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CICLO XXI

ANALYSIS OF PIGLET SURVIVAL IN A DRY-CURED HAM-PRODUCING CROSSBRED LINE

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Riassunto

RIASSUNTO

Gli obiettivi del moderno allevamento suinicolo mirano principalmente a garantire buone performance in termini produttivi e costi di produzione contenuti. Il controllo della mortalità neonatale rappresenta una delle tematiche più attuali, proprio per gli importanti riflessi sul bilancio aziendale. Attualmente, il tasso di mortalità totale sino allo svezzamento può variare dal 18 al 20% sia per la realtà europea che statunitense. Il numero di suinetti annualmente partoriti da una scrofa è probabilmente considerato il carattere riproduttivo più importante, in grado di condizionare il management aziendale e spesso, per tali motivazioni, l'unico ad essere considerato nei programmi di miglioramento genetico di linee suine. In realtà, gli indubbi miglioramenti ottenuti relativamente a questo aspetto sono spesso vanificati da peggioramenti nella capacità di sopravvivenza del suinetto, sia al parto che durante la fase di allattamento.

I dati utilizzati in questa tesi sono stati raccolti presso il nucleo di selezione Gorzagri, sito a Riese Pio X (TV, Italia) e il centro genetico, sito a Todi (PG, Italia) e dedicato al programma di sib-testing della linea verri C21. L'attività del centro è finalizzata alla produzione di famiglie di suinetti ibridi, originati dall'accoppiamento di verri C21 e scrofe ibride Goland. Questi suinetti, di costituzione genetica identica all'ibrido Gorzagri allevato negli allevamenti commerciali, producono le informazioni necessarie per la stima del valore genetico e successiva selezione dei verri e scrofe della linea C21. La linea verri C21 è oggetto di attività di selezione secondo obiettivi finalizzati al miglioramento delle performances di allevamento e dell'attitudine alla produzione del prosciutto crudo stagionato DOP. Obiettivi selettivi sono il miglioramento dell'attitudine alla trasformazione industriale della carcassa e della coscia, particolarmente in relazione alla copertura di grasso della coscia e alla sua qualità (numero di iodio e acido linoleico) e alla presenza di difetti della stessa quali la globosità ed il grado di marezzatura della carne. Inoltre, lo schema selettivo mira all'ottenimento di animali omogenei in termini di accrescimento al fine di permettere un'ottimale organizzazione produttiva all'interno degli allevamenti.

Recentemente, anche la capacità di sopravvivenza e la robustness del suinetto sono divenuti oggetto di interesse selettivo. I dati di mortalità dei suinetti sino allo svezzamento sono stati raccolti sia presso il nucleo di selezione che presso il centro genetico di Todi a partire dal 2000. Nel primo contributo i dati di sopravvivenza dei suinetti fino allo svezzamento rilevati presso il centro genetico di Todi, sono stati analizzati utilizzando un modello dei rischi proporzionali e testando diverse baseline hazard function. Il tempo di sopravvivenza del suinetto, considerato come l'intervallo tra la nascita e lo svezzamento, è stato analizzato prendendo in esame gli effetti sistematici dovuti al sesso, messa a balia, mese-anno di nascita, ordine di parto della scrofa allattante, dimensione della nidiata e classi di indice genetico standardizzato dei verri. Quest'ultimo si è reso indispensabile per poter apprezzare le relazioni intercorrenti tra obiettivi di selezione della linea verri C21 attualmente perseguiti e sopravvivenza della progenie; nel modello sono stati inclusi anche gli effetti casuali della nidiata e la componente genetico additiva del verro. La mortalità in allattamento dei suinetti ibridi Goland si è attestata intorno al 14% con un tempo di morte medio pari a 6 giorni. L'effetto del sesso è risultato essere un fattore rilevante sulla mortalità in allattamento: le femmine hanno una probabilità di morire del 20% in meno rispetto alla classe di riferimento (maschi). Lo spostamento a balia ha anch'esso un effetto significativo sulla mortalità in allattamento. I soggetti trasferiti dalla madre "biologica" ad una balia presentano una probabilità di morire del 40% inferiore rispetto ai suinetti non spostati (classe di riferimento). Tali risultati confermano l'importanza di questa pratica manageriale, che porta ad una significativa riduzione della mortalità neonatale. Considerando il fattore "ordine di parto della balia", si è osservato come i suinetti allattati principalmente da balie primipare abbiano manifestato una probabilità di morire superiore rispetto ai suinetti allattati da pluripare. In particolare, si è potuta osservare una diminuzione del rischio di morte fino al quarto ordine di parto, per il quale il rischio di morte si attestava attorno al 75% di quello che caratterizza suinetti nati da scrofe di secondo ordine di parto (considerate in questo caso come riferimento).

Considerando la dimensione della nidiata, è stato possibile constatare che la probabilità di morire aumenta in modo significativo per i suinetti allattati in nidiate di dimensioni ridotte (inferiore a 5 suinetti) oppure molto numerose (superiore ai 15 suinetti) rispetto alla classe di riferimento che in questo caso è rappresentata da nidiate costituite da 9-11 suinetti. Nidiate molto numerose comportano una maggiore competizione tra i suinetti per la mammella (difficoltà ad assumere il colostro) e sovraffollamento con maggiore possibilità di schiacciamento. Per quanto riguarda le nidiate di dimensioni ridotte queste sono state osservate, generalmente, in scrofe che hanno avuto una gestazione non regolare.

L'effetto dell'anno e della stagione di nascita è risultato essere un effetto altamente significativo, imputabile a fattori di natura climatica, epidemiologica e manageriale esistenti tra il 2000 ed il 2006. Per quanto riguarda l'indice genetico standardizzato dei verri, da questo studio è emerso che i verri con un indice di merito genetico globale superiore rispetto alla media di popolazione, hanno generato progenie, nel corso degli anni oggetto di studio, con un rischio di morte pre-svezzamento superiore a quelli con indice inferiore rispetto alla media di popolazione.

Per quanto riguarda la stima della variabilità genetico additiva indotta dal verro e quella ambientale permanente indotta dalla nidiata sulla capacità di sopravvivenza del suinetto, è emerso che l'effetto della nidiata ha una rilevanza maggiore rispetto a quella del verro e mette in evidenza l'importanza dell'effetto ambientale esercitato dalla scrofa allattante. L'ereditabilità stimata per la sopravvivenza è risultata bassa (0.03), in accordo con stime di ereditabilità reperite in bibliografia. Tuttavia la variabilità genetica associata a questa caratteristica è risultata molto elevata e di grado tale da permettere interventi specifici di miglioramento dei verri appartenenti alla linea C21.

I risultati ottenuti nell'ambito del presente lavoro sottolineano che la selezione per la diminuzione della mortalità dei suinetti in allattamento può essere perseguita attraverso l'inclusione della capacità di sopravvivenza del suinetto negli obiettivi di selezione della linea C21.

Al fine di testare la possibilità di includere il carattere “sopravvivenza” tra gli obiettivi di selezione della linea verri C21, nel secondo contributo si è proceduto alla valutazione dell’importanza delle informazioni fenotipiche provenienti dal nucleo di selezione Gorzagri per la stima del valore riproduttivo della sopravvivenza alla nascita dei suini ibridi allevati presso il centro genetico. Obiettivo specifico di questo lavoro è stato quello di stimare le correlazioni genetiche tra sopravvivenza alla nascita dei suini puri e sopravvivenza alla nascita dei suini ibridi, originati dai medesimi verri della linea pura, in modo da testare la possibilità di introdurre metodologie selettive CCPS (combined crossbred and purebred selection). La stima della correlazione genetico additiva permette di quantificare, in parte, l’entità dell’interazione genotipo – ambiente degli animali allevati presso strutture differenti. Lo studio è stato condotto su 30,919 (3,162 nidiate) suini di linea pura e 13,643 (1,213 nidiate) suini ibridi. Gli animali di linea pura sono stati generati da 168 verri C21 e 1,413 scrofe C21 di linea pura. I suini ibridi, allevati presso il centro genetico di Todi, sono stati generati impiegando gli stessi 168 verri e 319 scrofe ibride di derivazione Large-White. Il carattere analizzato è stato la sopravvivenza alla nascita come carattere categorico (vivo o morto). Per la stima dei parametri genetici è stato fittato un modello statistico bivariato a soglie (threshold model) utilizzando un approccio Bayesiano. Il modello statistico ha considerato l’effetto del sesso, dell’ordine di parto della scrofa, della dimensione della nidiata e l’anno mese di nascita dei suinetti. Per la stima dei parametri del modello sono state adottate, per gli effetti “fissi”, delle flat priors, mentre per l’effetto della nidiata, della scrofa e la componente genetico additiva del verro, sono state adottate delle Gaussian prior distributions. La media della distribuzione marginale a posteriori della componente di varianza del verro, della scrofa e della nidiata nei suini puri è risultata pari a 0.018 (0.008), 0.077 (0.020), 0.347 (0.025), rispettivamente. Per quanto riguarda le stime delle componenti di varianza nei suini meticci, le stime sono risultate pari a 0.030 (0.018), 0.120 (0.034), and 0.189 (0.032), rispettivamente per la componete del verro, della scrofa e della nidiata. L’ereditabilità per sopravvivenza alla nascita nei suini puri e risultata pari a 0.049

(0.023) mentre quella dei meticci 0.091 (0.054). Per quanto riguarda la correlazione genetica tra questi due caratteri (sopravvivenza in soggetti puri e meticci) è risultata pari a 0.248 (0.336). Tuttavia, l'ampia variabilità di stima (95% Bayesian confidence region: -0.406 - 0.821) suggerisce che il progresso genetico atteso, quando la selezione è basata solamente soggetti puri, potrebbe essere nullo.

Nel terzo contributo sono state messe a confronto diverse metodologie per la stima del valore riproduttivo della sopravvivenza del suinetto durante la fase di allattamento. Il modello dei rischi proporzionali, assumendo due differenti baseline hazard function (Cox e Weibull), è stato comparato con un thershold e un sequential threshold model, in termini di capacità predittiva del modello (predictive ability) e di goodness of fit. Le stime di ereditabilità sono risultate basse per tutti e quattro i modelli e variabili tra 0.04 e 0.06. Inoltre le stime dei valori riproduttivi dei verri non hanno provocato sostanziali re-ranking dei riproduttori. Tuttavia in termini di capacità predittiva dei modelli, il sequential threshold model ha manifestato le migliori performance e proprio per questo motivo e per la sua facile interpretazione, potrebbe essere proposto come sistema di valutazione genetica da implementare all'interno del programma di selezione della linea verri C21.

Summary

SUMMARY

Piglet death during the perinatal and lactation periods is one of the most detectable causes of reduced production efficiency in swine herds. and it has also been identified as an important welfare issue. Piglet mortality within the first three days of life is still a problem in intensive farms. Mortality rates vary between 10 and 20% depending on the housing system. Data used in this study were collected in the nucleus and sib testing program of the C21 Large White boar line (Gorzagri, Fonzaso, Italy) from 2000 to 2006. In the selection nucleus farm (Riese Pio X, Italy) the pure C21 boars are produced and mated to pure C21 sows, while in a sib testing program farm (Todi, Italy), the same C21 boars are mated to crossbred sows to produce crossbred piglets. The general aim of this thesis was to explore the genetic aspects of piglet survival in the aforementioned dry-cured ham-producing crossbred line. In chapter 2 the piglet pre-weaning survival and its relationship with a total merit index (TMI) used for selection of Large White terminal boars for dry-cured ham production was investigated. Piglet pre-weaning survival was analyzed under a frailty proportional hazards model, assuming different baseline hazard functions and including sire and nursed litter as random effects. Estimated hazard ratios (HR) indicated that sex, cross-fostering, year-month of birth, parity of the nurse sow, size of the nursed litter and class of TMI were significant effects for piglet pre-weaning survival. Female piglets had less risk of dying than males (HR = 0.81) as well as cross-fostered piglets (HR = 0.60). Survival increased when piglets were nursed by sows of third (HR = 0.85), fourth (HR = 0.76) and fifth (HR = 0.79) parity. Piglets of small (HR = 3.90) or very large litters (HR > 1.60) had less chances of surviving in comparison with litters of intermediate size. Class of TMI exhibited an unfavorable relationship with survival (HR = 1.20 for the TMI top class). The modal estimates of sire variance under different baseline hazard functions were 0.06 whereas the variance for the nursed litter was close to 0.7. The estimate of the nursed litter effect variance was higher than the sire, underlying the importance of the common environmental generated by the nurse sow. The relationships between sire ranking obtained from different survival models highly agreed each others. The heritability estimate in equivalent scale was low (0.03).

Nevertheless, the exploitable genetic variation for this trait justifies the inclusion of piglet pre-weaning survival in the current breeding program for selection of Large White terminal boars for dry-cured ham-production.

In order to evaluate the opportunity of including the survival trait in such breeding programme, the relevance of purebred information for predicting genetic merit of survival at birth of crossbred piglets was assessed (chapter 3). A question is whether purebred performance (in the nucleus) predicts accurately outcomes in crossbreds (commercial tier). This was investigated by considering the two performances as different traits in a model and by estimating the genetic correlation. The objective of chapter 3 was to infer (co)variance components for farrowing survival in purebred (P) and crossbred (C) pigs; the latter were from crosses between P boars and Large White-derived crossbred sows. If the genetic correlation between C and P traits is large enough, selection in P would produce a correlated response in C. Data were from 13,643 (1,213 litters) C and 30,919 (3,162 litters) P pigs, produced by mating the same 168 P boars to 319 Large White-derived crossbred females and 1,413 P sows, respectively. The outcome variable was pig survival at birth as a binary trait. A Bayesian bivariate threshold model was implemented via Gibbs sampling. Effects of sex, parity of the dam, litter size and year-month of birth were assigned flat priors; those of litters, dams and sires were given Gaussian prior distributions. Marginal posterior means (SD) of the sire, dam and litter variances in P were 0.018 (0.008), 0.077 (0.020), 0.347 (0.025), respectively in the liability scale. For C, corresponding estimates were 0.030 (0.018), 0.120 (0.034), and 0.189 (0.032), respectively. The posterior means (SD) of heritability of survival in P and C, and of the genetic correlation between these traits were 0.049 (0.023), 0.091 (0.054) and 0.248 (0.336), respectively. Heritability estimates were low and in agreement with previous reports. The genetic correlation was also low, and a 95% Bayesian confidence region (-0.406, 0.821) included zero. Even though variation of estimates is large, results suggest that genetic progress expected in C when selection is based on P may be nil.

In chapter 4 different methodologies (proportional hazard, threshold and sequential threshold model) for predicting genetic merit of piglet survival were compared in terms of predictive ability and

goodness of fit. Data structure was the same used in chapter 3. A frailty proportional hazard model, assuming two different baseline hazard function (Cox and Weibull) and including sire and nursed litter as a random effects were fitted. The threshold and sequential threshold model considered the same effects. Model fitting was evaluated in terms of goodness-of-fit and predictive ability, using the mean square error as reference parameters. Estimated sire variances for piglet pre-weaning mortality were low, and heritability ranged from 0.04 to 0.06. All four models led to similar ranking for sires, with strong correlation between methods. The sequential threshold model had a better performance for predicting piglet survival but it had a lower performance in terms of goodness-of-fit than Cox model. Results from this study suggest that sequential threshold model may, globally, be better than other methods tested, both for its better predictive ability of piglet survival in genetic evaluations and for its easier interpretation. Further, sequential threshold model is computationally less demanding and can be extended to allow for different variance components by different period from birth to weaning.

Chapter 1

General introducion

GENERAL INTRODUCTION

The number of piglets alive at birth and at weaning represent the two major sources of variation in the profitability of the swine industry (Legault, 1983; Tess et al., 1983). According to a survey of commercial farms in the U.S., the average number of piglets born per sow is 10.9. In each litter, an average of 8.3% piglets are stillborn, and of those piglets born alive, 11% die before weaning (USDA, 2002). This scenario seems to be similar to that reported by C.R.P.A (2004) for the Italian situation. The National Swine Improvement Federation estimates the value of each additional piglet alive at birth and weaning to be $13.50 and $6.00, respectively (NSIF, 1996). Even tough in absolute terms a weaned piglet is more valuable than a newborn, the expenses of rearing an extra piglet to weaning subtract from the initial value result in a lower marginal profit at weaning. Genetic and management strategies that increase the number of live piglets at these two critical periods would therefore be of great value to the swine industry.

Biological aspects of piglet survival

The birth process is the first stressful event for the piglet. On average, between 3-8% of the total number of piglets are delivered stillborn (Spiecer et al., 1986; Daza et al., 1999; Marchant et al., 2000). The major cause of stillbirth is asphyxiation (Randall and Penny, 1967; Randall, 1971). Asphyxiation may be induced by decreased placental blood flow associated with uterus contractions, damage or breakage of the umbilical cord, or premature detachment of the placenta (Curtis, 1974; English and Morrison, 1984). Piglets born in the late stages of farrowing have an increased risk to suffer from asphyxia, because of cumulative effects of successive uterus contractions, or higher risks of premature rupture of the umbilical cord or detachment of the placenta (Randall, 1972; English and Wilkinson, 1982).

The percentage of live-born piglets that die until weaning varies considerably, ranging from 5 to 30% (Bille et al., 1974; Daza et al., 1999). On average, between 50-70% of these pre-weaning losses occur within the first three day after birth (Fahmy and Bernard, 1971; Blasco et al., 1995; Marchant et al., 2000). Major causes of pre-weaning mortality are starvation and overlying by the sow.

Together, these causes constitute about 75% of the total mortality during the first days of life (Dyck and Swierstra, 1987). Other mortality causes are congenital abnormalities, savaging by the sow, and diarrhea. Infectious diseases as a primary cause of death play a minor role in mortality until weaning, accounting for approximately 5% of deaths (Vaillancourt and Tubbs, 1992).

Genetic aspects of piglet survival

Genetic effects on piglet survival can be expected from the biological mother (through uterine effects), the nurse sow (through differences in mothering ability), the piglet itself (through differences in adaptive behavior and differences in body reserves etc). To investigate the latter animal effect, observations on an individual piglet basis must be available. If observations are available on a litter basis the service sire, the father of the piglets, becomes important instead of the piglet itself. Most previous studies treated piglet survival as a maternal trait. However the piglet's genotype could also influence its survival (Van Arendonk et al., 1996; Arango et al., 2005). It is well known that the causes of stillbirth and of pre-weaning mortality are different, which suggests that the genetic backgrounds for piglet survival in different periods are not the same. In addition, several previous studies (e.g. Kerr and Cameron, 1995; Roehe and Kalm, 2000) have shown that the piglet's individual birth weight is the most important factor affecting pre-weaning mortality. This, opens the possibility of improving survival rate by selecting indirectly for high birth weigh. This is in disagreement with studies carried out by Leenhouwers (2001) because it was found that selection for increased birth weight would actually somewhat decrease piglet survival. Biological studies using piglets with different genetic merits for piglet survival (Leenhouwers, 2001) strongly suggest that selection for improved survival will increase the degree of maturity of the piglets at birth (Leenhouwers et al., 2002) rather than affect the progress of parturition or early neaonatal piglet behavior leading to earlier postpartum ingestion of colostrum (Leenhouwers et al., 2001). Leenhouwers et al. (2002) compared late foetal development in piglets with low and high genetically determined ability to survive from onset of parturition until weaning. This study indicated that selection for piglet survival will increase the proportional masses (g kg^{-1} body weight) of liver, small intestine, stomach and adrenals and enhance the maturation of the

hypothalamus-pituitary-adrenal (HPA) axis (and thus the glucocorticosteroid-dependent late foetal adaptive maturation of various organs, e. g. gastrointestinal tract and lungs). Furthermore, selection for piglet survival will increase liver and muscle glycogen concentrations, total amount of liver glycogen and body fat percentage, thus improving the thermoregulatory capacity of the newborn piglet (Leenhouwers et al., 2002).

