2B-105: GENETIC PARAMETERS FOR HEALTH, SURVIVAL, IMMUNE COMPETENCE, POST-WEANING GROWTH AND DISEASE RESILIENCE OF PIGS

Report prepared for the Co-operative Research Centre for High Integrity Australian Pork

By

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Executive Summary

Resilience is the ability to recover quickly from challenges. Measurements recorded shortly after weaning provide information about resilience of weaner pigs to cope with the weaning process. This approach was used to estimate genetic parameters for multiple immune and haematological traits recorded in weaner pigs along with multiple weight measurements post weaning for genetic improvement of disease resilience.

Weight measurements were collected on 2,025 Large White pigs from January 2013 to November 2014. A proportion of these pigs (813 pigs) also had total and differential white blood cells, haematological traits including haemoglobin, immunoglobulins (IgA, IgG, IgM) and haptoglobin recorded. Genetic parameters were estimated for these immune and haematological traits as well as multiple growth traits using an animal model.

Immune and haematological traits had moderate to high heritabilities. Further, multiple immune and haematological traits had significant genetic correlations with growth traits. Average growth of a group of pigs was lower for groups of pigs that required more medication. This finding confirms the concept of growth as a health indicator. It is recommended to measure white blood cell counts and haemoglobin as well as post-weaning growth in weaner pigs. Haptoglobin and immunoglobulin (Ig) G and IgM may be recorded if cost-effective measurement techniques are available.

A simple score about whether a pig was medicated or not was lowly heritable in this high-health herd which offers new opportunities for genetic improvement of health of pigs. The heritability was not significantly affected by the approach to account for non-medicated pigs which provides extra flexibility for the definition of this trait for genetic evaluations. Medication of pigs has economic and welfare costs. The economic value of medication score is based on the cost of medication and loss in productivity due to the disease incidence. Medication score should be incorporated in breeding objectives of pig breeding programs.

Disease resilience is a two-dimensional trait which requires definition of environmental challenges. In this regard, it is important to separate other environmental, non-infection stressors from infection challenges. Methodology was developed by Sarita Guy as part of her PhD thesis to obtain finer descriptions of possible infection challenges by removing climatic effects from environmental descriptors using performance data recorded on farms.

An on-farm measure of haemoglobin was genetically the same trait as haemoglobin measured in the laboratory. However, the on-farm measure of haemoglobin had a lower heritability than the laboratory measure due to larger residual variation which indicates measurement errors for the on-farm measure. On farm-measures of immune and haematological traits should be explored if it is not possible to obtain measurements from the laboratory.

Recording immune, haematological and growth traits in weaner pigs at five weeks of age has practical advantages for the application and adoption of these traits. At this age, weaners may also be recorded for juvenile IGF1 which is an early selection criterion for genetic improvement of efficient leant meat growth. Outcomes from this study warrant recording of white blood cell counts, haemoglobin, post-weaning growth and possibly haptoglobin and IgG and IgM in weaner pigs at 35 days in other pig populations that have information about feed intake, juvenile IGF1 or post-weaning survival available.

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1. Introduction

Disease resilience has been defined as the ability of a host to maintain a reasonable level of productivity when challenged by infection (Albers et al., 1987). This approach focuses on reducing the effects of infection rather than reducing the infection itself following the earlier work by Clunies-Ross (1932) who had made the distinction between 'resistance to infection' and 'resistance to the effects of infection'. Hermesch (2014) outlined requirements to define disease resilience in pigs highlighting the fact that disease resilience is a two-dimensional trait that requires information about infection challenge experienced by pigs and their performance when challenged by infection. Performance in this context may be described by measures of productivity or alternatively may relate to traits describing health status and survival of pigs.

Infection challenge experienced by pigs on farms is not only affected by the amount of potentially interacting pathogens and their virulence but also by environmental factors such as air quality, temperature and humidity (Collins, 2014). It was suggested that monitoring air quality may provide a better indicator of pig health and growth than monitoring individual pathogen loads because air quality affects growth and health of pigs. Air quality measures have been collected in the pilot study on disease resilience (Hermesch et al. 2015) in weaner, porker and finisher sheds. Considerable variation has been observed between pens within sheds in multiple air quality measures which indicates that the infection challenge for pigs differs between groups of pigs housed in different areas of a shed. Disease resilience relies on an accurate measure of infection challenge and information about air quality and average performance of pigs offer opportunities for quantifying disease challenge better. For example, Hermesch (2014) pointed out that mean levels of specific immune traits for groups of pigs provide additional information about infection challenge.

Immune competence is under genetic control in pigs (Clapperton et al., 2008, 2009; Henryon et al., 2001; Flori et al., 2011). Recently, Hine et al. (2014) reviewed selection strategies for immune competence. The authors concluded that selection for resistance to a specific disease carries the potential risk of increasing the susceptibility to other diseases. This risk is reduced by selection for general immune responsiveness as an alternative or complementary selection strategy to selection for a specific disease. Differential blood counts describe global and innate immunity (Flori et al., 2011) which have been recorded in the pilot study on disease resilience (2B-103) along with information about the acute phase protein haptoglobin and immunoglobulins. Haptoglobin has been recommended as an important marker of heard health in pigs (Petersen et al., 2004) while immunoglobulins may be used to quantify the effect of the dam on progeny health better (Collins, 2014). Maternal genetic effects for growth are an important trait for pig breeding (Amer et al., 2014) which should be used more in selection programs (Hermesch et al., 2014). Using immunoglobulins in breeding programs as selection criteria may provide further avenues to improve growth and health of pigs.

Unexpected and unfavourable consequences of selection for immune response can be avoided by focusing directly on health, survival and low incidence of clinical and subclinical diseases. Further, growth is regarded as a proxy for health status of animals. In pigs, Henryon et al. (2001) found genetic variation for clinical and sub-clinical disease in pigs which were based on veterinary records from the central test station in Denmark. In addition, genetic variation was found for a simple disease incidence score based on routine veterinary observations on non-specific digestive disorders in a commercial rabbit population (Garreau et al., 2008). This disease score has subsequently been implemented in commercial rabbit breeding programs in France (H. Garreau, personal communication). These results are noteworthy because simple disease incidence scores were derived from routine veterinary records and genetic variation for disease scores was found in the good health and housing conditions of a central test station in Denmark and nucleus farms for rabbits in France. Information about repeated weight measurements, disease incidence and survival of pigs has been collected in the pilot study for disease resilience. Genetic analyses of these measurements is required to identify simple and cost effective selection criteria to improve health status of pigs.

In summary, concepts to define disease resilience in pigs have been defined and data about air quality, health status, survival and growth have been collected along with various immune parameters. The study aimed to estimate genetic parameters for the large number of traits available, support a PhD candidate in defining genetic models for disease resilience, and provide guidelines to Australian breeding companies for genetic improvement of disease resilience, health and survival of pigs.

2. Methodology

2.1. Estimation of genetic parameters

2.1.1. Overview of data

Weight measurements were collected on 2,025 Large White pigs from January 2013 to November 2014 at the piggery of the University of Queensland in Gatton, Australia. A proportion of these pigs (813 pigs) also had total and differential white blood cells, haematological traits including haemoglobin, immunoglobulins (IgA, IgG, IgM)and haptoglobin recorded. Procedures to record traits were described in detail by Hermesch et al. (2015). Briefly, the weight of pigs was measured at weaning, at five weeks and at 10, 13 and 17 weeks of age.). Haemoglobin was recorded on farm (HbF) at five weeks with the HemoCue Hb 201+ analyser (HemoCue® 2012). Blood samples of pigs were collected at five weeks (at 36.4 ± 3.69 days) into vacutainers with anticoagulants (EDTA) and stored at 1 to 4o C until haematology analyses 0.72 ± 1.04 days later using a calibrated automated haematology analyser (Cel-Dyn® 3700, www.abbottdiagnostics.com). The haemogram included total white blood cells, red blood cell count, haemoglobin, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration and packed cell volume. The complete blood count was differentiated into the five main types (neutrophils, eosinophils, basophils, lymphocytes and monocytes).

These data were complemented by information from the general database that has been maintained since 1995 at the herd for genetic improvement purposes. This information included pedigree of pigs as well as reproductive records of sows and carcase information for growing pigs. Further, medication of pigs was part of the routine recording procedure at the piggery and medication records were available since 2011.

2.1.2. Statistical analyses

Outliers exceeding four standard deviations from the mean of each trait were eliminated from genetic analyses. The distribution of traits was evaluated with the Univariate procedure (SAS, 2014) and traits with a skewness and kurtosis number of smaller than -1.0 or greater than 1.0 were log transformed using a log to the base of 10. The log-transformed traits were neutrophils (LNEU), lymphocytes (LLYM), eosinophils (LEOS), mean corpuscular haemoglobin (LMCH), mean corpuscular haemoglobin concentration (LMCHC), red cell distribution width (LRDW) and mean platelet volume (LMPV).

Genetic parameters were estimated with ASReml (Gilmour et al. 2009) applying an animal model and fitting common litter effect as an additional random effect. Further, the program ASReml was also used to test significance of fixed effects which had previously been explore with the GLM and Mixed procedures (SAS, 2014). Weekly batch of pigs entering the weaning shed at the same time was used to define contemporary groups. This effect was fitted as a fixed effect for all traits as is common practice in animal breeding applications. In addition, this effect was fitted

as an additional random effect to compare estimates of variance components across different models. The phenotypic variance was the sum of the additive genetic variance, common litter effect and residual variance and did not include the variance due to the contemporary group effect.

Other fixed effects fitted included sex of the animal while age at weaning and time from collection of blood samples to haematological analysis were fitted as linear covariables for total and differential white blood counts, immunoglobulins and haptoglobin as shown in Table 2.1.1.

Table 2.1.1. Fixed effects fitted for total and differential white blood counts, red blood cell counts, immunoglobulins and haptoglobin.

