Genetic parameters for pre-fresh intake and the effects of lameness on feed intake and milk production in dairy cattle

by

Brittany N. Shonka

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Program of Study Committee:
Diane M. Spurlock, Major Professor
Jack C.M. Dekkers
Lee H. Kilmer

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DEDICATION

I would like to dedicate this thesis to my family to thank them for all the support they have provided throughout my schooling.

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ABSTRACT

Dry matter intake (DMI) is an important topic of research in dairy cattle. It is often studied relative to feed efficiency and disease. However, there are gaps in the current knowledge of DMI that remain to be filled. The objectives of the current study aimed to fill these gaps by estimating genetic parameters for DMI during the pre-fresh transition period, as well as examining the impact of lameness on DMI and milk production. In the current study, DMI during the dry period was moderately heritable, and had a high genetic correlation with lactating intake. This finding indicates DMI may be under similar genetic regulation during the dry period and lactation. Also, a low genetic correlation was found between the magnitude of intake depression before calving and other DMI traits, suggesting that the decline in DMI at parturition would be minimally affected by selection for DMI at other time points. It was confirmed that lameness has a negative effect on milk production and feed intake in lactating dairy cattle. Milk production decreased earlier than intake when comparing daily averages in the days before treatment, suggesting that decreased intake may not be the cause of decreased production surrounding a lameness event. Milk production also did not return to pre-treatment levels, indicating a lasting effect of lameness. When compared to the average of healthy cows, lame cows deviated in both intake and milk production for the days surrounding treatment for lameness. After treatment, milk production of multiparous cows gradually returned to pre-treatment levels, whereas primiparous cows recovered more quickly. The effects of lameness can be seen for at least two months after treatment for both DMI and milk production.

CHAPTER 1. GENERAL INTRODUCTION

1.1 Thesis Organization

Chapter 1 includes a general introduction to feed intake and its importance to the dairy industry, as well of the objectives of the thesis. A review of the literature is also included in Chapter 1. Chapters 2 and 3 were written for publication in scientific journals. Estimation of genetic parameters is described in Chapter 2. The effect of lameness on milk production and feed intake is described in Chapter 3. Finally, Chapter 4 includes general conclusions and discussion.

1.2. Introduction

Feed costs account for more than half of the expenses of a dairy enterprise, and producers often seek ways to lower this cost. Using alternative or non-traditional feeds is one option, however it can be challenging to find a consistent supply and nutrient content of these feeds. Another option to lower feed costs is to increase the feed efficiency of the cow. An efficient cow can be described as producing the same amount of milk as other cows while eating less feed, or as producing more milk while eating the same feed. To measure efficiency, feed intake data must be collected.

There are many obstacles when measuring feed intake in dairy cattle. Feed intake is relatively easy to measure on a pen basis in a commercial setting, but difficult to measure for individual cows as most dairy farms lack the necessary facilities, equipment, and labor. Therefore, most feed intake data come from research herds. Cows in research herds may be on multiple studies, and accounting for those factors can make analysis

difficult. In addition to collecting the intake data, samples of the feed must be analyzed for moisture content, which allows for calculation of dry matter intake (DMI). Expressing intake as DMI takes into account the large variety of moisture content in the diets of dairy cattle (Grummer et. al., 2004). Thus, DMI is a more appropriate comparison than the amount of total feed consumed because cows will eat more or less feed based on the moisture content.

Feed efficiency in livestock is most often described as the ratio of production outputs to feed inputs (Archer et al., 1999). In dairy cattle, the production output is usually the amount of milk energy produced, while the feed input is expressed as the energy consumed from feed. This ratio is also known as gross efficiency (Brody, 1945) and determines how efficiently a cow converts energy from feed to energy produced in milk. However, gross efficiency does not take into account that the cow uses energy not only for lactation, but also for maintenance and body tissue gain or loss (Veerkamp and Emmans, 1995). Maintenance energy is necessary for the cow to maintain basal metabolism, conduct voluntary body activity, and generate heat to maintain body temperature (Korver, 1988).

Though lactation utilizes over half of the energy consumed, other functions also impact the overall efficiency of dairy cows. Feed intake is different between animals based on their level of production as well as their maintenance requirements (Korver, 1988). Residual feed intake (RFI), first described by Koch et al. (1963), is an alternative way to look at efficiency, one that describes differences in biological efficiency independent of lactation and maintenance. Some countries, such as Ireland and New Zealand, have included feed efficiency in their breeding goals by placing a positive

weight on milk-solids production and a negative emphasis on body weight, but this method still does not take into account mobilization of body tissue (Coleman et al., 2010).

In addition to feed costs, health disorders are also costly to the dairy industry. When a cow becomes ill, she decreases the profit of a farm due to decreased milk production, increased labor to care for the cow, the cost of treatment, decreased reproductive health, and in extreme cases, premature culling or death. Health events can cost from $46 to $358 per case (Shaver, 1997; Oetzel, 2013; Overton and Fetrow, 2008; Guard, 1996; Cha et al., 2010). Mastitis alone costs the United States dairy industry $1.7 to $2 billion per year (Jones and Bailey, 2009). Decreased feed intake and milk production is a common symptom of disease. Quantifying the differences in intake between healthy and ill cows, and when these differences are observed relative to disease, can aid in diagnosis of disease, especially for events where obvious signs are not observed. These differences can also be used as a tool for educating producers of the importance of prevention, early detection, and treatment.

1.3 Objectives

The objectives of this thesis were to describe genetic parameters for intake during the pre-partum period in dairy cattle and to determine the effect of lameness on intake and milk production. To complete this objective, two studies were carried out to 1) estimate genetic parameters for pre-fresh dry matter intake and 2) determine the effect of lameness on intake and milk production in the periods before and after treatment. The

results from this thesis aid in filling the gaps in knowledge about feed intake and the vital role it plays in the life of a dairy cow.

1.4 Review of Relevant Literature

1.4.1 Pre-fresh intake

The dry period is the non-lactating period before calving in dairy cattle. Feed intake during the dry period is much lower than the lactating period, because the cow does not need energy for milk production. During this time, the cow is eating to support maintenance of body condition as well as the growing calf. Historically, it was thought that feed intake was constant throughout the dry period, and the recommended nutrient requirements for dry cows reflected that (Grummer, 1995). Intake is fairly constant during the early portion of the dry period, however, during the pre-partum transition period, feed intake is reduced as calving nears, while nutrient demands increase to support the fast growing calf and the onset of lactation (Grummer, 1995). During this time, intake gradually decreases by 20-40% (Hayirli et al., 2002). The sharpest decline in intake happens during the last week before calving (Grummer, 1995; Hayirli et al. 2002). Though the decline in intake has been clearly described, its cause is not known. There is evidence to suggest a relationship with endocrine changes, such as the increase in NEFA (Grummer, 1995; Allen and Piantoni, 2013). The depression in DMI is one of the factors that causes mobilization of energy from adipose tissue. If the cow is not consuming enough energy, more tissue will be mobilized, which increases the chance of metabolic disease following calving. It is therefore beneficial to encourage DMI by increasing the

nutrient density in the ration. It is also important to monitor pre-fresh cows, as the extent of DMI depression could be an indicator of metabolic disease (Grummer, 1995).

Endocrine changes may not be the only factors influencing intake during the pre-fresh transition period. Diet also plays a crucial role. Diets must not only contain all the nutrients required by the cow, but must also be fed in such a way that encourages intake and decreases sorting. Hayirli et al. (2002) composed a model for DMI that only accounted for 18% of the variation in DMI during the pre-fresh period. In that model, diet accounted for 24.2% of the variation. Similar results were found for lactating cows by Roseler et al. (1997), where diet accounted for 22% of the variation in DMI. Body condition score in both of the previous models significantly accounted for variation in DMI, at 9.7 and 6%, respectively. Parity affects DMI, as would be expected. Though primiparous animals are still growing during gestation, older cows are usually larger and have more capacity for feed consumption. However, multiparous cows have been shown to have a more severe depression of feed intake that occurs earlier than that of primiparous cows. Marquardt et al. (1977) found that primiparous and multiparous cows decreased DMI by 25% and 52% respectively, during the last 2 weeks of gestation. These results are comparable to Hayirli et al. (2002), who found that older cows decrease their DMI approximately 34% during the last three weeks, while primiparous cows remain constant until the final week before calving, in which their intake declines 28%. It must be noted that the previous two studies were published 25 years apart. The cows in the two studies most likely differ in stature and production, because of genetic selection during the intervening years. High body condition scores also increase the magnitude of intake depression. Grummer (1995) reviewed the literature relating body condition with pre-

partum feed intake and described numerous studies that found overconditioning is associated with lower dry matter intake. This causes a more negative energy balance and a higher risk of metabolic disease following calving (Grummer et al., 2004). Factors such as diet and body condition score are able to be controlled by management, and because metabolic diseases are costly to dairy producers, having a good transition program is crucial to having productive lactations.

