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Analysis of survival in laying hens using repeatability models

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Abstract

The commercial egg production is a huge and growing industry, which faces some important animal welfare issues. Feather pecking and cannibalistic behaviour are a multi-factorial problem that have a huge negative impact on the farmers' economy, as many layers die before the laying period is over. The solution to this problem has so far been beak trimming, which is also publicly regarded as a welfare issue. In addition to better management, breeding is starting to be looked upon as a more permanent solution, as the destructive behaviour has a heritable component. The challenge today is to find a breeding model that accurately predicts breeding values on the parental animals from survival data of the progeny. In traditional models, usually only the direct effect of the animal itself has been accounted for, neglecting the genetically inheritable social component that exists between grouped animals. In this thesis, survival records (0/1) from 6916 pure bred layers were analyzed using 6 different models. Model 1: Sire direct effects model, 2: Sire direct-random cage effects model, 3: Sire direct-indirect-random cage effects model, 4: Sire-Dam direct-indirect-random cage effects model, 5: Repeatability direct-indirect-random cage effects model, 6: Repeatability direct-indirect-random cage effects model with time dependent social effects. Genetic parameters were estimated, where the heritability ranged between 0.0121 and 0.1169, and the correlation between direct and indirect genetic effects ranged from -0.3704 to -0.4898. Cross validation of the models showed that for analyzing this type of survival data, the Sire-Dam direct-indirect-random cage effects model gave the most accurate breeding values compared to the other models. A Sire-Dam model can therefore be a good tool when breeding towards low-pecking strains of layers, where damaging behaviour is eliminated and beak trimming is unnecessary.

Sammendrag

Den kommersielle eggproduksjonen er en stor og voksende industri som har støtt på betydelige utfordringer innen dyrevelferd. Fjørpikking og kannibalistisk atferd er et multifaktorisk problem som har en stor negativ innvirkning på produsentens økonomi, da mange verpehøns dør lenge før verpeperioden er over. Løsningen på fjørpikking og kannibalisme har så langt vært nebbtrimming, noe som av opinionen også er ansett som dårlig dyrevelferd. I tillegg til generelt bedre daglig drift og administrering, begynner avl å bli sett på som en mer permanent løsning, da destruktiv pikkeatferd har en arvelig komponent. Utfordringen for dagens husdyravl er å finne en avlsmodell som med stor nøyaktighet kan predikere avlsverdier for foreldredyr basert på avkoms overlevelsesdata. I tradisjonelle modeller er det vanligvis bare den direkte effekten av dyret selv som blir inkludert, uten hensyn til at det også finnes en sosial komponent som påvirker atferd. I denne oppgaven er overlevelsesdata (0/1) fra 6916 renrasede verpehøns analysert med 6 forskjellige modeller. Modell 1: "Sire direct effects model", 2: "Sire direct-random cage effects model", 3: "Sire direct-indirect-random cage effects model", 4: "Sire-Dam direct-indirect-random cage effects model", 5: "Repeatability direct-indirect-random cage effects model", 6: "Repeatability direct-indirect-random cage effects model" med tidsavhengige sosialeffekter. Genetiske parametere ble estimert, hvor arvbarheten varierte mellom 0.0121 og 0.1169, og korrelasjonen mellom direkte og indirekte genetiske effekter varierte mellom -0.3704 til -0.4898. Kryssvalidering av modellene viste at Sire-Dam modellen gav de mest korrekte avlsverdiene ved analyse av denne typen overlevelsesdata, sammenlignet med de andre modellene. En slik modell kan derfor være et nyttig verktøy i arbeidet med å avle fram verpehønslinjer med lav pikkefrekvens, der skadelig atferd er eliminert og nebbtrimming er unødvendig.

Key words: layer, survival, cannibalism, feather pecking, sire model, repeatability

Preface

Through my whole life I have loved all kinds of animals, and felt very protective of them. I have many opinions about the commercial animal industries, and how intensification of the systems leads to suffering and bad animal welfare for the different species involved. I believe that as long as humans continue to exploit animals for the meat or other products, the animals should have the right to live a worthy life before death. For me, a worthy life means that the individual animal gets the chance to have proper life quality, to be itself and follow its motivations that are preserved deep in a species instincts and specific behaviour patterns. I think that the industry today is way too concerned about profit and effective production to care about the animals as individuals, and this makes me very frustrated. The pork industry and especially the poultry and egg production systems are in my opinion some of the worst ones, as very few measures are taken to make the environment closer to the animal's needs, as well as preferences. That is why I got intrigued when I looked at the different options for thesis topics, and saw a headline regarding laying hens and cannibalism. For me, that is an issue which feels meaningful to work with, to have the possibility to make a small difference. Even if it does not lead to anything spectacular, research like this can in the future collectively lead to a change for the better, for the birds whose welfare so few seem to worry about.

This thesis marks the end of my five years as a student of animal sciences, which has been exciting and inspirational the whole way, and I am looking forward to find "the perfect job" where all my new knowledge can come to good use! I want to thank my supervisor Esther Ellen for the superb guidance and help she gave along the way with my thesis. Our weekly meetings were always pleasant and inspirational. Further, I want to thank Theo Meuwissen for being co-supervisor, and Tessa Brinker for helping me when the computer programs got too complicated. In the end I also want to express my gratitude to the study advisors at IHA, NMBU; Stine Telneset and Marit Ensby for persuading me to broaden my horizons and study at Wageningen UR. I do not regret that decision!

WUR, Wageningen, June 2014

Content

Introduction

The commercial egg industry

The global commercial egg production today is a huge and growing industry which produces over 65 million tons of eggs every year (Web: FAO stat 2011). The industry is using only a few commercial layer breeds, where the breeding is controlled by only a couple of major breeding companies (Web: Breeding companies). As the demand for eggs in the world is increasing in pace with the population growth, the egg production systems have underwent a great intensification. Thousands of layers are put together in laying houses to produce more eggs, faster and to a lower cost. This intensification process has lead to several welfare problems as the breeding is somewhat unidirectional, and the production environment is to a low degree adjusted to the birds' motivations and behavioral needs. One of the biggest problems is the abnormal and destructive pecking behavior that develops in all types of commercial housing systems (Sherwin et al. 2010). Extreme pecking and cannibalism impact the farmer's economy negatively due to loss of eggs, veterinary expenses, and increased workload when layers die or get injured before the laying period is over. Mortality due to different types of cannibalism have been reported to range from 4-30% (Hocking et al. 2004; Usman & Diarra 2008), and being the main cause of all deaths in a flock (Fossum et al. 2009; Weitzenbürger et al. 2005), with the largest problems in loose systems where the pecking layers have access to all other layers, resulting in extreme mortality rates (Fossum et al. 2009; Hughes & Gentle 1995). Since the ban on battery cages in Europe in 2012 (Web: Council directive) the trend has moved towards using non-cage systems, like aviaries, floor- or free range systems rather than caged systems (Lukanov & Alexieva 2013), which means that the problem of cannibalism and pecking can affect even more farmers, and the mortality will increase within the flocks.

The main solution to the destructive pecking has so far been beak trimming. Beak trimming is a procedure that takes place before the chicken is 10 days of age, where a part of the beak is removed to make it blunter, so the pecking cannot cause serious injury on another hen (Gentle 1986). In Europe, all mutilations of laying hens are prohibited, except beak trimming performed in early age as a prevention of feather pecking and cannibalism (Web: Council directive). However, several countries in Europe have already outlawed the beak trimming procedure (Sweden, Finland, Switzerland, Norway) as it can cause serious pain for the layers both short and long term, and is itself looked upon as a welfare issue (Hughes & Gentle 1995). Some other European countries have as well made plans to implement a ban in the

nearest future, UK in 2016 and the Netherlands in 2018 (Web: UK, The Netherlands), as beak trimming may be unnecessary with proper housing systems and management (Jendral & Robinson 2004). Even though the trimming procedure can be a quick solution to the problem, it is not a long term solution, and the problem will remain the same year after year, if not take a turn for the worse. There have been some studies on the heritability of pecking and cannibalism (Kjaer & Sørensen 1997; Rodenburg et al. 2003; Savory 1995) which information can be used within breeding to give rise to strains of layers that are less destructive.

Breeding values on survival

Pecking, and being pecked are in fact inheritable traits (Kjaer & Sørensen 1997; Rodenburg et al. 2003; Rodenburg et al. 2004; Su et al. 2005) which makes it possible to select towards behavioral phenotypes that are less harmful. Calmer hens with a lower motivation for pecking on other individuals will increase the survival in the flock, both by lowering the incidence of pure cannibalism and injuries, but also by allowing the plumage of the hens to remain in good quality the whole laying period. Selecting for lower mortality due to pecking/cannibalism in layers requires a good model for breeding value estimations on survival. The traditional methods only account for the direct effect, the survival of the hen itself, but this will give an inaccurate picture of the reality. In a cage or in a flock, the hens will interact socially, and influence each other's behavior. A cannibalistic hen can trigger other hens as well to perform cannibalism (Cloutier et al. 2002), and feather pecking can be taught from one layer to another, spreading the behavior in the flock (Zeltner et al. 2000). Therefore, a social effect has to be included in the model for survival, as it can stand for up to 2/3 of the total genetic variation (Bijma et al. 2007).

