Review

Parasitic castration: the evolution and ecology of body snatchers

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Castration is a response to the tradeoff between consumption and longevity faced by parasites. Common parasitic castrators include larval trematodes in snails, and isopod and barnacle parasites of crustaceans. The infected host (with its many unique properties) is the extended phenotype of the parasitic castrator. Because an individual parasitic castrator can usurp all the reproductive energy from a host, and that energy is limited, intra- and interspecific competition among castrators is generally intense. These parasites can be abundant and can substantially depress host density. Host populations subject to high rates of parasitic castration appear to respond by maturing more rapidly.

The virulence tradeoff

In the classic Aesop's fable, a couple were fortunate enough to have a goose that laid a golden egg daily. Greed set in and, imagining the goose to be filled with gold, they slaughtered it, only to find its innards like any other goose. Parasites must heed the moral of this fable: those who take too much risk losing everything. A parasite eats a fraction of its host – how much to consume leads to the classic virulence tradeoff [1,2]. Parasite growth and reproduction should increase with parasite consumption, but reductions in host survivorship associated with high parasite consumption (virulence) can shorten the life of a parasite. Like Aesop's goose that laid the golden egg, the host might be more valuable to the parasite if it lives a long life than if it briefly provides a large meal. Regardless of the amount of energy that a parasite consumes, most parasites leave it to the host to allocate the remainder of the host's energy to host growth, defense, survivorship, and, ultimately, reproduction. Presumably, hosts allocate this residual energy in a manner that maximizes host fitness.

What is a parasitic castrator?

Parasites in many taxa can override the allocation strategy of the host by directing their 'take' of host energy solely to host reproductive energy, thereby avoiding a decrease in host viability [3,4]. Some models suggest that such a parasite should take all the reproductive energy of the host, if possible [5–7], a strategy that castrates the host. Formally defined, parasitic castration is an infectious strategy that requires the eventual intensity-independent elimination of host reproduction as the primary means of acquiring energy [8]. In other words, a single individual parasitic castrator will prevent or block host reproduction when the parasite matures. Parasitic castration is not necessarily instantaneous, nor must it be permanent (although it usually is), nor is it merely an *ante mortem* event for a parasitoid (see below). By defining parasitic castration as a parasite-trophic strategy, we take a parasite-centered view, one that is often ignored in evolutionary theory [9].

Castration in context

Fecundity reduction or suspension can be a host strategy difficult to distinguish from parasitic castration. For example, theoretically, if hosts can expect to outlive an infection, they might temporarily divert reproductive energy into defense to tackle the infection more effectively [2,10,11]. Also, there might be some conditions such that a host should respond to infection by partially reducing fecundity even if the infection is permanent [5,11]. Such host adaptations do not constitute parasitic castration, according to our definition. Macroparasites can sometimes reduce or prevent host reproduction in high-intensity infections. However, because such an effect varies with parasite intensity, the individuals are not castrators. Although most parasitoids attack juvenile hosts, some including mermithid nematodes, Nematomorpha, fecampiid flatworms, Microspora (e.g. of Cladocera), some braconid Hymenoptera and some Diptera, consume adult hosts [4] (Table 1). In doing so, they consume the reproductive tissues early – a prudent step in the lengthy and precise process of fully consuming an adult host [1]. Such parasitoids have been called parasitic castrators (or sterilizers) [1,12], but their life-history strategy is fully consistent with the parasitoid trophic strategy, a label we feel describes them best [8]. Male sterility or loss of pregnancy can result from infection with some pathogens (e.g. mumps, toxoplasmosis, brucellosis, herpes) but this seems to be a variable side effect of pathology. Finally, some larval acanthocephalans and cestodes (notably *Schistocephalus* spp.) can castrate (though not always) their intermediate hosts [11,13]. In these cases, the parasite also requires a final host to eat its intermediate host. This combination of traits is a distinctive and relatively uncommon strategy of trophically transmitted parasitic castration [8].

Just because an infected host can reproduce does not disqualify a parasite as a parasitic castrator. After the initial infection with a parasitic castrator, some hosts can

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Table 1. Examples of taxa of which most or all species are parasitic castrators, parasitoids with cessation of reproduction well before death and trophically transmitted parasitic castrators[a]

