Microwave Radiation Induces a Heat-Shock Response and Enhances Growth in the Nematode *Caenorhabditis Elegans*

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***Abstract*—This paper shows that prolonged (overnight) exposure to continuous microwave fields (750 MHz, 0.5 W) can induce both a heat-shock response and enhanced growth in the nematode worm *Caenorhabditis elegans*. Exposures were conducted in a TEM cell with matched load, producing an E-field of approximately 45 V m^{-1} at the center (where test worms are placed). Biomonitoring of heat–shock responses has been simplified by using two transgenic strains (PC72 and PC161), which both carry stress-inducible reporter constructs, respectively, placing *lacZ* (β-galactosidase) and *lacZ* plus green fluorescent protein expression under the control of *C. elegans hsp16-1* promoters. *In situ* localization of reporter expression reveals a minority of test worms, which respond strongly to microwave exposure. Enzyme activity measurements average these reporter responses across many thousands of individual worms, giving a reliable indication of the overall stress imposed on a population. The temperature profile of reporter responses induced by microwave exposure parallels that induced in controls by heat alone, but is displaced down the temperature scale by some 3 °C. Length measurements were conducted at intervals in synchronized *C. elegans* cultures seeded with $L1$ larvae. Using pooled data from nine separate runs, growth was stimulated by 8.5% after overnight microwave exposure (relative to controls), and this disparity increased to 11% after 24 h of further growth without irradiation. Both heat–shock responses and increased growth would be consistent with a modest increase in temperature, raising the possibility that microwave exposure might cause limited heating in this system. However, there is no detectable rise in the temperature of either medium or worms during overnight exposure under these conditions, discounting both generalized and localized (worm-specific) heating effects. We conclude that both growth and heat–shock responses are induced by microwave exposure through one or more nonthermal routes.**

***Index Terms*—Biological effects of electromagnetic radiation, genetics, heating, microwave communication, stress physiology.**

I. Introduction

CURRENT exposure limits for microwave radiation from mobile phones assume no adverse biological effects provided that heating is negligible ($\ll$1 °C) [1]. Recent health concerns about mobile phone use have focused on cognitive functions [2] and possible cancer-promoting effects [3] in humans, but the evidence remains inconsistent and unconvincing. In this paper, we show that prolonged exposure to continuous microwave emissions (as used in analog mobile phones) causes reproducible biological effects similar to those caused by heat, even though there is no measurable increase in temperature. Our test organism is the free-living soil nematode *Caenorhabditis elegans*, which offers many practical advantages thanks to its small size, rapid life cycle (three days at 25 °C), ease of culture and excellent genetics.

Excessive heat damages cellular proteins and triggers the production of heat-shock proteins (HSPs), a universal protective response found in all organisms. The HSPs act as molecular chaperones to refold and reactivate heat-damaged proteins, and dispose of those beyond rescue [4]. Other physical and chemical stressors also induce HSP expression, although damage may be indirect [e.g., via reactive oxygen species (ROS)]. Protein damage promotes the dissociation of HSP/heat-shock factor (HSF) complexes [5], such that free HSF can trimerise and bind to the HSEs (heat-shock elements) which preface all stress-inducible HSP genes, thereby activating HSP gene transcription [6]. HSP induction provides a summative biomarker response, reflecting the extent of underlying protein damage (proteotoxicity) under adverse environmental conditions [7], [8]. The difficulty and expense of HSP detection can be circumvented by using transgenic test organisms [7]. One such is the *C. elegans* strain PC72, which carries an *E. coli lacZ* reporter gene (encoding β-galactosidase) linked to the *C. elegans hsp16-1* promoter [9]. Like *hsp16* itself, reporter expression in this strain is strictly stress inducible throughout the life cycle [9].

PC72 and similar strains of *C. elegans* (e.g., PC161 carrying green fluorescent protein (GFP) as well as *lacZ* reporters; see Section II) provide stress-sensitive biosensors applicable in a variety of contexts. In this study, young adult worms were exposed overnight (18 h) to microwave radiation at 750 MHz and 0.5 W (27 dBm) in the TEM cell described previously [10], at exposure temperatures between 23 °C–25.5 °C. The *hsp16* promoter is heat-inducible above 27 °C [11], and optimal sensitivity to nonthermal stressors (such as pesticides and heavy metals) is apparent 2 °C–5 °C below this threshold [9]–[12].

