BIOLOGIE DES POPULATIONS DES MONOGENES POLYSTOMATIDAE

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RÉSUMÉ

Les cycles des monogènes polystomatidae montrent une très grande diversité. Parmi ceux qui infestent des amphibiens anoures, on devrait s'attendre à ce que la taille des populations parasites montre des différences prononcées, selon que des réinfestations interviennent régulièrement chaque année, ou qu'il n'y en ait qu'une dans la vie de l'hôte.

Toutefois, à quelques exceptions près, les niveaux d'infestation sont généralement bas, quelle que soit la durée de vie de l'hôte. Les facteurs susceptibles de réguler les populations de polystomatidae parasites d'amphibiens anoures sont récapitulés ici, et nous nous penchons plus particulièrement sur les mécanismes contrôlant l'infestation et, par voie de conséquence, la survie post-infestation. Les effets d'un éventail de facteurs sont envisagés, parmi lesquels les contraintes environnementales externes (en particulier, la température), les facteurs liés à l'hôte (dont le comportement et la durée de vie) et les facteurs propres au parasite (dont la compétition intraspécifique).

Deux genres de Polystomatidae témoignent d'une régulation densité-dépendante des infrapopulations unique, contrôlée par la production de deux types de larves. Il existe des données de terrain et de laboratoire qui permettent de quantifier les effets de ces différents paramètres pour un certain nombre d'espèces de Polystomes. Les résultats obtenus pour *Pseudodiplorchis americanus* suggèrent que, même lorsqu'ils sont combinés, les effets de ces différents facteurs ne suffisent pas à rendre compte de la puissante régulation que l'on observe dans les populations naturelles où, malgré de massives infestations annuelles, les populations de parasites adultes sont faibles en effectif et remarquablement stables d'une année à l'autre. C'est la preuve indirecte qu'une importante régulation intervient par l'intermédiaire de l'immunité-hôte.

Pour pouvoir pousser plus loin l'interprétation de la dynamique des populations parasites, il est donc maintenant nécessaire d'intégrer les composantes écologiques et immunologiques.

THE POPULATION BIOLOGY OF POLYSTOMATID MONOGENEANS

ABSTRACT

The life cycle patterns of polystomatid monogeneans show considerable diversity. Amongst representatives infecting anuran amphibians, parasite population size might be expected to show marked differences in relation to whether transmission occurs continuously, at annual intervals, or only once in the host's lifetime. However, with few exceptions, infection levels are more or less consistently low regardless of life cycle pattern. Factors which regulate the populations of polystomatids in anuran amphibians are reviewed, focusing on controls affecting parasite invasion and subsequent post-infection survival. Evidence for a range of constraints is considered, including external environmental factors (especially temperature), host factors (including behaviour and survivorship) and parasite factors (including intra-specific competition).

Two genera of polystomatids also demonstrate a unique density-dependent regulation of infra-populations controlled by production of dimorphic larvae. There are field and laboratory data which quantify the effects of these constraints for a number of polystomatid species. Specific evidence for *Pseudodiplorchis americanus* suggests that, even in combination, these factors are not sufficient to account for the powerful regulation seen in natural populations where, despite massive larval invasion occurring annually, populations of adult parasites are low and remarkably constant from year to year. There is circumstantial evidence that a major regulation is mediated by host immunity. Further interpretation of parasite population dynamics now requires integration of ecological and immunological research.

INTRODUCTION

Observations have been accumulating for over 120 years that the Polystomatidae exhibit a series of remarkable characteristics, many of which are without precedent in Parasitology. In addition to the unique life cycle of *Polystoma*, three other examples illustrate this distinctive evolution. *Eupolystoma alluaudi* produces two types of larvae, one designed to re-infect the host when worm burdens are low, the other released from heavily-infected hosts to transmit to others. This density-dependent regulation of the parasite population appears to be programmed into larval development (FOURNIER & COMBES, 1979). In *Pseudodiplorchis americanus*, the nutrition of encapsulated embryos in *utero* is achieved by a mechanism unrecorded elsewhere in platyhelminths : nutrients from the parent worm are channelled directly to the offspring via cytoplasmic connections which have a placenta-like function (CABLE & TINSLEY, 1991a). Studies on *Polystoma, Protopolystoma, Polystomoides, Eupolystoma, Diplorchis, Pseudodiplorchis* and *Neodiplorchis* show that polystomatid oncomiracidia have eyespots which form multilayer quarter-wavelength reflectors, an adaptation so far seen in no other platyhelminths but which has evolved independently in some representatives of four other invertebrate phyla (rotifers, archiannelids, lamellibranch molluscs, and decapod crustaceans) (FOURNIER, 1980; ZHANG, 1987; CABLE & TINSLEY, 1991b). Polystomatids also provide models for elucidating fundamental principles : for instance, studies on *Polystoma, Protopolystoma* and *Pseudodiplorchis* have demonstrated the operation of density-dependent factors, particularly the sensitivity of egg production to intra-specific competition (COMBES, 1972; JACKSON & TINSLEY, 1988; TOCQUE & TINSLEY, 1991a). There are many other examples which illustrate the richness of interest in this single family of monogeneans. This review aims to bring together life cycle data to assess the population dynamics of polystomatids ; it focuses on the representatives infecting anuran amphibians because these species are, at present, better documented and there is more comprehensive information on host ecology. This bias is certain to be redressed by future studies on other groups of polystomatids : thus, the initial reports of *Concinnocotyla australensis* reveal another series of unique features, including unusual characteristics of population biology (PICHELIN, WHITTINGTON & PEARSON, 1991).

Life cycle patterns

The hosts of most monogeneans occur continuously in the medium in which transmission takes place - in water - although the continuity of invasion may still be interrupted periodically by the effects of temperature, by spatial movements of the host populations, and by other aspects of host behaviour (TINSLEY, 1983). For the polystomatid monogeneans, however, there is a fundamental difference : this is the only major monogenean group (alongside some isolated examples) where transmission is periodic because the hosts may leave the aquatic medium, interrupting transmission for intervals ranging from hours within a diel cycle to many months within an annual cycle (see TINSLEY, 1990a) : transmission is absolutely precluded during these periods and there is a corresponding direct effect on parasite population dynamics. The life cycles of many polystomatids also incorporate more or less strict specificity in the stage of the host which is invaded. Amongst representatives infecting anuran amphibians, transmission biology shows three broad patterns :

1. The host is exposed continuously to invasion (the predominant case for monogeneans infecting fish);

2. The host is infected only as a larval /juvenile stage, and then free from further invasion throughout life;

3. The young stages of the host are free from infection, invasion is confined to adults.

These different modes of transmission result in a series of characteristics affecting the dynamics of infection. Thus, pattern 1 is illustrated by the aquatic anuran *Xenopus* which is exposed to infection by *Protopolystoma* more or less continually throughout life (after metamorphosis). This is also likely to be the case for the other polystomatids infecting fully-aquatic hosts, although there is little direct information (*Concinnocotyla* infecting Australian lungfish, *Polystomoides* and *Neopolystoma* infecting some aquatic chelonians). In some cases, transmission may still be interrupted in the short term by host behaviour (periodic emergence on land) and by environmental effects (drought which forces *Xenopus* and some terrapins to aestivate). Nevertheless, for hosts such as *Xenopus*, the relative continuity of transmission is confirmed by the presence of a range of parasite developing stages within the host population at virtually all times of the year.

Patterns 2 and 3 tend to be centred on host reproduction so that either the reproducing adult anuran or the tadpole generation become targets of invasion, and the adults and tadpoles may be carriers of the reproducing parasites. Thus, the life cycle of *Polystoma* is targeted on the tadpoles of species of *Rana, Hyla* and other mesic anurans (with eggs produced both by adult worms in spawning adult anurans and by so-called "neotenic" worms in tadpoles), and these hosts are then free from external re-infection for the whole of post-metamorphic life. In the life cycles of *Pseudodiplorchis*, *Neodiplorchis* and *Eupolystoma*, which are parasites of relatively terrestrial anurans such as *Scaphiopus* and *Bufo*, transmission occurs between adult hosts within spawning assemblies, the tadpoles are never involved, and re-infection may be repeated every year at each host spawning.

Parasite population dynamics in these three situations may be expected to follow three very different patterns :

1. A dynamic flux with recruitment and loss occurring continuously, the balance dependent on intensity of invasion, mortality rate, and duration of interruptions to transmission (when loss occurs in the absence of replacement).