Castrator	*Hosts*	*Remarks*
Parasitic castrators		
Ciliophora		
Orchitophrya stellarum	Starfish	Usually only of male hosts
Apicomplexa		
Mackinnonia tubificis	Oligochaetes	
Dinoflagellata		
Blastodinida[b]	Copepods	Pelagic species of hosts
Ellobiopsidae	Crustaceans	Pelagic species of hosts
Cnidaria		
Hydrichthys	Copepods	A hypercastrator
Orthonectida		
Rhopalura	Brittle stars	
Platyhelminthes		
Euplana takewakii	Brittle stars	
Trematoda: Digenea	Snails, bivalves	Only first intermediate hosts
Nematoda		
Sphaerularia bombi	Bumblebees	Behavior modification
Mollusca: Gastropoda		
Entoconchidae	Echinoderms	Highly modified morphology
Crustacea: Copepoda		Castration multiply evolved
Cardiodectes	Lantern fishes	
Sarcotretes	Lantern fishes	
Sarcotaces	Rockfishes	Highly modified morphology
Xenocoeloma	Polychaetes	
Ismaila	Nudibranchs	Some are partial castrators
Coelotrophus	Sipunculans	Highly modified morphology
Akessonia	Sipunculans	
Crustacea: Cirripedia		
Anelasma	Deep water sharks	
Rhizocephala	Decapod crustaceans	Behavior modification
Ascothoracica	Echinoderms	Highly modified morphology
Crustacea: Isopoda		
Epicaridea	Crustaceans	Behavior modification
Cymothoidae	Fishes	Small host species
Crustacea: Brachyura		
Pinnotheres	Mussels	
Insecta		
Strepsiptera	Bees, wasps	Feminization
Rodolia	Scale insects	First instar consumes brood
Vertebrata		
Encheliophis	Sea cucumbers	Some species consume gonad
Parasitoids with prior castration		
Bacteria		
Pasteuria ramosa	Cladocerans	Gigantism
Fungi		
Polycaryum laeve	Cladocerans	Gigantism
Microspora	Cladocerans, insects	Species infecting adult hosts
Dinoflagellata		
Syndinium	Copepods	
Paradinium	Copepods	
Orthonectida		Nemerteans
Platyhelminthes		
Fecampiida	Crustaceans	Species infecting adult hosts
Nematoda		
Mermithidae	Land arthropods	Sometimes cause intersexes
Daubaylia	Freshwater snails	Some species
Nematomorpha	Insects, crustaceans	Behavior modification
Insecta: Hymenoptera		
Braconidae	Weevils	Species infecting adult hosts
Trophically transmitted parasitic castrators		
Apicomplexa		
Aggregata	Crabs	Octopus final host
Platyhelminthes: Cestoda		
Dilepididae	Ants	Causes intersexes
Ligula	Minnows	Behavior modification
Schistocephalus	Sticklebacks	Precocious maturity in fish
Platyhelminthes: Digenea		
Microphallus	Snails	Hosts retain metacercariae
Acanthocephala	Beetles	Intensity-dependent impact

[a]Data are from Refs [1,4,14,63,64].
[b]Higher-level taxa are in bold font. They represent taxa with speciose radiations. Their widespread or universal trait of parasitic castration implies that castration was an ancient feature of that clade.

still reproduce although the parasite is developing [2]. For example, although most cardinalfish infected with cymothoid isopods are castrated, those with recent infections might still have functioning gonads [14]. A few hosts (particularly snails infected by trematode parthenitae) engage in a burst of reproduction before being castrated [2], a 'best of a bad situation' solution if the host can gauge that its reproductive life will soon be over [6]. Finally, although castration generally persists for the life of the parasitic castrator, host reproduction can sometimes resume after the death of the parasitic castrator, as reported infrequently for bopyrids, entoniscids and larval trematodes in snails [2,15]. Transient host reproduction before parasite maturation, or the occasional recovery of reproduction after parasite death, led early modelers [16] to assume that parasitic castration was merely an endpoint along a gradient in the reduction of host fecundity (as opposed to a distinctive parasitic strategy).

What do parasitic castrators take from their hosts?

The energetics of reproduction is not simply the mass of the gonads (generally 5–15% of the mass of the host). The host puts mass into the associated structures (e.g. sperm ducts, seminal receptacles and vitelline glands), secondary sexual characteristics (e.g. copulatory organs, ornaments and bright colors) and packaging material for offspring (e.g. egg shells). In addition, substantial behavioral and metabolic energy goes into activities such as mate selection, choice of oviposition sites, mate competition, nest construction and care of offspring. A parasitic castrator potentially redirects all this energy to parasite growth and reproduction. As a result, castrators are often similar to host reproductive organs in size, although allocation varies, and, in some cases, they can reach more than twice the size of the reproductive mass of the host [17]. Like reproductive organs, parasitic castrators grow in positive allometric proportion to host size [18–20]. Castration of the host allows parasites to be fecund and large (compared with macroparasites and pathogens). Most parasitic castrators range from 3% to 50% of the mass of their host [8,18] (Figure 1).

If parasitic castration is the ultimate solution for a parasite, why don't all parasites castrate their hosts? Some parasites simply do not have direct access to the reproductive organs of the host. Mathematical models which allow parasites to tap into host reproduction reach different conclusions, depending on assumptions such as whether the parasite drains nutrition in general, targets maintenance energy or targets reproductive energy [5]. Although all strategies can lead to changes in host reproductive output, and some parasites (e.g. bopyrid isopods) castrate their hosts without specifically targeting reproductive tissues, parasites that initially target portions of the reproductive energy of a host seem the most able to evolve as parasitic castrators.

