

Venom metering by juvenile prairie rattlesnakes, *Crotalus v. viridis*: effects of prey size and experience

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Abstract. Despite contradictory evidence, it is widely believed that venomous snakes carefully control, or 'meter', the quantities of venom expended when feeding upon rodent prey. The major purpose of this study was to clarify experimentally whether juvenile prairie rattlesnakes inject more venom into larger mice than smaller mice. The subjects ($N=7$) were videotaped as they struck at small, medium and large mice in each of two separate trials. The quantity of venom expended was measured by enzyme-linked immunosorbent assay (ELISA) of whole-animal homogenate. In the first ('naive') trial, the snakes injected similar quantities of venom into all size classes of prey. But in the second ('experienced') trial, the snakes injected significantly more venom into larger prey. No other aspect of striking varied among prey sizes or changed in the two trials. Thus, venom expenditure was probably not a consequence of, or constrained by, some extrinsic aspect of striking, such as duration of fang contact. More likely, the rattlesnakes, with experience, attempted to inject (or meter) more venom into larger prey through intrinsic control of venom delivery. Both natural (snakebite) and artificial (syringe) envenomations indicated that mice injected with larger quantities of venom died more quickly. Because larger prey succumb less rapidly to venom, metering more venom into larger prey may be an adaptive strategy for immobilizing and killing large prey more quickly.

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As opportunistic predators, rattlesnakes consume prey of diverse body sizes (e.g. Macartney 1989; Brown 1990; Wallace & Diller 1990). They procure their food by means of an envenomating strike, during which variable quantities of venom are injected into prey through a pair of hollow fangs (Hayes 1991a, 1992a, b, 1993; Hayes et al. 1992). Adult mice and larger rodents are usually released immediately after envenomation to avoid retaliatory injury, but smaller rodents may be held (Radcliffe et al. 1980; Kardong 1986a). Envenomated rodents that are released may subsequently wander several metres or more before succumbing to the venom (Kuhn et al. 1991; Hayes 1992a). The lifeless prey must then be relocated by strike-induced chemosensory searching (SICS), which allows the snake to follow the trail of and relocate dispatched prey through use of its tongue-vomer-nasal organ system (reviewed by Chiszar et al. 1983, 1992).

The quantity of venom expended during striking may significantly influence the success of predation for several reasons. First, larger prey are less susceptible to the effects of venom (Russell 1980), and thus more likely to wander beyond recovery range after envenomation (Klauber 1972). Consequently, snakes should attempt to inject more venom into larger prey. Second, the lower surface-to-mass ratio of larger prey offers less surface area on which digestive enzymes of the snake's gut can work, and putrefaction of large prey before digestion is completed poses a serious risk for snakes (Pough & Groves 1983). Because venom has proteolytic properties that accelerate prey digestion from within (Thomas & Pough 1979; Rodriguez-Robles & Thomas 1992), rattlesnakes might obtain digestive benefits by delivering more venom into larger prey. Finally, injection of too much venom into smaller prey could be metabolically wasteful and may deplete venom reserves, leaving the snake vulnerable to predation or unable to secure additional prey encountered soon thereafter.

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It is widely held that rattlesnakes and other venomous snakes do in fact inject (or 'meter') more venom into larger prey (e.g. Klauber 1972; Russell 1980, 1984; Dunkle 1981). However, 34 years after the question was first studied experimentally (Gennaro et al. 1961), evidence supporting the notion remains weak and contradictory. For example, Gennaro et al. (1961) reported that cottonmouths, *Agkistrodon piscivorus*, inject more venom into larger prey (rats) than smaller prey (mice). In contrast, Allon & Kochva (1974) concluded that *Vipera palaestinae* delivers similar quantities of venom into rats and mice. Thus, further study is needed to clarify whether venomous snakes possess control over the quantities of venom released, and thereby allocate venom differently when biting prey of unequal sizes. Also, in view of the many ontogenetic changes associated with diet, venom composition and feeding behaviour of rattlesnakes (Hayes 1991a), it would be meaningful to learn whether venom metering is a fixed action pattern, exhibited by snakes upon first opportunity to use the strategy, or whether metering skills develop or improve as a result of prior experience.