In a previous study, the direct and social (associative) genetic parameters were estimated by using a linear animal model, where the results were compared to a model that only included the direct effect (Ellen et al. 2008). Another study combined a survival analysis with a linear animal model including the associative effects (Ellen et al. 2010). Both methods gave more accurate results than only using the direct effect in the model, but it is still difficult to include survival time in a sufficient way. Censored records are a problem, where layers that do not die from cannibalism/pecking, but are being culled, lacks records on actual survival time and do not fit in a regular animal model. There is also a problem with the social effects, because they are not constant between the individuals during the whole laying period. The social effects are closely connected to the survival time, since a dead layer can no longer peck or influence the remaining hens in a cage or in a barn. Repeatability models have normally been used for

several production traits, e.g. milk yield per lactation (Carvalheira et al. 2002) or for type traits in dairy cattle (Gengler et al. 1997), and is believed to be a potentially better method for analyzing survival data in laying hens as well. Therefore, in addition to simpler sire models, two repeatability models will in this thesis be used to estimate the genetic parameters; one that accounts for the survival time, and one that accounts for survival time and time dependent social effects collectively.

Background theory

Destructive behaviour

In most of the modern animal production systems there are different challenges regarding animal welfare. If a husbandry system does not provide the animals with the proper opportunities to fulfill and express their natural motivations, frustration starts to build up. The animal will look for a way to escape or cope with the stressful situation, which often ends up with redirecting the motivation towards something else, or develop ambivalent or stereotypic behaviour patterns (Mason & Rushen 2006). The most controversial system within the egg industry is the traditional battery-cage system, where the general public opinion is that this way of caging has housing conditions that are inadequate for good life quality. The pressure to improve the animal welfare for layers has continuously been increasing (Web: Public pressure), with people rather buying eggs from loose housing systems, even though these systems may have the same, if not more problems with destructive behaviour than the cage systems (Fossum et al. 2009; Hughes & Gentle 1995).

The exact reason why chickens tend to adopt destructive behaviour like feather pecking and cannibalism is still not known, but it is speculated that it might be some sort of coping mechanism. The different types of pecking behaviour seen among chickens can either be harming/severe, or non-harming/gentle. Most pecking is non-harming and related to feeding, exploration and gentle preening on own and other birds' plumage (Leonard et al. 1995; Riedstra & Groothuis 2002; Webster & Hurnik 1990), while some pecking can be more aggressive and related to establishment of hierarchy or other social interactions (Guhl 1958; Wood-Gush 1955). However, this is normal chicken behaviour, and should not be confused with severe feather pecking behaviour.

Feather pecking

The term feather pecking is used in a situation where a bird grabs other birds' feathers, and damage or pulls the feather out. The behaviour is a multi-factorial problem that leads to extensive feather loss, damage and injuries that can cause illness and death (Blokhuis & Wiepkema 1998; Savory 1995). The feather pecking is most often directed toward the rump feathers, but can also progress to the back, wings, tail, feathers around the cloacae and the rest of the body (Bright 2009). Most often feather pecking develops at an early age, starting even before transfer from the rearing house to the laying house (Hocking et al. 2004; Van Hierden et al. 2002). The pecking is actually not regarded as aggressiveness, as aggressive pecking is more directed against the head and comb and delivered with considerate force, as if to start a fight (Savory 1995). At the same time, severe feather pecking and gentle feather pecking cannot be considered as the same trait, as gentle feather pecking in young age cannot be used as a predictor of severe feather pecking in adult laying hens (Newberry et al. 2007; Rodenburg et al. 2004). It is believed that the behaviour is connected to ground pecking (Blokhuis 1986), dustbading (Vestergaard et al. 1993) or foraging (Huber-Eicher & Wechsler 1997), except that the pecking is redirected towards other individuals instead of the environment. This can happen especially when the living environment is plain and unstimulating, where the ground does not encourage exploration (Hughes & Duncan 1972; Johnsen et al. 1998) and the feed is low in fiber and/or too grinded so it can be consumed within a short amount of time (Hetland et al. 2004).

Other factors that may trigger feather pecking are nutritional deficiencies (Ambrosen & Petersen 1997; Van Krimpen et al. 2005) where the hens are looking for other food sources, overcrowding that leads to stress and competition for resources (Hughes & Duncan 1972; Pötzsch et al. 2001; Savory 1995), bright light in the laying house (Kjaer & Vestergaard 1999) and general bad housing conditions and management. It is even believed that the preen-gland oil composition (smell and taste) may have something to do with attraction to pecking (Sandilands et al. 2004). Motivation for feather pecking is also different between gender, where it is more common in females and rare in males (Hughes 1973), and between breeds (Hocking et al. 2004; Kjaer & Sørensen 2002). Differences between strains are also documented, where the lines have unequal motivation for pecking and preening (Hughes & Duncan 1972; Kjaer et al. 2001; Rodenburg et al. 2004; Van Hierden et al. 2002). Farmers can experience no problems with one batch, and full outbreak in another. The severe feather

pecking can be damaging enough by itself, but in many cases it also develops into cannibalistic behaviour among the layers.

Cannibalism

Cannibalism is by definition when an individual consumes body parts of the same species as itself, and is a wide spread natural adaptation mechanism for several species against competition, starvation and as defense (Fox 1975). While this is the law of nature in many wild populations, cannibalism is not by any means wanted in domesticated livestock populations. Cannibalism in chickens is recognized when a bird pecks on other birds' feathers and skin until wounds appear, and proceeds with consummation of blood and tissue. The wound is made bigger by continued pecking until it inflicts death upon the victim, followed by pecking on the dead body (Blokhuis & Wiepkema 1998). The pecking is mostly directed towards the rump, like in feather pecking, but also towards the vent (vent pecking), where the sight of skin and the intestinal entrance seems to be tempting for the bird (Savory 1995). Weitzenbürger et al. 2005 showed that over 38% of the mortality in a flock can be because of vent cannibalism. The development of cannibalism can proceed directly from feather pecking, where the sight of damaged feathers (McAdie & Keeling 2000) or blood becomes a stimulus, or it can be triggered by other factors without having any connection to feather pecking in advance. These factors are mostly the same as what triggers feather pecking, like nutritional factors (Ambrosen & Petersen 1997; Hughes & Duncan 1972), high light intensity (Kjaer & Vestergaard 1999), and unstimulating (Johnsen et al. 1998; Savory 1995), stressful and crowded environment (Allen & Perry 1975; Hughes & Duncan 1972). Breed and strain also have an impact, where some are more likely to be cannibalistic (Hocking et al. 2004; Hughes & Duncan 1972) or victimized than others. Harmful pecking and cannibalism are unfortunately not only genetically conditional, but have a quite big social component where cannibalism or feather pecking can spread in a flock by social learning (Cloutier et al. 2002). A few layers may start pecking, and soon after other observers will follow the same pattern and create an "epidemic" of death.

Other harmful pecking behaviours

Besides feather pecking and cannibalism, other abnormal pecking patterns are sometimes seen in layers. Pecking directed towards the toes of other hens increases mortality, and may develop into cannibalism (Craig & Lee 1990; Krause et al. 2011; Leonard et al. 1995) where the skin and the appearing wounds become a stimulus. Other behavioural problems are feather plucking from own body and feather eating. The feather plucking is especially seen among

parrots in captivity (Lumeij & Hommers 2008), but shows up occasionally in poultry as well, and may be a response to different environmental stress. Feather eating is when the bird rips of feathers from self or conspesifics and swallows them (McKeegan & Savory 1999) and is often seen in relation with feather pecking where the bird eats the feathers of its victim. Reasons for feather eating can be due to some kind of nutritional deficiency where the bird uses the feather as a substitute, as with cannibalistic behaviour.

Destructive pecking and cannibalism are huge problems in the egg industry as it impacts the economy negatively due to loss of eggs, increased feed requirements (Tauson & Svensson 1980), higher electrical expenses for heating (bad plumage quality), and potential veterinary/medication bills. The behaviour does not only develop in laying hens, but is also seen among ducks, broilers, pheasants, geese, turkeys and other avian production species (Dalton et al. 2013; Gustafson et al. 2007; Hoffmeier 1969; Hughes & Gentle 1995). Due to the extent of the problem, and the economical losses the farmers experience, the industry has developed different methods to prevent the unwanted behaviour.

Prevention of destructive pecking behaviour

Appropriate housing systems, good management and experienced farmers are crucial factors when it comes to preventing or stopping outbreaks of pecking and cannibalism. Throughout the years, many methods have been tried out to decrease the mortality, some more successful than others.

Beak trimming

The most dangerous weapon of a bird is the beak, and different approaches has been tried to make it harmless. Beak trimming is a procedure where the sharp tip of the beak is removed, and has so far been the most successful way to stop injurious pecking. With a blunter beak it is harder for the hens to make wounds by pecking, and the decreased sensibility does not give the same tactile satisfaction/stimulation as an intact beak (Hughes & Michie 1982; Hughes & Gentle 1995). Severity of the beak trimming may differ, but the normal range is removal of 1/4 to 1/2 of the beak, with less taken of the lower beak than the upper part (Gentle 1986). The term beak trimming should not be confused with the more drastic method full *debeaking* where almost the whole beak is removed (Hargreaves & Champion 1965), which is not a procedure that is favored or practiced in great numbers.

