C. officinalis and *C. robusta* grown in South India and our findings are recorded below.

Adopting the standard procedure for the isolation of flavonoids, the aglycones present in the leaves were found to be mainly quercetin with small quantities of kaempferol. Of the three glycosides present, the one in appreciable quantity was isolated in pure form as light yellow needles, m.p. 210–12° (decomp). It was purple under U.V. changing to yellow with NH_3 and had λ_{max} (nm) 259, 359 and λ_{min} 238, 286 (EtOH) and showed shifts with diagnostic reagents[5] characteristic for a quercetin-3–0–glycoside. Its IR spectrum (KBr) exhibited bands at 1650, 1600, 1500, 1360, 1195, 1110, 1040, 1000, 945, 920; 825 and 800 cm^{-1}. On acid hydrolysis it yielded quercetin and L–arabinose in equal proportion. The identity of the sugar as L–arabinose was established by co-chromatography of the sample with authentic arabinose and xylose. They had R_f (arabinose, xylose) : 0·27, 0·27 (BAW) ; 0·55, 0·50 (phenol); 0·45, 0·48 (*t*-BAW) ; 0·30, 0·35 (ethyl acetate : pyridine : water = 10 : 4 : 3) ; 0·25, 0·27 (butanol : ethanol : water = 4 : 1 : 1) and 0·36, 0·44 (benzene : butanol : pyridine : water = 1 : 5 : 3 : 3). Thus, the glycoside was identified as quercetin–3–0–L–arabinoside and a direct comparison, co-chromatography and superimposable I.R. spectra with an authentic compound showed it to be avicularin (quercetin–3–0–α–L–arabinoside).

In order to ascertain whether the quercetin glycoside isolated from *C. ledgeriana* was really quercetin–3–xyloside or the so far misidentified reynoutrin, an attempt made to procure a small quantity of the sample isolated from *C. ledgeriana*[4] for comparison was unsuccessful. The absence of record of the occurrence of quercetin–3–0–xyloside so far in nature, the earlier misidentification of reynoutrin as a xyloside and the present isolation of avicularin from *C. officinalis* and *C. robusta* warrant a re-examination of the true nature of the quercetin pentoside[4] isolated from *C. ledgeriana*[4].

Our grateful thanks are due to Shri S. Kalyanasundaram, Botanist, Government Cinchona Plantations, Dodabetta, Nilgiris, for authentic leaves of *C. officinalis* and *C. robusta,* Drs. T. R. Govindachari and B. S. Joshi, Ciba–Geigy Research Centre, Bombay–63, and Dr. L. Horhammer, University of Munich, Germany, for the spectral data and the Principal, JIPMER, for encouragement.

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THE EFFECT OF TOLUENE, PHENOBARBITAL AND 3-METHYLCHOLANTHRENE ON HEPATIC MICROSOMAL LIPID PEROXIDATION

PHENOBARBITAL AND 3-METHYLCHOLANTHRENE are known to be potent inducers of a range of hepatic microsomal xenobiotic metabolizing enzymes. It has been reported that the pretreatment of rats with either of these compounds produces inhibition of hepatic microsomal lipid peroxidation[1]. Numerous attempts have been made to study the levels of liver lipid peroxides in carbon tetrachloride treated rats[2–4] and it has been reported that the butylated hydroxytoluene[5,6] and benzene[7] significantly reduce the lipid peroxide formation.

Except for a preliminary study[8], involving some aromatic hydrocarbons, there is no recorded information about the effect of organic solvents on hepatic microsomal lipid peroxidation in rat. Accordingly, the present paper reports the effect of toluene on liver microsomal lipid peroxidation. Furthermore, the effect of pretreatment of phenobarbital and 3-methylcholanthrene on the toxicity of toluene was also studied.

Materials and Methods

Hindustan antibiotic strain adult male (200–250 gm) and female (135–160 gm) albino rats were obtained from Hindustan-Antibiotics, Poona. The animals were kept in an air-conditioned room and supplied with rat pellets (obtained from Hindustan-Lever Ltd., Bombay) *ad libitum* prior to the initiation of the experiments. All animals were fed on a synthetic diet for 1 week before initiation of the experiments. The composition of the diet was as reported earlier[9] except that the casein content was increased to 18%. The rats were then allotted to the following groups and pair fed during the experimental period. (1) Control, (2) Toluene treated, (3) Phenobarbital treated, (4) Phenobarbital and toluene treated, (5) 3-Methylcholanthrene treated and (6) 3-Methylcholanthrene and toluene treated. Toluene (0·72 ml/kg body wt.) was administered orally to the rats using corn oil as a carrier in the morning (before feeding) for 2 successive days. Sodium phenobarbital (80 mg/kg body wt.) was injected intraperitoneally, daily, in the morning between 8·0–9·0 a.m. for 3 days,

Rats in group 4 which were treated with phenobarbital were further treated with toluene for 2 successive days. 3-Methylcholanthrene (obtained from Calbiochem, Switzerland) was injected intraperitoneally at a dose of 20 mg/kg body wt. in corn oil in the morning for 2 days. The rats in group 6 which were treated with 3-methylcholanthrene were further treated with toluene for 2 successive days.

