The influence of the juvenile hormone analogue (S)-hydroprene on *Aprostocetus hagenowii* (Hymenoptera: Eulophidae), an oothecal parasitoid of the oriental cockroach *Blatta orientalis* (Dictyoptera: Blattidae)

H.A. Bell*, G.C. Marris and J.P. Edwards

Central Science Laboratory, Ministry of Agriculture, Fisheries and Food, Sand Hutton, York, Yorkshire, Y04 1LZ, UK

Abstract

The synthetic juvenile hormone analogue (S)-hydroprene can control populations of the oriental cockroach *Blatta orientalis* Linnaeus. Eradication of *B. orientalis* infestations, however, can take in excess of two years. In an attempt to reduce the time (S)-hydroprene takes to eliminate a population of oriental cockroaches, we explored the possibility of using the oothecal endoparasitoid *Aprostocetus hagenowii* Ratzeburg in combination with (S)-hydroprene. For such a strategy to be successful, it is important that the parasitoid remains substantially unaffected by (S)-hydroprene. When *A. hagenowii* was exposed to *B. orientalis* oothecae in the presence of (S)-hydroprene, female parasitoids showed no reduction in their capacity to attack hosts and their fecundity was not compromised. (S)-hydroprene, at dose rates of 18 mg/m² and 100 mg/m², induced deformity in approximately 12% and 33% respectively of parasitoids that emerged. No reduction in reproductive viability was seen in morphologically normal F₁ parasitoids. Deformed F₁ parasitoids, exposed to the higher (S)-hydroprene dose, showed a 71% reduction in the number of oothecae attacked and a 50% reduction in the number of offspring produced. These results indicate that *A. hagenowii* could be used in combination with (S)-hydroprene in an integrated pest management programme against *B. orientalis*.

Introduction

The oriental cockroach, *Blatta orientalis* Linnaeus (Dictyoptera: Blattidae) is the major cockroach pest in the United Kingdom (Alexander et al., 1991). It occurs in domestic, industrial and institutional premises, and a survey carried out by the UK Department of Health has shown that up to 63% of British hospitals may be infested (Baker, 1990). This species is also a common pest in many other parts of the World. Although control can be achieved with insecticidal sprays and dusts, such as carbamates and synthetic pyrethroids (Bajomi & Elek, 1979; Barson, 1979), or with toxic baits, such as abamectin and hydramethylnon (Koehler et al., 1991; Short et al., 1993), for a variety of reasons, these preparations are not universally effective (Piper & Frankie, 1978). Moreover, increasing public concern about the possibility that harmful side-effects might arise from the use of certain insecticides in domestic, industrial and institutional premises has resulted in a general need to reduce conventional pesticide usage in such situations. Thus, alternative techniques for cockroach control need investigating.

One such alternative technique is the use of insect juvenile hormone analogues such as (S)-hydroprene (ethyl (S)-3,7,11-trimethyl-2(E),4(E)-dodecadienoate) (Edwards &...
This highly-specific compound has negligible vertebrate toxicity (acute oral LD₅₀ (rat) >34 g/kg), making it a particularly desirable candidate for use in situations where other pesticide types may be precluded. (S)-Hydroprene disrupts metamorphosis, leading to deformity and sterility in adults that have been exposed during the final nymphal instar (Bao & Robinson, 1990; Short & Edwards, 1992). The loss of reproductive capacity of the population results in its eventual eradication and, in a simulated domestic environment, applications of (S)-hydroprene have been shown to eliminate semi-natural populations of B. orientalis in approximately two years (Edwards & Short, 1993). The long period of time required for (S)-hydroprene to eliminate a population of B. orientalis is partly due to the relatively long developmental period of the nymphs of this species (by comparison with other pest cockroaches). Additionally, the fact that any adult females present at the time of treatment are unaffected by (S)-hydroprene and remain capable of producing viable oothecae until they die, results in the cockroach population increasing for several months after treatment (Edwards & Short, 1993). There are two possible ways in which the problem of continued production of viable oothecae after (S)-hydroprene treatment can be addressed.

One possibility would be to treat the population with a conventional neurotoxic insecticide in combination with a juvenile hormone analogue, thereby killing extant adult insects to reduce or eliminate the production of oothecae. This method, however, would still require the use of a toxic compound. Alternatively, deployment of complementary biological control agents, specifically directed towards the destruction of oothecae, would target any oothecae produced after treatment and could reduce the time required to completely eradicate the infestation.

