TOPICS
TOPICS IN COGNITIVE SCIENCE

Topics in Cognitive Science 8 (2016) 361–370

ISSN:1756-8757 print / 1756-8765 online
DOI: 10.1111/tops.12195

Part A: Theoretical foundations

Language Evolution: Constraints and Opportunities From Modern Genetics

Dan Dediu,[a] Morten H. Christiansen[b]

[a]*Language and Genetics Department, Max Planck Institute for Psycholinguistics*
[b]*Department of Psychology, Cornell University*

Received 6 January 2014; received in revised form 14 August 2014; accepted 14 August 2014

Abstract

Our understanding of language, its origins and subsequent evolution (including language change), is shaped not only by data and theories from the language sciences, but also fundamentally by the biological sciences. Recent developments in genetics and evolutionary theory offer both very strong constraints on what scenarios of language evolution are possible and probable, but also offer exciting opportunities for understanding otherwise puzzling phenomena. Due to the intrinsic breathtaking rate of advancement in these fields, and the complexity, subtlety, and sometimes apparent non-intuitiveness of the phenomena discovered, some of these recent developments have either being completely missed by language scientists or misperceived and misrepresented. In this short paper, we offer an update on some of these findings and theoretical developments through a selection of illustrative examples and discussions that cast new light on current debates in the language sciences. The main message of our paper is that life is much more complex and nuanced than anybody could have predicted even a few decades ago, and that we need to be flexible in our theorizing instead of embracing a priori dogmas and trying to patch paradigms that are no longer satisfactory.

Keywords: Language evolution; Genetics of language; Hearing loss; Speech deficits; Epigenetics; Development

1. Introduction

Language is an incredibly complex and multi-faceted human behavior, whose many interacting components have different origins and evolutionary histories. We know from neuropsychology and cognitive neuroscience that language involves many different parts

Correspondence should be sent to Dan Dediu, Language and Genetics Department, Max Planck Institute for Psycholinguistics, Nijmegen, 6525 XD The Netherlands. E-mail: dan.dediu@mpi.nl

of the brain that work in concert (Christiansen & Müller, 2015; Hagoort, 2013). Understanding the genetic underpinnings of the careful neural orchestration needed to accommodate this unique human phenotype is therefore likely not to be straightforward. The relationship between genes and language is further complicated by the fact that there are many different theoretical definitions of what "language" is. To side-step such theoretical uncertainties, we focus instead on the genetic underpinnings of the ability to hear, also touching on speech production, in order to illustrate that even these relatively simple phenotypes are products of nontrivial genetic processes. In this context, it is worth noting that it is not only the language scientists that would profit from a better understanding of modern genetics, but that geneticists and neuroscientists interested in what makes us human should pay closer attention to current advances in the various disciplines studying language and linguistic diversity.

Language scientists do not always seem to appreciate the complex reality that lies behind the concept of a "gene." Indeed, it has even been claimed in the past that a mutation in a single gene gave us language and everything else making us human (e.g., Crow, 2002; Klein, 2002). But we know now (e.g., Jobling, Hollox, Hurles, Kivisild, & Tyler-Smith, 2013; Snustad & Simmons, 2010) that genes are far from the abstract, mathematical entities assumed earlier. Protein-coding genes are composed of *exons* (the coding bits) and *introns*, and the same gene can produce many different proteins (*isoforms*) through the expression of only a subset of its exons (*alternative splicing*) in different tissues and at different times. Moreover, not all genes end up producing proteins but instead result in RNA molecules (such as *microRNAs*) with essential functions in gene regulation. And, finally, it has been discovered that probably the most important variation from an evolutionary point of view is not in the protein-coding bits of our genome (the *exome*, only about 2% of the whole genome), but in the extremely complex *regulatory machinery* that controls when, where, and how much of what is produced. These *regulatory networks* involve "standard" protein-producing genes whose products control the transcription of other genes (the *transcription factors* such as the famous *Hox* genes and *FOXP2*) but also RNA molecules and *epigenetic markers* that do not change the actual message in the DNA but how it is read and interpreted. Thus, the same gene can be (and usually is) involved in very different processes in different places in the organism and at different times, consequently further complicating the understanding of multifaceted phenotypes such as language.

