

Impact matrix analysis and cost-benefit calculations to improve management practices regarding health status in organic dairy farming

Project Number: 311824

- Deliverable -

D5.4 – LP module accounting for limited resources; supporting tool for vets/advisors

Due Date of Deliverable:	30-June-2015
Actual submission to EC date:	31-Dec2015
Deliverable Lead Partner:	P4 Wageningen University
Deliverable Author(s):	Felix van Soest, Monique Mourits & Henk Hogeveen

Executive Summary

This document provides a theoretical framework which can support farmers, veterinarians and/or animal health advisors in their decision making process towards animal health management and were to best allocate their scarce resources given the individual nature of each dairy farm. The proposed framework the "Animal Health Management Frontier" reveals the trade-off between preventive effort made to reduce disease incidence and the effect this has on failure costs. Via a linear programming technique a technical efficient frontier and current frontier can be constructed. These frontiers can be directly compared and yields useful insights in the efficiency of the current preventive effort made. Moreover, comparisons between multiple production disorders can be made to determine priority areas in animal health management.

	Dissemination Level	
PU	Public	PU





Table of Contents

1	Ir	ntroduct	lion	.3	
2	In	nportar	t production disorders in dairy cattle	.4	
2	2.1	Mas	titis	.5	
	2	.1.1	Occurrence	.5	
	2	.1.2	Consequences	.5	
	2	.1.3	Risk factors	.6	
	2	.1.4	Economic impact	.6	
2	2.2	Keto	osis	.6	
	2	.2.1	Occurrence	.6	
	2	.2.2	Consequences	.7	
	2	.2.3	Risk factors	.7	
	2	.2.4	Economic impact	.8	
2	2.3	Lam	neness	.8	
	2	.3.1	Occurrence	.8	
	2	.3.2	Consequences	.8	
	2	.3.3	Risk factors	.9	
	2	.3.4	Economic impact	.9	
3	A	nimal h	al health management frontier9		
	3.1	Frar	nework	.9	
	3	.1.1	Determine disease incidences.	10	
	3	.1.2	Determine failure costs	10	
	3	.1.3	Construct technical efficient and current frontier	11	
	3	.1.4	Report outcome	11	
	3.2	Fun	ctional form	12	
	3.3	Esti	mating the farm specific frontiers	12	
	3	.3.1	Technical efficient frontier	12	
	3	.3.2	Farm frontier	13	
	3.4	Exa	mple	13	
4	D	iscussi	on	14	
5	Conclusion15				
6	References				



A Framework to determine priorities in Animal Health

1 Introduction

Economic losses from livestock diseases can be extremely large. To illustrate, in the dairy sector, average production losses from two of the more common production diseases, i.e. mastitis and lameness, have been estimated at \in 210 and \in 89^d respectively, per clinical case and respectively \in 78 and \in 55 per average cow on the farm per year (Huijps et al., 2008; Bruijnis et al., 2010).

In spite of the potential scale of disease costs, there has been a general failure, across the EU, to significantly improve farmed animal health status over the last decade (Hovi et al., 2003). One of the possible reasons for this is the observation that many farmers do not take up the full range of health measures suggested to them by their health advisers (Huijps et al., 2009a, 2010; Vaarst et al., 2011). If further improvements in health status are to be achieved, a dual approach is needed, involving both the provision of better advice to farmers on animal health measures, and incentives to farmers to greater rates of uptake of these recommended health measures.

It has been suggested that one of the reasons why farmers do not always adopt measures recommended by health advisers is that they do not have a clear idea of the scale of the economic losses that they incur through livestock diseases (van Asseldonk et al., 2010). Providing farm specific cost estimates of the scale of economic losses resulting from livestock diseases is therefore seen as a useful way of incentivize farmers to higher rates of uptake health improvement measures.

However, as McInerney et al. (1992) pointed out, estimating only the magnitude of disease costs, i.e. the failure costs, provides insufficient information on which to make economically optional management decisions. What is required is an estimate of total economic costs arising from diseases, where this accounts for two components: (i) output losses following disease occurrence (the 'failure' costs), and (ii) expenditures arising from prevention or treatment actions.

According to the McInerney's theoretical framework for the analysis of livestock disease costs (McInerney et al., 1992), there exists a trade-off between the costs and benefits of disease control, i.e. there exists an economically optimal level of disease prevention/control expenditure, beyond which further investment in disease control is not repaid in output gains. Hogeveen et al. (2011) further explored the nature of the trade-off between failure costs and preventive costs, suggesting that they share a substitution relationship, following a downward convex curve, in-line with the economic principle of diminishing returns. It is information on these expenditure–failure cost trade-offs that therefore needs to be conveyed to farmers.

The problem with doing this is that the nature of these trade-offs varies, not only from disease to disease, but also from farm to farm. Because there is a trade-off between control expenditure and failure costs for individual diseases, farmers must consider the nature of this trade-off for each disease challenge in order to determine the optimal level of investment in the control of each. However, there is also a trade-off between diseases, such that optimal levels of investment in

¹ Value converted from \$ to \in following a conversion rate in which \$1 equals \$1,07



control of one disease may yield more, or less, in terms of reductions in failure costs, than optimal expenditure in the control of other diseases. Farmers are therefore faced with a very complex management decision, i.e. within the constraints of available resources, first, which production diseases should be targeted for investment, and second, what is the optimal level of investment to be made in each?

As a way of reducing the complexity of these offsetting calculations, a number of farm animal health decision-support tools have been developed (e.g. Huijps, 2009). However, these existing models suffer from a number of important limitations. In most of the models the estimation of disease costs is based on the extraction and application of general rules to individual farms, with no account taken of farm specific characteristics, including limitations on the availability of farm resources.

Presenting farmers with generalized data on these questions, i.e. a one-size-fits-all approach, would have limited value, because these trade-off functions vary from farm to farm due to the fact that the building blocks of the functions themselves vary from farm to farm. To illustrate, the magnitude of failure costs for individual diseases varies between farms as a consequence of disease incidence and severity, which in turn is impacted by type of livestock housing, number of animals, farming system, management practice and the quality of farm resources and inputs used etc.

Moreover, the majority of these support tools only estimate the expected impact of one production disease at a time. This is a significant limitation because, in reality, farmers will typically face multiple disease challenges, and have to decide which of these diseases they should focus on, given limited resources. To be able to make such decisions there is a need for a structural framework which provides farmers with insights into the trade-offs between effort and failure costs not just for single diseases, but across multiple diseases simultaneously.

The aim of the IMPRO WP5 work reported in deliverable 5.4 is therefore the development of a linear programming-based approach to improve the allocation of resources in managing multi-factorial diseases in dairy cattle, such as mastitis, ketosis and lameness. This Animal Health Management Frontier model uses farm specific data on disease situation to establish the farm-specific animal health management frontiers for different production diseases and by that means enables the farmer to select a production disease with the highest expected benefits for the additionally spent resources to focus on for further preventive measures. It provides farmers and/or veterinarians with insight into the trade-off between effort and failure costs of production diseases, in which the farm specific constraints regarding animal health are taken into account, as a means to better allocation of resources to maximize the reduction in failure costs.

This deliverable first describes the three production diseases of interest in this work: mastitis, ketosis and lameness. Per disease, the occurrence, consequences and economic effects are provided. In chapter 3, the animal health management frontier framework is described, including an example. After a discussion in chapter 4 a conclusion is provided in chapter 5.

2 Important production disorders in dairy cattle

Three production disorders are currently of importance amongst dairy farmers due to different reasons. In the next section we will describe the occurrence, consequences, risk factors and economic costs of mastitis, ketosis and lameness.



