Anim. Behav., 1998, 55, 1255–1261

Pathogen transmission as a selective force against cannibalism

D. W. PFENNIG, S. G. HO & E. A. HOFFMAN
Department of Ecology, Ethology and Evolution, University of Illinois at Urbana-Champaign

*(Received 15 July 1997; initial acceptance 22 July 1997;
final acceptance 29 September 1997; MS. number: A7891R)*

Abstract. Cannibalism is uncommon in most species despite being taxonomically widespread. This rarity is surprising, because cannibalism can confer important nutritional and competitive advantages to the cannibal. A general, but untested, explanation for why cannibalism is rare is that cannibals may be especially likely to acquire pathogens from conspecifics, owing to greater genetic similarity among conspecifics and selection for host specificity and resistance to host immune defences among pathogens. We tested this hypothesis by contrasting the fitness consequences of intra- versus interspecific predation of diseased and non-diseased prey. We fed cannibalistic tiger salamander (*Ambystoma tigrinum*) larvae diseased conspecifics, healthy conspecifics, diseased heterospecifics (a sympatric congener, small-mouthed salamanders, *A. texanum*), or healthy heterospecifics. Cannibals that ate diseased conspecifics were significantly less likely to survive to metamorphosis and grew significantly less than those that ate diseased heterospecifics, but none of the other groups differed. Tiger salamander larvae also preferentially preyed on heterospecifics when given a choice between healthy conspecifics and heterospecifics. These results suggest that pathogen transmission is an important cost of cannibalism and provide a general explanation for why cannibalism is infrequent in most species.

Cannibalism, the ingestion of all or part of a conspecific, is taxonomically widespread (reviewed in Elgar & Crespi 1992) but uncommon in most species (Dawkins 1976). This rarity is perplexing, because cannibals can accrue important fitness benefits over and above those associated with obtaining a meal. For instance, cannibals may obtain a better balance of nutrients, necessary for growth and body maintenance, than individuals that prey on heterospecifics (Meffe & Crump 1987; Crump 1990). Cannibals can also accrue the competitive advantage of killing a close competitor.

The rarity of cannibalism suggests that it carries important fitness costs (Elgar & Crespi 1992). Two that are mentioned frequently are (1) that cannibalism may increase the cannibal's risk of injury, because cannibals and their prey may be evenly matched in fighting ability (Dawkins 1976; Sherman 1981) and (2) that cannibals may diminish the indirect component of their inclusive fitness by killing relatives (Hamilton 1964). However, both costs may be less important than is generally assumed. Many cannibals do not risk injury by preying on conspecifics, but instead attack smaller, defenceless individuals (Elgar & Crespi 1992). Moreover, the indirect inclusive fitness costs of killing a relative are often not incurred, because many cannibals preferentially avoid eating kin (reviewed in Pfennig 1997; for exceptions see Walls & Blaustein 1995 and references therein). Thus, we sought other possible costs of cannibalism that might explain why this behaviour is uncommon in nature.

A potentially important cost of cannibalism is that this behaviour may heighten the probability of acquiring deleterious parasites or pathogens above that experienced by non-cannibalistic predators. There are numerous accounts of pathogens being transmitted via cannibalism (Polis 1981; Klitzman et al. 1984; Schaub et al. 1989; Pfennig et al. 1991a), indicating that cannibalism is an effective means of pathogen transmission.

Correspondence: D. W. Pfennig, Department of Biology, Coker Hall, CB#3280, University of North Carolina, Chapel Hill, NC 27599-3280, U.S.A. (email: dpfennig@email.unc.edu). S. G. Ho is at the Center for Biotechnology, Northwestern University, 1801 Maple Avenue, Evanston, IL 60201, U.S.A. E. A. Hoffman is at the Department of Zoology, Oregon State University, Cordley Hall, Corvallis, OR 97331-2914, U.S.A.

