Evolutionary potential of natal dispersal in multiple blue tit populations while accounting for spatial autocorrelation among relatives

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Abstract

Natal dispersal is a key driver of ecological and evolutionary dynamics in populations. However, our understanding of genetic mechanisms underlying inter-individual variation in dispersal behaviour is still incomplete, despite their crucial role in the evolutionary potential and persistence of wild populations. Here, we investigate the genetic basis of natal dispersal distance in four blue tit (*Cyanistes caeruleus*) populations monitored for 26 to 48 years, using multi-generational social pedigrees and Restriction-site Association DNA sequencing genomic data. We combine quantitative genetic analyses (2,718 individuals), a permutation-based approach and genome-wide associations analyses (139 individuals). We find some evidence for additive genetic variance in natal dispersal distance, with heritability varying from 0.03 [<0.001-0.11] to 0.13 [<0.001-0.22] and evolvability ranging from 0.09 to 0.16. Permutation tests and power analyses reveal that spatial arrangement of nest boxes can generate spurious estimates of additive genetic variance in three of our four focal populations. We identified four single-nucleotide polymorphisms associated with natal dispersal distance, related with central nervous, metabolism and endocrine systems, which are key determinants of movement abilities. Overall, our study suggests that natal dispersal exhibits evolutionary potential, although this signal is partially obscured by spatial autocorrelation, potentially constraining population persistence under increasing habitat loss and fragmentation.

Keywords: Dispersal evolution - additive genetic variance - heritability - genotype – environment correlation - genome-wide association study - *Cyanistes caeruleus*.

Introduction

Natal dispersal, *i.e.* the movement of individuals from their birthplace to a new location where they reproduce, is a key driver of ecological and evolutionary dynamics for populations [1]. Natal dispersal shapes genetic diversity, influences local adaptation by facilitating or constraining gene flow, contributes to the persistence of declining populations, and plays a crucial role in the colonization and extinction of habitat patches [2–4]. Individuals can track suitable habitats (*e.g.* high resource abundance, few competitors, numerous mating partners, preferred temperature) through natal dispersal processes enabling them to cope with rapidly changing environments [5–7]. Thus, understanding whether dispersal itself can be modified in response to environmental changes is critical to determining whether individuals can adjust to such changes by tracking suitable habitats. For instance, the adjustment of dispersal may allow some populations to better track their climatic niche and, in the present global warming, help rescue populations [8,9].

Like many life-history traits, natal dispersal depends on interactions between the organism and its environment. Several ecological factors (*e.g.* population density, resource availability and habitat fragmentation) [10–12], and internal state factors (*e.g.* exploratory activity, body mass, immunity, hormone levels) [13–15], have been identified as key drivers of dispersal behaviour, generating among-individual variance (within or across populations) in natal dispersal. Consequently, dispersal is often considered as a highly plastic trait with strong environmental dependency and complex multi-determinisms [16–18]. However, among-individual variance in dispersal could be partly explained by genetic variance like other behavioural traits [19–21], and microevolutionary changes in natal dispersal could play a

crucial role in long-term persistence and adaptation of populations to strongly altered environments [22,23]. Unravelling the genetic basis of dispersal is therefore essential for predicting its evolutionary dynamics and deciphering adaptive responses to environmental changes through shifts in animal movements.

Despite its ecological importance, genetic variance in dispersal remains poorly documented. In 2018, Saastamoinen and colleagues reviewed 84 studies over 30 years that had reported heritability (average 0.35) for dispersal-related traits such as locomotion speed, wing shape or flight capacity [20], while a broader analysis of behavioural movement found higher mean heritability (0.46, [95% CI: 0.33 – 0.56]) [21]. Notably, fewer than 20% of studies in Saastamoinen et al. (2018) focused on traits directly linked to dispersal, such as dispersal distance or propensity to disperse outside a native habitat. In addition, studies investigating the genetic basis of dispersal in natural populations have traditionally relied on parent – offspring regressions (69% of studies in Saastamoinen et al., 2018). However, this approach tends to overestimate heritability compared to more sophisticated linear mixed quantitative genetic models, such as the animal model [24,25], which account for non-genetic resemblance among relatives. To date and to our knowledge, only eleven studies have explored the genetic components of dispersal in vertebrates using animal models (Table S1). Notably, animal models generally provide more accurate, albeit lower, heritability estimates than traditional regression methods [24,26]. For example, Doligez et al. (2012) reported a 17 – 30% reduction in the estimated heritability of dispersal probability in collared flycatchers (*Ficedula albicollis*) when comparing the animal model to parent – offspring regression [27], while Charmantier et al. (2011) found reductions of up to 58% in wandering

albatrosses (*Diomedea exulans*) [28]. These findings highlight that, in natural populations, non-genetic factors are often shared among relatives, potentially confounding genetic estimates and biasing heritability inferred from simple regression approaches. Disentangling genetic and environmental contributions to natal dispersal in wild populations remains challenging, as it requires extensive individual-level data and robust statistical methods to avoid misestimating quantitative genetic parameters, particularly in the presence of shared environmental effects among relatives [29–31].

Natal dispersal can also generate spatial structure in phenotypic traits that may confound estimates of genetic variation [30]. Limited dispersal increases the likelihood that relatives experience similar environments, including breeding locations and maternal conditions, potentially inflating phenotypic resemblance among relatives and leading shared environmental effects to be attributed to genetic similarity [30,32,33]. This is particularly relevant when dispersal itself is influenced by the spatial configuration of breeding sites [34–36]. For example, siblings born in peripheral nest boxes may have different dispersal opportunities from those born in centrally located boxes, causing relatives to resemble one another in dispersal distance because they share similar spatial constraints rather than genes. Such spatial autocorrelation can therefore generate spurious additive genetic variance and inflate estimates of heritability and evolvability. Although this issue was first highlighted by Van Noordwijk (1984), to our knowledge only two studies has explicitly accounted for spatial configuration when estimating quantitative genetic parameters of dispersal [32,37].

The evolutionary potential of a quantitative trait also depends on its underlying genetic architecture, including the number and effect sizes of contributing loci [38,39]. Traits controlled by relatively few loci of large effect are generally expected to respond more

rapidly to selection than highly polygenic traits, although rapid evolution of polygenic traits is also well documented [40–45]. Natal dispersal is expected to have a polygenic basis, as is common for quantitative traits [46,47], but direct evidence remains scarce, particularly in vertebrates [19]. Several genes have nevertheless been associated with dispersal-related phenotypes across taxa. In Drosophila melanogaster, variation in the foraging gene influences movement distance [48]. In vertebrates, NPAS2 is associated with dispersal timing and distance in common buzzards (*Buteo buteo*) [49], CPNE7 differs in expression between dispersing and non-dispersing yellow-bellied marmots (*Marmota flaviventer*) [50], and BCAT2 and KLF15 are overexpressed in dispersing cane toads (*Rhinella marina*) at the range edge [51].

Only two studies, to our knowledge, have jointly quantified heritability and genomic associations for dispersal in natural vertebrate populations. Saatoglu et al. [33] estimated a heritability of 0.09 [95% CI: 0.07–0.12] for dispersal propensity and found that approximately 3.8% of its variance was associated with ADORA2A, a gene involved in metabolic processes. San-José et al. [52] estimated a heritability of 0.17 [<0.01–0.29] for dispersal propensity in the common lizard (*Zootoca vivipara*) and identified a significant SNP near CA10, a gene involved in nervous-system function. These findings provide initial evidence for a genetic basis of dispersal but remain insufficient to determine whether dispersal is generally controlled by a small number of loci or by many loci of small effect. The genomic basis of dispersal in natural vertebrate populations therefore remains poorly understood [53].

Here, we investigated the genetic basis of natal dispersal by combining quantitative genetic, permutation-based and genomic approaches in four blue tit (*Cyanistes caeruleus*) populations monitored over several decades in southern France. These populations occupy

heterogeneous landscapes and differ in forest composition, providing an opportunity to examine genetic variation in dispersal across contrasting environments [54]. The spatial arrangement of nest boxes also imposes constraints on measured dispersal distance, because individuals dispersing outside the monitored areas cannot be detected. Previous work has documented weak but significant genetic differentiation between deciduous and evergreen populations [55] and heterogeneous selection pressures among populations [56], providing a context in which population-specific genetic variation and evolutionary potential may arise.

We first estimated additive genetic and environmental contributions to natal dispersal distance using animal models based on long-term capture–mark–recapture data and social pedigrees spanning 8–19 generations (26–48 years). Second, we used permutation-based simulations to test whether the spatial configuration of nest boxes could generate spurious additive genetic variance through non-genetic resemblance among relatives. Finally, we conducted a genome-wide association study (GWAS) to identify loci associated with dispersal distance, characterize their biological functions and quantify their contribution to phenotypic variation. We predicted that natal dispersal would show low to moderate heritability, consistent with estimates from other tit species [32,57], and a polygenic genetic architecture. We further predicted that spatial autocorrelation among relatives could inflate estimates of additive genetic variance if not explicitly accounted for.

Materials & Methods

1 *Study populations and field monitoring*

This study was conducted in four populations sedentary breeding in artificial nest boxes, located on the island of Corsica and in southern mainland France. The populations D-

Rouvière (Montarnaud, France, Lat: 43.66; Long: 8.75) and D-Muro (Muro, Corsica, 42.55; 8.92) are dominated by deciduous oak (*Quercus pubescens*) whereas E-Muro (Feliceto, Corsica, 42.59; 8.96) and E-Pirio (Manso, Corsica, 42.38; 8.75) are dominated by evergreen oak (*Quercus ilex*) (Figure S1). The sites have been monitored since 1976, 1991, 1993, and 1998, corresponding to 48, 33, 31, and 26 years of monitoring, respectively, and include 182, 284, 113, and 75 nest boxes used in the dispersal analyses for E-Pirio, D-Rouvière, D-Muro, and E-Muro, respectively. Blue tit populations from mainland and Corsica are genetically differentiated and represent two distinct subspecies (*C. c. caeruleus* for mainland and *C. c. ogliastrae* for Corsica) [55].

During the breeding season (March – July), from the onset of monitoring at each site until 2023, nestlings were individually marked with unique metal rings at 15 days after hatching. Adults were (re)captured at the nest boxes during the nestlings provisioning, sexed based on the presence of a brood patch and belly plumage pattern, thenidentified or ringed with a unique metal ring provided by the Centre de Recherche sur la Biologie des Populations d'Oiseaux (CRBPO, France). Local recruitment rate (*i.e.* proportion of ringed fledglings born in a site and subsequently recruited as breeders in nest boxes of the same site) ranged between 4.3 and 6.9 % among populations. Hatching date, nest box of origin and parental identities were known for all local recruits. Since monitoring began, 9,005 breeding events have been recorded in nest boxes across the four sites, yielding 53,753 ringed individuals, including 2,718 local recruits.