2. Highest infection levels in the youngest age classes, then a continuous decline throughout the rest of life.

3. Pre-adults uninfected, then a cycle of sharp "pulses" of recruitment (usually occurring annually) leading to heavy burdens followed by decline until the next invasion episode.

Given these fundamental differences in population dynamics, it is interesting to compare the outcome : the prevalence and intensity of infection in representative species.

Parasite population size

In most cases, the published data provide a "snap-shot" - a single measure in time of parasite population size by examination of a population sample of hosts. This does not take account of the different dynamic patterns which are strongly influenced by factors such as host age and sex, seasonal fluctuations, etc. Nevertheless, many studies are based on breeding populations of anuran hosts (when normally scattered and elusive individuals aggregate together in large numbers) : this introduces some uniformity into the measurements of the parasite population since the records then concern the effective population of reproducing parasites (adult worms in adult hosts at the time of transmission) or the realised success of invasion (larval worms in recently-invaded tadpoles or adults). Table I gives a selection of data from the most intensively-studied region, Africa, together with one set of comparative data from North America which contributes to the discussion (*vide infra*). Some of these studies include a proportion of juvenile hosts which may distort the general trends : thus, MAEDER (1973) remarked that all the hosts found infected with P. *dorsalis* were juveniles (8 out of 15 examined) whereas 27 adult frogs examined were entirely uninfected. Despite these limitations, the data serve to demonstrate a general pattern of relatively low prevalence and /or intensity. The parameter which best reflects the exploitation of the host population by each species is relative density. In general, there is less (often far less) than one parasite for every individual in the host population : a majority of *Polystoma* species occur in a ratio of less than 1 adult parasite for every 5 hosts.

Some exceptions stand out from this general pattern : these are distinguished in Table I by having a relative density greater than unity. *P. grassei* and the *Metapolystoma* and *Eupolystoma* species have the capacity to boost existing infrapopulations by an internal cycle of infection

Tableau I : Caractéristiques des populations de Polystomatidae d'Amphibiens anoures.

(1) Données concernant des hôtes juvéniles (53 % infestés, n=15) ; aucun adulte de cette étude n'était infesté (n=27) (Maeder, 1973).

(2) Comprend les données pour le synonyme *P. vaucheri.*

(3) Données relatives à des infestations avec des parasites adultes reproducteurs.

(*) Valeurs approximatives.

Table I : Population characteristics of polystomatids from anuran amphibians.

(1) Data concern juvenile hosts (53% infected, n = 15) ; all adults in this survey were uninfected (n = 27) (MAEDER, 1973).

(2) Includes data for the synonymous *P. vaucheri.*

(3) Data record infections with adult reproducing parasites.

(*) Approximate values.

Species	Locality	Prevalence %	Range	Intensity	Relative density	Sample size	Reference
Polystoma							
P. aeschlimanni	Togo	5				298	Bourgat & Murith,1980
P. africanum	Togo	42				209	Combes *et al.*, 1976
P. assoulinei	Togo	13	1	1.0	0.13	16	Bourgat, 1975
P. australis	South Africa	18	1-6	2.3	0.42	38	Kok & van Wyk, 1986
P. baeri	Togo	17				60	Bourgat, 1977
P. baeri	Togo	6				54	Bourgat, 1977
P. baeri	Ivory Coast	26	1-15	3.5		174	Murith, 1981a
P. batchvarovi	Cameroun	3	2	2.0	0.05	39	Murith *et al.*, 1978
P. dorsalis	Liberia	38	1-2			16	Maeder *et al.*, 1970
P. dorsalis	Ivory Coast	19(1)	1-5			42	Maeder, 1973
P. ebriensis	Ivory Coast	5	1-3			20	Murith, 1981a
P. gabonesis	Gabon	13	1-2	1.3	0.17	54	Euzet *et al.*, 1966
P. galamensis	Togo	21	1-11	3.0	0.64	47	Euzet *et al.*, 1974
P. galamensis	Togo		1-17	2.8			Bourgat, 1977
P. grassei	Gabon	11	6	6.0	0.67	9	Euzet *et al.*, 1966
P. grassei	Cameroun	11	116	116	12.9	9	Murith *et al.*, 1978
P. ivindoi	Gabon	25	7-24	12.7	3.17	24	Euzet *et al.*, 1966
P. ivindoi	Cameroun	14	3-34	17.7	2.41	22	Murith *et al.*, 1978
P. lamottei	Togo	10				298	Bourgat & Murith, 1980
P. llewellyni	Cameroun	3	3	3.0	0.10	30	Murith *et al.*, 1978
P. mangenoti	Liberia	44	1-2	1-4	0.63	16	Maeder *et al.*, 1970
P. mangenoti (2)	Ivory Coast	8	1-2	1.4(*)	0.12(*)	107	Maeder, 1973
P. mangenoti	Ivory Coast	11	1-5	1.5		281	Murith, 1981a
P. perreti	Ivory Coast	8	2-3	2.3	0.19	37	Maeder, 1973
P. perreti	Togo	5					Bourgat, 1977
P. perreti	Ivory Coast	10	1	1.0	0.10	63	Murith, 1981a
P. ragnari	Ivory Coast	6	1-2	1.3	0.08	50	Maeder, 1973
P. ragnari	Togo	3					Bourgat, 1977
P. sodwanensis	South Africa	31		1.1	0.58	26	Du Preez & Kok, 1992
P. togoensis	Togo	8				153	Bourgat, 1977
P. togoensis	Cameroun	23	1-4			31	Murith *et al.*, 1978
P. togoensis	Ivory Coast	30	1-5	1.7		49	Murith, 1981a
P. umthakathi	South Africa	11		1.4	0.15	47	Kok & Seaman, 1987
Metapolystoma							
M. cachani	Ivory Coast	25	1-11			126	Murith *et al.*, 1977
M. cachani	Ivory Coast	24	1-15	3.5		174	Murith, 1981a
M. porosissimae	South Africa	50		2.2	1.88	26	Du Preez & Kok, 1992
Eupolystoma							
E. alluaudi	Togo	5	1-2000	240	11.6	269	Combes *et al.*, 1973
E. alluaudi	Togo	19				348	Combes *et al.*, 1976
Protopolystoma							
P. xenopodis (3)	South Africa	40	1-6	1.8		1200	Tinsley, 1972
P. xenopodis (3)	Uganda	10	1-6	2.4	0.24	241	Tinsley, 1973
Pseudodiplorchis							
P. americanus (3)	U.S.A.	41	1-27	5.7	2.34	423	Tinsley, 1989

(TINSLEY, 1983 and references); as a result, although prevalence may be relatively low, infected hosts accumulate increasing numbers of reproducing worms. An internal cycle has not yet been reported for *P. ivindoi* but it is interesting that independent studies (in Gabon and Cameroun) recorded similar, unusually high, intensities (Table I). It may be that these exceptions could also indicate other fundamental features of the host-parasite relationship. Thus, the *Polystoma* species - *P. grassei* - which has the capacity to produce the highest worm burdens in adult frogs (up to 116 worms /host (MURITH, GASSMANN & VAUCHER, 1978)), also has exceptional levels of infection in the "neotenic" generation on tadpoles (49% prevalence and up to 29 worms /host (MAEDER, 1973)). Whilst the heavy infections in adults may be explained by the operation of an internal cycle of infection, those in tadpoles must reflect other processes (perhaps a different immunological interaction with the host species - see concluding paragraph to this review).

Internal auto-infection is an exceptional process amongst all adult platyhelminths and has a dramatic effect on polystomatid dynamics, reversing the infrapopulation decline typical of pattern 2 (above). Setting aside this influence and taking a broad overview of the data in Table I, it is surprising that there are not major differences in parasite population size which reflect the fundamental differences in transmission biology. Thus, extensive samples show that *Protopolystoma* which is transmitted virtually continuously throughout the life of its host does not achieve higher prevalence or intensity than *Pseudodiplorchis* which is transmitted during less than 24h each year. The question arises, why is there so little variation in infection levels in different polystomatids ? In this review, two key determinants of population dynamics will be considered : a) the pattern and efficiency of recruitment (the factors which generate the initial invasion levels) ; b) the subsequent regulation of the surviving parasite population (the mortality factors operating post-infection which determine the potential for reproduction). These factors are influenced both by host biology and by parasite biology, and each in turn is regulated by external environmental factors.

