



Swedish University of Agricultural Sciences
Faculty of Veterinary Medicine and Animal Science

Genotype by environment interactions of claw health in Swedish dairy cattle in tie stalls and loose-housing

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Genotyp-miljö samspel för klövhälsa hos Svenska mjölkkor i uppbundna stallar och lösdrift

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Abstract

Claw diseases are common diseases in modern dairy production. They are painful for the cow and costly for the producer. Differences in the prevalence of claw disease depending on housing system have previously been observed. This raises the question if there are genotype by environment (GxE) interactions for claw diseases in different housing systems. To investigate this claw trimming records for Swedish Red dairy cattle (SR) and Swedish Holstein cows (SH) were retrieved from the Swedish Dairy Association. The data contained information concerning hygiene-related claw diseases; dermatitis (interdigital and digital), heel horn erosion and skin proliferation (interdigital hyperplasia and warts), and feed-related claw diseases; sole hemorrhage, sole ulcer, corkscrew claws and the combination of white line separation and double sole. The data also contained information about housing; tie stalls or loose-housing. Disease frequencies were studied depending on breed, lactation number and housing system and genetic parameters were estimated for the same breed-lactation-housing-combinations. To investigate the presence of a GxE interaction, genetic correlations between the same claw disease in different housing systems was estimated. The results showed lower frequencies of claw diseases for the SR compared to the SH. There was an increase in disease prevalence with increasing lactation number for the hygiene-related claw diseases in both breeds. For both breeds there was a higher prevalence of hygiene-related claw diseases in loose-housing, while there only was a tendency towards the same for feed-related claw diseases. Despite the smaller differences in disease frequency depending on housing system indications of GxE interactions for claw diseases in different housing systems were found mostly for the feed-related diseases.

Sammanfattning

Klövsjukdomar utgör en av de vanligaste typerna av sjukdomar i dagens mjölkproduktion. De orsakar smärta för korna samt förhöjda kostnader för lantbrukaren. I tidigare studier har sjukdomsprevalensen visat sig vara olika i olika stallsystem. Det innebär att det kan finnas genotyp-miljö samspel för klövsjukdomar i olika stallsystem. För att undersöka detta användes klövverkningsdata för svenska röda kor och svenska Holstein kor. All data som användes tillhandahölls av Svensk Mjölk. I klövverkningsdata fanns information om hygienrelaterade klövsjukdomar; dermatit (interdigital och digital), klövröta och limax/vårtor, samt foderrelaterade klövsjukdomar; sulblödningar, klövsulesår, korkskruvsklövar och hålvägg och dubbelsula som en kombinerad egenskap. Dessutom fanns information om stallsystem; uppbunden eller lösdrift. Sjukdomsfrekvensen undersöktes i de olika raserna, olika laktationer samt olika stallsystem. Genetiska parametrar skattades för samma kombinationer av ras, laktation och stallsystem och för att undersöka om det fanns några genotyp-miljö samspel skattades även genetisk korrelation för samma klövsjukdom men i olika stallsystem. Resultaten visade på en lägre prevalens för klövsjukdomar hos röda kor. För hygienrelaterade klövsjukdomar fanns en tydlig trend mot stigande sjukdomsförekomst med laktationsnummer för båda raser. Förekomsten av hygienrelaterade klövsjukdomar var högre i lösdrift för båda raser. För de foderrelaterade klövsjukdomarna fanns en liknande trend men inte lika tydlig. Trots detta var det framförallt bland foderrelaterade klövsjukdomar som det fanns indikationer på genotyp-miljö samspel för klövhälsa i olika stallsystem.

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Introduction

Background

In Sweden 39% of the dairy cows suffer from a claw disease (The Swedish Dairy Association, 2011). In the Netherlands the percentage of cows who suffer from at least one claw disease is 57.5-81% (depending on housing and flooring) (Somers *et al.*, 2003) and in Norway the prevalence is 48-71.8% (depending on housing system) (Sogstad *et al.*, 2005). This makes claw disease a very common disease in modern dairy production (Manske *et al.*, 2002a, Somers *et al.*, 2003 and The Swedish Dairy Association, 2011).

Claw diseases are painful for the cow and therefore result in changed behavior and impaired locomotion (Manske *et al.*, 2002b). Claw diseases also contribute to lower milk yield (Koenig *et al.*, 2005) and to problems with deteriorated fertility (Hinrichs *et al.*, 2006). Loose-housed cows suffering from a claw disease resulting in lameness have a lowered number of visits to the feeding table (Bach *et al.*, 2007) which could put extra stress on a high yielding dairy cow. In addition to causing problems with animal welfare, claw diseases are costly for the producer (Oskarsson, 2008 and Pedersen *et al.*, 2011) as it leads to extra costs for veterinary treatments, extra workload and involuntary culling (Oskarsson, 2008).

The heritability of claw health traits is low (van der Waaij *et al.*, 2005, Meeuwes, 2009, van der Linde *et al.*, 2010 and Johansson *et al.*, 2011). But since the claw diseases are both painful for the cows (Manske *et al.*, 2002b) and costly for the producer (Kossaibati & Esslemont, 1997 and Oskarsson, 2008) there are breeding associations that despite the low heritability have formed claw health indexes. In these indexes claw trimming registrations are used as a basis for estimating breeding values. The low heritability is somewhat compensated by a high number of progenies per bull and repeated measurements of each individual cow (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011), two factors which increases the accuracy of the selection (Simm, 2000).

During the last decade the number of cows in loose-housing has increased. Loose-housing is more strenuous for the claws, which means that the increased numbers of cows in loose-housing could lead to increased issues with claw health (Manske *et al.*, 2002b). This is verified in a study by Sogstad *et al.* (2005) where the prevalence of claw disease in loose-housed systems is higher than the prevalence of claw disease in tie stalls. It can therefore be hypothesized that claw health is a different trait in tie stalls and loose-housing.

One way to examine this is to look at genotype by environment (GxE) interactions. GxE interactions can be described as a varying performance of different genotypes in different environments. One way to study GxE interaction is by looking at the genetic correlation between the same trait in different environment. The genetic correlation shows if it is different genes controlling the trait in the two environments (Falconer, 1952). When the genetic correlation is close to zero there are completely different genes affecting the trait in the two environments and when the genetic correlation is close to plus or minus one the same genes affect the trait in the two environments (Eisen, 2005).

Aim and objective

The objective of this study is to investigate if cows in tie stalls and cows in loose-housing are equally resistant to claw diseases, if the two major Swedish dairy breeds differ in susceptibility and if the susceptibility differs in different lactations. Disease frequencies of all breed-trait-housing-lactation combinations will be studied in order to investigate the phenotypic expression of the traits. Genetic correlations between claw health traits of Swedish dairy cows in tie stalls and in loose-housing will be estimated. These genetic correlations can help to determine if there is a GxE interaction of claw health in different housing systems. It will further be investigated if there is re-ranking among sires for the same claw disease in different housing system. Lastly, the aim is to study if there is a genetic trend for the traits analyzed.

Literature review

Claw diseases

Dermatitis, digital and interdigital

Dermatitis refers to different types of eczema and is common among cows held indoors. Usually it does not cause any issues with pain. However, dermatitis can lead to problems with skin proliferation and heel horn erosion. Dermatitis is often caused by poor hygiene (Manske *et al.*, 2002b).

Interdigital and digital dermatitis are two forms of dermatitis. Interdigital dermatitis can be both husky and discharging and cause cracks in the interdigital skin. Digital dermatitis is contagious and a more aggressive form of eczema visible as strawberry-like ulcers on the back of the claw (Manske *et al.*, 2002b). The digital form of dermatitis is believed to be very contagious and caused by bacteria of *Treponema* genus within the *Spirochaetes* strain (SVA, 2012). Digital dermatitis did not show up in Sweden until the end of the 1990s' and beginning of the 2000s' (Manske *et al.*, 2002b). However, the disease might have been present in the country earlier but diagnosed and treated as heel horn erosion, a disease which can be caused by dermatitis (Manske *et al.*, 2002c). Today digital dermatitis is described as an increasing issue in Swedish herds due to the fact that when it enters a loose-housed herd it is almost impossible to become free from the disease again (Olsson, 2010).

Dermatitis is a part of both the Nordic and the Dutch Claw Health Indexes (CHI), but it is recorded differently (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011), see Table 1. In the Dutch CHI digital and interdigital dermatitis is recorded as two different traits (van der Linde *et al.*, 2010) and in the Nordic CHI digital and interdigital dermatitis is recorded as two versions of the same trait (Johansson *et al.*, 2011).

Heel horn erosion

In 2002 about half of all Swedish dairy cows had problems with heel horn erosion. Heel horn erosion means that there is a loss of bulbar horn at the back of the claw. It can be a result of either degradation of tissue due to poor hygiene in the barn or impaired production of new tissue due to for example dermatitis (Manske *et al.*, 2002b). Heel horn erosion and digital dermatitis can appear on the same areas on the claws and heel horn erosion is a common symptom of dermatitis. Therefore some of the diagnosed heel horn erosions might in fact also be cases of dermatitis (Manske *et al.*, 2002c). Heel horn erosion is a part of the Nordic CHI (Johansson *et al.*, 2011).

Skin proliferation: Interdigital hyperplasia and warts

Interdigital hyperplasia is uncontrolled skin proliferation between the claws. The protuberance can become rather big and cause the claws to sprawl. Both interdigital hyperplasia and warts can be caused by dermatitis and both diseases are related to hygiene (Manske *et al.*, 2002b). In the Nordic CHI interdigital hyperplasia and warts are combined under a claw health trait called skin proliferation (Johansson *et al.*, 2011). In the Dutch CHI only interdigital hyperplasia is recorded (van der Linde *et al.*, 2010).

Laminitis

Laminitis is a disease believed to be caused by imbalance in the rumen resulting in degradation of the claw due to impaired blood flow (Manske *et al.*, 2002b). One common cause for imbalance in the rumen is acidosis which is a result from feeding too high amounts of easily degradable carbohydrates or to small amounts of fiber (Nocek, 1995).

In the first phase of laminitis there is an increase of total blood flow and thereby also an increase in blood pressure. In the claws the increased blood pressure leads to ruptures in the blood vessel walls. This results in internal hemorrhages and swelling of the claw corium, ultimately causing pain. In the second phase of laminitis the vessels are damaged and the blood flow becomes impaired. This leads to phase three; a degradation of the claw due to lack of nutrients. In phase four the degradation of the claw leads to ruptures in the adherence between the corium and claw wall (Nocek, 1995). In the Nordic CHI sole hemorrhages, sole ulcer, white line separation, double sole and corkscrew claws are considered to be related to laminitis and thereby also considered to be feed-related (Johansson *et al.*, 2011).

Sole hemorrhage

Sole hemorrhage is characterized by a bleeding in the sole tissue (Manske *et al.*, 2002b). Sole hemorrhage can occur as a response to stress from a change in housing or flooring or from stress caused during the peri-parturient period (Bergsten & Frank, 1996). Sole hemorrhage is also considered as a sign of subclinical laminitis (Livesey *et al.*, 1998). The disease is a part of both the Nordic and the Dutch CHI (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011).

Sole ulcer

Sole ulcers arise when the claw capsule is damaged and the corium is laid bare. When the corium is laid bare there are bleeding wounds on the surface of the sole. Sole ulcers are often consequences of laminitis. The disease is both costly, hurtful and takes a long time to heal. Relapses are fairly common due to the stretch of tendons and ligaments caused by the disease which results in a higher pressure on the sole (Manske *et al.*, 2002b). Sole ulcer is a part of both the Nordic and the Dutch CHI (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011).

White line disease: White line separation, white line abscess and double sole

White line separation means that there are deep cracks in the white line. The white line is where the claw capsule and the claw bone meets in approximately 1300 small folds creating a strong but still flexible adherence. Cracks in the white line causes a loose claw wall and bleeding wounds as the corium is laid bare. Double sole occurs when a new sole of poor quality starts forming behind the old sole. Both white line separation and double sole are associated with laminitis (Manske *et al.*, 2002b). White line disease is a term referring to both white line separation and white line abscess. Wall ulcer is similar to white line abscess but with an open wound. This means that wall ulcers are related to white line separation (Bergsten, personal message, 2012).

White line disease and wall ulcer is a part of the Dutch CHI as two separate traits (van der Linde *et al.*, 2010). In the Nordic CHI both white line separation and double sole is recorded but as one trait (Johansson *et al.*, 2011).

Corkscrew claws

Animals with corkscrew claws have a deformed claw capsule (Manske *et al.*, 2002b). The background to the disease seems unclear but there are genetic correlations to sole hemorrhage, sole ulcers, and white line separation and double sole, which are diseases related to laminitis (Johansson *et al.*, 2011).

The disease is said to be mainly genetic (Manske *et al.* 2002b and Backlin, 2006). However, the heritability found by Johansson *et al.* (2011) is low, which indicates that the disease is mostly affected by environment. The disease corkscrew claw is a part of the Nordic CHI (Johansson *et al.*, 2011).

Breeding for improved claw health

The possibility of a genetic evaluation based on claw trimming registrations was studied in Sweden in 2006 (Eriksson, 2006). At the moment a claw health index (CHI) is included in the Nordic Total Merit Index (NTM) (Johansson *et al.*, 2011) and the possibility of a Dutch index has also been investigated (van der Linde *et al.*, 2010). The NTM for dairy cattle is published by the Nordic Cattle Genetic Evaluation (NAV) which is a joint evaluation between Denmark, Finland and Sweden (The Nordic Cattle Genetic Evaluation, 2012). In both indexes the claw health data is recorded by professional claw trimmers and cows can have repeated observations both within and across lactations (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011). The diseases included in the two indexes are presented in Table 1.

Table 1. Diseases in the Dutch and Nordic claw health indexes and their economic value (EV)

Disease	<u>Dutch index</u>	<u>Nordic index</u>
	EV relative to the value of sole ulcer*	EV relative to the value of sole ulcer
Dermatitis (digital & interdigital)	Interdigital dermatitis: 3.16 Digital dermatitis: 2.63	0.22
Heel horn erosion	-	0.22
Sole hemorrhage	2.13	0.13
Sole ulcer	1.00	1.00
Skin proliferation (interdigital hyperplasia & warts)	-	0.39
Interdigital hyperplasia	0.52	-
White line disease	0.27	-
White line separation & double sole	-	0.15
Cork screw claws	-	0.13

***The EV's per genetic standard deviation in the study concerning a Dutch CHI was multiplied by the genetic standard deviation and then calculated as EV's relative to the value of sole ulcer to enable comparison**

In the study concerning a Dutch CHI, the economic values (EV's) was based on costs per case of the disease and prevalence per genetic standard deviation. The highest EV relative to the value of sole ulcer was in this study on sole hemorrhage, digital dermatitis and interdigital dermatitis (van der Linde *et al.*, 2010), as seen in Table 1. In the Nordic CHI the highest EV relative to the value of sole ulcer is on sole ulcer (Pedersen *et al.*, 2011). This means that the traits are ranked differently according to EV despite the fact that the EV's are set as relative to

sole ulcer for both indexes (van der Linde *et al.*, 2010 and Pedersen *et al.*, 2011). This can be explained by the fact that even though sole ulcers had the highest cost per case in the study concerning a Dutch index, the prevalence of sole hemorrhage, digital dermatitis and interdigital dermatitis was higher than the prevalence of sole ulcers (van der Linde *et al.*, 2010).

Genetic studies on claw health

The heritability of different claw health traits is low and the genetic correlation between different claw health traits is varying (van der Waaij *et al.*, 2005, Meeuwes, 2009, van der Linde *et al.*, 2010 and Johansson *et al.*, 2011). Different studies on genetic parameters for claw health are presented in Appendix 1. The different studies have looked at different diseases and used different models for estimation of genetic parameters. The fixed and random effects used in the models can be different and in some studies the same effect was used, but measured in a different ways. Van der Linde *et al.* (2010) and van der Waaij *et al.* (2005) studied claw health in Dutch dairy cows, while Laursen *et al.*, (2009) studied claw health in Danish cows. Johansson *et al.* (2011) studied claw health in cows from Denmark, Finland and Sweden and Meeuwes (2009) studied claw health in Swedish cows.