Information about the genetics of pre-partum feed intake is limited, and genetic parameters associated with dry period DMI are non-existent. Most genetic studies that include intake focus on the genetics of feed efficiency, so milk production and lactating DMI are needed. Since lactation also accounts for a large portion of the life of a dairy cow, it is logical that most studies focus on this time period. However, because the pre-partum transition period plays such an important role in a profitable lactation, it is important to understand the relationship between feed intake during dry and lactating periods.

1.4.2 Lactating intake

Feed intake studies during lactation focus heavily on the optimization and genetic regulation of feed efficiency. Historically, selection for feed efficiency has been done by selecting for increased milk yield. Freeman (1967) found that by selecting for milk production, the indirect response of feed efficiency was 70 to 95% of the response achieved by direct selection for feed efficiency. Because intake data are difficult to obtain, selecting directly for gross feed efficiency is not necessary economically feasible. However, an eventual goal for dairy breeders is to incorporate feed efficiency directly

into a selection index. This has led to many studies focusing on genetic parameters for DMI and its relationship to other traits.

Heritabilities reported for DMI are given in Table 1. Differences in estimates may be attributed to sample size, statistical methodology, and population differences. The number of cows on each study ranges from 289 to 970. The study by Van Arendonk et al. (1991) is the most different, as the animals in that study were selected based on high and low intake. Koenen and Veerkamp (1998) also used selection to create different lines, but their selection criteria was based on production instead of intake. Berry et al. (2007) found low to moderate heritability using records on grass fed cows, while Vallimont et al. (2010) found a low heritability when collecting feed intake data from cows housed in tie stall barns. The low estimates could be due to measurement errors, as there were no distinct barriers between the cows, making it difficult to determine which feed belonged to which cow. The upper range of these estimates are moderately heritable. Together, these studies demonstrate that the regulation of DMI has a genetic component.

The genetic correlation between DMI at different stages of lactation has been studied to determine if the genes controlling DMI change between early and late lactation (Table 2). Reported positive genetic correlations range from 0.10 to 0.97 (Tetens et al., 2014; Spurlock et al., 2012; Buttchereit et al., 2011; Karacaören et al., 2006; Koenen and Veerkamp, 1998; Veerkamp and Thompson, 1999). The range is large, mostly due to relatively small sample sizes and therefore large standard errors, making some estimates not significantly different from zero ($P>0.05$). Most of the studies found that the strength of the correlation is inversely related to the amount of time between the stages of lactation being compared (Tetens et al., 2014; Berry et al., 2007; Karacaören et al., 2006;

Koenen and Veerkamp, 1998; Veerkamp and Thompson, 1999). In general, these results indicate that genetic regulation of DMI during lactation changes according to stage of lactation, and therefore, DMI at different stages should be considered separate traits. In contrast, Spurlock et al. (2012) found correlations above 0.70 between daily estimates during early and mid-lactation, as well as high correlations when using monthly averages for the first five months. Correlations between different stages of lactation are important to consider, especially during early lactation when cows are experiencing negative energy balance. Reducing DMI during this period could have negative effects on fertility and health (Tetens et al., 2014). If the genes affecting DMI do change with stage of lactation, selection will need to adjust to the changing biology of the cow with different stages of lactation. Between early and mid-lactation, low to negative correlations were found by Veerkamp and Thompson (1999), Berry et al. (2007), Hüttmann et al. (2009), Koenen and Veerkamp (1998), Coffey et al.(2001), and Buttchereit et al. (2011). These studies differ from Spurlock et al. (2012) where high positive correlations were found between early and mid-lactation. Between early and late lactation, correlations range from highly positive (Coffey et al., 2001) to low (Berry et al., 2007). These results stress that more work in this area needs to be done to better quantify these correlations. However, based on the majority of studies, it appears that early, mid, and late lactation should be considered separate traits, due to the low genetic correlations among DMI measures at these times.

1.4.3 Health

Health disorders such as mastitis and lameness can occur throughout lactation, and even during the dry period. In most circumstances, these issues can be prevented by

proper management techniques, such as a clean living and milking environment for the cow. Other disorders, such as milk fever and displaced abomasum, usually occur during the postpartum phase of the transition period. These diseases are usually considered to be metabolic, brought on by the dramatic increase in energy demands for lactation as well as the decrease in feed intake before calving. This results in negative energy balance, which causes the cow to mobilize adipose tissue. Overconditioning cows and severe negative energy balance put cows at high risk for developing these diseases. Also, metabolic health events often form disease complexes, meaning a cow can easily have more than one illness around the same time period (Urton et al., 2005). Prevention, early detection, and timely treatment, are necessary to minimize economic losses due to disease.

1.4.3.1 Transition cow diseases

Metabolic diseases in dairy cattle are thought to be caused in part by the decrease in feed intake before calving as well as the higher energy demand brought on by lactation (Grummer, 1995). Lower feed intake means the cow is not consuming enough energy to transition from dry to lactating, so she must use body tissue reserves (Grummer, 1995). Low intake is thought to be one of the reasons for the abomasum shifts that allow displaced abomasums (DAs), due to low ruminal fill (Shaver, 1997; Van Winden et al., 2003). Mobilizing adipose tissue increases the amount of ketones in blood and urine, which can cause a cow to have ketosis. Milk fever, another common transition cow disease, is associated with the sudden demand for calcium needed for milk production. The stresses associated with the transition period, especially if cows develop one of the previously mentioned disease, can lower immune function, which increases the risk of getting infections such as metritis. Incidence rates differ according to disease, and range

from 40 to 60%, 0.7 to 69%, and 0 to 10% for ketosis, metritis, and milk fever, respectively (Oetzel, 2013, Huzzey et al., 2007, DeGaris and Lean, 2009).

The effect of metabolic diseases on intake, as well as the cost for specific diseases, is summarized in Table 3. Significant differences in intake between diseased and healthy cows have been reported for metabolic diseases. When those significant changes in intake occur is disputable, but most studies found that changes occur close to the day of diagnosis. In some cases, cows with metabolic diseases showed lower intake up to two weeks before calving (Goldhawk et al., 2009; Huzzey et al., 2007; Bareille et al., 2003). For cows diagnosed with milk fever however, contrasting results were found. Jawor et al. (2012) reported that cows with milk fever ate more 2 weeks before and after calving. The authors found a large parity effect, with older cows more likely to be diagnosed with milk fever, which is common in the industry. Older cows tend to be larger and higher producers and therefore need more feed. It is clear that transition diseases cause changes in intake, but since intake is difficult to measure, other studies focused not just on intake specifically, but feeding behavior. Huzzey et al. (2007) and Urton et al. (2005) found that cows were more likely to be diagnosed with metritis if they spent less time at the feed bunk before and after calving.

1.4.3.2 Lameness

Lameness is the second most costly disease to the dairy industry. Because there are many reasons a cow can be lame, the disease is complex. Determining if a cow is lame is subjective, making it is difficult to determine incidence rates. Reported values range from 0 to 70% (Green et al., 2002). Cows can suffer from lameness due to injury, sole ulcers, digital dermatitis, foot rot, white line disease, and other causes. A lame cow

means increased treatment costs, increased labor costs, decreased milk production, and possibly the loss of the animal by culling or death (González et al., 2008). These costs vary according to disease. Cha et al. (2010) estimated the cost of lameness to be $216.07, $132.96, and $120.70 per case for sole ulcers, digital dermatitis, and foot rot, respectively. A relationship between increased lameness and high milk yields may exist (Potterton et al., 2012), likely because high yielding cows are under more stress. Therefore, as selection for milk yield continues, lameness will continue to be an issue. Determining when lameness affects DMI and milk production and how long the effects last are important factors in fully understanding how important it is to promptly detect and treat lame cows.

It is clear from the literature that lameness has a definite impact on DMI. González et al. (2008) found that lame cows on average had less daily feeding time, fewer visits to the feed bunk, and a higher feeding rate in the 30 days before being treated. When comparing intake during the 30 day period after treatment, cows seemed to have fully recovered and showed more typical feeding behaviors. Similar results were found by Norring et al. (2014). Palmer et al. (2012) also found that lame cows had lower DMI, but those results were only significant for cows around 60 DIM, not for cows around 120 DIM. This result suggests that lameness may impact cows differently according to stage of lactation.

Milk production is also significantly affected by lameness. Cows diagnosed with sole ulcers and white line disease had a significant decrease in milk yield in the month before treatment (Green et al., 2014). Determining both when the decline starts and when the cow recovers can help in understanding the total impact across lactation. Green et al.

(2002) found that clinically lame cows can show decreased production up to 4 months before diagnosis and treatment, and that lower milk yields can last up to 5 months after treatment. This results in a loss of 360 kg of milk during a typical 305-day lactation, on average. Decreased milk yield for lame cows could be caused in part by these changes in feeding behavior, as milk yield is reliant on feed intake.

