Control of Gastrointestinal Parasites in First- and Second-Season Grazing Cattle in Sweden

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Abstract

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Gastrointestinal (GI) parasite infections are of major concern for productivity and health of first-season grazing (FSG) cattle in Sweden. Both GI nematodes and the coccidium *Eimeria alabamensis* overwinter on the pasture, which has major epidemiological implications. Clinical infections are seen as diarrhoea, anorexia and weight loss, while sub-clinical infections induce weight gain penalties that are easily overlooked. This thesis is based on grazing experiments that focus on the control of pasture-borne GI parasites in FSG cattle in Sweden, and the consequences of various levels of infection during their second grazing season (SSG cattle).

For 3 consecutive years, FSG cattle were allocated to 4 groups with 10 animals in each, and subjected to either 1) rotational grazing with a 2-paddock system, 2) nutritional supplementation in early season, 3) no treatment (minimum parasite control) and 4) regular treatment with anthelmintics (maximum parasite control). During the first grazing season, animal performance and parasitology were recorded, and pasture infectivity was investigated through tracer animals and pasture larval counts. The same 40 animals were monitored on communal pasturelands during their second grazing season. In addition, the effects of artificial single or concurrent infections with *E. alabamensis* and GI nematodes were assessed in a separate grazing experiment.

Rotational grazing for FSG cattle was successful, and the same aftermath paddock proved parasitologically safe by mid-July for consecutive years. In contrast, severe diarrhoea and weight loss were seen in supplementary-fed and untreated FSG cattle, which may be explained by significant numbers of overwintered GI nematode larvae and *E. alabamensis* oocysts in the paddocks at spring turn-out. In the study with SSG on communal pasturelands, the weight gain penalties induced in the first grazing season largely remained at the end of the second grazing season in 2 out of 3 years. In addition, the importance of lungworm infections was demonstrated in previously unexposed SSG cattle.

The experiment with artificial infections with *E. alabamensis* and GI nematodes showed no synergy between *E. alabamensis* and GI nematode infections, whereas dramatic clinical symptoms and production losses from *E. alabamensis* infections were seen.

Key words: *Eimeria alabamensis*; cattle-nematoda; interaction; epidemiology; pasture-infectivity; grazing; pasture-borne; control

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I will always remember the years at Ånhammar farm!

/Anna Larsson

Svensk sammanfattning

Kontroll av betesburna mag-tarmparasiter hos första- och andragångsbetande nötkreatur i Sverige

Mag-tarmparasiter förekommer i löpmage och tarm hos nästan alla förstagångsbetande nötkreatur i Sverige. Både parasitära maskar och koccidier (encelliga parasiter, protozoer) kan finnas i det gräs som kalvarna betar. Parasiterna övervintrar på betet vilket gör att ett bete kan vara hårt smittat på våren när kalvarna ska släppas ut, trots att vintern varit både snörik och kall. Nötkreatur som släpps på bete för första gången är mest känsliga för betesburna mag-tarmparasiter. Kraftigt infekterade djur insjuknar i parasitär gastroenterit (PGE) som kännetecknas av diarré, aptitlöshet och dålig tillväxt, medan lindrigare infektioner enbart ger nedsatt tillväxt hos djuren utan att kalvarna ser sjuka ut ens för ett tränat djuröga.

Eftersom mag-tarmparasiter förekommer i princip överallt där betande nötkreatur hålls, så måste de känsliga förstagångsbetande kalvarna skyddas på något sätt. Vanligast är att rutinmässigt avmaska kalvarna förebyggande, men detta är inte tillåtet i ekologisk produktion, varför alternativa metoder måste tillämpas istället.

Denna avhandling bygger på tre olika betesförsök under svenska förhållanden och syftar till att förbättra kunskapen om mag-tarmparasiternas skadeverkningar hos infekterade kalvar under deras första och andra betessäsong. För att åstadkomma detta måste kunskapen öka om hur smittan på betet varierar under en och samma betessäsong, och från det ena året till det andra. Med denna förbättrade kunskap är målsättningen att utvärdera och utveckla alternativa och praktiskt genomförbara kontrollstrategier där användningen av avmaskningsmedel så långt möjligt kan undvikas, men utan att djurens välfärd och lantbrukarens ekonomi påverkas negativt.

Treårigt betesförsök med förstagångsbetande kalvar

Ett treårigt betesförsök med 40 förstagångsbetande kastrerade tjurkalvar (stutar) i 4 olika grupper startade sommaren 2002 och upprepades under 2003 och 2004. Försöket utfördes i samma betesfållor för respektive grupp under de tre åren, men med 40 nya förstagångsbetande kalvar varje år. Syftet med försöket var att långsiktigt utvärdera antiparasitära effekter av olika betesstrategier. Följande strategier för parasitkontroll utan förebyggande avmaskning utvärderades: 1) betessläpp på marker som nyttjats av andragångsbetande nötkreatur föregående sensommar, följt av betesbyte till återväxtbete i mitten av sommaren (rotation) och 2) tillskottsutfodring på bete de första 4 veckorna efter betessläpp (foder). Som jämförelse följdes 2 kontrollgrupper, varav den ena gruppen avmaskades regelbundet under hela betesperioden (maximal parasitkontroll) och den andra var obehandlad (minimal parasitkontroll). Som komplement till studierna av de direkta

effekterna på djuren i de olika grupperna genomfördes analys av betessmittans storlek i de olika betesfållorna för försöksgrupperna.

Den första sommaren (2002) visade resultaten att parasittrycket var relativt lågt vid tidpunkten för betessläpp och endast lindriga infektioner med parasiter konstaterades under betessäsongen. Andra sommaren (2003) släpptes nya kalvar i samma fållor som året innan. Kraftiga koccidie-infektioner påvisades i utfodringsgruppen strax efter betessläpp och efter ytterligare några veckor konstaterades måttliga till kraftiga infektioner med mag-tarmmaskar i samtliga oavmaskade försöksgrupper. Djuren med de tydligaste symtomen återfanns i utfodringsgruppen trots att dessa utskiljde färre maskägg än andra grupper. Denna grupp, tillsammans med den obehandlade kontrollgruppen, hade en avsevärt sämre tillväxt under betessäsongen jämfört med den avmaskade kontrollgruppen där skillnaden i medelvikten per djur vid slutet av betessäsongen var ca 40 kg. Djuren i rotationsgruppen uppvisade inga tydliga symptom på parasitinfektion, trots att de i medeltal vägde 21 kg mindre per djur. Resultaten från det avslutande året (2004) visade att gruppen som flyttades till ett parasitfritt återväxtbete i mitten av juli till och med växte bättre än kalvarna som avmaskades. De obehandlade kalvarna på permanent bete, däremot, växte betydligt sämre än de övriga tre grupperna och förklaras av att kalvarna drabbades av kraftiga infektioner med både koccidier och mag-tarmmaskar.

Slutsatsen från försöket är att det är möjligt att förebygga parasitproblem utan att använda avmaskningsmedel. Nyckeln till detta är att kalvarna erbjuds ett relativt parasitfritt välkomstbete på våren och att de flyttas till ett likaledes parasitfritt bete i mitten på sommaren. I detta försök bestod höstbetet av en återväxt vilket förutom att det var parasitfritt, sannolikt även höll högre näringsmässig kvalitet eftersom dessa kalvar ibland till och med växte bättre än de kalvar som avmaskades. Tillskottsutfodringen misslyckades i just detta försök, men utesluter inte att andra sätt att tillämpa tillskottsutfodring i början av betessäsongen kan vara mer lyckosamma.

Treårigt betesförsök med andragångsbetande stutar

I försöket med andragångsbetande stutar användes helt enkelt samma djur som ingick i det treåriga försöket med förstagångsbetande kalvar ovan, men i detta försök betade samtliga djur gemensamt under hela betssäsongen. Försöket pågick således under åren 2003-2005. Den fråga många ställer sig är om olika nivåer på parasitsmittans storlek under kalvarnas första år påverkar immunitetsutvecklingen mot parasitinfektioner och därmed risken för att drabbas av parasitsjukdom och sämre tillväxt under den andra betessäsongen. Moderna avmaskningsmedel gör till exempel att djur som behandlas det första året ur parasitologisk synvinkel kan vara att betrakta som förstagångsbetande kalvar deras andra betessommar. Resultaten visade att ingen skillnad kunde ses i tillväxt hos djuren i de olika grupperna det andra året, men att de skillnader som uppstått under det första året bestod vid försökets slut. Dock var parasittrycket lågt samtliga tre år vilket naturligtvis inte utesluter att högre smittryck kan ge upphov till skillnader i tillväxt på grund av skillnader i motståndskraft mot parasitinfektioner. En intressant observation var att

nötkreaturens lungmask förekom hos de andragångsbetande djuren under 2004 och 2005 vilket kan förklara att tillväxten hos infekterade djur var dålig mot slutet av betessäsongen.

Samtidig infektion med beteskoccidier och mag-tarmmaskar

I ett separat betesförsök med 4 olika grupper av förstagångsbetande stutar genomfördes ett experiment med konstgjorda infektioner där bestämda infektionsdoser gavs som innehöll antingen beteskoccidier eller mag-tarmmaskar enbart, eller koccidier och maskar tillsammans. En grupp med oinfekterade kalvar fungerade som kontroll. Den frågeställning vi ville få svar på var om samtidig infektion med både koccidier och mag-tarmmaskar orsakar allvarligare symptom och sämre tillväxt än då infektionerna sker var för sig.

Resultaten visade att de kalvar som infekterades med koccidier drabbades av tydliga symptom på koccidie-infektion i form av vattnig diarré, upphörd foderlust och påverkat allmäntillstånd. Detta oberoende av om koccidierna gavs enbart eller tillsammans med mag-tarmmaskarna. De kalvar som enbart fick mag-tarmmaskar visade däremot inga symptom på parasitinfektion och växte lika bra som de oinfekterade kalvarna. Skillnaden i tillväxt vid försökets slut var ca 24 kg per kalv, en skillnad som uppstod redan under den första veckan av försöket och som alltså bestod under de resterande 9 veckorna av försöket. Slutsatsen från försöket är att beteskoccidier är mycket viktiga betesburna parasiter men att frågan om betydelsen av samtidig infektion med koccidier och mag-tarmmaskar inte gav något bra svar. Sannolikt var infektionsdosen mag-tarmmaskar för låg i detta försök.

Avslutningsvis ges i avhandlingen en konservativ definition på när ett bete under svenska förhållanden kan betraktas som ”parasitfritt”. För att ett välkomstbete till förstagångsbetande kalvar på våren ska betraktas som parasitfritt bör detta bete inte ha använts av obehandlade förstagångsbetande kalvar någon gång under det föregående året. Däremot kan ett återväxtbete i mitten av juli, efter en ensilage eller höskörd i början på sommaren, säkert betraktas som parasitfritt oavsett hur nedsmittat betet blev föregående år.

Contents

Appendix

Papers I-IV

This thesis is based on the following papers, which will be referred to by their Roman numerals:

I. Larsson, A., Dimander, S.-O., Rydzik, A., Uggla, A., Waller, P.J. & Höglund, J. 2006. A 3-year field evaluation of pasture rotation and supplementary feeding to control parasite infection in first-season grazing cattle – Effects on animal performance. *Veterinary Parasitology 142*, 197-206.

II. Larsson, A., Dimander, S.-O., Rydzik, A., Uggla, A., Waller, P.J. & Höglund, J. 2006. A 3-year field evaluation of pasture rotation and supplementary feeding to control parasite infection in first-season grazing cattle – Dynamics of pasture infectivity. *Veterinary Parasitology* (in press).

