OMB No. 3145-0279 (Expiration Date 10/31/2026)

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Coleman, Leon Garland Garland

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-1693-3799

Position Title: Assistant Professor

Organization and Location: University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina, United States	DOCTOR OF PHILOSOPHY	06/2005	08/2010	Neurobiology
University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina, United States	DOCTOR OF MEDICINE	06/2003	05/2012	Medicine (MD/PhD program)
University of Virginia, Charlottesville, Virginia, United States	BACHELOR OF SCIENCE	09/1999	05/2003	Chemical Engineering

Appointments and Positions

2020 - present Assistant Professor, University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina, United States
2017 - 2020 Research Assistant Professor, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
2014 - 2017 Research Associate, UNIV OF NORTH CAROLINA CHAPEL HILL, Chapel Hill, NC, USA

Products

Products Closely Related to the Proposed Project

1. Barnett AM, McNair EM, Dawkins L, Zou J, Nikolova VD, Moy SS, Sutherland GT, Stevens J, Colie M, Katemboh K, Kellner H, Ho K, Damian C, DeCastro S, Vetreno RP, Coleman LG Jr. Loss of lysosomal acid lipase contributes to Alzheimer's disease pathology and cognitive decline. Alzheimers Dement. 2025 Jul;21(7):e70486. PubMed Central PMCID: PMC12271982.
2. McNair EM, Dawkins LW, Materia B, Ross G, Barnett A, Nakkala P, Qin L, Zou J, Nikolova V, Moy S, Coleman LG Jr. Microglia Promote Neurodegeneration and Hyperkatifeia during Withdrawal and Abstinence from Binge Alcohol. Am J Pathol. 2026 Jan;196(1):306-325. PubMed Central PMCID: PMC12799517.
3. Zou J, McNair E, DeCastro S, Lyons SP, Mordant A, Herring LE, Vetreno RP, Coleman LG Jr. Microglia either promote or restrain TRAIL-mediated excitotoxicity caused by Aβ(1-42) oligomers. J Neuroinflammation. 2024 Sep 1;21(1):215. PubMed Central PMCID: PMC11367981.
4. Barnett A, David E, Rohlman A, Nikolova VD, Moy SS, Vetreno RP, Coleman LG Jr. Adolescent Binge Alcohol Enhances Early Alzheimer's Disease Pathology in Adulthood Through Proinflammatory Neuroimmune Activation. Front Pharmacol. 2022;13:884170. PubMed Central PMCID: PMC9086457.
5. Coleman LG Jr, Zou J, Crews FT. Microglial-derived miRNA let-7 and HMGB1 contribute to ethanol-induced neurotoxicity via TLR7. J Neuroinflammation. 2017 Jan 25;14(1):22. PubMed Central PMCID: PMC5264311.

Other Significant Products Highlighting Contributions to Science

1. Coleman LG Jr, He J, Lee J, Styner M, Crews FT. Adolescent binge drinking alters adult brain neurotransmitter gene expression, behavior, brain regional volumes, and neurochemistry in mice. Alcohol Clin Exp Res. 2011 Apr;35(4):671-88. PubMed Central PMCID: PMC3544413.
2. Coleman LG Jr, Zou J, Crews FT. Microglial depletion and repopulation in brain slice culture normalizes sensitized proinflammatory signaling. J Neuroinflammation. 2020 Jan 18;17(1):27. PubMed Central PMCID: PMC6969463.

3. Zou J, Walter TJ, Barnett A, Rohlman A, Crews FT, Coleman LG Jr. Ethanol Induces Secretion of Proinflammatory Extracellular Vesicles That Inhibit Adult Hippocampal Neurogenesis Through G9a/GLP-Epigenetic Signaling. Front Immunol. 2022;13:866073. PubMed Central PMCID: PMC9136051.
4. Willis ML, Mahung C, Wallet SM, Barnett A, Cairns BA, Coleman LG Jr, Maile R. Plasma extracellular vesicles released after severe burn injury modulate macrophage phenotype and function. J Leukoc Biol. 2022 Jan;111(1):33-49. PubMed Central PMCID: PMC8716518.
5. Coleman LG Jr, Zou J, Qin L, Crews FT. HMGB1/IL-1β complexes regulate neuroimmune responses in alcoholism. Brain Behav Immun. 2018 Aug;72:61-77. PubMed Central PMCID: PMC5932292.