The other side to this question is: why are all hosts not castrated by their parasites? The answer relates to why Aesop used a goose instead of a moose (with its microscopic moose ova) to illustrate his fable; only some host life histories make castration profitable [18]. A high reproductive effort is probably the most important host trait for a parasitic castrator. Investment in reproductive tissues as a proportion of body mass tends to be large in smaller-bodied species, particularly ectotherms. Perhaps for this reason, parasitic castrators are most common in invertebrates and small fishes. This contention could help to explain why there are no parasitic castrators of humans or other large vertebrates. Consuming only our small testes or ovaries would restrict such a parasite, rewarding it with relatively little of our available energy. Longevity is another important life-history feature of the host that is associated with susceptibility to parasitic castrators. Castration is an investment that yields enhanced parasite reproduction only over the long term. In short-lived hosts (such as many very small species), the parasites might as well consume everything (as do parasitoids). A notable exception is castration of annual freshwater snails by larval trematodes [2].

Are parasitic castrators important?

Although unfamiliar to medical and veterinary parasitology, castration is common among parasites of fishes and invertebrates (Table 1) [1,8–11]. For instance, in three estuaries along the coast of southern California and Baja California, parasitic castrators comprised 20% of the 150 detectable species of infectious agents [21]. The pooled biomass of parasitic castrators was 3–11 kg/ha, which exceeded the biomass of the macroparasites by two orders of magnitude. The biomass of parasitic castrators even surpassed the biomass of top predators (mostly shorebirds) in two of the three estuaries (Figure 2).

Host taxa that fit the bill for parasitic castrators include crustaceans, gastropods and echinoderms (Table 1). Larval digenean trematodes are the most common castrators of gastropods and some bivalves. Although just a single clade, these trematodes have radiated, forming a speciose assemblage of castrators. A few other parasites castrate mollusks, such as some *Ismaila* copepods in nudibranchs [22]. Crustaceans are the host group that is perhaps most affected by castrators. Castrators of crustaceans include several crustacean groups: the epicaridean isopods (bopyrids, entoniscids, dajids and several cryptoniscine families). Other important castrators of crustaceans include rhizocephalan barnacles and many parasitic dinoflagellates (including the enigmatic Ellobiopsidae). For the echinoderms, the major castrator groups are the ascothoracican barnacles in sea stars, eulimid gastropods (some of which are highly evolved endoparasitic worm-like forms in sea stars, sea urchins and sea cucumbers), *Orchitophrya* ciliates in sea stars and orthonectids in brittle stars. Pearl fish of the genus *Encheliophis* eat the gonads of the sea cucumbers in which they reside [23], and these are the only endoparasitic vertebrates familiar to us. A surprisingly diverse array of parasitic castrators infect polychaetes and fishes. Fungi commonly castrate plants [7] (a comparison and contrast between plant and animal hosts is beyond the scope of this review).

The extended phenotype

Parasitized hosts present a complex extended phenotype [24] (which sometimes differs considerably from an uninfected host phenotype, even if they visually appear

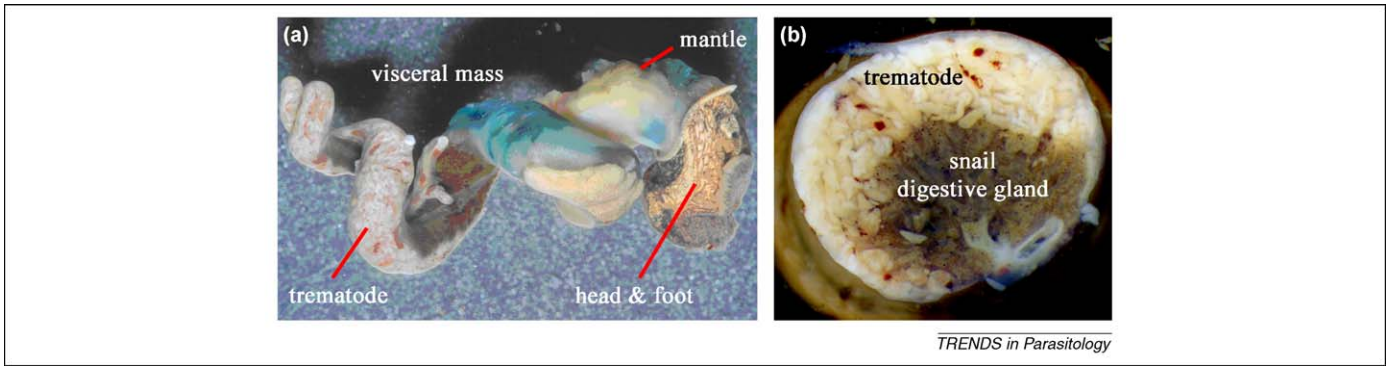

Figure 1. The snail *Cerithidea californica* castrated by trematode parthenitae. **(a)** Snail infected with *Himasthla rhigedana* extracted from the shell. **(b)** Cross-section of the visceral mass infected with *Cloacitrema michiganensis*. Reproduced, with permission, from Ref. [17].