Different methods for beak trimming have been in use throughout time, with better methods continuously developing. One of the first methods was cutting the beak manually with a knife or blade, but it was not an efficient procedure, and soon machines were built to do it faster and more accurately. These machines were equipped with a cold or hot blade for cutting, and a metal plate with a hole in it for the beak so it could be cut approximately like a guillotine (Glatz & Hinch 2008; Web: Poultryhub). Newer methods that are preferred today are the use of infrared rays that do not directly cut through the beak, but destroys the underlying tissues and blood supply. After short amount of time the tip of the beak will dissolve and fall off. This is regarded as a better method than mechanically removal of the tip, since the procedure leaves no open wounds, is more accurate, and the chicken will have some time to adjust to a shorter beak before the tip disappears completely (Glatz & Hinch 2008; Web: Poultryhub). Another method that does approximately the same is a machine using an electronic current (Bio-Beaker), which burns a hole on the top of the beak and also destroys the tissue inside. This method is suspected to be a worse method for chicken beak trimming, as it is hard to execute properly and seems to produce more extensive damage and acute pain, even though the method has shown better results with turkey (Gentle et al. 1995; Glatz 2000). Regardless of method for beak trimming, the procedure has for a long time been in the public spotlight as a welfare issue as it has strong indications of causing both acute and long term pain for the chickens (Duncan et al. 1989; Gentle et al. 1997). The injured nerve endings in the beak can form neuromas that have been connected to the phenomenon of phantom pain (Breward & Gentle 1985; Gentle 1986). This can be avoided to a certain degree by trimming at an early age (<10 days), but then the chance of regrowth and having to beak trim twice will increase (Glatz 2000; Hughes & Gentle 1995).

Beak trimmed hens have also showed lower feed intake (Gentle 1986; Hartini et al. 2002), more inactivity and a decrease in general pecking behaviour (Gentle et al. 1997), something that also can be interpreted as signs of pain. Whether the pain caused by the procedure is preferred over the suffering from feather pecking and cannibalism is still being discussed, and is a complicated issue to solve ethically. Other methods related directly to the beak are the use of different bumper bits or clips that hinder the beak to close completely, and thereby prevent pulling and biting (Glatz 2000; Web: Nationalband). They have mostly been rejected as a solution commercially as it is difficult to adjust to the individual bird and its age, and requires a considerable amount of work when having thousands of layers.

Light manipulation

The ancestor of the modern chicken is the Red Jungle fowl which is still found living in some parts of the Asian forest. The main habitat of the bird is the forest floor where it hides in dim light under the trees and bushes (Collias & Collias 1967; Subhani et al. 2010; Web: Gallus Gallus). The modern chicken has kept this instinct and the preference for staying and nesting at places with a lower light intensity as protection for predators. As high light intensity seems to increase pecking (Allen & Perry 1975; Kjaer & Vestergaard 1999), lowering the light intensity in the laying house has proven to decrease the level of aggression among the birds (Hughes & Duncan 1972) as it calms them, and also makes it harder to see triggering stimuli as blood and wounds on other hens.

Other light manipulation has also been tried, like using red light bulbs to further mask the color of blood (Savory 1995; Schumaier et al. 1968), and different lighting programs with time scheduled day and night hours, with dimming intensity to mimic dusk and dawn so the chickens have time to settle for night or morning (Web: Agrilight). Some methods have not been successful, like lowering the light intensity to a very low level. This will affect the egg production negatively (Renema et al. 2001), and in the worst cases, the layers develop eye abnormalities (Jenkins et al. 1979) and strange behaviour (Collins et al. 2011). Other unsuccessful methods regarding vision has been the use of rosy contact lenses for not seeing blood, and different types of blinders to impede aim (Glatz 2000; Web: Poultryhub; Web: Nationalband) In most of the cases the lenses caused irritation on the eyes, and the blinders gave the birds difficulties with seeing the food as well as conspesifics, and have not been recommended as a solution on commercial level.

Housing conditions and nutrition

In general, good housing conditions will to a certain degree be able to decrease pecking and cannibalistic behaviour. Wide distribution of all the resources like food, water and nesting boxes will give the layers equal opportunities, and prevent encounters due to competition. Providing good nutrition that does not allow for any deficiencies, will also take away potential pecking in search of the lacking foodstuff. Often, small adjustments in the housing environment can make a difference, most of them being easy and cheap to conduct. Studies on different housing environments show that enrichments that make the environment more interesting for the birds have a positive effect on lowering harmful pecking. Providing perches to sit on (Gunnarsson 1999; Wechsler & Huber-Eicher 1998), toys/objects to investigate (Gvaryahu et al. 1994; Jones & Carmichael 1998; Jones et al. 2002), and suitable types of

bedding that stimulate ground pecking and dust bathing, have a decreasing effect on feather pecking and cannibalism (Blokhuis & Wiepkema 1998; Braastad 1990). Feeding fibrous feed that takes longer time to consume will activate the layers around the food tray, and stimulate the behavioural need of foraging (Aerni et al. 2000; El-Lethey et al. 2000; Hartini et al. 2002). The more time the layers spend on other activities, the less time they will have to do destructive pecking.

There have also been trials on group sizes and stocking density. In the wild, the chicken would be living in smaller groups widely spaced in the forest (Web: Gallus Gallus; Web: Red Junglefowl). In commercial systems, the birds are crowded tightly in cages, or in large numbers on a restricted area in aviary or free range systems. Large groups (Allen & Perry 1975; Bilčík & Keeling 2000) and high density has shown to increase the level of pecking and cannibalism (Hughes & Duncan 1972; Nicol et al. 1999), and in none-cage systems, also the natural, stable flock composition is disturbed. Giving the layers enough space and "privacy" are factors that can decrease destructive behaviour, but are hard to defend economically and efficiency-wise. However, some farmers are defending the extra cost of having roosters mixed in with the hens. Even though they do not lay eggs, they may seem to have a calming effect on the hens, raising the threshold for performing destructive pecking (personal conversations with farmers).

Rearing

The first days and weeks in an animal's life are for many species the most important for learning normal behaviour (Bolhuis & Honey 1998; Rice & Barone 2000; Scott 1958). Today, the commercial layers are hatched in a hatchery, where heat is given by lamps, and no mother is there when the chickens comes out. After hatching, the chickens are reared together with other chickens of the same age in large groups. Normally, the mother would have been there to take care of her young, teaching them how to search for food by exploring, and directing pecking towards the ground instead of towards other individuals. Research has shown that chickens brooded by a mother hen had a stronger exploratory motivation (Rodenburg et al. 2009), and a lower mortality rate due to cannibalism and feather pecking (Riber et al. 2007). However, to allow maternal brooding/rearing in great scale can be a challenge, but is still a potential way to lower feather pecking and cannibalistic behaviour. Another option that might be more achievable is to rear the chickens on litter ground, that will, even though the mother is not there, stimulate ground pecking (Blokhuis & Wiepkema 1998; Johnsen et al. 1998) and imprint this behaviour instead of feather pecking.

Breeding

Next to good management, breeding is now starting to be regarded as the best way to permanently solve the feather pecking and cannibalism problem. Developing strains of layers that have little, to no motivation for destructive pecking will make other extreme measures like beak trimming unnecessary. Research has shown that it is possible to breed for low feather pecking strains, where the layers show less motivation for pecking activity, without influencing the production negatively (Muir 1996; Su et al. 2006). Low pecking strains combined with good management and housing systems should together lead to a low morbidity and mortality rate among the animals, and benefit the farmer economically as well as ethically, since no farmer enjoys watching his birds suffer. There are different approaches of selection. One method can be behavioural observations to see which individuals perform cannibalism and/or pecking (Hocking et al. 2004; Kjaer et al. 2001; Van Hierden et al. 2002), but this method can be time consuming, costly and inaccurate. Another method is to record the survival time of the individual layers, which can be analyzed using e.g. a direct-indirect effects model, resulting in a breeding value for the individual layer (direct effect) and a breeding value for the influence the layer has on her associates (social effect) (Bijma et al. 2007; Ellen et al. 2008).

As mentioned in the introduction, new methods have been tried to improve the estimation of genetic parameters, but the models face some difficulties with time-dependent effects and censored records, which might be overcome using more complicated models like the repeatability model. The next sections will explain the dataset used for the analysis, and what is included in the different models.

Material

The dataset

The data used in this thesis was the same data as was described in the two papers on survival time in laying hens (Ellen et al. 2008; Ellen et al. 2010). The survival time records were produced from three purebred White Leghorn layer lines collected from Institut de Sélection Animale B.V, a Hendrix Genetics Company. In this study, only data of line WB (6916 records) were analyzed.

Housing

The survival time of the hens were observed for only a single generation. Chickens were hatched in 2 batches, but as it was not possible to hatch all the chickens at once, 4 age groups separated by 2 weeks each were within each batch. All the chickens had intact beaks, and were wing banded in one wing. After birth, the chickens were reared in bigger group cages, first with 60 individuals per cage, then down to 20 per cage from 5 weeks onwards. At an average of 17 weeks of age, the chickens were allocated at random to traditional 4-bird battery cages, each batch placed in its own laying house along with mostly unrelated birds of same line and age. After housing, hens were wing banded in the other wing as well, to prevent loss of wing band numbers.

Laying house 1 had windows and 8 double racks of cages that were 3 levels tall, while laying house 2 did not have windows, and only used the bottom and middle level of the racks. Corridors for cage access separated the racks. The hens could make contact with the hens in the back cage through the mesh, but not with their neighbors on the sides which had solid walls as separation. Because of the contact in the back wall, a cage had mostly hens of same line and age in the corresponding back cage. To allow equal spread of the lines and age groups in the laying houses, each level was divided into blocks of 10 cages of the same line and age. Feed were given ad libitum in the front of the cages, while the drinking nipples were in the back, and shared with the back cage. The light intensity was on average higher in laying house 2, but since laying house 1 had windows, the lighting there was highly variable (Ellen et al. 2008). The layers started with a light period of 9 hours/day, up to 16 hours/day at 26 weeks of age.

