Journal of Genetics, Vol. 97, No. 5, December 2018, pp. 1485–1491
https://doi.org/10.1007/s12041-018-1006-9

REVIEW ARTICLE

Genetics of language and its implications on language interventions

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Received 10 July 2018; accepted 14 August 2018; published online 23 October 2018

Abstract. Genetic variation of language genes affect neurophysiology of brain and can thus influence the way people respond to environmental language input, leading to differences in terms of their response to environmental language learning. Conversely, language learning environment too can affect gene expressions through neuroepigenetic mechanisms, leading to increased interindividual differences. Further, language-related cognitive processes such as learning, working memory and perception; and language-related affective factors such as stress and positive emotion involve neuroplasticity, which is also epigenetically regulated. Language intervention methods must understand the extent and the type of difficulties, and must offer personalized learning and medical solutions. Medical intervention in terms of epigenetics and neurotransmitter regulation is proposed in addition to effective teaching methods to aid in effective language acquisition.

Keywords. language acquisition; language genes; neuroepigenetics; personalized medicine; personalized learning.

Genetics of language ability

Genetic research into language-related disorders such as specific language impairment, developmental dyspraxia, autism spectrum disorder (ASD) and developmental dyslexia points to language being a part of our genetic makeup. An entry-point into the genetic research of language was provided by the pathbreaking discovery of a language-related gene, *FOXP2*, a transcription factor gene which when mutated was known to cause child apraxia of speech (CAS) (Lai *et al.* 2001). *FOXP2* has since been used as a molecular framework to link genes, neurons, brain and speech among different population (Fisher and Scharff 2009). For instance, its genetic variants are known to cause language disorders (Estruch *et al.* 2016). A single-nucleotide polymorphism (SNP) of *FOXP2*, rs6980093 modulates several language-related and reading-related traits (Chandrasekaran *et al.* 2015; Mozzi *et al.* 2017). Intragenic deletions in *FOXP2* were also observed to pose risk for CAS (Turner *et al.* 2013). In addition to genetic variants of *FOXP2*, its transcription factor too regulates other language-related genes, perpetrating genetic differences in language capacity. For example, a *FOXP2* regulated gene, *SRPX2* (Roll *et al.* 2010) is associated with speech difficulties; another such gene *CNTNAP2* with peak association at SNP rs17236239 is linked to language in ASD-affected individuals (Alarcón *et al.* 2008; Vernes *et al.* 2008).

In addition to the genes regulated by the *FOXP2* transcription factor, several other genes are associated with language abilities. *ASPM* and microcephalin genes are linked to linguistic tone (Ladd *et al.* 2008); and *CMIP* and *ATP2C2* are linked to phonological memory (Newbury *et al.* 2009). Deletions and duplications in the 16p11.2 locus modulates phonology, written language and vocabulary (Hippolyte *et al.* 2016). A single rare coding variant of *NFX1* gene rs144169475 is significantly associated with language impairment (Villanueva *et al.* 2015). Similarly, the *GLI3* gene is found to be significantly associated with language performance in Alzheimer's disease (Deters *et al.* 2017). *GRIN2A* (Lesca *et al.* 2013), *GRIN2B* (Ocklenburg *et al.* 2011), *SETBP1* (Filges *et al.* 2011), *ERC1* (Thevenon *et al.* 2013), *AUTS2* (Amarillo *et al.* 2014), *KMT2D* (Morgan *et al.* 2015a), *OXR1* (Liu *et al.* 2015) and *SCN9A* (Chen *et al.* 2017a) are some of other genes that are observed to have different levels of associations with language disabilities. The variants of these genes and their developmental consequences will have effects on the language-related abilities associated with them.

'Reading', which is a language-related human capacity, also involves a number of genes, namely, *DYX1C1* (Taipale *et al.* 2003), *ROBO1* (Hannula-Jouppi *et al.* 2005;

Tran *et al.* 2014, *DCDC2* (Meng *et al.* 2005), *KIAA0319* (Cope *et al.* 2005) and *MRPL19*/*C2ORF3* (Scerri *et al.* 2012). Further, *CCDC136*/*FLNC* and *RBFOX2* were also identified as candidate genes associated with both reading and language disabilities (Gialluisi *et al.* 2014). Variation in these genes influences the reading ability. For instance, two haplotypes and six SNPs of *DCDC2*, namely rs807724, rs2274305, rs4599626, rs9467075, rs6456593 and rs6922023, were found to be associated with developmental dyslexia among Chinese Uyghur children (Chen *et al.* 2017b).

Genetics of environmental language acquisition

Language capacity, which involves complex cognitive processes, is considered to be a product of gene–environment coevolution (Moya and Henrich 2016). In fact, the cognitive computational aspects of language are the result of coevolution of cultural abilities and genetic capacities of human brain (Morgan *et al.* 2015b; Sterelny 2012). Also, recursion, which is considered to be the core property of language, is a product of culture-gene coevolution (Hauser *et al.* 2002; Berwick and Chomsky 2016). The innate gene–environment coordination influences the way in which an individual's cognitive system interacts with language input from the cultural environment. This means that along with genetics of language capacity, genetics of environmental language acquisition is essential in understanding the acquisition of individual languages.