2.1 Mastitis

2.1.1 Occurrence

Clinical mastitis is defined as a cow with visual abnormalities in the milk and/or quarter. Lam et al. (2013) reported incidence rates for clinical mastitis of 28.1 (95%Cl, 25.7-30.7) per 100 cow-years at risk in 2009 on 175 Dutch dairy herds. Van den Borne (2010) reported an incidence rate for clinical mastitis of 33.5 (95%Cl, 31.3-35.8) per 100 cow-years at risk in 2004 on 205 Dutch dairy herds. Zwald et al. (2004) reported an incidence of mastitis of 23% for the first 30 DIM. Suthar et al. (2013) however, reported a prevalence of clinical mastitis in Europe at an average of 6.1% for the first 30 DIM. The same study found a large variation in average prevalence, 1.8% - 29.5%, between countries. An average incidence rate of 9.1 per 100 cow-years for veterinary treated cases of clinical mastitis was found on Swedish organic dairy herds, whereas a significant higher incidence rate of 14.7 was found in conventional Swedish dairy herds (Hamilton et al., 2006). Generally, recording only cases of clinical mastitis treated by a veterinarian may represent an underestimation of the true number of clinical cases of mastitis (Hamilton et al., 2006). The incidence of mastitis might therefore be better revealed from farmers reports (van den Borne et al., 2010; Lam et al., 2013), contrary to the veterinary records where only cases treated by the veterinarian are reported. However, also reports from farmers do not ensure that all cases of clinical mastitis are captured.

Generally, subclinical mastitis is reflected by individual cow somatic cell counts (SCC). Different cutoff values have been used to determine whether or not a cow has subclinical mastitis e.g. 100,000/150,000 cells/mL (Pyörälä, 2003), 200,000 cells/mL (Schukken et al., 2003) or 250,000 cells/mL (Dohoo and Meek, 1982). Incidence rates per 100 cow-years at-risk for subclinical mastitis, with a cut-off SCC value of 200,000 cells/mL, were reported at an average of 23.0 (95%CI, 22.2-23.9) in 2004 and 22.2 (21.4-23.2) in 2009 (van den Borne et al., 2010; Lam et al., 2013). A prevalence of 14.3% for subclinical mastitis, with a threshold of 250,000 cells/mL, was reported for Flemish dairy herds with a certified good animal health status (Piepers et al., 2007).

2.1.2 Consequences

Milk yield losses due to clinical mastitis vary on average from 4% to 6% (300-400kg) at the lactation level, similarly clinical mastitis affects fat (0.10% reduction) and protein levels (0.05% reduction) (Hortet and Seegers, 1998). Rajala-Schultz et al. (1999) reported similar production losses as a consequence of clinical mastitis varying from 110-552 kg depending on parity and the time of mastitis occurrence. Although subclinical mastitis generally is diagnosed around a threshold of 200,000 cells/mL, milk losses already occur once an SCC level of 50,000 cells/mL is passed (Halasa et al., 2009b). Similarly, Dürr et al. (2008) showed that milk losses are influenced by SCC and DIM for each individual cow. This would suggest that the majority of the dairy cows contribute to a reduced milk yield, even without ever reaching the 200,000 cells/mL threshold. Studies using threshold levels to determine milk production losses due to SCM might therefore underestimate the production losses due to subclinical mastitis.

A review by Fourichon et al. (2000) found no effect of mastitis on fertility. It was, however, found that occurrence of mastitis <20 days after AI was associated with a >50% reduction in pregnancy risk. Occurrence of mastitis before AI had a weak effect on pregnancy (Pryce et al., 2004). Similarly both Chebel et al. (2004) and Santos et al. (2004) found a reduced conception rate and a higher risk of embryonic loss of mastitis when cows had a clinical case of mastitis between AI and 50 days of pregnancy. A delayed onset of oestrus behaviour was experienced when clinical mastitis was



observed within 28 days after calving (Huszenicza et al., 2005). These findings suggest that mastitis affects fertility (Walsh et al., 2011).

Ahlman et al. (2011) reported that out of all culled animals on a farm 30.7% and 22.5% were due to clinical mastitis for respectively organic and conventional farms with Swedish Holstein dairy cows. Mastitis was therefore seen as the most important reason for on farm culling. Seegers et al. (1998) used more specific reasons for culling and reported that mastitis attributed 7.8% to all culled animals. Nevertheless, combining all udder disorders in one would attribute to 18% of all culled animals. Since some cases of mastitis occur in early lactation it is likely that not all cows are culled for mastitis reasons but for low milk yield or for reduced fertility. Cows with repeated episodes of clinical mastitis, with higher risks for cows in their third parity or higher (Bar et al., 2008).

2.1.3 Risk factors

On cow level, high milk production was found to be a risk factor for clinical mastitis (e.g. Ingvartsen et al., 2003). Other identified risk factors are associated to housing conditions, hygiene, nutrition, milking technique and machine milking (Barkema et al., 1999b). Moreover, it was found that one case of clinical mastitis can cause other cows to attain clinical mastitis (Halasa et al., 2009a). Bulk tank somatic cell count was found to be influenced by stockman ship and hygienic conditions (Barkema et al., 1999a). Generally, appropriate management measures were found to influence herd somatic cell count in a positive way (Dufour et al., 2011).

2.1.4 Economic impact

Hogeveen et al. (2011) gave an overview of the costs of mastitis up to 2009, and found that costs per average cow varied between €61 and €97. After hat review other studies estimated the costs of more specific cases of mastitis. Huijps et al. (2009b) estimated the costs of (sub)clinical heifer mastitis at € 31 / heifer / yr. Cha et al. (2011) estimated the costs of Gram positive, Gram negative and other mastitis causing pathogens, at €123, €193 and €87 per case. Similarly, Sørensen et al. (2010) found the costs of mastitis to vary between €149 and €570 per case, depending on the mastitis causing pathogen. Heikilä et al. (2012), estimated costs of mastitis at €458 / case or alternatively €147 / cow / yr. Although estimates of the costs of mastitis differ, it is clear that mastitis has a substantial impact on farm economics. Mastitis does not only affect farm income, the costs of mastitis are directed throughout the dairy processing chain and affect processors profitability (Geary et al., 2013). Within the IMPRO project (part of deliverable D5.3) farm specific failure costs of mastitis were estimated for organic dairy farmers in Germany, France, Spain and Sweden and estimated at an average of respectively €106, €114,€149 and €124 /cow / yr.

2.2 Ketosis

2.2.1 Occurrence

Clinical ketosis is diagnosed when cattle show wasting with decreased appetite, fall in body condition and milk production where some cows have short periods of bizarre neurological and behavioural abnormality. Subclinical ketosis is detected by measuring ketones in milk or urine and defined as abnormal concentrations of circulating ketone bodies in the absence of clinical signs of ketosis (Radostits and Done, 2007). Both subclinical and clinical ketosis are a consequence of a disturbed energy balance during calving, where energy intake is unable to keep up with demand. Different methods of testing have been reported in which the golden standard is measuring plasma β -hydroxybutyrate (BHBA) which reflects the amount of circulating ketone bodies (Duffield, 2000).



The observed prevalence of subclinical ketosis in Dutch dairy cows between 5-60 days in milk at a plasma BHBA level \geq 1,2 µmol/L was 11.2% (van der Drift et al., 2012). Dohoo and Martin (1984) found that 92% of the cases of subclinical ketosis were found within the first 65 days of the lactation and a corresponding prevalence of 12.1%. Similarly, Andersson and Emanuelson (1985) found that the majority of cases occurred within the first two months of lactation with a reported prevalence of 8.9%, 4.7% and 1.1%, respectively, at the first three months. Reported prevalences of 14.1%, 5.3%, 3.2% and 1.6% for early lactation(<65 DIM), mid lactation (65-149 DIM), late lactation (>149 DIM) and dry period by Duffield (1997) again show similar findings. Recently, McArt et al. (2015) reported a peak incidence of 22.3% at 5 DIM for subclinical ketosis. Suthar et al. (2013) found an average prevalence of 21.8% for subclinical ketosis during 2-15 DIM and 3.7% for clinical ketosis. Vanholder et al. (2015) reported a prevalence of 47.2% and 11.2% for subclinical and clinical ketosis in the period 7-14 DIM and found high milk yield to be one of the risk factor for ketosis. This would suggest that with a tendency towards a higher production level per dairy cow, ketosis could potentially become more prevalent in the future.

