The compatible combination of resistance monitoring with population level monitoring would aid the grower by allowing more judicious use of insecticides and use of a material that remains effective at low application rates. This technique is ideally suited for *P. gossypiella* where treatments are targeted at the adult stage[13]. Resistance monitoring with sticky traps can be adapted to other insecticides, to other species that use pheromones in mate-location, to species that rely on specific host odors for food location, and to insects that orient to well-defined visual cues. In fact many of the major agricultural pests are amenable to monitoring of insecticide resistance using traps. For many species it will be important to establish the relationship between the toxicity of the insecticide to the adult and larval stages, since resistance may only be found in the targeted stage[14]. The need to preserve effective and environmentally sound pest control tactics is of paramount importance, and is providing the impetus for management of insecticide resistance.

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Ivermectin prevents head eversion in the blowfly *Calliphora vomitoria* L.

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Summary. Failure to complete adult development after treating final-stage larvae of *Calliphora vomitoria* with ivermectin is due mainly to the inhibition of head eversion in the pupal stage.
Key words. Diptera; head eversion; ivermectin; larvae; metamorphosis.

Ivermectin, a product of the soil microorganism *Streptomyces avermitilis*, is used against nematode and arthropod parasites of livestock[1]. It acts upon inhibitory neuromuscular synapses, stimulating the release of the transmitter gamma-aminobutyric acid (GABA), enhancing the binding of GABA to muscle membranes, resulting in hyperpolarisation of the muscle[1,2]. 1-2 µg of ivermectin prevents final-stage, post-feeding larvae of *Calliphora vomitoria* from forming puparia, while 0.1 µg allows pupariation but hinders adult development[3]. The neuromuscular events in pupariation[4] might be expected to be susceptible to ivermectin, but the disruption of adult development is obscure. This paper shows why development fails after ivermectin treatment.

500 post-feeding larvae of *Calliphora*[3] were used 2 days before pupariation. 100 were untreated; 100 were given 1 µl of ethanol; 300 were given 0.3 µg ivermectin in 1 µl ethanol applied topically to the posterior region of the abdomen. The larvae were allowed to form puparia in glass jars at room temperature which took about 14 days. While most of the controls completed development, only 48% of the treated insects produced flies. The remaining puparia were dissected to assess adult development, and the observations are shown in the table.

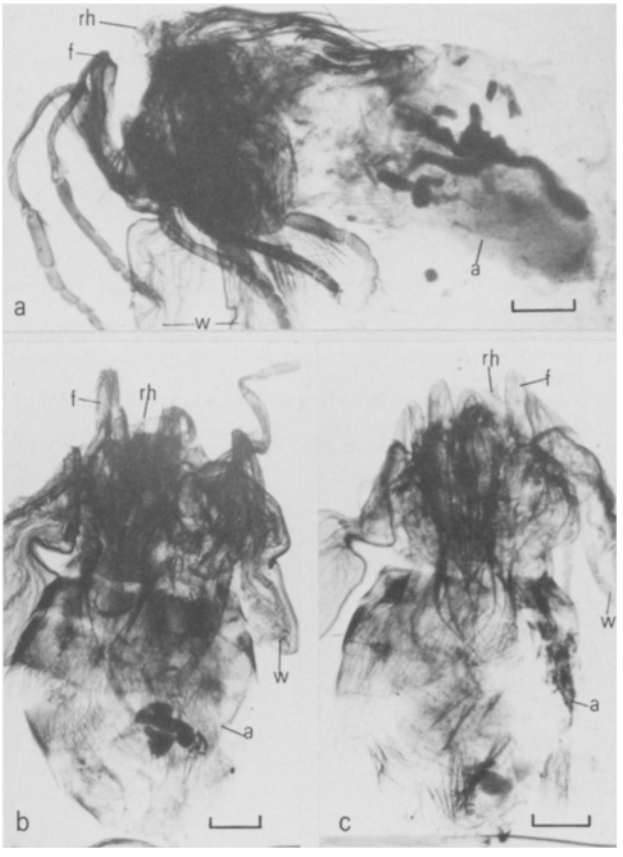
Untreated and ethanol-treated controls produced adult flies almost completely, but 52% of the puparia from ivermectin-treated larvae failed to do so. 2% had died, as had 2% of the ethanol-treated controls: no significance is attached to this finding. 15% of the puparia contained normal-looking pharate adults with a functional ptilinum and moving limbs. Whether these flies would have emerged eventually is not known, but as similar delayed adults were not found in the controls, ivermectin clearly impedes the emergence of flies that otherwise appear to be normal.

