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LETTER

The role of maternally transferred antibodies in maternal performance in red deer

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Abstract

Maternal effects are ubiquitous. Yet, the pathways through which maternal effects occur in wild mammals remain largely unknown. We hypothesise that maternal immune transfer is a key mechanism by which mothers can affect their offspring fitness, and that individual variation in maternally derived antibodies mainly depends on a mother's characteristics and the environmental conditions she experiences. To test this, we assayed six colostrum-derived antibodies in the plasma of 1447 neonates in a wild red deer population. Neonatal antibody levels were mainly affected by maternal genes, environmental variation and costs of prior reproductive investment. We found consistent heterogeneity in maternal performance across traits, with mothers producing the heaviest calves also having calves with more antibodies. Unexpectedly, antibody levels were not associated with calf survival. We provide a unique example of how evolutionary theory on maternal effects can be used to gain insight into the causes of maternal effects in wild populations.

KEYWORDS

ecological immunology, individual quality, mammal ecology, maternal effects, maternal immune transfer, maternal investments, natural population, trade-off, viability selection

INTRODUCTION

Mothers strongly influence the environmental conditions experienced by their offspring during development (pre- and post-natal) and, consequently, they often have profound consequences for offspring phenotype and fitness (Bernardo, 1996; Moore et al., 1997; Mousseau & Fox, 1998). These maternal effects are an important source of phenotypic variation in nature (Moore & Kruk, 2002) and arise from both environmental and genetic variation (Räsänen & Kruuk, 2007). Despite increasing interest in maternal effects, and the role they can play in ecological and evolutionary processes, we still know very little about the traits that might mediate these effects. This is especially true in wild mammals,

because of the difficulties of identifying relevant maternal traits and phenotyping free-living individuals without disturbance. Resource allocation by the transfer of nutrients, hormones and antibodies is a key pathway through which mothers can affect their neonates, and so present a promising set of traits with which to characterise maternal reproductive investment (Mousseau & Fox, 1998) and gain insight into how maternal effects arise in nature (Grindstaff et al., 2003; Roth et al., 2018).

In vertebrates, neonates are born with an immature immune system and their immune defence depends on the transfer of antibodies (or immunoglobulins; Ig) from immunocompetent mothers through the placenta, milk or yolk (Boulinier & Staszewski, 2008; Hasselquist & Nilsson, 2009). An effective immune response is critical

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for protection against parasites and pathogens but can come at a cost to an individual's reproduction and development (Garnier et al., 2017; Lochmiller & Deerenberg, 2000; Schmid-Hempel, 2003). Positive effects of maternally transferred antibodies on offspring growth, health and survival have been reported in domestic species and experimental populations (e.g. Lora et al., 2018 in cows, Martyka et al., 2018 in birds), and in wild mammal populations (e.g., Marchandeau et al., 2014 in rabbits, Gomez-Chamorro et al., 2019 in bank voles, Sparks et al., 2020 in Soay sheep). This effect is often interpreted as a direct beneficial effect of passively acquired immunity for neonates (transgenerational immune priming; Hasselquist & Nilsson, 2009; Roth et al., 2018). An effective protection also allows neonates to limit their investment in immunity and devote energy to rapid growth (Hasselquist & Nilsson, 2009; Lochmiller & Deerenberg, 2000). The ability of mothers to transmit their antibodies can thus be an important source of variation in offspring traits. Yet, causes of individual variation in maternally transferred immune traits have very rarely been characterised in the wild, especially in mammals (but see Sparks et al., 2020).

Variation in the amount of antibody transferred from mothers to their neonates is multicausal. In ruminants for instance, colostrum is the main source of circulating antibodies in neonates (no evidence of placental immune transfer in domestic ruminants because of their endotheliochorial placenta; Butler & Kehrli, 2005). After the colostrum stage, maternally derived antibodies can no longer reach the calf's bloodstream, but they may have a local effect in the intestinal tract. Individual variation in neonates' circulating antibody levels could either be due to: (1) variation in antibody concentrations circulating in mother's serum close to calving, that should mainly reflect on the mother's body condition and past exposure to pathogens and parasites; (2) variation in the mother's ability to actively transfer antibodies from serum to colostrum; (3) variation in the mother's ability to locally produce lacteal immunoglobulins; (4) variation in the ability of offspring to absorb them (Boulinier & Staszewski, 2008; Butler & Kehrli, 2005). These physiological processes, which depend on a mother's and a neonate's characteristics, as well as environmental factors, critically determine the success of transferring passive immunity.

Several environmental factors have been identified as potentially important for the transfer of antibodies, such as the effect of maternal condition and age, nutrition, breeding density and local disease environment (reviewed in Boulinier & Staszewski, 2008; Grindstaff et al., 2003). In birds, higher breeding density and the presence of parasites are associated with higher antibody concentration in eggs (e.g. Gasparini et al., 2001), but these effects have never been reported in other taxa. Reproduction can come with high costs for mothers. Past reproductive effort might therefore affect maternal

investment in transfer of antibodies. In mammals, lactation is the phase of reproduction requiring highest resources (Clutton-Brock et al., 1989; Froy et al., 2016). In resource-limited environments, mothers might invest less in immune defence during pregnancy, because of a trade-off between investment in different functions (immunocompetence, foetus growth, milk production). However, this hypothesis remains largely untested (see Landete-Castillejos et al., 2002 in captive ruminants). Moreover, trade-offs in performance and fitness-related traits are notoriously difficult to detect in the wild, due to heterogeneity in individual quality (Wilson & Nussey, 2010). Finally, genetic differences might explain part of the variation in maternally derived immune traits (as suggested by studies in domestic animals; e.g. Dardillat et al., 1978; Bumstead et al., 1993; Cordero-Solorzano et al. in preparation), with consequences for a population's evolutionary dynamics (Kirkpatrick & Lande, 1989). To date, no study has investigated the relative influence of these genetic and environmental factors on maternal immune transfer in a wild population (see Ruuskanen et al., 2016 for a study in a wild bird reared in captivity).

