

# Genetic intersections of language and neuropsychiatric conditions

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Received: date / Accepted: date

**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 · Psychiatry · 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-established 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 down-regulated 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* through 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 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 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  $\text{Ca}^{+}/\text{Mn}^{2+}$  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
<i>ABCC13</i>	[126]	no					
<i>ABCG4</i>	[100]	yes					
<i>ATP2C2</i>	[147]	no				[48]	[116]
<i>ATXN10</i>	[49]	yes	[136]				
<i>AUTS2</i>	[6, 33]	yes	[232]	[114]		[12, 19, 199, 212]	[50]
<i>CACNA1C</i>	[207]	yes	[5, 66, 83, 121, 142, 155, 179]	[5, 31, 55, 124, 179]	[5, 29, 66, 124]	[194, 206]	
<i>CAND1</i>	[165]	yes					
<i>CDC2L2</i>	[126]	yes					
<i>CDH2</i>	[100]	yes		[67]			
<i>CMIP</i>	[146, 147]	yes				[1, 127]	
<i>CNTNAP2</i>		yes	[88, 218]	[218]	[65, 88]	[2, 8, 10]	[50]
	[216, 221, 222]						
<i>CNTNAP5</i>	[150]	yes	[117, 228]			[150]	
<i>COL4A2</i>	[49]	low	[218]	[218]			
<i>COMT</i>	[198]	yes	[24, 63, 106, 152, 188]				
<i>CUBN</i>	[49]	no		[134]			
<i>DAB2</i>	[148]	low					
<i>DAPK3</i>	[165]	yes					
<i>DAZAP1</i>	[126]	yes					
<i>DCDC2</i>	[138]	low	[87]		[80]		[42, 175]
<i>DGKB</i>	[100]	yes			[75]		
<i>DOCK4</i>	[150]	4yes	[4]			[122, 128, 150]	
<i>DPAGT1</i>	[100]	yes					
<i>ERC1</i>	[33, 207]	yes				[189]	
<i>ENTHD1</i>	[100]	no					
<i>ESR1</i>	[100]	low	[74, 137, 219]		[95, 139, 180, 215]	[45, 230, 231]	[36, 135, 200]
<i>FOXP1</i>	[72, 78]	yes	[63, 83, 84, 121]			[72, 190, 206]	[110]
<i>FOXP2</i>	[109, 141]	yes	[120, 133, 168, 182, 192]			[56, 236]	[43, 172]
<i>GRIN2A</i>	[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
<i>GRIN2B</i>	[149]	yes	[63, 129, 131, 226]	[53, 131, 234]	[123, 213, 233]	[153, 204, 227]	[46, 96]
<i>H2AFX</i>	[100]	yes					
<i>HINFP</i>	[100]	yes					
<i>HLCS</i>	[100]	yes					
<i>HYOU1</i>	[100]	yes					
<i>ILK</i>	[100]	yes					
<i>INSC</i>	[165]	low					
<i>KIAA0319</i>	[39, 132, 146, 154]	yes					
<i>KMT2D</i>	[33]	yes		[82]			
<i>NDST4</i>	[49]	low					
<i>NECAB1</i>	[100]	yes					
<i>NFXL1</i>	[217]	low					
<i>NOP9</i>	[148]	yes	[62]				
<i>NOS1AP</i>	[126]	yes	[25, 70, 103, 224, 235]		[32, 112]		
<i>NT5DC2</i>	[100]	yes	[173]	[68]			
<i>NUAK1</i>	[49]	yes					[3]
<i>OPA3</i>	[49]	low					
<i>OXR1</i>	[33]	yes	[119]				
<i>PALB2</i>	[33]	yes		[38, 89, 205]			
<i>PLEKHA1</i>	[49]	yes					
<i>PPP2R1B</i>	[100]	yes					
<i>PRKCH</i>	[126]	yes			[223]		
<i>PTGER4</i>	[148]	low					
<i>RCAN3</i>	[126]	yes					
<i>RNLS</i>	[210]	low		[52]			
<i>ROBO1</i>	[14, 132]	yes	[83, 121, 164]			[7]	
<i>ROBO2</i>	[165]	yes	[63, 164]	[69]	[144]	[7]	
<i>SCN9A</i>	[33]	yes	[63]			[178]	
<i>SEMA6D</i>	[33, 51]	yes			[79, 144]		[43, 130]
<i>SETBP1</i>	[35, 57, 100]	yes					
<i>SIK2</i>	[100]	yes					
<i>SLC6A13</i>	[207]	low					
<i>SRPX2</i>	[33]	low					
<i>STARD9</i>	[33]	yes					
<i>SYNPR</i>	[33]	yes			[71]		
<i>FAM19A1</i>	[49]	yes					
<i>TCP10L2</i>	[100]	low				[91]	
<i>TNC</i>	[100]	yes					

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

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

## References

1. der Aa, N.V., Vandeweyer, G., Reyniers, E., Kenis, S., Dom, L., Mortier, G., Rooms, L., Kooy, R.F.: Haploinsufficiency of CMIP in a girl with autism spectrum disorder and developmental delay due to a de novo deletion on chromosome 16q23.2. *Autism Research* **5**(4), 277–281 (2012). DOI 10.1002/aur.1240. URL <https://doi.org/10.1002%2Faur.1240>
2. Alarcón, M., Abrahams, B.S., Stone, J.L., Duvall, J.A., Perederiy, J.V., Bomar, J.M., Sebat, J., Wigler, M., Martin, C.L., Ledbetter, D.H., Nelson, S.F., Cantor, R.M., Geschwind, D.H.: Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. *The American Journal of Human Genetics* **82**(1), 150–159 (2008). DOI 10.1016/j.ajhg.2007.09.005. URL <https://doi.org/10.1016%2Fj.ajhg.2007.09.005>
3. Alemany, S., Ribasés, M., Vilor-Tejedor, N., Bustamante, M., Sánchez-Mora, C., Bosch, R., Richarte, V., Cormand, B., Casas, M., Ramos-Quiroga, J.A., Sunyer, J.: New suggestive genetic loci and biological pathways for attention function in adult attention-deficit/hyperactivity disorder. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **168**(6), 459–470 (2015). DOI 10.1002/ajmg.b.32341. URL <https://doi.org/10.1002%2Fajmg.b.32341>
4. Alkelai, A., Lupoli, S., Greenbaum, L., Kohn, Y., Kanyas-Sarner, K., Ben-Asher, E., Lancet, D., Macciardi, F., Lerer, B.: DOCK4 and CEACAM21 as novel schizophrenia candidate genes in the jewish population. *The International Journal of Neuropsychopharmacology* **15**(04), 459–469 (2012). DOI 10.1017/s1461145711000903. URL <https://doi.org/10.1017%2Fs1461145711000903>
5. Amare, A.T., Vaez, A., Hsu, Y.H., Direk, N., Kamali, Z., Howard, D.M., McIntosh, A.M., Tiemeier, H., Bltman, U., Snieder, H., Hartman, C.A.: Bivariate genome-wide association analyses of the broad depression phenotype combined with major depressive disorder, bipolar disorder or schizophrenia reveal eight novel genetic loci for depression. *Molecular Psychiatry* (2019). DOI 10.1038/s41380-018-0336-6. URL <https://doi.org/10.1038%2Fs41380-018-0336-6>
6. Amarillo, I.E., Li, W.L., Li, X., Vilain, E., Kantarci, S.: De novo single exon deletion ofAUTS2in a patient with speech and language disorder: A review of disruptedAUTS2and further evidence for its role in neurodevelopmental disorders. *American Journal of Medical Genetics Part A* **164**(4), 958–965 (2014). DOI 10.1002/ajmg.a.36393. URL <https://doi.org/10.1002%2Fajmg.a.36393>
7. Anitha, A., Nakamura, K., Yamada, K., Suda, S., Thanseem, I., Tsujii, M., Iwayama, Y., Hattori, E., Toyota, T., Miyachi, T., Iwata, Y., Suzuki, K., Matsuzaki, H., Kawai, M., Sekine, Y., Tsuchiya, K., ichi Sugihara, G., Ouchi, Y., Sugiyama, T., Koizumi, K., Higashida, H., Takei, N., Yoshikawa, T., Mori, N.: Genetic analyses ofRoundabout(ROBO) axon guidance receptors in autism. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **147B**(7), 1019–1027 (2008). DOI 10.1002/ajmg.b.30697. URL <https://doi.org/10.1002%2Fajmg.b.30697>
8. Arking, D.E., Cutler, D.J., Brune, C.W., Teslovich, T.M., West, K., Ikeda, M., Rea, A., Guy, M., Lin, S., Cook, E.H., Chakravarti, A.: A common genetic variant in the neurexin superfamily member CNTNAP2 increases familial risk of autism. *The American Journal of Human Genetics* **82**(1), 160–164 (2008). DOI 10.1016/j.ajhg.2007.09.015. URL <https://doi.org/10.1016%2Fj.ajhg.2007.09.015>
9. Atagn, M., kolu, E., Can, S., Karaka-Uurlu, G., Ulusoy-Kaymak, S., aykyl, A., Algn, O., Phillips, M., Moore, C., ngr, D.: Investigation of heschl's gyrus and planum temporale in patients with schizophrenia and bipolar disorder: A proton magnetic resonance spectroscopy study. *chizophrenia Research* **161**(2), 202–209 (2015). DOI 10.1016/j.schres.2014.11.012. URL <https://doi.org/10.1016/j.schres.2014.11.012>
10. Bakkaloglu, B., O'Roak, B.J., Louvi, A., Gupta, A.R., Abelson, J.F., Morgan, T.M., Chawarska, K., Klin, A., Ercan-Sencicek, A.G., Stillman, A.A., Tanriover, G., Abrahams, B.S., Duvall, J.A., Robbins, E.M., Geschwind, D.H., Biederer, T., Gunel, M., Lifton, R.P., State, M.W.: Molecular cytogenetic analysis and resequencing of contactin associated protein-like 2 in autism spectrum disorders. *The American Journal of Human Genetics* **82**(1), 165–173 (2008). DOI 10.1016/j.ajhg.2007.09.017. URL <https://doi.org/10.1016%2Fj.ajhg.2007.09.017>
11. Barnby, G., Abbott, A., Sykes, N., Morris, A., Weeks, D.E., Mott, R., Lamb, J., Bailey, A.J., Monaco, A.P.: Candidate-gene screening and association analysis at the autism-susceptibility locus on chromosome 16p: Evidence of association at GRIN2a and ABAT.

- The American Journal of Human Genetics **76**(6), 950–966 (2005). DOI 10.1086/430454. URL <https://doi.org/10.1086%2F430454>
12. de la Barra M, F., C, V.S., R, A.A., B, E.R., M, F.C., S, Y.L.: Gemelas con autismo y retardo mental asociado a translocación cromosómica balanceada (7;20). Revista chilena de pediatría **57**(6) (1986). DOI 10.4067/s0370-41061986000600016. URL <https://doi.org/10.4067%2Fs0370-41061986000600016>
  13. Bartczak, M., Bokus, B.: Semantic distances in depression: Relations between ME and PAST, FUTURE, JOY, SADNESS, HAPPINESS. Journal of Psycholinguistic Research **46**(2), 345–366 (2016). DOI 10.1007/s10936-016-9442-2. URL <https://doi.org/10.1007%2Fs10936-016-9442-2>
  14. Bates, T.C., Luciano, M., Medland, S.E., Montgomery, G.W., Wright, M.J., Martin, N.G.: Genetic variance in a component of the language acquisition device: ROBO1 polymorphisms associated with phonological buffer deficits. Behavior Genetics **41**(1), 50–57 (2010). DOI 10.1007/s10519-010-9402-9. URL <https://doi.org/10.1007%2Fs10519-010-9402-9>
  15. Bearden, C.E., Wu, K.N., Caplan, R., Cannon, T.D.: Thought disorder and communication deviance as predictors of outcome in youth at clinical high risk for psychosis. Journal of the American Academy of Child & Adolescent Psychiatry **50**(7), 669–680 (2011). DOI 10.1016/j.jaac.2011.03.021. URL <https://doi.org/10.1016%2Fj.jaac.2011.03.021>
  16. Beitchman, J.H., Brownlie, E.B., Inglis, A., Wild, J., Ferguson, B., Schachter, D., Lancee, W., Wilson, B., Mathews, R.: Seven-year follow-up of speech/language impaired and control children: Psychiatric outcome. Journal of Child Psychology and Psychiatry **37**(8), 961–970 (1996). DOI 10.1111/j.1469-7610.1996.tb01493.x. URL <https://doi.org/10.1111%2Fj.1469-7610.1996.tb01493.x>
  17. Bennabi, D., Vandell, P., Papaxanthis, C., Pozzo, T., Haffen, E.: Psychomotor retardation in depression: A systematic review of diagnostic, pathophysiologic, and therapeutic implications. BioMed Research International **2013**, 1–18 (2013). DOI 10.1155/2013/158746. URL <https://doi.org/10.1155%2F2013%2F158746>
  18. Besche-Richard, C.: Lexical decision tasks in depressive patients: semantic priming before and after clinical improvement. European Psychiatry **17**(2), 69–74 (2002). DOI 10.1016/s0924-9338(02)00630-2. URL <https://doi.org/10.1016%2Fs0924-9338%2802%2900630-2>
  19. Beunders, G., Voorhoeve, E., Golzio, C., Pardo, L.M., Rosenfeld, J.A., Talkowski, M.E., Simonin, I., Lionel, A.C., Vergult, S., Pyatt, R.E., van de Kamp, J., Nieuwint, A., Weiss, M.M., Rizzu, P., Verwer, L.E., van Spaendonk, R.M., Shen, Y., Lin Wu, B., Yu, T., Yu, Y., Chiang, C., Gusella, J.F., Lindgren, A.M., Morton, C.C., van Binsbergen, E., Bulk, S., van Rossem, E., Vanakker, O., Armstrong, R., Park, S.M., Greenhalgh, L., Maye, U., Neill, N.J., Abbott, K.M., Sell, S., Ladda, R., Farber, D.M., Bader, P.I., Cushing, T., Drautz, J.M., Konczal, L., Nash, P., de Los Reyes, E., Carter, M.T., Hopkins, E., Marshall, C.R., Osborne, L.R., Gripp, K.W., Thrush, D.L., Hashimoto, S., Gastier-Foster, J.M., Astbury, C., Ylstra, B., Meijers-Heijboer, H., Posthuma, D., Menten, B., Mortier, G., Scherer, S.W., Eichler, E.E., Girirajan, S., Katsanis, N., Groffen, A.J., Sistermans, E.A.: Exonic deletions in AUTS2 cause a syndromic form of intellectual disability and suggest a critical role for the c terminus. The American Journal of Human Genetics **92**(2), 210–220 (2013). DOI 10.1016/j.ajhg.2012.12.011. URL <https://doi.org/10.1016%2Fj.ajhg.2012.12.011>
  20. Binder, J.R., Frost, J.A., Hammeke, T.A., Cox, R.W., Rao, S.M., Prieto, T.: Human brain language areas identified by functional magnetic resonance imaging. The Journal of Neuroscience **17**(1), 353–362 (1997). DOI 10.1523/jneurosci.17-01-00353.1997. URL <https://doi.org/10.1523%2Fjneurosci.17-01-00353.1997>
  21. Bleuler, E.: Dementia praecox or the group of schizophrenias(1908). American Journal of Psychiatry **149**(12), 1733–1734 (1992). DOI 10.1176/ajp.149.12.1733. URL <https://doi.org/10.1176%2Fajp.149.12.1733>
  22. Bottema-Beutel, K.: Associations between joint attention and language in autism spectrum disorder and typical development: A systematic review and meta-regression analysis. Autism Research **9**(10), 1021–1035 (2016). DOI 10.1002/aur.1624. URL <https://doi.org/10.1002%2Faur.1624>
  23. Bourne, C., Aydemir, ., Balanzá-Martínez, V., Bora, E., Brissos, S., Cavanagh, J.T.O., Clark, L., Cubukcuoglu, Z., Dias, V.V., Dittmann, S., Ferrier, I.N., Fleck, D.E., Frangou, S., Gallagher, P., Jones, L., Kiesepp, T., Martínez-Aran, A., Melle, I., Moore, P.B., Mur, M., Pfennig, A., Raust, A., Senturk, V., Simonsen, C., Smith, D.J., Bio, D.S., de Souza, M.G.S., Stoddart, S.D.R., Sundet, K., Szke, A., Thompson, J.M., Torrent, C.,

