Online Supplement for

Genetic neuroimaging of bipolar disorder: A systematic 2017-2020 update

by

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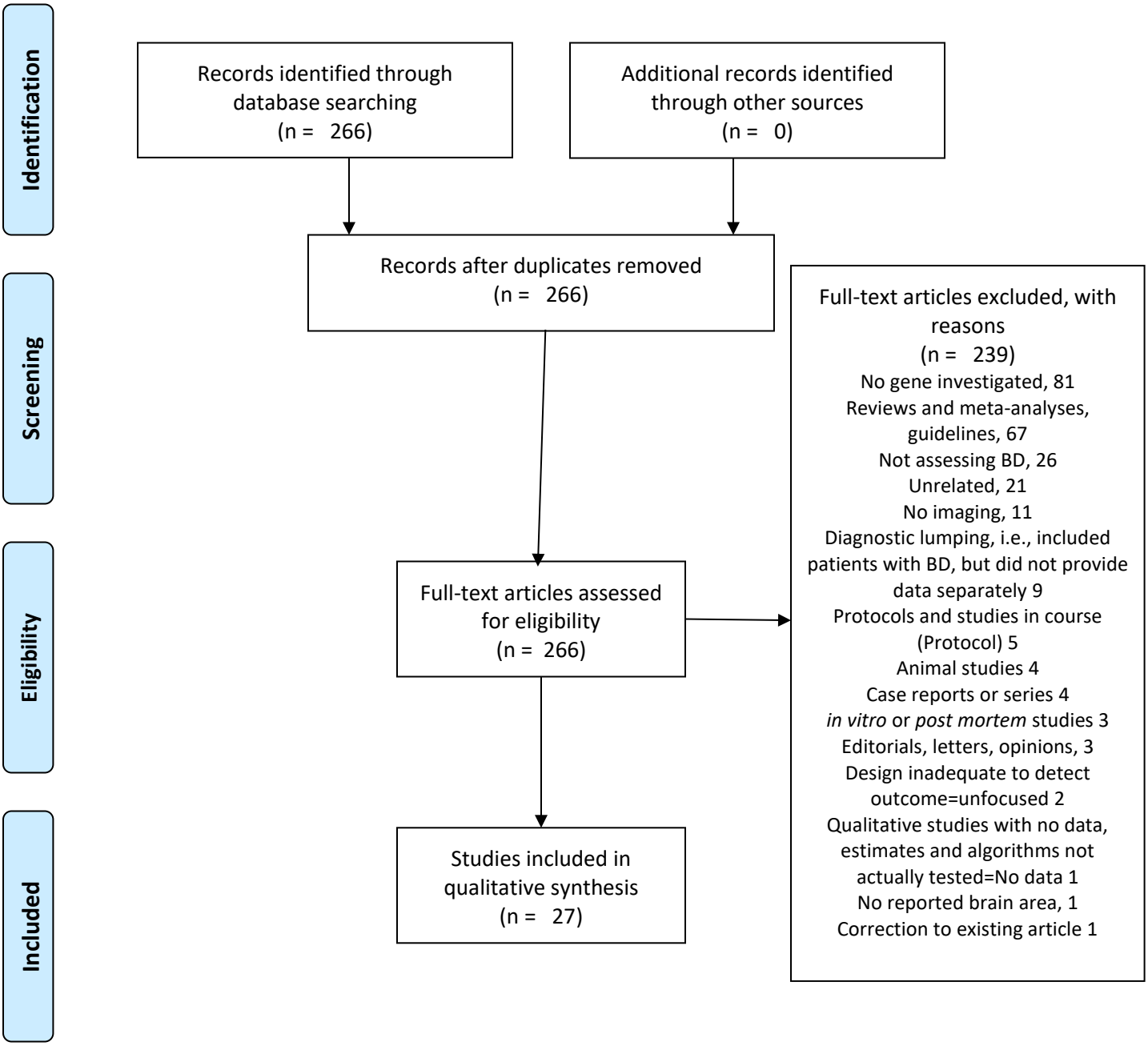
(SNPs OR single nucleotide polymorphism* OR haplotype* OR gene expression OR gene OR genes OR genetic score OR genetic* OR methylome OR telomere length OR epigenetic* OR genome OR transcriptome OR polymorphism OR genetic polymorphism OR genome wide OR genome-wide OR GWAS OR "polygenic risk score") AND ("bipolar disorder" OR "bipolar depression" OR mania OR manic) AND (neuroimaging OR "functional magnetic resonance" OR "structural magnetic resonance" OR fMRI OR BOLD fMRI OR "blood oxygen level dependent" OR tractography OR "voxel based morphometry" OR "positron emission tomography" OR "single photon" OR spect OR spet OR "magnetic resonance spectroscopy" OR DTI OR "diffusion tensor imaging" OR "fractional anisotropy" OR "white matter hyperintensity" OR "mean diffusivity" OR "radial diffusivity") on PubMed, 10-July-2020 → 265 records after restricting to 2017-2020



From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

PRISMA 2009 Checklist

Section/topic	#	Checklist item	Reported on page #
TITLE			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	1
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	1-2
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	1-2
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	N/A
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	2
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	2
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	2
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	2
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	2
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	Supplement
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	N/A, 10
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	Similarities between studies
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	2-7

PRISMA 2009 Checklist

Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	N/A
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	N/A
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	3
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	3-7
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	NA, 10
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	2-7
Synthesis of results	21	Present the main results of the review. If meta-analyses are done, include for each, confidence intervals and measures of consistency	3-6
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	N/A
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	None, N/A
DISCUSSION			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	7-10
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	10
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	10
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	None

From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

For more information, visit: **www.prisma-statement.org**.

PICO Worksheet and Search Strategy

Name_Janiri D et al._

1. Define your question using PICO by identifying: Problem, Intervention, Comparison Group and Outcomes.

Your question should be used to help establish your search strategy.

Patient/Problem___Having bipolar disorder (BD)___

Intervention__Analysis of polymorphisms, Being subjected to neuroimaging procedures___

Comparison___Bipolar disorder *vs.* other control groups (*vs*. healthy controls *vs.* patients with schizophrenia, *vs*. nonaffected relatives)___

Outcome__Presence of polymorphisms affecting brain structure and/or function__

Write out your question(s):____Is the risk gene carrier status in bipolar disorder patients paralleled by structural and/or functional brain alterations?_____

2. Type of question/problem: **Circle one:** Therapy/Prevention **Diagnosis Etiology** Prognosis

3. Type of study (Publication Type) to include in the search: **Check all that apply:**

Meta-Analysis **Systematic Review** Randomized Controlled Trial Cohort Study Case Control Study Case series or Case Report Editorials, Letters, Opinions Animal Research In Vitro/Lab Research

4. List main topics and alternate terms from your PICO question that can be used for your search

SNP (single nucleotide polymorphism) haplotype gene expression gene genetic score genetics methylome telomere length epigenetics genome transcriptome polymorphism polygenic risk score bipolar disorder bipolar depression mania manic neuroimaging functional magnetic resonance structural magnetic resonance blood oxygen level dependent tractography voxel based morphometry positron emission tomography