1.4.3.3 Mastitis

Mastitis is the most costly disease affecting dairy cattle. Mastitis is usually caused by a bacteria, which forces the cow to have an immune response, which then causes inflammation of the udder. If untreated, the infection can cause damage to the mammary tissue, and is thought to cause the cow pain. The immune response causes a rise in the leukocyte or somatic cells in the milk. If the level of somatic cells is too high in a herd, the milk will be unsaleable. Though proper management techniques can significantly reduce the incidence of mastitis, it cannot be completely eliminated. Documenting changes in feed intake for cows diagnosed with mastitis helps with the understanding of the impact of mastitis, as well as if changes in intake can be used as detection aids.

Fogsgaard et al. (2012) found that cows induced with *E. coli*, so as to have an acute clinical infection, spent less time feeding on the day of infection compared to two days before and two days after. Time spent feeding two days before compared to two days after was not significantly different. However, DMI decreased between 12 to 36 hours after infection, and had not recovered to pre-infection levels at 60 hours after infection (Fogsgaard et al., 2012). Bareille et al. (2003) also indicated a reduction in DMI by 30.3 kg on average for cows diagnosed with systemic mastitis. Similar results were found by Yieser et al. (2012) for cows induced with *E. coli*, which caused a

significant drop in intake on the day after infection. This study also included cows induced and then treated for mastitis. Both the infected cows and treated cows ate less than control cows two days after infection, but after that time intake was not significantly different between groups. However, this indicates that treatment can reduce the decline in intake due to mastitis and contribute to a fast recovery (Yieser et al., 2012).

1.4.4 Summary of key points

Intake measurements, though difficult to obtain, are incredibly useful in understanding the dairy cow. Feed efficiency will continue to be an important issue in dairy cattle as the industry faces more pressure to be economically and environmentally sustainable in the coming years. Genetic parameters have been extensively studied for lactating intake, but not for the dry period. Feeding behavior and intake can also be a useful indicator of disease, especially during the post-partum transition period. Intake can be used to determine when a disease starts to have an effect, and how long that effect lasts, as well as the benefit of treatment.

1.5 Tables

Table 1. Summary of reported heritabilities in the literature for lactating feed intake.

Author(s)	Year	N	h^2	Time
Van Arendonk et al.	1991	360	0.31[1] and 0.46[2]	First 105 DIM
Koenen and Veerkamp	1998	469	0.18 to 0.37	First 25 weeks
Veerkamp and Thompson	1999	628	0.13 to 0.54	First 15 weeks
Karacaören et al.	2006	320	0.12 to 0.34	305 DIM
Berry et al.	2007	755	0.10 to 0.30	305 DIM
Hüttmann et al.	2009	289	0.11 to 0.47	11 to 180 DIM
Vallimont et al.	2010	970	0.18	305 DIM
Buttchereit et al.	2011	682	0.04 to 0.19	11 to 180 DIM
Spurlock et al.	2012	402	0.18 to 0.30[3]	First 150 DIM
			0.22 to 0.60[4]	
Tetens et al.	2014	910	0.26 to 0.37[3]	11 to 180 DIM
			0.27 to 0.37[4]	
Berry et al.	2014	6,957	0.34	70 DIM

[1]Energy intake

[2]Roughage intake

[3]Daily estimates

[4]Monthly estimates

Table 2. Summary of reported genetic correlations in literature between dry matter intake (DMI) at different stages of lactation.

Author(s)	Year	N	Correlation	Time
Koenen and Veerkamp	1998	469	0.04 to 0.98	First 25 weeks
			-0.14	Week 3 and week 25
Veerkamp and Thompson	1999	628	0.24 to 1.00	First 15 weeks
Karacaören et al.	2006	320	0.14	Early to mid
			-0.07	Early to late
Hüttmann et al.	2009	289	0.29 to 0.97	11 to 180 DIM
Buttchereit et al.	2011	682	0.15 to 0.83	Early to mid/mid to late
			-0.51 to -0.55	Early to late
Spurlock et al.	2012	402	> 0.70	First 150 DIM
Tetens et al.	2014	910	0.29 to 0.97	11 to 180 DIM

Table 3. Cost and intake effect of metabolic diseases.

Disease	Cost/case (US$)	Effect on intake
DA	100 to 200	Lower intake 4 days before diagnosis[1]
Ketosis	46 to 92	10.38 kg lower several days before diagnosis[2]
		Lower DMI at -1, +1, and +2 weeks relative to calving[3]
Metritis	358	Lower DMI at -2 through +3 weeks relative to calving[4]
Milk fever	334	Lower DMI on day of onset[5]
		14.7 kg lower between onset and diagnosis[6]
		Greater DMI -2 to +2 weeks relative to calving[7]

[1]Van Winden et al., 2003

[2]González et al., 2008

[3]Goldhawk et al., 2009

[4]Huzzey et al., 2007

[5]Martinez et al., 2014

[6]Bareille et al., 2003

[7]Jawor et al., 2012

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CHAPTER 2. GENETIC PARAMETERS FOR PRE-FRESH INTAKE IN DAIRY CATTLE

A paper to be submitted to *Journal of Dairy Science*.

Brittany N. Shonka and Diane M. Spurlock

2.1 Abstract

The objective of this study was to estimate genetic parameters for dry matter intake (DMI) for Holstein cows during the dry period and lactation. Measurements were recorded on cows from Iowa State University (ISU) and the University of Florida (UF) dairy herds. Individual feed intake data were recorded daily at ISU starting at approximately 30 days before expected calving date, and continued to 150 days in milk (DIM). Individual intake from cows at UF were recorded for approximately 42 days before and after calving. Pre-calving DMI traits were defined as DMI on d -15 (multiparous) or d -7 (primiparous) relative to calving date (DRYDMI), DMI on d -1 before parturition (CALVEDMI), and the slope of a regression line fitted through the last 14 (multiparous) or 7 (primiparous) days before calving (DECLINE). Lactation DMI traits were defined as DMI at 30 DIM (DMI30) and 100 DIM (DMI100), as well as the slope of the increase in intake after calving (INCREASE). The final data set included 245 primiparous and 221 multiparous cows from ISU, and 125 multiparous cows from UF. Heritability estimates were 0.39, 0.64, 0.28, and 0.64 for DRYDMI, CALVEDMI, DECLINE, and DMI30, respectively. The increase in intake after calving was not heritable, and the estimate of heritability for DMI100 (ISU only) was 0.52. The genetic

correlations of DECLINE with DRYDMI and DMI30 were 0.52 and 0.30, and all other correlations were above 0.70. Specifically, the genetic correlation between DRYDMI and DMI30 was 0.94. Intake during the dry period is a moderately heritable trait that is highly correlated with intake during lactation. This result suggests that selection against feed intake during lactation for improved feed efficiency will result in lower genetic potential for feed intake during the dry period, but will not result in a significant effect on the genetic potential for the decline in intake before parturition.

Key words: Holstein cow, heritability, pre-calving intake

2.2 Introduction

Feed efficiency is a critical factor in the economic and environmental sustainability of the dairy industry, as feed costs account for approximately 55% of the total production costs of dairy operations (USDA-ERS, 2014). Genetic selection and improved management methods have increased milk production significantly over the past years, causing a rise in feed intake and feed costs, but also improved feed efficiency. When commodity prices are especially high, feed costs cause major concern among producers. One approach to lower feed costs in an environmentally sustainable way is to select for improved feed efficiency.

To improve feed efficiency through selection, a thorough understanding of the genetic regulation of DMI is needed. Genetic parameters for feed intake have been estimated for lactating dairy cattle, with heritability estimates ranging from 0.04 to 0.54 (Veerkamp and Thompson, 1999; Coffey et al., 2001; Hüttmann et al., 2009, Buttchereit et al., 2011, Spurlock et al., 2012; Tetens et al., 2014). These results suggest that there is

a genetic component to the regulation of feed intake and that selection on it would likely result in a response. It is also important to consider DMI at different stages of lactation, as the genes controlling DMI may vary throughout lactation. Estimates of genetic correlations between DMI at different time intervals range from 0.10 to 0.97, with the general conclusion that the closer the time points are, the stronger the correlation (Veerkamp and Thompson, 1999; Berry et al., 2007; Hüttmann et al., 2009; Buttchereit et al., 2011; Spurlock et al., 2012; Tetens et al., 2014). The genetic regulation of DMI during the non-lactating phase has received minimal attention to date, most likely because the dry period accounts for less than 60 days per year per cow. Also, as milk production is not occurring, production efficiency cannot be determined during this time. However, dry period intake, especially during the final weeks of gestation, plays a crucial role for a profitable lactation. The pre-partum transition period is characterized by a sharp decline in feed intake, and the magnitude of this decline may impact health and production after calving (Grummer, 1995). It is therefore necessary to determine the genetic relationship between intake during the dry period and intake during lactation to fully appreciate potential consequences of selection for decreased feed intake.

The objectives of the current study were to estimate heritabilities for pre-fresh intake traits and to investigate the genetic relationship between pre-fresh intake and intake during the lactating period.

2.3 Materials and Methods

2.3.1 Animals and Data Collection

All animal experiments conducted at ISU were approved by the Iowa State University Institutional Animal Care and Use Committee (Ames). Feed intake data were

collected from 466 cows at the Iowa State University Dairy Farm from March 2008 through November 2010. Records from a single lactation were used for each cow, and cows ranged from parity one to parity seven. Feed intake data collection began approximately 30 days before calving and continued through approximately 150 days in milk (DIM). Cows on the study descended from 103 sires and 362 dams.