The first study in Appendix 1 is by van der Waaij *et al.* (2005) and deals with genetic parameters for claw disorders and correlation between claw disorders and conformation traits. Heritability and correlations was in this study estimated for interdigital dermatitis and heel horn erosion (combined), sole hemorrhage, digital dermatitis, white line disease, interdigital hyperplasia, sole ulcer and chronic laminitis (van der Waaij, *et al.*, 2005). Combining heel horn erosion and interdigital dermatitis could be explained by the fact that some cases of dermatitis can be diagnosed as heel horn erosion (Manske *et al.*, 2002c). The two diseases also have a common background as hygiene-related diseases (Johansson *et al.*, 2011).

The study by Laursen *et al.*, (2009) had a different approach when estimating heritability for claw health. Claw health was defined as absence of heel horn erosion, sole ulcer, interdigital hyperplasia, interdigital phlegmon and laminitis. The data originated from the Danish Cattle Database and the recording of claw health was performed by veterinarians instead of claw trimmers as in the other studies. It was thereby a risk for the registrations being only for cows that have shown symptoms of claw disease, not for entire herds. In this study, diseases such as dermatitis, warts, sole hemorrhage and white line disease was missing (Laursen *et al.*, 2009). However, both sole hemorrhage and white line disease are related to laminitis (Manske *et al.*, 2002b). The model used in the study by Laursen *et al.*, (2009) was similar to the other models used, however the fixed effect of lactation stage was missing.

Meeuwes (2009) looked at the Holstein breed and the diseases dermatitis (digital and interdigital), heel horn erosion, sole hemorrhage and sole ulcer. In this study dermatitis and heel horn erosion as well as sole hemorrhage and sole ulcer was grouped into two combined traits. In Johansson *et al.* (2011) the traits were grouped according to background. The hygiene-related traits; dermatitis, heel horn erosion and skin proliferation was in one group and the feed-related traits; sole ulcer, sole hemorrhage and white line disease were in the other group together with cork screw claws. However there were no combined traits in the study by

Johansson *et al.* (2011). In the study by Meeuwes (2009) the combination of traits did not remarkably affect the heritability.

The cows in the study by van der Linde *et al.* (2010) had at least 75% Holstein genes and were in lactation 1-5. The claw diseases analyzed were sole hemorrhage, wall ulcer, sole ulcer, interdigital hyperplasia, white line disease and digital dermatitis and interdigital dermatitis as two separate diseases (van der Linde *et al.*, 2010). The heritability for the different claw diseases was similar for first and later lactations for most claw diseases. However, for digital dermatitis, sole ulcer and interdigital hyperplasia the genetic correlation between the same disease in different lactations ($=1$ or ≥ 2) was significantly different from one (van der Linde *et al.*, 2010). When the genetic correlation is significantly different from one the traits expressed in different environments (in this case lactations) are influenced by slightly different genes (Eisen, 2005).

Johansson *et al.* (2011) looked at the claw diseases dermatitis (digital and interdigital), heel horn erosion, skin proliferation (interdigital hyperplasia and warts), sole hemorrhage, sole ulcer, corkscrew claws and the combination of white line separation and double sole in Holstein and Red dairy cows in Denmark, Finland and Sweden. The study found breed differences in heritability for all diseases except dermatitis and white line separation. The genetic parameters were estimated for three lactations and the results were similar in all three except for skin proliferation where the heritability increased with lactation number. Genetic correlations between hygiene-related traits and feed-related traits varied between -0.13 and 0.40 (Johansson *et al.*, 2011).

As previously mentioned the genetic correlation between different claw health traits is varying (Meeuwes, 2009, van der Linde *et al.*, 2010 and Johansson *et al.*, 2011). It ranges from -0.01 for sole ulcer and dermatitis to 0.78 for sole ulcer and white line separation (Johansson *et al.*, 2011). This motivates the choice of recording several diseases in the claw health index (van der Linde *et al.*, 2010). The correlations between the different claw diseases can according to van der Linde *et al.* (2010) incite grouping of claw health diseases into hygiene-related diseases and laminitis-related diseases. This is supported by Naeslund *et al.* (2008) who suggests combining diseases according to disease background.

Environmental impact on claw health

The low heritability of claw health traits (Meeuwes, 2009, van der Linde *et al.*, 2010 and Johansson *et al.*, 2011) means that the environmental impact is large (Simm, 2000). Several studies have been made to map the effect of environment on claw health in dairy cattle (Livesey *et al.*, 1998, Somers *et al.*, 2003, Sogstad *et al.*, 2005, Telezhenko *et al.*, 2008, Baird *et al.*, 2009, Buch *et al.*, 2011 and Fjelddas *et al.*, 2011). Amongst other factors, housing system affects the prevalence of claw disease (Sogstad *et al.*, 2005).

In a study by Livesey *et al.* (1998) the amount of concentrate in the diet affected the presence of lameness and sole hemorrhages while housing in straw bedding or in cubicles affects white line separation, heel horn erosion and sole hemorrhages. Baird *et al.* (2009) shows results where the level of sole and white line lesions increased with grazing. When animals were

moved from stable to grazing, both housing and diet was changed. The fact that the study by Livesey *et al.* (1998) showed different results of the effect of housing on one hand and the effect of diet on the other is in compliance with Buch *et al.* (2011). The study by Buch *et al.* (2011) found indications for diseases related to hygiene on one hand and feed on the other only to a certain extent being controlled by the same genes.

When different forms of loose-housing were compared, straw bedding resulted in a lower number of claw disease compared to concrete flooring. Within concrete floor systems, slatted floor with manure scraper had a preferable effect on the presence of claw diseases related to hygiene such as digital dermatitis, interdigital dermatitis, heel horn erosion and interdigital hyperplasia (Somers *et al.*, 2003). This is agreed on by Fjeldaas *et al.* (2011) who found slatted floors, but without manure scraper, to be preferable compared to solid concrete floors with manure scraper or manually cleaned when trying to decrease the amount of dermatitis, heel horn erosion, sole ulcers and corkscrew claws. When it comes to laminitis-related claw lesions solid rubber floors with manure scrapers or manually cleaned had the lowest frequency of white line disease, double sole and sole hemorrhage (Fjeldaas *et al.*, 2011). One possible explanation to this could be the fact that the properties of the floor surface affect the distribution of weight on the claws (Telezhenko *et al.*, 2008). However, Fjeldaas *et al.* (2011) also found that sole ulcers had a higher frequency on rubber flooring than on slatted concrete. This is surprising since sole ulcer is considered to be a laminitis- or feed-related disease just as sole hemorrhage, white line diseases and double sole (Fjeldaas *et al.*, 2011 and Johansson *et al.*, 2011). One reason for this could be that the influence of housing system on the frequency of sole ulcers is small (Sogstad *et al.*, 2005).

GxE Interaction

What is a GxE interaction?

An animals' phenotypic performance (P) can be described as a combination of the animals genotype or genetic potential (G) and the effect of the environment it lives in (E). This can be presented in the formula: $P = G + E$. This is however not a full explanation of what determines an animals' performance. The performance of different genotypes is varying in different environments and show diversity in their response to changes in the environment. This is called genotype by environment (GxE) interaction. When adding GxE interaction the formula is: $P = G + E + GxE$ (Falconer, 1952, Simm, 2000 and Eisen, 2005).

Estimating GxE interaction

One way to study GxE interaction is by looking at genetic correlations. In this case one trait expressed in two different environments is considered as two traits. If these two traits are influenced by the same genes can be investigated by studying the genetic correlation between the traits (Falconer, 1952). When the genetic correlation is close to zero there are completely different genes affecting the two traits and when the genetic correlation is close to plus or minus one the same genes affect the two traits. When the genetic correlation is somewhere between zero and plus or minus one there are genes at some loci which affect both the traits, but they do not have all genes in common. When the two traits in fact are one trait but expressed in two environments the genetic correlation describes the differences in the

expression of a trait in different environments. In this case, a genetic correlation close to zero means that there are completely different genes controlling the same trait in the two different environments and there is a clear GxE interaction. When the genetic correlation is close to plus or minus one, there is no GxE interaction (Eisen, 2005).

Another way to study GxE interaction is through analysis of variance. This can be done by quantifying the contribution of the genomic variance, the environmental variance as well as the covariance between genotype and environment to the total variance. Phenotypes can be recorded for the same trait in animals with two or more distinct genotypes in each of two or more distinct environments. After this the statistical significance of the difference in genotype, environment and combinations of genotype and environment is estimated. The statistical significance helps quantify the contributions of genotype, environment and GxE interaction (Eisen, 2005).

Previous studies on GxE interactions in different housing systems

Feet and leg conformation and locomotion

Fatehi *et al.* (2003) estimated genetic parameters for feet and leg conformation in tie stalls and loose-housed systems. Lassen & Mark (2008) estimated genotype by housing interactions for 21 conformation traits and two workability traits. Among the conformation traits seven traits were associated to feet and legs. The feet and leg conformation traits studied, as well as the results from both papers, are found in Table 2.

Table 2. Heritability and correlations for feet and leg conformation traits in different stable systems

	Fatehi <i>et al.</i> , 2003			Lassen & Mark, 2008		
	<u>Heritability</u>		<u>Correlation</u>	<u>Heritability</u>		<u>Correlation</u>
	<u>Tie stalls</u>	<u>Loose-housed system</u>	(Standard errors)	<u>Tie stalls</u>	<u>Loose-housed system</u>	(Standard errors)
Rear legs, rear view	0.11	0.08	<u>0.79(0.07)</u>	0.15	0.16	0.92(0.06)
Rear legs, side view	0.21	0.20	<u>0.86(0.04)</u>	0.26	0.19	0.98(0.03)
Foot angle	0.12	0.11	<u>0.89(0.05)</u>	0.15	0.13	0.98(0.04)
Bone quality	0.29	0.24	<u>0.94(0.02)</u>			
Bone structure				0.30	0.24	0.99(0.02)
Claw uniformity	0.03	0.04	0.98(0.03)			
Depth of heel	0.07	0.06	0.90(0.06)			
Hock quality				0.24	0.19	0.99(0.03)
Feet and legs	0.17	0.15	<u>0.88(0.03)</u>			
Rump width				0.28	0.26	-
Rump angle				0.42	0.36	0.99(0.02)

Correlations marked *italic and underlined* can be considered significantly different from one.

The correlations in both papers are high which suggests that there are little differences between the same trait in different environments (Fatehi *et al.*, 2003 and Lassen & Mark, 2008). However, some of the correlations are significantly different from one which means that genotype by environment interactions cannot be excluded (Fatehi *et al.*, 2003).

The genetic correlation between feet and leg conformation and claw health is however questioned. According to Uggla *et al.* (2008) there are only low genetic correlations between feet and leg conformation and dermatitis, heel horn erosion, sole hemorrhage and sole ulcer. However, it is interesting to notice is that for “rear leg, side view”, a conformation trait which according to Fatehi *et al.* (2003) might be considered as two different traits in loose-housing and tie stalls, there are significant genetic correlations for dermatitis, heel horn erosion and sole ulcers (Uggla *et al.*, 2008). In the study by van der Linde *et al.* (2010) it is stated that there is a genetic correlation, but not for all feet and leg traits. Among the feet and leg traits “locomotion” and the descriptive “overall feet and legs” the majority of the correlations to different claw health traits are significantly different from zero, indicating that “locomotion” and “overall feet and legs” are correlated to claw health. The genetic correlation between locomotion and the claw diseases digital and interdigital dermatitis, sole ulcer and interdigital hyperplasia is significantly different from zero in all lactations. For the descriptive trait “overall feet and legs”, there are genetic correlations significantly different from zero for the same claw health traits except for sole ulcer in lactation number one (van der Linde *et al.*, 2010). Neither “locomotion” or “overall feet and legs” were a part of the study by Uggla *et al.* (2008).

GxE interactions for longevity

In a study by Petersson *et al.* (2005) genotype by environment interactions were estimated for length of productive life in different herds. The herd environment was characterized by their yearly average in number of primiparous cows, peak yield, protein yield and productive life. Overall the genetic correlations between length of productive life in different herds were high, however there were some re-ranking of sires between average environments and more extreme environments. The reason for this indication of GxE interaction might be that the traits which make up the length of the productive life could differ in importance depending on the environment (Petersson *et al.*, 2005). However, milk yield was seen as a part of the environment and the re-ranking could be caused by differences in yield.

Since claw disease is a reason for involuntary culling (Oskarsson, 2008) it is reasonable to believe that there is a correlation between claw disease and longevity. In a study by Meeuwes (2009) significant negative genetic correlations was found for longevity and sole hemorrhage, sole ulcer and the combined trait of sole hemorrhage and sole ulcer. Sole ulcers and sole hemorrhage are signs of laminitis which means that the diseases have a common background (Manske *et al.*, 2002b). The negative genetic correlation to longevity could be due to different factors. Firstly there is probably a direct effect since poor claw health causes culling. Sole ulcers have a poor recovery rate due to relapses being fairly common (Manske *et al.*, 2002b) which implies that the disease would cause culling. Also, sole ulcers seem to occur more frequently in high yielding cows, but since the disease decreases the milk production these animals cannot show their full potential (Hultgren *et al.*, 2004).

Impact of possible GxE interactions of claw health in different housing systems in future breeding plans

Since May 6th, 2010 it is no longer allowed to build new tie stalls in Sweden (The Swedish Board of Agriculture. 2010). This means that tie stalls are being phased out. A GxE interaction for claw health in different housing systems would indicate that there are different genes influencing the trait in different housing systems (Eisen, 2005). If this is the case it might be motivated to do the genetic evaluation on recordings only from loose-housing systems to assure that the genes selected are suited for the environment which will be provided in the future. In this case the assumption is that claw health in loose-housing is a different trait than claw health in tie stall and that the trait for selection is claw health in loose-housing. However, there are other factors to have in mind before making a change like this. For example the formula for genetic progress:

$$\textit{Genetic progress} = (\textit{accuracy of selection} * \textit{Standard deviation of the additive genetic variation} * \textit{intensity of selection}) / \textit{generation interval}$$

This formula shows that a decreased number of cows in the genetic evaluation, which this change would imply, would lead to a lower accuracy of selection. With a lower accuracy there is a risk of choosing a breeding animal with overestimated breeding value which results in lower selection intensity for the sought trait. Thereby the genetic progress can be slowed down if the number of cows in the genetic evaluation is decreased. However, it is not necessary to discard all information from cows housed in tie stalls. If a bivariate analysis is used for estimation of genetic parameters, correlations from cows housed in tie stalls will still affect the result of the breeding evaluation for cows in loose-housing.

Material and Method

Data

The datasets contained claw trimmings from 136 548 Swedish Red dairy cows (SR) and 152 399 Swedish Holstein cows (SH) with registrations for a maximum of three lactations and with a maximum of four registrations per lactation. In these two datasets the maximum amount of claw trimming registrations for one cow was nine. The total number of claw trimming registrations was 412 329 for SR and 467 520 for SH. However, some registrations lacked information concerning housing system and after removing those, the number of cows was 121 925 SR and 137 628 SH and the number of claw trimming registrations was 375 249 for SR and 428 930 for SH. The claw trimming registrations were carried out in the period of 2003 to 2011 and retrieved from the Swedish Dairy Association. In the SR dataset there was a mean of 49 daughters per bull and in the SH the mean was 35 daughters per bull. Sire-maternal grand sire (S-MGS) pedigree files corresponding to the claw datasets were also provided by the Swedish Dairy Association. The SR pedigree file contained 18 113 registrations while the SH pedigree file contained 24 834 registrations.

