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Expulsion of Intraperitoneally-Implanted Radiotransmitters by Australian Pythons

DAVID J. PEARSON

Western Australian Department of Conservation and Land Management
PO Box 51, Wanneroo, Western Australia 6065, Australia

and

RICHARD SHINE

Biological Sciences A08, University of Sydney
New South Wales 2006, Australia
e-mail: rics@bio.usyd.edu.au

The availability of miniature radiotransmitters has revolutionized the study of snake ecology (Fitch 1987; Shine and Bonnet 2000). Most workers rely upon surgical insertion of transmitters into the animal's peritoneal cavity, and methods for the surgical implantation of transmitters are now sophisticated and effective (e.g., Reinert and Cundall 1982). As more workers use these techniques, new complications will undoubtedly appear. In this paper we report a bizarre example of such a complication. Free-ranging carpet pythons (*Morelia spilota*) frequently rid themselves of surgically-implanted transmitters by incorporating the transmitter into the alimentary tract and then expelling it with the feces.

Table 1 summarizes 14 occasions when radio-telemetered pythons appear to have expelled transmitters in this way. The transmitters (Holohil models SI-2T and PD-2T) weighed 4 to 22 g, and constituted 0.7 to 3.3% of the mass of the snake into which they were implanted. The transmitters were inserted under isoflurane anesthesia, via a midventral incision approximately 10% of the snake's snout-vent length anterior to the vent. The entire unit (transmitter plus 19–30 cm antenna) was placed inside the peritoneal cavity, with the body of the transmitter positioned anterior to the antenna. The transmitter was not sutured to a rib. The incision was sutured closed, and the snake released 1–14 days after surgery.

In the course of the study, many telemetered snakes were recaptured for transmitter replacement. In all such cases except those described below, the transmitter had remained in place within the peritoneum, and was surrounded by fibrous tissue. In the cases listed in Table 1, however, attempts to relocate snakes in the field revealed only the transmitter, usually in conjunction with fecal material from the snake. Python feces are very easily distinguished from those of other large predators because the python feces are distinctive in shape, are always associated with uric acid deposits, and detailed examination reveals the presence of python teeth.

Large carnivorous mammals (e.g., cats, dogs, foxes) are absent from our Garden Island study area and very rare (because of a prolonged control program) at the Dryandra site. Subsequent recapture of two of these free-ranging pythons that had lost their telemeters confirmed that the snake was in good health, and showed no scarring that would indicate loss of the transmitter through the body wall. Evidence of the route of transmitter expulsion was available for two additional snakes. First, an adult diamond python (*Morelia spilota spilota*; not listed in Table 1) was captured and kept in captivity after several months radio-tracking, prior to surgery to remove the transmitter. Before we could remove it, the snake defecated the transmitter in its cage. Second, we dissected a radio-tracked carpet python (*M. s. imbricata*; again not listed in Table 1) that died in the field after 22 months of radio-tracking. The transmitter in this snake was partially incorporated into the stomach, but the antenna remained attached in the peritoneal cavity. It seemed that fecal impaction might have led to the snake's death.

Data in Table 1 show that cloacal expulsion of transmitters was relatively frequent, although by no means universal (these snakes were 14 of 75 pythons radio-tracked during the study). The snakes that expelled transmitters covered a wide range of body sizes, included both sexes, and carried the transmitters prior to expulsion for periods of 1–24 months. At the Dryandra study site, 3 female and 7 male pythons expelled their transmitters from a total of 48 transmitters implanted. This involved 33 individual pythons, as some females were implanted twice. At the Garden Island study site, 1 female and 3 male pythons expelled their transmitters from a total of 52 transmitters implanted (consisting of 42 individual pythons).

To examine correlates of transmitter expulsion, we conducted a logistic regression on data from all radio-tracked pythons. Although some snakes were fitted with more than one transmitter in succession during our study, each animal appeared only once in the data set to avoid pseudoreplication. The dependent variable was whether or not the transmitter was expelled, and the independent variables were the snake's sex and snout-vent length. We excluded four cases where we had less reliable evidence for expulsion (no feces with transmitters: see Table 1) but inclusion or exclusion of these data did not change any of the conclusions from the analysis. The regression was based on 38 female snakes (3 of which expelled transmitters) and 36 males (7 of which expelled transmitters). Likelihood ratio tests from this regression showed that the probability of expulsion was linked both to the snake's sex ($\chi^2 = 6.75$, 1 df, $P < 0.01$) and its body length ($\chi^2 = 4.90$, 1 df, $P < 0.03$). Males expelled transmitters more frequently than did females (7 of 36, = 19% of males, vs 3 of 38, = 8% of females). The mean snout-vent lengths of snakes that expelled transmitters were larger than those of other snakes, in both sexes (males: 145 vs 119 cm; females, 188 vs 175 cm). There was no significant difference in rates of expulsion between the two study areas.