Variable	CG	sex	Age at weaning	Time period
WBC (10^9/L)	✓	✓		
LNEU (log 10^9/L)	✓	✓		
LLYM (log 10^9/L)	✓			
MONO (10^9/L)	✓			✓
LEOS (log 10^9/L)	✓		✓	
BASO (10^9/L)	✓			✓
HbF (g/L)	✓	✓	□	
HbL (g/L)	✓	✓	✓	
RBC (10^{12}/L)	✓			
HCT (%)	✓	✓	✓	✓
MCV (fL)	✓		✓	✓
LMCH (log pg/cell)	✓		✓	
LMCHC (log g/dL)	✓			✓
RDW (%)	✓	✓	✓	
PLT (10^9/L)	✓	✓	✓	✓
LMPV (log fl)	✓	✓		✓
LIgA (log mg/ml)	✓	✓	✓	
LIgG (log mg/ml)	✓	✓		
LIgM (log mg/ml)	✓		✓	
LHAP (log mg/ml)	✓			

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, RBC: red blood cell count, HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL) , HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, MPV: mean platelet volume, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

2.2. Genetic aspects of disease tolerance and disease resilience in the pig – objectives of Sarita Guy's PhD project

The primary objective of Sarita Guy's thesis is to define and propose a method to quantify the traits of disease tolerance and disease resilience in the pig using records routinely collected on Australian commercial piggeries. It was hypothesised that standard records are sufficient to quantify disease tolerance and disease resilience, and selection criteria for these traits will be able to be defined. In order to test these hypotheses, several research questions were investigated:

- How do the mechanisms of disease tolerance, resilience and resistance differ, and are existing data structures sufficient to model and quantify these traits?

This question was addressed in chapters two and three which provide a literature review about disease tolerance and disease resistance (published in Guy et al., 2012, chapter two) and disease resilience (chapter three).

- How can the pig environment be quantified using routinely collected records? If through contemporary group estimates, which phenotypic traits should we base these estimates on?

The average performance of a group of pigs was used to quantify environmental conditions. Multiple performance measures were evaluated as an environmental descriptor and principal component analyses were explored to combine multiple descriptors into one overall environmental descriptor. These procedures and the subsequent findings were summarised in chapters four (published in Guy et al. 2015), five (published in Guy et al. 2017a) and six.

- Since contemporary group estimates capture all known and unknown challenges experienced, how do we refine this measure to include only infection challenges?

Average performance of a group of pigs is affected by multiple environmental factors. Methodology was developed to separate climatic effects from other unknown environmental effects using spline functions and developing new genetic models. The development of these methods and models and their application in analyses of genotype by environment interactions were outlined in chapters seven (Guy et al. 2017b) and eight (Guy et al. 2017c).

- Do these refined contemporary group estimates reflect health challenges captured by medication records? Can these be used as a proxy of infection challenges for the definition of disease resilience?

Genetic analyses of medication records defined as a simple binary (0/1) trait were conducted to answer this key question. Whether a pig was medicated for a disease or not was outlined in chapter nine (Guy et al., 2018, submitted).

The application of these findings was explored in the General Discussion of the PhD thesis (chapter 10).

2.3. Investigation of environmental conditions in a weaner shed

Air quality measures were recorded for 12 pens of a weaner shed in south-east Queensland on five dates from May to July 2013 and on five occasions from June to August 2014. For each pen, air quality measures were recorded in each corner area about 50 to 70 cm from the side of each pen (four measurements per pen). The pen size was 2.80 m times 2.64 m equivalent of a pen area of 7.4 m^2. The front area of each pen represented the feeding and dunging area with a size of 4.1 m^2. The sheltered resting area was at the back of the pen. The resting area consisted of a hutch that could have boards and heat lamps in use. This was noted at recording. A board was present for 39% of pens and the heat lamp was turned on in 15% of pens.

Air quality measures included temperature, humidity and velocity which were recorded with the vane anemometer test 410-2. The velocity measurement did not have sufficient precision to detect variation in air movement and was dropped from recording procedures. Carbon dioxide (CO_2) levels were measured with the Testo 535 (www.testo.com.au). Ammonium levels (NH_3) were recorded using 2-cm long hydrion ammonia test paper strips (QA supplies). Outliers exceeding four standard deviations from the mean were eliminated from the analyses for all air quality measures. The distribution of air quality measures was evaluated with the univariate procedure (SAS, 2014) which provided the histogram and information about skewness and kurtosis for each measurement. These two parameters (skewness and kurtosis) were larger than 1.0 for CO_2 and NH_3. A log and square root transformation was evaluated for each air quality measure in order to determine the best transformation for each trait. Skewness and kurtosis were improved for CO_2 and NH_3 by applying a square root and log transformation, respectively.

Fixed effect models were developed with the GLM procedure (SAS, 2014) applying a linear general model. Fixed effects that were evaluated were the date of recording, pen, the recording position within each pen and whether the area was covered by a board or whether the heat lamp was turned on. Furthermore, the time of recording was evaluated as an additional fixed effect. Air quality measures were recorded in the morning from 9 am to 11 am in 2013 and from 11 am to 2 pm in 2014. Temperatures are increasing during the day. Therefore, temperature was lower in 2013 in comparison to 2014 due to the different time of recording procedures. This difference between years was confounded with date of recording and alternative models were explored to evaluate this confounding and its effects on air quality measures.

3. Outcomes

3.1. Overview of data

Data statistics for total and differential blood counts, immunoglobulins and haptoglobin traits are shown in Table 3.1.1. Total and differential white blood counts were more variable than traits describing red blood cell measures. Coefficients of variation varied from 22 to 56% for white blood cell measures. Haemoglobin measured on farm was more variable (CV% of 12%) than the corresponding haemoglobin measure recorded in the laboratory (CV% of 9%). Mean corpuscular haemoglobin (LMCH) and mean corpuscular haemoglobin concentration (LMCHC) were least variable with coefficients of variation of two and one percent, which may limit ability to estimate genetic parameters reliably.

Table 3.1.1. Data statistics for total and differential white blood counts, red blood cell counts, immunoglobulins and haptoglobin.

Variable	N	Mean	SD	CV%
BC (10^9/L)	811	16.82	4.711	28
LNEU (log 10^9/L)	806	0.792	0.173	22
LLYM (log 10^9/L)	812	0.854	0.186	22
MONO (10^9/L)	813	1.593	0.601	38
LEOS (log 10^9/L)	810	-0.56	0.229	41
BASO (10^9/L)	813	0.328	0.181	56
HbF (g/dL)	1152	110.3	13.38	12
HbL (g/dL)	812	114	10.05	9
RBC (10^{12}/L)	811	6.46	0.577	9
HCT (%)	810	36.14	3.1	9
MCV (fL)	811	56.03	3.413	6
LMCH (log pg/cell)	811	1.246	0.028	2
LMCHC (log g/dL)	810	1.499	0.012	1
RDW (%)	805	23.49	3.643	16
PLT (10^9/L)	811	675.8	230.9	34
LMPV (log fl)	565	0.944	0.104	11
LIgA (log mg/ml)	799	-0.42	0.35	-
LIgG (log mg/ml)	797	0.902	0.223	25
LIgM (log mg/ml)	797	0.013	0.229	-
LHAP (log mg/ml)	736	-2.25	1.506	67

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, RBC: red blood cell count, HbF, HbL: haemoglobin measured on farm (HgF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Multiple growth traits were investigated based on the repeated weight measurements recorded on farm as outlined in Table 3.1.2. Average growth rate from birth to a specific weight measure increased for longer growth periods as expected. Coefficients of variation were slightly lower for longer growth periods

partly due to the reduced effect of random differences in gut fill for longer growth periods. The coefficient of variation was considerably higher for growth rate shortly after weaning with 66% for growth from weaning to five weeks. This high variability is in part due to the short test period and it highlights variability in the ability of weaner pigs to cope with the weaning process.

Table 3.1.2. Data statistics for growth traits.

Variable	N	Mean	SD	CV%
Average daily gain (g/day) from birth to				
Weaning (ADGb-w)	2025	332.4	47.11	14
Five weeks (ADGb-5)	1889	298.7	45.23	15
Nine weeks (ADGb-9)	1911	474.9	69.8	15
12 weeks (ADGb-12)	1356	575.2	69.61	12
17 weeks (ADGb-17)	1246	679.2	63.92	9
Average daily gain (g/day) for different periods				
Weaning to 5 weeks (ADGw-5)	1780	189.9	125.1	66
Weaning to 9 weeks (ADGw-9)	1870	574.0	110.9	19
Weaning to 12 weeks (ADGw-12)	1334	683.9	94.6	14
Weaning to 17 weeks (ADGw-17)	1217	776.5	78.4	10
Five to nine weeks (ADG5-9)	1796	694.7	136.7	20
Five to 12 weeks (ADG5-12)	1059	785.9	111.6	14
Five to 17e weeks (ADG5-13)	1105	844.4	87.67	10
Nine to 12 weeks (ADG9-12)	1328	911.0	243.7	27
Nine to 17 weeks (ADG9-17)	1180	920.5	120.9	13
12 to 17 weeks (ADG12-17)	1116	924.4	169.8	18

3.2. Estimates of genetic parameters

3.2.1. Heritabilities for immune parameters

Heritabilities for total and differential white blood counts varied from 0.11 (± 0.09) for lymphocytes (LLYM) to 0.46 (± 0.10) for eosinophils (Table 3.2.1.). Two traits (LNEU, LEOS) had low common litter effects which were omitted from the model. Omitting common litter effect caused an increase in heritability estimates as has been observed for other traits in previous genetic analyses. Most estimates of heritabilities from this study were similar to the range of estimates (0.22 to 0.30) presented by Henryon et al. (2006) for differential white blood cells recorded in pigs at 52 days of age. Flori et al. (2011) found higher heritabilities for these traits (range: 0.38 to 0.80). However, standard errors of these estimates were also larger (0.20 and 0.21). Jointly, these studies demonstrate that total and differential blood cells are heritable traits that can be used for pig breeding. All of these studies collected blood samples in weaner pigs between 36 and 57 days of age thereby providing early selection criteria for pig breeding.

The measure of haemoglobin recorded on farm with a handheld device (HbF) had a lower heritability of 0.15 ± 0.08 than the lab measure (HbL: 0.30 ± 0.14, Table

3.2.2.). The residual variance was twice as high in HbF in comparison to HbL indicating larger measurement errors. However, additive genetic variances were the same for both traits. The heritability for HbF found in this study was higher than the estimate of 0.04 ± 0.02 presented by Hermesch and Jones (2012) for the same trait. These results demonstrate that haemoglobin levels in weaner pigs have genetic variation. Handheld on-farm measures may be used to record haemoglobin provided that operators are trained and accuracy of measurements are evaluated through repeated records.

All three immunoglobulin traits had high heritability estimates of 0.44 or above (Table 3.2.3.). In comparison, the heritability estimate for haptoglobin was lower with an estimate of 0.22 (±0.13). Common litter effects were negligible for immunoglobulin A and G.

Table 3.2.1. Estimates of heritability (h^2) and common litter effect (c^2) with standard errors (se) and phenotypic variance (Vp) for total and differential white blood cell counts.

