

**Phenotypic Relationships between Lactation persistency and  
Common Health Disorders in Dairy Cows**

Jayasooriya Arachchige Don Ranga Niroshan Appuhamy

**Thesis submitted to the faculty of the Virginia Polytechnic Institute and  
State University in partial fulfillment of the requirements for the  
degree of Master of Science**

**In  
Dairy Science**

**Approved by**

**Dr. B. G. Cassell**

**Dr. R. E. Pearson**

**Dr. R. M. Akers**

**Dr. R. M. Lewis**

**November 10, 2006**

**Blacksburg, VA**

**Keywords: cow, disease, lactation persistency,  
phenotypic relationship**

**Copyright 2006, Ranga Appuhamy**

# **Phenotypic Relationships between Lactation Persistency and Common Health Disorders in Dairy Cows**

**Jayasooriya Arachchige Don Ranga Niroshan Appuhamy**

## **Abstract**

Lactation persistency is defined as the ability of a cow to maintain production at a higher level after peak yield. Hypothetically, more persistent cows are less susceptible to health and reproductive disorders. The objective of this research was to investigate the phenotypic relationships of common health disorders in dairy cows to lactation persistency. The relationships with peak yield and days in milk (DIM) at peak yield were also studied. Two separate investigations (Study 1 and Study 2) were performed. Study 1 used treatment incidence data and daily milk weights of 991 lactations from experimental dairy herds at Virginia Tech and Pennsylvania State University. Milk yield persistency (PM) was estimated for individual lactations using daily milk weights. In Study 2, producer recorded health data of 87555 lactations from 398 commercial herds were used. PM, fat (PF), and protein (PP) persistencies were estimated from TD yields. Mastitis only in the first 100 days, only after 100 DIM, and at any stage of lactation, and reproductive disorders including metritis, retained placenta, cystic ovaries, metabolic diseases including ketosis, milk fever and displaced abomasums, and lameness were considered in both studies. Mastitis both before and after 100 DIM was considered only in Study 1. Each disease was defined as a binary trait distinguishing between lactations with at least one incidence (1) and lactations with no incidences (0). Standardized measures of the persistencies, uncorrelated with yield, were calculated as a function of yield deviations from standard curves and DIM deviations around reference dates. Peak milk yield and DIM at peak of individual lactations were computed using Wood's function. Effects of persistency (PM, PF, and PP) on probability of the diseases in current and next lactations were examined through odds ratios from a logistic regression model. Conversely, the effects of diseases on persistencies, peak milk yield, and DIM at peak milk yield were also examined. Increasing PM, PF, and PP tend to reduce the incidence

of mastitis, specifically in late stages of current and next lactation. PM and PP appear to have greater impact on mastitis than PF. No other likelihood of a disease was affected by the increasing persistencies. Post partum reproductive and metabolic diseases often had substantially positive effect on persistencies of both primiparous and multiparous cows ( $p < 0.001$  in Study 1 and  $p < 0.001$  in Study 2). Mastitis in early lactation appeared to increase persistency more often in multiparous cows ( $p < 0.05$  in Study 1 and  $p < 0.005$  in Study 2). Mastitis in late lactation had considerable but negative impact on persistency in both primiparous and multiparous cows ( $p < 0.05$  in Study 1 and  $p < 0.005$  in Study 2). Cows, which developed mastitis in both early and late lactations tended to have lower PM ( $p < 0.05$  in Study 1). Irrespective to the time of occurrence, effect of mastitis on milk, fat and protein yield persistencies was negative. Most of the diseases significantly affected DIM at peak milk yield in multiparous cows ( $p < 0.05$  in Study 1). Reproductive and metabolic disorders tended to delay DIM at peak milk yield while Mastitis in late lactation was associated with early DIM at peak milk yield. Lameness had no phenotypic relationships with shape of the lactation curve. Overall, diseases tend to affect milk, fat, and protein persistencies more strongly than the impact of persistency on likelihood of disease.

**TO**  
**MY BELOVED PARENTS, RANJAN & NEETA**  
**MY DEAR BROTHERS, SANJEEWA, SANJAYA & SAMEERA**  
**&**  
**MY EVERLOVING GRANDMOTHER, BEETA**

## ACKNOWLEDGEMENTS

Thanks to my committee members: Dr. Bennet Cassell, for giving me the opportunity to pursue my master's studies at Virginia Tech and for his guidance throughout this research, Dr. Ronald Pearson and Dr. R. M. Lewis for their valuable comments and suggestions towards the success of this study and Dr. Michael Akers for his help and attempts made to make a very good working environment in the Department of dairy science.

I am grateful for financial support and data provided by Animal Improvement Program Laboratory (AIPL), USDA, Beltsville, MD. A special thank to Dr. John B. Cole (AIPL, USDA) for being generous to share his ideas and information of lactation persistency. I acknowledge the support I received from Dr. Chad Dechow through providing me health data and daily milk weights from the experimental dairy herd at Pennsylvania State University.

I want to thank Dr. (Ms.) Susanne Aref, former director of statistical consultant service unit at Virginia Tech, for her valuable instructions in regression analysis. I highly appreciate all support I received from faculty, staff, graduate students and farm crew in the Department of dairy science during last two years. Moreover, I must be thankful for Dr. Ned Lester and Dr (Ms.) Rosalyn Lester, who have been taking care of me as my host family at Blacksburg.

I am deeply grateful to my parents, brothers, relatives, and friends for their love, encouragement, and help.

Most importantly, I need to thank God for the innumerable blessings my family and I receive every day: without His help I could have done nothing.

## TABLE OF CONTENTS

TITLE .....	i
ABSTRACT .....	ii
DEDICATION .....	iv
ACKNOWLEDGEMENTS .....	v
TABLE OF CONTENTS .....	vi
LIST OF TABLES .....	viii
LIST OF FIGURES .....	x
<b>CHAPTER I</b> .....	1
<b>Introduction</b> .....	2
Objectives .....	3
<b>CHAPTER II</b> .....	4
<b>Literature Review</b> .....	5
Common health disorders in dairy cows .....	5
Selection of dairy cattle for disease resistance .....	10
Persistence .....	17
References .....	27
<b>CHAPTER III</b> .....	33
<b>Manuscript I: Phenotypic relationships of common health disorders in dairy cows to lactation persistency, estimated from daily milk weights</b>	
Abstract .....	34
Introduction .....	35
Materials and Methods .....	36
Results .....	41
Discussion .....	45
Conclusions .....	49
Acknowledgements .....	49



## LIST OF TABLES

<b>Table 1.1</b> Number of lactations (out of total 1212 lactations) and frequency (in parenthesis) of lameness diagnosed at different stages of lactation (Booth et al., 2004) .....	9
<b>Table 1.2</b> Heritability estimates ( $\pm$ SD) for common health disorders in US Holsteins with respect to the analysis in first and all lactations (Zwald et al., 2004) .....	12
<b>Table 1.3</b> Disease frequencies of mastitis (MAST), displaced abomasums (DA), ketosis (KET), milk fever (MKFV), lameness (LAME), Cystic ovaries (CYST) and metritis (MET) in US (USH), Canadian (CH), German (GH) and Danish (DH) Holsteins .....	13
<b>Table 1.4</b> On-farm codes or acronyms used to record the six health disorders in 379 farms located in Northeast, Southeast, South, Midwest, and Great plain areas in USA (Zwald et al., 2004) .....	14
<b>Table 1.5</b> Individual diseases in each composite disease trait (Lyons et al., 1991) .....	15
<b>Table 1.6</b> Equations that are commonly used to depict the lactation curve .....	20
<b>Table 2.1</b> Correlations between 305 d yield and persistency calculated for different $d_0$ in first (FL) and later lactations (LL) .....	56
<b>Table 2.2</b> Incidence frequencies (%) for the diseases in first lactations (FL), later lactations (LL), and across parities (ALL) .....	56
<b>Table 2.3</b> Least Square Means (LSM) of persistency and significance (p-value) of the effect of each disease trait on persistency in first (FL) and later lactations (LL) .....	57
<b>Table 2.4</b> Least Square Means (LSM) of peak yield (kg/d) and significance (p-value) of the effect of each disease trait on peak yield in first (FL) and later lactations (LL) .....	58
<b>Table 2.5</b> Least Square Means (LSM) of DIM at peak and significance (p-value) of the effect of each disease trait on DIM at peak in first (FL) and later lactations (LL) .....	59
<b>Table 2.6</b> Means and phenotypic correlations (r) for 305 d yield (305-Y), peak yield (PY), DIM at peak (DIMP), and persistency in first (FL) and later lactations .....	60

<b>Table 2.7</b> Odds ratios and 95% confidence intervals (CI) for the effect of persistency on probability of disease occurrence in current lactation .....	60
<b>Table 2.8</b> Odds ratios and associated 95% confidence intervals (CI) for the effect of persistency on probability of disease occurrence in next lactation .....	60
<b>Table 3.1</b> Summary statistics for first (FL) and later lactations (LL) .....	80
<b>Table 3.2</b> Lactational Incidence Rates (LIR) for each disease trait in first lactations (FL), later lactations (LL), and all lactations (ALL) .....	80
<b>Table 3.3</b> Coding disease traits for mastitis .....	81
<b>Table 3.4</b> Frequencies for the first incidence of health disorders by month of lactation (across parities) .....	81
<b>Table 3.5</b> Least Square Means (LSM), significance (p-value) for the effect of each disease trait and correlations (r) to disease traits for PM, PF and PP in first lactations (FL) ....	82
<b>Table 3.6</b> Least Square Means (LSM), significance (p-value) for the effect of each disease trait and correlations (r) to disease traits for PM, PF and PP in later lactations (LL) ....	83
<b>Table 3.7</b> Odds ratios and 95% confidence interval (CI) for the effect of the persistency on the likelihood of the diseases in first (FL) and later lactations (LL) .....	84
<b>Table 3.8</b> Odds ratios and associated 95% confidence intervals (CI) related to the effect of each persistency trait on the likelihood of the diseases in next lactation .....	85

## LIST OF FIGURES

<b>Figure 1.1</b> Total number of cows (solid circle) and total milk production (empty circle) in USA by year (Powell and Norman, 2006) .....	5
<b>Figure 1.2</b> Incidence of mastitis during the first 300d of lactation (Hinrichs et al., 2005) .....	6
<b>Figure 1.3</b> Least square means of monthly milk production (kg/d) for multiparous (dashed line) and primiparous (solid line) cows that were not affected (square) and affected (triangle) by LDA (Raizman et al., 2002) .....	8
<b>Figure 1.4</b> Relative emphasis on production (gray), longevity (white) and health and reproduction (black) components in selection indices of countries in August 2003 (Miglior et al., 2005) .....	10
<b>Figure 1.5</b> Typical lactation curve of dairy cows: shaded portion shows the additional yield from greater persistency (Jamrozic et al., 1997) .....	18
<b>Figure 1.6</b> Lactation curves for milk, protein and fat yields of 1 <sup>st</sup> , 2 <sup>nd</sup> , and 3 <sup>rd</sup> lactation Holstein cows (Stanton et al., 1992) .....	19
<b>Figure 1.7</b> A schematic representation of the biology of the mammary gland during pregnancy and 40-week lactation showing parenchyma cell production (◆), secretory cell differentiation (■), cell dying by apoptosis (▲), potentially active cells (○), secretory rate (—) and milk production (—) (Pollot et al., 2000) .....	22
<b>Figure 1.8</b> The standard curve (solid line) vs. a lactation curve of an individual cow (dot line) .....	26
<b>Figure 2.1</b> Standard lactations curves <sup>1</sup> for first (FL) and later lactations (LL) developed from daily milk yields in two experimental herds .....	53
<b>Figure 2.2</b> Frequencies of persistency classes in first (FL) and later lactations (LL) ...	53
<b>Figure 2.3a</b> Comparison of first (FL) <sup>1</sup> and later lactations (LL) <sup>2</sup> of high persistency <sup>3</sup> with corresponding standard lactation curves <sup>4</sup> .....	54

<b>Figure 2.3b</b> Comparison of first (FL) <sup>1</sup> and later lactations (LL) <sup>2</sup> of average persistency <sup>3</sup> with corresponding standard lactation curves <sup>4</sup> .....	54
<b>Figure 2.3c</b> Comparison of first (FL) <sup>1</sup> and later lactations (LL) <sup>2</sup> of low persistency <sup>3</sup> with corresponding standard lactation curves <sup>4</sup> .....	55
<b>Figure 2.4</b> Distribution of treatment incidence for mastitis over 305 d lactation .....	55
<b>Figure 3.1</b> Distribution of standardized milk (PM), fat (PF) and protein (PP) persistencies (frequencies (%) in each persistency classes were estimated across parities) .....	78
<b>Figure 3.2</b> Frequency distribution of MAST and MAST2 across PM, PF, and PP classes	79

# **CHAPTER I**

## **Introduction**

## INTRODUCTION

It is generally accepted that undesirable genetic relationships exist between production and health (Carlen et al., 2004). Disease resistance in the global dairy cattle population has been adversely affected because the majority of selection emphasis during last 50 yr focused on higher milk production per cow (Powell and Norman, 2006). Zwald et al. (2004a) identified mastitis, lameness, displaced abomasums, ketosis, metritis (including retained placenta), and cystic ovaries to be the most common health disorders affecting the profitability of dairy herds through involuntary culling, veterinary cost, added labor and lost of milk sales. These diseases cost \$151 per case for ketosis to \$312 per case for displaced abomasum (Zwald et al., 2004a). In spite of relatively low heritabilities (Cassell, 2001) the economic importance justifies the inclusion of disease traits in breeding goal for dairy cattle selection.

Until 1994, when genetic evaluation for somatic cell score (SCS) and productive life (PL) were introduced, selection indices for US dairy cattle included only yield traits. A positive genetic relationship between SCS and clinical mastitis suggests selection for lower SCS may reduce mastitis incidence in dairy cows. Selection for increased PL could reduce diseases because diseases are primary causes of premature culling (Rogers et al., 1999). Since 1994, selection for health has proceeded primarily through these two indirect indicators that are routinely measured within the DHI system (Zwald et al., 2004b). Direct selection for disease traits is yet to be established in USA as health data recording practices are not standardized across farms and mechanisms for routine retrieval of such data do not exist (Zwald et al., 2004b).

Besides SCS and PL, many investigators (Dekkers et al., 1998, Jakobsen et al., 2003, and Cole and VanRaden, 2006) have suggested favorable relationships between lactation persistency and health disorders in dairy cows. Lactation persistency is defined as the ability of a cow to maintain milk production at a high level after the peak yield (Jamrozik et al., 1998). Persistent lactations are associated with lower peak yields at later days in milk (Ferris et al., 1985) indicating less severe metabolic stress in early stages of the lactations. Metabolic stress in early lactation tends to suppress physiological processes that maintain general health and reproduction (Collard et al., 2000). This suggests that cows with good persistency may have fewer health and reproductive

problems than cows that are less persistent. Thus, including persistency in breeding goals could help to improve the disease resistance if genetic correlations are favorable and sufficiently strong. Studies on correlations between diseases and milk yield are abundant in the literature but investigations into the relationships between diseases and other traits are lacking (Muir et al., 2004).

Lack of clear consensus on how best to define persistency is a key issue in genetic evaluation for lactation persistency (Grossman et al., 1999 and Cole and VanRaden, 2006). Many available persistency measures are negatively correlated with 305 d yield (Dekkers et al., 1998, Jakobsen et al., 2003, and Muir et al., 2004) suggesting selection for higher persistency would occur at the expense of total yield. A persistency measure that is independent of yield will therefore allow more efficient selection for total yield and persistency simultaneously (Muir et al., 2004). Cole and VanRaden (2006) suggested a method to estimate persistency, phenotypically uncorrelated with yield, using a function of test day (TD) yield deviations from a standard curve and TD DIM deviations around a reference date.

## **Objectives**

The general objective of this research was to investigate phenotypic relationships between lactation persistency and common health disorders in dairy cows.

The specific objectives were:

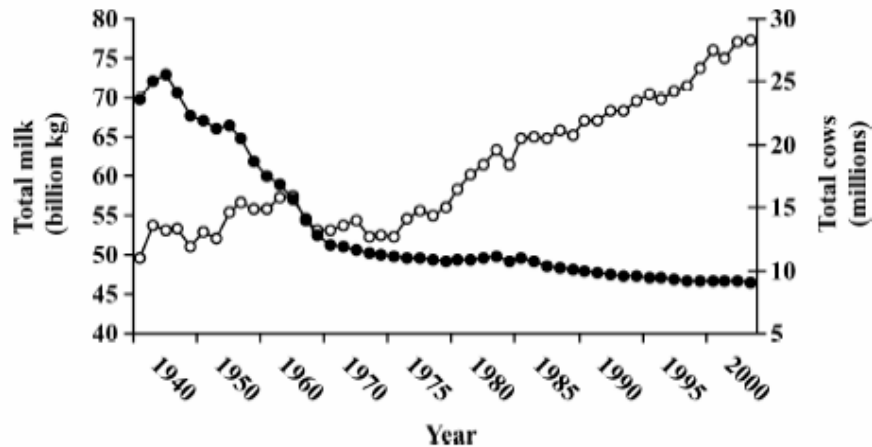
- i. to compare the use of TD yields in calculating persistency that is uncorrelated with yield, relative to more frequent recording of daily milk weights
- ii. to study the relationships between persistency and diseases using two types of data: producer recorded health data from commercial dairy farms and the data from experimental dairy farms operating under relatively intensive health management programs

**CHAPTER II**  
**Literature Review**

## LITERATURE REVIEW

### Common health disorders in dairy cows

Since 1940s, selection emphasis on total yield per cow has been very successful (Powell and Norman, 2006). The data in Figure 1.1 can be used to estimate that average milk production per cow has increased more than ten times (from about 700 kg in 1940 to approximately 8500 kg in 2000) in USA during last half century.

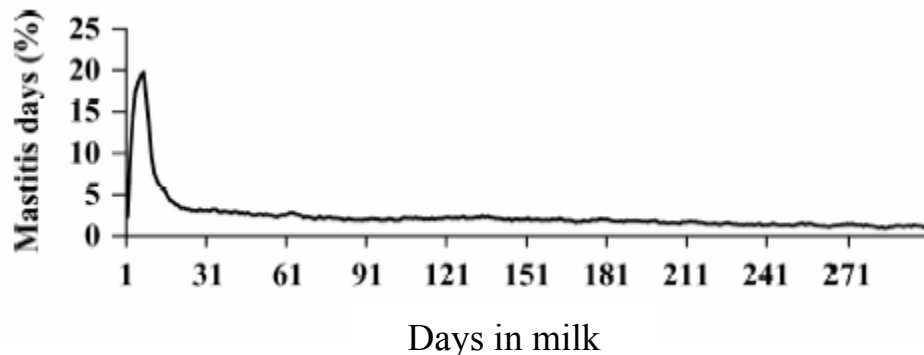


**Figure 1.1** Total number of cows (solid circle) and total milk production (empty circle) in USA by year (Powell and Norman, 2006)

But, it is generally accepted that undesirable genetic relationships exist between production and health disorders (Carlen et al., 2004). Increased incidence rates for many diseases in global dairy cattle population suggest that present day cows are highly susceptible to health disorders (Zwald et al., 2004). There are two major reasons why higher susceptibility to diseases is not desirable. First, there are ethical concerns related to animal welfare and consumer interest (Jakobsen et al., 2003 and Van Dorp et al., 1998). Second, diseases can dramatically affect the profitability of dairy production through the cost of veterinary treatment and added labor, and loss of revenue through involuntary culling and discarded milk (Van Dorp et al., 1998, Zwald et al., 2004, and Harder et al., 2006). Zwald et al. (2004) listed mastitis, lameness, displaced abomasums,

ketosis, metritis (including retained placenta), and cystic ovaries as common health disorders of US Holsteins having a significant impact on profitability of dairy operation.

**Mastitis:** Mastitis is the most costly disease affecting dairy producers. In USA, mastitis is estimated to cost dairy producers \$1.2 to \$1.7 billion per year (Shim et al., 2004). It is generally agreed that 70 to 80% of the estimated annual cost for mastitis per cow is associated with reduced milk production due to non-symptomatic, sub-clinical mastitis (Gill et al., 1990). When clinical mastitis occurs, additional costs result from discard of abnormal milk, drugs, veterinary services and risk of contamination in milk sold. Each case of clinical mastitis has been estimated to cost between \$100 and \$200 per cow within the lactation (Wilsen et al., 2004). Zwald et al. (2004) presented frequencies of mastitis ranging from 5% to 60% across 379 US Holstein herds with mean frequency of 20%. As Figure 1.2 shows, most cases of mastitis tend to occur during the first 30 d of lactation (Hinrichs et al., 2005). Moreover Zwald et al. (2006) suggested that it is more appropriate to consider mastitis with respect to at least two segments of lactation (e.g. early and late) as early lactation mastitis is likely to be lowly correlated with mastitis in mid and late lactation.



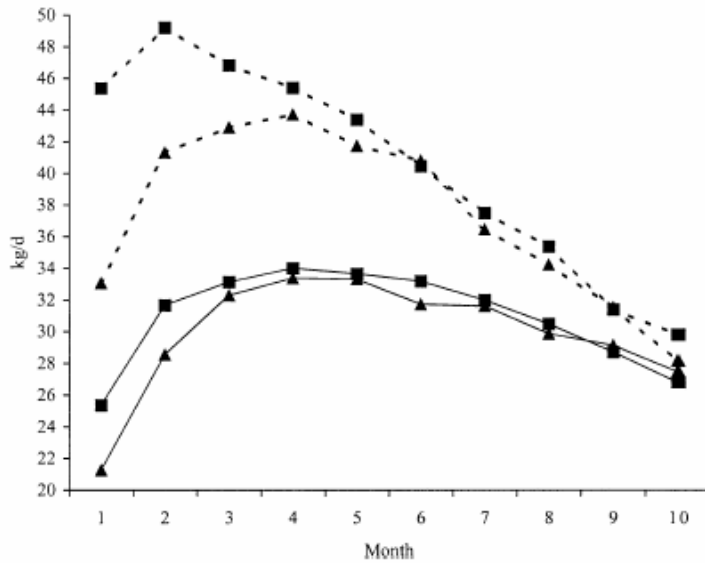
**Figure 1.2** Incidence of mastitis during the first 300d of lactation (Hinrichs et al., 2005)

Many dairy producing countries have included Somatic Cell Scores (SCS) with a negative weight in selection indexes to avoid a further increase of mastitis susceptibility of dairy cows (Miglior et al., 2006). A standard procedure for diagnosis and recording clinical mastitis is available in Scandinavian countries (Carlen et al., 2006). Such a standard procedure is yet to be established in US dairy herds (Zwald et al., 2004b).

However, Zwald et al. (2006) concluded that “farmer-recorded clinical mastitis data can make a valuable contribution to genetic selection programs, but additional systems for gathering and storing this information must be developed, and more extensive data recording in progeny test herds should be encouraged”.

**Metritis:** Inflammatory uterine disease or metritis frequently occurs soon after calving and may severely compromise reproductive efficiency. The incidence rates of metritis ranges from 7.8 to 51.3% in US dairy cows (Lewis, 1997). This wide range in incidence rates, in part, can be attributed to inconsistency in diagnosis and defining the disease. A lack of easily identifiable signs of metritis may also result in cases going unnoticed (Lewis, 1997 and Urton et al., 2005). A path analysis (Correa et al., 1993) to metritis indicated that dystocia, retained placenta, and metabolic stress increased the likelihood that a cow would develop metritis. Lewis (1997) reported that the culling rate during lactations with metritis was 26.6% but was 20.5% in lactations without metritis and the cost to producers for each lactating cow with metritis was \$106.

