed source of microwave energy 20. The pulsed microwave energy is fed to a microwave antenna system 22 and radiated as primary microwave energy 12. Pulse control circuitry 18 preferably outputs fixed duration pulses having a pulse width in the range of about 5  $\mu$ s to about 75  $\mu$ s. For a given pulse width, the duty cycle of the pulse train is determined from the average power level of microwave energy to be provided. A narrower pulse width will have a higher duty cycle for a given level of average power than will a longer pulse width.

[00033] Optionally, a pulse modulator unit 26 is coupled to the pulse control circuitry 18. Modulator unit 26 introduces a modulated variation in the time between preferably fixed-duration pulse widths, which duration varies from about 0.001 Hz to about 10 KHz. Modulation from unit 26 can advantageously reduce adaptation in recipient birds by varying in a complex manner the patterns of output radiation 12. Further, by suitably programming unit 26, the auditory system of recipient birds 14 may be stimulated using non-thermal microwave energy 12 to evoke biologically relevant responses. For example, a complex pattern of pulsed microwave radiation 12 ideally would evoke the same response in a recipient bird 14 that the alarm call or warning shriek of another bird would evoke. However, evoking other responses could also attain the birds' attention but perhaps less effectively.

[00034] Central control unit 16 monitors and controls pulse control circuit 18, which in turn controls the pulse parameters associated with microwave energy source 20. Central control unit 16 typically will also include bi-directional communications to a remote operator site. Safety interlock 24 provides for shutdown of system 10 in case of any potential danger to service personnel or to system components.

[00035] Pulse energy source 20 preferably includes a magnetron or a cavity oscillator operating in the range of about 1.0 GHz to about 40 GHz. Applicant has found that pulsed microwave radiation the range 1.0 GHz to about 2.5 GHz is quite effective, and radiation in the approximate range 15 GHz to about 25 GHz should be similarly quite useful. This microwave energy is coupled from source 20 to antenna system 22 using coaxial cable or waveguides. Antenna system 22 includes an antenna whose direction of radiation preferably can be steered to direct the primary microwave emissions 12 toward targets 14.

[00036] Applicant has discovered that the emission of such pulsed microwave radiation is sensed by pigeons, apparently by affecting their auditory system in a non-thermal manner. Even without emitting a complex pattern of pulses, the resultant effect upon the birds is believed perhaps to be analogous to a buzzing, clicking, or popping sensation in the ears of a human.

[00037] During testing by applicant, applicant exposed pigeons to S and L band pulsed microwave radiation, using fixed pulse widths in the approximate range 5  $\mu$ s to 75  $\mu$ s, and more preferably approximately 5  $\mu$ s to about 25  $\mu$ s. In providing this microwave energy, pulse width, pulse duty cycle and repetition rate into the pulsed microwave source were controlled to limit the radiation density to less than about 1 mw/cm.sup.2 to 10 mw/cm.sup.2, an environmentally safe level.

[00038] Under laboratory conditions, the ability of such birds to sense the microwave radiation was confirmed by monitoring a standard conditioned cardiac response. Within about three seconds after exposure to this microwave radiation, pigeons having a baseline heartbeat of about 100 beats/minute were found to experience a relatively sudden increase of about 40 beats/minute, a 40% increase. The onset of the heartbeat change occurred within a second or so of the onset of exposure to the pulsed microwave radiation. In reality, the pulsed microwave radiation is sensed instantly by the pigeons, and the delay in attaining a detectable change in heart rate represents a normal response latency.

[00039] While applicant's testing was directed to pigeons, it is believed that other birds and possibly bats would also sense their exposure to the pulsed microwave radiation.

[00040] As noted, applicant's pulsed microwave energy is sensed by birds, thus attaining their attention, which may include immediate communication to the birds of the presence of the area of object to be protected. Once aware, the birds can avoid knowingly colliding with the protected area or object. With reference to FIG. 1, after the presence of an area or object protected by radiation 12 is communicated to birds 14 by the radiation, the birds should take evasive action. Such action can include veering off a collision course, preferably by flying out of the target zone that is radiated by microwave antenna system 22.

[00041] Because avian hazard communication according to the present invention uses microwave rather than visible light or acoustic energy, it is transparent to humans not within the effective radiation path of the antenna system 22. Further, microwave radiation travels at the speed of light, and thus the attention-attaining effect of radiation 12 upon targets 14 can be realized relatively instantaneously. Further, it will be appreciated that the effectiveness of the emanating microwave radiation 12 is not diminished by inclement weather, or the direction in which target 14 may be at the moment.

[00042] Optionally, system 10 includes a hazard proximity detector 28 that can include motion detectors, heat detectors, simple radar systems, and the like. The function of detector unit 28 is to detect when targets 14 have approached sufficiently close to the area protected by system 10 to constitute a potential hazard.

[00043] Until detector 28 signals a potential hazard, control unit 16 and the remainder of system 10 may remain in a standby mode. In standby mode, no radiation is emanated by antenna system 22, and system 10 consumes relatively little operating power. However, when detector 28 signals that a target 14 is nearing the protected zone, control unit 16 causes pulsed microwave source 20 to output microwave energy that is radiated by antenna system 22.

[00044] Once targets 14 have sensed pulsed microwave radiation 12, are alerted to the presence of the area protected by system 10, and have veered their course or otherwise avoided the target zone, detector 28 once again returns system 10 to the standby mode. If desired, hazard proximity detector 28 can share the microwave antenna system 22 for detecting purposes. Further, upon actually detecting a target 14, the hazard proximity detector 28 can cause antenna system 22 to be pointed more directly at target 14.

[00045] Thus, the inclusion of a hazard proximity detector 26 can enhance the operating accuracy of system 10. In addition, adaptation by target birds 14 is minimized because radiation 12 is not always present. Further, by permitting standby mode operation until the need to emanate microwave radiation is actually at hand, proximity detector 28 helps conserve system operating power, and also increases system 10 operational lifetime.

[00046] Optionally, system 10 can also emit secondary radiations. For example, secondary source unit 30 can provide control signals and voltages to secondary emitter unit 32, which may include flashing lamps used with various lenses and/or filters, acoustic loudspeakers, and the like. These secondary light and/or acoustic emanations can provide additional warning to targets 14, and are more likely to be noticed after the target birds have sensed the primary radiation 12.

[00047] One useful secondary emission is acoustic energy 34, especially energy containing frequencies in the approximate range 0.001 Hz to about 10 KHz. Applicant has discovered that the auditory system of birds is quite sensitive at infrasound frequencies. Further, the emission of infrasound frequencies, e.g., 0.001 Hz to about 10 Hz advantageously is transparent to humans.

[00048] An earlier discovery of applicant is that birds recognize ultraviolet radiation 36 in the 305 nm to 400 nm range, and especially in the approximate range 325 nm to about 375 nm. Applicant has discovered that an effective way to generate such radiation transparently to humans is to filter components from the output of a strobe-type lamp 38. More specifically, the strobe-lamp output is passed through a filter sandwich comprising a type UG-1 Schott glass layer 40, one surface of which includes a preferably vacuum-deposited anti-red blocking coating 42. (While FIG. 1 shows coating 42 facing away from lamp 38, the sandwich may be reversed so coating 42 faces toward lamp 38.) The net effect is that radiation 36 is broad bandwidth ultraviolet, with no red components that would be visible to humans. Of course, if the radiation were not required to be transparent to humans, the anti-red coating layer could be omitted. The resultant radiation 36 would be broad bandwidth ultraviolet that included red components visible to humans.

[00049] In some applications, it may in fact be possible to warn birds 14 of a hazard by using a system 10 that includes secondary emitters 32, but that omits the pulsed microwave source 20 and antenna system 32.

[00050] FIG. 2 depicts the use of system 10 to warn birds 14 of the presence of a wind turbine generator system 50. Typically generator system 50 includes a tower 52 that may be perhaps 70 m or more in height, atop which is located an electrical generator 54 that is rotated by wind-blown turbine blades 56. Because the power generating efficiency of system 50 increases with the size of the blades, blades 56 may be 30 m or more in length.

[00051] Although FIG. 2 depicts system 10 as mounted at the top of tower 52, system 10 may be disposed elsewhere. It suffices if the pulsed microwave radiation 12 emanating from the microwave antenna system 22 effectively covers the region whose presence is to be communicated to birds 14 to avoid avian impact. If desired, operating voltage for system 10 may be obtained from generator 54, which typically is coupled to an electrical grid carrying electrical power. In applications where there is no coupling to an electrical grid, the generator 54 output voltage could be supplemented by a rechargeable storage battery. This would permit powering system 10, even when the absence of wind caused generator 54 to cease generating voltage.

[00052] Birds, bats or the like 14 approaching the protected area of system 10 sense the presence of the pulsating microwave energy 12, apparently by the radiation's effect upon their auditory system. The resultant auditory effect serves to attract the attention of the birds. The microwave energy 12 itself may cause the birds to avoid flying into the protected area, perhaps to minimize the auditory effect. Alternatively, the now more attentive birds may observe the protected area of system 10, and veer off course to avoid contact.

[00053] Optionally, if secondary sources and emitters (e.g., elements 28, 30 in FIG. 1) are included, such second radiation may also alert and cause the birds to change course to avoid a collision. Eventually the birds will fly out of regions of the antenna radiation path having sufficient energy density to affect their auditory system. Once the birds have so veered, they no longer endanger the portion of system 50 being protected. Conversely, the protected region would no longer threaten the birds.

[00054] The net result is that the hazard presented to system 50 and the hazard to birds 14 from an avian collision will have been avoided. Further, this hazard warning can occur at the speed of light, independently of weather conditions, and occurs transparently to humans in an area not encompassed by the antenna radiation field. In addition, it will be appreciated that the warning is benign in that the levels of emitted microwave energy preferably are sufficiently low in density to alert but not harm the recipient target birds 14.

[00055] FIG. 3 depicts the application of system 10 to warn birds 14 as to the presence of a protected area of an electric power distribution system 60. The purpose of such warning is to prevent a collision by the birds with the protected area. System 10 may (but need not) include the secondary sources and emitters 30, 32 and the hazard proximity detector 28 described earlier.

[00056] In FIG. 3, system 60 includes a utility tower 62 that carries various high voltage conductors 64, a transformer and/or other equipment 66. Of course, system 60 may include other components as well, or as alternatives to what is shown in FIG. 3. Although system 10 is depicted as mounted atop tower 62, system 10 may be disposed elsewhere providing that radiation 12 emanating from the microwave antenna system 22 covers the region whose presence is to be communicated to birds 14, to avoid avian impact. Those skilled in the relevant art will appreciate that operating voltage for system 10 may be obtained by stepping-down voltages present in lines 64.

[00057] Although FIG. 3 shows system 10 as protecting a power distribution system 60, tower 62 could in fact represent some other object whose presence is to be communicated to oncoming avians. As such, protected object 62 could represent a skyscraper, a tall monument, among other objects.

[00058] Again, flying birds or bats 14 will be warned of the presence of the protected portion of system 60 by the pulsed microwave energy 12 and/or secondary emitters 32. As was described with respect to FIG. 2, the likelihood of avian contact is reduced in a benign manner, preferably transparently to humans in the area.

[00059] FIG. 4 shows another application of the present invention 10, whose components are mounted within an aircraft 70. In flight, system 10 emanates pulsed microwave radiation 12 that is intended to attract the attention of birds and the like 14 to the aircraft, whereupon the birds will veer off course. Possibly system 10 can share existing microwave antenna and other system facilities already present in aircraft 70. Alternatively, system 10 can include its own microwave antenna system 22 as shown in FIG. 1. It is understood that FIG. 4, like FIGS. 2 and 3, is not drawn to scale.



[00060] In military aircraft applications, the use of a hazard proximity detector 28 may advantageously permit aircraft 70 to fly over terrain without needlessly emanating radiation 12 until actually required to warn and/or deter targets 14. In this fashion, the presence of aircraft 70 is less likely to be detected by hostile aircraft or ground forces monitoring for pulsed microwave radiation frequencies in the 1.0 GHz to 40 GHz range.

[00061] Normally, system 10 emits a benign density of radiation 12 that complies with environmental safety standards. In contrast to such benign use, for an aircraft protection system such as shown in FIG. 4, it may be feasible to use substantially larger magnitudes of pulsed microwave radiation that intentionally damages one or more organs in the birds. Such damage may in fact impair the birds' ability to orient themselves and to continue flying. In this fashion, the safety of aircraft 70 and all on board could be ensured.

[00062] Other features and advantages of the invention will appear from the following description in which the preferred embodiments have been set forth in detail, in conjunction with the accompanying drawings. For example, although the preferred embodiments use fixed-width pulses, those skilled in the art will recognize that pulses of variable width may be used as well. Modifications and variations may be made to the disclosed embodiments without departing from the subject and spirit of the invention as defined by the following claims.

(57) **What is claimed is:**

1. A method for benignly communicating the presence of an object to a flying vertebrate, the method comprising the following steps:

propagating a region at least partially surrounding said object with pulses of microwave energy having an average power level of about 1 mw/cm.sup.2, wherein said pulses of microwave energy are selected to elicit a warning signal within said flying vertebrate's auditory system without physically harming said flying vertebrate.

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## Chick embryo development can be irreversibly altered by early exposure to weak extremely-low-frequency magnetic fields.

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### Abstract

Several reports have shown that weak, extremely-low-frequency (ELF), pulsed magnetic fields (PMFs) can adversely affect the early embryonic development of the chick. In this study, freshly fertilized chicken eggs were exposed during the first 48 h of postlaying incubation to PMFs with 100 Hz repetition rate, 1.0 microT peak-to-peak amplitude, and 500 microseconds pulse duration. Two different pulse waveforms were used, having rise and fall times of 85 microseconds (PMF-A) or 2.1 microseconds (PMF-B). It has been reported that, with 2 day exposure, these fields significantly increase the proportion of developmental abnormalities. In the present study, following exposure, the eggs were allowed to incubate for an additional 9 days in the absence of the PMFs. The embryos were taken out of the eggs and studied blind. Each of the two PMF-exposed groups showed an excess in the percentage of developmental anomalies compared with the respective sham-exposed samples. This excess of anomalies was not significant for the PMF-A-treated embryos ( $P = 0.173$ ), whereas it was significant for the PMF-B-exposed group ( $P = 0.007$ ), which showed a particularly high rate of early embryonic death. These results reveal that PMFs can induce irreversible developmental alterations and confirm that the pulse waveform can be a determinant factor in the embryonic response to ELF magnetic fields. The data also validate previous work based on the study of PMFs' effects at day 2 of embryonic development under field exposure.

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## **Pulse shape of magnetic fields influences chick embryogenesis\***

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### INTRODUCTION

Recent studies have demonstrated that biological systems are sensitive to electromagnetic fields (EMF), as shown by changes in growth rate of bacteria (Blackman, Bennane, Weil & Ali, 1975), eucaryote (Adey, 1981; Grundler, Keilmann & Fröhlich, 1977) and tumoural cell cultures (Kim, 1976; Rockwell, 1977), by mutagenic effects (Diebolt, 1978), and by metabolic alterations (Weissbluth, 1979). Most of these studies have been performed using static electromagnetic fields of high intensity or pulsating fields at frequencies above MHz, and results have been contradictory.

Cerebral calcium ion binding (Bawin & Adey, 1976; Bawin, Kaczmarek & Adey, 1975; Bawin, Adey & Sabbot, 1978) and osteogenesis (Bassett, Pawluk & Pilla, 1974*a, b*; Bassett, Chiokshi, Hernandez & Pawluk, 1979) are influenced by pulsating fields of very low frequencies, demonstrating 'window' effects at specific frequencies and intensities. Slight modifications of electromagnetic field parameters may have opposite effects; for example, frog erythrocyte dedifferentiation in Ringer solution is inhibited by exposure to 40 and 71 Hz electromagnetic fields, and accelerated with a frequency of 55 Hz (Hinsenkamp, Chiabrera, Pilla & Bassett, 1978).

The waveform appears to be an important factor determining biological responses. When exposed to sinusoidal electrical fields, chick cerebral tissues released a varied amount of preincorporated  $^{45}\text{Ca}^{2+}$  according to the frequency and intensity of the applied fields. A sinusoidally modulated amplitude caused progressive increase in  $^{45}\text{Ca}^{2+}$  efflux at modulation frequencies from 6 to 16 Hz, whereas 0.5 and 3.0 Hz did not change this efflux (Bawin *et al.* 1975).

There are few studies on the effects of electromagnetic fields on embryonic development. Levengood (1967) reported that an electromagnetic field of 150 Gauss induced retardation of development in *Drosophila*, and Mulay & Mulay (1964) found that exposure to 100, 600, and 1500 Oe had no effect, although exposure to 3000-4000 Oe for more than one generation resulted in increased abnormalities. This report, however, was not confirmed by Beischer (1964) or by Steen & Ofted (1967).

Studying the effects of electromagnetic fields on amphibian development, Iwasaki, Ohara, Matsumoto & Matsudaira, (1978) reported that the exposure of fertilized or early cleavage eggs of *Xenopus laevis* to a uniform electromagnetic field of 5000 Gauss for up to 72 hours did not affect development or hatchability, while higher intensities of 10000 or more than 17000 Gauss induce pathological disturbances in frog and salamander embryos (Levengood, 1969; Neurath, 1968).

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Veneziano (1965) demonstrated the sensitivity of chick embryos to uniform magnetostatic fields ranging from 1.1 to 31 Gauss. The exposed embryos present a mean blastoderm diameter smaller than in controls, as well as developmental anomalies. Exposure to the strongest electromagnetic field induces central nervous system defects in 9% of the embryos, including hyperplasia and general growth retardation. Histological examination reveals cellular disorganization and extra-neural tube formation.

Significant effects on chick development have also been described by Joshi, Khan & Damle (1978) in 25 embryos which were exposed, *in vitro* and at room temperature, to a vertical homogeneous magnetic field of 5000 Oe for 1 hour and then returned to the incubator for 24 hours. All embryos show abnormalities in the nervous system, heart, and somites, and generally retarded development, as well as an increased number of dividing cells in the neuroepithelium.

In a previous paper (Delgado, Leal, Monteagudo & Garcia Gracia, 1982), we described the effects on chick embryos of low frequency electromagnetic fields of 10, 100 and 1000 Hz with intensities of 0.12, 1.2 and 12 microTeslas, ( $\mu\text{T}$ ) showing the different sensitivity of developing organs to specific frequencies and intensities.

In the present study these investigations have been continued, paying special attention to the differential embryological effects of pulse shape.

#### MATERIALS AND METHODS

A total of 659 freshly fertilized White Leghorn hen eggs was used (295 exposed to electromagnetic fields and 364 unexposed controls). Pairs of eggs were placed inside cylindrical coils 170 mm in length and 68.6 mm in diameter, each made of 1000 turns of 0.33 mm enamelled copper wire. Eggs and coils were kept at 38 °C for 48 hours inside Memmert incubators. Additional experiments were performed placing groups of 12 eggs between two square Helmholtz coils 30 cm long, 20 cm apart, and made of 30 turns of 1.5 mm enamelled copper wire. These eggs and coils were also placed in Memmert incubators.

