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Genetic intersections of language and neuropsychiatric conditions

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Abstract Abnormal language and communication are common manifestations of neuropsychiatric conditions. Inversely, children with impaired language are more likely to develop psychiatric disorders than their peers. To better understand the shared basis of language and mental health, this review examines the behavioral and neurobiological features of aberrant language in five major neuropsychiatric conditions. Special attention is paid to genes implicated in both language and neuropsychiatric disorders, as they reveal biological domains likely to underpin the processes controlling both. These themes include master transcriptional regulators, like *FOXP2*, key developmental regulators, like *AUTS2*, and mediators of neurotransmission, like *GRIN2A* and *CACNA1C*.

Keywords Language · Genetics · Psychaitry · Neurodevelopment

1 Introduction

Changes in verbal communication patterns are endemic to neuropsychiatric conditions. From unintelligible speech in many cases of schizophrenia to a total lack of verbal communication often seen in autism spectrum disorder, these features can be reliably observed at the neurological and behavioral level. Additionally, children with language impairments are at increased risk for adult-onset psychiatric conditions [90]. These observations suggest common mechanisms that perturb both language and mental health, but dissection of the genetic factors underlying this connection has been challenging. This difficulty is due to the relative immaturity of the field of language genetics (reviewed in [44]), the difficulty of collecting language phenotypes at the scales required for well-powered genetic studies, and the complex etiology of neuropsychiatric conditions. A better understanding of the molecular, cellular, and circuit-level changes that perturb language and men-

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tal health will ultimately improve therapeutic prospects, both for those suffering from mental illness and those living with a language disorder.

This review provides a broad overview of the behavioral and neurological connections of language to each of five major neuropsychiatric conditions (schizophrenia, bipolar disorder, major depressive disorder, autism spectrum disorder, and attention deficit/hyperactivity disorder). Each section includes a description of evidence supporting the shared genetics of language and the respective condition. In most cases, specific linguistic features of a disorder cannot be linked to genetic variations in the genes discussed, however, the presence of these commonalities is notable and deserves further research. For convenience, a summary of the association of language-implicated genes with neuropsychiatric conditions can be found in Table 1.

2 Schizophrenia

In addition to delusions and hallucinations, disorganized language has long been recognized as a feature of schizophrenia [21]. This trait — typically referred to as "thought disorder" — has been actively studied for over 50 years [158], and has two primary distinctions. Negative thought disorder is typified by poverty of speech, while positive thought disorder results in confusing speech interrupted by irrelevant word associations. There was long a hope that quantifying these language disturbances in schizophrenia would reflect the state of overall symptom severity, but years of studies rarely bore fruit [37, 225]. More recently, automated semantic and linguistic analyses have shown a remarkable ability to predict outcomes, like psychosis, from speech patterns in schizophrenic patients [15, 40]. An excellent overview of the language features and psycholinguistics of schizophrenia can be found in [107, 108].

The advent of high resolution structural brain imaging in recent decades has also helped identify overlap between schizophrenia and known language centers of the brain [151, 187, 197]. Functional imaging has provided additional insights, such as task-based [97] and resting state [195] studies that identified perturbations in Wernicke's area (Brodmann 22) in schizophrenics with thought disorder. In addition, response to speech between Brodmann area 22 and area 21 was able to discriminate schizophrenic patients from controls [174], and functional lateralization of language was decreased in schizophrenics [41, 177].

Neural signalling is a primary focus in mechanistic and pharmacological research on schizophrenia, and several genes implicated in language development play key roles in neurotransmission. *GRIN2A* and *GRIN2B* — subunits of the NMDA receptor that modulates the activity of dopamine, critical for long-term potentiation and memory — were implicated in multiple schizophrenia case-control studies [85, 86, 129, 131, 226] and Genome Wide Association Studies (GWAS) [63, 83, 121, 160, 206]. Major disruptions to *GRIN2A* cause a focal epilepsy with comorbid speech and language deficits [115], and a child with language impairment was found to have a *de novo* missense variant in the gene [33]. Genetic variation in *GRIN2B* is associated with alterations to language lateralization within the brain [149]. Microdeletions in another ion channel linked to schizophrenia through case-control [66] and GWAS [5, 83, 121, 142, 155] — *CACNA1C* — have also been shown to result in language delays [207].