**Displaced abomasum:** Displaced abomasum is a condition primarily of dairy cows in which the abomasum becomes enlarged with fluid and gas and is mechanically displaced from its normal position to left or right side (Raizman et al., 2002). Shaver (1997) noted that “90% of all abomasal displacements are left sided” and frequency of left displaced abomasums (LDA) ranged from 0 to 21.7% (across 71 herds that had 5,742 cows). On the other hand, based on the treatment incidences from 379 US Holstein herds, Zwald et al. (2004a) estimated the mean frequency of 3% for metritis. Periparturiant low feed consumption is a risk factor for LDA through reduced rumen fill and accumulation of gas in abomasums (Shaver, 1997). Dairy cows are more likely to develop LDA if they have recently experienced one or more periparturiant disorders including dystocia, stillbirth, twins, retained placenta, metritis, ketosis or milk fever (Raizman et al., 2002). A survival analysis done by Raizman et al. (2002) reported that 15% and 52% of LDA cows died and were sold respectively. They also observed that the negative effect of LDA on milk production is only apparent during the first 4 mo of lactation and afterwards the affected cows reached the production of their non-affected herd-mates (Figure.1.3). Zwald et al. (2004) reported that LDA cost \$ 312 per case.



**Figure 1.3** Least Square means of monthly milk production (kg/d) for multiparous (dashed line) and primiparous (solid line) cows that were not affected (square) and affected (triangle) by LDA (Raizman et al., 2002).

**Lameness:** With respect to animal welfare considerations, lameness is the most common cause of distress in dairy cattle (Booth et al, 2004). Nutrition and feeding, housing and environment, concurrent diseases, genetic influence, and management factors all predispose a cow to foot and leg problems. The greater incidences (90%) of lameness involve the foot and claw (Sogstad et al., 2005) and of these about 90 percent involve rear feet. The most frequent causes of lameness are: laminitis, sole ulcers, foot wart, abscess, foot rot, digital dermatitis, white line disease, interdigital hyperplasia, and over-grown claw (Warnick et al., 2001). The nature of lameness data can be binary (Koenig et al., 2005) or discrete, when lameness is recorded using a lameness-scoring sheet. Annual incidence rate of lameness ranges from 4% to 55%. As Table.1.1 shows, intensity of lameness is generally high in early lactation (Booth et al., 2004). Based on results from a study of two dairy herds in New York, Booth et al. (2004) concluded that culling rates due to lameness could vary from 15.6 to 35.4 depending on parity, season of calving and stage of lactation during which lameness occurs.

**Table 1.1** Number of lactations (out of total 1212 lactations) and frequency (in parenthesis) of lameness diagnosed at different stages of lactation (Booth et al., 2004).

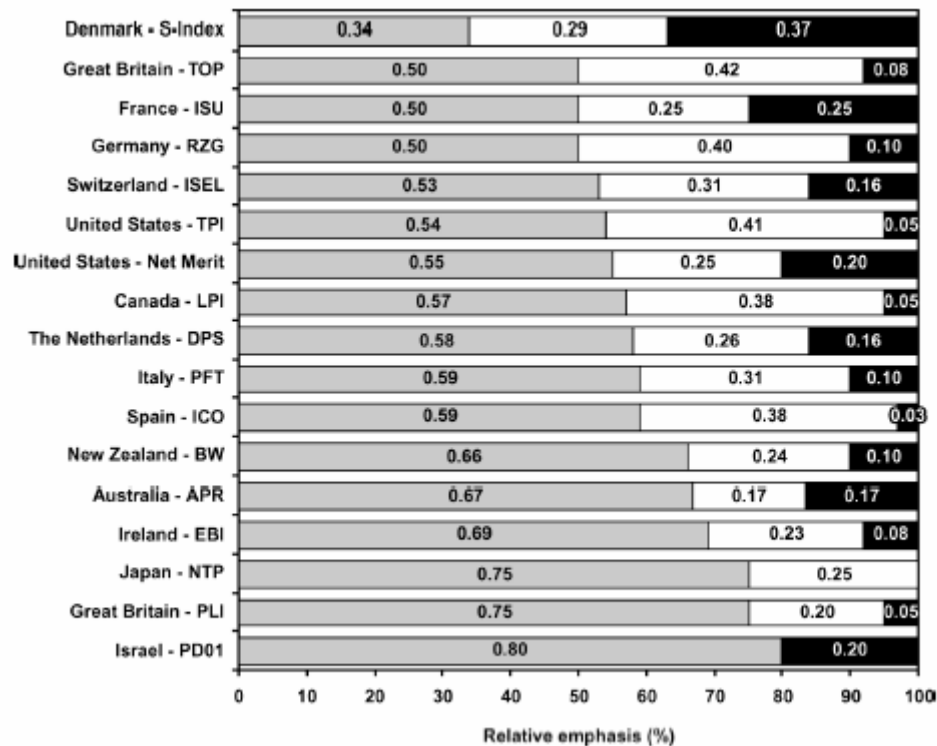
Diagnosis	Days in milk				Total
	≤60	61 to 120	121 to 240	>240	
	No. (%)				
Foot rot	71 (58.7)	18 (14.9)	21 (17.4)	11 (9.1)	121
Foot warts	70 (26.7)	81 (30.9)	79 (30.2)	32 (12.2)	262
Sole ulcer	64 (26.4)	77 (31.8)	76 (31.4)	25 (10.3)	242
Multiple diag. <sup>1</sup>	5 (50.0)	0 (0.0)	5 (50.0)	0 (0.0)	10
Other lameness	273 (47.3)	118 (20.5)	111 (19.2)	75 (13.0)	577
All lameness	488 (39.9)	294 (24.3)	292 (24.1)	143 (11.8)	1212

**Ketosis:** Ketosis is disease related to the high demand of glucose in mammary gland and the inability of cows to meet the glucose demand through appetite (Baird et al., 1982). As a consequence, adipose tissue is mobilized, and free fatty acids are oxidized in the liver, which results in ketone body production. Over production and accumulation of ketone bodies are toxic (Green et al., 1999). In general ketosis is diagnosed through the detection of ketone bodies in milk, urine and breath besides reduced feed intake and milk production. A loss of milk production of 1.0 to 1.4 kg/d is associated with ketosis (Green et al., 1999). On average, about one tenth of cows are likely to develop ketosis (Green et al., 1999, Zwald et al., 2004, Wilson et al., 2004). Ketosis is more highly correlated with LDA than other perparturiant diseases and increases predisposition to LDA (Shaver, 1997). Ketosis in US dairy cattle costs \$151 per case (Zwald et al., 2004).

**Ovarian follicular cysts:** In a number of mammalian species, ovarian follicular cysts have been identified as anovulatory follicular structures. Cows are infertile as long as the condition persists. From 5.6 to 18.8% of dairy cows develop the condition; the mean percentage probably ranges between 10 and 13%. Thus, the condition is estimated to affect at least one million dairy cows in the US annually. Cows with ovarian follicular cysts have extended calving intervals from 22 to 64 d; the mean interval ranges from 40 to 50 d. The mean interval from diagnosis to conception is 50 d. Thus, ovarian follicular cysts are an important cause of infertility and economic loss for dairy farm enterprises (Garverick, 1997)

## Selection of dairy cattle for disease resistance

The involvement of genetic factors in the incidence of the health disorders is suspected, since the incidence rates are higher among relatives of affected individuals than in the general population (Lin et al., 1989). The presence of an underlying genetic component suggests the potential for improvement in disease resistance through selection (Jakobsen et al., 2003). The greater economic importance of health disorders in dairy cows may adequately justify inclusion of disease traits in breeding goals, despite their relatively low heritabilities (Shook, 1989).



**Figure 1.4** Relative emphasis on production (gray), longevity (white) and health and reproduction (black) components in selection indices of countries in August 2003 (Miglior et al., 2005).

Selection goals in many countries are initiating positive selection pressure on disease resistance by incorporating correlated traits such as conformation traits, SCS, and longevity in selection indices (Jakobsen et al., 2003). Miglior et al. (2005) investigated the selection indices in Holstein cattle of various countries. They presented the relative

emphasis on health and reproduction components in selection indices of 15 dairy producing countries (Figure 1.4). The health and reproduction component in Figure 1.4 included traits such as udder health, milking speed and calving ease. In most of the indices SCS (an indicator trait for mastitis resistant) was the only trait contributing to udder health. Only the Dutch udder health index included a trait for direct selection of clinical mastitis (Miglior et al., 2005).

Greater emphasis on non-production traits reflects the industry's desire for functional dairy cattle. Shook (2006) noted that until 1994, when SCS and productive life were introduced, selection indexes for US dairy cattle included only yield traits. Composite type indexes for udder, feet and legs, and body size were added in 2000 and daughter pregnancy rate and calving ease were included in 2003. Low heritability of disease traits, lack of industry-wide standard for recording and accumulating field data for disease (Shook, 1989), and lack of clear consensus on how best to analyze the subjectively measured disease events (Carlen et al., 2006) have been identified as a major challenge in direct selection of dairy cattle for disease resistance.

**Heritability of disease traits:** In general, traits related to fertility, fitness, health and survival have low heritabilities of less than 0.15. For lowly heritable traits, an animal's performance is much less useful in identifying the individuals with the best genes for the trait (Cassell, 2001). Uribe et al. (1995) estimated heritability of common health disorders, namely mastitis, metritis, cystic ovaries, milk fever, ketosis, displaced abomasum and leg and foot problems in Holstein cows, and concluded there was relatively little genetic variation for the diseases. However, they also suggested that the estimated genetic variation may be sufficient for most of the diseases to be potentially useful in breeding programs. Moreover, Zwald et al (2004) estimated heritabilities of 6 common health disorders (Table. 1.2) using producer recorded health data from 379 commercial dairy farms located in Northeast, Southeast, South, Midwest, and Great plains in USA. They also concluded that the estimated heritabilities were sufficient to incorporate those disease traits, with appropriate economic weights into a selection index. In spite of relatively low heritabilities, the economic importance seems to justify the inclusion of disease traits in breeding goal (Shook, 1989).

**Table 1.2** Heritability estimates ( $\pm$ SD) for common health disorders in US Holsteins with respect to the analysis in first and all lactations (Zwald et al., 2004).

Disorder	Analysis	Heritability
Displaced abomasum	1st lactation	0.18 $\pm$ 0.010
	All lactations	0.15 $\pm$ 0.006
Ketosis	1st lactation	0.11 $\pm$ 0.007
	All lactations	0.06 $\pm$ 0.005
Mastitis	1st lactation	0.10 $\pm$ 0.003
	All lactations	0.09 $\pm$ 0.003
Lameness	1st lactation	0.07 $\pm$ 0.003
	All lactations	0.06 $\pm$ 0.003
Cystic ovaries	1st lactation	0.08 $\pm$ 0.005
	All lactations	0.05 $\pm$ 0.005
Metritis	1st lactation	0.08 $\pm$ 0.004
	All lactations	0.07 $\pm$ 0.003
Any disease within 50 d postpartum	1st lactation	0.12 $\pm$ 0.005
	All lactations	0.10 $\pm$ 0.004

**Health data and definition of disease traits:** Table 1.3 presents some estimates of disease frequencies in Holstein cows available in literature. These estimates indicate that the disease frequencies vary widely from a study to another. Hence, it is difficult to compare disease frequencies across different studies as demography, data source (e.g. large scale or small scale dairy farm), disease diagnosis, data recording methods (e.g. computer software), and disease definitions widely vary from one study to another (Harder et al. 2006). For instance, the disease frequencies (Table 1.4) reported by Zwald et al. (2004) were based on producer recorded health data from 379 commercial dairy farms whereas Wilson et al. (2004) reported frequencies of disease incidences from only two large scale dairy farms in New York, where farm crew and veterinarians were provided with written definitions of diseases. The disease frequencies pertaining to lameness differ across studies since they used widely varying combinations of several leg and foot disorders. For instance, Sander-Nielsen et al. (1996) defined lameness considering only the incidences related to foot such as foot rot, foot abscesses, laminitis, sole ulcers, sole dermatitis, and claw trimming by veterinarians, whereas Collard et al. (2000) defined lameness considering also leg disorders such as hock problems, and inflamed thigh in addition to the foot disorders. Similarly, some investigators tend to define metritis considering incidences of both uterine infections and retain placenta while some account only for the infections.

**Table 1.3** Disease frequencies (%) of mastitis (MAST), displaced abomasums (DA), ketosis (KET), milk fever (MKFV), lameness (LAME), Cystic ovaries (CYST) and metritis (MET) in US (USH), Canadian (CH), German (GH) and Danish (DH) Holsteins.

Disease	Zwald et al. (2004)	Lyons et al (1991)	Wilson et al. (2004)		Domecq et al. (1997)	Grohn et al. (1995)	Collard et al. (2000)	Bigras-Poulin, (1990)	Harder et al (2006)	Nielsen et al. (1996)
	USH						CH		GH	DH
	All <sup>1</sup>	All	First <sup>2</sup>	Later <sup>3</sup>	Later	All	All	All	First	All
MAST	20	48.7	19.6	28.8	--	5.4	35	24.2	--	26.3
DA	3	2.2	2.2	2.9	0.5	--	--	--	--	--
KET	10	6.6	12.3	12.6	--	6		3.3	--	--
MKFV	--	9.4	--	--	--	3.8	--	5.6	--	--
LAME	10	19.0	33	31	2.8		35		11.4	6.2
CYST	8	5.8	--	--	--	6.8	--	5	--	--
METR	21	14.7	13.9	4.4	19.9	2.3	--	10.2	--	--

<sup>1</sup>across all lactations, <sup>2</sup>first lactation, <sup>3</sup>second and later lactations

Heterogeneity of codes and acronyms used to record health events also tend to cause disease definitions to be different from study to study (Kelton et al., 1998). Zwald et al. (2004) noted that besides the variability of the health codes among dairy management software (e.g. PCDART and DAIRY COMP-305), many herds developed their own user-defined codes or acronyms for various diseases. Table 1.4 presents some on-farm codes or acronyms used to record six health disorders in the study performed by Zwald et al. (2004). This inconsistency in disease diagnosis and recording may reduce the reliability of health data. The lack of reliability has limited the inclusion of health traits in breeding goals, although many countries have realized the economic importance of selection for disease resistance (Koenig et al., 2005). Zwald et al. (2004) and Dechow et al. (2004) noted that there was no standard program for recording, centralizing, storing, or analyzing information about disease traits in US dairy cattle. However, Carlen et al. (2006) noted that although some Scandinavian countries record veterinary treated cases of clinical mastitis (after the decision made by the farmer), still it is important to choose the best approach to analyze these data to get accurate information for genetic evaluation.

**Table 1.4** On-farm codes or acronyms used to record six health disorders in 379 farms located in Northeast, Southeast, South, Midwest, and Great plain areas in USA (Zwald et al., 2004).

Displaced abomasum	Ketosis	Mastitis	Lameness	Cystic ovaries	Metritis
DA	KETOSIS	MAST	ABCS	CYST	MET/RP
D.A.	KETOTIC	RF	ABSS	CYSTG	MET
LDA	KET	LF	HROT	CYSTO	METR
RDA	KETO	RR	HFROT	CYSTIC	RP
L-DA	KETOS	LR	LAMINIT	RCYST	RETAINP
R-DA	KET1	MLFQ	LAME	LCYST	RETP
DAS	KET2	MLRQ	WRAP	CYSTRO	INFU
DALF	KET3	MRRQ	LAMI	CYSTLO	INF
DART	KETI	MRFQ	LIMP		MTRI
DAR	KETR	MLF	SOEFT		RETN
DAL	KETS	MLR	ABCSRR		RPL
	KETH	MRR	ABCSLR		RPIN
	KETD	MRF	FOOT		RPRE
	KETP	RFMT	FEET		UCND
	METB <sup>1</sup>	LFMT			RTPL
		LRMT			UINF
		RRMT			PYOM
		M2TIT			UTCN
		MASTALL			RE-PLA
		MAST2Q			
		MAST3Q			

<sup>1</sup>Used as an acronym for ketosis in herds with the PCDART software only.

**The best approach to analyze health data in genetic evaluations:** Binary nature of health data challenges choice of the best approach to analyze them in genetic evaluation for disease resistance (Koenig et al., 2005). The widely used method for health data analyses is a linear model (LM), in which a disease is defined as a binary trait distinguishing cows with at least one incidence of the disease (1) and cows without incidences (0) (Carlen et al., 2004). This trait definition utilizes only one disease incidence (mostly the first) in a defined period (mostly whole lactation). By excluding the incidences other than the first, some of the available information, especially the timing of the disease, is lost in the LM approach (Carlen et al., 2006). Timing of the incidences is important with respect to the diseases that can occur through out the lactation. Therefore it is more appropriate to consider diseases like mastitis pertaining to at least two stages of lactation (e.g. early and late lactation or before and after peak yield). Another potential disadvantage of LM methodology is that the assumption of “normally distributed observations” is not fulfilled (Van Dorp et al., 1998), causing linear models to be less suitable for estimation of genetic parameters of disease traits.

The other approach is using a threshold model (TM), which takes the binary character into account and that would be theoretically more appropriate (Van Dorp et al., 1998, Henrichs et al., 2005, Carlen et al., 2006). Based on the amount of data used, two

types of TM can be identified: cross-sectional TM using only a single incidence from each animal/lactation (Similar to the LM) and multivariate TM, considering multiple cases and time aspects of a disease (Heringstad et al., 2004). However, Mantysaari et al. (1991) noted that “if the disease frequency is more than 10% and the difference in frequency between animal groups (e.g parity groups and herds) is less than 3-fold, the estimates for genetic correlations from a linear model will be very similar to the estimates from a threshold model”. Therefore, it seems to be more appropriate to use LM for the diseases that tend to occur more frequently. Some investigators have used LM for analyzing diseases with lower incidence (e. g. milk fever) in composite disease traits as shown in Table 1.5.

**Table 1.5** Individual diseases in each composite disease trait (Lyons et al., 1991)

<b>Individual trait</b>	<b>Composite trait</b>
Abortion	Reproductive
Cystic ovaries	
Retained placenta	
Uterus infections	
Other reproductive problems	
Mastitis	Mammary
Udder injury	
Other udder problems	
Milk fever	Digestive
Ketosis	
Displaced abomasum	
Other digestive problems	
Trimmed feet	Locomotive
Leg problems	
Foot problems	
Crampy	Respiratory
Other locomotive problems	
Pneumonia	
Other respiratory problems	

Moreover, Carlen et al., 2006 compared LM, TM and survival analysis (SA) to estimated genetic parameters and found little difference among these three approaches.

**Alternative approaches to improve disease resistant in dairy cattle:** When direct selection for disease resistance is difficult, industry has to look for other measures that may be useful to improve disease resistance or at least moderate the undesirable response of disease that occurs with intense selection for yield. In response to concerns about fertility, longevity, and disease susceptibility in Holstein cattle, some producers have looked at crossbreeding to reduce disease susceptibility (Zwald et al, 2004). But, the investigations on this approach seem to be at very initial stage.

Some investigators suggested that using genetic markers associated with disease traits in dairy cattle may be useful to evaluate sires for disease resistant. Completing the bovine genome sequence will be very useful for this approach. However, identification of genetic markers specific to a particular disease will not be an easy task as each disease is controlled by many loci in the genome (Shook, 1989). Lucy (2005) noted that “finding genes in the genome is difficult and scanning billions of base pairs of DNA is an imperfect task. At the present time, our capacity to generate information is great but our capacity to understand the information is small. The long-term prognosis for genome science is good but advances will take time. Traditional methods of genetic selection in dairy cattle will be used for the foreseeable future.”

Some possible traits that could be useful in improving disease resistance in dairy cows are measures of longevity such as productive life (PL), and measures of physical characteristics such as body weight, body condition score (BCS) or linear type traits that reflect body structure (Rogers et al., 1999). Selection for increased PL could reduce diseases because diseases contribute to premature culling. Selection for increased body condition or less change in body condition during the lactation may reduce metabolic diseases in dairy cattle. Rogers et al. (1999) investigated the genetic relationships of PL to diseases other than mastitis in Holstein cows. They found a favorable genetic correlation (ranged from 0.29 to 0.51) between PL and disease resistance, suggesting that selection for increased PL may reduce disease incidence. Dechow et al. (2004) estimated genetic correlations of BCS and dairy form to a composite of all diseases in US Holsteins to be -0.79 and 0.85 respectively. These results suggest that selection for higher body condition or lower dairy form with continued selection for yield may slow deterioration in cow health. However, Dechow et al. (2004) also noted “it is not clear that genetic

evaluation for body condition would provide valuable genetic information beyond current dairy form evaluation”.

Selection for traits correlated with disease resistant seems to ultimately improve genetic changes towards less severe negative energy balance or reduced metabolic stress. For example, selection for increased body condition will indirectly select cows for less severe negative energy balance as severe negative energy balance results in a considerable loss of body reserves. Moreover, Dekkers et al. (1998) suggested that persistent cows might lose less body weight indicating a favorable relationship between persistency and reduced negative energy balance.

## **Persistency**

### **Relationships among lactation persistency, metabolic stress and disease resistance:**

Success in selection for higher total yield per cow has led to a significant increase in milk production in early lactation. On the other hand, dietary intake is unable to meet the demands of high milk production in early lactation and consequently cows enter a period of metabolic stress (Harder et al., 2006). Metabolic stress can be defined as “the amount of metabolic load which cannot be sustained, such that some energetic process, including those that maintain general health and reproduction, must be down regulated” (Collard et al., 2000). The high producing cows that undergo a higher peak yield in early lactation are therefore more likely to be susceptible to health and reproductive disorders (Dekkers et al., 1997).

Ferris et al. (1985) and Muir et al. (2004) found persistent lactations tend to be correlated with low peak yields and later time to peak. Moreover, Ferris et al. (1985) mentioned that both later DIM at peak and lower peak yield are indications of less severe negative energy balance. It may be hypothesized that cows having more persistent lactations may be less liable to health disorders as they undergo less metabolic stress in early lactation. Thus, a genetic change towards a persistent lactation curve could be used as a means to lower the disease susceptibility in dairy cows. Relationships of diseases to production traits are abundant in literature, but the investigations into the relationships of diseases to other traits are lacking (Muir et al., 2004).

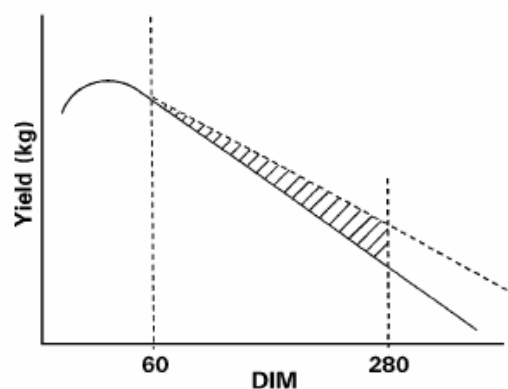
**Definition of persistency:** A key issue in genetic evaluation for persistency is trait definition and there is no clear consensus on the best way of measuring persistency (Cole and VanRaden, 2006). However, Dekkers et al. (1998) suggested the following criteria in selecting a better measure of persistency.

- i. The measure of persistency must be uncorrelated with 305 d yield, because the antagonistic relationship between persistency and 305 d yield
- ii. The measure of persistency must have substantial genetic variance
- iii. The measure of persistency must explain a large proportion of genetic variability in the factors that contribute to economic importance of persistency (i. e. cost of feed , health, and reproduction).

Grossman et al (1999) categorized persistency measures in literature into three groups:

1. Measures expressed as a ratio (or a rate) of yields,
2. Measures derived from variation of test day yields, and
3. Measures constructed of parameter estimates from mathematical models of lactation curves.

