Table 1. *Mean number of different stages of* Globodera rostochiensis *found in 5 g potato root tissue at two sampling dates.*

Sampling date	Developmental stage	Aldicarb dose (g/m^2)			
		before 1991	in 1991		
			0.6	0.3	0
30.05.1991	Second stage juveniles	0.6	7 *b*	0 *b*	727 *a*
		0.3	7 *b*	20 *b*	520 *a*
		0	0 *b*	6 *b*	2313 *a*
	Swollen juvenile stages	0.6	13 *b*	33 *b*	2240 *a*
		0.3	7 *b*	0 *b*	1420 *a*
		0	0 *b*	0 *b*	3793 *a*
	Females	0.6	0 *c*	0 *c*	160 *b*
		0.3	0 *c*	0 *c*	293 *a*
		0	0 *c*	0 *c*	467*a*
	Males	0.6	0 *b*	0 *b*	20 *b*
		0.3	0 *b*	0 *b*	13 *b*
		0	0 *b*	0 *b*	53 *a*
16.07.1991	Second stage juveniles	0.6	7 *a*	8 *a*	13 *a*
		0.3	20 *a*	47*a*	53 *a*
		0	33 *a*	72*a*	40 *a*
	Swollen juvenile stages	0.6	0 *c*	0 *c*	33 *ab*
		0.3	13 *bc*	13 *bc*	33 *ab*
		0	0 *c*	33 *ab*	140 *a*
	Females	0.6	0 *d*	0 *d*	106*ab*
		0.3	0 *d*	20 *cd*	186 *a*
		0	0 *d*	60 *bc*	353 *a*
	Males	0.6	7 *b*	0 *b*	73 *a*
		0.3	27 *ab*	53 *ab*	27 *ab*
		0	20 *b*	33 *ab*	147 *a*

Per sampling date and stage, numbers followed by the same letter are not significantly different according to the Duncan test ($P \leq 0.05$).

INDIFFERENT RESPONSE TO NEMATICIDE APPLICATIONS

An indifferent response to nematicide applications can be interpreted as a development of resistance. When treatment decreases a wild-type population (0-x) to a level below that of the wild-type population control (0-0), a similar effect might be expected in exposed populations (x-0). However, if an exposed population (x-x) is not comparably reduced to below the level of an exposed population control (x-0), resistance to the nematicide may be inferred (Yamashita *et al.*, 1986).

G. rostochiensis: At the first sampling, the nematicide-exposed males showed an indifferent response. The number of males (0 and 0) was significantly lower in 0-x populations than in 0-0 populations (53; Table 1). Male production in x-x populations, however, was not significantly different from that in 0.6-0 and 0.3-0 populations (four 0 values vs. 20 and 13). At the second sampling, an indifferent response was observed for the swollen stages and the males. Aldicarb treatments (0.6 g/m^2) significantly reduced these stages in the wild-type population (0 vs. 140 and 20 vs. 147 for swollen stages and males, respectively). The same

Table 2. *Mean number of different stages of* Meloidogyne naasi *found in 5 g wheat root tissue at two sampling dates.*

Sampling date	Developmental stage	Aldicarb dose (g/m^2)			
		before 1991	in 1991		
			0.6	0.3	0
06.06.1991	Second stage juveniles	0.6	673 *d*	906 *cd*	2220 *abc*
		0.3	987 *bcd*	3067 *ab*	4467 *a*
		0	133 *e*	800 *cd*	1493 *abcd*
	Swollen juvenile stages	0.6	0 *b*	0 *b*	773 *a*
		0.3	13 *b*	13 *b*	2333 *a*
		0	13 *b*	0 *b*	347*a*
23.07.1991	Second stage juveniles	0.6	40 *b*	133 *b*	2333 *a*
		0.3	33 *b*	7 *b*	8000 *a*
		0	7 *b*	93 *b*	3333 *a*
	Females	0.6	27 *c*	47 *c*	6000 *a*
		0.3	20 *c*	100 *c*	3333 *a*
		0	0 *d*	20 *c*	1033 *b*
	Eggs	0.6	813 *c*	8580 *bc*	450 000 *a*
		0.3	1227 *c*	3867 *c*	403 333 *a*
		0	193 *d*	4427 *c*	93 333 *ab*

Per sampling date and stage, numbers followed by the same letter are not significantly different according to the Duncan test ($P \leq 0.05$).

treatments, however, did not influence significantly the 0.3 g/m^2 aldicarb exposed populations (13 vs. 33 and 27 vs. 27 for swollen stages and males, respectively).

M. naasi and *P. crenatus:* none of the stages of these two nematodes showed an indifferent response at both samplings.

LARGER NEMATODE NUMBERS IN PREVIOUSLY EXPOSED POPULATIONS THAN IN WILD-TYPE POPULATIONS

An increase in nematode numbers following nematicide treatment of previously exposed populations (x-x) greater than the nematode number increase following treatment of wild-type populations (0-x) would provide evidence of the development of resistance in the exposed population.

