Journal of Scientific Exploration, Vol. 14, No. 2, pp. 217–231, 2000 0892-3310/00

Energy, Fitness, and Information-Augmented Electromagnetic Fields in *Drosophila melanogaster*

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Abstract—Exposure of developing larvae to a few specific electromagnetic fields (EMFs) and information-augmented EMFs modified (a) the expression of larval development time, a genetically based trait relevant to development and whole organism fitness, and (b) a measure of energy metabolism, the [adenine triphosphate/adenine diphosphate] ([ATP]/[ADP]) ratio in isofemale strains of *Drosophila melanogaster*. The study represents a compilation of approximately 10,000 larvae and 7,000 adults counted. The specific EM frequencies used in this study, 5.0, 7.3, 8.0, and 9.3 MHz at output power levels in the approximately 1 microwatt range, were produced by two small electronic devices of physically identical nature, but one was intentionally imprinted with specific information. Exposure periods varied from 4 hours to one life cycle. Larval development time was significantly shortened (approximately 10%) and the [ATP]/[ADP] ratio significantly increased (approximately 30%) in a Faraday cage without the EMFs compared to a Faraday cage with the specific EMFs. The Faraday cage represents a shielded environment that facilitates exposure to both fewer and specific electromagnetic frequencies. Larval development time results for development in the laboratory environment, which represents exposure to background EMFs of various frequencies, were intermediate. The information-augmented EMFs also gave intermediate results. Overall, there were no significant effects observed for the other measured fitness components—third instar larval weight, adult survival, and surviving adult weight. We discuss a thermodynamic model to account for our results and general bioelectromagnetic effects and attribute the change in development time to EMFs modifying electron transport chain activity and the [ATP]/[ADP] ratio via the influence of the EMF/magnetic vector potential upon electron availability and nicotinamide adenine dinucleotide levels.

Keywords: fitness experiments—[ATP]/[ADP] ratio—Faraday cages—information-augmented and normal EMFs, theoretical models

1. Introduction

Bioelectromagnetic studies have advanced considerably in the past decade (Goodman, Greenbaum, and Marron, 1995). For example, studies of nonthermal effects on cells of the immune system from exposure to electromagnetic fields (EMFs) in the extremely low frequency range (<300 Hz) indicate that stimulatory, inhibitory, and no field exposure effects exist even for identical field parameters. The results depend upon the degree of cellular activation and the physical and biochemical boundary conditions experienced (Eichwald and Walleczek, 1996). Furthermore, low frequency EMFs influence specific RNA transcripts in human cells and transcription in *Drosophila melanogaster* cells (Goodman, Wei, and Henderson, 1989; Goodman *et al.,* 1992), and Ho *et al.* (1992) have shown that weak static magnetic fields cause abnormalities in first instar larvae of *D. melanogaster*.

A number of models have been proposed to account for EMF effects on biological systems. Ho *et al.* (1992) suggested that the weak static magnetic fields they studied must affect some cooperative process involved in pattern determination during critical stages of early *Drosophila* development. On the basis of the immune cell experiments, Eichwald and Walleczek (1996) suggested that external EMFs interact with cellular systems at the level of intracellular signal transduction pathways, specifically, Ca-signaling processes.

Nossol, Buse, and Silny (1993) reported magnetic field influences on the *in vitro* redox activity of cytochrome oxidase activity. Additionally, weak EMFs stimulate adenine triphosphate (ATP) synthesis and alter Na,K-ATPase activity (Blank, Soo, and Papstein, 1995; Lei and Berg, 1998). Thus, magnetic fields may influence cellular energy metabolism, and Menendez (1996) has suggested that an electromagnetic coupling process may explain the proton translocation mechanism in cellular energy transfer.

A significant association has been observed between stress, larval development time and aspects of energy metabolism, the cofactor nicotinamide adenine dinucleotide (NAD), and the [ATP]/[ADP] ratio in *D. melanogaster* (Kohane, 1988, 1994). In the present paper, we use the theoretical and experimental approach presented in these earlier papers and expand it to incorporate the intention-imprinted electronic device (IIED) techniques of Dibble and Tiller (1999) and investigate the hypothesis that both normal and information-augmented EMFs may influence fitness and energy metabolism.

We study fitness and the [ATP]/[ADP] ratio under nonstressful nutrient conditions, in the presence and absence of small electronic devices that produce EMFs of frequencies much higher than those used in previous studies. We assess these frequency effects using exposure periods from 4 hours to one life cycle in order to detect EMF effects that may not be observed at more conservative levels of EMF frequency and exposure period.

