SQUEEZING THE MID GUT OF PARASITIZED Spodoptera Littoralis LARVAE BY PARASITOGENIC TRACHEOLS

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ABSTRACT

The mid gut of the parasitized larvae was decreased in diameter compared with unparasitized ones. Th epithelial cells were loose, full with vacuoles, turned to folds more thick and faintly stained. The tracheals occupied a considerable space of the perivesceral protein of the body cavity of the parasitized larvae. The parasitized Spodoptera littoralis larvae showed parasitogenic degeneration in their fat bodies.

INTRODUCTION

Microplitis rufiventris, a solitary endoparasitoid, is considered an important and efficient agent in controlling the population of the cotton leaf worm. This braconid parasitoid can also attack other Lepidopterous hosts, but hosts from genus Spodoptera are the most preferred. Hymenopterous endoparasitoids are known to cause many physiological changes in their hosts in action known as "host regulation". This regulation alters many aspects of host physiology and ontogeny (Harvey, 1996). In some host parasite system it includes an acceleration in host growth rate/or assimilation efficiency by the host (Slansky, 1978, Cloutier and Mackauer, 1979 & 1980, Alleyne and Beckage, 1997).

In contrast with other systems; feeding and locomotory activities are inhibited (Adamo et al., 1997; Beckage and Riddiford, 1978; Beckage and Templeton, 1986; Eid et al., 2001), novel virally encoded haemolymph protein are produced (Harwood and Beckage, 1994; Harwood et al., 1994), the relative amounts of several endogenous host haemolymph protein are altered (Beckage and Kanost, 1993), and pathological changes in the host fat body are found (Dahlman and Vinson, 1980; Lardner and Hales, 1990; Zhang et al., 1991).

However, the changes in host tissues associated with the parasitoid's development have seldom been documented. In addition, the cause of such changes is poorly understood. This work describes the histopathological changes observed in the parasitized S. littoralis larvae and discusses these changes. Relatively few studies have focused on the effects of parasitoid on feeding and growth of hosts, particularly in those koinobiotic species.

MATERIALS AND METHODS

Larvae of the host Spodoptera littoralis were reared in glass jars (15x15x20cm.), at room temperature. The larvae were fed fresh castor leaves washed and sterilized by formaldehyde 0.25-0.5%. The parasitoid M. rufiventris was reared on early third instar larvae of the host in glass rearing units, where droplets of diluted honey were scattered.
on the inner walls as a source of food for the adult parasitoids. Twenty early third instar larvae were introduced daily to one fertilized female wasp every 24 hours.

Parasitized larvae were then transferred to clean pots and reared on sterilized castor leaves till parasitoid egression. The rearing process of adult parasitoid was carried out in an incubator under controlled conditions of temperature (20±5°C), humidity (60-70% R.H.) and a 9:15 L/D photoperiod regime.

The parasitized larvae were histologically studied to investigate the effect of the endoparasitoid on hosts integument and tracheal system. It was observed that some parasitized larvae attack their parasitoid and these individuals were considered as a category from the normal parasitized (PI) and are referred to as (PII). Three replicates were taken for each category (unparasitized, PI and PII) and longitudinal sections were made in the thoracic and abdominal regions after fixing in aqueous buin and then drying in descending alcohol concentrations. The sections were then double embedded in celloidin and paraffin wax. The 7μ sections were stained with Relish's hematoxylin for staining the nucleus and Eosin for staining the cytoplasm.

RESULTS AND DISCUSSIONS

1- Effect on the mid gut:

The parasitized larvae become sluggish and loses its appetite gradually until it finally stop feeding about two days before egression of the parasite. However, it was not easy to differentiate between the parasitized and nonparasitized larvae on basis of size as no obvious differences were detected.

The histological inspection showed that the mid gut in the parasitized larvae was less in diameter than in the unparasitized ones. It's lumen was nearly empty of food material or residue (Figs. 1 & 2). In the unparasitized larvae (Fig. 1), it is noticed that the epithelium is consisted of a single layer of columnar cells as reported by House (1965), with microvilli forming a striated border (Chapman, 1973), and lined with an inner layer of circular muscles and outer longitudinal one (Wigglesworth, 1972). The epithelial cells were deeply stained without vacuoles. Some granules are seen in the cytoplasm of the cells and others were liberated in the lumen close to the inner border of the epithelial cells and considered as digestive enzymes.

Figure (2) shows the mid gut of parasitized larvae where the epithelial cells are faintly stained, compared with those of the unparasitized larvae, due to the decrease in the digestive enzymes. The epithelial cells appear to be loose and separated and the lumen is full with gaps and vacuoles. In the mid gut of the parasitized larva; the circular and longitudinal muscles are detached from the epithelial layer, the epithelial layer turns into adjacent folds. By measuring the thickness of the epithelial layer in parasitized and nonparasitized larvae (Table, 1), it was 122.1 μ and 119.4μ, respectively. The differences were not significant (Table, 1).
Figure 1: Longitudinal section in the mid gut of a healthy larva of *S. littoralis* (10x)
Epth: Epithelial cells, em: Epithelial muscles, L: Mid gut lumen, g: Ganglion

Figure 2: Longitudinal section in the mid gut of a parasitized larva of *S. littoralis* (10x)
Epth: Epithelial cells, em: Epithelial muscles, T: trachea, g: Ganglion
**Figure 3** Longitudinal section in the fat body of a healthy larva of *S. littoralis* (40x)

**Figure 4** Longitudinal section in the fat body of a parasitized larva of *S. littoralis* (40x)

- **l**: Integument
- **m**: Muscles
- **T**: Trachea
- **f**: Fat body
The most striking feature in the sections of the parasitized larvae is numerous sections of tracheels which occupy a considerable space of the section approximating that of the mid gut. It may be of concern to note that the sections of the unparasitized larvae are devoid of these sections of tracheels and the mid gut consequently occupies, its normal size with a lumen full of foodstuff (Figs. 1 & 2). The diameter of the mid gut in the unparasitized larvae may thus exceed two folds the diameter of the parasitized ones.