Trait	h^2	se	c^2	se	V_p
WBC	0.26	0.12	0.07	0.05	21.2
LLYM	0.11	0.09	0.10	0.05	0.0326
LNEU	0.28	0.13	0.02	0.05	0.0286
LNEU	0.32	0.09			0.0288
MONO	0.18	0.11	0.12	0.05	0.3322
LEOS	0.41	0.14	0.02	0.05	0.0489
LEOS	0.46	0.10			0.0494
BASO	0.12	0.08	0.08	0.04	0.0289

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.2. Estimates of heritability (h^2) and common litter effect (c^2) with standard errors (se) and phenotypic variance (Vp) for red blood cell traits.

Trait	h^2	se	c^2	se	V_p
HbF	0.15	0.08	0.11	0.04	156
HbL	0.31	0.14	0.06	0.05	80.7
RBC	0.48	0.16	0.02	0.05	0.2700
RBC	0.53	0.10			0.274
HCT	0.30	0.14	0.04	0.05	7.12
MCV	0.63	0.18	0.09	0.06	10.090
LMCH	0.60	0.18	0.12	0.07	0.00077
LMCHC	0.11	0.08	0.08	0.04	0.000085
LRDW	0.39	0.18	0.11	0.07	0.00311
PLT	0.26	0.14	0.10	0.06	49613
LMPV	0.25	0.15	0.14	0.07	0.0085

Abbreviations: HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.3. Estimates of heritability (h^2) and common litter effect (c^2) with standard errors (se) and phenotypic variance (Vp) for immunoglobulin and haptoglobin traits.

Trait	h^2	se	c^2	se	V_p
LIGA	0.44	0.16	0.04	0.07	0.0454
LIGG	0.51	0.17	0.01	0.06	0.0310
LIGM	0.44	0.19	0.10	0.07	0.0335
LHAP	0.22	0.13	0.07	0.05	1.792

Abbreviations: IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Contemporary group was fitted as a random effect in order to compare the magnitude of this effect with additive genetic effects and permanent environmental effects of the litter. Estimates of contemporary groups represent environmental effects influencing all pigs grown out each month. These analyses showed that total and differential white blood count traits were predominantly affected by additive genetic effects and environmental effects captured by contemporary group effects were of minor importance. For these traits, contemporary group effects accounted for 2 to 9% of the phenotypic variance only (Table 3.2.4).

Contemporary group effects were more important for haematological traits accounting for 9 to 35% of the phenotypic variance for these traits. However, contemporary group effects were most important for immunoglobulins and in particular immunoglobulin A accounting for 52 to 172 % of the phenotypic variance of these traits. Variance due to contemporary group could be larger than phenotypic variance because phenotypic variance was derived from the sum of additive genetic variance, common litter variance and residual variance and did not include variance due to contemporary group. Haptoglobin has been suggested as a measure of herd health (e.g. Petersen et al., 2004). This was confirmed by a higher contemporary group estimate of 31% in comparison to a heritability of 0.19 (19%).

Table 3.2.4. Estimates of heritability (h^2) and common litter effect (c^2) with standard errors (se) and phenotypic variance (Vp) for immune and haematological traits when contemporary group (CG^2) was fitted as an additional random effect.

Trait	h^2	se	c^2	se	CG^2	se	V_p
WBC	0.27	0.12	0.07	0.05	0.02	0.02	21.4
LLYM	0.04	0.08	0.12	0.05	0.08	0.04	0.0319
LNEU	0.28	0.12	0.02	0.05	0.02	0.02	0.0287
MONO	0.16	0.10	0.12	0.05	0.08	0.04	0.3337
LEOS	0.41	0.14	0.02	0.05	0.09	0.04	0.0490
BASO	0.12	0.08	0.09	0.04	0.07	0.04	0.0292
HbF	0.13	0.08	0.12	0.04	0.16	0.06	156
HGB	0.27	0.14	0.06	0.05	0.29	0.11	78.895
RBC	0.54	0.16	0.01	0.05	0.21	0.09	0.2764
HCT	0.25	0.13	0.04	0.05	0.35	0.13	6.963
MCV	0.65	0.17	0.07	0.06	0.10	0.05	10.050
LMCH	0.60	0.17	0.10	0.06	0.09	0.04	0.00077
LMCHC	0.12	0.09	0.09	0.05	0.13	0.06	0.000086
LRDW	0.45	0.18	0.09	0.06	0.24	0.10	0.00315
PLT	0.13	0.10	0.13	0.05	0.01	0.02	47559
LMPV	0.29	0.16	0.13	0.07	0.13	0.07	0.0085
LIGA	0.43	0.16	0.05	0.06	1.75	0.60	0.0454
LIGG	0.53	0.10	0.00	0.00	0.52	0.19	0.0311
LIGM	0.47	0.19	0.09	0.07	0.57	0.21	0.0338
LHAP	0.19	0.12	0.08	0.05	0.31	0.12	1.783

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, RBC: red blood cell count, HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

3.2.2. Genetic correlations between immune parameters

Estimates of genetic correlations between total and differential white blood cells were moderate to high ranging from 0.29 ± 0.26 to 0.91 ± 0.09 (Table 3.2.5). Differential white blood cells had higher genetic correlations with WBC and lower genetic correlations with LEOS. A result that was also found by Flori et al. (2011).

Both the on-farm and the laboratory measure of haemoglobin were genetically the same trait (Table 3.2.6). These two haemoglobin measures also had a genetic correlation of close to one with haematocrit demonstrating that these three traits are genetically the same. These high genetic correlations imply that genetic correlations of these three traits with other traits should be similar.

In comparison, genetic correlations between haemoglobin levels and red blood cell count were lower (0.74 for HbF and 0.53 for HbL). This lower genetic correlation is reflected in differing genetic correlations of haemoglobin and red blood cell count with other traits. For example, haemoglobin and haematocrit had no genetic association with mean corpuscular volume which had a genetic correlation of -0.73 (±0.11) with red blood cell count.

Table 3.2.5. Estimates of genetic and common litter correlations (first and second row above diagonal) and residual and phenotypic correlations (first and second row below diagonal) with standard errors (se) for total and differential white blood count traits.

	WBC	LLYM	LNEU	MONO	LEOS	BASO
WBC		.80±.23 .99±.15	.91±.09	.56±.29 .42±.33	.64±.20	.60±.32 .24±.41
LLYM	.64±.04 .68±.02		.41±.38	.45±.52 .28±.30	.59±.32	.77±.50 .02±.35
LNEU	.63±.04 .69±.02	.00±.07 .07±.04		.41±.25	.38±.19	.38±.27
MONO	.47±.06 .48±.03	.03±.06 .12±.04	.48±.06 .43±.04		.29±.26	.36±.39 .95±.20
LEOS	.16±.08 .30±.04	.09±.08 .19±.04	.18±.09 .25±.04	.23±.08 .23±.04		.19±.31
BASO	.32±.06 .36±.04	.01±.06 .09±.04	.36±.06 .35±.04	.51±.05 .53±.03	.03±.08 .07±.04	

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.6. Estimates of genetic and common litter correlations (first and second row above diagonal) and residual and phenotypic correlations (first and second row below diagonal) with standard errors (se) for haemoglobin and red blood cell count traits.

	HbF	HbL	RBC	HCT	MCV
HbF		.95±.19[1] .88±.19	.74±.13	.98±.11[1] .84±.25	-.16±.31 .77±.39
HbL	.64±.03 .68±.02		.53±.17	.99±.01 .97±.03	.31±.27 .67±.47
RBC	.43±.07 .50±.03	.85±.06 .68±.03		.61±.16	-.73±.11
HCT	.59±.04 .67±.02	.95±.01 .96±.003	.85±.05 .71±.03		.22±.29 .72±.59
MCV	.32±.14 .16±.05	.25±.16 .29±.05	-.08±.18 -.47±.04	.35±.15 .29±.05	

Abbreviations: HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), RBC: red blood cell count, HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, [1] Log Likelihood not converged indicating that correlations were at the end of the parameters space, e.g. bigger than 1.

Mean corpuscular haemoglobin was genetically the same trait as red cell distribution width only in the opposite direction as shown by a genetic correlation of -0.78 (±0.16) between these two traits (Table 3.2.7). In addition, corpuscular haemoglobin had negative genetic correlation with platelet count of -0.55 (±0.29). Other genetic correlations between haematological traits shown in Table 3.2.7 had a lower magnitude and were not significant given the standard errors of estimates.

Similarly, haemoglobin levels, red blood cell count and haematocrit had no genetic associations with mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration, red cell distribution width or platelet count (Table 3.2.8). However, high positive correlations due to common litter effect were found between mean corpuscular haemoglobin and haemoglobin levels or haematocrit indicating the permanent environment effect of the common litter affect these haematological traits in a similar way.

There were, however, strong negative genetic correlations between mean corpuscular volume (MCV) and red cell distribution with (LRDW: -0.86 ± 0.12) and platelet count (PLT: -0.60 ± 0.25) indicating that these traits are genetically similar traits only in the opposite direction.

In contrast, different immunoglobulins and haptoglobin were genetically different traits (Table 3.2.9). Genetic correlations varied from 0.13 ± 0.19 to 0.36 ± 0.16 between immunoglobin traits. Haptoglobin had negative genetic correlations of -0.13 ± 0.24 and -0.28 ± 0.36 with immunoglobulin A and M and a lowly positive genetic correlation of 0.08 ± 0.23 with immunoglobulin G.

Table 3.2.7. Estimates of genetic and common litter correlations (first and second row above diagonal) and residual and phenotypic correlations (first and second row below diagonal) with standard errors (se) for red blood cell and platelet traits.

	LMCH	LRDW	PLT	LMPV
LMCH		-.78±.16 -.57±.21	-.55±.29 -.06±.38	-.31±.35 .13±.40
LRDW	-.22±.15 -.48±.04		.42±.34 .14±.39	.29±.38 .01±.38
PLT	.24±.19 -.12±.05	-.02±.12 .14±.05		-.34±.38 -.30±.35
LMPV	.06±.18 -.07±.06	.00±.13 .10±.05	-.10±.11 -.19±.05	

Abbreviations: MCH: mean corpuscular haemoglobin, RDW: red cell distribution width, PLT: platelet count, MPV: mean platelet volume, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.8. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between total and differential white blood cell count and red blood cell and platelet traits.

