

**BIOGRAPHICAL SKETCH**

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NAME: Alison V. Roland

eRA COMMONS USER NAME (credential, e.g., agency login): AVR3KNIH

POSITION TITLE: Research Assistant Professor

EDUCATION/TRAINING (*Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.*)

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
University of Maryland, College Park, MD	B.S.	05/2003	Biology
University of Virginia, Charlottesville, VA	Ph.D.	12/2010	Pharmacology
University of Pennsylvania, Philadelphia, PA	Post-doc	06/2012	Neuroscience
University of North Carolina, Chapel Hill, NC	Post-doc	01/2025	Alcohol Studies

**A. Personal Statement**

My research investigates the neurobiological mechanisms underlying alcohol use disorder (AUD), with a focus on how stress and affective dysregulation contribute to harmful drinking behaviors. I have a broad neuroscience background studying the contribution of sex hormones, stress, and alcohol to neuronal and circuit function in models of disease. In my first postdoctoral position, I examined the intersection of stress and neuroinflammation in the regulation of affective behaviors via serotonergic pathways. Following a hiatus from laboratory work to focus on family commitments, I joined the Kash lab in October 2019 funded by an NIH Reentry Supplement. My work in the Kash lab focuses on how chronic alcohol drives plasticity in brain circuits to promote pathological drinking behavior. Here I led a collaborative project employing whole-brain imaging and network analysis that identified the cortical amygdala (CoA) as a critical hub in the alcohol withdrawal circuitry. The CoA is implicated in drinking behavior and several other psychiatric disorders but is a largely unexplored brain region. As a neural hub, the CoA can drive large-scale changes in brain activity and thus is uniquely poised as a potential target for therapeutics. I aim to integrate assessments of large-scale brain activity with molecular, surgical, and pharmacological manipulations in mice, enabling improved understanding of brain dysfunction in AUD.

1. **Roland AV\***, Coelho CAO\*, Haun HL, Gianessi CA, Lopez MF, D'Ambrosio S, Machinski SN, Kroenke CD, Frankland PW, Becker HC, Kash TL. Alcohol Dependence Modifies Brain Networks Activated During Withdrawal and Reaccess: A c-Fos-Based Analysis in Mice. *Biol Psychiatry*. 2023 Sep 1;94(5):393-404. PMID: 36736419.
2. **Roland AV**, Harry Chao TH, Hon OJ, Machinski SN, Sides TR, Lee SI, Ian Shih YY, Kash TL. Acute and chronic alcohol modulation of extended amygdala calcium dynamics. *Alcohol*. 2024 May;116:53-64. doi: 10.1016/j.alcohol.2024.02.004. PMID: 38423261.
3. Xiao T, **Roland A**, Chen Y, Guffey S, Kash T, Kimbrough A. A role for circuitry of the cortical amygdala in excessive alcohol drinking, withdrawal, and alcohol use disorder. *Alcohol*. 2024 Dec;121:151-159. doi: 10.1016/j.alcohol.2024.02.008. PMID: 38447789.

**B. Positions, Scientific Appointments, and Honors**

## Positions

2025 – present	Research Assistant Professor, Psychiatry, Bowles Center for Alcohol Studies, University of North Carolina
2019 – 2025	Postdoctoral Research Associate, University of North Carolina, Chapel Hill, NC
2017 – 2018	Technical Writer, United States Pharmacopeia, Rockville, MD
2016 – 2019	Freelance Scientific Writer/Editor, Various
2010 – 2012	Postdoctoral Researcher, University of Pennsylvania, Philadelphia, PA
2004 – 2010	Graduate Student Researcher, University of Virginia, Charlottesville, VA
2003 – 2003	Research Assistant, Neuroendocrinology, Walter Reed Army Institute of Research, Silver Spring, MD
2001 – 2003	Physical Sciences Aide, Naval Research Laboratory, Washington, DC

