query: dc.description = "autism" OR dc.subject = "autism" OR dc.title = "autism" AND prism.publicationDate > "2017-11-01"

Records found: 4 | << Back to Search

Records

Position: 1 Schema: PRISM Aggregator Message

dc:identifier	doi:10.1038/npp.2017.195
dc:title	Modeling Non-Syndromic Autism with Human-Induced Pluripotent Stem Cells
dc:creator	Luke AD Bury
dc:creator	Anthony Wynshaw-Boris
prism:productCode	npp
prism:publicationName	Neuropsychopharmacology
prism:issn	0893-133X
prism:elssn	1740-634X
prism:doi	10.1038/npp.2017.195
dc:publisher	Nature Publishing Group
prism:publicationDate	2017-12-01
prism:volume	43
prism:number	1
prism:startingPage	219
prism:endingPage	220
prism:url	http://dx.doi.org/10.1038/npp.2017.195
dc:subject	Cell biology
dc:subject	Diseases
dc:description	Genetically, autism can broadly be segregated into syndromic and non-syndromic forms. Accounting for a small percentage of total ASD cases, syndromic ASD includes incidences of the disease with known genetic cause and unique clinical presentation, while non-syndromic ASD with unknown genetic etiology accounts for the remaining majority of ASD cases (Sztainberg and Zoghbi, 2016).
prism:copyright	© 2018 American College of Neuropsychopharmacology
prism:aggregationType	issue

Position: 2 Schema: PRISM Aggregator Message

dc:identifier	doi:10.1038/pr.2017.254
dc:title	Cell therapy for diverse central nervous system disorders: inherited metabolic diseases and autism
dc:creator	Jessica M Sun
dc:creator	Joanne Kurtzberg
prism:productCode	pr
prism:publicationName	Pediatric Research
prism:issn	0031-3998
prism:elssn	1530-0447
prism:doi	10.1038/pr.2017.254
dc:publisher	Nature Publishing Group
prism:publicationDate	2017-11-08
prism:url	http://dx.doi.org/10.1038/pr.2017.254
prism:genre	Reviews
dc:description	The concept of utilizing human cells for the treatment of medical conditions is not new. In its simplest form, blood product transfusion as treatment of severe hemorrhage has been practiced since the 1800s. The advent of hematopoietic stem cell transplantation (HSCT) began with the development of bone marrow transplantation for hematological malignancies in the mid-1900s and is now the standard of care for many hematological disorders. In the past few decades, HSCT has expanded to additional sources of donor cells, a wider range of indications, and the development of novel cell products. This trajectory has sparked a rapidly growing interest in the pursuit of innovative cell therapies to treat presently incurable diseases, including neurological conditions. HSCT is currently an established therapy for certain neurologically devastating inherited metabolic diseases, in which engrafting donor cells provide lifelong enzyme replacement that prevents neurological deterioration and significantly extends the lives of affected children. Knowledge gained from the treatment of these rare conditions has led to refinement of the indications and timing of HSCT, the study of additional cellular products and techniques to address its limitations, and the investigation of cellular therapies without transplantation to treat more common neurological conditions, such as autism spectrum disorder. Pediatric Research advance online publication, 8 November 2017; doi:10.1038/pr.2017.254
prism:copyright	© 2017 International Pediatric Research Foundation, Inc.
prism:aggregationType	aop

