PERSPECTIVE

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What developmental disorders can tell us about the nature and origins of language

Gary Marcus & Hugh Rabagliati

Few areas in the cognitive sciences evoke more controversy than language evolution, due in part to the difficulty in gathering relevant empirical data. The study of developmental disorders is well placed to provide important new clues, but has been hampered by a lack of consensus on the aims and interpretation of the research project. We suggest that the application of the Darwinian principle of 'descent with modification' can help to reconcile much apparently inconsistent data. We close by illustrating how systematic analyses within and between disorders, suitably informed by evolutionary theory—and ideally facilitated by the creation of an open-access database—could provide new insights into language evolution.

Language is arguably the defining characteristic of the human species, yet the biological basis of our ability to speak, listen and comprehend remains largely mysterious; about its evolution, we know even less. As in other areas of cognition, comparative animal research can yield valuable insights^{1–4}, but the study of the nature and origin of human language poses special challenges. No system of animal communication approaches human language in its complexity, and many of the powerful techniques that have been used in animal studies (for example, gene knockout studies and the 'deprivation studies' used to study how isolated songbirds develop songs) cannot ethically be applied to human beings.

Human developmental disorders could offer special insight into the genetic, neural and behavioral basis of language because they provide a way to study naturalistically what cannot be controlled in the lab. For example, studies of developmental disorders have been particularly prominent in a central issue in cognitive neuroscience: the relation between the biological (and psychological) basis of language and the biological (and psychological) basis of language and the biological (and psychological) basis of other cognitive or neural systems. One classic view holds that language is a "modular system" that should be studied largely on its own terms, and another view is that language is simply a particular byproduct or instantiation of powerful "domain-general" cognitive systems. Advocates of both views have pointed to studies of developmental disorders. One set of studies, invoked by critics of modularity, has shown that impairments in language often co-occur with impairments in other spheres of cognition, such as motor control⁵ and general intelligence. Variability that can be

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attributed to genetics in one domain (say language) also typically correlates strongly with genetically attributable variability in other domains⁶. Such correlations may indicate that language is mediated largely by "generalist genes" and therefore, "genetic input into brain structure and function is general, not modular"⁶.

Advocates of modularity have focused on what we will call dissociability. One prominent case study⁷, for instance, focused on a single 10-year-old child, AZ, with a particular grammatically focused form of specific language impairment. AZ showed a significant deficit in language comprehension and production, while otherwise showing normal cognitive functioning. On tests of auditory processing, analogical and logical reasoning as well as nonverbal I.Q., AZ performed as well as age-matched controls. In contrast, he frequently omitted grammatical inflections (for example, the plural –*s*) and proved unable to use or properly understand subordinate clauses or complex sentence constructions. Likewise, in sentences such as Grandpa says Granny is tickling him, AZ could use context to accurately infer the referent of him (i.e., Grandpa), but where context alone was inadequate, AZ performed at chance. In the sentence Mowgli says Baloo is tickling him, a normal native speaker recognizes that the pronoun him can only refer to Mowgli; conversely in Mowgli says Baloo is tickling himself, a normal speaker recognizes that *himself* must refer to Baloo. Despite normal intelligence, AZ was never able to make such distinctions. The authors concluded that "The case of AZ provides evidence supporting the existence of a genetically determined, specialized mechanism that is necessary for the normal development of human language."

We very much admire both lines of work—the care taken by modularity's critics in investigating the overlap between language and other aspects of cognition, and the detailed case studies of how specific aspects of language can be dissociated within well-defined subgroups, as undertaken by modularity's advocates. (There is also interesting work on the opposite sorts of cases such as Williams syndrome, in which afflicted members have marked deficits in domains such as spatial cognition, but comparatively spared language^{8,9}.) At the same time, the mutual inconsistency of the two conclusions is striking.