G. rostochiensis and *P. crenatus:* With regard to the larger population effect, no significant differences were observed for these species.

M. naasi: Aldicarb treatment (0.6 g/m^2 aldicarb) of populations previously exposed to nematicide (x-0.6), produced, at the first sampling, a significantly larger number of second stage juveniles (673 and 987) than did wild-type (0-0.6) populations (133; Table 2). The 0.3 g/m^2 aldicarb treatment apparently induced resistance as observed after a 0.3 g/m^2 aldicarb exposure (0.3-0.3 vs. 0-0.3; 3067 vs. 800). At the second sampling, the egg number found after a 0.6 g/m^2 aldicarb

Table 3. *Mean number of* Pratylenchus crenatus *individuals found in 5 g maize root tissue at two sampling dates.*

Sampling date	Aldicarb dose (g/m^2)			
	before 1991	in 1991		
		0.6	0.3	0
06.06.91	0.6	139 *de*	1667 *c*	24 617 *b*
	0.3	108 *e*	317 *cd*	39 538 *ab*
	0	172 *de*	603 *cd*	62 083 *a*
23.07.91	0.6	600 *c*	520 *c*	90 200 *a*
	0.3	1020 *bc*	4740 *b*	80 200 *a*
	0	1180 *bc*	2560 *b*	86 400 *a*

Per sampling date and stage, numbers followed by the same letter are not significantly different according to the Duncan test ($P \leq 0.05$).

treatment was significantly larger in x-0.6 populations than in the 0-0.6 population (813 and 1227 vs. 193).

Discussion

Our results show that long term aldicarb applications affected the development of *G. rostochiensis*, *M. naasi*, and *P. crenatus* inside their host roots. Different kinds of effects on the developmental stages were observed: *i*) lower population levels in x-0 populations than in x-x populations; *ii*) increased susceptibility in x-x populations; *iii*) resistance phenomena as expressed by indifference to nematicides; and *iv*) resistance as demonstrated by larger population levels in x-x populations. These effects were influenced by the stage of the nematode, the exposure dose, and the time of observation. The effects, however, were not constant and not consistent from one stage to another. If any effects were present in adult stages, they were nearly always of the non resistance-type. Resistance in adults was only observed with males of *G. rostochiensis* (indifferent response at both samplings).

In our experiment, selective pressures, required for the development of non-fumigant nematicide resistance under field conditions, were apparently present. However, at the end of a series of fifteen consecutive annual aldicarb applications, differences between wild-type and nematicide-exposed populations were not significant. Therefore, it can be concluded that the required degree of selective pressures (Wright, 1981) was absent. The buffer effect of the soil probably contributed to this result. In the field, part of the nematode population will likely lie out of the zone of action of the nematicide. Nematicides are seldom equally distributed throughout the soil profile and it is probable that nematodes are not all exposed to the same rate of nematicide. Moreover, aldicarb can leach to deeper soil layers, depending on climatic conditions (Smelt & Leistra, 1992).

The influence of soil-inhabiting micro-organisms on pesticide degradation is another factor which reduces nematicide efficacy in soil (Smelt & Leistra, 1992). Occasionally, accelerated degradation develops after only one application, as observed by Harris *et al.* (1984). However, this may not occur with other combinations of nematicide and soil, even after a long series of applications. It appears unlikely that accelerated degradation occurred in our experiment: if it had occurred, it would have neutralised, at least partially, the nematicide treatments in the sixteenth year.

Yamashita and Viglierchio (1987*a*) were able to demonstrate the development of field resistance to non-fumigant nematicides in *X. index*. The results of their tests suggested that the development of non-fumigant nematicide resistance under field conditions does not require a continuous monthly exposure regimen. Apparently, in the vineyard used for their tests, sufficient selective pressures were available to enable resistance to develop. The authors suggested that the development of resistance may vary with the specific host, nematode species and strain, nematicide, soil types, and related factors. An important difference between their experiment and ours is found in the perennial character of the vineyard, which authorizes a continuous multiplication of the nematode.

In the last 8 years, no reports of field resistance were published. Apparently, if resistance does appear, it is very scarce. Resistance may be masked by opposing forces. As Yamashita and Viglierchio (1987*a*) suggest, nematode selection for resistance may not necessarily evolve concomitant qualities of fitness for the environment. The same authors also accept a low reproductive potential of treated populations as one of the possible masking effects. Our experiment, however, clearly demonstrates that long term nematicide applications do not influence the reproductive potential of *G. rostochiensis, M. naasi*, and *P. crenatus*.

Some of the criteria for demonstrating resistance advocated by Yamashita *et al.* (1986) and used in this paper may not be sufficiently rigorous to sustain the conclusion for evidence for resistance. The recording of nematode numbers greater in exposed populations than in wild-type populations is seen as a development of resistance. This effect was observed only in *M. naasi*. This behaviour, however, could be explained by the effect of a natural mechanism causing biological control in the absence of a nematicide treatment. In presence of such a treatment, the natural control may have been removed, allowing a greater survival of second stage juveniles. Further experimental work in which more factors can be controlled is needed.

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