

The Association Between Sire Estimated Breeding Value for Antibody-Mediated Immune Response (AMIR) and Offspring AMIR Phenotype

M. Emam^{1,2}, M. Paibomesai¹, K. Thompson-Crispi^{1,2}, F. Schenkel², F. Miglior^{2,3}, M. Sargolzaei^{2,4}, B. Mallard^{1,2}

¹Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON, ²Centre for Genetic Improvement of Livestock, University of Guelph, ³Canadian Dairy Network, Guelph,

⁴The Semex Alliance, Guelph, ON, Canada



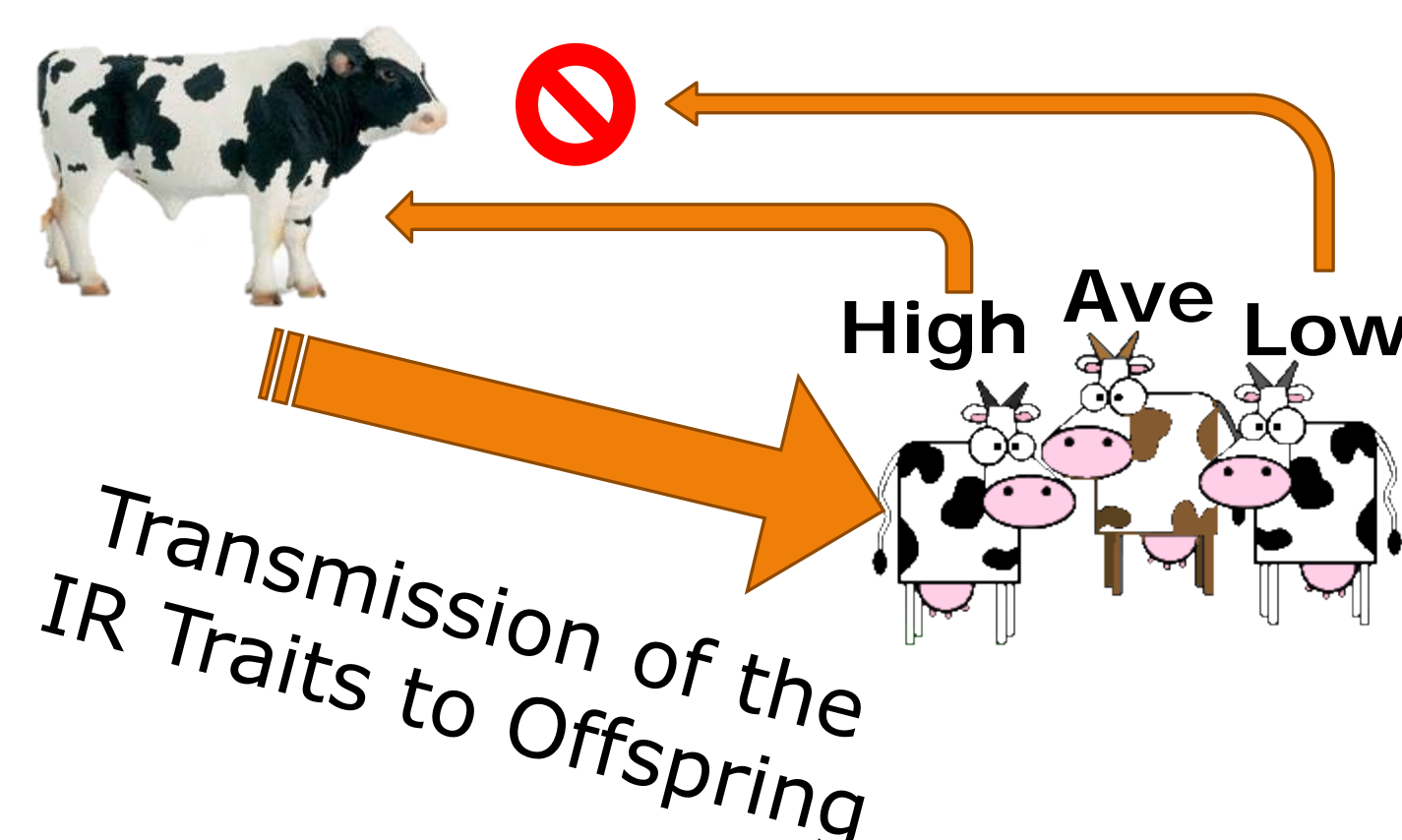
Introduction

- The **immune system** is a complex network of tissues, cells and molecules critical to the **maintenance of animal health**.
- The Two key components of the adaptive immune system, antibody- (**AMIR**) and cell-mediated immune response (**CMIR**) are critical to the control of extracellular and intracellular pathogens, respectively.
- The **High Immune Response (HIR)TM** technology has been successfully applied to measure the performance of the adaptive immune system of dairy cattle.

