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Feeding innovations and parasitism in birds

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The rate of behavioural innovation, such as opportunistic feeding innovation, may facilitate adaptation to novel environments. Because parasites may affect how their hosts adopt novel means of resource acquisition, or because opportunistic behaviours may involve the risk of being exposed to a large parasite fauna, we hypothesize an evolutionary link between the rate of feeding innovations and parasitism. We investigated the phylogenetic relationship between relative frequency of feeding innovations (adjusted for research effort and population size) and relative size of immune defense organs (as a relative measure of parasite-mediated selection) and the prevalence of blood parasites in birds. Using generalized least squares models, we found that species with relatively large bursa of Fabricius, thymus, and spleen had higher rates of feeding innovations than species with small immune defense organs. Similarly, there was a positive interspecific association between feeding innovation and haematozoa prevalence. These relationships were not confounded by migration, relative brain size, geographical distribution, and male plumage brightness. Analyses of causality relying on evolutionary modelling of discrete variables and path analysis suggest that increasing rate of feeding innovation may place species under intense selection due to parasitism. Therefore, behavioural adaptation by feeding innovation seems to have consequences for the coevolutionary arm race between parasites and hosts. *Biological Journal of the Linnean Society*, 2007, **90**, 441–455.

ADDITIONAL KEYWORDS: bursa of Fabricius – cognition – malaria – spleen.

INTRODUCTION

Feeding innovations may allow animals to exploit different kinds of resources, or exploit resources in a novel way. Hence, the frequency of feeding innovation (or a correlate thereof) may not only promote the success of individuals reaching novel environments, but also the ability of individuals to survive in changing environments. The frequency of feeding innovations is determined by cognitive abilities as shown by its association with tool use and learning and with forebrain size in birds and primates (Nicolakakis & Lefebvre, 2000; Lefebvre, Nicolakakis & Boire, 2002; Lefebvre, Reader & Sol, 2004). Feeding innovations have ecological implications because they predict the success of the introduction of birds to a non-native location (Sol *et al.*, 2005a; Sol & Lefebvre, 2000; Sol, Timmermans & Lefebvre, 2002). Feeding innovations predict intraspecific diversification as reflected by the relative abundance of subspecies and species richness in avian clades (Nicolakakis, Sol & Lefebvre, 2003; Sol, 2003).

The ability to perform novel behaviours relies on complex learning and cognitive processes, which may be facilitated by the absence of parasites and disease. A large amount of the available literature on mammals, including humans, has shown that discrimination learning and spatial and nonspatial cognition are all impaired by different kinds of parasitemias (Jukes *et al.*, 2002; Stolzfus *et al.*, 2001; Kavaliers, Colwell & Galea, 1995; Sahti *et al.*, 1999; Al Serouri *et al.*, 2000; Fiore *et al.*, 2002). Generally, because the brain that governs cognition (Lefebvre *et al.*, 2004; Shettleworth, 2001; Roth & Dicke, 2005), the detrimental effect of

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parasites on cognition should also affect the brain. Hence, species evolving larger brains should also evolve more efficient immune defenses, which, appears to be the case in birds (Møller, Erritzøe & Garamszegi, 2005). Given the positive interspecific relationship between brain size and feeding innovation (Lefebvre *et al*., 2004), and between brain size and immune defense (Møller *et al*., 2005), it is possible that feeding innovation evolves simultaneously with brain size, leading to a relationship between immune defence and innovation.

Long dispersal distances and habitat specialization may have evolved in situations where local parasite impact on host reproductive success is severe (Møller & Erritzøe, 2001; Møller, Martín-Vivaldi & Soler, 2004; Snoeijs *et al.*, 2004). Several examples exist of hosts moving out of the range of parasites, resulting in a decrease of parasite impact on host fitness, as described for reindeer, *Rangifer tarandus* L, and mangabey, *Cercocebus albigena* Gray (Freeland, 1980; Folstad *et al.*, 1991). However, migration is not the only means by which hosts can temporarily avoid or reduce the effects of parasites. Other possible defences include feeding innovations that involve different ways of finding, treating or exploiting food, which may result in the exploitation of novel niches. Such innovations could reduce the efficiency of transmission of parasites and thereby reduce the impact of parasites on host fitness. In addition, foraging innovations may allow hosts to encounter substances that have beneficial effects on the ability of hosts to rid themselves of parasites. For example, self-medication or other aspects of food-derived antiparasite defences may have arisen as a foraging innovation. Consumption of soil by ungulates or elephants as a way of reducing intestinal helminth infections (Holdo, Dudley & McDowell, 2002; Knezevich, 1998), or consumption of plant leaves with anthelminthic properties by primates (Lozano, 1998), must initially have been based on feeding innovations.

On the other hand, increased parasitism may not only be the cause, but also it may be the consequence of increased frequency of feeding innovations. Animals exploiting novel ecological strategies (e.g. by including new food types in their diet, may encounter a more diverse parasite fauna). When opportunistic species innovate feeding or other behaviour, they are confronted with new environmental factors that potentially involve parasite species not encountered before the innovation event occurred. This is likely to be even more frequent in the case of invading species, whose colonization success is partly affected by innovation rate (Sol & Lefebvre, 2000; Sol *et al*., 2002, 2005a). Hence, an opportunistic species with a broad spectrum of feeding habits is also exposed to a broad spectrum of diseases and infections. Analogically, migrating bird species that exploit two different habitats during their annual cycle, and have to cope with two different parasite faunas, evolve larger immune defence organs than resident species (Møller & Erritzøe, 1998).

The present study aimed to investigate the evolutionary link between feeding innovation and the impact of parasites as reflected by relative investment in immune defence, and the prevalence of blood parasites. We expected two patterns to occur. First, if parasite-mediated natural selection impairs learning, or if behaviourally flexible species are able to reduce parasite pressure by successfully adapting novel environments by behavioural means, we predicted the frequency of feeding innovations to be negatively related to estimates of parasitism. Second, if high parasite pressure selects for alternative feeding styles that provide hosts with novel parasite defence mechanisms, or if feeding innovation leads to extended parasitism, we predicted a positive interspecific relationship between feeding innovation frequencies and measures of parasitism. We investigated the underlying causal mechanism further by applying evolutionary modelling based on discrete variables (Pagel, 1994) and path analysis (Li, 1975). which allowed us to characterize the temporal order of evolutionary changes in feeding innovation and parasite pressure.

