Oikos 125: 1790–1801, 2016
doi: 10.1111/oik.02904

Subject Editor: Hamish McCallum. Editor-in-Chief: Dries Bonte. Accepted 10 April 2016

Immune gene variability influences roe deer natal dispersal

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Dispersal is a key life-history trait governing the response of individuals, populations and species to changing environmental conditions. In the context of global change, it is therefore essential to better understand the respective role of condition-, phenotype- and genetic-dependent drivers of dispersal behaviour. Although the importance of immune function and pathogen infestation in determining patterns of dispersal is increasingly recognised, no study to our knowledge has yet investigated the influence of immune gene variability on dispersal behaviour. Here, we filled this knowledge gap by assessing whether individual heterozygosity at five immune gene loci (one from the Major histocompatibility complex and four from encoding Toll-like receptors) influences roe deer natal dispersal. We found that dispersal propensity was affected by immune gene diversity, suggesting potential pathogen-mediated selection through over-dominance. However, the direction of this effect differed between high and low quality individuals, suggesting that dispersal propensity is driven by two different mechanisms. In support of the condition-dependent dispersal hypothesis, dispersal propensity increased with increasing body mass and, among high quality individuals only (standardized body mass $>$ 18 kg), with increasing immune gene diversity. However, among poor quality individuals, we observed the opposite pattern such that dispersal propensity was higher for individuals with lower immune gene diversity. We suggest that these poor quality individuals expressed an emergency dispersal tactic in an attempt to escape a heavily infested environment associated with poor fitness prospects. Our results have potentially important consequences in terms of population genetics and demography, as well as host–pathogen evolution.

In the context of global change, it is critical to better understand how individuals, populations and species cope with environmental modification. Dispersal is a crucial mechanism allowing individuals to respond to changing environmental conditions (Ronce 2007). First, in the context of ongoing climate change, dispersal provides individuals with the potential to track suitable environmental conditions. Second, by affecting spatial variation in genetic diversity among and within populations, dispersal can help mitigate the effects of genetic drift in small populations and decrease mutation load, hence reducing extinction risk. Finally, dispersal-mediated gene flow can also favor local adaptation. For instance, when strong co-evolutionary dynamics occur, increasing parasite dispersal can enhance local adaptation of parasites to their hosts (Gandon et al. 1996, Morgan et al. 2005, Garant et al. 2007). In this context, it is therefore essential to better understand the ultimate and proximate causes of dispersal and, in particular, the respective role of condition-, phenotype- and genetic-dependent drivers of dispersal propensity and distance (Clobert et al. 2009, Zera and Brisson 2012).

Recent studies have highlighted that an individual's decision to disperse or remain philopatric depends, in part, on local environmental conditions such as population density, resource availability or parasitism (condition-dependent dispersal; Massot et al. 2002). For this reason, as noted by Doligez et al. (2009), "dispersal is generally considered as a highly plastic, condition-dependent behaviour, with a complex external multi-determinism and thus low heritability". However, dispersal is highly heritable in several animal species (reviewed by Roff and Fairbairn 2001, Zera and Brisson 2012), including insects, birds and reptiles. Surprisingly, studies of the link between dispersal and genetics in mammals remain scarce (Zera and Brisson 2012) and, strikingly, have so far not identified any detectable heritability of dispersal (Waser and Jones 1989 in banner-tailed kangaroo rats *Dipodomys spectabilis*; Boonstra and Hochachka 1997 in collared lemmings *Dicrostonyx groenlandicus*), although a few studies have reported that microsatellite multilocus heterozygosity affects dispersal behaviour in mammals (Selonen and Hanski 2010 in flying squirrels *Pteromys volans*; but see Vanpé et al. 2015 in European roe deer *Capreolus capreolus*).

The genetic basis of dispersal is thought to be linked to genetic correlations between dispersal propensity and other phenotypic traits (Clobert et al. 2009). Indeed, because the phenotype of individuals can affect the balance between the costs and benefits of dispersal, dispersers often differ from residents in terms of their morphology, physiology, life-history traits and/or behaviour (phenotype-dependent dispersal). Many of these phenotypic traits are genetically determined to some degree (Clobert et al. 2009). However, only a few studies have identified a gene of known molecular function that influences dispersal rate or distance in animals (Haag et al. 2005). For instance, a positive association between allelic variation at the pgi enzymatic locus – a gene that plays an important role in controlling flight metabolism – and spatial variation in dispersal rate was found in the butterfly *Melitaea cinxia* (Haag et al. 2005). The difficulty of such candidate gene studies lies in the fact that the determinism of variation in dispersal or dispersal-related traits is often polygenic (Ronce 2007).

The influence of immune function and pathogen infestation on dispersal propensity is becoming increasingly recognised (reviewed by Boulinier et al. 2001, Møller et al. 2004). However, whether high levels of pathogen infestation or immune responsiveness should promote or inhibit natal dispersal is hard to predict. Dispersal might provide a way for highly parasitized or infected individuals to escape from heavily infested environments. For example, in cliff swallows *Hirundo pyrrhonota,* heavily parasitized nestlings dispersed to non-natal colonies to breed, whereas lightly parasitized nestlings returned to their natal colony (Brown and Brown 1992). Alternatively, low-quality individuals, with presumed low immune responsiveness, may be forced to disperse by highly competitive, high-quality individuals (Snoeijs et al. 2004). However, highly parasitized or infected individuals should also attempt to minimize costs by dispersing less often and/or less far than healthy individuals, as found in great tits *Parus major* in which flea infestation led to higher local recruitment of male fledglings and lower dispersal distances (Heeb et al. 1999).

Immune function and pathogen infestation are both potentially affected by immune gene diversity. There is increasing evidence for associations between specific immune gene alleles and pathogen resistance or susceptibility in wild mammalian populations. For instance, Ditchkoff et al. (2005) found a relationship between nematode abundance and allelic composition at the MHC-DRB gene in white-tailed deer *Odocoileus virginianus*. In comparison, a few studies (exclusively on rodents) have reported a lower pathogen susceptibility of heterozygous over homozygous individuals (i.e. over-dominance) in wild mammal populations. For example, Oliver et al. (2009) reported that MHC heterozygotes were co-infected by fewer parasites than homozygotes in water voles *Arvicola terrestris*. Despite this, no study has yet investigated the potential link between immune gene variability and dispersal propensity.

We therefore aimed to fill the gap by assessing whether immune gene polymorphism influences natal dispersal in a roe deer population inhabiting a heterogeneous agricultural landscape. This population is known to be heavily infested by intestinal parasites (Debeffe et al. 2014) and to have a high prevalence of abortive diseases (Candela et al. 2014). Interestingly, in the same roe deer population, Debeffe et al. (2014) recently found that dispersal propensity generally increased with both decreasing nematode burden and increasing body mass, supporting the condition-dependent dispersal hypothesis (Bonte and de la Peña 2009). However, the lowest quality individuals (i.e. both light and heavily parasitized) also dispersed, suggesting the existence of a "leave-it" emergency life-history tactic (sensu Wingfield (2003) who coined this term for behavioural and physiological responses for escaping long-term perturbations of the physical environment) in response to high parasite levels in the natal environment. In addition, we recently reported that individual heterozygosity at 12 putatively neutral microsatellite markers had no detectable effect on either dispersal propensity or dispersal distance in the roe deer population under study (Vanpé et al. 2015).

