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The genetic basis of dispersal in a vertebrate metapopulation

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Abstract

Dispersal affects evolutionary processes by changing population size and genetic composition, influencing the viability and persistence of populations. Investigating which mechanisms underlie variation in dispersal phenotypes and whether populations harbour adaptive potential for dispersal is crucial to understanding the eco-evolutionary dynamics of this important trait. Here, we investigate the genetic architecture of dispersal among successfully recruited individuals in an insular metapopulation of house sparrows. We use an extensive long-term individual-based ecological data set and high-density single-nucleotide polymorphism (SNP) genotypes for over 2500 individuals. We conducted a genome-wide association study (GWAS), and found a relationship between dispersal probability and a SNP located near genes known to regulate circadian rhythm, glycogenesis and exercise performance, among other functions. However, this SNP only explained 3.8% of variance, suggesting that dispersal is a polygenic trait. We then used an animal model to estimate heritable genetic variation (σ_A^2), which composes 10% of the total variation in dispersal probability. Finally, we investigated differences in σ_A^2 across populations occupying ecologically relevant habitat types (farm vs. non-farm) using a genetic groups animal model. We found different adaptive potentials across habitats, with higher mean breeding value, σ_A^2, and heritability for the habitat presenting lower dispersal rates, suggesting also different roles of environmental variation. Our results suggest a complex genetic architecture of dispersal and demonstrate that adaptive potential may be environment dependent in key eco-evolutionary traits. The eco-evolutionary implications of such environment dependence and consequent spatial variation are likely to become ever more important with the increased fragmentation and loss of suitable habitats for many natural populations.

KEYWORDS
adaptive potential, additive genetic variance, dispersal, genotype-by-environment interaction, GWAS, heritability

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1 | INTRODUCTION

Dispersal is a complex life-history trait that influences demographic and genetic processes, hence dispersal plays an important role in the eco-evolutionary dynamics of geographically structured populations (Legrand et al., 2017; Van Dyck & Baguette, 2005). Dispersal affects evolutionary processes by leading to gene flow that increases genetic variation within and affects the genetic structure of populations (Holsinger & Weir, 2009). Variation in dispersal also impacts local population sizes and densities, habitat use and (re)colonization in fragmented populations, and, ultimately, these effects can influence the viability and persistence of populations and species (Clobert et al., 2012; Saastamoinen, 2008). Because of its fundamental importance, it is essential to understand whether and how fast dispersal rates can evolve (Ronce, 2007; Saastamoinen et al., 2018). Estimating the heritable genetic component and examining the genetic architecture of dispersal are needed to understand causes of variation in the dispersal phenotype and to predict its adaptive evolutionary potential (Orr, 2005; Zera & Brisson, 2012).

Dispersal-related traits (such as wing shape, locomotion performance or speed) have previously been shown to be heritable in birds and insects with an average heritability (h^2) of 0.35 (Saastamoinen et al., 2018). Another meta-analysis revealed that the average heritability of movement behaviour over 15 different studies (including both dispersal and migration) was 0.46 (Dochtermann et al., 2019). However, estimating the heritability of dispersal or dispersal syndromes (i.e. traits associated with dispersal) is a challenging task due to the complexity of the dispersal event itself. Dispersal propensity may be affected not only in one or more of the dispersal stages (departure, transfer and settlement) but also by dispersal-related phenotypic traits and their interactions with the environment (Bowler & Benton, 2005; Ronce, 2007; Saastamoinen et al., 2018). Due to the need for accurate identification of dispersers and resident individuals, which relies on the quality and extent of mark–recapture data over sufficiently large geographic areas to cover normal dispersal distances, estimating heritability of dispersal and dispersal-related traits is challenging, but such estimates have been obtained in birds and insects more often than any other taxa (Brown et al., 2014; McGaugh et al., 2010; Saastamoinen et al., 2018; Waser & Jones, 1989; Zera & Brisson, 2012).

Additive genetic variance (σ_A^2) and the proportion of the phenotypic variance explained by σ_A^2 (i.e. narrow sense heritability; h^2) reflect the heritable genetic component of a trait and determine the potential rate of any evolutionary response to selection acting on the trait (Lande, 1979). A specific linear mixed-effects model called the 'animal model' uses information on the relatedness of individuals with phenotypic data and is widely used to estimate additive genetic variances in phenotypic traits of domestic animals as well as wild populations of many species (Kruuk, 2004; Lynch & Walsh, 1998; Wilson et al., 2010). However, most animal models assume that the populations under study are genetically homogeneous, which is often not the case in natural populations, and this assumption may therefore introduce biases in estimates (Muff et al., 2019; Wolak & Reid, 2017). A recent extension called genetic groups animal model (GGAM) enables us to account for genetic admixture within and between populations and allows estimating heterogeneous and population-specific mean genetic values (basic GGAM; Wolak & Reid, 2017) and additive genetic variances (extended GGAM; Aase et al., 2022; Muff et al., 2019).

Genome-wide association studies (GWAS) are commonly performed to investigate underlying genetics of phenotypic traits and to detect Quantitative Trait Loci (QTL; Korte & Farlow, 2013). In relation to dispersal, it has, for instance, been shown that a foraging gene in *Drosophila melanogaster* is linked with locomotion behaviour, causing adults with the dominant 'rover' allele to have longer dispersal distances (Edelsparre et al., 2014). Similarly, the *Pgi* gene in the Glanville fritillary butterfly (*Melitaea cinxia*) codes for a metabolic enzyme associated with cellular energetics (Mattila & Hanski, 2014), and has an allelic variant that causes a higher flight metabolic rate and dispersal propensity (Haag et al., 2005; Niitepõld et al., 2009; Niitepõld et al., 2011). However, research on genetic variation in dispersal in natural populations, as well as other complex life-history traits, indicates that underlying genetic variation is often caused by many genes of small effect (i.e. are polygenic; Saastamoinen et al., 2018; Tiffin & Ross-Ibarra, 2014; Zera & Brisson, 2012). Polygenic traits may covary with several different fitness traits and are often influenced by multiple environmental factors and can hence show complex evolutionary trajectories (Remington, 2015).

Studies on the genetic architecture of dispersal pave the road to a better understanding of the ecological and evolutionary consequences of dispersal and movement in fragmented populations and species invasions, and hence the capacity to spread and ultimately survive in the face of environmental change (Saastamoinen et al., 2018). In the present study, we investigated the heritable genetic basis of dispersal probability among successfully recruited individuals in an insular metapopulation of a small passerine bird, the house sparrow (*Passer domesticus*). Previous studies in this metapopulation have shown differences in dispersal rates related to island habitat type (Ranke et al., 2021; Saatoglu et al., 2021). Initially, we, therefore, assumed that the heritable genetic variation in dispersal was similar across islands but allowed the mean genetic values of dispersal to differ between island habitat types. That is, we used a basic genetic groups animal model (basic GGAM) to estimate the additive genetic variance (σ_A^2) and narrow-sense heritability (h^2) of dispersal probability. Subsequently, in order to test our initial assumption, we used an extended GGAM to allow for different σ_A^2 of dispersal for the two habitat types. Lastly, we used GWAS to identify genes that might explain variation between individuals in dispersal probability. To achieve these goals, we used high-quality information on dispersal and high-density genome-wide single-nucleotide polymorphism (SNP) genotype data from over 2500 recruited individuals in a long-term study of house sparrows on eight study islands in a metapopulation off the coast of northern Norway, where relatedness is available through a genetically determined multi-generational pedigree (Lundregan et al., 2018; Niskanen et al., 2020; Saatoglu et al., 2021).

2 | METHODS

2.1 | Study populations and field methods

The study comprises data from eight subpopulations of an insular house sparrow metapopulation located in the Helgeland archipelago in northern Norway (66°30′ N, 12°30′ E; Figure 1). These eight islands are part of a larger metapopulation study system covering ca. 1600 km^2, where extensive fieldwork during the breeding seasons (May–August) and autumns (September–October) since 1993 has ensured that most house sparrows have been individually marked and followed through their lives from hatching to death (Baalsrud et al., 2014; Ranke et al., 2021; Saatoglu et al., 2021). From each ringed individual, we collected a small blood sample (~25 μL) to obtain DNA for genotyping purposes, genetic parentage determination and pedigree construction (Niskanen et al., 2020). The subpopulations on islands in this metapopulation are almost solely interconnected by natal dispersal (fewer than 0.02% of adults performed breeding dispersal during their lifetimes; Altwegg et al., 2000) with an overall dispersal rate of 22.2% among individuals that successfully recruited (Saatoglu et al., 2021). However, dispersal rates also differ among islands and years and depend on environmental conditions related to the habitat type of the subpopulations (Ranke et al., 2021; Saatoglu et al., 2021). On five islands (Aldra, Gjerøy, Hestmannøy, Indre Kvarøy and Nesøy), which are also denoted as 'farm' islands, house sparrows nest in a colony-like manner on and near dairy farms, and thus have higher local densities but are also more sheltered during harsh weather conditions (Araya-Ajoy et al., 2019). Conversely, in the subpopulations located farther from the mainland (Myken, Træna and Selvær), denoted 'non-farm' islands, the house sparrows are less colonial and breed in nest boxes and other suitable cavities on the houses in small villages. Non-farm islands have less sheltering opportunities and probably less stable food availability during winter time (Araya-Ajoy et al., 2019). We have previously shown that the dispersal rate is higher among the non-farm islands (24.3%) than among the farm islands (9.6%) and that the dispersal rate is higher from non-farm to farm islands (7.2%) than from farm to non-farm islands (2.1%; see Saatoglu et al., 2021).

