Dermal Lesions and Amebocytic Accumulations in Snails

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Pathology in mollusks such as *Biomphalaria glabrata* is sometimes accompanied by activation of the hemopoietic organ in the pericardial wall (Lie, Heyneman, and Yau, 1975; Richards, 1975). Hemopoietic activity has been observed in *B. glabrata* with dermal lesions and with amebocytic accumulations.

During studies on the genetic variation in susceptibility of *B. glabrata* for infection with *Schistosoma mansoni* much had been learned about the role of genetics in other infections and pathologic conditions. Two types of dermal lesions have been observed in different stocks of *B. glabrata.* Experimental studies suggest that each of these pathologic conditions involves an infectious agent and genetic susceptibility. Although lesions are remote from the heart, the hemopoietic organ in the pericardial wall is activated.

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The occurrence of proliferative amebocytic accumulations in four different clonal stocks of *B. glabrata* was reported by Richards (1975). The conditions in the four snail stocks have common features: occurrence of the amebocytic accumulations is genetically regulated; the accumulations originate as aggregations of amebocytes; amebocytic infiltration of hemolymph sinuses around stomach, intestine, albumen gland, digestive gland, and in the kidney may occur persistently; and the hemopoietic organ in the pericardial wall becomes enlarged in production of amebocytes. In vivo manifestation, supported by genetic experiments, demonstrated consistently different patterns of amebocytic accumulations in the four snail stocks. One type involves atrial nodules (Fig. 1) which first appear shortly after onset of egg laying, increase for a few weeks, then usually regress and disappear. In a second type, after maturity, accumulations appear in the pericardial cavity and develop progressively thereafter (Fig. 2). In a third type nodules appear around the aorta after maturity. In the fourth type nodules appear in juvenile snails on and between the loops of the intestine (Figs. 3, 4).

Selection and selfing through several generations in all four clonal snail stocks has resulted in amebocytic accumulations in 50-100 percent of the snails. Crossing experiments suggest that each of the four types of amebocytic accumulations is determined by a complex of genetic factors, and that each type is genetically different

Lie, et al. (1975) reported acquired resistance to infection in *B. glabrata* exposed first to irradiated echinostome miracidia and then challenged with normal echinostome miracidia. Following the challenge exposure the hemopoietic organ rapidly became much enlarged but returned to normal a few days after destruction of the parasites. In the case of the dermal lesions and amebocytic accumulations, which developed slowly but persistently, enlargement of the hemopoietic organ (Fig. 2) occurred more slowly and to a lesser extent but persisted longer. When the amebocytic accumulations are transferred to tissue culture media, the cells separate and form a monolayer of motile amebocytes. Although cell division has not been observed, some of the amebocytes survived and remained motile for several months in vitro. Since the effective life span of *B. glabrata* is about 1 year, it would appear possible that some amebocytes may survive for the life of the snail. If the primary function of these amebocytes is defense against infection or injury, then in the absence of such stress there may be little need for hemopoietic activity. When the hemopoietic organ is activated, the abruptness, magnitude, and duration of its activity probably depends on the nature of the stress to the snail.

LITERATURE CITED

Lie, K. J., D. Heyneman, and P. Yau. 1975. The origin of amebocytes in *Biomphalaria glabrata.* J. Parasitol. 61:574-576.

Richards, C. S. 1975. Genetic studies of pathologic conditions and susceptibility to infection in *Biomphalaria glabrata.* Ann. N.Y. Acad. Sci. 266:394-410.

Figure 1.—Amebocytic accumulation (arrow) in atrium (A) of *B. glabrata*; kidney (K), pericardial cavity (C); 100×, Gomori's trichrome.

Figure 2.—Amebocytic accumulations (arrow) in pericardial cavity (C) of *B. glabrata*; also showing hemopoietic organ (H) in pericardial wall (P), atrium (A), pulmonary cavity (L); 100×, hematoxylin and azure-eosin.