Electrical current to produce electromagnetic fields in the coils was provided by an SD-9 or S-44 Grass stimulator set to deliver biphasic stimulation with zero net value. Uniformity and strength of the electromagnetic field were tested with a Gaussmeter model 750 AR with accessory 1D75 (RFL Industries, Boonton, N.Y). The electromagnetic field did not vary more than 2% in different coils in series, and when calculated for 1  $\mu\text{T}$ , the energy changes due to eddy currents were less than  $3.3 \times 10^{-16}$  watts. Since the eggs were stationary, they did not cause spurious electrical fields.

Electrical current parameters used to activate the coils were monitored by a Tektronic 5113 oscilloscope. Repetition rate was always 100 Hz and pulse duration was 500  $\mu\text{sec}$ . As explained later, electromagnetic field intensity varied between 0.4 and 104  $\mu\text{T}$ . The following types of pulse shape were tested:

##### *Pulse A*

Rise and fall times were 100  $\mu\text{sec}$  with a declining plateau, with some ripples in the curve, and a long duration post-pulse negative amplitude, as shown in Figure 1 A. The pulse was generated by a Grass SD-9 stimulator feeding five cylindrical coils connected in series, permitting simultaneous experiments with five pairs of eggs.

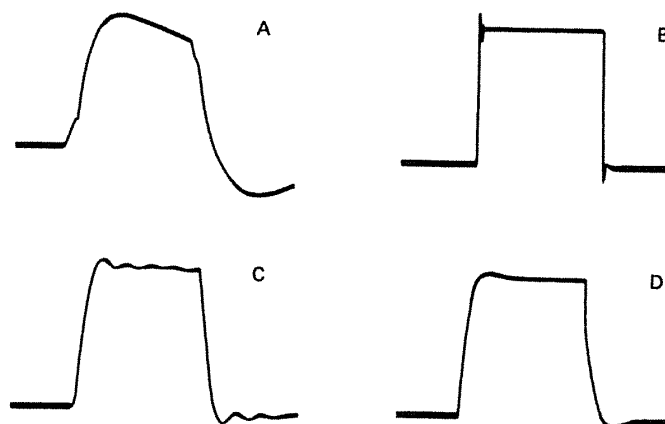


Fig. 1. Pulse signals: In all experiments, pulse duration ( $500 \mu\text{sec}$ ) and repetition rate ( $100 \text{ Hz}$ ) were constant. Four different pulse shapes were used: (A) rise time  $100 \mu\text{sec}$ ; (B) rise time  $2 \mu\text{sec}$ ; (C) rise time  $42 \mu\text{sec}$ ; (D) rise time  $42 \mu\text{sec}$ .

#### *Pulse B*

Rise and fall times were  $2 \mu\text{sec}$ , as shown in Figure 1 B. The pulse was generated by a Grass SD-9 stimulator feeding two square Helmholtz coils in parallel. The low resistance permitted rapid transit of current with great uniformity of pulses.

#### *Pulse C*

Rise and fall times were  $42 \mu\text{sec}$  with several ripples in the curve (Fig. 1 C). The pulse was generated by a Grass S-44 stimulator feeding five cylindrical coils as in Pulse A.

#### *Pulse D*

This pulse was similar to pulse C but without ripples. It was generated by a Grass S-44 stimulator feeding only two cylindrical coils (Fig. 1 D).

#### *Study of embryos*

After 48 hours of incubation the eggs were opened and embryos were immersed in Tyrode solution and then fixed in Carnoy's solution (60% absolute ethanol, 30% chloroform, 10% glacial acetic acid). Gross examination of embryos was performed at  $\times 30$  magnification with a Nikon binocular stereomicroscope, and pictures were taken with a photographic attachment. The developmental stage of each embryo was determined according to the Hamburger & Hamilton (1951) scale. The cephalic nervous system, truncal nervous system, heart, extra-embryonic vascularisation, and somites were studied in detail for possible abnormalities.

Histological examination of the embryos was performed by dehydrating samples through an alcohol series and embedding them in paraffin. Transverse sections  $7 \mu\text{m}$  thick were cut serially using a rotary Leitz microtome. Sections were mounted, stained with periodic acid-Schiff (Luna, 1968) and alcian blue at pH 2.5 (Gabe, 1968), and counterstained with haematoxylin. Preparations were studied with a Nikon Apophot microscope. Photographs were taken with a 35 mm Nikon camera.

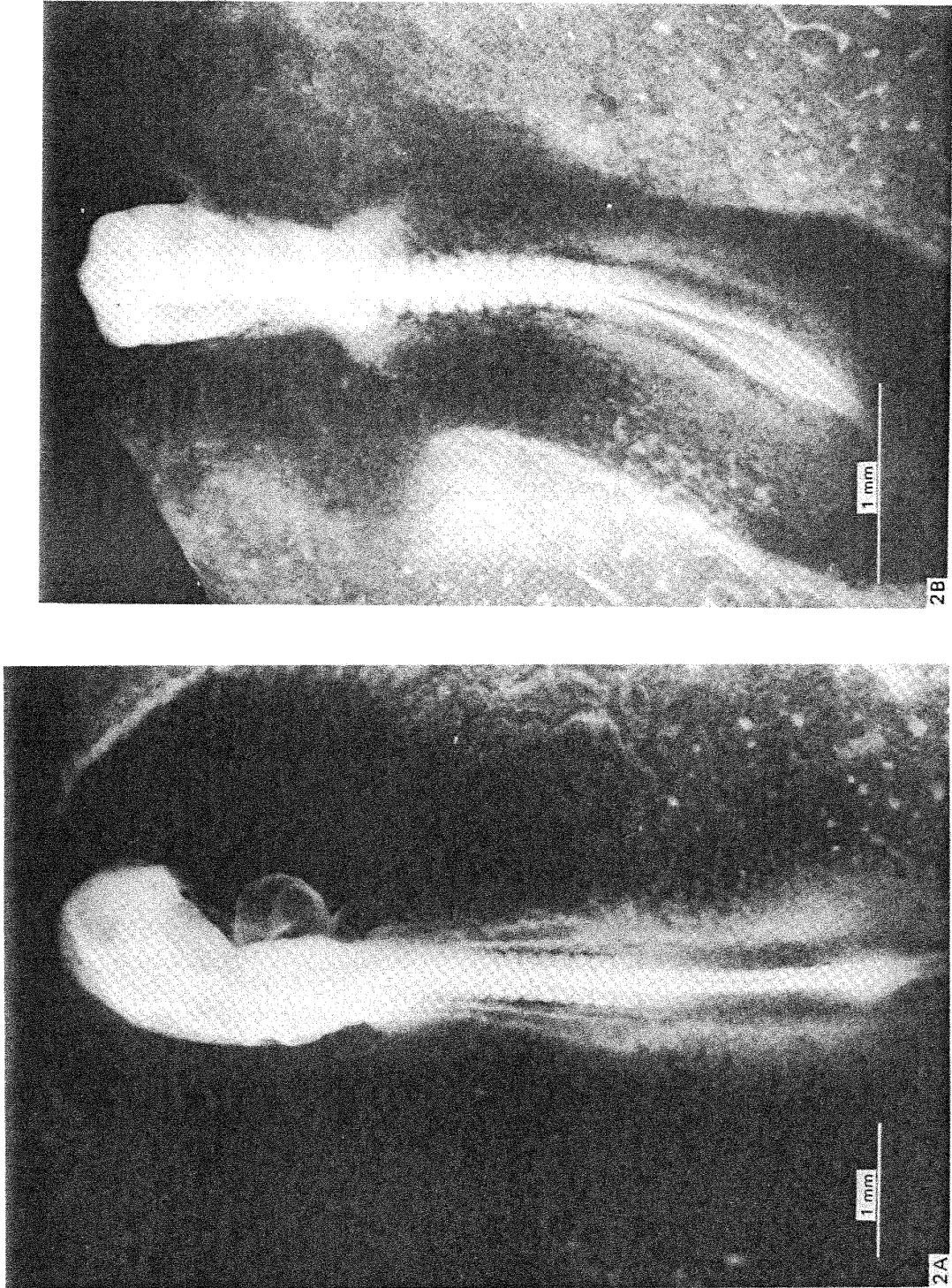


Fig. 2. (A) Stage 12 control embryo.  $\times 20$ . (B) Stage 10 embryo exposed to pulse A,  $1.0 \mu T$ , showing abnormal truncal torsion.  $\times 30$ .

## RESULTS

Observation of each embryo with the stereomicroscope permitted classification of its stage of development according to the Hamburger–Hamilton scale. Stage 11 was characterised as follows: cephalic torsion to the right was clearly visible and the cephalic nervous system showed five primitive regions. The division of the prosencephalon into telencephalon and diencephalon had begun, and the rhombencephalon was segmented into 6–7 monomeric regions, the first to become the metencephalon and the others to become the myelencephalon. At this stage, the anterior neuropore was closed and the optic vesicles were constricted at the base. The auditory pits were wide open. The heart was located at the right of the embryo and the extra-embryonic vessels were well developed. There were 13–15 somite pairs.

At the end of stage 12 (Fig. 2A), cephalic flexion was almost complete and torsion was detectable as far as the fifth or sixth somite pair. The five cerebral vesicles were clearly differentiated and there were 19 somite pairs. The heart, in 'S' shape, was beating and primitive blood was circulating between the embryo and the extra-embryonic zone. Extra-embryonic vascularisation was well developed; the two anterior vitelline veins were visible as well as the sinus vein at the base of the heart, formed by fusion of the two omphalomesenteric veins.

In our study, histological examination of the embryos was concentrated on the five systems (cephalic and truncal nervous systems, heart, extra-embryonic vascularisation and somites).

*Classification of embryos*

According to the Hamburger–Hamilton scale, normal embryos should reach stage 12 after 48 hours of incubation. We therefore placed the lower limit of normality at stage 9 which corresponds to about threequarters of the total development time. Embryos which had reached at least stage 9 and exhibited normal morphological and histological characteristics in the five systems were classified as *normal*. Embryos which had not reached stage 9, representing a delay of at least 11 hours in their development, or those which presented morphological and/or histological alterations in any of the five systems, were classified as *abnormal*. In this group, sub-embryos which had not reached stage 4 (the definitive primitive streak stage) were classified as 'non-developed'.

Spontaneous abnormalities varied among the lots of eggs, as demonstrated by comparing the controls in different experiments. Therefore, the significance of experimental results was established by evaluating the simultaneously incubated exposed and control embryos of the same lot. As the control samples had a high frequency of spontaneous abnormalities, they provided a basis for evaluation of detrimental, neutral, or beneficial effects of applied electromagnetic fields.

*Null effects of presence of coils on embryonic development*

Exposure to electromagnetic fields required the placement of eggs within the coils. Even in the absence of an electromagnetic field, this procedure might modify the uniform temperature in the incubator, constituted a shield for the egg, or introduced unknown elements disturbing embryonic development.

To evaluate these possibilities, 39 eggs were incubated inside cylindrical coils with no electromagnetic field and 43 eggs were simultaneously incubated outside the coils.

Table 1. *Development of embryos lying inside and outside coils in the absence of electromagnetic fields*

Embryos	Inside coils (N = 39)	Outside coils (N = 43)
<b>Normals</b>		
No.	30	34
%	76.9	79.0
Mean stage	11.8	11.4
<b>Abnormals</b>		
No.	9	9
% (1)	23.0	20.9
Mean stage	7.7	9.1
<b>Non-developed</b>		
No.	2	3
%	5.1	6.9
Abnormality ratio (1) (% inside/% outside)		$\frac{1.10}{(P \approx 0.75)}$

N, total number of embryos.

Table 2. *Abnormalities of systems of embryos in Table 1*

Systems	Inside coils (N = 39)	Outside coils (N = 43)
<b>CNS</b>		
Abnormals	8	8
%	20.5	18.6
Ratio		$\frac{1.10}{(P \approx 0.75)}$
<b>TNS</b>		
Abnormals	7	6
%	17.9	13.9
Ratio		$\frac{1.28}{(0.75 > P > 0.50)}$
<b>Heart</b>		
Abnormals	6	6
%	15.3	13.9
Ratio		$\frac{1.10}{(P \approx 0.75)}$
<b>Vessels</b>		
Abnormals	6	7
%	15.3	16.2
Ratio		$\frac{0.94}{(P \approx 0.90)}$
<b>Somites</b>		
Abnormals	6	7
%	15.3	16.2
Ratio		$\frac{0.94}{(P \approx 0.90)}$

CNS, cephalic nervous system; TNS, truncal nervous system; N, total number of embryos.



Table 3. Normal and abnormal embryos in controls and after exposure to electromagnetic fields, pulse A, at indicated intensities

Total no. of embryos (N)	0.4 μT		1.0 μT		10.4 μT		13.9 μT		104 μT	
	Exposed	Controls	Exposed	Controls	Exposed	Controls	Exposed	Controls	Exposed	Controls
Normal	21	25	32	42	27	29	15	21	42	40
No.	70.0	67.5	58.1	76.3	65.8	70.7	46.8	65.6	71.1	75.4
%	12.3	12.4	13.0	12.9	11.3	11.3	10.5	11.5	11.6	11.6
Mean stage										
Abnormals	9	12	23	13	14	12	17	11	17	13
No.	30.0	32.4	41.8	23.6	34.1	29.2	53.1	34.3	28.8	24.5
% (1)	10.8	10.5	12.0	10.5	9.4	9.7	8.4	8.1	8.2	8.8
Mean stage										
Non-developed	1	4	2	2	1	1	5	1	3	4
No.	3.3	10.8	3.6	3.6	2.4	2.4	15.6	3.1	5.0	7.5
%										
Abnormality ratio (1)										
% Exposed/% controls	0.92		1.77		1.16		1.54		1.17	
	(P ≈ 1.0)		(0.10 > P > 0.05)		(0.90 > P > 0.75)		(0.25 > P > 0.10)		(P ≈ 1.0)	

N, total number of embryos.

Table 4. Abnormalities in systems of embryos in Table 3

Systems	0.4 μT		1.0 μT		10.4 μT		13.9 μT		104 μT	
	Exposed (N = 30)	Controls (N = 37)	Exposed (N = 55)	Controls (N = 55)	Exposed (N = 41)	Controls (N = 41)	Exposed (N = 32)	Controls (N = 32)	Exposed (N = 59)	Controls (N = 53)
<b>CNS</b>										
Abnormals	7	9	16	12	14	11	16	11	17	13
%	23.3	24.3	29.0	21.8	34.1	26.8	50.0	34.3	28.8	24.5
Ratio	0.95 (0.90 > P > 0.75)		1.33 (0.90 > P > 0.75)		1.27 (0.75 > P > 0.50)		1.45 (0.50 > P > 0.25)		1.17 (0.90 > P > 0.75)	
<b>TNS</b>										
Abnormals	7	12	14	8	11	9	15	11	16	11
%	23.3	32.4	25.4	14.5	26.8	21.9	46.8	34.3	27.1	20.7
Ratio	0.71 (0.75 > P > 0.50)		1.75 (0.10 > P > 0.05)		1.22 (P ≈ 0.75)		1.36 (0.50 > P > 0.25)		1.30 (0.75 > P > 0.50)	
<b>Heart</b>										
Abnormals	4	8	9	9	11	10	17	9	14	13
%	13.3	21.6	16.3	16.3	26.8	24.3	53.1	28.1	23.7	24.5
Ratio	0.61 (P ≈ 0.50)		1.00 (P = 1)		1.10 (P ≈ 1)		1.88 (0.10 > P > 0.05)		0.96 (P = 0.90)	
<b>Vessels</b>										
Abnormals	3	7	14	9	11	9	17	9	14	10
%	10.0	18.9	25.4	16.3	26.8	21.9	53.1	28.1	23.7	18.8
Ratio	0.52 (0.50 > P > 0.25)		1.55 (0.50 > P > 0.25)		1.22 (P ≈ 0.75)		1.88 (0.10 > P > 0.05)		1.26 (P ≈ 0.90)	
<b>Somites</b>										
Abnormals	4	9	9	10	10	10	16	11	14	11
%	13.3	24.3	16.3	18.1	24.3	24.3	50.0	34.3	23.7	20.7
Ratio	0.54 (0.50 > P > 0.25)		0.90 (P ≈ 1)		1.00 (P = 1)		1.45 (0.50 > P > 0.25)		1.14 (0.90 > P > 0.75)	

CNS, cephalic nervous system; TNS, truncal nervous system; N, number of embryos.

Table 5. Embryos exposed to pulse A, 1.0  $\mu$ T: detail of six different experiments

Experiment	1		2		3		4		5		6		Total	
	Exp	C	Exp	C	Exp	C	Exp	C	Exp	C	Exp	C	Exp	C
No. of embryos	10	9	9	10	10	8	9	9	8	10	9	9	55	55
Normals														
No.	2	4	7	9	7	8	6	7	3	6	7	8	32	42
%	20.0	44.4	77.7	90.0	70.0	100	66.6	77.7	37.5	60.0	77.7	88.8	58.1	76.3
Abnormals														
No.	8	5	2	1	3	0	3	2	5	4	2	1	23	13
% (1)	80.0	55.5	22.2	10.0	30.0	—	33.3	22.2	62.5	40.0	22.2	11.1	41.8	23.6
Abnormality ratio (1) (% Exposed/ % controls)	1.44		2.22		—		1.50		1.56		2.00		1.77	(0.10 > P > 0.05)

Exp, exposed embryos; C, controls.

Table 6. *Effect of pulse shape at the indicated intensities ( $\mu T$ )*

Pulse shape Intensity	B 0.4 $\mu T$		C 1.0 $\mu T$		D 1.0 $\mu T$	
	Exposed	Controls	Exposed	Controls	Exposed	Controls
Total no. of embryos	24	23	30	29	24	12
Normals						
No.	4	15	19	22	7	10
%	16.6	62.2	63.3	75.8	29.1	83.3
Mean stage	11.7	10.4	12.3	12.5	10.7	11.9
Abnormals						
No.	20	8	11	7	17	2
% (1)	83.3	34.7	36.6	24.1	70.8	16.6
Mean stage	8.8	7.9	11.7	11.7	8.7	Non developed
Non-developed						
No.	4	2	2	0	5	2
%	16.6	8.6	6.6	—	20.8	16.6
Abnormality ratio (1) (% exposed/% controls)	2.40 (0.005 > P)		1.51 (0.50 > P > 0.25)		4.26 (0.01 > P > 0.005)	

As shown in Table 1, frequencies of abnormal (23 and 20.9% inside and outside the coils, respectively) and non-developed (5.1 and 6.9%) embryos were similar in eggs placed inside and outside the coils. The abnormality ratio (the percentage of faults inside expressed in relation to the percentage outside the coils) was 1.10, which indicated that incubation of the eggs inside the coils produced no significant differences. The mean developmental stage reached by normal embryos was also similar when eggs were placed inside and outside the coils (stages 11.8 and 11.4 respectively). The only difference was in the mean developmental stage of abnormal embryos which was slightly earlier in eggs placed inside the coils (stage 7.7) as compared with eggs placed outside the coils (stage 9.1). This difference corresponded to approximately 3 hours of development and was not statistically significant because of the spontaneous variability of abnormal embryos in controls (Tables 3-6).

