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Can dominance genetic variance be ignored in evolutionary quantitative genetic analyses of wild populations?

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Accurately estimating genetic variance components is important for studying evolution in the wild. Empirical work on domesticated and wild outbred populations suggests that dominance genetic variance represents a substantial part of genetic variance, and theoretical work predicts that ignoring dominance can inflate estimates of additive genetic variance. Whether this issue is pervasive in natural systems is unknown, because we lack estimates of dominance variance in wild populations obtained in situ. Here, we estimate dominance and additive genetic variance, maternal variance, and other sources of nongenetic variance in eight traits measured in over 9000 wild nestlings linked through a genetically resolved pedigree. We find that dominance variance, when estimable, does not statistically differ from zero and represents a modest amount (2-36%) of genetic variance. Simulations show that (1) inferences of all variance components for an average trait are unbiased; (2) the power to detect dominance variance is low; (3) ignoring dominance can mildly inflate additive genetic variance and heritability estimates but such inflation becomes substantial when maternal effects are also ignored. These findings hence suggest that dominance is a small source of phenotypic variance in the wild and highlight the importance of proper model construction for accurately estimating evolutionary potential.

KEY WORDS: Animal model, *Cyanistes caeruleus*, dominance genetic variance, juveniles.

Predicting the evolution of quantitative traits in wild animals requires determining how these traits respond to selection (Lynch and Walsh 1998). Because the evolutionary response depends upon additive genetic variance, it is this component of genetic variance that has been the focus of many empirical quantitative genetic studies. Additive genetic variance is, however, only one segment of genetic variance, the other two being nonadditive: dominance and epistatic variances. Compared to additive genetic variance, estimates of nonadditive genetic variance components are uncommon. Although studies in animal and plant breeding, lab experiments, and humans have provided estimates of dominance variance (reviewed in Wolak and Keller 2014), studies estimating nonadditive genetic variance in wild animal populations in situ (i.e., under ecologically relevant environmental conditions) remain scarce. This lack of knowledge of dominance variance in wild populations hampers critical evaluation of some of the core theories in evolutionary genetics. In particular, the Fisherian assumption that population size is (near) infinite implies that dominance genetic variance can be ignored. This is because nonadditive genetic effects average out to zero when a (near) infinite number of genetic backgrounds are present in a population (Fisher 1958). Furthermore, Hill et al. (2008) predicted using a biallelic single locus model that dominance variance can at best explain a small proportion of genetic variance under drift-mutation equilibrium and speculated the same scenario would apply under selection. In contrast, under a Wrightian view of populations as finite, structured, and often small, nonadditive genetic effects cannot be ignored in shaping local adaptation and fitness landscapes (Wright 1931; Whitlock et al. 1995; Fenster et al. 1997). From this perspective, dominance variance could be

pronounced in the wild, because most natural population are considered to have low effective population sizes (Roff 1997).

With theory taking two opposite stands regarding the importance of dominance, the resolution of this question lies in empirical work. Compilation of empirical estimates, however, has not led to a consistent conclusion. Notably, Hill et al. (2008) conducted a literature review showing that narrow-sense heritability is only a little less than repeatability and heritability in the broad sense, and thus concluded that dominance variance is negligible. In contrast, Crnokrak and Roff (1995) found that life history traits are affected particularly strongly by dominance and the more recent and more extensive compilation by Wolak and Keller (2014) underlines that the dominance variance is a pronounced fraction of total genetic variance in various traits (on average 38%; $n =$ 553 point estimates). However, most of these estimates are based on domesticated organisms or laboratory studies, where the environmental variance is reduced to a minimum and the genetic architecture has likely been altered by domestication or adaptation to the lab environment. Two studies conducted in humans found dominance variance to be either negligible or to account for most of the genetic variance in life history and physiological traits (Abney et al. 2000; Kosova et al. 2010). One study in zoo-housed orangutans showed that dominance accounts for most of the genetic variance in personality (Adams et al. 2012). In a few field trials studies conducted in trees, the proportion of dominance to genetic variance was found to be 1 for survival (Mullin et al. 1992), 0.59 and 0.53 for diameter (Waldmann et al. 2008; Costa et Silva et al. 2010), and 0.34 and 0.01 for height (Mullin et al. 1992; Waldmann et al. 2008). Finally, one study using a combination of a long-term pedigree and cross-fostering design in wild birds showed that variance explained by the nest of origin (after accounting for additive genetic and nest of rearing effects), which is the upper limit to dominance variance, was small in proportion to genetic variance (0.09; Merilä et al. 2001). Clearly, more estimates of dominance in wild populations in situ are needed to inform us of the magnitude of its effects sizes in the wild.

