combinations with the *DGAT1* mutation was higher than for the individual SNP, rather than a true epistatic interaction.

Five additive × additive interactions were found significant ($P < 0.0001$) for CI in Holsteins with a FDR of 18%. However, none of these was validated ($P < 0.01$) in the Jersey population.

Table 2. *P*-value thresholds, number of significant pairwise additive × additive interactions and calculated false discovery rates (FDR) for milk yield (MY) and calving interval (CI)

		Discovery			Validation			
Trait	No. of interactions	*P*	Holstein	FDR (%)	*P*	Jersey	FDR (%)	Same Dir.[1]
MY	255,255	10^{-7}	3700	0	10^{-5}	165	0	163
CI	9,180	10^{-4}	5	18	0.01	0	NA	NA

[1]Number of same direction SNP effects in discovery and validation populations

Implications. One critical parameter determining the power of a GWAS is the amount of LD between the observed SNP and the unobserved causal variant. The success of a GWAS in identifying QTLs with additive effects is controlled by r^2 (r is the correlation between genetic marker and causative mutation) while detection of dominance or pairwise additive by additive effects depends on r^4. This indicates a much higher reliance on LD in searching for non-additive effects compared to additive effects, if LD between the markers and QTL is incomplete (Wei *et al.* 2014). This, and possibly the relatively small size of individual dominance and epistatic effects, was reflected in results of this study in which a larger number of additive markers were identified than the markers with dominance and epistasis effects for both traits under investigation.

The standard in reporting GWAS results is validation and before genotype-phenotype relationships can be used in selection decisions, they should be replicated in an independent population to confirm generalized effects in multiple populations. Validation of GWAS results across breeds can refine QTL regions to narrower intervals and is powerful in identifying positional candidate genes. This is because the extent of LD across cattle breeds is limited in contrast to within a breed, where considerable LD can be maintained in intervals up to 1 Mbp as a result of a relatively small effective population size. We validated a lower number of non-additive genetic associations than additive effects such that very few dominance effects for MY and CI were confirmed and no epistasis effect was common across Holstein and Jersey cows for CI. This trend is in agreement with the fact that the higher dependence on LD in searching for dominance and epistatic effects compared to additive effects significantly decreases the chance of validating associations in independent populations for non-additive effects of the markers (Wei *et al.* 2014).

CONCLUSION

We identified and validated a small number of SNPs with suggested dominance effects on MY and CI in Australian Holstein and Jersey cows. Given our results, identifying non-additive gene actions using single SNP regression in a GWAS setting will require very large datasets to capture the likely very small individual non-additive genetic effects.

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