Using RePORT to Your Advantage as a PIO

Nicole J. Garbarini, Ph.D.
Media & Communications Specialist
NIH Office of Extramural Research

September 27, 2016
What is RePORT?

Website and database that provides access to reports, data and analyses on NIH supported research activities

Launched in 2008

New features continually implemented since then

Past 25 years of project data available!

http://report.nih.gov
How can this help public information officers?

- Find current and historical information about NIH funding to your institution
- Monitor awards to your institution through saved search alerts
- Increase press release reach and visibility of scientific experts
- Find key facts, statistics, and figures that can be used in your outreach
Project Information

Abstract Text:

DESCRIPTION (provided by applicant): The amygdala plays a critical role in the genesis of defensive behaviors. Moreover, it is hyperactive in humans afflicted with anxiety disorders. Thus, it is commonly believed that many anxiety disorders result, at least in part, from a dysregulation of amygdala processes normally mediating fear or defensive behaviors. Accordingly, research on the mechanisms controlling amygdala excitability might open new approaches for the treatment of anxiety disorders. This proposal aims to tap into the potential of the bilaterally projecting midline thalamic (MTh) nuclei on the amygdala. Prior studies on thalamic influences over the amygdala have focused on inputs arising from the posterior thalamus, particularly from the medial portion of the medial geniculate nucleus. Yet, a number of tracing studies have revealed that MTh nuclei also contribute massive projections to the basolateral (BLA) and central (CeA) amygdala. However, other than anatomically, little is known about the role of these strong glutamatergic inputs. The work proposed here aims to shed light on the influence of MTh inputs to the amygdala. To this end, we will first identify the targets and postsynaptic mechanisms of MTh inputs in the amygdala using anatomical (Aim #1) and physiological (Aim #2) methods. Indeed, BLA and CeA both contain multiple cell types that express different peptides/receptors and form contrasting connections with each other and extrinsic afferents. Therefore, in Aim #1, we will combine anterograde tracing with immunocytochemistry for various neuronal markers to identify the targets of MTh axon terminals in the amygdala at the light and electron microscopic levels. Building on these results, Aim #2 will combine optogenetic and patch-clamp recording techniques in vitro to study the impact of MTh inputs on amygdala cells. Armed with this information, the last two aims will examine the influence of MTh cells on amygdala-dependent functions. Indeed, recent studies have revealed that following muscimol infusions in MTh nuclei, the expression of amygdala-dependent learned and innate fear is drastically reduced. However, it is unclear whether these muscimol findings result from the inhibition of nearby thalamic cells (e.g., mediodorsal nucleus), or the disinhibition of cell targets of MTh nuclei (e.g., prefrontal cortex), that project to the amygdala. Two different approaches will be used to address this question. First, in Aim #3, we will perform simultaneously extracellular recordings of MTh and amygdala cells during the expression of learned and innate fear. Next, in Aim #4, we will use a dual viral strategy allowing us to express halorhodopsin or channelrhodopsin, but only in MTh cells that project to the amygdala. We will then optogenetically inhibit or excite amygdala-projecting MTh cells and examine this effect on amygdala-dependent tasks that probe learned or innate fear. Together, the experiments proposed here will reveal how MTh neurons regulate the excitability of the amygdala during the expression of learned and innate fear. This knowledge will pave the way for pharmacological interventions aiming to regulate the activity of midline thalamic cells by taking advantage of their unusual profile of receptor expression.

Public Health Relevance Statement:

PUBLIC HEALTH RELEVANCE: Congruent findings from animal and human studies indicate that a highly conserved network of brain structures regulates the expression of fear and anxiety in mammals. The amygdala in particular plays a critical role in various aspects of emotional regulation including the expression of innate fear responses or defensive behaviors, the acquisition of new fear responses as a result of experience and the facilitation of memory by emotions. Importantly, functional imaging studies indicate that the amygdala is often hyper-responsive in humans afflicted with anxiety disorders. As a result, it is commonly believed that many anxiety disorders result, at least in part, from a dysregulation of amygdala processes normally mediating fear/defensive behaviors. Thus, identifying inputs that exert a potent effect on amygdala excitability might open new approaches for the treatment of anxiety disorders. This proposal aims to do just that, by studying the influence of midline thalamic nuclei on the amygdala. Together, the experiments proposed here promise to advance our understanding of the neurobiological basis of anxiety disorders.
### NIH Awards by Location & Organization

**NOTE:** These data do not include projects funded by the American Recovery and Reinvestment Act of 2009. For a list of those projects, please visit [http://report.nih.gov/recovery](http://report.nih.gov/recovery).

