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THEORETICAL APPROACH TO THE EFFECTS OF
EXTREMELY LOW FREQUENCY ELECTROMAGNETIC
FIELDS ON PHYSARUM POLYCEPHALUM

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MASTER

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ABSTRACT

Absolute reaction rate theory is applied to explain the increased time between successive synchronous mitotic division of the slime mold Physarum polycephalum when exposed to extremely low frequency electromagnetic fields. By ignoring the transition period from the normal (control) mitotic cycle to the lengthened mitotic cycle of exposed P. polycephalum, the change in the activation energy is established. This activation energy is in the range of enzyme-catalyzed reactions. The influence of changing frequency on the above transition period is considered to complete the model. Suggestions are made for new experimentation.

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THEORETICAL APPROACH TO THE EFFECTS OF EXTREMELY LOW
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I. Introduction

Goodman, Greenebaum and Marron (1976)¹ (GGM) recently reported the effects of extremely low frequency electromagnetic fields (ELF EMF) upon the microplasmodia of the slime mold Physarum polycephalum. They made a comparative study of the time required, after being placed in rich nutrients, for control and cultures exposed to ELF EMF to reach the first, second, and third postfusion nuclear division. For a magnetic field of 2.0 G and an electric field of 0.7 V/m, the time between mitoses for exposed cultures increased by 0.5 to 2.0 hour, as compared to unexposed controls. The mitotic delay displayed some frequency dependency, the onset of this effect occurring earlier for lower frequency EMF. Experimental cultures were exposed to ELF EMF for more than 700 days.

The synchronons mitotic cycle of P. polycephalum is well-known. About one hour before metaphase, the nucleolus swells followed by condensation of the chromosomes. The nucleolus breakup begins about 20 minutes before metaphase. Prometa- meta- ana-, and telophase are all about 5 minutes long and interphase is greater than or equal to 8 hours (Mohberg and Rusch, 1969).² The divided nuclei have many small irregular bodies which fuse over the next 2 hours to form the nucleolus. This fusing process occurs concurrent with the S period. The G₁ phase is less than 10 minutes in length and may not exist (Kessler, 1967).³ DNA begins synthesization in the plasmodium almost immediately after telophase (within 10 minutes) and continues at a high rate for about 2 hours. The synthesization of DNA then decreases to a low level at approximately 3 hours after telophase. The replication of DNA is finished at the end of the S period about 5 hours after metaphase. Cummins and Rusch (1966)⁴ found that DNA synthesis occurred in cycles and protein synthesis was necessary for the start of new cycles. A G₂ period of about 5 to 6 hours is required before the beginning of mitosis (Rausch, et al., 1964).⁵ Mitotic synchrony, a complex mechanism which is

important to the understanding of the theoretical considerations of this paper, will be discussed in detail in the next section.

In this paper, absolute reaction rate theory will be used to develop a model of the effects of ELF EMF upon critical biochemical reactions in the mitotic cycle of the slime mold Physarum polycephalum. In Section II the model is developed for the frequency independent steady-state effects. The consequences of different frequency ELF EMF are investigated in Section III. A number of critical experiments that can greatly help to expand our understanding of the interaction of EMF with biological systems are presented in Section IV.

II. Frequency Independent Theoretical Formulation

GGM exposed P. polycephalum to 75 Hz, 2.0 G, and 0.7 V/m electromagnetic fields (EMF) for more than 700 days and found the times for successive nuclear division in macroplasmodia given in Table I.

Table I

Time (in hours) for Successive Postfusion Nuclear Division

	Without EMF* (control)	With EMF* (75 Hz, 2 G and 0.7 V/m)
Mitosis I	5.2 $\pm$ 0.2	5.2 $\pm$ 0.1
Mitosis II	15.1 $\pm$ 0.2	16.1 $\pm$ 0.1
Mitosis III	25.6 $\pm$ 0.2	28.0 $\pm$ 0.2

*Error bounds are 95% confidence limits.

The steady lengthening of the time between mitotic division in the presence of the ELF EMF suggests an impact upon the mechanisms that regulate mitotic synchrony. These results are for a fixed frequency and ignore the time required for the establishment of the mitotic delay. Similar steady-state results are given for frequencies of 45 and 60 Hz. In this section, we are concerned only with the steady-state results.

In the next section (III) the influence of frequency upon the early transitional period of exposure will be considered.

