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Impact of *Nosema sp.* Infection on Nutritional Physiology and Growth of the Tasar Silkworm *Antheraea mylitta*

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Nosema sp. **infection in the Indian tasar silkworm,** ***Antheraea mylitta*** **exerts a complex of influences on its host. The instar duration was extended significantly ($P<0.001$) except in 1st instar. The infected larvae took about 48 days to reach the spinning stage against 40 days in the uninfected ones. The final weight attained by the larva at the end of each instar of development declined significantly following infection, as did weight gain and relative growth rate (RGR). The growth recorded/ day declined in infected larvae compared to uninfected ones from 8.2% during 1st instar to 43.3% during 5th instar. Food ingestion and digestion increased with advancement of the instar significantly irrespective of the status of the larvae but the relative consumption rate (RCR) declined. These parameters significantly declined in infected larvae (except food digested during 2nd instar). The decline was more during 3rd instar. In contrast, the approximate digestibility (AD %) was significantly higher in infected larvae than uninfected ones leaving the 1st instar larvae unaffected. The efficiency of conversion of ingested food (ECI) and efficiency of conversion of digested food (ECD) did not change in a patterned way following the microsporidia (*** *Nosema sp.)* **infection. The values of ECI significantly changed during 2nd, 3rd and 5th instars; while the change in ECD during 2nd, 4th and 5th were significant. During the entire larval life all the parameters tends to decline significantly following microsporidia infection but AD registered a significant increase.** ***Nosema sp.*** **spore concentration has increased 270.7 times during larval development in the course of experimentation.**

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Introduction

The Indian tasar silkworm, *Antheraea mylitta* Drury, is a wild sericigenous insect that produces tasar silk of commercial importance. Among many diseases that affect the silkworms, infection by the microsporidian *Nosema* sp. is the most devastating, since it inflicts severe silkworm crop loss and it is passed on to the next generation transovarially (Jolly and Sen, 1972; Rao *et al.*, 2004). The disease is popularly known as 'pebrine' and the infected silkworm exhibit black spots on the integument and irregular growth. *Nosema* sp. spores infect almost all the host tissues, but the degree of infection varies from tissue to tissue. Because the fat body and gut tissues are very intensely infected (next to testis and ovary) which are involved in uptake and metabolism of food, the utilization of host resources and injury to the cells caused by the microsporidium may have a strong influence on the development of the host (Bauer and Nordin, 1988; Bansal *et al.*, 1997; Henn and Solter, 2000; Jolly and Sen, 1972; Rath *et al.*, 2003). The amount of food ingested and absorbed through the gut may be influenced following invasion of microsporidia into the gut tissues. The relative consumption rate (RCR) and approximate digestibility (AD %) are used to quantify physiological activities in the host. Further disruption of the fat body cells may lead to a decrease in function and may influence the conversion of ingested and digested food into body substance (ECI and ECD) and ultimately growth is inhibited (Henn and Solter, 2000).

Our aim in the present communication was to investigate the impact of *Nosema* infection on nutritional physiology of the larvae through different instars of development in *A. mylitta* that are still unknown.

Materials and Methods

Insect and bioassay

Antheraea mylitta (Lepidoptera: Saturniidae) eggs were collected from inseminated females infected with *Nosema* sp. (1.08×10^7 spores/female) and after incubation (at 28 ± 2°C and 70~80% relative humidity in a pre-sterilized room) the eggs hatch out on the 9th day after oviposition. Newly hatch larvae were examined under microscope at 675X for infection. All the larvae from infected mothers were infected (100%). Uninfected control larvae were obtained from the eggs laid by healthy uninfected females.

Ten replications with 100 larvae during instars 1st and 2nd, 50 during instars 3rd and 4th and 25 during instar 5th each were used for uninfected and infected groups. The uninfected and infected larvae were reared separately in indoor condition (28 ± 2°C, 75 ± 2% R.H., photoperiod 11 h L: 13 h D) in large plastic tubs (60 × 45 × 30 cm), and fed with sufficient quantity of *Terminalia tomentosa* leaves twice a day (Rath *et al.*, 2003; Rath and Sinha, 2005) till the feeding ceased at the end of instar 5th. Larval populations in experimental conditions were maintained by replacing worms from buffer stocks against mortality and larvae used for dry weight determination as detailed before (Rath *et al.*, 2003).

