Letters

Genome size, longevity and development time in birds

On the basis of a comparative analysis among species of birds, Monaghan and Metcalfe[1] suggested that longevity and genome size are positively correlated. Although this result is intriguing, a recent comparative study based on a different measure of ageing failed to find a relationship between genome size and senescence in birds, with or without development-time variables included in the analysis[2]. The inconsistency between these studies could, in part, reflect the different measures of longevity. Monaghan and Metcalfe used oldest recorded lifespan, which depends on both age-independent and senescent mortality, and is sensitive to sample size and the duration of the study. For example, recorded lifespans of 13.2 years for the greater flamingo and 7.0 years for the rock dove are not consistent with the known longevity of these species in captivity. Ricklefs and Scheuerlein[2] used parameters of Weibull models fitted to survival curves of birds in captive populations as a measure of rate of ageing.

The two studies might also differ because of the manner in which the data were handled. Monaghan and Metcalfe calculated phylogenetically independent contrasts (PICs) based on differences between families, whereas Ricklefs and Scheuerlein used species values without correcting for phylogenetic relationship. However, analyses of PICs rarely contradict regressions based on species values[3]. When we applied multiple regression to species values in Monaghan and Metcalfe's dataset, we found that log-transformed longevity was unrelated to genome size, regardless of which other variables were entered in the regression, whether longevity and development time were normalized with respect to body mass, or whether order was entered as an effect when normalizing variables.

Accordingly, we have reanalysed Monaghan and Metcalfe's data (concerning 63 species of birds) using the independent contrasts method[4] based on a species-level phylogeny. We used the DNA-hybridization phylogeny constructed by Sibley and Ahlquist[5], which included 58 of the species in Monaghan and Metcalfe's data, to extract independent contrasts. Using the CAIC program[6], we obtained 50 independent contrasts. Data were log-transformed before analysis. All correlations between contrasts were forced through the origin[7]. To verify that contrasts were properly standardized, we checked that the standard deviations of the standardized contrasts did not vary in relation to their absolute values ($P = 0.51$)[7].

We applied a multiple regression to contrasts and found that the total period of development in the nest (egg laying to fledging) is positively related to genome size and to body size ($R = 0.81$, $P < 0.0001$). We found that longevity is positively related to body size ($R = 0.47$, $P < 0.0005$) and that genome size explained no additional variation. Development period controlled for body mass was also significantly related to genome size ($R = 0.35$, $P < 0.2$, Fig. 1a), but was unrelated to longevity controlled for body mass (Fig. 1b, $P = 0.84$). If we consider the incubation period (egg laying to hatching, controlled for body mass) alone, a significant relationship also exists with genome size, although the relationship is weaker ($R = 0.31$, $P < 0.05$). Incubation period and longevity were not significantly correlated (both variables controlled for body mass, $P = 0.98$).

Our results contradict the finding of Monaghan and Metcalfe[1], but the analyses were conducted on different taxonomic levels. To determine whether correlations absent on the species level might appear at the family level, we performed a hierarchical analysis of covariance on the Monaghan and Metcalfe dataset, treating the data in the same way that they did. We used the deviations from a log–log regression of lifespan on body size to obtain a relative lifespan, and then assessed the covariation between relative lifespan and genome size at the levels of order, family within order, and error (species- and genus-level variation). The results of the variance component correlations show very strong correlation at the family level, consistent with the results of Monaghan and Metcalfe, but none among the error variance or at the order level. The slope of the regression at the family level was 0.214, which is essentially identical to the value obtained by Monaghan and Metcalfe (0.217). However, it is also clear that there is no relationship between relative lifespan and genome size among genera and species within families, nor in the dataset overall, that is, among species. The significant result at the family level represents only a small amount of the total variance in the data (26% for the relative lifespan and 6% for genome size). It is possible that the family-level correlation results from values for a small number of species. When we partitioned the dataset into equal halves, the family-level correlation for one half of the data remained significant, but it was close to zero for the other half.

Correlation studies cannot determine whether the positive relationship between genome size and development time is a result of direct causation or a fortuitous relationship through an unspecified variable. Olmo *et al.*[8] suggested that genome size is linked to development rate in birds and Vinogradov[9] reported a relationship between resting metabolic rate and genome size within passerine birds. A link between genome size and

