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Hypersensitive reaction of *Solanum dulcamara* to the gall mite *Aceria cladophthirus* causes an increased susceptibility to *Tetranychus urticae*

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ABSTRACT

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The hypersensitive reaction induced by the eriophyid mite *Aceria cladophthirus* (Nalepa) on detached leaves of *Solanum dulcamara* L. did not protect them against subsequent attacks by the spider mite *Tetranychus urticae* Koch. This reaction stimulated the oviposition of *T. urticae*; the increase of fecundity was about 40%. As the survival rate and the life-cycle were not affected, higher populations of *T. urticae* developed on leaves previously infested by *A. cladophthirus* than on healthy ones. The hypersensitive reaction caused by members of one family of phytophagous mites induced an increased susceptibility to attacks by mites of an unrelated family.

INTRODUCTION

The hypersensitive reaction in plants is generally held to be one of the most effective defence mechanisms by which invaders are rapidly localized in small necrotic lesions on resistant hosts (Kiraly et al., 1972). This reaction is non-specific in that quite similar local lesions may develop after infection by many pathogens (Klement and Goodman, 1967; Wood, 1982), after infestation by nematodes (Kaplan and Keen, 1980), insects (Shapiro and De Vay, 1987) or eriophyoid mites (Westphal et al., 1981) and after abiotic stresses (van Loon, 1977).

The hypersensitive reaction triggers in plants an induced resistance which protects them locally and/or systemically against challenge inoculation of the same or other pathogens (Deverall, 1977; Matta, 1980; Sequeira, 1983), and even against further attack by insects (McIntyre et al., 1981) or eriophyoid

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mites (Westphal et al., 1991). The protective effect reduces plant damage and prevents the establishment of invading organisms or limits their subsequent activities (Ross, 1961a,b; Novaky et al., 1973; Niblett et al., 1978; Kuc, 1983).

The objective of this study was to determine if the hypersensitive reaction occurring in *Solanum dulcamara* L. plants after attack by the eriophyid gall mite *Aceria cladophthirus* (Nalepa) triggers a local induced resistance that protects them against challenger spider mites. Therefore, we have assayed the effects of necrotic local lesions occurring on *S. dulcamara* detached leaves, on the development of *Tetranychus urticae* Koch. Variations in damage rate on *S. dulcamara* leaves during challenge infestation by the spider mite were not investigated in this study.

MATERIALS AND METHODS

Plants, mites and culture of detached leaves

Susceptible and resistant *S. dulcamara* plants were grown at $20 \pm 2°C$, under long-day conditions (16L/8D) and 8 W m^{-2} illumination. *Aceria cladophthirus* multiplies for extended periods within the galls induced on susceptible plants. The spider mite *T. urticae* developed just as well on resistant and susceptible host plant varieties. Nevertheless, to avoid possible interference through an eventual effect of feeding on the source host plant as reported in *T. urticae* by De Ponti (1977), the spider mites utilized in this study were reared only on the resistant variety. Infested plants were maintained under the same conditions as healthy ones.

Detached leaves of resistant plants were cultivated in vitro under non-aseptic conditions in petri dishes containing Murashige and Skoog medium (1962) solidified by 0.7% Difco Bacto-Agar and without sucrose, to prevent bacterial and fungal contamination. The dishes remained slightly open to avoid water condensation on the leaves. This arrangement entailed some drying of the medium. In order to maintain the hygrometric conditions fairly constant, the leaves were transplanted in new petri dishes every two or three days. The dishes were placed into a growth chamber maintained at $22 \pm 2°C$, under long-day conditions (16L/8D), 50–60% relative humidity and 8 W m^{-2} illumination. Under these conditions, the detached leaves developed well for several weeks.

Leaf position fn-1 was that of the last emerging leaf, about 2 mm long; the next leaves on the stem were numbered basipetally (Fig. 1). Only the actively growing leaves fn-5 to fn-7 were used in the experiments.