Farrowing survival. For stillbirth (or its complement farrowing survival) the piglet and the biological mother effects could be of influence. The additive genetic effect of the dam includes both a direct effect of the dam's genes transmitted to the piglets on the survivability of the offspring and also a pure maternal genetic effect which is related to aspects of the dam that are relevant for the chance of surviving of the piglets and are influenced by the additive effects of the dam's genes (e.g., the uterine influence of the dam on piglet mortality at birth and an influence during the expulsion phase of the birth process). The sire effect accounts only for differences of piglet survival that are caused by the additive effects of genes that the piglets inherit from the sire. When included in a model along with additive genetic effects of the sire and of the dam, litter effects are expected to account for influences, common to all piglets joining the same litter and causing variation across litters, due to a strictly-environmental component and to non-additive gene effects shared by members of a full-sibs family. Heritability estimates, for stillbirth, are generally low ranging, from 0 to 0.10 (Siewerdt and Cardellino, 1996; Haneberg et al., 2001). Johnson et al. (1999) reported heritability estimates equal to 0.17 whereas Grandison et al. (2000) reported heritability estimates equal to 0.23 and 0.27 (obtained using linear or threshold model respectively). It is well known that the use of linear models with categorical data ignores their non-linear distribution and tends to produce underestimates of heritability (Gianola, 1982).

Preweaning survival. As reviewed by many authors (Knol et al., 2000; Leenhouwers, 2001; Arango et al., 2005) a way to improve piglet survival through weaning is selecting against preweaning mortality (PWM); however, individual preweaning mortality is not routinely recorded by producers. Furthermore, low heritability, categorical nature of the trait and cross-fostering impose additional

challenges to model PWM variation and to predict breeding values. Threshold models account for the non-linear nature of categorical traits and yield larger estimates of heritability for categorical traits than most common and widely used linear models (i.e., Gianola, 1982).

However, most research reports of piglet mortality (or its complement trait piglet survival) have used linear models assuming a continuously distributed trait; thus ignoring its categorical nature (van Arendonk et al., 1996; Knol et al., 2002; Serenius et al., 2003). Few studies have addressed variance component estimation of piglet mortality using threshold models (Roehe and Kalm, 2000; Grandinson et al., 2002; Arango et al., 2005), and only one had modeled the trait at the individual piglet level (Grandinson et al., 2002) but ignored maternal components. Roehe and Kalm (2000) estimated variance components with a generalized mixed model approach, and obtained heritability estimates for preweaning mortality using only the sire components. Separating the genetic components associated with preweaning piglet mortality is complex under field conditions due to the common management practice of cross-fostering, and the consequently combined effects of biological and nurse dams and their respective litters.

Scheme selection of the C21 boar line

Data used in this thesis were collected from year 2000 to 2006 in two locations: a selection nucleus farm (Riese Pio X, Italy) where pure C21 boars are produced and mated to pure C21 sows, and in a sib testing program farm (Todi, Italy), where the same C21 boars are mated to crossbred sows to produce crossbred piglets. Boars from the C21 line are used in commercial farms as sires of crossbred pigs which are fattened and slaughtered at heavy body weights (165 kg) for production of dry-cured hams. In the sib testing program of the C21 line, crossbred paternal half sib families are produced by mating C21 nucleus boars to a group of crossbred sows which is submitted to minimum intensity replacement policies. Crossbred sows originated from a cross involving boars of a synthetic line, derived from Large White and Pietrain breeds, and sows of a Large White line selected for maternal ability and prolificacy. Crossbred paternal half sib families provide the genetic evaluation program of C21 purebred breeding candidates with crossbred half sibs phenotypes for quality traits of raw and dry

cured hams. Besides growth and residual feed efficiency, the breeding goal of the C21 line includes traits related to the quality of dry-cured ham. Selection is addressed to an intermediate optimum for marbling and for the amount of subcutaneous fat evaluated on the raw ham, to enhance the quality of fat covering, to reduce excessive ham roundness, and to reduce curing weight losses at a fixed level of dry-cured ham quality.

Objective of this Thesis

The main objective of this thesis is to gain knowledge in the genetic aspects of piglet survival in a dry-cured ham-producing crossbred line and, in detail:

I. to investigate sources of variation of piglet pre-weaning survival in a crossbred slaughter pigs population, the relationship of crossbred piglet survival with a total merit index (TMI) used for selection of breeding candidates in a Large White boar line and to estimate variance components and genetic parameters through survival analysis techniques;
II. to investigate sources of variation of piglet survival at birth and to infer genetic parameters including the genetic correlation between survival at birth of purebred and crossbred piglets originated by the same sires;
III. to infer parameters of piglet pre-weaning survival with the survival analysis, threshold model and sequential threshold model in a crossbred slaughter pigs populations and then to asses their relative predictive abilities and goodness of fit.

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Chapter 2

Piglet pre-weaning survival

Survival analysis of pre-weaning piglet survival in a dry-cured ham-producing crossbred line[1]

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ABSTRACT

The aim of this study was to investigate piglet pre-weaning survival and its relationship with a total merit index (TMI) used for selection of Large White terminal boars for dry cured ham production. Data on 13,924 crossbred piglets (1,347 litters), originated by 189 Large White boars and 328 Large White-derived crossbred sow, were analyzed under a frailty proportional hazards model, assuming different baseline hazard functions and including sire and nursed litter as random effects. Estimated hazard ratios (HR) indicated that sex, cross-fostering, year-month of birth, parity of the nurse sow, size of the nursed litter and class of TMI were significant effects for piglet pre-weaning survival. Female piglets had less risk of dying than males (HR = 0.81) as well as cross-fostered piglets (HR = 0.60). Survival increased when piglets were nursed by sows of third (HR = 0.85), fourth (HR = 0.76) and fifth (HR = 0.79) parity in comparison with first and second parity sows. Piglets of small (HR = 3.90) or very large litters (HR > 1.60) had less chances of surviving in comparison with litters of intermediate size. Class of TMI exhibited an unfavourable relationship with survival (HR = 1.20 for the TMI top class). The modal estimates of sire variance under different baseline hazard functions were 0.06 whereas the variance for the nursed litter was close to 0.7. The estimate of the nursed litter effect variance was higher than that of the sire and this shows the importance of the common environmental generated by the nurse sow. The relationships between sire ranking obtained from different survival models highly agree each others. The heritability estimate in equivalent scale was low and reached a value of 0.03. Nevertheless, the exploitable genetic variation for this trait justifies the inclusion of piglet pre-weaning survival in the current breeding programme for selection of Large White terminal boars for dry cured ham production.

Key words: dry-cured hams, heritability, pre-weaning mortality, piglet, survival analysis

INTRODUCTION

On average 12% of newborn piglets die before weaning and half of these losses occurs during the first three days of life (Blasco et al., 1995). Enhancing piglet survival is relevant both for ethical

and economical implications and for the acceptability of the production system by the consumer. Additive genetic variation of piglet pre-weaning survival is large enough to be exploited in specific breeding programs, but selection for improvement of direct survival is difficult due to the binary nature of the trait and large environmental variance (Knol et al., 2002).

The threshold model is the method of choice for the analysis of binary traits (Gianola, 1982) and can account for the categorical nature of survival, but it suffers from a severe loss of information because piglets dying at d 1 or in wk 2 after birth are treated alike (Casellas et al., 2004). The analysis of failure time makes use of all available information and does not restrict observations to an arbitrarily defined point (Ducrocq, 1997). The availability of survival analysis techniques offers the opportunity of new approaches to the investigation of pre-weaning piglet survival (Ducrocq et al., 1988).

The relationships of piglet survival with production traits have been investigated in lines selected for increased efficiency of lean meat production. In Italy, the breeding goal for boar lines originating slaughter pigs for dry-cured ham production differs greatly from the one pursued to enhance efficiency of pork production and the relationship between the breeding goal and piglet survival is currently unknown.

The aims of this study were to investigate sources of variation of piglet pre-weaning survival in a crossbred slaughter pigs population, the relationship of crossbred piglet survival with a total merit index (TMI) used for selection of breeding candidates in a Large White boar line and to estimate variance components and genetic parameters through survival analysis techniques.

MATERIAL AND METHODS

Animals and Data

Data used in this study were collected in the sib testing program of the C21 Large White boar line (Gorzagri, Fonzaso, Italy) from 2000 to 2006. Boars from the C21 line are used in commercial farms as sires of crossbred pigs which are fattened and slaughtered at heavy body weights (165 kg)

for production of dry-cured hams. In the sib testing program of the C21 line, crossbred paternal half sib families are produced by mating C21 nucleus boars to a group of crossbred sows which is submitted to minimum intensity replacement policies. Crossbred sows originated from a cross involving boars of a synthetic line, derived from Large White and Pietrain breeds, and sows of a Large White line selected for maternal ability and prolificacy. Crossbred paternal half sib families provide the genetic evaluation program of C21 purebred breeding candidates with crossbred half sibs phenotypes for quality traits of raw and dry cured hams. Besides growth and residual feed efficiency, the breeding goal of the C21 line includes traits related to the quality of dry-cured ham. Selection is addressed to an intermediate optimum for marbling and for the amount of subcutaneous fat evaluated on the raw ham, to enhance the quality of fat covering, to reduce excessive ham roundness, and to reduce curing weight losses at a fixed level of dry-cured ham quality.

Data on survival of piglets at birth and up to weaning were routinely collected in the sib testing program since 2000 and included birth litter description (sow identification and parity, sire, date of farrowing, and size of the litter at birth), and individual piglet information (identification, sex, stillborn or alive at birth, weaning date or date of death if the piglet died during the suckling period, and date of transfer and foster dam identification for cross-fostered piglets). Cross-fostering occurred for 46% of piglets and was of similar proportion for male and female piglets. For piglets which died before weaning, survival time was computed as the difference between the date of death and date of birth whereas for piglets still alive at weaning survival time was computed as the difference between the date of weaning and the date of birth, but all these records were considered censored records.

After application of editing procedures, which aimed to discard records with incomplete or inconsistent information (120 piglets) and with unknown sire (105 piglets), a total of 13,924 individual survival records of piglets (1,347 litters) sired by 189 C21 boars mated to 328 crossbred sows were available for the study.

Survival Analysis

The individual piglet survival time was analyzed using survival analysis methodology. Preliminarily, the survivor function for the general population was estimated by the Kaplan-Meier method (Kaplan and Meier, 1958). The linear regression of $\ln\{-\ln[S(t)]\}$ on $\ln(t)$, where $S(t)$ is the Kaplan-Meier estimated survivor function, was considered to check the suitability of the assumption of a Weibull baseline hazard (Ducrocq et al., 1988). Because the relation between $\ln\{-\ln[S(t)]\}$ and $\ln(t)$ was not linear, the assumption of a Weibull distribution function for the baseline hazard was not suitable for these data and alternative models were considered. All these models were from the group of proportional hazard frailty models (Cox, 1972) and of the general form:

$$h(t) = h_0(t) \exp\{\mathbf{x}_1'\boldsymbol{\beta}_1 + \mathbf{x}_2'(t)\boldsymbol{\beta}_2 + \mathbf{z}'\mathbf{u} + \mathbf{w}'(t)\mathbf{q}\}$$

where $h(t)$ is the hazard of death at time t (age of piglets), $h_0(t)$ is the baseline hazard function, $\boldsymbol{\beta}_1$ is an unknown vector of fixed regression coefficients for a set of nongenetic time-independent effects, $\boldsymbol{\beta}_2$ is an unknown vector of fixed regression coefficients for a set of nongenetic time-dependent effects, $\mathbf{x}_1$ is a vector of indicator variables for nongenetic time-independent effects, $\mathbf{x}_2(t)$ is a vector of indicator variables for nongenetic time-dependent effects, $\mathbf{u}$ is an unknown vector of regression coefficients for random effects due to sires, $\mathbf{z}$ is a vector of indicator variables for sire effects, $\mathbf{q}$ is an unknown vector of regression coefficients for time-dependent random effects due to the nursed litter, and $\mathbf{x}_2(t)$ is a vector of indicator variables for time-dependent nursed litter effects.

Three different models were considered. For the first model (model COX), the distribution function for the baseline $h_0(t)$ was left completely unspecified (Cox, 1972) and a semi-parametric

proportional hazard model was used. The second model (model WTD) was a parametric model where a Weibull distribution was assumed as a baseline distribution function after the inclusion of a time-dependent covariate, i.e., a Weibull time-dependent function was used as baseline function for this model (Tarres et al.,2005; Casellas et al., 2006).

An additional model (model GDM) was considered because it is well adapted for analyses of timing of events occurring in short periods of time and with high incidence of ties in timing of occurrence, which is a common situation in analysis of piglet pre-weaning survival (Casellas et al., 2004). This model, which is based on Prentice and Gloeckler (1978) and is a grouped data version of the proportional hazard model, does not make any assumption about the baseline distribution function, but it can be viewed as a fully parametric model that include a time-dependent covariate that changes its value at each day of the observed time space (Ducrocq, 1999).

Model Selection for Fixed Effects

Prior to survival analysis, TMI of the sire of the piglet and the size of the nursed litter, which were considered in all models as potential effects influencing the hazard of death of piglets, were categorized (Table 1). Because the form of the relationship between these variables and the hazard was unknown, this ensured that no assumption had to be made about the form of that relationship. Before categorization, TMI of sires were standardized to a mean of zero and standard deviation of one.

For all models, effects included in $\boldsymbol{\beta}_1$ were the effects of cross-fostering (yes or not), sex (male or female), year-month of birth (72 monthly classes from July 2000 to July 2006) and class of standardized TMI of the sire of the piglet (class 1: TMI < -1 SD; class 2: -1 SD ≤ TMI ≤ 1 SD; class 3: TMI > 1 SD; the number of sires for piglets in class 1, 2, and 3 was 25, 132, and 32, respectively). The parity of the nurse sow (parity 1, 2, 3, 4, 5, 6, and 7 or more) and the class for the size of the nursed litter (class 1: ≤ 5 piglets, class 2: from 6 to 8, class 3: from 9 to 11, class 4: from 12 to 14, class 5: ≥ 15 piglets) were effects included in $\boldsymbol{\beta}_2$ as time-dependent covariates that could

change value in the time space as a consequence of cross-fostering, carried out to homogenize size of litters, and of piglet mortality. Besides these effects, model WTD included also a time-dependent covariates changing value at d 6 and 12 for all piglets. Cut points at 6 and 12 d of age of piglets were identified with a spline regression of the log of the Kaplan-Meier survival function on time following the same approach used by Tarrés et al. (2005). To do that, all possible combinations of 1, 2, …, *k* cut points in the time space (pre-weaning period) were explored, avoiding combinations of adjacent days.

A preliminary analysis was carried out to identify fixed effects that were statistically significant at $P < 0.05$. Before ultimate rejection of effects which were not significant, each of them was tested with the group of fixed effects initially significant to determine whether any became significant. To test the proportional hazards assumption, time-dependent factors, i.e., interaction terms between the time-independent effects and function of time (changing at 6 and 12 d), were defined. The inclusion of these interaction terms did not significantly increase ($P > 0.05$) the likelihood for any of the models analyzed (results not shown in tables), and, thus, the proportionality hypothesis was not rejected.

Random Effects and Heritability

Random effects included in all models were the effects of the sire of the piglet and of the nursed litter which was treated as a time-dependent effect that, after the first day of life, could change value as a consequence of cross-fostering. Sire additive genetic effects, under polygenic inheritance, were assumed to follow a multivariate normal distribution: $\mathbf{u} \sim MVN(\mathbf{0}, \mathbf{A}\sigma_u^2)$, where **A** is the additive genetic relationship matrix among sires and σ_u^2 is a variance component for sire effects. The effects of the nursed litter in **q** were assumed to be log-gamma distributed following a single parameter γ, from which the variance of the nursed litter effect σ_q^2 can be derived. Normal priors for σ_u^2 and log-gamma priors for σ_q^2 were combined with the likelihood function of the data

to obtain an expression proportional to the joint posterior density of all parameters (Ducrocq and Casella, 1996) and estimates of variance components were obtained by Laplacian approximation of the marginal posterior densities.

Effective and equivalent heritabilities (Yazdi et al., 2002) of piglet pre-weaning survival were obtained as :

$$h^2 = \frac{4\sigma_u^2}{1+\sigma_u^2+\sigma_q^2}$$

and

$$h_{eq}^2 = \frac{4\sigma_u^2}{\sigma_u^2+\sigma_q^2+\frac{1}{p}}$$

where h^2 and h_{eq}^2 are effective and equivalent heritability, respectively, σ_u^2 and σ_q^2 are variance components for sire and nursed litter effects (calculated as σ_q^2 = trigamma(γ) where trigamma(.) is the trigamma function), respectively, and *p* is the average proportion of uncensored records. All analyses were carried out using the "Survival Kit" software, version 3.12 (Ducrocq and Sölkner, 1994).

RESULTS AND DISCUSSION

Survival and Hazard Function

The survival and hazard functions estimated by the Kaplan-Meier method are presented in Figure 1 and 2, respectively. Eighty-six percent of records were censored (animals still alive) at the end of the weaning period which occurred at an average time of 28 d. Hence, piglet mortality from birth to weaning was 14% and average failure time for uncensored records (death of piglets) was 6 d. The survival experience was not constant over the pre-weaning period. The estimated hazard function (Figure 2) indicates that the hazard of death for piglets progressively decreased from birth to d 14 and was much higher in the first week after birth than afterwards. As a consequence, nearly

70% of overall mortality (Figure 1) occurred during the first week of life. From d 14 onwards, the hazard remained unchanged. These results imply that each litter lost, on average, one piglet from birth to weaning, giving rise to important economic losses and ethical considerations. Svendsen and Bengtsson (1982) reported that a fraction ranging from 10 to 35% of newborn piglets may die within the early three weeks of age. Moreover, over 50% of deaths occur in the first three days after birth (Dyck and Swierstra, 1987) with crushing accounting for 70 to 80% of deaths (English and Morrison, 1984). Most causes of death are due to interactions between the piglet and its environment (Le Dividich and Herpin, 1994). Also, low immune-competence at birth may play a role by increasing susceptibility to pathogens and leading to death in lactation (Xu et al., 2000).

Nongenetic Effects

Results of likelihood ratio tests (LRT), obtained with COX, for statistical significance of fixed effects are summarized in Table 2. All analyzed factors were significantly related to the risk of mortality when they were entered sequentially in the model or were excluded from the full model one at a time giving similar results and showing very little redundancy when explaining variation of the investigated trait. Parity of the nurse sow ($P < 0.01$) and TMI ($P < 0.05$) had a lower impact on pre-weaning survival than the one of other effects. The year-month of birth had a marked influence on the hazard function ($P < 0.001$) as well as cross-fostering ($P < 0.001$), sex ($P < 0.001$) and size of the nursed litter ($P < 0.001$). These results are in agreement with what was expected intuitively from percentages of uncensored records for levels of fixed effects reported in Table 1.