Tissue Preparation.—The animals were killed by decapitation, 24 hours after the last injection. The whole livers were carefully perfused with 0·9% ice-cold saline, excised, weighed, minced and homogenized (1 : 4 w/v) in ice-cold 50 mM Tris-HCl buffer, pH 7·4 containing 1·15% KCl. All tissue preparations were made at — 2° C. The microsomes were isolated by the procedure of Baker *et al.*[10]. The microsomal protein was determined by the biuret method[11] using crystalline bovine serum albumin as the standard.

Lipid Peroxidation.—NADPH linked lipid peroxidation was assayed according to the method of Ernster and Nordenbrand[12]. Ascorbate-induced lipid peroxidation was carried out in the same medium but nicotinamide was omitted and NADPH was replaced by 1 mM ascorbate. The malonaldehyde formation was measured by the thiobarbituric acid reaction[13].

Results and Discussion

Table I shows the effect of various treatments on lipid peroxidation. Phenobarbital and 3-methylcholanthrene treatment resulted in lowering the NADPH linked and ascorbate induced lipid peroxidation irrespective of the sex. The magnitude of decrease in 3-methylcholanthrene treated male rats was more than in phenobarbital treated ones. Phenobarbital and 3-methylcholanthrene treatment resulted in a similar reduction in lipid peroxidation in female rats.

Toluene treatment lowered both the NADPH linked and ascorbate-induced lipid peroxidation irrespective of sex ; however, the decrease was more in male as compared to female rats. Lipid peroxide formation was further decreased in rats pretreated with phenobarbital prior to the administration of toluene in both sexes. However, pretreatment of 3-methylcholanthrene prior to the administration of toluene reduced lipid peroxidation significantly only in the male rats.

The inhibitory effect of toluene on lipid peroxidation may be due to the antioxidant property of toluene metabolites. Further decrease in lipid peroxidation in rats pretreated with phenobarbital or 3-methylcholanthrene may be due to competition for a common flavoprotein cytochrome *c* reductase[1] for the metabolism of these drugs. These results agree with the report of Wills[7].

TABLE I

*Effect of treatment of toluene on NADPH linked and ascorbate induced microsomal lipid peroxidation with and without pretreatment of phenobarbital and 3-methylcholanthrene in adult male and female rats**

Group	NADPH-linked lipid peroxidation†		Ascorbate-induced lipid peroxidation†	
	Male	Female	Male	Female
Control	16·20 ±0·20	27·40 ±0·20	17·80 ±0·20	19·40 ±0·20
Toluene treated	13·80 ±1·40[a]	25·80 ±0·20[a]	13·95 ±0·85[b]	17·40 ±0·20[a]
Phenobarbital treated	14·00 ±0·40[a]	17·20 ±0·40[c]	14·80 ±0·40[b]	16·20 ±0·20[b]
Phenobarbital + toluene treated	13·00 ±0·20[a]	19·00 ±0·20[b]	12·68 ±0·20[a]	17·80 ±0·20[a]
3-Methylcholanthrene treated	11·20 ±1·60[c]	21·20 ±1·20[b]	12·20 ±0·20[c]	17·00 ±1·00[a]
3-Methylcholanthrene + toluene terated	8·40 ±0·40[c]	21·20 ±0·40[b]	9·20 ±0·40[c]	16·80 ±0·80[a]

* The results are mean values (±SEM; 10 rats in each group).

† Activity is expressed as *n*-moles malonaldehyde formed/min/mg protein.

[a] = $P < 0·05$; [b] = $P < 0·01$; [c] = $P < 0·001$.

AMM is a Junior Research Fellow of U.G.C., New Delhi. The authors wish to thank Dr. J. M. Patel for his help during the early stages of the work.

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ON THE OCCURRENCE OF PRIAPULIDS IN THE LITTORAL REGION OF BHIMILIPATNAM (VISAKHAPATNAM, BAY OF BENGAL)

VERY little is known about the eucoelomate phylum Priapulida, which are marine vermiform cylindrical organisms attaining a length of over 12 cm with a bulbous anterior end, destitute of appendages and with an eversible proboscis. Surface of the body is superficially segmented and covered with spines.

Priapulids are hitherto known to occur only in the high latitudes and it is agreed that they have bipolar distribution and are absent in the tropics and subtropics[1-3]. Of late they have been reported from the Red Sea, Bermuda and Curacuo as meiofaunal components[4-6]. It is interesting to note that these organisms wherever reported were based on a small number of specimens only, whilst Coul[5] observed them as an important faunule constantly occurring in the meiobenthos forming 4·7% of the total proportion and ranked fourth in abundance accounting for 2% of all the meiobenthic animals collected. However, these animals were not known from Indian Ocean excepting for the Red Sea record. In the present communication the authors report on the occurrence of priapulids for the first time from the peninsular India and for the second time from Indian Ocean.

