
Adding a bit of **color** and **light** to the brain

Matt Carter
Department of Biology
Williams College

August 8, 2022





A discrete parasubthalamic nucleus subpopulation plays a critical role in appetite suppression

Jessica H Kim¹, Grace H Kronen¹, Olivia K Barnhill², Jacob Sparber¹, Lauren B Heuer¹, Sierra Loomis¹, Matthew C Newman¹, Kenneth Han¹, Felix F Gulamali¹, Theresa B Logan¹, Katharine E Jensen¹, Samuel C Funderburk¹, Michael J Krashes¹, Matthew E Carter^{1*}

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Abstract: Food intake behavior is regulated by a network of appetite-inducing and appetite-suppressing neuronal populations throughout the brain. The parasubthalamic nucleus (PSTN), a relatively unexplored population of neurons in the posterior hypothalamus, has been hypothesized to regulate appetite due to its connectivity with other anorexigenic neuronal populations and because these neurons express *Fox1*, a marker of neuronal activation following a meal. However, the individual cell types that make up the PSTN are not well characterized, nor are their functional roles in food intake behavior. Here, we identify and distinguish between two discrete PSTN subpopulations, those that express neuropeptide Y (PSTN^{NPY} neurons) and those that express corticotropin-releasing hormone (PSTN^{CRH} neurons), and use a panel of genetically encoded tools *in vivo* to show that PSTN^{CRH} neurons play an important role in appetite suppression. Both subpopulations increase activity following a meal and in response to administration of the anorexigenic hormones amylin, cholecystikinin (CCK), and peptide YY (PYY). Interestingly, chemogenetic inhibition of PSTN^{CRH}, but not PSTN^{NPY} neurons, reduces the appetite-suppressing effects of these hormones. Consistently, optogenetic and chemogenetic stimulation of PSTN^{CRH} neurons, but not PSTN^{NPY} neurons, reduces food intake in hungry mice. PSTN^{CRH} and PSTN^{NPY} neurons project to distinct downstream brain regions, and stimulation of PSTN^{CRH} projections to individual anorexigenic populations reduces food consumption. Taken together, these results reveal the functional properties and projection patterns of distinct PSTN cell types and demonstrate an anorexigenic role for PSTN^{CRH} neurons in the hormonal and central regulation of appetite.

Editor's evaluation

This work has identified a previously unexplored role of the parasubthalamic nucleus in the regulation of feeding behavior. The combination of genetic and pharmacological approaches nicely demonstrates the physiological role of this group of neurons in regulating appetite. These studies will be of interest to the field and more broadly to the readers of eLife.

Introduction

The brain regulates food intake behavior through the coordinated activity of several distinct neuronal populations (Anderson and Lowell, 2012; Stevenson and Bell, 2012). Activity in anorexigenic

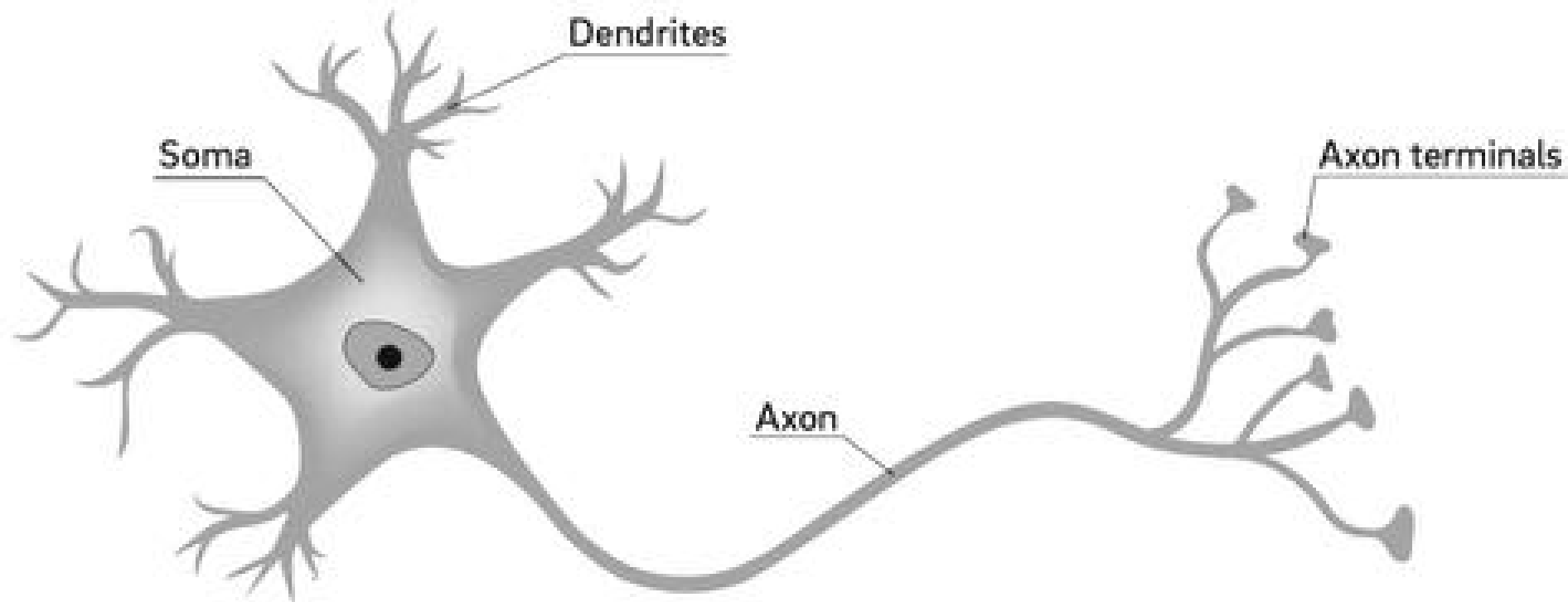
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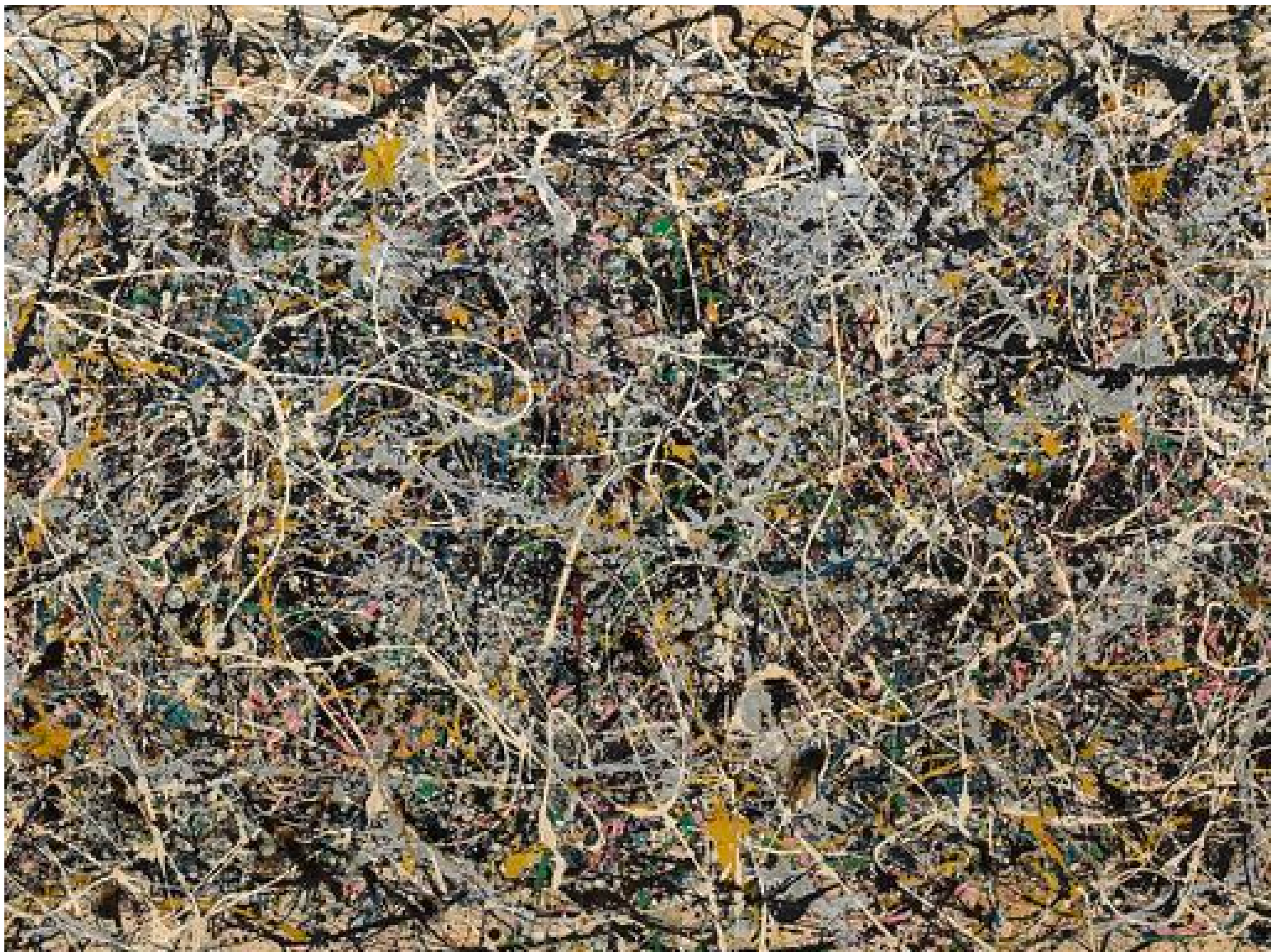