The main limitation for inferring dominance variance in wild animal populations in situ is that most organisms do not easily allow obtaining the necessary knowledge of relatedness between individuals. Indeed, dominance variance contributes to the phenotypic resemblance of both full-sibs and double first cousins, the latter category of relatives being very rare in the wild (Wolak and Keller 2014). Hence, the most viable option for inferring dominance is—in principle—based on comparing the phenotypic resemblance of full-sibs (affected by additive genetic and dominance effects) to that of half-sibs (affected by additive genetic effects alone) (Cockerham and Weir 1977; Lynch and Walsh 1998). In many organisms where parentage can be readily assessed, full and half-sibs typically share common environment and maternal effects, which are hence confounded with additive and dominance genetic effects. Multigenerational phenotypic information on full sibs from different breeding attempts, maternal and paternal half-sibs, as well as more distant relatives is then additionally required to avoid complete confounding (Wolak and Keller 2014). Clearly, therefore, inferring dominance in a wild population is demanding. On the other hand, simulations show that ignoring dominance variance results in inflated estimates of additive genetic variance (Ovaskainen et al. 2008) thereby overestimating evolvability. Importantly, the strength of this bias depends on the amount of dominance variance. Under controlled environmental conditions, dominance variance was shown to represent a large (>0.5) fraction of the total genetic variance in fitness-related traits (Crnokrak and Roff 1995; Wolak and Keller 2014). It is hence possible that by ignoring dominance, additive genetic variance is strongly overestimated in wild populations for traits that are under selection. In addition, the tight relationship between maternal and both genetic variances suggests that even greater biases can be expected when also maternal effects are ignored, which corresponds to a commonly fitted model in ecology and evolution. Such overestimation of additive genetic variance could provide one explanation for why wild populations suffer from an inflated expectation of evolutionary response compared to what is observed (Pujol et al. 2018).

Our objective in this article is to test whether dominance can be a nonnegligible part of genetic variance in a wild population, and whether ignoring such dominance variance in quantitative genetic analyses of traits in a wild population entails inflation of additive genetic variance. To this end, we first obtain point estimates of dominance genetic variance (in addition to other sources of variance) in a wild population of blue tits. We focus on eight morphological and behavioral traits measured in nestlings. We estimate (when possible) additive genetic, dominance, common environmental, and maternal variances underlying each of these traits. After thus establishing what are realistic effect sizes of dominance and other sources of variance in this wild population, we use simulations to demonstrate that our data structure allows providing unbiased estimates of all these variance components and that ignoring dominance causes little bias in additive genetic variance and heritability estimates. In contrast, we show that models ignoring both dominance and maternal variance result in clearly inflated estimates of additive genetic variance and heritability.

Material and Methods

DATA COLLECTION

Data were collected in a wild population of blue tits breeding in nest boxes in South-Western Finland (Tammisaari, 60°01′N, 23°31′E) and monitored yearly since 2003. Nest boxes were

Table 1. Sample size, average, and standard deviation (SD) of each studied trait.

Trait	Number of nestlings	Average	SD	Years
Handling aggression	9376	2.85	1.26	2006-2019
Breath rate	9114	1.92	0.41	2007-2019
Docility	8315	−0.21	0.17	2008-2019
Tarsus	9857	17.06	0.64	2003, 2005-2019
Mass	9865	11.47	1.11	2003, 2005-2019
Wing	9875	46.01	3.59	2003, 2005-2019
Head	9701	22.67	0.67	2005-2019
Tail	9858	24.98	4.28	2003, 2005-2019

visited weekly in May, and daily starting from their expected hatching date until hatching was observed (D0). Two days after hatching (D2), nestlings were weighed (using a scale with 0.1g precision) and individually marked by clipping their nails. Between 2006 and 2010, reciprocal cross-fostering was performed between pairs of nests with similar hatching date and average mass (for more details, see Brommer and Kluen 2012). Between D5 and D9, parents were caught in the box when feeding their young and identified based on unique alphanumeric codes on their metal ring or ringed if previously unringed. On D9, nestlings were weighed and ringed after their nail code, which provides information on their nest of origin, was read. On D16, nestlings were all transferred in a large paper bag and morphometric and behavioral measurements were taken following a fixed sequence (cf. Brommer and Kluen 2012). First, each individual was held still on its back by an observer, who counted how many times it struggled during 10 seconds. Docility was calculated by multiplying the number of struggles per second by –1, such that higher docility values indicate a more docile animal. Directly following the docility assay, the time each bird took to take 30 breaths was recorded twice using a stopwatch. Breath rate (BR), which captures an individual's stress response to handling (Carere and van Oers 2004), was calculated as 30 divided by the average of these two measures and expressed in number of breath/second. Morphometric measurements were then taken: First, the bird's right tarsus and head-bill length were measured using a digital sliding caliper (0.1-mm accuracy). Then, wing and tail lengths were measured using a ruler (1-mm accuracy). A score was then given to each bird based on its behavior (struggling, flapping wings) during morphometric measurements. This score, which is similarly measured in adults and called handling aggression (HA), ranges from 1 (for completely passive individuals) to 5 (for the individuals struggling continuously) and reflects the time it takes for each bird to calm down during handling. Finally, each nestling was weighed using a Pesola spring balance (0.1-g accuracy) before being placed in a second large paper bag where it remained with its already measured siblings until the entire brood was processed and put back to its nest. In total, eight traits (three behavioral and five morphological) were measured in nestlings (Table 1) and analyzed using quantitative genetic models.

