

ON THE QUESTION OF IMMUNITY OF SNAKES.

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Contents: Introduction - Two cases in captivity involving Death Adders (*Acanthophis ant-arcticus*), documented in detail - Three miscellaneous cases - Discussion and conclusions - The significance of the results of the five cases documented here and of other cases - Evolving immunity - Causes of immunity of snakes to snake venoms - Summary - References.

INTRODUCTION

Whether or not snakes are immune to their own venom is still a point of controversy despite the fact that detailed observation to do with this phenomenon and much research on the subject has been carried out by many people including Douglas, Nicholl & Peck (1933) and Kellaway (1931).

Kellaway (1931) concluded that Australian venomous snakes were not only immune to their own venoms, but also to similar types of venoms from other species of snakes. Douglas, Nicholl & Peck (1933) concluded that some highly venomous North American snakes exhibited no exceptional immunity to their own venoms within the context of injection by one snake into another similar snake if the species has highly potent venom.

Most herpetological workers conclude or assume that snakes are immune to the effects of their own venoms, including Fleay (1937, 1951), Kinghorn (1964), and Worrell (1970). My own observations tend to support this view.

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TWO CASES IN CAPTIVITY INVOLVING DEATH ADDERS
(*ACANTHOPHIS ANTARCTICUS*), DOCUMENTED IN DETAIL

The first case involved two Death Adders one red male 46.5 cm in snout-vent length (catalogued as AAA-9), and one grey female 53.5 cm in snout-vent length (catalogued as AAA-3), both originally captured in the Sydney district. Throughout this article all snakes will be referred to by their catalogue numbers when assigned one. This case occurred on 19 January 1980.

The incident occurred where the Death Adders were housed in a 180 cm long x 38 cm wide x 45 cm high 'heated' cage during feeding time.

At 2.00 pm one adult live brown mouse was introduced into the cage and shortly afterwards (within 60 seconds) it was bitten by AAA-3 just behind the head from an ambush position. AAA-9 who was in an ambush position adjacent to AAA-3 struck for the mouse a split second after AAA-3 had already bitten the mouse and bit AAA-3 on her neck, prompting AAA-3 to release the mouse immediately. The mouse rapidly moved to another part of the cage where it rested before dying in about one minute.

Meanwhile as soon as AAA-3 had released the mouse she bit AAA-9 mid-body and both snakes struggled with one another by twisting and turning themselves for roughly twenty seconds, until AAA-9 released his bite on AAA-3's neck region. This fighting scene appeared somewhat similar to that of male combat in other species (Shine, 1978).

AAA-3 maintained her bite on AAA-9 until 2.10 pm with neither snake attempting to make any major bodily movements, AAA-9 making no significant attempts to crawl away or resist AAA-3 in any manner, despite the fact that AAA-3's fangs were well embedded in AAA-9's spinal chord and that AAA-3 was almost certainly injecting massive quantities of venom into AAA-9's body. AAA-9's limited slow and apparently normal bodily move-

ments throughout this part of the incident seemed to indicate that AAA-9 was not feeling any pain or real discomfort.

After roughly ten minutes AAA-3 having her fangs well embedded into AAA-9's spinal chord, I intervened and initially tried to force AAA-3 to release her grip by prising open her jaws with the aid of a plastic rod and by hand. This was not successful due to the determination of AAA-3 to maintain her grip and her extremely deep penetration into AAA-9's body. In fact AAA-3 appeared to continue to try to pump more venom into AAA-9. Following this approximately 30 second attempt at separation, I placed and initially held AAA-3's head and AAA-9's mid-body under water in the water dish in the cage, upon which AAA-3 released her bite almost immediately. After the separation they moved away from each other in an apparently normal manner.

Both snakes had scars left from their biting each other in the form of puncture marks and damaged scales. AAA-9 had several fang puncture marks in the region where he had been bitten, indicating that AAA-3 had chewed when biting AAA-9.

As an epilogue at 2.50 pm AAA-3 ate the mouse she had bitten at 2.00 pm in an apparently normal manner. At 3.45 pm AAA-9 was seen to take then eat one adult live white mouse in an apparently normal manner, and then a second one at 4.40 pm. At no point in time during or after the incident did either snake show any ill effects, although both snakes almost certainly gave each other theoretically multiple lethal doses of venom. Two other adult male Death Adders in the cage at the same time as the incident were not involved and in fact one of them ate two live adult mice placed in the cage on 19 January 1980.

