HOST REGULATION BY ENDOPARASITIOD Microplitis rufiventris

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ABSTRACT

The Microplitis rufiventris-Spodoptera littoralis system provides an obvious parasitoid host regulative system. The parasitized larvae were histologically studies to examine the integument and tracheal system, which were capitalized for the own benefit of the parasitoid.

The thickness of the integument was reduced 8% and 32% in the thoracic and abdominal regions of the parasitized larvae. Also, the integument had curled twisted cuticle, diminished hypodermal cells with small nuclei. In the unparasitized larval integument the ecdysial space was formed while in the parasitized larvae the apolysis was blocked and host larvae died few days after parasitoid egression.

The haemocoele of parasitoid larvae was decreased in diameter and was full with trachea higher in number and larger in size compared with the unparasitized larvae.

INTRODUCTION

Parasitization by several parasitic insects has been shown to cause hormonal changes in their hosts. The relationship between host and parasitoid has been considered in terms of effects of parasitism on host growth and development, parasitism (Vinson, 1972; Strand et al., 1988; Dushay and Beckage, 1993; Beckage et al., 1994) or the larval parasitoid's growth and development response to physiological state of the host (Vinson and Iwantsch, 1980; Gunsena et al., 1989; Sequeira and Mackauer, 1992; Harvey et al., 1994).

Parasitism alters host physiology (Dover and Vinson, 1990; Strand and Dover, 1991; Pennachio *et al.*, 1994). Lawrence(1986; 1990) classified endoparasitoids into two groups. "Regulators" that alter host development to increase its suitability for parasitoid by injection of factors from the calyx and /or poison glands (Dover and Venson, 1990; Strand and Dover, 1991; Dushay and Beckage, 1993). Secretions released by larval parasitoids may also regulate host growth. Alternatively, "conformers" are parasitoids which synchronize their physiology and development with those of the host (Lawrence, 1986; Hegazi *et al.*, 1988; Ohnuma and Kainoh, 1989).

The braconid larval endoparasite *Microplitis refiventris* (Kok.) parasitizes wide range of *Lepidopterous* insects preferring larvae of *Spodoptera littoralis* (Boisd). This investigation examines the effect of the parasitoid on the integument and tracheal system of that host. We aim to determine the state of regulation or conformation in the parasitoid/host system examined.

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 $(20\pm5C^{\circ})$, 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 longitudual sections were made in the thoracic and abdominal regions after fixing in aqueous buin and then drying in ascending alcohol concentrations. The sections were then double embedded in celloidin and parafin wax. The 7u sections were stained with Relish's heamatoxylin for staining the nucleus and Eosin for staining the cytoplasm.

RESULTS AND DISCUSSIONS

1- Effects of parasitism on the integument.

Data presented in table (1) show that the mean thickness of the integument in the thoracic region were 20.5, 18.5 and 18.3 u for the unparasitized, PI and P II, respectively. The corresponding values for the abdominal region were 20.4, 14.1 and 13.7 u. The reduction in the thickness of the integument caused by parasitism reached 8% and 32 % for thoracic and abdominal regions, respectively. From table (2) the differences between control and (PI, PII) were significant while between PI and PII it were not.

The microscopic examination showed that the integument of the parasitized larvae had curlied and twisted cuticle layer (Figs 1 and 2). Also, the hypodermal cells were diminished in size with very small nuclei and consequently, the hypodermics layer was thin. The unparasitized larvae had regular thick hypodermal layer with hypertrophied cells having distinct nuclei. It may be of critical value, that in the unparasitized larval integument the ecdysial space was formed and the moulting fluid had already worked causing relatively thinner cuticuler layer in unparasitized compared with parasitized larvae, despite that the whole integument was thicker in the former than the latter. It is evident that apolysis in the parasitized larvae was blocked and host larvae died few days after parasitoid egression.

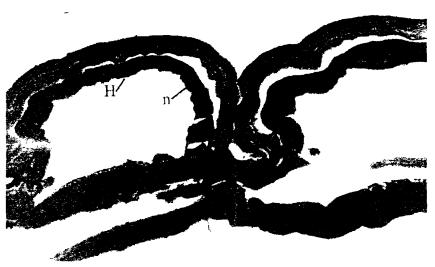


Figure (\mathcal{J}) Longitudinal section in the integument of a healthy larva of S. littoralis (40x)

Cu. Cuticle H Hypodermis

Cu. Cuticle H Hypodermis m: Muscles



Figure ($\frac{2}{S}$) Longitudinal section in the integument of a parasitized larva of $\frac{2}{S}$. littoralis $\frac{2}{S}$ (40x)

S. littoralis (40x)
Cu Cuttele H Hypodermis Muscles F fat body

Parasitized *S. littoralis* were unable to initiate larval apolysis due presumably to: (a) their inability to secrete molting gel and (b) high ecdysteriod titre that may have inhibited the synthesis of a new cuticle. Many reports suggest that parasitoid can produce their own ecdysteriods for their moult, but also they might control their host's moult by releasing ecdysteroids (Jones, 1985 & 1986), Grossniklaus-Burgin and lanzerin (1990), Brown *et al.*, (1988), Brown and Kainoh (1992). Some parasitoids may cause stage specific degeneration of host prothoracic glands (Dover and Venson, 1990) to establish sole control of host apolysis while others may influence the release of PTTH (Dahlman *et al.*, 1990); Tanaka and Venson, 1991).