Our results indicate that larval development time is significantly shortened and the [ATP]/[ADP] ratio significantly increased in a Faraday cage without the devices compared to a Faraday cage with the devices. The Faraday cage

represents a shielded environment with respect to electromagnetic (EM) radiation and facilitates exposure to fewer frequencies in the absence of devices and specific EM frequencies in the presence of devices. In addition, the IIEDs gave significantly better results on larval development time than did the unimprinted control devices. Overall, there are no significant effects for the other fitness components assessed, third instar larval weight, adult survival, and surviving adult weight.

Thus, a reduction in exposure to EMFs increases one component of fitness, suggesting that EMFs may act as a biological stress (Goodman, Greenbaum, and Marron, 1995; Smith, 1996). Although we acknowledge the fact that EMFs may modify the larval environment (*e.g.,* the food), we attribute the effects of the specific EMFs in this study to the modified [ATP]/[ADP] ratio as a consequence of altered NAD levels, electron availability, and electron transport chain activity. Finally, we discuss a thermodynamic model that may rationalize our observed fitness and energy changes and general bioelectromagnetic effects.

2. Experimental Methods

(a) Strains

We studied larvae obtained from two isofemale strains, Strains 1 and 2 of Kohane (1994). Nonstressful food was used for strain culture and experiments, and the food composition was as follows: 36 g agar, 108 g sugar, 72 g dry yeast, 10 ml propionic acid, and 24 ml nipagen in 2,000 ml water (Kohane, 1987). Separate constant temperature rooms (18ºC, 55% relative humidity) were used for (a) device storage, (b) strain culture and unexposed adult culture, and (c) experiments.

(b) Faraday Cages

A standard Faraday cage consisted of a copper mesh screen enclosing a certain spatial volume. It is electrically grounded so the EM waves of wavelength larger than the mesh size, which impinge on the screen, will leak off to ground and only minimally penetrate to the interior space. Thus, the interior space has a greatly reduced EM integrated power density in the wavelength range larger than the copper mesh spacing. The one layer of copper mesh cages (dimensions: $40 \times 40 \times 30$ cm) used here can be expected to reduce the EM field strength by a factor of approximately 10.

(c) Electronic Devices

Our experiments used two electronic devices in order to assess exposure to multiple frequencies and a single frequency as follows: (d1), a triple oscillator device producing frequencies of 5.0, 8.0, and 9.3 MHz; and (d2), a single oscillator device, producing a frequency of 7.3 MHz. Additionally, we studied two categories of EMFs produced by these devices. The first category

involved devices (d1, o) and (d2, o), which had not been exposed to human informational influences. The second category involved devices (d1, j) and (d2, j), which had been exposed to human informational influences (see below). Thus, (d1, o) and (d1, j) and (d2, o) and (d2, j) constituted physically identical pairs of devices that differed only in the fact that one of each pair, (d1, j) and (d2, j), respectively, had been exposed to the human informational influence. The devices were individually wrapped in aluminum foil and stored in separate Faraday cages and were fabricated to be identical to those produced by Clarus Corporation (La Costa, CA). The triple oscillator device was powered by line voltage to 9V DC, and the single oscillator device was previously charged for 24 h by line voltage to 9 V DC and used with battery power.

(d) Intentions

The actual imprinting procedure was as follows: (a) The device was placed along with its current transformer on a table around which the imprinters sit; (b) Four people (two men plus two women) who were all accomplished meditators, coherent, inner-self managed and readily capable of entering an ordered mode of heart function (Tiller, 1997) and sustaining it for an extended period of time, sat around the table ready to enter a deep meditative state; (c) A signal was then given to enter such an internal state, to cleanse the environment and create a sacred space for the intention, then, a signal was given by one of the four to put attention on the tabletop objects and begin a mental cleansing process to erase any prior imprints from the device; (d) After 3 or 4 minutes, another signal was given to begin focusing on the specific prearranged intention statement for about 10–15 minutes; (e) Next, a final signal was given to shift focus to a closing intention designed to seal off the imprint into the device and minimize the leakage of the essential energy/information from the devices. This completed the process, so the four people withdrew from the meditative state and returned to their normal state of consciousness.