The second case involved two Death Adders in a 'temporarily heated' cage 180 cm long x 38 cm wide

x 45 cm high during a fairly typical mating attempt. It involved AAA-9 which was attempting to copulate with a grey 52.3 cm in snout-vent length female Death Adder from the Sydney district (catalogued as AAA-2). This incident occurred on 16 October 1979 at approximately 4.30 pm.

AAA-2 was simply coiled up in one corner of the cage and AAA-9 was crawling on top of AAA-2, back and forth caressing AAA-2 with his head and tail, often attempting to force his tail underneath AAA-2's body and raise her body slightly. AAA-9 was observed to open his mouth and bite AAA-2 relatively softly in various places between the dorsal mid-body and the upper neck regions. AAA-2 did not appear to respond to AAA-9 biting her, indicating no discomfort. AAA-2 was however being generally responsive to AAA-9's overtures by raising her tail, twitching it and appearing to try to force out her vulva in a characteristic manner. Whether or not AAA-9 did inject venom into AAA-2 in this particular case is questionable. In the cage at the same time as this incident although in no way directly involved were four other adult Death Adders two of which were actually copulating throughout the incident. AAA-2 at no time indicated any adverse effects to having been bitten.

All other observations of Death Adders biting themselves or one another in captivity by myself have been well documented and these Death Adders have never shown adverse effects; they have remained in perfect health.

THREE MISCELLANEOUS CASES

Case 1.

On 18 January 1980 Mr. Bill Miles found a grey adult 50 cm in snout-vent length, male Death Adder (*Acanthophis antarcticus*) on a bush road at night

in the Sydney district. When found, the snake opened its mouth wide to strike at Mr. Miles who was inspecting the snake and the snake bit itself. The snake died shortly afterwards. It was immediately preserved in formalin. It was at first concluded that the snake had died from the effects of its own bite, but on closer inspection of the specimen by the author in May 1980, it was revealed that the anterior end of the snake had been run over by a car just prior to Mr. Miles locating the snake. It therefore appeared that the likely cause of death was not from the effects of its own venom, but more probably from internal injury.

Case 2.

In early 1980 Mr. Gary Stephenson reported an adult captive highlands Copperhead (*Austrelaps superba*) biting and envenomating an adult lowlands Copperhead (*Austrelaps superba*). The lowlands Copperhead died shortly afterwards, apparently due to the effects of the bite, exhibiting according to Mr. Stephenson, typical snakebite symptoms. The highlands Copperhead was still alive and in perfect health at the time of writing this article.

Case 3.

Between 3.50 pm and 4.00 pm on 28 August 1980 I forced six Death Adders (*Acanthophis antarcticus*) from the Sydney district to envenomate one white adult pre-killed 40 g mouse. The snakes of average snout vent length of 56.8 cm (catalogue numbers AAA-2 and AAA-7) were forced one at a time to bite into the mouse's body in a similar manner to the milking of snakes. Immediately after this, the mouse having now been envenomated six times, was fed to a female 155 cm snout-vent length Diamond Python (*Morelia spilotes*) originally caught in the Sydney district (catalogued as MSS-12). She ate the mouse in a normal manner over a short period. At no time did she ever show any adverse effects

from having eaten this mouse.

Using the criterion that each Death Adder injected an average of the equivalent of 40 mg of dry venom into the mouse (an extremely conservative estimate), one may assume that MSS-12 had ingested some 240 mg of dry venom. A fraction of this amount of venom in the blood stream of this snake would presumably constitute a lethal dose.

The five cited cases here typify a much larger number of cases that have come to my notice. I have documented approximately ten cases of Death Adders (*Acanthophis antarcticus*) apparently being injected with Death Adder venom, all with similar results as those previously mentioned.

I have received numerous reports, often of a dubious nature of snakes biting themselves or being bitten by others of the same species and dying, which is similar to what happened in the first of the miscellaneous cases cited here. The second case is more typical of cases involving different subspecies or species, rather than of the same species. Over the past eighteen months I have fed Carpet Pythons (*Morelia spilotes variegata*) and Diamond Pythons (*Morelia spilotes spilotes*) food which contained venom from other species on approximately fifty occasions with the same results as in the third miscellaneous case.

