Data analysis to identify inherited conditions in an Australian commercial pig herd[1]

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Abstract

Piglet mortality is a significant area of economic loss for pig breeders and a predominant welfare issue. Certain genetic mutations cause pre- and peri-parturient piglet mortality, although information about such conditions is limited and management of these mutations are not commonly incorporated into breeding programs. The role of genetic mutations in piglet mortality within the Australian industry is unknown, however the population's closed nature increases risk of recessive inherited diseases. This study aimed to fill this knowledge gap and to work towards developing a method by which the impact of genetics on piglet mortality can be assessed using industry recorded data. The study utilised pedigree and production data from an Australian pig breeder to perform data analysis on reproductive and mortality traits with the aim to quantify the impact of inbreeding and potentially discover inherited disorders. Data from a commercial piggery from 1995 to 2019 were analysed and included records on 76901 litters, 17557 sows and 2143 sires. The parental population was predominantly purebred Landrace (R) and Large White (LW) pigs with a small proportion of Duroc (D). Litters were a combination of purebred and crossbred. Preliminary research involved curating the pig traits recorded within OMIA, followed by construction of mixed models in R and utilisation of the ASReml-R package. Two reproductive traits, litter size (total number born) and number born alive (NBA) were analysed along with five piglet mortality traits; mummified, stillborn, haemophilia, splay leg and atresia ani. Heritability estimates were low at 0.16 (litter size), 0.14 (NBA), 0.07 (mummified), 0.13 (stillborn), 0.10 (haemophilia), 0.18 (splay leg) and 0.27 (atresia ani), which

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indicates a small role of genetics. Litter inbreeding coefficients indicated that completely inbred litters would have 1.8 piglets less, 2.2 piglets less born alive, a 511% increase in the odds of at least one piglet with splayed legs and an 87% increase in the average number of stillborn piglets, compared with those with a zero-inbreeding coefficient. Data analysis was useful to observe trends in mortality incidence and estimation of heritability. However, in the absence of DNA analysis it was difficult to confirm the role and presence of recessive traits. This study may be built upon in the future to further investigate the approach and may be supplemented by the use of laboratory analysis to confirm results.

Keywords

breeding, genetics, novel approach, piglet mortality

Introduction

Piglet mortality continues to be one of the largest areas of economic loss for pig breeders, as well as posing a major animal welfare issue (Walters, 2010). Piglet death can be attributed to a range of causes, with crushing by the sow and low birth weight among the most common (Olsson et al., 2019). Some mortality causes, such as haemophilia and splay leg, have been associated with genetic mutations in some populations (Wiedemann et al., 2005; Maak et al., 2009), however, these are not extensively incorporated into breeding and selection programs.

The global pig industry is continuously growing in response to the growing human population and demand for animal protein (Australian Government, 2020). According to FAO data, over two billion pigs were processed globally in 2018 and more than 175 million tonnes of pig meat were produced (Food and Agriculture Organization of the United Nations, 2020). Of the 190 pig-producing countries and regions around the world, the Australian pig industry ranks 33rd in terms of head produced and 30th in terms of tonnes produced, at 5,378,100 head and 417,426 tonnes in 2018 (Food and Agriculture Organization of the United Nations, 2020).

In order to keep up with the growing demand, many pig breeders utilise selective breeding programs to enhance the genetics of their animals, particularly in terms of production traits such as carcase weight and backfat (Hermesch et al., 2005). In Australia, computer software programs, such as PIGBLUP (Animal Genetics and Breeding Unit, 2010), are available to industry breeders to assist them in performing genetic evaluation on their animals, by calculating individual animal estimated breeding values (EBV) for various traits (Animal Genetics and Breeding Unit, 2010). Initially, these selection programs focused on production traits, such as average daily gain and backfat (Hermesch et al., 2005), but newer versions have incorporated reproduction traits such as number of mummified piglets and 21-day litter weight (Crump et al., 2009). Many breeders utilise these programs to increase litter size (Schodl et al., 2019), however studies have shown that this can have detrimental effects on piglet health and

survival (Hayes et al., 2013). Additionally, it has been suggested that selecting for larger litters may also have an inadvertent influence on the incidence of pre- and post-parturient mortality in piglets (Sorensen et al., 2000).

Currently in Australia, disease prevention in the pig industry is focused on infectious diseases (Department of Primary Industries, 2020), rather than those with a genetic basis and are often not heavily incorporated into breeding programs (Hermesch et al., 2005). Additionally, research on these mortality causes, particularly those of recessive inheritance, is not extensive and their impact in Australia is currently unknown (Walters, 2010). However, there is a higher risk of mortality due to recessive causes within the Australian population, due to its closed nature as a result of importation restrictions (Department of Agriculture Water and the Environment, 2018) and the associated risk of inbreeding and inbreeding depression (Charlesworth and Willis, 2009; Kock et al., 2009).

The aim of this project was to investigate the current status of inherited conditions within one Australian pig breeding herd and the factors influencing their incidence. To do so, current information about inherited conditions in pigs was reviewed with the aim to facilitate future development of DNA diagnostics and a novel approach was formulated to mine industry data from an Australian pig breeder via data analysis, to analyse the impact of inbreeding and identify evidence for the presence of recessive traits causing piglet mortality within a population. This included the estimation of heritability values and observation of genetic and non-genetic trends within the population, to determine the role of genetics in piglet mortality. The results of these analyses were then used to suggest ways in which practical applications, such as DNA testing and selective breeding programs could be modified to incorporate causative mortality traits and reduce piglet mortality within the industry overall. It hypothesised that industry data could be mined to identify recessive traits causing pre- and post-parturient piglet mortality.

Material and methods

Online Mendelian Inheritance in Animals (OMIA) analysis

Preparation of OMIA data

At the time of data collection, all traits and disorders observed in pigs (so called phenes) recorded within OMIA were exported into an Excel file for further analysis. Additional fields including 'phene description' and 'impact on reproductive performance' were added to the existing Excel file in order to better suit the aims of this project (Table 1).

OMIA curation

A thorough literature search for new phenes, missing publications and mutations identified in pigs but not recorded in OMIA was performed via PubMed (PubMed, 2020), using key search words, such as 'pigs', 'disease', 'inherited' and 'mutation', in various combinations. Identified references and phenes were added to the database.

For phenes with no gene recorded, the associated papers on OMIA were searched for missing information, including analysing the figures for clues, such as images analysed sequence. Phenes with no gene and no recorded mutation were not investigated further.

For phenes with a recorded gene, information about the causal variants in the database are based on published information. To facilitate future development of diagnostic tools information needed to be updated to reflect the location on the current reference genome. Mutation locations and descriptions were determined through various methods depending on availability of published data and information (Figure 1).

Any mutations mapped in a reference genome other than the most recent, Sscrofa11.1, were remapped using the NCBI Genome Remap tool (NCBI, 2020b). The input for the tool required selection of the source organism, source assembly, target assembly, mutation location and chromosome. No settings under the 'remapping options' or 'data' headings were altered. The

input format for the mutation location and chromosome followed the input guide provided by NCBI (NCBI, 2020c).

To confirm mutation locations and determine the reference allele and strand, NCBI Genome Viewer was utilised (NCBI, 2020a). For phenes that impact reproductive performance and have a recorded gene, the gene sequence surrounding the mutation was determined using NCBI Genome Viewer (NCBI, 2020a) and recorded as this information is needed to develop a diagnostic SNP panel.

Estimation of population parameters using POPREP

The online software system, POPREP (Groeneveld, 2009), was used to generate three reports providing figures and summaries on key parameters including age structure, generation interval and effective population size, as well as inbreeding coefficients. To generate the reports, pedigree data for the population was uploaded in the input format specified by POPREP (Groeneveld et al., 2009).

Analysis of industry data

Provided industry data

The project was provided with six initial data files from a commercial pig breeder within the Australian industry. This included PIGBLUP data for three breeds (Duroc (D), Landrace (R), Large White (W)), containing information on production and pedigree data, as well as records of mummified piglets, piglet abortions and other piglet death events. Litter breeds were identified as an abbreviation composed of the sow breed, followed by the sire breed (e.g. Landrace sow and Large White sire produce a RW litter), resulting in four main litter breeds/crossbreeds; RR, WW, DD, RW, WR. All production data ranged from farrow dates in 1987 to early 2020 while pedigree data dated back to 1980.

Preparation of industry data

In preparation for analysis, the provided data files required 'cleaning' to ensure that all data was included in the final analysis. This included using Excel to delete additional spaces within cells using the 'Find and Replace' function and all remaining analysis, including the merging of files together to create a single record for each litter, was performed via R version 4.0 (R Core Team, 2020). Abortion data were excluded due to the inability to merge with other data as sows were culled post-abortion and therefore did not have corresponding records. Litter records with farrow dates before 1995 were excluded and all parities of 10 or more were collapsed into a single category. The provided piglet death events data contained 64 mortality causes, however this was collapsed into 14 new categories using R, for ease of analysis (Table 2). Selection of these categories was based on incidence, where causes with high incidence were retained, as well as those with previously reported or suspected genetic causes.

Analysis of prepared data

Exploratory data analysis was performed to obtain summary statistics for variables in the combined dataset, including the selected piglet mortality causes. Pedigree data were analysed in R using the ASReml-R package (Butler et al., 2018) to calculate the inbreeding coefficients of sires and dams and each litter (assuming that all litter mates have the same inbreeding coefficients when the inbreeding coefficient is calculated using pedigree information). Quantitative genetic analyses were then undertaken on seven traits by fitting mixed models to the data, these being five piglet mortality causes, as well as litter size and number born alive (NBA). Litter size was defined as the total number of piglets born per litter (i.e. sum of NBA and number of piglet born dead) to differentiate it from NBA. The fixed effects for each model were litter breed, parity, litter inbreeding and contemporary group, while the random effects were sow (polygenic effect, linked to the pedigree) as well as sire. The type of model used depended on the residual configuration for each response variable, including linear, logistic and Poisson models, as well as the trait type. For example, count traits, such as stillborn, were fitted

with Poisson models, while binary traits, such as mummified, were fitted with logistic models and quantitative traits, such as little size, were fitted with linear traits. Statistical significance was assessed at a level of $p < 0.05$ for each of the fixed effects; model-based predictions for each level of the fixed effects were obtained. For each trait, heritability, and repeatability were estimated and individual animal EBVs were calculated to determine genetic effects. Contemporary group estimates (CGEs) were determined using the models and plotted over time to analyse environmental/non-genetic effects.

Results

Online Mendelian Inheritance in Animals (OMIA)

Pig data in OMIA

At the time of data collection, the OMIA database contained 277 phenes in pigs (Online Mendelian Inheritance in Animals, 2020). Of these, 87 were classified as Mendelian traits/disorders and 37 were Mendelian traits with known causal variants (Online Mendelian Inheritance in Animals, 2020).

