suspensions with equal numbers of cells. Data are expressed as: mean ± SE (No. of observations).

Results. a) *Preparation of isolated cells.* Percentage viabilities varied between 30–60%, as they did for VAHOUNY et al.[2], whereas for GLICK et al.[5] they were 95% or more. Attempts to improve viability (e.g. use of plastic vessels) were without success, except that of reducing the temperature to 30 °C (Table). The important variable is the collagenase, since its dispersive activity is due to proteolytic contamination, which can vary considerably from batch to batch. 4 preparations were used: CLS 45BI94 and 45D006 (Worthington Biochemical Corp., Freehold, N. J., USA) and I 44C 0080 and II 15C 6890 (Sigma London Chemical Co., Kingston, Surrey, U.K.). The first 3 gave similar results (Sigma I being used routinely), but Sigma II was less useful. A positive correlation was obtained between percentage viability and yield of cells/g tissue (Table, at 37 °C; Harvest 3, $r = +0.58$, $p \simeq 0.02$; Harvest 4, $r = +0.60$, $p < 0.02$). This is also shown by the parallelism of viability and yield of cells for all but the earliest experiments performed with Sigma I (Figure).

b) *Incorporation into heart cells of ^{14}C from [^{14}C]glucose.* Cells were incubated for 30, 60 or 120 min. Maximum incorporation was at 60 min; at 30 and 120 min the incorporation was 49% and 61% respectively of that at 60 min. Incubating the cells with [^{14}C]glucose in 5 m*M* glucose or galactose reduced incorporation of ^{14}C (cpm) from 16740 ± 1300 (5) (0.5 m*M* glucose) to 11,100 ± 1,300 (5) and 9,180 ± 560 (6) respectively. Thus the carrier transport system for glucose was still operating in most of the cells, and was rate limiting for glucose uptake.

Discussion. GLICK et al.[5] tested 14 different commercial preparations of collagenase, all from either Sigma or Worthington. Since the yield of live cells per gram tissue varied between 4,500 and 270,000, their experiments were less successful than those given here (Table), whereas for percentage viability (95% or more) theirs were the more successful. This reciprocal difference does not reflect simply a greater proteolytic activity of the collagenase used here – greater cell dispersion with greater cell disruption – since there is a significant correlation between % viability and yield of cells, and a parallelism between them for almost all experiments (Figure).

Effect of *Schistosoma mansoni* on Plasma Cholinesterase Activity in Rhesus Monkeys

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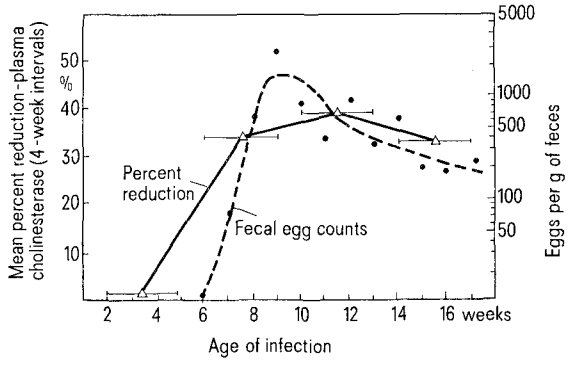
Summary. A significant reduction in plasma cholinesterase activity at a time when fecal egg counts indicated a patent infection has been found in a limited study with schistosome infected Rhesus monkeys.

In working with a group of *Schistosoma mansoni* infected Rhesus monkeys[1], a limited number of monkeys were not treated with any drugs and represented the infected ($n = 3$) and noninfected ($n = 2$) control animals. These monkeys, however, did contribute to a series of blood tests which included the determination of plasma cholinesterase (ChE) activity. This report describes the changes of plasma ChE activity during the course of early and patent schistosome infection in Rhesus monkeys.

Materials and methods. Each monkey (3–4 kg) was anesthetized with nembutal, placed in dorsal recumbancy and exposed to approximately 750 *S. mansoni* cercariae via the percutaneous route. At weekly intervals, plasma ChE activity was determined by the electrometric titration method[2]. Beginning at 5 weeks post exposure to the schistosome infection, twice weekly fecal egg counts (AMS III method[3]) were made with each monkey for the remainder of the study. An analysis of variance was conducted on the ChE data.



Comparison of fecal egg counts with reduction of plasma cholinesterase in *Schistosoma mansoni* infected Rhesus monkeys.