**By Organization**

- **Organization:** 1ST PLAYABLE PRODUCTIONS, LLC
- **City:** TROY
- **State:** NY
- **Country:** UNITED STATES
- **Awards:** 1
- **Funding:** $225,000

- **Organization:** 21ST CENTURY THERAPEUTICS, INC.
- **City:** DETROIT
- **State:** MI
- **Country:** UNITED STATES
- **Awards:** 1
- **Funding:** $4,784

- **Organization:** 23ANDME, INC.
- **City:** MOUNTAIN VIEW
- **State:** CA
- **Country:** UNITED STATES
- **Awards:** 1
- **Funding:** $260,360

- **Organization:** 2B TECHNOLOGIES, INC.
- **City:** BOULDER
- **State:** CO
- **Country:** UNITED STATES
- **Awards:** 1
- **Funding:** $484,446

- **Organization:** 3-C INSTITUTE FOR SOCIAL DEVELOPMENT
- **City:** DURHAM
- **State:** NC
- **Country:** UNITED STATES
- **Awards:** 4
- **Funding:** $1,988,645
NIH Awards by Location & Organization

NOTE: These data do not include projects funded by the American Recovery and Reinvestment Act of 2009. For a list of those projects, please visit http://report.nih.gov/recovery.

Fiscal Year: 2016
Institute/Center: All
Funding Mechanism: All
FOA: Format: RFA-IC-09-003 or PA-09-003
Location: NJ
Congressional District: All
Organization Type: Domestic Higher Education (DHE), Schools of Organization:

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**DESCRIPTION:** The amygdala plays a critical role in the genesis of defensive behaviors. Moreover, it is hyperactive in humans afflicted with anxiety disorders. Thus, it is commonly believed that many anxiety disorders result, at least in part, from a dysregulation of amygdala processes normally mediating fear or defensive behaviors. Accordingly, research on the mechanisms controlling amygdala excitability might open new approaches for the treatment of anxiety disorders. This proposal aims to shed light on the influence of MTh inputs to the amygdala. To this end, we will first identify the targets and post-synaptic mechanisms of MTh inputs to the amygdala using physiological and anatomical methods. Indeed, BLA and CeA both contain multiple cell types that express different peptides/receptors and form contrasting connections with each other and extrinsic afferents. Therefore, in Aim #1, we will combine anterograde tracing with immunocytochemistry for various neuronal markers to identify the targets of MTh axon terminals in the amygdala at the light and electron microscopic levels. Building on these results, Aim #2 will combine optogenetic and patch clamp recording techniques in vitro to study the impact of MTh inputs on amygdala cells. Armed with this information, the last two aims will examine the influence of MTh cells on amygdala-dependent functions. Indeed, recent studies have revealed that following muscimol infusions in MTh nuclei, the expression of amygdala-dependent learned and innate fear is drastically reduced. However, it is unclear whether these muscimol findings result from the inhibition of nearby thalamic cells (e.g., mediodorsal nucleus), or the disinhibition of other targets of MTh nuclei (e.g., prefrontal cortex), that project to the amygdala. Two different approaches will be used to address this question. First, in Aim #3, we will perform simultaneous extracellular recordings of MTh and amygdala cells during the expression of learned and innate fear. Next, in Aim #4, we will use a dual viral strategy allowing us to express halorhodopsin or channelrhodopsin, but only in MTh cells that project to the amygdala. We will then optogenetically inhibit or excite amygdala-projecting MTh cells and examine how this affects behavior on amygdala-dependent tasks that probe learned or innate fear. Together, the experiments proposed here will reveal how MTh neurons regulate the excitability of the amygdala during the expression of learned and innate fear. This knowledge will pave the way for pharmacological interventions aiming to regulate the activity of midline thalamic cells by taking advantage of their unusual profile of receptor expression.