Probably the best-known (and among the most misrepresented) gene involved in speech (and possibly language) is *FOXP2*. There is no space in this paper for a thorough review of this gene (see Fisher & Scharff, 2009; Graham & Fisher, 2013), but suffice to say that some of its mutations are involved in a severe dominant pathology (Developmental Verbal Dyspraxia or DVD; OMIM[1] 602081) affecting speech and possibly more broadly language and cognition in far from simple ways. Crucially, *FOXP2* is not only involved in brain development and the functioning of specific brain areas (presumably mediating its effects on speech, language, and cognition), but also in the development of lung and esophagus. The homologous gene (*FoxP2*) in songbirds seems to be involved in song learning and plasticity through its effects on specific brain regions such as area X, while in mice *FoxP2* affects motor coordination, learning, ultrasonic vocalizations, and

the development of the brain (as well as of the lungs and the esophagus). Importantly, *FOXP2* is a transcription factor controlling the expression of other genes (downstream targets), presumably through its interactions with yet other genes such as the closely related *FOXP1*, by binding to the *regulatory regions* of these target genes. *Normal variants*[2] of the *FOXP2* gene in humans have been associated with covert variation in brain activation patterns (fMRI) during language tasks (Pinel et al., 2012), but a recent relatively large-scale study was unable to find any association between normal variation in *FOXP2* and individual differences in language ability (Mueller et al., unpublished data).

One of *FOXP2*'s targets is the very promising *CNTNAP2* (downregulated by *FOXP2*), which is a member of the *neurexin family* and is involved in neural development and probably nerve conduction. Variants of *CNTNAP2* are associated with autism, intellectual disability, dyslexia and specific language impairment (SLI), as well as with variation in the normal population in phonological memory (Rodenas-Cuadrado, Ho, & Vernes, 2014). But *FOXP2* regulates many more targets (Vernes et al., 2011), some of them producing regulatory factors in their turn, such as the microRNAs *miR-124* and *miR-137*. In turn, other microRNAs such as *miR-9* and *miR-132* regulate *FOXP2* (Clovis, Enard, Marinaro, Huttner, & De Pietri Tonelli, 2012), showing that *FOXP2* really is a hub in a *complex regulatory network* whose activity and effects are highly context-dependent, one facet of which apparently affects speech and possibly language (or processes such as sequence learning that may subserve language).

2. The complexities of gene–behavior relationships

To exemplify the complexity and sometimes surprising beauty of the link between genes and phenotypes that are far from the abstract way of thinking about genetics and evolution (that makes simplistic proposals possible; see Fisher, 2006, for a thorough criticism), we focus here, in a necessarily brief manner, on several genes involved in *hearing loss*. These examples are informative for several reasons: (a) hearing loss is very relevant to language and speech, and in some special cases (village sign languages; Levinson & Dediu, 2013) it provides one of the clearest cases of gene-culture co-evolution, (b) it is relatively well understood and studied, and (c) it provides a rich set of fascinating and illustrative examples.

Sound (see Stover & Diensthuber, 2012, for a review) is captured and channeled by the external ear to the eardrum and the ossicles that further transmit it into the inner ear. Here, the fluid in the cochlea transmits the mechanical waves to the *tectorial membrane*, whose movements bend and activate the *stereocillia* (or hairs) of the hair cells, producing electrical depolarizations and finally a nerve impulse that is sent to the brain for processing. The tectorial membrane must have certain mechanical properties for the efficient transmission of mechanical energy. One of its components is the so-called *α-tectorin* protein produced by the *TECTA* gene on chromosome 11. Certain mutations in *TECTA* result in a *dominant* form of hearing loss (known as *DFNA12*; OMIM 601543), meaning that a person carrying one mutated copy and one normal copy will still be deaf, probably

because the altered α-tectorin protein interferes with other components of the membrane and disrupts its functioning so badly that sound transmission fails. However, other mutations in the *same* gene result in a *recessive* form of hearing loss (*DFNB21*; OMIM 603629), meaning that one needs to have both copies of *TECTA* mutated for deafness to occur; in this case, a single normal copy of *TECTA* is still able to produce enough normal α-tectorin protein to compensate for the non-functional mutated form. Thus, while both types of mutation in the *same* gene result in the "same" phenotype, the inheritance patterns and actual mechanisms are different.