Variables used

The variables present in the datasets are presented in Appendix 2 together with variable information. However, not all variables in these datasets were used. Since the purpose of the study was to study claw health in different housing systems, variables with information about the diseases dermatitis (interdigital and digital), heel horn erosion, skin proliferation (interdigital hyperplasia and warts), sole hemorrhage, sole ulcer, white line separation and double sole combined and corkscrew claws were used as well as variables concerning housing system. The choice of variables tested as effects for the model was based on previous studies on claw health (van der Waaij *et al.*, 2005, Ugglå *et al.*, 2008, Meeuwes, 2009, van der Linde *et al.*, 2010 and Johansson *et al.*, 2011).

The cows were registered in four different housing systems: loose-housed without isolated walls, loose-housed with isolated walls, tie stalls with short cubicles and tie stalls with long cubicles. The distribution of cows in different housing systems and in different lactation numbers is presented in Table 3. The aim of the study was to compare loose-housing and tie stalls and therefore registrations for cows in loose-housing without isolation and loose-housing with isolation were merged into loose-housing and the same for registrations for cows in tie stalls where tie stalls with short cubicles were merged with tie stalls with long cubicles.

Table 3. The distribution of SR and SH in different housing systems and different number of lactations

Lactation	Housing system	Number of SR	Number SH	Total
1	Loose-housed without isolation	16621	26950	43571
	Loose-housed with isolation	48877	62995	111872
	Tie stalls with short cubicles	44411	40141	84552
	Tie stalls with long cubicles	11593	7157	18750
	Total Loose-housed	65498	89945	155443
	Total Tie stalls	56004	47298	103302
	Total in lactation 1	121502	137243	258745
2	Loose-housed without isolation	9871	14557	24428
	Loose-housed with isolation	28025	34859	62884
	Tied-up on short cubicles	24590	21624	46214
	Tied-up on long cubicles	6384	3701	10085
	Total Loose-housed	37896	49416	87312
	Total Tie stalls	30974	25325	56299
	Total in lactation 2	68870	74741	143611
3	Loose-housed without isolation	5154	6956	12110
	Loose-housed with isolation	13905	16125	30030
	Tied-up on short cubicles	12100	9952	22052
	Tied-up on long cubicles	3128	1648	4776
	Total Loose-housed	19059	23081	42140
	Total Tie stalls	15228	11600	26828
	Total in lactation 3	34287	34681	68968

The different claw diseases were scored by educated claw trimmers. Dermatitis was scored as no disease (0), interdigital dermatitis (1) and digital dermatitis (2). Heel horn erosion, Sole hemorrhage and Sole ulcer were scored as no disease (0), disease (1), and severe disease (2). Corkscrew claws and the two disease pairs White line separation together with Double sole and Skin proliferation (Interdigital hyperplasia and Warts) were scored on 0-1 scale, where zero (0) denotes no disease and one (1) denotes disease.

Statistical methods

When calculating frequencies only the first registration for each cow in each lactation was used. The original dataset was derived from a larger dataset with registrations from Sweden, Finland and Denmark and the observations for the different diseases were corrected for heterogeneous variances. In order to study claw disease frequencies the corrected observations had to be rounded off to 0, 1 or 2. Frequencies were calculated for the different claw diseases in the different breeds, different housing systems and for three lactations.

The difference between claw disease frequencies in different housing systems was further studied by using least square means (SAS, 2003). Least square means was estimated with the fixed effects calving age, month and year of calving, week in milk and housing system. Due to limitations in memory it wasn't possible to use any random effects when estimating the least square means. This made it likely that the significance of the differences was overestimated and therefore the significance level of the test was set to 0.01 instead of the commonly used 0.05. The matrix was found singular and a generalized inversed matrix was used. For the least square means estimations the observations for the claw diseases were kept variance corrected. This could cause differences in the results when comparing the frequencies with the least square means.

The claw diseases were studied one by one and grouped according to disease background. The disease-groups were feed-related claw diseases; sole hemorrhage, sole ulcer, corkscrew claws and white line separation and double sole, and hygiene-related claw diseases; dermatitis, heel horn erosion and skin proliferation (warts and interdigital hyperplasia). Two additional traits called feed-related claw diseases and hygiene-related claw diseases were formed by taking the highest score for either of the feed-related or hygiene-related claw diseases at each claw trimming.

Fixed effects for the diseases combined with housing system in each breed and lactation were examined using PROC GLM (SAS, 2003). Due to the results the fixed effects of calving age, month and year of calving, 5-year herd effect and week in milk was used. It was not possible to include housing system as a fixed effect as a 5-year herd effect was already included in the model. Housing system was coupled with disease in order to enable keeping the 5-year herd effect and later bivariate analyses for estimation of genetic parameters. The variables in the model are listed below.

Variables in the model (more information concerning the variables is found in Appendix 2):

$$Y_{ijklmn} = CA_i + MY_j + 5Y_k + WM_l + sID_m + a_n + e_{ijklmn}$$

Y_{ijklmn}	Claw disease in either loose-housing or tie stalls
CA_i	fixed effect, calving age in months (SH - lactation 1: $i=1, \dots, 20$, lactation 2: $i=1, \dots, 34$, lactation 3: $i=1, \dots, 38$) (SR - lactation 1: $i=1, \dots, 20$, lactation 2: $i=1, \dots, 34$, lactation 3: $i=1, \dots, 38$)
MY_j	fixed effect, month and year of calving (SH - lactation 1: $j=1, \dots, 109$, lactation 2: $j=1, \dots, 100$, lactation 3: $j=1, \dots, 90$) (SR - lactation 1: $j=1, \dots, 109$, lactation 2: $j=1, \dots, 98$, lactation 3: $j=1, \dots, 88$)
$5Y_k$	fixed effect, 5-year herd effect (SH: $k=1, \dots, 3792$, SR: $k=1, \dots, 3856$)
WM_l	fixed effect, week in milk (SH - lactation 1: $l=1, \dots, 62$, lactation 2: $l=1, \dots, 62$, lactation 3: $l=1, \dots, 62$) (SR - lactation 1: $l=1, \dots, 62$, lactation 2: $l=1, \dots, 62$, lactation 3: $l=1, \dots, 62$)
sID_m	random sire effect (SH: $m=1, \dots, 4036$, SR: $m=1, \dots, 2574$)
a_n	random animal effect (since one animal can have several registrations/lactation) (SH: $n=1, \dots, 137408$, SR: $n=1, \dots, 121608$)
e_{ijklmn}	random residual effect

Estimation of genetic parameters was done using average information (AI)-REML in the DMU package (Madsen and Jensen, 2007). A series of bivariate analyses were performed for each claw disease and for the two groups of claw diseases for two different housing systems, loose-housed and tie stalls, for the two breeds and the three lactations individually using the bivariate sire model shown below.

Bivariate sire model:

$$\begin{bmatrix} Y_{tie} \\ Y_{loose} \end{bmatrix} = \begin{bmatrix} X_1 & 0 \\ 0 & X_2 \end{bmatrix} \begin{bmatrix} fix_{tie} \\ fix_{loose} \end{bmatrix} + \begin{bmatrix} Z_1 & 0 \\ 0 & Z_1 \end{bmatrix} \begin{bmatrix} PE_{tie} \\ PE_{loose} \end{bmatrix} + \begin{bmatrix} Z_2 & 0 \\ 0 & Z_2 \end{bmatrix} \begin{bmatrix} Sire_{tie} \\ Sire_{loose} \end{bmatrix} + \begin{bmatrix} e_{tie} \\ e_{loose} \end{bmatrix}$$

Where $\begin{bmatrix} X_1 & 0 \\ 0 & X_2 \end{bmatrix}$, is the incidence matrix and $\begin{bmatrix} fix_{tie} \\ fix_{loose} \end{bmatrix}$ is the vector of the fixed effects,

$\begin{bmatrix} Z_1 & 0 \\ 0 & Z_1 \end{bmatrix}$, is the incidence matrix and $\begin{bmatrix} PE_{tie} \\ PE_{loose} \end{bmatrix}$ is the vector of the permanent environment,

$\begin{bmatrix} Z_2 & 0 \\ 0 & Z_2 \end{bmatrix}$, is the incidence matrix and $\begin{bmatrix} Sire_{tie} \\ Sire_{loose} \end{bmatrix}$ is the vector of sires and $\begin{bmatrix} e_{tie} \\ e_{loose} \end{bmatrix}$ is the vector of the residuals.

$Var \begin{bmatrix} PE_{tie} \\ PE_{loose} \end{bmatrix} = PE \otimes I$, where I is the identity matrix, \otimes is the Kronecker product and

$$\text{where, } PE = \begin{bmatrix} \sigma_{PE_{tie}}^2 & 0 \\ 0 & \sigma_{PE_{loose}}^2 \end{bmatrix}$$

$Var \begin{bmatrix} Sire_{tie} \\ Sire_{loose} \end{bmatrix} = G_0 \otimes A$, where A is the relationship matrix, \otimes is the Kronecker product

$$\text{and where, } G_0 = \begin{bmatrix} 1/4 \cdot \sigma_{stie}^2 & \sigma_{stie,loose} \\ \sigma_{stie,loose} & 1/4 \cdot \sigma_{sloose}^2 \end{bmatrix}$$

$Var \begin{bmatrix} e_{tie} \\ e_{loose} \end{bmatrix} = R \otimes I$, where I is the identity matrix, \otimes is the Kronecker product and where,

$$R = \begin{bmatrix} \sigma_{e_{tie}}^2 & 0 \\ 0 & \sigma_{e_{loose}}^2 \end{bmatrix}$$

The genetic correlations between the same trait in different housing systems combined with its standard error was then used as an indicator for GxE interactions. Genetic correlations were not estimated across breed or lactations.

For the combined diseases, feed-related claw diseases and hygiene-related claw diseases, breeding values were predicted using DMU4 (Madsen and Jensen, 2007). These breeding values were then used to further investigate the presence of GxE interactions. By using the estimated breeding values, sires were ranked and the ranking was compared across different housing systems. In addition, genetic trends computed from the breeding values were plotted by birth year of bull, starting on a birth year of 1997 since the first claw trimming registrations were from 2003.

Results

Dermatitis (interdigital and digital)

Prevalence of dermatitis in different breeds, lactations and housing systems

The frequency of dermatitis in different housing systems was investigated in two ways. Firstly as a percentage of sick cows in either loose-housing or tie stalls and secondly as least square means for loose-housing and tie stalls. The percentage of sick cows is shown in Figure 1 and the least square means are seen in Table 4. As seen in Figure 1, Swedish Holstein cows (SH) have a higher percentage of Dermatitis than Swedish Red dairy cows (SR) in both housing systems. The percentage of dermatitis is increasing with lactation number for both breeds in both housing systems. In Figure 1 there is a visibly higher frequency of dermatitis in loose-housing compared to tie stalls and according to the least square means in Table 4 the differences between the housing systems within the same breed and lactation are significant.

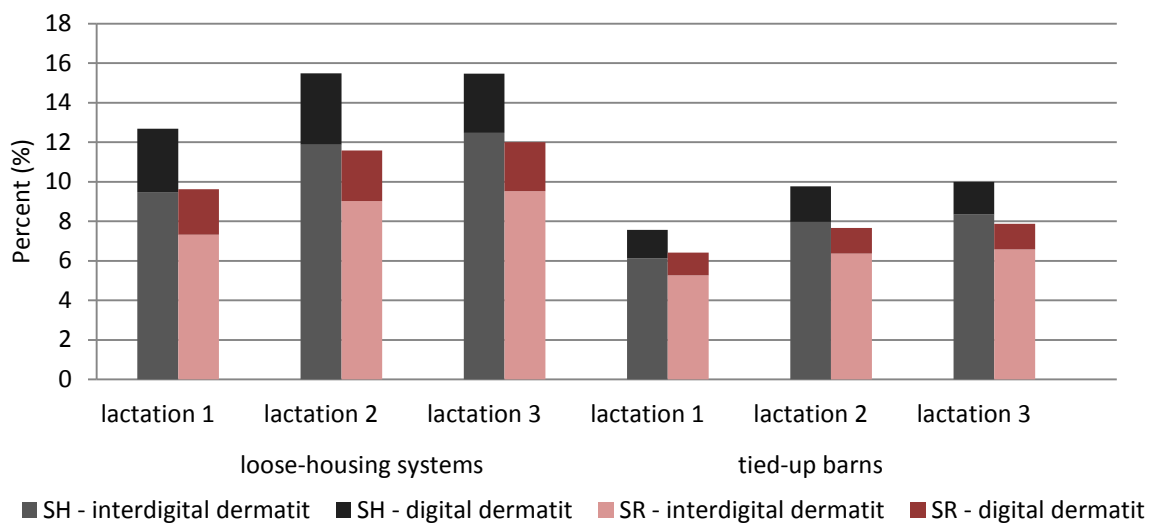


Figure 1. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with dermatitis in different housing systems

Table 4. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference) for dermatitis in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		Ls-means in loose-housing	Ls-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.2008	0.1506	S
	Lactation 2	0.2132	0.1694	S
	Lactation 3	0.1688	0.1313	S
SR	Lactation 1	0.1689	0.1264	S
	Lactation 2	0.1833	0.1411	S
	Lactation 3	0.1683	0.1297	S

Genetic parameters for dermatitis (interdigital and digital)

Genetic parameters for dermatitis are presented in Table 5 and show differences in heritability and genetic variances depending on breed, lactation number and housing system. The heritability and genetic variances for SH are decreasing with lactation number in both housing systems but there is no decrease in heritability or genetic variances depending on lactation number for SR. The decrease in heritability and genetic variances with lactation number for SH is in contrast to the increase in disease frequency with lactation number. For SH cows the heritability is higher in tie stalls in lactation one, similar in both housing systems in lactation two and higher in loose-housing in lactation three. For SR the heritability is higher in tie stalls for all lactations. The higher heritability for SR in tie stalls compared to loose-housing cannot be explained by a higher genetic variance since the genetic variances are comparable for both housing systems. This indicates that it is a lower phenotypic variance which causes the higher heritability in tie stalls. None of the genetic correlations between dermatitis in loose-housing and tie stalls was significantly different from one.

Table 5. Heritability (h^2) and additive genetic variance (σ_A^2) for dermatitis in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactations 1-3 and genetic correlation (r_G) with Standard Error (S.E.) between dermatitis in loose-housing and dermatitis in tie stalls

		h^2_{loose}	$\sigma_{A,loose}^2$	h^2_{tied}	$\sigma_{A,tied}^2$	$r_{G,loose-tied}$	S.E
SH	lactation 1	0,0505	0,0097	0,0656	0,0102	0,9562	0,0307
	lactation 2	0,0456	0,0084	0,0469	0,0074	0,9018	0,0625
	lactation 3	0,0383	0,0062	0,0204	0,0030	0,8417	0,2060
SR	lactation 1	0,0318	0,0054	0,0402	0,0053	0,9517	0,0408
	lactation 2	0,0377	0,0063	0,0430	0,0056	0,9758	0,0403
	lactation 3	0,0352	0,0055	0,0462	0,0057	0,9999	0,0961

Heel horn erosion

Prevalence of heel horn erosion in different breeds, lactations and housing systems

The percentage of cows with heel horn erosion in different housing systems is shown in Figure 2 together with information about breed and lactation number. As seen in this figure the percentage of cows with heel horn erosion is almost equal between breeds but slightly higher for Swedish Red dairy cows (SR) compared to Swedish Holstein cows (SH). This is in agreement with Figure 2 in Appendix 3 which shows disease percentage regardless of housing system for the two breeds. The influence of lactation number on the percentage of cows with heel horn erosion is the same for both breeds with an increase in percentage of cows with heel horn erosion with increasing lactation number. Figure 2 also shows that there are differences in disease frequency depending on housing systems, with higher frequencies in loose-housing compared to tie stalls. According to the least square means in Table 6 these differences are significant for both breeds and all lactations.