Certification:

I certify that the information provided is current, accurate, and complete. This includes, but is not limited to, information related to current, pending, and other support (both foreign and domestic) as defined in 42 U.S.C. § 6605.

In accordance with Section 10632 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19232), each individual identified as a senior/key person must certify that they are not a party to a malign foreign talent recruitment program.

Research Security Training Requirement for Federal Award Personnel: In accordance with Section 10634 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19234), each individual identified as a senior/key person must certify that they have completed the requisite research security training that meets the requirements specified in Item 2 of Important Notice No. 149 within 12 months prior to proposal submission.

Misrepresentations and/or omissions may be subject to prosecution and liability pursuant to, but not limited to, 18 U.S.C. §§287, 1001, 1031 and 31 U.S.C. §§3729-3733 and 3802.

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NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Coleman, Leon Garland Garland

Persistent Identifier (PID) of the Senior/Key Person: https://orcid.org/0000-0003-1693-3799

Position Title: Assistant Professor

Organization and Location: University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina, United States

Personal Statement

My scientific goal is to identify novel therapeutic targets for chronic diseases. I have witnessed first-hand the merciless effects cancer, Alzheimer's disease, addiction and other conditions have on families – including my own. I have made discoveries regarding the long-term effects of adolescent alcohol on adult neurobiology as well as immune and epigenetic mechanisms underlying pathology in alcohol use disorder, Alzheimer's disease, and trauma. I discovered that adolescent binge alcohol causes long-lasting deficits in basal forebrain cholinergic neurons and cognitive flexibility (Coleman et al 2011) as well as an important role of EHMT/G9a epigenetic reprogramming in alcohol neuropathology (Zou et al 2022). We have found that microglia and Toll-like receptor ligands play key roles in alcohol use disorder, with microglia promoting the long-lasting negative affect seen in alcohol use disorder (Coleman et al 2017, Coleman et al 2018, Coleman 2020, and McNair et al 2026). We identified an important role for extracellular vesicles in alcohol use disorder and severe burn injury (Willis et al 2022 and Zou et al 2022), and recently identified lysosomal acid lipase as a promising therapeutic target for Alzheimer's disease (Barnett et al 2025). Our mission is to identify novel therapeutic targets for these diseases by employing multiple in vivo, in vitro and human new approach methodologies

I have been funded by NIH to study cellular and molecular mechanisms that contribute to both central and peripheral inflammatory processes. This includes an R01 studying the contribution of alcohol abuse to Alzheimer's pathology, an R21 studying the effect of alcohol on anti-PD1 cancer immunotherapy efficacy, an R21 studying the role of microglia in neuronal metabolic dysfunction, and a P60 component as well as a U54 component studying neuroinflammation in alcohol use disorder. I am experienced in studying pharmacology and immune biology using a broad array of technical approaches including: Proteomics, microscopy, cell culture, extracellular vesicle assays, animal models, immunohistochemistry, molecular biochemical assays (e.g. RT-PCR, ELISA, and Western Blot, proteomics), immunological assays (e.g. flow cytometry, nanostring), electron microscopy, and MRI. I have completed a K08 on the central and peripheral immune effects of alcohol and completed the MD/PhD program at the University of North Carolina at Chapel Hill. During that time, I received and completed an F30 Ruth L. Kirschstein NRSA award for MD/PhD students investigating the effects of alcohol on brain development during the third trimester and adolescence.