The gregarious endoparasitoid Aprostocetus (=Tetrastichus) hagenowii Ratzburg (Hymenoptera: Eulophidae) attacks oothecae of several cockroach species, including B. orientalis (Roth & Willis, 1954; Edmunds, 1955). Broods of parasitoid eggs, sometimes in excess of 100, are laid directly into cockroach eggs within the ootheca and the parasitoid larvae usually consume all the cockroach eggs or embryos within each parasitized ootheca prior to pupation and emergence as adults. Since this leads to the death of embryonic cockroach nymphs (Roth & Willis, 1954; Cameron, 1957), this parasitoid has become the subject of a number of investigations as a biocontrol agent against various cockroach pests (Patterson et al., 1988; Hagenbuch et al., 1989; Pawson & Gold, 1993). The fact that A. hagenowii specifically parasitizes oothecae means that it might be feasible to use this parasitoid in conjunction with (S)-hydroprene, to target those egg cases produced after juvenile hormone analogue treatment. However, because juvenile hormone analogues work by interfering with insect development and reproduction, it is possible that (S)-hydroprene could also adversely affect a number of aspects of parasitoid biology. The following study describes a series of laboratory experiments designed to compare the biology of A. hagenowii on B. orientalis, reared in the presence or absence of two alternative (S)-hydroprene doses. Such information is necessary to allow an initial assessment of the likely efficiency of this parasitoid in an integrated pest management (IPM) programme against this widespread urban pest.

**Materials and methods**

**Preparation of experimental organisms**

Newly-produced oothecae were harvested within 24 h of deposition from a laboratory strain of B. orientalis derived from an initial population originally collected from a London hospital in 1986. This strain was maintained at 27°C and 25% r.h. on a diet of wheatfeed, rolled oats, yeast, fishmeal, dog chow and ground peanuts (14:14:3:6:6:2 w/w).

The strain of A. hagenowii used in these experiments was originally obtained in 1992, from Texas A & M University, USA. This strain was originally reared on oothecae of Periplaneta americana (Linnaeus) (Dictyoptera: Blattidae), but subsequently we have reared the parasitoid on B. orientalis hosts. Parasitoid cultures are maintained through exposing approximately 500 oothecae weekly to approximately 1000 newly-emerged A. hagenowii (males and females). A new generation of A. hagenowii emerge 30–60 days after oothecae were initially exposed to parasitoids. Prior to the experiments, parasitized oothecae were removed from the parasitoid culture and incubated singly in individual glass tubes in order to provide newly-emerged parasitoids which had not had access to any potential hosts. Upon emergence, adult parasitoids were provided with a 50% (v/v) aqueous honey solution as a food source and used within 48 h. All cultures were kept at 25°C, 70% r.h., L:D 16:8.

The effect of (S)-hydroprene on B. orientalis oothecae

Prior to evaluating any effects that (S)-hydroprene-exposed B. orientalis oothecae may have had on the biology of A. hagenowii, it was necessary to investigate the biology of B. orientalis oothecae following their exposure to selected (S)-hydroprene doses. In these trials, (S)-hydroprene treatment was achieved using the method described by Short & Edwards (1992), whereby a non-racemic solution containing 90 g/l of (S)-hydroprene only was mixed with distilled water, to provide concentrations of 0.86 mg and 4.80 mg (S)-hydroprene per ml. Aliquots of each solution (1 ml) were then applied to the top surfaces of two sets of vinyl tiles (area 0.048 m²), and spread evenly over the surface using a small paintbrush which had been previously moistened with the same solution. In this way, vinyl tiles were treated with (S)-hydroprene solutions to give final treatment rates that corresponded to the manufacturer’s recommended treatment rate of 18 mg/m² (S)-hydroprene (lower dose rate) and a much higher dose rate than would normally be used in practice of 100 mg/m² (S)-hydroprene (higher dose rate). In a separate room, a third set of tiles was treated with distilled water only, to serve as untreated controls. In total, 45 tiles were prepared; 15 tiles of each treatment type. All treated tiles were left to dry at room temperature for 24 h, before being laid into the bottom of separate plastic tanks (30 x 17.5 x 21 cm). Ten newly-formed B. orientalis oothecae were placed directly onto the surface of each treated tile. These treatments were designated as direct contact (S)-hydroprene treatments.

