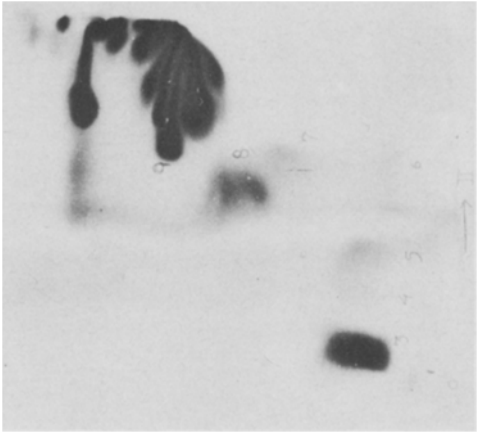
choline represent the major phospholipids of this parasite. On chromatography of these lipids in chloroform-methanol-ammonia (80:20:0.4, vol/vol) 1 sugar positive component was identified with alpha naphthol sulfuric acid. This component was identified as sulfatides with authentic markers. The presence of this lipid was further confirmed by its chromatography with the respective glycolipid.

The chemical constitution of the phospholipids has received detailed attention only in recent years. In general, it appears that the major phospholipid group known from vertebrates occur also in parasites. The phospholipids detected in *D. caninum*, namely lysolecithin, phosphatidyl choline, lysophosphatidyl ethanolamine, phosphatidyl inositol, phosphatidyl serine and phosphatidyl ethanolamine were the same as those reported in *Hymenlepis diminuta*[6] and *Taenia hydatigena*[7]. They differed, however, from those of *T. saginata*[8] and *T. taneniaeformis*[9] in that the former contained no lecithin, and the latter no phosphatidyl inositol. Ethanolamine and choline containing lipids comprise about 74% of total phospholipids similar to *Ascaris lumricoides*[10] and *Ancylostoma caninum*[11]. The identification of sulfatides in *D. caninum* is similar to its presence in nematodes and trematodes[3]. The function of these lipids in the metabolism of *D. caninum* is unknown and warrants further work.



Two-dimensional thin layer chromatogram of lipids of *Dipylidium caninum*. Chloroform-methanol-water (65:25:4, vol/vol) was used in the first direction and chloroform-methanol-acetic acid-water (50:35:8:1, vol/vol) in the 2nd direction. Components are visualized with iodine vapors: 1, lysolecithin; 3, phosphatidyl choline; 4, lysophosphatidyl ethanolamine; 5, phosphatidyl inositol; 6, phosphatidyl serine; 7, sulfatide; 8, phosphatidyl ethanolamine; 9, neutral lipids.

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Chaetogaster limnaei K von Baer 1872 on *Lymnaea tomentosa:* Ingestion of *Fasciola hepatica* cercariae

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Summary. The fresh water mollusc, *Lymnaea tomentosa,* the intermediate host of the liver fluke is heavily infested under natural conditions with ectocommensal annelid, *Chaetogaster limnaei.* These annelids which destroy the larval stages of *Fasciola hepatica* have been observed in the laboratory. The association of *C. limnaei* with the snail intermediate host is of value in exploiting a control measure against economically important liver fluke disease.

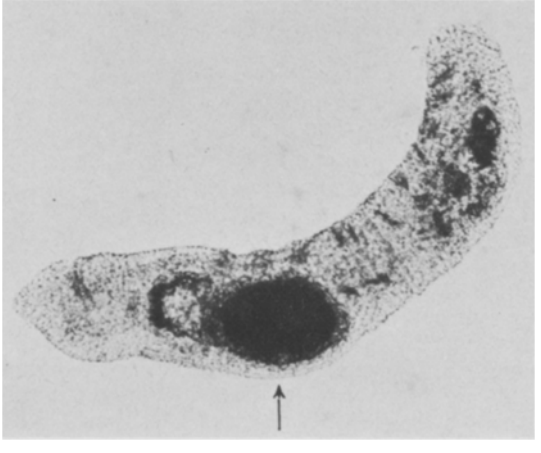
Lymnaea tomentosa is the intermediate host of the liver fluke, *Fasciola hepatica,* in Australia. Its ecology has been studied in detail[2] and many biological and chemical measures aimed at controlling populations of this snail have been suggested[3]. The former include artificially creating competition with other snails, encouraging the predatory habits of sciomycid flies and rearing ducks and other water fowl in snail localities. A competition between the larvae of echinostomes and *F. hepatica* in snails also seems to lessen the prevalence of *F. hepatica* cercariae (Boray; personal communication). Although such methods have been found to be quite successful in controlling lymnaeid snails in other parts of the world, none were found successful for the control of *L. tomentosa* in Australia.