2.2 | Genotyping, natal population assignment and dispersal phenotypes

A total of 3253 adult individuals captured and sampled for blood during the period 1998–2013 were successfully genotyped with our custom house sparrow Affymetrix Axiom 200,000 SNP array (Lundregan et al., 2018). Based on the MonoHigh and PolyHigh quality criteria of Affymetrix, 185,587 SNPs were passed on to further quality control, where potential duplicates (identity by state above 0.98) and low-quality samples (genotyping rate < 0.90) were removed from the data set. Moreover, loci with potentially high levels of genotyping errors (SNP call rate < 95%; Mendelian error rate based on parental relationships > 5%) or low minor allele frequency (MAF < 0.01)

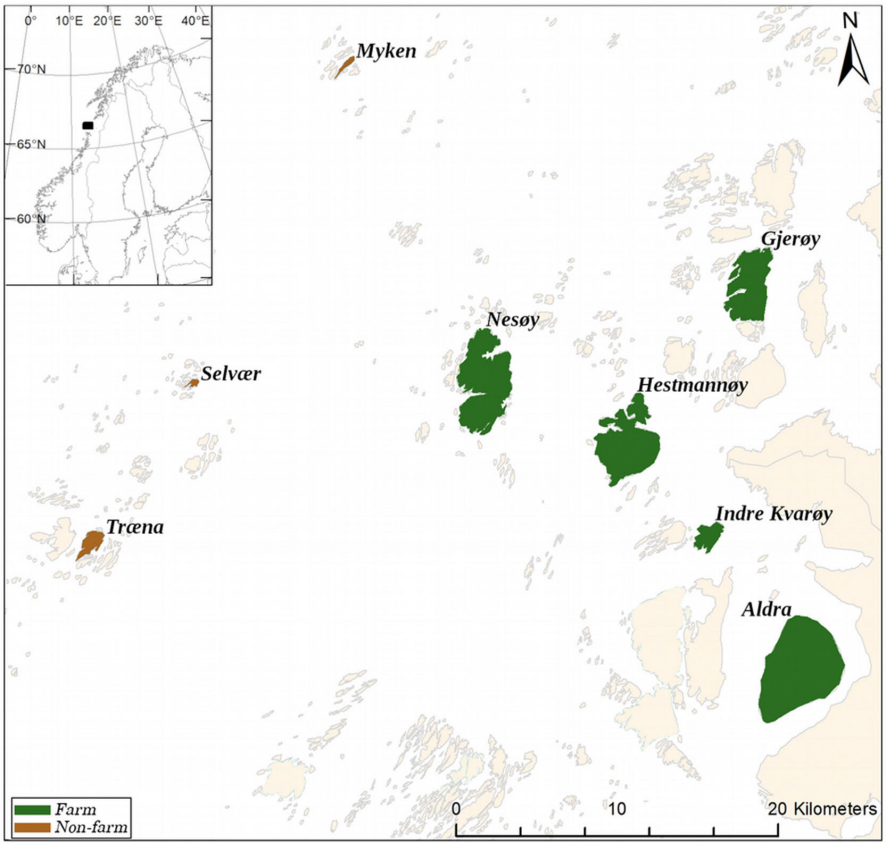

FIGURE 1 Map showing the house sparrow metapopulation study system in northern Norway, with different colours for farm (green) and non-farm (brown) habitat islands included in the present study.

were also excluded. In total, 3116 individuals and 183,145 SNPs passed the overall quality check (Lundregan et al., 2018). In this data set, any missing genotypes (0.76% of the total 570,679,820 genotypes) were imputed using LinkImpute (Money et al., 2015) to improve statistical power in our GWAS. Finally, a metapopulation-level pedigree was constructed based on parentage analyses using individual high-density SNP genotype data in the R-package sequoia (Huisman, 2017; Niskanen et al., 2020). Counting only real individuals, both parents were known for 52.7% of the 3116 individuals in the pedigree, one parent was known for 25.0% of the individuals and the rest of the individuals did not have any parental information in the pedigree. However, sequoia also assigned 440 dummy parents to, for example, group full- and half-siblings and linked grandparents and grandoffspring if the real parents were not genotyped. Thus, this extended pedigree included in total of 3556 (real and dummy) individuals, of which both parents were known for 74.8% of individuals and only one parent was known for 7.0% of individuals.

High-quality information on natal dispersal was available for 2741 adult birds present on one of the eight main study islands during the years 1998–2013, where 60% of individuals could be assigned to a natal island due to known nest identity from mark–recapture data. Meanwhile, 40% of individuals were assigned a natal island using a low-error (< 5%) high-density SNP genotype-based genetic assignment procedure (BONE; Kuismin et al., 2020) calibrated with a genetically determined pedigree built at the metapopulation level, as described in Saatoglu et al. (2021). For the remaining 375 individuals that were successfully genotyped, we only had information on which island they were first recorded (either as a fledged juvenile in the autumn or a 1-year-old recruit during summer). Therefore, we removed these individuals from the phenotype data set. We also removed individuals hatched outside one of our eight main study islands ($N = 98$), and individuals for which we could only genetically assign a group of possible natal islands due to logistical constraints ($N = 41$; see Saatoglu et al., 2021). Thus, phenotypic data on dispersal for a total of 2602 individuals which successfully recruited to the adult population on one of the eight main study islands, with an estimated overall dispersal rate of 18.6%, were used in the animal model analyses and GWAS.

2.3 | Heritable genetic variation in dispersal

We first estimated the variance in dispersal probability (i.e. natal dispersal of recruits between any of the study islands) that was attributable to additive genetic effects and several environmental effects using a basic genetic group animal model (basic GGAM), where individuals born in the farm and non-farm island habitat types were allowed to differ in mean breeding values for dispersal, but where the additive genetic variances in dispersal were assumed identical in both habitat types (Muff et al., 2019; Wolak & Reid, 2017). Next, we formulated an extended genetic groups animal model (extended GGAM; Aase et al., 2022; Muff et al., 2019), where the additive genetic variance in dispersal was also allowed to differ for farm and non-farm island habitat types. Here, the island habitat types corresponded to two baseline populations or genetic groups, where values for the proportions of farm (group 1) and non-farm (group 2) genetic group contributions (q_{ij}, with i indicating the individual and j indicating the genetic group) to the genomes of all individuals were calculated from the metapopulation-level pedigree. This pedigree included all 3116 successfully SNP-typed real individuals and 440 dummy individuals that were assigned as parents to identify relationships among recruits (such as grandparent–grandoffspring, and full- or half-sibling relationships) when one or both of the true genetic parents were not genotyped (Niskanen et al., 2020). Additionally, in order to define the farm and non-farm genetic group contributions of all individuals further down in the pedigree, phantom parents were assigned to individuals or dummy individuals without a parent or dummy parent (Wolak & Reid, 2017). The natal island of these phantom individuals was assumed to be the same natal island as their (real or dummy) offspring. Therefore, to obtain assumed natal island habitat type and hence define the 100% genetic group belonging of phantom parents, we first identified the known or most likely natal island habitat type of each individual in the pedigree. For 2741 of the 3116 real individuals, their natal island was known either from ecological or genetic assignment data (Saatoglu et al., 2021). Because most house sparrows in our study metapopulation are resident individuals (Ranke et al., 2021; Saatoglu et al., 2021), we used the first island they were recorded on as the most likely proxy for the natal island of the remaining 375 real individuals. Furthermore, dummy individuals that had at least one known parent ($N = 169$) were assigned the same natal island as their parent(s). Finally, dummy individuals without any known parent(s) ($N = 271$) were assigned the island where their offspring were born as their natal island. The proportional genetic group contribution values to the farm (q_{i1}) and non-farm (q_{i2}) genetic groups for each individual in the metapopulation-level pedigree were derived with the phantom parents as the starting point (i.e. with either 100% farm or 100% non-farm genetic group assignment), and where genetic group contributions of other individuals were 'propagated' through the pedigree and calculated using the ggcontrib function in the R package NADIV (Wolak, 2012). See Figure S1 for proportional genetic group contribution values to the non-farm genetic group (q_{i2}) for the 2602 individuals in the dispersal phenotype data set.

Our basic GGAM partitioning variation in dispersal probability, which accounts for differences in group-specific mean breeding values, was formulated using a binomial regression model where the linear predictor

$$\eta_i = \mu + sex_i\beta + \underbrace{q_{i2}g_2 + a_i}_{u_i} + island_i + hyear_i \quad (1)$$

for individual i was linked to its phenotype y_i via the logistic link function, where μ is the intercept and sex_i indicates sex of individual i with β as the estimated effect of sex. Individual sex (encoded as 0 = male and 1 = female) was included as a fixed effect to account for differences between sexes in dispersal propensity (Saatoglu et al., 2021).

