nusoidal interference term, $\delta_j(k)$ being the phase shift. A full analysis of the spectrum on the basis of Eq. 1 requires the use of computer-assisted Fourier transform techniques (*1*, *2*). However, it is more illustrative for the present purpose to employ a straightforward graphical technique (*1*) to deduce the nearest-neighbor distance.

The graphical technique is based on the fact that the EXAFS curve is usually dominated by scattering from the nearest neighbor. This is especially true of the positions of the principal maxima and minima, which are determined mainly by the first sine term in Eq. 1, namely $\sin[2kR_1 + 2\delta_1(k)]$. If δ_1 is linear in k, then $\delta_1 = \alpha_1 k + \beta_1$, and the argument of the sine term takes the form $2k(R_1 - \alpha_1) + 2\beta_1$. The approximate positions of the maxima and the minima of the EXAFS curve are thus given by

$$n\pi = 2k(R_1 - \alpha_1) + 2\beta_1 \qquad (2)$$

where $n = 0, 2, 4, \ldots$ for maxima and 1, 3, 5, . . . for minima. A plot of n against k for the dominant maxima and minima of the EXAFS spectrum shown in Fig. 2 is given in Fig. 3 with $k = 0$ taken to correspond to the inflection point, $E_{K\,\mathrm{edge}} = 1552$ eV, of the measured x-ray absorption coefficient. The points closely fit a straight line with a slope $(2/\pi)(R_1 - \alpha_1)$ of 1.7 Å. This leads to the basic result $(R_1 - \alpha_1) \approx 2.6$Å. Since $R_1 >> \alpha_1$ (α_1 is typically a few tenths of an angstrom), this result is in good agreement with the known nearest-neighbor distance of 2.86 Å for the aluminum face-centered-cubic lattice (*8*).

The spectrum presented in Fig. 2 is noteworthy for several reasons. It illustrates the capability of the laser-EXAFS technique to record EXAFS spectra of light elements with absorption edges below about 3 keV, which are difficult to study with other x-ray sources. The technique is particularly suitable at present for the study of *K*-edge EXAFS spectra of the elements from carbon to sulfur and of *L*-edge EXAFS spectra of the elements from sulfur to molybdenum. More important, however, the complete laser-EXAFS spectrum presented in Fig. 2 was obtained in only a few nanoseconds with a single pulse of laser-produced x-rays. This represents a dramatic improvement in the speed and ease of obtaining EXAFS data compared to what is possible with other known x-ray sources. The technique also makes possible the measurement of "flash-EXAFS" spectra of transient species having lifetimes of a few nanoseconds or less. Thus, with this technique it may soon be possible to make "snapshots" or "movies" of the structural changes that occur in molecules when they are excited by optical or other means. If this proves to be the case, laser-EXAFS will have provided an important new dimension to the study of chemical structure by x-ray absorption techniques.

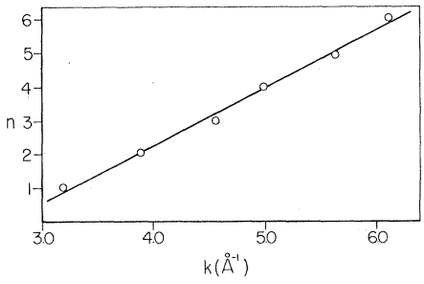

Fig. 3. Graph of n versus k for aluminum. The points correspond to the features indicated by arrows in Fig. 2.

P. J. MALLOZZI, R. E. SCHWERZEL
H. M. EPSTEIN, B. E. CAMPBELL
Battelle, Columbus Laboratories, Columbus, Ohio 43201

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Juvenile Hormone: Evidence of Its Role in the Reproduction of Ticks

Abstract. *Ovarian development, vitellogenesis, and embryogenesis in recently mated fed females of the soft tick* Ornithodoros parkeri *Cooley were prevented by topical application of the insect antiallatotropin precocene 2. The blockage was relieved by topical application of juvenile hormone. Cancellation of the anti-juvenile hormone effects of precocene 2 and the reestablishment of oogenesis (and oviposition in one specimen) by a naturally occurring insect juvenile hormone argues strongly for a physiological role of juvenile hormone in acarine reproduction.*

In this study we demonstrate that application of the antiallatotropic compound precocene 2 prevents oocyte development in the soft tick (Argasidae) *Ornithodoros parkeri* and that exogenous juvenile hormone III (JH III) repairs the blockage. Precocene 2 has been shown to cause precocious metamorphosis, prevent ovarian development, and induce diapause in various insects presumably by suppressing the function of the corpora allata (*1*). When applied to the tick *Argas persicus*, precocene is reported to induce sterility in adult females (*2*). However, it is not clear whether this effect results from a suppression of JH production. Indeed, precocene also blocks nymphal molting in these ticks, suggesting a more widespread disruption of normal development. Moreover, no convincing evidence of JH activity in ticks has yet been reported. We sought to determine (i) whether the effects of precocene on oocyte development in ticks are similar to those in some insects, and if so (ii) whether the effects are reversed by JH application.