List your inclusion criteria –gender, age, List irrelevant terms that you may want year of publication, language to exclude in your search: For **eligibility**: papers ought to (i) be an original research article; (ii) include patients with a diagnosis of BD, based on Structured Clinical Interview for DSM-IV/5 (SCID or Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) or other recognised clinical diagnostic criteria e.g., diagnostic interview for genetic studies or ICD-9/10); (iii) include use of neuroimaging acquisition (structural MRI or functional MRI) of participants; (iv) include genetic analyses of participants; and (v) provide data separately for BD; Studies 2017-2020; No language restriction. Gender, any; Ages: All. **Exclusion** criteria were: (i) reviews and meta-analyses (although we used their reference lists to seek possible additional eligible studies that could have eluded our search strategy); (ii) not assessing genetics of participants; (iii) not assessing people with diagnosis of BD; (iii) editorials, letters to the editor with no data, opinion papers not supported by data, surveys of what doctors think about genetics of BD and neuroimaging; (iv) serendipitous search result with no actual relation to the subject matter; (v) not assessing neuroimaging of participants; (vi) in vitro or post-mortem studies; (vii) studies conducted on animals only; (viii) case reports or case series; (ix) diagnostic lumping, (i.e., included patients with BD, but did not provide data separately for BD-diagnosed participants); (x) protocols and ongoing studies; (xi) qualitative studies with no data or using estimates and algorithms not actually tested; (xii) surveys or consensus meetings; (xiii) design inadequate to assess changes; (xiv) correction to existing article; (xv) MRI and genetics on different samples; (xvi) not providing data for specific brain areas, but only general metabolic data

5. List where you plan to search, i.e. EBM Reviews, Medline, AIDSLINE, CINAHL, PubMed
MEDLINE/PubMed/Index Medicus

(SNPs OR single nucleotide polymorphism* OR haplotype* OR gene expression OR gene OR genes OR genetic score OR genetic* OR methylome OR telomere length OR epigenetic* OR genome OR transcriptome OR polymorphism OR genetic polymorphism OR genome wide OR genome-wide OR GWAS OR "polygenic risk score") AND ("bipolar disorder" OR "bipolar depression" OR mania OR manic) AND (neuroimaging OR "functional magnetic resonance" OR "structural magnetic resonance" OR fMRI OR BOLD fMRI OR "blood oxygen level dependent" OR tractography OR "voxel based morphometry" OR "positron emission tomography" OR "single photon" OR spect OR spet OR "magnetic resonance spectroscopy" OR DTI OR "diffusion tensor imaging" OR "fractional anisotropy" OR "white matter hyperintensity" OR "mean diffusivity" OR "radial diffusivity") → 825 results, 265 restricting to 2017-2020 on PubMed, 10-July-2020.

1.	Fears SC, Service SK, Kremeyer B, Araya C, Araya X, Bejarano J, Ramirez M, Castrillón G, Gomez-Franco J, Lopez MC, Montoya G, Montoya P, Aldana I, Teshiba TM, Al-Sharif NB, Jalbrzikowski M, Tishler TA, Escobar J, Ruiz-Linares A, Lopez-Jaramillo C, Macaya G, Molina J, Reus VI, Cantor RM, Sabatti C, Freimer NB, Bearden CE. Genome-wide mapping of brain phenotypes in extended pedigrees with strong genetic loading for bipolar disorder. Mol Psychiatry. 2020 Jun 30. doi: 10.1038/s41380-020-0805-6.	No BD
2.	Assmann A, Richter A, Schütze H, Soch J, Barman A, Behnisch G, Knopf L, Raschick M, Schult A, Wüstenberg T, Behr J, Düzel E, Seidenbecher CI, Schott BH. Neurocan genome-wide psychiatric risk variant affects explicit memory performance and hippocampal function in healthy humans. Eur J Neurosci. 2020 Jun 24. doi: 10.1111/ejn.14872.	No BD
3.	Sankar A, Purves K, Colic L, Lippard ETC, Millard H, Fan S, Spencer L, Wang F, Pittman B, Constable RT, Gross JJ, Blumberg HP. Altered frontal cortex functioning in emotion regulation and hopelessness in bipolar disorder. Bipolar Disord. 2020 Jun 10. doi: 10.1111/bdi.12954.	No gene
4.	Koshiyama D, Miura K, Nemoto K, Okada N, Matsumoto J, Fukunaga M, Hashimoto R. Neuroimaging studies within Cognitive Genetics Collaborative Research Organization aiming to replicate and extend works of ENIGMA. Hum Brain Mapp. 2020 Jun 5. doi: 10.1002/hbm.25040.	No gene
5.	Mendez MF, Parand L, Akhlaghipour G. Bipolar disorder among patients diagnosed with frontotemporal dementia. J Neuropsychiatry Clin Neurosci. 2020 Jun 5:appineuropsych20010003. doi: 10.1176/appi.neuropsych.20010003.	Review
6.	**Rodríguez-Ramírez AM, Meza-Urzúa F, Cedillo-Ríos V, Becerra-Palars C, Jiménez-Pavón J, Morales-Cedillo IP, Sanabrais-Jiménez MA, Hernández-Muñoz S, Camarena-Medellín B. *CACNA1C* risk variant and mood stabilizers effects in the prefrontal cortical thickness of Mexican patients with bipolar disorder. Neuropsychiatr Dis Treat. 2020;16:1199-1206. doi: 10.2147/NDT.S245911.**	**Included**
7.	Phillips JL, Jaworska N, Kamler E, Bhat V, Blier J, Foster JA, Hassel S, Ho K, McMurray L, Milev R, Moazamigoudarzi Z, Placenza FM, Richard-Devantoy S, Rotzinger S, Turecki G, Vazquez GH, Kennedy SH, Blier P; CAN-BIND Investigator Team. A randomized, crossover comparison of ketamine and electroconvulsive therapy for treatment of major depressive episodes: a Canadian biomarker integration network in depression (CAN-BIND) study protocol. BMC Psychiatry. 2020 Jun 2;20(1):268. doi: 10.1186/s12888-020-02672-3.	No gene
8.	Jabbi M, Arasappan D, Eickhoff SB, Strakowski SM, Nemeroff CB, Hofmann HA. Neuro-transcriptomic signatures for mood disorder morbidity and suicide mortality. J Psychiatr Res. 2020;127:62-74. doi: 10.1016/j.jpsychires.2020.05.013. Epub 2020 May 18.	*In vitro*
9.	McPhilemy G, Nabulsi L, Kilmartin L, Whittaker JR, Martyn FM, Hallahan B, McDonald C, Murphy K, Cannon DM. Resting-state network patterns underlying cognitive function in bipolar disorder: A graph theoretical analysis. Brain Connect. 2020 May 27. doi: 10.1089/brain.2019.0709.	No gene
10.	De la Serna E, Camprodon-Boadas P, Ilzarbe D, Sugranyes G, Baeza I, Moreno D, Díaz-Caneja CM, Rosa-Justicia M, Llorente C, Ayora M, Borras R, Torrent C, Bernardo M, Castro-Fornieles J. Neuropsychological development in the child and adolescent offspring of patients diagnosed with schizophrenia or bipolar disorder: A two-year follow-up comparative study. Prog Neuropsychopharmacol Biol Psychiatry. 2020 Dec 20;103:109972. doi: 10.1016/j.pnpbp.2020.109972. Epub 2020 May 23.	No BD
11.	Serykh A, Khrapova MV, Dubrovina NI, Petrova ES, Mikhnevich N, Starostina MV, Amstislavskaya TG, Lipina TV. The increased density of the habenular neurons, high impulsivity, aggression and resistant fear memory in Disc1-Q31L genetic mouse model of depression. Behav Brain Res. 2020 May 15;392:112693. doi: 10.1016/j.bbr.2020.112693.	Animal
12.	Navarrete F, García-Gutiérrez MS, Jurado-Barba R, Rubio G, Gasparyan A, Austrich-Olivares A, Manzanares J. Endocannabinoid System Components as Potential Biomarkers in Psychiatry. Front Psychiatry. 2020 Apr 27;11:315. doi: 10.3389/fpsyt.2020.00315.	Review
13.	Taquet M, Smith SM, Prohl AK, Peters JM, Warfield SK, Scherrer B, Harrison PJ. A structural brain network of genetic vulnerability to psychiatric illness. Mol Psychiatry. 2020 May 6. doi: 10.1038/s41380-020-0723-7.	No BD
14.	Zhang W, Sweeney JA, Yao L, Li S, Zeng J, Xu M, Tallman MJ, Gong Q, DelBello MP, Lui S, Nery FG. Brain structural correlates of familial risk for mental illness: a meta-analysis of voxel-based morphometry studies in relatives of patients with psychotic or mood disorders. Neuropsychopharmacology. 2020;45(8):1369-1379. doi: 10.1038/s41386-020-0687-y. Epub 2020 Apr 30.	Review
15.	Alloza C, Blesa-Cábez M, Bastin ME, Madole JW, Buchanan CR, Janssen J, Gibson J, Deary IJ, Tucker-Drob EM, Whalley HC, Arango C, McIntosh AM, Cox SR, Lawrie SM. Psychotic-like experiences, polygenic risk scores for schizophrenia, and structural properties of the salience, default mode, and central-executive networks in healthy participants from UK Biobank. Transl Psychiatry. 2020 Apr 27;10(1):122. doi: 10.1038/s41398-020-0794-x.	No BD
16.	Vismara M, Cirnigliaro G, Piccoli E, Giorgetti F, Molteni L, Cremaschi L, Fumagalli GG, D'addario C, Dell'Osso B. Crossing Borders Between Frontotemporal Dementia and Psychiatric Disorders: An Updated Overview. J Alzheimers Dis. 2020;75(2):661-673. doi: 10.3233/JAD-191333.	Review
17.	Zhuo C, Wang C, Song X, Xu X, Li G, Lin X, Xu Y, Tian H, Jiang D, Wang W, Zhou C. A unified model of shared brain structural alterations in patients with different mental disorders who experience own-thought auditory verbal hallucinations-A pilot study. Brain Behav. 2020 Jun;10(6):e01614. doi: 10.1002/brb3.1614. Epub 2020 Apr 18.	No gene
18.	Zhuo C, Ji F, Lin X, Tian H, Wang L, Xu Y, Wang W, Jiang D. Global functional connectivity density alterations in patients with bipolar disorder with auditory verbal hallucinations and modest short-term effects of transcranial direct	No gene

