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Linguistics: Evolution and Language Change

Linguists have long identified sound changes that occur in parallel. Now novel research shows how Bayesian modeling can capture complex concerted changes, revealing how evolution of sounds proceeds.

Claire Bowern

English speakers who study languages such as German, French, or Spanish are accustomed to coming across words that are similar to their English counterparts. For example, many words that start with *p* in English start with *pf* in German, such as *plum – Pflaume, pan – Pfanne, penny – Pfennig*, and so on. Sister languages show many such regularities, and this has formed the cornerstone for research on language change for nearly two centuries [1,2]. These regularities have allowed linguists to discover many of the processes of language evolution, and how language evolution may be similar to biological evolution. Now, in this issue of *Current Biology*, Hruschka and colleagues [3] have identified regular sound change as a process similar to concerted evolution in biology. They provide the first statistical model which allows us to study the properties of regular sound change systematically, as well as to compare it to concerted evolution.

How Sound Change Works

All languages have a set of distinctive sounds, called 'phonemes'. These are abstract sound categories, which in combination make up words. The word *pat*, for example, has three phonemes (*p*, *a*, and *t*). The substitution of one phoneme for another changes the meaning of the word, or turns a word into a non-word. For example, the difference between *pat* and *cat* is the first phoneme (*p* in the first case, *k* in the second).

Phonemes are articulated in different ways. The realization phonemes change according to the position at which they occur in the word, the surrounding phonemes, and physiological traits of the speaker pronouncing the word. For example, the pronunciation of the /k/ phoneme in *cat* is different from the same phoneme in *key*. In the latter word, the front vowel pulls the tongue blade forward, leading to a more forward pronunciation. Aspiration is another example. Consider the difference between the *t* in *pat* and the *t* in *tap*. In the second case, the *t* has a puff of air (called 'aspiration') which is absent from the *t* in *pat*. However, no English speaker would consider that *pat* and *tap* don't otherwise have the same phonemes. Finally, the distinctive realization of phonemes is what produces different accents. The distinction between phonemes and their realization is somewhat akin to the genotype–phenotype distinction in biology.

The pronunciation of phonemes can change over time. This is called 'sound change'. Within a language, individuals have different realizations of phonemes. These realizations are subject to selection pressures at both the individual and population level [4]. For example, some variants undergo positive selection and spread because they are easier to perceive. In our example of the different realizations of *k* above, for example, a fronted *k* before a front vowel enhances the cues for the following vowel. Other variants may be positively or negatively selected because they are associated with particular social groups [5]. Over time, these changes may lead to changes in the phonemes themselves. In my German example above, for example, *p* has become an affricate *pf*.

Linguistics and Biology

At this point, biologists will no doubt be thinking of numerous parallels between linguistic and biological evolution. Words are somewhat like genes: they are transmitted vertically, and the nucleotide or phoneme sequences they comprise can change individually or concertedly. There are many broad similarities between linguistic and biological evolution [4,6]; for example, both involve homologous units which descend from common ancestors, which allow us to trace the history of those descent patterns using evolutionary models.

'Concerted change' is central to historical linguistics. It was the regularities in correspondences which first allowed linguists to provide principled definitions of language relationships [7], by showing that such changes lead to systematic similarities which could not arise by chance. Sporadic, irregular changes do occur, but they are concentrated in certain sound sequences, or are the result of changes in word structure, or reflect loans from related languages.

Though the parallels between linguistic and biological evolution have been discussed since Darwin [8,9], until the last ten years the two disciplines have used different

methods; this has obscured both similarities and differences between the two evolutionary systems [6]. More recently, more historical work has taken advantage of quantitative biological and particularly phylogenetic methods [10–12], but much of that work looks at how words change, not at sounds as the building blocks of words. As a linguist, I see these approaches not as replacing more familiar types of argument, but rather expanding the questions we ask about language change.