However, the same phenotype (hearing loss) might also emerge through intricate gene-environment interactions, as exemplified by particular mitochondrial genes. Mitochondria are essential components of every cell in our body as they are their "power plants," and due to their particular evolutionary history (once free-living bacteria engulfed by ancestral eukaryotic cells that have developed an *obligate symbiotic* relationship during billions of years of evolution) they still have their own tiny genome using a slightly different genetic code. One such mitochondrial gene (Bindu & Reddy, 2008), *MTRNR1*, produces a component of the translation machinery (a piece of RNA of the small subunit of the *ribosome*) responsible for turning the messenger RNA into protein. Certain mutations in this gene presumably make the already bacterial-like (due to its ancestry) mitochondrial ribozome even more bacterial-like, which means that certain *antibiotics* (such as gentamycin) mistake it for their genuine target and disrupt its function, resulting in hearing loss. Interestingly, hearing loss can in some cases result just from mutations in *MTRNR1* without this environmental factor (the antibiotic treatment) while in others antibiotic treatment does not result in deafness, suggesting that this mutation is modulated by the right *environment* (antibiotics) and/or its *genomic context* (other genetic variants that the individual might have). Another important lesson is that this complex of factors affects ribosomes in every cell in the body and yet results in a very *specific* phenotype (hearing loss), showing that tissue-specific context is fundamental.

More generally, there are over 25 genetic loci involved in *dominant* hearing loss and over 40 in the *recessive* forms, one of the latter being represented by *DFNB1* (OMIM 220290, 612645). This locus in fact encompasses two very closely linked genes, *GJB2* and *GJB6*, both of them encoding components of the so-called *gap junction connexons*—small tubes connecting two neighboring cells and allowing them to exchange small molecules and information. Several mutations in these genes (over 90 in *GJB2* have been cataloged, for example) result in *non-syndromic* hearing loss (i.e., hearing loss not accompanied by other disorders), but there are other mutations that either result in skin disorders only or syndromes involving both skin disorders and hearing loss. Thus, mutations in the same gene could suggest that it has apparently narrow functions ("hearing"), but others reveal it to be involved in multiple phenotypes (*pleiotropy*).

Another recessive form of hearing loss is due to mutations at the *DFNB3* locus (OMIM 600316), involving the gene *MYO15A*, a member of the so-called *unconventional myosins* family of genes. The myosins are usually involved in moving things, resulting in bodily movement (muscle cells) or in other molecules being shuttled inside the cell (e.g., from where they are produced to where they are needed). Within the hair cells,

MYO15A[3] seems to be required for another protein, Whirlin, to be transported to the top of the stereocillia, allowing mechanical displacement to produce electrical changes. Interestingly, it also seems that just moving Whirlin is not enough: MYO15A further interacts with it as well as with another protein, EPS8, in order for the stereocillia to do their job. Thus, this same protein, MYO15A plays *multiple roles* (transport and interaction) in influencing the same phenotype (hearing).

2.1. From hearing loss to speech deficits

It is sometimes argued that genes discovered through their involvement in rare diseases, such as *FOXP2*, cannot say much about speech (and language). However, as clearly shown by the research program built around this gene, we can peer into complex networks of many interacting genes starting from there and uncover unexpected mechanisms and new candidate genes, some involved in the normal variation in the phenotype of interest. Thus, *FOXP2* has been called a "molecular window" (Fisher & Scharff, 2009) into the genetics of speech and language-related functions as its study reveals the genes it regulates and genes that regulate its own expression.