For a language to be acquired, favourable expressions of genes responsible for environmental learning ability along with favourable expressions of language genes are necessary. An analogy to this can be drawn using the genetics of intelligence quotient (IQ). Although several genes are responsible for IQ (Sniekers *et al.* 2017), gene–environment interaction is also required for the favourable phenotype (i.e. higher IQ). One such environmental input is breastfeeding. Breastfed children are known to possess higher IQ but only when a specific variant of *FADS2* is present in the genome (Caspi *et al.* 2007). Here, *FADS2* acts as a gene that enables favourable interaction of the external environment with genes. *BDNF* gene is another gene found to be one of the important genes, which is found to influence learning from environmental stimuli (Keifer 2017). In language acquisition too, both language genes and genes responsible for environmental learning (such as *BDNF*) are crucial. Simply put, favourable expression of both language genes and genes that mediate gene and environment interactions gives rise to a favourable language phenotype.

It is thus clear that genes enable an individual to be receptive to a socio-cultural learning environment. During language acquisition, this reception takes place in the human brain through neurotransmitter signalling, i.e. neural connections among brain areas responsible for language subskills are developed by the neurotransmitter signal transmission via synaptic junctions. Increased levels of a neurotransmitter named dopamine in the amygdala region of the brain aids in performing language-related cognitive tasks such as learning and memory (Fried *et al.* 2001). The levels of dopamine, in turn, are found to be regulated by dopaminergic genes (Kitayama *et al.* 2016). Thus, it can be deduced that the variation in neurotransmitter-regulator genes too will have its effect on learning. Rightly so, a study emphasizes that interindividual variation in environment learning could be due to genetic variation in serotonin transporter genes (Caspi *et al.* 2010).

Cultural language acquisition triggers the development of novel neural connections of brain areas corresponding to various language functions, changing the structure and functions of the brain. Such modified brain structure and function is the reason why people learn languages differently (Golestani and Zatorre 2009). This change in the neural organization in response to environmental learning is known as neural plasticity. Neural plasticity is regulated by epigenetic processes which in turn modify gene expressions (Allen 2008; Borrelli *et al.* 2008; Kennedy *et al.* 2016).

Epigenetics in language acquisition

Language acquisition is no longer viewed in terms of dichotomy between nature and nurture but is increasingly recognized that genetic and environmental factors interact. It is evident that genes influence the way individuals are receptive to environmental stimuli. Specifically, changes in the environmental factors influence the gene expression through a biological process known as epigenetics. Epigenetic processes such as DNA methylation and histone modification alter the gene expression depending on the cues from the environment. Since environmental stimuli vary at various levels such as families, communities and countries, epigenetically modified gene expressions too vary.

Epigenetic processes pervade neuronal genes as well (Telese *et al.* 2013; Satterlee *et al.* 2015) and therefore affect cognition and behaviour. For instance, neurodevelopmental plasticity and synaptic plasticity are influenced by epigenetic mechanisms (Allen 2008; Bagot and Meaney 2010; Fass *et al.* 2014; Felling and Song 2015). Neural plasticity corresponding to acquisition of languages also involves epigenetic processes which can affect gene expressions (Reich and Richards 2004).

Role of cognition and emotion in language acquisition

Language acquisition involves activation of several language-associated cognitive processes of human brain such as learning, working and long-term memory, attention and perception in addition to language-specific cognitive processes such as lexical processing, phonological

decoding and meaning association. Similar to language subskills, cognitive subskills too have significant associations with genes. A gene named *PCCH17* is found to be associated with cognition (Chang *et al.* 2017). Cognitive processes, namely, learning and memory were deficient when *DYX1C1*, a dyslexia candidate gene was mutated (Rendall *et al.* 2017). Learning is influenced by different polymorphisms of the *FOXP2* gene and it modulates neural correlates of nonnative speech (Chandrasekaran *et al.* 2015). Working memory, an important cognitive ability required for language is also known to have genetic basis (Owens *et al.* 2011; Knowles *et al.* 2014). More precisely, phonological working memory is also found to be associated with *FOXP2* (Schulze *et al.* 2018).

Neuronal genes corresponding to cognitive processes are epigenetically regulated. Cognition is linked to early childhood epigenetic modifications (Peter *et al.* 2016). Neural epigenetics influences the cognitive abilities such as learning (Mc Ewen 2015), memory (Levenson and Sweatt 2005) and object recognition (Gräff and Mansuy 2008), contributing to an increase in interindividual differences (IDs) in language acquisition due to the environmental causes. Moreover, synaptic plasticity of neurons, an essential property of neurons that forms brain connections that enable language acquisition and other cognitive functions, is also found to be epigenetically regulated (Borrelli *et al.* 2008; Kennedy *et al.* 2016). Thus, epigenetic regulation of cognition and synaptic plasticity plays a key role in language acquisition.

