mesencephalon as parameters. This method also has one drawback—it is not fully applicable in laterally compressed fishes (*e.g., E. orbis*) in which the lobes increase in dorso-ventral direction and the length alone might not reflect the true allometric growth of the lobes. Nevertheless, a fairly correct deduction can be predicted regarding the olfactory capacity of the species by studying the two methods.

Histology of the olfactory epithelium of *A. testudineus* and *C. fasciatus* has been studied in detail and it is found that it does not differ from the basic vertebrate plan. Ciliated and non-ciliated supporting cells are present. Unidirectional beating of cilia creates a gentle flow of water current inside the olfactory chamber. The ethmoidal and lachrymal sacs do not differ in their cellular arrangements.

This study shows that *A. testudineus* and *E. orbis* are nose fishes (macrosmat), while *C. oblongus* is eye-nose fish, and *C. fasciatus*, *N. nandus* and *O. argenteus* are eye-fishes (microsmat). The conclusions drawn by morphological and histological studies can be corroborated by behavioural and ecological findings.

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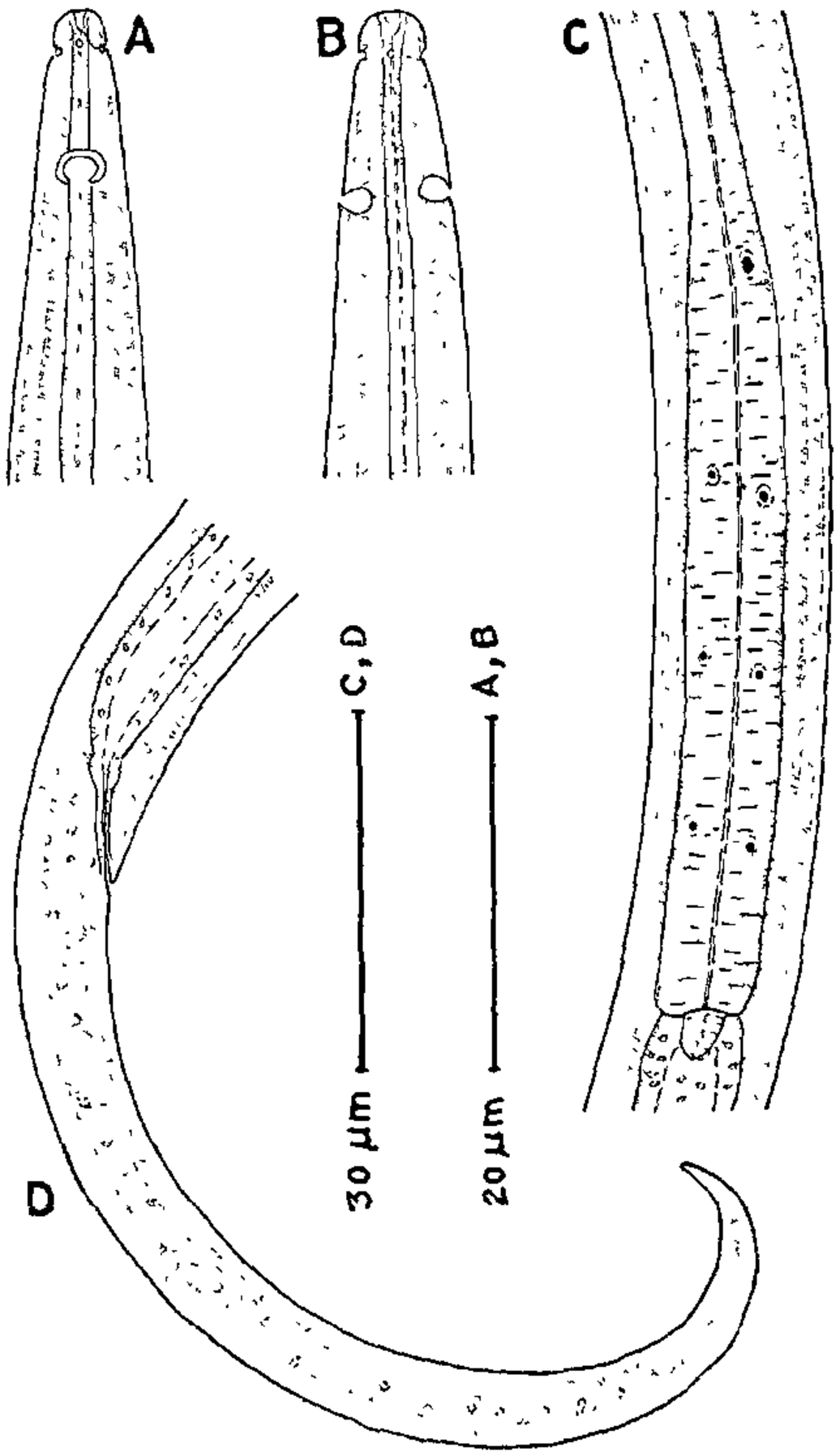

FIG. 1. A : Anterior end, lateral view; B : Anterior end, ventral view; C : Expanded part of oesophagus; D : Tail region.