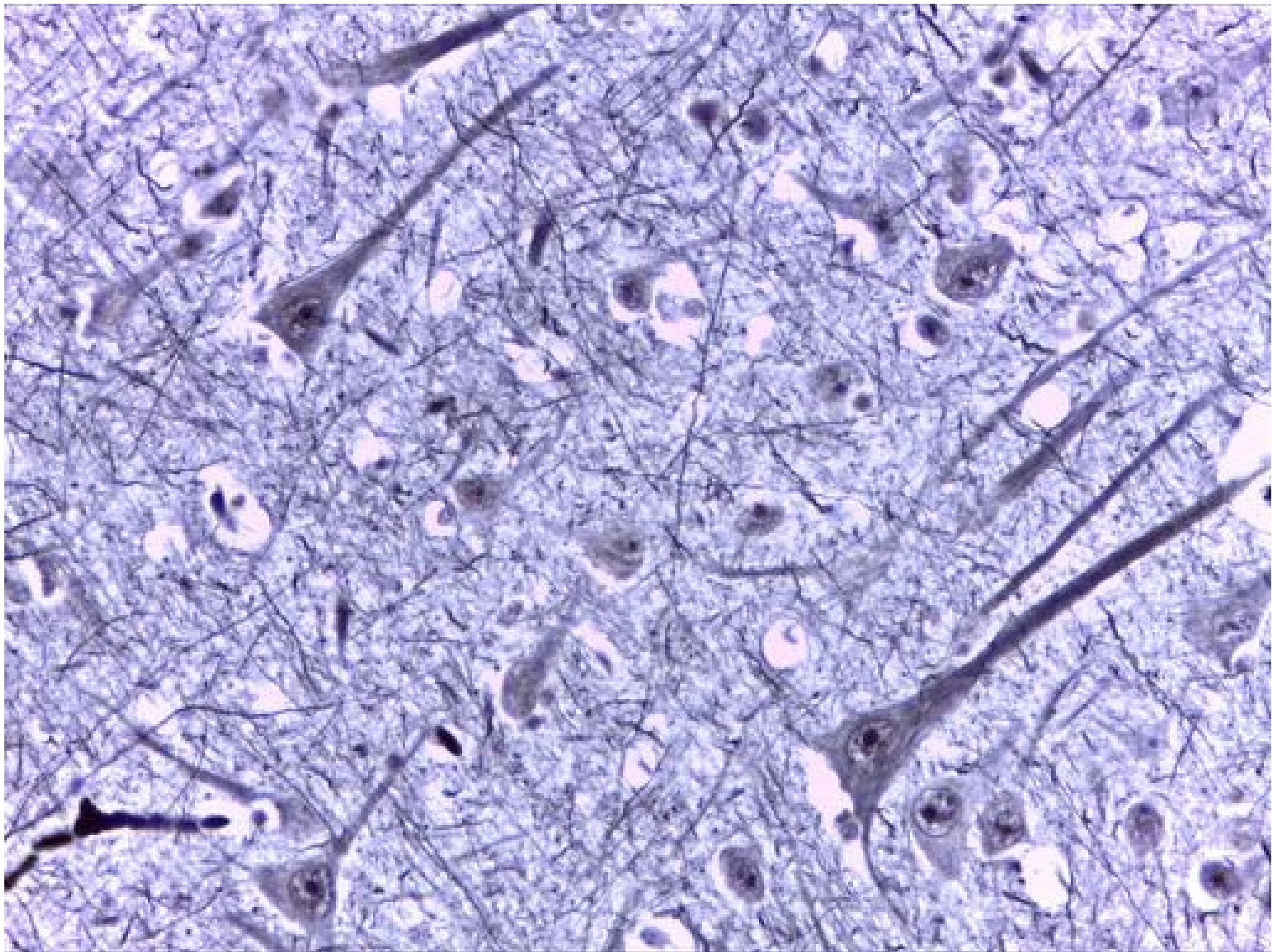


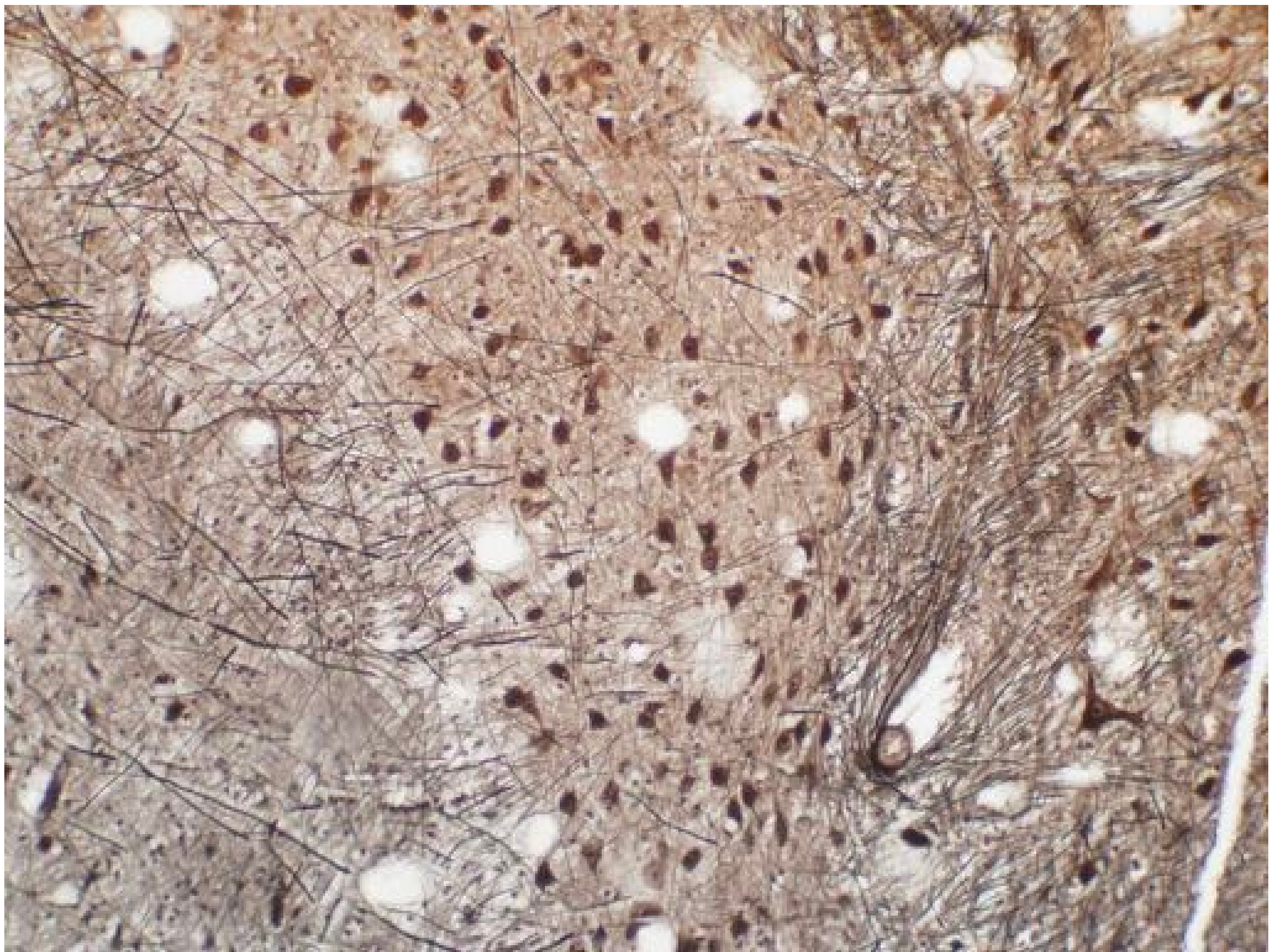


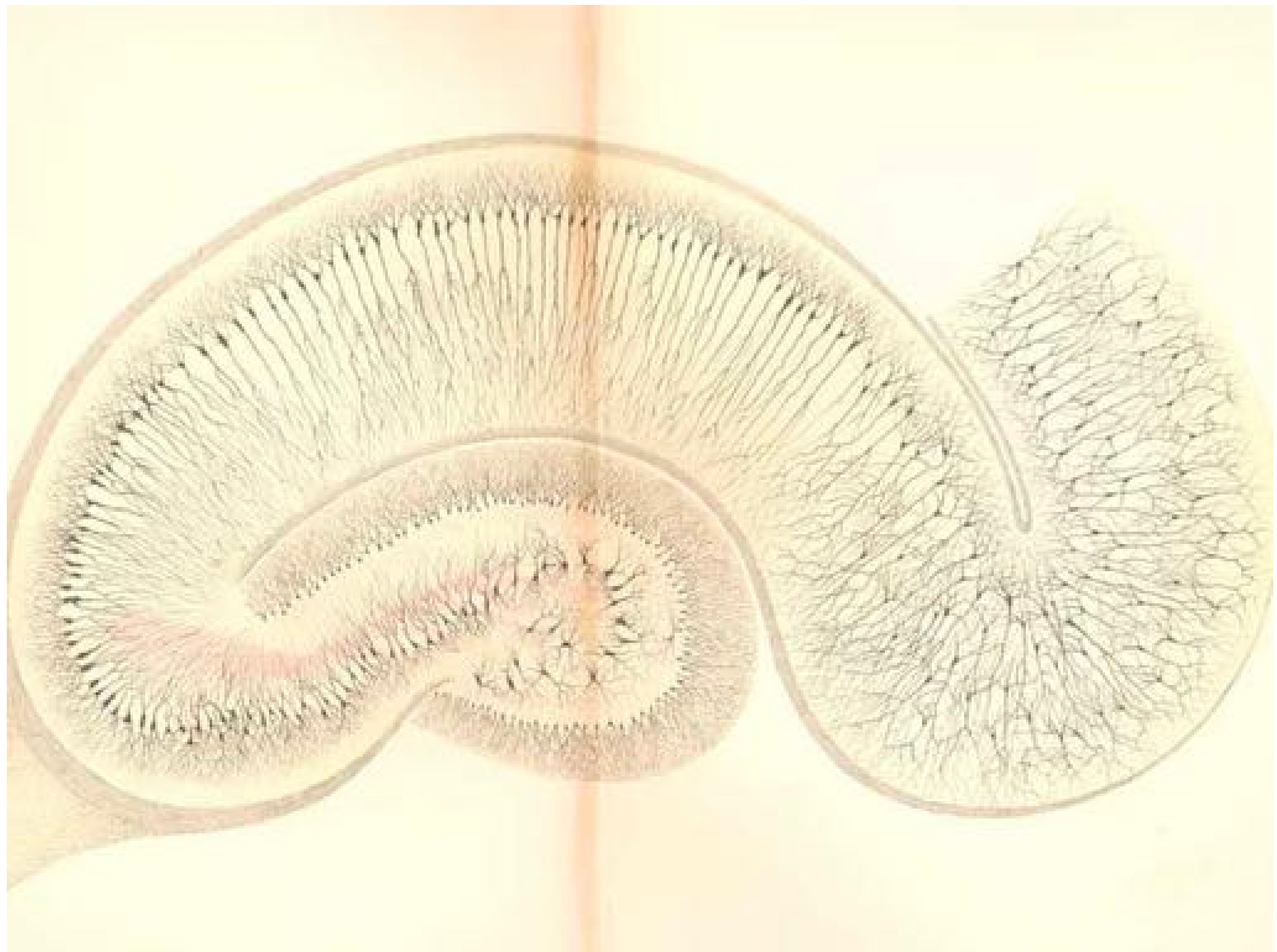




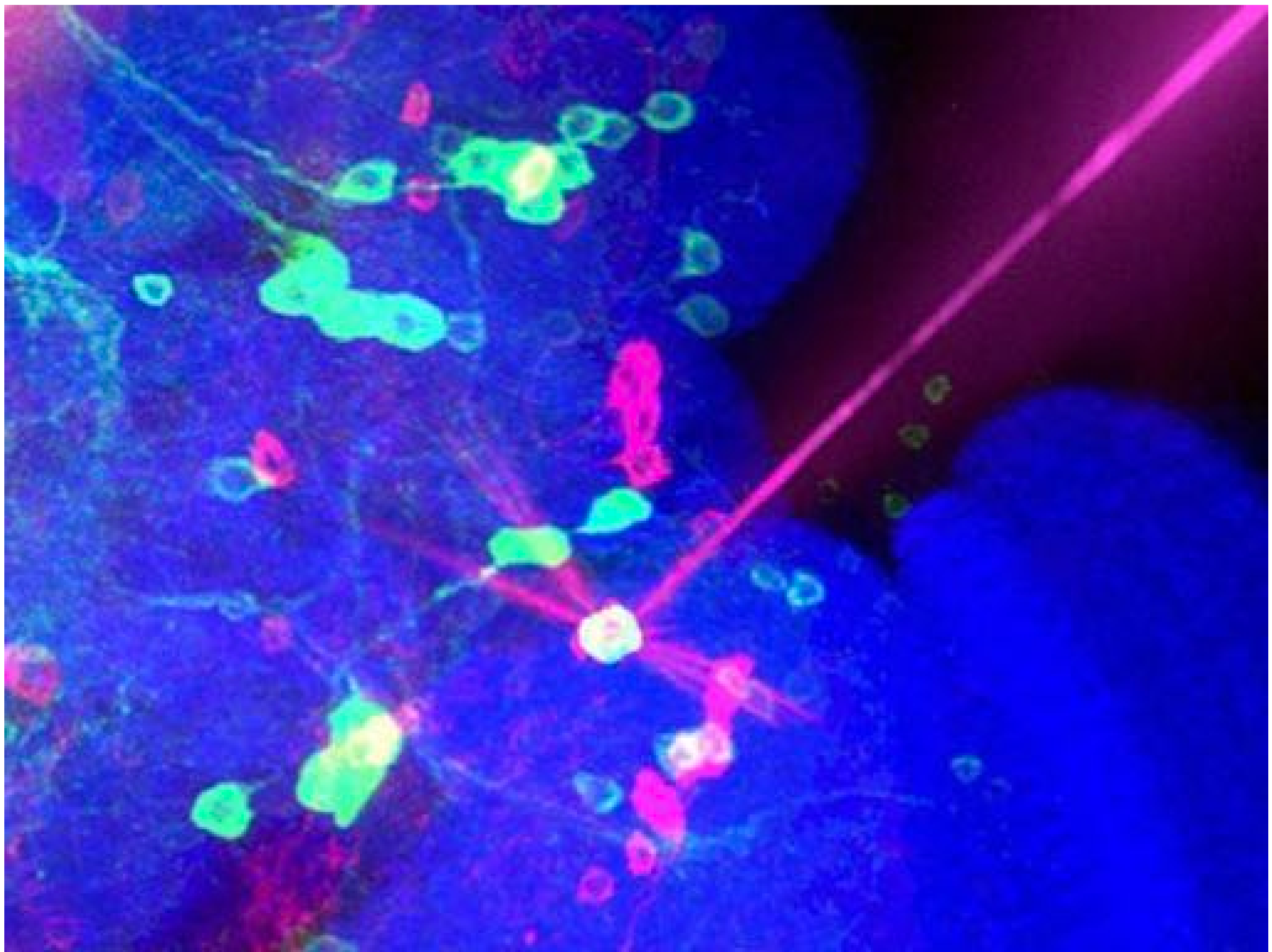


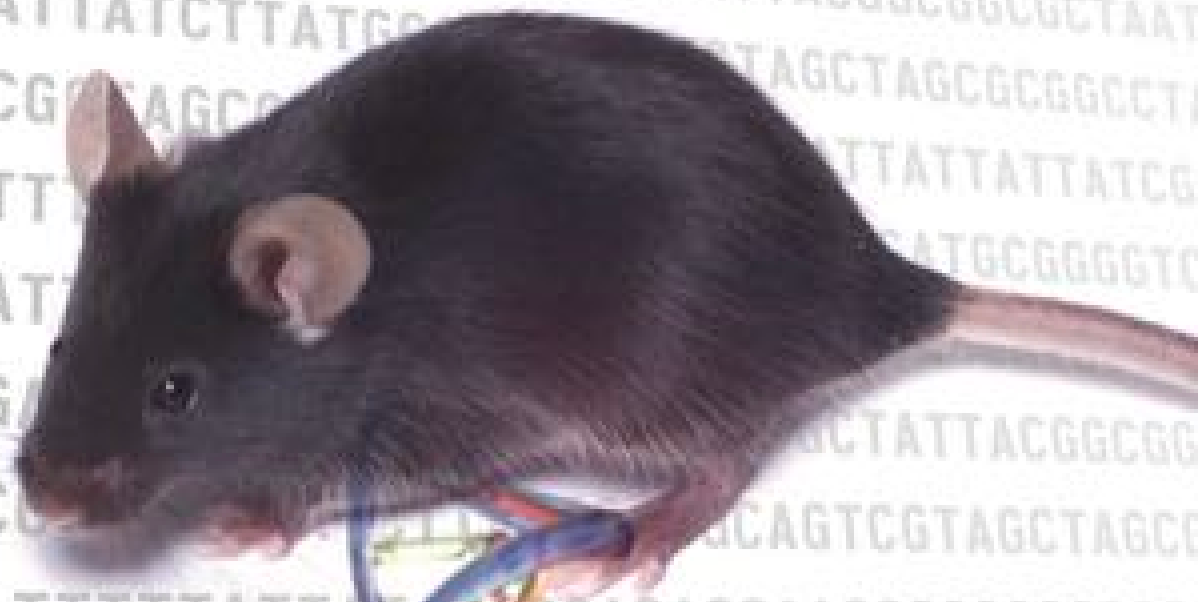






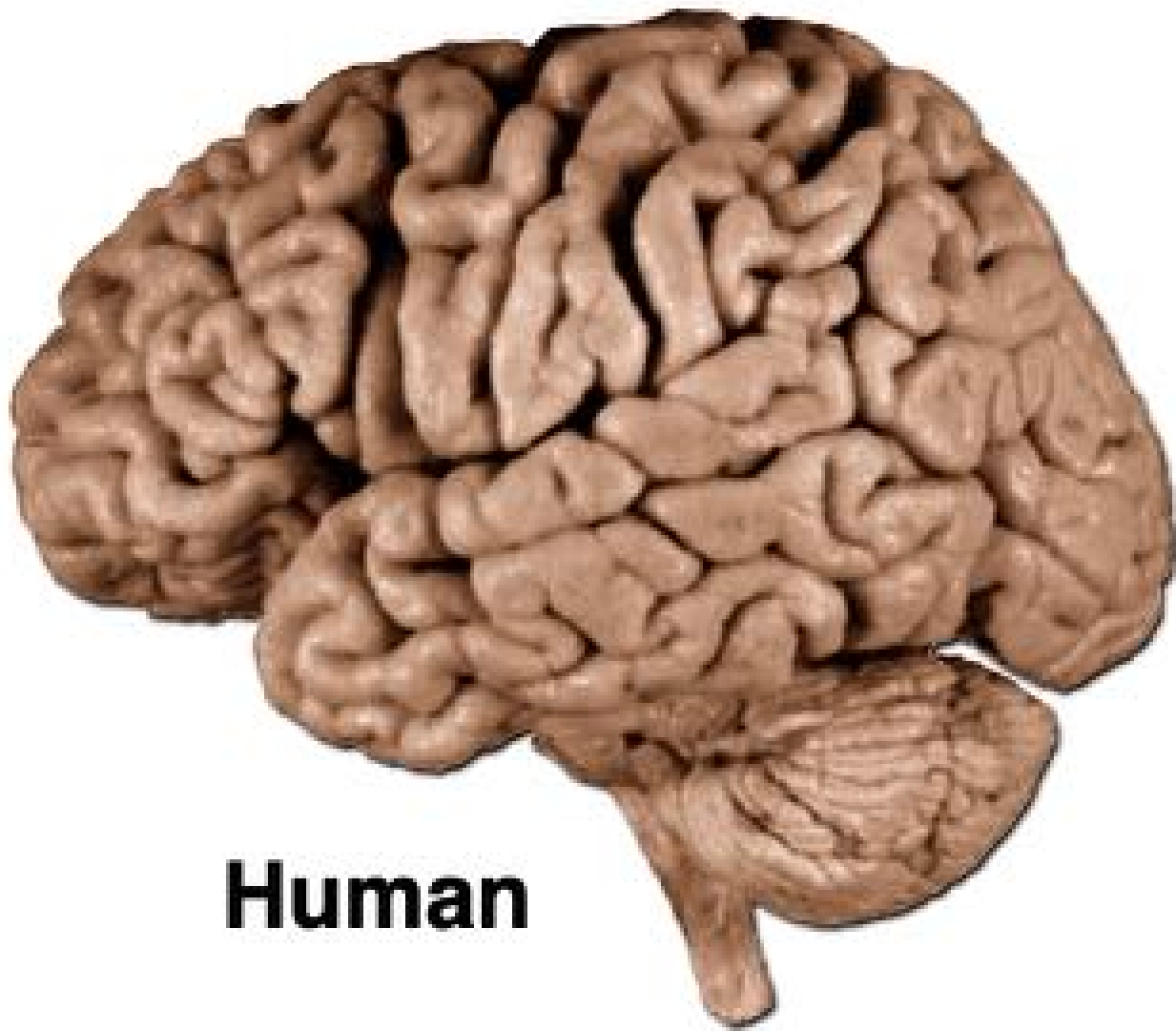






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CTCTTATT
GCGGATAT
CAGTCG
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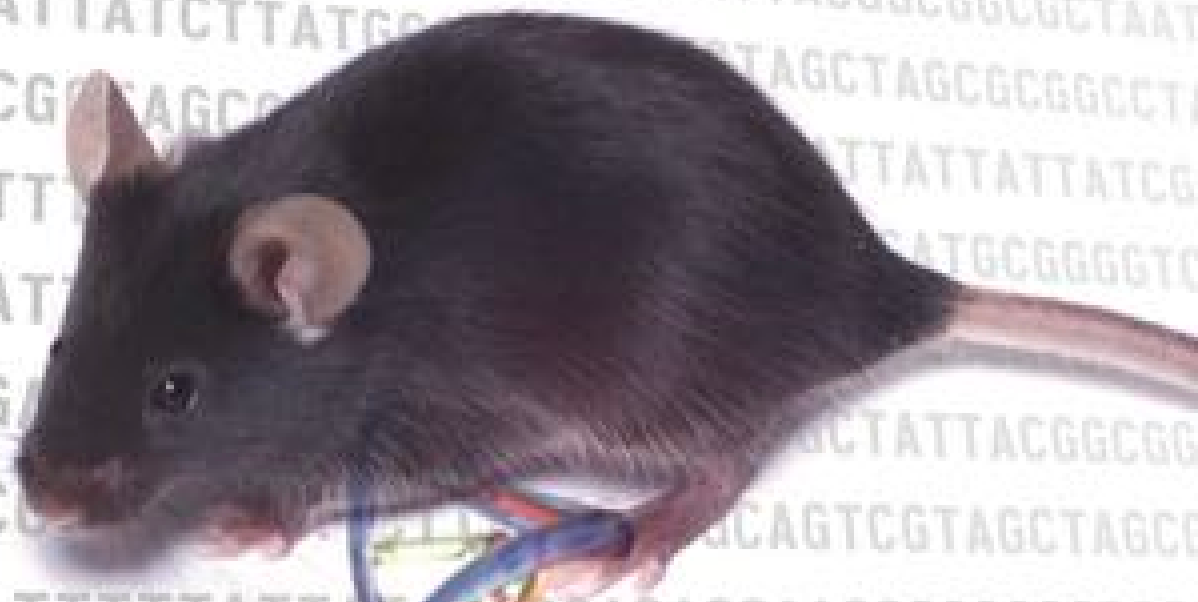




Human



Mouse



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Neural Structure

Neural Activity

Neuromodulation



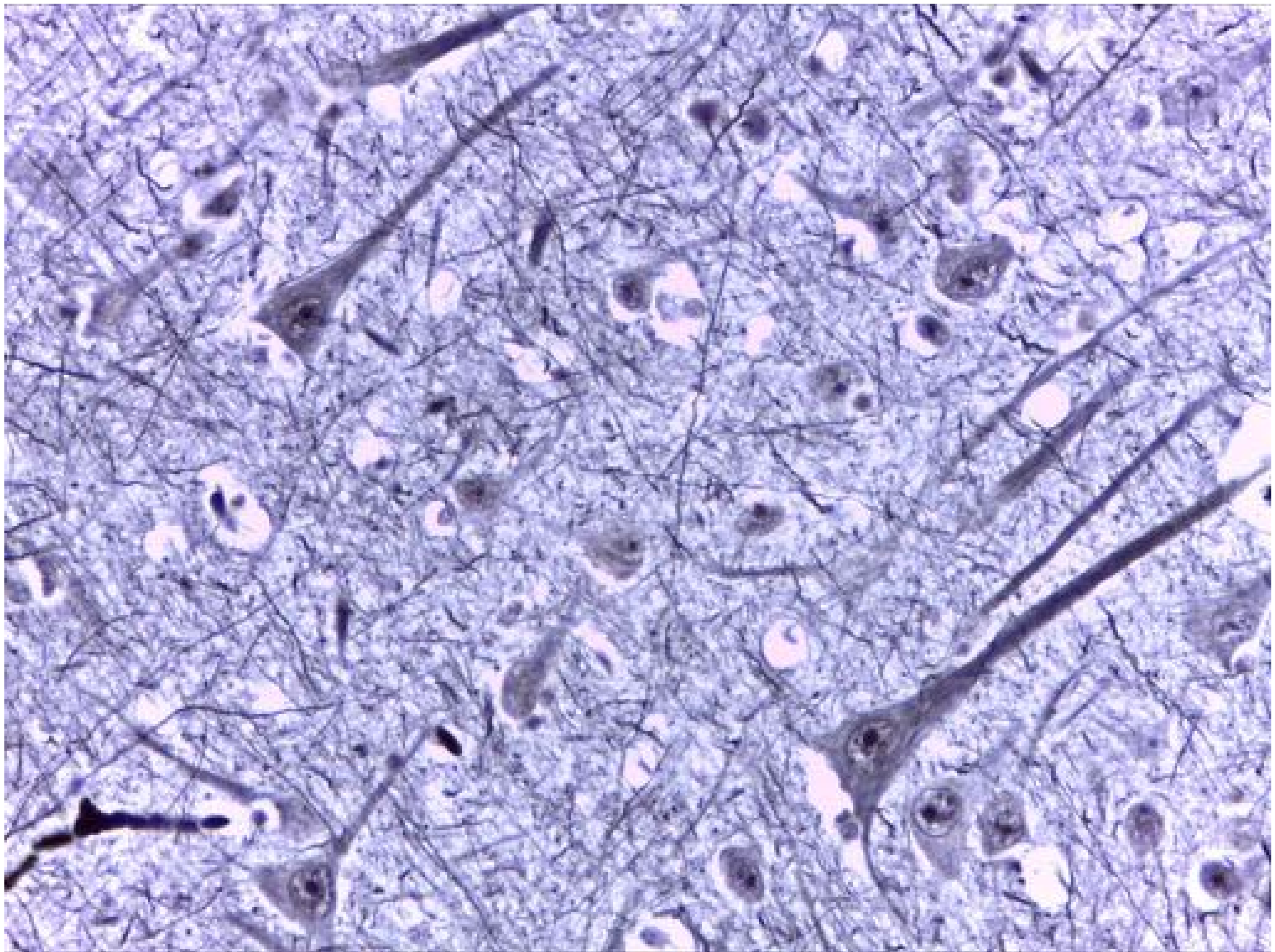
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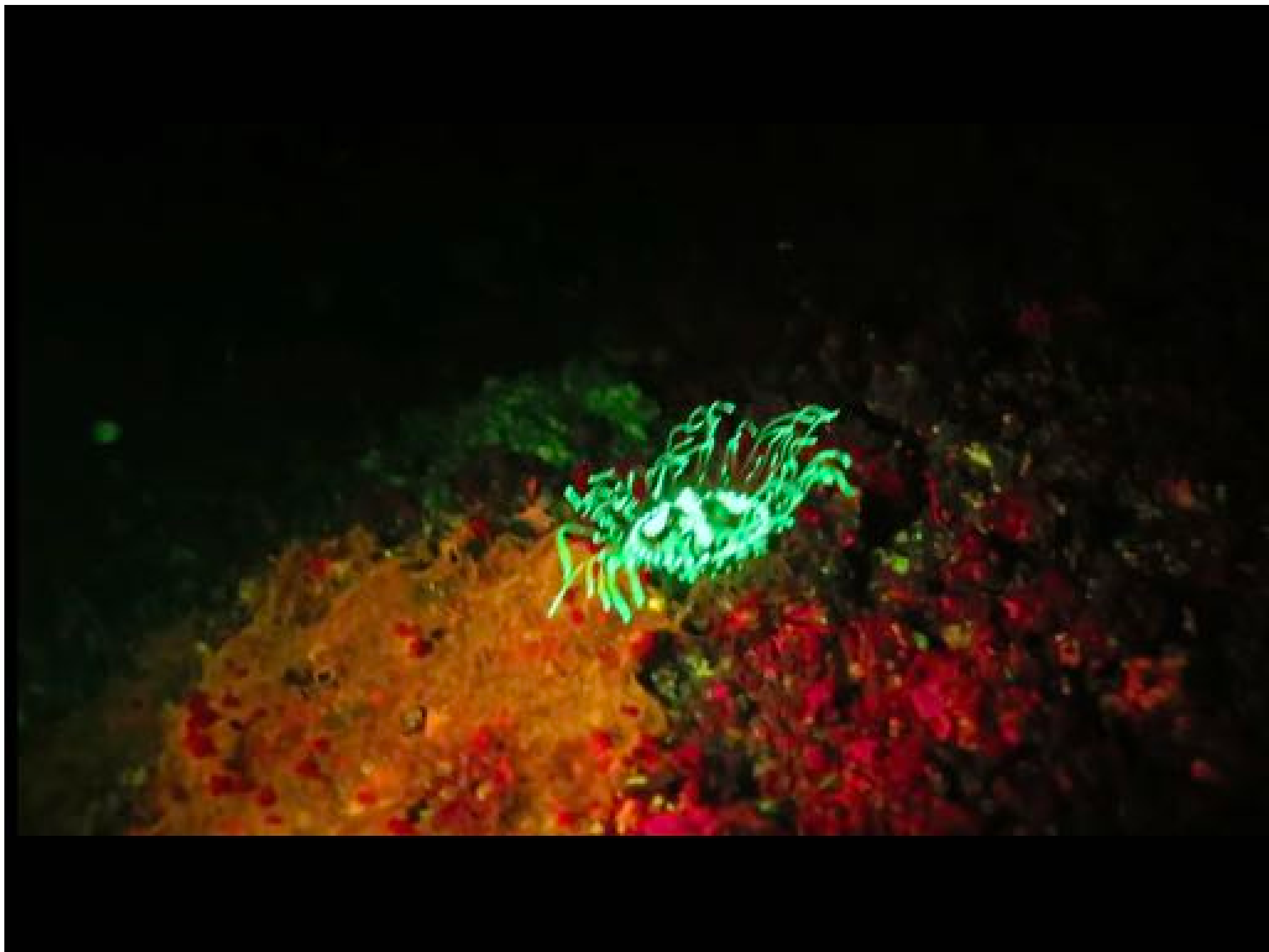
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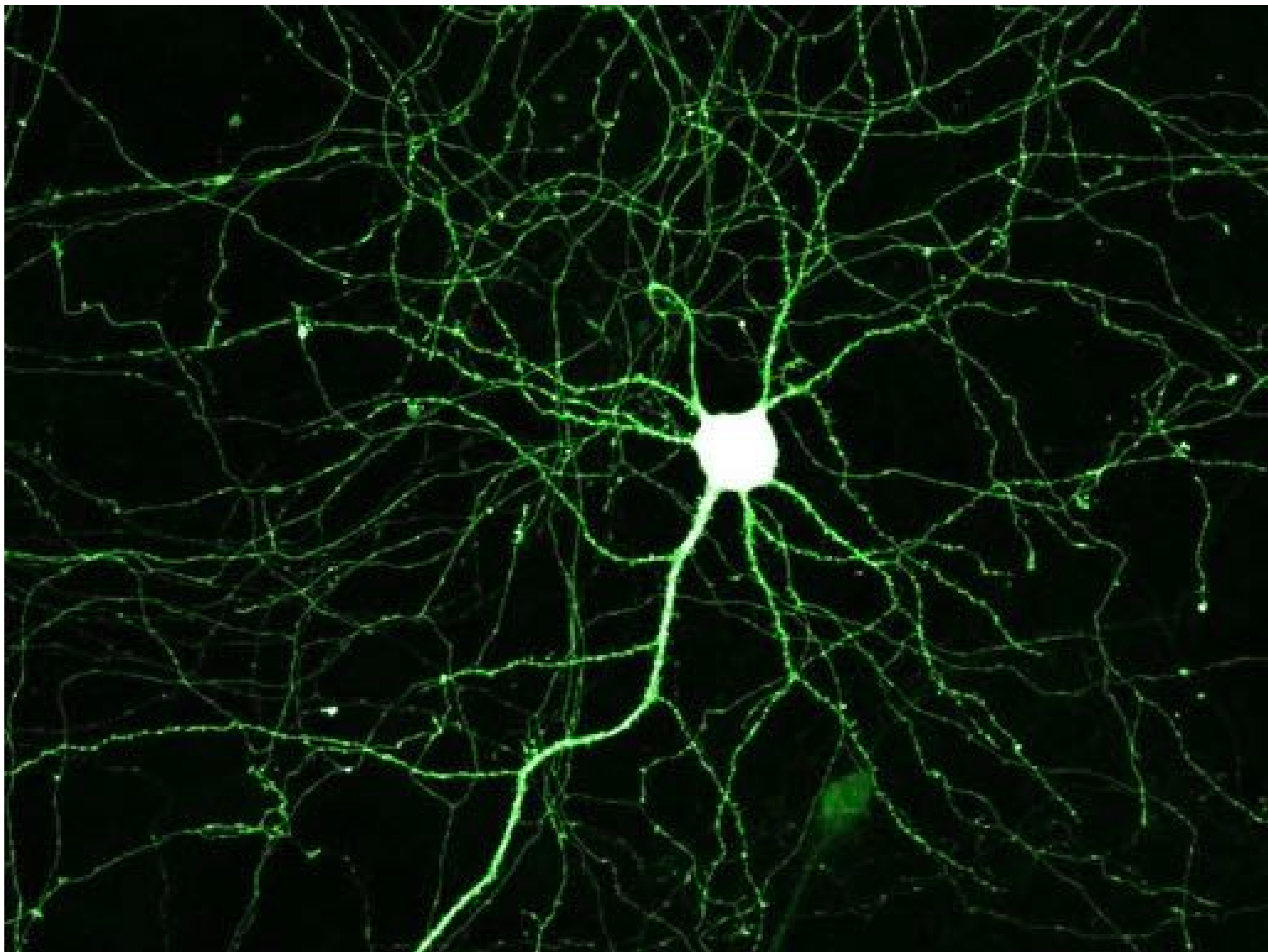