One sees a similar lack of consensus in discussion of the significance of the gene *FOXP2*, initially noted in studies of a severe inherited speech and language disorder^{10–12} found in the British KE family¹³. Early debate (before the gene was identified) focused on the specificity of the disorder, which apparently afflicts both language and orofacial motor control¹³. Recent debate has focused on the relation between *FOXP2* and recursion, the ability to embed and conjoin units of a language (such as phrases or words), which underlies much of modern linguistic theory. Advocates of a link between the two have argued that

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FOXP2 may "confer enhanced human reiterative ability [recursion] in domains as different as syntax and dancing¹⁴." In contrast, one recent high-profile paper states that it is highly unlikely that FOXP2 has anything to do with recursion¹⁵. A third perspective¹⁶, meanwhile, uses the wide-ranging effects of FOXP2 expression¹³ to criticize the suggestion⁴ that recursion might be the only mechanism unique to both humans and to language, suggesting that the FOXP2 facts "refute the hypothesis that the only evolutionary change for language in the human lineage was one that grafted syntactic recursion onto unchanged primate inputoutput abilities." Among these three reports, one sees FOXP2 as essential for recursion and by extension language evolution, one sees that FOXP2 as not important for what is unique about human language, and a third provides evidence that recursion is not the only unique contribution to language. As FOXP2 is expressed in many species apart from humans¹⁷, and as the gene is expressed in the lungs as well as as the brain,¹⁸ the situation is clearly complex¹⁹.

In our view, although developmental disorders hold great promise for informing these debates, no firm consensus has yet been reached. We raise these inconsistencies not to cast doubt on the possibility of useful data emerging from developmental disorders, but to suggest that a change in perspective may be necessary.

A more plausible perspective?

We suggest a return to first principles—specifically the logic of evolution, or what Charles Darwin called "descent with modification". As Darwin wrote, "the existing forms of life are the descendants by true generation of pre-existing form." That is, the systems that currently exist have a twopart evolutionary history: something borrowed, something modified.

Although the "survival of the fittest" strand of Darwin's thought has, in the form of adaptationism, often been prominent in evolutionary approaches to psychology, the importance of descent with modification has generally received less attention. A focus on adaptation alone appears to be implicit, for example, in a suggestion from evolutionary psychology that "There is no more reason to expect any two cognitive mechanisms to be alike than to expect the eye and the spleen, or the pancreas and the pituitary to be alike."²⁰ But this position overlooks the importance of the causal mechanism of descent with modification. Language evolved only relatively recently, probably within the last few hundred thousand years, presumably out of other neural substrates. It would thus seem unlikely that language and cognition would really be as distant as eye and spleen (which have been diverging for roughly a thousand times as long).

At the same time, the element of modification implies important evolutionary change, even where two systems may stem from common origins; as a consequence, "generalist genes"²¹ on their own are unlikely to be adequate^{22,23}. Consider for example the forelimb and hindlimb. There is enormous overlap in which genes are expressed²⁴. Yet that overlap is less than total, and differences between hindlimb and forelimb are presumably largely attributable to the small genetic differences. In a similar way, differences between language and cognition may depend on small amounts of change, genetic, neural and psychological, relative to a largely shared common background.

Seen in this light, one might imagine three caricatures (**Fig. 1**), depicting how the different perspectives on language evolution would predict the relations between the genes, neural substrates and cognitive substrates underlying language and the full range of other cognitive systems. In our view, the logic of descent with modification clearly points to the relation seen in **Figure 1c**.

Provisional acceptance of this perspective has important implications, both theoretical and empirical, for how the nature and origin of language should be studied.

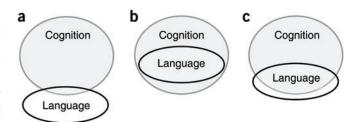


Figure 1 The relation between language and cognition under three evolutionary scenarios. (a) The genes, neural substrates or cognitive substrates underlying language are largely separate from the genes, neural substrates or cognitive substrates underlying other neural systems. (b) A scenario in which language would depend on a subset of the machinery used for building cognition in general. (c) A scenario, analogous to the hindlimb/forelimb case, in which the machinery underlying language would largely but not entirely overlap with the machinery that underlies other neural/cognitive systems.

On the theoretical side, the logic of descent with modification speaks against the two extreme views that currently characterize the debate. On the one hand, descent with modification argues against "*sui generis* modularity," according to which modules are treated as independent neurocognitive entities that owe nothing to one another²⁵; on the other hand, it suggests that exclusive study of overlapping "generalist" contributions is likely to miss some of the most important evolutionary contributions. On this view, language must be understood as the joint product of domaingeneral ancestral inheritance and domain-specific adaptations.

