Vol. 6. No. 1, pp. 98-101 Afro-Asian Journal of Nematology June, 1996

Nematicidal impact on the embryogenesis of *Cephalobus persegnis*

Qudsia Tahseen, M. Shamim Jairajpuri and Irfan Ahmad

Section of Nematology, Department of Zoology, Aligarh Muslim University, Aligarh 202 002, India

Abstract. The impact of nematicides *viz.*, carbofuran, phorate, posse, aldicarb and endosulfan was tested on the embryogenesis of *Cephalobus persegnis*. The nematicides enhanced the embryonation period with the maximum effect being for endosulfan and minimum for carbofuran and phorate. In 50 ppm concentration hatching could not take place only in eggs treated with endosulfan. Development did not proceed beyond gastrulation in single celled eggs treated with 100 and 150 ppm of all nematicides. However, the development continued up to late pretzel stage if the eggs were treated at gastrula stage. The nematicides at 200 ppm drastically affected the developmental process.

Keywords: *Cephalobus persegnis*, concentration, embryogensis, hatching, nematicides, nematode development, toxicity.

INTRODUCTION

The impact of nematicides is definitely adverse not only on the target organisms but also on the free living nematodes which form an integral part of the soil-ecosystem. The studies revealing the effects of nematicides on the reproduction and development of the latter are rather few apparently because of their unimportance in agriculture. It has been concluded by Van Gundy and McKenry (1977) that unhatched juveniles of nematodes were less sensitive to the same concentration of nematicides than the hatched ones. Wright (1981) observed that the embryonic development of nematodes was blocked by certain nematicides/antihelminthic compounds. He emphasised the impact of different nematicides on the embryogenesis and hatching of a free living cephalobid, *Cephalobus persegnis* Bastian, 1965.

MATERIALS AND METHODS

The nematicides used for this study belong either to the carbamate group (carbofuran 3%, posse 25%, phorate 3% and aldicarb 10%) or the organochlorine group (endosulfan 35%). Concentrations of 50 ppm, 100 ppm, 150 ppm and 200 ppm were tested (25 ppm concentration caused insignificant effects on the embryonic development).

For studying the effects of nematicides on embryogenesis, boiled agar was poured into special observation chambers (Ahmad and Jairajpuri, 1979) and allowed to gelate. The eggs were placed on the surface in a drop of nematicide and covered with a coverslip. Observations were made till hatching or cessation of development. The experiments were replicated five times and for each observation at least 10 eggs were studied. Controls were run without nematicides at 25±2°C.

RESULTS

Effect at 50 ppm (Fig. 1): All the nematicides prolonged the embryonation period of *C. persegnis*, the maximum effect being in endosulfan. Carbofuran and phorate had the least effects. The total duration up to the formation of a full fledged juvenile in endosulfan was about three times greater than that of the control. Also, the eggs treated with endosulfan in early embryonic (pregastrulation) stages did not hatch, although they developed to the late pretzel stage. However, the eggs treated with the same after gastrulation hatched successfully.

Effect at 100 ppm (Fig. 2): 100 ppm concentration of all nematicides further increased the duration of embryogenesis. At this particular concentration the eggs, if placed in pre-gastrula stage, did not develop beyond gastrulation in any nematicide. However, eggs treated at gastrula or postgastrula stages successfully formed juveniles although hatching was slightly delayed. It was further observed that the exposure time/duration of the eggs from single cell to gastrulation and from gastrula to hatching was more or less the same. Endosulfan increased the duration of embryogenesis the most.

Effect at 150 ppm (Fig. 3): The nematicides at 150 ppm lengthened the duration between embryonic stages more than at 100 ppm. Endosulfan appeared to be the most toxic while carbofuran and phorate the least toxic nematicides. The eggs, if placed in endosulfan at gastrula or post gastrula stages, failed to undergo eclosion and hatching could occur only in 20% cases. However, the percentage hatch was relatively higher in phorate (83%), carbofuran (80%), aldicarb (60%) and posse (50%). Juveniles within the treated eggs showed slower body movements and weaker oesophageal pulsations prior to hatching as compared to the control.