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II. Methods

A. Worm Strains

Strain PC72 carries multiple copies of a reporter construct in which the *E. coli lacZ* gene and SV40 nuclear localization signal (NLS) are fused in-frame into the second exon of the *C. elegans hsp16-1* gene [9], [13]. To produce PC161, a promoterless S65C GFP/*lacZ*/SV40 NLS construct (pPD96.04, obtained from A. Fire) was inserted at the same position into *hsp16-1* and this fusion gene microinjected into adult hermaphrodites along with the pRF4 *rol6* selectable marker [14]; multiple copies were then integrated into the genome by γ irradiation. Homozygous rolling animals were selected and outcrossed over six generations.

B. Worm Culture

Worms were grown on nematode growth medium (NGM) agar plates at 15 °C, as previously described [12], and $L1$ larvae isolated by filtration through a 5-μm mesh (Wilson Sieves, Nottingham, U.K.) [15]. Larvae were exposed overnight to microwaves (below) at 24 °C or 25 °C for growth studies, or grown on for a further three days at 15 °C (reaching $L4$ early adulthood) for stress reporter assays.

C. Microwave Exposure and Dose Estimation

$L1$ larvae or $L4$/adult stages were exposed overnight (18 h) to microwave radiation at 750 MHz and 0.5 W (27 dBm) in the TEM cell previously described [10] within a Leec $LT3$ heating/cooling incubator (temperature stability $\pm$0.2 °C). For stress responses, worms were exposed in a shallow depth of aqueous K medium (53 mM NaCl, 32 mM KCl) in six- or 12-well multiwell dishes (Corning). For growth studies, $L1$ larvae were plated on fresh NGM agar plus food bacteria in six- or 12-well dishes. Control worms from the same source population were treated identically, but shielded from microwave exposure (foil-wrapped outside the TEM cell in the same incubator). The E-field in the centre of the TEM cell (where worm dishes are placed) is approximately 45 V m^{-1} (power density of about 10 W m^{-2}); however, penetration of the electromagnetic field (EMF) into the aqueous/agar medium and into the worms will be much less than at the surface. We have measured the relative permittivity of the worms (50%-w/v suspension in K medium, as compared to K medium alone) at 615 MHz using the cavity perturbation technique. This implies a conductivity of about 0.48 mho m^{-1}, which is comparable to that measured for a range of human tissues [16]. From this, we estimate an electric field penetration of about 1.3 V m^{-1} (based on an external field of 45 V m^{-1}), which gives a specific absorption rate (SAR) of the order of 0.001 W kg^{-1}. This compares with SAR values of 0.02–1.0 W kg^{-1} within the human head when irradiated by mobile phones at 835 or 1900 MHz [17].

D. Assay Procedures

For growth studies, worms were washed off the plates and frozen in blind-coded tubes either immediately after exposure, or after a further 24 h of growth at the exposure temperature of 24 °C or 25 °C. Thawed worms were mounted in saline for microscopy, visualized via a charge-coupled device (CCD) camera and video (on-screen magnification $\times$140), and the lengths of 200 worms measured for each test condition (including all worms within each microscope field before moving on) [15]. Data from nine runs are included in the analysis for Fig. 3. For stress response assays, worms were washed, frozen, acetone-fixed, and dried, prior to fluorometric assays for β-galactosidase activity (using a Perkin-Elmer HTS7000 microplate reader), as previously described [12]. All activities (pmoles product h^{-1} mg^{-1} protein) were normalized against 15 °C controls (=100%), so as to compare runs at different temperatures. β-galactosidase activity was detected by Xgal staining, and GFP was visualized under UV.

E. Statistical Procedures

Because of inter-run variability, pooled growth data from nine independent runs at two different temperatures (24 °C and 25 °C) was subjected to iterative general linear model (GLIM) analysis to identify the principal sources of variation by three-way analysis of variance (ANOVA) [18]. None of this variation was attributable to temperature; hence, two-way least-squares ANOVA using StatGraphics [12] allowed derived means to be calculated for each test treatment (exposed versus control), after taking account of inter-run variation. A student's t-test was used to compare microwave-induced with control reporter expression at each temperature tested.