Nosema spore multiplication during embryonic development

Five eggs from the infected female were crushed and examined for *Nosema* spores under microscope at 675X at different stages of embryonic development to gain knowledge on multiplication of spores.

Infection of larval tissues

To identify the level of infection in tissues important to nutritional physiology, the gut and fat body, were dissected out during instars 3rd, 4th and 5th and homogenates of the tissues were prepared in insect saline (7.5 g NaCl, 0.5 g KCl, 0.2 g $CaCl_2 2H_2O$, 0.05 g $NaH_2PO_4 2H_2O$, 0.05 g $NaHCO_3$, 0.2 g $C_6H_{12}O_6$ and water to make 1 litre). The homogenates were examined under a phase contrast microscope to observe the extent of infection.

Food ingestion, digestion and utilization

Food consumed, fecal matter excreted, and larval growth was determined on dry weight basis. Fresh weight of larvae were also noted to asses the live weight during experimentation. Dry weight of larvae, faces and leaves were determined in the same way as mentioned earlier (Rath *et al.*, 2003). Larvae of known weight were killed by placing them in a freezer for short time and then dried in an oven at 60°C to a constant weight. Samples of leaves of equal size and weight were taken each day and dried in an oven at 60°C to determine the dry weight. Left over food and faces were removed each time when fresh food was given and dried in the same way.

Indices of food consumption, digestion, and conversion efficiencies followed Waldbauer (1968): relative growth rate (RGR) = P/TA, relative consumption rate (RCR) = E/TA, approximate digestibility (AD %) = 100 (E – F)/E, efficiency of conversion of ingested food (ECI %) = 100 P/E, efficiency of conversion of digested food (ECD %) = 100 P/ (E – F) (where, A = mean dry weight of larva during the feeding period, E = dry weight of food eaten, F = dry weight of feces produced, P = dry weight gain of larva, T = duration of feeding (days). Amount of food ingested (E) was obtained by subtracting the dry weight of residual food from the dry weight of food given. The difference in weight of food ingested and fecal matter produced (E – F) was treated as food digested. Gain in body weight (P) was the difference in the weight of larvae recorded at the beginning and at the end of the instar under study. Mean weight of the larva (A) was calculated as (initial weight + final weight)/2 in dry weight basis.

Nosema spore concentration

At the end of each larval instar 5 larvae were sacrificed to record the spore concentration by an improved Neubauer hemocytometer (Cantwell, 1974).

Statistical analyses

Differences between means of uninfected and infected groups were tested for significance using student's *t* test. Data within different instar development were checked for significance using one way ANOVA. All the data sets with significant *F* values in ANOVA were analyzed post hoc using Tukey's HSD test. Correlation studies were under taken to established relationship with instar development and nutritional parameters.

Results

Larval examination during instars 4th and 5th reveal shrinkage in gut wall epithelium showing diminishing area of absorptive surface of gut. The microscopic observations of temporary microtome section (5 μ) of gut revealed that the villi in the mid gut epithelium were short in infected larvae compared to healthier ones where it was longer showing enlarged surface area for absorption. Gut muscle and epithelium and haemocytes were found to be heavily infected. Black spots appeared on the integument of the infected larvae from instar 3rd onwards. The fat

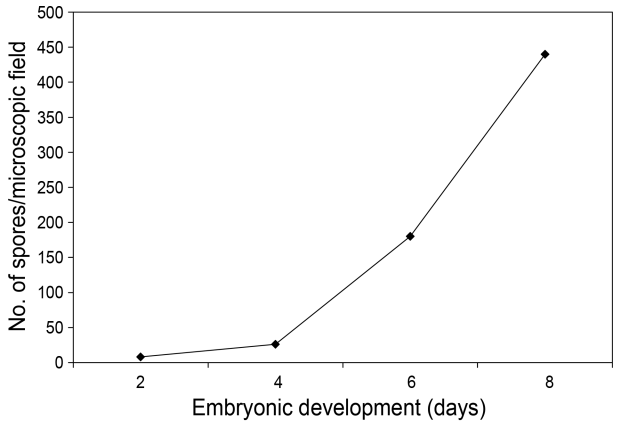

Fig. 1. *Nosema* spore multiplication during embryonic development in *A. mylitta.*