As a proxy of immune responsiveness, we evaluated individual heterozygosity at a panel of five immunity-related genes: the widely-used MHC class II DRB exon 2 locus (hereafter noted MHC-DRB), and four non-MHC loci encoding Toll-like receptors (TLRs). These five loci play a central role in pathogen recognition and host defense in domestic and/or wild ungulates, with known associations to health and fitness components, as well as to susceptibility to pathogens known to occur in roe deer (Quéméré et al. 2015). Quéméré et al. (2015) recently found marked immune genetic diversity in the study population and consistent signatures of balancing selection that indicate the key role of these immunity-related genes in the co-evolutionary dynamics with pathogens. We assumed that heterozygosity at these five immune gene loci positively affects immune responsiveness and pathogen resistance in roe deer through overdominance, with more heterozygous individuals being able to recognize a broader range of pathogens than homozygotes and consequently being less heavily infected, leading to higher fitness (Sommer 2005). Since there is no clear consensus on how pathogens and/or immune responsiveness impact dispersal, we chose not to formulate a priori predictions. Rather, our objective was to explore the relationship between the level of heterozygosity at immune gene loci and roe deer natal dispersal, in terms of both dispersal propensity and distance, while controlling for variation in individual quality (indexed by body mass).

Methods

Study area

The study was carried out in a heterogeneous agricultural landscape of the Aurignac canton (43°13′N, 0°52′E) in southwest France. The study area, which covers approximately 10 000 ha, is fragmented, with a mix of open fields (including 38% meadows and pastures for cattle, sheep and horses, and 32% crops), hedges (16%), small woodland patches (14%), and two larger forest blocks (5%) (see Hewison et al. 2009 for further details). Small villages and farms are distributed along the extensive road network.

We identified two sectors of contrasting landscape structure based on woodland extent: the closed sector included the two large forest blocks, whereas the open sector

included the remaining part of the landscape and was composed of a more open landscape of fragmented woodland (details in Debeffe et al. 2012). These sectors have previously been shown to affect roe deer life-history traits, including body mass (Hewison et al. 2009) and both the rate and distance of natal dispersal (Debeffe et al. 2012). Roe deer population density was estimated to be 9.3 ± 1.3 deer per 100 ha in the open sector, but 2–3 times higher in the forest sector (Debeffe et al. 2012).

Roe deer in this population are often infested by intestinal parasites such as strongyle nematodes and *Nematodirus*, which negatively impact their body condition (Debeffe et al. 2014). High prevalence of *Toxoplasma gondii* (46%), *Chlamydophila abortus* (17%) and *Coxiella burnetii* (11%), which may provoke abortion and reproductive failure or increase adult mortality rates in large herbivores (Shewen 1980), have also been reported in this population (Candela et al. 2014).

Capture and handling

The roe deer population of Aurignac has been intensively monitored by capture–mark–recapture (CMR) methods for more than 15 years. Each year since 2001, roe deer are caught by drive-netting during winter from November to March. For each captured animal, sex, age class (juveniles: ≤ 1 year of age versus adults: > 1 year of age; based on the presence or absence of a tricuspid third premolar milk tooth; Hewison et al. 1999), body mass (to the nearest 0.1 kg), geographical coordinates and habitat type (closed versus open sector) of the capture site are also recorded. An ear skin sample is also collected and stored in 95% ethanol prior to DNA extraction. Some individuals (including all caught juveniles) are equipped with a GPS collar before release (Debeffe et al. 2012).

In roe deer, only juveniles (approximately 1 year old) disperse and both sexes disperse in similar proportions (Debeffe et al. 2012). Hence, here, we only considered individuals that were captured as juveniles when between five and ten months old and subsequently monitored using GPS collars for approximately one year. In order to control for sex-specific body growth that occurs over the winter (Hewison et al. 2002), we controlled for capture date (as the Julian date) by adjusting juvenile body mass to 1 February using sex-specific linear regressions. We also standardized body mass for sex by adding the mean between-sex difference in body mass to the body mass of all females (see Vanpé et al. 2015 for a similar approach).

Assessment of natal dispersal

Dispersal status was assessed by comparing the location of the juvenile home range (from 1 January to 31 March) with that of the adult home range (from 1 July to 30 September) based on GPS tracking data and using the range stability index (Debeffe et al. 2012). Following Debeffe et al. (2012), individuals were considered to have dispersed when their average annual range stability index was equal to 0.5, corresponding to an absence of overlap between the juvenile and adult home ranges, or to be philopatric otherwise. To estimate dispersal distance as the individual based standardized dispersal distance (Debeffe et al. 2012), we considered the residual value from the linear regression of the Euclidean distance between juvenile and adult home range centres of gravity on the juvenile home range size in order to account for individual differences in spatial behaviour.

Choice of candidate genes

We selected five immunity-related genes, i.e. the MHC-DRB locus and four TLR genes. The MHC-DRB locus shows a signature of strong positive selection in cervids (Schaschl et al. 2006), as well as associations with nematode and tick resistance (e.g. in red deer *Cervus elaphus*: Fernández-de-Mera et al. 2009). TLRs comprise a family of transmembrane sensor proteins that sense the presence of microbial associated molecular patterns and induce innate immune responses (Akira et al. 2001). Genes encoding TLRs are involved in the host frontline defence against a wide repertoire of invasive microorganisms (Akira et al. 2001). A growing number of studies on free-ranging populations have illustrated the key role of pathogen-mediated balancing selection in driving the evolution of mammalian TLR genes (Tschirren et al. 2011, Takaki et al. 2012, Fornůsková et al. 2013). Here, we focused on the polymorphism of four TLR genes – TLR2, TLR3, TLR4 and TLR5. TLR2 and TLR4 are involved in resistance to *Toxoplama gondii* and Chlamydiae in mice (Rodriguez et al. 2006, Miller et al. 2009), two abortive pathogens affecting both domestic and wild ungulates, with high prevalence in the studied roe deer population (Candela et al. 2014). Genetic variation in TLR2 also plays a key role in susceptibility to *Mycobacterium avium* (the causative agent of paratuberculosis) (Mucha et al. 2009) and Anaplasma (Choi et al. 2004), two pathogenic bacteria regularly found in the studied roe deer population (Candela et al. 2014). Variability in TLR3 expression may play an important role in disease susceptibility of white-tailed deer to bluetongue and epizootic hemorrhagic disease viruses (Vos et al. 2009). TLR3 is also involved in the susceptibility or resistance to 'Peste des petits ruminants' viral infection, which is responsible for the ovine rinderpest or goat plague (Dhanasekaran et al. 2014). Finally, TLR5 is known to bind bacterial flagellin and is important in host defense against bacterial pathogens in several mammalian species (Metcalfe et al. 2014).