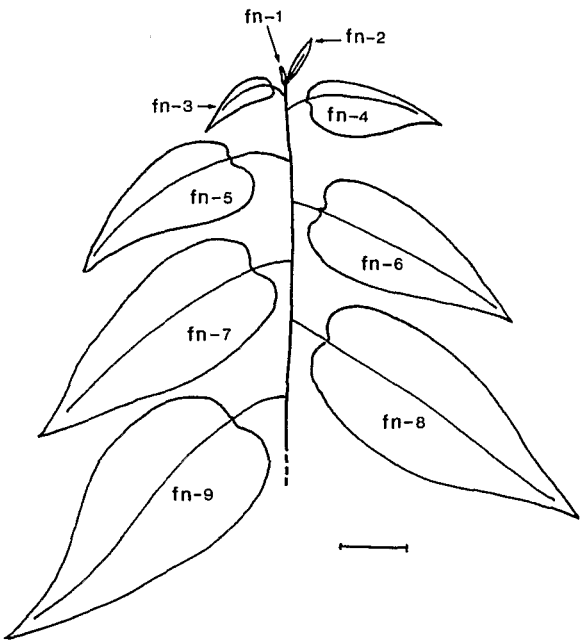

Fig. 1. Schematic representation of leaf size and leaf position on the stem of a vegetative shoot of *S. dulcamara*. The bar represents 1 cm.

Infestation of detached leaves

Primary infestation by A. cladophthirus

The females of *A. cladophthirus* were taken from gall fragments under a dissecting microscope using a single eyelash mounted on a glass rod and transferred to healthy leaves in the petri dishes. As the feeding activity of one female induced 2–3 local lesions a day (Westphal et al., 1990), infestation was carried out to obtain different local lesion densities: low, moderate and high with about 10, 50–100, and 400–1000 local lesions per leaf, respectively. The mites were allowed to feed for only 24 h to avoid the induction of new lesions during the experiments, and were then manually removed from each leaf side. Mite removal does not impede full development of the necrotic local lesions, which reach their final size (350 μm in diameter) after 3 or 4 days. This technique allowed us to obtain leaves with well-dated local lesions.

Infestation with T. urticae

Healthy detached leaves were exposed to one to six 1-day-old females of *T. urticae* for a period of 8 to 16 days. Corresponding leaves with necrotic local lesions were challenged, at various times after the primary infestation (1 to 11 days), by infesting them in the same way with females of the same age.

Simultaneously, the transfer of 1 to 3 males to each leaf allowed for the fertilization of the females.

Daily examination of the test leaves allowed us to detect the effects of the hypersensitive reaction on the subsequent development of *T. urticae*. Survival rates were estimated from 18 to 60 females 3, 4 and 6 days after their transfer. To determine the larval survival in relatively homogeneous populations, all females and non-hatched eggs were manually removed after 6 days; dead larvae were counted 4 and 6 days later.

Fecundity, expressed as the mean number of eggs laid per female per day, was measured over an 8-day period. Data obtained from about 70 females were analyzed by using Student's *t*-test ($P<0.05$).

Hourly observations at critical periods permitted us to determine the time of the first egg hatching, the occurrence of males and first eggs laid by the females of the succeeding generation. Effects of the primary infestation by *A. cladophthirus* on duration of the development of each larval stage were not analyzed.

RESULTS

Behavior of T. urticae

After their transfer to healthy leaves, the females of *T. urticae* did some short exploratory probings on the upper leaf surface before settling down to feed on the undersize. The behavior of females on leaves previously infested by *A. cladophthirus* did not deviate from this pattern. During the following hours, discolored leaf spots developed at each feeding site in both experiments. The presence of necrotic local lesions did not prevent the females from feeding; they even fed on the tissues surrounding the necrotic lesions which then underwent typical discoloration. Egg deposition occurred as early as the first hour after the transfer of the females on intact leaves or on leaves with local lesions; in the latter case, the eggs were deposited without discrimination on healthy tissues or in the small depressions formed by the local lesions. After hatching, the larvae behaved on leaves with local lesions as on healthy ones, and sometimes fed on the tissues adjacent to necrotic lesions like the females did.

Survival rate of T. urticae

During the first two days after mite transfer, the female survival rate was nearly 100% both on intact leaves and on leaves with necrotic local lesions. In the following days, this rate decreased for both kinds of leaves but without significant differences: i.e. about 70% after 6 days (Table 1). Loss of females was mostly caused by entrapment on the medium.