MATERIAL AND METHODS

Feeding innovations can be quantified from the ornithological literature, using descriptions of novel kinds of feeding behaviour (Lefebvre *et al.*, 1997). Louis Lefebvre kindly provided us with a list of reported evidence for feeding innovation in birds. This data set was collated from an exhaustive survey of 30 years (1970–2000) of the short note sections of 65 generalist ornithology journals covering six geographical areas of the world (Lefebvre *et al*., 2004). For a detailed description of the systematic data collection, see Lefebvre *et al*. (2001, 1997), Nicolakakis & Lefebvre (2000), Nicolakakis *et al*. (2003), and Sol *et al*. (2002). For each continent, we calculated the frequency of opportunistic feeding innovations, as the number of reported cases of novel feeding habits (food type or feeding technique described by an observer as novel for the species). We only considered species for which at least one report was available because the meaning of an innovation frequency of zero is obscure (Nicolakakis *et al*., 2003). Species in the European continent are ranked similarly to those in North American continent based on feeding innovation frequencies of common species for both continents (Kendall $\tau = 0.474$, $N = 41$, $P < 0.001$). Hence, feeding innovations measured in different continents appear to be species-specific attributes. We used data for European birds

because information on size of immune defence organs and parasitism were mainly available for these birds.

The probability of finding a feeding innovation in different species may depend on several factors, and absolute counts of innovation events should thus be corrected (Lefebvre *et al*., 1997; Sol *et al*., 2002). First, there may be more reports available for intensely studied species. We estimated research effort by using the number of studies published since 1972 on each species as cited in the ISI Web of Science (http://www.isiknowledge.com/). Second, we assessed the importance of population size that may affect the probability of detection of feeding innovations. We used the minimum breeding population size (in number of pairs) given in Tucker & Heath (1994), which rounds population size to the nearest million, if more than a million pairs were found for a species. Third, we also assessed the potential confounding effect of body mass on feeding innovations because larger birds may be more likely to be observed when feeding. We used our own data for body mass (see below).

To estimate relative feeding innovation that is independent of research effort, population size and body mass, we used the following approach. First, to obtain a normal distribution for feeding innovation, we calculated ranks of this variable. Then, these ranks were used in a multiple regression model as dependent variable with $\log_{10}$-transformed research effort, population size, and body mass as independent variables. This model was highly significant, indicating strong effects of research effort and population size on feeding innovation rate (overall model: $F_{3,107} = 8.454$, $P < 0.001$; effect for research effort: $F_{1,107} = 12.147$, $P < 0.001$; effect for population size: $F_{1,107} = 4.815$, $P = 0.030$; effect for body mass: $F_{1,107} = 0.458$, $P = 0.500$). From this regression line, after the exclusion of body mass, we calculated residuals that were subsequently used as measures of relative feeding innovation. Positive residuals thus indicate that the reported numbers of feeding innovations are larger than expected from research intensity and abundance.

Selection pressures arising from parasitism can be reflected by the investment of the host in immune defence that can be reflected by the relative size of immune organs (Møller & Erritzøe, 1996, 1998; Møller *et al*., 1998a, Møller, Sorci & Erritzoe, 1998b). The bursa of Fabricius synthesizes antibody, and is responsible for differentiation of the repertoire of B-cell in juvenile birds (Glick, 1983, 1994; Toivanen & Toivanen, 1987). The spleen is a part of peripheral lymphoid system, which is the main site of lymphocyte differentiation (B cells) and proliferation (B and T cells), producing cells involved in the production of humoral and cell-mediated immune responses (Rose, 1981; John, 1994, 1995). The thymus is the organ where stem cells differentiate into the three main populations of lymphocytes during the embryonic development (Rose, Payne & Freeman, 1981). Interspecific studies revealed a positive association between nematode species richness and relative spleen mass in birds (John, 1995; Morand & Poulin, 2000). Additionally, bird species with relatively large spleens appear to suffer more from parasite-induced mortality (Møller & Erritzøe, 2002).

Relying on postmortem examinations of dead birds, the size of immune organs (spleen, bursa of Fabricius and thymus) and body mass were measured by a taxidermist (J.E.) on a balance to the nearest 0.001 g, blindly with respect to the hypotheses under test. The detailed description of the standardized preparation procedure is available at: http://www.birdresearch.dk/. Because birds were frozen when received until examination, we assumed that any effects of storage on measurements should only cause noise in the data set. Although the size of these immune organs may be the subject to annual fluctuations, our interspecific data are not confounded by consistent seasonal effects, and the mass of immune organs is significantly repeatable within species (spleen: $R = 0.881$; bursa of Fabricius: $R = 0.783$, thymus: $R = 0.605$) (Møller *et al*., 2005). In the present study, we used data for feeding innovation and relative size of immune defence organs for 108 species, with information on spleen size for 97 species, on bursa size for 77 species, and on thymus size for 48 species. We controlled for allometric effects by using residuals from the phylogenetically-corrected linear regression of $\log_{10}$-transformed organ sizes on $\log_{10}$-transformed body size (see below). These residuals are subsequently termed relative organ sizes. When we used alternative approaches (e.g. partial correlation) to control for body size, the results and conclusions were identical to findings based on residuals.

Because the use of the relative size of immune defence organs to infer the effectiveness of the immune system may reserve some limitations (Smith & Hunt, 2004), we also tested our predictions by using real parasite load data (i.e. haematoza prevalence from the literature) as an alternative measure of parasitism. The prevalence of blood parasites varies consistently among bird species, and this variation may arise from various selection forces, such as sexual selection, offspring development, disease resistance, and vector abundance (Scheuerlein & Ricklefs, 2004; and references therein). Although, the immunological mechanisms involved in resistance to these pathogens are poorly understood (Buckling & Read, 2001), it has been proposed that haematozoan prevalence may primarily reflect immunocompetence because it is closely linked to the immunological capacity of the host that ultimately determines parasite resistance (Hamilton & Zuk, 1982; Atkinson & van Riper, 1991; Ricklefs, 1992; Tella *et al.*, 1999). We used the square-root-

arcsine transformed proportion of individuals infected with haematozoa (*Plasmodium*, *Haemoproteus*, *Leucocytozoon*, and *Trypanosoma* combined) in 45 passerine species from Scheuerlein & Ricklefs (2004).

Migratory habit has been shown to be associated with immune defence (Møller & Erritzøe, 1998) and feeding innovation (Sol, Lefebvre & Rodriguez-Teijeiro, 2005b). Therefore, migration should be controlled in our comparative study. Migration was scored on a three point scale as: (1) resident (a score of 1); (2) partial migrant (species having resident and migratory populations; a score of 2); or (3) migrant (a score of 3). Information on migration originated from handbooks and field guides (Heinzel, Fitter & Parslow, 1997; Cramp & Perrins, 1985–94), and was treated as a continuous variable in the comparative analyses because intermediate states are biologically meaningful.