	current stimulation augmentation treatment-Baseline and follow-up study. Brain Behav. 2020;10(6):e01637. doi: 10.1002/brb3.1637. Epub 2020 Apr 18.	
19.	Kochunov P, Hong LE, Dennis EL, Morey RA, Tate DF, Wilde EA, Logue M, Kelly S, Donohoe G, Favre P, Houenou J, Ching CRK, Holleran L, Andreassen OA, van Velzen LS, Schmaal L, Villalón-Reina JE, Bearden CE, Piras F, Spalletta G, van den Heuvel OA, Veltman DJ, Stein DJ, Ryan MC, Tan Y, van Erp TGM, Turner JA, Haddad L, Nir TM, Glahn DC, Thompson PM, Jahanshad N. ENIGMA-DTI: Translating reproducible white matter deficits into personalized vulnerability metrics in cross-diagnostic psychiatric research. Hum Brain Mapp. 2020 Apr 16. doi: 10.1002/hbm.24998.	No gene
20.	Zhuo C, Lin X, Wang C, Song X, Xu X, Li G, Xu Y, Tian H, Zhang Y, Wang W, Zhou C. Unified and disease specific alterations to brain structure in patients across six categories of mental disorders who experience own-thought auditory verbal hallucinations: A pilot study. Brain Res Bull. 2020 Jul;160:33-39. doi: 10.1016/j.brainresbull.2020.04.001. Epub 2020 Apr 13.	No gene
21.	Wei Y, Chen Q, Curtin A, Tu L, Tang X, Tang Y, Xu L, Qian Z, Zhou J, Zhu C, Zhang T, Wang J. Functional near-infrared spectroscopy (fNIRS) as a tool to assist the diagnosis of major psychiatric disorders in a Chinese population. Eur Arch Psychiatry Clin Neurosci. 2020 Apr 11. doi: 10.1007/s00406-020-01125-y.	No gene
22.	Bond DJ, Torres IJ, Lam RW, Yatham LN. Serum epidermal growth factor, clinical illness course, and limbic brain volumes in early-stage bipolar disorder. J Affect Disord. 2020 Jun 1;270:30-35. doi: 10.1016/j.jad.2020.03.055. Epub 2020 Mar 27.	No gene
23.	Scaini G, Valvassori SS, Diaz AP, Lima CN, Benevenuto D, Fries GR, Quevedo J. Neurobiology of bipolar disorders: a review of genetic components, signaling pathways, biochemical changes, and neuroimaging findings. Braz J Psychiatry. 2020 Apr 3:S1516-44462020005007202. doi: 10.1590/1516-4446-2019-0732.	Review
24.	Thompson PM, Jahanshad N, Ching CRK, Salminen LE, Thomopoulos SI, Bright J, Baune BT, Bertolín S, Bralten J, Bruin WB, Bülow R, Chen J, Chye Y, Dannlowski U, de Kovel CGF, Donohoe G, Eyler LT, Faraone SV, Favre P, Filippi CA, Frodl T, Garijo D, Gil Y, Grabe HJ, Grasby KL, Hajek T, Han LKM, Hatton SN, Hilbert K, Ho TC, Holleran L, Homuth G, Hosten N, Houenou J, Ivanov I, Jia T, Kelly S, Klein M, Kwon JS, Laansma MA, Leerssen J, Lueken U, Nunes A, Neill JO, Opel N, Piras F, Piras F, Postema MC, Pozzi E, Shatokhina N, Soriano-Mas C, Spalletta G, Sun D, Teumer A, Tilot AK, Tozzi L, van der Merwe C, Van Someren EJW, van Wingen GA, Völzke H, Walton E, Wang L, Winkler AM, Wittfeld K, Wright MJ, Yun JY, Zhang G, Zhang-James Y, Adhikari BM, Agartz I, Aghajani M, Aleman A, Althoff RR, Altmann A, Andreassen OA, Baron DA, Bartnik-Olson BL, Marie Bas-Hoogendam J, Baskin-Sommers AR, Bearden CE, Berner LA, Boedhoe PSW, Brouwer RM, Buitelaar JK, Caeyenberghs K, Cecil CAM, Cohen RA, Cole JH, Conrod PJ, De Brito SA, de Zwarte SMC, Dennis EL, Desrivieres S, Dima D, Ehrlich S, Esopenko C, Fairchild G, Fisher SE, Fouche JP, Francks C, Frangou S, Franke B, Garavan HP, Glahn DC, Groenewold NA, Gurholt TP, Gutman BA, Hahn T, Harding IH, Hernaus D, Hibar DP, Hillary FG, Hoogman M, Hulshoff Pol HE, Jalbrzikowski M, Karkashadze GA, Klapwijk ET, Knickmeyer RC, Kochunov P, Koerte IK, Kong XZ, Liew SL, Lin AP, Logue MW, Luders E, Macciardi F, Mackey S, Mayer AR, McDonald CR, McMahon AB, Medland SE, Modinos G, Morey RA, Mueller SC, Mukherjee P, Namazova-Baranova L, Nir TM, Olsen A, Paschou P, Pine DS, Pizzagalli F, Rentería ME, Rohrer JD, Sämann PG, Schmaal L, Schumann G, Shiroishi MS, Sisodiya SM, Smit DJA, Sønderby IE, Stein DJ, Stein JL, Tahmasian M, Tate DF, Turner JA, van den Heuvel OA, van der Wee NJA, van der Werf YD, van Erp TGM, van Haren NEM, van Rooij D, van Velzen LS, Veer IM, Veltman DJ, Villalon-Reina JE, Walter H, Whelan CD, Wilde EA, Zarei M, Zelman V; ENIGMA Consortium. ENIGMA and global neuroscience: A decade of large-scale studies of the brain in health and disease across more than 40 countries. Transl Psychiatry. 2020 Mar 20;10(1):100. doi: 10.1038/s41398-020-0705-1.	Review
25.	Benedetti F, Aggio V, Pratesi ML, Greco G, Furlan R. Neuroinflammation in Bipolar Depression. Front Psychiatry. 2020 Feb 26;11:71. doi: 10.3389/fpsyt.2020.00071.	Review
26.	**Chen J, Tan J, Greenshaw AJ, Sawalha J, Liu Y, Zhang X, Zou W, Cheng X, Deng W, Zhang Y, Cui L, Liu C, Sun J, Cheng X, Wu Q, Li S, Mai S, Lan X, Chen Y, Cai Y, Zheng C, Cheng D, Zhang B, Yang C, Li X, Li X, Ye B, Yousefnezhad M, Zhang Y, Zhao L, Soares JC, Zhang X, Li T, Cao B, Cao L. CACNB2 rs11013860 polymorphism correlates of prefrontal cortex thickness in bipolar patients with first-episode mania. J Affect Disord. 2020 May 1;268:82-87. doi: 10.1016/j.jad.2020.02.007. Epub 2020 Feb 3.**	**Included**
27.	Madeira N, Duarte JV, Martins R, Costa GN, Macedo A, Castelo-Branco M. Morphometry and gyrification in bipolar disorder and schizophrenia: A comparative MRI study. Neuroimage Clin. 2020 Feb 19;26:102220. doi: 10.1016/j.nicl.2020.102220.	No gene
28.	Ducharme S, Dols A, Laforce R, Devenney E, Kumfor F, van den Stock J, Dallaire-Théroux C, Seelaar H, Gossink F, Vijverberg E, Huey E, Vandenbulcke M, Masellis M, Trieu C, Onyike C, Caramelli P, de Souza LC, Santillo A, Waldö ML, Landin-Romero R, Piguet O, Kelso W, Eratne D, Velakoulis D, Ikeda M, Perry D, Pressman P, Boeve B, Vandenberghe R, Mendez M, Azuar C, Levy R, Le Ber I, Baez S, Lerner A, Ellajosyula R, Pasquier F, Galimberti D, Scarpini E, van Swieten J, Hornberger M, Rosen H, Hodges J, Diehl-Schmid J, Pijnenburg Y. Recommendations to distinguish behavioural variant frontotemporal dementia from psychiatric disorders. Brain. 2020;143(6):1632-1650. doi: 10.1093/brain/awaa018.	Review
29.	Resende R, Fernandes T, Pereira AC, De Pascale J, Marques AP, Oliveira P, Morais S, Santos V, Madeira N, Pereira CF, Moreira PI. Mitochondria, endoplasmic reticulum and innate immune dysfunction in mood disorders: Do Mitochondria-Associated Membranes (MAMs) play a role? Biochim Biophys Acta Mol Basis Dis. 2020 Jun 1;1866(6):165752. doi: 10.1016/j.bbadis.2020.165752. Epub 2020 Feb 29.	Review
30.	Claude LA, Houenou J, Duchesnay E, Favre P. Will machine learning applied to neuroimaging in bipolar disorder help the clinician? A critical review and methodological suggestions. Bipolar Disord. 2020 Jun;22(4):334-355. doi: 10.1111/bdi.12895. Epub 2020 Mar 20.	Review