Analysis of abnormalities present in the five systems (Table 2) revealed no differences between embryos developed inside or outside the inactivated coils. It was therefore concluded that in the absence of an electromagnetic field, placement of eggs inside the coils caused no significant disturbance of early development.

#### *Embryological effects of electromagnetic fields with pulse shape A*

With pulse A (Fig. 1) and constant parameters of 100 Hz, 500  $\mu$ sec pulse duration experiments were performed on 435 eggs (217 exposed and 218 controls) to test the effects of electromagnetic fields with intensities of 0.4, 1.0, 10.4, 13.9, and 104  $\mu T$ . Data on the general development of embryos are shown in Table 3. Analysis of disturbances in each system is presented in Table 4 which also indicates for each system the ratio of alterations in exposed compared with control embryos. For example, with 0.4  $\mu T$  intensity, the total number of exposed embryos was 30, and seven (23.3%) had cephalic nervous system abnormalities. The total number of control embryos in this group was 37, and nine (24.3%) had cephalic nervous system abnormalities. Therefore, the ratio of these abnormalities at 0.4  $\mu T$  was 0.95 (23.3% in exposed: 24.3% in controls) which was not significant. Calculations were made in the same way for

abnormalities in each system. A ratio above 1.0 suggested that electromagnetic field effects were teratogenic, while a ratio below 1.0 suggested 'beneficial' effects.

#### *Exposure to 0.4 $\mu$ T*

A total of 30 eggs was exposed to an electromagnetic field of 0.4  $\mu$ T intensity and compared with 37 controls. In both groups, about 70% of the embryos were normal, developing just beyond stage 12 (Table 3). Abnormal embryos in exposed and control groups (30 and 32.4% respectively) had a ratio (0.92) which was not significant, and both groups developed to between stages 10 and 11.

Although the analysis of abnormalities in the five systems showed no significant differences (Table 4), there was a beneficial 'trend' in exposed embryos, which had ratios below 1.0 for all developing systems. Possible beneficial effects of the 0.4  $\mu$ T electromagnetic field were also suggested by the respective frequencies of non-developed embryos in the exposed (3.3%) and control (10.8%) groups (Table 3).

#### *Exposure to 1.0 $\mu$ T*

A total of 55 eggs was exposed to an electromagnetic field of 1.0  $\mu$ T in six experiments, with 55 controls. As shown in Table 3, 41.8% of the exposed embryos were abnormal, compared with 23.6% of the controls, the ratio being 1.77 ( $0.1 > P > 0.05$ ). As shown in Table 5, the number of abnormal embryos was always greater in the exposed than in the control group, indicating a slight but significant teratogenic effect.

Normal embryos reached a similar mean stage of development in exposed eggs (stage 13) and in controls (stage 12.9), while abnormal exposed embryos reached a more advanced stage of development (stage 12.0) than did controls (stage 10.5), this advance corresponding to approximately 12 hours of development (Table 3). A possible explanation might be that spontaneous abnormalities affected general development, while induced abnormalities appeared to be more system-specific, indicating the greater sensitivity of some systems to electromagnetic fields. Abnormalities in the truncal nervous system were more frequent in exposed than in control embryos, whereas cephalic nervous system, heart, vessel, and somite abnormalities were not significantly different (Table 4). Gross examination of the embryos showed that malformations of the neural tube in exposed embryos presented two types of disturbances not found in controls: abnormal torsion of the axis (six cases; Fig. 2B), and abnormally dense folds (five cases). In addition, short neural tubes were less common in abnormal exposed embryos (three out of 14 cases) than in abnormal controls (seven out of eight). An open neural tube was found as often in exposed as in control embryos.

Histological analysis of the abnormal exposed embryos showed a general dorso-ventral flattening of the organs. Compared with controls of the same stage (Fig. 3A), truncal neural tubes were thick and poorly organised, somites were slightly retarded, and alcian blue-stained components were less abundant in the basal membranes of the neuroepithelium, ectoderm, notochordal sheath, and around the somites (Fig. 3B).

#### *Exposure to 10.4 $\mu$ T*

A total of 41 embryos was exposed to an electromagnetic field of 10.4  $\mu$ T, and development was compared to the same number of controls. The frequency of abnormal embryos was similar (close to 30%) in both groups with no differences in the developmental stage (Table 3). Ratios of abnormalities in the different systems

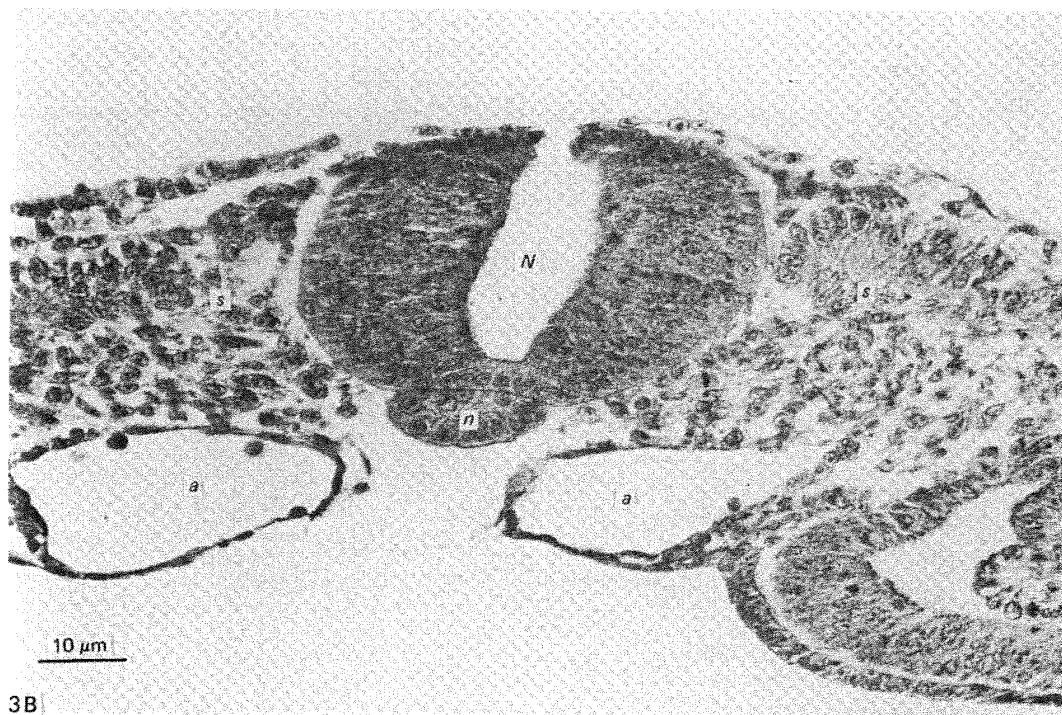
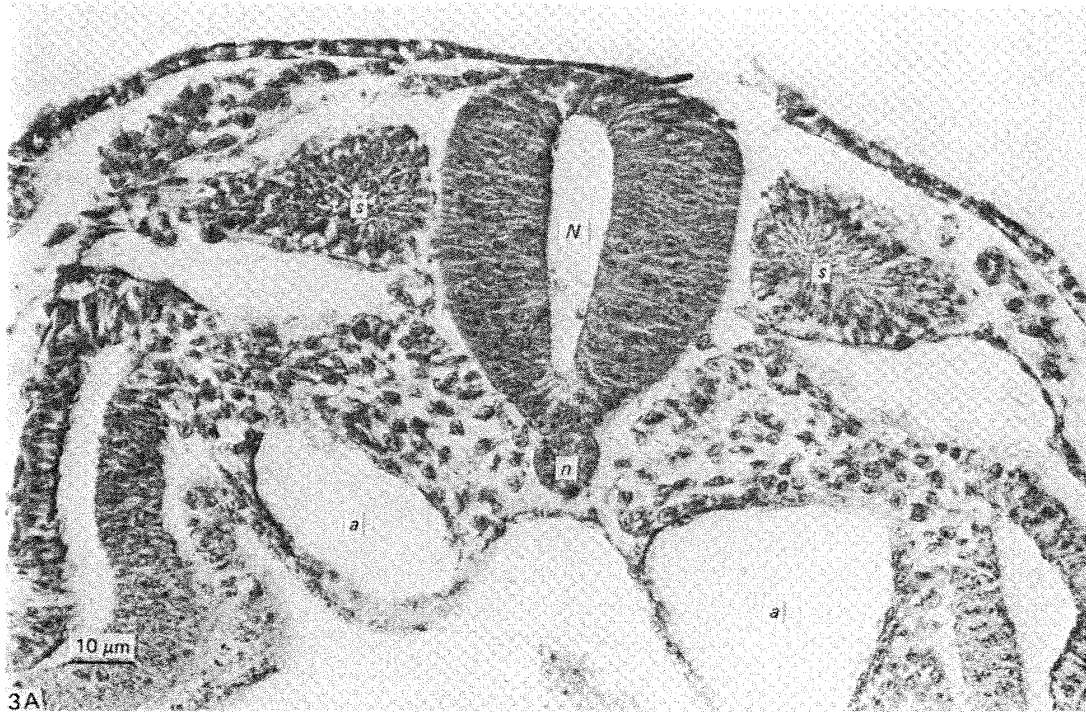
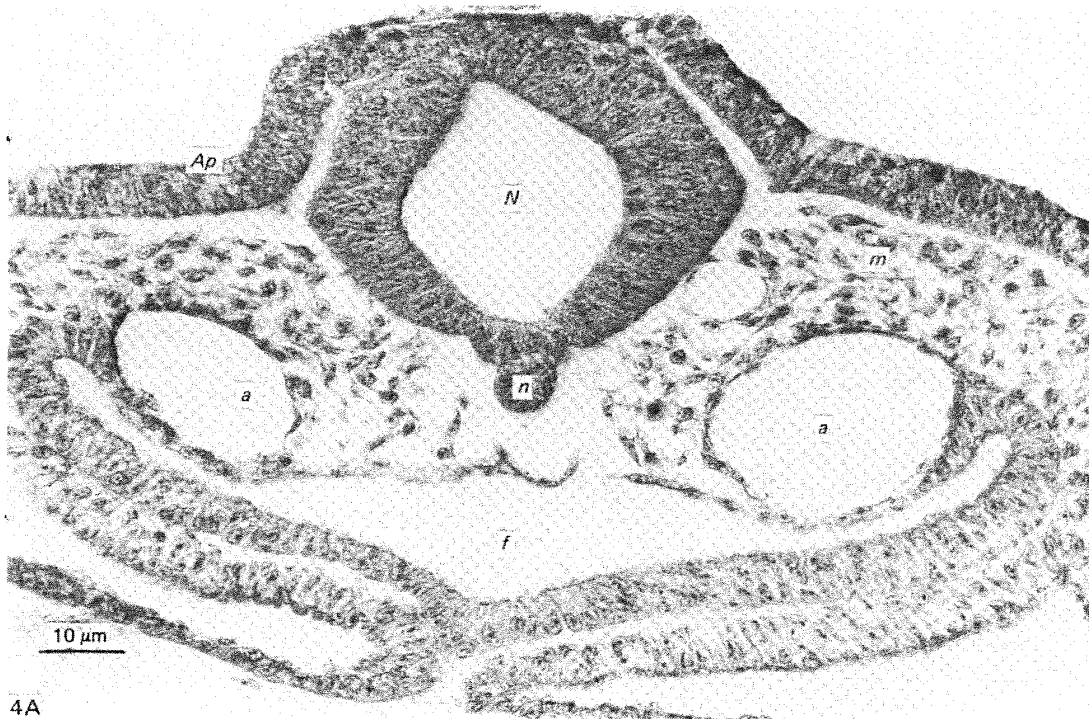
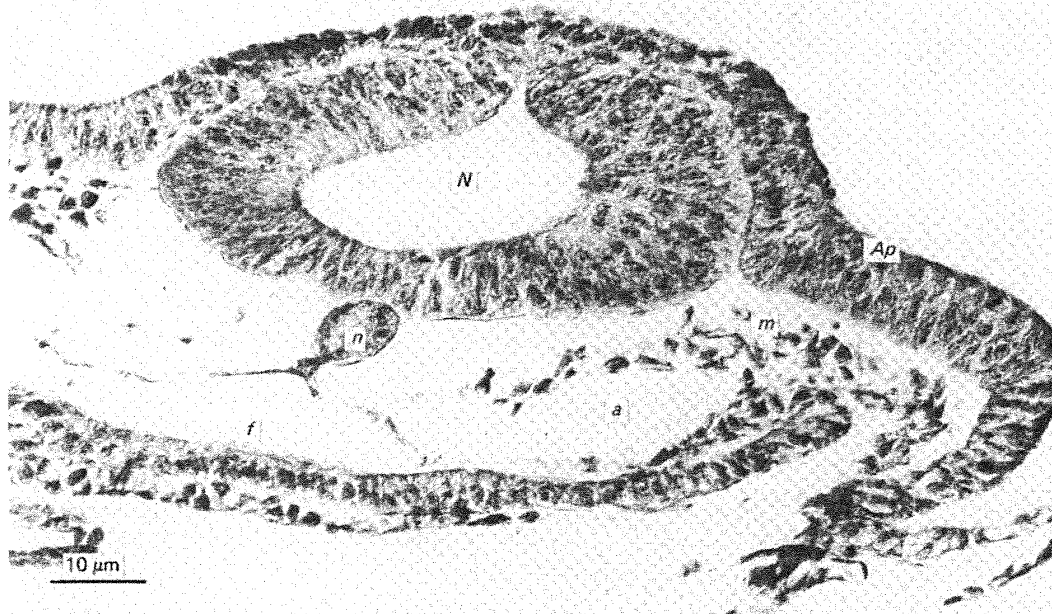


Fig. 3. Transverse sections through the third somite pair. (A) Stage 12 control embryo. (B) Embryo exposed to pulse A, 1.0  $\mu$ T. There is dorsoventral flattening of all organs and a thick, poorly organised truncal nervous system. *N*, neural tube; *a*, aorta; *s*, somite; *n*, notochord.  $\times 800$ .



4A



4B

Fig. 4. Transverse sections at the auditory pit level in stage 12 embryos. (A) Control. (B) Exposed to pulse A, 13.9  $\mu$ T. There is dorsoventral flattening of all organs and poorly developed vessels, foregut, and mesenchymal tissue. *N*, neural tube; *Ap*, auditory pit; *a*, aorta; *n*, notochord; *m*, mesenchyme; *f*, foregut.  $\times 800$ .

(Table 4) were between 1.27 for the cephalic nervous system and 1.00 for somites. It was concluded that this intensity had no notable effect on embryonic development.

#### *Exposure to 13.9 $\mu$ T*

In this group there were 32 exposed and 32 control eggs. As shown in Table 3, there was an increase in the number of abnormal embryos in the exposed group (53.1%) as compared to controls (34.3%), but the ratio, 1.54, ( $0.25 > P > 0.1$ ) was not significant.

In exposed eggs, the frequency of non-developed embryos (15.6%) was five times higher than in controls (3.1%). Furthermore, the developmental stage reached by normal embryos was slightly delayed in the exposed sample (stage 10.5) when compared with controls (mean stage 11.5), corresponding to a difference of seven to eight hours. Heart and vessel abnormalities were significantly more frequent in exposed than in control embryos, with a ratio of 1.88 ( $0.10 > P > 0.05$ ).

Microscopic examination of the 17 exposed embryos with heart and vessel abnormalities revealed that, in 14 cases, the heart was absent and vascularisation had not developed. Of these 14 cases, seven had not yet reached stage 8 (i.e. the stage of normal initiation of heart and vascular system organogenesis). In the other seven cases, however, embryos had reached beyond stage 8 and showed undeveloped heart and extra-embryonic vascularisation. The intra-embryonic vessels had poorly organised walls. One example is shown in Figure 4 where a transverse section at the auditory pit level in a stage 12 normal embryo (Fig. 4A) may be compared with a similar section of an exposed abnormal embryo of the same stage (Fig. 4B). These sections reveal a lack of endothelial organisation in the dorsal aortae and an underdeveloped foregut.

In another three embryos, the heart was displaced and abnormally twisted, with large blood islands but no vessels. In all these abnormal embryos, transverse sections showed a dorsoventral flattening of the organs (Fig. 4B) as previously observed in embryos exposed to an electromagnetic field of 1.0  $\mu$ T intensity.

#### *Exposure to 104 $\mu$ T*

The highest electromagnetic field intensity used (104  $\mu$ T) was tested on 59 embryos and compared with 53 controls. The frequency of abnormal specimens was similar in the exposed (28.8%) and control (24.5%) groups (Table 3). Analysis of abnormalities in the different developing systems (Table 4) revealed no significant disturbances, contrasting with the effects of intensities seven times (13.9  $\mu$ T) and one hundred times (1.0  $\mu$ T) weaker.

#### *Effects of pulse shape changes*

All the experiments described above were performed with pulse shape A (Fig. 1), and in order to evaluate possible effects related to the use of different pulse shapes, three other types of pulse were tested.

#### *Pulse B*

This pulse was tested with a field intensity of 0.4  $\mu$ T in 24 embryos and results were compared with 23 controls, as indicated in Tables 6 and 7. A strong teratogenic effect was demonstrated: while 34.7% of controls were abnormal, 83.3% of the exposed group presented abnormalities, giving a significant ratio of 2.40 ( $0.005 > P$ ).



Table 7. Abnormalities in systems of embryos of Table 6

Intensity	0.4 $\mu$ T		1.0 $\mu$ T		1.0 $\mu$ T	
	Exposed (N = 24)	Controls (N = 23)	Exposed (N = 30)	Controls (N = 29)	Exposed (N = 24)	Controls (N = 12)
Pulse shape	B		C		D	
CNS						
Abnormals	16	8	10	5	17	2
%	66.6	34.7	33.3	17.2	70.8	16.6
Ratio	1.91		1.93		4.26	
	(0.05 > P > 0.025)		(0.50 > P > 0.25)		(0.01 > P > 0.005)	
TNS						
Abnormals	16	8	8	3	17	2
%	66.6	34.7	26.6	10.3	70.8	16.6
Ratio	1.91		2.58		4.26	
	(0.05 > P > 0.025)		(0.25 > P > 0.10)		(0.01 > P > 0.005)	
Heart						
Abnormals	15	8	6	3	15	2
%	62.5	34.7	20.0	10.3	62.5	16.6
Ratio	1.80		1.94		3.76	
	(0.25 > P > 0.10)		(P $\approx$ 0.50)		(P $\approx$ 0.025)	
Vessels						
Abnormals	16	7	5	2	15	2
%	66.6	30.4	16.6	6.8	62.5	16.6
Ratio	2.19		2.44		3.76	
	(0.05 > P > 0.025)		(0.50 > P > 0.25)		(P $\approx$ 0.025)	
Somites						
Abnormals	17	7	9	3	15	2
%	70.8	30.4	30.0	10.3	62.5	16.6
Ratio	2.32		2.91		3.76	
	(0.01 > P > 0.05)		(0.25 > P > 0.10)		(P $\approx$ 0.025)	

CNS, cephalic nervous system; TNS, truncal nervous system; N, number of embryos.

