Shell Opening by Crabs of the Genus Calappa

Abstract. *Oxystomatous crabs of the subfamily Calappinae, particularly the genus* Calappa, *possess a large tooth on the dactyl and a pair of protuberances on the propodus of the right cheliped. With these modifications and an associated behavior pattern, these crabs can efficiently open shells of gastropods and other mollusks and thus feed on the soft parts or enclosed hermit crabs.*

Decapod crustaceans of various groups break-open mollusk shells in order to feed on the soft parts. The behavior associated with opening of such shells has been reported for the Xanthidae (*1–3*), Portunidae (*1*, *4*, *5*), Ocypodidae (*6*), Palinuridae (*7*), and Paguridae (*3*, *7*). Both the Pelecypoda and the Gastropoda are preyed upon, although most of the records concern economically important pelecypods such as oysters and clams.

The methods of opening so far noted have been those of unspecialized types. Portunids, xanthids, and pagurids use their powerful chelipeds to crush the main body of the shell of the mollusk. The spiny lobster *Panulirus argus* (Latreille) probably uses its mandibles to break away the edges of gastropod shells and so open them (*7*); *Palinurus elephas* (Fabr.) reportedly "pulls" hermit crabs from their shells and eats them (*8*).

The most specialized morphological and behavioral adaptations for opening mollusk shells yet discovered are found in oxystomatous crabs of the subfamily Calappinae. These crabs, particularly those of the genus *Calappa,* have one modified cheliped, usually the right one, that enables them to crush particular portions of mollusk shells and procure the soft parts or animals enclosed. This cheliped has been used as a taxonomic character for the genera *Calappa* (*9*) and *Cycloës* (*10*). It has a set of heavy teeth: a large tooth that projects outward and downward from the dactyl, and two shorter but heavier protuberances that project outward from the propodus (Fig. 1). The teeth fit together when the dactylus of the cheliped is closed, thus breaking the portion of the shell that is bridged between them.

Tauber (*11*) noted that members of the genus *Calappa* were probably responsible for breaking the edges of various scaphopod shells, but he made no mention of the tooth or of its use. Schäfer (*3*) illustrated the right chela of *C. lophos* (Herbst) and compared its tooth with similar but less well-developed teeth of some members of the family Portunidae; he made no mention of a specific function.

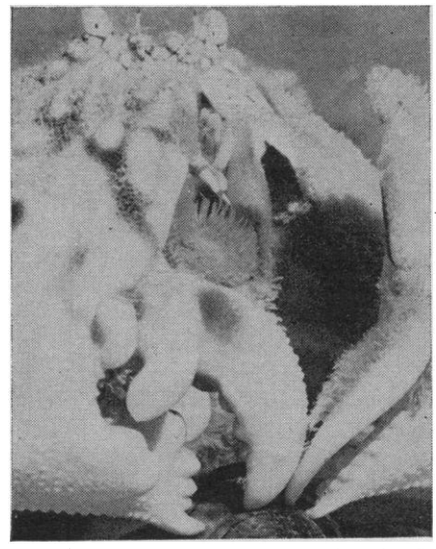

Fig. 1. The right cheliped of a female *C. flammea* (width of carapace, 91 mm), showing shell-opening mechanism. Note the large tooth on the dactyl, and how it fits between the protuberances of the propodus.