III. Results

Fig. 1(a) shows *in situ* staining for reporter product (nuclear-localized β-galactosidase) in PC72 worms after 18 h of microwave exposure at 25 °C; many exposed worms show weaker staining, but all control worms are unstained [see Fig. 1(b)]. Similar exposure of PC161 worms at 25 °C induces GFP expression in post-blastula embryos within adult hermaphrodites [see Fig. 1(c)], which is never seen in control worms [see Fig. 1(d)]. The ribbons of yellow–green fluorescence apparent in both Fig. 1(d) and (c) represent autofluorescent gut granules, not GFP. Strong reporter expression is confined to a minority (circa 10%) of exposed worms in both strains, but probably most worms express weak (below detection threshold) rather than zero reporter activity. Although microwave-exposed PC72 worms show occasional staining in eggs and embryos [as for PC161, see Fig. 1(c)], high background autofluorescence will swamp any GFP signal from the gut in PC161 worms. Between them, gut and embryos comprise more than half the internal tissues of an adult nematode; such widespread expression is inconsistent with localized tissue-specific heating.

Fig. 2 compares the temperature profile for *lacZ* reporter expression in control PC72 worms (22 °C–28 °C) with that in microwave-exposed worms (24 °C–25.5 °C). For consistency between runs at different temperatures, all reporter activities were normalized relative to those of worms from the same batch grown at 15 °C (15 °C controls = 100%). On this basis, controls at temperatures up to 27 °C do not exceed 500% relative to those at 15 °C. At 24 °C, there is no difference in re-

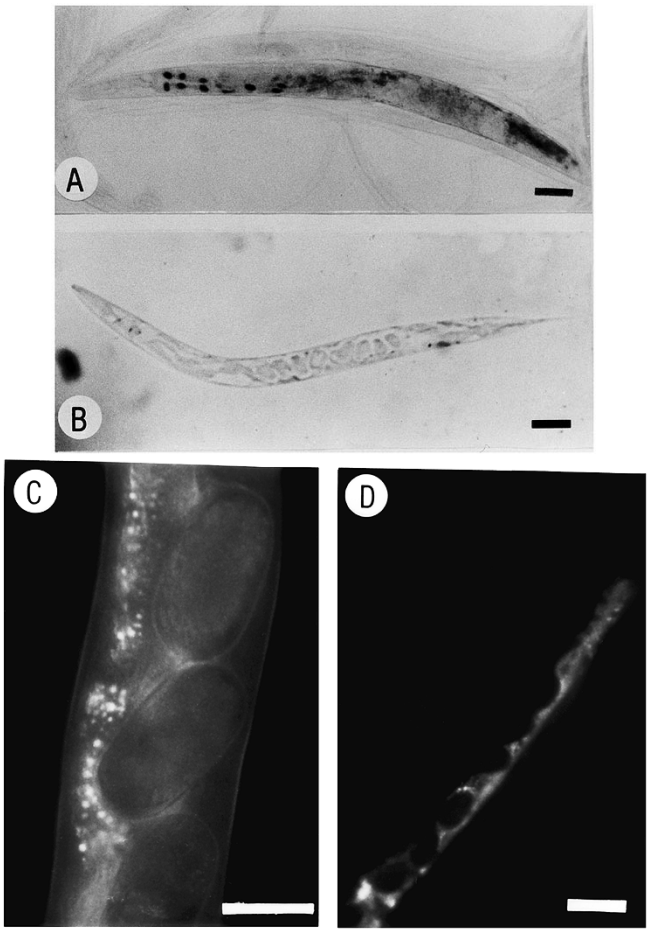

Fig. 1. Stress responses in microwave-exposed transgenic worms. PC72 worms exposed to microwaves for 18 h at 25 °C were stained for β-galactosidase activity using Xgal alongside shielded controls then mounted in glycerol and photographed. Part A: microwave-exposed (note dark nuclear staining in gut nuclei). Part B: typical unstained control. A similar comparison was made for strain PC161 (carrying a stress-inducible GFP reporter) exposed at 25 °C. Photography used fluorescein isothiocyanate (FITC) settings on an Olympus fluorescence microscope. Part C: microwave-exposed (note widespread GFP expression in ovoid embryos); Part D: typical control with no GFP expression. The brighter ribbon of fluorescence in parts C and D is caused by autofluorescent granules in the intestinal tissue (gut), and is yellower than GFP. Bars in all four parts show 50 μm.