The frequency of non-developed embryos was twice as great in the exposed (16.6%) as in the control (8.6%) eggs.

Disturbances in the five systems were twice as numerous in the exposed as in the control embryos. Asymmetry was observed in eight of the 16 cases of cephalic nervous system abnormality in exposed embryos, while it was detected in only two of the eight abnormal controls. Other characteristic disturbances, seen in seven of 16 cases, were short truncal nervous systems with large open folds, grossly under-developed somites and an abnormal torsion of the embryo (Fig. 5A) which was not observed in either normal or abnormal controls.

Transverse sections of the embryos (Fig. 5B) showed extensive necrotic cells in the cephalic neural ectoderm and ectoderm, absence of basal membrane around the tissues, and diminution of acid glycosaminoglycans in extracellular regions. The truncal neural ectoderm was not organised and somites appeared as small groups of cells.

Exposure to this type of electromagnetic field also changed the timing of embryonic development. While normal controls reached a mean stage of 10.4 and abnormal controls reached 7.9, the exposed embryos advanced further, normal embryos reaching stage 11.7 and abnormal 8.8. This advance represented a difference of 8-9 hours in the case of the normal embryos and indicated the complexity of electromagnetic field effects.

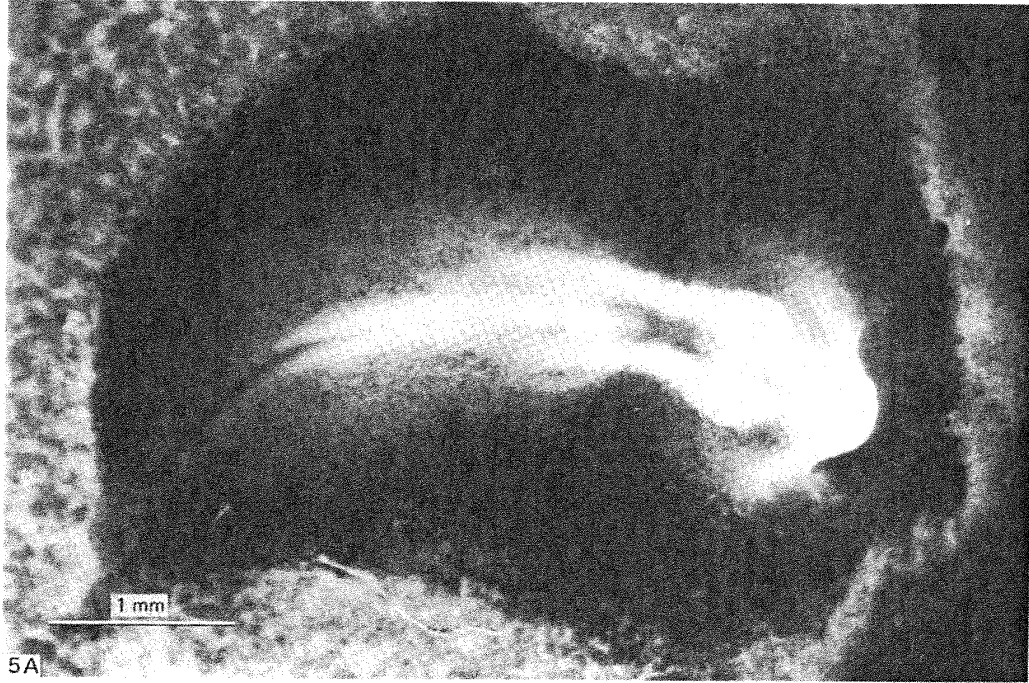
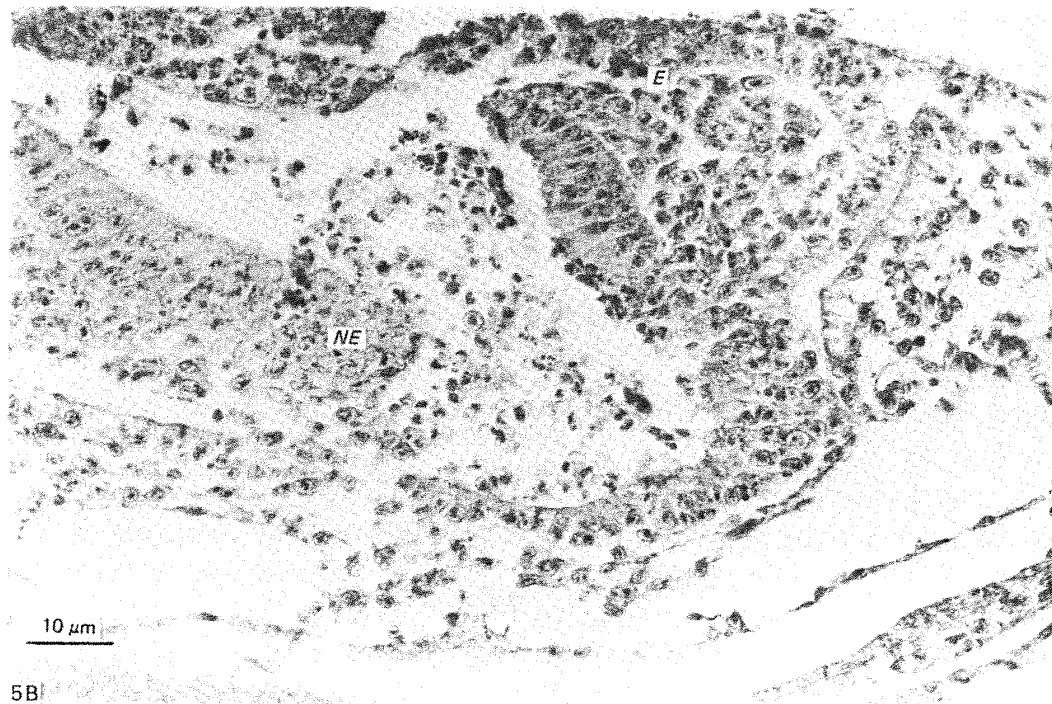


Fig. 5. Embryo exposed to pulse B, 0.4  $\mu$ T. (A) Gross morphology showing abnormal torsion of the neural tube with large open folds.  $\times 26$ . (B) Transverse section through the cephalic region showing extensive zones of necrotic cells in cephalic neural ectoderm (*NE*) and ectoderm (*E*).  $\times 800$ .



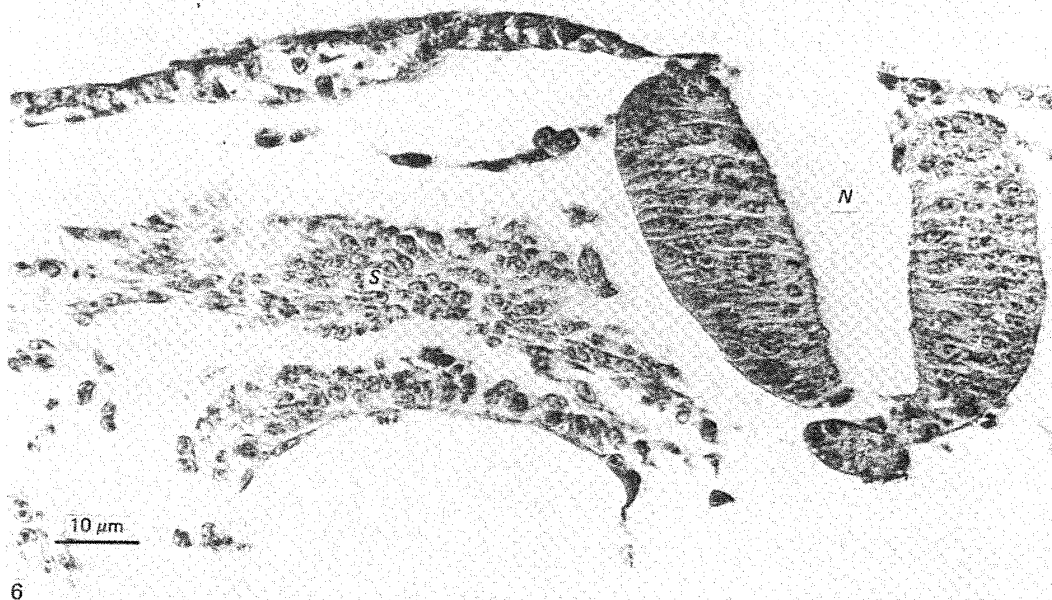


Fig. 6. Embryo exposed to pulse C, 1.0  $\mu$ T. Transverse section through the fourth somite pair, showing open neural folds (N), poorly organised somites (S), and a general loss of cohesion.  $\times 800$ .

### Pulse C

A total of 30 embryos was exposed to pulse C with 1.0  $\mu$ T and compared with 29 controls. Results, summarised in Table 6, indicated no significant increase in the frequency of abnormal embryos in the exposed group, with a ratio of 1.51 (0.50 >  $P$  > 0.25). The mean stage of development of normal embryos was similar in exposed and in controls (12.3 and 12.5), and the mean stage of abnormal embryos was the same (11.7) in both groups.

Abnormalities were different in exposed and control embryos. Controls were affected in only one or two developing systems, while exposed embryos had abnormalities in three or four systems. Thus, abnormalities in all five systems (Table 7) were twice as frequent in exposed as in control embryos, with a ratio of 1.93 for cephalic nervous system and 2.91 for the somites. Although these results did not reach statistical significance, there were specific disturbances in the exposed abnormal embryos: in the 10 cases of malformation of cephalic nervous system, cephalic torsion and flexion were absent although these embryos reached a mean stage of 11.7. In the eight cases of a malformed truncal nervous system, six showed open folds and abnormal torsion. This effect was similar to that observed in the embryos exposed to pulse B with an electromagnetic field of 0.4  $\mu$ T (Fig. 5A).

Transverse sections of the exposed abnormal embryos confirmed these morphological data, showing poorly organised somites. A loose cohesion of the organs and an increased tissue fragility were also noted (Fig. 6).

*Pulse D*

A total of 24 embryos was exposed to pulse D with a field intensity of  $1.0 \mu\text{T}$  and compared with 12 controls. Results (Tables 6, 7) demonstrated the most powerful teratogenic effects of the experiments, as shown by increased numbers of abnormal embryos, with a ratio of 4.26 in cephalic and truncal nervous systems, and 3.76 in heart, vessels, and somites. In the exposed group, abnormal embryos were very small, and the development of the normal embryos was delayed, reaching a mean stage of 10.7 instead of 11.9 in controls. This corresponded to a delay of about 10 hours in development, indicating that electromagnetic fields disturbed the development of all exposed embryos.

Histological studies confirmed the importance of alterations in the exposed embryos. They presented a disorganised appearance, with abnormal and poorly developed cephalic nervous systems and truncal nervous systems which remained open, poorly formed hearts, altered somites, and a lack of vessel development (Fig. 7). Acid glycosaminoglycans in basal membranes and extracellular matrix were drastically reduced.

*Electromagnetic field effects compared to pulse shapes*

A summary of the results of the embryonic effects of electromagnetic fields with intensities of  $0.4$  and  $1.0 \mu\text{T}$ , using pulses A, B and D, is shown in Figure 8. This illustrates the powerful effects of a field intensity of  $1.0 \mu\text{T}$  using pulse D on the development of the five systems, which was far greater than the effects of the same intensity with pulse A. Figure 8 also indicates the trend of 'beneficial' effects when an intensity of  $0.4 \mu\text{T}$  was used with pulse A, contrasting with its teratogenic influence when pulse B was employed.

## DISCUSSION

*Effects of electromagnetic fields with pulse A characteristics*

Teratogenic disturbances are observed with field intensities of  $1.0$  and  $13.9 \mu\text{T}$  and not at the lower or higher intensities used, ruling out the possible influence of a rise in internal temperature on the embryonic tissues and confirming the existence of 'windows' in the teratogenic effects of electromagnetic fields (Adey, 1981; Delgado *et al.* 1982). Direct measurement of temperature during pulsating field exposure has been performed in another experimental series, confirming the absence of thermal changes.

Intensities of  $1.0$  and  $13.9 \mu\text{T}$  have only slight, specific effects on development. Exposure to  $1.0 \mu\text{T}$  disturbs the organogenesis of the truncal nervous system, and the developmental stage in abnormal exposed embryos (stage 12) is more advanced than in abnormal controls (stage 10.5). This advance corresponds to about 12 of the total 48 hours of incubation and, among other factors, may be related to an increase of mitotic activity. The frequency of abnormally thin and short neural tubes actually decreases in exposed embryos and very dense truncal folds are detected.

In recent experiments (M. A. Jimenez, in preparation), it has been observed that, after a brief exposure to electromagnetic fields at stages 7, 8 or 9, there is accelerated development accompanied by an increased mitotic index of the neuro-epithelium.

Goodman, Bassett & Henderson (1982) have demonstrated that 15 minutes' exposure of *Sciara coprofila* polytene chromosomes to pulsing fields can provoke a

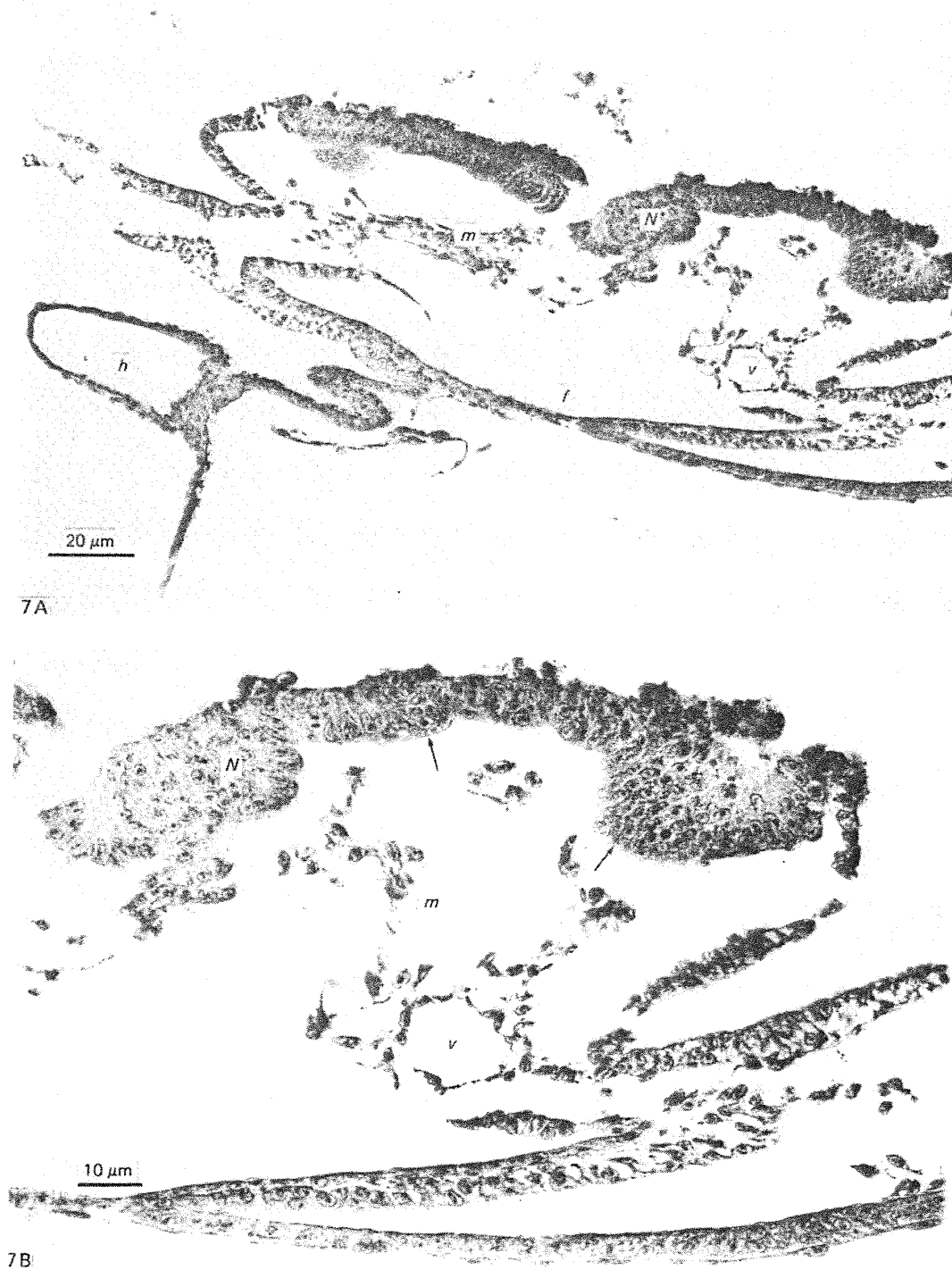


Fig. 7. Embryo exposed to pulse D,  $1.0 \mu\text{T}$ . (A) Transverse section at the level of the heart showing underdeveloped and abnormal nervous system (*N*), foregut (*f*), heart (*h*), and vessels (*v*).  $\times 400$ . (B) The notochord is absent, the basal membrane of the neural ectoderm (arrows) is discontinuous, and the somitic mesenchyme (*m*) is greatly reduced.  $\times 800$ .