The functional polymorphisms (i.e. non-synonymous single nucleotide polymorphisms, SNPSs) of these five immunity-related genes in domains responsible for antigen binding affect the type of antigens recognized by the receptor. Hence, we can expect that individuals that are heterozygous for these five immune genes should be able to detect a wider range of pathogen-driven antigens than homozygous individuals due to a higher number of different MHC and TLR molecules and, consequently, be less heavily infected. This should be particularly true when measuring heterozygosity based on only non-synonymous SNPs.

Estimation of individual immune genetic diversity

For each individual, genomic DNA was extracted from alcohol-preserved tissues using the DNeasy Blood and Tissue kit (QIAGEN). Full details concerning laboratory

procedures, methods to identify the TLR and MHC-DRB gene haplotypes, with basic statistics about the polymorphism of the five immunity-related genes in the studied population, are provided in Quéméré et al. (2015).

For each individual, we estimated two measures of individual genetic diversity based on the MHC-DRB and the four TLR genes pooled together. First, we simply calculated the total number of alleles per individual (hereafter denoted N_A), estimated as the sum of all detected alleles across the five loci. Second, standardized mean observed heterozygosity (hereafter denoted H_O) was calculated using the GENHET R function as the proportion of heterozygous typed loci across the five immune genes divided by the mean heterozygosity of these loci in the population (Coulon 2010). Observed heterozygosity, which is usually defined as the proportion of heterozygous loci in an individual, is an effective and widely used measure of individual genetic diversity (Coulon 2010). To calculate H_O while avoiding potential problems linked to the presence of related individuals in the dataset (Coulon 2010), allele frequencies were estimated using a larger dataset from the same population (n = 382) than that used for the dispersal study (n = 71) (see Vanpé et al. 2015 for a similar approach). Note that we retained only individuals for which we had haplotype data for all the five immune genes.

We estimated these two measures of individual multilocus genetic diversity based on haplotypes reconstructed using: 1) all SNPs (hereafter denoted $H_{O(allSNPs)}$ and $N_{A(allSNPs)}$) and 2) non-synonymous SNPs only, hence, reflecting amino acid sequence diversity (hereafter denoted $H_{O(AA)}$ and $N_{A(AA)}$). Note that, contrary to non-synonymous SNPs, synonymous SNPs do not affect the amino-acid composition of the antigen-binding site of receptors so that estimates of immune gene diversity based on haplotypes reconstructed using synonymous SNPs only are uninformative (we do however include results of the analysis based on the synonymous SNPs only in the Supplementary material Appendix 1). The variance-covariance matrix of the heterozygosity measures, $H_{O(allSNPs)}$, $H_{O(AA)}$, but also a measure of H_O based on haplotypes reconstructed using synonymous SNPs only, as well as H_O based on the 12 putatively neutral microsatellite markers that we studied in Vanpé et al. (2015) are provided as Supplementary material Appendix 1 Table A1. If the potential effect of individual multilocus genetic diversity on dispersal behaviour is actually due to the functional role of the five immune gene loci (rather than simply to the genome-wide level of inbreeding), this effect should be stronger when using non-synonymous SNPs only than when using all SNPs to reconstruct haplotypes. Finally, in order to investigate the potential effect of heterozygosity at each of the five individual immune gene loci, we also assessed individual heterozygosity at TLR2, TLR3, TLR4, TLR5 and MHC separately (noted $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR4)}$, $H_{O(TLR5)}$ and $H_{O(MHC)}$, respectively) based on all SNPs and coded as a binary factor describing whether each locus was either heterozygous (coded as 1) or homozygous (coded as 0).

Statistical analyses

Complete data were available for 71 roe deer juveniles born between 2003 and 2012. We assessed the influence of immune gene diversity on natal dispersal propensity (a binary response variable: disperser versus philopatric) using generalized linear mixed-effect models with a binomial family (see Debeffe et al. 2012 for a similar approach) and the glmer function implemented in the lme4 package (Bates et al. 2013) of R (<www.r-project.org>). To investigate the effect of immune gene diversity on dispersal distance, we used mixed linear models (see Debeffe et al. 2012 for a similar approach) implemented in the lme function of R nlme package (Pinheiro et al. 2013). For both logistic and linear regressions, we included immune gene diversity, as well as standardized body mass (noted BM_{std}) and landscape sector (as a two modality factor: open versus closed), in our models as fixed effects to account for known condition-dependent dispersal and the effect of landscape sector on dispersal in this population. We also included the year of capture as a random factor to control for the potential non-independence of dispersal propensity and dispersal distance within cohorts (Debeffe et al. 2012).

We assessed the influence of immune gene diversity on natal dispersal propensity and dispersal distance by testing the effects of: 1) $H_{O(allSNPs)}$ and $N_{A(allSNPs)}$, 2) $H_{O(AA)}$ and $N_{A(AA)}$, and 3) $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR4)}$, $H_{O(TLR5)}$ and $H_{O(MHC)}$.

We first fitted the full model (including the main effects of immune gene diversity, standardized body mass BM_{std} and landscape sector, as well as the two-way interaction between immune gene diversity and BM_{std}), and then all possible simpler models derived from this full model (backward procedure, Searle 1971). Model selection was performed using the Akaike information criterion corrected for small sample size (AICc) as recommended by Burnham and Anderson (2002). We retained the model with the lowest AICc value, reflecting the best compromise between precision and accuracy. ΔAICc is the difference in AICc between a given model and the model with the lowest AICc. However, according to the parsimony rule, when simpler models (with a lower number of parameters, K) had AICc values which differed from the best model by < 2, we retained the simplest model. We also calculated AICc weights (wAICc) as a measure of the likelihood that a given model was the best among the set of fitted candidate models. All statistical analyses were carried out using R software (ver. 3.2.0) (<www.r-project.org>). Note that due to scaling issues, BM_{std} was centered around the mean for the generalized linear mixed-effect models testing the effects of $H_{O(allSNPs)}$, $N_{A(allSNPs)}$, $H_{O(AA)}$, or $N_{A(AA)}$ on dispersal propensity. Dispersal and genotyping data are available as Supplementary material Appendix 1 Table A2.

Note that neither *Ho* nor N_A were correlated with BM_{std} (Pearson correlation tests: r = –0.145, p = 0.23 for $H_{O(allSNPs)}$ and $N_{A(allSNPs)}$ and r = –0.200, p = 0.10 for $H_{O(AA)}$ and $N_{A(AA)}$). We also tested whether $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR4)}$, $H_{O(TLR5)}$ and $H_{O(MHC)}$ were related to BM_{std} using generalized linear mixed-effect models with a binomial family and whether BM_{std} was related to either $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR4)}$, $H_{O(TLR5)}$ or $H_{O(MHC)}$ using mixed linear models, with the year of capture as a random effect. Results showed that no statistically significant correlation occurred between either $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR4)}$, $H_{O(TLR5)}$ or $H_{O(MHC)}$ and BM_{std}, but a statistically significant relationship was found between BM_{std} and $H_{O(TLR4)}$ (Supplementary material Appendix 1 Table A3). BM_{std} was lower in heterozygous than in homozygous

individuals at TLR4 (for heterozygous individuals: mean coefficient ± SE = –0.910 ± 0.386, DF = 60, t = –2.359, p = 0.02, with homozygous individuals as a reference).

