Correlations between genetic and behavioural dissimilarities in wild chimpanzees (Pan troglodytes) do not undermine the case for culture

1. INTRODUCTION
A key issue in the ongoing debate about the existence of culture in chimpanzees is the nature of the behavioural differences documented among wild populations. Some argue that many of the behaviours in question are socially learned and that the interpopulation differences can be considered cultural [1–4]. Others contend that a genetic origin for the behavioural differences cannot be dismissed, because many of the behavioural patterns occur in a single subspecies, and genetic studies suggest chimpanzee subspecies have been genetically isolated for hundreds of thousands of years [5].

Recently, Langergraber et al. [6] have made what appears to be an important intervention in this debate. Langergraber et al. do two things in their paper. One is criticise two studies we have reported that refute the genetic hypothesis [2,3]. The other is describe new analyses that they argue show 'genetic dissimilarity cannot be eliminated as a playing a major role in generating group differences in chimpanzee behaviour' (p. 408). As such, Langergraber et al.'s piece seems simultaneously to weaken the case for the culture hypothesis and to strengthen the case for the genetic hypothesis.

Here, we show that Langergraber et al.'s paper does not change the debate in this way. We begin by explaining why Langergraber et al.'s criticisms do not invalidate our findings. We then demonstrate that, contrary to what Langergraber et al. claim, their analyses do not strengthen the case for the genetic hypothesis.

2. LANGERGRABER ET AL.'S [6] CRITICISMS OF LYCETT ET AL. [2,3]
In the studies we reported in 2007 and 2009, we made use of the fact that there are greater genetic differences between chimpanzee subspecies than within them. We reasoned that, if the hypothesis that genes drive the behavioural differences among wild chimpanzee groups is correct, the behavioural data should mirror the genetic data in terms of structure. With this in mind, we carried out two cladistic analyses of Whiten et al.'s [1] behavioural dataset. One analysis included groups from multiple subspecies. The other focused on groups from just one subspecies. We then measured the fit between the trees and the data. In our 2007 study, we used the Retention Index to do this; in our 2009 study, we used the permutation tail probability test and the phylogenetic bootstrap. Both studies showed that the goodness-of-fit values for the single subspecies tree were higher than the goodness-of-fit values for the multiple subspecies tree. As this is the inverse of the predicted pattern, we concluded that the analyses refuted the genetic hypothesis and, by extension, supported the culture hypothesis.

Langergraber et al.'s first criticism is that there is no way of assessing the statistical significance of a difference in phylogenetic structure measures, and therefore no way of knowing whether the difference is meaningful. This argument would hold if we had no expectations about the direction of the change in phylogenetic structure required to support the genetic hypothesis. However, such is not the case. Phylogenetic analyses of chimpanzee genetic data have consistently yielded results suggesting that if the genetic hypothesis is to be supported, behavioural data should exhibit decreased phylogenetic structure when data from a single subspecies are analysed as opposed to when two subspecies are analysed. Hence, even if the two trees were indistinguishable in terms of structure, this would still be inconsistent with the predictions of the genetic hypothesis. As our analyses consistently returned higher measures of phylogenetic structure for the single-subspecies sample than for the multi-subspecies sample, they unambiguously refute the genetic hypothesis.

Langergraber et al.'s second criticism concerns our assumption that the behavioural data should exhibit greater phylogenetic structure when groups from multiple subspecies are analysed than when groups from only a single subspecies are analysed. Langergraber et al. argue this assumption is flawed because simulations carried out by Nunn et al. [7] show that if the rate of evolution is sufficiently high, goodness-of-fit measures can be higher for a subset of the branches of a tree than for the entire tree. This criticism is erroneous. For the purposes of testing the genetic hypothesis, what matters is the relationship between the behavioural data and the genetic data. Under the genetic hypothesis, a high rate of evolution should impact both genes and behaviour because the two are linked. Accordingly, any difference in degree of phylogenetic structure among parts of a tree should be found in both the genetic and behavioural data. Since our analyses of the chimpanzee behavioural dataset do not conform to phylogenetic predictions based on previously published phylogenetic analyses of chimpanzee genetic data, our results do indeed fail to support the genetic hypothesis.
Langergraber et al.’s third criticism is that we tested the genetic hypothesis only by analysing the behavioural patterns en masse but did not examine the distribution of individual behaviours. Analysing individual behaviours is a potentially informative approach. However, the lack of analyses of individual patterns is not a reason to disregard our findings. Proponents of the genetic hypothesis have argued that entire suites of traits correspond to subspecies groupings [5]. Thus, treating the behaviours as a group for the purposes of testing the genetic hypothesis is valid.