body tissues were damaged to a great extent such that fat cells were completely atrophied and cell boundary could not be seen. Larval survivability was 40~45% in infected lots while it was 70~74% in uninfected lots. The spore concentrations in developing embryos of infected *A. mylitta* experienced a sharp rise in spore production in 8 days of embryonic development from 8 to 440 (Fig. 1).

Developmental time and larval growth

The developmental time during 1st instar was not altered in the infected groups, but was extended significantly ($P < 0.001$) in later instars. The whole larval period was extended by 8 days in infected larvae (Table 1 and 6). The weight of both infected and uninfected *A. mylitta* larva

Table 1. Impact of *Nosema sp.* infection on growth and developmental time in *A. mylitta* during different instars of development

Instar	Developmental time (instar duration, days)			Fresh weight measures (final weight, g)		
	Uninfected	*P* (t test) (% change)	Infected	Uninfected	*P* (t test) (% change)	Infected
1st	4.2 ± 0.2	NS (+ 2.4)	4.3 ± 0.3	0.089 ± 0.002^{a}	< 0.01 (– 4.494)	0.085 ± 0.003^{A}
2nd	3.2 ± 0.2	< 0.001 (+12.5)	3.6 ± 0.2	0.569 ± 0.010^{a}	< 0.001 (– 9.490)	0.515 ± 0.010^{A}
3rd	4.0 ± 0.4	< 0.001 (+25.0)	5.0 ± 0.3	2.484 ± 0.042^{b}	< 0.001 (– 10.064)	2.234 ± 0.040^{B}
4th	7.0 ± 0.4	< 0.001 (+28.6)	9.0 ± 0.3	11.208 ± 0.523^{c}	< 0.001 (– 19.656)	9.005 ± 0.393^{C}
5th	22.0 ± 1.2	< 0.001 (+19.1)	26.2 ± 0.8	36.725 ± 1.091^{d}	< 0.001 (– 26.372)	27.040 ± 1.967^{D}
r				0.8562 (P<0.001)		0.8654 (P<0.001)

Means ± SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, $P < 0.05$). NS- Not significant. r-Correlation coefficient.

Table 2. Effect of *Nosema sp.* infection on weight gain and RGR in *A. mylitta*

Instar	Weight gain (dry mass, g)			Relative growth rate (RGR)		
	Uninfected	*P* (t test) (% change)	Infected	Uninfected	*P* (t test) (% change)	Infected
1st	0.023 ± 0.001^{a}	< 0.05 (– 4.348)	0.022 ± 0.001^{A}	0.412 ± 0.022^{a}	NS (– 4.126)	0.395 ± 0.024^{A}
2nd	0.140 ± 0.003^{a}	< 0.001 (– 10.000)	0.126 ± 0.003^{A}	0.498 ± 0.033^{b}	< 0.001 (– 13.253)	0.432 ± 0.022^{B}
3rd	0.554 ± 0.014^{b}	< 0.001 (– 10.288)	0.497 ± 0.010^{B}	0.338 ± 0.033^{c}	< 0.001 (– 20.710)	0.268 ± 0.020^{C}
4th	2.514 ± 0.139^{c}	< 0.001 (– 22.116)	1.958 ± 0.100^{C}	0.143 ± 0.008^{d}	< 0.001 (– 22.378)	0.111 ± 0.007^{D}
5th	7.414 ± 0.324^{d}	< 0.001 (– 28.338)	5.313 ± 0.526^{D}	0.063 ± 0.004^{e}	< 0.001 (– 23.810)	0.048 ± 0.003^{E}
r	0.8678 (P<0.001)		0.8769 (P<0.001)	-0.9022 (P<0.001)		– 0.9423 (P<0.001)

Means ± SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, $P < 0.05$). NS- Not significant. r-Correlation coefficient.