Note that because near identical results were obtained when analysing either the standardized mean observed heterozygosity or the number of alleles, for simplicity we present detailed results only for heterozygosity measures below.

Results

Effect of heterozygosity at the five immune gene loci on dispersal propensity

Irrespective of the measure of heterozygosity used (with haplotypes based either on all SNPs or on non-synonymous SNPs only), the selected model describing variation in dispersal propensity included interactive effects of heterozygosity and standardized body mass (Table 1A–B). Dispersal propensity increased with increasing heterozygosity, but only for heavy individuals (with $BM_{std} \geq 18$ kg; Fig. 1A–B). For light individuals (with $BM_{std} < 18$ kg), dispersal propensity decreased with increasing heterozygosity (Table 2A–2B, Fig. 1A–B). Interestingly, no dispersal occurred among individuals with particularly low standardized body mass (< 15 kg) but high heterozygosity (> 0.7; Fig. 1A–B). Landscape sector had no effect on dispersal propensity (for the open sector: mean coefficient ± SE = 1.196 ± 0.869 and 0.805 ± 0.916 for when using all SNPs and non-synonymous SNPs only, respectively, to measure heterozygosity, with the closed sector as the reference). We found a stronger relationship between heterozygosity and dispersal propensity when haplotypes were based on non-synonymous SNPs only than on all SNPs (Table 2A–B) (note, however, that dispersal propensity was not affected by heterozygosity measured at synonymous SNPs only: mean coefficient ± SE = –0.050 ± 0.513, p = 0.92; Supplementary material Appendix 1 Tables A6–A7).

Effect of heterozygosity at the five immune gene loci on dispersal distance

When using all SNPs to measure heterozygosity, the selected model of variation in dispersal distance only included

Table 1. Performance of the ten candidate mixed logistic regression and mixed linear regression models of variation in dispersal propensity and in dispersal distance (measured as individual-based standardized dispersal distance), respectively, for juvenile roe deer, including the effects of individual standardized observed heterozygosity at the five immune gene loci with haplotype reconstruction based on all SNPs (labelled $H_{O(allSNPs)}$) (A) or on non-synonymous SNPs only (labelled $H_{O(AA)}$) (B), standardized body mass (labelled BM_{std}) and landscape sector (labelled Sector) as fixed effects, as well as the year of capture as a random effect. Note: BM_{std} was centered around the mean for the mixed logistic regressions. Model selection was performed using the Akaike information criterion corrected for small sample size (AICc) as recommended by Burnham and Anderson (2002). ΔAICc is the AICc difference between the given model and the model with the lowest AICc. K is the number of parameters. wAICc is the AICc weight that is a measure of the likelihood that the given model is the best among the set of fitted models. The selected model (in bold) is generally the model with the lowest AICc value, reflecting the best compromise between precision and accuracy. However, according to the parsimony rule, when simpler models (with a lower number of parameters K) had an AICc which differed from the best model by < 2, we retained the simplest model. In such cases, the model with the lowest AICc occurs in italics and the retained simplest model occurs in bold.

(A)

	Dispersal propensity				Dispersal distance			
	K	AICc	ΔAICc	wAICc	K	AICc	ΔAICc	wAICc
$H_{O(allSNPs)} \times BM_{std}$ + Sector	6	91.24	0.25	0.35	*7*	*281.06*	*0.00*	*0.34*
$H_{O(allSNPs)} \times BM_{std}$	***5***	***90.99***	***0.00***	***0.40***	6	282.43	1.37	0.17
$H_{O(allSNPs)}$ + BM_{std} + Sector	5	95.26	4.27	0.05	6	282.88	1.81	0.14
$H_{O(allSNPs)}$ + BM_{std}	4	96.62	5.62	0.02	5	284.95	3.89	0.05
$H_{O(allSNPs)}$ + Sector	4	98.54	7.55	0.01	5	288.33	7.27	0.01
BM_{std} + Sector	4	93.80	2.80	0.10	**5**	**282.15**	**1.08**	**0.20**
$H_{O(allSNPs)}$	3	103.88	12.89	0.00	4	295.78	14.72	0.00
BM_{std}	3	95.15	4.16	0.05	4	284.03	2.97	0.08
Sector	3	96.70	5.71	0.02	4	286.82	5.76	0.02
Constant	2	101.89	10.90	0.00	3	293.89	12.82	0.00

(B)

	Dispersal propensity				Dispersal distance			
	K	AICc	ΔAICc	wAICc	K	AICc	AAICc	wAICc
$H_{O(AA)} \times BM_{std}$ + Sector	6	89.20	1.57	0.29	*7*	283.76	1.61	0.15
$H_{O(AA)} \times BM_{std}$	***5***	***87.63***	***0.00***	***0.64***	6	283.23	1.08	0.20
$H_{O(AA)}$ + BM_{std} + Sector	5	95.98	8.35	0.01	6	284.53	2.38	0.10
$H_{O(AA)}$ + BM_{std}	4	97.34	9.71	0.00	5	286.25	4.10	0.04
$H_{O(AA)}$ + Sector	4	98.94	11.31	0.00	5	288.92	6.77	0.01
BM_{std} + Sector	4	93.80	6.17	0.03	*5*	*282.15*	*0.00*	*0.33*
$H_{O(AA)}$	3	103.96	16.33	0.00	4	295.24	13.09	0.00
BM_{std}	3	95.15	7.53	0.01	**4**	**284.03**	**1.88**	**0.13**
Sector	3	96.70	9.08	0.01	4	286.82	4.67	0.03
Constant	2	101.89	14.27	0.00	3	293.89	11.74	0.00

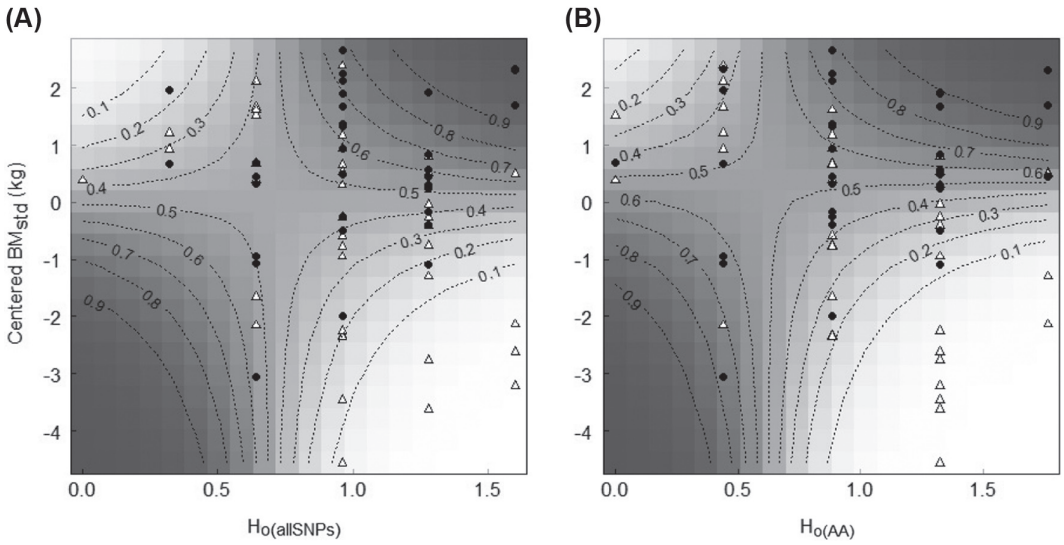

Figure 1. Dispersal propensity of roe deer juveniles (n = 71) as a function of standardized body mass centered around the mean (in kg) (labelled centered BM_{std}) and individual standardized observed heterozygosity at the five immune gene loci based on all SNPs (labelled $H_{O(allSNPs)}$) (A), and non-synonymous SNPs only (labelled $H_{O(AA)}$) (B). Note: the colours in the grid of gray-scale rectangles and the contour lines correspond to the values of dispersal propensity predicted by the selected models ($BM_{std} \times Ho_{(allSNPs)}$ and $BM_{std} \times H_{O(AA)}$ for (A) and (B), respectively). Dispersal propensity increases with increasing intensity of the grey shading. The black circles and the white triangles represent the dispersing and philopatric individuals, respectively.