Similarly, relative brain size can be a potential confounding factor because it correlates interspecifically with both immune defence and feeding innovation (Lefebvre *et al*., 1997; Møller *et al*., 2005); thus, an apparent relationship between them may mediated by relative brain size. Therefore, we also used information on brain size that was available from our post-mortem measurement. The reliability and repeatability of this trait are given in detail elsewhere (Garamszegi, Møller & Erritzøe, 2002; Garamszegi, Eens, Erritzøe & Møller, 2005; Møller *et al*., 2005). Brain mass was $\log_{10}$-transformed, and was adjusted for allometry, as described previously.

Because male plumage brightness and geographical distribution are phylogentically associated with haematozoa prevalence (Scheuerlein & Ricklefs, 2004), we also assessed the importance of these traits. Data on plumage brightness and geographical distribution (first detrended correspondence analysis axis) were available from Scheuerlein & Ricklefs (2004). The full dataset, without information on feeding innovation is given in the Appendix. Data on feeding innovation can be obtained from L. Lefebvre.

Because species are not statistically independent observations, a phylogenetic control is required to eliminate the confounding effects of common ancestry. We constructed a composite phylogenetic hypothesis at the family level mainly based on information available in Sibley & Ahlquist (1990), which was obtained from extensive studies of DNA–DNA hybridization. This phylogeny was supplemented at the subfamily level with information from Arnaiz-Villena *et al*. (1998), Blondel, Catzeflis & Perret (1996), Cibois & Pasquet (1999), Grapputo *et al*. (2001), and Thomas, Wills & Székely (2004). We applied branch lengths from the tapestry tree of Sibley & Ahlquist (1990) for higher taxonomic levels. Within families, the distance between different genera was set to 3.4 $\Delta T_{50}H$ units, and between species within genera to 1.1 $\Delta T_{50}H$ units (Sibley & Ahlquist, 1990; Bennett & Owens, 2002). The phylogenetic tree we used in our comparative analyses is given in Figure 1.

We applied the general method of comparative analysis for correlated evolution of traits based on generalized least squares (GLS) models implemented in the software Continuous (Pagel, 1997, 1999). Hypothesis testing in this program relies on likelihood ratio (LR) test statistic that compares the log-likelihood of the model corresponding to a null hypothesis (H_0) over the model for an alternative hypothesis (H_1). First, we assessed the contribution of different branch lengths and the importance of phylogenetic relationships by adjusting the appropriate scaling parameters. Second, we tested the correlation between pairs of traits. We present the phylogenetically corrected correlation coefficients (r_{phyl}) and the corresponding LR statistics. To control for allometric effects, we calculated the phylogenetically corrected regression of organ size on body mass, using Continuous. Based on this phylogenetic equation, residuals were obtained for the raw species (Purvis & Rambaut, 1995). When we controlled for the potentially confounding effects arising from migratory habit, relative brain size, male plumage brightness, and geographical distribution, we entered these variables together with the variables of interest in the model, and tested for correlated trait evolution. We then calculated the partial phylogenetic correlation for the relationship between the relative innovation rate and organ size.

We also analysed the data by using used Pagel's discrete variable method available in the software 'Discrete' (Pagel, 1994). This method applies a continuous-time Markov model to characterize evolutionary changes along each branch of a phylogenetic tree. The LR statistic for the discrete model (omnibus test) compares models H_0 and H_1, with a Monte Carlo simulation procedure being used to derive the null hypothesis distribution of significance. The advantage of the discrete model is that it not only tests for correlated trait evolution, but also it examines whether changes in one variable make changes in another more or less likely than would be expected from random. We used the discrete variables method to test the temporal ordering and direction of evolutionary change of feeding innovation and parasitism. The method allows various tests of whether specified character transitions are significantly different from zero or from each other. Transition rate parameters, q_{ij}, denote the rate of change from state i to state j. The subscripts refer to the beginning and end character states for each particular transition, where 1 = 0,0, 2 = 0,1, 3 = 1,0, and 4 = 1,1. We tested models of evolution in which certain types of transitions are excluded a priori, by forcing the relevant parameters