Cows were housed in group pens equipped with a Calan Broadbent Feeding system, and each cow was assigned a bin and trained to gain access only to that bin. A TMR was fed ad libitum once (during the dry period) or twice (during lactation) daily. The TMR was formulated to meet or exceed nutritional requirements as determined by the NRC (2001). Once a day, the refusals from each bin were removed. Both the quantity of feed dispensed and the feed reclaimed were recorded electronically to obtain daily intake. Samples of the TMR were taken four times per week and pooled to a weekly sample that was analyzed for DM content (Dairyland Laboratories, Inc., Arcadia, WI). Daily DMI records were discarded if a cow consumed all feed provided on a particular day and if a cow was removed from the pen for medical treatments. Cows were weighed and scored weekly for body condition (Elanco Animal Health, 1996) by a single trained evaluator.

2.3.1.1 University of Florida (UF)

Feed intake data were recorded for a total of 144 cows from August 2007 through November 2010 at the University of Florida Dairy Research Unit, as described by do Amaral et al. (2009), do Amaral et al. (2011), Tao et al. (2011), and Tao et al. (2012). Data from June 2011 through October 2011 were collected similarly to Tao et al. (2012).

All experimental procedures in the aforementioned studies were approved by the Institutional Animal Care and Use Committee of the University of Florida.

2.3.2 Definition of Dry Matter Intake traits

In order to predict missing values and minimize day to day variation, smoothing splines were applied to the daily intake data using the PROC TRANSREG procedure and a smoothing parameter of 70 (SAS Institute, 1999). Cows were excluded if they did not have two or more feed intake records before one week (primiparous) or two weeks (multiparous) prior to parturition. Some cows also did not have both lactating and pre-partum data because intake was not recorded if a cow was undergoing treatment for a medical condition and some cows did not continue on the study after parturition. The final ISU data set included 466 cows with pre-partum and 400 cows with post-partum data, while the final UF data set included 125 cows with pre-partum and 103 with post-partum data.

Multiple traits were defined to characterize DMI at different stages before and after parturition. Intake during the dry period was represented by DRYDMI, defined as DMI on the day prior to initiation of the rapid decline in feed intake that precedes parturition. For primiparous cows, DRYDMI was represented by day -8 relative to parturition (day 0); for multiparous animals, DRYDMI was represented by day -15. Intake immediately before calving (**CALVEDMI**) was DMI on day -1 for all cows. The decrease in intake prior to parturition (**DECLINE**) was calculated as the slope of the regression line fit through the daily DMI prior to parturition. The regression for primiparous cows was fitted for days -7 through -1 while the regression was fitted through days -14 through -1 for multiparous cows. Similarly, INCREASE represented

the rate of increase in DMI after parturition and was calculated as the slope of the regression line fitted through days 1 to 21 post-partum. The DMI at 30 DIM (DMI30) was chosen to represent a point in early lactation, after the transition period, and DMI at 100 DIM (DMI100) was selected as a time when lactating cows had returned to positive energy balance, on average (Spurlock et al., 2012).

2.3.3 Genetic analysis

Heritabilities and genetic correlations were estimated using ASReml software (Gilmour et al., 2008). Fixed effects included parity (1, 2, or 3 through 7), calving age (divided into 7 groups based on parity and age), year-season of calving (year, 2007 through 2011; season as winter (December to February), spring (March to May), summer (June to August), and fall (September to November)), calf gender (male, female, or twins), and live or dead status of the calf. When the ISU and UF data were combined, farm (1 or 2) was also included as a fixed effect. The model for analysis was

$$y_i = \mu + lac_i + calveage_i + seascalv_i + calf_i + status_i + a_i + e_i,$$

where y_i = individual phenotype; μ = overall mean; lac_i = fixed effect of lactation (1 to 3) for cow i; $calveage_i$ = fixed effect of age at calving class (1 to 7) for cow i; $seascalv_i$ = fixed effect of year and season of calving (1 to 12) for cow i; $calf_i$ = fixed effect of calf gender (1 to 3) of cow i; $status_i$ = fixed effect of born dead or alive (1 or 2) for cow i; a_i = additive genetic effect for the ith cow; and e_i = random residual error. To calculate the genetic and phenotypic correlations among the traits, the model was extended to be a multivariate model. A five generation pedigree was used that included 3156 individuals for the ISU data set, 1880 individuals for the UF data set, and 4358 individuals when combined.

2.4 Results and Discussion

Average daily DMI during the dry period and the predicted DMI after applying the smoothing spline are shown in Figure 1. Intake was relatively constant from 30 to 15 days prior to parturition and then gradually declined as calving approached. As expected, multiparous cows ate more than primiparous cows, on average. This is likely due to differences in body weight, as multiparous cows from ISU and UF were larger than primiparous cows by 109 and 64 kg, respectively (Table 1). Body condition scores were similar between primiparous and multiparous cows from ISU, but cows from UF were much less conditioned than ISU cows (Table 1). The depression of DMI as parturition approaches is also evident in Figure 1. On average, DMI of multiparous cows began to decline approximately 2 weeks before calving with the sharpest decline occurring during the last week, whereas primiparous cows sharply declined around -7 DIM. This is consistent with Hayirli et al. (2002), who found that primiparous cows had consistent DMI until the week before calving, while older cows experienced a more gradual decline in DMI. During the final two weeks of gestation in the current study, DMI of primiparous cows decreased by 15%, with 80% of that occurring during the final week of gestation. Also, ISU cows decreased DMI by 14% during the final two weeks, with 73% of that decrease occurring during the week prior to calving. The UF cows had the greatest decrease in DMI, declining 25% during the final 2 weeks, with 80% of that occurring during the last week. A reason for the differences in DMI between ISU and UF multiparous cows could be that animals from UF were more likely to be experiencing heat stress, as all UF data were collected during the summer months. The declines in

DMI observed in this study are less than those reported by Hayirli et al. (2002) and Marquardt et al. (1977), who observed decreases in DMI between 32.2 and 52%.

Heritability estimates and correlations for the ISU data and for the combined data set are shown in Tables 3 and 4, respectively. Dry matter intake during the dry period was moderately to highly heritable, and heritability estimates for DMI measured during the dry period and lactation were similar. The estimates for lactating DMI were similar to monthly averages reported by Spurlock et al. (2012) for the same population, which ranged from 0.22 to 0.60 for the first five months of lactation. In general, estimates of DMI heritability were similar to or slightly higher than those reported in the literature, which range from 0.04 to 0.54 (Veerkamp and Thompson, 1999; Coffey et al., 2001; Hüttmann et al., 2009, Buttchereit et al., 2011, Spurlock et al., 2012; Tetens et al., 2014). Many of these previous studies only used records for primiparous animals (Veerkamp and Thompson, 1999; Coffey et al., 2001; Hüttmann et al. 2009; Buttchereit et al., 2011; Tetens et al., 2014), population sizes were variable (289 to 910 animals) and a variety of statistical approaches were used to estimate DMI heritability. All of these factors may contribute to the range in estimates found in current literature. Estimates of DRYDMI, CALVEDMI, and DMI30 heritability were not different from zero for the UF data alone, due to the high standard errors resulting from a relatively small dataset.

Estimates of genetic correlations between intake traits ranged from 0.30 to 0.94 in the combined data set (Table 4). Most notably, the correlations of DRYDMI and CALVEDMI with DMI30 were 0.88 and 0.94, respectively. In the ISU data set, the correlation between DRYDMI and DMI100 was also high (0.86). These correlations suggest that DMI in the dry period is under similar genetic regulation as DMI during

early lactation. Positive estimates of genetic correlations in the literature between different stages of lactation range from 0.10 to 0.97 (Veerkamp and Thompson, 1999; Berry et al., 2007; Hüttmann et al., 2009; Buttchereit et al., 2011; Spurlock et al., 2012; Tetens et al., 2014). In general, the correlations are strong between time points that are close together, and weaken as the interval grows larger, leading others (Tetens, et al., 2014; Hüttmann et al., 2009; Berry et al., 2007; Koenen and Veerkamp, 1998) to conclude that the genetic regulation of DMI differs with stage of lactation. However, Spurlock et al. (2012) found correlations above 0.70 for DMI measured in the first five months of lactation. Thus, at least in this ISU population, the genetic regulation of DMI appears to be consistent throughout the dry period and first half of lactation.