Concerted Evolution

The model of Hruschka *et al.* [3] works on aligned series of words from languages of the Turkic family. The model simultaneously estimates character alignment — that is, which sounds correspond across languages — and the sound changes that such an alignment entails. The sound changes are classified as regular or sporadic and regular sound changes are assigned to positions in a phylogeny. Parameters for the model are estimated using Monte Carlo Markov Chain (MCMC) methods. Within Turkic, the phylogeny derived from the concerted-evolution model conforms fairly closely to the linguistic family tree accepted by linguists. The sound changes derived by the model are all well-known from study of language families other than Turkic [13], reinforcing plausibility of the model. The model can also be used to study the details of sound change. For example, nearly 80% of the regular changes involving consonants (and 70% of those involving vowels) were changes between a single feature, such as voicing, height, or constriction. This is unexpected but plausible, assuming that sound change proceeds by gradual, incremental changes in pronunciation targets, as has been claimed [14,15].

The methods of Hruschka and colleagues [3] expand historical linguistics in several ways. While previous research relies on regularities in change to identify families, evaluation of the evidence has been subject to expert agreement rather than statistical rigour. Bayesian identification of concerted evolution could be useful for statistical detection of regularities, for example in long-distance relationships. The data used as proof of concept here are from Turkic languages, which are closely related to one another and show extensive regularities even at cursory examination [16]. It would be fruitful to see whether this approach finds statistical support for some of the claims of more remote relationships, such as those that are claimed to link the languages of Eurasia [17].

This approach is also likely to be particularly useful in cases of language hybridization. If a language borrows words extensively from a relation, 'doublets' are created – these are multiple correspondence sets. For example, English and Latin have *f –p* correspondences in words such as 'foot' (: *ped-*) and 'father' (: *pater*); but there are also *p–p* correspondences in other words such as 'paternal' and 'pedestrian', where English has borrowed the Latin word. Quantitative evaluation of both the frequency of the two correspondence sets, as well as the types of words which show each correspondence, would allow us to better elucidate borrowing histories. For the English–Latin case, documentary evidence is sufficient that we know the histories of these words already, but for the more than 95% of the world's language without long written histories, this approach would be invaluable.

Finally, this model will be invaluable for investigating the relationships between linguistic evolution and lineage branching. Biologists might be surprised to learn that linguists typically make no distinction between anagenetic and cladogenetic changes, and while we know a great deal about how changes advance through languages, general models of cladogenesis are lacking; that is, linguists implicitly assume that in the normal course of events, changes accrue across a community of language speakers. Cladogenesis arises when, for some reason, those changes do not spread to a subsection of the population. Cladogenesis in linguistics cannot simply be the result of migration, as many language families have evolved while their speakers have remained in contact with one another [18]. But we do not yet understand whether some changes spread less easily through communities, and are therefore more likely to lead to cladogenesis; or whether cladogenesis occurs in the presence of rapid parallel changes. A formal model of concerted evolution in sounds allows us to investigate how this interacts with tree structures. For example, Atkinson and colleagues [19] found that changes involving word replacements occur in punctuational bursts – is the same true of sound changes? That is, do we find more change occurring on lineages that are also diverging?

Hruschka *et al.* [3] state that their model can be used "wherever discrete elements – such as genes, words, cultural trends, technologies or morphological traits – can change in parallel within an organism or other evolving group." This gets to the heart of problems in comparative evolution. We have no clear evidence that change within different parts of language work in the same ways. But now we have a way to generate the hypotheses to find out.

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Epithelial Cell Division: Aurora Kicks Lgl to the Cytoplasmic Curb

The *Drosophila* neoplastic tumor suppressor Lethal giant larvae (Lgl) regulates apico–basal polarity in epithelia as well as the asymmetric segregation of cell fate in neural progenitors. Two new studies uncover a new facet of its regulation in epithelia, where Aurora-dependent phosphorylation triggers Lgl dissociation from the basolateral cortex to facilitate planar orientation of the mitotic spindle.