❖ **Classify animals into high, average and low immune responders.**

Objective

Does the actual AMIR performance of an offspring associate with sire AMIR EBV as genetic theory would suggest?



Materials and Methods

Sample Population

The data set consisted of 1,044 IR-phenotyped Holstein bulls (n=763) and cows (n=281). A total of 254 sire-offspring IR-tested pairs were identified. Two groups of high and low responders, each consisting of 50 sire-offspring pairs, were selected based on AMIR performance of the offspring (Detailed sample population structure).

Phenotypic Analysis:

$$y_{ijkl} = \mu + h_i + p_j + m_k + hp_{ij} + \beta_1 \times a_l + \beta_2 \times d_l + e_{ijkl} \quad [1]$$

[Click here for description](#)

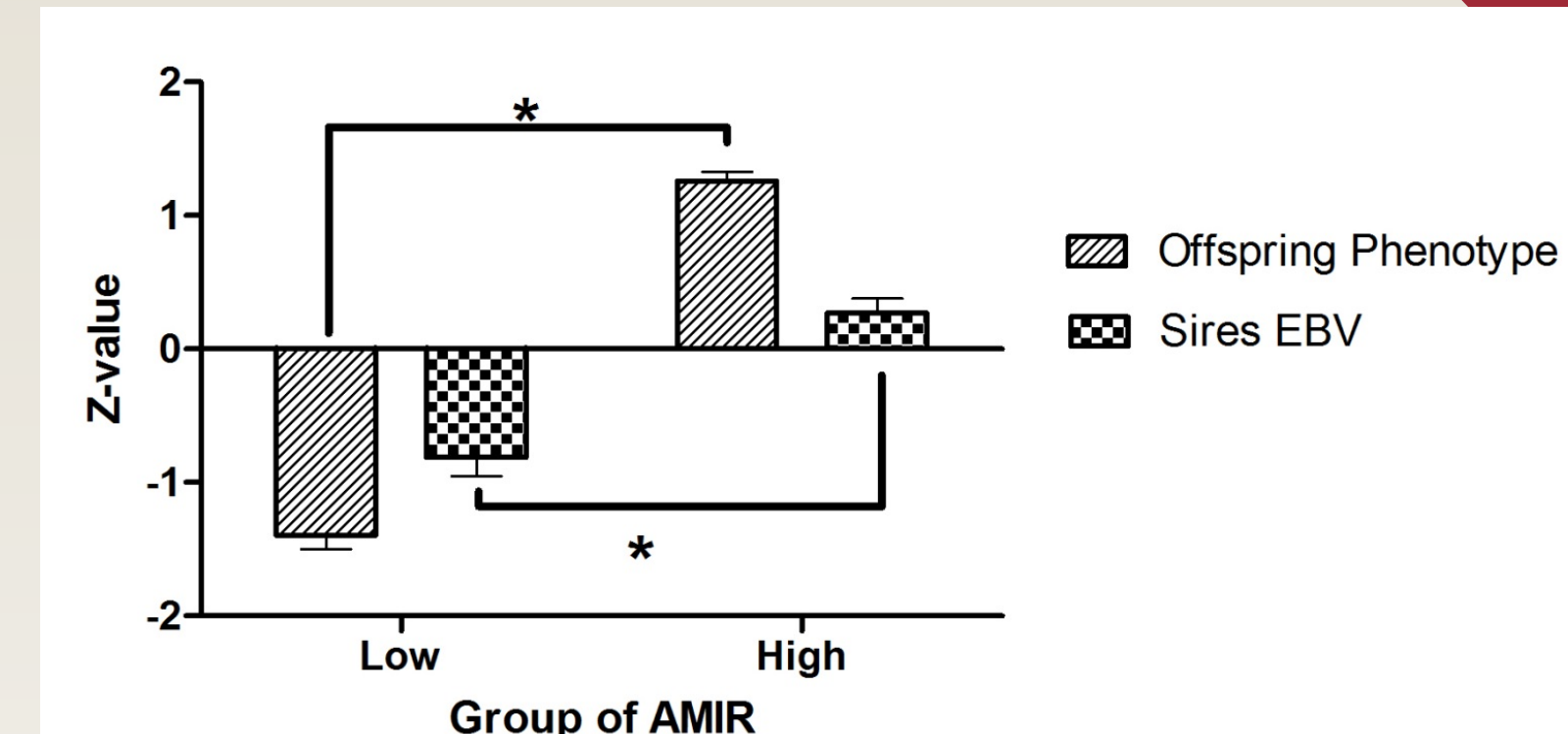
Standardized Z-values of the animals' residual effects in model [1] (adjusted log₁₀ of OD at day 14) were calculated and animals with the 50 highest and 50 lowest Z-values were categorized into high responder and low responder groups, respectively.