Although the summary table in OMIA suggested that there was a total of 277 traits/disorders recorded for pigs (Online Mendelian Inheritance in Animals, 2020), after compilation of all categories of data, it was noted that for several phenes different alleles had unique names. When considering all alleles with uniquely named phenes, there were a total of 288 pig phenes. Descriptions and impact on reproductive performance were added to all 288 phenes. Of these 288 phenes, 17 were identified to be Mendelian traits associated with disease in pigs, all of which have known mutations (Table 3).

OMIA curation

Changes were made to a total of 34 phenes on OMIA, most of which were Mendelian phenes (Table 3). Chromosome numbers were added to two additional non-Mendelian phenes; ear size

195 (OMIA 001579-9823) and ectodermal dysplasia-9 (OMIA 002157-9823) on chromosome 8 and
196 5, respectively.

197 ***Causal variant curation***

198 Eight phenes with recorded genes, had mutations recorded within Sscrofa10.2 and were
199 remapped to Sscrofa11.1. Two phenes had mutations located within earlier reference genomes
200 (i.e. Sscrofa7.0, Sscrofa9.0) which are not options on the NCBI tool as source assemblies and
201 were therefore not remapped. The remaining phenes had no reference genomes recorded in their
202 associated papers and were not edited. The chromosome number, genomic location and protein
203 location were added for 28, 9 and 4 causal variants, respectively.

204 ***POPREP reports***

205 The reports generated by POPREP indicated that the pedigree data included within the provided
206 dataset were relatively complete, with pedigree completeness index (PCI) values ranging
207 between 99.2 to 100 in 2019 across all three breeds in the herd (Table 4). In comparison to other
208 studies, these values were relatively high, indicating good data collection and record keeping
209 as well as very limited importations of pigs into the herd.

210 The reports also displayed effective population size (N_e) using various calculation methods
211 (Table 5). Some methods produced negative N_e values, indicating that the sample size was too
212 small for the tested method.

213 Each report produced included a short introductory section which stated that pedigree data were
214 only analysed for a maximum of 2000 pigs of each breed for every year, resulting in many
215 records being excluded from analysis. This was performed via random selection; however, this
216 may have influenced the accuracy and reliability of the results, particularly those regarding
217 reproduction and inbreeding. Hence, all further investigation was performed via R version 4.0
218 (R Core Team, 2020).

Industry data

Statistical summary

Between 1995 and 2019, there were 76901 litters, 17557 sows and 2143 sires. The predominant breeds within the population for both sows and sires were R and W (maternal lines), accounting for 88.5% and 75.8% of the total population for sows and sires respectively, while D (paternal line) pigs made up a smaller portion (Figure 2). The population contained a mixture of both crossbred and purebred litters, where purebred RR litters were most predominant with 23933 litters, accounting for 31% of the total number of litters in the population (Figure 3). Purebred DD litters were the least common, accounting for only 9.0% (6921 litters).

Graphical analysis of the provided pedigree data indicated a clear increase in inbreeding over time in all three sow and sire breeds (Figure 4), as well as in all analysed litter breeds (Figure 5).

Across all three breeds, parities 1-3 had the highest frequency, with an overall average parity of 3.62. R and W sows had slightly higher average parities at 3.82 and 3.49, respectively, compared to D sows with an average parity of 2.96 (Figure 6).

Litter size ranged from 0-30, with an average of 12.8 piglets (Figure 7). The crossbred litters had lower average litter sizes of 12.6 and 12.7, for RW and WR, respectively, compared to their purebred counterparts with average litter sizes of 13.3 and 13.5 for RR and WW, respectively (Figure 8). Purebred Duroc litters had the smallest average litter size at 12.2 piglets.

The average NBA per litter was lower than the average litter size for each litter breed, however showed the same trend. The crossbred litters had lower NBA per litter with averages of 11.4 (RW) and 11.5 (WR), compared to their purebred counterparts, with averages of 11.6 (RR) and 12.0 (WW). DD had the lowest average NBA at 10.7. The total NBA between 1995 and 2019

differed between sow breeds, where D sows had the lowest NBA of 68,587, while R and W had similar numbers of 478,160 and 345,844, respectively.

RW and WR had lower average numbers of dead piglets per litter at 1.13 (RW) and 1.01 (WR), compared to RR and WW with averages of 1.64 and 1.29, respectively. Despite having smaller average litter sizes and NBA, DD had an average of 1.40 piglets born dead per litter.

Piglet mortality data

In addition to the routinely reported data collected for BLUP analysis, this farm recorded specific piglet mortality data for each litter. The most common cause of piglet mortality across all breeds was 'Laid On', causing 4,398 (6.41% of total NBA) deaths in D piglets, 23,181 (4.85% of total NBA) in R piglets and 18,577 (5.37% of total NBA) in W piglets (Table 6).

Of the 64 original mortality causes, three were identified as having potential underlying genetic causes: haemophilia, atresia ani and splay leg. Of these, haemophilia had the lowest incidence within all three breeds contributing to the mortality of 0.001%, 0.03% and 0.01% of the total NBA for D, R and W piglets, respectively. Atresia ani affected R and W piglets at a similar rate, contributing to the mortality of 18% and 19% of piglets, respectively. Unlike the other previous two mortality causes, splay leg had the highest incidence in D piglets (0.93%), compared to R (0.85%) and W (0.73%).

Summary statistics for the above piglet mortality causes, as well as for mummified and stillborn are shown in Table 7. Stillborn had the highest incidence, affecting a total of 35466 litters and had the highest average number of affected piglets per litter of 0.85. All traits ranged from 0 affected piglets to above 10 affected piglets per litter. Splay leg had the highest maximum of 20 affected piglets in a single litter, while atresia ani had the lowest at 7.

Mixed models

Litter inbreeding coefficients

The litter inbreeding coefficient for litter size had a regression coefficient of -1.806, NBA had a coefficient of -2.23, splay legs had a coefficient of 1.632 and stillborn had a coefficient of 0.628. These results indicate that a completely inbred litter would have 1.8 piglets less 2.2 piglets less born alive, a 5.1-fold ($e^{1.632}$) increase in the odds of at least one piglet having splayed legs and an 1.87-fold ($e^{0.628}$) increase in the average number of stillborn piglets.

Trait EBVs

Plots of EBVs versus year of birth for each model are shown in Figure 9. The individual animal EBVs showed an increasing trend over time for litter size and NBA, indicating an increase in the genetic effects influencing the traits within the population. The EBVs for mummified, atresia ani and stillborn showed very little variation over time and had small amounts of fanning in the data, indicating little to no change to the genetic impact over time. Haemophilia had little variation in trendline but increased variability in EBVs over time, indicating no change in the influence of genetics over time, but large amounts of genetic variation in the animals. Splay leg had little variation over time, but a gradual decrease is visible in the trendline, indicating an overall reduction in the genetic effects influencing the trait.

Contemporary group estimates

The plotted CGEs versus year of birth for each model can be seen in Figure 10. Splay leg increased between 1995 and ~2010, but declined until 2018 where it increased again. This fluctuation was also observed in stillborn, where CGEs increased steadily since 1995, with a slight decrease between 2010 and 2015, followed by a further increase into 2019. Both mummified and atresia ani showed increasing trends, indicating a gradual increase in the probability of affected litters, while haemophilia showed a dramatic increase in probability from 0 in 2008, highlighting a new and emerging issue.

Model predictions

Litter breed predictions

The model-based means for each model trait, based on litter breed and parity are detailed in Table 8. Duroc (DD) had the highest probability of having at least one mummified piglet (0.43) and atresia ani (0.0293), while RR had the highest probability of having a litter affected by haemophilia (8.14×10^{-5}). Large White (WR) had the highest probability of having at least one piglet with splay leg per litter (0.11). Duroc (DD) had the highest average number of stillborn piglets per litter at 0.99.

Parity predictions

The probability of having a litter affected by mummification increases as parity increases, which is evident up until parity five, beyond which there is some fluctuation in results. There is a clear parity effect on the average number of stillborn piglets per litter, where the average number increases as parity increases. A clear parity effect is also seen for splay leg and atresia ani, where the probability of having an affected litter, increases as parity increases. Haemophilia showed no clear parity effect, however parities above five have a higher probability of having an affected litter compared to those below five.

Statistical significance

The statistical significance of the fixed effects in each model are listed in Table 9. Litter breed was statistically significant for all traits, except litter size and haemophilia. Parity was statistically significant for all traits and was the only significant fixed effect in the haemophilia model. Contemporary group (CG) was statistically significant for all traits, except haemophilia. Litter inbreeding was statistically significant for all traits, except mummified and haemophilia.

Heritability and repeatability

The heritability and repeatability values for each trait are listed in Table 10. Heritability ranged from 0.068 (mummified) to 0.267 (atresia ani). The heritability values for mummified and

haemophilia (0.101) were considered low, while the values for litter size (0.160), NBA (0.143), stillborn (0.126) and splay leg (0.182), were considered low to moderate. Atresia ani (0.267) was considered to have a moderate level of heritability. Splay leg had the highest repeatability (0.410) and mummified had the lowest (0.114).

Discussion

Selective breeding programs are often utilised within the Australian pig industry to ensure optimal production for human consumption and population growth (Hayes et al., 2013). However, selection of breeding animals is often focused on improving production traits, such as meat quality, carcase weight and litter size, which has been shown to have a detrimental impact on piglet mortality (Sorensen et al., 2000). Walters (2010) highlighted the importance of considering inherited conditions within pig production due to their association with piglet disease and mortality. Currently, there are no reports of tools that assist pig breeders in identifying and selecting against genetic traits, particularly those of recessive nature. However, due to the substantial economic and welfare impact associated with piglet mortality, multiple studies have suggested that piglet health and survival traits be incorporated into animal selection programs (Schodl et al., 2019). According to OMIA, several inherited disease-causing traits have been identified in pigs globally (Online Mendelian Inheritance in Animals, 2020), however there is little evidence to indicate the extent to which they are prevalent within the Australian pig population (Walters, 2010).

This study aimed to better understand the current situation regarding inherited conditions within the Australian population, followed by data analysis to identify areas of future research of possible recessive traits. This would be a first step towards developing DNA diagnostic tools that may greatly enhance the Australian pig population in the future. As there are no current tools used to identify recessive traits causing piglet mortality within the Australian pig population, the study design is a novel approach to determine whether data analysis is a feasible

method to achieve the desired result. Previous studies have utilised data analysis to estimate heritability values and investigate piglet mortality via creation of statistical models (Holl et al., 2004; Hermesch et al., 2005). However, most were focused on the impact of genetics on litter size, mummification and stillborn only, with little consideration of further recessive traits. Strange et al. (2013) used multivariate mixed linear models to estimate the heritability of piglet mortality traits, such as crushing, starvation and stillborn piglets, with the inclusion of splay leg, observed to be inherited in a recessive pattern (Matika et al., 2019). However, splay leg was combined with other traits, such as tail biting and hernias under the heading 'miscellaneous', making it difficult to discern the results regarding splay leg specifically. Alternatively, other studies have used regression models and data analysis to identify quantitative trait loci for reproductive traits, such as mummification and stillborn (Holl et al., 2004), while others incorporated halothane testing to determine the heritability of malignant hypothermia, an autosomal recessive trait (McPhee et al., 1979). Unlike many of these studies investigating the role of genetics in piglet mortality, the current study did not involve the physical testing of pigs or laboratory analysis, rather data were provided by an Australian industry pig breeder.