MICROSATELLITE GENOTYPING

Blood was taken on all adults when caught and feathers were taken on nestlings on D9 for DNA extraction and genotyping. All laboratory work was carried out by the Center of Evolutionary Applications (University of Turku, Finland). DNA from feather samples was extracted using a silica fine and filter plate-based method modified from Elphinstone et al. (2003). DNA from blood samples was extracted with a method modified from Aljnabi and Martinez (1997). All samples were genotyped with nine microsatellite markers using a multiplex PCR approach. PCR was carried out in one 8 μl reaction using QIAGEN Multiplex PCR Kit (Qiagen Inc. Valencia, CA, USA) with the annealing temperature of 57°C and the primer concentration varying from 0.09 to 0.5 μM following the standard protocol (Table S1). To improve the microsatellite peak profiles, a GTTT-tail was added to the 5' end of each reverse primer (Brownstein et al. 1996). The sex of the offspring was determined by amplifying sex-specific genes CHD1W and CHD1Z using P2 and P8 primers in an additional amplification reaction using the same standard protocol with annealing temperature at 55°C (Griffiths et al. 1998).

Amplifications were performed on Bio-Rad S1000 thermal cyclers and the size of the fragments was determined by capillary electrophoresis on an ABI PrismTM 3130xl genetic analysis instrument. To minimize fragment analysis costs, two samples were pooled for capillary electrophoresis. To enable pooling of samples, two alternative sets of fluorescent labels, that is, all nine markers (+sexing marker) with both FAM/VIC and NED/PET labels, were used. The peak profiles of the pooled samples could then be separated during scoring and visual inspection, using GeneMarker version 2.4.0 (SoftGenetics).

POPULATION PEDIGREE

Parentage assignment was done for each year separately (2007-2019), by combining genotype data and social pedigree and using the R package MasterBayes (Hadfield et al. 2006). After

validating parent-offspring relationships between females and nestlings sampled on the same territory (mismatch tolerance = 1), we assigned genetic fathers, among all males genotyped on the same year for each offspring, with a 95% probability threshold. These analyses revealed that between 2007 and 2019, extra-pair young represented 15% (standard deviation [SD] = 3) of the young in the population and occurred in 45% (SD = 6) of broods. In nests that were not genotyped (before 2007, or when the mother or offspring was not genotyped), we assumed social parents to be the genetic parents of the offspring they reared. The resulting population pedigree hence combines social and genetic pedigrees and represents our best inference of the true pedigree in this population.

Phenotypic data are available for 9887 individuals and the pruned pedigree (which only includes informative individuals) holds record for 10,946 individuals, 9890 maternities, 8620 paternities, 38,507 full sibs, 82,487 maternal sibs, 68,748 paternal sibs, 43,980 maternal half-sibs, 30,241 paternal half-sibs, a mean family size of 12.4, a mean pairwise relatedness of 1.5×10^{-03}, and a maximum pedigree depth of 9. Several features of this dataset are expected to provide information that allows obtaining unbiased estimates of dominance, additive genetic, maternal, and common environment variance. First, full sibs and maternal half sibs share common environment effects and maternal effects. Because dominance is expected to contribute to phenotypic covariance between full sibs but not between half-sibs, comparing full to half-sibs allows estimating dominance variance. In this dataset, 1304 phenotyped nestlings in 473 nests are identified as extra-pair offspring. Second, paternal half sibs, regardless of whether they are sired through extra-pair or within-pair mating, do not share common environment nor maternal effects and provide unconfounded information on additive genetic variance. In this population, 220 of the identified 563 sires mated with more than one female (1.67 females per male on average). Third, comparing full sibs from different breeding attempts allows estimating consistent differences in offspring traits across mothers (maternal effects). Of the 794 known mothers, 271 reproduced in multiple years (1.54 broods per female on average), 151 are known to have reproduced more than once with the same male. Finally, cross-fostering experiments allow estimating environmental effects due to a common nest of rearing (common environment). In this dataset, 1437 nestlings from 350 broods were raised in a different nest than their nest of origin. There were no double first cousins in this population. In natural populations, the estimation of dominance can be complicated by the presence of inbreeding (see Wolak and Keller 2014). Based on this population's pedigree, we found inbreeding to be rare: 66 individuals (0.6% of the population) have a nonzero inbreeding coefficient (f), among which 37 closely inbred ($f = 0.25$) individuals (0.3% of the population) produced by seven pairs (0.4% of the pairs). These numbers and the average inbreeding coefficient in the population (9.8×10^{-04}) are lower than what was found in a noninbred great tit population showing low inbreeding variance (Szulkin and Sheldon 2008; Chapman and Sheldon 2011). We hence assume inbreeding to have negligible impacts on our variance component estimates.

ANIMAL MODELS

Quantitative genetic analyses were performed by running univariate animal models for each trait separately. Animal models are a type of linear mixed model in which the additive genetic and dominance relatedness matrices derived from a population pedigree can be fitted as random effects to estimate additive genetic and dominance variance (Henderson 1984; Kruuk 2004; Wilson et al. 2010). In all models, we also fitted year, brood of rearing, mother identity, and observer identity as additional random effects to estimate variance among years, broods (common environment variance), mothers (maternal variance), and observers. Sex was fitted as a fixed effect in all models to account for sexual dimorphism, which can already occur at the nestling stage. Cross-sex additive genetic correlations for all traits were calculated and they were all $\geq$0.96 (standard error [SE] $\leq$ 0.29). Finally, handling order was fitted as a fixed effect for behaviors to correct for potential effects of waiting time before measurement, and mass was corrected for tarsus length to reflect body condition. Animal models were solved using Restricted Maximum Likelihood (REML) and implemented in ASReml-R version 4.0 (Butler et al. 2009; VSN International, Hemel Hempstead, U.K.). Statistical significance of fixed effects was tested using conditional Wald F tests. Statistical significance of dominance variance was tested using likelihood ratio tests (LRT) tested against a chi-square distribution with mixture of zero and one degree of freedom to account for the fact that variances cannot be below zero (Self and Liang 1987). For each variance component, we calculated its approximated 95% confidence interval (CI) by adding/subtracting $1.96 \times$ SE to its point estimate that we use for visible display purposes only and not for statistical testing. Heritability (h^2) of each trait was calculated as the ratio VA/VP, where we calculated VP, the phenotypic variance, as the sum of the REML estimates of additive genetic effects, dominance, maternal, common environment effects, and residuals, conditional on the fixed-effect structure of the model. Standard errors of variance ratios were approximated using the delta method (Fischer et al. 2004). All statistical analyses were performed in R (R Development Core Team 2019). Residuals of all animal models were approximately normally distributed (Shapiro-Wilk test values >0.91; Figure S1). The script of the quantitative genetic analysis is provided in Text S1.