2 *Estimating natal dispersal distances*

166 To estimate natal dispersal distances, we considered locally recruited blue tits for which sex,
167 natal year and nest box were known. Natal dispersal distance was calculated using the
168 Haversine formula, which accounts for Earth's curvature, between the natal nest box and the
169 nest box of first reproduction [58]. Since breeding events were only monitored in nest boxes,
170 natal dispersal distance opportunities within a site were inherently constrained by spatial
171 configuration of nest boxes [37]. Each site has a different spatial configuration with a distinct
172 distribution of natal dispersal distance possibilities for each nest box (Figure S1). For instance,
173 possible dispersal distances from a centrally located nest box differed from that of a
174 peripheral one (Figure S3). Based on reproductive monitoring data collected through 2023,
175 the median breeding dispersal distance (i.e. the distance between two successive breeding
176 events detected in nest boxes) was 41 m for males and 58 m for females, indicating high
177 breeding-site fidelity in this species. Our natal dispersal distance estimates included recruits
178 for which breeding during the first year was not detected, either because they did not
179 reproduce during their first years or because their first breeding occurred outside the study
180 sites (27.1% of all natal dispersal events in our dataset). Including individuals with
181 undetected first breeding did not impact mean natal dispersal distance (Wilcoxon-Mann-
182 Whitney = 2648158 ; p = 0.34).

183 3 *Social pedigree building*

184 Social pedigrees were constructed from the complete set of breeding records, assigning each
185 offspring to its mother and father, identified at capture. We built one pedigree per site,
186 because of the very low number of dispersal events among sites and the genetic structure
187 revealed by genomic analyses [55]. Unknown parents of a given brood were assigned dummy

identities to preserve sibship information. Using the "*Pedantics*" R-package [59], we defined population founders as the individuals with unknown parents, including the first breeders ringed at the beginning of the survey as well as immigrants. Social pedigrees were pruned to keep only individuals informative for quantitative genetic analyses of natal dispersal distances (i.e., individuals with known dispersal phenotype and/ or known relatives with or without phenotypes; see Table S2 for pedigree descriptive statistics). In our populations, extra-pair paternityoccurs at relative high rate, representing 14-25% of nestlings across populations [60], which is expected to underestimate heritability by an average of 0-17% [61].

4 *Animal model and quantitative genetic parameters estimations*

We used population-specific animal models implemented in the *MCMCglmm* R package [62] to partition phenotypic variance in natal dispersal distance into additive genetic, maternal, nest, year and residual components. The models included additive genetic effects derived from the pedigree (V_A), birth year (V_Y), mother identity (V_M), hatching nest (V_N) and residual variance (V_R). To account for variation in the spatial configuration of nest boxes, we included as a fixed effect the median distance from each natal nest box to all nest boxes available for future reproduction. Sex and age at first recapture were also included as fixed effects to account for potential differences in measured dispersal distance associated with delayed first capture.

Because breeding pairs showed spatial fidelity, mother and hatching-nest identities were partially confounded. We therefore assessed this potential confounding by quantifying the proportion of offspring sharing the same mother–nest association and by comparing the full model with models excluding either maternal or nest effects. Although 31–45% of individuals

shared the same mother–nest association within a year (Figure S2), estimates of variance components and heritability were largely robust to the exclusion of either effect (see supplementary materials, Text S1, Figure S2 and Table S3).

The "*Full*" animal model was: $Y_i = \mu + X_i b + a_i + year_i + mother_i + nest_i + e_i$ (Eq.1)

where $_{Yi}$ is the natal dispersal distance of individual i, μ is the intercept, X_i b represents the fixed effects (sex, age at first recapture and median potential dispersal distance), a_i is the additive genetic effect, and $year_i$, $mother_i$, $nest_i$ and e_i are the birth-year, maternal, hatching-nest and residual effects, respectively.

We used inverse-gamma priors for variance components (V = 1, ν = 1, Vα = 1000 [63]); results were qualitatively similar under less informative priors (V = 1, ν = 0.002). Natal dispersal distance was log-transformed as log(x + 10) for E-Muro, E-Pirio and D-Muro to meet normality assumptions; D-Rouvière was analysed without transformation. Models assumed a Gaussian distribution and were run for 1,000,000 iterations, with sampling every 100 iterations and a burn-in of 15,000 iterations. Convergence was assessed from trace and density plots, and by checking autocorrelation and effective sample sizes. Autocorrelation was <0.05 and effective sample sizes exceeded 1,000 for all parameters.

For the log-transformed populations, variance components were estimated on the latent scale and back-transformed to the observed scale using the *QGglmm* R package [64]. Narrow-sense heritability was calculated as $h^2 = V_A/V_P$, where $V_P = V_A + V_R + V_M + V_N + V_Y$. Heritability was estimated on both latent and observed scales for the log-transformed populations and on the observed scale for D-Rouvière. The relative contributions of the other variance components were calculated as their ratio to V_P.

We estimated evolvability (I_A), defined as the additive genetic variance standardized by the squared mean of the trait [65]. For D-Rouvière, I_A was calculated as V_A/μ^2, whereas for the log-transformed populations we used additive genetic variance as an approximation of I_A on the raw scale [66,67]. Quantitative genetic parameters are reported as posterior medians, means and 95% credible intervals. To assess the contribution of pedigree information, we compared models with and without the additive genetic effect using the Deviance Information Criterion (DIC), retaining the model with the lower DIC. We further ensured the ability of our sampling design and pedigree structure to estimate genetic variance using simulations following Pick et al. (2023) [68] (see supplementary materials, Text S2, Tables S4a,b).

5 *Simulations of spatial-configuration bias*

We used a permutation approach following Korsten et al. (2013) [32] to test whether the spatial configuration of nest boxes could generate spurious additive genetic variance in natal dispersal distance. For each population, we randomly reassigned the natal nest box and parental pair of each locally recruited individual to those of another individual born in the same year. We then recalculated dispersal distances and pedigrees from these reassigned data. This procedure preserved the spatial configuration of nest boxes while removing the correspondence between dispersal distance and pedigree, such that heritability was expected to approach zero.

We generated 1,000 permuted datasets and fitted the same animal model to each. The resulting estimates were used to construct null distributions for quantitative genetic parameters. For each observed dataset, we calculated a p-value as the proportion of

estimates in the corresponding null distribution that exceeded the median estimate obtained from the observed data [68].

6 *Genomic approaches*

6.1 *DNA extraction and SNP filtering*

Fledglings were generally not blood-sampled; blood was collected from breeding individuals at their first recapture. DNA was extracted using Qiagen DNeasy Blood and Tissue Kits and sent to the Montpellier GenomiX platform (MGX) for paired-end RAD sequencing following Caizergues et al. (2022) [69]. We sequenced 459 individuals from the Corsican populations (150 E-Pirio, 180 D-Muro and 141 E-Muro). Paired-end sequencing (2 × 150 bp) generated 5,435,974,084 reads, with a mean depth of 19.8× per individual before filtering.

Reads were mapped to the great tit (*Parus major*) reference genome [70] using *BWA-MEM* 0.7.19 [71], with 97% mapping properly, following Perrier et al. (2020). SNPs were called using the gstacks and populations modules of *STACKS* v2.64 [72]. We used default settings except for --min_mapq=20 and --rm-pcr-duplicates in gstacks, and --r=0.9, --max_obs_het=0.65 and --p=2 in populations. This yielded 243,762 SNPs. The analyses were implemented in a Snakemake v7.25 pipeline [73] generated and run using the MBB-workflow framework [74].

6.2 *Genome-wide association study*

We tested for associations between natal dispersal distance and genome-wide SNP variation using *GAPIT* v3.1.0 [75]. Dispersal distance was rank-based inverse-normal transformed using the *RNOmni* package [76]. After applying *GAPIT*'s minor allele frequency (MAF) threshold of

0.05, 243,669 SNPs remained for association testing in 137 individuals with known natal dispersal distances. We used the Bayesian-information and linkage-disequilibrium iteratively nested keyway (BLINK) model, a multi-locus method implemented in *GAPIT* [77]. Model fit was assessed using QQ plots, and we retained the BLINK results because they showed the best fit.

We corrected for multiple testing using both the Bonferroni threshold ($\alpha = 0.05/243{,}669 = 2.05 \times 10^{-7}$) and the less conservative Benjamini–Hochberg false discovery rate (FDR) procedure [78] (Table S8). To identify candidate genes, we considered genes within 5 kb of significant SNPs, based on evidence that linkage disequilibrium in blue tits declines to near-baseline levels within 10 kb [79]. Gene functions were annotated using NCBI/RefSeq orthologues and Gene Ontology terms.

We estimated the proportion of variance in dispersal distance explained by significant SNPs by fitting the same animal model described previously, with each significant SNP coded as 0, 1 or 2 (homozygous alternative, heterozygous and homozygous reference, respectively) as the only numeric fixed effect. This analysis included the 137 sequenced individuals with known natal dispersal distances.

Finally, we assessed the statistical power of the GWAS using the GAPIT.Power.compare function. Phenotypes were simulated under four scenarios combining 20 or 100 quantitative trait nucleotides (QTNs) with narrow-sense heritabilities (h^2) of 0.1 or 0.2. Each scenario was replicated 100 times, and power was defined as the proportion of causal SNPs detected at the corresponding significance threshold (Figure S4).

The data and computer code for replicating the analyses are freely available via Zenodo [80].

Results

1 *Natal dispersal distance in four blue tit populations*

Among 48,987 nestlings ringed over four monitored sites between 1976 and 2023, 2,718 were phenotyped for natal dispersal. In all populations, females displayed significantly greater natal dispersal distances than males (E-Muro: Wilcoxon-Mann-Whitney , W = 15396, p <0,001 ; E-Pirio: W = 43035, p = 0.006 ; D-Muro: W = 54600, p < 0.001 ; D-Rouvière: W = 196590, p = 0.027) ; see Table S5 and Figure S5 for sex-specific distributions). Natal dispersal distance also differed significantly between populations (Table S5, Kruskal-Wallis: $\chi^2_3 = 579.6$, $p < 0.001$).

2 *Heritability and evolvability of blue tit natal dispersal distance*

Heritability of natal dispersal distance was low to moderate across populations, ranging from 3% [95% CI: <0.1–11%] in E-Muro to 13% [<0.1–22%] in E-Pirio (Figure 1; Figure 2a; Table S6; Table S7). Evolvability showed a similar pattern, ranging from 9% [<0.1–37%] in E-Muro to 16% [2–32%] in E-Pirio (Figure 2b; Table S6). Model comparison based on the Deviance Information Criterion supported the inclusion of additive genetic variance in all populations (Table S9).

Permutation tests revealed, however, that the apparent genetic signal could be partly attributable to the spatial configuration of nest boxes. In E-Muro, D-Pirio and D-Rouvière, observed heritability and evolvability did not differ from the null distributions generated by permutations (p = 0.164, 0.086 and 0.11, respectively; Figure 3). In contrast, E-Pirio showed strong evidence for a genetic component: observed heritability and evolvability exceeded

318 those obtained under the null model in >99% of permutations (p = 0.003; Figure 3).
319 Additional simulations showed that estimates of small additive genetic variances (h^2 < 10%)
320 were substantially biased and imprecise, making weak genetic signals difficult to distinguish
321 from the null expectation of VA = 0 across the four populations (Text S2, Tables S4a,b).