Empirically, it follows that developmental disorders are particularly well placed to yield insight into the evolution of language by providing insight into both halves of the equation: that which is unique to language, and that which is not. The logic of descent with modification predicts that impairments of particular aspects of language will correlate with impairments in their ancestral cognitive structures. As such, it follows that a great deal can be learned by comparing impaired and spared language and cognition in individuals both within and between disorders over the course of development.

As an example of a within-disorder comparison, consider word learning in children with autism. Several researchers have recently suggested that to a large extent, learning the meanings of words is driven by an understanding of the intentions of other speakers^{26,27}. Accordingly, children with autism, generally considered to have a deficit in understanding other people's intentions (a "theory of mind")²⁸, have difficulty learning the names of objects when cues such as eye gaze are crucial^{29,30}. In most contexts, however, the capacity to infer other people's intentions might be merely helpful, not essential. A number of researchers have suggested that children might also be able to use logical reasoning strategies, independent of social apprehension, to infer the referents of words^{31,32}. Consistent with this idea, autistic children do not appear to be impaired on word learning tasks that only require them to use a reasoning strategy known as "mutual exclusivity" (presuming that each object tends to have only one label)³⁰, suggesting two distinct evolutionary contributions to word learning, one from general capacities for logical reasoning³³, the other from sophisticated mechanisms evolved for the purpose of social cognition³⁴.

According to this perspective, a series of intriguing correlations should emerge within the domain of word learning. First, the ability to learn words when the most critical cues come from the social situation should be positively correlated with performance on theory-of-mind tasks; yet this ability should also be comparatively independent of performance on logical reasoning tasks. At the same time, correct use of strategies like mutual exclusivity should be correlated with performance on (nonlinguistic) logical reasoning tasks even after the ability to use referential intent as a cue to meaning is factored out. Alternatively, if word-learning depended only on general reasoning capacities, one would expect positive correlations between word-learning ability and general intelligence, with no additional variance attributable to performance on theory of mind tasks. The opposite pattern would be expected if word learning were solely descended from structures supporting social cognition rather than general intelligence.

Patterns of dissociation and comorbidity between disorders can cast further light. For example, consider the relation between fragile X syndrome, Down syndrome and Williams syndrome, each of which impairs cognitive capacities, but in markedly different ways. In fragile X syndrome, an X chromosome–linked disorder common in males, cognitive abilities are significantly compromised, but receptive language skills are typically on par with those of normal children of equivalent mental age, albeit with a number of pragmatic difficulties^{35–38}. Within Down syndrome, a disorder resulting in physical and learning disabilities linked to chromosome 21, language is delayed relative to mental age^{39,40}. In Williams syndrome, another genetic disorder resulting in a broad range of cognitive impairments, language is significantly advanced relative to mental age^{8,9}.

In themselves, these three disorders point to a degree of dissociability between language and cognitive skills; closer examination may reveal considerably more. For example, one component of the linguistic impairment in fragile X syndrome³⁷, perseverative language, may be attributable not to a problem with, say, grammar, but rather to a difficulty with (a domain-general process of) inhibitory control, which suggests that evolution co-opted a domain-general process (inhibition) for at least one aspect of the language faculty. In other cases, such as in the relation between impaired spatial cognition and impaired language, there appears to be no systematic pattern either within or between disorders^{8,9,41–44}, suggesting (contrary to a common suggestion) that the ability to represent relations between syntactic and semantic elements evolved separately from the ability to represent relations between physical elements in space.

Fine-grained data on how developmental disorders impair (and leave intact) both cognition and language could thus lead to important insights into the ways in which language builds on and departs from particular aspects of domain-general cognition.

Maximizing what we can learn from developmental disorders

The contribution of developmental disorders to our efforts to reconstruct the nature and origins of language could be considerably enhanced through the creation of a large-scale open-acess database. Although current data already provide some clues about comorbidity and dissociability, the extant data are largely proprietary (rather than publicly available), fragmentary (in that most studies focus either on comorbidity or on dissociation, but not both) and unstandardized (in that fine-grained assessments of particular linguistic or cognitive functions typically depend on *ad hoc* tests designed in individual labs. To a certain extent, even when asking the same questions (for example, about the relationship between phonological and syntactic impairments), each lab uses its own measures. Furthermore, the samples from which these data are drawn are not necessarily representative, as groups of subjects are often selected based on intrinsic interest (for example, because particular subjects are twins, or precisely because those subjects face problems with language that do not immediately appear to be due to other cognitive limitations). As a consequence, reliable cross-study estimates of comorbidity and the prevalence and nature of strong dissociations are difficult or impossible to reconstruct.