The purposes of this study were: (1) to test experimentally the hypothesis that juvenile prairie rattlesnakes inject more venom into larger prey than smaller prey; (2) to determine whether prior experience influences the quantities of venom delivered; (3) to elucidate whether venom expenditure is influenced mostly by incidental, extrinsic factors (e.g. jaw kinematics or reactions of prey) or regulated instead by intrinsic factors under nervous system control; and (4) to determine whether injection of more venom into larger prey could be an adaptive strategy.

MATERIALS AND METHODS

Subjects

I individually maintained seven 18-month-old juvenile prairie rattlesnakes (38–45 cm snout-vent length, SVL), captive-born from gravid females collected in Carbon County, Wyoming, in plastic boxes (4 litres) with pine shavings and a glass vessel containing water. With the exception of a 5-month hibernation at 8–10°C (which ended 10 months before experimentation), the temperature was 24–26°C during a 14:10 h light:dark cycle. I fed juvenile laboratory mice, *Mus musculus*

(2–5 g), to the snakes on a weekly basis; predatory behaviour exhibited towards wild and laboratory mice is indistinguishable (Hayes 1991b). None of the present subjects had been exposed previously to an adult mouse.

I selected juvenile snakes for three reasons. First, preliminary observations indicated that juveniles initially respond to all sizes of mice in a predatory manner (i.e. they do not strike defensively at adult mice; cf. Klauber 1972; Graves 1991). Predatory and defensive strikes are easily distinguished in prairie rattlesnakes (Duvall et al. 1985; Hayes 1991b; Hayes & Duvall 1991). Second, it was thought that with experience the juveniles might either switch to striking defensively at large mice (which they did not do), or improve their success at metering venom. Changes in prey-handling behaviour as a result of experience have been implicated previously in natricine snakes (e.g. Halloy & Burghardt 1990) and in rattlesnakes (Kardong 1986a). Finally, to successfully demonstrate venom metering by snakes, it is important to rule out extrinsic factors that may influence venom delivery, such as success and duration of fang contact. Because rattlesnakes occasionally insert only one of their two fangs into prey (Kardong 1986b), it seemed that juvenile snakes, by virtue of their size, should experience greater success than adults in placing both of their fangs into prey, especially smaller prey whose width may be less than the space between fangs of large snakes.

Procedures

I observed each snake striking a small (2–5 g), medium (7–11 g) and large (25–44 g) laboratory mouse at 1-week (6–8-day) intervals in a randomly balanced sequence (i.e. in a repeated-measures design). Because snakes had no prior experience with medium or large prey, the first trial was considered the 'naive' trial. An 'experienced' trial was conducted 8 weeks later when the experiment was replicated. Thus, the study conformed to a 3×2 (prey size \times replication) repeated-measures analysis of variance (ANOVA) design. I transferred snakes individually by hook to a black Plexiglas arena measuring 30.5 \times 40 \times 44 cm in which the strike trials were conducted. The snakes were well habituated to this transfer. The arena floor was covered with light brown outdoor carpet. Both the arena and carpet were washed in

soap and water between trials. I observed and recorded all interactions by closed circuit VHS format video.

After allowing the snake 5 min of acclimation, I dropped one live prey into a corner just beyond striking range. If the snake failed to strike within 10 min, I prodded the prey to move within striking range of the snake using a snake hook; this assistance simply hastened data collection without unduly affecting snake behaviour (Hayes 1992a). All strikes were clearly predatory in motivation (according to the criteria of Hayes & Duvall 1991), with the possible exception of one strike at an adult mouse that was preceded by brief rattling; however, no other defensive behaviour patterns were exhibited by that snake. Once bitten, the envenomated prey was quickly removed by forceps from the arena to avoid receiving multiple bites, and transferred to a small plastic box (6 litres) for further observation. If the envenomated animal remained alive after 5 min, I quickly removed and sacrificed it by cervical dislocation to minimize suffering. I immediately transferred dead animals in a plastic bag to a freezer for storage (-20°C). After being returned to its home cage, I permitted the snake to ingest a small juvenile mouse that had been previously sacrificed to avoid further expenditure of venom supplies by the snake.

