

# Brandon C Mckinney

## List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/2712533/publications.pdf>

Version: 2024-02-01

20  
papers

1,141  
citations

471509

17  
h-index

752698

20  
g-index

21  
all docs

21  
docs citations

21  
times ranked

1767  
citing authors

#	ARTICLE	IF	CITATIONS
1	DNA methylation in the human frontal cortex reveals a putative mechanism for age-by-disease interactions. <i>Translational Psychiatry</i> , 2019, 9, 39.	4.8	16
2	Density of small dendritic spines and microtubule-associated-protein-2 immunoreactivity in the primary auditory cortex of subjects with schizophrenia. <i>Neuropsychopharmacology</i> , 2019, 44, 1055-1061.	5.4	27
3	DNA methylation age is not accelerated in brain or blood of subjects with schizophrenia. <i>Schizophrenia Research</i> , 2018, 196, 39-44.	2.0	41
4	DNA methylation as a putative mechanism for reduced dendritic spine density in the superior temporal gyrus of subjects with schizophrenia. <i>Translational Psychiatry</i> , 2017, 7, e1032-e1032.	4.8	28
5	DNA methylation evidence against the accelerated aging hypothesis of schizophrenia. <i>NPJ Schizophrenia</i> , 2017, 3, 13.	3.6	36
6	A novel mouse model of the aged brain: Over-expression of the L-type voltage-gated calcium channel Ca V 1.3. <i>Behavioural Brain Research</i> , 2017, 322, 241-249.	2.2	14
7	Hypermethylation of BDNF and SST Genes in the Orbital Frontal Cortex of Older Individuals: A Putative Mechanism for Declining Gene Expression with Age. <i>Neuropsychopharmacology</i> , 2015, 40, 2604-2613.	5.4	24
8	The Age-by-Disease Interaction Hypothesis of Late-Life Depression. <i>American Journal of Geriatric Psychiatry</i> , 2013, 21, 418-432.	1.2	58
9	Age-by-disease biological interactions: implications for late-life depression. <i>Frontiers in Genetics</i> , 2012, 3, 237.	2.3	17
10	The Age-by-Disease Interaction Hypothesis of Late-Life Depression. <i>American Journal of Geriatric Psychiatry</i> , 2012, , 1.	1.2	5
11	Deletion of the L-type calcium channel Ca <sub>v</sub> 1.3 but not Ca <sub>v</sub> 1.2 results in a diminished sAHP in mouse CA1 pyramidal neurons. <i>Hippocampus</i> , 2011, 21, 133-141.	1.9	45
12	Impaired long-term potentiation and enhanced neuronal excitability in the amygdala of Ca <sub>v</sub> 1.3 knockout mice. <i>Neurobiology of Learning and Memory</i> , 2009, 92, 519-528.	1.9	51
13	Decreased locomotor activity in mice expressing tTA under control of the CaMKII $\alpha$ promoter. <i>Genes, Brain and Behavior</i> , 2008, 7, 203-213.	2.2	29
14	Exaggerated emotional behavior in mice heterozygous null for the sodium channel <i>Scn8a</i> (Na <sub>v</sub> 1.6). <i>Genes, Brain and Behavior</i> , 2008, 7, 629-638.	2.2	44
15	Conditional forebrain deletion of the L-type calcium channel Ca <sub>v</sub> 1.2 disrupts remote spatial memories in mice. <i>Learning and Memory</i> , 2008, 15, 1-5.	1.3	112
16	L-type voltage-gated calcium channels in conditioned fear: A genetic and pharmacological analysis. <i>Learning and Memory</i> , 2008, 15, 326-334.	1.3	40
17	Hippocampal pyramidal cells in adult <i>Fmr1</i> knockout mice exhibit an immature-appearing profile of dendritic spines. <i>Brain Research</i> , 2006, 1084, 158-164.	2.2	168
18	The L-Type voltage-gated calcium channel Cav1.3 mediates consolidation, but not extinction, of contextually conditioned fear in mice. <i>Learning and Memory</i> , 2006, 13, 584-589.	1.3	83

#	ARTICLE	IF	CITATIONS
19	Dendritic spine abnormalities in the occipital cortex of C57BL/6Fmr1 knockout mice. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 2005, 136B, 98-102.	1.7	153
20	Experience effects on brain development: possible contributions to psychopathology. Journal of Child Psychology and Psychiatry and Allied Disciplines, 2003, 44, 33-63.	5.2	147