III. Larsson, A., Uggla, A., Waller, P.J. & Höglund, J. 2006. Performance of second-season grazing cattle subjected to different levels of parasite control during their first grazing season. (Manuscript).

IV. Larsson, A., Dimander, S.-O., Rydzik, A., Uggla, A., Waller, P.J. & Höglund, J. 2006. Effects of single or concurrent infections with *Eimeria alabamensis* and gastrointestinal nematodes on the performance of calves on pasture. *Parasitology Research 99*, 84-89.

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Abbreviations

AM	Aftermath group/paddock
BW	Body weight
C. oncophora	*Cooperia oncophora*
DM	Dry matter
DO	Doramectin-treated control group/paddock
E. alabamensis	*Eimeria alabamensis*
EL4	Early fourth-stage larva/-ae
Epg	Trichostrongylid nematode eggs per gram of faeces
FD	Supplementary-fed group/paddock
FSG	First-season grazing
GLM	General linear model
GI	Gastrointestinal
KRAV	Kontrollförening för ekologisk produktion (the Swedish certification body for organic production)
L3	Infective third-stage trichostrongylid nematode larva/-ae
O. ostertagi	*Ostertagia ostertagi*
Opg	Oocysts per gram of faeces
PGE	Parasitic gastroenteritis
SAS	Statistical analysis system
SLU	Sveriges lantbruksuniversitet (Swedish University of Agricultural Sciences)
SPC	Serum pepsinogen concentration
SSG	Second-season grazing
SVA	Statens Veterinärmedicinska Anstalt (National Veterinary Institute)
SWEPAR	Department of Parasitology, SVA/SLU
U	Units
UT	Untreated control group/paddock

Introduction

Pasture-borne gastrointestinal (GI) parasites are of major concern to the productivity and health of grazing cattle. Inadequately controlled parasite infections may cause severe symptoms such as watery diarrhoea, anorexia, weight loss and even death of affected animals, and should therefore be regarded as major animal welfare concerns. However, more common and of major economical impact are sub-clinical infections, which may cause significantly reduced performance of infected animals without obvious clinical symptoms.

The GI nematodes *Ostertagia ostertagi* and *Cooperia oncophora* can be found on virtually all cattle pastures. Even if these parasites often are associated with sub-clinical infections, they still have the potential to cause significantly reduced weight gain of infected first-season grazing (FSG) cattle, and thus may have a great economical impact (Corwin, 1997). Of additional interest among pasture-borne GI parasites is the intestinal protozoan *Eimeria alabamensis*, which has been identified as an important pathogen of FSG cattle (Svensson, Uggla & Pehrson, 1994). To date, both GI nematodes (Dimander, 2003) and *E. alabamensis* (Svensson, 1994) have been investigated separately under Swedish conditions. However, these parasites have to be acknowledged together, as they are both potential health hazards for young cattle turned out onto permanent pastures under temperate conditions. Although the present thesis has its primary focus on control of GI nematodes, *E. alabamensis* is recognised as a potential pathogen throughout the studies.

The parasites have the ability to survive from one grazing season to another, either on pasture or inside the host, which has major epidemiological implications. Thus, to achieve sound economic productivity, and avoid animal welfare concerns, cattle production systems need to control parasite infections. Control of nematode infections is often achieved by the use of anthelmintic drugs (Nansen, 1987). However, there is a growing trend toward use of environmentally sustainable agriculture systems, which has created an interest for non-chemical methods of parasite control that can replace, or minimise, traditional chemotherapy (Thamsborg, 2001). The forerunner for this change in agricultural production is the organic farming industry, which aims to develop sustainable and environmentally acceptable farming practices that also encapsulate high standards of animal welfare. The statutes developed by organic farming certification bodies (i.e. KRAV in Sweden) have quite rigid guidelines, particularly with regard to the use of synthetic compounds such as anthelmintics. This implies a potential threat to animal welfare unless alternatives to anthelmintics are developed (Thamsborg, 2001; Lund, 2002). Therefore, implementation of non-chemical approaches to control parasites of cattle is urgently required to assist organic cattle production. Accordingly, refined grazing management techniques along with other alternatives to chemotherapy must be further developed and evaluated.

Aims of the thesis

The objective of this series of studies was to evaluate rotational grazing and supplementary feeding as non-chemotherapeutic approaches to control gastrointestinal parasites, specifically *Ostertagia ostertagi*, *Cooperia oncophora* and *Eimeria alabamensis*, in FSG cattle. Further aims were to study the dynamics of pasture infectivity within and between grazing seasons, and to monitor animal parasitology and performance during the second grazing season on communal pasturelands. More specifically the aims of the studies were to:

- Evaluate non-chemical parasite control strategies for first-season grazing cattle subjected to the following managements: 1) turn-out on pastures grazed the previous season by second-season grazing cattle, followed by one move to aftermath in mid-season, and 2) nutritional supplementation during the early grazing season.
- Investigate the dynamics of the free-living larval stages of nematode parasites in relation to these treatments.
- Investigate the influence of these strategies and anthelmintic treatment during the first year on the ability of cattle to withstand parasite infections during their second grazing season.
- Investigate effects of concurrent or single artificial infections with the protozoan *Eimeria alabamensis* and the nematodes *Ostertagia ostertagi* and *Cooperia oncophora* on the performance of FSG cattle.

Background

Pasture-borne parasites of cattle

To the grazing animal, the inviting pasture is a combined dining area and lavatory, and may be the source of numerous internal parasites including nematodes, cestodes, trematodes and protozoa. However, it is beyond the scope of this thesis to cover them all. Instead, the focus of attention is on non-anthelmintic control of GI nematodes and the impact of *E. alabamensis* coccidiosis on FSG cattle shortly after turn-out. In addition, infection with the cattle lungworm *Dictyocaulus viviparus* had a clear impact during the course of study III, and it will therefore be covered briefly in this background. However, pasture-borne parasites not mentioned in this thesis may still be important to grazing cattle under different contexts and in other countries. The animals of primarily concern are first-season grazing (FSG) cattle that are turned out onto pasture without their dams.

Gastrointestinal nematodes

In temperate regions, the GI nematodes *O. ostertagi* and *C. oncophora* are considered to be among the economically most important internal parasites of FSG cattle (Corwin, 1997). Both *O. ostertagi* and *C. oncophora* belong to the superfamily Trichostrongyloidea of the phylum Nematoda. Other related genera of this superfamily that are found in the alimentary tract of cattle are *Nematodirus*, *Trichostrongylus* and *Haemonchus*. *Ostertagia ostertagi* and *C. oncophora* infect the abomasum and small intestine, respectively, and heavy infections may result in diarrhoea, anorexia and weight loss (Anderson*, et al.*, 1965). However, infections are often sub-clinical, which may result in significantly reduced weight gain without obvious signs of disease (Shaw*, et al.*, 1998b).

Life cycles

Ostertagia ostertagi and *C. oncophora* typically occur as mixed infections in grazing cattle. Their direct life cycles are illustrated in Fig. 1 and described in detail by Frankena (1987). The life cycle has a pre-parasitic (free-living) phase outside the host and a parasitic phase inside the host. The parasitic phase begins when infective third-stage larvae (L3) are ingested with herbage (1). These larvae travel to their predilection site (2) in the abomasum (*O. ostertagi*) and small intestine (*C. oncophora*), where the larvae moult and develop through the fourth (L4) and fifth (L5) larval stages, to become sexually mature adult worms that produces offspring (eggs). Parasite eggs from infected cattle are shed with the faeces onto the pasture (3). Each egg contains a first-stage larva (L1) that hatches, moults twice and develops to L3 within the faecal pat (4). The time for this development, and the proportion of developed L3, is variable and temperature dependent, but may be less than 10 days at 25 °C. Insignificant development takes place below 6 °C, and above 32 °C the mortality rate is high (Ciordia & Bizzel, 1963). When the development to L3 is completed, larvae migrate onto the surrounding herbage. This occurs under moist conditions that provide a water film

to assist infective larvae to make their way to the surrounding herbage (Rose, 1961).

The prepatent period (from ingestion of L3 to shedding of eggs) for both *O. ostertagi* and *C. oncophora* is approximately 3 weeks. However, completion to the adult stage inside the cattle may, under certain conditions, become delayed for several months due to larval inhibition or 'arrested development'. The reason for this is not fully understood, although evidence suggests that host immunity, population size and seasonal conditioning of infective larvae are important factors (Michel, Lancaster & Hong, 1978; Armour & Duncan, 1987; Eysker, 1997).

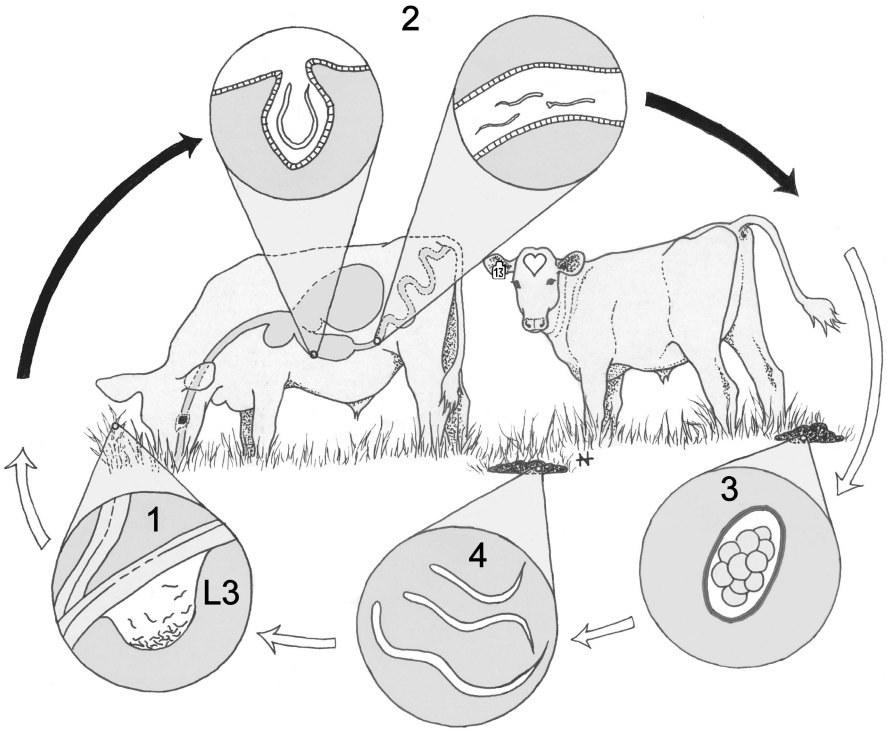

Fig. 1. The life cycles of *Ostertagia ostertagi* and *Cooperia oncophora*. For description of the life cycles, see the text. The filled arrows represent the parasitic part of the life cycle and the open arrows the pre-parasitic part. Drawing: Helena Nordenfors

Clinical picture

The clinical effects of GI nematodes depend on the parasite burden and the ability of the infected animal to withstand such infection (for review, see Fox, 1997). There is no multiplication of the parasitic stages following infection, which means that the larval uptake over time is crucial for the severity of disease. GI nematode infections are often associated with impaired productivity, such as reduced weight gain, that may be more or less pronounced. The outcome is defined as parasitic

gastroenteritis (PGE), and may be sub-clinical or clinical, where the distinction is not always straightforward. Sub-clinical PGE is characterised by reduced weight gain without clinical signs (Vercruysse & Claerebout, 2001), whereas clinical PGE is characterised by watery diarrhoea, reduced weight gain or weight loss, anorexia and a general loss of condition (Anderson*, et al.*, 1965). Reduced voluntary food intake is a common observation, and may be substantial (Fox*, et al.*, 1989; Forbes*, et al.*, 2000).