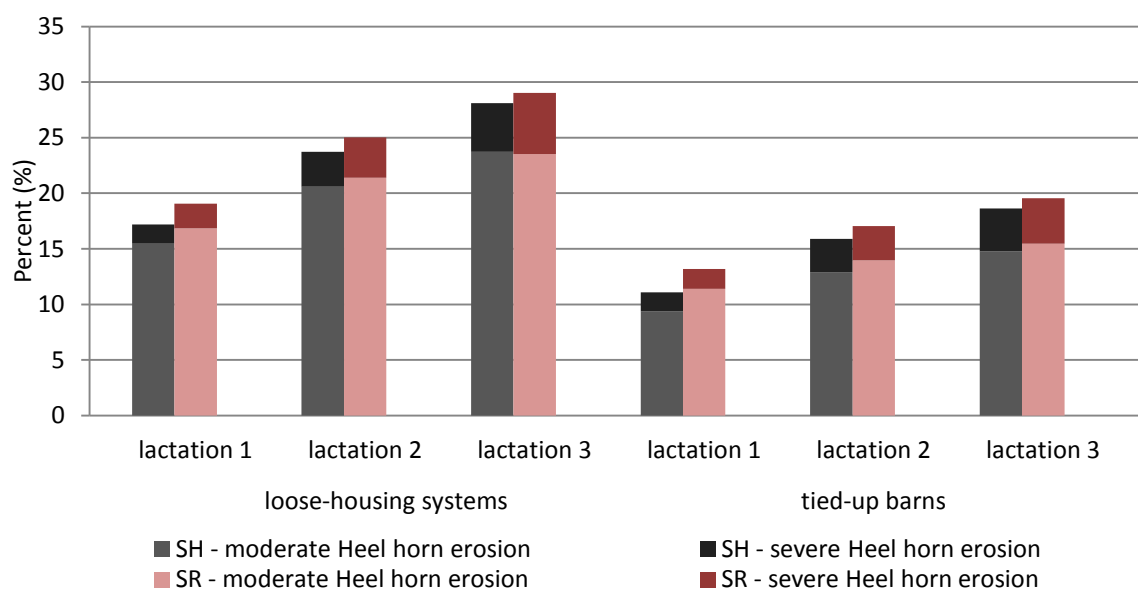


Figure 2. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with heel horn erosion in different housing systems

Table 6. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference) for heel horn erosion in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.2559	0.1886	S
	Lactation 2	0.3703	0.2888	S
	Lactation 3	0.4260	0.3350	S
SR	Lactation 1	0.2698	0.2015	S
	Lactation 2	0.3957	0.3093	S
	Lactation 3	0.4312	0.3307	S

Genetic parameters for heel horn erosion

Genetic parameters for heel horn erosion for (SH) and (SR) in different housing systems are presented in Table 7. For heel horn erosion the heritability is slightly higher in tie stalls except for SR in lactation one, as seen in Table 7. The genetic variance is also slightly higher in tie stalls for four of the six breed-lactation combinations, but the differences are very small. Since the disease frequency is lower in tie stalls an explanation for the slightly higher genetic variances cannot be found in higher frequencies. The higher heritability in tie stalls compared to loose-housing could be due to a lower phenotypic variance in tie stalls. For SH the heritability is highest in lactation two and for SR it is highest in lactation three. The heritability for heel horn erosion is higher than the heritability for the other two hygiene-related diseases, dermatitis and skin proliferation. None of the genetic correlations for heel horn erosion in different housing systems were significantly different from one.

Table 7. Heritability (h^2) and additive genetic variance (σ_A^2) for heel horn erosion in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactations 1-3

and genetic correlation (r_G) with Standard Error (S.E.) between heel horn erosion in loose-housing and heel horn erosion in tie stalls

		h_{loose}^2	$\sigma_{A,loose}^2$	h_{tied}^2	$\sigma_{A,tied}^2$	$r_{G,loose-tied}$	S.E.
SH	lactation 1	0,0577	0,0103	0,0592	0,0098	0,9979	0,0165
	lactation 2	0,0593	0,0132	0,0674	0,0145	1,0000	0,0238
	lactation 3	0,0573	0,0148	0,0614	0,0159	1,0000	0,0781
SR	lactation 1	0,0583	0,0113	0,0450	0,0081	0,9884	0,0160
	lactation 2	0,0540	0,0124	0,0586	0,0131	0,9629	0,0289
	lactation 3	0,0617	0,0160	0,0665	0,0170	0,9795	0,0385

Skin proliferation (interdigital hyperplasia and/or warts)

Prevalence of skin proliferation (interdigital hyperplasia and/or warts) in different breeds, lactations and housing systems

The percentage of cows with skin proliferation is presented in Figure 3. Swedish Holstein cows (SH) has a higher percentage of cows with skin proliferation compared to Swedish Red dairy cows (SR). This is in accordance with the frequencies of claw disease in different breeds seen in Appendix 3, Figure 3. For both breeds and all housing systems the percentage of sick cows is increasing with lactation number. There are also differences in skin proliferation frequency in the different housing systems and the higher frequency is found in loose-housing systems, see Figure 3. As seen in Table 8 the differences are significant for both breeds and all three lactations.

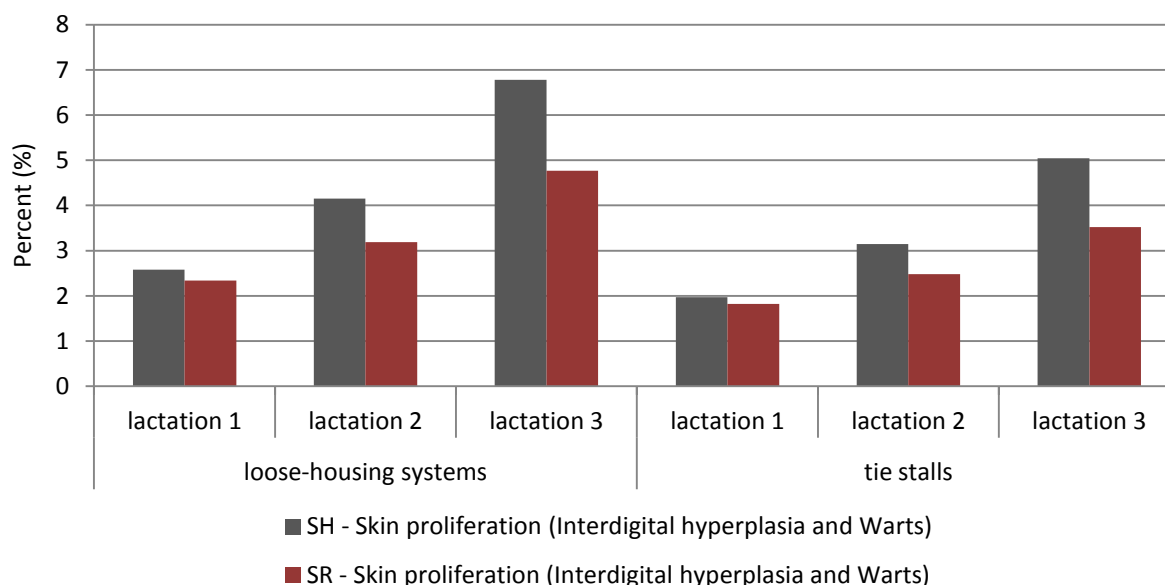


Figure 3. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with skin proliferation (interdigital hyperplasia and warts) in different housing systems

Table 8. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference,) for skin proliferation in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.0367	0.0320	S
	Lactation 2	0.0672	0.0581	S
	Lactation 3	0.0904	0.0775	S
SR	Lactation 1	0.0345	0.0307	S
	Lactation 2	0.0594	0.0542	S
	Lactation 3	0.0782	0.0655	S

Genetic parameters for skin proliferation (interdigital hyperplasia and warts)

Genetic parameters for skin proliferation in SH and SR in different lactation and housing systems are presented in Table 9. Considering the low frequencies of the disease it is not surprising to find low heritability and genetic variance compared to the other two hygiene-related claw diseases; dermatitis and heel horn erosion. The heritability is higher in loose-housing in both breeds and all lactations except for SR in lactation one. The genetic variance is also slightly higher in loose-housing, even though the differences are very small. Both the heritability and genetic variance are increasing with lactation number for both breeds. This means that the higher disease frequency in loose-housing compared to tie stalls and the increase in disease frequency with lactation number could be due to a larger genetic impact in loose housing and an increasing genetic impact with lactation number. The genetic correlation for skin proliferation in loose-housing and tie stalls is significantly different from one for SH cows in lactation one. The standard errors for the correlations between skin proliferation in different housing system were large for all breed-lactation combinations which is most likely caused by the very low frequencies of this trait.

Table 9. Heritability (h^2) and additive genetic variance (σ_A^2) for skin proliferation in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3 and genetic correlation (r_G) with Standard Error (S.E.) between skin proliferation in loose-housing and skin proliferation in tie stalls

		h^2_{loose}	$\sigma^2_{A,loose}$	h^2_{tied}	$\sigma^2_{A,tied}$	$r_{G,loose-tied}$	S.E.
SH	lactation 1	0,0081	0,0002	0,0079	0,0002	0,6453*	0,1699
	lactation 2	0,0164	0,0007	0,0086	0,0003	0,6873	0,1895
	lactation 3	0,0287	0,0020	0,0099	0,0006	0,8394	0,2906
SR	lactation 1	0,0032	0,0001	0,0039	0,0001	0,9985	0,2913
	lactation 2	0,0077	0,0003	0,0048	0,0002	0,9983	0,2092
	lactation 3	0,0136	0,0007	0,0090	0,0004	0,9983	0,2735

*Genetic correlations significantly different from one.

Sole hemorrhage

Prevalence of sole hemorrhage in different breeds, lactations and housing systems

The percentage of cows with sole hemorrhage is higher for Swedish Holstein cows (SH) compared to Swedish Red dairy cows (SR) in all three lactations and in all housing systems, as seen in Figure 4. Unlike the frequencies of dermatitis, heel horn erosion and skin proliferation there is no clear increase in disease frequency with lactation number, see Figures 1-4. In Figure 4 there are no visible trends towards higher frequency in either of the housing systems. However, in Table 10 the differences are presented as least square means instead, and here differences are clearer with significantly higher least square means in loose-housing than in tie stalls for both breeds and lactations, except for SH cows in lactation three.

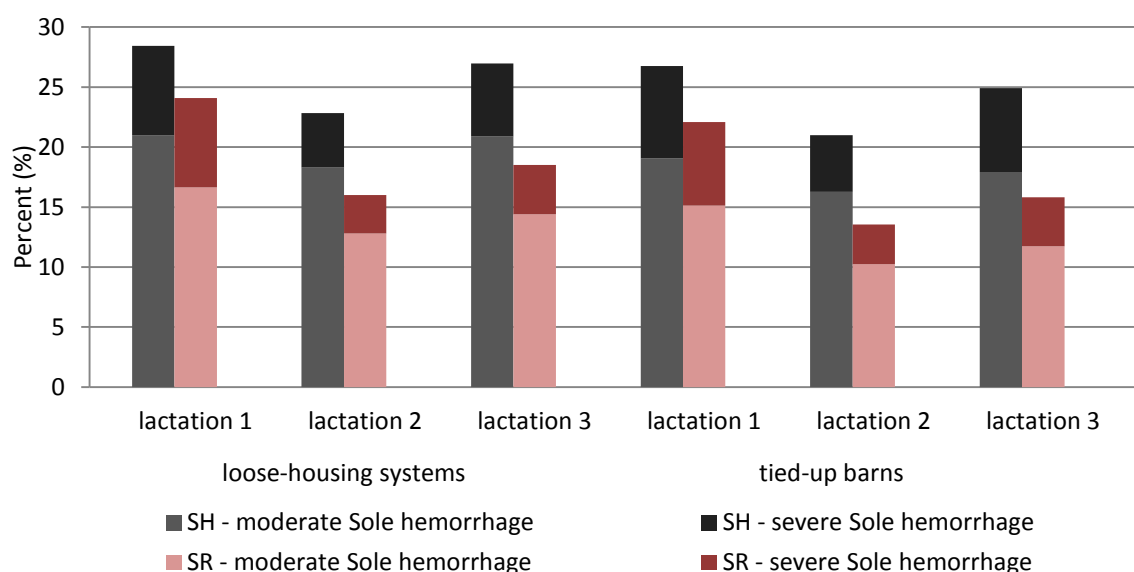


Figure 4. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with sole hemorrhage in different housing systems

Table 10. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference, NS=no significant difference) for sole hemorrhage in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		Least square means in loose-housing	Least square means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.3116	0.2925	S
	Lactation 2	0.3298	0.3124	S
	Lactation 3	0.4048	0.3950	NS
SR	Lactation 1	0.2832	0.2653	S
	Lactation 2	0.2366	0.2069	S
	Lactation 3	0.3004	0.2674	S

Genetic parameters for sole hemorrhage

Genetic parameters for sole hemorrhage in different breeds, lactation and housing systems are presented in Table 11. The results show no clear differences in heritability depending on housing system. For SH the heritability is highest in lactation one and is then decreasing with lactation number. For SR it is highest for lactation two and lowest for lactation three. The genetic variances in the different housing systems are also difficult to map. This is in

accordance with the disease frequencies where there are no visible differences between housing systems or trends in the disease frequency depending on lactation number, as seen in Figure 4. Apart from the correlation for SR in lactation two, none of the genetic correlation for sole hemorrhage in the two different housing systems was significantly different from one.

Table 11. Heritability (h^2) and additive genetic variance (σ_A^2) for sole hemorrhage in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3 and genetic correlation (r_G) with Standard Error (S.E.) between sole hemorrhage in loose-housing and sole hemorrhage in tie stalls

		h^2_{loose}	$\sigma_{A,\text{loose}}^2$	h^2_{tied}	$\sigma_{A,\text{tied}}^2$	$r_{G,\text{loose-tied}}$	S.E.
SH	lactation 1	0,0358	0,0094	0,0426	0,0118	0,9296	0,0387
	lactation 2	0,0346	0,0080	0,0389	0,0096	0,9226	0,0569
	lactation 3	0,0305	0,0082	0,0280	0,0083	0,9997	0,0995
SR	lactation 1	0,0430	0,0117	0,0437	0,0121	0,9697	0,0227
	lactation 2	0,0484	0,0100	0,0455	0,0092	0,8598*	0,0602
	lactation 3	0,0294	0,0068	0,0311	0,0072	0,9421	0,0827

*Genetic correlations significantly different from one.

Sole ulcer

Prevalence of sole ulcer in different breeds, lactations and housing systems

The percentage of cows with sole ulcer is presented in Figure 5. Swedish Holstein cows (SH) show a higher percentage of the disease compared to Swedish Red dairy cows (SR) in all lactations and housing systems. When looking at the distribution of the disease over different lactations there seem to be a drop in percentage of sick cows in lactation two. Just as for sole hemorrhage there are no visible differences in disease frequency in the different housing systems. The differences are also presented with least square means in Table 12. However, this table shows that there are three combinations of breed and lactation number which have significant differences between least square means in different lactations. For all of these the least square means are higher in tie stalls compared to loose-housing.

