Significance of digitate appendage of *Hexamermis* sp.

K. Subbiah
Department of Zoology,
Government Arts College, Ootacamund 643 002, India

The juveniles of *Hexamermis* sp. are parasitic on tea-flushworms. They have caudal appendages. The functional significance of the appendages is discussed.

THE occurrence of juveniles of *Hexamermis* sp. (Nematoda: Mermithidae), on tea-flushworm *Cydia leucostoma* was reported by Subbiah[1]. During an investigation, it was observed that the juveniles were found to emerge from the thoracic region of the host with the posterior end protruding out first (Figure 1*a*). This region has a digitate appendage at the tip (Figure 1*b*). It is also called caudal appendage or tail appendage. This organ measures about 55 µm in length and 18.5 um width at the base both in the smaller and bigger juveniles. It is broad at the base and pointed at the tip (Figure 1*c*). According to Wouts[2], this characterstic organ is formed during the parasitic stage of *Hexamermis* juveniles. He also reported that it is the result of extensive enlargement of the body in which the tail-tip does not take part. The post-parasitic juveniles also continue to possess this appendage. The significance of this appendage has not so far been reported.

Figure 1. ***a***, *Hexamermis* juvenile emerging from thoracic region of flush-worm (× 12). ***b***, Posterior end of *Hexamermis* showing digitate appendage (× 240). ***c***, Digitate appendage enlarged; note the slightly serrated profile of the appendage (× 600).

The juveniles in this instance lack odontostyle, which is the piercing organ either to enter the host-body or to emerge from the haemocoel. The jabbing of the stylet and the flow of the digestive enzyme for juvenile emergence have been reported earlier[3]. The caudal appendage has also been observed to secrete a milky-fluid. That may probably contain enzymes for the dissolution of cuticle while emerging from the body-cavity. In the light of the above observation it is suggested that the caudal appendage might be helpful in piercing through the hosts body-wall for the swift emergence in the absence of odontostyle.

1. Subbiah, *Curr. Sci.*, 1986, **55**, 1047.
2. Wouts, W. M., in *Plant and Insect nematodes* (ed. Nickle, W. R.), Marcel Dekker, New York and Basel, 1984, p. 925.
3. Welch, H. E., in *Insect Pathology* (ed. Steinhaus, E. A.), Academic Press, New York, 1963, Vol II pp. 363–393.

10 May 1989

Effects of methyl isocyanate on renal function

S. Sundaramoorthy, K. Prabhakaran and A. Namasivayam
Department of Physiology,
Dr A. L. Mudaliar PG Institute of Basic Medical Sciences,
University of Madras, Madras 600 113, India

Freshly prepared methyl isocyanate when administered intraperitoneally (0.02 ml) diluted with equal volume of clinical dextran to albino rats induces nephrotoxic effects as manifested by decreased glomerular filtration rate, renal plasma and blood flow, filtration fraction and tubular maximum for *para*-aminohippuric acid at 30 min. The renal functions recovered at 24 h indicating acute nephrotoxic stress induced by sublethal doses of methyl isocyanate.

THE pathophysiological effects of methyl isocyanate (MIC) have gained importance in recent times after the Bhopal disaster. Early findings have shown that MIC is a potent irritant of the respiratory tract[1-3]. Subsequent investigations

Table 1. Glomerular filtration rate (ml/min) after MIC administration to Albino rats.

Controls (n=12)	0.5 h (n=10)	1 h (n=7)	4 h (n=7)	8 h (n=7)	24 h (n=5)	48 h (n=5)	72 h (n=5)
0:181 ±0.07	0.029 ±0.016	0.03 ± 0.011	0.048 ±0.025	0.25 ±0.03	0.125 ±0.025	0.137 ± 0.03	0.197 ± 0.13

Values are expressed as mean ± SD.

Table 2. Renal function tests after MIC administration to Albino rats.

Parameters	Controls (n = 12)	0.5 h (n = 10)	24 h (n = 7)	48 h (n = 7)	72 h (n = 5)
Renal plasma flow (ml/min)	0.464 ± 0.27	0.085 ± 0.04	0.414 ± 0.19	0.36 ± 0.04	0.358 ± 0.2
Renal blood flow (ml/min)	0.85 ± 0.56	0.171 ± 0.08	0.780 ± 0.40	0.698 ± 0.38	0.46 ± 0.38
Filtration fraction	0.391 ± 0.17	0.351 ± 0.25	0.341 ± 0.10	0.378 ± 0.10	0.552 ± 0.19
TmPAH (mg/min)	23.52 ± 19.3	9.576 ± 4.8	18.06 ± 0.5	22.0 ± 3.7	29.27 ± 4.3