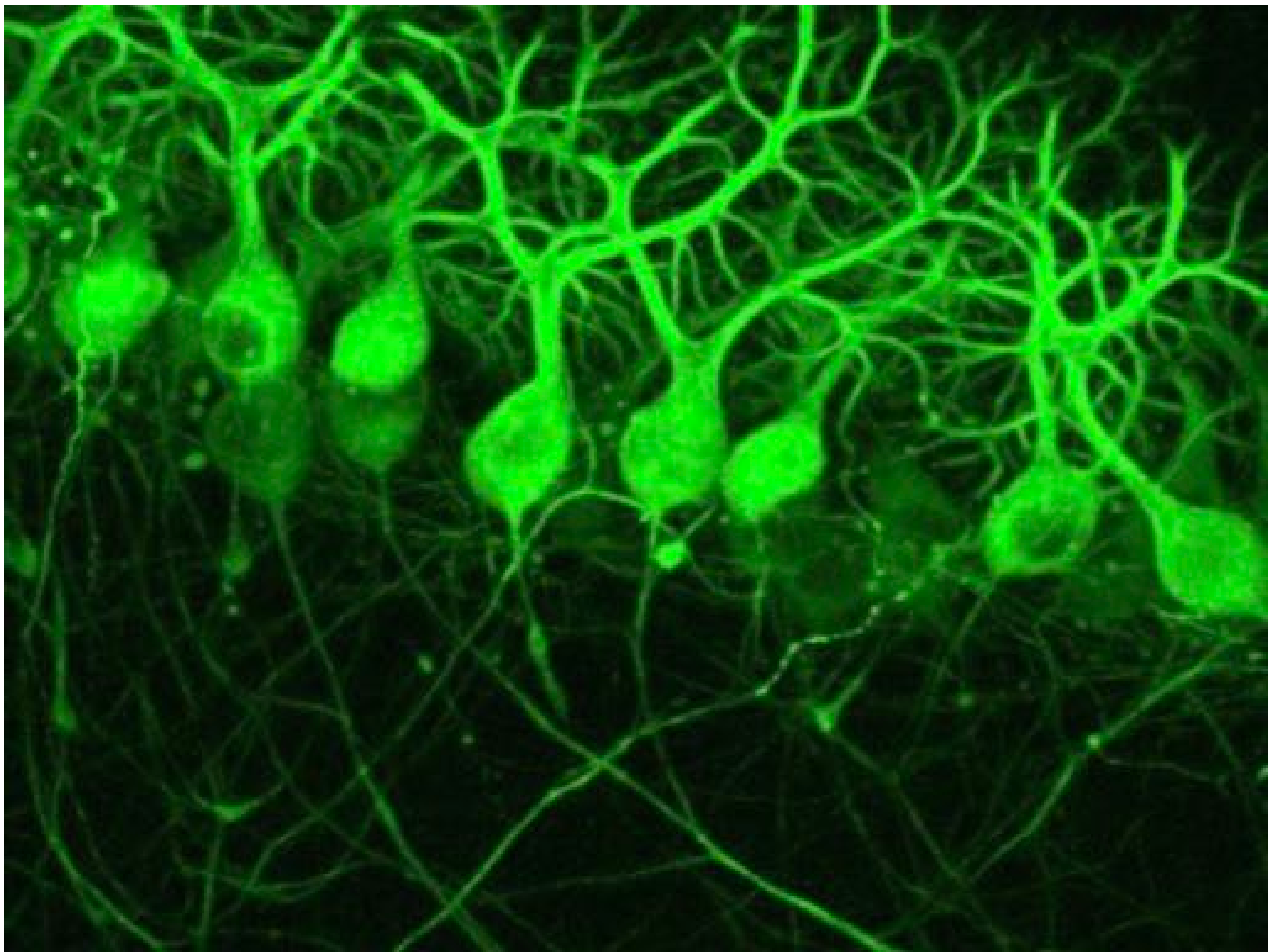


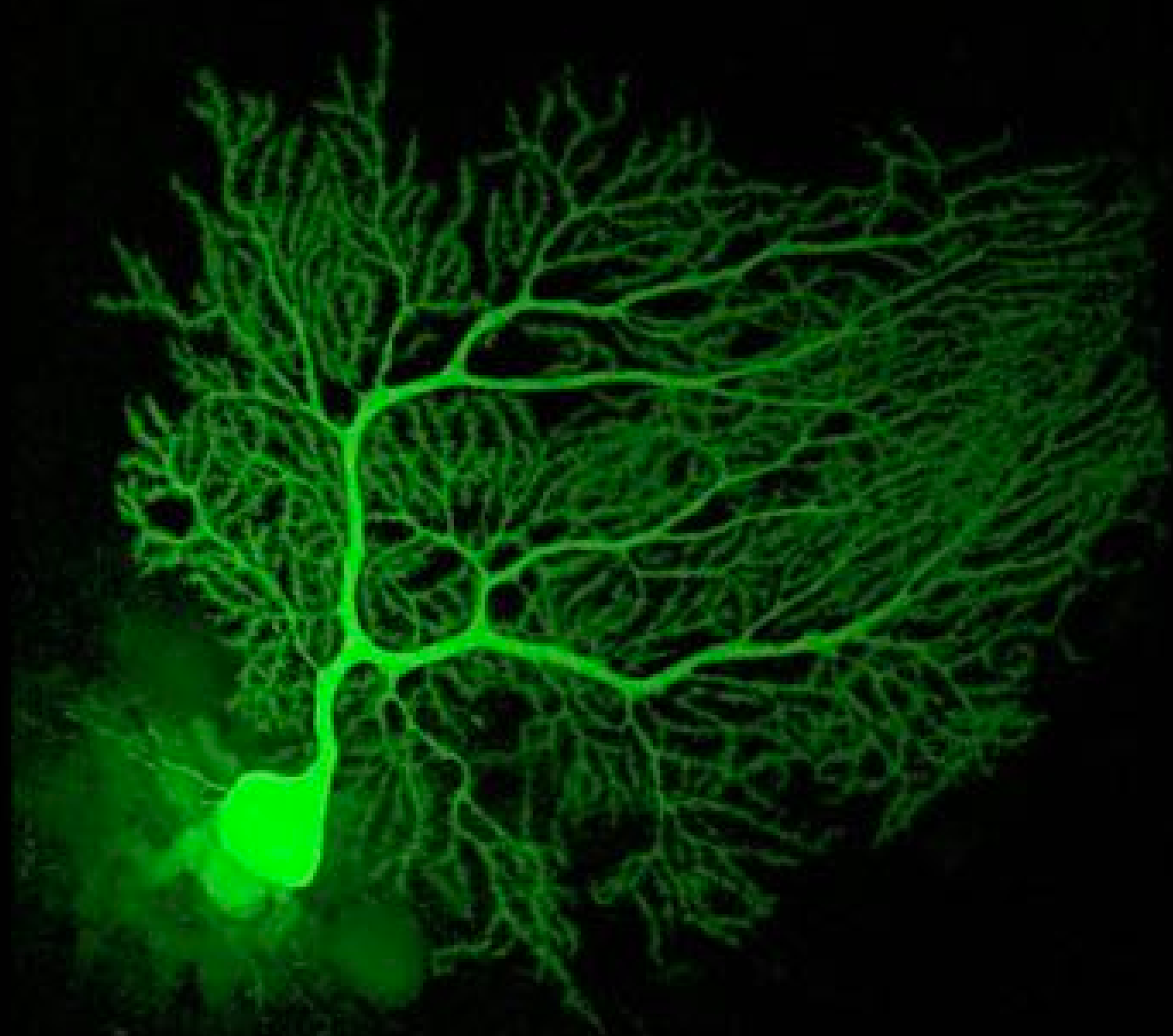


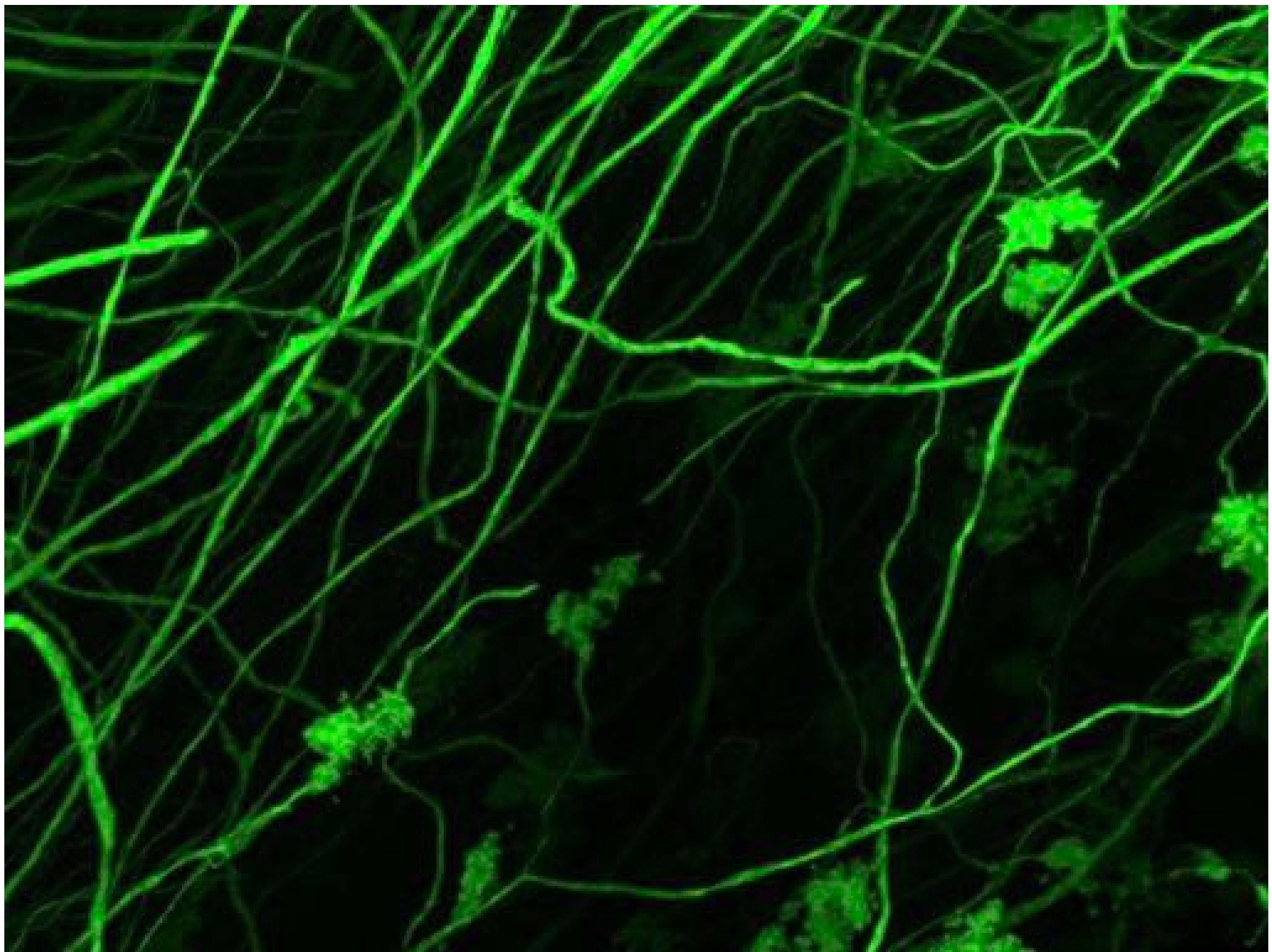


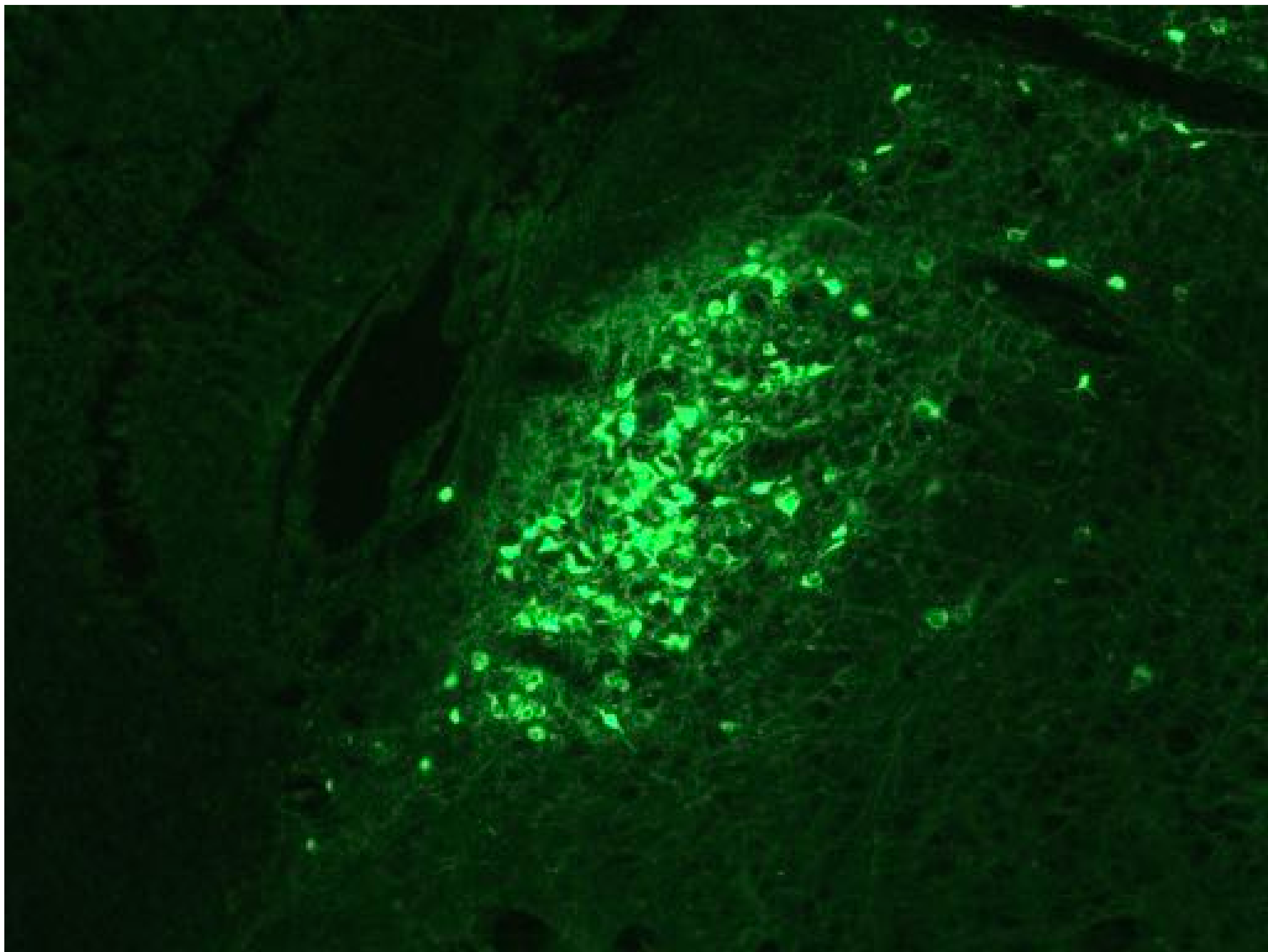
Green Fluorescent Protein (GFP)