Calappa spp. were observed, in the laboratory and in the field, opening a great variety of mollusk shells: *C. hepatica* (Linn.) and *C. calappa* (Linn.) (*12*), and *C. angusta* Milne-Edwards, *C. flammea* (Herbst), *C. gallus* (Herbst), *C. ocellata* Holthuis, and *C. sulcata* Rathbun (*13*). Usually they opened gastropods or gastropod shells containing hermit crabs. The behavior pattern is relatively consistent in these seven species, although it can be modified by the crab in unusual situations. When hunting for food the crab moves about the sand, sweeping the surface with its ambulatory legs, especially the first two pairs, and pushing them into the sand up to the distal ends of the meri. The intensity of this searching increases if the crab is deprived of food or if body fluids of prey animals are disseminated. Upon finding a possible prey, the crab grasps the shell, usually by trapping it between the chalae and the body. Using the two chelae and the first two pairs of legs, the crab rolls the shell over several times, frequently inserting a dactyl into its aperture. The shell is normally held on the crab's right side, oriented with the aperture directed dorsally and supported by the first two pairs of ambulatory legs; the crab grasps the edge of the aperture with the chela and positions the shell for insertion of the large tooth of the right dactyl. The crab then inserts the tooth in the aperture, with the outside edge of the shell bridging the gap between the two heavy teeth on the propodus (Fig. 2). The dactyl is then closed on the propodus, and the bridging portion of the shell is broken. The shell is rolled toward the tooth of the dactyl by the other chela and several of the ambulatory legs, and successive pieces of shell are broken out until the enclosed animal is exposed and

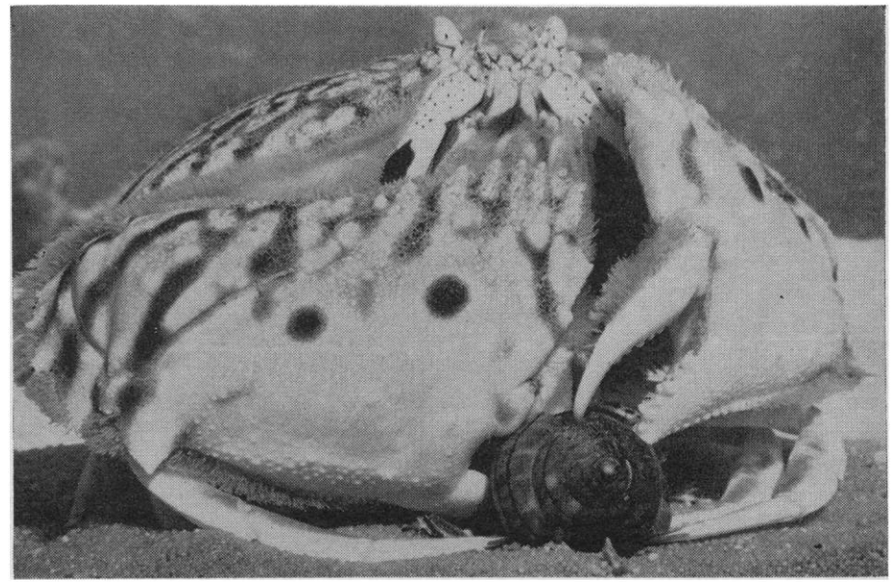

Fig. 2. Same crab as in Fig. 1 opening a shell of *Fasciolaria hunteria* containing a hermit crab (*Clibanarius vittatus*). The tooth of the dactyl is inserted into a slot in the shell that has been broken; the portion of shell bridged will be broken when the dactyl is closed on the propodus.

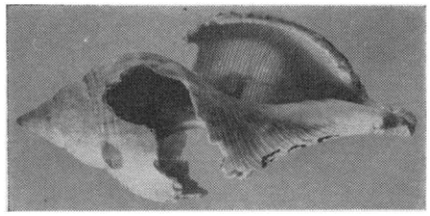

Fig. 3. Shell of *Fasciolaria tulipa* (length, 66.6 mm), containing a hermit crab (*Clibanarius vittatus*), that was opened by *C. flammea*.

removed by the chela or maxillipeds for eating.

An opened tulip shell, *Fasciolaria tulipa* Linn., appears in Fig. 3; similarly broken shells are common in the field. While opening a shell, the crab reacts immediately with quick grasps with the ambulatory legs and chelae to movements of escape by the prey.

This previously unrecognized mechanism, with the associated behavior pattern, enables these crabs to open shells that they could not otherwise open and thus expose shell-protected animals. Certainly this is the most refined shell-opening mechanism yet reported for the Crustacea.