31.	Reddy-Thootkur M, Kraguljac NV, Lahti AC. The role of glutamate and GABA in cognitive dysfunction in schizophrenia and mood disorders - A systematic review of magnetic resonance spectroscopy studies. Schizophr Res. 2020 Feb 24:S0920-9964(20)30077-3. doi: 10.1016/j.schres.2020.02.001.	Review
32.	Petrasch-Parwez E, Schöbel A, Benali A, Moinfar Z, Förster E, Brüne M, Juckel G. Lateralization of increased density of Iba1-immunopositive microglial cells in the anterior midcingulate cortex of schizophrenia and bipolar disorder. Eur Arch Psychiatry Clin Neurosci. 2020 Feb 15. doi: 10.1007/s00406-020-01107-0.	*In vitro*
33.	Steardo L Jr, Manchia M, Carpiniello B, Pisanu C, Steardo L, Squassina A. Clinical, genetic, and brain imaging predictors of risk for bipolar disorder in high-risk individuals. Expert Rev Mol Diagn. 2020 Mar;20(3):327-333. doi: 10.1080/14737159.2020.1727743. Epub 2020 Feb 13.	Review
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219.	O'Donoghue S, Kilmartin L, O'Hora D, Emsell L, Langan C, McInerney S, Forde NJ, Leemans A, Jeurissen B, Barker GJ, McCarthy P, Cannon DM, McDonald C. Anatomical integration and rich-club connectivity in euthymic bipolar disorder. Psychol Med. 2017;47(9):1609-1623. doi: 10.1017/S0033291717000058.	No gene
220.	Birur B, Kraguljac NV, Shelton RC, Lahti AC. Brain structure, function, and neurochemistry in schizophrenia and bipolar disorder-a systematic review of the magnetic resonance neuroimaging literature. NPJ Schizophr. 2017;3:15. doi: 10.1038/s41537-017-0013-9.	Review
221.	**Miskowiak KW, Kjaerstad HL, Støttrup MM, Svendsen AM, Demant KM, Hoeffding LK, Werge TM, Burdick KE, Domschke K, Carvalho AF, Vieta E, Vinberg M, Kessing LV, Siebner HR, Macoveanu J. The catechol-O-methyltransferase (COMT) Val158Met genotype modulates working memory-related dorsolateral prefrontal response and performance in bipolar disorder. Bipolar Disord. 2017;19(3):214-224. doi: 10.1111/bdi.12497. Epub 2017 May 23.**	**Included**
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223.	Pereira LP, Köhler CA, de Sousa RT, Solmi M, de Freitas BP, Fornaro M, Machado-Vieira R, Miskowiak KW, Vieta E, Veronese N, Stubbs B, Carvalho AF. The relationship between genetic risk variants with brain structure and function in bipolar disorder: A systematic review of genetic-neuroimaging studies. Neurosci Biobehav Rev. 2017;79:87-109. doi: 10.1016/j.neubiorev.2017.05.002. Epub 2017 May 4.	Review
224.	Hibar DP, Westlye LT, Doan NT, Jahanshad N, Cheung JW, Ching CRK, Versace A, Bilderbeck AC, Uhlmann A, Mwangi B, Krämer B, Overs B, Hartberg CB, Abé C, Dima D, Grotegerd D, Sprooten E, Bøen E, Jimenez E, Howells FM, Delvecchio G, Temmingh H, Starke J, Almeida JRC, Goikolea JM, Houenou J, Beard LM, Rauer L, Abramovic L, Bonnin M, Ponteduro MF, Keil M, Rive MM, Yao N, Yalin N, Najt P, Rosa PG, Redlich R, Trost S, Hagenaars S, Fears SC, Alonso-Lana S, van Erp TGM, Nickson T, Chaim-Avancini TM, Meier TB, Elvsåshagen T, Haukvik UK, Lee WH, Schene AH, Lloyd AJ, Young AH, Nugent A, Dale AM, Pfennig A, McIntosh AM, Lafer B, Baune BT, Ekman CJ, Zarate CA, Bearden CE, Henry C, Simhandl C, McDonald C, Bourne C, Stein DJ, Wolf DH, Cannon DM, Glahn DC, Veltman DJ, Pomarol-Clotet E, Vieta E, Canales-Rodriguez EJ, Nery FG, Duran FLS, Busatto GF, Roberts G, Pearlson GD, Goodwin GM, Kugel H, Whalley HC, Ruhe HG, Soares JC, Fullerton JM, Rybakowski JK, Savitz J, Chaim KT, Fatjó-Vilas M, Soeiro-de-Souza MG, Boks MP, Zanetti MV, Otaduy MCG, Schaufelberger MS, Alda M, Ingvar M, Phillips ML, Kempton MJ, Bauer M, Landén M, Lawrence NS, van Haren NEM, Horn NR, Freimer NB, Gruber O, Schofield PR, Mitchell PB, Kahn RS, Lenroot R, Machado-Vieira R, Ophoff RA, Sarró S, Frangou S, Satterthwaite TD, Hajek T, Dannlowski U, Malt UF, Arolt V, Gattaz WF, Drevets WC, Caseras X, Agartz I, Thompson PM, Andreassen OA. Cortical abnormalities in bipolar disorder: an MRI analysis of 6503 individuals from the ENIGMA Bipolar Disorder Working Group. Version 2. Mol Psychiatry. 2018;23(4):932-942. doi: 10.1038/mp.2017.73. Epub 2017 May 2.	No gene
225.	**Soeiro-de-Souza MG, Lafer B, Moreno RA, Nery FG, Chile T, Chaim K, da Costa Leite C, Machado-Vieira R, Otaduy MC, Vallada H. The *CACNA1C* risk allele rs1006737 is associated with age-related prefrontal cortical thinning in bipolar I disorder. Transl Psychiatry. 2017;7(4):e1086. doi: 10.1038/tp.2017.57.**	**Included**
226.	Sugihara G, Kane F, Picchioni MM, Chaddock CA, Kravariti E, Kalidindi S, Rijsdijk F, Toulopoulou T, Curtis VA, McDonald C, Murray RM, McGuire P. Effects of risk for bipolar disorder on brain function: A twin and family study. Eur Neuropsychopharmacol. 2017;27(5):494-503. doi: 10.1016/j.euroneuro.2017.03.001. Epub 2017 Apr 6.	No gene
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228.	Nenadic I, Yotter RA, Dietzek M, Langbein K, Sauer H, Gaser C. Cortical complexity in bipolar disorder applying a spherical harmonics approach. Psychiatry Res Neuroimaging. 2017;263:44-47. doi: 10.1016/j.pscychresns.2017.02.007. Epub 2017 Feb 21.	No gene
229.	Wang T, Zhang X, Li A, Zhu M, Liu S, Qin W, Li J, Yu C, Jiang T, Liu B. Polygenic risk for five psychiatric disorders and cross-disorder and disorder-specific neural connectivity in two independent populations. Neuroimage Clin. 2017;14:441-449. doi: 10.1016/j.nicl.2017.02.011.	No BD
230.	Verkooijen S, Stevelink R, Abramovic L, Vinkers CH, Ophoff RA, Kahn RS, Boks MP, van Haren NE. The association of sleep and physical activity with integrity of white matter microstructure in bipolar disorder patients and healthy controls. Psychiatry Res Neuroimaging. 2017 Apr 30;262:71-80. doi: 10.1016/j.pscychresns.2017.01.013. Epub 2017 Feb 9.	No gene
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233.	Reus LM, Shen X, Gibson J, Wigmore E, Ligthart L, Adams MJ, Davies G, Cox SR, Hagenaars SP, Bastin ME, Deary IJ, Whalley HC, McIntosh AM. Association of polygenic risk for major psychiatric illness with subcortical volumes and white matter integrity in UK Biobank. Sci Rep. 2017;7:42140. doi: 10.1038/srep42140.	No BD
234.	Ohgidani M, Kato TA, Haraguchi Y, Matsushima T, Mizoguchi Y, Murakawa-Hirachi T, Sagata N, Monji A, Kanba S. Microglial CD206 gene has potential as a state marker of bipolar disorder. Front Immunol. 2017;7:676. doi: 10.3389/fimmu.2016.00676.	No imaging
235.	Luykx JJ, Broersen JL, de Leeuw M. The DRD2 rs1076560 polymorphism and schizophrenia-related intermediate phenotypes: A systematic review and meta-analysis. Neurosci Biobehav Rev. 2017;74(Pt A):214-224. doi: 10.1016/j.neubiorev.2017.01.006. Epub 2017 Jan 16.	Review