Studies that have investigated the consequences of maternal immune transfer for offspring traits and fitness have not related these effects to maternal effect theory (as highlighted by Grindstaff et al., 2003). Consequently, we have so far ignored how much maternal effect variance for an offspring trait can be explained by variation in maternal investment in immunity (Moore et al., 2019). The quantitative genetics theory of maternal effects usually treats maternal effects as a general feature of the mother, known as 'maternal performance' (Willham, 1963, 1972). Therefore, we often have little insight into which traits are relevant to characterise maternal performance, except in a few cases, such as egg size in birds (Hadfield et al., 2013; Pick et al., 2016), or litter/clutch size in mammals and reptiles (McAdam & Boutin, 2004; Noble et al., 2014). Here, we hypothesise that maternal antibody transmission, while being a trait in which we can measure maternal performance, is also a good candidate with which to characterise maternal performance for key offspring traits such as survival in mammals.

In the present study, we use a long-term individual-based study to investigate the causes and consequences of variation in maternally transferred antibodies in a wild red deer population (*Cervus elaphus*). In domestic ruminants, IgG is the main antibody isotype found in serum and milk, representing 70–80% of the colostrum protein content (Korhonen et al., 2000). Other isotypes, such as IgA and IgM, which play an important role in the intestinal protection of neonates during and after the colostrum stage, are found at lower concentrations (Butler & Kehrli, 2005). We measured total levels and levels against a specific antigen of three isotypes (IgG, IgA and IgM) in plasma samples of 1447 red deer calves. We address three major questions: First, what are the sources of (co)

variation in maternally derived antibody? We focus on the effects of mothers' characteristics (environmental, genetic, reproductive status) in affecting antibody levels measured in neonates; second, to what extent are differences in antibody transfer between mothers associated with other aspects of maternal performance, namely offspring birth weight?; and third, is maternal immune transfer a causal pathway explaining maternal effects for juvenile survival? To answer these questions, we use models derived from maternal effect theory (Hadfield, 2012; McAdam et al., 2014) and apply them for the first time to a wild system.

MATERIAL AND METHODS

Study population, individual monitoring and pedigree

The red deer living in the population in the 12 km² North Block of the Isle of Rum (Inner Hebrides, Scotland; 57°03'N, 06°21'W) have been intensively monitored since 1972. Regular censuring and mortality searching throughout the year provide close monitoring of deaths within the study area. During the calving season (May–June), all pregnant females are monitored daily to identify when and where calves are born. Calves are caught soon after birth (often within 24 h) to be weighed, measured, tagged and sampled. Age at capture is estimated based on several field insights. Some births are observed, but otherwise observations of the mother (changes in shape, absent from her regular group of hinds) and the calf (how well the calf moves when first observed, inspection of the umbilicus and the hooves at capture) are used to estimate age at capture as accurately as possible. During the calving season a large field team is deployed so that the entire study area is well monitored and variation in the estimation of age at capture is minimised and likely random across calves. Culling has not occurred in the study area since 1973 and therefore mortality mainly occurs from natural causes. Since 1993, jugular blood samples have been collected from neonates. Samples were centrifuged at 10,000 g for 10 min and the resulting plasma was aliquoted and stored at –20°C until measurement of immunoglobulin levels in the plasma (see below).

All sampled individuals within the study area have been genotyped on the Illumina Cervine 50K SNP array. These genomic data were used for pedigree inference with the R-package SEQUOIA (Huisman, 2017). The resulting multigenerational pedigree has already been used to decompose the genetic and environmental sources of variation in quantitative traits in previous studies (Gauzere et al., 2020; Huisman, 2017). For the traits expressed early in life, there is significant maternal effect variance, explaining 35% of the total phenotypic variance in birth weight and 2–3% in juvenile survival

(Gauzere et al., 2020; Kruuk & Hadfield, 2007; Walling et al., 2014).

Measurement of immunoglobulin levels

Immunoglobulins (Ig) were assayed from plasma using ELISA. We used the optimised antibody assays developed in Soay sheep (Nussey et al., 2014; Sparks et al., 2018), and validated in red deer (Albery et al., 2020), to quantify the concentration of three different isotypes, IgA, IgM and IgG, for a parasite-specific antigen (anti-*Teladorsagia circumcincta*) and their total levels (anti-*Tc* Igs are a component of total Igs). Anti-*Tc* antibodies show a high cross-reactivity with other strongyles (Froy et al., 2019); their variation can therefore be interpreted as a general anti-strongyle response (Albery et al., 2020). All antibody concentrations were recorded as optical density (OD) values measured by spectrophotometer (Thermo Scientific Multiskan GO Spectrophotometer). All samples were randomised across plates, with each sample duplicated, and each plate included two negative controls (i.e. sample free wells) and two positive controls (serum from an infected sheep). For each sample, we calculated a standardised OD:

$$OD_{sd} = \frac{\text{sample OD} - \text{negative control OD}}{\text{positive control OD} - \text{negative control OD}}$$

We analysed the average OD_{sd} values between the two repeated samples as the neonatal immune trait. In total 1646 calves were measured for six different antibody levels. See Part S1 in Supporting Information for more details about the ELISA assay protocols. We analysed data from 1993 to 2017.