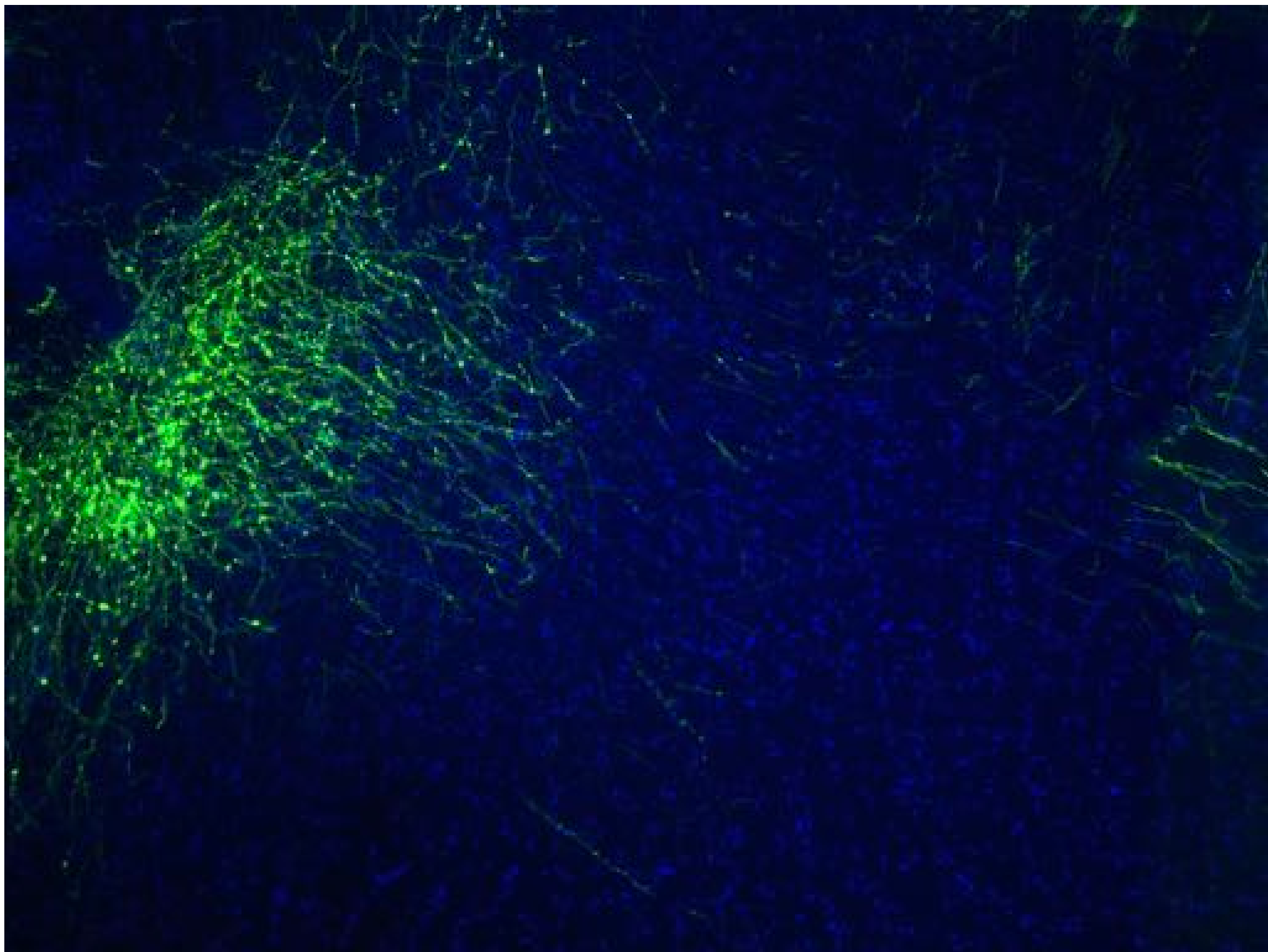


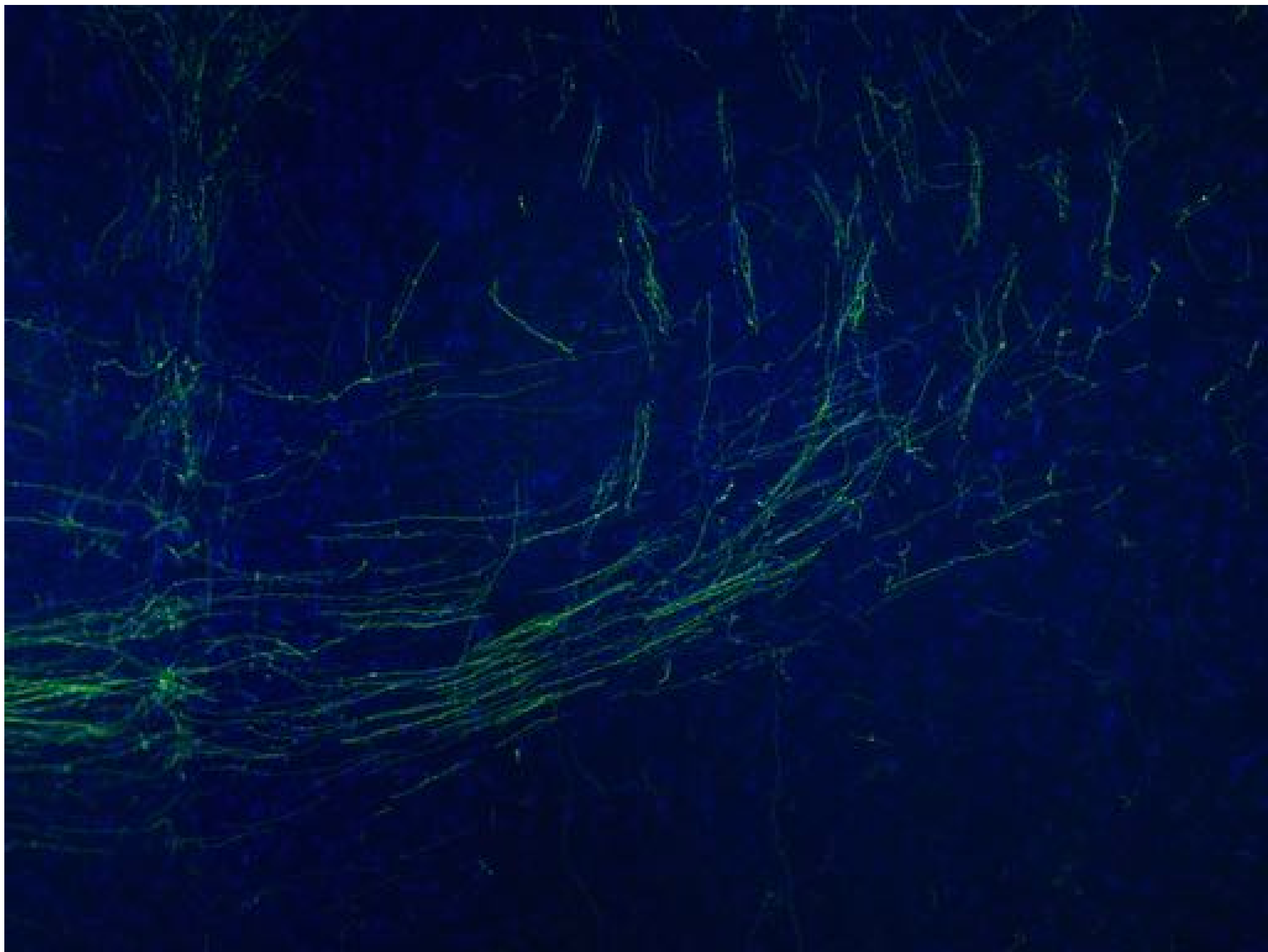




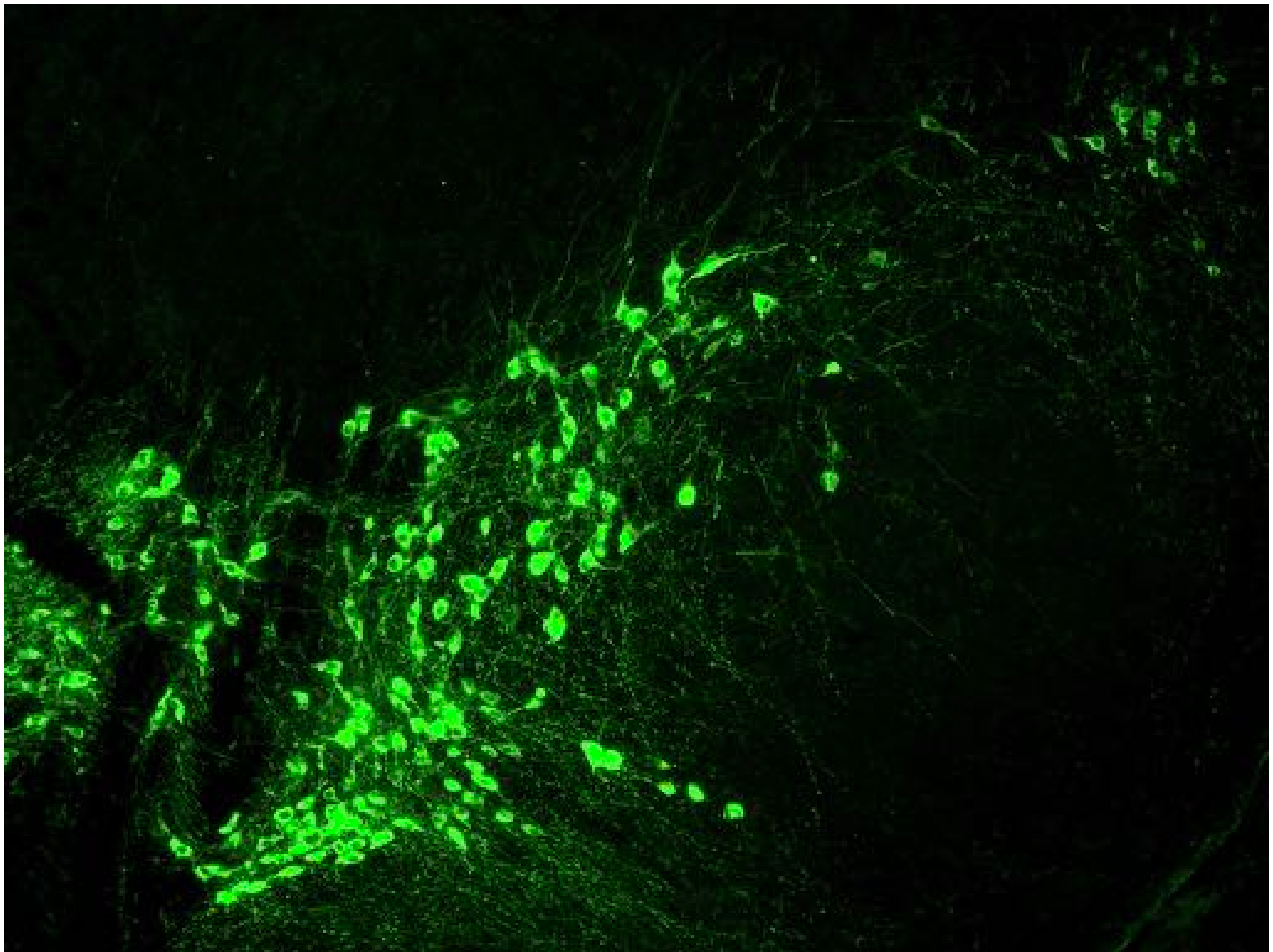


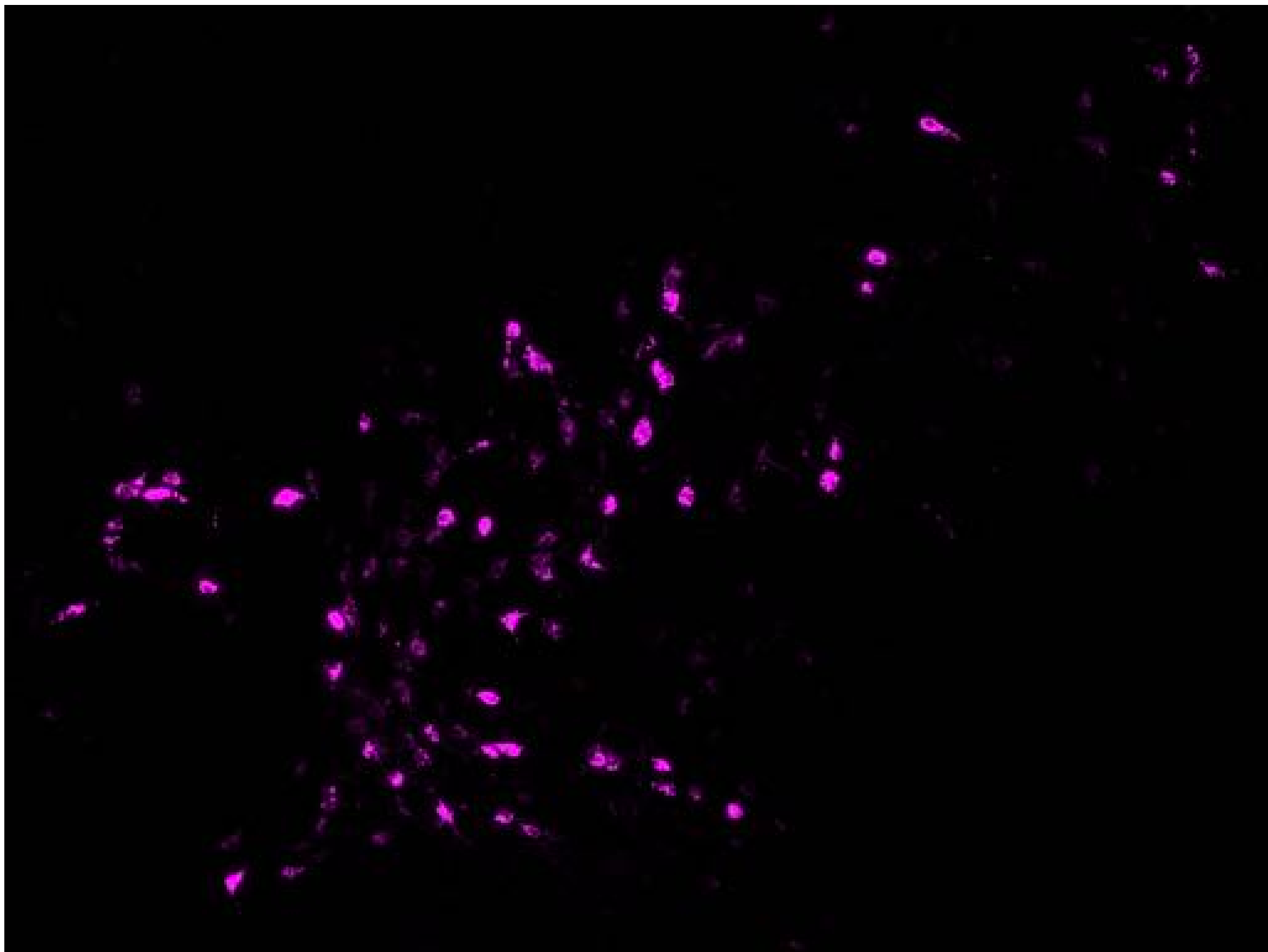


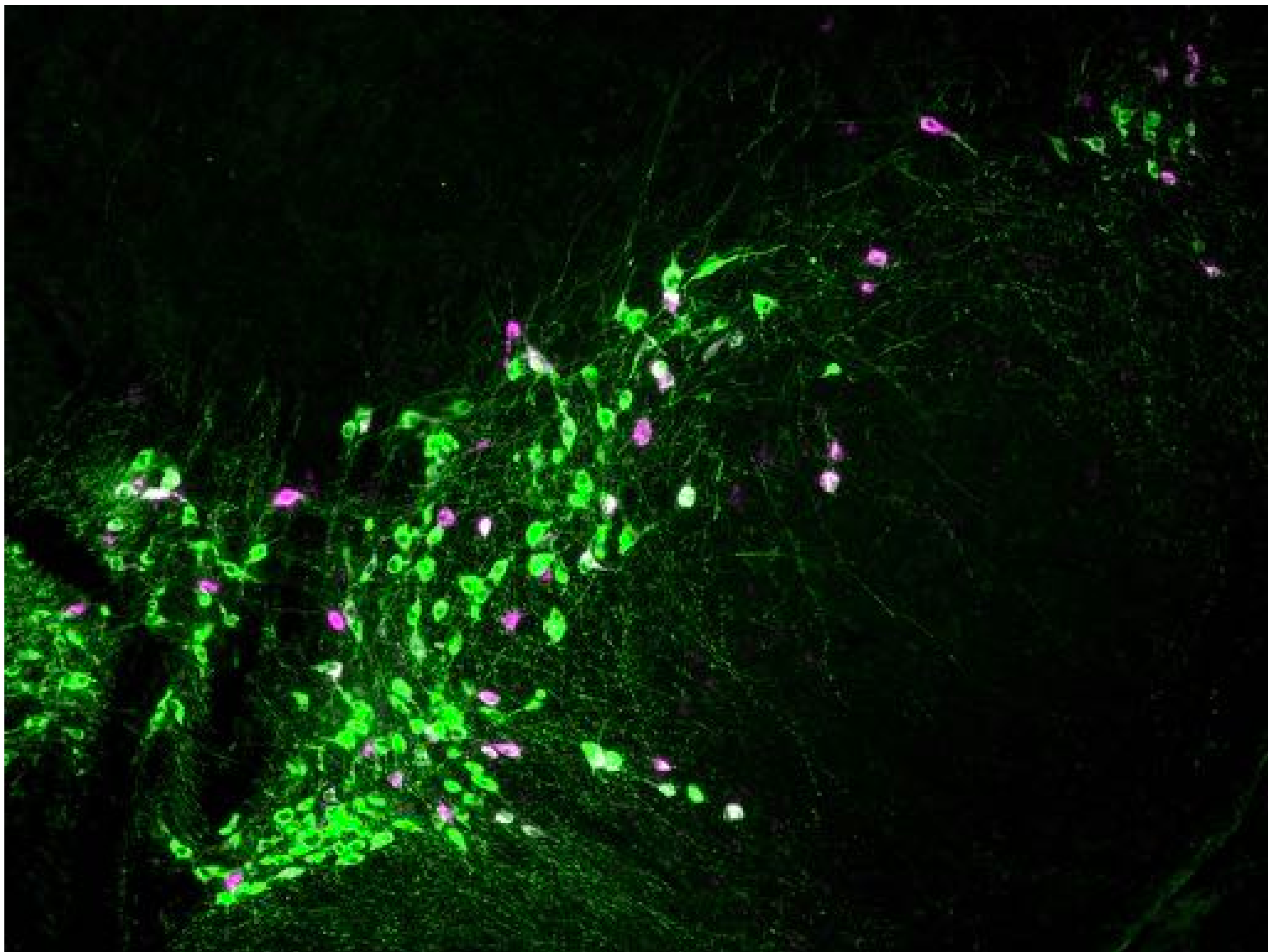


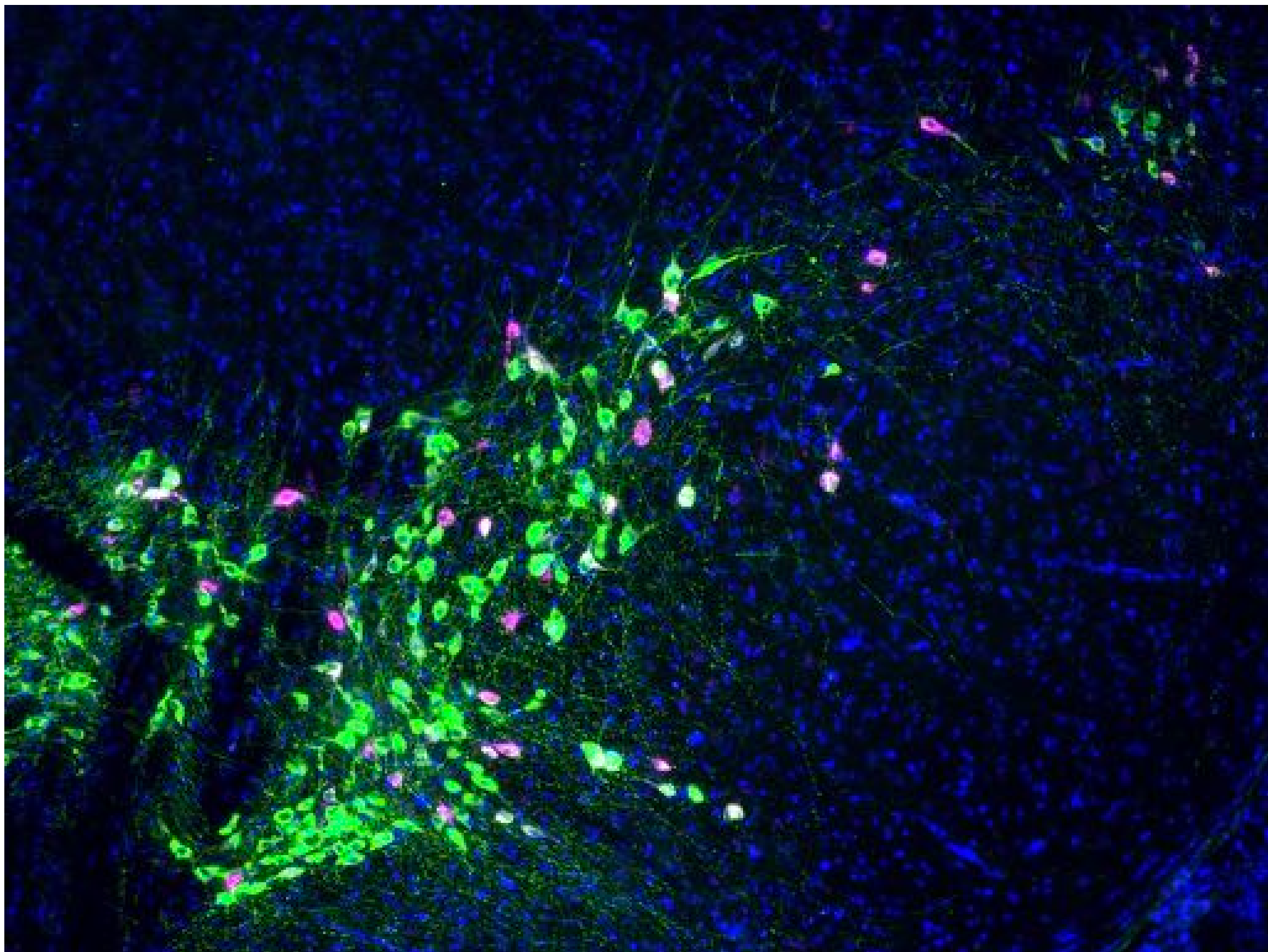


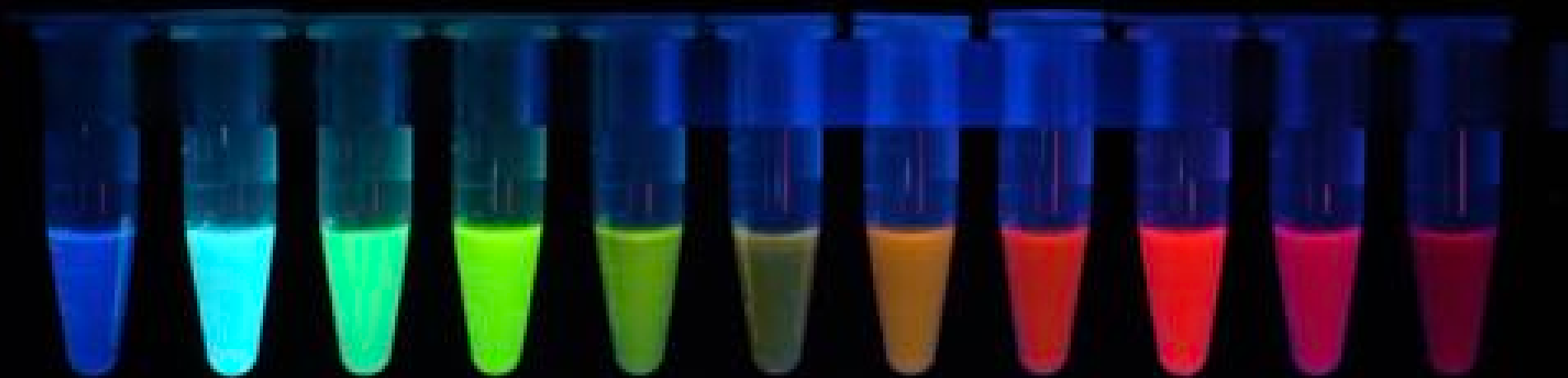


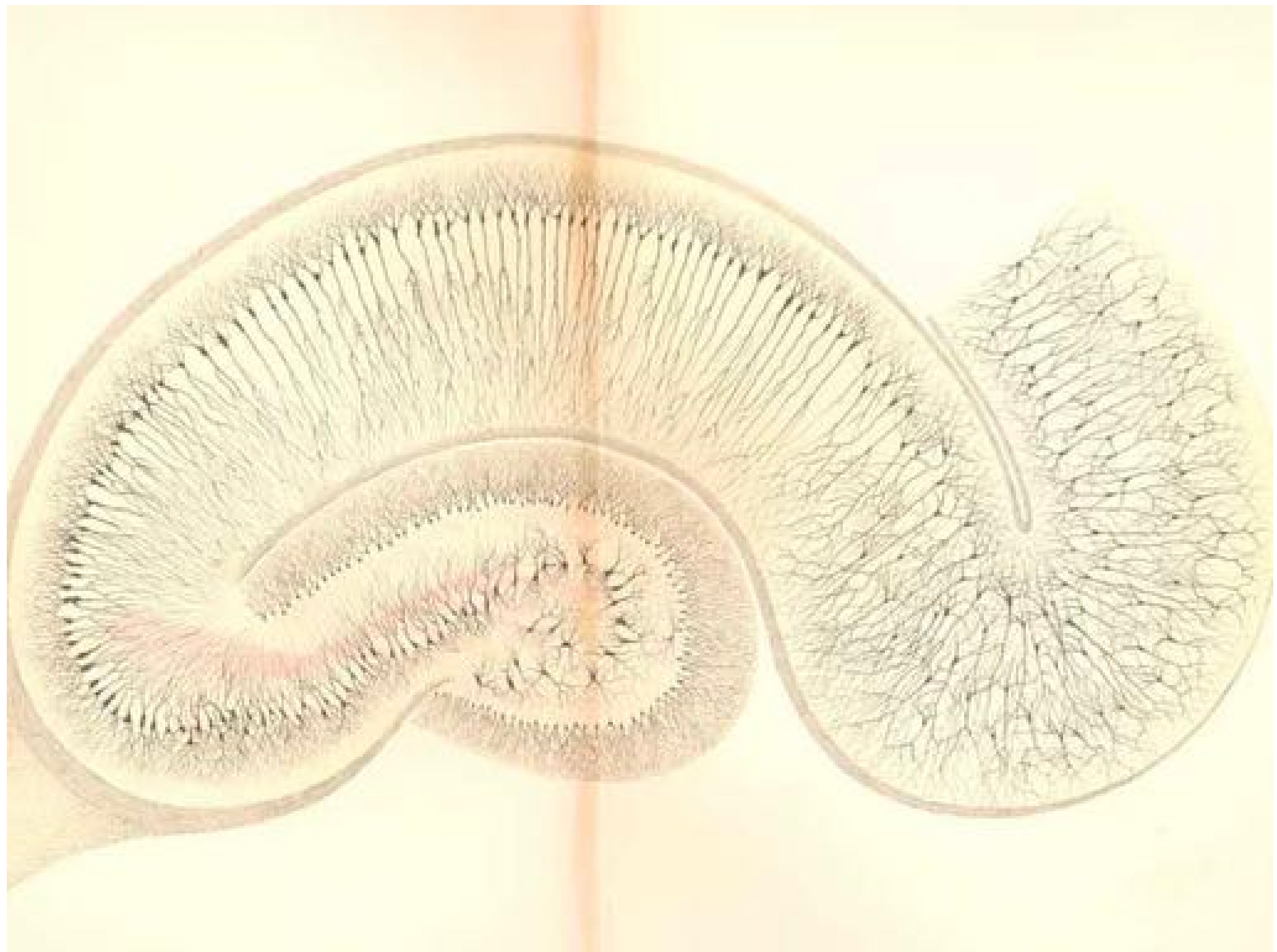


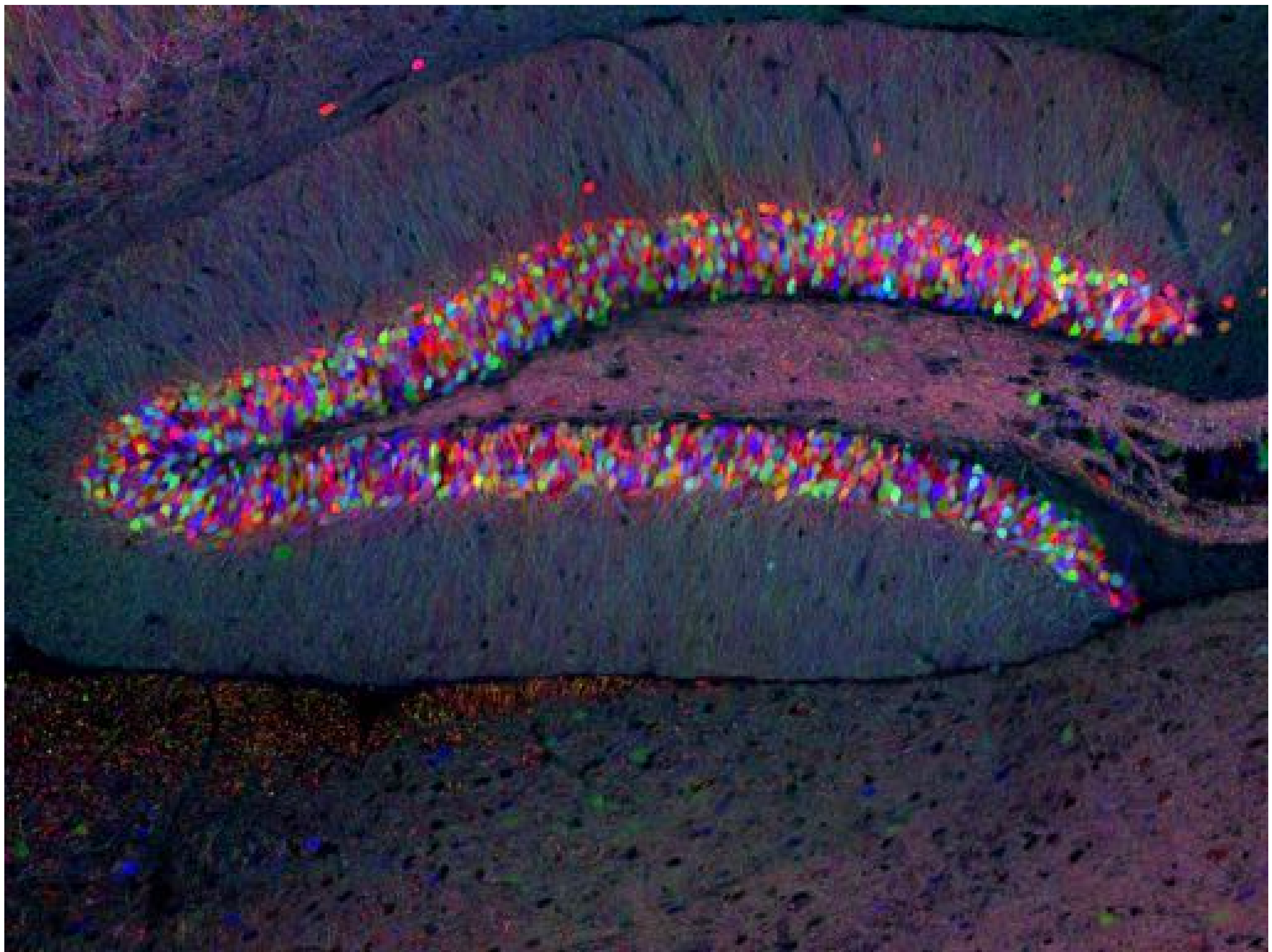