(S)-Hydroprene has been shown to be a highly mobile molecule, capable of diffusing from point sources to treat a larger area (Short & Edwards, 1993). In order to ascertain any indirect effects of (S)-hydroprene vapour on cockroach development, a further 15 vinyl tiles were treated with (S)-hydroprene at the higher dose rate and placed in plastic
effects of (S)-hydroprene on Aprostocetus hagenowii

Table 1. The hatch of Blatta orientalis oothecae exposed to (S)-hydroprene but not to Aprostocetus hagenowii.

<table>
<thead>
<tr>
<th>Tile treatment (mg/m²)</th>
<th>Exposure method</th>
<th>Replicates (10 oothecae per replicate)</th>
<th>Mean no. of oothecae hatching ± SE</th>
<th>Mean no. of nymphs emerging per hatched oothecae ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>–</td>
<td>15</td>
<td>8.6 ± 0.3a</td>
<td>15.0 ± 0.2a</td>
</tr>
<tr>
<td>18</td>
<td>Direct</td>
<td>15</td>
<td>8.5 ± 0.3a</td>
<td>14.6 ± 0.2a</td>
</tr>
<tr>
<td>100</td>
<td>Direct</td>
<td>15</td>
<td>9.2 ± 0.2a</td>
<td>15.0 ± 0.1a</td>
</tr>
<tr>
<td>100</td>
<td>Vapour</td>
<td>15</td>
<td>8.9 ± 0.2a</td>
<td>14.7 ± 0.2a</td>
</tr>
</tbody>
</table>

Values followed by the same letter in each column are not significantly different (P > 0.05, single factor ANOVA).

tanks as above. A group of ten oothecae were placed in an open Petri dish (7 cm diam.) which was placed onto each of the treated tiles. These treatments were designated as vapour action treatments.

The control and (S)-hydroprene treatments were conducted in separate rooms maintained at 25°C, 70% r.h., L:D 16:8. The three (S)-hydroprene treatments (lower dose, higher dose and vapour action), although conducted in the same room, were completed at different times to reduce the possibility of cross-contamination of the tiles. During all experiments, oothecae were left exposed to the (S)-hydroprene treated surfaces until they were 44 days old. This allowed the oothecae to be exposed continuously to (S)-hydroprene for the same period of time as oothecae exposed to (S)-hydroprene and parasitoids (see below). Subsequently, individual oothecae were placed into separate glass vials (5 × 2.5 cm) and were inspected daily whereupon the numbers of nymphs that emerged from each ootheca were recorded.

Exposure of A. hagenowii to oothecae in the presence of (S)-hydroprene

Following the same experimental design as above, a further 45 tiles were prepared: 15 were treated with distilled water; 15 were treated with (S)-hydroprene at the lower dose; 15 were treated with (S)-hydroprene at the higher dose rate. All tiles were placed into the base of separate plastic tanks (30 × 17.5 × 21 cm), and a group of ten newly-deposited B. orientalis oothecae was placed onto the surface of each tile. These (S)-hydroprene treatments were designated direct contact treatments. Oothecae were subsequently left undisturbed for the next 14 days, to allow them to reach the age at which they are known to be highly susceptible to parasitoid attack (H. A. Bell, unpublished data). A mating pair of A. hagenowii was then introduced into each tank. The parasitoids were provided with a 50% (v/v) aqueous honey solution as a food source, and the tanks were sealed with a gauze lid to prevent escape.

In order to ascertain any effects of (S)-hydroprene vapour (as opposed to direct contact with (S)-hydroprene-treated surfaces) on parasitoid biology, a further 15 tiles were treated with (S)-hydroprene at the higher dose rate. Groups of ten oothecae were placed on open plastic Petri dishes (7 cm diam.) and one of these was then placed on each treated surface. These (S)-hydroprene treatments were designated as vapour action treatments. Parasitoids were exposed to the oothecae 14 days after placement on the treated surfaces, as described above.