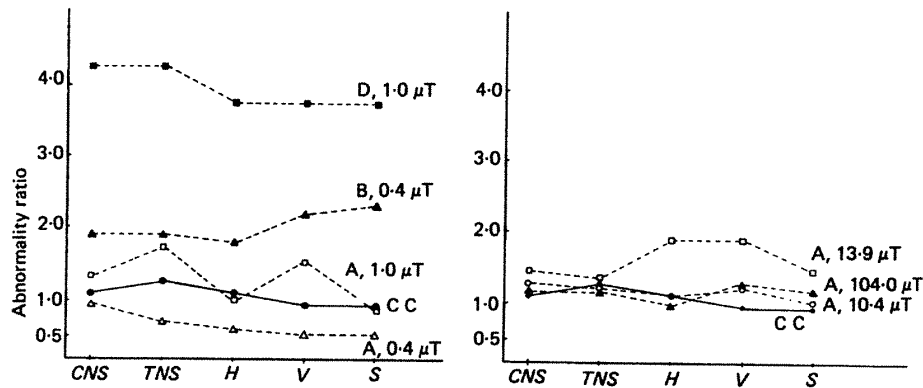


Fig. 8. Abnormality ratio in the cephalic nervous system (CNS), truncal nervous system (TNS), heart (H), vessels (V) and somites (S) in exposed embryos compared with their controls. The ratios were those indicated in Tables 2, 4 and 7. As shown, pulse D, 1.0  $\mu$ T, caused the most teratogenic effects while pulse A, 0.4  $\mu$ T, had a 'beneficial' trend on organic development. CC, Controls inside coils. A, B and D refer to pulse shapes.

fourfold increase in total RNA synthesis. A longer exposure reduces this effect, and RNA transcription diminishes to control levels or below. In the present experiments, exposure of chick embryos for two days to an electromagnetic field of 1.0  $\mu$ T with pulse A could increase mitotic and metabolic activities during what may be a specific sensitive phase of development. In histological sections of the abnormal embryos, truncal neural tubes appear thicker and less organised than in controls at the same developmental stage (Fig. 3 A, B). In these embryos, there is also a drastic reduction of alcian blue-stained components in the basal membrane of the truncal neuroepithelium and in the perinotochordal sheath.

It is known that acid glycosaminoglycans, which are components of tissue basal membranes and extracellular matrix (Kosher & Searls, 1973; Thesleff, 1978; Hay, 1978; Belsky, Vasan & Lash, 1980; Thesleff & Hurmerinta, 1981) play an important role in the regulation of cellular proliferation (Ohnishi, Ohshima & Ohtsuka, 1975; Roblin, Albert, Gelb & Black, 1975; Glimelius & Pintar, 1981), and also in organogenesis. Therefore the two types of histological alteration observed in neural tubes of the exposed embryos may be related to local changes in glycosaminoglycan synthesis, secretion or structure (Takaya, 1977).

Normal embryos of the exposed group reach the same stage of development as controls and no field effect is detected in the histological preparations. Thus it cannot be determined whether only a fraction of the exposed population is sensitive to the electromagnetic field or whether the apparently normal embryos might reveal alterations during their subsequent development. This question could be clarified by allowing the exposed embryos to develop until hatching and by performing physiological studies.

Organogenesis of the circulatory system is altered specifically at a field intensity of 13.9  $\mu$ T without notable effects on the other systems. Transverse sections of malformed embryos show histological anomalies in the foregut which could be related to the specific abnormality of heart development (Manasek, 1976).

No relationship has yet been described between foregut tissue and vascular system organogenesis, but a common regulatory mechanism for development of the circulatory system (i.e. heart, intra-embryonic and extra-embryonic vessels and blood) may be altered by the application of an electromagnetic field. At least for vessel organo-

genesis, it may be proposed that the field acts upon membranes of the pre-endothelial cells, since the cell-to-cell contacts necessary for normal endothelialisation processes are altered in the abnormal embryos.

The normal surface coat of endothelial cells carries a net negative charge due to sialic acid. Bassett (1982) suggests that, in the bone 'gaps' of fracture non-union, the negative charge of fibrocartilage (which is rich in proteoglycans) prevents vascular invasion, so that electromagnetic fields could act by neutralising this electrical charge. In another series of experiments (M. A. Trillo, in preparation) stage 9 chick embryos have been treated *in vivo* with streptomyces hyaluronidase, which specifically hydrolyzes hyaluronic acid. After 10 hours, the embryos develop an enormous vascular system. Therefore, the disappearance of a major, negatively charged, component of the mesenchymal extracellular matrix may stimulate endothelial proliferation. In these experiments, however, the exposed embryos show normal glycosaminoglycan components, as demonstrated by a histological study performed with light microscopy, while abnormal cell-to-cell contacts are observed in the pre-endothelial cells. Therefore, rather than a direct action upon mesenchymal extracellular matrix, the electromagnetic field exposure could alter normal membrane processes of vascular wall differentiation, as in the Pilla (1974) model of living cell membranes.

A developmental delay in normal embryos and an increase in the frequency of non-developed embryos have also been noted in the group exposed to an electromagnetic field of  $13.9 \mu\text{T}$ , indicating that all exposed embryos are affected. The biological response is either morphological, or produces a delay or arrest of the developmental programme.

Transverse sections of all abnormal embryos in groups exposed to field intensities of  $1.0$  and  $13.9 \mu\text{T}$  (pulse A) show a common feature. A dorsoventral flattening is found, which is never observed in the normal embryos or in those exposed to other intensities, or in controls. While the explanation is not yet clear, the effect seems to be specific and typical.

Unlike fields of  $13.9 \mu\text{T}$ , exposure to  $10.4 \mu\text{T}$  has no effect on morphology or on the mean developmental stage attained by the exposed embryos. Chick embryo development appears to be very sensitive to slight variations in the intensity of the applied field.

#### *Electromagnetic field effects with pulse forms B, C and D*

Application of waveform B at an intensity of  $0.4 \mu\text{T}$  has a significant teratogenic effect (Table 6). This intensity, which is 75 times less than the mean value of the Earth's static magnetic field, increases twofold the number of organic abnormalities as compared with controls, and accelerates embryological development. These results demonstrate the fine sensitivity of chick embryos to pulsating electromagnetic fields of extremely low frequency and intensity.

Comparison of field effects with pulses C and D at an intensity of  $1.0 \mu\text{T}$  (Table 6) shows that the biological response is markedly diminished when high frequency noise is added to the magnetic pulse, indicating that embryological effects of pulsed electromagnetic fields are also related to the noise components of the waves. Exposure of eggs to pulse D at an intensity of  $1.0 \mu\text{T}$  induces four times more abnormal embryos than in controls. This effect is similar to our previous findings with comparable electromagnetic field parameters (Delgado *et al.* 1982).

The following specific characteristics of abnormal embryos exposed to waveforms B, C and D may be emphasised. (1) Each abnormal embryo shows anomalies in

various systems, especially those exposed to a field of 0.4  $\mu\text{T}$ , pulse B, and to 1.0  $\mu\text{T}$ , pulse D (Table 7). (2) Cephalic flexion is absent in embryos which have developed beyond stage 11, while marked truncal torsions are present. (3) Truncal neural folds remain open. (4) Mesenchymal cell density decreases in the cephalic region and somites remain as small, poorly organised groups of cells. (5) Lack of cellular cohesion is observed in the different tissues. (6) Acid glycosaminoglycans are considerably reduced in basal membranes and in the extracellular matrix.

It is not yet known whether these effects result from metabolic changes occurring during gastrulation and/or organogenesis, or whether they are caused by direct action of the electromagnetic field on cell membranes and/or on glycosaminoglycan components.

Our results coincide partly with the teratogenic effects of electromagnetic fields on chick embryo development reported by Joshi *et al.* (1978) who describe embryonic abnormalities, including open neural tubes and diffuse somites, caused by exposure to a vertical and continuous electromagnetic field of 5000 Gauss. Similar malformations have been observed in the present study, but the intensity is 500,000 times lower. Hence pulsating fields seem to be far more effective than static fields. Findings presented in this study suggest that embryonic development can be more or less modified by varying the magnetic intensity or pulse shape.

#### SUMMARY

A total of 295 chick embryos was exposed during the first 48 hours of development to pulsed electromagnetic fields of 100 Hz and 0.4 to 104 microTeslas ( $\mu\text{T}$ ), and findings were compared with those in 364 control embryos. General morphology was analysed and supplemented by light microscopy studies.

Exposure to electromagnetic fields with a pulse rise time of 100  $\mu\text{sec}$  produced teratogenic changes when intensities of 1.0 and 13.9  $\mu\text{T}$  were used but not with lower or higher intensities, demonstrating a 'window' effect and ruling out the possible influence of a rise in internal embryonic temperature. Exposure to an electromagnetic field of 1.0  $\mu\text{T}$  specifically altered organogenesis of the truncal nervous system and drastically reduced the alcian blue-stained components, whereas with an intensity of 13.9  $\mu\text{T}$ , there were abnormalities in the circulatory system and foregut, altering cell-to-cell contacts in the walls of developing vessels.

When embryos were exposed to intensities of 0.4 and 1.0  $\mu\text{T}$  with 2.0 and 42  $\mu\text{sec}$  pulse rise times, teratogenic effects were greater and alterations involved all developing systems. The most powerful effects were obtained with 1.0  $\mu\text{T}$  and 42  $\mu\text{sec}$  rise time.

The findings confirm the sensitivity of chick embryos to electromagnetic fields of extremely low frequency and intensity and indicate that pulse shape may be a decisive parameter determining strong, slight, or no modification of embryonic development. Mechanisms of action of electromagnetic fields are still unclear, but induced alterations in extracellular glycosaminoglycans could be a causal factor in the observed malformations.

The authors would like to thank Dr José L. Monteagudo for very helpful discussions and Caroline S. Delgado for her essential support in preparation of the manuscript.



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## The properties of bird feathers as converse piezoelectric transducers and as receptors of microwave radiation. I. Bird feathers as converse piezoelectric transducers.

[Bigu-del-Blanco J, Romero-Sierra C.](#)

### Abstract

An investigation was made of the properties of bird feathers as piezoelectric transducers in the audiofrequency range and as dielectric receptors of electromagnetic radiation in the microwave region. In the first case, cartridges of the ceramic and magnetic type and an electromagnetic transducer probe were used as detecting devices. Results show piezoelectric resonances in the 1 to 20-kHz region for the calami of feathers.

PMID: 1235241

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## The urban decline of the house sparrow (*Passer domesticus*): a possible link with electromagnetic radiation.

[Balmori A<sup>1</sup>](#), [Hallberg O](#).

### Author information

### Abstract

During recent decades, there has been a marked decline of the house sparrow (*Passer domesticus*) population in the United Kingdom and in several western European countries. The aims of this study were to determine whether the population is also declining in Spain and to evaluate the hypothesis that electromagnetic radiation (microwaves) from phone antennae is correlated with the decline in the sparrow population. Between October 2002 and May 2006, point transect sampling was performed at 30 points during 40 visits to Valladolid, Spain. At each point, we carried out counts of sparrows and measured the mean electric field strength (radiofrequencies and microwaves: 1 MHz-3 GHz range). Significant declines ( $P = 0.0037$ ) were observed in the mean bird density over time, and significantly low bird density was observed in areas with high electric field strength. The logarithmic regression of the mean bird density vs. field strength groups (considering field strength in 0.1 V/m increments) was  $R = -0.87$  ( $P = 0.0001$ ). The results of this article support the hypothesis that electromagnetic signals are associated with the observed decline in the sparrow population. We conclude that electromagnetic pollution may be responsible, either by itself or in combination with other factors, for the observed decline of the species in European cities during recent years. The apparently strong dependence between bird density and field strength according to this work could be used for a more controlled study to test the hypothesis.

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Comp Biochem Physiol A Comp Physiol. 1988;89(4):511-30.

## Effects of nonionizing radiation on birds.

Bryan TE<sup>1</sup>, Gildersleeve RP.

### Author information

#### Abstract

1. With the ability to fly comes a greater probability of direct irradiation by nonionizing radiation. The effect of nonionizing radiation on birds is, therefore, of environmental significance. 2. Most biological effects of exposure to nonionizing radiation in avian species are a result of radiation-induced temperature increases. 3. The incubating avian egg provides a model to study nonthermal effects of microwave exposure since ambient incubation temperature can be adjusted to compensate for absorbed thermal energy. 4. Some studies have shown that exposure to nonthermal levels of nonionizing radiation affect a bird's ability to recover from acute physiological stressors. 5. Although earlier research indicated that modulated radiofrequency radiation increased calcium-ion efflux in chick forebrain tissue, criticism of experimental techniques and contradictory results between related studies have made final conclusions elusive. 6. Birds have been shown to be able to reliably detect magnetic fields in both the field and laboratory. Some researchers have reported malformations in chicken embryos exposed to a sinusoidal bipolar oscillating magnetic field.

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EFFECTS OF THE ELECTROMAGNETIC FIELDS OF PHONE MASTS ON A  
POPULATION OF WHITE STORK (*Ciconia ciconia*)

©Alfonso Balmori Martínez. Biologue.  
Valladolid. Spain. March, 2004

*SUMMARY.*— *effects of the electromagnetic fields of phone masts on a population of White Stork (Ciconia ciconia).* Monitoring of a population of White Stork in the vicinity of Cellular Phone Base Stations was carried out in Valladolid (Spain) with the objective to detect possible effects. **Very significant differences among the total productivity of the nests located within 200 meters and those located further than 300 meters of phone masts were found (U=240; p=0,001, test of Mann-Whitney).** In another intensive monitoring carried out around four monuments, **failures in the breeding of this species in the nests near to antennae were observed.** The productivity obtained in this study shows very low levels regarding the censuses previously realized. **The most affected couples could not build the nest,** had disputes for the placement of the sticks and the sticks dropped to the ground. These results are compatible with the possibility that **the microwaves are interfering especially with the reproduction of the white storks** in the inhabited nuclei and would corroborate the results of laboratory research by other authors.

*Key words:* *Ciconia ciconia*, electromagnetic fields, microwaves, non thermal effects, phone masts, White stork

#### INTRODUCTION

The objective of this study was to investigate if the cellsites cause effects in wild birds effects similar to the laboratory studies, and to those recorded in studies carried out on people exposed to this radiation (Lilienfeld, 1978; Hyland, 2000, Hutter *et al.*, 2002; Santini *et al.*, 2003; Navarro *et al.*, 2003) that could cause the abandonment of the area, the failure of the breeding, the decrease of the brood or other anomalies.

The white stork (*Ciconia ciconia*) was chosen to study because it is one of the most vulnerable species. The couple builds their nests on pinnacles and other very high places exposed to the microwaves. Also, they usually live inside urban environment, where the electromagnetic contamination is higher.

## MATERIALS AND METHOD

During the 2002, 2003 and 2004 springs we carried out a monitoring of the reproduction of White Stork (*Ciconia ciconia*) in several nests of Valladolid (Spain).

The spring 2002 we take contact with the problem of the effects of phone masts on the species. That year we began to observe problems in the white storks breeding nearer phone masts. Historical nests disappeared and we began to observe a reduction on the productivity (chicks/nest) and a high mortality of the youngs.

During the spring of 2003 we carried out a more exhaustive monitoring on the breeding success of white stork population. 30 nests were selected located within 200 meters of one or several cellsite antennae and another 30 nests located further than 300 meters of any cellsites, in Valladolid (Spain) (Table 1). The nests were visited in May and June of 2003. To compare the breeding success of both groups of nests a non parametric test was applied (U of Mann - Whitney).

We also carried out 15 visits between the months of February and June 2003 to four historical building (monuments) of Valladolid, with more than 20 nests of the species. The visits embraced all the phases of breeding, from the construction of the nest, until the appearance of youngs exercising the wings and practising flight. The monuments studied were San Pablo (A, B), San Martin (C), Las Angustias (D, E) and La Catedral (F, G) (Fig. 1). We made measurements of the of Electric Field Intensity (radiofrequencies) in the proximity of the monuments.

The results of the previous censuses of white stork carried out in Valladolid and in other Spanish counties were consulted for comparison as a reference for the results obtained in this study.

During the spring of 2004 we are studying by means of observations, the behavior of the most affected white storks.

## RESULTS

**The total productivity (number of young flown by couple, including the nests with 0 chicks), in the nests located within 200 meters of antennae, was  $0,86\pm 0,16$ . For those located further than 300 meters, the result was practically duplicated, with**

an average of  $1,6\pm 0,14$  (Fig. 2). Both groups showed very significant differences in the breeding success ( $U=240$ ;  $P=0,001$ , Test U of Mann-Whitney) (Fig. 2).

In partial productivity (number of young flown by number of couples with some chicks, excluding the nests with 0 chicks), an average of  $1,44\pm 0,16$  was obtained for the first group (within 200 m. of antennae.) and of  $1,65\pm 0,13$  for the second (further than 300 m. of antennae) respectively. The difference between both groups of nests in this case were not statistically significant ( $U=216$ ;  $P=0,26$ , Test U of Mann-Whitney).

Twelve nests (40%) located within 200 meters of antennae didn't have any chicks, while only one (3,3%) of those located further than 300 meters didn't have chicks.

The results of the monitoring realized during the spring of 2003 near the four monuments studied in Valladolid are presented (Fig. 1 and Table 2). The white storks had a total productivity of  $0,6\pm 0,18$  chicks per nest, while the partial productivity was  $1,33\pm 0,23$ . The nests that didn't have chicks generally presented a very scruffy and compressed aspect, as if the couple had not placed sticks in the last months. This happened especially in those located within 200 meters of antennae and on those that the main beam impacted directly. In the nests from San Martin (Fig. 1-C) and San Pablo (Fig. 1-A and B) at least one or two young died from unknown causes. Five nesting sites located within 200 meters from antennae, that received the direct beam of waves, were never built in spite of the couple's repeated attempts (Fig. 1 and Table 2). In their vicinity high intensity levels of Electric Field (higher than 2 V/m) were measured.

The results of the bibliographical revision are presented. The results of productivity of this study are generally less than those obtained in previous studies, especially for the nests located within 200 meters of the cellsite antennae (Table 3).

From the behavior of most affected white storks, the most interesting observations include:

- The couple frequently dispute for the sticks
- Fall of the sticks to the ground while the copuple try to build the nest.
- The couple don't advance in the construction of the nest.
- The most affected nests don't end up being built.
- Increases the number of nests without chicks.
- Frequent death of young in their first stages.
- The storks remain passively in front of phone masts.



## DISCUSSION

The results of the difference of total productivity between the near nests and those far from the antennae indicate the existence of nests without chicks, or the death of young in their first stages in the nests most affected by the microwaves. In the monitoring (more exhaustive) of the monuments near to cellsite antennae, dead young were observed (Fig. 1 and Table 2). Also, several couples (adults) never built the nest. The results of productivity of this study are generally less than those obtained in previous studies, especially for the nests located within 200 meters of the cellsite antennae (Table 3).

**Keeping in mind these results, the microwaves could be affecting one or several reproductive stages: the construction of the nest, the number of eggs, the embryonic development, the hatching or to the mortality of chicks and young in their first stages.**

Other authors have obtained similar results in studies with birds carried out in laboratories (Farrel *et al.*, 1997; Youbicier - Simo *et al.*, 1998; Grigoriew, 2003). Our observations indicate that the most affected nests would be those that are within 200 meters of the cellsite antennae (exposed to the incident beams of one or several antennae focused directly).

A Greek study (Magras & Xenos, 1997) relates to a progressive drop in the number of births of rodents. The mice exposed to  $0.168 \mu\text{W}/\text{cm}^2$  become sterile after five generations, while those exposed to  $1.053 \mu\text{W}/\text{cm}^2$  became sterile after only three generations. The interaction seems to take place through the central nervous system more than on the reproductive gland directly. In the areas of breeding of white storks in this study intensity of electric field levels are overcome (Pers. Obs.).