Honors

2026	Keynote Speaker, SYNAPSE2026
2025	Keynote Speaker, Gordon Research Seminar on Alcohol
2025	Neuropharmacology Early Career Investigator Award, American Society of Pharmacology and Experimental Therapeutics
2025	Woods Early Career Investigator Award, University of North Carolina at Chapel Hill School of Medicine
2024	Distinguished Local Speaker, Triangle Society for Neuroscience
2023	Early Career Travel Award, Winter Brain Conference on Brain Research
2017	Early Career Investigator Showcase Award, NIDA/NIAAA
2017 - 2022	K08 Clinical Scientist Research Career Development Award, NIAAA
2016	Junior Investigator Award, International Society on Brain Research on Alcohol/European Society on Brain Research on Alcohol
2008	F30 Ruth L. Kirschstein National Research Service Award , NIAAA

Contributions to Science

1. Proinflammatory Signaling Promotes Neuropathology and Behavioral Dysfunction in Alcohol Use Disorder. Neuroimmune activation plays a key role in the pathology of alcohol use disorder. We found that alcohol causes long-lasting proinflammatory polarization of microglia that drives innate immune signaling in brain (McNair et al 2026, American Journal of Pathology). This directly contributes to pathologic features such as neurodegeneration and negative affect. Formation of regenerative microglia is promising intervention, restoring aspects of alcohol-induced neuroinflammation (Coleman et al 2020, Journal of Neuroinflammation). Together, these findings have warrant investigation into therapeutic strategies targeting neuroimmune signaling for AUD.

2. Lysosomal Dysfunction and Proinflammatory Signaling Promote the Progression of Alzheimer's Disease (AD). We have reported that lysosome dysfunction and neuroimmune activation play key roles in the pathology of Alzheimer's disease. Specifically, we recently identified lysosomal acid lipase (LAL) as an important contributor to AD progression. LAL is lost in AD, which promotes lysosomal dysfunction and AD proteopathy. Gene replacement of LAL was protective, improving cognitive function and reducing AD neuropathological progression. Thus, LAL represents a novel preventative and therapeutic target for AD. The development of LAL-PET enables the possibility of a precision medicine approach, wherein individuals with abnormally low LAL are identified as at-risk for AD and stratified to preventative treatment. We have further found that microglia are pertinent regulators of AD pathology, with regenerative phenotypes protecting against neurotoxicity. Modifiable AD risk exposures such as alcohol misuse or midlife obesity were found to enhance AD pathology through neuroimmune and lysosomal mechanisms. Relevant products are in the "Products Closely Related to this Project" section.

3. Extracellular Microvesicle signaling mediates pathology in alcohol use disorder. Extracellular microvesicles (MVs) have emerged as mediators of innate immune dysfunction. We found that MVs released in response to ethanol are enriched in damage-associated molecular pattern molecules (DAMPs) such as HMGB1 and miRNA let-7b (Coleman et al 2017, Journal of Neuroinflammation). These DAMPs are endogenous agonists for endosomal immune TLRs (HMGB1-TLR4, let-7b-TLR7). Further, we found that MVs are critical mediators of alcohol-induced neuro-inflammation and loss of adult hippocampal neurogenesis, with blockade of their secretion preventing immune responses (Zou et al 2022 Frontiers in Pharmacology). Thus, MVs may represent novel therapeutic targets for neuroinflammation.

4. Extracellular microvesicles contribute to immune dysfunction with trauma and sepsis. Extracellular microvesicles (MVs) have emerged as mediators of innate immune dysfunction. We found that MVs released in response to burn injury and other trauma are enriched in damage-associated molecular pattern molecules (DAMPs) such as HMGB1 and IL-1 beta that promote immune dysfunction after injury. Surprisingly, MVs from burned subjects reproduce much of the immune dysfunction associated with burn in naive subjects and immune cells. This implicates MVs as novel therapeutic targets after severe trauma.

5. Persistent effects of Alcohol on Brain Development During Adolescence and Late Pregnancy. Alcohol abuse during the adolescent period in particular is associated with increased risk of developing Alcohol Use Disorder (AUD) in adulthood. We reported that binge alcohol during adolescence causes long-lasting brain cellular, structural, and behavioral deficits that create a persistent neuro-environment that is vulnerable to addiction and neurodegeneration (Coleman et al 2011, ACER). Further, we found that ethanol treatment during the analog of the human third trimester causes persistent reductions in adult brain volume, frontal cortical neuron number and adult neurogenesis. This may proceed through the NMDA antagonism effect of ethanol. These findings show that adolescence and the third trimester of pregnancy are 'danger periods' for alcohol use. Both local and national media covered the findings from these projects.

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