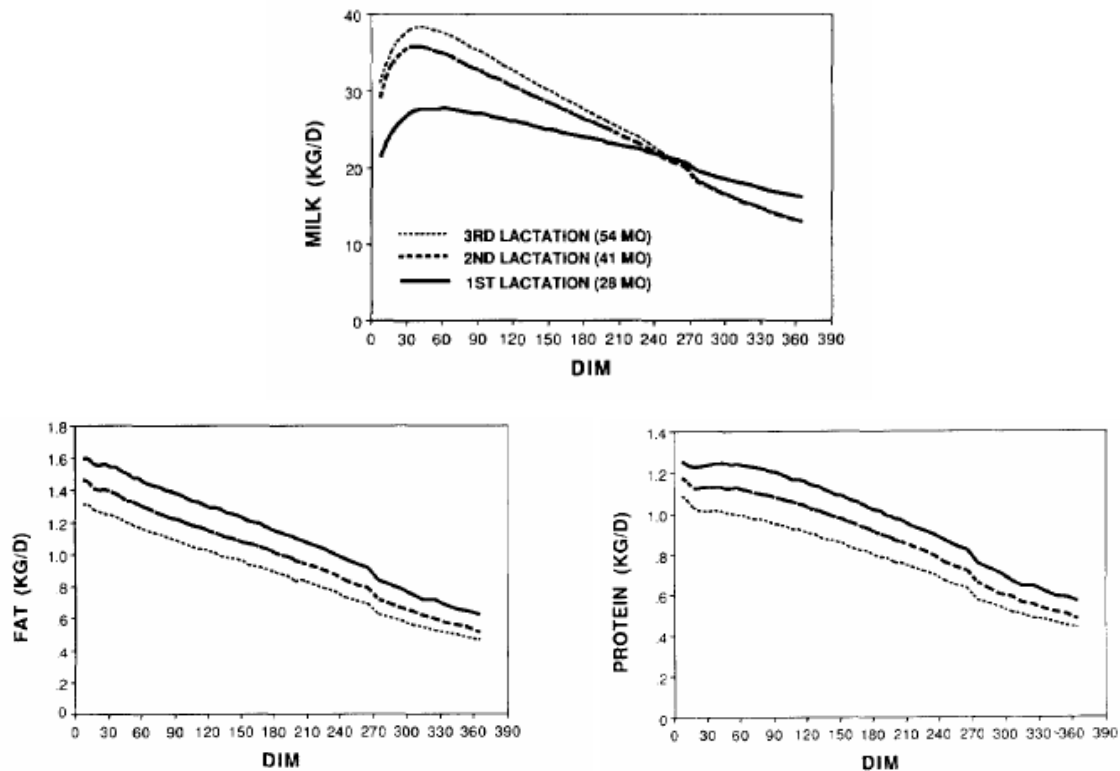
Jamrozik et al. (1997) compared 3 measures of persistency for milk, fat and protein yields using a random regression models. The first definition, D1, was the yield, gained or lost, from 60 to 280 DIM relative to a lactation curve of an animal with average persistency (shaded area of figure 1.5).



**Figure 1.5** Typical lactation curve of dairy cows: shaded portion shows the additional yield from greater persistency (Jamrozik et al., 1997).

The second definition, D2, was based on the ratio of partial lactation yield from d 201 to 305 divided by partial lactation yield from d 1 to 100. The third definition, D3, was based on the ratio of partial lactation yield from 201 to 305 DIM divided by total 305-d yield.

**Lactation curve and persistency:** A typical lactation curve depicts a cow's milk yield distribution after colostrum to drying-off. The distribution of protein and fat yield can also be described using a lactation curve. Lactation curves reflect peak production level, time of lactation at peak production, and persistency that usually refers to the rate of decline in yield after peak. The classic shape is a rapid increase of milk yield after calving to a peak a few weeks later followed by a gradual decline until the cow is dried off. Protein yield tends to reach peak level at earlier days in milk (DIM). Fat yield does not "peak", it only declines from initial level of production (Figure 1.6).



**Figure 1.6** Lactation curves for milk, protein and fat yields of 1<sup>st</sup>, 2<sup>nd</sup>, and 3<sup>rd</sup> lactation Holstein cows (Stanton et al., 1992).

Shape of the lactation curve is affected by several factors such as body weight, age of cow, gestation, dry period, rations, season, temperature and humidity (Togashi and Lin, 2004). However, for milk, fat, and protein, the effect of parity has been shown to be significant on the shape of lactation curve as primiparous cows have lower peak yield, delayed time to peak and more persistent persistency yield than multiparous cows (Stanton et al., 1992). Moreover, lactation curves of multiparous cows in different parities are similar except total yield can be slightly high in third and greater lactations (Scott et al. 1996). Jamrozic et al. (1997) found that the correlations among persistency of milk, fat and protein yields ranged from 0.70 to 0.90 and suggested, “Persistency has a similar meaning for milk, fat, and protein yields”.

**Lactation curve equations and persistency:** Lactation curve equations are helpful to depict the lactation curve when the milk yields through out the lactation are lacking (e.g. TD yields). Using the parameters of these equations peak yield, time to peak and persistency of lactation can be predicted. Lactation curve equations can be categorized into empirical (i.e. equations by Gaines, Wood, and Wilmink ) and mechanistic (i.e. equation by Dijkstra) classes. Four equations, which are widely used to depict lactation curve are presented in Table 1.6.

**Table 1.6** Equations that are commonly used to depict the lactation curve.

Equation	Functional form <sup>1</sup>
Gaines	$Y = ae^{-bt}$
Wood	$Y = at^b e^{-ct}$
Wilmink	$Y = a + bt + c \exp^{-0.05t}$
Dijkstra	$Y = a \exp[b(1 - e^{-ct})/c - dt]$

<sup>1</sup>Y is milk yield (kg/d), t is time of lactation (days), and a, b, c, d, g, h (all>0) are parameters that define the scale and shape of the curve

In 1927, Gaines developed a simple two-parameter (a and b) model of exponential decay (Val-Arreola et al., 2004). The model does not represent a rise to peak yield after calving. The parameter “b” in Gaines equation describes rate of decline in milk yield as lactation progresses.

Wood (1967) proposed the gamma equation, which consists of 3 parameters and accounts for rise to peak yield. The “a” parameter in Wood’s equation is a scaling factor estimating production at time zero, the “b” parameter is rate of ascent to peak, and “c” is rate of descent after peak. Moreover, Wood’s equation offers relatively simple functions;  $a[b/c]^c e^{-b}$  and  $[b/c]$  to calculate peak yield and time to peak yield respectively (Ferris et al, 1985).

In Wilmink’s equation, “a” is regression coefficient related to the maximum daily level of production, “b” is a regression coefficient related to production decrease after peak yield, and “c” is a regression coefficient related to production increase towards peak, and the factor 0.05 is related to the approximate DIM when peak milk yield occurs. Muit et al. (2004) used parameter “b” in Wilmink’s function to measure persistency in Canadian Holstein cows. Estimated heritability for milk yield persistency and its genetic correlation to total yield were 0.18 and -0.12 respectively. “b” parameter in Gain’s and Wood’s equations also describe lactation persistency.

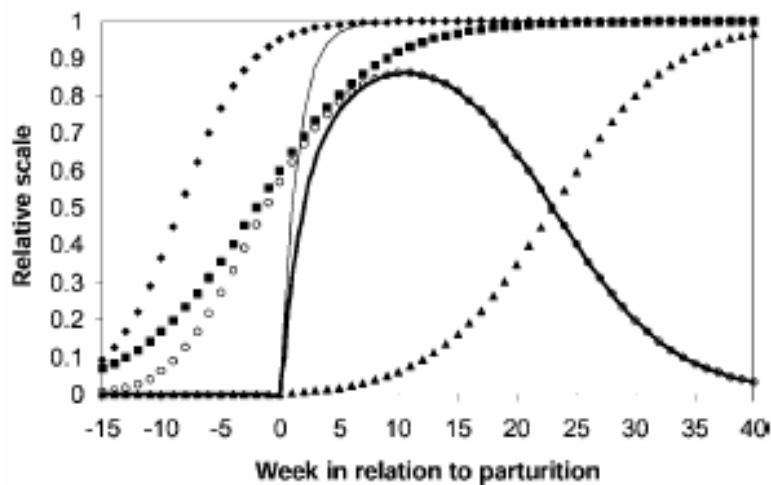
The parameters of some lactation curves derived by empirical equations usually have little or no biological meaning and provide little insight into what is happening to the animal during pregnancy and lactation (Pollot et al., 2000). Dijkstra et al. (1997) developed a four-parameter mechanistic model describing the pattern of mammary growth throughout pregnancy and lactation (Table 1.6). In Dijkstra’s equation, a = theoretical initial milk production (kg/day), b = specific rate of secretory cell proliferation at parturition, c = decay parameter (per day), and d = specific rate of secretory cell death (per day). The parameter d describes the degree of persistency as cell death has been identified as the main course of milk yield decline after peak lactation (Capuco et al., 2003).

**Lactation persistency and mammary gland proliferation:** Three major processes are involved in the changes of cell number in the mammary gland during gestation and lactation (Pollot et al., 2000).

- i. Mammary parenchyma cell proliferation,
- ii. Differentiation of parenchyma cell into milk secretory cells, and
- iii. Decline in mammary gland cell number through apoptosis

As Figure 1.7 shows, the cell proliferation starts in early gestation and rises exponentially until it reaches its maximum soon after parturition. Dijkstra et al. (1997) found that more than 50 percent of the proliferation was completed by the time of parturition.

Furthermore, a study on mammary growth in Holstein cows during the dry period by Capuco et al. (1997) found that around the time of calving, 83% of mammary cells were epithelial cells. The enzyme activity per cell did not reach maximum until several days or few weeks after parturition, after which the activity remained constant (Capuco et al., 2003). In contrast, after peak lactation cell number tends to decline exponentially (Figure 1.7).



**Figure 1.7** A schematic representation of the biology of the mammary gland during pregnancy and 40-week lactation showing parenchyma cell production (◆), secretory cell differentiation (■), cell dying by apoptosis (▲), potentially active cells (○), secretory rate (—) and milk production (—) (Pollot et al., 2000).

“Therefore, as Knight and Parker, (1984) demonstrated, increasing milk production in early lactation can first be the result of an increase in mammary cell number and followed by increasing secretory activity per cell. After peak lactation, decreased milk yield with advancing lactation was primarily the result of declining cell number” (Capuco et al., 2003).

However when cows are concomitantly lactating and pregnant, the conflicting nutrition demand of gestation and lactation may lead to decrease in secretory capacity of mammary cells (Capuco et al., 2003). Bachman et al. (2002) noted a negative effect of pregnancy on milk production as early as the 100<sup>th</sup> day of pregnancy that coincided with the onset of estrogen secreted by the fetal-placental unit, indicating that estrogen could be a mediator of the inhibitory effects of pregnancy on lactation. Nevertheless, the effect of pregnancy is likely to be readily apparent during late pregnancy when milk production declines rapidly. Hence, the stage of pregnancy seems to be of particular interest when persistency of milk production is studied.

**Systemic and local regulatory factors affecting persistency:** As lactation progresses, the secretory cells gradually regress from a state of active synthesis and secretion to a non-secretory state through a process called “involution”. In a typical lactation curve, gradual involution starts around peak lactation and continues up to the time when the animals are dried off.

Maintenance of milk synthesis and secretion (or persistency) is controlled by both systemic and local regulatory factors. The systemic factors involve hormones such as prolactin and growth hormone (GH). Changes of their circulating levels affect the rate of secretion of milk. As lactation progresses, the level of growth hormone and prolactin decrease causing a reduction of milk synthesis (Dijkstra et al., 1997). The influence of these hormones in reducing involution is mediated by the insulin-like growth factor-I (IGF-I), a hormone that is well known to increase milk yield (Tonner et al., 2000).

In addition to systematic factors, local factors such as the feedback inhibitor of lactation (FIL) and the plasminogen-plasmin system are involved in the involution process. FIL is a peptide that is synthesized by mammary epithelial cells and secreted with the milk into alveoli. As time from last milking increases, milk accumulates in the

alveoli with this peptide causing a progressive reduction of milk synthesis and secretion. Therefore, frequent removal of milk from mammary gland reduces local inhibitory effects on milk secretion (Wilde and Knight, 1990).

On the other hand, Politis (1996) reported the involvement of plasmin-plasminogen system during gradual involution of the mammary gland. Plasmin is the predominant protease in milk. Both plasmin and its precursor plasminogen are present simultaneously in milk. Administration of exogenous GH in cows increases milk yield and lactation persistency and reduces plasmin activity, probably through its mediator IGF-I (Politis, 1996). However, the effect of GH, mediated through IGF-I, is more likely to be significant in well-fed animals, indicating a positive relationship between nutritional status of cows and lactation persistency.

**Altering lactation persistency through management:** Three potential means to alter the lactation curve have been investigated in recent years (Cannas et al., 2002).

- i. Bovine somatotrophin (bST) administration
- ii. Alteration in milk frequency
- iii. Photoperiodic manipulation.

Administration of bST increased the persistency of milk yield after the peak. This suggests the effect of bST on lactation persistency is due to the maintenance of mammary cell population rather than maintenance of cellular activity in advance lactation. However, in early lactation, the positive effect of bST tends to be insignificant because its impact is blunted by post partum negative energy balance (Cannas et al., 2002).

Capuco et al. (2003) noted that increased milking frequency (IMF) at the beginning of lactation of cows has shown to increase milk yield not only during IMF, but also after it is stopped. Based upon this finding, they suggested that increased milking frequency during early lactation may increase mammary growth and thus produce a carryover effect on milk production for the majority of lactation.

A review on photoperiodic effects of dairy cattle, by Dahl et al. (2000 noted) “lactating dairy cows exposed to long-day photoperiods (16 to 18 hours of light) produced more milk than cows exposed to short-day (<12 hours) photoperiods”. The effects of long-day photoperiods were associated with increasing levels of IGF-I secretion and IGF-I appears the most likely mediator for the galactophoietic effects of increased photoperiod (Capuco et al., 2003).

**Best prediction of lactation persistency:** There may be an antagonistic relationship between 305-d lactation yield and persistency (Dekkers et al., 1998 and Jakobsen et al., 2003). Selection for persistency correlated with yield may lead to a decrease in total production per cow. Therefore a measure of persistency uncorrelated with milk yield is of particular importance because it allows an effective selection for 305 d yield and persistency simultaneously (Muir et al., 2004).

Cole and VanRaden (2006) introduced a method of calculating persistency that is independent of yield. According to this method, persistency is a function of TD yield deviation from trait specific (milk, fat, and protein) standard curve ( $Y_i - S_i$ ) and TD DIM deviation around a reference date ( $d_i - d_0$ ):

$$p = \sum_{i=1}^n (Y_i - S_i) \times (d_i - d_0)$$

Where,

$p$  = persistency of an individual lactation

$Y_i$  =  $i^{\text{th}}$  TD yield

$S_i$  = standard yield on  $i^{\text{th}}$  TD

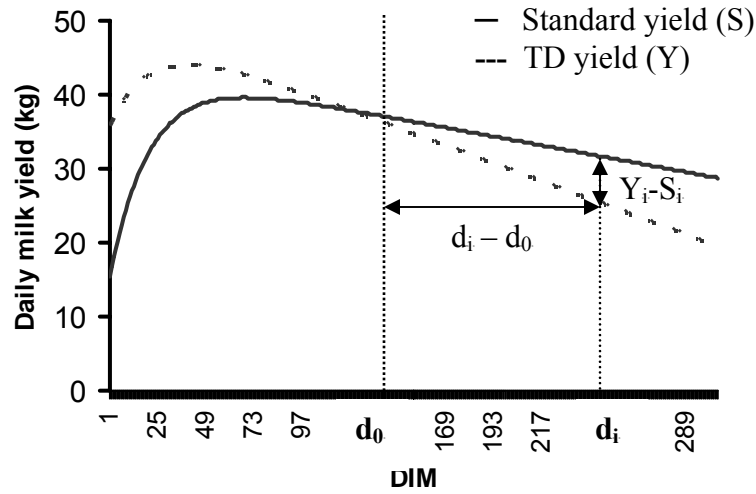
$d_i$  = DIM at  $i^{\text{th}}$  TD

$d_0$  = DIM at the reference date

$Y_i - S_i$  =  $i^{\text{th}}$  TD yield deviation from the standard yield (Figure 1.8)

$d_i - d_0$  =  $i^{\text{th}}$  TD DIM deviation from the reference date (Figure 1.8)

$n$  = total number of TD yield records used to calculate persistency



**Figure 1.8** The standard curve (solid line) vs. a lactation curve of an individual cow (dot line).

In matrix form, let  $\mathbf{t}$  represent the vector whose elements are TD yield deviations ( $Y_i - S_i$ ) and  $\mathbf{q}$  represent the vector whose elements are TD DIM deviations ( $d_i - d_0$ ).

$$\text{Persistence} = \mathbf{q}'\mathbf{t} \text{ or}$$

If  $\mathbf{d}$  is the vector with elements,  $d_i$ , then  $\mathbf{q} = \mathbf{d} - \mathbf{1}d_0$  and Persistence =  $(\mathbf{d}' - \mathbf{1}'d_0) \mathbf{t}$

The reference date,  $d_0$ , is a constant that acts as a balance point between yields in early and late lactation and makes persistency uncorrelated with yield. Therefore  $d_0$  is obtained by setting Cov ( $\mathbf{q}'\mathbf{t}$ ,  $\mathbf{1}'\mathbf{t}$ ) to 0 and solving for  $d_0$ :

$$\text{Cov}(\mathbf{q}'\mathbf{t}, \mathbf{1}'\mathbf{t}) = \mathbf{q}'\mathbf{V}\mathbf{1} = (\mathbf{d}' - \mathbf{1}'d_0) \mathbf{V}\mathbf{1} = \mathbf{d}'\mathbf{V}\mathbf{1} - \mathbf{1}'\mathbf{V}\mathbf{1}d_0 = 0$$

$$d_0 = \mathbf{d}'\mathbf{V}\mathbf{1} / \mathbf{1}'\mathbf{V}\mathbf{1}, \text{ where } \mathbf{V} \text{ is the variance of } \mathbf{t}$$

VanRaden (1998) determined  $d_0$  to be 128, 146, and 135 for milk, fat and protein yields.

A standardized estimate of persistency,  $\hat{s}$ , is obtained by converting persistency to a unit normal variable using population (e.g. herd or parity group) mean and standard deviation of persistency.

$$\hat{s} = \frac{p - \mu_p}{SD}$$

The mean and variance of  $\hat{s}$  are 0 and 1, respectively. Positive values of  $\hat{s}$  indicate increased persistency relative to standard curve (curve of an averagely persistent cow) and negative values of  $\hat{s}$  indicate decreased persistency.

**Economic aspects of lactation persistency in dairy cattle:** Besides the anticipated relationship between persistency and reduced probability of diseases, there may be some additional advantages from selecting for higher persistency. Dekkers et al (1998) investigated the impact of lactation persistency on feed costs and milk returns. The results suggested that increasing persistency tended to reduce feed costs per unit of milk returns in multiparous cows. “Greater persistency resulted in a distribution of production over the lactation period that was more in line with dry matter intake capacity. As a result, a greater portion of energy requirements throughout the lactation could be met from less expensive roughages versus concentrates, thereby lowering the feed cost” (Dekkers et al., 1998).

Dekkers et al. (1998) found that persistency had a negative effect on milk returns for short lactations (calving interval <12 months) and a positive effect for long lactations (calving interval >12 months). Thus, persistent lactations become more profitable when lactation length is greater than 305 d or the calving interval is greater than 12 months. Therefore, persistency can have significant implications for insemination and culling decisions. For high producing cows with highly persistent lactations, the optimum time of voluntary culling and first insemination can be delayed (Dekkers et al., 1998).

## REFERENCES

- Bachman, K. C. 2001. Milk production of dairy cows treated with estrogen at the onset of a short dry period. *J. Dairy Sci.* 85: 797-803.
- Baird, G. D. 1982. Primary ketosis in the high-producing dairy cows: clinical and subclinical disorders, treatment, prevention, and outlook. *J. Dairy Sci.* 65(1): 1-10.
- Booth, C. J., L. D. Warnick, Y. T. Grohn, D. O. Maizon, C. L. Guard, and D. Janssen. 2004. Effect of lameness on culling dairy cows. *J. Dairy Sci.* 87: 4115-4122.
- Cassell, B. G. 2001. using heritability for genetic improvement. <http://www.ext.vt.edu/pubs/dairy/404-084/404-084.html>. Accessed October 21, 2006.

- Capuco, A. V., R. M. Akers, and J. J. Smith. 1997. Mammary growth in Holstein cows during the dry period: quantification of nucleic acids and histology. *J. Dairy Sci.* 80 (3): 477-487
- Capuco, A. V., S. E. Ellis, S. A. Hale, R. A. Edman, X. Zhao, and M. J. Paape. 2003. Lactation persistency, insights from mammary cell proliferation studies. *J. Anim. Sci.* 81: 18-31
- Carlen, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.*: 87(9): 3062-3070.
- Carlen, E., U. Emanuelson, and E. Strandberg. 2006. Genetic evaluation of mastitis using linear models, threshold models, and survival analysis: a simulation study. *J. Dairy Sci.* 89: 4049-4057.
- Cole, J. B. and P. M. VanRaden. 2006. Genetic evaluation and best prediction of lactation Persistency. *J. Dairy Sci.* 89: 2722-2728.
- Cannas, A., A. Nudda, and G. Pulina. 2002. Nutritional strategies to improve lactation persistency in dairy ewes. <http://www.uwex.edu/animalscience/sheep/> Accessed October 21, 2006.
- Collard, B. L., P. J. Boettcher, J. C. Dekkers, D. Petitcler, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. *J. Dairy Sci.* 83(11): 2683-2690.
- Correa, M. T., H. Erb, J. Scarlett. 1993. Path analysis for seven postpartum disorders of Holstein cows. *J. Dairy Sci.* 76: 1305-1312.
- Dahl, G. E., B. A. Bachman, and H. A. Tucker. 2000. Photoperiodic effects on dairy cattle: a review. *J. Dairy Sci.* 83: 885-893.
- Dechow, C. D., G. W. Rogers, L. Klei, T. J. Lawlor, and P. M. VanRaden. 2004. Body condition scores and dairy form evaluations as indicators of days open in US Holsteins. *J. Dairy Sci.* 2004: 87(10):3534-41.
- Dekkers, J. C. M., J. H. Ten Haag, and A. Weersink. 1997. Economic aspects of persistency in dairy cattle. *Livest. Prod. Sci.* 53:237-252.

- Dijkstra, J., J. France, M. S. Dhanoa, J. A. Maas, M. D. Hanigan, A. J. Rook, and D. E. Beever. 1997. A model to describe growth patterns of the mammary gland during pregnancy and lactation. *J. Dairy Sci.* 80(10): 2340-2354.
- Ferris, T. A., I. L. Mao, and C. R. Anderson. 1985. Selection for lactation curve and milk yield in cattle. *J. Dairy Sci.* 68:1438-1448.
- Garverick, H. A. 1997. Ovarian follicular cysts in dairy cows. *J. Dairy Sci.* 80: 995-1004
- Gill, R., H. H. Wayne, L. E. Kenneth, and K. Lissersmore. 1990. Economics of mastitis control. *J. Dairy Sci.* 73: 3340-3348.
- Green, B. L., B. W. McBride, D. Sandals, K. E. Leslie, R. Bagg , and P. Dick. 1999. The impact of monensin controlled-release capsule on subclinical ketosis in the transition dairy cow. *J. Dairy Sci.* 82: 333-342.
- Grossman, M., S. M. Hartz, and W. J. Koops. 1999. Persistency of lactation yield: a Novel approach. *J. Dairy Sci.* 82:2192-2197
- Harder, B., J. Bennewitz, D. Hinrichs, and E. Kalm. 2006. Genetic parameters for health traits and their relationship to different persistency traits in German Holstein dairy cattle. *J. Dairy Sci.* 89:3202-3212
- Henringstad, B., Y. M. Chang, I. M. Andersen-Ranahag, and D. Gianola. 2004. Genetic analysis of number of mastitis cases and number of services to conception using a censored threshold model. *J. Dairy Sci.* 89(10): 4042-4048.
- Hinrichs, D., E. Stamer, W. Junge, and E. Kalm. 2005. Genetic analysis of mastitis data using animal threshold model and genetic correlation with production traits. *J. Dairy Sci.* 88: 2260-2268.
- Jakobsen, J. H., R. Rekaya, J. Jensen, D.A. Sorensen, P. Madsen, D. Gianola, L.G. Christensen, and J. Pedersen. 2003. Bayesian estimates of covariance components between lactation curve parameters and disease liability in Danish Holstein cows. *J. Dairy Sci.* 86:3000-3007.
- Jamrozik, J., L. R. Schaeffer, and J. L. Dekkers. 1997. Genetic evaluation of dairy cattle using test day yields and random regression model. *J. Dairy Sci.* 80(6): 1217-1226.

- Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. *J. Dairy Sci.* 81 (9): 2502-2509.
- Koenig, S., A. R. Sharifi, H. Wentrot, D. Landmann, M. Eise, and H. Simianer. 2005. Genetic parameters of claw and foot disorders estimated with logistic model. *J. Dairy Sci.* 88:3316-3325.
- Lewis, G. S. 1997. Uterine health and disorders. *J. Dairy Sci.* 80(5):984-994.
- Lin, C. Y. and K. Togashi. 2005. Maximization of lactation milk production without decreasing persistency. *J Dairy Sci.* 2005 :88(8):2975-2980.
- Lin, H. K., P. A. Oltenacu, I. D. Van Vleck, H. N. Erb, and R. D. Smith. 1989. Heritabilities of and genetic correlations among six health problems in Holstein Cows. *J. dairy Sci.* 72: 180-186.
- Lyons D. T., and A. E. Freeman .1991. Genetics of health traits in Holstein Cattle, *J. Dairy. Sci.* 74: 1092-1100.
- Lucy, M. C. 2005. Non-lactational traits of importance in dairy cows and applications for emerging biotechnologies. [N Z Vet J.](#) 53(6):406-415.
- Mantysaari, E.A., Y.T. Grohn, R.L. Quaas. 1991. Clinical ketosis: phenotypic and genetic correlations between occurrence and with milk yield. *J. Dairy Sci.* 74(11): 827-833.
- Miglior, F., B. L. Muir, and B. J. Van Doormaal. 2005. Selection indices in Holstein cattle of various countries. *J. Dairy Sci.* 88: 1255-1263.
- Muir, B. L., J. Fatehi, and L. R. Schaeffer. 2004. Genetic relationships between persistency and reproductive performances in first-lactation Canadian Holsteins. *J. Dairy Sci.* 87: 3029-3037.
- Pollot, G. E. 2000. A biological approach to lactation curve analysis for milk yield. *J. Dairy Sci.* 83(11): 2448-2456.
- Politis, I. 1996. Plasminogen activator system: implications for mammary cell growth and involution. *J Dairy Sci.* 79(6):1097-1107.
- Powell, R. L. and H. D. Norman. 2006. Major advances in genetic evaluation techniques. *J. Dairy Sci.* 89(4): 1337-1348.

- Raizman, E.A., and J. E. Santos. 2002. The effect of left displacement of abomasums corrected by Toggle-pin suture on lactation, reproduction, and health of Holstein dairy cows. *J. Dairy Sci.* 85: 1157-1164.
- Rogers G. W., G. Banos, and U. Sander-Nielsen. 1999. Genetic correlations among protein yield, productive life and type traits from the United States and diseases other than mastitis from Denmark and Sweden, *J. Dairy. Sci.* 82 : 1331-1338.
- Sander Nielsen, U., G. A. Pedersen, J. Pederson, and J. Jensen. 1996. Genetic parameters for mastitis, other diseases and somatic cell count in different parities in Danish dairy breeds. Page 10 in 47<sup>th</sup> Annu. Mgt. of EAAP, Lillehammer, Norway.
- Scott., T. A., B. Yandell, L. Zepeda, R.D. Shaver, and T.R. Smith. 1996. Use of lactation curves for analyzing of milk production data. *J. Dairy Sci.* 79: 1885-1894.
- Shaver, R. D. 1997. Nutrition risk factors in the etiology of left displaced abomasum in dairy cows: a review. *J. Dairy Sci.* 80: 2449-2453.
- Shim, E. H., R. D. Shank, and D. E. Morin. 2004. Milk loss and treatment costs associated with two treatment protocols for clinical mastitis in dairy cows. *J. Dairy Sci.* 87(8):2702-2708.
- Shook,G. E. 1989. Major advances in genetic evaluation techniques. *J. Dairy Sci.* 89(4):1337-1348.
- Shook, G. E. 2006. Major advances in determining appropriate selection goals. *J. Dairy Sci.* 89(4):1349-1361.
- Sogstad, A. M., T. Fjeldaas, and O. Osteras. 2005. Lameness and claw lesions of the Norwegian red dairy cattle housed in free stalls in relation to environment, parity and stage of lactation. *Acta Vet. Scand.:* 46(4):203-217.
- Solkner, J., and W. Funchs. 1987. A comparison of different measures of persistency with special respect to variation of test-day milk yields. *Livest. Prod. Sci.* 16:305-319.
- Stanton, T. L., L. R. Jones, R. W. Everett, and S. D. Kachman. 1992. Estimating milk, fat, and protein lactation curves with a test day model. *J. Dairy Sci.:*75(6):1691-1700.

- Tonner, E., G. J. Allen, and D. J. Flint. 2000. Hormonal control of plasmin and tissue-type plasminogen activator activity in rat milk during involution of mammary gland. *J. Endocrinol.* 167: 265-273.
- Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Ketton. 1994. Genetic parameters for common health disorders of Holstein cows. *J. Dairy Sci.* 78:421-430.
- Urton, G., M. A. G. von Keyserlingk, and D. M. Weary. 2005. Feeding behavior identifies dairy cows at risk for metritis. *J. Dairy Sci.* 88: 2843-2849.
- Val-Areola, D., E. Kebreab, J. Dijkstra, and J. France. 2004. Study of the lactation curve in dairy cattle on farms in central Mexico. *J. Dairy Sci.* : 87(11):3789-99.
- Van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders and relationships with 305-day milk yield and conformation traits of registered Holstein cows. *J. Dairy Sci.* 81:2264–2270.
- VanRaden, P. M., 1998. Best prediction of lactation yield and persistency. Proc. 6<sup>th</sup> World Congr. Genet. Appl. Livest. Prod., Armidale, Australia XXIII:347-350.
- Warnick, L. D., D. Janssen, C. L. Guard, and Y. T. Grohn. 2001. The effect of lameness on milk production in dairy cows. *J. Dairy Sci.* 84: 1988-1997.
- Wilde, C. J. and C. H. Knight. 1990. Milk yield and mammary function in goats during and after once-daily milking. *J. Dairy Res.* 57(4): 441-447.
- Wilson, D. J., R. N. Gonzalez, J. Hertl, H. F. Schulte, G. J. Bennett, Y. H. Schukken, and Y. T. Grohn. 2004. Effect of clinical mastitis on the lactation curve: a mixed model estimation using daily milk weight. *J. Dairy Sci.* 87:2073-2084.
- Wood, P.D.R. 1997. Algebraic model of the lactation curve in cattle. *Nature* 216:164-165.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer recorded data. I. incidence rates, heritability estimates, and sire breeding values. *J. Dairy Sci.* 87:4287-4294.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2006. Genetic analysis of clinical mastitis data from on-farm management software using threshold models. *J. Dairy Sci.* 89(1): 330-336.

## **CHAPTER III**

### **Manuscript 1**

## **Phenotypic Relationships of Common Health Disorders in Dairy Cows to Lactation Persistency, Estimated from Daily Milk Weights**

### **ABSTRACT**

The objective of this study was to investigate the phenotypic relationship between common health disorders in dairy cows and lactation persistency, uncorrelated with 305 d yield. The relationships with peak yield and days in milk (DIM) at peak were also studied. Daily milk weights and treatment incidence records of 991 Holstein lactations from experimental dairy herds at Virginia Tech and Pennsylvania State University were used. Persistency was calculated as a function of daily yield deviations from standard lactations curves, separately developed for first (FL) and later lactations (LL), and deviations of DIM around reference dates: 128 for FL and 125 for LL. DIM at peak and peak yield were computed for each lactation using Wood's function. The disease traits studied were mastitis only during first 100 days (MAST1), only after 100 DIM (MAST2), both before and after 100 DIM (MAST12) and at any stage of lactation (MAST1/2), and Metritis (MET), displaced abomasums (DA), lameness (LAME), and metabolic diseases (METAB). Each disease was defined as a binary trait distinguishing between lactations with at least one incidence (1) and lactations with no incidences (0). Effect of diseases on persistency, DIM at peak and peak yield were investigated separately for FL and LL for all disease traits except MAST12, which was investigated across parities. The effect of persistency on probability of diseases in same lactation and in next lactation was examined using odds ratios from a logistic regression model. MET and DA had significantly positive effects on persistency in both FL and LL. The positive effects of METB and MAST1 on persistency were significant in LL. The effects of MAST2, in both FL and LL, and MAST12, across parities, were significant but negative. Overall, cows affected by mastitis tended to have less persistent lactations. Majority of the diseases tended to significantly affect DIM at peak in LL. In LL, MET, METAB, and DA tended to significantly delay DIM at peak while MAST2 was associated with significantly early DIM at peak in LL. Increasing persistency tended to reduce likelihood

of MAST2 and MAST1/2 in current lactations of primiparous cows. None of the diseases studied was significantly affected by persistency of previous lactation.

**(Key words:** cow, health disorder, phenotypic relationship, persistency)

## INTRODUCTION

Persistency is defined as the ability of a cow to maintain milk production at a high level after the peak yield (Jamrozik et al., 1998). High persistency is associated with a slow rate of decline in yield after peak production, while low persistency is associated with a rapid rate of decline. A cow with a higher persistency tends to incur less feed, health and reproductive costs (Solkner and Funch, 1987). Persistent lactations are characterized by lower peak yield (Dekkers et al., 1998) reached at later days in milk, indicating their association to reduced metabolic stress in early lactation (Ferris et al., 1985).

Antagonistic genetic correlations between milk production and disease traits (Semianer et al., 1991) indicate that increased disease incidence in today's dairy herd (Zwald et al., 2004) is in part a consequence of genetic improvement in milk production. Sick cows are less profitable and can lead to ethical concerns related to animal welfare and consumer interest (Jakobsen et al., 2003). Diseases such as mastitis, displaced abomasums, ketosis, cystic ovaries, metritis and lameness can dramatically affect the profitability of dairy herds through involuntary culling, veterinary treatments, added labor, and lost milk sales (Zwald et al., 2004). Many countries are initiating negative selection pressure on disease susceptibility by including disease resistance in breeding goals (Jakobsen et al., 2003).

Direct selection for disease resistant requires accurate records of disease incidence and severity. Many producers do not record diseases in a manner useful for the purpose. When direct selection for disease resistant is not possible, correlated traits could be useful in indirect selection. Hypothetically (Dekkers et al., 1998), cows having highly persistent lactations are less liable to diseases as they undergo less metabolic stress specially from calving to peak. Thus, genetic changes towards more persistent lactations could be used as a mean to lower disease susceptibility in dairy cows. However,

persistence may not be justified at the expense of milk yield, as 305 d yield tends to be negatively affected by increasing persistence (Dekkers et al., 1998, Togashi and Lin, 2003). Therefore, persistence measures uncorrelated with total yield will allow more efficient selection for total lactation yield and persistence simultaneously (Muir et al, 2004). A phenotypic measure of persistence that is independent of yield can be calculated as a function of a standard lactation curve and a linear function of a cow's test day deviations on days in milk (Cole and VanRaden, 2006).

The objective of current study was to examine phenotypic relationships between lactation persistence, uncorrelated with 305 d yield, and common health disorders in dairy cows using daily milk records from experimental dairy farms at Virginia Tech (VT), and Pennsylvania State University (PSU).

## **MATERIALS AND METHODS**

### **Data**

Daily milk yield and treatment incidence of Holstein lactations initiated by calving on or after July 7, 2001 at PSU and July 18, 2004 at VT, were used. Lactations of at least 260 DIM were chosen to calculate persistence. The edited dataset contained 326 first lactations (FL) and 511 later lactations (LL) from PSU, and 59 FL and 95 LL from VT.

### **Calculation of persistence**

VanRaden (1998) reported a method of calculating lactation persistence by multiplying test-day (TD) deviations from a standard lactation curve by corresponding days in milk (DIM) deviations around a reference date,  $d_0$ .

$$p = \sum_{i=1}^n (Y_i - S_i) \times (d_i - d_0)$$

Where,  $p$  = persistency of an individual lactation  
 $Y_i$  =  $i^{\text{th}}$  TD yield  
 $S_i$  = standard yield on  $i^{\text{th}}$  TD  
 $d_i$  = DIM at  $i^{\text{th}}$  TD  
 $d_0$  = DIM at the reference date  
 $n$  = total number of TD yield records used to calculate persistency.

A measure of persistency that is phenotypically uncorrelated with lactation yield may be obtained by defining  $d_0$  as a balance point between yields in early and late lactation (Cole and VanRaden, 2006). We used 128 and 125 DIM as the reference dates for FL and LL respectively in this study. As the shape of the lactation curve differs between primiparous and multiparous cows (Jakobsen et al., 2003), two standard lactation curves were developed to calculate persistency for FL and LL separately. We separately fit mean daily milk yields in FL and LL, across herds to Wood's function (Wood, 1967) and developed the two standard lactation curves shown in Figure 2.1.

A standardized estimate of persistency ( $\hat{s}$ ) was obtained by subtracting within-lactation (FL or LL) mean ( $\mu_p$ ) and dividing by within-lactation (FL or LL) phenotypic standard deviation (SD) of calculated persistency.

$$\hat{s} = \frac{p - \mu_p}{SD}$$

Positive values of  $\hat{s}$  indicate increased persistency relative to an average cow and negative values of  $\hat{s}$  indicate decreased persistency.

### **Defining disease traits**

Herd treatment incidence records were used to define disease traits for mastitis, metritis, ketosis, milk fever, displaced abomasums and lameness that were recognized as common health disorders in dairy cows (Zwald et al, 2004). The farm crew at PSU and VT used DAIRY COMP 305 and PCDART respectively to record the treatment events. Both herds are frequently supported by veterinarians and have very thorough recording of health events. Treatment incidences for all udder infections were considered as mastitis.

We chose to consider mastitis under two separate stages of lactation: early (before 100 DIM) and late (after 100 DIM), because mastitis in early lactation is only slightly correlated with mastitis in late lactation (Zwald et al., 2006). Four disease traits were formed with respect to mastitis: MAST1, MAST2, MAST12, and MAST1/2, representing mastitis, only in the early stage, only in the late stage, in both the early and late stages, and in either of the stages respectively. A disease variable for lameness (LAME) was formed considering treatment incidences for all causes of limping and abnormal weight bearing including laminitis, foot rot, hoof abscess, over-grown hoof, and pelvic abscess. Treatments for vaginal discharge or an enlarged uterus diagnosed through veterinary palpation were considered to be metritis (MET). Treatment incidences for both ketosis (KET) and milk fever (MKFV) were considered together as one disease trait, metabolic diseases (METAB). Disease trait for displaced abomasums (DA) was formed considering the treatment incidences for both left and right abomasal displacements. Each disease trait was defined as a binary trait distinguishing between cows with at least one reported incidence during the defined period (1) and cows without cases (0) (Carlen et al., 2004). Besides aforementioned disease traits, we chose three other health disorders: retained placenta, ovarian cyst and diarrhea, for inclusion in the statistical models.

### **Computation of peak yield and DIM at peak**

Although the relationships between persistency and diseases were our main interest, we also examined the effect of diseases on other lactation curve characteristics, in particular peak yield and DIM at peak as they would be useful in explaining the relationship between diseases and persistency. Wood's equation was chosen to depict the shape of the lactation curve:

$$Y_t = at^b e^{-ct}$$

Where  $Y_t$  is production (kg) on day  $t$ ,  $a$  is a scaling factor estimating production at time zero,  $b$  is rate of ascent to peak,  $c$  is rate of descent after peak. Two functions using  $a$ ,  $b$ , and  $c$ , were computed;  $a[b/c]^c e^{-b}$  to calculate peak yield and  $[b/c]$  to estimate DIM at peak (Ferris et al, 1985). Parameter estimates for individual lactations of at least 260 d length, were obtained through the Gauss-Newton method in the nonlinear (NLIN) procedure in SAS (1999).

## Statistical Analysis

We examined the phenotypic relationships of the disease traits to milk yield persistency in two directions: first, effects of the diseases on the persistency and then, the effect of persistency on probabilities of disease occurrence.

**Effects of the diseases on persistency, peak yield, and DIM at peak:** The following model was used to investigate the effect of each disease trait on persistency, peak yield, and DIM at peak.

$$Y_{ijklmn} = \mu + H_i + YR_j + S_k + D_l + O_m + \beta_1 DO_{ijklmn} + \beta_2 AGE_{ijklmn} + e_{ijklmn}$$

Where;

- Y = standardized persistency, peak yield or DIM at peak
- $\mu$  = Overall mean of persistency, peak yield or DIM at peak
- H = fixed effect of Herd [VT or PSU]
- YR = fixed effect of calving year [2001, 2002, 2003, 2004 or 2005]
- S = fixed effect of calving season [1(February – April), 2 (May – July), 3 (August – October), and 4 (November – January)]
- D = fixed effect of presence (1) or absence (1) of at least one incidence of the main disease of interest
- O = fixed effect of presence (1) or absence (1) of at least one incidence of any other disease besides main disease of interest
- $\beta_1$  = regression for days open (DO)
- $\beta_2$  = regression for age at calving (AGE) in mo
- $e_{ijklmn}$  = residual error  $\sim N(0, I\sigma_e^2)$ .

The variable other diseases (O) included retained placenta, ovarian cyst, diarrhea and the other defined disease traits besides the main disease trait of interest, i. e, the other diseases for MAST1 were MAST2, MAST12, MET, METAB, DA, LAME, retained placenta, ovarian cyst, and diarrhea. Days open less than 50 were set to 50, and days open greater than 250 were set to 250 (Cole and VanRaden, 2006). Primiparous cows differ from multiparous cows as they produce less milk and have different incidence rates for many diseases (Oltenacu et al, 1990). On the other hand, Jumrozik et al. (1998) suggested that persistency in different lactations can be considered as different traits. We chose to perform separate analyses for FL and LL. However, when disease frequency was <5%, as

for MAST12, data for primiparous and multiparous cows were pooled to avoid loss of information by empty cells (Uribe et al, 1995). When cows are concomitantly lactating and pregnant, conflicting metabolic demands of gestation and lactation in advanced pregnancy might enhance the decline in milk yield in late lactation (Capuco et al, 2003). We included DO in the statistical model to account for this effect. AGE accounted for some parity differences in LL and the negative correlation between persistency and age of heifers at breeding, as reported by Muir et al (2004).

**Effect of persistency on the probability of diseases:** We examined effect of persistency on the likelihood of the diseases in current lactation as well as next lactation. We chose not to include MAST1, DA, MET, and METAB in this analyses for the effect of persistency on diseases in current lactation because expression of these disease traits preceded the expression of persistency.

A linear logistic model was chosen to investigate the effect of persistency on the probability of disease occurrence (Domecq et al., 1997). The probability of observing the disease of interest ( $Y_i=1$ ) is  $\pi_i$  and the logit of observing the disease ( $Y_i$ ) is:

$$\text{Log} [ \pi_i / (1 - \pi_i) ] = \eta_i$$

Where,  $\eta_i$  is the linear predictor of the logistic regression model,  $Y_i = 1 / 1 + e^{-\eta_i}$ .

Because  $\pi$  is the probability that  $Y_i = 1$ , it follows that  $1 - \pi$  is the probability of  $Y = 0$ ; then,  $\pi / (1 - \pi)$  is the odds ratio of the 2 probabilities. Any factor that increases  $\eta_i$  leads to a concomitant increase in  $\pi_i$  (Koenig et al, 2005).

We computed several logistic regression models, including indicator variables for class effects such as herd, year of calving, season of calving and presence or absence of the other diseases, linear and quadratic effects of persistency, days open, cow age at calving, and interactions among independent variables. We removed the non-significant regression coefficients from the initial model based on Type 3  $\chi^2$ -statistics for likelihood ratios at  $p < 0.1$  given by GENMOD procedure in SAS (1999) (Montgomery et al, 2001). The following regression model was chosen.

$$\eta_{ijkl} = \beta_0 + \beta_1 S_{2i} + \beta_2 Y_{2j} + \beta_3 O_{1k} + \beta_4 P_{ijkl} + \beta_5 AGE_{ijkl}$$

Where:  $\eta$  = logit of observing the disease

$\beta_0$  = intercept

$S_2$  = effect of season 2 (March –May)

$Y_2$  = effect of calving year 2002

$O$  = effect of other diseases

$P$  = effect of standardized persistency

$AGE$  = effect of age at calving.

Effects of persistency on probability of disease occurrence ( $Y=1$ ) were investigated in terms of the corresponding odds ratios. The significance of the odds ratio was determined, based on its 95% confidence interval (CI). CI including 1 was considered to represent a non-significant association between disease incidence and persistency. We expressed persistency in SD units. Therefore, the estimated odds ratios in this study were standardized odds ratios (Domecq et al., 1997) that describe change in probabilities of the diseases in response to a SD unit increase in persistency.

The effect of persistency on the probabilities of MAST2, MAST12, MAST1/2, and LAME in same lactation was examined separately for FL and LL. We used the same logistic model to investigate the effect of persistency on likelihood of the diseases in next lactation. 181 cows with both first and second lactations were involved in this analysis.

## RESULTS

### Calculation of persistency

In this study, persistency was calculated using daily milk records from 305 d lactations. However, lactations that failed to complete 305 d but reached at least 260 DIM were also considered for persistency calculation. We made this decision based on the estimated correlations of persistency calculated using daily yields up to 305 DIM to those were calculated using daily milk yields up to 280, 260, 240, 210, and 180 DIM. The estimated correlations were 0.95, 0.89, 0.73, 0.30, and 0.002 respectively. Moreover, the notable shape difference between the standard lactation curves (Figure 2.1) indicated the necessity of two values for  $d_0$  to calculate persistency for FL and LL. VanRaden (1998)

determined  $d_0$  to be 128 for first lactation Holsteins. Considering 128 as an orientation point, correlations between 305-day milk yield and persistency were estimated for  $d_0$  values of 124, 125, 126, 127, 128, 129, and 130. The correlations are given in Table 2.1. We chose 128 and 125 to calculate persistency for FL and LL respectively, as these days produced phenotypic correlations between persistency and 305 d yield that were nearest to zero.

Persistency ranged from -3.78 to 3.88 in FL and from -3.03 to 2.88 in LL. The frequency histograms of the persistency for both FL and LL appear in figure 2.2. The persistency values were rounded up to form persistency classes for the histograms (e.g. persistency class 3 included lactations having persistency less than 3.5 and greater than 2.5). Distribution of the persistency was virtually normal in both FL and LL. The distributions of mean daily milk yields of FL and LL with high (persistency class 2), average (persistency class 0), and low (persistency class -2) persistencies, compared to the standard curves are presented in Figure 2.3a–2.3c. High, average, and low persistency classes consisted of 62, 399, and 50 lactations respectively. Mean daily yields of lactations with average persistency (Figure 2.3b) were virtually identical to the standard lactation curves. As expected, high persistent lactations (Figure 2.3a) produced less milk at the beginning of lactation and more milk at the end of lactation compared to lactations with the same level of production and average persistency (Cole and VanRaden, 2006). In contrast, lactations of low persistency (Figure 2.3c) produced more milk in early lactation and less milk during late lactation.

This study used data of 991 Holstein lactations from two experimental dairy herds, where daily milk yield was recorded. Larger studies would rely on test day (TD) records from commercial herds. We used the Cole and VanRaden (2006) method to investigate the number of TD yield records (per lactation) required to estimate persistency compared to the use of daily milk weights. The correlations of persistency calculated using daily milk weights ( $P_{305}$ ) to persistency calculated using one record per week ( $P_{wk}$ ), per two weeks ( $P_{2wk}$ ) and per month ( $P_{mon}$ ) were studied.  $P_{wk}$ ,  $P_{2wk}$ , and  $P_{mon}$  involved 44, 22, and 11 yield records respectively, so that  $P_{mon}$  was similar to using TD records. The correlations of  $P_{305}$  to  $P_{wk}$ ,  $P_{2wk}$ , and  $P_{mon}$ , were 0.99, 0.97 and 0.96

respectively, suggesting that TD records can be used to estimate persistency satisfactorily.