identical) that attempts to express two genotypes: host and parasite [9]. Parasitic castration resolves this internal conflict because the residual fitness of the host is reduced to zero, and its morphology, behavior and physiology now simply supply the reproductive success of the castrator by functioning as its extended phenotype [2]. A castrated host resembles a host species but it is now a member of the parasite population, and, so long as there is little chance that the host will recover, natural selection on infected hosts operates solely on the parasite. This change can lead to differences in distribution, feeding rates, timing of reproduction and energy allocation between castrated and uninfected hosts. For example, green crabs, *Carcinus maenas*, parasitically castrated by the rhizocephalan barnacle, *Sacculina carcini*, show some striking alterations upon becoming a barnacle genotype and crab phenotype, such as feminization of male crabs [25]. Snails infected with the heterophyid trematode, *Cercaria batillariae*, migrate lower in the intertidal zone compared to the uninfected snails, putting them in closer proximity to the fishes sought out by their cercariae [26]. *Physa acuta* snails infected with *Posthodiplostomum minimum* consume significantly more periphyton than do unparasitized snails, which can be advantageous for growth of the trematode genotype [27]. In other cases, and for unknown reasons, infected snails feed less than do uninfected snails [28]. Infected snails might devote more of their total mass to reproduction of trematodes than uninfected snails devote to reproduction of snails [17]. Some larval trematodes have masses (rediae or sporocysts) comparable to that of a snail gonad (Figure 1) but most are significantly greater [29]. Infected hosts sometimes grow faster and to larger sizes than do uninfected hosts, leading to a phenomenon termed gigantism (Box 1).

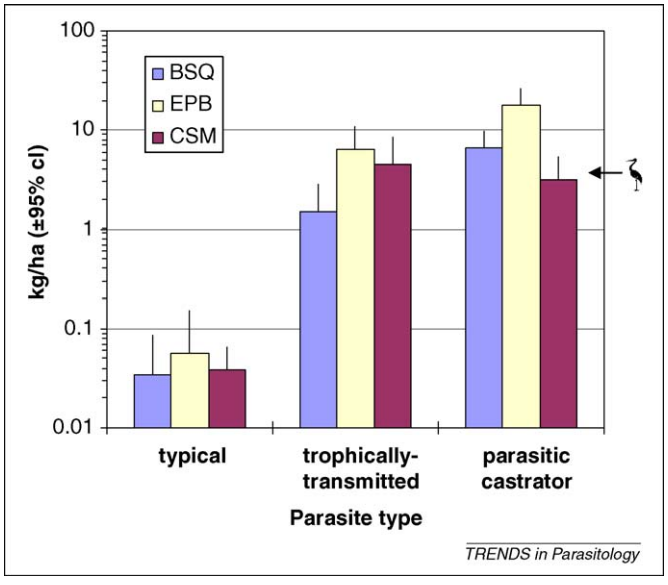

Figure 2. The biomass density (parasite mass per hectare of habitat) of parasitic castrators from three estuaries compared with the biomass of trophically transmitted parasite stages (e.g. trematode metacercariae) or macroparasites (mostly adult worms). The icon and arrow indicates the biomass of birds, which are the dominant guild of top predators in these systems. CSM: Carpinteria Salt Marsh; EPB: Estero de Punta Banda; BSQ: Bahia San Quintin. Reproduced, with permission, from Ref. [21].

How is parasitic castration achieved?

Though castration is often achieved through the selective targeting of reproductive energy, castration can also simply be the consequence of a nutritional drain, and hosts might be programmed to make up for the energetic loss by first sacrificing reproduction [11]. The energy drain hypothesis fits some bopyrid isopods that parasitize certain shrimps and crabs. Castration could also target host reproductive energy by consuming gonads [7] or by indirectly manipulating the optimal resource allocation strategy of the host [5]. Only careful empirical work can separate these options. Extensive research with larval trematodes in snails shows that sophisticated hormonal control of host physiology by the parasites directly targets reproductive energy. Molecular studies provide insight into how trematodes alter the reproductive physiology of the snail. Using humoral mechanisms, the larval trematodes alter the neuroendocrine system that normally regulates snail growth and reproduction [30]. Rhizocephalan barnacles use equally sophisticated means to castrate their hosts [31]. The high degree of host specificity of many parasitic castrators might be tied to the probable requirement of precise physiological interventions with the reproductive processes of the host. In particular, yolk proteins can be surprisingly variable among invertebrate species [32]. There is a complex control mechanism enabling expression of the genes that produce yolk proteins [33]. In effect, the parasitic castrator probably has highly specific genes for 'lock and key' biochemical mechanisms that allow the parasite to intervene and redirect nutritive yolk for its own benefit.