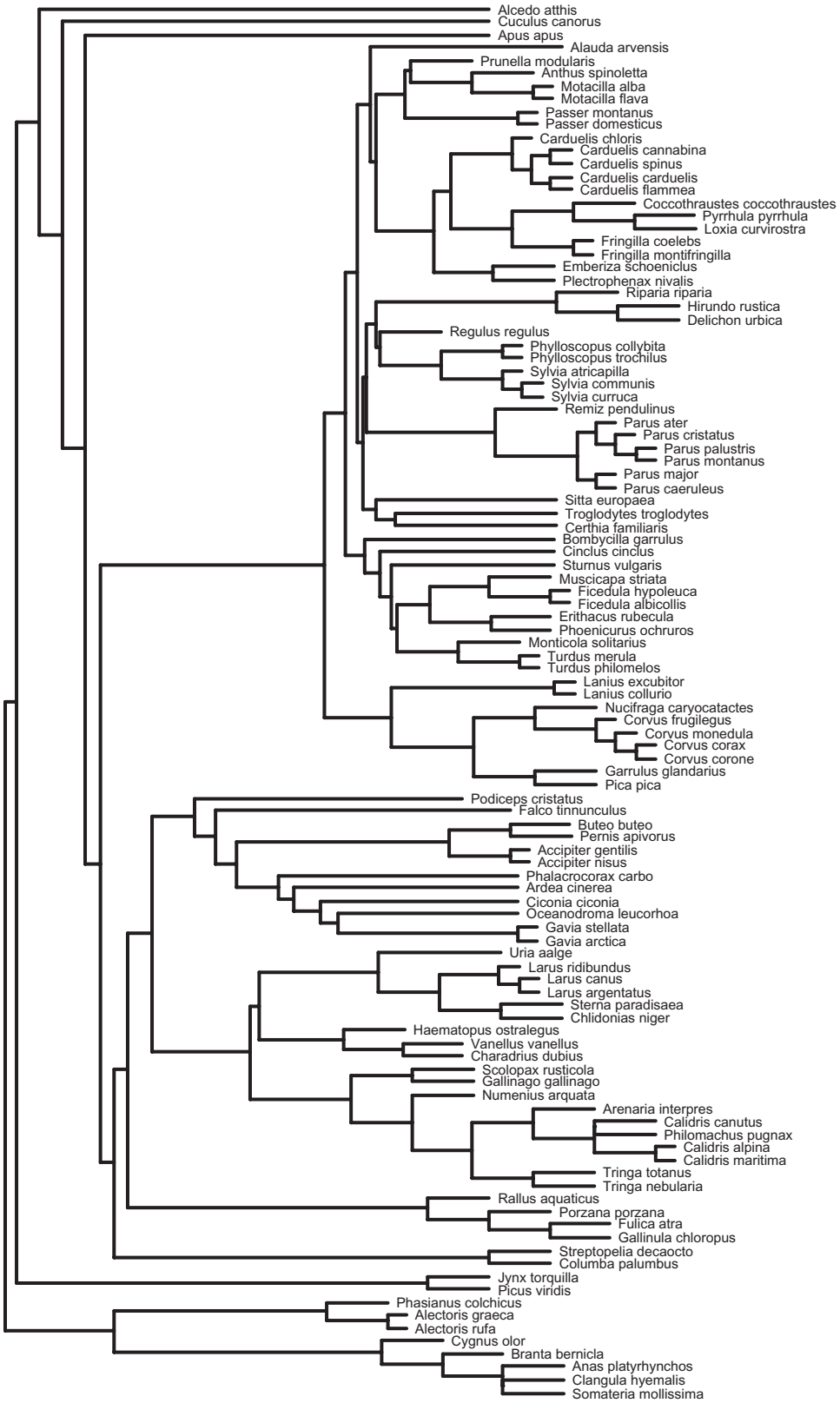

Figure 1. Phylogenetic hypotheses of birds used to investigate the interspecific relationship between feeding innovation and parasitism. The sources are provided in the Material and Methods section. Scales are shown at the bottom left of the figure.

(q_{ij}) to zero. The fit of the reduced model, in which one parameter is constrained, was then compared to the full model of eight parameters. We chose relative spleen size and relative haematozoa prevalence (adjusted for geographical distribution and male plumage brightness) for this evolutionary modelling. For the discrete approach, we categorized each variable as being larger or smaller than predicted (positive or negative residuals). However, collapsing continuous variables into two-state categories might reduce the power of the analysis, due to loss of information.

To investigate the causal relationship between measures of parasitism and feeding innovations, relying on the phylogenetic correlation coefficients from Program Continuous, we also used path analysis (Li, 1975). We implemented previous findings in this modelling, and predicted that feeding innovation is determined by brain size (Nicolakakis *et al*., 2003; Sol *et al*., 2005a), We created three different evolutionary models. The first model predicted that, arising from the same selection pressures, parasitism and relative brain size mutually influence each other and both affect the evolution of the relative innovation rate (Møller *et al*., 2005). According to the second model, a relatively larger brain evolves to allow a high innovation rate that has a secondary impact on parasite levels. The third model predicted that parasitism primarily has an evolutionary effect on brain size that subsequently favours higher rates of innovation. We identified the statistical model that explained the most variance in the relationship between the relative innovation rate, parasitism, and brain size. As different estimates of the impact of parasites, we used both relative spleen size and haematozoa prevalence in our evolutionary path modelling.

RESULTS

The GLS modelling of continuous variables revealed that relative feeding innovation was significantly and positively related to the relative size of the spleen and the bursa of Fabricius (relative spleen size: $r_{phyl} = 0.354$, $P < 0.001$, $N = 98$, Fig. 2A; relative size of the bursa of Fabricius: $r_{phyl} = 0.417$, $P < 0.001$, $N = 77$, Fig. 2B) However, a very similar tendency was found for the relative size of the thymus, but this was non-significant, probably due to the lower sample size ($r_{phyl} = 0.262$, $P = 0.064$, $N = 48$, Fig. 2C). These results appear to be independent of the potentially confounding effect of migration and brain size because we found similar patterns when we calculated the partial phylogenetic correlations between the relative innovation rate and measures of immune defence (relative spleen size, partial $r_{phyl} = 0.227$, $P = 0.036$, $N = 88$; relative size of the bursa of Fabricius, partial $r_{phyl} = 0.225$, $P = 0.087$, $N = 61$; relative thymus size, partial $r_{phyl} = 0.444$, $P = 0.003$, $N = 44$).

We also found a positive phylogenetic association between the prevalence of blood parasites and relative feeding innovation in passerines ($r_{phyl} = 0.424$, $P = 0.003$, $N = 45$, Fig. 2D). Again, this pattern appeared to be independent of potentially confounding factors, such as male plumage brightness and geographical distributions (partial $r_{phyl} = 0.410$, $P = 0.006$, $N = 45$) or migration and relative brain size (partial $r_{phyl} = 0.337$, $P = 0.041$, $N = 39$).

We investigated the casual relationship between parasitism and relative rate of feeding innovation, using the phylogenetic method developed for discrete variables. We found significant evidence for correlated evolution of relative spleen size and the relative innovation rate (LR = 7.69, $P < 0.01$ after 100 simulations). However, the categorization decreased the significance of the evolutionary correlation between the relative innovation rate and the relative prevalence of blood parasites (LR = 2.14, $P = 0.18$, after 100 simulations). Hence, we proceeded with hypothesis testing corresponding to the temporal order of changes by using the discrete model for relative spleen size.

First, we estimated ancestral states for the variables. When using continuous variables in the program Continuous, we found that the estimated ancestral state for the relative rate of feeding innovation was –16.48 (SE = 10.08), whereas it was –0.239 (SE = 0.07) for relative spleen size. Therefore, the most likely evolutionary scenario for the ancestral state is that species had a low innovation rate and relatively small spleens at the root of the phylogenetic tree. We characterized the most likely route of evolution from this probable ancestral state. Table 1 shows

Table 1. Comparisons of likelihood values for alternative discrete models of evolution, in which one transition is excluded, vs. an eight-parameter model of dependent evolution of feeding innovation and relative spleen size reflecting investment in immune defense

Alternative models	$L(D_7)$	LR	*P*
$q_{12} = 0$	–118.57	0.00	NS
$q_{13} = 0$	–121.58	6.02	< 0.05
$q_{21} = 0$	–121.68	6.40	< 0.01
$q_{24} = 0$	–122.12	7.10	< 0.01
$q_{31} = 0$	–118.59	0.06	NS
$q_{34} = 0$	–122.85	8.56	< 0.01
$q_{42} = 0$	–125.77	14.40	< 0.001
$q_{43} = 0$	–123.50	9.86	< 0.01

The log-likelihood of the full, eight-parameter model was –118.57.
NS, not significant.