Another, unexpected "molecular window" might have been recently opened for *stuttering* (Kang & Drayna, 2012). An essential and ubiquitous cellular component is the *lysosome* where waste products and dangerous molecules are digested and possibly recycled, using strong digestive enzymes. However, these enzymes are produced in a different organelle (the endoplasmic reticulum) and need to be transported to their final destination. This transport system uses a system of *tags* which are attached to the molecules needing to be moved around and which identify their destination—wrong or absent tags mean transport into the wrong place or no transport at all. These tags are added by complicated systems, in this case involving two proteins: NAGPA and GNPT (Kang & Drayna, 2012). While NAGPA is encoded by a single gene (*NAGPA*) on chromosome 16, GNPT is actually composed of three subunits, α, β, and γ, the first two encoded by the *GNPTAB* gene on chromosome 12 and the third by the *GNPTG* gene on chromosome 16. Initially, only *GNPTAB* was identified as involved in stuttering, but knowing this molecular process in detail allowed the researchers to predict that *GNPTG* and *NAGPA* might be involved in stuttering as well, a prediction wonderfully confirmed later (Kang et al., 2010). Therefore, we see not only that totally unexpected molecular pathways might subtend interesting phenotypes, but that the knowledge and study of these sometimes apparently irrelevant and abstruse mechanisms are a necessary prerequisite for progress.

It may be objected that the genetics of hearing loss and stuttering (and even *FOXP2*) do not really tell us anything important language. However, such an objection would ignore the wealth of data from the language sciences underscoring the multifaceted nature of our linguistic abilities. Thus, we need to understand how evolutionarily older systems and genes may have been co-opted for novel functions and potentially tweaked under selective pressures to include additional functions (see e.g., Christiansen & Müller, 2015, for a discussion of such processes in cognitive neuroscience terms).

3. Epigenetics is not the holy grail

Recent developments in the field of *epigenetics* seem to hold much promise in understanding gene regulation and how environmental factors can affect it, resulting in environmentally informed phenotypic variants, sometimes transmitted across generations. On the one hand, epigenetics can be understood to refer to any sort of non-genetic transmission of information within an organism, but also across generations (Jablonka & Raz, 2009). Phenomena such as self-maintaining metabolic loops, prions (proteins that can transmit their shape and properties without recourse to the genetic material), and RNA interference (small RNA molecules regulating gene expression) involve the maintenance of information in the absence of genetic change, but also niche construction and even culture can be seen as parallel, epigenetic channels of information transmission. On the other hand (e.g., Smith & Meissner, 2013), epigenetics is used in a very narrow sense referring to specific chemical markers on the DNA bases themselves or on the histones (proteins around which DNA is coiled), controlling how genes are accessed, read, and interpreted.

These chemical marks (most often *methylation* or acetylation) can be abstractly understood as changing the way the genetic message is read in the absence of changes to this message itself. Thus, while the actual sequence of a gene is unchanged, its methylation might make it harder to be expressed, thus affecting the resulting phenotype. The epigenetic markers are managed by complex systems of proteins being part of networks of gene regulation and are involved in the differential expression of genes in various tissues and developmental stages, as well as in diseases such as cancer.

In the vast majority of cases, the epigenetic marks are erased and reinitialized during gametogenesis, thus making sure that the offspring's epigenetically influenced gene regulation is not influenced by their parents'. However, in some cases, such as the parent of origin effects or *imprinting* (where the same gene variant has different phenotypic effects depending on which parent it has been inherited from; Clift & Schuh, 2013) and the celebrated epigenetic inheritance of stress preparedness (in rats and humans; Rozanov, 2012), some genomic regions escape this resetting and instead allow epigenetic marks to be transmitted to the next generation. While fascinating, it is crucial to realize that this transmission is not Lamarkian in the sense that the environment could modify the inherited information in unconstrained and directed ways, but that what is transmitted concerns the differential expression of pre-existing genetic networks highly adapted to their tasks by previous episodes of selection (Lester et al., 2011). In the case of the stress response, for example, the epigenetic information transmitted to the next generation switches on or off a pre-adapted preparedness to face a stressful environment encountered by the mother and likely to persist into the next generation. Thus, it is highly unlikely that epigenetics would somehow instruct chemical markers on the genome to encode a new and never before encountered environmental or cultural feature, such as a specific linguistic property, making it, as it were, "innate."[4]

4. Conclusions: What (probably) can and cannot be genetic

Given the facts and examples briefly reviewed above, as well as many other considerations not included here for reasons of space,[5] but treated in the other papers in this special issue, we can begin to sketch some constraints on what can and cannot be the case in language evolution.

