sponse was obtained in 1 dorsal preparation (9×10^{-4} g/ml).

Discussion. The sensitivity of the muscle to ACh and its potentiation by eserine are similar to previous observations[10,11], suggesting that ACh is a neuro-muscular transmitter in Onychophora.

As found by previous authors[10,11] muscle preparations without the central nervous system do not respond to catecholamines. Ventral preparations, however, respond with increases in tone and spontaneous activity. This suggests that these agents act within the ventral nerve cords. The different effects of NA and DA on tone and spontaneous activity suggest possibly that these 2 catecholamines may act by different mechanisms. The action of iproniazid suggests the presence of monoamine oxidase in the nervous system.

5HT probably has an inhibitory effect on the musculature but a mixed excitatory-inhibitory effect on preparations containing the central nervous system. It seems possible that 5HT increases muscle tone and spontaneous activity when it acts on the nervous system, but if acting directly on the muscles as well it may inhibit their activity.

The monoamine responses of *Peripatopsis* are qualitatively similar to those in the annelid *Lumbricus terrestris*[14], except for the inhibitory effect of 5HT on the muscle. Inhibition was observed, however, in preparations from other annelids[15,16]. Our results may be physiologically significant since catecholamines and 5HT are present within the nervous system[17].

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The presence of monoamines in the nervous system of *Peripatopsis* (Onychophora)

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Summary. Preliminary observations of formaldehyde-induced fluorescence support the suggestion that monoamines such as noradrenaline, dopamine and 5-hydroxytryptamine are transmitter agents in the central nervous system of Onychophora.

Experiments with preparations of the body wall of *Peripatopsis moseleyi* show that a response of the longitudinal muscle to noradrenaline and dopamine depends on the presence of the ventral nerve cord[2]. We therefore examined the distribution of formaldehyde-induced fluorescence in the nervous system, following the method of Falck-Hillarp[3].

The results reported here were obtained with a specimen of *Peripatopsis sedgwicki* and have since been confirmed with *P. moseleyi*[4]. The specimen was anaesthetized with ether and cut into pieces which were quenched in isopentane cooled over liquid nitrogen, freeze-dried and treated with formaldehyde, and embedded in paraffin wax. Orientated 10 µm sections were trimmed and mounted in liquid paraffin. They were examined using a Leitz Ortholux microscope equipped with filters BG12 and K490.

As seen in figure 1 there are several brightly fluorescent tracts in the ventral nerve cords. Their green colour is consistent with the presence of noradrenaline or dopamine or related substances. A few green fluorescent nerve cell bodies were seen from time to time in the cortex of the nerve cord. Yellow cells, which probably contain 5-hydroxytryptamine (or similar compounds) were also observed there, and in some sections rather tenuous yellow fluorescent tracts were seen next to green fibre bundles.

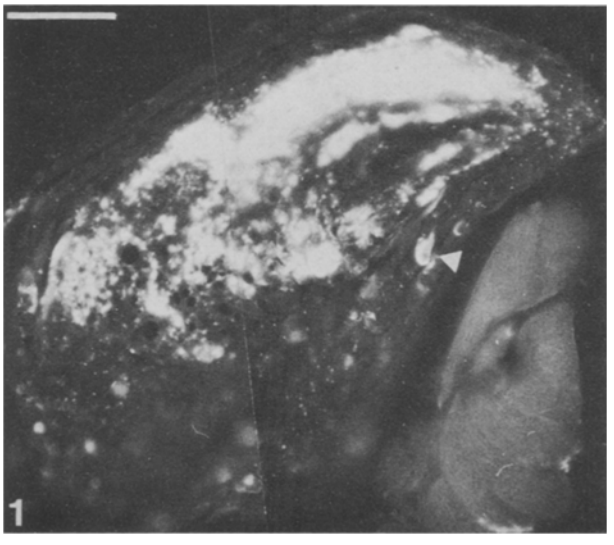

Fig. 1. Right nerve cord of *Peripatopsis sedgwicki* in transverse section, showing green fluorescent tracts. Arrow indicates a small green nerve cell. Bar = 50 µm.