We failed to detect total IgG in stillborn calves (Figure S1), as expected because of the endotheliochorial placenta in ruminants. Therefore, all the antibodies circulating in the neonates' blood originate from passive immunity and the absorption of antibodies from the colostrum. In domestic ruminants, the gut wall becomes impermeable to antibodies around 48 h after birth (Butler & Kehrl, 2005). The observed variation in antibody levels with the age at which a calf was captured is consistent with these expectations, with an increase in circulating immunoglobulins up to about 50 h and a decline afterwards (because of gut closure and catabolism of absorbed immunoglobulins; Figure S2).

Model for variation in maternally transferred antibodies

We investigated the causes of variation in the six assayed maternally transferred antibody measures. To do this we used a univariate mixed model framework and pedigree information allowing us to decompose the contribution

of calf's own genes (a ; direct genetic effects), maternal genes (mg ; maternal genetic effects), maternal environment effects (me) and cohort effects (c) on the phenotypic variation of a quantitative trait y . The general matrix form of this model is:

$$y = Xb + Z_1a + Z_2m_g + Z_3m_e + Z_4c + e \quad (1)$$

with y the phenotypic observations, b the vector of fixed effects fitted in the model and e the vector of residual error. The design matrices X and Z link the individual observations to the relevant fixed and random effects.

Models for maternally derived antibodies accounted for the effect of calf sex (male or female) and age at capture (in hours; linear and quadratic effects) as fixed terms. We also considered some effects likely to be related to maternal condition, such as maternal age (in years; linear and quadratic effects) and maternal reproductive status. Depending on their breeding status in the previous year, females can be categorised as (1) 'milk' when a female calved and the calf survived to at least 1 May the year after the birth, (2) 'winter yield' when she calved and the calf died during the winter after birth (between 1 October and 1 May), (3) 'summer yield' when she calved and the calf died during the summer (before 1 October), (4) 'true yield' when a female did not calve and (5) 'naive' if she had never calved (following Stopher et al., 2012). We also accounted for the effect of the birth region and local population density (following Gauzere et al., 2020). The study area was divided into six regions (Shamhnan Insir 'SI,' Intermediate area 'IM,' Laundry 'LA,' North glen 'NG,' Mid glen 'MG' and South glen 'SG') and calf birth locations were attributed to one of these areas using the mother's census locations. Population density was calculated as the number of females sharing the same region during spring in the calf's year of birth.

Following previous analyses on neonatal traits, we only analysed the immune traits of calves caught within 7 days of birth and born before 1 August (Gauzere et al., 2020; Huisman et al., 2016). We also excluded stillborn calves ($n = 10$), calves thought not to have suckled before capture ($n = 12$) and calves that produced very haemolysed samples ($n = 9$), all of which had almost null immunoglobulin levels (Figure S1). A total of 1447 calves, born to 463 different mothers, with non-missing information for the fixed and random effects defined in model (1) were analysed (Table S1).

We estimated the proportion of total phenotypic variance explained by each random component fitted in model (1) to compare their relative contribution to individual variation in maternally transferred immune traits. The maternal repeatability of the trait was estimated as the proportion of total phenotypic variance explained by maternal identity, testing for consistent differences between mothers in a model that only included maternal identity and cohort as random effects. Finally, we ran a multivariate analysis of all six immune

traits together to measure their phenotypic covariances, testing whether these covariances were due to consistent differences between mothers (using the same fixed effects as above). These models were fitted using the software AsReml-R v3 (Gilmour et al., 2006), assuming Gaussian error distributions. We tested the significance of each fixed term in the model using a Wald conditional test.

Models of maternal effects

Quantitative genetic theory offers two main frameworks to study maternal effects, called the 'variance partitioning' and 'trait-based' approaches (Kirkpatrick & Lande, 1989; Willham, 1963). The 'variance partitioning' approach treats maternal effects as a general feature of the mothers and separates the phenotypic variance in an offspring trait explained by direct genetic effects and indirect effects due to differences between mothers (maternal performance), whereas the 'trait-based' approach focuses on the strength of potentially causal relationships between maternal traits and offspring traits. Here, we build on previous work (Hadfield, 2012; McAdam et al., 2014) and apply a 'hybrid' strategy that lies between these two approaches. Typically, univariate models are used to implement this hybrid approach (Noble et al., 2014; Pick et al., 2016), but here we develop a multivariate approach to analyse the sources of covariance between a specific neonatal circulating antibody and offspring trait(s).

We focussed on three different offspring traits y , birth weight (in kg), neonatal survival from birth in May/June to 30 September (0/1; about 11% of calves die in this period) and first winter survival from 1 October to 1 May the following year (0/1; another 30% of calves die in this interval; 36 shot calves were excluded from this analysis). We directly modelled the trivariate association between a focal neonatal antibody level, weight and neonatal or first winter survival, partitioning the phenotypic (co)variances into total maternal effects, cohort effects and residual effects. The outputs of this model were then treated in two different ways depending on our hypotheses.

