

## Gypsy moth revisited: Studies on the natural enemy complex of *Lymantria dispar* L. (Lep., Lymantriidae) during an outbreak in a well known gypsy moth area

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**Abstract:** Untersuchungen zum Gegenspielerkomplex von *Lymantria dispar* während einer Massenvermehrung auf einer bekannten Gradationsfläche.

Seit 1992 führen wir in einem Eichenmischwald bei Klingenbach, nahe Eisenstadt, Österreich, Abundanzhebungen des Schwammspinners, *Lymantria dispar* (Lep., Lymantriidae) mittels Eigelegezählungen durch. Im Jahre 2002 zeichnete sich nach sieben Jahren der Latenz ein Anstieg der Populationsdichte ab. Die Zahl von 1,2 Gelegen/Baum im Winter 2002/03 deutete auf eine beginnende Massenvermehrung. Die Dichte an Eigelegen war im folgenden Winterhalbjahr mit 9,7 pro Baum extrem hoch. Durch stadienspezifische Aufsammlungen von *L. dispar* Raupen oder Puppen und deren Zucht im Labor ermittelten wir die durch Parasitoide verursachte Mortalität sowohl im Progradationsjahr 2003 als auch im Jahr der Kulmination 2004. Generell war die Mortalität der Raupen und Puppen sehr gering. Im Jahr der Progradation vermochte einzig *Parasetigena silvestris* (Dipt., Tachinidae) nennenswerte Mortalität von 23,7% bei Altraupen zu verursachen. Die sehr warme, trockene Witterung im Mai-Juni 2003 bedingte eine ausgesprochen schnelle Raupenentwicklung. Im Frühjahr 2004 zeigten die Raupenaufsammlungen noch geringere Parasitierungsraten. Es dominierten *P. silvestris* und *Blepharipa* sp. (Dipt., Tachinidae) mit 8,5% bzw. 8,0% bei den Altraupen. Aufsammlungen von Puppen im Jahr 2004 zeigten anhand der typischen Fraßbilder eine Mortalität durch *Calosoma* spp. (Col., Carabidae) von 13% an den Zweigen des Baumbestandes bis 38% in der Strauchschicht. Die Ergebnisse unserer Untersuchung zum Antagonistenkomplex in der Phase der Progradation und Kulmination werden im Vergleich mit entsprechenden Daten aus der letzten Gradation 1993 diskutiert.

**Key words:** *Lymantria dispar*, parasitoid, predator, *Parasetigena silvestris*, *Calosoma sycophanta*

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We have been surveying a gypsy moth, *Lymantria dispar* (Lep., Lymantriidae), population in the oak forest of Klingenbach near Eisenstadt, Austria, since 1992. During the last gradation from 1993 to 1996, we studied the natural enemy complex at this site in comparison with other locations where no outbreak occurred (HOCH et al. 2001). During the latency years, an experimental study on the impact of predators on *L. dispar* pupal populations was performed (GSCHWANTNER et al. 2002). The population density was recorded regularly; in the winter 2001/02, the egg mass surveys indicated a rising population after seven years of latency. We used this opportunity to study the parasitoid complex in the progradation phase. This phase of gypsy moth population dynamics was not studied in our previous work. Moreover, it allowed us to repeat the investigation during the outbreak after 11 years.

### Material and Methods

Gypsy moth population densities were evaluated by egg mass counts in the winter season. We performed these counts following Turcek's method as described in HOCH et al. (2001). Therefore, starting from the center of the study plot, 50 trees in each cardinal direction were searched for egg masses up to a stem height of 4 m. Parasitism was studied from larval and pupal samples that were collected stage specifically on the study plot. Collections were carried out when the majority of the gypsy moth population was in the requested

stage. Observation of the prevalence of the larval stadia was also used to reconstruct larval development. The sampled larvae were reared individually on wheat germ diet (BELL et al. 1981) at 20°C, 16:8 L:D photo-period. Parasitism was based on emergence of parasitoids from the test larvae; percent parasitism is expressed as number of larvae out of which parasitoids emerged divided by the total number of larvae in the sample of the respective instar. Braconid parasitoids were determined according to cocoon and adult wasp; tachinids were determined based on puparia morphology following the key for gypsy moth parasitoids provided by ZUBRIK (1998).