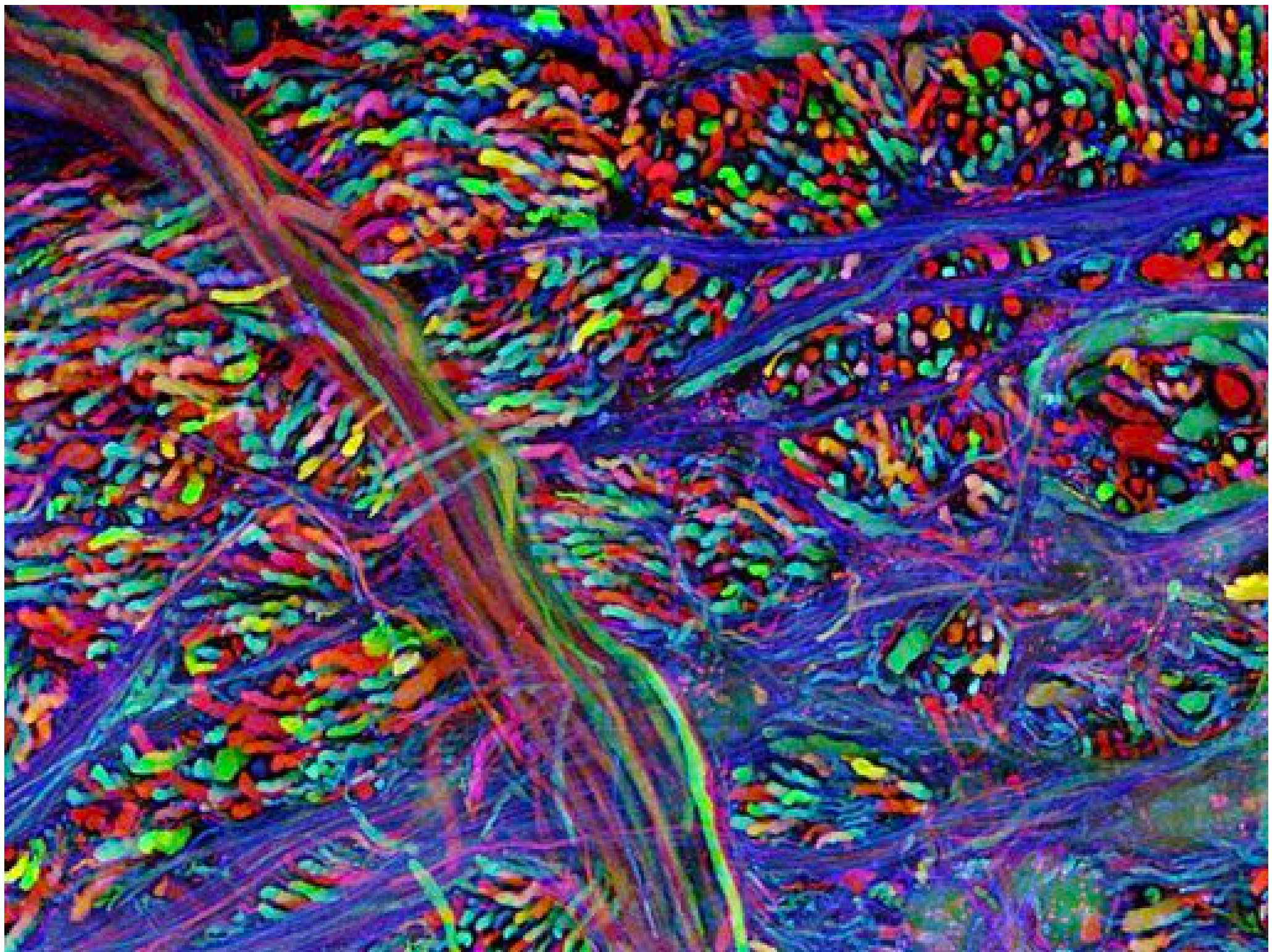


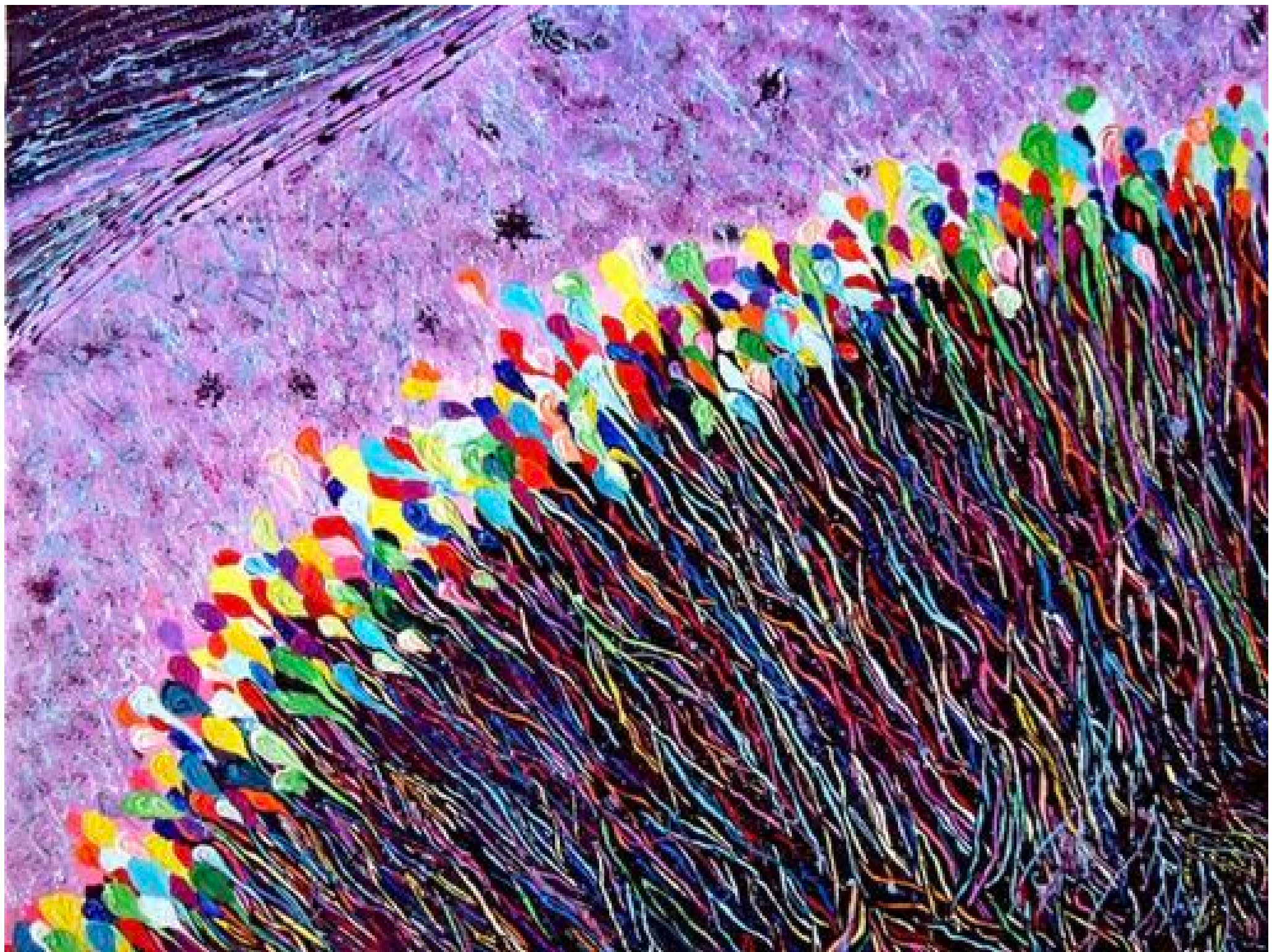


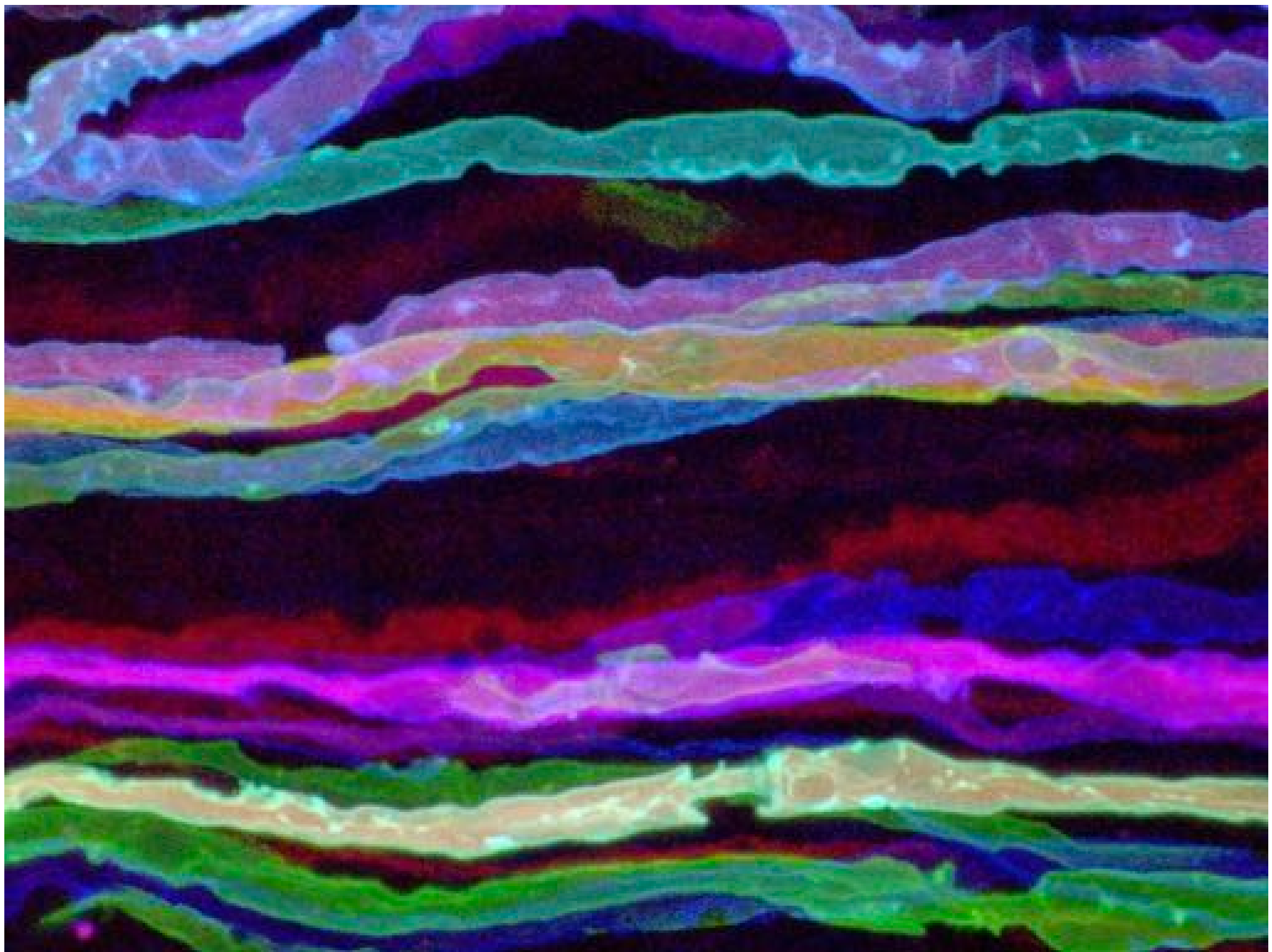


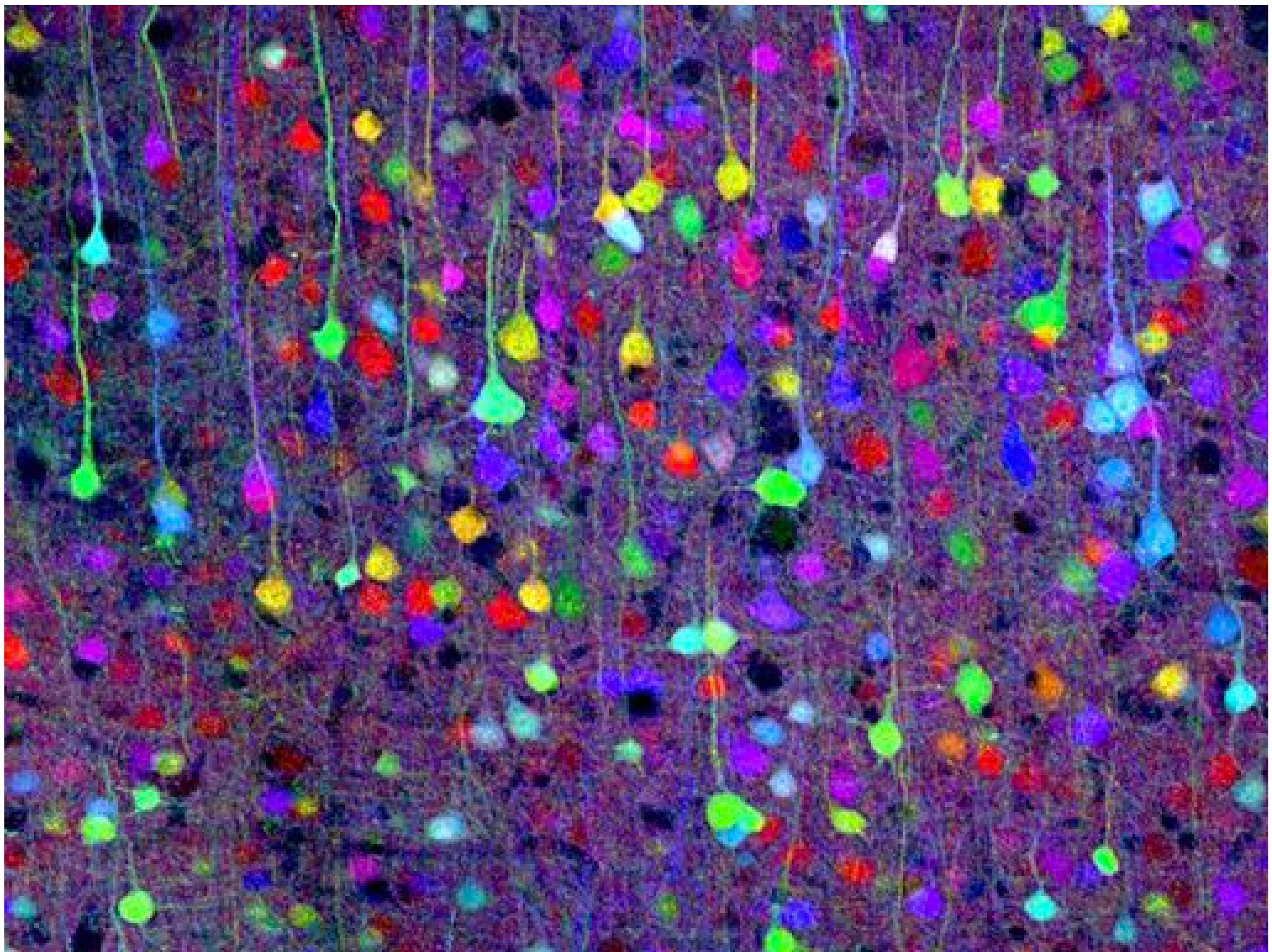


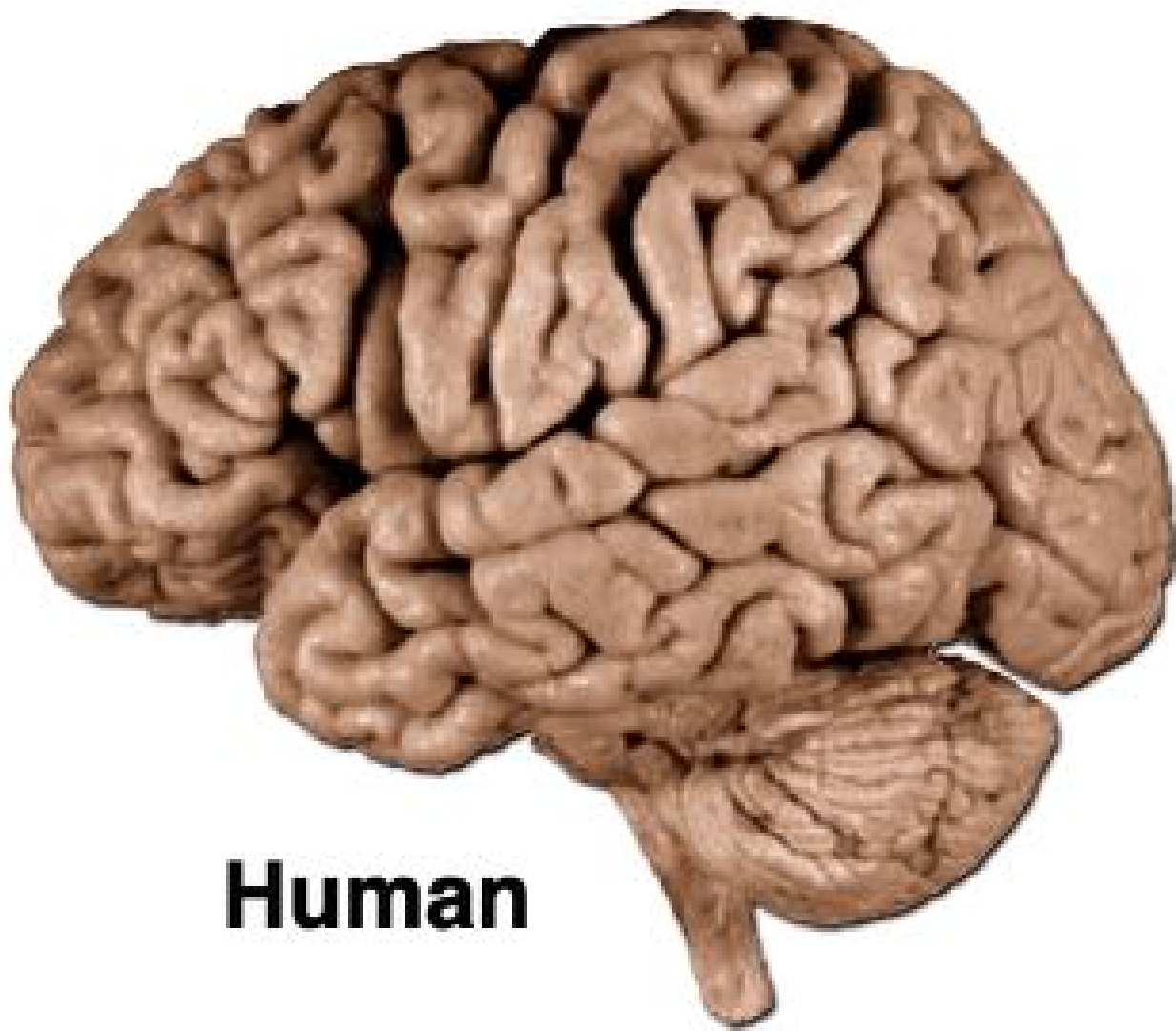










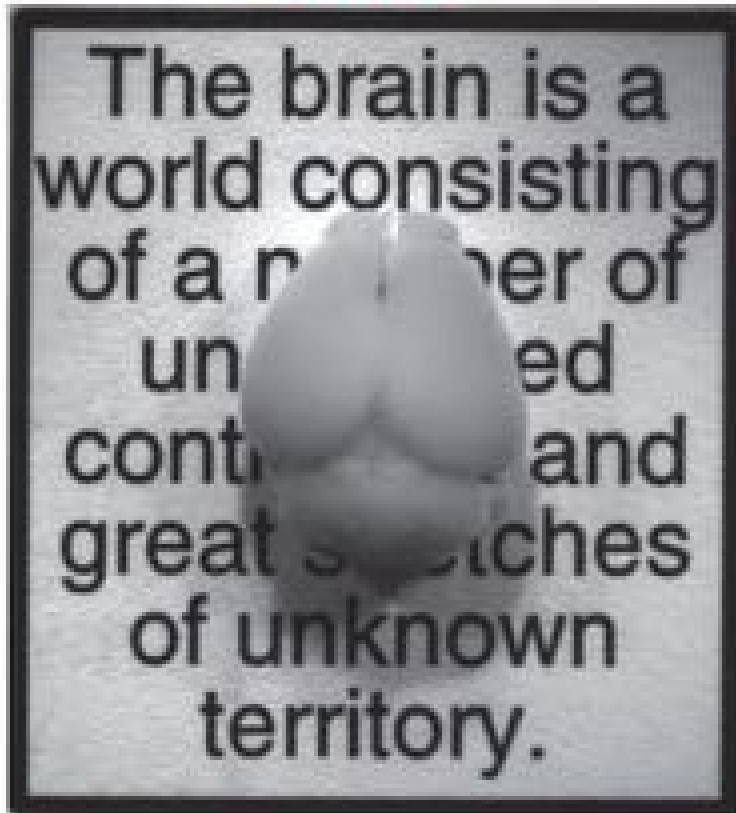


Human

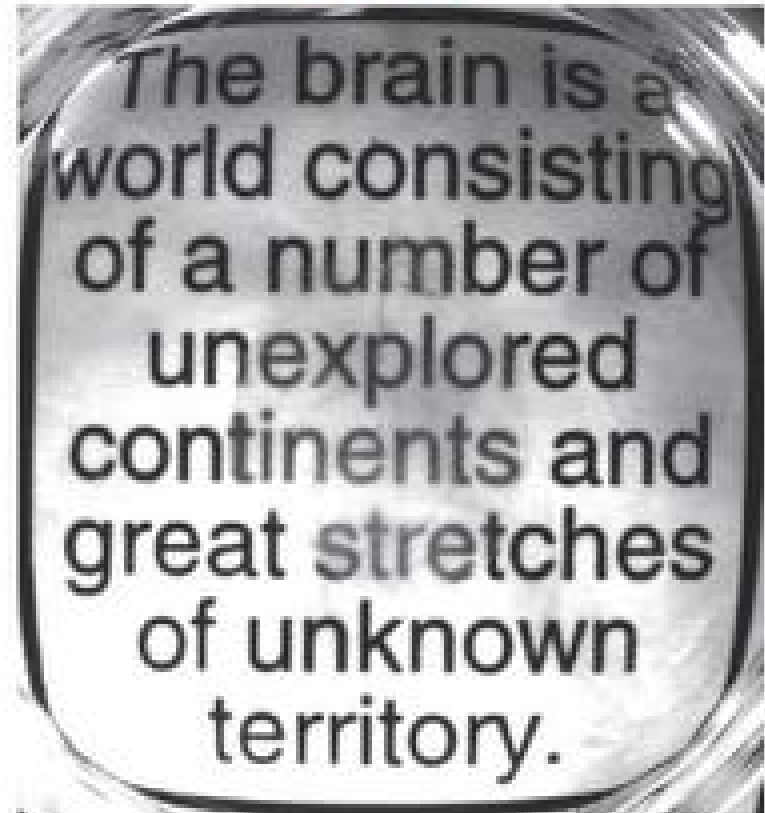


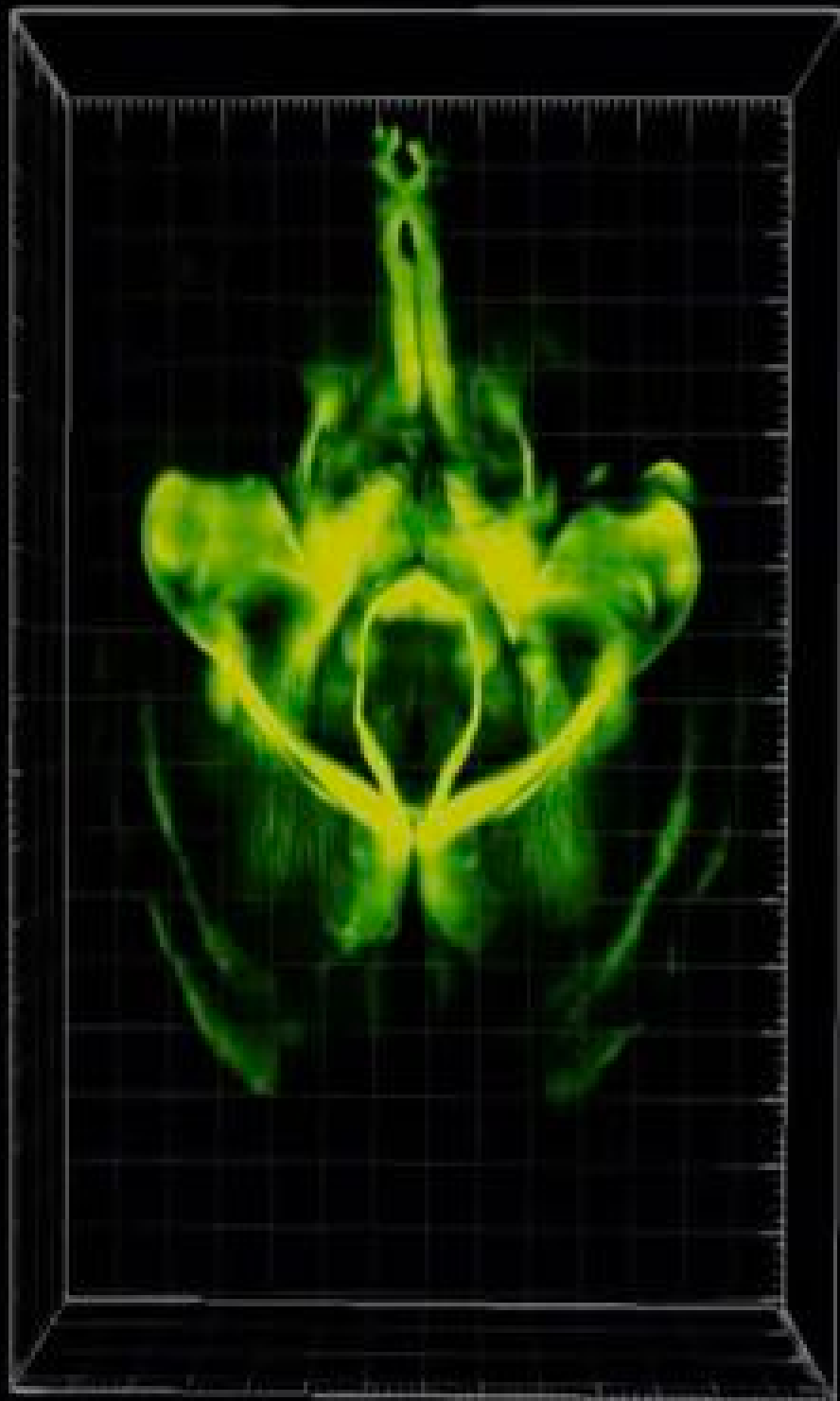
Mouse

