ABSTRACT

Infection with gastrointestinal nematode parasites appears to be widespread in the cattle population. The pathologically important genera of worms all belong to the family Trichostrongylidae. The most pathogenic species is *Ostertagia ostertagi*, which can produce profound changes in abomasal mucosa of affected animals. These nematodes show developmental adaptations, such as seasonally arrested development, that are of prime importance to their survival and persistence on a year to year basis. Whereas nematode prevalence in cattle is high, their association with outbreaks of clinical disease is lower. This has focused attention on their so-called subclinical effects, particularly reduction of milk production in lactating cows. Evidence for this effect is still equivocal, but constant exposure to low infection apparently can depress milk production. Subclinical effects also have been demonstrated in young animals and include depressed rates of gain and interference in energy and nitrogen metabolism. Evidence suggests that these effects may be partly due to hypersensitivity reactions at the mucosal level in sensitized animals.

INTRODUCTION

Nematode parasites of cattle continue to be recognized as potential, widespread causes of disease in these animals. For example, in a recent survey in Maine (46) of 94 cows, 78 heifers, and 91 calves examined regularly over a year, 95.7%, 90.7%, and 96.7% were positive for strongyle nematode infection. These data were substantiated further in a subsequent experiment (35) where gastrointestinal tracts from 48 dairy heifers were examined over a year at the rate of eight animals every 2 mo. Strongyle nematode parasitism was in animals sampled during every period, and the overall incidence was 93.7%.

These high prevalences are fairly typical of findings in other parts of the country (11, 17, 19, 31) and raise interesting questions relative to the pathogenic significance of these infections, their economic impact, and the success of efforts directed at their control.

In this paper it is proposed to discuss some of the more recent information relative to epidemiology and pathogenesis of these infections that may have special importance for their eventual control.

DISCUSSION

Life Cycle Phenomena

Several genera of strongyle nematodes occur in the gastrointestinal tract of cattle. These are mainly members of the family Trichostrongylidae and include such genera as Haemonchus, Ostertagia, Cooperia, Nematodirus, and Trichostrongylus. By far the most pathogenic of these appears to be *Ostertagia ostertagi* (1, 2), which is in the abomasum. This parasite is not confined to North America but appears to be present and can cause problems wherever cattle are raised in the temperate zones. Its life cycle is direct (1) and is fairly typical of those of the other genera. Eggs are laid by the adult female worms and pass out in the feces. Outside the host, if environmental conditions are appropriate, they develop through two molts to an infective or third larval stage (L3). If the L3 are ingested by grazing cattle, further development takes place in the crypts of the gastric glands. After two further molts, to the fourth (L4) and fifth larval stages, the adult stage is reached. Completion of this life cycle in the host takes approximately 3 wk. However, at certain times of the year, depending on the geographical location, development in the host...
may be arrested at the L4 stage for up to 5 or 6 mo. This is the phenomenon known as hypo-
biosis (32) and has occurred with most of the
other genera of strongyle nematodes that
inhabit the gastrointestinal tract of cattle.

This phenomenon has important implications
relative to the epidemiology of these parasites.
It allows the parasites to synchronize or modify
their developmental and reproductive patterns
so that infective larvae are available at the most
appropriate times and locations for transfer
from one host to another to be accomplished
(21). Because these arrested larvae appear to be
metabolically less active than unarrested larvae,
they can accumulate in large numbers in the
host without producing a fraction of the
pathological effects of actively developing and
metabolizing worms (32). The stimuli for onset
of this phenomenon at first were thought to be
associated primarily with adverse environmental
changes, particularly low temperatures,
associated with late fall and winter in northern
regions (2). Recent studies, however, suggest
that these patterns of arrested development
differ geographically from north to south. In
the more northerly regions with severe winters,
large populations of parasites undergo arrested
development in late fall and winter (38, 46, 35,
31). On the contrary, in southern states with
milder winters the patterns seem to be different.
In California (6) arrested development has been
seen in both spring and fall, whereas in Louisiana
(43, 44, 45) peak arrest in development has
been in the spring with accumulation of large
numbers of arrested larvae over the summer
until August. This appears to facilitate per-
sistence of these infections over the hot dry
conditions of summer in the south, with
greatest availability of infective larvae during
the winter months of January and February.