The use of live laboratory mice was preferable to models of mice for several reasons: (1) rattlesnakes treat models differently than they do live mice (Hayes & Hayes 1993); (2) reactions of prey may influence the mass of venom injected; (3) envenomated models provide no information on the effects of venom and, therefore, live mice are necessary to learn whether it could be adaptive to inject more venom into larger prey; and (4) the snakes were accustomed to feeding on live mice. Death from envenomation is very rapid, and is thought to be relatively painless (Russell 1980).

Strike Behaviour

Videotapes (30 fields/s) were subjected to field-by-field analyses to quantify aspects of striking. When multiple bites occurred (7% of trials), only data from the first strike were analysed. The rationale and discussion of important predatory variables are given elsewhere (Kardong 1986a; Hayes 1992a). The measures scored here included: (1) strike distance (cm from snake's snout at

launch of strike to nearest portion of prey); (2) overall duration (in s) of strike, including (a) time from launch of strike to contact of prey ('launch'), (b) time that fangs were in contact with prey ('contact') and (c) time of strike retraction ('recoil'); and lastly, (3) site of fang penetration (site 1: anterior; site 2: middle; and site 3: posterior of bitten prey). The snakes nearly always released mice immediately after envenomation (93% of trials), but two subjects were dropped from analyses of strike duration because they each held on to rather than released one of the two small mice that they bit.

Mass of Venom Expended

I measured the total quantity of venom expended (dry mass) on each prey animal to the nearest 0.5 mg by enzyme-linked immunosorbent assay (ELISA) of whole-mouse homogenates, as described elsewhere (Hayes et al. 1992, 1993; cf. Morrison et al. 1982, 1983a; Pe et al. 1991). I also calculated the mass of venom expended per bite (total venom expended divided by number of bites), but since both measures lead to identical conclusions, only total venom expended is hereafter considered.

To conduct venom assays, I obtained *in vivo* standard curve homogenates for each size class of prey. To prepare these (see Hayes et al. 1992, for more details), I extracted venom (using the 'voluntary' technique; Glenn & Straight 1982) from each snake after completion of the study. I then pooled venom samples and stored them collectively in lyophilized form. I injected six negative and positive control mice from each size class by tuberculin syringe with known quantities of reconstituted venom (0–5 mg in 0.2 ml volumes of phosphate buffered saline) in the right mid-dorsal region (cf. Hayes et al. 1992); I then prepared and stored homogenates of these control mice in the same manner as homogenates from experimental mice (Hayes et al. 1992). I assayed the venom content of each size class of prey on separate microtitre plates containing *in vivo* control homogenates of matching size class. I assayed homogenates of all experimental and control mice in triplicate.

Finally, I estimated venom content in experimental mice by regression analysis of calorimetric data, as described in Hayes et al. (1992, 1993).

Table I. Summary ($\bar{X} \pm \text{SE}$) of dependent measures associated with juvenile prairie rattlesnakes feeding on three size classes of laboratory mice in two trials

Dependent measures	Prey size		
	Small	Medium	Large
'Naive' trial			
Strike distance (cm)	2.5 \pm 0.7	4.3 \pm 0.9	3.0 \pm 0.9
Duration of strike (s)	0.36 \pm 0.05	0.33 \pm 0.02	0.30 \pm 0.03
Launch	0.07 \pm 0.02	0.07 \pm 0.02	0.07 \pm 0.01
Contact	0.14 \pm 0.03	0.12 \pm 0.02	0.09 \pm 0.02
Recoil	0.15 \pm 0.02	0.13 \pm 0.01	0.15 \pm 0.02
Anterior of animal struck (%)	43	57	43
Time to prey death (s)	216 \pm 40	167 \pm 48	286 \pm 14
'Experienced' trial			
Strike distance (cm)	3.2 \pm 1.5	4.0 \pm 1.1	4.1 \pm 0.8
Duration of strike (s)	0.32 \pm 0.05	0.28 \pm 0.02	0.35 \pm 0.03
Launch	0.06 \pm 0.02	0.08 \pm 0.02	0.09 \pm 0.01
Contact	0.14 \pm 0.03	0.10 \pm 0.01	0.10 \pm 0.02
Recoil	0.12 \pm 0.02	0.10 \pm 0.00	0.17 \pm 0.02
Anterior of animal struck (%)	29	33	100
Time to prey death (s)	282 \pm 18	177 \pm 33	246 \pm 33

For each mean, $N=7$ except that $N=5$ for measures of strike duration (see Methods). Only the main effect of prey size for time to prey death was significant (see Results). Snakes in the first trial were 'naive' to medium and large prey, but 'experienced' to them in the second trial.