Before

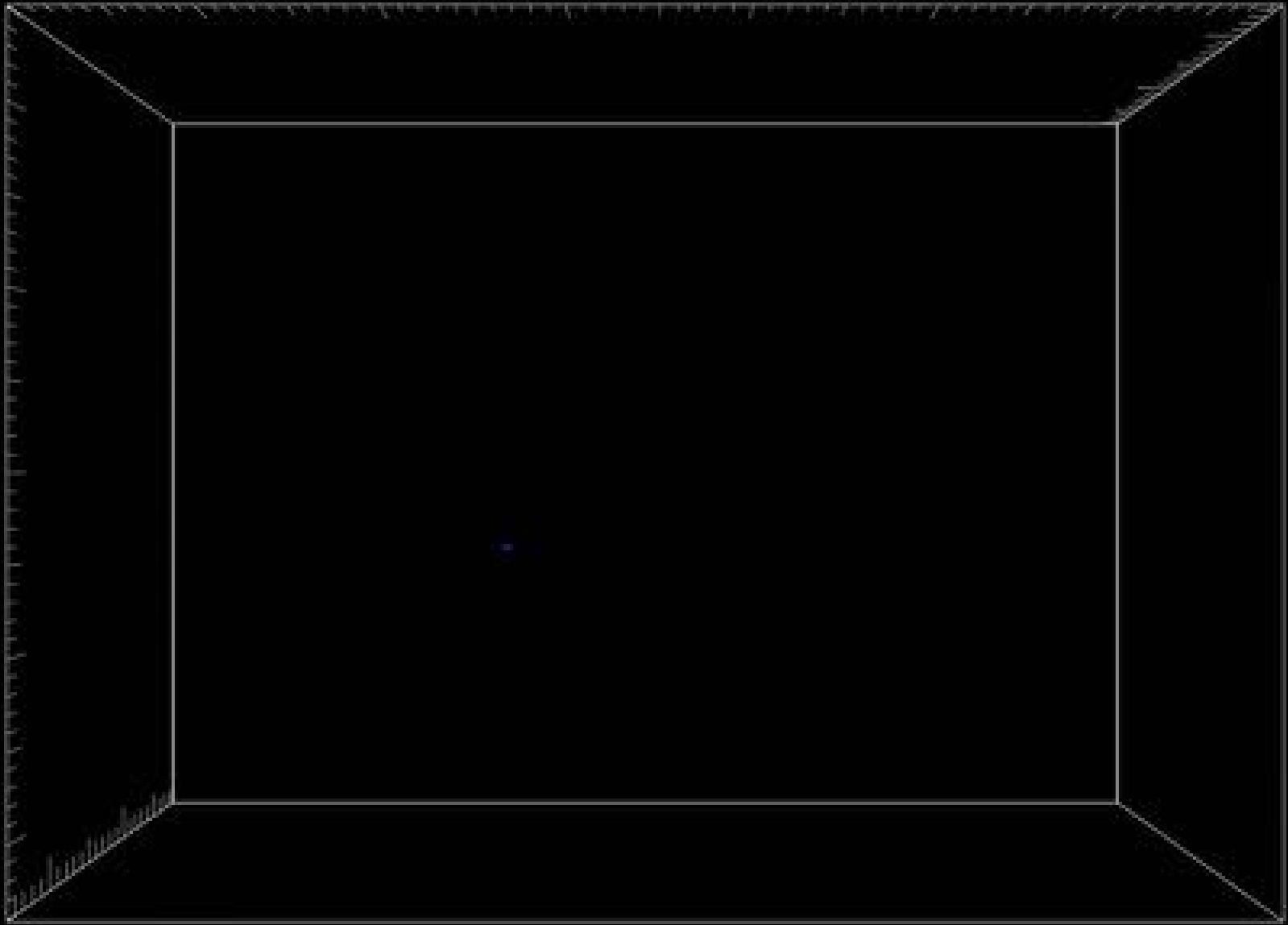


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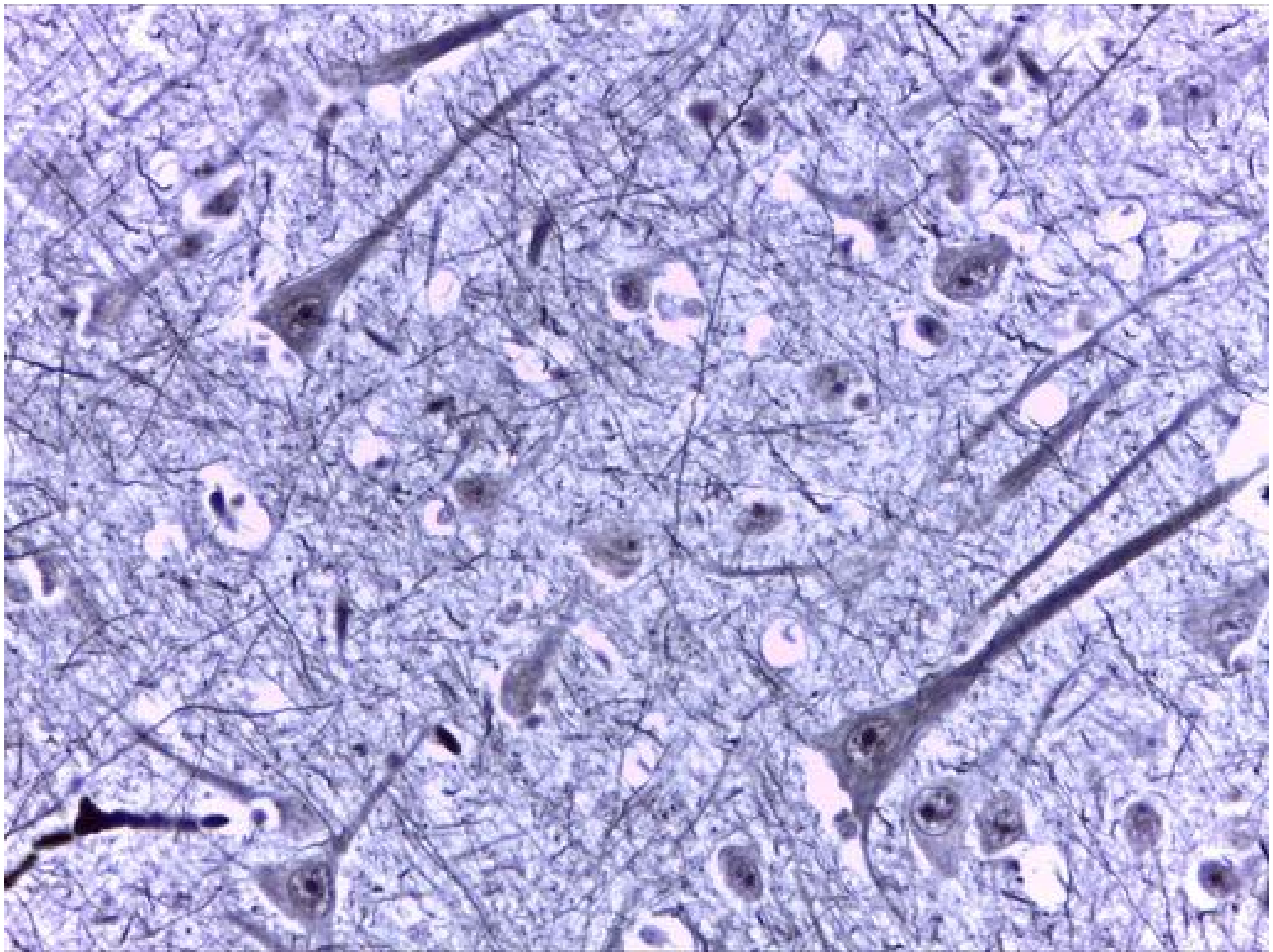




200 μm



100 μm





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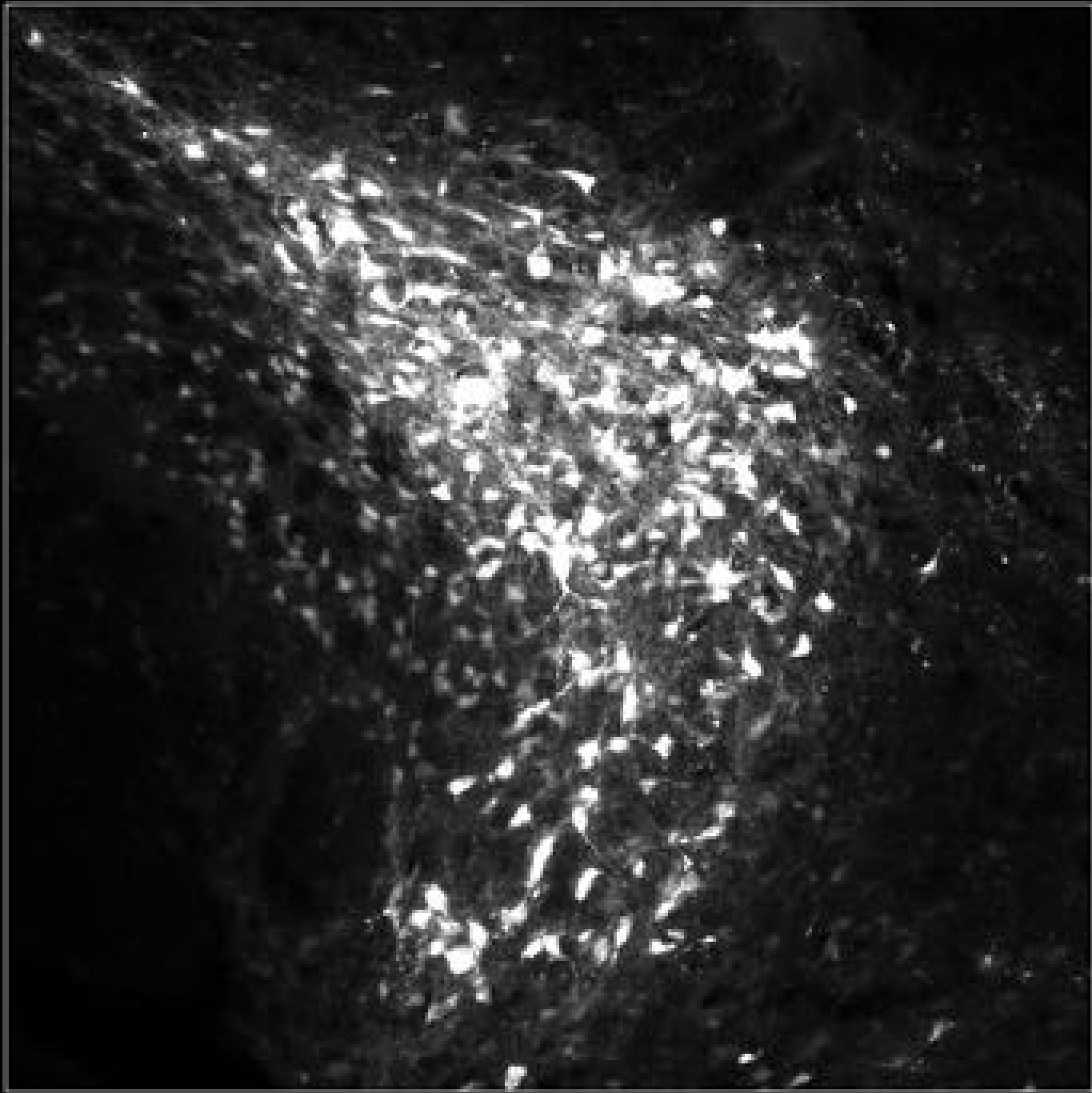
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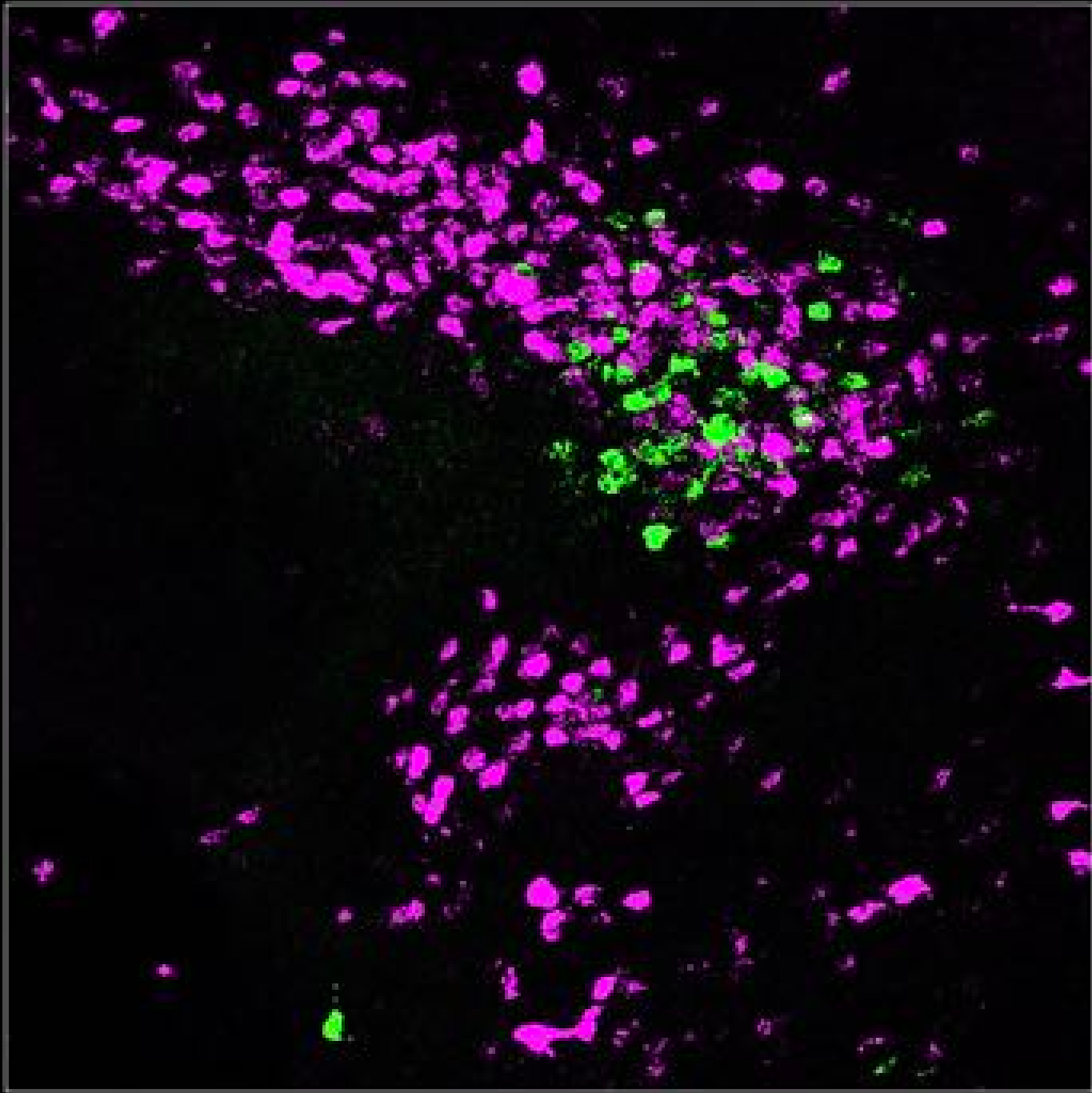
Editor's evaluation