Table 3. Effect of *Nosema sp.* infection on ingestion and digestion of food in *A. mylitta*

Instar	Ingestion (dry mass, g)			Digestion (dry mass, g)		
	Uninfected	*P* (t test) (% change)	Infected	Uninfected	*P* (t test) (% change)	Infected
1st	0.145 ± 0.004^{a}	< 0.01 (−8.966)	0.132 ± 0.010^{A}	0.108 ± 0.002^{a}	< 0.01 (−8.333)	0.099 ± 0.009^{A}
2nd	0.582 ± 0.017^{a}	< 0.001 (−6.186)	0.546 ± 0.020^{A}	0.396 ± 0.010^{a}	NS (3.030)	0.384 ± 0.020^{A}
3rd	2.137 ± 0.124^{b}	< 0.001 (−20.870)	1.691 ± 0.089^{B}	1.312 ± 0.087^{b}	< 0.001 (−14.939)	1.116 ± 0.071^{B}
4th	8.131 ± 0.353^{c}	< 0.001 (−19.198)	6.570 ± 0.382^{C}	5.678 ± 0.165^{c}	< 0.001 (−14.847)	4.835 ± 0.300^{C}
5th	45.730 ± 1.647^{d}	< 0.001 (−18.412)	37.310 ± 2.082^{D}	18.080 ± 1.026^{d}	< 0.01 (−8.684)	16.510 ± 0.716^{D}
r	0.8006 (P<0.001)		0.7981 (P<0.001)	0.8573 (P<0.001)		0.8491 (P<0.001)

Means ± SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, P < 0.05). NS- Not significant. r-Correlation coefficient.

Table 4. Effect of *Nosema sp.* infection on RCR and AD% in *A. mylitta*

Instar	Relative consumption rate (RCR)			Approximate digestibility (AD%)		
	Uninfected	*P* (t test) (% change)	Infected	Uninfected	*P* (t test) (% change)	Infected
1st	2.583 ± 0.130^{a}	< 0.01 (−9.059)	2.349 ± 0.164^{A}	74.360 ± 2.575^{a}	NS (+ 0.833)	74.980 ± 2.269^{A}
2nd	2.077 ± 0.178^{b}	< 0.01 (−9.774)	1.874 ± 0.105^{B}	68.201 ± 1.733^{b}	< 0.01 (+ 3.107)	70.320 ± 1.540^{B}
3rd	1.307 ± 0.176^{c}	< 0.001 (−30.145)	0.913 ± 0.084^{C}	61.388 ± 1.448^{c}	< 0.001 (+ 7.497)	65.990 ± 3.020^{C}
4th	0.464 ± 0.024^{d}	< 0.001 (−19.612)	0.373 ± 0.020^{D}	69.884 ± 1.887^{b}	< 0.001 (+ 5.317)	73.600 ± 1.887^{A}
5th	0.390 ± 0.029^{e}	< 0.001 (−13.590)	0.337 ± 0.027^{E}	39.572 ± 2.490^{d}	< 0.01 (+ 11.925)	44.291 ± 1.261^{D}
r	−0.9695 (P<0.001)		−0.9586 (P<0.001)	−0.7720 (P<0.001)		−0.7218 (P<0.001)

Means ± SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, P < 0.05). NS- Not significant. r-Correlation coefficient.

increased significantly in the same manner with increasing in instars. The weight of uninfected larva reached its maximum (36.7 g) on 18th day of 5th instar; while in the infected group it was 27 g on 21st day of 5th instar (Fig. 2). The infected larvae always had significantly lower weights compared to their uninfected counterparts and the decline in weight increases with instar from 4.5% in 1st instar to 26.4% in 5th instar. During their entire larval lives uninfected larvae gained about 10.2 g of dry weight while the infected larvae gained 7.5 g (a decline by 26.4%, P<0.001). Thus, during larval life the weight of uninfected mature larvae increases approximately 5246 times, while the infected ones increase by approximately 3863 times. Irrespective of larval condition, RGR declined significantly with the enhancement of the instar (P<0.001) after attaining the peak during 2nd instar. The infected larvae always underwent a lower growth rate than the uninfected ones (except during 1st instar) and the decline in the rate of growth increased with development from 4.1% to 23.8%. When the entire larval life was considered, the gain in weight and RGR declined in the infected group by 26.4% and 18.2% respectively over uninfected ones. The decline in average increase in larval weight increases from 8.2% in 1st instar to 43.3% in 5th instar (Table 2 and 6).