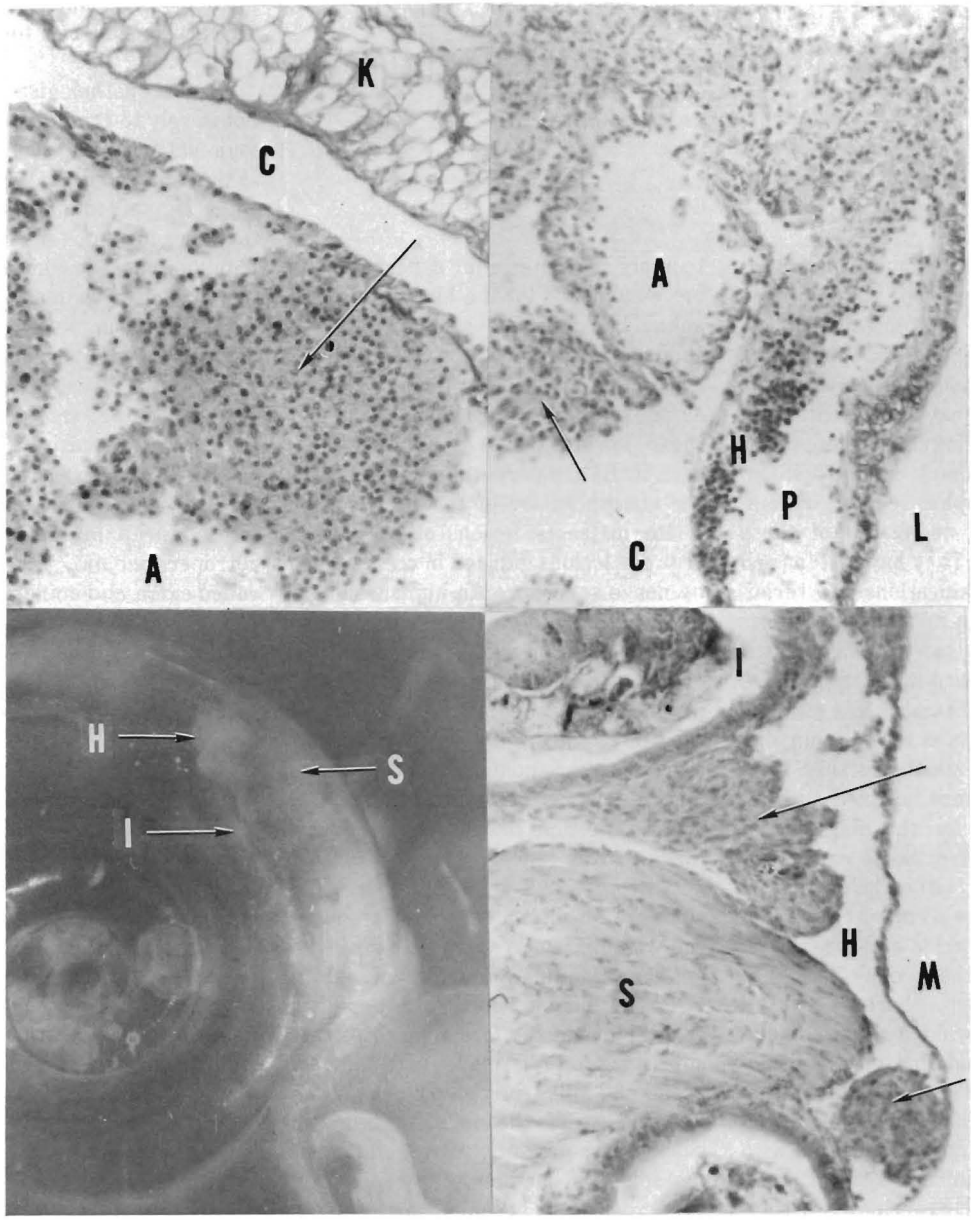

Figure 3.—Left side of live *B. glabrata* showing amebocytic accumulation in hemocele (H), stomach (S), intestine (I); 12×.

Figure 4.—Amebocytic accumulations (arrows) in hemocele (H) of *B. glabrata*; stomach muscle (S), intestine (I), mantle cavity (M); 100×, hematoxylin and azure-eosin.

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