SIMULATIONS

Simulations were performed using the population pedigree and the R package nadiv (Wolak 2012) to generate values for different random effects of a hypothetical "average trait" (with phenotypic variance equal to 1 and additive genetic, common environment, maternal, dominance, and residual variances equal to their average proportion of phenotypic variance calculated across all traits). In total, 1000 datasets were simulated and for each dataset, five different models were run. In model 1 ("full model"), all five variance components were estimated. Model 2 was similar to model 1 but dominance variance was constrained to be equal to the variance of the simulated dominance values. Model 2 was then compared to model 1 using LRT and we calculated the percentage of simulations in which the estimated dominance variance differed statistically from the true dominance variance. In model 3 ("Model –D"), dominance variance was not fitted as a random effect. Model 3 was then compared to model 1 using LRT and we calculated the percentage of simulations in which a significant dominance variance was found (power). Maternal and dominance variances are likely to be confounded in natural datasets and we wanted to investigate our capacity to disentangle them and test whether not fitting one can create bias in the other. In model 4 ("Model –M"), maternal variance was hence not fitted as a random effect. In model 5 ("Model –D–M"), neither maternal nor dominance variance were fitted as random effects. We calculated the distribution (average and 95% confidence interval [CI]) of the error (estimated-simulated value) of all variance estimates obtained in models 1, 3, 4, and 5. The script of the simulations is provided in Text S2. To assess the identifiability of all random effects, we (i) estimated the correlations between each pair of variance components estimated by the "full model" across all simulated datasets (as done in Bourret and Garant 2017) and (ii) extracted each empirical model's average information matrix and performed a multivariate sampling approach (with $n = 1000$) advocated in Houle and Meyer (2015) to estimate sampling correlations between all variance components and their 95% CI. A strong negative correlation is expected when two components are confounded (low identifiability).

Results

ANIMAL MODEL ESTIMATES

We found significant differences between sexes for all traits except tail length. Males were larger and heavier than females on D16, behaved more aggressively, had a lower BR, and were less docile than females. We also found that handling order increased HA and decreased BR but did not significantly affect docility (Tables S2-S9).

Regarding variance components, all traits exhibited additive genetic and common environment variance as their nonoverlapping zero 95% CI illustrates (Fig. 1). For all behaviors, both components had comparable magnitude and for tarsus length, additive genetic variance was greater than common environment variance. In contrast, for all other morphological traits, common environment variance was greater than additive genetic variance. Maternal variance was a relatively small variance component for all right traits and its 95% CI overlapped zero for all traits except BR and tarsus length. Morphological traits exhibited important among-year variance compared to behaviors for which this variance component was relatively low (and overlaps zero for HA and docility). Among-observer variance varied substantially among traits. It was relatively important for HA, BR, head, and tail length, but its 95% CI always overlapped zero. Finally, dominance variance was one of the smallest variance components for all traits with maternal variance. Dominance variance was indeed bound to zero and hence not estimable for HA and tail length, whereas for the other 6 traits, its estimated 95% CIs overlapped zero. LRT tests confirmed that dominance variance was not statistically different from zero for BR ($\chi^2 = 0.003$, $P = 0.48$), docility ($\chi^2 = 1.83$, $P = 0.09$), tarsus ($\chi^2 = 0.06$, $P = 0.40$), size-corrected mass ($\chi^2 = 0.11$, $P = 0.37$), wing length ($\chi^2 = 1.93$, $P = 0.08$), and head-bill length ($\chi^2 = 0.64$, $P = 0.21$). All estimates of variance and their standard errors are provided in Tables S2-S9.

We computed proportional contributions of the above variance components excluding year and observer variances. Proportionally, residual variance was the largest variance component for all behaviors (Fig. 2). In contrast, common environment variance was the major source of phenotypic variance for most morphometric traits except tarsus length, for which it was additive genetic variance. Heritability varied between traits, being low for tail length ($h^2 = 0.074$, SE $= 0.024$), moderate for behaviors ($h^2 = 0.238$, SE $= 0.035$ for HA; $h^2 = 0.158$, SE $= 0.037$ for BR; $h^2 = 0.156$, SE $= 0.036$ for docility), for mass ($h^2 = 0.252$, SE $= 0.036$), wing ($h^2 = 0.136$, SE $= 0.034$), and head-bill length ($h^2 = 0.227$, SE $= 0.040$), and the highest for tarsus length ($h^2 = 0.387$, SE $= 0.045$). Assuming unbiased point estimates, dominance variance represented a modest part of genetic variance for BR (0.021, SE $= 0.355$), tarsus (0.034, SE $= 0.132$), and mass (0.056, SE $= 0.153$), but less so for head length (0.160, SE $= 0.181$), docility (0.360, SE $= 0.196$), and wing length (0.351, SE $= 0.193$).