- Zalla, T., Craddock, N., Andreassen, O.A., Leboyer, M., Vieta, E., Bauer, M., Worhunsky, P.D., Tzagarakis, C., Rogers, R.D., Geddes, J.R., Goodwin, G.M.: Neuropsychological testing of cognitive impairment in euthymic bipolar disorder: an individual patient data meta-analysis. *Acta Psychiatrica Scandinavica* **128**(3), 149–162 (2013). DOI 10.1111/acps.12133. URL <https://doi.org/10.1111%2Facps.12133>
24. Bray, N.J., Buckland, P.R., Williams, N.M., Williams, H.J., Norton, N., Owen, M.J., O'Donovan, M.C.: A haplotype implicated in schizophrenia susceptibility is associated with reduced COMT expression in human brain. *The American Journal of Human Genetics* **73**(1), 152–161 (2003). DOI 10.1086/376578. URL <https://doi.org/10.1086%2F376578>
  25. Brzustowicz, L.M., Simone, J., Mohseni, P., Hayter, J.E., Hodgkinson, K.A., Chow, E.W., Bassett, A.S.: Linkage disequilibrium mapping of schizophrenia susceptibility to the CAPON region of chromosome 1q22. *The American Journal of Human Genetics* **74**(5), 1057–1063 (2004). DOI 10.1086/420774. URL <https://doi.org/10.1086%2F420774>
  26. Bucci, W., Freedman, N.: The language of depression. *Bulletin of the Menninger Clinic* **45**(4), 334 (1981). URL <https://search-proquest-com.proxy.lib.uiowa.edu/docview/1298126062?accountid=14663>. Last updated - 2013-02-23
  27. Buyukdura, J.S., McClintock, S.M., Croarkin, P.E.: Psychomotor retardation in depression: Biological underpinnings, measurement, and treatment. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* **35**(2), 395–409 (2011). DOI 10.1016/j.pnpbp.2010.10.019. URL <https://doi.org/10.1016%2Fj.pnpbp.2010.10.019>
  28. C. Sobin, H.S.: Psychomotor symptoms of depression. *American Journal of Psychiatry* **154**(1), 4–17 (1997). DOI 10.1176/ajp.154.1.4. URL <https://doi.org/10.1176%2Fajp.154.1.4>
  29. Casamassima, F., Huang, J., Fava, M., Sachs, G.S., Smoller, J.W., Cassano, G.B., Lattanzi, L., Fagerness, J., Stange, J.P., Perlis, R.H.: Phenotypic effects of a bipolar liability gene among individuals with major depressive disorder. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **9999B**, n/a–n/a (2009). DOI 10.1002/ajmg.b.30962. URL <https://doi.org/10.1002%2Fajmg.b.30962>
  30. Chang, J.S., Choi, S., Ha, K., Ha, T.H., Cho, H.S., Choi, J.E., Cha, B., Moon, E.: Differential pattern of semantic memory organization between bipolar I and II disorders. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* **35**(4), 1053–1058 (2011). DOI 10.1016/j.pnpbp.2011.02.020. URL <https://doi.org/10.1016%2Fj.pnpbp.2011.02.020>
  31. Charney, A.W., Ruderfer, D.M., Stahl, E.A., Moran, J.L., Chambert, K., Belliveau, R.A., Forty, L., Gordon-Smith, K., Florio, A.D., Lee, P.H., Bromet, E.J., Buckley, P.F., Escamilla, M.A., Fanous, A.H., Fochtmann, L.J., Lehrer, D.S., Malaspina, D., Marder, S.R., Morley, C.P., Nicolini, H., Perkins, D.O., Rakofsky, J.J., Rapaport, M.H., Medeiros, H., Sobell, J.L., Green, E.K., Backlund, L., Bergen, S.E., Juréus, A., Schalling, M., Lichtenstein, P., Roussos, P., Knowles, J.A., Jones, I., Jones, L.A., Hultman, C.M., Perlis, R.H., Purcell, S.M., McCarroll, S.A., Pato, C.N., Pato, M.T., Craddock, N., Landén, M., Smoller, J.W., Sklar, P.: Evidence for genetic heterogeneity between clinical subtypes of bipolar disorder. *Translational Psychiatry* **7**(1), e993–e993 (2017). DOI 10.1038/tp.2016.242. URL <https://doi.org/10.1038%2Ftp.2016.242>
  32. Cheah, S.Y., Lawford, B.R., Young, R.M., Morris, C.P., Voisey, J.: Association of NOS1ap variants and depression phenotypes in schizophrenia. *Journal of Affective Disorders* **188**, 263–269 (2015). DOI 10.1016/j.jad.2015.08.069. URL <https://doi.org/10.1016%2Fj.jad.2015.08.069>
  33. Chen, X.S., Reader, R.H., Hoischen, A., Veltman, J.A., Simpson, N.H., Francks, C., Newbury, D.F., Fisher, S.E.: Next-generation DNA sequencing identifies novel gene variants and pathways involved in specific language impairment. *Scientific Reports* **7**(1) (2017). DOI 10.1038/srep46105. URL <https://doi.org/10.1038%2Fsrep46105>
  34. Cheng, W., Rolls, E.T., Qiu, J., Liu, W., Tang, Y., Huang, C.C., Wang, X., Zhang, J., Lin, W., Zheng, L., Pu, J., Tsai, S.J., Yang, A.C., Lin, C.P., Wang, F., Xie, P., Feng, J.: Medial reward and lateral non-reward orbitofrontal cortex circuits change in opposite directions in depression. *Brain* **139**(12), 3296–3309 (2016). DOI 10.1093/brain/aww255. URL <https://doi.org/10.1093%2Fbrain%2Faww255>
  35. Coe, B.P., Witherspoon, K., Rosenfeld, J.A., van Bon, B.W.M., van Silfhout, A.T.V., Bosco, P., Friend, K.L., Baker, C., Buono, S., Vissers, L.E.L.M., Schuurs-Hoeijmakers, J.H., Hoischen, A., Pfundt, R., Krumm, N., Carvill, G.L., Li, D., Amaral, D., Brown, N., Lockhart, P.J., Scheffer, I.E., Alberti, A., Shaw, M., Pettinato, R., Tervo, R., de Leeuw, N., Reijnders, M.R.F., Torchia, B.S., Peeters, H., Thompson, E., O'Roak, B.J., Fichera,

- M., Hehir-Kwa, J.Y., Shendure, J., Mefford, H.C., Haan, E., Géczy, J., de Vries, B.B.A., Romano, C., Eichler, E.E.: Refining analyses of copy number variation identifies specific genes associated with developmental delay. *Nature Genetics* **46**(10), 1063–1071 (2014). DOI 10.1038/ng.3092. URL <https://doi.org/10.1038%2Fng.3092>
36. Comings, D.E., Gade-Andavolu, R., Gonzalez, N., Wu, S., Muhleman, D., Blake, H., Chiu, F., Wang, E., Farwell, K., Darakjy, S., Baker, R., Dietz, G., Saucier, G., MacMurray, J.P.: Multivariate analysis of associations of 42 genes in ADHD, ODD and conduct disorder. *Clinical Genetics* **58**(1), 31–40 (2001). DOI 10.1034/j.1399-0004.2000.580106.x. URL <https://doi.org/10.1034%2Fj.1399-0004.2000.580106.x>
  37. Condray, R., van Kammen, D.P., Steinhauer, S.R., Kasperek, A., Yao, J.K.: Language comprehension in schizophrenia: Trait or state indicator? *Biological Psychiatry* **38**(5), 287–296 (1995). DOI 10.1016/0006-3223(95)00378-t. URL [https://doi.org/10.1016%2F0006-3223\(95\)00378-t](https://doi.org/10.1016%2F0006-3223(95)00378-t)
  38. Consortium, W.T.C.C.: Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. *Nature* **447**(7145), 661–678 (2007). DOI 10.1038/nature05911. URL <https://doi.org/10.1038%2Fnature05911>
  39. Cope, N., Harold, D., Hill, G., Moskvina, V., Stevenson, J., Holmans, P., Owen, M.J., O'Donovan, M.C., Williams, J.: Strong evidence that KIAA0319 on chromosome 6p is a susceptibility gene for developmental dyslexia. *The American Journal of Human Genetics* **76**(4), 581–591 (2005). DOI 10.1086/429131. URL <https://doi.org/10.1086%2F429131>
  40. Corcoran, C.M., Carrillo, F., Fernández-Slezak, D., Bedi, G., Klim, C., Javitt, D.C., Bearden, C.E., Cecchi, G.A.: Prediction of psychosis across protocols and risk cohorts using automated language analysis. *World Psychiatry* **17**(1), 67–75 (2018). DOI 10.1002/wps.20491. URL <https://doi.org/10.1002%2Fwps.20491>
  41. Costafreda, S.G., Fu, C.H., Picchioni, M., Touloupoulou, T., McDonald, C., Kravartiti, E., Walshe, M., Prata, D., Murray, R.M., McGuire, P.K.: Pattern of neural responses to verbal fluency shows diagnostic specificity for schizophrenia and bipolar disorder. *BMC Psychiatry* **11**(1) (2011). DOI 10.1186/1471-244x-11-18. URL <https://doi.org/10.1186%2F1471-244x-11-18>
  42. Couto, J.M., Gomez, L., Wigg, K., Ickowicz, A., Pathare, T., Malone, M., Kennedy, J.L., Schachar, R., Barr, C.L.: Association of attention-deficit/hyperactivity disorder with a candidate region for reading disabilities on chromosome 6p. *Biological Psychiatry* **66**(4), 368–375 (2009). DOI 10.1016/j.biopsych.2009.02.016. URL <https://doi.org/10.1016%2Fj.biopsych.2009.02.016>
  43. Demontis, D., Walters, R.K., Martin, J., Mattheisen, M., Als, T.D., Agerbo, E., Baldursson, G., Belliveau, R., Bybjerg-Grauholm, J., Bækvad-Hansen, M., Cerrato, F., Chambert, K., Churchhouse, C., Dumont, A., Eriksson, N., Gandal, M., Goldstein, J.I., Grasby, K.L., Grove, J., Gudmundsson, O.O., Hansen, C.S., Hauberg, M.E., Hollegaard, M.V., Howrigan, D.P., Huang, H., Maller, J.B., Martin, A.R., Martin, N.G., Moran, J., Pallesen, J., Palmer, D.S., Pedersen, C.B., Pedersen, M.G., Poterba, T., Poulsen, J.B., Ripke, S., Robinson, E.B., Satterstrom, F.K., Stefansson, H., Stevens, C., Turley, P., Walters, G.B., Won, H., Wright, M.J., Andreassen, O.A., Asherson, P., Burton, C.L., Boomsma, D.I., Cormand, B., Dalsgaard, S., Franke, B., Gelernter, J., Geschwind, D., Hakonarson, H., Haavik, J., Kranzler, H.R., Kuntsi, J., Langley, K., Lesch, K.P., Middeldorp, C., Reif, A., Rohde, L.A., Roussos, P., Schachar, R., Sklar, P., Sonuga-Barke, E.J.S., Sullivan, P.F., Thapar, A., Tung, J.Y., Waldman, I.D., Medland, S.E., Stefansson, K., Nordentoft, M., Hougaard, D.M., Werge, T., Mors, O., Mortensen, P.B., Daly, M.J., Faraone, S.V., Børghlum, A.D., Neale, B.M., and: Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature Genetics* **51**(1), 63–75 (2018). DOI 10.1038/s41588-018-0269-7. URL <https://doi.org/10.1038%2Fs41588-018-0269-7>
  44. Deriziotis, P., Fisher, S.E.: Speech and language: Translating the genome. *Trends in Genetics* **33**(9), 642–656 (2017). DOI 10.1016/j.tig.2017.07.002. URL <https://doi.org/10.1016%2Fj.tig.2017.07.002>
  45. Doi, H., Fujisawa, T.X., Iwanaga, R., Matsuzaki, J., Kawasaki, C., Tochigi, M., Sasaki, T., Kato, N., Shinohara, K.: Association between single nucleotide polymorphisms in estrogen receptor 1/2 genes and symptomatic severity of autism spectrum disorder. *Research in Developmental Disabilities* **82**, 20–26 (2018). DOI 10.1016/j.ridd.2018.02.014. URL <https://doi.org/10.1016%2Fj.ridd.2018.02.014>
  46. Dorval, K.M., Wigg, K.G., Crosbie, J., Tannock, R., Kennedy, J.L., Ickowicz, A., Pathare, T., Malone, M., Schachar, R., Barr, C.L.: Association of the glutamate receptor subunit gene GRIN2b with attention-deficit/hyperactivity disorder. *Genes, Brain*



- and Behavior **6**(5), 444–452 (2007). DOI 10.1111/j.1601-183x.2006.00273.x. URL <https://doi.org/10.1111%2Fj.1601-183x.2006.00273.x>
47. Drevets, W., Videen, T., Price, J., Preskorn, S., Carmichael, S., Raichle, M.: A functional anatomical study of unipolar depression. *The Journal of Neuroscience* **12**(9), 3628–3641 (1992). DOI 10.1523/jneurosci.12-09-03628.1992. URL <https://doi.org/10.1523%2Fjneurosci.12-09-03628.1992>
  48. Eicher, J.D., Gruen, J.R.: Language impairment and dyslexia genes influence language skills in children with autism spectrum disorders. *Autism Research* **8**(2), 229–234 (2014). DOI 10.1002/aur.1436. URL <https://doi.org/10.1002%2Faur.1436>
  49. Eicher, J.D., Powers, N.R., Miller, L.L., Akshoomoff, N., Amaral, D.G., Bloss, C.S., Libiger, O., Schork, N.J., Darst, B.F., Casey, B.J., Chang, L., Ernst, T., Frazier, J., Kaufmann, W.E., Keating, B., Kenet, T., Kennedy, D., Mostofsky, S., Murray, S.S., Sowell, E.R., Bartsch, H., Kuperman, J.M., Brown, T.T., Hagler, D.J., Dale, A.M., Jernigan, T.L., Pourcain, B.S., Smith, G.D., Ring, S.M., and, J.R.G.: Genome-wide association study of shared components of reading disability and language impairment. *Genes, Brain and Behavior* **12**(8), 792–801 (2013). DOI 10.1111/gbb.12085. URL <https://doi.org/10.1111%2Fgbb.12085>
  50. Elia, J., Gai, X., Xie, H.M., Perin, J.C., Geiger, E., Glessner, J.T., D'arcy, M., deBerardinis, R., Frackelton, E., Kim, C., Lantieri, F., Muganga, B.M., Wang, L., Takeda, T., Rappaport, E.F., Grant, S.F.A., Berrettini, W., Devoto, M., Shaikh, T.H., Hakonarson, H., White, P.S.: Rare structural variants found in attention-deficit hyperactivity disorder are preferentially associated with neurodevelopmental genes. *Molecular Psychiatry* **15**(6), 637–646 (2009). DOI 10.1038/mp.2009.57. URL <https://doi.org/10.1038%2Fmp.2009.57>
  51. Ercan-Sencicek, A.G., Wright, N.R.D., Sanders, S.J., Oakman, N., Valdes, L., Bakkaloglu, B., Doyle, N., Yrigollen, C.M., Morgan, T.M., Grigorenko, E.L.: A balanced t(10;15) translocation in a male patient with developmental language disorder. *European Journal of Medical Genetics* **55**(2), 128–131 (2012). DOI 10.1016/j.ejmg.2011.12.005. URL <https://doi.org/10.1016%2Fj.ejmg.2011.12.005>
  52. Fabbri, C., Serretti, A.: Genetics of long-term treatment outcome in bipolar disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* **65**, 17–24 (2016). DOI 10.1016/j.pnpbp.2015.08.008. URL <https://doi.org/10.1016%2Fj.pnpbp.2015.08.008>
  53. Fallin, M.D., Lasseret, V.K., Avramopoulos, D., Nicodemus, K.K., Wolyniec, P.S., McGrath, J.A., Steel, G., Nestadt, G., Liang, K.Y., Haganir, R.L., Valle, D., Pulver, A.E.: Bipolar i disorder and schizophrenia: A 440–single-nucleotide polymorphism screen of 64 candidate genes among ashkenazi jewish case-parent trios. *The American Journal of Human Genetics* **77**(6), 918–936 (2005). DOI 10.1086/497703. URL <https://doi.org/10.1086%2F497703>
  54. Fanous, A.H., Zhou, B., Aggen, S.H., Bergen, S.E., Amdur, R.L., Duan, J., Sanders, A.R., Shi, J., Mowry, B.J., Olincy, A., Amin, F., Cloninger, C.R., Silverman, J.M., Buccola, N.G., Byerley, W.F., Black, D.W., Freedman, R., Dudbridge, F., Holmans, P.A., Ripke, S., Gejman, P.V., Kendler, K.S., and, D.F.L.: Genome-wide association study of clinical dimensions of schizophrenia: Polygenic effect on disorganized symptoms. *American Journal of Psychiatry* **169**(12), 1309–1317 (2012). DOI 10.1176/appi.ajp.2012.12020218. URL <https://doi.org/10.1176%2Fappi.ajp.2012.12020218>
  55. Ferreira, M.A.R., , O'Donovan, M.C., Meng, Y.A., Jones, I.R., Ruderfer, D.M., Jones, L., Fan, J., Kirov, G., Perlis, R.H., Green, E.K., Smoller, J.W., Grozeva, D., Stone, J., Nikolov, I., Chambert, K., Hamshere, M.L., Nimgaonkar, V.L., Moskvina, V., Thase, M.E., Caesar, S., Sachs, G.S., Franklin, J., Gordon-Smith, K., Ardlie, K.G., Gabriel, S.B., Fraser, C., Blumenstiel, B., Defelice, M., Breen, G., Gill, M., Morris, D.W., Elkin, A., Muir, W.J., McGhee, K.A., Williamson, R., MacIntyre, D.J., MacLean, A.W., Clair, D.S., Robinson, M., Beck, M.V., Pereira, A.C.P., Kandaswamy, R., McQuillin, A., Collier, D.A., Bass, N.J., Young, A.H., Lawrence, J., Ferrier, I.N., Anjorin, A., Farmer, A., Curtis, D., Scolnick, E.M., McGuffin, P., Daly, M.J., Corvin, A.P., Holmans, P.A., Blackwood, D.H., Gurling, H.M., Owen, M.J., Purcell, S.M., Sklar, P., Craddock, N.: Collaborative genome-wide association analysis supports a role for ANK3 and CACNA1c in bipolar disorder. *Nature Genetics* **40**(9), 1056–1058 (2008). DOI 10.1038/ng.209. URL <https://doi.org/10.1038%2Fng.209>
  56. Feuk, L., Kalervo, A., Lipsanen-Nyman, M., Skaug, J., Nakabayashi, K., Finucane, B., Hartung, D., Innes, M., Kerem, B., Nowaczyk, M.J., Rivlin, J., Roberts, W., Senman, L., Summers, A., Szatmari, P., Wong, V., Vincent, J.B., Zeeman, S., Osborne, L.R., Cardy, J.O., Kere, J., Scherer, S.W., Hannula-Jouppi, K.: Absence of a paternally inherited