Position: 3 Schema: PRISM Aggregator Message

dc:identifier	doi:10.1038/mp.2017.213
dc:title	Identifying specific prefrontal neurons that contribute to autism-associated abnormalities in physiology and social behavior
dc:creator	A C Brumback
dc:creator	I T Ellwood
dc:creator	C Kjaerby
dc:creator	J Iafrati
dc:creator	S Robinson
dc:creator	A T Lee
dc:creator	T Patel
dc:creator	S Nagaraj
dc:creator	F Davatolhagh
dc:creator	V S Sohal
prism:productCode	mp
prism:publicationName	Molecular Psychiatry
prism:issn	1359-4184
prism:elssn	1476-5578
prism:doi	10.1038/mp.2017.213
dc:publisher	Nature Publishing Group
prism:publicationDate	2017-11-07
prism:url	http://dx.doi.org/10.1038/mp.2017.213
dc:subject	Neuroscience
dc:subject	Autism spectrum disorders
prism:genre	Research
dc:description	Functional imaging and gene expression studies both implicate the medial prefrontal cortex (mPFC), particularly deep-layer projection neurons, as a potential locus for autism pathology. Here, we explored how specific deep-layer prefrontal neurons contribute to abnormal physiology and behavior in mouse models of autism. First, we find that across three etiologically distinct models—in utero valproic acid (VPA) exposure, CNTNAP2 knockout and FMR1 knockout—layer 5 subcortically projecting (SC) neurons consistently exhibit reduced input resistance and action potential firing. To explore how altered SC neuron physiology might impact behavior, we took advantage of the fact that in deep layers of the mPFC, dopamine D2 receptors (D2Rs) are mainly expressed by SC neurons, and used D2-Cre mice to label D2R+ neurons for calcium imaging or optogenetics. We found that social exploration preferentially recruits mPFC D2R+ cells, but that this recruitment is attenuated in VPA-exposed mice. Stimulating mPFC D2R+ neurons disrupts normal social interaction. Conversely, inhibiting these cells enhances social behavior in VPA-exposed mice. Importantly, this effect was not reproduced by nonspecifically inhibiting mPFC neurons in VPA-exposed mice, or by inhibiting D2R+ neurons in wild-type mice. These findings suggest that multiple forms of autism may alter the physiology of specific deep-layer prefrontal neurons that project to subcortical targets. Furthermore, a highly overlapping population—prefrontal D2R+ neurons—plays an important role in both normal and abnormal social behavior, such that targeting these cells can elicit potentially therapeutic effects. Molecular Psychiatry advance online publication, 7 November 2017; doi:10.1038/mp.2017.213
prism:copyright	© 2017 Macmillan Publishers Limited, part of Springer Nature.
prism:aggregationType	aop

Position: 4 Schema: PRISM Aggregator Message

dc:identifier	doi:10.1038/npp.2017.274
dc:title	Brain-Behavior Participant Similarity Networks Among Youth and Emerging Adults with Schizophrenia Spectrum, Autism Spectrum, or Bipolar Disorder and Matched Controls
dc:creator	Laura Stefanik
dc:creator	Lauren Erdman
dc:creator	Stephanie H Ameis
dc:creator	George Foussias
dc:creator	Benoit H Mulsant
dc:creator	Tina Behdinan
dc:creator	Anna Goldenberg
dc:creator	Lauren J O'Donnell
dc:creator	Aristotle N Voineskos
prism:productCode	npp
prism:publicationName	Neuropsychopharmacology
prism:issn	0893-133X
prism:elssn	1740-634X
prism:doi	10.1038/npp.2017.274
dc:publisher	Nature Publishing Group
prism:publicationDate	2017-11-06
prism:url	http://dx.doi.org/10.1038/npp.2017.274
dc:subject	Cognitive neuroscience
dc:subject	Biomarkers
dc:subject	Schizophrenia
dc:subject	Autism spectrum disorders
dc:subject	Bipolar disorder
prism:genre	Research
dc:description	There is considerable heterogeneity in social cognitive and neurocognitive performance among

	people with schizophrenia spectrum disorders (SSD), autism spectrum disorders (ASD), bipolar disorder (BD), and healthy individuals. This study used Similarity Network Fusion (SNF), a novel data-driven approach, to identify participant similarity networks based on relationships among demographic, brain imaging, and behavioral data. T1-weighted and diffusion-weighted magnetic resonance images were obtained for 174 adolescents and young adults (aged 16–35 years) with an SSD (n=51), an ASD without intellectual disability (n=38), euthymic BD (n=34), and healthy controls (n=51). A battery of social cognitive and neurocognitive tasks were administered. Data integration, cluster determination, and biological group formation were then obtained using SNF. We identified four new groups of individuals, each with distinct neural circuit-cognitive profiles. The most influential variables driving the formation of the new groups were robustly reliable across embedded resampling techniques. The data-driven groups showed considerably greater differentiation on key social and neurocognitive circuit nodes than groups generated by diagnostic analyses or dimensional social cognitive analyses. The data-driven groups were validated through functional outcome and brain network property measures not included in the SNF model. Cutting across diagnostic boundaries, our approach can effectively identify new groups of people based on a profile of neuroimaging and behavioral data. Our findings bring us closer to disease subtyping that can be leveraged toward the targeting of specific neural circuitry among participant subgroups to ameliorate social cognitive and neurocognitive deficits.</p> Neuropsychopharmacology advance online publication, 6 December; doi: 10.1038/npp.2017.274
prism:copyright	
prism:aggregationType	aop