Genetic Analysis:

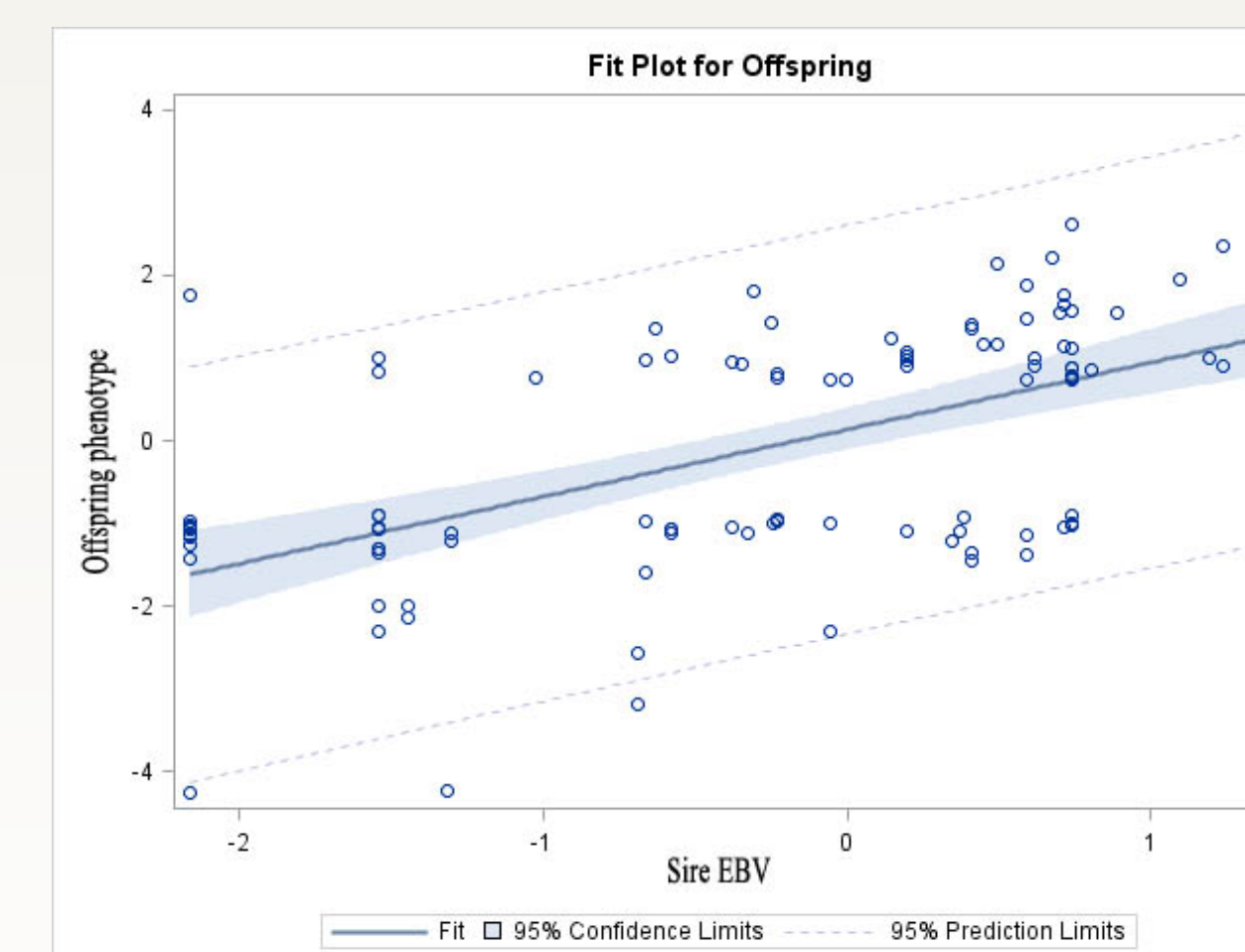
To estimate (co)variance components and breeding values for AMIR, the same statistical model [1] as for the phenotypic analysis was used, but with the addition of the animal random additive genetic effect (u). The covariance between animals was modeled by the additive genetic relationship matrix, using the pedigree information extracted from Canadian Dairy Network database, including a total of 23,913 records.