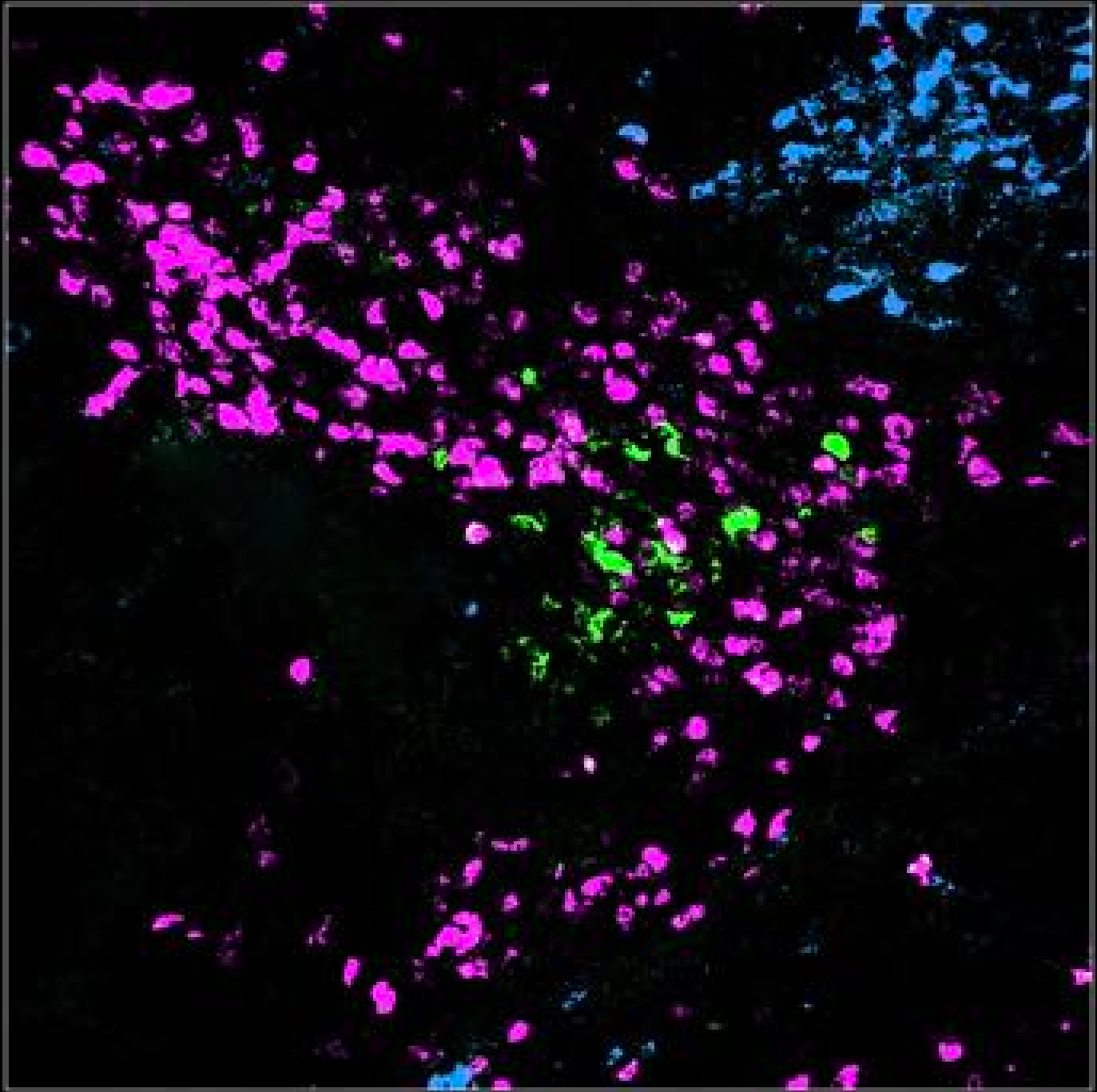
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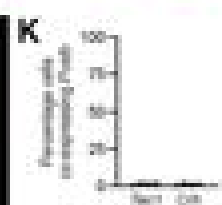
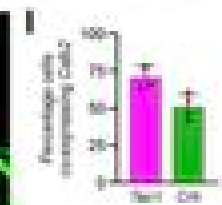
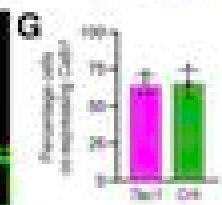
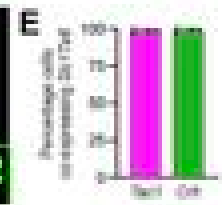
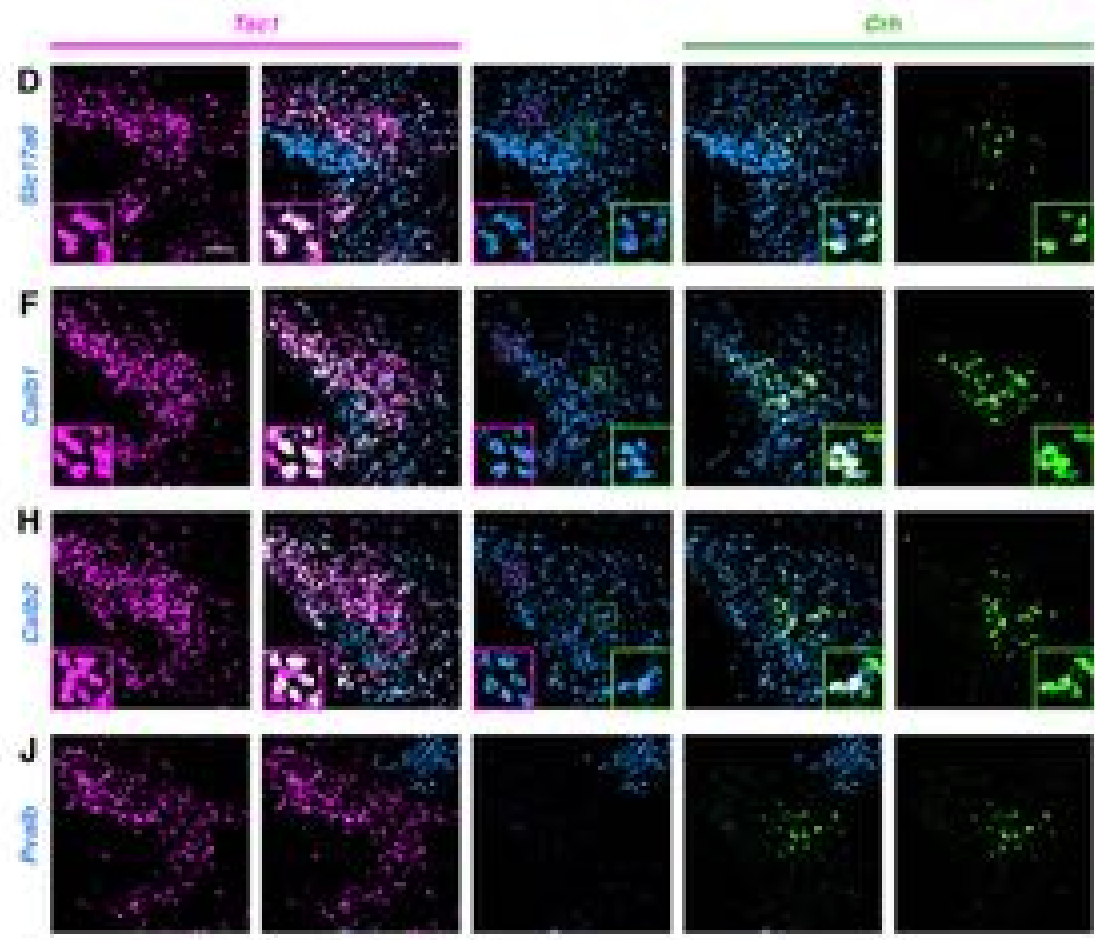
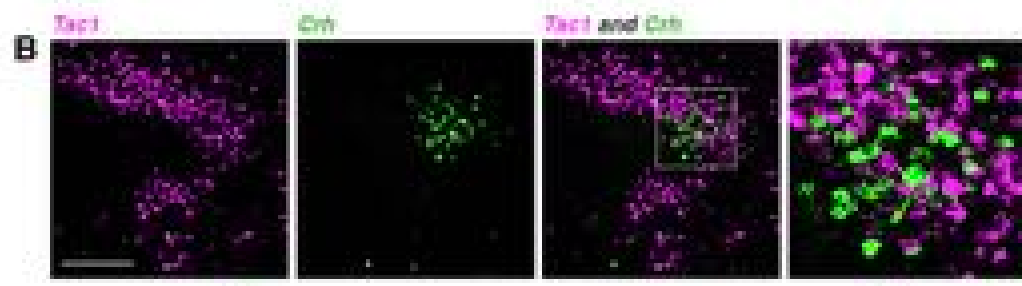
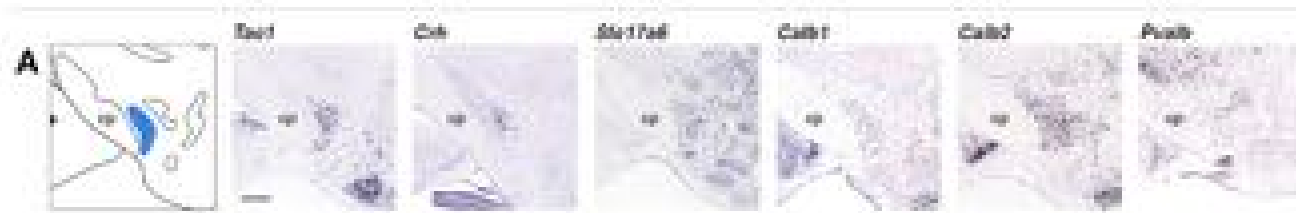
Introduction

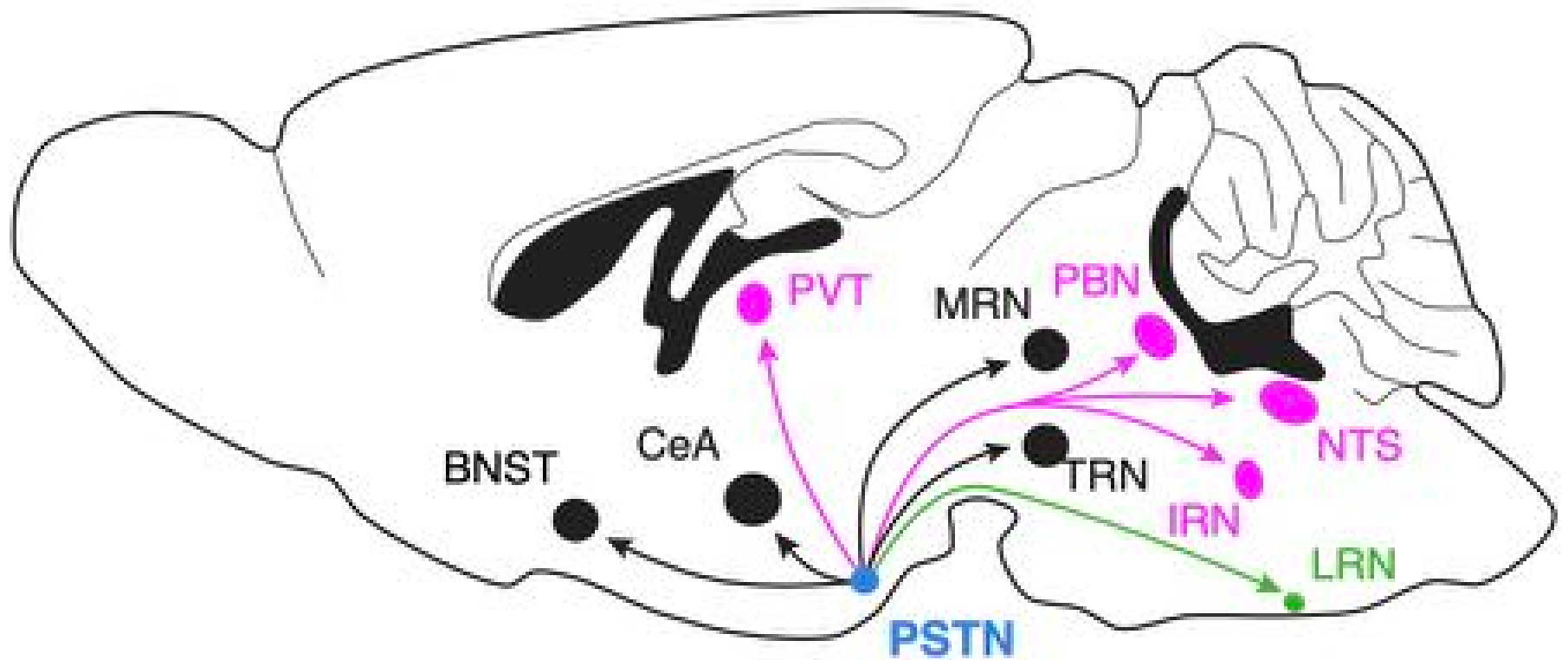
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— *Tac1* — *Crh* — *Tac1 and Crh*

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Neural Structure

Neural Activity

Neuromodulation



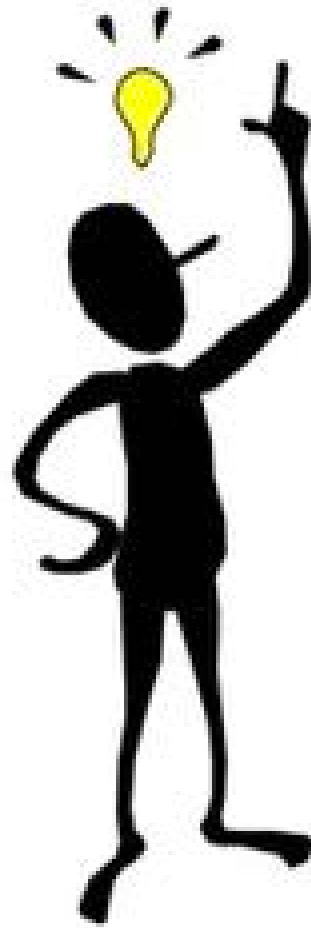
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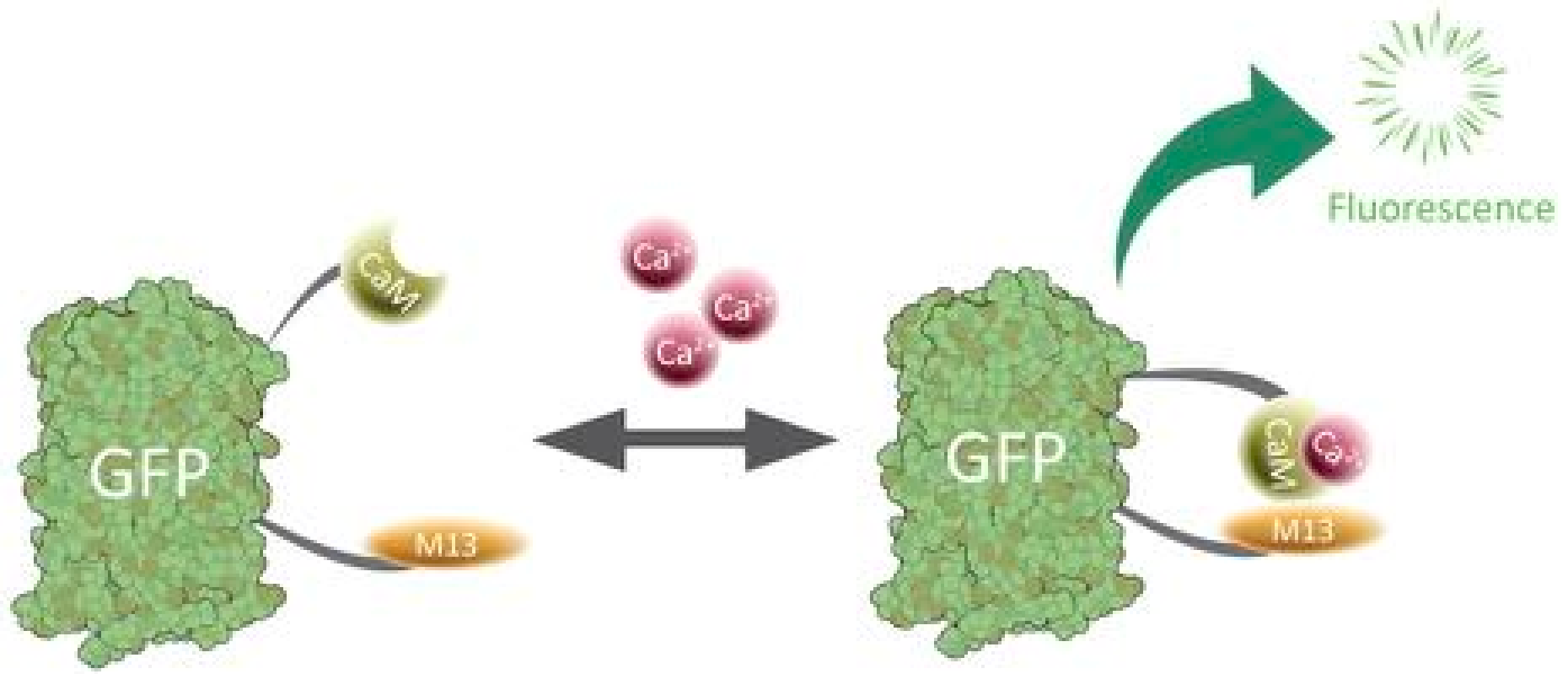
Neural Structure

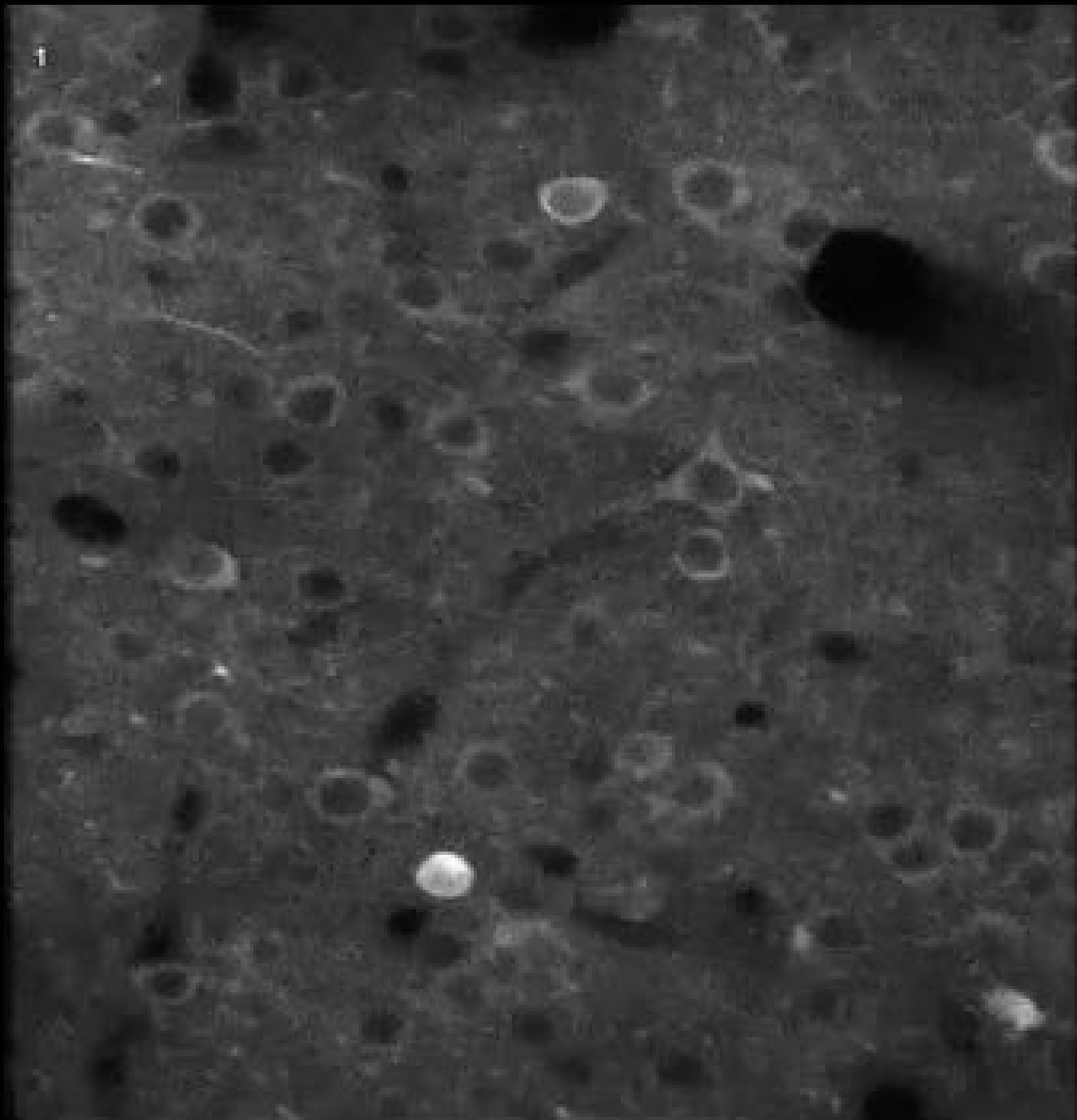
Neural Activity

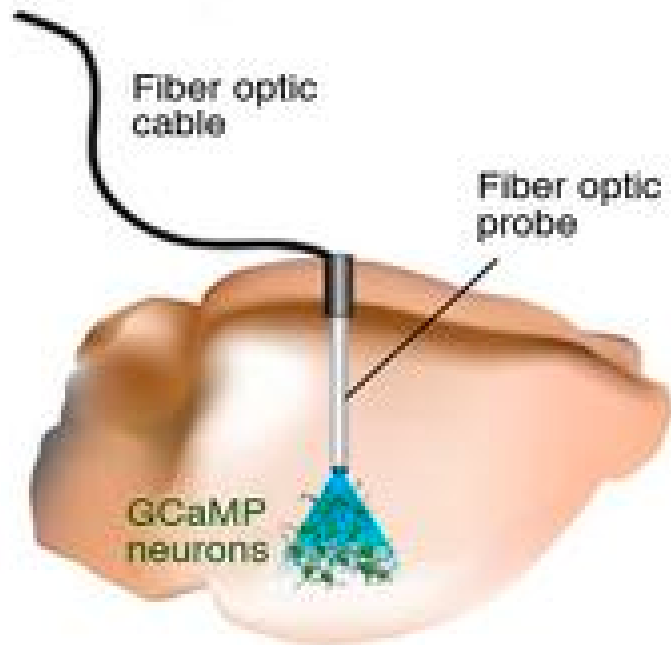
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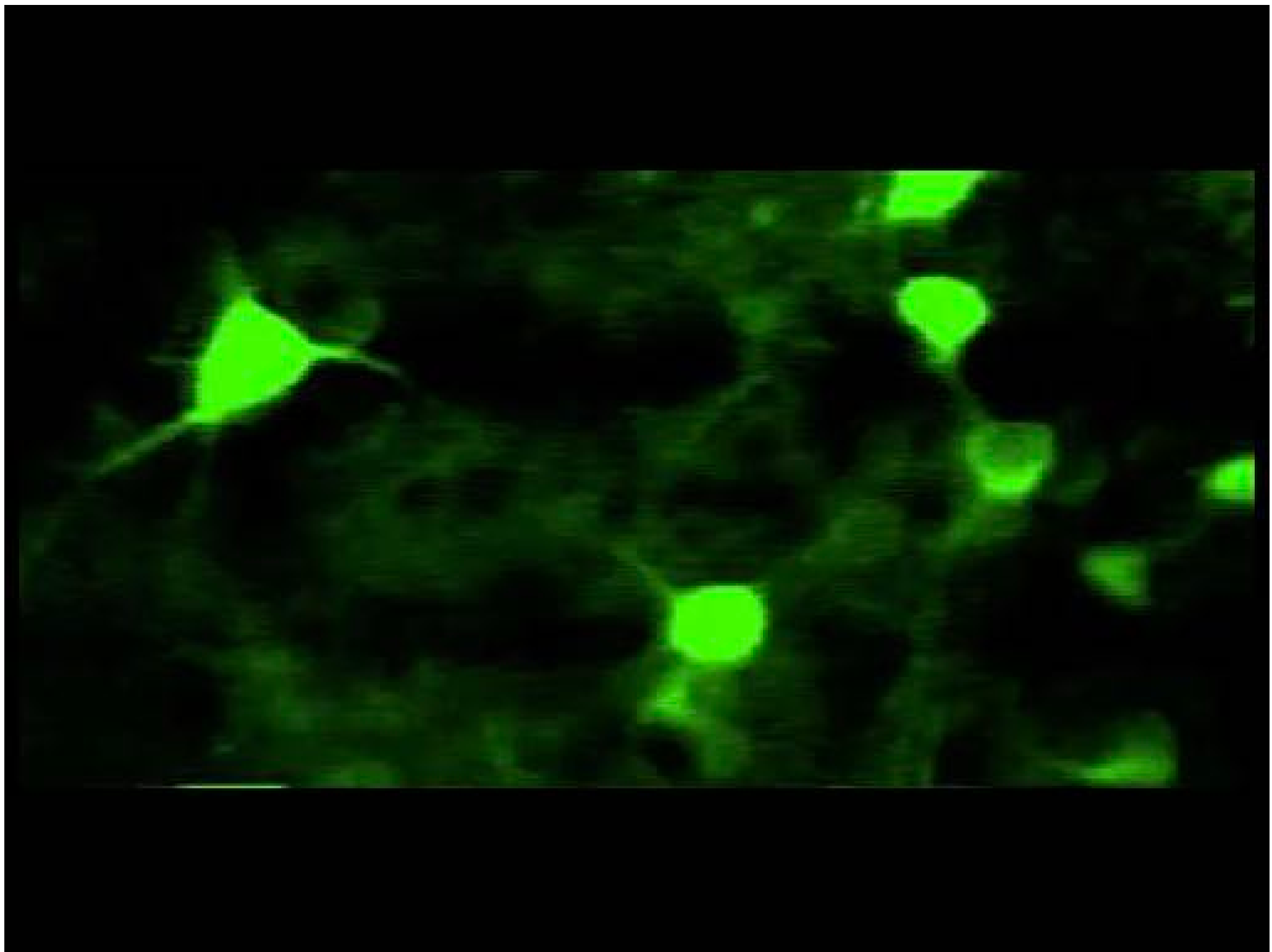


