

ABSTRACT

Thompson, Bianca Merrick. Effects of Milk Feeding Period and Anthelmintic Treatment on Fecal Egg Counts and Growth in Pastured Dairy Steers. (Steven P. Washburn)

A 2 x 2 factorial trial with 2 phases was conducted to evaluate the effects of weaning age (6 wk vs 12 wk) and anthelmintic treatment (non vs dewormed) on average daily weight gain (ADG) in 36 Holstein and Jersey X Holstein crossbred steers born in fall (Oct - Nov) and winter (Dec – Feb) 2003 – 2004. Steers were blocked into 4 treatment groups by birth weight and breed. Calves of similar age were managed together in pastures regardless of treatment and group-fed 3.8 to 7.6 L of whole milk/d until weaning. Phase 1 extended from birth until July 15, 2004. Phase 2 started on July 15 and ended on Nov 18, 2004. Dewormed calves received 1 mL ivermectin pour-on formulation/ 10 kg body weight (BW) at 12 and 20 wk of age, and again on July 15 and Sept 23. Fecal samples and BW (birth to Nov 18, 2004) were taken from each calf at 4-wk intervals. Fecal egg counts (FEC), BW, and ADG (during Phase 1, Phase 2, and Phases 1 & 2 combined) were compared among deworm, wean age, % Holstein, birth season, and their interactions. Parasite eggs were not detected until April and were lower in dewormed calves after July and Sept treatments. Fall-born calves usually had lower FEC than winter-born calves. Gains during Phase 1 were higher for fall-born calves. In Phase 2, dewormed calves actually had faster ADG than non-dewormed calves. Gain across Phase 1 and Phase 2 were higher in fall-born calves and tended to be higher in calves weaned at 6 wk. Gains and BW generally were higher with increasing % Holstein. Although steers that were not dewormed had higher FEC and differing ADG during parts

of the trial, their overall performance was similar to those that received 4 doses of ivermectin.

Key words: (anthelmintic, weaning, dairy steer, gain)

EFFECTS OF MILK FEEDING PERIOD AND ANTHELMINTIC TREATMENT ON
FECAL EGG COUNTS AND GROWTH IN PASTURED DAIRY STEERS

by
BIANCA MERRICK THOMPSON

A thesis submitted to the Graduate Faculty of
North Carolina State University
in partial fulfillment of the
requirements for the Degree of
Master of Science

DEPARTMENT OF ANIMAL SCIENCE

Raleigh

2005

APPROVED BY:

Steven P. Washburn
Chair of Advisory Committee

Jean-Marie Luginbuhl
Committee Member

Brinton A. Hopkins
Committee Member

DEDICATION

This thesis is dedicated to my late grandfather, Dr. Fred D. Rosi, PhD in Materials Science, who was a pioneer in his field and an inspiration for me to finish strong.

BIOGRAPHY

Bianca Rosi Merrick was born in Manhasset, New York, on May 4, 1979, to Bree Rosi Merrick and Frederic DuPuy Merrick. She was raised in Old Brookeville, New York, until the age of 11 when she moved to Richmond, Virginia, where she attended middle school and high school. She graduated from J. R. Tucker High School in May of 1997 and began her freshman year at North Carolina State University in the fall of 1997. In May, 2001, Bianca graduated from North Carolina State University with a Bachelor of Science degree in Animal Science. Soon thereafter, she married Jonathan Forrest Thompson on June 23, 2001, and became Bianca Merrick Thompson.

Bianca then spent the next two years working out in the veterinary field until returning to North Carolina State University for her Master's degree under the direction of Dr. Steve Washburn. Bianca was given a teaching assistantship for her first year of graduate school, which allowed her to TA the Anatomy and Physiology class for two semesters in a row. For this work, she was given an Outstanding Teaching Assistant Award in 2005. She successfully defended her thesis on May 16, 2005, and will graduate in August, 2005, with her Master's degree in Animal Science. Bianca will then pursue a teaching career at the collegiate level.

Over the years, Bianca has been blessed by being exposed to various aspects of the animal science field. As an undergraduate, she was able to work on the university dairy educational unit. Subsequently, she spent a brief stay in the Netherlands in cooperation with the Wageningen Research Station. She has worked at the NCSU College of Veterinary Medicine with Hepatitis research involving swine farmers. She is very thankful for this diverse exposure to the field.

ACKNOWLEDGEMENTS

I would like to sincerely thank all of my family and friends who have supported me over the past 2 years while I have been in graduate school. I would like to specifically thank my mom, dad, stepmom and stepdad for their unending support and encouragement. I would also like to thank my in-laws, who prayed me through graduate school. Perhaps the biggest thank you (which is hard to rank) goes to my husband, Josh, whose selflessness, patience, and unconditional love helped me endure two of the hardest years in her life.

Thank you also to all of the graduate students who lent moral support and who understood my pain. Thank you to Heather Glennon, my technician and friend, for pouring herself out for my research and me and for never complaining. Her maturity and compassion meant the world to me.

Thank you to my committee for their guidance and for passing me. Thank you, most of all, to Jean-Marie, who kept me laughing and who shared wisdom and understanding with me that extended far beyond the world of research.

TABLE OF CONTENTS

	Page
LIST OF FIGURES	vii
LIST OF TABLES	viii
REVIEW OF LITERATURE	
Introduction	1
Internal Parasites	2
<i>Classification</i>	2
<i>Anatomy</i>	3
<i>Life cycle</i>	3
<i>Infection</i>	5
<i>Immunity</i>	8
Measures of Parasitism	9
<i>Fecal Egg Counts</i>	9
<i>Pepsinogen Assay</i>	9
Anthelmintics	10
<i>Traditional Chemicals</i>	10
<i>Parasite Resistance</i>	15
<i>Organic Alternatives</i>	18
Biological Control	21
<i>Duddingtonia flagrans</i>	21
<i>Dung Beetles</i>	22
Grazing Strategies	22
<i>Rotational Grazing</i>	22
<i>Stocking Rate</i>	24
<i>Alternate Grazing</i>	24

Genetics	25
Milk and weaning Age	26
List of References	28
MATERIALS AND METHODS	43
RESULTS	48
DISCUSSION	51
CONCLUSIONS	55
FURTHER APPLICATION	56
REFERENCES	61

LIST OF FIGURES

	Page
Figure 1. Timeline of Phases 1 and 2 for the duration of the trial.....	65
Figure 2. Lsmean average daily gains (kg/d +/- S.E.) of treated and untreated animals during Phase 1, Phase 2, and Phases 1 and 2 combined.....	66
Figure 3a. Lsmean of weights (kg +/- S.E.) of 4 different treatment groups throughout Phases 1 and 2.....	67
Figure 3b. Lsmean of average daily gain (kg/d +/- S.E.) of 4 different treatment groups.....	68
Figure 4. Phase 2 (July 15, 2004 to Nov 18, 2004) lsmean weights (kg + S.E.) of calves weaned at 6 wk (n=18) or 12 wk (n=18).....	69
Figure 5. Phase 2 lsmean weights (kg +/- S.E.) of calves of varying breeds.....	70
Figure 6a. Phase 2 fecal egg counts (eggs per gram +/- S.E.) of treated and untreated calves.....	71
Figure 6b. Phase 1 and Phase 2 fecal egg counts (eggs per gram) of each treatment group.....	72
Figure 7. Phase 1 and Phase 2 fecal egg counts (eggs per gram) of fall-born (Oct-Nov) and winter-born (Dec-Feb) calves.....	73

LIST OF TABLES

	Page
Table 1. 2 x 2 factorial arrangement of treatments with weaning age and treatment as classification variables.	74
Table 2. Feed analyses throughout Phases 1 and 2.....	75
Table 3. Lsmean weight (kg +/- S.E.) and lsmean average daily gain (ADG; kg/d +/- S.E.) comparisons between calves weaned at 6 wk or 12 wk and either treated or untreated with ivermectin pour on.....	76
Table 4. Lsmean ADG (kg/d +/- S.E.) for fall-born (October and November) and winter-born (December – February) calves during Phases 1 and 2.....	77
Table 5. Fecal egg counts (lsmean: eggs per gram +/- S.E.) of fall-born (October and November) and winter-born (December - February) calves during Phase 2.....	78

REVIEW OF LITERATURE

Introduction

Internal parasites have been shown to decrease weight gain, negatively impact reproductive measures in cows and reduce overall health of calves, which leads to severe economic losses in production (Perry and Randolph, 1999; McLeod, 1995). Ploeger and Kloosterman (1989) estimated yearly costs associated with gastrointestinal parasites in the Netherlands as \$100 million. Estimating the economic losses of cattle on pasture is difficult, though, because of having to factor in variations in cattle, parasite infections, weather, management, etc (Hawkins, 1993).

The exact mechanisms of how parasites cause decreased growth and other clinical signs are unclear, but scientists hypothesize that due to damage to the intestinal wall by inhabitation of parasites, amino nitrogen is sequestered to the gastrointestinal tract for repair instead of being partitioned for growth and development (Hawkins, 1993). To confirm the involvement of protein loss in parasitic infections, studies have shown that calves consuming low protein diets will have lingering effects from nematode infection even after being treated with an anthelmintic (Parkins et al., 1982).

In the southeastern part of the United States where summers are extremely hot and humid and the winters are not cold enough for long enough to impact the parasite life cycle (Anderson, 2000), whole farm approaches to decreasing parasite burdens in calves, especially in the first grazing season, have to be taken. Developing alternatives to chemical anthelmintics via organic dewormers and integrated pasture management for the control of nematodal parasites is also becoming a necessity in the dairy cattle industry as

a whole due to the increased interest in organic farming and also the growing anthelmintic resistance issue in cattle.

Internal Parasites

Classification

All cattle gastrointestinal parasites fall under the Kingdom: Animalia, the Phylum: Nematoda, the Class: Secementea and the Order: Strongylida (Johnstone, 1998). Common characteristics of all nematodes in the Strongylida order are: the presence of a bursa, a copulatory bursa in all males, a buccal capsule that varies in size, both direct and indirect life cycles and, lastly, strongyle eggs that are thin shelled, smooth surfaced, and ellipsoidal in shape strongyle type eggs. This order contains two superfamilies that encompass cattle parasites: the Trichostrongyloidea and the Strongyloidea.

The Trichostrongyloidea superfamily is broken down into two families: Trichostrongylidae and Dictyocaulidae (Johnstone, 1998). *Ostertagia* is the predominant Trichostrongylidae genus in cattle with *Ostertagia ostertagi* spp. (brown stomach worm) being the most prevalent parasitic species in cattle. The other parasites in the Trichostrongylidae family that are of importance in cattle are: *Trichostrongylus* (*T. axei* and *T. colubriformis* spp.), *Haemonchus* (*H. placei* spp.), *Nematodirus* (*N. helveticus* spp.) and *Cooperia* (*C. oncophora* spp.). *Dictyocaulus viviparus* is the only parasite in the Dictyocaulidae family that infests cattle (Johnstone, 1998).

Oesophagostomum radiatum is in Strongyloidea superfamily and is the only member in this family that affects cattle production (Johnstone, 1998).

Anatomy

Nematodes are often called roundworms because they are cylindrical worms that taper at both the head and tail ends. They have an outer skin called a "cuticle" which is often modified. At the tail end of many nematodes are the caudal alae, and caudal papillae. The caudal ala is a cuticle that is expanded to form a bursa at the end. The bursa is supported by rays, which are papillae, all of which are expansions of the cuticle. This bursa is the copulatory bursa and is used to grasp the female during copulation (Anderson, 2000).

The GI tract consists of three parts: a mouth opening that leads to a buccal cavity, an esophagus, and a simple intestine. The mouth opening varies by species from simple opening to large opening with thick cuticular walls and teeth. The esophagus is thick walled, lined with cuticle, contains muscle, nerve, and glands in its walls. The esophageal muscles serve to dilate the lumen of the esophagus in a pumping fashion, to suck in liquid food and pump it to intestine. In some worms, the sucking action can reach 120 pulsations/minute and this voracious feeding can lead to pathogenicity. The intestine opens into the rectum in females or the cloaca in males, which is lined in cuticle and terminates in an anus that opens as excretory duct or pore (Anderson, 2000).

Life Cycle

Life cycles of the more common cattle internal parasites are direct which means that they do not require an intermediate host like a snail (Anderson, 2000). Adult males and females reproduce and the females lay eggs called ova. The ova get passed into the environment through the cattle feces and the ova hatch into the first larval stage 1 (L1). With favorable weather conditions, L1 larvae will develop and molt their protective outer

cuticle, which constrains growth, twice before becoming the infective stage L3 larvae. Stage L3 larvae migrate from the manure up the moist blades of grass and then are eaten as the cattle graze the grass. The larvae usually only ascend 2 inches on the blade of grass, so grazing pressure is an important principle in integrated parasite management.

The parasites travel to their site of incubation (abomasum, small intestine, large intestine or lungs), molt through two more larval stages (L4 and L5), and then mature into egg-laying adults. The time for newly hatched eggs to develop into the L3 infective stage (called the preparasitic phase) is typically 4 to 6 days. The time from the ingestion of the L3 larvae to adults hatching eggs is around 3 weeks and is called the parasitic phase or prepatent period (Georgi and Georgi, 1990).

The life cycle of *Ostertagia* differs slightly from the other parasites. Similar to the other parasites, *Ostertagia*'s L3 larvae are ingested from the grass and travel to the abomasum where they gather together and form swellings and nodules in the gastric pits and glands of the abomasum (Sommerville, 1953). The parasites move out of the mucosa and into lumen for each molt and then migrate back into the mucosa until the next phase. The fourth phase is where *Ostertagia* diverge from the other parasites.

The fourth-stage larvae are able to stay in the stomach glands for up to six months. These are called inhibited or arrested larvae (Armour and Osborne, 1982; Melancon, 2001). The ability to arrest and then leave the stomach a gland seems to be triggered by weather or nutritional factors and is a heritable trait. When favorable weather occurs, larvae will leave the stomach lining and become adults, which is the fifth stage. Adults survive by living in the mucosal lining of the abomasum of infected hosts.

Coccidia are the only cattle parasite that causes pathogenicity that does not belong to the nematodal phylum. The coccidia life cycle is very complex, short and fast spreading. Coccidia produce eggs (oocysts) in very large numbers like *Haemonchus*, but the complete life cycle takes only 21 days (Melancon, 2001). Unlike other parasites, the calves ingest coccidial eggs (instead of larvae) called oocysts and then the eggs hatch inside the animal. The coccidia develop through several life stages during which they damage the digestive tract. Cattle may pick up coccidia eggs from contaminated forage, water or by licking themselves or other cattle soiled with contaminated manure. Eggs are very hardy and will contaminate premises for a long time (Herrick, 1990).

Infection

A parasitic relationship exists between nematodes and cattle when the parasite profits at the expense of the host animal. Parasitic infestations irritate and inflame the stomach and intestinal linings, or mucosa, and cause damage called parasitic gastroenteritis (PGE). The damage to the GI tract prevents cattle from getting the proper nutrients, due to the decreased digestive and absorptive ability of the mucosal surface.

Ostertagia are brown in color and act on the abomasum of calves; hence the nickname brown stomach worm. Development of *Ostertagia* was observed as early as 1892 by Stadelmann and then in 1901 by Stodter. Threlkeld (1934) was the first scientist to carry out a study with *Ostertagia*. Its pathogenesis is one of the most important among helminthes in cattle in temperate climates (Myers and Taylor, 1989; Vercruyssen et al., 1986). The optimal temperature for hatching and developing into infective L3 larvae is 25°C, which makes areas with temperate climates highly susceptible to these parasites (Pandey, 1972). Because of their specific temperature parameters, adult *Ostertagia* will

deposit eggs in the feces in the spring months where the eggs will wait and delay maturity into 3rd-stage larvae until late August. Calves grazing in the late summer will eat these larvae and show clinical signs of Ostertagiosis in the fall months (Michel, 1969; Armour and Osborne, 1982). These L3 larvae also have the ability to survive the winter by going into the arrested state and infect the calves in the following spring. Increased fecal egg counts will therefore be seen in calves both in the spring and the fall with a drop in the summer when temperatures are above 30 degrees Celsius (Williams and Knox, SR 6004).

Types 1 Ostertagiosis occurs when large numbers of infective larvae are eaten over a short time and quickly complete their life cycle to become adults (Anderson, 1988). Type 2 Ostertagia infection occurs when the larvae leave the stomach lining (Anderson, 1988). Severe problems occur only when large numbers of larvae mature and leave at the same time. A Pre-Type 2 stage occurs when the fourth-stage larvae burrow deep into the stomach lining and are inhibited. There may be as many as 100,000 of the inhibited larvae in the stomach wall (Anderson, 1988).

Ostertagia infection generally manifests in the calf as diarrhea and decreased appetite (Hawkins, 1993). Specifically, Type 1 Ostertagia clinical disease is characterized by severe greenish diarrhea, swelling under the jaw (bottle jaw) and rapid weight loss (Forbes et al., 1999). Young animals show the most severe symptoms, which will continue until the calves die or are treated. All age animals may show symptoms, especially weight loss (Organic Livestock Research Group, 2000).

A Pre-Type 2 Ostertagia infection doesn't normally produce any damage or symptoms when the larvae enter the stomach glands. They're arrested or inhibited until weather changes trigger their release. Adults suddenly leaving the stomach glands cause

type 2 *Ostertagia* infections (Anderson, 1988). Their leaving may be gradual or in a sudden burst. With gradual releasing of the adults, the symptoms are similar to those exhibited in Type 1. But a sudden weather change may trigger the release of all inhibited worms and cause acute symptoms with high death loss of the host.

Cattle dying with Type 2 *Ostertagia* infection will show yellowish and flat-topped lesions in the lining of the stomach. The stomach fluid will have a high pH, and a blood test may help make the diagnosis. Type 2 *Ostertagia* infections are dangerous and need to be diagnosed quickly because not all dewormers are effective in treating this type of internal parasite infection (Anderson, 1988).

Like *Ostertagi ostertagi*, *Haemonchus placei* also affects the abomasum of cattle. These worms are called the barberpole worm because of the females who are red and white because of the blood vessels encircling the white egg-filled uterus (Georgi and Georgi, 1990). The female produces a large amount of eggs, which will permeate the pasture and heavily infect the cattle grazing there. Both the larvae and adults feed on the blood in the abomasums and are known for being voracious bloodsuckers. They have a small lancet in the buccal capsule that they extrude and use to slit open the host's capillaries and then they ingest the blood, which causes anemia and hypoproteinemia (Benz, 1985). Small numbers of these worms can cause acute symptoms with blood and protein loss. A heavy infestation will present as severe anemia or sudden death.