Other studies find a decrease of fertility, increase of deaths after the birth in rats and dystrophic changes in their reproductive organs (Nikolaevich *et al.*, 2001). An increase in the mortality (Youbicier-Simo *et al.*, 1999) and the appearance of morphological abnormalities, especially of the neural tube (Farrel *et al.*, 1997) has been notified in chicken embryos exposed to pulsed magnetic fields, with different susceptibility among individuals probably for genetic reasons. A recent study shows a statistically significant high mortality of chicken embryos subjected to the radiation from a cellphone, compared to the control group (Grigoriew, 2003). These waves can be affecting the wild birds in the polluted areas in the same way (Balmori, 2003).

The radiofrequency electromagnetic contamination from antennae in cities is much higher than in the rural environment (Pers. Obs.). For this reason urban birds especially can suffer the effects of this radiation. One of the effects that can take place is reduction of the population (specially urban), in places with high electromagnetic contamination. The birds are specially sensitive to the magnetic fields (Liboff & Jenrow, 2000) For this reason they abandon the electromagnetic polluted areas (Balmori, 2003). It is probable that each species, even each individual, show different susceptibility to the radiation, since the susceptibility depends on the genetic bias (Fedrowitz *et al.*, 2004), and of the irradiated living organisms physiologic and neurological state (Hyland, 2001).

In the electromagnetic polluted areas (within an approximate radius of 300-500 meters of an antenna, in the direct emission of the main beam), a deterioration of the good habitat, for the permanency of the birds, takes place. That can cause the abandonment of the breeding areas, sleeping places etc. (Balmori, 2003). In far away areas, where the radiation decreases progressively, the chronic exposure can also have long term effects (Adey, 1996; Magras & Xenos, 1997). The effects from phone masts on the habitat of birds are difficult to quantify, but they can cause a serious deterioration, generating silent areas without male singers nor reproductive couples. The deterioration of the ecosystem can also take place from the impact of the radiation on the populations of invertebrate prey and on the plants (Balmori, 2003).

Microwaves have the potential to induce adverse reactions in the health of people (Hyland, 2000 and 2001, Santini *et al.*, 2002 and 2003; Navarro *et al.*, 2003) and on the fauna that lives in the vicinity of the antennae (Balmori, 2003). The freedom of movement of the birds and their habit of settling in the proximity and even on the cellsites makes them potentially susceptible to the effects. The small organisms (children, birds, small mammals, etc...) are specially vulnerable, so much to approach their size to the frequency of resonance, like for the smallest thickness from their skull that facilitates a higher penetration of the radiation in the brain (Magras & Xenos, 1997; Santini, 2000; Hyland, 2001; Maisch, 2003; Balmori, 2003).

When the experimental conditions (power density, frequency, duration, composition of the tissue irradiated etc.) change, their biological effects also change (Kemerov *et al.*, 1999; Dasdag *et al.*, 1999). Below the levels ( $0,1 \mu\text{W}/\text{cm}^2$ ) recommended in the Salzburg conference adverse effects on health have never notified. At the same time when going away to more than 300 meters distance from the antennas, most of the symptoms notified in people diminish or disappear (Santini, 2003).

Recently it has also been demonstrated that the microwaves used in cellphones produce a non thermal response in several types of neurons of the Nervous System in the birds (Beason & Semm, 2002) and they can affect the blood brain barrier like it has been observed in rats (Salford *et al.*, 2003).

**It is recommended to consider the electromagnetic contamination in the microwave range a risk factor in the decline of some populations, especially the urban birds, subjected to higher radiation levels.**

We consider that the birds most affected from the microwave electromagnetic contamination could be: 1) The ones bound to urban environments with more sedentary customs. In general those that pass a lot of time in the vicinity of the base stations. 2) Those that live or breed in high places, more exposed to the radiations and at higher power density levels. 3) Those that breed on open structures where the radiation impacts directly on adults and chickens in the nest. 4) those that spend the night outside of holes or structures that attenuate the radiation.

**Future investigation should be carried out with long term monitoring of the breeding succes, of the sleeping places and of the uses of the habitat for species more vulnerable to the waves for its behaviour. Of special interest should be the investigations that try to correlate the numeric evolution with the results of the radiofrequency electromagnetic field measurements. Field studies investigating populations of urban parks and territories surrounding cellsites should be high-priority.**

ACKNOWLEDGMENTS. - Denise Ward revised the English translation of the text. To Manuel González for their company in the visit to San Pablo. Juan Matute and José Antonio García provided the information of some white stork censuses carried out in Valladolid.

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FIG. 1

*Sketch with the situation of the building monuments that have been studied. It is also represented the nests or colonies of White Storks (with capital letters) and the phone masts (black triangles with numbers) nearer to the same ones. The vertexes of the triangles point out the approximate direction of main lobe (beam)*

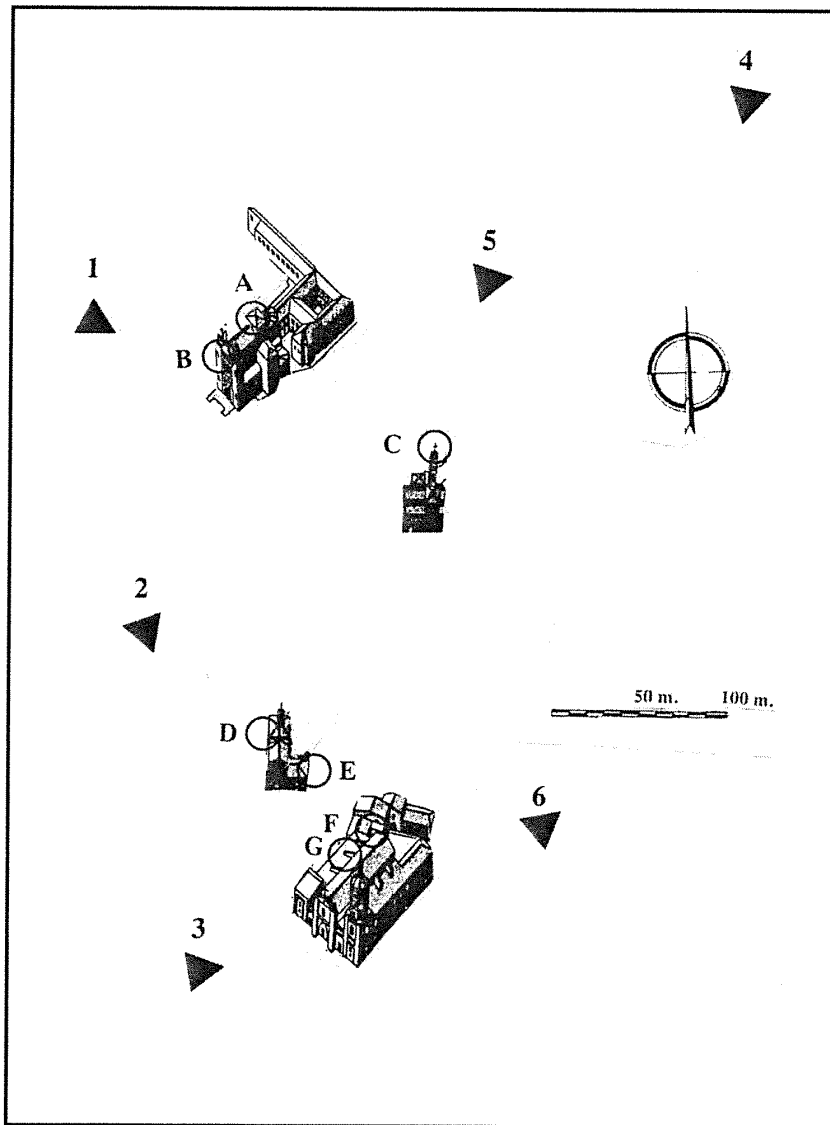


FIG.2

*Comparison of the total productivity (breeding success or n° of chickens for nest) of white stork (Ciconia ciconia) in 30 nests located nearer than 200 meters and 30 located far away than 300 meters of the phone masts.*

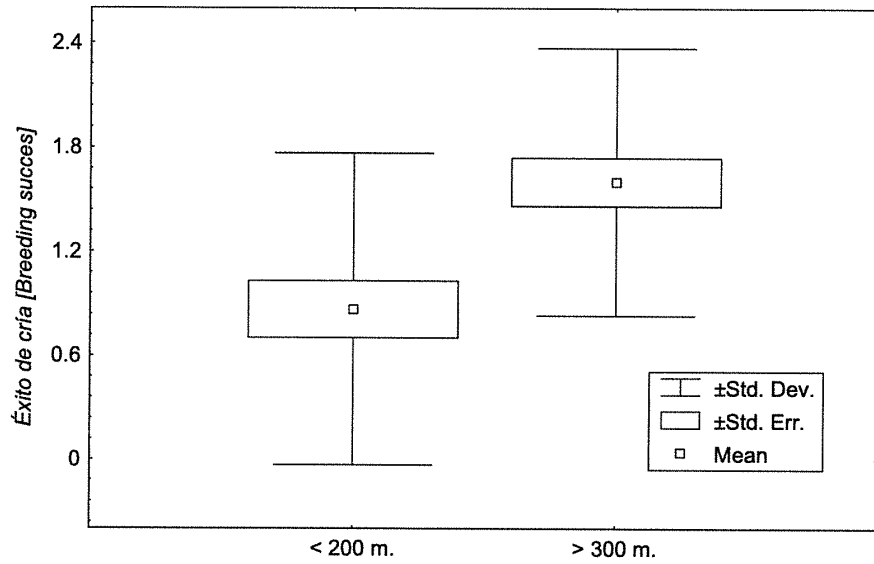


Table 1: Municipalities studied and number of white stork nests in each one

Distance to the more next phone masts			
< 200 meters		> 300 meters	
Boecillo	7	Tordesillas	6
Laguna de Duero	1	Serrada	4
Pesquera de Duero	1	Villanueva de Duero	1
Villanubla	1	Viana de Cega	1
San Pablo (VA)	7	San Bernardo	1
San Martín (VA)	4	Esguevillas de Esgueva	1
Angustias (VA)	2	Villanueva de los Infantes	1
Catedral (VA)	7	Pozaldez	1
		Iscar	1
		Megeces	1
		Dueñas	2
		Cigales	1
		Mucientes	1
		Fuensaldaña	1
		Puenteduero	1
		Simancas	1
		Geria	1
		Villavieja del Cerro	1
		Mota del Marqués	1
		San Cebrián de Mazote	1
		Torrelobatón	1



TABLE 2.- Results of monitoring of breeding white stork (*Ciconia ciconia*) at four building/monuments in Valladolid. Spring of 2003. (See also Fig. 1)

MONUMENT	NEST	NOT FINISH NEST	FINISH NEST		NUMBER OF CHICKS	PHONE MASTS NEAR
			COUPLES WITHOUT CHICKS	COUPLES WITH CHICKS		
SAN PABLO	A	0	4	3	1, 3, 1, 0, 0, 0, 0	1,2,4,5
	B	3	0	0	–	1,2,4,5
SAN MARTÍN	C	0	4	0	0,0,0,0	1,2,4,5,6
ANGUSTIAS	D	0	0	1	2	2,3,6
	E	0	1	0	0	2,3,6
CATEDRAL	F	1	1	2	1,1,0	3,6
	G	1	1	3	1,1,1, 0	3,6
<b>TOTAL</b>		<b>5 (20%)</b>	<b>11 (44%)</b>	<b>9 (36%)</b>	<b>12 (0,6 chicks/nest)</b>	

TABLE 3.

**Results of bibliographical revision**

COUNTY	YEAR	NUMBER OF COUPLES	TOTAL PRODUCTIVITY	PARTIAL PRODUCTIVITY	NUMBER OF UNSUCCESSFUL COUPLES (%)	REFERENCES
Palencia	1984	110	1,51	2,26	24,5	Lázaro <i>et al.</i> , 1986
Soria	1984	61	1,6	2	1,6	Lázaro <i>et al.</i> , 1987
Segovia	1984	246	1,01	2,06	45,9	Lázaro <i>et al.</i> , 1988
Ávila	1984	188	0,97	1,81	42	Lázaro <i>et al.</i> , 1989
Burgos	1984	77	1,39	2,04	27,2	Lázaro <i>et al.</i> , 1990
León	1984	397	1,44	1,99	23,9	Lázaro <i>et al.</i> , 1991
Salamanca	1984	591	1,68	2,03	11,1	Lázaro <i>et al.</i> , 1992
Zamora	1984	260	0,96	2,14	16,5	Lázaro <i>et al.</i> , 1993
Ávila (Valle del Tietar)	1985	78	2,69	3,04	8,97	Muñoz <i>et al.</i> 1988
Ávila (Valle del Tietar)	1986	71	2,17	2,62	14,08	Muñoz <i>et al.</i> 1988
Ávila	1986	151	2,22		18,5	Hernández, 1987
Zamora	1986	201		2,32		Ocellum durii, 1986
Ávila	1989	150	1,77	2,46		Hernández, 1989
León	1990	509	2,56	2,75	6	Urz, 1990
Palencia	1991	205	1,85	2,5		Gepopn, 1991
SPAIN	1984	6753	1,39	2,12	16,6	Lázaro <i>et al.</i> , 1986
SPAIN	1994	16643	1,6	2,5	7,9	Martí <i>et al.</i> , 1999
Valladolid (provincial)	1984	113	1,69	2,13	7	Lázaro <i>et al.</i> , 1986
Valladolid (provincial)	1992	115		1,93	5,2	Alauda, 1992
Valladolid (capital)	1994	24	1,84		7,6	Alauda, 1994
Valladolid (capital)	2001	35		2,43		García, 2001
VALLADOLID	2003 (<200 m.)	30	0,83	1,44	40	THIS STUDY
VALLADOLID	2003 (>300 m.)	30	1,6	1,65	3,3	THIS STUDY

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Bioelectromagnetics. 1984;5(3):331-9.

## The effects of microwave radiation on avian dominance behavior.

Wasserman FE, Dowd C, Schlinger BA, Byman D, Battista SP, Kunz TH.

### Abstract

Seventeen birds from 12 flocks were exposed to microwave radiation under various combinations of power density and duration; three birds from two additional flocks served as sham-exposed controls. Experiments were conducted outdoors at Manomet, Massachusetts (41 degrees 56'N, 70 degrees 35'W) under normal winter ambient temperatures. Although irradiated birds maintained their positions within a flock hierarchy with one exception, some appeared to have a change in their level of aggression after exposure.

PMID: 6487384

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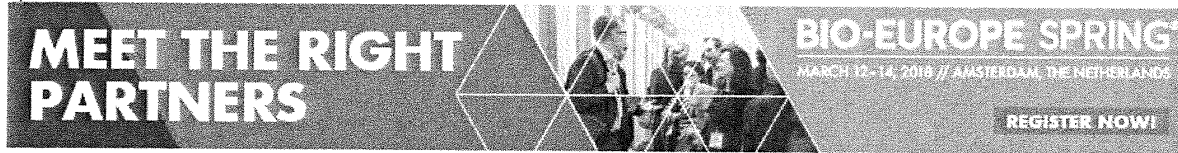
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Original Article

# The Effects of Chronic Exposure to Very Low Intensity Microwave Radiation on Domestic Fowl

J. A. Tanner & C. Romero-Sierra

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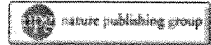
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# RETRACTED ARTICLE

**See: Retraction Notice**

Int J Impot Res. 2010 Mar-Apr;22(2):127-33. doi: 10.1038/ijir.2009.57. Epub 2009 Nov 26.

## Effects of exposure to a mobile phone on sexual behavior in adult male rabbit: an observational study.

Salama N<sup>1</sup>, Kishimoto T, Kanayama HO, Kagawa S.

### Author information

### Retraction in

Retraction. Effects of exposure to a mobile phone on sexual behavior in adult male rabbit: an observational study. [Int J Impot Res. 2012]

### Abstract

The accumulating effects of exposure to electromagnetic radiation emitted by a conventional mobile phone (MP) on male sexual behaviour have not yet been analyzed. Therefore, we studied these effects in 18 male rabbits that were randomly divided into phone and control groups. Six female teasers were taken successively to the male's cage and the copulatory behavior was recorded. Serum total testosterone, dopamine and cortisol were evaluated. The animals of the phone group were exposed to MPs (800 MHz) in a standby position for 8 h daily for 12 weeks. At the end of the study, the copulatory behavior and hormonal assays were re-evaluated. Mounts without ejaculation were the main mounts in the phone group and its duration and frequency increased significantly compared with the controls, whereas the reverse was observed in its mounts with ejaculation. Ejaculation frequency dropped significantly, biting/grasping against teasers increased notably and mounting latency in accumulated means from the first to the fourth teasers were noted in the phone group. The hormonal assays did not show any significant differences between the study groups. Therefore, the pulsed radiofrequency emitted by a conventional MP, which was kept on a standby position, could affect the sexual behavior in the rabbit.

PMID: 19940851 DOI: [10.1038/ijir.2009.57](https://doi.org/10.1038/ijir.2009.57)

[Indexed for MEDLINE]

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## Electromagnetic Radiation (EMR) Clashes with Honey Bees

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### ABSTRACT

Apiculture has developed in to an important industry in India as honey and bee-wax have become common products. Recently a sharp decline in population of honey bees has been observed in Kerala. Although the bees are susceptible to diseases and attacked by natural enemies like wasps, ants and wax moth, constant vigilance on the part of the bee keepers can over come these adverse conditions. The present plunge in population ( $< 0.01$ ) was not due to these reasons. It was caused by man due to unscientific proliferation of towers and mobile phones.

**Key words:** Electromagnetic radiation, Apiculture, Colony collapse disorder.

### 1. Introduction

Bees and other insects have survived and evolved complex immune system on this planet over a span of millions of years. It is not logical that they would now suddenly die out now due to diseases and natural parasites. This suggests another factor has been introduced to their environment that disrupts their immune system. This man made factor is the mobile towers and mobile phones.

The public is not being informed of the threat due to deliberate attempts on the part of mobile phone makers to mask the direct causal relationship. Over the past several months a cadre of scientists, funded by the deep pockets of the mobile phone industry, has suggested viruses, bacteria, and pesticides are to blame for the unprecedented honey bee decline. Rather than critically assessing the problem, the industry is dealing with it as a politics and public relation problems thus manipulating perception of the appropriate remedy. Sadly, this deceptive practice is business as usual for the mobile phone industry. If the reason behind the population decrease were biological or chemical there would be a pattern of epidemic spread. Observers would be able to trace the spread of bee disappearance from a source similar to the spread of SARS a few years ago. This pattern did not occur, however mobile towers and mobile phones meet the criterion.