Results and discussion. An initial analysis of the data revealed that changes in ChE activity were more pronounced when the values were grouped at 4 week intervals. These results are graphically displayed (Figure) as the mean percentage reduction of ChE activity relative to the noninfected controls. Included in the Figure are the mean weekly fecal egg counts to illustrate patency of the schistosome infection. Schistosome eggs were first noted in the feces during the 6th week of infection. These egg counts increased in numbers to greater than 1000 eggs/g of feces during the 8th and 9th weeks followed by a gradual reduction in numbers thereafter. Coincident with the rise of schistosome egg counts was a significant reduction of plasma ChE in the infected monkeys. It would appear that reduction of enzymatic activity was initiated at a time when the schistosome infection became patent and continued throughout the remainder of the study.

An altered serum ChE is sometimes indicative of impaired hepatic function[4] and certain other abnormalities including schistosome infections of man[5]. The mode of

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action by which parasitic infection affects enzymatic activity must be speculated at this time. It is known that the liver is affected in schistosomiasis when the young parasite is migrating to the mesenteric vessels and later when egg and toxic metabolic products are passed by the mature worms[3]. In Rhesus monkeys infected with *S. mansoni*, a change of serum protein occurred 6 to 7 weeks following the initial infection and about the time that eggs appeared in the feces[6]. Associated with this change of serum protein was a fall of total body and intravascular albumin. The same authors[6] mention that similar protein changes have been observed with malaria, trypanosomiasis, trichenelliasis, trichostrongylosis and fascioliasis. In analogous studies with *S. matheei* in sheep[7,8], a more detailed account of the sequential changes of albumin degradation associated with the parasitic disease has been reported. These authors[8] state that 'the underlying cause of increased albumin degradation is due to an abnormal loss of plasma into the gastrointestinal tract'.

In the present study, it would appear that enzymatic function as measured by serum ChE was reduced at a time coincident with schistosome egg production of a patent infection. There was, however, no significant liver damage as indicated by additional clinical blood chemistry[1]. Therefore, it might be suspect that with egg passage into the gastrointestinal tract, a loss of plasma fluids would reduce the serum ChE activity faster than its replacement by the liver.

Fasciola hepatica is another trematode parasite which markedly affects the liver and its associated functions. A recent report[9] has shown that in infected rats, the ChE activity was reduced within 2 weeks of infection with metacercariae. In this case, however, tissue destruction during the time of parasite migration in the liver would be a more probable cause for the reduction of ChE activity. Egg production by mature flukes would not be found until many weeks later, and the eggs would follow a different pathway via the bile ducts as a means of entering the intestinal tract.

From the foregoing information, it is suggested that parasitic trematode infections are associated with a variety of pathological effects which include a reduction of ChE activity. This reduction of enzymatic function may be a result of tissue destruction by the parasite or the parasite's eggs. In the present study, loss of plasma protein associated with egg perforation of the intestinal wall is thought to be responsible for the reduction of plasma ChE activity in schistosome infected monkeys.

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In situ Accumulation of Marine Algal Exudate by a Polychaete Worm (*Schizobranchia insignis*)[1,2]

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Summary. For the first time, heterotrophic accumulation of dissolved carbon by a soft-bodied marine invertebrate under in situ conditions has been demonstrated. The polychaete worm *Schizobranchia insignis* Bush concentrated ^{14}C-labelled dissolved carbon (DC) exudated by the large brown alga, *Macrocystis integrifolia* Bory, 14 times over the killed controls. Our evidence suggests that algal exudate may be a significant nutritional supplement to some invertebrates cohabitating with *M. integrifolia*.

Heterotrophic utilization of dissolved organic carbon (DOC) compounds in seawater, resulting predominately from algal primary production, has been the basis of much speculation[3–8], since Pütter's hypothesis[9] was put forward near the turn of the century. Recent reviews[8,10,11] indicate that some cyclostome fishes and a wide range of marine invertebrates accumulate DOC and that this material provides a nutritional supplement to these organisms.

Exudation of significant amounts of photosynthetically produced DOC has been reported for many unstressed, actively growing algae, including both benthic macrophytes and phytoplankton[12–14]. To date, however, accumulation of DOC by freeliving invertebrates has been demonstrated solely in the laboratory using either synthetically prepared substrates or algal hydrolysates. Such conditions are not experienced in toto by the organisms in their natural environment and lead to inconclusive results. In the following, we have conducted experiments which establish, for the first time, heterotrophic accumulation in situ of algal extracellular products by a free-living marine invertebrate.

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