Trichostrongylus axei (Small stomach worms) affect the abomasum and *Trichostrongylus colubriformis* (Bankrupt worms) affect the duodenum of the small intestine. The *Trichostrongylus* genus tolerates low temperatures but can be killed when the temperature drops to freezing levels (Anderson, 2000). Calves with an infection from

these parasites are anemic, weak, thin, and have soft swelling under the jaw and abdomen.

Nematodirus helventianus (Thread necked Intestinal worms) and *Cooperia oncophora* (small intestinal worms) are duodenal parasite species whose infections manifest similarly to a *Haemonchus* infection. *Dictyocaulus* are found in the bronchi and trachea of cattle and present clinically as bronchitis and a condition called husk (Allan and Johnson, 1960). The last nematode causing parasitic infection in cattle is *Oesophagostomum radiatum* (Nodular worms) in the Strongyloidea Superfamily, which affects the large intestine of cattle (Anderson, 2000).

Coccidiosis may strike any time of year, but most severe outbreaks occur in stressful weather, especially cool, wet months of fall, winter and early spring. A calf with coccidiosis will have bloody and mucous-like diarrhea (Melancon, 2001). These symptoms, if not treated early enough, will lead to dehydration, anemia, weight loss, lethargy, pneumonia and eventual death (Melancon, 2001). Some infections may be self-limiting, meaning that the population of *Coccidia* will grow to a maximum level and then fade away within a week if reinfection does not take place.

Immunity

Calves in the first grazing season (FGS) are most susceptible to infection (Berghen et al., 1993) and will often exhibit the signs of Ostertagiosis such as diarrhea, weight loss, and inappetance. However, as cattle go through one grazing season and become yearlings, they develop immunity to parasites as long as they have sufficient exposure to parasites (Armour, 1989). Adult cattle continue to shed eggs, but in very low numbers which cause no adverse effects.

Measures of Parasitism

Fecal Egg Counts

Fecal egg counts are commonly used as indicators of parasitic infection. The procedure involves taking fecal samples and counting how many parasite eggs are in the sample and then dividing the eggs by the total number of grams of sample and reporting eggs per gram (epg). The preparation of the fecal sample can vary between techniques. Some techniques are not sensitive enough to detect the low numbers of parasites that cattle have because the procedure is designed to detect larger numbers of eggs, as in small ruminant samples. This method is used frequently because it is inexpensive and easy to perform. This method, though commonly used in parasite trials, is not an accurate indicator of parasitic load because the amount of eggs being shed does not directly relate to the number of adults in the GI tract (Rohrbacher, 1958); i.e. there can be a few females shedding a large portion of the eggs and a large percentage of the females shedding very few.

Pepsinogen Assay

Pepsinogen is measured in an assay (Dorny and Vercruysse, 1998) as an indicator of *Ostertagia* infection because as these parasites reside in the abomasum of cattle, they create lesions in the abomasal wall, where pepsinogen (a precursor for pepsin which breaks down protein) will leak out into the bloodstream. An *Ostertagia* infection causes damage to the mucosal and parietal cells in the abomasum, which will decrease the acid (secreted by the parietal cells) that converts pepsinogen into pepsin, leading to a buildup of pepsinogen (Berghen et al., 1993). Other parasites can cause rises in pepsinogen levels, but these results are often inconsistent (Ross et al., 1967), and the other parasites

are of little concern in cattle in temperate regions (Vercruysse et al., 1986). In the pepsinogen assay, tyrosine units are measured due to the fact that in the assay, the substrate in the assay breaks pepsinogen down into tyrosine where it can be measured spectrophotometrically.

In two cattle experiments where calves were infected with *Ostertagia ostertagi*, strong correlations were found between increased plasma pepsinogen and variables associated with parasitic infection such as diarrhea and weight loss (Jennings et al., 1966; Anderson et al., 1966). Calves uninfected with parasites have pepsinogen levels below 1000mU tyrosine (Xiao et al., 1991; Snider, 1981). When correlating the pepsinogen values with PGE, Selman et al. (1977) concluded that plasma pepsinogen concentrations of > 3,000 mU of tyrosine are indicative of *Ostertagia* infection when calves are naturally infected. Subsequently, researchers have further clarified that Type I *Ostertagiosis* exists when pepsinogen levels are above 5000mU tyrosine and subclinical infections to be between 3000 and 4000mU tyrosine (Hilderson et al., 1989).

Anthelmintics

Traditional Chemicals

There are four classes of chemical dewormers for cattle (Merck & Co., Inc., 2003). These classes include: benzimidazoles, imidazothiazoles, tetrahydropyrimidines, and macrocyclic lactones. Benzimidazoles have been in use for about 40 years (Vercruysse and Dorny, 1999) and include thiabendazole, cambendazole, parabendazole, mebendazole, fenbendazole, oxfendazole, oxibendazole, albendazole, albendazole sulfoxide, thiophanate, febantel, netobimin, and triclabendazole. Special cases among these subclasses are: albendazole and triclabendazole which are active against liver flukes

and triclabendazole, which does not affect roundworms. The most effective of the group are oxfendazole, fenbendazole, and albendazole, which have the longest half-life in the body. Benzimidazoles act by binding to β -tubulin, a structural protein, and block polymerization of β -tubulin into microtubules (Roos, 1993). This damages the integrity and transport functions of intestinal cells within the parasite. The parasite starves to death and is therefore lethal, but can take longer than other classes of anthelmintics to show a response.

Imidazothiazoles are primarily in the form of levamisole, which is given orally or subcutaneously. Levamisole was put on the market over 30 years ago (Vercruysse and Dorny, 1999) and is a cholinergic agonist, which causes neuromuscular paralysis of the parasites. Levamisole is ineffective against inhibited *Ostertagia ostertagi* larvae and against flukes and tapeworms, so is not used alone as an anthelmintic in cattle (Harder et al., 2003).

The macrocyclic lactones are the newest class of anthelmintics, being about 20 years old (Vercruysse and Dorny, 1999), and are produced in two main subclasses: avermectins and milbemycins. Avermectins are further delineated into ivermectin, abamectin, and doramectin. Milbemycins are further delineated into milbemycin oxime and moxidectin. Macrocyclic lactones bind to glutamate-gated chloride channel receptors in the parasite, which causes the channel to open, allowing an influx of chloride ion that leads to paralysis (Merck & Co., Inc., 2003). Simply put, the class binds to receptors on inhibitory neurons, which leads to parasite paralysis.

Macrocyclic lactones can be administered in various forms such as subcutaneous injections, pour-on formulations, and sustained release boluses. There are differing

efficacies among the different formulations, but as a group, this class is highly effective at significantly reducing fecal nematodal egg counts when compared to non-treated animals (Mercier, 2001; Williams, 2003; Loyacano, 2001; Dorny, 2000; Skogerboe, 2000; Sarkunas, 1999, Barth, 1997).

Not only do chemical dewormers consistently and significantly reduce fecal egg counts in treated animals; numerous studies show that the chemical dewormers also consistently increase weight and weight gain in treated animals when compared to untreated animals. Upon a review of over 60 studies where anthelmintic efficacy on weight gain was tested, a majority of them found increases in weight and weight gain in treated animals.

Stromberg et al. (1997) found that over a 2-year repeated study in a beef cow/calf herd, the calves in the treated group that were treated in midsummer after a move to a new pasture significantly ($P < 0.0001$) outgained untreated animals in both years (by 19.3kg in the first year and 13.2kg in the second). Average daily gain over the 2-year study was 0.13 kg greater for the treated calves than the control calves (0.83 vs. 0.70; $P < 0.0001$). Ryan et al. (1997) reported a 34% increase ($P < 0.02$) in weight gain of treated calves ($n=29$) over untreated calves ($n=29$), which was a 33.9kg over 168 days after turnout.

When investigating the different effects of various forms of the avermectin class, Williams et al. (1997) reported that calves ($n=80$ crossbred steer calves; 6 months of age; average weight 208kg) treated with injectable doramectin (DOR; $n=18$), ivermectin injectable (IVM-INJ; $n=18$) and ivermectin pour-on (IVM-PO; $n=18$) formulation gained 159kg, 147kg and 150kg respectively over a 161-day trial that began in-mid January.

While these weights were not significantly different from one another, they were all significantly ($P < 0.05$) higher than the untreated group ($n=18$), which gained only 96kg.

Similarly designed studies were done in Wisconsin ($n=42$; days=140), Arkansas ($n=32$; days=84), Georgia ($n=60$; days=112) and Mississippi ($n=72$; days=112) and reported summarily (Ballweber et al., 1997). In these studies, calves were all weaned, 6-12 months of age, and between 133 to 293 kg in BW. The WI and MS studies used female crossbred calves and AR (crossbred calves) and GA studies (Angus calves) used steers. The results showed that treated animals had a 0.152-0.272kg ADG advantage over animals that were untreated, which was significant in three of the four studies. The differences between gains were only significant in three of the four studies because FEC were low in the trial with insignificant differences.

These results were further confirmed in studies in three different locations lasting 140-day each. Studies were performed in Tennessee (TN) between October 1995 and March of 1996, in Louisiana (LA) between January and June of 1997, and in Wisconsin (WI) between May and September of 1997. All calves were steer beef calves between 6 and 10 months of age and weighed between 155 and 334 kg at the beginning of the trials. All studies except TN used crossbred steer calves; TN used Angus and Angus crossbreds. Average daily weight gain advantages of treated animals over control animals were 0.055kg ($P \leq 0.05$) in TN, 0.208kg ($P \leq 0.05$) in LA, and 0.116kg ($P \leq 0.05$) in WI (Skogerboe et al., 2000).

Many studies will confirm the increase in weight gain with using a chemical anthelmintic, but there are some trials that show no significant differences. Williams et al. (1999) showed only a trend for increased weight on the last 2 weigh dates in a 112-day

trial with 75 crossbred (BrahmanXBrangus) beef calves that were 9-12 months old and weighed on average 216 kg on day 0. On day 84, untreated calves weighed 327 kg and IVM-PO treated animals weighed 338kg, which the authors state is a trend, but give no *P*-value. Similarly, when comparing final weights on day 112, IVM-PO treated animals weighed 358kg and the untreated animals weighed 346, which was trend towards a difference. Barger (1981) found that regular anthelmintic treatment of suckling beef calves usually failed to demonstrate increased weight gains.

When evaluating the weight gain results on parasite studies, many factors need to be taken into consideration. Weather, number of calves, time of year, duration for the trial, nutrition of the diet, pasture contamination, and pen effects can have an impact on how well the animals perform. The trials listed above addressed these issues in their study designs. If weather was too dry or too cool, parasite counts were low and the results were presented with a caveat about the weather. Most studies were conducted over the first year grazing season and lasted for the duration of the whole season. Pasture parasite contamination was measured and reported in each study. If pasture contamination was low, then the results were presented with that in mind.

Logically, if the variables such as weather, number of calves, time of year and duration for the trial, nutrition of the diet and pasture contamination are the same for every treatment group and there is severe enough nematodal contamination to show PGE, the control animals will perform poorly compared to the treated animals since the detrimental effects of parasites is not debatable. However, when comparing the efficacy of chemical dewormers to no treatment, there is more than one option for managing the control group. All of the trials listed above kept their animals in the same pasture

throughout the duration of the trial without any rotation (i.e. set stocked); therefore, the decreased performance in untreated animals is predictable.

Dimander et al. (2003) conducted a three-year grazing experiment from 1998-2000 in Sweden where they compared calves in several treatment groups. Calves in group 1 were set-stocked and untreated. Calves in group 2, 3, and 4 were treated with an ivermectin bolus, nematophagus fungus or a copper wire particle bolus, respectively. Calves in group 5 were left untreated and were turned out onto a pasture that had been grazed by older cows (i.e. low levels of contamination) the year before and then were moved mid-season to a silage aftermath pasture. They reported that weight gain trajectories for the calves that were rotated improved after the move to the silage aftermath plots, which was different than the set stocked animals whose trajectory remained the same. In 1999, the rotation group grew significantly better than all set stocked groups ($P = 0.03$). In 2000, calves in the rotation group gained 65kg ($P < 0.0001$) more than calves treated with the IVM bolus. This study proves that while chemically treated animals perform better than untreated animals that are raised in identical set- stocked conditions, untreated animals can compete with treated animals when raised in conditions that will combat the parasites in different ways.

Parasite Resistance

Parasitic resistance is present in a population when “there is greater frequency of an individual within a population able to tolerate doses of a compound than in a normal population of the same species” (Prichard, 1980). Prichard, 1980 also stated that such resistance is heritable. Occurrence of anthelmintic resistance in different species is ranked from most frequently to least frequent in the following way: goats>sheep>horses>cattle.

Among the specific species of parasites, the ones most resistant are *H. contortus*, *T. circumcincta*, *Trichostrongylus* spp, *Oesophagostomum* spp, and *Cooperia* species (Vercruyse and Dorny, 1999). Of those in the list, *Cooperia* is the only resistant parasite typically reported in cattle. This list shows the infrequency to which cattle parasites develop resistance. Resistance in general is enhanced by the increased anthelmintic usage or pressure and also dosing at suboptimal levels where all worms are not removed, thus ensuring that reinfection will primarily occur through the progeny of survivors.

Parasitic resistance to the benzimidazole class of anthelmintics manifests as a decreased ability of the benzimidazoles to bind to β -tubulin (Prichard, 1990). *Cooperia oncophora*, *O. ostertagi*, and *H. placei* were all found to be resistant to benzimidazoles in Argentina (Mejia, 2003). Williams et al. (1997b) reported that efficiencies of albendazole, fenbendazole, and oxfendazole were lower than expected against immature and adult *O. ostertagi*. Resistance to oxfendazole has been reported in *Cooperia* strains in New Zealand cattle (Jackson et al., 1987; Vermunt et al., 1995) and in *T. axei* strains in Australia (Eagleson and Bowie, 1986). Resistance to albendazole and oxfendazole has been reported in *Haemonchus* species in southern Brazil (Pinheiro and Echevarria, 1990).

Parasite resistance to levamisole and morantel groups prevents such anthelmintics from causing neuromuscular paralysis of the parasites. A species of *Ostertagia* was reported to be resistant to Levamisole as early as 1979 (Sangster), again in Belgium cattle based on an in vitro larval paralysis test (Geerts, 1987), and most recently in Louisiana (Williams, 1991). Instances of *O. ostertagi* strains being resistant to morantel have been reported (Borgsteede, 1988). This resistance was found after using a sustained release

bolus of morantel. This same strain was later found to have a side resistance to Levamisole. Side resistance occurs when a parasite that is resistant to one dewormer becomes resistant to another dewormer that is similar in mode of action to the first one, whether or not it has been exposed to the second dewormer (Prichard et al., 1980).

Macrocyclic lactones cannot bind to the inhibitory neurons when parasites are resistant and paralysis cannot occur (Coles, 2001). Resistance of *Cooperia* to this class of dewormers has been documented in cattle in various parts of Argentina (Fiel, 2001; Anziani et al., 2001; Mejia, 2003) where cattle are treated at frequent intervals throughout the year, in New Zealand (West et al., 1994; Vermunt et al., 1995) in the UK (Coles et al., 1998; Stafford and Coles, 1999), and in Brazil (Echevarria and Pinheiro, 2001). Most startling is the study in New Zealand where 16 out of 18 farms had *Cooperia* resistance to macrocyclic lactones (Familton et al., 2001). This resistance most likely occurred from farmers trying to maximize growth of their cattle with frequent dewormings, which led to pastures being primarily contaminated with parasites that were able to survive anthelmintic treatment.

Resistance in cattle is infrequent because of the way in which cattle are managed. Cattle develop immunity to most parasites shortly after their first grazing season, so the frequency in which they get treated and the proportion of animals who get treated is not as much, for example as in goats whose parasites have high resistance to anthelmintics (Coles, 2002; Vercruysse and Dorny, 1999). In order to prevent development of resistance in cattle, highly effective treatment or a combination of anthelmintics must be used in order for there to be few survivors (Barnes, 1995). Vercruysse and Dorny (1999)

contend that resistance to anthelmintics cannot be prevented, but can only be slowed by carefully managing the frequency and amount of usage.

Resistance to anthelmintics is not only found in parasites of calves that have been treated with anthelmintics previously. Some strains of parasites are resistant to anthelmintics without ever having been treated. Bisset et al. (1990) found that the pour-on formulation of ivermectin failed to control species of *Cooperia* in cattle that had not been treated with the pour-on formulation previously.

There is little expectation of new classes of anthelmintics being developed in the near future to aid in alleviating the problem of increasing resistance to the current classes of anthelmintics. The reason for no new production is that the cost of developing a new anthelmintic from production to market is approximately \$230 million and because current anthelmintics are widely used, very safe for the animals and usually highly effective, the anticipation for new anthelmintics is low (McKellar, 1994). Two other deterrents for new products are the fact that working with cattle in experiments is very expensive and difficult and also the resistance that is so heavily reported is occurring mainly in areas of the world with the smallest population of marketable cattle such as South Africa and South America.

Organic Alternatives

Condensed tannins (CT), as observed by Bertrand (2004), have anthelmintic effects against GI parasites but the mechanism is still unknown. Waghorn et al. (1994) and Terrill et al. (1992) hypothesize that CT increase performance of parasitized animals by decreasing protein degradation in the rumen and therefore, more protein is available to the small intestine for increased absorption and wall repair of the gastrointestinal tract.

Niezen et al. (1995) report highly significant increased performance of lambs feeding on different forages that contain condensed tannins but such results have yet to be reproduced in cattle.

Copper sulphate, a mineral substance that already meets organic farming specifications for plant production, has a strong deworming action against certain parasites, particularly *Haemonchus contortus* and *Trichostrongylus axei*, which affect the abomasum. This dewormer may be administered with a funnel and flexible tube.

Duval (2003) gives a list of many botanical dewormers that farmers may try in lieu of chemical anthelmintics. While the plants below have exhibited anthelmintic effects (many of which have been used for their anthelmintic properties before chemical dewormers were on the market), Duval cautions the user to remember that these plants can be toxic to animals at high levels and should be used with caution.