	HbF	HbL	RBC	HCT	MCV
LMCH	-.06±.32	.33±.26	NA[1]	.25±.29	NA[1]
	.87±.32	.90±.38		.89±.53	
	.37±.13	.40±.14		.27±.15	
	.24±.05	.39±.04		.29±.05	
LRDW	.14±.39	-.10±.35	.71±.19	-.07±.37	-.86±.12
	-.46±.34	-.60±.49		-.48±.60	-.23±.42
	-.16±.09	-.09±.12	-.06±.12	-.06±.12	-.05±.23
	-.12±.04	-.13±.05	.26±.04	-.09±.05	-.47±.04
PLT	.26±.47	-.22±.24	.36±.24	-.35±.23	-.60±.25
	-.24±.30				-.23±.43
	.00±.07	.02±.10	-.16±.10	.00±.09	.20±.20
	00±.04	-.07±.05	.04±.05	-.12±.05	-.18±.05
LMPV	-.42±.42	-.37±.36	-.40±.23	-.44±.25	-.24±.35
	-46±.30	-.39±.45			.25±.43
	.15±.09	.04±.11	.17±.13	.07±.11	.04±.18
	-.03±.05	-.12±.05	-.08±.06	-.11±.05	-.05±.06

Abbreviations: HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed, [1] it was not possible to estimate a correlations.

Table 3.2.9. Estimates of genetic and common litter correlations (first and second row above diagonal) and residual and phenotypic correlations (first and second row below diagonal) with standard errors (se) between immunoglobulins and haptoglobin traits.

	LIGA	LIGG	LIGM	LHAB
LIGA		.31±18	.36±.16	-.13±.24
LIGG	.33±.11		.13±.19	.08±.23
	.31±.04			
LIGM	.28±.15	.41±.13		-.28±.36
	.32±.05	.25±.05		-.42±.48
LHAP	.13±.11	.12±.10	.18±.14	
	.03±.05	.10±.05	-.03±.05	

Abbreviations: IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Genetic correlations between total or differential white blood cells and haemoglobin, red blood cell count or haematocrit were predominantly negative (Table 3.2.10). However, the magnitude was variable and often genetic correlations were not significant given the high standard errors of these estimates. Therefore, it is important to observe patterns in estimates of genetic correlations and consistency of estimates across trait groups.

Lymphocytes had negative genetic correlations with mean corpuscular volume (MCV: -0.51 ± 0.38) and mean corpuscular haemoglobin (LMCH -0.53 ± 0.39, Table 3.2.10) that corresponded with positive genetic correlations between lymphocytes and red cell distribution width (LRDW: 0.64 ± 0.42), platelet count (PLT: 0.79 ± 0.43) and mean platelet volume (LMPV: 0.89 ± 0.67) given that mean corpuscular volume and haemoglobin had negative genetic correlations with platelet traits.

Platelet count had positive genetic correlations with all white blood cell counts except basophils. Estimates of genetic correlations between platelet count and the main white blood cells varied from 0.47 to 0.79 indicating a strong genetic association between these traits.

Basophils represent only a small proportion of white blood cells (< 1-2%) which may explain why no genetic associations between basophils and red blood cell or platelet traits were found.

Table 3.2.10. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between total and differential white blood cell count and haemoglobin, red blood cell and platelet traits.

	WBC	LLYM	LNEU	MONO	LEOS	BASO
HbF	-.33±.40	.25±.58	-.54±0.31	-.18±.42	-.18±.27	.12±.47
	-.06±.37	.05±.31		-.52±.28		-.64±.34
	.12±0.07	.09±.06	.09±.07	.02±.07	.04±.08	.06±.06
	.02±.04	.10±.04	-.05±0.04	-.08±.04	-.03±.04	.00±.04
HbL	-.30±.35	-.19±.46	-.09±.27	-.51±.20	-.06±.22	.17±.43
	.06±.55	-.03±.46				-.80±.54
	.19±.09	.18±.08	.04±.08	.11±.11	.04±.10	-.04±.07
	.04±.05	.09±.04	.00±.01	-.12±.05	.01±.05	-.05±.04
RBC	-.09±.22	.27±0.31	-.13±.20	-.55±.17	.03±.19	-.37±.24
	.17±.10	.10±.09	.09±.10	.17±.13	.05±.11	.07±.10
	.06±.05	.13±.04	-.00±.05	-.14±.05	.04±.05	-.07±.05
HCT	-.25±.38	-.11±.48	.02±.28	-.43±.23	-.05±.29	-.43±.23
	.11±62	.00±.54				
	.18±.09	.17±.07	.00±.08	.06±-.10	.03±.08	.06±.09
	.06±.04	.10±.04	.00±.04	-.10±.05	.01±.04	-.10±.05
MCV	.08±.30	-.51±.38	.13±.20	-.16±.37	-.07±0.18	.12±.37
	-.15±.52	.02±.40		.76±.44		.16±.44
	.05±.15	.21±.14	-.11±.14	.05±.15	.01±.15	-.12±.13
	.01±.05	-.03±.05	.01±.05	.05±.05	-.03±.05	.00±.05
LMCH	-.11±.31	-.53±.39	.05±.21	-.23±.40	-.06±.27	-.08±.39
	-.01±.44	.05±.36		.50±.36	-.07±.78	.29±.39
	.08±.15	.19±.14	-.04±.13	.07±.14	-.01±.17	.00±.12
	.00±.05	-.04±.05	.01±.05	.02±.05	-.04±.05	.01±.05
LRDW	.15±.35	.64±.42	.16±.24	-.27±.40	.15±.21	.40±.41
	.41±.43	.13±.33		.15±.35		-.14±.38
	.03±.11	-.06±.09	.07±.09	.15±.11	.00±.11	-.03±.08
	.10±.05	.10±.04	.09±.05	.03±.05	.06±.05	.05±.04
PLT	.74±.31	.79±.43	.63±.35	.47±.43	.51±.33	-.14±.48
	-.65±.51	-.51±.38		-.48±.35		.23±.36
	.03±.08	.04±.07	.05±.07	.09±.08	.04±.08	.15±.07
	.14±.04	.10±.04	.16±.04	.09±.04	.14±.04	.11±.04
LMPV	.64±.50	.89±.67	-.23±.27	NA[1]	-.15±.25	NA[1]
	-.69±.41	-.43±.34				
	-.13±.09	-.16±.09	.12±.10		.01±.11	
	-.05±.05	-.05±.05	.01±.05		-.05±.05	

For abbreviations see Table 3.0.1. [1] it was not possible to estimate correlations.

Total and differential white blood cells had no genetic associations with immunoglobulins (Table 3.2.11). In contrast, genetic correlations between haptoglobin and total and differential white blood cells were positive ranging from 0.11 to 0.56. This consistency in genetic correlations indicates a positive genetic association between haptoglobin and total and differential white blood count despite the high standard errors of estimates. Phenotypic correlations were also positive between haptoglobin and white blood cell counts further indicating positive associations between haptoglobin and white blood cell count.

White blood cell count traits had negative genetic correlations with haemoglobin, red blood cell and haematocrit. Negative genetic correlations between haptoglobin and haemoglobin levels, red blood cell count or haematocrit are therefore consistent among trait groups. Estimates of genetic correlations between haptoglobin and haemoglobin levels, red blood count or haematocrit varied from -0.60 to -0.11 (Table 3.2.12).

Genetic correlations between immunoglobulins and haemoglobin levels, red blood cell count or haematocrit were predominantly positive (Table 3.2.12). In particular, immunoglobulin A had high positive genetic correlation with these traits ranging from 0.30 to 0.64.

Table 3.2.11. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between total and differential white blood cell count and immunoglobulin and haptoglobin traits.

	WBC	LLYM	LNEU	MONO	LEOS	BASO
LIGA	-.07±.22	.10±.32	-.29±.22	.24±.27	-.04±.18	.38±.33
	.06±.10	-.03±.09	.11±.10	-.06±.10	.06±.12	-.01±.09
	.00±.05	.01±.04	-.04±.05	.04±.05	.01±.05	.08±.04
LIGG	.18±.21	.32±.31	-.08±.20	.05±.26	.01±.17	.24±.32
	-.09±.10	-.06±.08	-.01±.10	-.03±.09	-.10±.11	-.08±.09
	.02±.05	.04±.04	-.04±.05	.00±.05	-.05±.05	.01±.04
LIGM	-.05±.35	.24±.48	-.07±.23	.21±.40	.12±.20	-.24±.42
	-.01±.48	.04±.36		.26±.37		.03±.39
	.14±.13	.09±.10	.00±.10	.08±.11	-.05±.12	.05±.10
	.06±.05	.11±.04	-.03±.05	.13±.05	.03±.05	-.02±.04
LHAP	.28±.40	.11±.50	.26±.28	.56±.39	.22±.27	.52±.46
	.19±.50	.23±.42		-.46±.48		-.13±.45
	.06±.08	.00±.07	.06±.08	.00±.08	-.08±.09	.03±.07
	.12±.04	.03±.04	.11±.04	.07±.04	.01±.05	.09±.04

Abbreviations: WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.12. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between haemoglobin levels or red blood cell count traits and immunoglobulins or haptoglobin.

	HbF	HbL	RBC	HCT	MCV
LIGA	.64±.19	.49±.20	.30±.16	.57±.20	.00±.18
	-.22±.11	-.26±.12	-.28±.14	-.25±.12	.12±.18
	.09±.05	.06±.05	.04±.05	.08±.05	.04±.05
LIGG	.20±.28	.23±.21	.18±.17	.34±.21	.06±.17
	.01±.08	.03±.11	.06±.12	-.02±.10	-.14±.17
	.05±.04	.11±.05	.12±.05	.12±.05	-.02±.05
LIGM	.21±.36	0.11±.22	-.25±.18	.44±.38	.48±.27
	-.14±.38			-.63±.45	-.29±.40
	-.11±.10	-.09±.12	.11±.15	-.09±.11	-.24±.21
	-.02±.05	.003±.05	-.08±.05	.00±.05	.10±.05
LHAP	-.18±.42	-.60±.31	-.11±.24	-.45±.26	-.33±.33
	-.26±.38	.31±.64			.10±.46
	.03±.07	.07±.10	.07±.10	.04±.09	-.05±.13
	-.04±.04	-.09±.05	.00±.05	-.10±.05	-.13±.05

Abbreviations: HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Both immunoglobulin A and M had negative genetic correlations with red cell distribution width and platelet count ranging from -0.49 to -0.20 (Table 3.2.13). Mean platelet volume had a positive genetic correlation with immunoglobulin A (0.55 ± 0.27) which corresponds to the negative genetic correlations between immunoglobulin A and red cell distribution width or platelet count given the negative genetic correlation between mean platelet volume and red cell distribution width or platelet count.

Haptoglobin had a negative genetic correlation with mean corpuscular haemoglobin (-0.30 ± 0.33) and a positive genetic correlation with men platelet volume (0.43 ± 0.41) which were consistent with other genetic correlations involving these traits.

Table 3.2.13. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between red blood cell or platelet traits and immunoglobulin or haptoglobin traits.