additive effects of standardized body mass and landscape sector (Table 1A). Dispersal distance increased with increasing standardized body mass (mean coefficient ± SE = 0.340 ± 0.128) and was higher in the open than in the closed sector (for the open sector: mean coefficient ± SE = 1.092 ± 0.536, closed sector as a reference) (Table 3, Fig. 2A), but was not influenced by heterozygosity (mean coefficient ± SE = 0.720 ± 0.582).

When using non-synonymous SNPs to measure heterozygosity, the selected model of variation in dispersal distance only included standardized body mass (Table 1B). Dispersal distance increased with increasing standardized body mass (mean coefficient ± SE = 0.437 ± 0.122, $p = 7.10^{-4}$) (Fig. 2B), but was not affected by either heterozygosity (mean coefficient ± SE = –0.042 ± 0.465) or landscape sector (for the open sector: mean coefficient ± SE = 1.086 ± 0.543, with the closed sector as the reference).

However, note that the model including interactive effects between heterozygosity and standardized body mass and an additive effect of the landscape sector also received substantial support irrespective of the heterozygosity measure used (wAICc = 0.34 and 0.15 when using all SNPSs and non-synonymous SNPs only, respectively), although it was less parsimonious than the selected models (Table 1A–B). As for dispersal propensity, this model suggests that dispersal distance increased with heterozygosity among heavy individuals, but decreased with increasing heterozygosity among light individuals (see Supplementary material Appendix 1 Table A4 for the heterozygosity measure using all SNPs as an example) (note, however, that dispersal distance was not affected by heterozygosity measured at synonymous SNPs only: mean coefficient ± SE = 0.146 ± 0.409, p = 0.72; Supplementary materials Appendix 1 Table A6, A8).

Table 2. Parameter estimates of the mixed logistic regression models selected to describe variation in dispersal propensity of juvenile roe deer and including individual standardized observed heterozygosity at the five immune gene loci with haplotype reconstruction based on all SNPs (labelled $H_{O(allSNPs)}$) (A) or non-synonymous SNPs only (labelled $H_{O(AA)}$) (B), and standardized body mass centered around the mean (labelled BM_{std}) as fixed effects, as well as the year of capture as a random effect.

(A)

	Estimate	SE	z	p
Intercept	–0.049	0.901	–0.054	–
$H_{O(allSNPs)}$	–0.125	0.921	–0.136	–
BM_{std}	–1.412	0.792	–1.782	–
$H_{O(allSNPs)} : BM_{std}$	2.015	0.875	2.304	0.021

(B)

	Estimate	SE	z	p
Intercept	0.527	0.739	0.712	–
$H_{O(AA)}$	–0.731	0.734	–0.996	–
BM_{std}	–1.172	0.575	–2.036	–
$H_{O(AA)} : BM_{std}$	1.903	0.694	2.742	0.006

Effects of heterozygosity at individual immune gene loci on dispersal propensity

When analysing the effects of heterozygosity at individual immune gene loci, the selected models for variation in

Table 3. Parameter estimates of the mixed linear regression model selected to describe variation in individual-based standardized dispersal distance of juvenile roe deer and including standardized body mass (labelled BM_{std}) and landscape sector (labelled Sector) as fixed effects, as well as the year of capture as a random effect.

	Estimate	SE	DF	t	p
Intercept	–6.504	2.064	59	–3.151	–
BM_{std}	0.340	0.128	59	2.653	0.010
Sector	1.092	0.536	59	2.035	0.046

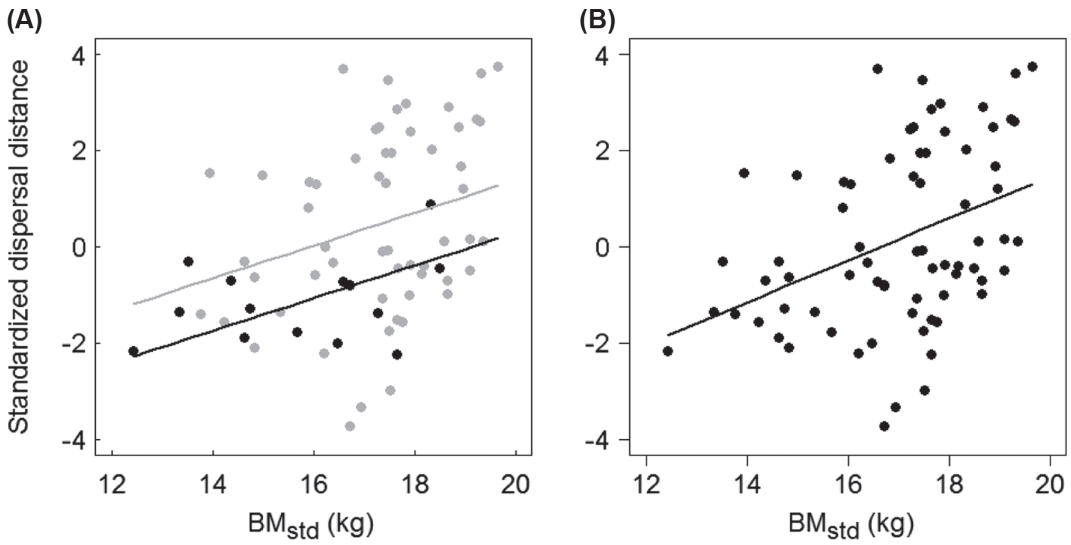

Figure 2. Individual-based standardized dispersal distance (labelled standardized dispersal distance) of roe deer juveniles (n = 71) as a function of standardized body mass (in kg) (labelled BM_{std}) for individuals from closed (black circles) and open (grey circles) landscape sectors (labelled Sector) (A) or for individuals from all sectors (B). Note: the black and grey lines in (A) represent the relationships predicted by the selected model (BM_{std} + Sector) for the closed and open landscape sectors, respectively, when using the $H_{O(allSNPs)}$ heterozygosity measure. The black line in (B) represents the relationship predicted by the selected model (BM_{std}) when using the $H_{O(AA)}$ heterozygosity measure.

dispersal propensity only included standardized body mass for all the five immune gene loci except TLR4 (Table 4A). For these four immune gene loci, dispersal propensity increased with standardized body mass (mean coefficient ± SE = 0.477 ± 0.176), but was not affected by genetic diversity at individual immune genes (for heterozygous individuals: mean coefficient ± SE = –0.072 ± 0.525, 0.792 ± 0.531, 0.440 ± 0.612 and –0.378 ± 0.512 for $H_{O(TLR2)}$, $H_{O(TLR3)}$, $H_{O(TLR5)}$ and $H_{O(MHC)}$, respectively, with homozygous individuals as the reference).

