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Genome size is thought to be independent of organismal complexity, but as a cell component is subject to selection. Andrews *et al.* (2009) and Organ *et al.* (2007) present cases of genome size evolution through natural selection. Both suggest that reduction in genome size is linked to an increase in active metabolic rate (AMR) due to flying. Yet the latter suggest that the avian small genome size was achieved by selection acting on standing variation in non-avian reptiles rather than a synapomorphy acquired by flying birds.

Both studies present similar approaches but their objectives and scopes are different. Andrews *et al.* (AE) inferred genome size using densitometry of erythrocytes, measure nucleus, cell and cytoplasm area in 74 temperate passerine birds. AE hypothesized that smaller genome is necessary for flying birds; as smaller cells, with smaller genomes, increase density of hemoglobin molecules per volume and thus facilitate respiration. AE used a body-size-unrelated measurement, wing loading index (WLI), as a surrogate for AMR. Similarly, Organ *et al.* (OE) measured osteocytes size in extant vertebrates and generate linear regressions with genome size. As cell size was an adequate predictor of genome size; OE estimated genome size in extant and extinct reptiles, including birds and inferred the ancestral character state.

A common ground of these studies is the acceptance of cell-nucleus-genome size relationship, yet there is unaccounted variation in their regression models which may introduce uncertainty in their overall trends. Since OE work is designed to correlate cell size with genome size and make predictions on the ancestral state, this work is likely to be less affected by the effects of unaccounted variance accumulation after each correlational step (*i. e*. OE make a direct correlation, one step). Conversely, AE uses mean values of genome size to link them with wing parameters, yet it is uncertain what is the fate of the genome size variance derived from densitometry measures and if taken into count would unaltered confidence in their trends. More importantly, Fig. 2 shows genome size variation among passerine families. The graph shows the standard error (SE) bars, which provides a measure of error around the standard deviation (SD) but not the mean value. Although both are correlated, ES are drawn from a decrease variance (*e.g*. ES= SD/√sample size; Sokal and Rohlf 1995). Note that if the graph is drawn with SD and the Y axis is set to zero, differences among families become less clear and the error bars will cover much of the X axis. Additionally, families with low genome sizes (< 1.25pg) are sample in one species; while families with higher genome sizes were sampled in multiple species. Thus genome size at the family level spuriously increases with increasing sampling. Altogether this suggests that associations (WLI and genome size) at the family level becomes non significant, a trend noted by AE “**though this became non-significant due to the smaller sample size (r=0.31, p=0.2, n=19)”.**

OE suggest that extinct reptiles had small genomes thus was ancestral, decreasing the role of selection in driving the trait evolution. Surprisingly, OE does provide evidence of a salient difference between reptile and mammal genome size, which would merit further studies to see if such increase is correlated with a mammal life history trait or if relaxation of selection for smaller genomes is the cause. Conversely, AE report an earlier correlation with body size in birds (Gregory 2002). It would be desirable to see if in the passerines studied, the linear relationship with body mass equally explains the cell size and thus the genome size. Preliminary work has shown that body size correlate with metabolic rate (Nagy 1987, Glazier 2008) and cell size in fast evolving cells (including erythrocytes) (Savage *et al.* 2007). Alternatively, it is known that sister species with similar morphologies are prone to niche diversification, including significantly different metabolic rates (Clarke 1999), which affect other molecular processes (*e. g.* nucleotide substitution rate; Gillooly *et al.* 2005).

Both studies illustrate genome size evolution in vertebrates. AE claim that WLI is a good predictor of genome size. Yet it would be informative to see if other non-flying organisms with high AMR are subjected to similar levels of genome reduction or if small genomes in birds are link to other trait yet to be found. Although densitometry is a widely accepted way of estimating genome size, until more data is available the correlation between AMR and genome size evolution and the possible case of adaptation in avian systems will remain contentious. Yet, the methodology presented by both studies could be used to infer cases of polyploidy in non-model organisms or at least as a preliminary survey to generate testable hypothesis.

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