	LMCH	LRDW	PLT	LMPV
LIGA	.00±.18	-.20±.20	-.49±.25	.55±.27
	.05±.17	.14±.13	.07±.10	-.20±.13
	.02±.05	-.03±.05	-.12±.05	.09±.06
LIGG	.03±.18	-.07±.17	.00±.23	.12±.26
	-.08±.17	.19±.15	.03±.10	-.07±.12
	-.01±.05	.04±.05	.02±.05	.01±.05
LIGM	.56±.24	-.49±.27	-.42±.30	.13±.39
	-.51±.46	.33±.59	-.14±.46	.44±.42
	-.43±.29	.32±.24	.09±.16	-.05±.15
	.09±.06	-.09±.06	-.13±.05	.07±.06
LHAP	-.30±.33	.09±.40	.26±.41	.43±.41
	.03±.44	-.32±.46	-.02±.45	-.08±.44
	-.02±.15	.06±.10	.04±.09	-.04±.10
	-.11±.05	.03±.05	.09±.04	.06±.05

Abbreviations: MCH: mean corpuscular haemoglobin, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

3.2.3. Heritability estimates for growth traits

All growth traits were moderately heritable (Table 3.2.10) including ADGw-5 which covers only a short time period of ten days from weaning to 5 weeks. Variation in gut fill contributes to measurement errors in weight and longer test periods are required to measure growth accurately (Arthur et al., 2008). The highest variability was observed for ADGw-5, however, the heritability estimate of 0.26 ± 0.08 for ADGw-5 demonstrates genetic variation in the ability of weaner pigs to cope with the weaning process. Colditz and Hine (2016) suggested to use husbandry practices such as weaning that provide physical and social stressors to animals for characterisation of resilience phenotypes. The genetic variation found for ADGw-5 supports the notion to use growth shortly after weaning for genetic improvement of resilience of pigs. This growth trait was genetically a different trait than growth rate until 17 weeks of age given the genetic correlation of 0.21 (± 0.28) between these two growth traits further demonstrating that this trait provides information for genetic improvement of pigs in addition to growth until 17 weeks.

As expected, the common litter effect was more important for early growth traits with estimates of 0.17 (± 0.03) and 0.13 (± 0.03) for growth from birth or weaning until five weeks of age. Heritabilities are increased when common litter effects are not fitted in the model due to sampling correlations between these effects. The moderate heritability of 0.26 mentioned above for ADGw-5 was obtained for a model that included both common litter effect and heritability demonstrating that a high

proportion of the phenotypic variance could be explained by these two random effects. Despite these higher effects of the common litter effect, heritabilities were still similar Further, it was slightly higher when the pre-weaning period was included in the growth trait. Omitting the common litter effect from growth traits until 17 weeks of age, increased heritability estimates as has been observed previously.

Table 3.2.10. Estimates of heritability (h^2) and common litter effect (c^2) with standard errors (se) and phenotypic variance (Vp) for growth traits.

Trait	h^2	se	c^2	se	Vp
ADGb-5	0.27	0.08	0.17	0.03	1682
ADGw-5	0.26	0.08	0.13	0.03	8648
ADGb-17	0.22	0.09	0.04	0.04	3694
ADGb-17	0.30	0.08	-		3752
ADGw-17	0.23	0.10	0.02	0.04	6546
ADGw-17	0.27	0.08	-		6598

Abbreviations: ADGb-5: average daily gain from birth to five weeks; ADGw-5: average daily gain from weaning to five weeks; ADGb-17: average daily gain from birth to 17 weeks; ADGw-17: average daily gain from weaning to 17 weeks.

3.2.4. Genetic correlations between immune or haematological and growth traits

Total white blood count and lymphocytes had positive genetic correlations with growth until 17 weeks ranging from 0.24 (±0.25) to 0.86 (±.41, Table 3.2.11) indicating that selection for higher levels of total white blood count and lymphocytes at weaning is beneficial for growth until slaughter. Positive genetic correlations were also found between these two immune traits and growth from birth until weaning. In contrast, growth from weaning to five weeks had no genetic correlations with these two immune traits. This genetic correlation may have been affected by health status of pigs during this time, e.g. sick pigs with a health challenge are expected to have low growth and high levels of total white blood cell count to fight any disease. A similar shift in genetic correlations was also observed between neutrophils and growth traits, although the magnitude of positive genetic correlations between neutrophils and growth until 17 weeks was lower ranging from 0.13 (±0.24) to 0.29 (±0.22). Both monocytes and basophils had positive genetic correlations with all growth traits.

Early growth measures had negative genetic correlations with haemoglobin levels varying from -0.66 ± 0.22 between haemoglobin recorded on farm and growth from birth to five weeks to -0.09 ± 0.27 between haemoglobin recorded in the laboratory and growth until five weeks (Table 3.2.12). Estimates of genetic correlations were higher between later growth traits and haemoglobin levels with estimates ranging from -0.19 ± 0.29 to 0.36 ± 0.23. Similarly, the genetic correlation between on-farm haemoglobin and growth until 21 weeks of age was -0.26 ± 0.20 in the study by Hermesch and Jones (2012). At the phenotypic level, Perri et al. (2016) found lower haemoglobin levels in larger weaner pigs and highlight the larger iron requirements of pigs with higher growth rate. The higher haemoglobin requirements of faster-growing pigs may influence estimates of genetic correlations between these traits, and the genetic correlation between haemoglobin and growth may be affected when

haemoglobin is measured, i.e. at the start or the end of the growth period. These aspects need to be considered when genetic correlations between haemoglobin and growth are evaluated.

Table 3.2.11. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between total and differential white blood cell count and growth traits.

	ADGb-5	ADGw-5	ADGb-17	ADGw-17
WBC	.26±31	-.06±.30	.46 ± .24	.24±.25
	.41±.29	-.17±.29	not fitted	not fitted
	-.16±.07	-.09±.08	-.17 ± .08	-.10±.08
	.01±.04	-.09±.04	.01 ± .05	-.01±.05
LLYM	.47±.43	-.05±.39	.86±.41	.60±.38
	.21±.24	-.24±.23		
	-.08±.06	-.05±.07	-.08±.07	-.05±.07
	.05±.04	-.08±.04	.07±.05	.06±.05
LNEU	.07±.23	-.12±.24	.29±.22	.13±.24
	not fitted	not fitted	not fitted	not fitted
	-.14±.08	-.04±.08	-.20±.08	-.13±.08
	.00±.00	.00±.0002	-.05±.05	-.05±.05
MONO	not conv.	.77±.29	.70±.23	.45±.27
		.35±.21	not fitted	not fitted
		-.16±.08	-.25±.08	-.18±.07
		.10±.04	-.01±.05	-.03±.05
LEOS	.37±.19	.01±.22	-.42±.19	-.49±.19
	not fitted	not fitted	not fitted	not fitted
	-.08±.09	.09±.09	.14±.10	.13±.09
	.09±.04	.05±.04	-.07±.05	-.09±.05
BASO	.50±.34	.52±.30	.75±.21	.54±.26
	.52±.27	.42±.27	not fitted	not fitted
	-.16±.07	-.10±.07	-.19±.08	-.13±.07
	.04±.04	.08±.04	.03±.05	.01±.05

Abbreviations: ADGb-5: average daily gain from birth to five weeks; ADGw-5: average daily gain from weaning to five weeks; ADGb-17: average daily gain from birth to 17 weeks; ADGw-17: average daily gain from weaning to 17 weeks; WBC: white blood cell count, NEU: neutrophils, LYM: lymphocytes, MONO: monocytes, EOS: eosinophils, BASO: basophils, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

Table 3.2.12. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between haemoglobin, red blood cell or platelet traits and growth traits.

	ADGb-5	ADGw-5	ADGb-17	ADGw-17
HbF	-.25±.30	-.66±-.22	-.19±.27	.25±.36
	-.27±.20	-.70±.13	not fitted	-.87±.87
	-.26±.06	-.24±.06	-.12±.06	-.11±.06
	-.25±.03	-.39±.03	-.14±.04	-.08±.04
HbL	-.09±.28	-.24±.28	.14±.25	.23±.26
	-.36±.31	-.80±.24	not fitted	not fitted
	-.41±.08	-.42±.08	-.25±.08	-.18±.08
	-.30±.04	-.42±.04	-.13±.05	-.06±.05
RBC	.17±.20	-.57±.15	.25±.20	.13±.22
	not fitted	not fitted	not fitted	not fitted
	-.35±.10	-.28±.09	-.18±.10	-.09±.09
	-.12±.04	-.37±.04	-.01±.05	-.01±.05
HCT	.02±.29	-.18±.29	.26±.25	.32±.26
	-.34±.41	-.86±.37	not fitted	not fitted
	-.36±.08	-.34±.07	-.24±.08	-.18±.08
	-.24±.04	-.34±.04	-.10±.05	-.05±.05
MCV	-.22±.23	.29±.22	-.12±.20	.05±.21
	-.19±.30	-.12±.15	not fitted	not fitted
	-.04±.13	-.03±.15	-.08±.13	-.13±.13
	-.13±.05	.09±.05	-.09±.05	-.04±.05
LMCH	-.29±.22	.21±.24	-.19±.20	.01±.21
	-.37±.26	-.36±.28	not fitted	not fitted
	-.14±.13	-.18±.16	-.09±.13	-.12±.12
	-.22±.04	-.03±.05	-.13±.05	-.06±.05
LRDW	.24±.25	-.42±.25	.29±.23	.11±.24
	.30±.31	.20±.29	not fitted	not fitted
	.08±.11	-.02±.10	-.02±.10	.01±.09
	.16±.04	-.12±.05	.09±.05	.04±.05
PLT	.21±.31	-.25±.31	.19±.27	.09±.28
	-.02±.30	.04±.26	not fitted	not fitted
	-.08±.08	.01±.08	.00±.08	.02±.08
	.00±.04	-.05±.04	.04±.03	.03±.04
LMPV	.75±.48	.87±.37	.25±.33	.13±.32
	-.07±.23	.23±.21	not fitted	not fitted
	-.14±.08	-.23±.09	-.01±.09	.03±.09
	.04±.05	.07±.05	.06±.06	.05±.06

Abbreviations: ADGb-5: average daily gain from birth to five weeks; ADGw-5: average daily gain from weaning to five weeks; ADGb-17: average daily gain from birth to 17 weeks; ADGw-17: average daily gain from weaning to 17 weeks, HbF, HbL: haemoglobin measured on farm (HbF) or in the laboratory (HbL), HCT: haematocrit (packed cell volume), MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, RDW: red cell distribution width, PLT: platelet count, LMPV: mean platelet volume, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed, [1] it was not possible to estimate a correlations.