Estimated hazard ratios (HR) for fixed effects included in models are presented in Table 3. Estimated HR were similar for different models with the exception of HR estimates for the size of the nursed litter. For litters ranging from 12 to 14 piglets or with more than 14 piglets, the estimated HR were greater when obtained with WTD than when estimated by COX and GDM (1.78 vs 1.40 for litters from 12 to 14 piglets; 2.98 vs 1.60 for litters with more than 14 piglets).

The sex of the piglet was a relevant effect for survival: female piglets (HR = 0.81) had 19% lower risk of dying than male piglets. Several authors have reported that female piglets have a greater survival advantage than males (Svendsen et al., 1986, Becker, 1995, Knol et al., 2002). Becker (1995) reported that the increased mortality in males was due to more males being crushed and to chilling. Although the underlying mechanism responsible for this sexual dimorphism in pre-weaning mortality rates has not been elucidated, there are differences across sexes that may provide biological explanations to these observations. A greater basal concentration of cortisol observed in male piglets in comparison with female piglets (Ruis et al., 1997) may cause male piglets to be more susceptible to detrimental stress effects and succumb to subsequent disease challenges.

Cross-fostering exerted favourable effects on survival chances of piglets. Fostered piglets had 40% greater probability of survival than piglets raised by the biological mother. This is in agreement with results obtained by Knol et al. (2002) and Leenhouwers et al. (2001). Cross-fostering of piglets was performed to reduce variation in size of nursed litters and occurred for 46% of piglets. Cross-fostering is one of the most effective methods to increase postnatal survival. This practice enhances the survival probability of small piglets because they have to compete less to reach the last available teat (English et al., 1982) in litters of average size than in large or very large litters. Moreover, cross-fostered piglets might introduce a disease in their new litters and might be at lower risk than their new littermates because of immunity due to colostrum suckled from the biological mother. This phenomenon decreases the average survival of piglets that are not cross-fostered (Knol, 2001). Since 70% of deaths occur during the first week of life, cross-fostering should be performed as soon as possible as reported in previous studies (Svendsen et al., 1986; Straw et al., 1998). A further biological explanation of these effects is related to the increase of piglet weights uniformity within a nursed litter caused by cross-fostering and to possible association between within-litter variation of piglet weights and pre-weaning losses (English et al.,

1982; Roehe and Kalm, 2000; Marchant et al., 2001; Milligan et al., 2002a,b). However, Milligan et al. (2001) reported that their data provided little support for the hypothesis that high birth weight variation resulted in decreased survival. Leenhouwers et al. (1999) did not find any relation at the phenotypic level between the within-litter standard deviation of birth weight and the proportion of stillborn piglets.

In this study, the group of sows originating crossbred piglets was submitted to minimum replacement policies and culling occurred mostly for reduced fertility due to aging or occurrence of severe disease. As a consequence, the chance of repeated farrowing was not influenced by selection of sows based on reproductive performance. The survivability increased when piglets were nursed by sows of third, fourth, and fifth parity in comparison with first- and second-parity sows. However, the hazard of dying for piglets nursed by sow of sixth, seventh or greater parity did not differ ($P > 0.05$) from that of animals nursed by first- and second-parity sows. These results are consistent with those of a number of studies (Leenhouwers et al. 2001, Knol et al., 2002; Damgaard et al., 2003; Grandison et al., 2005; Arango et al., 2005). Conversely, Weary et al. (1998) reported a higher probability of crushing for greater parities. The authors justified this result because litters originated by sows of higher parity tended to exhibit lower average weight gains from d 1 to 3 after birth, and because older sows were heavier and clumsier. However, their results did not allow clear conclusions about the causes of crushing because several potential predisposing factors (low early weight gains, high sow parity number, larger litter size, and low birth weight) were closely related. Another important aspect related to piglet pre-weaning mortality is colostrum production of sows. After birth, the piglet is fed initially with colostrum and after with milk, which exhibit high fat and low carbohydrate contents, implying that the intestine must be functional at birth and the piglet rapidly able to synthesize glucose by gluconeogenesis to supply its glucose-dependent tissues and to oxidize fats. In this context, ingestion of colostrum, which provides both energy and maternal antibodies protecting the piglets until their immune system matures, is of the utmost importance for

survival. There are several sow-related factors, including health, premature farrowing, changes in reproductive hormones and metabolism, parity, nutrition and genetics, that might be involved in colostrum production (Le Dividich et al., 2005). There is no clear evidence of parity effects on colostrum production by the sow (Le Dividich et al., 2005). Inoue et al. (1980) and Klobasa et al. (1986) reported that first parity sows have lower colostrum IgG concentrations than multiparous sow.

Consistently with a number of studies (Kerr and Cameron, 1995; Knol et al., 2002; Grandison et al., 2005), the probability of survival decreased for piglets joining small (less than 6 piglets), large (from 12 to 14 piglets) or very large (more than 14 piglets) litters in comparison with litters of intermediate size (from 6 to 11 piglets). Because of occurrence of cross-fostering, a few litters were classified as litters of small size and were likely to be those of sows with physiological inabilities or difficulties to have a normal gestation. For large or very large litters, increased risk of pre-weaning mortality might have been caused by excessive crowding and reduced milk availability for piglets of limited competing ability for suckling. Large litters and wide ranges of birth-weight variation are claimed to cause decreases of piglet survival because of competitive exclusion of light littermates from access to productive teats (English and Morrison, 1984).

The year-month of birth had a marked influence on piglet survival. Piglet pre-weaning mortality changed across years and across months of the same year as a consequence of changes in the hazard due to several sources of variation such as climate, epidemiologic and management effects. The magnitude of the estimated HR for year-month of birth effects (data not presented) changed erratically across year-month classes and did not exhibit a consistent trend over time. Roehe and Kalm (2000) analyzed pre-weaning mortality in piglets and found that year-season was the most important fixed effect for pre-weaning mortality.

For model WTD, the instantaneous mortality rate was more pronounced during the first week of life, diminished from d 6 to d 12 (HR = 0.21), and was very low from d 12 up to weaning (HR =

0.06). Heterogeneous mortality rates in different periods caused the Weibull distribution to fail in the validation of the baseline distribution, and a time-dependent distribution was needed. In our analysis the parametric survival function has been replaced with piecewise survival functions whose slopes change at givens points as suggested by Yazdi et al. (2002).

Relationship Between Sire Total Merit Index and Piglet Survival

The TMI of boars exhibited significant relationships with piglet pre-weaning survival (Table 2). Piglets originated by top TMI boars exhibited a 17% (20% for WTD) greater instantaneous risk of mortality than did piglets sired by intermediate or low TMI boars. The Kaplan-Meier estimate of survival functions stratified according to TMI class are presented in Figure 3. Some studies investigated the genetic relationship between piglet survival and performance traits such as backfat and fat and protein deposition in pig lines selected for efficiency of pork production (Herpin et al., 1993; McKay, 1993; Knol, 2001). The genetic correlation between piglet survival and backfat has been reported to be moderate (Knol et al. 2002). Knol (2001) reported that selection to reduce backfat is expected to increase birth weight and decrease piglet survival. Unfortunately, the relationship of piglet survival with production traits has been investigated only in lines used for pork production. In Italy, the breeding goal for boar lines originating slaughter pigs for dry-cured ham production differs greatly from the one pursued to enhance efficiency of pork production. Class of TMI exhibited an unfavourable relationship with survival of piglets. Biological explanations for these results could be related to selection of C21 boar line which did not include any crossbred survival trait during the studied period. Another interpretation is that, in the past, one of the major goals for this line, was to reduce excessive marbling of the raw ham. This trait is related to fat deposition and a reduced ability to metabolize triglycerides; it might play an important role in the thermoregulatory ability of the neonate and survival.

Variance Components and Heritability

Parameters of the approximated marginal posterior distributions of sire and nursed litter variance components and estimates of effective and equivalent heritability for COX, GDM, and WTD models are reported in Table 4. Estimated variance components and heritabilities obtained using different survival models were similar with the only exception of the WTD estimate of the nursed litter variance that was slightly greater than the corresponding estimates obtained with COX and GDM. The estimated nursed litter variance component was much greater than the sire variance estimate, confirming the importance of the common environmental generated by the nurse sow as a key factor affecting the survival of piglets before weaning (Casellas et al., 2004; Wolf et al., 2007). This effect was included as a time-dependent effect to account for variation in litter membership due to cross-fostering. Piglets of a nursed litter share common environmental effects due to occurrence of infectious diseases like diarrhoea and incidentals like diseased udders, and are affected by the maternal ability of the nurse sow. The nurse sow exerts effects on piglet survival which are strictly environmental for the piglets, but, for the sow, are affected by both genetic and environmental components. In this study cross-fostering occurred for 46% of the piglets. This raised a question in modelling simultaneously both maternal and permanent environmental effects. For a piglet which was not moved to a different litter, accounting for both the biological mother and the nurse sow effects was not feasible because these effects were confounded. In the present study, the choice was to model the permanent environmental effect determined by the nursed litter.

The modal estimates of the sire variance were close to 0.06 and the standard deviation of the approximate marginal posterior of this parameter was small for all models. The marginal posterior distributions of the sire variance are depicted in Figure 4. When a parametric (WTD and GDM) or semiparametric (COX) model was used in the construction of the likelihood function, it was observed that the resulting marginal posterior densities of σ_s^2 were very similar, with a slightly larger variance in the case of COX. In spite of a very high censoring rate, the standard deviations of

the posterior densities were small and this could be due to the size of the data set and the good pedigree structure which allowed a precise estimation of sire variance (Ducrocq et al., 2000). The estimated sire variance obtained in this study using proportional hazard models can not be compared directly with estimates obtained in other studies where alternative methodologies were used (e.g., linear o threshold models) because of scale differences. Van Arendonk et al. (1996) and Knol et al. (2002) used linear models ignoring the categorical nature of the trait. Few studies addressed estimation of variance component for piglet mortality using threshold models (Roehe and Kalm, 2000; Grandison et al., 2002; Arango et al., 2005) and only one had modelled the trait at the individual piglet level (Grandison et al., 2002).

Previous studies have reported low estimates of heritability for piglet mortality or survival rate, with an average of 0.05 (at the level of the litter and as a trait of the sow), as reviewed by Rothschild and Bidanel (1998). There is large variation across estimates obtained in different studies. Lamberson and Johnson (1984) reported an estimate of heritability for pre-weaning survival of 0.03, whereas Ferguson et al. (1985) reported values of 0.14 and 0.18 for in Yorkshire and Duroc, respectively.

In our analysis, using the formula of Yazdi et al. (2002), the estimated heritability, in the unrealistic situation of no censoring, was 0.14. After correction for the large censoring rate (86%), the equivalent heritability was very low (0.03) and comparable to estimates reported by other authors (Lamberson and Johnson, 1984; Knol et al., 2002; Damgaard et al., 2003; Casellas et al., 2004; Wolf et al., 2007).

Piglet pre-weaning survival additive genetic variance is large enough to allow economically viable selection as suggested by Knol et al. (2002). Furthermore, the implementation of a routine genetic evaluation based on survival models is feasible, even for large populations. The inclusion of the results of such an evaluation in breeding programs seems possible and is probably advisable, for

economic as well as for ethical reasons. However, the relationships between survival and breeding goal traits should be considered to optimize selection strategies.

Sire Rankings Under Different Models

The relationships between sire rankings obtained using different survival models are depicted in Figure 5. The rank correlations between COX and GDM, COX and WTD, and GDM and WTD were 0.99, 0.98, and, 0.97, respectively. Changes in sire rankings due to use of different models were limited and occurred preferentially at intermediate rank positions. Because COX is a semiparametric model, it is less sensitive to an incorrect model choice. Nevertheless, when analyzing short periods of time, the dates of failure are rather broadly grouped and the time scale has to be considered as discrete. This is a common situation in the analysis of piglet pre-weaning survival and the assumption of continuity in the baseline hazard distribution and absence of ties between ordered failure time (Cox, 1972) associated to the use of proportional hazard models may be violated. Since the Weibull model is *a priori* not sensitive to ties, and WTD is more flexible than a pure Weibull model (Yazdi et al., 2002; Tarrés et al., 2006), WTD seems to be a better option than COX for the analysis of piglet pre-weaning survival. Results of this study indicate that, as suggested by Casellas (2007), the fitting of parametric survival models can be easily improved with the simple addition of a time dependent effect. Although the high flexibility of COX is advantageous, semi-parametric approaches imply greater demands in computational requirements and time needs (Ducrocq et al., 2000). Hence, WTD seems to be a more advantageous model for the genetic evaluation of piglet pre-weaning survival because the vector of first derivatives of the log-likelihood function is much easier to be computed and the Hessian matrix is usually very sparse (Ducrocq et al., 2000).

IMPLICATIONS

The relationship between crossbred piglets pre-weaning survival and a total merit index used for selection of terminal boars for dry cured ham production was investigated using survival analysis techniques. Because this relationship was unfavorable, the inclusion of piglet pre-weaning

survival in the breeding goal of the line is advisable. Sire rankings provided by different survival models were very similar. Comparison of the predictive ability and goodness of fit of different survival models as well as of performance of survival and threshold models for the analysis of piglet survival will be the matter of future studies.

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TABLES AND FIGURES

Table 1. Number of records and percentage of uncensored records in each level of fixed effects included in survival models

Effect	Level	Records	
		N	uncensored, %
Sex	Male	6,936	17.8
	Female	6,988	10.8
Cross-fostering	No	7,515	16.5
	Yes	6,409	11.2
Parity of the nurse sow	1	2,241	15.7
	2	2,727	14.2
	3	2,241	12.9
	4	1,892	12.7
	5	1,597	13.6
	6	1,177	14.1
	7 or higher	2,049	14.7
Size of the nursed litter	less than 6	300	59.3
	from 6 to 8	1,246	13.7
	from 9 to 11	6,175	12.4
	from 12 to 14	4,696	12.9
	greater than 14	1,507	15.8
Sire total merit index	less than -1 SD	2,120	14.0
	from -1 SD to 1 SD	9,689	13.9
	greater than 1 SD	2,115	14.9
Total		13,924	14.1

Table 2. Tests for statistical significance of fixed effects based on changes of likelihood statistic obtained with a semiparametric proportional hazard model

Effect	df	Test[1]	
		Sequential	Last
Crossfostering	1	95.7 ***	106.7 ***
Sex	1	18.8 ***	21.0 ***
Year-month of birth	71	412.3 ***	254.9 ***
Parity of the nurse sow[2]	6	16.9 **	17.1 **
Size of the nursed litter[2]	4	151.3 ***	150.8 ***
Sire total merit index	2	6.2 *	6.2 *

[1]Sequential: effects were included in the model in sequential order, the numbers expressing the increase of -2 × log likelihood after including the effect in the model. Last: the full model was compared with models excluding one effect at a time, the numbers indicating the loss of -2 × log likelihood after excluding the effect from the model

[2]Time-dependent effect

$P < 0.05$, $^{**} P < 0.01$, $^{***} P < 0.001$

Table 3. Estimated hazard ratios (HR) and 95% confidence interval of HR for fixed effects included in Cox, grouped data (GDM) and Weibull time-dependent (WTD) models

Effect	Level	Model[1]								
		COX			GDM			WTD		
		HR	LB	UB	HR	LB	UB	HR	LB	UB
Sex	Male	1.00			1.00			1.00		
	Female	0.81	0.75	0.88	0.81	0.75	0.88	0.81	0.75	0.88
Crossfostering	No	1.00			1.00			1.00		
	Yes	0.61	0.57	0.67	0.60	0.56	0.66	0.60	0.56	0.65
Parity of the nurse sow	1	1.02	0.88	1.59	1.02	0.88	1.59	1.06	0.92	1.21
	2	1.00			1.00			1.00		
	3	0.84	0.73	0.99	0.84	0.72	0.99	0.85	0.73	1.00
	4	0.76	0.65	0.89	0.76	0.65	0.89	0.76	0.66	0.90
	5	0.79	0.67	0.96	0.79	0.66	0.95	0.80	0.68	0.97
	6	0.88	0.74	1.06	0.88	0.74	1.06	0.89	0.75	1.07
	7 or higher	0.85	0.74	1.00	0.85	0.73	1.00	0.88	0.76	1.04
Size of the nursed litter	< 6	3.89	3.10	4.60	3.94	3.23	4.79	3.94	3.43	5.08
	from 6 to 8	1.15	0.98	1.34	1.15	0.98	1.34	1.15	1.01	1.39
	from 9 to 11	1.00			1.00			1.00		
	from 12 to 14	1.40	1.25	1.58	1.40	1.25	1.58	1.78	1.57	1.98
	> 14	1.60	1.29	1.98	1.62	1.30	2.00	2.98	2.39	3.70
Sire total merit index	< - 1 SD	0.95	0.84	1.10	0.95	0.83	1.10	0.97	0.85	1.12
	from -1 to 1 SD	1.00			1.00			1.00		
	>1 SD	1.17	1.01	1.33	1.17	1.20	1.35	1.20	1.04	1.37
Period	< 6 d							1.00		
	from 6 to 12 d							0.21	0.18	0.24
	>12 d							0.06	0.05	0.07

[1]LB: lower bound of 95% confidence interval of HR; UB: upper bound of 95% confidence interval of HR.