REGULATION OF PARASITE POPULATIONS BY EXTERNAL ENVIRONMENTAL FACTORS

The external environment has a range of obvious effects on the population dynamics of most parasites through its direct and indirect effects on transmission and hence recruitment. The following examples illustrate these effects for polystomatids. In the case of *P. integerrimum*, COMBES (1968) has documented how differences in water temperature between deep and shallow water and between sunlit and shaded regions of ponds influence rates of egg development (for both host and parasite) and hence the formation of "neotenic" adults which have the capacity to boost transmission. Rainfall, of course, has a primary role in the maintenance of aquatic habitats in which transmission occurs. In North American deserts, torrential rainfall is the vital cue which triggers the spawning of *Scaphiopus couchii* (and thus the transmission of *P. americanus*) : indeed, even if water is present in temporary ponds (from previous rainfall) the toads will not enter the ponds to spawn without a fresh rainfall stimulus (TINSLEY, 1990a). In this case, therefore, rainfall provides not just the medium for infection, it has a direct role in initiating each episode of transmission. Other studies on *P. americanus* demonstrate that temperature has a major effect in regulating the life cycle : parasite growth and reproductive preparation are negligible below 20°C and are totally inhibited at 16°C. The seasonal temperature cycle in the Sonoran Desert permits optimum development for only 4 to 5.5 months per year. In years when this period is short, 1st year *P. americanus* may not be able to manufacture infective stages in time for the annual opportunity for transmission and the population of reproducing parasites is correspondingly reduced. In years when the period above 20°C exceeds 5 months, there will be a greatly increased reproducing adult population contributing larvae to transmission (TOCQUE & TINSLEY, 1991b).

REGULATION OF PARASITE POPULATIONS BY HOST BIOLOGY

Host behaviour

The population biology of many anuran polystomatids is controlled very exactly by host behaviour because of the synchrony of host and parasite oviposition. The correlation between the life cycles of *P. integerrimum* and its host is so precise that it has led to the hypothesis that parasite reproductive biology is controlled by host sex hormones (although there has never been experimental confirmation - see KEARN, 1986; TINSLEY, 1990a). The temporal coincidence

is even closer for *P. americanus* and *S. couchii* because each opportunity for host and parasite egg-laying is restricted to less than 7 h. Parasites in male toads begin to discharge oncomiracidia as soon as the hosts become sexually excited (as they enter breeding ponds), whereas parasites in females respond at around the time of spawn discharge a few hours later (TINSLEY, 1990a). This control of parasite transmission is so strict that parasites in toads which are in poor physical condition after hibernation and which do not become aroused sexually, fail to release their oncomiracidia : even if these parasites are fully-prepared for transmission they may make no contribution because they do not receive the essential stimulus provided by the host (TINSLEY, 1990b). For *P. americanus*, host behaviour also determines that male *S. couchii* receive heavier invasions than females : males remain in water at breeding sites for the maximum duration of each nocturnal exposure (up to 7 h), and if there are repeated rainstorms males may return to further mating assemblies, multiplying their exposure duration. Female *S. couchii*, on the other hand, enter spawning ponds only once and then leave water as soon as they have spawned, restricting their exposure to infection to only 3 or 4 h each year. As a result of these sex-specific differences in host behaviour, there are about 4 times more parasites in the males than the females in a population of *S. couchii* (see TINSLEY, 1989). COMBES, BOURGAT & SALAMI-CADOUX (1976) recorded a higher prevalence of *Eupolystoma alluaudi* in male (21%) compared with female (13%) *Bufo regularis* where transmission is also venereal and is probably regulated in the same way by host behaviour.

Field studies on *S. couchii* have indicated other, more subtle, effects of host behaviour on invasion levels. Male toads which fail to secure a mate usually acquire heavier infections of *P. americanus* than males which mate successfully. One explanation might be that unsuccessful males are more mobile, swimming through a greater volume of water during the brief assembly and encountering larger numbers of swimming oncomiracidia ; males which mate with a female are far more sedentary during amplexus and may have correspondingly reduced encounters with infective larvae (TINSLEY, 1989).

Host activity has other profound effects on the life cycle of *P. americanus*. Larvae which initially invade the respiratory tract subsequently migrate via the digestive tract to the urinary bladder where they begin preparation for transmission. Migration is triggered in some way by host activity (TINSLEY & JACKSON, 1986) and tegumental vesicles are discharged which protect the surface of worms from digestive attack (CABLE & TINSLEY, 1992a). If the toads are inactive at the time when migration would normally occur (one month post infection), the parasites remain arrested in the respiratory tract throughout hibernation until the host is next active (at the start of the next summer's breeding season) : their reproductive development is then delayed by nearly one year. These events involve an interaction between environmental factors (the early onset of unfavourable conditions), host behaviour (an end to the activity season and early hibernation), and parasite development (which is arrested because of the failure of an essential trigger), and the outcome is that a fraction of the recently-invaded parasite population fails to contribute to the next opportunity for transmission. In some years, this fraction which fails to reproduce (because of delayed migration and /or reduced developmental rate at low temperatures (see above)) exceeds the proportion which have accumulated uterine larvae ready for the once-a-year transmission (TINSLEY & JACKSON, 1988).

These effects of host activity, on larval recruitment and on the timing of reproductive development, are important in determining the size of the adult parasite population which contributes to future episodes of transmission : other host factors are important in reducing this potential.

Host mortality

Anuran amphibians show a wide variety of reproductive modes (DUELLMAN & TRUEB, 1986). For a majority of anurans which are hosts of polystomatid monogeneans, typical life history patterns involve high egg production, large populations of tadpoles, and high mortality of offspring both before and soon after metamorphosis. Anurans experience a wide range of environmental hazards, especially in the dual requirement for aquatic and moist terrestrial habitats, and predation risks are probably high. SAVAGE (1950) considered that the mortality of pre-metamorphic *Rana temporaria* exceeds 99%. For polystomatids which infect the tadpole and begin reproduction only in sexually mature hosts, the mortality of pre-adult hosts automatically eliminates all contribution to transmission from this fraction of the parasite population.

Although there is little precise documentation, the mortality rates of most anurans are likely to be greatly reduced by the time of maturity. Adult anurans therefore represent much safer targets for invasion and a higher proportion of invaded parasites are likely to survive to reproduce. (TINSLEY, 1983 provides a comparison of bionomics of infection of *Pseudodiplorchis* and *Polystoma* and especially the influence of generation time on parasite fecundity.)

Host longevity

In addition to the effects of host mortality due to environmental hazards, parasite reproduction is also regulated by the intrinsic longevity of the host species. For the fraction of the parasite population which survives to maturity, the number of opportunities for transmission is strictly limited by the number of breeding seasons during the life of the adult host. Recent application of skeletochronological techniques to age analysis of anuran populations provides valuable data on potential life-time reproductive output of polystomatid parasites. Thus, for *Rana temporaria* in Ireland, which survives up to 7 years (GIBBONS & McCARTHY, 1983), *P. integerrimum* could contribute to transmission in up to 5 seasons (although a diminishing proportion of parasites in a diminishing proportion of hosts would contribute through this time period). *Bufo pentoni* is a host of *Eupolystoma alluaudi* in West Africa, and age determination for one population sample indicated 24% of toads were aged 5 years, with maximum survival 6 years (FRANCILLON *et al.*, 1984). In this case, *E. alluaudi* invading at host maturity (2 years) would potentially have several years for transmission. Recent age analysis of other anuran species indicates that polystomatid reproduction may be significantly reduced by a short host life span. CHERRY & FRANCILLON-VIEILLOT (1992) recorded an age range of 1-3 years for male *Bufo pardalis* in breeding assemblies, and 2-6 years for females. *E. anterorchis* has an annual cycle of infection (TINSLEY, 1978a,b and unpublished studies) and therefore the first entry of *B. pardalis* into breeding ponds provides the first chance for invasion. These parasites will then have their first opportunity to release infective stages in the second breeding season. However, the data of CHERRY & FRANCILLON-VIEILLOT (admittedly based on rather small samples : 16 males, 12 females) suggest that only 19% of males and 25% of females survive to breed more than twice, and this automatically means that the majority of *E. anterorchis* make only a single contribution to transmission. Indeed, since release and recruitment of infective stages occur simultaneously, most of the oncomiracidia responsible for transmission come from the adult parasites in 2 year-old males and 3 year-old females, but most of the larvae re-invading these same hosts will never reproduce because their hosts will not survive.