Box 1. Gigantism

Hall *et al.* [1] provide many examples of increased growth of hosts associated with parasitic castration. Reports of gigantism (parasitized hosts being larger than unparasitized hosts of the same age) based on size differences might be in question because it can be difficult to separate an effect of parasitism on size from an effect of size on parasitism [51]. For instance, the cumulative risk of infection increases as hosts age so that older or larger hosts are more likely to be infected [2]. Gigantism is not a general response to parasitic castration because some castrators reduce the growth of their hosts, sometimes modestly, sometimes markedly (e.g. the cymothoid isopod *Anilocra apogonae* on a cardinalfish [14], and *Sacculina carcini* on the European green crab [52]). The snail *Biomphalaria glabrata* infected with *Schistosoma mansoni* shows the complexity of castrator–host growth dynamics. Recently infected snails grow more rapidly than do uninfected snails (perhaps because the early parasitic stages are unable to use all of the available host reproductive energy), whereas older infected snails grow more slowly than do uninfected snails, perhaps reflecting their decreased life expectancy after infection [2]. Yet, some castrated hosts, including crustacean–pathogen, plant–ant, fish–tapeworm, beetle–fungus and snail–trematode interactions, clearly grow larger than their uninfected hosts [1]. Several species of short-lived, freshwater snails infected with certain larval trematodes grow larger than do uninfected individuals [2]. Explanations for increased growth have tended to consider that some castrators are too inefficient to consume all of the energy liberated by the cessation of reproduction, 'releasing' or redirecting the residual energy to the castrated phenotype for further growth [5]. In addition, some workers have postulated that gigantism could be beneficial for the castrators because of greater fecundity or survivorship [29,53]. A recent model indicates that castration prior to parasitoid-induced host death can lead to increases in host growth – as seen in *Daphnia magna* hosts infected with the castrating parasitoid *Pasteuria ramosa*, a bacterium [12]. One simple answer for gigantism is that the tradeoff between reproduction and growth is an outcome of the selective consequences of life expectancy, which differs between castrators and uninfected hosts [29]. Uninfected hosts under risk of castration mature earlier [54–56], energy thereby being diverted from growth to reproduction. An infected host with the same general sources of mortality as an uninfected host might, ironically, be relieved from the risk of infection by parasitic castrators [29]. Many castrators can defend their hosts from subsequent infections so that the parasite has a longer expected reproductive lifespan than does an uninfected host. The resulting increased expected reproductive lifespan biases the optimal allocation of energy for the castrator towards less investment in reproduction [29], potentially freeing energy for growth. Recent comparisons of trematode species that use the same host provide the clearest empirical understanding for why parasitic castrators vary in how they affect growth (R.F. Hechinger, unpublished data); the growth rate of castrated snails increases with the expected lifespan of the trematode and variation in lifespan among trematodes is attributable to the chance that a dominant trematode might replace them.

By minimizing impacts to host viability, parasitic castrators have lifted a common constraint on the growth and productivity experienced by most other types of infectious agents. However, because they focus on host reproductive effort, castrators have a rather clearly defined limit to their resource. Theoretically, they can remove host energy without impairing host longevity until they approach a ceiling dictated by the energy a host could potentially use for reproduction.

Ecological and evolutionary consequences

Parasitic worms are often aggregated, meaning that, for a given level of infection, more hosts are uninfected or are heavily infected than would randomly be expected. Highly aggregated distributions of parasites among hosts are so often reported that this dispersion pattern is almost axiomatic for macroparasites [34]. In marked contrast, dispersion of individual parasitic castrators (or a mated pair) often approaches a uniform distribution with a distinct mode and mean of one parasite per infected host [4]. The ability of a single castrator to consume all the reproductive energy of the host leads to severe intraspecific and interspecific competition and selects for mechanisms of competitive advantage among parasitic castrators [35] in a manner comparable to that used by parasitoids that face a similar cap on resources [4,36]. Because a second individual, either conspecific or heterospecific, decreases the resources a competitor could use, the fitness of both will necessarily be lower than if they were solitary. In many systems, the dominant individual eliminates the subordinate or prevents it from establishing. For example, adult female bopyrid isopods, generally accompanied by a single dwarf male consort, usually occur in single infections [37,38]. Rarely, two parasites might be present but never three or more. Sometimes, one or two juvenile females are present with a single mature female but the juveniles never appear to mature. For entoniscid isopod parasites, a single dwarf male and one or more presumptive male cryptoniscus larvae accompany one or two adult females [15]. Only at locations with a high prevalence (>70%) do multiple infections of female entoniscids become common. These sites always include several juvenile females but never more than two adult females. Strict numerical limitations on adult female parasitic castrators derive from the high-site specificity within the host crustaceans. For instance, an adult female bopyrid fills a host gill chamber, and adult female entoniscids occupy the space remaining from the obliterated ovary of the adult crab [4]. This leaves room for only two female parasites. Cymothoid isopods show a similar pattern. The cymothoid isopod parasitic castrator *Anilocra apogonae* of the five-lined cardinalfish occurs only as a single female [14]. Multiple infections of rhizocephalan barnacles sometimes occur but they are infrequent. Even in heavily infected populations, usually only one genetic individual occurs per parasitized host [39]. Multiple infections of Strepsiptera [40] in insects and the dinoflagellate *Blastodinium contortum* in copepods are also uncommon [4].