Garlic (Anonymous, 1953) is a common plant dewormer that is easy to find. It is effective against lungworm in cattle. Garlic should be used as a preventative measure because its anthelmintic mode of action is to prevent the eggs of certain parasites from developing into larvae (Bastidas, 1969). The numerous therapeutic properties of garlic originate mainly from its high sulphur content (Duval, 2003). Garlic juice or garlic milk has been suggested as an alternate form of administration (Grieve, 1971). When giving garlic to dairy cows, farmers should give garlic during or immediately after milking so that the milk does not pick up the garlic taste.

Many species of wormwood such as *absinthium*, *vulgaris*, *cina*, *herba alba* and *dracunculus* have demonstrated deworming properties. Santonin, which is extracted from the Eurasian wormwood acts against many parasites but must be used carefully because it

can lead to eye problems. The dried, powdered shoots of *Artemisia herba-alba* wormwood (a species common to North Africa) has proved to be effective against *Haemonchus* in sheep (Idris, Um El A.A., et al., 1982), but has not been tested in cattle. Duval (2003) suggests that because several wormwood species grow wild in North America, a low-cost option for using wormwood as an anthelmintic would be to incorporate it in pastures where animals would naturally graze it.

There are a couple plants that are effective against liver fluke in ruminants. Cabret (1986) recommends using turpentine that is extracted from pine and various other conifers. The turpentine should be distilled and the spirits that are produced should be added to castor oil. Turpentine spirits can also be mixed with comestible linseed oil, but should be used with caution and given exactly as prescribed since turpentine can cause spasmodic closure of the mouth if it enters the respiratory tract. Common juniper is another conifer that is effective against liver fluke (Duval, 2003).

There are species of plants that have anthelmintic effects that can be grown in the pasture where cattle can readily graze them. The oil in the seeds of tansy flowers (*Tanacetum vulgare*) is used against *Nematodirus* in sheep (Papchenkov, 1968) and should be investigated in cattle because cows readily eat it. Wild ginger or snakeroot (*Asarum canadense*) that grows in wooded areas has been historically used as an anthelmintic purge for horses and cattle, but it requires several years to reproduce. Cabret (1986) states that beech creosote is another biological anthelmintic used against lungworm in ruminants.

Blackberries, raspberries, and young ash and elder shoots are also other plant species with deworming properties that should be accessible in pastures. Duke (1985)

lists the following plants that are naturally grown or can be cultivated in Québec that are anthelmintic: Yarrow (*Achilea millefolium*), which is highly toxic to calves; Sweet flag or calamus (*Acorus calamus*); Agrimony (*Agrimonia*); Roots or root infusions of Indian hemp (*Apocynum cannabinum*); Calendula (*Calendula officinalis*); Hemp (*Cannabis saliva*); Blue cohosh (*Caulophyllum thalictroides*); Lady slipper root extract (*Cypripedium calceolus*); Sweet gale or bog myrtle (*Myrica gale*); Pokeweed (*Phytolacca americana*); Common knotgrass (*Polygonum aviculare*); Rue (*Ruta graveolens*); Bloodroot (*Sanguinaria canadensis*); Savory (*Satureja montana*); Skullcap (*Scutellaria lateriflora*); Skunk cabbage or skunk weed (*Symplocarpus foetidus*); Nettle (*Urtica dioica*) seeds and roots; Valerian (*Valeriana officinalis*); Verbena (*Verbena officinalis*); and Periwinkle (*Vinca minor*).

Biological Control

Waller and Faedo (1996) define biological control of nematode parasites as “the action of natural enemies which maintain a host population at levels lower than would occur in the absence of the enemies”. When introducing a biological control to a pasture, it is crucial that if the enemy or agent is foreign to the local environment, it does not adversely affect non-target organisms. A better alternative would be to enhance the natural agents that are indigenous to the region (Waller and Faedo, 1996).

Duddingtonia flagrans

Biological control can affect the parasites in their parasitic phase inside the host or in the outside environment while the parasite is in the free-living phase. Nematode-trapping microfungi have been shown to be abundant in organic systems and act by germinating and growing in the feces of the host and then trapping and destroying

digesting the free-living stages of the parasites (Larsen, 1999). Larsen et al. (1991) identified *Duddingtonia flagrans* as a nematode-trapping fungus that is found in the soil, and then when ingested by a ruminant, can pass through the rumen and not be digested by the microbes. Subsequent studies have confirmed *D. flagrans*' ability to reduce the nematodal larval populations in the feces and herbage (Gronvold et al., 1993, Wolstrup et al., 1994) and also reduce *O. ostertagi* worm burden in calves that were fed *D. flagrans* (Wolstrup et al., 1994). The success of these fungi as biological control products depends on the interest of the pharmaceutical industry and the price compared to the price of chemical anthelmintics (Vercruysse and Dorny, 1999).

Dung Beetles

Dung beetles are also a biological control that can reduce nematodal larvae in the feces and surrounding herbage in cattle (Fincher, 1973; 1975; Waller and Faedo, 1996) by degrading the dung patty and exposing it to the sun and rain where the eggs can be washed away or dehydrated. However, dung beetles are very susceptible to the avermectin class of anthelmintics. The residue in dung from these anthelmintics can greatly reduce dung beetle populations (Herd et al., 1993; Barth et al., 1993, 1994; Wratten, 1996) and other insects such as flies.

Grazing strategies

Rotational grazing

Grazing strategies aimed at decreasing parasitic load are not meant to be used in isolation, but instead should be used in conjunction with efficient use of anthelmintics, proper nutrition, etc. Foundational research into grazing strategies that will decrease cattle exposure to parasites began in 1969 when Michel proposed his dose and move

system. Michel claimed that nematode infections could be controlled by a combination of preventative grazing strategies and preventative deworming. Based on Michel's claim, he recommended that cattle should be dosed with an anthelmintic and then moved to a fresh pasture before eggs from the previous pasture became infective larvae (Michel, 1969). This type of rotational grazing is a preventative strategy that is based on knowledge of the parasitic life cycle. This system proved effective in controlling *Ostertagia* and *Cooperia* infections (Smeal et al., 1980) and also lungworm infections in calves.

Michel further classified his dose and move system into three categories of grazing strategies: preventative, evasive, and diluting (Michel, 1985). Preventative strategies involved putting worm-free animals on clean pasture and suppressing egg output by treating early in the grazing season. Evasive strategies involved moving calves to a new pasture before the eggs in the current pasture could develop into infective larvae (usually 4 to 6 days). Diluting strategies involve adding animals that will decrease the parasitic load in the pasture, which would decrease the amount of larvae eaten by susceptible stock. This is frequently seen in beef cattle cow-calf operations where the older animals dilute the amount of eggs shed because the adults are immune to parasites. This can also be accomplished by co-grazing or alternate grazing of different species.

Various manipulations of grazing strategies have been researched. In an early study, Ostendorp and Harmsen (1968) found that moving cattle every 2 to 3 weeks prevented a build up of eggs and infective larvae on a given pasture. In a more recent study, Eysker et al. (1998) sought to alleviate the burden on farmers having to move cattle so frequently by trying to find the least amount of times a farmer would have to move his cattle in order to benefit from pasture rotation. After two years of

experimenting, Eysker reported a significant control of gastrointestinal nematode infections when calves were moved at monthly intervals from July onwards and when the last move was 1 month before housing. They did recognize that heavy parasite infections tended to be in the fall when the weather was cooler and that more frequent moves may be required. Among various studies including Oostendorp and Harmsen (1968), Eysker et al. (1998), rotating calves to different pastures to optimize herbage utilization and to stay ahead of the parasite life cycle has proven effective in managing parasites.

Stocking Rate

Another grazing strategy is having a low stocking density of cattle to reduce the amount of infective larvae to which calves are exposed which also reduces helminthosis (Ciordia et al., 1971; Nansen et al., 1988; Shaw et al., 1998).

Alternate grazing

Pasture rotation is in itself an effective parasite management tool, but Michel also recommends the alternate grazing of small ruminants and cattle. The principle of alternate grazing is that the cattle and small ruminants have different infective species of parasites and, by grazing each species together or alternately, the cattle will ingest some of the small ruminant infective larvae and vice versa without risk of cross contamination (Waller, 1997). This practice is widely used as a management practice for sheep and goat operations where anthelmintic resistance is rampant. The recommended ratio of cattle to sheep stock is approximately 1:1 (Niezen, 1996).

Alternating sheep and cattle every 2 to 6 months have led to sheep only having to be drenched 1 to 2 times every three years (Barger and Southcott, 1978) or not at all (Donald et al., 1987). While alternate grazing has been adopted in the small ruminant

industry because of its positive results in the small ruminant, the effects on the cattle are not always beneficial. Lambs grazing with steers led to a decrease in sheep parasites *O. circumcincta* and *N. spathiger*, but an increase in cattle parasites *T. axei* and *C. oncophora* (Arundel and Hamilton, 1975). Jordan et al. (1988) noted similar results by reporting a decrease in sheep parasitism, an increase in lamb production, an increase in cattle parasites and a decrease in calf production in an alternate grazing system.

Bairden et al. (1995) conducted a 4-year study to investigate controlling cattle parasites by alternately grazing sheep and cattle. Cattle parasites were significantly lower up to the second grazing season but, by the end of the study period, the calves' parasite burdens were equal to, or greater than, those of set-stocked control animals. A suggestion for the decrease in efficacy of the system was a possible selection of strains of *O. ostertagi* and *C. oncophora* that were capable of surviving for longer periods of time on the pasture. Due to the positive results in small ruminants, this management practice should be investigated further to determine if there can be beneficial effects on calves as well.

Genetics

While trying to improve the environment's ability to destroy parasites, there is also research that focuses on trying to improve the animal's response to parasites by selecting animals who either develop immunity to parasites more quickly or who consistently gain well in spite of infection. Research has shown that the number of eggs found in the feces of calves during a first season grazing period was significantly related to the calf's sire and that the heritability for that trait was consistently 20 to 30% (Leighton et al., 1989). Mackinnon et al. (1991) reports the heritability of total and

individual species for counts of worm eggs as 0.12-0.25. Estimates of total heritability from other studies range from 0.40-0.93. An obstacle to selecting animals for genetic resistance is the fact that no genetic marker for parasite resistance has been identified yet. Identifying this marker is difficult because researchers have been unable to differentiate between markers for resistance and markers for resilience (Gasbarre et al., 1990).

A second obstacle to genetic selection against parasites is that scientists have reported a negative correlation between cow fertility and resistance to parasite infection. Cows with high fertility rates (higher line's pregnancy rate was 28-31% greater than low line) typically have higher fecal egg counts (258 log(epg) vs. 213 log(epg)) than cows with lower fertility rates (Mackinnon et al., 1990) and having better fertility will take precedence over having lower parasite loads. Similarly, when measuring phenotypic correlations, increased weight gain in tropical cattle is positively correlated with higher FEC (Mackinnon et al., 1991), which leads to decreased resistance as cattle are selected for higher growth. Due to these observations, Kloosterman (1992) concludes that selecting cattle for parasite resistance will never occur.

Milk and weaning age

The role of milk and its affects on GI parasitism has been researched because milk is a component of all calf diets. Some studies have reported higher number of parasites in rats, dogs and cats on milk as opposed to weaned animals (Porter, 1941). On the other hand, some studies have found beneficial effects of milk on parasite infections. Leese (1943) found that strongyle-infected colts that were given cows milk helped to rid the colt of infection. Shorb and Spindler (1947) and Spindler et al. (1944) found a similar reduction in parasites when pigs were given skim milk.

Rohrbacher et al. (1958) conducted 3 experiments aimed at identifying the differences in parasite load in calves on milk alone and calves on a mixture of milk and grain and hay. In the first study, calves were either fed skim milk, whole milk, skim and whole milk, skim milk/grain/hay diet or a whole milk/grain/hay diet. The calves were then experimentally infected with nematodes. Calves on milk alone (irregardless of whether it was skim or whole milk) had fewer and smaller parasites, but gained less than calves on a milk/grain/hay diet. These results are surprising considering the fact that, typically, undernourished calves are more susceptible to parasitism. In experiment 2, calves were either fed a milk/grain/hay diet or a grain/hay diet and were experimentally infected with nematodes. The weaned calves had higher parasite load gained less weight than the calves on milk.

Bovine milk proteins, or components associated with the proteins, reduced the motility of both sheathed and exsheathed L3 *Ostertagia* in an *in vitro* study performed by Zeng et al. (2003). Lower larval motility may reduce worm establishment and be a contributing factor to the smaller burdens of gastrointestinal nematodes in milk-fed animals compared with animals after weaning (Zeng et al., 2003). Milk lowers the pH of the rumen, which Zeng et al. (2001) partly attribute the cause of lower worm establishment in lambs fed only milk to because parasites have previously been shown to die more rapidly *in vitro* at low pH.

Satrijia et al. (1991) reported a lower rate of exsheathed larvae establishment when calves were fed only milk rather than milk, hay and concentrates. The results suggest that the degree of development of the ruminal function influences the establishment of *O. ostertagi*.

LIST OF REFERENCES

- Allan, D. and Johnson, A.W. 1960. A short history of husk. *Veterinary Record* 72, 42–44.
- Anderson, N., Armour, J., Eadie, M. R., Jarrett, W. F. H., Jennings, F. W., Ritchie, J. S. D., & Urquhart, G. M. 1966. Experimental Ostertagia Ostertagi Infections in Calves - Results of Single Infections with 5 Graded Dose Levels of Larvae. *Am. J. Vet. Res.* 27: 1259.
- Anderson, R. C. 2000. *Nematode Parasites of Vertebrates: Their Development and Transmission*. CABI Publishing.
- Anonymous. 1953. Garlic as an anthelmintic. *Veterinary Record*, 65 (28): 436.
- Anziani, O. S., G. Zimmermann, A. A. Guglielmo, R. Vazquez, and V. Suarez. 2001. Avermectin resistance in *Cooperia pectinata* in cattle in Argentina. *Vet. Rec.* 149:58-59.
- Armour, J., M. p. N. o. t. ., and Commonwealth Agricultural Bureaux, 93 pp. 1982. *Bovine Ostertagiasis: a Review and Annotated Bibliography*. In C.P. Ogbourne, Ed. Commonwealth Institute of Parasitology, Farnham Royal, UK.
- Armour, J. 1989. The Influence of Host Immunity on the Epidemiology of Trichostrongyle Infections in Cattle. *Vet. Parasitol.* 32:5-19.
- Arundel, J. H., and D. Hamilton. 1975. Effect of Mixed Grazing of Sheep and Cattle on Worm Burdens in Lambs. *Aust. Vet. J.* 51:436-439.
- Bairden, K., J. Armour, and J. L. Duncan. 1995. A 4-Year Study on the Effectiveness of Alternate Grazing of Cattle and Sheep in the Control of Bovine Parasitic Gastroenteritis. *Vet. Parasitol.* 60:119-132.
- Ballweber, L. R., L. L. Smith, J. A. Stuedemann, T. A. Yazwinski, and T. L. Skogerboe. 1997. The effectiveness of a single treatment with doramectin or ivermectin in the control of gastrointestinal nematodes in grazing yearling stocker cattle. *Vet. Parasitol.* 72:53-68.
- Barger, I. A., and W. H. Southcott. 1978. Parasitism and Production in Weaner Sheep Grazing Alternately with Cattle. *Aust. J. Exp. Agr.* 18:340-346.
- Barger, I. A., & Gibbs, H. C. 1981. Milk-Production of Cows Infected Experimentally with Trichostrongylid Parasites. *Vet. Parasitol.* 9: 69-73.
- Barnes, E. H., R. J. Dobson, and I. A. Barger. 1995. Worm Control and Anthelmintic Resistance - Adventures with a Model. *Parasitol. Today.* 11:56-63.

- Barth, D., E. M. Heinzemutz, R. A. Roncalli, D. Schluter, and S. J. Gross. 1993. The Degradation of Dung Produced by Cattle Treated with an Ivermectin Slow-Release Bolus. *Vet. Parasitol.* 48:215-227.
- Barth, D., M. Karrer, E. M. Heinzemutz, and N. Elster. 1994. Colonization and Degradation of Cattle Dung - Aspects of Sampling, Fecal Composition, and Artificially Formed Pats. *Environ. Entomol.* 23:571-578.
- Barth, D., G. F. Ericsson, B. N. Kunkle, S. Rehbein, W. G. Ryan, and D. H. Wallace. 1997. Evaluation of the persistence of the effect of ivermectin and abamectin against gastrointestinal and pulmonary nematodes in cattle. *Vet. Rec.* 140:278-279.
- Bastidas, G.J. 1969. Effect of ingested garlic on necator americanus and ancylostoma caninum. *Am. J. Trop. Med. Hyg.*, 18 (6): pp 920-923.
- Benz, G. W. 1985. Trichostrongylosis in ruminants. In: S.M. Gaafar, W. E. Howard and R. E. Marsh (Eds.): *Parasites, Pests and Predators*, pp. 237-248. Elsevier Science Publishing Co., Amsterdam.
- Berghen, P., H. Hilderson, J. Vercruysee, and P. Dorny. 1993. Evaluation of Pepsinogen, Gastrin and Antibody-Response in Diagnosing Ostertagiasis. *Vet. Parasitol.* 46:175-195.
- Bisset, S. A., R. V. Brunson, and S. Forbes. 1990. Efficacy of a Topical Formulation of Ivermectin against Naturally Acquired Gastrointestinal Nematodes in Weaner Cattle. *N.Z. Vet. J.* 38:4-6.
- Borgsteede, F. H. M. 1988. The Difference between 2 Strains of Ostertagia-Ostertagi in Resistance to Morantel Tartrate. *Int. J. Parasitol.* 18:499-502.
- Cabret, J. 1986. 167 plantes Pour soigner les animaux. Editions du Point Veterinaire, Maisons-Alfort, France. 192 pages.
- Ciordia, H., W. E. Neville, D. M. Baird, and McCampbe.Hc. 1971. Internal Parasitism of Beef Cattle on Winter Pastures - Level of Parasitism as Affected by Stocking Rates. *Am. J. Vet. Res.* 32:1353.
- Coles, G. C., K. A. Stafford, and P. H. S. MacKay. 1998. Ivermectin-resistant Cooperia species from calves on a farm in Somerset. *Vet. Rec.* 142:255-256.
- Coles, G. C., C. L. Watson, and O. S. Anziani. 2001. Ivermectin-resistant Cooperia in cattle. *Vet. Rec.* 148:283-284.
- Coles, G. C. 2002. Cattle nematodes resistant to anthelmintics: why so few cases? *Vet. Res.* 33:481-489.

