A review on parasitic castration in veterinary parasitology

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Abstract

Internal and external parasites are the most common organisms present in the different animals including humans. Some of the parasites are specific to individual species while others may be transmissible to humans as zoonotic in nature. Impairment of the health condition of the animals leads to huge economic loss interns of productivity and reproduction. Parasitic diseases in livestock can be controlled by geographical location, type of host and their manage mental practices. Conventional methods of controlling parasites use synthetic chemotherapeutic drugs. In a safe manner, control of the animal parasitic can be done by the biological control which means maintenance of natural enemies which maintain a parasitic population at the lower level. Recently, parasitic castration has a role in the control of the parasitic diseases. In this, with help of different parasites, reproduction of the other parasites which are harmful to the animals can be reduced. This would be a case of direct parasitic castration by feeding on gonads of the hosts or indirectly, diverting the host energy from gonad development. Reproductive status and the body condition of the animals influenced by the different external and internal factors. Among the different factors temperature, photoperiod, availability of the food and space, individual animal habitat, predation behaviour and close association with other organisms as parasites which causes parasitic castration. One of the recent technique but, ignored by most of the population and community ecologist is parasitic castration. The present paper discussed the important role of parasites in the parasitic castration and importance in veterinary science in bri.

Keywords: Parasitic castration, strategy, gigantism, early, infection fecundity compensation

Introduction

Parasitic diseases are one of the important global problems and considered as major issues in milk-producing as well as meat producing animals. Most common economically parasitic diseases are caused by haemoproteozans, nematodes and trematodes in livestock [33]. Huge literature was available on different parasitic infections in livestock and importance of diseases including clinical signs, life cycle, pathological changes and treatment [32]. Most of the parasitic infections are obstacles in curtailing health and lowering productivity of animals. Many of the parasitic diseases caused by transmission by the vectors [29, 30]. Control of the parasitic infection can be done by using chemotherapeutic agents and reduction in the vector population [39]. Nowadays due to an indiscriminate utilization of the chemotherapeutic agents, parasitic resistance was noticed in the different hosts against the parasites [34]. Conditional factors of target therapeutic agents are providing the chance to select an alternative parasitic control programme. One of the new methods for controlling the parasitic diseases is the parasitic castration. Parasitic population can be influenced by the environment and host interactions with the parasites [39]. The term Parasitic castration was first introduced by Giard and Bonnier in 1887 and has attracted scientific attention ever since it was first reported by Maln in 1881 [11]. Parasitic castration is a phenomenon in which a parasite prevents its host from reproducing. In this procedure, host's gonad tissue altered by the parasites [27]. Baudoin (1975) stated as “a destruction or alteration of gonad tissue, reproductive behaviour, hormonal balance, or other modification that results in the reduction in host reproduction above and beyond that which results from non-selective use of host energy reserves by the parasite” [3]. Lafferty and Kuris defined parasitic castration as “an infectious strategy that requires the eventual intensity independent elimination of host reproduction as the primary means of acquiring energy” [20]. Some of the parasites specifically reduce the host reproductive function [5].
When parasites are consuming the nutrient-rich host reproductive tissue, it increases the own fecundity or survival. It is an alternative strategy for the evolution of virulence. Most of the protozoan parasites are found to castrate their vertebrate hosts. It may be either partially or fully. Common protozoan parasites include some ciliates, sporocysts, cestodes, mermithids, strepsipterans, dipterans and hymenopterans. Most of the times, parasites will do very minimum physical damage to the gonads. Always they will not destroy the host’s gonads, but can also be the indirect result of a nutritional drain or hormonal interference by the parasite. By this, the host may be temporarily too sick to reproduce or complete elimination of the host reproduction takes place. Parasitic castrators will take the time for the infection to mature to the point of complete castration. In few hosts, it is not permanent castration, the ability of the reproduction is as normal after the course of castrator parasites.

Parasitic castration
Most of the parasitic castrators directly affect the utilization of the energy resource and they stop hosts from spending any energy on reproduction. The non-castrating parasite may reduce the host reproductive output without actually targeting the energy that the host spends on reproduction. But, few non-castrating parasites, they will not have any effect on the host energy including reproduction energy. But they will reduce the overall growth of the hosts (Figure 1 and 2). Direct parasitic castration may be done by feeding on host gonads. During the indirect strategies, castrators diverting host energy from gonad development or secreting castrating hormones. Lafferty and Kuris explained that “when a host is infected by parasites that manipulate host behaviour, it might still look like a host, but it’s actually just a parasite vehicle”.[21]

Extended host phenotype
It is the phenotype of the infected host after the infection with castrator. Changes in the phenotype based on the castrator infection. During the extended host phenotypes, one common phenotypic change is host gigantism and the second change is called fecundity compensation.[19, 22]

1. Host Gigantism. Hosts that are parasitized by castrators should be bigger than un-parasitized hosts and hosts parasitized by non-castrating parasites.
2. Lifespan. Hosts that are parasitized by castrators should live longer than hosts parasitized by non-castrating parasites.
3. Fecundity compensation. Hosts that are parasitized by castrators should begin reproducing sooner than hosts parasitized by non-castrating parasites.
4. Reproductive rates. Hosts that are parasitized by castrators should have lower mean reproductive rates than hosts parasitized by non-castrating parasites because castrated hosts are sterilized, either partially or completely.
5. Parasite reproduction. Hosts that are parasitized by castrators should produce more new parasites than hosts parasitized by non-castrating parasites.