Association between Ig levels and birth weight: Neonatal morphological traits usually display the largest maternal effect variance (Gauzere et al., 2020; Moore et al., 2019), and are thus relevant to characterise the maternal reproductive investment. In ruminants, variation in maternally transferred immune traits cannot be causally linked to birth weight as immune transfer occurs after birth (Schultz et al., 1971). However, any (negative) association between these two traits might reveal short-term trade-offs between these different kinds of maternal investment. Such trade-offs are notoriously difficult to demonstrate, especially when heterogeneity in individual performance exists. The benefit of our approach

is that it allows us to test for the correlation between Ig and birth weight at different hierarchical levels (e.g. mother, cohort) and to identify such trade-offs while accounting for variation in between-mother performance (see more details in Part S2).

Association between Ig levels and survival: We predicted a causal association between neonatal Ig levels and a calf's probability of survival. To test this hypothesis, we estimated and compared the between- and within-mother effects, that is, the regression coefficients of survival on Ig estimated at the maternal and residual levels (following Froy et al., 2019). The benefit of our multivariate approach is that it takes into account uncertainty in the estimation of these effects (Phillimore et al., 2010), making it superior to other proposed methods for testing causality (van de Pol & Wright, 2009). Similar between- and within-mother slopes are indicative of causation, rather than another maternal trait causing the correlation (Hadfield et al., 2013; Pick et al., 2016). We accounted for potential indirect effects of Ig on survival arising through their association with birth weight when estimating regression slopes (see Part S2).

We analysed the causes of variation in antibody levels and capture weight assuming a Gaussian error distribution, and juvenile survival assuming a binomial error distribution (logit link function) using the Markov Chain Monte Carlo method implemented in the R-package MCMCglmm v.2.29 (Hadfield, 2010). We included as fixed effects the covariates and factors known to be important for each offspring trait (Gauzere et al., 2020; Huisman et al., 2016). For the trait(s) for which it was relevant, we further decomposed maternal effects into maternal genetic and maternal environmental effects.

RESULTS

Non-genetic predictors of neonatal circulating immunoglobulins

Almost all maternally transferred Igs were affected by the same fixed factors and covariates (Table S2). We found that all maternally transferred immune traits, except anti-*Tc* IgG, were affected by maternal reproductive status and the region of birth. Specifically, neonatal Ig levels were higher in calves of mature females which had not calved the year before (Figure 1), and Ig levels also tended to be higher in calves of females which calved the year before but lost their calf during summer ('summer yield' status). The neonatal Ig levels were consistently higher in the glen than in the rest of the study area (Figure 1; Figure S3). This spatial pattern matched the spatial variation in birth weight (Figure S4). Calves born in the region with the highest density ('SI') were also the lightest and had the lowest circulating Ig levels. Therefore, spatial variation in neonatal immune traits most likely reflects differences in habitat quality. The year-to-year variation in local population density had no significant effect on antibody levels. We found that male calves had lower anti-*Tc* IgG levels (Table S2). A significant linear effect of maternal age on anti-*Tc* IgM was also detected, with calves of older mothers having lower Ig levels.

The six maternally transferred immune traits analysed were all highly positively correlated at the phenotypic level, with a particularly strong correlation among the anti-*Tc* and total levels for IgA and IgM ($R > 0.82$; Table 1). Overall, covariances between immune traits were mostly due to within-mother effects, that is, factors varying throughout a female's life (e.g. temporal environmental variation). At the between-mother level, only the amount

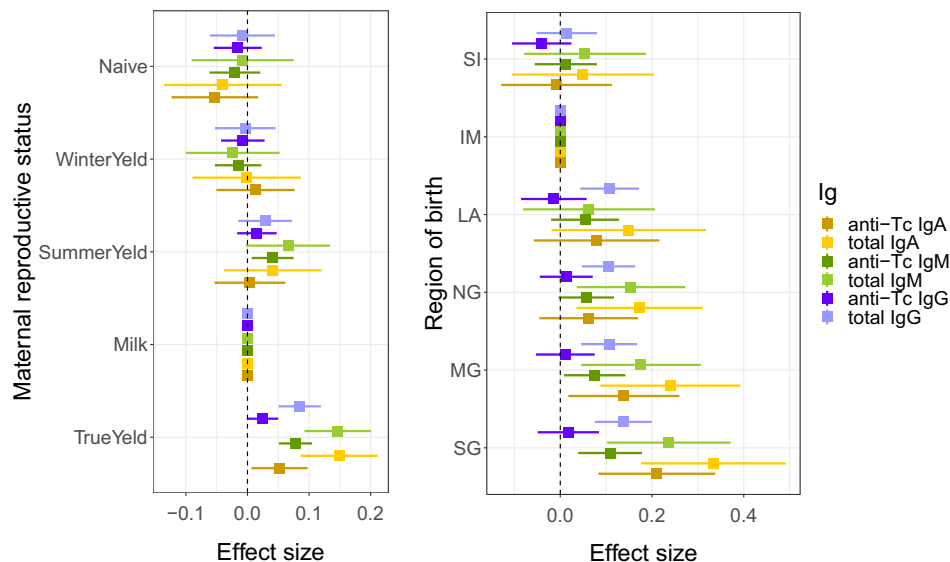


FIGURE 1 Effect of the region of birth and maternal reproductive status on maternally transferred antibody levels (point estimate and 95% confidence intervals of the effect size). These two predictors had very consistent effects on the immune traits. Offspring of mature females, which had not calved the year before ('true yield'), and of females inhabiting the glen (North glen NG, Mid glen MG and South glen SG) had higher antibody levels

TABLE 1 Correlations between the six neonatal immune traits estimated at the between-mother (maternal effect) level (lower diagonal) and within-mother (residual) level (upper diagonal)