- FOXP2 gene in developmental verbal dyspraxia. *The American Journal of Human Genetics* **79**(5), 965–972 (2006). DOI 10.1086/508902. URL <https://doi.org/10.1086/508902>
57. Filges, I., Shimojima, K., Okamoto, N., Rothlisberger, B., Weber, P., Huber, A.R., Nishizawa, T., Datta, A.N., Miny, P., Yamamoto, T.: Reduced expression by SETBP1 haploinsufficiency causes developmental and expressive language delay indicating a phenotype distinct from schinzel-giedion syndrome. *Journal of Medical Genetics* **48**(2), 117–122 (2010). DOI 10.1136/jmg.2010.084582. URL <https://doi.org/10.1136/jmg.2010.084582>
  58. Frangou, S.: The maudsley bipolar disorder project. *Epilepsia* **46**, 19–25 (2005). DOI 10.1111/j.1528-1167.2005.463005.x. URL <https://doi.org/10.1111/j.1528-1167.2005.463005.x>
  59. Fusaroli, R., Lambrechts, A., Bang, D., Bowler, D.M., Gaigg, S.B.: “is voice a marker for autism spectrum disorder? a systematic review and meta-analysis”. *Autism Research* **10**(3), 384–407 (2016). DOI 10.1002/aur.1678. URL <https://doi.org/10.1002/aur.1678>
  60. Gehricke, J.G., Kruggel, F., Thampipop, T., Alejo, S.D., Tatos, E., Fallon, J., Muf-tuler, L.T.: The brain anatomy of attention-deficit/hyperactivity disorder in young adults – a magnetic resonance imaging study. *PLOS ONE* **12**(4), e0175433 (2017). DOI 10.1371/journal.pone.0175433. URL <https://doi.org/10.1371/journal.pone.0175433>
  61. Georgieff, N., Dominey, P.F., Michel, F., Marie-cardine, M., Dalery, J.: Semantic priming in major depressive state. *Psychiatry Research* **78**(1-2), 29–44 (1998). DOI 10.1016/S0165-1781(97)00155-8. URL [https://doi.org/10.1016/S0165-1781\(97\)00155-8](https://doi.org/10.1016/S0165-1781(97)00155-8)
  62. Glatt, S.J., Stone, W.S., Nossova, N., Liew, C.C., Seidman, L.J., Tsuang, M.T.: Similarities and differences in peripheral blood gene-expression signatures of individuals with schizophrenia and their first-degree biological relatives. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **156**(8), 869–887 (2011). DOI 10.1002/ajmg.b.31239. URL <https://doi.org/10.1002/ajmg.b.31239>
  63. Goes, F.S., McGrath, J., Avramopoulos, D., Wolyniec, P., Pirooznia, M., Ruczinski, I., Nestadt, G., Kenny, E.E., Vacic, V., Peters, I., Lencz, T., Darvasi, A., Mülle, J.G., Warren, S.T., Pulver, A.E.: Genome-wide association study of schizophrenia in ashkenazi jews. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **168**(8), 649–659 (2015). DOI 10.1002/ajmg.b.32349. URL <https://doi.org/10.1002/ajmg.b.32349>
  64. Grabenhorst, F., Rolls, E.T.: Value, pleasure and choice in the ventral prefrontal cortex. *Trends in Cognitive Sciences* **15**(2), 56–67 (2011). DOI 10.1016/j.tics.2010.12.004. URL <https://doi.org/10.1016/j.tics.2010.12.004>
  65. Gratacòs, M., Costas, J., de Cid, R., Bayés, M., González, J.R., Baca-García, E., de Diego, Y., Fernández-Aranda, F., Fernández-Piqueras, J., Guitart, M., Martín-Santos, R., Martorell, L., Menchón, J.M., Roca, M., Sáiz-Ruiz, J., Sanjuán, J., Torrens, M., Urretavizcaya, M., Valero, J., Vilella, E., Estivill, X., and, Á.C.: Identification of new putative susceptibility genes for several psychiatric disorders by association analysis of regulatory and non-synonymous SNPs of 306 genes involved in neurotransmission and neurodevelopment. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **150B**(6), 808–816 (2009). DOI 10.1002/ajmg.b.30902. URL <https://doi.org/10.1002/ajmg.b.30902>
  66. Green, E.K., Grozeva, D., Jones, I., Jones, L., Kirov, G., Caesar, S., Gordon-Smith, K., Fraser, C., Forty, L., Russell, E., Hamshere, M.L., Moskvina, V., Nikolov, I., Farmer, A., McGuffin, P., Holmans, P.A., Owen, M.J., O'Donovan, M.C., Craddock, N.: The bipolar disorder risk allele at CACNA1c also confers risk of recurrent major depression and of schizophrenia. *Molecular Psychiatry* **15**(10), 1016–1022 (2009). DOI 10.1038/mp.2009.49. URL <https://doi.org/10.1038/mp.2009.49>
  67. Greenwood, T.A., Akiskal, H.S., Akiskal, K.K., Kelsoe, J.R.: Genome-wide association study of temperament in bipolar disorder reveals significant associations with three novel loci. *Biological Psychiatry* **72**(4), 303–310 (2012). DOI 10.1016/j.biopsych.2012.01.018. URL <https://doi.org/10.1016/j.biopsych.2012.01.018>
  68. Group, P.G.C.B.D.W.: Large-scale genome-wide association analysis of bipolar disorder identifies a new susceptibility locus near ODZ4. *Nature Genetics* **43**(10), 977–983 (2011). DOI 10.1038/ng.943. URL <https://doi.org/10.1038/ng.943>
  69. Guo, W., Machado-Vieira, R., Mathew, S., Murrough, J.W., Charney, D.S., Grunebaum, M., Oquendo, M.A., Kadriu, B., Akula, N., Henter, I., Yuan, P., Merikangas, K., Drevets, W., Furey, M., Mann, J.J., McMahon, F.J., Zarate, C.A., Shugart, Y.Y.: Exploratory genome-wide association analysis of response to ketamine and a polygenic analysis of

- response to scopolamine in depression. *Translational Psychiatry* **8**(1) (2018). DOI 10.1038/s41398-018-0311-7. URL <https://doi.org/10.1038/s41398-018-0311-7>
70. Hadzimichalis, N.M., Previtiera, M.L., Moreau, M.P., Li, B., Lee, G.H., Dulencin, A.M., Matteson, P.G., Buyske, S., Millonig, J.H., Brzustowicz, L.M., Firestein, B.L.: NOS1ap protein levels are altered in BA46 and cerebellum of patients with schizophrenia. *Schizophrenia Research* **124**(1-3), 248–250 (2010). DOI 10.1016/j.schres.2010.05.009. URL <https://doi.org/10.1016/j.schres.2010.05.009>
  71. Hall, L.S., , Adams, M.J., Arnau-Soler, A., Clarke, T.K., Howard, D.M., Zeng, Y., Davies, G., Hagenaaars, S.P., Fernandez-Pujals, A.M., Gibson, J., Wigmore, E.M., Boutin, T.S., Hayward, C., Scotland, G., Porteous, D.J., Deary, I.J., Thomson, P.A., Haley, C.S., McIntosh, A.M.: Genome-wide meta-analyses of stratified depression in generation scotland and UK biobank. *Translational Psychiatry* **8**(1) (2018). DOI 10.1038/s41398-017-0034-1. URL <https://doi.org/10.1038/s41398-017-0034-1>
  72. Hamdan, F.F., Daoud, H., Rochefort, D., Piton, A., Gauthier, J., Langlois, M., Foomani, G., Dobrzyniecka, S., Krebs, M.O., Joobar, R., Lafrenière, R.G., Lacaille, J.C., Mottron, L., Drapeau, P., Beauchamp, M.H., Phillips, M.S., Fombonne, E., Rouleau, G.A., Michaud, J.L.: De novo mutations in FOXP1 in cases with intellectual disability, autism, and language impairment. *The American Journal of Human Genetics* **87**(5), 671–678 (2010). DOI 10.1016/j.ajhg.2010.09.017. URL <https://doi.org/10.1016/j.ajhg.2010.09.017>
  73. Hampton, L.H., Kaiser, A.P.: Intervention effects on spoken-language outcomes for children with autism: a systematic review and meta-analysis. *Journal of Intellectual Disability Research* **60**(5), 444–463 (2016). DOI 10.1111/jir.12283. URL <https://doi.org/10.1111/jir.12283>
  74. Hass, J., Walton, E., Wright, C., Beyer, A., Scholz, M., Turner, J., Liu, J., Smolka, M.N., Roessner, V., Sponheim, S.R., Gollub, R.L., Calhoun, V.D., Ehrlich, S.: Associations between DNA methylation and schizophrenia-related intermediate phenotypes — a gene set enrichment analysis. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* **59**, 31–39 (2015). DOI 10.1016/j.pnpbp.2015.01.006. URL <https://doi.org/10.1016/j.pnpbp.2015.01.006>
  75. Ho, K.W.D., Han, S., Nielsen, J.V., Jancic, D., Hing, B., Fiedorowicz, J., Weissman, M.M., Levinson, D.F., Potash, J.B.: Genome-wide association study of seasonal affective disorder. *Translational Psychiatry* **8**(1) (2018). DOI 10.1038/s41398-018-0246-z. URL <https://doi.org/10.1038/s41398-018-0246-z>
  76. Hoogman, M., Muetzel, R., Guimaraes, J.P., Shumskaya, E., Mennes, M., Zwiers, M.P., Jahanshad, N., Sudre, G., Wolfers, T., Earl, E.A., Vila, J.C.S., Vives-Gilabert, Y., Khadka, S., Novotny, S.E., Hartman, C.A., Heslenfeld, D.J., Schweren, L.J., Ambrosino, S., Oranje, B., de Zeeuw, P., Chaim-Avincini, T.M., Rosa, P.G., Zanetti, M.V., Malpas, C.B., Kohls, G., von Polier, G.G., Seitz, J., Biederman, J., Doyle, A.E., Dale, A.M., van Erp, T.G., Epstein, J.N., Jernigan, T.L., Baur-Streubel, R., Ziegler, G.C., Zierhut, K.C., Schranter, A., Høvik, M.F., Lundervold, A.J., Kelly, C., McCarthy, H., Skokauskas, N., Tuura, R.L.O., Calvo, A., Lera-Miguel, S., Nicolau, R., Chantiluke, K.C., Christakou, A., Vance, A., Cercignani, M., Gabel, M.C., Asherson, P., Baumeister, S., Brandeis, D., Hohmann, S., Bramati, I.E., Tovar-Moll, F., Fallgatter, A.J., Kardatzki, B., Schwarz, L., Anikin, A., Baranov, A., Gogberashvili, T., Kapilushniy, D., Solovieva, A., Marroun, H.E., White, T., Karkashadze, G., Namazova-Baranova, L., Ethofer, T., Mattos, P., Banaschewski, T., Coghill, D., Plessen, K.J., Kuntsi, J., Mehta, M.A., Paloyelis, Y., Harrison, N.A., Bellgrove, M.A., Silk, T.J., Cubillo, A.I., Rubia, K., Lazaro, L., Brem, S., Walitza, S., Frodl, T., Zentis, M., Castellanos, F.X., Yoncheva, Y.N., Haavik, J., Reneman, L., Conzelmann, A., Lesch, K.P., Pauli, P., Reif, A., Tamm, L., Konrad, K., Weiss, E.O., Busatto, G.F., Louza, M.R., Durston, S., Hoekstra, P.J., Oosterlaan, J., Stevens, M.C., Ramos-Quiroga, J.A., Vilarroya, O., Fair, D.A., Nigg, J.T., Thompson, P.M., Buitelaar, J.K., Faraone, S.V., Shaw, P., Tiemeier, H., Bralten, J., Franke, B.: Brain imaging of the cortex in ADHD: A coordinated analysis of large-scale clinical and population-based samples. *American Journal of Psychiatry* p. appi.ajp.2019.1 (2019). DOI 10.1176/appi.ajp.2019.18091033. URL <https://doi.org/10.1176/appi.ajp.2019.18091033>
  77. Hoppenbrouwers, M., Vandermosten, M., Boets, B.: Autism as a disconnection syndrome: A qualitative and quantitative review of diffusion tensor imaging studies. *Research in Autism Spectrum Disorders* **8**(4), 387–412 (2014). DOI 10.1016/j.rasd.2013.12.018. URL <https://doi.org/10.1016/j.rasd.2013.12.018>