The host gigantism, the phenomenon commonly noticed in molluscs, crustacean, vertebrate and plant hosts and bacterial, fungal and helminth parasites.[21] Castration - gigantism combination occurs is most commonly observed in crustacean–microparasite and snail-trematode systems but also seen in plant–ant, fish–worm, and beetle–fungus pairs. Usually, the many snail–trematode castrator systems, hosts are relatively larger but the crustacean-microparasite cases, especially those with the zooplanktonic Daphnia as host; broaden that host size-parasite size perspective (Figure 3 and 4).[23, 31]

Different suitable examples for host gigantism are given in Table-1. During the “early-infection fecundity compensation”, after getting the infection, hosts reproduce earlier and/or produce more offspring. It was documented by the field and laboratory studies and it might be due to this response by the host could indicate enhanced investment in reproduction before maximal burden from castrators drops fecundity to zero.[14] Two recent studies with parasites of Daphnia highlight this fecundity compensation and even show variation in this response among host genotypes. Such variation perhaps indicates that parasite-mediated natural selection could act on this strategy.[6, 8].

Categories of castrators
Based on the castrator’s effect on the internal organs of the host, castrators can be divided into three groups[27].

1. Castrators which have a direct effect on a number of organs including the gonads (e.g., Metacercariae).
2. Castrators which have a direct effect on the gonads but have little effect on other organs (e.g., Rhopalura).
3. Parasites which have a little direct effect on the gonads but where there is a strong positive correlation between the presence of the parasite and a reduction of the gonads (e.g., Eoxenos, Hemiarthrus).

Mechanisms of Parasitic Castration
A. Parasites of insects often reduce host fecundity with minor or no effects on survival.

Reproductive effects range from complete castration (destruction of host reproductive tissues) to minor reductions in egg production and disruption of mating behaviour.[14, 21]

1. This is known to occur by altering the expression of a range of host neuropeptide genes
2. This occurs because of the susceptibility of neuro-endocrine-nerve-ganglion-gonad axis
3. Many parasites of insects regulate their host’s physiology by altering host endocrine function, effects on juvenile hormone and ecdysteroids.


Many microsporidians reside in specific host tissues, with the fat body (the site of egg yolk protein synthesis) a common target. Eugregarians (Protozoan, Phylum Apicomplexa) are a large family of castrating parasites found in insect’s guts and body cavities. Gregarians often invade the fat body, where they are likely to influence nutritional status and number of endocrine functions. Parasitic nematodes frequently reduce fecundity of insect hosts via effects on the fat body and/or corpora allata, implying endocrine disruption.

B. Parasitic castration (partial or total inhibition of host gamete formation by parasites)

Most commonly noticed in molluscs - trematode associations, in which larval trematodes (sporocysts, rediae and cercariae) infect the gonads of their molluscan hosts, finally leads to
partial or complete castration. It was recorded in several species of snails and bivalves and it was proved that the presence of larval trematodes disturbs gametogenesis in oysters during the annual reproductive cycle. The reduction in reproductive capacity caused by larval trematodes may affect the overall fecundity of the oyster and may increase the ability of larval trematodes to act as a regulator of its population. Bopyrids are known to cause parasitic castration, which involves two associated phenomena: gonads of female host do not mature and parasitized males are feminized [19, 15, 31]. The results vary depending on several factors including a). Host and parasitic interaction. b). Host age. c). The lifespan and typical reproductive pattern of the host and d). Whether the experiments were conducted in a laboratory or in the field [4].

One of the unique features of parasitic castrators is that they grow in larger size in accordance with their hosts by utilization of the available resources. In general, castration process persists for the throughout the life of the castrator host reproduction. But, it may be resumed after the death of the parasitic castrator. But in little literature, it reported later stages also for bioprids entoniscids and larval trematodes in snails [7]. Literature was available on how parasitic castration promotes the coevolutionary cycling but also imposes a cost on sex [1]. Different perspectives of the parasitic castration by models and sharing of the dynamic energies also mentioned in the earlier publication [13].

![Fig 1: Showing the utilization of the energy for body maintenance and reproductive maintenance by the host (reduction in the fertility) (Not a research finding – Graphical representation of theoretical concept)](image1)

![Fig 2: Showing the size of the hosts after getting the parasitism (giantism) (Not a research finding – Graphical representation of theoretical concept)](image2)

![Fig 3: Snail – Most common vector for transmission of the trematodes in livestock](image3)

![Fig 4: Trematode parasites in livestock – controlled by parasitic castration](image4)
Conclusion
Parasitic castration is a phenomenon can be viewed as a parasite’s adaptation and the castration effects on the hosts are beneficial to the parasite but not to the host and that advantages derived from this adaptation are a result of a reduction in host reproductive effort, which in turn gives rise to increased host survivorship, increased host growth and/or increased energy available to the parasite.

References