Careful studies of the factors controlling synchrony have been made by observing the timing of mitosis in coalesced plasmodia from different stages of the mitotic cycle (Mohberg, 1974).⁶ Rusch, et al. (1966),⁷ and Guttess and Guttess (1968),⁸ Murakami and Ohta (1971),⁹ and Chin, et al. (1972)¹⁰ found that fused plasmodia from early and later interphase undergo synchronous mitosis, at a time intermediate between those of the parent cultures. Rusch, et al.,⁷ and Murakami and Ohta⁹ proposed that a stimulator accumulation was responsible for cytoplasmic control of mitosis. Rusch (1970)¹¹ points out that the DNA must be completely replicated before mitosis can occur. During the G₂ period there is growth of the plasmodium and synthesis of substances needed for mitosis. Muldone, et al. (1971)¹² found evidence that the Physarum polycephalum genome might be made of at least 10 replicative units. Each unit may be controlled by a protein which is synthesized at a specific time in the S phase.

Another bit of information that offers an important insight is ultraviolet light delayed mitosis with the strongest effects occurring during the first 30 percent of the interphase period (Devi, et al., 1968).¹³ DNA synthesis was depressed by ultraviolet light. This may be the cause of delayed mitosis.^{11,13}

The time to first mitosis is unaffected by the presences of the ELF EMF. One possible interpretation is that for GGM experiments the G₂ period of Physarum polycephalum is 5.2 hours. Also, the ELF EMF has an effect during the S period that causes a delayed mitotic cycle. The interaction could be with the stimulator (activator) that controls mitotic activity or perhaps at a specific step in the full replication of DNA via a controlling protein (enzyme). The interaction of ELF EMF with the critical biochemical reaction may be analogous to the case of ultraviolet light. UV influence upon DNA synthesis is a strong suggestion that the above may be correct.

Another interpretation is that the normal activator for mitotic activity is suppressed by the ELF EMF. Then another activator, that is not affected by the ELF EMF but that is synthesized at a slower rate, initiates the mitotic cycle. Both of the above interpretations are developed within a common framework below.

Let the concentration of the stimulator be n and assume that it increases via a first-order reaction, then

$$\frac{dn}{dt} = k_r n \quad (1)$$

or

$$n = n_o e^{k_r t} \quad (2)$$

where n_o is the concentration at $t = 0$. Higher order reactions may be involved in the production of the stimulator/activator. Because of the limited nature of the data being analyzed, the simplifying assumption of first order reaction is made. Of course, enzyme reactions can be first, second, or zero order. The variation of the velocity of the reaction with the concentration of the enzyme and with the nature and concentration of the reactants is not known. By considering first order reactions, we can help to narrow the actual choice of the specific reaction that is being altered by ELF EMF effects.

Mitosis occurs when the concentration of n is built up to a critical level n_c . The amount of time required is

$$t_c = \frac{1}{k_r} \ln \frac{n_c}{n_o} \quad (3)$$

Without knowledge of which biochemical compound is the important one and careful measurements of its concentration as a function of time in the

mitotic cycle, n_o , n_c , or k_r is not known. However, there is information about the amount of time required for DNA replication. Although not critical for the development of this theory, let us assume that the stimulator follows the development of DNA in that at the end of the S period it has doubled its concentration, i.e., $n_c/n_o \equiv 2$. The time required can be estimated as the period of the S period. From the data in Table I, the critical time for the second mitotic division of the control is

$$\begin{aligned}
 t_{IIS} &= (\text{time to mitosis II} - \\
 &\quad \text{time in } G_2 \text{ phase of mitosis II}) - \\
 &\quad \text{time in } G_2 \text{ phase of mitosis I} \\
 &= [(15.1 \pm 0.2) - (5.2 \pm 0.2)] - (5.2 \pm 0.2) \\
 &= 7 \pm 0.35 \text{ hr.}
 \end{aligned}$$

Similarly, the other critical times can be calculated and the results are given in Table II.

Table II

Critical Time for Development of Critical
Concentration of Stimulator

Mitotic Division	Critical Time (Hr)	
	Without ELF EMF	With ELF EMF
II	4.7 ± 0.35	5.7 ± 0.17
III	5.3 ± 0.35	6.7 ± 0.24

The reaction rates k_r are calculated using these critical times and $n_c/n_o = 2$ and are shown in Figure 1 as a function of the time for

Figure 1. The reaction rate constant k for the production of the mitotic stimulator/activator as a function of time t_m into the synchronous mitotic cycles of the plasmodia of the slime mold, *P. polcephalum*. The functional form for the curves shown for k in the control (○) and for exposure to ELF EMF (●) is $k = k_0 + k_1 t_m$. As the slopes are the same (i.e., k_1 is the same) for both lines, the implication is that the influence of the ELF EMF is upon k_0 .