Infection with *O. ostertagi* has been categorised into ostertagiosis type I and type II (Anderson*, et al.*, 1965). The type I disease is usually seen in calves grazed intensively during their first grazing season, and is a result of ingested larvae beginning 3-4 weeks previously. This may occur approximately one month after turn-out to heavily contaminated pastures (Nansen*, et al.*, 1989), or from mid-season onwards when the second generation of larvae appears on pasture (Eysker*, et al.*, 1998a). The type II disease typically occurs in cattle after housing, usually in late winter or spring, following their first grazing season. This is considered to be a consequence of the maturation of larvae ingested during the previous grazing season, which subsequently became arrested in their development at the early fourth larval stage (EL4) (Armour & Bruce, 1974). Generally, type I ostertagiosis is characterised by high morbidity and low mortality, whereas type II ostertagiosis has the feature to cause low morbidity and high mortality (Urquhart*, et al.*, 1996).

Despite the frequent occurrence of *C. oncophora*, this parasite has been less discussed in favour of *O. ostertagi*. However, experimental single infections with *C. oncophora* have resulted in reduced weight gain in growing calves (Coop, Sykes & Angus, 1979; Armour*, et al.*, 1987), and Parkins*, et al.* (1990) observed an increased establishment of *O. ostertagi* in animals concurrently infected with *C. oncophora*.

Prevalence and epidemiology

Virtually all grazing cattle are infected with GI nematodes (Nilsson & Sorelius, 1973; Ploeger*, et al.*, 1990), and larvae of *O. ostertagi* and *C. oncophora* are found on most cattle pastures. Overwintering survival between grazing seasons is possible through predominantly the third larval stage, and has major implications for the development of clinical PGE in FSG calves (Nilsson & Sorelius, 1973). Instead, a low or moderate number of overwintering L3 that infect calves at turn-out may not cause disease, but develop to maturity and contaminate the pasture with eggs that are the source of a new generation of larvae later in the season. As previously mentioned, this development and timing for migration onto herbage is dependent on prevailing weather conditions. Under optimal conditions, this second generation of larvae infect the cattle from mid-season onwards, and if sufficient numbers are ingested, clinical outbreaks of PGE occur in the latter part of the grazing season (Anderson*, et al.*, 1969). However, the peak of second generation larvae may be delayed until the end of the grazing season, i.e. during dry conditions (Dimander*, et al.*, 2003), and cause problems the following spring (Dimander, Höglund & Waller, 1999).

FSG cattle are inexperienced to these infections and as such the most susceptible class of animals. Therefore, large amount of eggs are shed onto pastures by FSG cattle. Continuous use of the same pastures by susceptible FSG cattle increases the risk of heavy pasture contamination (Törnquist & Tolling, 1987). For young parasite-naïve cattle, it is therefore strongly recommended to avoid turn-out pastures that have been grazed by FSG cattle the previous year.

Acquired immunity against GI nematode infections affects the establishment, development, fecundity and survival of GI nematodes within the infected host, (for review, see Claerebout & Vercruysse, 2000), where decreased egg output is the first indication of a developing acquired immunity. The rate of development and the degree of immunity to GI nematodes vary according to the species of parasite and the period of exposure, and there is large variation between individuals in their susceptibility to parasite infections (Gasbarre, 1997; Gasbarre, Leighton & Sonstegard, 2001). Protective immunity against *O. ostertagi* is considered to develop slowly (Klesius, 1988), and although a reduction in faecal egg output may occur after some months of exposure, immune responses that reduce the actual number of established nematodes in the host are not evident until the second grazing season (Gasbarre, 1997). Protective immunity has been shown to develop more quickly against *C. oncophora* (Armour, 1989; Hilderson*, et al.*, 1995), and establishment is significantly reduced after 4 months of exposure (Hilderson*, et al.*, 1995) or at the end of the first grazing season (Gasbarre, Leighton & Sonstegard, 2001). However, even older cattle with a history of multiple grazing seasons may harbour low numbers of predominantly *O. ostertagi* (Agneessens*, et al.*, 2000; Claerebout & Vercruysse, 2000). Considering the large amount of faeces produced by older and larger animals, even animals with low faecal egg counts (FEC) may generate large numbers of eggs on pastures (Stromberg, 1997).

Eimeria alabamensis

Eimeria alabamensis is a coccidium that infects the nucleus of the epithelial cells of the lower part of the small intestine (Davis, Bowman & Boughton, 1957). This parasite was described already in 1941 (Christensen, 1941), but it has received far less research attention compared with GI nematodes. In contrast to the nematodes, coccidia are unicellular organisms that belong to the phylum protozoa. Twelve species of the genus *Eimeria* have been described in FSG cattle in Sweden (Svensson, 1994), although the majority of these are regarded as non-pathogenic. *Eimeria bovis* and *E. zuernii* are considered important pathogens to housed calves and young cattle, whereas *E. alabamensis* is a cause of early-season coccidiosis in grazing cattle, and was thoroughly investigated under Swedish conditions by Svensson (1994). Nowadays this coccidium has become recognised as an important pathogen in grazing calves shortly after turn-out (Svensson*, et al.*, 1993; Svensson, Uggla & Pehrson, 1994; von Samson-Himmelstjerna*, et al.*, 2006).

Life cycle

The life cycle of *Eimeria* spp. (Fig. 2) is direct, and it comprises both asexual (schizogony) and sexual (gametogony) multiplication that finally results in the

shedding of oocysts in faeces (Urquhart*, et al.*, 1996). Cattle turned out onto a contaminated pasture ingest sporulated oocysts together with herbage (1). Inside the host, the wall of the oocysts breaks, and the sporocysts infect epithelial cells in the intestinal tract (2). The following intracellular multiplication of the parasite results in destruction of the epithelial cells (3), and shedding of oocysts with faeces (4). The oocyst is the free-living stage of the parasite and must sporulate to become infective (5). Sporulation time is variable and dependent on temperature and humidity. At 25 °C sufficient sporulation may occur in 5 to 8 days, but it is limited below 15 °C and above 33 °C (Soekardono, 1975). The prepatent period of *E. alabamensis* is approximately 6 to 8 days (Hooshmand-Rad, Svensson & Uggla, 1994).

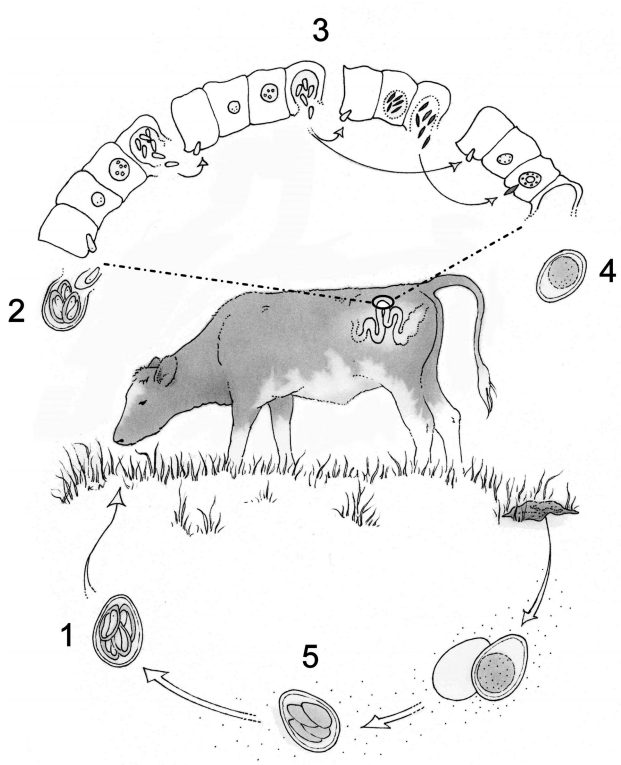

Fig. 2. The life cycle of *Eimeria alabamensis.* For more detailed description of the life cycle, see the text. Drawing: Katarina Näslund.

Clinical picture

Eimeria alabamensis infections may result in severe watery diarrhoea and total loss of appetite within a week after turn-out to pastures that have been grazed by FSG cattle for consecutive years (Svensson, Uggla & Pehrson, 1994). Heavy infections can result in rapid weight loss that is rarely compensated for even if infection is interrupted (Hooshmand-Rad, Svensson & Uggla, 1994), whereas infection remains sub-clinical under moderate infection pressure (Svensson*, et al.*, 1993). A distinct peak of oocysts in faeces appears after approximately 8 to 9 days in infected cattle, when millions of oocysts can be shed during a couple of days

(Svensson, Uggla & Pehrson, 1994). Typically, by this time the diarrhoea has become more porridge-like in consistency. Although these levels do not always correlate with infection dose and severity of symptoms, 850,000 oocysts per gram of faeces (opg) has been associated with clinical coccidiosis (Svensson, 1994). Although mortality due to *E. alabamensis* coccidiosis is low, deaths have been reported in heavily infected calves in Sweden (A. Uggla, pers. comm.).

Prevalence and epidemiology

The severity of *E. alabamensis* infections seems to be highly variable and difficult to predict. The prevalence of *Eimeria* spp. infection in cattle can reach 100%, but it may vary markedly in different age classes and between farms (Cornelissen*, et al.*, 1995). In a Dutch study of housed cattle, the prevalence of *Eimeria* oocysts was 46% in calves, 43% in yearlings and 15% in cows (Cornelissen*, et al.*, 1995). *E. alabamensis* was found on 12% of the farms, and, although *E. bovis* was the most abundant species, *E. alabamensis* was still found in 15% of the housed calves and in 6% of the yearlings. However, the situation with *E. alabamensis* infections is different among grazing cattle. In a Swedish study performed shortly after spring turn-out, *E. alabamensis* oocysts were found to have overwintered on 46% of the farms (Svensson, 1995). In another Swedish study, faecal specimens with more than 100,000 opg were found from calves sampled 8 to 10 days after turn-out on 7 out of 15 farms (Höglund, Svensson & Hessle, 2001), indicating extensive pasture contamination by *E. alabamensis*. Outbreaks with coccidiosis are accompanied by shedding of millions of oocysts, thus contaminating the environment considerably with oocysts. It was estimated by Svensson (1995) that FSG cattle on 20% of Swedish dairy farms are at risk of developing *E. alabamensis* coccidiosis. This is considered a consequence of using the same turn-out pastures for consecutive years, as the infective oocysts of *E. alabamensis* are capable to overwinter on pasture in all regions of Sweden (Svensson, 1995).

Detailed information about the development of immunity to *E. alabamensis* is limited. Most experiments with this parasite have focused on the first part of the grazing season, and less is known about the responses to subsequent infections with *E. alabamensis*. Young cattle that have been experimentally infected with high numbers of *E. alabamensis* have still developed symptoms of clinical coccidiosis when reinfected (Hooshmand-Rad, Svensson & Uggla, 1994). On the other hand, inoculation of calves with sporulated *E. alabamensis* oocysts 16 days before turn-out onto contaminated pasture protected the animals against clinical coccidiosis, and strongly reduced the shedding of oocysts (Svensson, Olofsson & Uggla, 1996). Further, under natural conditions Svensson (2000) concluded that reinfection with *E. alabamensis* after 17 days on pasture in the first grazing season was of little clinical importance. In addition, heifers that were naturally infected with *E. alabamensis* during their first grazing season appeared to be resistant to clinical coccidiosis during their second grazing season on contaminated pasture (Svensson, 2000).

Dictyocaulus viviparus

The lungworm of cattle, *D. viviparus*, also belongs to the superfamily Trichostrongyloidea, and is a pathogenic nematode of grazing cattle that deserves attention. The adult worms reside in the lungs, and may induce disease referred to as dictyocaulosis, husk, or bovine parasitic bronchitis (Eysker, 1994).