The logic of descent with modification, however, suggests that fine-grained data drawn from anonymous individual profiles of particular children could be critical to elucidating rare but theoretically important developmental trajectories. A composite measure of cognitive ability, for example, might not correlate with a composite measure of language, even though deficits in more specific cognitive abilities such as inhibition might correlate with a specific deficit in language such as perseveration.

At present, it is remarkably difficult to answer basic questions such as "what proportion of children with autism have difficulty with inflectional morphology?" Certainly no publicly available data allow a researcher to ask "how does the capacity of autistic children to acquire inflectional morphology relate to their capacities to learn the meanings of concrete nouns as opposed to words describing abstract personal relationships?" There is likewise no way to gather comparable data for children with Williams syndrome, Down syndrome or fragile X. Despite the plethora of quantitative data available from individual studies, answers to specific theoretical questions continue to rely on data that is often anecdotal, distributed and possibly unsystematic.

A systematic, publicly shared database of developmental disorders, combined with a capacity to tap (or at least request) pedigree data and genetic data, could provide a powerful new tool for investigating the origins of language. It would not supplant traditional approaches such as comparative psychology, developmental psycholinguistics or neuroimaging, but in combination with them, could yield considerable insight.

Conclusions

The comparative study of language disorders provides researchers with a natural experiment, a rare chance to examine how variance within the genome influences cognition. The variation and covariation in abilities seen between the normal population and those with language disorders, as well as between those with different disorders, provides a situation logically equivalent to the results of a knockout study. Much as knockout studies can tell us about common heritage in different abilities, studies of language disorders could tell us about the diverse heritage of the many different aspects of language.

Whereas initial studies typically merely described disorders^{45,46}, more recent approaches have emphasized the importance of causal factors that change over the course of development⁴⁷. Our proposal is complementary to this, and emphasizes how vital it is to study both what is impaired and what is unimpaired within a disorder, over the course of development. As an example, evidence that auditory processing deficits cause certain forms of specific language impairment⁴⁸ would show that language is dependent upon a correctly functioning auditory system, but would not lead to many further insights on language evolution. In contrast, a detailed comparison between the phenotypes of two particular disorders where language skills show analogous impairments but cognitive patterns are very different (for example, Down syndrome and specific language impairment⁴⁹) could yield an excellent testing ground for tying nonlinguistic abilities to their possible language counterparts.

Many challenges remain; for example, cognitive capacities may dissociate only at particular points of time⁴⁷; because of the possibility, indeed inevitability, of change over development, the growth of language and related cognitive systems must be studied dynamically, rather than statically. Studies of disorders do not obviate the need for careful linguistic analysis, for neuroimaging, or for careful analyses of both preand postnatal environmental input^{22,47}. Nevertheless, through careful cross-disorder comparison, we can begin to discern which symptoms of language disorders are necessarily causally related, which are correlated by virtue of their shared mechanisms, and which are correlated accidentally, for example simply by being linked to genes with proximal loci. In this way, through careful, systematic analyses of co-morbidity and dissociation, developmental disorders have the potential to provide important insight into the nature and evolution of language, and how language relates to other aspects of cognition.

What developmental disorders reveal about the nature of language has been a hotbed of discussion for over two decades, but perhaps stymied by a commitment to extreme views. The logic of descent with modification suggests a move away from an all-or-nothing perspective on modularity that could lead to important new insights into the nature and evolution of language.

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COMPETING INTERESTS STATEMENT

The authors declare that they have no competing financial interests.