One advantage of the *Polystoma life* cycle (involving invasion of tadpoles) is that parasites contribute to transmission at the first entry of their hosts into breeding assemblies. However, for short-lived hosts there may still be few opportunities for transmission. Studies (TINSLEY, TOCQUE & MARUBINI, in preparation) on the age structure of spawning assemblies of *Hyla versicolor* show that the great majority of treefrogs breed only once or twice, so this sets a limit on the reproductive potential of the major part of the *P. nearcticum* suprapopulation.

Current analysis of the population age structure of *Scaphiopus couchii* shows that the desert toads are relatively long-lived with maximum survival exceeding 12 years (TINSLEY & TOCQUE, in preparation). In cases such as this, host longevity has a reduced influence on parasite population dynamics and other factors (including parasite longevity) become more important (*vide infra*).

REGULATION OF PARASITE POPULATIONS BY PARASITE BIOLOGY

Aspects of parasite biology have the same dual effect on parasite population dynamics as has already been considered for host factors, influencing the process of recruitment (and hence initial infection levels), and influencing parasite mortality (and hence the size of the population surviving to contribute to further transmission).

Intra-specific competition

As mentioned in the opening paragraph, competitive effects have been documented for 3 polystomatid species. A remarkable aspect of the data for *Polystoma integerrimum* and *Protopolystoma xenopodis* is the extreme sensitivity of parasite egg production to "crowding effects". In both cases, mean *per capita* egg output was reduced in burdens of 2,3 and 4

parasites /host in comparison with output from single worm burdens (COMBES, 1972 ; JACKSON & TINSLEY, 1988). In *Pseudodiplorchis americanus*, competitive effects are evident only in much larger burdens but, since the total annual production of offspring is retained *in utero* within individual parasites, this system provides a means of distinguishing between "scramble competition" (where all individuals show a decrease in fitness) and "contest competition" (where some individuals suffer more than others) (TOCQUE & TINSLEY, 1991a). Field and lab data demonstrate that a small proportion of *P. americanus* can produce larvae at close to the highest rates at all parasite densities (up to 30 worms /host); however, as parasite density increases a greater proportion of individuals show reduced production of larvae : in other words, there is an increasing reproductive asymmetry within parasite infrapopulations with numbers of larvae /parasite skewed towards lower output in heavier infections. Since these effects operate unequally within infrapopulations, competition alters the relative fitness of individual parasites.

Although the threshold at which reduced reproduction is detectable in *P. americanus* (above 5 worms /host) is higher than in *P. integerrimum* and *P. xenopodis*, in all three cases this critical level is close to the mean worm burden observed in natural populations of these species (around 6 worms /host for *P. americanus*, just below 2 worms /host for the other two parasites). It is possible, therefore, that intraspecific competition results not only in depressed reproductive output but also in reduced parasite survival.

Parasite mortality

There are consistent reports in the literature of the continuous losses of parasites from gills of tadpoles beginning immediately after invasion (see, for instance, accounts for *Polystoma* species by SAVAGE (1950), KOK & DU PREEZ (1987) and for *Metapolystoma* by MURITH (1981b)). GALLIEN (1935) showed that if metamorphosis is delayed all the parasites eventually disappear. KOK (1990) recorded this mortality directly from observations of *P. umthakathi* neotenics which infect a transparent host tadpole (*Natalobatrachus bonebergi*). Normally these losses will be counteracted by continuing recruitment, and this led SAVAGE (1950) to suggest that an important role of the "neotenic cycle" is to boost invasion levels up to the last moment before tadpole metamorphosis which ends the possibility of further external invasion. Nevertheless, parasite losses continue after the parasites have migrated to the urinary bladder.

The single period of host invasion for *Polystoma* species provides a useful indication of the losses of worms from the bladder since these are not normally replaced in the parasite suprapopulation and parasite mortality will be reflected by a decline in intensity and prevalence. Such simple interpretation is, however, complicated by the finding of COMBES (1967) that after a period of oviposition by *P. integerrimum* and *P. pelobatis* a single egg may remain in the uterus, hatch and establish alongside its parent on the bladder wall. This simple process could potentially double the worm burden within each host, although Combes considered that its occurrence was rare in European *P. integerrimum*. However, the exceptionally heavy burdens recorded in some African polystomes may result from the more effective operation of this internal cycle (*vide supra*). So, with this qualification, the reports in the literature of a decline in infection levels must represent a conservative estimate of actual worm losses. There are general indications of the numerical importance of this process of loss : ZELLER (1872) reported the following age-prevalence data for *P. integerrimum* in *R. temporaria* : in frogs aged 6-7 months, 90% were infected; at 1.5 years, 33%; 2.5 years, 42%; 3-5 years, 27%; 4.5 years, 10%.

In some unpublished experimental studies on *Polystoma nearcticum* in *Hyla versicolor* (in Missouri, USA), I maintained infected treefrogs individually in plastic boxes in order to collect parasite eggs which were expelled with the urine into water in the bottom of each box. This regime meant that any parasites expelled from these hosts could also be recorded. In total, 28 infected frogs were maintained in the lab for 2-24 days from collection in the field until dissection. Worm losses were calculated for a cohort of 40 parasites whose survival was followed for a total of 266 parasite days (the period of observation until expulsion or host dissection). The loss of worms from the parasite population was 0.075% per day leading to a total mortality of 20% within the observation period (TINSLEY, in preparation). *H. versicolor* hibernates for 3-4 months /year at low temperatures when the risks of worm loss are presumably greatly reduced. The total annual period "at risk" for *P. nearcticum* may therefore be close to the 266 days of host activity followed in this study : the 20% mortality recorded may give a reasonable approximation of normal annual parasite mortality.

Parasite longevity

Apart from parasite mortality which is "premature" (in the sense that death occurs before the parasites can reproduce), parasite population biology may also be regulated by the intrinsic life span of the parasite which limits the extent of the contribution to transmission (duration and number of occasions when offspring are produced). There is little information on parasite longevity in populations of most polystomatids. A guide to life-time reproductive output is provided by records of maximum longevity : for *Protopolystoma xenopodis* this is about 2 years (JACKSON & TINSLEY, 1988), for *Pseudodiplorchis americanus* about 4 years (TOCQUE & TINSLEY, 1991a), for *Polystoma integerrimum* at least 5 years (ZELLER, 1872). However, the pre-reproductive period (respectively 3 months, 1 year and 3 years in these examples) reduces this potential. Clearly, maximum survival has limited relevance for interpretation of population biology : the shape of the survivorship curve will have a more significant effect on reproductive contribution in each population age class. ZELLER'S records for *P. integerrimum* showed that prevalence had dropped to around 10% in hosts (and parasites) aged 4.5 years (compared with a prevalence over 40% at the time when reproduction begins), and intensity is reduced to 1 or 2 in mature frogs. COMBES, BOURGAT & SALAMI-CADOUX (1976) recorded a strong inverse relationship between parasite prevalence and host body size for *P. africanum* in Togo. Up to 70% of individuals in the smallest size classes of *Bufo regularis* were infected, but toads above 10 cm body length were all uninfected. In these cases, where a potentially long-lived host can only be infected as a tadpole, parasite mortality leaves an increasing fraction of the host population unexploited. In *P. americanus* and *P. xenopodis* where parasite longevity is considerably less than host longevity, the life cycle involves repeated re-infection so a succession of parasite age cohorts occurs in the respective host populations. TOCQUE & TINSLEY (1991a) recorded age-specific data for *P. americanus* which show population age structure together with age-related changes in reproductive output by individual worms. The total parasite population recovered from one host population sample showed almost equal representation of 1st and 2nd year worms (both about 40% of the total) ; the proportion of 3 year old worms was about half that of the previous cohorts (19%) ; and a single parasite (0.6% of the sample) was judged to be 4 years old, representing maximum survivorship. This study demonstrated the progressive increase in reproductive output with parasite age : thus, the 20% of the suprapopulation aged 3 and 4 years produced more infective stages than the 80% of the total population in the first two year classes.

Two further mortality factors which regulate parasite populations arise from interactions between parasite and host biology : parasite-induced host mortality and host-mediated parasite mortality (the immune response).