Table 4. Parameters of the approximated marginal posterior distributions for Cox, grouped data (GDM) and Weibull time-dependent (WTD) models

Effect[1]	Parameter	Model		
		Cox	GDM	WTD
Sire	σ_s^2	0.058	0.059	0.060
Nursed litter	σ_{nl}^2	0.638	0.637	0.683
Effective h^2		0.137	0.139	0.137
Equivalent h^2		0.029	0.030	0.030

[1]Effective h^2: $h^2 = \frac{4\sigma_s^2}{1+\sigma_s^2+\sigma_{nl}^2}$, where σ_s^2 is the mode of the approximated marginal density of the sire variance component, σ_{ns}^2 is the nursed litter variance calculated as σ_{ns}^2 = trigamma(γ) and γ is the parameter of the log-gamma distribution; equivalent h^2: $h_{eq}^2 = \frac{4\sigma_s^2}{\sigma_s^2+\sigma_{nl}^2+\frac{1}{\bar{p}}}$ where $\bar{p}$ is the average proportion of uncensored records

Figure 1. Survivor function estimated by the Kaplan-Meier method

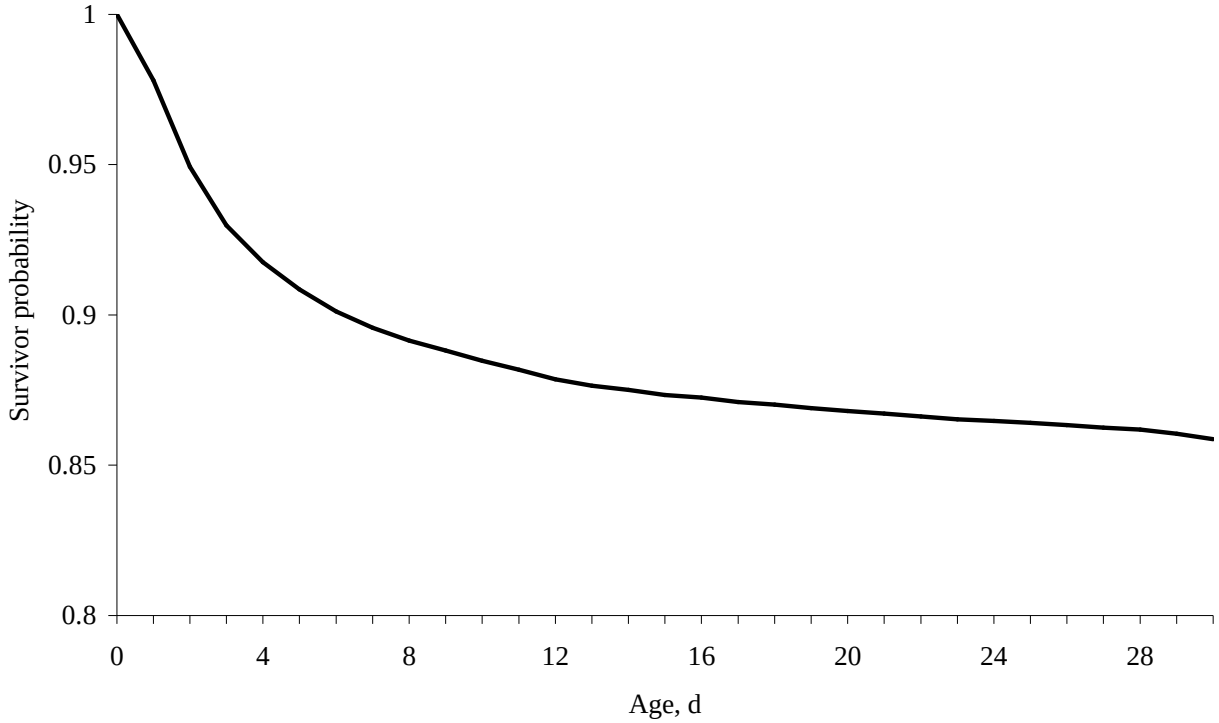


Figure 2. Estimate of the hazard function based on the Kaplan-Meier estimate of the survivor function

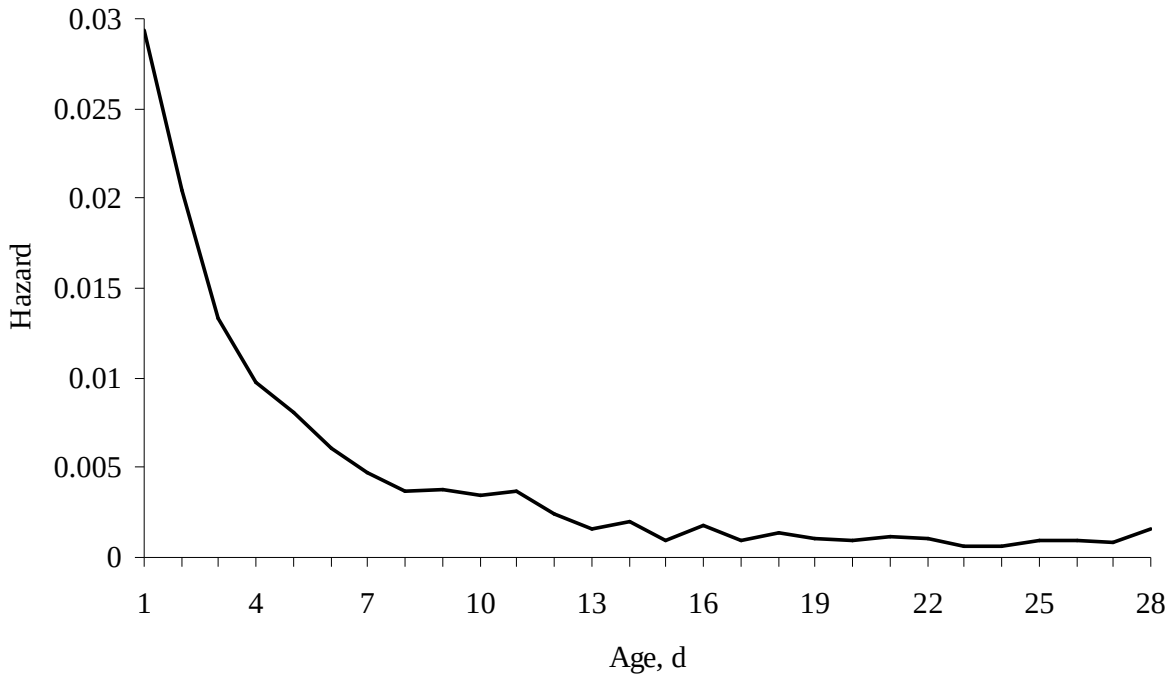


Figure 3. Kaplan-Meier estimate of survival functions stratified by total merit index (TMI) sire. TMI 1: less than -1 SD. TMI 2: from -1 SD to 1 SD. TMI 3: greater than 1 SD

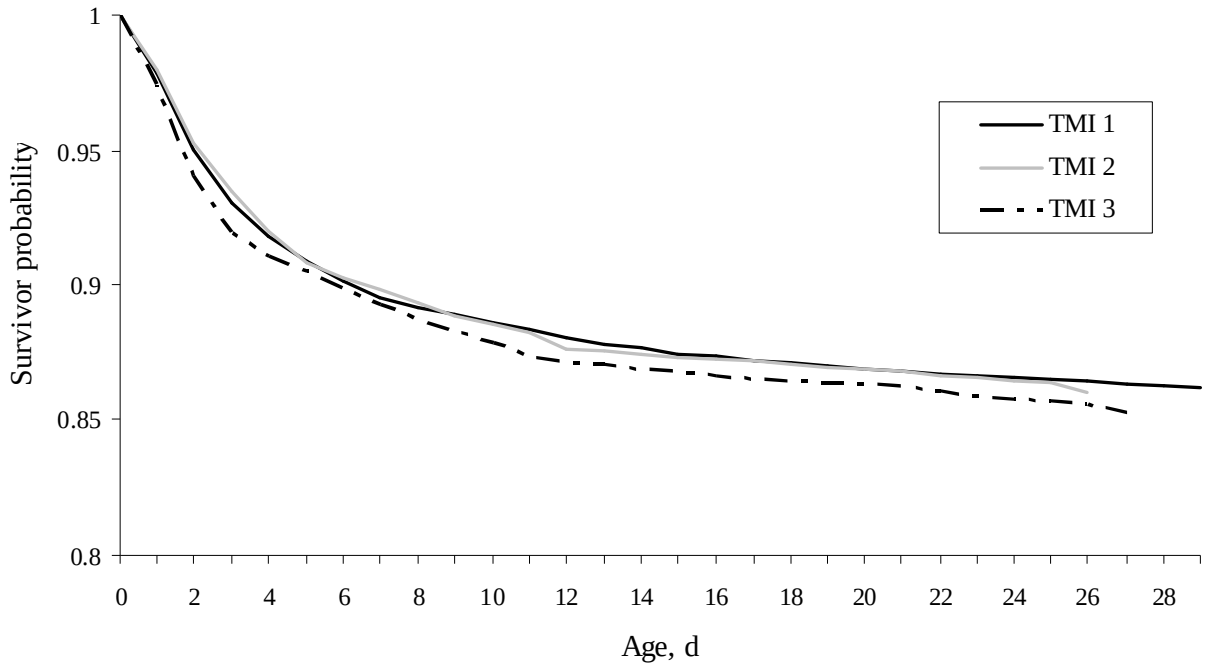


Figure 4. Gram-Charlier approximation of the marginal posterior density of the sire variance obtained with Cox, Grouped data (GDM) and Weibull time dependent (WTD) model

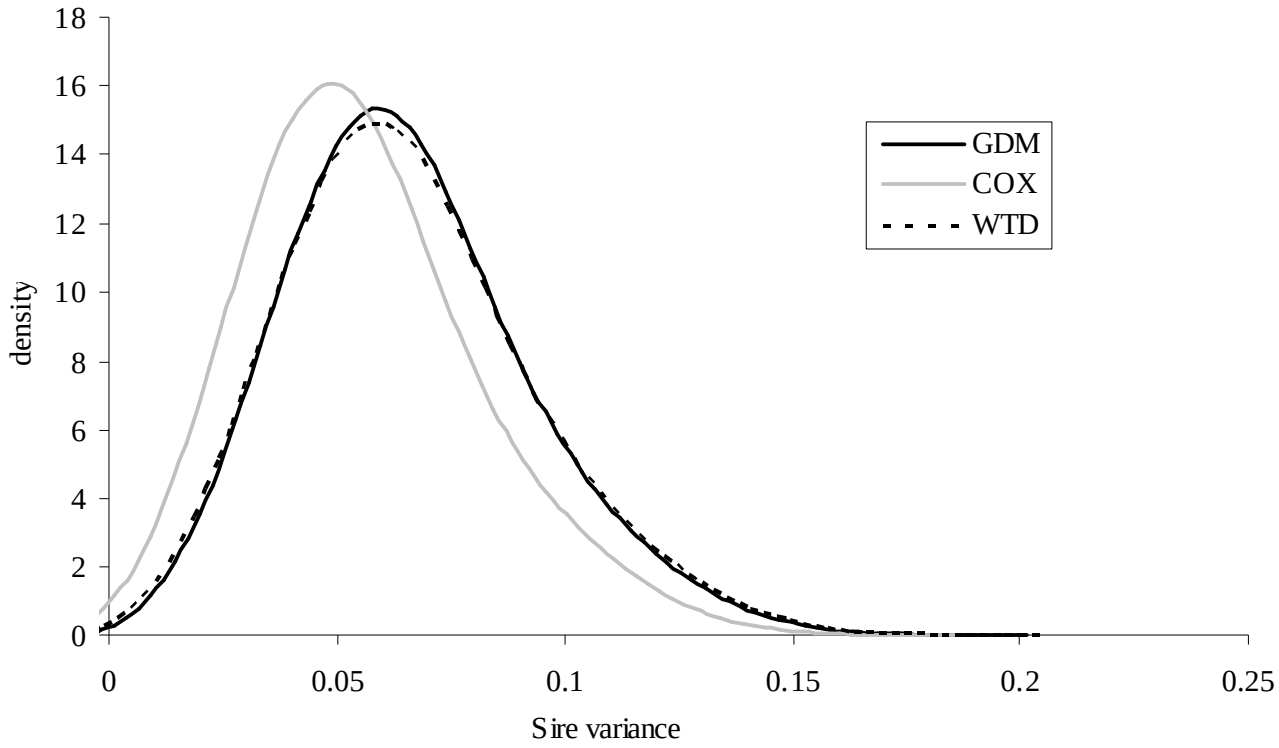
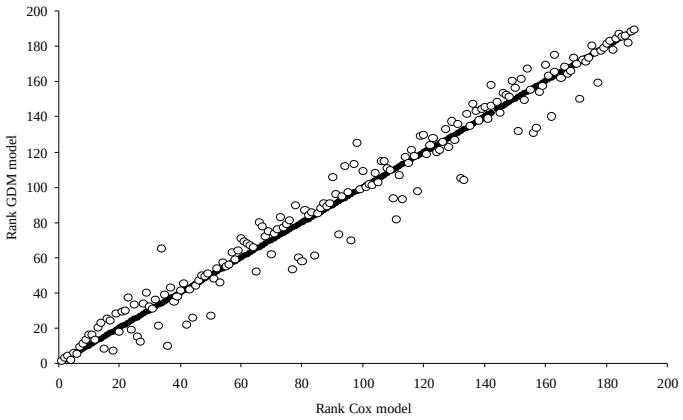
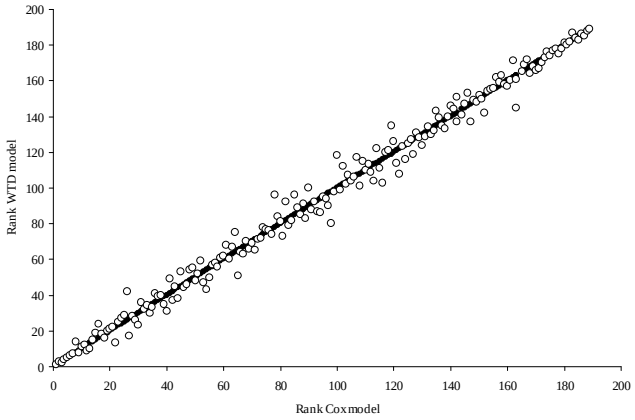


Figure 5. Relationships between boars ranking obtained from different survival models: A) Cox and Weibull time dependent (WTD) model. B) Cox and Grouped data (GDM) model. C) Weibull time dependent and Grouped data model

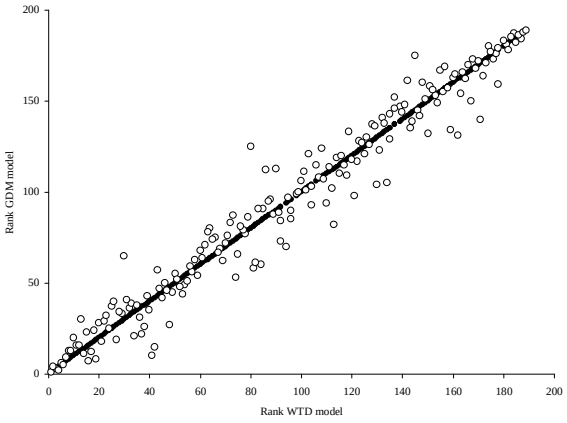
A)



B)



C)



Chapter 3

Piglet survival at birth

The relevance of purebred information for predicting genetic merit of survival at birth of crossbred piglets[1]

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ABSTRACT

The objective of this study was to infer (co)variance components for piglet survival at birth in purebred (P) and crossbred (C) pigs. Data were from 13,643 (1,213 litters) C and 30,919 (3,162 litters) P pigs, produced by mating the same 168 P boars to 319 Large White-derived crossbred females and 1,413 P sows, respectively. The outcome variable was piglet survival at birth as a binary trait. A Bayesian bivariate threshold model was implemented via Gibbs sampling. Flat priors were assigned to the effects of sex, parity of the dam, litter size and year-month of birth. To those of litters, dams and sires, Gaussian prior distributions were given. Marginal posterior means (SD) of the sire and dam variances for liability of piglet survival in P were 0.018 (0.008) and 0.077 (0.020), respectively. For C, sire and dam variance estimates were 0.030 (0.018) and 0.120 (0.034), respectively. The posterior means (SD) of the heritability of liability of survival in P and C and of the genetic correlation between these traits were 0.049 (0.023), 0.091 (0.054), and 0.248 (0.336), respectively. The symmetrical 95% Bayesian confidence region (-0.406, 0.821) for the genetic correlation between P and C liabilities of piglet survival included zero. Results suggest that the expected genetic progress for piglet survival in C when selection is based on P information may be nil.

Key words: Bayesian analysis, piglet survival, threshold model, genotype x environment interaction

INTRODUCTION

Crossbreeding of swine is widely accepted as an effective commercial production practice (Merks, 1988). Structured crossbreeding can exploit additive and non-additive genetic effects to advantage (Siegel, 1988). Although economic returns in pig production derive mainly from crossbred performance, selection of prospective parents is usually based on purebred performance.

The genetic correlation between purebred and crossbred performance provides an indicator for evaluating the effectiveness of reciprocal recurrent selection (Comstock et al., 1949), combined purebred and crossbred selection (Wei and van der Werf, 1994), and of the use of data recorded only in crossbreds for evaluation of purebreds (Lutaaya et al. 2001). Often, estimates of such genetic correlations for production traits have been less than unity (Merks, 1988; Wei and van der Werf, 1995) indicating that selection of parents in one type of mating system may not optimize progeny performance in another type of system (Mulder and Bijma, 2005).

Mortality at birth constitutes a major problem in pig production because up to 8% of newborns are stillborn (Van der Lende et al., 2000) arising ethical and economical problems. Evidence of genetic influences on stillbirth has been provided by Johnson et al. (1999) and Knol et al. (2002). Survival at birth has traditionally been considered as a trait of the sow (Grandison et al., 2002; Arango et al., 2005) and no previous studies have been conducted on a sire level.

Even though genetic variation for stillbirth rates seems to exist, the development of a successful breeding program needs to investigate whether purebred performance (in a nucleus) predicts accurately outcomes in crossbreds (commercial tier).

The objectives of this study were to investigate sources of variation of piglet survival at birth and to infer genetic parameters including the genetic correlation between survival at birth of purebred and crossbred piglets originated by the same sires.

MATERIALS AND METHODS

Animal Care and Use Committee approval was not obtained for this study because the data were obtained from an existing database; the analyzed records were registered in the nucleus farm and in the sib testing program of the C21 Large White boar line (Gorzagri, Fonzaso, Italy) from 2000 to 2006.

Animals and Data

Data were collected from year 2000 to 2006 in two locations: a selection nucleus farm (Riese Pio X, Italy) where pure C21 boars are produced and mated to pure C21 sows, and in a sib testing program farm (Todi, Italy), where the same C21 boars are mated to crossbred sows to produce crossbred piglets. The hybrid dams originated from a cross involving boars of a synthetic line, derived from Large White and Pietrain breeds, and sows of a Large White line selected for maternal ability and prolificacy. Crossbred paternal half-sib families provide the genetic evaluation program of C21 purebreds with phenotypic information for quality traits of raw and dry cured hams.

Data on survival of piglets at birth and up to weaning had been collected routinely in the nucleus and in the sib testing farm since 2000. Records included birth litter description (sow identification and parity, sire, date of farrowing, and size of the litter at birth), and individual piglet information (identification, sex, stillborn or alive at birth, weaning date or date of death if the piglet died during the suckling period, and date of transfer and foster dam identification for cross-fostered piglets). After discarding records with incomplete or inconsistent information, 13,643 individual survival records of crossbred piglets (1,213 litters) and 30,919 individual survival records of purebred piglets (3,162 litters), progeny of the same 168 C21 boars mated to 460 crossbred sows and 1,413 purebred sows, respectively, were available.

Statistical analysis

Falconer (1952) suggested that genotype x environment (G x E) interaction could be gauged via the genetic correlation between performance measurements in each of the environments regarded as different traits. Following this author, a Bayesian bivariate threshold model (Gianola and Foulley, 1983; Foulley et al., 1987) was fitted, where binary survival at birth (0 = alive, 1 = dead) was treated as the outcome of different traits when observed on purebred or crossbred piglets. With this, one can predict breeding values for piglet survival of C21 boars at the commercial level

(i.e., when mated with crossbred sows) by using information generated both at the nucleus and at commercial farms. This bivariate model produces estimates of the covariance and genetic correlations between the two traits with a high correlation suggesting mainly additive gene action. These estimates may be used to evaluate whether it is reasonable to select boars based on information of purebred piglets only or not.