Affect or emotion is another dimension through which language acquisition can be analysed. Language acquisition occurs ideally an anxiety-free environment, just as a baby learns its mother tongue. Studies on second language (L2) acquisition point to negative effects of an environment that induces fear and anxiety (Krashen 1982; Anyadubalu *et al.* 2010; Atasheneh and Izadi 2012). Research has shown that reduced negative affective factors such as anxiety and stress improves language learning in addition to improvements in personality growth and self-efficacy beliefs (Ni 2012; Jennifer and Ponniah 2017). Moreover, positive emotional aspects such as motivation and self-regulation mediate the language learning process in a favourable manner (Méndez López and Fabela Cárdenas 2014; Ölmez 2015). The claims made by the above studies are strengthened by evidences from human biology: cognitive and emotional brain circuitry is intertwined in a way that many of their functions are shared by the same areas in the brain (Gray 1990; Pessoa 2008; Williams *et al.* 2010; Dolcos *et al.* 2011; Scheidegger *et al.* 2016; Scult and Hariri 2018). Thus, it is highly possible that language circuitry too is affected by the emotion circuitry of the brain (Mondal 2016).

Genetic studies (as reviewed by Canli *et al.* 2009) show that emotion is gene-regulated similar to cognition. Rs322931, an SNP of *LINC01221*, along with some microRNAs, is found to be significantly associated with positive emotion (Wingo *et al.* 2017). An SNP of the oxytocin receptor gene (*OXTR*), rs1042778, too is linked with positive emotion for learning (Isgett *et al.* 2016). Association between high polygenic scores for schizophrenia and neural correlates of emotion also points to genetic links of emotion (Dzafic *et al.* 2018). Studies on epigenetics (McEwen 2016; Nieto *et al.* 2016) suggest that emotion too is epigenetically regulated.

Gene variants and IDs

Language capacity tends to be different among individuals and these IDs are perpetrated by genetic variations. The genetic variation in language-associated genes renders people to be different in terms of their brain neurophysiology (Wong and Ettlinger 2011), thereby affecting their cognitive abilities that aid in language acquisition, and resulting in IDs in language capacity. Genetic variants in language-associated genes are either missense or nonsense gene mutations, SNPs (Crespi *et al.* 2017), copy number variants (Laffin *et al.* 2012), intragenic deletions (Turner *et al.* 2013), duplications (Hippolyte *et al.* 2016) or exome variants (Worthey *et al.* 2013). In addition to language gene variation, the variants of the genes corresponding to reading, environmental language learning, neurotransmitter regulation, synaptic plasticity, cognition and emotion processes that aid language multiply the IDs. These variants lead to variation in protein coding at the DNA level, further to the differences in brain development and neural plasticity, thus perpetrating differences at various levels of human biology pertaining to language. These differences result in IDs in language capacity.

Along with the variations perpetrated through language genes, environmental modification of such genes increases the IDs. The cues from the environment can influence the way in which language is acquired. Epigenetic mechanisms are known to regulate several psychological and behavioural aspects. Research on neuroepigenetics has proved that stress, cognition and neuroplasticity are epigenetically regulated (Day and Sweatt 2011; McEwen *et al.* 2012; Toyokawa *et al.* 2012). These mechanisms have a prolonged effect on language learners and can further accentuate the individual differences among learners.

Handling IDs in L2 acquisition

IDs in language and cognition is all-pervasive in human biology and cognitive psychology, spreading through genetic mechanisms, neural development, epigenetic mechanisms and synaptic plasticity. Yet, genetic studies on language difficulties ignore IDs by focussing mainly on group-based differences. Moreover, IDs have long been neglected by cognitive science research which worked largely on explaining universal human cognitive capacities (Levinson 2012). Such studies have not considered the

inherent variety characterized by every human being. The lack of importance to IDs and the above-mentioned shortcoming of cognitive science is a bottleneck in finding a holistic solution for L2 pedagogy.

A problem that a L2 teacher often faces in a classroom setup is the wide variation in proficiency levels and speed of language acquisition and the difference in reading ability. Firstly, language learners are different in their language-related genetic makeup. Secondly, epigenetic regulation of neural plasticity plays a role in language acquisition. An added classroom situation is where students differ in socio-cultural background. For example, a child may have exposed to an environment where their parents are proficient at the L2 and may use it at their home as well. The ubiquitous and multidimensional nature of IDs in language acquisition means that individuals learn languages in several ways. In fact, IDs are the reason why varying levels of language proficiency and the speed of acquisition are observed among learners in spite of the same exposure provided to learners. Teachers must be viewed as solution providers. Just as a doctor must diagnose the ailments of the patient, a teacher must possess the knowledge of the extent and the type of language disability and choose the course of action in accordance with the language difficulties (Sriganesh *et al.* 2018).

Another challenge faced in language classrooms is the lack of understanding among teachers as to how a language is acquired. Still, many English as a second language classrooms follow a stressful rote learning of language grammar. The most scientific arguments on language acquisition is provided by Chomsky and his proponents who state that language is innate and we learn languages through biological instinct of acquiring language using a limited input (Chomsky 2006; Pinker 2007). Biological evidences too point out that language is genetic. This is in line with Krashen's L2 theories, which further adds that language is acquired in the absence of any stress and anxiety about consciously learning the structure.