Although many trematode rediae or sporocysts can occupy a snail, these are generally clones derived from a single genetic individual. Multiple genotypes can occur within a snail but the extent to which these are stable over time is not known. For instance, most *Zeacumantus subcarinatus* snails in New Zealand infected with *Maritrema novaezealandensis* were a single genotype [41]. Some snails had as many as five genotypes but the lack of multiple genotypes in large snails suggests that intraspecific competition among castrators for the snail gonad eventually leads to competitive exclusion. In the same trematode, non-castrating metacercarial stages in crabs showed very high intraspecific genetic diversity. Experimental challenge exposures failed to reinfect snails

already harboring that species, suggesting a priority effect [41].

Interspecific competition among parasitic castrators has been studied most frequently using larval trematodes because diverse guilds of these parasitic castrators can infect molluscan first intermediate hosts [35,42,43]. These guilds have a hierarchy that is generally linear and transitive. Dominant species successfully replace, or block, the infection of all species subordinate to them. In general, trematodes with rediae are dominant over species that have only sporocysts. Where subordinate species can survive in the presence of competitive dominant species, they often occupy suboptimal sites (sometimes displaced to these sites) [44]. Here, they presumably have reduced access to resources. For example, *Renicola buchanani* does not lose out to other species of larval trematodes in the snail *Cerithidea californica*. Although most trematodes occupy the site of the former gonad, *R. buchanani* lives in the mantle tissues, where it seems to avoid strong competitive interactions. Consequently, *R. buchanani* has less reproductive mass and produces fewer cercariae than do other trematodes [17].

By eliminating the reproductive output of a fraction of the host population, parasitic castrators might reduce host density [45–47]. In addition, castrated hosts can compete with uninfected hosts for limited resources [47]. However, a reduction in host density becomes apparent only at the scale at which reproductive output of a population contributes directly to recruitment (otherwise recruits from other, perhaps less infected, populations could drive host density). At small spatial scales, or for hosts with widespread larval dispersal, the effects of castration might not be apparent but should be expected [48]. Also, parasitic castrators can increase the mortality of infected hosts [45]. Differential mortality can have further effects on host population density, independent of the spatial scale of recruitment (Box 2).

Because castrated hosts suffer reproductive death, there are probably strong adaptations to prevent infection by parasitic castrators. Selective forces acting on the immune system of the host might contribute to the pattern that parasitic castrators primarily initially infect young hosts [14,49]. For a host parasitized by a castrator, selection for defenses against the castrator necessarily ends soon after the maturation of the castrator within that host, because the residual reproductive value of such a host is effectively nil, even when an occasional host eventually outlives the castrator and regains some reproductive capability (at this age, the residual fitness of the host is probably relatively low). This contention implies that defensive selective pressures focus on prevention of an infection rather than on tolerance or recovery. Hemocytes that move, encapsulate, release cytotoxic superoxides and phagocytose are the main parts of the internal defense system that attack trematodes, and the more hemocytes that the mollusk has at its disposal, the more effective is the response, which means that adult snails are generally less susceptible to infection than are juvenile snails [50].

Box 2. How do castrators affect host populations?

Negovetich and Esch [57] developed a matrix mathematical model that indicates that trematodes reduce the population growth rate of the snail *Helisoma anceps* in Charlie's Pond, North Carolina, by 40%. General models by Antonovics [58] indicate that frequency-dependent transmission of parasitic castrators can drive host populations to even lower levels. How general are these results? A simple method for estimating the potential effect of parasitic castration on host populations is presented. This approach is unique in that it takes advantage of the ability to assess population impact from parasitic castrator prevalence – a statistic that is easy to measure in the field – and applies this to existing well-known population models. Although it is not always obvious, most population models imply that the density of hosts at equilibrium increases with the average per capita reproductive output of the population. The relationship between reproductive output and carrying capacity at equilibrium can be indicated by a re-expression of the logistic equation. A familiar form of the logistic is:

$$\partial N/\partial t = b - m(N - N/K) \quad (1)$$

where N is density, b is per capita birth, m is per capita death and K is the carrying capacity of N at equilibrium. Here, K appears to be independent of birth rates. However, an equal form of the logistic is:

$$\partial N/\partial t = N(b - m - dN) \quad (2)$$

where d is a measure of density dependence. Solving for N at equilibrium indicates that $K=(b–m)/d$. If the host suffers from parasitic castration at an equilibrium prevalence, p, the density will decline to:

$$N* = [b(1 - p) - m(1 - p) - zm\,p]/d \quad (3)$$

where z is the proportional change in mortality for infected individuals, assuming that the prevalence of castration equals the proportional reduction in the reproductive output of the population as a result of parasitism. The solution can be simplified by expressing m as a proportion of the birth rate, or $m=xb$. One measure of the effect of parasitic castration on a host population is $1–N*/K$. In other words, if the host density in the presence of a parasitic castrator is 80% of the carrying capacity, the host population is 20% less abundant than it would be without the castrator. Rearranging these equations indicates that density reduction $=p[1+zx/(1–x)]$. Hence, the minimum effect of a parasitic castrator on a host population is equal to the prevalence of the castrator in the population. Departure from the stated assumptions can affect this relationship. For instance, if some infected hosts still reproduce, the effect of parasitism will be less. If prevalence increases with host size (and larger hosts are more fecund) the effect of castration will be greater. Both assumptions could be treated if additional information were available. Additional impacts occur as parasite-induced mortality increases, and the ratio of mortality to birth rate increases, in uninfected hosts. As an example, for freshwater snail hosts of the trematode *Schistosoma mansoni*, an estimate of x is 0.15, and an estimate of z is 2 [59]. With these parameters, the impact of a parasitic castrator on the host population would be 1.35 times the prevalence. Clearly, parasitic castration is increasingly able to depress host density when castrators reach high prevalence. For example, *Sacculina carcini* had a mean prevalence of 20% in the crab *Carcinus maenas* (N=2221) at ten European locations [52], and the entoniscid isopod *Portunion conformis* averaged 41% prevalence for 20 populations of the crab *Hemigrapsus oregonensis* (N=2604) on the Pacific coast of North America [15]. A general prediction is for a negative association between the prevalence of a parasitic castrator and the density of the host population. As an example, data from two studies of green crabs (*Carcinus maenas*) and rhizocephalan barnacles *(Sacculina carcini*) in Europe are plotted (Figure I). A similar effect occurs with the experimental addition of trematode eggs to snail populations (Figure II). This strong effect indicates that parasitic castrators could be effective biological control agents [46,60].