Each pair of parasitoids was left in association with the oothecae for 5 days which is a sufficient period of time for females to complete the majority of their potential attacks (H. A. Bell, unpublished data). After this period, parasitoids were removed and the condition of females, either dead or alive, was noted. Male mortality was not recorded because, under normal circumstances, a large proportion would have died naturally over the 5 day exposure period (Narasimhan, 1984). Subsequently, the oothecae were left in their respective treatment tanks for a further 25 days, giving a total period of exposure to the (S)-hydroprene treated surfaces of 44 days. This interval allowed us to collect oothecae just before they would normally begin to yield parasitoids, while ensuring that they had been subjected to the same period of (S)-hydroprene treatment as that experienced by oothecae used in our first experiment. Oothecae were placed into separate glass vials (5 × 2.5 cm), and inspected at daily intervals for the emergence of A. hagenowii or cockroaches (or both). The following information was recorded: (i) the number of oothecae which produced adult A. hagenowii only; (ii) the number of oothecae which produced both adult A. hagenowii and cockroaches; and (iii) the number of oothecae which did not produce adult A. hagenowii but were found to contain partially-developed parasitoids when inspected after cockroach emergence. In addition to

Table 2. The mortality of Aprostocetus hagenowii and mean number of oothecae attacked under different tile treatment conditions (ten ootheca exposed per replicate).

<table>
<thead>
<tr>
<th>Tile treatment (mg/m²)</th>
<th>Exposure method</th>
<th>No. of pairs of parasitoids</th>
<th>No. of females dead after five days¹</th>
<th>Mean number of oothecae attacked per female parasitoid ± SE</th>
<th>Oothecae from which no parasitoids emerged but which contained parasitoid larvae²</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>–</td>
<td>15</td>
<td>2a</td>
<td>2.00 ± 0.40a</td>
<td>0.40 ± 0.16a</td>
</tr>
<tr>
<td>18</td>
<td>Direct</td>
<td>15</td>
<td>3a</td>
<td>2.60 ± 0.36a</td>
<td>0.20 ± 0.11a</td>
</tr>
<tr>
<td>100</td>
<td>Direct</td>
<td>15</td>
<td>2a</td>
<td>2.06 ± 0.35a</td>
<td>0.13 ± 0.09a</td>
</tr>
<tr>
<td>100</td>
<td>Vapour</td>
<td>15</td>
<td>1a</td>
<td>2.00 ± 0.31a</td>
<td>0.13 ± 0.13a</td>
</tr>
</tbody>
</table>

¹Values followed by the same letter in this column are not significantly different (P > 0.05, chi-square).

²Values followed by the same letter within a column are not significantly different (P > 0.05, single factor ANOVA).
recording the different types of attack, we also monitored the number of cockroach nymphs emerging from each ootheca, the date of emergence of parasitoid offspring, parasitoid brood size per egg case and the total number of offspring produced by each A. hagenowii female. Any signs of (S)-hydroprene-induced deformity in the parasitoid progeny (e.g. twisted wings) were also recorded.

Viability of A. hagenowii emerging from (S)-hydroprene-treated oothecae

Ten non-deformed males and ten non-deformed female parasitoid offspring (<48 h old), were collected from oothecae that had been exposed to each of the (S)-hydroprene treatments. These parasitoids were placed in pairs into (S)-hydroprene-free tanks (in the same way as described above) and presented with ten B. orientalis oothecae (14 days old) for 5 days. The fate of each ootheca was subsequently monitored, and the emergence of parasitoids or cockroach nymphs was recorded. The sex of offspring was noted to determine whether the progeny were produced by mated females (mixed sex broods) or unmated females (exclusively male broods) (Edmunds, 1955). Additionally, where deformed female parasitoids emerged from (S)-hydroprene-treated oothecae, ten deformed females (<48 h old) were chosen randomly and paired with newly-emerged males taken from the laboratory culture. The use of males from the culture was undertaken in order to maximize the likelihood of the deformed females mating. Each pair of parasitoids was subsequently exposed to ten untreated oothecae, as described above, and the outcome recorded.

Results

The effect of (S)-hydroprene on B. orientalis oothecae

The mean number of oothecae that hatched after exposure to the different (S)-hydroprene treatments and the controls was not significantly different (F = 1.29, d.f. = 3, 56, P > 0.05). In all cases, more than 85% of oothecae hatched to give broods of live cockroach nymphs (table 1). The mean number of cockroach nymphs produced per hatched ootheca, approximately 15, was similar in the controls and (S)-hydroprene treatments (table 1). The differences between the mean number of cockroach nymphs that emerged from hatched oothecae in the different treatments was not significant (F = 1.71, d.f. = 3, 525, P > 0.05).