0003–3472/98/051255+07 $25.00/0/ar970689

Moreover, although pathogens can be transmitted via interspecific predation (Lozano 1991), individuals may be more likely to acquire pathogens from conspecifics than from heterospecifics (Freeland 1983), owing to greater genetic similarity among conspecifics and selection for host specificity, and resistance to host immune defences among pathogens (Burdon & Jarosz 1988). Thus, pathogen transmission may be a general and important cost that explains the rarity of cannibalism.

We addressed this issue by experimentally contrasting the fitness consequences of intra- versus interspecific predation of diseased and non-diseased prey using facultatively cannibalistic salamander larvae and naturally occurring pathogens. We also examined whether normally cannibalistic salamanders preferentially prey on heterospecifics, when given a choice between a healthy conspecific and a healthy heterospecific.

METHODS

Study System and Experimental Subjects

We investigated disease transmission and cannibalism in natural populations of eastern tiger salamanders, *Ambystoma t. tigrinum*. The larvae of this species often exhibit one of two alternative feeding phenotypes: a 'typical' morph that feeds mostly on invertebrate prey and a larger, physically distinctive 'cannibal' morph that has specialized oral structures to facilitate ingestion of conspecifics (Lannoo & Bachmann 1984; Pedersen 1991; Reilly et al. 1992). Cannibals are induced facultatively by high densities of conspecifics (Collins & Cheek 1983).

A link between cannibalism and disease was first suggested in tiger salamanders by the significant, inverse correlation between the frequency of cannibal morphs and bacterial density in natural ponds (Pfennig et al. 1991a). In populations where cannibals occur at low frequencies, late summer bacterial blooms correlate with massive die-offs of salamanders (Worthylake & Hovingh 1989). The deaths are apparently caused by *Clostridium* bacteria (Pfennig et al. 1991a). During epidemics, most salamanders in any given pond die, but cannibal morph larvae are disproportionately represented among the dead (Pfennig et al. 1991a). Pathogenic bacteria may lethally bioaccumulate in tissues of cannibalistic larvae that feed on sublethally infected conspecifics, thereby selecting against cannibalism. In support of this, cannibalistic larvae in a laboratory setting that were fed a single diseased conspecific were more likely to die from disease than were non-cannibalistic typical-morph larvae that were simply exposed to (but did not eat) a diseased animal, or cannibals that consumed only a single healthy conspecific (Pfennig et al. 1991a). Thus, ingestion of a diseased animal, rather than being in the same tank with it, is the more effective mode of disease transmission in this system.

We collected eggs of tiger salamanders, *A. tigrinum*, and small-mouthed salamanders, *A. texanum*, from two ponds in Illinois and *A. tigrinum* eggs from one pond in Nebraska, U.S.A. These two species co-occur in the central U.S.A. (Conant & Collins 1975), implying that they experience similar selective environments (however, only *A. tigrinum* produce cannibal morphs). The Nebraska animals, which were from a location where the two species do not co-occur, were used only to crowd the older, larger Illinois larvae and cause the Illinois larvae to become cannibals for use in the experiments below. Thus, the Nebraska larvae were not used in either of the experiments described below. We separated embryos according to species. After the eggs hatched, we randomly chose groups of 16 larvae of each species and placed them into separate aquaria (38 litres) with 23 litres of dechlorinated tap water from a common source. Larvae were reared under identical conditions. During rearing, animals were fed ad libitum live brine shrimp (*Artemia*) daily. At 7 weeks after hatching, we scored *A. tigrinum* larvae as being typical or cannibal morphs using criteria in Pedersen (1991). We then separated these cannibals from their tankmates and placed them individually in separate aquaria (38 litres) with 23 litres of dechlorinated tap water from a common source.