The five cases documented here were stated in detail in order to highlight the circumstances in which one snake may become envenomated by another and the reactions that may result in these circumstances.

DISCUSSION AND CONCLUSIONS

Snakes may have venom from other snakes of the same or other species injected by the following means:

- a. during feeding as seen in case one. This could include cannibalism within a species or one venomous species attempting to feed on another species.
- b. during reproduction as seen in case two. This could include one snake biting another in attempting to stimulate or resist copulation, or snakes biting one another during sexual combat.
- c. through exceptional circumstances such as injury causing snakes to accidentally bite themselves as in miscellaneous case one, or as a result of unusually high snake population densities increasing the likelihood of the first two possibilities.

Snakes are only likely to ingest venom from another venomous snake if they eat food killed by other snakes, or eat other venomous snakes. All venomous snakes would ingest their own venoms. Obviously snakes are more likely to be bitten by other venomous snakes or to bite themselves more frequently within the confines of captivity than in the wild, and the occurrence of ingestion of snake venom from other snakes or from themselves is also higher within the confines of captivity than in the wild.

THE SIGNIFICANCE OF THE RESULTS OF THE FIVE CASES DOCUMENTED HERE AND OF OTHER CASES

In all cases known to me and in all published literature, there is no evidence to support the idea that Death Adders (*Acanthophis antarcticus*) are not highly immune to the effects of their own venom. These conclusions cannot be interpolated to all species of snakes due to the outcome in miscellaneous case number two, whereby one deadly venomous snake appeared to show no immunity to the venom of a geographical variation of the same species. Also



Fig. 1. *Acanthophis antarcticus*, West Head and St. Ives, Sydney District, N.S.W. Foto: R.T. Hoser.



Fig. 2. *Acanthophis antarcticus*, Glenbrook, N.S.W. "Melanistic" form. Foto: R.T. Hoser.

Douglas, Nicholl & Peck (1933), found a relative lack of immunity in certain highly venomous North American snakes to the venom of their own species. Species of snakes that engage in sexual combat would appear to be among those most likely to display immunity to the venom of their own species, as this would be virtually a necessity for their survival. As many highly venomous Australian species of snake do engage in sexual combat, it is not surprising that in general Australian snakes have a high degree of immunity to their own venom. Miscellaneous case two appears to contradict Kellaway (1931) who concluded that venomous snakes show a high degree of immunity to the venoms of snakes possessing similar venoms to their own.

Although it has been proven that snake venom in the alimentary canal of an animal is relatively harmless, it has been shown that if any venom enters the bloodstream from any part of the alimentary canal due to a cut or lesion in it, the venom's effect on the animal is the same as if it had been injected intramuscularly initially. Kellaway (1931) noted that pythons of the genus *Morelia* showed a relatively high degree of immunity to the venoms of common deadly Australian snakes. It is possible that at one time or another Diamond or Carpet Pythons (*Morelia spilotes*) held by myself received Death Adder venom in their bloodstreams indirectly via the digestive tract, although no ill effects have ever been shown by any of these snakes. This tends to support Kellaway's experimental results, and is significant as *Morelia spilotes* is a non-venomous species with no single venom type on which to base evolution of immunity to. This suggests that cases where various species of snakes have shown apparent lack of immunity to their own venoms warrants particular attention and further investigation.

EVOLVING IMMUNITY

The advantages of immunity of snakes to the venoms of other snakes particularly of the same species are obvious. Snakes would evolve immunity to their own venom and to the venoms of other snakes only if the natural selection pressures in this direction were strong enough. This could suggest that some species of snake such as the Death Adder are more likely to be injected with venom from the same species in the wild state than are other species of snake such as the Western Diamond Backed Rattlesnake (*Crotalus atrox*) which shows a lesser degree of immunity to its own venom.

This inference is significant as *Crotalus atrox* is known to engage in both sexual combat and "denning" in large numbers.

As corollary to this, it appears that snakes are only likely to become immune to venom types with which they are most likely to come into contact. The above reasoning helps to explain the apparent lack of immunity of one variety of Copperhead (*Austrelaps superba*) to the venom of another variety of Copperhead.

The hypothesis that a snake which is highly immune to the effects of one type of venom, is also highly immune to similar types of venom in many cases, as put forward by Kellaway (1931) is also supported by the preceding reasoning. It therefore appears that there is at least one component in the venom of the highlands Copperhead which is probably not present in the venom of the lowlands Copperhead to which the lowlands Copperhead is not immune.