2.2.2 Consequences

Milk yield losses due to subclinical ketosis have been reported by McArt et al. (2012) at an average of 0.5kg/day within the first 30 DIM. Rabboison et al. (2014) reported a production loss by subclinical ketosis of 112 kg in 305 d milk production, whereas a correction for other diseases resulted in a production loss of 251 kg in 305 d milk production. According to Duffield et al. (2009), part of the losses might be cloaked by the fact that moderate cases of subclinical cases were able to produce more milk than cows without subclinical ketosis which was also found by Vanholder et al. (2015).

Fertility was affected by subclinical ketosis and it was found that the probability of pregnancy was reduced by 20% in cows with a diagnosed case of subclinical ketosis within the first two weeks of the lactation (Walsh et al., 2007). Return to normal probabilities of pregnancy after a case of subclinical ketosis was on average reported after 160 DIM and the probability of a first successful insemination was reduced by 50% (Walsh et al., 2007).

Cows with diagnosed ketosis were more likely to be culled throughout the lactation than non-ketotic cows. Nevertheless, the direct risk of ketosis was found to be reduced when conception status was included in the model (Gröhn et al., 1998). An increased BHBA level was found to be associated with an increased risk of culling within the first 60 DIM (Roberts et al., 2012). Relations of subclinical ketosis on increased culling are, however, not clear since subclinical ketosis primarily impedes milk production losses and reduced fertility it is possible that cows are culled for either production or fertility problems than for subclinical ketosis.

2.2.3 Risk factors

A higher body condition score post calving was found to be a risk factor for dairy cows to develop either subclinical or clinical ketosis (Gillund et al., 2001; Vanholder et al., 2015). Herd size, feeding regime, calving period and parity were risk factors for developing subclinical ketosis (Berge and Vertenten, 2014). According to Stengärde et al. (2012), a large herd size, a high maximum daily milk yield, housing of dry cows and feeding regime were risk factors for the development of clinical ketosis. Feeding a larger amount of concentrate in early lactation was deemed to increase the risk of ketosis but was not consistent (Gustafsson et al., 1995).



2.2.4 Economic impact

Current estimates of the costs of ketosis are only scarcely available in scientific literature. Costs of ketosis were estimated to vary from US\$78 to US\$289 per case of ketosis (Geishauser et al., 2001; Guard, 2008; McArt et al., 2015). Due to the relation of ketosis with other diseases, it is essential that the increased risk of other diseases is included in costs estimations. Which, until now, has only been done by Mc Art et al. (2014) who found that 88% of displaced abomasum cases, and 70% of metritis cases were attributable to ketosis. Within the IMPRO project (part of deliverable D5.3) farm specific failure costs of ketosis were estimated for organic dairy farmers in Germany, France, Spain and Sweden and estimated at an average of respectively ≤ 28 , ≤ 11 , ≤ 5 and ≤ 29 /cow / yr.

2.3 Lameness

2.3.1 Occurrence

There does not exist one true definition for clinical lameness. A commonly used definition, however, is based upon a locomotion score (Whay et al., 1997; Thomsen et al., 2008). Most of these scoring systems use a 5 point ordinal scale (Sprecher et al., 1997; Winckler and Willen, 2001; Flower and Weary, 2006). These five points vary from normal to severe lameness. However as noted by Engel et al. and Thomsen et al. (2008), the decision which score is appropriate is arbitrary and observers may attribute different scores to the same individual. In the future, computerized vision techniques to detect lame cows could prove to be a perspective to cope with observers bias (Song et al., 2008).

Prevalence of claw disorders in dairy cows was reported to be more than 70% where cows had at least one claw disorder at the time of hoof trimming (Somers et al., 2003; van der Waaij et al., 2005). Claw disorders comprise multiple lesions. Van der Linde et al. (2010) found sole haemorrhage, interdigital dermatitis and digital dermatitis to be the most prevalent claw disorders, their prevalence being respectively 38%, 29% and 22%. Similarly, Somers et al. (2003) and Van der Waaij et al. (2005) found the same predominant lesions. A study by Manske et al. (2002b) on Swedish dairy cows found sole haemorrhage, heel horn erosion and dermatitis to be most prevalent.

2.3.2 Consequences

Several studies found that lameness affected milk production. Warnick et al. (2001) reported that milk yield was reduced by $1,5 \text{ kg/d} \ge 2 \text{ wks}$ after diagnosis and the reduction was found to be greater in multiparous than in primiparous cows. Clinical lameness amongst UK dairy herds was found to reduce the 305d milk yield by 360 kg (Green et al., 2002). Contrary, Barkema et al. (1994), found that lame cows had a higher milk yield than non-lame cows most likely because a higher milk yield was a risk factor for developing lameness.

Lame cows were found to have longer lying times and spend less time standing, walking and expressing oestrus behaviour (Walker et al., 2008). Non-lame cows were more likely to conceive at first service, cows experiencing lameness within the first 30 DIM were more likely to develop ovarian cysts and, overall, lame cows were less likely to conceive than non-lame cows (Melendez et al., 2003). Pregnancy was found to be delayed by, on average, 12 days for lame cows when compared to non-lame cows (Alawneh et al., 2011). Barkema et al. (1994), however, found no effect of lameness on pregnancy rate at first service.

Approximately, 5% of all culled dairy cows on Swedish dairy herds were culled because of leg problems (Ahlman et al., 2011). Culling cows because of lameness was found to be 2,5% of all



culled cows on French dairy herds (Seegers et al., 1998). Green et al. (Green et al., 2002) found that the risk of locomotion disorders were highest the first 3 months in lactation. Culling dairy cows is mostly because of multiple reasons and culling a cow at the end of a lactation could be due to a history of lameness which may at that time not be the primary reason for culling. A low prevalence of culled animals due to lameness might therefore be clouded by such effects.

2.3.3 Risk factors

Claw trimming was found to reduce the odds of lameness (Manske et al., 2002a). The risk for lameness was found to be increased when stall lying comfort decreased and when a cow was in the lowest body condition score quartile, depending on breed (Dippel et al., 2009). Also concrete flooring was found to be a risk factor for developing lameness (Somers et al., 2003). Cubicle characteristics, bedding material, access to pasture were found to be risk factors which were associated with hock lesions in dairy cattle (Kester et al., 2014). Hock injuries were found to be more prevalent among cows with a shorter calving interval, increased parity, duration of summer grazing, cow milk yield, low feed space per cow, cleanliness of passageway and type of bedding material (Rutherford et al., 2008).

2.3.4 Economic impact

Costs of lameness were estimated at an average of \$216, \$133 and \$121/ per case of respectively sole ulcer, digital dermatitis and foot rot (Cha et al., 2010). Total costs of different lameness causing foot disorders were estimated for an average herd at \$479, \$825, \$1,517, \$641 / yr. for respectively interdigital phlegmon, interdigital dermatitis and heel erosion, digital dermatitis and sole ulcer (Bruijnis et al., 2010). The costs of lameness were generally based on the underlying lameness, causing disorders and varied substantially depending on the disorder. Within the IMPRO project (part of deliverable D5.3) farm specific failure costs of mastitis were estimated for organic dairy farmers in Germany, France, Spain and Sweden and estimated at an average of respectively \in 48, \in 31, \in 53 and \in 33 /cow / yr.

3 Animal health management frontier

3.1 Framework

For this study we developed a theoretical framework (figure 1). The basis for this theoretical framework basically consists of the animal health management frontier: a substitution relationship between failure costs and preventive costs that follows a downward convex curve. Since this relationship is farm-specific, depending on, for instance, housing facilities and management capabilities of the farmer, this relationship differs between farms. We distinguish 2 different farm-specific frontiers: the "technical efficient" frontier (denoting the animal health management frontier when measures are carried out in the most efficient and correct way) and the "current" frontier (denoting the animal health management frontier as it is). In our framework we designed an approach consisting of 4 steps to estimate the farm-specific animal health management frontiers:

- 1. Determine disease incidences;
- 2. Determine failure costs;
- 3. Construct technical efficient and current frontier;
- 4. Report outcome.