REGULATION BY PARASITE-INDUCED HOST MORTALITY

An important negative feedback control of parasite suprapopulations could result from the selective deaths of heavily-infected hosts due to pathological damage. Life cycles of the *Polystoma* type should, theoretically, have the most serious pathogenic effects on the host population : maximum infection levels occur in the youngest age classes which might be least able to tolerate damage. However, there is no evidence of parasite-induced mortality. SAVAGE (1950) tested field data for a series of correlations between parasite infection and tadpole size and concluded that the gill stages of *P. integerrimum* had no significant effect on host growth or survival. For metamorphosed hosts, the effects of infection should diminish with age (as worm burdens decline), although there might be a complex interaction between the effects of large numbers of small parasites succeeded by smaller numbers of large parasites.

Life cycles restricted to sexually-mature hosts focus infection on individuals which should potentially be better able to tolerate the drain on resources due to blood-feeding parasites. On the other hand, the larval stages of some of these parasites infect organs in which pathogenic effects could be serious. SALAMI-CADOUX (1975) has described tissue damage caused by *E. alluaudi* in the kidneys of *Bufo regularis*. *Protopolystoma xenopodis* also initially invades the host's kidneys : all post-metamorphic stages may carry a succession of developing stages until migration to the urinary bladder occurs 10 - 12 weeks p.i. (TINSLEY & OWEN, 1975). The initial development of *Pseudodiplorchis* and *Neodiplorchis* occurs in the host's lungs and there may be heavy burdens of blood-feeding larvae (TINSLEY & EARLE, 1983). However, since each year's invasions are more or less synchronized, the duration of this potentially-

damaging phase in the life cycle is limited, and no instances of host mortality associated with heavy lung infections have been recorded during extensive field and laboratory studies (TINSLEY, unpublished).

Amongst polystomatids of anurans, the long-term pathogenic effects of *Pseudodiplorchis americanus* infections are potentially the most severe. (There is little information on *Eupolystoma* species which can create much heavier burdens, although the worms are much smaller.) The desert host of *P. americanus* hibernates for 10-11 months each year and during this period of total starvation there can be no replacement of resources, including blood, removed by the parasite except by utilization of reserves laid down for host survival. In this extreme situation, therefore, the polystomatid infection competes with the host for essential reserves. Comprehensive studies, both in the field and in laboratory-controlled conditions, have documented the pathogenic effects of *P. americanus*. Adult worms remove up to 5µl of blood /parasite /week at 25°C (TOCQUE & TINSLEY, 1992), and this drain is reflected in a decrease in host lipid reserves. Field data show that, during hibernation, the parasites are responsible for the loss of about 7% of the lipid reserves which are the main energy resource for host survival (TOCQUE, 1993). Toads which feed very successfully during the short activity season and lay down a surplus of lipid can tolerate this drain and survive hibernation in good condition. On the other hand, toads which fail to feed adequately, or which experience an unusually severe or prolonged period of hibernation, could exhaust their energy reserves at a faster rate because of heavy parasite burdens and die. Of course, this outcome cannot be recorded in the field since these hosts would not normally be found. However, despite this theoretical possibility, there is no evidence from laboratory experiments that parasite pathology is a significant cause of host mortality. Instead, lab studies show consistently that heavy parasite burdens (up to 300 worms /host) administered at the start of hibernation disappear from the host population : toads aroused after 9 months are generally in good condition with fewer than 30 worms /host (TINSLEY, 1990b).

HOST-MEDIATED PARASITE MORTALITY

There is now good evidence, largely derived from research on African *Xenopus* species, that anuran amphibians possess immune defences that compare in all essential details with those of mammals. Anurans display cell-mediated and humoral immunity, they possess immunoglobulins, distinct T- and B-cells and lymphoid tissues, and the defences involve immunological memory. The operation of this system in anurans, as in other ectothermic vertebrates, is temperature-dependent (see TINSLEY, 1989). There is no information on the potential of the amphibian immune response to operate against helminth parasites. Nevertheless, data presented in the concluding section of this review provide circumstantial evidence from parasite population dynamics that there may be a major control of polystomatid infections by the host immune response (*vide infra*).

PARASITE-MEDIATED REGULATION OF PARASITE POPULATIONS

All the factors so far considered which may exert a regulatory effect on the population dynamics of polystomatids have close parallels elsewhere in parasitology and, in many cases, in animal ecology. However, an additional regulatory mechanism occurs within the Polystomatidae which, so far, appears to be unique. *Eupolystoma alluaudi* produces two different types of oncomiracidia : ciliated larvae which are released from the host and are immediately infective to other toads congregating together in water, and non-ciliated larvae which hatch *in utero* within the parent worm, emerge into the urinary bladder and re-infect the host internally. This latter type lacks not only the cilia required for locomotion in the external environment but also the sense organs presumed to be involved in host invasion. The production of these alternative types of larvae appears to be controlled by the numbers of parasites infecting individual hosts : when infrapopulations are small, a majority of non-ciliated larvae are produced which boost the numbers within that host ; when infrapopulations are large, ciliated larvae are produced which are destined to transmit to other hosts. Thus, the mode of infection is programmed into the development of the oncomiracidia whilst *in utero* and this achieves a remarkable density-dependent regulation of the parasite population (FOURNIER & COMBES, 1979). The same dimorphic development of oncomiracidia has subsequently been recorded in *Polystomoides nabedei* which infects a chelonian, confirming the distinctiveness of the life cycle patterns in polystomatid monogeneans (LAMBERT & KULO, 1982).

DETERMINANTS OF PARASITE POPULATION BIOLOGY

Parasite recruitment

This review has focused primarily on two components of parasite population dynamics : entry into the parasite population (the pattern of invasion) and the regulation of parasite populations surviving to reproduce (which governs the potential for transmission). Most data indicate that initial invasion of anuran amphibians is a relatively efficient process (as would be predicted for a water-borne infection involving more or less aggregated targets). At a detailed level there is evidence of how this efficiency is enhanced : thus, a circadian rhythm of egg hatching improves the chances of contact between infective stages of *P. integerrimum* and the tadpole hosts (MACDONALD & COMBES, 1978).

Following recent field and laboratory studies, *P. americanus* now provides a relatively well-documented system for illustrating parasite population dynamics. The circumstances permitting transmission are particularly well-defined because the host, the North American desert toad *Scaphiopus couchii*, is terrestrial for almost all of each year but enters temporary ponds to breed on only 1-3 nights per year, after torrential rainfall. These occasions represent the only opportunities for water-borne invasion, but the strictly nocturnal behaviour of the toads reduces each opportunity still further to a maximum of 7 hours (21.00 - 04.00h) so that transmission is restricted to a total of less than 24h each year. TINSLEY & JACKSON (1988) documented the effectiveness of the process in a year when there were 3 episodes of transmission : by the end of the third night, prevalence in male toads reached 100% and mean intensity exceeded 80 larvae /toad (burdens in females were lower, corresponding with the shorter duration of exposure, usually on just one night each year). Subsequent field studies in the same area of desert have shown that this virtual saturation of the host population is more or less typical (TINSLEY, 1989 and in preparation).

The parasites develop to maturity and accumulate infective stages *in utero* during the long period of host hibernation. However, when these toads emerge from dormancy in the following summer, both prevalence and intensity show a major reduction. Consistently, the prevalence of adult parasites is around 50% and mean intensity is around 6 worms /infected host. The maximum burdens recorded after transmission, 300-400 larvae /host, fall to less than 30. Successive annual samples from the same population demonstrate that the magnitude of this population reduction is very uniform from year to year. Preliminary data for 1983 - 1987 (TINSLEY, 1989) have since been extended to 1991 : despite the very high invasion success, the relative density of adult worms one year post-infection remains remarkably constant at around 3 worms /toad. The scale of the losses can be quantified relatively precisely : up to 50% of toads lose their infection and only 3 - 4% of the parasites successfully invading the host population will survive to reproduce. This constancy of worm burdens is remarkable because it persists despite relatively wide year-to-year variations in the environmental conditions which, intuitively, should affect invasion success. Thus, during the years of this study, there have been major differences in the amount and frequency of rainfall during the summer activity seasons which have directly affected the number and duration of breeding assemblies and hence exposure to oncomiracidial invasion. However, the effects of this variation in parasite recruitment appear to have been overridden by some other factors which exert a dominant control over surviving parasite numbers. Key determinants in the population dynamics of *P. americanus* are, therefore, the factors responsible for parasite mortality.