The language acquisition from biological perspective thus calls for language training methods that are in tune with how every individual best acquires the ability. In other words, language teaching methods must cater to every individual instead of catering to the arbitrarily chosen general needs of learners in a classroom setup. Additionally, they must also suit the cultural differences since socio-cultural factors are different for learners. Every classroom presents a unique milieu of socio-cultural differences which can also contribute to biological difference especially in terms of neurobiology (neuroplasticity and neuroepigenetics) since the structure and function of the brain changes during any kind of learning.

L2 classrooms mostly ignore such biological and psychological differences and follow a one-size-fits-all curriculum that ignores the language problems faced by learners. Not addressing the specific language problems during language acquisition will only lead to increased difference in language skills when they become adults. A vast difference in language subskills is seen among L2 classroom learners despite them being taught in a similar setup. A study in an Indian classroom, notes that children differ widely in their language skills in spite of they being from a similar socio-economic background and the schools with the same medium of instruction (Jennifer and Ponniah 2018). This study suggests that if IDs are ignored in the childhood, it might pose a bigger problem in adulthood.

Language teaching methods can only give a theoretical basis for L2 acquisition and cannot provide a context-sensitive solution to language learning. The role of a language teacher is to identify the type and the level of language difficulty and to provide a learning environment where language input is personalized (in accordance with the language difficulties faced by the learner) and self-selected, with comprehensible, yet challenging input as in Krashen's i+1 (Krashen 1982). Research has proven that such self-selected reading plays an important role in language acquisition, reduces anxiety and improves cognition (Jennifer and Ponniah 2018). However, second language teaching still focusses on tasks and excercises that give importance to grammar rules and list learning of vacabulary. Such decontextualized learning cannot provide comprehensible input which is a necessary condition for language acquisition. Comprehensible input facilitates that incidental acquisition of grammar (Ponniah 2009) and vocabulary (Ponniah 2011; Jennifer and Ponniah 2017). Such incidental acquisition of language is the most effective way of acquiring a language and is most natural too since mother tongue is also acquired in a similar manner.

In addition to personalized language teaching methods, personalized medical intervention (especially for people with language impairments) must also be considered. Epigenetic and neurotransmitter regulation are two suitable medical interventions for language difficulties. Since brain processes such as synaptic plasticity and cognition are epigenetically regulated, epigenetic treatment is viable for individuals for whom the language deficiency is a result of epigenetic modifications. For instance, neuronal DNA methylation constitute an effective regulation of memory (Day and Sweatt 2010, 2011), which is an essential subskill for language. Epigenetic treatment involving histone deacetylase inhibitors and DNA methyltransferases inhibitors improves memory, learning and enhances synaptic plasticity. Also, vitamin B12, which aids in DNA methylation, is known to influence academic performance, motivation and learning strategies (Wang *et al.* 2017). DNA methylation benefits triggered by vitamin B12 can aid in language acquisition too since the development of motivation and learning is essential for language acquisition.

Interventions based on neurotransmitters are also workable solutions for people with language disorders.

Studies have pointed out that dopamine is positively correlated with synaptic plasticity (Andrzejewski *et al.* 2005). Dopamine is widely known to influence motivation, which is a key factor in language acquisition. It is also linked to language learning in Parkinson's disease (McNamara and Durso 2018). An optimized intake of dopamine among learners with dopamine deficiency may influence language learning.

Conclusion

Genetics and epigenetics of language, brain development, neuroplasticity, language-related cognition and emotion lead to IDs. IDs are also perpetrated due to multilevel socio-cultural differences such as family, school, community and nation, which in turn affect the genes through neuroepigenetics increasing the IDs. Language training approaches must recognize IDs to diagnose the language problems precisely and adopt the methods that are both context-sensitive and learner-oriented. In addition, medical intervention for people with language difficulties should also be in tune with the way in which an individual differs in terms of genetics, epigenetics and neuroscience.

References

Alarcón M., Abrahams B. S., Stone J. L., Duvall J. A., Perederiy J. V., Bomar J. M. *et al*. 2008 Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. *Am. J. Hum. Genet*. **82**, 150–159.

Allen N. D. 2008 Temporal and epigenetic regulation of neurodevelopmental plasticity. *Philos. Trans. Biol. Sci*. **363**, 23–38.

Amarillo I. E., Li W. L., Li X., Vilain E. and Kantarci S. 2014 De novo single exon deletion of AUTS2 in a patient with speech and language disorder: a review of disrupted AUTS2 and further evidence for its role in neurodevelopmental disorders. *Am. J. Med. Genet*. **164**, 958–965.

Andrzejewski M. E., Spencer R. C. and Kelley A. E. 2005 Instrumental learning, but not performance, requires dopamine D1-receptor activation in the amygdala. *Neuroscience* **135**, 335–345.

Anyadubalu C. C., Atasheneh N., Izadi A. and Krashen S. D. 2010 The role of teachers in reducing/increasing listening comprehension test anxiety: a case of Iranian EFL learners. *English Lang. Teach*. **5**, 193–198.