Experiment 1: Fitness Consequences of Intra- Versus Interspecific Predation of Diseased and Non-diseased Prey

We randomly assigned 48 7-week-old cannibal-morph *A. tigrinum* larvae to one of four treatment groups (12 cannibals per treatment group), which differed in the type of prey the cannibal received: (1) diseased conspecifics, (2) healthy conspecifics, (3) diseased heterospecifics (*A. texanum*), or (4)

healthy heterospecifics. Cannibals were initially similar to each other in snout–vent length ($\bar{X} \pm$ SD initial snout–vent length=39.14 ± 3.98 mm; there were no significant differences across treatments; ANOVA: $F_{3,47}$=0.19, NS). Throughout the experiment, each cannibal was fed daily the hatch from 0.015 g of brine shrimp. To create the prey that would be fed to the experimental cannibals, we randomly assigned smaller typical-morph larvae of each species (differing from each other in snout–vent length by no more than 5%) to one of two groups: 'diseased' prey and 'healthy' prey.

To create diseased prey, we used naturally occurring pathogens present on some of the *A. tigrinum* larvae. Many animals collected from the field appear to carry bacteria that subsequently causes disease outbreaks in the laboratory similar to those observed in nature. When dissected, these diseased animals exhibit bacterial lesions identical to those present in wild-caught diseased animals. In an earlier study, Pfennig et al. (1991a) isolated numerous colonies of the pathogenic bacterium *Clostridium* from these lesions. We created diseased prey by individually placing larvae of both species in perforated 2-litre bottles and positioning these inside aquaria that had formerly contained 15 diseased *A. tigrinum* larvae in 23 litres of water. We exposed diseased prey to this disease-containing water for 2 h (larvae exposed longer invariably died of disease). We did not expose healthy prey to disease-containing water but treated them the same otherwise. We fed each focal cannibal 4–10 prey animals over the 7-week experimental interval. There was, however, no significant effect of the number of animals consumed on final growth rate (Pearson correlation for cannibals fed diseased prey: $r=-0.15$, N=24, NS; Pearson correlation for cannibals fed healthy prey: r=0.18, N=24, NS).

As response variables, we measured survival and growth rate of experimental animals over 7 weeks, by which time the focal cannibals were beginning to metamorphose. Aquaria were coded by number, and, without knowledge of which treatment group was in an aquarium, one of us observed each focal cannibal daily and measured its snout–vent length once a week using hand-held digital calipers. Growth rate was used as a response variable because (1) it is a continuous measure and (2) an important fitness consequence of disease is that infected individuals often experience diminished growth compared with uninfected individuals (Burnet & White 1972). Growth rate is particularly critical for larval amphibians, as size at metamorphosis correlates significantly with several components of fitness; for example adult survival (Pfennig et al. 1991b) and age at first reproduction (Semlitsch et al. 1988).

Experiment 2: Cannibal Preferences for Healthy Conspecific Versus Healthy Heterospecific Prey

We sought to determine whether well-fed tiger salamander larvae would engage in risk-aversive behaviour by preferentially preying on heterospecifics, when given a choice between healthy conspecific prey and healthy heterospecific prey.

We tested the discrimination abilities of larvae 7–9 weeks after they had hatched. To start an experiment, we put 23 litres of dechlorinated tap water into a 38-litre aquarium and introduced one 'test' animal (a cannibal morph) and two 'stimulus' animals (both typical-morph larvae) that were matched for size. One stimulus animal was a heterospecific (*A. texanum*) and the other was a conspecific. Stimulus larvae were always smaller than the test animal (about half the snout–vent length) and all three animals were from the same natal pond. Stimulus animals had been reared apart from test animals since they were less than 2 weeks old, so they had not been exposed to each other for 5–7 weeks. An observer watched each aquarium continuously after the animals were introduced and noted when predation had occurred (i.e. when a tank-mate was consumed) and the species identity of the surviving stimulus animal. Non-lethal attacks were also recorded. Test and stimulus animals were used only once.