Estimated parameters for DECLINE and INCREASE were lower than for actual intake traits (Tables 3 and 4). INCREASE was not heritable in this data set, while DECLINE was moderately heritable. Genetic correlations between DECLINE and other intake traits were 0.30 to 0.86, with the highest correlation between DECLINE and CALVEDMI. Phenotypic correlations were low, and in some cases not different from zero. The magnitude of decline in feed intake prior to parturition has been associated with metabolic disorders post-partum (Grummer et al., 2004). Thus, it is important to define how selection for DMI during lactation may impact DECLINE at both the genetic and phenotypic levels. Although the population used to investigate these correlations in the current study was relatively small, moderate and low genetic and phenotypic correlations were estimated for DECLINE and DMI30. In the ISU population estimates for genetic and phenotypic correlations between DECLINE and DMI100 were slightly lower than those for DECLINE and DMI30. These results are important because they indicate

selection for feed intake during lactation will likely not contribute to an exaggerated decrease in DMI prior to parturition, at least at the phenotypic level. Since the decline in intake, rather than the level of DMI, contributes to metabolic issues after parturition in most cases (Grummer et al., 2004) the current research indicates selection for reduced DMI during lactation will likely not have a deleterious effect on DMI during the transition period.

2.5 Conclusions

Intake during the dry period is a moderately heritable trait that is genetically similar to intake during lactation. Correlations between intake and intake depression prior to calving suggest that selecting for feed efficiency will not have a negative impact on the magnitude of intake depression in DMI that occurs prior to parturition, but selection for reduced feed intake during lactation will likely contribute to reduced DMI throughout the dry period.

2.6 Acknowledgements

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2.7 Tables

Table 1. Summary of animals.

		BCS[1]			BW[1]		
	N	Dry	30 DIM	100 DIM	Dry	30 DIM	100 DIM
ISU							
Parity 1	245	4.34 ± 0.25	3.63 ± 0.44	3.33 ± 0.48	647 ± 60	551 ± 55	548 ± 48
Parity 2+	221	4.23 ± 0.37	3.47 ± 0.58	2.99 ± 0.61	756 ± 79	637 ± 62	623 ± 55
UF							
Parity 2+	125	3.52 ± 0.44	3.17 ± 0.41	-	711 ± 79	621 ± 60	-

[1]Body condition score (BCS) and body weight (BW) calculated as the average throughout the dry period, the average from 1 to 30 DIM, and the average from 1 to 100 DIM, all plus or minus the standard deviation.

Table 2. Summary of traits.

Trait	N	Mean (kg)	SD	Minimum	Maximum
DRYDMI	557	11.37	2.11	4.09	17.15
CALVEDMI	557	10.04	2.56	2.44	18.39
DECLINE	557	-0.12	0.17	-0.86	0.44
DMI30	503	19.91	3.48	6.74	31.20
DMI100[1]	400	23.74	3.39	15.09	32.91
INCREASE	459	0.33	0.22	-0.54	1.23

[1]Records on ISU cows only.

Table 3. Heritability estimates (SE) and correlations (SE) for all traits in the ISU data set. Heritabilities are shown on the diagonal, genetic correlations are shown above the diagonal, and phenotypic correlations are shown below the diagonal.

Item	DRYDMI	CALVEDMI	DECLINE	DMI30	DMI100
DRYDMI	0.40 (0.11)	0.91 (0.08)	0.52 (0.31)	0.90 (0.12)	0.86 (0.17)
CALVEDMI	0.66 (0.03)	0.62 (0.11)	0.81 (0. 13)	0.73 (0.12)	0.62 (0.15)
DECLINE	-0.15 (0.05)	0.59 (0.03)	0.25 (0.11)	0.30 (0.24)	0.13 (0.27)
DMI30	0.40 (0.05)	0.45 (0.05)	0.16 (0.05)	0.65 (0.13)	0.95 (0.07)
DMI100	0.30 (0.05)	0.30 (.05)	0.08 (0.06)	0.70 (0.03)	0.52 (.13)

Table 4. Heritability estimates (SE) and correlations (SE) for all traits in the combined data set. Heritabilities are shown on the diagonal, genetic correlations are shown above the diagonal, and phenotypic correlations are shown below the diagonal.

Item	DRYDMI	CALVEDMI	DECLINE	DMI30
DRYDMI	0.39 (0.11)	0.88 (0.08)	0.52 (0.28)	0.94 (0.12)
CALVEDMI	0.65 (0.03)	0.64 (0.11)	0.86 (0.10)	0.70 (0.12)
DECLINE	-0.13 (0.05)	0.62 (0.02)	0.28 (0.11)	0.30 (0.23)
DMI30	0.40 (0.04)	0.43 (0.04)	0.15 (0.05)	0.64 (0.13)

2.8 Figures

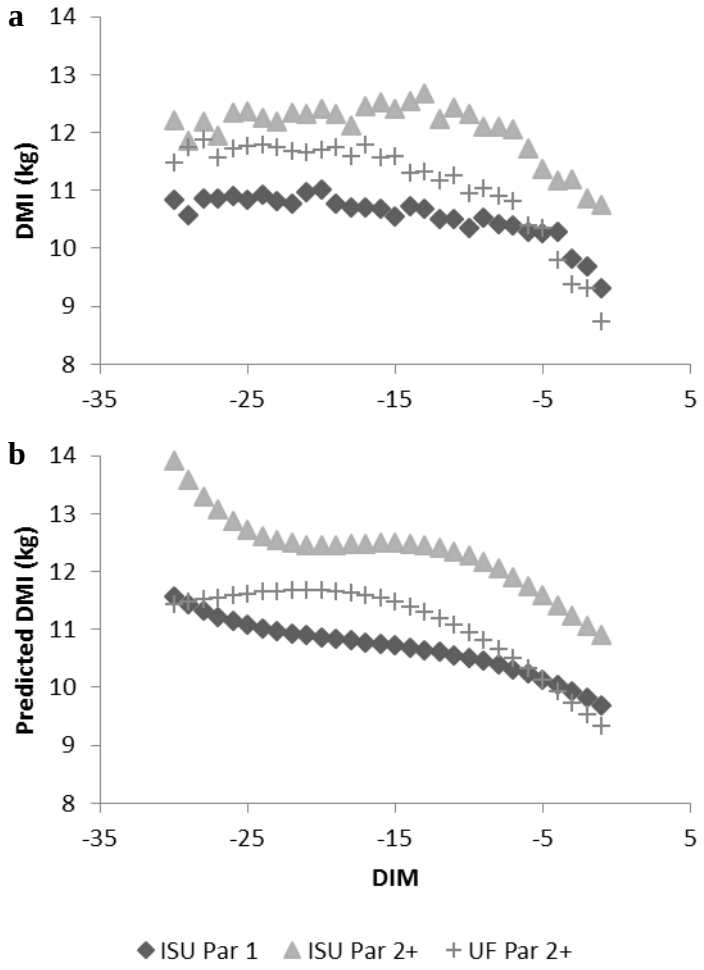

Figure 1. Average daily DMI (a) and average predicted DMI (b) during the dry period.

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CHAPTER 3. THE IMPACT OF LAMENESS ON MILK YIELD AND INTAKE IN HOLSTEIN COWS

A paper to be submitted to *Journal of Dairy Science*.

Brittany N. Shonka and Diane M. Spurlock

3.1 Abstract

Lameness is costly to the dairy industry and compromises the welfare of the animal. Lameness causes a depression in milk production and intake, though the magnitude of this depression is not well quantified. The objective of the current study was to determine the effect of lameness on dry matter intake (DMI) and milk production. In this study, 400 Holstein cows, including 49 cows that experienced at least one occurrence of lameness during the first 150 days in milk (DIM), were monitored for daily milk yield and dry matter intake (DMI). Data were split into two groups based on whether lameness occurred in early lactation (<90 DIM) or mid-lactation (91 to 150 DIM) to determine differences based on stage of lactation To analyze the impact of lameness on yield and intake 2 weeks before treatment, the day of treatment, and 2 weeks after treatment, mixed model procedures were used. Milk production and DMI decreased by 2% and 14%, respectively, between pre-treatment and treatment intake, for cows in the early group, and by 17% and 11% for cows in the mid-lactation group. Two weeks after treatment, intake recovered to pre-treatment levels while milk production did not. Lame cows had decreased intake and milk production when compared to healthy cows immediately before treatment. Milk yield was affected for at least 2 months after

treatment. These results help to quantify loss of profitability and the impact of treatment for cows affected by lameness. To maintain the welfare of the animal and decrease the loss in profit due to milk yield, prevention, early detection, and treatment for lameness are crucial.

3.2 Introduction

Lameness is a complex disease of major concern to dairy producers due to its negative impact on profitability and animal welfare. It is the second most costly disease after mastitis, and costs associated with lameness include increased veterinary costs, increased labor costs, decreased milk production, and possibly the loss of the animal by culling or death (González et al., 2008). Cha et al. (2010) estimated the cost of a sole ulcer, digital dermatitis, and foot rot to be $216.07, $132.96, and $120.70 per case, respectively. There is evidence that high milk yields and high lameness prevalence are associated, so lameness will likely continue to be a challenge as selection for increased production continues (Potterton et al., 2012). Incidence rates of lameness are extremely variable. In the United Kingdom, Australia, and the Netherlands, reported rates were 5 to 70%, 0 to 50%, and 9 to 50%, respectively (Green et al., 2002). This high variability likely reflects differences not only in detecting lameness but also differences among farms in types and prevalence of lameness (Green et al., 2002). The high degree of subjectivity in defining lameness makes lameness difficult to analyze.