Atasheneh N. and Izadi A. 2012 The role of teachers in reducing/increasing listening comprehension test anxiety: a case of Iranian EFL learners. *English Lang. Teach*. **5**, 178–187.

Bagot R. C. and Meaney M. J. 2010 Epigenetics and the biological basis of gene? environment interactions. *J. Am. Acad. Child Adolesc. Psychiatry* **49**, 752–771.

Berwick R. C. and Chomsky N. 2016 *Why only us: language and evolution*. MIT Press, Cambridge, MS.

Borrelli E., Nestler E. J., Allis C. D. and Sassone-Corsi P. 2008 Decoding the epigenetic language of neuronal plasticity. *Neuron* **60**, 961–974.

Canli T., Ferri J. and Duman E. A. 2009 Genetics of emotion regulation. *Neuroscience* **164**, 43–54.

Caspi A., Williams B., Kim-Cohen J., Craig I. W., Milne B. J., Poulton R. *et al*. 2007 Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proc. Natl. Acad. Sci. USA* **104**, 18860–18865.

Caspi A., Hariri A. R., Andrew H., Uher R. and Moffitt T. E. 2010 Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits. *Am. J. Psychiatry Part A* **167**, 509–527.

Chandrasekaran B., Yi H. G., Blanco N. J., McGeary J. E. and Maddox W. T. 2015 Enhanced procedural learning of speech sound categories in a genetic variant of FOXP2. *J. Neurosci*. **35**, 7808–7812.

Chang H., Hoshina N., Zhang C., Ma Y., Cao H., Wang Y. *et al*. 2017 The protocadherin 17 gene affects cognition, personality, amygdala structure and function, synapse development and risk of major mood disorders. *Mol. Psychiatry*, **23**, 400–412

Chen X. S., Reader R. H., Hoischen A., Veltman J. A., Simpson N. H., Francks C. *et al*. 2017a Next-generation DNA sequencing identifies novel gene variants and pathways involved in specific language impairment. *Sci. Rep*. **7**, 1–17.

Chen Y., Zhao H., Zhang Y. X. and Zuo P. X. 2017b DCDC2 gene polymorphisms are associated with developmental dyslexia in Chinese Uyghur children. *Neural Regener. Res*. **12**, 259–266.

Chomsky N. 2006 *Language and mind*, 3rd edition. Cambridge University Press, Cambridge.

Cope N., Harold D., Hill G., Moskvina V., Stevenson J., Holmans P. *et al*. 2005 Strong evidence that KIAA0319 on chromosome 6p is a susceptibility gene for developmental dyslexia. *Am. J. Hum. Genet*. **76**, 581–591.

Crespi B., Read S. and Hurd P. 2017 Segregating polymorphisms of FOXP2 are associated with measures of inner speech, speech fluency and strength of handedness in a healthy population. *Brain Lang*. **173**, 33–40.

Day J. J. and Sweatt J. D. 2010 DNA methylation and memory formation. *Nat. Neurosci*. **13**, 1319–1323.

Day J. J. and Sweatt J. D. 2011 Epigenetic mechanisms in cognition. *Neuron* **70**, 813–829.

Deters K. D., Nho K., Risacher S. L., Kim S., Ramanan V. K., Crane P. K. *et al*. 2017 Genome-wide association study of language performance in Alzheimer's disease. *Brain Lang*. **172**, 22–29.

Dolcos F., Iordan A. D. and Dolcos S. 2011 Neural correlates of emotion–cognition interactions: a review of evidence from brain imaging investigations. *J. Cognit. Psychol*. **23**, 669–694.

Dzafic I., Burianova H., Periyasamy S. and Mowry B. 2018 Association between schizophrenia polygenic risk and neural correlates of emotion perception. *Psychiatry Res.: Neuroimaging* **276**, 33–40.

Estruch S. B., Graham S. A., Chinnappa S. M., Deriziotis P. and Fisher S. E. 2016 Functional characterization of rare FOXP2 variants in neurodevelopmental disorder. *J. Neurodev Disord*. **8**, 44.

Fass D. M., Schroeder F. A., Perlis R. H. and Haggarty S. J. 2014 Epigenetic mechanisms in mood disorders: targeting neuroplasticity. *Neuroscience* **264**, 112–130.

Felling R. J. and Song H. 2015 Epigenetic mechanisms of neuroplasticity and the implications for stroke recovery. *Exp. Neurol*. **268**, 37–45.

Filges I., Shimojima K., Okamoto N., Röthlisberger B., Weber P., Huber A. R. *et al*. 2011 Reduced expression by SETBP1 haploinsufficiency causes developmental and expressive language delay indicating a phenotype distinct from Schinzel-Giedion syndrome. *J. Med. Genet*. **48**, 117–122.

Fisher S. E. and Scharff C. 2009 FOXP2 as a molecular window into speech and language. *Trends Genet*. **25**, 166–177.

