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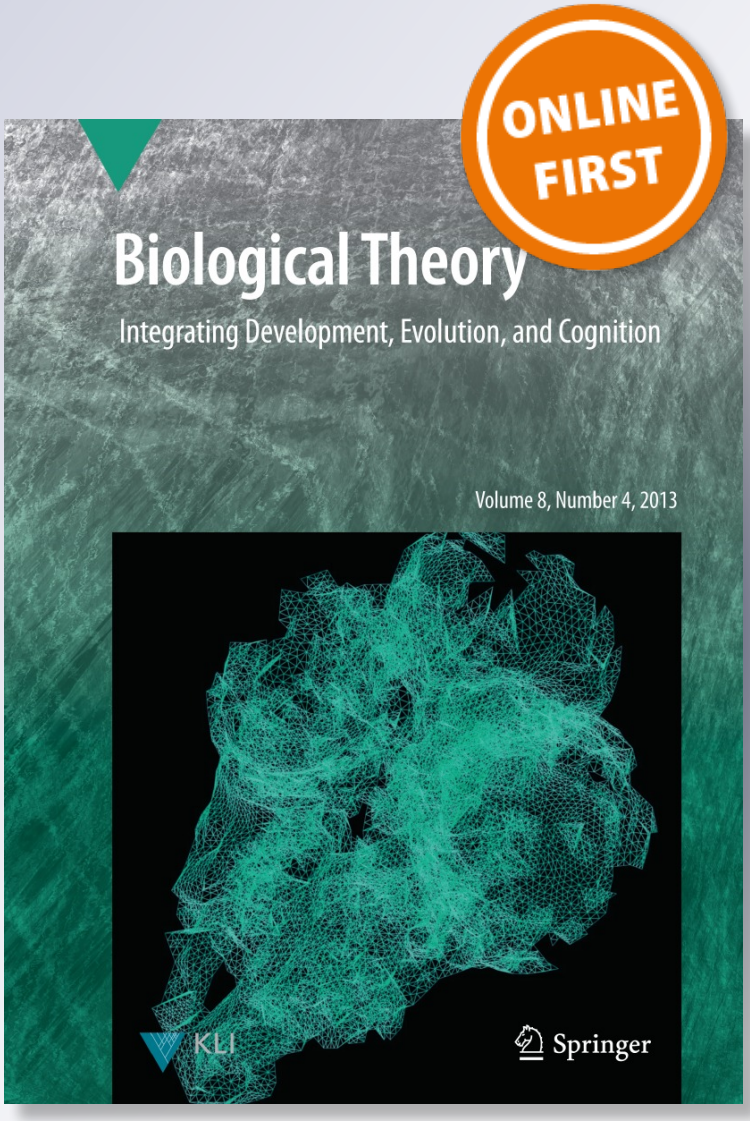

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SHORT ARTICLE

Language Disorders and Language Evolution: Constraints on Hypotheses

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Abstract It has been suggested that language disorders can serve as real windows onto language evolution. We examine this claim in this paper. We see ourselves forced to qualify three central assumptions of the the 'disorders-as-windows' hypothesis. After discussing the main outcome of decades of research on the linguistic ontogeny of pathological populations, we argue that language disorders should be construed as conditions for which canalization has failed to cope fully with developmental perturbations. We conclude that a robust link exists between developmental disturbances and evolutionary history that allows language disorders to be used as real windows onto the evolution of the neuronal substrate of language, and emphasize that our conclusion is more compatible with views of language evolution as the result of reorganizational changes as opposed to radically novel emergences.