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- Gentner, T.Q., Fenn, K.M., Margoliash, D. & Nusbaum, H.C. Recursive syntactic pattern learning by songbirds. *Nature* 440, 1204–1207 (2006).
- 2. Marcus, G.F. Language: startling starlings. Nature 440, 1117–1118 (2006).
- Arnold, K. & Zuberbuhler, K. Language evolution: semantic combinations in primate calls. *Nature* 441, 303 (2006).
- Hauser, M.D., Chomsky, N. & Fitch, W.T. The faculty of language: what is it, who has it, and how did it evolve? *Science* 298, 1569–1579 (2002).
- Hill, E.L. Non-specific nature of specific language impairment: a review of the literature with regard to concomitant motor impairments. *Int. J. Lang. Commun. Disord.* 36, 149–171 (2001).
- Kovas, Y. & Plomin, R. Generalist genes: implications for the cognitive sciences. Trends Cogn. Sci. 10, 198–203 (2006).
- van der Lely, H.K., Rosen, S. & McClelland, A. Evidence for a grammar-specific deficit in children. *Curr. Biol.* 8, 1253–1258 (1998).
- Bellugi, U., Lichtenberger, L., Jones, W., Lai, Z. & St. George, M.I. The neurocognitive profile of Williams syndrome: a complex pattern of strengths and weaknesses. *J. Cogn. Neurosci.* 12 (suppl. 1), 7–29 (2000).
- Mervis, C.B., Morris, C.A., Bertrand, J. & Robinson, B.F. Williams syndrome, findings from an integrated program of research. in *Neurodevelopmental Disorders* (ed. Tager-Flusberg, H.) Ch. 4, 65–110 (MIT Press, Cambridge, Massachusetts, 1999).
- Lai, C.S., Fisher, S.E., Hurst, J.A., Vargha-Khadem, F. & Monaco, A.P. A forkheaddomain gene is mutated in a severe speech and language disorder. *Nature* 413, 519–523 (2001).
- Gopnik, M. & Crago, M.B. Familial aggregation of a developmental language disorder. Cognition 39, 1–50 (1991).
- 12. Vargha-Khadem, F. *et al.* Neural basis of an inherited speech and language disorder. *Proc. Natl. Acad. Sci. USA* **95**, 12695–12700 (1998).
- Vargha-Khadem, F., Watkins, K., Alcock, K., Fletcher, P. & Passingham, R. Praxic and nonverbal cognitive deficits in a large family with a genetically transmitted speech and language disorder. *Proc. Natl. Acad. Sci. USA* 92, 930–933 (1995).
- 14. Lieberman, P. The pied piper of Cambridge. *The Linguistic Review* **22**, 289–301 (2005).
- Fitch, W.T., Hauser, M.D. & Chomsky, N. The evolution of the language faculty: clarifications and implications. *Cognition* 97, 179–210 (2005).
- 16. Pinker, S. & Jackendoff, R. The faculty of language: what's special about it? *Cognition* **95**, 201–236 (2005).
- Enard, W. et al. Molecular evolution of FOXP2, a gene involved in speech and language. Nature 418, 869–872 (2002).
- Teramitsu, I., Kudo, L.C., London, S.E., Geschwind, D.H. & White, S.A. Parallel FoxP1 and FoxP2 expression in songbird and human brain predicts functional interaction. J. Neurosci. 24, 3152–3163 (2004).
- Fisher, S.E. & Marcus, G.F. The eloquent ape: genes, brains and the evolution of language. *Nat. Rev. Genet.* 7, 9–20 (2006).
- 20. Cosmides, L. & Tooby, J. Origins of domain specificity: the evolutioin of functional

organization. in *Mapping the Mind: Domain Specificity in Cognition and Culture* (eds. Hirschfeld, L.A. & Gelman, S.A.) Ch. 4, 85–116 (Cambridge Univ. Press, Cambridge, UK, 1994).