- Corwin, R. M. 1997. Economics of gastrointestinal parasitism of cattle. *Vet. Parasitol.* 72:451-457.
- Dimander, S. O., J. Høglund, A. Ugglå, E. Spröndly, and P. J. Waller. 2003. Evaluation of gastro-intestinal nematode parasite control strategies for first-season grazing cattle in Sweden. *Vet. Parasitol.* 111:193-209.
- Donald A.D., F.H.W. Morley, A. Axelsen, J.B. Donnelly and P.J. Waller. 1987. Integration of grazing management and anthelmintic treatment for the control of nematode infections in young sheep. In: JL Wheeler, CJ Pearson and GE Robards, Editors. 1987. *Temperate pastures, their production, use and management*, Australian Wool Corporation/CSIRO, Melbourne. pp. 567–569.
- Dorny, P., and J. Vercruyse. 1998. Evaluation of a micro method for the routine determination of serum pepsinogen in cattle. *Res. Vet. Sci.* 65:259-262.
- Dorny, P., D. Demeulenaere, K. Smets, and J. Vercruyse. 2000. Control of gastrointestinal nematodes in first season grazing calves by two strategic treatments with eprinomectin. *Vet. Parasitol.* 89:277-286.
- Duke, J. A. 1985. *CRC Handbook of medicinal herbs*. CRC Press, Boca Ration, Florida, 677 pgs.
- Duval, J. 2003. A Review: Alternative Methods of Controlling Internal Parasites in Ruminants. University of Aberdeen. Available online: http://www.abdn.ac.uk/organic/organic_14c.php
- Eagleson, J. S., and J. Y. Bowie. 1986. Oxfendazole Resistance in *Trichostrongylus-Axei* in Cattle in Australia. *Vet. Rec.* 119:604-604.
- Echevarria, F., and A. Pinheiro. 2001. Efficiency of anthelmintics in cattle. 18th Int. Conf. W.A.A.V.P., Stresa, Italy p. 147.
- Eysker, M., W. M. van der Aar, J. H. Boersema, J. B. Githiori, and F. N. J. Kooyman. 1998. The effect of repeated moves to clean pasture on the build up of gastrointestinal nematode infections in calves. *Vet. Parasitol.* 76:81-94.
- Familton, A. S., P. Mason, and G. C. Coles. 2001. Anthelmintic-resistant *Cooperia* species in cattle. *Vet. Rec.* 149:719-720.
- Fiel, C. A., C. A. Saumell, P. E. Steffan, and E. M. Rodriguez. 2001. Resistance of *Cooperia* to ivermectin treatments in grazing cattle of the Humid Pampa, Argentina. *Vet. Parasitol.* 97:211-217.

- Fincher, G. T. 1973. Dung Beetles as Biological-Control Agents for Gastrointestinal Parasites of Livestock. *J. Parasitol.* 59:396-399.
- Fincher, G. T. 1975. Effects of Dung Beetle Activity on Number of Nematode Parasites Acquired by Grazing Cattle. *J. Parasitol.* 61:759-762.
- Gasbarre, L. C., E. A. Leighton, and C. J. Davies. 1990. Genetic-Control of Immunity to Gastrointestinal Nematodes of Cattle. *Vet. Parasitol.* 37:257-272.
- Geerts, S., J. Brandt, V. Kumar, and L. Biesemans. 1987. Suspected Resistance of *Ostertagia-Ostertagi* in Cattle to Levamisole. *Vet. Parasitol.* 23:77-82.
- Grieve, M. 1971. *A modern herbal*. Volume 1 Dover, New York, 427 pages.
- Gronvold, J., J. Wolstrup, M. Larsen, S. A. Henriksen, and P. Nansen. 1993. Biological-Control of *Ostertagia-Ostertagi* by Feeding Selected Nematode-Trapping Fungi to Calves. *J. Helminth.* 67:31-36.
- Harder, A., Schmitt-Wrede, H. P., Krucken, J., Marinovski, P., Wunderlich, F., Willson, J., Amliwala, K., Holden-Dye, L., and Walker, R. 2003. Cyclooctadepsipeptides - an anthelmintically active class of compounds exhibiting a novel mode of action. *Int. J. Antimicrob. Agents.* 22: 318-331.
- Hawkins, J. A. 1993. Economic-Benefits of Parasite Control in Cattle. *Vet. Parasitol.* 46:159-173.
- Herd, R., and K. Wardhaugh. 1993. Environmental-Impact of Avermectin Usage in Livestock - Preface. *Vet. Parasitol.* 48:1-2.
- Herrick, J. B. 1990. Hidden losses: conquering coccidia. *Large Animal Veterinarian*, Sept./Oct.: 29-30.
- Hilderson, H., Berghen, P., Vercruyssen, J., Dorny, P., and Braem, L. 1989. Diagnostic-Value of Pepsinogen for Clinical *Ostertagiosis*. *Vet. Rec.* 125: 376-377.
- Idris, Um El A.A., S.E.I. Adam and G. Tartour. 1982. The anthelmintic efficacy of *Artemisia herba-alba* against *Haemonchus contortus* infection in goats. *National Institute of Animal Health Quarterly, Japan*, 22(3):138-143.
- Jackson, R. A., K. G. Townsend, C. Pyke, and D. M. Lance. 1987. Isolation of Oxfendazole Resistant *Cooperia-Oncophora* in Cattle. *N.Z. Vet. J.* 35:187-189.
- Jennings, F. W., Armour, J., Lawson, D. D., and Roberts, R. 1966. Experimental *Ostertagia Ostertagi* Infections in Calves - Studies with Abomasal Cannulas. *Am. J. Vet. Res.* 27: 1249.

- Johnstone, Colin. 1998. Parasites and Parasitic Diseases of Domestic Animals. University of Pennsylvania.
http://cal.vet.upenn.edu/merial/Nematodes/nems_2.htm
- Jordan, H. E., W. A. Phillips, R. D. Morrison, J. J. Doyle, and K. McKenzie. 1988. A 3-Year Study of Continuous Mixed Grazing of Cattle and Sheep - Parasitism of Offspring. *Int. J. Parasitol.* 18:779-784.
- Kloosterman, A., H. K. Parmentier, and H. W. Ploeger. 1992. Breeding Cattle and Sheep for Resistance to Gastrointestinal Nematodes. *Parasitol. Today.* 8:330-335.
- Larsen, M., J. Wolstrup, S. A. Henriksen, C. Dackman, J. Gronvold, and P. Nansen. 1991. Invitro Stress Selection of Nematophagous Fungi for Biocontrol of Parasitic Nematodes in Ruminants. *J. Helminth.* 65:193-200.
- Larsen, M. 1999. Biological control of helminths. *Int. J. Parasitol.* 29:139-146.
- Leighton, E. A., K. D. Murrell, and L. C. Gasbarre. 1989. Evidence for Genetic-Control of Nematode Egg-Shedding Rates in Calves. *J. Parasitol.* 75:498-504.
- Leese AS. 1943. Parasitic aneurism in foal. *Vet. J.* 99: 276.
- Loyacano, A. F., T. L. Skogerboe, J. C. Williams, A. A. DeRosa, J. A. Gurie, and V. K. Shostrom. 2001. Effects of parenteral administration of doramectin or a combination of ivermectin and clorsulon on control of gastrointestinal nematode and liver fluke infections and on growth performance in cattle. *JAVMA.* 218:1465-1468.
- Mackinnon, M. J., D. J. S. Hetzel, N. J. Corbet, R. P. Bryan, and R. Dixon. 1990. Correlated Responses to Selection for Cow Fertility in a Tropical Beef Herd. *Anim. Prod.* 50:417-424.
- Mackinnon, M. J., K. Meyer, and D. J. S. Hetzel. 1991. Genetic-Variation and Covariation for Growth, Parasite Resistance and Heat Tolerance in Tropical Cattle. *Livest. Prod. Sci.* 27:105-122.
- McKellar, Q. A. 1994. Chemotherapy and Delivery Systems Helminths. *Vet. Parasitol.* 54:249-258.
- McLeod, R. S. 1995. Costs Of Major Parasites To The Australian Livestock Industries. *International Journal For Parasitology.* 25(11):1363-1367. Online.
- Mejia, M. E., B. M. F. Igartua, E. E. Schmidt, and J. Cabaret. 2003. Multispecies and multiple anthelmintic resistance on cattle nematodes in a farm in Argentina: the beginning of high resistance? *Vet. Res.* 34:461-467.

- Melancon, J. 2001. Parasites of Dairy Cattle. Veterinary Professional Services, Merial Limited.
- Mercier, P., P. E. Steffan, and C. R. White. 2001. Effects of treatments with endectocide on the weight gain of grazing cattle in a warm temperate climate. *Vet. Rec.* 149:266-269.
- Merck and Co. Inc. 2003. <http://www.merckvetmanual.com>.
- Michel, J. F. 1969. Control of Some Nematode Infections in Calves. *Vet. Rec.* 85:326.
- Michel, J. F. 1985. Strategies for the Use of Anthelmintics in Livestock and Their Implications for the Development of Drug-Resistance. *Parasitol.* 90:621-628.
- Myers, G. H. and R.F. Taylor. 1989. Ostertagiasis in cattle. *J. Vet. Diag.* 1:195–200.
- Nansen, P., J. Foldager, J. W. Hansen, S. A. Henriksen, and R. J. Jorgensen. 1988. Grazing Pressure and Acquisition of Ostertagia-Ostertagi in Calves. *Vet. Parasitol.* 27:325-335.
- Niezen, J. H., Waghorn, T. S., Charleston, W. A. G., and Waghorn, G. C. 1995. Growth and Gastrointestinal Nematode Parasitism in Lambs Grazing Either Lucerne (Medicago-Sativa) or Sulla (Hedysarum-Coronarium) Which Contains Condensed Tannins. *Journal of Agricultural Science* 125: 281-289.
- Niezen, J. H., W. A. G. Charleston, J. Hodgson, A. D. Mackay, and D. M. Leathwick. 1996. Controlling internal parasites in grazing ruminants without recourse to anthelmintics: Approaches, experiences and prospects. *Int. J. Parasitol.* 26:983-992.
- Organic Livestock Research Group. 2000. VEERU, The University of Reading. ISBN 0-70-491093-5. Available online: <http://www.organic-vet.reading.ac.uk/Cattleweb/Home.htm>.
- Oostendorp, D. and Harmsen, H.E., 1968. Agricultural control measures against intestinal parasites in cattle. *Neth. J. Agric. Sci.* 16, pp. 177–185.
- Pandey, V.S. (1972) Effect of temperature on development of the free-living stages of *Ostertagia ostertagi*. *Journal of Parasitology* 58, 1037–1041.
- Papchenkov, N. Y. 1968. Tanacetum vulgare seed and naphtaman against Nematodirus infections in sheep. *Veterinarya Mosk.*, 45 (8): 48-49.
- Parkins, J. J., K. Bairden, and J. Armour. 1982. Ostertagia-Ostertagi in Calves - a Growth, Nitrogen-Balance and Digestibility Study Conducted During Winter

- Feeding Following Thiabendazole Anthelmintic Therapy. *J. Comp. Pathol.* 92:219-227.
- Perry, B. D. and T. F. Randolph. 1999. Improving the assessment of the economic impact of parasitic diseases and of their control in production animals. *Veterinary Parasitology.* 84(3-4):145-168. Online.
- Pinheiro, A. C., and F. A. M. Echevarria. 1990. Susceptibility of *Haemonchus* Spp in Cattle to Anthelmintic Treatment with Albendazole and Oxfendazole. *Pesquisa Vet. Brasil.* 10:19-21.
- Ploeger, H. W. and A. Kloosterman. 1989. The Economic-Impact Of Nematode Parasitism In Cattle. *Magyar Allatorvosok Lapja.* 44(5):272-275. Online.
- Porter DA. 1941. The effect of milk diet on the development of *Haemonchus contortus* in calves. *Journal of Parasitology* 27(supplement), 18-9.
- Prichard, R. K., C. A. Hall, J. D. Kelly, I. C. A. Martin, and A. D. Donald. 1980. The Problem of Anthelmintic Resistance in Nematodes. *Aust. Vet. J.* 56:239-251.
- Prichard, R. K. 1990. Anthelmintic Resistance in Nematodes - Extent, Recent Understanding and Future-Directions for Control and Research. *Int. J. Parasitol.* 20: 515-523.
- Rohrbacher GH, Porter DA, Herlich H. 1958. The effect of milk in the diet of calves and rabbits upon the development of Trichostrongylid nematodes. *Am. J. of Veterinary Research* 19, 625-31.
- Roos, M. H., Kwa, M. S. G., Veenstra, J. G., Kooyman, F. N. J., and Boersema, J. H. 1993. Molecular Aspects of Drug-Resistance in Parasitic Helminths. *Pharm. Therap.* 60: 331-336.
- Ross, J. G., Purcell, D. A., Dow, C., and Todd, J. R. 1967. Experimental Infections of Calves with *Trichostrongylus Axie* - Course and Development of Infection and Lesions in Low Level Infections. *Res. Vet. Sci.* 8: 201.
- Ryan, W. G., R. J. Crawford, S. J. Gross, and D. H. Wallace. 1997. Assessment of parasite control and weight gain after use of an ivermectin sustained-release bolus in calves. *JAVMA.* 211:754.
- Sangster, N. C., H. V. Whitlock, I. G. Russ, M. Gunawan, D. L. Griffin, and J. D. Kelly. 1979. *Trichostrongylus-Colubriformis* and *Ostertagia-Circumcincta* Resistant to Levamisole, Morantel Tartrate and Thiabendazole - Occurrence of Field Strains. *Res. Vet. Sci.* 27:106-110.

- Sarkunas, M., A. Malakauskas, P. Nansen, J. W. Hansen, and V. Paulikas. 1999. Effect of strategic treatments with ivermectin on parasitism of set-stocked calves exposed to natural trichostrongyle infection in Lithuania. *Acta Vet. Scand.* 40:163-171.
- Satrija, F., P. Nansen, C. M. Christensen. 1991. Ostertagia-ostertagi in neonatal calves – Establishment of infection in ruminating and non-ruminating calves. *Res. Vet. Sci.* 51(3): 344-346.
- Schaper, R., and A. Liebisch. 1991. Influence of a Systemic Acting Antiparasitic Drug (Ivermectin) on the Dung Biocoenosis and Dungdegradation of Grazing Cattle. *Tierarztl. Umschau.* 46:12-18.
- Selman, I.E., J. Armour, F.W. Jennings and J.F.S. Reid. 1977. Interpretation of the plasma pepsinogen test (letter). *Vet. Rec.* **100** (1977), pp. 249–250.
- Shaw, D. J., J. Vercruyse, E. Claerebout, and P. Dorny. 1998. Gastrointestinal nematode infections of first-grazing season calves in western Europe: Associations between parasitological, physiological and physical factors. *Vet. Parasitol.* 75:133-151.
- Shorb D. A. and L. A. Spindler. 1947. Growth rate of pigs fed skim milk to control intestinal parasites. *Proceedings of the Helminthological Society of Washington* 14, 30-4.
- Skogerboe, T. L., L. Thompson, J. M. Cunningham, A. C. Brake, and V. K. Karle. 2000. The effectiveness of a single dose of doramectin pour-on in the control of gastrointestinal nematodes in yearling stocker cattle. *Vet. Parasitol.* 87:173-181.
- Smeal, M. G., G. G. Robinson, and G. C. Fraser. 1980. Seasonal Availability of Nematode Larvae on Pastures Grazed by Cattle in New-South-Wales. *Aust. Vet. J.* 56:74-79.
- Snider, T.G., J.C. Williams, D.S. Sheehan and R.H. Fuselier. 1981. Plasma pepsinogen inhibited larval development, and abomasal lesions in experimental infections of calves with *Ostertagia ostertagi*. *Vet. Parasitol.* **8** (1981), pp. 176–183.
- Sommerville, R. I. 1953. Development of *Ostertagia circumcincta* in the abomasal mucosa of sheep. *Nature (Lond.)*. 171:482–483.
- Spindler LA, Zimmerman HE. Effect of skim milk on the growth and acquisition of parasites by pigs under conditions of constant exposure to infection. *Proceedings of the Helminthological Society of Washington* 11, 49-54, 1944
- Stadelmann, H. 1892. Ueber den anatomischen Bau des *Strongylus convolutus* Ostertag nebst einigen Bemerkungen zu seiner Biologie. *Archiv für Naturgeschichte* 1, 149–176.

- Stafford, K., and G. C. Coles. 1999. Nematode control practices and anthelmintic resistance in dairy calves in the south west of England. *Vet. Rec.* 144:659-661.
- Stödter, W. (1901) Die Strongyliden in dem Labmagen der gazähmten Wiederkäuer und die Magenwurmseuche. Dissertation, Hamburg, 108 pp.
- Stromberg, B. E., R. J. Vatthauer, J. C. Schlotthauer, G. H. Myers, D. L. Haggard, V. L. King, and H. Hanke. 1997. Production responses following strategic parasite control in a beef cow/calf herd. *Vet. Parasitol.* 68:315-322.
- Terrill, T. H., Douglas, G. B., Foote, A. G., Purchas, R. W., Wilson, G. F., and Barry, T. N. 1992. Effect of Condensed Tannins Upon Body Growth, Wool Growth and Rumen Metabolism in Sheep Grazing Sulla (*Hedysarum-Coronarium*) and Perennial Pasture. *J. Agric. Sci.* 119: 265-273.
- Threlkeld, W.L. 1934. The life history of *Ostertagia circumcincta*. *Technical Bulletin* No. 5, Virginia Polytechnic Institute, Virginia Agricultural Experimental Station, Blacksburg, Virginia, 24 pp.
- Vercruyse, J., Dorny, P., Berghen, P., and Geeraerts, J. 1986. Abomasal Parasitism in Dairy-Cows in Belgium. *Vet. Parasitol.* 22: 285-291.
- Vercruyse, J., and P. Dorny. 1999. Integrated control of nematode infections in cattle: A reality? A need? A future? *Int. J. Parasitol.* 29:165-175.
- Vermunt, J. J., and D. M. West. 1994. Predicting Copper Status in Beef-Cattle Using Serum Copper Concentrations. *N.Z. Vet. J.* 42:194-195.
- Vermunt, J. J., D. M. West, and W. E. Pomroy. 1995. Multiple Resistance to Ivermectin and Oxfendazole in *Cooperia* Species of Cattle in New-Zealand. *Vet. Rec.* 137:43-45.
- Waghorn, G. C., Shelton, I. D., McNabb, W. C., and McCutcheon, S. N. 1994. Effects of Condensed Tannins in *Lotus-Pedunculatus* on Its Nutritive-Value for Sheep .2. Nitrogenous Aspects. *Journal of Agricultural Science* 123: 109-119.
- Waller, P. J., and M. Faedo. 1996. The prospects for biological control of the free-living stages of nematode parasites of livestock. *Int. J. Parasitol.* 26:915-925.
- Waller, P. J. 1997. Sustainable helminth control of ruminants in developing countries. *Vet. Parasitol.* 71:195-207.
- West, D. M., J. J. Vermunt, W. E. Pomroy, and H. P. Bentall. 1994. Inefficacy of Ivermectin against *Cooperia* Spp Infection in Cattle. *N.Z. Vet. J.* 42:192-193.