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Agent of Disease Contracted from Green Monkeys

Abstract. *An infectious agent obtained from patients who became ill after exposure to tissues of African green monkeys is viral in character. By electron microscopy, the agent appeared cylindrical, 90 to 100 nanometers in diameter, and 130 to 2600 nanometers in length. Cross-striations at 5-nanometer intervals and a core diameter of 45 nanometers were observed. The agent was completely resistant to the effects of the metabolic inhibitor 5-bromodeoxyuridine, which may mean that RNA is the genetic material. It was sensitive to ether and relatively sensitive to destruction by heat.*

In August and September 1967, an outbreak of disease occurred in Germany and Yugoslavia among laboratory workers engaged in removal and processing of kidneys from African green monkeys for cell culture production; additional cases involved medical personnel attending the patients. At least 30 cases with seven deaths were observed. Gordon Smith *et al.* (*1*) attempted to define the etiology of this disease.

Efforts to culture bacteria or leptospires were unsuccessful; its filtration characteristics indicated that the agent was larger than most viruses. The apparent size of the organism and its failure to propagate on artificial media led to the consideration that the agent might be a rickettsia. Accumulated evidence indicates that it is a true virus. Using guinea pigs, Gordon Smith and co-workers produced complement-fixing (CF) antigens and antiserums, but these reagents did not react with any of a variety of viral reference antigens and antiserums. However, patients developed antibody to the experimental antigen.

A Porton isolate was provided by Simpson (*2*); it had been passaged nine times through guinea pigs, twice through monkeys, and once again through a guinea pig. Frozen blood from a nonfatal case ("Flak"), collected on the 6th day of illness, and liver from the first guinea-pig passage of liver from a fatal case ("Popp") were provided by May (*3*).

Each specimen was inoculated into guinea pigs (350 g, male, Hartley strain) by the intraperitoneal route. The Porton agent produced a rapidly progressing disease, resulting in death 6 to 7 days after inoculation. Clinical signs in the guinea pigs consisted of fever (to 40.3°C), inappetence, conjunctivitis, dyspnea, and progressive weakness. The pathological changes have been described (*1*). The Popp isolate killed guinea pigs on the 11th and 12th days after inoculation. The clinical signs and pathological changes were similar to those observed with the Porton specimen.

The Flak specimen initially produced a more slowly progressing disease in guinea pigs and was not uniformly fatal until the fourth passage. The lesions observed in these animals were similar to those previously described (*1*), and guinea pigs that survived the disease developed complement-fixing antibodies to an antigenic extract produced with the Porton agent.

Crude complement-fixing antigens for the Porton agent prepared from liver from infected guinea pig (20 percent suspension) in barbital-buffered saline had titers of 1 : 16 to 1 : 32, by the Laboratory Branch complement-fixation micromethod (*4*). Treatment of the antigens with 1 : 2000 β-propiolactone reduced the titer to 1 : 8, whereas heat (56°C for 30 minutes) lowered titers to 1 : 4.

Immune serum was produced in guinea pigs by intraperitoneal inoculation of liver from infected guinea pig (0.7 ml of a 20 percent suspension) (Porton) previously treated for 24 hours with 1 : 2000 β-propiolactone. Thirteen days after this injection 0.1 ml of infectious liver suspension was injected intraperitoneally. Serums were collected 12 days later. Complement-fixing antibody titers ranged from 1 : 64 to 1 : 512. These serums did not react in complement-fixation tests against antigens prepared from vesicular stomatitis (New Jersey and Indiana serotypes), Cocal, Kern Canyon, or Hart Park viruses, or against a wide variety of arboviruses.

We also attempted to establish the agent in cell cultures. The Porton agent was inoculated into a variety of cell cultures including VERO and GMK AH-1 stable lines of kidney of vervet monkey, the WI2 and CCL10 clones of BHK21, and diploid strains of fibroblasts from guinea pig heart and human foreskin. The growth and maintenance media have been described (*5*). After three passages in BHK21-CCL10 and AH-1 cells, a similar complete cyto-