	Anti- <i>Tc</i> IgA	Total IgA	Anti- <i>Tc</i> IgM	Total IgM	Anti- <i>Tc</i> IgG	Total IgG
Anti- <i>Tc</i> IgA		0.82 (0.01)	0.69 (0.02)	0.61 (0.02)	0.60 (0.02)	0.71 (0.02)
Total IgA	0.76 (0.03)		0.82 (0.01)	0.77 (0.01)	0.71 (0.02)	0.86 (0.01)
Anti- <i>Tc</i> IgM	0.00 (0.07)	-0.03 (0.07)		0.89 (0.01)	0.67 (0.02)	0.77 (0.01)
Total IgM	-0.04 (0.07)	-0.08 (0.07)	0.88 (0.02)		0.64 (0.02)	0.74 (0.01)
Anti- <i>Tc</i> IgG	0.04 (0.06)	0.06 (0.07)	0.03 (0.07)	0.07 (0.07)		0.71 (0.02)
Total IgG	0.23 (0.10)	0.36 (0.10)	0.37 (0.10)	0.37 (0.10)	0.22 (0.11)	

These correlations (point estimate and standard error) were estimated from a model that only included maternal effects as a random component and included the same fixed effects as in the univariate model eq. 1. Significant terms are highlighted in bold.

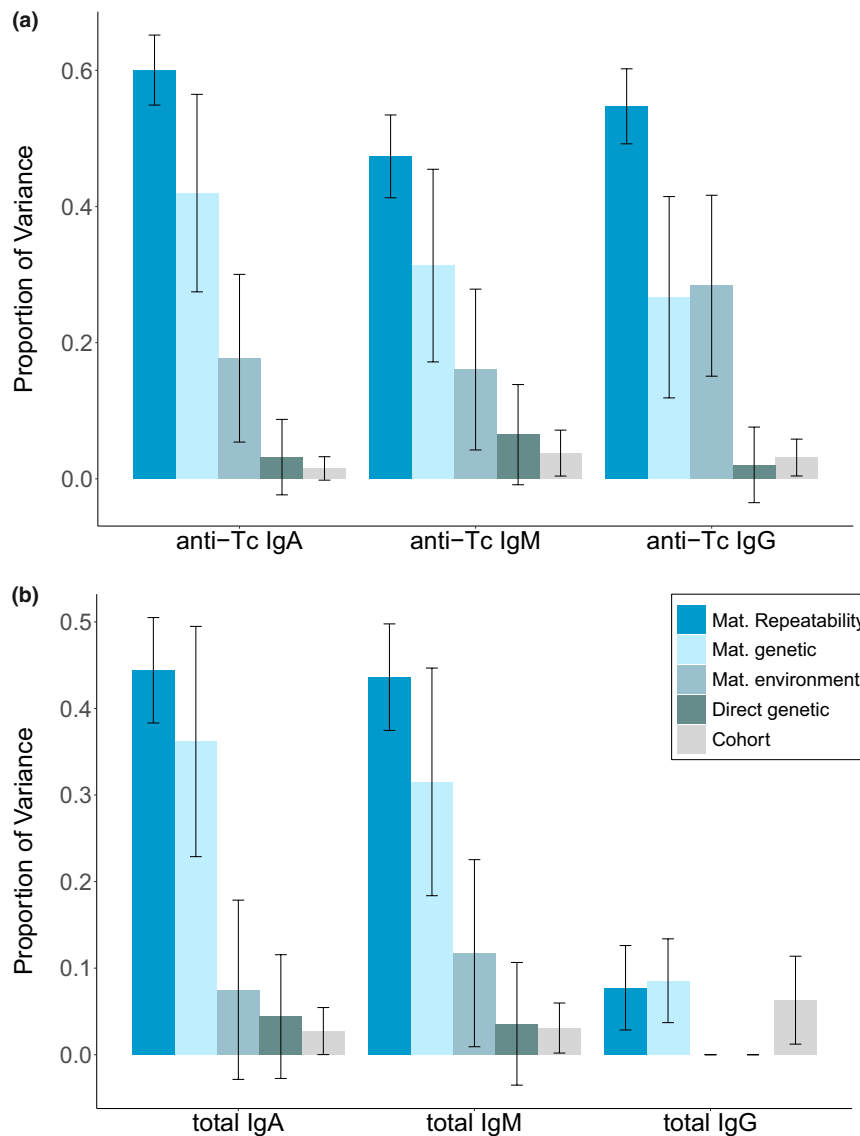


FIGURE 2 Proportion of total phenotypic variance explained by maternal genetic effects, maternal environment effects, direct genetic effects (calf's genes) and cohort effects (model eq. 1) for the three isotopes of a parasite-specific antigen (anti-*Tc*; panel a) and their total levels (b). The maternal repeatability was estimated as the proportion of variance explained by consistent differences between mothers, encompassing their genetic and environmental effects. The cohort variance represents any year-to-year variation in environmental quality not captured by our fixed effects. Error bars represent the 95% confidence intervals around the point estimate

of maternally transferred total IgG was associated with higher levels for other antibodies (Table 1).

Genetic architecture of maternally transferred immune traits

The amount of antibody measured was repeatable among mothers (Figure 2). The variance in neonatal antibody levels due to consistent differences among mothers explained between 44% and 60% of the total phenotypic variance for all traits, except total IgG, for which only 8% [3%; 13%] (point estimate and 95% confidence intervals) of the variance was explained. We found no evidence that the calf's own genes affected the phenotypic variance in neonatal Ig levels (Figure 2; Table S3), which suggests no direct genetic effects. The only significant additive genetic effects detected were due to the mother's genes. This maternal genetic effect variance explained between 26 and 42% of the total phenotypic variance in anti-*Tc* IgA, IgM and IgG and between 9 and 37% of total phenotypic variance in their total levels. Therefore, maternal genetic effects explained most of the maternal repeatability of the traits (Figure 2).

