

Reply to Bowles (2008)

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In response to Bowles (2008), we wish above all to reiterate one of the main points of our original discussion (henceforth LDK 2008), which is the following: If there is to be a field of biolinguistics that makes a useful contribution to our understanding of the human language faculty, then it is important to adopt Boeckx & Grohmann's (2007) 'strong' sense of biolinguistics. We are naturally pleased that our work has elicited detailed comment from an adherent of 'weak biolinguistics', but we feel that, in his eagerness to equate biolinguistics with Minimalist research in formal linguistics and to evaluate our work according to the standards of that research paradigm, Bowles has missed our point about the need for genuinely interdisciplinary investigation. We are well aware of the logical problems associated with conclusions based on correlations. However, as we tried to make clear in LDK, the consequence we draw is that we need to look for evidence in other sources of data, not (as Bowles does) merely think harder about the logic of our claims.

When Bowles says (p. 247) that biolinguistic research "still faces classic problems related to the issues of correlation and causality, evidence, counterexample, and refutation", he ignores our general suggestion (LDK 2008: 122) that we need to bring together "linguists and others *in equal measure*, making use of their respective methodologies with a full understanding of their assumptions, and trying to resolve any incompatibilities using shared standards of falsifiability and argumentation". When he rightly points out (p. 248) that there is "no clear way in which to distinguish the natural development of tonogenesis in languages with speakers who do not have the muted [sic] allele pairs from the development of tonogenesis in languages with speakers who do have the muted allele pairs", he ignores the fact that nothing in our work suggests that there should be. When he wonders (p. 249, fn. 3, and again p. 250, questions (1c) and (1e)) "what would constitute a genuine counter-example" to our claim, he is thinking in terms of the kinds of theoretical enquiry in which counter-examples play an important role in shaping conclusions; he ignores Dediu & Ladd's (2007: 10947) explicit suggestion that their correlational finding "warrants future experimental work, which will help test and refine the hypothesis of a causal effect".

A more concrete problem with Bowles's discussion is that, despite his disclaimers, many of his points seem to be based on the assumption that there are specific genes that code for specific linguistic features in the individual. Among

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the issues he considers at some length is whether positing a correlation between a typological feature and a genetic feature “assume[s] that the typological feature has been around approximately as long as the genetic feature it correlates with” (p. 250, question (1a)). This question reveals a profound misunderstanding of how genetics works. It seems pretty clear, for example, that the *FOXP2* gene makes some essential contribution to human linguistic abilities, but *FOXP2* has been “around” for millions of years and is found in many other species without allowing any of those other species to talk. The phenotypic effects of a gene are highly dependent on context, where context includes the rest of the genome, the physical environment, and (in the case of humans) culture. This is part of the reason that it is still far from clear exactly what *FOXP2* does to facilitate language in humans.

Note in this connection that Dediu & Ladd and LDK suggest a number of general cognitive and perceptual differences that might be relevant to a bias for or against linguistic tone, including phonological working memory, low-level pitch tracking, and the ability to process rapid sequences of sounds. These do not appear to be the kinds of traits Bowles has in mind when he talks about “possible genetic factors related to UG principles and parameters” (p. 246) or about linguistic features being “expressed” (p. 252) or “somewhat actively constant” (p. 250, question (1b)) in a population. They are, however, the kinds of differences that can be investigated experimentally and related to observable differences in brain anatomy and physiology, and are biologically far more plausible candidates for the substance of the hypothesized bias than a specific instruction to the language acquirer to assume that the language they are exposed to is tonal. We also note that Bowles seems not to appreciate the importance of the fact that the correlation under discussion is between genetic variation and linguistic variation *in populations*. In both the original Dediu & Ladd paper and in LDK, we went out of our way to emphasize that the contribution of intergenerational transmission of language is essential to any proposed link between population genetics and linguistic typology. No specific *linguistic* predictions about individuals are implied by our work.

Nevertheless, Bowles does raise one important issue that is primarily linguistic, concerning the historical stability of typological features and specifically the historical stability of tone (p. 251, question (2a)). If the distribution of tone (or any other typological feature) is affected by a genetically-mediated bias, it is reasonable to expect that it may be more stable over time. That is, once tone is present in a genetically predisposed group, it should be less likely to disappear through the ordinary mechanisms of language change; by the same token, a language that lacks tone should be less likely to acquire it through those mechanisms if it is spoken by a group genetically disposed against it. Bowles argues that these expectations are not met: Tonogenesis and tone loss, he says, are as common as any other historical change, and the idea of a genetically-mediated bias is therefore problematical. But the idea that tone comes and goes like any other typological feature is actually open to discussion, *pace* Bowles and the authorities he cites. For one thing, the languages of sub-Saharan Africa, across three major language phyla, are overwhelmingly tonal, and for most of them there is no evidence that they have ever been anything else. Loss of tone in

Swahili, for example, is relatively recent and almost certainly related to contact and use as a lingua franca. More generally, it may be important to distinguish between the structural pressures that bring about tonogenesis and the long-term historical developments that follow. In East and Southeast Asia, it is generally accepted that many previously non-tonal languages rapidly became tonal two or three thousand years ago (e.g., Haudricourt 1954), and tone is now central to the phonology of most of these languages. In Northern Europe, by contrast, it is similarly uncontroversial that some sort of tonogenesis took place about 800 years ago, yet tone remains marginal. Norwegian probably has the best claim of any European language to be called tonal, but it is an obvious typological oversimplification to put Norwegian in a class with Chinese, and there are researchers (e.g., Morén 2005) who argue that the Scandinavian languages do not actually have lexically-specified tone at all.

These considerations suggest a refinement of what Bowles says about the historical stability of tone: Tonogenesis itself may indeed be a rather ordinary historical process of phonologization or secondary split, but the thoroughgoing incorporation of tone into a language depends heavily on other factors — almost certainly including areal language contact, and possibly including genetically-mediated biases. The idea of drawing such a distinction — between structural triggers for phonologization of phonetic differences and the long-term establishment of new phonemic contrasts — is discussed by Kiparsky (1995: 655ff.), who specifically (citing Svantesson 1989) mentions tone as a likely case in point. If some such distinction is valid, then Dediu & Ladd's hypothesis suggests a historical account along the following lines. Tonogenesis 'happened' in Southeast Asia and in Northern Europe, in both cases through well-established mechanisms of diachronic change. In Southeast Asia, the population genetic environment was favourable, and tone took hold and spread to become a thoroughly ingrained feature of the phonology of the languages involved. In Northern Europe, the population genetic environment was unfavourable, and tone remained marginal and continues to struggle to this day.

It is thus possible that typological change involving tone is different from typological change in, say, word order. This is a matter that can best be studied on the basis of descriptive and historical linguistic work, and typological theorising about the nature of tone. But such research is not biolinguistics: A finding that tone is exceptionally stable in Africa, or that tonogenesis happens regularly everywhere but only catches on in certain areas, might be *consistent* with the Dediu-Ladd hypothesis, but on its own would do nothing to prove it. If we are serious about learning more about the biological foundations of language, we have to integrate what we know about language with what we know about biology. Research into the formal properties of language is useful and important, but describing it as "biolinguistics" is just wishful thinking.

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