Results and Discussion

- ✓ The mean AMIR phenotype Z-value of the high responder offspring was +1.26 and the mean AMIR phenotype Z-value of the low responder offspring was -1.39 ([Figure 1](#)). This difference was statistically significant ($p < 0.0001$).
- ✓ The mean EBV Z-values among the sires of high responder offspring was +0.27 and the mean was -0.81 among sires of low responder offspring ([Figure 1](#)).
- ✓ In addition, **the linear regression of the adjusted phenotypes of offspring on the sires' EBV was positive ($b = 0.81$) and significant ($P < 0.0001$)** ([Figure 2](#)).



[Figure 1](#)



[Figure 2](#)

- ✓ The significant association between sires' EBV and offspring phenotype shows the possibility of genetic improvement for AMIR in dairy Holstein through sire selection.
- ✓ The contribution of genetic potential of sires for AMIR is similar to some other production traits, such as milk production.
- ✓ The positive association between sires' EBVs and their offsprings' phenotypes for AMIR emphasizes the possibility of breeding for higher AMIR in Holstein dairy cattle.
- ✓ Since AMIR is associated with resistance to diseases, such as mastitis, disease occurrence can be decreased by using bulls with superior immune responses.

Acknowledgement

This research was funded by grants to Dr. B. Mallard from NSERC and OMAF. Authors appreciate Dr. D.Hodgins for his constructive comments.

References

The Association Between Sire Estimated Breeding Value for Antibody-Mediated Immune Response (AMIR) and Offspring AMIR Phenotype

M. Emam^{1,2}, M. Paibomesai¹, K. Thompson-Crispi^{1,2}, F. Schenkel², F. Miglior^{2,3}, M. Sargolzaei^{2,4}, B. Mallard^{1,2}

¹Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON, ²Centre for Genetic Improvement of Livestock, University of Guelph, ³Canadian Dairy Network, Guelph, ⁴The Semex Alliance, Guelph, ON, Canada