Pedigree

Basically the same sires were used for the two batches, minus a few sires for batch number 2 due to death. 35 sires and 276 dams were used for the layers in laying house 1, and 33 sires and 261 dams for laying house 2. The dams were different for the two batches. The mating was at random, where on average each sire was mated with 8 dams, and each dam gave rise to 12.3 female offspring. A pedigree of five generations was available to include in the calculations. Hens with unknown or double ID, were marked as having unknown pedigree.

Recordings

Every day dead hens were removed, and identity, cage number and cause of death (subjectively determined) were recorded. The dead hens were not replaced, and at 75 weeks,

the study was terminated. Each hen then had information on survival (alive or dead, 0/1) and survival days (from start of study to death or end of study (max 447 days)).

Methods

Data analysis

The data was analyzed using R, an R expansion pack; *R commander* and ASReml. First, R commander was used to find which effects in the experiment had a significant effect on survival (0/1). The variables that were the most important and showed significant effect (on 0.05 level) were; 1: the stable-rack-level combination for housing, 2: if the cage had back-cage neighbors; yes or no (1/0), and 3: the back-cage mortality (1-4). Other possible fixed effects were investigated too, like hatch-week, and which line the layers in the back-cage belonged to. Even though age is a common effect to include in a model, it was excluded here, as in this experiment; age was nested within cage and house and therefore not suitable to have as a separate fixed effect. Also, when arranging the layers, the same lines were always tried placed back-to back, so only hens of the same line could be in contact. The cases where this was not true were not substantial, so back-cage line as a fixed effect was excluded as well, in favor of having back-cage neighbor (0/1) as a fixed effect instead.

A difficulty in the dataset was that the cages that did not have a back-neighbor, resulted in missing values for the back-cage mortality effect. However, it could be that having no neighbors instead of neighbors has an effect on the individual's survival. Therefore, the layers which had no neighbors in the back-cage got a 0 for back-cage effect, and the back-cage mortality of these empty cages was set as 2, the average mortality of 4 hens. Further, regular R was used to arrange and duplicate the dataset when needed, and ASReml to estimate the genetic variances, covariances and correlations, as well as the heritability of survival, breeding values and the values of other random effects.

Models

In this study, survival (0/1) was analyzed using six different models; a linear direct-effect sire model, a sire model with a random cage effect, a direct-indirect-random cage effects sire model, a sire-dam direct-indirect-random cage effects model, a repeatability direct-indirect-random cage effects model, and a repeatability direct-indirect-random cage effects model with time dependent social effects.

Sire model - direct effect

The first model is a classical model that has been used often in animal breeding, the sire model that only accounts for the direct effect of the animal itself. In this case, the direct effect of interest is survival; if the animal died or survived (1/0), which gives information that can be used to estimate the genetic parameters, and predict the breeding values of the animals' sires; the random effect. Depending on which animals that is included in the pedigree, the model will predict breeding values for all of these, but based on the connections to the sires (BV_{layer} ~$1/2BV_{sire}$). Even though survival is a threshold character (e.g. dead/alive, yes/no, sick/healthy) which usually is treated as a binomial trait, it will be analyzed as a linear trait to overcome some difficulties with including it in more complicated models (further discussed later). The model also corrects for the systematic non-genetic differences between the observations- the fixed effects; here, the stable-rack-level combination, back-cage neighbors (1/0) and back-cage mortality (1-4). For calculating the estimations and predictions, Best Linear Unbiased Prediction/ Estimation (BLU(P/E)) can be used (Mrode & Thompson 2005).

The sire model in matrix notation:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Zs} + \mathbf{e} \quad (1)$$

where

y = *n* x 1 vector of events - dead (1) or alive (0); *n* = number of observations
b = *p* x 1 vector of fixed effects; *p* = number of levels of fixed effects
s = *q* x 1 vector of random effects; here- breeding values of the sires; *q* = number of sires
e = *n* x 1 vector of random residual effects; *n* = number of observations
X = design matrix of order *n* x *p* which connects the observations to the fixed effects
Z = design matrix of order *n* x *q* which connects observations to the random effects

The variance of the residuals, which include random environmental effects and non-additive genetic effects, are independently distributed so that var(**e**) = $\mathbf{I}\sigma^2_e$ = **R**, and var(**s**) = $\mathbf{A}\sigma^2_{Sire}$ = $\mathbf{A}^*0.25\sigma^2_{AD}$ = **G** and var(**y**) = **ZGZ**' + **R**, where **I** is an identity matrix, and **A** is the relationship matrix that connects the pedigree to the sire variance. The solutions for **b** and **s** are estimated and predicted by Mixed Model Equations (MME):

$$\begin{bmatrix} \mathbf{X}'\mathbf{R}^{-1}\mathbf{X} & \mathbf{X}'\mathbf{R}^{-1}\mathbf{Z} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{X} & \mathbf{Z}'\mathbf{R}^{-1}\mathbf{Z} + \mathbf{G}^{-1} \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{s}} \end{bmatrix} = \begin{bmatrix} \mathbf{X}'\mathbf{R}^{-1}\mathbf{y} \\ \mathbf{Z}'\mathbf{R}^{-1}\mathbf{y} \end{bmatrix} \quad (2)$$

Sire model - direct and random cage effect

Since the layers were confined in permanent cages during the whole experiment, with no exchange or moving of hens between locations, one can assume that there will be a unique environment within each cage that may have a non-genetic effect on survival. Therefore, the second model is identical to the sire model above, but with a random cage effect included; **Wc**, where **W** is a design matrix of order *n* x *t* which connects the observations to each cage, and **c** is a *t* x 1 vector of random cage effects. This model will give the $\hat{\mathbf{b}}$ values for the fixed effects and the breeding values of the sires as usual, and additionally a cage effect value $\hat{\mathbf{c}}$ for each of the *t* cages. The random cage effect will capture differences in survival among the cages compared to the average, and explain more of the non-heritable social relationship.

Sire model - direct, indirect and random cage effect

Within each cage, there are 4 layers that have the chance to interact with each other. This is known as the indirect genetic effect (or social effect/associative effect), where the behaviour or phenotype of one individual has an effect on another individual. The indirect effect will in other words give a measurement on an individual's effect on the survival of the cage-mates, as it is closely related to its own pecking behaviour. All the birds have known identity and records on survival (0/1), which can be connected to the survival of the other cage members. In this third model, the fixed effects stays the same as before, but a random indirect effect (using the sires of the cage-mates) is added, in addition to the direct effect and the random cage effect. The model will estimate an indirect breeding value (IBV) for all the birds in the pedigree, based on the survival of the layer's 3 cage-mates, in addition to the direct breeding values (DBV). The indirect effect still only consist of the information if cage-mates died of cannibalism or not, and not at which particular time this happened, which will be added later.

The model in matrix notation:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_D s_D} + \mathbf{Z_I s_I} + \mathbf{Wc} + \mathbf{e} \qquad (3)$$

where

$\mathbf{y}$ = *n* x 1 vector of events - dead (1) or alive (0); *n* = number of observations
$\mathbf{b}$ = *p* x 1 vector of fixed effects; *p* = number of levels of fixed effects
$\mathbf{s_D}$ = *q* x 1 vector of random effects - direct breeding values; *q* = number of sires
$\mathbf{s_I}$ = *q* x 1 vector of random effects - indirect breeding values; *q* = number of sires
$\mathbf{c}$ = *t* x 1 vector of random cage effects; *t* = number of cages

e = *n* x 1 vector of random residual effects; *n* = number of observations

X = design matrix of order *n* x *p* which connects the observations to the fixed effects

$\mathbf{Z_D}$ = design matrix of order *n* x *q* which connects observations to the random direct effect

$\mathbf{Z_I}$ = design matrix of order *n* x *q* which connects observations to the random indirect effect

W = design matrix of order *n* x *t* which connects the observations to each cage

The covariance structure of the direct and the indirect effects is var $\begin{bmatrix} \mathbf{s_D} \\ \mathbf{s_I} \end{bmatrix} = \mathbf{C} \otimes \mathbf{A}$ where

$\mathbf{C} = \begin{bmatrix} \sigma^2_{SireD} & \sigma_{DI} \\ \sigma_{DI} & \sigma^2_{SireI} \end{bmatrix} = \begin{bmatrix} g^{11} & g^{12} \\ g^{21} & g^{22} \end{bmatrix}$. The σ^2_{SireD} is the direct sire variance ($0.25\sigma^2_{AD}$), σ^2_{SireI} is the indirect sire effect ($0.25\sigma^2_{AI}$) and σ_{DI} is the genetic covariance between direct and indirect effects ($0.25\sigma_{A_{DI}}$). The $\otimes$ sign indicates the Kronecker product of the matrices.