There were no genetic associations between immunoglobulin A and growth traits (Table 3.2.13). In comparison, negative genetic correlations ranging from -0.41 (±0.19) to -0.12 (±0.26) were found between immunoglobulin M and growth traits. Genetic correlations between immunoglobulin G and growth were negative (-0.15 ± 0.15 and -0.21 ± 0.15) for the two early growth traits and positive (0.32 ± 0.21 and 0.34 ± 0.20) for average daily gain until 17 weeks. This shift in correlations was also observed for phenotypic correlations. Haptoglobin levels in weaner pigs had low positive genetic correlations with growth rate (range: 0.11 to 0.39) which were not significant given the number of records available.

Table 3.2.13. Estimates of genetic and common litter correlations (first and second row) and residual and phenotypic correlations (third and fourth row) with standard errors (se) between immunoglobulins or haptoglobin and growth traits.

	ADGb-5	ADGw-5	ADGb-17	ADGw-17
LIGA	.24±.18	.04±.17	.02±.22	-.07±.22
	.09±.11	.21±.15	.04±.10	.02±.09
	.16±.05	.11±.05	.03±.05	-.01±.05
LIGM	-.12±.26	-.37±.26	-.41±.19	-.37±.20
	-.05±.30	.44±.31		
	.07±.11	.09±.11	.06±.11	.02±.10
	-.02±.05	-.02±.05	-.14±.05	-.13±.05
LIGG	-.15±.15	-.21±.15	.34±.20	.32±.21
	.13±.12	.09±.15	-.05±.10	-.04±.08
	-.03±.05	-.10±.05	.10±.05	.09±.04
LHAP	.18±.32	.39±.29	.27±.25	.11±.28
	.25±.30	.07±.31		
	-.07±.07	-.12±.08	-.03±.08	.02±.08
	.03±.04	.03±.04	.05±.05	.04±.05

Abbreviations: ADGb-5: average daily gain from birth to five weeks; ADGw-5: average daily gain from weaning to five weeks; ADGb-17: average daily gain from birth to 17 weeks; ADGw-17: average daily gain from weaning to 17 weeks, IgA, IgG, IgM: immunoglobulin A, G, M, HAP: haptoglobin, note the letter L at the beginning of the trait abbreviation indicates that the trait was log transformed.

3.3. Genetic aspects of disease tolerance and disease resilience in the pig – results from Sarita Guy's PhD thesis

3.3.1. Defining disease resilience (from Chapter 3)

Disease resilience has been defined in various ways and inconsistently in literature. After taking into consideration how disease resilience has been defined in previous studies, the unknowns about the specific mechanisms responsible, and the data that may be available to identify the pigs that are able to cope with infection challenges, a definition of disease resilience is provided in this thesis. This thesis defines disease resilience as the ability to maintain performance and health despite increasing

infection challenges in the environment, by utilising both mechanisms of disease resistance and disease tolerance. Further, the way that disease tolerance and disease resilience are primarily distinguished from each other is through the location of the infection challenge: tolerance considers pathogen burden within the animal, while resilience looks at the infection level within the environment. If investigations are on natural infectious challenges that occur on-farm, other environmental challenges need to be accounted for, along with environmental infection challenges. Therefore, descriptors of the overall environmental challenges, as well as infection challenges, are needed for a multi-dimensional approach to modelling this trait.

The environmental conditions that pigs experience can be quantified through specific environmental variables, such as air quality, temperature and humidity. These variables were previously highlighted as the ones that also reflect the health status of a farm. Managerial processes and feed quality can also influence the quality of environment, and data on these factors may be examined. The social environment may also be taken into consideration, as social interactions can affect the quality of environment of the pig (Bijma 2014). Pigs can influence the performance of other pen mates genetically, termed indirect genetic effects. This social interaction challenge may be taken into account provided data is available on pen allocation and pen mates. Alternatively, an environmental descriptor can be derived through the phenotypic averages of a group of animals under the same environmental and managerial conditions (known as contemporary groups). The Finlay and Wilkinson (1963) approach of quantifying the environment through contemporary group means, adjusted for genetic and systematic effects, can be used if the environment is complex, or there are no measures of the environment available. Therefore, contemporary group estimates may provide a practical way to quantify the challenges in the pig environment. This measure of the environment quantifies the interactions between environmental variables as challenges are taken into account simultaneously. If specific challenges in the overall environmental descriptor can be partitioned, a measure of infection challenge may then be derived for the evaluation of disease resilience.

3.3.2. Backfat as an environmental descriptor for a GxE analysis of growth (Chapter 4)

This publication contributes to the second research question, where contemporary group estimates of various phenotypic traits are explored and used as environmental descriptors for genetic analyses. In this chapter, the contemporary group estimates of backfat were explored. Genotype by environment interaction for growth was evaluated using the multi-trait approach, whereby Pearson's correlations between the estimated breeding values (EBVs) for each animal were used to estimate the level of genotype by environment interaction. These results were compared to when using an environmental descriptor based on contemporary group estimates of average daily gain.

The conclusion of this publication was that contemporary group estimates of backfat captured different aspects of the environment from those captured by

contemporary group estimates of growth. This may warrant use of both traits simultaneously in the quantification of environments. The genetic associations described by Pearson's correlations between EBVs of all animals in the herd suggested significant interaction, using environments defined by either of the two performance traits.

Pearson's correlations between EBVs are known to provide an underestimation of genetic correlations because they do not completely reflect the genetic relationship between traits. Therefore, a postscript to this published chapter was included to provide more accurate estimates of the level of genotype by environment interaction. Bivariate models were used to construct genetic correlation matrices. Estimates of genetic correlations contradicted the published results– there were no significant genotype by environment interactions for growth, using either of the traits to define the environment. This discrepancy was explored in detail in the postscript of the published paper. While Pearson's correlations between EBVs underestimated genetic correlations, genetic correlations estimated through bivariate analyses may be inflated if there are poor links between environments. Evaluation of G×E can be performed on a sire level, provided that sires are well represented by their progeny across all environments. Use of the environmental descriptor of backfat increased the ability to detect G×E compared to use of an environmental descriptor of growth. The potential use of alternative performance traits as environmental descriptors should be further investigated.

3.3.3. Extension of environmental descriptors used to analyse sire by environment interaction for growth of pigs (Chapter 5)

This publication further contributes to the second research question, where additional performance traits were used to derive contemporary group estimates as environmental descriptors. A different herd was used for this chapter as there were more traits available. Further, a different genetic model was used to evaluate the extent of variation in growth across environmental trajectories.

Contemporary group estimates of average daily gain, backfat, daily feed intake, and muscle depth were used to define the environment. These four environmental descriptors were used individually, and simultaneously (combined through principal component analysis), to evaluate sire by environment interaction for growth using the sire interaction model.

This publication concluded that contemporary group estimates of average daily gain, backfat, muscle depth and daily feed intake, captured different aspects of the environment. This warranted combining these traits through principal component analysis. There was significant sire by environment interaction when environments were defined by average daily gain. A significant sire by environment interaction was also detected using the first principal component as the overall descriptor, however this overall descriptor was heavily made up by average daily gain. While use of all traits simultaneously is expected to capture more environmental variation and can be argued to be a more objective measure of the environment, use of the environmental descriptor based on average daily gain alone appears adequate in describing phenotypic variability attributed to sire by environment interaction for growth.

3.3.4. Reaction norm analysis of pig growth using environmental descriptors based on alternative traits (Chapter 6)

The phenotypic average of a contemporary group (CG) is a practical way to quantify the environment as it uses standard performance records collected on farm. The objective of this study was to extend the traits used to derive environmental descriptors of the growing pig, to include early growth between birth and start of feed intake test (ADG1), growth during feed intake test (TADG), lifetime growth (LADG), daily feed intake (DFI), backfat (BF) and muscle depth (MD). Pedigree and performance records from a commercial Australian piggery were used to derive CG estimates of these six traits, which were further combined using principal component analysis. These definitions of the environment were used in reaction norm analysis of growth, to assess variation in performance of sire lines across the environmental trajectory (Sire×E for growth). The CG estimates of growth traits described different aspects of the environment from the CG estimates of carcass traits ($r < 0.10$). The most appropriate reaction norm model to evaluate Sire×E for growth depended on the environmental descriptor. The environmental descriptor based on CG estimates of BF and MD had a non-significant overall environment effect, resulting in inflated estimates of the common litter environment effect and sire intercept variance. Inclusion of CG as an additional random effect absorbed the unaccounted environmental variability. There was no detectable Sire×E using all definitions of the environment, however, this may be due to sires not being represented in sufficient environmental ranges. Nevertheless, the estimated variance in sire slopes was largest when environments were defined by CG estimates of BF (97 ± 83 (g/day)). There was no value in using the overall descriptor derived by principal component analysis. Improved data structure is firstly required to better assess these environmental descriptors based on alterative traits, used for reaction norm analysis of pig growth. Nevertheless, measures of the environment derived from a different trait from that being analysed can be argued to be a more objective measure of the environment.

3.3.5. Contemporary group estimates adjusted for climatic effects provide a finer definition of the unknown environmental challenges experienced by growing pigs (Chapter 7)

Environmental descriptors (EDs) derived from mean performances of contemporary groups (CGs) are assumed to capture any known and unknown environmental challenges. The objective of this paper was to obtain a finer definition of the unknown challenges, by adjusting CG estimates for the known climatic effects of monthly maximum air temperature (MaxT), minimum air temperature (MinT) and monthly rainfall (Rain). Since the unknown component could include infection challenges, and these EDs may help to better model varying responses of sire progeny to environmental infection challenges for the definition of disease resilience. Data were recorded from 1999 to 2013 at a piggery in south-east Queensland, Australia (n = 31,230). In the first step of analysis, CG estimates of average daily gain (ADG) and backfat (BF) were adjusted for MaxT, MinT and Rain, which were fitted as splines. For the ED based on ADG, MaxT and MinT were significant variables, reducing the estimated variance component for the random CG effect. Other variance component estimates remained stable, suggesting that

these significant climatic variables accounted for some known environmental variation captured in CG estimates. No climatic variables were significant in the models to derive the ED based on BF. Environments were categorised using the EDs from the first step of analysis. There was no observable sire by environment interaction (S×E) for ADG when using the EDs based on BF. For the EDs based on ADG, there was significant S×E only when MinT was included in the model ($p = 0.01$). Therefore, this new definition of the environment, pre-adjusted by MinT, increased the ability to detect S×E. While the unknown challenges captured in refined CG estimates need verification for infection challenges, this is a practical approach to help identify and breed for disease-resilient pigs using routinely collected data.