Food consumption, digestion and utilization efficiency

Food ingestion and digestion in *A. mylitta* larvae increased significantly with advancement of the instar, but

Table 5. Effect of *Nosema sp.* infection on ECI and ECD in *A. mylitta*

Instar	Efficiency of conversion of ingested food (ECI)			Efficiency of conversion of digested food (ECD)		
	Uninfected	*P* (t test) (% change)	Infected	Uninfected	*P* (t test) (% change)	Infected
1st	15.960 ± 0.553[a]	NS (+ 5.827)	16.890 ± 1.500[A]	21.465 ± 0.359[a]	NS (+ 5.008)	22.540 ± 2.005[A]
2nd	24.029 ± 1.013[b]	< 0.05 (− 4.033)	23.060 ± 0.891[B]	35.238 ± 1.359[b]	< 0.001 (− 6.919)	32.800 ± 1.151[B]
3rd	26.018 ± 1.666[c]	< 0.001 (+ 13.383)	29.500 ± 2.058[C]	42.400 ± 2.761[c]	NS (+ 5.613)	44.780 ± 3.752[C]
4th	30.905 ± 0.846[d]	NS (− 3.252)	29.900 ± 2.327[C]	44.264 ± 1.991[d]	< 0.01 (− 8.187)	40.640 ± 3.253[D]
5th	16.230 ± 0.875[a]	< 0.01 (− 12.082)	14.269 ± 1.549[D]	41.141 ± 3.207[e]	< 0.001 (− 21.572)	32.266 ± 3.831[B]
r	0.1789 (P, NS)		0.0316 (P, NS)	0.8006 (P <0.001)		0.4722 (P <0.001)

Means ± SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, P < 0.05). NS- Not significant. r-Correlation coefficient.

Table 6. Effect of *Nosema sp.* infection on growth, developmental and nutritional parameters in *Antheraea mylitta* during the entire larval life.

Parameters	Uninfected	*P* (t-test)	Infected	% change
Total larval duration	40.1 ± 1.6	< 0.001	48.1 ± 0.5	+20.0
Fresh weight measures				
Maximum weight (g)	36.7 ± 1.1	< 0.001	27.0 ± 2.0	−26.4
Dry weight measures				
Food ingestion (g)	56.7 ± 1.8	< 0.001	46.2 ± 2.2	−18.5
Food digestion (g)	25.6 ± 1.1	< 0.001	22.9 ± 0.7	−10.3
RCR	0.306 ± 0.017	< 0.01	0.276 ± 0.024	−9.8
AD	45.2 ± 2.1	< 0.001	49.6 ± 1.2	+10.1
ECI	17.9 ± 0.8	< 0.01	16.2 ± 1.3	−9.7
ECD	39.8 ± 2.3	< 0.001	32.8 ± 2.8	−18.0
Gain body weight (g)	10.2 ± 0.3	< 0.001	7.5 ± 0.5	−26.4
RGR	0.055 ± 0.002	< 0.001	0.045 ± 0.001	− 18. 2

Values mean ± SD.

the RCR declined irrespective of the status of the larva (Table 3 and 4). Following *Nosema sp.* infection food consumption and digestion (except during 2nd instar) and RCR declined significantly compared to uninfected ones and the decline was more during 3rd instar (by 20.9%, 14.9% and 30.1% in ingestion, digestion and RCR respectively). The absolute value for ingestion and digestion continuously increased during 5th instar up to 16th day in uninfected larvae and up to 18th day in the infected ones, thereafter it declined up to 18th day in uninfected groups and 23rd day in infected group after which the feeding stops and the body weight continuously declined till spinning starts (Fig. 2).