While engaged in the studies of littoral benthos of a backwater region at Bhimilipatnam about 35 km north of Visakhapatnam (70° 44' N and 83° 23' E) on the east coast of India, two priapulids (Fig. 1) were collected from a sediment comprising mud with sand, rock and shell pieces and rich in organic debris. The variation of ambient salinity ranged from 10‰ (during monsoon) to 37‰ (during summer) and temperature from 20° to 35° C.

Absence of tentacles, eversible proboscis with longitudinal rows of papillae, superficially segmented body covered with spines, mouth associated with recurved spines are the salient features of the specimens collected.

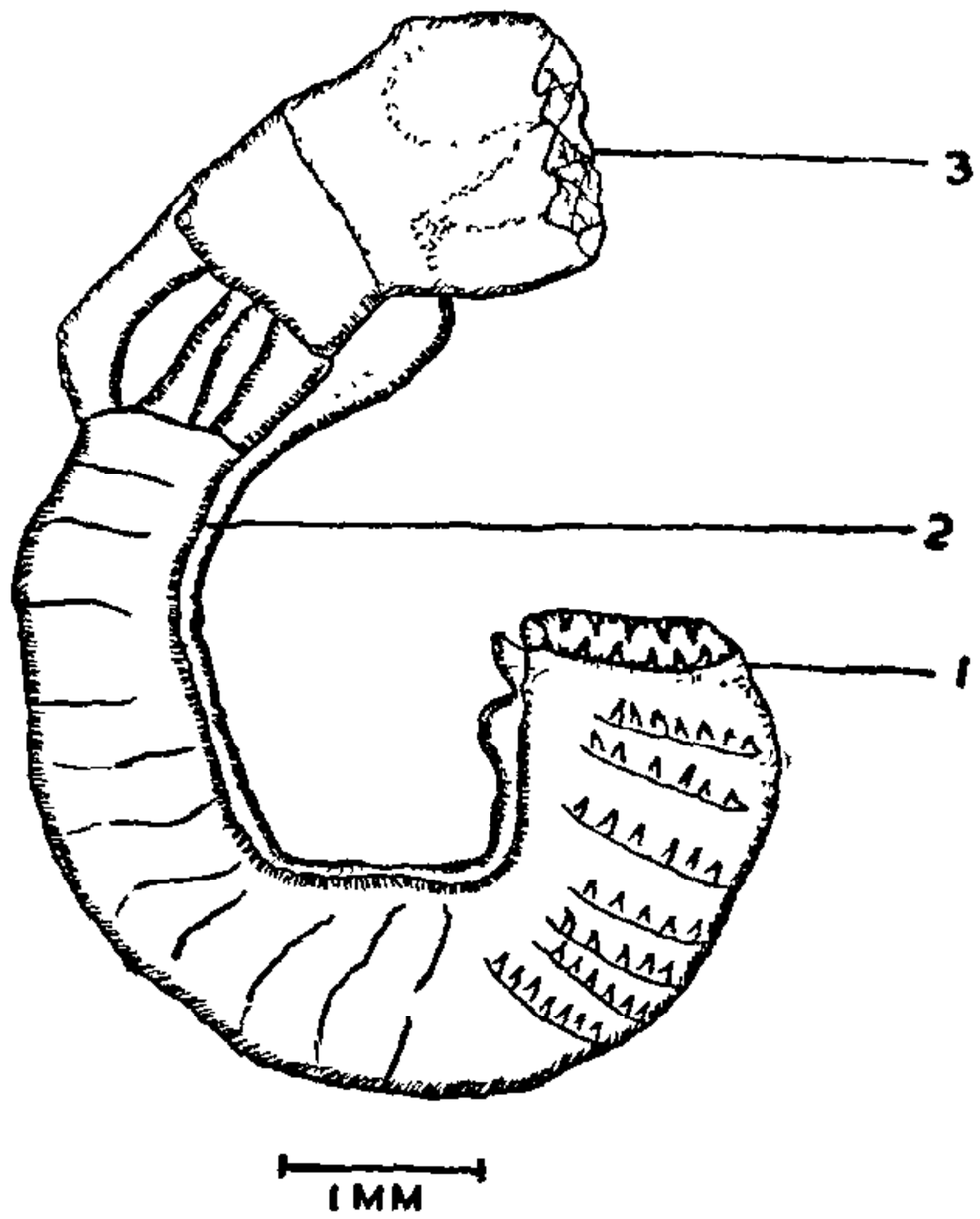

FIG. 1. 1, Introvert partially extruded showing the hooks; 2, Ventral fold; 3, Caudal region.

The present record of the occurrence of priapulids from a tropical location, further extends their traditional habit and distribution into the tropical Indian Ocean region. Thus the hitherto accepted bipolar distribution of these organisms appears to be no more valid.

One of us (GSR) is thankful to the CSIR, New Delhi, for awarding a fellowship. We are thankful to Prof. K. Hanumantha Rao for providing the necessary facilities.

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