The total additive genetic effect of individual i is given as u_i, which is the genetic group mean effect for the non-farm genetic group (g_2) weighted by the proportional non-farm genetic group contribution (q_{i2}) plus the breeding value a_i, distributed as $\mathbf{a}^\top=(a_1,\ldots,\ a_n)\sim N(\mathbf{0},\ \sigma_A{}^2\mathbf{A})$, with additive genetic variance $\sigma_A{}^2$ and additive genetic relatedness matrix $\mathbf{A}$ that represents the relatedness among individuals (Kruuk, 2004). To estimate the mean difference in the additive genetic effects for dispersal probability between the genetic groups, we considered the mean dispersal probability of the farm genetic group as the value of reference. Thus, the genetic group mean effect for the farm group was set to 0 (i.e. $g_1=0$) for identifiability reasons, and the estimate for g_2 thus encodes for the difference in the non-farm group's mean total additive genetic effect for dispersal probability compared to the farm group's mean total additive genetic effect for dispersal probability. Furthermore, individual i's natal island ($island_i\sim N(0,\sigma^2_{island})$) and hatch year ($hyear_i\sim N(0,\sigma^2_{year})$) were included as random effects to capture the variance in dispersal attributable to spatio-temporal environmental variation (Ranke et al., 2021; Saatoglu et al., 2021). Sex and q_{i2} were mean-centred prior to analyses. Note that, because our animal models were formulated as logistic regression models, the variance of the distribution when calculating heritability was set to $\pi^2/3$ (de Villemereuil et al., 2016).

The basic GGAM was then further extended to allow estimation of group-specific additive genetic variances. Our extended GGAM was thus formulated as a binomial regression model with logistic link function and linear predictor η_i given as

$$\eta_i = \mu + sex_i\beta + \underbrace{q_{i2}g_2 + a_{i1} + a_{i2}}_{u_i} + island_i + hyear_i, \quad (2)$$

where the total additive genetic effect of individual i is again given as u_i, which is now the sum of the genetic group mean effect for the non-farm genetic group (g_2) multiplied by the proportional non-farm genetic group contribution (q_{i2}), plus the group-specific additive genetic values of the farm genetic group (a_{i1}) and the non-farm genetic group (a_{i2}). Note that the breeding value a_i in model (1) is now split into two group-specific components a_{i1} and a_{i2}, with $\mathbf{a}_j^\top=(a_{1j},\ldots,a_{nj})\sim N(\mathbf{0},\sigma_{Aj}{}^2\mathbf{A}_j)$ for both groups $j=1, 2$, where $\sigma_{Aj}{}^2$ is the additive genetic variance in group j, and $\mathbf{A}_j$ are group-specific relatedness matrices calculated as in Muff et al. (2019). We denote a_{i1} and a_{i2} as the partial breeding values because they represent the contributions to the breeding value of individual i that are inherited from the farm and non-farm genetic groups, respectively.

Narrow-sense heritabilities for dispersal probability were obtained from (i) the basic GGAM for the whole study population combined, and (ii) the extended GGAM, where $\sigma_{Aj}{}^2$ was estimated group specific for the farm and non-farm genetic groups from the variance component estimates by using the formula (showing the basic GGAM case):

$$h^2 = \frac{\sigma^2_A}{\sigma^2_A + \sigma^2_{\text{island}} + \sigma^2_{\text{year}} + (\pi^2/3)}, \quad (3)$$

where the variances are defined as above, and the distribution-specific variance was approximated by $\pi^2/3$ (Nakagawa & Schielzeth, 2010). The group-specific heritability estimates for dispersal from the extended GGAM (h_j^2) were obtained by replacing $\sigma_A{}^2$ with group-specific $\sigma_{Aj}{}^2$ in formula (3). The proportion of phenotypic variance in dispersal explained by the natal island and hatch year was also estimated for the basic GGAM and the extended GGAM using the same formulas, but with σ^2_{island} or σ^2_{year} as the numerators respectively (instead of $\sigma_A{}^2$ or $\sigma_{Aj}{}^2$). Note that, for the sake of feasibility, we assume that σ^2_{island} and σ^2_{year} are the same within the farm and non-farm habitats for heritabilities and other proportions of phenotypic variance explained.

The basic GGAM and the extended GGAM were fitted in a Bayesian framework with integrated nested Laplace approximations using R-INLA (Rue et al., 2009), which is a fast and accurate alternative to MCMC (Holand et al., 2013; Steinsland et al., 2014). In order to prevent overfitting, a penalized complexity prior was used for the precisions of the environmental random components (with $u=2$ and $\alpha=0.02$) (Simpson et al., 2017).

2.4 | Genome-wide association analyses

To identify the potential associations of genomic regions with dispersal probability in the Helgeland house sparrow metapopulation, a GWA analysis on dispersal was carried out using the R package RepeatABEL (Rönnegård et al., 2016). A second round of SNP and sample filtering was carried out prior to the GWA analysis, resulting in a data set with 2602 individuals with phenotype data and genotypes at 183,070 SNPs (genotyping rate > 0.95, SNP call rate > 0.95, MAF > 0.01 and pairwise identity by state < 0.9). In the GWA model, sex was included as fixed factor to control its effect on dispersal, whereas hatch year and natal island were used as random factors to account for these environmental effects, and finally, the genomic relatedness matrix (GRM) was also added as a random effect to account for the genetic population structure and relatedness between individuals. The GWA analysis was carried out with a logistic model where dispersal was a binary response variable (disperser = 1; resident = 0). To reveal the SNPs that potentially may be in linkage disequilibrium (LD) with genetic variants affecting dispersal propensity, Bonferroni correction was applied with a family-wise error rate (FWER) of 0.05 (i.e. α was set to $0.05/183{,}070=2.73^{*}10^{-7}$). The genes flanking any SNP markers associated with dispersal were determined using the annotated house sparrow genome (Elgvin et al., 2017), and any annotation of these genes was investigated.

3 | RESULTS

3.1 | Heritable genetic variation in dispersal

In our house sparrow metapopulation data set, 484 of 2602 adults (18.6%) had dispersed between islands prior to recruitment (Table S1). Among the dispersers, 399 individuals (82.4% of

dispersers, and 15.3% of all recruits) dispersed to an island of the same habitat type as their natal island, whereas 85 individuals (17.6% of dispersers, and 3.3% of all recruits) either dispersed from a farm habitat island to a non-farm habitat island ($N=42$; 8.7% of dispersers, and 1.6% of recruits) or in the opposite direction ($N=43$; 8.9% of dispersers, and 1.7% of recruits). Considering the 2005 adults born on one of the farm habitat islands, a total of 13.5% had dispersed prior to recruitment: 11.4% had dispersed to another farm habitat island and 2.1% had dispersed to a non-farm habitat island. In contrast, 35.8% of the 597 adults born on one of the non-farm islands had dispersed prior to recruitment: 28.6% had dispersed to another non-farm habitat island and 7.2% had dispersed to a farm habitat island. Proportions of dispersing recruits produced by adult sparrows were considerably higher on non-farm habitat islands than on farm habitat islands (Figure 2; Table S1), and within each habitat type, there was a tendency for disperser parents of both sexes to produce a somewhat higher proportion of dispersing recruits than parents that were residents (Figure 2). The interchange of individuals between islands of different habitat types enabled us to use genetic groups animal models to separate heritable genetic causes from environmental causes of spatial variation in individual dispersal propensity because house sparrows with genomes partially originating from the farm genetic group were present in the non-farm habitat and vice versa (Figure S1).

The basic GGAM analysis suggested that the dispersal probability in our house sparrow metapopulation had a heritable genetic basis, with additive genetic variance explaining approximately 10% of the observed variation in dispersal probability (Table 1). Furthermore, the basic GGAM indicated that a considerable portion of the observed variation in dispersal probability among individuals was explained by environmental differences between natal islands (ca. 25%), in contrast to hatch years (ca. 1%), and provided strong evidence that females had a higher probability to disperse than males (Table 1). Finally, there was strong evidence from our basic GGAM that the estimated mean genetic value (i.e. mean breeding value) for dispersal was lower for the non-farm than the farm genetic group (Table 1).

Our extended GGAM analysis indicated that, despite lower overall dispersal rates, the farm genetic group had a higher additive genetic variance for dispersal probability than the non-farm genetic group (Figure 3, Table 1). The posterior difference in additive genetic variance between the farm and non-farm genetic groups had a mode of 0.556, with a 95% credible interval (CI) ranging from 0.045 to 1.134 (Figure S2), providing considerable evidence that the additive genetic variances were different. Correspondingly, the heritability of dispersal was higher in the farm habitat than in the non-farm habitat ($h^2=0.124$ and 0.017, respectively; Table 1). In contrast, the proportions of variation in dispersal probability explained by differences between hatch years and natal islands were similar in the two habitat types and similar to estimates from the basic GGAM (Table 1). In agreement with the basic GGAM, there was also strong evidence from the extended GGAM for a sex difference in dispersal probability, and that the estimated mean genetic value (i.e. mean breeding value) for dispersal was lower for the non-farm than the farm genetic group (Figure 3, Table 1).