Fried I., Wilson C. L., Morrow J. W., Cameron K. A., Behnke E. D., Ackerson L. C. and Maidment N. T. 2001 Increased dopamine release in the human amygdala during performance of cognitive tasks. *Nat. Neurosci*. **4**, 201–206.
Gialluisi A., Newbury D. F., Wilcutt E. G., Consortium T. S. L. I. and Luciano M. 2014 Genome-wide screening for DNA variants associated with reading and language traits. *Genes Brain Behav*. **13**, 686–701.
Golestani N. and Zatorre R. J. 2009 Individual differences in the acquisition of second language phonology. *Brain Lang*. **109**, 55–67.
Gräff J. and Mansuy I. M. 2008 Epigenetic codes in cognition and behaviour. *Behav. Brain Res*. **192**, 70–87.
Gray J. A. 1990 Brain systems that mediate both emotion and cognition. *Cognit. Emotion* **4**, 269–288.
Hannula-Jouppi K., Kaminen-Ahola N., Taipale M., Eklund R., Nopola-Hemmi J., Kääriäinen H. and Kere J. 2005 The axon guidance receptor gene ROBO1 is a candidate gene for developmental dyslexia. *PLoS Genet*. **1**, 0467–0474.
Hauser M. D., Chomsky N. and Fitch W. T. S. 2002 The faculty of language: what is it, who has it, and how did it evolve? *Science* **298**, 1569–1579.
Hippolyte L., Maillard A. M., Rodriguez-Herreros B., Pain A., Martin-Brevet S., Ferrari C. *et al*. 2016 The number of genomic copies at the 16p11.2 locus modulates language, verbal memory and inhibition. *Biol. Psychiatry* **80**, 129–139.
Isgett S. F., Algoe S. B., Boulton A. J., Way B. M. and Fredrickson B. L. 2016 Common variant in OXTR predicts growth in positive emotions from loving-kindness training. *Psychoneuroendocrinology* **73**, 244–251.
Jennifer J. M. and Ponniah R. J. 2017 Investigating the levels, types and causes of second language writing anxiety among Indian freshmen. *J. Asia TEFL* **14**, 557–563.
Jennifer J. M. and Ponniah J. R. 2018 Acquisition of writing by reading and its impact on cognition. In *The idea and practice of reading* (ed. R. J. Ponniah and S. Venkatesan), pp. 41–56. Springer, Singapore.
Keifer J. 2017 Primetime for learning genes. *Genes* **8**, 69.
Kennedy A. J., Rahn E. J., Paulukaitis B. S., Michael T. P., Day J. J., David J. *et al*. 2016 Tcf4 regulates synaptic plasticity, DNA methylation and memory function. *Cell Rep*. **16**, 2666–2685.
Kitayama S., King A., Hsu M., Liberzon I. and Yoon C. 2016 Dopamine-system genes and cultural acquisition: the norm sensitivity hypothesis. *Curr. Opin. Psychol*. **8**, 167–174.
Knowles E. E. M., Mathias S. R., McKay D. R., Sprooten E., Blangero J., Almasy L. and Glahn D. C. 2014 Genome-wide analyses of working-memory ability: a review. *Curr. Behav. Neurosci. Rep*. **1**, 224–233.
Krashen S. D. 1982 *Principles and practice in second language acquisition*, 1st edition. Pergamon Press Inc., Oxford.
Ladd D., Dediu D. and Kinsella A. 2008 Languages and genes: reflections on biolinguistics and the nature-nurture question. *Biolinguistics* **2**, 114–126. Retrieved from http://www.biolinguistics.eu/index.php/biolinguistics/article/view/39/61.
Laffin J. J. S., Raca G., Jackson C. A., Strand E. A., Jakielski K. J. and Shriberg L. D. 2012 Novel candidate genes and regions for childhood apraxia of speech (CAS) identified by array comparative genomic hybridization. *Genet. Med*. **14**, 928–936.
Lai C. S. L., Fisher S. E., Hurst J. A. and Vargha-khadem F. 2001 A forkhead-domain gene is mutated in a severe speech and language disorder. *Nature* **413**, 519–523.
Lesca G., Rudolf G., Bruneau N., Lozovaya N., Labalme A., Boutry-Kryza N. *et al*. 2013 GRIN2A mutations in acquired epileptic aphasia and related childhood focal epilepsies and encephalopathies with speech and language dysfunction. *Nat. Genet*. **45**, 1061–1066.
Levenson J. and Sweatt J. 2005 Epigenetic mechanisms in memory formation. *Nat. Rev. Neurosci*. **6**, 108–118.
Levinson S. C. 2012 The original sin of cognitive science. *Top. Cognit. Sci*. **4**, 396–403.
Liu K. X., Edwards B., Lee S., Finelli M. J., Davies B., Davies K. E. and Oliver P. L. 2015 Neuron-specific antioxidant OXR1 extends survival of a mouse model of amyotrophic lateral sclerosis. *Brain* **138**, 1167–1181.
Mc Ewen B. 2015 Epigenetics and learning. *Trends Neurosci. Educ*. **4**, 108–111.
McEwen B. S. 2016 In pursuit of resilience: stress, epigenetics, and brain plasticity. *Ann. N Y Acad. Sci*. **1373**, 56–64.
McEwen B. S., Eiland L., Hunter R. G. and Miller M. M. 2012 Stress and anxiety: structural plasticity and epigenetic regulation as a consequence of stress. *Neuropharmacology* **62**, 3–12.
McNamara P. and Durso R. 2018 The dopamine system, Parkinson's disease and language function. *Curr. Opin. Behav. Sci*. **21**, 1–5.
Méndez López M. G. and Fabela Cárdenas M. A. 2014 Emotions and their effects in a language learning Mexican context. *System* **42**, 298–307.
Meng H., Smith S. D., Hager K., Held M., Liu J., Olson R. K. *et al*. 2005 DCDC2 is associated with reading disability and modulates neuronal development in the brain. *Proc. Natl. Acad. Sci. USA* **102**, 17053–17058.
Mondal P. 2016 *Language and cognitive structures of emotion*. Palgrave Macmillan.
Morgan A. T., Mei C., Da Costa A., Fifer J., Lederer D., Benoit V. *et al*. 2015a Speech and language in a genotyped cohort of individuals with Kabuki syndrome. *Am. J. Med. Genet. Part A* **167**, 1483–1492.
Morgan T. J. H., Uomini N. T., Rendell L. E., Chouinard-Thuly L., Street S. E., Lewis H. M. *et al*. 2015b Experimental evidence for the co-evolution of hominin tool-making teaching and language. *Nat. Commun*. **6**, 1–8.
Moya C. and Henrich J. 2016 Culture-gene coevolutionary psychology: cultural learning, language, and ethnic psychology. *Curr. Opin. Psychol*. **8**, 112–118.
Mozzi A., Riva V., Forni D., Sironi M., Marino C., Molteni M. *et al*. 2017 A common genetic variant in FOXP2 is associated with language-based learning (dis)abilities: evidence from two Italian independent samples. *Am. J. Med. Genet. Part B* **174**, 578–586.
Newbury D. F., Winchester L., Addis L., Paracchini S., Buckingham L. L., Clark A. *et al*. 2009 CMIP and ATP2C2 modulate phonological short-term memory in language impairment. *Am. J. Hum. Genet*. **85**, 264–272.
Ni H. 2012 The effects of affective factors in SLA and pedagogical implications. *Theory Pract. Lang. Stud*. **2**, 1508–1513.
Nieto S. J., Patriquin M. A., Nielsen D. A. and Kosten T. A. 2016 Don't worry; be informed about the epigenetics of anxiety. *Pharmacol. Biochem. Behav*. **146–147**, 60–72.
Ocklenburg S., Arning L., Hahn C., Gerding W. M., Epplen J. T., Güntürkün O. and Beste C. 2011 Variation in the NMDA receptor 2B subunit gene GRIN2B is associated with differential language lateralization. *Behav. Brain Res*. **225**, 284–289.
Ölmez F. 2015 An investigation into the relationship between L2 reading motivation and reading achievement. *Procedia – Soc. Behav. Sci*. **199**, 597–603.
Owens S. F., Picchioni M. M., Rijsdijk F. V., Stahl D., Vassos E., Rodger A. K. *et al*. 2011 Genetic overlap between episodic memory deficits and schizophrenia: results from the Maudsley Twin Study. *Psychol. Med*. **41**, 521–532.
Pessoa L. 2008 On the relationship between emotion and cognition. *Nat. Rev. Neurosci*. **9**, 148–158.