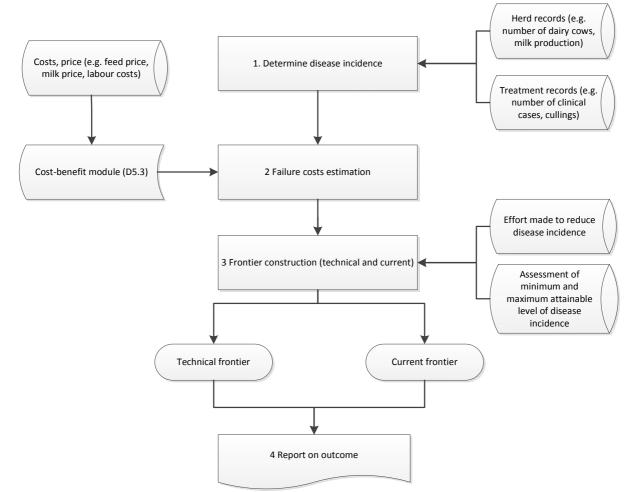


Figure 1 Theoretical framework of the animal health management frontier

3.1.1 Determine disease incidences.

In the first step, the current number of contracted cases of each disease (N_{curr}) on the target farm will be determined, and expressed as the total number of cases for a given disease. This can be done using existing herd records, such as treatment records, information from herd management systems etc. If time does not allow for this or if herd records are not available, the farmer and/or advisor may make an expert-based estimate of N_{curr} .

3.1.2 Determine failure costs.

Using the N_{curr} and information about the losses associated with the disease as well as information about price levels, the total farm specific failure costs (FC_T) will be estimated. The failure costs are defined as the costs which are associated with animals suffering from a disease and consist of production losses, discarded milk, treatments and culling. The topic of failure costs has been extensively studied elsewhere in work package 5 and results have been published in deliverable 5.3. The failure cost calculation tool developed and described in D5.3 can assist in the estimation of FC_T . By combining FC_T with N_{curr} and the herd size, the failure costs per contracted case of the disease (FC_C) can be determined.



3.1.3 Construct technical efficient and current frontier.

In a third step, the two frontiers representing the trade-off between failure costs and preventive effort are constructed. One frontier represents the trade-offs between disease failure costs and preventive effort when the preventative measures are applied as efficiently as theoretically possible. The second frontier represents the trade-offs between preventive efforts and failure costs under conditions of sub-optimal application of preventative measures. In the original animal health management frontier, as described by Hogeveen et al. (2011), the failure costs are inversely related to preventive costs (although this is a non-linear relationship). The preventive costs are, however, very difficult to estimate (see Deliverable 5.3). It is difficult to determine exactly which measures are applied and how much a general (e.g. hygienic) measure contributes to the prevention of one specific disease. It is also difficult to determine the potential maximum preventive costs for a farm as some of the preventative measures deployed on farms, such as provision of adequate nutrient supply, are not in fact seen as preventive measures, but rather are seen as part of general management. Although in theory possible, we decided not to quantify the current preventive costs but to use an ordinal measure of "preventive effort" instead. Preventive effort is defined as the relative effort made by the farmer or farm manager to reduce disease incidence on a farm. The preventive effort is expressed on a 7-point Likert scale in which 0 means 'do nothing' and 6 means 'every possible effort is made'. In order to produce both the efficient and the current animal health management frontiers, the hypothetical maximum number of contracted cases of a disease (N_{max}) is needed. N_{max} is the number of cases that should occur if no efforts are made to control a disease. To establish the efficient animal health management frontier, the theoretical minimum number of cases due to a disease (N_{min}) also has to be determined. N_{min} is the number of cases of a disease that would occur when every possible preventive effort is made (this number will always be nonzero). N_{max} and N_{min} cannot typically be derived from herd health recordings. The estimation of these figures therefore has to be derived from the expertise and knowledge of the farmer, veterinarian and/or health advisor. To obtain these estimates, a benchmark, or generalised, value for N_{max} is first provided and this is, if deemed necessary, adjusted for the specific farm. To determine N_{min} , a dialogue between farmer, veterinarian and/or animal health advisor is needed. The estimation of N_{max} , with a preventive effort of 0 and N_{min} , with a preventive effort of 6, are input to derive the technically efficient animal health management frontier. Based on the estimated curve, a technically efficient FC frontier can be constructed in which at each effort point the associated FC_{τ} can be determined. The technically efficient frontier now shows the trade-off between FC_T and preventive effort.

Next, the farmer, veterinarian and/or health advisor discuss the amount of preventive effort that is currently applied using a 7-point Likert scale. The $FC_{T,max}$ is derived from the technically efficient frontier and the $FC_{T,curr}$ is derived from the cost-benefit module. To construct the current frontier, $FC_{T,max}$ is attained at an effort of 0 and $FC_{T,curr}$ is attained at the current preventive effort level. Based on these presumptions the current animal health management frontier can be established

3.1.4 Report outcome

For each disease, the efficient and current animal health management frontiers are presented graphically, showing the trade-off between preventive effort and FC in both the current state of the farm and in a situation where the farm would be fully efficient in terms of disease prevention. The graph shows: (i) the economic consequences of being fully efficient at current levels of preventive effort; and (ii) the economic consequences of increasing, or decreasing, the preventive effort by 1 or



more units. By changing the parameter values in the graph and discussing the outcomes, new insights can be gained into animal health management and how improvements can be achieved.

3.2 Functional form

Technical efficient frontier

The functional form of with the technically efficient frontier can be expressed as:

$$N_{e\!f\!f} = \frac{(N_{\max} - N_{\min})}{(X+1)^{\alpha_{e\!f\!f}}} + N_{\min}$$

With restrictions:

 $lpha_{eff} \ge 0$ $N_{\max} \ge N_{\min} \ge 0$

In this formula the number of contracted cases of a disease is a function of N_{max} , N_{min} , the preventive effort (X) and the curvature of the frontier (α_{eff}). In the technically efficient frontier α_{eff} is estimated such that at X = 0, N equals N_{max} and at X = 6, N equals N_{min} . This information is used to construct the failure costs frontier for a technically efficient farm. From this it follows that:

$$FC_{T,eff} = \left(\frac{(N_{\max} - N_{\min})}{(X+1)^{\alpha_{eff}}} + N_{\min}\right) FC_C$$

Current frontier

Similarly, the functional form of the current frontier can be expressed as:

$$FC_{T,curr} = \left(\frac{(N_{\max} - N_{\min})}{(X+1)^{\alpha_{cur}}} + N_{\min}\right) FC_C \quad \text{In the}$$

In the current frontier α_{curr} is estimated such that at X = 0, FC_T equals $FC_{T,max}$ and at the assessed preventive effort (X)

 $FC_{T,curr}$ equals $FC_{T,X}$ derived from the cost benefit module (deliverable 5.3).

3.3 Estimating the farm specific frontiers

If a farm was fully efficient, both frontiers would be similar. It is therefore important to estimate the values of α_{eff} and α_{curr} since these influence the shape of the frontier.

3.3.1 Technical efficient frontier

To construct the technical efficient frontier we need to solve the following model, using linear programming, to determine the efficient alfa value (α_{eff}):

$$N_{\min} = \frac{(N_{\max} - N_{\min})}{(X+1)^{\alpha_{eff}}}$$

With restrictions:



 $\alpha_{eff} \ge 0$ $N_{\max} \ge N_{\min} \ge 0$ X = 6

This gives us a value of α_{eff} which reaches N_{max} at an effort (X) of 0 and N_{min} at an effort (X) of 6. This α_{eff} can be entered in the FC model to estimate the FC frontier for a fully efficient farm.

3.3.2 Farm frontier

To construct the farm frontier we need to solve the following model for the farm alfa (α_{farm}) value, using the linear programming method:

$$FC_{T,curr} = \left(\frac{N_{\max} - N_{\min}}{\left(X_{curr} + 1\right)^{\alpha_{jarm}}} + N_{\min}\right) FC_{C}$$

With restrictions:

$$\alpha_{farm} \ge 0$$
$$N_{\max} \ge N_{\min} \ge 0$$

In which X_{curr} is the level of effort that best reflects the current effort to reduce disease incidence and $FC_{T,curr}$ is the current level of failure costs as estimated by the cost-estimation tool. Entering the solved value of α_{farm} in the FC model allows us to estimate the FC frontier for an individual farm.