Yu-ichiro Nakajima[1] and Matthew C. Gibson[1,2,*]

Epithelia are the fundamental building blocks of animal organ and appendage structures, and thus play prominent roles in both development and disease. In broad terms, epithelial architecture requires the localized assembly of adhesive junctions in concert with the apico-basal polarity of each cell. Considering the complexity associated with establishing and maintaining this degree of structural order, how do proliferating epithelial cells maintain polarity and tissue integrity while cyclically disassembling their interphase morphologies, rounding up, and dividing into co-equal daughters? One key hypothesis is that during division, the mitotic spindle is oriented to the plane of the epithelium in order to facilitate the conservation of cell junctions and the correct integration of post-mitotic cells into the monolayer. While both classic papers and more recent studies have implicated polarity determinants as cues for planar spindle orientation [1], precisely how epithelial polarity is modulated during mitosis *in vivo* remains poorly understood. Addressing this problem head-on, two reports in this issue of *Current Biology* reveal a novel mechanism for the mitotic regulation of the conserved polarity regulator Lethal giant larvae (Lgl) in *Drosophila* epithelia [2,3].

Lgl was the first reported tumor suppressor in *Drosophila*, named for mutant larvae that exhibit dramatic overgrowth and a corresponding disruption of tissue architecture in the imaginal discs and neuroblasts [4]. Similar phenotypes are observed in mutants of *discs large* (*dlg*) and *scribble* (*scrib*), and subsequent work has demonstrated that these three neoplastic tumor suppressor genes function in the same genetic pathway [5]. In epithelia, the protein products of *lgl*, *dlg*, and *scrib* co-localize at the basolateral membrane and work together as a protein complex that controls cell polarity (the Scrib complex) [5]. Consistent with its neoplastic phenotypes, Lgl is implicated in the regulation of apico-basal polarity in epithelia and asymmetric cell division in neuroblasts [6]. In the last decade, further studies have suggested a contribution of Scrib complex mutations to tumorigenesis by investigating *Drosophila* models and by exploring the association of mutations in human orthologs with cancer [7]. Lgl is a cytoskeletal protein that primarily localizes at the cell cortex and plasma membrane, but it is also found in the cytoplasm [6]. In epithelia, basolateral Lgl, Dlg and Scrib regulate cell polarity through mutually antagonistic interactions with the apical Par (Par3–Par6–aPKC) and Crumbs (Crumbs–PatJ–Stardust) complexes [8]. Similarly, during asymmetric cell division of neuroblasts, Lgl targets fate determinants to the basal cortex by mutually inhibiting the activity of the Par complex [9]. Thus, in two very different cellular contexts, the role of Lgl is to restrict the spatial localization and activity of polarity determinants along the cortex and membrane.

The subcellular localization of Lgl is controlled, in part, by aPKC-dependent phosphorylation at three conserved serine residues (S656, S660, and S664) (Figure 1A). Upon phosphorylation, Lgl dissociates from the cell cortex, leading to its cytoplasmic localization and inactivation [10]. In epithelia and asymmetrically dividing neuroblasts, Lgl is excluded from the apical cell cortex by aPKC-dependent phosphorylation, which is necessary to maintain epithelial polarity and direct fate determinants, respectively [10,11]. Interestingly, during neuroblast cell division Lgl translocates from the cell cortex to the cytoplasm at prophase entry [12]. This event, termed Lgl cortical release, is triggered by the mitotic kinase Aurora A (AurA). At the onset of mitosis, AurA activates aPKC by directly phosphorylating Par-6, thus relieving aPKC from negative regulation by Par-6. The mitotically activated aPKC then phosphorylates Lgl and remodels the Par complex [12]. Combined, studies from neuroblast cell division indicate that protein localization is dynamically reorganized to coordinate cell polarity with mitosis. Until now, however, whether and how polarity determinants are remodeled during epithelial cell division has remained poorly understood.

Using genetic analysis and *in vivo* live-imaging, Carvalho *et al.* [3] and Bell *et al.* [2] share the finding that Lgl relocalizes from the cortex to the cytoplasm during mitosis in imaginal and follicular epithelia. How is Lgl relocalization controlled? Like in neuroblasts, cortical release of epithelial Lgl depends on its aPKC phosphorylation motifs. Further, Lgl relocalization is strongly delayed in *aurA* kinase-defective mutants, suggesting that AurA activity is required for Lgl cortical release at