The genetic basis of dispersal in a vertebrate metapopulation

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Abstract

Dispersal affects evolutionary processes by changing population sizes and their genetic composition, influencing the viability and persistence of populations. Investigating which mechanisms underlie variation in dispersal phenotypes and whether populations harbor adaptive potential for dispersal is crucial to understanding the eco-evolutionary dynamics of this important trait. Here, we investigate the genetic architecture of dispersal in an insular metapopulation of house sparrows. We use an extensive long-term individual-based ecological dataset and high-density single nucleotide polymorphism (SNP) genotypes for over 2500 individuals. We conducted a genome-wide association study (GWAS), finding a relationship between dispersal probability and a SNP located near genes known to regulate circadian rhythmic, 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 (Va), which composes 10% of the total overall variation in dispersal probability. Finally, we investigated differences in Va across populations occupying ecologically relevant habitat types (farm vs. non-farm) using a genetic-groups animal model. We found higher mean breeding value, Va, and heritability for the farm habitat, suggesting different adaptive potentials across habitats. Moreover, dispersal phenotypes may depend on genotype-by-environment interactions. 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 be ever more important with the increased fragmentation and loss of suitable habitats for many natural populations.

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, Baguette, Benton, & Bullock, 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 is needed to understand causes of variation in the dispersal phenotype and for predicting 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 behavior over 15 different studies (including both dispersal and migration) was found to be 0.46 (Dochtermann, Schwab, Anderson Berdal, Dalos, & Royauté, 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, Phillips, & Shine, 2014; McGaugh, Schwanz, Bowden, Gonzalez, & Janzen, 2010; Saastamoinen et al., 2018; Waser & Jones, 1989; Zera & Brisson, 2012).

Additive genetic variance ($\sigma_A{}^2$) and the proportion of the phenotypic variance explained by$\sigma_A{}^2$ (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 of 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, Niskanen, Saatoglu, Jensen, & Keller, 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, Jensen, & Muff, 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 behavior, causing adults with the dominant 'rover' allele to have longer dispersal distances (Edelsparre, Vesterberg, Lim, Anwari, & Fitzpatrick, 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, Saastamoinen, Marden, & Hanski, 2005; Niitepõald et al., 2009; Niitepõld, Mattila, Harrison, & Hanski, 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 used successful natal dispersal between islands as the phenotypic trait in order to investigate the heritable genetic basis of dispersal in an insular metapopulation of a small passerine bird, the house sparrow (*Passer domesticus*). Previous studies have shown spatial 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, and used a basic genetic groups animal model (basic GGAM) to estimate the $\sigma_A{}^2$ of dispersal probability. Subsequently, we used an extended GGAM to allow for different $\sigma_A{}^2$ 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 individuals in a long-term study of house sparrows on 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).

METHODS

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, 12deg30'E; Figure 1). These 8 islands are part of a larger metapopulation study system covering ca. 1600 km^2, where extensive field work 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 (breeding dispersal $< 0.3\%$; Altwegg, Ringsby, & Sæther, 2000; Jensen et al., 2013; Tufto, Ringsby, Dhondt, Adriaensen, & Matthysen, 2005) with an overall dispersal rate of 22.2% (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 live 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).

Genotyping and population assignment

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 level of genotyping errors (SNP call rate $< 95\%$; Mendelian error rate based on parental relationships $> 5\%$) or low minor allele frequency (MAF < 0.01) 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 in 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 (Niskanen et al., 2020). Both parents were known for 52.7% of the 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.

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 either from mark-recapture or genetic assignment information (Saatoglu et al., 2021). For the remaining 375 individuals that were successfully genotyped, we 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). These individuals, as well as individuals which had a natal island not among one of our eight main study islands (N = 98), and individuals which could only be assigned to a group of natal islands and not a specific one out of our 8 main study islands because the SNP genotyping of birds from the farm and non-farm islands had been initiated in different years (N = 41; see Saatoglu et al., 2021), were removed from the phenotype data set. Thus, phenotypic data on dispersal for a total of 2602 individuals were used in the animal model analyses and GWAS.