The hope that a single relatively recent mutation gave us language in a single "hopeful monster" is highly improbable and must be replaced by a more nuanced view. Such a mutation would presumably affect a hub in a regulatory network changing the location and/or timing of gene expression in highly coordinated ways, given the complex and multi-faceted architecture of language and speech. It is true that evolutionary developmental biology (or *Evo-Devo*; for a gentle introduction, see Carroll, 2011; De Robertis, 2008) has uncovered fascinating cases of developmental master genes (such as the celebrated *Hox* genes; Mallo & Alonso, 2013) whose duplication or altered patterns of expression gave rise to striking and sometimes dramatic variations in form (Pick & Heffer, 2012), but such saltations do not result in a finished end product.[6] Their catastrophic results must be accommodated by developmental and phenotypic *plasticity* (e.g., West-Eberhard, 2003) and be further incorporated into the genome and shaped into useful and intricate functional phenotypes by natural selection affecting many other genes. For example, although mammals like cows, whales, and bats have the same overall body plan, their forelimbs are exquisitely adapted to their ecological niches as legs, flippers, and wings (Schneider & Shubin, 2013; see also Christiansen & Chater, 2008, for discussion).

The genetic foundations of language and speech are extremely complex, and there is no gene "for" language (Fisher, 2006). Instead, there are many genes interacting in complex regulatory networks tuned to many contextual cues and influencing many aspects of the phenotype. Genes are not monolithic units with simple and clear functions but instead there is pervasive gene regulation at multiple levels and constant interaction with the environment. This genetic architecture mostly affects old genes by tweaking them and especially their regulation, being honed by millions of years of biological evolution, resulting in a robust, redundant, and incredibly complex system. The more we look into the genetics of complex phenotypes (including language), the more we realize that simplistic assumptions are doomed to fail and that the path from DNA to grammaticality judgments is not only complicated but complex and more often than not counterintuitive. Uncovering the genetics of this complex architecture will require more detailed and sophisticated measures of the many aspects and components of language, including on-line learning tasks, processing-based brain imaging, eye tracking studies, etc., investigating everything from phonetics to pragmatics and naturalistic discourse settings. This approach promises to provide a more complete picture of what language is and of how it is used than we currently have.

Finally, "genes" do not do anything by themselves, and the "environment" is not some external reality influencing their expression. Crucially, the genome is not read only once during development (modulated or not by this environment) and then packed away and stored, but is continuously active, reacting instantly—on the order of milliseconds—to

changes both within and outside the organism, continuously blurring the border between the genotype and the environment. Modern approaches to evolution such as *niche construction* (Odling-Smee, Laland, & Feldman, 2003), *gene-culture co-evolution* (Richerson & Boyd, 2008), and *developmental systems theory* (Oyama, Griffiths, & Gray, 2003) make clear that while not completely the same, genes and their environments are intertwined and intimately connected during development and evolution. The emergence of language through such evolutionary processes has further affected the evolution of our species, allowing *Homo loquacious* (or "talkative humans") to change the Earth in profound ways through processes of cultural evolution (Richerson & Christiansen, 2013).

Acknowledgments

We thank the organizers and the participants at the Origins of Complex Communication and Language Workshop, Konrad Lorenz Institute, Altenberg, Austria, Bart de Boer, and one other anonymous reviewer for very helpful comments and suggestions. DD is funded by the NWO (Netherlands Organisation for Scientific Research) Vidi grant no. 276-70-022.

Notes

1. OMIM (Online Mendelian Inheritance in Man; http://omim.org) is a vast and up-to-date online database containing information on many phenotypes, genes, and molecular mechanisms, as well as comprehensive references to the primary literature. Therefore, due to space constraints, we will use OMIM IDs instead of actual references and details.
2. Here, normal alleles (variants) are relatively frequent and are not involved in pathologies; by contrast, the KE mutation is both rare and causes DVD.
3. Note that while the names of genes are written in *italic* form (e.g., *MYO15A*), their protein products are denoted by non-italicized letters (e.g., MYO15A).
4. The term "innate" is extremely loaded and unclear, and we point the interested reader to the lucid discussion in Mameli and Bateson (2006).
5. For more relevant examples and discussion, see Dediu (2015).
6. It is also important to note that it is unclear just how far this approach may generalize to explain a putative emergence of a completely novel behavior as hypothesized for language (e.g., by Chomsky, 2010).