In the United States, the effect of lameness on production and non-production traits is not well quantified, due in part to variability in reporting of lameness events. However, clinical lameness has been shown to have a detrimental effect on milk yield

both before and after treatment (Green et al., 2014). This decline in production is likely due to an effect of lameness on feed intake, and feeding behavior is clearly altered by lameness. Fewer trips to the feeder with increased feeding rates are associated with lameness (González et al., 2008; Palmer et al., 2012; Norring et al., 2014). González et al. (2008) found lower intake starting at the onset and continuing until treatment for acute lameness, while Palmer et al. (2012) found a decrease in intake at 60 DIM, but not at 120 DIM, compared to pre-treatment levels. Norring et al. (2014) determined lameness by locomotion score, and found that cows with higher scores had lower intake. The aim of the current study was to quantify the impact of clinical lameness on milk yield and dry matter intake (DMI) in lactating Holstein cows, before and after treatment for lameness.

3.3 Materials and Methods

Milk production and feed intake data were collected on 400 Holstein cows at the Iowa State University Dairy Farm from 2008 through 2010 as described by Spurlock et al. (2012). Lameness was diagnosed, treated, and recorded by farm staff and veterinarians. For this study, clinical lameness was defined as a health event that affected cow motility and resulted in treatment, typically either foot trimming or medication. Injuries that affected motility were not included as lameness.

The final data set included 49 cows that were treated for lameness during their first 150 DIM of the lactation studied. The effect of lameness on milk yield and DMI was evaluated using 2 approaches. First, average milk yield and DMI were compared at 3 time points relative to treatment: 2 weeks prior to treatment, at treatment, and 2 weeks after treatment. Cows treated between 0 and 90 DIM were considered to be in early

lactation (primiparous=23; multiparous=11), while cows treated between 91 and 150 DIM were considered to be mid-lactation (primiparous=7; multiparous=8) when lameness occurred. All analyses were done separately for cows treated for lameness in early versus mid-lactation. Mixed model procedures in SAS were used to investigate changes in milk yield and DMI with time relative to treatment. Parity (1 or 2 and greater) and time relative to treatment (-2, 0, +2) were fitted as fixed effects and cow was fit as a random effect. The main effect of time relative to treatment was significant, and differences among means at the three time points were determined using Tukey's test.

Secondly, milk production and DMI of cows diagnosed as lame were compared to the average of healthy cows. There were 100 primiparous and 69 multiparous animals with no recorded health events during the first 150 DIM. Intake and production data from these cows were used to generate an average lactation curve for healthy cows based on parity (first lactation or second and greater lactation) through 150 DIM (intake) or 305 DIM (milk production). Intake data for lame cows in the months surrounding the lameness event was then compared to the average of healthy cows on the same day.

3.4 Results and Discussion

Cows in the final data set were considered lame for multiple reasons. There were 7, 34, and 4 cows recorded with abscesses, foot/hoof rot, and sore feet, respectively. Four cows were coded as lame, and no apparent cause of lameness was given. On average, cows diagnosed as lame in early lactation were treated at 31 ± 27.13 DIM, whereas cows diagnosed in mid-lactation were treated at 124.4 ± 15.42 DIM, indicating a high variability of time of treatment for lameness (Figure 1). Least square means for DMI and

milk yield, as well as p-values for parity, time relative to treatment, and the interaction between the two are given in Table 1. For both DMI and milk yield in either group, the interaction of parity and time relative to treatment was not significant. For intake in the early group, parity was not significant, but time relative to treatment was, while for milk yield both parity and time were significant. This indicates that intake is affected similarly between primiparous and multiparous cows, but decreases in milk yield due to lameness differ by parity. In the mid-lactation group, parity and time relative to treatment were both significant for intake and milk yield.

The impact of lameness on DMI for cows diagnosed as lame in early lactation and mid-lactation is shown in Table 1 and Figure 2a. Cows diagnosed with lameness in early lactation consumed significantly less dry matter at treatment than at 2 weeks before treatment ($p<0.0001$). This was also realized for cows diagnosed in mid-lactation, but not to the same extent ($p=0.002$). This suggests that lameness affects DMI differently in different stages of lactation, which is consistent with the results of Palmer et al. (2012) who found that high locomotion scores were associated with a decrease in intake at 60 DIM but not at 120 DIM. Figure 2a shows a clear decrease in intake for both groups approximately 4 days before treatment. After treatment, intake returned to pre-treatment levels very quickly, at approximately 2 days after treatment. The difference between pre-treatment and post-treatment DMI was significant for early ($p=0.0014$) but not for mid-lactation lameness ($p=0.60$). Because data for this study were collected during the first half of lactation, the higher intake after treatment in the early group may reflect either a compensatory increase in intake following lameness, or the expected increase in DMI associated with increasing DIM. González et al. (2008) found that cows classified as

lame showed significant changes in feeding behavior, including less daily feeding time, fewer number of visits to the feeder, and higher feeding rate during a 30 day period before treatment for lameness. In the same time period after treatment, cows were shown to recover by increasing feeding time, meals per day, and lowering the feeding rate. Norring et al. (2014) also determined that lameness is associated with these same changes in behavior.

The comparison of DMI between lame cows and healthy cows is shown in Figure 3. Primiparous animals that became lame in early lactation had less intake compared to healthy cows on the days immediately before and after treatment (Figure 3a). The DMI of multiparous cows did not recover as quickly as the first lactation cows, and did not return to pre-treatment intake levels. This indicates that lameness has a lasting effect on intake in multiparous cows. However, it must be noted that there are not as many records before treatment, especially for cows that were treated within the first 30 days of lactation. First lactation cows diagnosed as lame in mid-lactation show a clear deviation from pre-treatment levels, and do not fully recover after treatment (Figure 3b). Multiparous cows also show a deviation, but do return to pre-treatment intake levels soon after treatment (Figure 3b). Intake data are limited in this study for cows diagnosed as lame in mid-lactation, as intake records were only available through 150 DIM. This explains in part the large amount of variation shown after treatment in Figure 3b, and why there are no records after 50 DIM following treatment. The deviations from average for both the early and mid-lactation group follow the trends shown in Table 1 and Figure 2a.

The impact of lameness on milk yield for the 4 weeks surrounding treatment differed between the cows treated in early versus mid-lactation (Table 1 and Figure 2b).

Milk yield of cows treated in early lactation did not decline at treatment (P=0.88), but was higher after treatment compared to pre-treatment and treatment yield (P=0.0027 and P<0.0001, respectively). Milk yield of cows treated in mid-lactation showed a different pattern, with lower yields at treatment compared to the pre-treatment period (P=0.0004). In Figure 2b, a sharp decline in milk production is observed approximately 10 days before treatment for the mid-lactation group, while cows in the early group did not decline in milk production until closer to the day of treatment for lameness. This result is comparable to the findings of Green et al. (2014), who found that milk yield significantly decreased in the month before the treatment of the specific lameness events of sole ulcers and white line disease. Green et al. (2002) found that in general, lameness may impact milk yield as early as 4 months before diagnosis and treatment. In the current study, the day of decline was much earlier for milk production than for intake, indicating that monitoring cows that are down in milk may be more useful than monitoring intake in early lactation. In the mid-lactation group, post-treatment yield was not different from treatment yield (P=0.44). Green et al. (2002) observed that lameness affected milk yield for up to 5 months after treatment. Figure 2b shows that, although milk yield recovers quickly (though not as quickly as DMI), the cow has not fully recovered from lameness after treatment, causing a loss in profit. Overall, Green et al. (2002) estimated that lameness accounted for a loss of 360 kg of milk during a 305-day lactation.

The milk yield comparison between lame cows and healthy cows is displayed in Figure 4. There are less milk yield records before treatment for cows diagnosed as lame in early lactation, accounting for part of the high variability in deviations before treatment day. Primiparous cows that were lame in early lactation deviated from average close to

treatment day, and returned to average quickly (Figure 4a). Multiparous cows, however, did not reflect a change in yield until after treatment, and had a more gradual recovery to pre-treatment levels (Figure 4a). Both multiparous and primiparous animals diagnosed as lame in mid-lactation show the lasting impact of lameness on milk yield. Primiparous cows drop in production immediately before treatment, but do not recover as quickly as primiparous cows diagnosed in early lactation. As lactation continues, primiparous animals deviate further from the average of healthy cows. Multiparous cows show a similar pattern in mid-lactation as they do in early lactation, where they deviated from healthy cows immediately before treatment and gradually returned to pre-treatment levels.

Previous studies focus on feeding behavior instead of DMI, and rarely describe the effect on milk yield and DMI together. Changes in feeding behavior do not necessarily imply a decrease in DMI, especially considering that lameness increases feeding rate (González et al., 2008; Norring et al., 2014). Knowledge of the impact of lameness can aid in advocating for rapid detection and treatment, which will improve animal welfare. Examining feed intake and milk yield curves can give researchers and producers a better idea of when these decreases occur and how long the impact lasts, allowing for more insight as to the negative economic impact lameness.