3.4 Example

To illustrate this approach with an example, two diseases mastitis (M) and ketosis (K) are compared on a farm with a herd size of 100 dairy cows. In this case $C_{m,j}$ is \in 235 and $C_{k,j}$ is \in 120, $Nmax_{M,}$ is 35 $Nmin_{M}$ is 10, $Nmax_{K}$ is 90, $Nmin_{K}$ is 5%. Effort to reduce M is indicated by a rank score of 5 and effort to reduce K is indicated by a score of 2. Current FC_{T,curr} of M are estimated at \in 4,500 and FC_{T,curr} of K are estimated at \in 3,000. Solving for $\alpha_{eff,M}$, $\alpha_{eff,K}$, $\alpha_{farm,M}$ and $\alpha_{farm,K}$ gives alpha values of 1.6, 0.6, 3.2 and 1.3 respectively. Two constructed frontiers (technically efficient and current) are presented for each disease in figure 2, where the horizontal arrows represent an increase in effort and vertical arrows represents an increase in efficiency.

If the farmer would increase the preventive effort by 1, this would result in a decrease of \in 178 in FCM and a decrease of \in 757 in FCK. So, increasing efforts in reducing ketosis prevention gives a higher pay-off than increasing efforts in mastitis prevention, even when the failure costs of mastitis are higher than the failure costs of ketosis. However, it is obvious in this example, that the farmer is not efficient. The current animal health management frontiers, both for ketosis as well as for mastitis are far above the technically efficient frontiers. If the farmer could become fully efficient the same level of effort would yield a decrease of \in 1,816 in FCM and a decrease of \in 2,097 in FCK. Unless the estimated minimum incidence of disease was wrong, there is quite some room to improve the efficiency of the currently applied preventive measures. As the figure shows, for both diseases the biggest gains can be attained by becoming more efficient, rather than increasing the levels of effort.



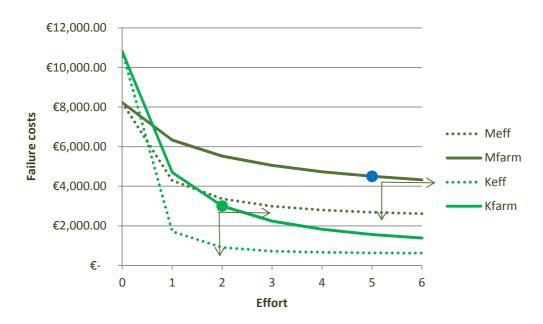


Figure 2 Animal health management frontier for mastitis (M) and ketosis (K) in which the solid lines are the current frontiers and the dotted lines are the technically efficient frontiers.

4 Discussion

One of the primary assumptions made in the use of the current model is that with respect of the ordinal scale use to measure effort, the units are equidistant (this effectively implies a cardinal/interval scale) and assessments of effort are made as such. This means that moving between any two categories on this scale equates to the same magnitude of change in actual effort for different assessors. This might not be the case in reality. Failure to comply with this assumption will classify a farmer in the wrong scale of effort and thereby consequently assume wrong levels of efficacy. Similarly, the problem with non-equidistant scaling in which the same lame cow was classified in different categories of the ordinal scale of the lameness scoring system by different assessors has been pointed out by Engel et al. (2003) and Thomsen et al. (2008). This makes clear that the system of scaling relies on the capacities and experience of the assessor. There does not seem to be a clear solution for this problem, although one possible solution would be to translate preventive notional effort into actual preventive costs. This would however, require a more intense assessment of what is being done on a specific farm to control disease costs currently and an assessment of the maximum attainable level expenditures on preventive measures is on a farm by farm basis.

Traditionally, disease cost estimations distinguish diseases into clinical or subclinical forms (Dijkhuizen and Morris, 1997). Nowadays, for cost estimations of mastitis it is considered more important to distinguish other aspects of the disease e.g. heifer mastitis (Huijps et al., 2009b) or pathogen specific mastitis (Cha et al., 2011). The clinical and subclinical differentiation in ketosis has also been abandoned by some (Berge and Vertenten, 2014), while most studies nowadays use the level of ketone bodies present in the urine, blood or milk, as an indicator for ketosis (de Roos et al., 2007; van der Drift et al., 2012). Lameness is typically differentiated on the basis of different types of hoof lesion, e.g. digital dermatitis, sole ulcer, interdigital phlegmon (Manske et al., 2002b; Brenninkmeyer et al., 2013). Each of these separate "forms" within a disease impose different failure costs, as explained in the disease incidence section of this deliverable, because of the nature of the disorder. This level of detail in failure costs cannot be determined based on the cost estimation tool. Given the specific purpose of the animal health management frontier and the aims



of the farmer or herd manager, this does not need to be problematic. On the one hand, the costbenefit module is flexible enough to capture specific forms of a disease and on the other hand, the animal health management frontier should provide the farmer and/or veterinarian with insights into the trade-off between effort and failure costs of production diseases, given their specific farming situation and how preventive effort should be allocated. Moreover, the framework could be adapted to multiple forms of the same disease, much the same as in the example given in this document. To illustrate, the mastitis and ketosis examples could be replaced with the FCs of sole ulcer and digital dermatitis (i.e. two forms of lameness). Therefore, with an increasing knowledge on the form specific costs of different disorders within a disease, a farmer and/or veterinarian can adopt the animal health management frontier to determine were to allocate resource given two forms within the same disease.

One of the pitfalls of the animal health management frontier in the current form is that it relies on the number of cases due to one disease (N) and consequently estimates total failure costs by multiplying N with the failure costs per case. In reality, this disease information should be seen as a complex of multiple disorders and included in the animal health management frontier as such, and in which the complex relation between disorders is taken into account, e.g. an increase in the incidence of ketosis may increase the risk of mastitis. Typical costs components such as milk production losses and culling may therefore be accounted for twice, i.e. once for each of two diseases, whereas in practice losses or culling in individual animals will have multiple causes and so will overlap. Failure costs may therefore, wrongfully, be overestimated and potential gains, either by increasing effort or improving efficiency, may also be overestimated. This true estimation of total failure costs, i.e. a complex of multiple disorders with a fair degree of correlation, should ideally be implemented in the animal health management frontier. Given the current state of epidemiological research on these correlations there is the possibility of estimating failure costs as such. Estimation of these total failure costs would solve the issue of attribution of preventive costs of general measures to specific disorders, since all are reflected in the total failure costs. The general framework of the animal health management frontier would be able to accommodate these total failure costs and result in only two 'all disease' curves: the "technically efficient frontier" and the "current frontier". With further advances in epidemiological and economic research the animal health management frontier would become more relevant since input becomes more reliable.

Reporting only the magnitude of the failure costs of a disease is of no use to the farmer, veterinarian or farm health advisors since it does not reveal which part of the costs could truly be reduced. The cost-benefit module as reported in deliverable 5.3 is suffering from this same fault. Combining the cost-benefit module from deliverable 5.3 with the animal health management frontier described in this deliverable allows for a better understanding of the trade-offs between failure costs and preventive efforts made and reveals the potential gains to be made and compares multiple disorders. Moreover, with the addition of the animal health management frontier, decision making has been made farm specific, something which, until now has been neglected in the economics of animal health.

5 Conclusion

The animal health management frontier allows farmers, veterinarians and advisors to gain farm specific insight in the relationships between effort made in implementing disease prevention measures and the effects on failure costs. Moreover, the model allows for economically efficient decision making, specific to individual farms on the allocation of preventive effort to control the multiple production disorders and gives insight in the economic impact of inefficient preventive effort.