### **Disease incidence rates**

Table 2.2 shows the incidence rates (%) of health disorders considered in this study. The number of primiparous cows that develop mastitis only during early lactation (MAST1) was approximately similar to cows that developed mastitis only in late lactation (MAST2). But, many more multiparous cows tended to develop MAST2 than MAST1. The frequency of lactations with mastitis in both early and late lactation (MAST12) was low (3%) in FL but considerably higher (10.3%) in LL. The overall frequency of mastitis in early lactation is the summation of the frequencies for MAST1 and MAST12 (e.g.  $10.3\% + 7.5\% = 17.8\%$  for all lactations). Similarly, the overall frequency of mastitis in late lactation is the summation of MAST2 and MAST12 frequencies (e.g.  $12.7\% + 7.5\% = 20.2\%$  for all lactations). These frequencies suggest that cows in our data were more likely to have mastitis in late lactation (after 100 DIM). Mastitis at any time in lactation (MAST1/2) was greater in LL than FL. Approximately 60% of the multiparous cows escaped an incidence of mastitis. MET was more common in FL than LL (19.1% vs. 9.5%). DA and LAME frequencies in FL and LL were similar. The frequency of METAB increased from 10.6% in FL to 13.5% in LL as a consequence of increasing MKFV frequency (from 3.0% to 5.3%) while KET frequency remained fairly constant (7.6% in FL and 8.2% in LL). More than 85% of the incidences of METAB, DA and MET occurred during first 30 d after calving (not shown).

### **Effects of diseases on persistency**

Table 2.3 shows the least square means (LSMs) of persistency in lactations with (1) and without (0) each disease and significance (p-values) of the diseases on persistency. The effect of MAST1 on persistency was significant ( $p < 0.05$ ) in LL. The relatively greater LSM of lactations with MAST1 implies that multiparous cows, which were affected by mastitis only during early lactation, tended to have more persistent lactations than cows, which avoided mastitis. Conversely, MAST2 had significant but negative effect on persistency in both FL ( $p < 0.05$ ) and LL ( $p < 0.005$ ). The difference in

LSMs between affected and non-affected cows reflects mastitis in late lactation tended to reduce persistency. The effect of MAST12 on persistency was significant ( $p < 0.01$ ) across parities. The associated LSMs indicate a negative relationship between persistency and mastitis occurred in both early and late lactation. Similarly, the effect of MAST1/2 on persistency was also negative and significant ( $p < 0.05$ ) in LL. The positive effect of METAB (KET+MKFV) on persistency was significant ( $p < 0.05$ ) only in LL. DA and MET had significantly ( $p < 0.05$ ) positive effects on persistency in both FL and LL. The effect of LAME on persistency was clearly non-significant ( $p > 0.1$ ) in both FL and LL suggesting a weaker relationship between LAME and persistency.

### **Effects of diseases on peak yield and DIM at peak**

Table 2.4 presents the effect of each disease trait on peak yield in FL and LL. Of all disease traits only DA in FL and MAST2 in LL had significant ( $p < 0.05$ ) effects on peak yield. Conversely, METAB in both FL and LL, MET and DA in LL had significant ( $p < 0.05$ ) effects on DIM at peak (Table 2.5). The difference between least square means indicate that periparturiant diseases tended to delay the occurrence of lactation peak specifically in LL. The negative effect of MAST12 on DIM at peak was also significant across parities. As Table 2.6 shows the correlations of persistency to peak yield, and DIM at peak were -0.04 and 0.70 respectively in FL and -0.13 and 0.82 respectively in LL. Higher persistency was associated with low peak yield and later DIM at peak. However the phenotypic relationship between persistency and DIM at peak was much stronger as Ferris et al. (1985) reported.

### **Effect of persistency on probability of disease occurrence**

Odds ratio and associated confidence interval (CI) for the effect of persistency on likelihood of each disease in current lactation are presented in Table 2.7. The odds ratio for MAST2 in FL (0.46) indicates that for a standard deviation unit (SD-unit) increase in persistency, risk of mastitis only in late stage of lactation reduced by 0.54 times. Similarly, the probability of mastitis at any stage of lactation (MAST1/2) reduced by 0.41 times in response to a SD unit increase in persistency.

The odds ratios indicate that increasing persistency would reduce the probability of mastitis (MAST2, MAST12, and MAST1/2) in LL. However, the associated CIs, nearest to 1.0, suggest that the effect of persistency on mastitis in LL would be non-significant. Increasing persistency resulted in almost zero change in the probability of LAME in both FL and LL. Moreover, the odds ratios and associated CI (Table 2.8) indicate that likelihood of none of the diseases was significantly affected by the lactation persistency in previous lactation.

## **DISCUSSION**

### **Disease frequencies**

It is difficult to compare disease incidence frequencies across different studies as procedures for diagnosis, data recording methods, and disease trait definitions vary from study to study (Harder et al, 2006). Zwald et al (2004) reported mean lactational incidence rates (across parities) of 3%, 10%, 20%, 10%, and 21% for DA, KET, MAST1/2, LAME, and MET. The corresponding disease frequencies estimated by Wilson et al. (2006) were 2.2%, 12.3%, 19.6%, 33%, 13.9% respectively for FL and 2.9%, 12.6%, 28.8%, 31% , 4.4% respectively for LL. Wilson et al. (2004) studied diseases in 2 large commercial Holstein dairy herds in New York State, provided with standard procedures for diagnosis. The frequency estimates by Zwald et al (2004) were based on producer-recorded health data from commercial Holstein herds located in Northeast, Southeast, South, Midwest, and Great Plains areas of the USA. The frequencies for MAST12, LAME and DA in this study were considerably bigger than that for commercial herds reported by Zwald et al. (2004). Relatively more intensive health management practices in the experimental dairy herds (VT and PSU) might have inflated the incidence rates of these diseases. However, the trends of disease frequencies from FL to LL were similar to many studies. The disease frequencies of MAST1, MAST2, MAST12, MAST1/2 increased, whereas that of MET decreased from FL to LL. Frequencies of KET, LAME and DA were relatively unchanged in FL and LL.

We defined disease frequency as a percentage of total lactations (FL, LL or all) that had at least one incidence of the disease of interest. Therefore, these frequencies can be considered as minimum lactational incidence rates for the diseases such as mastitis because multiple cases can occur in whole lactation or a defined period of lactation. The incidence rates indicate that cows tended to develop mastitis more frequently in late lactation than early lactation. On the other hand, Figure 2.4 presents the distribution of total number of mastitis treatment incidences in our data. This distribution suggests that the frequency of mastitis was greater in early lactation than late lactation as observed by Wilson et al. (2004) and Hinrichs et al. (2005).

### **Effect of diseases on persistency, peak yield and DIM at peak**

Overall, the effects of mastitis (MAST1, MAST2, and MAST1/2) on persistency appeared to be more substantial in multiparous cows than primiparous cows. The weak positive effect of MAST1 in primiparous cows became much stronger in multiparous cows. Moreover, the negative effect of MAST2 in primiparous cows became more significant in multiparous cows. The corresponding disease frequencies (Table 2.2) indicate that multiparous cows tended to develop mastitis more frequently than primiparous cows. Estimated correlations between persistency and DIM at peak (0.70 in FL and 0.82 in LL) indicate that lactations, associated with delayed peak yield are likely to be more persistent (Muir et al., 2004). The positive relationships of MAST1 to persistency suggest that health disorders in early lactation may suppress increasing rate of milk secretion and prevent milk yield from reaching its maximum level soon. This may allow cows to utilize their energy reserves slowly and efficiently while maintaining their production without developing a rapid decline after peak (Ferris et al., 1985). On the other hand, MAST2 did not get the opportunity to control peak yield so as MAST1, METAB, MET, and DA. However, we observed an association between MAST2 and early DIM at peak, which was significant in multiparous cows. Early peak production results in a severe negative energy balance that may down regulate the energetic process maintaining general health status of cows (Collard et al., 2000). Hence, one can argue that mastitis tends to occur in lactations associated with early DIM as they create a favorable environment for it to occur. Nonetheless, MAST2 had significantly negative

effects on persistency in both FL and LL. Capuco et al. (2003) noted that “increased milk yield during early lactation appeared mainly due to increased secretory activity per cell but the decline in milk yield after peak was solely associated with decreased cell number due to apoptosis”. These authors also noted that mastitis could enhance mammary cell apoptosis. Therefore the impact of mastitis on persistency can be negative when it occurs after peak. The significant negative effect of MAST12 on persistency indicates that when mastitis occur in both early and late stages of lactation the effect of later is more likely to be stronger.

The positive effect of METAB on persistency tended to be more substantial in multiparous cows. The incidence rate of METAB was greater in multiparous cows than primiparous cows (13% vs. 10%). The significantly positive impact of the other periparturiant diseases (DA and MET) indicate that illness in early lactation tend to produce more persistent lactations. In connection to this contention, Muir et al. (2004) reported that Canadian Holstein heifers that had a difficult first calving had more persistent first lactations while Harder et al. (2006) reported post partum metabolic diseases (KET, MKFV and DA) appeared to increase persistency in German Holstein cows. Moreover, we observed that cows experiencing periparturiant diseases tended to have low peak yields and late DIM to peak. However the relationship between DIM at peak and persistency was much stronger than that between persistency and peak yield, reflecting that periparturiant diseases positively affect persistency more by delaying DIM at peak than reducing peak yield. This observation suggests that diseases cause persistency, rather than the other way around, at the phenotypic level.

Table 2.4 shows that majority of disease traits did not have a significant impact on peak yield. The antagonistic relationship between disease resistance and total production (Simianer et al., 1991) suggests that high producing cows are more susceptible to health disorders than low producing cows. Wilson et al. (2004) noted that even after contracted with diseases, milk yield of high producing sick animals can be better or similar to their healthy low producing herd mates. In this context, the stronger correlation between peak yield and 305 d production (0.91 in FL and 0.96 in LL in our data) shows that difference in peak yields between sick and healthy cows could also be non-significant. However, we estimated persistency to be uncorrelated with 305 d yield. Thus, the phenotypic

relationships between persistency and diseases in this study are independent of the antagonistic association between level of production and diseases. The phenotypic relationships of LAME to peak yield, DIM at peak and persistency seem to be very weak. But from a genetic relationships view point, Harder et al. (2006) found LAME to be much more strongly correlated with milk yield persistency.

### **Effect of persistency on probability of disease occurrence**

Overall, the effect of persistency on mastitis is more pronounced in primiparous cows than multiparous cows. Increasing persistency tended to reduce the likelihood of MAST2 in current lactation of primiparous cows. Furthermore, the primiparous cows with more persistent lactation were less likely to develop mastitis in any stage of the lactation (MAST1/2). We examined the question of the effect of persistency in previous lactation on the likelihood of diseases in present lactation. However, none of the disease traits were affected by the persistency in previous lactation. Thus, no such relationship could be established in these data. Nevertheless, majority of disease traits had significant effects on persistency. One general conclusion is that many common health traits in dairy cows tend to significantly affect persistency, while the occurrence of many diseases is not much affected by increasing persistency. The relationship between LAME and persistency was not strong, regardless of whether we treated it as a causative factor or a result of persistency. Frequency of lameness in these data was over twice that of some estimates in literature (i. e., Zwald et al., 2004). Perhaps a more restricted definition of LAME would produce different results. Furthermore, we defined disease traits considering only the presence or absence of at least one incidence of the disorders. Hence, the estimated relationships in this study do not account for the severity and the repeated incidence of diseases such as mastitis and lameness (Domecq et al., 1997). Investigations that define disease traits accounting for these factors may also produce different results.

## **CONCLUSIONS**

Mastitis in early lactation tends to significantly increase persistency in multiparous cows. Conversely, both primiparous and multiparous cows that develop mastitis in late lactation have significantly less persistent lactations. Effect of mastitis in both early and late lactation on persistency is significant but negative across parities. Irrespective to time of occurrence, mastitis tends to reduce milk yield persistency. Postpartum metabolic diseases, displaced abomasum, and metritis have significantly positive effects on persistency and tend to force peak yield to occur at later DIM.

More persistent primiparous cows tend to develop mastitis less frequently in late stage of lactation. Persistency has no significant association with likelihood of diseases in next lactation. Overall, this study suggests that diseases tend to significantly affect lactation persistency compared to the anticipated influence of persistency on disease occurrence. Relationships in this study are phenotypic. Inclusion of persistency in the breeding goal to improve disease resistance needs to be based on genetic relationships.

## **ACKNOWLEDGEMENTS**

The authors are grateful for financial support provided by AIPL, USDA and health and daily milk weight data received from Dr. Chad Dechow at Pennsylvania State University.

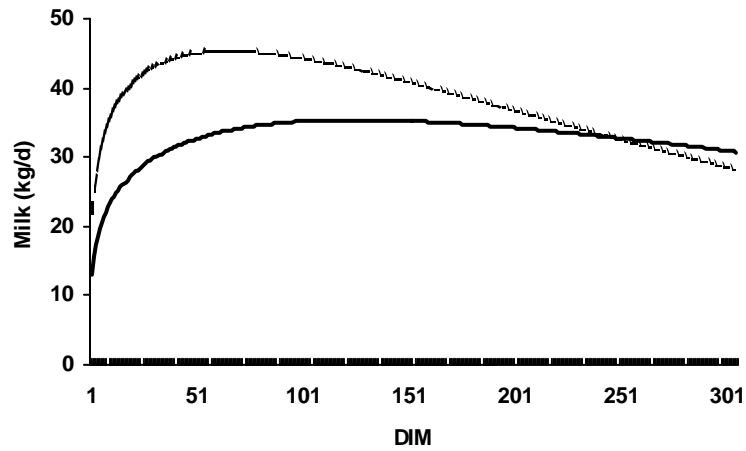
## REFERENCES

- Capuco, A. V., S. E. Ellis, S. A. Hale, R. A. Edman, X. Zhao, and M. J. Paape. 2003. Lactation persistency, insights from mammary cell proliferation studies. *J. Anim. Sci.* 81: 18-31.
- Carlen, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.*: 87(9): 3062-3070.
- Cole, J. B. and P. M. VanRaden. 2006. Genetic evaluation and best prediction of lactation Persistency. *J. Dairy Sci.* 89: 2722-2728.
- Dekkers, J. C. M., J. H. Ten Haag, and A. Weersink. 1997. Economic aspects of persistency in dairy cattle. *Livest. Prod. Sci.* 53:237-252.
- Domecq, J. J., A. L. Skidmore, J. W. Lloyd, and J. B. Kaneene. 1997. Relationships between body condition scores and conception at first artificial insemination in a large dairy herd of high yielding Holstein cows. *J. Dairy Sci.* 80: 113-120
- Ferris, T. A., I. L. Mao, and C. R. Anderson. 1985. Selection for lactation curve and milk yield in cattle. *J. Dairy Sci.* 68:1438-1448.
- Harder, B., J. Bennewitz, D. Hinrichs, and E. Kalm. 2006. Genetic parameters for health traits and their relationship to different persistency traits in German Holstein dairy cattle. *J. Dairy Sci.* 89:3202-3212.
- Jakobsen, J. H., R. Rekaya, J. Jensen, D.A. Sorensen, P. Madsen, D. Gianola, L.G. Christensen, and J. Pedersen. 2003. Bayesian estimates of covariance components between lactation curve parameters and disease liability in Danish Holstein cows. *J. Dairy Sci.* 86:3000-3007.
- Jamrozik, J., L. R. Schaeffer, and J. L. Dekkers. 1997. Genetic evaluation of dairy cattle using test day yields and random regression model. *J. Dairy Sci.* 80(6): 1217-1226.
- Koenig, S., A. R. Sharifi, H. Wentrot, D. Landmann, M. Eise, and H. Simianer. 2005. Genetic parameters of claw and foot disorders estimated with logistic model. *J. Dairy. Sci.* 88:3316-3325.

- Miglior, F., B. L. Muir, and B. J. Van Doormaal. 2005. Selection indices in Holstein cattle of various countries. *J. Dairy Sci.* 88: 1255-1263.
- Montgomery, D. C., E. A. Peck, and G. G. Vining. 2001. Introduction to linear regression analysis, John Wiley and sons, Inc., New York, NY.
- Muir, B. L., J. Fatehi, and L. R. Schaeffer. 2004. Genetic relationships between persistency and reproductive performances in first-lactation Canadian Holsteins. *J. Dairy Sci.* 87: 3029-3037.
- Scott., T. A., B. Yandell, L. Zepeda, R.D. Shaver, and T.R. Smith. 1996. Use of lactation curves for analyzing of milk production data. *J. Dairy Sci.* 79: 1885-1894.
- Simianer, H., H. Solbu, and L. R. Schaeffer. 1991. Estimated genetic correlations Between diseases and yield traits in dairy cattle. *J. Dairy. Sci.* 74(12): 4358-4365.
- Solkner, J., and W. Funchs. 1987. A comparison of different measures of persistency with special respect to variation of test-day milk yields. *Livest. Prod. Sci.* 16:305-319.
- Tekerli, M., Z.Akinci, I. Dogan, and A. Akcon. 2000. Factors affecting the shape of the lactation curves of Holstein cows from the Bulikesir province of Turkey. *J. Dairy Sci.* 83:1381-1386.
- Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Ketton. 1994. Genetic parameters for common health disorders of Holstein cows. *J. Dairy Sci.* 78:421-430.
- Van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders and relationships with 305-day milk yield and conformation traits of registered Holstein cows. *J. Dairy Sci.* 81:2264–2270
- VanRaden, P. M., 1998. Best prediction of lactation yield and persistency. *Proc. 6<sup>th</sup> World Congr. Genet. Appl. Livest. Prod., Armidale, Australia XXIII:347-350.*
- Wilson, D. J., R. N. Gonzalez, J. Hertl, H. F. Schulte, G. J. Bennett, Y. H. Schukken, and Y. T. Grohn. 2004. Effect of clinical mastitis on the lactation curve: a mixed model estimation using daily milk weight. *J. Dairy Sci.* 87:2073-2084.
- Wood, P.D.R. 1997. Algebraic model of the lactation curve in cattle. *Nature* 216:164-165.

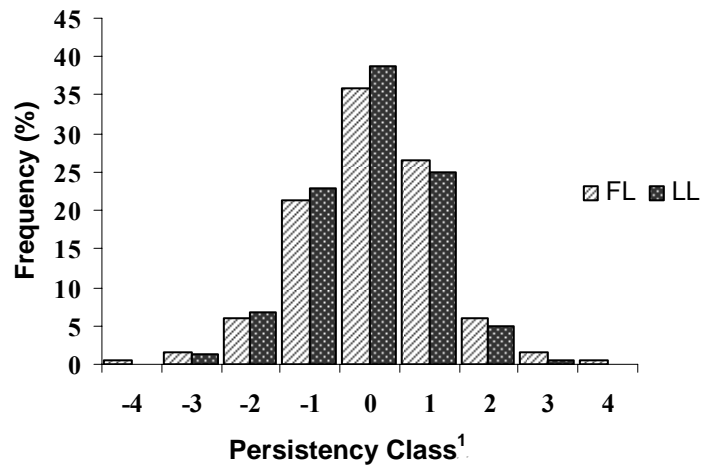
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer recorded data.1. incidence rates, heritability estimates, and sire breeding values. *J. Dairy Sci.* 87:4287-4294.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2006. Genetic analysis of clinical mastitis data from on-farm management software using threshold models. *J. Dairy Sci.* 89(1):330-336.

**Figure 2.1** Standard lactations curves<sup>1</sup> for first (FL) and later lactations (LL) developed from daily milk yields in two experimental herds.



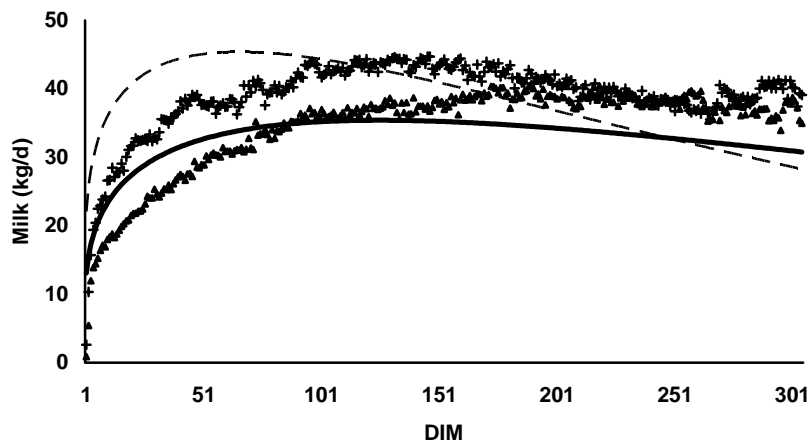
<sup>1</sup>The standard curves for FL (—) and LL (---)

**Figure 2.2** Frequencies of persistency classes in first (FL) and later lactations (LL)



<sup>1</sup>Standardized persistency values were rounded to the nearest integer to form persistency classes

**Figure 2.3a** Comparison of first (FL)<sup>1</sup> and later lactations (LL)<sup>2</sup> of high persistency<sup>3</sup> with corresponding standard lactation curves<sup>4</sup>.



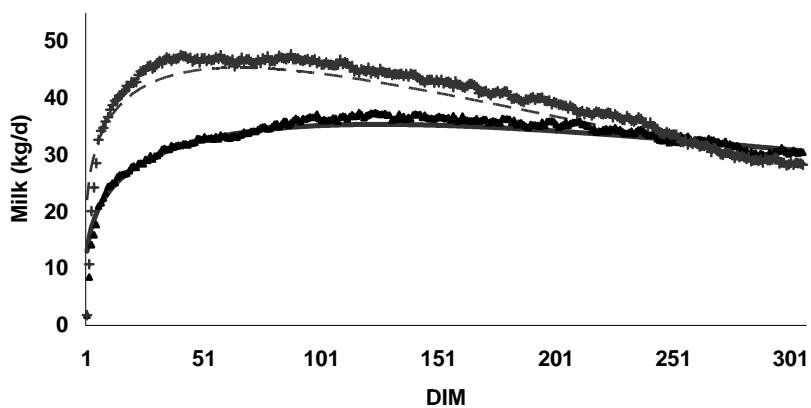
<sup>1</sup>Mean daily milk yields for 25 FL with high persistency (solid triangle)

<sup>2</sup>Mean daily milk yields for 37 LL with high persistency (cross)

<sup>3</sup>Highly persistent lactations included those having standardized persistency values within the range from 1.5 to 2.5

<sup>4</sup>The standard curves for FL (solid line) and LL (broken line)

**Figure 2.3b** Comparison of first (FL)<sup>1</sup> and later lactations (LL)<sup>2</sup> of average persistency<sup>3</sup> with corresponding standard lactation curves<sup>4</sup>.

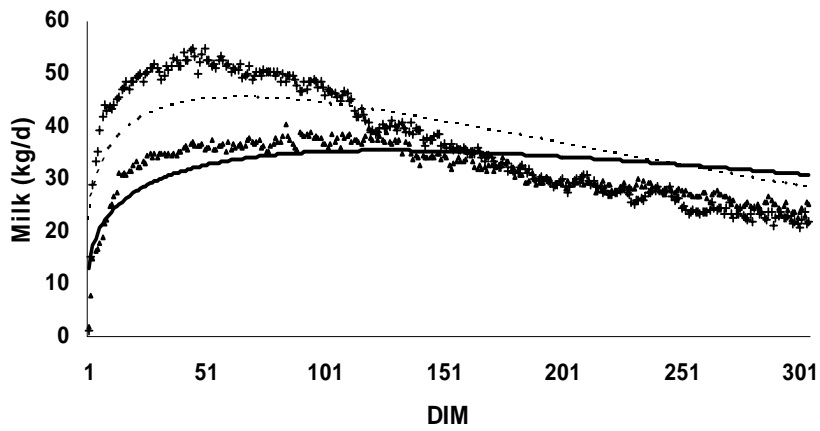


<sup>1</sup>Mean daily milk yields for 157 FL with average persistency (solid triangle)

<sup>2</sup>Mean daily milk yields for 242 LL with average persistency (cross)

<sup>3</sup>Lactations with average persistency included those having standardized persistency values within the range from -0.5 to 0.5, <sup>4</sup>The standard curves for FL (solid line) and LL (broken line)

**Figure 2.3c** Comparison of first (FL)<sup>1</sup> and later lactations (LL)<sup>2</sup> of low persistency<sup>3</sup> with corresponding standard lactation curves<sup>4</sup>.