To determine whether any differences reflected actual preferences by cannibals, and not differences between prey species in behaviour, we performed the following three experiments. First, we examined whether heterospecifics were easier for cannibals to catch by subjecting similarly sized typical-morph larvae of each species to a simulated attack. An observer touched the distal portion of the tail of each type of larva and assigned each animal to an ordinal score depending on its response to being tapped. In particular, we categorized the salamander's response as no response (score=0), short slow swim (score=1), long slow swim (score=2), short fast swim (score=3), long fast swim (score=4), or moved before being touched (score=5). Second, we

Table I. Two-way ANOVA showing effects of prey species identity (conspecifics versus heterospecifics) and disease state (healthy versus diseased) on cannibal growth rate

Source of variance	*df*	*MS*	*F*	*P*
Prey species identity	1	1.129	0.381	0.540
Prey disease state	1	16.685	5.636	0.022
Prey species identity × prey disease state	1	15.985	5.399	0.025
Residual	44	2.960		

examined whether heterospecifics were more 'apparent' to cannibals by noting the proportion of time each species spent swimming in the presence of a cannibal enclosed in a clear, perforated bottle. Third, we examined whether heterospecifics were more likely to approach a cannibal by recording the proportion of time each species spent swimming within 5 cm of a cannibal enclosed in a clear, perforated bottle.

We minimized the numbers of animals used in our study in light of the Association for the Study of Animal Behaviour's (1997) guidelines and Elwood's (1991, page 847) recommendation that, 'It should be a general aim in studies on aggression and predation to use experimental designs that keep numbers of animals to a minimum.' For Experiment 2, a total of 11 larvae were subjected to predation.

RESULTS

Experiment 1: Fitness Consequences of Intra- Versus Interspecific Predation of Diseased and Non-diseased Prey

Cannibals that ate diseased conspecifics were significantly less likely to survive to metamorphosis than were cannibals that ate diseased heterospecifics. Five of 12 cannibals (42%) that ate diseased conspecifics died before metamorphosis; none of the 12 cannibals that ate diseased heterospecifics died over the same interval (chi-square test: $\chi^2_1=6.32$, $P<0.02$). Prey disease state also had a significant effect on cannibal growth rate (Table I). Cannibals that ate diseased conspecifics grew significantly less than those that ate diseased heterospecifics, but none of the other groups differed (Fig. 1; Fisher's protected least significant difference multiple comparisons test: $P<0.05$).

That 'diseased' heterospecifics in our experiment were actually infected is supported by two lines of evidence. First, diseased heterospecific prey that were kept in isolation, and not fed to cannibals, later exhibited symptoms of disease and died (healthy heterospecific prey did not). Second, by day 35, cannibals that had eaten diseased heterospecifics had grown 10% less than those that had consumed healthy heterospecifics. These differences had disappeared by metamorphosis (which started at day 49), presumably because infection obtained from heterospecifics was not severe, and the cannibals had recovered by metamorphosis.

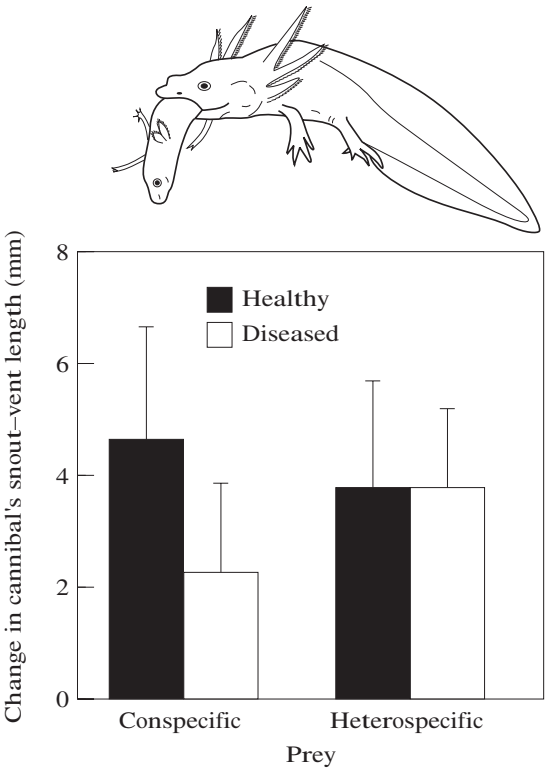

Figure 1. Effect of prey type on growth rate ($\bar{X} \pm$ SD snout–vent length) of cannibalistic salamanders.