- Williams, J. C. 1991. Efficacy of Albendazole, Levamisole and Fenbendazole against Gastrointestinal Nematodes of Cattle, with Emphasis on Inhibited Early 4th Stage Ostertagia-Ostertagi Larvae. *Vet. Parasitol.* 40:59-71.
- Williams, J. C., A. F. Loyacano, A. DeRosa, J. Gurie, D. F. Coombs, and T. L. Skogerboe. 1997. A comparison of the efficacy of two treatments of doramectin injectable, ivermectin injectable and ivermectin pour-on against naturally acquired gastrointestinal nematode infections of cattle during a winter-spring grazing season. *Vet. Parasitol.* 72:69-77.
- Williams, J. C., A. DeRosa, Y. Nakamura, and A. F. Loyacano. 1997. Comparative efficacy of ivermectin pour-on, albendazole, oxfendazole and fenbendazole against *Ostertagia ostertagi* inhibited larvae, other gastrointestinal nematodes and lungworm of cattle. *Vet. Parasitol.* 73:73-82.
- Williams, J. C., A. F. Loyacano, A. DeRosa, J. Gurie, B. C. Clymer, and F. Guerino. 1999. A comparison of persistent anthelmintic efficacy of topical formulations of doramectin, ivermectin, eprinomectin and moxidectin against naturally acquired nematode infections of beef calves. *Vet. Parasitol.* 85:277-288.
- Williams, J. C., and A. DeRosa. 2003. Dose confirmation of moxidectin 0.5% pour-on against adults and fourth-stage larvae of various *Cooperia* spp. and *Trichostrongylus colubriformis* in Louisiana. *Vet. Parasitol.* 114:295-303.
- Wolstrup, J., J. Gronvold, S. A. Henriksen, P. Nansen, M. Larsen, H. O. Bogh, and B. Ilsoe. 1994. An Attempt to Implement the Nematode-Trapping Fungus *Duddingtonia-Flagrans* in Biological-Control of *Trichostrongyle* Infections of 1st Year Grazing Calves. *J. Helminth.* 68:175-180.
- Wratten, S. D., and A. B. Forbes. 1996. Environmental assessment of veterinary avermectins in temperate pastoral ecosystems. *Ann. Appl. Biol.* 128:329-348.
- Xiao, L., H. C. Gibbs, and C. Yang. 1991. Pathophysiologic Effects of *Ostertagia-Ostertagi* in Calves and Their Prevention by Strategic Anthelmintic Treatments. *Am. J. Vet. Res.* 52:1706-1711.
- Zeng S., D. E. B. Lawton, S. M. C. Przemec, D. C. Simcock, and H. V. Simpson. 2001. Reduced *Ostertagia circumcincta* burdens in milk-fed lambs. *NZ Vet J.* 49(1): 2-7.
- Zeng S., S. Brown, M.C. Przemec, and H. V. Simpson. 2003. Milk and milk components reduce the motility of *Ostertagia circumcincta* larvae in vitro. *NZ Vet J.* 51(4): 174-178.

**RUNNING HEAD: DAIRY STEERS, WEAN AGE, DEWORMING, AND
GROWTH**

**Effects of Milk Feeding Period and Anthelmintic Treatment on Fecal Egg Counts
and Growth in Pastured Dairy Steers.**

**B. M. Thompson,* S. P. Washburn,*¹ J.- M. Luginbuhl,*[†] B. A. Hopkins,* and H.
M. Glennon***

North Carolina State University, Raleigh 27695

*Department of Animal Science.

[†]Department of Crop Science

¹ Corresponding Author:

Steven P. Washburn

North Carolina State University

Box 7621

Raleigh, NC 27695

TEL: 919-515-7726

FAX: 919-515-2152

email: steve_washburn@ncsu.edu

Introduction

Internal parasites have been shown to decrease weight gain, negatively impact reproductive measures in cows and reduce overall health of calves, which leads to severe economic losses in production (McLeod, 1995, Perry and Randolph, 1999). Estimating the economic losses of cattle on pasture is difficult, though, because of having to factor in variations in cattle, parasite infections, weather, management, etc. (Hawkins, 1993). The exact mechanisms of how parasites cause decreased growth and other clinical signs are unclear, but scientists hypothesize that due to damage to the abomasal or intestinal wall by inhabitation of parasites, amino nitrogen is sequestered to the gastrointestinal tract for repair instead of being partitioned for growth and development (Hawkins, 1993).

Ostertagi is one of the most important helminthes in cattle in temperate climates (Myers, 1989); Vercruysse et al., 1986). The optimal temperature for hatching and developing into infective L3 larvae is 25°C, which makes areas with temperate climates highly susceptible to these parasites (Anderson, 2000). Clinical cases of Ostertagiosis are rare due to prophylactic dewormings common on most dairy farms, but the subclinical infections that commonly exist can cause detrimental effects on the calves (Berghen, et al., 1993).

Ivermectin is in the macrocyclic lactone class of chemical anthelmintic that acts on parasites such as *Ostertagia* by binding to glutamate-gated chloride channel receptors in the parasite, which causes the channel to open, allowing an influx of chloride ion that leads to paralysis (Merck & Co., Inc., 2003). However, parasitic resistance to this class of dewormers has been documented in cattle in various parts of Argentina (Fiel, et al., 2001), in New Zealand (Familton, et al., 2001, West, et al., 1994) in the UK (Coles, et al.,

1998), and in Brazil (Echevarria and Pinheiro, 2001) and may become a reality in the United States if anthelmintics are used without correct dosing or used too frequently.

Generally, chemical anthelmintics significantly reduce fecal egg counts (Barth, et al., 1997, Dorny, et al., 2000, Loyacano, et al., 2001, Mercier, et al., 2001, Sarkunas, et al., 1999, Skogerboe, et al., 2000, Williams and DeRosa, 2003) and increase weight and weight gain in treated calves when compared to untreated calves (Ballweber, et al., 1997, Ryan, et al., 1997, Skogerboe, et al., 2000, Stromberg, et al., 1997, Williams, et al., 1997). However, there are some trials that show no significant differences in weight and weight gains between treated and untreated cattle (Williams, et al., 1999).

In the southeastern part of the United States, where summers are extremely hot and humid and the winters are not cold enough for long enough to impact the parasite life cycle (Anderson, 2000), alternative strategies and whole farm approaches to decreasing parasite burdens in calves, especially in the first grazing season, have to be taken. Rotational grazing is a management strategy that is used on pasture-based dairies to utilize pasture efficiently and also is effective at breaking the parasitic life cycle. Calves that are rotationally grazed and not dewormed have been shown to gain similarly to calves that are treated and not rotationally grazed (Dimander, et al., 2003). Also, increasing weaning age of calves can possibly combat parasites because bovine milk contributes to fewer gastrointestinal nematodes and comparable weight gains in milk-fed animals compared with animals after weaning (Satrijia et al., 1991; Zeng et al., 2001; Zeng et al., 2003).

Developing alternatives to chemical anthelmintics via organic dewormers and integrated pasture management for the control of nematodal parasites is important to the

dairy cattle industry because of concerns about parasite resistance to anthelmintics and also due to increased interest in organic farming over the past several years. This trial was designed to investigate if pasture-reared dairy steers that are weaned at 12 wk vs 6 wk would have similar or different growth responses after being treated or not treated with ivermectin.

MATERIALS AND METHODS

Animals and treatment

Two phases comprised the study at the Center for Environment Farming Systems (CEFS) in Goldsboro, North Carolina (Figure 1). Phase 1 extended from birth (Oct, 2003- Feb, 2004) to July 15, 2004, with measurements taken according to age of individual calves. Phase 2 started on July 15, 2004, and ended on Nov 18, 2004, with measurements taken at calendar intervals. Thirty-six Holstein ($n = 15$) and Jersey x Holstein steer calves were blocked into a 2x2 factorial arrangement of treatments (Table 1) according to birth weight and breed. Fifty percent Holstein steers ($n = 9$) were crosses using a 100% Jersey sire and 100% Holstein dam. Twenty-five percent Holstein steers ($n = 4$) were crosses using a 100% Jersey sire to a 50% Jersey-Holstein dam. Seventy-five percent Holstein steers ($n = 8$) were crosses using a 100% Holstein sire to breed a 50% Jersey-Holstein dam.

Treatment group 1 consisted of 9 calves (birth weight = 35.7 ± 1.6 kg, $75 \pm 11\%$ Holstein) weaned at 6 wk and treated with ivermectin pour-on formulation (1ml/ 10kg BW) at 12 wk and 20 wk of age and then again on July 15 and September 23. Treatment group 2 consisted of 9 calves (birth weight = 35.1 ± 1.6 kg; $75 \pm 7\%$ Holstein) weaned at 6 wk but not treated with ivermectin. Treatment group 3 consisted of 9 calves (birth weight = 35.2 ± 1.7 kg; $64 \pm 12\%$ Holstein) weaned at 12 wk and treated with ivermectin pour-on formulation (1ml/ 10kg BW) at 12 wk and 20 wk and then on July 15 and September 23. Treatment 4 consisted of 9 calves (birth weight = 35.6 ± 1.7 kg; $75 \pm 9\%$ Holstein) weaned at 12 wk but not treated with ivermectin. Weights from each calf were taken at birth and then at 4-wk intervals until the end of the trial (November 18, 2004).

At the end of Phase 2, weights were taken on 2 d (November 17 and November 18) and were averaged and then reported as final weight.

Feeding management

After birth, calves were put into individual hutches and given 3.8L colostrum within 12 hours of birth. Calves were provided with fresh water, 3.8L whole milk from the morning milking (first from a bottle and then transitioned to drinking out of a bucket), and a fresh allotment of a commercial calf starter grain ration daily. When calves were successfully drinking milk out of a bucket, they were transferred to pasture in groups of similar age (regardless of treatment) and group-fed whole milk once each day from a common trough at a rate of 7.6 liters per calf per day until weaning to increase the likelihood that all calves received an ample amount of milk. The calves readily drank all the milk daily.

The starter ration that the calves received in the hutch included a coccidiostat, decoquinate (10mg/lb), and was fed on pasture to each group of similarly aged calves until 12 wk of age in a trough, regardless of weaning age. The starter ration was fed at a rate of 0.9kg/calf/day both in the hutch and on pasture. Orts were not recorded and daily group intakes were not obtained. After 12 wk of age, calves were supplemented throughout the trial with a concentrate ration made onsite at a rate of 2.27 kg/head/day.

Calves grazed on a pasture used only for seasonally-born calves from a few days after birth through at least 12 to 20 wk of age depending on availability of pasture. Afterwards, calves were switched to a location (where older calves and yearlings had grazed previously the year before) with larger pasture areas to meet the increased DMI requirements of older calves. Calves born in the winter (December through February)

were similarly raised on calf-only pastures through weaning but were moved sooner after weaning to pastures that had been previously grazed by older calves than were calves born in the fall (October through November). Overall grazing management involved moving calves grouped by similar ages to fresh paddocks within each pasture every 2 to 3 days based on herbage stand height in the paddocks. While paddock DM estimates (kg/hectare) were not measured, eye estimates were taken to estimate stand height and calves were moved to fresh paddocks when the stand was approximately 5 to 10 cm tall.

In the cool season months (October to January with limited amounts in February and March), calves grazed annual ryegrass (*Lolium multiflorum* Lam.). On the second pasture site, both annual ryegrass and a mixture of fescue and clover were provided from March through May. In the warm season months (June through September), calves grazed either perennial bermudagrass (*Cynodon dactylon* L.) or a mix of summer annuals (crabgrass-*Digitaria sanguinalis* L., millet-*Setaria italica* L., and sorghum/sudangrass-*Sorghum bicolor* L.). All feed samples were analyzed at Cumberland Valley Analytical Services, Inc., Hagerstown, Maryland (Table 2).

Assessment of parasitic load

Starting at 4 wk of age, rectal fecal samples were collected from each calf at 4-wk intervals throughout the trial and analyzed at North Carolina State University for fecal egg counts using the Modified Wisconsin Sugar Fecal Worm Egg Flotation Method. Individual fecal samples were stirred thoroughly and a 3-gm subsample was placed into a cup. The subsample was manually mixed with 10mL of sugar solution (454 gm of table sugar in 355 ml of very hot water and then cooled at 4° C). The mixture was filtered through a tea strainer and then poured into a tapered bottom test tube. The tube was

placed in a free-swinging centrifuge and a 22 x 22mm cover slip was placed on top of the tube. The tube was centrifuged for 10 min and then the coverslip was removed and placed on a microscope slide. The total number of nematode eggs on each slide was counted, then the total was divided by 3 gm and eggs per gram (epg) were reported. Each sample was analyzed in duplicate and the average of the two results were reported.

For Phase 1, April sampling is the composite of fecal egg counts of each treatment group from April 1 - 22. May sampling is the composite of fecal egg counts of each treatment group from April 29 - May 20. June sampling is the composite of fecal egg counts of each group from May 27 - June 17. July sampling is the composite of fecal egg counts from June 24 - July 15. During Phase 2, fecal samples were gathered on July 15, July 29, August 26, September 23, October 21, and November 18 for FEC analysis.

Statistical analyses

Body weight and ADG were compared (during Phase 1, Phase 2 and Phases 1& 2 combined) among treatment, weaning age, breed (% Holstein), birth season (fall-born = October and November; winter-born = December through February), and their interactions. The final reduced model included treatment, weaning age, treatment*weaning age, breed, and birth season.

Fecal egg counts (FEC) during Phases 1 and 2 were compared among treatment, weaning age, breed (% Holstein), birth season (fall born = October and November; winter-born = December through February), and their interactions. The final reduced model for Phase 1 was the same as for BW and ADG. The final reduced model for Phase 2 included treatment, weaning age, treatment*weaning age, breed, birth season and birth

season*treatment. All analyses were run through the GLM procedure of SAS (SAS version 8.02, 1999).

RESULTS

Weight and ADG

Phase 1. Neither main effect of weaning age (6wk: 0.77 ± 0.03 kg/d; 12wk: 0.73 ± 0.03 kg/d) or anthelmintic treatment (Figure 2) had significant ADG differences within levels throughout Phase 1. Neither weight (Figure 3a, Table 3) nor ADG differed between treatment groups (Figure 3b, Table 3). Crossbred calves that were 25% Holstein weighed less than calves that were 50%, 75% or 100% Holstein at the beginning of phase 1 (29.4 ± 2.3 kg, 36.2 ± 1.8 kg, 37.0 ± 1.8 kg, and 39.0 ± 1.2 kg respectively; $P \leq 0.01$). Also during this phase, breed tended ($P = 0.08$) to have a significant effect on ADG with 25% Holstein calves having a slower rate of gain than 100% Holsteins (0.68 ± 0.05 kg/d vs. 0.82 ± 0.03 kg/d; $P < 0.05$). The calves that were 25% Holstein gained slower than 50% (0.74 ± 0.04 kg/d) and 75% (0.76 ± 0.03 kg/d) Holsteins, but not significantly slower. Fall-born calves had higher rates of gain than for the younger, winter-born calves (Table 4).

Phase 2. Over Phase 2, ADG was not significantly different between calves weaned at 6 wk or 12 wk (0.38 ± 0.03 kg/d and 0.36 ± 0.03 kg/d, respectively). However, calves weaned at 6 wk tended to weigh more than calves weaned at 12 wk on certain dates (Figure 4). Dewormed animals gained faster than untreated animals from July until November (Figure 2). No interactions between weaning age and treatment were seen during Phase 2 (Figure 3a, 3b; Table 3). Similarly to Phase 1, 25% Holstein calves weighed less than the 100% Holstein steers (Figure 5). Throughout Phase 2, the older, fall-born calves had similar gains to those of the younger winter-born calves (Table 4).

Phases 1 and 2 combined. Calves weaned at 6 wk tended ($P = 0.06$) to have higher ADG than calves weaned at 12 wk (0.63 ± 0.02 kg/d vs. 0.59 ± 0.02 kg/d). Dewormed animals gained similarly to untreated animals (Figure 2). Average daily gain over Phases 1 and 2 combined showed that fall-born calves had higher gains than the younger winter-born calves based on the advantage observed in Phase 1 (Table 4).

Fecal egg counts

Phase 1. Phase 1 FEC are reported as eggs per gram (lsmean epg +/- S. E.) for April, May, June, and July. Weaning age had no significant effect on fecal egg counts. Treated calves had lower FEC than calves not receiving ivermectin only in April (2 ± 6 epg vs. 20 ± 6 epg; $P < 0.05$). Interactions were not significant during this phase (Figure 6a). There was a trend ($P = 0.07$) towards a weaning age*treatment interaction in July. Untreated calves that were weaned at 12 wk (group 4) tended to have higher FEC than untreated calves that were weaned at 6 wk (526 ± 118 epg vs. 229 ± 112 epg; $P = 0.06$) and also higher FEC than treated calves who were weaned at 12 wk (203 ± 116 epg; $P = 0.05$). Fall-born calves tended to have higher FEC than winter-born calves in April (19 ± 5 epg vs. 2 ± 8 epg; $P = 0.06$; Figure 7) and had significant higher counts in May (50 ± 6 epg vs. 23 ± 11 epg; $P < 0.05$; Figure 7).