Keywords Atavisms · Canalization · Language evolution · Language faculty · Language disorders

Language does not fossilize. Additionally, it is a human-specific phenotypic trait. This is why research on language evolution necessarily relies on indirect evidence, and why inference is a key step if we want to gain a confident view about how language emerged in our clade, and which (proto)linguistic abilities, if any, extinct hominins were endowed with. Evidence of this kind has been the subject of intense investigation in recent years. For instance, Botha (2003, 2006, among others) has extensively written about "windows on language evolution." "A phenomenon X is considered to offer a window on a distinct phenomenon Y if by 'looking at' X it is possible 'to see' something of Y" (Botha 2006, p. 132). Similarly, Johansson (2013) has coined the term "proxy" for language evolution. Proxies are construed as uniquely human features that ideally allow for a two-sided entailment: if they are present one can confidently infer the presence of language; if they are absent, one can likewise infer the absence of language. Many different "windows" or "proxies" have been proposed: symbolic artifacts, complex tools (under the assumption that technological innovations and the spread of the know-how seem to depend on language), fossil evidence of modern behavior (big-game hunting strategies, complex uses of space, seasonal displacements, etc.), brain endocasts, fossil remains of the speech organs, "language genes," and so on. Nonetheless, their real value is hotly disputed. Usually the controversy focuses on the inferential steps needed to render them informative (see, e.g., Botha 2003, 2006).

Among the less explored "windows" or "proxies" are perhaps the so-called "degraded" forms of language (after Jackendoff 1999). According to Jackendoff and others (e.g., Bickerton 1990), they contain remains of previous, less complex, stages in the evolution of language. Pidgins, home signs, child language, interlanguages, and agrammatic speech are commonly cited as examples. More neutrally speaking, these are formally simplified, functionally restricted linguistic systems. As with other "windows," the main criticism around their use (i.e., evolutionary informativeness) concerns the bridging theories that allegedly allow moving from these "degraded languages" to the domain of language evolution (see, e.g., Botha 2006; Botha and de Swart 2009). In the

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absence of further evidence, it is not clear why hominin (proto)languages, if any, should have had (some of) the formal and functional properties that these modern forms of language are endowed with. Ultimately, the bridging theory underlying this claim seems to be grounded on a particular, but still disputable view of evolution, i.e., that ontogeny recapitulates phylogeny (after Haeckel 1866).

Here we will specifically focus on language disorders as putative windows on language evolution. For instance, it has been suggested that aphasic speech offers information concerning a putative stage of protolanguage and that its recovery parallels early language evolution in the species (Code 2011). This would eventually mean that language disorders are, or can be construed as, atavisms that can shed light on the evolution of language in modern humans. We will explore this possibility, but in doing so we will move from the phenotype, i.e., linguistic data, whose analysis seems to be highly controversial, to the biological machinery underlying the faculty of language (brain tissues, neural networks, proteins, genes, and the like), which we find less controversial and more illuminating at present. Many of the mutations that give rise to different language disorders have already been identified (see Benítez-Burraco 2009 or Szalontai and Csiszar 2013 for comprehensive reviews). Furthermore, we know which brain areas are structurally and functionally impaired in people bearing them. For instance, mutations on *MCPH1* (a gene involved in chromosome condensation during the cellular cycle and in DNA repair) reduce human brain size to that observed in extant great apes (Woods 2004). People suffering from microcephalia vera (the clinical condition linked to the mutation of *MCPH1*, but also of other genes such as *ASPM*) have mild to moderate mental retardation and show a delayed speech acquisition (Garshasbi et al. 2006; Passemard et al. 2009). If we eventually proved that microcephaly (or any other regressive trait linked to some other language disorder) is an atavistic trait in modern humans, we would be in a better position to speculate about the primitive features, important for the emergence of language, exhibited by extinct hominins, and ultimately about the way in which the modern phenotype (i.e., a full-fledged language) emerged. In fact, some of the candidate genes for language disorders have been positively selected in our clade. Famously, the "language gene" *FOXP2* gained two non-synonymous changes in the common ancestor of Neanderthals and modern humans (Krause et al. 2007). But interestingly, in some positions Neanderthals exhibited the ancestral allele of the gene *MCPH1*, whose mutation gives rise to microcephaly (Green et al. 2010).