Data analysis was useful to gain a detailed understanding of the structure and trends within the population, including the incidence of disease, partially confirming our original hypothesis that industry data could be mined to identify recessive traits causing piglet mortality. However, without DNA testing and the recording of specific genetic information within the population, it is difficult to determine whether prevalent diseases and piglet mortality are caused by recessive inherited traits, other genetic abnormalities or environmental factors. In an attempt to determine the role of recessive traits in piglet mortality within this herd, Pedigree Viewer version 6.5 (Kinghorn, n.d.), a pedigree visualisation program was used to analyse the pedigrees of litters affected by haemophilia; a trait known to have a recessive mode of inheritance (Lozier and

Nichols, 2013). While the pedigree did show that multiple sires fathered more than one affected litter and clearly showed evidence of inbreeding within the investigated herd (Figure 11) it was difficult to explicitly conclude that the prevalence of haemophilia was associated with a recessive trait. This would be most heavily attributed to the occurrence of cross-fostering within the population, making it impossible to determine genetic relationships between individuals and therefore the pattern of inheritance for haemophilia.

It is possible that recessive lethal traits contribute to piglet mortality, however piglet mortality itself is a complex trait (Zak et al., 2017), hence the requirement to analyse heritability and the impact of inbreeding on various traits. In the investigated population, the contribution of genetics to the incidence of inherited disease was apparent, although environmental effects appeared to be more influential. This was evident through the increasing incidence of various mortality causes (Figure 12), despite low to moderate heritability values, where low heritability was <0.20 and moderate was between 0.21 and 0.40 (Bailey, 2014). A study conducted by Strange et al. (2013) calculated a heritability value of 0.16 for the 'miscellaneous' group, including splay leg. This estimate is close to our calculated value for splay leg of 0.18, however it is important to note that the value of 0.16 is not specific to splay leg and is therefore not an accurate comparison to our estimate. As litter size is an important reproductive trait, many studies have analysed its heritability, with varying estimates of 0.06 (Hermesch et al., 2005) to 0.12 (Ogawa et al., 2019), compared to our estimated value of 0.16. A potential explanation for this range of estimated values may be related to the variation in the number of litters analysed to estimate the heritability value. Both this study and that performed by Ogawa et al. (2019) analysed over 50,000 litters, whereas the study performed by Hermesch et al. (2005) analysed just over 2,000. Similarly, the study performed by Ogawa et al. (2019) and the current study had comparable heritability estimates for NBA at 0.12 and 0.14, respectively. Although, their estimated heritability for stillborn piglets was slightly lower at 0.08 (Ogawa et al., 2019)

compared to our estimate of 0.13. Interestingly, the study performed by Strange et al. (2013) estimated a heritability of 0.08 for sires and 0.24 for dams, which suggests that there may be an influence of sex, however the study analysed the litters of crossbred piglets (Duroc × Yorkshire), whereas both the current study and that performed by Ogawa et al. (2019) analysed purebred litters and excluded sires. These factors may influence the resulting heritability estimates which would explain the variation between results, however without further research and laboratory testing this cannot be confirmed. Despite the differences between our heritability estimates and those of previous studies, it appears that piglet mortality traits generally have a low heritability, therefore non-genetic effects must have a significant influence on their incidence.

This study showed that the incidence of haemophilia rapidly increased from around 2013 (Figure 12). This spike is believed to be associated with the consumption of wood shavings used as bedding in farrowing pens by sows, as indicated by the industry breeder, and this may contain harmful mycotoxins (Beynon, 2014). However, while studies indicate that mycotoxins in wood shavings can predispose piglets to pre-natal umbilical cord damage, navel bleeding following birth (Pig Progress, 2020) and abortions (Weaver et al., 1978), there are no findings that associate these toxins with haemophilia directly. This suggests that piglets impacted by the wood shavings were misdiagnosed as being haemophilic or other factors influenced the spike. It is important to note that the piggery identified haemophilic piglets as those with pale skin and haemorrhagic bruises along the body, while piglets affected by navel bleeding are pale but do not have haemorrhagic bruises (Pig Progress, 2020), therefore misdiagnosis is not likely. Additionally, incidence trend plots indicated a decrease in the total number of mummified piglets from around 2014 (Figure 12), at which time the breeder noted a feed change, suggesting a potential association between feed components and the incidence of mummification. However, a major decrease in total population size (loss of ~50%) between 2014-15 was

observed (Figure 13), which the breeder indicated to be an intentional removal of animals to be housed on a different property or sold to market. This drastic decrease in population size may explain the decrease in the number of piglets affected by various mortality causes, therefore the results and conclusions regarding mortality incidence are debatable.

Although we were able to analyse and plot the incidence of piglet mortality traits over time, it became apparent that determining whether they were caused by a recessive genetic mutation was difficult without DNA testing. The mortality causes we chose to perform quantitative genetic analysis on (i.e. haemophilia, splay leg and atresia ani), have all been reported to have underlying genetic causes according to their OMIA records (Online Mendelian Inheritance in Animals, 2020). Both splay leg and atresia ani have been observed to be inherited in a recessive pattern in pigs (Cassini et al., 2004; Matika et al., 2019). However, conflicting studies indicated that atresia ani is inherited in an autosomal dominant pattern in mice (Kluth et al., 1991) and is a complex multifactorial trait in humans (Winkler and Weinstein, 1970) which may make it more difficult to identify through a quantitative genetic pedigree-based analysis alone. Haemophilia was arguably the most difficult to analyse in terms of relation to a recessive trait, as various types of haemophilia have slightly different inheritance modes and there is currently no indication of which of these are present within the investigated population.

Although preliminary data analysis was unable to confirm the inheritance patterns of the mortality traits, it is possible that they are recessive. If so, this poses a major risk to the Australian pig population due to its closed nature and subsequent increased risk of inbreeding (Charlesworth and Willis, 2009; Kock et al., 2009). Pedigree analysis of the investigated population showed a clear increase in inbreeding for sows (Figure 4) and litters (Figure 5) over time, confirming the influence of a closed population. The litter breed coefficients indicated the substantial impact that inbreeding can have on litter size and piglet mortality, however the low heritability values for each of the investigated traits, suggests that while inbreeding does have

some impact on piglet mortality, other non-genetic factors may be more influential. Previous studies evaluated the impact of inbreeding on the Australian pig population, concluding that the genetic resources currently available within the population are sufficient at ensuring the maintenance of productivity and genetic diversity at acceptable levels (Hermesch et al., 2005; Bunter and Hermesch, 2017). Additionally, a focused study analysing the impact of inbreeding within a single pig breeding population found that the impact of inbreeding was relatively minimal, for example for every 1% increase in inbreeding, NBA reduced by 0.0278 piglets (Do et al., 2015). This is comparable to the litter inbreeding coefficient calculated for NBA in this study, of -2.233 for a completely inbred litter, therefore a 0.0223 decrease in NBA per 1% increase in inbreeding (-2.233/100). Similar to (Bunter and Hermesch, 2017), the study concluded that increasing inbreeding levels were manageable as long as appropriate measures were implemented to minimise inbreeding where possible (Do et al., 2015).

Study limitations

One of the major limitations of this study was the absence of laboratory work, specifically DNA testing to confirm results found through data analysis. This made it difficult to differentiate disease and piglet mortality caused by genetic abnormalities and those caused by environmental factors. Additionally, the provided dataset contained data for a single population, which resulted in some piglet mortality traits, such as haemophilia and splay leg having limited numbers of records which may have skewed the results. In order to efficiently analyse the piglet mortality causes we collapsed the 64 original causes into 14, which involved combining some into new categories and conserving others. By combining categories, there is the possibility that some trends were missed, while others may be misinterpreted, as similar phenotypes were combined in some instances. An issue that became evident through analysis was the recording of cross-fostering within the population. This is a common husbandry practice which involves the removal of piglets from their birth mother due to various factors, such as lack of adequate milk

or maternal skills, to be raised by another sow. We were informed that cross-fostering occurred within the population, but the dataset did not indicate which and how many piglets were cross-fostered as opposed to the biological offspring. While the breeder did an outstanding job recording other additional information, such as mortality cause, the absence of cross-fostering data made it difficult to analyse pedigrees and determine whether the mortality traits were inherited or a result of environmental factors.

Opportunities for future research

This study uncovered a range of areas suitable for future research. Using the prepared data and findings from this study, laboratory DNA analysis could be performed to further investigate the role of genetics in the incidence of various piglet mortality causes. This may require utilising OMIA to identify the mutations associated with the lethal phenotype. Once identified, a SNP panel could be produced to enable industry breeders to quickly and efficiently identify these lethal genetic variants on-farm and may be incorporated into breeding programs, to minimise piglet mortality and enhance welfare. Alternatively, further analysis can be performed on the data to gain a more thorough understanding of the effects of various factors such as parity, breed and environmental impacts on piglet mortality. This may include using Pearson correlation to identify and determine any genetic correlations between the EBVs of investigated traits as an indication of how selecting for a particular trait can impact the incidence of others. Additionally, pedigree visualisation and segregation analysis would be useful in determining the mode of inheritance of some traits. Some results suggested a potential breed difference in piglet mortality traits, particularly mummification being more prevalent in DD litters. These breed differences may be further investigated, and findings could be incorporated into breeding programs to further reduce piglet mortality. The same study design may also be repeated with more complex models, including additional traits, including incorporating the sire effect to a

greater extent. Ideally, the findings of this study will assist in developing tools and methods which can be applied to the Australian pig industry to reduce piglet mortality.

Conclusion

This study confirmed that data analysis was effective at determining that piglet mortality traits are prevalent within a sample of the Australian pig industry, however the role of recessive traits is still unclear. Without further pedigree analysis and DNA sampling of the investigated population, it is difficult to accurately identify inheritance patterns of mortality traits. Based on known information about inheritance patterns of various piglet mortality traits and estimated heritability values, it is suspected that a portion of the cases relating to piglet mortality are caused by underlying genetic conditions. This may also be exacerbated by increased levels of inbreeding within the Australian pig population due to its closed nature. In order to draw definitive conclusions and estimates of the role of genetics in piglet mortality, further research investigating the influence of all factors, both genetic and non-genetic is required. The findings may then be incorporated into current selective breeding programs to enhance the health, welfare and economic value of the Australian pig population.