For anti-*Tc* IgA, IgM and IgG, we also found significant variance in maternal environment effects, with 16 to 29% of phenotypic variance explained by these non-genetic differences between mothers (Figure 2a; Table S3). Maternal environment effect variance was lower for the total Ig levels, being significant only for total IgM, and non-significant to negligible for total IgG and total IgA (Figure 2b). Finally, the calf cohort effect explained a small to negligible proportion of total phenotypic variance for the six maternally transferred immune traits, with 2–6% of variance explained. Among all the maternally transferred immune traits, total IgG had the lowest proportion

of variance explained by the included random components, with only 15% of the phenotypic variance explained while we explained more than 51% of the other traits (Sparks et al., 2020 also reported a low repeatability for total IgG).

The role of maternally transferred antibodies in maternal performance

Covariance between maternally transferred immune traits being mostly independent among mothers (Table 1), we investigated the consequence of variance in each trait independently. We detected a significant positive association between all antibody levels and birth weight, except for anti-*Tc* IgA (Figure S5, see also Part S2). This association was driven by a significant positive correlation between birth weight and two isotypes, IgM and IgG (for both their antigen-specific and total levels), at the between-mother level, and a significant positive correlation between total IgA, total IgG, and (anti-*Tc* and total) IgM and birth weight at the within-mother level (Figure 3; Figure S5). At the between-mother level, the strongest association was found between birth weight and total IgG, with a correlation of 0.40 [0.16; 0.68] (mode and 95% credible intervals). Correlations at the within-mother level were usually lower than those estimated at the between-mother level. The maternally transferred immune traits and birth weight were never associated at the cohort level (Figure 3; Figure S5). From the same models, we calculated the regression slopes of the effect of survival on neonatal antibody levels. These regression coefficients were never significantly different from zero, indicating no association between maternally transferred immune traits and neonatal or overwinter survival (Figure 4; Figure S5).

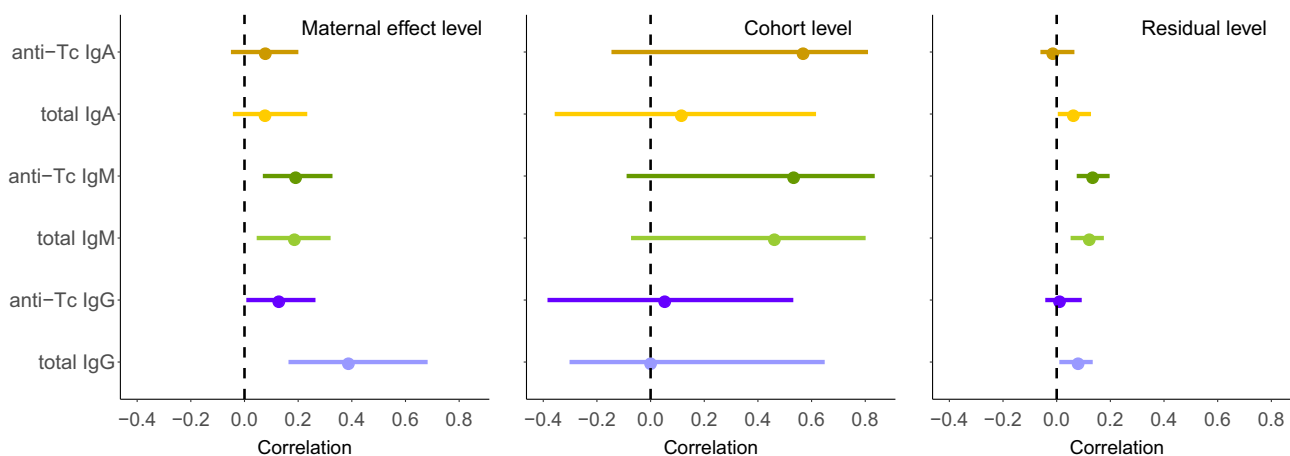


FIGURE 3 Correlations between maternally transferred antibodies and birth weight estimated at different hierarchical levels (one panel per level). The point estimates and 95% credible intervals are represented. Associations between maternal immune transfer and birth weight were due to within-mother correlation (residual level) and between-mother correlations (maternal effect level). We found no significant correlation across years (cohort level)