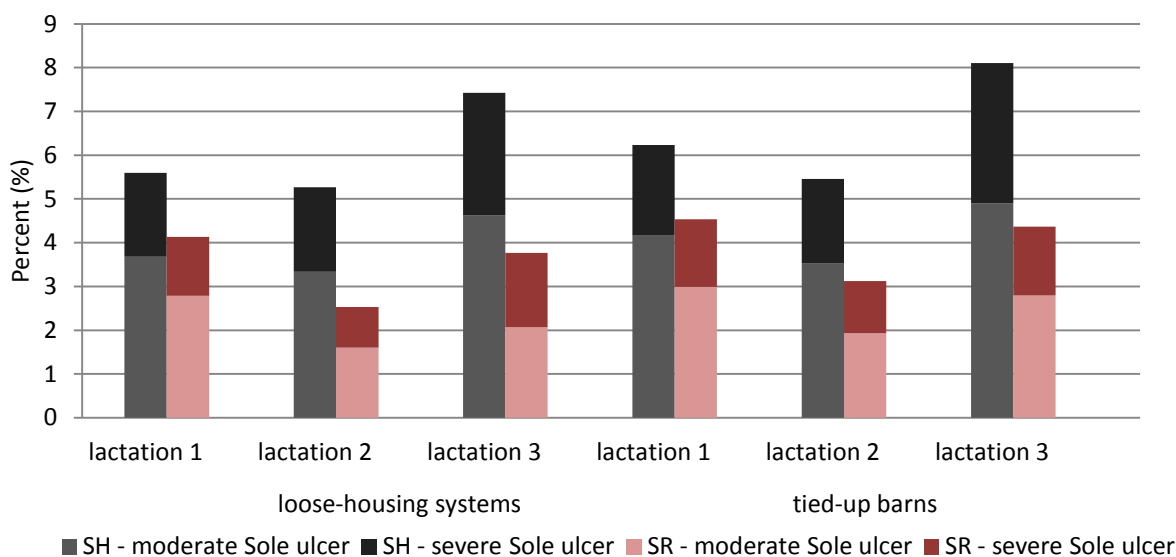


Figure 5. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with sole ulcer in different housing systems

Table 12. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference, NS=no significant difference) for sole ulcer in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		ls-means in loose-housing	ls-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.0701	0.0752	S
	Lactation 2	0.0995	0.0989	NS
	Lactation 3	0.1340	0.1432	NS
SR	Lactation 1	0.0449	0.0515	S
	Lactation 2	0.0566	0.0642	S
	Lactation 3	0.0629	0.0687	NS

Genetic parameters for sole ulcer

The genetic parameters for sole ulcers are presented in Table 13. The heritability for the disease is differing between breeds and lactations. For SH the heritability is highest in loose-housing in lactation three. For SR the heritability is highest in lactation one and in lactation two it is very low. In general the difference in both heritability and genetic variance in SH between the two housing systems is increasing with lactation number. This is in contrast with the disease frequency where significant differences between the housing systems are found only in lactation one. For SH there is a tendency towards higher heritability in loose-housing, in SR the situation is the opposite. The genetic correlation for sole ulcer in the two different housing systems is significantly different from one for SH in lactation one.

Table 13. Heritability (h^2) and additive genetic variance (σ_A^2) for sole ulcer in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3 and genetic correlation (r_G) with Standard Error (S.E.) between sole ulcer in loose-housing and sole ulcer in tie stalls

		h^2_{loose}	$\sigma_{A,loose}^2$	h^2_{tied}	$\sigma_{A,tied}^2$	$r_{G,loose-tied}$	S.E.
SH	lactation 1	0,0440	0,0042	0,0451	0,0047	0,9208*	0,0365
	lactation 2	0,0425	0,0047	0,0331	0,0037	0,9995	0,0473
	lactation 3	0,0574	0,0091	0,0354	0,0064	0,9999	0,0774
SR	lactation 1	0,0235	0,0015	0,0266	0,0021	0,9951	0,0231
	lactation 2	0,0007	0,0008	0,0072	0,0005	0,9998	0,1369
	lactation 3	0,0180	0,0015	0,0235	0,0022	0,9484	0,1104

*Genetic correlations significantly different from one.

White line separation and/or double sole

Prevalence of white line separation and/or double sole in different breeds, lactations and housing systems

Figure 6 shows the percentage of cows with white line separation and/or double sole in different breeds, lactation number and in different housing systems. When investigating breed differences for the frequency of white line separation and double sole, there are only small differences visible in Figure 6. However, when looking at Appendix 3, Figure 3, the frequency of Swedish Holstein cows (SH) with white line separation and/or double sole is higher than the frequency of Swedish Red dairy cows (SR) with the diseases. The percentage of cows with white line separation and/or double sole is increasing with lactation number in both breeds, which is shown in Figure 6. When looking at the disease frequency in different housing systems there is a visible difference in Figure 6. Cows in loose-housing have a higher disease frequency compared to cows in tie stalls. According to the least square means seen in Table 14, these differences are significant.

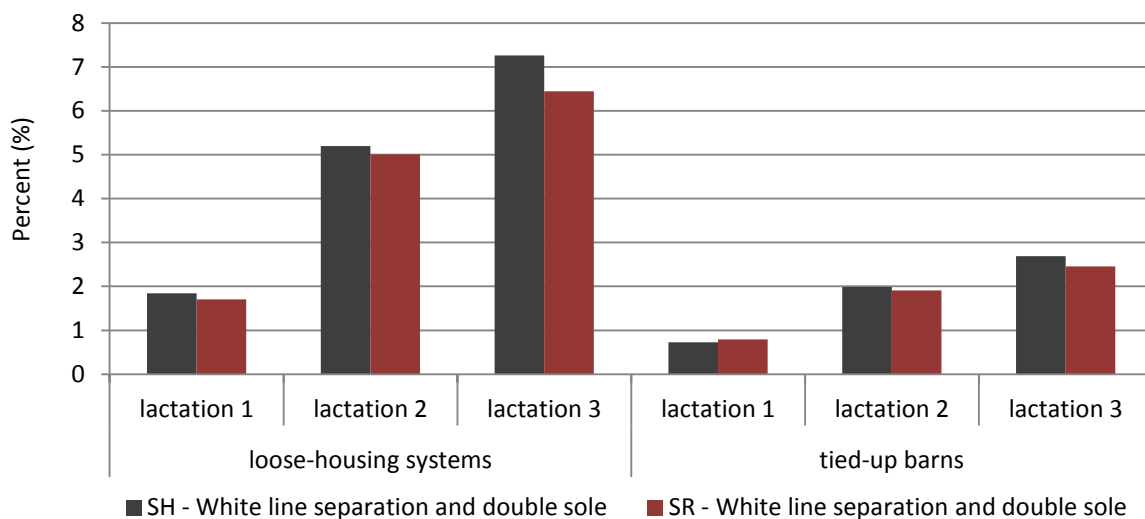


Figure 6. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with white line separation and/or double sole in different housing systems

Table 14. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference) for white line separation and/or double sole in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.0325	0.0172	S
	Lactation 2	0.0596	0.0284	S
	Lactation 3	0.0688	0.0291	S
SR	Lactation 1	0.0311	0.0187	S
	Lactation 2	0.0556	0.0252	S
	Lactation 3	0.0609	0.0242	S

Genetic parameters for white line separation and/or double sole

To further explore the differences between loose-housing and tie stalls, genetic parameters were estimated for the disease in the two different housing systems. In general the heritability and genetic variation is higher for SH, as seen in Table 15. However, the tendency of the heritability and the genetic variation is similar for both breeds with higher heritability and genetic variation with increasing lactation number and higher heritability in loose-housing systems for both breeds, see Table 15. This is in accordance with the disease frequencies which are higher in loose-housing and increasing with lactation number. Only the genetic correlation for SR in lactation one is significantly different from one.

Table 15. Heritability (h^2) and additive genetic variance (σ_A^2) for white line separation and/or double sole in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3 and genetic correlation (r_G) with Standard Error (S.E.) between white line separation and/or double sole in loose-housing and white line separation and/or double sole in tie stalls

		h^2_{loose}	$\sigma_{A,loose}^2$	h^2_{tied}	$\sigma_{A,tied}^2$	$r_{G,loose-tied}$	S.E.
SH	lactation 1	0,0298	0,0008	0,0113	0,0002	1,0000	0,0805
	lactation 2	0,0687	0,0035	0,0337	0,0009	0,9514	0,0602
	lactation 3	0,0848	0,0051	0,0392	0,0012	1,0000	0,1007
SR	lactation 1	0,0209	0,0005	0,0119	0,0002	0,9262	0,0642
	lactation 2	0,0402	0,0018	0,0228	0,0005	1,0000	0,0540
	lactation 3	0,0526	0,0028	0,0352	0,0009	0,9791	0,0690

Corkscrew claws

Prevalence of corkscrew claws in different breeds, lactations and housing systems

The frequency of corkscrew claws in different housing systems is presented in Figure 7 together with information about breed and lactation number. As seen in Figure 7, the frequency of corkscrew claws is increasing with lactation number, but only for cows held in loose-housing. For cows held in tie stalls there is no clear increase in frequency depending on lactation number. There is also a breed difference which causes the differences due to housing systems being larger for Swedish Red dairy cows (SR) compared to Swedish Holstein cows

(SH). Therefore there is a trend towards SR in tie stalls being less susceptible to corkscrew claws than SR in loose housing while there is no significant trend for SH, as seen in Table 16.

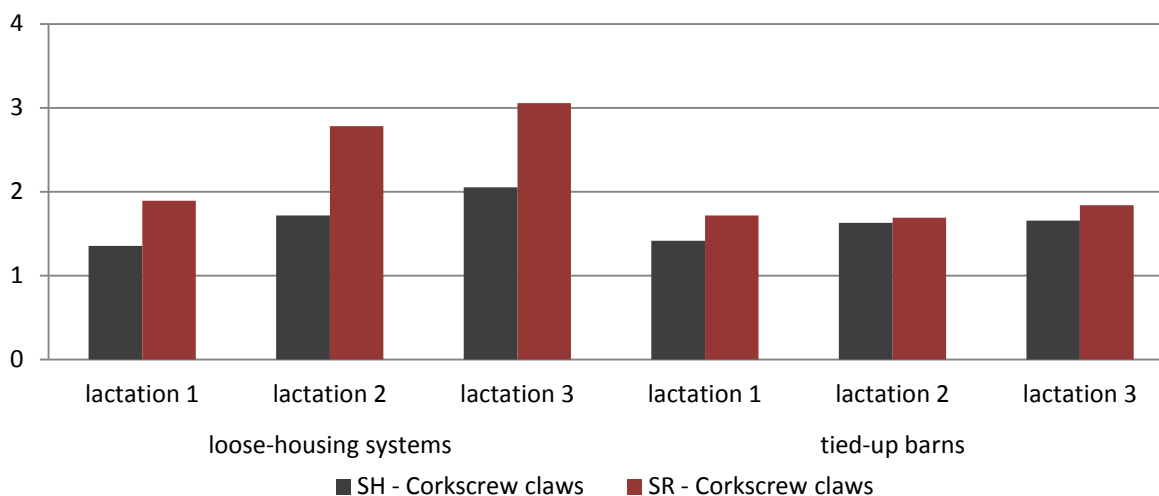


Figure 7. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with corkscrew claws in different housing systems

Table 16. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference, NS=no significant difference) for dermatitis in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.0188	0.0202	NS
	Lactation 2	0.0303	0.0313	NS
	Lactation 3	0.0186	0.0169	NS
SR	Lactation 1	0.0252	0.0213	S
	Lactation 2	0.0327	0.0198	S
	Lactation 3	0.0622	0.0507	S

Genetic parameters for corkscrew claws

As seen in Table 17, the heritability for corkscrew claws in different housing systems is highest in tie stalls for SH in all lactations and in the third lactation for SR. In SH in tie stalls there is a clear increase in heritability with increasing lactation number. For SR it is the opposite with decreasing heritability with lactation number for both housing systems. The highest heritability for SH is found in tie stalls for cows in the third lactation. For SR the highest heritability is for loose-housed cows in lactation one. The difference between breeds is in accordance with the disease frequencies where only SR has significant disease frequency differences for the two housing systems. There are also frequency differences between housing system where SR have a clear increase in disease frequency with lactation number, but only in loose-housing. The difference of the effect of lactation number depending on housing system is not seen among the genetic parameters where the heritability in decreasing with lactation number for both housing systems. There is a genetic correlation significantly different from one for SH in lactation three.

Table 17. Heritability (h^2) and additive genetic variance (σ_A^2) for corkscrew claws in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3

and genetic correlation (r_G) with Standard Error (S.E.) between corkscrew claws in loose-housing and corkscrew claws in tie stalls

		h^2_{loose}	$\sigma^2_{A,loose}$	h^2_{tied}	$\sigma^2_{A,tied}$	$r_{G,loose-tied}$	S.E.
SH	lactation 1	0,0156	0,0002	0,0162	0,0003	0,9999	0,0648
	lactation 2	0,0193	0,0003	0,0203	0,0004	1,0000	0,1008
	lactation 3	0,0163	0,0003	0,0918	0,0016	0,4584*	0,2008
SR	lactation 1	0,0500	0,0011	0,0420	0,0008	0,9888	0,0208
	lactation 2	0,0477	0,0014	0,0383	0,0007	1,0000	0,0260
	lactation 3	0,0244	0,0007	0,0281	0,0006	0,9998	0,1492

*Genetic correlations significantly different from one.

Feed-related and hygiene-related diseases

Prevalence of feed-related and hygiene-related claw diseases in different breeds, lactations and housing systems

As seen in Figures 1, 2 and 3 the frequency of the hygiene-related claw diseases dermatitis, heel horn erosion and skin proliferation differs between breeds. For dermatitis (Figure 1) and skin proliferation (Figure 3) the frequency is higher for Swedish Holstein cows (SH) and for heel horn erosion (Figure 2) it is higher for Swedish Red dairy cows (SR). When the diseases are combined there is a higher frequency for SH, as seen in Figure 8. The three diseases follow the same pattern concerning the influence of lactation number where a higher lactation number gives a higher disease frequency. This pattern is also seen in Figure 8 where the diseases have been combined. For dermatitis, heel horn erosion and skin proliferation (Figures 1, 2 and 3) there is a clear trend towards higher frequencies in loose-housing. This is seen for the hygiene-related diseases combined as well, see Figure 8. As seen in Table 18 these differences are significant.

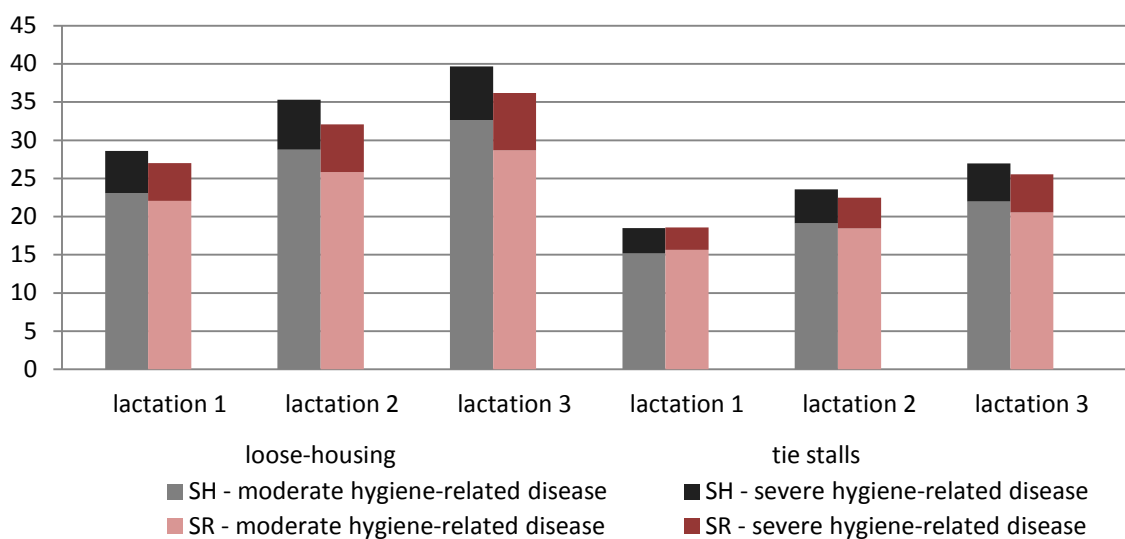


Figure 8. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with hygiene-related claw diseases in different housing systems

Table 18. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference,) for hygiene-related diseases in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.3477	0.2312	S
	Lactation 2	0.4579	0.3266	S
	Lactation 3	0.4730	0.3326	S
SR	Lactation 1	0.3359	0.2311	S
	Lactation 2	0.4410	0.3244	S
	Lactation 3	0.4608	0.3324	S

Sole hemorrhage, sole ulcer, white line separation, double sole and corkscrew claws are considered feed-related diseases. The results in Figures 4, 5 and 6 show that the claw disease frequency is higher in SH compared to SR for sole hemorrhage, sole ulcer, white line separation and double sole. For corkscrew claws (Figure 7) the frequency is higher in SR. When combining the four diseases, see Figure 9, the frequency is higher in SH. Sole hemorrhage and sole ulcer (Figures 4 and 5) have similar patterns for frequency depending on lactation number where the frequency decreases from lactation one to lactation two only to increase again for lactation three. For white line separation (Figure 6) there is a clear trend towards higher frequencies with higher lactation number. For corkscrew claws (Figure 7) there is a similar pattern, but only for cows in loose-housing. Figure 9 which show the diseases combined into feed-related diseases show no clear trend in disease frequency over different lactations.