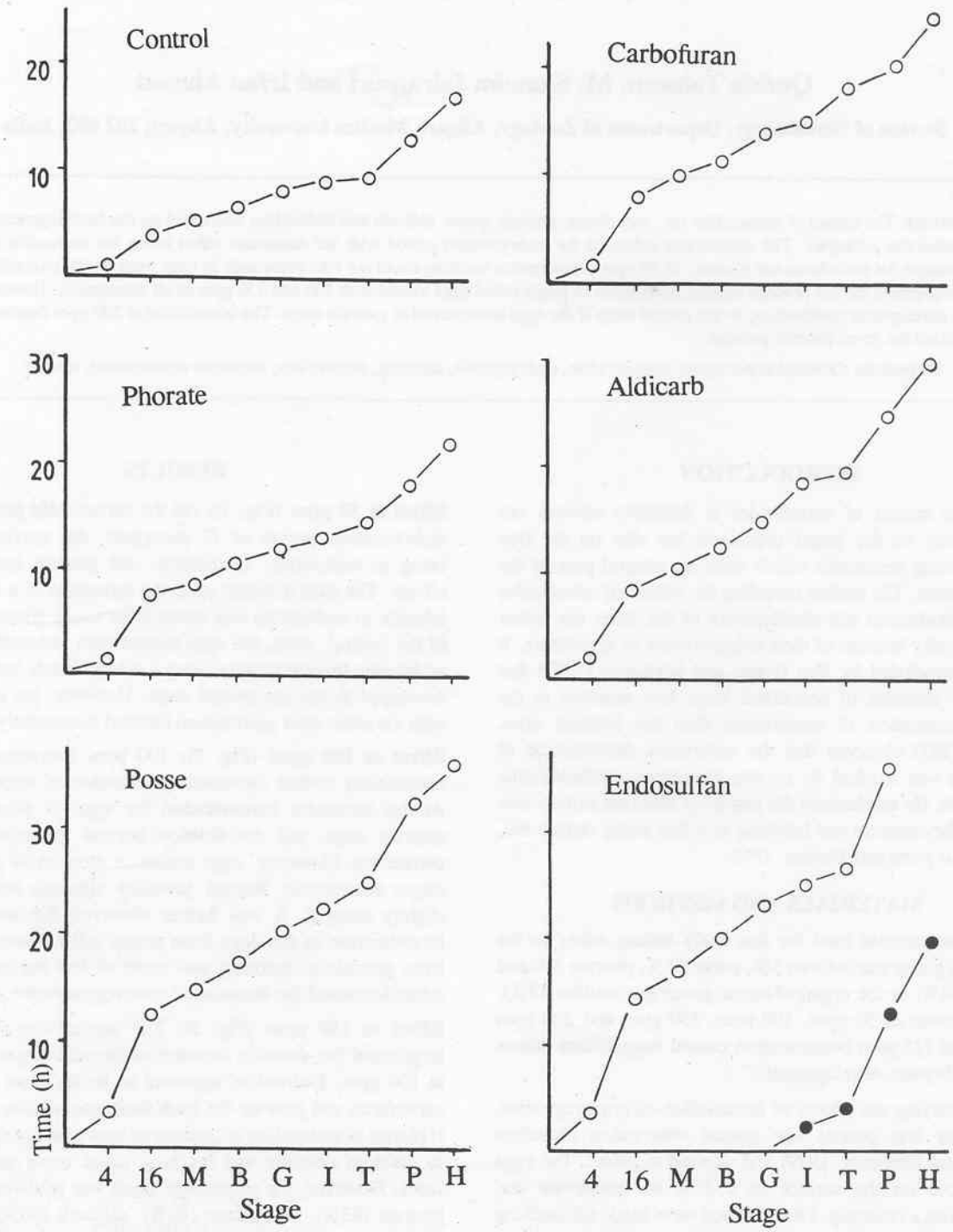

Fig. 1. Duration of successive embryonic stages of *Cephalobus persegnis* in 50 ppm concentration of nematicides. Stages: 4 = four celled stage; 16 = sixteen celled stage; I = invagination stage; T = tadpole stage; p = pretzel stage; H = hatching.