The solutions for $\mathbf{b}$, $\mathbf{s_D}$, $\mathbf{s_I}$ and $\mathbf{c}$ is estimated by an expanded MME (Muir 2005):

$$\begin{bmatrix} \mathbf{X'R^{-1}X} & \mathbf{X'R^{-1}Z_D} & \mathbf{X'R^{-1}Z_I} & \mathbf{X'R^{-1}W} \\ \mathbf{Z_D'R^{-1}X} & \mathbf{Z_D'R^{-1}Z_D + g^{11}A^{-1}} & \mathbf{Z_D'R^{-1}Z_I + g^{12}A^{-1}} & \mathbf{Z_D'R^{-1}W} \\ \mathbf{Z_I'R^{-1}X} & \mathbf{Z_I'R^{-1}Z_D + g^{21}A^{-1}} & \mathbf{Z_I'R^{-1}Z_I + g^{22}A^{-1}} & \mathbf{Z_I'R^{-1}W} \\ \mathbf{W'R^{-1}X} & \mathbf{W'R^{-1}Z_D} & \mathbf{W'R^{-1}Z_I} & \mathbf{W'R^{-1}W + T^{-1}} \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{s}}_\mathbf{D} \\ \hat{\mathbf{s}}_\mathbf{I} \\ \hat{\mathbf{c}} \end{bmatrix} = \begin{bmatrix} \mathbf{X'R^{-1}y} \\ \mathbf{Z_D'R^{-1}y} \\ \mathbf{Z_I'R^{-1}y} \\ \mathbf{W'R^{-1}y} \end{bmatrix} \quad (4)$$

Where the g^{ij} is an expression for the elements of the inverse of **C**, $\mathbf{T^{-1}}$ is the inverse of $\mathbf{I}\sigma^2_{Cage}$ and $\mathbf{R^{-1}}$ is the inverse of the $\mathbf{I}\sigma^2_e$ matrix.

Sire and dam model - direct, indirect and random cage effect

In livestock, the sire is usually the parent that has the biggest influence on the gene pool of a population, as he is able to mate with several females in the same breeding season, and give rise to a bigger number of progeny than the individual dams do through their whole production time. In modern breeding programs, the dam's influence is most often also taken into account in maternal effect-models, and/or they are included in the pedigree to be used for breeding value prediction in animal models. In contrary to bigger production animals like cattle, which most often only give birth to one calf a year, chickens can produce a large amount of offspring in a relatively short amount of time. This means that the hen will have more than only a couple of records on the offsprings' survival time, and her breeding value will be more accurate. In this experiment, the sires were mated to around 8 dams, and each dam produced approximately 12.3 female chickens each. This will make also the dam a somewhat large contributor to the breeding, even though not as substantial as the sires. In this model, the dams are included to see how this will affect the genetic parameters. The dam is not added as a new random effect, but in one random effect together with the sire, so that the

relationship of full-sibs is also included. The direct additive genetic effect will therefore be based on not only the sire, but the sire and dam together. The dams of the cage-mates are also added for the random social effect, to play a part in estimating the indirect genetic effect. The fixed effects stay the same, as well as the random cage effect, and the model in matrix notation will look the same as Equation (-3-).

Repeatability model

For a farmer, the actual time of death of his layers is important, as he will lose more money from layers that die early in the laying period, compared to the ones that die on a later stage right before flock replacement. In this model, the time of death is accounted for by expanding the dataset to include time with repeated observations on survival each month. The experiment lasted for maximum 447 days, which means about 14.5 months, but not all hens were slaughtered after exactly 447 days as some groups were slaughtered a bit earlier. To have equal sized "months", the trial was divided into 14 periods (447/14). The dataset was duplicated 14 times, and added a time variable of 14 periods. Then a new y-vector was made, where every time period got a record of survival based on the number of survival days. If the hen was still alive in period 1, period 1 got a 1, if it was still alive in period 2, it got another 1 in period 2, etc. The period where the layer died of cannibalism received a 0, and all periods after this received a "NA" for missing value. For the censored animals, the same procedure was followed, except they did not receive a 0 for death, since as they got slaughtered, the actual "natural" time of death is not known. Therefore they received either a 1 or a NA in the last period depending on the day of slaughter (late or early). Notice that the record for death is now switched to a 0 instead of a 1, as this is easier for the computer programs to understand when dealing with repeated records. The fixed effect "period" was also included in the model in addition to the previous fixed effects. Both sire and dam were included for the random effects like in the Sire-Dam model. Since the model now is time dependent, so is the random cage effect, as it is logical to believe that the cage environment may change through the periods. The random cage effect was made time dependent by fusing cage number and period number.

The repeatability model is usually in the form

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wpe} + \mathbf{e} \qquad (5)$$

in an animal model, where **pe** is a vector of permanent environmental effects and non-additive genetic effects, in case an individual e.g. had a better or worse rearing than another individual

that will affect the repeated records. This effect is assumed uncorrelated with the additive genetic effects, and allows partitioning of the environmental effect (residual) into permanent and temporary components (Mrode & Thompson 2005). The permanent effect will be individual for all the layers, but in this case when working with a threshold trait, the hen can only be dead or alive, and it is not possible to fit a permanent environmental effect (discussed further later). Therefore, the model in matrix notation would look the same as Equation (-3-), but with differences in the vectors and matrices:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_D p_D} + \mathbf{Z_I p_I} + \mathbf{Wc} + \mathbf{e} \qquad (6)$$

where

$\mathbf{y}$ = 14n x 1 vector of repeated records on survival (alive (1), dead (0), after death (NA)); n = number of observations x 14 periods

$\mathbf{b}$ = p x 1 vector of fixed effects; p = number of levels of fixed effects

$\mathbf{p_D}$ = q x 1 vector of random effects- direct breeding values; q = number of sires and dams

$\mathbf{p_I}$ = q x 1 vector of random effects - social breeding values; q = number of sires and dams

$\mathbf{c}$ = t x 1 vector of random cage effects; t = number of cages

$\mathbf{e}$ = 14n x 1 vector of random residual effects; n = number of observations x 14 periods

$\mathbf{X}$ = design matrix of order 14n x p which connects the observations to the fixed effects

$\mathbf{Z_D}$ = design matrix of order 14n x q which connects observations to the random direct effect

$\mathbf{Z_I}$ =design matrix of order 14n x q which connects observations to the random indirect effects

$\mathbf{W}$ = design matrix of order 14n x t which connects the observations to each cage

Repeatability model - time dependent social effects.

As layers die of cannibalism (or for other reasons) along the laying period, the environment is no longer the same within a cage. A dead hen can no longer peck another hen, and this might change the dynamics within the cage. In this model the social effects are made time dependent by replacing the IDs of the sires and dams of the cage-mates with a 0 in the periods where the cage-mate was dead and no longer in the cage. For example, for a layer i that had a cage-mate that died in period 4, the cage-mate will get a 0 for its sire and dam from period 4 to 14 in the Z_I matrix. This will make the social effect change through the periods, which is more realistic than keeping it constant. All other components of the model are the same as in the repeatability model, and the model will look like Equation (-6-) in matrix notation.

Total heritable variation

When dealing with social interactions in a group, the animal's heritable impact on the group members' phenotype has to be included in the breeding value. The total breeding value (TBV) of an individual will therefore consist of the sum of its own direct breeding value (DBV), and *n*-1 times its indirect breeding value (IBV); TBV = DBV_i + (n-1) IBV_i, where *n* is the number of animals in the group/cage (in this case; 4) (Muir 2005). The total heritable variance is calculated as the variance of the TBV among the animals (Bijma et al. 2007); $\sigma^2_{TBV} = \sigma^2_{AD}+2(n-1)\sigma_{DI}+ (n-1)^2 \sigma^2_{AI}$. If assuming that the layers in each cage are mostly unrelated, the phenotypic variance equals $\sigma^2_P = \sigma^2_{SireD}+ (n-1)\sigma^2_{SireI}+ \sigma^2_{Cage}+ \sigma^2_e$, including the random cage effect. The total heritability (T^2) is given as the ratio between σ^2_{TBV} and σ^2_P; $\sigma^2_{TBV}/\sigma^2_P$.

Model comparison

To compare the fit of the different models, some methods for model comparison are needed. The three methods chosen are the Log likelihood ratio test, the Akaike information criterion (AIC), and Cross validation.

Log Likelihood Ratio Test

The Log likelihood (LogL) ratio test is a common test that can be performed between nested models, where the simpler model lays within the more complicated one with additional parameters (Lynch & Walsh 1998). The likelihood is the probability of obtaining such data, given the parameter estimates. The best model will find values for the coefficients that maximize the value of the likelihood function, which means that it finds the parameter estimates that make the data most likely to occur. With a LogL ratio test, the models are tested against each other to see whether the observed difference is statistically significant (Lynch & Walsh 1998). The log likelihood of the model can be obtained from the ASReml output, where the value is shown for each iteration when estimating the parameters. The last iteration will show the final log likelihood value.

The test statistic is often called D, and follows a chi square distribution with *k*-1 degrees of freedom, where *k* is the number of parameters estimated;

D = -2 ln(likelihood null model) + 2 ln(likelihood alternative model) (7)

where the null model denotes the first model, which is the simplest, against the more complex model in the comparison, the alternative model. P-values < 0.05 are considered as a significant difference between the models, and the simplest model in the test can be rejected.

When two models are not completely nested in each other, it is not possible to compare them using Log likelihood ratio test. Another way of comparing them can instead be by looking at the standard error, where the model with the smallest parameter SE will most likely be the one that has the best fit (as the real values come closer to the estimated), or perform the Akaike information criterion test.

Akaike Information Criterion Test

The Akaike Information Criterion test (AIC) is based on the Kullback-Leibler divergence which represents the loss of information when using one model to approximate another model (Posada & Buckley 2004), and the maximized log likelihood. The formula is defined as

$$AIC = -2 \ln(\text{likelihood}) + 2K \quad (8)$$

where the log likelihood can be taken from computer outputs from the models, and the K is the number of estimated parameters in the model (all variables + mean) (Lynch & Walsh 1998). AIC deals with the tradeoff between the fit of a model and its complexity when giving a "penalty" (the K). The penalty discourage over-fitting by not favoring the more complex models just because of their increased fit to the data. Since the AIC represents "loss of information", the model with the lowest AIC is preferred. Since AIC is on a relative scale, it is common to present the AIC differences (ΔAIC) as well. This is the difference between the AIC of a model *i* and the AIC_{min}, which is the smallest AIC value among the models that are compared (Posada & Buckley 2004).

$$\Delta AIC = \Delta i = AIC_i - AIC_{min} \quad (9)$$

As a "rule of thumb" $\Delta i < 2$ suggest that model *i* is a good model, Δi between 4 and 7 indicate a less good model, and $\Delta i > 10$ receives no support as it has large distance to the best model (Posada & Buckley 2004).