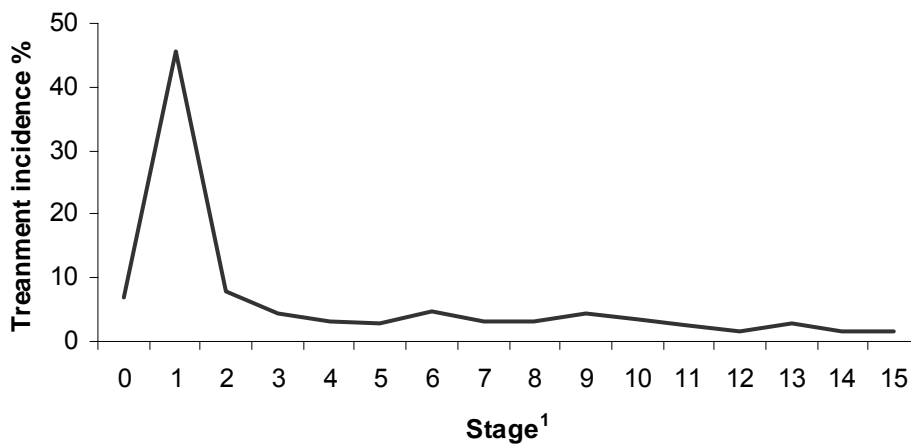
<sup>1</sup>Mean daily milk yields for 20 FL with low persistency (solid triangle)

<sup>2</sup>Mean daily milk yields for 30 LL with low persistency (cross)

<sup>3</sup>Lactations with low persistency included those having standardized persistency values within the range from -2.5 to -1.5

<sup>4</sup>The standard curves for FL (solid line) and LL (broken line)

**Figure 2.4** Distribution of treatment incidence for mastitis over 305 d lactation



<sup>1</sup>Stage, 0 = day of calving, stage 1-15 = 20 d stages

**Table 2.1** Correlations between 305 d yield and persistency calculated for different  $d_0$  in first (FL) and later lactations (LL)

$d_0$	FL	LL
130	-0.063	-0.124
129	-0.025	-0.114
128	0.009	-0.083
127	0.043	-0.056
126	0.084	-0.023
125	0.115	-0.003
124	0.125	0.032

**Table 2.2** Incidence frequencies (%) for the diseases in first lactations (FL), later lactations (LL), and across parities (ALL)

Disease traits <sup>1</sup>	FL		LL		ALL	
	n <sup>2</sup>	%	n	%	n	%
MAST1	24	6.4	78	12.9	102	10.3
MAST2	22	5.8	106	17.5	128	12.7
MAST12	12	3.0	63	10.3	75	7.5
MAST1/2	58	15.2	247	40.7	305	30.5
MET	73	19.1	58	9.5	131	13.2
DA	30	7.9	45	7.4	75	7.6
MKFV	12	3.0	32	5.3	44	4.4
KET	29	7.6	50	8.2	79	8.0
METAB	41	10.6	82	13.5	123	12.4
LAME	94	24.0	164	27.0	258	26.0

<sup>1</sup>Mastitis only before 100 d (MAST1), mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), metritis (MET), displaced abomasums (DA), milk fever (MKFV), ketosis (KET), metabolic diseases (METAB), and lameness (LAME).

<sup>2</sup>Number of lactations had at least one incidence of the disease

**Table 2.3** Least Square Means (LSM) of persistency and significance (p-value) of the effect of each disease trait on persistency in first (FL) and later lactations (LL)

Disease trait <sup>1</sup>	FL			LL		
	LSM		p-value	LSM		p-value
	0 <sup>2</sup>	1 <sup>3</sup>		0	1	
MAST1	-0.051	0.122	0.341	-0.124	0.171	0.021
MAST2	0.021	-0.472	0.031	-0.001	-0.371	0.002
MAST1/2	-0.058	-0.187	0.379	0.014	-0.279	0.023
MAST12 <sup>4</sup>	-0.068	-0.417	0.007			
METAB	-0.34	-0.276	0.649	-0.118	0.3	0.0001
MET	-0.388	-0.076	0.0008	-0.128	0.322	0.0002
DA	-0.355	0.296	<0.0001	-0.117	0.453	<0.0001
LAME	-0.283	-0.44	0.251	-0.092	-0.201	0.263

<sup>1</sup>Mastitis only before 100 d (MAST1), mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), metabolic diseases (METAB) metritis (MET), displaced abomasums (DA), and lameness (LAME)

<sup>2</sup>LSM for lactations with no incidence of the disease

<sup>3</sup>LSM for lactations with at least one incidence for the disease

<sup>4</sup>Estimated across parities

**Table 2.4** Least Square Means (LSM) of peak yield (kg/d) and significance (p-value) of the effect of each disease trait on peak yield in first (FL) and later lactations (LL)

Disease trait <sup>1</sup>	FL			LL		
	LSM		p-value	LSM		p-value
	0 <sup>2</sup>	1 <sup>3</sup>		0	1	
MAST1	33.1	32.3	0.708	38.1	36.7	0.569
MAST2	32.3	32.9	0.804	37	41.6	0.021
MAST1/2	32.2	34.7	0.071	38.6	37.6	0.551
MAST12 <sup>4</sup>	36.5	33.6	0.103			
METAB	32.9	30.1	0.091	38.6	37.8	0.753
MET	33.3	32.6	0.571	38.2	39.3	0.404
DA	33.5	29.3	0.026	39.5	38.3	0.324
LAME	32.9	32.7	0.881	38.6	40.7	0.202

<sup>1</sup>Mastitis only before 100 d (MAST1), mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), metabolic diseases (METAB) metritis (MET), displaced abomasums (DA), and lameness (LAME)

<sup>2</sup>LSM for lactations with no incidence for the disease

<sup>3</sup>LSM for lactations with at least one incidence of the disease

<sup>4</sup>Estimated across parities

**Table 2.5** Least Square Means (LSM) of DIM at peak and significance (p-value) of the effect of each disease trait on DIM at peak in first (FL) and later lactations (LL)

Disease trait <sup>1</sup>	FL			LL		
	LSM		p-value	LSM		p-value
	0 <sup>2</sup>	1 <sup>3</sup>		0	1	
MAST1	138	147	0.594	68	72	0.420
MAST2	141	107	0.081	73	68	0.246
MAST1/2	140	131	0.427	77	63	0.003
MAST12 <sup>4</sup>	106	82	0.004			
METAB	137	167	0.004	69	85	0.003
MET	136	151	0.125	67	92	<0.0001
DA	139	152	0.327	69	96	0.002
LAME	108	110	0.599	64	66	0.533

<sup>1</sup>Mastitis only before 100 d (MAST1), mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), metabolic diseases (METAB) metritis (MET), displaced abomasums (DA), and lameness (LAME)

<sup>2</sup>LSM for lactations with no incidence of the disease

<sup>3</sup>LSM for lactations with at least one incidence for the disease

<sup>4</sup>Estimated across parities

**Table 2.6** Means and phenotypic correlations (r) for 305 d yield (305-Y), peak yield (PY), DIM at peak (DIMP), and persistency in first (FL) and later lactations (LL)

Trait	Mean		r <sup>1</sup>			
	FL	LL	Yield	PY	DIMP	Persistency
Yield (kg)	10257	11889		0.96	0.23	0.009
PY(kg/day)	37.2	47.2	0.91		0.14	-0.04
DIMP	115	67	0.01	-0.01		0.70
Persistency	0	0	-0.003	-0.13	0.82	

<sup>1</sup>FL (above diagonal) and LL (below diagonal)

**Table 2.7** Odds ratios and 95% confidence intervals (CI) for the effect of persistency on probability of disease occurrence in current lactation

Disease <sup>1</sup>	FL		LL	
	Odds ratio	CI	Odds ratio	CI
MAST2	0.46	0.30 – 0.72	0.77	0.61 – 0.97
MAST12	0.87	0.48 – 1.60	0.70	0.53 – 0.95
MAST1/2	0.59	0.42 – 0.83	0.82	0.65 – 1.03
LAME	0.95	0.74 – 1.24	0.94	0.77 – 1.15

<sup>1</sup>mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), and lameness (LAME)

**Table 2.8** Odds ratios and associated 95% confidence intervals (CI) for the effect of persistency on probability of disease occurrence in next lactation.

Disease trait <sup>1</sup>	Odds ratio	CI
MAST1	1.13	0.96 - 1.33
MAST2	0.96	0.84 - 1.09
MAST12	1.17	0.99 - 1.39
MAST1/2	1.08	0.96 - 1.21
METAB	1.13	0.98 - 1.32
MET	1.04	0.91 - 1.19
DA	1.00	0.88 - 1.16
LAME	0.87	0.78 - 1.01

<sup>1</sup>Mastitis only before 100 d (MAST1), mastitis only after 100 d (MAST2), mastitis before and after 100 d (MAST12), mastitis at any time of lactation (MAST1/2), metabolic diseases (METAB) metritis (MET), displaced abomasums (DA), and lameness (LAME)

**CHAPTER IV**  
**Manuscript 2**

# **Investigation of Phenotypic Relationships between Common Health Disorders and Milk, Fat, and Protein Persistencies Using Producer Recorded Health Data and Test Day Yields**

## **ABSTRACT**

The objective of this study was to investigate the phenotypic relationships of common health disorders in dairy cows to milk, fat, and protein yield persistencies (PM, PF, and PP respectively), uncorrelated with yield. Producer recorded health data from 87555 lactations in 398 herds were used to define disease traits for mastitis only in the first 100 d of lactation (MAST1), only after 100 DIM (MAST2) and at any stage of lactation (MAST), and reproductive disorders (REPRO), metabolic disorders (METAB) and lameness (LAME). Each disorder was defined as a binary trait distinguishing between lactations with at least one reported case (1) and lactations with no cases (0). Standardized PM, PF and PP were calculated as a function of test day (TD) yield deviations from a trait specific standard curve and TD DIM deviations around trait specific reference dates of 128, 146, and 155 DIM for milk, fat, and protein respectively. Effects of diseases on the persistencies were investigated separately for first (FL) and later (LL) lactations. Effects of the persistencies on likelihood of diseases in current lactation (in both FL and LL) and next lactation were examined using odds ratios from a logistic regression model. METAB and REPRO had significantly positive effects on PM, PF, and PP in both FL and LL. Multiparous cows positive for MAST1 tended to have significantly higher PM and PF. MAST2 had a significant but negative impact on PM, PF, and PP in both FL and LL while MAST tended to significantly reduce PM, PF, and PP in FL and only PP in LL. Probability of MAST and MAST2 decreased to approximately 0.1 to 0.2 times for each SD unit increase in persistency of present lactation and previous lactation. Occurrence of REPRO and METAB were not affected by persistencies in previous lactation. Phenotypically, LAME seemed to have no association with PM, PF, or PP.

**(Key words:** disease, fat, milk, persistency, phenotypic relationship, protein)

## INTRODUCTION

The antagonistic relationship between advanced production and disease resistance (Semianer et al., 1991) seems to be associated with increased disease incidence rates in dairy cows (Zwald et al., 2004a). Higher susceptibility to diseases is not desirable, as it leads to a considerable financial loss, besides its adverse impact on animal welfare (Jakobsen et al. 2003). Many dairy producing countries are placing more emphasis on health relative to production in their selection programs (Miglior et al., 2005). However, direct selection of dairy cattle for disease traits is impossible in many countries including USA as health data recording practices are not standardized across farms and mechanisms for routine retrieval of such data do not exist (Zwald et al., 2004b).

Some investigators have looked to indirect selection for disease resistance using correlated traits such as productive life (Rogers et al., 1999) and body condition score (Dechow et al., 2004). Moreover, Jakobsen et al. (2002), Muir et al. (2004) and Harder et al. (2006) suggested that selection for more persistent lactations could be used to lower the likelihood of health disorders in dairy cows. Besides anticipated improvement in disease resistance, persistent lactations tend to be more profitable as they incur less feed cost and generate more return from milk when lactations extend beyond 305 d (Dekkers et al., 1998).

Lack of clear consensus on how best to define persistency is a key issue in genetic evaluation for lactation persistency (Cole and VanRaden, 2006). Many available persistency measures are negatively correlated with 305 d yield (Dekkers et al., 1998, Jakobsen et al., 2003, Muir et al., 2004) suggesting selection for higher persistency would occur at the expense of total yield. A persistency measure that is independent of yield will therefore allow more efficient selection for total yield and persistency simultaneously (Muir et al., 2004).

Cole and VanRaden (2006) suggested a method to estimate persistency, uncorrelated with yield. We performed a preliminary investigation (unpublished) for this method and found a phenotypic correlation of 0.96 between persistencies calculated using daily milk weights and monthly milk weights. This suggests that test day (TD) milk can be used to estimate persistency satisfactorily. The objective of this study was to investigate the phenotypic relationships of milk, fat, and protein yield persistencies to

common health disorders in Holstein cows using producer recorded health data and TD yield records.

## **MATERIALS AND METHODS**

### **Data**

Health data and estimated persistency for milk (PM), fat (PF) and protein (PP) yields of 90237 lactations with calving between January 1, 1997 and June 1, 2002 in 405 herds were obtained from Animal Improvement Program Laboratory (AIPL), USDA (Beltsville, MD). Health data were from Dairy Record Management Systems (DRMS), Raleigh, NC.

All lactations in the data did not include records for all diseases because some diseases were not recorded in some herd-years. Lactations that had records (presence=1 or absence=0 of the disease) for at least two diseases were first chosen from the data. Although information on lactation number was given, a general restriction of age at calving was constructed to exclude lactations with wrong lactation number (Carlen et al., 2004). The defined minimum and maximum ages for first, second, third, and fourth calving were 20 to 38, 32 to 52, 43 to 70, and 54 to 88 mo respectively. The fifth calving at <66 mo were deleted from the data. Edited data included 87555 lactations (43232 first (FL) and 44323 later lactations (LL)) from 398 herds. Table 3.1 presents some summary statistics of the edited data.

### **Defining disease traits**

Disease traits regarding mastitis, metritis, retained placenta, cystic ovaries, ketosis, displaced abomasum and lameness were developed as they were recognized as common health disorders in dairy cows (Zwald et al, 2004). We chose to consider mastitis under two separate stages of lactation: early (before 100 DIM) and late (after 100 DIM), because Zwald et al. (2004) reported that mastitis in early lactation is lowly correlated with mastitis in late lactation. Three disease traits were formed with respect to mastitis: MAST, MAST1, and MAST2 representing mastitis, at any stage, only in the early stage, and only in the late stage respectively. A disease variable for lameness (LAME) was formed considering treatment incidences for foot rot, hoof abscess, heel

warts, laminitis and other causes of lameness. Health events pertaining to metritis, retained placenta and cystic ovaries were pooled to form a disease trait, reproductive disorders (REPRO). Treatment incidences for both ketosis (KET) and displace abomasum (DA) were considered together to form one disease trait, metabolic diseases (METAB). Each disease trait was defined as a binary trait distinguishing between cows with at least one reported incidence during the defined period (1) and cows without cases (0) (Carlen et al., 2004).

### **Disease incidence**

Disease incidence was expressed as lactational incidence rate (LIR):

$$\text{LIR} = \frac{\text{Number of lactations with at least one incidence of the disease} \times 100}{\text{Total number of lactations at risk}}$$

Lactations, during which the disease of interest was recorded, were chosen as the total number of lactations at risk of the disease, i. e, only 49220 lactations in 226 herds were initiated in herd-year when at least one disease for KET and DA was recorded. Thus, 49220 were at risk of METAB (across parities). Similarly, 58118 lactations from 260 herds, 43168 lactations from 193 herds, 77106 lactations from 342 herds were at risk of MAST, LAME and REPRO respectively (Table 3.2).

The data included dates for the first disease incidence (but no dates for subsequent incidences) and total number of the incidence (n) in an individual lactation. We could identify the lactations that had first mastitis incidence before 100 DIM. In defining MAST1 as a binary trait, lactations with only one mastitis incidence (n=1), occurred before 100 DIM were considered as MAST1=1 (Table 3.3). Lactations that had first occurrence of mastitis before 100 DIM and with multiple incidences (n>1) were deleted because we could not identify the timing of second and later incidence. As Table 3.3 shows, rest of the lactations (that had first mastitis incidence after 100 DIM) were considered as MAST1=0. After editing, 56115 lactations from 260 herds were left as the number of lactations at the risk of MAST1. Lactations with first mastitis incidence after 100 DIM were considered to be positive for mastitis only in late lactation (MAST2=1).

### Calculation of persistency

PM, PF and PP estimates were received from AIPL, USDA. These estimates have been calculated based on the method proposed by Cole and VanRaden (2006). According to this method persistency can be calculated by multiplying test-day (TD) deviations from a trait specific (milk, fat and protein) standard lactation curve by TD DIM deviations around a trait specific reference date,  $d_0$ .

$$p = \sum_{i=1}^n (Y_i - S_i) \times (d_i - d_0)$$

Where,  $p$  = PM, PF or PP of an individual lactation  
 $Y_i$  =  $i^{\text{th}}$  TD milk, fat or protein yield  
 $S_i$  = milk, fat or protein yield from the standard lactation curve on  $i^{\text{th}}$  TD  
 $d_i$  = DIM at  $i^{\text{th}}$  TD  
 $d_0$  = reference DIM for milk, fat or protein  
 $n$  = total number of TD yield records used to calculate the persistency.

Let  $\mathbf{t}$  represent the vector whose elements are TD yield deviations ( $Y_i - S_i$ ) and  $\mathbf{q}$  represent the vector whose elements are TD DIM deviations ( $d_i - d_0$ ). Then,

$$\text{persistency} = \mathbf{q}'\mathbf{t}$$

If  $\mathbf{d}$  is the vector with elements,  $d_i$ , then  $\mathbf{q} = \mathbf{d} - \mathbf{1}d_0$  and Persistency =  $(\mathbf{d}' - \mathbf{1}'d_0) \mathbf{t}$

The reference date,  $d_0$ , is a constant that acts as a balance point between yields in early and late lactation and makes persistency uncorrelated with yield. Therefore  $d_0$  was obtained by setting Cov ( $\mathbf{q}'\mathbf{t}$ ,  $\mathbf{1}'\mathbf{t}$ ) to 0 and solving for  $d_0$ :

$$\text{Cov} (\mathbf{q}'\mathbf{t}, \mathbf{1}'\mathbf{t}) = \mathbf{q}'\mathbf{V}\mathbf{1} = (\mathbf{d}' - \mathbf{1}'d_0) \mathbf{V}\mathbf{1} = \mathbf{d}'\mathbf{V}\mathbf{1} - \mathbf{1}'\mathbf{V}\mathbf{1}d_0 = 0$$

$$d_0 = \mathbf{d}'\mathbf{V}\mathbf{1} / \mathbf{1}'\mathbf{V}\mathbf{1}, \text{ where } \mathbf{V} \text{ is the variance of } \mathbf{t} \text{ (Cole and VanRaden, 2006).}$$

AIPL developed trait specific standard curves for each herd and used 128, 146, and 155 DIM as the reference dates for milk, fat and protein yields respectively. Standardized estimates ( $\hat{s}$ ) of PM, PF, and PP were obtained by subtracting the corresponding within-herd mean ( $\mu_p$ ) and dividing by the within-herd standard deviation (SD) of the persistencies ( $p$ ).

$$\hat{s} = \frac{p - \mu_p}{SD}$$

Positive values of  $\hat{s}$  indicate increased persistency relative to an averagely persistent cow in a herd and negative values of  $\hat{s}$  indicate decreased persistency. Persistencies that exceeded  $\pm 4.00$  were limited to an absolute value of 4 (Cole and VanRaden, 2006).

### Statistical Analysis

We examined the phenotypic relationships of the disease traits to persistencies in two ways: first, we evaluated effects of the diseases on the persistency and then, the effect of persistency on probabilities of disease occurrence.

**Effects of diseases on PM, PF and PP:** The following generalized linear model was used to investigate the effect of each disease trait on persistency.

$$Y_{ijkl} = \mu + \text{HYS}_i + D_j + O_k + \beta_1 \text{DO}_{ijkl} + \beta_2 \text{AGE}_{ijkl} + e_{ijkl}$$

Where

Y = standardized PM, PF, or PP

$\mu$  = overall mean of PM, PF, or PP

HYS = fixed effect of Herd-Year-Season [calving years: 1997, 1998, 1999, 2000, 2001, and 2002 and seasons: 1(January– April), 2 (May –August), and 3 (September– December)]

D = fixed effect of the main disease of interest

O = fixed effect of the other diseases, besides main disease of interest

$\beta_1$  = regression for days open (DO)

$\beta_2$  = regression for age at calving (AGE) in mo

$e_{ijklmn}$  = residual error  $\sim N(0, I\sigma_e^2)$ .

The variable other diseases (O) included respiratory diseases, dystocia, fever and the other defined disease traits besides the main disease trait of interest, i. e, the other diseases for MAST1 were MAST2, REPRO, METAB, LAME, respiratory diseases, dystocia, and, fever. The trait “O” was also defined as a binary variable distinguishing between lactations with at least one incidence of any other diseases (1) and lactations with no any other disease incidence. Days open less than 50 were set to 50, and days open greater than 250 were set to 250 (Cole and VanRaden, 2006).

Primiparous cows differ from multiparous cows as they have different incidence rates for many diseases (Oltenucu et al, 1990). Jumrozic et al (1998) suggested that persistency in different lactations can be considered as different traits. Hence, separate analyses were performed to FL and LL. Additionally, the correlation between each disease and persistency was estimated. We included DO in the statistical model to account for the negative effect of pregnancy on persistency (Capuco et al., 2003). AGE accounted for some parity differences in LL and the negative correlation between persistency and age of heifers at breeding (Muir et al., 2004).

**Effect of PM, PF and PP on probability of disease occurrence:** We examined the effect of persistency on likelihood of the diseases in same lactation as well as next lactation through odds ratios and associated confidence intervals (CIs) from a linear logistic model.

Odds ratios measure the strength of associations between independent and dependent variables. An odds ratio can be interpreted as the estimated increase in the dependent variable in response to a unit increase in an independent variable (Montgomery et al, 2001). An odds ratio of 1.0 suggests no association between the dependent variable and the independent variable of interest.

We computed several logistic regression models, including indicator variables for class effects such as herd, year of calving, season of calving and presence or absence of the other diseases, linear and quadratic effects of persistency, days open, cow age at calving, and interactions among independent variables. We removed the non-significant regression coefficients from the initial model based on Type 3  $\chi^2$ -statistics for likelihood ratios at  $p < 0.1$  given by GENMOD procedure in SAS (1999) (Montgomery et al, 2001). The following regression model was chosen to be the most appropriate for all disease traits.

$$\eta_{ijkl} = \beta_0 + \beta_1 S_{2i} + \beta_1 S_{3i} + \beta_2 YR_{2j} + \beta_3 YR_{3j} + \beta_4 YR_{4j} + \beta_5 O_{1k} + \beta_6 P_{ijkl} + \beta_7 AGE_{ijkl} + \beta_8 DO_{ijkl}$$

Where,  $\eta$  = logit of observing the disease

$\beta_0$  = intercept

$S_2$  = effect of season 2 (May –August)

$S_3$  = effect of season 3 (September-December)

$YR_2$  = effect of calving year 1999

$YR_3$  = effect of calving year 2000

$YR_4$  = effect of calving year 2001

$O$  = effect of other diseases

$P$  = effect of standardized persistency

$AGE$  = effect of age (mo) at calving

$DO$  = effect of days open.

Effects of persistency on probability of disease occurrence ( $Y=1$ ) were investigated in terms of the corresponding odds ratios. The significance of the odds ratio was determined, based on its 95% confidence interval (CI). CI including 1 indicates a non-significant change in probability of the disease in response to increasing persistency. The effect of persistency on the probabilities of MAST2, MAST, and LAME in same lactation was examined separately for FL and LL. We used the same logistic model with addition of DO in previous lactation to investigate the effect of persistency on likelihood of disease in the next lactation. Data included 9035 cows from 184 herds for MAST1, 10184 cows from 186 herd for MAST2 and MAST, 14500 cows from 253 herds for REPRO, 7562 cows from 137 herds for LOCO, and 8088 cows from 157 herds that had both first and second lactations.