3.2 | Genome-wide association analyses

GWA analyses on dispersal phenotype revealed only a single region on chromosome 15 that may predict dispersal propensity (Figure 4). The top SNP identified in this region, SNPa105044, explained 3.8%

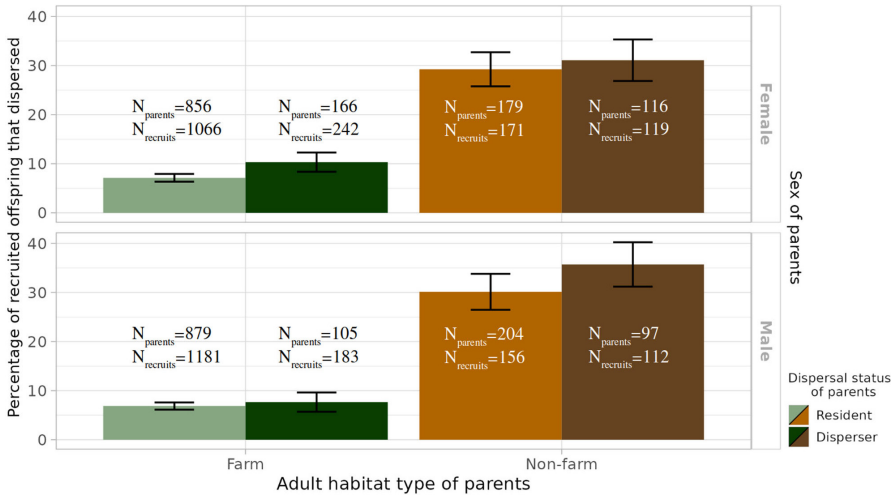

FIGURE 2 The proportion (in %) of recruited offspring produced by different types of female (top) and male (bottom) house sparrow parents in an insular metapopulation. Parents were classified as either residents (i.e. they were adults on the same island they were born; light-shaded bars) or dispersers (i.e. they were adults on a different island than the one they were born on; dark-shaded bars). Standard errors of percentages are indicated with black bars and were estimated assuming that the proportions were described by a binomial distribution (Whitlock & Schluter, 2014). Dispersal can occur between any of the islands in the metapopulation, but parents were further divided into those that were adult on one of the islands of the farm habitat type (green coloured bars) or one of the islands of the non-farm (brown coloured bars) habitat type.

TABLE 1 Posterior statistics for fixed effects and random effects from a basic genetic groups animal model (GGAM) and an extended GGAM for dispersal probability in house sparrows.

		Posterior estimate of fixed or random effect (variance component)		Proportion variation explained by random effect (variance component)	
	Parameter	Basic GGAM	Extended GGAM	Basic GGAM	Extended GGAM
Fixed effects	Sex (female)	0.470 (0.249, 0.690)	0.474 (0.250, 0.690)	–	–
	Genetic Group (non-farm)	−1.154 (−1.793, −0.519)	−0.819 (−1.440, −0.201)	–	–
Random effects	Natal island	1.067; 1.619 (0.537, 3.963)	1.028; 1.541 (0.497, 3.724)	0.271; 0.262 (0.180, 0.372)	–
	Natal island (farm)	–	–	–	0.210; 0.243 (0.167, 0.320)
	Natal island (non-farm)	–	–	–	0.280; 0.272 (0.188, 0.343)
	Hatch year	0.042; 0.074 (0.017, 0.198)	0.035; 0.067 (0.013, 0.187)	0.019; 0.014 (0.008, 0.022)	–
	Hatch year (farm)	–	–	–	0.013; 0.012 (0.006, 0.018)
	Hatch year (non-farm)	–	–	–	0.009; 0.013 (0.007, 0.020)
	Additive genetic variance	0.448; 0.489 (0.236, 0.821)	–	0.098; 0.121 (0.070, 0.123)	–
	Additive genetic variance (farm)	–	0.696; 0.769 (0.405, 1.278)	–	0.124; 0.140 (0.107, 0.167)
	Additive genetic variance (non-farm)	–	0.034; 0.167 (0.011, 0.580)	–	0.017; 0.038 (0.017, 0.067)

Note: The GGAM included two genetic groups, corresponding to the farm and non-farm island habitats. The basic GGAM allowed mean breeding values for dispersal to differ between the two genetic groups, whereas the extended GGAM also allowed the additive genetic variances to differ between the two genetic groups. The mean posterior estimate with 95% credible interval (CI, in parenthesis) is presented for fixed effects. For random-effects variances and proportions of phenotypic variance explained, the posterior mode and mean (formatted: mode; mean) are presented, with the 95% CI in parenthesis. The proportion phenotypic variance explained by additive genetic variance is equal to the heritability of dispersal probability.

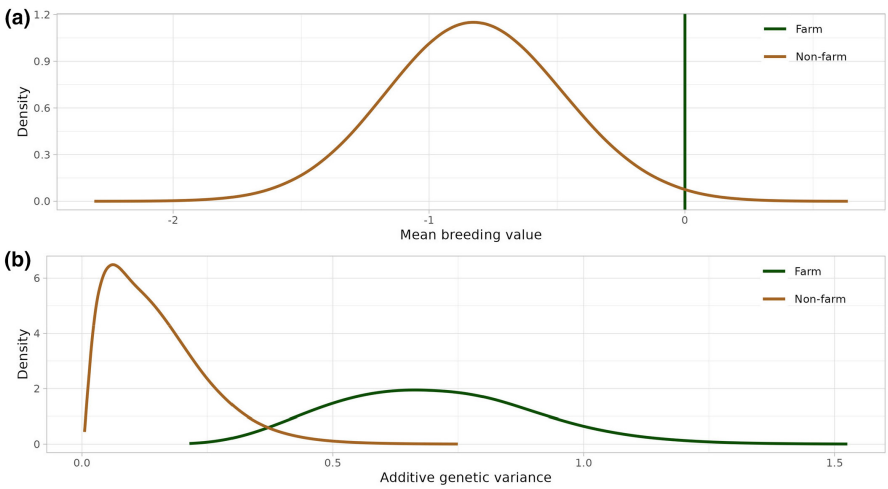

FIGURE 3 Posterior distributions of mean breeding values (a) and additive genetic variances (b) from the extended genetic group animal model. Panel a shows the estimated posterior distribution of the mean breeding value for the non-farm genetic group in brown. Because the farm genetic group was set to be the baseline mean (equal to zero), the mean breeding value for this habitat type is shown as a green vertical line. In panel b, the posterior distributions for additive genetic variances for the farm and non-farm genetic groups are shown in green and brown respectively.

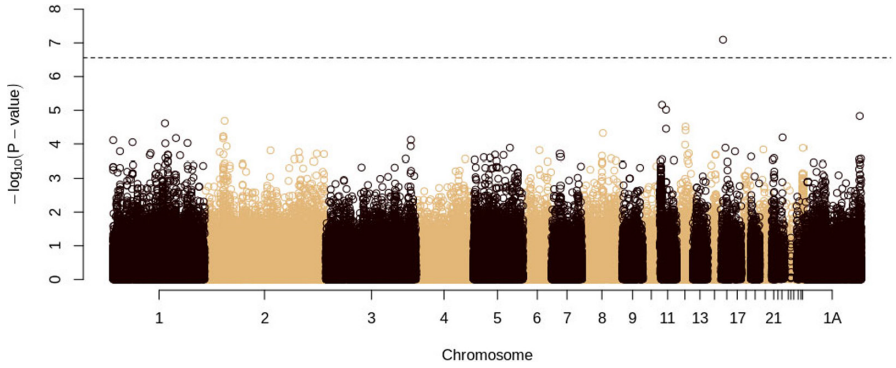

FIGURE 4 Results from the GWAS on dispersal phenotype for 2602 house sparrows showing the negative logarithm of the *p*-value for 183,070 autosomal markers against their chromosomal position in the house sparrow genome. Markers on chromosome 16 were not on the genotyping array, and those markers located on the Z and W chromosomes or without a known position were not included in the GWAS. Sex was included as a fixed factor in the model; and hatch year, natal island and GRM were included as random effects. One marker, SNPa105044 on chromosome 15, was associated with dispersal phenotype at an FWER of 0.05 (equivalent to an α of $0.05/183{,}070 = 2.73*10^{-7}$, given by the horizontal dashed line). This marker is 16 Kbp from ADORA2A in the house sparrow genome and 22 Kbp from UPB1.

of the variance in dispersal probability (Table S2; Figure S4) and is located 16 Kbp downstream from adenosine receptor A2a (ADORA2A) and 22 Kbp upstream from beta-ureidopropionase (UPB1) in the house sparrow genome. It has been shown that ADORA2A encodes a member of the *G* protein-coupled receptor superfamily that is involved in increasing intracellular cAMP levels and is a regulator of functions including sleep cycles, cardiac rhythm and circulation, immune function and pain regulation (NCBI), as well as glycogenesis (González-Benítez et al., 2002), whereas UPB1 encodes a highly conserved protein that catalyses a late step in the nucleic acid pyrimidine degradation leading to biosynthesis of beta-alanine in animals (Matthews et al., 1992). In humans, UPB1 deficiency is associated with neurological problems (Dobritzsch et al., 2022; Van Kuilenburg et al., 2004), and beta-alanine supplementation has been shown to increase performance during intense exercise by acid buffering of the blood (Hobson et al., 2012; Milioni et al., 2019).