Heritable genetic variation in dispersal

We first estimated the variance in dispersal (i.e. natal dispersal between any of the study islands) that was attributable to additive genetic effects and several environmental effects using a basic genetic groups 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 of dispersal was similar in the two 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 allowed to differ for farm and non-farm island habitat types. The two genetic groups corresponded to the farm and non-farm island habitat types, where the genomes of individuals were proportionally assigned to their origin from either the farm or non-farm genetic group. The proportional assignment to farm or non-farm genetic group origin was based on the metapopulation level pedigree that included all 3116 successfully SNP-typed individuals and an additional 440 dummy individuals that were assigned as parents to identify known relationships among recruits (such as full- or half-sibling relationships) when one or both of the true genetic parents were not genotyped (Niskanen et al., 2020). More specifically, the assignment of these 3556 real or dummy individuals' genomes to the two genetic groups was done based on information on the assumed natal island habitat type of the phantom (i.e. unknown) parents of individuals in our metapopulation level pedigree. To obtain assumed natal island habitat type 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) was 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. In the metapopulation level pedigree, 592 real and 303 dummy individuals had either both parents (N = 646), their mother (N = 93) or their father (N = 156) missing. These unknown parents represent the pedigree's phantom parents, and were assigned to the same natal island habitat type as their (real or dummy) offspring. Finally, the proportional genetic group contribution (q_{ij}) values to the farm and non-farm genetic groups for each individual in the metapopulation level pedigree were calculated from the pedigree based on the phantom parents' assumed natal island habitat types using the "ggcontrib" function from the R package NADIV (Wolak, 2012).

Our basic GGAM partitioning variation in dispersal probability allowing differences in group-specific mean breeding values was defined using a binomial regression model with logistic link function and linear predictor for individual i given as

, (1)

where μ is the intercept, X_i is a vector indicating the fixed covariates of individual i and β is a vector of fixed effects. Individual sex was included as a fixed effect to account for differences between sexes in dispersal propensity (Saatoglu et al., 2021), and the proportional genetic contribution from the non-farm genetic group was included as a fixed effect (continuous covariate) to account for any mean differences in dispersal probability between the genetic groups. Both fixed effects variables were mean centered. The random effects include individual i 's natal island ($island_i$ ~ $N(0,\sigma_{\iota\sigma\lambda\alpha\nu\delta}{}^2)$) and hatch year ($hyear_i$ ~ $N(0,\sigma_{\psi\epsilon\alpha\rho}{}^2)$), and captured the variance in dispersal attributable to spatio-temporal environmental variation. Furthermore, the total additive genetic effect of individual$i$ is given as u_i , which is the weighted genetic group mean effect for group 2 (g_2; we defined group 2 as the non-farm genetic group) plus the breeding valuea_i , distributed as $\mathbf{a}^{[?]} = (a_1, \ldots, a_n)^{[?]} \quad N(\mathbf{0}, \sigma_A{}^2\mathbf{A})$ with additive genetic variance $\sigma_A{}^2$ and additive genetic relatedness matrix $\mathbf{A}$ that represents the relatedness among individuals (Kruuk, 2004). 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 is the difference in the non-farm group's mean total additive genetic effect compared to the farm group's mean total additive genetic effect. Note that, because our animal models were formulated as logistic regression

models, there is no residual variance component (de Villemereuil, Schielzeth, Nakagawa, & Morrissey, 2016).

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

, (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 group 2 (g_2; the non-farm genetic group) multiplied by the genetic group 2 proportion of individual i (q_{i2}), plus group-specific additive genetic values of group 1 (a_{i1}, the farm genetic group) and group 2 (a_{i2}, the non-farm genetic group). As in model (1) the genetic group mean effect for the farm group was set to 0 (i.e. g_1=0) so that the estimate for g_2 is the difference in the non-farm group's mean total additive genetic effect compared to the farm group's mean total additive genetic effect. However, the breeding valuea_i in model (1) is now split into two group-specific components a_{i1} anda_{i2}, with$\mathbf{a}_j^{[?]} = (a_{1j},\ldots,a_{nj})^{[?]}$ $\mathrm{N}(\mathbf{0}, \sigma_{A\theta}^2 \mathbf{A}_j)$ for both groups j = 1, 2, where$\sigma_{A\theta}^2$ 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} anda_{i2} as the partial breeding values, because they represent the contributions to the breeding value of individual i that are inherited from group 1 and 2, 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 for farm and non-farm genetic groups separately, from the variance component estimates by using the formula (showing the extended GGAM case)

, (3)

where the variances are defined as above, and residual variance was approximated by $\pi^2/3$ (Nakagawa & Schielzeth, 2010). The heritability estimate for dispersal from the basic GGAM (h^2) was obtained by usingσ_A^2 instead of$\sigma_{A\theta}^2$ 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$\sigma_{\iota\sigma\lambda\alpha\nu\delta}^2$ or$\sigma_{\psi\epsilon\alpha\rho}^2$ as the numerators, respectively (instead of σ_A^2 or$\sigma_{A\theta}^2$). Note that we for heritabilities and other proportions of phenotypic variance explained assume that the island and year variances are the same within the farm and non-farm habitats.