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607 **Figures and Tables**

608 **Table 1: Additional fields added to analyse the phenes previously recorded in OMIA. Most are aimed towards identification of traits that play a role in piglet mortality and genetic**
609 **information useful for the development of a SNP panel to detect these on-farm.**

New field name	Contents
Description	Description of the phene in layman terms
Mendelian Trait (Y/N)	Whether the phene is a mendelian trait
Coat Colour (Y/N)	Whether the phene is related to coat colour
Disease (Y/N)	Whether the phene is associated with a disease
Disease affecting reproductive performance/piglet health? (Y/N/P)	Whether the phene impacts reproductive performance and/or piglet health
Impact on Reproductive Performance	Explanation of the previous field if Y or P
Notes for Frank	Additional notes for Frank about changes made or questions
Reference genome	Genome where the mutation location has been found
Start	Starting position of the mutated sequence within the gene
End location	Ending position of the mutated sequence within the gene
Reference allele	Allele without mutation
Variant allele	Allele with mutation
Strand	Can be positive or negative
Marker type	Type of mutation
Sequence	Genetic sequence showing the mutated region
NCBI ID	Code which links the mutation to related papers on NCBI

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Table 2: Incidence of the original piglet mortality causes included within the provided dataset compared to the new, collapsed categories and their incidence within the population over the entire duration of data collection (1995-2019).

611

Original mortality causes	Incidence	New mortality cause categories	Incidence
Broken back	13	All_accidental	98
Broken leg	29	Anaemia	2642
Fractured bones	29	Atresia ani	1496
Anaemia	2642	Deformed	5487
Atresia ani	1496	Haemophilia	180
Deformed	5487	Ill_thrift	5292
Bleeding	10	Laid_on	42212
Haemophiliac	170	Other	2043
Ill thrift	5292	Runt	13322
Laid on	42212	Savaged	3961
Accident	5	Scours	3863
Accidental	22	Splay_leg	5970
Boar pen death	32	Starvation	8641
Drowned	129	Unviable	2453
Injury/trauma	97		
Stepped on	24		
Chilling	413		
Arthritis	21		
Blood poisoning	14		
Campylobacter	2		
E coli.	1		
Ear infection	13		
Greasy pig	106		
Kidney infection	1		
Meningitis	9		
Pericarditis	4		
Pneumonia	33		
Septicemia	3		
Strept	5		

Oedema	24
Abscess	1
Burst intestine	9
Congential	52
Dehydrated	10
Destroyed	248
Haemorrhage	1
Heart disease	1
Heart failure	2
Heat stress	2
Joint ill	12
Lame	29
Low viability	1
Melanoma	3
Omphalocele	8
Other disease	3
Ruptured spleen	1
Ruptures	23
Sick pen	1
Stress	11
Twisted bowel	28
Twisted liver	1
Unknown	180
Premature	364
Anal prolapse	21
Prolapse	14
C-section	7
Retained piglet	79
Runt	13322
Savaged	3961
Scours	3863
Splay leg	5970
Dry sow	309
Starvation	8332
Unviable	2453

Table 3: Pig phenes recorded within the OMIA database that have been identified as Mendelian traits. Some are associated with the manifestation of disease in pigs, while others are associated with coat colour. Several phenes had changes made on OMIA as indicated.

612

OMIA ID	Phene	Disease? (Y/N)	Updates in OMIA
OMIA 000209-9823	Coat colour, dominant white	N	Chr: 8
OMIA 002232-9823	Myopathy, congenital, SPTBN4-related	Y	
OMIA 001745-9823	Coat colour, white belt, KIT-related	N	
OMIA 001745-9823	Coat colour, white belt, KIT-related	N	
OMIA 001128-9823	Pale soft exudative meat	N	
OMIA 002210-9823	Hypothyroidism, congenital, DUOX2-related	Y	
OMIA 001058-9823	Von Willebrand disease III	Y	
OMIA 001752-9823	Resistance to porcine reproductive and respiratory syndrome (PRRS) virus	Y	
OMIA 002180-9823	Abortion due to haplotype DU1	Y	
OMIA 002181-9823	Abortion due to haplotype LA1	Y	
OMIA 002182-9823	Abortion due to haplotype LA2	Y	
OMIA 002183-9823	Abortion due to haplotype LA3	Y	
OMIA 002178-9823	Abortion, BBS9-related	Y	
OMIA 001579-9823	Ear size	N	
OMIA 002161-9823	Leg weakness, MSTN-related	Y	
OMIA 002157-9823	Ectodermal dysplasia-9	Y	Chr: 5 Ref seq: Sscrofa10.2 g.: g.70193783C>T Verbal description of mutation Ref genome: Sscrofa11.1 Start: 69841413 End: 69841413 Ref allele: C Variant allele: T Strand: PLUS
OMIA 000499-9823	Hypercholesterolaemia	Y	
OMIA 001436-9823	Non-shivering thermiogenesis, absence of	Y	Ref seq: Sscrofa11.1 Chr: 8
OMIA 001401-9823	Waardenburg syndrome, type 2A	N	Ref seq: Sscrofa10.2

OMIA 001199-9823	Coat colour, extension	N	Chr: 6 Ref seq: Sscrofa10.2
OMIA 000683-9823	Muscular hypertrophy (double muscling)	Y	Chr:15 g.: g.105740019A>G
OMIA 001089-9823	Blood group system ABO	N	Verbal description of mutation Ref seq: Sscrofa11.1
OMIA 001362-9823	Coat colour, blond	N	
OMIA 001986-9823	Severe combined immunodeficiency disease, autosomal, T cell-negative, B cell-negative, NK cell-positive, with sensitivity to ionizing radiation	Y	Verbal description of mutation Ref genome: Sscrofa11.1 Start: 46845535 End: 46845535 Ref allele: G Variant allele: A Strand: PLUS
OMIA 001986-9823	Severe combined immunodeficiency disease, autosomal, T cell-negative, B cell-negative, NK cell-positive, with sensitivity to ionizing radiation	Y	Verbal description of mutation Ref genome: Sscrofa11.1 Start: 46851262 End: 46851262 Ref allele: G Variant allele: A Strand: PLUS
OMIA 001952-9823	Microtia	Y	Ref seq: Sscrofa10.2 Chr: 18 g.: g.50111252delinsTC p.: p. (Leu151fs) Verbal description of mutation Ref genome: Sscrofa11.1 Start: 45478109 End: 45478110 Ref allele: G Variant allele: TC Strand: PLUS
OMIA 001085-9823	Meat quality (Rendement Napole)	Y	Ref seq: Sscrofa10.2 Chr: 15 Allele: RN -

			g.: g.133803828A>G p.: p.I249V Verbal description of mutation
			Allele: RN – p.: p.R250Q
OMIA 001085-9823	Meat quality (Rendement Napole)	Y	Verbal description of mutation Ref genome: Sscrofa11.1 Start: 120863533 Strand: MINUS
			Verbal description of mutation Ref genome: Sscrofa11.1 Start: 47368496
OMIA 000621-9823	Malignant hyperthermia	Y	End: 47368496 Ref allele: C Variant allele: T Strand: PLUS
			Ref seq: Sscrofa9.2 Chr: X
OMIA 001685-9823	Stress syndrome	Y	Allele: RN -
OMIA 001743-9823	Coat colour, patch	N	g.: g.27387827T>C Chr: 8 Ref seq: Sscrofa10.2
OMIA 001200-9823	Tremor, high-frequency	Y	Chr: 7 Allele: RN -
			g.: g.81070838_81070839insGGCGGG Ref seq: Sscrofa11.1
OMIA 000636-9823	Membranoproliferative glomerulonephritis type II	Y	g.: g.2553907T>G
OMIA 000837-9823	Vitamin D-deficiency rickets, type I	Y	Chr: 5
OMIA 000837-9823	Vitamin D-deficiency rickets, type I	Y	Chr: 5 Ref seq: Sscrofa11.1 Chr: 1
OMIA 001718-9823	Dwarfism, Schmid metaphyseal chondrodysplasia	Y	g.: g.81767089G>A Ref genome: Sscrofa11.1 Start: 81767089 End: 81767089

			Ref allele: G Variant allele: A Strand: MINUS
OMIA 000862-9823	Resistance to oedema disease (F18 receptor)	N	Chr: 6
OMIA 001249-9823	Coat colour, brown	N	Ref seq: Sscrofa7.0 Chr: 1
OMIA 001216-9823	Coat colour, roan	N	Chr: 8
OMIA 001673-9823	Spermatogenic arrest	Y	Chr: 12
OMIA 001334-9823	Sperm, short tail	Y	Chr: 16
			Verbal description of mutation Ref genome: Sscrofa11.1 Start: 31281804
OMIA 001579-9823	Large floppy ears	N	End: 31281804
			Ref allele: G Variant allele: A Strand: PLUS
OMIA 001199-9823	Red	N	Breeds: D Chr: 6
OMIA 001199-9823	Red	N	Chr: 6 p.: p. A240T
OMIA 001199-9823	Dominant black	N	Chr: 6
OMIA 001199-9823	Dominant black	N	Chr: 6
OMIA 001199-9823	Recessive white	N	Chr: 6
OMIA 001199-9823	Coat colour, black spotting on red or white background	N	Chr: 16

Table 4: Pedigree completeness index values for all three main breeds within the population as estimated by the reports generated by the pedigree analysis software program, POPREP.

Breed	Pedigree completeness index (%)					
	PCI1	PCI2	PCI3	PCI4	PCI5	PCI6
Duroc	100	100	100	100	99.9	99.2
Duroc[1]	-[2]	100	99	98	96	94
Landrace	100	100	100	100	100	99.9
Landrace[1]	-	100	100	98	97	95
Large White	100	100	100	100	100	99.9

[1]Data from study outlined in Groeneveld et al. (2009) on South African populations of Duroc and Landrace pigs.
[2]Data not included in Groeneveld et al. (2009) study.
PCI 1-6 = PCI for pedigree depths of 2 to 6 generations

Table 5: Effective population sizes calculated using various methods as performed by the pedigree analysis software program, POPREP.

Breed	Method					
	***Ne*-Cens**	***Ne*-ΔFp**	***Ne*-ΔF_g**	***Ne*-Coan**	***Ne*-Ln**	***Ne*-Ecg**
Duroc	141	52	-66	58	-28	65
Duroc[1]	247	40	119	99	219	143
Landrace	143	36	37	31	34	78
Landrace[1]	546	50	13	44	222	102
Large White	137	37	35	28	37	59

[1]Data from study outlined in Groeneveld et al. (2009) on South African populations of Duroc and Landrace pigs.

Table 6: Total number of piglets affected by recorded mortality causes between 1995-2019. The proportion of piglets affected by a particular mortality cause is indicated as a percentage of the total number of piglets born alive (TBA) within the same period.

Variable	Total number of piglets died due to death cause					
	Breed					
	Duroc (68587 TBA)		Landrace (478160 TBA)		Large White (345844 TBA)	
	Total affected	%	Total affected	%	Total affected	%
Haemophilia[1]	1	0.001	156	0.03	33	0.01
All accidental	9	0.01	64	0.01	33	0.01
Anaemia	92	0.13	1619	0.34	1295	0.37
Atresia ani[1]	82	0.12	874	0.18	652	0.19
Splay leg[1]	636	0.93	4060	0.85	2538	0.73
Deformed	506	0.74	3509	0.73	2025	0.59
Ill thrift	589	0.86	2794	0.58	1770	0.51
Laid on	4398	6.41	23181	4.85	18577	5.37
Other	255	0.37	930	0.19	936	0.27
Runt	1131	1.65	7848	1.64	6369	1.84
Savaged	325	0.47	1066	0.22	2504	0.72
Scours	331	0.48	1828	0.38	1802	0.52
Starvation	1113	1.62	5256	1.10	3757	1.09
Unviable	179	0.26	993	0.21	1034	0.30

[1]Mortality causes identified to have known underlying genetic causes

Table 7: Summary statistics for piglet mortality traits chosen for complex analysis as calculated in R version 4.0.