322 Most variation in natal dispersal distance remained unexplained by the model. Year, maternal
323 and hatching-nest effects together accounted for less than 10% of phenotypic variance in all
324 populations. Year effects were particularly small, explaining at most 1.7% in D-Rouvière,
325 whereas maternal effects accounted for up to 6.7% in D-Rouvière and hatching-nest effects
326 up to 5.3% in E-Muro (Figure 1; Table S6; Table S7).

327 Fixed effects accounted for 4% [95% CI: 2–7%] and 6% [3–9%] of phenotypic variance in D-
328 Muro and D-Rouvière, respectively (Figure 1; Table S6). Sex was the main contributor in E-
329 Muro and D-Muro, explaining 5% [2–8%] and 4% [2–7%] of the phenotypic variance,
330 respectively.

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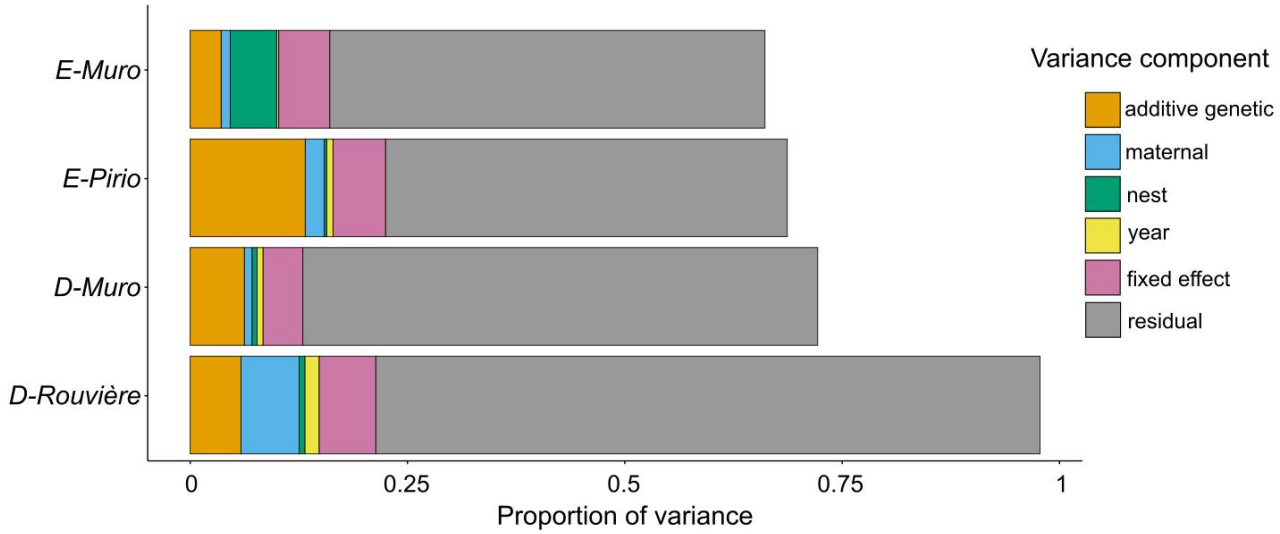

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Figure 1 : Decomposition of natal dispersal distance variation into genetic and environmental components. For each site (E-Muro, E-Pirio, D-Muro, D-Rouvière), results were reported from the "*Full*" model (presented in Table S6) on the observed scale. Bars represent the proportion of dispersal variance explained by each component. The proportion value of each variance component was calculated from the median of the posterior distribution. The variance components were back-transformed to the observed scale for log-transformed models (i.e.E-Muro, E-Pirio and D-Muro). Note that due to backtranformations variances do not sum to one anymore [85].

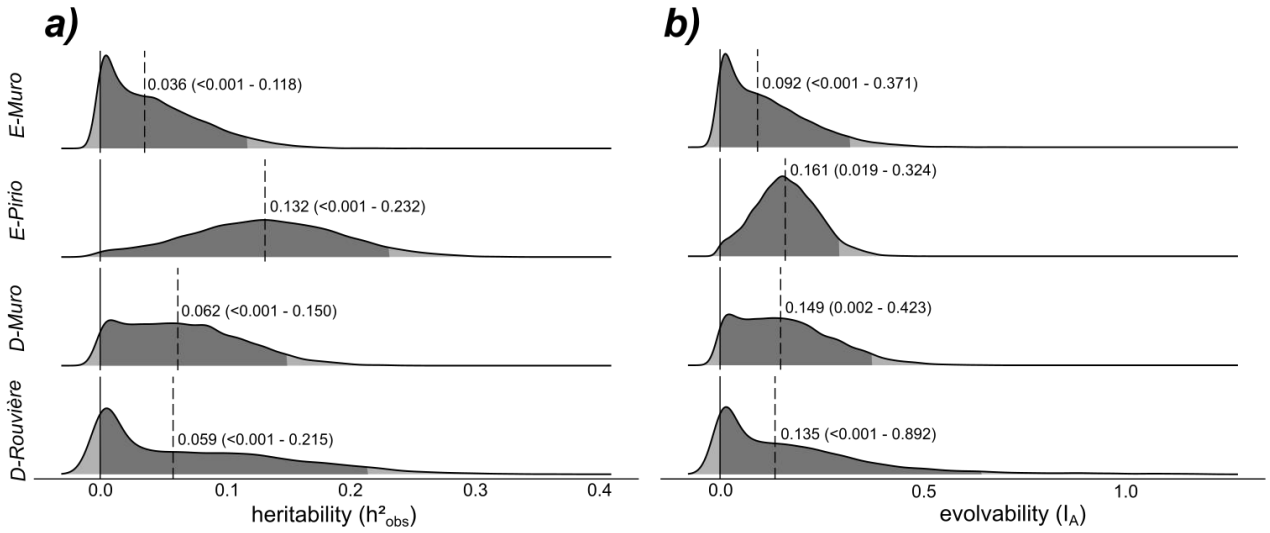

Figure 2: Posterior distributions for the heritability (h^2_{obs}, on observed scale, panel a) and evolvability (I_A, panel b) of blue tit natal dispersal distance. For each population (E-muro, E-Pirio, D-Muro, D-Rouviere), the posterior distributions were obtained from 9850 iterations (see detailed results in Table S6). 95% CI are in dark blue. Vertical solid lines correspond to zero and the dashed lines correspond to the median value for each posterior distribution.

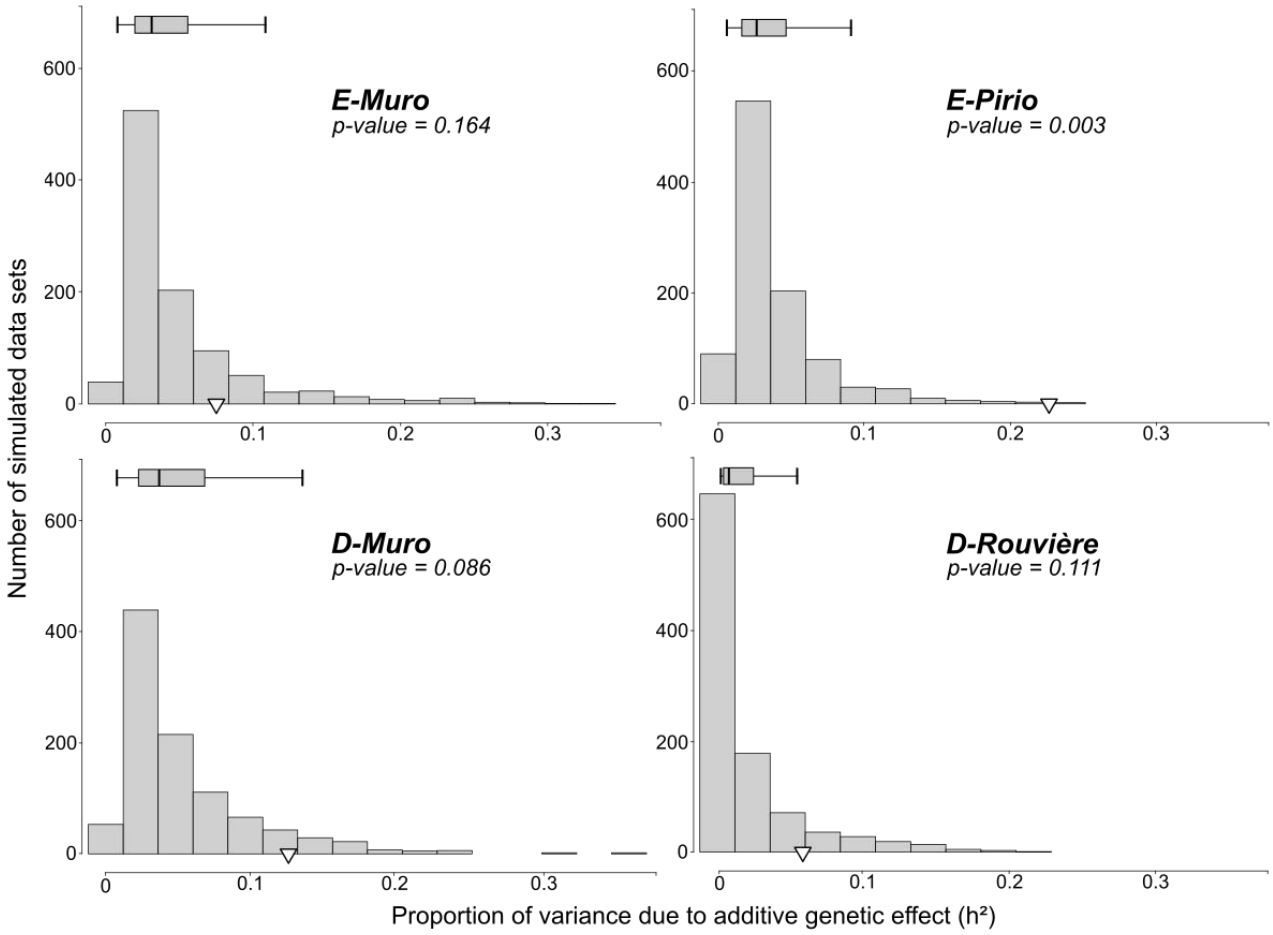

Figure 3: Distribution of proportion of natal dispersal distance variance attributable to additive genetic variance estimated from permuted dataset (*i.e.* null model). Grey bars represent the frequency distribution of heritabilities estimated from permutation models fitted in each population. Boxes indicate the median, 25th and 75th percentiles, whiskers indicate the 10th and 90th percentiles. Triangles indicate the heritability of natal dispersal distance estimated from the animal model described in the "*Full*" model equation. Note that heritability estimates from the observed and permuted datasets were reported on the latent scale for E-Muro, E-Pirio, and D-Muro. Consequently, these estimates differ from those presented in the main text, which were reported on the observed scale. This difference does not affect the interpretation of the results. P-values were calculated by the percentage of permuted data sets with higher heritability than observed heritability. A p-value below 0.05 indicates that the observed heritability estimate is unlikely to have arisen solely by chance. Note that results were qualitatively similar with additive genetic variance and evolvability estimates.