The specific intention was "to activate the indwelling consciousness of the device (d, j) so as to increase the concentration of NAD plus the activity of the available enzymes, dehydrogenases and ATP synthase in the mitochondria so that production of ATP is significantly increased relative to that produced in the unimprinted device (d, o)." This chemical transformation process in the cells of the fruit fly larvae was expected to significantly influence their fitness, which would manifest itself via a reduced larval development time for these larvae because they have a larger pool of ATP to work with (Kohane, 1994).

(e) Fitness

We conducted four similar experiments over a 6-month period and assessed EMF effects on the fitness of the strains using the above devices and different exposure periods. The experiments are summarized in Tables 1 and 2. We measured fitness at 18ºC using the procedures given in Kohane (1988; 1994).

TABLE 1
Summary of Fitness Experiments

Experiment	Strain[a]	Device[b]	Treatment and replicate number				Exposure period[e]	Date
			C	F[c]	d, j[d]	d, o[d]		
1	2	d1	16	15	15	16	4 hours	February 1997
2	1	d2	16	15	15	16	4 hours	February 1997
3	1	d1	15	14	15	15	4 days	July 1997
4[f,g]	1	d1	15	15	16	16	Life cycle plus 4 days	July 1997

Note: Fitness components assessed were larval development time, adult survival, and surviving adult weight.

[a] Strain 1 and strain 2 refer to two isofemale strains.

[b] (d1) refers to a triple oscillator device producing frequencies of 5.0, 8.0, and 9.3 MHz; and (d2) to a single oscillator device, producing a frequency of 7.3 MHz. The output power of the devices at the exposure distances is expected to be less than 1 μW. Categories of EMFs produced by these devices were as follows: (a) (d, o), devices which had not been exposed to human informational influences; (b) (d, j), devices which had been exposed to human informational influences—an intention concerned with significantly increasing the [ATP]/[ADP] ratio and decreasing larval development time.

[c] (F) refers to culture in a Faraday cage without a device, and (C) refers to culture in the laboratory environment.

[d] (d, o) and (d, j) refer to culture in a Faraday cage with a single device.

[e] Experiments were conducted at 18ºC. Experimental vial cultures involved 30 larvae (0–4 h old) transferred to a single vial containing nonstressful food.

[f] For Experiments 1–3, larvae were derived from unexposed adults, and for Experiment 4, larvae were derived from exposed adults.

[g] The [ATP]/[ADP] ratio and third instar larval weight were assessed in Experiment 4 (see Materials and Methods).

Experiments were conducted at 18ºC since development time differences have been detected at this temperature for the strains studied here (Kohane, 1994).

The treatments investigated in the experiments were as follows: (a) (C)—culture in the random EMF environment of the laboratory; (b) (F)—culture in the relatively reduced EMF environment of the Faraday cage without a device; (c) (d1, o) and (d2, o)—culture in the relatively reduced EMF environment of the Faraday cage in the presence of a device that had not been associated with human informational influences; and (d) (d1, j) and (d2, j)—culture in the relatively reduced EMF environment of the Faraday cage in the presence of a device that had been associated with human informational influences.

A single replicate involved 30 larvae (0–4 h old) transferred to a single vial containing nonstressful food (see above). For each experiment, all vials were established within a 3-hour period. Vials were transferred to Faraday cages and the cages were placed, at the same time, immediately next to each other on the same bench in a constant temperature room. Treatment (C) involved vials concurrently transferred to a tray, which was placed on the lid of treatment (F). We used vial cultures as a thermocouple and could not detect temperature variation between treatments in each experiment.

Exposure of vials to devices in Faraday cages was achieved as follows: The

TABLE 2
Results for Each Experimental Condition Described in Table 1

	Treatment			
Variable	C	F	j	o
Experiment[a]				
1	16.276 (0.170)	15.503 (0.097)	16.080 (0.351)	16.838 (0.145)
2	15.504 (0.097)	15.238 (0.111)	16.047 (0.336)	16.837 (0.141)
3	15.964 (0.315)	14.703 (0.248)	15.739 (0.151)	16.778 (0.152)
4	16.507 (0.087)	15.348 (0.106)	16.228 (0.197)	17.323 (0.186)
Experiment[b]				
1	0.917 (0.833) (0.933)	0.900 (0.842) (0.915)	0.933 (0.800) (0.933)	0.850 (0.767) (0.900)
2	0.900 (0.842) (0.915)	0.917 (0.833) (0.933)	0.933 (0.833) (0.933)	0.850 (0.779) (0.900)
3	0.933 (0.830) (0.938)	0.933 (0.871) (0.941)	0.867 (0.778) (0.917)	0.933 (0.867) (0.958)
4	0.867 (0.833) (0.967)	0.933 (0.879) (0.969)	0.933 (0.867) (0.967)	0.950 (0.890) (0.975)
Experiment[c]				
1	1.140 (0.049)	1.148 (0.057)	1.124 (0.036)	1.147 (0.056)
2	1.155 (0.045)	1.158 (0.089)	1.125 (0.036)	1.136 (0.063)
3	1.267 (0.099)	1.360 (0.083)	1.349 (0.056)	1.352 (0.039)
4	1.254 (0.047)	1.388 (0.037)	1.249 (0.066)	1.238 (0.085)
Experiment 4[d]				
	1.478 (0.157)	1.524 (0.138)	1.575 (0.277)	1.579 (0.163)
Assay[e]				
NAD	1.305 (0.046)	1.630 (0.077)	1.273 (0.039)	1.177 (0.043)
Pure water	0.486 (0.035)	0.482 (0.046)	0.424 (0.042)	0.368 (0.024)

