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Interpretation of dominant and additive variances from genomic modelsZ.G. Vitezica¹, L. Varona² and A. Legarra³¹INRA, INPT, UMR 1289 TANDEM, Avenue de l'Agrobiopole, PB 32607, F31326 Castanet-Tolosan, France,²Facultad de Veterinaria, Universidad de Zaragoza, Miguel Servet, 177, 50013 Zaragoza, Spain, ³INRA, UR 631 SAGA, 24 Chemin de Borde Rouge, 31326 Castanet-Tolosan, France; zulma.vitezica@ensat.fr


Genomic evaluation models typically fit only SNP additive effects. However, it is possible to include also dominant SNP effects. Under quantitative genetics theory, breeding values (A) of individuals are generated by substitution effects, which involve both additive and dominant effects, whereas the dominance deviations (D) only include dominant effects. From the genotypic value, we can also define (A*) and (D*) as the parts attributable to the additive and dominant effect of the markers. Note that this A* is not a breeding value. We show that the variance of genotypic values due to additive effects of markers ($\sigma_{A^*}^2$), is not equal to additive genetic variance (among breeding values) and that the variance of genotypic dominant effects ($\sigma_{D^*}^2$) is not the dominance variance. In fact, only when the allele frequencies are equal to 0.5 all variances are identical. In addition, dominant relationship matrices constructed from markers depend on which of the two decompositions is used. As the total genetic variance can be partitioned into additive and dominance components, we know that $\sigma_A^2 + \sigma_D^2$ is equal to $\sigma_{A^*}^2 + \sigma_{D^*}^2$. In our study, we show that it is easy to define a one on one relation between 'breeding' and 'genotypic' additive and dominance variance and that even being equivalent, the estimated variance components will be different. The 'genotypic' model overestimates the dominance variance and, consequently, underestimates additive variance. We illustrate these results with parameter estimations in mice data. The differences between 'breeding' and 'genotypic' model must be taken into account in the interpretation and use of genomic predictions and genomic estimates of variance components.

Genomic prediction of heterosis for egg production traits in White Leghorn crossesE.N. Amuzu-Aweh^{1,2}, P. Bijma¹, B.P. Kinghorn³, A. Vereijken⁴, J. Visscher⁴, J.A.M. Van Arendonk¹ and H. Bovenhuis¹¹Wageningen University and Research Centre, Animal Breeding and Genomics Centre, P.O. Box 338, 6700 AH Wageningen, the Netherlands, ²Swedish University of Agricultural Sciences, Department of Animal Breeding and Genetics, P.O. Box 7023, 750 07 Uppsala, Sweden, ³University of New England, School of Environmental and Rural Science, NSW 2351, Armidale, Australia, ⁴Hendrix Genetics, Institut de Sélection Animale B.V., P.O. Box 30, 5830 AE Boxmeer, the Netherlands; esinam.amuzu@wur.nl

The genetic basis of heterosis has puzzled geneticists for decades. Accurate prediction of heterosis would benefit animal and plant breeding by identifying parental lines suitable for crossbreeding. Prediction of heterosis has a long history with mixed success, partly due to low numbers of genetic markers and/or small data sets. We investigated prediction of heterosis for egg number, egg weight and survival time in domestic White leghorns, using ~400,000 individuals from 47 crosses and allele frequencies on ~53,000 genome-wide SNPs. For a single locus, heterosis is solely due to dominance and proportional to the squared difference in allele frequency between parental lines (SDAF). We, therefore, used linear mixed models where phenotypes of crossbreds were regressed on the SDAF between parental lines. Accuracy of prediction was determined using leave-one-out cross-validation. SDAF predicted heterosis for egg number and weight with an accuracy of ~0.5, but not for survival time. Heterosis predictions allowed pre-selection of pure lines prior to field-testing, saving ~50% of field-testing costs with only 4% loss in heterosis. Accuracies from cross-validation were lower than those from the model-fit, indicating that values in the literature may be overestimated. Cross-validation also indicated dominance cannot fully explain heterosis. Nevertheless, the dominance model yielded a considerable accuracy, clearly greater than that of a general-specific combining-ability model. Our results show that SDAF can be used to predict heterosis in commercial layer breeding.

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