359 *3. A few SNPS associated with natal dispersal in island populations*

360 The genome-wide association analysis of natal dispersal distance in Corsican blue tit
361 populations identified four SNPs exceeding the genome-wide Bonferroni significance
362 threshold. These SNPs were located on chromosomes 4 ($p = 3.58 \times 10^{-14}$), 3 ($p = 4.53 \times 10^{-8}$),
363 18 ($p = 5.58 \times 10^{-8}$), and 12 ($p = 8.94 \times 10^{-8}$) (Figures 4a, b, Table S8). One additional SNP
364 located on chromosome 1 ($p = 1.01 \times 10^{-6}$) was significantly associated with variation in natal
365 dispersal distance based on its H&B p-value but did not reach the Bonferroni threshold
366 (Figures 4a, b, Table S8). The SNP on chromosome 4 (1,579,274 bp) was located within
367 *LOC107203094*, the vertebrate ortholog of *MTHFD2L*, which encodes a mitochondrial
368 enzyme involved in folate-dependent one-carbon metabolism and purine biosynthesis. The
369 SNP on chromosome 3 (1,335,245 bp) occurred within *SPTBN1,* which encodes β II-spectrin,
370 a cytoskeletal protein linking the plasma membrane to the actin cytoskeleton and involved in
371 cellular organization and synaptic function. The SNP on chromosome 12 (1,421,770 bp),
372 negatively associated with natal dispersal distance, was located within the *PHF2* encodes a
373 histone demethylase and involved in transcriptional activation by RNA polymerase II. Finally,
374 the SNP on chromosome 18 (6,766,807 bp) was located within the *SAP30BP* which encodes a
375 protein interacting with transcriptional repression complex, regulating gene expression
376 through chromatin remodelling. Moreover, the SNP on the first chromosome (27,602,551 bp)
377 was found near to the *ECRG4* gene at more than 10 kbp, which is involved in neuropeptide
378 hormone activity, acting in the central nervous system (Table S8).

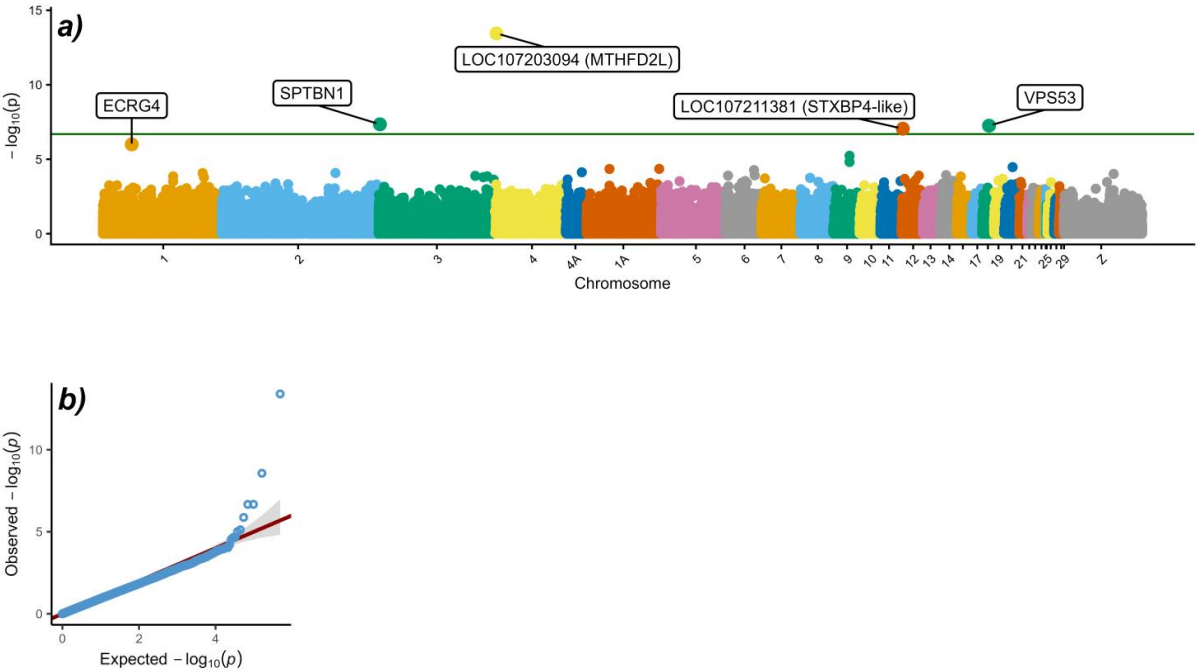

Figure 4: Manhattan plot (a) for the association between marker genotype and natal dispersal distance for all 243,629 SNPs and 139 Corsica individuals and associated Q-Qplot (b). a) The X-axis represents the SNPs (coloured points) positions and chromosome across the Great tit reference genome, and the Y-axis is the negative log base 10 of the p-values of SNPs. The green solid line shows the Bonferroni significant threshold (−log10(p) = 6.69). SNPs strongly associated with the trait will have a higher Y-coordinate value due to lower p-value. **b)** The Y and X-axis of the quantile-quantile plot indicate, respectively, the observed and expected negative base 10 logarithm of p-values. The majority of SNPs (blue circles) closely follow the null hypothesis of no association between SNPs and natal dispersal distance (red line). The significant SNPs deviate strongly from the diagonal, producing an excess of small p-values consistent with true association signals. The grey surface shows the 95% CI for the Q-Qplot under the null hypothesis.

Discussion

This study quantifies the genetic architecture of a key life-history trait to assess its evolutionary potential under rapid anthropogenic change. Using several decades of breeding data from four blue tit populations and combining quantitative genetics, genome-wide

association, and permutation approaches, we reveal a genetic basis for natal dispersal, supported by significant SNP effects, heritability, and evolvability in at least one of our four populations. However, our results show that, without accounting for spatial structure, we would have erroneously inferred high evolvability across all populations. Accounting for spatial structure influences proved highly challenging, as its omission can lead to misleading inferences about adaptive potential.

Shared environmental effects and spatial structure

Dispersal and its behavioural components, including movement decisions and habitat selection, are strongly influenced by environmental conditions [16,17] and are therefore expected to exhibit limited additive genetic variance [19,20]. Consistent with this expectation, we found low heritability for natal dispersal distance (3–13% across populations; Table S6). These estimates were further reduced after accounting for spatial structure, indicating that the spatial configuration of nest boxes can generate "phantom" genetic variance [30,31,37,81–83]. Only E-Pirio retained significant heritability after accounting for spatial structure ($p = 0.001$), suggesting that the estimates obtained in the other populations may partly reflect spatial bias rather than biological signal (Figure 3). This bias arises because the spatial arrangement of nest boxes generates correlations between genetic relatedness and local environmental conditions, allowing shared environments among relatives to mimic genetic effects. However, our limited statistical power to distinguish very low heritability estimates (<0.10) from zero (see supplementary material, Table S4b) means that the loss of significant heritability after accounting for spatial structure should not necessarily be interpreted as an absence of genetic variation. Instead, it may reflect heritability that is too

low to be reliably distinguished from spatial effects. Thus, while our results do not imply that heritability in the three populations is driven solely by shared environmental effects, they highlight the difficulty of disentangling genetic and environmental sources of variation in wild populations when relatives experience similar habitat configurations.

Importantly, accounting for nest-of-origin effects alone was insufficient to remove this bias, indicating that broader spatial structure in the nest-box network contributes to estimates of genetic variance. Partitioning variance associated with spatial configuration from additive genetic variance will therefore be an important next step, although such models may require larger datasets and may face identifiability issues if habitat choice itself has a genetic component, generating gene–environment correlations [84]. Automated tracking systems such as ATLAS could partly overcome these limitations by allowing continuous monitoring of individuals across landscapes rather than restricting detection to predefined nest boxes. Nevertheless, explicitly incorporating spatial structure into quantitative genetic and genomic analyses will remain essential for reliably estimating the genetic architecture of dispersal.

Evolutionary potential and its constraints

Although only one heritability estimate remained significant after accounting for spatial structure (h^2 = 13.2%), the estimates in the other populations were of similar magnitude and were within the range reported for dispersal and fitness-related traits [21,85]. They were lower than the average heritability reported for behavioural traits (mean h^2 = 23.5% and 35%; [21,85]), but comparable to estimates for dispersal and life-history traits (mean h^2 = 12% and 21%, respectively; [85]). If natal dispersal is tightly linked to fitness, as suggested by previous

studies, directional selection could contribute to the relatively low additive genetic variance expected for traits under strong selection [86–90].

Low heritability, however, does not necessarily imply limited evolutionary potential. Evolvability estimates ranged from 9.2% to 16.1% across populations (Table S6), comparable to and slightly higher than estimates reported for fitness-related traits (8.56%; [85]). After accounting for spatial structure, only E-Pirio retained significant evolvability (IA = 16.1%). These values were higher than those reported for fine-scale movement behaviours in roe deer (1.1–4.3%; [91]), although such movements differ substantially from large-scale natal dispersal in their ecological context and potential selective pressures. Taken together, our results suggest that natal dispersal can retain appreciable evolutionary potential despite relatively low heritability. Higher evolvability compare to heritability may also indicate that a substantial proportion of phenotypic variation arises from environmental and non-additive genetic effects, such as dominance and epistasis [65]. The high residual variance observed in our analyses is consistent with this interpretation. Investigating the non-additive genetic architecture of dispersal [92,93] will therefore be important for identifying additional sources of variation and improving predictions of its evolutionary dynamics.

Limits to estimating evolutionary potential in wild populations

The evolutionary potential of natal dispersal should nevertheless be interpreted cautiously because several features of our study system may further bias estimates of additive genetic variance. First, dispersal distances are measured only for individuals that survive the post-fledging period and subsequently reproduce in a nest box. Between 23% and 87% of individuals die within three weeks of fledging [94], suggesting strong early viability selection

during dispersal. Such mortality could non-randomly remove individuals with extreme dispersal phenotypes before they enter the breeding population, thereby reducing the additive genetic variance observed among recruits.

Second, the artificial nest-box network restricts detection to a subset of the population and is likely to underestimate both mean dispersal distance and its genetic variance. In particular, our analyses capture predominantly short-distance dispersal because highly dispersive individuals are more likely to leave the study area undetected. An experimental study conducted over two years outside the nest-box network found that 45% of recruited males and 54% of recruited females dispersed more than 1.5 km from their natal nest (de Franceschi, unpublished). These undetected movements are likely to affect estimates of both phenotypic and genetic variation. Integrating capture–recapture approaches with quantitative genetic models could therefore provide a more explicit framework for accounting for imperfect detection [27,95–97].