Effects of Venom on Prey

Using a hand-held stopwatch I recorded the time (s) to death of bitten mice. Mice that survived more than 5 min (52%) were assigned a score of 300 s. To better clarify the relationships between prey size, venom quantity and time to death, I also observed the effects of envenomation on control mice after their syringe injections (described above). Specifically, I recorded time to death (s) with an endpoint of 10 min, at which time I sacrificed surviving animals (20%, including 0 mg controls) and assigned each a score of 600 s. With three size classes and six venom quantities, the artificial injections conformed to a 3×6 independent ANOVA design with one subject per cell (necessitating use of the interaction as the error term; Woodward et al. 1990).

Analyses

Most dependent measures met the assumptions of parametric tests, and therefore were analysed by repeated-measure ANOVAs (followed by post-hoc Scheffé contrasts) and Pearson correlation coefficients. In the case of time to death following artificial injections of control mice, however, I

rank-transformed data prior to conducting parametric ANOVA and correlation analyses (Conover 1980). I performed all tests using Statistix software (Heisey & Nimis 1985) with an alpha level of 0.05.

RESULTS

Strike Behaviour

The variables associated with striking are summarized in Table I. No interactions between prey size and replication were detected. Juvenile rattlesnakes struck at prey from a range of 0–9 cm (up to 20% of their SVL; cf. Hayes 1992a), but the mean distance did not vary when striking different sizes of prey, nor did it change in the two trials. Similarly, no other aspect of striking appeared to change between trials or vary among size classes of prey, including duration of the strike and its components and the site of envenomation.

Mass of Venom Expended

There was a significant interaction between prey size and replication for mass of venom expended

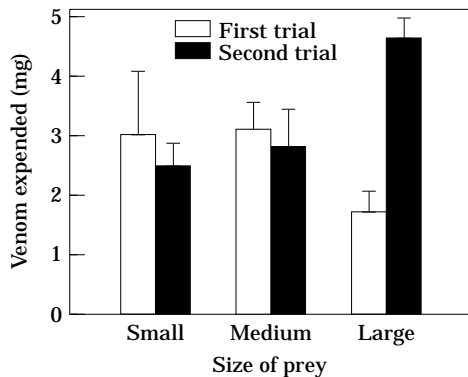


Figure 1. Mean (\pm SE) mass of venom expended by juvenile prairie rattlesnakes feeding on three size classes of laboratory mice. Snakes were considered 'naive' in the first trial and 'experienced' in the second trial. For each mean, $N=7$.

(Fig. 1; $F_{2,12}=70.2$, $P<0.0001$). Multiple comparisons indicated that rattlesnakes expended similar quantities of venom for the different prey sizes in the first strike trial ($\bar{X} \pm \text{SE} = 3.1 \pm 1.0$ mg, 3.2 ± 0.4 mg and 1.8 ± 0.3 mg for small, medium and large mice, respectively), but released significantly more venom when striking large mice in the second trial (2.6 ± 0.3 mg, 2.9 ± 0.6 mg and 4.7 ± 0.3 mg for small, medium and large mice, respectively). In fact, the mean for large mice in the second trial differed significantly from all other means. The mass of venom expended ranged from 1 to 6 mg.