The effect of (S)-hydroprene on the biology of A. hagenowii

The total number of oothecae attacked per female A. hagenowii did not differ significantly (F = 1.21, d.f. = 3, 56, P > 0.05) between any of the respective (S)-hydroprene treatments and the controls (table 2). Typically, irrespective of exposure to either dose of (S)-hydroprene, each female parasitoid attacked 2.0 to 2.6 oothecae during the course of the experiments. In all cases, regardless of treatment, the majority of attacks led to the development of parasitoids only. Female parasitoid mortality over the exposure period ranged from one parasitoid in the vapour action treatment to three parasitoids in the lower dose rate treatment, a difference that was not significant ($\chi^2 = 1.15, P > 0.05$). Females exposed to (S)-hydroprene successfully attacked
Table 4. The deformity of F₁ parasitoids emerging from oothecae exposed to (S)-hydroprene.

<table>
<thead>
<tr>
<th>Tile treatment (mg/m²)</th>
<th>Exposure method</th>
<th>Total no. of deformed parasitoid offspring</th>
<th>Total deformity of offspring (%)</th>
<th>Deformed males (%)</th>
<th>Deformed females (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>–</td>
<td>0.0</td>
<td>0.0a</td>
<td>0.0a(a)</td>
<td>0.00a(a)</td>
</tr>
<tr>
<td>18</td>
<td>Direct</td>
<td>152</td>
<td>11.6b</td>
<td>13.80b(a)</td>
<td>10.2b(b)</td>
</tr>
<tr>
<td>100</td>
<td>Direct</td>
<td>357</td>
<td>33.3c</td>
<td>39.6c(a)</td>
<td>31.7c(b)</td>
</tr>
<tr>
<td>100</td>
<td>Vapour</td>
<td>0.0</td>
<td>0.0a</td>
<td>0.0a(a)</td>
<td>0.00a(a)</td>
</tr>
</tbody>
</table>

Values followed by the same letter in each column are not significantly different (P > 0.05, chi-square analysis of the total number of deformed parasitoids that emerged per treatment).

Viability of A. hagenowii emerging from (S)-hydroprene treated ootheca

Aprostocetus hagenowii that emerged from oothecae which had been exposed to (S)-hydroprene, in both the lower, higher and vapour action treatments, were found to be fertile (table 5) and were able to attack oothecae. Severely deformed F₁ female parasitoids were reproductively viable and, in several cases, produced normal sized broods. However, for parasitoids exposed to the higher dose rate, the mean attack rate, at 0.60 oothecae per parasitoid, was significantly lower than that of the parent females (F = 10.12, d.f. = 1, 23, P < 0.01). Normal A. hagenowii females derived from the lower and higher dose rate treatments attacked a mean of 1.9 and 1.7 oothecae respectively, figures not statistically lower than the parental generations (P > 0.05, single factor ANOVA).

Similarly, deformed A. hagenowii females derived from the lower dose rate treatment attacked a mean of 1.8 oothecae each, which was not statistically different from the attack rate

<table>
<thead>
<tr>
<th>Tile treatment of F₁ parasitoids (mg/m²)</th>
<th>Exposure method</th>
<th>F₁ parasitoid condition</th>
<th>No. of pairs of parasitoids</th>
<th>Female parasitoid mortality</th>
<th>Mean no. of oothecae attacked per parasitoid ± SE³</th>
<th>Mean no. offspring per parasitoid ± SE¹</th>
<th>Sex ratio of F₁ parasitoids (female: male)⁴</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>Direct</td>
<td>Normal</td>
<td>10</td>
<td>2</td>
<td>1.70 ± 0.42a</td>
<td>76.0 ± 13.1a</td>
<td>3.7:1a</td>
</tr>
<tr>
<td>18</td>
<td>Direct</td>
<td>Deformed</td>
<td>10</td>
<td>5</td>
<td>1.80 ± 0.32a</td>
<td>67.3 ± 13.5a</td>
<td>4.3:1a</td>
</tr>
<tr>
<td>100</td>
<td>Direct</td>
<td>Normal</td>
<td>10</td>
<td>1</td>
<td>1.90 ± 0.35a</td>
<td>91.4 ± 10.7a</td>
<td>7.5:1b</td>
</tr>
<tr>
<td>100</td>
<td>Direct</td>
<td>Deformed</td>
<td>10</td>
<td>9</td>
<td>0.60 ± 0.30b†</td>
<td>36.1 ± 16.9b†</td>
<td>7.2:1b</td>
</tr>
<tr>
<td>100</td>
<td>Vapour</td>
<td>Normal</td>
<td>10</td>
<td>0</td>
<td>2.60 ± 0.48a</td>
<td>79.1 ± 15.8a</td>
<td>-</td>
</tr>
</tbody>
</table>