Cross Validation

To see how well a model predicts breeding values, a cross validation can be done. When using cross validation, known phenotypes are set as unknown and predicted using estimated breeding values. The predicted phenotype will then be compared with the observed phenotype. To do this, a random number was assigned to each cage, and used to divide the full dataset into 5 parts of 20% each. From the full dataset, the 20% parts were set as missing once, giving 5 larger subsets all containing 80% of the cages. In this way, all cages were removed only once, and all classes of fixed effects were present in all the remaining subsets.

For each dataset, with the 20% missing individuals, breeding values for the direct effect (DBV) and the indirect effect (IBV) were estimated. To predict the phenotypes for the animals set to missing, the DBVs and the IBVs were taken from the model outputs of the subset where they were not present, as these values are predicted without having the observed phenotype included. The predicted phenotype (P_{pred}) was set as a combination of the DBV of the animal itself, and the IBVs of its 3 cage-mates; $P_{pred,i} = DBV_i + IBV_1 + IBV_2 + IBV_3$. To correct for the fixed effects, the whole dataset was then analyzed upon survival time (in days), with only the fixed effects as variables. From this, the model output gave a predicted survival time ($\hat{y}$) and a residual for each layer. The residual is denoted as $P_{corrected}$, as it includes the layers' breeding values. The $P_{corrected}$ and P_{pred} values were then converted to ranks (best BVs get highest ranks), which could be compared by Spearman's rank correlation coefficient ρ (rho). Spearman's rho is a way to measure the strength of the association between two ranked variables, and is defined as

$$\rho = \frac{\sum_i (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_i (x_i - \bar{x})^2 \sum_i (y_i - \bar{y})^2}} \quad (10)$$

where x_i is the rank of $P_{corrected}$, and y_i is the rank of P_{pred} for layer i. If some phenotypes have identical values (ties), they are assigned a rank equal to the average of what their ranks would otherwise be (Web: Spearman). For the $P_{corrected}$ values, the censored animals could not be ranked based on their survival days, since you do not know their true time of death if they were allowed to live on. Therefore, the ranks of the censored animals were set as the mean of all the ranks they would have otherwise been given (Ellen et al. 2010). All the subsets were ranked and rho-scored separately.

Results

Survival rate

Attachment 1 gives an overview of the survival rate and time of the layers in the different laying houses, racks and levels. Overall, in laying house 1, the survival rate for the racks range between 43% and 65%, and levels between 48% (top level, closest to the lights) and 53% (bottom level), with an average of 52% over the whole house. In laying house 2, the survival rate of the racks range between 50% and 69%, and levels between 56% and 57% (middle and bottom, only two levels), with a house average of 57%. The fixed effects used in the models were significant with a p-value < 0.05.

Direct effects

Table 1 shows the genetic parameters for the Sire direct effect and the Sire direct-random cage effects model. They both gave the same results for the direct genetic variance (σ^2_{AD}) and the phenotypic variance (σ^2_P), 0.0282 ± 0.0081 and 0.2414 ± 0.0044 respectively. The random cage variance (σ^2_{Cage}) was very small; 0.0001 ± 0.0023 and not significantly different from zero. The heritabilities (h^2) for both models were similar ~0.1168 ± 0.0327 (Table 1).

Direct-indirect effects

The Sire direct-indirect-random cage model and the Sire-Dam model are the models that include the effect of social interactions without using repeated observations on survival. Both gave comparable results for σ^2_{AD} (0.0278 ± 0.0080 and 0.0279 ± 0.0053 respectively) and σ^2_P (0.2414 ± 0.0045 and 0.2345 ± 0.0040 respectively), and were very similar to the direct effects models. The indirect additive variances (σ^2_{AI}) were also comparable, with 0.0019 ± 0.0008 for the Sire direct-indirect-random cage model and 0.0016 ± 0.0006 for the Sire-Dam model. The covariance between the direct and indirect effect (σ_{DI}) were moderately negative, but not significantly different from zero, with lowest σ_{DI} for the Sire direct-indirect-random cage model (-0.0034 ± 0.0019). The total heritable variance (σ^2_{TBV}) differed between the two models, where the Sire-Dam model yielded the highest σ^2_{TBV} (0.0273 ± 0.0091), compared to the Sire direct-indirect-random cage model (0.0252 ± 0.0108). The random cage effects (σ^2_{Cage}) were very small, and not significant different from zero. The genetic direct-indirect correlation (r_{DI}) was strongest and significant for the sire direct-indirect-random cage model (-0.4587 ± 0.2185) compared to the Sire-Dam model (-0.3704 ± 0.1941) where r_{DI} was not significant. The total heritability (T^2) was highest for the Sire-Dam model with 0.1165 ± 0.0387 compared to the Sire direct-indirect-random cage model with 0.1045 ± 0.0444 (Table 1).

Repeatability

Table 2 shows the results of the repeatability models. The additive direct effect (σ^2_{AD}) for both repeatability models were quite similar, with the highest value for the repeatability model without time dependent social effects (REP; 0.599E-3 ± 0.109E-3). The indirect effect (σ^2_{AI}) was highest for the model with time dependent social effect (REP_{time}), with 0.102E-3 ± 0.278E-4 compared to 0.427E-4 ± 0.149E-4. REP showed the lowest covariance (σ_{DI}) between direct and indirect effects (-0.784E-4 ±0.299E-4) compared to REP_{time} with -0.101E-3 ± 0.397E-4. The σ^2_{TBV} was highest for REP_{time} (0.898E-3 ± 0.283E-3).

Table1. Estimates of the genetic parameters[1] ± SE for the direct models and the direct-indirect effects models.

Parameter	**Sire** **- direct**	**Sire** **- direct** **- random cage**	**Sire** **- direct** **- indirect** **- random cage**	**Sire - Dam** **- direct** **- indirect** **- random cage**
σ^2_{AD}	0.0282 ± 0.0081	0.0282 ± 0.0081	0.0278 ± 0.0080	0.0279 ± 0.0053
σ^2_{AI}	-	-	0.0019 ± 0.0008	0.0016 ± 0.0006
σ_{DI}	-	-	-0.0034 ± 0.0019	-0.0024 ± 0.0013
σ^2_{TBV}	-	-	0.0252 ± 0.0108	0.0273 ± 0.0091
σ^2_{Cage}	-	0.0001 ± 0.0023	0.64E-7 ± 0.110E-8	0.607E-7 ± 0.107E-8
σ^2_e	0.2344	0.2342	0.2330	0.2263
σ^2_P	0.2414 ± 0,0044	0.2414 ± 0.0045	0.2414 ± 0.0045	0.2345 ± 0.0040
r_{DI}	-	-	-0.4587 ± 0.2185	-0.3704 ± 0.1941
h^2	0.1168 ± 0.0327	0.1169 ± 0.0327	-	-
T^2	-	-	0.1045 ± 0.0444	0.1165 ± 0.0387

[1]σ^2_{AD}= the additive direct genetic effect: $4\sigma^2_{SireD}$; σ^2_{AI}= the additive indirect genetic effect: $4\sigma^2_{sireS}$; σ_{DI} = the genetic covariance between direct and indirect effects: $4\sigma_{covar}$; σ^2_{TBV} = the total heritable variance: σ^2_{AD}+2(n-1)σ_{DI}+ (n-1)$^2\sigma^2_{AI}$; σ^2_P = the phenotypic variance: σ^2_{SireD}+ (n-1)σ^2_{sireI}+ σ^2_{Cage}+ σ^2_e; σ^2_e = the residual variance; r_{DI} = the genetic correlation between direct and indirect effects; h^2= the additive heritability (σ^2_{AD}/σ^2_P); T^2 = the total heritability ($\sigma^2_{TBV}/\sigma^2_P$).

Table 2. Estimates of the genetic parameters[1] ± SE for the repeatability model and the repeatability model with time dependent social effects.

Parameter	**Repeatability**	**Repeatability** **- time dependent social effects**
σ^2_{AD}	0.599E-3 ± 0.109E-3	0.584E-3 ± 0.108E-3
σ^2_{AI}	0.427E-4 ± 0.149-4	0.102E-3 ± 0.278E-4
σ_{DI}	-0.784E-4 ±0.299E-4	-0.101E-3 ± 0.397E-4
σ^2_{TBV}	0.513E-3 ± 0.177E-3	0.898E-3 ± 0.283E-3
σ^2_{Cage}	0.0029 ± 0.162E-3	0.0027 ± 0.174E-3
σ^2_e	0.0392	0.0393
σ^2_P	0.0423 ± 0.226E-3	0.0423 ± 0.226E-3
r_{DI}	-0.4898 ± 0.1621	-0.4131 ± 0.1485
T^2	0.0121 ± 0.0042	0.0212 ± 0.0067

[1]σ^2_{AD}= the additive direct genetic effect: $4\sigma^2_{SireD}$; σ^2_{AI}= the additive indirect genetic effect: $4\sigma^2_{sireI}$; σ_{DI} = the genetic covariance between direct and indirect effects:$4\sigma_{covar}$; σ^2_{TBV} = the total heritable variance: σ^2_{AD}+2(n-1)σ_{DI}+ (n-1)$^2\sigma^2_{AI}$; σ^2_P = the phenotypic variance: σ^2_{SireD}+ (n-1)σ^2_{sireI}+ σ^2_{Cage}+ σ^2_e; σ^2_e = the residual variance; r_{DI} = the genetic correlation between direct and indirect effects; T^2 = the total heritability ($\sigma^2_{TBV}/\sigma^2_P$).