4 | DISCUSSION

In the present study, we investigated the genetic architecture of dispersal in an insular metapopulation of house sparrows by estimating additive genetic and environmental variance components complemented by a genome-wide association analysis. Our house sparrow metapopulation is particularly interesting for such a study, as previous publications have shown that birds differ in dispersal probability depending on whether they originate from a farm or non-farm habitat type of island (Pärn et al., 2012; Ranke et al., 2021; Saatoglu et al., 2021). We found that in this metapopulation, heritable genetic variation explained only approximately 10% of the variation in the dispersal probability of recruited adults. However, by using novel statistical methods that allow for mean and variance in heritable genetic variation to differ between genetic groups, we revealed that the farm and non-farm habitats differ in both mean breeding values and additive genetic variances for dispersal. Specifically, although phenotypic dispersal probabilities are higher in the non-farm habitat, the mean breeding value and the additive genetic variance (as well as the heritability) for dispersal were higher in the farm habitat than in the non-farm habitat.

It is challenging to obtain high-quality data on dispersal because the study system needs to be sufficiently large to cover normal dispersal distances of the organisms, resident and dispersing individuals need to be individually recognizable, and to estimate either the heritable genetic component of dispersal or its fitness consequences, cross-generational data that include information also on the descendants of dispersers and residents are advantageous (Cayuela et al., 2018; Holyoak et al., 2008; Millon et al., 2019). Despite these challenges, the genetic basis of dispersal phenotype and dispersal-related traits have been researched on occasion even in vertebrates in the wild, using either parent–offspring regressions or animal models (Table S3). Parameter estimates derived from animal models, which account for all kinds of genetic relatives and are regarded as potentially less biased by, for example, non-genetic environment effects than parent–offspring regressions (Kruuk et al., 2008), tend to report more modest magnitudes of ${\sigma_A}^2$ and h^2 than parent–offspring regressions (Saastamoinen et al., 2018). Furthermore, animal model-based h^2 estimates of dispersal propensity for birds in natural populations were reported to have a range of 0.36–0.95 (Table S3). Moreover, the h^2 of dispersal were estimated to be 0.170 and 0.280 in a semi-natural common lizards (*Zootoca vivipara*; San-Jose et al., 2023) study and an experimental cane toad (*Bufo marinus/Rhinella marina*; Phillips et al., 2010) study, respectively. Hence, the h^2 estimates of dispersal for other species are usually higher than those we documented in house sparrows, although our estimate of ${\sigma_A}^2$ for the farm habitat was slightly higher than in the metapopulation as a whole, with a h^2 of 0.12 in this habitat type (Table 1). In combination, the relatively few studies on the heritable genetic basis for dispersal propensity that exist from natural vertebrate populations (Table S3) suggest that this

key life-history trait has the capacity for adaptive evolution on ecological time scales if any selection is acting on it, but that its rate of micro-evolution may differ somewhat between species and even between populations within the same species. Indeed, in another study of the same house sparrow metapopulation, we have shown that immigrants have higher lifetime fitness than resident individuals (as estimated by annual production of recruiting offspring and number of recruiting offspring produced over the life span; Saatoglu et al., in prep.). Consequently, and assuming similar positive directional selection in both environments, dispersal rates could be expected to increase across generations at higher rates for the farm habitat in our metapopulation.

Moreover, the contrasting results for the two habitat types such as lower mean breeding values but higher dispersal probabilities in the non-farm habitat type and differences between islands within each habitat type in dispersal probabilities (Figure S3) suggest that there may be a genotype-by-environment interaction (*GxE*) for dispersal in our house sparrow metapopulation. If such an interaction exists, one would expect that birds with genomes originating from non-farm islands will respond differently to the farm environment with respect to their dispersal probabilities, and vice versa. Birds that disperse between habitat types and the descendants of such inter-habitat dispersers (see Figure S1) can not only be used to separate additive genetic effects from environmental causes of observed differences in dispersal probabilities such as we have done here (Table 1; Figure 3) but they also allow for examining *GxE* in dispersal. Testing whether there is a *GxE* for dispersal in our study metapopulation, and investigating any causes and consequences of such an interaction is, however, outside the scope of the current paper and should be examined in a future study.

Dispersal in our house sparrow metapopulation occurs during the fledged juvenile phase in the autumn before the juveniles' first winter (Pärn et al., 2009; Ranke et al., 2021; Saatoglu et al., 2021). Thus, it seems likely that environmental conditions related to the population density, weather or various habitat characteristics that offspring experience during development may also affect the propensity to disperse. Accordingly, we have previously shown in the same study system that dispersal rates were higher when preceding springs were warmer, breeding started early and total population sizes at the end of the breeding season were higher (Pärn et al., 2012). Condition-dependent dispersal probabilities that are influenced by environmental conditions such as population density, prenatal/postnatal environmental conditions and/or physiological traits underlying the movement capacity have also been documented in many other studies of vertebrates (Boualit et al., 2019; Leon et al., 2022; Maag et al., 2018; Massot et al., 2002; Matthysen, 2005; McCaslin et al., 2020; Messier et al., 2012; Saastamoinen et al., 2018; Walls et al., 2005; Wu & Seebacher, 2022). Interestingly, the relationships between dispersal and environmental conditions in our house sparrow metapopulation actually differed between habitat types: dispersal rates were positively related to spring temperature, onset of breeding and total population density in non-farm habitat islands, while dispersal was independent of these environmental conditions in farm habitat islands (Pärn et al., 2012). Despite higher average dispersal rates in the non-farm habitat than in the farm habitat (Ranke et al., 2021; Saatoglu et al., 2021), the results in the current study show lower estimated mean breeding values and lower additive genetic variances for dispersal in the non-farm habitat than the farm habitat (Table 1), which suggest that when individuals make their dispersal decisions, environmental components are more influential than heritable genetic effects in the non-farm habitat. In any case, although gene flow is expected to reduce genetic differences between populations (Holsinger & Weir, 2009), the low dispersal rates between the two habitat types relative to dispersal rates within habitat types (Table S1) may have contributed to generating, and could help maintaining, the differences in breeding values, additive genetic variances and importance of environmental conditions for dispersal between farm and non-farm habitats we found in our study system.

Recently, gene mapping studies using a GWAS approach have been able to identify genes underlying phenotypic variation in various heritable life-history- and fitness-related traits even in natural vertebrate populations (e.g. Barson et al., 2015; Husby et al., 2015; Johnston et al., 2011; Lawson & Petren, 2017; Lundregan et al., 2018; Tietgen et al., 2021). Here, we have revealed that a single marker on chromosome 15 was linked with dispersal trait in the house sparrow metapopulation and this marker was closest to the ADORA2A receptor gene. This receptor gene is located near the UPB1 gene both in the house sparrow genome (Elgvin et al., 2017) and the zebra finch genome (Warren et al., 2010). ADORA2A is involved in glycogenolysis (i.e. release of glucose into the bloodstream; see González-Benítez et al., 2002), thus ADORA2A may influence energy dynamics. Glucose metabolism has been shown to affect dispersal rate in the Glanville fritillary butterfly (*Melitaea cinxia*), for which the *Pgi* gene explains variation in dispersal rate and is involved in breakdown of glucose to produce ATP (Hanski et al., 2017; Niitepõld & Saastamoinen, 2017). Interestingly, another function of ADORA2A is to increase intracellular cAMP levels which are not only important in metabolism and wakefulness but are also an important aspect of the circadian regulatory mechanism that has direct influence on the clock phase (O'Neill & Reddy, 2012). A recent study on a semi-natural population of common lizards showed that expression of circadian clock genes differed between dispersers and residents. However, ADORA2A or UPB1 were not among the dispersal-related genes identified in this species (San-Jose et al., 2023). Moreover, clock-linked genes may also influence migratory timing in the American kestrel (*Falco sparverius*; Bossu et al., 2022). Hence, although few studies exist and the functional relationship between putative genes and dispersal in most cases needs to be explored further, there appears to be some evidence that genes related to (flight) energy metabolism and circadian rhythms are related to the individual dispersal processes. The top SNP in our GWAS was estimated to explain 3.8% of the variance in dispersal probability (Table S2), which is a considerable proportion of our estimated heritability for dispersal (Table 1). However, effect sizes are likely overestimated in GWAS

where sample sizes are relatively small and effect sizes are estimated in the same data set where associations are discovered (Uffelmann et al., 2021). Thus, it seems likely that dispersal propensity, at least in our house sparrow metapopulation, is a polygenic trait with a complex basis that involves both genes and environmental effects.

In summary, we have shown that there is a habitat-dependent heritable basis for dispersal, which is an important life-history trait because of its close connection with spatio-temporal ecological and evolutionary dynamics across geographically structured populations (Clobert et al., 2012; Saastamoinen et al., 2018). The ability of evolutionary ecologists to partition a natural population's phenotypic variance in key traits into a heritable genetic component and environmental components of variation advanced when animal models were introduced to the field approximately two decades ago (Kruuk, 2004; Wilson et al., 2010). Here, we have exploited the recent development of genetic groups animal models that allow for exploring and quantifying spatial variation in heritable genetic variation (Aase et al., 2022; Muff et al., 2019) and showed that the rate of any adaptive evolutionary change in dispersal may differ across space in a fragmented population. In a rapidly changing world, where many populations become increasingly fragmented and range shifts may be necessary to avoid extinction, quantifying such spatial variation and understanding its consequences for ecological and evolutionary processes are likely to be of increasing importance.