Recently mated unfed female *O. parkeri* were allowed to feed to repletion on white laboratory mice 24 hours before we applied 0.5 mg of precocene in 2 μl of dimethyl sulfoxide (DMSO) to the dorsum of each tick (Table 1). Control ticks remained untreated or we applied 2 μl of DMSO. Seven days after the application of precocene, one group of ticks was given a second application of 0.5 mg of precocene in DMSO. All groups (ten ticks each) were maintained at 26°C and 85 percent relative humidity. Control ticks began oviposition on day 12 after feeding; they deposited an average of 316 eggs (untreated group) and 311 eggs (DMSO-treated group) per female with normal hatch beginning in both groups approximately 14 days after initiation of oviposition. Since no oviposition had occurred by day 56 after feeding in either of

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Table 1. Effects of 0.5 mg of precocene 2 (P) (experiment 1) and 0.5 mg of precocene 2 plus 1, 10, or 100 μg of JH III (experiment 2) on ovum maturation and oviposition of female *O. parkeri*.

Treatment	*N*	Mortality (%)	Percentage of ticks			Number of eggs oviposited†
			With well-developed ova*	With eggs in uterus	Ovipositing	
			Experiment 1			
Control (untreated)	10	0	100	100	100	135
Control (DMSO)	10	0	100	100	100	158
One P application‡	10	0	0	0	0	0
Two P applications	10	10	0	0	0	0
			Experiment 2			
Control (untreated)	10	0	100	100	100	198
Control (DMSO and acetone)	10	0	100	100	100	158
P + 1 μg of JH III	10	0	70	40	10	26
P + 10 μg of JH III	10	0	40	20	0	0
P + 100 μg of JH III	10	70	0	0	0	0

*See (*10*). †Per ovipositing female on day 22 after feeding. ‡0.5 mg of P.

the precocene-treated groups, all of these ticks were dissected and no evidence of oocyte maturation beyond that seen in the unfed state was observed.

In a second series of experiments (Table 1), we applied 0.5 mg of precocene in DMSO to the dorsum 24 hours after feeding and then, 24 hours later, we applied 1, 10, or 100 μg of JH III in 1 μl of acetone to the ventral surface of each tick. We used untreated ticks and ticks treated with both DMSO and acetone as controls. Two days after treatment, seven out of ten of the ticks treated with 100 μg of JH III had red legs, indicative of gut rupture, and soon died. The three survivors showed no significant ovarian recovery upon later dissection. All of the ticks treated with 10 μg of JH III lived: four of them contained ovaries at advanced developmental stages (two of those four had fully developed eggs in the uterus).

The most spectacular recovery of ovarian development and egg maturation occurred among the ticks treated with 1.0 μg of JH III. One of these ten ticks began oviposition on day 15 after feeding (only 3 days after the earliest oviposition by control ticks and well within the range of other controls). Though only 26 eggs were laid, dissection revealed a uterus, oviducts, and ovary full of mature and advanced eggs when compared with controls and precocene-treated ticks not receiving JH III (Fig. 1). In this group (1.0 μg of JH III), seven of the ten ticks showed substantial ovarian recovery and four (including the one that oviposited) had mature eggs in the uterus. Apparently, the 1-μg dosage of JH III is physiologically near normal for the conditions and timing of this experiment. However, the actual quantity of precocene or JH III penetrating the cuticle and reaching receptive tissues is unknown.

Certain developmental and reproductive aspects appear similar in insects and acarines, and its seems reasonable to expect that these events might be regulated by similar hormonal controls. Previous attempts to demonstrate these similarities produced results that could be interpreted as pharmacological disruption of naturally occurring cellular regulatory events. Blockage of nymphal molt, initiation and breakage of ovipositional diapause, embryocidal activity, and functional sterility in ticks as a result of exogenous application of insect hormones, their analogs, or antihormones may all result from toxic rather than endocrinological effects on cells. Our method of experimentally interfering with the production or function of a presumed JH-like compound, and then reversing the effects by topical application of JH, effectively demonstrates the presence of a naturally occurring JH in ticks.