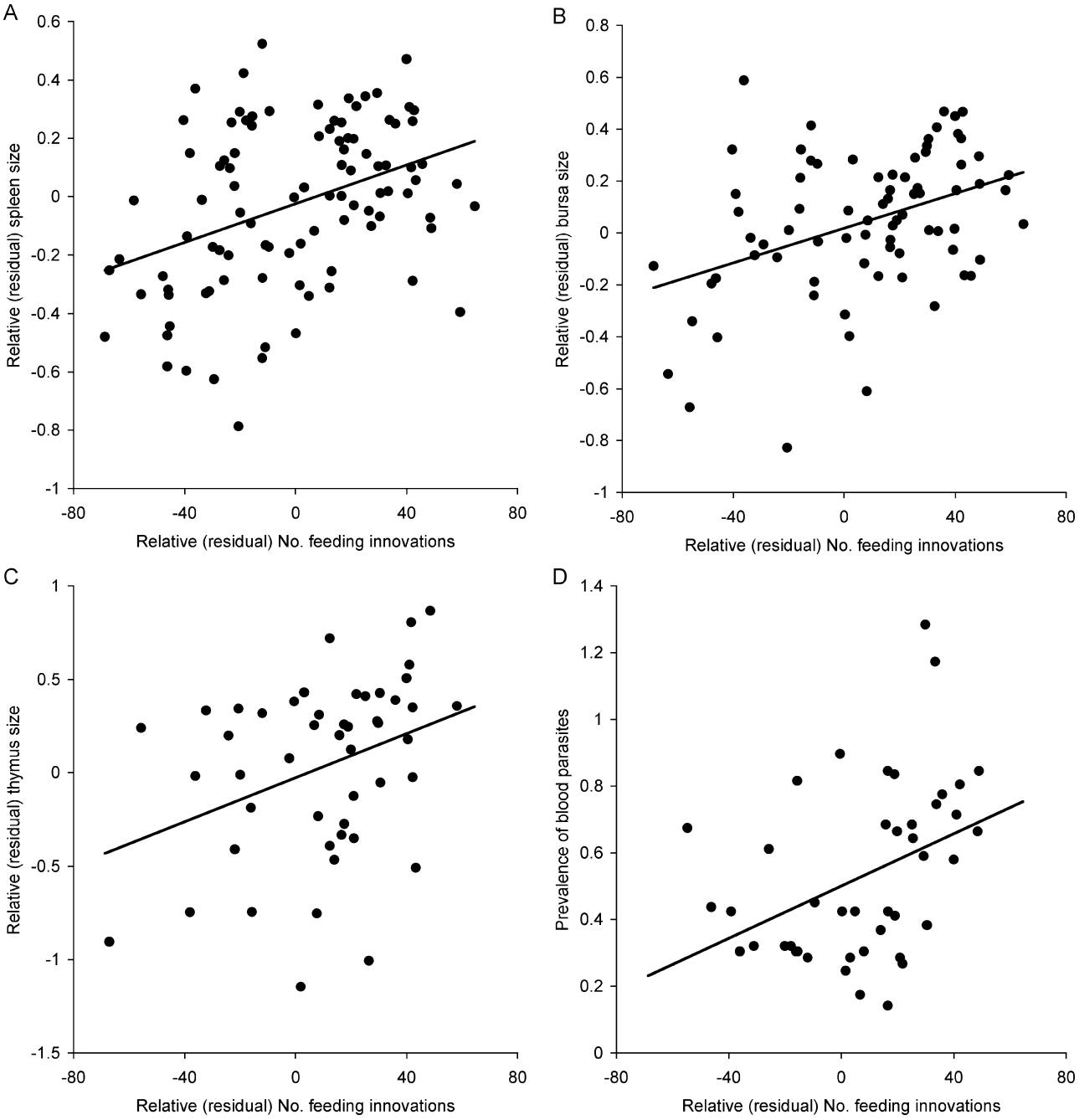

Figure 2. Interspecific relationship between relative frequencies of feeding innovation in European species (corrected for research effort and population size) and the relative size of immune defense organs (A, spleen; B, bursa of Fabricius; C, thymus; all are corrected for body size). D, prevalence of blood parasites (square-root-arcsine transformed). Data are raw species data and their associated linear regression lines.

the log-likelihood values for the alternative models. The coupled evolution of feeding innovation and relative spleen size can be summarized by the following major events (Fig. 3). First, innovation rate increased without the evolutionary enlargement of spleen size (transition rate q_{13}). Second, relative spleen size increased (transition rate q_{34}). In a third stage, both relative spleen size and the rate of feeding innovation varied independently of each other (transition rates q_{42}, q_{43}, and q_{24}).

Path analysis revealed that the second evolutionary model, which assumes a causal mechanism with feed-

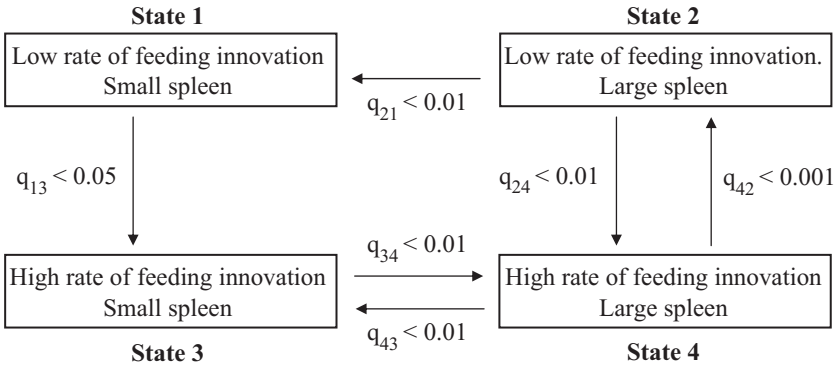

Figure 3. Flow diagram tracing the most likely evolutionary scenario for the coupled evolution of feeding innovation and relative spleen size in birds. State 1 corresponds to the most probable ancestral state. Only significant pathways are shown. The q_{ij}s are the transition rate parameters, with subscripts referring to the beginning and end character states for each particular transition (1 = 0,0; 2 = 0,1; 3 = 1,0; and 4 = 1,1); see also Table 1.

ing innovation affecting relative spleen size, has the largest explanatory power ($r^2 = 25.27\%$, Fig. 4). However, this model explained only 1.34% more of the variance than the third model predicting the reverse causal chains of evolutionary events. The first model, which involved independent effects for parasitism and brain size, had the smallest explanatory power ($r^2 = 19.39\%$). When we used haematozoa prevalence instead of relative spleen size to reflect parasitism, we also found that the second model explained the most of the variance (model 1: $r^2 = 21.30\%$, model 2: $r^2 = 24.76\%$, model 3: $r^2 = 11.35\%$).

DISCUSSION

The main finding of the present was that bird species with relatively larger immune defence organs and higher prevalence of blood parasites had higher rates of feeding innovations than species with small immune defence organs or lower parasite prevalence. We interpret these findings as implying, for the first time, that the evolution of feeding innovations has implications for parasite-mediated natural selection. We briefly discuss each of these findings below.

The relative size of both bursa of Fabricius and spleen, after adjusting for body size, predicted the relative frequency of feeding innovations in birds (Fig. 2). In a smaller sample, a similar tendency was found for the relative size of the thymus. Because relatively large immune defence organs imply that a host species suffers severely from parasite-mediated natural selection (Møller & Erritzøe, 2002), we can speculate that species with a higher rate of feeding innovations have a greater parasite-induced mortality. Although caution is warranted when using the size of immune defence organs to infer the general impact of parasites (Smith & Hunt, 2004), our findings were very similar when our analyses were based on parasite prevalence. This suggests that differences in rates of feeding innovations have generally given rise to parasite-mediated selection.