AUTHOR CONTRIBUTIONS

The fieldwork was carried out by H.J., D.S., S.L.L. and A.H.. A.K.N. and H.J. developed the pedigree. D.S. carried out genetic assignment. D.S. and S.L.L. performed the statistical analyses and interpreted the results with input from A.K.N., D.G., S.M. and H.J.. D.S. drafted the initial version of the manuscript with input from S.L.L., D.G., A.K.N., S.M. and H.J.. All authors contributed to later versions of the manuscript, and all authors gave final approval for publication.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data associated with this study have been made available on the Dryad Digital Repository (DOI: 10.5061/dryad.80gb5mkxh).

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REFERENCES

Aase, K., Jensen, H., & Muff, S. (2022). Genomic estimation of quantitative genetic parameters in wild admixed populations. *Methods in Ecology and Evolution*, *13*(5), 1014–1026. https://doi.org/10.1111/2041-210X.13810

Altwegg, R., Ringsby, T. H., & Sæther, B.-E. (2000). Phenotypic correlates and consequences of dispersal in a metapopulation of house sparrows Passer domesticus. *Journal of Animal Ecology*, *69*(5), 762–770. https://doi.org/10.1046/j.1365-2656.2000.00431.x

Araya-Ajoy, Y. G., Ranke, P. S., Kvalnes, T., Rønning, B., Holand, H., Myhre, A. M., Pärn, H., Jensen, H., Ringsby, T. H., Sæther, B-E., & Wright, J. (2019). Characterizing morphological (co)variation using structural equation models: Body size, allometric relationships and evolvability in a house sparrow metapopulation. *Evolution*, *73*(3), 452–466. https://doi.org/10.1111/evo.13668

Baalsrud, H. T., Sæther, B. E., Hagen, I. J., Myhre, A. M., Ringsby, T. H., Pärn, H., & Jensen, H. (2014). Effects of population characteristics and structure on estimates of effective population size in a house sparrow metapopulation. *Molecular Ecology*, *23*(11), 2653–2668. https://doi.org/10.1111/mec.12770

Barson, N. J., Aykanat, T., Hindar, K., Baranski, M., Bolstad, G. H., Fiske, P., Jacq, C., Jensen, A. J., Johnston, S. E., Karlsson, S., Kent, M., Moen, T., Niemelä, E., Nome, T., Næsje, T. F., Orell, P., Romakkaniemi, A., Sægrov, H., Urdal, K., … Primmer, C. R. (2015). Sex-dependent dominance at a single locus maintains variation in age at maturity in salmon. *Nature*, *528*(7582), 405–408. https://doi.org/10.1038/nature16062

Bossu, C. M., Heath, J. A., Kaltenecker, G. S., Helm, B., & Ruegg, K. C. (2022). Clock-linked genes underlie seasonal migratory timing in a diurnal raptor. *Proceedings of the Royal Society B: Biological Sciences*, *289*, 20212507. https://doi.org/10.1098/rspb.2021.2507

Boualit, L., Pichenot, J., Besnard, A., Helder, R., Joly, P., & Cayuela, H. (2019). Environmentally mediated reproductive success predicts breeding dispersal decisions in an early successional amphibian. *Animal Behaviour*, *149*, 107–120. https://doi.org/10.1016/j.anbehav.2019.01.008

Bowler, D. E., & Benton, T. G. (2005). Causes and consequences of animal dispersal strategies: Relating individual behaviour to spatial dynamics. *Biological Reviews*, *80*(2), 205–225. https://doi.org/10.1017/S1464793104006645

Brown, G. P., Phillips, B. L., & Shine, R. (2014). The straight and narrow path: The evolution of straight-line dispersal at a cane toad invasion front. *Proceedings of the Royal Society B: Biological Sciences*, *281*, 20141385. https://doi.org/10.1098/rspb.2014.1385

Cayuela, H., Rougemont, Q., Prunier, J. G., Moore, J. S., Clobert, J., Besnard, A., & Bernatchez, L. (2018). Demographic and genetic approaches to study dispersal in wild animal populations: A methodological review. *Molecular Ecology*, *27*(20), 3976–4010. https://doi.org/10.1111/mec.14848

Clobert, J., Baguette, M., Benton, T. G., & Bullock, J. M. (2012). *Dispersal ecology and evolution* (2012th ed.). Oxford University Press.

de Villemereuil, P., Schielzeth, H., Nakagawa, S., & Morrissey, M. (2016). General methods for evolutionary quantitative genetic inference from generalized mixed models. *Genetics*, *204*(3), 1281–1294. https://doi.org/10.1534/genetics.115.186536

Dobritzsch, D., Meijer, J., Meinsma, R., Maurer, D., Monavari, A. A., Gummesson, A., Reims, A., Cayuela, J. A., Kuklina, N., Benoist, J-F., Perrin, L., Assmann, B., Hoffmann, G. F., Bierau, J., Kaindl, A. M., & van Kuilenburg, A. B. P. (2022). β-Ureidopropionase deficiency due to novel and rare UPB1 mutations affecting pre-mRNA splicing and protein structural integrity and catalytic activity. *Molecular Genetics and Metabolism*, *136*(3), 177–185. https://doi.org/10.1016/j.ymgme.2022.01.102

Dochtermann, N. A., Schwab, T., Anderson Berdal, M., Dalos, J., & Royauté, R. (2019). The heritability of behavior: A meta-analysis. *Journal of Heredity*, *110*(4), 403–410. https://doi.org/10.1093/jhered/esz023

Edelsparre, A. H., Vesterberg, A., Lim, J. H., Anwari, M., & Fitzpatrick, M. J. (2014). Alleles underlying larval foraging behaviour influence adult dispersal in nature. *Ecology Letters*, *17*(3), 333–339. https://doi.org/10.1111/ele.12234

Elgvin, T. O., Trier, C. N., Tørresen, O. K., Hagen, I. J., Lien, S., Nederbragt, A. J., Ravinet, M., Jensen, H., & Sætre, G.-P. (2017). The genomic mosaicism of hybrid speciation. *Science Advances*, *3*(6), e1602996. https://doi.org/10.1126/sciadv.1602996

González-Benítez, E., Guinzberg, R., Díaz-Cruz, A., & Pia, E. (2002). Regulation of glycogen metabolism in hepatocytes through adenosine receptors. Role of Ca2+ and cAMP. *European Journal of Pharmacology*, *437*(3), 105–111. https://doi.org/10.1016/S0014-2999(02)01299-2

Haag, C. R., Saastamoinen, M., Marden, J. H., & Hanski, I. (2005). A candidate locus for variation in dispersal rate in a butterfly metapopulation. *Proceedings of the Royal Society B: Biological Sciences*, *272*(1580), 2449–2456. https://doi.org/10.1098/rspb.2005.3235

Hanski, I., Schulz, T., Wong, S. C., Ahola, V., Ruokolainen, A., & Ojanen, S. P. (2017). Ecological and genetic basis of metapopulation persistence of the Glanville fritillary butterfly in fragmented landscapes. *Nature Communications*, *8*, 14504. https://doi.org/10.1038/ncomms14504

Hobson, R. M., Saunders, B., Ball, G., Harris, R. C., & Sale, C. (2012). Effects of β-alanine supplementation on exercise performance: A meta-analysis. *Amino Acids*, *43*(1), 25–37. https://doi.org/10.1007/s00726-011-1200-z

Holand, A. M., Steinsland, I., Martino, S., & Jensen, H. (2013). Animal models and integrated nested laplace approximations. *G3: Genes, Genomes, Genetics*, *3*(8), 1241–1251. https://doi.org/10.1534/g3.113.006700

Holsinger, K. E., & Weir, B. S. (2009). Genetics in geographically structured populations: Defining, estimating and interpreting FST. *Nature Reviews Genetics*, *10*(9), 639–650. https://doi.org/10.1038/nrg2611

Holyoak, M., Casagrandi, R., Nathan, R., Revilla, E., & Spiegel, O. (2008). Trends and missing parts in the study of movement ecology. *Proceedings of the National Academy of Sciences of the United States of America*, *105*(49), 19060–19065. https://doi.org/10.1073/pnas.0800483105

Huisman, J. (2017). Pedigree reconstruction from SNP data: Parentage assignment, sibship clustering and beyond. *Molecular Ecology Resources*, *17*(5), 1009–1024. https://doi.org/10.1111/1755-0998.12665

Husby, A., Kawakami, T., Rönnegård, L., Smeds, L., Ellegren, H., & Qvarnström, A. (2015). Genome-wide association mapping in a wild avian population identifies a link between genetic and phenotypic variation in a life-history trait. *Proceedings of the Royal Society B: Biological Sciences*, *282*(1806), 20150156. https://doi.org/10.1098/rspb.2015.0156

Johnston, S. E., McEwan, J. C., Pickering, N. K., Kijas, J. W., Beraldi, D., Pilkington, J. G., Pemberton, J. M., & Slate, J. (2011). Genome-wide association mapping identifies the genetic basis of discrete and quantitative variation in sexual weaponry in a wild sheep population. *Molecular Ecology*, *20*(12), 2555–2566. https://doi.org/10.1111/j.1365-294X.2011.05076.x

Korte, A., & Farlow, A. (2013). The advantages and limitations of trait analysis with GWAS: A review self-fertilisation makes Arabidopsis particularly well suited to GWAS. *Plant Methods*, *9*(1), 29.

