File S1

Supplementary Methods

For all univariate traits, the proportion of phenotypic variance accounted for by common environment (vial) was less than one percent, and highly non-significant in all cases. While common environment is often cited as an important source of variance, the lack of evidence here is not surprising. The wing traits examined are aligned inter-landmark distances that describe wing shape variation free from size variation, a trait that is much more likely to be influenced by environment. Additionally, flies were reared at a low density, which may have mitigated any effects of larval competition that could contribute to common environmental variance. This finding is also consistent with the lack common environmental variance found for wing shape phenotypes in *Drosophila melanogaster* (MEZEY and HOULE 2005). Therefore, we excluded common environment as a random effect from our models, and proceeded to examine whether additive x additive epistasis contributed substantially to the phenotypic variance in these traits.

There are two lines of evidence that strongly suggest additive x additive epistasis contributes marginally to phenotypic variance, compared to dominance, in our experiment. First, when comparing univariate models that estimate additive x additive epistasis to those that estimate dominance, in all cases the likelihoods of the two models are almost identical (Table S1), despite the substantially increased power we have for detecting epistasis, that comes from an AA-matrix with 20 times more non-zero elements than the D-matrix (1 982 541 vs. 101 272 non-zero elements in the AA and D matrices, respectively). Of this difference in non-zero elements, 42% were sizeable relatedness coefficients between 0.0156215 and 0.25, adding considerable power to detect epistatic variance. The variance component estimates for epistasis vs. dominance were also very similar for each trait, although in all cases the epistatic estimates were higher on average by 16% (Table S1). The more powerful epistatic models should, however, yield more precise estimates of the variance components. In order to determine whether epistatic estimates were, in fact, more precise, we relied on large sample theory that indicates maximum likelihood estimates are normally distributed with a covariance matrix equal to the inverse of the information matrix (MEYER and HILL 1992). We took 50 000 samples from these multivariate normal distributions for models that estimated epistasis vs. dominance, in order to generate sampling distributions for each of these parameters for each trait. Here, the mean of these distributions converge on our variance component estimates from REML, with the spread indicating the precision of the estimates. In no cases were sampling distributions from models estimating epistasis more precise than models estimating dominance (Figure

S1), consistent with the near-identical likelihoods we found for the two models for all traits. This finding is consistent with variance component estimates arising from dominance and not epistasis.

Our second line of evidence comes from examining the trait, 'fitness'. This univariate trait is unlike our wing shape traits, in that it does not have significant additive genetic variance (see results). It follows, then, that we are unlikely to pick up substantial additive x additive epistatic variance. However, we observed the same patterns of both variance component estimates and sampling distributions when we compared epistatic vs. dominance models for fitness to those for wing shape traits. This, again, indicated that although we cannot tease apart epistatic and dominance variance, the epistatic component of variance is likely to contribute little to our dominance estimates. We, therefore, excluded additive x additive epistasis from our models and subsequently examined additive genetic, dominance genetic, and residual variance only.

Meyer K., Hill W. G., 1992 Approximation of sampling variances and confidence intervals for maximum likelihood estimates of variance components. *Journal of Animal Breeding and Genetics* **109**: 264–280.

Mezey J. G., Houle D., 2005 The dimensionality of genetic variation for wing shape in Drosophila melanogaster. *Evolution* **59**: 1027–1038.

Files S2-S3

Available for download at www.genetics.org/lookup/suppl/doi:10.1534/genetics.115.175489/-/DC1

File S2 Pedigree

File S3 Raw Data

Table S1 Variance component estimates and corresponding log-likelihoods of models fitting 'animal' and either 'dominance' or 'epistasis' as random effects. In all cases the log-likelihoods of the two models are almost identical, with variance component estimates for 'animal' and 'residual' also corresponding well between the two models.