Genetic architecture of dispersal

Natal dispersal is a multifactorial life-history trait involving behavioural, personality, cognitive and locomotor components [18,98]. Consistent with a genetic contribution to these underlying traits [32,99,100], we identified four candidate genes—*SPTBN*, *PHF2*, *SAP30BP* and *MTHFD2L*—that may contribute to variation in dispersal through effects on neurodevelopment, cognition, gene regulation and cellular metabolism (Table S8). Their functions suggest several potential pathways linking genotype to dispersal behaviour, including locomotor capacity, spatial learning, behavioural responsiveness and metabolic performance [101–105]. These traits shape how individuals perceive and respond to

environmental variation, thereby contributing to among-individual differences in dispersal patterns. However, these functional interpretations remain indirect, and further validation will be required to establish their roles in dispersal.

None of the loci identified here corresponded to candidate genes previously reported in GWAS of dispersal, highlighting the difficulty of detecting consistent genetic signals for this complex trait. Although some previous studies have suggested an oligogenic architecture, dispersal is generally expected to be highly polygenic, with many loci of small effect [46,47]. Our apparently oligogenic signal may therefore reflect limited statistical power: simulations indicated that approximately 25–40% of causal SNPs remained undetected in our study systems (Figure S4), with detection power declining further at low heritability and under highly polygenic architectures. Moreover, RADseq captured only a small fraction of genomic variation (~4%), further limiting our ability to detect loci of small effect.

Spatial structure may also influence genotype–phenotype associations if genetically related individuals experience similar environments, as observed for genetic variance. Although the magnitude of this potential bias remains unclear, spatial genetic structure has been proposed as a source of confounding in genotype–phenotype associations [106]. Whole-genome sequencing, combined with chromosome partitioning approaches such as those used in great tits [107], could therefore help determine how genetic variance is distributed across the genome and assess the contribution of polygenic and non-additive effects to dispersal variation.

Sex-specific variation and early-life effects

503 Despite accounting for maternal effects, sex and cohort [34,35,108], more than 50% of the
504 phenotypic variance in natal dispersal remained unexplained (Figure 1, Table S6). Sex
505 accounted for only ~4% of the variance, with females being the primary dispersing sex in
506 blue tits and generally dispersing farther than males [109]. This sex difference was also
507 evident in our data, with mean dispersal distances of 487–791.9 m in females compared with
508 286.3–732 m in males. However, the comparison between observed sex-specific dispersal
509 distances and the distances possible given the nest-box distribution (Table S5) suggests that
510 female dispersal is more strongly underestimated, consistent with the greater frequency of
511 undetected long-distance movements.

512 Early-life factors also contributed to variation in dispersal. Maternal identity explained 6% of
513 phenotypic variance, while hatch year and nest identity explained 2% and 5%, respectively
514 (Table S6). Although these effects were modest, they indicate that parental and natal
515 environments contribute measurably to dispersal phenotypes. Such effects may also operate
516 indirectly through habitat selection processes such as natal habitat preference induction
517 [36,110–113], suggesting that dispersal and settlement should be considered jointly in future
518 studies.

519 **Conclusion**

520 Our results demonstrate that spatial structure can substantially alter estimates of genetic
521 variance and evolutionary potential, even among populations of the same species.
522 Accounting for spatial autocorrelation reduced evidence for heritability and evolvability in
523 most populations, emphasizing the need to incorporate environmental and spatial structure
524 into quantitative genetic and genomic analyses of dispersal.

At the same time, the persistence of significant genetic variation in one population suggests that natal dispersal may retain the potential to evolve under environmental change. The magnitude and direction of such responses will depend not only on additive genetic variance, but also on selection, imperfect detection and genetic correlations with other behavioural and life-history traits [32,114–118]. In blue tits, coordinated variation among morphology, behaviour and life history [119] further suggests that dispersal may be embedded within broader phenotypic syndromes. Integrating quantitative genetics with high-resolution tracking and genomic approaches will therefore be important for determining how dispersal evolves and how such evolution feeds back on population connectivity and eco-evolutionary dynamics.

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553 **Author Contributions**

554 M.B.: Conceptualization, Data curation, Formal analysis, Methodology, Visualization,
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560 All authors gave final approval for publication and agreed to be held accountable for the
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562 **Conflict of interest declaration**

563 We declare we have no competing interests.

564 **Data accessibility**

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Ethics

Captures were performed under personal ringing permits delivered by the CRBPO (A.C. & CDF ringing permits no. 1907 & 1820) for the research program no. 369. All experimental protocols described were reviewed by the Ethics Committee for Animal Experimentation no. 036 (Languedoc Roussillon CEEA-LR) and approved on 12 December 2012, 5 June 2018, 11 October 2021 and 9 July 2024.

Declaration of AI use

AI-assisted technologies, namely ChatGPT, were used with human oversight in the writing process to improve the manuscript's readability and language once a full first draft was written.

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Supplementary Materials

Table S1: Empirical studies on the heritability of dispersal traits estimated through the animal model, with species names, type of dispersal trait, heritability (h^2), additive genetic variance (V_A) and the reference. Articles were selected based on the literature reviews conducted by Saastamoinen et al. (2018) and Dochtermann et al. (2019) [20,21]. More recent studies were identified using the following keywords in google scholar: "dispersal distance", "dispersal propensity", "dispersal heritability", "genetic architecture", and "animal model". Uncertainty around the mean estimates is not reported because of the high heterogeneity among studies.

Species	Dispersal Trait	Heritability (h^2)	Additive genetic variance (VA)	References
Great tit (*Parus major*)	Dispersal distance	0.253 (for female) 0.247 (for male)	0.157 0.168	McCleery et al., 2004
Western bluebirds (*Sialia mexicana*)	Dispersal propensity	0.60 (observed scale) 0.95 (underlying scale)	0.13	Duckworth et Kruuk, 2009
Cane Toad (*Rhinella marina*)	Dispersal rate	0.28	0.29	Phillips et al., 2010
Wandering albatross (*Diomedea exulans*)	Dispersal propensity	0.36 (liability scale)	0.64	Charmantier et al., 2011
Collared flycatchers (*Ficedula albicolis*)	Dispersal probability	0.21 (all dispersal event) 0.39 (natal dispersal)	0.024 0.053	Doligez et al., 2012
Great tit (*Parus major*)	Dispersal distance	0.15	0.024	Korsten et al., 2013
Piping plovers (*Charadrius melodus*)	Dispersal distance	0.03	0.0004	Saunders & Cuthbert, 2014
Alpine swift (*Apus melba*)	Dispersal propensity	0.598	7.608	Bize et al., 2017
Common lizard (*Zootoca vivipara*)	Dispersal propensity	0.35 (liability scale) 0.17 (observed scale)	1.148	San-Jose et al., 2023
House sparrows (*Passer domesticus*)	Dispersal propensity	0.121	0.489	Saatoglu et al., 2024
Common lizard (Zootoca vivipara)	Dispersal distance Dispersal statue	0.121 (natal dispersal) 0.217 (adult dispersal) 0.192 (natal statue) 0.105 (adult statue)	0.022 0.047 0.513 0.189	Koch et al., 2026

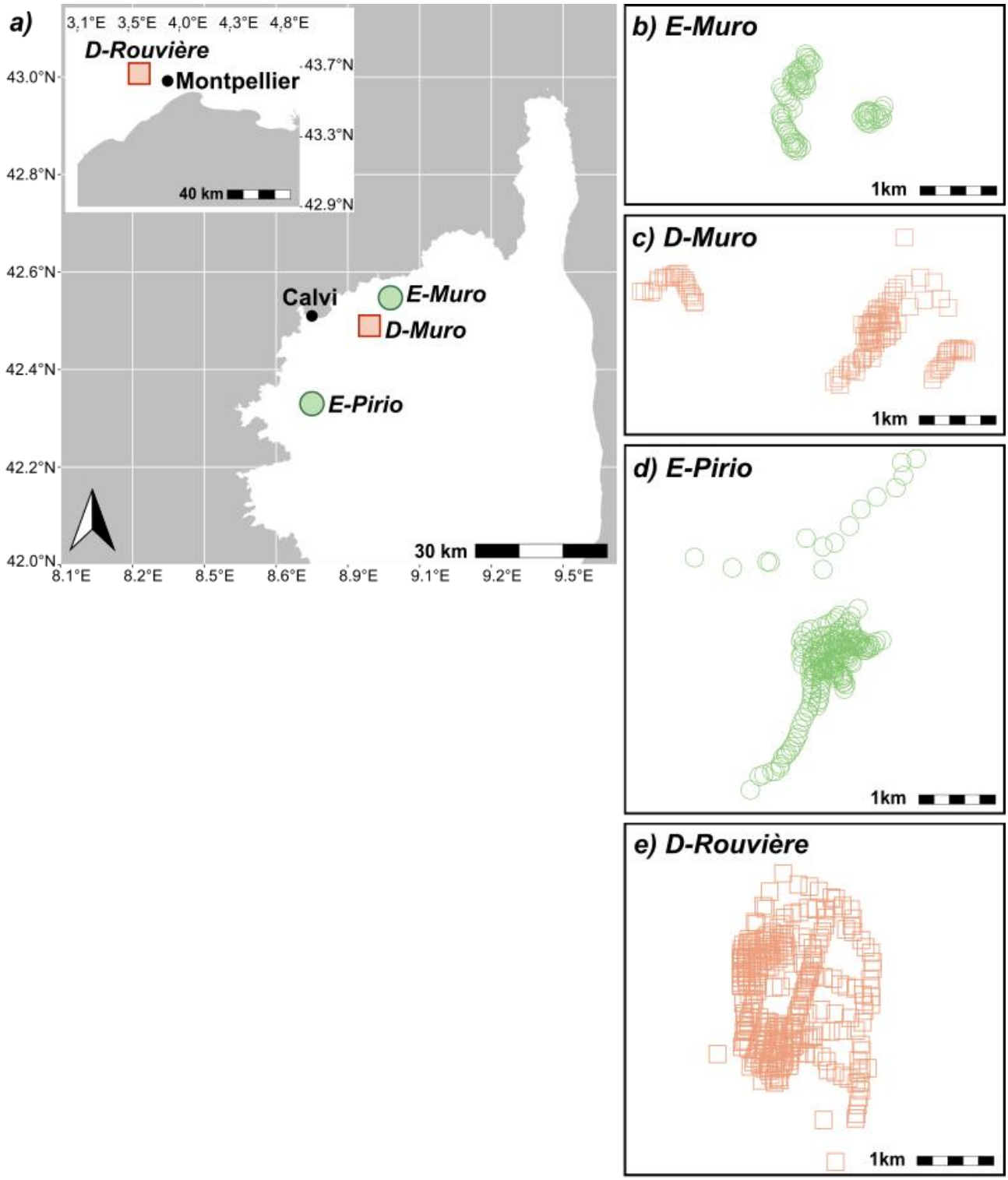


Figure S1: Map and distribution of nest boxes across mainland and Corsican blue tit populations. a) The top-left panel shows the location of the D-Rouvière site on the mainland. The main panel displays the locations of the E-Muro, E-Pirio, and D-Muro sites on the island of Corsica. Red squares indicate sites dominated by deciduous oaks, while green circles represent sites dominated by evergreen oaks. **b)** E-Muro, **c)** D-Muro, **d)** E-Pirio, and **e)** D-Rouvière panels show zoomed-in views of nest box locations, with symbols (red squares or green circles) corresponding to the dominant oak species.