Considerable speculation has been on
possible mechanisms underlying this phe-
nomenon of seasonally arrested larval develop-
ment (21). Most workers seem to agree that it
probably represents a "diapause phenomenon",
comparable to that in insects, and could be
triggered by a number of factors, both host and
environmental. Another important epidemi-
ological consideration with respect to this
phenomenon is the unreliability that it in-
troduces into assessment of parasitism based on
fecal worm egg counts. Because arrested larval
worms do not produce eggs, they may be in
large numbers in animals with absence of
external evidence in the form of high egg
counts. Also of importance for control of these
infections is that these arrested larvae appear to
be relatively unresponsive to most of the
anthelmintics available for treatment (2).

Pathologic Aspects

Large numbers of actively developing and
adult stages of these parasites in the gut of
cattle can cause severe pathophysiological
changes with accompanying clinical signs. It
seems that acute clinical disease is primarily a
disease of young animals, and there appears
to be a definite relationship between host age
and susceptibility. Young animals are more
prone to severe outbreaks of clinical disease
than older animals (1).

As previously mentioned, O. ostertagi is
probably the most pathogenic and economically
important of these worms, and the path-
ophysiological effects it produces are in many
respects similar to those with many of the other
species. Acute Ostertagiasis, or Type I O
stertagiasis, primarily affects young animals
grazing heavily contaminated pastures (1).
Calves introduced to such pastures can show
clinical effects within 3 wk of pasturing. In this
form of the disease the emergency of the L4
larvae from the gastric gland crypts 18 to 21
days after ingestion results in pathological
disruption of functional tissues responsible for
production of gastric juice. Parietal cells, which
produce hydrochloric acid, are replaced by
undifferentiated, nonsecretory cells. This causes
an increase in the gastric pH; pepsinogen is not
activated to pepsin and protein, therefore, is
not denatured; there is loss of bacteriostatic
effect and a consequent overgrowth of micro-
organisms. In addition, the increased per-
meability of the gastric mucosa leads to passage
of macromolecules into and out of the epi-
thelium. There is loss of albumin from the
plasma resulting in a hypoalbuminemia and an
elevation in pepsinogen of plasma. These
changes result in loss of appetite, profuse
diarrhea, weight loss, high morbidity, and often
mortality.

Other genera of these parasites produce their
effects by variations of this process. Trich-
strongylus produces similar effects to Ostertagia (37). Haemonchus is primarily a blood
sucker, causing an anemia (5). Nematodirus and Cooperia can cause a form of protein losing enteropathy because of their effects on the intestinal mucosal cells (30, 31).

In the northern parts of the country this type of disease most usually occurs from midsummer to the end of the grazing season when pastures tend to become heavily contaminated with infective larvae. In the more southern regions, outbreaks are less restricted and can occur over most of the year (26).

Animals that survive clinical outbreaks of disease apparently develop immunity by the end of the first grazing season. We have observed that this immunity will protect animals in the face of heavy pasture contamination that can lead to death in nonimmune animals. Furthermore, it apparently extends over to the subsequent grazing season (22).

Another acute form of ostertagiasis known as Type II also has been described (1). This type of disease results when the large numbers of arrested larvae in the gastric glands resume development. Disease results some 14 to 17 days after resumption of development and tends to be more severe than Type I because of the protracted damage by continuous development of the larvae. It mainly affects yearling animals as they are about to enter their second grazing season. In my experience, often only a few animals in a group are affected, but these frequently succumb to effects of disease.

In our studies on gastrointestinal parasitism in the northeastern United States and in eastern Canada I always have been impressed that even though prevalence is high, instances of clinical disease tend to be low. In one of our studies (46), we attempted to correlate farm management practices to the degree of parasitism in the young animals in our surveyed herds. Factors that were used for classifying the farm operations into good, fair, and poor, included pasture topography, housing, feeding practices, replacement policies, anthelmintic programs, and histories of recent parasitism. Those herds classified as poor had significantly ($P<.05$) higher parasitism in their young animals than those classified as having fair or good management. Predisposing factors, therefore, appear to be important in determining whether individual farms will be faced with clinical outbreaks of disease.