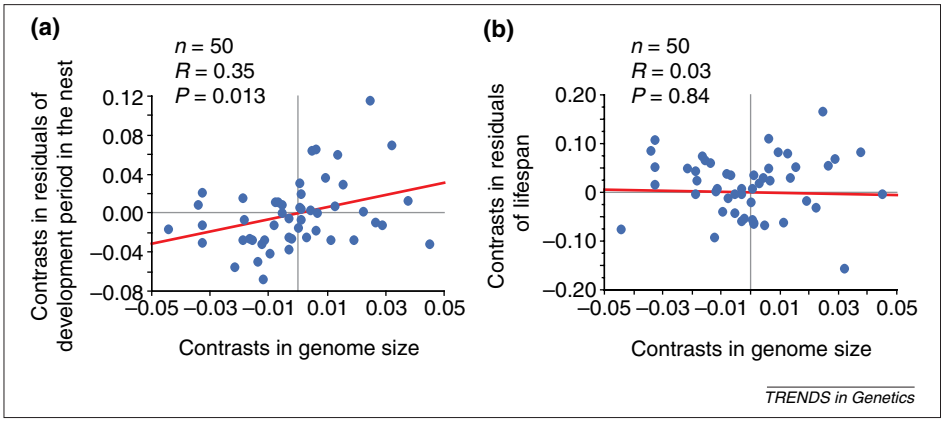

Fig. 1. Relationship between independent contrasts in genome size and (a) development period in the nest – from eggs being laid until fledging (controlled for body mass using residuals of linear regression), and (b) lifespan (controlled for body mass using residuals of linear regression).

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development time is plausible, as the time taken for DNA synthesis and the duration of the cell cycle during development depend on genome size[10].

Serge Morand*
Laboratoire de Biologie Animale (UMR 5555 CNRS). Centre de Biologie et d'Ecologie Tropicale et Méditerranéenne. Université de Perpignan, 66860 Perpignan Cedex, France.
*e-mail: morand@univ-perp.fr

Robert E. Ricklefs
Dept of Biology, University of Missouri-St Louis, 8001 Natural Bridge Road, St Louis, MO 63121-4449, USA.

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Response from Pat Monaghan and Neil B. Metcalfe

We found a significant positive correlation between genome size and longevity in birds[1], whereas a subsequent paper[2] found no such relationship. There are at least three possible reasons for this discrepancy. First, in our analysis we used maximum recorded lifespans of wild birds, whereas Ricklefs and Scheuerlein[2] based their estimates of longevity on captive (zoo) animals. Neither source of data is perfect. We accept that there could be some inaccuracies in using records from ringing studies of wild birds. But it is equally true that the wear and tear of natural life, and hence the age to which zoo animals live, can be artificially altered to an unknown degree by their diet, minimal activity rates, standard of veterinary care and variable opportunity to breed. Moreover, sample sizes of documented lifespans for a species in captivity can be very small (e.g. $n = 20$ individuals[2]), and all the data for a species can come from a single zoo and hence be based on limited environmental and genetic variation.

Second, the range of species included in the two papers was limited by the data available, and this range is unlikely to have been the same (Ricklefs and Scheuerlein do not indicate which species they included, but it is likely that their dataset was biased towards parrots and other typical aviary birds, groups which were absent from our sample). It is clear that patterns relating to genome size in one subset of the avian taxon[3] might not be present in birds as a whole[4], and so it is conceivable that different sub-sampling of avian groups yielded contrasting results.

The third, and perhaps most likely, explanation is the choice of analytical approach, because the results of comparative studies can be heavily influenced by the precise method of statistical analysis. Ricklefs and Scheuerlein did not correct for phylogenetic relationships and treated each species as an independent data point, whereas we used the more robust approach of phylogenetically independent contrasts (PICs). In an interesting re-analysis of a subset of our own database, Morand and Ricklefs (this issue) have now shown that they obtain the same result as we did when using our approach of examining variation at the family level and above, but they fail to find a relationship between genome size and lifespan at the species level, with or without the use of PICs.

This valuable comparison suggests that the discrepancy between the two studies does indeed rest with the taxonomic level at which the analysis is conducted. Morand and Ricklefs then suggest that ours is the less reliable result, because it is not found at the level of the species. They imply that our result is a chance outcome, possibly because of a few species (presumably distorting the overall pattern). However, because our analysis is actually based on a larger sample size (67 species) than either Morand and Ricklefs (58 species) or Ricklefs and Scheuerlein (53 species), it seems unlikely that our result is the one most influenced by a few atypical species. Moreover, it is commonly found in comparative analyses (including those investigating genome size[4]) that a pattern is stronger at higher taxonomic levels, but this is attributed to the much greater signal-to-noise ratio in contrasts between less-closely related taxa, which would make it easier to detect real trends[5]. Analyses at the family level and above could therefore be more reliable than those that compare among species, because of the greater influence of measurement error in comparisons among close relatives[5]. Both of these factors suggest (but do not prove) that our result is the more robust.

Finally, the relationship between resting metabolic rate and genome size, used by Morand and Ricklefs in support of the argument for a link between genome size and development rate, has only been found in one order of birds[3] and is not evident at any taxonomic level (from species to order) when the analysis is broadened to include a fuller range of bird species[4]. Equally plausible links could be constructed between genome size and longevity, but we agree with Morand and Ricklefs that correlational studies will not prove causation. Nonetheless, we hope that they will prompt further investigations into the links between genome size, lifespan and development rate.

Pat Monaghan*
Neil B. Metcalfe
Division of Environmental and Evolutionary Biology, Graham Kerr Building, Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow, UK G12 8QQ.
*e-mail: p.monaghan@bio.gla.ac.uk

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