The threshold model poses

$$y_{hijkl} \mid \tilde{y}_{hijkl} = \begin{cases} 1 & \text{if } \tilde{y}_{ihjkl} > 0 \\ 0 & \text{otherwise} \end{cases}$$

where y_{hijkl} ($h = 1$ if purebred or $h = 2$ if crossbred) indicates whether the $ijkl^{th}$ animal survived (1) or not (0) at birth, and $\tilde{y}_{hijkl}$ is the unobserved liability for survival of the animal in question. Above, *i,j,k,l*, indexes sire, dam, litter and piglet, respectively. The conditional distribution of the data, given the liabilities, is

$$p(\mathbf{y} \mid \tilde{\mathbf{y}}) = \prod_{h,i,j,k,l} 1(\tilde{y}_{hijkl} > 0)^{y_{hijkl}} 1(\tilde{y}_{hijkl} \le 0)^{1-y_{hijkl}} \qquad [1]$$

The model equation for the liability of survival is expressed as,

$$\tilde{y}_{hijkl} = 1(h=1)(\mathbf{x}'_{hijkl}\boldsymbol{\beta}_1 + s_{1i} + d_{1j} + l_{1k} + \varepsilon_{1l}) + 1(h=2)(\mathbf{x}'_{hijkl}\boldsymbol{\beta}_2 + s_{2i} + d_{2j} + l_{2k} + \varepsilon_{2l})$$

Above, $\boldsymbol{\beta}_1$ ($\boldsymbol{\beta}_2$) is a vector of 'fixed effects' and $\mathbf{x}'_{hijkl}$ is an incidence row vector; s_{1i}, s_{2i} are 'random' sire effects on the liability of purebred and crossbred piglets, respectively; similarly, d_{1j} (d_{2j}) and l_{1k} (l_{2k}) are 'random' dam and litter effects, respectively, on liability of purebred (crossbred) piglets. Finally, ε_{1l} and ε_{2l} are model residuals for purebred and crossbred liabilities, respectively. The $\boldsymbol{\beta}$'s included effects of sex (male or female); year-month of birth (72 classes from July 2000 through July 2006); parity number of the dam (1, 2, 3, 4, 5, and 6 or more) and litter size classes (1: ≤ 6 piglets, 2: from 7 to 9, 3: from 10 to 11, 4: from 12 to 13, 5: ≥ 14 piglets). Expected survival probabilities for piglets born from sows of different parities or born in litters of different

size were calculated as $1-\Phi\left[\hat{\gamma}_j, \overline{\mathbf{x}}'\hat{\boldsymbol{\beta}}\right]$, where: $\Phi[.]$ is the CDF of a standard-normal random variable, $\hat{\gamma}_j$ is the posterior mean of the effect of the j[th] parity or litter size class, $\overline{\mathbf{x}}'$ is an incidence vector for the fixed different that parity (litter size) evaluated at the mean of the data, and $\hat{\boldsymbol{\beta}}$ is the posterior mean of these effect.

Letting $\tilde{\mathbf{y}}_1$, $\tilde{\mathbf{y}}_2$ be the vectors of liabilities for purebreds and crossbreds, respectively, the bivariate model was

$$\begin{pmatrix}\tilde{\mathbf{y}}_1\\ \tilde{\mathbf{y}}_2\end{pmatrix}=\begin{pmatrix}\mathbf{X}_1 & \mathbf{0}\\ \mathbf{0} & \mathbf{X}_2\end{pmatrix}\begin{pmatrix}\boldsymbol{\beta}_1\\ \boldsymbol{\beta}_2\end{pmatrix}+\begin{pmatrix}\mathbf{Z}_{1s} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{2s}\end{pmatrix}\begin{pmatrix}\mathbf{s}_1\\ \mathbf{s}_2\end{pmatrix}+\begin{pmatrix}\mathbf{Z}_{1d} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{2d}\end{pmatrix}\begin{pmatrix}\mathbf{d}_1\\ \mathbf{d}_2\end{pmatrix}+\begin{pmatrix}\mathbf{Z}_{1l} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{2l}\end{pmatrix}\begin{pmatrix}\mathbf{l}_1\\ \mathbf{l}_2\end{pmatrix}+\begin{pmatrix}\boldsymbol{\varepsilon}_1\\ \boldsymbol{\varepsilon}_2\end{pmatrix} \quad [2]$$

where the $\mathbf{X}$'s and $\mathbf{Z}$'s are appropriate incidence matrices. Following a standard setting, it was assumed that model residuals in [2] followed a multivariate normal distribution with null means and the residual variance was set equal to one for both traits. Since no piglet had records on both traits, model residuals were treated as independent. Hence, the conditional distribution of all liabilities, given the location effects, was

$$\begin{aligned}p\left(\tilde{\mathbf{y}}_1,\tilde{\mathbf{y}}_2\middle|\mathbf{s}_1,\mathbf{s}_2,\mathbf{d}_1,\mathbf{d}_2,\mathbf{l}_1,\mathbf{l}_2,\boldsymbol{\beta}_1,\boldsymbol{\beta}_2\right)= N\left(\tilde{\mathbf{y}}_1 \mid \mathbf{X}_1\boldsymbol{\beta}_1+\mathbf{Z}_{1s}\mathbf{s}_1+\mathbf{Z}_{1d}\mathbf{d}_1+\mathbf{Z}_{1l}\mathbf{l}_1,\mathbf{I}\right)\\ \text{x } N\left(\tilde{\mathbf{y}}_2 \mid \mathbf{X}_2\boldsymbol{\beta}_2+\mathbf{Z}_{2s}\mathbf{s}_2+\mathbf{Z}_{2d}\mathbf{d}_2+\mathbf{Z}_{2l}\mathbf{l}_2,\mathbf{I}\right)\end{aligned} \quad [3]$$

Prior assumptions

The joint prior distribution was assumed to have the form

$$\begin{aligned}&p\left(\mathbf{s}_1,\mathbf{s}_2,\mathbf{d}_1,\mathbf{d}_2,\mathbf{l}_1,\mathbf{l}_2,\boldsymbol{\beta}_1,\boldsymbol{\beta}_2,\mathbf{G}_0,\sigma^2_{d2},\sigma^2_{l1},\sigma^2_{l2}\right)\propto\\ &\qquad p(\boldsymbol{\beta}_1,\boldsymbol{\beta}_2)p(\mathbf{s}_1,\mathbf{s}_2,\mathbf{d}_1\mid\mathbf{G}_0)p\left(\mathbf{d}_2\mid\sigma^2_{d2}\right)p\left(\mathbf{l}_1\mid\sigma^2_{l1}\right)p\left(\mathbf{l}_2\mid\sigma^2_{l2}\right)p(\mathbf{G}_0)p\left(\sigma^2_{d2}\right)p\left(\sigma^2_{l1}\right)p\left(\sigma^2_{l2}\right)\end{aligned} \quad [4]$$

$$\text{Above, } \mathbf{G}_0=\begin{pmatrix}\sigma^2_{s1} & Symm & \\ \sigma_{s1,s2} & \sigma^2_{s2} & \\ \sigma_{d1,s1} & \sigma_{d1,s2} & \sigma^2_{d1}\end{pmatrix}.$$

Thus, the model assumed the following covariances: between sire effects on purebreds and crossbreds; between sire and dam effects on purebreds, and between dam effects on purebreds and

sire effects on crossbreds. Because there was no additive genetic relationship between sires and dams of crossbred piglets and between dams of purebred and dams of crossbred piglets, [4] imposed that effects in $\mathbf{d}_2$ were uncorrelated with effects in $\mathbf{s}_1$, $\mathbf{s}_2$ and $\mathbf{d}_1$. A bounded [-100,000,100,000] uniform prior was assumed for the fixed effects so that $p(\boldsymbol{\beta}_1, \boldsymbol{\beta}_2)$ was flat. Using standard Gaussian assumptions and the factorization provided by [4], the joint distribution of litter (environmental), dam and sire effects was

$$\begin{pmatrix} \mathbf{l}_1 \\ \mathbf{l}_2 \\ \mathbf{d}_2 \\ \mathbf{s}_1 \\ \mathbf{s}_2 \\ \mathbf{d}_1 \end{pmatrix} \sim N\left[\mathbf{0}, \begin{pmatrix} \mathbf{I}\sigi_{l1}^2 & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma_{l2}^2 & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{A}_1\sigma_{d2}^2 & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{G}_0 \otimes \mathbf{A}_2 \end{pmatrix} \right] \qquad [5]$$

Above, $\mathbf{A}_2$ is the numerator of Wright's relationship matrix for C21 sires and C21 dams in the nucleus and $\mathbf{A}_1$ is the numerator of Wright's relationship matrix for crossbred sows (i.e., the dams of crossbred piglets). Note that $\mathbf{s}_1$, $\mathbf{s}_2$, $\mathbf{d}_1$ are augmented as in a standard maternal effects model, so that the covariance can be written using Kronecker product ($\otimes$) notation. The variance components associated with litter effects were assumed to reflect environmental variability across litters, and σ_{d2}^2 is the variance component for dam effects on liability of crossbred piglets.

Finally, following standard assumptions, an inverted Wishart distribution was adopted as prior for $\mathbf{G}_0$ and inverted chi-square distributions were assumed for the remaining variances. In short, $p(\mathbf{G}_0) = IW(\mathbf{G}_0 \mid \mathbf{S}_0, \nu_0)$; $p(\sigma_{l1}^2) = \chi^{-2}(\sigma_{l1}^2 \mid S_{0l1}, df_{l1})$; $p(\sigma_{l2}^2) = \chi^{-2}(\sigma_{l2}^2 \mid S_{0l2}, df_{0l2})$; and, $p(\sigma_{d2}^2) = \chi^{-2}(\sigma_{d2}^2 \mid S_{0d2}, df_{0d2})$. Here, the *S*'s and the *df*'s are scale and degrees of freedom parameters, respectively. With this setting, all fully conditional distributions have closed form and a Gibbs sampler can be used to draw samples from the posterior distribution of all unknowns (Sorensen and Gianola, 2002).

Model Comparison

Four models differing for the specification of the 'random' effects were evaluated. In addition to 'fixed' effects in [2], model 1 included the 'random' effects of litters; model 2 had the 'random' effects of litters and of sires; model 3 considered litters and dams effects, and model 4 had litter, dam and sire effects. BIC (Schwartz, 1978) was calculated for each of these models; BIC provides and approximation (on a logarithmic scale) of the Bayes Factor and was computed as

$$BIC(\log Lik, P) = -2\log Lik + P\log(N)$$

where $\log Lik$ is the log-likelihood of a fitted model, and P is the number of dispersion parameters. Models with smaller BIC are preferred. The model for inferring genetic parameters had to be kept simple and robust because of the (relatively) low number of sires, dams and litters. Other authors have reported problems in models that included permanent environmental and genetic effects of the sow (Wolf et al., 2007; Grandison et al., 2005; Kremer et al., 1999). However, omitting some of these effects may lead to biased estimates (Van Arendonk et al., 1996). Because of these reasons, rather than picking up estimated parameters from a single model, estimates from all models are reported.

Convergence diagnostics

Convergence of the Gibbs Sampler was assessed by inspection of trace plots, and the length of the chains was defined using the estimated Monte-Carlo standard error and the equivalent number of effective samples. Mixing differed across models. Because of this, chains of different length were run. After burn-in, the number of samples kept for inference was 300,000, 300,000, 300,000, 500,000 for model 1, model 2, model 3 and model 4, respectively.

RESULTS AND DISCUSSION

Table 1 shows total number of records, litters, dams and boars and rate of survival at birth for the purebred and crossbred lines. A slight difference in raw survival at birth was observed between purebred and crossbred piglets, reflecting both the effect of the cross (individual and maternal heterosis and maternal effects) and differences between environments. Knol (2001) reported that the amount of relative heterosis for litter survival was 1.55%. Blasco et al. (1995) also reported higher stillbirth rates for purebred than for crossbred litters.

The survival rate at birth for the crossbred population was similar to the one presented by Knol et al. (2002) for dam and sire lines, and by Moeller et al. (2004) for commercial dam lines. Purebred piglets exhibited a survival rate slightly lower and similar to that reported by Serenius et al. (2003) for Finnish Landrace. Variability of results across studies is large as a consequence of genetic and environmental differences between populations and of different trait definitions that might consider or not, as stillborn piglets, piglets dying in the early hours after birth.

The number of dams in the crossbred line was smaller than the one for the purebred line and the average number of litters per dam was larger for the crossbreds than for the purebreds. This was due to different numbers of sows in the nucleus and in the sib testing farm and to the occurrence of different replacement policies between the two locations. In the nucleus, replacement of sows was intense and mainly related to breeding decisions whereas sows producing crossbred piglets had less stringent culling which was mostly related to reduced fertility due to aging or occurrence of severe disease.

To provide a description of the data structure and of the degree of unbalancedness, the numbers of records per level of fixed effect for purebreds and crossbreds are reported in Table 2.

Models comparison

Posterior means of the log-likelihoods and BIC values for each of the investigated models are reported in Table 3. All models exhibited similar log-likelihoods, with a slight improvement in the goodness of fit when dam effects were included. As reported by Willham (1972), the choice of the appropriate model to examine traits affected by both a direct and a maternal effect is critical. The model must be reasonably accurate in describing the biology involved and yet simple enough to manipulate so that deduction can be made (Willham, 1972).

However, since all models had a similar goodness of fit, BIC favored (lower values) the most parsimonious models always. Results from models comparison suggest that the genetic variance, as stemming from sire effects, may be nil.

Non genetic effects

Posterior means of nongenetic effects did not vary much across models. Only results obtained with model 4 are reported. Female piglets had a smaller probability of survival than male piglets (0.93 vs 0.94 and 0.92 vs 0.95 for purebreds and crossbreds, respectively), and this is in disagreement with previous studies. Several authors reported that female piglets have a greater survival advantage than males (Svendsen et al., 1986; Becker, 1995; Knol et al., 2002). Figures 1 and 2 provide summaries for the effects of parity number and litter size in the probability scale. Due to the fact that purebred and crossbred pigs were raised in two different environments, differences between estimates of nongenetic effects for purebreds and crossbreds might be the result of environmental differences also. Moreover, with this data structure, any heterosis effect that might have affected the chance of survival of crossbred piglets cannot be disentangled from that of the environment. The influence of parity on piglet survival of crossbreds and purebreds is depicted in Figure 1. The probability of survival for piglets born from gilts was slightly lower than that for animals born from sows of parity 2, 3, and 4. The probability did not vary much from parity 2 to parity 4 and decreased thereafter, but more markedly in purebreds. As noted, sows producing

crossbred piglets were subjected to less stringent culling than dams of purebreds. As a consequence, the chance of an additional farrowing for dams of crossbred piglets was not influenced by selection. The situation in the nucleus was different, because the replacement rate was greatly influenced by selection. This may explain the difference between curves depicted in Figure 1. The decrease in probability of survival for advanced parities is in agreement with literature reports (Leenhouwers et al., 1999; Knol et al., 2002; Borges et al., 2005). This decrease might result from excessive fatness of old sows or aging of the uterus (e.g., a reduced muscular tone may hamper the farrowing process; Pejsak, 1984). The greater probability of stillbirth for the first parity also agrees with other studies (Cutler et al., 1992; Leenhouwers et al., 1999) and might be related to insufficient size of the birth canal in young gilts (Pejsak, 1984; Cutler et al., 1992).

The influence of litter size on piglet survival is depicted in Figure 2. In purebreds, the chance of survival was lower for larger litters, but the same figure was not observed in crossbreds. The negative influence of larger litter sizes on survival is well documented (Kerr and Cameron, 1995; Leenhouwers et al., 1999; Knol et al., 2002). A reason may be the association between litter size and farrowing duration causing greater risks of hypoxia (Herpin et al., 2001) when prolonged farrowings due to large litters occur. In agreement with several studies (Fahmy et al., 1978; Kerr and Cameron, 1995), an increased probability of stillbirth was observed for small crossbred litters. This might result from difficulties that younger sows have in carrying out a normal gestation.

Year-month of birth had a marked influence on variation of piglet survival. Piglet mortality varied much across years and across months of the same year. Likely, this is due to variation in climate, infectious pressure and management practices. The estimated probabilities of survival for different year-month of birth effects (results not presented) changed erratically across year-month classes, and did not exhibit a consistent trend over time, being in agreement with results obtained by Cecchinato et al. (2008).

(Co)variance components in purebred and crossbreds

Posterior means of dispersion parameters for purebred and crossbred piglet survival at birth are presented in Table 4. The variance due to litter effects ranged from 0.347 to 0.407 for purebreds, depending on the model, whereas the between-litter variance for crossbred ranged from 0.189 to 0.295. These estimates were similar to those reported by Ibáñez-Escriche et al. (2008) for Large White, Landrace and Pietrain populations. The (genetic) variance associated with sow effects was close to 0.08 in purebreds, but it was lower in crossbreds and estimated at 0.012 and 0.121 in model 1 and model 2, respectively. Genetic variance from sire effects decreased when maternal genetic effects of the sow were fitted. Fitting sow genetic effects also changed the estimated covariance between purebred and crossbred sire effects from 0.0015 in model 3 to 0.006 in model 4. However, the genetic (co)variance was small in both models.

The additive genetic effect of the dam includes both a direct effect of a dam's genes transmitted to the piglets on the survivability of the offspring and also a pure maternal genetic effect which is related to aspects of the dam that are relevant for the chance of surviving of the piglets and are influenced by the additive effects of the dam's genes (e.g., the uterine influence of the dam on piglet mortality at birth).

The sire effect accounts only for differences of piglet survival that are caused by the additive effects of genes that the piglets inherit from the sire. When included in a model along with additive genetic effects of the sire and of the dam, litter effects are expected to account for influences, common to all piglets joining the same litter and causing variation across litters, due to a strictly-environmental component and to non-additive gene effects shared by members of a full-sibs family. Hence, the inclusion of litter effects in the model should enhance the accuracy of breeding value predictions.

Heritabilities and Genetic Correlation

Figure 3 depicts trace plots and estimated posterior densities for the heritabilities of the liability of purebred and crossbred survivals and for the genetic correlation between liabilities of purebred and of crossbred piglets. Trace plots indicated that the algorithm mixed well. The skewed densities reflect the scant statistical information in the sample (i.e., a small number of boars). Summaries of the posterior densities of genetic parameters for the liability of piglet survival at birth are presented in Table 5. Heritability was low in both cases (0.05 and 0.09 for purebreds and crossbreds, respectively), but higher than the estimates reported by Knol (2001) and Roehe and Kalm (2000). Rothschild and Bidanel (1998) reviewed 96 studies on the number of piglets born alive (i.e., survival at birth as a trait of the sow) and reported a mean heritability of 0.09. For the same trait, Rydhmer (2000) reported a median heritability of 0.10, whereas Siewert and Cardellino (1996) obtained an estimate of 0.004. For the number of stillborns, Johnson et al. (1999) found a heritability of 0.17 and high estimates of heritabilities for litter size, number born alive and number of mummified piglets also. Most estimates of heritability for farrowing mortality or piglet survival using linear models are lower than the values obtained in our study, ranging from 0 to 0.04 (Haneberg et al., 2001; Grandison et al., 2002; Knol et al., 2002). Grandison et al. (2002) fitted linear and threshold models to stillbirth, crushing, total piglet mortality and birth weight. Estimates of heritability (based on sow components) with the threshold model were larger for all traits, especially for mortality. Only a limited number of studies (Grandison et al., 2002; Arango et al., 2005) have obtained (co)variance estimates for piglet survival using non-linear models. Individual piglet mortality or survival at birth has been traditionally analyzed by applying the classical linear model (van Arendonk et al., 1996; Knol et al., 2002; Mesa et al., 2006), albeit the nature of the trait is categorical. It is well known that the use of linear models with categorical data ignores their non-linear distribution and tends to produce underestimates of heritability (Gianola, 1982).