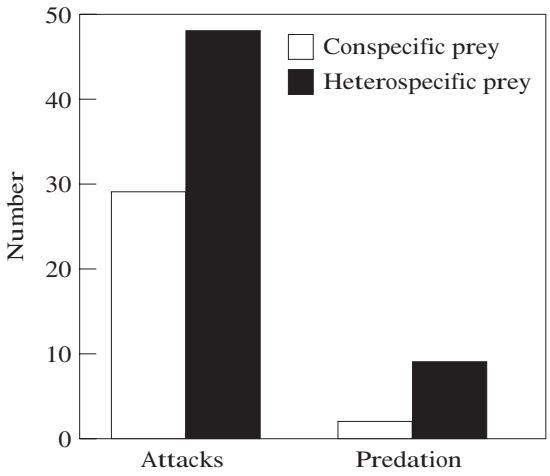

Figure 2. Results of prey choice experiments in which 11 focal cannibal morph *A. tigrinum* larvae were each given a choice of preying on a conspecific or a heterospecific larva (*A. texanum*).

Experiment 2: Cannibal Preferences for Healthy Conspecific Versus Healthy Heterospecific Prey

Cannibals were significantly more likely to attack (chi-square test: $\chi^2_1=7.52$, $P<0.01$) and consume ($\chi^2_1=5.44$, $P<0.02$) heterospecific than similarly sized conspecific larvae (Fig. 2). These differences reflected actual preferences by cannibals for heterospecifics and not differences in the behaviour of the two prey species. In particular, heterospecifics were not easier for cannibals to catch: when subjected to a simulated attack, the response of *A. texanum* larvae ($\bar{X}\pm$ SD response score$=2.29\pm1.49$) did not differ significantly from that of similarly sized *A. tigrinum* larvae ($\bar{X}\pm$ SD response score$=2.86\pm1.51$; two-tailed Mann–Whitney *U*-test: $U=78$, $N_1=N_2=14$, NS). It was also unlikely that heterospecifics were more 'apparent' to cannibals: proportion of time spent swimming in the presence of a cannibal by *A. texanum* larvae ($\bar{X}\pm$ SD$=0.36\pm0.18$, $N=6$) did not differ significantly from that of similarly sized *A. tigrinum* larvae ($\bar{X}\pm$ SD$=0.42\pm0.07$, $N=6$; two-tailed paired *t*-test: $t_5=0.84$, NS). Finally, heterospecifics were not more likely to approach a cannibal: proportion of time within 5 cm of a cannibal enclosed in a clear perforated bottle by *A. texanum* larvae ($\bar{X}\pm$ SD$=0.37\pm0.3$, $N=13$) did not differ significantly from that of similarly sized *A. tigrinum* larvae ($\bar{X}\pm$ SD$=0.35\pm0.38$, $N=13$; two-tailed paired *t*-test: $t_{12}=-0.25$, NS).

DISCUSSION

Our results help clarify why cannibalism is infrequent in most species. Cannibalistic tiger salamander larvae that ate diseased conspecifics were significantly less likely to survive to metamorphosis and were significantly smaller at metamorphosis than were cannibals that ate diseased heterospecifics, but none of the other groups differed (Table I, Fig. 1). Thus, we suggest that cannibalism is more dangerous than carnivory, because individuals are more likely to acquire pathogens from conspecifics than from heterospecifics.