A dramatic finding was that 35% of the puparia contained flies that lacked heads (fig.). The abdomen and thorax (with wings and legs) were present, but nothing anterior to the respiratory horns. This was not simply a deformity of the head: the latter was not present as such in any of these insects. Some abdomens were white and pupal-like (fig., a), while others were clearly adult, segmented, and covered with setae (fig., b and c). A constant feature was that the insects were packed into the anterior part of the puparium, with the thorax occupying the region where the head should have been: this left a space posterior to the abdomen.

Developmental fates *Calliphora vomitoria* treated with 0.3 µg ivermectin as final-stage, post-feeding larvae

	Puparia produced	Undeveloped adults (%)	Headless adults (%)	Pharate adults (%)	Normal adults (%)
Untreated controls (100)	99	0	0	0	100
Ethanol-treated controls (100)	99	2	0	0	98
Ivermectin-treated (100)	294	2	35	15	48

Ivermectin was given topically in 1 µl ethanol. Ethanol-treated controls were given ethanol only. Numbers in parantheses indicate number of larvae treated.



Whole mounts of headless flies from pupuria formed after treating post-feeding, final-stage larvae of *Calliphora* with 0.3 µg ivermectin in 1 µl ethanol. Scale = 1 mm. *a* Side view showing advanced thorax terminating at respiratory horns: abdomen is pupal-like. *b* Ventral view showing respiratory horn between the front femora: abdomen is more advanced and segmented. *c* Dorsal view showing complete thorax and well-developed abdomen with long setae. a, abdomen; f, front femur; rh, respiratory horn; w, wing.

This curious finding can be explained. Fraenkel[5] described vigorous peristaltic activity in newly formed pupal abdomens, activity resulting in the rapid eversion of the head from the thorax. At this early stage in metamorphosis, the peristalsis must be caused by larval muscles. Pupae dissected from the puparia of ivermectin-treated larvae do not show this muscular activity (unpublished observations). Since ivermectin has known effects upon inhibitory neuromuscular synapses[2], it is not unreasonable to attribute the lack of muscular activity to the presence of ivermectin in the treated insects. However, there are GABA binding sites in the central system of insects[6], and ivermectin might also exert its effects on muscular activity through the central nervous system.

Since headless insects develop in the wrong location in the puparium, one function of head eversion might be to align the pupa in the correct position. Even so, the insects can still progress to an advanced stage in the wrong place. Whether the 15% pharate adults that remain within the puparia is due to residual action of ivermectin on adult muscles is beyond the scope of this paper, although it is unlikely that ivermectin would prevent adult emergence without affecting head eversion at the earlier stage. In conclusion, this work demonstrates how ivermectin can prevent adult development during metamorphosis in *Calliphora*, and attempts to explain the inhibition in terms of known effects of ivermectin.

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Structure of minor host-selective toxins from *Cochliobolus victoriae*

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Summary. Structures **2**–**5** have been assigned to four minor host-selective toxins from *Cochliobolus victoriae*, namely victorin B, victorin D, victorin E, and victoricine, by using as a lead the established structure **1** of victorin C, the main component of the toxin complex.

Key words. Blight of oats; host-specific phytotoxins; *Cochliobolus victoriae;* victorins, victoricine.

In a previous communication on the host-selective toxins produced by *Cochliobolus victoriae*, the causal agent of victoria blight of oats, we described the isolation of five fractions displaying host-selective toxic activity which were named victorin A, B, C, D and E, respectively, according to their order of elution from a reversed phase HPLC column[2]. Preliminary chemical examination of the most abundant compound, victorin C, which is probably identical with the toxic preparation reported by two other groups[3,4], revealed the presence of six subunits, namely 5,5-dichloroleucine (Cl_2leu)[2,5], erythro-β-hydroxyleucine (OHleu)[2,5], threo-β-hydroxylysine (OHlys)[2,5], α-amino-β-chloroacrylic acid (aClaa)[2], victalanine (victala)[2], and glyoxylic acid[2]. Further work on victorin C and its degradation products eventually led to the establishment of the unusual structure **1** for the toxin[6]. We now report on the structure of the minor toxic companions of victorin C.

The fractions from the RP-HPLC column previously designated as victorins A, B, and E were found homogeneous when analyzed by TLC and NMR techniques; in contrast, NMR analysis of fraction D revealed it to be a mixture of two components which could be resolved into the homogeneous compounds by TLC (table 1). To avoid upsetting the original alphabetical nomenclature we have retained the designation victorin D for the compound with R_f 0.59 and have named the other compound with R_f 0.83 victoricine.

The main toxin, victorin C, was obtained routinely in yields of