TABLE 1

Comparative survival rates, after 3, 4 and 6 days, of females of *T. urticae* transferred onto healthy leaves or leaves with 1-, 3- or 11-day-old necrotic local lesions (CI = 95% confidence interval)

	Number of females	Days		
		3	4	6
Healthy leaves	60	98% CI: 91–100	95% CI: 86–99	80% CI: 67–90
Leaves with local lesions				
Moderate infestation				
1-day-old lesions	60	97% CI: 88–100	87% CI: 75–95	73% CI: 60–84
11-day-old lesions	18	94% CI: 72–100	89% CI: 65–99	72% CI: 46–91
Strong infestation				
3-day-old lesions	34	97% CI: 84–100	85% CI: 68–96	68% CI: 49–83

TABLE 2

Comparison of survival rates of *T. urticae* larvae, 10 and 12 days after the transfer of females to healthy leaves or to leaves bearing 11-day-old necrotic local lesions (CI: 95% confidence interval; *N*: number of larvae)

		fn-5		fn-6	
		Days		Days	
		10	12	10	12
Healthy leaves	%	91	84	88	81
	N	118	118	186	186
	CI	84–96	75–90	82–93	74–87
Leaves with local lesions	%	89	81	86	84
	N	170	170	270	270
	CI	83–94	74–87	80–90	78–89

Less than 0.3% of the eggs were found desiccated after 5 days on both control and previously infested leaves. Egg viability was not affected by the presence of local lesions since even direct contact of eggs with necrosed tissues did not prevent hatching.

The survival rate of larvae on leaves with local lesions was not significantly different from that on healthy ones: i.e. 88% ten days after the transfer of the females and 82% two days later (Table 2).

The age of local lesions had no significant effect on mite survival (Table 1). High lesion densities did not increase the mortality of the females (Table

1), but the resulting intensive leaf senescence did not allow further investigation into survival of larval populations.

Fecundity of T. urticae

The mean number of eggs laid per female showed daily fluctuations both on healthy leaves and on leaves with local lesions (Fig. 2). On healthy leaves, the mean oviposition rate was 7.0 ± 2.3 ($N=70$) eggs per female per day over the 8-day period of our experiments. On leaves previously infested by *A. cladophthirus*, spider mites laid significantly more eggs during the same period regardless of the local lesion density: 8.3 ± 2.3 ($N=19$), 9.9 ± 3.4 ($N=40$) and 10.1 ± 3.2 ($N=21$) eggs per female per day on leaves with local lesions resulting from high, moderate and low infestations respectively. On leaves with high lesion density, fecundity was strongly stimulated the first day, remained at a high level the next days, and went back to normal values after 7 to 8 days. In this case, the overall oviposition stimulation was only about 18% whereas it was much higher (40%) on leaves with moderate or low lesion densities.

Improved oviposition was already detectable after 1 day, and occurred regardless of the age of the lesions (1-day-old lesions: 9.5 ± 3.5 eggs/female/day ($N=37$); 11-day-old lesions: 9.2 ± 3.8 eggs/female/day) ($N=18$). Moreover, leaf age had no significant effect on *T. urticae* fecundity (Table 3).

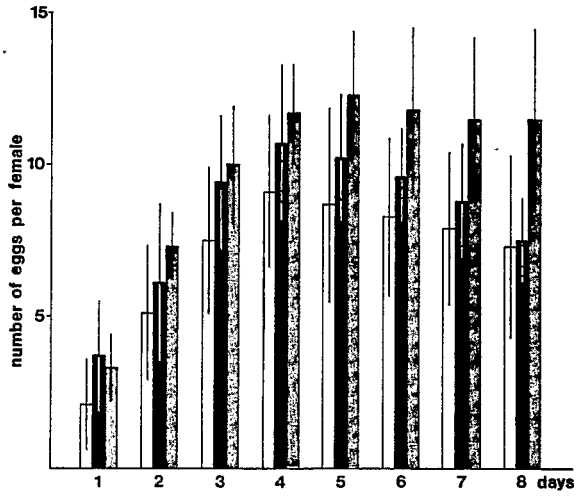

Fig. 2. Comparison of mean daily oviposition rates per female of *T. urticae* on healthy leaves (open bars, $N=70$) and on leaves previously infested by *A. cladophthirus*: leaves with high (dark bars, $N=19$), moderate and low (grey bars, $N=61$) densities of necrotic local lesions. Vertical lines delimit the 95% confidence intervals on the means.