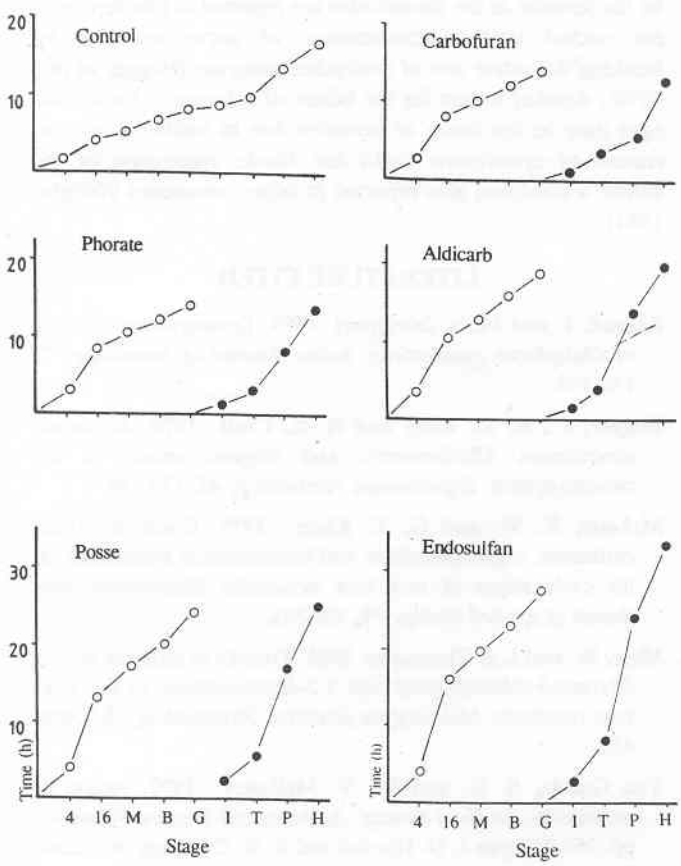

Fig. 2. Duration of successive embryonic stages of *Cephalobus persegnis* in 100 ppm concentration of nematicides. Stages: 4 = four celled stage; 16 = sixteen celled stage; I = invagination stage; T = tadpole stage; p = pretzel stage; H = hatching.

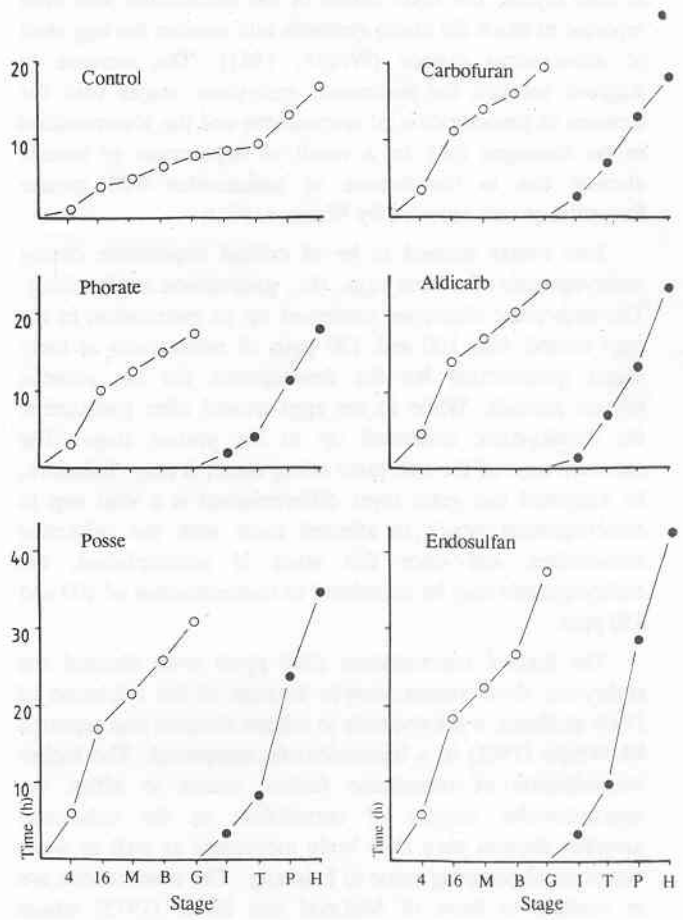

Fig. 3. Duration of successive embryonic stages of *Cephalobus persegnis* in 150 ppm concentration of nematicides. Stages: 4 = four celled stage; 16 = sixteen celled stage; I = invagination stage; T = tadpole stage; p = pretzel stage; H = hatching.

