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Are the Duvernoy's Gland Secretions of Colubrid Snakes Venoms?

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A toxin is a biologically produced substance that has deleterious effects on, and that may even kill, an

organism through chemical actions that can damage or destroy its tissues (Gans, 1978). A venom is a mixture of various components (among them are toxins) produced in specialized glands of an animal that when injected into another animal by spurs, stings, spines, or biting acts for feeding or defensive purposes (Mebs, 1978).

Most snakes of the family Colubridae possess Duvernoy's glands, branched, tubular, salivary glands located behind each eye. The glands discharge a serous secretion to the bases of the posterior maxillary teeth (Taub, 1967; Kochva, 1978). According to McDowell (1986), Duvernoy's glands are not venom glands because direct muscle attachment does not play an important role in the mechanical emptying of the glands in most colubrids, as is the case with the "truly venomous" viperid, atractaspidid, and elapid snakes. Venoms are recognized, however, for their functions and biological roles and not for the anatomical features of the glands where they are produced. Kardong (1980, 1982a), Kardong and Luchtel (1986), and Rochelle and Kardong (1993) also have objected to the use of the term venom for the Duvernoy's gland secretion (DGS) of colubrid snakes based on a putative functional difference between DGSs and venoms: most DGSs do not kill prey animals rapidly, as venoms supposedly do. According to Kardong (1980, 1982a), colubrids use their DGSs for prey lubrication, tooth hygiene, digestion, anti-putrefaction of ingested food, and/or detoxifying functions, rather than to capture prey. My purpose here is to emphasize that DGSs of several colubrids do aid in prey capture, and that the criterion of rapid prey death (RPD) cannot be used objectively to distinguish between venomous and nonvenomous snakes.

Venoms and Biological Roles.—The function of a phenotypic feature is its action or how it works, facilitated by its physical and chemical properties, whereas its biological role is how the organism uses that feature in the course of its life history (Bock, 1980). Kardong (1982a) maintains that to call DGSs venoms implies a biological role with a demonstrated contribution to survival. In fact, it would be difficult or impossible for the West Indian *Arrhyton exiguum* to feed on the relatively large *Anolis* lizards that constitute a major part of its diet if the DGS did not enable the snakes to immobilize the lizards prior to ingestion (Thomas and Leal, 1993). In Central America, *Leptodeira septentrionalis* frequently feeds on arboreal frogs (*Agalychnis* spp.) with adhesive toe tips that impede swallowing while the prey struggles. Only after the DGS immobilizes a tree frog can the snake ingest it (H. W. Greene and W. E. Roberts, unpubl. data). Similarly, the DGSs of the North American *Hypsiglena torquata* (Cowles, 1941; Goodman, 1953) and *Rhadinaea flavilata* (Neill, 1954; Willard, 1967), the Australasian *Fordonia leucobalia* (Shine, 1991), and the West Indian *Alsophis portoricensis* (Thomas and Prieto-Hernández, 1985; Rodríguez-Robles, 1992) allow these snakes to incapacitate their prey. The immobilizing property of these DGSs presumably allows snakes to reduce the risks of being injured during the ingestion process (Gans, 1978). Hence, DGSs contribute to the survival of some colubrid snakes.

Problems with the Concept of Rapid Prey Death.—A major difficulty with the criterion of RPD to distinguish between DGSs and venoms is the precise definition of RPD: how fast is fast enough? Would time

to death or cessation of movements after injection of DGSs or envenomation of 10, 15, 30, or 60 min all be considered rapid? Supposing that death or immobilization before, e.g., 15 min, is defined as fast, would this figure be applicable to all venomous snakes, or should different times for viperids, atractaspidids, and elapids, or even distinct ones for each snake species be defined, according to the toxicity of their venom? Another problem with this concept is that it ignores instances in which venom is used for purposes other than prey killing or immobilization. For example, helodermatid lizards use their venom for defensive purposes (Lowe et al., 1986), as often do the Asian and African cobras *Naja* spp. and *Hemachatus haemachatus* when they spray their venom through modified fangs at the eyes of predators and blind them (Bogert, 1943; Wüster and Thorpe, 1992). According to the RPD concept, none of these commonly regarded venomous animals would be considered venomous.

The following examples show the inadequacy of the concept of RPD to distinguish between venomous and nonvenomous snakes.