SIMULATIONS: POWER, BIAS, AND IMPACT OF THE MODEL'S RANDOM STRUCTURE

Simulations showed that for a hypothetical average trait measured in this population, the estimated dominance variance statistically differed from the simulated dominance variance in 5.5% of the simulations. Although our power to statistically

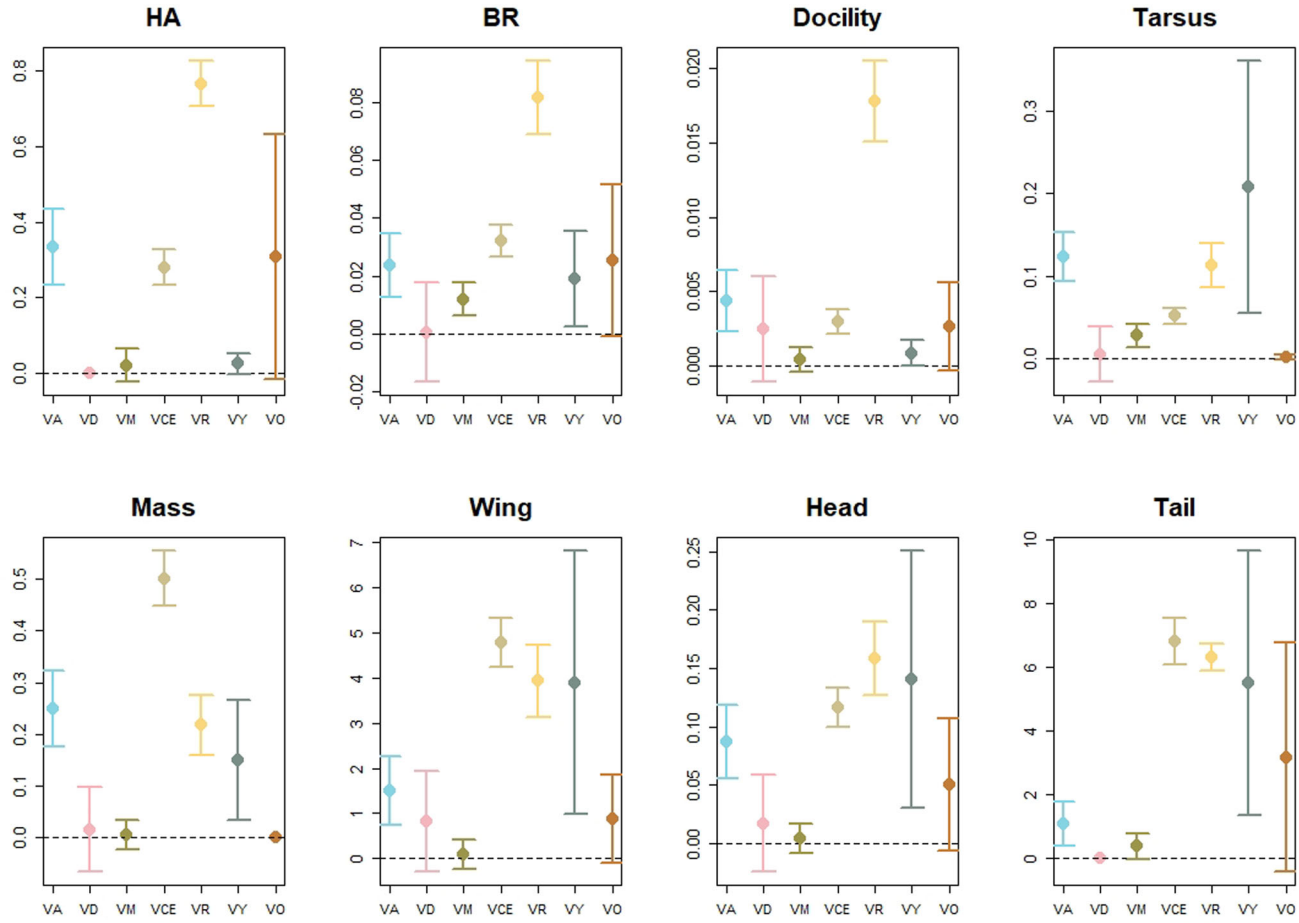

Figure 1. Point estimate and approximated 95% CI of each variance component in each trait. Each variance component is assigned a unique color (consistent across all figures) to ease visual comparison between traits. Title acronyms: HA = handling aggression; BR = breath rate. Axes acronyms: VA = additive genetic variance; VD = dominance variance; VM = maternal variance; VCE = common environment variance; VR = residual variance; VY = year variance; VO = observer variance.

detect dominance variance was low (15.2%), these simulations also showed that estimates of all variance components were unbiased when all fitted in the model ("Full model," Fig. 3 upper panel).