3.5 Conclusions

Lameness significantly reduces feed intake and milk yield. DMI decreased by 14% and 11% for cows in early and mid-lactation, respectively, from intake and yield levels two weeks before treatment to the day of treatment. Two weeks after treatment,

intake returned to pre-treatment levels. Milk yield decreased by 3% and 17% between pre-treatment and treatment time points for early and mid-lactation cows, respectively. However, milk yield did not return to pre-treatment levels by two weeks after treatment. The results stress the importance of early detection and treatment of lameness to minimize the decrease in intake and possibly deter the decrease in milk yield that is associated with lameness. Lameness caused deviations from averages of healthy cows for DMI and milk yield, and lasting impacts can be seen for at least 2 months after treatment. This indicates the importance of lameness prevention strategies.

3.6 Tables

Table 1. Least square means (in kg) of intake and milk yield two weeks before treatment (pre-treat), at treatment (treat), and two weeks after treatment (post-treat) for cows diagnosed at lame in early lactation (n=34) or mid-lactation (n=15). P-values are shown for the effects of parity, time relative to treatment, and the interaction between them. Means with different letters differ significantly ($p \leq 0.05$).

Trait	Stage of Lactation	Pre-treat (kg)	Treat (kg)	Post-treat (kg)	Parity (p-value)	Time (p-value)	Parity*Time (p-value)
DMI	Early	18.56^{a} (0.85)	15.93^{b} (0.69)	20.55^{c} (0.68)	0.1234	<0.0001	0.9224
	Mid	23.92^{a} (0.83)	21.31^{b} (0.87)	23.19^{ab} (0.95)	0.0106	0.0028	0.8832
Milk	Early	35.61^{a} (1.57)	34.87^{a} (1.36)	40.98^{b} (1.36)	0.0004	<0.0001	0.6990
	Mid	44.46^{a} (2.13)	37.06^{b} (2.13)	39.36^{b} (2.33)	0.0335	0.0005	0.1816

3.7 Figures

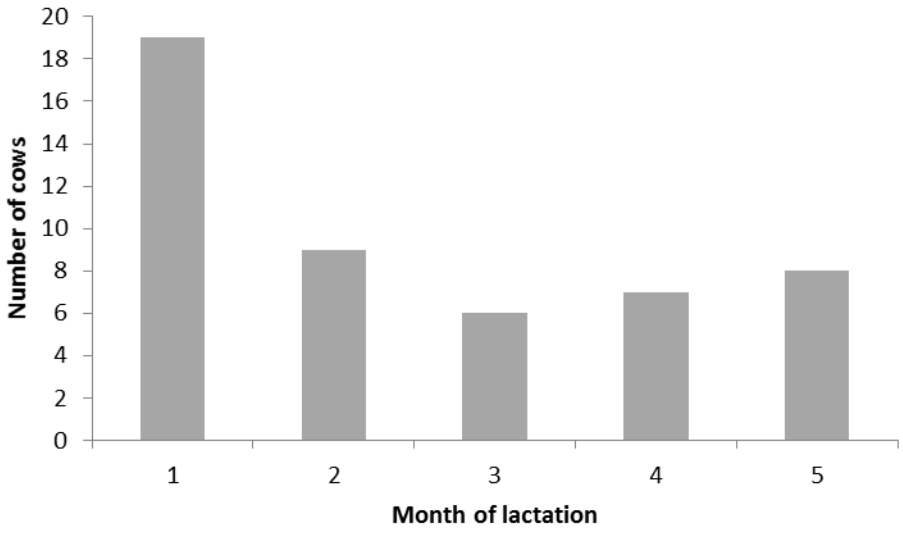

Figure 1. Number of cows diagnosed with lameness by month of lactation.

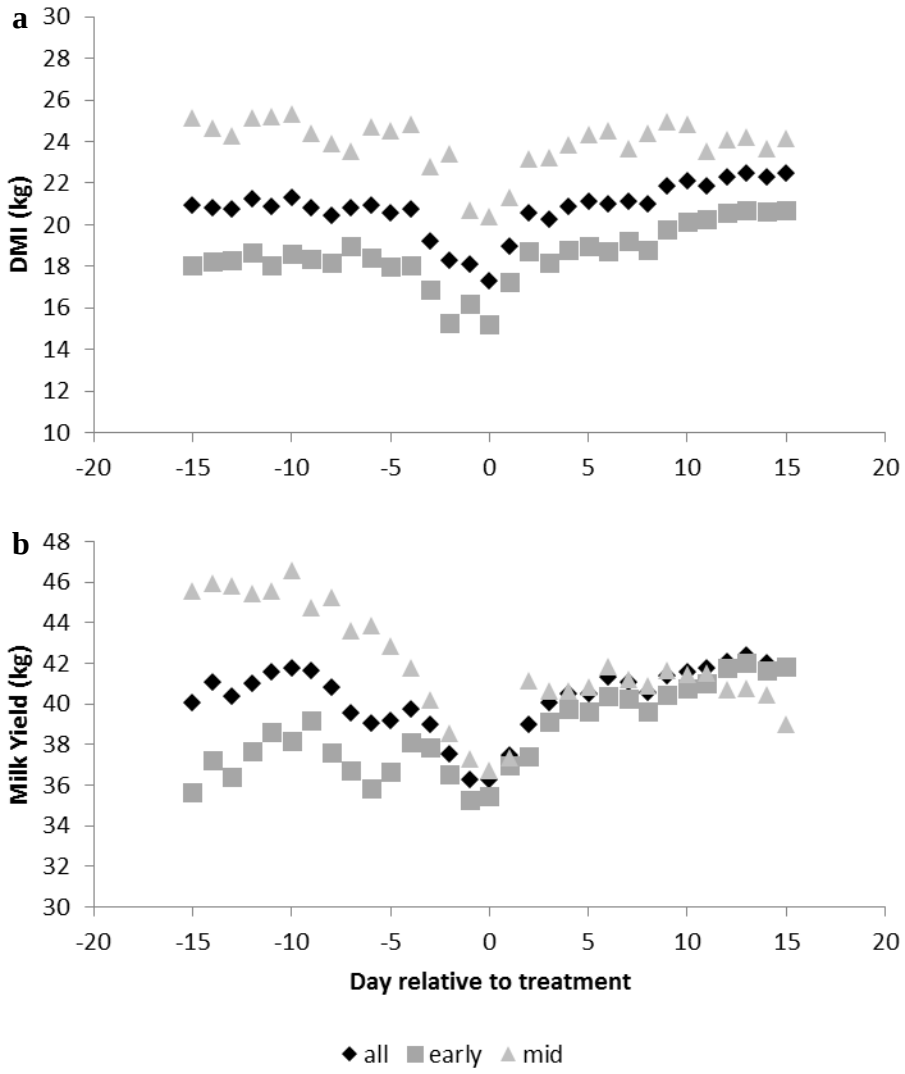

Figure 2. Lsmeans for daily DMI (a) and milk yield (b) relative to treatment day for cows diagnosed as lame in early lactation (n=34) and mid-lactation (n=15).

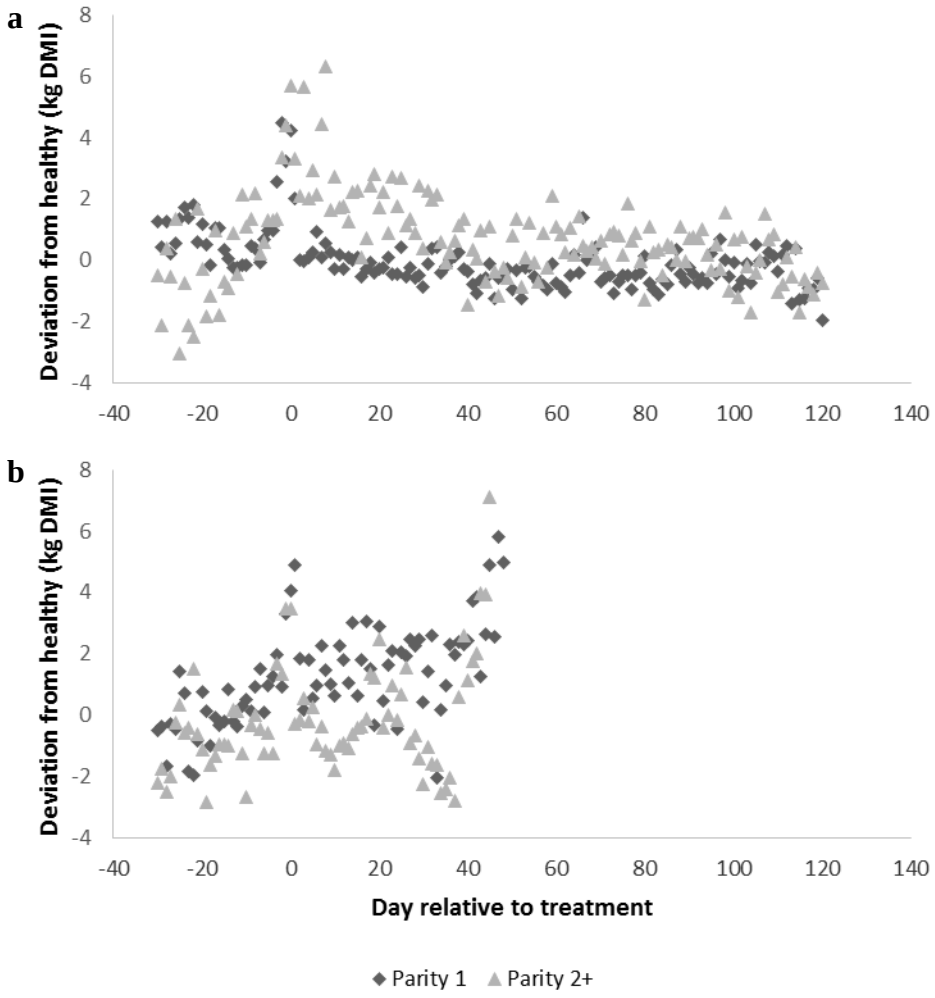

Figure 3. Deviation of DMI of lame cows from the average of healthy cows for cows diagnosed in early lactation (a; n=34) and mid-lactation (b; n=15) relative to day of treatment (day 0).