FIRST RECORD OF THE GENUS *ETAMPHIDELUS* ANDRÁSSY, 1977 (NEMATODA) FROM INDIA

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ANDRÁSSY[1] proposed the genus *Etamphidelus* with the type species, *E. japonicus* and based its description on females and one male specimen collected in Japan. Five females of *E. japonicus* were found in the soil from around the roots of tea plants, *Camellia sinensis* L. from Maranda, district Palampur, Himachal Pradesh. This is the first record of the genus from India. The dimensions and description of the species are given below.

Etamphidelus japonicus Andrássy, 1977
(Fig. 1)

Dimensions

Females (5) : $L = 0{\cdot}90$–$1{\cdot}10$ mm;
$a = 75$–82; $b = 3{\cdot}6$–$4{\cdot}0$;
$c = 10$–12; $c' = 10$–17; $V = 62$–64.

Description

Body almost C-shaped upon fixation and tapering towards both extremities. Cuticle about 1 µm thick, finely striated. Lip region rounded, 3 µm or 1/5th–1/4th of midbody width. Stoma very small measuring

1–2 μm or about half of lip-width. Amphidial apertures oval, 3 μm wide, situated at 6–9 μm or 2–3 lip-widths from anterior end of body. Nerve ring at 117–127 μm from anterior end of body. Oesophagus 255–310 μm long, basal part occupying 25–29% of its length. Oesophago-intestinal junction 4–5 μm long. Rectum 9–12 μm or more than one anal body-width long. Female reproductive system mono-prodelphic, 186–205 μm long. Vulva transverse, vagina thick-walled, inclined anteriorly, 7–9 μm or 0·5–0·6 vulval body-width long. Tail elongate, conoid, 90–117 μm or 10–17 anal body-widths long. Vulva-anus distance 279–355 μm or 2·5–3·0 times the tail length.

Male : Not found.

Remarks

The present specimens conform with the description and dimensions of the species as given by Andrássy[1] except that they have a smaller and slender body, anteriorly situated amphidial apertures and vulva ($L = 1·14–1·16$ mm; $a = 54–59$; distance of amphid from anterior end = 15 μm; $V = 65–66$ according to Andrássy).

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LIPOFUSCIN ACCUMULATION AND LIPID PEROXIDATION IN RAT MYOCARDIUM AS A FUNCTION OF AGE

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Lipofuscin accumulation in the myocardium of 12½-, 25-, 50-, 75- and 100-week-old rats was investigated spectrophotofluorometrically by chloroform-methanol extraction method. Since heart is an organ that is particularly sensitive to lipid peroxidation, malonaldehyde (an end product of lipid peroxidation) production was also studied. A dramatic age-related increase in the lipofuscin concentration was observed. Lipid peroxidation rate also increased as a function of age. It is suggested that lipofuscin is formed by peroxidative destruction of polyunsaturated lipids of the subcellular membranes and their consequent co-polymerization with other biological molecules. Lipofuscin accumulation as a function of age is considered as a strong evidence of the occurrence of lipid peroxidation process in cells *in vivo* and it is suggested that there is a positive correlation between aging, lipofuscin and lipid peroxidation. The mode of formation and the possible functional significance of lipofuscin in the myocardium are discussed.

Introduction

Accumulation of age pigments or lipofuscin is one of the most prominent age associated cytological alterations in a variety of long-lived post-mitotic cells including the myocardial fibres[1]. The nature of the relationship between the lipofuscin granules and the process of aging, however, remains obscure[2]. Lipofuscin accumulation has been linked to the aging process because of a striking correlation found between the degree of accumulation and the actual age[3]. A major reason for the paucity of information on the functional significance of lipofuscin appears to be the lack of techniques for the quantitative determination of lipofuscin.

The origin of lipofuscin is not certain, but, it is generally thought to arise from the oxidative polymerization of unsaturated lipids and to co-exist in combination with the components of the lysosomes[4]. Evidence suggests that lipofuscin granules may originate from the peroxidation of unsaturated fatty acids, resulting in carbonyl compounds such as malonaldehyde[5]. These compounds may cross link with biological molecules, resulting in cross-polymers[5], that may be hydrolytically undigestible by lysosome and accumulate intracellularly as tertiary lysosomes or residual bodies[6].

In non-dividing cells like the myocardial cells, the indigestible substances can be observed without the evidence of exocytic elimination[7]. Cellular dysfunction in these cells may affect the metabolism in such a way as to cause a mechanical disruption of the cellular organization and lead to the eventual death of the cell[8].

The objective of the present study is to investigate the accumulation of lipofuscin and the lipid peroxidation potential of the myocardium as a function of age.

Materials and Methods

Male albino rats of 12½-, 25-, 50-, 75- and 100-week-old were used for the present study. The rats were decapitated and the myocardium was excised and washed in 0·9% saline to remove the blood clots.

Analysis of the fluorescent products was performed according to Fletcher *et al.*[9] on 200 mg of the tissue after homogenization and extraction with 2 : 1 chloroform-methanol mixture. The solvent to tissue ratio was 20 : 1 (V : W). Spectrophotofluorometric measurements were made with a Perkin Elmer MPF–44 fluorescence spectrophotometer. The excitation maximum was 365 nm and the emission maximum was 445 nm. The spectrofluorometer was standardized each time with a fresh solution of quinine sulphate. The instrument was calibrated to read 100 units for 1 μg of quinine sulphate/ml of 0·1 N sulphuric acid.