Although we did not determine the precise reason(s) why pathogens are more dangerous to cannibals than to non-cannibalistic carnivores, our results support the hypothesis that individuals may be more likely to acquire pathogens from conspecifics than from heterospecifics because of greater genetic similarity among conspecifics, and selection for host specificity and resistance to host immune defences among pathogens. In support of this prediction, some pathogens exhibit such a high degree of host specificity that they only infect hosts with a single genotype (Burdon & Jarosz 1988). Moreover, there is mounting evidence that contagious, debilitating pathogens may be more highly transmissible among genetically similar conspecifics (kin) than among less genetically similar conspecifics (non-kin; Shykoff & Schmid-Hempel 1991; Black 1994). Again, this may be because individuals that have diverged from one another more recently (e.g. conspecifics and kin as compared with heterospecifics and non-kin) have more similar immune systems and are thus afflicted by similar varieties of pathogens.

As an alternative explanation, cannibals may have died at a higher rate after eating diseased conspecifics than after eating diseased heterospecifics because of the lower nutritional value of diseased conspecifics. However, three lines of evidence argue against this possibility. First, the conspecifics and heterospecifics that were used as prey in our experiments were similar in size and reared under similar conditions. Second, it is not clear why diseased conspecifics would be less nutritious as prey than diseased heterospecifics. If anything, conspecific prey may actually be more nutritious than heterospecific prey (e.g. Meffe & Crump 1987; Crump 1990). Third, autopsies revealed that the dead cannibals had bacterial

lesions on their internal organs similar to those found on diseased animals from the field. Thus, it seems most likely that the cannibals died at a higher rate after eating diseased conspecifics than after eating diseased heterospecifics because of enhanced pathogen transmission between conspecifics.

If cannibalism is potentially costly, then why do some species, such as tiger salamanders, evolve specialized cannibal morphs (Polis 1981)? Cannibal morphs may be favoured when the benefits of cannibalism outweigh the costs. One such benefit is enhanced growth (Crump 1990). As predicted, cannibal morphs appear most frequently in species and life stages (such as insect and amphibian larvae) that occur in rapidly deteriorating environments, where the benefits of such rapid growth brought on by cannibalism may exceed the risks associated with disease (Polis 1981; Pfennig et al. 1991a; Crump 1992; Elgar & Crespi 1992). After all, failure to escape a drying pond is always deadly, but disease can sometimes be avoided or overcome.

For most animals, however, cannibalism is uncommon (Dawkins 1976). The relative infrequency of cannibalism has been attributed to many factors, including the risk of injury or retaliation (Dawkins 1976; Sherman 1981) and the indirect inclusive fitness costs of killing a relative (Hamilton 1964). These costs, however, cannot explain cases where food-limited animals kill conspecifics but do not consume them. For instance, lactating female Belding's ground squirrels, *Spermophilus beldingi*, kill infants of nearby mothers but rarely ingest their victims (Sherman 1981), the larvae of two species of mosquitoes (*Toxorhynchites*) attempt to kill, but do not eat, all other larvae in their tree hole or bamboo hollow (Hamilton 1970), and in some species of birds, nestlings kill siblings but do not eat them (Stanback & Koenig 1992). Presumably, these animals forgo consuming a freshly killed prey item, even though there would be no risk of retaliation, or of diminishing the indirect component of the cannibal's inclusive fitness (because the victim is already dead), because of enhanced risk of acquiring deleterious pathogens.

The notion that cannibalism may be generally infrequent because it increases the cannibal's risk of disease has not been studied in detail (Elgar & Crespi 1992), despite mounting evidence that selection to avoid pathogens may influence many behaviour patterns (Hamilton 1987; Sherman et al. 1988; Hamilton et al. 1990; Hart 1990; Lozano 1991; Shykoff & Schmid-Hempel 1991; Andersson 1994; Loehle 1995). Previous studies have pointed to pathogen transmission as a potential cost of cannibalism (Klitzman et al. 1984; Schaub et al. 1989; Pfennig et al. 1991a). None of these studies, however, had compared the fitness costs of cannibalism with the alternative, interspecific predation. Our investigation, the first to contrast directly the fitness consequences of intra- versus interspecific predation, implicates enhanced pathogen transmission as an important and general cost of cannibalism and helps explain why this behaviour is relatively rare in nature.