References

Bindu, L. H., & Reddy, P. P. (2008). Genetics of aminoglycoside-induced and prelingual non-syndromic mitochondrial hearing impairment: A review. *International Journal of Audiology*, *47*(11), 702–707. doi:10.1080/14992020802215862

Carroll, S. B. (2011). *Endless forms most beautiful: The new science of evo devo, and the making of the animal kingdom*. London: Quercus Publishing.

Chomsky, N. (2010). Some simple evo devo theses: How true might they be for language? In R. K. Larson, V. Déprez, & H. Yamakido (Eds.), *The evolution of human language* (pp. 45–62). Cambridge, UK: Cambridge University Press.

Christiansen, M. H., & Chater, N. (2008). Language as shaped by the brain. *Behavioral & Brain Sciences*, *31*, 489–558.

Christiansen, M. H., & Müller, R.-A. (2015). Cultural recycling of neural substrates during language evolution and development. In M. S. Gazzaniga & G. R. Mangun (Eds.), *The cognitive neurosciences V* (pp. 675–682). Cambridge, MA: MIT Press.

Clift, D., & Schuh, M. (2013). Restarting life: Fertilization and the transition from meiosis to mitosis. *Nature Reviews Molecular Cell Biology*, *14*(9), 549–562. doi:10.1038/nrm3643

Clovis, Y. M., Enard, W., Marinaro, F., Huttner, W. B., & De Pietri Tonelli, D. (2012). Convergent repression of Foxp2 3'UTR by miR-9 and miR-132 in embryonic mouse neocortex: Implications for radial migration of neurons. *Development*, *139*(18), 3332–3342. doi:10.1242/dev.078063

Crow, T. J. (2002). Sexual selection, timing and an X-Y homologous gene: Did Homo sapiens speciate on the Y chromosome? In T. J. Crow (Ed.), *The speciation of modern Homo sapiens* (pp. 195–216). Oxford, UK: Oxford University Press.

De Robertis, E. M. (2008). Evo-devo: Variations on ancestral themes. *Cell*, *132*(2), 185–195. doi:10.1016/j.cell.2008.01.003

Dediu, D. (2015). *An introduction to genetics for language scientists*. Cambridge, UK: Cambridge University Press.

Fisher, S. E. (2006). Tangled webs: Tracing the connections between genes and cognition. *Cognition*, *101*(2), 270–297. doi:10.1016/j.cognition.2006.04.004

Fisher, S. E., & Scharff, C. (2009). FOXP2 as a molecular window into speech and language. *Trends in Genetics*, *25*(4), 166–177. doi:10.1016/j.tig.2009.03.002

Graham, S. A., & Fisher, S. E. (2013). Decoding the genetics of speech and language. *Current Opinion in Neurobiology*, *23*(1), 43–51. doi:10.1016/j.conb.2012.11.006

Hagoort, P. (2013). MUC (Memory, Unification, Control) and beyond. *Frontiers in Language Sciences*, *4*, 416. doi:10.3389/fpsyg.2013.00416

Jablonka, E., & Raz, G. (2009). Transgenerational epigenetic inheritance: Prevalence, mechanisms, and implications for the study of heredity and evolution. *The Quarterly Review of Biology*, *84*(2), 131–176.

Jobling, M. A., Hollox, E., Hurles, M., Kivisild, T., & Tyler-Smith, C. (2013). *Human evolutionary genetics*. New York: Garland Science.

Kang, C., & Drayna, D. (2012). A role for inherited metabolic deficits in persistent developmental stuttering. *Molecular Genetics and Metabolism*, *107*(3), 276–280. doi:10.1016/j.ymgme.2012.07.020

Kang, C., Riazuddin, S., Mundorff, J., Krasnewich, D., Friedman, P., Mullikin, J. C., & Drayna, D. (2010). Mutations in the lysosomal enzyme-targeting pathway and persistent stuttering. *New England Journal of Medicine*, *362*(8), 677–685. doi:10.1056/NEJMoa0902630

Klein, R. G. (2002). *The dawn of human culture*. New York: John Wiley & Sons.