**PUBLIC HEALTH RELEVANCE:** Congruent findings from animal and human studies indicate that a highly conserved network of brain structures regulates the expression of fear and anxiety in mammals. The amygdala in particular plays a critical role in various aspects of emotional regulation, including the expression of innate fear responses or defensive behaviors, the acquisition of new fear responses as a result of experience, and the facilitation of memory by emotions. Importantly, functional imaging studies indicate that the amygdala is often hyper-responsive in humans afflicted with anxiety disorders. As a result, it is commonly believed that many anxiety disorders result, at least in part, from a dysregulation of amygdala processes normally mediating fear or defensive behaviors. Thus, identifying inputs that exert a potent effect on amygdala excitability might open new approaches for the treatment of anxiety disorders. This proposal aims to do just that, by studying the influence of midline thalamic nuclei on the amygdala. Together, the experiments proposed here will reveal how MTh neurons regulate the excitability of the amygdala during the expression of learned and innate fear. This knowledge will pave the way for pharmacological interventions aiming to regulate the activity of midline thalamic cells by taking advantage of their unusual profile of receptor expression.
Monitor awards to your institution

New Query Form

Fiscal Year (FY): Current FY is 2016

RESEARCHER AND ORGANIZATION
Principal Investigator (PI) / Project Leader:
(Last Name, First Name)
Use '%' for wildcard in PI names
Enter several PI/Project Leader names OR PI Profile IDs
Organization:
Organization Type:
Department:
City:
State:
Country:
Congressional District:
DUNS Number:

TEXT SEARCH
Text Search (Logic):
Search in
Projects
Publications
News
Limit Project search to
Start Year
End Year
Limit Publication search to

PROJECT DETAILS
Project Number/ Application ID:
Agency/Institute/Center:
NIH Spending Category:
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Monitor awards to your institution

**Save New Query**

* Title: Vanderbilt Active Projects

- New Projects Alert: ✔
- New Publications Alert: ✔
- New News Alert: ✔

Notes: All funding agencies, active projects only

**RESEARCHER AND ORGANIZATION**

- Principal Investigator (PI) / Project Leader: 
  (Last Name, First Name)
  Use ‘%’ for wildcard in PI names
  Enter several PI/Project Leader names OR PI Profile IDs

- Organization: VANDERBILT UNIVERSITY
  Use ‘%’ for wildcard
  Please enter at least 3 characters to use Lookup.
  Contains, Begins with, Exact

- City:

- State:

- Country:

- Congressional District:

- DUNS Number:

**TEXT SEARCH**

- Text Search (Logic):

- Search in:

- Limit Project search to:

- Limit Publication search to:
Links to press releases on RePORT

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PSA: Acknowledge NIH funding

http://grants.nih.gov/grants/acknow.htm

Communicating and Acknowledging Federal Funding

Publicizing the outcomes of NIH-funded projects and communicating the role of NIH support in biomedical research improves public understanding of how the biomedical research community as a whole, are working to improve human health.

This important information for researchers and public information officers (PIOs) describes how to correctly acknowledge NIH in your presentations, papers, posters, and press releases.

On This Page:
- Requirements for Acknowledging NIH-Supported Research
- Proper Grant Number Format
- Information for Researchers
- Information for Public Information Officers
- Frequently Asked Questions

Requirements for Acknowledging NIH-Supported Research

According to NIH grants policy, all grantee publications, research publications, press releases, other publications or documents about research that must include the following two statements:

1. A specific acknowledgment of NIH grant support

"Research reported in this [publication/press release] was supported by xxx of the Institute(s), Center, or other NIH elements) of the National Institutes of Health under award number [specific NIH grant number(s) in this format: R01GM987654]."

2. A disclaimer that says:
PI profiles: one example

PI Profile and website of an NIH funded PI accessed through RePORT
Key facts & figures: NIH Data Book

The NIH Data Book (NDB) provides basic summary statistics on extramural grants and contract awards, grant applications, the organizations that NIH supports, the trainees and fellows supported through NIH programs, and the national biomedical workforce. Tables and charts are provided in a variety of formats, including PowerPoint (PPT) slides and Portable Documents Files (PDF) files.
How much did NIH spend on a particular disease or research area?

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Federal RePORTER

- http://federalreporter.nih.gov/
- Trans-federal agency searchable database of science awards
- STAR METRICS: a federal and research institution collaboration to create a repository of data and tools that will be useful to assess the impact of federal R&D investments
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- RePORT Frequently Asked Questions (FAQs)
- RePORTER Frequently Asked Questions (FAQs)
- Tutorials
- Communicating and acknowledging NIH funding
  - [http://grants.nih.gov/grants/acknow.htm](http://grants.nih.gov/grants/acknow.htm)
  - [http://grants.nih.gov/grants/acknow.htm#info_public](http://grants.nih.gov/grants/acknow.htm#info_public)