Another well-esablished schizophrenia risk gene, *COMT*, encodes a methyltransferase responsible for dopamine metabolism. One allele (Val158) of *COMT* has significantly higher activity than the other common allele (Met158). Homozygosity for Val158 *COMT* was associated with worse performance on non-word repetition task in younger children, along with decreased activation in the temporal region around Wernicke's area [198]. The Val158 allele has also been repeatedly linked to schizophrenia through family [106], case-control [188], functional studies [24, 152], and recently in GWAS [63].

Genes with the broadest impact on language development and psychiatric disorders are often master regulators — those that control the expression of other genes through a variety of mechanisms. *FOXP1* and *FOXP2* are transcription factors that regulate the expression of hundreds of genes in the brain. Early family studies of rare inherited language disorders identified the cause as a loss of function mutation in *FOXP2* [109], making it one of the best established language-related genes [141]. Loss of function mutations in the closely related *FOXP1* were subsequently found to cause language impairments and intellectual disabilities in several case studies [72, 78]. *FOXP1* has shown stronger signal in schizophrenia GWAS [63, 83, 121], while *FOXP2* has been linked to auditory hallucinations [133, 182] and altered grey matter concentration [192]. Some studies suggest common genetic variation in *FOXP2* contributes to schizophrenia risk [120], while others only find effects on symptomatology within schizophrenia cases [168].

Although an adult onset condition, schizophrenia has neurodevelopmental roots [220], underscored by the presence of developmental genes among its risk factors. For example, the *ROBO1* and *ROBO2* genes are critical for axon guidance and neuronal cell migration, and show evidence for involvement both in language and schizophrenia risk. *ROBO1* was implicated in a recent schizophrenia GWAS [83], and variation in both genes was associated with task-based fMRI activation in schizophrenic patients [164]. *ROBO1* was identified in a targeted investigation of a linkage region for language impairment in the context of reading disability [14] and dyslexia [132], while a GWAS of infant vocabulary found significant signal near *ROBO2* [165].

Another developmental protein responsible for neuronal migration, encoded by *DCDC2*, aids ciliary signalling by promoting microtubule polymerization. Deletions in *DCDC2* are linked to reading disorders [138], and common variation in the gene correlates with temporal and prefrontal grey matter volume in schizophrenic patients [87]. The *CNTNAP2* gene encodes a neurexin that also aids neural projection, by facilitating interactions between neurons and glia. *CNTNAP2* is downregulated by *FOXP2* and variation within the gene correlates with both language impairments [216], and overall language ability [221, 222]. Schizophrenia has been linked to *CNTNAP2* though GWAS meta-analysis [218], as well as a targeted investigation [88].

3 Bipolar Disorder

Pressured speech (i.e., rapid speech with unwarranted urgency) is a hallmark of manic episodes in bipolar disorder [94]. Whether performance on language assessments reflects this state-dependent behavior has been contested for some time. Mania-specific deficits in semantic fluency were observed in some studies [102],

while others observed deficits regardless of state [145,181]. Semantic fluency tasks measure the ability to correctly categorize words — not overall verbosity — where lower scores primarily result from aberrant word associations [201]. In a recent meta-analysis, semantic fluency was found to be decreased across mood states, but significantly better in patients during manic episodes [169]. Greater fluency impairments have also been observed in bipolar I compared to bipolar II [30], and meta-analysis of improvements in fluency with pharmacological treatment found no effect [23].

The potential effects of bipolar disorder on language can also be observed directly within the brain. Bipolar I patients in remission from a manic episode were found to have reduced volume in the left dorsolateral prefrontal cortex (Brodmann 46) [58], which has been implicated in language by fMRI [98,183]. Bipolar patients also showed altered functional connectivity between the auditory cortex and regions of the temporal lobe – including Wernicke's area [171] — while several metabolites had lower relative concentrations in these regions in a proton magnetic resonance spectroscopy study [9]. Evidence for altered activity extends to EEG analysis, where bipolar patients had reduced responsivity to syntactic mistakes in sentences [113].