Table (1): Average values of the integument thickness (in microns) for the different treatments of the Spodoptera littoralis larvae parasitized by the parasitoid Microplitis rufiventris

Treatment	Thoracic Integument	Abdominal Integument 20.4 14.1		
Control larvae	20.5			
Parasitized larvae PI	18.5			
Parasitized larvae attacking their parasitoid PII	18.3	13.7		

Each figure is the average of three replicates (each replicate has 21 readin

Table (2): Analysis of variance for the effect of the parasitoid *Microplitis* rufiventris on the integument thickness for the different treatments of its host *Spodoptera litoralis*

	Integument of the thoracic region				Integument of the abdominal region				
	F	Р	F	Significance of	F	Р	F	Significance	
		value	critical	Differences		value	critical	of Differences	
Control &PI	5.3	0.02	3.9	Significant	68.2	1.8	3.9	Significant	
Control &PII	7.2	0.01	3.9	Significant	81.9	2.4	3.9	Significant	
PI & PII	0.1	0.75	3.9	Not Significant	0.35	0.6	3.9	Not Significant	
Control &PI&PII	4.1	0.02	3.0	Significant	54.7	2.1	3.04	Significant	

Control: Healthy larvae

PI: Parasitized larvae

PII: Parasitized larvae attacking their parasitoid

F: Fisher value

2-Effect of parasitism on the tracheal system

The microscopic examination of the longitudinal sections of the parasitized larvae revealed that the haemocoel was decreased in diameter and completely full with circular and oval cavities that were absent in the sections of the unparasitized larvae (Figs 3 & 4). By examining these cavities, it was found that their inner space is empty. The interna of the walls of these cavities is similar to the structure of the cuticle having ciliated protrusions (tanidia). Figurs 5 and 6 show the normal tracheal system in healthy larvae and that of parasitized ones, respectively. These observations suggest that these cavities are sections of the trachea of the host larvae which has been stimulated by the presence of the parasitoid to increase in number and size to meet the oxygen needs of both the host and parasitoid.

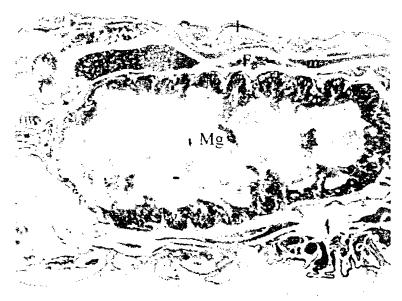


Figure (3) Longitudinal section in a 4th instar larva of Spodotera littoralis showing a general view for the abdomen of a healthy larva (4x) t Integument Mg Mid gut G Female Gonads g Ganghon F tat body m Muscles

G. Female Gonads m. Muscles

F m 😁

Figure (4) Longitudinal section in a 4th instar larva of S. littoralis showing a general view for the abdomen of a parasitized larva (4x)

I Integument:

My Mid gut
F trachea
g Ganglion
F fat body
Muscies



Figure (5) Longitudinal section in the tracheal system of a parasitized larva of S. littoralis (40x)

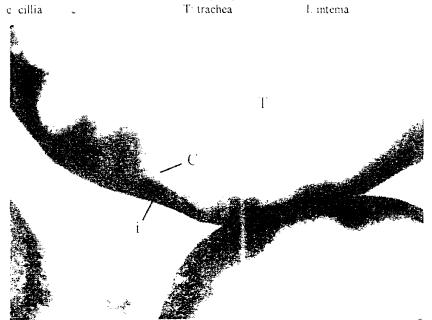


Figure (6) Longitudinal section in the tracheal system of a parasitized larva of S. littoralis (100x)

c cillia T trachea I interna

It appears from the present results that the interaction between the parasitoid *M. rufiventris* and its host *S. littoralis* as a parasite-host system is significantly a type of regulating the host growth in order to retain it in a particular state. In causing its effects, parasitoides use all factors, namely, virus, venom and teratocytes. In the system *M. creceips-H. virescens*, injection of calyx fluid inhibited growth and development as well as JHE activity (Dover and Vinson, 1990).

The effects of endoparasitoid on host development are numerous and diverse (Vinson and Iwantsch, 1980; Beckage, 1985 & 1990; Lawrence, 1986&1988). In most cases it is not known whether parasitoid directly interfere with the host's endocrine system or weather its effects are indirect, caused by stress and/or influences on the nutritional status of the host. Feeding of the parasitoid larvae depletes the host's nutrients and those nutritional changes likely alter the endocrine milieu of the host, thereby impacting the parasitoid. Endoparasitoides react to the prevailing host environment to minimize damage to themselves and capitalize on those nutritional and physiological conditions that will ultimately enhance their own fitness.

In conclusion the *M. rufiventris* endoparasitoid regulated its host, as regulators in the opinion of Lawrence (1986 & 1990)are parasitoids that disrupt the normal metabolic and endocrine processes of their hosts whose micro environmental conditions are suboptimal for parasitoid growth and development. In the *M. rufiventris –S. littoralis* system, the cuticle as well as the tracheal system, as a continous organ, were regulated by the parasitoid for its own benefit.

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تنظيم العائل بو اسطة الطفيل الداخلي Microplitis Rufiventris محمد احمد عيد - جمال السيد - صلاح المعصراوى - حنان عبد الصمد قسم الحشرات الاقتصادية والمبيدات - كلية الزراعة - جامعة القاهرة

تم فحص كل من جدار الجسم والجهاز القصبي هستولوجيا ليرقبات دودة ورق القطن المتطفل عليها. أوضح الفحص الهستولوجي انخفاض سمك جدار الجسم في منطقتي الصدر والبطن بمعدل ٨ و ٣٦ % على التوالي, كذلك ظهرت منطقة الكيوتيكل مجعدة وكانت الخلايا الطلائية ذات انويه صغيرة. لوحظ أيضا أن التجويف الداخلي لليرقات المتطفل عليها ممتلئ بعبصات هوائية ذات حجم كبير واليرقات المتطفل عليها ماتت بعد أيام قليلة من التطفل.