Life cycle

Similar to other nematode parasite infections of cattle, infective larvae (L3) are ingested during grazing. The L3 penetrate the intestinal wall and travels via the lymph and blood to the lungs, which are reached about one week after ingestion (Urquhart*, et al.*, 1996). In the bronchi of the lungs, the parasite reaches the adult stage and the female starts producing eggs. In contrast to the gastrointestinal nematodes, the female lungworm produces eggs that contain fully developed larvae, which hatch immediately after being laid. The newly released first-stage larva, still an L1, migrates up the trachea and is then swallowed, and finally passed with the faeces. In the faecal pat on pasture, L1 develops via L2 to the infective L3 stage and migrates onto the surrounding herbage, and thereby completes its life cycle when ingested by susceptible cattle.

Prevalence and epidemiology

In Sweden, like in other parts of northern Europe, approximately 40% of the farms are infected (Schnieder, Bellmer & Tenter, 1993; Ploeger*, et al.*, 2000; Höglund, Viring & Törnquist, 2004). Dictyocaulosis typically appears in non-immune young cattle during their first grazing season on permanent pastures (Eysker, 1994), but may also be of importance in previously uninfected older cattle. Further, older cattle often act as carrier animals, that maintain and spread the infection between grazing seasons, as small numbers of adult worms or arrested larval stages survive in the bronchi until the next grazing season (Saatkamp, Eysker & Verhoeff, 1994; Urquhart*, et al.*, 1996). In addition, L3 may survive on pasture over winter in sufficient numbers to initiate infections, and occasionally disease, in susceptible grazing animals. However, overwintering on pasture probably plays a minor role in transmission between grazing seasons, compared to carrier animals (Saatkamp, Eysker & Verhoeff, 1994). Outbreaks of disease typically occur 2-5 months following turnout, when a gradual increase of pasture contamination by infected animals results in sufficient numbers of larvae to cause clinical disease (Urquhart*, et al.*, 1996).

In contrast to the GI nematodes, *D. viviparus* induces a stronger immune response, associated with high levels of protection against reinfection (Gilleard, Duncan & Tait, 1995; Kooyman*, et al.*, 2006). However, low primary infections may result in a slow development of immunity that may be insufficient to protect cattle from lungworm disease after challenge infections (Eysker, Saatkamp & Kloosterman, 1993; Eysker, Kooyman & Ploeger, 2001). In general, some individuals always remain infected in afflicted herds (Höglund, 2006), and the immunity will also gradually disappear in the absence of larval challenge.

Lately, there have been reports of a shift towards older age classes of cattle that develop dictyocaulosis (McKeand, 2000). The reason for this shift is unknown, but it may be associated with extensive use of chemoprophylaxis in the first grazing season (Ploeger, 2002). Thus, a suppression of lungworm infections may negatively affect the build up of immunity against *D. viviparus* infections in young cattle. On the other hand, some degree of protection will still establish after a challenge infection, and lungworm-naïve animals that are exposed to small infections in the second grazing season may still be able to develop immunity naturally (Borgsteede*, et al.*, 1998). Further, although this interference of development of immunity in suppressed animals was concluded by Höglund, Gånheim & Alenius (2003), they still recommended treatment of infected calves to prevent further transmission of the parasite.

Control of GI nematodes in FSG cattle

Anthelmintics

Control of parasite infections in cattle in Scandinavia, and other developed countries of the world, has its foundation in anthelmintic drugs (Nansen, 1987; Svensson, Hessle & Höglund, 2000). Unless resistance has developed, anthelmintics effectively reduce pasture contamination by removing helminths inside the host. Thus, anthelmintics break the life cycle and prevent shedding of eggs onto pasture. Anthelmintics may be given as salvage treatments to clinically diseased animals, or as planned strategic treatment regimes for FSG cattle, based on knowledge about the epidemiology of the parasite infections in the local context. Contemporary anthelmintic treatments can be given through an intraruminal sustained release bolus (Borgsteede*, et al.*, 1990; Claerebout*, et al.*, 1997b; Borgsteede*, et al.*, 1998), or by single or repeated drenching with pour-on or injectable anthelmintics (Vercruysse, Hilderson & Claerebout, 1995; Vercruysse*, et al.*, 1995; Satrija*, et al.*, 1996; Epe*, et al.*, 1999).

However, prophylactic strategic control based on chemotherapy is not an issue for organic cattle producers, who instead are urged to implement other means for parasite control.

Non-anthelmintic control of nematodes

An essential component of an effective parasite-control program is knowledge about the dynamics of pasture infectivity, i.e. when parasite populations are likely to reach maximum numbers on pasture, and when ingested nematode larvae are induced to become hypobiotic (Stromberg & Averbeck, 1999). Although understanding these principles may seem relatively straightforward, access to detailed information is often difficult to obtain, and this may explain unexpected outcomes of various applied control methods that occur more or less irregularly. Understandably, as the pre-parasitic stages occur outside the host the combinations of environmental factors that act on these stages, such as temperature and humidity, make exposure levels to grazing animals difficult to predict at any given

time and place. Thus, control methods should be designed under the conditions that prevail locally.

Grazing management for parasite control is a concept that embraces different practices aimed to reduce, or prevent, exposure of susceptible cattle to high levels of infection on pasture, and the subsequent pasture contamination from infected animals (Thamsborg, Roepstorff & Larsen, 1999). Grazing management procedures can offer relatively simple and effective solutions to improve GI nematode control that can reduce the use of anthelmintics (Barger, 1997; Waller & Thamsborg, 2004). The purpose with parasite control systems is to keep populations of the infective stages as low as possible, especially to FSG cattle.

Michel (1985) categorized grazing management for nematode parasite control into preventive, evasive or diluting strategies:

Preventive strategies involve the availability of 'parasite-safe' pastures, i.e. pastures not contaminated by cattle parasites during the previous season, or aftermath pastures that have been mown. If such pastures are not available, prevention is instead obtained by suppressive use of anthelmintics, to eliminate pasture contamination until the initial exposure of L3 has decreased to a very low level.

Evasive strategies are to prevent cattle from grazing on contaminated pasture. Even a 'clean' pasture will become contaminated later in the season if grazed by FSG cattle. One example of an evasive strategy is to move grazing cattle to an aftermath before the appearance of a second generation of L3 on herbage (Henriksen*, et al.*, 1976; Dimander*, et al.*, 2003).

Dilution strategies make use of helminthologically inert animals of the same or different species for mixed or alternate grazing. The effect is achieved by reducing availability of existing infective larvae through ingestion of contaminated herbage by the helminthologically inert animals. Examples of dilution strategies are mixed grazing between FSG cattle and second-season grazing (SSG) cattle (Šarkūnas*, et al.*, 2000), and between sheep and cattle (Marley*, et al.*, 2006). However, Bairden, Armour & Duncan (1995) proved this method to be unsuccessful. Notably, the efficacy requires that the grazing alternations be related to the seasonal larval availability on pasture.

Rotational grazing

The term 'rotational grazing' is an evasive strategy that refers to moving cattle to new segments of pasture at regular intervals. The purpose may be to escape parasites before pasture infectivity reaches dangerous levels, or just to graze most of the forage to stimulate optimal re-growth of herbage. An intense rotational grazing strategy with a high stocking rate may force the animals to graze close to the faecal pats, which will increase the risk to be exposed to heavily infected grass (Williams & Bilkovich, 1973; Bransby, 1993). Further, a limited number of 'clean' paddocks in the system may increase the risk of return to previously grazed

paddocks, where pasture infectivity may be high. Parasite burdens have been found to increase in rotationally grazed cattle compared to continuously grazed cattle (Kunkel & Murphy, 1988). However, results are often conflicting, and good results based on clinical symptoms and weight gain have also been reported (Eysker*, et al.*, 1998b). Thus, the term rotational grazing embraces an infinite set of combinations, and any unique rotational strategy evaluated must be judged with sense.

Supplementary feeding

Dietary supplementation is considered to be a sustainable control strategy aimed at enhancing the natural ability of the host to cope with a parasitic challenge, but it may also substitute parts of herbage, which will reduce parasite exposure (Bransby, 1993). The positive effect of good host nutrition in ruminants on their ability to withstand GI nematode infections has been the subject for several reviews e.g. (Coop & Holmes, 1996; van Houtert & Sykes, 1996). Recently, Knox, Torres-Acosta & Aguilar-Caballero (2006) concluded that nutritional supplementation to small ruminants frequently increases resistance to nematode infection, which results in decreased faecal egg counts, and thus has the potential to reduce the needs for anthelmintic treatment.

Two terms are often used to describe the response of a host to parasitic infection, namely 'resistance' and 'resilience'. Resistance to parasitic infection is used to define the ability of an animal to prevent or limit establishment and maintenance of a parasite population in the GI tract, and is judged by a decreased faecal egg output by the infected host. Resilience describes the ability of an infected animal to maintain good production (i.e. weight gain) during a parasite challenge (van Houtert, 1997; Coop & Sykes, 2002). It has become increasingly clear in small ruminants that both resistance and resilience are significantly affected by, in particular, the protein nutrition of the host (Steel, 2003), possibly reflecting the considerable change in protein metabolism induced by infection with gastrointestinal nematodes (see review by Coop & Sykes, 2002). Protein supplementation seems to have a positive influence on the development of acquired immunity after a parasite challenge, which is manifested as reduced survival and decreased fecundity of an established parasite population (see review by Coop & Sykes, 2002). These effects seem to be most pronounced in young animals, which have the greatest demand for a high protein/energy ratio (Kambara*, et al.*, 1993). However, protein supplementation does not always have an effect on the establishment rate of the nematodes, even if egg output is reduced (Wallace*, et al.*, 1995).

Studies with grazing ruminants, where roughage is the main feed source, are limited. However, production losses have been significantly reduced when protein supplement has been offered to grazing sheep (van Houtert, Barger & Steel, 1995), and positive results have been reported on the clinical condition (Jørgensen*, et al.*, 1992) and animal performance (Jørgensen*, et al.*, 1992; Magaya*, et al.*, 2000) of supplementary-fed grazing cattle.

Despite limited information in this area, nutritional supplementation seems to be a fairly common strategy for parasite control among Swedish cattle farmers. In a questionnaire among organic farmers in Sweden, nearly 50% stated that they provided nutritional supplementation following turn-out in spring, with the intention to reduce parasite problems in FSG cattle (Svensson, Hessle & Höglund, 2000). However, the scheme for supplementation of their grazing ruminants, what quantity and quality of the supplement was used, how often it was used and when, was not revealed by the study.

Practical application can be complex in many production systems, and generalization may be difficult. In general, strategic supplementation should target those periods when nutrient requirements are greatest and supply those nutrients which are deficient, whether protein, energy, minerals or trace elements. However, nutrient requirements will differ at different stages of growth, with differing seasonal availability of forage, with different species of nematodes, and with different levels of exposure to parasites (Knox, Torres-Acosta & Aguilar-Caballero, 2006). Because of limited information about the effect of supplementary feeding on nematode infections in grazing ruminants, further studies within this area are suggested (van Houtert, 1997). Nevertheless, as a starting point, the provision of nutrients to optimise rumen function and animal performance in the particular production system should assist in maintaining resilience to nematode infections (Knox, Torres-Acosta & Aguilar-Caballero, 2006).

Methodological considerations

The research included in this thesis is based on 4 studies (I-IV) performed on a commercial cattle farm situated in south-central Sweden. Each year, approximately 250 male dairy breed calves were purchased for the farm, castrated and raised as steers to an age of 22-24 months. This provided an excellent opportunity to monitor performance and parasitology of the cattle under established management conditions, during their first- and second grazing seasons as well as the intermediate housing period (although these results are not included in the thesis).