Phase 2. Treated calves had lower FEC than untreated animals at 2 ($P < 0.001$) and 6 wk ($P < 0.01$) after the July 15 anthelmintic treatment and also 4 ($P = 0.001$) and 8 ($P < 0.01$) wk after the September 23 treatment (Figure 6b, Table 5). As opposed to Phase 1, fall-born calves had lower FEC than winter-born calves throughout phase 2 (Table 5; Figure 7). On July 15, there was a trend towards weaning age*treatment interaction by which treated calves that were weaned at 12-wk had lower FEC than

untreated weaned calves of the same weaning age (305 ± 144 epg vs. 698 ± 142 epg; $P = 0.06$) whereas calves weaned at 6 wk had intermediate FEC whether dewormed (488 ± 140 epg) or not (359 ± 134 epg). An interaction of treatment with birth season was significant ($P < 0.05$) on July 29 and October 21 and tended to be significant ($P = 0.08$) on July 15. In this case, untreated winter-born calves usually had higher FEC compared to their ivermectin-treated contemporaries relative to comparisons of FEC between treated and untreated fall-born calves.

DISCUSSION

Weight gain and ADG

Our hypothesis that increasing the weaning age of untreated calves will allow such calves to compete with treated calves was not supported by this research. Calves that were weaned at 12 wk did not perform better than those weaned at 6 wk. In fact, the calves weaned at 6 wk tended to have increased gain over all of the study and tended to weigh more at the end of the study. These results are contradictory to studies that show comparable or better weights for animals on milk for longer periods of time or for calves who intake milk as the sole dietary component (Satrijia et al., 1991; Zeng et al., 2001; Zeng et al., 2003). Calves that were weaned later may have had reduced grain or pasture intake (neither of which was measured directly), which could delay rumen development and thereby affect subsequent growth. Hopkins (1997) reported that calves weaned at 4 wk vs 8 wk compensated by increasing intake of starter ration after early weaning such that growth rates were similar through 180 d of age.

The lack of treatment effect on BW or ADG could be due to a couple of factors. First of all, the parasite load may not have been sufficient in any of the animals to cause much of an effect on growth. Most of the groups stayed below 500 epg with only 3 untreated calves weaned at 12 wk being above 500 epg. Because fecal egg counts do not always correlate well with worm burdens during the first-grazing season (Ploeger et al., 1994; Eysker and Ploeger, 2000), the extent of parasitism in the treated versus untreated groups is not certain. Parasitized animals that have lower weight gain due to the parasites can shed anywhere from several hundred of eggs per gram of feces to several thousand (Anderson et al., 1965; Hilderson et al., 1987).

Secondly, pasture rotation management of the calves could have sufficiently prevented them from being continually reinfected with infective L3 larvae. Pasture rotation as a means for controlling parasite infection was introduced by Michel (1969;1985) who suggested moving calves to a fresh pasture before eggs from the previous pasture became infective larvae. This type of rotational grazing is a preventative strategy that is based on knowledge of the parasitic life cycle. This system proved effective in controlling *Ostertagia*, *Cooperia* and lungworm infections in calves (Smeal, 1978; Smeal et al., 1980; Jorgensen, 1981; Eysker, 1995; 1998; Ostendorp and Harmsen, 1968). It is logical to expect that the older animals would gain faster than the younger animals (fall-born vs. winter-born, Table 3) but some of the differences may be related to having a longer period of time on pastures, which had not been exposed to older animals for several months.

It is interesting to note how the only differences in weight and gain between different breeds occurred between the 25% Holstein and the 100% Holstein steers (Figure 5). However, numerical means for body weights were increasingly greater with higher percentages of Holstein. Performance data, such as BW and ADG, on crossbred calves has not been widely reported.

Fecal egg counts

Managing the calves on pastures that had not been previously grazed by older cattle was important because as animals age, they develop an immune response which develops because of repeated exposure to parasites. The developed immunity is not absolute, so older growing animals or adult cows may still be infected with nematodes

and can serve as a source of pasture contamination and infection for younger cattle that graze after them (Melancon, 2001).

The fecal egg count trend (Figure 6a) shows a peak in late spring/beginning of summer and in the late summer/early fall and a drop in between where temperatures are hot and dry which is consistent with what is known of the life cycle of *Ostertagia*. We would have expected to see the peak in egg counts occur before July 15 and the low counts occur for a longer period of time.

The fecal egg count data also show an reduction in FEC to <100 epg with ivermectin treatment for at least 42 days post treatment in Phase 2 (Figure 6b) which is longer than the reported residual efficacy of macrocyclic lactones in other studies (Barth, et al., 1997; Yazwinski et al., 1994). Variation in persistency between studies can be due to animal differences such as breed, nutritional status, and amount of body fat, and also parasite factors such as level of infection (Barth, 1997).

The fall-born calves had higher fecal egg counts in April and May than winter-born calves because the fall-born calves had been exposed to pastures since January that had been previously grazed by older calves who had been shedding eggs; whereas the younger calves had only been moved to the infected pastures in March. This indicates that calves that are raised on pastures not previously grazed by older calves (that shed parasite eggs) will have lower parasitic infection and burden. It is likely that these older calves ingested overwintered larvae late in the winter that matured in the spring and began to hatch. This trend reversed to some extent in the summer as the fall-born calves approached being yearling when resistance to parasites begins to set in. The fact that the older animals had less fecal egg counts than younger calves during late summer and fall

would suggest that such calves had begun to develop immunity to parasites, as calves do as they approach one year of age; however, no indicators of immunity other than fecal egg counts can substantiate this hypothesis.

Interactions between treatment and weaning age in fecal egg counts were rare in this study and not strong when they did exist. Only 1 calf out of the 27 in groups 1 to 3 ever had a fecal egg count over 600epg, but in treatment group 4, there were 3 of 9 calves that had FEC between 600 and 1800 epg. The higher FEC of these 3 calves explain all of the significant interactions observed, because all such interactions involved treatment group 4. If these 3 calves were removed from the study or more evenly dispersed over the other groups, the interactions would not have existed. Overall, spring fecal egg counts were too low to demonstrate any real challenge to the calves.

Conclusions

In this study, increasing weaning age of dairy steers did not significantly improve subsequent performance whether treated with ivermectin or not. Also, treating animals with a chemical dewormer did not have a major impact on weight or weight gain. The lack of differences in weight gain between treated and untreated animals could be attributable to pasture rotation management, possible reinfection of the treated animals by the untreated animals (although FEC in the treated animals remained low), or due to other unknown variables. This study begins to provide some evidence of the possibility for raising dairy calves organically in the southeastern US where parasites can be a year-round problem. However, calves were not fed organic feeds or pastures and did receive a coccidiostat in the calf starter. Further investigation into the possible efficacy or and better understanding of effects of alternate pasture management systems is needed. An integrated approach that accounts for the parasitic life cycle, biological control with fungi and dung beetles, and forage components such as condensed tannins that have anthelmintic properties should lead to successful strategies for raising dairy calves organically in the southeastern US.

FURTHER APPLICATION

Research Rationale

The rationale behind this research was pursuing alternative methods for preventing the production losses associated with parasite gastroenteritis in calves. As the North Carolina dairy industry shifts to fewer, larger farms carrying the bulk of milk production, small farmers are left with few options. The shift is seen in milk cow numbers and milk production numbers given by the NCDA (Agricultural Statistics Division, 2005) where the number of milk cows in North Carolina had steadily decreased over the past 50 years from 300,000 head in 1958 to approximately 60,000 in 2003. Milk production has not changed too dramatically (1,600 million pounds in 1958 to 1,200 million pounds in 2003). Therefore, if farmers with smaller herds desire to stay in business, they have to keep costs lower or create a niche' market. The possibility of organic production may be a viable option for them, although not without challenges.

Holstein and Jersey*Holstein results

Perhaps one of the most interesting findings in this study was the lack of weight differences and weight gains between the different breeds albeit the number of observations of each breed were small. When addressing the declining reproductive efficiency of Holsteins, the dominant dairy breed in the US, this study begins to support the possibility of introducing other breeds into a herd to introduce heterogeneity. Jerseys are able to endure the heat better than the Holsteins due to their size. Also, Jerseys have better reproductive efficiency than the Holsteins because they have not been as heavily selected for milk yield, which often leads to sacrificing reproductive measures. One possible application of this study is to begin to support the concept of introducing

crossbreeding into a dairy herd to alleviate reproductive problems without sacrificing weight and weight gain. However, comparisons between heifers of different breeds needs to be measured to be sure that weight gains are adequate to ensure they reach puberty by a year of age.

Limitations of the study

Reproductive measures such as time required to reach breeding weight could not be measured here because the study was done on steers. Researchers have determined that treating cattle with anthelmintics reduce the time required to reach breeding weight (Zajac et al., 1991); therefore, repeating the study with heifers would be valuable data to collect. While significant reduction in time to breeding weight has consistently been reported with the use of anthelmintics, pregnancy rate has not shown the same consistency. Some studies have shown treated animals having a higher pregnancy rate than untreated animals (Holmes, 1987), but other studies have shown no difference between the two groups (Loyacano et al., 1991).

Pasture management plays a large role in parasite trials on pasture-based animals. In this study, all calves were managed together so as not to introduce a pasture effect, but this may not have given the advantage to the treated animals that they would have in another trial. In other words, in most management situations, calves would all be treated; so, the untreated animals would not continually reinfect treated calves. Due to this reinfection of the treated animals, the lack of weight differences may reflect the fact that treated animals were not managed as they are in other studies where weight and ADG differences between untreated and treated calves are seen. Multiple replicated pastures

with similar forage, DM, and larvae infection are optimal in parasite trials, but were not available during this study.

Future study ideas

Restructuring the current study:

Design and calves: A similar project to the pilot study would be informative with the following changes: Remove the weaning age dimension of the factorial and have 18 calves (36 total) in each group or be able to decrease the amount of calves on trial to 18 total. If fall calving, begin by weighing all calves at birth and then monthly at 4 week intervals based on day 0 of the trial (the x axis on weight vs. time will be *day*, not *age*) until November of the next year. If using heifers in the study, the trial may be extended until January or perhaps even through calving at 2 years of age to incorporate reproductive measures in the analysis and comparison. The trial must have a calving day window of no more than 45 days. If spring calving, begin weighing calves at birth and monthly at 4 week intervals based on day 0 of the trial (the x axis on weight vs time will be *day*, not *age*) until December of the same year. The calving window must be no more than 30 days. This experiment would be more difficult to carry out due to the desired project start date of March, but if sufficient calves are available and if this management practice needs to be considered, then the study should be conducted. However, the results of this study may not be comparable to the fall-calving study because the fall-born calves may be better able to endure parasitic infection that would be beginning in the spring.

Pastures: Raise treated and untreated calves separately on clean pastures (no cows/calves on them within 1 year) until March or throughout the trial if available. Have

clean pastures sufficient for having the dewormed group in one set of paddocks and the untreated group in a different set of paddocks from March until November. Sufficient pasture DM must be available so that calves can be rotated to a new paddock when the canopy stand height reaches 10 cm in the presently-grazed paddock for all species of forages. All pastures must have the same type of forage (mixture of warm season and cool season grasses). Contaminate with infected larvae of known amount (see literature for 2 ways of doing this – trickle or dose).

Treatment: Deworm all treated calves one month after contaminating pastures and then monthly until November. If parasite load diminishes in the summer as it should, treated calves do not need to be dewormed at that time. Use ivermectin pour-on formulation or another dewormer common in the cattle industry. Due to the unlikelihood of having sufficient pasture for adding an additional group at the facilities used in this study, the study would not be able to compare two different dewormers.

Data Collection: Take monthly fecal samples starting in March. Also, take fecal samples 2 weeks after each deworming to estimate efficacy of dewormer. Estimate fecal scores on each sample. Analyze fecal samples for fecal egg counts using the McMasters and Modified Wisconsin technique and compare results. These results will be of interest to the field. Take monthly blood samples and analyze for serum pepsinogen values as indicators of *Ostertagia* infection. Compare the results of this assay with the results of the two fecal egg count techniques to see which the most effective measurement of parasitism is. Take monthly BCS and wither heights.

Other project ideas

Conduct a similar study with different forages that are known to have anthelmintic properties.

Cograzing studies where cattle graze in front of goats and then goats clean up behind the cattle or where cattle and goats co-graze in the same paddocks concurrently.

REFERENCES

- Anderson, R. C. 2000. Nematode Parasites of Vertebrates: Their Development and Transmission. CABI Publishing.
- Ballweber, L. R., L. L. Smith, J. A. Stuedemann, T. A. Yazwinski, and T. L. Skogerboe. 1997. The effectiveness of a single treatment with doramectin or ivermectin in the control of gastrointestinal nematodes in grazing yearling stocker cattle. *Veterinary Parasitology*. 72(1):53-68.
- Barth, D., G. F. Ericsson, B. N. Kunkle, S. Rehbein, W. G. Ryan, and D. H. Wallace. 1997. Evaluation of the persistence of the effect of ivermectin and abamectin against gastrointestinal and pulmonary nematodes in cattle. *Veterinary Record*. 140(11):278-279.
- Berghen, P., H. Hilderson, J. Vercruysse, and P. Dorny. 1993. Evaluation of Pepsinogen, Gastrin and Antibody-Response in Diagnosing Ostertagiasis. *Veterinary Parasitology*. 46(1-4):175-195.
- Coles, G. C., K. A. Stafford, and P. H. S. MacKay. 1998. Ivermectin-resistant *Cooperia* species from calves on a farm in Somerset. *Veterinary Record*. 142(10):255-256.
- Dimander, S. O., J. Hoglund, A. Ugglå, E. Sprondly, and P. J. Waller. 2003. Evaluation of gastro-intestinal nematode parasite control strategies for first-season grazing cattle in Sweden. *Veterinary Parasitology*. 111(2-3):193-209.
- Dorny, P., D. Demeulenaere, K. Smets, and J. Vercruysse. 2000. Control of gastrointestinal nematodes in first season grazing calves by two strategic treatments with eprinomectin. *Veterinary Parasitology*. 89(4):277-286.
- Echevarria, F., and A. Pinheiro. 2001. Efficiency of anthelmintics in cattle. 18th Int. Conf. W.A.A.V.P., Stresa, Italy. p. 147.
- Familton, A. S., P. Mason, and G. C. Coles. 2001. Anthelmintic-resistant *Cooperia* species in cattle. *Veterinary Record*. 149(23):719-720.
- Fiel, C. A., C. A. Saumell, P. E. Steffan, and E. M. Rodriguez. 2001. Resistance of *Cooperia* to ivermectin treatments in grazing cattle of the Humid Pampa, Argentina. *Veterinary Parasitology*. 97(3):211-217.
- Georgi, J. R., and M. E. Georgi. 1990, Parasitology for veterinarians, 5th edition. W. B Saunders Company. Philadelphia. Pennsylvania. 412 pages.
- Hawkins, J. A. 1993. Economic-Benefits of Parasite Control in Cattle. *Veterinary Parasitology*. 46(1-4):159-173.

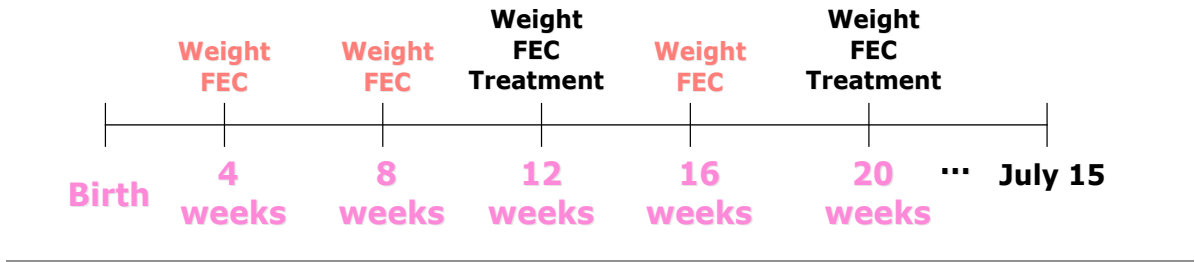
- Hopkins, B. A. 1997. Effects of the method of calf starter delivery and effects of weaning age on starter intake and growth of Holstein calves fed milk once daily. *J Dairy Sci.* 80 (9): 2200-2203.
- Johnstone, Colin. 1998. *Parasites and Parasitic Diseases of Domestic Animals.* University of Pennsylvania. http://cal.vet.upenn.edu/merial/Nematodes/nems_2.htm
- Loyacano, A. F., T. L. Skogerboe, J. C. Williams, A. A. DeRosa, J. A. Gurie, and V. K. Shostrom. 2001. Effects of parenteral administration of doramectin or a combination of ivermectin and clorsulon on control of gastrointestinal nematode and liver fluke infections and on growth performance in cattle. *Journal of the American Veterinary Medical Association.* 218(9):1465-1468.
- McLeod, R. S. 1995. Costs Of Major Parasites To The Australian Livestock Industries. *International Journal For Parasitology.* 25(11):1363-1367. Online.
- Melancon, J. 2001. *Parasites of Dairy Cattle.* Veterinary Professional Services, Merial Limited. <http://www.vet.ksu.edu/cdroms/merial/misc/99014.htm>
- Mercier, P., P. E. Steffan, and C. R. White. 2001. Effects of treatments with endectocide on the weight gain of grazing cattle in a warm temperate climate. *Veterinary Record.* 149(9):266-269.
- Merck and Co. Inc. 2003. <http://www.merckvetmanual.com>.
- Michel, J. F. 1969. Control of Some Nematode Infections in Calves. *Veterinary Record.* 85(12):326.
- Michel, J. F. 1985. Strategies for the Use of Anthelmintics in Livestock and Their Implications for the Development of Drug-Resistance. *Parasitology.* 90(APR):621-628.
- Myers, G. H. 1989. Ostertagiasis in cattle. *Journal of the Veterinary Diagnostic Investigation.* 1:195-200.
- Perry, B. D., and T. F. Randolph. 1999. Improving the assessment of the economic impact of parasitic diseases and of their control in production animals. *Veterinary Parasitology.* 84(3-4):145-168. Online.
- Ploeger, H. W., and A. Kloosterman. 1989. The Economic-Impact Of Nematode Parasitism In Cattle. *Magyar Allatorvosok Lapja.* 44(5):272-275. Online.
- Ryan, W. G., R. J. Crawford, S. J. Gross, and D. H. Wallace. 1997. Assessment of parasite control and weight gain after use of an ivermectin sustained-release bolus in calves. *Journal of the American Veterinary Medical Association.* 211(6):754.