## RESULTS AND DISCUSSION

Table 3.2 shows that most of the herds (86%) tended to record reproductive disorders (REPRO) while the ketosis (KET) was recorded by only 30% of total herds in the data. Zwald et al. (2004) noted that fewer commercial Holstein herds recorded KET and LAME in their data. Difficulties in uniform diagnosis and high variation of the threshold level for veterinary treatment might have restricted the recording of these diseases in commercial herds. Furthermore Zwald et al. (2004) noted that the herd management software might also influence the health data recording in dairy farms. According to these authors, herds that use DAIRY COMP 305 and PCDART are more likely to record health events.

Table 3.2 presents the LIR of 6.2, 5.0, 11.2, 15.3, 19.0, 5.6, 3.9, and 6.8% (across parities) for MAST1, MAST2, MAST, LOCO, REPRO, KET, DA, and METAB respectively. It is difficult to compare disease incidence across different studies as procedures for diagnosis, data recording methods, and disease trait definitions vary from study to study (Harder et al., 2006). However, Zwald et al. (2004) reported mean LIR for DA, KET, MAST, LAME and MET (including retain placenta) were 3, 10, 20, 10, and 21% respectively. LIRs for MAST and KET in this study were much smaller (11.2 vs. 20% for MAST and 5.6 vs. 10% for KET) and that for LAME was greater (15.6 vs. 10%). In addition to DRMS (Raleigh, NC), Zwald et al. (2004) obtained health data from Alta Genetics (Watertown, WI) Advantage Progeny Test Program cooperators. However, the trends of disease frequencies from FL to LL in this study were similar to several studies (i.e. Wilson et al., 2004). The disease frequencies of MAST and LOCO increased from FL to LL, while REPRO and METAB were relatively unchanged in FL and LL.

Table 3.4 shows the distribution of first incidence of each disorder according to month of lactation. As expected, most of KET (94%), DA (82%), and REPRO (67%) incidence occurred during the first 30 d of lactation. Zwald et al. (2004) observed quite similar frequencies; 90% for KETO, 79% for DA, and 61% REPRO (excluding cystic ovaries), in first month of lactation. About 36% of MAST incidences occurred in first month of lactation with the rest of the incidences equally distributed. Wilson et al. (2004)

and Hinrichs et al. (2005) observed a similar pattern of mastitis incidence distribution. Incidences of LAME were evenly distributed throughout the lactation.

### **Persistency estimates**

Means for PM, PF and PP in FL and LL are in Table 3.1. The positive mean values indicate that data included many highly persistent cows. The frequency histograms of PM, PF, and PP appear in figure 3.1. The persistency values were rounded to form persistency classes for the histograms (e.g. persistency class 0 included lactations having persistency less than 0.5 and greater than -0.5). Greater frequencies of the positive persistency classes indicate that data included many more highly persistent cows (persistency>0) than lowly persistent cows (persistency<0). The mean PM, and PF were greater in FL than LL, consistent with the general consensus that lactation curves of primiparous cows are more persistent than that of multiparous cows. However, the difference in PP between FL and LL (0.324 vs. 0.347) was small.

### **Effects of diseases on persistencies**

**First lactations (FL):** Table 3.5 presents the least square means (LSM) for PM, PF and PP in FL with (1) or without (0) each health disorder, significance (p-value) of each health disorder on the persistencies, and correlation (Pearson's correlation coefficient) between each health disorder and each persistency trait. Mastitis only in late lactation (MAST2), mastitis at any stage of lactation (MAST), reproductive disorders (REPRO), and metabolic disorders (METAB) had significant ( $p<0.01$ ) effects on PM, PF, and PP in FL. Greater LSM of diseased cows (1) than healthy (0) cows and the positive correlations reflect that primiparous cows exhibiting METAB or REPRO problems tended to have more persistent lactations. As Table 3.4 shows, majority (67%) of METAB and REPRO incidences occurred during first 30 d of lactation. Thus, illness in early lactation of primiparous cows seems to produce more persistent lactations. Muir et al. (2004) and Harder et al., (2006) observed similar relationship with respect to Calving difficulty and post partum metabolic diseases respectively. Furthermore Muir et al. (2004) reported a positive genetic correlation (0.54) between persistency and DIM at peak in FL. Hence,

one can argue that diseases may prevent milk production from reaching its maximum soon and thereby make the lactation more persistent. Ferris et al. (1985) suggested that delayed peak allow cows to utilize energy slowly and efficiently without experiencing a rapid decline of yield in late lactation. On the other hand, the primiparous cows that developed MAST and MAST2 tended to have significantly ( $p < 0.05$ ) lower PM, PF, and PP.

**Later lactations (LL):** Table 3.6 presents the LSM of PM, PF and PP in LL with (1) or without (0) each health disorder, significance (p-value) of each health disorder on persistencies, and correlation (Pearson's correlation coefficient) between each health disorder and each persistency trait in LL. The positive effect of MAST1 on PM, that was non-significant ( $P > 0.05$ ) in FL, was significant ( $p < 0.001$ ) in LL. A sharp peak followed by rapid decline in milk yield often characterizes lactation curves of multiparous cows. Conversely, primiparous cows have more flatter lactation curves representing greater persistency (Stanton et al., 1992). Hence, effect of MAST1 that tends to produce a highly persistent lactation can be more significant in multiparous cows than primiparous cows.

MAST2 significantly reduced PM, PF, and PP in LL indicating multiparous cows contracted with mastitis in late lactation tended to be less persistent than primiparous cows. Mastitis is able to enhance apoptosis of mammary secretory cells that is solely responsible for the decline in milk yield after peak lactation (Capuco et al., 2001). Therefore, the impact of mastitis on persistency can be negative when it occurs in late lactation. Effect of MAST on all persistency traits in multiparous cows was negative, but significant ( $p < 0.05$ ) only on PP. METAB and REPRO had highly significant ( $p < 0.0001$ ) positive effect on all persistencies in LL like with FL. This suggests that both primiparous and multiparous cows that develop periparturiant health disorders tend to have more persistent lactations for milk, fat and protein yields. Non-significant effects of LAME on all persistency traits in both FL and LL indicate that the effect of locomotive diseases on lactation curve shape is almost negligible.

### **Effects of persistency on diseases**

Odds ratios and associated CIs for the effect of persistency on probabilities of diseases in current lactation are presented in Table 3.7. Majority (>66%) of MAST1, METAB and REPRO incidences occurred during first 30 days of lactation (Table 3.4). Phenotypically, the expression of these periparturiant disease traits was under minimal influence of persistency in same lactation. Hence we chose not to include MAST1, METAB and REPRO in the analyses for the effects of diseases on persistencies in same lactation.

**First lactations (FL):** The odds ratios and associated CIs (Table 3.7) show that all three persistencies, PM, PF, and PP, tended to reduce the likelihood that a cow would develop mastitis at any stage (MAST) and mastitis specifically at late lactation (MAST2). The odds ratios (0.88 for MAST and 0.79 for MAST2) indicate that the probabilities of MAST and MAST2 in preparturient cows were reduced by 0.12 and 0.21 times in response to a SD unit increase in PM. Moreover, the corresponding 95% CIs (0.85-0.90 for MAST and 0.75-0.83 for MAST2) suggest relationships of PM to MAST and MAST2 are significant. Similarly, increasing PF and PP tended to reduce the likelihood of MAST and MAST2 in primiparous cows.

**Later lactations (LL):** The odds ratios associated with PM, PF, and PP (0.84, 0.91, and 0.86 for MAST2 and 0.92, 0.97, and 0.90 for MAST) were closer to 1.0 than those in primiparous cows (Table 3.7). This suggests that the impact of persistencies on mastitis is likely to be weaker in multiparous cows than primiparous cows. The odds ratios (0.84 and 0.86) indicate that probability of MAST2 in multiparous cows tended to decrease by 0.16 and 0.14 times respectively for each SD unit increase in PM and PP respectively. The effect of PF on MAST2 was barely significant as upper bound of the CI was relatively closer to 1.0.

Figure 3.2 shows that MAST and MAST2 frequencies (estimated across parities) tended to decrease as PM, PF, and PP increased. Overall, the effect of increasing persistency on MAST2 was greater than MAST. MAST included mastitis incidences in both early and late lactation. Phenotypically, the mastitis incidences in early lactation

(before 100 DIM) are not under control of persistencies in same lactation. Hence, the impact of persistency on MAST is basically on mastitis in late lactation.

Table 3.8 shows the odds ratios and CIs for the effect of the persistency traits on probability of disease in next lactation. The model included effect of DO in previous lactation. Thus impact of the persistency on disease incidence was independent of the influence of reproductive status in previous lactation. The PM, PF and PP tended to reduce the incidence of MAST and MAST2 in next lactation. The odds ratios for MAST2 (0.75, 0.83, and 0.74 respectively) and MAST (0.86, 0.90, and 0.82 respectively) indicate that a SD unit increase in persistency tended to reduce the probabilities of MAST and MAST at least by 0.17 and 0.10 respectively in next lactation. The effects of PM, PF and PP on disease probabilities seem to be similar. This may be because of the high inter-relationships among milk, fat and protein as protein and fat are components of milk. The magnitude of the effect of PM was more often similar to PP than PF. We found a stronger phenotypic correlation of PM to PP (0.92) than to PF (0.72).

The odds ratios near 1.0 and associated CIs reflect that there were no significant associations between the PM, PF, and PP and likelihood of METAB and REPRO in next lactation. On the other hand, we observed highly significant ( $<0.001$ ) effects of these periparturiant diseases on all three persistencies in both primiparous and multiparous cows. One general conclusion is that periparturiant health disorders in dairy cows tend to significantly affect persistency, while their occurrence is not much affected by increasing persistency in current or previous lactation. The probability of LAME was not significantly affected by PM, PF and PP of either same lactation or previous lactation. Moreover we observed Lameness had no significant impact on the persistencies in both primiparous and multiparous cows. Perhaps lameness was not severe enough to affect daily yields in a meaningful way.

## **CONCLUSIONS**

Cows that develop post partum metabolic and reproductive disorders tend to have significantly higher milk, fat, and protein yield persistencies. The positive impact of mastitis in early lactation on persistencies is often significant in multiparous cows. Conversely, mastitis in late stages of lactation has significantly negative impact on persistencies in both primiparous and multiparous cows. Irrespective to a particular stage of lactation, mastitis tends to reduce persistencies significantly in primiparous cows. Increasing persistencies tend to lower the likelihood of mastitis specifically in late stages of current lactation, and to a lesser extent in next lactation. LAME has no phenotypic relationships to persistency. Overall, diseases tend to have stronger effects on milk, fat, and protein persistencies than persistencies on disease occurrence. Except the negative impact of persistencies on likelihood of mastitis, present study does not support the hypothesis that increasing persistency lowers disease susceptibility in dairy cows. Relationships in this study are phenotypic. Investigations into genetic relationships may produce different results.

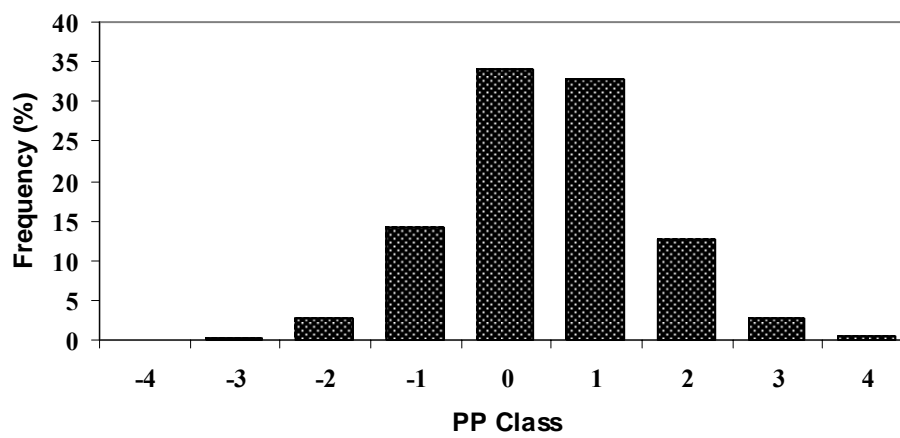
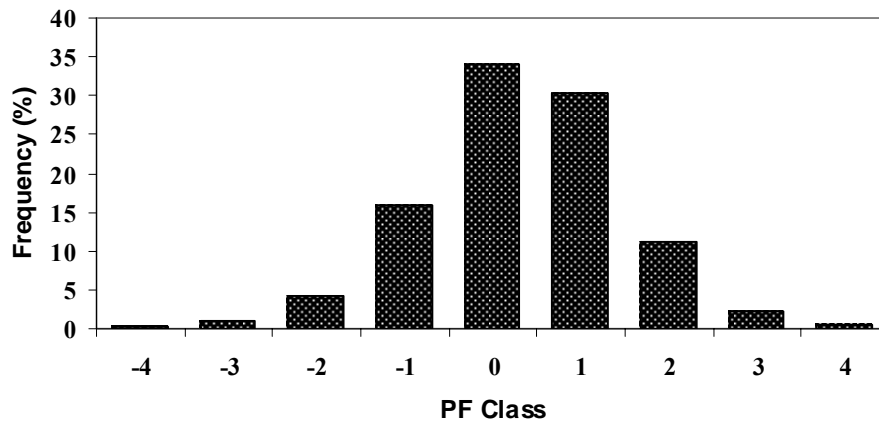
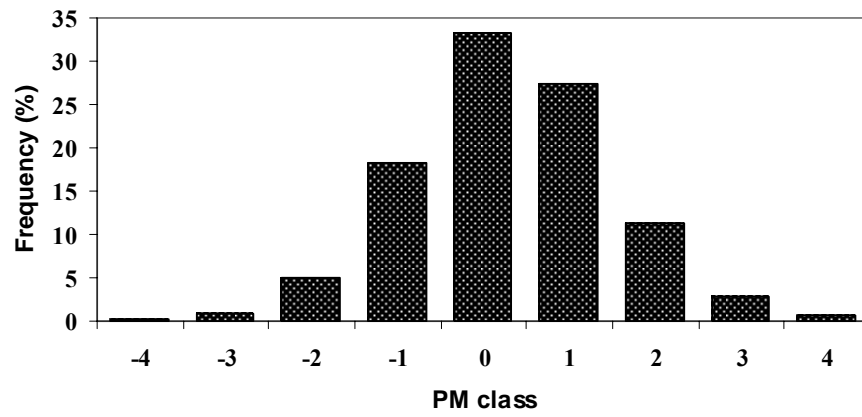
## **ACKNOWLEDGEMENT**

The authors are grateful for financial support and data provided by AIPL, USDA

## REFERENCES

- Appuhamy, J. A. D. R. N., B. G. Cassell, J. B. Cole, and C. D. Dechow. 2006. Phenotypic relationships of common health disorders to lactation persistency estimated from daily milk weights. (in press)
- Capuca, A. V., S. E. Ellis, S. A. Hale, R. A. Edman, X. Zhao, and M. J. Paape. 2003. Lactation persistency, insights from mammary cell proliferation studies. *J. Anim. Sci.* 81: 18-31
- Carlen, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.*: 87(9): 3062-3070.
- Cole, J. B., P. M. VanRaden. 2006. Genetic evaluation and best prediction of lactation Persistency. *J. Dairy Sci.* 89: 2722-2728.
- Dekkers, J. C. M., J. H. Ten Haag, and A. Weersink. 1997. Economic aspects of persistency in dairy cattle. *Livest. Prod. Sci.* 53:237-252.
- Domecq, J. J., A. L. Skidmore, J. W. Lloyd, and J. B. Kaneene. 1997. Relationships between body condition scores and conception at first artificial insemination in a large dairy herd of high yielding Holstein cows. *J. Dairy Sci.* 80: 113-120
- Ferris, T. A., I. L. Mao, and C. R. Anderson. 1985. Selection for lactation curve and milk yield in cattle. *J. Dairy Sci.* 68:1438-1448.
- Harder, B., J. Bennewitz, D. Hinrichs, and E. Kalm. 2006. Genetic parameters for health traits and their relationship to different persistency traits in German Holstein dairy cattle. *J. Dairy Sci.* 89:3202-3212
- Hinrichs., D, E. Steamer, W. Junge, Kalm E. 2005. Genetic analyses of mastitis data using animal threshold models and genetic correlation with production traits. *J. Dairy Sci.* 2005 : 88(6):2260-2268.
- Koenig, S., A. R. Sharifi, H. Wentrot, D. Landmann, M. Eise, and H. Simianer. 2005. Genetic parameters of claw and foot disorders estimated with logistic model. *J. Dairy. Sci.* 88:3316-3325
- Montgomery, D. C., E. A. Peck, and G. G. Vining. 2001. Introduction to linear regression analysis, [John Wiley & Sons, Inc.](#), New York, NY.

- Muir, B. L., J. Fatehi, and L. R. Schaeffer. 2004. Genetic relationships between persistency and reproductive performances in first-lactation Canadian Holsteins. *J. Dairy Sci.* 87: 3029-3037.
- Scott., T. A., B. Yandell, L. Zepeda, R.D. Shaver, and T.R. Smith. 1996. Use of lactation curves for analyzing of milk production data. *J. Dairy Sci.* 79: 1885-1894.
- Simianer, H., H. Solbu, and L. R. Schaeffer. 1991. Estimated genetic correlations Between diseases and yield traits in dairy cattle. *J. Dairy. Sci.* 74(12): 4358-4365
- VanRaden, P. M., 1998. Best prediction of lactation yield and persistency. Proc. 6<sup>th</sup> World Congr. Genet. Appl. Livest. Prod., Armidale, Australia XXIII:347-350.
- Wilson, D. J., R. N. Gonzalez, J. Hertl, H. F. Schulte, G. J. Bennett, Y. H. Schukken, and Y. T. Grohn. 2004. Effect of clinical mastitis on the lactation curve: a mixed model estimation using daily milk weight. *J. Dairy Sci.* 87:2073-2084
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer recorded data.1. incidence rates, heritability estimates, and sire breeding values. *J. Dairy Sci.* 87:4287-4294



**Figure 3.1.** Distribution of standardized milk (PM), fat (PF) and protein (PP) persistencies (frequencies (%)) in each persistency classes were estimated across parities)

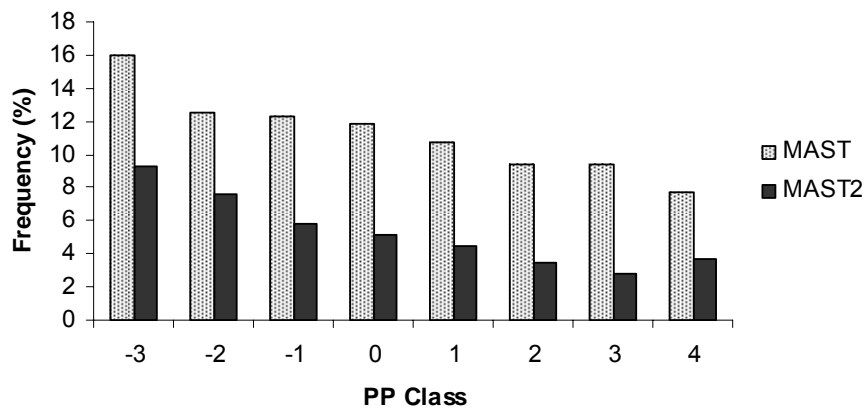
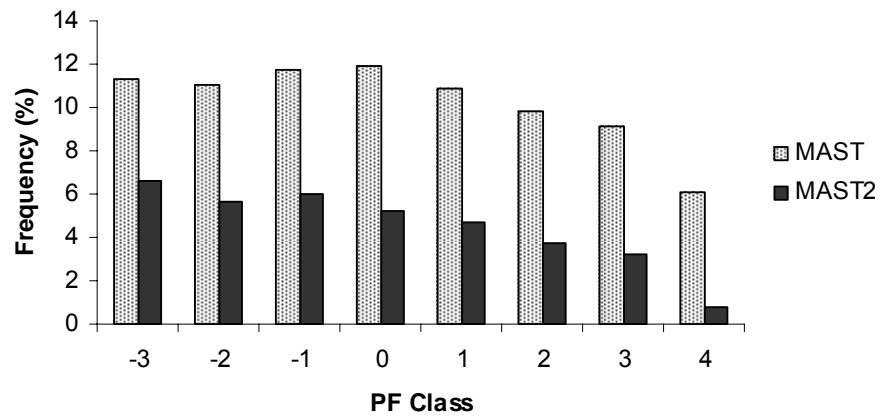
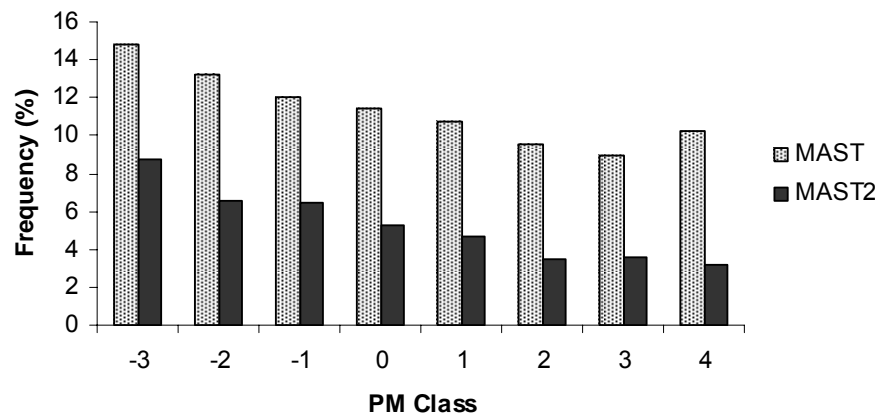


Figure 3.2. Frequency distribution of MAST and MAST2 across PM, PF, and PP classes

**Table 3.1** Summary statistics for first (FL), and later lactations (LL)

Variable	FL		LL	
	Mean	SD	Mean	SD
Milk (kg) <sup>1</sup>	11738	1974	11568	2035
Fat (kg) <sup>2</sup>	433	84	427	88
Protein (kg) <sup>3</sup>	348	55	342	57
DIM <sup>4</sup>	381	110	367	95
DO <sup>5</sup>	166	112	166	101
PM <sup>6</sup>	0.533	1.192	0.430	0.922
PF <sup>7</sup>	0.366	1.330	0.247	1.027
PP <sup>8</sup>	0.324	1.280	0.347	1.009

<sup>1</sup>Total milk yield (kg), <sup>2</sup>Total fat yield (kg), <sup>3</sup>Total protein yield (kg), <sup>4</sup>Length of lactation (days), <sup>5</sup>Days open, <sup>6</sup>Milk yield persistency, <sup>7</sup>Fat yield persistency, <sup>8</sup>Protein yield persistency

**Table 3.2** Lactational Incidence Rates (LIR (%)) for each disease trait in first lactations (FL), later lactations (LL), and all lactations (ALL)

Disease	Herds <sup>2</sup>	N <sup>3</sup>	LIR(%) <sup>1</sup>		
			FL	LL	ALL
MAST1 <sup>4</sup>	260	56115	2.7	3.0	2.9
MAST2 <sup>5</sup>	260	58118	3.9	6.0	5.0
MAST <sup>6</sup>	260	58118	9.5	12.7	11.2
LAME <sup>7</sup>	193	43168	13.3	17.3	15.3
REPRO <sup>8</sup>	342	77106	17.7	20.3	19.0
KET <sup>9</sup>	118	24528	5.2	5.9	5.6
DA <sup>10</sup>	202	44839	4.1	3.7	3.9
METAB <sup>11</sup>	226	49220	6.4	7.0	6.8

<sup>1</sup>LIR= lactations had at least one incidence of the disease trait/ total number of lactations at risk

<sup>2</sup>Number of herds that recorded the disease, <sup>3</sup>Total number of lactations had records for the disease,

<sup>4</sup>Mastitis in only first 100 d of lactation, <sup>5</sup>Mastitis only after 100 DIM, <sup>6</sup>Mastitis at any stage of lactation,