78. Horn, D., Kapeller, J., Rivera-Brugués, N., Moog, U., Lorenz-Depiereux, B., Eck, S., Hempel, M., Wagenstaller, J., Gawthrop, A., Monaco, A.P., Bonin, M., Riess, O., Wohlleber, E., Illig, T., Bezzina, C.R., Franke, A., Spranger, S., Villavicencio-Lorini, P., Seifert, W., Rosenfeld, J., Klopocki, E., Rappold, G.A., Strom, T.M.: Identification of FOXP1 deletions in three unrelated patients with mental retardation and significant speech and language deficits. *Human Mutation* **31**(11), E1851–E1860 (2010). DOI 10.1002/humu.21362. URL <https://doi.org/10.1002%2Fhumu.21362>
79. Howard, D.M., , Adams, M.J., Clarke, T.K., Hafferty, J.D., Gibson, J., Shirali, M., Coleman, J.R.I., Hagenaars, S.P., Ward, J., Wigmore, E.M., Alloza, C., Shen, X., Barbu, M.C., Xu, E.Y., Whalley, H.C., Marioni, R.E., Porteous, D.J., Davies, G., Deary, I.J., Hemani, G., Berger, K., Teismann, H., Rawal, R., Arolt, V., Baune, B.T., Dannlowski, U., Domschke, K., Tian, C., Hinds, D.A., Trzaskowski, M., Byrne, E.M., Ripke, S., Smith, D.J., Sullivan, P.F., Wray, N.R., Breen, G., Lewis, C.M., and, A.M.M.: Genome-wide meta-analysis of depression identifies 102 independent variants and highlights the importance of the prefrontal brain regions. *Nature Neuroscience* **22**(3), 343–352 (2019). DOI 10.1038/s41593-018-0326-7. URL <https://doi.org/10.1038%2Fs41593-018-0326-7>
80. Howard, D.M., , Adams, M.J., Shirali, M., Clarke, T.K., Marioni, R.E., Davies, G., Coleman, J.R.I., Alloza, C., Shen, X., Barbu, M.C., Wigmore, E.M., Gibson, J., Hagenaars, S.P., Lewis, C.M., Ward, J., Smith, D.J., Sullivan, P.F., Haley, C.S., Breen, G., Deary, I.J., McIntosh, A.M.: Genome-wide association study of depression phenotypes in UK biobank identifies variants in excitatory synaptic pathways. *Nature Communications* **9**(1) (2018). DOI 10.1038/s41467-018-03819-3. URL <https://doi.org/10.1038%2Fs41467-018-03819-3>
81. Hu, J., Bi, Y., Shi, L., Xu, F., Yuan, F., Niu, W., Ren, D., Guo, Z., Yang, F., He, L., He, G.: No association of GRIN2a polymorphisms with the major depressive disorder in the chinese han origin. *Psychiatric Genetics* p. 1 (2018). DOI 10.1097/ypg.0000000000000207. URL <https://doi.org/10.1097%2Fypg.0000000000000207>
82. Ikeda, M., , Takahashi, A., Kamatani, Y., Okahisa, Y., Kunugi, H., Mori, N., Sasaki, T., Ohmori, T., Okamoto, Y., Kawasaki, H., Shimodera, S., Kato, T., Yoneda, H., Yoshimura, R., Iyo, M., Matsuda, K., Akiyama, M., Ashikawa, K., Kashiwase, K., Tokunaga, K., Kondo, K., Saito, T., Shimasaki, A., Kawase, K., Kitajima, T., Matsuo, K., Itokawa, M., Someya, T., Inada, T., Hashimoto, R., Inoue, T., Akiyama, K., Tanii, H., Arai, H., Kanba, S., Ozaki, N., Kusumi, I., Yoshikawa, T., Kubo, M., Iwata, N.: A genome-wide association study identifies two novel susceptibility loci and trans population polygenicity associated with bipolar disorder. *Molecular Psychiatry* **23**(3), 639–647 (2017). DOI 10.1038/mp.2016.259. URL <https://doi.org/10.1038%2Fmp.2016.259>
83. Ikeda, M., Takahashi, A., Kamatani, Y., Momozawa, Y., Saito, T., Kondo, K., Shimasaki, A., Kawase, K., Sakusabe, T., Iwayama, Y., Toyota, T., Wakuda, T., Kikuchi, M., Kanahara, N., Yamamori, H., Yasuda, Y., Watanabe, Y., Hoya, S., Aleksic, B., Kushima, I., Arai, H., Takaki, M., Hattori, K., Kunugi, H., Okahisa, Y., Ohnuma, T., Ozaki, N., Someya, T., Hashimoto, R., Yoshikawa, T., Kubo, M., Iwata, N.: Genome-wide association study detected novel susceptibility genes for schizophrenia and shared trans-populations/diseases genetic effect. *Schizophrenia Bulletin* **45**(4), 824–834 (2018). DOI 10.1093/schbul/sby140. URL <https://doi.org/10.1093%2Fschbul%2Fsby140>
84. Ingason, A., , Giegling, I., Hartmann, A.M., Genius, J., Konte, B., Friedl, M., Ripke, S., Sullivan, P.F., Clair, D.S., Collier, D.A., O'Donovan, M.C., Mirnics, K., Rujescu, D.: Expression analysis in a rat psychosis model identifies novel candidate genes validated in a large case–control sample of schizophrenia. *Translational Psychiatry* **5**(10), e656–e656 (2015). DOI 10.1038/tp.2015.151. URL <https://doi.org/10.1038%2Ftp.2015.151>
85. Itokawa, M., Yamada, K., Yoshitsugu, K., Toyota, T., Suga, T., Ohba, H., Watanabe, A., Hattori, E., Shimizu, H., Kumakura, T., Ebihara, M., Meerabux, J.M., Toru, M., Yoshikawa, T.: A microsatellite repeat in the promoter of the n-methyl-d-aspartate receptor 2a subunit (GRIN2a) gene suppresses transcriptional activity and correlates with chronic outcome in schizophrenia. *Pharmacogenetics* **13**(5), 271–278 (2003). DOI 10.1097/00008571-200305000-00006. URL <https://doi.org/10.1097%2F00008571-200305000-00006>
86. Iwayama-Shigeno, Y., Yamada, K., Itokawa, M., Toyota, T., Meerabux, J.M., Minabe, Y., Mori, N., Inada, T., Yoshikawa, T.: Extended analyses support the association of a functional (GT)<sub>n</sub> polymorphism in the GRIN2a promoter with japanese schizophrenia. *Neuroscience Letters* **378**(2), 102–105 (2005). DOI 10.1016/j.neulet.2004.12.013. URL <https://doi.org/10.1016%2Fj.neulet.2004.12.013>

87. Jamadar, S., Powers, N., Meda, S., Gelernter, J., Gruen, J., Pearlson, G.: Genetic influences of cortical gray matter in language-related regions in healthy controls and schizophrenia. *Schizophrenia Research* **129**(2-3), 141–148 (2011). DOI 10.1016/j.schres.2011.03.027. URL <https://doi.org/10.1016%2Fj.schres.2011.03.027>
88. Ji, W., Li, T., Pan, Y., Tao, H., Ju, K., Wen, Z., Fu, Y., An, Z., Zhao, Q., Wang, T., He, L., Feng, G., Yi, Q., Shi, Y.: CNTNAP2 is significantly associated with schizophrenia and major depression in the han chinese population. *Psychiatry Research* **207**(3), 225–228 (2013). DOI 10.1016/j.psychres.2012.09.024. URL <https://doi.org/10.1016%2Fj.psychres.2012.09.024>
89. Jiang, Y., Zhang, H.: Propensity score-based nonparametric test revealing genetic variants underlying bipolar disorder. *Genetic Epidemiology* **35**(2), 125–132 (2011). DOI 10.1002/gepi.20558. URL <https://doi.org/10.1002%2Fgepi.20558>
90. Johnson, C.J., Beitchman, J.H., Young, A., Escobar, M., Atkinson, L., Wilson, B., Brownlie, E.B., Douglas, L., Taback, N., Lam, I., Wang, M.: Fourteen-year follow-up of children with and without speech/language impairments. *Journal of Speech, Language, and Hearing Research* **42**(3), 744–760 (1999). DOI 10.1044/jslhr.4203.744. URL <https://doi.org/10.1044%2Fjslhr.4203.744>
91. Jones, R.M., Cadby, G., Melton, P.E., Abraham, L.J., Whitehouse, A.J., Moses, E.K.: Genome-wide association study of autistic-like traits in a general population study of young adults. *Frontiers in Human Neuroscience* **7** (2013). DOI 10.3389/fnhum.2013.00658. URL <https://doi.org/10.3389%2Ffnhum.2013.00658>
92. Jou, R.J., Minshew, N.J., Keshavan, M.S., Vitale, M.P., Hardan, A.Y.: Enlarged right superior temporal gyrus in children and adolescents with autism. *Brain Research* **1360**, 205–212 (2010). DOI 10.1016/j.brainres.2010.09.005. URL <https://doi.org/10.1016%2Fj.brainres.2010.09.005>
93. Karolewicz, B., Szebeni, K., Gilmore, T., Maciag, D., Stockmeier, C.A., Ordway, G.A.: Elevated levels of NR2a and PSD-95 in the lateral amygdala in depression. *The International Journal of Neuropsychopharmacology* **12**(02), 143 (2008). DOI 10.1017/s1461145708008985. URL <https://doi.org/10.1017%2Fs1461145708008985>
94. Kendler, K.S.: The clinical features of mania and their representation in modern diagnostic criteria. *Psychological Medicine* **47**(6), 1013–1029 (2016). DOI 10.1017/s0033291716003238. URL <https://doi.org/10.1017%2Fs0033291716003238>
95. Keyes, K., Agnew-Blais, J., Roberts, A.L., Hamilton, A., Vivo, I.D., Ranu, H., Koenen, K.: The role of allelic variation in estrogen receptor genes and major depression in the nurses health study. *Social Psychiatry and Psychiatric Epidemiology* **50**(12), 1893–1904 (2015). DOI 10.1007/s00127-015-1087-1. URL <https://doi.org/10.1007%2Fs00127-015-1087-1>
96. Kim, J.I., Yoo, J.H., Kim, D., Jeong, B., Kim, B.N.: The effects of GRIN2b and DRD4 gene variants on local functional connectivity in attention-deficit/hyperactivity disorder. *Brain Imaging and Behavior* **12**(1), 247–257 (2017). DOI 10.1007/s11682-017-9690-2. URL <https://doi.org/10.1007%2Fs11682-017-9690-2>
97. Kircher, T.T.J., Liddle, P.F., Brammer, M.J., Williams, S.C.R., Murray, R.M., McGuire, P.K.: Neural correlates of formal thought disorder in schizophrenia. *Archives of General Psychiatry* **58**(8), 769 (2001). DOI 10.1001/archpsyc.58.8.769. URL <https://doi.org/10.1001%2Farchpsyc.58.8.769>
98. Klaus, J., Schutter, D.J.: The role of left dorsolateral prefrontal cortex in language processing. *Neuroscience* **377**, 197–205 (2018). DOI 10.1016/j.neuroscience.2018.03.002. URL <https://doi.org/10.1016%2Fj.neuroscience.2018.03.002>
99. Klumpp, H., Deldin, P.: Review of brain functioning in depression for semantic processing and verbal fluency. *International Journal of Psychophysiology* **75**(2), 77–85 (2010). DOI 10.1016/j.ijpsycho.2009.10.003. URL <https://doi.org/10.1016%2Fj.ijpsycho.2009.10.003>
100. Kornilov, S.A., Rakhlin, N., Kuposov, R., Lee, M., Yrigollen, C., Caglayan, A.O., Magnuson, J.S., Mane, S., Chang, J.T., Grigorenko, E.L.: Genome-wide association and exome sequencing study of language disorder in an isolated population. *PEDIATRICS* **137**(4), e20152469–e20152469 (2016). DOI 10.1542/peds.2015-2469. URL <https://doi.org/10.1542%2Fpeds.2015-2469>
101. Korrel, H., Mueller, K.L., Silk, T., Anderson, V., Sciberras, E.: Research review: Language problems in children with attention-deficit hyperactivity disorder - a systematic meta-analytic review. *Journal of Child Psychology and Psychiatry* **58**(6), 640–654 (2017). DOI 10.1111/jcpp.12688. URL <https://doi.org/10.1111%2Fjcpp.12688>

102. Kravariti, E., Dixon, T., Frith, C., Murray, R., McGuire, P.: Association of symptoms and executive function in schizophrenia and bipolar disorder. *Schizophrenia Research* **74**(2-3), 221–231 (2005). DOI 10.1016/j.schres.2004.06.008. URL <https://doi.org/10.1016%2Fj.schres.2004.06.008>
103. Kremeyer, B., García, J., Kymilinen, H., Wratten, N., Restrepo, G., Palacio, C., Miranda, A.L., López, C., Restrepo, M., Bedoya, G., Brzustowicz, L.M., Ospina-Duque, J., Arbeláez, M.P., Ruiz-Linares, A.: Evidence for a role of the nos1ap (capon) gene in schizophrenia and its clinical dimensions: An association study in a south american population isolate. *Human Heredity* **67**(3), 163–173 (2008). DOI 10.1159/000181154. URL <https://doi.org/10.1159%2F000181154>
104. de Krom, M., Staal, W.G., Ophoff, R.A., Hendriks, J., Buitelaar, J., Franke, B., de Jonge, M.V., Bolton, P., Collier, D., Curran, S., van Engeland, H., van Ree, J.M.: A common variant in DRD3 receptor is associated with autism spectrum disorder. *Biological Psychiatry* **65**(7), 625–630 (2009). DOI 10.1016/j.biopsych.2008.09.035. URL <https://doi.org/10.1016%2Fj.biopsych.2008.09.035>
105. Kujala, T., Lepist, T., Ntinen, R.: The neural basis of aberrant speech and audition in autism spectrum disorders. *Neuroscience & Biobehavioral Reviews* **37**(4), 697–704 (2013). DOI 10.1016/j.neubiorev.2013.01.006. URL <https://doi.org/10.1016%2Fj.neubiorev.2013.01.006>
106. Kunugi, H., Vallada, H.P., Sham, P.C., Hoda, F., Arranz, M.J., Li, T., Nanko, S., Murray, R.M., McGuffin, P., Owen, M., Gill, M., Collier, D.A.: Catechol-o-methyltransferase polymorphisms and schizophrenia. *Psychiatric Genetics* **7**(3), 97–102 (1997). DOI 10.1097/00041444-199723000-00001. URL <https://doi.org/10.1097%2F00041444-199723000-00001>
107. Kuperberg, G.R.: Language in schizophrenia part 1: An introduction. *Language and Linguistics Compass* **4**(8), 576–589 (2010). DOI 10.1111/j.1749-818x.2010.00216.x. URL <https://doi.org/10.1111%2Fj.1749-818x.2010.00216.x>
108. Kuperberg, G.R.: Language in schizophrenia part 2: What can psycholinguistics bring to the study of schizophrenia...and vice versa? *Language and Linguistics Compass* **4**(8), 590–604 (2010). DOI 10.1111/j.1749-818x.2010.00217.x. URL <https://doi.org/10.1111%2Fj.1749-818x.2010.00217.x>
109. Lai, C.S.L., Fisher, S.E., Hurst, J.A., Vargha-Khadem, F., Monaco, A.P.: A forkhead-domain gene is mutated in a severe speech and language disorder. *Nature* **413**(6855), 519–523 (2001). DOI 10.1038/35097076. URL <https://doi.org/10.1038%2F35097076>
110. Lasky-Su, J., Neale, B.M., Franke, B., Anney, R.J., Zhou, K., Maller, J.B., Vasquez, A.A., Chen, W., Asherson, P., Buitelaar, J., Banaschewski, T., Ebstein, R., Gill, M., Miranda, A., Mulas, F., Oades, R.D., Roeyers, H., Rothenberger, A., Sergeant, J., Sonuga-Barke, E., Steinhausen, H.C., Taylor, E., Daly, M., Laird, N., Lange, C., Faraone, S.V.: Genome-wide association scan of quantitative traits for attention deficit hyperactivity disorder identifies novel associations and confirms candidate gene associations. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **147B**(8), 1345–1354 (2008). DOI 10.1002/ajmg.b.30867. URL <https://doi.org/10.1002%2Fajmg.b.30867>
111. Lattner, S., Meyer, M.E., Friederici, A.D.: Voice perception: Sex, pitch, and the right hemisphere. *Human Brain Mapping* **24**(1), 11–20 (2004). DOI 10.1002/hbm.20065. URL <https://doi.org/10.1002%2Fhbm.20065>
112. Lawford, B.R., Morris, C.P., Swagell, C.D., Hughes, I.P., Young, R.M., Voisey, J.: NOS1ap is associated with increased severity of PTSD and depression in untreated combat veterans. *Journal of Affective Disorders* **147**(1-3), 87–93 (2013). DOI 10.1016/j.jad.2012.10.013. URL <https://doi.org/10.1016%2Fj.jad.2012.10.013>
113. Lee, C.W., Kim, S.H., Shim, M., Ryu, V., Ha, R.Y., Lee, S.J., Cho, H.S.: P600 alteration of syntactic language processing in patients with bipolar mania: Comparison to schizophrenic patients and healthy subjects. *Journal of Affective Disorders* **201**, 101–111 (2016). DOI 10.1016/j.jad.2016.05.008. URL <https://doi.org/10.1016%2Fj.jad.2016.05.008>
114. Lee, H.J., Woo, H.G., Greenwood, T.A., Kripke, D.F., Kelsoe, J.R.: A genome-wide association study of seasonal pattern mania identifies NF1a as a possible susceptibility gene for bipolar disorder. *Journal of Affective Disorders* **145**(2), 200–207 (2013). DOI 10.1016/j.jad.2012.07.032. URL <https://doi.org/10.1016%2Fj.jad.2012.07.032>
115. Lesca, G., Rudolf, G., Bruneau, N., Lozovaya, N., Labalme, A., Boutry-Kryza, N., Salmi, M., Tsintsadze, T., Addis, L., Motte, J., Wright, S., Tsintsadze, V., Michel, A., Doummar, D., Lascelles, K., Strug, L., Waters, P., de Bellescize, J., Vrielynck, P., de Saint Martin,