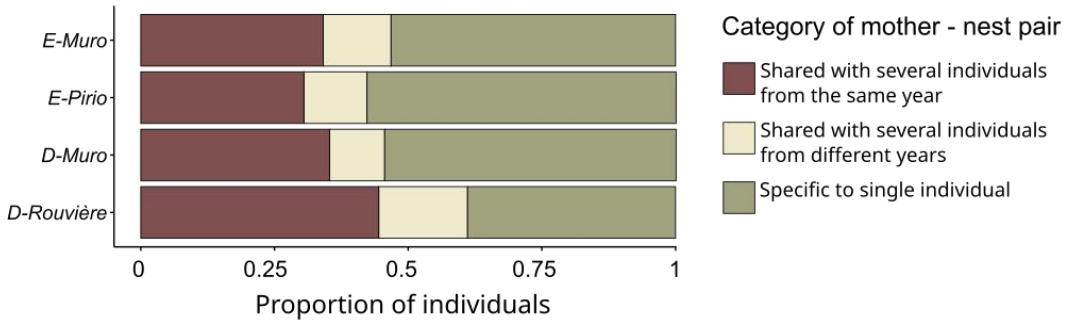
Table S2: Statistics overview for the pruned pedigrees of mainland (D-Rouvière) and Corsican (E-Muro, E-Pirio, D-Muro) blue tit populations. Pruned pedigrees retained only individuals informative for quantitative genetic analyses of natal dispersal distance.

Pedigree statistic	*E-Muro*	*E-Pirio*	*D-Muro*	*D-Rouvière*
Number of years included in the pedigree	26	45	31	33
N individuals	527	1,086	1,092	1,820
Number of links				
Maternal	295	569	565	1217
Paternal	287	565	536	1159
Full-sibs	89	177	166	752
Maternal half-sibs	171	323	299	1,256
Paternal half sibs	197	311	273	1,139
Maternal grandparents	155	279	232	931
Paternal grandparents	284	501	390	1,426
Maximum pedigree depth (N generations)	11	17	8	19
Number of pairwise relatedness estimated (≥ 0.025)				
≥ 0.5	676	1,312	1,268	3,141
≥ 0.25 - < 0.5	782	1,312	1,050	4,739
≥ 0.125 - < 0.25	845	1,318	8,72	6,631
≥ 0.025 - < 0.125	2,180	2,794	1,519	37,521
Total	4,483	6,736	4,709	52032

Text S1 - Evaluation of redundancy between the mother" and "nest" random effects on variance partitioning.

To assess whether the random effect of mother identity and birth nest were confounded between individuals, we calculated different proportions representing the degree of redundancy between these effects in our data sets. For all sites, we considered each mother–birth nest association and examined whether this association was unique to a single individual or shared among several individuals. In cases where multiple individuals shared the same mother and birth nest, we further determined whether sharing mother and birth nest occurred among individuals born in the same year or in different years. In this way, the redundancy between mother and nest effects was assessed through the proportion of individuals born in the same year and sharing the same mother–nest association. The mother and nest variables are considered correlated when individuals share the same mother–nest association across different years.

Figure S2: Proportion of individuals sharing or not, the same mother and birth nest across sites. In red, the proportion of individuals sharing a same mother–nest pair, born the same year, in yellow, the proportion of individuals sharing the same mother–nest pair, born in a different year and in green the proportion of individuals not sharing their mother–nest pair with any other individual in the dataset



These results show that the proportion of individuals born the same year and sharing the same mother and nest varies between 0.31 for E-Pirio and 0.45 for D-Rouvière. Thus, we can expect that our model for the D-Rouvière population may have difficulties distinguishing Vm and Vn.Hence, to evaluate whether our models are able to accurately estimate Vm and Vn, as well as the proportion of variance they account for, we fit on the basis of the "*Full*" model,

three supplementary models for each site. The models "*Without nest*" and "*Without mother*" no longer have the nest and mother effects, respectively.

Table S3: Proportion of variance for natal dispersal distance estimated from three models for each site. The "*Full*" model is presented in the 4th part of Material & Methods (Eq. 1). The model "*Without nest*" and "*Without mother*" are derived from the "*Full*" model, by removing nest then mother effect respectively. Estimation of variance proportion is on the latent scale for the three first populations (log-transformed scale), due to excessive computation time, but not in D-Rouvière. Ratio of phenotypic variance components are reported in columns (h^2: heritability; m^2: maternal ; n^2: birth nest ; y^2: birth year ; f^2: fixed effects ; r^2: residual variance). 95% CI are given in brackets.

Model	h^2	m^2	n^2	y^2	f^2	r^2
E-Muro (latente scale)						
Without nest	0.136 (<0.001 - 0.33)	0.035 (<0.001 - 0.17)	-	0.005 (<0.001 - 0.042)	0.110 (0.053 - 0.18)	0.68 (0.47 - 0.87)
Without mother	0.082 (<0.001 - 0.26)	-	0.105 (<0.001 - 0.20)	0.005 (<0.001 - 0.037)	0.111 (0.053 - 0.18)	0.68 (0.50 - 0.83)
Full	0.075 (<0.001 - 0.25)	0.021 (<0.001 - 0.12)	0.100 (<0.001 - 0.20)	0.005 (<0.001 - 0.038)	0.110 (0.055 - 0.18)	0.65 (0.46 - 0.82)
E-Pirio (latent scale)						
Without nest	0.233 (0.023 - 0.43)	0.039 (<0.001 - 0.15)	-	0.012 (<0.001 - 0.047)	0.094 (0.053 - 0.14)	0.60 (0.42 - 0.80)
Without mother	0.259 (0.075 - 0.45)	-	0.005 (<0.001 - 0.032)	0.012 (<0.001 - 0.047)	0.095 (0.052 - 0.14)	0.62 (0.43 - 0.81)
Full	0.227 (<0.001 - 0.39)	0.035 (<0.001 - 0.14)	0.004 (<0.001 - 0.031)	0.012 (<0.001 - 0.046)	0.095 (0.051 - 0.14)	0.60 (0.42 - 0.79)
D-Muro (latent scale)						
Without nest	0.148 (<0.001 - 0.32)	0.018 (<0.001 - 0.10)	-	0.013 (<0.001 - 0.053)	0.083 (0.046 - 0.13)	0.72 (0.52 - 0.89)
Without mother	0.155 (<0.001 - 0.33)	-	0.011 (<0.001 - 0.052)	0.014 (<0.001 - 0.054	0.084 (0.046 - 0.13)	0.72 (0.52 - 0.90)
Full	0.125 (<0.001 - 0.31)	0.017 (<0.001 - 0.099)	0.011 (<0.001 - 0.051)	0.013 (<0.001 - 0.056)	0.084 (0.046 - 0.13)	0.7^2 (0.53 - 0.88)
D-Rouvière (observed scale)						

Without nest	0.059 (<0.001 - 0.22)	0.078 (<0.001 - 0.15)	-	0.017 (<0.001 - 0.042)	0.064 (0.036 - 0.095)	0.77 (0.65 - 0.86)
Without mother	0.140 (<0.001 - 0.26)	-	0.012 (<0.001 - 0.057)	0.014 (<0.001 - 0.038)	0.061 (0.032 - 0.091)	0.76 (0.64 - 0.88)
Full	0.059 (<0.001 - 0.22)	0.067 (<0.001 - 0.14)	0.007 (<0.001 - 0.047)	0.017 (<0.001 - 0.042)	0.065 (0.036 - 0.096)	0.76 (0.65 - 0.86)

943

944 Table S3 shows that maternal and nest effects influence each other's estimation when one of
945 them is removed from the model, and that this also affects heritability estimates. The
946 magnitude of this connection differs between sites. When comparing the proportion of
947 variance explained by the maternal effect between the model "*Full*" and the model "*Without*
948 *nest*", the estimates remain almost unchanged for E-Pirio and D-Muro sites. The same
949 pattern is observed for the variance explained by the nest effect. These results suggest that,
950 for these sites, the partial confounding between maternal and nest effects has little impact
951 on the proportion of variance attributed to each effect.

952 In contrast, for site E-Muro and D-Rouvière, the variance explained by maternal and nest
953 effects is lower in the "*Full*" model than in the models "*Without nest"* and "*Without mother"*.
954 In D-Rouvière, m^2 decreases from 7.8% in the model "*Without nest"* to 6.7% in the model
955 "*Full"*, and n^2 decreases from 1.2% in the model "*Without mother*" to 0.7% in the model
956 "*Full"*. For E-Muro, m^2 decreases from 3.5% in the model "*Without nest*" to 2.1% in the model
957 "*Full*", and n^2 decreases slightly from 10.5% in the model "*Without mother*" to 10% in the
958 model "*Full*". These results indicate that when both maternal and nest effects are included in
959 the same model, the proportion of variance explained by each effect differs from that
960 obtained in models where they are fitted individually. Confounding between maternal and

nest effects occurs in these populations, making it more difficult for the models to distinguish the variance associated with each effect.

Overall, these results are consistent with the patterns shown in Figure S1 and indicate that the proportion of individuals for which maternal and nest effects are confounded, has a limited impact on the variance estimates associated with these two effects.

Text S2 - Statistical power analysis and relative bias estimates of variance components.

To verify that our sampling design was sufficient to detect genetic variance and reliably estimate variance components, we conducted simulation analyses following the approach of Pick et al. (2023). For simplicity, we simulated only additive genetic (V_A) and maternal (V_M) variance components. Using the *squidSim* R package, we generated synthetic populations that mirrored our empirical data structure (e.g., number of individuals, sex ratio, and number of years) and trait distribution (Gaussian) for each population separately. Simulations were performed across the following combinations of variance components:

- V_A=0,05; 0,1; 0,2
- V_M=0,05; 0,1; 0,2
- V_R (within-individual/residual) = 1 - (V_A+V_M)

, so that the total phenotypic variance was standardized to 1 and ratio are equal to variance. For each combination, we generated 100 datasets and fitted a linear mixed model to each dataset, using the same model structure as in our primary analysis. We then calculated the relative bias for each parameter set as:

$$\frac{1}{n}\sum_{i=0}^{n}\frac{\widehat{\theta_i}-\theta}{\theta}$$

where θ is the true parameter value, $\widehat{\theta_i}$ is the estimate from the i^{th} simulation, and n is the number of simulations (Pick et al., 2023 ; TableS2a). In addition, to assess the statistical power to detect variance components different from zero, we simulated 100 null distributions (i.e., datasets where the simulated variance of interest is equal to zero) with different set of V_M, following Pick et al. (2023) and using the squidSim R-package. Based on these 100 null distributions (V_A is expected to be 0), we estimated statistical power as the percentage of null distributions that were greater than different V_A thresholds ($V_{A\_null} > V_{A\_threshold}$).

Table S4a: Percentage bias in average heritability and maternal effect estimates obtained from simulated datasets across the four study populations. Bias is expressed as a percentage, with positive values indicating an upward bias and negative values a downward bias. Because total phenotypic variance was standardized to one, V_A is equivalent to heritability (h^2), and V_M corresponds to the proportion of variance explained by maternal effects. Bias was calculated as the percentage deviation between the median parameter estimate obtained across the 100 simulated datasets and the true simulated value, thereby reflecting the accuracy of our model in estimating variance components.