Ontogeny and Phylogeny

Three underlying assumptions converge in the "disorders-as-a-window" hypothesis:

(1) ontogeny recapitulates phylogeny;
(2) language disorders are atavistic phenotypes;
(3) evolution is not unidirectional.

We feel compelled to revisit each of these assumptions, as appropriate qualifications of each lead to a better use of language disorders as windows onto language evolution.

The idea that ontogeny recapitulates phylogeny as such is just plain wrong. At present we cannot go on claiming that the developmental milestones achieved by the child when she acquires language parallel, or are the record of, the milestones achieved by the species as language evolved in our clade. At the very least, this is untenable concerning the biological underpinnings of the faculty of language. Haeckel himself was aware that even at the embryological level recapitulation is neither absolute nor complete, because ontogenetic itineraries are also a consequence of embryonic adaptations and/or displacements of developmental stages in space and time (more on this below). As a consequence, in the absence of further evidence it is not clear what aspects of language can be confidently treated as inheritances, and which ones must be regarded as innovations. In sum, if the so-called biogenetic hypothesis is false as a whole, it should be false as well regarding language (eventually this disqualifies the very existence of "language fossils" in modern languages). At the same time, modern biology has found a deep link between development and evolution. At the very heart of the current Evo-Devo theories we find the claim that evolution is prompted by changes in developmental processes. In other words, ontogeny *creates* phylogeny.

When it comes to language disorders, one must also bear in mind an additional concern: from the discovery that some structural damage in the brain or a gene mutation gives rise to a language deficit in some individuals, one cannot just conclude that this brain region, when unimpaired, or the wild version of the gene plays the opposite role in typically developing people (see Bateson and Mamelli 2007 for a detailed discussion). This possibility has to be probed. Notice as well that as for acquired language disorders (e.g., aphasia) what we actually have is an impaired neuronal device inside an anatomically modern human brain otherwise normally developed and organized. The consequences for language of this loss of function cannot just be conflated to the putative gain of function resulting from this very piece of brain placed in a hominin brain. As for developmental language disorders, the entailment is even more problematic as they cannot be merely construed as a breakdown of a functional faculty of language. Instead they are alternative ways of implementing (or trying to implement) a more or less functional faculty of language at the term of growth. Not surprisingly, in these conditions we usually observe diffuse effects on

the whole brain and on different cognitive processes (at the same time deficits in low-level processes commonly manifest as deficits in high-level capacities). Moreover, disordered systems are adaptive and react to compensate for damages, to the extent that the signals of breakdowns and compensations can be observed at different levels, places, and times during development. Consequently, if we eventually concluded that some pathogenic allele impairs some specific component of language during development (but see Benítez-Burraco and Longa 2011 on the biological unsoundness of such a direct link between the genotype and the phenotype), we could not automatically assume that the wild copy of the gene was once selected to improve some hominin (proto)language with this very component.

A second important concern is whether language disorders can be actually construed as atavisms. Of course, atavisms *do* exist. According to Hall (2010, p. R807) "an atavism is the occasional re-appearance in individual species members of a single generation of a character—a structure or behavior—that is present in all ancestors within the lineage." However, as Hall also points out, "by definition, atavisms can be identified only in a lineage for which we know the evolutionary history." But this is precisely what we want to uncover: we do not know the evolutionary history of language. However, when we move to the biological underpinnings of language, atavistic traits can be more confidently identified. For example, the brains of people suffering from microcephaly can be claimed to be a genuinely atavistic feature. On the one hand, the trait has been sufficiently preserved in our lineage to be identified as atavistic (all extant apes and many extinct hominins have small brains). On the other hand, we can speculate about the developmental program that was modified to give rise to bigger brains in modern humans (and that has been substantially preserved in them). Hence human brains are in essence hypermorphic hominin brains (McKinney 2000), resulting from a late offset of the symmetric cellular division phase of neuronal precursors and of their cytogenesis (Rakic and Kornack 2001). That said, the real problem is, of course, whether brain size is an informative proxy for language evolution (see Balari et al. 2013; and Boeckx and Benítez-Burraco submitted, for relevant discussion).