- A., Ville, D., Ryvlin, P., Arzimanoglou, A., Hirsch, E., Vincent, A., Pal, D., Burnashev, N., Sanlaville, D., Szepietowski, P.: GRIN2a mutations in acquired epileptic aphasia and related childhood focal epilepsies and encephalopathies with speech and language dysfunction. *Nature Genetics* **45**(9), 1061–1066 (2013). DOI 10.1038/ng.2726. URL <https://doi.org/10.1038%2Fng.2726>
116. Lesch, K.P., Timmesfeld, N., Renner, T.J., Halperin, R., Rser, C., Nguyen, T.T., Craig, D.W., Romanos, J., Heine, M., Meyer, J., Freitag, C., Warnke, A., Romanos, M., Schfer, H., Walitza, S., Reif, A., Stephan, D.A., Jacob, C.: Molecular genetics of adult ADHD: converging evidence from genome-wide association and extended pedigree linkage studies. *Journal of Neural Transmission* **115**(11), 1573–1585 (2008). DOI 10.1007/s00702-008-0119-3. URL <https://doi.org/10.1007%2Fs00702-008-0119-3>
  117. Levinson, D.F., Shi, J., Wang, K., Oh, S., Riley, B., Pulver, A.E., Wildenauer, D.B., Laurent, C., Mowry, B.J., Gejman, P.V., Owen, M.J., Kendler, K.S., Nestadt, G., Schwab, S.G., Mallet, J., Nertney, D., Sanders, A.R., Williams, N.M., Wormley, B., Lasseter, V.K., Albus, M., Godard-Bauché, S., Alexander, M., Duan, J., O'Donovan, M.C., Walsh, D., O'Neill, A., Papadimitriou, G.N., Dikeos, D., Maier, W., Lerer, B., Campion, D., Cohen, D., Jay, M., Fanous, A., Eichhammer, P., Silverman, J.M., Norton, N., Zhang, N., Hakonarson, H., Gao, C., Citri, A., Hansen, M., Ripke, S., Dudbridge, F., and, P.A.H.: Genome-wide association study of multiplex schizophrenia pedigrees. *American Journal of Psychiatry* **169**(9), 963–973 (2012). DOI 10.1176/appi.ajp.2012.11091423. URL <https://doi.org/10.1176%2Fappi.ajp.2012.11091423>
  118. Lewis, J.D., Evans, A.C., Pruett, J.R., Botteron, K., Zwaigenbaum, L., Estes, A., Gerig, G., Collins, L., Kostopoulos, P., McKinstry, R., Dager, S., Paterson, S., Schultz, R.T., Styner, M., Hazlett, H., Piven, J.: Network inefficiencies in autism spectrum disorder at 24 months. *Translational Psychiatry* **4**(5), e388–e388 (2014). DOI 10.1038/tp.2014.24. URL <https://doi.org/10.1038%2Ftp.2014.24>
  119. Li, Q., Wineinger, N.E., Fu, D.J., Libiger, O., Alphs, L., Savitz, A., Gopal, S., Cohen, N., Schork, N.J.: Genome-wide association study of paliperidone efficacy. *Pharmacogenetics and Genomics* **27**(1), 7–18 (2017). DOI 10.1097/fpc.0000000000000250. URL <https://doi.org/10.1097%2Ffpc.0000000000000250>
  120. Li, T., Zeng, Z., Zhao, Q., Wang, T., Huang, K., Li, J., Li, Y., Liu, J., Wei, Z., Wang, Y., Feng, G., He, L., Shi, Y.: FoxP2 is significantly associated with schizophrenia and major depression in the Chinese Han population. *The World Journal of Biological Psychiatry* **14**(2), 146–150 (2012). DOI 10.3109/15622975.2011.615860. URL <https://doi.org/10.3109%2F15622975.2011.615860>
  121. Li, Z., Chen, J., Yu, H., He, L., Xu, Y., Zhang, D., Yi, Q., Li, C., Li, X., Shen, J., Song, Z., Ji, W., Wang, M., Zhou, J., Chen, B., Liu, Y., Wang, J., Wang, P., Yang, P., Wang, Q., Feng, G., Liu, B., Sun, W., Li, B., He, G., Li, W., Wan, C., Xu, Q., Li, W., Wen, Z., Liu, K., Huang, F., Ji, J., Ripke, S., Yue, W., Sullivan, P.F., O'Donovan, M.C., Shi, Y.: Genome-wide association analysis identifies 30 new susceptibility loci for schizophrenia. *Nature Genetics* **49**(11), 1576–1583 (2017). DOI 10.1038/ng.3973. URL <https://doi.org/10.1038%2Fng.3973>
  122. Liang, S., Lai Wang, X., Yang Zou, M., Wang, H., Zhou, X., Hong Sun, C., Xia, W., Jie Wu, L., Fujisawa, T.X., Tomoda, A.: Family-based association study of ZNF533, DOCK4 and IMMP2L gene polymorphisms linked to autism in a northeastern Chinese Han population. *Journal of Zhejiang University SCIENCE B* **15**(3), 264–271 (2014). DOI 10.1631/jzus.b1300133. URL <https://doi.org/10.1631%2Fjzus.b1300133>
  123. Lin, E., Kuo, P.H., Liu, Y.L., Yu, Y.W.Y., Yang, A.C., Tsai, S.J.: A deep learning approach for predicting antidepressant response in major depression using clinical and genetic biomarkers. *Frontiers in Psychiatry* **9** (2018). DOI 10.3389/fpsy.2018.00290. URL <https://doi.org/10.3389%2Fpsy.2018.00290>
  124. Liu, Y., Blackwood, D.H., Caesar, S., de Geus, E.J.C., Farmer, A., Ferreira, M.A.R., Ferrier, I.N., Fraser, C., Gordon-Smith, K., Green, E.K., Grozeva, D., Gurling, H.M., Hamshere, M.L., Heutink, P., Holmans, P.A., Hoogendijk, W.J., Hottenga, J.J., Jones, L., Jones, I.R., Kirov, G., Lin, D., McGuffin, P., Moskvina, V., Nolen, W.A., Perlis, R.H., Posthuma, D., Scolnick, E.M., Smit, A.B., Smit, J.H., Smoller, J.W., Clair, D.S., van Dyck, R., Verhage, M., Willemsen, G., Young, A.H., Zandbelt, T., Boomsma, D.I., Craddock, N., O'Donovan, M.C., Owen, M.J., Penninx, B.W.J.H., Purcell, S., Sklar, P., Sullivan, P.F.: Meta-analysis of genome-wide association data of bipolar disorder and major depressive disorder. *Molecular Psychiatry* **16**(1), 2–4 (2010). DOI 10.1038/mp.2009.107. URL <https://doi.org/10.1038%2Fmp.2009.107>

125. Lott, P., Guggenbhl, S., Schneeberger, A., Pulver, A., Stassen, H.: Linguistic analysis of the speech output of schizophrenic, bipolar, and depressive patients. *Psychopathology* **35**(4), 220–227 (2002). DOI 10.1159/000063831. URL <https://doi.org/10.1159/000063831>
126. Luciano, M., Evans, D.M., Hansell, N.K., Medland, S.E., Montgomery, G.W., Martin, N.G., Wright, M.J., Bates, T.C.: A genome-wide association study for reading and language abilities in two population cohorts. *Genes, Brain and Behavior* **12**(6), 645–652 (2013). DOI 10.1111/gbb.12053. URL <https://doi.org/10.1111/2Fgbb.12053>
127. Luo, M., Fan, J., Wenger, T.L., Harr, M.H., Racobaldo, M., Mulchandani, S., Dubbs, H., Zackai, E.H., Spinner, N.B., Conlin, L.K.: CMIP haploinsufficiency in two patients with autism spectrum disorder and co-occurring gastrointestinal issues. *American Journal of Medical Genetics Part A* **173**(8), 2101–2107 (2017). DOI 10.1002/ajmg.a.38277. URL <https://doi.org/10.1002/2Fajmg.a.38277>
128. Maestrini, E., Pagnamenta, A.T., Lamb, J.A., Bacchelli, E., Sykes, N.H., Sousa, I., Toma, C., Barnby, G., Butler, H., Winchester, L., Scerri, T.S., Minopoli, F., Reichert, J., Cai, G., Buxbaum, J.D., Korvatska, O., Schellenberg, G.D., Dawson, G., de Bildt, A., Minderaa, R.B., Mulder, E.J., Morris, A.P., Bailey, A.J., Monaco, A.P.: High-density SNP association study and copy number variation analysis of the AUTS1 and AUTS5 loci implicate the IMMP2L–DOCK4 gene region in autism susceptibility. *Molecular Psychiatry* **15**(9), 954–968 (2009). DOI 10.1038/mp.2009.34. URL <https://doi.org/10.1038/2Fmp.2009.34>
129. Maria, E.D., Gulli, R., Begni, S., Luca, A.D., Bignotti, S., Pasini, A., Bellone, E., Pizuti, A., Dallapiccola, B., Novelli, G., Ajmar, F., Gennarelli, M., Mandich, P.: Variations in the NMDA receptor subunit 2b gene (GRIN2b) and schizophrenia: A case-control study. *American Journal of Medical Genetics* **128B**(1), 27–29 (2004). DOI 10.1002/ajmg.b.30028. URL <https://doi.org/10.1002/2Fajmg.b.30028>
130. Martin, J., Walters, R.K., Demontis, D., Mattheisen, M., Lee, S.H., Robinson, E., Brikell, I., Ghirardi, L., Larsson, H., Lichtenstein, P., Eriksson, N., Werge, T., Mortensen, P.B., Pedersen, M.G., Mors, O., Nordentoft, M., Hougaard, D.M., Bybjerg-Grauholm, J., Wray, N.R., Franke, B., Faraone, S.V., O'Donovan, M.C., Thapar, A., Børghlum, A.D., Neale, B.M., Ager, M., Alipanahi, B., Auton, A., Bell, R.K., Bryc, K., Elson, S.L., Fontanillas, P., Furlotte, N.A., Hinds, D.A., Hromatka, B.S., Huber, K.E., Kleinman, A., Litterman, N.K., McIntyre, M.H., Mountain, J.L., Northover, C.A., Pitts, S.J., Sathirapongsasuti, J.F., Sazonova, O.V., Shelton, J.F., Shringarpure, S., Tian, C., Tung, J.Y., Vacic, V., Wilson, C.H., zgr Albayrak, Anney, R.J., Vasquez, A.A., Arranz, M.J., Asherson, P., Banaschewski, T., Banaschewski, T.J., Bau, C., Biederman, J., Mortensen, P.B., Børghlum, A., Buitelaar, J.K., Casas, M., Charach, A., Cormand, B., Crosbie, J., Dalsgaard, S., Daly, M.J., Demontis, D., Dempfle, A., Doyle, A.E., Ebstein, R.P., Elia, J., Faraone, S.V., Faraone, S.V., Fcker, M., Franke, B., Freitag, C., Gelernter, J., Gill, M., Grevet, E., Haavik, J., Hakonarson, H., Hawi, Z., Hebebrand, J., Herpertz-Dahlmann, B., Hervas, A., Hinney, A., Hohmann, S., Holmans, P., Hutz, M., Ickowitz, A., Johansson, S., Kent, L., Kittel-Schneider, S., Kranzler, H., Kuntsi, J., Lambregts-Rommelse, N., Langley, K., Lehmkuhl, G., Lesch, K.P., Loo, S.K., Martin, J., McGough, J.J., Medland, S.E., Meyer, J., Mick, E., Middleton, F., Miranda, A., Mulas, F., Mulligan, A., Neale, B.M., Nelson, S.F., Nguyen, T.T., O'Donovan, M.C., Oades, R.D., Owen, M.J., Palmason, H., Ramos-Quiroga, J.A., Reif, A., Renner, T.J., Rhode, L., Ribasés, M., Rietschel, M., Ripke, S., Rivero, O., Roeyers, H., Romanos, M., Romanos, J., Mota, N.R., Rothenberger, A., Sánchez-Mora, C., Schachar, R., Schfer, H., Scherag, A., Schimmelfmann, B.G., Sergeant, J., Sinzig, J., Smalley, S.L., Sonuga-Barke, E.J., Steinhausen, H.C., Sullivan, P.F., Thapar, A., Thompsom, M., Todorov, A., Waldman, I., Walitza, S., Walters, R., Wang, Y., Warnke, A., Williams, N., Witt, S.H., Yang, L., Zayats, T., Zhang-James, Y., Agerbo, E., Als, T.D., Bækved-Hansen, M., Belliveau, R., Børghlum, A.D., Bybjerg-Grauholm, J., Cerrato, F., Chambert, K., Churchhouse, C., Dalsgaard, S., Daly, M.J., Demontis, D., Dumont, A., Goldstein, J., Grove, J., Hansen, C.S., Hauberg, M.E., Hollegaard, M.V., Hougaard, D.M., Howrigan, D.P., Huang, H., Maller, J., Martin, A.R., Martin, J., Mattheisen, M., Moran, J., Mors, O., Mortensen, P.B., Neale, B.M., Nordentoft, M., Pallesen, J., Palmer, D.S., Pedersen, C.B., Pedersen, M.G., Poterba, T., Poulsen, J.B., Ripke, S., Robinson, E.B., Satterstrom, F.K., Stevens, C., Turley, P., Walters, R.K., Werge, T.: A genetic investigation of sex bias in the prevalence of attention-deficit/hyperactivity disorder. *Biological Psychiatry* **83**(12), 1044–1053 (2018). DOI 10.1016/j.biopsych.2017.11.026. URL <https://doi.org/10.1016/2Fj.biopsych.2017.11.026>



131. Martucci, L., Wong, A.H., Luca, V.D., Likhodi, O., Wong, G.W., King, N., Kennedy, J.L.: N-methyl-d-aspartate receptor NR2b subunit gene GRIN2b in schizophrenia and bipolar disorder: Polymorphisms and mRNA levels. *Schizophrenia Research* **84**(2-3), 214–221 (2006). DOI 10.1016/j.schres.2006.02.001. URL <https://doi.org/10.1016%2Fj.schres.2006.02.001>
132. Mascheretti, S., Riva, V., Giorda, R., Beri, S., Lanzoni, L.F.E., Cellino, M.R., Marino, C.: KIAA0319 and ROBO1: evidence on association with reading and pleiotropic effects on language and mathematics abilities in developmental dyslexia. *Journal of Human Genetics* **59**(4), 189–197 (2014). DOI 10.1038/jhg.2013.141. URL <https://doi.org/10.1038%2Fjhg.2013.141>
133. McCarthy-Jones, S., Green, M.J., Scott, R.J., Tooney, P.A., Cairns, M.J., Wu, J.Q., Oldmeadow, C., Carr, V.: Preliminary evidence of an interaction between the FOXP2 gene and childhood emotional abuse predicting likelihood of auditory verbal hallucinations in schizophrenia. *Journal of Psychiatric Research* **50**, 66–72 (2014). DOI 10.1016/j.jpsychires.2013.11.012. URL <https://doi.org/10.1016%2Fj.jpsychires.2013.11.012>
134. McElroy, S.L., Winham, S.J., Cuellar-Barboza, A.B., Colby, C.L., Ho, A.M.C., Sicotte, H., Larrabee, B.R., Crow, S., Frye, M.A., Biernacka, J.M.: Bipolar disorder with binge eating behavior: a genome-wide association study implicates PRR5-ARHGAP8. *Translational Psychiatry* **8**(1) (2018). DOI 10.1038/s41398-017-0085-3. URL <https://doi.org/10.1038%2Fs41398-017-0085-3>
135. van der Meer, D., Hoekstra, P.J., van Donkelaar, M., Bralten, J., Oosterlaan, J., Heslenfeld, D., Faraone, S.V., Franke, B., Buitelaar, J.K., Hartman, C.A.: Predicting attention-deficit/hyperactivity disorder severity from psychosocial stress and stress-response genes: a random forest regression approach. *Translational Psychiatry* **7**(6), e1145–e1145 (2017). DOI 10.1038/tp.2017.114. URL <https://doi.org/10.1038%2Ftp.2017.114>
136. Meffre, J., Chaumont-Dubel, S., la Cour, C.M., Loiseau, F., Watson, D.J.G., Dekeyne, A., Séveno, M., Rivet, J.M., Gaven, F., Délérès, P., Hervé, D., Fone, K.C.F., Bockaert, J., Millan, M.J., Marin, P.: 5-HT6receptor recruitment of mTOR as a mechanism for perturbed cognition in schizophrenia. *EMBO Molecular Medicine* **4**(10), 1043–1056 (2012). DOI 10.1002/emmm.201201410. URL <https://doi.org/10.1002%2Femmm.201201410>
137. Mellios, N., Galdzicka, M., Ginns, E., Baker, S.P., Rogaev, E., Xu, J., Akbarian, S.: Gender-specific reduction of estrogen-sensitive small RNA, miR-30b, in subjects with schizophrenia. *Schizophrenia Bulletin* **38**(3), 433–443 (2010). DOI 10.1093/schbul/sbq091. URL <https://doi.org/10.1093%2Fschbul%2Fsbq091>
138. Meng, H., Smith, S.D., Hager, K., Held, M., Liu, J., Olson, R.K., Pennington, B.F., DeFries, J.C., Gelernter, J., O'Reilly-Pol, T., Somlo, S., Skudlarski, P., Shaywitz, S.E., Shaywitz, B.A., Marchione, K., Wang, Y., Paramasivam, M., LoTurco, J.J., Page, G.P., Gruen, J.R.: From the cover: DCDC2 is associated with reading disability and modulates neuronal development in the brain. *Proceedings of the National Academy of Sciences* **102**(47), 17053–17058 (2005). DOI 10.1073/pnas.0508591102. URL <https://doi.org/10.1073%2Fpnas.0508591102>
139. Mill, J., Kiss, E., Baji, I., Kapornai, K., Daróczy, G., Vetró, Á., Kennedy, J., Kovacs, M., and, C.B.: Association study of the estrogen receptor alpha gene (ESR1) and childhood-onset mood disorders. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **147B**(7), 1323–1326 (2008). DOI 10.1002/ajmg.b.30751. URL <https://doi.org/10.1002%2Fajmg.b.30751>
140. MITCHELL, S., BRIAN, J., ZWAIGENBAUM, L., ROBERTS, W., SZATMARI, P., SMITH, I., BRYSON, S.: Early language and communication development of infants later diagnosed with autism spectrum disorder. *Journal of Developmental & Behavioral Pediatrics* **27**(Supplement 2), S69–S78 (2006). DOI 10.1097/00004703-200604002-00004. URL <https://doi.org/10.1097%2F00004703-200604002-00004>
141. Morgan, A., Fisher, S.E., Scheffer, I., Hildebrand, M.: FOXP2-Related Speech and Language Disorders. University of Washington (2016). URL <https://www.ncbi.nlm.nih.gov/books/NBK368474/>
142. Moskvina, V., , Craddock, N., Holmans, P., Nikolov, I., Pahwa, J.S., Green, E., Owen, M.J., O'Donovan, M.C.: Gene-wide analyses of genome-wide association data sets: evidence for multiple common risk alleles for schizophrenia and bipolar disorder and for overlap in genetic risk. *Molecular Psychiatry* **14**(3), 252–260 (2008). DOI 10.1038/mp.2008.133. URL <https://doi.org/10.1038%2Fmp.2008.133>