Because heritability estimates of mortality traits are low, with the possible exception of stillbirth, the potential for effective progress by selection is limited. As an alternative, Roehe and Kalm (2000) suggested selection for individual birth weight as a means for improving survival rate. The purported advantages of applying this strategy are not so clear because Knol et al. (2002) found a negative effect of weight at birth on farrowing mortality. Additionally, other studies have indicated that extreme selection based on weight at birth could produce a considerable increase in farrowing mortality, partly due to dystocia and prolonged parturition (Grandison et al., 2002; Damgaard et al., 2003; Holm et al., 2004). Roehe (1999) found direct and maternal heritabilities of birth weight of 0.08 and 0.22, respectively.

The genetic correlation between dam and sire effects on purebred piglet survival was 0.16 whereas the one between dam effects on purebred survival and sire effects on crossbred survival was 0.03, indicating that the two animal effects address at least partially the same trait. The sow effect includes the influence of the sow by means of uterine quality and her contribution to the genotype of the piglet. The sire only influences the genotype of the piglet.

The estimate of genetic correlation between survival at birth of purebred and crossbred piglets was moderate, with a posterior mean of 0.24 (Table 6) and a posterior mode at about 0.4 (Figure 3). A 95% Bayesian credibility region (-0.3856, 0.8203) included zero, and the posterior distribution was skewed. The reason for this was the low number of boars in the data. The low point estimate suggests G x E interactions. Other studies have reported relatively low genetic correlations between traits measured at different tiers of a genetic program (Standal, 1977; Groeneveld et al., 1984; Ollivier et al., 1984). The effect of G x E interaction on the efficiency of breeding programs is inversely proportional to the magnitude of the genetic correlation among genotypes in the different environments for same traits (Falconer, 1952). When genetic correlations are low, G x E requires a reappraisal of breeding strategies (Brascamp et al., 1985).

IMPLICATIONS

Covariance components for piglet survival at birth in purebred and crossbred pigs were inferred using bivariate threshold models with different degrees of model complexity. Results indicated that the direct additive genetic variance (due to sire effects) of piglet survival at birth is small and perhaps nil. Because the estimated correlation between additive genetic effects at the nucleus and at the commercial tier was low, results suggest that selection of sires for direct effects on piglet survival in a nucleus may lead to negligible genetic progress at the commercial level. Inclusion of this trait in the breeding goal of the line designed to produce sires for terminal mating seems questionable.

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TABLES AND FIGURES

Table 1. Number of records, boars, dams and litters and survival rate at birth for purebred and crossbred piglets

Item	Number or value	
	Purebred	Crossbred
Total number of piglets born	30,919	13,643
Survival at birth, %	89.41	92.79
Number of litters	3,162	1,213
Number of dams	1,413	460
Number of boars	168	168

Table 2. Number of records and fraction of stillborn per level of fixed effect in the purebred and crossbred line

Effect	Level	Number of records			
		Purebreds		Crossbreds	
		N	Stillborn, %	N	Stillborn, %
Sex	Male	15,438	10.19	6,863	7.44
	Female	15,481	11.04	6,780	6.94
Parity of the dam	1	11,706	12.05	2,208	8.78
	2	7,339	8.62	2,736	4.93
	3	5,032	9.16	2,218	6.98
	4	3,104	8.73	1,744	6.30
	5	1,886	15.27	1,519	7.50
	6 or higher	1,852	14.63	3,218	8.57
Litter size	less than 7	1,773	9.24	507	14.00
	from 7 to 9	3,793	10.04	972	6.48
	from 10 to 11	7,930	9.58	2,081	6.48
	from 12 to 13	9,749	10.60	3,269	6.51
	more than 13	7,674	12.19	6,814	7.36

Table 3. Average of likelihood values and Bayesian information criterion (BIC) for different models[1]

Model (random effects)	Number of dispersion parameters	Log-likelihood	BIC
M1 (Litter)	2	-11469.67	22960.75
M2 (Litter + Dam)	4	-11466.88	22976.58
M3 (Litter + Sire)	5	-11471.98	22997.48
M4 (Litter + Dam + Sire)	8	-11467.86	23021.36

[1] $BIC(\log Lik, P) = -2\log Lik + P\log(N)$ where $\log Lik$ is the log-likelihoods of a fitted model, and P is the number of dispersion parameters. Models with smaller BIC are preferred.

Table 4. Estimates of (co)variance components for piglet survival at birth on the liability scale[1]

Model (random effects)	Parameter[2]	Purebred	Crossbred
M1 (Litter)	σ_l^2	0.407 (0.025)	0.295 (0.035)
M2 (Litter + Dam)	σ_l^2	0.357 (0.024)	0.214 (0.032)
	σ_d^2	0.077 (0.023)	0.012 (0.034)
M3 (Litter + Sire)	σ_l^2	0.393 (0.024)	0.268 (0.035)
	σ_s^2	0.021 (0.009)	0.033 (0.021)
	$\sigma_{s1,s2}$	0.0015 (0.011)	
M4 (Litter + Dam + Sire)	σ_l^2	0.347 (0.025)	0.189 (0.032)
	σ_d^2	0.078 (0.020)	0.121 (0.034)
	σ_s^2	0.018 (0.008)	0.031 (0.018)
	$\sigma_{s1,s2}$	0.006 (0.008)	
	$\sigma_{d1,s1}$	0.005 (0.011)	
	$\sigma_{d1,s2}$	0.002 (0.016)	

[1]Estimates are the means (SD) of the marginal posterior densities of the (co)variance components.

[2] σ_l^2, σ_d^2, and σ_s^2 denote the variance of litter effects, additive genetic effects of the dam, and additive genetic effects of the sire, respectively; $\sigma_{s1,s2}$, $\sigma_{d1,s1}$, and $\sigma_{d1,s2}$, denote, for the additive genetic effects, the (co)variance between sire effects on purebred and those on crossbred animals, between dam and sire effects on purebred animals, and between dam effects on purebreds and sire effects on crossbreds, respectively.

Table 5. Estimates of genetic parameters for piglet survival at birth on the liability scale

				HPD95%[5]	
Parameter[1]	Mean[2]	SD[3]	MC_{se}[4]	Lower bound	Upper bound
h_1^2	0.0491	0.0233	0.0020	0.0127	0.0975
h_2^2	0.0916	0.0543	0.0061	0.0127	0.1971
r_A	0.2480	0.3363	0.0378	-0.3856	0.8203
$r_{d1,s1}$	0.1565	0.2808	0.0249	-0.4263	0.6839
$r_{d1,s2}$	0.0323	0.3273	0.0337	-0.5625	0.6366

[1] h_1^2 = heritability for purebreds; h_2^2 = heritability for crossbred; r_A = genetic correlation between purebred and crossbred piglet survival at birth; $r_{d1,s1}$ = correlation between dam and sire additive genetic effects on purebred piglet survival; $r_{d1,s2}$ = correlation between dam additive genetic effects on purebred piglet survival and sire additive genetic effects on crossbred piglet survival.

[2] Mean of the marginal posterior density of the parameter.

[3] SD of the marginal posterior density of the parameter.

[4] Monte Carlo Standard error.

[5] Symmetric 95% posterior density region.

Figure 1. Effect of parity of the dam on piglet survival at birth in the crossbred and in the purebred line

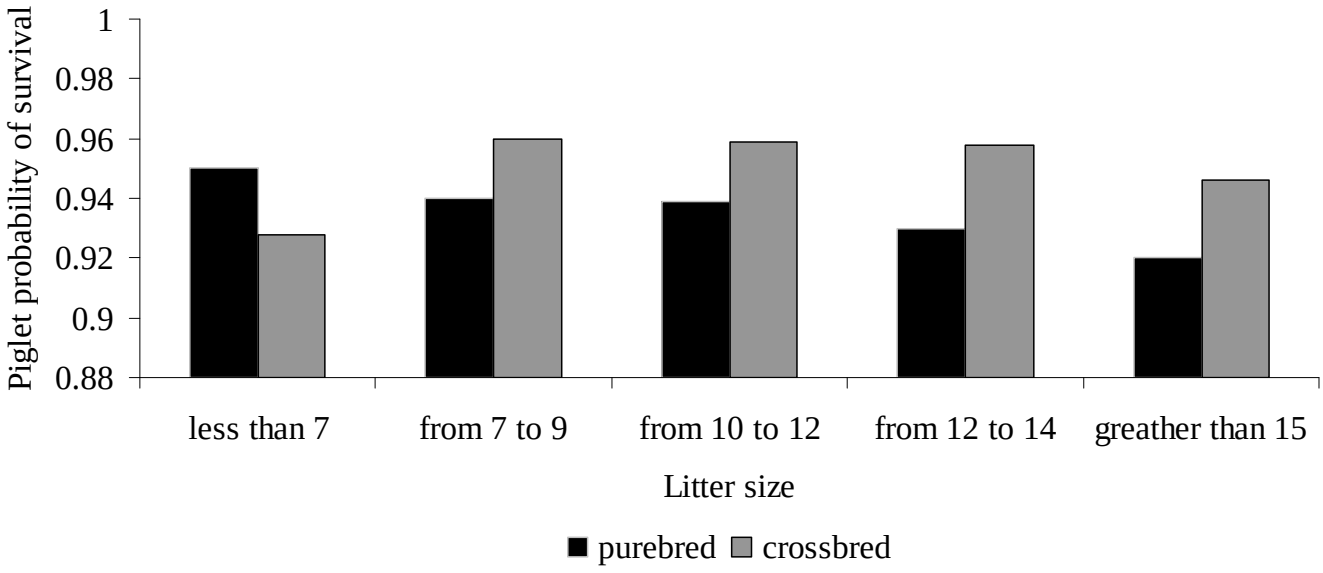


Figure 2. Effect of litter size on piglet survival at birth in the crossbred and in the purebred line

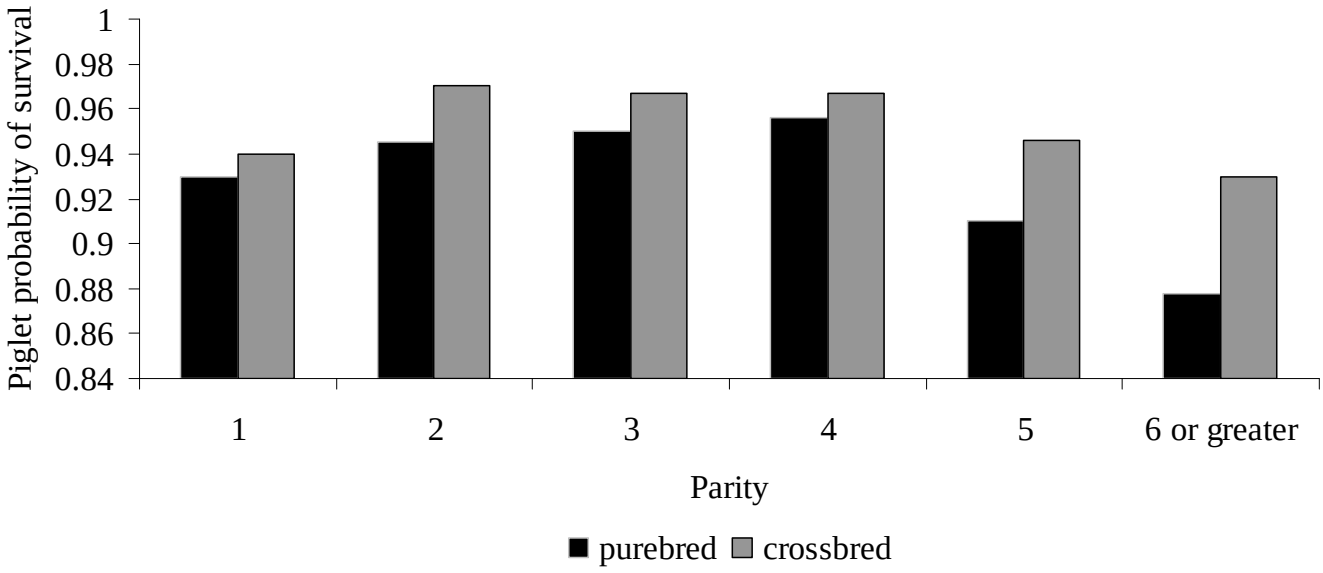
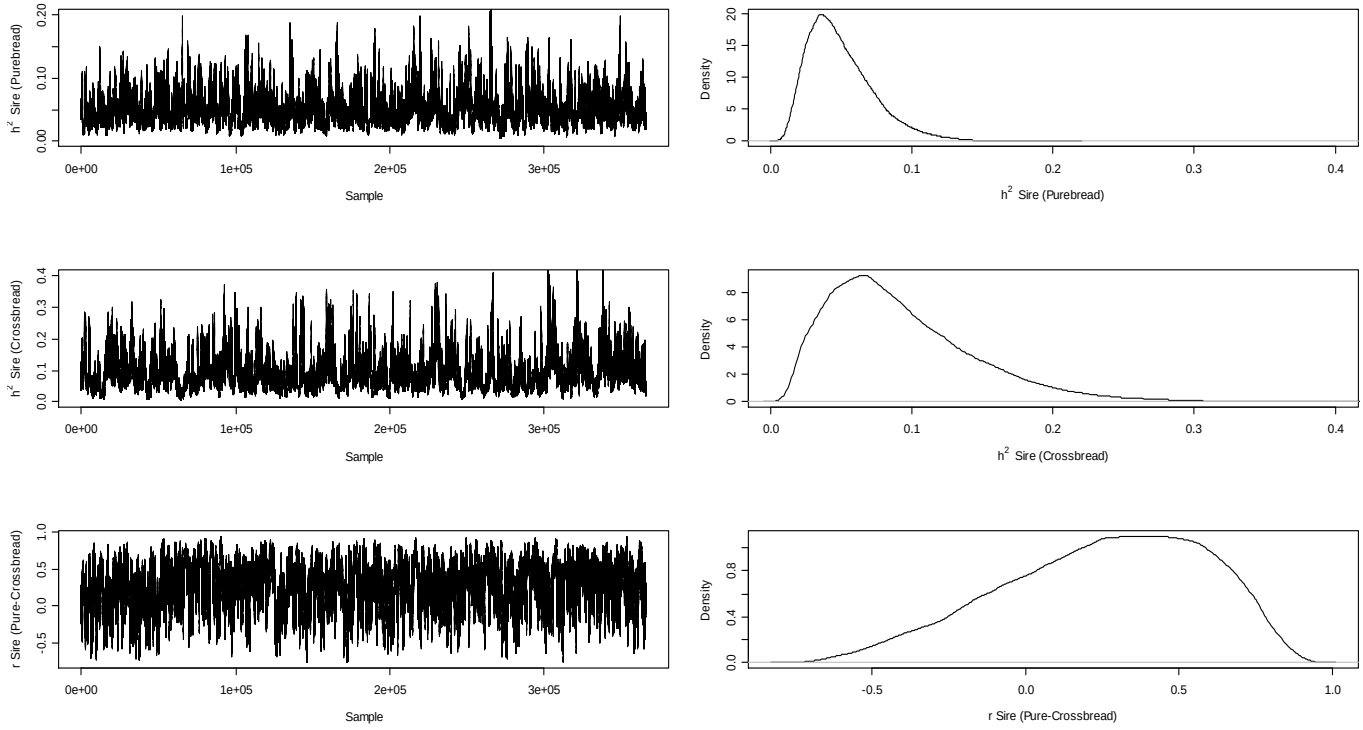


Figure 3. Estimated marginal posterior densities and trace plot of heritability of sire variances and of the correlation between the sire effects on pure and crossbred animals



Chapter 4

Model comparison for predicting genetic merit of piglet pre-weaning survival

A comparison between different Survival and Threshold models with an application to piglet pre-weaning survival in a dry-cured ham-producing crossbred line

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ABSTRACT

Different approaches for predicting genetic merit of piglet pre-weaning survival were compared using proportional hazard, threshold (TM) and sequential threshold model (STM). Data were from 13,924 crossbred piglets (1347 litters) born from 2000 to 2006 and originated by mating 189 Large White C21 Gorzagri boars with 328 Large White-derived crossbred sows. A frailty proportional hazard model, assuming two different baseline hazard function (Cox and Weibull) and including sire and nursed litter as a random effects were fitted. The TM and STM considered the same effects. Model fitting was evaluated in terms of goodness-of-fit and predictive ability, using the mean square error as reference parameters. Estimated sire variances for piglet pre-weaning mortality were low, and heritability ranged from 0.04 to 0.06. All four models led to similar ranking for sires, with strong correlation between methods. The STM had a better performance for predicting piglet survival but it had a lower performance in terms of goodness-of-fit than COX. The results in this study suggest that STM may, globally, be better than other methods tested, both for its better predictive ability of piglet survival in genetic evaluations and for its easier interpretation. Further, STM is computationally less demanding and can be extended to allow for different variance components by different period from birth to weaning.

Key words: pre-weaning mortality, piglet, survival analysis, threshold model, sequential threshold model

Introduction

Pre-weaning mortality of piglets has become an important issue from both economical and animal welfare point of views (Grandison et al., 2002). In last decades, different approaches were used to analyze pre-weaning survival. One possible approach is to consider survivability as a binary outcome (e.g. 0=alive at time t; 1=death at time t) and this may be done using a threshold model (Gianola, 1982; Gianola and Foulley, 1983). However, most researches dealing with piglet

mortality (or its complement trait piglet survival) have traditionally used linear models assuming a continuously distributed trait, thus ignoring its categorical nature, skewed distribution and censoring (Van Arendonk et al., 1996; Knol et al., 2002). Another alternative is the analysis of failure time (i.e. time of death) with regression models (Cox, 1972; Prentice and Gloeckler, 1978). An advantage of using a time-to event model is that there is no need to restrict the observations to an arbitrarily defined point as it is the case where a binary response is considered as in the threshold model (Ducrocq, 1997) Additionally, there are good strategies to handle censoring in survival regressions. Recently, a sequential threshold model (Albert and Chib, 2001) which can analyze categorical traits that occur in sequential order and accommodate time-dependent covariate was used to compare predictive ability against other models for number of inseminations to conception (González-Recio et al., 2005).

The aims of this study were to infer parameters of piglet pre-weaning survival with the survival analysis, threshold model and sequential threshold model in a crossbred slaughter pigs populations and then to asses their relative predictive abilities and goodness of fit.

MATERIAL AND METHODS

Animals and data

Data used in this study were collected in the sib testing program of the C21 Large White boar line (Gorzagri, Fonzaso, Italy) from 2000 to 2006. The description of the sib testing program as well as the breeding goals of the C21 Large White boar line are fully described in Cecchinato et al. (2008). Briefly, data on survival of piglets at birth and up to weaning were routinely collected in the sib testing program since 2000 and included birth litter description (sow identification and parity, sire, date of farrowing, and size of the litter at birth), and individual piglet information, i.e., identification, sex, status at birth (stillborn or alive), weaning date or date of death if the piglet died during the suckling period, and date of transfer and foster dam identification for cross-fostered piglets. Cross-fostering occurred for 46% of the litters and was of similar proportion for male and

female piglets. For piglets which died before weaning, survival time was computed as the difference between the date of death and the date of birth whereas for piglets still alive at weaning survival time to censoring was computed as the difference between the date of weaning and the date of birth.