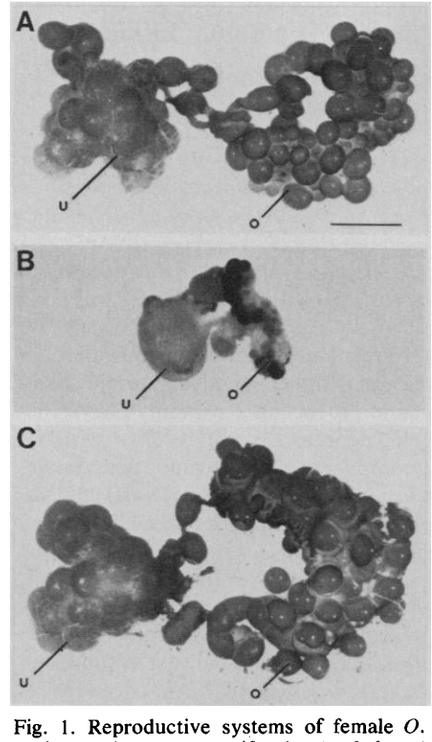

Fig. 1. Reproductive systems of female *O. parkeri* at the same magnification (scale bar, 1 mm). (A) From a control tick treated with DMSO and acetone showing mature eggs in the uterus (*u*), the oviducts and ovary (*o*); the tick had previously laid 185 eggs. (B) From a precocene-treated tick, showing undeveloped eggs in the ovary and the absence of eggs in the uterus. (C) From a precocene-treated tick that was subsequently treated with JH III. The reproductive system contains mature eggs in the uterus, oviducts, and ovary; this tick had previously laid 26 eggs.

Molting hormones (ecdysteroids) are known to occur in insects and crustaceans (*3*), and strong evidence indicates that they are also present in the chelicerates *Limulus polyphemus* (*4*) and the spider *Pisaura mirabilis* (*5*). A few reports dealing with the physiological effects of exogenous ecdysteroids in ticks (*6*, *6a*) and one study in which radioimmunoassay and gas-liquid chromatography combined with mass fragmentography or mass spectrometry were used, established their presence in the hard tick *Amblyomma hebraeum* (*7*). Topical application of several juvenile hormone analogs (JHA) including farnesyl methyl ether (FME), farnesynic acid (FA), Altozar (ZR 512, Zoecon Corporation), Altosid (ZR 515, Zoecon Corporation), ZR 777 (Zoecon Corporation), R-20458 (Stauffer), and others (*6a*, *8*) has shown detrimental effects on egg development in both hard (Ixodidae) and soft (Argasidae) tick species. However, application of the JHA acetaldehyde-2-(2-ethoxyethoxy) - ethyl - *p* - (methylthio)phenyl acetal to the soft tick *Argas arboreus* terminated diapause as evidenced by vitellogenesis and oviposition (*9*).

Since it appears the JH plays similar reproductive roles in the Chelicerata and Mandibulata, we suggest that JH's are ancient molecules that have been used by arthropods for over 500 million years. The present discovery along with reports of the presence of ecdysone in ticks, also suggests the feasibility of considering insect hormones (and analogs) and antihormones in initiating new tick control strategies.

J. Mathews Pound
James H. Oliver, Jr.
Department of Biology,
Georgia Southern College,
Statesboro 30458

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Phycomyces: Interference Between the Light Growth Response and the Avoidance Response

Abstract. Phycomyces *sporangiophores show a growth response to a light stimulus and an avoidance bending response to a physical barrier. A blue-light stimulus administered in conjunction with a barrier interferes with the avoidance bending response. This interference begins after a latency of about 3 minutes and continues for a period of 4 to 5 minutes.*

The most puzzling and least studied sensory pathway in *Phycomyces* is the avoidance response. By a yet unknown mechanism, a sporangiophore senses the presence of a solid barrier a short distance away and will bend away from it after a latency period of about 3 minutes (*1*). We have found that this response can be inhibited by a blue-light stimulus given in combination with the avoidance stimulus. Such interference is evidence of a significant physiological interaction between these two sensory pathways in the regulation of cell growth.