New experiments suggests a strong correlation between population decline and cellular equipment. . The massive amount of radiation produced by towers and mobile phones is actually frying the navigational skills of the honey bees and preventing them from returning back to their hives. The thriving hives suddenly left with only queens, eggs and hive bound immature worker bees. Thus electromagnetic radiation exposure provides a better explanation for Colony Collapse Disorder (CCD) than other theories. The path of CCD in India has followed the rapid development of cell phone towers, which cause atmospheric electromagnetic radiation.

Insects and other small animals would naturally be the first to obviously be affected by this increase in ambient radiation since naturally they have smaller bodies and hence less flesh to be penetrated by exposure to microwaves. The behavioral pattern of bees alters when they are in close proximity to mobile phones and towers. The vanished bees are never found, but thought to die singly far from home. Bee keepers told that several hives have been abruptly abandoned. If towers and mobile phones increase the honey bees might be wiped out in ten years. Radiation of 900 MHz is highly bioactive, causing significant alternation in the physiological function of living organisms <sup>7</sup>.

## **2. Materials and Methods**

Six colonies of honeybees (*Apis mellifera*) were selected. Three colonies were selected as test colonies (T<sub>1</sub>, T<sub>2</sub> & T<sub>3</sub>) and the rest were as control (C<sub>1</sub>, C<sub>2</sub> & C<sub>3</sub>). The test colonies were provided with mobile phones in working conditions with frequency of 900 MHz for 10 minutes for a short period of ten days. EMF (Electromotive field) power density was measured with the help of RF Power density meter. The control colonies had not provided with mobile phones. Queen prolificacy was calculated in terms of egg laying rate of the queen <sup>1</sup>. Flight activity and returning ability were measured as number of worker bees leaving and returning respectively to the hives per minute: before exposure, during exposure and after exposure.

## **3. Results**

The results of the studies are presented in Table. The present study showed that after ten days the worker bees never returned hives in the test colonies. The massive amount of radiation produced by mobile phones and towers is actually frying the navigational skills of the honey bees and preventing them from returning back to their hives <sup>1-4</sup>. It was shown that the total bee strength was significantly higher in the control colonies being nine comb frames as compared to one in the test colony at the end of the experiment. The thriving hives suddenly left with only queens, eggs and hive bound immature worker bees. The queens in the test colonies produced fewer eggs/day (100) compared to the control (350). It has previously been reported that there is low egg laying rate in queens exposed to high voltage transmission lines <sup>5</sup> or exposure of the queen bees to cell phone radiation stimulated her to produce only drones <sup>6</sup>. Thus electromagnetic radiation (EMR) exposure provides a better explanation for Colony Collapse Disorder (CCD) than other theories. The path of CCD in India has followed the rapid development of cell phone towers and cell phones, which cause atmospheric electromagnetic radiation.

## **4. Discussion**

Some countries have sought to limit the proliferation of mobile towers with strict rules. But in India no such rules have been formulated or implemented. Given the proliferation of mobile phone towers and their vital role in communications, solutions to the problem will not be as simple as eliminating the towers. One possibility is shielding the bee hives with EMR resistant materials.

Another solution would be granting local communities the ability to control whether or not to install mobile towers. On one hand, community members would be able to exert some control over their environment and determine whether the benefits outweigh the costs and risks. On the other, it is highly susceptible to manipulation by powerful influences, especially since the



bee keepers have significantly less influence, power and wealth than the mobile phone companies.

However, Indians could risk losing even this right to self determination if the cellular providers can impose a country wide mandate prohibiting regulation against them, similar to the Telecommunications Act of 1996 in the United States. The Act prohibited local governments from making sitting decisions based on the perceived health impacts of wireless facilities. Indian advocates are concerned that such regulations might be upheld in India as they were in the United States in order to “eliminate service gaps in its cellular telephone service area.” In Kerala there are about 600,000 beehives and over 100,000 workers are engaged in Apiculture. A single hive may yield 4-5 kg of honey. Moreover, the destruction of bee hives could be a major environmental disaster. Honeybees are responsible for pollinating over 100 commonly eaten fruit and vegetable crops and without bees the food system would be in serious trouble. Rural village dependent on locally grown foods would be most vulnerable. The need of the hour is to check unscientific proliferations of mobile phone towers. More research is essential on how to protect the bee hives from the electromagnetic exposure, but perhaps more to study the impacts on humans.

**Table 1:** Change in colony status of honeybees exposed to mobile phones

Parameter	Control ( mean ± SD)	Treated ( 10 m exposure for 10 days.
( No. of worker bees leaving the hive entrance/ minute)		
Before exposure	40.7±15	38.2±12
During exposure	41.5±14	18.5±13
After exposure	42.4±14	Nil
<b>Returning ability</b>		
Before exposure	42.5±15	39.5±14
During exposure	43.6±14	15.6±13
After exposure	44.6±13	Nil
<b>Bee strength</b>		
Before exposure	9 Frame	9 Frame
During exposure	9 Frame	5 Frame
After exposure	9 Frame	1 Frame
<b>Egg laying rate of queen /day</b>		
Before exposure	365.25	355.10
During exposure	362.15	198.60
After exposure	350.15	100.00

All mobile phone towers emit microwave radiations, which is in the radio frequency radiation (RFR), part of the spectrum of electromagnetic waves. Though RFR, like Ultra -violet (UV) and Infra-red light, is a source of non-ionizing radiation, these radiations, together with ionizing electromagnetic radiations such as X- rays, gamma rays make up the electromagnetic spectrum. Radio frequency of the electromagnetic waves ranged from 100 kilo hertz (KHz) to 300 Giga hertz (GHz). Radio frequency radiation is a source of thermal energy and in adequate doses, has all the known effects of heating on biological systems <sup>7</sup>.

Despite a growing number of warnings from scientists, like me, the Government has done nothing to protect people and the environment. Steps must be taken to control the installations of mobile phone towers by imposing restrictions. Installation of towers should be regulated near thickly populated areas, educational institutions, hospitals etc. Sharing of towers by different companies should be encouraged, if not mandated. To prevent overlapping high radiations fields, new towers should not be permitted within a radius of one kilometer of existing towers.

More must also be done to compensate individuals and communities put at risk. Insurance covering diseases related to towers, such as cancer, should be provided for free to people living in 1 km radius around the tower. Independent monitoring of radiation levels and overall health of the community and nature surrounding towers is necessary to identify hazards early. Communities need to be given the opportunity to reject cell towers and national governments need to consider ways of growing their cellular networks without constantly exposing people to radiation.

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## DOSE RELATED SHIFTS IN THE DEVELOPMENTAL PROGRESS OF CHICK EMBRYOS EXPOSED TO MOBILE PHONE INDUCED ELECTROMAGNETIC FIELDS

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**Background:** The possible adverse effects of Electromagnetic Fields (EMFs) emitted from mobile phones present a major public concern today. Some studies indicate EMFs effects on genes, free radical production, immunological and carcinogenic effects. On the other hand there are studies which do not support the hypothesis of any biological impacts of EMFs. This study was designed to observe the effects of mobile phone induced EMFs on survival and general growth and development of chick embryo, investigating dose-response relationship if any. **Methods:** This was an experimental study in which developing chick embryos were exposed to different doses of mobile phone induced EMFs. For this purpose a mobile phone was placed in the incubator in the centre of fertilised eggs in silent ringing mode and was 'rung' upon from any other line or cell phone. After incubation for 10 or 15 days the eggs were opened and the developmental milestones of the surviving embryos were compared with the non exposed subgroup. **Results:** EMFs exposure significantly decreased the survivability of the chick embryos. The lower doses of EMFs caused growth retardation. However, this effect of growth retardation reallocated to partial growth enhancement on increasing the dose of EMFs and shifted over to definite growth enhancement on further raising the dose. **Conclusion:** There is an adverse effect of EMFs exposure on embryo survivability. Chick embryos developmental process is influenced by EMFs. However, these effects are variable depending upon the dose of EMFs exposure.

**Keywords:** EMFs, Chick embryo, Mobile phone

### INTRODUCTION

The possible health hazards of mobile phone induced Electromagnetic Fields (EMFs) is no longer a new debate now. The 'interference phenomenon' of the mobile phone induced EMFs and those from the biological cellular activities, has been described as the potential cause of non thermal health effects. There are many studies which have contributed to the still unresolved safety issue of the EMFs. Many have reported the toxicological effects of EMFs, ranging from cognitive effects to genotoxicity, and from skin ulcers to brain tumours, however there are contrasting studies in which EMFs have been utilised for therapeutic purposes.

The mobile phone operates upon radiofrequency EMFs, is held very close to human body, and requires base stations for its functionality. Hence the user's body is exposed to two sources of EMFs; one from the mobile phones itself and the other from the base stations usually implanted in residential areas. Obviously the dose of EMFs irradiation is different in different cases.

Keeping in view the casual implantation of the base stations in residential areas, and long talk times of the consumer this study was designed to investigate the dose dependant effects of mobile phone induced EMFs on the general growth and survivability of developing chick embryos.

### MATERIALS AND METHODS

This Experimental study was carried out at the Department of Anatomy, College of Physicians and Surgeons Pakistan, Regional Centre, Islamabad. Fertilised chicken eggs of 'Desi' breed were obtained from Poultry Research Institute of Punjab, Rawalpindi and were randomly divided into two main groups; Control A (n=60 eggs), and experimental B (n=120 eggs). Group A was further subdivided into 2 subgroups, Aa (n=30 eggs) sacrificed on day 10, and Ab (n=30 eggs) sacrificed on day 15 of incubation. The first day of incubation was taken as day one. Group B was subdivided into two series B1 and B2 based on the dosage of EMFs exposure. B1 (n=60 eggs), was exposed to 15 minutes of mobile phone 'silent ringing' twice daily while series B2 (n=60 eggs) was exposed to 25 minutes of the same. Each series of the Group B, i.e., B1 and B2 were again divided into 2 subgroups labelled as 'a' and 'b' according to their day of sacrifice, each subgroup having 30 eggs. Subgroups B1a and B2a were sacrificed on day 10, while subgroup B1b and B2b were sacrificed on day 15 of incubation.

For the purpose of description the exposure levels were graded in ascending order as under:

- GRADE I: Low dose, less duration exposure level (for B1a subgroup)
- GRADE II: High dose, less duration exposure level (for B2a subgroup).

- GRADE III: Low dose, longer duration exposure level (for B1b subgroup)
- GRADE IV: High dose, longer duration exposure level (for B2b subgroup)

The exposure grading was one of the methodological limitations of the study. Grade II was high dose exposure till day 10 of embryonal life, while grade III was low dose exposure till day 15 of the embryonal life. It was difficult to mathematically grade the net exposed dose between these two exposure levels. However for the purpose of study the longer duration of exposure, i.e., till day 15 was taken as more intense intervention in the developmental process and considered as a higher dose level.

The eggs were arranged in double ring pattern on a double storey circular plate with 15 eggs on each plate. This plate system was placed in the incubator under standard incubation conditions. In the experimental group a mobile phone was placed in the centre of the lower plate on a small rectangular stand (Figure-1). This mobile was periodically rotated for maintaining almost equal levels of EMFs exposure in all directions through its built in antenna. The mobile phone was 'rung' upon (in silent and non-vibrant mode) from any

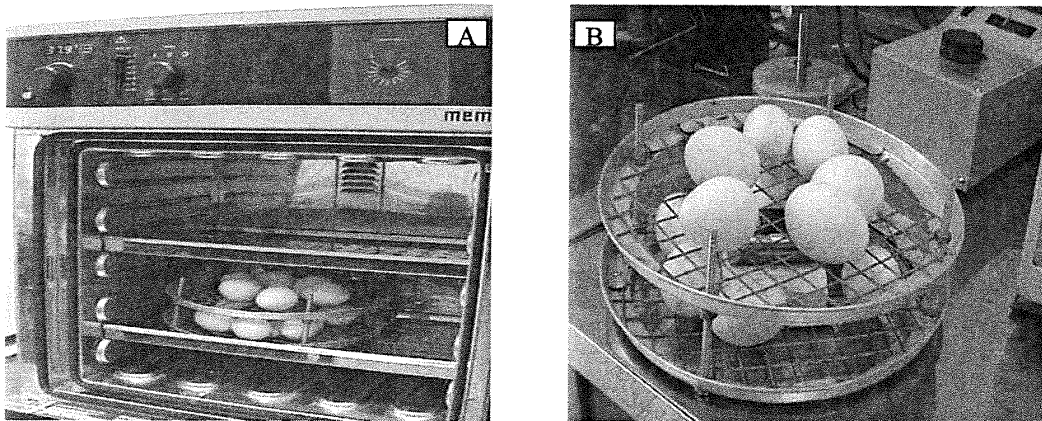
other line or cell phone for the respective schedule of timings set for different subgroups. On the day of sacrifice the eggs were taken out of the incubator and broken open by conventional methods. The embryos were dissected out of their membranes and their survivability was noticed. The live embryos were processed further.

After a fixation time of 48 hours in 10% buffered formalin the embryos were taken out of the fixative, blotted dry on a piece of tissue paper and their lengths and weights were measured.

The length was taken from the vertex (highest point between the eye balls) and the tip of coccyx along the curvature of the spine. For this purpose a thread was stretched over the contours of the embryo between the above mentioned two points and the distance covered by the thread was measured by a scale.

The embryos were weighed using a precision digital balance with 0.001 gm readability.

Student's *t*-test was applied to detect any significant differences in means of the gross weights and lengths, and to the percentage survival of the embryos. A *p*-value of 0.05 or less was taken as significant.



**Figure-1: The exposure system**

A: Double storey plate carrying fertilised chicken eggs kept in the incubator under standard conditions, with a mobile phone positioned in the centre of the lower storey. B: Magnified view of the exposure plate showing the position of the mobile phone. Note that all eggs are within a distance of one wavelength of the EMFs from the mobile phone.

## RESULTS

The effect of EMFs on the survivability of chick embryos was evaluated by applying student's *t* test on the percentages of the dead embryos which were 3% in the control group A, and 21.06% in the treated group B. A *p*-value of < 0.001 indicated a significant increase in percentage of dead embryos in the Experimental group B as compared to control group A.

### Assessment of growth parameters with respect to different exposure grades of EMFs:

#### Exposure Grade I:

The post fixed embryonal weights and lengths of the embryos exposed to Grade I were significantly less than those of the control embryos ( $p < 0.001$ ). This indicated a delayed growth in the treated subgroup as compared to the control one.

**Exposure Grade II:**

The post fixed weight of this subgroup was more than the control subgroup ( $p<0.001$ ). The embryonal length was less than the control; however difference of mean lengths noticed was not significant. Hence, ignoring the non significant factors, this exposure level has caused a partial growth enhancement of the developing embryos i.e. with respect to the weight of the developing embryos only.

**Exposure Grade III:**

The post fixed weights and lengths of the treated subgroup were significantly more than the control embryos. These findings suggest a developmental stage more advanced than the exposure level II, where only the parameter of weight was significantly more than the control. (In this exposure level, however, the significance of weight difference was more ( $p<0.001$ ) as compared to length difference ( $p<0.01$ .)

**Exposure Grade IV:**

Grossly the embryos were the most advanced in development as compared to the other exposure levels. The weights and lengths of embryos of this subgroup were significantly more than the controls with a  $p<0.001$ .

**Table-1: Comparison of mean weights of treated and control groups by student's t-test**

Exposure Grades	Treated subgroups Mean weight±SE (grams)	Control subgroups Mean weight±SE (grams)	p value
I	B1a (1.207±0.02)	Aa (1.394±0.02)	<0.001
	B2a (1.513±0.02)	Aa (1.394±0.02)	
II	B1b (9.563±0.285)	Ab (7.399±0.259)	0.001
	B2b 11.563±0.311	Ab (7.399±0.259)	

**Table-2: Comparison of mean lengths of treated and control groups by student's t-test**

Exposure Grades	Treated subgroups Mean length±SE (cm)	Control subgroups Mean length±SE (cm)	p value
I	B1a (4.129±0.050)	Aa (4.341±.032)	0.001
	B2a (4.288±0.039)	Aa (4.341±.032)	
II	B1b (7.375±0.156)	Ab (6.845±0.098)	<0.01
	B2b (7.721±0.124)	Ab (6.845±0.098)	

**DISCUSSION**

As noticed in the results the survivability of embryo worsened by the exposure to EMFs. However, these effects were not dose dependant. Most of the previous studies have also ruled out the dose dependant association of the radio frequency EMFs to their biological effects. However, Lai and Singh in their study earlier had exposed rats to 2450 MHz microwaves and demonstrated damage to DNA, which was dose dependant.<sup>1</sup>

The present study shows that in the surviving embryos, lower doses of EMFs caused growth retardation of the developing embryo. This effect of 'growth retardation' reallocated to 'partial' growth enhancement, on increasing the dose of EMFs and shifted over to 'definite' growth enhancement on further raising the dose.

**GROWTH DEPRESSION:**

Growth suppression of embryos exposed to EMFs has been reported by other researchers. Atli and Unlu have studied the effects of microwave frequency electromagnetic fields on the development of *Drosophila melanogaster* and have reported that EMF can cause developmental delay.<sup>2</sup>

Following are some of the factors which have been shown to cause growth retardation and may have contributed to the developmental delay noticed in this experiment.

• **Hormonal Stress:**

The EMFs are a source of stress induction in biologically active tissue.<sup>3</sup> Stress hormones although protective in the immediate aftermath of stress can promote damage when they are overproduced or not turned off. This wear-and-tear of the body has been called allostastic load.<sup>4</sup> Allostasis is the active process of maintaining stability, or homeostasis but allostastic load is the almost inevitable cost to the body of doing so. Intense or prolonged provocation of this response contributes to what have been called 'diseases of adaptation'. Recent research suggests that increased adrenocortical and sympathoadrenal responses are associated with small size at birth.<sup>5</sup>

• **Oxidative stress:**

EMFs also cause oxidative stress resulting in an increased likelihood of cell injury and cell death. Recent study on human volunteers exposed to EMFs for up to 4 hours showed statistically significant oxidative stress.<sup>6</sup> Same has been seen in animal studies.<sup>7</sup> Oxidative stress has been shown to cause delay in the embryonal development.<sup>8</sup>

• **Interference between EMFs and the biological electromagnetism:**

The sequences of developmental events are guided by endogenous ionic currents and electric fields. Disruption of these fields through the EMFs exposure can adversely affect these events<sup>9</sup> hindering the normal developmental process.

• **Genotoxicity:**

The genotoxic effect of the EMFs has come into discussion recently.<sup>10</sup> When cells encounter DNA damage, a cascade of signal transduction pathways activate cell cycle checkpoints leading to blocked cell cycle progression.<sup>11</sup> Such checkpoints have been reported to suppress cellular growth and in an active

process like embryogenesis these reactions could result in growth retardation.