Values are expressed as mean ± SD.

have shown that a single exposure of MIC can induce toxic changes in the bone marrow[4]. Experiments with another isocyanate, -toluene diisocyanate, showed that, this chemical after a single exposure by inhalation, combined with plasma proteins irreversibly[5]. Recently Bhattacharya *et al.*[6] have shown the binding of ^{14}C-labelled MIC to various tissue proteins in rats and the binding was detectable even after 10 days following a single administration of labelled MIC. Isocyanates react with sulphhydryl groups of protein molecules[7]. It is presumed that MIC after gaining entry into the body through the blood might influence a number of physiological systems either directly or indirectly. A perusal of the available literature shows that virtually nothing is known about the effects of MIC on renal function. This report gives an account of exploratory investigations on the renotoxic effect in rats after a single exposure to MIC.

MIC synthesized in this laboratory by treating sodium azide with acetic anhydride was used. Wistar strain albino rats of either sex weighing 150–160 g were used. Untreated animals were served as controls (n=12). Freshly prepared MIC 0.02 ml (which is one half the LD_{50}) diluted with an equal volume of clinical dextran was injected intraperitoneally to the test animals. This dose was used in this study as it was well tolerated by the animals. In MIC-treated animals glomerular filtration was estimated by inulin clearance[8] at 0.5 (n=10), 1 (n=7), 2 (n=7), 4 (n=7), 6 (n=7), 8 (n=7), 24 (n=5), 48 (n=5), and 72 (n=5) hours (Table 1). Renal plasma flow was estimated in MIC-treated animals at 0.5 (n=7), 24 (n=7), 48 (n=7) and 72 (n=5) hours using *para*-aminohippuric acid clearance test[9]. Renal blood flow, filtration fraction and tubular maximum for PAH were calculated from these values (Table 2).

The present results indicate that all the parameters studied were adversely affected in a statistically significant manner at 30 min. The renal functions recovered at 24 h indicating an acute nephrotoxic stress induced by sublethal dose of MIC. In addition there was mild to moderate proteinuria in the early samples of urine, the pattern of which changed at 48 h. In this connection it is pertinent to point out the work of Jeevarathinam *et al.*[10] who have shown that kidney parenchyma in 0.5 LD_{50} MIC-treated animals was essentially normal except for a mild to moderate degree of congestion and focal lymphocytic infiltration. In 1 LD_{50}-treated animals in addition to these changes, the proximal convoluted tubules showed cloudy swelling and some degree of coagulative necrosis. These authors suggest that acute toxicity of MIC *in vivo* may be mediated by its effect on vascular beds.

The nephrotoxic effects of MIC are under further investigation in order to elucidate the mechanism of action of the toxin.

1. Ferguson, J. S., Schaper, M., Stock, M. F., Weyel, D. A. and Alarie, Y., *Toxicol. Appl. Pharmacol.*, 1986, **94**, 329.
2. Alarie, Y., Ferguson, J. S., Stock, M. F., Weyel, D. A. and Schaper, M. *Environ. Health. Perspect.*, 1987, **72**, 159.
3. Nemer, B., Dinsdale, D., Sparrow, S. and Ray, D. E., *Br. J. Ind. Med.*, 1985, **42**, 799.
4. Conner, M. K., Rubinson, H. F., Ferguson, J. S., Stock, M. F. and Alarie, Y., *Environ. Health-Prespect.*, 1987, **72**, 177.
5. Hill, B. L. (as quoted by Ferguson *et al.*). *Toxicol. Appl. Pharmacol.*, 1986, **94**, 104.
6. Bhattacharya, B. K., Sharma, S. K. and Jaiswal, D. K., *Biochem. Pharmacol.*, 1988, **37**, 2489.
7. Brown, W. E., Green, A. H., Cedal, T. E. and Cairns, *J., Environ. Health. Perspect.*, 1987, **72**, 5.
8. Bratton, A. C. and Marshall, F. K., *J. Biol. Chem.*, 1939, **128**, 537.
9. Bacon, J. S. D. and Bell, D. J., *Biochem. J.*, 1948, **42**, 397.
10. Jeevarathinam, K. Selvamurthy, W., Ray, U. S., Mukhopadhyay, S. and Thakur, L., *Toxicology*, 1988, **51**, 223.

ACKNOWLEDGEMENT. We are grateful to The Scientific Commission for Continuing Studies on the Effects of Bhopal Gas Leakage on Life Systems, Cabinet Secretariat, Government of India, for their financial support.

24 October 1988; revised 2 November 1989