Effects of Venom on Prey

The time to death of mice, presented in Table I, varied significantly between the prey size classes bitten by snakes ($F_{2,12}=6.2$, $P=0.014$). Multiple comparisons indicated that large mice survived longer than those of medium size, but survival time of small mice was similar to other size classes. The main effect of replication was not significant, nor was there any interaction between prey size and replication. No significant correlations were detected between mass of venom expended and time to death within small, medium and large prey size classes ($r = -0.12$, -0.47 and -0.50 , respectively; $N=14$ for each); however, all correlation coefficients were negative. When data were pooled for all size classes, the correlation was significant ($r = -0.35$, $N=42$, $P<0.05$), indicating that time

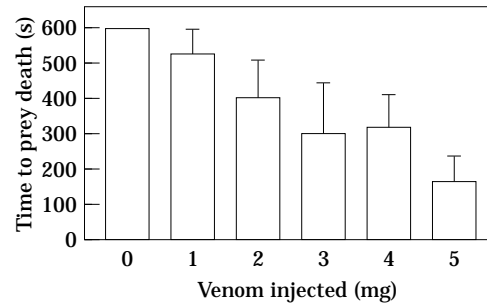


Figure 2. Mean (\pm SE) time to death (with endpoint of 600 s) of laboratory mice injected by syringe with six different quantities of prairie rattlesnake venom. Results are pooled for small, medium and large mice due to lack of significance for the main effect of prey size. For each mean, $N=3$.

to death decreased with increasing mass of venom expended. The majority of mice (52%) survived to the 5-min endpoint (at which point they were sacrificed), which may have weakened the correlations because mice injected with smaller doses could have survived longer.

Artificial injection results are summarized in Fig. 2. From analyses of variance, the main effect of prey size was not significant ($P>0.30$), but time to death varied significantly ($F_{5,10}=4.39$, $P=0.023$) and linearly ($P<0.001$) with quantity of venom injected. Consequently, there was a strong negative correlation between time to death and quantity of venom injected ($r = -0.77$, $P<0.001$). When the 0 mg control injections were excluded to make the data set comparable to natural envenomation (i.e. 1–5 mg venom injected), neither ANOVA main effect (prey size and venom quantity) was significant. However, there remained a negative correlation between time to death and quantity of venom expended ($r = -0.65$, $P=0.007$), indicating that mice injected with greater doses of venom died more quickly. The proportion of mice (excluding 0 mg controls) surviving more than 5 min (47%) was similar to natural envenomation (52%), but many also lived to the 10 min endpoint (20%). Thus, as mentioned previously for natural envenomation, a longer endpoint for time to death of mice could have yielded even stronger correlations.

DISCUSSION

The most important finding of this study was that, in the second ('experienced') trial, juvenile

prairie rattlesnakes expended more venom when striking larger prey. But the question remains: did they deliberately meter more venom into larger prey, or was it an incidental consequence of some other aspect of striking or reaction of the prey? To answer this, two alternative mechanisms regarding control of venom delivery must be considered.

First, the quantity of venom injected could be simply a consequence of some extrinsic aspect of striking. For example, venom delivery could be influenced by the duration of fang contact or by misalignment of jaws (Kardong 1986b; Rowe & Owings 1990; Hayes 1992a). The strike behaviour of snakes in this study, however, appeared similar in every respect when feeding on different sizes of prey. Furthermore, no aspects of striking changed between the two trials, while the mass of venom injected into large prey increased with experience. Moreover, it would seem unlikely that the reactions of large prey in the second trial would be different from those of the first trial. Thus, it seems improbable that any extrinsic aspect of striking or prey reaction could account for differences in venom expenditure.

The second possibility is that snakes possess an intrinsic ability to control, or meter, their venom supplies. This would imply that snakes discriminate prey size (see Radcliffe et al. 1980; Kardong 1986a) and judge the appropriate dose of venom to inject either prior to striking or during the strike itself. Such decision making by snakes can only be inferred by a demonstrated lack of extrinsic factors associated with venom expenditure, as shown here. Accordingly, the present data suggest that juvenile snakes, with experience, most likely injected more venom into larger prey through intrinsic control of venom delivery under the influence of the central nervous system.