- Sarkunas, M., A. Malakauskas, P. Nansen, J. W. Hansen, and V. Paulikas. 1999. Effect of strategic treatments with ivermectin on parasitism of set-stocked calves exposed to natural trichostrongyle infection in Lithuania. *Acta Veterinaria Scandinavica*. 40(2):163-171.
- Satrija, F., P. Nansen, C. M. Christensen. 1991. Ostertagia-ostertagi in neonatal calves – Establishment of infection in ruminating and non-ruminating calves. *Res. Vet Sci*. 51(3): 344-346.
- Skogerboe, T. L., L. Thompson, J. M. Cunningham, A. C. Brake, and V. K. Karle. 2000. The effectiveness of a single dose of doramectin pour-on in the control of gastrointestinal nematodes in yearling stocker cattle. *Veterinary Parasitology*. 87(2-3):173-181.
- Stromberg, B. E., R. J. Vathauer, J. C. Schlotthauer, G. H. Myers, D. L. Haggard, V. L. King, and H. Hanke. 1997. Production responses following strategic parasite control in a beef cow/calf herd. *Veterinary Parasitology*. 68(4):315-322.
- West, D. M., J. J. Vermunt, W. E. Pomroy, and H. P. Bentall. 1994. Inefficacy of Ivermectin against Cooperia Spp Infection in Cattle. *New Zealand Veterinary Journal*. 42(5):192-193.
- Williams, J. C. and A. DeRosa. 2003. Dose confirmation of moxidectin 0.5% pour-on against adults and fourth-stage larvae of various Cooperia spp. and Trichostrongylus colubriformis in Louisiana. *Veterinary Parasitology*. 114(4):295-303.
- Williams, J. C., A. F. Loyacano, A. DeRosa, J. Gurie, B. C. Clymer, and F. Guerino. 1999. A comparison of persistent anthelmintic efficacy of topical formulations of doramectin, ivermectin, eprinomectin and moxidectin against naturally acquired nematode infections of beef calves. *Veterinary Parasitology*. 85(4):277-288.
- Williams, J. C., A. F. Loyacano, A. DeRosa, J. Gurie, D. F. Coombs, and T. L. Skogerboe. 1997. A comparison of the efficacy of two treatments of doramectin injectable, ivermectin injectable and ivermectin pour-on against naturally acquired gastrointestinal nematode infections of cattle during a winter-spring grazing season. *Veterinary Parasitology*. 72(1):69-77.
- Yazwinski, T. A., H. Featherstone, C. Tucker, and Z. Johnson. 1994. Residual nematocidal effectiveness of ivermectin in cattle. *Am J. Vet. Res*. 55 (10): 1416-1420.
- Zajac, A. M., J. W. Hansen, W. D. Whittier, and D. E. Eversole. 1991. The effect of parasite control on fertility in beef heifers. *Vet Parasitol*. 40 (3-4): 281-291.

- Zeng S., D. E. B. Lawton, S. M. C. Przemec, D. C. Simcock, and H. V. Simpson. 2001. Reduced *Ostertagia circumcincta* burdens in milk-fed lambs. *NZ Vet J.* 49(1): 2-7.
- Zeng S., S. Brown, M.C. Przemec, and H. V. Simpson. 2003. Milk and milk components reduce the motility of *Ostertagia circumcincta* larvae in vitro. *NZ Vet J.* 51(4): 174-178.

FIGURES

•Phase 1 (based on age)



•Phase 2 (based on date)



Figure 1. Timeline of Phases 1 and 2 for the duration of the trial. Phase 1 started at birth and endured to July 15. Phase 2 began on July 15 and lasted until November 18. Calves were weighed every 4 weeks. Fecal samples were taken every 4 weeks also and fecal egg counts (FEC) were analyzed at North Carolina State University. Calves that were in treatment groups received ivermectin pour-on formulation at 12 wk and 20 wk of age and then again on July 15 and Sept 23.

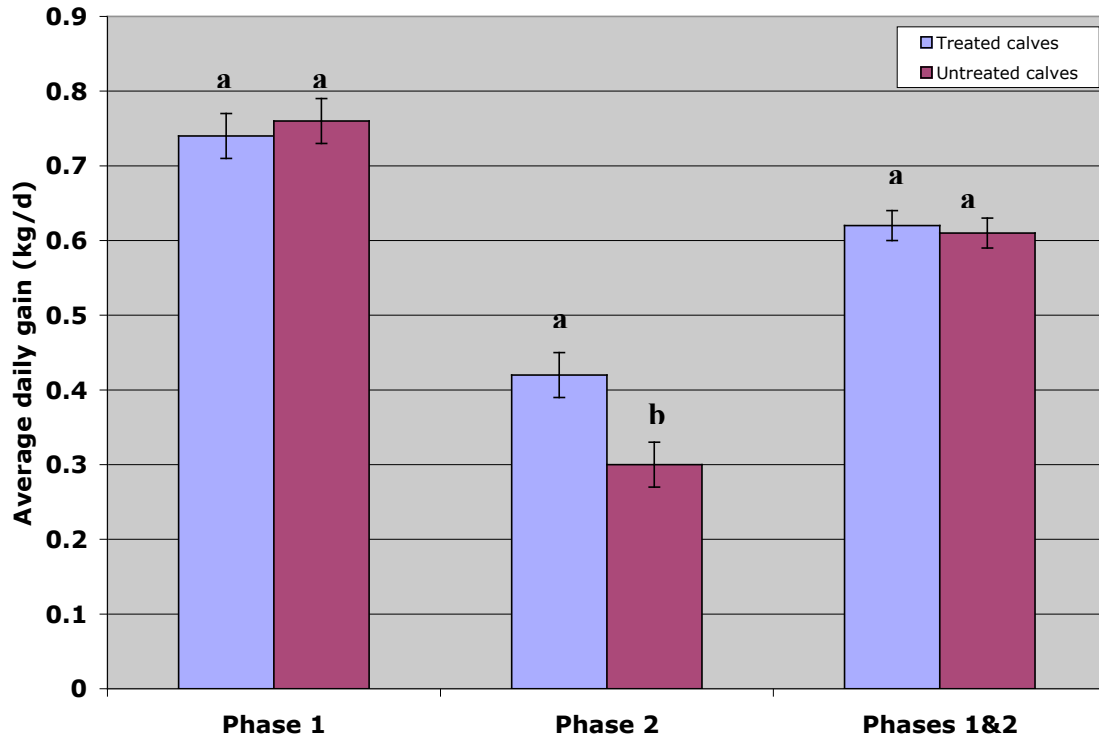


FIGURE 2. Lsmean average daily gains (kg/d +/- S.E.) of treated and untreated animals during Phase 1, Phase 2, and Phases 1 and 2 combined. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf. Treated animals were given 1mL/kg ivermectin pour on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and Sept 23 (Phase 2). Treated animals gained significantly more than untreated animals during Phase 2 only ($P < 0.05$).

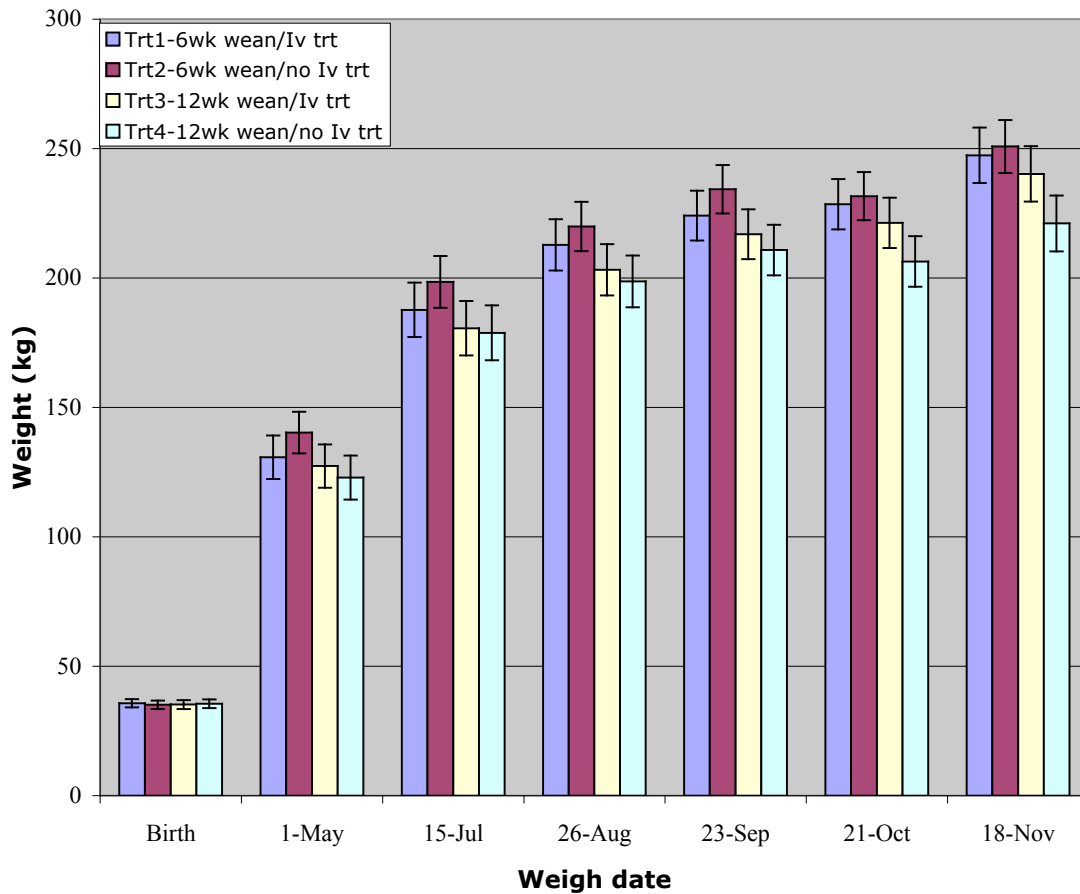


FIGURE 3a. Lsmean of weights (kg +/- S.E.) of 4 different treatment groups throughout Phases 1 and 2. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf. Trt1: calves weaned at 6 wk of age and treated with ivermectin. Trt2: calves weaned at 6 wk of age and untreated. Trt3: calves weaned at 12 wk of age and treated with ivermectin. Trt4: calves weaned at 12 wk of age and untreated. Treated calves were dosed with 1mL/10 kg BW ivermectin pour-on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and Sept 23 (Phase 2). All weigh dates represent actual weights from each treatment group, whereas May weights are calculated using a regression model.

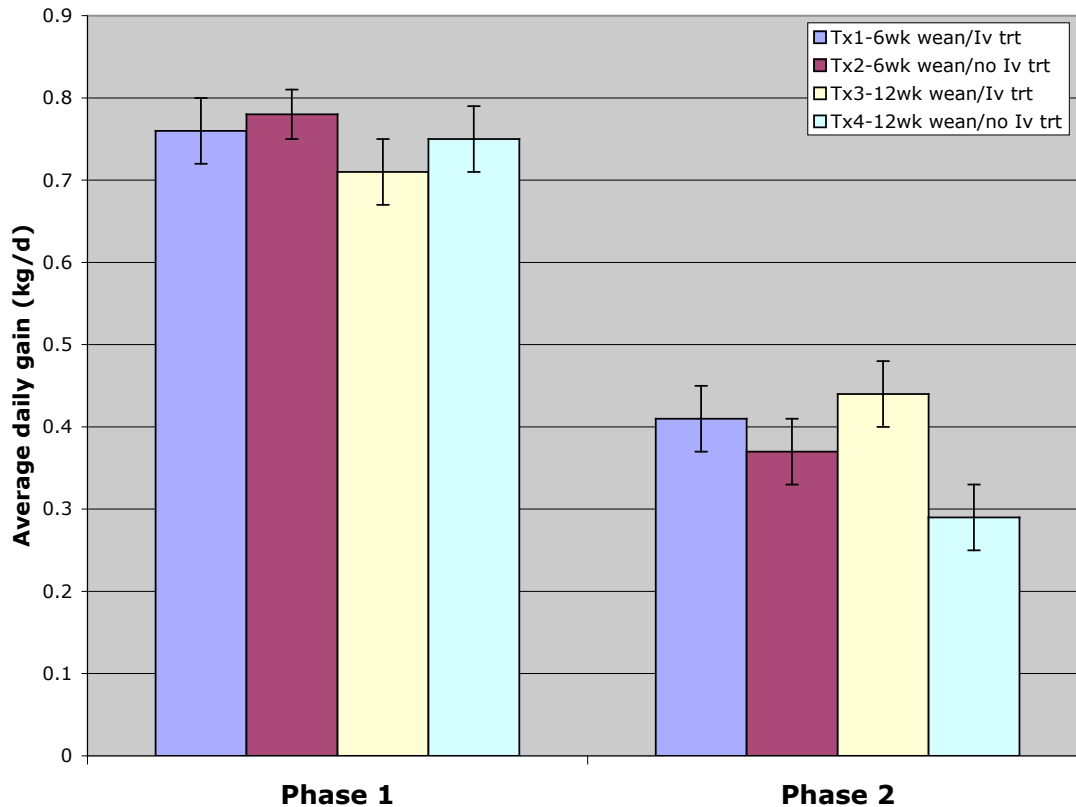


FIGURE 3b. Lsmean of average daily gain (kg/d +/- S.E.) of 4 different treatment groups. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf. Trt1: calves weaned at 6 wk of age and treated with ivermectin. Trt2: calves weaned at 6 wk of age and untreated. Trt3: calves weaned at 12 wk of age and treated with ivermectin. Trt4: calves weaned at 12 wk of age and untreated. Treated calves were dosed with 1mL/10 kg BW ivermectin pour-on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and Sept 23 (Phase 2).

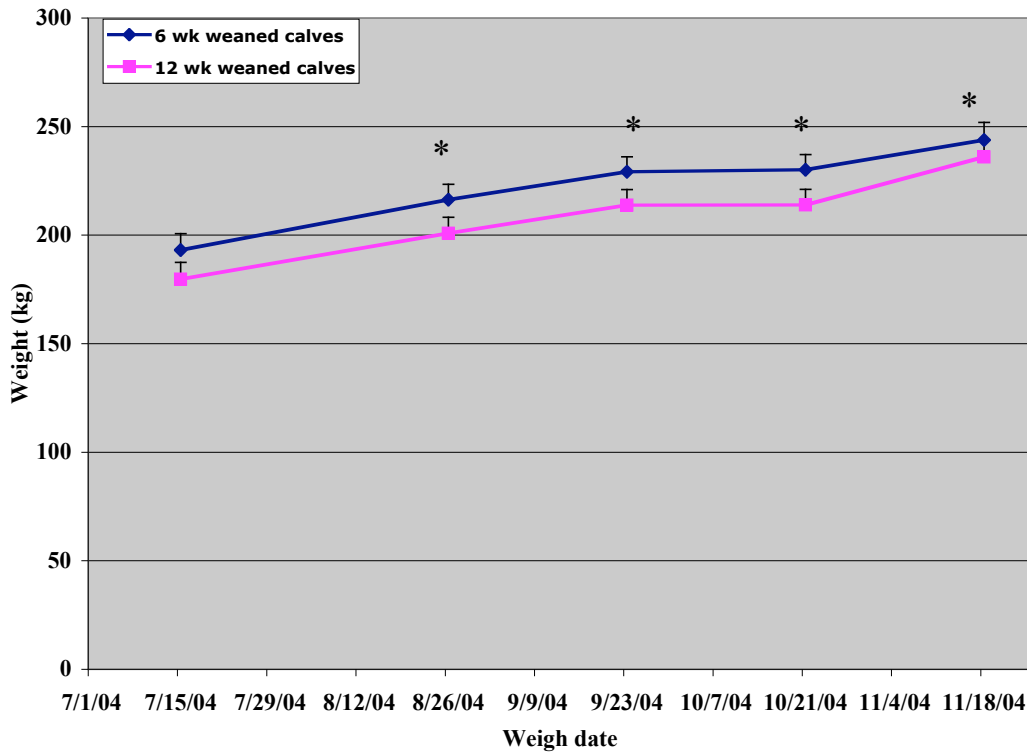


Figure 4. Phase 2 (July 15, 2004 to Nov 18, 2004) lsmean weights (kg + S.E.) of calves weaned at 6 wk (n=18) or 12 wk (n=18). 6 wk-weaned calves tended to weigh more than 12 wk-weaned calves on 8/26 (P = 0.1); 9/23 (P = 0.1); 10/21 (P = 0.08); 11/18 (P = 0.07) as denoted in the graph by *. Average daily gain for calves weaned at 6 wk or 12 wk were not different (0.38 ± 0.03 kg/d and 0.36 ± 0.03 kg/d, respectively).