Traits	Number of affected litters	Minimum	Maximum	Average
Mummified	24463	0	18	0.4942
Stillborn	35466	0	17	0.8517
Haemophilia	60	0	17	0.00247
Splay leg	4080	0	20	0.0941
Atresia ani	1337	0	7	0.0209

633 **Table 8: Litter breed and parity predictions based on mixed models created in R version 4.0 for piglet mortality traits.**

[1]Linear trait: values represent an actual number of piglets. [2]Binary trait: values represent a probability. [3]Count trait: values represent an actual number of piglets.

Fixed effects		Response variables				
		Mummified[2]	Stillborn[3]	Haemophilia[2]	Atresia ani[2]	Splay leg[2]
		Prediction ± SE				
Litter breed	DD	0.43 ± 0.032	0.99 ± 0.115	$2.66e^{-05}$ ± $1.67e^{-05}$	0.0105 ± 0.0034	0.11 ± 0.026
	RR	0.33 ± 0.019	0.94 ± 0.072	$8.14e^{-05}$ ± $3.44e^{-05}$	0.0125 ± 0.0027	0.04 ± 0.008
	RW	0.29 ± 0.018	0.77 ± 0.059	$1.10e^{-04}$ ± $5.24e^{-05}$	0.0200 ± 0.0044	0.07 ± 0.013
	WR	0.28 ± 0.019	0.90 ± 0.075	$4.84e^{-05}$ ± $2.53e^{-05}$	0.0293 ± 0.0068	0.08 ± 0.015
	WW	0.28 ± 0.019	0.90 ± 0.076	$4.37e^{-05}$ ± $2.26e^{-05}$	0.0118 ± 0.0028	0.04 ± 0.007
Parity	1	0.26 ± 0.013	0.47 ± 0.027	$1.09e^{-05}$ ± $5.06e^{-06}$	0.0012 ± 0.00026	0.003 ± $6.80e^{-04}$
	2	0.26 ± 0.013	0.46 ± 0.027	$4.06e^{-06}$ ± $2.28e^{-06}$	0.0030 ± 0.00061	0.01 ± 0.001
	3	0.31 ± 0.014	0.62 ± 0.035	$3.54e^{-05}$ ± $1.56e^{-06}$	0.0050 ± 0.00097	0.01 ± 0.002
	4	0.34 ± 0.015	0.78 ± 0.044	$3.52e^{-05}$ ± $1.61e^{-05}$	0.0082 ± 0.00156	0.03 ± 0.004
	5	0.35 ± 0.015	0.91 ± 0.051	$8.85e^{-05}$ ± $3.83e^{-05}$	0.0145 ± 0.00267	0.04 ± 0.006
	6	0.34 ± 0.015	1.04 ± 0.059	$1.59e^{-04}$ ± $6.77e^{-05}$	0.0230 ± 0.00418	0.09 ± 0.013
	7	0.34 ± 0.015	1.18 ± 0.066	$1.73e^{-04}$ ± $7.71e^{-05}$	0.0346 ± 0.00628	0.17 ± 0.023
	8	0.33 ± 0.016	1.28 ± 0.073	$1.28e^{-04}$ ± $6.44e^{-05}$	0.0516 ± 0.00936	0.26 ± 0.033
	9	0.36 ± 0.019	1.39 ± 0.081	$1.44e^{-04}$ ± $7.69e^{-05}$	0.0702 ± 0.01304	0.35 ± 0.040
	10+	0.32 ± 0.018	1.66 ± 0.097	$1.03e^{-04}$ ± $6.01e^{-05}$	0.1095 ± 0.01950	0.57 ± 0.045

634

635 **Table 9: Statistical significance of fixed effects on response variables in mixed models created for selected piglet mortality traits.**

Variable	Trait	p-value	Statistical significance
Litter breed	Litter size	0.107730	✗
	Number born alive	0.00000	✓
	Mummified	0.00000	✓
	Stillborn	0.00000	✓
	Haemophilia	0.60910	✗
	Atresia ani	0.000000	✓
	Splay leg	0.000000	✓
Parity	Litter size	0.000000	✓
	Number born alive	0.000000	✓
	Mummified	0.000000	✓
	Stillborn	0.000000	✓
	Haemophilia	0.00000	✓
	Atresia ani	0.000000	✓
	Splay leg	0.000000	✓
Litter inbreeding	Litter size	0.000344	✓
	Number born alive	$1.65446e^{-06}$	✓
	Mummified	0.277643	✗
	Stillborn	0.00084689	✓
	Haemophilia	0.84502	✗
	Atresia ani	0.076281	✓
	Splay leg	0.032831	✓
Contemporary group	Litter size	0.000000	✓
	Number born alive	0.000000	✓
	Mummified	0.000000	✓
	Stillborn	0.000000	✓
	Haemophilia	1.00000	✗
	Atresia ani	0.034010	✓
	Splay leg	0.000000	✓

636 **Table 10: Heritability and repeatability estimates calculated for each selected mortality trait, indicating the role of genetics in the prevalence of the traits.**

Trait	h^2 ± SE	Repeatability ± SE
Litter size	0.160 ± 0.010	0.283 ± 0.012
Number born alive	0.143 ± 0.009	0.254 ± 0.011
Mummified	0.068 ± 0.006	0.114 ± 0.004
Stillborn	0.126 ± 0.008	0.245 ± 0.004
Haemophilia	0.101 ± 0.199	0.239 ± 0.135
Splay leg	0.182 ± 0.020	0.410 ± 0.011
Atresia ani	0.267 ± 0.032	0.385 ± 0.016

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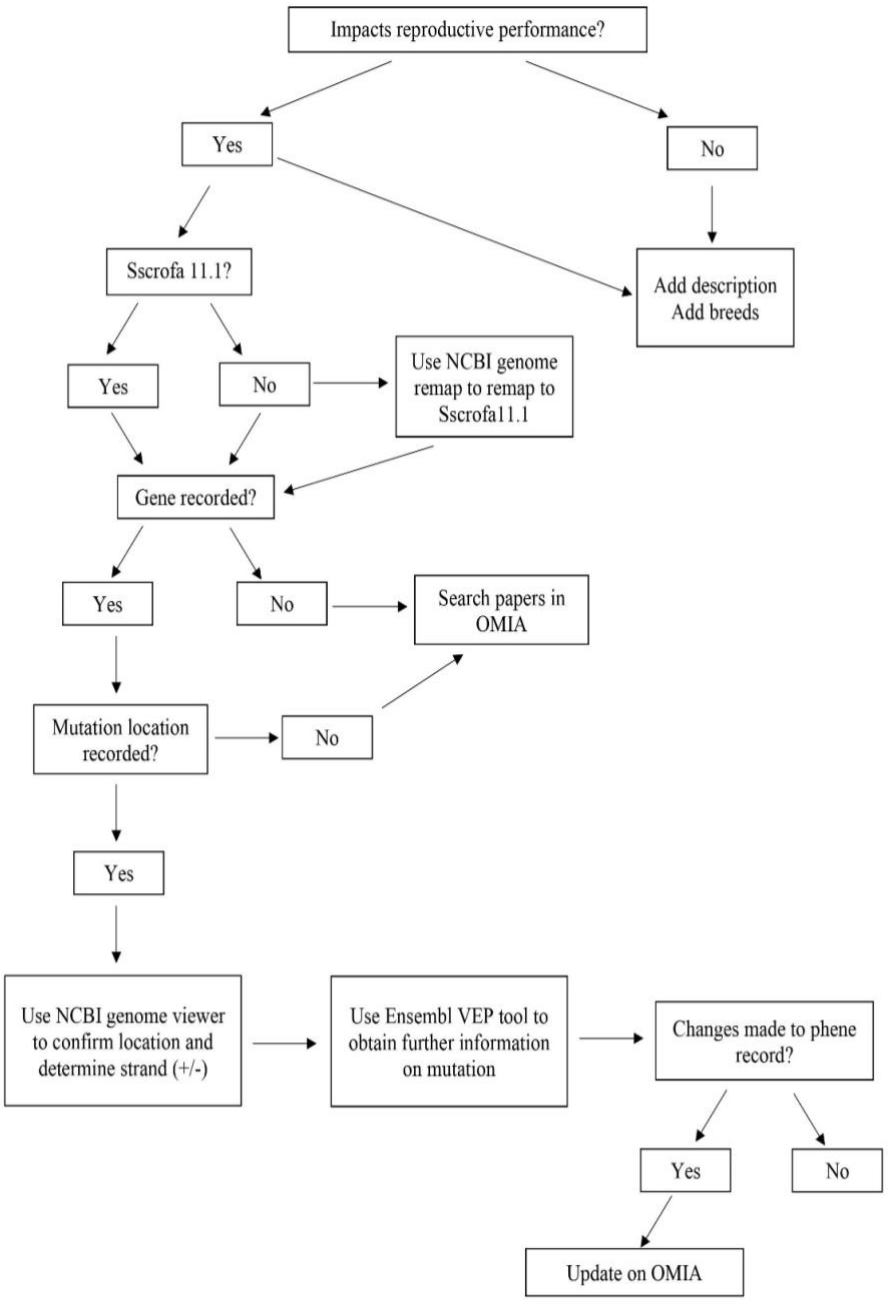

Figure 1: Decision tree for OMIA database analysis and curation created for this project.

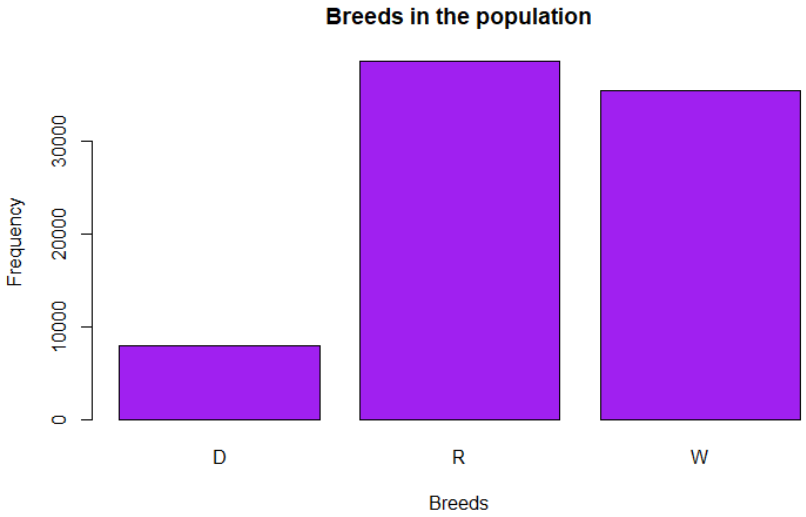

Figure 2: Sow and sire breed distribution within the analysed population between 1995-2019.