Kruuk, L. E. B. (2004). Estimating genetic parameters in natural populations using the "animal model.". *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *359*(1446), 873–890. https://doi.org/10.1098/rstb.2003.1437

Kruuk, L. E. B., Slate, J., & Wilson, A. J. (2008). New answers for old questions: The evolutionary quantitative genetics of wild animal populations. *Annual Review of Ecology, Evolution, and Systematics*, *39*, 525–548. https://doi.org/10.1146/annurev.ecolsys.39.110707.173542

Kuismin, M., Saatoglu, D., Niskanen, A. K., Jensen, H., & Sillanpää, M. J. (2020). Genetic assignment of individuals to source populations using network estimation tools. *Methods in Ecology and Evolution*, *11*(2), 333–344. https://doi.org/10.1111/2041-210X.13323

Lande, R. (1979). Quantitative genetic analysis of multivariate evolution, applied to brain: Body size Allometry. *Evolution*, *33*(1), 402. https://doi.org/10.2307/2407630

Lawson, L. P., & Petren, K. (2017). The adaptive genomic landscape of beak morphology in Darwin's finches. *Molecular Ecology*, *26*(19), 4978–4989. https://doi.org/10.1111/mec.14166

Legrand, D., Cote, J., Fronhofer, E. A., Holt, R. D., Ronce, O., Schtickzelle, N., Travis, J. M. J., & Clobert, J. (2017). Eco-evolutionary dynamics in fragmented landscapes. *Ecography*, *40*(1), 9–25. https://doi.org/10.1111/ecog.02537

Leon, C., Banks, S., Beck, N., & Heinsohn, R. (2022). Population genetic structure and dispersal patterns of a cooperative breeding bird in variable environmental conditions. *Animal Behaviour*, *183*, 127–137. https://doi.org/10.1016/j.anbehav.2021.11.005

Lundregan, S. L., Hagen, I. J., Gohli, J., Niskanen, A. K., Kemppainen, P., Ringsby, T.-H., Kvalnes, T., Pärn, H., Rønning, B., Holand, H., Ranke, P. S., Båtnes, A. S., Selvik, L-K., Lien, S., Sæther, B-E., Husby, A., & Jensen, H. (2018). Inferences of genetic architecture of bill morphology in house sparrow using a high – density SNP array point to a polygenic basis. *Molecular Ecology*, *27*, 3498–3514. https://doi.org/10.1111/mec.14811

Lynch, M., & Walsh, B. (1998). *Genetics and analysis of quantitative traits* (Vol. *1*). Sinauer Sunderland.

Maag, N., Cozzi, G., Clutton-Brock, T., & Ozgul, A. (2018). Density-dependent dispersal strategies in a cooperative breeder. *Ecology*, *99*(9), 1932–1941. https://doi.org/10.1002/ecy.2433

Massot, M., Clobert, J., Lorenzon, P., & Rossi, J. M. (2002). Condition-dependent dispersal and ontogeny of the dispersal behaviour: An experimental approach. *Journal of Animal Ecology*, *71*(2), 253–261. https://doi.org/10.1046/j.1365-2656.2002.00592.x

Matthews, M. M., Liao, W., Kvalnes-Krick, K. L., & Traut, T. W. (1992). β-Alanine synthase: Purification and allosteric properties. *Archives of Biochemistry and Biophysics*, *293*(2), 254–263. https://doi.org/10.1016/0003-9861(92)90393-B

Matthysen, E. (2005). Density-dependent dispersal in birds and mammals. *Ecography*, *28*(3), 403–416. https://doi.org/10.1111/j.0906-7590.2005.04073.x

Mattila, A. L. K., & Hanski, I. (2014). Heritability of flight and resting metabolic rates in the Glanville fritillary butterfly. *Journal of Evolutionary Biology*, *27*(8), 1733–1743. https://doi.org/10.1111/jeb.12426

McCaslin, H. M., Caughlin, T. T., & Heath, J. A. (2020). Long-distance natal dispersal is relatively frequent and correlated with environmental factors in a widespread raptor. *Journal of Animal Ecology*, *89*(9), 2077–2088. https://doi.org/10.1111/1365-2656.13272

McGaugh, S. E., Schwanz, L. E., Bowden, R. M., Gonzalez, J. E., & Janzen, F. J. (2010). Inheritance of nesting behaviour across natural environmental variation in a turtle with temperature-dependent sex determination. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1685), 1219–1226. https://doi.org/10.1098/rspb.2009.1883

Messier, G. D., Garant, D., Bergeron, P., & Réale, D. (2012). Environmental conditions affect spatial genetic structures and dispersal patterns in a solitary rodent. *Molecular Ecology*, *21*(21), 5363–5373. https://doi.org/10.1111/mec.12022

Milioni, F., De Poli, R. A. B., Saunders, B., Gualano, B., Da Rocha, A. L., Da Silva, A. S. R., De Tarso Guerrero Muller, P., & Zagatto, A. M. (2019). Effect of β-alanine supplementation during high-intensity interval training on repeated sprint ability performance and neuromuscular fatigue. *Journal of Applied Physiology*, *127*(6), 1599–1610. https://doi.org/10.1152/japplphysiol.00321.2019

Millon, A., Lambin, X., Devillard, S., & Schaub, M. (2019). Quantifying the contribution of immigration to population dynamics: A review of methods, evidence and perspectives in birds and mammals. *Biological Reviews*, *94*, 2049–2067. https://doi.org/10.1111/brv.12549

Money, D., Gardner, K., Migicovsky, Z., Schwaninger, H., Zhong, G. Y., & Myles, S. (2015). LinkImpute: fast and accurate genotype imputation for nonmodel organisms. *G3: Genes, Genomes, Genetics*, *5*(11), 2383–2390. https://doi.org/10.1534/g3.115.021667

Muff, S., Niskanen, A. K., Saatoglu, D., Jensen, H., & Keller, L. F. (2019). Animal models with group-specific additive genetic variances: Extending genetic group models. *Genetics Selection Evolution*, *51*, 7. https://doi.org/10.1186/s12711-019-0449-7

Nakagawa, S., & Schielzeth, H. (2010). Repeatability for Gaussian and non-Gaussian data: A practical guide for biologists. *Biological Reviews*, *85*(4), 935–956. https://doi.org/10.1111/j.1469-185X.2010.00141.x

Niitepõld, K., Smith, A. D., Osborne, J. L., Reynolds, D. R., Carreck, N. L., Martin, A. P., Marden, J. H., Ovaskainen, O., & Hanski, I. (2009). Flight metabolic rate and Pgi genotype influence butterfly dispersal rate in the field. *Ecology*, *90*(8), 2223–2232. https://doi.org/10.1890/08-1498.1

Niitepõld, K., Mattila, A. L. K., Harrison, P. J., & Hanski, I. (2011). Flight metabolic rate has contrasting effects on dispersal in the two sexes of the Glanville fritillary butterfly. *Oecologia*, *165*(4), 847–854. https://doi.org/10.1007/s00442-010-1886-8

Niitepõld, K., & Saastamoinen, M. (2017). A candidate gene in an ecological model species: Phosphoglucose isomerase (Pgi) in the glanville fritillary butterfly (*Melitaea cinxia*). *Annales Zoologici Fennici*, *54*(1–4), 259–273. https://doi.org/10.5735/086.054.0122

Niskanen, A. K., Billing, A. M., Holand, H., Hagen, I. J., Araya-Ajoy, Y. G., Husby, A., Rønning, B., Myhre, A. M., Ranke, P. S., Kvalnes, T., Pärn, H., Ringsby, T. H., Lien, S., Sæther, B.-E., Muff, S., & Jensen, H. (2020). Consistent scaling of inbreeding depression in space and time in a house sparrow metapopulation. *Proceedings of the National Academy of Sciences of the United States of America*, *117*(25), 14584–14592. https://doi.org/10.1073/pnas.1909599117

O'Neill, J. S., & Reddy, A. B. (2012). The essential role of cAMP/Ca 2+ signalling in mammalian circadian timekeeping. *Biochemical Society Transactions*, *40*(1), 44–50. https://doi.org/10.1042/BST20110691

Orr, H. A. (2005). The genetic theory of adaptation: A brief history. *Nature Reviews Genetics*, *6*(2), 119–127. https://doi.org/10.1038/nrg1523

Pärn, H., Jensen, H., Ringsby, T. H., & Sæther, B. E. (2009). Sex-specific fitness correlates of dispersal in a house sparrow metapopulation. *Journal of Animal Ecology*, *78*(6), 1216–1225. https://doi.org/10.1111/j.1365-2656.2009.01597.x