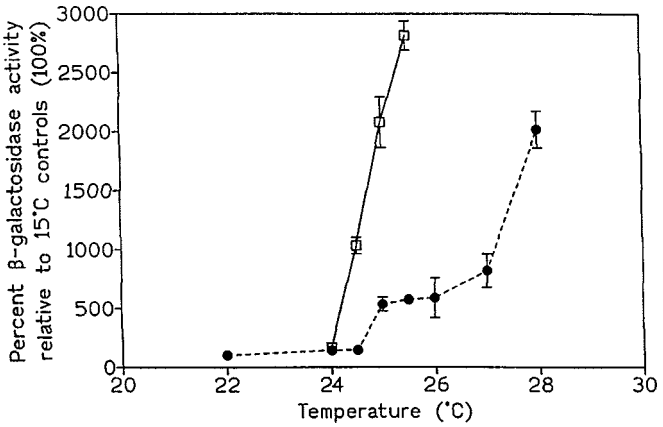

Fig. 2. Temperature profile for stress transgene expression in microwave-exposed versus control PC72 worms. Reporter enzyme activities (see Section II) were compared between microwave-exposed and control worms at half-degree temperature intervals across the range 24.0 °C–25.5 °C ($n = 12$ for each data point), while controls only were assayed outside this range ($n = 6$). All results at each temperature tested were normalized relative to 15 °C controls (100%) included in each such run and derived from the same batch of filtered worms. Filled circles, dashed line: reporter responses to heat alone. Open squares, solid line: microwave-induced reporter responses at different temperatures. All data points show mean values ± SEM.

TABLE I
TEMPERATURE MEASUREMENTS FOR EXPOSED VERSUS CONTROL SAMPLES IN WORM SUSPENSION AND IN K MEDIUM

Shielded Controls – K medium	Shielded Controls – 50% worm suspension	Exposed Samples – K medium	Exposed Samples – 50% worm suspension
24.7°C	24.7°C	24.8°C	24.8°C
24.7°C	24.7°C	24.8°C	24.8°C
24.6°C	24.5°C	24.8°C	24.7°C
24.5°C	24.5°C	24.6°C	24.5°C
Mean 24.63°C	**Mean 24.60°C**	**Mean 24.75°C**	**Mean 24.70°C**
± 0.096°C (SD)	**± 0.116°C (SD)**	**± 0.100°C (SD)**	**± 0.141°C (SD)**

porter expression between microwave-exposed and control populations ($p > 0.05$), but microwave-induced reporter activities rise steeply through 24.5 °C and 25.0 °C to 25.5 °C ($p \ll 0.01$, as compared to controls). Above 26 °C, controls show variable heat-induced expression so that the differences attributable to microwave exposure become less clear (data not shown). Thus, microwave exposure induces reporter-gene expression at temperatures some 3 °C below those required to induce comparable responses in nonexposed controls (Fig. 2). To investigate whether this effect could possibly be due to microwave heating, the medium temperature was monitored continuously during exposure at 25 °C, using a Luxtron fiber-optic probe; no inflection of the temperature record could be detected during several hours of exposure. Immediately after exposure, the mean medium temperature was 24.70 °C± 0.07 °C (SEM) in 25 microwave-exposed wells and 24.74 °C± 0.081 °C in 25 similar shielded control wells. To test for possible selective heating of the worms, we exposed small volumes (0.2 mL) of concentrated worm suspension (50%-w/v packed worms in K medium) alongside K medium alone for 20 h at 25 °C, in parallel with identical shielded control samples in the same incubator. At the end of this period, plates were removed and the sample temperatures measured rapidly (alternately K medium, then worm suspension) with a fast-response microthermocouple, giving the results shown in Table I. Although all eight measurements were completed within 1 min of removing each plate from the incubator, the last samples measured were always detectably cooler than the first few. Despite this slight cooling, it is clear that the mean temperatures differ neither between K medium and 50% worm suspension, nor appreciably between microwave-exposed and control conditions. This not only rules out generalized heating by microwaves, but also discounts the possibility that worms might be selectively heated much more than their surrounding medium. Fig. 2 implies that worms would need to be 3 °C hotter than their surroundings in order for heating alone to account for the induction of heat-shock reporter responses at 25 °C. In a 50% (W/V) worm suspension, this model would predict that the overall temperature should rise by around 1.5 °C (heat diffusion being inevitable over 18 h), yet no such rise is seen in Table I. The TEM cell used in this paper delivers an