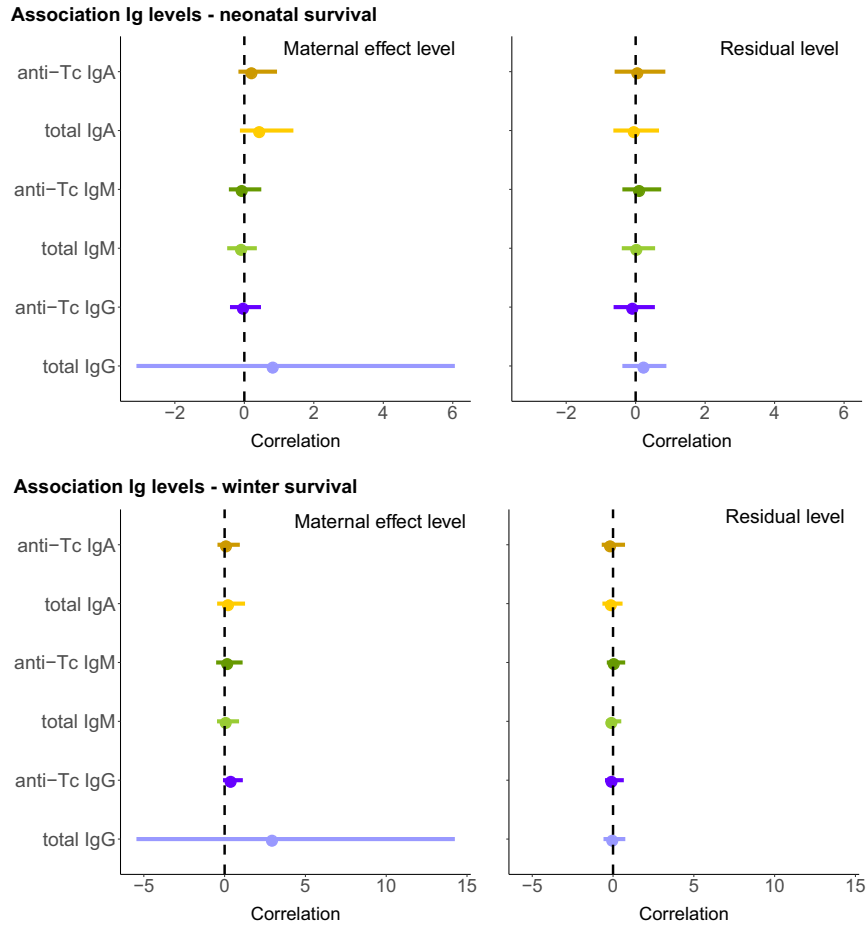


FIGURE 4 Regression slopes of the effect of neonatal antibody levels on survival estimated at different hierarchical levels (point estimates and 95% credible intervals). Causality can be inferred by comparing the within- and between-mother slopes, that is, the slopes estimated at the maternal effect level and residual level with the multivariate hybrid model. We found no significant association between the maternally transferred Ig and offspring survival

Using pedigree information, we further decomposed the maternal covariance between total IgG and birth weight into genetic and non-genetic components. We found that this maternal correlation was only due to genetic effects, with an estimated genetic correlation between these two traits of 0.44 [0.17; 0.84] (Figure 5).

DISCUSSION

Despite a relatively good understanding of the physiology of maternal immune transfer, we are remarkably ignorant about the causes of its variation in natural populations, notably regarding the respective contributions of genetic and environmental effects (Boulinier & Staszewski, 2008; Grindstaff et al., 2003). Most studies interested in maternal immune transfer have focussed on the concentration of total IgG (or IgY in birds), the most abundant maternally transferred antigen, and have paid less attention to variation in antibodies

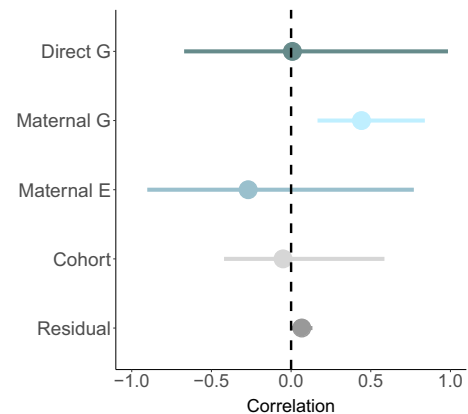


FIGURE 5 Correlation between total IgG and birth weight decomposed over genetic and environmental/residual levels (point estimate and 95% credible intervals). The phenotypic (co)variances of these two traits were decomposed into an effect of direct genetic effects ('Direct G'; i.e. due to calf's own genes), maternal genetic effects ('Maternal G') and maternal environmental effects ('Maternal E'), cohort and residual effects

against specific antigens (but see Gomez-Chamorro et al., 2019). Here, our results showed little difference in the environmental causes of variation according to the type of antibodies. Using data from one of the largest individual-based studies in a wild mammal population, we found high maternal repeatability for the circulating antibody levels in neonates' blood, with about half of the variation being due to consistent differences among mothers in their ability to produce and transmit antibodies (except for total IgG). This result is consistent with what has been found recently in another wild mammal population (Sparks et al., 2020). Most interestingly, we showed for the first time that these maternal effects were mostly due to genetic differences between mothers, which highlights a moderate to high genetic basis and a capacity for these traits to evolve. We found that maternal permanent environmental effects—due to consistent non-genetic differences between mothers—had a higher influence on the antigen-specific antibody levels than on their total levels. This variation in the anti-strongyle response could reflect between-mother differences in their pathogen load (which we do not yet have the data to test comprehensively) or persistent early-life effects. Evidence that the local disease environment affects the amount of antibodies transferred to offspring is commonly reported in cattle, captive animals and bird species (Hasselquist & Nilsson, 2009).