Langergraber et al. reported three analyses that they claim test the genetic hypothesis—one primary analysis and two secondary analyses. They used a modified version of the Whiten et al. [1] dataset. In their primary analysis, Langergraber et al. employed the Mantel test to assess the significance of correlation between genetic dissimilarity and behavioural dissimilarity. In the first of the two secondary analyses, they identified genetically indistinguishable populations and calculated the number of behavioural differences between them. In the remaining analysis, they employed the Mantel test to assess the relationship between behavioural dissimilarity and genetic dissimilarity for each behavioural variant.

Langergraber et al.’s primary analysis returned a significant correlation between genetic and behavioural dissimilarities. In the first of the two secondary analyses, behavioural differences were correlated with genetic differences in 47.4–86.8% of comparisons. In the other secondary analysis, genetic and behavioural dissimilarities were found to be significantly correlated in seven of the 38 cases (table 2). Based on these results, Langergraber et al. argued that genetic differences cannot be ruled out as playing a ‘major role’ (p. 414) in generating differences in the behaviour of chimpanzee groups.

Langergraber et al.’s results seem to contrast strikingly with the results of our 2007 and 2009 studies. However, the difference is illusory. This is because the method Langergraber et al. employed in their primary analysis can only distinguish between the genetic hypothesis and the culture hypothesis if genetic and behavioural dissimilarities are uncorrelated. Genetic and cultural transmission can be expected to often parallel each other across time and space. Thus, finding a significant correlation between genetic and behavioural dissimilarities is not informative about the causes of the behavioural differences. It may indicate that the differences are genetic, but it is also possible that the differences are cultural. Given this ambiguity, it is inappropriate to conclude that Langergraber et al.’s primary analysis shows that genetic differences cannot be excluded as a potential cause of the behavioural differences among chimpanzee groups. The appropriate conclusion to draw is that the results of the analysis are inconclusive owing to methodological limitations.

Langergraber et al.’s secondary analyses also suffer from the limitation that a correlation between genes and behaviour is consistent with both the genetic hypothesis and the culture hypothesis. Thus, the significance of these analyses also differs from what Langergraber et al. suggest. Langergraber et al. take the results quoted above to indicate that genetic differences cannot be excluded as a potential cause of the behavioural differences. However, it is only in cases where genetic and behavioural dissimilarities are uncorrelated that the results are informative. Thus, the correct interpretation of the first of the secondary analyses is that 47–87% of the comparisons were inconclusive owing to methodological limitations, while the remaining 13–53% refuted the genetic hypothesis. Similarly, the correct interpretation of the other secondary analysis is that the results for seven behavioural patterns (18% of the total) were inconclusive owing to methodological limitations, while the genetic hypothesis was not supported in the other 31 behaviours. Hence, Langergraber et al.’s secondary analyses do not add weight to their contention that genes cannot be excluded as a cause of the behavioural variation among wild chimpanzee populations. Indeed, in the few instances where Langergraber et al.’s results shed any unambiguous light at all on the causes of chimpanzee behavioural variation, they are supportive of the cultural hypothesis for those behaviours.

4. CONCLUSIONS

We have made two points in this comment. The first is that Langergraber et al.’s suggested ‘limitations’ of our 2007 and 2009 studies in no way invalidate our findings that genetics plays only a minor role (if any) in determining chimpanzee behavioural variation. The second is that the majority of their own results are ambiguous on this point, and in the small number of cases where their results are unambiguous they support the culture hypothesis. As such, in the light of what has been learned over the last 40 years—and the analyses we have presented previously—the majority of evidence is consistent with the cultural hypothesis for chimpanzee behavioural variation. This includes the results of Langergraber et al. and claims of a potential ‘major role’ for genetic causation are inconsistent with the available evidence.

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