E-field of about 45 V m^{-1} at the center of the cell, from which we estimate an SAR of 0.001 W kg^{-1} (see Section II). Mobile phones deliver SARs in the range of 0.02–1.0 W kg^{-1} at 835 or 1900 MHz [17], using pulsed and modulated signals in the case of digital models. Clearly, our test conditions most closely resemble those experienced by users of older-style analog cell phones. Tests are now in progress with digital mobile phones to determine whether these also induce heat–shock responses in transgenic worms.

Worm growth is also stimulated, both during and after exposing synchronous populations of $L1$ larvae to microwave radiation overnight at 24 °C or 25 °C, as compared to controls at the same temperature. Worms were frozen for length measurements either immediately after exposure (18 h) or else after 24 h of further growth at 24 °C–25 °C (42 h). There is considerable variation in the final size distributions attained, due in part to a variable proportion of $L2$ larvae included alongside the $L1$ larvae isolated by size fractionation [15]. Iterative GLIM [15], [18] analysis of the data pooled from nine independent runs confirms this highly significant effect of run ($F_{425915} = 275.76$, $P < 0.001$ after 18 h, $F_{357375} = 60.3$, $P < 0.001$ after 42 h). But over and above this, there is a clear and consistent effect of treatment attributable to microwave exposure ($F_{113565} = 54.91$, $P < 0.001$ after 18 h, $F_{151955} = 31.07$, $P < 0.001$ after 42 h), and a less significant interaction between run and treatment ($F_{413239} = 7.44$, $P < 0.001$ after 18 h, $F_{350976} = 9.8$, $P < 0.005$ after 42 h). Four of the nine runs were conducted at 24 °C and five at 25 °C; however, the same GLIM analysis found that none of the variability in the overall data set was attributable to temperature (24 °C versus 25 °C). This implies that the growth effects observable at 24 °C are indistinguishable statistically from those at 25 °C, in contrast to the patterns of heat–shock reporter expression (undetectable at 24 °C, but significant at 25 °C; see Fig. 2). Growth stimulation is one predicted consequence of mild heating, but neither our temperature measurements (above) nor the lack of any discernible temperature effect (24 °C versus 25 °C) offers any support for this explanation. Fig. 3 shows the overall difference between exposed and control populations at both time points (four runs at 24 °C plus five runs at 25 °C), in terms of the derived mean lengths obtained from least-squares two-way ANOVA of the pooled data set (using StatGraphics [12]), after taking account of the inter-run variation. Immediately after microwave exposure for 18 h, worms are 8.5% longer than controls, whereas after 24 h of further growth, this size difference increases to 11.2% (42 h). By the third day (66 h), many worms reach adulthood and size differences become obscured by large numbers of new $L1$ larvae (data not shown). Since the somatic cell lineage of *C. elegans* is invariant, the observed size differences (Fig. 3) cannot arise from extra cell divisions, but probably reflect a faster developmental rate in the exposed worms. Consistent with this, eggs (diagnostic of adulthood) appear markedly earlier in exposed as compared to control cultures (unpublished observations).

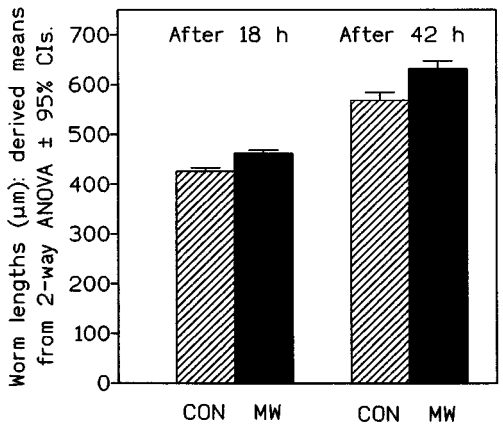

Fig. 3. Growth stimulation in PC72 worms following microwave exposure. Worm growth at 24 °C–25 °C was monitored as described in Section II, and data from nine independent runs (four at 24 °C and five at 25 °C) were pooled for statistical analysis (see text). Derived mean lengths from two-way least-squares ANOVA (StatGraphics) are shown both immediately after exposure (18 h) and after 24 h of further growth at 24 °C or 25 °C (42 h). Left-hand-side group, comparison after 18 h; Right-hand-side group, comparison after 42 h. In both groups, hatched bars (left-hand side) show the mean lengths of control worms, while solid bars (right-hand side) show the corresponding mean lengths of microwave-exposed worms. Error bars show 95% confidence intervals for these means. Ambient incubator temperature (24 °C versus 25 °C) did not contribute to the variation observed between these size distributions.