Three evolutionary scenarios can mediate a link between feeding innovations and parasitism (see Introduction). First, parasite-mediated selection may depress the rate of foraging innovations because the presence of parasites may depress learning ability. This mechanism was not supported by our data because it would predict a negative association between estimates of parasitism and innovation rate, which was not the case. Second, feeding innovations may provide means by which infected hosts change their ecological niche and escape or reduce the negative impact of parasitism on host fitness. This scenario predicts both positive and negative relationships between parasitism and innovation. If parasites drive behavioural responses, a causal link could be that strong parasite pressure facilitates feeding innovation, which should result in their positive association. However, the same driving force, but with opposite causal links, may mediate behaviourally flexible species to invest less in immune defence because they are able to reduce the pressure of parasites throughout behavioural adjustments to different ecological niches. This mechanism, predicting a negative relationship between traits, is unlikely because we observed positive associations. In addition, we not only found evidence for the efficiency of the immune system, but also for parasite prevalence. Hence, innovative species apparently do not decrease their parasite loads. Third, feeding innovation being correlated with other opportunistic behaviours (Nicolakakis & Lefebvre, 2000; Lefebvre *et al.*, 2002) may involve the risk of exposure to a wide range of parasites, thereby resulting in an increased parasite load in innovative species. This

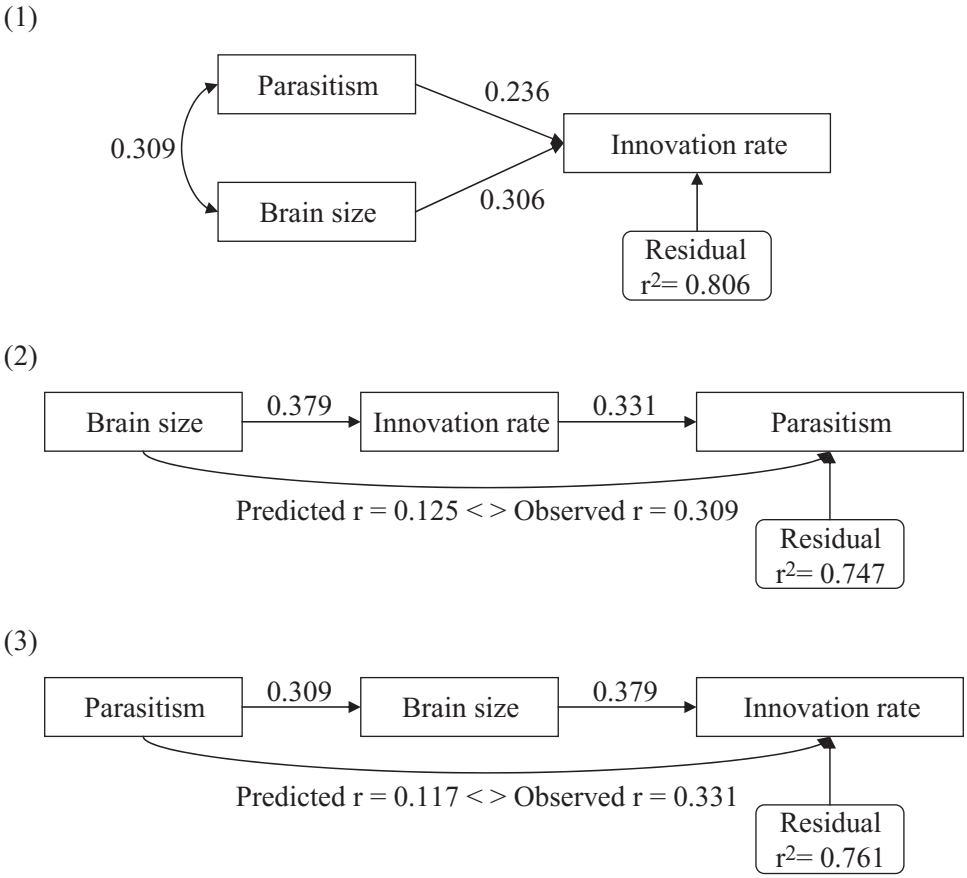

Figure 4. Path analysis of relative innovation rate, measures of parasitism, and relative brain size, when relative spleen mass is used to reflect parasitism. Values are path coefficients calculated from the pairwise phylogenetic correlation among the three traits. The three models correspond to the causal mechanisms in which (1) parasitism and relative brain size mutually influence each other and both affect the evolution of feeding innovation; (2) relative brain size evolves first to allow high rate of innovation that has a secondary consequences for parasitism; and (3) parasites have an evolutionary effect on relative brain size that subsequently favours higher rates of innovation. Correlation coefficients between indirectly related traits as predicted by different evolutionary models (predicted r) are compared with observed phylogenetic correlations (observed r). Residual variance is the amount of variance that is not explained by the model, the explained variance can be calculated as $1 - r^2$. Explanatory power of the models that rely on haematozoa prevalence instead of relative spleen size is given in the main text.

hypothetical mechanism predicts a positive relationship between feeding innovation and measures of parasitism. In summary, our correlative findings appear to be consistent with the hypotheses that: (1) innovative species are evolutionarily more successful under high parasite pressure and (2) feeding innovation and other opportunistic behaviours result in higher parasite pressure.

However, our detailed evolutionary modelling indicates that the latter mechanism is more likely because the evolutionary changes in innovation rate precede evolutionary changes in parasitism (Figs 3, 4). The phylogenetic analyses of causality based on discrete variables revealed that, from an ancestral state with low innovation rate and small immune defence organs (state 1), only one evolutionary route (q_{13} and q_{34}) can lead to the correlative evolution of traits. In this route, innovation rate increases first (state 3), which is followed by an increased investment in immune defense (state 4). Additionally, subsequent evolutionary changes from high innovation rates and large immune organs (state 4) towards low innovation rates and small immune organs (state 1) along q_{42} and q_{21} may also result in a correlative evolution. This evolutionary route also predicts that evolutionary steps in innovation rate occur before evolutionary steps in immune defence. Moreover, the path analyses have similar implications for the temporal order of changes. Most of the variance was explained by the evolutionary models that predicted that relative brain size and innovation

rate evolve first, and these evolutionary changes have secondary impact for parasitism. Although the explanatory power of these models was only 1.3–3.5% higher compared with the other models, such differences may be evolutionary important (Møller & Jennions, 2002). Therefore, the interspecific relationship between feeding innovation and parasitism is more likely to be explained by the hypothesis that enhanced innovation puts species under higher parasite pressure.