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237.	Nazeri A, Mulsant BH, Rajji TK, Levesque ML, Pipitone J, Stefanik L, Shahab S, Roostaei T, Wheeler AL, Chavez S, Voineskos AN. Gray Matter Neuritic Microstructure Deficits in Schizophrenia and Bipolar Disorder. Biol Psychiatry. 2017 Nov 15;82(10):726-736. doi: 10.1016/j.biopsych.2016.12.005. Epub 2016 Dec 8.	No gene
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239.	**Vai B, Riberto M, Poletti S, Bollettini I, Lorenzi C, Colombo C, Benedetti F. Catechol-O-methyltransferase Val(108/158)Met polymorphism affects fronto-limbic connectivity during emotional processing in bipolar disorder. Eur Psychiatry. 2017;41:53-59. doi: 10.1016/j.eurpsy.2016.10.002. Epub 2017 Feb 3.**	**Included**
240.	Tamminga CA, Pearlson GD, Stan AD, Gibbons RD, Padmanabhan J, Keshavan M, Clementz BA. Strategies for advancing disease definition using biomarkers and genetics: the Bipolar and Schizophrenia Network for Intermediate Phenotypes. Biol Psychiatry Cogn Neurosci Neuroimaging. 2017;2(1):20-27. doi: 10.1016/j.bpsc.2016.07.005. Epub 2016 Aug 2.	Review
241.	Roberts G, Lord A, Frankland A, Wright A, Lau P, Levy F, Lenroot RK, Mitchell PB, Breakspear M. Functional Dysconnection of the Inferior Frontal Gyrus in Young People With Bipolar Disorder or at Genetic High Risk. Biol Psychiatry. 2017 Apr 15;81(8):718-727. doi: 10.1016/j.biopsych.2016.08.018. Epub 2016 Aug 18.	No gene
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243.	**Bollettini I, Melloni EM, Aggio V, Poletti S, Lorenzi C, Pirovano A, Vai B, Dallaspezia S, Colombo C, Benedetti F. Clock genes associate with white matter integrity in depressed bipolar patients. Chronobiol Int. 2017;34(2):212-224. doi: 10.1080/07420528.2016.1260026. Epub 2016 Dec 20.**	**Included**
244.	Roberts G, Perry A, Lord A, Frankland A, Leung V, Holmes-Preston E, Levy F, Lenroot RK, Mitchell PB, Breakspear M. Structural dysconnectivity of key cognitive and emotional hubs in young people at high genetic risk for bipolar disorder. Mol Psychiatry. 2018;23(2):413-421. doi: 10.1038/mp.2016.216. Epub 2016 Dec 20.	No gene
245.	Sugranyes G, Solé-Padullés C, de la Serna E, Borras R, Romero S, Sanchez-Gistau V, Garcia-Rizo C, Goikolea JM, Bargallo N, Moreno D, Baeza I, Castro-Fornieles J. Cortical morphology characteristics of young offspring of patients with schizophrenia or bipolar disorder. J Am Acad Child Adolesc Psychiatry. 2017;56(1):79-88. doi: 10.1016/j.jaac.2016.09.516. Epub 2016 Oct 25.	No gene
246.	Wiggins JL, Brotman MA, Adleman NE, Kim P, Wambach CG, Reynolds RC, Chen G, Towbin K, Pine DS, Leibenluft E. Neural Markers in Pediatric Bipolar Disorder and Familial Risk for Bipolar Disorder. J Am Acad Child Adolesc Psychiatry. 2017;56(1):67-78. doi: 10.1016/j.jaac.2016.10.009. Epub 2016 Nov 2.	No gene
247.	Neilson E, Bois C, Gibson J, Duff B, Watson A, Roberts N, Brandon NJ, Dunlop J, Hall J, McIntosh AM, Whalley HC, Lawrie SM. Effects of environmental risks and polygenic loading for schizophrenia on cortical thickness. Schizophr Res. 2017;184:128-136. doi: 10.1016/j.schres.2016.12.011. Epub 2016 Dec 15.	Lumping
248.	Schroeder FA, Gilbert TM, Feng N, Taillon BD, Volkow ND, Innis RB, Hooker JM, Lipska BK. Expression of HDAC2 but not HDAC1 transcript is reduced in dorsolateral prefrontal cortex of patients with schizophrenia. ACS Chem Neurosci. 2017;8(3):662-668. doi: 10.1021/acschemneuro.6b00372. Epub 2016 Dec 13.	*In vitro*
249.	O'Donoghue S, Holleran L, Cannon DM, McDonald C. Anatomical dysconnectivity in bipolar disorder compared with schizophrenia: A selective review of structural network analyses using diffusion MRI. J Affect Disord. 2017;209:217-228. doi: 10.1016/j.jad.2016.11.015. Epub 2016 Nov 17.	Review
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252.	de la Serna E, Sugranyes G, Sanchez-Gistau V, Rodriguez-Toscano E, Baeza I, Vila M, Romero S, Sanchez-Gutierrez T, Penzol MJ, Moreno D, Castro-Fornieles J. Neuropsychological characteristics of child and adolescent offspring of patients with schizophrenia or bipolar disorder. Schizophr Res. 2017;183:110-115. doi: 10.1016/j.schres.2016.11.007. Epub 2016 Nov 12.	Unrelated
253.	Pagliaccio D, Wiggins JL, Adleman NE, Harkins E, Curhan A, Towbin KE, Brotman MA, Pine DS, Leibenluft E. Behavioral and neural sustained attention deficits in bipolar disorder and familial risk of bipolar disorder. Biol Psychiatry. 2017;82(9):669-678. doi: 10.1016/j.biopsych.2016.09.006. Epub 2016 Sep 16.	No gene
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255.	**Mallas E, Carletti F, Chaddock CA, Shergill S, Woolley J, Picchioni MM, McDonald C, Toulopoulou T, Kravariti E, Kalidindi S, Bramon E, Murray R, Barker GJ, Prata DP. The impact of CACNA1C gene, and its epistasis with ZNF804A, on white matter microstructure in health, schizophrenia and bipolar disorder[1]. Genes Brain Behav. 2017;16(4):479-488. doi: 10.1111/gbb.12355. Epub 2016 Nov 29.**	**Included**
256.	Tandon N, Nanda P, Padmanabhan JL, Mathew IT, Eack SM, Narayanan B, Meda SA, Bergen SE, Ruaño G, Windemuth A, Kocherla M, Petryshen TL, Clementz B, Sweeney J, Tamminga C, Pearlson G, Keshavan MS. Novel gene-brain	Lumping