Lester, B. M., Tronick, E., Nestler, E., Abel, T., Kosofsky, B., Kuzawa, C. W., Maze, I., Meaney, M. J., Monteggia, L. M., Reul, J. M. H. M., Skuse, D. H., Sweatt, J. D., Wood, M. A. (2011). Behavioral epigenetics. *Annals of the New York Academy of Sciences*, *1226*, 14–33. doi:10.1111/j.1749-6632.2011.06037.x

Levinson, S. C., & Dediu, D. (2013). The interplay of genetic and cultural factors in ongoing language evolution. In P. J. Richerson & M. H. Christiansen (Eds.), *Cultural evolution: Society, technology, language, and religion* (vol. *12*, pp. 219–232). Cambridge, MA: MIT Press.

Mallo, M., & Alonso, C. R. (2013). The regulation of Hox gene expression during animal development. *Development*, *140*(19), 3951–3963. doi:10.1242/dev.068346

Mameli, M., & Bateson, P. (2006). Innateness and the sciences. *Biology and Philosophy*, *21*, 155–188.

Odling-Smee, F. J., Laland, K. N., & Feldman, M. W. (2003). *Niche construction: The neglected process in evolution*. Princeton, NJ: Princeton University Press.

Online Mendelian Inheritance in Man, OMIM®. Baltimore, MD: McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University. Available at http://omim.org/. Accessed June 15, 2014.

Oyama, S., Griffiths, P. E., & Gray, R. D. (2003). *Cycles of contingency: Developmental systems and evolution*. Cambridge, MA: MIT Press.

Pick, L., & Heffer, A. (2012). Hox gene evolution: Multiple mechanisms contributing to evolutionary novelties. *Annals of the New York Academy of Sciences*, *1256*, 15–32. doi:10.1111/j.1749-6632.2011.06385.x

Pinel, P., Fauchereau, F., Moreno, A., Barbot, A., Lathrop, M., Zelenika, D., Le Bihan, D., Poline, J. B., Bourgeron, T., & Dehaene, S. (2012). Genetic variants of FOXP2 and KIAA0319/TTRAP/THEM2 locus are associated with altered brain activation in distinct language-related regions. *The Journal of Neuroscience*, *32*(3), 817–825. doi:10.1523/JNEUROSCI.5996-10.2012

Richerson, P. J., & Boyd, R. (2008). *Not by genes alone: How culture transformed human evolution*. Chicago: University of Chicago Press.

Richerson, P. J., & Christiansen, M. H. (Eds.) (2013). *Cultural evolution: Society, technology, language and religion*. Cambridge, MA: MIT Press.

Rodenas-Cuadrado, P., Ho, J., & Vernes, S. C. (2014). Shining a light on CNTNAP2: Complex functions to complex disorders. *European Journal of Human Genetics*, *22*(2), 171–178. doi:10.1038/ejhg.2013.100

Rozanov, V. A. (2012). Epigenetics: Stress and behavior. *Neurophysiology*, *44*, 332–350.

Schneider, I., & Shubin, N. H. (2013). The origin of the tetrapod limb: From expeditions to enhancers. *Trends in Genetics*, *29*(7), 419–426. doi:10.1016/j.tig.2013.01.012

Smith, Z. D., & Meissner, A. (2013). DNA methylation: Roles in mammalian development. *Nature Reviews Genetics*, *14*(3), 204–220. doi:10.1038/nrg3354

Snustad, D. P., & Simmons, M. J. (2010). *Principles of genetics* (5th ed.). Hoboken, NJ: John Wiley & Sons Inc.

Stover, T., & Diensthuber, M. (2012). Molecular biology of hearing. *GMS Current Topics in Otorhinolaryngology, Head and Neck Surgery*, *10*, doi:10.3205/cto000079

Vernes, S. C., Oliver, P. L., Spiteri, E., Lockstone, H. E., Puliyadi, R., Taylor, J. M., Ho, J., Mombereau, C., Brewer, A., Lowy, E., Nicod, J., Groszer, M., Baban, D., Sahgal, N., Cazier, J. B., Ragoussis, J., Davies, K. E., Geschwind, D. H., & Fisher, S. E. (2011). Foxp2 regulates gene networks implicated in neurite outgrowth in the developing brain. *PLoS Genetics*, *7*(7), e1002145.

West-Eberhard, M. J. (2003). *Developmental plasticity and evolution* (1st ed.). Oxford, UK: Oxford University Press.