successive postfusion nuclear division t_m . From an inspection of Figure 1, the simplest functional forms for the reaction rate constants* are

$$k_r = k_o + k_1 t_m \quad \text{w/o EMF (control)} \quad (4)$$

and

$$k' = k'_o + k'_1 t_m \quad \text{w/ EMF} \quad (5)$$

Because the slopes of the lines are the same within the accuracy of the data†, assume that

$$k_1 = k'_1 = 0.00224 \text{ (Hr}^{-2}\text{)} \quad .$$

This implies that the effect of the ELF EMF is upon the k_o term. For the straight lines of Figure 1, note that

$$k_o = 0.249 \text{ Hr}^{-1} \quad (\text{control})$$

and

$$k'_o = 0.212 \text{ Hr}^{-1} \quad (\text{with ELF EMF}) \quad .$$

The nature of the change in the reaction rates suggests the application of absolute reaction rate theory. The rate constant is given by

$$k = f \frac{k_B T}{h} K^\ddagger \quad (6)$$

*This form is valid only within the range of the GGM experiment. The functional form for longer t_m could be different. Within this small range this form is a good approximation.

†Actually to within 4.4%.

where: f is the probability that a reaction will occur, i.e., f is the transmission coefficient and is usually of order unity (Johnson, Eyring and Stover, 1974);¹⁴ $K^\ddagger$ is the quasi-equilibrium constant for the transition and the normal states; k_B is Boltzmann's constant; T is the temperature in degrees Kelvin; and h is Planck's constant. The quasi-equilibrium constant can be written as

$$K^\ddagger = \exp\left(\frac{-\Delta F^\ddagger}{RT}\right) \quad (7)$$

where R is the universal gas constant. The term $\Delta F^\ddagger$ is the activation energy -- that is, the energy required to change the reactants from the initial to the transition state. The activation energy can be written as a sum of contributions from normal environmental factors, $\Delta F^{(c)}$, from electric fields, $\Delta F^{(e)}$, and from magnetic fields, $\Delta F^{(m)}$, (Brannen and Wayland, 1976; Wayland, 1976),^{15,16}

$$\begin{aligned} \Delta F^\ddagger &= \Delta F^{(c)} + \Delta F^{(e)} + \Delta F^{(m)} \\ &= \Delta F^{(c)} + \Delta F^{(em)} \end{aligned} \quad (8)$$

The assumption is made that the electromagnetic field has introduced a change in the activation energy of magnitude $\Delta F^{(em)}$ for the critical molecules. If the k_o term is given by absolute reaction rate theory, then

$$k_o = f \frac{k_B T}{h} \exp(-\Delta F^{(c)}/RT) \quad (9)$$

and

$$k'_o = f \frac{k_B T}{h} \exp[-(\Delta F^{(c)} + \Delta F^{(em)})/RT] \quad (10)$$

The effect of the $\Delta F^{(em)}$ term is given by

$$\Delta F^{(em)} = RT \ln (k_o/k'_o) \quad (11)$$

or for a more usable dimensionless parameter

$$A = \frac{\Delta F^{(em)}}{RT} = \ln (k_o/k'_o) \quad (12)$$

For the values of k_o and k'_o given above, $A = 0.161$. A is about 0.4% of $\Delta F^{(c)}/RT$. Using the data given in GGM, the values of A can be found at 45 and 60 Hz. To within experimental error, the values of A are all the same.

The magnitude of the free-energy difference per mole between products and reactants can be estimated from the reaction rate k_o . The value is about 23 Kcal/mole. Without more information, the specific molecules cannot be identified. However, this is in the range of enzyme-catalyzed reactions.

III. Frequency Dependent Theoretical Formulation

In the preceding analysis, the assumption is that the changes in the mitotic cycle occur simultaneously with the introduction of the ELF EMF and are equal to the average throughout exposure. GGM found that this was not the complete description of what occurred. The changes in the lengthening of the mitotic cycle are dependent on frequency and duration of exposure to the ELF EMF. For example, the lower frequency, 45 Hz, caused the effect to occur earlier after the ELF EMF was introduced than in the case of 75 Hz. In order to estimate the effect of frequency, the data given in GGM was grouped into 25-day bins for the frequencies of 45, 60, and 75 Hz, and 2.0 G, 0.7 V/m EMF. For each data point a rate constant was found using the equation

$$k' = \frac{1}{t_{IIS}} \ln 2.$$

Then the values of k'_o were found using Equation 5.

The effect of the EMF on k'_0 can be considered as an alteration of a specific type of molecule or sites within molecules. The removal of the unaltered molecules causes the time dependency (in terms of the exposure time to ELF EMF, t_{em}) in the delayed mitotic cycle. The probability of a molecule not being changed can be shown to be¹⁴

$$q = \exp(-k_{em} t_{em}) \quad (13)$$

where k_{em} is the rate constant for the destructive reaction. At all frequencies, as t_{em} approaches zero, k'_0 approaches k_0 . This implies that k_0 is a constant which does not vary with frequency. As the number of molecules that can behave in a normal manner is reduced, the probability that a reaction will occur should be proportional to the remaining number of molecules so that

$$k'_0 = k_0 q = k_0 e^{-k_{em} t_{em}} \quad (14)$$

when Equation 14 is fitted, by a least square routine, to the data for k'_0 , the values of k_{em} given in Table III were found.