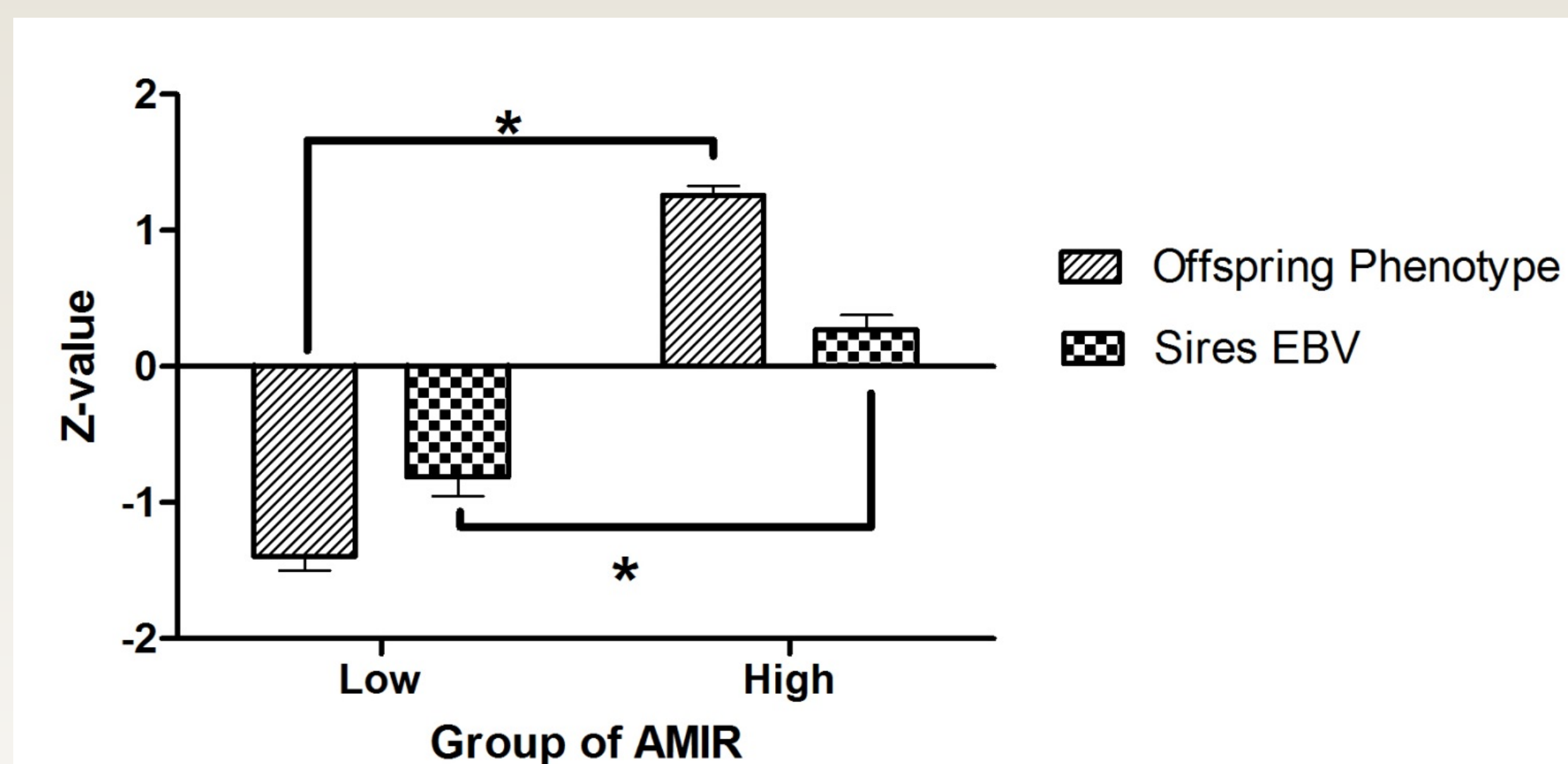


Figure 1- Mean standardized adjusted offspring phenotypes in high responder and low responder groups and mean of the corresponded sire EBVs . * indicates significant differences (p < 0.0001).