[a] Larval development time ($T_{½}$, in days). Means and standard deviations are given in parentheses.
[b] Adult survival given as proportions for an input of 30 larvae. Medians and 25 and 75 percentiles are shown in parentheses for adult survival since the data distribution curves for each batch were not normal. The 25 percentiles are given before the 75 percentiles.
[c] Surviving adult weight (mg). Means and standard deviations are given in parentheses.
[d] Larval weight in mg. Means and standard deviations are given in parentheses.
[e] [ATP]/[ADP] ratio. Means and standard deviations are given in parentheses.

respective device was placed in the center of a Faraday cage and vials/bottles were transferred to cages and placed around the perimeter. The device was then removed at the end of a specific time period and larval development proceeded until adults eclosed. The device was approximately 15 cm from the vials/bottles on average, where the output power of the devices in their specific frequency ranges is expected to be less than 1 microwatt.

Vials were monitored daily and surviving adults were collected and weighed. Surviving adult weight was calculated for each vial as the weight of the number of flies surviving divided by the number of flies surviving and is given in mg. Larval development time is given as $T_{½}$, the time taken for half of the surviving adults to emerge.

In Experiments 1–3 (Table 1), larvae were derived from unexposed adults. In Experiment 4 (Table 1), larvae were derived from exposed adults of Strain 1 as follows: Replicate bottle cultures were exposed to all treatments—(C), (F) (d1, j), (d1, o) for one life cycle. Emerging adults were synchronously collected from the respective cultures and used to generate larvae for experimental vials. These were then exposed to the same treatment as the parents using a 4-day exposure period for devices.

(f) Energy Levels During Larval Development

The energy assay follows Kohane (1994) and primarily detects electron transport chain activity. We measured the [ATP]/[ADP] ratio in third instar larval homogenates where the glucose-hexokinase reaction, ADP-regenerating reactions, and glycolysis and the citric acid cycle were not functional or were severely impaired (Kohane, 1994). Supplemental dH_2O does not alter the [ATP]/[ADP] ratio in these homogenates. Supplemental 0.01M NAD increases the [ATP]/[ADP] ratio as a consequence of altered electron transport chain activity (Kohane, 1994). Kohane (1994) stored larval homogenates supplemented with NAD and dH_2O on ice for a reaction time of 7 minutes to facilitate metabolic activity, prior to determination of the [ATP]/[ADP] ratio.

In order to assess treatment, fitness, and the [ATP]/[ADP] ratio in the same experiment, we established experimental vials for energy assays concurrently with Experiment 4 of Table 1. We also adopted a more conservative approach and stored the homogenates, supplemented with NAD and dH_2O, in their respective treatments for a longer reaction time of 40 minutes. Thus, the assay assesses *in vivo* and *in vitro* effects of EMFs on the [ATP]/[ADP] ratio (Kohane, 1994). We avoided age-dependent effects on energy metabolism by preparing larval homogenates on the same day as follows: 15 third instar larvae were collected, transferred to microcentrifuge tubes, weighed (mg), and homogenized in 250 μl ice-cold dH_2O to which 250 μl of ice-cold 0.01 M NAD or dH_2O were immediately added.