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

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). 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. 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 were investigated.

RESULTS

Heritable genetic variation of dispersal

In our house sparrow metapopulation data set there were 2118 resident adults and 484 adults that had dispersed between islands prior to recruitment. Among the dispersers, 399 individuals dispersed to an island of the same habitat type as their natal island, whereas 85 individuals either dispersed from a farm habitat island to a non-farm habitat island (N = 42) or in the opposite direction (N = 43). Proportions of dispersing recruits produced by adult sparrows was considerably higher on non-farm habitat islands than farm habitat islands (Figure 2; Supplementary 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 (Supplementary Figure S1).

The basic GGAM analysis showed 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%) and 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 habitat than the farm genetic group (Table 1).

Our extended GGAM analysis showed that 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 (Supplementary Materials, Figure S2), providing clear evidence that the additive genetic variances were different. Correspondingly, the heritability of dispersal was higher in the farm habitat than 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).

Genome wide association analyses

GWAS analyses on dispersal phenotype revealed a single genomic region that has been linked to regulation of diverse biological functions (Figure 4). The top SNP in this region was SNPa105044 which explained 3.8% of the variance in dispersal probability (Supplementary Table S2). SNPa105044 is located 16 Kbp downstream from Adenosine Receptor A2a (ADORA2A) and 22 Kbp upstream from Beta-Ureidopropionase (UPB1) on chromosome 15 in the house sparrow genome. 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, Guinzberg, Díaz-Cruz, & Pia, 2002). UPB1 encodes a highly conserved protein that catalyzes a late step in the nucleic acid pyrimidine degradation leading to biosynthesis of beta-alanine in animals (Matthews, Liao, Kvalnes-Krick, & Traut, 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, Saunders, Ball, Harris, & Sale, 2012; Milioni et al., 2019).

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, Ringsby, Jensen, & Sæther, 2012; Ranke et al., 2021; Saatoglu et al., 2021). We found that in this metapopulation, heritable genetic variation explained approximately 10% of the variation in individual dispersal probability. 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 was 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 necessary (Cayuela et al., 2018; Holyoak, Casagrandi, Nathan, Revilla, & Spiegel, 2008; Millon, Lambin, Devillard, & Schaub, 2019). Despite these challenges, the genetic basis of dispersal phenotype and dispersal-related traits had been researched on occasion even in vertebrates in the wild, using either parent-offspring regressions or animal models. Parameter estimates derived from animal models, which account for all kinds of genetic relatives in the models and are regarded as potentially less biased by for example non-genetic environment effects than parent-offspring regressions (Kruuk, Slate, & Wilson, 2008), tend to report more modest magnitudes of ${\sigma_A}^2$ andh^2 than parent-offspring regressions (Saastamoinen et al., 2018). Furthermore, animal model-based heritable genetic parameters of dispersal propensity for Aves class members of natural populations were reported as 0.024 – 7.608 and 0.36 – 0.95 for${\sigma_A}^2$ andh^2 , respectively (Supplementary Table S3). Moreover, the ${\sigma_A}^2$ andh^2 of dispersal were estimated to be 0.290 and 0.280, respectively, in an experimental cane toad study (*Bufo marinus/Rhinella marina* ; Phillips, Brown, & Shine, 2010), and in a semi-natural common lizards (*Zootoca vivipara*) study as 1.148 and 0.170, respectively (San-Jose et al., 2023). Hence, heritability 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 approximately 12% of the phenotypic variance in dispersal explained by heritable genetic variation 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 (Supplementary 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 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 preparation). Consequently, dispersal rates are expected to increase across generations in our metapopulation.

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 a single region on chromosome 15 that was linked with dispersal trait in the house sparrow metapopulation and it has been found that 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 the 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 (*Zootoca vivipara*) 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 these species (San-Jose et al., 2023). Moreover, clock-linked genes may also influence migratory timing in the American kestrel (*Falco sparverius* ; Bossu, Heath, Kaltenecker, Helm, & Ruegg, 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. However, it seems clear 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.