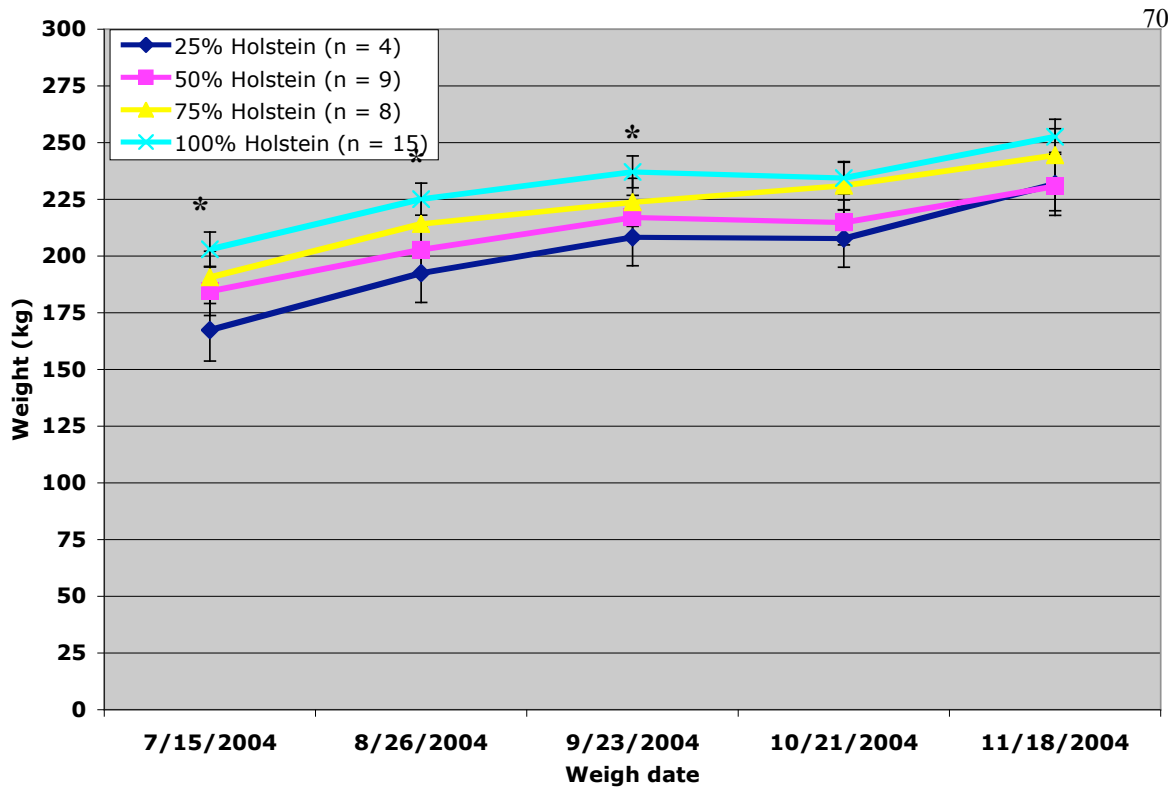


Figure 5. Phase 2 lsmean weights (kg +/- S.E.) of calves of varying breeds. Fifty percent Holstein steers were crosses using a 100% Jersey sire and 100% Holstein dam. Twenty-five percent Holstein steers were crosses using a 100% Jersey sire to a 50% Jersey-Holstein dam. Seventy-five percent Holstein steers were crosses using a 100% Holstein sire to breed a 50% Jersey-Holstein dam. Differences in weights between breeds occurred between 25% and 100% Holstein on 7/15, 8/26, and 9/23 ($P < 0.05$; differences between 25% and 100% Holstein denoted above by *). Although not significantly different in most cases, numerical means were generally higher with increasing percentage of Holstein.

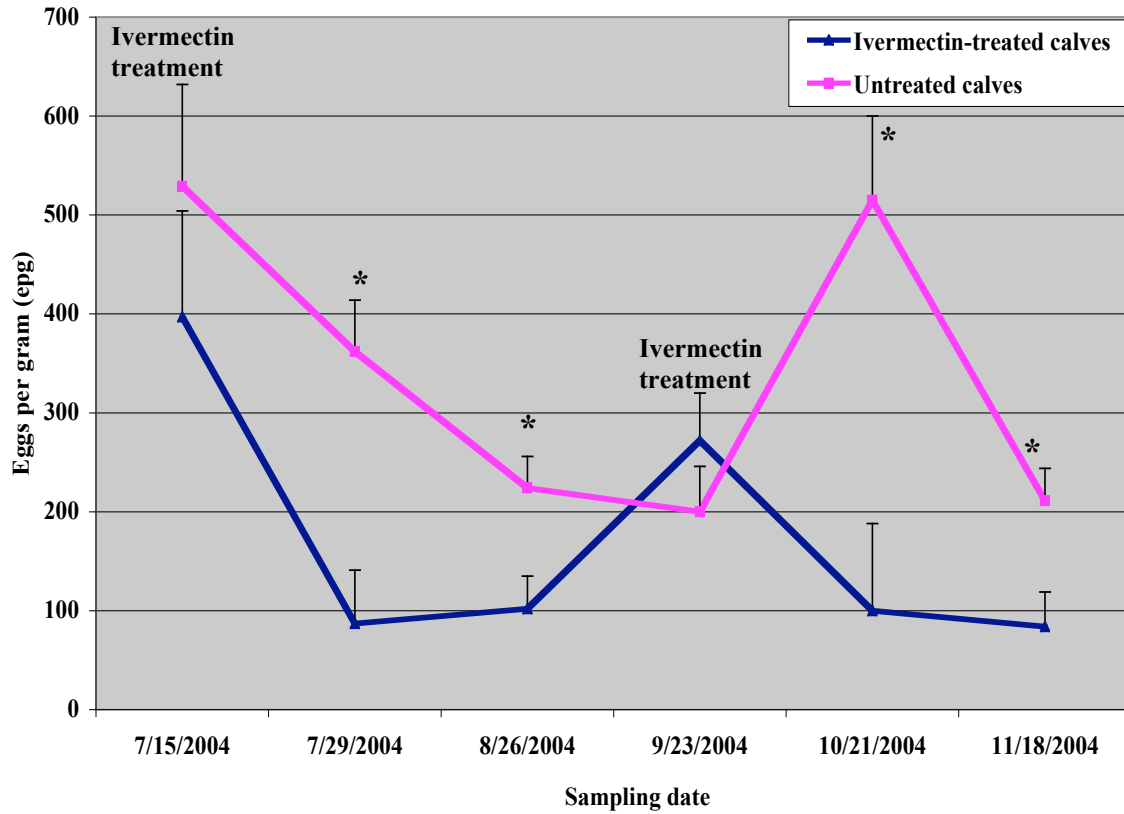


Figure 6a. Phase 2 fecal egg counts (eggs per gram +/- S.E.) of treated and untreated calves. Treated calves (Treatment groups 1 and 3) were treated with 1mL/10kg BW ivermectin pour-on formulation on July 15 and September 23. Treated calves had lower FEC than untreated calves on 7/29/04, 8/26/04, 10/21/04, and 11/18/04 ($P < 0.05$; differences between groups denoted above by *).

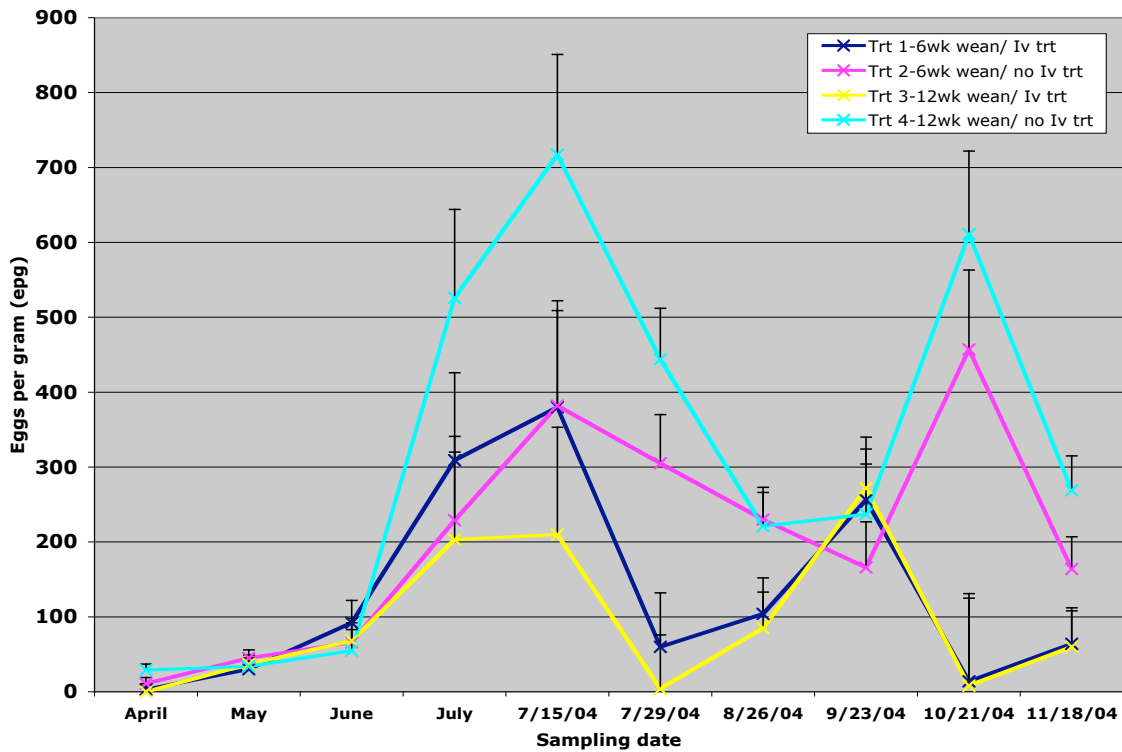


Figure 6b. Phase 1 and Phase 2 fecal egg counts (eggs per gram) of each treatment group. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf. Treated calves (Treatment groups 1 and 3) were treated with 1mL/10kg BW ivermectin pour-on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and September 23 (Phase 2). April sampling is the composite of fecal egg counts from April 1-22. May sampling is the composite of fecal egg counts from April 29-May 20. June sampling is the composite of fecal egg counts from May 27-June 17. July sampling is the composite of fecal egg counts from June 24-July 15. During Phase 2, all animals were sampled on the same date.

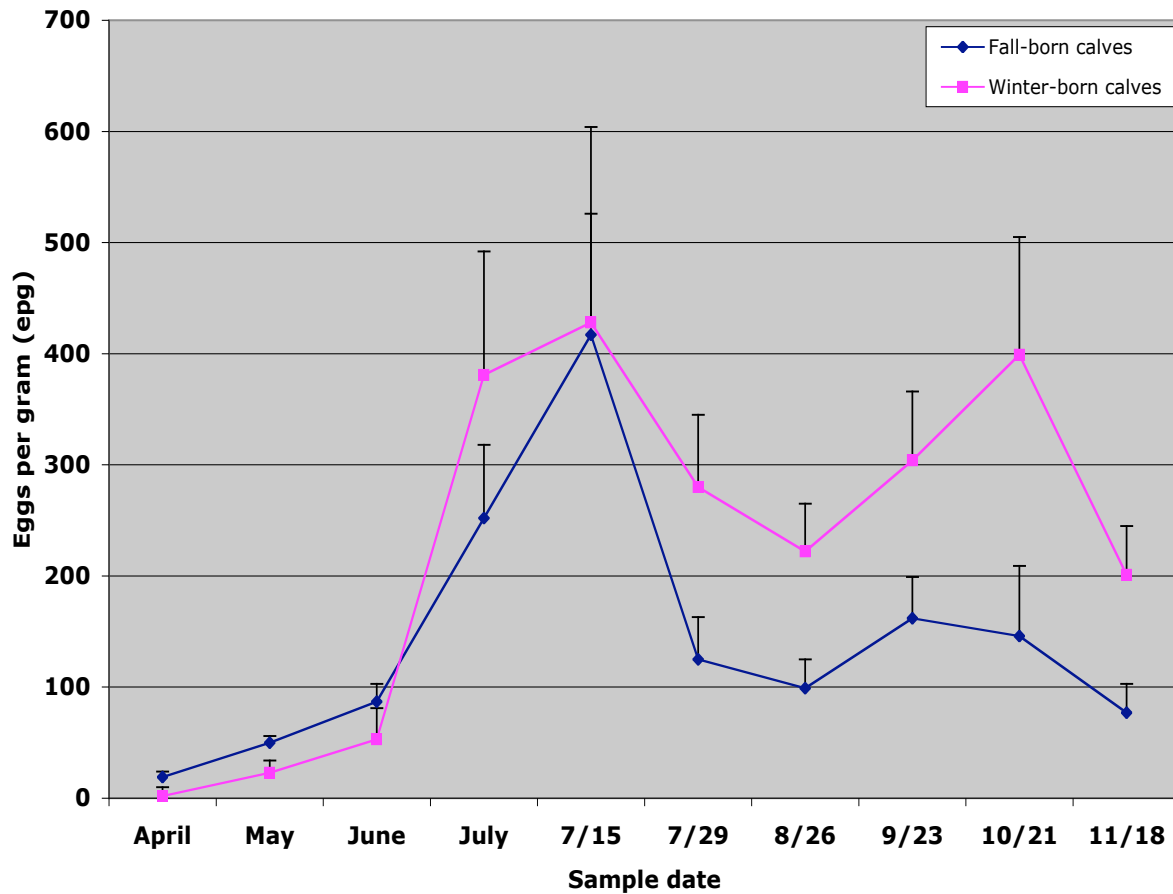


Figure 7. Phase 1 and Phase 2 fecal egg counts (eggs per gram) of fall-born (Oct-Nov) and winter-born (Dec-Feb) calves. Calves receiving anthelmintic treatment were dosed with 1mL/10kg BW ivermectin pour-on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and September 23 (Phase 2). April sampling is the composite of fecal egg counts from April 1-22. May sampling is the composite of fecal egg counts from April 29-May 20. June sampling is the composite of fecal egg counts from May 27-June 17. July sampling is the composite of fecal egg counts from June 24-July 15. During Phase 2 (July 15 - Nov 18), all animals were sampled on the same date.

Table 1. 2 x 2 factorial arrangement of treatments with weaning age and treatment as classification variables. Treatment group 1= 9 calves weaned at 6 wk and treated with ivermectin pour-on formulation (1mL/10kg BW) at 12 and 20 wk of age (Phase 1) and again on July 15 and September 23 (Phase 2). Treatment group 2= 9 calves weaned at 6 wk and untreated. Treatment group 3= 9 calves weaned at 12 wk and treated with ivermectin pour-on formulation (1mL/10kg BW) at 12 and 20 wk of age (Phase 1) and again on July 15 and September 23 (Phase 2). Treatment group 4= 9 calves weaned at 12 wk and untreated.

		Weaning age	
		6 wk	12 wk
Ivermectin treatment	+	9 calves	9 calves
	-	9 calves	9 calves

Table 2. Feed analyses throughout Phases 1 and 2¹.

	DM, %	CP % DM	TDN % DM	ADF % DM	NDF % DM
Calf Starter Concentrate²	96.7	26.4	78.5	6.2	13.0
Grower Concentrate³	89.8	17.0	79.4	4.1	9.3
Cool season ryegrass⁴	26.7	19.6	84.5	16.0	34.3
Warm season Bermudagrass⁴	38.5	21.7	69.1	22.9	62.1
Warm season annuals⁴	22.8	18.8	67.8	31.0	57.2
Bermudagrass Hay⁵	86.5	10.9	55.4	42.2	70.7

¹ Calves ate calf starter concentrate (80% corn, 13.9% SBM, 6.1% mineral) from birth until 12 wk of age at a rate of 0.9 kg/head/d. Grower concentrate (83.9% corn, 10.6% SBM, 5.5% mineral) was fed after 12 wk of age at a rate of 2.27kg/head/d along with forage. Cool season Ryegrass was grazed from October to January and March to June. Warm season Bermudagrass and summer annual mix was grazed from June – September. Bermuda Hay was fed from December – March when grass supply was insufficient for DMI. All feed analyses were done at Cumberland Valley Analytical Services.

² Calf starter concentrate analyses were performed on a composite sample consisting of 5 samples: 1 subsample taken from various places within each of 5 bags.

³ Grower concentrate analysis was performed on a composite consisting of 4 subsamples: 1 subsample taken from each of 4 batches.

⁴ Cool season ryegrass, warm season Bermudagrass, and warm season annuals analyses were performed on a composite sample from each of 3 paddocks.

⁵ Bermudagrass hay analysis was performed on a composite sample from cored samples from each of 6 bales.

Table 3. Lsmean weight (kg +/- S.E.) and lsmean average daily gain (ADG; kg/d +/- S.E.) comparisons between calves weaned at 6 wk or 12 wk and either treated or untreated with ivermectin pour on. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf. All treated calves were dosed with 1mL/10kg BW ivermectin pour-on formulation at 12 and 20 wk of age (Phase 1) and again on July 15 and September 23 (Phase 2). No statistical differences existing between group weights or ADG throughout Phases 1 and 2.

		6wk wean Ivermectin	6wk wean no ivermectin	12wk wean Ivermectin	12wk wean no ivermectin
Weight (kg)	Birth	35.7 ± 1.6kg	35.1 ± 1.6	35.2 ± 1.7	35.6 ± 1.7kg
	July 15	187.7 ± 10.5	198.5 ± 10.1	180.6 ± 10.5	178.8 ± 10.6
	Nov 18	245.0 ± 10.4	251.1 ± 10.0	240.1 ± 10.4	221.2 ± 10.6
ADG (kg/d)	Phase 1	0.76 ± 0.04	0.77 ± 0.03	0.71 ± 0.04	0.75 ± 0.04
	Phase 2	0.40 ± 0.04	0.37 ± 0.04	0.44 ± 0.04	0.28 ± 0.04
	Phase 1 and 2	0.63 ± 0.02	0.64 ± 0.02	0.60 ± 0.02	0.58 ± 0.02

Table 4. Lsmean ADG (kg/d +/- S.E.) for fall-born (October and November) and winter-born (December – February) calves during Phases 1 and 2. Phase 1 began at birth and continued to July 15, lasting an average of 212 d. Phase 2 started on July 15 and ended on November 18, lasting 126 d for each calf.

	Fall-born calves n = 26	Winter-born calves n = 10
Phase 1	0.81 ± 0.02 ^a	0.69 ± 0.03 ^b
Phase 2	0.39 ± 0.02 ^a	0.36 ± 0.04 ^a
Phases 1 and 2	0.68 ± 0.01 ^a	0.55 ± 0.02 ^b
^{a,b} Superscripts differing across a row represent values which are significantly different by $P < 0.01$.		

Table 5. Fecal egg counts (lsmean: eggs per gram +/- S.E.) of fall-born (October and November) and winter-born (December - February) calves during Phase 2.

		Fall-born calves		Winter-born calves	
		Treated	Untreated	Treated	Untreated
July 15	^{c1}	446±102	387±116	144±197	712±155
July 29	^{abc}	37±51	214±59	27±100	534±79
August 26	^{ab}	44±34	154±39	146±66	298±52
September 23	^b	205±48	119±59	324±95	285±75
October 21	^{abc}	16±83	276±97	6±163	792±129
November 18	^{ab}	33±34	122±40	91±67	311±53

Superscripts that differ across a row reference values that are statistically different.
¹ Represents a trend towards a treatment effect ($P = 0.08$)
^a Represents a treatment effect ($P \leq 0.05$)
^b Represents a season (fall- or winter-born) effect ($P \leq 0.05$)
^c Represents a treatment*season effect ($P \leq 0.05$)