Effect at 200 ppm: All nematicides at 200 ppm completely inhibited the eclosion of juveniles. The eggs also failed to complete embryogenesis and the development stopped shortly after treatment with nematicides. Nevertheless, the division continued up to a maximum of 18 h, 26 h, 30 h, 32 h and 38 h in endosulfan, posse, aldicarb, carbofuran and phorate, respectively after which the eggs became non-viable. Eggs treated with carbofuran and phorate in the postgastrula stage completed organogensis and attained late pretzel condition. However, in aldicarb and posse only those embryos completed the organogenesis which were treated at the tadpole stage. In the case of endosulfan, even the embryos treated at the tadpole stage failed to undergo organogenesis and the eggs became non-viable before attaining the pretzel stage. In some cases the cleavage patterns were also affected and the blastomeres divided randomly without any definite pattern resulting in a mass of irregularly arranged blastomeres. The division in such cases did not proceed further.

DISCUSSION

Eggs of nematodes are comparatively less sensitive to nematicides than the hatched juveniles (Moje and Thomason, 1963) because they possess an efficient barrier in form of an egg shell to prevent the sudden entry of nematicides. The nematicides later gain entry because of the porosity of the shell as well as due to their toxic nature, which may cause distortion

in shell layers. The toxic nature of the nematicides was even reported to block the chitin synthesis and weaken the egg shell of *Aphelenchus avenae* (Wright, 1981). The increase in duration between the successive embryonic stages with the increase in concentration of nematicides and the abnormalities in the cleavages may be a result of impairment of mitotic division due to interference of nematicides with spindle formation as also reported by Wright (1981).

Two events seemed to be of critical importance during embryogenesis of treated eggs, *viz.*, gastrulation and hatching. The embryonic cleavages continued up to gastrulation in the eggs treated with 100 and 150 ppm of nematicides at early stages (pregastrula) but the development did not proceed beyond gastrula. While in the eggs treated after gastrulation the development continued up to the pretzel stage. The exposure time of the two cases being equal, it may, therefore, be suggested that germ layer differentiation is a vital step in embryogenesis which is affected most with the infiltrated nematicides, and once this stage is accomplished, the embryogenesis may be completed in concentrations of 100 and 150 ppm.

The highest concentration (200 ppm) even blocked the embryonic development, maybe because of the inhibition of DNA synthesis, a pre-requisite to mitotic division also reported by Wright (1981) in a benzimidazole compound. The higher concentration of nematicide further seems to affect the neuromuscular activity of nematodes, as the unhatched juveniles showed very slow body movement as well as weak oesophageal pumping prior to hatching. The observations are in contrast to those of McLeod and Khair (1975) where *Meloidogyne* spp. juveniles showed normal movement and spear thrusting. The eclosion of juveniles is probably inhibited because of the entry of large volume of nematicide through the thin shell which stretches and expands due to growth and enlargement of the embryo and also due to emulsification of the lipid layer. The hatching further seemed to be inhibited because of lack of perception of extrinsic and intrinsic stimuli by the juvenile as the nematicides are reported to interfere with the normal synaptic transmission of nerve impulses by blocking the active site of acetlycholinesterase (Hogger *et al.*, 1978). Another reason for the failure of eclosion in the treated eggs may be the death of juveniles due to oxidation of iron centres of cytochrome chain that blocks respiration of the worm, a condition also reported in other nematodes (Wright, 1981).

LITERATURE CITED

Ahmad, I. and M. S. Jairajpuri 1979. Developmental biology of *Chiloplacus symmetricus*. *Indian Journal of Nematology* **7**, 123-139.

Hogger, C., R. H. Estey and N. A. Croll 1978. *Xiphinema americanum*. Cholinesterase and biogenic amines in the nervous system. *Experimental Parasitology* **45**, 139-149.

McLeod, R. W. and G. T. Khair 1975. Effect of oxime-carbamate, organophosphate and benzimidazole nematicides on life cycle stages of root knot nematodes *Meloidogyne* spp. *Annals of Applied Biology* **79**, 329-341.

Moje, W. and I. J. Thomason 1963. Toxicity of mixtures of 1,2-dibromo-3-chloropropane and 1,2-dibromoethane to the root-knot nematode, *Meloidogyne javanica*. *Phytopatholgy* **53**, 428-431.

Van Gundy, S. D. and M. V. McKenry 1977. Action of nematicides. In *Plant disease*: *An Advanced Treatise Volume 1*, pp. 263-283 (eds J. G. Horsfall and E. B. Cowling). Academic Press.

Wright, D. J. 1981. Nematicides: Mode of action and new approaches to chemical control. In: *Plant Parasitic Nematodes Vol. III*, pp. 421-449 (eds B. M. Zuckerman and R. A. Rohde). Academic Press.