After editing procedures, which aimed to discard records with incomplete or inconsistent information (120 piglets) and with unknown sire (105 piglets), a total of 13,924 individual survival records of piglets (1,347 litters) sired by 189 C21 boars mated to 328 crossbred sows were available for the study.

Survival Analysis

The individual piglet survival time was analyzed using survival analysis methodology. The survivor function for the general population was estimated previously using the Kaplan-Meier method (Kaplan and Meier, 1958). To test whether a Weibull distribution properly fitted the data, the log of the Kaplan-Meier estimate (nonparametric) of the survivor curves was plotted against the log of time. If the assumption for Weibull holds, a straight line should be obtained. Because the relation was not linear the assumption of a Weibull distribution function for the baseline hazard was not suitable for these data and alternative models were considered.

Cox model. In the Cox model (COX) the distribution function for the baseline $h_0(t)$ was left completely unspecified (Cox, 1972) and a semi-parametric proportional hazard model was used. The hazard function can be expressed as:

$$h_{ijklmnop}(t) = h_0(t)\exp\left[CF_i + SEX_j + YM_k + TMI_l + P_m(t') + NLS_n(t') + nl_o(t') + s_p\right]$$

where $h_{ijklmnop}(t)$ = hazard function (instantaneous probability of death) for a given piglet at time *t*; *t* = time (measured in days) from birth until death or weaning (censoring); $h_0(t)$ = baseline hazard function; CF_i = time-independent fixed effect of cross fostering (*i*, 0 = no; 1 = yes); SEX_j = time-independent fixed effect of sex (*j*, 0 = male; 1 = female); YM_k = time-independent fixed effect of year-month of birth (*k* = 72 monthly classes from July 2000 to July 2006); TMI_l = time-

independent fixed effect of class of standardized total merit index of the sires of the piglet (l = class 1: TMI < -1 SD; class 2: -1 SD ≤ TMI ≤ 1 SD; class 3: TMI > 1 SD); $P_m(t')$ = time-dependent fixed effect of parity of the nurse sow that could change value in the time space as a consequence of cross fostering (m = 1 to 7 or more); $NLS_n(t')$ = time-dependent fixed effect of nursed litter size that could change value in the time space as a consequence of cross fostering, carried out to homogenize size of litters, and of piglet mortality (n = class 1: ≤ 5 piglets, class 2: from 6 to 8, class 3: from 9 to 11, class 4: from 12 to 14, class 5: ≥ 15 piglets); $nl_o(t')$ = time-dependent random effect of the nursed litter which might change as a consequence of cross-fostering (o = 1 to 1,346), assumed to be independently distributed as a log-gamma with shape parameter γ from which the variance of the nursed litter effect σ^2_{nl} can be derived; s_p = time-independent random effect of sire effects (p = 1 to 189) assumed to follow a multivariate normal distribution with mean 0 and variance $\mathbf{A}\sigma^2_s$, where $\mathbf{A}$ is the additive genetic relationship matrix among sires and σ^2_s is a variance component for sire.

The sire variance σ^2_s, as well as the γ parameter of the log-gamma distribution were estimated using the Bayesian approach described in Ducrocq and Casella (1996). Multivariate normal for the sire effect and log-gamma priors for the nursed litter effect were combined with the likelihood function of the data to obtain an expression proportional to the joint posterior density of all parameters. Whenever a Cox model was used, the likelihood function was replaced by a partial likelihood (Cox, 1972) which does not contain any information about the arbitrary baseline hazard function. Further technical details of the random parameter estimation under survival analysis are given in Ducrocq and Casella (1996).

Weibull time dependent model. This model is a parametric model where a Weibull distribution ($\lambda\rho(\lambda t)^{\rho-1}$) was assumed as a baseline distribution function after the inclusion of a time-dependent covariate (Tarrés et al., 2005). The inclusion of time-dependent covariates P_c converts the Weibull hazard function into a Weibull time-dependent (WTD) hazard function:

$$h_0(t) = \begin{cases} \lambda_1(t) = \lambda\rho(\lambda t)^{\rho-1}\exp(P_1) & if\ t \in (0, c_1) \\ \lambda_1(t) = \lambda\rho(\lambda t)^{\rho-1}\exp(P_2) & if\ t \in (1, c_2) \\ \vdots & \\ \lambda_1(t) = \lambda\rho(\lambda t)^{\rho-1}\exp(P_{k+1}) & if\ t \geq c_k \end{cases}$$

This model assumes that the hazard for an individual is constant within each period of time but is different for the same individual between the c = 1, 2, …, k + 1 different periods, corresponding to k cut points. Cut points were identified with a spline regression of the log of the Kaplan-Meier survival function on time following the same approach used by Tarrés et al. (2005). To cope with this approach, all possible combinations of 1, 2, …, k cut points in the time space (pre-weaning period) were explored, avoiding combinations of adjacent days. In this study a time-dependent effect changing at d 6 and 12 was found. We estimated the Kaplan-Meier survival function (S) and tested possible "cut points" (different numbers and different locations) with a spline regression of ln(S) on time (t). We used the spline regression for variable number (up to 6) and variable location of cut points, and we choose the option providing an R^2 greater than 0.99 with the minimum number of cut points. This was achieved with 2 cut points located at 6 and 12 d. Details on the procedure are reported in Tarrés et al. (2005).

Heritabilities on the effective and equivalent scale were determined according to Yazdi et al. (2002). All survival analyses were carried out using the "Survival Kit" software, version 3.12 (Ducrocq and Sölkner, 1994).

Threshold Model

A threshold model (Wright, 1934; Dempster and Lerner, 1950; Gianola, 1982; Gianola and Foulley, 1983) was used for the analysis of pre-weaning mortality as a binary response. The threshold model postulates an underlying continuous random variable, called liability, λ_i, i = 1,…, n, such that the observed binary response (y_i) are the result of the fallowing relationship:

$$y_i = \begin{cases} 0 \, if \; \lambda_i \le \tau \\ 1 \, if \; \lambda_i > \tau \end{cases}$$

where τ is a fixed threshold, and y_i = 1 and y_i = 0 corresponds to record dead before weaning or not for observation *i*. The joint distribution of liability, $p(\boldsymbol{\lambda}) = p(\lambda_1,...,\lambda_n)$, liability is assumed to be normally distributed with mean vector $\boldsymbol{\mu}$ and covariance matrix $\mathbf{R} = \mathbf{I}\sigma_e^2$ where σ_e^2 is the variance in the underlying scale. Since the threshold and σ_e^2 are not identifiable, these parameters were set to some arbitrary values (τ = 0 and σ_e^2 = 1) to denote origin and scale of measurement, respectively. Hence, it was assumed that the vector of liabilities, given $\boldsymbol{\mu}$, had the distribution: $\boldsymbol{\lambda} \mid \boldsymbol{\mu} \sim N(\boldsymbol{\mu}, \mathbf{I})$. Then the probability that observation *i* is scored as 1, given μ_i model parameter vector , is defined to be:

$$\pi_i = \text{Prob}(y_i = 1 \mid \mu_i) = \text{Prob}(\lambda_i > 0 \mid \theta) = \Phi(\mu_i),$$

where $\Phi(.)$ is the standard normal cumulative distribution function. The statistical model for liability was:

$$\lambda_{ijklmnop} = CF_i + SEX_j + YM_k + TMI_l + P_m + NLS_n + nl_o + s_p + e_{ijklmnop}$$

where $\lambda_{ijklmnop}$ is the value of the underlying variable for the piglet of sire *p*, suckled into litter *o* with litter size *n* from a sow with parity *m*, with a class of total merit index of the sire *l*, born in the year-month class *k*, with sex *j* and belonging in class of cross fostering *i*. Some justification of the model is required. Effects were as in previous model with the only exception of the nurse sow parity (P_m), nursed litter size (NLS_n) and the permanent environmental effect determined by the nursed litter (nl_o), because this methodology is not able to handle time-dependent covariate. In this case, the levels belonging to the sows suckling piglets for the largest period were assigned to each observation. In this study cross-fostering occurred for 46% of the piglets. This raised a question in modelling simultaneously both maternal and permanent environmental effects. For a piglet which

was not moved to a different litter, accounting for both the biological mother and the nurse sow effects was not feasible because these effects were confounded. In the present study, the choice was to model the permanent environmental effect determined by the nursed litter.

As in the previous model the random effects were: nl_o = nursed litter (o = 1 to 1,347 levels) distributed independently as N ($\mathbf{0}$, $\mathbf{I}\sigma^2_{nl}$), where σ^2_{nl} is the variance among litters; s_p = sire effect (p = 1 to 189 levels) distributed as MVN ($\mathbf{0}$, $\mathbf{A}\sigma^2_s$), where $\mathbf{A}$ is the additive genetic relationship matrix among sire and σ^2_s is a variance component for sire effects and $e_{ijklmnop}$ =random residual term assumed independently distributed as N (0,1).

In matrix notation, the model can be expressed as:

$$\boldsymbol{\lambda} = \boldsymbol{\mu} + \mathbf{e} = \mathbf{X}\boldsymbol{\beta} + \mathbf{W}\mathbf{q} + \mathbf{Z}\mathbf{s} + \mathbf{e}$$

where, $\boldsymbol{\lambda}$ is an n x 1 vector, $\boldsymbol{\beta}$ is a vector of location effects, $\mathbf{q}$ is a vector of nursed litter effects, $\mathbf{s}$ is a vector of sire effects, and $\mathbf{e}$ is the vector of residual effects, $\mathbf{X}$, $\mathbf{W}$ and $\mathbf{Z}$ are corresponding incidence matrices having appropriate dimensions.

A Bayesian implementation via the Gibbs sampler, based on Sorensen et al. (1995), for a multi-tier or hierarchical structure was adopted. Conditionally on the parameter vector, $\boldsymbol{\mu}$, the observed binary data was assumed to be the result on n independent Bernoulli trials with success probability π_i. Hence the conditional probability distribution of the observation was:

$$p(\mathbf{y} \mid \theta) = \prod_{i=1}^{n} \pi_i^{y_i} \left(1 - \pi_i\right)^{(1-y_i)}$$

Flat priors were used for fixed effects and variance components. Parameters were drawn from the posterior distributions using Gibbs sampling, as implemented in the program TM by Legarra et al. (unpublished),. Based on the visual inspection of the trace plots, a chain of 200,000 iterations was used, with a burn-in of 20,000 rounds and thinning interval of 10 samples.

Sequential Threshold Model

Survivability of piglets to weaning can be expressed in a sequential order and analyzed using a sequential threshold model (Albert and Chib, 2001). As described above, there exist three important stages in the weaning process (from 0 - 3, 3 - 6, 6 - weaning). This means that for an observation to be present at a given stage of the sequence, it must have passed through all previous stages (Gonzalez-Recio et al., 2005). In the case of this study, a piglet that was alive at weaning had to present and survive in all previous stages. Therefore, a single latent variable can be used to represent the piglet's propensity to survive to the next stage (1 - 3). The response y_i can take the value *j* only after stages 1, …, *j*-1 are reached, and then either "survive" or "failure" (death) in stage *j* is observed. Hence, the probability to survive at stage *j*, conditionally on the event that the piglet survived to stage $(j\text{-}1)^{th}$, is given by

$$\Pr(y_i = j \mid y_i \geq j-1, \boldsymbol{\gamma}, \boldsymbol{\beta}, \mathbf{q}, \mathbf{s}) = \Phi\left[\gamma_j - \left(\mathbf{x}_i'\boldsymbol{\beta} + \mathbf{w}_i'\mathbf{q} + \mathbf{z}_i'\mathbf{s}\right)\right]$$

where now $\boldsymbol{\beta}$ represents the systematic effects of cross fostering (2 levels), sex of the piglet (2 levels), year-month of birth (72 levels), class of TMI of the sire of the piglet (3 levels), parity of the nurse sow (7 levels), class of the size of the nursed litter (5 levels) and $\mathbf{x}_i$ is the corresponding incidence vector. As before $\mathbf{q}$ and $\mathbf{s}$ represent the random effects of nursed litter and sire additive genetic effect. Further, the vector $\gamma = (\gamma_1, \gamma_2, \gamma_3)$ represents unordered cutpoints; these cutpoints do not need to be ordered as in the case of an ordinal threshold model (Albert and Chib, 2001).

This model can also be formulated in terms of latent variables expressing the propensity of a piglet to survive to the next stage. It is possible to define latent variables $\{w_{ij}\}$ corresponding to each of the *j* stages, where $w_{ij} = \mathbf{x}_i'\beta + \mathbf{w}_i'\mathbf{q} + \mathbf{z}_i'\mathbf{s} + \mathbf{e}_{ij}$. The residuals e_{ij} were assumed distributed as $e_{ij} \sim \text{NIID}(0,1)$ We observe $y_i = 1$ if $w_{i1} \leq \gamma_1$, and we observe $y_i = 2$ if the first latent variable $w_{i1} > \gamma_1$ and the second latent variable $w_{i2} \leq \gamma_2$. In general:

$$y_i = \begin{cases} 1 & \text{if } w_{i1} \le \gamma_1 \\ 2 & \text{if } w_{i1} > \gamma_1,\ w_{i2} \le \gamma_2 \\ 3 & \text{if } w_{i1} > \gamma_1,\ w_{i2} > \gamma_2,\ w_{i3} \le \gamma_3 \end{cases}$$

These latent variables can be incorporated into a Markov chain Monte Carlo sampling scheme. The latent variable representation can be simplified by incorporating the cutpoints $\{\gamma_i\}$ into the mean function and fixing one of the cutpoints, usually $\gamma_1 = 0$. Each latent variable can have different explanatory variable (Gonzalez-Recio et al., 2005), therefore, accommodating time-dependent effects (including the vector of cutpoints), and a binary-threshold model can be fitted for the survivability at each stage event. The response variable is: "fail" (death at the present stage) = 0, or "survive" (pass to the next stage) = 1. Further details on this model may be found in Albert and Chib (2001) and Gonzalez-Recio et al. (2005) in an animal breeding context.

Effects, other than the cutpoints, were as in previous models. The litter effect was assumed distributed as: N ($\mathbf{0}$, $\mathbf{I}\sigma^2_{nl}$), where σ^2_{nl} is the variance among litters; the $\mathbf{s_p}$ = sire effect (p= 1 to 189 levels) was distributed as MVN ($\mathbf{0}$, $\mathbf{A}\sigma^2_s$), where $\mathbf{A}$ is the additive genetic relationship matrix among sire and σ^2_s is a variance component for sire effects and $\mathbf{e}_{ij}$ =random residual assumed independently distributed as N ($\mathbf{0}$, $\mathbf{I}\sigma^2_e$), where σ^2_e is the residual variance, which was fixed to one and, for simplicity, it was assumed to be constant at each step of the sequence.

Posterior distributions of the parameters were estimated using a Gibbs sampling algorithm for STM, (Sorensen et al., 1995; Sorensen and Gianola, 2002), drawing samples from a single chain of 200,000 iterations, with the first 10,000 samples discarded.

Comparison among models

Predictive ability. Prediction of future observations given past data is a concern for animal breeders that might be answered using the predictive ability, a notation that arises naturally in Bayesian statistics (Matos et al., 1997). Cross validation may provide a suitable scenario to check for better predictive ability between different models (Shao,1993). The method usually involves

omitting a portion of the available data, fitting a predicting model to the remaining data and then testing the model fit on the omitted portion. To asses predictive ability in this study, a cross validation was carried out following the same approach of Caraviello et al. (2003) and González Recio et al. (2005). Data set was randomly partitioned into four datasets, to performed a 4-fold cross validation, with the restriction that all levels of fixed effects were represented in each partitions. In addition, the number of records per sire or litter were as evenly distributed among the four partitions as possible. The PTA for sires in each training sets were calculated by survival analysis using the two different baseline (Cox and WTD), the TM and the STM. Survivability observations (i.e., binary indicators of survival to 3, 6 or 28 d of age among piglets that had the opportunity to survive that long) for piglets in one of each training sets were regressed (using logistic regression) on PTA obtained from each methodology, such that each sire's breeding value could be converted into his piglets probability of survival to 3, 6, and 28 d. Next, the expected number of piglets of each sire that would survive to 3, 6 or 28 d age (among those that had an opportunity to survive that long) in the testing set was calculated by multiplying the probability of survival to a given age from training set by the total number of piglets in the independent data set. The actual number of piglets in the independent data set that survived to 3, 6 or 28 d of life, among those that had an opportunity to stay that long, was subsequently determined for each sire, and the observe and expected numbers of "survivors" and "failures" were compared using the following χ^2 statistic:

$$\chi^2 = \left[(\text{Observed Survivors} - \text{Expeted Survivors})^2 + (\text{Observed Failures} - \text{Expted Failure})^2\right]$$

These χ^2 statistics were summed across sire, and the model that produced the smallest sum was regarded as the more accurate predictor of survivability. In addition Spearman and Pearson correlations between estimated breeding values obtained with the different models were calculated.

Goodness-of-fit. The goodness-of-fit were assessed using the local weighted regression and the mean square error (MSE). After obtaining predicted transmitting abilities (PTA) of all sires,

these were matched with the observed mortality rates in the original data set. Local regression is a nonparametric approach to fitting curves and surfaces to data based on smoothing (Cleveland and Loader, 1996). This method was used to approximate the relationship between mortality rate (response variable) and PTA estimates (explanatory variables) locally by a smooth curve based on a non-parametric function, using locally weighted least square. Weights are assigned such that points close (in the Euclidean distance) to the predictor value of interest receive a higher weight. The regressions were computed using the R software (R Development Core Team, 2007). The mean square error (MSE) for each method was calculated as the average of the squares of the differences between the actual average and the local weighted regression estimate at each point.

RESULTS AND DISCUSSION

Descriptive statistics

Brief description of data used in this study are summarized in Table 1. Data set contained 4.90%, 4.55% and 4.65% uncensored records (dead piglets) for stage one (from birth to 3d), two (from 3d to 6d) and three (from 6 to weaning), respectively. In total, 86 % of records were censored (animals still alive) at the end of the weaning period, which occurred at an average time of 28 d. Hence, piglet mortality from birth to weaning was 14% and average failure time for uncensored records (death of piglets) was 6 d. A detailed description of the influence of the non-genetic effects involved in the survival process is reported in Cecchinato et al (2008).

Variance Components and Heritability

Parameters of the approximated marginal posterior distributions of sire and nursed litter variance components and estimates of effective and equivalent heritability for COX and WTD models are reported in Table 2. The discussion about the estimated variance components and heritabilities obtained with both survival models are extensively reported in Cecchinato et al (2008).

Summaries of the posterior distribution of the parameters of the TM and STM are presented in Table 3. The estimated sire variance using the TM was 0.011 and the nursed litter variance was

0.147. The estimated sire variance from STM was similar to that from the TM (0.012), but estimates of nursed litter variance (0.166) was slightly larger. Besides, smaller posterior standard deviations were obtained for the genetic parameter and for the permanent environment generated by the nurse sow.