The impact of predators was evaluated based on a collection of samples of pupae after the majority of adult moths had eclosed. Samples were collected separately from the ground vegetation and twigs of trees; hardly any pupae could be found on the trunks of the trees. Pupal remains were examined and the cause of mortality was determined based on feeding marks as outlined in GSCHWANTNER et al. (2002).

**Results and Discussion**

Ten years after the last culmination, egg mass density in winter 2002/03 indicated an ongoing rapid increase of the population. A density of almost 10 egg masses per tree in the following winter led to the prognosis of total defoliation on the site in spring 2004 (Figure 1).

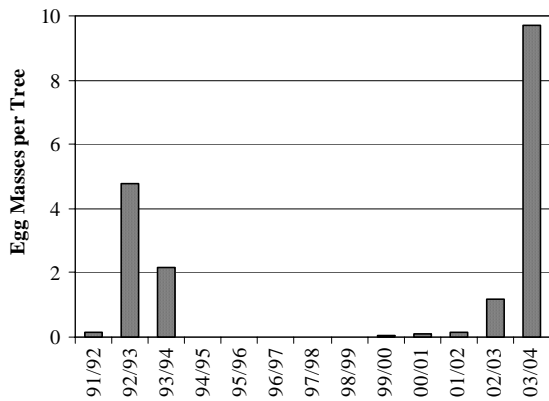


Fig. 1: Average number of *L. dispar* egg masses per tree on the study plot Klingenberg.

Larval development was obviously accelerated by the unusually warm and dry spring in 2003. The timing was comparable to the year of the last outbreak in 1993 (SCHOPF & HOCH 1997). Spring 2004 was markedly cooler and consequently the larval development was retarded (Figure 2). Due to the slow and staggered development the fastest insects started pupating before trees were totally defoliated.

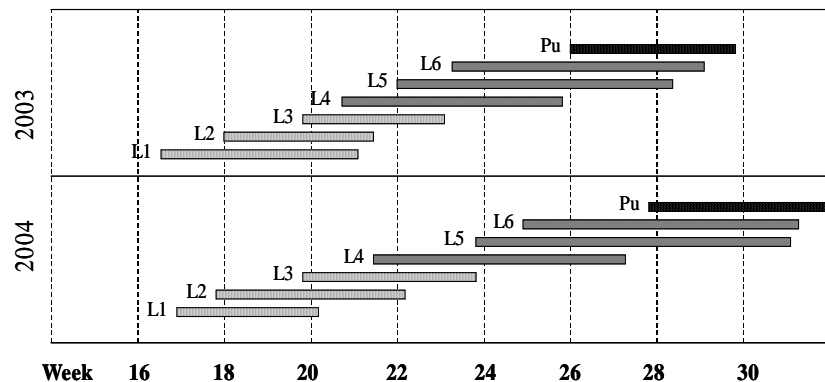


Fig. 2: Larval development on the study site in 2003 and 2004. Average temperatures were 18.1°C in May and 22.6°C in June 2003, precipitation was 68 and 43 mm, respectively. In 2004, average temperatures were 14.2°C in May and 18.3°C in June, precipitation was 86 and 109 mm, respectively (ZAMG 2005).

In the progradation year, parasitism of *L. dispar* was generally low (Table 1). Only a few specimens of the braconids *Glyptapanteles liparidis* and *G. porthetriae* were recovered. The only significant mortality was caused by the tachinid fly *Parasetigena silvestris* in older larvae. The samples from the outbreak years revealed even a lower percent parasitism. *P. silvestris* parasitism in old larvae (L5+6) was significantly lower than in 2003 ( $\text{Chi}^2=17.0$ ,  $P<0.001$ ) and, although *Blepharipa* sp. caused higher parasitism than the year before ( $\text{Chi}^2=8.9$ ,  $P<0.01$ ), total parasitism by tachinids was also significantly lower ( $\text{Chi}^2=5.09$ ,  $P<0.05$ ). Pupal parasitism did not differ between the years ( $\text{Chi}^2=1.72$ ).

Table 1: Percentage of *Lymantria dispar* collected stage specifically on the study plot with emerged parasitoids, killed by pathogens and unknown causes, and successful development into adults (L1 to L6 = instars 1 to 6; Pu = pupae).