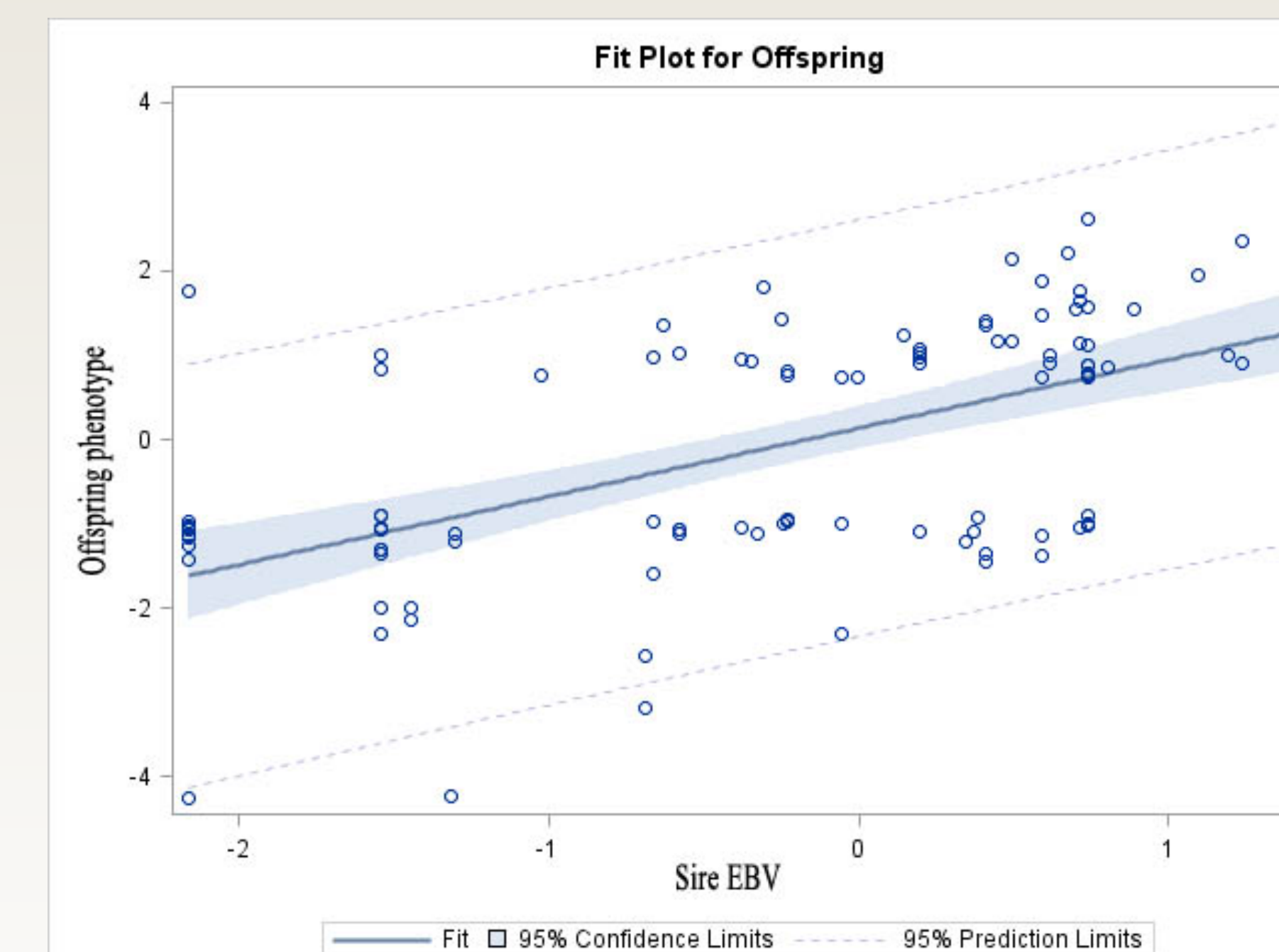


Figure 2 - Regression analysis of standardized adjusted offspring phenotypes (y axis) on sire EBVs (x axis).