Finally, the role of language disorders as "windows on language evolution" has been regarded as controversial because of some loose construals of Dollo's law, according to which organisms are unable to return, even partially, to previous evolutionary stages. In other words, even pathogenic ontogeny would not recapitulate phylogeny. This can be true if we think about evolutionary innovations as the accumulative result of many different gene mutations (i.e., it is unlikely that they all can be simultaneously reversed). However, real reversions actually exist in nature [to be fair, Dollo's law was soon reformulated to only account for complex characters that have been lost in evolution and that (allegedly) cannot be regained (Collin and Miglietta 2008)]. Current EvoDevo theory tells us that true reversions can appear if an ancestral developmental program is preserved (but silenced) in the derived populations. In fact, reversions can even promote evolution (Cabej 2012).

Our own claim is that we should analyze the putative role of language disorders as windows on language evolution first of all from this EvoDevo perspective. In doing so we can arrive at relevant insights about the way in which the biological substrate of language has evolved and ultimately about how the language-ready brain appeared in our clade (we find less ideologically loaded than others (e.g., Chomsky's Universal Grammar) this way of referring to the aspect of our biology that allows us to spontaneously develop mental rules that are used for thinking and communicating).

Language Disorders and the Evolution of the Language-Ready Brain

For the sake of the argument we will limit our discussion to developmental language disorders (as we pointed out above, we feel that acquired language disorders are less suitable for evolutionary concerns, as they usually entail the breakdown of a full-fledged linguistic brain). Let us summarize what we actually observe as language growths in pathological populations. Contrary to the simplistic view of the disordered brain as a juxtaposition of impaired and preserved modules, we find diffuse effects on the brain architecture and function, and on diverse cognitive capacities/abilities. Certainly some aspects are more impaired than others, and specific deficits can be identified in each disorder (more frequently in the adult stage), but they usually result from the impairment of low-level, more generalized (as opposed to domain-specific) processes (Karmiloff-Smith 2009). Moreover, disordered brains are not static entities, i.e., they are able to compensate damages at different levels and throughout growth (Sirois et al. 2008). Ultimately, this explains why the linguistic profile of affected people varies across populations and throughout development. Eventually, substantially preserved linguistic capacities can be achieved in spite of deeper cognitive impairments. Importantly, breakdowns and compensations do not occur randomly, to the extent that some defective phenotypes are never observed, while some deficits are repeatedly found in many (if not all) disorders. For instance, problems with inflectional morphology are observed not only in specific language impairment (SLI) (Marchman et al. 1999), but also in people with speech-sound disorder (SSD) (Mortimer and Rvachew 2010), Down's syndrome

(Eadie et al. 2002), and autism (Roberts et al. 2004). This plausibly means that some aspects of language processing are particularly vulnerable to damage. On the whole, this suggests to us that there exist not one, but several ways of implementing a functional faculty of language at the term of growth (see also Hancock and Bever 2013 for discussion).

This complex scene is easier to understand under the umbrella of the EvoDevo approach. Hence, we have observed that in all conditions (pathological or non-pathological) language development is both plastic (i.e., sensitive to environmental change) and canalized (i.e., resistant to environmental perturbation), both robust (i.e., resistant to evolutionary modification) and evolvable (i.e., prompted to evolutionary change). At the same time, certain phenotypes are never seen, while others are always observed (either ontogenetically or phylogenetically or both). Our point is that we must rely on these key EvoDevo concepts (canalization, development plasticity, robustness, evolvability) if we want to probe the putative role of language disorders as a "window on language evolution." In doing so we will rely on these important EvoDevo insights:

- developmental processes are regulated by many different factors (not only by genes!) that interact in a nonlinear way;
- all these factors are equally necessary for the final phenotype to emerge;
- as a corollary, evolution can be prompted by the modification of any of them (Oyama et al. 2001; Griffiths and Gray 2004);
- developmental dynamics tends to strongly canalize development (and in particular, all the existing variation at different levels: molecular, genetic, histological, physiological, etc.);
- as a consequence, diverse genotypes (or even different brain architectures) can render the same phenotype (i.e., similar faculties of language) at the term of growth (the other way around also holds and we refer to it as *developmental plasticity*) (West-Eberhard 2003; Dworkin 2005);
- phenotypic variability is always constrained (i.e., only certain normal, impaired, delayed, or deviant faculties of language emerge at the term of growth);
- this limited set of phenotypes can be properly described as *phenotypic morphospaces* or *adaptive landscapes* (Svensson and Calsbeek 2012).

On the basis of this, we wish to argue that language disorders should be construed as conditions for which canalization has failed to (fully) cope with developmental perturbations (deleterious gene mutations, brain damage, and the like) (Benítez-Burraco 2013). As a consequence, certain developmental milestones are never reached or are instead (incompletely) achieved via compensatory mechanisms. Following a recent model by Gibson (2009), disorders can be also construed as de-canalized conditions, resulting from the uncovering of cryptic genetic variation as a consequence of genomic, environmental, or even cultural perturbations. However, the crucial question is why this is the case, i.e., why buffering mechanisms are unable to deal with these perturbations, and why adaptability is eventually limited (this entailing in turn that some perturbations are never compensated for by developmental dynamics, that certain developmental itineraries are never tracked, and that some phenotypic traits are always disturbed in all conditions). It has been hypothesized that this is probably due to the fact that certain cognitive processes are more vulnerable per se than others to developmental disturbances or to damage because they rely on less resilient neural networks that have less robust compensatory mechanisms. Importantly, there seems to exist an inverse relationship between resilience and evolutionary novelty (Toro et al. 2010). This is an appealing view that robustly links (abnormal) development and evolution at deep (i.e., biologically grounded) levels of analysis.

Notice that one remarkable outcome of the current biolinguistic approach (in the broad sense) to language is that the genetic, physiological, and even cognitive mechanisms supporting the faculty are quite resistant to damage, in spite of language itself being so sensitive to perturbation. Plausibly this is due to the long evolutionary history of the former: after millions of years of stabilizing selection they have become particularly robust. At the same time, our idiosyncratic evolutionary history (characterized by acute population bottlenecks and widespread migratory movements), some specific mutations, and/or some cultural changes disturbed this stable equilibrium (otherwise still observed in primates), de-canalized the whole system, and uncovered all the existing cryptic variation. According to Gibson (2009), this explains why complex genetic diseases are so pervasive and prevalent among modern populations. On our account, these were the changes that brought about modern language. In some sense, modern, full-fledged language can be construed as a de-canalized state. And this is why it is so sensitive to damage (although only to some kind of damages). In other words, the faculty of language is easy to impair because it is an evolutionary novelty. At the same time it relies on robust biological mechanisms that are difficult to disturb and that are able to compensate many kinds of damage because they are significantly older. Plausibly, this explains why the same deficits are found in nearly all disorders and why others aspects of language processing are substantially preserved in all of them. Plausibly also, this provides us with a real "window" on language evolution.

We think that this picture nicely fits fresh views of evolution as the result of reorganizational processes rather than as a product of innovative genes (West-Eberhard

2003), and implicitly, the view of language as a cognitive faculty resulting from the interface of different components (cognitive, neural, genetic) that are not otherwise specifically linguistic (see, e.g., Hauser et al. 2002; Balari et al. 2013; Boeckx 2013). The idiosyncrasy of language would thus mostly rely on the pervasive predisposition of its components to interact whenever growth occurs under the proper amount of linguistic stimuli.

Conclusions

In conclusion, language disorders can constitute real windows onto the evolution of language, but only to the extent that they tell us something about the evolutionary novelty (or ancestry) of the biological underpinnings of the faculty. Still, we think that this is progress. Considering the amount of speculation in the field of evolutionary linguistics, we should welcome constraints on hypotheses of the sort we have outlined in this paper.

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