Stadium	2003						2004					
	L1	L2	L3	L4	L5+6	Pu	L1	L2	L3	L4	L5+6	Pu
Collection date	25.-28.4.	2.-6.5.	6.-10.5.	16.5.	24.5.-4.6.	15.6.	2.5.	2.-8.5.	8.-14.5.	26.5.	7.-16.6.	30.6.
Surviving	90.8	84.8	91.9	81.8	60.8	78.6	73.5	74.7	86.9	85.9	61.5	25.0
<i>Glyptapanteles liparidis</i>	-	3.0	3.0	2.0	-	-	-	-	3.0	-	-	-
<i>Glyptapanteles porthetriae</i>	1.0	4.0	1.0	1.0	-	-	1.0	-	-	-	-	-
<i>Parasetigena silvestris</i>	-	-	1.0	10.1	23.7	3.1	-	-	1.0	9.1	8.5	-
<i>Blepharipa</i> sp.	-	-	-	-	1.5	8.2	-	-	-	-	8.0	6.0
<i>Zenillia libatrix</i>	-	-	-	-	0.5	-	-	-	-	-	-	-
<i>Brachymeria intermedia</i>	-	-	-	-	-	1.0	-	-	-	-	-	1.0
Pathogens / unknown	8.2	8.1	3.0	5.1	13.4	9.2	25.5	25.3	9.1	5.1	22.0	68.0
n	98	99	99	99	194	98	98	99	99	99	200	100

The tachinids *P. silvestris* and *Blepharipa* sp. are considered to be specialist species and typically cause highest parasitism in high density host populations (FUESTER et al. 1983; MAIER & BOGENSCHÜTZ 1990). Our previous study showed, however, that peak parasitism occurs in the early post-culmination phase (HOCH et al., 2001), which we may also expect for this gradation cycle. Parasitism by *P. silvestris* (8.5% in old larvae) was even lower this time than during the last outbreak in 1993 (20.4%;  $\text{Chi}^2=10.30$ ,  $P<0.01$ ) rather resembling parasitism on an outbreak site in Slovakia in 1993 where gypsy moth occurred at extremely high abundance with > 25 egg masses per tree (HOCH et al. 2001).

In the outbreak year 2004, high mortality in our sampled insects was due to undetermined causes. One part, collected as L1 or L2, died shortly after collection. The other part, collected as old larvae or pupae, died as fully developed imagines that were unable to eclose from the pupa. This may be due to unfavorable abiotic conditions. However, also food quality can be the cause of problems during metamorphosis and adult eclosion (KLAUS 1999; SCHOPF et al. 1999). An already deteriorated leaf quality for the last larval instar could, therefore, also play a role.

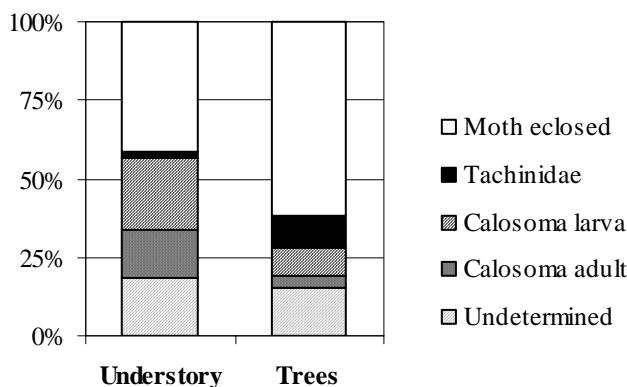


Fig. 3: Pupal mortality based on feeding marks of predators. *Calosoma* spp. caused significantly higher mortality in pupae on the understory vegetation than on twigs of trees ( $\text{Chi}^2= 24.8$ ,  $P<0.001$ ).

*Calosoma* sp. destroyed many *L. dispar* pupae (Figure 3); particular *C. sycophanta* was abundant on the study plot, both, in adult and larval stage. The former was also frequently seen preying on *L. dispar* larvae. This supports the opinion that this species can cause substantial mortality among outbreak populations (BURGESS & CROSSMAN 1929; WESELOH 1985). Besides *Calosoma*, the silphid beetle *Xylodrepa quadripunctata* was abundant on the study plot and was observed preying on younger *L. dispar* larvae. In 1998, under latency conditions, *Calosoma* killed only 1.4% of an experimental pupal population on this site and not a single specimen was captured (GSCHWANTNER et al. 2002). The impact of rodents, such as *Apodemus* spp. or *Clethrionomys glareolus*, that were the most important predators of pupae during the latency period (GSCHWANTNER et al., 2002) was not evaluated in the present study.

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