Peter C. J., Fischer L. K., Kundakovic M., Garg P., Jakovcevski M., Dincer A. *et al*. 2016 DNA Methylation signatures of early childhood malnutrition associated with impairments in attention and cognition. *Biol. Psychiatry* **80**, 765–774.

Pinker S. 2007 *The language instinct*, 3rd edition. William Moorw and Company, New York.

Ponniah R. J. 2009 The role of grammar: an insight into the skill-building and the output hypotheses. *Mod. J. Appl. Linguist*. **1**, 275–285.

Ponniah R. J. 2011 Incidental acquisition of vocabulary by reading. *Read Matrix* **11**, 135–139.

Reich P. A. and Richards B. A. 2004 Epigenetics and language: the minimalist program, connectionism, and biology. *Linguist. Atl*. **25**, 7–21.

Rendall A. R., Tarkar A., Contreras-Mora H. M., LoTurco J. J. and Fitch R. H. 2017 Deficits in learning and memory in mice with a mutation of the candidate dyslexia susceptibility gene Dyx1c1. *Brain Lang*. **172**, 30–38.

Roll P., Vernes S. C., Bruneau N., Cillario J., Ponsole-Lenfant M., Massacrier A. *et al*. 2010 Molecular networks implicated in speech-related disorders: FOXP2 regulates the SRPX2/uPAR complex. *Hum. Mol. Genet*. **19**, 4848–4860.

Satterlee J. S., Beckel-Mitchener A., Little R. A., Procaccini D., Rutter J. L. and Lossie A. C. 2015 Neuroepigenomics: resources, obstacles, and opportunities. *Neuroepigenetics* **1**, 2–13.

