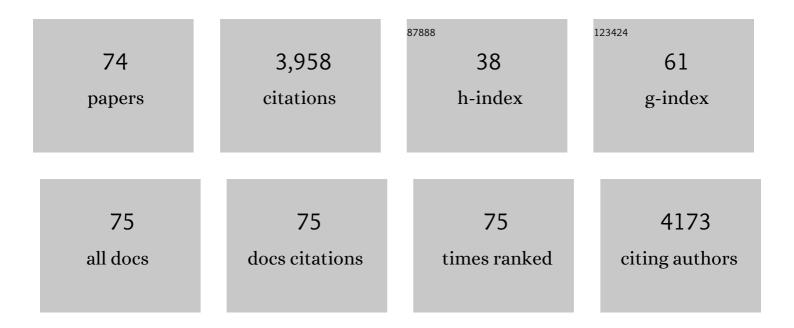
## Steven R Laviolette

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Identification of a novel fatty acid binding protein-5-CB2 receptor-dependent mechanism regulating anxiety behaviors in the prefrontal cortex. Cerebral Cortex, 2023, 33, 2470-2484.	2.9	4
2	Functional interactions between cannabinoids, omegaâ€3 fatty acids, and peroxisome proliferatorâ€activated receptors: Implications for mental health pharmacotherapies. European Journal of Neuroscience, 2022, 55, 1088-1100.	2.6	8
3	Anxiety and cognitive-related effects of Δ 9-tetrahydrocannabinol (THC) are differentially mediated through distinct GSK-3 vs. Akt-mTOR pathways in the nucleus accumbens of male rats. Psychopharmacology, 2022, 239, 509-524.	3.1	1
4	Adolescent nicotine induces depressive and anxiogenic effects through ERK 1â€2 and Aktâ€GSKâ€3 pathways and neuronal dysregulation in the nucleus accumbens. Addiction Biology, 2021, 26, e12891.	2.6	11
5	THC and CBD produce divergent effects on perception and panic behaviours via distinct cortical molecular pathways. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 2021, 104, 110029.	4.8	14
6	Molecular and neuronal mechanisms underlying the effects of adolescent nicotine exposure on anxiety and mood disorders. Neuropharmacology, 2021, 184, 108411.	4.1	26
7	l-Theanine Prevents Long-Term Affective and Cognitive Side Effects of Adolescent Δ-9-Tetrahydrocannabinol Exposure and Blocks Associated Molecular and Neuronal Abnormalities in the Mesocorticolimbic Circuitry. Journal of Neuroscience, 2021, 41, 739-750.	3.6	9
8	Could Cannabidiol Be a Treatment for Coronavirus Disease-19-Related Anxiety Disorders?. Cannabis and Cannabinoid Research, 2021, 6, 7-18.	2.9	18
9	Exploring the impact of adolescent exposure to cannabinoids and nicotine on psychiatric risk: insights from translational animal models. Psychological Medicine, 2021, 51, 940-947.	4.5	7
10	In Utero Exposure to Δ9-Tetrahydrocannabinol Leads to Postnatal Catch-Up Growth and Dysmetabolism in the Adult Rat Liver. International Journal of Molecular Sciences, 2021, 22, 7502.	4.1	14
11	Reversing the Psychiatric Effects of Neurodevelopmental Cannabinoid Exposure: Exploring Pharmacotherapeutic Interventions for Symptom Improvement. International Journal of Molecular Sciences, 2021, 22, 7861.	4.1	8
12	Deciphering midbrain mechanisms underlying prepulse inhibition of startle. Progress in Neurobiology, 2020, 185, 101734.	5.7	26
13	Prenatal Cannabinoid Exposure: Emerging Evidence of Physiological and Neuropsychiatric Abnormalities. Frontiers in Psychiatry, 2020, 11, 624275.	2.6	32
14	Adolescent Nicotine Exposure Induces Dysregulation of Mesocorticolimbic Activity States and Depressive and Anxiety-like Prefrontal Cortical Molecular Phenotypes Persisting into Adulthood. Cerebral Cortex, 2019, 29, 3140-3153.	2.9	36
15	The Bivalent Rewarding and Aversive properties of Δ9-tetrahydrocannabinol are Mediated Through Dissociable Opioid Receptor Substrates and Neuronal Modulation Mechanisms in Distinct Striatal Sub-Regions. Scientific Reports, 2019, 9, 9760.	3.3	20
16	Cannabidiol Counteracts the Psychotropic Side-Effects of Δ-9-Tetrahydrocannabinol in the Ventral Hippocampus through Bidirectional Control of ERK1–2 Phosphorylation. Journal of Neuroscience, 2019, 39, 8762-8777.	3.6	52
17	Δ-9-Tetrahydrocannabinol and Cannabidiol produce dissociable effects on prefrontal cortical executive function and regulation of affective behaviors. Neuropsychopharmacology, 2019, 44, 817-825.	5.4	40
18	Fear Memory Recall Potentiates Opiate Reward Sensitivity through Dissociable Dopamine D1 versus D4 Receptor-Dependent Memory Mechanisms in the Prefrontal Cortex. Journal of Neuroscience, 2018, 38, 4543-4555.	3.6	16