Several genes important for neurotransmission in bipolar are also implicated in language development. The *RNLS* gene — which encodes the renalase responsible for metabolizing hormones like epinephrine and norepinephrine — has been linked to remission of bipolar symptom in a GWAS pathway analysis [52]. *RNLS* was identified in a recent cross-population GWAS of performance on rapid automatized naming, a robust reading and language task [210].

A number of bipolar-associated genes are similarly linked to schizophrenia and language development. For example, *CACNA1C* was one one of the first genome-wide significant loci in bipolar disorder [191], with strong subsequent support [5,31,55,124]. Similarly, *GRIN2B* was identified in multiple bipolar linkage studies [53,131,234], and a variant in *ROBO2* was the strongest signal in a GWAS of the dissociation effects of ketamine in bipolar [69].

The shared genetic etiology of language development, bipolar disorder, and schizophrenia extends to developmental genes as well. For instance, meta-analysis GWAS for bipolar and schizophrenia identified the key *FOXP2* target *CNTNAP2* [218] as a risk gene. *CDH2*, the gene encoding Cadherin 2 — an integral protein at cell junctions with key roles in brain lateralization — contained rare missense variants in 6 of 12 individuals from an isolated population enriched for language impairments [100], and showed some GWAS signal for dysthymic temperament in bipolar disorder [67]. The neurogenesis-promoting *PALB2* gene was also identified with modest GWAS signal for bipolar [38], then subsequently supported by non-parametric reanalysis [89] and a targeted case-control investigation [205]. In a family-based exome sequencing study of language impairment, one proband was found to be a compound heterozygote for damaging variation in *PALB2* [33].

The developmentally critical *AUTS2* is involved in dendrite extension, neuronal migration, H3-K4 methylation and H4-K16 acetylation (marks of active transcription). Rare deletions [6] and non-synonymous variants [33] in *AUTS2* were identified in children with language impairments, and showed nominal signal in GWAS for seasonal pattern mania in bipolar [114]. Another regulator of H3-K4 methylation (*KMT2D*) was identified in a bipolar GWAS [82], and found in a compound heterozygous state in a language impaired child [33]. The under-

studied DNA binding protein *ZNF385D* was the strongest signal in a reading and language GWAS [49], and has shown some association with bipolar in several GWAS [55, 124].

4 Major Depressive Disorder

Decreased verbal communication — long recognized as a hallmark of major depressive disorder (MDD) [26] — has been linked to other psychomotor symptoms, like reduced reaction time and gross motor activity [28]. Manual linguistic analysis was better able to identify MDD patients than schizophrenic or bipolar patients, indicating the poverty of speech in MDD is more easily quantified than the linguistic traits of the other disorders [125].

Across studies, the psychomotor deficits of MDD primarily impact processing speed and capability [18, 61]. Reviews of assessments for psychomotor retardation in MDD can be found in both [27] and [17], with particularly thorough discussions of conflicting evidence for the use of psychomotor phenotypes to inform the course of pharmacological treatment. Most evidence for the semantic impacts of MDD, is more complex, indicating an interplay between the emotional context of speech and its semantic associations, rather than an overall compromised semantic ability [13, 99].

The nuances of language perturbations in MDD are reflected in brain structure and function. Functional imaging in a pair of studies found increased connectivity in MDD patients from Brodmann area 47 to the left angular gyrus [34, 176]. The left angular gyrus was one of the first language-related areas identified by fMRI [20], while Brodmann 47 (ventrolateral orbital frontal cortex) has been implicated in both language and depression. Linguistically, Brodmann 47 seems to largely control semantic processing [163, 214, 229], while also connecting to the amygdala, linking it to negative reward or punishment [47, 64, 166]. In both [34] and [176] this phenomenon is taken as evidence for a circuit which promotes rumination on negative-self perception.