These simulations also suggested that not fitting dominance variance for an average trait (Model "–D") did not bias estimates of maternal and common environmental variances. However, it caused an inflated residual variance and a slightly overestimated additive genetic variance. In contrast, not fitting maternal variance (Model "–M") generated clearly inflated estimates of additive genetic and dominance variance, slightly overestimated common environment variance, and an underestimated residual variance. Finally, in the model not fitting dominance and maternal variance (Model "–D–M"), estimates of additive genetic and common environment variance were even more inflated, whereas residual variance was unbiased (Fig. 3 upper panel). Hence, ignoring dominance and maternal variance can bias estimates of additive genetic and other components of the phenotypic variance, and therefore estimates of heritability. Indeed, applying these four models to simulated data showed that heritability estimates were inflated when fitting reduced models (Fig. 3 lower panel): heritability went from 0.198 ("Full model") to 0.209 (+5.71%) when ignoring dominance variance, and to 0.229 (+15.67%) when ignoring maternal variance, whereas not fitting both components resulted in a heritability of 0.246 (+24.46%). Applying these four models to empirical data also revealed differences in heritability estimates between full and reduced models, which varied in amplitude across traits due to different amounts of dominance and maternal variance (Table 3). Importantly, heritability estimates were systematically higher when both dominance and maternal effects were not fitted. In addition, differences in heritability between the full and the most reduced model were often higher than the sum of the differences between the full model and each of the two other reduced models, in particular for BR and docility (Table 3) but also for simulated data (Table 2). For both simulated and empirical data, higher heritability estimates obtained in

Table 2. Variance components (and their 95% CI) estimated by the four animal models for a simulated trait.

Variance component		Full model	Model-D	Model-M	Model-D-M
Additive genetic	VA	0.20 [0.13, 0.27]	0.21 [0.14, 0.28]	0.23 [0.16, 0.29]	0.25 [0.19, 0.31]
Dominance	VD	0.03 [0.00, 0.15]		0.05 [0.00, 0.17]	
Maternal	VM	0.03 [0.00, 0.06]	0.03 [0.00, 0.06]		
Common environment	VE	0.30 [0.26, 0.34]	0.30 [0.26, 0.34]	0.31 [0.27, 0.35]	0.31 [0.28, 0.36]
Residual	VR	0.43 [0.36, 0.49]	0.46 [0.42, 0.50]	0.41 [0.33, 0.46]	0.44 [0.40, 0.47]
Phenotypic	VP	1.00 [0.96, 1.05]	1.00 [0.96, 1.05]	1.00 [0.96, 1.05]	1.00 [0.96, 1.05]

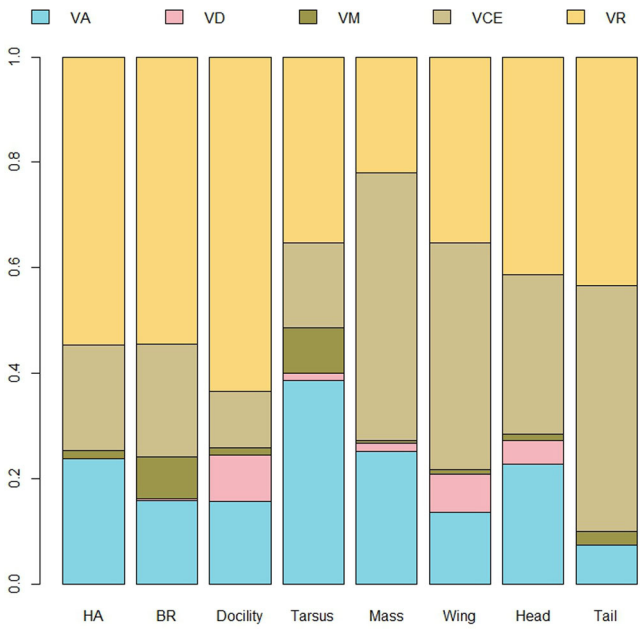

Figure 2. Proportions of the phenotypic variance (total column height) explained by additive genetic (VA), dominance (VD), maternal (VM), common environment (VCE), and residual (VR) variances calculated based on REML point estimates conditional upon the fixed effects in the model. To facilitate trait comparisons, we here ignore variances due to year and observer, although these were included in the model.

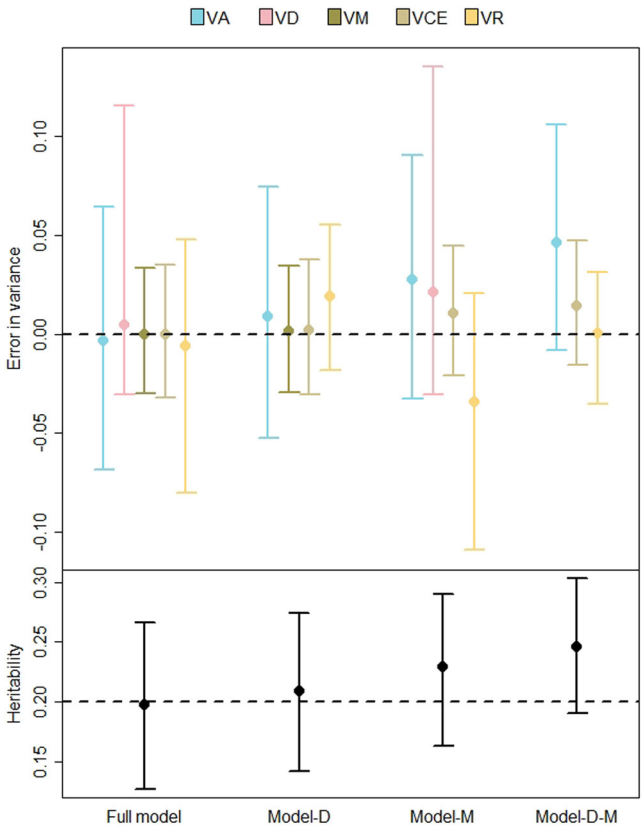

Figure 3. Median estimate and 95% CI of variance errors (estimated – simulated values) and heritability for each model. Each color denotes a different variance component (VA = additive genetic variance; VD = dominance variance; VM = maternal variance; VCE = common environment variance; VR = residual variance).