Table III

Values of k_{em} as a Function of Frequency for all
Electromagnetic Field of 2.0 G and 0.7 V/m

Frequency (Hz)	k_{em} (day ⁻¹)
45	4.8×10^{-3}
60	4.5×10^{-3}
75	1.9×10^{-3}

Note that for lower frequencies, there is a larger effect upon k_{em} which implies a stronger coupling of the ELF EMF to the P. polycephalum as the frequency decreases. However, care must be exercised in using these values of k_{em} because of their large uncertainties.

The reaction rate k_{em} is very difficult to estimate. There is not enough information to find the frequency dependency of the activation energy for k_{em} . It is formally possible to give the functional form by absolute reaction rate theory^{15,16} as

$$k_{em} = f_{em} \frac{k_B T}{h} \exp(aE^2/RT) \quad (15)$$

where f_{em} is the transmission coefficient for the electromagnetic altered reaction. The sign of a , the coupling coefficient, is chosen to reflect the requirement that as the electric field E increases, there is a decrease in k'_0 which in turn increases the time t'_{IIS} . In addition, f_{em} must approach zero as E goes to zero. For small changes in E , the dominant dependence of k_{em} should be in the exponential which suggests an approximate scaling relation for a new electric field strength E , as

$$k_{em}(E=E_1) = k_{em}(E=.7 \text{ V/m}) \exp\left[\frac{a}{RT} (E_1^2 - 0.49)\right].$$

IV. Discussion

A model based upon absolute reaction theory to explain the delays induced by ELF EMF in the mitotic cycle of Physarum polycephalum has been developed. The delay is introduced by an alteration of a molecule which must reach a critical concentration before mitotic activity begins. The precise molecule (or molecules) has not as yet been identified. In a comparison to experimental results, the reaction rate constants were found for the overall mitotic cycle activity as a function of the time for successive postfusion nuclear divisions. In the steady-state approximation of an instantaneously introduced delay in the time spent in the S period of mitosis, the rate constant for the buildup of the mitotic stimulating molecules was found to be given by

$$k'_0 = k_0 \exp(-\Delta F^{(em)}/RT),$$

where the alteration of the critical molecule is represented as a change in the activation energy term, $\Delta F^{(em)}$. This change is not permanent as can be seen from the evidence presented by GGM of the slow return to the normal mitotic activity after the removal of the ELF EMF.

The basic concept of the above model was expanded to include the observed frequency dependency in the delay to the time of postfusion nuclear division. The assumption was made that the rate constant for the altered biochemical reaction for mitotic activity was dependent upon the number of stimulator molecules which were unaltered in exposure to the ELF EMF. When the Physarum polycephalum has been exposed only a short time, there are more of the unaltered stimulator molecules around to initiate the mitotic activity. The changes in the delay in the time spent in the S period are less than for the steady-state. In the longer exposure time, more altered stimulator molecules are produced and steady-state approximation is reached. For weak ELF EMF, the effects upon the stimulator molecules should reach an equilibrium if the repair mechanism is of the same efficiency as the damage mechanism. This was not investigated because of a lack of data in the critical first 100 days of exposure to the ELF EMF.

The specific experimental detail that is needed to develop a coherent theory without the use of phenomenological parameters does not exist for the interaction of electromagnetic fields with Physarum polycephalum. The pioneering experiments of GGM offer clues to what the mechanisms might be. The use of absolute reaction rate theory allows a model to be developed which can be expanded into a theory that will explain the interaction processes. To do this requires the expansion of experimental efforts to new areas. Some suggestions of profitable measurements are given below. It is fully understood that to suggest experiments is easy. The suggestions offered are in the hope of establishing a cooperative relationship between experimental and theoretical activities.

Perhaps the most useful immediate experiments would be to make measurements, with high statistical accuracy, of the changes in the synchronous

mitotic cycle of Physarum polycephalum during the first 100 days of exposure to electromagnetic fields. The electric and magnetic fields should be varied in the range of 1 to 100 V/m and 1 to 100 G, respectively. The frequency should be decreased into the 10 Hz range and increased into the 100 MHz range. By careful inspection of the DNA replication process, specific changes of chemical concentrations of critical RNA and protein molecules during the mitotic cycle for different exposure conditions would be invaluable. These experiments would help in the identification of the stimulator molecules and their interaction with EMF.

V. Conclusion

Absolute reaction rate theory has been used to develop a model of the interaction of ELF EMF with the slime mold Physarum polycephalum. The effects upon possible enzyme-catalyzed reactions indicate the need for future experimentation.

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