Because of the apparent low incidence of clinical outbreaks of disease in spite of the high prevalence of infection, great interest has been focused in recent years on any possible subclinical effects that might result from this low parasitism. This interest was stimulated greatly by the work of Todd and his coworkers (14, 15, 16, 17, 40) on effects of low mixed gastrointestinal nematode infections on lactation performance in mature dairy cows. Basically, these workers claimed that routine anthelmintic treatment of dairy cows at parturition and again at 60 to 90 days postcalving will result in a significant increase in milk production over the 305-day lactation period of 200 to 300 kg. A number of studies in various parts of the world attempted to substantiate or disprove the validity of these claims. Studies in Belgium (34), in Wisconsin (15), Vermont (16), North Carolina (40), and Pennsylvania (40) all have given positive results, but results have been negative in California (33), Florida (25), Maine (23), Mississippi (10), Texas (13), Australia, (7) and New Zealand (18).

Results conflict. Consideration of the published information shows that only limited statistical data have been presented. In general, examination of milk production data from these trials shows that high variation of yield despite careful pairing in some trials of treated and control cows. Variation in yield is similar when current yield is compared with that of the previous lactation. Yet another complicating factor is the difference between current and previous yields because of increasing age of the animals. These may be as much as 400 to 500 kg from first to third lactations. Because of this inherent variability, which is particularly evident for small numbers of animals, British workers have concluded that meaningful results can be obtained only by using a large population of animals for this type of study. Currently there is a large trial underway in the United Kingdom under the supervision of the Agricultural Development and Advisory Service (3). At this stage, therefore, the evidence on effects of subclinical parasitism on milk production still appears equivocal.

As Herd (26) has pointed out, in attempting to relate a milk production response to anthelmintic treatment it is important to know whether helminth parasites are capable of depressing milk production in clinically healthy
animals. So far, there has only been one study of direct effects of nematode parasitism on milk production in dairy cows. Bliss and Todd (17) found that administration of 200,000 trichostrongyle infective larvae over 1 to 3 days resulted in a reduction of 1 to 3 kg/day over the next 30 days. It could, however, be argued that this size of infective dose is not representative of the way that nematode infections normally are acquired. In a study recently completed, Barger and Gibbs (8) attempted to assess the effect on milk production of a mixed trichostrongyle infection acquired at a relatively constant low infection over an extended period. The milk production of six cows infected with 15,000 trichostrongyle infective larvae per week over 9 wk in early lactation was compared with milk production of six uninfected paired animals. After adjustment for differences in preinfection milk production by covariance analysis, the infected cows produced 2.16 kg/day less milk than the uninfected cows (P<.076). The infection rate of 15,000 larvae per week was based on an estimate of a cow consuming approximately 20 kg of dry matter daily while grazing a pasture containing 100 larvae/kg of dry matter. Such a pasture would be considered relatively clean but representative of pastures grazed by adult cattle. The egg counts produced by this infection were low, but variable and typical of those in previous studies on dairy cows. None of the infected cows showed any clinical evidence of infection, and we were confident that the infections in these cows were equivalent to subclinical infections usually found in dairy cows in this region. The uninfected group remained uninfected during the course of the study. No eggs were in their feces nor in any of the cows at calving; all cows had been confined to the barn since birth.

Bliss and Todd (16) and Grisi and Todd (24) claimed that transmission of trichostrongyle infections occurred readily in a barn environment, but no evidence of this was in the study. The detailed study by Herd et al. (28) suggests that trichostrongyle transmission within a barn is highly unlikely.

The small number of cows available for this study meant that the results did not reach acceptable statistical significance. However, a consistent depression of milk production in infected cows throughout the period of exposure to larvae suggests that the effect was real.

These results support those of Bliss and Todd (17) that subclinical infections can depress milk production by about 2 kg/day for a period of at least 9 wk if infection is continuous. It therefore appears that in situations where there is continuous exposure to reinfection, anthelmintic treatment would be justified economically if reinfection could be prevented.