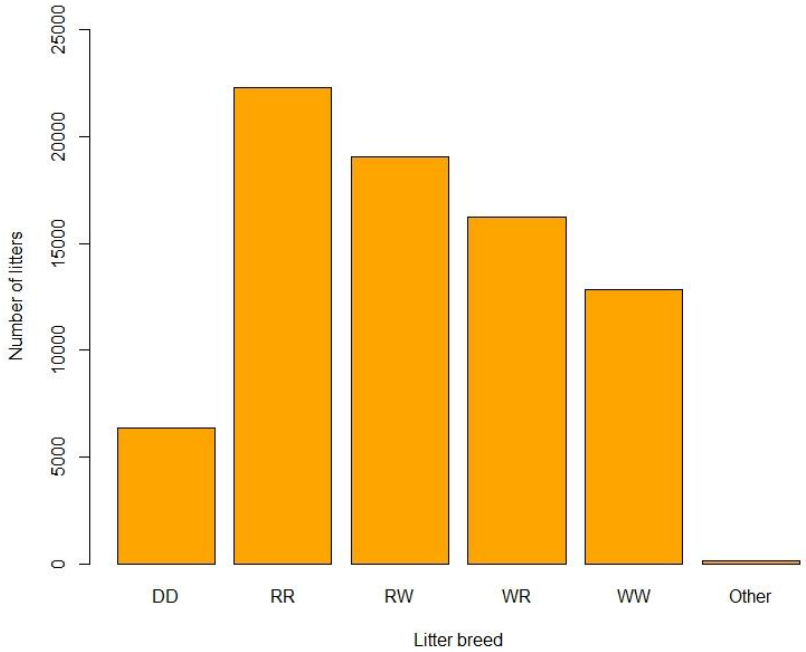

Figure 3: Litter breed distribution within the analysed population between 1995-2019.
DD - purebred Duroc; RR - purebred Landrace; RW: - Landrace sow, Large White sire; WR - Large White sow, Landrace sire; WW – purebred Large white; Other – various sow-sire combinations with very small numbers of litters over the data collection period.

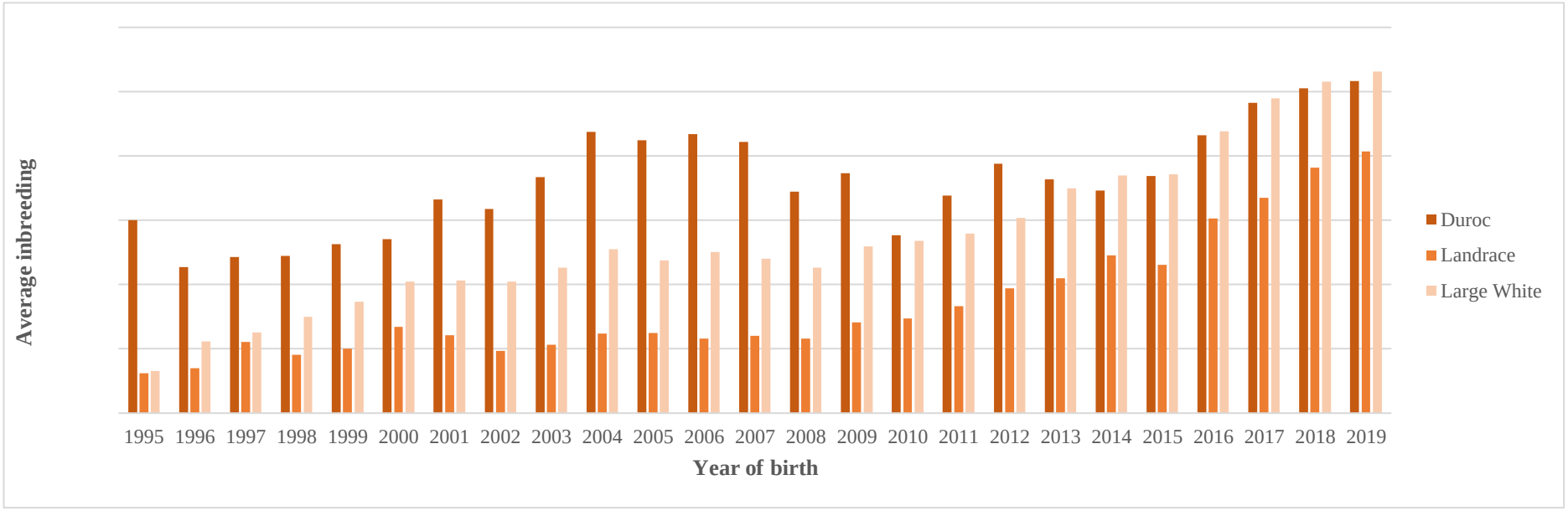

Figure 4: Average inbreeding value for sows of all three breeds between 1995-2019.

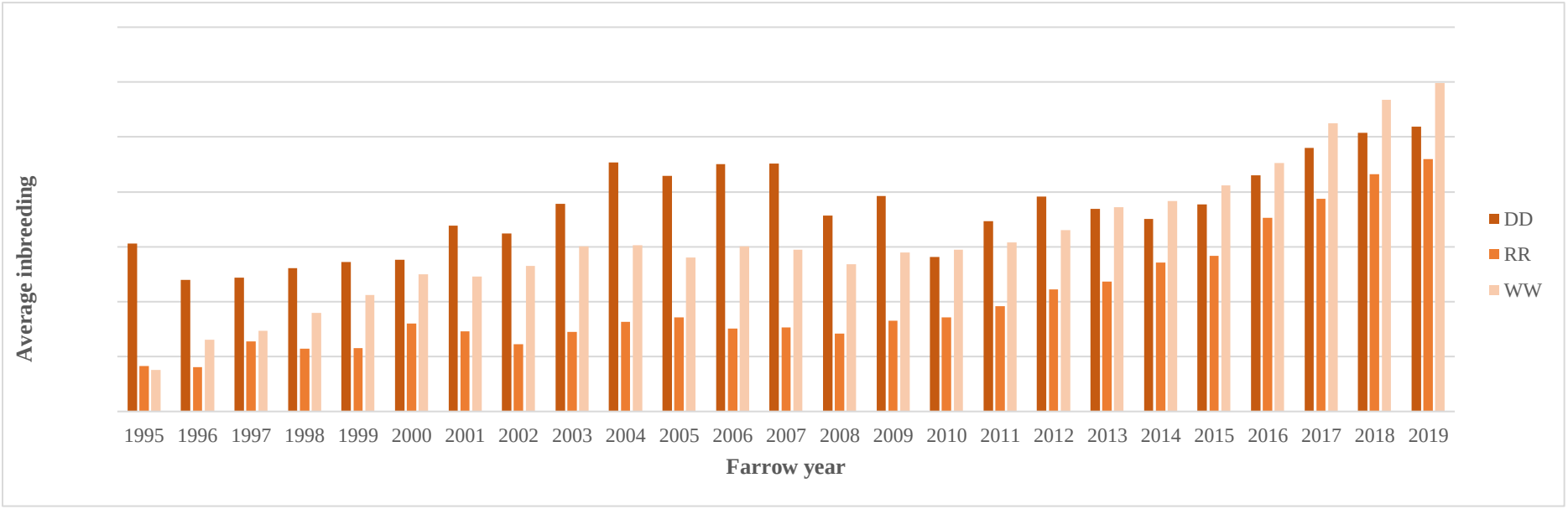

Figure 5: Average inbreeding values for purebred litters between 1995-2019.

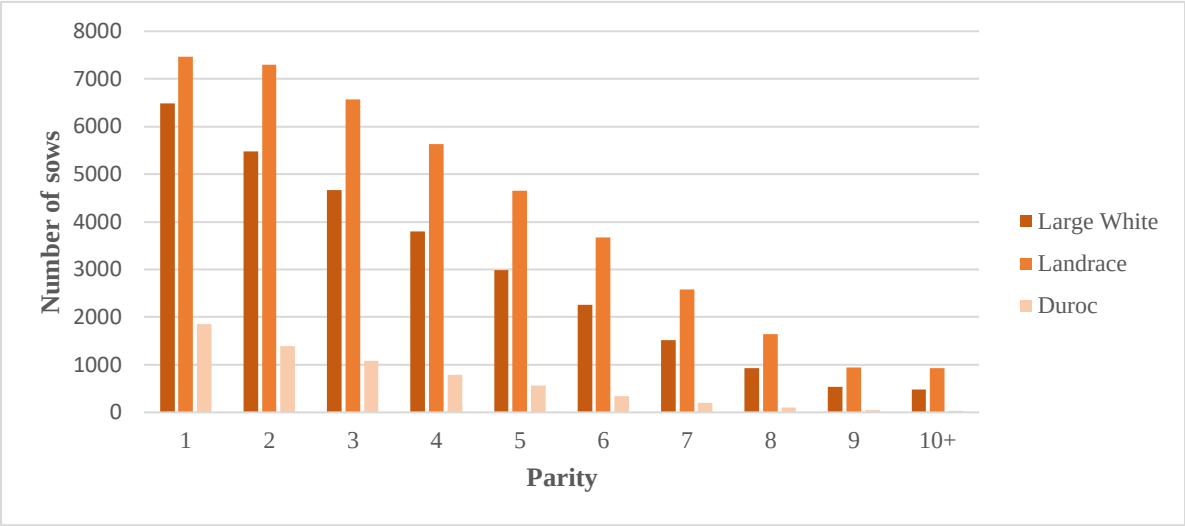

Figure 6: Total number of sows from each breed in each parity level between 1995-2019.

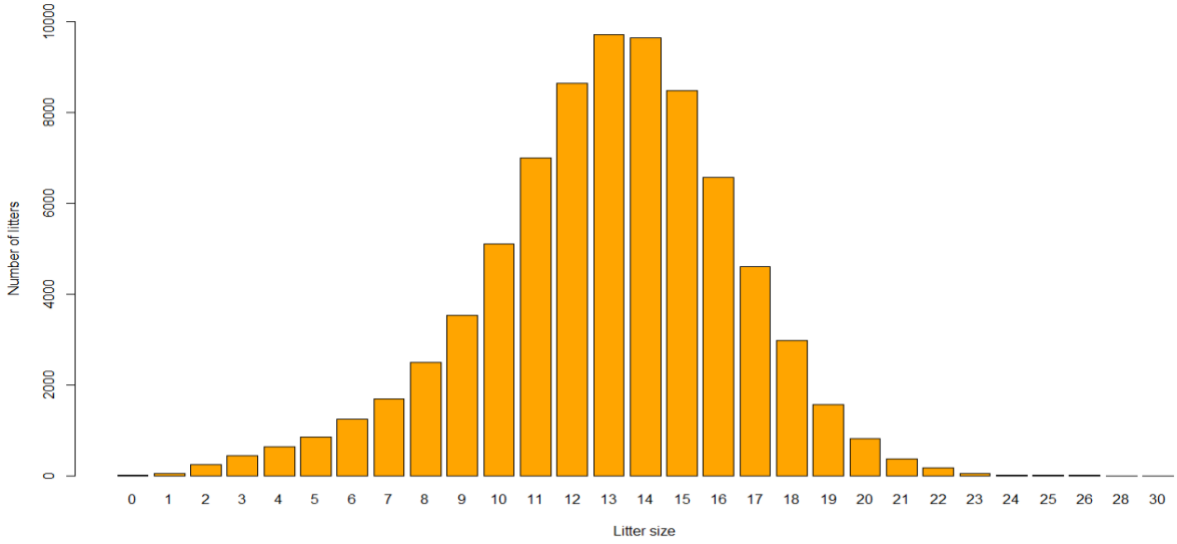

Figure 7: Total number of sows with litters of varying litter size between 1995-2019.