The most consistent correlate of expulsion involved feeding. Ten of the 14 expulsions were associated with fecal material (Table 1), suggesting that transmitters were expelled soon after the snake had consumed a large prey item. Although many of the other radio-tracked snakes took equally large meals and did not expel transmitters afterwards, we suspect that there is a functional link between feeding on a large meal, and transmitter expulsion. Not only

TABLE 1. Cases in which radio-telemetered carpet pythons (*Morelia spilota imbricata*) expelled intraperitoneally-implanted transmitters. SVL = snout-vent length (cm). The Table shows the characteristics of snakes, the dates at which transmitters were surgically implanted and were later found in the field (without the snake), and whether or not snake feces were present with the relocated transmitter. *For snake 22, the original transmitter was not found, but the snake was recaptured 25 Nov 1998 without its transmitter. Note that snake 80 was also recaptured 14 Nov 1995 and reimplanted after voiding its first transmitter.

Snake ID #	Location	Date implanted	Date recovered	Days before expulsion	Sex	SVL (cm)	Mass (g)	Transmitter found in fecal pellet?
2	Dryandra	9 Jan 1995	10 Feb 1995	32	F	172	1401	yes
3	Dryandra	5 April 1997	30 Dec 1997	269	F	196	1620	yes
10	Dryandra	31 March 1995	27 Feb 1996	333	M	147	905	yes
17	Dryandra	8 March 1995	22 Dec 1997	196	M	130	633	no
18	Dryandra	10 Dec 1995	22 Sept 1996	287	M	145	712	yes
22	Dryandra	7 Feb 1996	?	?	M	113	248	*
25	Dryandra	5 April 1996	12 Nov 1996	221	M	146	693	yes
30	Dryandra	3 Dec 1996	22 May 1998	535	F	179	2389	no
32	Dryandra	23 Feb 1997	30 Dec 1997	310	M	160	955	yes
36	Dryandra	29 Oct 1997	30 Dec 1997	62	M	152	960	yes
80	Garden Is.	1 Dec 1995	9 Feb 1996	70	M	134	744	yes*
80	Garden Is.	14 Nov 1995	29 Jan 1997	442	M	135	759	yes
83	Garden Is.	1 Dec 1995	22 Dec 1997	752	M	129	608	no
40	Garden Is.	15 April 1995	16 Feb 1996	307	F	195	2309	yes

was transmitter loss linked to feeding events (Table 1), but the sexes and sizes of snakes that most frequently expelled transmitters (large specimens, and males rather than females) are those that tend to take very large prey (relative to snake size) in our study populations (unpubl. data).

Why should a snake consuming a large meal be likely (or indeed, able) to transfer an object in its peritoneal cavity into its alimentary tract? This ability might be linked to massive temporal shifts in size and activity of the alimentary tract in ambush predators such as pythons (Secor and Diamond 1995), including carpet pythons (Bedford 1996). The gut shrinks during non-feeding periods, but is massively up-regulated soon after a prey item is ingested. It might be able to incorporate foreign objects during this rapid increase in size. This ability might function in removal of objects such as fish spines that can penetrate the stomach wall and lodge in the peritoneum (Shine 1991).

The same ability to expel intraperitoneally-implanted transmitters through the gut has been reported in fishes (Chisholm and Hubert 1985; Marty and Summerfelt 1986), and might prove to be widespread. The only analogous phenomenon in snakes (to our knowledge) involves passive integrated transponder microchips surgically implanted in the necks of corn snakes (*Elaphe guttata*); the tags frequently (and sometimes rapidly) moved posteriorly through the body and were expelled in the feces (Roark and Dorcas 2000). If such abilities are widespread, there is an obvious caveat for interpretation of radiotelemetry studies. Researchers finding a transmitter in the field would be tempted to conclude that the animal carrying that telemeter had died and/or been consumed by a predator. Marks on a transmitter left by teeth or claws of a predator species would be interpreted as evidence for such an event, but may have been inflicted after expulsion of the unit. We advocate caution in inferring predation as a cause of death of radio-tracked

snakes, unless the carcass (or part of it) is available.

Studies on fishes have attempted to determine whether changes in factors such as the size or coating of a transmitter can reduce the probability of expulsion. Smaller transmitters were less likely to be expelled (Marty and Summerfield 1986), but the transmitters used in our own study were smaller relative to snake body mass (< 4%) than is the case in most such studies. Anchoring the transmitter by suturing it to a rib during the initial surgery or by running the antenna subcutaneously (e.g., Reinert and Cundall 1982; Reinert 1992) is one obvious solution, but experimental work on fishes found that anchoring the transmitters greatly increased the rate at which they were expelled via rupturing the animal's body wall (Marty and Summerfield 1986).

Our results are interesting not only in providing a cautionary note about interpreting cases of transmitter loss, but also in revealing a hitherto-unsuspected ability of snakes to encapsulate and expel a foreign object from the peritoneum. The apparent link between expulsion and feeding (and especially to ingestion of prey that are very large relative to the predator), in a lineage of snakes known to show massive fluctuations in size and activity of the alimentary tract, strongly suggests the ability to regulate organ size in this way might have more diverse consequences than has hitherto been recognized.