Figures 4 and 5, show no clear difference between the frequency of sole hemorrhage or sole ulcer in different housing systems. However, Table 10 and 12 show tendencies towards higher frequency of sole hemorrhage in loose-housing and higher frequency of sole ulcer in tie stalls. For white line separation and double sole, see Figure 6 and Table 14, there is a clear trend towards higher disease frequency in loose-housing. There is a higher frequency of corkscrew claws for SR in loose-housing, as seen in Figure 7. In Figure 9, where the diseases are combined, there is a slightly higher disease frequency in loose-housing. This is confirmed by the least square means for the combined feed-related diseases, as shown in Table 19.

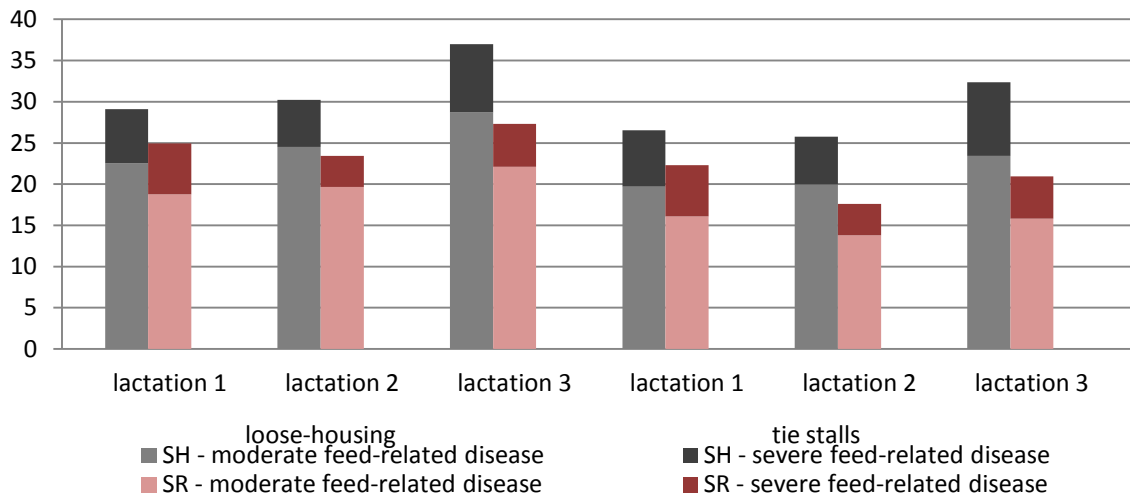


Figure 9. Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) with feed-related claw diseases in different housing systems

Table 19. Least square means (ls-means), and significance of the difference in least square means in loose-housing and tie stalls (S=significant difference) for feed related diseases in Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different lactations

		LS-means in loose-housing	LS-means in tie stalls	Significance (sign. level: 0.01)
SH	Lactation 1	0.3672	0.3337	S
	Lactation 2	0.4211	0.3728	S
	Lactation 3	0.5256	0.4810	S
SR	Lactation 1	0.3313	0.2916	S
	Lactation 2	0.3272	0.2633	S
	Lactation 3	0.3831	0.3124	S

Genetic parameters for grouped claw diseases

Table 20 summarizes the genetic parameters found for the two combinations of claw diseases; feed-related claw diseases and hygiene-related claw diseases. For the feed-related traits there is a slightly higher heritability and genetic variation for the trait in loose-housing, except for SH in lactation one. This is in accordance with a slightly higher frequency of feed-related diseases in loose-housing. For SH the heritability and the genetic variance of feed-related claw diseases is increasing with lactation number. A similar pattern cannot be seen for the frequency of the feed-related diseases where there is no real pattern for the influence of lactation number. For SR the heritability is lowest in lactation two and the frequency of the feed-related claw diseases in SR is also lowest in lactation two. When looking at hygiene-related diseases the heritability does not follow any pattern. There are several genetic correlations for the same group of diseases in the two different housing systems significantly different from one, especially for feed-related diseases indicating more GxE interactions for feed-related diseases compared to hygiene-related diseases.

Table 20. Heritability (h^2) and additive genetic variance (σ_A^2) for grouped diseases in loose-housing (loose) and in tie stalls (tied) for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in lactation 1-3

and genetic correlation (r_G) with Standard Error (S.E.) between the grouped diseases in loose-housing and the grouped diseases in tie stalls

			h^2_{loose}	$\sigma^2_{A,loose}$	h^2_{tied}	$\sigma^2_{A,tied}$	$r_{G,loose \rightarrow tied}$	S.E.
Feed-related claw diseases ¹	SH	lactation 1	0.0432	0.0069	0.0448	0.0070	0.9313*	0.0326
		lactation 2	0.0552	0.0092	0.0465	0.0074	0.8657*	0.0538
		lactation 3	0.0574	0.0104	0.0493	0.0089	1.0000	0.0812
	SR	lactation 1	0.0519	0.0079	0.0490	0.0070	0.9592	0.0228
		lactation 2	0.0480	0.0073	0.0402	0.0051	0.8815*	0.0527
		lactation 3	0.0536	0.0088	0.0450	0.0064	0.7990	0.1014
Hygiene-related claw diseases ²	SH	lactation 1	0.0501	0.0077	0.0576	0.0071	0.9377*	0.0310
		lactation 2	0.0489	0.0082	0.0605	0.0086	0.9822	0.0265
		lactation 3	0.0515	0.0089	0.0276	0.0042	1.0000	0.1049
	SR	lactation 1	0.0487	0.0071	0.0385	0.0048	0.9742	0.0229
		lactation 2	0.0467	0.0070	0.0509	0.0071	0.9629	0.0317
		lactation 3	0.0636	0.0101	0.0615	0.0093	0.9899	0.0348

*Correlations significantly different from one.

¹Feed-related claw diseases: Sole hemorrhage, sole ulcer, Corkscrew claws, White line separation and Double sole, ²Hygiene-related claw diseases: Dermatitis (Interdigital and digital), Heel horn erosion, Skin proliferation (Interdigital hyperplasia and warts).

Changes in ranking of sires depending on housing system

Breeding values were estimated for feed-related claw diseases and hygiene-related claw diseases in both breeds and in lactation one and three. These breeding values were then used to investigate if there were any GxE interactions causing re-ranking among sires. The top 20-lists are shown in Tables 21 and 22. In Table 21 results for feed-related claw diseases are shown and re-ranking occurs among SH sires in lactation one and SR sires in lactation one and three. In Table 22 results are shown for hygiene-related diseases. Re-ranking occurred for the same lactations for both breeds as for feed-related diseases. However, the re-ranking was not as cogent for hygiene-related diseases as for the feed-related diseases. This is supported by the higher number of genetic correlations significantly different from one for feed-related diseases compared to hygiene-related diseases, see Table 20.

Table 21. Ranking of sires depending on breeding values estimated for feed-related claw diseases in either loose-housing or tie stalls. The shaded rankings show re-ranking depending on housing system. The bold figures represent genetic correlations of the trait in the two housing systems with the standard error within brackets.

Feed-related claw diseases							
Swedish Holstein				Swedish Red			
lactation 1		lactation 3		lactation 1		lactation 3	
Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls
0.9313 (0.0326)*		1.0000 (0.0812)		0.9592 (0.0228)		0.7990 (0.1014)	
1	2	1	1	1	1	1	10
2	3	2	2	2	4	2	1
3	1	3	3	3	3	3	3
4	5	4	4	4	2	4	8
5	6	5	5	5	7	5	5
6	7	6	6	6	6	6	7
7	9	7	7	7	5	7	2
8	14	8	8	8	8	8	4
9	10	9	9	9	18	9	82
10	13	10	10	10	11	10	39
11	16	11	11	11	10	11	9
12	4	12	12	12	9	12	28
13	26	13	13	13	13	13	11
14	17	14	14	14	15	14	17
15	23	15	15	15	21	15	66
16	43	16	16	16	12	16	80
17	29	17	17	17	14	17	23
18	30	18	18	18	19	18	13
19	37	19	19	19	20	19	76
20	8	20	20	20	17	20	83

*Genetic correlation significantly different from one.

Table 22. Ranking of sires depending on breeding values estimated for hygiene-related claw diseases in either loose-housing or tie stalls. The marked rankings show re-ranking depending on housing system. The bold figures represent genetic correlation of the trait in the two housing systems with the standard error within brackets.

Swedish Holstein				Swedish Red			
lactation 1		lactation 3		lactation 1		lactation 3	
Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls	Ranking in loose-housing	Ranking in tie stalls
0.9377(0.0310)*		1.0000(0.1049)		0.9742(0.0229)		0.9899(0.0348)	
1	1	1	1	1	1	1	1
2	2	2	2	2	2	2	2
3	3	3	3	3	3	3	3
4	4	4	4	4	4	4	4
5	5	5	5	5	5	5	5
6	6	6	6	6	8	6	6
7	7	7	7	7	6	7	8
8	8	8	8	8	7	8	7
9	9	9	9	9	10	9	10
10	13	10	10	10	9	10	9
11	10	11	11	11	15	11	11
12	9	12	12	12	11	12	12
13	19	13	13	13	16	13	13
14	23	14	14	14	12	14	14
15	26	15	15	15	13	15	15
16	20	16	16	16	14	16	16
17	18	17	17	17	17	17	17
18	17	18	18	18	20	18	18
19	15	19	19	19	21	19	19
20	25	20	20	20	18	20	21

*Genetic correlation significantly different from one.

Genetic trends for claw diseases

The estimated genetic parameters were also used in order to investigate genetic trends for sires in different housing systems. The results, seen in Figures 10 to 13, are shown as estimated breeding values for susceptibility to claw disease over sire birth year. The trends show differences between the two breeds, types of claw diseases and housing systems. For the feed-related claw diseases in lactation one SH has a favorable genetic trend indicating a decrease in susceptibility to feed-related claw diseases. For SR the trend is the opposite. In lactation three both breeds show an adverse trend towards higher susceptibility to feed-related claw diseases. Both SH and SR has a favorable genetic trend towards decreasing susceptibility to hygiene-related claw diseases in lactation one. In lactation three however, only SR has continued the favorable trend.

Differences in genetic trend for the same disease in different housing systems are affected by genetic correlation between the traits in the different housing systems. The lower genetic correlations found for feed-related claw diseases in different housing systems have resulted in

genetic trends differing from each other depending on for which housing system the estimation has been performed. In lactation one for feed-related claw diseases the difference between the genetic trend in different housing system has become smaller for SR, but larger for SH. For SR in lactation three there is an increasing difference in the genetic trend depending on housing system, but for SH the difference between genetic trends in the different housing system is small.

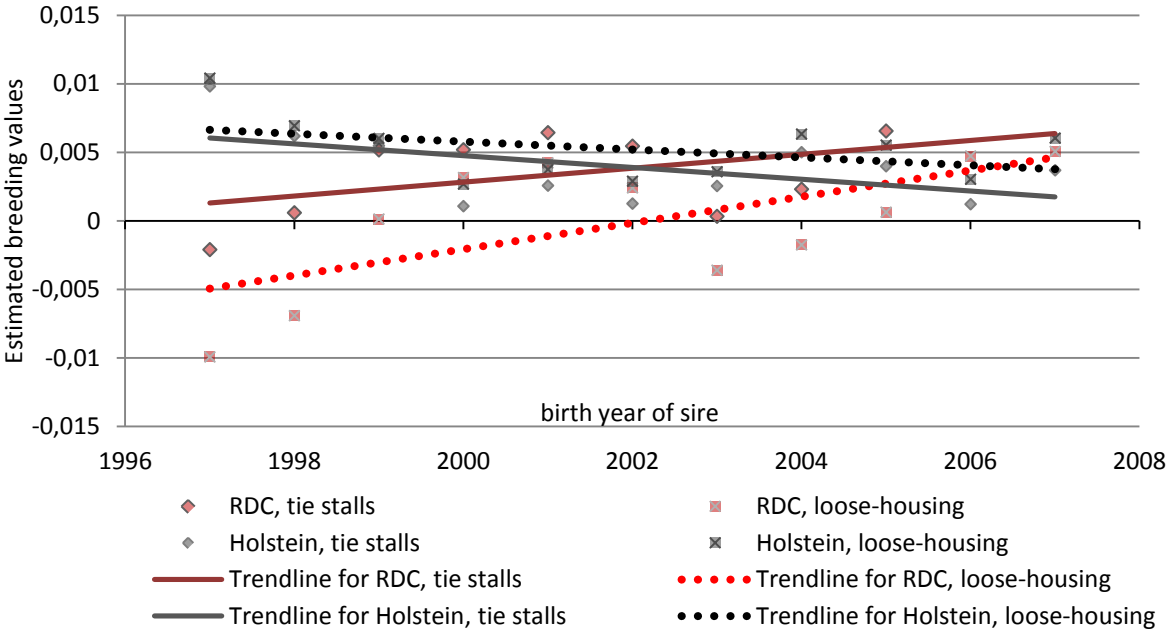


Figure 10. Genetic trend by birth year of sire for feed-related claw diseases in lactation one for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different housing systems, where a negative trend indicates a favorable decrease in susceptibility to claw disease.

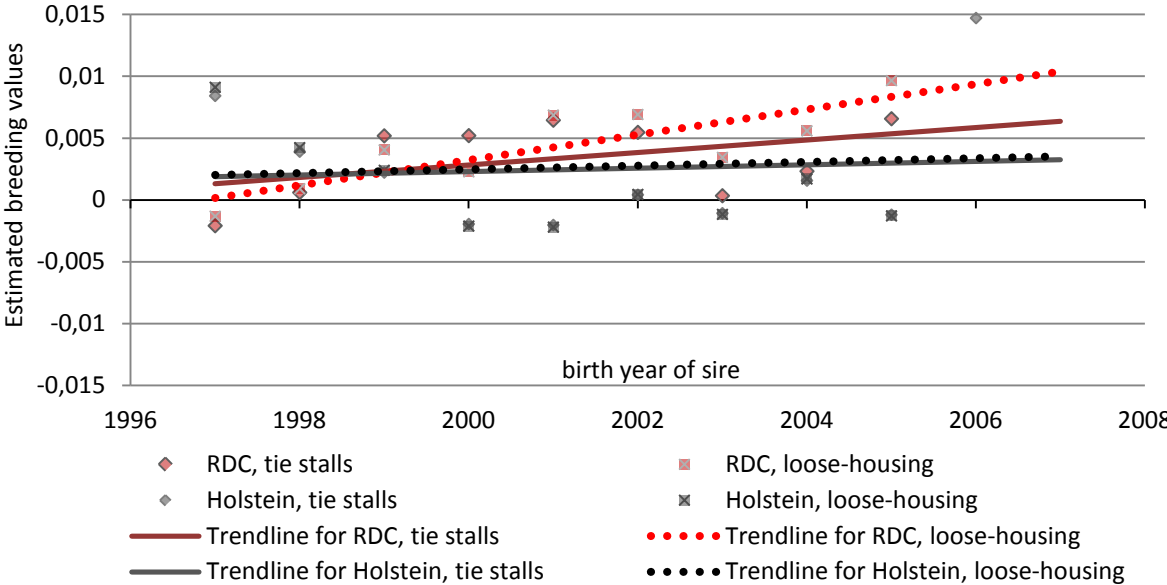


Figure 11. Genetic trend by birth year of sire for feed-related claw diseases in lactation three for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different housing systems, where a negative trend indicates a favorable decrease in susceptibility to claw disease.