**REVERSAL OF DOSE-EFFECT RELATIONSHIP:**

The delayed development induced by the EMFs in the project shifted to initially partial, and then definite growth enhancement with rising doses. There could be three possible explanations to this dose-effect reversal phenomenon, namely:

• **A pre-conditioning effect of EMFs:**

According to this effect any external influence at first manifests its damaging effect through stress proteins. Later, intracellular over production of these same proteins can render cytoprotection.<sup>12</sup> Hence the reversal of the growth retardation to growth stimulation may be explained on the same grounds.

• **Dose cumulating effect:**

EMFs have been scientifically and clinically shown and approved to be highly active 'medicines' for cells, animals and plants. So the prolonged exposure of EMFs may have had a cumulative effect leading to growth stimulation rather than depression as noticed with lower doses.

• **Role of genetics:**

It may have been a role of individual genetic make-up, that different flock of embryos (although belonging to the same species), had responded or suffered differently to the EMFs. Although this does not explain the dose related reversal of effects but may be a contributing factor in the diversity of the observed results.

**GROWTH ENHANCEMENT:**

The increase in growth noticed with higher doses can be explained as simple controlled growth activation or it can be debated as an uncontrolled preneoplastic manifestation. Three possible mechanisms involved could be a direct stimulatory effect, improved developmental environment or carcinogenic influences.

• **Direct stimulatory influence:**

Several different factors can cause embryonal growth activation as discussed under:

**i. Growth factors**

Recent studies suggest that EMFs exposure might function as soluble growth factors.<sup>13</sup> Roles of growth factors in early embryogenesis is also coming into picture. Insulin-like growth factor-I has been shown to stimulate DNA replication<sup>14</sup>; insulin-like growth factor II has a potential role in the endothelial-mesenchymal transition<sup>15</sup> while Fibroblast Growth Factor has been found to orchestrate gastrulation movements.<sup>16</sup>

**ii. Increased cell proliferation**

A study in 2006 suggested that EMFs had progressive stimulatory effects on both cellular proliferation and differentiation of mouse embryo limb bud.<sup>17</sup> Understandingly where such kind of

effects can lead to advanced growth in developing embryos, they also hint the possibility of cancer transformation. Also this potential of EMFs has been utilized clinically for healing and repair of wounds and fractures.<sup>18</sup>

• **Improved environment:**

**i. Cytoprotection/Role of Stress proteins**

Intracellular over expression of heat shock proteins has been shown to render cytoprotection in many studies in which EMFs were used as stress inducers.<sup>20</sup> These proteins can render protection to the cells against other potential challenges of the embryonal life and render protection against cytotoxic and proteotoxic effects.<sup>21</sup> There could be a possibility that production and protective influence of stress proteins against the EMFs biological effects was dose dependant and required a specific threshold. This working mechanism has supportive evidence from the past in which prolonged heat stress leading to heat acclimation increases the basal Hsp72 level<sup>22</sup> and predisposed the Hsp molecular machinery to respond faster increasing the cytoprotection.<sup>23</sup>

**ii. Improving hypoxia**

Electromagnetic field exposure of chick embryos has been shown to protect against hypoxic insults.<sup>24</sup> In a reported study EMFs limited the area of necrosis after ischemic injury caused by permanent ligation of the left anterior descending artery of rats.<sup>25</sup> An improved hypoxia protection definitely is a better environmental condition for embryonal development.

**iii. Increased metabolic processes**

EMFs are said to activate metabolic processes, specifically the synthesis of factors controlling early embryonic development. In a study two cell mouse embryos were cultured in vitro. After exposure to EMFs, they acquired the ability to develop on their own and reached the stage of blastocyst without serum or growth factors in the culture.<sup>26</sup> These findings indicate that EMFs have a stimulating effect on the early development of embryos, increasing the resistance of embryos to unfavourable environmental conditions.

• **Carcinogenic influences:**

Findings suggest changes in polyamines, activation of c-myc and c-fos gene expression in cells exposed to electromagnetic fields.<sup>19</sup> Polyamines are compounds playing a role in both protein synthesis and cell differentiation through c-myc and c-fos gene activation. Thus EMFs affecting these proteins are affecting the mechanisms involved in cell proliferation and differentiation. These altered mechanisms also raise the probability of an uncontrolled neoplastic cellular proliferation induced by EMFs.

## CONCLUSION

The results of this project suggest that the exposure of the developing chick embryo to EMFs decreases their survivability. Regarding the surviving embryos, different exposure doses exhibited different developmental patterns. Whereas the lower doses of EMFs exposure delayed the developmental process of the treated embryos, there was a partial and then definite growth enhancement on progressively increasing the exposure levels. The prevailing controversy regarding the health issues of EMFs may be evaluated in the light of dose related response diversities. Results of this project also demand focus of future studies on the tumorigenic tendency of EMFs.

It is suggested that till the emergence of more definite facts, use of EMFs devices including mobile phone should be limited to minimum.

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PC 2-8-18

#3

Planning Department <planning@edcgov.us>

## Deny the proposed conditional use permit for AT&T Cell Tower Site 5 on parcel APN #087-181-10 – 7160 Dragon Point Road

1 message

Amy Haydt <amyhaydt@sbcglobal.net>

Mon, Jan 29, 2018 at 4:14 PM

Reply-To: Amy Haydt <amyhaydt@sbcglobal.net>

To: "planning@edcgov.us" <planning@edcgov.us>

Cc: "bostwo@edcgov.us" <bostwo@edcgov.us>, "Gary.miller@edcgov.us" <Gary.miller@edcgov.us>, Eric Haydt <erichaydt70@gmail.com>, "brad@vaminvest.com" <brad@vaminvest.com>

To Whom it May Concern,

We are writing to ask that you **deny** the proposed permit referenced above for AT&T to put a cell tower on our neighbor's property. We are the titled owners of the property at 7180 Dragon Point Road, directly next to the parcel upon which this tower is proposed to be built. We are opposed to this proposed project for the following reasons.

(1) We live in a very rural area, each with 20 acre parcels that are currently zoned for 40 acres. Our properties are filled with rolling hills, quiet creeks and ponds, meandering wildlife, and acres and acres of beautiful old oaks. Building a 120-160 foot cell phone tower in the middle of this type of rural residential area will completely **deduct from the rural setting** in which we currently live. Even the biggest digger pines that grace our properties will be overshadowed by such a monstrosity. Such a detraction will inevitably have a **negative impact on our property values**, as has been confirmed by numerous real estate specialists in our area with whom I have discussed this situation.

(2) Moreover, the **peace and tranquility of our area will be marred** by the constant buzzing sound that will echo through the valley of our hills if this tower is built. Our properties sit in an area that is so quiet I can sometimes hear conversations that occur on the decks of the houses that sit on the 20 acre parcels on either side of us. You can be sure we will be negatively affected by even the slightest buzzing sound that I know these towers emit day in, day out.

(2) There is **NO NEED** for the service that would be provided by such a tower. There are currently at least 7 other such towers within four miles of our property. Thus the tower would simply be providing additional service, not necessary new service. We already have high speed internet provided to our residence. Thus, when you consider the cost of the negative impact to our property values as described above, it seems that any slight benefit yet another tower may provide to our area generally would be substantially outweighed.

(3) Dragon Point Road is a privately owned and paved road, personally maintained by each of the owners of properties that sit on Dragon Point Road. It would have an unwelcome burden and financial impact on the maintenance and upkeep on our privately owned road for AT&T to use the road for all of the necessary equipment and upkeep during both the initial building and then continued maintenance of their proposed cell tower. Moreover, the owners of the properties over which Dragon Point Road runs **WILL NOT give easements** to AT&T for the use of the road to build and maintain this tower. Thus, if you approve the permit, you'll be aiding them in perpetrating a knowingly **illegal trespass** with substantial damage. I find it highly unlikely the county would want to be a party to such an issue.

(4) Placing an unmanned cell phone tower to which electricity will be run and a diesel generator will be used during power failures substantially **increases the risk of extreme wildfire danger** in an area that is already at high risk of such a fire. Moreover, given the rural nature of our area, we have power outages constantly; I'd estimate at least four times a year. Every time that unmanned propane generator kicks in it will increase the risk of starting a wildfire in an area that is already under served by fire service.

By way of example, when our property, which again is right next door to the parcel upon which this tower is proposed to be built, was struck by lightning two years ago, it started a fire on our property. It took almost 30 minutes for the El Dorado Hills fire department to respond. Thankfully, we were able to contain the fire ourselves and with help of the Amador Fire Protection District who responded before our own department.

Imagine what would happen if the propane generator used by the tower malfunctioned. There would be no warning like the crack of lightning and rolling thunder that warned us of the starting fire. In a rural area such as ours, in fire season, with dry conditions, and the winds we get sweeping through our area, it would be almost inevitable that a wildfire with the size and destruction of the one that just occurred in Napa County because of unmanned utilities owned by PG&E would ensue.



1/29/2018 Edcgov.us Mail - Deny the proposed conditional use permit for AT&T Cell Tower Site 5 on parcel APN #087-181-10 – 7160 Dragon Point Road

For all of these reasons **we object** to the proposal to build a tower on 7160 Dragon Point Road and we therefore **ask that you vote to deny** the conditional use permit.

Sincerely,

Eric and Amy Haydt  
Property owners and residents of:  
7180 Dragon Point Road  
Shingle Springs, CA 95682



PC-2-8-18  
# 3  
Planning Department <planning@edcgov.us>

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**Cell Tower Site 5 on parcel APN 087-181-10 Dragon Point Rd. - (SEE CORRECTED ADDRESS FOR OUR LATROBE CA PROPERTY)**

1 message

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parksr4me@aol.com <parksr4me@aol.com>

Mon, Jan 29, 2018 at 12:22 AM

To: planning@edcgov.us, Gary.miller@edcgov.us, bostwo@edcgov.us, brad@vaminvest.com, sandellp@hotmail.com

William & Lorena Lochhead  
3466 Laketree Dr.  
Fallbrook, CA 92028  
760-723-0074 parksr4me@aol.com

January 28, 2018

RE: our property at - 7101 Dragon Point Rd. APN 087-181-16-100  
Latrobe, CA 95682

We strongly oppose the approval of the cell tower, site 5, to be located at 7160 Dragon Point Rd. APN 087-181-10

The main reasons are:

- 1) Aesthetics – The tower would create a visual blight. This would be directly across from my property and would ruin a beautiful view.
- 2) Decreased property values – Property values would decrease by approximately 20%. Potential buyers are not interested in a property that has the baggage of a cell tower that may affect the future value of their property. Buyers see this risk of the investment as too great.

1/29/2018

Edcgov.us Mail - Cell Tower Site 5 on parcel APN #087-181-10

PC 2-8-18

#3  
2 pages

Planning Department <planning@edcgov.us>



**Cell Tower Site 5 on parcel APN #087-181-10**

1 message

**Julie Murray** <j\_murray@pacbell.net>  
Reply-To: Julie Murray <j\_murray@pacbell.net>  
To: "Planning@edcgov.us" <Planning@edcgov.us>

Mon, Jan 29, 2018 at 5:29 AM

The attached letter pertains to our opposition of the cell tower intent to be installed at this location.

Julie Murray  
Murray Excavating Inc.  
j\_murray@pacbell.net  
530-676-9187 cell 530-305-6995



Marc Murray  
cell 916-798-0108  
530-676-9287



**Dragon Point January 28.pdf**  
437K

January 28, 2018

El Dorado County

Planning Department

Placerville, CA

Dear Planning Department,

We are writing in regards to learning of our neighbor's intent to allow an installation of an AT&T cell tower on their property at 7140 Dragon Point Road. We strongly oppose the approval of this tower due to a number of reasons.

We purchased our property on Lost Horizon Road in 2003 which looks directly at 7140 Dragon Point Road. We purchased the land and moved to it in 2008 to enjoy the quiet rural location away from the commercial and housing expansion in El Dorado Hills. We want to keep it that way. Although all of the parcels surrounding Dragon point on Lost Horizon and Chaparral Road, because of the location of 7140 Dragon Point located on the high ridge, we all look at this parcel. We oppose the cell tower being proposed as it will be extremely close to our parcel and our neighbor's parcels. Installation of this tower will totally destroy the rural attractiveness of our community and negatively impact ours and our neighbor's property values.

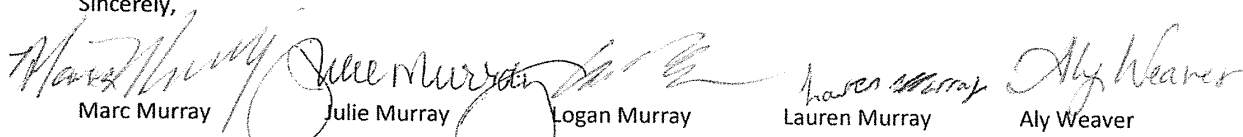
We all live on a private road, the improvements of which were privately paid for by ourselves and our neighbors. Allowing this cell tower will bring additional traffic and wear and tear to our private road. The cell tower traffic will aggravate this situation. One neighbor will gain and take advantage of the equity road maintenance split at the expense of all of the other neighbors.

In addition, we are especially concerned with the potential health risks associated with EMF emitted from this tower as research has been published to truly identify our personal risk from constant exposure to a cell tower. The homes located along Dragon Point are within 2 tenths of a mile from the tower. That is way too close for a commercial operation to be established within our rural location exposing our residents to EMF without our consent.

We are concerned as well with the impact to the environment we live in. Has an environment impact report been published to understand the impact of this tower installation? If not, we request seeing an impact report so we can better understand all of the risks.

Finally, although only one carrier is apparently starting the process, we feel it will not end there. If a tower is installed, we fear it will not be long until Verizon and other carriers also start mounting additional devices on the structure, further increasing the EMF exposure and visibility to the tower. We understand the allure to a potential revenue stream for our neighbor, but at devastation to our rural community along with the potential personal health implications. We are strongly against it.

Sincerely,

  
Marc Murray      Julie Murray      Logan Murray      Lauren Murray      Aly Weaver

8920 Lost Horizon Road, Latrobe, CA 95682 \* [j\\_murray@pacbell.net](mailto:j_murray@pacbell.net) \* 530-676-9187

1/29/2018

Edcgov.us Mail - Planning Commission Document Submission For Conditional Use Permit S17-0016/AT&T CAF4 for Site 6-Zee Estates

PC 2-8-18  
#3  
3 pages



Planning Department <planning@edcgov.us>

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## Planning Commission Document Submission For Conditional Use Permit S17-0016/AT&T CAF4 for Site 6-Zee Estates

1 message

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**Rick Wolfe** <rwolfe@resero.com>

Fri, Jan 26, 2018 at 11:55 PM

To: "james.williams@edcgov.us" <james.williams@edcgov.us>, "planning@edcgov.us" <planning@edcgov.us>

Mr. Williams & Planning Department-

Attached is my statement of support for the Conditional Use Permit S17-0016/AT&T CAF4 for Site 6-Zee Estates. Thank you for your consideration.

Rick Wolfe

Owner, Lotus View Ranch

5081 Salmon Falls Road,

Pilot Hill, CA 95664

916-417-5937



**Planning Commission Statement Conditional Use Permit S17-0016-AT&T CAF4.docx**  
17K

James Williams, Planning Commissioner, District 4  
County of El Dorado Planning Commission  
2850 Fairlane Court  
Placerville, CA 95667  
Email: [james.williams@edcgov.us](mailto:james.williams@edcgov.us)

Reference: Conditional Use Permit S17-0016/AT&T CAF4

Dear James Williams,

I am writing regarding the conditional use permit for construction and operation of the wireless telecommunication facility at Site 6-Zee Estates - Assessor's parcel Number 104-370-24. I am the owner of the named parcel for which the conditional use permit is being requested. When I was initially approached by AT&T about this facility a year ago, I was concerned about the impacts of installing and operating a telecommunication facility on our property. As an engineer, I spent a great deal of time researching and working closely with Epic Wireless, the siting consultant to address my concerns about safety, visual impact, noise, power usage, fire suppression, and RF hazards. Ultimately, I was satisfied that these issues were adequately addressed in their design, and agreed to the installation at a site which would minimize impact to our neighbors to the greatest degree possible while still allowing for the technical needs of AT&T. Since we have intentions in the future to build a cottage near the tower site I wanted to make sure that there would be no adverse effects on my family from the presence of the tower as well.

I was informed by Epic that this tower was to be part of a federal grant program to improve connectivity to rural communities across the country. Because of this we believe it to be a worthwhile use of the land for benefit of the local community. The area where the tower site is proposed currently lacks connectivity, and this tower would significantly improve connectivity to the local community. I was also given the sense that whether or not we agreed to host the tower, that a tower would be installed somewhere in the immediate vicinity. By agreeing to host the site, I have been able to have some control over the placement, and make other requests aimed at minimizing the visual impact to as many neighbors as possible. The site that was chosen was a compromise that simultaneously met the needs for the AT&Ts wireless coverage footprint, was close to necessary utilities (power and fiber) and would minimize visual impact to most of homes in the neighborhood. Of course, it is impossible to completely hide a facility such as this, but by locating it away from the main thoroughfare, on the downhill slope facing the interior of our property, hidden by trees, and requiring the tower to be fully camouflaged, I believe that Epic Wireless has minimized the visual impact to the greatest extent possible.

In talking with neighbors and our siting consultant, I learned that several of my neighbors were interested in having the tower placed on their property. This suggested to me that even if this application was not being made for the installation on our parcel, another application would likely have

been made for a neighboring parcel. As a result, it seems that a vote by the commission to accept or reject this application should consider whether either there is a flaw in the placement of this specific tower or whether the commission rejects out of hand the notion of placing a tower in this community generally. Given my investigation I do believe there are any flaws to this specific site.

Our ranch, of which this parcel is part of, comprises nearly 1100 acres of unspoiled land that provides a habitat for wildlife and a buffer from civilization, affording peace and quiet to our neighbors, as well as a place to occasionally ride horses, hike, and cut firewood. While our neighbors enjoy the benefit of this quiet and serene setting, there are significant costs to us associated with the holding of such a piece of property in this undeveloped state. The income from this tower, while modest, would help to offset some of the ongoing costs of ownership of our ranch.

On a personal note, I have a deep connection with these lands being a native of this area, and grew up exploring its hills, creeks and oak woodlands. While it saddens me at times to witness the development and influx of people from our cities, and the resultant need for infrastructure such as wireless towers, there is little that I can do in response other than protecting and preserving the property that I am fortunate to have. In offering to host this wireless site, I have been able to influence the design and siting to minimize the visual and environmental impact. The modest compensation we receive from the tower lease may help to pay some of the costs associated with preserving these historic rangelands. In summary, I respectfully urge the Commission to approve this use permit as proposed because I feel it is a necessary and inevitable infrastructure expansion to support the needs of our community as it grows. I have worked closely with Epic Wireless in attempting to find an optimal site that considers both functionality and esthetics.

Thank you for your consideration; I plan to attend the meeting and would be happy to address any of your questions that I can at that time.

Respectfully,  
Rick Wolfe  
Owner, Lotus View Ranch