For the first time in a wild mammal, we report that past reproductive effort negatively affected maternal immune transfer. Offspring of mature females, which did not calve the year before, had higher antibody levels than offspring of females which did calve. Neonatal antibody levels also tended to be higher in offspring of females which calved the year before but lost their calves early in the season. This result suggests that the gestation and the duration of lactation have a cost for maternal immune transfer, and perhaps for female immunity in general (as suggested by Albery et al., 2020). In contrast to Sparks et al., (2020), we did not find an effect of maternal age on the amount of antibody transmitted (except for anti-*Tc* IgM). Variation in neonatal antibody levels also showed an interesting spatial pattern. In theory, this spatial pattern can be due to local levels of resource availability and/or variation in pathogen assault provoking enhanced immune responses (Holt & Boulinier, 2005). Previous analyses have shown that spatial patterns of faecal total IgA and parasitism in females are independent in the study area (Albery et al., 2019). Here, we show that the spatial pattern in neonatal antibodies matches the variation in environmental quality (using birth weight as a descriptor of habitat quality), suggesting that resource availability might be the main factor affecting the amount of antibodies transferred to offspring. Overall, our results highlight the main influence of maternal genes, maternal environment effects and mother's condition on neonatal circulating antibody levels, while the calf's own genes had a negligible influence. In contrast, some studies in

domestic mammals have found that the neonate's characteristics and genes can impact passive transfer, for example because of genetic differences in their capacity to absorb antibodies from the milk (Gilbert et al., 1988; Cordero-Solorzano *et al.*, in preparation). The relative influence of mothers versus neonates in determining variation in maternally transferred antibody levels might thus differ across species. Moreover, it might not be possible to fully (statistically) dissociate the influence of offspring and mothers on traits involved in the transfer of nutrients, hormones and antibodies, as such traits almost always involve a maternal–zygotic or maternal–neonate interaction (Grindstaff et al., 2003).

Measuring maternal allocation of resources in large free-living mammals is particularly difficult as compared to oviparous animals. Consequently, characterising maternal performance for offspring traits has remained challenging in wild mammals (see e.g. Krist, 2011; Pick et al., 2016 in birds). Here, we overcame this challenge by using longitudinal data on maternally derived immune traits and a new approach to decompose the sources of covariance between maternal reproductive investments and to identify the pathways through which maternal effects may occur in nature. In doing so, we provide a unique example of how maternal effect theory can be used to better understand how maternal immune transfer contributes to maternal performance. Different approaches have been proposed and used to investigate the potentially causal association between maternal and offspring traits (Hadfield et al., 2013; McAdam et al., 2014; Noble et al., 2014; Pick et al., 2016), but the multivariate hybrid approach we developed is the most flexible, as it allows us to fit different covariances/regression coefficients for different levels of variation. While we had strong expectations about the causal influence of maternal immune transfer on offspring viability (Boulinier & Staszewski, 2008; Grindstaff et al., 2003), we found no association between the maternally transferred immune traits and neonatal or winter survival. This is in contrast to the animal breeding literature in which failure of passive transfer strongly influences offspring health and early-life viability in ruminants (e.g. cows; Lora et al., 2018; Raboisson et al., 2016). Explanations include the possibility that efficient transfer is more strongly selected (and thus less variable) in wild mammals than in domestic animals with veterinary support. The lack of long-lasting effects on offspring viability is perhaps less surprising since winter survival occurs well after gut closure and is more likely to be influenced by the local action of antibodies in the intestinal tract, which we did not measure. However, a very recent study has reported an effect of neonatal anti-*Tc* IgG on winter survival in a wild mammal population experiencing strong pathogen pressure (Sparks et al., 2020). The consequences of maternal immune transfer for offspring traits might thus be species and/or context specific. Testing these hypotheses would require more studies on the variation in

maternally transferred immune traits and juvenile survival at different stages (pre- and post-colostral) in wild animals.

Interestingly, we found a significant positive association between most maternally transferred immune traits and birth weight at both the phenotypic and genetic level. We demonstrated that this association was due to a positive phenotypic correlation both at within- and between-mother levels. Phenotypic correlations between performance traits are rarely investigated in such details (Moyes et al., 2009; Wilson & Nussey, 2010). Nonetheless, no trade-off between maternal performance traits can be detected in the conditions experienced by our study population. Our results particularly highlighted a strong positive maternal correlation between total IgG and birth weight, suggesting that maternal investment into immune transfer and foetus growth—that both happen at the end of the pregnancy (Butler & Kehrli, 2005)—are positively associated. Variation in total IgG might better reflect the overall quality of the colostrum/milk produced by a mother, encompassing both its immunological and nutritional content (Kehoe et al., 2008), and be a proxy for maternal condition that also influences offspring birth weight. Finally, we revealed that this phenotypic association was mostly due to genetic effects, suggesting that maternal performance traits have co-evolved through build-up of linkage disequilibrium or that they involve the same genes and molecular pathways.

Studies aiming to shed light on the pathways through which maternal effects occur in nature are essential to improve models of maternal effects (McGlothlin & Brodie, 2009). For instance, they enable us to explicitly model plasticity of interacting phenotypes (Ramakers et al., 2018) or to account for ‘cascading maternal effects’ (Pick et al., 2019), that occur when a trait causing maternal effects is itself affected by maternal effects. The approach we developed here could be applied to other species and traits potentially underlying maternal effects. The development of non-invasive methods to measure antibody levels (e.g. from faecal samples; Watt et al., 2016) opens exciting avenues to track both the amount of circulating antibodies in the mother and the calf, which would lead to better characterisation of mother–calf interactions, the rate of catabolism of antibodies, and the post-colostral effects of the maternal immune transfer on offspring traits.

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AUTHORSHIP

JMP and CAW designed the research. SM and AM collected field data. KW and PJ performed the laboratory work and QC of the antibody data. JG, CAW, JLP and JMP discussed and interpreted the findings. JG ran the analyses and wrote the first draft of the manuscript. JLP and CAW helped with the statistical analyses. All authors contributed to write the manuscript.

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DATA AVAILABILITY STATEMENT

The data used in this paper have been deposited on Figshare, <https://doi.org/10.6084/m9.figshare.14541030.v1>.

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