TABLE 3

Comparison of means of daily fecundity of *T. urticae* during 8 days on healthy leaves and on corresponding leaves with 2-day-old necrotic local lesions (N=number of females; n=number of leaves)

	Leaf position		
	fn-5	fn-6	fn-7
Healthy leaves	7.1 ± 2.6 ($N=40$; $n=10$)	7.2 ± 2.6 ($N=43$; $n=11$)	6.5 ± 1.4 ($N=28$; $n=6$)
Leaves with local lesions	10.1 ± 2.4 ($N=11$; $n=4$)	10.8 ± 2.8 ($N=14$; $n=5$)	10.2 ± 2.6 ($N=11$; $n=4$)

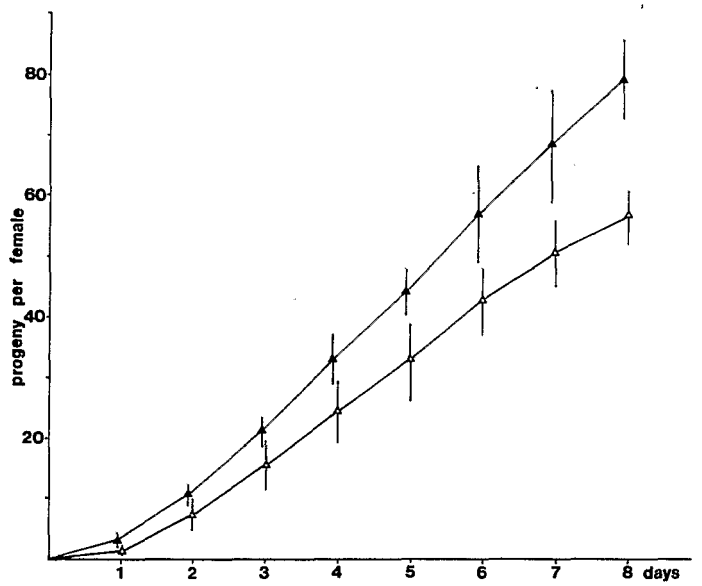

Fig. 3. Cumulative curves showing the comparative development of *T. urticae* populations on healthy leaves (△) and on leaves with low and moderate local lesions (▲). Vertical lines delimit standard error of means.

Life cycle of T. urticae

The presence of local lesions did not significantly affect the egg incubation period. The first larvae appeared at 97.5 ± 3.8 h ($N=14$) on healthy leaves and at 95.0 ± 2.6 h ($N=21$) on leaves with local lesions. No statistically significant differences were found between development times of males on healthy leaves (9.9 ± 0.4 days; $N=10$) and those on leaves with local lesions (9.4 ± 0.7 days; $N=10$). Similarly, development of females was not affected; the first eggs laid by the new females were detected at 10.9 ± 0.8 days ($N=8$) (on healthy leaves and in 10.7 ± 0.4 days ($N=8$) on leaves with local lesions. Thus, the hypersensitive reaction triggered by *A. cladophthirus* did not signif-

icantly modify the duration of *T. urticae* life cycle. The rough average of 40% increase of *T. urticae* populations occurring on leaves with necrotic local lesions (Fig. 3) was due mainly to increased fecundity.

DISCUSSION

The data presented here clearly indicate that the hypersensitive reaction triggered by *A. cladophthirus* in *S. dulcamara* leaves did not provoke the expected local induced resistance against challenge infestation with *T. urticae*. This spider mite did not discriminate between healthy and previously infested leaves since its behaviour was similar in both cases. The hypersensitive reaction had no adverse effects either on spider mite survival or on life cycle. On the contrary, the presence of local lesions increased the susceptibility of *S. dulcamara* leaves to *T. urticae*, an effect which was expressed by a strong stimulation of fecundity. This positive effect on spider mite oviposition occurred within 24 h and led to higher populations than on healthy leaves.