An alternative biological control measure for fascioliasis might derive from *Chaetogaster limnaei,* an oligochaete annelid, which is found attached to the bodies of snails. This species feeds on protozoa, rotifers, nematodes, crustaceans, water mites and chironomid larvae[4]. There are also reports that they feed on miracidia[2,5,6] and cercariae[7,8]. Becklund[8] and Khalil[6] considered that these annelids play a part in limiting the distribution of trematodes. This view seems to be supported by the fact that a) Boray[2] found few or no sporocysts of *F. hepatica* in snails infested with this annelid, and b) Khalil[6] noted an inverse relationship between the prevalence of *F. hepatica* infections in snails and the number of *C. limnaei* present. *C. limnaei* may also kill snails since Bendezu[9] found many snails dying due to excessive protozoan accumulation in the intrashell space following the death of the annelid. This report provides additional observations of the feeding behaviour of *C. limnaei* on cercariae of *F. hepatica.*

Experimental observations. Adult *L. tomentosa* collected from Brooks' Creek, which flows through sheep grazing areas near Canberra, Australia, were examined for the presence of larval stages of *F. hepatica.* It was observed that all snails carried heavy infestations of *C. limnaei* (ranging from 70 to 90 per snail) on their external surfaces and shells, but they were apparently not infected with trematodes. 88 snails were each exposed in the laboratory to 20 miracidia of *F. hepatica.* During the next 2 months many snails died and most of them were devoid of any larval

stages of *F. hepatica,* and on the 68th day, only 4 snails remained alive and all released cercariae. When cercariae were emerging, 10–15% of the *C. limnaei* present on snails caught and ingested some of them (at the rate of 1/2 cercariae/chaetogaster). Most cercariae were ingested tail-end first and became lodged in the gut (figure). Encysted metacercariae were never seen inside *C. limnaei* and these annelids did not even ingest any of the encysted metacercariae which were present in the environment. Those chaetogaster which were dislodged from the body of snails, and came to lie in the immediate environment, became less active and were unable to catch cercariae, whereas those in contact with snails remained active and capable of catching and ingesting cercariae.

Discussion. In this situation one would normally expect a control group of infected snails without *C. limnaei.* Strictly speaking, such a control group is not required as it has been clearly understood that infected snails release cercariae[10]. Alternatively, it is very difficult to carry out any observation on predation by chaetogaster which are dislodged from snails because the former lose activity soon after their separation from the latter. Hence no such control observations were carried out. However, it reveals the importance of commensalistic relationship of snails and chaetogaster to maintain the predatory habits. The present observation of the devouring of *F. hepatica* cercariae by *C. limnaei* provides additional information on their feeding behaviour[4].



Chaetogaster limnaei: after ingestion of *Fasciola hepatica* cercariae (indicated by arrow).

It is common to note that snails free from *C. limnaei* successfully 'take' infection and release cercariae of *F. hepatica,* whereas snails infested with this annelid do not readily 'take' infection of *F. hepatica* in the laboratory[10]. This clearly indicates that *C. limnaei* inhibits the invasion of miracidia into snails, and devours cercariae as they emerge. Previous workers considered the association of *C. limnaei* with snails as either commensalistic[4], predacious[6], or mutualistic[2]. Nevertheless, whatever the nature of the relationship, it would seem that *C. limnaei* may control snail populations as well as limit trematode infections in snails. The latter may occur directly, by the ingestion of miracidia, and indirectly, by the ingestion of cercariae. The ingestion of cercariae is also of direct importance as far as limiting *F. hepatica* infection in the definitive hosts. Although a number of control measures are being suggested and implemented to eradicate fascioliasis, successful control has not been achieved.

Recently Samson and Wilson[11] have shown ducks to be an effective biological control agents for *F. hepatica* in USA; other biological methods need to be explored. Hence, it would seem possible that the presence of *C. limnaei* on *L. tomentosa* could be exploited as a multi-facet control measure for fascioliasis. However, much more information on the ecology and physiology of this annelid seem warranted before such a possibility could be realized.

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Collagen synthesis of cultured fibroblast from Werner's syndromes of premature aging[1]

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Summary. The difference of collagen producibility between 2 groups of skin fibroblasts from patients with Werner's syndrome with skin change and with normal skin, and the difference of collagen accumulation to cell layer between skin fibroblast from Werner's syndrome and controls were studied.

In recent years, some reports indicate that the cultured skin fibroblasts from patients with Werner's syndrome, a typical inherited premature aging disease, have shorter life span in culture[2,3] increased portion of heat-labile enzymes[4,5] and retarded rate of DNA replication[6]. It is assumed that collagen synthesis or fibre formation disorder may exist in the skin of the patients, because dermal atrophy on extremities and face is a typical clinical sign of this syndrome[2].

We now report the difference of collagen synthesis, differentiated function of skin fibroblasts from 2 cases of Werner's syndromes compared with that of normal skin firboblast and human diploid fibroblast.

The cultures were derived and propagated from the thigh skin of dermal atrophy (WF52-1), and from a trunk skin in where no obvious pathological change was detected (WF52-3) of a 52-year-old male patient, and from normal