Trait	Residual	Animal	Dominance	Additive x Additive Epistasis	Log Likelihood
Fitness	0.733	0.001	0.265	-	-1422.461
	0.737	0.000	-	0.262	-1422.525
aLM2-4	0.409	0.562	0.029	-	-2041.995
	0.405	0.558	-	0.036	-2041.742
aLM2-5	0.270	0.408	0.319	-	-2059.721
	0.225	0.360	-	0.410	-2059.465
aLM2-8	0.225	0.551	0.209		-1898.784
	0.213	0.544	-	0.231	-1898.82
aLM3-7	0.225	0.465	0.309	-	-2000.418
	0.197	0.441	-	0.362	-2000.675
aLM3-9	0.386	0.292	0.317	-	-2195.059
	0.354	0.262	-	0.380	-2195.264
aLM4-5	0.271	0.482	0.249	-	-2023.281
	0.233	0.440	-	0.326	-2022.891
aLM5-6	0.390	0.475	0.129	-	-2075.378
	0.382	0.469	-	0.144	-2075.242
aLM5-8	0.270	0.488	0.257	-	-2054.424
	0.221	0.432	-	0.355	-2053.947

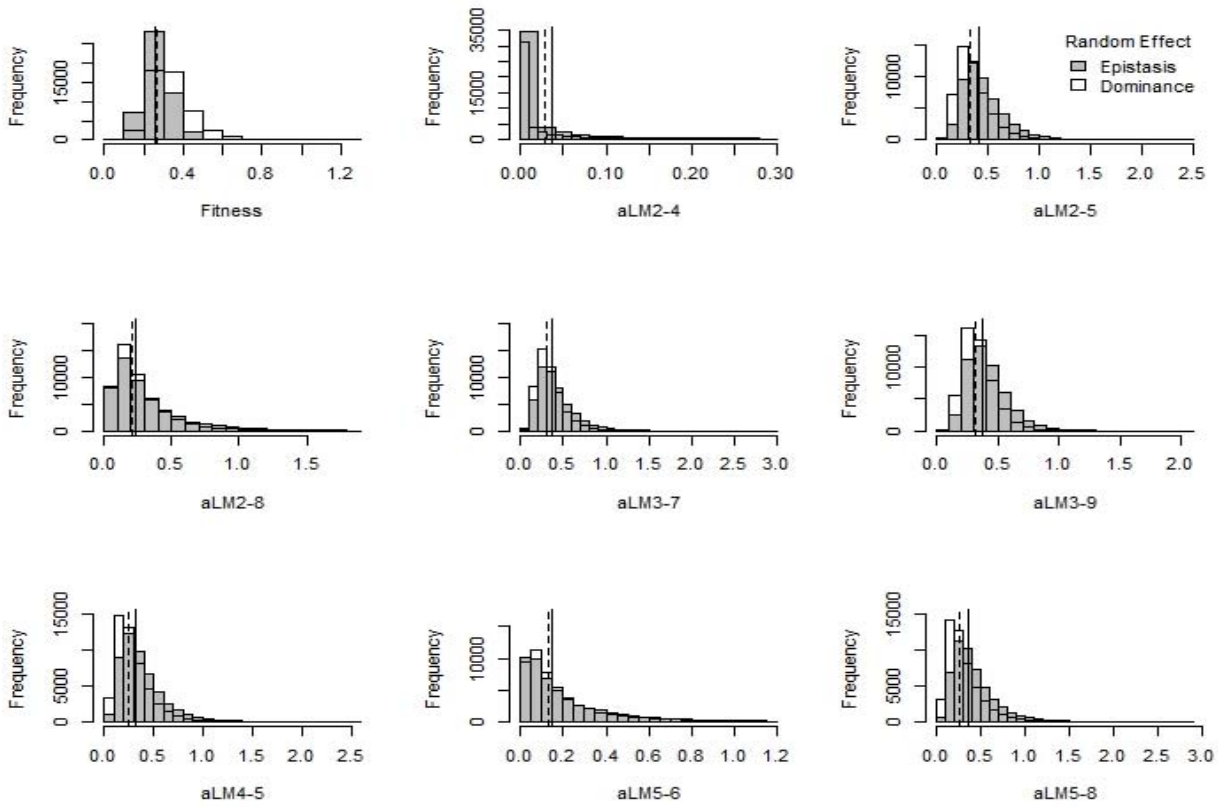

Figure S1 Sampling distributions for epistasis and dominance from 50 000 samples of the multivariate normal distribution defined by our data for each univariate wing trait and fitness.