ACKNOWLEDGMENTS

We thank Rex Cocroft, Karin Pfennig, Kern Reeve and Paul Sherman for helpful discussion, Mark Elgar, Karin Pfennig, Ronald Rutowski and an anonymous referee for comments on the manuscript, and Mikel Pulins, Sameer Naseerdon and Matt Reetz for laboratory assistance. This work was supported by a grant from the Arnold O. Beckman Research Fund and by NSF grant IBN-9512110 to D.W.P.

REFERENCES

Andersson, M. 1994. *Sexual Selection*. Princeton, New Jersey: Princeton University Press.

Association for the Study of Animal Behaviour 1997. Guidelines for the treatment of animals in behavioural research and teaching. *Anim. Behav.*, 53, 229–234.

Black, F. L. 1994. An explanation of high death rates among New World peoples when in contact with Old World diseases. *Perspect. Biol. Med.*, **37**, 292–307.

Burdon, J. J. & Jarosz, A. M. 1988. The ecological genetics of plant-pathogen interactions in natural communities. *Phil. Trans. R. Soc. Ser. B*, **321**, 349–363.

Burnet, M. & White, D. O. 1972. *Natural History of Infectious Disease*. 4th edn. Cambridge: Cambridge University Press.

Collins, J. P. & Cheek, J. E. 1983. Effect of food and density on development of typical and cannibalistic salamander larvae in *Ambystoma tigrinum nebulosum*. *Am. Zool.*, 23, 77–84.

Conant, R. & Collins, J. T. 1975. *Reptiles and Amphibians of Eastern and Central North America*. Boston: Houghton Mifflin.

Crump, M. L. 1990. Possible enhancement of growth in tadpoles through cannibalism. *Copeia*, **1990**, 560–564.

Crump, M. L. 1992. In: *Cannibalism. Ecology and Evolution among Diverse Taxa* (Ed. by M. A. Elgar & B. J. Crespi), pp. 256–276. Oxford: Oxford University Press.

Dawkins, R. 1976. *The Selfish Gene*. Oxford: Oxford University Press.

Elgar, M. A. & Crespi, B. J. 1992. *Cannibalism. Ecology and Evolution among Diverse Taxa*. Oxford: Oxford University Press.

Elwood, R. W. 1991. Implications of studies on infanticide and maternal aggression in rodents. *Anim. Behav.*, **42**, 841–849.

Freeland, W. J. 1983. Parasites and the coexistence of animal host species. *Am. Nat.*, **121**, 223–236.

Hamilton, W. D. 1964. The genetical evolution of social behaviour. I. II. *J. theor. Biol.*, **7**, 1–52.

Hamilton, W. D. 1970. Selfish and spiteful behaviour in an evolutionary model. *Nature, Lond.*, 228, 1218–1220.

Hamilton, W. D. 1987. Kinship, recognition, disease and intelligence: constraints on social evolution. In: *Animal Societies: Theories and Facts* (Ed. by Y. Itô, J. L. Brown & J. Kikkawa), pp. 81–102. Tokyo: Japan Scientific Society Press.

Hamilton, W. D., Axelrod, R. & Tanese, R. 1990. Sexual reproduction as an adaptation to resist parasites (a review). *Proc. natn. Acad. Sci. U.S.A.*, 87, 3566–3573.

Hart, B. J. 1990. Behavioral adaptations to pathogens and parasites: five strategies. *Neurosci. Biobehav. Rev.*, **14**, 273–294.