	structure relationships in psychotic disorder revealed using parallel independent component analyses. Schizophr Res. 2017;182:74-83. doi: 10.1016/j.schres.2016.10.026. Epub 2016 Oct 24.	
257.	Frye MA, Ryu E, Nassan M, Jenkins GD, Andreazza AC, Evans JM, McElroy SL, Oglesbee D Jr, Highsmith WE, Biernacka JM. Mitochondrial DNA sequence data reveals association of haplogroup U with psychosis in bipolar disorder. J Psychiatr Res. 2017;84:221-226. doi: 10.1016/j.jpsychires.2016.09.027. Epub 2016 Sep 30.	No imaging
258.	Prendes-Alvarez S, Nemeroff CB. Personalized medicine: Prediction of disease vulnerability in mood disorders. Neurosci Lett. 2018;669:10-13. doi: 10.1016/j.neulet.2016.09.049. Epub 2016 Oct 13.	Review
259.	Romme IA, de Reus MA, Ophoff RA, Kahn RS, van den Heuvel MP. Connectome disconnectivity and cortical gene expression in patients with schizophrenia. Biol Psychiatry. 2017 Mar 15;81(6):495-502. doi: 10.1016/j.biopsych.2016.07.012. Epub 2016 Jul 27.	No BD
260.	Knöchel C, Kniep J, Cooper JD, Stäblein M, Wenzler S, Sarlon J, Prvulovic D, Linden DE, Bahn S, Stocki P, Ozcan S, Alves G, Carvalho AF, Reif A, Oertel-Knöchel V. Altered apolipoprotein C expression in association with cognition impairments and hippocampus volume in schizophrenia and bipolar disorder. Eur Arch Psychiatry Clin Neurosci. 2017;267(3):199-212. doi: 10.1007/s00406-016-0724-3. Epub 2016 Aug 22.	No gene
261.	Zarinabad N, Wilson M, Gill SK, Manias KA, Davies NP, Peet AC. Multiclass imbalance learning: Improving classification of pediatric brain tumors from magnetic resonance spectroscopy. Magn Reson Med. 2017;77(6):2114-2124. doi: 10.1002/mrm.26318. Epub 2016 Jul 12.	Unrelated
262.	Reich M, Girard E, Le Rhun E. Breast leptomeningeal metastasis recurrence presenting as a manic episode. Palliat Support Care. 2017;15(2):272-275. doi: 10.1017/S1478951516000456. Epub 2016 Jun 27.	Unrelated
263.	**Lippard ETC, Jensen KP, Wang F, Johnston JAY, Spencer L, Pittman B, Gelernter J, Blumberg HP. Effects of ANK3 variation on gray and white matter in bipolar disorder. Mol Psychiatry. 2017;22(9):1345-1351. doi: 10.1038/mp.2016.76. Epub 2016 May 31.**	**Included**
264.	**Tseng CJ, Gilbert TM, Catanese MC, Hightower BG, Peters AT, Parmar AJ, Kim M, Wang C, Roffman JL, Brown HE, Perlis RH, Zürcher NR, Hooker JM. In vivo human brain expression of histone deacetylases in bipolar disorder. Transl Psychiatry. 2020;10(1):224. doi: 10.1038/s41398-020-00911-5.**	**Included**
265.	Navarri X, Afzali MH, Lavoie J, Sinha R, Stein DJ, Momenan R, Veltman DJ, Korucuoglu O, Sjoerds Z, van Holst RJ, Hester R, Orr C, Cousijn J, Yucel M, Lorenzetti V, Wiers R, Jahanshad N, Glahn DC, Thompson PM, Mackey S, Conrod PJ. How do substance use disorders compare to other psychiatric conditions on structural brain abnormalities? A cross-disorder meta-analytic comparison using the ENIGMA consortium findings. Hum Brain Mapp. 2020 Jul 9. doi: 10.1002/hbm.25114.	Review
266.	Opel N, Goltermann J, Hermesdorf M, Berger K, Baune BT, Dannlowski U. Cross-disorder analysis of brain structural abnormalities in six major psychiatric disorders: a secondary analysis of mega- and meta-analytical findings from the ENIGMA Consortium. Biol Psychiatry. 2020 May 11:S0006-3223(20)31585-7. doi: 10.1016/j.biopsych.2020.04.027.	Review

Total output: 266 articles.
Included: 27
Excluded: 239
Reasons:

- No gene: 81
- Reviews (meta-analyses): 67
- No BD: 26
- Unrelated: 21
- No imaging: 11
- Lumping data of different patient populations, thus not allowing to infer on data of BD patients: 9
- Protocols: 5
- Case reports/series: 4
- Animal studies: 4
- Opinion papers (editorials, letters asking clarifications, hypotheses etc.): 3
- *In vitro*/*post mortem:* 3
- Unfocused: 2
- No reported brain area: 1
- No data: 1
- Correction to already reported article (errata corrige as self-standing record): 1

Supplementary results. Included studies analysed 25 different genes totalling 31 polymorphisms (Table 1); these were CACNA1C rs1006737 (five studies), CACNB2 rs11013860 (two studies), ZNF804A rs1344706 (three studies), ANK3 rs9804190 (one study), BDNF Val66Met (or rs6265 or G196A) (one study) COMT rs4680 (two studies), SNAP25 rs6039769 (one study), IL-1β rs16944 (one study), Homer rs7713917 (one study), 5-HT1A receptor promoter gene rs6295 (one study), CLOCK rs1801260 (one study), PER3 PER4 (one study), EAAT1 rs 231880 (one study) and EAAT2-181A>C (SLC1A2) rs4354668 (one study), and one study each investigated ADCY3 rs11676272, ASPDH rs7248272, CLN3 rs77595156, HNF4G rs1805098, LMO7 rs2241913, NREP rs11559, PRTN3 rs351111, TDRD7 rs2045732, KMT2C rs4639425, rs74483926, rs201834857, and rs138627563, OR51G1 rs1378739 and rs10836954, LIMCH1 rs2289342 and rs11734372. Five studies investigated polygenic risk scores (PGR) using data from six databases (Psychiatric GWAS Consortium Bipolar Disorder Working Group, 2011; Schizophrenia Psychiatric Genome-Wide Association Study (GWAS), 2011; Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Codd et al., 2013; Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014; Stahl et al., 2019).