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19	Phytocannabinoids modulate emotional memory processing through interactions with the ventral hippocampus and mesolimbic dopamine system: implications for neuropsychiatric pathology. Psychopharmacology, 2018, 235, 447-458.	3.1	26
20	The Role of Cholinergic Midbrain Neurons in Startle and Prepulse Inhibition. Journal of Neuroscience, 2018, 38, 8798-8808.	3.6	47
21	Deltaâ€9â€ŧetrahydrocannabinol potentiates fear memory salience through functional modulation of mesolimbic dopaminergic activity states. European Journal of Neuroscience, 2018, 47, 1385-1400.	2.6	10
22	Effects of Adolescent THC Exposure on the Prefrontal GABAergic System: Implications for Schizophrenia-Related Psychopathology. Frontiers in Psychiatry, 2018, 9, 281.	2.6	43
23	Adolescent Nicotine Exposure Induces Molecular and Neuronal Features of Depressive Disorder in Adulthood in the Mesolimbic Dopamine System. FASEB Journal, 2018, 32, 783.6.	0.5	Ο
24	Adolescent Cannabinoid Exposure Induces a Persistent Sub-Cortical Hyper-Dopaminergic State and Associated Molecular Adaptations in the Prefrontal Cortex. Cerebral Cortex, 2017, 27, bhv335.	2.9	72
25	Bi-directional cannabinoid signalling in the basolateral amygdala controls rewarding and aversive emotional processing via functional regulation of the nucleus accumbens. Addiction Biology, 2017, 22, 1218-1231.	2.6	19
26	Palmitoylethanolamide Modulates GPR55 Receptor Signaling in the Ventral Hippocampus to Regulate Mesolimbic Dopamine Activity, Social Interaction, and Memory Processing. Cannabis and Cannabinoid Research, 2017, 2, 8-20.	2.9	44
27	Neuronal and molecular effects of cannabidiol on the mesolimbic dopamine system: Implications for novel schizophrenia treatments. Neuroscience and Biobehavioral Reviews, 2017, 75, 157-165.	6.1	71
28	Opiate exposure state controls dopamine D3 receptor and cdk5/calcineurin signaling in the basolateral amygdala during reward and withdrawal aversion memory formation. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 2017, 79, 59-66.	4.8	17
29	Adolescent THC Exposure Causes Enduring Prefrontal Cortical Disruption of GABAergic Inhibition and Dysregulation of Sub-Cortical Dopamine Function. Scientific Reports, 2017, 7, 11420.	3.3	91
30	Cannabinoid reward and aversion effects in the posterior ventral tegmental area are mediated through dissociable opiate receptor subtypes and separate amygdalar and accumbal dopamine receptor substrates. Psychopharmacology, 2017, 234, 2325-2336.	3.1	8
31	Cannabinoid regulation of opiate motivational processing in the mesolimbic system: the integrative roles of amygdala, prefrontal cortical and ventral hippocampal input pathways. Current Opinion in Behavioral Sciences, 2017, 13, 46-54.	3.9	4
32	Seeing through the smoke: Human and animal studies of cannabis use and endocannabinoid signalling in corticolimbic networks. Neuroscience and Biobehavioral Reviews, 2017, 76, 380-395.	6.1	28
33	Cannabidiol Counteracts Amphetamine-Induced Neuronal and Behavioral Sensitization of the Mesolimbic Dopamine Pathway through a Novel mTOR/p70S6 Kinase Signaling Pathway. Journal of Neuroscience, 2016, 36, 5160-5169.	3.6	106
34	What Can Rats Tell Us about Adolescent Cannabis Exposure? Insights from Preclinical Research. Canadian Journal of Psychiatry, 2016, 61, 328-334.	1.9	52
35	Cannabidiol Modulates Fear Memory Formation Through Interactions with Serotonergic Transmission in the Mesolimbic System. Neuropsychopharmacology, 2016, 41, 2839-2850.	5.4	55
36	Opiate Exposure State Controls a D2-CaMKIIα-Dependent Memory Switch in the Amygdala-Prefrontal Cortical Circuit. Neuropsychopharmacology, 2016, 41, 847-857.	5.4	21

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37	Cannabinoid Transmission in the Hippocampus Activates Nucleus Accumbens Neurons and Modulates Reward and Aversion-Related Emotional Salience. Biological Psychiatry, 2016, 80, 216-225.	1.3	41