Scerri T. S., Darki F., Newbury D. F., Whitehouse A. J. O., Peyrard-Janvid M., Matsson H. *et al.* 2012 The dyslexia candidate locus on 2p12 is associated with general cognitive ability and white matter structure. *PLoS One* **7**, e50321.

Scheidegger M., Henning A., Walter M., Boeker H., Weigand A., Seifritz E. and Grimm S. 2016 Effects of ketamine on cognition-emotion interaction in the brain. *NeuroImage*. **124** (Pt A), 8–15.

Schulze K., Vargha-Khadem F. and Mishkin M. 2018 Phonological working memory and FOXP2. *Neuropsychologia* **108**, 147–152.

Scult M. A. and Hariri A. R. 2018 A brief introduction to the neurogenetics of cognition-emotion interactions. *Curr. Opin. Behav. Sci*. **19**, 50–54.

Sniekers S., Stringer S., Watanabe K., Jansen P. R., Coleman J. R. I., Krapohl E. *et al*. 2017 Genome-wide association meta-analysis of 78,308 individuals identifies new loci and genes influencing human intelligence. *Nat. Genet*. **49**, 1107–1112.

Sriganesh R., Rahul D. R. and Ponniah R. J. 2018 Genetics of reading ability and its role in solving reading difficulties. In *The idea and practice of reading* (ed. R. J. Ponniah and S. Venkatesan), pp. 125–139. Springer, Singapore.

Sterelny K. 2012 Language, gesture, skill: the co-evolutionary foundations of language. *Philos. Trans. R. Soc. B: Biol. Sci*. **367**, 2141–2151.

Taipale M., Kaminen N., Nopola-Hemmi J., Haltia T., Myllyluoma B., Lyytinen H. *et al*. 2003 A candidate gene for developmental dyslexia encodes a nuclear tetratricopeptide repeat domain protein dynamically regulated in brain. *Proc. Natl. Acad. Sci. USA* **100**, 11553–11558.

Telese F., Gamliel A., Skowronska-Krawczyk D., Garcia-Bassets I. and Rosenfeld M. G. 2013 Seq-ing insights into the epigenetics of neuronal gene regulation. *Neuron* **77**, 606–623.

Thevenon J., Callier P., Andrieux J., Delobel B., David A., Sukno S. *et al*. 2013 12p13.33 microdeletion including ELKS/ERC1, a new locus associated with childhood apraxia of speech. *Eur. J. Hum. Genet*. **21**, 82–88.

Toyokawa S., Uddin M., Koenen K. C. and Galea S. 2012 How does the social environment 'get into the mind'? Epigenetics at the intersection of social and psychiatric epidemiology. *Soc. Sci. Med*. **74**, 67–74.

Tran C., Wigg K. G., Zhang K., Cate-Carter T. D., Kerr E., Field L. L. *et al*. 2014 Association of the ROBO1 gene with reading disabilities in a family-based analysis. *Genes, Brain Behav*. **13**, 430–438.

Turner S. J., Hildebrand M. S., Block S., Damiano J., Fahey M., Reilly S. *et al*. 2013 Small intragenic deletion in FOXP2 associated with childhood apraxia of speech and dysarthria. *Am. J. Med. Genet. Part A* **161**, 2321–2326.

Vernes S. C., Newbury D. F., Abrahams B. S., Winchester L., Nicod J., Groszer M. *et al*. 2008 A functional genetic link between distinct developmental language disorders. *N. Engl. J. Med*. **359**, 2337–2345.

Villanueva P., Nudel R., Hoischen A., Fernández M. A., Simpson N. H., Gilissen C. *et al*. 2015 Exome sequencing in an admixed isolated population indicates NFXL1 variants confer a risk for specific language impairment. *PLoS Genet*. **11**, e1004925.

Wang X., Hui Z., Dai X., Terry P. D., Zhang Y., Ma M. *et al*. 2017 Micronutrient-fortified milk and academic performance among Chinese middle school students: a cluster-randomized controlled trial. *Nutrients* **9**, 226.

Williams L. M., Tsang T. W., Clarke S. and Kohn M. 2010 An "integrative neuroscience" perspective on ADHD: linking cognition, emotion, brain and genetic measures with implications for clinical support. *Expert Rev. Neurother*. **10**, 1607–1621.

Wingo A. P., Almli L. M., Stevens J. S., Jovanovic T., Wingo T. S., Tharp G. *et al*. 2017 Genome-wide association study of positive emotion identifies a genetic variant and a role for microRNAs. *Mol. Psychiatry* **22**, 774–783.

Wong P. C. M. and Ettlinger M. 2011 Predictors of spoken language learning. *J. Commun. Disord*. **44**, 564–567.

Worthey E. A., Raca G., Laffin J. J., Wilk B. M., Harris J. M., Jakielski K. J. *et al*. 2013 Whole-exome sequencing supports genetic heterogeneity in childhood apraxia of speech. *J. Neurodev. Disord*. **5**, 29.

Corresponding editor: S. Ganesh