The use of detached leaves for the experiments cannot be responsible for the increasing of spider mite populations on leaves previously infested by *A. cladophthirus*. Indeed, the duration of the life-cycle and the fecundity rate of *T. urticae* reared on healthy detached *S. dulcamara* leaves were similar to those obtained by Carey and Bradley (1982) on healthy cotton seedlings at the same temperature ($22 \pm 2°C$). This shows that the technique of infestation of detached leaves is a reliable and efficient method for analyzing plant–spider mite interactions, but only for limited periods since, after 1 or 2 weeks, leaves infested by *T. urticae* became too badly damaged.

Improved performance of *T. urticae* on leaves with local lesions strongly contrasts with the negative effects of the same *S. dulcamara* hypersensitive reaction on subsequent eriophyid mite populations (Westphal et al., 1991). In that earlier report, the protective effect was manifested by an increased mortality either of the inducing mite, *A. cladophthirus*, or of another eriophyid, the rust mite *Thamnacus solani* Boczek and Michalska. It seems therefore that the local induced resistance triggered in *S. dulcamara* leaves by *A. cladophthirus* was only efficient against further invasions by these host-specific eriophyid mites but not against the polyphagous *T. urticae*, which belongs to the unrelated family Tetranychidae. Further investigations are needed to determine whether the hypersensitive reaction of *S. dulcamara* has negative effects on subsequent attacks by other unrelated invading organisms (aphids, fungi, bacteria or viruses).

The results reported here also conflict with those establishing negative effects of induced resistance-like phenomena triggered in plants by previous attacks by eriophyid mites (Croft and Hoying, 1977) or spider mites (Karban and Carey, 1984; Harrisson and Karban, 1986; Karban, 1986; English-Loeb and Karban, 1988; Karban and English-Loeb, 1988) on subsequent mite

development. Nevertheless, it should be stressed that no hypersensitive reaction was implicated in those plant–mite interactions.

Hypersensitive reaction triggers drastic change in plant metabolism regardless of the nature of the inducing agent and leads to the accumulation of compounds considered to be defensive substances such as phenolic compounds (Bell, 1981), phytoalexins (Bailey, 1982; Kuc, 1987) and pathogenesis-related proteins (van Loon, 1983). Phenolics may have adverse effects on insects (Fenny, 1968; Rhoades, 1985) or spider mites (Larson and Berry, 1984) and phytoalexins may act as feeding deterrents. (Sutherland et al., 1980; Hart et al., 1983; Kogan and Paxton, 1983). Some pathogenesis-related proteins have chitinase and glucanase activities (Kaufmann et al., 1987; Legrand et al., 1987) and may be defense enzymes against invaders. Here is the paradox: all of these substances have been shown to occur in *S. dulcamara* leaves undergoing hypersensitive reaction to *A. cladophthirus* (Bronner and Westphal, 1987; Westphal et al., 1989; Bronner et al., 1991) but they are completely ineffective in stopping or hindering spider mite development. These results suggest either that defensive substances were not toxic for *T. urticae* or that detoxification mechanisms may develop rapidly in spider mites as is the case in insects (Rhoades, 1985; Bell, 1987; Harvell, 1990). However, this does not explain why spider mite performance was improved. Increased spider mite fecundity and higher populations could result from enhancement of nutritional value of leaves with local lesions. Indeed, positive correlations between food plants of better quality and increased fecundity have been established for spider mites (Rodriguez and Rodriguez, 1987) and insects (Myers and Post, 1981). This does not exclude the possibility that some substances induced by the hypersensitive reaction in *S. dulcamara* leaves may be used by *T. urticae* as kairomones, stimulating either feeding activity or fecundity. Nevertheless, it is far from clear how *T. urticae* circumvents the defense reaction in leaves which earlier had reacted in a hypersensitive manner to *A. cladophthirus*.

Infestation by unimportant herbivorous arthropods in order to protect plants against more damaging pests has been suggested by some acarologists (English-Loeb and Karban, 1988; Karban and English-Loeb, 1990). However, the same authors have recently proven that previous spider mite feeding injury can also induce an unexpected increased systemic susceptibility in beans to subsequent infestation by *T. urticae* (English-Loeb and Karban, 1991). These results are consistent with ours, although the increased susceptibility of *S. dulcamara* leaves to *T. urticae* was shown to occur locally and no hypersensitive reaction had occurred in beans. Our results also demonstrate that the problem is more complex than previously supposed, and that possible practical applications of induced resistance for pest control should be carefully reexamined to determine their actual limits.

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