Although genetic studies by Bergman *et al.* (*2*) indicate that the two pathways share a common element, previous work has provided no physiological evidence for such interaction. A study by Cohen *et al.* (*3*) reports that the avoidance bending response is not affected by steady illumination, even at light intensities as high as 20 mW/cm^2, conditions under which the sporangiophore has lost the ability to respond to any light stimulus. Similarly, Ortega and Gamow (*4*) report that, after a saturating 10-minute light stimulus at 82 mW/cm^2, a normal avoidance growth response (bilateral barrier stimulus) still occurs.

In the work we report here, the avoidance stimulus was a plastic cover slip placed about 1 mm from a stage IV sporangiophore and kept at a constant distance throughout each experiment. Sporangiophores were grown in white light and adapted to red light for at least 5 minutes before each experiment. The blue-light stimulus was either a 1-minute pulse or a step (continuous light) at 315 μW/cm^2, with light from a 100-W tungsten bulb filtered through a Corning 5-61 filter. The light was split into two equal beams that struck the sporangiophore on each side at an angle of 30° from the horizontal. The angle of bending was measured in red light by use of a microscope with a goniometer eyepiece.

The application of a blue light step stimulus at 3, 7, or 10 minutes after the avoidance stimulus causes a temporary reversal of avoidance bending (Fig. 1, curves c, d, and e). Table 1 shows that, regardless of the time the light stimulus is applied and the amount of forward bending that has occurred, the interference period begins about 3 minutes after the light stimulus is applied and has a duration of 4 to 5 minutes. Similar results were obtained with 1-minute light pulse stimuli.

Not only does a light stimulus interfere with an ongoing avoidance response but if given earlier it can also interfere with the onset of this response (Fig. 1, curves a and b). Table 2 shows that the avoidance latency period, defined as the interval between application of the barrier and the start of avoidance bending, is 4 to 5 minutes longer in the presence of a light stimulus than in the absence of blue light. The light stimulus clearly interferes with the onset of avoidance bending, and the additional delay is roughly the same as the interference periods described above.

We also found that a succession of 1-minute light pulse stimuli, given at 10-minute intervals during an avoidance response, elicits a corresponding succession of bending reversals. Each reversal begins about 3 minutes after the pulse and lasts 4 to 5 minutes. When two pulses are spaced only 3 minutes apart, there is an overlap of the two interference periods, resulting in an unusually long reversal.

From the fact that the interference caused by a 1-minute pulse of light is comparable to that caused by a step, we conclude that the interference is not due to the presence of light per se but is instead a result of the growth response to

Table 1. The time of onset and duration of the interference period and the magnitude of the reversal induced by a light stimulus applied during an ongoing avoidance response. Steps were continuous blue light at 315 μW/cm^2; pulses were 1-minute flashes of the same light. Light stimuli were begun 3, 7, or 10 minutes after the barrier stimulus was applied. Values are means ± standard errors of the means. The number of experiments is given in parentheses. Time of onset is the interval between the beginning of the light stimulus and the beginning of the reversal. Interference duration is the duration of reversed bending.

Light stimulus	Time of interference onset (min)	Interference duration (min)	Reversal magnitude (deg)
Step (t = 3)	3.38 ± 0.13 (4)	3.75 ± 0.48 (4)	2.45 ± 0.61 (4)
Step (t = 7)	2.63 ± 0.24 (4)	5.50 ± 0.64 (4)	5.25 ± 1.08 (4)
Step (t = 10)	2.13 ± 0.23	5.00 ± 0.35 (4)	7.75 ± 0.92 (4)
Mean, all steps	2.71 ± 0.23	4.75 ± 0.35	
Pulse (t = 3)	3.00 ± 0.00 (5)	4.33 ± 1.64 (2)	6.25 ± 0.25 (2)
Pulse (t = 7)	2.67 ± 0.17 (3)	4.83 ± 0.66 (3)	8.33 ± 2.19 (3)
Mean, all pulses	2.88 ± 0.08	4.58 ± 0.79	

Table 2. The avoidance latency following light treatments. Dark indicates barrier applied in the absence of blue light; step, barrier applied 1 minute after the beginning of a blue light step at 315 μW/cm^2; and pulse, barrier applied immediately following a 1-minute pulse of the same light. Latency is the interval between application of the barrier and the beginning of avoidance bending. Values are means ± standard errors of the means. The number of experiments is given in parentheses.

Light treatment	Latency (min)
Dark	3.01 ± 0.78 (15)
Step	8.07 ± 0.80 (7)
Pulse	7.13 ± 0.37 (3)

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