Parasite mortality factors

The information reviewed above shows that polystomatid populations are regulated by a range of mortality factors. Some such as host mortality are direct, others are the result of complex interaction between independent influences. Thus, for *P. americanus*, environmental conditions in the desert affect host activity ; this determines the timing of parasite migration to the urinary bladder which governs the size of the adult parasite population contributing to transmission. Indeed, there is a close interaction with host biology for all polystomatid monogeneans such that fundamental characteristics of the parasite are actually controlled by host characteristics. For instance, the synchrony of parasite and host reproduction automatically imposes a long generation time on *Polystoma* - longer than that of most parasites and even most free-living invertebrates. This means that *P. integerrimum* makes no contribution to transmission for up to 3 years ; host death during this time will eliminate all parasite reproductive potential ; and,

as outlined above, host longevity may then restrict parasite egg production to only a few occasions.

The losses of polystomatids from hosts which have been invaded successfully is well documented although the factors responsible are not identified. The general impression (see, for instance, SAVAGE, 1950; TINSLEY, 1972; KOK & DU PREEZ, 1989) is that expulsion of worms both from the gill chamber and the urinary bladder is "accidental" : the chance loss of attachment in the gill-ventilating current or during host urination (when there is a rapid change in the surface area of the elastic bladder wall). The worms expelled are often recovered still active, indeed they may even re-establish if immediately re-introduced into the host (TINSLEY, 1972).

In the *Pseudodiplorchis /Scaphiopus* system there is more information to document the multi-factorial nature of parasite mortality. In samples of all life cycle stages examined by CABLE & TINSLEY (1992b) for electronmicroscopy, up to 10% of worms were infected by a microsporidean hyperparasite. Most tissues were infected including encapsulated embryos *in utero* : oncomiracidia may therefore emerge already contaminated and carry the pathogen to other toads, infecting other polystomatid infrapopulations. The precise role of this hyperparasite in parasite mortality is unknown (although microsporideans are known to be pathogenic in digeneans), but the degree of structural damage in certain tissues suggested that some interference with function is likely (CABLE & TINSLEY, 1992b).

The migration through the gut of the host by developing juvenile worms (noted above) may be responsible for the deaths of another fraction of the parasite population : CABLE & TINSLEY (1992a) found that, following experimental triggering of migration, 11% of parasites recovered from the host's gut had died during the passage.

Deaths of heavily-infected hosts may constitute a further source of parasite mortality. The pathogenic effects of *P. americanus* have been measured in field and laboratory populations of *S. couchii* (see TOCQUE, 1993; TOCQUE & TINSLEY, 1993), and blood loss due to infection has been quantified (TOCQUE & TINSLEY, 1992). It may be predicted from these studies that heavy infections of *P. americanus* have the capacity to kill hosts which are in poor physical condition during hibernation. However, as noted above, attempts to simulate this in the laboratory have consistently resulted in a major reduction in the parasite burdens from most hosts - the situation which is encountered in natural populations (TINSLEY, 1990b).

Even in combination, these potential effects cannot contribute to more than a part of the total worm losses. Competitive interactions, which have a measurable effect on reproductive output (TOCQUE & TINSLEY, 1991a) and may affect parasite survival (*vide supra*), cannot account for the total loss of infection by up to 50% of toads. Two important features of the population reduction of *P. americanus* are its scale and the apparent constancy of its effect. The scale (amounting to a loss of around 96% of the worms initially invading) overwhelms even apparently major environmental effects such as the frequency and duration of rainstorms, and hence extent of larval recruitment. The constancy, such that in successive years there is only very minor variation in prevalence and intensity (around a relative density of 3 worms /toad), is suggestive of a dominant characteristic of the host population rather than a combination of the minor factors outlined above. This control of parasite populations strongly resembles - although there is not yet any experimental evidence - the operation of an immune response. A number of characteristics support this hypothesis. In experimental infections maintained in the laboratory, there is a marked inverse relationship between parasite survival and temperature : at 15°C, worm burdens survive without decrease for over 14 months; at 25°C, burdens decline and all toads become uninfected after 10 months (TOCQUE, 1990 and in preparation). In the desert, soil temperatures are below 15°C for at least 6 months, and above 25°C for around 3 months (TOCQUE & TINSLEY, 1991b) ; every adult male *S. couchii* becomes infected each year, but up to 50% lose all parasites during hibernation. These events are consistent with the operation of a temperature-dependent immune response. Current studies correlating parasite infection with host age (TINSLEY & TOCQUE, in preparation) show trends consistent with age-related immunity. It might be predicted that repeated annual infection by *P. americanus* would lead to progressively greater burdens of adult worms during the host's life-time. This does not occur : infection levels rise to a plateau in toads aged about 6 years but then decline in the oldest age groups despite continued re-infection. Clearly, these ideas can only be conjectural and specific immunological studies are now required to determine the role of host immunity in regulating polystomatid populations.

POPULATION CHARACTERISTICS OF *PSEUDODIPLORCHIS AMERICANUS*

Continuing studies on *P. americanus* have now produced some detailed data enabling this species to be employed as a model illustrating some fundamental characteristics of polystomatid population biology. The distribution of parasite infection levels within the host population provides a framework for identifying essential features : Fig. 1 illustrates data for adult parasites containing oncomiracidia *in utero* at the time of host spawning, i.e. the effective reproducing parasite population. Data are derived from male *S. couchii* in one area of the Sonoran Desert, Arizona (see TINSLEY, 1989, 1990a).

The frequency distribution of worm burdens is overdispersed, as is typical of most parasite populations. In a host breeding assembly with the pattern of infection shown in Fig. 1, 52% of the male toads are uninfected, and 25% carry small burdens of 1-4 worms /host. Decreasing numbers of hosts carry heavier burdens - 9% of toads carry over 9 worms each up to a maximum of 27, but this fraction of hosts actually contains nearly half (48%) of the total parasite suprapopulation. This pattern has implications for many of the aspects of parasite population biology considered above. In relation to numbers of reproducing parasites, the majority of hosts entering spawning sites are each responsible for the release of relatively small numbers of infective stages. A few hosts will have a disproportionate effect in infecting others : from the field data used in Fig. 1, 3 hosts carrying the heaviest infections (two with 24 worms and 1 with 27 worms) contain the same total potential for infection as 48 hosts carrying the lightest burdens (1 or 2 worms /host). This potential, for a minority of host individuals to be responsible for a major part of transmission within the host population, emphasises the role of chance in the dynamic process : the entry of these few individuals into a spawning assembly may greatly alter the pattern of

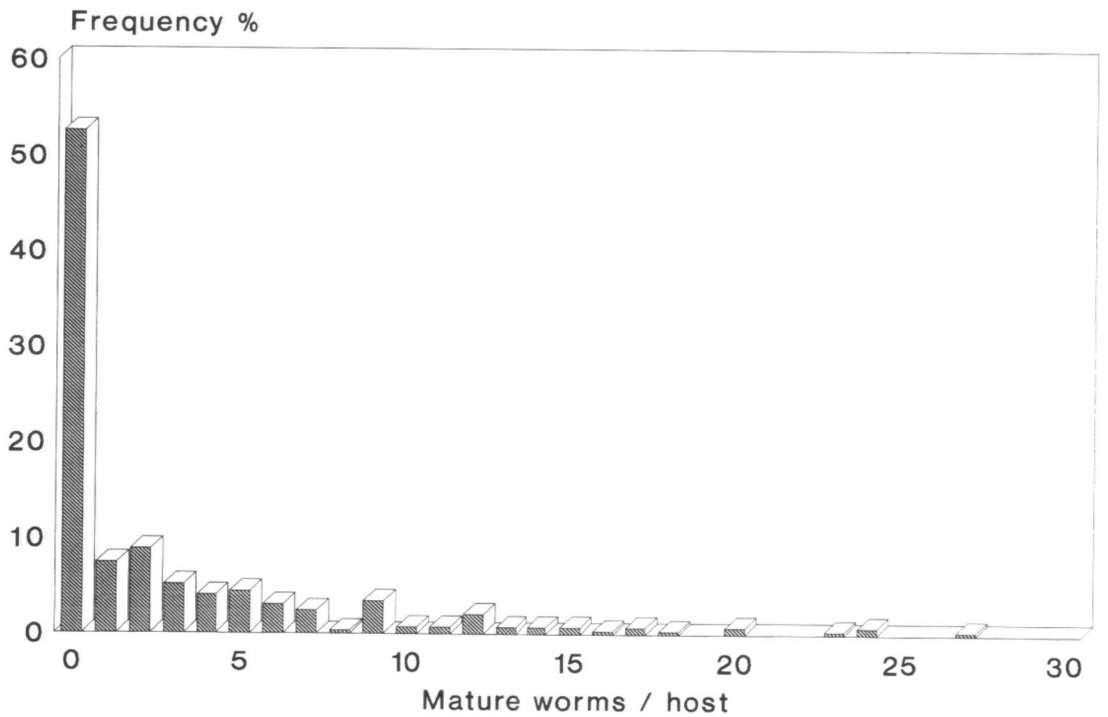

Figure 1 : Distribution des fréquences des niveaux d'infestation des adultes de *Pseudodiplorchis americanus* chez des *Scaphiopus couchii* mâles (n=297), désert de Sonoran Arizona. Les données (de Tinsley, 1989, 1990 a) sont relatives aux parasites contenant des stades infestants *in utero* et donc issus d'une infestation d'au moins un an.