For both REP and REP_{time}, the random cage effect was time dependent, resulting in a higher σ^2_{Cage} than for the direct and direct-indirect effects models, with the highest value for REP (0.0029 ± 0.162E-3). The phenotypic variances (σ^2_P) were the same in both models (0.0423 ± 0.226E-3). As the covariances were negative, so were the correlations (r_{DI}) between the direct

and indirect genetic effects, with a stronger correlation for REP (-0.4898 ± 0.1621) compared to REP_{time} (-0.4131 ± 0.1485). The total heritability (T^2) was very low for both models, with REP having 0.0121 ± 0.0042 and REP_{time} having 0.0212 ± 0.0067.

Model testing

Table 3 shows the results of the Log likelihood ratio test. Statistical comparison of the Sire direct effects model and the Sire direct-random cage effects model did not show a significant improvement. This means that the cage effect in this case was not substantial enough to make a huge difference on the estimated parameters. Statistical comparison of the Sire-direct effect and the Sire-direct-indirect-random cage effects model showed significant improvement of the goodness of fit ($P < 0.05$). This means that for this dataset, the more complex model is the better one for estimating the genetic parameters.

Table 4 shows the results of the Akaike information criterion test for all models. The AIC value was clearly lowest for the REP_{time} model, with close value to the REP model, but large differences to the sire models. As REP_{time} had the lowest AIC value, it was set as AIC_{min} for calculating ΔAIC for all models. All models had ΔAIC values > 10, which is, based by "the rule of thumb", considered a lower fit than the AIC_{min} model (Posada & Buckley 2004).

The results for the Cross validation are shown in table 5. The Sire direct effects model yielded the lowest average ρ (0.13605) followed by the Sire direct-random cage model (0.13612), REP (0.15454), REP_{time} (0.15671), and Sire direct-indirect-random cage effects model (0.15779), with the Sire-Dam model giving the highest average value (0.17235).

Table 3: Log Likelihood Ratio Test[1] between the different models.

Test	**Model 1**	**Model 2**	**Log likelihood model 1**	**Log likelihood model 2**	**D**	**df_2- df_1**	**p-value**
1	Sire - direct	Sire - direct - random cage	1401.43	1401.43	0	2-1 = 1	1
2	Sire - direct	Sire - direct - indirect - random cage	1401.43	1409.96	17.06	4-1 = 3	0.00068

[1]*The log likelihood values for each model is retrieved from ASReml output. D = -2 ln(likelihood$_1$) + 2 ln(likelihood$_2$). P-values < 0.05 = significant difference, and model 1 can be rejected.*

Table 4. AIC test[1] and ΔAIC values[2] of all the models.

Model	**K**	**Log likelihood**	**AIC**	**ΔAIC_i**
Sire - direct	5	1401.43	-2792.86	10446.9
Sire -direct - random cage	6	1401.43	-2790.86	10448.9
Sire - -direct -indirect -random cage	8	1409.96	-2803.92	10435.84
Sire- Dam -direct -indirect -random cage	8	1427.51	-2839.02	10400.74
Repeatability	9	6616.33	-13214.66	25.1
Repeatability - time dependent	9	6628.88	-13239.76	0

[1]AIC = -2 ln(likelihood) + 2K where K is the number of all estimated parameters in the model (all variables + mean).[2]$\Delta AIC = \Delta i = AIC_i - AIC_{min}$ = the difference between the AIC of a model i and the AIC_{min} (smallest AIC value among compared models).

Table 5. Cross validation of all models using Spearman's rank correlation coefficient (ρ values)[1], with the average over the 5 subset for each model at the bottom.

Subset	**Sire - direct**	**Sire -direct - random cage**	**Sire - -direct -indirect -random cage**	**Sire- Dam -direct -indirect -random cage**	**REP**	**REP_{time}**
1	0.07906	0.07912	0.12291	0.15654	0.16119	0.18092
2	0.18233	0.18240	0.20129	0.21301	0.18159	0.15729
3	0.13995	0.14004	0.14375	0.16471	0.14047	0.15659
4	0.16344	0.16335	0.16690	0.16425	0.12333	0.11593
5	0.11551	0.11570	0.15412	0.16319	0.16612	0.17283
Average	0.13605	0.13612	0.15779	0.17235	0.15454	0.15671

[1]Spearman's rank correlation coefficient is explained in Methods- Cross validation. The subsets are the parts (20%) that were set as missing.

Discussion

In this thesis, six different models were compared to analyze survival (0/1) in a purebred layer line. Based on the cross validation, the Sire-Dam model was the best model for predicting the phenotypes using estimated breeding values.

Animal model vs. Sire model

To analyze the survival data, a sire model was chosen as a base for building the different breeding models upon. Other studies have used an animal model to analyze data on survival days (Ellen et al. 2008; Peeters et al. 2012), in opposite to this study where the trait of interest was survival (0/1) instead of survival days. Survival is a binary trait that can only have two values, 0 or 1. The layers have only one observation on themselves that can tell something about the breeding value, and no offspring for themselves, which makes an animal model unsuitable for analyzing such survival data (0/1). The sires on the other hand, are fathers to several layers, which makes it possible to calculate a mean survival rate of the offspring. This makes the sire model a good alternative to the animal model. Unfortunately, using a sire model may give EBVs that are less accurate than an animal model, as well as giving potential bias since the relationship between the dams, and the full-sib relationship between some layers are not corrected for (some of this is corrected when using a sire-dam model).

Analyzing threshold data

Normally, a threshold trait is treated as a binomial trait, with a categorical observed scale, and a continuous underlying threshold scale (liability with normal distribution) (Lynch & Walsh 1998). The heritability has to be calculated as the underlying heritability, where the variances of the effects are on a logistic scale (residual variance = $\pi^2/3 \sim 3.289$) (Fikret 2011; Web: ASReml). This will work well in less complicated models, but when dealing with more complex models such as the repeatability models, it might give computer problems or results that are difficult to interpret. For the sire models, survival (0/1) could have been analyzed as a binomial trait, but to get comparable numbers that are easy to interpret, survival was set as a linear trait for all the models, not only the repeatability models.

Repeatability models usually include a permanent environmental effect that separates the environmental effects into a permanent and a temporary part. But when dealing with a binary trait with repeated records, this is not possible. The permanent environmental factor assume a positive relationship between the repeated records of an individual, like if you survived period one, it is more likely that you also survive period two and three, etc. But the problem with

survival data is that you can only die once, so the 0 cannot be repeated, which makes the data unsuitable for estimation of a permanent environmental factor (Ødegård et al. 2006).

Model choices

The models were constructed in an ascending order where more and more effects were accounted for to see if they would give a better model for survival analysis. The classical Sire direct effects model is a model that has been widely used in breeding. However, there has been doubt about its accuracy when dealing with survival data, since factors other than the direct genetic effect may have a large impact on mortality. These factors can be genetical, but also non-genetical where an individual's phenotype does not directly reflect its genotype. The phenotypic expression does in fact also contain a non-heritable environmental effect that can be influenced by group dynamics (P = G + E) (Lynch & Walsh 1998). Group effect is something that has shown to be important in husbandry systems with social animals (Camerlink et al. 2013; Muir & Cheng 2014; Peeters et al. 2012), and should therefore be fitted in the model, in this case, as a random cage effect. By fitting a random cage effect, the variation between the cages can be predicted. If the random cage effect is negative, it cannot be fitted as random effect. Instead, the correlation between the cages could be of interest. When looking at the Log Likelihood Ratio tests in table 3, the random cage effect does not show significance when compared to the Sire direct effect model, which would normally mean that it should be taken out. The random cage effect was still decided to be kept in the models, because of the known importance of the non-heritable effect.

Another factor that can have a large impact on survival is, as mentioned earlier, the social interactions between the animals, which is genetically inheritable. Including the indirect effect in a model and finding variance estimates for it is not always possible, especially if the animals live in large flocks where it is difficult to record which animals are affecting each other. But for animals that live in stable groups (at least for longer periods), as caged layers, pigs, rabbits etc., it is easier. For some traits, selection based solely on the direct effect of an individual may give no effect, or even the opposite effect of what you are breeding towards, while selection among groups (accounting for the indirect effect) results in large genetic improvement (Craig & Muir 1996; Muir 2005; Wade et al. 2010). This is because there is some heritable variation hidden in the social interactions and in the composition of the group, which is not fully understood yet.

Especially when the correlation between the direct and indirect genetic effect is negative (the stronger relationship, the worse), selection based on the direct effect only will result in even lower survival rate in the next generation. This can be interpreted as a competing relationship, where individuals with a good breeding value for their own phenotype (DBV) have a negative impact on the cage-mates phenotype (IBV) (Ellen et al. 2008). In other words, the surviving birds could have stayed alive by killing one or several of the other group members. Breeding with the fathers/grandfathers of these destructive layers, will give rise to a generation with a higher number of cannibalistic and pecking layers, and thereby higher mortality rates. Birds with a good TBV (high if based on survival days, low if based on survival where alive = 0) will have a high survival themselves, with minimal damage to the rest of the group. Selection of sires based on these, and on groups with high mean survival, will result in capture of the full heritable variation. The next generation will then mirror this, with calmer and less destructive layers (Wilson 1975). Because of this, direct-indirect effects models were included in this thesis to investigate the effect of social interactions on survival.