The solutions were mixed and transferred to each treatment for a reaction time of 40 minutes to facilitate metabolic activity. The microcentrifuge tubes were then freeze-clamped in liquid nitrogen. ATP, ADP, and AMP (adenine monophosophate) were extracted using 4.2 M formic acid and 4.2 M ammonium hydroxide (Kohane, 1994). They were quantified using an automated Isco high performance liquid chromatography (HPLC) apparatus, a Vydac column (302IC4.6), and a preprogrammed gradient from 0.025 M sodium phosphate monobasic pH 2.8 to 0.5 M sodium phosphate monobasic pH 2.8 (modified from Kohane, 1994). There were eight replicates per treatment for both supplemental NAD and dH_2O. All extractions were completed within a 4-hour period. Replicates were stored at 2–4ºC and assayed in random order during a 3-day period.

3. Results

(a) EMFs and Fitness

We have used visual inspection of Figure 1 to assess the homogeneity of the treatment results across experiments. Considering larval development time, (C) and (F) were not homogeneous and rankings for (C) were as follows: (C2) < (C3) ≤ (C1) < (C4). The rankings for (F) were as follows: (F3) < (F2) ≤ (F4) ≤ (F1). (d, j) was essentially homogeneous. Considering (d, o), the rankings were as follows: (o1) = (o2) = (o3) < (o4). All treatments appeared to be homogeneous across experiments for adult survival. Considering surviving adult weight, treatments were not homogenous across experiments. Rankings were as follows:

$$(C) - (C1) \leq (C2) < (C4) \leq (C3);$$
$$(F) - (F1) = (F2) < (F3) = (F4);$$
$$(d, j) - (j1) = (j2) < (j4) < (j3);$$

and

$$(d, o) - (o1) = (o\ 2) < (o4) < (o3).$$

Finally, larval weight was only assessed in Experiment 4.

On the basis of the observed heterogeneity, we have considered treatment comparisons for each experiment and have used visual inspection of Figure 1 to assess these comparisons. In our experiments, comparisons between (d, o) and (d, j) indicate the effects of exposure to the two categories of EMFs—the intention effect. The presence of (d, j) significantly decreased larval development time in comparison to (d, o) in all experiments. We did not observe a significant device effect for adult survival, surviving adult weight, and larval weight.

Comparisons between (C) and (F) indicate the effect of a reduction in exposure to random EMFs—the Faraday cage effect. The presence of the Faraday cage significantly decreased larval development time in comparison to (C) in all experiments. We did not observe a significant Faraday cage effect for adult survival and larval weight. Considering surviving adult weight, a significantly higher weight was observed in Experiments 3 and 4 for (F) in comparison to (C).

Comparisons between (F) and (d, o) and (d, j) indicate the effects of the addition of specific EMFs to the Faraday cage environment—the oscillator effect. In all experiments, the presence of the Faraday cage without a device (F) significantly decreased development time in comparison to both (d, j) and (d, o). As noted above, the effect of (d, j) was to decrease development time in comparison to (d, o). We did not detect any significant comparisons for both adult survival and larval weight. Considering surviving adult weight, no treatment effects were observed for Experiments 1, 2, and 3. However, in Experiment 4 the treatment rankings were as follows: (F) > (d, j) = (d, o).