The importance of subclinical effects of parasitism in younger animals has not received as much attention as have effects on milk production in the adult. Factors that might be expected to be influenced by subclinical parasitism in this age group would include rates of development, feed intake and utilization, weight gains, and long effects on lifetime productivity.

Cornwell et al. (20) demonstrated the impact of subclinical parasitism when they reported that growth rates in calves were increased significantly after deworming. In another study by Van Adrichem (41) a single anthelmintic treatment of 7-mo-old calves resulted in weight gains of 50.8 kg/animal over the nontreated controls, being greatest during the latter part of the 321 day trial. It was concluded that this indicated a long lasting harmful physiological effect from the moderate helminthiasis in these young calves. The long lasting effects of parasitism on weight gains suggested that these deleterious effects might extend over to affect subsequent milk production. A subsequent study (42) was conducted with monozygous twin heifers, beginning at less than 6 mo of age, in which gastrointestinal nematodiasis was maintained low by frequent anthelmintic treatments. A moderate natural infection was maintained for 1 yr in the nontreated animals. Twenty-three pairs of calves completed lactations. The treated animals produced significantly more milk, milk fat, and fat-corrected milk (FCM) than their controls, FCM amounting to an average of 191 kg or 6.58%. During the 1st yr of the study, both feed intake and feed utilization were improved in the treated animals. It was concluded that gastrointestinal nematodiasis of young cattle could have a relatively harmful, long effect upon growth performance as well as upon
subsequent milk production during their first lactation period.

Additional evidence of the importance of low infections in growing animals recently was given by Herd et al. (27) who showed that two strategic anthelmintic treatments given to heifers in spring and in summer effectively controlled parasitism as well as significantly increased weight gains over the grazing period.

In a recent experiment, Smith and Gibbs (39) studied effects of mixed gastrointestinal parasitism on hematological parameters and weight gains of three groups of yearling calves. One group served as an uninfected control, one group was exposed to contaminated pastures and treated at monthly intervals with an anthelmintic, the third group was exposed to contaminated pasture but left untreated. Weight gain studies showed that the uninfected group gained an average of 2.2 kg/wk as compared to 1.0 kg/wk for the treated group and a loss of .2 kg/wk for the untreated calves. Overall the control group gained 26.8 kg for the 12 wk of the trial, which was significantly higher than an 11.4 kg gain for the treated and a 2.3 kg loss for the untreated. Whereas anthelmintic treatment appeared to remove most of the adult worms from the treated animals, it did not appear to be effective in the face of constant pasture challenge in completely reversing the adverse effects of parasitism. Of particular interest in this respect were the changes in serum pepsinogen in the three groups of calves. These did not increase significantly in the parasitized groups (both treated and untreated) until the last 2 wk of the experiment. This result was similar to that of Armour et al. (4) and was probably due to the increased availability of infective larvae during the latter part of the grazing season. Armour et al. (4) suggested that this might be caused by an allergic reaction to ingested larvae which increased permeability of the mucosa to macromolecules like pepsinogen. Thus, an important part of the long-term pathogenic effects of parasitism (subclinical effects) may not be simply attributable to the presence and activities of adult worms but could be from the continuous challenge experienced by the grazing animals and the host’s reaction to that challenge. Additional support for this suggestion has been provided in studies by Barger and Southcott (9) on the effects of ovine trich-
be marked variations in the epidemiological behavior of various species of nematode depending on the particular geographical location in which they occur. It is thus important that epidemiological data be developed for the various geographical areas as there is an inherent problem in drawing generalized conclusions on the epidemiology of these infections based on limited regional data.

Although parasitism is widespread, reports of outbreaks of obvious clinical disease tend to be limited. Much of this condition, therefore, exists in the so-called subclinical forms. Much of the information that is available on the latter type of disease is still speculative, and even in instances where there are data, these are often contradictory. However, there is sufficient information available to suggest that this form of parasitism can represent a significant constraint on maximum productivity in both young and mature animals.

REFERENCES
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