## Honors

2009	Travel Award, Endocrine Society
2008	Travel Award, International Symposium on Neural Sex Differences
2003	Gemstone Citation for Team Research, University of Maryland
2001	Outstanding Service Award, Naval Research Laboratory
2001	Honors Program Citation, University of Maryland
1999 – 2003	National Merit Scholarship
1999 – 2003	Banneker/Key Full Scholarship, University of Maryland

## Professional Memberships

2024—present	Member, Society for Biological Psychiatry
2022 – present	Member, Research Society on Alcoholism
2007 – present	Member, Society for Neuroscience
2017 – 2018	Member, American Medical Writers Association
2011 – 2012	Member, Penn Postdoctoral Editors Association
2006 – 2010	Member, Endocrine Society

## C. Contributions to Science

### 1. Modulation of brain-wide networks and extended amygdala circuitry by chronic alcohol

As a postdoc in the Kash lab, I led a project examining whole-brain networks activated during alcohol withdrawal. Utilizing a cutting-edge brain clearing technique and light-sheet microscopy, I acquired a snapshot of c-Fos activation throughout the entire mouse brain 24 hours after binge alcohol consumption, and immediately after a reaccess drinking session, in mice made dependent by chronic intermittent ethanol exposure. Through a collaboration with the Frankland Lab at the University of Toronto, we were able to leverage this large dataset to identify important neural hubs activated during alcohol withdrawal. We then validated one of these hubs, the cortical amygdala, via chemogenetic interrogation, solidifying a novel role for this brain region in alcohol-dependent drinking behavior. This work was published in *Biological Psychiatry*. I also spearheaded a project using fiber photometry to study neuronal activity dynamics in the extended amygdala during acute alcohol consumption, and how chronic alcohol exposure alters these dynamics. I identified dynorphin as a key subpopulation participating in acute responses to alcohol and other salient consummatory stimuli, and I demonstrated that these responses were significantly modulated by stimulus novelty. I then collaborated with the Shih lab to examine coherence between the bed nucleus of the stria terminalis and central amygdala during alcohol drinking bouts. This work was published in *Alcohol*. I am currently following up on these observations using a CeA-specific kappa-opioid receptor knockdown combined with simultaneous GCaMP recording in BNST and CeA to determine how interregional coherence is altered in concert with behavioral changes produced by this manipulation, such as reduced drinking.

- a. **Roland AV\***, Coelho CAO\*, Haun HL, Gianessi CA, Lopez MF, D'Ambrosio S, Machinski SN, Kroenke CD, Frankland PW, Becker HC, Kash TL. Alcohol Dependence Modifies Brain Networks Activated During Withdrawal and Reaccess: A c-Fos-Based Analysis in Mice. *Biol Psychiatry*. 2023 Sep 1;94(5):393-404. PMID: 36736419.

- b. **Roland AV**, Harry Chao TH, Hon OJ, Machinski SN, Sides TR, Lee SI, Ian Shih YY, Kash TL. Acute and chronic alcohol modulation of extended amygdala calcium dynamics. *Alcohol*. 2024 May;116:53-64. doi: 10.1016/j.alcohol.2024.02.004. PMID: 38423261.
- c. Xiao T, **Roland A**, Chen Y, Guffey S, Kash T, Kimbrough A. A role for circuitry of the cortical amygdala in excessive alcohol drinking, withdrawal, and alcohol use disorder. *Alcohol*. 2024 Dec;121:151-159. doi: 10.1016/j.alcohol.2024.02.008. PMID: 38447789.