Parameters		E-Muro		E-Pirio		D-Muro		D-Rouvière	
V_A (h^2)	V_M (m^2)	bias V_A	bias V_M	bias V_A	bias V_M	bias V_A	bias V_M	bias V_A	bias V_M
0,05	0,05	70,07	16,60	22,20	-2,53	16,76	-10,42	17,38	-11,01
0,05	0,10	55,36	-6,31	51,84	-10,85	40,33	-18,05	10,51	-3,89

0,05	0,20	102,82	-9,90	42,53	-5,29	69,18	-16,26	3,24	-2,38
0,10	0,05	-0,06	34,52	-4,66	14,94	3,97	10,19	-6,81	-5,33
0,10	0,10	4,72	-1,39	2,19	-10,04	14,76	-15,18	-5,02	1,73
0,10	0,20	27,25	-12,11	4,49	-10,78	6,69	-7,29	-5,52	-4,89
0,20	0,05	-13,54	40,59	-4,95	11,65	-8,13	15,98	1,53	-9,59
0,20	0,10	-3,69	-11,89	1,84	-4,54	-1,71	0,89	1,00	-1,52
0,20	0,20	3,13	-7,97	-4,76	-2,70	2,85	-2,01	-2,79	-0,89

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Table S4b : Statistical power to detect additive genetic variance (V_A) greater than zero for natal dispersal distance from simulated datasets across the four study populations. For each population, statistical power was estimated as the percentage of V_{A_null} values drawn from the null distribution ($V_A = 0$) that exceeded given threshold values (V_A = 0.05, 0.10 and 0.20), for three levels of V_M (0.05, 0.10 and 0.20). Bold values indicate that fewer than 5% of the null distribution exceeded the corresponding threshold, indicating sufficient statistical power to distinguish true heritability from the null expectation.

Parameters		E-Muro			E-Pirio			D-Muro			D-Rouvière		
V_{Anull}	V_M	% >	% >	% >	% >	% >	% >	% >	% >	% >	% >	% >	% >

		0,05	0,10	0,20	0,05	0,10	0,20	0,05	0,10	0,20	0,05	0,10	0,20
0	0,05	27	10	**3**	19	6	**1**	22	**2**	**1**	9	**2**	**0**
0	0.10	50	19	**4**	24	8	**0**	31	10	**0**	16	**4**	**0**
0	0.20	45	17	**3**	26	11	**0**	44	23	**4**	16	**1**	**0**

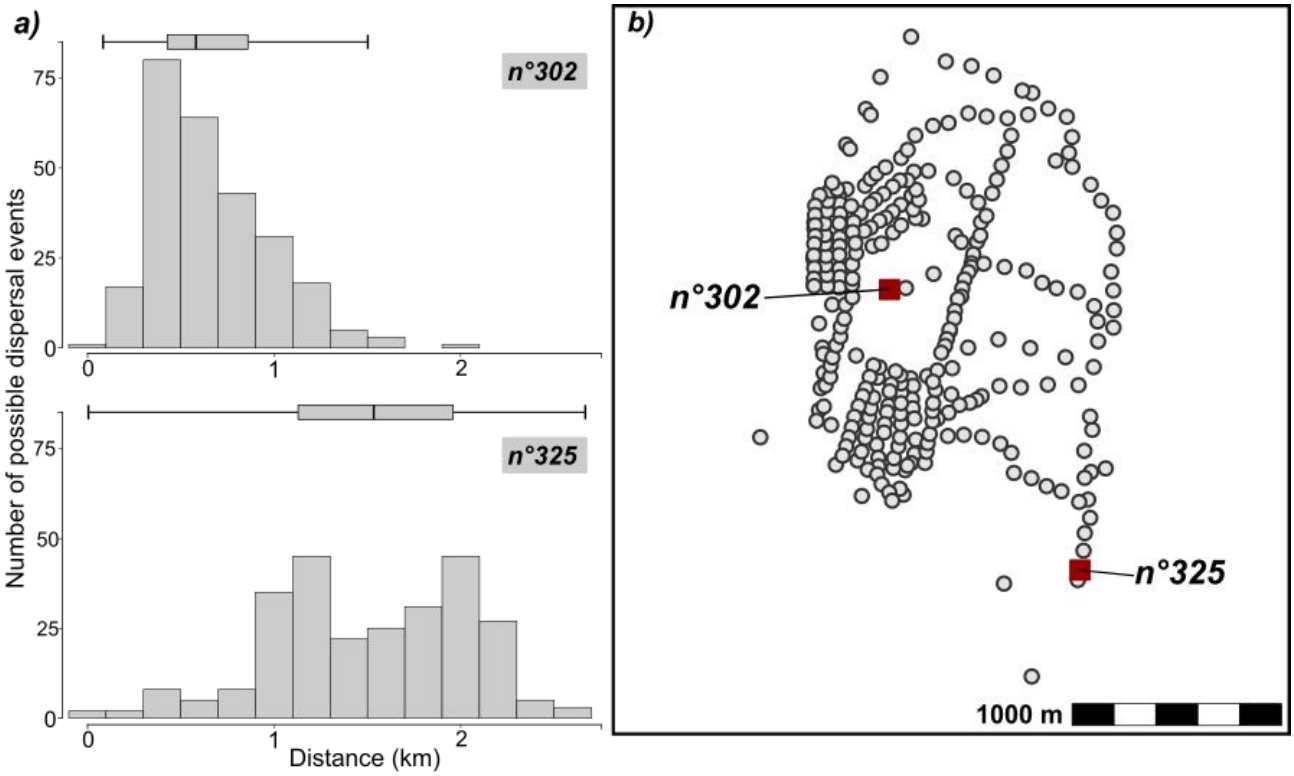

Figure S3 : Natal dispersal distance possibilities depend on spatial location of nest boxes. a) The top left panel shows the distribution of all possible natal dispersal distances for a fledgling that hatched in nest box n°302 and the bottom left panel similarly for a fledgling hatching in nest box n°325 in D-Rouvière. Boxes indicate the median, 25th and 75th percentiles, whiskers indicate the 10th and 90th percentiles. Minimum distances with other nest boxes in this site are 78m and 49m, maximum distances are 1,97km and 2,67km, respectively for the

nest boxes n°302 and n°325. b) Map of D-Rouvière with grey dots representing the location of nest boxes. Red squares are nests n°302 and n°325, while nest n°325 is at the edge.

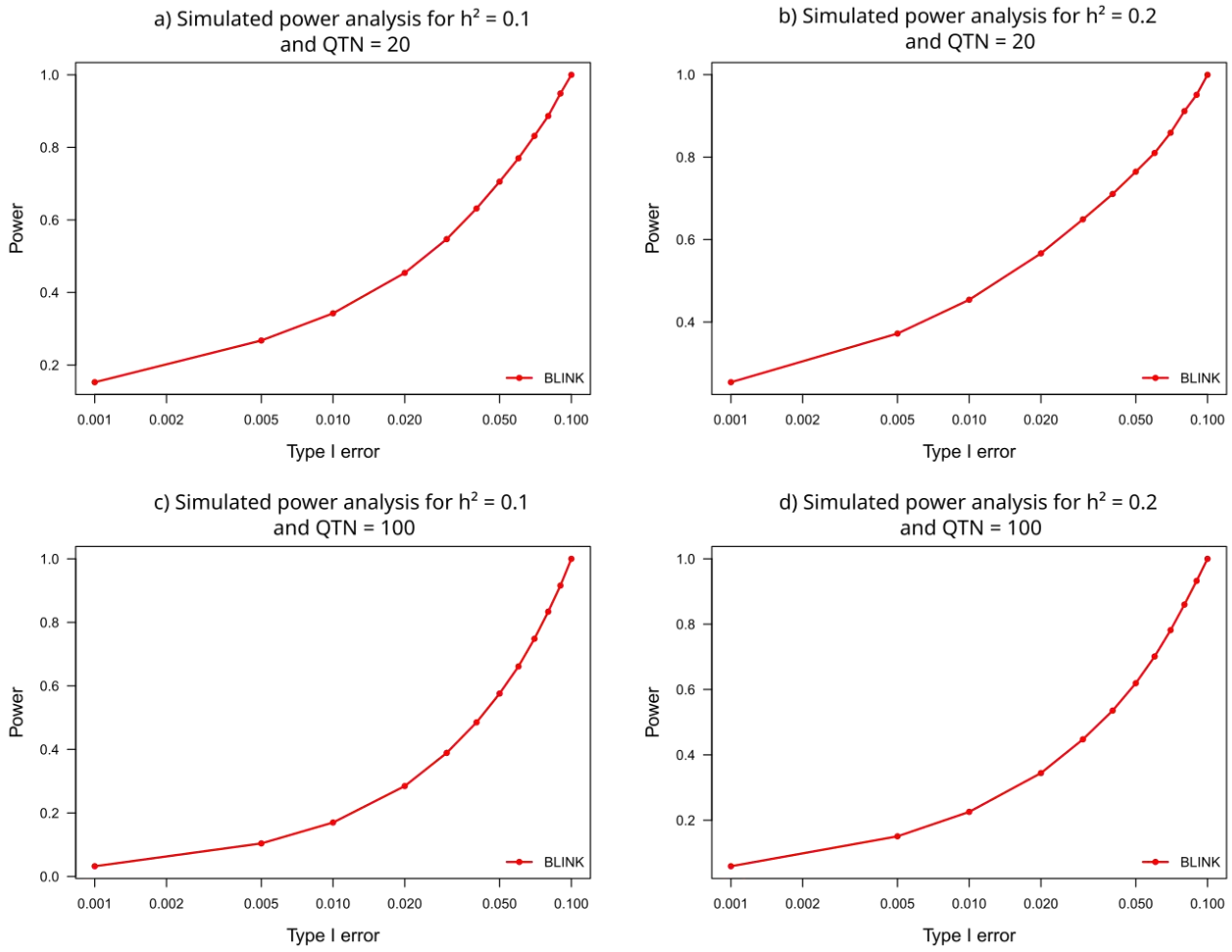

Figure S4 : Power of a simulated model to detect SNPs associated with a quantitative trait. The power analysis is based on a simulated trait using 137 blue tit individuals and 243,629 SNPs, under four different scenarios. The simulated trait harboured a heritability of 0.1 (a,c) or 0.2 (b,d), and 20 (a,b) or 100 (c,d) Quantitative Trait Nucleotides (QTN). The simulations were run 100 times using BLINK GWAS methods. Power was calculated as the proportion of QTN detected. Type I error was calculated as the proportion of tests with false positives.

Table S5: Descriptive statistics for sex-specific natal dispersal distances. Number of individuals phenotyped for natal dispersal (local recruits), with mean, median, and SD by population and sex. Mean of dispersal distance possibilities was estimated by calculating each possible dispersal distance from each nest box to the others.