Dispersal in our house sparrow metapopulation occurs during the fledged juvenile phase, in the autumn before the juveniles' first winter (Parn, Jensen, Ringsby, & Saether, 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 springs were warmer, breeding started early, and when total population sizes at the end of the breeding season were higher (Parn 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, Banks, Beck, & Heinsohn, 2022; Maag, Cozzi, Clutton-Brock, & Ozgul, 2018; Massot, Clobert, Lorenzon, & Rossi, 2002; Matthysen, 2005; McCaslin, Caughlin, & Heath, 2020; Messier, Garant, Bergeron, & Reale, 2012; Saastamoinen et al., 2018; Walls, Kenward, & Holloway, 2005; Wu & Seebacher, 2022). Interestingly, the relationships between dispersal and environmental conditions in our house sparrow metapopulation mentioned above 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 (Parn 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 that show lower estimated mean breeding values and lower additive genetic variances for dispersal in the non-farm habitat than the farm habitat (Table 1), suggest that when individuals make their dispersal decisions, environmental components are more influential than heritable genetic effects in the non-farm habitat.

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 (Supplementary 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 Supplementary 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), 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.

Here 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 and 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 show 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 is likely to be of increasing importance.

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TABLES AND FIGURES

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. The GGAM had 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.

	Posterior estimate of fixed or random effect (variance component)	Posterior estimate of fixed or random effect (variance component)	Posterior estimate of fixed or random effect (variance component)	Proportion variation explained by random effect (variance component)	Proportion variation explained by random effect (variance component)
Parameter	Basic GGAM	Extended GGAM	Basic GGAM	Basic GGAM	Extended GGAM
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)	-	-	-
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)	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)	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)	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)

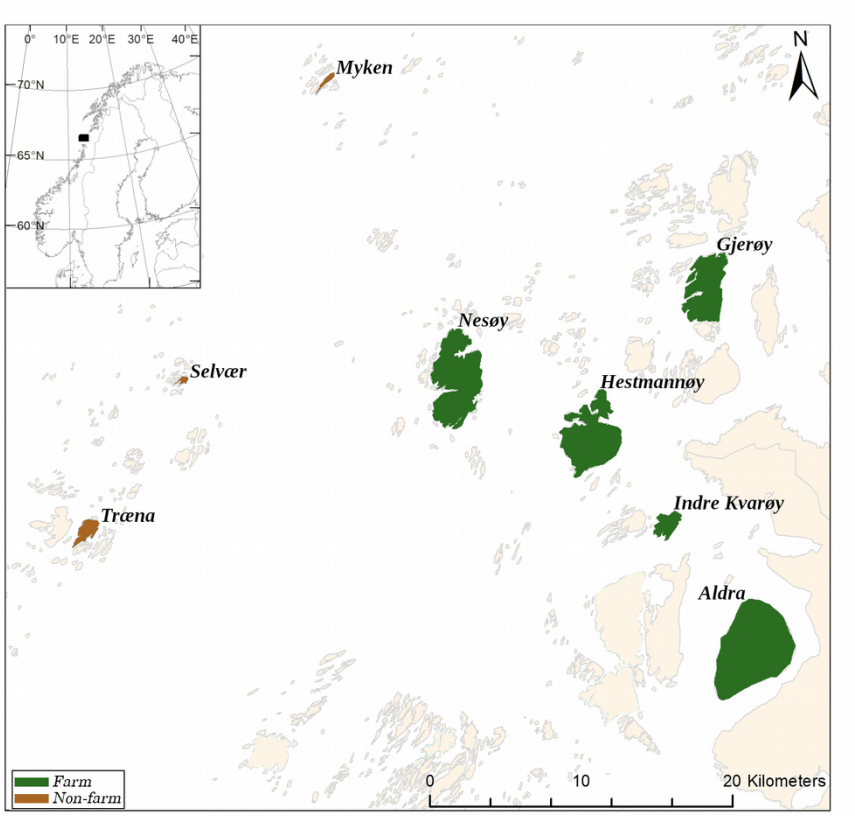

Figure 1 . Map showing the house sparrow metapopulation study system in northern Norway, with different colors for farm (green) and non-farm (brown) habitat islands included in the present study.