White matter (WM)-DTI studies.

Some diffusion tensor imaging (DTI) studies analysed the relationship between genetic variants and white matter abnormalities in patients with BD.

FA reduction indicates a disorganisation of WM fibre directionality, which would be greater in patients with BD with genetic polymorphisms regarding the genes that follow suit.

A homozygotes with the *Homer* gene rs7713917 polymorphism show reduced FA in frontal WM tracts, particularly in the forceps minor, body of the corpus callosum (CC) including bilateral superior longitudinal fasciculus, in the cingulate zone, including its right hippocampal part, as well as in a series of left hemisphere regions, i.e., uncinate fasciculus (UF), inferior fronto-occipital fasciculus, anterior thalamic radiation, anterior corona radiate, and superior corona radiate (Benedetti *et al.,* 2018).

Reduced FA was found in individuals homozygous for PER3$^{4/4}$ of the *PER3* gene in bilateral anterior thalamic radiation, bilateral superior and inferior longitudinal fasciculi, bilateral cingulate gyrus, forceps major, splenium of the CC, and left fronto-occipital fasciculus, peaking in the inferior left fronto-occipital fasciculus (Bollettini *et al.,* 2017).

ANK3 rs9804190 T carriers showed decreased FA in the UF in ROI analysis, while whole-brain showed reduced FA in the right temporo-parietal region, left posterior cingulate and posterior CC/fornix (Lippard *et al.,* 2017). Furthermore, reduced FA was shown in in a series of WM tracts, like the left UF extending to the ventro-frontal WM and including the anterior cingulate and the CC, then the dorso-frontal WM including the forceps, and in the right UF extending to the ventro-frontal WM including the anterior cingulate and the CC, then also in the anterior branch of the internal capsule (Lippard *et al.,* 2017). Furthermore, FA is reduced in the frontal dorso-medial WM, including bilateral dorsal anterior cingulate, extending to the corona radiata and the external capsule; moreover, FA is reduced in the left temporo-parietal WM and in the posterior dorso-medial WM, including areas of the left dorsal cingulate, then the left parieto-occipital WM and finally the right parietal WM extending to the temporo-parietal WM (Lippard *et al.,* 2017).

So we may stress that in BD patients matching particular gene polymorphisms, some brain regions show decreased FA, namely, bilateral frontal lobe WM, over the entire cingulate gyrus, and the parietal and occipital lobes, as well as in some inner structures, like bilateral internal capsule.

One study reported **increased radial (RD) and mean diffusivity (MD)** in WM fibre tracts. The meaning is one of increased space between fibres, thus suggesting de- or dys-myelination.

Increased MD values have been shown in *CLOCK rs1801260* C carriers, while increased RD has been found in *PER3*$^{4/4}$ homozygotes (Bollettini *et al.,* 2017).

CLOCK rs1801260 C carriers show increased MD only in several WM regions that are all located in the left hemisphere and include UF, superior and inferior longitudinal fasciculus, anterior thalamic radiation, inferior fronto-occipital fasciculus, cortico-spinal tract, retrolenticular part of internal capsule, posterior and superior corona radiata, thalamic medullar laminae, with signal peaking in superior corona radiata and inferior fronto-occipital fasciculus (Bollettini *et al.,* 2017).

PER3$^{4/4}$ homozygotes show increased RD in right anterior thalamic radiation, bilateral inferior longitudinal fasciculus, right superior longitudinal fasciculus, splenium and body of the CC, right cortico-spinal tract, right

fronto-occipital fasciculus, and UF, with signal peaking in right inferior fronto-occipital fasciculus and right anterior thalamic radiation (Bollettini *et al.,* 2017).

Another study investigated the rôle of the *EAAT2-181A* > *C* (*SLC1A2* gene) (rs4354668) on WM integrity and found a complex interaction between childhood adversity and axial diffusivity (Poletti *et al.*, 2019); the risk G allele carriers had lower axial diffusivity than T/T homozygotes when exposed to high stress and higher diffusivity than T/T when exposed to low stress.

A recent study found no effect of a variety of risk genes on DTI measures in the whole brain and in cortical thickness (Han *et al.,* 2019). Another study that used a PGR score for schizophrenia and analysed whole brain DTI, found no effects of PGR on FA or MD in patients with BD, schizophrenia, their relatives and HC, and no genetic risk-diagnosis interaction (Simões *et al*., 2020).

Summarising, MD measure are increased in BD patients who are also risk gene carriers, mostly in left corona radiata and in left inferior fronto-occipital fasciculus. In BD, RD is increased mostly in the right hemisphere and particularly in inferior fronto-occipital fasciculus, thalamic radiation, and inferior longitudinal fasciculus. Indirect support for the involvement of risk genes in WM alterations in BD is provided by the fact that patients with BD with the risk variants of *CACNA1C* and *ANK3* genes have reduced ventral prefrontal activation and prefrontal-visual cortex effective connectivity while performing an emotional face recognition task, in contrast with HCs who are carriers of one of these risk genes, who show both increased ventral prefrontal activation and increased effective connectivity between ventral frontal and visual cortices (Dima *et al.,* 2013). However, when the effect of *CACNA1C* on WM was studied through DTI MRI in a BD population, no significant findings emerged (Mallas *et al.,* 2017). Also, there were no associations of either *ZNF804A* alone or *CACNA1C* in epistasis with *ZNF804A* with any WM alteration (Mallas *et al.,* 2017), thus confirming previous results (Mallas *et al.,* 2016).

Grey matter (GM)- fMRI and sMRI studies

Cortical GM

Different genes were reported to affect brain cortex structure and function in BD patients. Here we display results divided by cortical region, identifying three main clusters, i.e., frontal, associative, and occipital regions. For each group we first provide fMRI data, then sMRI

A study investigating whole brain effective connectivity with fRMI during a facial recognition paradigm as related to telomere length GWAS and found no relationship (Powell *et al.,* 2019). Similarly, no significant effects of diagnosis in a sMRI study investigating PGR for schizophrenia and for BD as related to cortical volume, thickness, and GM density (Doan *et al.,* 2017). Another whole brain study investigating PGR for autism spectrum and schizophrenia in patients with BD *vs*. MDD *vs*. HCs, found PGR-GM associations not to be driven by diagnosis (Ranlund *et al.,* 2018). Finally, another whole-brain study of cortical thickness investigating the effects of PGR score in BD patients from the Stahl *et al.* (2019) database and comparing BD patients with HC, found the PGR score to correlate with cortical thickness over a 6-yr follow-up in the entire BD/HC sample, but no significant correlation in the BD sample alone (Abé *et al.,* 2020).

Frontal and cingulate

fMRI. *CACNA1C* rs1006737 influenced frontocortical activity of BD patients during a verbal fluency task. In particular, A carriers showed increased activation in left middle and superior frontal gyri (Tecelão *et al.,* 2019). Furthermore, *ZNF804A* rs1344706 A carriers exhibited increased activation during a verbal fluency task in the left inferior frontal gyrus, in pars opercularis/pars triangularis (Broca's area) (Tecelão *et al.,* 2018). In spite of this, in BD patients there was no epistatic effect of *CACNA1C* rs1006737 with *ZNF804A* rs1344706 on activity of frontal cortex during verbal fluency task (Tecelão *et al.,* 2019).