¹Values followed by the same letter in these columns are not significantly different (P > 0.05, single factor ANOVA).
²Chi-squared analysis of the total number of male and female parasitoids produced in each of the treatments.
³Significantly different from parent rates of parasitism (P < 0.05, single factor ANOVA).
⁴Sex ratio of this treatment was not recorded.
of the parental generation \((F = 2.56, \text{d.f.} = 1, 23, P > 0.05)\). F1 \(A. hagenowii\) females from the vapour action treatment, all of which were normal, attacked oothecae in higher numbers, at 2.6 oothecae per parasitoid, than the parental generation that attacked 2.0 oothecae per parasitoid.

With the exception of deformed females that had developed at the higher dose rate, no significant differences \((P > 0.05, \text{single factor ANOVA})\) were seen between the number of progeny produced by the F1 females and their parents. \(Aprostocetus hagenowii\) females taken from the lower dose rate produced, on average, 76 and 67 offspring for the normal and deformed parasitoids respectively. Normal \(A. hagenowii\) females from the higher dose rate treatment produced a mean of 91 parasitoids each, while females derived from the vapour action treatment produced a mean of 79 offspring. Deformed \(A. hagenowii\) females derived from the higher dose rate produced significantly fewer progeny than their parental females \((F = 4.35, \text{d.f.} = 1, 23, P < 0.05)\) and their non-deformed siblings \((F = 11.19, \text{d.f.} = 1, 18, P < 0.01)\). The sex ratios of the F1 offspring were all biased in favour of females, more heavily so in the higher dose rate \((S)\)-hydroprene treatment than in the corresponding lower dose rate treatment (table 5). All reproductively viable F1 females, both deformed and normal, produced mixed broods of male and female offspring.

The mortality of F1 female parasitoids during the five day exposure period was highest among deformed females, particularly in those that emerged from oothecae exposed to the higher dose rate \((S)\)-hydroprene treatment, where nine out of the ten females died within 5 days. However, of the F1 females that died during this experiment, four successfully attacked oothecae prior to death, whilst five females died without successfully parasitizing any oothecae. Similarly, five out of ten deformed F1 females taken from the lower dose rate \((S)\)-hydroprene treatment died during the 5 days exposure period, although three of these successfully attacked oothecae prior to death. Of the non-deformed females derived from the lower and higher treatments, two females died over the 5 days from those taken from the lower dose and one died from those taken from the higher dose. All F1 parasitoids derived from the vapour action treatment survived 5 days (table 5).

**Discussion**

In the present study, we found that when \(A. hagenowii\) was exposed to \((S)\)-hydroprene, adult female parasitoids did not show any reduction in their capacity to attack and parasitize oothecae of \(B. orientalis\). \(Aprostocetus hagenowii\) larvae successfully developed to adults in \((S)\)-hydroprene-treated oothecae and were reproductively viable in most cases, although some detrimental effects were observed.

In \(A. hagenowii\) that developed within oothecae in direct contact with \((S)\)-hydroprene-treated surfaces, at both the lower and higher doses, deformity was induced in a proportion of the offspring that emerged, while parasitoids that developed in oothecae exposed to \((S)\)-hydroprene vapour only exhibited no deformity. Deformity was seen in both sexes although male parasitoids were apparently more susceptible to the effects of the \((S)\)-hydroprene than females. Deformed females that emerged from \((S)\)-hydroprene-treated oothecae were viable in most cases, and capable of mating, suggesting that the morphogenetic disruption was probably marginal. Deformed females were, however, paired with non-deformed males taken from laboratory culture and the effect of \((S)\)-hydroprene on the ability of deformed males to mate was not ascertained. The reduced host-attack rate, observed in the deformed F1 females obtained from oothecae in direct contact with the higher dose rate \((S)\)-hydroprene treatment, was probably due, in part, to the fact that several of the parasitoids failed to live for 5 days, and died before achieving their full parasitic potential. The effect that the elimination of flight in deformed females parasitoids would have had on the location of hosts, and the subsequent production of new generations of parasitoids, was not investigated. However, it is probable that flightless, deformed female parasitoids would have reduced foraging ability.