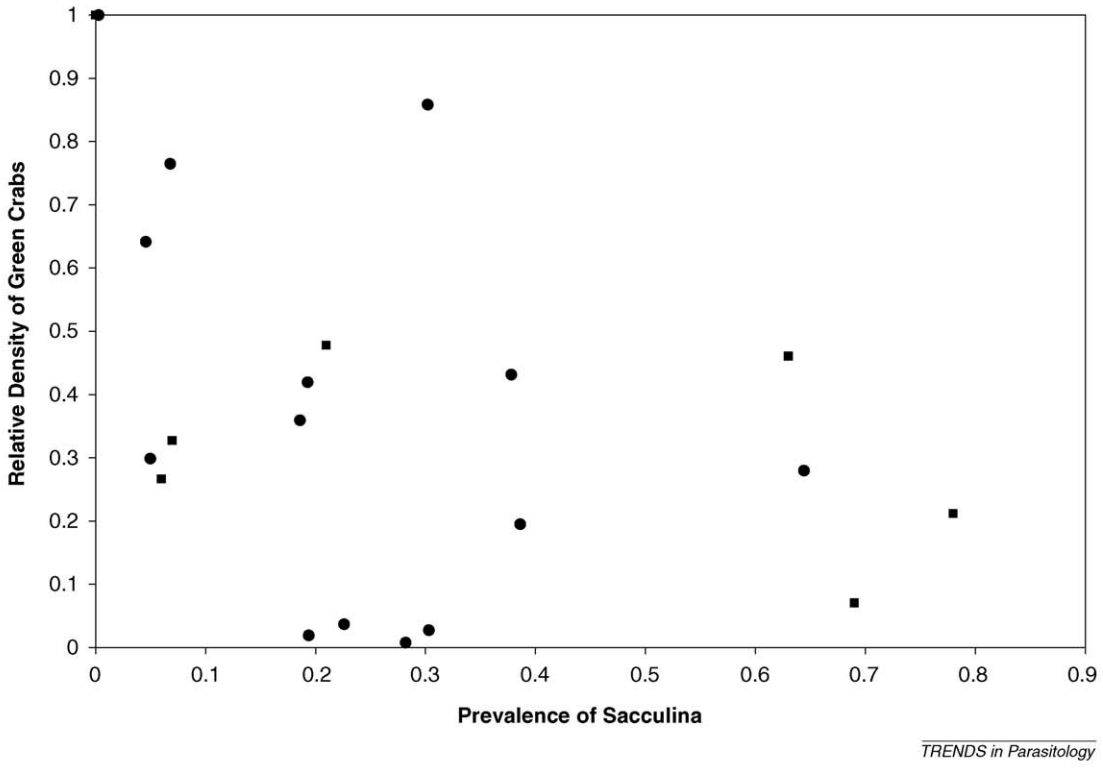

Figure I. The association between the prevalence of the parasitic castrator *Sacculina carcini* (rhizocephalan barnacle) and the estimated ratio of the host density to the host carrying capacity without parasitism. Although the association between host density and parasitic castrator prevalence is relatively easy to obtain from field data, interpreting this pattern in the context of the predictions of the models required an estimate of the carrying capacity of each population. Torchin *et al.* [52] (squares) found several locations throughout the range of the host where prevalence was zero (where the crabs should hypothetically approach carrying capacity), so carrying capacity was estimated as the average crab biomass density of the zero prevalence sites. Crab biomass at zero prevalence did vary, although not appreciably (uninfected population mean =638 g per trap, standard deviation =294, *N*=12). Data on green crab biomass and barnacle prevalence from the second study [61] are entirely from Ireland (circles). Although none of the sites had a prevalence of zero, one of the sites had a prevalence of only 0.3%, and this value was used as an estimate of the carrying capacity. For both data sets, all biomass values are expressed as a fraction of the estimate for the carrying capacity for each study. This approach allowed the carrying capacities to be standardized and data from both studies to be displayed on a single figure, which shows clearly that crabs subject to parasitic castration were below the estimated carrying capacity.

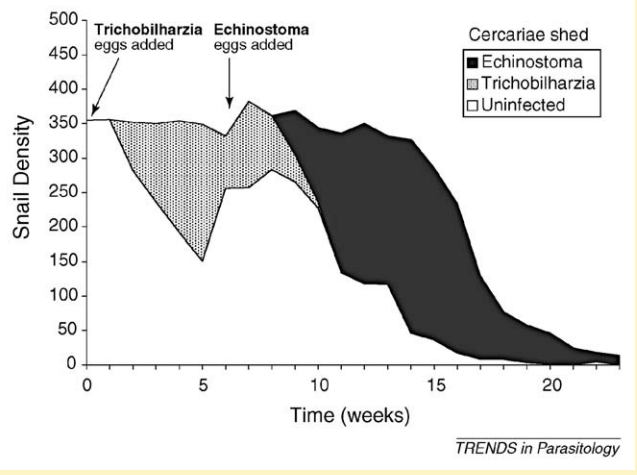

Figure II. Decreases in snail density over time after the experimental addition of trematode eggs to a pond. Uninfected snails are rapidly infected. Infected snails then die out without being replaced by uninfected snails. Data are from Ref. [62]. Reproduced, with permission, from Ref. [35].

Concluding remarks

The parasitic strategy of castration is a unique interaction between consumer and resource. Parasitic castration is a response to the classic virulence tradeoff between consumption and longevity. The most commonly observed castrators are larval trematodes in snails and isopod and barnacle parasites of crustaceans, though many other taxonomic groups have independently adopted the strategy. Invertebrates and small fishes appear to be the primary hosts for parasitic castrators because a combination of high investment into reproduction and a relatively long adult life makes castration profitable relative to other consumer strategies. While castrated, the infected host phenotype is the extended phenotype of the parasitic

castrator. The parasite alters some behaviors and other traits of the castrated host to maximize parasite fitness. This strategy can lead to distinctive, sometimes marked differences between infected and uninfected hosts, including increased or decreased growth rates and a substantially greater investment in reproductive output (of the parasite). Castration appears, in many cases, to be an elegant and sophisticated strategy that involves rewiring the neuroendocrine system of the host, probably explaining why many parasitic castrators are host-specific. Because an individual parasitic castrator can usurp all the reproductive energy from a host, these resources become limited. Hence, intra- and interspecific competition can be intense within the host. Although parasitic castrators can be an unfamiliar life-history strategy, they also can be common in terms of numbers of species and biomass. Models and empirical evidence suggest that prevalent parasitic castrators can substantially depress host density. Castrated hosts are more likely to evolve resistance than tolerance, at least to the degree that castration tends to be a permanent state. Tolerance seems unlikely given that those rare individuals that can recover will generally be near the natural end of their reproductive lives, with little residual reproductive value on which to capitalize. Instead, host populations subject to high rates of parasitic castration appear to adjust their life history so that they can mature and reproduce before becoming castrated. To understand fully the evolutionary and ecological significance of parasitic castrators will require exploration of their effects in the context of the extended phenotype. In particular, understanding the mechanistic basis of castration will help to distinguish this strategy from other cases of parasite-associated reduction in host fecundity. Considerable field and experimental work is needed to determine the effects of castration on host population dynamics and life histories.

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