Note that the STM accounts for variation in litter membership due to cross-fostering, whereas TM does not. The estimated nursed litter variance was much larger than the sire variance estimate in both models, confirming the nurse sow common environment as a key factor affecting the survival of piglets before weaning (Casellas et al., 2004; Wolf et al., 2007). Estimates of heritability for pre-weaning mortality were low, which is in agreement with results of Kerr and Cameron (1995) and Roehe and Kalm (2000). Roehe and Kalm (2000) estimated heritability using linear, logit and probit scale. As theoretically expected for categorical traits, heritability was lower on the linear observed scale than on the logit or probit scale. Boettcher et al. (1999), studying longevity in dairy cattle, observed that the estimate of heritability obtained by survival analysis was superior to those obtained by the linear and threshold models (for binary traits), that could result from a better adjustment of the data to the survival model (Pereira et al. 2007). In a more recent study, Carlén et al. (2005), working with Swedish Holstein data, reported a higher estimate of heritability for the continuous trait (time to first mastitis or censoring - TFM), analysed by survival model, in relation to the binary one (mastitis - MAST), analysed by mixed linear model, suggesting that this result was probably partly due to an increased observed variation among cows using the trait TFM (more continuously distributed than MAST).

However, when comparing these heritabilities, one must consider that the traits evaluated by the threshold model versus survival analysis are slightly different. Van Arendonk et al. (1996) and Knol et al. (2002) used linear models ignoring the categorical nature of the trait. Few studies addressed estimation of variance component for piglet mortality using threshold models (Roehe and

Kalm, 2000; Grandison et al., 2002; Arango et al., 2005) and only one had modelled the trait at the individual piglet level (Grandison et al., 2002).

Previous studies have reported low estimates of heritability for piglet mortality or survival rate, with an average of 0.05 (at the level of the litter and as a trait of the sow), as reviewed by Rothschild and Bidanel (1998). There is large variation across estimates obtained in different studies. Lamberson and Johnson (1984) reported an estimate of heritability for pre-weaning survival of 0.03, whereas Ferguson et al. (1985) reported values of 0.14 and 0.18 for in Yorkshire and Duroc, respectively.

Predictive Ability

Table 4 shows Spearman (r_s) and Pearson (r_p) correlations between estimates of sire effects from the different models ranged between 0.82 and 0.98 (in absolute values). The WTD model had a higher agreement with the COX and the STM models ($r_s = 0.98$, $r_p = 0.96$ and $r_s = -0.96$, $r_p = -0.97$ respectively). The correlations between STM with the other 3 models were negative because this the trait for this model stands for survivability instead of risk to death (i.e. higher PTA estimate for survival models and TM indicates a larger probability of progeny mortality, whereas higher PTA for STM mean larger probability to survive to next stage). The four methodologies result in very similar sire rankings in a routine genetic evaluation, being the TM and COX model the most different in terms of ranking of sires ($r_s = 0.82$, $r_p = 0.84$). When comparing binary and continuous traits the sire's rank correlations found in the literature were, in general, lower, indicating a moderate reranking of sires, which is in agreement with the present study. Boettcher et al. (1999), analysing longevity in dairy cattle by threshold (binary trait) and survival models (continuous trait), found a ranking correlations between PTA equal to 0.90. Carlén et al. (2005) found correlations of 0.93, 0.89 and 0.88 (for lactation 1 to 3), between PTA of sires obtained for the continuous trait (time to first mastitis or censoring) analysed by survival analysis and for the binary one (mastitis)

analysed by mixed linear model. However, no previous estimates for genetic evaluation of piglet pre-weaning survival are available.

A stronger correlation between WTD and COX as well as between WTD and STM was expected because of the ability of the methods to handle time-dependent covariate. Similar results were reported by González-Recio et al. (2005) in dairy cattle studying number of services to conception, where sires' rank correlation was equal to -0.98, between a discrete proportional hazard model and STM.

Results from the predictive cross-validation are shown in Table 5, for each of the methods. The STM provided more accurate predictions for piglet pre-weaning survival than other models tested. It resulted in the smallest sum of χ^2 statistics in 3 out of 4 testing sets for stage 2 (3-6 days) and birth to weaning period, outperforming WTD and TM. The STM had also the best predictive ability for stage 1 (0-3 days) in two out of four testing sets, whereas WTD and COX model were better in the other two testing sets. The COX model seemed to predict better in the third stage, where fewer piglet death take place. Difference, in terms of sum of χ^2 statistics across sire, between COX and WTD, seemed to be similar in magnitude. Casellas et al. (2006) reported that a Weibull baseline hazard function with the inclusion of time-dependent effect might approximate a smooth function similar to the COX model. A time dependent effect (in our case changing at d 6 and 12) may be difficult to interpret as an independent effect in the model, but it has to be understood as a new component of the survival baseline, adjusting the aging process of the whole population (Casellas, 2007). The TM showed the worst predictive ability among methods used and did not performed better than any other methods in any of the tests. This was expected since there is loss of information when collapsing into a binary outcome. The cross validation were performed over the most important pre-weaning stages from a productive point of view (0-3, 3-6 and 6-28 d), which where also used to check survivability with the STM. This might be favouring the STM, however it

also performed better in a global stage (0-28). The flexibility of STM to discriminate between important productive stages is an important advantage for this model.

Goodness-of-Fit

Figure 1 shows the nonparametric fit relating progeny raw mortality the sire PTA for each of the four models. There was an association between PTA and progeny mortality and all the models seemed to have similar fitness. However, COX model performed better than other methods. A smaller bandwidth parameter was determined for this model, which led to a slight overfit, as shown in the regression line in Figure 1. After trial-error learning, this parameter value was chosen because it provided the smallest MSE, among those tested. The MSE for COX model showed differences greater than 11% regarding the other survival analyses model (WTD) (MSE = 75.88 x 10^{-4} vs. 85.55 x 10^{-4}). This result highlights the high flexibility of nonparametric approaches to fit the survival data, although it implies greater demands in computational requirements and time needs. Note that differences in time required between Weibull and COX models are substantial: e.g., 30 times greater in the survival analysis of laying hens reported by Ducrocq et al. (2000). The MSE from STM and TM were similar, 85.51 x 10^{-4} vs. 83.62 x 10^{-4} (differences close to 2%). These are the first available results comparing goodness-of-fit of TM and STM of piglet pre-weaning survival.

In general, the COX model showed better fit between progeny raw mortality and sire PTA than other models, with its MSE showing differences greater than 11% regarding STM, whereas the difference with TM was lower and close to 9%. Discrepancies between WTD and STM were minimal, lower than 0.1% (MSE = 85.55 x 10^{-4} vs. 85.51 x 10^{-4}) (Figure 1).

IMPLICATIONS

Four methods to analyzing pre-weaning survival in pigs were tested, leading to generally similar results. Estimated sire variances for piglet pre-weaning mortality were low, and heritability ranged from 0.04 to 0.06. All four models led to similar ranking for sires, with strong correlation

between methods. The STM had a better performance for predicting piglet survival but it had a lower performance in terms of goodness-of-fit than COX. The sampling distribution of the χ^2 statistic used to test predictive ability is unknown, therefore a very computationally demanding method, such as permutation test or bootstrapping would be needed to asses their significance. However, consistency of better predictive ability throughout cross validation folds was obtained for the STM, mainly in important productive stages such as survivability from birth to day 6 of life, where higher mortality occurs, and also in the global weaning period (birth to day 28).

The results in this study suggest that STM may, globally, be better than other methods tested, both for its better predictive ability of piglet survival in genetic evaluations and for its easier interpretation. Further, STM is computationally less demanding and can be extended to allow for different variance components by different period from birth to weaning.

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TABLES AND FIGURES

Table 1. Number of records, percentage of uncensored (i.e. dead piglets) and mean number of progeny per boars in each stage

Stage	No. progeny per boars	Records	
		N	Uncensored
From birth to 3 days	74	13,924	4.90
From 3 to 6 days	69	13,218	4.55
From 6 to weaning	66	12,548	4.65
From birth to weaning	74	13,924	14.1

Table 2. Parameters of the approximated marginal posterior distributions for Cox, and Weibull time-dependent (WTD) models

Effect[1]	Parameter	Model	
		COX	WTD
Sire	σ_s^2	0.058	0.060
Nursed litter	σ_{nl}^2	0.638	0.683
Effective h^2		0.137	0.137
Equivalent h^2		0.029	0.030

[1]Effective h^2: $h^2 = \frac{4\sigma_s^2}{1+\sigma_s^2+\sigma_{nl}^2}$, where σ_s^2 is the mode of the approximated marginal density of the sire variance component, σ_{ns}^2 is the nursed litter variance calculated as σ_{ns}^2 = trigamma(γ) and γ is the parameter of the log-gamma distribution; equivalent h^2: $h_{eq}^2 = \frac{4\sigma_s^2}{\sigma_s^2+\sigma_{nl}^2+\frac{1}{\bar{p}}}$ where $\bar{p}$ is the average proportion of uncensored records

Table 3. Estimated variance for sire (σ_s^2), nursed litter (σ_{nl}^2) and heritability (h^2) from the threshold model (TM) and sequential threshold models (STM).

Item[1]	Parameter	Model	
		TM	STM
σ_s^2	Mean	0.0116	0.0122
	SD	0.0078	0.0055
	Monte Carlo error	0.0003	0.00008
σ_{nl}^2	Mean	0.1472	0.1667
	SD	0.0198	0.0186
	Monte Carlo error	0.0003	0.00002
h^2		0.0400	0.0414

[1]Heritability calculated in the liability scale: $h^2 = \frac{4\sigma_s^2}{1+\sigma_s^2+\sigma_{nl}^2}$, where σ_s^2 and σ_{ns}^2 are the means of the marginal posterior distributions of the sire and nursed litter variance components respectively.

Table 4. Spearman (above diagonal) and Pearson correlations (below diagonal) between sire effects from the four different models[1]

	Models[1]			
	WTD	COX	TM	STM
WTD	-	0.98	0.90	-0.96
COX	0.96	-	0.82	-0.91
TM	0.91	0.84	-	-0.93
STM	-0.97	-0.89	-0.94	-

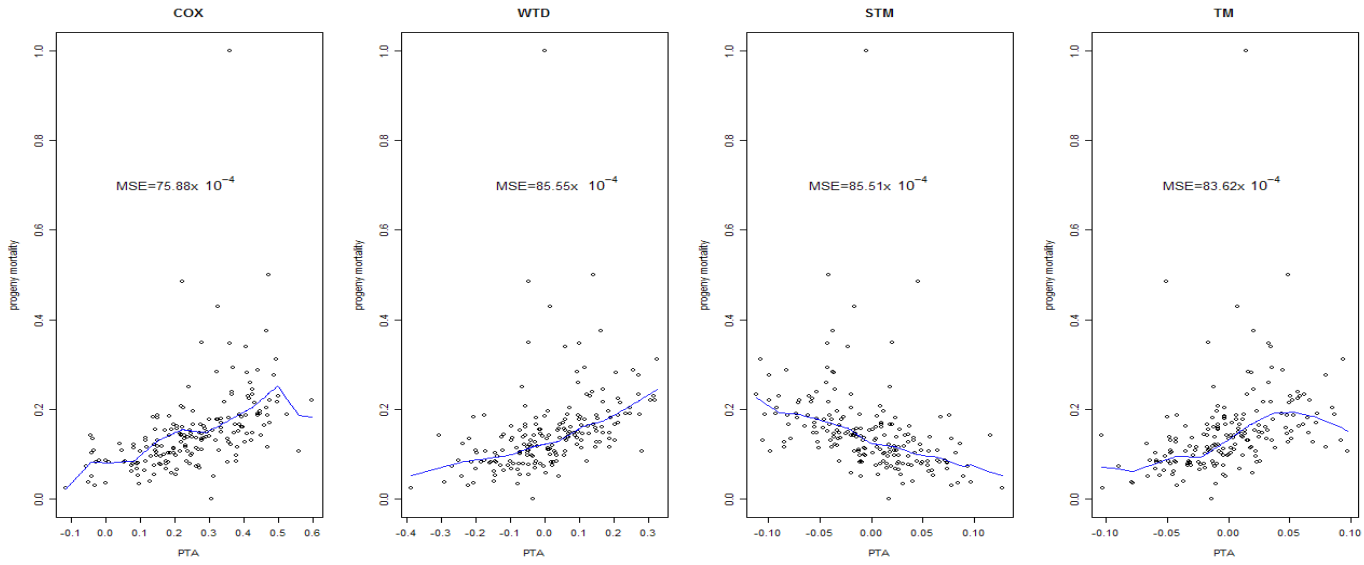
[1]COX = Cox model; WTD = Weibul time dependent model; STM = Sequential threshold model; TM=Threshold model.

Table 5. Sum of χ^2 statistics across sires, based on comparing the predicted vs observed number of survivors to 3, 6 or 28 days, among piglets that had an opportunity to survive that long, in each subset of the data (using probabilities of survival derived from the other subset), with smaller sums indicating more accurate predictions of piglets survivability.

Period	N. of boars[1]	Models			
		WTD	COX	TM	STM
A) training set N = 10,451 testing set N = 3,463					
From birth to 3 days	171	262.128	260.673	286.149	**255.348**
From 3 to 6 days	168	286.965	286.689	293.833	**285.106**
From 6 to weaning	163	**270.795**	275.147	289.414	280.286
From birth to weaning	171	802.926	809.830	904.209	**793.836**
B) training set N = 10,368 testing set N = 3,546					
From birth to 3 days	174	372.349	378.487	384.503	**351.187**
From 3 to 6 days	171	356.991	358.205	378.790	**346.179**
From 6 to weaning	166	342.645	**339.711**	368.577	363.308
From birth to weaning	174	798.560	810.305	957.356	**775.620**
C) training set N = 10,510 testing set N = 3,404					
From birth to 3 days	167	**389.805**	391.707	511.434	392.202
From 3 to 6 days	186	389.262	393.054	410.840	**368.790**
From 6 to weaning	184	267.349	**266.695**	279.903	270.444
From birth to weaning	167	799.496	820.856	883.633	**784.741**
D) training set N = 10,413 testing set N = 3,501					
From birth to 3 days	172	499.353	**498.463**	534.088	504.437
From 3 to 6 days	170	289.654	289.649	300.803	**283.527**
From 6 to weaning	170	279.964	**278.405**	306.701	285.420
From birth to weaning	172	874.504	**815.222**	1,079.12	876.622

[1] Boars with at least 10 piglets

Figure 1. Nonparametric locally weighted regression of raw progeny mortality on estimated sire predicted transmitting ability (PTA) for each of the four models. Mean square errors (MSE) are given for each method[1].



[1]COX=Cox model; WTD= Weibul time dependent model; STM= Sequential threshold model; TM=Threshold model.

Chapter 5

General discussion

GENERAL DISCUSSION

The main goal of this thesis was to gain knowledge in the genetic and non-genetic aspects of piglet survival and to evaluate the possibility of introducing this trait in the breeding goal of C21 boar line. Since genetic backgrounds of survival at birth and survival at weaning are different, independent analysis have been carried out for farrowing survival and pre-weaning survival. In the first contribution, pre-weaning survival has been considered and the relationship between crossbred piglets pre-weaning survival and a total merit index used for selection of terminal boars for dry cured ham production was investigated using survival analysis techniques. Because this relationship was unfavourable, the inclusion of piglet pre-weaning survival in the breeding goal of the line seems to be advisable. However in order to predict the consequences of the inclusion of piglet survival in a pig-breeding program with various other traits under selection, it is important to evaluate the genetic correlations between piglet survival and these other traits. Unfortunately, this type of analysis is difficult to be carried out under proportional hazard framework. Knol (2001) estimated the genetic correlations of piglet survival with several other economically important traits by using the linear model ignoring the categorical nature of the trait. Results indicated that single trait selection for improved piglet survival will increase feed intake, daily gain, and backfat thickness and decrease residual feed intake (Knol, 2001). It should be noted that the increased daily gain and backfat thickness as a correlated response to selection for piglet survival may be related to the better development of gastrointestinal tract and higher carcass fat percentage at birth in piglets with a high genetic merit for piglet survival, in comparison with those with a low genetic merit (Leenhouwers et al., 2002).

From the aforementioned it is clear that knowledge of genetic relations between production, reproduction and survival traits undoubtedly make it possible to build a selection index, which will allow a more balanced genetic progress in all traits of interest for pig producers, including an increase in both litter size and piglet survival while still maintaining progress in all other traits

of interest. It should be a challenge for all pig breeders, now and in the future, to achieve such breeding programs.

However the development of a successful breeding program needs to investigate whether purebred performance (in a nucleus) predicts accurately outcomes in crossbreds (commercial tier). This can be investigated by considering the two performances as different traits in a model and by estimating the genetic correlation between them. In the second contribution covariance components for piglet survival at birth in purebred and crossbred pigs were inferred using bivariate threshold models with different degrees of model complexity. Results indicated that the direct additive genetic variance (due to sire effects) of piglet survival at birth is small and perhaps nil. Because the estimated correlation between additive genetic effects at the nucleus and at the commercial tier was low, results suggest that selection of sires for direct effects on piglet survival in a nucleus may lead to negligible genetic progress at the commercial level. Hence, the inclusion of this trait in the breeding goal of the line designed to produce sires for terminal mating seems questionable.

In order to predict the genetic merit of piglet survival, many different statistical approaches have been proposed in literature. The categorical nature of the trait impose some difficulties in estimating genetic parameters. Most researches dealing with piglet mortality (or its complement trait piglet survival) have traditionally used linear models assuming a continuously distributed trait, thus ignoring its categorical nature, skewed distribution and censoring (Van Arendonk et al., 1996; Knol et al., 2002). Another alternative is the analysis of failure time (i.e. time of death) with regression models (Cox, 1972; Prentice and Gloeckler, 1978). An advantage of using a time-to event model is that there is no need to restrict the observations to an arbitrarily defined point as it is the case where a binary response is considered as in the threshold model (Ducrocq, 1997). Additionally, there are good strategies to handle censoring in survival regressions. Recently, a sequential threshold model (Albert and Chib, 2001) which can analyze categorical traits that occur in sequential order and accommodate time-dependent covariate was used to compare predictive

ability against other models for number of inseminations to conception (González-Recio et al., 2005).

In this thesis it has been shown that all models fitted led to similar ranking for sires, with strong correlation between methods. However the sequential threshold model may, globally, be better than other methods tested, both for its better predictive ability of piglet survival in genetic evaluations and for its easier interpretation. In literature several authors compared statistical models with different degree of complexity. For example, Lopez-Romero and Carabano (2003) compared random regression models using Legendre polynomials of orders 2–6. While more complex models fit the data better, the predictive ability of all the models was almost identical, indicating almost identical rankings of sires. Good arguments for following productivity in model comparisons were made by Blasco (2006). Reports from literature that simple and complicated models provide similar estimated breeding values are abound, e.g. Piles et al. (2006).

In conclusion, according to G. Box it is possible to state that ‘all models are wrong but some models are useful’, thus the search for a perfect model seems to be futile (Misztal, 2008). While more complex models may be needed to reveal the biology of traits, simpler models may suffice for genetic evaluation.

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