As in schizophrenia and bipolar disorder, interest in improved pharmacological treatment has driven substantial genetic research on MDD. Investigations into *GRIN2A* and *GRIN2B* largely focused on how variation in those genes may modulate the efficacy of ketamine, which targets the NMDA receptor. Patients with treatment resistant depression are more likely to have a regulatory variant in *GRIN2B* (rs1805502) [233], which has shown some association with drug response in MDD patients [213]. Experimental evidence in rodents [203] and postmortem gene expression [93] have also suggested a role for *GRIN2A* in depression, but studies in human populations did not show variation within *GRIN2A* to be associated with MDD [81]. In addition to receptors, variations in some intracellular signalling genes were linked to altered response to antidepressants in MDD, like the *PRKCH* gene [223]. Widely expressed and interacting with a number of cellular signalling pathways depending on the tissue, *PRKCH* was also identified in a GWAS for reading and language ability [126].

Developmental genes show the greatest degree of intersection between MDD and language. *ROBO2* and the *SEMA6D* gene — encoding a transmembrane semaphorin with roles in axon guidance and branching — were identified in a GWAS of neuroticism and depressive symptoms [144]. A recent MDD meta-analysis

GWAS also identified significant signal in *SEMA6D* [79]. In one case study, a balanced translocation interrupting *SEMA6D* was found in a child with language impairment, while a novel missense mutation was identified in another study [33]. The same proband from [33] also had a loss of function mutation in the *SYNPR* gene, which encodes the cell junction synaptoporin and was identified in one MDD GWAS [71].

The estrogen receptor *ESR1* has a substantial impact on neural growth and development, in addition to its more obvious role in sex characteristics and reproductive development. Genetic variation in *ESR1* had the strongest association with language in a multivariate GWAS of an isolated population enriched for language development problems [100]. Depressive symptoms in children were repeatedly found to be mediated by sex and genetic variation in *ESR1* [139, 215]. Studies focusing on *ESR1* in women alone found the gene to be associated with lifetime depression (acute or chronic) [180], and modulated by post-menopausal hormone therapy [95].

Developmental genes linked to bipolar, schizophrenia, and language are also strongly associated with MDD risk. For instance, *CACNA1C* was identified in targeted studies of MDD [66], with one finding that bipolar-associated genetic variants correlated with drug-induced suicidality [29]. This association has been backed by multiple MDD GWAS [5, 124]. Functional and regulatory variation in *CNTNAP2* is also associated with the combined effects of major depression and anxiety [65]. Targeted investigation of language and autism-related SNPs in the *CNTNAP2* gene [88] identified two alleles linked to MDD.

5 Autism Spectrum Disorder

Social communication deficits in autism spectrum disorder (ASD) — particularly those linked to language usage — are often much more severe than the disorders described thus far. Given the early onset of ASD, these deficits are unsurprisingly linked to developmental processes. Children with compromised language skills as young as 12-24 months are more likely to be later diagnosed with ASD [140], though this may be confounded by expressive ability deficits. The level of expressive language deficits in adolescents with ASD is correlated with infantile vocalizations [157], and early interventions to support language development repeatedly showed significant beneficial effects [73]. Such evidence has prompted significant research into how and when differences in language skills manifest, and how to support their development.

An important technique for engaging language skills in children is joint attention, where an adult directs the focus of communication to a particular object. Children with ASD are less likely to respond to joint attention, and meta-analysis has found the extent of ASD probands' response to joint attention is more predictive of eventual language ability than that of typically developing children [22]. Another relatively distinctive hallmark of ASD is echolalia. Much of the early research on language in ASD focused on this phenomenon [196], as it raised the possibility that the compromised communication of many with ASD may result from a lack of ability — and not a lack of desire — to communicate. Even more unique to ASD is a true "loss" of previously acquired language ability. In the broader literature, this is more commonly associated with neurodegeneration or

injury than aberrant neurodevelopment, but some estimates find that up to 15% of children with ASD experience language regression [161]. An excellent review of the many dimensions of language in ASD can be found in [202].