In contrast, for TLR4, the selected model for variation in dispersal propensity included interactive effects of $H_{O(TLR4)}$ and standardized body mass (Table 4A). Dispersal propensity increased with standardized body mass for heterozygous individuals at TLR4, whereas it decreased with increasing standardized body mass for homozygous ones (Table 5, Fig. 3). Landscape sector did not influence dispersal propensity in any of the models for the five immune gene loci (for the open sector: mean coefficient ± SE = 1.463 ± 0.842, with the closed sector as the reference).

Effects of heterozygosity at individual immune gene loci on dispersal distance

When analysing the effects of heterozygosity at individual immune gene loci, the selected model of variation in dispersal distance only included standardized body mass for TLR2, TLR5 and MHC (Table 4B). For these three immune gene loci, dispersal distance increased with standardized body mass (mean coefficient ± SE = 0.437 ± 0.122), but was not affected by genetic diversity at individual immune genes (for heterozygous individuals: mean coefficient ± SE = –0.176 ± 0.421, –0.037 ± 0.480 and –0.095 ± 0.411 for $H_{O(TLR2)}$, $H_{O(TLR5)}$ and $H_{O(MHC)}$, respectively, with homozygous individuals as the reference) or by the landscape sector (for the open sector: mean coefficient ± SE = 1.092 ± 0.536, with the closed sector as the reference).

For TLR4, the selected model of variation in dispersal distance included additive effects of standardized body mass and landscape sector (Table 4B). According to this model, dispersal distance increased with standardized body mass (mean coefficient ± SE = 0.340 ± 0.128) and was higher in the open than in the closed sector (for the open sector: mean coefficient ± SE = 1.092 ± 0.536, with the closed sector as the reference), but was not affected by $H_{O(TLR4)}$ (for heterozygous individuals: mean coefficient ± SE = 0.630 ± 0.426, with homozygous individuals as the reference). However, models including interactive effects between $H_{O(TLR4)}$ and standardized body mass also received substantial support, although they were less parsimonious than the selected model (Table 4B). These two models both suggested that dispersal distance increased with $H_{O(TLR4)}$ in heavy individuals, but decreased with increasing $H_{O(TLR4)}$ in light individuals (see Supplementary material Appendix 1 Table A5 for the model including the interaction between standardized body mass and heterozygosity).

Finally, for TLR3, the selected model of variation in dispersal distance included additive effects of $H_{O(TLR3)}$ and standardized body mass (Table 4B). While dispersal distance increased with standardized body mass (mean coefficient ± SE = 0.452 ± 0.119), it was higher in heterozygous individuals at TLR3 than in homozygous ones (for heterozygous individuals: mean coefficient ± SE = 0.827 ± 0.402, with homozygous individuals as the reference; Table 6, Fig. 4). In addition, dispersal distance was not affected by landscape sector (for the open sector: mean coefficient ± SE = 0.918 ± 0.538, with the closed sector as the reference).

Table 4. Performance of the ten candidate models of variation in dispersal propensity (A) and individual-based standardized dispersal distance (B) for juvenile roe deer, including the effects of heterozygosity at individual immune gene loci (labelled H_O), standardized body mass (labelled BM_{std}) and landscape sector (labelled Sector) for TLR2, TLR3, TLR4, TLR5 and MHC. Note: Model selection was performed using the Akaike information criterion corrected for small sample size (AICc) as recommended by Burnham and Anderson (2002). ΔAICc is the AICc difference between the given model and the model with the lowest AICc. K is the number of parameters. wAICc is the AICc weight that is a measure of the likelihood that the given model is the best among the set of fitted models. The selected model (in bold) generally is the model with the lowest AICc value, reflecting the best compromise between precision and accuracy. However, according to the parsimony rule, when simpler models (with a lower number of parameters K) had an AICc which differed from the best model by <2, we retained the simplest model. In such cases, the model with the lowest AICc occurs in italics and the retained simplest model occurs in bold.

(A)

		TLR2			TLR3			TLR4			TLR5			MHC		
	K	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc
$H_O \times BM_{std}$ + Sector	6	94.34	0.54	0.22	96.54	2.75	0.07	88.18	0.49	0.41	97.53	3.73	0.05	96.81	3.02	0.08
$H_O \times BM_{std}$	5	96.01	2.21	0.10	96.88	3.08	0.06	**87.68**	**0.00**	**0.53**	99.15	5.35	0.02	98.04	4.24	0.04
$H_O + BM_{std}$ + Sector	5	96.11	2.31	0.09	94.50	0.70	0.20	95.50	7.82	0.01	95.16	1.36	0.18	95.33	1.53	0.16
$H_O + BM_{std}$	4	97.38	3.58	0.05	95.11	1.31	0.15	96.87	9.19	0.01	96.88	3.08	0.07	96.86	3.06	0.07
H_O + Sector	4	98.83	5.03	0.02	98.17	4.37	0.03	98.87	11.19	0.00	97.33	3.53	0.06	97.66	3.86	0.05
BM_{std} + Sector	4	*93.80*	*0.00*	*0.29*	*93.80*	*0.00*	*0.28*	93.80	6.12	0.02	*93.80*	*0.00*	*0.35*	*93.80*	*0.00*	*0.34*
H_O	3	104.07	10.27	0.00	102.52	8.72	0.00	104.06	16.38	0.00	103.18	9.38	0.00	102.96	9.17	0.00
BM_{std}	3	**95.15**	**1.35**	**0.15**	**95.15**	**1.35**	**0.14**	95.15	7.47	0.01	**95.15**	**1.35**	**0.18**	**95.15**	**1.35**	**0.17**
Sector	3	96.70	2.90	0.07	96.70	2.90	0.07	96.70	9.02	0.01	96.70	2.90	0.08	96.70	2.90	0.08
Constant	2	101.89	8.10	0.01	101.89	8.10	0.00	101.89	14.21	0.00	101.89	8.10	0.01	101.89	8.10	0.01

(B)