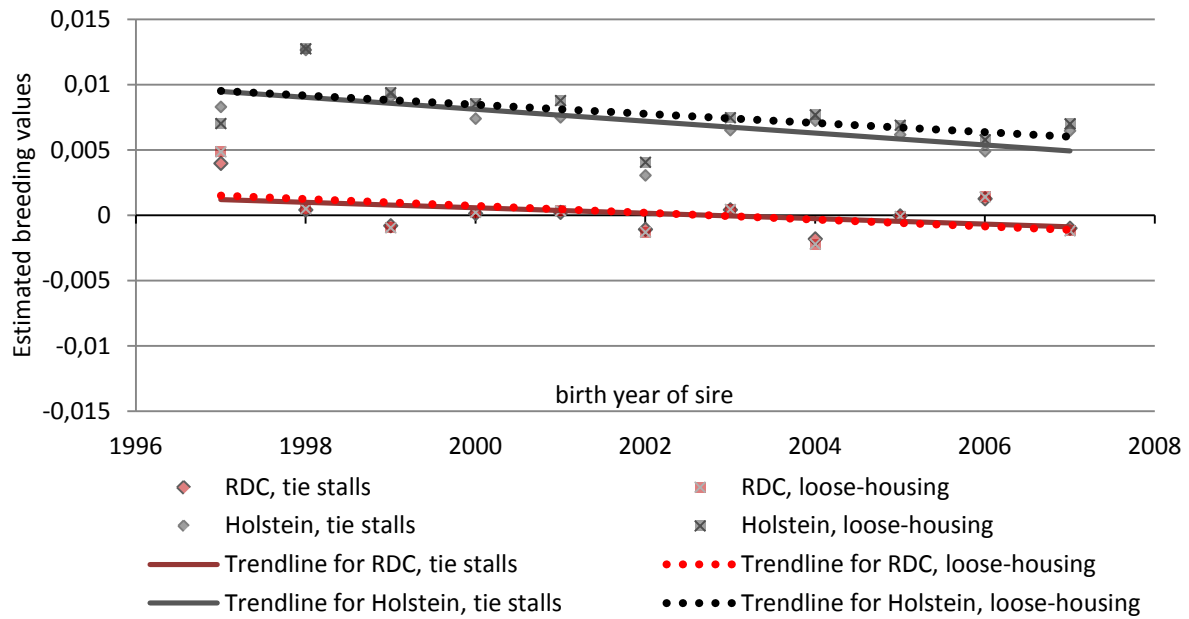


Figure 12. Genetic trend by birth year of sire for hygiene-related claw diseases in lactation one for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different housing systems, where a negative trend indicates a favorable decrease in susceptibility to claw disease.

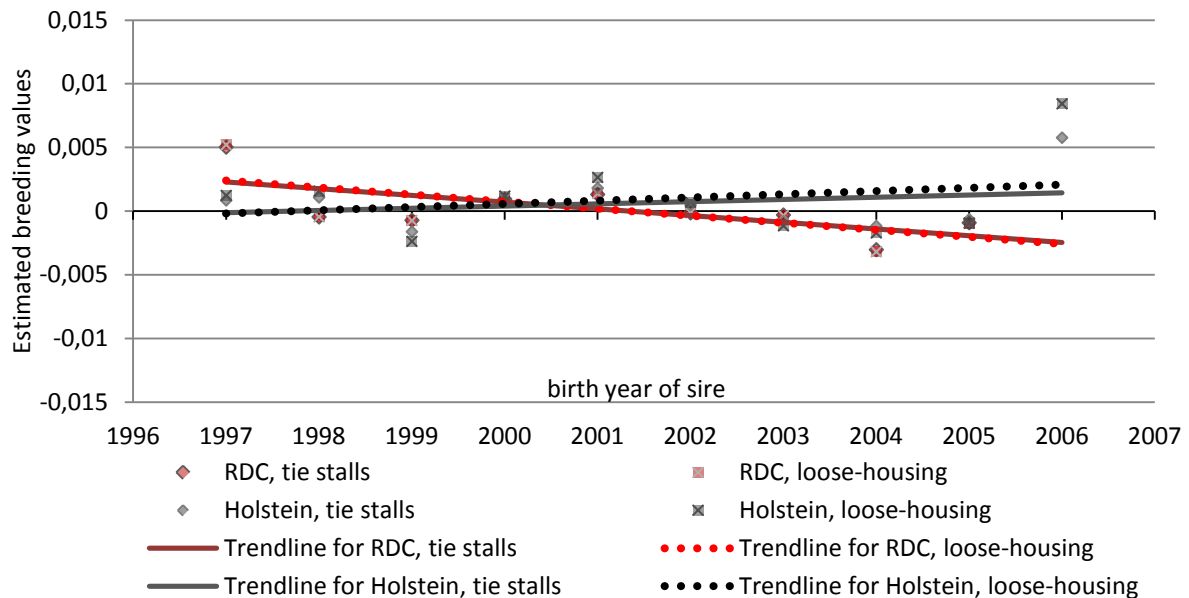


Figure 13. Genetic trend by birth year of sire for hygiene-related claw diseases in lactation three for Swedish Holstein cows (SH) and Swedish Red dairy cows (SR) in different housing systems, where a negative trend indicates a favorable decrease in susceptibility to claw disease.

Discussion

The importance of improving claw health traits

Claw diseases are costly for the producer (Oskarsson, 2008) and painful for the cow (Manske *et al.*, 2002b) and diseases like dermatitis, heel horn erosion and sole hemorrhages are alarmingly common in Swedish dairy cows, see Figures 1, 2 and 4. These three diseases also seem to be more common in loose-housing as seen in Figures 1, 2 and 4 and Tables 4, 6 and 10. Cows suffering from sole hemorrhage will have a decreased milk production as well as a decrease in the number of visits to the feeding area and time spent there (Carlsson *et al.*, 2012). But it is not just sole hemorrhage which affects visits to the feeding table, lameness in general has the same effect (Bach *et al.*, 2007). Visits to and time spent at the feeding table are two important factors for loose-housing systems since being able to eat a sufficient amount of feed is important for the high producing cow. This means that the importance of good claw health will increase during the transition from tie stalls towards loose-housing.

Claw diseases related to hygiene

The influence of lactation number

For the three hygiene-related diseases dermatitis, heel horn erosion and skin proliferation the disease frequencies are increasing with lactation number, seen in Figure 1, 2 and 3. Considering that the background of the diseases is hygiene-related this is logical (Manske *et al.*, 2002b). With increasing lactation number the cow has also been exposed to the general hygiene-level in the herd for a longer time. The increase in disease frequency with lactation number could also imply that once a cow has been infected, full recovery is difficult.

There is no common pattern for the influence of lactation number on the heritability of the three diseases, see Table 4, 5 and 6. An increase in heritability with lactation number, as for skin proliferation (Table 6) could mean that the genetic impact increases. In the case of hygiene-related diseases this implies an increased importance of genes related to protection against infection. However, skin proliferation has a lower heritability than the two other hygiene-related diseases. The increased heritability could therefore be a direct result of the increase in skin proliferation frequency which comes with increasing lactation number. In the study by Johansson *et al.* (2011) the heritability of the different diseases in different lactations was comparable, with exception for skin proliferation which showed an increasing heritability with increasing lactation number as well. For dermatitis in Swedish Holstein the situation is the opposite with decreasing heritability with lactation number and an increasing disease frequency with lactation number. This implies that the environmental effect on the presence of dermatitis is larger in later lactations.

Difference in heritability of claw health traits in different lactations has been noticed in other studies as well (van der Linde *et al.*, 2010 and Johansson *et al.*, 2011). In the study by van der Linde *et al.* (2010) small differences were found, but what is interesting to observe is that for digital dermatitis, interdigital hyperplasia (a form of skin proliferation) and sole hemorrhage the correlation between the disease in lactation one and the consecutive lactations was significantly different from one. This implies that digital dermatitis, interdigital hyperplasia

and sole hemorrhage in lactation one and the same disease in the consecutive lactations are controlled by slightly different genes. The variation in heritability depending on lactation number shows the importance of repeated registrations for several lactations.

Even though the heritability for hygiene-related traits was comparable to those from the individual traits no pattern of the heritability or genetic variance depending on lactation number could be seen when the diseases were grouped. This is despite the clear increase in disease frequency with increasing lactation number. The increase in disease frequency combined with the lack of such a pattern for the heritability and genetic variation imply that the cause for the higher hygiene-related disease frequencies in later lactations is related to environmental factors.

The influence of housing system

Since dermatitis, heel horn erosion and skin proliferation are related to hygiene (Manske *et al.*, 2002b) higher disease frequencies are expected in loose-housing compared to tie stalls, see Figure 1, 2 and 3. Since there is a ban on building new tie stalls in Sweden (The Swedish Board of Agriculture, 2010) the number of cows housed in loose-housing will increase. This makes these results important to consider when aiming to retaining a good animal welfare.

In general, the frequencies of the different hygiene-related diseases are lower in tie stalls compared to loose-housing. This could be due to the environment being easier to keep clean in tie stalls. In previous studies on different forms of loose-housing the effect of flooring has been investigated. These studies show lower disease frequency for slatted floors (Somers *et al.*, 2003, Fjeldaas *et al.*, 2011) which further denotes the importance of a clean environment. Another reason for the differences in disease frequency depending on housing system could be due to a higher risk of infection with cows moving freely, as in loose-housing.

The heritability of the different diseases in different housing systems is varying. For dermatitis and heel horn erosion there is a tendency towards higher heritability in tie stalls compared to loose-housing. Since the disease frequency is lower in tie stalls compared to loose-housing this cannot be explained by a higher disease frequency. Instead these results entail that the hygienic level of the environment have a larger impact on the presence of the disease in loose-housing compared to tie stalls. For skin proliferation there is a tendency towards higher heritability in loose-housing systems. However, the heritability of skin proliferation is low which implies that it is the presence of contagion or not which determines the existence of the disease. Loose-housing entail a higher infection pressure which means that the very small genetic variation which exists probably is due to variation in the immune defence.

Feed-related claw diseases

Influence of lactation number

Based on the study by Manske *et al.* (2002b) sole hemorrhage, sole ulcer, white line separation and/or double sole are considered to be feed-related claw diseases since they are common symptoms of laminitis. Corkscrew claws are also considered a feed-related disease based on genetic correlations to the diseases mentioned above (Johansson *et al.*, 2011). For

sole hemorrhage and sole ulcer there is no clear increase in percentage of the diseases with increasing lactation number, see Figures 4 and 5. Instead the number of sick cows is decreasing from lactation one to lactation two and then increasing again in lactation three. The reason for this might be found in that the transition from heifer to milking cow is larger than the transition between the subsequent lactations. The results concerning disease frequency and lactation number is in contrast to a previous study by Pryce *et al.* (1999) where the prevalence of lameness was increasing with lactation number. However this study was not for any individual claw diseases. The fact that this pattern occurs for both sole ulcers and sole hemorrhage suggests that this pattern might be related to laminitis. It is possible that the cows have a higher resistance to laminitis in lactation two when they are more used to the housing and feed. Another reason could be that cows who suffer from sole ulcers already in the first lactation are culled since, as mentioned by Manske *et al.* (2002b), relapses are common with sole ulcers.

Even though white line separation and double sole also are laminitis-related, these diseases show another pattern with a clear increase in disease prevalence with lactation number, see Figure 6 and Table 14. An explanation for this can be due to the fact that white line separation and double sole occur in phase four-laminitis (Nocek, 1995) meaning that the diseases do not occur until the claw has been severely damaged by laminitis. This implies that for a cow prone to subclinical laminitis, each lactation can cause a little bit more damage to the claw tissue. This is in line with the increase in heritability with lactation number, seen in Table 9. The increase in heritability suggests that the process leading to white line separation or double sole is additive over subsequent lactations.

Corkscrew claws have a different but distinct pattern where there is an increase in disease frequency with lactation number, but only in loose-housing and not in tie stalls. The differences in the pattern of corkscrew claws compared to the other feed-related disease might be explained by other factors except feed affecting the presence of corkscrew claws. An alternative explanation for the correlation between corkscrew claws and the laminitis-related diseases might be that corkscrew claws cause unevenly distributed weight on the claws which might cause laminitis-related claw damage to turn into for example a sole ulcer.

Influence of housing system

Since sole hemorrhage, sole ulcers, white line separation and double sole are common consequences of laminitis, they are dependent both on feed and environment. An unbalanced rumen, caused by the feeding, is believed to be the cause to laminitis which causes degradation in the claw tissue (Manske *et al.*, 2002b). Livesey *et al.* (1998) looked at the effect of concentrate feeding on sole hemorrhages until weeks 12 in milk, and found that high concentrate feeding gives a higher prevalence of sole hemorrhages which further strengthens the idea of sole hemorrhage being related to laminitis. When the claw tissue becomes degraded due to laminitis it is fairly logical if it also becomes more sensitive to pressure or strain.

For sole hemorrhage, white line separation and double sole in both breeds and corkscrew claws in Swedish Red dairy cows tie stalls seem to provide lower disease frequencies

compared to loose-housing. This could be due to different factors. Sole hemorrhage can occur as a response to stress from a change in housing or flooring (Bergsten & Frank, 1996) which implies that higher strain on the claws can cause sole hemorrhage. Since, as mentioned by Manske *et al.* (2002b), loose-housing is more strenuous for the claws this might cause the higher frequency of cows with sole hemorrhage in loose-housing. One reason for higher strain on the claws causing sole hemorrhage could be the fact that sole hemorrhage is a laminitis-related disease. Laminitis causes damage on the claw tissue (Nocek, 1995 and Manske *et al.*, 2002b) and might thereby make the claws more sensitive to strain. An example of the varied causes to sole hemorrhage is seen in the study by Livesey *et al.* (1998) where the presence of sole hemorrhage is affected both by flooring and by feed. In the case of white line separation the extra strain put on the claws in loose-housing could cause the adherence between the claw capsule and the claw bone to rupture. Loose-housing may not only cause higher strain on the claws, it might also cause higher frequency of laminitis since it makes individual feeding more difficult.

Concerning sole ulcers, Sogstad *et al.* (2005) state that the influence of housing system on the frequency of sole ulcers is low. In this study there are small differences between disease frequencies in different housing systems, but some of the differences are still significant. Sole ulcers also lack clear tendencies concerning the heritability in different housing systems. For sole hemorrhage however, there is a tendency towards a higher heritability in tie stalls. This might be due to the fact that the environmental impact could be lower in tie stalls since the claws are subject to a less strenuous environment. For white line separation and double sole the situation is the opposite with higher heritability in loose-housing. This could however be a direct cause of a higher frequency of the diseases in loose-housing, which since the trait overall have a low frequency, could have quite a large impact.

When analysing the heritability of corkscrew claws in different housing systems there are quite clear breed differences. These breed differences might however be due to the lower disease frequency for Holstein cows. There is also a difference between the effects of lactation number depending on housing system. This is not seen for the heritability where there is a decreasing heritability with lactation number for both housing systems. This suggests that the differences between the frequencies of corkscrew claws in different housing systems are mainly due to environmental factors.

Combination of traits

Whether or not different traits should be combined is debated. Combining traits can increase the frequency and a higher frequency makes it easier to estimate variance components since it provides a larger amount of data to estimate the variances from. However, if the estimation of variance components is made in order to estimate breeding values for selection of a certain trait, combining traits can lead to that the animals chosen for selection in the end are not the best for the individual traits. Based on this it is important to always keep in mind on which trait selection is made. If breeding values are estimated for trait A combined with trait B (trait A+B), genetic improvement will occur for trait A+B, not for trait A and trait B individually.

In this study the combination of traits into feed-related and hygiene-related diseases led to disease frequencies which were fairly coherent with the results for the individual diseases. The genetic parameters were also in cohesion with the results for the individual traits when looking at the level of the heritability. However, patterns that were seen for the heritability of some of the individual traits were not seen when they were combined. These differences in heritability show the importance of always keeping in mind on which trait selection is made.