Solid and dashed vertical lines indicate the variance component estimates for epistasis and dominance, respectively.

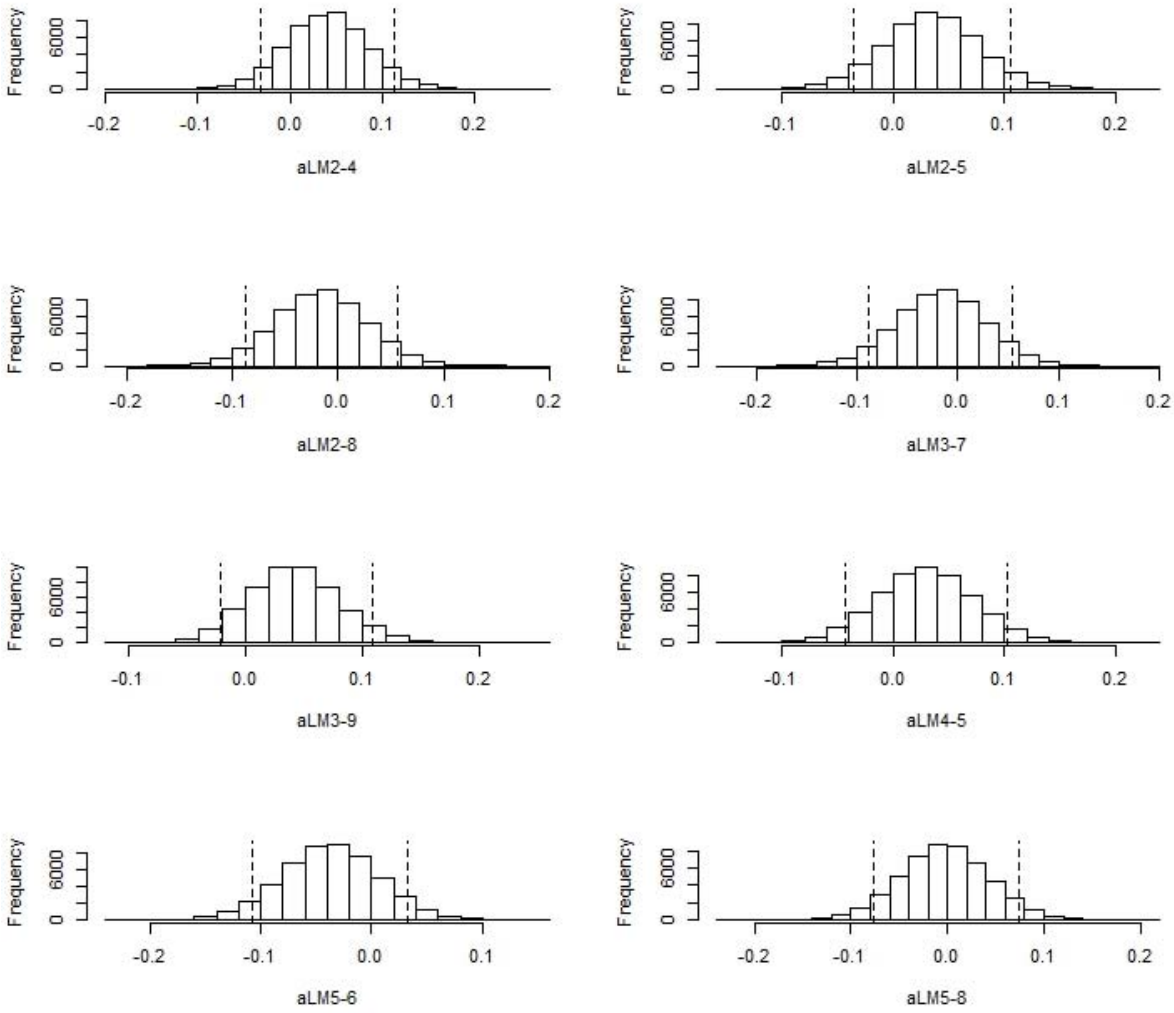

Figure S2 Sampling distributions for the additive genetic covariance between each wing trait and fitness.

Vertical dashed lines indicate 95% confidence intervals of the covariances.