## 2. Behavioral effects of CRF and neuroinflammation in the dorsal raphe nucleus

As a postdoctoral researcher in the Bale lab, I investigated sex differences in the role of the stress peptide corticotropin-releasing factor (CRF) and its type 1 receptor (CRF1) in the dorsal raphe nucleus (DRN), a major serotonergic nucleus involved in stress and affect regulation. My work demonstrated that infusion of CRF or a CRF1-selective antagonist into the DRN had sex-specific effects on anxiety- and depression-like behavior driven by differential activation of neuronal subpopulations in the dorsal raphe. This work revealed potential molecular substrates underlying sex differences in mood disorders and culminated in a co-first author manuscript in *Biological Psychiatry*. I also tested the hypothesis that the link between neuroinflammation and behavior may be mediated by serotonergic neurocircuitry by inducing localized neuroinflammation in the DRN through viral-mediated proinflammatory IL-1 $\beta$  overexpression. This manipulation induced a profound behavioral phenotype characterized by increased motor activity and risk-taking, providing evidence that neuroinflammatory processes acting specifically on serotonergic circuits might contribute to the pathophysiology of manic behavior.

- a. Howerton AR\*, **Roland AV\***, Fluharty JM, Marshall A, Chen A, Daniels D, Beck SG, Bale TL. Sex differences in corticotropin-releasing factor receptor-1 action within the dorsal raphe nucleus in stress responsivity. *Biol Psychiatry*. 2014 Jun 1;75(11):873-83. PubMed PMID: 24289884; PubMed Central PMCID: PMC3997756.
- b. Howerton AR, **Roland AV**, Bale TL. Dorsal raphe neuroinflammation promotes dramatic behavioral stress dysregulation. *J Neurosci*. 2014 May 21;34(21):7113-23. PubMed PMID: 24849347; PubMed Central PMCID: PMC4028491.

## 3. Metabolic-Reproductive interactions in polycystic ovary syndrome

My graduate work focused on polycystic ovary syndrome (PCOS), the most common cause of infertility in women. PCOS is characterized by abnormal patterns of gonadotropin-releasing hormone (GnRH) secretion and metabolic abnormalities. I used a mouse model to demonstrate that prenatal androgenization of females, a putative pathogenic mechanism for PCOS, programs impaired reproductive cyclicity in part by increasing the activity of GnRH neurons. These studies provided evidence for a previously unknown mechanism for a common PCOS therapeutic, metformin, which simultaneously restored reproductive cyclicity and reduced GnRH neuron hyperactivity in this model. In a related line of inquiry examining the link between reproduction and metabolic substrate availability, I demonstrated that GnRH neuronal activity is modulated by the extracellular glucose concentration in brain slices, and that glucosensing is mediated by adenosine monophosphate-activated protein kinase (AMPK). This work established AMPK as a novel molecular link between metabolism and the central control of fertility. These findings have been confirmed and extended by in vivo studies in other labs demonstrating that AMPK mediates the suppressive effects of short-term food deprivation on the reproductive axis. I authored five manuscripts based on this work, including two review articles.

- a. **Roland AV**, Moenter SM. Regulation of gonadotropin-releasing hormone neurons by glucose. *Trends Endocrinol Metab*. 2011 Nov;22(11):443-9. PubMed PMID: 21855365; PubMed Central PMCID: PMC3205187.
- b. **Roland AV**, Moenter SM. Glucosensing by GnRH neurons: inhibition by androgens and involvement of AMP-activated protein kinase. *Mol Endocrinol*. 2011 May;25(5):847-58. PubMed PMID: 21393446; PubMed Central PMCID: PMC3082325.

- c. **Roland AV**, Moenter SM. Prenatal androgenization of female mice programs an increase in firing activity of gonadotropin-releasing hormone (GnRH) neurons that is reversed by metformin treatment in adulthood. *Endocrinology*. 2011 Feb;152(2):618-28. PubMed PMID: 21159854; PubMed Central PMCID: PMC3037157.
- d. **Roland AV**, Nunemaker CS, Keller SR, Moenter SM. Prenatal androgen exposure programs metabolic dysfunction in female mice. *J Endocrinol*. 2010 Nov;207(2):213-23. PubMed PMID: 20713501; PubMed Central PMCID: PMC3612271.

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