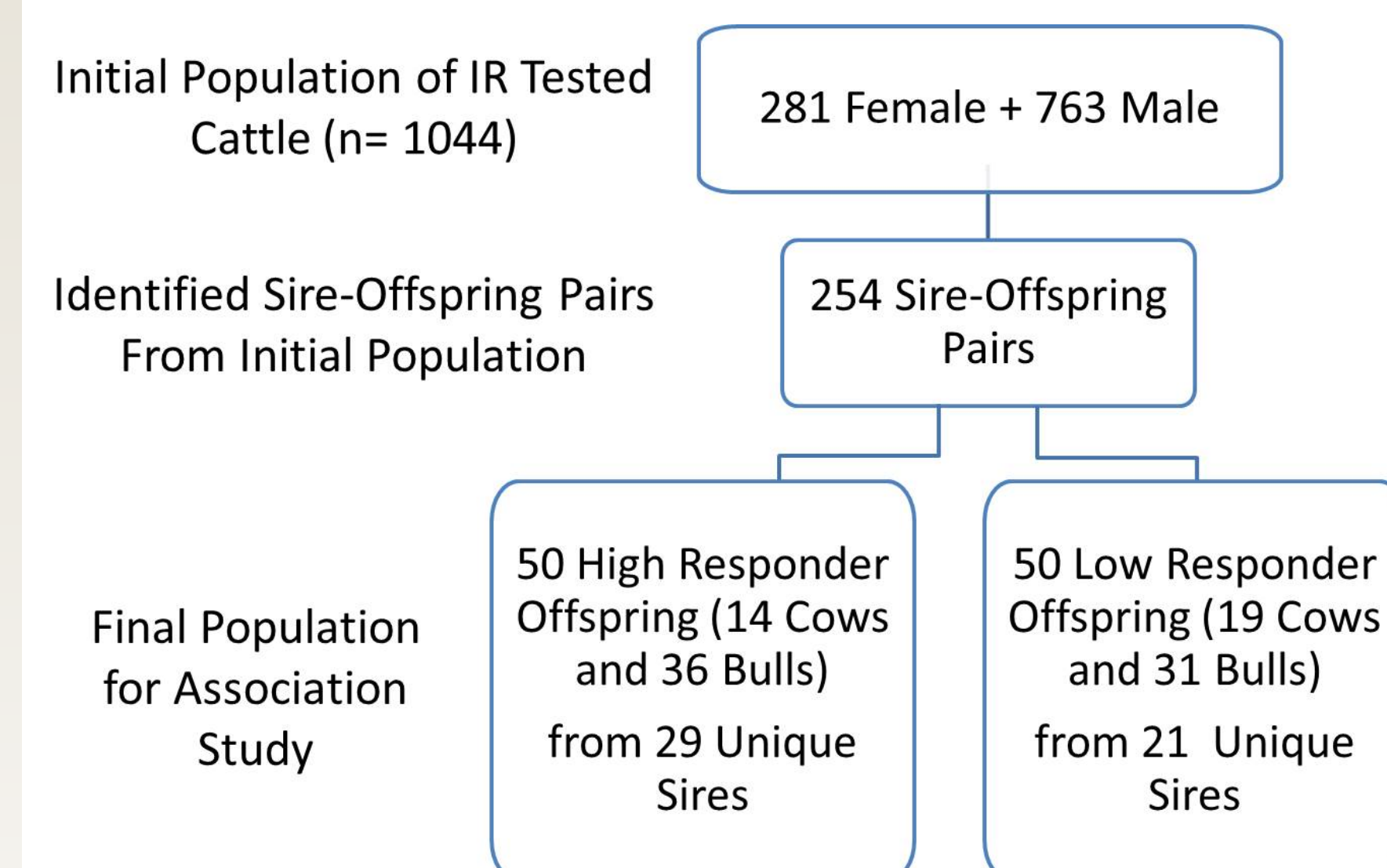
The Association Between Sire Estimated Breeding Value for Antibody-Mediated Immune Response (AMIR) and Offspring AMIR Phenotype

M. Emam^{1,2}, M. Paibomesai¹, K. Thompson-Crispi^{1,2}, F. Schenkel², F. Miglior^{2,3}, M. Sargolzaei^{2,4}, B. Mallard^{1,2}

¹Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON, ²Centre for Genetic Improvement of Livestock, University of Guelph, ³Canadian Dairy Network, Guelph, ⁴The Semex Alliance, Guelph, ON, Canada



Sample population structure



Phenotypic Analysis:

$$y_{ijkl} = \mu + h_i + p_j + m_k + hp_{ij} + \beta_1 \times a_l + \beta_2 \times d_l + e_{ijkl} \quad [1]$$

Where y_{ijkl} = Log10 of OD at day 14 for the l^{th} animal; h_i = fixed effect of i^{th} housing facility; p_j = fixed effect of j^{th} phase of testing; t_k = fixed effect of k^{th} pregnancy status (heifer, pregnant cow, non-pregnant cow and bulls); hp_{ij} = interaction effect of i^{th} phase and j^{th} housing facility; β_1 = linear coefficient of the fixed regression on age (a_l) of the l^{th} animal (in months); β_2 = linear coefficient of the fixed regression on = Log10 of OD at day 0 (d_l) of the l^{th} animal; e_{ijkl} = random residual effect.

References

- Paibomesai, M., Hussey, B., Nino-Soto, M., et al., (2013). Can. J. Vet. Res. 77, 54–62.
- Thompson-Crispi, K.A., Sewalem, A., Miglior, F., et al., (2012). J. Dairy Sci. 95, 401–409.
- Thompson-Crispi, K.A., Hine, B., Quinton, M., et al. (2012). J. Dairy Sci. 95, 3888–3893.
- Wagter, L.C., Mallard, B.A., Wilkie, B.N., et al., (2000). J. Dairy Sci. 83, 488–498.
- Wiggans, G.R., Cooper, T.A., VanRaden, P.M., et al., (2011). J. Dairy Sci. 94, 6188-6193.
- Wilkie, B.N., Mallard, B.A., (1999). Vet. Immunol. Immunopathol. 72, 231–235.