143. Mueller, K.L., Tomblin, J.B.: Examining the comorbidity of language impairment and attention-deficit/hyperactivity disorder. *Topics in Language Disorders* **32**(3), 228–246 (2012). DOI 10.1097/tld.0b013e318262010d. URL <https://doi.org/10.1097%2Ftld.0b013e318262010d>
144. Nagel, M., Jansen, P.R., Stringer, S., Watanabe, K., de Leeuw, C.A., Bryois, J., Savage, J.E., Hammerschlag, A.R., Skene, N.G., Munoz-Manchado, A.B., White, T., Tiemeier, H., Linnarsson, S., Hjerling-Leffler, J., Polderman, T.J.C., Sullivan, P.F., van der Sluis, S., Posthuma, D.: Meta-analysis of genome-wide association studies for neuroticism in 449,484 individuals identifies novel genetic loci and pathways. *Nature Genetics* **50**(7), 920–927 (2018). DOI 10.1038/s41588-018-0151-7. URL <https://doi.org/10.1038%2Fs41588-018-0151-7>
145. Nenadic, I., Langbein, K., Dietzek, M., Forberg, A., Smesny, S., Sauer, H.: Cognitive function in euthymic bipolar disorder (BP i) patients with a history of psychotic symptoms vs. schizophrenia. *Psychiatry Research* **230**(1), 65–69 (2015). DOI 10.1016/j.psychres.2015.08.012. URL <https://doi.org/10.1016%2Fj.psychres.2015.08.012>
146. Newbury, D.F., Paracchini, S., Scerri, T.S., Winchester, L., Addis, L., Richardson, A.J., Walter, J., Stein, J.F., Talcott, J.B., Monaco, A.P.: Investigation of dyslexia and SLI risk variants in reading- and language-impaired subjects. *Behavior Genetics* **41**(1), 90–104 (2010). DOI 10.1007/s10519-010-9424-3. URL <https://doi.org/10.1007%2Fs10519-010-9424-3>
147. Newbury, D.F., Winchester, L., Addis, L., Paracchini, S., Buckingham, L.L., Clark, A., Cohen, W., Cowie, H., Dworzynski, K., Everitt, A., Goodyer, I.M., Hennessy, E., Kindley, A.D., Miller, L.L., Nasir, J., O'Hare, A., Shaw, D., Simkin, Z., Simonoff, E., Slonims, V., Watson, J., Ragoussis, J., Fisher, S.E., Seckl, J.R., Helms, P.J., Bolton, P.F., Pickles, A., Conti-Ramsden, G., Baird, G., Bishop, D.V., Monaco, A.P.: CMIP and ATP2c2 modulate phonological short-term memory in language impairment. *The American Journal of Human Genetics* **85**(2), 264–272 (2009). DOI 10.1016/j.ajhg.2009.07.004. URL <https://doi.org/10.1016%2Fj.ajhg.2009.07.004>
148. Nudel, R., Simpson, N.H., Baird, G., O'Hare, A., Conti-Ramsden, G., Bolton, P.F., Hennessy, E.R., Ring, S.M., Smith, G.D., Francks, C., Paracchini, S., Monaco, A.P., Fisher, S.E., and, D.F.N.: Genome-wide association analyses of child genotype effects and parent-of-origin effects in specific language impairment. *Genes, Brain and Behavior* **13**(4), 418–429 (2014). DOI 10.1111/gbb.12127. URL <https://doi.org/10.1111%2Fgbb.12127>
149. Ocklenburg, S., Arning, L., Hahn, C., Gerding, W.M., Epplen, J.T., Gntrkn, O., Beste, C.: Variation in the NMDA receptor 2b subunit gene GRIN2b is associated with differential language lateralization. *Behavioural Brain Research* **225**(1), 284–289 (2011). DOI 10.1016/j.bbr.2011.07.042. URL <https://doi.org/10.1016%2Fj.bbr.2011.07.042>
150. Pagnamenta, A.T., Bacchelli, E., de Jonge, M.V., Mirza, G., Scerri, T.S., Minopoli, F., Chiocchetti, A., Ludwig, K.U., Hoffmann, P., Paracchini, S., Lowy, E., Harold, D.H., Chapman, J.A., Klauck, S.M., Poustka, F., Houben, R.H., Staal, W.G., Ophoff, R.A., O'Donovan, M.C., Williams, J., Nthen, M.M., Schulte-Krue, G., Deloukas, P., Ragoussis, J., Bailey, A.J., Maestrini, E., Monaco, A.P.: Characterization of a family with rare deletions in CNTNAP5 and DOCK4 suggests novel risk loci for autism and dyslexia. *Biological Psychiatry* **68**(4), 320–328 (2010). DOI 10.1016/j.biopsych.2010.02.002. URL <https://doi.org/10.1016%2Fj.biopsych.2010.02.002>
151. Palaniyappan, L., Mahmood, J., Balain, V., Mouglin, O., Gowland, P.A., Liddle, P.F.: Structural correlates of formal thought disorder in schizophrenia: An ultra-high field multivariate morphometry study. *Schizophrenia Research* **168**(1-2), 305–312 (2015). DOI 10.1016/j.schres.2015.07.022. URL <https://doi.org/10.1016%2Fj.schres.2015.07.022>
152. Palmatier, M.A., Pakstis, A.J., Speed, W., Paschou, P., Goldman, D., Odunsi, A., Okonofua, F., Kajuna, S., Karoma, N., Kungulilo, S., Grigorenko, E., Zhukova, O.V., Bonne-Tamir, B., Lu, R.B., Parnas, J., Kidd, J.R., DeMille, M.M.C., Kidd, K.K.: COMT haplotypes suggest p2 promoter region relevance for schizophrenia. *Molecular Psychiatry* **9**(9), 859–870 (2004). DOI 10.1038/sj.mp.4001496. URL <https://doi.org/10.1038%2Fsj.mp.4001496>
153. Pan, Y., Chen, J., Guo, H., Ou, J., Peng, Y., Liu, Q., Shen, Y., Shi, L., Liu, Y., Xiong, Z., Zhu, T., Luo, S., Hu, Z., Zhao, J., Xia, K.: Association of genetic variants of GRIN2b with autism. *Scientific Reports* **5**(1) (2015). DOI 10.1038/srep08296. URL <https://doi.org/10.1038%2Fsrep08296>
154. Paracchini, S., Steer, C.D., Buckingham, L.L., Morris, A.P., Ring, S., Scerri, T., Stein, J., Pembrey, M.E., Ragoussis, J., Golding, J., Monaco,

- A.P.: Association of the KIAA0319 dyslexia susceptibility gene with reading skills in the general population. *American Journal of Psychiatry* **165**(12), 1576–1584 (2008). DOI 10.1176/appi.ajp.2008.07121872. URL <https://doi.org/10.1176%2Fappi.ajp.2008.07121872>
155. Pardiñas, A.F., Holmans, P., Pocklington, A.J., Escott-Price, V., Ripke, S., Carrera, N., Legge, S.E., Bishop, S., Cameron, D., Hamshere, M.L., Han, J., Hubbard, L., Lynham, A., Mantripragada, K., Rees, E., MacCabe, J.H., McCarroll, S.A., Baune, B.T., Breen, G., Byrne, E.M., Dannlowski, U., Eley, T.C., Hayward, C., Martin, N.G., McIntosh, A.M., Plomin, R., Porteous, D.J., Wray, N.R., Caballero, A., Geschwind, D.H., Huckins, L.M., Ruderfer, D.M., Santiago, E., Sklar, P., Stahl, E.A., Won, H., Agerbo, E., Als, T.D., Andreassen, O.A., Bækvad-Hansen, M., Mortensen, P.B., Pedersen, C.B., Børghlum, A.D., Bybjerg-Grauholm, J., Djurovic, S., Durmishi, N., Pedersen, M.G., Golimbet, V., Grove, J., Hougaard, D.M., Mattheisen, M., Molden, E., Mors, O., Nordentoft, M., Pejovic-Milovancevic, M., Sigurdsson, E., Silagadze, T., Hansen, C.S., Stefansson, K., Stefansson, H., Steinberg, S., Tosato, S., Werge, T., Collier, D.A., Rujescu, D., Kirov, G., Owen, M.J., O'Donovan, M.C., and, J.T.R.W.: Common schizophrenia alleles are enriched in mutation-intolerant genes and in regions under strong background selection. *Nature Genetics* **50**(3), 381–389 (2018). DOI 10.1038/s41588-018-0059-2. URL <https://doi.org/10.1038%2Fs41588-018-0059-2>
  156. Patel, S.P., Kim, J.H., Larson, C.R., Losh, M.: Mechanisms of voice control related to prosody in autism spectrum disorder and first-degree relatives. *Autism Research* (2019). DOI 10.1002/aur.2156. URL <https://doi.org/10.1002%2Faur.2156>
  157. Patten, E., Belardi, K., Baranek, G.T., Watson, L.R., Labban, J.D., Oller, D.K.: Vocal patterns in infants with autism spectrum disorder: Canonical babbling status and vocalization frequency. *Journal of Autism and Developmental Disorders* **44**(10), 2413–2428 (2014). DOI 10.1007/s10803-014-2047-4. URL <https://doi.org/10.1007%2Fs10803-014-2047-4>
  158. Pavy, D.: Verbal behavior in schizophrenia: A review of recent studies. *Psychological Bulletin* **70**(3, Pt.1), 164–178 (1968). DOI 10.1037/h0020191. URL <https://doi.org/10.1037%2Fh0020191>
  159. Petrides, M.: Connectivity of the core language areas. In: *Neuroanatomy of Language Regions of the Human Brain*, pp. 139–174. Elsevier (2014). DOI 10.1016/b978-0-12-405514-8.50006-2. URL <https://doi.org/10.1016%2Fb978-0-12-405514-8.50006-2>
  160. PGC: Biological insights from 108 schizophrenia-associated genetic loci. *Nature* **511**(7510), 421–427 (2014). DOI 10.1038/nature13595. URL <https://doi.org/10.1038%2Fnature13595>
  161. Pickles, A., Simonoff, E., Conti-Ramsden, G., Falcato, M., Simkin, Z., Charman, T., Chandler, S., Loucas, T., Baird, G.: Loss of language in early development of autism and specific language impairment. *Journal of Child Psychology and Psychiatry* **50**(7), 843–852 (2009). DOI 10.1111/j.1469-7610.2008.02032.x. URL <https://doi.org/10.1111%2Fj.1469-7610.2008.02032.x>
  162. Pineda, D.A., Puerta, I.C., Aguirre, D.C., García-Barrera, M.A., Kamphaus, R.W.: The role of neuropsychologic tests in the diagnosis of attention deficit hyperactivity disorder. *Pediatric Neurology* **36**(6), 373–381 (2007). DOI 10.1016/j.pediatrneurol.2007.02.002. URL <https://doi.org/10.1016%2Fj.pediatrneurol.2007.02.002>
  163. Poldrack, R.A., Wagner, A.D., Prull, M.W., Desmond, J.E., Glover, G.H., Gabrieli, J.D.: Functional specialization for semantic and phonological processing in the left inferior prefrontal cortex. *NeuroImage* **10**(1), 15–35 (1999). DOI 10.1006/nimg.1999.0441. URL <https://doi.org/10.1006%2Fnimg.1999.0441>
  164. Potkin, S.G., Turner, J.A., Guffanti, G., Lakatos, A., Fallon, J.H., Nguyen, D.D., Mathalon, D., Ford, J., Lauriello, J., Macciardi, F.: A genome-wide association study of schizophrenia using brain activation as a quantitative phenotype. *Schizophrenia Bulletin* **35**(1), 96–108 (2008). DOI 10.1093/schbul/sbn155. URL <https://doi.org/10.1093%2Fschbul%2Fsbn155>
  165. Pourcain, B.S., Cents, R.A., Whitehouse, A.J., Haworth, C.M., Davis, O.S., O'Reilly, P.F., Roulstone, S., Wren, Y., Ang, Q.W., Velders, F.P., Evans, D.M., Kemp, J.P., Warrington, N.M., Miller, L., Timpson, N.J., Ring, S.M., Verhulst, F.C., Hofman, A., Rivadeneira, F., Meaburn, E.L., Price, T.S., Dale, P.S., Pillas, D., Ylihera, A., Rodriguez, A., Golding, J., Jaddoe, V.W., Jarvelin, M.R., Plomin, R., Pennell, C.E., Tiemeier, H., Smith, G.D.: Common variation near ROBO2 is associated with expressive vocabulary