It remains unresolved whether it is the increasing feeding innovation rate or a correlate thereof that affects parasite level on an evolutionary scale. Opportunistic feeding behaviour by exploiting novel food sources and/or acquiring novel feeding styles may per se increase the risk of parasitism. However, variation in feeding innovation may predict variation in other cognitive tasks, and behavioural flexibility, opportunism, and social learning may evolve simultaneously (Nicolakakis & Lefebvre, 2000; Lefebvre *et al*., 2002; Reader & Laland, 2002). Therefore, feeding innovation reflecting complex cognitive functions may correlate with parasitism, because opportunistic behaviours ameliorating the occupation of new niches in general may have consequences for host–parasite interactions. Moreover, behavioural flexibility and opportunism may be associated with environmental variability, width of the ecological niche, social structure, morphological variability, and population density (Lefebvre & Bolhuis, 2003), which may all involve increased parasitism pressures. Clearly, further analyses are required to distinguish whether feeding innovation alone or its cognitive or ecological correlates enhance the risk of parasitism.

If innovative species having higher success in coping with the environment or a superior ability to colonize novel habitats suffer from high parasite pressure, such species would pay fitness costs for their superior capacity. The evolutionary costs of being innovative can be well-expected. For example, feeding innovation should positively correlate with the length of the nestling period and metabolic rate (Lefebvre *et al*., 2004). These relationships would arise because development and maintenance of brain functions corresponding with enhanced cognition are traded against other self-maintenance mechanisms.

A higher rate of feeding innovation may facilitate successful establishment in a novel environment (Sol & Lefebvre, 2000; Sol *et al*., 2002; Sol *et al*., 2005a). However, parasite-mediated selection may also be involved in successful invasion. Introduced populations of animals had a considerably reduced parasite fauna compared with the populations of origin, implying that invasion success is a direct function of the ability to leave parasites behind (Torchin *et al*., 2003). The loss of parasites should be particularly important in host species with a high degree of parasite-induced mortality (Møller & Cassey, 2004). Hence, immune function, especially antibody-mediated immunity, should be of much greater importance in such successful invaders than in unsuccessful ones (Lee & Klasing, 2004). Therefore, the results of the present suggest that feeding invasion and parasitism may mediate invasion success act along the same axis.

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APPENDIX

Research effort (number of papers published), population size (number of breeding pairs), body mass (g), mass of the spleen (g), and mass of the bursa of Fabricius (g), mass of the thymus, mass of the brain, migration, prevalence of blood parasites (haematozoa), geographical distribution (first DCA), and male plumage brightness of European birds

Species	Research effort	Population size	Body mass (g)	Spleen mass (g)	Bursa mass (g)	Thymus mass (g)	Brain mass (g)	Migration	Haematozoa prevalence	DCA1	Male plumage brightness
Accipiter gentilis	151	130 000	830.6	0.827	0.332	0.032	7.697	1			
Accipiter nisus	164	270 000	206.6	0.149	0.165	0.017	2.936	2			
Alauda arvensis	104	1 000 000	40.2	0.028	0.05	0.006	0.957	2	0.09	184.43	1.5
Alcedo atthis	14	46 000	38.7	0.031	0.046	0.004	0.817	2			
Alectoris graeca	26	34 000	542	0.12				1			
Alectoris rufa	77	1 000 000	410.2	0.167	0.222	0.123	1.72	1			
Anas platyrhynchos	1233	1 000 000	1 224.5	0.74	0.266	0.01	5.86	2			
Anthus spinoletta	27	380 000	23.9		0.043			2	0.09	167.95	1.5
Apus apus	52	1 000 000	42	0.022	0.012		0.684	3			
Ardea cinerea	90	130 000	2 000	0.709	1.663		8.096	2			
Arenaria interpres	67	27 000	119		0.102			3			
Bombycilla garrulus	3	130 000	63.4	0.055	0.04	0.005	1.102	2			
Branta bernicla	168	1 100	1 340	0.4			6.041	3			
Buteo buteo	175	740 000	769.3	0.689	0.663	0.144	7.969	2			
Calidris alpina	158	390 000	37.5	0.018		0.001	0.861	2			
Calidris canutus	152	10 000	166	0.069			1.3	3			
Calidris maritima	29	25 000	70.2	0.059	0.061			2			
Carduelis cannabina	21	1 000 000	18.6	0.03	0.045		0.663	2	0.09	247.81	3
Carduelis carduelis	18	1 000 000	15.4	0.006			0.605	2	0.17	283.95	4
Carduelis chloris	76	1 000 000	26.6	0.028	0.024	0.008	0.88	2	0.38	251.05	3
Carduelis flammea	28	1 000 000	11.8	0.005	0.018		0.539	2	0.06	104.77	3.5
Carduelis spinus	34	1 000 000	11.9	0.013	0.014		0.573	2	0.17	61.12	2
Certhia familiaris	37	1 000 000	9.6	0.016			0.51	1	0.1	126.81	1.5
Charadrius dubius	9	110 000	39.3	0.008			0.824	3			
Chlidonias niger	31	57 000	73.5		0.057			3			
Ciconia ciconia	34	120 000	3 342	1.439			16.01	3			
Cinclus cinclus	103	110 000	62.7	0.03	.		1.267	2			
Clangula hyemalis	27	370 000	812	0.327	0.188		5.187	2			
Coccothraustes coccothraustes	6	920 000	55.7	0.076	0.059	0.02	1.569	2	0.55	202.55	4
Columba palumbus	43	1 000 000	520	0.074	0.049	0.152	2.524	2			
Corvus corax	108	280 000	1 322.5	1.207	1.812		15.491	1	0.85	185.04	1.5