Experimental design

First grazing season (studies I & II)

Studies I and II were designed to evaluate the effects of different parasite control strategies on performance of FSG cattle (study I), and on the dynamics of pasture infectivity (study II), during 3 consecutive years (2002-2004). Each year in May, 40 calves without previous grazing experience were assigned to 4 different treatments on naturally infected pasture in separate paddocks (Table 1). All paddocks were assigned a new set of experimental animals each year, although the different parasite-control strategies on each paddock remained the same. After the mid-summer move of one group (group RT) to aftermath grazing, their turn-out paddock was grazed by SSG cattle until housing each year. The other 3 groups remained on their paddocks until October, when all animals were housed. Animals in the supplementary-fed group received concentrate from the same batch as for the previous housing period, although half the amount, and hay *ad libitum*.

Table 1. Experimental groups and management of the first-season grazing animals.

Experimental group	Abbreviation	Management
Rotational grazing	RT	Spring turn-out onto pastures grazed the previous late season by SSG[a] cattle. Mid-July move of FSG[b] cattle to aftermath (AM) after 10 weeks on pasture
Supplementary feeding	FD	Supplementary-fed with concentrate 2 x 0.5 kg/calf and hay *ad libitum* daily for 4 weeks after turn-out, set stocked
Negative control	UT	Untreated control group, set stocked
Treated control	DO	Treated with doramectin (Dectomax® vet, Pfizer, Amboise, France) given as subcutaneous injection (10 mg/kg live weight) from turn-out and every 4 weeks throughout the grazing season, set stocked

All groups consisted of 10 FSG animals that grazed in separate 2 ha paddocks for approximately 20 weeks during 3 consecutive years.
[a] Second-season grazing cattle.
[b] First-season grazing cattle.

Second grazing season (study III)

Study III involved the same animals that were monitored during their first grazing season. Each year in May of 2003, 2004 and 2005, these 40 SSG cattle were assigned to a communal pastureland, and allowed to graze for approximately 20 weeks. The annual group of 40 SSG cattle was kept as a single group, and each year used 4 different pastures. Timing of the moves between pastures was decided *ad hoc* according to pasture availability.

Artificial infection of FSG cattle with nematodes and coccidia (study IV)

In study IV, performance and parasitological variables of artificially infected FSG cattle were compared between groups of animals infected with either coccidial oocysts (*E. alabamensis*) or GI nematode larvae (*O. ostertagi* and *C. oncophora*), or concurrently infected with both coccidia and GI nematodes. In June of 2004, 24 calves with no previous grazing experience were assigned to 4 different treatments, and inoculated with either 2 doses of 5 million sporulated *E. alabamensis* oocysts at turn-out, 90,000 L3 of *O. ostertagi* and *C. oncophora* divided into 6 occasions, or both oocysts and larvae as above. A control group was left uninoculated. All groups grazed on separate paddocks and were monitored for 10 weeks.

Animals

Male castrated calves (steers) of either Swedish Red and White or Holstein breeds, with no experience of grazing, were used in all experiments. The calves had been purchased from several dairy farms, and were kept indoors on the experimental farm for at least 2 months before each study. The age of the calves at the start of the studies was 5-9 months and, except in study IV, each animal was monitored continuously for two consecutive grazing seasons inclusive of the intermediate housing period.

Pasturelands

An improved pasture that had been established for 12 years, and previously grazed by anthelmintic-treated FSG cattle, was divided into 4 contiguous experimental paddocks of approximately 2 ha, and used throughout studies I and II. A similar-sized previously ungrazed paddock was used for aftermath grazing. From this paddock a silage cut was taken in June each year, and the aftermath was then grazed by the experimental cattle from mid-July. During the second grazing seasons (study III) all animals grazed on communal pasturelands, and were rotated between 4 different pastures (approximately 25 ha) depending on pasture availability. Before the start of study III, anthelmintic-treated FSG cattle had previously grazed these areas. In study IV, new leys were used to minimize adventitious infection with coccidia and nematodes. A uniform pasture was divided in 4 paddocks of approximately 1 ha each.

Parasite inocula (study IV)

The *E. alabamensis* oocysts that were used in study IV were harvested from faeces of calves collected 9 days after turn-out to naturally infected pasture. Later, a fresh stock of oocysts was obtained by passage through 2 calves that were inexperienced to grazing. On days 8 and 9 after inoculation, faeces were collected and the oocysts isolated and sporulated as described by Anonymous (1986). The species were identified based on morphology of sporulated oocysts (Anonymous, 1995), and maintained in 2% potassium dichromate at 6 °C before use. Before the oocysts were administered to the experimental calves, the potassium dichromate was removed from the oocysts by repeated washing and centrifugation.

Infective larvae of *Ostertagia* and *Cooperia* spp. were isolated from faeces collected from naturally infected calves. Sufficient numbers of larvae were obtained by passage through 4 calves that had never been on pasture. Larvae were harvested from faecal cultures (Anonymous, 1986) and identified according to Borgsteede & Hendriks (1974). The larvae were kept in small volumes of tap water in aerated flat-bottomed tissue culture flasks at 6 °C. Before administration, the larval suspension was poured into a conical glass and aerated to get a homogenous solution. Larvae were counted and individual doses prepared in 50 ml Falcon tubes.

Analyses

Clinical symptoms and weight gain

All animals were checked daily, and diarrhoea, reduced appetite, depression or any other visual abnormalities were recorded. In case of uncertainty about the severity of the clinical symptoms, the local veterinarian was consulted. If required, adequate treatment was given as stated in each paper. The animals were weighed prior to all studies, and then regularly on all sampling occasions throughout the experiments.

Faecal examinations

The numbers of trichostrongylid nematode eggs (epg) and *Eimeria* oocysts (opg) were determined by a McMaster method (Anonymous, 1986), based on 3 g of faeces and with a sensitivity of 50 epg/opg. To estimate proportions of trichostrongylid nematode genera, faecal cultures were prepared either individually or as pooled cultures from each experimental group. In either case, samples of 10 to 20 g of faeces from each animal were mixed with vermiculite and incubated for approximately 2 weeks at 26-27 °C. Third-stage infective larvae were harvested (Anonymous, 1986) and approximately 100 L3 were then differentiated to genus level by morphological identification according to Borgsteede & Hendriks (1974).

Blood samples

Blood samples were collected in vacutainer tubes, both without additive (studies I, III and IV) and with EDTA (study IV). Sera were prepared and stored at –20 °C until analysed for individual serum pepsinogen concentrations (SPC) according to Dorny & Vercruysse (1998). The diagnostic threshold for sub-clinical and clinical ostertagiosis was set to 3.6 and 5.0 U tyrosine, respectively, as a group mean (Hilderson*, et al.*, 1989). Serum antibodies to *Dictyocaulus viviparus* infection were determined in studies I and III, using the lungworm Ceditest-ELISA (ID-DLO, Lelystad, The Netherlands) as described by Cornelissen, Borgsteede & van Milligen (1997). Whenever seropositivity was ≥15% at housing, i.e. in relation to a control sample on each ELISA plate, all sera collected the same year were analysed retrospectively. EDTA blood samples were analysed individually for eosinophil leukocytes in study IV. This was performed at the Department of Clinical Chemistry, Swedish University of Agricultural Sciences, using Cell-Dyn 3500 with software for veterinary specimens (Abbott Laboratories, North Chicago, IL, USA).

Pasture larval counts (study II)

To assess the number of infective larvae on pasture (study II), replicate herbage samples were collected following two 'W-shaped' paths starting from opposite directions (Taylor, 1939), in each paddock. Pasture samplings were performed regularly, starting before turn-out in spring until approximately one month after housing in autumn. Infective larvae (L3) were then recovered by washing and sedimentation using a Baermann procedure (Persson, 1974). The infective larvae were enumerated and identified according to Borgsteede & Hendriks (1974), and expressed as amount L3/kg dry herbage. Notably, one disadvantage with this method is that there may be wide variation in pasture sampling and processing techniques, which implies difficulties in comparing results between laboratories and research studies (Couvillion, 1993; Eysker & Ploeger, 2000). Further, pasture sampling is only a spot-estimate of the amounts of larvae on pasture at a particular time.

Tracer worm counts (study II)

In addition to herbage samples, tracer animals were used to assess pasture infectivity (study II). Two tracer calves grazed each paddock along with the FSG cattle for 3 weeks after turn-out, and before housing. These calves had been raised indoors until allocated to the pastures, by which time they were 6-9 months old. Following the 3-week grazing period, they were housed for an additional 3 weeks, before transfer to the abattoir for slaughter and viscera collection. Following slaughter, the abomasum and approximately 10 m of the proximal small intestine were cut open. Bowel contents and washings of the mucosal surfaces were collected separately in individual buckets, and adjusted to a total volume of 4 l. To analyse the proportion of arrested inhibited larvae (EL4), the abomasal mucosa was scraped off and digested in a pepsin-HCl solution (10 g pepsin + 17 ml concentrated HCl dissolved in 1 l water) and the final volume adjusted to 2 l. Four

20 ml sub-samples were taken from each of the contents and stored at –20 °C until analysed. After thawing, worm counts of 2-3 sub-samples from each organ were examined. Nematodes were stained with Lugols iodine, counted, and identified to genus using the keys by Barth & Visser (1991).

Compared to herbage samples, the tracer technique provides a total acquisition of L3 over a defined period of time, and the subsequent establishment of these nematodes in the host (Waller*, et al.*, 1981). In addition, the tracer technique makes it possible to determine arrested development of the nematodes. One drawback with tracer animals may be the general variation in intake and establishment of nematodes between individuals, and to overcome this a fairly large number of tracer calves would be required to obtain representative worm counts (Eysker & Ploeger, 2000).

Herbage availability (study I)

Herbage availability (sward height) was estimated by using a Massey grass meter (Holmes, 1974). Recordings were made following a 'W-shaped' path, with at least 60 readings per paddock every 2-3 weeks, starting 3 weeks after turn-out in 2002 and at turn-out in 2003 and 2004.

Meteorological data

Daily precipitation and temperatures were recorded at a meteorological station located 14 km from the experimental area. These data were presented as monthly means, and expressed in relation to the long-term (1961-1990) averages (LTA).

Statistics

Data were summarised using Microsoft Excel® 2000, and analysed using the statistical analysis system (SAS) version 9.1 (SAS Institute Inc., NC, USA) or by using Intercooled Stata® version 8.2 (Stata Corporation, College Station, TX, USA). In study I, differences in epg, opg, weight gain and pepsinogen were calculated by repeated measures analysis of variance using the Mixed Model procedure with SAS. In study III and IV, cumulative epg and opg values were calculated and log (x+1) transformed, and then analysed using the GLM procedure in SAS or Student's t-test. Pepsinogen and weight gain trajectories were analysed separately with repeated measures multivariate analysis of variance (MANOVA). In all experiments, opg and epg were presented as geometric means, whereas pepsinogen and weight gain were presented as arithmetic means, and the significance level was set to $P<0.05$.

Results

Rotational grazing and nutritional supplementation to FSG cattle (studies I & II)

Clinical observations and performance (study I)

The rotational grazing strategy (RT) overall resulted in healthy cattle without clinical symptoms of parasite infections. The average daily weight gain in this group ranged between 0.82 and 0.91 kg during the 3-year study, which is comparable to the doramectin-treated group (DO), with a daily weight gain of between 0.80 and 1.0 kg (Fig. 3). In contrast, the supplementary-fed animals (FD) performed poorly the second year (2003), and some animals showed clinical symptoms of PGE, including diarrhoea and reduced weight gain, early in the season. Accordingly, group FD were salvage treated with doramectin 10 mg/50 kg BW (Dectomax® vet., Pfizer, Amboise, France), 7 weeks after turn-out. In contrast in 2004, group FD were healthy throughout the season and performed as well as group DO. However, this year PGE were observed in group UT. The daily concentrate ration of 2 x 0.5 kg/calf offered to the supplementary-fed group was readily consumed on each feeding occasion, and the hay consumption was estimated as a daily intake between 0.5 and 0.75 kg/calf.