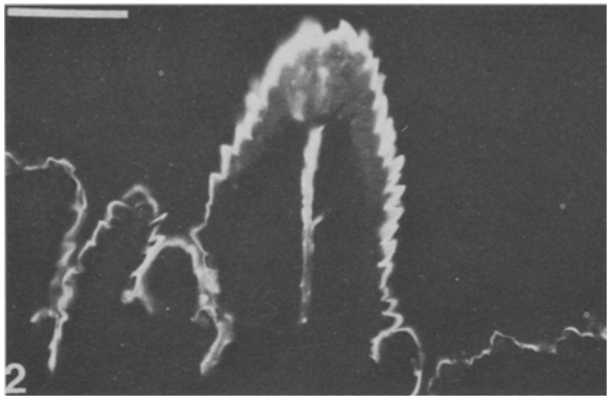

Fig. 2. Transverse section of epidermis showing the green fluorescent nerve leaving a sense organ. The cuticle is autofluorescent. Bar = 50 µm.

The sensory nerves of peripheral sense organs fluoresce with a blue-green colour similar to that of tracts in the nerve cords (figure 2). As they join segmental nerves, some of the fluorescent tracts in the nerve cords may be afferent. It has been established that the sensory nerves of many invertebrates contain catecholamines[5].

Fine fluorescent fibres occur among the body wall muscle. They appear to be yellow in colour but their distribution has not yet been ascertained. Should they prove to be motor axons they could be either excitatory or inhibitory, the latter possibility being particularly interesting[6].

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Induction of deoxyribonucleic acid degradation in *Escherichia coli* by ozone[1]

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Summary. Cell survival and deoxyribonucleic acid (DNA) degradation were measured for wild-type *Escherichia coli* B251 cells after exposure to different concentrations of ozone. The results show that extensive breakdown of DNA occurs after ozonation and that the extent of ozone-induced DNA degradation generally correlates with the colony-forming ability of the cells.

Previous work on the effect of ozone on the survival of radiation-resistant and -sensitive strains of *Escherichia coli* suggested that lesions to deoxyribonucleic acid (DNA) might be responsible for killing of bacteria by ozone[3]. In agreement with this, ozone has been shown to modify markedly nucleic acids and bases[4,5] and to produce specific mutants in *E. coli*[6]. Therefore, it seemed interesting to determine the amount of DNA degraded in wild-type strain B251 of *E. coli* following exposure to different concentrations of ozone.

Materials and methods. Cells were grown overnight at 37 °C in glucose-salts medium (M9)[7] containing 10 µCi of thymidine-^{3}H (New England Nuclear Corp.; 40-60 Ci/mM) and 250 µg deoxyadenosine per ml. After several washings, cell suspensions were exposed in M9 medium to 5, 10, 25 and 50 µl/l ozone for 30 min as described[6]. Ozone survival curves were done on nutrient agar plates (Difco) as previously described[6]. The procedure for measuring ozone-induced DNA degradation was that of Strike and Emmerson[8]. Control experiments were carried out as above, except that clean air was used instead of ozone.

Results and discussion. The survival of strain B251 to different concentrations of ozone and for different intervals of treatment-time is represented in figure 1. According to these results, ozone showed no discernable effect on survival for exposure times ranging between 1 and 30 min at 5 µl/l. However, the killing capacity of ozone which appeared first at 10 µl/l (about 55% lethality after 30 min), was greatly increased at higher ozone concentrations (25 and 50 µl/l), more than 3 decades of killing occurring within the 30-min treatment period.

The amount of DNA degraded in B251 cells following a 30-min exposure to ozone was examined (figure 2). Expressed as a percentage of total radioactivity, the data show that more DNA is degraded and released into the medium by ozonated than by unozonated cells. Furthermore, the extent of this DNA breakdown increases both with ozone treatment and incubation time. This suggests that ozone produces lesions in the DNA which stimulate degradation; and

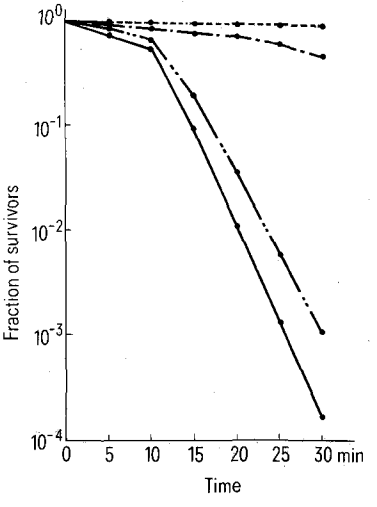

Fig. 1. Survival of wild-type strain B251 of *Escherichia coli* treated with different concentrations of ozone in complete growth medium: 5 µl/l (●– – –●); 10 µl/l (●– · –●); 25 µl/l (●– · · –●); 50 µl/l (●——●). Average of 3 independent experiments.