<sup>7</sup>Lameness, <sup>8</sup>Reproductive disorders, <sup>9</sup>Ketosis, <sup>10</sup>Displaced abomasums, <sup>11</sup>Metabolic disorders

**Table 3.3** Coding disease traits for mastitis

Disease trait <sup>1</sup>	FDIM <sup>2</sup> =0 <sup>3</sup>		FDIM<100		FDIM>100
	n <sup>4</sup> =0	n=1	n>1		n>0
MAST1 <sup>5</sup>	0	1	Delete		0
MAST2 <sup>6</sup>	0	0	0		1
MAST <sup>7</sup>	0	1	1		1

<sup>1</sup>each disease trait was defined as a binary variable distinguishing between lactations with at least one reported incidence (1) and lactations without cases (0)

<sup>2</sup>DIM at first incidence

<sup>3</sup>mastitis didn't occur in corresponding lactation

<sup>4</sup>number of mastitis incidence in an individual lactation

<sup>5</sup>mastitis in only during first 100 days of lactation, <sup>6</sup>mastitis only after 100 DIM, <sup>7</sup>mastitis at any stage of lactation

**Table 3.4** Frequencies for the first incidence of health disorders by month of lactation (across parities)

Month	MAST1 <sup>1</sup>	MAST2 <sup>2</sup>	MAST <sup>3</sup>	KET <sup>4</sup>	DA <sup>5</sup>	METAB <sup>6</sup>	LAME <sup>7</sup>	REPRO <sup>8</sup>
1	64.88	--	36.47	93.92	82.65	88.3	11.61	66.54
2	14.75	--	8.42	2.86	5.67	4.27	10	9.42
3	14.19	--	8.29	0.88	1.39	1.14	10.01	6.80
4	6.18	12.81	8.39	0.37	1.04	0.71	11.24	5.51
5	--	16.67	7.35	0.22	1.68	0.95	10.48	3.50
6	--	15.85	6.99	0.29	1.27	0.78	9.89	2.91
7	--	15.56	6.86	0.22	1.56	0.89	9.70	1.75
8	--	14.81	6.53	0.44	1.04	0.74	8.63	1.31
9	--	12.06	5.32	0.29	1.50	0.90	8.47	1.21
10	--	12.24	5.40	0.18	2.20	1.19	9.97	1.02

<sup>1</sup>Mastitis in only first 100 d of lactation, <sup>2</sup>Mastitis only after 100 DIM, <sup>3</sup>Mastitis at any stage of lactation, <sup>4</sup>Ketosis, <sup>5</sup>Displaced abomasums, <sup>6</sup>Metabolic disorders, <sup>7</sup>Lameness, <sup>8</sup>Reproductive disorders

**Table 3.5** Least Square Means (LSM), significance (p-value) for the effect of each disease trait and correlations (r) to disease traits for PM, PF and PP in first lactations (FL)

Persistency trait	Disease trait	LSM		p-value	r <sup>12</sup>
		0 <sup>10</sup>	1 <sup>11</sup>		
PM <sup>1</sup>	MAST1 <sup>4</sup>	0.30	0.44	0.1152	0.01
	MAST2 <sup>5</sup>	0.36	0.09	<0.0001	-0.05
	MAST <sup>6</sup>	0.36	0.28	0.0020	-0.03
	REPRO <sup>7</sup>	0.29	0.46	<0.0001	0.06
	METAB <sup>8</sup>	0.37	0.96	<0.0001	0.13
	LAME <sup>9</sup>	0.33	0.25	0.0900	0.01
PF <sup>2</sup>	MAST1	0.33	0.42	0.0745	0.00
	MAST2	0.35	0.11	<0.0001	-0.04
	MAST	0.36	0.26	<0.0001	-0.03
	REPRO	0.31	0.40	<0.0001	0.03
	METAB	0.34	0.74	<0.0001	0.08
	LAME	0.30	0.25	0.1279	-0.01
PP <sup>3</sup>	MAST1	0.52	0.56	0.2247	0.00
	MAST2	0.56	0.34	0.0013	-0.04
	MAST	0.57	0.46	<0.0001	-0.04
	REPRO	0.42	0.50	<0.0001	0.05
	METAB	0.58	1.08	<0.0001	0.13
	LAME	0.52	0.51	0.6316	-0.02

<sup>1</sup>Persistency of milk yield, <sup>2</sup>Persistency of fat yield, <sup>3</sup>Persistency of protein yield

<sup>4</sup>Mastitis only in first 100 d, <sup>5</sup>Mastitis only after 100 DIM, <sup>6</sup>Mastitis stage of lactation <sup>7</sup>Reproductive disorders, <sup>8</sup>Metabolic disorders, <sup>9</sup>Lameness.

<sup>10</sup>Lactations with no disease incidence

<sup>11</sup>Lactations with at least one incidence

<sup>12</sup>Pearson's correlation coefficient

**Table 3.6** Least Square Means (LSM), significance (p-value) for the effect of each disease trait and correlations (r) to disease traits for PM, PF and PP in later lactations (LL)

trait	Disease trait	LSM		p-value	r <sup>12</sup>
		0 <sup>10</sup>	1 <sup>11</sup>		
PM <sup>1</sup>	MAST1 <sup>4</sup>	0.23	0.39	<0.0001	0.02
	MAST2 <sup>5</sup>	0.26	0.11	<0.0001	-0.04
	MAST <sup>6</sup>	0.26	0.24	0.2412	-0.02
	REPRO <sup>7</sup>	0.21	0.33	<0.0001	0.06
	METAB <sup>8</sup>	0.26	0.71	<0.0001	0.11
	LAME <sup>9</sup>	0.25	0.23	0.4916	0.00
PF <sup>2</sup>	MAST1	0.38	0.49	0.0022	0.01
	MAST2	0.39	0.29	<0.0001	-0.03
	MAST	0.39	0.37	0.3520	-0.01
	REPRO	0.36	0.45	<0.0001	0.04
	METAB	0.37	0.65	<0.0001	0.06
	LAME	0.38	0.36	0.5092	-0.01
PP <sup>3</sup>	MAST1	0.46	0.54	0.0048	0.00
	MAST2	0.48	0.37	<0.0001	-0.03
	MAST	0.48	0.44	0.0124	-0.03
	REPRO	0.44	0.63	<0.0001	0.05
	METAB	0.47	0.59	<0.0001	0.12
	LAME	0.47	0.48	0.3456	-0.01

<sup>1</sup>Persistency of milk yield, <sup>2</sup>Persistency of fat yield, <sup>3</sup>Persistency of protein yield

<sup>4</sup>Mastitis only in first 100 d, <sup>5</sup>Mastitis only after 100 DIM, <sup>6</sup>Mastitis at any stage of lactation <sup>7</sup>Reproductive disorders, <sup>8</sup>Metabolic disorders, <sup>9</sup>Lameness,

<sup>10</sup>Lactations with no disease incidence

<sup>11</sup>Lactations with at least one incidence

<sup>12</sup>Pearson's correlation coefficient

**Table 3.7** Odds ratios and 95% confidence interval (CI) for the effect of the persistency on the likelihood of diseases in first (FL) and later lactations (LL)

Persistency		FL		LL	
trait	Disease trait	Odds ratio	CI	Odds ratio	CI
<b>PM</b> <sup>1</sup>	MAST <sup>4</sup>	0.88	0.85-0.90	0.92	0.88-0.95
	MAST2 <sup>5</sup>	0.79	0.75-0.83	0.84	0.80-0.88
	LAME <sup>6</sup>	0.98	0.95-1.02	1.01	0.98-1.05
<b>PF</b> <sup>2</sup>	MAST	0.9	0.87-0.93	0.97	0.93-1.01
	MAST2	0.83	0.79-0.87	0.91	0.87-0.96
	LAME	1.01	0.97-1.04	0.99	0.95-1.03
<b>PP</b> <sup>3</sup>	MAST	0.85	0.82-0.88	0.9	0.86-0.94
	MAST2	0.79	0.74-0.83	0.86	0.81-0.91
	LAME	1.06	1.02-1.09	1.03	0.99-1.07

<sup>1</sup>Persistency of milk yield, <sup>2</sup>Persistency of fat yield, <sup>3</sup>Persistency of protein yield

<sup>4</sup>Mastitis at any stage of lactation, <sup>5</sup>Mastitis only after 100 DIM, <sup>6</sup>Lameness

**Table 3.8** Odds ratios and associated 95% confidence intervals (CI) related to the effect of each persistency trait on the likelihood of diseases in next lactation

Persistency trait	Disease trait	Odds ratio	CI
PM <sup>1</sup>	MAST1 <sup>4</sup>	1.00	0.89-1.01
	MAST2 <sup>5</sup>	0.75	0.70-0.82
	MAST <sup>6</sup>	0.86	0.82-0.91
	REPRO <sup>7</sup>	1.00	0.97-1.03
	METAB <sup>8</sup>	1.04	0.98-1.12
	LAME <sup>9</sup>	1.00	0.94-1.03
PF <sup>2</sup>	MAST1	0.97	0.78-1.56
	MAST2	0.83	0.76-0.94
	MAST	0.90	0.85-0.95
	REPRO	0.97	0.98-1.01
	METAB	1.03	0.96-1.10
	LAME	0.97	0.93-1.02
PP <sup>3</sup>	MAST1	0.93	0.83-1.04
	MAST2	0.74	0.68-0.82
	MAST	0.82	0.77-0.87
	REPRO	0.96	0.93-1.00
	METAB	1.04	0.96-1.12
	LAME	0.99	0.94-1.04

<sup>1</sup>Persistency of milk yield, <sup>2</sup>Persistency of fat yield, <sup>3</sup>Persistency of protein yield

<sup>4</sup>Mastitis only in first 100 d, <sup>5</sup>Mastitis only after 100 DIM, <sup>6</sup>Mastitis at any stage of lactation <sup>7</sup>Reproductive disorders, <sup>8</sup>Metabolic disorders, <sup>9</sup>Lameness

# **CHAPTER V**

## **General conclusions and implications**

## GENERAL CONCLUSIONS AND IMPLICATIONS

Both primiparous and multiparous cows, which develop postpartum metabolic disorders (ketosis, milk fever, and displaced abomasums) and periparturiant reproductive disorders (metritis, retained placenta, and ovarian cysts) tend to reach peak yield at later days in milk (DIM) and have flatter lactation curves for milk, fat and protein yields. Conversely, mastitis in late lactation (after 100 DIM) tends to produce less persistent lactations characterized by faster rate of decline in milk, fat and protein yields after peak production in both primiparous and multiparous cows. Multiparous cows, which reach peak production at earlier DIM develop mastitis more frequently in late lactation. Mastitis in early lactation (before DIM) tends to produce highly persistent lactations more often in multiparous cows than primiparous cows. However, when mastitis occurs in both early and late stages of lactation, it tends to reduce milk yield persistency. Overall, mastitis incidence is associated with less milk, fat, and protein yield persistencies. Effect of lameness on milk, fat, and protein yield persistencies was negligible.

Cows with more persistent lactations for milk, fat, and protein yields are less likely to develop mastitis specifically in late stages of same lactation, and to a lesser extent in next lactation. The favorable relationship between persistency and likelihood of mastitis in same lactation is much stronger in primiparous cows than multiparous cows. However, no other likelihood of a disease is significantly affected by increasing persistency in both current and previous lactation.

Both types of data; producer recorded health data from commercial farms and health data from experimental herd having intensive health management programs, provided evidence that diseases tend to affect lactation persistency more strongly than the impact of persistency on likelihood of disease. Results of present study do not support the introductory hypothesis that good persistency leads to fewer health problems. However, the relationships in this study are phenotypic. Inclusion of persistency in the breeding goal to improve disease resistance needs to be based on genetic relationships.

The investigations into genetic relationships have to deal with large data sets of test day yields from commercial dairy herds. This study demonstrated that persistency could be satisfactorily measured using monthly test day milk weights from standardized DHA programs.

## BIBLIOGRAPHY

- Appuhamy, J. A. D. R. N., B. G. Cassell, J. B. Cole, and C. D. Dechow. 2006. Phenotypic relationships of common health disorders to lactation persistency estimated from daily milk weights. (in press)
- Bachman, K. C. 2001. Milk production of dairy cows treated with estrogen at the onset of a short dry period. *J. Dairy Sci.* 85: 797-803.
- Baird, G. D. 1982. Primary ketosis in the high-producing dairy cows: clinical and subclinical disorders, treatment, prevention, and outlook. *J. Dairy Sci.* 65(1): 1-10.
- Booth, C. J., L. D. Warnick, Y. T. Grohn, D. O. Maizon, C. L. Guard, and D. Janssen. 2004. Effect of lameness on culling dairy cows. *J. Dairy Sci.* 87: 4115-4122.
- Cassell, B. G. 2001. Using heritability for genetic improvement. <http://www.ext.vt.edu/pubs/dairy/404-084/404-084.html>. Accessed October 21, 2006.
- Capuco, A. V., S. E. Ellis, S. A. Hale, R. A. Edman, X. Zhao, and M. J. Paape. 2003. Lactation persistency, insights from mammary cell proliferation studies. *J. Anim. Sci.* 81: 18-31
- Carlen, E., E. Strandberg, and A. Roth. 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci.*: 87(9): 3062-3070.
- Carlen, E., U. Emanuelson, and E. Strandberg. 2006. Genetic evaluation of mastitis using linear models, threshold models, and survival analysis: a simulation study. *J. Dairy Sci.* 89: 4049-4057.
- Cole, J. B. and P. M. VanRaden. 2006. Genetic evaluation and best prediction of lactation Persistency. *J. Dairy Sci.* 89: 2722-2728.
- Cannas, A., A. Nudda, and G. Pulina. 2002. Nutritional strategies to improve lactation persistency in dairy ewes. <http://www.uwex.edu/animalscience/sheep/> Accessed October 21, 2006.
- Collard, B. L., P. J. Boettcher, J. C. Dekkers, D. Petitcler, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. *J. Dairy Sci.* 83(11): 2683-2690.

- Correa, M. T., H. Erb, J. Scarlett. 1993. Path analysis for seven postpartum disorders of Holstein cows. *J. Dairy Sci.* 76: 1305-1312.
- Dahl, G. E., B. A. Bachman, and H. A. Tucker. 2000. Photoperiodic effects on dairy cattle: a review. *J. Dairy Sci.* 83: 885-893.
- Dechow, C. D., G. W. Rogers, L. Klei, T. J. Lawlor, and P. M. VanRaden. 2004. Body condition scores and dairy form evaluations as indicators of days open in US Holsteins. *J. Dairy Sci.* 2004: 87(10):3534-41.
- Dekkers, J. C. M., J. H. Ten Haag, and A. Weersink. 1997. Economic aspects of persistency in dairy cattle. *Livest. Prod. Sci.* 53:237-252.
- Dijkstra, J., J. France, M. S. Dhanoa, J. A. Maas, M. D. Hanigan, A. J. Rook, and D. E. Beever. 1997. A model to describe growth patterns of the mammary gland during pregnancy and lactation. *J. Dairy Sci.* 80(10): 2340-2354.
- Ferris, T. A., I. L. Mao, and C. R. Anderson. 1985. Selection for lactation curve and milk yield in cattle. *J. Dairy Sci.* 68:1438-1448.
- Garverick, H. A. 1997. Ovarian follicular cysts in dairy cows. *J. Dairy Sci.* 80: 995-1004
- Gill, Ravinderpal, Howard H. Wayne, Leslie E. Kenneth, and Kerry Lissersmore. 1990. Economics of mastitis control. *J. Dairy Sci.* 73: 3340-3348.
- Green, B. L., B. W. McBride, D. Sandals, K. E. Leslie, R. Bagg, and P. Dick. 1999. The impact of monensin controlled-release capsule on subclinical ketosis in the transition dairy cow. *J. Dairy Sci.* 82: 333-342.
- Grossman, M., S. M. Hartz, and W. J. Koops. 1999. Persistency of lactation yield: a Novel approach. *J. Dairy Sci.* 82:2192-2197
- Harder, B., J. Bennewitz, D. Hinrichs, and E. Kalm. 2006. Genetic parameters for health traits and their relationship to different persistency traits in German Holstein dairy cattle. *J. Dairy Sci.* 89:3202-3212
- Henringstad, B., Y. M. Chang, I. M. Andersen-Ranvag, and D. Gianola. 2004. Genetic analysis of number of mastitis cases and number of services to conception using a censored threshold model. *J. Dairy Sci.* 89(10): 4042-4048.
- Hinrichs, D., E. Stamer, W. Junge, and E. Kalm. 2005. Genetic analysis of mastitis data using animal threshold model and genetic correlation with production traits. *J. Dairy Sci.* 88: 2260-2268.

- Jakobsen, J. H., R. Rekaya, J. Jensen, D.A. Sorensen, P. Madsen, D. Gianola, L.G. Christensen, and J. Pedersen. 2003. Bayesian estimates of covariance components between lactation curve parameters and disease liability in Danish Holstein cows. *J. Dairy Sci.* 86:3000-3007.
- Jamrozik, J., L. R. Schaeffer, and J. L. Dekkers. 1997. Genetic evaluation of dairy cattle using test day yields and random regression model. *J. Dairy Sci.* 80(6): 1217-1226.
- Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. *J. Dairy Sci.* 81 (9): 2502-2509.
- Koenig, S., A. R. Sharifi, H. Wentrot, D. Landmann, M. Eise, and H. Simianer. 2005. Genetic parameters of claw and foot disorders estimated with logistic model. *J. Dairy. Sci.* 88:3316-3325.
- Lewis, G. S. 1997. Uterine health and disorders. *J. Dairy Sci.* 80(5):984-994.
- Lin, C. Y. and K. Togashi. 2005. Maximization of lactation milk production without decreasing persistency. *J Dairy Sci.* 2005 :88(8):2975-2980.
- Lin, H. K., P. A. Oltenacu, I. D. Van Vleck, H. N. Erb, and R. D. Smith. 1989. Heritabilities of and genetic correlations among six health problems in Holstein Cows. *J. dairy Sci.* 72: 180-186.
- Lyons D. T., and A. E. Freeman .1991. Genetics of health traits in Holstein Cattle, *J. Dairy. Sci.* 74: 1092-1100.
- Lucy, M. C. 2005. Non-lactational traits of importance in dairy cows and applications for emerging biotechnologies. [N Z Vet J.](#) 53(6):406-415.
- Mantysaari, E.A., Y.T. Grohn, R.L. Quaas. 1991. Clinical ketosis: phenotypic and genetic correlations between occurrence and with milk yield. *J. Dairy Sci.* 74(11): 827-833.
- Miglior, F., B. L. Muir, and B. J. Van Doormaal. 2005. Selection indices in Holstein cattle of various countries. *J. Dairy Sci.* 88: 1255-1263.
- Montgomery, D. C., E. A. Peck, and G. G. Vining. 2001. Introduction to linear regression analysis, [John Wiley & Sons, Inc.](#), New York, NY.

- Muir, B. L., J. Fatehi, and L. R. Schaeffer. 2004. Genetic relationships between persistency and reproductive performances in first-lactation Canadian Holsteins. *J. Dairy Sci.* 87: 3029-3037.
- Pollot, G. E. 2000. A biological approach to lactation curve analysis for milk yield. *J. Dairy Sci.* 83(11): 2448-2456.
- Politis, I. 1996. Plasminogen activator system: implications for mammary cell growth and involution. *J Dairy Sci.* 79(6):1097-1107.
- Powell, R. L. and H. D. Norman. 2006. Major advances in genetic evaluation techniques. *J. Dairy Sci.* 89(4): 1337-1348.
- Raizman, E.A., and J. E. Santos. 2002. The effect of left displacement of abomasums corrected by Toggle-pin suture on lactation, reproduction, and health of Holstein dairy cows. *J. Dairy Sci.* 85: 1157-1164.
- Rogers G. W., G. Banos, and U. Sander-Nielsen. 1999. Genetic correlations among protein yield, productive life and type traits from the United States and diseases other than mastitis from Denmark and Sweden, *J. Dairy. Sci.* 82 : 1331-1338.
- Sander Nielsen, U., G. A. Pedersen, J. Pederson, and J. Jensen. 1996. Genetic parameters for mastitis, other diseases and somatic cell count in different parities in Danish dairy breeds. Page 10 in 47<sup>th</sup> Annu. Mgt. of EAAP, Lillehammer, Norway.
- Scott., T. A., B. Yandell, L. Zepeda, R.D. Shaver, and T.R. Smith. 1996. Use of lactation curves for analyzing of milk production data. *J. Dairy Sci.* 79: 1885-1894.
- Shaver, R. D. 1997. Nutrition risk factors in the etiology of left displaced abomasum in dairy cows: a review. *J. Dairy Sci.* 80: 2449-2453.
- Shim, E. H., R. D. Shank, and D. E. Morin. 2004. Milk loss and treatment costs associated with two treatment protocols for clinical mastitis in dairy cows. *J. Dairy Sci.* 87(8):2702-2708.
- Shook, G. E. 1989. Major advances in genetic evaluation techniques. *J. Dairy Sci.* 89(4):1337-1348.
- Shook, G. E. 2006. Major advances in determining appropriate selection goals. *J. Dairy Sci.* 89(4):1349-1361.

- Sogstad, A. M., T. Fjeldaas, and O. Osteras. 2005. Lameness and claw lesions of the Norwegian red dairy cattle housed in free stalls in relation to environment, parity and stage of lactation. *Acta Vet. Scand.*: 46(4):203-217.
- Solkner, J., and W. Funchs. 1987. A comparison of different measures of persistency with special respect to variation of test-day milk yields. *Livest. Prod. Sci.* 16:305-319.
- Stanton, T. L., L. R. Jones, R. W. Everett, and S. D. Kachman. 1992. Estimating milk, fat, and protein lactation curves with a test day model. *J. Dairy Sci.*:75(6):1691-1700.
- Tonner, E., G. J. Allen, and D. J. Flint. 2000. Hormonal control of plasmin and tissue-type plasminogen activator activity in rat milk during involution of mammary gland. *J. Endocrinol.* 167: 265-273.
- Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Ketton. 1994. Genetic parameters for common health disorders of Holstein cows. *J. Dairy Sci.* 78:421-430.
- Urton, G., M. A. G. von Keyserlingk, and D. M. Weary. 2005. Feeding behavior identifies dairy cows at risk for metritis. *J. Dairy Sci.* 88: 2843-2849.
- Val-Areola, D., E. Kebreab, J. Dijkstra, and J. France. 2004. Study of the lactation curve in dairy cattle on farms in central Mexico. *J. Dairy Sci.* : 87(11):3789-99.
- Van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders and relationships with 305-day milk yield and conformation traits of registered Holstein cows. *J. Dairy Sci.* 81:2264–2270.
- VanRaden, P. M., 1998. Best prediction of lactation yield and persistency. Proc. 6<sup>th</sup> World Congr. Genet. Appl. Livest. Prod., Armidale, Australia XXIII:347-350.
- Warnick, L. D., D. Janssen, C. L. Guard, and Y. T. Grohn. 2001. The effect of lameness on milk production in dairy cows. *J. Dairy Sci.* 84: 1988-1997.
- Wilde, C. J. and C. H. Knight. 1990. Milk yield and mammary function in goats during and after once-daily milking. *J. Dairy Res.* 57(4): 441-447.
- Wilson, D. J., R. N. Gonzalez, J. Hertl, H. F. Schulte, G. J. Bennett, Y. H. Schukken, and Y. T. Grohn. 2004. Effect of clinical mastitis on the lactation curve: a mixed model estimation using daily milk weight. *J. Dairy Sci.* 87:2073-2084.

- Wood, P.D.R. 1997. Algebraic model of the lactation curve in cattle. *Nature* 216:164-165.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer recorded data. I. incidence rates, heritability estimates, and sire breeding values. *J. Dairy Sci.* 87:4287-4294.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer-recorded data. II. Genetic correlations, disease probabilities, and relationships with existing traits. *J. Dairy Sci.* 87(12):4295-4302.
- Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2006. Genetic analysis of clinical mastitis data from on-farm management software using threshold models. *J. Dairy Sci.* 89(1): 330-336.