Recent studies of anti-predator and feeding responses of neonatal snakes underscore the potential for confounding of ontogenetic effects with experimental effects (Halloy & Burghardt 1990; Burghardt 1992; Herzog et al. 1992). In the present case, however, it is unlikely that the 1.5-year-old rattlesnakes underwent an ontogenetic change in venom-metering capability during the 8-week interval between successive trials. More likely, the snakes acquired venom-metering skills through experience, possibly involving learning. Because large mice were removed after envenomation and replaced with smaller mice to be

consumed, the 'loss' (from the snake's perspective) of a large meal in the first trial may have created a learning experience, thus leading to delivery of more venom in the second trial. Alternatively, practice during the first trial simply may have led to improvement of venom metering. Regardless of how the skill is developed, metering more venom into larger prey does not appear to be a fixed action pattern in prairie rattlesnakes.

This study provides the strongest evidence to date that rattlesnakes can control and adjust the amount of venom expended during predatory strikes. Other studies that claim to demonstrate such an ability should be reconsidered for various reasons. Gennaro et al. (1961) stated that cottonmouths (of unreported size) inject more venom into large prey (rats) than into small prey (mice). It is unclear, however, from their study whether strikes at rats were predatory, and defensive strikes may differ kinematically in ways that might affect venom delivery (e.g. reduced fang contact; Hayes 1991b). Morrison et al. (1982) observed that several Australian elapids inject less venom in successive mice when presented several in quick succession, and attributed the decline to deterioration of strike coordination and efficiency. Thus, when they measured an increase in venom expenditure during successive strikes by the taipan, *Oxyuranus scutellatus*, they concluded that it was evidence of control over venom delivery. However, their claim contradicted the statistics (i.e. non-significance) presented in their results. The same investigators also compared venom expenditure by the Australian rough-scaled snake, *Tropidechis carinatus*, during predatory and defensive bites at mice and agar-filled gloves, respectively (Morrison et al. 1983b). They interpreted the differences as evidence for venom metering, but they failed to provide any measures of variance or statistical tests to support their conclusions (i.e. they reported only mean values). Elsewhere, I have shown that prairie rattlesnakes expend less venom when hungry (Hayes 1993) and more venom when feeding on birds than mice (Hayes 1992b), but in each case I reasoned that some other aspect of striking or unknown constraints on venom delivery could also explain the differences.

How might rattlesnakes benefit by injecting more venom into larger prey? The negative correlations between mass of venom expended and time to death for bitten mice corresponded well with

the artificial injection data. Together, these correlations suggest that injection of more venom can hasten immobilization and death of prey (time to immobilization, although not measured here, is highly correlated with time to death; Hayes 1992a). Compared with small prey, large prey generally survive longer and can travel further after being bitten (compare medium and large mice in Table I; see also Russell 1980). Therefore, metering more venom into larger prey may serve an adaptive function by immobilizing and killing large prey more quickly, thereby mitigating the possibility of losing a meal. Rattlesnakes observed in the wild occasionally lose envenomated animals, usually because their wounded prey travelled too far to be recovered (Fitch & Twining 1946; Hennessy & Owings 1988; Diller 1990). Furthermore, by injecting less venom into smaller prey, snakes would not unnecessarily deplete their venom supplies. Too much venom injected could be metabolically wasteful and leave the snake vulnerable to predation or unable to secure additional prey encountered soon thereafter. Thus, delivery of more venom into larger prey may represent an important but overlooked component of optimal foraging by rattlesnakes (cf. Duvall et al. 1985, 1990). It is possible that injection of more venom into larger prey also results in additional digestive benefits, but more study is needed to explore this hypothesis.

The fact that large mice survived longer after being bitten than medium mice (Table I) was expected because of their larger size and correspondingly reduced susceptibility to venom (Russell 1980). The surprisingly long survival of small mice was documented previously (Hayes 1991a), and thought to be the result of poor venom dispersion due to an underdeveloped circulatory system. The lack of a difference between medium and large mice artificially injected with venom (Fig. 2) was probably the result of the comparatively small sample size.

In contrast to this study, no comparable correlation between venom quantity and time to death was detected in a separate study for adult mice naturally bitten or artificially injected with 5–25 mg of adult rattlesnake venom (Hayes 1992a). Reasons for the discrepancy between the two studies are not immediately clear, but may be related to the different lethality of juvenile and adult venoms (Kardong 1986a; Mackessy 1988),

or to the ranges of venom quantities injected (1–5 mg of juvenile venom versus 5–25 mg of adult venom).

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