		TLR2			TLR3			TLR4			TLR5			MHC		
	K	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc	AICc	ΔAICc	wAICc
$H_O \times BM_{std}$ + Sector	7	283.16	1.01	0.21	283.82	2.40	0.09	*280.56*	*0.00*	*0.33*	286.71	4.57	0.05	286.86	4.72	0.04
$H_O \times BM_{std}$	6	284.88	2.74	0.09	284.44	3.01	0.07	281.04	0.47	0.26	288.18	6.03	0.02	288.61	6.46	0.02
$H_O + BM_{std}$ + Sector	6	284.51	2.36	0.11	*281.43*	*0.00*	*0.30*	282.25	1.68	0.14	284.48	2.33	0.15	284.41	2.26	0.15
$H_O + BM_{std}$	5	286.16	4.02	0.05	**282.06**	**0.63**	**0.22**	284.54	3.97	0.04	286.34	4.19	0.06	286.29	4.14	0.06
H_O + Sector	5	289.12	6.98	0.01	287.39	5.96	0.02	288.55	7.99	0.01	288.81	6.67	0.02	288.70	6.55	0.02
BM_{std} + Sector	5	*282.15*	*0.00*	*0.35*	282.15	0.72	0.21	**282.15**	**1.58**	**0.15**	*282.15*	*0.00*	*0.47*	*282.15*	*0.00*	*0.47*
H_O	4	296.10	13.95	0.00	293.30	11.88	0.00	296.07	15.51	0.00	296.09	13.94	0.00	295.79	13.64	0.00
BM_{std}	4	**284.03**	**1.88**	**0.14**	284.03	2.60	0.08	284.03	3.46	0.06	**284.03**	**1.88**	**0.18**	**284.03**	**1.88**	**0.18**
Sector	4	286.82	4.67	0.03	286.82	5.39	0.02	286.82	6.26	0.01	286.82	4.67	0.05	286.82	4.67	0.05
Constant	3	293.89	11.74	0.00	293.89	12.46	0.00	293.89	13.32	0.00	293.89	11.74	0.00	293.89	11.74	0.00

Table 5. Parameter estimates of the model selected to describe the relationship between dispersal propensity of juvenile roe deer and heterozygosity at TLR4 (labelled $H_{O(TLR4)}$) and standardized body mass (labelled BM_{std}) as fixed effects, as well as the year of capture as a random effect.

	Estimate	SE	z	p
Intercept	7.497	6.381	1.175	–
$H_{O(TLR4)}$	–25.122	8.389	–2.994	–
BM_{std}	–0.435	0.362	–1.203	–
$H_{O(TLR4)}$: BM_{std}	1.467	0.482	3.045	0.002

Table 6. Parameter estimates of the mixed linear regression model selected to describe the relationship between individual-based standardized dispersal distance) of juvenile roe deer and heterozygosity at TLR3 (labelled $H_{O(TLR3)}$) and standardized body mass (labelled BM_{std}) as fixed effects, as well as the year of capture as a random effect.

	Estimate	SE	DF	t	p
Intercept	8.103	5.826	58	1.391	–
Ho	–13.719	4.848	58	–2.830	–
stdBM	–0.439	0.331	58	–1.329	–
Ho : stdBM	0.786	0.277	58	2.842	0.006

Discussion

We provide here a first empirical demonstration that immune gene variability can influence natal dispersal, but that this influence depends on individual quality. Whereas dispersal propensity of roe deer juveniles increased with increasing heterozygosity at our five immune gene loci for high quality individuals ($BM_{std} \geq 18$ kg), it decreased with increasing heterozygosity for low quality individuals ($BM_{std} < 18$ kg). Interestingly, all juvenile roe deer with a standardized body mass lower than 15 kg and heterozygosity above 0.7 were philopatric. These effects may be mainly driven by the TLR4 locus because this was the only individual immune gene locus in which heterozygosity was associated with dispersal propensity: dispersal propensity increased with body mass for heterozygous individuals at TLR4, whereas it decreased with increasing mass for homozygous ones. In contrast, while dispersal distance clearly increased with standardized body mass, the effects of heterozygosity at our five immune gene loci and of heterozygosity at TLR4 on dispersal distance were less clear. Interestingly, we found that heterozygous individuals at TLR3 dispersed longer distances than homozygous ones.

A likely explanation for these observations is that individuals with high levels of heterozygosity at immune gene loci are able to recognize a larger spectrum of pathogens and therefore are more resistant to pathogens and less heavily parasitized than individuals with lower heterozygosity (pathogen-mediated selection through over-dominance; Oliver et al. 2009). Indeed, a recent study investigating the genetic effects of several immune gene loci (including the five studied here) on pathogen infestation in the same roe deer population reported that heterozygous individuals at TLR3 were less likely to be infected by *Toxoplasma gondii* than homozygous ones (Fernandez 2013). However, further studies are required to confirm that pathogen-mediated selection occurs through over-dominance for all or part of the five immune gene loci we studied.

Our findings nicely fit with the associations among nematode abundance, body mass and dispersal propensity recently reported by Debeffe et al. (2014) in this roe deer population. These authors found that dispersal propensity generally decreased with both increasing nematode abundance and with decreasing body mass; however, individuals that were both very light and heavily parasitized also had a high propensity to disperse. Since dispersal is costly in terms

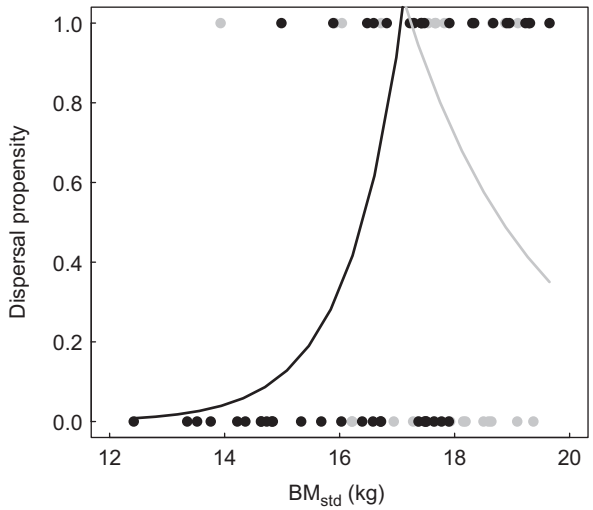

Figure 3. Dispersal propensity of roe deer juveniles (n = 71) as a function of standardized body mass (in kg) (labelled BM_{std}) for heterozygous (black circles) and homozygous (grey circles) individuals at TLR4. Note: the black and grey lines represent the relationships predicted by the selected model ($BM_{std} + H_{O(TLR4)}$) for heterozygous and homozygous individuals at TLR4, respectively.

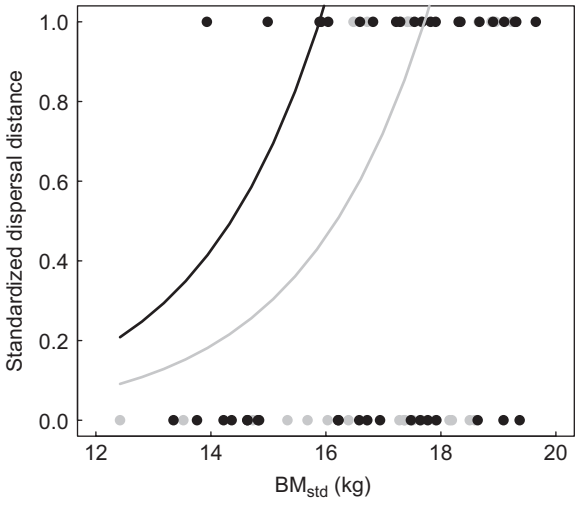

Figure 4. Individual-based standardized dispersal distance (labelled standardized dispersal distance) of roe deer juveniles (n = 71) as a function of standardized body mass (in kg) (labelled BM_{std}) for heterozygous (black circles) and homozygous (grey circles) individuals at TLR3. Note: the black and grey lines represent the relationships predicted by the selected model ($BM_{std} + H_{O(TLR3)}$) for heterozygous and for homozygous individuals at TLR3, respectively.