The overall ingestion and digestion during entire larval stage declined by 18.5%, and 10.3% respectively in infected larvae compared to uninfected ones, while AD increased by 10.1%. The ECI and ECD also declined by 9.7% and 18% respectively followed by a decline in body weight gain (by 26.4%) owing to declines in RCR and RGR by 9.8% and 18.2% respectively (Table 6).

ECI in both infected and uninfected increased significantly with instars up to 4th instar but declined during 5th instar to the level of 1st instar in uninfected larvae and even below in infected ones (Table 5). A significant increase in ECD was observed in uninfected group up to 4th instar and then declined significantly during 5th instar. In contrast, ECD in infected group recorded its high value during 3rd instar and then declined significantly in the sub-

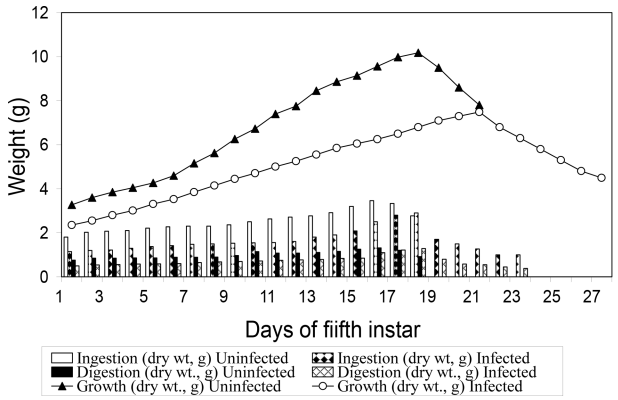

Fig. 2. Dry matter ingestion, digestion and growth in *A. mylitta* larva during 5th instar of development infected with *Nosema* sp.

sequent instars. *Nosema* infection did not have a similar effect in all the instars. The declines in ECI in infected larvae compared to uninfected ones during 2nd and 5th instar were significant, but at the same time it significantly increased during 3rd instar (13.4%). In contrast, the decline in ECD registered in infected larvae during instars 2nd, 4th and 5th were significant.

Spore concentration

Nosema sp. spore concentration was 6000 spores/larva at the end of 1st instar. It was then increased by 13.4, 47.8, 144.2 and 270.7 times during instars 2nd, 3rd, 4th and 5th respectively (Table 7).

Table 7. *Nosema* sp. spore concentration during different instars of development in *A. mylitta*

Instar	*Nosema* sp. spore concentration	Increase over previous instar	Increase over 1st instar
1st	$6.000 \pm 0.477 \times 10^{3a}$		
2nd	$8.042 \pm 0.127 \times 10^{4b}$	13.4 times	13.4 times
3rd	$2.866 \pm 0.358 \times 10^{5c}$	3.564 times	47.8 times
4th	$8.650 \pm 0.518 \times 10^{5d}$	3.018 times	144.2 times
5th	$1.624 \pm 0.308 \times 10^{6e}$	1.877 times	270.7 times

Means SD. Significant difference between instars of development are indicated by different superscripts (ANOVA and Tukey's HSD test, $P < 0.05$).

Discussion

Host-parasite interactions in insects affect growth, developmental time and nutritional physiology (Nakamatsu *et al.,* 2001; Thompson *et al*., 2001, 2005; Rath *et al.,* 2003; Rath and Sinha, 2005). Infection of *A. mylitta* larvae with microsporidia *Nosema* sp. influenced larval developmental time, growth, food ingestion, digestion and conversion of food in to body substance.

The most obvious influence of infection was the extended developmental time of the infected larvae. The developmental time in uninfected larvae was 40 days, which was extended by 8 days in infected larvae. Such extensions in developmental time have been reported in Lepidoptera challenged by microsporidia (Mitchel and Cali, 1994; Henn and Solter, 2000; Rath *et al,* 2003). Extension of larval period in infected larvae seems necessary to reach the intake and growth target toward functional optima (Raubenheimer and Simpson, 1999). Host-parasite competition for available nutrition demands more food to be ingested by the host to acquire optimum growth for pupation and to accumulate sufficient nutrients for the rest of the life. Lepidopteran species infected with parasites other than microsporidia also exhibit extended period of development (Schopf and Steinberger, 1996; Rath *et al.,* 2000; Rath and Sinha, 2005). We infer from our data that extended developmental time is coupled with lower RCR might be due to longer retention of food in the gut to maximize AD to meet the increased demand of nutrients in the infected host (Reynolds *et al.,* 1985). The AD in infected *A. mylitta* larvae significantly increased over the uninfected ones through out the development (except during 1st instar), in agreement with this observation.