The developmental period of parasitoid broods (measured as the period from the first exposure of \(A. hagenowii\) to oothecae up to the emergence of live offspring), and the numbers of \(A. hagenowii\) emerging from parasitized \((S)\)-hydroprene-treated oothecae, were within previously reported limits (Roth & Willis, 1954; Cameron, 1957). The longer developmental time of the control parasitoids, however, suggests that the presence of \((S)\)-hydroprene may shorten the pre-adult lifespan of this parasitoid. However, the preponderance of all-male broods that occurred in the control replicates probably contributed to the comparatively long mean developmental time of parasitoids in this treatment, as exclusively male broods take significantly longer to emerge than mixed sex broods (H.A. Bell, unpublished data).

Exposure to \((S)\)-hydroprene appeared to increase the female bias of the sex ratio of the parasitoid offspring. Sex determination occurs in the Hymenoptera by haplodiploidy, whereby unfertilized eggs give rise to males, and fertilized eggs give rise to females. However, the ratio of male and female parasitoids, although determined by how many eggs the female parasitoid fertilizes, may be influenced by environmental conditions (Godfray, 1994). Exposure to \((S)\)-hydroprene may indeed have contributed to the female bias seen in the \(A. hagenowii\) adults that developed from \((S)\)-hydroprene-treated oothecae. However, this seems unlikely as the sex ratio of \(A. hagenowii\) offspring is usually female biased (Roth & Willis, 1954) and, furthermore, the male dominated sex ratio apparent in the controls could have been a result of the failure of a proportion of the female parasitoids to mate. Unfertilized females frequently occur in laboratory populations of \(A. hagenowii\) (H.A. Bell, unpublished data) although the reason for their prevalence in our control replicates was not readily apparent.

The effects of various juvenile hormone analogues on parasitoids have been widely examined (Vinson, 1974; Hamlen, 1975; McNeil, 1975; Lawrence et al., 1978; Beckage & Riddiford, 1981; Abd El-Kareim et al., 1988; Peleg, 1988). Relatively few of those parasitoid species which have been studied develop completely normally in the presence of juvenile hormone analogues (Wright & Spates, 1972; Wilkinson & Ignoffo, 1973; Guerra et al., 1977; Peleg, 1988). In many cases, exposure of parasitoids to juvenile hormone analogues, either directly or indirectly via the host, has been seen to be detrimental, sometimes severely so (Poe, 1974; Grannett et al., 1975; McNeil, 1975; Smilotwitz et al., 1975; LeMA et al., 1978). The nature of negative effects depends on the life stage of the parasitoid at the time of treatment, as well as the method of application of the juvenile hormone analogue (Riviere, 1975; Smilotwitz et al., 1975; De LooL et al., 1979; Fashing & Sagan, 1979). Acute symptoms include high
mortality in developing parasitoid larvae, deformity in adults, or a complete failure of parasitoids to eclose from the pupal stage (Poe, 1974; Hamlen, 1975; McNeil, 1975). More subtle effects, such as altered developmental time (Vinson, 1974; Grannett et al., 1975), reduction in egg size and viability (Wissinger & Grosch, 1979), and termination of diapause (Ascerno et al., 1980), have also been recorded. Moreover, exposure of the developing stages of parasitoids to juvenile hormone analogues has also been shown to reduce the fecundity of the resultant F₁ adult parasitoids, as well as that of subsequent (unexposed) generations (Ascerno et al., 1980).

Aprostocetus hagenowii, in the experiments described, remained relatively unaffected by exposure to (S)-hydroprene, particularly when developing parasitoids were exposed to the vapour only, or to the lower (direct contact) dose. Given the low incidence of deformity in adult parasitoids, and the fact that neither the number of oothecae attacked, nor the number of progeny subsequently emerging, was markedly affected by the presence of the lower dose of 18 mg/m² (S)-hydroprene, it seems that A. hagenowii could be used in conjunction with this juvenile hormone analogue to control populations of B. orientalis. Moreover, the fact that most of the parasitoids that developed in the (S)-hydroprene-treated oothecae were reproducibly viable suggest it may be possible to use A. hagenowii as an inoculative biological agent in an integrated programme with (S)-hydroprene.

We conclude that A. hagenowii shows potential as a biological control agent that could be used in combination with the (S)-hydroprene, to achieve effective control of B. orientalis populations. Such a combination could possibly reduce the time required for the elimination of a cockroach population when (S)-hydroprene is used alone. Furthermore, the combined use of a juvenile hormone analogue and a biocontrol agent would be compatible with the minimization of toxic pesticide use in domestic and industrial environments.

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