38	Molecular and neuronal plasticity mechanisms in the amygdala-prefrontal cortical circuit: implications for opiate addiction memory formation. Frontiers in Neuroscience, 2015, 9, 399.	2.8	23
39	Hippocampal Cannabinoid Transmission Modulates Dopamine Neuron Activity: Impact on Rewarding Memory Formation and Social Interaction. Neuropsychopharmacology, 2015, 40, 1436-1447.	5.4	54
40	The Role of Cannabinoid Transmission in Emotional Memory Formation: Implications for Addiction and Schizophrenia. Frontiers in Psychiatry, 2014, 5, 73.	2.6	63
41	Dopamine Receptor Blockade Modulates the Rewarding and Aversive Properties of Nicotine via Dissociable Neuronal Activity Patterns in the Nucleus Accumbens. Neuropsychopharmacology, 2014, 39, 2799-2815.	5.4	21
42	NMDA Receptor Blockade in the Prelimbic Cortex Activates the Mesolimbic System and Dopamine-Dependent Opiate Reward Signaling. Psychopharmacology, 2014, 231, 4669-4679.	3.1	22
43	Endogenous Opioid-Induced Neuroplasticity of Dopaminergic Neurons in the Ventral Tegmental Area Influences Natural and Opiate Reward. Journal of Neuroscience, 2014, 34, 8825-8836.	3.6	46
44	Cannabinoid Transmission in the Prefrontal Cortex Bi-Phasically Controls Emotional Memory Formation via Functional Interactions with the Ventral Tegmental Area. Journal of Neuroscience, 2014, 34, 13096-13109.	3.6	55
45	The effects of AMPA receptor blockade in the prelimbic cortex on systemic and ventral tegmental area opiate reward sensitivity. Psychopharmacology, 2013, 225, 687-695.	3.1	23
46	Opiate Exposure and Withdrawal Induces a Molecular Memory Switch in the Basolateral Amygdala between ERK1/2 and CaMKIIα-Dependent Signaling Substrates. Journal of Neuroscience, 2013, 33, 14693-14704.	3.6	49
47	Supra-normal stimulation of dopamine D1 receptors in the prelimbic cortex blocks behavioral expression of both aversive and rewarding associative memories through a cyclic-AMP-dependent signaling pathway. Neuropharmacology, 2013, 67, 104-114.	4.1	17
48	Natural and Drug Rewards Act on Common Neural Plasticity Mechanisms with ΔFosB as a Key Mediator. Journal of Neuroscience, 2013, 33, 3434-3442.	3.6	100
49	Cannabinoid Transmission in the Prelimbic Cortex Bidirectionally Controls Opiate Reward and Aversion Signaling through Dissociable Kappa Versus μ-Opiate Receptor Dependent Mechanisms. Journal of Neuroscience, 2013, 33, 15642-15651.	3.6	47
50	Early versus Late-Phase Consolidation of Opiate Reward Memories Requires Distinct Molecular and Temporal Mechanisms in the Amygdala-Prefrontal Cortical Pathway. PLoS ONE, 2013, 8, e63612.	2.5	23
51	Phasic D1 and tonic D2 dopamine receptor signaling double dissociate the motivational effects of acute nicotine and chronic nicotine withdrawal. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 3101-3106.	7.1	110
52	Dopamine D4 Receptor Transmission in the Prefrontal Cortex Controls the Salience of Emotional Memory via Modulation of Calcium Calmodulin-Dependent Kinase II. Cerebral Cortex, 2012, 22, 2486-2494.	2.9	19
53	Inactivation of the basolateral amygdala during opiate reward learning disinhibits prelimbic cortical neurons and modulates associative memory extinction. Psychopharmacology, 2012, 222, 645-661.	3.1	27
54	Inputs from the basolateral amygdala to the nucleus accumbens shell control opiate reward magnitude via differential dopamine D1 or D2 receptor transmission. European Journal of Neuroscience, 2012, 35, 279-290.	2.6	49

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55	NMDA Receptor Hypofunction in the Prelimbic Cortex Increases Sensitivity to the Rewarding Properties of Opiates via Dopaminergic and Amygdalar Substrates. Cerebral Cortex, 2011, 21, 68-80.	2.9	50