reduced models were explained by a higher additive genetic variance because phenotypic variance remained constant (Tables 2 and S10-S17).

The correlation matrix between variance components estimated by the full model for simulated data revealed that maternal variance was moderately confounded with additive genetic and common environmental variance ($r = -0.42$ and -0.32, respectively, Table S18) and positively correlated to residual variance ($r = 0.34$). In contrast, dominance variance had weak, moderate, and strong negative correlations with maternal ($r = -0.09$), additive genetic ($r = -0.35$), and residual variance ($r = -0.80$), respectively. Common environmental variance was not and weakly confounded with additive and dominance genetic variances ($r = -0.01$ and -0.11, respectively). Sampling correlation matrices between variance components estimated for each trait in each empirical model were overall qualitatively similar, although correlations between additive and dominance genetic variances were weaker and both genetic components were positively correlated with common environmental variance (Tables S19-S26).

Table 3. Heritability (and SE) estimated by the four empirical animal models for each studied trait. Traits are ranked by decreasing order of their respective proportion of maternal to phenotypic variance (VM/VP) and proportion of dominance to phenotypic variance (VD/VP). Relative difference (in %) between the heritability estimate from each reduced model and the full model estimate is printed in italics below each heritability estimate.

	Full model			Model-D	Model-M	Model-D-M
Trait	VM/VP	VD/VP	h^2	h^2	h^2	h^2
Tarsus	0.085	0.014	0.39 (0.04)	0.39 (0.04)	0.48 (0.04)	0.49 (0.03)
				1.32	*25.09*	*27.79*
BR	0.080	0.003	0.16 (0.04)	0.16 (0.03)	0.23 (0.03)	0.26 (0.03)
				0.64	*44.8*	*62.83*
Tail	0.026	0	0.07 (0.02)	0.07 (0.02)	0.10 (0.02)	0.10 (0.02)
				0	*35.76*	*37.1*
Docility	0.015	0.088	0.16 (0.04)	0.18 (0.03)	0.17 (0.03)	0.20 (0.03)
				12.46	*11.51*	*27.81*
HA	0.015	0	0.24 (0.04)	0.24 (0.04)	0.26 (0.03)	0.26 (0.03)
				0	*7.8*	*7.8*
Head	0.012	0.045	0.23 (0.04)	0.24 (0.04)	0.24 (0.04)	0.26 (0.03)
				7.24	*5.36*	*13.22*
Wing	0.007	0.073	0.14 (0.03)	0.16 (0.03)	0.14 (0.03)	0.17 (0.03)
				20.77	*5.45*	*27.45*
Mass	0.006	0.015	0.25 (0.04)	0.26 (0.03)	0.26 (0.03)	0.27 (0.03)
				3.25	*3.08*	*6.16*

Discussion

We infer genetic dominance variance for different nestling traits in a wild population and use simulations to study repercussions of ignoring genetic dominance across various modelling schemes. Our findings provide three clear conclusions.

First, dominance variance can be estimated in the wild, although when estimable (6/8 traits), dominance variance does not statistically differ from zero. These results combined with our simulations demonstrate that the power to detect a realistic level of dominance variance in this population is low despite the availability of long-term phenotypic data of over 9000 animals and a pedigree resolved for paternities. This issue clearly illustrates the fact that these analyses are data hungry and thus that inferring dominance in the wild is challenging (cf. Kruuk and Hill 2008; Wolak 2012; Wolak and Keller 2014) and explains the paucity of empirical estimates from wild populations to date. However, our simulations underline that this wild population's data structure including half-sibs, full sibs, reciprocally cross-fostered nestlings, and multiple breeding attempts for females permits gathering sufficient information to produce estimates of dominance variance and other variance components that are unbiased and weakly to moderately confounded. Estimates of dominance variance, although not statistically different from zero, add to the few effect sizes of dominance in relation to other variance components in a nondomestic species and constitute rare estimates of dominance variance in a wild population.

Second, dominance variance—when estimable—explains a small portion of the genetic variance, with point estimates ranging from 2% to 36%. In contrast, based on 553 literature point estimates, Wolak and Keller (2014) computed that on average 38% of the genetic variance was due to dominance. Hence, our study implies that dominance variance may be relatively modest in wild populations compared to measures obtained under controlled environmental conditions, although further attempts to estimate dominance variance in other systems is clearly required. Not surprisingly, point estimates of dominance represented only a small fraction (less than 9%) of phenotypic variance in our wild study population subjected to ecological relevant environment variance, whereas it was on average 14% (475 point estimates) under controlled environmental conditions (Wolak and Keller 2014). Interestingly, estimates of dominance variance for behavioral traits align with earlier findings (Brommer and Kluen 2012) in which nest-of-origin variance (which represented the upper limit to dominance variance) was zero for HA and small for BR and docility (7.3% and 4% of phenotypic variance, respectively). The low proportion of phenotypic variance explained by dominance in this population is also consistent with findings based on nest-of-origin variance from another wild bird population (Merilä et al. 2001), in which dominance variance in body

condition was considered to explain at most 9% of the phenotypic variance.