In *A. mylitta*, the amount of food ingested, digested and gain in body weight declined significantly in all the instars following *Nosema* infection (except food digestion in 2nd instar). The same affect also prevails when the entire larval period was accounted for. Similar findings were reported in insects following parasitization by *Nosema* and other parasites including parasitoids (Hoch and Schopf, 2001; Rath *et al,* 2003; Rath and Sinha, 2005). Metabolic profile of the haemolymph and fat body declined in insects during host-parasite interaction resulted in inhibition of growth (Lue *et al.,* 2001; Nakamatsu *et al*., 2001; Thomson *et al*, 2001, 2002).

We observed that a considerable part of the fat body cells of the host *A. mylitta* were damaged following heavy infection with *Nosema* spores. Such damage might lead to failure in fat body metabolic events. Findings in *Bombyx mori* infected with *Nosema bombycis*, *Lymantria dispar* infected with *Vairimorpha sp*., and *A. mylitta* with *Nosema sp.* support our interpretations (Jolly *et al.,* 1972; Henn and Solter, 2000; Rao *et al.,* 2004). *Menduca sexta* exhibited altered food selection following parasitization to maintain haemolymph trehalose through gluconeogenesis but failed to attain proper growth (Thomson *et al.,* 2001).

The physiological disturbance caused by the parasite in action might have consumed the essential nutrients required for growth affecting the food utilization by the host. Exhaustion of protein to maintain trehalose level instead of being utilized for host growth may be another possible reason for retard host growth (Thompson and Redak, 2000; Thompson *et al.,* 2003, 2005).

In the present study we observed that, although mid-gut infection is mild some portions show heavy infection, the fat body is damaged to a great extent in the infected larvae of *A. mylitta* as recorded in an earlier study (Jolly and Sen, 1972). *Bombyx mori* infected with *Nosema bombycis* and new microsporidian isolates show high infection in mid-gut. Formation of hypertrophied sacs in the midgut filled with spores has been reported in *B. mori* infected with a new microsporidian isolate (NIK-4m) (Rao *et al.,* 2004). Since mid-gut and fat body are chief sites of metabolism, the invasion of *Nosema* sp. reduces food ingestion and digestion but AD increases in all instars (except during 1st instar) in infected *A. mylitta,* apparently to compensate the nutrient requirement. Similar observations were also reported in gypsy moth (Henn and Solter, 2000) and eastern spruce bud worm (Bauer and Nordin, 1988) and *A. mylitta* (Rath *et al.,* 2003).

Both ECI and ECD behave almost in the same manner during infection. Over all, when the entire larval period was considered both the parameters declined significantly. Lower values of ECI and ECD were observed following microsporidia infection in spruce bud worm (Bauer and Nordin, 1988) and in gypsy moth (Henna and Solter, 2000) supporting our findings. This likely resulted in dysfunction of fat tissue, thus affecting conversion potential. This may also affect nutritional absorption critical for growth (Giordana *et al.,* 2002; Leonardi *et al.,* 2001), which aligns with findings of our present observation of shrinkage in absorptive area of the gut in the infected larvae of *A. mylitta.*

During parasitization, the nutrients available to the host are directed towards protein and carbohydrate synthesis, which lead to reduced growth and longer developmental period Thomson *et al.* 2001; Lee *et al.,* 2002). Nutrition interacts with parasite to influence growth and physiology of the insect (Thompson *et al.,* 2005).

It is plausible that the development of *Nosema* sp. spores strongly impedes larval nutritional physiology in *A. mylitta* seen elsewhere (Henn and Solter, 2000). Parasitism brings about long-term physiological effects that may influence the ultimate success of parasite development and multiplication. Depressed host growth and increased developmental time are common responses. Thus, it can be inferred the microsporidial infection in the host impedes its growth, metabolism and restricts its efficiency.

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