Homer rs7713917 gene A homozygotes showed increased frontocortical activation during a face-matching task. Specifically, increased activation was observed in right dorso-anterior cingulate cortex and in right precentral gyrus in BD patients(Benedetti *et al.,* 2018). Furthermore, patients showed reduced volumes in left frontal cortex, in medial prefrontal region, especially in medial and superior frontal gyri (Benedetti *et al.,* 2018).

COMT rs4680 SNP Val/Val risk gene was associated with bilateral decreased DLPFC activation during a high-load working memory spatial task (2-back *vs*. 1-back) (Miskowiak *et al.*, 2017). This effect was evident during both ROI– (focused on DLPFC) and whole-brain–analyses.

sMRI. *IL-1β rs16944* gene carrier status in BD patients might influence the frontal cortex area, especially pars triangularis. T carriers showed increased pars triangularis surface area than C homozygotes. In contrast, HC T carriers showed a decreased surface area compared to C homozygotes (Shonibare *et al.,* 2020).

A homozygotes for the *Homer rs7713917* gene showed reduced volumes in left frontal and medial prefrontal regions, especially in medial and superior frontal gyri (Benedetti *et al.,* 2018).

One study found the *CACNA1C* rs1006737 risk allele to correlate with decreased cortical thickness of the left medial orbitofrontal cortex (OFC) (Soeiro-de-Souza *et al*., 2017), another with decreased cortical thickness in superior frontal, left lateral OFC, rostral anterior cingulate, right precentral, and right paracentral cortices and increased left paracentral cortex (Smedler et al., 2019), but a more recent study found no association between this risk allele and frontal cortical thickness in BD patients (Rodríguez-Ramírez *et al.,* 2020).

Another study tested the effect of the *CACNB2* rs11013860 risk allele (A carrier) on cortical thickness; BD A carriers showed reduced thickness in right superior frontal gyrus compared to HC A carriers and BD C homozygotes (non-risk) (J. Chen *et al*., 2020).

Parietal

sMRI. *CACNA1C* rs1006737 A carriers showed decreased cortical thickness in different regions of the parietal lobe compared to G homozygotes, including the precuneus, bilateral inferior parietal, and superior parietal cortices (Smedler et al., 2019).

Temporal

fMRI. *CACNA1C rs1006737* influenced temporal cortex activity during a verbal fluency task in BD patients. In particular, A carriers showed increased activity in bilateral middle temporal gyrus and in right superior temporal gyrus (Tecelão *et al.,* 2019).

Occipital

fMRI. *CACNA1C* rs1006737 influenced occipital cortex activity during a verbal fluency task in BD patients. In fact, A carriers showed increased activation only in the left hemisphere, at the level of the occipital gyrus, calcarine sulcus, and lingual gyrus (Tecelão *et al.,* 2019). During a picture-encoding task, a fMRI study examining BD-only patients and comparing *BDNF* Val66Met gene non-risk (Val/Val) to risk (Met carriers) on their activity in the lateral occipital cortex, found the non-risk to display higher activity in this area during task performance than the risk carriers (Hørlyck *et al.*, 2019).

sMRI. The *IL-1β* gene rs16944 polymorphism in BD patients might affect left lateral occipital cortex volume. In fact, T carriers show increased volume in left lateral occipital cortex than C homozygotes. This cortical region comprises the pericalcarine area and the inferior temporal area. In contrast, HC T carriers show decreased volumes in these regions, compared to C homozygotes (Shonibare *et al.,* 2020).

Subcortical GM

In the following section we provide results of studies that analysed gene effects on subcortical grey matter structure in BD populations. Findings were obtained using different neuroimaging techniques, i.e., fMRI and sMRI, and will be provided according to this order.

fMRI. *CACNA1C* gene rs1006737 A carriers showed increased activation in right thalamus during a verbal fluency task (Tecelão *et al.,* 2019). When *CACNA1C* rs1006737 was analysed in epistasis with *ZNF804A* rs1344706 A homozygote state, a greater activation was observed, during a verbal fluency task, in right thalamus, anterior cerebellum (in particular, in the vermis), and caudate nucleus (Tecelão *et al.,* 2019). However, this latter effect was reversed when *CACNA1C* rs1006737 was in epistasis with *ZNF804A* rs1344706 C carriers, so the same regions showed decreased activation than *CACNA1C* rs1006737 alone (Tecelão *et al.,* 2019).

sMRI. In both BD and HC *SNAP25* rs6039769 C homozygotes, ROI analysis (amygdala and hippocampus) showed increased amygdala volumes compared to A carriers, but no effect on the hippocampus. Increased amygdala volumes were restricted to males only (Houenou *et al.,* 2017).

In another study, *ANK3* rs9804190 T BD carriers showed decreased thalamic volume compared to C homozygotes (Lippard *et al.,* 2017). Still another, investigating whole brain telomere length in BD, focused on hippocampus; although associations betwixt telomere length, hippocampal volume and episodic memory were found and telomere length explained a considerable proportion of left and right hippocampal volume variance, no significant interaction between BD and telomere length emerged (Powell *et al.,* 2018).

Functional connectivity (FC)

In the last section we provide results of studies investigating FC between brain areas during fMRI in BD patients. Only three studies found significant effects; in particular, they focused on connectivity between the amygdala and the prefrontal cortex (PFC). These two areas are involved in emotional control and processing. Abnormalities in BD patients with specific SNPs were reported during both emotional tasks and resting state.

EAAT1 rs2731880 T homozygotes in BD patients showed a reduction in FC in the right hemisphere, between the amygdala and the anterior subgenual cingulate cortex during a face-matching task, then during emotional processing of faces (Poletti *et al.,* 2018).

In BD patients or HCs with *SNAP25* rs6039769 C homozygosis, increased FC is present between the amygdala and the ventro-medial PFC during resting state fMRI. This effect was significant only in the male population (Houenou *et al.,* 2017).

One study tested the effects of the *5-HT1A* receptor promoter gene polymorphism (rs6295) during a face-matching task on VLPFC-amygdalar FC and found a weak bilateral reduction of connectivity in risk G carriers, that disappeared after correcting for multiple comparisons (Vai *et al.*, 2017a).

Looking at the *COMT* rs4680 SNP, Val/Val homozygotes showed increased FC between left amygdala and right dorso-lateral PFC, as well as between the left amygdala and the right supramarginal gyrus during emotional processing of a face-matching task. In contrast, Met carriers showed decreased FC between left amygdala and right dorso-lateral PFC and no variation in FC between amygdala and supramarginal gyrus during a face-matching task (Vai *et al.,* 2017b).

One study used resting state fMRI to study the effect of the *CACNB2* rs11013860 risk allele (A carrier status) (F. Liu et al., 2019). A carriers showed reduced resting state FC between the hippocampus and the pars triangularis of the right inferior frontal gyrus than C homozygotes in both BD and HC; furthermore, BD C homozygotes displayed greater FC between these two areas compared to their HC counterparts.

A study that used whole brain fMRI to test effective connectivity during a facial recognition task, found no effect of group (BD *vs*. HC) on PGR telomere length affecting connectivity and facial affect activation (Powell *et al.,* 2019).

Epigenetic studies

The only epigenetic study investigated histone deacetylase expression during cognitive task performance with PET, using [^{11}C]Martinostat uptake as a tracer and had a combined whole-brain/ROI approach focusing especially on left amygdala and DLPFC; it showed lower histone deacetylase expression in right amygdala in BD, but the result was too weak to resist post hoc testing due to small sample size (Tseng *et al.,* 2020).

Summarising, we identified a tendency of studies investigating small samples to report positive results for their target genes, while PGR score-based studies, using large databases, produced negative results.

References refer to the original references of the paper.