56	Identification of a Dopamine Receptor-Mediated Opiate Reward Memory Switch in the Basolateral Amygdala–Nucleus Accumbens Circuit. Journal of Neuroscience, 2011, 31, 11172-11183.	3.6	75
57	Cannabinoid Transmission in the Basolateral Amygdala Modulates Fear Memory Formation via Functional Inputs to the Prelimbic Cortex. Journal of Neuroscience, 2011, 31, 5300-5312.	3.6	90
58	Acquisition, Extinction, and Recall of Opiate Reward Memory Are Signaled by Dynamic Neuronal Activity Patterns in the Prefrontal Cortex. Cerebral Cortex, 2011, 21, 2665-2680.	2.9	43
59	Integrated Cannabinoid CB1 Receptor Transmission within the Amygdala-Prefrontal Cortical Pathway Modulates Neuronal Plasticity and Emotional Memory Encoding. Cerebral Cortex, 2010, 20, 1486-1496.	2.9	72
60	Dopamine D4-receptor modulation of cortical neuronal network activity and emotional processing: Implications for neuropsychiatric disorders. Behavioural Brain Research, 2010, 208, 12-22.	2.2	54
61	Dopamine D1 versus D4 Receptors Differentially Modulate the Encoding of Salient versus Nonsalient Emotional Information in the Medial Prefrontal Cortex. Journal of Neuroscience, 2009, 29, 4836-4845.	3.6	58
62	Chronic nicotine exposure switches the functional role of mesolimbic dopamine transmission in the processing of nicotine's rewarding and aversive effects. Neuropharmacology, 2009, 56, 741-751.	4.1	40
63	Dopamine Signaling through D <sub>1</sub> -Like versus D <sub>2</sub> -Like Receptors in the Nucleus Accumbens Core versus Shell Differentially Modulates Nicotine Reward Sensitivity. Journal of Neuroscience, 2008, 28, 8025-8033.	3.6	49
64	Dopamine Modulation of Emotional Processing in Cortical and Subcortical Neural Circuits: Evidence for a Final Common Pathway in Schizophrenia?. Schizophrenia Bulletin, 2007, 33, 971-981.	4.3	138
65	Cannabinoids Potentiate Emotional Learning Plasticity in Neurons of the Medial Prefrontal Cortex through Basolateral Amygdala Inputs. Journal of Neuroscience, 2006, 26, 6458-6468.	3.6	166
66	A Subpopulation of Neurons in the Medial Prefrontal Cortex Encodes Emotional Learning with Burst and Frequency Codes through a Dopamine D4 Receptor-Dependent Basolateral Amygdala Input. Journal of Neuroscience, 2005, 25, 6066-6075.	3.6	218
67	GABAA receptors signal bidirectional reward transmission from the ventral tegmental area to the tegmental pedunculopontine nucleus as a function of opiate state. European Journal of Neuroscience, 2004, 20, 2179-2187.	2.6	38
68	DREAM ablation selectively alters THC place aversion and analgesia but leaves intact the motivational and analgesic effects of morphine. European Journal of Neuroscience, 2004, 19, 3033-3041.	2.6	36
69	Opiate state controls bi-directional reward signaling via GABAA receptors in the ventral tegmental area. Nature Neuroscience, 2004, 7, 160-169.	14.8	203
70	The neurobiology of nicotine addiction: bridging the gap from molecules to behaviour. Nature Reviews Neuroscience, 2004, 5, 55-65.	10.2	381
71	The motivational valence of nicotine in the rat ventral tegmental area is switched from rewarding to aversive following blockade of the α7-subunit-containing nicotinic acetylcholine receptor. Psychopharmacology, 2003, 166, 306-313.	3.1	97
72	Motivational state determines the functional role of the mesolimbic dopamine system in the mediation of opiate reward processes. Behavioural Brain Research, 2002, 129, 17-29.	2.2	90

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73	Lesions of the Tegmental Pedunculopontine Nucleus Block the Rewarding Effects and Reveal the Aversive Effects of Nicotine in the Ventral Tegmental Area. Journal of Neuroscience, 2002, 22, 8653-8660.	3.6	89
74	GABA <sub>A</sub> receptors in the ventral tegmental area control bidirectional reward signalling between dopaminergic and nonâ€dopaminergic neural motivational systems. European Journal of Neuroscience, 2001, 13, 1009-1015.	2.6	121