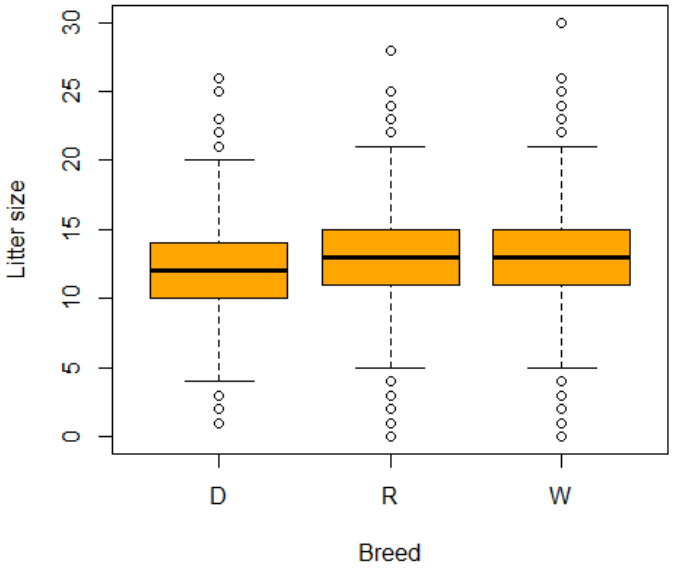

Figure 8: Distribution of litter size for all three sow breeds within the population between 1995-2019.

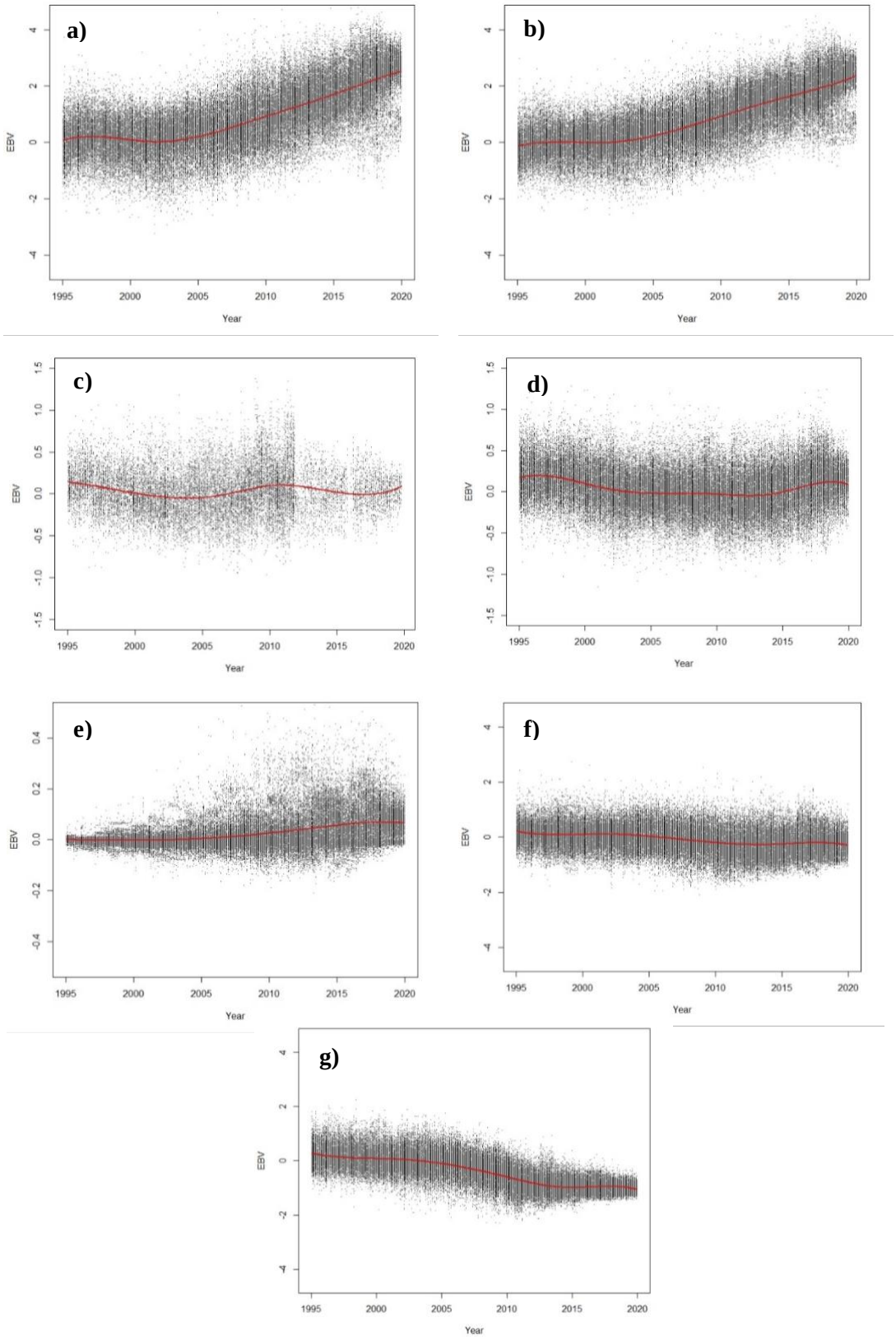

Figure 9: Model plots of individual animal EBVs for all response variables indicating the influence of genetic effects over time.

a) litter size, b) number born alive, c) mummified, d) stillborn, e) haemophilia, f) atresia ani, g) splay leg

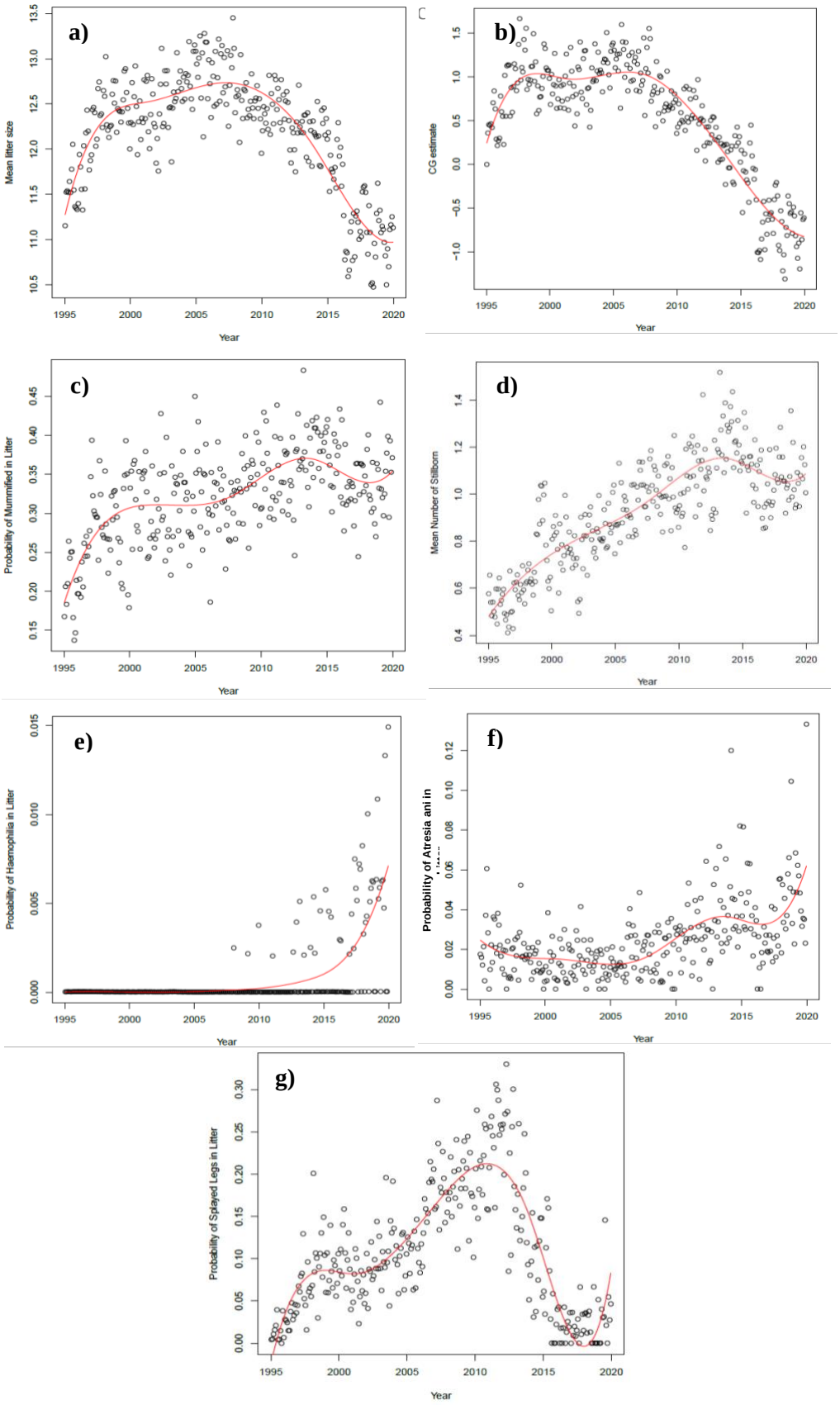

Figure 10: Model plots of contemporary groups estimates for response variables indicating the influence of non-genetic effects over time.

a) means of litter size, b) estimates of number born alive, c) probabilities of mummified, d) means of stillborn, e) probabilities of haemophilia, f) probabilities of atresia ani, g) probabilities of splay leg

