Pharmaceutical Control of Endoparasitic Helminth Infections in Sheep

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KEYWORDS

• Anthelmintics • Endoparasites • Helminths
• Pharmaceutics • Sheep

Sheep are hosts to numerous genera and species of helminth parasites. For most of these parasites, a balance has evolved between them and their sheep hosts, whereby the host provides the environment and nutrients required by the parasitic population, while the parasite does not compromise the host to an extent that will threaten the survival of its future generations. Circumstances that upset this evolutionary balance can give rise to production-limiting disease, for example, when sheep are exposed to a previously unrecognized or new helminth species, as illustrated by the fact that the common large intestinal nematode species, *Oesophagostomum venulosum*, is not considered pathogenic, whereas the closely related, but rare species, *Oesophagostomum columbianum*, is highly pathogenic.[1] The balance between sheep hosts and helminth parasites has evolved over millions of years, but has been upset in relatively recent times by domestication and farming practices that favor the parasites by the inadvertent selection of more susceptible hosts or by the creation of environments that enable the establishment of larger populations of free-living stages of the parasites. This upset to the evolutionary host-parasite balance affects different parasite species to differing extents, enabling some nematode, trematode, or cestode species to be potentially production limiting while others are not.

Helminth parasites, such as *Haemonchus contortus, Bunostomum trigonocephalum*, and *Fasciola hepatica*, limit sheep production, due to the direct effects of their blood feeding behavior, while the pathogenic effects of most helminth species arise

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as a consequence of host innate and adaptive immune responses.[2] In fact, the parasites may have evolved to stimulate these responses in order to create the optimal environment for their own nutrition and survival, while differences in components of the host immune responses influence the extent to which the parasites limit production.[3] This has been shown by the demonstration of higher cumulative liveweight gains in corticosteroid-immunosuppressed sheep compared with control animals exposed to the same daily challenge of *Teladorsagia circumcincta* or *Trichostrongylus colubriformis*.[4,5] Furthermore, sheep that are naturally immune to gastrointestinal nematode parasites may suffer production loss despite harboring relatively low parasite burdens, whereas those that prioritize their protein resources toward survival, rather than immune responses that ensure maximal productivity, may harbor relatively high and epidemiologically relevant parasite burdens with relatively little effect on their productivity, and are resilient to the effects of helminth parasitism.[6,7]

The major production-limiting nematode parasite species affecting sheep in temperate climates are *Teladorsagia circumcincta*, *H contortus*, *Trichostrongylus vitrinus* or *Trichostrongylus colubriformis*, and *Nematodirus battus*. These parasites limit the productivity of susceptible animals because of their direct feeding activities that remove nutrients from the ingesta, and due to indirect effects on the immune response in their hosts, damaging the absorptive lining of the gastrointestinal tract or, in the case of *H contortus*, feeding on blood. The net pathophysiological effects of these activities are inefficient feed utilization, inducing a state of relative protein deficiency, fluid and electrolyte or macroelement imbalances, and anemia, leading to clinical signs such as reduced appetite, poor weight gains, diarrhea, and death.[8–10] Overall, the greatest economic importance of nematode parasites is suboptimal productivity arising from continuous low-level exposure to infective larvae.[11] Sheep can also be affected by several trematode parasites, in particular *F hepatica*, *Fasciola gigantica*, *Fascioloides magna*, *Dicrocoelium dendriticum*, and *Paramphistomum* spp. The feeding and migratory activities of these parasites are direct causes of production loss, because they remove blood and nutrients and cause tissue damage. Cestode parasites, such as *Taenia multiceps*, *Taenia hydatigena*, and *Taenia ovis*, or *Echinococcus granulosus*, for which sheep are intermediate hosts, cause disease through the development and space-occupying nature of their second-stage coenurus, cysticercus, or hydatid larval cysts, respectively. The cestode tapeworm parasite, *Monezia expansa*, which has sheep as its final host, passively absorbs nutrients from the intestinal digesta and has few, if any, adverse effects on productivity.[12] Helminth parasitism also causes production loss, due to the considerable cost incurred by its treatment and management.

Gastrointestinal helminth parasites are arguably the most important causes of suboptimal productivity in sheep, albeit that they often occur concurrently with other problems. The principal reason for keeping farmed sheep is to convert primary forage, herbage, or cereal crops into a marketable product. The efficiency of conversion of feed to meat is greater in sheep that achieve maximal growth rates than in ill-thrifty animals, because there is a daily feed requirement for maintenance that must be met before growth can occur, irrespective of the time taken to reach slaughter weight. Furthermore, sheep that are slow to finish are more susceptible to compounding effects of production-limiting diseases than rapidly growing animals, which may leave the farm before the main risk period for these problems. Therefore, the profitability of global sheep farming is heavily influenced by the efficiency of feed conversion to meat; control of gastrointestinal helminth parasites is a prerequisite for economically sustainable production.