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Mouthpart Pigmentation in *Rana muscosa* Tadpoles: Seasonal Changes Without Chytridiomycosis

LARA J. RACHOWICZ

Department of Integrative Biology, University of California
Berkeley, California 94720-3140, USA
e-mail: larar@socrates.berkeley.edu

Chytridiomycosis, an amphibian skin disease caused by the fungus *Batrachochytrium dendrobatidis*, has been implicated in amphibian declines in Australia, Europe, North America, and the Neotropics (Berger et al. 1998; Bosch et al. 2001; Daszak et al. 1999; Nichols et al. 2001). At least 93 amphibian species over six continents have been found to be infected with this chytridiomycete (Speare and Berger 2000). *Batrachochytrium* infections have been found in both tadpoles and adults. In postmetamorphic anurans, chytridiomycosis is associated with high mortality, but infections in tadpoles are restricted to the mouthparts and are not associated with mortality (Berger et al. 1998; Pessier et al. 1999).

Batrachochytrium grows on the keratinized epithelial cells of adult amphibians (Longcore et al. 1999) and keratinized mouthparts of some ranid and hylid tadpoles (Fellers et al. 2001; G. Fellers, pers. comm.). The superficial keratinized cells of the upper and lower jaw sheaths and tooth rows of tadpoles are typically heavily pigmented (Luckenbill 1965). Recent attention has been focused on the loss of pigmentation in mouthparts as an indicator of chytridiomycosis (Fellers et al. 2001; Vredenburg and Summers 2001). Fellers et al. (2001) found that conspicuous oral disc abnormalities including depigmented tooth rows, depigmented jaw sheaths, and swollen labial papillae (along the margins of the oral disc) are strongly associated with *Batrachochytrium* infection in the mountain yellow-legged frog, *Rana muscosa*. However, evidence presented here suggests that loss of pigmentation in tadpole mouthparts might not always be associated with chytridiomycosis

and that pigmentation might vary seasonally. This paper (1) describes mouthparts and histological results for tadpoles showing abnormalities characteristic of chytridiomycosis; (2) describes seasonal changes in mouthpart pigmentation in tadpoles from several populations; and (3) describes changes in lab-maintained animals that resemble seasonal changes.

Vredenburg and Summers (2001) identified two levels of mouthpart loss in *R. muscosa* tadpoles collected at Ebbetts Pass, Alpine County, California, USA in June 2001: (1) a lack of pigmentation in the tooth rows and only a thin pigmented line on the top and bottom jaw sheaths (Fig. b in Vredenburg and Summers 2001) and (2) a complete lack of pigmentation in the tooth rows and jaw sheaths (Fig. c in Vredenburg and Summers 2001; these figures can also be viewed in color on Amphibia Web at <http://elib.cs.berkeley.edu/aw/chytrid/index.html>). Large-scale surveys have identified more than 100 *R. muscosa* populations throughout the Sierra Nevada in California that were characterized by a loss of pigmentation in the mouthparts (R. Knapp, L. Rachowicz, V. Vredenburg, unpubl. data). Following Fellers et al. (2001), these tadpoles would be assumed to be infected with *Batrachochytrium*.

Histological evidence suggests that the amount of pigmentation in *R. muscosa* tadpole mouthparts might vary without the presence of *Batrachochytrium*. *Rana muscosa* tadpoles experience one to two winters before metamorphosis and overwinter in ice-covered lakes (Bradford 1983). Fifteen tadpoles from Ebbetts Pass, Alpine County, California, USA were inspected early in the ice-free season (2 June 2001), and all had partially or fully depigmented jaw sheaths and tooth rows (V. Vredenburg, pers. comm.). Two of these tadpoles were collected and photographed (Figs. b and c in Vredenburg and Summers 2001). A survey late in the season (6 September 2001) found that all of the tadpoles surveyed (N = 20) from this same population had fully pigmented jaw sheaths and tooth rows. The population appeared healthy during both surveys (e.g., no die-offs, no emaciated tadpoles, no behavioral changes). The two tadpoles collected from Ebbetts Pass on 2 June 2001 (Figs. b and c in Vredenburg and Summers 2001) and six tadpoles collected from this same location on 6 September 2001 were sectioned and processed routinely for histologic examination, and no *Batrachochytrium* organisms were found.

Field surveys from the 60 Lake Basin, Fresno County, California, USA, also provide evidence for a seasonal trend in mouthpart pigmentation. During the summer of 2001, five populations of *R. muscosa* in the 60 Lake Basin were examined for loss of mouthpart pigmentation on 9 June and then again on 29 July. The June surveys took place approximately one week after winter ice melted. In each survey, 20–26 tadpoles representing two tadpole age classes at each of the five sites were captured, and their jaw sheaths were examined for pigmentation with a 10x hand lens (except at two sites where fewer tadpoles were found). The percentage of tadpoles showing thin or complete loss of pigmentation in their jaw sheaths (resembling Figs. b and c in Vredenburg and Summers 2001) at each site was high in the early season survey and very low in the later season survey (Table 1). All populations seemed healthy during surveys. These populations were not tested for chytridiomycosis.

Laboratory-maintained tadpoles showed variation in mouthpart pigmentation with temperature changes. Ten *R. muscosa* tadpoles representing three age classes were collected on 5 October 2001