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## BIOGRAPHICAL SKETCH

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NAME Joshua W. Callahan		POSITION TITLE Postdoctoral Fellow	
eRA COMMONS USER NAME (credential, e.g., agency login) JCALLAHAN			
EDUCATION/TRAINING <i>(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)</i>			
INSTITUTION AND LOCATION	DEGREE <i>(if applicable)</i>	MM/YY	FIELD OF STUDY
Western Washington University	B.A.	06/05	Biopsychology
Rutgers, The State University of New Jersey	Ph.D.	09/07-09/13	Neuroscience
Northwestern University	Postdoc.	10/13- Present	Neuroscience

### A. Personal Statement

Broadly, my research encompasses understanding the functional organization of the basal ganglia and limbic system (and their target structures) at the circuit level. I am interested in examining the complex mechanisms that mediate synaptic transmission within these neural networks in the context of normal and pathological states. This is fundamental for understanding the cellular dysfunction underlying – and possible therapies for – diseases like Parkinson's disease and Huntington's disease.

### B. Positions and Honors

#### Positions and Employment

2005-2007 Research associate II, Efficacy Pharmacology, MDS Pharma Services, Inc., Bothell, WA

#### Other Experience and Professional Memberships

2005- Member, American Association for Laboratory Animal Science  
2009- Member, Society for Neuroscience  
2010- Member, International Basal Ganglia Society  
2010- Member, New York Academy of Sciences  
2012- Member, American Association for the Advancement of Science

#### Academic and Professional Honors

2008 Travel Fellowship Award, Hereditary Disease Foundation Symposium  
2009 Young Investigator Award, Gordon Research Conference on CAG Triplet Repeat Disorders  
2010 Award for best student/postdoctoral oral presentation, Rutgers University Minisymposium  
2010 Travel Fellowship Award, 10<sup>th</sup> Triennial Meeting of the International Basal Ganglia Society  
2011 Award for best student/postdoctoral poster presentation, Rutgers University Minisymposium  
2013 Award for best student/postdoctoral oral presentation, Rutgers University Minisymposium

### C. Selected Publications

#### Peer-reviewed Publications

1. **Callahan, JW**, Abercrombie, ED. *In vivo* dopamine efflux is decreased in striatum of both fragment (R6/2) and full-length (YAC128) transgenic mouse models of Huntington's disease. *Frontiers in Systems Neuroscience*. 2011, 5: 61. PMID: PMC3139944
2. Farrar, AM, **Callahan, JW**, Abercrombie, ED. Reduced striatal acetylcholine efflux in the R6/2 mouse model of Huntington's disease: an examination of the role of altered inhibitory and excitatory mechanisms. *Experimental Neurology*. 2011, 232(2): 119-125.

3. Basso, JC, **Callahan, JW**, Farrar, AM, Morrel, JI. Voluntary lifelong activity in the rat alters monoamine content in brain regions with motor and motivational functions, monoamine responses to acute cocaine challenge, and cocaine-seeking behavior. (*In review*)
4. Farrar, AM, **Callahan, JW**, Abercrombie, ED. Prefrontal cortical monoamine release is reduced in the R6/2 mouse model of Huntington's disease. (*In preparation*)
5. **Callahan, JW**, Abercrombie, ED. Relationship between cortical EEG and STN neuronal activity is altered in the R6/2 mouse model of Huntington's disease. (*In preparation*)

#### **Abstracts (2008-present)**

1. **Callahan, JW**, Abercrombie, ED. *In vivo* analysis of nigrostriatal dopamine function and behavior in multiple mouse models of Huntington's disease. Hereditary Disease Foundation Symposium, Cambridge, MA, 2008.
2. **Callahan, JW**, Farrar, AM, Abercrombie, ED. Altered extracellular release of dopamine and serotonin in the YAC128 mouse model of Huntington's disease. Gordon Research Conference (CAG Triplet Repeat Disorders), Waterville Valley, NH, 2009.
3. **Callahan, JW**, Farrar, AM, Abercrombie, ED. Decreased efflux of *in vivo* dopamine and serotonin in striatum of the YAC128 mouse model of Huntington's disease. Society for Neuroscience Conference, Chicago, IL, 2009.
4. Farrar, AM, **Callahan, JW**, Abercrombie, ED. Abercrombie, ED. Striatal acetylcholine is altered in the R6/2 mouse model of Huntington's disease. Hereditary Disease Foundation Symposium, Cambridge, MA, 2010.
5. **Callahan, JW**, Abercrombie, ED. Cortical EEG and subthalamic unit activity *in vivo* in the R6/2 mouse model of Huntington's disease. Hereditary Disease Foundation Symposium, Cambridge, MA, 2010.
6. Farrar, AM, **Callahan, JW**, Abercrombie, ED. Striatal acetylcholine is altered in the R6/2 mouse model of Huntington's disease. International Basal Ganglia Society Conference, Long Branch, NJ, 2010.
7. **Callahan, JW**, Abercrombie, ED. Cortical EEG and subthalamic unit activity *in vivo* in the R6/2 mouse model of Huntington's disease. International Basal Ganglia Society Conference, Long Branch, NJ, 2010.
8. Farrar, AM, **Callahan, JW**, Abercrombie, ED. Monoamine release dynamics are altered in medial prefrontal cortex in the R6/2 mouse model of Huntington's disease. Society for Neuroscience Conference, Washington, DC, 2011.
9. Basso, JC, **Callahan, JW**, Farrar, AM, Abercrombie, ED, Morrell, JI. Voluntary wheel running throughout rearing in the rat alters baseline monoamine content and content levels in response to cocaine in brain regions that mediate motivation. Society for Neuroscience Conference, Washington, DC, 2011.
10. **Callahan, JW**, Farrar, AM, Abercrombie, ED. Cortical EEG and subthalamic unit activity *in vivo* in the YAC128 mouse model of Huntington's disease. Society for Neuroscience Conference, Washington, DC, 2011.
11. **Callahan, JW**, Abercrombie, ED. Comparison of nigrostriatal synaptic function across both fragment and full-length transgenic mouse models of Huntington's disease. Society for Neuroscience Conference, New Orleans, LA, 2012.