6 References

- Ahlman, T., B. Berglund, L. Rydhmer, and E. Strandberg. 2011. Culling reasons in organic and conventional dairy herds and genotype by environment interaction for longevity. *J. Dairy Sci.* 94:1568–1575. doi:http://dx.doi.org/10.3168/jds.2010-3483.
- Alawneh, J.I., R.A. Laven, and M.A. Stevenson. 2011. The effect of lameness on the fertility of dairy cattle in a seasonally breeding pasture-based system. *J. Dairy Sci.* 94:5487–93. doi:10.3168/jds.2011-4395.
- Andersson, L., and U. Emanuelson. 1985. An epidemiological study of hyperketonaemia in Swedish dairy cows; Determinants and the relation to fertility. *Prev. Vet. Med.* 3:449–462. doi:10.1016/0167-5877(85)90006-6.
- van Asseldonk, M.A.P.M., R.J. Renes, T.J.G.M. Lam, and H. Hogeveen. 2010. Awareness and perceived value of economic information in controlling somatic cell count. *Vet. Rec.*166: 263–267. doi:10.1136/vr.b4713.
- Bar, D., Y.T. Gröhn, G. Bennett, R.N. González, J.A. Hertl, H.F. Schulte, L.W. Tauer, F.L. Welcome, and Y.H. Schukken. 2008. Effects of repeated episodes of generic clinical mastitis on mortality and culling in dairy cows. J. Dairy Sci. 91:2196–204. doi:10.3168/jds.2007-0460.
- Barkema, H.W., J.D. Van der Ploeg, Y.H. Schukken, T.J.G.M. Lam, G. Benedictus, and A. Brand. 1999a. Management Style and Its Association with Bulk Milk Somatic Cell Count and Incidence Rate of Clinical Mastitis. J. Dairy Sci. 82:1655–1663. doi:10.3168/jds.S0022-0302(99)75394-4.
- Barkema, H.W., Y.H. Schukken, T.J. Lam, M.L. Beiboer, G. Benedictus, and A. Brand. 1999b. Management practices associated with the incidence rate of clinical mastitis. *J. Dairy Sci.* 82:1643–54. doi:10.3168/jds.S0022-0302(99)75393-2.
- Barkema, H.W., J.D. Westrik, K.A.S. van Keulen, Y.H. Schukken, and A. Brand. 1994. The effects of lameness on reproductive performance, milk production and culling in Dutch dairy farms. *Prev. Vet. Med.* 20:249–259. doi:http://dx.doi.org/10.1016/0167-5877(94)90058-2.
- Berge, A.C., and G. Vertenten. 2014. A field study to determine the prevalence, dairy herd management systems, and fresh cow clinical conditions associated with ketosis in western European dairy herds. *J. Dairy Sci.* 97:2145–54. doi:10.3168/jds.2013-7163.
- van den Borne, B.H.P., G. van Schaik, T.J.G.M. Lam, and M. Nielen. 2010. Variation in herd level mastitis indicators between primi- and multiparae in Dutch dairy herds. *Prev. Vet. Med.* 96:49–55. doi:10.1016/j.prevetmed.2010.05.010.
- Brenninkmeyer, C., S. Dippel, J. Brinkmann, S. March, C. Winckler, and U. Knierim. 2013. Hock lesion epidemiology in cubicle housed dairy cows across two breeds, farming systems and countries. *Prev. Vet. Med.* 109:236–245. doi:http://dx.doi.org/10.1016/j.prevetmed.2012.10.014.
- Bruijnis, M.R.N., H. Hogeveen, and E.N. Stassen. 2010. Assessing economic consequences of foot disorders in dairy cattle using a dynamic stochastic simulation model. *J. Dairy Sci.* 93:2419–2432. doi:http://dx.doi.org/10.3168/jds.2009-2721.
- Cha, E., D. Bar, J.A. Hertl, L.W. Tauer, G. Bennett, R.N. González, Y.H. Schukken, F.L. Welcome, and Y.T. Gröhn. 2011. The cost and management of different types of clinical mastitis in dairy cows estimated by dynamic programming. *J. Dairy Sci.* 94:4476–4487. doi:http://dx.doi.org/10.3168/jds.2010-4123.
- Cha, E., J.A. Hertl, D. Bar, and Y.T. Gröhn. 2010. The cost of different types of lameness in dairy cows calculated by dynamic programming. *Prev. Vet. Med.* 97:1–8. doi:http://dx.doi.org/10.1016/j.prevetmed.2010.07.011.
- Chebel, R.C., J.E.P. Santos, J.P. Reynolds, R.L.A. Cerri, S.O. Juchem, and M. Overton. 2004. Factors affecting conception rate after artificial insemination and pregnancy loss in lactating dairy cows. *Anim. Reprod. Sci.* 84:239–255. doi:10.1016/j.anireprosci.2003.12.012.
- Dijkhuizen, A.A., and R.S. Morris. 1997. Animal health economics: principles and applications.
- Dippel, S., M. Dolezal, C. Brenninkmeyer, J. Brinkmann, S. March, U. Knierim, and C. Winckler. 2009. Risk factors for lameness in freestall-housed dairy cows across two breeds, farming systems, and countries. *J. Dairy Sci.* 92:5476–5486. doi:10.3168/jds.2009-2288.
- Dohoo, I.R., and S.W. Martin. 1984. Subclinical ketosis: prevalence and associations with production and disease. *Can. J. Comp. Med.* 48:1–5.
- Dohoo, I.R., and A.H. Meek. 1982. Somatic cell counts in bovine milk. Can. Vet. J. 23:119-25.
- van der Drift, S.G. a, R. Jorritsma, J.T. Schonewille, H.M. Knijn, and J. a Stegeman. 2012. Routine detection of hyperketonemia in dairy cows using Fourier transform infrared spectroscopy analysis of β-hydroxybutyrate and acetone in milk in combination with test-day information. *J. Dairy Sci.* 95:4886–98. doi:10.3168/jds.2011-4417.



Duffield, T. 2000. Subclinical ketosis in lactating dairy cattle. Vet. Clin. NORTH Am. Anim. Pract. 16:231-+.