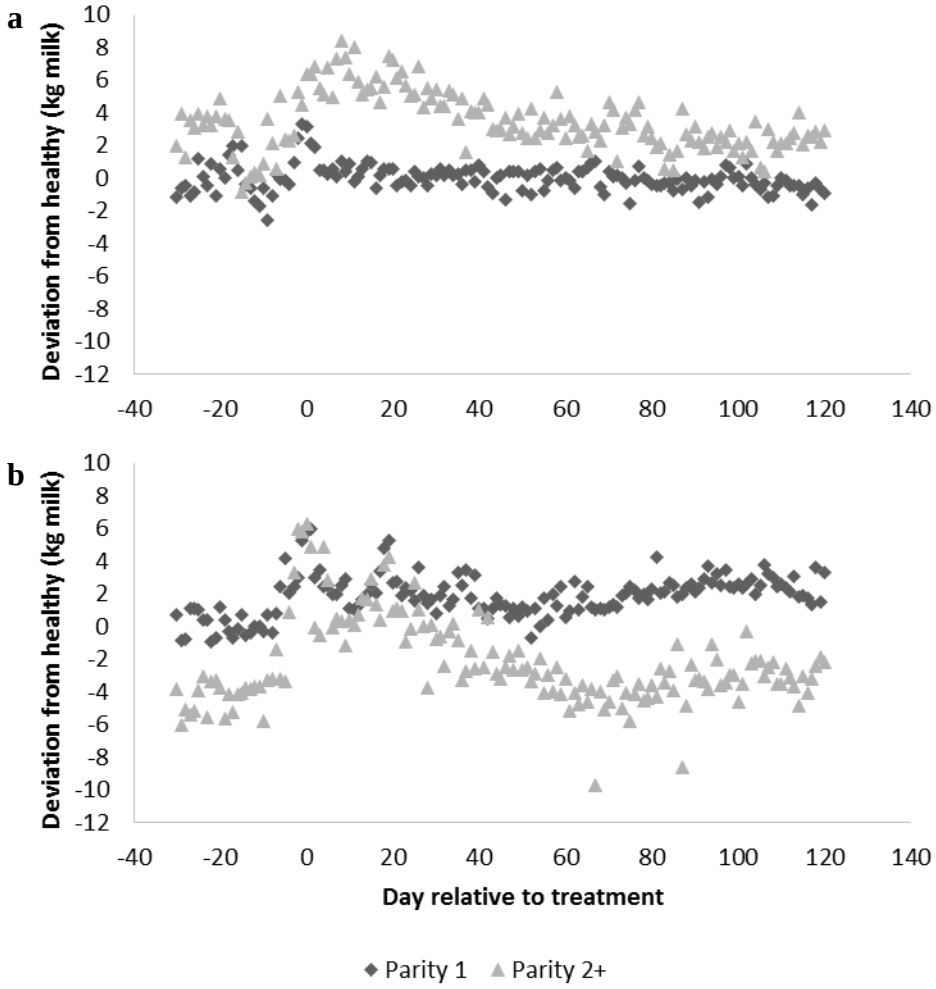

Figure 4. Deviation of milk yield of lame cows from the average of healthy cows for cows diagnosed in early lactation (a; n=34) and mid-lactation (b; n=15) relative to day of treatment (day 0).

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CHAPTER 4. GENERAL CONCLUSIONS AND DISCUSSION

High feed costs, consumer interest in animal welfare and environmental sustainability, and the pressure to feed the growing population have pushed the dairy industry to strive to better understand the dairy cow. To do this, feed intake, more specifically dry matter intake (DMI) has been extensively studied. The genetic regulation of DMI has been studied during lactation because DMI during lactation is used in calculations of feed efficiency, and to understand the genetic regulation behind it in attempts to one day incorporate it into a breeding goal to promote economic and environmental sustainability. However, the dry period, though short, has been overlooked. Feeding behavior and DMI during lactation have also been studied to determine whether it is useful in predicting disease to improve animal welfare and minimize health costs. Therefore, intake can be used in a variety of ways to improve the economics of a dairy farm. The ideas presented in this thesis fill gaps in the knowledge about the genetic regulation of DMI, as well as the relationship of lameness with DMI and milk yield.

Dry period DMI is a trait that will likely respond to selection, due to its moderate heritability and presence of genetic variation. The high correlations between dry period DMI and lactating DMI suggest they are under similar genetic regulation, and that changes in one will affect the other at the genetic level. Selecting for more efficient cows by selecting against DMI could lower feed costs throughout the entire life cycle of the cow, not just during lactation. Suggestions have been made that the genes controlling intake change as lactation continues. This would mean that intake at different stages of

lactation are separate traits, and would need to be treated as such if used as a selection tool. The results of the current study found a high correlation between DMI in early and mid-lactation, suggesting that intake is not regulated differently throughout lactation. However, low feed intake during the dry period is thought to be a factor in development of transition cow diseases, such as displaced abomasum. Therefore, whether or not lower intake during the dry period, especially the time very near to calving, is a negative or positive change needs to be investigated further before feed efficiency becomes part of a selection index. The low correlation between lactating DMI and the magnitude of intake depression just prior to parturition indicates that lowering DMI in lactation will merely shift the pre-fresh DMI curve downward, and not change the slope of the depression. Cows with a more severe depression in DMI are more inclined to develop transition cow diseases, so the low correlation is a positive finding. However, estimates from the current study would benefit from a larger population size. Validation in other Holstein populations is necessary. It can be difficult to compare genetic parameter estimates across studies due to differences in animal numbers, population structure, and statistical methods. Measurement errors between studies can also make comparisons difficult. Future research needs to be done to further investigate dry period DMI and the relationship it has with other traits of interest. An understanding of these areas will allow for a more complete understanding of intake in the dairy cow and the positive and negative consequences of selecting for feed efficiency.

Lameness was found to have impactful effects on both milk production and feed intake. Lameness in multiparous cows had a more severe impact on milk production, as it took longer for their milk production to recover to pre-treatment levels. Primiparous

animals recovered quite quickly. For all cows, using DMI and milk production as a tool for detecting lameness early is difficult, as declines may not occur until a few days before treatment. Multiparous cows with lameness occurring during the first 90 days of lactation did not deviate from average until just after treatment. Cows treated in early lactation showed significantly lower DMI at treatment when compared to pre-treatment levels, and had higher intake after treatment than they did before treatment. This is most likely a reflection of where the cows are on the lactation curve, as these cows may or may not have peaked in milk production. This was not seen in mid-lactation cows, as their intake returned to pre-treatment levels. Milk yield was very different based on stage of lactation. Lame cows in early lactation did not drop significantly in milk production, but produced more after treatment. Again, this is mostly likely because the cow has not yet peaked. Lame mid-lactation cows significantly decreased in milk production at treatment, but production after treatment was not different than treatment levels. Since these cows recovered in feed intake, it suggests that intake may not be the cause for lower milk yield. Deviations of milk yield from the average of healthy cows were apparent for at least 2 months following treatment, indicating a significant loss in profit due to lower milk production. The findings of the current study emphasize the importance of lameness prevention on farms to avoid profit loss. The results, particularly for the cows diagnosed in mid-lactation, are only for a small number of animals, however, the difficulty in collecting individual intake data hinders the ability to evaluate lameness on a larger scale. Future research directions should focus not only on lameness but the impact of other diseases on DMI as well. More knowledge about these effects will stress the importance of monitoring animals carefully and quickly diagnosing and treating. This will not only

decrease health costs on a dairy farm, but it will also positively impact the welfare of the farm.

The studies included in this thesis show that individual intake in dairy cattle is an important tool not only in understanding the biology and genetics of the cow, but also the important economic role it plays in a dairy operation. The continued collection and analysis of intake data will be important in future research to improve both economic and environmental sustainability, as well as animal welfare.

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