IV. Discussion

We have demonstrated consistent and significant induction of the heat–shock response in *C. elegans* during prolonged exposure to continuous microwave fields at 750 MHz and 0.5 W, conditions comparable to those produced by analogue mobile phones. There is also a modest stimulation of growth, which is sustained for some time after cessation of exposure. Since there is no measurable increase in ambient or worm temperature during exposure, both effects appear to be mediated by nonthermal mechanisms. A heat–shock response might be inducible through disruption of the hydrophobic and other weak noncovalent interactions that maintain proteins in their active three-dimensional (3-D) conformations. This would not necessarily entail gross heating of the entire cell contents (the bulk of which consists of water), and certainly would not require breakage of covalent bonds. Other possible mechanisms underlying heat–shock induction by microwaves include: 1) enhanced formation of ROS, which are classic inducers of the heat–shock response and/or 2) interference with cellular signalling systems involved in HSF activation by phosphorylation. The excellent genetics and recently completed genome sequence of *C. elegans* make it an ideal organism in which to explore the genetic basis of the heat–shock response to microwaves. Our observation of enhanced larval growth at first sight seems anomalous since most chemical toxicants **reduce** the growth rate as well as inducing a heat–shock response. However, an acceleration of development (resulting in earlier reproduction) may be an adaptive response to mildly stressful conditions. Further exploration of the longer term life-history consequences of microwave irradiation is needed to confirm this suggestion. Our temperature data (Table I) discount alternative explanations in terms of differential heating of worms versus their surrounding medium. However, this could only be rigorously excluded through real-time thermal imaging of exposed worms.

Our findings suggest that the stress-sensor strains used in this paper are particularly suitable for investigating the effects of

microwave exposure and of other EMFs [19]. The strong interaction noted here between temperature and microwave-induced responses suggests that temperature may be a key consideration when assessing contradictory claims as to the biological effects of EMFs [2], [3]. Although overnight exposures were used in this paper to demonstrate unequivocal biological responses, heat–shock responses are detectable over exposure periods as low as 2 h [10]. Shorter exposures cannot be tested using this system since sufficient time must elapse for reporter induction, translation, and enzyme accumulation. Our *in situ* localization studies (Fig. 1) suggest that only a minority of test worms (circa 10%) express the reporter gene strongly [see Fig. 1(a) and (c)], whereas the majority show little or no expression. This is unexpected, given the genetic homogeneity of the test worms (which reproduce largely by hermaphrodite self-fertilization) and the fact that they were all at the same developmental stage (mainly adults in three-day cultures of synchronized $L1$ larvae [15]). It is possible that the $L4$-to-adult moult represents a stage of particular stress sensitivity, or alternatively, this may be a stochastic effect. It is likely that several internal damage-limitation systems (including presynthesized HSPs [4]) may have to be overcome before the reporter genes become activated. Highly toxic stressors (e.g., cadmium) will overpower these defences in almost all worms, but weaker stressors (including microwaves) may do so only in a minority of more susceptible individuals [9]. Nevertheless, measurements of reporter enzyme activity (as in Fig. 2) average these variable responses across many thousands of individual worms and, thus, provide a reliable overall estimate of the level of stress experienced by a population [12].

Our findings do not directly address the vexed question of whether prolonged use of mobile phones might be damaging to human health [2], [3], but given the universality of the heat–shock response [4], it is likely that similar effects will be detectable in vertebrate systems. There is only a 4 °C difference between the ambient human body temperature of 37 °C and the human heat–shock temperature of circa 41 °C. This is scarcely greater than the 3 °C disparity (Fig. 2) between control and microwave-exposed temperature profiles for heat–shock reporter expression in PC72 worms. Further studies are needed to determine whether microwave exposure might have longer term effects on worm fecundity or life span.

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