Population	Sex	N. recruits	Mean(m) ± SD	Mean of dispersal distance possibilities (m) ± SD	Study period

E-Muro	Male	198	286.3 ± 273	592.9 ± 340	1998 - 2023
	Female	110	543.3 ± 345		
E-Pirio	Male	396	396.9 ± 330	732.2 ± 490	1976 - 2023
	Female	191	487.0 ± 394		
D-Muro	Male	356	297.7 ± 294	1197 ± 860	1993 - 2023
	Female	228	565.3 ± 567		
D-Rouvière	Male	753	732.0 ± 388	877.3 ± 850	1991 - 2023
	Female	486	791.9 ± 423		

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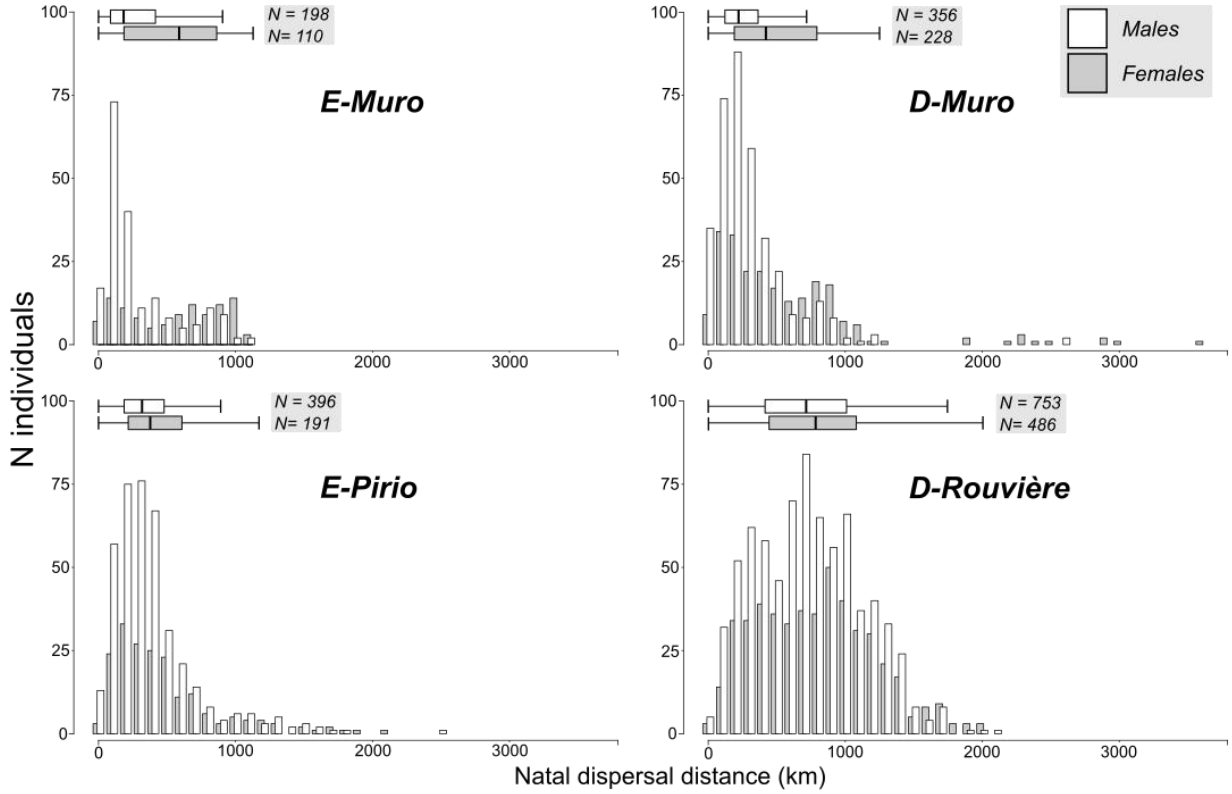

1005 **Figure S5 : Frequency distribution of natal dispersal distance of four blue tit populations for males (in white)**

1006 **and females (in grey)**. Boxes indicate the median, and 25th and 75th percentiles, whiskers indicate the 10th and

1007 90th percentiles. Sample sizes for each site are indicated separately for males and females.

Table S6: Proportion of natal dispersal distance variance for each component and evolvabilities. Quantitative genetic parameters were estimated for each site on the observed scale from the *"Full"* model. Ratios of phenotypic variance components are reported in columns for each of the following effects (h^2_obs: observed-scale heritability; m^2: maternal; n^2: birth nest; y^2: birth year; f^2: fixed-effect; r^2: residual), together with evolvability (I_A), defined as the additive genetic variance standardized by the squared phenotypic mean. Posterior distributions for each parameter are summarized by median in bold, then mean and 95% credible intervals in brackets. Note that the sum of variance proportions does not equal 1 because the sum of the medians differs from the median of the summed components. This deviation is larger for E-Muro, E-Pirio and D-Muro, as the back-transformation of variance components on the observed scale is non-linear and therefore does not perfectly recover the total phenotypic variance. Estimates shown in bold are considered to have moderate to strong support (i.e., CI > 0.001).

Site	h^2_{obs}	m^2	n^2	y^2	f^2	r^2	I_A
E-Muro	**0.036**; 0.044 (<0.001 - 0.118)	**0.011**; 0.019 (<0.001 - 0.068)	**0.053**; 0.056 (<0.001 - 0.110)	**0.002**; 0.005 (<0.001 - 0.019)	**0.059; 0.060 (0.028 - 0.097)**	**0.501; 0.500 (0.296 - 0.704)**	**0.092**; 0.117 (<0.001 - 0.371)
E-Pirio	**0.132**; 0.132 (<0.001 - 0.232)	**0.022**; 0.031 (<0.001 - 0.093)	**0.002**; 0.005 (<0.001 - 0.019)	**0.007**; 0.010 (<0.001 - 0.029)	**0.060; 0.060 (0.036 - 0.083)**	**0.462; 0.465 (0.273 - 0.666)**	**0.161; 0.163 (0.019 - 0.324)**
D-Muro	**0.062**; 0.066 (<0.001 - 0.150)	**0.009**; 0.016 (<0.001 - 0.054)	**0.006**; 0.009 (<0.001 - 0.027)	**0.007**; 0.010 (<0.001 - 0.029)	**0.046; 0.046 (0.024 - 0.070)**	**0.592; 0.591 (0.376 - 0.813)**	**0.149 ; 0.163 (0.002 - 0.423)**
D-Rouvière	**0.059**; 0.076 (<0.001 - 0.215)	**0.067**; 0.067 (<0.001 - 0.144)	**0.007**; 0.013 (<0.001 - 0.047)	**0.017**; 0;019 (<0.001 - 0.042)	**0.065; 0.066 (0.036 - 0.096)**	**0.764; 0.759 (0.648 - 0.860)**	**0.135**; 0.312 (<0.001 - 0.892)

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Table S7: Variance component estimates for natal dispersal distance of each population. Component of phenotypic variance (V_A: additive genetic variance, V_M: maternal variance, V_N: natal nest variance, V_Y: birth year variance, V_F: fixed-effect variance, V_R: residual variance, V_P: total phenotypic variance, μ: trait mean (adjusted for fixed effect) estimating from animal models. The posterior distribution of each variance component is described by the median in bold, the mean and 95% credible intervals are in brackets. Note that for E-Muro, E-Pirio, D-Muro, the sum of variance components after back-transformation does not exactly match the total phenotypic variance on the observed scale, as non-linear transformations alter the relationships among variance components.

Site	V_A	V_M	V_N	V_Y	V_F	V_R	V_P	μ
E-Muro	**18327** ; 25033 (57 - 89077)	**5553** ; 11032 (12 - 51361)	**27628** ; 32883 (3495 -	**1266** ; 2990 (3 - 15600)	**31128** ; 33977 (12606 -	**262629** ; 276258 (143335 -	**531068** ; 567923 (302897 -	**451** ; 455 (372 - 560)

			92647)		70652)	482396)	1044756)	
E-Pirio	**32407** ; 33720 (3854 - 70842)	**5421** ; 7882 (17 - 28518)	**665** ; 1329 (1 - 6141)	**1787** ; 2496 (8 - 8966)	**14879** ; 15444 (7635 - 26452)	**113579** ; 116023 (72551 - 172767)	**247596** ; 253640 (177295 - 363567)	**451** ; 452 (408 - 500)
D-Muro	**32574** ; 37607 (363 - 107020)	**4705** ; 8577 (11 - 37932)	**2904** ; 4759 (8 - 19420)	**3619** ; 5486 (17 - 22145)	**24356** ; 25564 (11838 - 46298)	**311064** ; 320119 (192657 - 496556)	**532019** ; 549631 (358299 - 842526)	**471** ; 473 (408 - 500)
D-Rouvière	**9398** ; 12222 (8 - 39044)	**10728** ; 10860 (36 - 25880)	**1046** ; 2087 (2 - 9298)	**2667** ; 3006 (91 - 8025)	**10456** ; 10618 (6030 - 16174)	**122528** ;122264 (102561 - 140415)	**160886** ; 161057 (148450 - 174742)	**258** ; 259 (108 - 412)

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Table S8: Single Nucleotide Polymorphisms (SNPs) and genes significantly associated with natal dispersal behaviour detected by a genome-wide association study. The SNP names come from the HapMap files (The International HapMap Consortium). We provide SNP information including the chromosome number and its reference on NCBI, the position in base pairs, the p-value indicating the significance of the association between SNPs and natal dispersal distance, the minor allele frequency (MAF), the number of genotyped individuals (Genotypes), the effect size, and the Bonferroni-corrected p-value for multiple testing (H&B.P.Value). The names of genes associated with the SNPs within a 5 kbp window are also provided.

SNP name	Chromosome name NCBI	Chromosome name RefSeq	Position(bp)	p.value	MAF	Genotypes	Effect	H&B.P.Value	Gene name
884127:191:-	4	NC_031771.1	1579274	3,580E-14	0,219	137	0,778	0,000	within gene *LOC107203094*

616711:32:-	3	NC_031770.1	1335245	4,530E-08	0,398	137	0,424	0,005	within gene *SPTBN1*
2130822:66:-	18	NC_031786.1	6766806	5,584E-08	0,146	137	0,556	0,005	within gene *SAP30BP*
1881676:202:+	12	NC_031781.1	1421770	8,937E-08	0,226	137	-0,462	0,005	within *PHF2*
65700:18:+	1	NC_031768.1	27602551	1,006E-06	0,212	137	-0,403	0,049	near *ECRG4* (>10 kb)

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Table S9: Comparison of Deviance Information Criterion (DIC) between the "*Full*" model and a model without the additive genetic variance component (no pedigree). The lower DIC value indicates a better fit. Removing pedigree information from our model and estimating the DIC provides a quality index for model comparison and allows assessment of whether additive genetic variance arises from genetic resemblance between relatives.

Model	"Full" model without additive genetic variance	"Full" model
Random effect	year + mother + nest	Year + mother + nest + ped
E-Muro	883.115	**874.302**

E-Pirio	1383.229	**1335.289**
D-Muro	1696.41	**1674.3**
D-Rouvière	18231.8	**18220.2**

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