of energy, time, risk and opportunity, and since dispersers are expected to be exposed to a broader range of infectious agents than philopatric individuals (Snoeijs et al. 2004), only high quality individuals with high resistance to pathogens and high heterozygosity may be capable of dispersing successfully, suggesting a mechanism for the occurrence of condition-dependent dispersal (Bonte and de la Peña 2009). However, for poor quality individuals that are both light and have very low levels of heterozygosity with poor resistance to pathogens, natal dispersal may be a way to escape heavily infested environments, constituting a 'leave-it' emergency life-history tactic (cf. the 'take-it-or-leave-it' emergency life-history tactics defined by Wingfield 2003). These 'take-it-or-leave-it' tactics are behavioural responses to long-term perturbations of the physical environment (such as major weather changes, increased predation risk or increased pathogen infestation), which are mediated by the hypothalamo–pituitary–adrenal cortex and which serve to enhance Darwinian fitness by redirecting the individual away from its normal life-history stages into survival mode until the perturbation passes (Wingfield 2003). Alternatively, if we make the reasonable supposition that roe deer individuals with low levels of heterozygosity are also the most heavily infested, the high dispersal propensity among low quality individuals with a low level of heterozygosity at immune genes may indirectly result from manipulation of the host's dispersal behaviour by pathogens (i.e. pathogen-mediated dispersal; Moore 2002, Nunn et al. 2008). However, we lack evidence for a direct link between nematode abundance and heterozygosity at immune gene loci in this species, although preliminary results suggest that a dominant allele effect may occur for TLR4 and nematode abundance in the Aurignac population (Fernandez 2013).

To date, few studies in natural populations of birds or mammals have investigated the association between immune gene variation and phenotypic traits whose expression may be influenced by pathogens. In the ring-necked pheasant *Phasianus colchicus*, von Schantz et al. (1997) provided evidence for an association between MHC genotype and a condition-dependent selected male trait (spur length). Spur length was, in turn, positively correlated with age, body size and viability in males, although no direct link was inferred between disease resistance and spur length or MHC haplotypes. These authors also showed female mate preference for males with long spurs, which may result in improved chick survival. In white-tailed deer, Ditchkoff et al. (2001) detected associations between some allelic combinations at the MHC-DRB locus with antler development and body mass in adult males, and found a negative relationship between the degree of antler development and abundance of abomasal helminths. They suggested that antler development could therefore provide an honest signal of a male's genetic quality, health and body condition.

Since multi-locus heterozygosity is often assumed to be a good proxy for the level of genome-wide inbreeding (Jensen et al. 2007), the increase in dispersal propensity with increasing individual genetic diversity at immune genes that we observed for most individuals may be explained by a negative effect of inbreeding depression. Through an increased expression of recessive deleterious alleles and the loss of heterozygote advantage, inbreeding may indeed result in a decrease in the dispersal capacity of individuals of low genetic diversity, as found in great tits (Szulkin and Sheldon 2008). In addition, the fact that the lightest individuals with low genetic heterozygosity actually dispersed more than expected could indicate inbreeding avoidance. Dispersal may indeed benefit individuals with low genetic diversity by reducing the likelihood that they mate with closely related kin, so increasing fitness through heterosis and by purging deleterious mutations (Shafer et al. 2011). However, we did not find any clear effect of heterozygosity at immune gene loci on dispersal distance, whereas the benefits of inbreeding avoidance decrease very quickly at relatively short distances from the natal area (Szulkin and Sheldon 2008). Furthermore, the relationship between dispersal propensity and heterozygosity at immune gene loci was stronger when considering non-synonymous SNPs only than when considering all SNPs to reconstruct haplotypes. Note also that dispersal propensity was not affected by heterozygosity measured at synonymous SNPs only (Supplementary material Appendix 1 Table A6, A8). This suggests that the effects of immune gene diversity on dispersal behaviour that we observed were due to the functional role of the five immune gene loci we used, and not simply to the genome-wide level of inbreeding. Finally, all individuals used in this study were also genotyped at a set of 12 presumably neutral and non-linked microsatellites to provide an evaluation of the background level of neutral genetic variation for comparison with immune genes (Vanpé et al. 2015). Based on these data, we recently showed that individual heterozygosity at these putatively neutral microsatellite markers had no effect on dispersal propensity and distance in three contrasted populations of roe deer, including the population studied here (Vanpé et al. 2015). If we assume that our measure of individual heterozygosity at 12 microsatellite loci reliably reflects the level of inbreeding (but note that we did not find significant statistical support for identity disequilibrium with these 12 microsatellites: $n = 77$, $g_2 \pm SD = 0.011 \pm 0.011$, $p = 0.09$; see Vanpé et al. 2015 for more details), we can conclude that inbreeding depression and avoidance are unlikely to explain the link we report here between individual heterozygosity at immune genes and dispersal propensity. In addition, this suggests that our findings are specific to the immune genes studied and are not due to a genome-wide effect.

We found a clear effect of heterozygosity on dispersal propensity, but not on dispersal distance. This means that high quality individuals with high resistance to pathogens and high levels of heterozygosity at immune genes were generally more likely to disperse, but may not disperse over greater distances, compared to low quality individuals with low resistance to pathogens and low heterozygosity. This is also in agreement with the recent results of Debeffe et al. (2014) who found that nematode abundance affected dispersal propensity, but not dispersal distance. One potential explanation is that the distance that an individual has to travel to modify its environment in terms of local pathogen prevalence differs markedly from one individual to another, especially in a heterogeneous agricultural landscape such as our study site.

To conclude, our findings demonstrate that dispersal propensity can be affected by the diversity of genes involved in immune recognition. In roe deer, this effect is dependent on individual quality, suggesting that dispersal propensity

is driven by two different mechanisms, and likely linked to pathogen-mediated selection through over-dominance. Whereas dispersal of high quality individuals appears to be purely condition-dependent such that deer with higher body mass and higher levels of immune gene heterozygosity disperse more often, dispersal among poor quality individuals may be governed by a 'leave-it' emergency life-history tactic where light, homozygous individuals with poor resistance to pathogens also disperse so as to escape heavily infested environments. Further studies are needed to evaluate the potential consequences of these findings in terms of population genetics, demography and host–pathogen evolution.

Acknowledgements – We thank the local hunting associations, the Fédération Départementale des Chasseurs de la Haute Garonne, as well as numerous co-workers and volunteers for their assistance during roe deer capture.
Funding – CV and LD were funded by the 'PATCH' RPDOC ANR project (ANR-12-PDOC-0017-01) attributed to CV from the French National Research Agency. This study was supported by the 'PATCH' RPDOC ANR project, the 'INDHET'ANR project (ANR-12-BSV7-0023-02) and the French Natl Inst. for Agricultural Research (INRA).

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Supplementary material (available online as Appendix oik-02904 at <www.oikosjournal.org/appendix/oik-02904>). Appendix 1.