Figure 1 : Frequency distribution of infection levels of adult *Pseudodiplorchis americanus* in male *Scaphiopus couchii* (n = 297), Sonoran Desert, Arizona. Data (from TINSLEY, 1989, 1990a) relate to parasites containing infective stages *in utero* and therefore at least one year post-infection.

infection in the majority. However, the documentation which is now available for this system indicates that this relationship is modified by other regulatory factors. First, in these highest worm burdens, competition has a marked effect on total production of oncomiracidia : although some of the adults may release as many larvae as those in small burdens, a majority will have reduced *per capita* output. Second, worm age has an even greater influence : in the data set of TOCQUE & TINSLEY (1991a), a single 4th year parasite contained the same number of infective stages as 73 1st year parasites. This demonstrates that numbers of parasites per host actually provide an incomplete indication of the role that hosts have in transmission dynamics. Furthermore, since the trigger for release of oncomiracidia is controlled by the level of host sexual excitement (*vide supra* and TINSLEY, 1990a,b), the final determinant of transmission is the host itself.

The frequency distribution of infection levels (Fig. 1) also indicates that the majority of toads entering spawning (and transmission) sites will suffer none or little of the potential pathogenic effects of this blood-feeding parasite. Parasite-induced pathology is most likely to occur amongst the small proportion of hosts to the right of the distribution curve, but the potential for damage is determined primarily by host condition - toads which accumulated good energy reserves in the previous summer will tolerate the range of burdens recorded here. Of course, the data on potential pathogenic effects relate only to the hosts entering transmission sites : there is no information on the levels of infection, and pathology, which may have occurred earlier during host hibernation nor on the hosts which may have suffered from parasite-induced pathology and died. This latter theoretical possibility would represent an important regulatory influence : the deaths of very heavily-infected hosts would result in loss, first, of a disproportionate number of parasites and, second, in the loss of a host genotype which may be less able to tolerate or control infection. Parasite-induced host mortality could therefore regulate transmission potential, but it could also represent a selective force modifying the pattern of susceptibility in the host population.

The characteristics of population biology illustrated in Fig. 1 relate to the fraction of reproducing parasites. The scale of the regulation of parasite reproductive potential is emphasised by the fact that these numbers represent only about 4% of the parasite suprapopulation which successfully invaded the host population during the previous transmission season (TINSLEY, 1989). Host immunity may have a dominant role in this population regulation. If this is the case, then the 50% of the toad population which is free of infection should include a majority of those which are most competent immunologically (there will also be some just-mature toads entering the breeding population for the first time and not previously infected). On the other hand, the small proportion of hosts which carry the heaviest burdens may include those least capable of eliminating their infections. There is no immunological evidence to support this extrapolation, but there is now a challenge to apply immunological techniques to research in monogenean biology.

The opening section of this review and Table I showed that, with few exceptions, the infection levels of polystomatids in anuran hosts are relatively low regardless of the mode of transmission. It is remarkable that there may be so little variation in parasite population size whether hosts are exposed to infection continuously, at yearly intervals, or at only one period in the lifetime. This suggests that some other factor(s), additional to the efficiency of recruitment, provide the major regulation of infection levels and over-ride external environmental constraints. Circumstantial evidence suggests that this dominant control of polystomatid populations is due to the host immune response : further interpretation of parasite population dynamics now requires integration of ecological and immunological research.

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REFERENCES

BOURGAT R., 1975. *Polystoma assoulinei* n.sp. (Monogenea, Polystomatidae) parasite vesical de *Ptychadena huguettae* (Anura) au Togo. *Vie Milieu*, 25, 197-202.

BOURGAT R., 1977. Étude comparative des polystomes (Monogènes) de ranidés (Anoures) du sud Togo. Description de *Polystoma togoensis* sp.n. *Bull. Mus. natn. Hist. nat., Paris, 3 sér., Zoologie, 312*, 447-463.

BOURGAT R., MURITH D., 1980. *Polystoma lamottei* n.sp. et *P. aeschlimanni* n.sp., deux Polystomes (Monogènes) de la même espèce d'Amphibien : *Ptychadena pumilio* (Boulenger, 1920). *Z. Parasitenk., 62*, 293-301.

CABLE J., TINSLEY R.C., 1991a. Intra-uterine larval development in the polystomatid monogeneans *Pseudodiplorchis americanus* and *Neodiplorchis scaphiopodis*. *Parasitology, 103*, 253-266.

CABLE J., TINSLEY R.C., 1991b. The ultrastructure of photoreceptors in *Pseudodiplorchis americanus* and *Neodiplorchis scaphiopodis* (Monogenea : Polystomatidae). *Int. J. Parasitol, 21.*, 81-90.

CABLE J., TINSLEY R.C., 1992a. Unique ultrastructural adaptations of *Pseudodiplorchis americanus* (Polystomatidae : Monogenea) to a sequence of hostile conditions following host infection. *Parasitology, 105*, 229-241.

CABLE J., TINSLEY R.C., 1992b. Microsporidean hyperparasites and bacteria associated with *Pseudodiplorchis americanus* (Monogenea : Polystomatidae). *Can. J. Zool., 70*, 523-529.

CHERRY M.I., FRANCILLON-VIEILLOT H., 1992. Body size, age and reproduction in the leopard toad, *Bufo pardalis. J. Zool., Lond., 228*, 41-50.

COMBES C., 1967. Biologie des Polystomatidae (Monogenea) : existence et démonstration expérimentale des possibilités de cycle interne direct. *Bull. Soc. Zool. France, 92*, 129-133.

COMBES C., 1968. Biologie, écologie des cycles et biogéographie de digènes et monogènes d'amphibiens dans l'est des Pyrénées. *Mém. Mus. natn. Hist. nat., Paris, 51*, 1-195.

COMBES C., 1972. Écologie des Polystomatidae (Monogenea) : facteurs influençant le volume et le rythme de la ponte. *Int. J. Parasitol., 2*, 233-238.

COMBES C., BOURGAT R., SALAMI-CADOUX M.L., 1973. Biologie des Polystomatidae : Le cycle interne direct chez *Eupolystoma alluaudi* (de Beauchamp, 1913). *Z. Parasitenk., 42,* 69-75.

COMBES C., BOURGAT R., SALAMI-CADOUX M.L., 1976. Valeur adaptative du mode de transmission chez les Polystomatidae (Monogenea). *Bull. Ecol., 7*, 207-214.

DU PREEZ L.H., KOK D.J., 1992. Syntopic occurrence of new species of *Polystoma* and *Metapolystoma* (Monogenea : Polystomatidae) in *Ptychadena porosissima* in South Africa. *Syst. Parasitol., 22*, 141-150.

DUELLMAN W.E., TRUEB L., 1986. Biology of Amphibians. McGraw-Hill Book Company New York, 670 p.

EUZET L., BOURGAT R., SALAMI-CADOUX M.L., 1974. *Polystoma galamensis* (Monogenea) parasite de *Rana galamensis* Dumeril et Bibron, 1841, au Togo. *Annls. Parasit. hum. comp.*, 49, 63-68.

EUZET L., COMBES C., KNOEPFFLER L-Ph., 1966. Parasites d'Amphibiens du Gabon Polystomatidae (Monogenea). *Biologia Gabon.*, 2 (3), 215-233.

FOURNIER A., 1980. Les Monogènes d'Amphibiens, Approche ultrastructure des modifications adaptives au cours du cycle et des relations phylétiques. Thèse, Université de Perpignan.