Third, based on simulations we find that ignoring dominance variance in our population indeed inflates the estimate of additive genetic variance and heritability, which aligns with theoretical predictions (cf. Ovaskainen et al. 2008) and empirical findings (Waldmann et al. 2008). However, this inflation can remain relatively small as long as maternal variance is included. In terms of our variance partitioning, maternal variance is the sum of both maternal genetic and maternal environmental variances, and captures differences in offspring trait values that are maintained across repeated records of the same mother. Not including maternal variance can clearly inflate the estimates of additive and dominance genetic variances and, to a lesser extent, common environmental variance. This finding is not surprising, as it reflects the fact that having the same mother is a major source of resemblance in full- and half-sibs produced and reared in one or multiple broods by the same female. Interestingly, although we find that ignoring maternal variance can inflate estimates of dominance variance when explicitly accounted for in the model, we do not find the converse to hold; that is, maternal variance does not increase when the dominance variance component is not accounted for in the model. Instead, dominance variance ends up in both additive genetic and residual components. This "leaking" of variance between components in reduced models is consistent with the confounding between them in the full models. It also aligns with earlier predictions that dominance and maternal effects (both included in the nest-of-origin component in Hadfield and Owens 2006) can inflate estimates of additive genetic variance (Lynch and Walsh 1998; Hadfield and Owens 2006) and that ignoring dominance variance often inflates individual-level variance here estimated as residual variance (Kruuk 2004; Adams et al. 2012). Importantly, biases in the different variance components resulting from each reduced model systematically cause inflated heritability estimates.

The greater bias in heritability when ignoring maternal effects than when ignoring dominance, even when these two components have an equal variance, is likely explained by maternal variance being more strongly confounded with additive genetic variance, at least in our study population. Strikingly, the animal model producing the most inflated heritability estimates (i.e., the one ignoring both dominance and maternal effects) is commonly fitted in evolutionary quantitative genetic studies. Indeed, maternal variance is commonly not included in animal models estimating the heritability of juvenile traits in pedigreed populations (e.g., Frentiu et al. 2007, Dingemanse et al. 2009, Weiß and Foerster 2013, Pavitt et al. 2014, Class and Brommer 2015, Stedman et al. 2017). Ignoring maternal variance is often justified by a limited number of breeding attempts per female or by testing beforehand that the inclusion of maternal identity returns nonsignificant or nonestimable maternal variance. In this population, for example, 66% of females breed only once and maternal variance represents a rather small portion of phenotypic variance. However, we show that not fitting maternal effects, even when small and nonsignificant, can substantially bias estimates of additive genetic variance and therefore should be avoided.

The above conclusions are moderated by the caveat that the dominance variance estimates we obtain and use in the simulations are point estimates that do not significantly differ from zero. As stated above, we view these point estimates are relevant, as they present the maximum likelihood and unbiasedly estimated effect sizes of dominance for a variety of traits in a wild population. Knowledge of effect sizes from a wild population is required to realistically explore power of detecting dominance as well as possible erroneous inferences of additive genetic and other variance components when ignoring dominance. This is because a simulation is always conditional upon the effect sizes one assumes in addition to the data structure (pedigree and phenotyped individuals). Using estimates of dominance variance obtained from lab and domesticated animals would risk being nonrepresentative of wild populations. Our findings imply that assuming 38% of genetic variance is due to dominance is a maximal value in wild populations, rather than an average, as studies in controlled environments suggest (Wolak and Keller 2014). Still, the conclusions of our simulations are an a posteriori exploration specific to our study population that may or may not be relevant in other systems. To this end, we recommend that analysts explore the outcome of simulations tailored to their specific study system to quantify in which variance component dominance variance ends up when ignored. Our findings combined with Wolak (2012), Wolak and Keller (2014), and our script (Text S2) provide a good starting point for straightforward construction of such simulations.

To conclude, this study demonstrates that unbiased point estimates for dominance variance can be inferred in a wild population in situ and that dominance variance in the wild is likely lower than estimates from controlled environments suggest. Further, ignoring such low level of dominance does not strongly inflate estimates of additive genetic variance and heritability as long as variance across mothers is accounted for. We stress that this conclusion is specific to our study and may or may not be generalizable to other systems or to other type of traits (e.g., life history traits). We therefore encourage conducting simulations to explore the sensitivity of inferences for ignoring dominance and to report point estimates of dominance whenever estimable, as we need to compile estimates in a variety of natural systems to deepen our understanding of its importance in the wild.

AUTHOR CONTRIBUTIONS

Both authors conceived the study. BC performed the analyses contributed to data collection, and authored the manuscript. JEB set up the study population, collected data, and authored the manuscript. Both authors approved the final version of the manuscript.

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DATA ARCHIVING

Analyses reported in this article can be reproduced using data and code provided on Dryad (https://doi.org/10.5061/dryad.zpc866t6d).

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