Klitzman, R. L., Alpers, M. P. & Gajdusek, D. C. 1984. The natural incubation period of kuru and the episodes of transmission in three clusters of patients. *Neuroepidemiology*, **3**, 3–20.

Lannoo, M. J. & Bachmann, M. D. 1984. Aspects of cannibalistic morphs in a population of *Ambystoma t. tigrinum* larvae. *Am. Midl. Nat.*, **112**, 103–109.

Loehle, C. 1995. Social barriers to pathogen transmission in wild animal populations. *Ecology*, **76**, 326–335.

Lozano, G. A. 1991. Optimal foraging theory: a possible role for parasites. *Oikos*, **60**, 391–395.

Meffe, G. K. & Crump, M. L. 1987. Possible growth and reproductive benefits of cannibalism in the mosquitofish. *Am. Nat.*, **129**, 203–212.

Pedersen, S. C. 1991. Dental morphology of the cannibal morph in the tiger salamander, *Ambystoma tigrinum*. *Amphib.-Reptil.*, **12**, 1–14.

Pfennig, D. W. 1997. Kinship and cannibalism. *BioScience*, **47**, 667–675.

Pfennig, D. W., Loeb, M. L. G. & Collins, J. P. 1991a. Pathogens as a factor limiting the spread of cannibalism in tiger salamanders. *Oecologia (Berl.)*, 88, 161–166.

Pfennig, D. W., Mabry, A. & Orange, D. 1991b. Environmental causes of correlations between age and size at metamorphosis in *Scaphiopus multiplicatus*. *Ecology*, 72, 2240–2248.

Polis, G. A. 1981. The evolution and dynamics of intraspecific predation. *A. Rev. Ecol. Syst.*, **12**, 225–251.

Reilly, S. M., Lauder, G. V. & Collins, J. P. 1992. Performance consequences of a trophic polymorphism: feeding behavior in typical and cannibal phenotypes of *Ambystoma tigrinum*. *Copeia*, **1992**, 672–679.

Schaub, G. A., Böker, C. A., Jensen, C. & Reduth, D. 1989. Cannibalism and coprophagy are modes of transmission of *Blastocrithidia triatomae* (Trypanosomatidae) between triatomines. *J. Protozool.*, **36**, 171–175.

Semlitsch, R. D., Scott, D. E. & Pechmann, J. H. K. 1988. Time and size at metamorphosis related to adult fitness in *Ambystoma talpoideum*. *Ecology*, **69**, 184–192.

Sherman, P. W. 1981. Reproductive competition and infanticide in Belding's ground squirrels and other organisms. In: *Natural Selection and Social Behavior* (Ed. by R. D. Alexander & D. W. Tinkle), pp. 311–331. New York: Chiron Press.

Sherman, P. W., Seeley, T. D. & Reeve, H. K. 1988. Parasites, pathogens, and polyandry in social Hymenoptera. *Am. Nat.*, **131**, 602–610.

Shykoff, J. A. & Schmid-Hempel, P. S. 1991. Genetic relatedness and eusociality: parasite-mediated selection on the genetic composition of groups. *Behav. Ecol. Sociobiol.*, 28, 371–376.

Stanback, M. T. & Koenig, W. D. 1992. Cannibalism in birds. In: *Cannibalism. Ecology and Evolution among Diverse Taxa* (Ed. by M. A. Elgar & B. J. Crespi), pp. 277–298. Oxford: Oxford University Press.

Walls, S. C. & Blaustein, A. R. 1995. Larval marbled salamanders, *Ambystoma opacum*, eat their kin. *Anim. Behav.*, **50**, 537–545.

Worthylake, K. M. & Hovingh, P. 1989. Mass mortality of salamanders (*Ambystoma tigrinum*) by bacteria (*Acinetobacter*) in an oligotrophic seepage mountain lake. *Gt Basin Nat.*, **49**, 364–372.