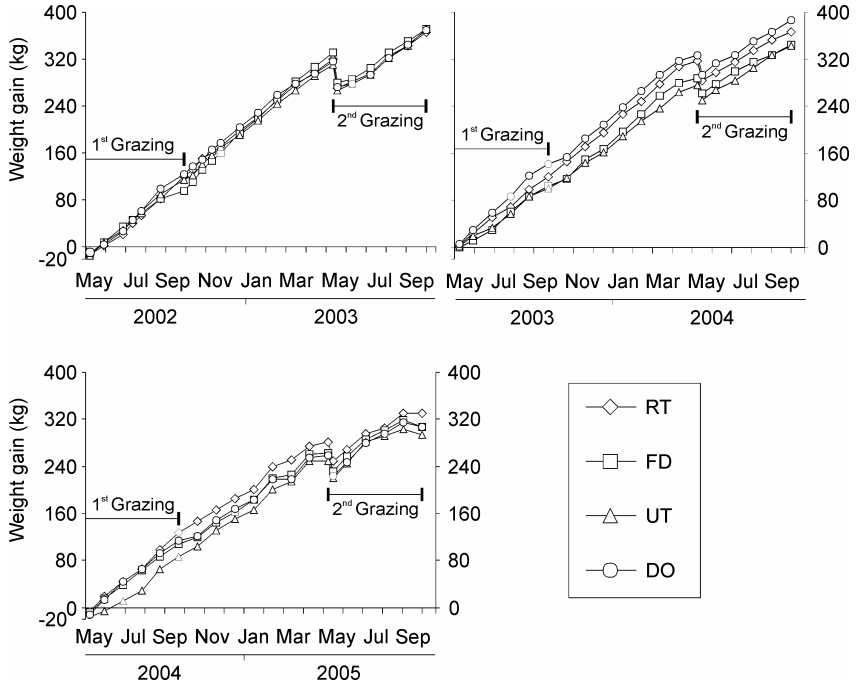

Fig. 3. Mean weight gains (kg) for the experimental animals during the first grazing season on 4 separate paddock, the following housing period and finally the second grazing season on communal pasturelands. Rotational grazing (RT), supplementary feeding (FD), untreated control (UT) and doramectin treated control (DO) indicate the treatments during the first grazing season.

Parasitology

A significant increase in oocyst output of mainly *E. alabamensis* was observed 9 days after turn-out. Individual samples up to 100,000 opg were observed in all four experimental groups, in all years. The highest mean numbers were observed in group FD in 2002 and 2003, and in group UT in 2004, with individual values ranging between 500,000 and 2 million opg. Faecal samples taken 4 weeks after turn-out showed geometric mean values of <1500 opg in all years. Eighty-nine percent of all faecal samples from non-anthelmintic-treated FSG cattle analysed between week 4 and 10 had detectable levels of trichostrongylid eggs, compared to 50% between weeks 12 and 20. Peak values were generally observed 4 weeks after turn-out (study I). In general, faecal egg counts (FEC) decreased following these early-season peaks, and mean values <100 epg were recorded by August. No significant differences in FEC were observed between the non-anthelmintic-treated groups, except in 2003 when a geometric mean of only 82 epg was observed in group FD (arithmetic mean was 495 epg). Faecal cultures showed that the most abundant species were *Cooperia* followed by *Ostertagia* spp. and only a minor proportion of *Nematodirus* spp.

In 2002, SPC increased to 3.6 U tyrosine (sub-clinical levels) in groups FD and UT towards the end of the season. In contrast, during 2003 and 2004, SPC in these groups increased within 8 weeks to levels >5 U tyrosine, followed by a decrease towards mid-July. Mean SPC in group RT increased to 3.5 U tyrosine 8 weeks after turn-out in 2004.

Dynamics and overwintering of infective nematode larvae

Pasture larval counts

Overall, the pattern of L3 in herbage samples from paddocks FD and UT showed moderate to very high levels in April and May, comparatively low levels between early June and August, and eventually moderate to high levels in September and October. On paddock RT, only low to moderate levels were observed at times when animals were kept grazing. Although extremely high values (up to 17,000 L3/kg dry herbage) were observed on the aftermath paddock (AM) in April, values were <200 L3/kg dry herbage by July. The observed high levels of infective larvae on paddocks FD, UT and AM in spring before turn-out suggest that the overwintering capacity of infective larvae on pasture was substantial.

Tracer worm counts

The principal genera observed in the tracer animals were *Cooperia* followed by *Ostertagia* (Fig. 1, study II). In general, tracers grazing on paddock RT harboured <30,000 nematodes in May and between 18,000 and 100,000 in September. In contrast, tracers on paddock FD harboured approximately 65,000 nematodes in May 2003 but on average 30,000 at hosing the same year. In 2004 the pattern was the opposite, with approximately 25,000 nematodes in May and more than 90,000 in September-tracers. Although, tracers on paddock AM harboured up to 83,000 nematodes at housing, values were less than 4000 in mid-July, after a silage cut in early summer. Virtually all tracer animals grazing in September harboured

inhibited EL4 of both *Ostertagia* and *Cooperia* spp. With tracers on the DO paddock excluded, generally 30 to 50% of the *Ostertagia* population and 64 to 78% of the *Cooperia* population were found as EL4. In contrast, only sporadic EL4 were found in the May-tracers.

Communal grazing during the second grazing season (study III)

Clinical observations and performance

The typical clinical signs of coughing associated with dictyocaulosis were obvious in the majority of the SSG animals from late August both in 2004 and 2005, but not in 2003. No other symptoms of GI-parasite disease were observed, although intermittent diarrhoea was present in the group throughout the grazing periods.

The mean weight gains during 2003, 2004 and 2005 are shown in Fig. 3. No significant difference in weight gain that could be related to the previous years parasite exposure was observed during the second grazing season on communal pasturelands. A dramatic weight loss of between 30 and 47 kg was observed for the first 10 days on pasture. This was followed by a daily average weight gain of 0.74, 0.66 and 0.59 kg in 2003, 2004 and 2005, respectively. The differences in live weights in 2004 and 2005 resulted from the previous years grazing seasons, although some convergence was observed during housing and the second grazing season. In 2005, the weight gain trajectories started to level out towards the end of the grazing season, and at week 16 the SSG cattle started to loose weight. These weight gain penalties coincided with lungworm infections in the majority of the animals.

Parasitology

Approximately 83% of the faecal samples analysed during the 3-year period had <50 trichostrongylid epg, and only 4% had values >100 epg. Cumulative epg increased between years and were significantly higher in 2005 compared to in 2003 ($P<0.05$). Cumulative epg were also significantly higher in group DO compared to other groups ($P<0.01$). The faecal oocyst counts during the second grazing season were of less significance compared to the first grazing season. The highest values were observed 10 days after turn-out in 2005, when 10 (25%) of the 40 SSG cattle excreted >10,000 opg, with the majority being *E. alabamensis* oocysts.

Elevated antibody levels to *D. viviparus* were detected only in 2004 and 2005, when a total of 64% and 83% of the animals were found seropositive. Seropositive animals were observed from week 8 in 2004 and from week 4 in 2005, and both seroprevalence and seropositivity gradually increased during the grazing seasons. In general, there was a slight increase in SPC for the first half of the grazing season, but there were no significant differences between former groups.

Artificial infection with coccidia and GI nematodes (study IV)

Clinical observations and performance

Four to five days after the first inoculation with coccidia, most calves developed more or less profuse and watery diarrhoea that lasted for 3-6 days. Their appetite was significantly reduced, and a few of the calves were weak and reluctant to rise. Following this first 1.5 weeks on pasture, only intermittent diarrhoea was seen. In non-infected control calves and in those calves only infected with nematodes, softening of the faeces was observed during the first week on pasture but their clinical condition was not affected. Differences in weight gains were observed after 1 week on pasture. After the 10-week study period, weight gains were still reduced by approximately 24 kg for animals in coccidia-infected groups, compared to groups that were non-infected or infected with nematodes only ($P<0.01$).

Faecal examinations and blood analysis

A marked increase in oocyst output in faeces from coccidia-infected animals was observed 8 to 9 days after the first inoculation. Geometric mean values were >1 million opg, and the highest individual value 21 million opg. By day 11 after inoculation, values had fallen to below 500,000 opg, and 14 days after the *E. alabamensis* inoculation opg values were less than 1200. There was no statistical difference in the peak mean excretion between the groups receiving only coccidia or coccidia plus GI nematode larvae. Virtually all oocysts excreted during days 8 to 11 were *E. alabamensis*. Trichostrongylid eggs in faeces were observed in nematode-infected calves 21 days after the first inoculation with GI nematode larvae. The geometric mean number of eggs never exceeded 300 epg, and followed a similar fluctuating pattern between animals infected with nematodes only and nematode plus coccidia. Mean serum pepsinogen levels were up to 3.8 U tyrosine in nematode infected groups. In groups that were non-infected or infected with coccidia only, mean serum pepsinogen levels never exceeded 1.9 U tyrosine.

Discussion

The first grazing season

Eimeria alabamensis infection

The impact of *E. alabamensis* coccidiosis in FSG cattle was obvious during the course of both studies I and IV. Study IV showed that most calves that were artificially infected with *E. alabamensis*, alone or concurrently with GI nematodes, developed severe watery diarrhoea 4-5 days after the first inoculation. This, in combination with some days of reduced appetite, or even total inappetence, significantly impaired performance of the young grazing calves. Despite the relatively quick clinical recovery of most calves, the initial weight loss was substantial and remained uncompensated even after another 9 weeks on pasture.

Although the calves in study IV were artificially inoculated, and it may be argued that the infection dose was unnaturally high, the importance of *E. alabamensis* as a pathogen in naturally infected FSG cattle has previously been shown (Svensson, Uggla & Pehrson, 1994; von Samson-Himmelstjerna*, et al.*, 2006).

Study I was performed on naturally infected pastures, and the impact of *E. alabamensis* infection was again evident in the animals that excreted high numbers of oocysts 9 days after turn-out. These animals suffered from weight losses of up to 49 kg already 4 weeks after turn-out, and, although they later showed signs of nematode PGE, it is likely that the spell of coccidiosis soon after turn-out contributed strongly to the weight loss. This is in accordance with Svensson, Uggla & Pehrson (1994) who observed weight losses of 18 kg 24 days following turn-out. The actual infection dose of *E. alabamensis* oocysts on naturally contaminated pastures is difficult to estimate. However, oocyst excretion of 2 million opg in FSG cattle in study I clearly indicated that the numbers of overwintered *E. alabamensis* oocysts on the permanent pastures was high. Similar situations have previously been reported in Sweden (Svensson, 1995), UK (Marshall*, et al.*, 1998) and Germany (von Samson-Himmelstjerna*, et al.*, 2006).

The severity of clinical *E. alabamensis* infections in study I was unpredictable between years, although this was not a surprise since problems with clinical coccidiosis shortly after turn-out had previously been experienced on the farm. Not all farms in Sweden seem to experience problems with clinical coccidiosis in their grazing calves. A previous Swedish survey showed that 34% of farms that had used the same turn-out pasture for 5 consecutive years for FSG cattle had calves that excreted more than 850,000 oocysts (Svensson, 1995). In contrast, in another field survey without the criteria of permanent pastures none of the 15 farms included had experienced clinical coccidiosis (Höglund, Svensson & Hessle, 2001). However, economical losses due to subclinical disease may still be substantial, and in fact exceed those resulting from clinical coccidiosis (Fitzgerald, 1980).