- Duffield, T., D. Kelton, K. Leslie, K. Lissemore, J. Lumsden. 1997. Use of test day milk fat and milk protein to detect subclinical ketosis in dairy cattle in Ontario. *Can. Vet. JOURNAL-REVUE Vet. Can.* 38:713–718.
- Duffield, T.F., K.D. Lissemore, B.W. McBride, and K.E. Leslie. 2009. Impact of hyperketonemia in early lactation dairy cows on health and production. *J. Dairy Sci.* 92:571–580. doi:http://dx.doi.org/10.3168/jds.2008-1507.
- Dufour, S., A. Fréchette, H.W. Barkema, A. Mussell, D.T. Scholl. 2011. Invited review: effect of udder health management practices on herd somatic cell count. *J. Dairy Sci.* 94:563–79. doi:10.3168/jds.2010-3715.
- Dürr, J.W., R.I. Cue, H.G. Monardes, J. Moro-Méndez, and K.M. Wade. 2008. Milk losses associated with somatic cell counts per breed, parity and stage of lactation in Canadian dairy cattle. *Livest. Sci.* 117:225–232. doi:10.1016/j.livsci.2007.12.004.
- Engel, B., G. Bruin, G. Andre, and W. Buist. 2003. Assessment of observer performance in a subjective scoring system: visual classification of the gait of cows. *J. Agric. Sci.* 140:317–333. doi:10.1017/S0021859603002983.
- Flower, F.C., and D.M. Weary. 2006. Effect of hoof pathologies on subjective assessments of dairy cow gait. *J. Dairy Sci.* 89:139–46. doi:10.3168/jds.S0022-0302(06)72077-X.
- Fourichon, C., H. Seegers, and X. Malher. 2000. Effect of disease on reproduction in the dairy cow: a metaanalysis. *Theriogenology*. 53:1729–1759. doi:http://dx.doi.org/10.1016/S0093-691X(00)00311-3.
- Geary, U., N. Lopez-Villalobos, B. O'Brien, D.J. Garrick, and L. Shalloo. 2013. Examining the impact of mastitis on the profitability of the Irish dairy industry. *Irish J. Agric. Food Res.* 52:135–149.
- Geishauser, T., K. Leslie, D. Kelton, and T. Duffield. 2001. Monitoring for subclinical ketosis in dairy herds. *Compend. Contin. Educ. Pract. Vet.* 23:S65–S71.
- Gillund, P., O. Reksen, Y.T. Gröhn, and K. Karlberg. 2001. Body condition related to ketosis and reproductive performance in Norwegian dairy cows. *J. Dairy Sci.* 84:1390–6.doi:10.3168/jds.S0022-0302(01)70170-1.
- Green, L.E., V.J. Hedges, Y.H. Schukken, R.W. Blowey, and A.J. Packington. 2002. The impact of clinical lameness on the milk yield of dairy cows. *J. Dairy Sci.* 85:2250–6. doi:10.3168/jds.S0022-0302(02)74304.
- Gröhn, Y.T., S.W. Eicker, V. Ducrocq, and J.A. Hertl. 1998. Effect of diseases on the culling of Holstein dairy cows in New York State. *J. Dairy Sci.* 81:966–78. doi:10.3168/jds.S0022-0302(98)75657-7.
- Guard, C.L. 2008. T He Costs of Common Diseases of Dairy Cattle. 1-7.
- Gustafsson, A.H., L. Andersson, and U. Emanuelson. 1995. Influence of feeding management, concentrate intake and energy intake on the risk of hyperketonæmia in Swedish dairy herds. *Prev. Vet. Med.* 22:237–248. doi:10.1016/0167-5877(94)00423-G.
- Halasa, T., M. Nielen, R.B.M. Huirne, and H. Hogeveen. 2009a. Stochastic bio-economic model of bovine intramammary infection. *Livest. Sci.* 124:295–305. doi:10.1016/j.livsci.2009.02.019.
- Halasa, T., M. Nielen, A.P.W. De Roos, R. Van Hoorne, G. de Jong, T.J.G.M. Lam, T. van Werven, and H. Hogeveen. 2009b. Production loss due to new subclinical mastitis in Dutch dairy cows estimated with a test-day model. *J. Dairy Sci.* 92:599–606. doi:http://dx.doi.org/10.3168/jds.2008-1564.
- Hamilton, C., U. Emanuelson, K. Forslund, I. Hansson, and T. Ekman. 2006. Mastitis and related management factors in certified organic dairy herds in Sweden. *Acta Vet. Scand.* 48:11. doi:10.1186/1751-0147-48-11.
- Heikkilä, A.M., J.I. Nousiainen, and S. Pyörälä. 2012. Costs of clinical mastitis with special reference to premature culling. *J. Dairy Sci.* 95:139–150. doi:http://dx.doi.org/10.3168/jds.2011-4321.
- Hogeveen, H., K. Huijps, and T.J.G.M. Lam. 2011. Economic aspects of mastitis: new developments. *N. Z. Vet. J.* 59:16–23. doi:933054794 [pii]10.1080/00480169.2011.547165 [doi].
- Hortet, P., and H. Seegers. 1998. Loss in milk yield and related composition changes resulting from clinical mastitis in dairy cows. *Prev. Vet. Med.* 37:1–20. doi:http://dx.doi.org/10.1016/S0167-5877(98)00104-4.
- Hovi, M., A. Sundrum, and S.M. Thamsborg. 2003. Animal health and welfare in organic livestock production in Europe: current state and future challenges. *Livest. Prod. Sci.* 80:41–53. doi:http://dx.doi.org/10.1016/S0301-6226(02)00320-2.
- Huijps, K. 2009. Economic decisions in mastitis management. PhD thesis, Utrecht pp.
- Huijps, K., H. Hogeveen, T.J.G.M. Lam, and R.B.M. Huirne. 2009a. Preferences of cost factors for mastitis management among Dutch dairy farmers using adaptive conjoint analysis. *Prev. Vet. Med.* 92:351–359. doi:http://dx.doi.org/10.1016/j.prevetmed.2009.08.024.
- Huijps, K., H. Hogeveen, T.J.G.M. Lam, and A.G.J.M. Oude Lansink. 2010. Costs and efficacy of management measures to improve udder health on Dutch dairy farms. *J. Dairy Sci.* 93:115–124.



doi:http://dx.doi.org/10.3168/jds.2009-2412.

- Huijps, K., T.J. Lam, and H. Hogeveen. 2008. Costs of mastitis: facts and perception. J. Dairy Res. 75:113– 120. doi:10.1017/S0022029907002932.
- Huijps, K., S. De Vliegher, T. Lam, and H. Hogeveen. 2009b. Cost estimation of heifer mastitis in early lactation by stochastic modelling. *Vet. Microbiol.* 134:121–127. doi:http://dx.doi.org/10.1016/j.vetmic.2008.09.018.
- Huszenicza, G., S. Jánosi, M. Kulcsár, P. Kóródi, J. Reiczigel, L. Kátai, A.R. Peters, and F. De Rensis. 2005. Effects of clinical mastitis on ovarian function in post-partum dairy cows. *Reprod. Domest. Anim.* 40:199–204. doi:10.1111/j.1439-0531.2005.00571.x.
- Ingvartsen, K., R. Dewhurst, and N. Friggens. 2003. On the relationship between lactational performance and health: is it yield or metabolic imbalance that cause production diseases in dairy cattle? A position paper. *Livest. Prod. Sci.* 83:277–308. doi:10.1016/S0301-6226(03)00110-6.
- Kester, E., M. Holzhauer, and K. Frankena. 2014. A descriptive review of the prevalence and risk factors of hock lesions in dairy cows. *Vet. J.* 202:222–8. doi:10.1016/j.tvjl.2014.07.004.
- Lam, T.J.G.M., B.H.P. van den Borne, J. Jansen, K. Huijps, J.C.L. van Veersen, G. van Schaik, and H. Hogeveen. 2013. Improving bovine udder health: a national mastitis control program in the Netherlands. *J. Dairy Sci.* 96:1301–11. doi:10.3168/jds.2012-5958.
- van der Linde, C., G. de Jong, E.P.C. Koenen, and H. Eding. 2010. Claw health index for Dutch dairy cattle based on claw trimming and conformation data. *J. Dairy Sci.* 93:4883–91. doi:10.3168/jds.2010-3183.
- Manske, T., J. Hultgren, and C. Bergsten. 2002a. The effect of claw trimming on the hoof health of Swedish dairy cattle. *Prev. Vet. Med.* 54:113–129. doi:http://dx.doi.org/10.1016/S0167-5877(02)00020-X.
- Manske, T., J. Hultgren, and C. Bergsten. 2002b. Prevalence and interrelationships of hoof lesions and lameness in Swedish dairy cows. *Prev. Vet. Med.* 54:247–263. doi:10.1016/S0167-5877(02)00018-1.
- McArt, J.A.A., D. V Nydam, and G.R. Oetzel. 2012. Epidemiology of subclinical ketosis in early lactation dairy cattle. *J. Dairy Sci.* 95:5056–5066. doi:http://dx.doi.org/10.3168/jds.2012-5443.
- McArt, J.A.A., D. V Nydam, G.R. Oetzel, and C.L. Guard. 2014. An economic analysis of hyperketonemia testing and propylene glycol treatment strategies in early lactation dairy cattle. *Prev. Vet. Med.* 117:170–179. doi:10.1016/j.prevetmed.2014.06.017.
- McArt, J.A.A., D.V. Nydam, and M.W. Overton. 2015. Hyperketonemia in early lactation dairy cattle: A deterministic estimate of component and total cost per case. *J. Dairy Sci.* 98:2043–2054. doi:10.3168/jds.2014-8740.
- McInerney, J.P., K.S. Howe, and J.A. Schepers. 1992. A framework for the economic analysis of disease in farm livestock. *Prev. Vet. Med.* 13:137–154. doi:http://dx.doi.org/10.1016/0167-5877(92)90098-Z.
- Melendez, P., J. Bartolome, L.F. Archbald, and A. Donovan. 2003. The association between lameness, ovarian cysts and fertility in lactating dairy cows. *Theriogenology*. 59:927–937. doi:10.1016/S0093-691X(02)01152-4.
- Piepers, S., L. De Meulemeester, A. de Kruif, G. Opsomer, H.W. Barkema, and S. De Vliegher. 2007. Prevalence and distribution of mastitis pathogens in subclinically infected dairy cows in Flanders, Belgium. *J. Dairy Res.* 74:478–83. doi:10.1017/S0022029907002841.
- Pryce, J.E., M.D. Royal, P.C. Garnsworthy, and I.L. Mao. 2004. Fertility in the high-producing dairy cow. *Livest. Prod. Sci.* 86:125–135. doi:10.1016/S0301-6226(03)00145-3.
- Pyörälä, S. 2003. Indicators of inflammation in the diagnosis of mastitis. *Vet. Res.* 34:565–78. doi:10.1051/vetres:2003026.
- Raboisson, D., M. Mounié, and E. Maigné. 2014. Diseases, reproductive performance, and changes in milk production associated with subclinical ketosis in dairy cows: a meta-analysis and review. *J. Dairy Sci.* 97:7547–63. doi:10.3168/jds.2014-8237.
- Radostits, O.M., and S.H. Done. 2007. Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats. Elsevier/Saunders, Edinburgh [etc.].
- Rajala-Schultz, P.J., Y.T. Gröhn, C.E. McCulloch, and C.L. Guard. 1999. Effects of Clinical Mastitis on Milk Yield in Dairy Cows. *J. Dairy Sci.* 82:1213–1220. doi:http://dx.doi.org/10.3168/jds.S0022-0302(99)75344.
- Roberts, T., N. Chapinal, S.J. Leblanc, D.F. Kelton, J. Dubuc, and T.F. Duffield. 2012. Metabolic parameters in transition cows as indicators for early-lactation culling risk. *J. Dairy Sci.* 95:3057–63. doi:10.3168/jds.2011-4937.
- de Roos, A.P.W., H.J.C.M. van den Bijgaart, J. Hørlyk, and G. de Jong. 2007. Screening for subclinical ketosis in dairy cattle by Fourier transform infrared spectrometry. *J. Dairy Sci.* 90:1761–6. doi:10.3168/jds.2006-203.