Pärn, H., Ringsby, T.-H., Jensen, H., & Sæther, B.-E. (2012). Spatial heterogeneity in the effects of climate and density-dependence on dispersal in a house sparrow metapopulation. *Proceedings of the Royal Society B: Biological Sciences*, *279*(1726), 144–152. https://doi.org/10.1098/rspb.2011.0673

Phillips, B. L., Brown, G. P., & Shine, R. (2010). Evolutionarily accelerated invasions: The rate of dispersal evolves upwards during the range advance of cane toads. *Journal of Evolutionary Biology*, *23*(12), 2595–2601. https://doi.org/10.1111/j.1420-9101.2010.02118.x

Ranke, P. S., Araya-Ajoy, Y. G., Ringsby, T. H., Pärn, H., Rønning, B., Jensen, H., Wright, J., & Sæther, B. E. (2021). Spatial structure and dispersal dynamics in a house sparrow metapopulation. *Journal of Animal Ecology*, *90*(12), 2767–2781. https://doi.org/10.1111/1365-2656.13580

Remington, D. L. (2015). Alleles versus mutations: Understanding the evolution of genetic architecture requires a molecular perspective on allelic origins. *Evolution*, *69*(12), 3025–3038. https://doi.org/10.1111/evo.12775

Ronce, O. (2007). How does it feel to Be like a rolling stone? Ten questions about dispersal evolution. *Annual Review of Ecology, Evolution, and Systematics*, *38*, 231–253. https://doi.org/10.1146/annurev.ecolsys.38.091206.095611

Rönnegård, L., McFarlane, S. E., Husby, A., Kawakami, T., Ellegren, H., & Qvarnström, A. (2016). Increasing the power of genome wide association studies in natural populations using repeated measures – Evaluation and implementation. *Methods in Ecology and Evolution*, *7*(7), 792–799. https://doi.org/10.1111/2041-210X.12535

Rue, H., Martino, S., & Chopin, N. (2009). Approximate Bayesian inference for latent Gaussian models by using integrated nested Laplace approximations. *Journal of the Royal Statistical Society, Series B: Statistical Methodology*, *71*(2), 319–392. https://doi.org/10.1111/j.1467-9868.2008.00700.x

Saastamoinen, M. (2008). Heritability of dispersal rate and other life history traits in the Glanville fritillary butterfly. *Heredity*, *100*(1), 39–46. https://doi.org/10.1038/sj.hdy.6801056

Saastamoinen, M., Bocedi, G., Cote, J., Legrand, D., Guillaume, F., Wheat, C. W., Fronhofer, E. A., Garcia, C., Henry, R., Husby, A., Baguette, M., Bont, D., Coulon, A., Kokko, H., Matthysen, E., Niitepõld, K., Nonaka, E., Stevens, V. M., Travis, J. M. J., … del Mar Delgado, M. (2018). Genetics of dispersal. *Biological Reviews*, *93*(1), 574–599. https://doi.org/10.1111/brv.12356

Saatoglu, D., Niskanen, A. K., Froy, H., Ranke, P. S., Goedert, D., Reid, J. M., Ringsby, T. H., Sæther, B-E., Araya-Ajoy, Y. G., & Jensen, H. (in prep.). *Metapopulation-level analyses reveal positive fitness-consequences of dispersal in a small bird*.

Saatoglu, D., Niskanen, A. K., Kuismin, M., Ranke, P. S., Hagen, I. J., Araya-Ajoy, Y. G., Kvalnes, T., Pärn, H., Rønning, B., Ringsby, T. H., Sæther, B-E., Husby, A., Sillanpää, M. J., & Jensen, H. (2021). Dispersal in a house sparrow metapopulation: An integrative case study of genetic assignment calibrated with ecological data and pedigree information. *Molecular Ecology*, *30*(19), 4740–4756. https://doi.org/10.1111/mec.16083

San-Jose, L. M., Bestion, E., Pellerin, F., Richard, M., Di Gesu, L., Salmona, J., Winandy, L., Legrand, D., Bonneaud, C., Guillaume, O., Calvez, O., Elmer, K. R., Yurchenko, A. A., Recknagel, H., Clobert, J., & Cote, J. (2023). Investigating the genetic basis of vertebrate dispersal combining RNA-seq, RAD-seq and quantitative genetics. *Molecular Ecology*, *32*(12), 3060–3075. https://doi.org/10.1111/mec.16916

Simpson, D., Rue, H., Riebler, A., Martins, T. G., & Sørbye, S. H. (2017). Penalising model component complexity: A principled, practical approach to constructing priors. *Statistical Science*, *32*(1), 1–28. https://doi.org/10.1214/16-STS576

Steinsland, I., Larsen, C. T., Roulin, A., & Jensen, H. (2014). Quantitative genetic modeling and inference in the presence of nonignorable missing data. *Evolution*, *68*(6), 1735–1747. https://doi.org/10.1111/evo.12380

Tietgen, L., Hagen, I. J., Kleven, O., Di Bernardi, C., Kvalnes, T., Norén, K., Hasselgren, M., Wallen, J. F., Angerbjörn, A., Landa, A., Eide, N. E., Flagstad, Ø., & Jensen, H. (2021). Fur colour in the Arctic fox: Genetic architecture and consequences for fitness. *Proceedings of the Royal Society B: Biological Sciences*, *288*(1959), 158–170. https://doi.org/10.1098/rspb.2021.1452

Tiffin, P., & Ross-Ibarra, J. (2014). Advances and limits of using population genetics to understand local adaptation. *Trends in Ecology & Evolution*, *29*(12), 673–680. https://doi.org/10.1016/j.tree.2014.10.004

Uffelmann, E., Huang, Q. Q., Munung, N. S., De Vries, J., Okada, Y., Martin, A. R., Martin, H. C., Lappalainen, T., & Posthuma, D. (2021). Genome-wide association studies. *Nature Reviews Methods Primers*, *1*(1), 59. https://doi.org/10.1038/s43586-021-00056-9

Van Dyck, H., & Baguette, M. (2005). Dispersal behaviour in fragmented landscapes: Routine or special movements? *Basic and Applied Ecology*, *6*(6), 535–545. https://doi.org/10.1016/j.baae.2005.03.005

Van Kuilenburg, A. B. P., Meinsma, R., Beke, E., Assmann, B., Ribes, A., Lorente, I., Busch, R., Mayatepek, E., Abeling, N. G. G. M., Van Cruchten, A., Stroomer, A. E. M., Van Lenthe, H., Zoetekouw, L., Kulik, W., Hoffman, G. F., Voit, T., Wevers, R. A., Rutsch, F., & Van Gennip, A. H. (2004). β-Ureidopropionase deficiency: An inborn error of pyrimidine degradation associated with neurological abnormalities. *Human Molecular Genetics*, *13*(22), 2793–2801. https://doi.org/10.1093/hmg/ddh303

Walls, S. S., Kenward, R. E., & Holloway, G. J. (2005). Weather to disperse? Evidence that climatic conditions influence vertebrate dispersal. *Journal of Animal Ecology*, *74*(1), 190–197.

Warren, W. C., Clayton, D. F., Ellegren, H., Arnold, A. P., Hillier, L. W., Künstner, A., Searle, S., White, S., Vilella, A. J., Fairley, S., Heger, A., Kong, L., Ponting, C. P., Jarvis, E. D., Mello, C. V., Minx, P., Lovell, P., Velho, T. A. F., Ferris, M., … Wilson, R. K. (2010). The genome of a songbird. *Nature*, *464*(7289), 757–762.

Waser, P. M., & Jones, W. T. (1989). Heritability of dispersal in banner-tailed kangaroo rats, *Dipodomys spectabilis*. *Animal Behaviour*, *37*, 987–991.

Whitlock, M., & Schluter, D. (2014). *The analysis of biological data* (2nd ed.). Roberts and Company Publishers.

Wilson, A. J., Réale, D., Clements, M. N., Morrissey, M. M., Postma, E., Walling, C. A., Kruuk, L. E. B., & Nussey, D. H. (2010). An ecologist's guide to the animal model. *Journal of Animal Ecology*, *79*(1), 13–26. https://doi.org/10.1111/j.1365-2656.2009.01639.x

Wolak, M. E. (2012). Nadiv: An R package to create relatedness matrices for estimating non-additive genetic variances in animal models. *Methods in Ecology and Evolution*, *3*(5), 792–796. https://doi.org/10.1111/j.2041-210X.2012.00213.x

Wolak, M. E., & Reid, J. M. (2017). Accounting for genetic differences among unknown parents in microevolutionary studies: How to include genetic groups in quantitative genetic animal models. *Journal of Animal Ecology*, *86*(1), 7–20. https://doi.org/10.1111/1365-2656.12597

Wu, N. C., & Seebacher, F. (2022). Physiology can predict animal activity, exploration, and dispersal. *Communications Biology*, *5*(1), 1–11. https://doi.org/10.1038/s42003-022-03055-y

Zera, A. J., & Brisson, J. A. (2012). Quantitative, physiological, and molecular genetics of dispersal/migration. In J. Clobert, M. Baguette, T. G. Benton, & J. M. Bullock (Eds.), *Dispersal ecology and evolution* (pp. 63–82). Oxford University Press.

SUPPORTING INFORMATION

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