A neurological feature common in ASD is broad neural "overgrowth" in both grey and white matter [184]. Several structural MRI studies in children [111] and adolescents [92] with ASD found particularly large increases in the superior temporal gyrus, which includes the auditory cortex (Brodmann 41 and 42) and Wernicke's area (Brodmann 22). Diffusion imaging shed additional light on this phenomenon, finding long-term disruptions to white matter in individuals with ASD [77]. Together, these observations suggest a neural overgrowth followed by a sort of over-maturation [77], with aberrations in the structural connectivity of language areas reported as early as infancy [118].

Functionally, EEG studies found reduced neural responses to speech in children with ASD, and some increased responses to non-speech stimuli [105], which might reflect the sensory sensitivities experienced by people with ASD. EEG also suggests those with ASD have deficits utilizing audio feedback from their own vocalizations [156], which was hypothesized to underlie the difficulty some ASD individuals have with the patterns of stress and intonation of language (prosody) [59]. For an extensive review on neuroanatomy and functionality in ASD, see [193].

Given the unique patterns of neural overgrowth and disorganization apparent in ASD, it is unsurprising that several developmental genes underlying language also confer risk for ASD. For example, expression of the axon guidance regulators *ROBO1* and *ROBO2*, were found to be lower in a family-based study of ASD [7]. The previously described *AUTS2* is well-established as a risk gene for ASD [12,199, 212], and a regulator of neuron migration and axon extension. Full loss or deletion of *AUTS2* usually results in a syndromic phenotype including microcephaly and cerebral palsy [19].

Another set of related genes implicated in language — the neurexins *CNTNAP2* and *CNTNAP5* — are robustly linked to ASD. *CNTNAP2* was first identified in ASD via a slue of linkage, gene expression [2], SNP [8], and structural variation [10] findings. The less studied *CNTNAP5* may be even more interesting from a language perspective, as disruptions were found in several families with comorbid ASD and dyslexia [150].

The well-established language gene *FOXP2* has also been been linked to ASD risk. Some families with deletions or other losses of *FOXP2* display comorbid ASD and language impairment, while others display language impairments alone [141]. The comparatively less interrogated *FOXP1* has more substantial support as an ASD risk gene than *FOXP2*. In addition to reports of an ASD-like phenotype in families with loss of function mutations in *FOXP1* [72, 78], subsequent targeted studies identified recurrent mutations in the gene in individuals with ASD [190]. Common genetic variation in *FOXP1* is also associated with ASD, as identified in a recent large GWAS [206].

Common variation in some other language-associated genes is associated with joint risk for ASD and schizophrenia [206]. This shared risk between very different neuropsychiatric conditions has been interpreted as evidence for foundational neurodevelopmental roles of these genes. Mutations in one such gene (*CACNA1C*) causes Timothy Syndrome, which includes heart arrhythmia and autistic features [194]. Another of these genes is the NMDA subunit *GRIN2A*, which was

identified as a candidate for ASD risk in early studies of common genetic variation as well [11, 104].

CACNA1C and *GRIN2A* both play substantial roles in neurotransmission, as well as neurodevelopment. *DOCK4*, also involved in neurotransmission and neurodevelopment is a regulator of the adherens junctions between cells. *DOCK4* was identified as potentially causal gene within the AUTS5 linkage region by case-control [128], and family study [122]. *DOCK4* was further implicated in both ASD and dyslexia, where a deletion in the gene was found to segregate specifically with dyslexia [150]. Finally, the ATP Ca+/Mn2+ pump *ATP2C2* was linked to phonological memory in a combined family and population-based study of language impairment [147]. More recently, variation in this gene was found to correlate with receptive vocabulary in two large ASD cohorts [48], and it was tied to the reduced EEG response to speech sounds [105], and deficits of prosody [156] in individuals with ASD.

6 Attention Deficit/Hyperactivity Disorder

Attention Deficit Disorder / Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental conditions, with incidence estimates around 7% [208]. Broadly, children with ADHD perform worse on language assessments, but there is substantial heterogeneity, and relatively little investigation of the language features of the condition within the core ADHD research field. Instead, most work on the topic comes from the fields of speech pathology and psycholinguistics. This may be because children with ADHD are at risk for language impairments, which also occur in about 7% of children [209]. The largest ADHD case-control study examining language impairments found children with ADHD are at three times greater risk of having language impairments [185].