3.3.6. Separating seasonal effects from other challenges in the pig environment using time series analysis (Chapter 8).

Seasonal decomposition is an effective and simple way of partitioning the seasonal effects from other environmental challenges captured in CG estimates. The remaining trend and residual components for the CG estimates, unadjusted and adjusted for minimum monthly temperatures, were very highly correlated, suggesting this methodology may be sufficient to decompose the seasonal effects captured in CG estimates. These trend and residual component may need to be considered separately in genetic models for genetic improvement of disease resilience.

3.3.7. Quantifying the health challenges in Australian piggeries using medication records for the definition of disease resilience (Chapter 9)

Pedigree, production and medication records were available for an Australian herd of Large White pigs from between 2011 and 2016. These records were used to address the three objectives. The first objective was to characterise the health challenges of the herd. The majority of medication treatments were administered to sows that experienced farrowing issues. For the growing pig, the majority of the treatments were related to tail bite, followed by being generally unwell, issues involving feet and legs, and skin conditions. The prevalence of medication for the growing pig was calculated as the percentage of pigs born each month which were medicated at some point during production. Two pedigrees were used to quantify the number of pigs in the herd – a reduced pedigree based on available performance records, and a full pedigree, constructed using numbers weaned per litter. Since the full pedigree presents a more precise measure of the total number of pigs on-farm each month, use of the full pedigree provides a more accurate estimate of medication prevalence compared to using the reduced pedigree. The very low incidence of pathogenic challenges indicates that this herd is of high-health status. The second objective of this study was to estimate genetic parameters for health of the growing pig. A health trait was defined as a binary (case/control) outcome of medication treatment, and fitted as a generalised linear mixed sire model. There were 812 medicated pigs that were linked to pedigree records (cases), and the remaining 8,023 in the reduced pedigree and 21,352 in the full pedigree were assumed to have not been medicated (controls). Significant fixed effects included sex (male and female pigs with roughly approximately equal numbers) and a linear

covariate of the number post-weaning deaths in the litter. Estimates of fixed effects and genetic parameter estimates were fairly consistent using both pedigrees, with heritability estimated at 0.06 ± 0.04 (± SE) using the reduced pedigree, and 0.04 ± 0.03 using the full pedigree. Therefore, the reduced pedigree available from performance recording may be sufficient to derive genetic parameter estimates for health. In this herd, male pigs from litters with a higher number of post-weaning deaths were more likely to be medicated. Separate models were used to evaluate the association between medication status and the genetic merit for growth. Animals with higher estimated breeding values for growth were less likely to be medicated, which supports the use of growth as an indirect indicator for health. The final objective of this study was to validate the infection challenges captured in contemporary group (CG) estimates of growth rate. The five environmental descriptors based on CG estimates for growth using different models had moderately negative linear relationships with the frequency of pig medication for the corresponding months, in particular CG estimates unadjusted for climatic effects (r = -0.29, Figure 3.2.7.1). However, the ability to measure disease resilience for this herd using these records is restricted due to the minimal pathogenic challenges experienced in this herd. Nevertheless, as the quality of environments described by CG estimates increase, the pigs are less medicated, and robustness of sire lines can be evaluated.

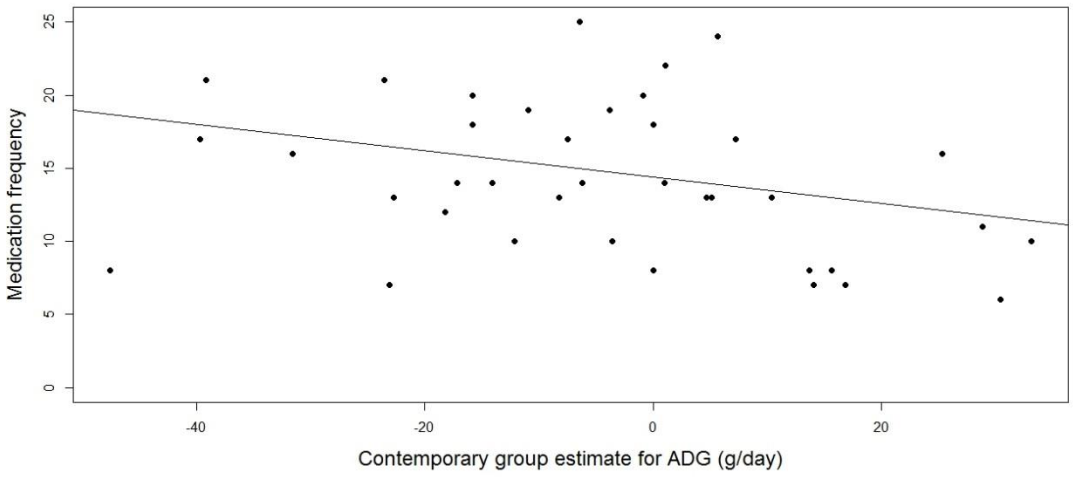

Figure 3.2.7.1 Medication frequency is higher in groups of pigs with lower growth rate.

3.3.8. General Discussion (Chapter 10)

The primary objective of this thesis was to define and propose a method to quantify the traits of disease tolerance and disease resilience in the pig. The main data source explored were the standard production records routinely collected on Australian commercial piggeries, which were already being used for routine genetic evaluations. Other data examined included on-farm medication treatment records and open source climatic information. It was hypothesised that these data are sufficient to quantify disease tolerance and disease resilience, and that selection criteria for these traits will be able to be defined.

This chapter discusses the important considerations for the evaluation of disease tolerance and disease resilience, which have arose from to thesis investigations. Firstly, the necessity to distinguish between the mechanisms of disease tolerance, disease resilience and disease resistance is examined. Then, aspects of how environments are quantified are discussed, and how these definitions of the environments can be further refined for the evaluation of disease resilience. The various methods used to assess genetic variation in response to environmental trajectories are then evaluated. Lastly, this chapter outlines the research implications and recommendations for the Australian pork industry, which will enable selection for not only productivity, but also for health and welfare.

3.4. Investigation of environmental conditions in a weaner shed

The data statistics for the climate and air quality measures are presented in Table 3.4.1. For these winter months, temperatures were already 23 degrees Celsius for these measurements that were predominantly recorded in the morning with an average recording hour of 10:30 am. Both carbon dioxide and ammonia were not normally distributed which contributed to their higher coefficients of variation for the original trait. The coefficients of variations were lower when these two traits were transformed which improved the distribution of these traits. Overall, the data statistics demonstrate that air quality measures recorded in one weaner shed have considerable variation. The causes of this variation are explored below.

Table 3.4.1. Data statistics for climate and air quality measures

Variable	N	Mean	Std Dev	Minimum	Maximum	CV
Temp	416	23.2	3.3	15.5	28.3	14.2
Hum	416	57.9	11.7	30.1	89.0	20.2
CO_2	410	1028.8	380.1	61.0	2400.0	36.9
CO_2-sqrt	410	31.6	5.7	7.8	49.0	18.1
NH_3	415	6.5	3.3	0.0	20.0	51.3
NH_3-log	404	1.8	0.4	0.0	3.0	24.2

Abbreviations: Temp: temperature; hum: humidity, %; CO_2: Carbon dioxide, ppm; CO2-sqrt: Carbon dioxide after square-root transformation, ppm; NH_3: ammonia, ppm; NH_3-log: ammonia after log transformation, ppm; Std Dev: standard deviation; CV: Coefficient of variation, %.

A rise in temperature was associated with lower humidity and higher carbon dioxide levels (Table 3.4.2.) The absolute magnitude of these associations varied from 0.38 to 0.46 indicating close associations. In comparison, correlations between temperature and ammonia were lower and not significant once ammonia was log transformed. Humidity had lowly positive correlations with all other air quality measures. The two air quality measures had positive correlations of 0.38 and 0.39 for different definitions of these variables. The transformation modified ammonia more in comparison to carbon dioxide because the distribution of ammonia

measures was not as continuous as the distribution of carbon dioxide. The main classes for ammonia observations were 2.5 (n: 24), 5 (n: 201), 7.5 (n: 42) and 10 (n: 82).

The development of fixed effect models showed that large proportions of the variation observed for climate and air quality measures could be explained (Table 3.1.3.). The date of recording was the most important effect for both climatic measures. In particular, temperature was highly variable between dates and 89% of the variation observed in temperature was attributed to this effect. There was a confounding between date of recording and hour of recording as outlined in the previous section. Hour of recording accounted for 70% of the variation observed for temperature when fitted as the single factor. When these two factors were fitted jointly (Model 3), 92% of the variation observed for temperature was accounted for, which is only slightly higher than the proportion of variation explained by fitting date of recording alone. Therefore, date of recording captures most of the observed variation that is due to these two effects. Similar results were obtained for humidity and to a lesser extent the other air quality measures.

Table 3.4.2. Pearson correlations (with P values on second row) between climate and air quality measures.

	Hum	CO_2	CO_2-sqrt	NH_3	NH_3-log
Temp	-0.46	0.38	0.39	0.17	0.08
	<0.0001	<0.0001	<0.0001	0.0006	0.12
Hum		0.11	0.10	0.14	0.19
		0.03	0.03	0.004	0.0001
CO_2			0.99	0.39	0.38
			<0.0001	<0.0001	<0.0001
CO_2-sqrt				0.39	0.38
					<0.0001
NH_3					0.94
					<0.0001

Abbreviations: Temp: temperature; hum: humidity; CO_2: Carbon dioxide; NH_3: ammonia; CO2-sqrt: Carbon dioxide after square-root transformation; NH_3-log: ammonia after log transformation;

Hour was fitted as a fixed effect that was nested within date of recording (Model 3, 4 and Final). The alternative model which fitted hour as a linear covariable was inferior because the coefficients of variations were lower for Model 3a in comparison to Model 3 for all variables.

Pen affected all climate and air quality measures. The location of pens within the weaner shed corresponded to estimates of pen effects for multiple climate and air variables; i.e. all variables were higher for some pens that were closely located. Further information is required from the farm to identify the underlying causes of these effects due to the characteristics and position of the building.

Effects for corner clearly showed climatic and air quality differences between the back and the front of the pen. The back of the pen was more enclosed and had less ventilation which led to higher humidity, carbon dioxide and ammonia levels. In addition, covering the back of the pen with a board increased all climate and air-

quality parameters demonstrating how specific designs in weaner sheds can affect microclimate within individual pens.

Overall, these analyses demonstrate to use temperature from either bureau of meteorology or data loggers on farm because individual measures are affected strongly by the time of measurement. Both, CO2 and ammonium were associated with higher temperatures highlighting the need to monitor these levels more closely in hot conditions.