(i) The DGS of *Hypsiglena torquata* killed one lizard *Cophosaurus texanus* in 3 min (Jameson and Jameson, 1956), whereas that of *Rhadinaea flavilata* killed skinks *Scincella lateralis* within 15 min (Myers, 1967). Similarly, the DGSs of the arboreal African colubrids *Dispholidus typus*, *Thelotornis capensis*, and *T. kirtlandii* kill chameleons (*Bradypodion* and *Chamaeleo*), geckos (*Lygodactylus*), and fledgling birds upon which these snakes feed in ca. 15 min (FitzSimons, 1962; W. R. Branch, unpubl. data). Such RPDs imply that the three species are venomous, as Kardong (1980:277) acknowledged when he stated that "... some of the most venomous snakes are colubrids (*Dispholidus*, *Thelotornis*)..."

(ii) The only colubrid snakes (*Scolecophis* and *Tantilla* in the New World and *Aparallactus* in Africa) that primarily eat centipedes immobilize their prey with DGS and are resistant to the venom of centipedes. The 15 species of Old and New World viperids that eat centipedes do so only occasionally, and no atractaspid or elapid snake preys on these arthropods. The DGS of one captive *Tantilla nigriceps* immobilized three scolopendromorph centipedes in 6–8 min, whereas it took the venom of the North American pitviper *Crotalus lepidus* more than 1 h to do so (H. W. Greene, unpubl. data). Thus, the DGS of *Tantilla* can immobilize a centipede as fast as the venom of *Crotalus* spp. kills some rodent prey (ca. 15 min, but see below). According to the criterion of RPD, the DGS of *T. nigriceps* would be a venom and the venom of *C. lepidus* would not when this viper feeds on centipedes, but it would be a venom when *C. lepidus* preys upon rodents.

(iii) The times of death of 31 goldfish preyed upon by the Australasian estuarine colubrid *Cerberus rynchops* ranged from 6–44 min after initial seizure, with nine goldfish dying in less than 16.5 min (Jayne et al., 1987). Following the criterion of RPD, sometimes the DGS of *C. rynchops* would be a venom, but sometimes it would not.

(iv) The eel *Gymnothorax hepaticus* survived, without persistent ill effects, injections of greater venom dosages than the maximum amount extracted from the venom glands of the Australasian sea snake *Laticauda colubrina* (H. Heatwole and N. S. Poran, unpubl. data), a known predator of *Gymnothorax* eels

(Glodek and Voris, 1982). The South American colubrid *Clelia clelia* is immune to the venoms of the pitvipers of the genus *Bothrops* (sensu lato) upon which it commonly feeds (Minton and Minton, 1980). The time until death of laboratory mice (*Mus musculus*) struck by the North American pitvipers *Agkistrodon piscivorus* and *Crotalus viridis* and by the African cobra *Naja haje* can be longer than 24, 80, and 52 min, respectively (calculated from Kardong, 1982b, 1986). Although most times mice died in less than 15 min, instances in which prey took much longer to succumb would force me to consider these three species nonvenomous at times.

(v) A population of California ground squirrels (*Spermophilus beecheyi*) from an area with a high density of *Crotalus viridis* showed a considerably higher resistance to the pitviper venom than a population of the same species from an area where no *Crotalus* occur (Poran et al., 1987). The hemorrhagic activity of the venom of viperids plays a major role in the lethality of their venom (Omori et al., 1964). Several mammals (*Erinaceus europaeus*, *Herpestes edwardsii*, *Microtus ochrogaster*, *Neotoma floridana*, *N. micropus*, *Sigmodon hispidus*) showed high resistance to viperid venoms due to antihemorrhagic factors in their blood serum that neutralize the venom proteins that cause massive hemorrhage (De Wit, 1982; Weissenberg et al., 1991 and references therein). On the other hand, the toxicity of the venom of *Crotalus scutulatus* varies more than tenfold across the geographical range of this snake in the western United States and Mexico (Glenn et al., 1983). These differences in venom toxicity and in venom resistance between prey populations, together with the fact that some prey are highly immune to pitviper venoms, mean that RPD may not follow after these animals are bitten by viperid snakes, which implies that those snakes are nonvenomous to those animals.

All these observations show that the concept of RPD is not an objective criterion to differentiate venomous from nonvenomous snakes. It ignores the natural variability in the time of death after envenomation or injection of DGSs and the degree of sensitivity of a given prey to a particular venom. Animal toxins injected into any other animal for the biological roles of feeding or defense are venoms, regardless of the time to death or immobilization of the animal into which the venom was injected. Clearly, Duvernoy's gland secretions of some colubrid snakes function as venoms, although as noted by Kardong (1980, 1982a), they can play other biological roles as well. Additional studies on a variety of taxa may enable us to assess the generality of diverse biological roles of snake venoms and their historical significance (Cadle, 1982; Greene, 1989).

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