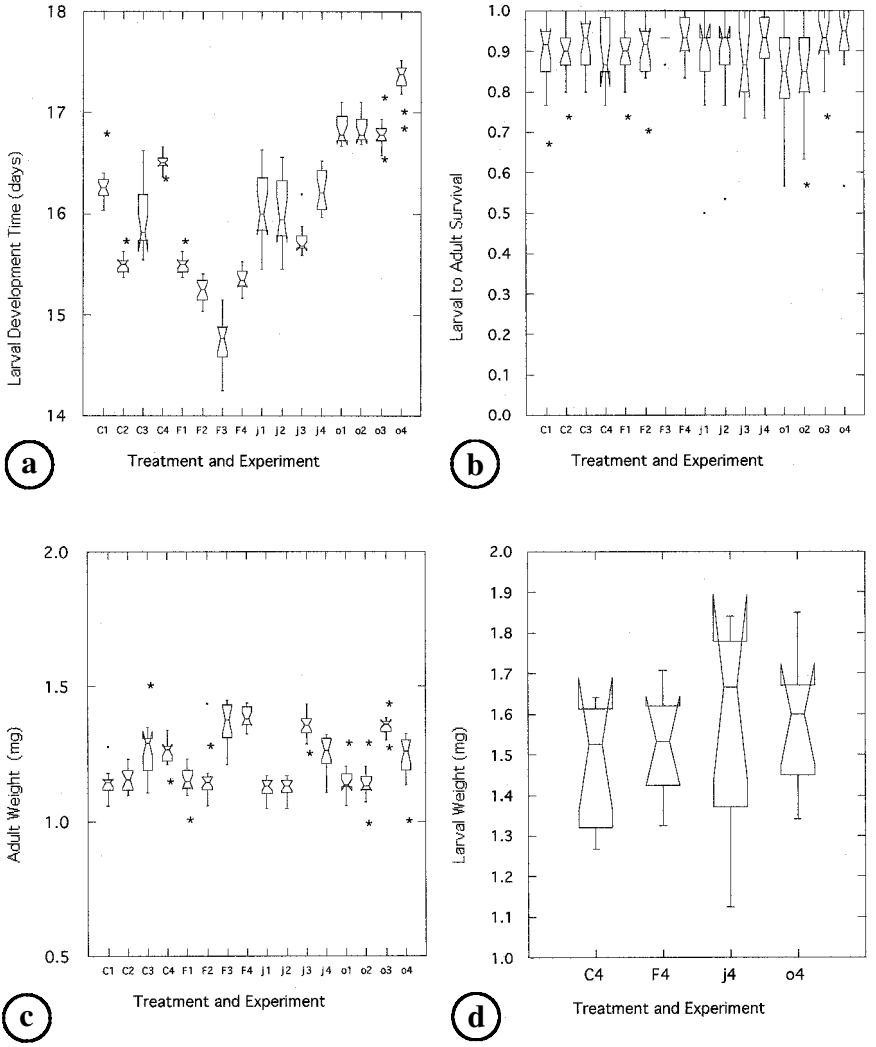

Fig. 1. *EMFs and fitness*—Notched box plots are given for the experiments described in Table 1 as follows: (a) larval development time (the time taken for half of the surviving flies to emerge, $T_{1/2}$, in days); (b) adult survival for an input of 30 larvae, given as a proportion; (c) surviving adult weight (mg); and (d) larval weight (Experiment 4 only, mg). The *x*-axis gives the treatment (C, F, j, and o) for each experiment (1, 2, 3, and 4) as described in Table 1. Note: A notched box plot provides a simple graphical summary of a batch of data and implements confidence intervals on the shown median values. The boxes are notched at the median and return to full width at the lower and upper confidence interval values. If the intervals around two medians do not overlap, then one can be confident at about the 95% level that the two population medians are different. Outside values are represented by an asterisk and far outside values by an open circle.

(b) EMFs and the [ATP]/[ADP] Ratio

Results for the [ATP]/[ADP] ratio in the presence of both NAD and dH_2O are given in Figure 2. Again, we have used visual inspection of the figure to assess the comparisons described above. Comparisons between (d, j) and (d, o) indicated significant differences for both assays, with a higher [ATP]/[ADP] ratio observed for (d, j). Comparisons between (F) and (C) indicated a dependence on the assay as follows: (F) > (C) for NAD and (F) = (C) for dH_2O. Comparisons between (F) and (d, j) and (d, o) indicated that (F) produced a higher [ATP]/[ADP] ratio than (d, j), which was greater than (d, o). Differences between treatments were more evident for supplemental NAD than dH_2O. Finally, the Pearson correlation coefficient for the [ATP]/[ADP] ratio and larval development time in Experiment 4 was –0.856.

4. Discussion

A reduction in exposure to random laboratory EMFs and two categories of EMF frequencies shortened larval development time and increased the [ATP]/[ADP] ratio in *D. melanogaster*. The magnitude of this effect appeared to be modified by the category of EMFs assessed, and we suggest that this may occur via modified and information-augmented EMFs (see below). This is another example of intention imprinting having a significant effect on physical reality (Dibble and Tiller, 1999). Differences observed for the [ATP]/[ADP] ratios were not large, but were significant, indicating that the EMFs studied here modified energy metabolism. Although our results for developing larvae

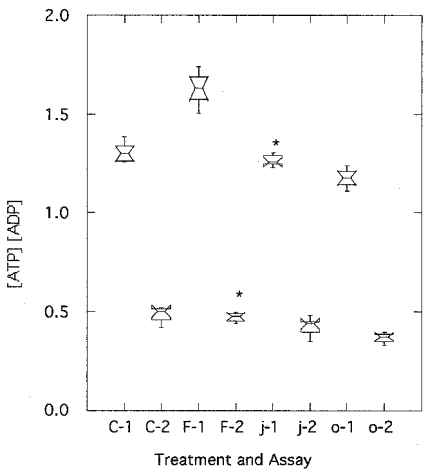

Fig. 2. EMFs and the [ATP]/[ADP] ratio in the presence of NAD and dH_2O as assayed in Experiment 4 of Table 1. The *x*-axis gives the treatment (C, F, j, and o) for each assay (1: NAD and 2: dH_2O).