- in infancy. *Nature Communications* **5**(1) (2014). DOI 10.1038/ncomms5831. URL <https://doi.org/10.1038%2Fncomms5831>
166. Price, J.L., Drevets, W.C.: Neurocircuitry of mood disorders. *Neuropsychopharmacology* **35**(1), 192–216 (2009). DOI 10.1038/npp.2009.104. URL <https://doi.org/10.1038%2Fnpp.2009.104>
  167. R, T., R, S.: *Language, Learning, and Behavior Disorders: Developmental, Biological, and Clinical Perspectives*. Cambridge University Press (1996)
  168. Rao, W., Du, X., Zhang, Y., Yu, Q., Hui, L., Yu, Y., Kou, C., Yin, G., Zhu, X., Man, L., Soares, J.C., Zhang, X.Y.: Association between forkhead-box p2 gene polymorphism and clinical symptoms in chronic schizophrenia in a chinese population. *Journal of Neural Transmission* **124**(7), 891–897 (2017). DOI 10.1007/s00702-017-1723-x. URL <https://doi.org/10.1007%2Fs00702-017-1723-x>
  169. Raucher-Ch  n  , D., Achim, A.M., Kaladjian, A., Besche-Richard, C.: Verbal fluency in bipolar disorders: A systematic review and meta-analysis. *Journal of Affective Disorders* **207**, 359–366 (2017). DOI 10.1016/j.jad.2016.09.039. URL <https://doi.org/10.1016%2Fj.jad.2016.09.039>
  170. Redmond, S.M.: Language impairment in the attention-deficit/hyperactivity disorder context. *Journal of Speech, Language, and Hearing Research* **59**(1), 133–142 (2016). DOI 10.1044/2015.jslhr-l-15-0038. URL <https://doi.org/10.1044%2F2015.jslhr-l-15-0038>
  171. Reinke, B., Ven, V., Matura, S., Linden, D., Oertel-Kn  chel, V.: Altered intrinsic functional connectivity in language-related brain regions in association with verbal memory performance in euthymic bipolar patients. *Brain Sciences* **3**(4), 1357–1373 (2013). DOI 10.3390/brainsci3031357. URL <https://doi.org/10.3390%2Fbrainsci3031357>
  172. Ribas  s, M., S  nchez-Mora, C., Ramos-Quiroga, J.A., Bosch, R., G  mez, N., Nogueira, M., Corrales, M., Palomar, G., Jacob, C.P., Gross-Lesch, S., Kreiker, S., Reif, A., Lesch, K.P., Cormand, B., Casas, M., Bay  s, M.: An association study of sequence variants in the forkhead box p2 (FOXP2) gene and adulthood attention-deficit/hyperactivity disorder in two european samples. *Psychiatric Genetics* **22**(4), 155–160 (2012). DOI 10.1097/ypg.0b013e328353957e. URL <https://doi.org/10.1097%2Fypg.0b013e328353957e>
  173. Ripke, S., O'Dushlaine, C., Chambert, K., Moran, J.L., Khler, A.K., Akterin, S., Bergen, S.E., Collins, A.L., Crowley, J.J., Fromer, M., Kim, Y., Lee, S.H., Magnusson, P.K.E., Sanchez, N., Stahl, E.A., Williams, S., Wray, N.R., Xia, K., Bettella, F., Borglum, A.D., Bulik-Sullivan, B.K., Cormican, P., Craddock, N., de Leeuw, C., Durmishi, N., Gill, M., Golimbet, V., Hamshire, M.L., Holmans, P., Hougaard, D.M., Kendler, K.S., Lin, K., Morris, D.W., Mors, O., Mortensen, P.B., Neale, B.M., O'Neill, F.A., Owen, M.J., Milovancevic, M.P., Posthuma, D., Powell, J., Richards, A.L., Riley, B.P., Ruderfer, D., Rujescu, D., Sigurdsson, E., Silagadze, T., Smit, A.B., Stefansson, H., Steinberg, S., Suvisaari, J., Tosato, S., Verhage, M., Walters, J.T., Bramon, E., Corvin, A.P., O'Donovan, M.C., Stefansson, K., Scolnick, E., Purcell, S., McCarroll, S.A., Sklar, P., Hultman, C.M., Sullivan, P.F., and: Genome-wide association analysis identifies 13 new risk loci for schizophrenia. *Nature Genetics* **45**(10), 1150–1159 (2013). DOI 10.1038/ng.2742. URL <https://doi.org/10.1038%2Fng.2742>
  174. Rish, I., Cecchi, G.A.: Functional network disruptions in schizophrenia. In: *Methods in Molecular Biology*, pp. 479–504. Springer New York (2017). DOI 10.1007/978-1-4939-7027-8\_19. URL [https://doi.org/10.1007%2F978-1-4939-7027-8\\_19](https://doi.org/10.1007%2F978-1-4939-7027-8_19)
  175. Riva, V., Marino, C., Giorda, R., Molteni, M., Nobile, M.: The role of DCDC2 genetic variants and low socioeconomic status in vulnerability to attention problems. *European Child & Adolescent Psychiatry* **24**(3), 309–318 (2014). DOI 10.1007/s00787-014-0580-5. URL <https://doi.org/10.1007%2Fs00787-014-0580-5>
  176. Rolls, E.T., Cheng, W., Gilson, M., Qiu, J., Hu, Z., Ruan, H., Li, Y., Huang, C.C., Yang, A.C., Tsai, S.J., Zhang, X., Zhuang, K., Lin, C.P., Deco, G., Xie, P., Feng, J.: Effective connectivity in depression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* **3**(2), 187–197 (2018). DOI 10.1016/j.bpsc.2017.10.004. URL <https://doi.org/10.1016%2Fj.bpsc.2017.10.004>
  177. Royer, C., Delcroix, N., Leroux, E., Alary, M., Razafimandimby, A., Brazo, P., Delamillieure, P., Dollfus, S.: Functional and structural brain asymmetries in patients with schizophrenia and bipolar disorders. *Schizophrenia Research* **161**(2-3), 210–214 (2015). DOI 10.1016/j.schres.2014.11.014. URL <https://doi.org/10.1016%2Fj.schres.2014.11.014>
  178. Rubinstein, M., Patowary, A., Stanaway, I.B., McCord, E., Nesbitt, R.R., Archer, M., Scheuer, T., Nickerson, D., Raskind, W.H., Wijsman, E.M., Bernier, R., Catterall, W.A.,

- Brkanac, Z.: Association of rare missense variants in the second intracellular loop of NaV1.7 sodium channels with familial autism. *Molecular Psychiatry* **23**(2), 231–239 (2016). DOI 10.1038/mp.2016.222. URL <https://doi.org/10.1038/mp.2016.222>
179. Ruderfer, D.M., Fanous, A.H., Ripke, S., McQuillin, A., Amdur, R.L., Gejman, P.V., O'Donovan, M.C., Andreassen, O.A., Djurovic, S., Hultman, C.M., Kelsoe, J.R., Jamain, S., Landén, M., Leboyer, M., Nimgaonkar, V., Nurnberger, J., Smoller, J.W., Craddock, N., Corvin, A., Sullivan, P.F., Holmans, P., Sklar, P., Kendler, K.S., and: Polygenic dissection of diagnosis and clinical dimensions of bipolar disorder and schizophrenia. *Molecular Psychiatry* **19**(9), 1017–1024 (2013). DOI 10.1038/mp.2013.138. URL <https://doi.org/10.1038/mp.2013.138>
  180. Ryan, J., Scali, J., Carrière, I., Peres, K., Rouaud, O., Scarabin, P.Y., Ritchie, K., Ancelin, M.L.: Estrogen receptor alpha gene variants and major depressive episodes. *Journal of Affective Disorders* **136**(3), 1222–1226 (2012). DOI 10.1016/j.jad.2011.10.010. URL <https://doi.org/10.1016/j.jad.2011.10.010>
  181. Ryan, K.A., Vederman, A.C., McFadden, E.M., Weldon, A.L., Kamali, M., Lange-necker, S.A., McInnis, M.G.: Differential executive functioning performance by phase of bipolar disorder. *Bipolar Disorders* **14**(5), 527–536 (2012). DOI 10.1111/j.1399-5618.2012.01032.x. URL <https://doi.org/10.1111/j.1399-5618.2012.01032.x>
  182. Sanjuán, J., Tolosa, A., González, J.C., Aguilar, E.J., Pérez, J., Nájera, C., Moltis, M.D., de Frutos, R.: Association between FOXP2 polymorphisms and schizophrenia with auditory hallucinations. *Psychiatric Genetics* **16**(2), 67–72 (2006). DOI 10.1097/01.ypg.0000185029.35558.bb. URL <https://doi.org/10.1097/01.ypg.0000185029.35558.bb>
  183. Saur, D., Kreher, B.W., Schnell, S., Kummerer, D., Kellmeyer, P., Vry, M.S., Umarova, R., Musso, M., Glauche, V., Abel, S., Huber, W., Rijntjes, M., Hennig, J., Weiller, C.: Ventral and dorsal pathways for language. *Proceedings of the National Academy of Sciences* **105**(46), 18035–18040 (2008). DOI 10.1073/pnas.0805234105. URL <https://doi.org/10.1073/pnas.0805234105>
  184. Schumann, C.M., Bloss, C.S., Barnes, C.C., Wideman, G.M., Carper, R.A., Akshoomoff, N., Pierce, K., Hagler, D., Schork, N., Lord, C., Courchesne, E.: Longitudinal magnetic resonance imaging study of cortical development through early childhood in autism. *Journal of Neuroscience* **30**(12), 4419–4427 (2010). DOI 10.1523/jneurosci.5714-09.2010. URL <https://doi.org/10.1523/jneurosci.5714-09.2010>
  185. Sciberras, E., Mueller, K.L., Efron, D., Bisset, M., Anderson, V., Schilpzand, E.J., Jongeling, B., Nicholson, J.M.: Language problems in children with ADHD: A community-based study. *PEDIATRICS* **133**(5), 793–800 (2014). DOI 10.1542/peds.2013-3355. URL <https://doi.org/10.1542/peds.2013-3355>
  186. Serrallach, B., Groß, C., Bernhofs, V., Engelmann, D., Benner, J., Gndert, N., Blatow, M., Wengenroth, M., Seitz, A., Brunner, M., Seither, S., Parncutt, R., Schneider, P., Seither-Preisler, A.: Neural biomarkers for dyslexia, ADHD, and ADD in the auditory cortex of children. *Frontiers in Neuroscience* **10** (2016). DOI 10.3389/fnins.2016.00324. URL <https://doi.org/10.3389/fnins.2016.00324>
  187. Shenton, M.E., Kikinis, R., Jolesz, F.A., Pollak, S.D., LeMay, M., Wible, C.G., Hokama, H., Martin, J., Metcalf, D., Coleman, M., McCarley, R.W.: Abnormalities of the left temporal lobe and thought disorder in schizophrenia. *New England Journal of Medicine* **327**(9), 604–612 (1992). DOI 10.1056/nejm199208273270905. URL <https://doi.org/10.1056/nejm199208273270905>
  188. Shifman, S., Bronstein, M., Sternfeld, M., Pisanté-Shalom, A., Lev-Lehman, E., Weizman, A., Reznik, I., Spivak, B., Grisaru, N., Karp, L., Schiffer, R., Kotler, M., Strous, R.D., Swartz-Vanetik, M., Knobler, H.Y., Shinar, E., Beckmann, J.S., Yakir, B., Risch, N., Zak, N.B., Darvasi, A.: A highly significant association between a COMT haplotype and schizophrenia. *The American Journal of Human Genetics* **71**(6), 1296–1302 (2002). DOI 10.1086/344514. URL <https://doi.org/10.1086/344514>
  189. Silva, I.M., Rosenfeld, J., Antoniuk, S.A., Raskin, S., Sotomaior, V.S.: A 1.5mb terminal deletion of 12p associated with autism spectrum disorder. *Gene* **542**(1), 83–86 (2014). DOI 10.1016/j.gene.2014.02.058. URL <https://doi.org/10.1016/j.gene.2014.02.058>
  190. Siper, P.M., Rubeis, S.D., del Pilar Trelles, M., Durkin, A., Marino, D.D., Muratet, F., Frank, Y., Lozano, R., Eichler, E.E., Kelly, M., Beighley, J., Gerdts, J., Wallace, A.S., Mefford, H.C., Bernier, R.A., Kolevzon, A., Buxbaum, J.D.: Prospective investigation of FOXP1 syndrome. *Molecular Autism* **8**(1) (2017). DOI 10.1186/s13229-017-0172-6. URL <https://doi.org/10.1186/s13229-017-0172-6>

191. Sklar, P., Smoller, J.W., Fan, J., Ferreira, M.A.R., Perlis, R.H., Chambert, K., Nimgaonkar, V.L., McQueen, M.B., Faraone, S.V., Kirby, A., de Bakker, P.I.W., Ogdie, M.N., Thase, M.E., Sachs, G.S., Todd-Brown, K., Gabriel, S.B., Sougnez, C., Gates, C., Blumenstiel, B., Defelice, M., Ardlie, K.G., Franklin, J., Muir, W.J., McGhee, K.A., MacIntyre, D.J., McLean, A., VanBeck, M., McQuillin, A., Bass, N.J., Robinson, M., Lawrence, J., Anjorin, A., Curtis, D., Scolnick, E.M., Daly, M.J., Blackwood, D.H., Gurling, H.M., Purcell, S.M.: Whole-genome association study of bipolar disorder. *Molecular Psychiatry* **13**(6), 558–569 (2008). DOI 10.1038/sj.mp.4002151. URL <https://doi.org/10.1038%2Fsj.mp.4002151>
192. Španiel, F., Horáček, J., Tintěra, J., Ibrahim, I., Novák, T., Čermák, J., Klířová, M., Hscl, C.: Genetic variation in FOXP2 alters grey matter concentrations in schizophrenia patients. *Neuroscience Letters* **493**(3), 131–135 (2011). DOI 10.1016/j.neulet.2011.02.024. URL <https://doi.org/10.1016%2Fj.neulet.2011.02.024>
193. Sperdin, H.F., Schaer, M.: Aberrant development of speech processing in young children with autism: New insights from neuroimaging biomarkers. *Frontiers in Neuroscience* **10** (2016). DOI 10.3389/fnins.2016.00393. URL <https://doi.org/10.3389%2Ffnins.2016.00393>
194. Splawski, I., Timothy, K.W., Sharpe, L.M., Decher, N., Kumar, P., Bloise, R., Napolitano, C., Schwartz, P.J., Joseph, R.M., Condouris, K., Tager-Flusberg, H., Priori, S.G., Sanguinetti, M.C., Keating, M.T.: CaV1.2 calcium channel dysfunction causes a multisystem disorder including arrhythmia and autism. *Cell* **119**(1), 19–31 (2004). DOI 10.1016/j.cell.2004.09.011. URL <https://doi.org/10.1016%2Fj.cell.2004.09.011>
195. Stegmayer, K., Stettler, M., Strik, W., Federspiel, A., Wiest, R., Bohlhalter, S., Walther, S.: Resting state perfusion in the language network is linked to formal thought disorder and poor functional outcome in schizophrenia. *Acta Psychiatrica Scandinavica* **136**(5), 506–516 (2017). DOI 10.1111/acps.12790. URL <https://doi.org/10.1111%2Fjacps.12790>
196. Sterponi, L., de Kirby, K., Shankey, J.: Rethinking language in autism. *Autism* **19**(5), 517–526 (2014). DOI 10.1177/1362361314537125. URL <https://doi.org/10.1177%2F1362361314537125>
197. Subotnik, K., Bartzokis, G., Green, M., Nuechterlein, K.: Neuroanatomical correlates of formal thought disorder in schizophrenia. *Cognitive Neuropsychiatry* **8**(2), 81–88 (2003). DOI 10.1080/13546800244000148. URL <https://doi.org/10.1080%2F13546800244000148>
198. Sugiura, L., Toyota, T., Matsuba-Kurita, H., Iwayama, Y., Mazuka, R., Yoshikawa, T., Hagiwara, H.: Age-dependent effects of catechol-o-methyltransferase (COMT) gene val158met polymorphism on language function in developing children. *Cerebral Cortex* **27**(1), 104–116 (2016). DOI 10.1093/cercor/bhw371. URL <https://doi.org/10.1093%2Fcercor%2Fbhw371>
199. Sultana, R., Yu, C.E., Yu, J., Munson, J., Chen, D., Hua, W., Estes, A., Cortes, F., de la Barra, F., Yu, D., Haider, S.T., Trask, B.J., Green, E.D., Raskind, W.H., Distèche, C.M., Wijsman, E., Dawson, G., Storm, D.R., Schellenberg, G.D., Villacres, E.C.: Identification of a novel gene on chromosome 7q11.2 interrupted by a translocation breakpoint in a pair of autistic twins. *Genomics* **80**(2), 129–134 (2002). DOI 10.1006/geno.2002.6810. URL <https://doi.org/10.1006%2Fgeno.2002.6810>
200. Sun, X., Wu, Z., Cao, Q., Qian, Y., Liu, Y., Yang, B., Chang, S., Yang, L., Wang, Y.: Genetic variant for behavioral regulation factor of executive function and its possible brain mechanism in attention deficit hyperactivity disorder. *Scientific Reports* **8**(1) (2018). DOI 10.1038/s41598-018-26042-y. URL <https://doi.org/10.1038%2Fs41598-018-26042-y>
201. Sung, K., Gordon, B., Vannorsdall, T.D., Ledoux, K., Schretlen, D.J.: Impaired retrieval of semantic information in bipolar disorder: A clustering analysis of category-fluency productions. *Journal of Abnormal Psychology* **122**(3), 624–634 (2013). DOI 10.1037/a0033068. URL <https://doi.org/10.1037%2Fa0033068>
202. Tager-Flusberg, H.: Risk factors associated with language in autism spectrum disorder: Clues to underlying mechanisms. *Journal of Speech, Language, and Hearing Research* **59**(1), 143–154 (2016). DOI 10.1044/2015.jslhr-l-15-0146. URL <https://doi.org/10.1044%2F2015.jslhr-l-15-0146>
203. Taniguchi, S., Nakazawa, T., Tanimura, A., Kiyama, Y., Tezuka, T., Watabe, A.M., Katayama, N., Yokoyama, K., Inoue, T., Izumi-Nakaseko, H., Kakuta, S., Sudo, K., Iwakura, Y., Umemori, H., Inoue, T., Murphy, N.P., Hashimoto, K., Kano, M., Manabe, T., Yamamoto, T.: Involvement of NMDAR2a tyrosine phosphorylation in depression-related behaviour. *The EMBO Journal* **28**(23), 3717–3729 (2009). DOI 10.1038/emboj.2009.300. URL <https://doi.org/10.1038%2Femboj.2009.300>