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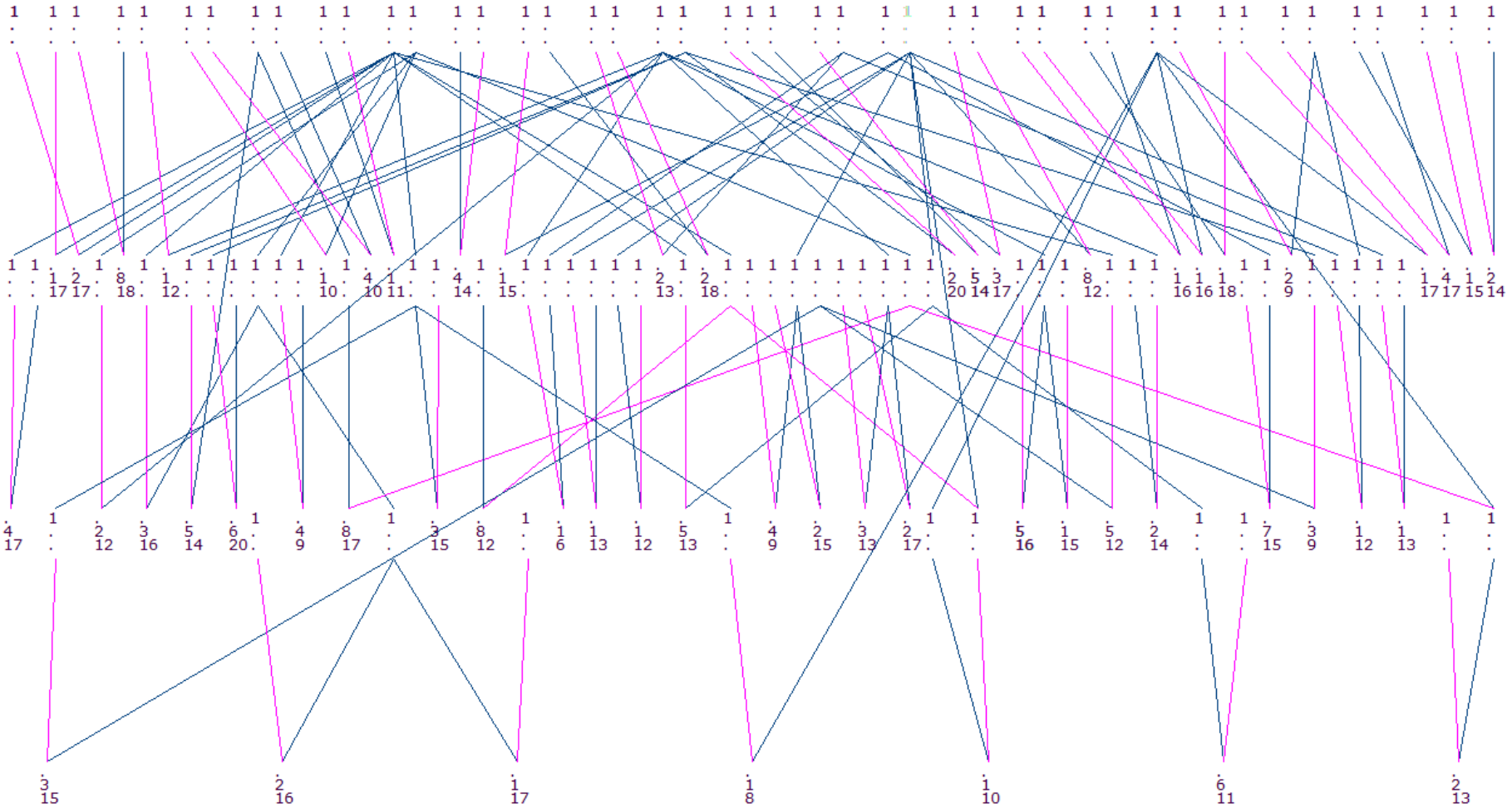

Figure 11: Pedigree showing the litters affected by haemophilia using Pedigree Viewer.
The bottom number is the total litter size, while the top number indicates the number of affected piglets. Maternal lines are represented by purple lines, while paternal lines are blue.

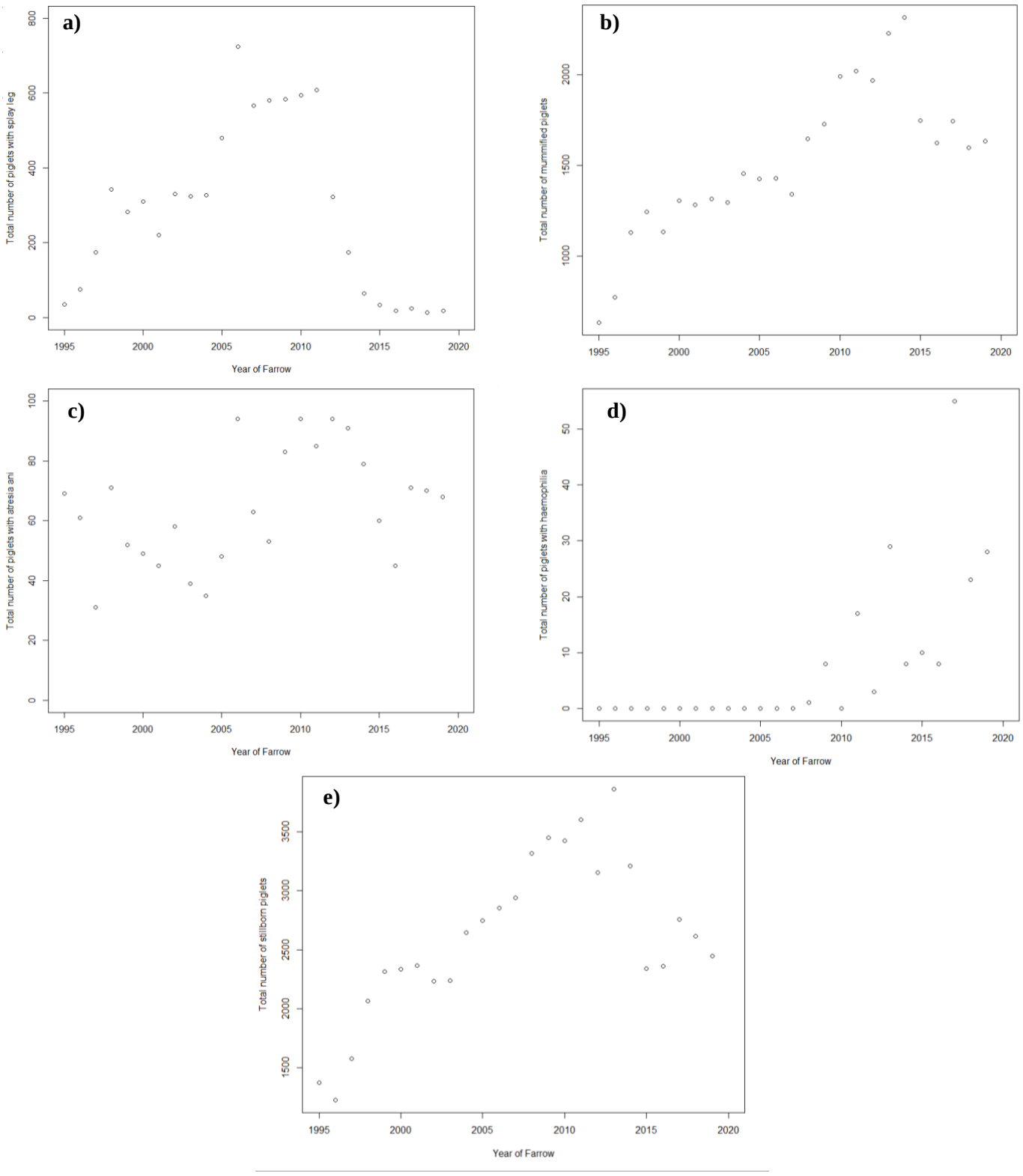

Figure 12: Total incidence of mortality traits over time for all litter breeds combined.

a) probability of splay leg, b) probability of mummified, c) probabilities of mummified, d) probability of haemophilia, e) number of stillborn

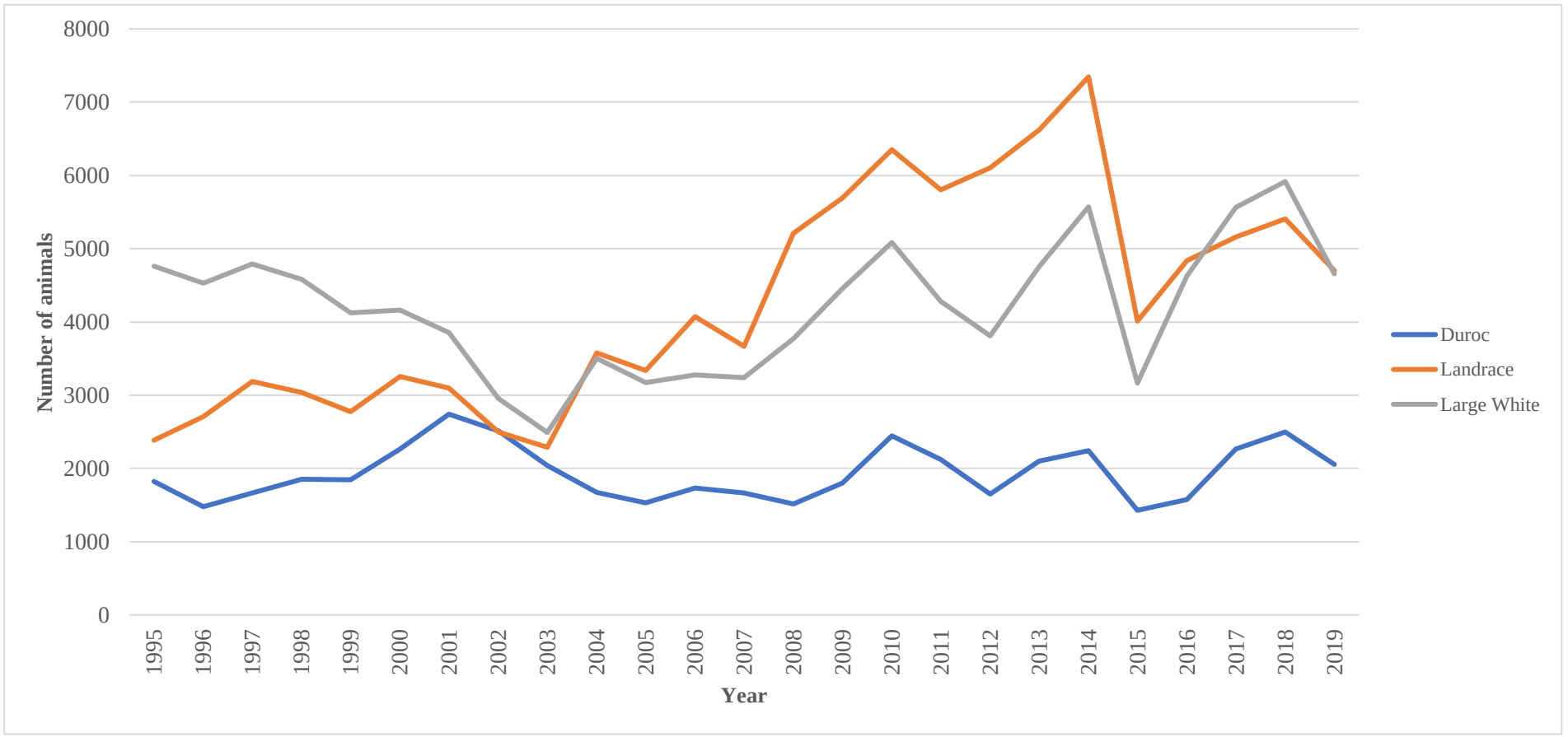

Figure 13: Total population size over time for all three main breeds in the population.