Conservative estimates of the comorbidity of ADHD and language impairment are around 30% [16], but language ability alone is not a strong predictor of ADHD status [162]. Meta analysis of 21 studies found overall lower expressive, receptive and pragmatic language in children with ADHD [101], yet the shared risk between ADHD and language impairment appears non-symmetric: Children with ADHD are more than twice as likely to have concurrent language problems as children with language problems are to have ADHD [167]. This suggests a lack of attention increases risk for language impairment, but not necessarily the other way around [143]. For a more detailed review on this subject, see [170].

Imaging studies on ADHD and language tend to focus narrowly on specific hypotheses, typically comparing ADHD to dyslexia. For example, one study [186] found both children with ADHD and dyslexic children had smaller left Herschel's gyri than children without either condition. In the large ENIGMA imaging consortium study, children with ADHD had lower surface area in the frontal and temporal lobes [76], while diffusion imaging found perturbations to the superior longitudinal fasciculus fiber tract in adolescents with ADHD [60]. This tract connects several classical language areas, like the angular gyrus, to the sensorimotor regions of the cortex [159].

Nearly all genes implicated in both language development and ADHD have substantial evidence for involvement with the other conditions described here. For example, recurrent copy number variations in *AUTS2* and *CNTNAP2* were found

in families with high incidence of ADHD [50]. The *DCDC2* gene, also linked to MDD, is in a linkage region for both ADHD and dyslexia. Targeted family-based investigation of this region found evidence for a role of *DCDC2*, but not the nearby *KIAA0319* [42], which has also been cited in connection to language. The estrogen receptor gene (*ESR1*) has been associated with conduct disorder and ADHD [36]. Variants upstream of *ESR1* were linked to regulation of executive function in ADHD [200], and variation within the gene was found to be useful in predicting ADHD symptom severity [135]. Finally, *FOXP2* has the most compelling evidence for involvement in both ADHD and language. A targeted investigation of common genetic variants near *FOXP2* showed some signal in a case-control study [172], and it is among the first genome-wide significant loci from GWAS for ADHD [43].

7 Conclusion

Compared to the massive genetic studies of neuropsychiatric conditions, studies of language are relatively modest in statistical power and findings. However, our examination of the results of these two fields together reveals themes of shared molecular risk involving neurotransmission (e.g., *GRIN2A*, *CACNA1C*), development (e.g., *AUTS2*, *CNTNAP2*, *ROBO1*), and transcriptional regulation (e.g., *FOXP2*). These biological themes are particularly well-established in autism genetic research, which is notable because autism is the only major neuropsychiatric condition that includes deficits in social communication as a defining feature. This suggests that in the search for genetic determinants of language, a focus on these themes — and genes associated with neuropsychiatric conditions — may yield more fruit than hypothesis-free approaches. This suggestion is supported by the relative lack of thematic coherence among the language-associated genes that are not known to confer neuropsychiatric risk (Table 1).

To take full advantage of the shared nature of the mechanisms underlying language and mental health, future genetic studies of these traits should cross-pollinate by collecting phenotypic data on mental health (in language studies) and language (in neuropsychiatric studies). This more thorough characterization of language and neuropsychiatric cohorts would allow for robust and informative genetic associations. Although language phenotypes have been historically time-consuming to collect, emerging modes of digital phenotyping will yield rich data from brief in-person or online encounters that will scale to the needs of large genetic studies.

Conflict of interest

The authors declare that they have no conflict of interest.