The fact that a non-venomous species such as *Morelia spilotes* appears to have developed some immunity to the venoms of some common species of snakes indicates that in the wild this species may come into contact with common species of venomous snakes or animals killed by them, the latter ap-

pearing to be the more likely reason in the case of *Morelia spilotes*. For example an elapid may kill an animal too large for it to eat due to reasons of self defence, although the animal may still be small enough to be subsequently eaten by a larger *Morelia spilotes* specimen. Using this criterion it appears likely that other non-venomous species of snakes might have a degree of immunity to the venoms of some venomous species of snakes.

CAUSES OF IMMUNITY OF SNAKES TO SNAKE VENOMS

My experiments in feeding various live foods to Death Adders confirm the published data in that ectothermic animals have a relatively higher degree of immunity to snake venoms than endothermic animals of equivalent mass. Also my own experiments support Kellaway's 1929 conclusion that smaller animals are in general relatively more resistant to snake venoms than larger animals. Therefore a small ectothermic animal will appear to have a relatively high immunity to snake venoms even if it actually has no special immunity mechanisms.

Ectothermic animals are probably relatively more resistant to the effects of snake venoms than endothermic animals, because of their generally slower blood circulation rates. Small animals are probably relatively more resistant to the effects of snake venoms than larger animals, due to the specific selective toxic actions of venoms.

Although many animals which develop immunity to snake venoms (for example the horse) carry most anti-toxins in their bloodstream, Kellaway (1931) found that most species of animal with an 'in-born' resistance to a given snake venom contained most anti-toxins only in the tissues or organs most likely to be attacked by the given venom com-

ponents. Because of the high neurotoxic component of many Australian snake venoms including that of the Death Adder, it appears that much of the immunity of these snakes to their own venoms stems from modifications of the tissues of their peripheral nervous and muscular systems.

Due to the fact that different types of venom attack different tissues in different ways, it is logical that no species of snake can ever become completely immune to the venoms of every other species of venomous snake in existence. Support in this conclusion is given in the report of H.K. Gloyd (1933) who found that a Rattlesnake (*Crotalus* sp.) showed virtually no resistance to the bite of a Cottonmouth Moccasin (*Agkistrodon piscivorus*) and subsequently died even though the Rattlesnake could tolerate envenomation by its own species.

SUMMARY

The five cited cases, which typify a larger number of cases which have come to my notice, and most relevant literature published to date, indicates that most species of snake are immune to the venom of their own species. Miscellaneous case number two therefore provides much support for the reclassification of the lowlands Copperhead (*Austrelaps superba*) as a distinct species to the highlands Copperhead (*Austrelaps superba*).

There does, however, appear to be enough anomalies in the above general pattern to warrant further investigation into why these discrepancies occur. Some published anomalies have almost certainly arisen due to erroneous conclusions being made by observers or researchers, as nearly occurred in the first documented miscellaneous case.

Further research into the mechanisms which venomous snakes have in giving them immunity to their

own venoms could have direct beneficial medical applications as well as increasing our understanding of the biology of venomous reptiles

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POSTSCRIPT TO THE ARTICLE "ON THE QUESTION OF
IMMUNITY OF SNAKES".

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From his own experiences and those of some friends Raymond Hoser concludes that Death adders are immune to the venom of their own species. Different observations are labelled as 'doubtful' or explained on the ground of 'extraordinary circumstances'.

In *Litteratura Serpentina* (1985), Vol. 5 (5): 170-180 (Dutch ed.: 174-183) Mr. Paul Heinrich Stettler described the death of two healthy Death adders as a result of the bite of a third Death adder all three young being from the same litter. I have contacted Mr. Stettler about this and he sent me the following additional information. The mother animal was collected from the wild already pregnant. Immediately after birth the five young were sent to Switzerland where Mr. Stettler came into possession of three (all males). Several times already two snakes had bitten each other while hunting for prey, with however, no symptoms of envenomation being observed in the bitten animals.

After three and a half years and during the mating season one male died as a result of a bite from his bigger brother, who also caused the death of the third snake under the same circumstances one year later. These clear occasions prove to me that Death adders are not per definition immune from the venom of their own species.

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