indicated a decrease in the [ATP]/[ADP] ratio in the presence of EMFs, other studies have shown that EMFs (15, 50 Hz) may stimulate both ATP synthesis and cell proliferation in *Corynebacterium glutamicum* and *Sacchharomyces cerevisiae* (Lei and Berg, 1998; Mehedintu and Berg, 1997). This contrast may be a consequence of the higher EMF frequencies studied here, the use of the Faraday cage, and the fact that both the biological system under study and its status influence the effects of EMFs (Goodman, Greenbaum, and Marron, 1995).

Larger treatment differences were observed for the [ATP]/[ADP] ratio and supplemental NAD, in comparison to supplemental dH_2O (Figure 2). This result was in accord with Kohane (1994) where the [ATP]/[ADP] ratios were also higher, as a consequence of the shorter reaction time. As previously noted, supplemental NAD increases electron transport chain activity and the [ATP]/[ADP] ratio in larval homogenates. Hence, EMF effects on energy metabolism may be greater when the electron transport chain is active. EMF effects on energy metabolism may manifest both *in vivo* and *in vitro,* and we will assess details of this in future experiments.

A higher [ATP]/[ADP] ratio was observed for the treatments that produced a shorter development time, providing additional evidence for a relationship between energetics and fitness (Parsons, 1997; Watt, 1985). Our results also suggest that that EMFs may function as a biological stress (Goodman, Greenbaum, and Marron, 1995; Smith, 1996), possibly via change in energy metabolism, since exposure to EMFs lengthened larval development time, which is associated with total fitness: Earlier sexual maturity leads to higher fitness (Cole, 1954; Hoffmann and Parsons, 1991; Parsons, 1997).

We detected significant effects on larval development time, but not on third instar larval weight, adult survival, and, in general, surviving adult weight. In this regard, Veicsteinas *et al.* (1996) studied the development of chicken embryos exposed to an intermittent EMF of 50 Hz. Statistical comparisons between exposed and sham-exposed values did not show significant differences for the variables studied—extracellular membrane components and histological examinations of brain liver and heart during development, egg fertility and egg weight, body weight of chickens at 90 days from hatching, and histological analysis of body organs.

Furthermore, Morganato *et al.* (1995) concluded that continuous exposure to a 50 Hz magnetic field during 70% of the life span of rats prior to sacrifice did not significantly alter growth rate; the morphology and histology of liver, heart, lymph nodes, testes, and bone marrow. Hematology, hematochemistry, and the neurotransmitters dopamine and serotonin were also unaffected by the exposure. The significant results observed here for larval development time may be a consequence of higher EMF frequencies, the Faraday cage, and the biological system under study (see above).

The decrease in larval development time observed here did not appear to be due to a trade off with the measured fitness components but may also arise as a trade off with other life-history traits (Hoffmann and Parsons, 1991; Odum,

1985). In the present study, we assessed EMF frequency effects under non-stressful nutrient conditions and a relatively low larval density (Kohane, 1988, 1994). Fitness differences are enhanced under stress, and, therefore, we may both detect differences for additional fitness components and clarify the surviving adult weight results under more stressful nutrient conditions (Kohane, 1994). We also note that the EMF effects may be due to modification of the larval environment, in particular the food, and we will assess this idea in future experiments studying EMF effects on larval energy metabolism *in vitro*.

Treatment results across the four experiments were not homogeneous for larval development time and adult survival. Of interest were the following: (a) the relative homogeneity of the effect of (d, j) on larval development time suggesting that the intention may produce a constrained larval development time and (b) the shorter development time observed for all treatments in Experiment 3, in comparison to Experiment 4, suggesting that exposure period may influence this fitness component. Considering surviving adult weight, significant differences were only observed in Experiments 3 and 4, where the Faraday cage effect produced the highest weights (F). The results for Experiment 4 suggest that cross-generational exposure to EMFs may affect fitness in ways that differ from single generation exposure. However, in general there did not appear to be specific patterns to the heterogeneity. Hence, at this time we cannot conclude much about strain, specific device (single or triple oscillator), and exposure period, and we will assess these aspects in detail in future experiments.