Table 1: Summary of genes implicated in language and their neuropsychiatric associations

Gene	Language Association	Brain Expressed	SCZ	BP	MDD	ASD	ADHD
ABCC13	[126]	no					
ABCG4	[100]	yes					
ATP2C2	[147]	no				[48]	[116]
ATXN10	[49]	yes	[136]				
AUTS2	[6, 33]	yes	[232]	[114]		[12, 19, 199, 212]	[50]
CACNA1C	[207]	yes	[5, 66, 83, 121, 142, 155, 179]	[5, 31, 55, 124, 179]	[5, 29, 66, 124]	[194, 206]	
CAND1	[165]	yes					
CDC2L2	[126]	yes					
CDH2	[100]	yes		[67]			
CMIP	[146, 147]	yes				[1, 127]	
CNTNAP2	[216, 221, 222]	yes	[88, 218]	[218]	[65, 88]	[2, 8, 10]	[50]
CNTNAP5	[150]	yes	[117, 228]			[150]	
COL4A2	[49]	low	[218]	[218]			
COMT	[198]	yes	[24, 63, 106, 152, 188]				
CUBN	[49]	no		[134]			
DAB2	[148]	low					
DAPK3	[165]	yes					
DAZAP1	[126]	yes					
DCDC2	[138]	low	[87]		[80]		[42,175]
DGKB	[100]	yes			[75]		
DOCK4	[150]	4yes	[4]			[122, 128, 150]	
DPAGT1	[100]	yes					
ERC1	[33, 207]	yes				[189]	
ENTHD1	[100]	no					
ESR1	[100]	low	[74, 137, 219]		[95, 139, 180, 215]	[45, 230, 231]	[36, 135, 200]
FOXP1	[72, 78]	yes	[63, 83, 84, 121]			[72, 190, 206]	[110]
FOXP2	[109, 141]	yes	[120, 133, 168, 182, 192]			[56, 236]	[43, 172]
GRIN2A	[33, 115]	yes	[63, 83, 85, 86, 121, 155, 160, 206]		[81, 93, 203]	[11, 104, 206]	[211]

Table 1: Summary of genes implicated in language and their neuropsychiatric associations

Gene	Language Association	Brain Expressed	SCZ	BP	MDD	ASD	ADHD
GRIN2B	[149]	yes	[63,129, 131,226]	[53, 131,234]	[123, 213,233]	[153, 204,227]	[46,96]
H2AFX	[100]	yes					
HINFP	[100]	yes					
HLCS	[100]	yes					
HYOU1	[100]	yes					
ILK	[100]	yes					
INSC	[165]	low					
KIAA0319	[39, 132, 146, 154]	yes					
KMT2D	[33]	yes		[82]			
NDST4	[49]	low					
NECAB1	[100]	yes					
NFXL1	[217]	low					
NOP9	[148]	yes	[62]				
NOS1AP	[126]	yes	[25,70, 103,224, 235]		[32,112]		
NT5DC2	[100]	yes	[173]	[68]			
NUAK1	[49]	yes					[3]
OPA3	[49]	low					
OXR1	[33]	yes	[119]				
PALB2	[33]	yes		[38,89, 205]			
PLEKHA1	[49]	yes					
PPP2R1B	[100]	yes					
PRKCH	[126]	yes			[223]		
PTGER4	[148]	low					
RCAN3	[126]	yes					
RNLS	[210]	low		[52]			
ROBO1	[14,132]	yes	[83, 121,164]			[7]	
ROBO2	[165]	yes	[63,164]	[69]	[144]	[7]	
SCN9A	[33]	yes	[63]			[178]	
SEMA6D	[33,51]	yes			[79,144]		[43,130]
SETBP1	[35,57,100]	yes					
SIK2	[100]	yes					
SLC6A13	[207]	low					
SRPX2	[33]	low					
STARD9	[33]	yes					
SYNPR	[33]	yes			[71]		
FAM19A1	[49]	yes					
TCP10L2	[100]	low				[91]	
TNC	[100]	yes					

Table 1: Summary of genes implicated in language and their neuropsychiatric associations

Gene	Language Association	Brain Expressed	SCZ	BP	MDD	ASD	ADHD
TRIP6	[100]	yes					
WASHC5	[49]	yes					
WFDC1	[147]	yes					
ZFYVE28	[49]	yes	[54]				
ZNF385D	[49]	yes		[55,124]			[110]

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