Endoparasitoids induce physiological and behavioural changes in their hosts, many of which can be considered as regulation of host environment to meet the needs of the parasitoids (Horton and Moore, 1993; Godfray, 1994). In S. littoralis/M. rufiventris system, known effects of parasitism include growth inhibition (Tawfik et al., 1977; Hegazi et al., 1991), behavioural changes such as the pre-emergence cessation of feeding and motor activity (Eid et al., 2001). These effects are induced by the polydnavirus of endoparasitoid females injected into hosts along with their eggs, other factors such as teratocytes and venom may also be important (Beckage, 1993a & b; Dahlman and Vinson, 1993; Levine and Beckage, 1995 & 1996; Jones and Coudron, 1993; Wani et al., 1993; Beckage et al., 1994; Strand and Pech, 1995).

In the S. littoralis / M. rufiventris system the host continues to feed after parasitization. But it gradually lose appetite and the decline in food uptake reach's its maximum two or three days before egression of the parasitoid. Similar to many parasitized insects larvae of S. littoralis cease feeding prior to parasite emergence until their death (Smilowitz et al., 1976; Hegazi et al., 2001). As reported by Hegazi et al., (1988) "The developmental stress of the parasite has an effect on the muscles of the host’s gut and its muscles lose the ability to process food. Indeed some food remained inside the alimentary canal of these parasitized hosts following cessation of food consumption". But the findings of this work show other view.

We suggest that the mechanical effect of the growing mass of tracheels in the mid gut is responsible for the gradual loss of appetite. The perivisceral protein of the body cavity available to mid gut extension by food injection become occupied by these tracheels or air sacs. This immense increase in the respiratory mass developed as consequence of the growing need for oxygen owing to parasitism as shown by Alleyne et al., (1997).

The lack of mid gut tissues damage makes such a suggestion acceptable. The alterations of the tracheal system in the parasitized larvae are not only an association but also a causal phenomenon. It may be of concern to point out the probable effect of the polydnavirus, teratocytes and venom in participating in losing appetite.
Table 1: Average values of the thickness of the epithelial layer of the mid gut of *S. littoralis* larvae parasitized by *M. rufiventris*.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Thickness of the epithelial layer (in microns)</th>
<th>Analysis of variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control larvae</td>
<td>119.8</td>
<td>F. 0.39 P. 0.54 F. critical 3.97</td>
</tr>
<tr>
<td>Parasitized larvae</td>
<td>122.1</td>
<td></td>
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</tbody>
</table>

Each figure is the average of three replicates, each of 24 readings. The differences were not significant.

2- Effect on the Fat body:

In the healthy larvae, the fat bodies (Fig. 3) were numerous and contained large cells with dense granular cytoplasm, indistinct cell margins, and a small rounded nucleus. The cytoplasm was deeply stained and lightly vacuolated.

The fat bodies of parasitized larvae were less in their mass (Fig. 4) and the staining was faint. The cytoplasm of the cells became less dense and contained numerous clear patches, probably representing fat droplets. In the parasitized larvae the fat bodies showed parasitogenic degeneration and increased vacuolation although it kept existing.

For assessing the pathological consequences of parasitism on the host, the histological measure of parasitism cost on the fat body level may, in our view, provide a parameter for the evaluation of the overall metabolic stress induced by parasitism. The parasitized *S. littoralis* larvae showed parasitogenic degeneration through faint staining, less dense cytoplasm, clear patches in the cytoplasm, increased vacuolization, and less mass of fat bodies (Fig 4).

The successful parasitism may be linked with synthesis of “early” proteins (Harwood, 1993; Harwood et al., 1993) produced by the fat body and haemocytes (Dunn, 1985; Harwood and Beckage, 1993). Several studies suggested that qualitative and quantitative changes in lipid metabolites accompany parasitism (Barras et al., 1970; Thompson, 1982; Hayakaw, 1986&1987).

The fat body of parasitized larvae almost glycogen depleted (Dahlman and Vinson, 1980). Altered host tissue carbohydrate levels have also been reported during many insect-parasite host relationships (Fuhrer, 1972; Hawlitzky and Boulay, 1986; Thompson et al., 1990). The host fat body curtailment may be due to parasite induced nutrient deprivation resulting in increased fat body catabolism and decreased anabolism (Tomalak *et al*., 1990 and Lieutier, 1984 a and b). It could be suggested that these effects are consequences of the parasitic-induced stresses on the mid gut and losing appetite.
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انكماش مدة برقات دودة ورق القطن بسبب ضغط قدصاتها الهوائية المتضخمة

. *Microplitis rufiventris*

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أوضح الفحص المستوحى لمدة برقات دودة ورق القطن المتطفل عليها صغر حجمها

بالمقارنة بالبرقات غير المتطفل عليها وكانت خلاياها الطلائية مفتككة ومتلازمة وممتلئة بفجوات القصصات الهوائية تشغل حيز كبير من الحجم الداخلي للبرقات المتطفل عليها. كانت الأجسام الدهنية للبرقات المتطفل عليها قليلة.

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