- Plomin, R. & Kovas, Y. Generalist genes and learning disabilities. *Psychol. Bull.* 131, 592–617 (2005).
- Marcus, G.F. The Birth of the Mind: How a Tiny Number of Genes Creates the Complexities of Human Thought (Basic Books, New York, 2004).
- Marcus, G.F. & Rabagliati, H. Genes and domain-specificity. *Trends Cogn. Sci.*, published online 8 August 2006 (doi:10.1016/j.tics.2006.07.003).
- Margulies, E.H., Kardia, S.L. & Innis, J.W. A comparative molecular analysis of developing mouse forelimbs and hindlimbs using serial analysis of gene expression (SAGE). *Genome Res.* 11, 1686–1698 (2001).
- Marcus, G.F. Cognitive architecture and descent with modification. Cognition 101, 43–65 (2206).
- Bloom, P. How Children Learn the Meanings of Words (MIT Press, Cambridge, Massachusetts, 2000).
- Tomasello, M. The Cultural Origins of Human Cognition (Harvard Univ. Press, Cambridge, Massachusetts, 1999).
- Baron-Cohen, S., Leslie, A.M. & Frith, U. Does the autistic child have a "theory of mind"? *Cognition* 21, 37–46 (1985).
- Baron-Cohen, S., Baldwin, D.A. & Crowson, M. Do children with autism use the speaker's direction of gaze strategy to crack the code of language? *Child Dev.* 68, 48–57 (1997).
- Preissler, M.A. & Carey, S. The role of inferences about referential intent in word learning: evidence from autism. *Cognition* 97, B13–B23 (2005).
- Halberda, J. The development of a word-learning strategy. Cognition 87, B23–B34 (2003).
- Golinkoff, R.M., Mervis, C.B. & Hirsh-Pasek, K. Early object labels: the case for a developmental lexical principles framework. J. Child Lang. 21, 125–155 (1994).
- Pepperberg, I.M. & Wilcox, S.E. Evidence for a form of mutual exclusivity during label acquisition by grey parrots (Psittacus erithacus)? J. Comp. Psychol. 114, 219–231 (2000).
- Seyfarth, R.M., Cheney, D.L. & Bergman, T.J. Primate social cognition and the origins of language. *Trends Cogn. Sci.* 9, 264–266 (2005).
- Abbeduto, L. *et al.* Receptive language skills of adolescents and young adults with down or fragile X syndrome. *Am. J. Ment. Retard.* **108**, 149–160 (2003).
- Hagerman, R.J. Fragile X syndrome. in *Neurodevelopmental Disorders* Ch. 2, 61–132 (ed. J.Hagerman, R.) (Oxford Univ. Press, Oxford 1999).
- Cornish, K., Sudhalter, V. & Turk, J. Attention and language in fragile X. Ment. Retard. Dev. Disabil. Res. Rev. 10, 11–16 (2004).
- Levy, Y., Gottesman, R., Borochowitz, Z., Frydman, M. & Sagi, M. Language in boys with fragile X syndrome. J. Child Lang. 33, 125–144 (2006).
- Nadel, L. Down syndrome in cognitive neuroscience perspective. in Neurodevelopmental Disorders (ed. Tager-Flusberg, H.) Ch. 9, 197–222 (MIT Press, Cambridge, Massachusetts, 1999).
- Rondal, J. Exceptional Language Development in Down Syndrome: Implications for the Cognition Language Relationship (Cambridge Univ. Press., New York, 1995).
- Jarrold, C., Baddeley, A.D. & Hewes, A.K. Verbal and nonverbal abilities in the Williams syndrome phenotype: evidence for diverging developmental trajectories. J. Child Psychol. Psychiatry 39, 511–523 (1998).
- Rice, M.L., Warren, S.F. & Betz, S.K. Language symptoms of developmental language disorders: an overview of autism, Down syndrome, fragile X specific language disorder and Williams syndrome. *Applied Psycholinguistics* 26, 7–28 (2005).
- Rosin, M.M., Swift, E., Bless, D. & Vetter, D.K. Communication profiles of adolescents with Down syndrome. J. Childhood Commun. Disord. 12, 49–64 (1988).
- Cornish, K.M., Munir, F. & Cross, G. Spatial cognition in males with Fragile-X syndrome: evidence for a neuropsychological phenotype. *Cortex* 35, 263–271 (1999).
- Rice, M.L., Wexler, K. & Cleave, P.L. Specific language impairment as a period of extended optional infinitive. J. Speech Hear. Res. 38, 850–863 (1995).
- Bellugi, U., Bihrle, A., Jernigan, T., Trauner, D. & Doherty, S. Neuropsychological, neurological, and neuroanatomical profile of Williams syndrome. *Am. J. Med. Genet. Suppl.* 6, 115–125 (1990).
- Thomas, M. & Karmiloff-Smith, A. Are developmental disorders like cases of adult brain damage? Implications from connectionist modelling. *Behav. Brain Sci.* 25, 727–750; discussion 750–787 (2002).
- Tallal, P., Miller, S. & Fitch, R.H. Neurobiological basis of speech: a case for the preeminence of temporal processing. Ann. NY Acad. Sci. 682, 27–47 (1993).
- Laws, G. & Bishop, D.V. Verbal deficits in Down's syndrome and specific language impairment: a comparison. Int. J. Lang. Commun. Disord. 39, 423–451 (2004).