FOURNIER A., COMBES C., 1979. Démonstration d'une dualité évolutive des embryons chez *Eupolystoma alluaudi* (Monogenea : Polystomatidae) et de son rôle dans la genèse du cycle interne. *C. R. Acad. Sc. Paris, 289*, 745-7.

FRANCILLON H., BARBAULT R., CASTANET J., DE RICQLES A., 1984. Étude complémentaire sur la biologie de l'amphibien déserticole *Bufo pentoni* : données de squelettochronologie et d'écodémographie. *Rev. Ecol. (Terre Vie), 39*, 209-224.

GALLIEN L., 1935. Recherches expérimentales sur la dimorphisme évolutif et la biologie de *Polystoma integerrimum* Frohl. *Trav. Stn. zool. Wimereux, 12*, 1-181.

GIBBONS M. M., MCCARTHY T.K., 1983. Age determination of frogs and toads (Amphibia, Anura) from North-western Europe. *Zool. Scr., 12*, 145-151.

JACKSON H.C., TINSLEY R.C., 1988. Environmental influences on egg production by the monogenean *Protopolystoma xenopodis*. *Parasitology, 97*, 115-128.

KEARN G., 1986. The eggs of Monogeneans. *Adv. Parasit., 25*, 175-273.

KOK D.J., 1990. *Polystoma umthakathi* (Monogenea) : establishment, mortality and reproduction of neotenic parasites in experimentally infected *Natalobatrachus bonebergi* tadpoles. *S. Afr. J. Zool., 25*, 26-29.

KOK D.J., DU PREEZ L.H., 1987. *Polystoma australis* (Monogenea) : life cycle studies in experimental and natural infections of normal and substitute hosts. *J. Zool., Lond., 212*, 235-243.

KOK D.J., DU PREEZ L.H., 1989. *Polystoma australis* (Monogenea) : Development and reproduction in neotenic parasites. *S. Afr. J. Zool., 24* (3), 225-230.

KOK D.J., SEAMAN M.T., 1987. Polystomatidae (Monogenea) parasitic in the anuran genus *Natalobatrachus* in South Africa. *S. Afr. J. Zool.*, 22 (4), 258-263.

KOK D.J., VAN WYK J.H., 1986. Polystomatidae (Monogenea) parasitic in the anuran genus *Kassina* in South Africa. *S. Afr. J. Zool., 21*, 189-196.

LAMBERT A., KULO S.D., 1982. Existence d'une dualité morphologique chez l'oncomiracidium de *Polystomoides nabedei* Kulo, 1980. *Annls Parasit. hum. comp., 57*, 237-243.

MACDONALD S., COMBES C., 1978. The hatching rhythm of *Polystoma integerrimum*, a monogenean from the frog *Rana temporaria*. *Chronobiologia, 5*, 277-285.

MAEDER A.M., EUZET L., COMBES C., 1970. Espèces nouvelles du genre *Polystoma* (Monogenea) en Afrique occidentale. Z. Parasitenk., 35, 140-155.

MAEDER A.M., 1973. Monogènes et trématodes parasites d'amphibiens en Côte d'Ivoire. *Revue suisse Zool., 80*, 267-322.

MURITH D., 1981a. Contribution à l'étude de la systématique des polystomes (Monogènes, Polystomatidae) parasites d'amphibiens Anoures de basse Côte d'Ivoire. *Revue suisse Zool., 88*, 475-533.

MURITH D., 1981b. Contribution à l'étude de la biologie du développement des polystomes (Monogenea) parasites d'amphibiens anoures de basse Côte d'Ivoire. *Bull. Soc. neuchatel. Sci. nat., 104*, 5-33.

MURITH D., GASSMANN M.M., VAUCHER C., 1978. Contribution à l'étude des polystomes d'amphibiens du Cameroun. *Revue suisse Zool., 85*, 681-698.

MURITH D., VAUCHER C., COMBES C., 1977. Coexistence de la néotenie et du cycle interne chez un Polystomatidae (Monogenea). *C. R. Acad. Sc. Paris*, D *284*, 187-190.

PICHELIN S., WHITTINGTON I., PEARSON J., 1991. *Concinnocotyla* (Monogenea: Polystomatidae), a new genus for the polystome from the Australian lungfish *Neoceratodus forsteri*. *Syst. Parasitol., 18*, 81-93.

SALAMI-CADOUX M.L., 1975. Transmission et développement du monogène réno-vésical *Eupolystoma alluaudi* (de Beauchamp, 1913) Euzet et Combes, 1967. *Bull. Soc. Zool. France, 100*, 283-292.

SAVAGE R.M., 1950. Observations on some natural epizootics of the trematode *Polystoma integerrimum* among tadpoles of *Rana temporaria temporaria*. *Proc. Zool. Soc., Lond., 120*, 15-37.

TINSLEY R.C., 1972. The adaptation for attachment by the Polystomatidae (Monogenoidea). *C.R. 1er Multicolloque eur. Parasitol.*, 65-68.

TINSLEY R.C., 1973. Observations of Polystomatidae (Monogenoidea) from East Africa with a description of *Polystoma makereri* n.sp.. *Z. Parasitenk., 42*, 251-263.

TINSLEY R.C., 1978a. The morphology and distribution of *Eupolystoma* species (Monogenoidea) in Africa, with the description of *E. anterorchis* sp.n. from *Bufo pardalis* at the Cape. *J. Helminthol.*, 52, 291-302.

TINSLEY R.C., 1978b. Oviposition, hatching and the oncomiracidium of *Eupolystoma anterorchis* (Monogenoidea). Parasitology, 77, 121-132.

TINSLEY R.C., 1983. Ovoviviparity in platyhelminth life cycles. *Parasitology, 86*, 161-196.

TINSLEY R.C., 1989. Effects of host sex on transmission success. *Parasitol. Today*, 5, 190-195.

TINSLEY R.C., 1990a. Host behaviour and opportunism in parasite life cycles. in BARNARD, C.J., BEHNKE, J., *Parasitism and host behaviour*, 158-192, Taylor & Francis, London.

TINSLEY R.C., 1990b. The influence of parasite infection on mating success in Spadefoot toads, *Scaphiopus couchii, Amer. Zool., 30*, 313-324.

TINSLEY R.C., EARLE C.M., 1983. Invasion of vertebrate lungs by the polystomatid monogeneans *Pseudodiplorchis americanus* and *Neodiplorchis scaphiopodis. Parasitology, 86*, 501-517.

TINSLEY R.C., JACKSON H.C., 1986. Intestinal migration in the life cycle of *Pseudodiplorchis americanus* (Monogenea). *Parasitology, 93*, 451-469.

TINSLEY, R.C., JACKSON, H.C., 1988. Pulsed transmission of *Pseudodiplorchis americanus* between desert hosts (*Scaphiopus couchii*). *Parasitology*, 97, 437-452.

TINSLEY R.C., OWEN R.W., 1975. Studies on the biology of *Protopolystoma xenopodis* (Monogenoidea) : the oncomiracidium and life cycle. *Parasitology*, 71, 445-463.

TOCQUE K., 1990. The reproductive strategy of a monogenean parasite in a desert environment. Ph.D. thesis, University of London.

TOCQUE K., 1993. The relationships between parasite burden and host reserves in the desert toad (*Scaphiopus couchii*), under natural environmental conditions. *J. Animal Ecol.* 62 (in press).

TOCQUE K., TINSLEY R.C., 1991a. Asymmetric reproductive output by the monogenean *Pseudodiplorchis americanus. Parasitology, 102*, 213-220.

TOCQUE K., TINSLEY R.C., 1991b. The influence of desert temperature cycles on the reproductive biology of *Pseudodiplorchis americanus* (Monogenea). *Parasitology, 103*, 111-120.

TOCQUE K., TINSLEY R.C., 1992. Ingestion of host blood by the monogenean *Pseudodiplorchis americanus* : a quantitative analysis. *Parasitology, 104*, 283-289.

TOCQUE K., TINSLEY R.C., 1993. The relationship between *Pseudodiplorchis americanus* (Monogenea) density and host resources under controlled environmental conditions. (In press).

ZELLER E., 1872. Untersuchungen über die Entwicklung und den Bau des *Polystomum integerrimum* Rud. *Z. Wiss. Zool., 22*, 1-28.

ZHANG S.Y., 1987. Ultrastructure of ocellus in the oncomiracidium of *Diplorchis* sp. (Monogenea : Polystomatidae). *Acta Zool. Sinica, 33*, 380.