Table 3.4.3. Proportion of variance explained by fixed effects of each model (R^2) and significance of fixed effects for climate and air quality measures.

	Model	R^2	Date	Hour	Date-Hour	Pen	Corner	Board	Lamp
Temp	1	0.892	<.0001						
	2	0.713		<.0001					
	3	0.923			<.0001				
	3a	0.901	<.0001	<.0001					
	4	0.950			<.0001	<.0001	0.46	.01	0.87
	Final	0.950			<.0001	<.0001		<.0001	
Hum	1	0.617	<.0001						
	2	0.405		<.0001					
	3	0.663			<.0001				
	3a	0.624	<.0001	0.0037					
	4	0.802			<.0001	0.0005	<.0001	0.089	0.52
	Final	0.802			<.0001	0.0005	<.0001	0.048	
CO2	1	0.286	<.0001						
	2	0.166		<.0001					
	3	0.336			<.0001				
	3a	0.291	0.089	<.0001					
	4	0.559			<.0001	0.0007	<.0001	0.025	0.30
	Final	0.558			<.0001	0.0003	<.0001	0.041	
CO2	1	0.286	<.0001						
-sqrt	2	0.173		<.0001					
	3	0.343			<.0001				
	3a	0.289	<.0001	0.248					
	4	0.572			<.0001	<.0001	<.0001	0.059	0.48
	Final	0.572			<.0001	<.0001	<.0001	0.077	
NH3	1	0.084	<.0001						
	2	0.042		<.0001					
	3	0.117			<.0001				
	3a	0.084	<.0001	<.0001					
	4	0.378			<.0001	<.0001	<.0001	0.47	0.25
	Final	0.374			<.0001	<.0001	<.0001		
NH2	1	0.039	<.0001						
-log	2	0.033		<.0001					
	3	0.087			<.0001				
	3a	0.040	<.0001	<.0001					
	4	0.351			0.0004	0.0001	<.0001	0.24	0.08
	Final	0.349			0.0004	0.0002	<.0001		0.032

4. Application of Research

Immune and haematological traits as well as growth recorded in weaner pigs had moderate to high heritabilities. Multiple immune and haematological traits also had significant genetic correlations with growth which is also a health indicator. These findings have practical significance for pig breeding programs because they offer early selection criteria for genetic improvement of growth and disease resilience.

Pig breeding programs should focus on total and differential white blood cells which are part of the innate immune system. High levels of white blood cells at weaning had positive genetic correlations with subsequent growth until slaughter. Activation of the innate immune system is non pathogen specific and selection for these traits reduces the risk of increased susceptibility to specific diseases which has been observed for selection for immune responsiveness to specific immunizations (e.g. Wilkie *et al*. 1998). After infection with *Mycoplasma hyorhinis*, the high-immune-response (HIR) line produced antibody earlier and at higher titres than the low-immune-response (LIR) line. However, HIR showed more severe signs of arthritis.

The immune phenotype used in the selection experiment included IgG which had positive genetic correlations with growth until the end of test in this study. In contrast, growth traits until five weeks of age had lowly negative genetic correlations with IgG because resources required to produce IgG at five weeks of age were not available for growth until five weeks of age. Overall, the positive genetic correlations between IgG at five weeks and subsequent growth rate found in this study support the results of the selection experiment for immune responsiveness.

Breeders should select for higher haemoglobin levels in sows, piglets and growing pigs because “cells of nearly all forms of life require well−defined amounts of iron for survival, replication and expression of differentiated processes.” (Svoboda and Drabek 2005). In their review, the authors point out that “iron deficiency causes anaemia and suppression of immune competence, impaired resistance to infectious and parasitic diseases, growth retardation and increased mortality rate.” There was a shift in genetic correlations between haemoglobin or haematocrit and growth traits in this study which supports these statements. Haemoglobin levels or haematocrit recorded at five weeks of age had negative genetic correlations with growth traits until five weeks of age because the higher growth rate of weaner pigs reduced haemoglobin levels. However, weaner pigs with higher haemoglobin at five weeks had positive genetic correlations with subsequent growth traits. This shift in genetic correlations demonstrates that higher haemoglobin levels are required for improved growth.

Haptoglobin is another measure of innate immunity. Haptoglobin had lowly positive genetic correlations with white blood cell counts and all growth traits. Selection for higher haptoglobin levels in weaner pigs is expected to improve innate immunity and growth rate. This trait may be considered in pig breeding programs if a cost-effective measurement technique is available.

Genetic correlations between immunoglobulins and white blood cell counts were generally low indicating that selection for total and higher white blood cell counts uses different mechanisms to combat diseases than selection for immunoglobulins. Negative genetic correlations between IgM and growth traits indicate that low levels of IgM are beneficial which should be explored further.

These traits were recorded in weaner pigs at five weeks of age which has practical advantages for the application and adoption of these traits. At five weeks of age, weaners may also be recorded for juvenile IGF1 which is an early selection criterion for genetic improvement of efficient leant meat growth. The ability to record multiple selection criteria at the same time will reduce cost of recording because pigs are only handled once.

Whether a pig was medicated or not was lowly heritable in this high-health herd. This finding has important practical implications because medication records are routinely collected on farms for veterinary auditing procedures. The heritability was not significantly affected by the approach to account for non-medicated pigs which provides extra flexibility for the definition of this trait for genetic evaluations. It was not possible in the current study to estimate genetic correlations between medication incidence and immune traits because there the number of pigs with both trait groups was too low. No information was found in the literature about genetic parameters for medication records in pigs and pig breeding companies should collect information about medication records and white blood cell counts for future genetic analyses.

The environmental descriptor based on growth rate was associated with incidence of medication, e.g. a higher mean growth rate of a group of pigs was associated with a lower mean medication incidence. This finding confirms the concept to use growth as a health indicator of clinical and sub-clinical diseases on farms.

Methodology was developed to separate specific, non-infection challenges like heat stress from environmental descriptors which provides opportunities to select pigs more specifically in regard to their ability to cope with individual environmental stressors. This methodology can be applied to disease resilience where environmental stressors are due to pathogen challenges.

4.1. Publications arising from this Research

Outcomes from this Research have been prepared for multiple conferences (AAABG, APSA and WCGALP) and a refereed journal paper has been published.

Guy, SZY, Hermesch, S, Thomson, PC (2015) Backfat as an Environmental Descriptor in Defining Growth Rate of the Pig: A GxE Analysis. In 'Proc. Assoc. Advmt. Anim. Breed. Genet. Lorne, Australia'. Volume 21 pp. 457-460. (Association for the Advancement of Animal Breeding and Genetics)

Guy, SZY, Harper, J, Li, L, Thomson, PC, Hermesch, S (2017a) 'Extension of environmental descriptors used to analyse sire by environment interaction for growth of pigs, 22nd Conference of the Association for the Advancement of Animal Breeding and Genetics.' Townsville, Australia, -. Australia)

Guy, SZY, Li, L, Thomson, PC, Hermesch, S (2017b) Contemporary group estimates adjusted for climatic effects provide a finer definition of the unknown environmental challenges experienced by growing pigs. Journal of Animal Breeding and Genetics doi: 10.1111/jbg.12282.

Guy, SZY, Li, L, Thomson, PC, Hermesch, S (2017c) Seasonal effects can be separated from other challenges in the pig environment using time series analysis. Animal Production Science 57, 2463.

Guy, SZY, Li, L, Thomson, PC, Hermesch, S (2018) Genetic parameters for health of the growing pig using medication records, 11th World Congress on Genetics Applied to LIvestock Production [Submitted]. Auckland, New Zealand.

Hermesch, S, McKenna, T, Bauer, MM, Sales, N (2017) The effect of dam parity on growth, white blood cell count, haemoglobin and immunoglobulin levels of weaner pigs, 16th conference of Australasian Pig Science Association, Animal Production Science, 57, 2482.

Hermesch, S, Luxford, BG (2018) Genetic parameters for white blood cells, haemoglobin and growth in weaner pigs for genetic improvement of disease resilience, 11th World Congress on Genetics Applied to Livestock Production [Submitted]. Auckland, New Zealand.

5. Conclusion

Moderate to high heritabilities and genetic variability were found for immune and haematological traits recorded in weaner pigs at five weeks of age. Estimates of genetic correlations provided information about genetic relationship between these traits. Genetic correlations between total and differential white blood count were high. Similarly, a number of haematological traits were genetically the same trait.

Genetic correlations between immunoglobulins and haptoglobin were generally low and not significantly different to zero. Low levels of immunoglobulin M and high levels of haptoglobin in weaner pigs had favourable genetic correlations with growth of pigs post weaning.

Incidence of medication was a heritable trait in this high-health herd. Medication of pigs has economic and welfare costs and this trait should be incorporated in breeding objectives of pig breeding programs.

Immune and haematological traits should be recorded in weaner pigs at five weeks of age for genetic improvement of disease resilience. In addition, weight of pigs should be recorded at weaning and at five weeks in order to measure growth of weaners during this critical period post weaning. Outcomes from this study warrant recording of these immune, haematological and post-weaning growth traits in other populations that have information about feed intake, juvenile IGF1 or post-weaning survival available.

6. Limitations/Risks

Information about genetic parameters of immune traits is still limited and most studies, including the current study, were based on small sample sizes of less than 1000 animals. Therefore, there is the risk that some estimates of genetic parameters may not be repeated in other populations because estimates of genetic parameters have a certain level of uncertainty. This aspect was considered by estimating genetic parameters among all available immune traits including traits that were genetically the same traits. The consistency of genetic correlations, e.g. traits that were genetically the same should have similar genetic correlations with other traits, was considered specifically as an approach to evaluate reliability of estimates of genetic.

There may be the risk that health status affects genetic parameters for immune, haematological and growth traits and estimates may not be applicable across herds with different health status. This aspect could not be addressed in the current study and it highlights the need to record immune and haematological traits more widely in commercial pig populations.

7. Recommendations

As a result of the outcomes in this study the following recommendations have been made:

White blood cell counts should be recorded in weaner pigs at five weeks of age for genetic improvement of disease resilience.

Haemoglobin levels should be monitored more widely on pig farms and hand-held devices provide cost-effective measurement tools that can be used on farms.

Weight of pigs should be recorded at weaning and at five weeks in order to measure growth of weaners during this critical period post weaning.

Haptoglobin may be considered in pig breeding programs if a cost-effective measurement technique is available.

Medication records available on farms for veterinary auditing procedures should be incorporated in electronic databases. The incidence of medication of pigs was heritable and should be considered in genetic evaluation systems.

The economic value of medication incidence should be derived in order to include this trait in pig breeding objectives in addition to other traits describing efficiency, productivity and survival of pigs post weaning.

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