Genetic correlations for the same claw disease in different housing systems

For the individual traits there are four genetic correlations for the same disease in different housing systems which are significantly different from one. These are for skin proliferation in Holstein cows in lactation one (Table 9), sole hemorrhage in Swedish Red in lactation two (Table 11), sole ulcers in Holstein cows in lactation one (Table 13) and finally corkscrew claws in Holstein cows in lactation three (Table 17). For the grouped diseases, there are genetic correlations significantly different from one for feed-related diseases in Holstein in lactation one and two and in Swedish Red in lactation two (Table 20). For hygiene-related diseases there is only one genetic correlation significantly different from one, for Holstein cows in lactation one (Table 20). The fact that there are more genetic correlations significantly different from one for feed-related disease is logical since three of the four individual diseases with genetic correlations significantly different from one are considered feed-related diseases. However, these results are in contrast with what was hypothesised.

The hypothesis was that it was more likely to find genetic correlations significantly different from one for diseases related to hygiene since these were thought to be more affected by housing system. However, the reality proved to be the opposite with more genetic correlations significantly different from one for feed-related diseases than for hygiene-related diseases. The fact that there is a difference between the genetic trend for feed-related diseases depending on housing systems further strengthens the idea of higher GxE interactions for feed related diseases than for hygiene-related diseases. The most probable reason for this is that the feed-related diseases are related to laminitis, a feed-induced disease resulting in degradation of the claw tissue (Nocek, 1995 and Manske *et al.*, 2002b). Genes associated to feed probably have the same impact on the feed-related diseases in both housing systems. However, the fact that laminitis causes degraded claw tissue makes it reasonable to believe that genes associated to soundness of the claws probably have a larger impact in loose-housing which, according to Manske *et al.* (2002b), is a more strenuous environment for the claws. Considering this, the lower number of cows with feed-related disease in tie stalls, see Table 19, might be due to an environment which holds the prevalence of the diseases down.

The only hygiene-related disease with a genetic correlation significantly different from one was skin proliferation. The correlation is lower than expected and might be due to the low frequencies of the disease causing the presence of the disease in the different housing systems causing difficulties in estimating the correlation. It is also possible that the cows' genetic potential to cope with infection is more important in loose-housing.

For the combination of diseases into feed-related or hygiene-related claw diseases in lactation one and three, ranking of sires in the two housing systems was compared. When comparing

the rankings in the two systems with a correlation of one, no re-ranking occurred. For correlations which differed from one, re-ranking was observed even though the differences were not significant. This indicates that some bulls might rank higher in one housing system than in another even though the genetic correlation is not significantly different from one.

Suggestions for future studies

Several of the claw diseases in this study are consequences of laminitis. Therefore it would be interesting to look at the cause of laminitis. One way to do this could be to study if there is a variation in how well a cow can cope with lower levels of kilogram dry matter of forage, i.e. higher amounts of concentrate. This could be done by combining a claw dataset with feed rations. If environment is defined as feed rations and if there is a variation between the responses to the different environments it could be possible to look at GxE interactions between laminitis-related diseases in environments with different feed rations.

Based on the genetic correlations significantly different from one for feed-related diseases it can be hypothesized that the strain on the cows' claws plays a more important role in loose-housing compared to tie stalls. With this background in mind it might be interesting to investigate the influence of cow weight on claw health. It would also be interesting to look at how the distribution of weight on the claws influences claw health.

It would also be interesting to investigate the possibility of separating interdigital and digital dermatitis into two traits. Interdigital dermatitis is said to be a hygiene-related eczema which can be both husky and discharging while digital dermatitis has a much more characteristic look with strawberry-like ulcers (Manske *et al.*, 2002b) and is caused by a particular strain of bacteria (SVA, 2012). There are thereby fairly large differences between digital and interdigital dermatitis. Since digital dermatitis is very difficult to become free from once the infection has entered a loose-housed herd (Olsson, 2010) the coming transition to loose-housing systems might motivate keeping digital dermatitis as an individual disease.

Conclusion

The genetic correlations between the same traits in different housing systems indicate that there are some differences in which genes that control the presence of claw disease in different housing systems but most correlations were not significantly different from one. However, differences non-significantly different from one still caused re-ranking among bulls. The differences in which genes that control claw disease is larger in claw diseases related to feed compared to claw diseases related to hygiene. The probable reason for this is that the feed-related diseases are multifactorial and dependent on feed, housing and flooring to a different extent depending on housing system.

Inclusion of claw health in the selection index can be recommended due to the high frequencies of claw diseases in modern dairy production, along with economical and ethical aspects. If the diseases are combined in a claw health index with appropriate weights as opposed to combining the diseases before estimation of variance components and breeding value prediction, larger progress may be expected. Due to the re-ranking among bulls it may be possible to run breeding value predictions in bivariate analyses and select bulls more suitable for each housing system. A transition towards loose-housing, which for example is occurring in Sweden, further motivates selection of bulls suited for loose-housing. Running the analyses bivariately with the other housing system as correlated information would utilize the claw health in the other housing system as correlated information.

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Appendix 1. Summary of genetic studies on claw health

Table 1. Summary of genetic studies on claw health including information concerning model, claw diseases and heritability

Reference	Model	Claw disease	Heritability (Standard error)	
			Holstein	Sw. Red
van der Waaij <i>et al.</i> , 2005	Claw disease = overall mean + fixed effect of parity + fixed effect of lactation stage + fixed effect of breed + effect of herd combined with scoring day and claw trimmer + random sire effect + random residual effect	Interdigital dermatitis and heel horn erosion	0.05 (0.01)	
		Sole hemorrhage	0.08 (0.02)	
		Digital dermatitis	0.10 (0.02)	
		White line disease	0.02 (0.01)	
		Interdigital hyperplasia	0.10 (0.02)	
		Sole ulcer	0.01 (0.01)	
		Chronic laminitis	0.01 (0.01)	
Laursen <i>et al.</i> , 2009	Claw health = overall mean + fixed effect of herd and year + fixed effect of season + fixed effect of calving age + fixed effect of calving-year-period + random sire effect + random residual effect	Claw health (defined as absence of heel horn erosion, interdigital phlegmon, interdigital hyperplasia, laminitis and sole ulcer)	0.01 (0.0005)	
Meeuwes, 2009	Claw disease = fixed effect of calving age at first calving + fixed effect of lactation stage + fixed effect of month of first calving + random effect of claw trimmer + random effect of herd and trimming year + random effect of sire + random residual effect	Digital dermatitis	0.04	
		Heel horn erosion	0.03	
		Sole hemorrhage	0.02	
		Sole ulcer	0.04	
		Digital dermatitis and Heel horn erosion combined	0.04	
		Sole hemorrhage and Sole ulcer combined	0.04	
Van der Linde <i>et al.</i> , 2010	Claw disease = overall mean + fixed effect of parity + fixed effect of age at calving + fixed effect of lactation stage at claw trimming + fixed effect of herd and date of visit + fixed effect of claw trimmer and half year of trimming + random effect of permanent environmental effect of animal + random sire effect + random residual effect	Sole hemorrhage	0.06	
		Digital dermatitis	0.09	
		Interdigital dermatitis	0.11	
		Wall ulcer	0.01	
		Sole ulcer	0.12	
		Interdigital hyperplasia	0.13	
		White line disease	0.03	
		Combined claw health trait	0.07	

Johansson <i>et al.</i> , 2011	Claw disease = fixed effect of herd and period + fixed effect of calving age and country + fixed effect of year, month and country of calving + fixed effect of lactation stage + random effect of herd, year and season + random effect of permanent environment + random animal effect + random residual effect	Dermatitis (digital and interdigital)	0.04	0.04
		Heel horn erosion	0.04	0.07
		Sole hemorrhage	0.04	0.05
		Sole ulcer	0.04	0.02
		Corkscrew claws	0.02	0.03
		White line separation and double sole	0.01	0.01
		Skin proliferation (interdigital hyperplasia and warts)	0.02	0.03

Appendix 2. Variables in the data set

Table 1. Integer variables

ID	NAV ID of the cow with claw trimming	
ca1	Calving age in months (+1000) lactation no 1	
ca2	Calving age in months (+1000) lactation no 2	
ca3	Calving age in months (+1000) lactation no 3	
hy1	Herd-year-season lactation no 1	
hy2	Herd-year-season lactation no 2	
hy3	Herd-year-season lactation no 3	
cmy1	Country-month-year of calving lactation no 1	
cmy2	Country-month-year of calving lactation no 2	
cmy3	Country-month-year of calving lactation no 3	
Y	A herd classification stretching over five years.	
wm1	Week in milk lactation no 1	
wm2	Week in milk lactation no 2	
wm3	Week in milk lactation no 3	
IDk	Claw trimmer ID	
L	Lactation number	
be1	Herd lactation no 1	
be2	Herd lactation no 2	
be3	Herd lactation no 3	
sID	NAV sire ID	
dID	NAV dam ID	
st1	Housing lactation no 1	
st2	Housing lactation no 2	
st3	Housing lactation no 3	
rkr1	Breed code SR lactation no 1	The breed code is on herd level. For example, if there is 100 cows in a herd and 30 of these are SR and 70 SHstein, then rkr=30 and SH=70
rkr2	Breed code SR lactation no 2	
rkr3	Breed code SR lactation no 3	
rkh1	Breed code SH lactation no 1	
rkh2	Breed code SH lactation no 2	
rkh3	Breed code SH lactation no 3	

Table2. Real variables

De1	Dermatitis lact no 1	<p>The claw disease records are adjusted to variance due to the fact that they are part of a breeding evaluation done in three different countries. These data will be treated as following:</p> <p>if claw disease < 0,5 then claw disease = 0</p> <p>If claw disease = 0,5-1,5 then claw disease = 1</p> <p>If claw disease > 1,5 then claw disease = 2</p>
HH1	Heel horn erosion lact no 1	
SH1	Sole hemorrhage lact no 1	
SU1	Sole ulcer lact no 1	
CS1	Corkscrew claws lact no 1	
WL1	White line separation and double sole lact no 1	
SP1	Skin proliferation: Interdigital hyperplasia and warts lact no 1	
De2	Dermatitis lact no 2	
HH2	Heel horn erosion lact no 2	
SH2	Sole hemorrhage lact no 2	
SU2	Sole ulcer lact no 2	
CS2	Corkscrew claws lact no 2	
WL2	White line separation and double sole lact no 2	
SP2	Skin proliferation: Interdigital hyperplasia and warts lact no 2	
De3	Dermatitis lact no 3	
HH3	Heel horn erosion lact no 3	
SH3	Sole hemorrhage lact no 3	
SU3	Sole ulcer lact no 3	
CS3	Corkscrew claws lact no 3	
WL3	White line separation and double sole lact no 3	
SP3	Skin proliferation: Interdigital hyperplasia and warts lact no 3	

Appendix 3. Frequency of claw diseases in Swedish dairy cows of different breeds across housing system

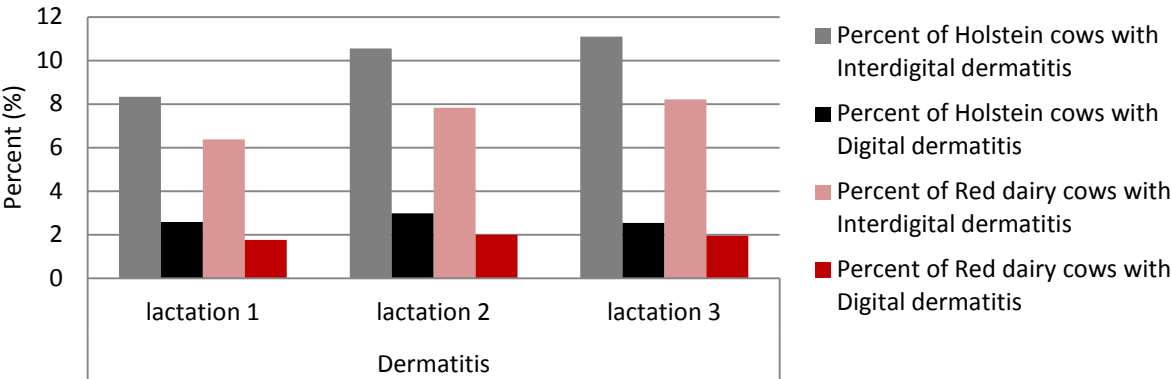


Figure 1. Cows of different breeds with Dermatitis

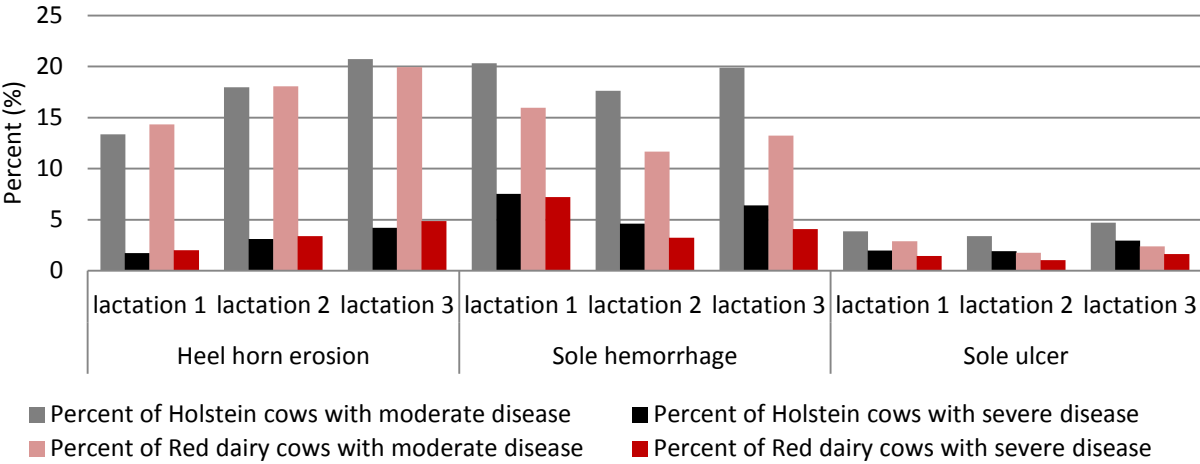


Figure 2. Percent of cows of different breeds with Heel horn erosion, Sole hemorrhage or Sole ulcer

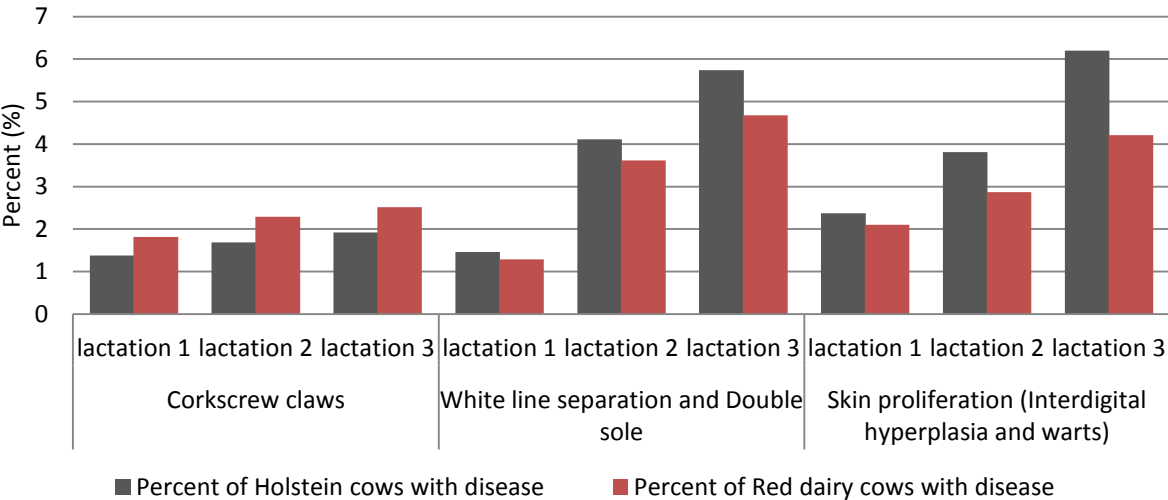


Figure 3. Percent of cows of different breeds with Corkscrew claws, White line separation, Double sole or Skin proliferation (Interdigital hyperplasia and warts)