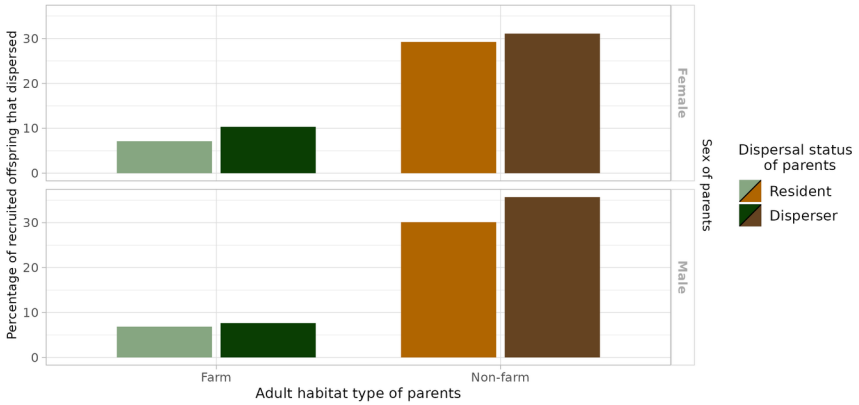

Figure 2. The proportion (in %) of recruited offspring produced by different types of female (top) and male (bottom) house sparrow parents in an insular metapopulation. Parents were classified as either resident (i.e. they were adult on the same island they were born; light-shaded bars), or dispersers (i.e. they were adult on a different island than the one they were born on; dark-shaded bars). 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 colored bars) or one of the islands of the non-farm (brown colored bars) habitat type.

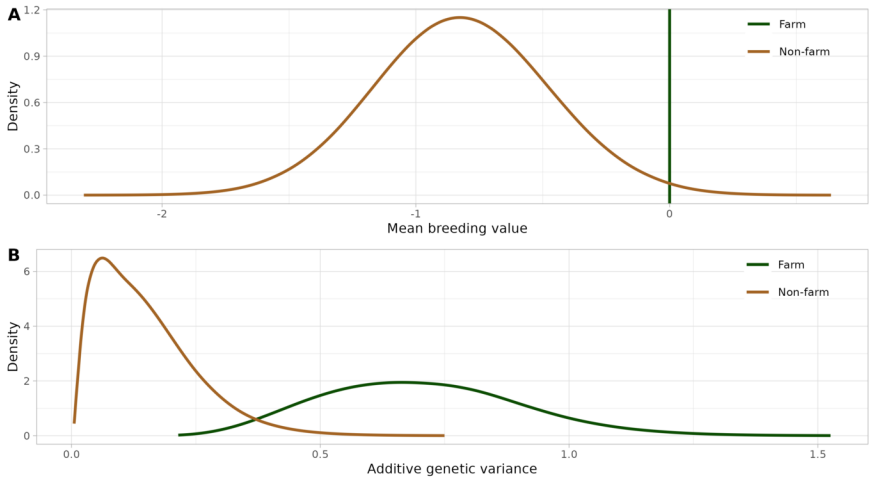

Figure 3. Posterior distributions of mean breeding values (A) and additive genetic variances (B) from the extended genetic groups animal model. Panel (A) shows the estimated posterior distribution of the mean breeding value for the non-farm genetic group in brown. Because the farm genetic group was set 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 distribution of additive genetic variance for the farm and non-farm genetic groups are shown in green and brown, respectively.

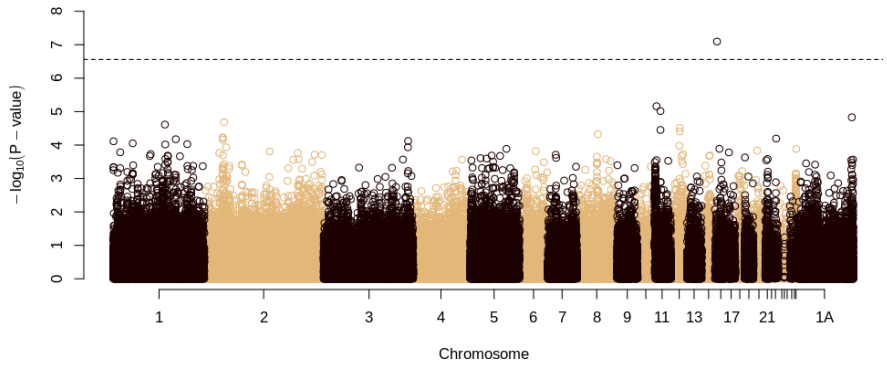

Figure 4. Results from the GWAS on dispersal phenotype for 2602 house sparrows, showing the negative logarithm of the p-value for 181,401 autosomal markers against their chromosomal position in the house sparrow genome. Markers on chromosome 16 were not on the genotyping array, and those markers located 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 a FWER of 0.05. This marker is 16 Kbp from ADORA2A in the house sparrow genome, and 22 Kbp from UPB1.

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DATA ACCESSIBILITY

Data associated with this study will be made available in the Dryad Digital Repository.

AUTHOR CONTRIBUTIONS

The fieldwork was carried out by HJ. DS, SLL and AH. AKN and HJ developed the pedigree. DS carried out genetic assignment. DS and SLL performed the statistical analyses and interpreted the results with input from AKN, DG, SM and HJ. DS drafted the initial version of the manuscript with input from SLL, DG, AKN, SM and HJ. All authors contributed to later versions of the manuscript, and all authors gave final approval for publication.