In the first 3 models, only the sires of the layers were accounted for in the pedigree, but from a breeders perspective, the more information you have available about your livestock, the better. If the whole pedigree is known, the whole pedigree should be included in BLUP to make the parameters and breeding values as accurate as possible. This is also the most common practice in modern animal breeding. The Sire-Dam model and the repeatability models also include the dam, as for the layers in this dataset, the whole pedigree for five generations was known. This will make a sire-dam model better than a sire model, since in a sire model, all the progeny of a sire is assumed to be half-sibs, but in reality they can be full-sibs. The dams can also have differences in breeding value. When including the dam in the pedigree, it will give the offspring a more accurate breeding value, since the sibling-relationship is now accounted for.

Repeatability models are usually used for different production traits, but can also be applied to survival as a 0/1 trait, as demonstrated. This is a logical approach, since for a farmer, it is economically important to keep each layer alive through the whole laying period. When including the indirect effect in a model, it is also logical to take the repeatability model a step further and account for the changing group environment as some layers will die at different times. In the time-dependent repeatability model, a problem appeared, but more related to the data and not the model. 16 of the layers had unknown parents, and the IDs of the parents were set to 0 in the pedigree, as if they were founders. This means that when changing the IDs of

the sires and dams to 0 after the death of a cage-mate, it will look like the cage-mate died already in the first period. After a closer inspection, only 9 of the 16 layers died late in the laying period, and then mostly from slaughter. Since this number is so small, and the layers are not connected to the other layers in the pedigree, it is assumed not to have a substantial effect on the indirect effect parameter, and is therefore ignored. If the number had been bigger, changing the parent IDs to a random number other than 0 could be a solution.

Findings

The results

Overall, the parameter estimates were separately comparable within the 4 sire models, and within the two repeatability models, with exception of the r_{DI} which does not show large differences between all the models. As expected, the Sire-Dam model have a larger σ^2_{TBV} than the Sire direct-indirect-random cage model since it can connect more of the relationship between the layers, and thereby capture more of the heritable variance. This can also be seen in the higher T^2.

An interesting finding is that the Sire direct-indirect-random cage model shows a lower total heritability than the heritability found in the Sire direct effects model and Sire direct- random cage effects model. This was expected to be the opposite, as adding a social component usually increases the heritability. A possible explanation is that because the covariance is negative between the direct and social effect, it makes the σ^2_{TBV} smaller than the σ^2_{AD}, and therefore gives a smaller heritability. If the covariance was positive, the heritability would have been much larger.

Another interesting finding is the random cage effect (σ^2_{Cage}), which was in general very low for the sire models, but showed a larger value when being time dependent in the repeatability models. As long as the random cage variance is positive, it can be fitted as a random effect, but it is clear that when using repeated records, the non-heritable effect was captured to a larger degree. Of the two repeatability models, the REP_{time} model gave a smaller value than REP, which might be because more of the variance is explained by the changing social interactions through time, instead of being a non-genetic effect.

The REP_{time} model had a higher σ^2_{AI} than REP, which may be because the time dependency make the social effect more accurate and thereby will find more of the variance. The σ^2_{TBV} for REP_{time} is also larger, most likely for the same reason. At the same time, the σ^2_{TBV} for the

repeatability models are lower than their additive direct genetic variances, supposedly of the same reason as with the Sire direct-indirect-random cage, and the Sire-Dam model. The negative relationship between the direct and indirect effects lowers the value.

A strange effect of the repeatability models was that the heritabilities (T^2) dropped to a very low level of only 1-2 %. The heritability was highest for the Rep_{time} model, but it is still very low compared to the results of the sire models. It seems to be no obvious reason for such a low number, and one can speculate about if something in the model makes it unable to give a good estimate. But after all, what a breeder is most interested in is the predicted breeding values, so the breeding animals can be selected. The heritability estimate itself is only a scaling factor that shows how large the response of selection will be, not the direction of the selection, or how accurate the breeding values are. The true heritability cannot be changed or increased by human interference, but if the estimated breeding values are as correct as possible, the selection would be more accurate, which makes the breeding more successful.

The results for some of the models could also be compared to what Ellen et al. (2008) found for the WB line. The Sire direct effects model gives a heritability (h^2) of 0.1168 ± 0.03, which is similar to 0.10 ± 0.02 which was found by a direct effect animal model on survival time. When comparing the total heritability (T^2) found for the Sire direct-indirect-random cage model to the direct-indirect animal model in Ellen et al. (2008), the results are more different; 0.1045 ± 0.04 vs. 0.15 ± 0.05 respectively. This is most likely because of the model differences (animal vs. sire), so the values cannot be strictly compared.

Model comparison

The LogL ratio test shows that the Sire direct-indirect-random cage model has a highly significant p-value, which makes this model a better model than both the Sire direct effects model, and the Sire direct-random cage effects model. In this case, there were no difference between the Sire direct effects model, and the Sire direct-random cage effects model, but one can assume that this might not always be the case. Depending on type of data and animal species, the group effect can be of great importance. The Sire-Dam model and the repeatability models could not be compared to the other models or each other by using LogL ratio test, as they are not truly nested in each other. However, the Sire-Dam model includes more pedigree information, which makes the estimations of the parameters closer to its true value, and can thereby be considered a better model than a sire model. Also when looking at the standard errors for the estimates, the Sire-Dam model has the lowest of all the sire models

(except for T^2), which is also an indicator of being a better fit model. The repeatability models are not consistent for standard errors, and cannot easily be compared using these.

Based on the AIC test, it is the REP_{time} that has the best fit of all models. The other models line up in a descending order as the fit decrease with lowered complexity. The Sire direct and Sire direct-random cage effects models have very close AIC values to each other, and can be considered within the boundary of equal fit.

The cross validation tells a different story than the AIC test. Looking at the average ρ values for the 6 models, the Sire-Dam model is the model that has the highest correlation between $P_{predicted}$ and $P_{corrected}$. This is a somewhat surprising result as one should believe that the more complex repeatability models would give more accurate breeding values, especially when correcting for time dependent social interactions and random cage effect. One possible reason for the lower ρ values of the repeatability models is that the trial was divided into too few time periods, so that there were not enough repeated survival records to capture all the variation that was available. Dividing the trial into more periods, like week periods or even day-periods might have given another result, as the AIC test gives a clue about the repeatability models being superior, but that is not within the scope of this thesis. If the Sire-Dam model is truly the best model for the prediction of breeding values, that could in fact be a good thing. A simpler model is easier to understand, handle and analyze in computer programs without the need of multiple records/observations, expanding the dataset and changing the presence or absence of cage-mates during time. Sometimes less can be more.

Further investigations

Besides expanding the repeatability models into week or day records, other breeding models could have been of interest for further investigation. This could e.g. be a random regression model, to see if there are differences in heritability between the periods the trial is divided into, or a bivariate model where survival time and survival (1/0) are the response variables.

Conclusion

To be able to eliminate the problem of feather pecking and cannibalistic behaviour in laying hens, the breeding model must be of sufficient complexity to capture all the heritable variance, giving accurate parameter estimates and breeding values. Including the effects of social interactions showed to be important, in addition to including as much pedigree information as possible. For this set of survival data, the Sire-Dam model including direct and

indirect genetic effects proved to be the model with the best fit, without excluding the possibility that an even more complex repeatability model in the end could give the most accurate breeding values.

List of abbreviations

AIC	= Akaike Information Criterion
BLUP	= Best Linear Unbiased Predictions
BLUE	= Best Linear Unbiased Estimations
DBV	= Direct Breeding Value
IBV	= Indirect (Social) Breeding Value
LogL	= Log Likelihood (Natural logarithm of the likelihood)
MME	= Mixed Model Equations
NA	= Missing value
REP	= Repeatability model
REP_{time}	= Repeatability model with time dependent social effect
SE	= Standard error
TBV	= Total Breeding Value
WB	= Name of the layer strain used in the analysis

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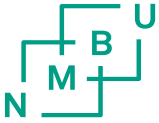
Attachments

Attachment 1: *Total number of layers, survival rate and mean survival time for in each house with their respective rows and levels.*

Housing	**Total**	**Alive**	**Dead**	**Survival rate**	**Mean survival time**
Laying house 1	3796	1971	1825	0,52	323.02
Row 1	464	251	213	0,54	323.57
Row 2	464	283	181	0,61	341.83
Row 3	428	245	183	0,57	342.11
Row 4	496	323	173	0,65	359.71
Row 5	488	236	252	0,48	315.59
Row 6	488	221	267	0,45	304.86
Row 7	480	205	275	0,43	297.86
Row 8	488	209	279	0,43	300.94
Level: Top	1256	609	647	0,48	310.67
Level: Middle	1232	655	577	0,53	325.74
Level: Bottom	1308	707	601	0,54	332.32
Laying house 2	3120	1767	1353	0,57	329.04
Row 1	404	279	125	0,69	350.72
Row 2	360	191	169	0,53	299.88
Row 3	400	240	160	0,60	329.09
Row 4	400	216	184	0,54	324.08
Row 5	320	170	150	0,53	326.58
Row 6	480	263	217	0,55	340.23
Row 7	360	181	179	0,50	321.83
Row 8	396	227	169	0,57	333.39
Level: Middle	1604	917	687	0,57	329.37
Level: Bottom	1516	850	666	0,56	328.70
Overall	6916	3738	3178	0,54	325.74



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