204. Tarabeux, J., Kebir, O., Gauthier, J., Hamdan, F.F., Xiong, L., Piton, A., Spiegelman, D., Henrion, É., Millet, B., Fathalli, F., Joob, R., Rapoport, J.L., DeLisi, L.E., Fombonne, É., Mottron, L., Forget-Dubois, N., Boivin, M., Michaud, J.L., Drapeau, P., Lafrenière, R.G., Rouleau, G.A., Krebs, M.O.: Rare mutations in n-methyl-d-aspartate glutamate receptors in autism spectrum disorders and schizophrenia. *Translational Psychiatry* **1**(11), e55–e55 (2011). DOI 10.1038/tp.2011.52. URL <https://doi.org/10.1038%2Ftp.2011.52>
205. Tesli, M., Athanasiu, L., Mattingsdal, M., Khler, A.K., Gustafsson, O., Andreassen, B.K., Werge, T., Hansen, T., Mors, O., Mellerup, E., Koefoed, P., Jnsson, E.G., Agartz, I., Melle, I., Morken, G., Djurovic, S., Andreassen, O.A.: Association analysis of PALB2 and BRCA2 in bipolar disorder and schizophrenia in a scandinavian case-control sample. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **153B**(7), 1276–1282 (2010). DOI 10.1002/ajmg.b.31098. URL <https://doi.org/10.1002%2Fajmg.b.31098>
206. Of The Psychiatric Genomics Consortium, A.S.D.W.G.: Meta-analysis of GWAS of over 16,000 individuals with autism spectrum disorder highlights a novel locus at 10q24.32 and a significant overlap with schizophrenia. *Molecular Autism* **8**(1) (2017). DOI 10.1186/s13229-017-0137-9. URL <https://doi.org/10.1186%2Fs13229-017-0137-9>
207. Thevenon, J., Callier, P., Andrieux, J., Delobel, B., David, A., Sukno, S., Minot, D., Anne, L.M., Marle, N., Sanlaville, D., Bonnet, M., Masurel-Paulet, A., Levy, F., Gaunt, L., Farrell, S., Caignec, C.L., Toutain, A., Carmignac, V., Mugneret, F., Clayton-Smith, J., Thauvin-Robinet, C., Faivre, L.: 12p13.33 microdeletion including ELKS/ERC1, a new locus associated with childhood apraxia of speech. *European Journal of Human Genetics* **21**(1), 82–88 (2012). DOI 10.1038/ejhg.2012.116. URL <https://doi.org/10.1038%2Fejhg.2012.116>
208. Thomas, R., Sanders, S., Doust, J., Beller, E., Glasziou, P.: Prevalence of attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *PEDIATRICS* **135**(4), e994–e1001 (2015). DOI 10.1542/peds.2014-3482. URL <https://doi.org/10.1542%2Fpeds.2014-3482>
209. Tomblin, J.B., Records, N.L., Buckwalter, P., Zhang, X., Smith, E., O'Brien, M.: Prevalence of specific language impairment in kindergarten children. *Journal of Speech, Language, and Hearing Research* **40**(6), 1245–1260 (1997). DOI 10.1044/jslhr.4006.1245. URL <https://doi.org/10.1044%2Fjslhr.4006.1245>
210. Truong, D.T., Adams, A.K., Paniagua, S., Frijters, J.C., Boada, R., Hill, D.E., Lovett, M.W., Mahone, E.M., Willcutt, E.G., Wolf, M., Defries, J.C., Gialluisi, A., Francks, C., Fisher, S.E., Olson, R.K., Pennington, B.F., Smith, S.D., Bosson-Heenan, J., Gruen, J.R.: Multivariate genome-wide association study of rapid automatized naming and rapid alternating stimulus in hispanic american and african-american youth. *Journal of Medical Genetics* pp. jmedgenet-2018-105874 (2019). DOI 10.1136/jmedgenet-2018-105874. URL <https://doi.org/10.1136%2Fjmedgenet-2018-105874>
211. Turic, D., Langley, K., Mills, S., Stephens, M., Lawson, D., Govan, C., Williams, N., den Bree, M.V., Craddock, N., Kent, L., Owen, M., O'Donovan, M., Thapar, A.: Follow-up of genetic linkage findings on chromosome 16p13: evidence of association of n-methyl-d aspartate glutamate receptor 2a gene polymorphism with ADHD. *Molecular Psychiatry* **9**(2), 169–173 (2004). DOI 10.1038/sj.mp.4001387. URL <https://doi.org/10.1038%2Fsmp.4001387>
212. Uddin, M., Tammimies, K., Pellicchia, G., Alipanahi, B., Hu, P., Wang, Z., Pinto, D., Lau, L., Nalpathamkalam, T., Marshall, C.R., Blencowe, B.J., Frey, B.J., Merico, D., Yuen, R.K.C., Scherer, S.W.: Brain-expressed exons under purifying selection are enriched for de novo mutations in autism spectrum disorder. *Nature Genetics* **46**(7), 742–747 (2014). DOI 10.1038/ng.2980. URL <https://doi.org/10.1038%2Fng.2980>
213. Uher, R., Tansey, K.E., Henigsberg, N., Wolfgang, M., Mors, O., Hauser, J., Placentino, A., Souery, D., Farmer, A., Aitchison, K.J., Craig, I., Peter, M., Lewis, C.M., Ising, M., Lucae, S., Binder, E.B., Kloiber, S., Holsboer, F., Mller-Myhsok, B., Ripke, S., Hamilton, S.P., Soundy, J., Laje, G., McMahon, F.J., Fava, M., Rush, A.J., Perlis, R.H.: Common genetic variation and antidepressant efficacy in major depressive disorder: A meta-analysis of three genome-wide pharmacogenetic studies. *American Journal of Psychiatry* **170**(2), 207–217 (2013). DOI 10.1176/appi.ajp.2012.12020237. URL <https://doi.org/10.1176%2Fappi.ajp.2012.12020237>
214. Vandenbergh, R., Nobre, A.C., Price, C.J.: The response of left temporal cortex to sentences. *Journal of Cognitive Neuroscience* **14**(4), 550–560 (2002). DOI 10.1162/08989290260045800. URL <https://doi.org/10.1162%2F08989290260045800>

215. Vermeersch, H., T'Sjoen, G., Kaufman, J.M., Houtte, M.V.: ESR1 polymorphisms, daily hassles, anger expression, and depressive symptoms in adolescent boys and girls. *Hormones and Behavior* **63**(3), 447–453 (2013). DOI 10.1016/j.yhbeh.2012.11.017. URL <https://doi.org/10.1016%2Fj.yhbeh.2012.11.017>
216. Vernes, S.C., Newbury, D.F., Abrahams, B.S., Winchester, L., Nicod, J., Groszer, M., Alarcón, M., Oliver, P.L., Davies, K.E., Geschwind, D.H., Monaco, A.P., Fisher, S.E.: A functional genetic link between distinct developmental language disorders. *New England Journal of Medicine* **359**(22), 2337–2345 (2008). DOI 10.1056/nejmoa0802828. URL <https://doi.org/10.1056%2Fnejmoa0802828>
217. Villanueva, P., Nudel, R., Hoischen, A., Fernández, M.A., Simpson, N.H., Gilissen, C., Reader, R.H., Jara, L., Echeverry, M.M., Francks, C., Baird, G., Conti-Ramsden, G., O'Hare, A., Bolton, P.F., Hennessy, E.R., Palomino, H., Carvajal-Carmona, L., Veltman, J.A., Cazier, J.B., Barbieri, Z.D., Fisher, S.E., and, D.F.N.: Exome sequencing in an admixed isolated population indicates NFXL1 variants confer a risk for specific language impairment. *PLOS Genetics* **11**(3), e1004925 (2015). DOI 10.1371/journal.pgen.1004925. URL <https://doi.org/10.1371%2Fjournal.pgen.1004925>
218. Wang, K.S., Liu, X.F., Aragam, N.: A genome-wide meta-analysis identifies novel loci associated with schizophrenia and bipolar disorder. *Schizophrenia Research* **124**(1-3), 192–199 (2010). DOI 10.1016/j.schres.2010.09.002. URL <https://doi.org/10.1016%2Fj.schres.2010.09.002>
219. Weickert, C.S., Miranda-Angulo, A.L., Wong, J., Perlman, W.R., Ward, S.E., Radhakrishna, V., Straub, R.E., Weinberger, D.R., Kleinman, J.E.: Variants in the estrogen receptor alpha gene and its mRNA contribute to risk for schizophrenia. *Human Molecular Genetics* **17**(15), 2293–2309 (2008). DOI 10.1093/hmg/ddn130. URL <https://doi.org/10.1093%2Fhmg%2Fddn130>
220. Weinberger, D.R.: Future of days past: Neurodevelopment and schizophrenia. *Schizophrenia Bulletin* **43**(6), 1164–1168 (2017). DOI 10.1093/schbul/sbx118. URL <https://doi.org/10.1093%2Fschbul%2Fsbx118>
221. Whalley, H.C., O'Connell, G., Sussmann, J.E., Peel, A., Stanfield, A.C., Hayiou-Thomas, M.E., Johnstone, E.C., Lawrie, S.M., McIntosh, A.M., Hall, J.: Genetic variation in cntnap2 alters brain function during linguistic processing in healthy individuals. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **156**(8), 941–948 (2011). DOI 10.1002/ajmg.b.31241. URL <https://doi.org/10.1002%2Fajmg.b.31241>
222. Whitehouse, A.J.O., Bishop, D.V.M., Ang, Q.W., Pennell, C.E., Fisher, S.E.: CNTNAP2 variants affect early language development in the general population. *Genes, Brain and Behavior* **10**(4), 451–456 (2011). DOI 10.1111/j.1601-183x.2011.00684.x. URL <https://doi.org/10.1111%2Fj.1601-183x.2011.00684.x>
223. Wong, M.L., Dong, C., Maestre-Mesa, J., Licinio, J.: Polymorphisms in inflammation-related genes are associated with susceptibility to major depression and antidepressant response. *Molecular Psychiatry* **13**(8), 800–812 (2008). DOI 10.1038/mp.2008.59. URL <https://doi.org/10.1038%2Fmp.2008.59>
224. Wratten, N.S., Memoli, H., Huang, Y., Dulencin, A.M., Matteson, P.G., Cornacchia, M.A., Azaro, M.A., Messenger, J., Hayter, J.E., Bassett, A.S., Buyske, S., Millonig, J.H., Vieland, V.J., Brzustowicz, L.M.: Identification of a schizophrenia-associated functional noncoding variant in NOS1ap. *American Journal of Psychiatry* **166**(4), 434–441 (2009). DOI 10.1176/appi.ajp.2008.08081266. URL <https://doi.org/10.1176%2Fappi.ajp.2008.08081266>
225. Wykes, T.: Language and schizophrenia. *Psychological Medicine* **10**(3), 403–406 (1980). DOI 10.1017/s0033291700047279. URL <https://doi.org/10.1017%2Fs0033291700047279>
226. Yang, Y., Li, W., Zhang, H., Yang, G., Wang, X., Ding, M., Jiang, T., Lv, L.: Association study of n-methyl-d-aspartate receptor subunit 2b (GRIN2b) polymorphisms and schizophrenia symptoms in the han chinese population. *PLOS ONE* **10**(5), e0125925 (2015). DOI 10.1371/journal.pone.0125925. URL <https://doi.org/10.1371%2Fjournal.pone.0125925>
227. Yoo, H.J., Cho, I.H., Park, M., Yang, S.Y., Kim, S.A.: Family based association of GRIN2a and GRIN2b with korean autism spectrum disorders. *Neuroscience Letters* **512**(2), 89–93 (2012). DOI 10.1016/j.neulet.2012.01.061. URL <https://doi.org/10.1016%2Fj.neulet.2012.01.061>
228. Yu, H., Yan, H., Wang, L., Li, J., Tan, L., Deng, W., Chen, Q., Yang, G., Zhang, F., Lu, T., Yang, J., Li, K., Lv, L., Tan, Q., Zhang, H., Xiao, X., Li, M., Ma, X., Yang, F., Li, L., Wang, C., Li, T., Zhang, D., Yue, W.: Five novel loci associated with antipsychotic



- treatment response in patients with schizophrenia: a genome-wide association study. *The Lancet Psychiatry* **5**(4), 327–338 (2018). DOI 10.1016/s2215-0366(18)30049-x. URL <https://doi.org/10.1016%2Fs2215-0366%2818%2930049-x>
229. Zatorre, R.J., Meyer, E., Gjedde, A., Evans, A.C.: PET studies of phonetic processing of speech: Review, replication, and reanalysis. *Cerebral Cortex* **6**(1), 21–30 (1996). DOI 10.1093/cercor/6.1.21. URL <https://doi.org/10.1093%2Fcercor%2F6.1.21>
  230. Zettergren, A., Jonsson, L., Johansson, D., Melke, J., Lundström, S., Anckarster, H., Lichtenstein, P., Westberg, L.: Associations between polymorphisms in sex steroid related genes and autistic-like traits. *Psychoneuroendocrinology* **38**(11), 2575–2584 (2013). DOI 10.1016/j.psyneuen.2013.06.004. URL <https://doi.org/10.1016%2Fj.psyneuen.2013.06.004>
  231. Zettergren, A., Karlsson, S., Hovey, D., Jonsson, L., Melke, J., Anckarster, H., Lichtenstein, P., Lundström, S., Westberg, L.: Further investigations of the relation between polymorphisms in sex steroid related genes and autistic-like traits. *Psychoneuroendocrinology* **68**, 1–5 (2016). DOI 10.1016/j.psyneuen.2016.02.020. URL <https://doi.org/10.1016%2Fj.psyneuen.2016.02.020>
  232. Zhang, B., Xu, Y.H., Wei, S.G., Zhang, H.B., Fu, D.K., Feng, Z.F., Guan, F.L., Zhu, Y.S., Li, S.B.: Association study identifying a new susceptibility gene (AUTS2) for schizophrenia. *International Journal of Molecular Sciences* **15**(11), 19406–19416 (2014). DOI 10.3390/ijms151119406. URL <https://doi.org/10.3390%2Fijms151119406>
  233. Zhang, C., Li, Z., Wu, Z., Chen, J., Wang, Z., Peng, D., Hong, W., Yuan, C., Wang, Z., Yu, S., Xu, Y., Xu, L., Xiao, Z., Fang, Y.: A study of n-methyl-d-aspartate receptor gene (GRIN2b) variants as predictors of treatment-resistant major depression. *Psychopharmacology* **231**(4), 685–693 (2013). DOI 10.1007/s00213-013-3297-0. URL <https://doi.org/10.1007%2Fs00213-013-3297-0>
  234. Zhao, Q., Che, R., Zhang, Z., Wang, P., Li, J., Li, Y., Huang, K., Tang, W., Feng, G., Lindpaintner, K., He, L., Shi, Y.: Positive association between GRIN2b gene and bipolar disorder in the chinese han population. *Psychiatry Research* **185**(1-2), 290–292 (2011). DOI 10.1016/j.psychres.2009.11.026. URL <https://doi.org/10.1016%2Fj.psychres.2009.11.026>
  235. Zheng, Y., Li, H., Qin, W., Chen, W., Duan, Y., Xiao, Y., Li, C., Zhang, J., Li, X., Feng, G., He, L.: Association of the carboxyl-terminal PDZ ligand of neuronal nitric oxide synthase gene with schizophrenia in the chinese han population. *Biochemical and Biophysical Research Communications* **328**(4), 809–815 (2005). DOI 10.1016/j.bbrc.2005.01.037. URL <https://doi.org/10.1016%2Fj.bbrc.2005.01.037>
  236. Žilina, O., Reimand, T., Zjablovskaja, P., Mnnik, K., Mnnamaa, M., Traat, A., Puusepp-Benazzouz, H., Kurg, A., Öunap, K.: Maternally and paternally inherited deletion of 7q31 involving the FOXP2 gene in two families. *American Journal of Medical Genetics Part A* **158A**(1), 254–256 (2011). DOI 10.1002/ajmg.a.34378. URL <https://doi.org/10.1002%2Fajmg.a.34378>