GI nematode infection

Clinical observations of PGE, including reduced weight gain and diarrhoea, were observed among FSG cattle on permanently grazed paddocks within the first 2 months of grazing both in 2003 (group FD) and in 2004 (group UT). In 2003, this was a result of only one previous grazing season with untreated FSG cattle on lightly contaminated pastures. Unquestionably, the massive numbers of overwintered larvae on pasture caused these symptoms, described as early-season type I trichostrongylosis (Nansen*, et al.*, 1989). These symptoms were associated with an increase in FEC after 4 weeks grazing, followed by a rapid decline, as well as increased serum pepsinogen concentration to clinical levels within 8 weeks (study I). This is in agreement with Kristensen*, et al.* (2006) who found the highest FEC in the first part of the season, but in contrast found the highest pepsinogen levels in the later part. This demonstrates that diagnosis of FEC and pepsinogen later in the season may be misleading (Kristensen*, et al.*, 2006).

Early-season PGE may be mitigated by a delayed turn-out until mid-June, due to the rapidly declining numbers of L3 on pasture in spring (study II) (Nansen*, et al.*, 1987), and may be especially important if the calves are less than 6 months old (Shaw*, et al.*, 1998a). The pronounced capacity of infective larvae to survive the Swedish winter and to induce parasitic disease early in the season, has previously been observed in Scandinavia (Nilsson & Sorelius, 1973; Tharaldsen, 1976; Oksanen & Nikander, 1981; Dimander*, et al.*, 2003). In contrast, in central and western parts of Europe the general pattern among non-anthelmintic-treated FSG cattle is a peak in FEC approximately 2 months after turn-out, which seems correlated with the appearance of PGE later in season (Shaw*, et al.*, 1998b).

Concurrent infections with E. alabamensis and GI nematodes

Under natural conditions, parasite-naïve cattle will often be exposed to both coccidia and GI-nematodes. Early-season PGE may therefore be caused by a combination of both infections. In study I, it was observed that the groups with the highest mean opg values on day 9 also developed PGE early in the grazing season in 2003 in group FD and in 2004 in group UT. Based on these observations it was decided to investigate the outcome of single or concurrent infections with *E. alabamensis* and GI nematodes in a more controlled experiment (study IV). Groups of FSG cattle in study IV were artificially infected with either *E. alabamensis* or GI nematodes alone or concurrently.

No evidence of synergy or additive effect of concurrent infections was observed, either on performance, parasitology or on peripheral eosinophils. In sheep Catchpole & Harris (1989) confirmed synergistic effects when they infected lambs concurrently with the coccidia *E. crandallis* and *E. ovinoidalis*, and the intestinal nematode *Nematodirus battus*. Lambs that were infected with both groups of parasites showed impaired weight gain and a higher FEC of *N. battus* compared to single-infected lambs, but no effect on oocyst production (Catchpole & Harris, 1989). In rats, Bristol, Pinon & Mayberry (1983) observed an increased egg production and a delay in the rejection of the nematode *Nippostrongylus brasiliensis* during a concurrent infection with *E. nieschulzi*. This was associated with a suppression of the normal immune response to *N. brasiliensis* (Bristol, Pinon & Mayberry, 1983). Later, Upton*, et al.* (1987) demonstrated a suppression of eosinophil mobilization from the bone marrow, and a delay in self-cure during concurrent infections with *E. nieschulzi* and *N. brasiliensis*. Thus, different species of parasites may be capable of altering the host's immune response.

However, study IV was not designed to study the immune response of the infected animals, but rather to focus on parasitological and clinical effects. Due to the patent effect of *E. alabamensis* on weight gains of FSG cattle, it seems reasonable to assume that grazing cattle recovering from a preceding coccidial infection may be more susceptible to infection with GI nematodes at levels higher than those used in study IV. More studies are therefore needed on the consequences of concurrent infections with *E. alabamensis* and other GI parasites.

Overwintering and seasonal dynamics of GI nematodes

As already indicated, overwintering of infective larvae is regarded as a key epidemiological factor of nematode parasitism in FSG cattle in Sweden, and was confirmed also in study I and II. However, elevated levels of infective larvae derived from within-season contamination (second generation) were not observed until early September or after housing in October. This is in agreement with previous findings from Sweden (Nilsson & Sorelius, 1973; Dimander*, et al.*, 2003).

Once the infective larvae have migrated from the faecal pat onto the surrounding herbage they are exposed to external weather, unless protected by vegetation or ingested by grazing cattle. Thus, the ecology of the pre-parasitic stages of GI nematodes is largely influenced by the prevailing climate and weather conditions (for review, see Stromberg, 1997). A combination of heavy rainfall followed by a prolonged period with drought may promote development and migration of infective larvae onto the surrounding herbage, followed by exposure to sunlight and low humidity that effectively reduces the numbers of larvae.

The high numbers of overwintered infective larvae observed in spring 2003 contrasted with the moderate numbers observed on the same paddocks in September 2002. The very dry conditions in August 2002 probably prevented breakdown of the dung pats deposited on the pasture, which protected the developing larval stages from detrimental external factors, such as direct sunlight, and prevented desiccation. In this scenario, infective larvae accumulate and the total number of overwintering larvae may be high even if FEC of the animals that have contaminated the pasture is low. Large numbers of overwintered infective larvae following extended periods of dry weather have previously been observed both in Sweden (Dimander*, et al.*, 2003) and Denmark (Nansen*, et al.*, 1989), as well as in the UK (Taylor*, et al.*, 1973) and Australia (Barger, Lewis & Brown, 1984).

Although the literature reports agree on some basic principles to understand, explain and predict high numbers of overwintered larvae on the spring turn-out pasture, or when the pasture infectivity can be expected to reach dangerous levels, general simple rules are lacking.

Arrested development

Arrested development at the early L4 stage was found almost exclusively in tracer animals grazing in September, and to a slightly higher extent for *Cooperia* spp. than to *Ostertagia* spp. (study II). Many factors, like climate, management, host age and immunity, may influence the induction of arrested development in nematodes (Armour & Duncan, 1987; Eysker, 1997). In the northern hemisphere, seasonal factors seem to be of significant importance (Michel, Lancaster & Hong, 1974, 1975), and low temperatures have been suggested as a primary stimulus (Armour & Bruce, 1974). Seasonal inhibition has been shown in many nematode species of both cattle and sheep in the cool temperate regions of the northern

hemisphere (Almeria, Llorente & Uriarte, 1996; Claerebout*, et al.*, 1997a; Waller*, et al.*, 2004). In a Danish study, an almost ten-fold increase in the proportion of inhibited EL4 *O. ostertagi* was observed in tracer animals in a period of only 6 weeks between August and September/October (Satrija & Nansen, 1993). By using parasite-naïve calves of approximately the same age on all occasions in study II, the clear difference in percentage of arrested EL4 found in late versus early season was primarily thought to be due to external seasonal factors acting directly on the L3.

Control of GI nematodes

Rotational grazing

In study I rotational grazing, to control GI parasite infections in FSG cattle, was carried out by using a spring turn-out pasture that had been grazed by SSG cattle for the latter part of the previous season, in combination with one move in mid-July to aftermath grazing (Fig. 4). Thus, the spring turn-out pasture was spelled from grazing with FSG cattle until the new set of FSG cattle was allocated the following spring. Results showed that infections with GI nematodes in FSG cattle could be acceptably controlled through this strategy without the use of anthelmintics. The FSG in the RT group showed no clinical symptoms of parasite infection, and their mean daily weight gain of at least 0.8 kg/day was similar to the animals that were treated every fourth week with doramectin. This was probably a result of comparatively low levels of overwintering infective larvae on paddock RT, and with a smaller proportion of those being *Ostertagia* spp. compared to contaminated paddocks not grazed by SSG cattle. On no occasion during the 3 years of this study were tracers grazing the RT paddock found to harbour >20,000 worms of *Ostertagia* spp. As this grazing management strategy involved both evasive grazing (the mid-summer move to aftermath) and dilution (SSG cattle on the spring turn-out pasture), their relative contribution to the positive effects on performance of FSG cattle in group RT is difficult to estimate.

Fig. 4. First-season grazing cattle on aftermath pasture.

The aftermath paddock seemed to be parasitologically safe, and was found to have only low levels of nematode infective larvae by July for 2 consecutive years, which implied an opportunity for excellent late season pasture for FSG cattle. Clinical symptoms were never observed in the animals (study I) in this paddock, despite high pasture larval counts in the aftermath paddock around the time for housing (study II). This is in accordance with Eysker*, et al.* (1998b), who also concluded that the move of FSG cattle resulted in a considerable avoidance of pasture contamination. To the farmer this means optimal usage of a pasture following a hay or silage cut, and a practical, reliable and labour-saving component in the tool box of non-chemical parasite control. The reason for the positive control effects achieved with this management is surely the natural reduction of the numbers of overwintered infective larvae from mid-May to mid-June (Nansen*, et al.*, 1987), in combination with the silage cut and removal of infected herbage in early June, as previously demonstrated in Sweden (Dimander, 2003) and in The Netherlands (Eysker, Kooyman & Wemmenhove, 1988).

For maximum parasite control with the rotation strategy evaluated in studies I and II, the SSG cattle should ingest as many infective larvae as possible but without allowing these to develop to patent infection that would re-contaminate the pasture. Thus, the beneficial effect of using SSG cattle would be attributed to their acquired immunity that would make them more or less parasitologically inert. Approximately 80% of all faecal samples (approximately 700 samples) taken from SSG cattle in study III were below the detection level of 50 epg. However, as reviewed by Stromberg & Averbeck (1999), the average total number of eggs deposited onto the pasture may be significantly higher from older (larger) cattle

compared to young calves because they produce more faeces. Thus, the slightly higher egg output of previously underexposed cattle observed in study III may possibly contribute to pasture infection (Herbert & Probert, 1987).

Because immunity develops earlier against *C. oncophora* than *O. ostertagi* (Armour, 1989; Hilderson*, et al.*, 1995), it would be expected that any contamination generated by SSG cattle would be derived from the more pathogenic *O. ostertagi*. However, the proportion of *Ostertagia* spp. to *Cooperia* spp. observed in tracers grazing paddock RT (study II) indicated in fact that *Cooperia* spp. dominated following grazing by SSG cattle. This may be explained either by a mixed *Ostertagia* and *Cooperia* spp. infection of the SSG cattle, or by survival of the early-season FSG cattle contamination on the RT paddock. Successful survival and overwintering of infective larvae derived from early season grazing is possible (Dimander, Höglund & Waller, 1999). If a majority of the early-season contamination from the FSG cattle survived and escaped ingestion of the SSG cattle, this indicates that these larvae actually never were available for the SSG during late-summer grazing.

Grazing with different age classes of the same species has previously been found to be successful in controlling nematode infections of FSG cattle, either by pasture alternation with older cattle (Axelsen*, et al.*, 1986; Dimander*, et al.*, 2003), or by mixed grazing (Nansen*, et al.*, 1990; Šarkūnas*, et al.*, 2000). Still, attention is recommended when this measure for control is advocated for consecutive years. This is illustrated in study I by the elevated mean pepsinogen concentration to almost sub-clinical levels in group RT 2 weeks before the move to the aftermath, indicating a possible build-up of pasture infectivity between years. To complicate things even more, the risk of lungworm infection in SSG cattle (study III), and thus the transmission to pasturelands intended for FSG cattle, must be carefully considered, since an important route of transmission of *D. viviparus* between years is through carrier animals (Saatkamp, Eysker & Verhoeff, 1994).