The thermodynamic basis for an EMF effect on biological systems is a consequence of the influence of the standard electrochemical potential upon biological processes. Standard thermodynamic theory (Tiller, 1990) shows us that the electrochemical potential, η_A^j, of species j in medium A, is given by

$$\eta_A^j = \mu_{oA}^j + kT \ln(a_A^j) + z^j eV = 1/2(\varepsilon_A \bar{E}^2 + m_A \bar{H}^2) \qquad (1)$$

Here, μ_o = standard state chemical potential, T = temperature, k = Boltzmann's constant, a = chemical activity, ($a = \gamma_c$ where c = concentration and γ = activity coefficient), e = electron charge, z = valence, V = electrostatic potential, ε = electrical permittivity, m = magnetic permeability, $\bar{E}$ = electric field, and $\bar{H}$ = magnetic field. One finds that $z^j = +1$ and - 1, respectively, for the proton and electron. Thus, the last term represents the applied EMF contribution to the standard electrochemical potential and, hence, to various biological systems.

The particular physical variable or quantum potential that may be important in our study is the magnetic vector potential (MVP), denoted $\bar{A}$, which creates an electric field $\bar{E}$, and magnetic field $\bar{H}$ (Jackson, 1962; Kraus and Carver, 1973; Tiller, 1993, 1997). Therefore, we attribute the EMF and augmented EMF effects on larval development time to the influence of the MVP upon the activity of the electron transport chain activity and the [ATP]/[ADP] ratio as follows: the MVP diminished electron transfer to the electron transport chain from the NAD pool and cellular electron availability. The diminished electron

transfer modified the redox potential causing lowered electron transport through the electron transport chain and decreased proton pumping and energy availability. This effect may have decreased the proton motive force across the inner mitochondrial membrane, subsequently modifying cytosolic phosphorylation status (Brand and Murphy, 1987; Kohane, 1994). The above approach suggests that the MVP may (a) modify NAD levels and (b) interact with the electron transport chain at positions beyond electron availability. We will assess these hypotheses in future experiments studying EMF effects on larval energy metabolism *in vitro*.

Discussion of the results concerning the categories of EMFs and the possibility of augmenting EMFs requires that one look to a deeper understanding of the quantum vacuum or so-called empty space. This has been established by quantum theory as a chaotic, virtual particle sea of boundless energy (energy density equivalent to approximately 10^{94} g/cc) at the quantum relativity level. An interaction occurs between this virtual particle sea and the fundamental particles of physical matter to ultimately determine the magnitude of μ_o in Equation 1 (Feynman and Hibbs, 1965; Lee, 1988; Tiller, 1997).

Change in characteristics of the physical vacuum should, in turn, change the ground state energy of fundamental particles, atoms, molecules, and biological moieties (Tiller, 1993). These changes may manifest in biological systems as a consequence of the relationship between the ground state energy of fundamental particles (or charge carrying species) and the standard state electrochemical potential (μ_o in Equation 1). Tiller (1997) suggested that directed human intention is capable of altering characteristics of the physical vacuum. Thus, if a human intention can shift these characteristics even a tiny amount, the ground state energy of the electron would be appreciably altered. Thus, the primary effect of intention on biological systems may occur by modification of this ground state energy for the charge carrying species and, thus, the standard state electrochemical potential for atoms containing such particles. Therefore, μ_{oA}^{j} in Equation 1 may be affected by directed human intention.

The magnetic vector potential is thought to be involved with human intention (Tiller, 1993, 1997). If we call the incremental change in $\bar{A}$ associated with a specific focused intention, $\Delta\bar{A}$, then standard electrodynamic equations yield changes $\Delta\bar{E}$ and $\Delta\bar{H}$ (Jackson, 1962; Kraus and Carver, 1973) and the intention effect may both enter physical reality via Equation 1 and influence energy metabolism and fitness.

Although others have proposed a kinetic rate-limiting step for EMF effects on biological systems (Eichwald and Walleczek, 1996, see above), a sound basis also exists for a thermodynamic potential change as the rate-limiting step. The final apportioning of the total effect between thermodynamic and kinetic categories awaits further research. In conclusion, the process path from EMFs to a biological effect in this work is likely to be via an influence of the magnetic vector potential on electron transport chain activity. The intention-augmented EMF effect is likely to just insert contributions ($\Delta\mu_o$ and $\Delta\bar{A}$) into this process path.

Acknowledgments

We wish to acknowledge Professor W. B. Watt, Department of Biological Sciences, Stanford University, for technical help and the loaning of facilities and equipment. We also acknowledge Durance Corporation and Ditron LLC for financial assistance and laboratory facilities.

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