- Rutherford, K.M.D., F.M. Langford, M.C. Jack, L. Sherwood, A.B. Lawrence, and M.J. Haskell. 2008. Hock Injury Prevalence and Associated Risk Factors on Organic and Nonorganic Dairy Farms in the United Kingdom. *J. Dairy Sci.* 91:2265–2274. doi:http://dx.doi.org/10.3168/jds.2007-0847.
- Santos, J.E.P., R.L.A. Cerri, M.A. Ballou, G.E. Higginbotham, and J.H. Kirk. 2004. Effect of timing of first clinical mastitis occurrence on lactational and reproductive performance of Holstein dairy cows. *Anim. Reprod. Sci.* 80:31–45. doi:10.1016/S0378-4320(03)00133-7.
- Schukken, Y.H., D.J. Wilson, F. Welcome, L. Garrison-Tikofsky, and R.N. Gonzalez. 2003. Monitoring udder health and milk quality using somatic cell counts. *Vet. Res.* 34:579–596. doi:10.1051/vetres.
- Seegers, H., F. Beaudeau, C. Fourichon, and N. Bareille. 1998. Reasons for culling in French Holstein cows. *Prev. Vet. Med.* 36:257–271. doi:10.1016/S0167-5877(98)00093-2.
- Somers, J.G.C.J., K. Frankena, E.N. Noordhuizen-Stassen, and J.H.M. Metz. 2003. Prevalence of claw disorders in Dutch dairy cows exposed to several floor systems. *J. Dairy Sci.* 86:2082–93. doi:10.3168/jds.S0022-0302(03)73797-7.
- Song, X., T. Leroy, E. Vranken, W. Maertens, B. Sonck, and D. Berckmans. 2008. Automatic detection of lameness in dairy cattle—Vision-based trackway analysis in cow's locomotion. *Comput. Electron. Agric.* 64:39–44. doi:10.1016/j.compag.2008.05.016.
- Sørensen, L.P., T. Mark, M.K. Sørensen, and S. Østergaard. 2010. Economic values and expected effect of selection index for pathogen-specific mastitis under Danish conditions. J. Dairy Sci. 93:358–369. doi:http://dx.doi.org/10.3168/jds.2009-2506.
- Sprecher, D.J., D.E. Hostetler, and J.B. Kaneene. 1997. A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. *Theriogenology*. 47:1179–1187. doi:10.1016/S0093-691X(97)00098-8.
- Stengärde, L., J. Hultgren, M. Tråvén, K. Holtenius, and U. Emanuelson. 2012. Risk factors for displaced abomasum or ketosis in Swedish dairy herds. *Prev. Vet. Med.* 103:280–6. doi:10.1016/j.prevetmed.2011.09.005.
- Suthar, V.S., J. Canelas-Raposo, a Deniz, and W. Heuwieser. 2013. Prevalence of subclinical ketosis and relationships with postpartum diseases in European dairy cows. *J. Dairy Sci.* 96:2925–38. doi:10.3168/jds.2012-6035.
- Thomsen, P.T., L. Munksgaard, and F.A. Tøgersen. 2008. Evaluation of a lameness scoring system for dairy cows. *J. Dairy Sci.* 91:119–26. doi:10.3168/jds.2007-0496.
- Vaarst, M., S. Roderick, G. Smolders, C. Leeb, M. Walkenhorst, C. Winckler, E. Gratzer, E. Stöger, L. Whistance, J. Brinkmann, S. March, S. Ivemeyer, C. Mejdell, B.I.F. Henriksen, P. Nicholas. 2011. The dialogue with farmers. *Process minimising Med. use through dialogue based Anim. Heal. Welf. Plan.*
- Vanholder, T., J. Papen, R. Bemers, G. Vertenten, and A.C.B. Berge. 2015. Risk factors for subclinical and clinical ketosis and association with production parameters in dairy cows in the Netherlands. *J. Dairy Sci.* 98:880–8. doi:10.3168/jds.2014-8362.
- van der Waaij, E.H., M. Holzhauer, E. Ellen, C. Kamphuis, and G. de Jong. 2005. Genetic parameters for claw disorders in Dutch dairy cattle and correlations with conformation traits. *J. Dairy Sci.* 88:3672–8. doi:10.3168/jds.S0022-0302(05)73053-8.
- Walker, S.L., R.F. Smith, J.E. Routly, D.N. Jones, M.J. Morris, and H. Dobson. 2008. Lameness, activity timebudgets, and estrus expression in dairy cattle. *J. Dairy Sci.* 91:4552–9. doi:10.3168/jds.2008-1048.
- Walsh, R.B., J.S. Walton, D.F. Kelton, S.J. LeBlanc, K.E. Leslie, and T.F. Duffield. 2007. The Effect of Subclinical Ketosis in Early Lactation on Reproductive Performance of Postpartum Dairy Cows. *J. Dairy Sci.* 90:2788–2796. doi:http://dx.doi.org/10.3168/jds.2006-560.
- Walsh, S.W., E.J. Williams, and A.C.O. Evans. 2011. A review of the causes of poor fertility in high milk producing dairy cows. *Anim. Reprod. Sci.* 123:127–38. doi:10.1016/j.anireprosci.2010.12.001.
- Warnick, L.D., D. Janssen, C.L. Guard, and Y.T. Gröhn. 2001. The effect of lameness on milk production in dairy cows. *J. Dairy Sci.* 84:1988–97. doi:10.3168/jds.S0022-0302(01)74642-5.
- Whay, H.R., A.E. Waterman, and A.J.F. Webster. 1997. Associations between locomotion, claw lesions and nociceptive threshold in dairy heifers during the peri-partum period. *Vet. J.* 154:155–161. doi:10.1016/S1090-0233(97)80053-6.
- Winckler, C., and S. Willen. 2001. The Reliability and Repeatability of a Lameness Scoring System for Use as an Indicator of Welfare in Dairy Cattle. *Acta Agric. Scand. Sect. A Anim. Sci.* 51:103–107. doi:10.1080/090647001316923162.
- 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.* 87:4287–94. doi:10.3168/jds.S0022-0302(04)73573-0.