APPENDIX *Continued*

Species	Research effort	Population size	Body mass (g)	Spleen mass (g)	Bursa mass (g)	Thymus mass (g)	Brain mass (g)	Migration	Haematozoa prevalence	DCA1	Male plumage brightness
Corvus corone	108	1 000 000	525.7	0.388	0.656	0.514	8.274	1	0.38	200.82	1.5
Corvus frugilegus	99	1 000 000	523.6	0.39	0.764	0.186	7.944	2	0.14	177.07	1.5
Corvus monedula	63	1 000 000	210.1	0.139		0.059	4.725	2	0.03	216.09	1.5
Cuculus canorus	183	1 000 000	108.4	0.031	0.068		1.467	3			
Cygnus olor	109	49 000	11 800	2.754			15.223	2			
Delichon urbica	91	1 000 000	18.3	0.034				3	0.16	227.03	3
Emberiza schoeniclus	36	1 000 000	20.5	0.032			0.698	2	0.1	92.79	2
Erithacus rubecula	171	1 000 000	18	0.028	0.027	0.002	0.65	2	0.13	160.63	2.5
Falco tinnunculus	276	290 000	190.6	0.085	0.265	0.068	3.828	2			
Ficedula albicollis	140	340 000	12.1	0.01				3			
Ficedula hypoleuca	640	1 000 000	13.2	0.015			0.46	3	0.33	69.72	3.5
Fringilla coelebs	157	1 000 000	24.1	0.032	0.036	0.009	0.755	2	0.4	127.44	4
Fringilla montifringilla	32	1 000 000	24	0.036		0.001	0.734	3	0.53	0	4
Fulica atra	76	1 000 000	518.3	0.811			3.213	2			
Gallinago gallinago	29	1 000 000	112.6	0.052	0.06		1.401	2			
Gallinula chloropus	53	790 000	299.3	0.336	0.143		2.007	2			
Garrulus glandarius	41	1 000 000	166.3	0.183	0.28	0.05	3.931	1	0.92	212.5	4
Gavia arctica	11	120 000	2 855	1.3		.		3			
Gavia stellata	21	61 000	1 232	0.708			5.6	3			
Haematopus ostralegus	321	200 000	531.2	0.59			4.02	2			
Hirundo rustica	487	1 000 000	16.6	0.029	0.032	0.011	0.561	3	0.07	238.68	3.5
Jynx torquilla	5	350 000	38.6	0.036			0.852	3			
Lanius collurio	52	1 000 000	28.2	0.024		0.015		3	0.61	250.71	4.5
Lanius excubitor	30	330 000	59.6	0.04	0.044		1.48	2	0.56	179.96	3
Larus argentatus	486	900 000	1 035	0.809	0.862	0.012		2			
Larus canus	41	420 000	376.6	0.413		0.34	4.07	2			
Larus ridibundus	144	1 000 000	240.7	0.413	0.514		2.824	2			
Loxia curvirostra	38	840 000	44.6	0.049	0.023		1.528	1			
Monticola solitarius	1	38 000	57		0.053	0.002		2			
Motacilla alba	36	1 000 000	20.6	0.032	0.054	0.005	0.562	2	0.52	139.03	3.5
Motacilla flava	22	1 000 000	17.6	0.011	0.029		0.426	3	0.17	137.54	3.5
Muscicapa striata	12	1 000 000	14.7	0.023	0.018		0.464	3	0.46	114.12	2.5
Nucifraga caryocatactes	12	150 000	193.8	0.236		0.012	5.82	2			
Numenius arquata	51	120 000	806	1.202	0.772	0.521		2			
Oceanodroma leucorhoa	40	91 000	39.8	0.023				3			
Parus ater	59	1 000 000	9.5		0.006			2	0.17	232.47	2.5
Parus caeruleus	273	1 000 000	10.9	0.013	0.027		0.606	1	0.36	216.75	5

Parus cristatus	38	1 000 000	10.9	0.017	0.029			1			
Parus major	1039	1 000 000	17.6	0.027	0.03	0.002	0.852	1	0.56	224.66	4.5
Parus montanus	185	1 000 000	10.2		0.006			2	0.39	103.97	2.5
Parus palustris	85	1 000 000	11	0.006		0.004	0.644	1			
Passer domesticus	580	1 000 000	28.5	0.054	0.043	0.016	0.887	1	0.4	223.87	2.5
Passer montanus	58	1 000 000	22.5	0.031	0.028	0.011	0.773	2			
Pernis apivorus	29	110 000	758	0.828				2			
Phalacrocorax carbo	198	140 000	1 991.2	1.386	1.405		9.784	2			
Phasianus colchicus	250	1 000 000	1 389.1	0.564	0.157	0.269	4.027	1			
Philomachus pugnax	69	1 000 000	138		0.106			3			
Phoenicurus ochruros	22	1 000 000	14.8	0.006			0.55	2	0.1	230.15	3.5
Phylloscopus collybita	52	1 000 000	8	0.007	0.011	0.002	0.309	3	0.14	197.47	3
Phylloscopus trochilus	116	1 000 000	10.5	0.003	0.009		0.335	3	0.18	87.17	3
Pica pica	213	1 000 000	215.2	0.332	0.47	0.082	5.314	1	0.49	187.31	4.5
Picus viridis	6	350 000	174.7	0.167	0.197	0.064	4.35	1			
Plectrophenax nivalis	27	690 000	42.2		0.038			2			
Podiceps cristatus	47	340 000	981.4	0.577	1.022		3.596	2			
Porzana porzana	3	52 000	77.9		0.053			3			
Prunella modularis	152	1 000 000	20.7	0.019	0.045	0.013	0.665	2	0.08	144.15	1.5
Pyrrhula pyrrhula	27	1 000 000	25.9	0.035	0.019	0.004	0.861	2	0.08	119.35	4
Rallus aquaticus	9	110 000	118.5	0.095	0.106		1.575	2			
Regulus regulus	36	1 000 000	5.7	0.01	0.002	0.001	0.367	2	0.09	78.92	3.5
Remiz pendulinus	28	140 000	9.3	0.021				2			
Riparia riparia	79	1 000 000	14	0.012	0.015			3	0.02	139.9	2
Scolopax rusticola	18	1 000 000	330.9	0.181	0.183	0.075	2.498	2			
Sitta europaea	84	1 000 000	24.1	0.029	0.032	0.001	1.043	1			
Somateria mollissima	232	740 000	2 218.1	0.703			7.916	2			
Sterna paradisaea	58	430 000	110	0.029	0.053			3			
Streptopelia decaocto	45	1 000 000	185.6	0.045			1.552	1			
Sturnus vulgaris	1046	1 000 000	79.2	0.099	0.118	0.027	1.802	2			
Sylvia atricapilla	149	1 000 000	18.6	0.036	0.044	0.009	0.62	2	0.31	255.34	2
Sylvia communis	38	1 000 000	14.3	0.024	0.016		0.528	3	0.19	166.75	3
Sylvia curruca	7	1 000 000	12.3	0.035	0.029	0.007	0.478	3	0.08	123.87	2.5
Tringa nebularia	9	61 000	174		0.207			3			
Tringa totanus	107	300 000	112.1	0.051	0.243		1.544	2			
Troglodytes troglodytes	104	1 000 000	9.5	0.019	0.048	0.003	0.504	2	0.09	245.14	1.5
Turdus merula	236	1 000 000	98.2	0.251	0.237	0.057	1.77	2	0.3	238.72	2
Turdus philomelos	50	1 000 000	69.8	0.122	0.153	0.051	1.494	2	0.43	108.86	3
Uria aalge	309	1 000 000	939.2	0.56	0.347		4.763	3			
Vanellus vanellus	139	1 000 000	217.3	0.166	0.165	0.033	2.109	2			

DCA, detrended correspondence analysis.