Information about pasture contamination from SSG cattle with *E. alabamensis* oocysts is limited. In study I, high levels of *E. alabamensis* oocysts were only occasionally found in FSG cattle turned out onto paddock RT, and no clinical coccidiosis was observed in these animals. Whether this was an effect from SSG cattle grazing that paddock in late season, or simply a coincidence, is unknown. However, results from a field survey including FSG cattle from 59 farms in Sweden (Svensson, 1995) showed that calves that were turned out onto pastures previously grazed by older cattle or horses excreted significantly fewer oocysts than the calves that were turned out onto pastures grazed only by FSG cattle. It was therefore suggested that cattle previously exposed to *E. alabamensis* may dilute oocyst contamination of a pasture because of their lower susceptibility to infection (Svensson, 1995).

Nutritional supplementation on pasture

As already indicated, nutritional supplementation to grazing cattle is used among many Swedish farmers as a means to minimize parasite infection (Svensson,

Hessle & Höglund, 2000). However, the supplementary feeding with concentrate and hay for 4 weeks from turn-out (study I) proved to be unsuccessful for parasite control. This was demonstrated in terms of clinical condition, weight gain and pasture infectivity. The rationale behind supplementary feeding at the time of turn-out was to provide a smooth transfer from pen feeding to a sole pasture diet for young parasite-naïve cattle. The theory behind this is partly to improve their level of nutrition when first exposed to parasitism, and partly to utilize the indirect reduction of exposure to infective larvae on pasture by replacing herbage intake with supplement. During the planning of the field trial presented here, it was assumed that supplementation to grazing cattle in practice is a balance between the cost for feed and the labour to provide the feed, as well as the value of any production benefits obtained. For simplicity, we decided to offer the same quality of concentrate as during the preceding housing-period, and to feed both concentrate and hay on a group basis.

Many studies with small ruminants show that both resistance (i.e. the ability to limit worm burden) and resilience (i.e. the ability to maintain production despite parasitism) to nematode infections are influenced by diet, and particularly by a metabolisable protein supply (van Houtert, Barger & Steel, 1995; Datta*, et al.*, 1999; Kahn*, et al.*, 2003). An increase in energy intake has also been shown to improve resilience of periparturient ewes (Kahn, 2003). By comparison, few studies have focused on this matter in grazing cattle managed under practical farm conditions. However, positive results on clinical condition, FEC and weight gain have been observed in grazing heifers offered lucerne pellets *ad libitum* from the end of July (Jørgensen*, et al.*, 1992). This was probably related to both a reduced intake of herbage because of a reduced grazing time and an increased resilience to infection (Jørgensen*, et al.*, 1992). Improved performance has also been observed in grazing cattle supplemented with cottonseed meal (Magaya*, et al.*, 2000) and urea-molasses (Waruiru, 2004), although there was no observed effect on FEC. However, Ciordia*, et al.* (1962) observed that grazing calves receiving long-term supplementation with grain had a higher daily weight gain, as well as a lower worm burden, compared to non-supplementary fed grazing animals.

The reasons for the poor results of supplementary feeding in study I are unclear. The timing, length of feeding, quantity and quality of the supplement are factors that require further investigation under field conditions. In any case, the supplementary feeding regime may not be too laborious and impractical. In study I, the same quality of concentrate used during the preceding housing period was offered after the turn-out. The cattle adapted quickly to the daily feeding, and the concentrate was readily consumed on each feeding occasion. However, the daily ration was halved from the first day on pasture compared to the housing period. An alternative feeding strategy would be to offer the full housing ration for the first week or two on pasture, followed by a gradual decrease. A higher amount of concentrate would increase the substitution effect for herbage, i.e. the concentrate would substitute for a certain amount of herbage (Bargo*, et al.*, 2003) and consequently reduce the intake of infective larvae. This would reduce the intake of larvae and also benefit by the declining numbers of overwintered larvae on pasture in spring (Nansen*, et al.*, 1987).

Early-season coccidiosis is a likely explanation for the negative performance of the animals in group FD the second year (2003). It may be speculated that the permanent location of the trough predisposed a local accumulation of *E. alabamensis* oocysts around this area, which triggered the coccidiosis in the FD group in 2003. This must be considered together with the fact that oocysts successfully overwinter on pasture (Svensson, 1995), as coccidiosis in young FSG cattle may obliterate any benefits from early-season feed supplementation. During the last year of study I, the faecal oocyst excretion was considerably lower in the FD group, and these animals performed as well as animals in the DO group. Notably, the doramectin salvage-treatment of group FD in 2003 resulted in reduced nematode contamination, which would lower the potential for the numbers of overwintered nematode larvae at spring turn-out in 2004. This, of course, compromised the evaluation of the FD group in the last year of the study.

Performance during the second grazing season

It has been speculated that overprotection to GI parasites during the first grazing period may lead to increased susceptibility to GI nematodes in the second grazing season (Vercruysse, Hilderson & Claerebout, 1994, 1995). However, in study III the SSG cattle (Fig. 5) showed no significant differences in weight gain between groups that were subjected to different levels of parasite exposure in their first grazing season. This is in agreement with results from other studies (Satrija*, et al.*, 1996; Eysker*, et al.*, 2000), and suggests that although immunity to GI nematodes depends on previous levels of exposure (Ploeger, Kloosterman & Rietveld, 1995), problems with poor weight gain may only be expected when exposure is very low in the first grazing season and high in the second (Eysker*, et al.*, 2000). In study III it was assumed that the pastures used for the SSG cattle were only lightly contaminated at the start of the trial. The possibility can therefore not be excluded that higher levels of pasture infectivity would have given a different result.

According to studies I and III, weight gain penalties observed at the end of the first grazing season generally remained throughout the housing period and the second grazing season, both in 2004 and in 2005. Thus, none or little compensation for lost productivity due to parasite infections during the first grazing season was observed in this trial. Accordingly, suppressive anthelmintic treatment during the first grazing season does not necessarily lead to reduced performance the subsequent grazing season. The weight loss in the last year of the study was certainly correlated to infections with *D. viviparus* which can significantly reduce performance of infected cattle (Boon, Kloosterman & Breukink, 1984).

A significantly higher output of trichostrongylid eggs from SSG cattle that had been treated with the long-acting anthelmintic doramectin every 4 weeks the previous year was observed in study III. Further, faecal egg counts increased between years, both in previously anthelmintic-treated and non-treated SSG cattle, indicating a potential build-up in pasture infectivity. Development of high pasture infectivity by SSG cattle is a clear possibility, and has been shown both by Herbert

& Probert (1987) and Eysker, *et al.* (2000). In contrast, Claerebout, *et al.* (1999) found no evidence that a slightly higher egg output by SSG cattle had an effect on pasture infectivity. Whether this was true in study III is not clear, since no herbage samples or tracer animal tests were performed.

Neither of the SSG cattle excreted significant numbers of *E. alabamensis* after turn-out in any of the years. Whether this was due to maintenance of immunity acquired during the first grazing season or related to low pasture infectivity of oocysts, cannot be concluded from results in the present trial. However, Svensson (2000) stated that reinfection with *E. alabamensis* is of little clinical importance, and that young cattle infected in their first grazing season may graze contaminated pastures the second grazing season without the risk of developing clinical coccidiosis.

Fig 5. A second-season grazing steer along the water front, enjoying the Swedish summer on one of the communal pasturelands.

Summary and applications of the results

Eimeria alabamensis coccidiosis had a significant impact on animal welfare, with clinical symptoms that caused direct and future consequences on the performance of affected FSG cattle. The severe diarrhoea, in combination with lack of appetite, claimed laborious, time consuming and expensive individual nursing to sustain

animal welfare. Infected animals excreted millions of oocysts 8 to 9 days after natural or artificial infections that contaminated the pasture exceedingly.

If there is a history of *E. alabamensis* coccidiosis on a farm, the calf-paddocks may be highly contaminated with *E. alabamensis* oocysts the following spring, even if only used for 2 weeks. It is therefore recommended that such pastures may not be allowed for FSG cattle. Faecal sampling for diagnose should be performed on day 8 to 10 after turn-out, but it has to be remembered that clinical disease may appear already within the first week on pasture.

Large numbers of overwintered GI nematode larvae were observed on permanently grazed paddocks, unless the animals were prophylactically treated with anthelmintics. A rapid decline was evident between April and June, but levels were still high enough in mid-May to induce early-season PGE in FSG cattle that where assigned to permanent cattle pastures. Elevated levels of serum pepsinogen concentrations (SPC) were observed in FSG cattle within 8 weeks of grazing, when high levels of overwintered GI nematodes were present. The peak in FEC was observed after 4 weeks in all 3 years. Thus sampling for FEC is preferably performed after 4 weeks grazing, and blood samples for SPC after 4 to 8 weeks. However, blood samples for SPC may still be indicative for disease in late season.

Adequate performance and comparatively low numbers of overwintered GI nematode larvae were observed in the 2-paddock rotational system. However, there were some indications that the numbers of overwintered infective larvae may reach dangerous levels in the spring turn-out paddock in this particular 2-paddock rotational system. Therefore, caution has to be used when this strategy for parasite control is used and possible reasons for a breakdown of this strategy for parasite control are discussed in detail elsewhere in the thesis. However, the aftermath paddock was parasite safe from mid-July after mowing in all years, despite presence of high numbers of overwintered GI nematode larvae in spring.

Supplementary feeding, as applied in this thesis, did not prove effective as a parasite control strategy for FSG cattle. However, the salvage treatment the second year obviously reduced pasture contamination, which compromised a thorough evaluation of this strategy the last year.

Based on this thesis and earlier experience from Sweden, a mid-summer move to aftermath following a silage or hay cut is strongly recommended from a parasitological point of view. This strategy can be practised safely for consecutive years regardless of the level of contamination from untreated FSG cattle. However, it is necessary to provide such animals with a ‘parasite-clean’ pasture at turn-out. Although the supplementary feeding regime was unsuccessful, the principles behind this method motivate further research and evaluation of other regimes that are known to be practised among Swedish farmers.

No differences in performance could be identified in SSG cattle that had experienced different levels of parasite exposure during their first grazing season. This was observed under conditions when the SSG cattle grazed pastures where

the level of pasture infectivity was low to moderate. However, only marginal compensation of the weight gain penalties that resulted from different levels of parasite infections the first grazing season was observed during the second grazing season, in 2 out of 3 years.

Taken together, when providing Swedish farmers with non-chemotherapeutic approaches to GI parasite control for FSG cattle, the overwintering capacity of infective larvae and coccidia should be emphasised. To prevent exposure to detrimental levels of parasite infection at spring turn-out, 'parasite-clean' pastures are strongly advocated for FSG cattle.

Finally, based on findings in this and previous studies with FSG cattle in Sweden, a guarded definition of a clean or parasite-free pasture may be a pasture that have been spelled from grazing with FSG cattle during the whole previous grazing season or an aftermath pasture where a silage cut has been taken in early summer.

Future research

- Continued research on the interaction between GI nematode and *Eimeria alabamensis* infections
- Continued research on the effects of using SSG cattle to 'vacuum-clean' heavily infected pastures, with special reference to pasture contamination, but also on the consequence on the performance of the SSG cattle.
- Further studies on supplementary feeding regimes to FSG cattle
- Studies on the effects of mixed grazing between sheep and cattle, or horses and cattle, on pasture contamination, pasture quality and the performance of the animal spices
- Improve the transfer and 'translation' of parasitological research to the farmers

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