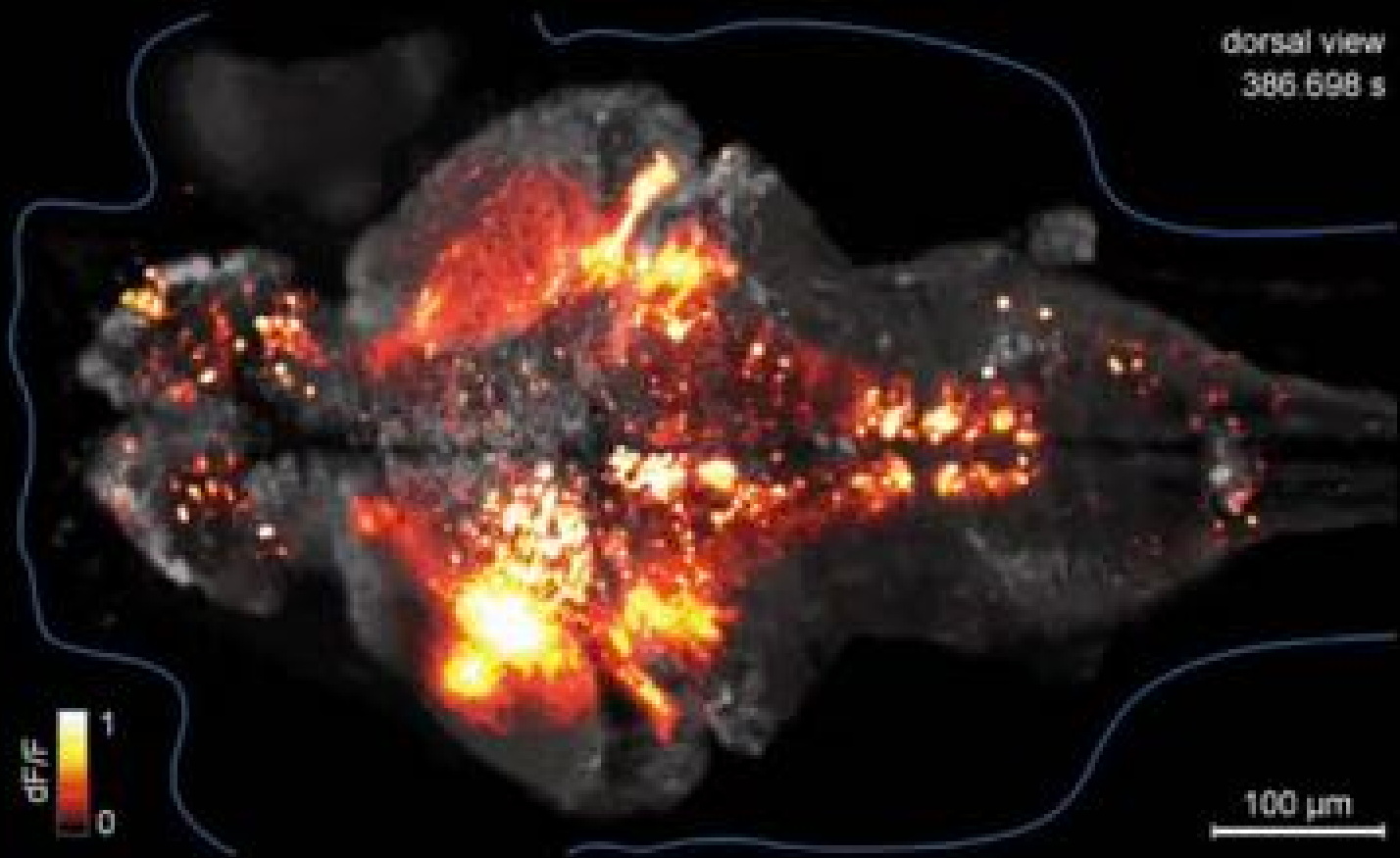




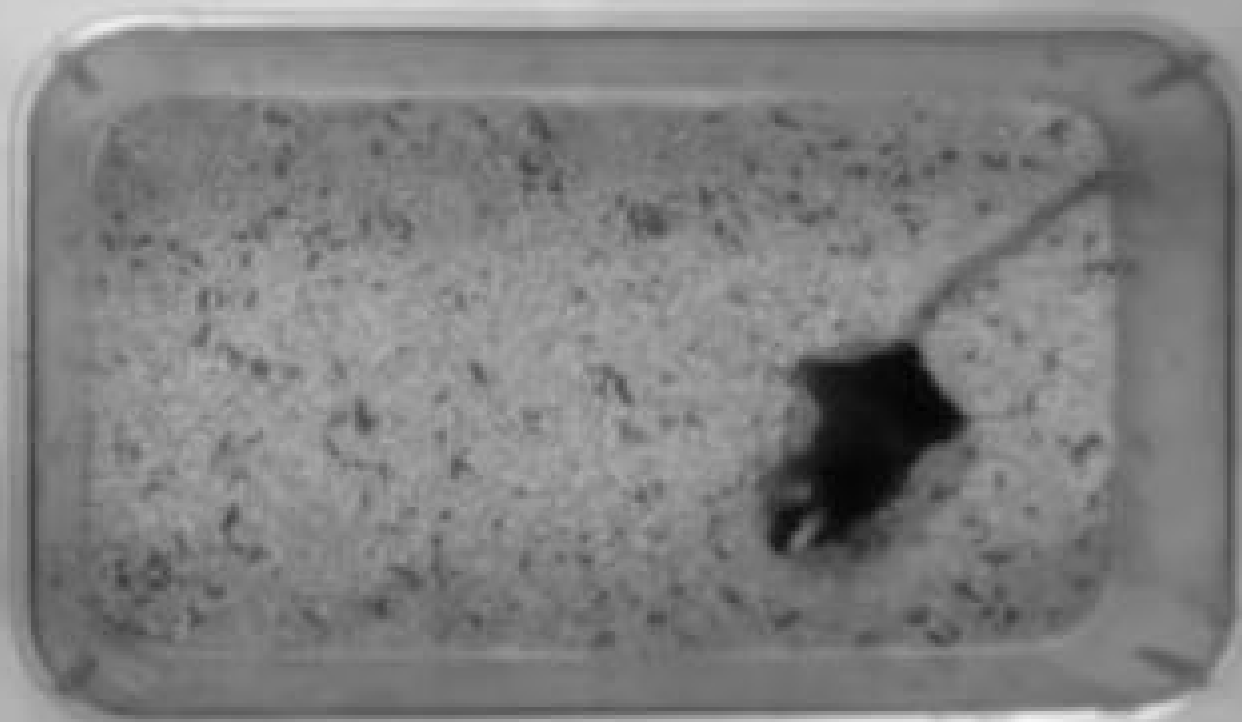








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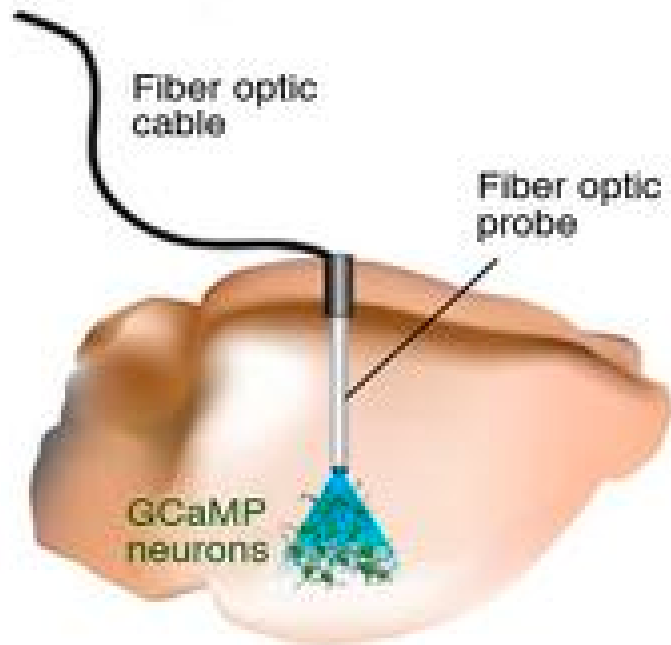
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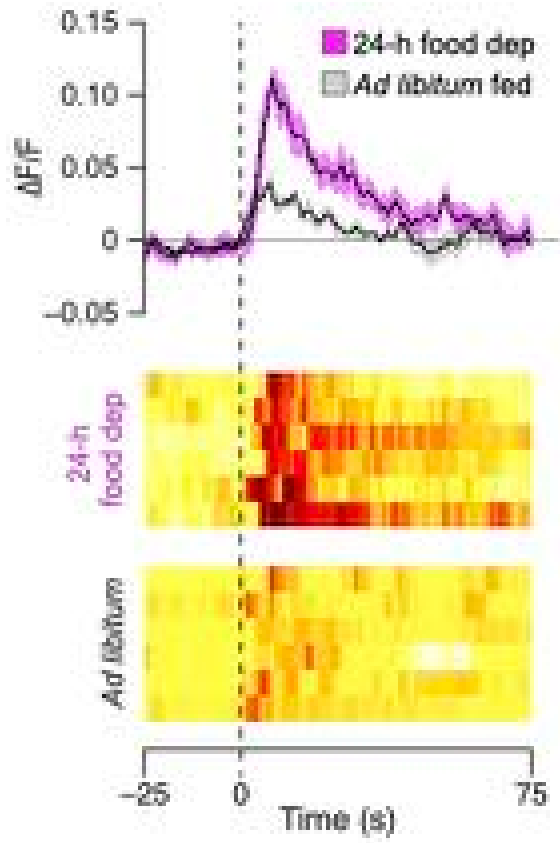
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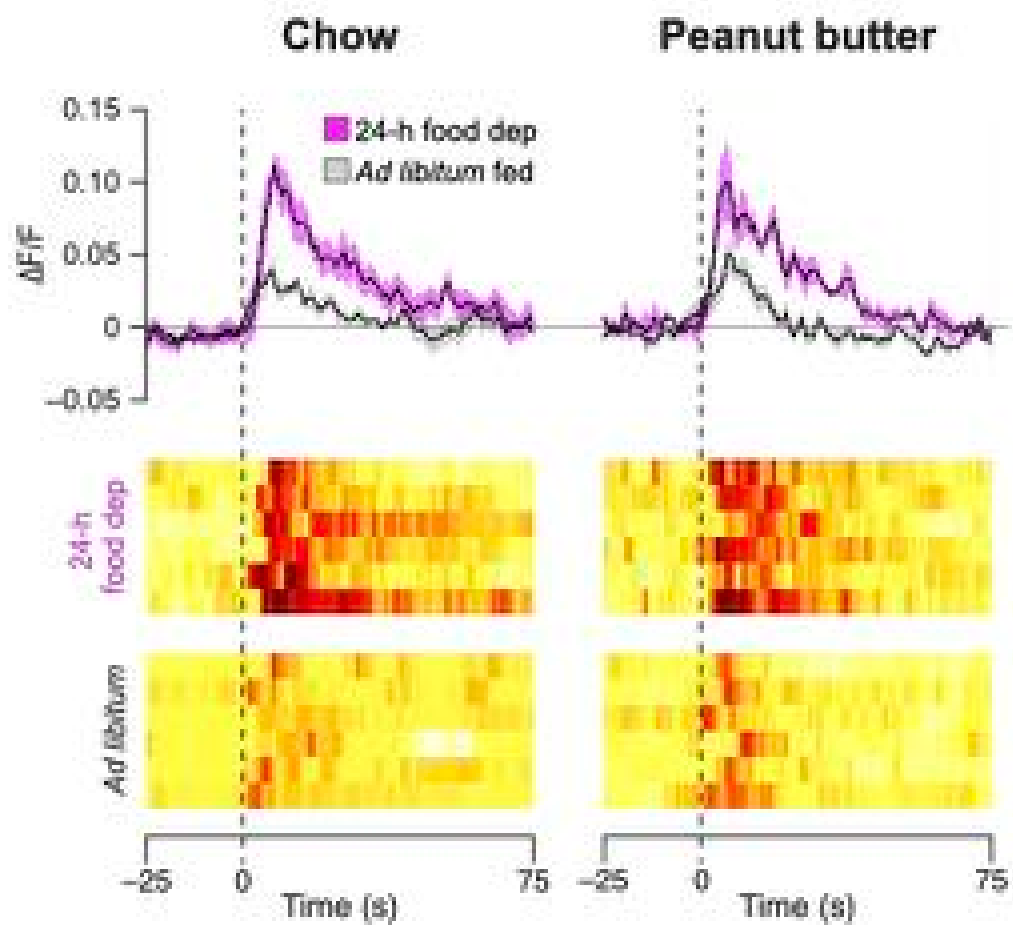
Introduction

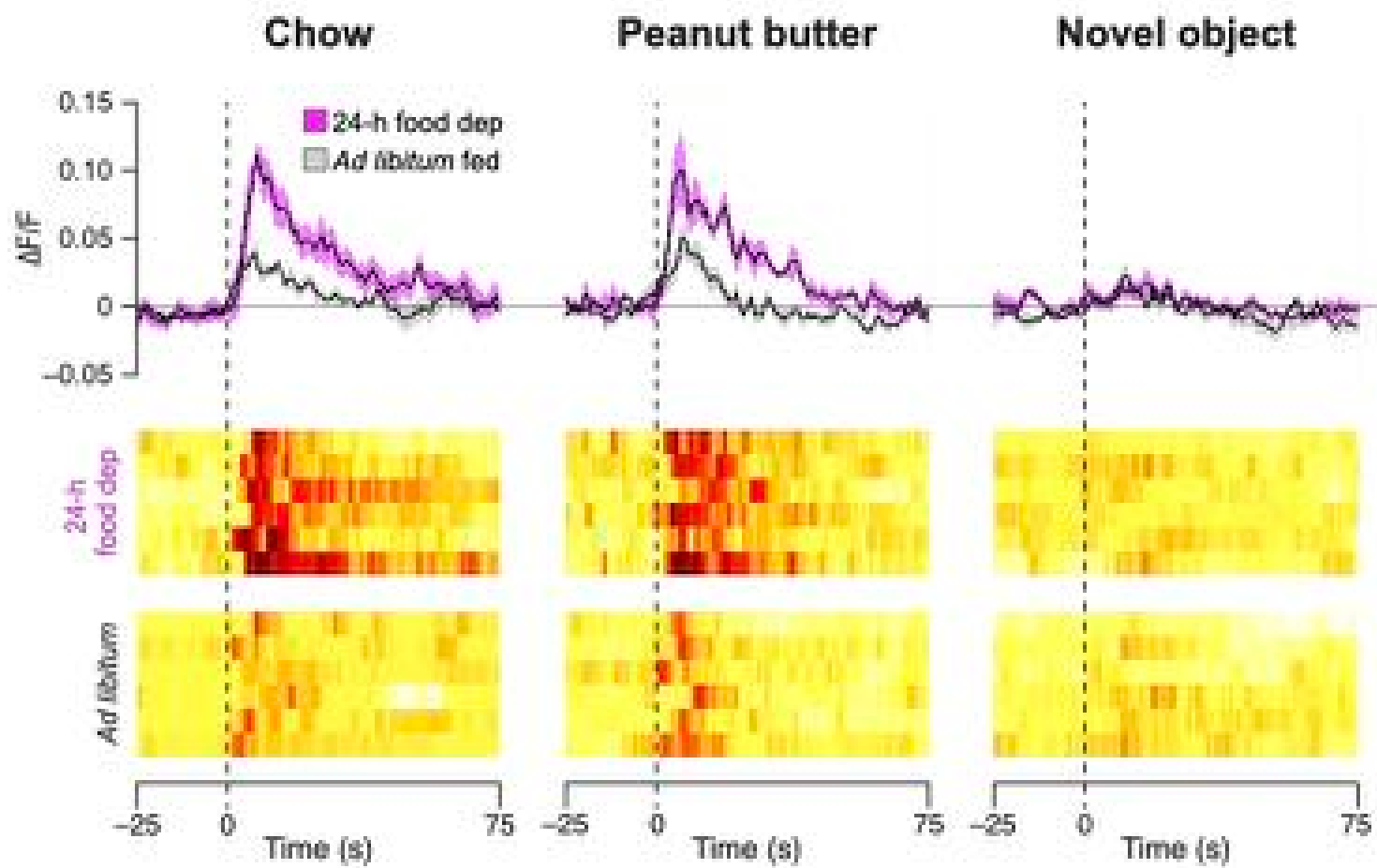
The brain regulates food intake behavior through the coordinated activity of several distinct neuronal populations (Anderson and Lowell, 2012; Stevenson and Bell, 2012). Activity in anorexigenic

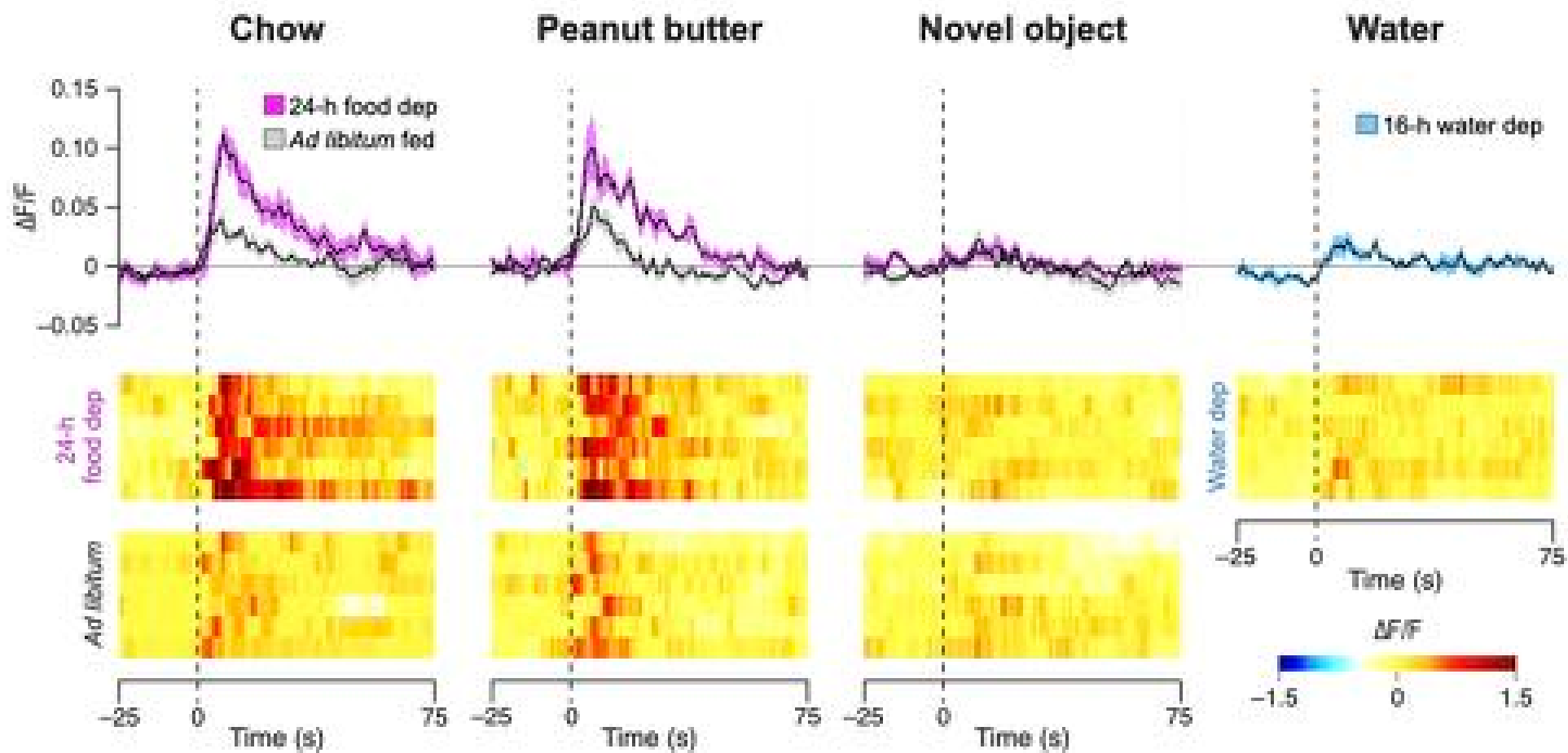


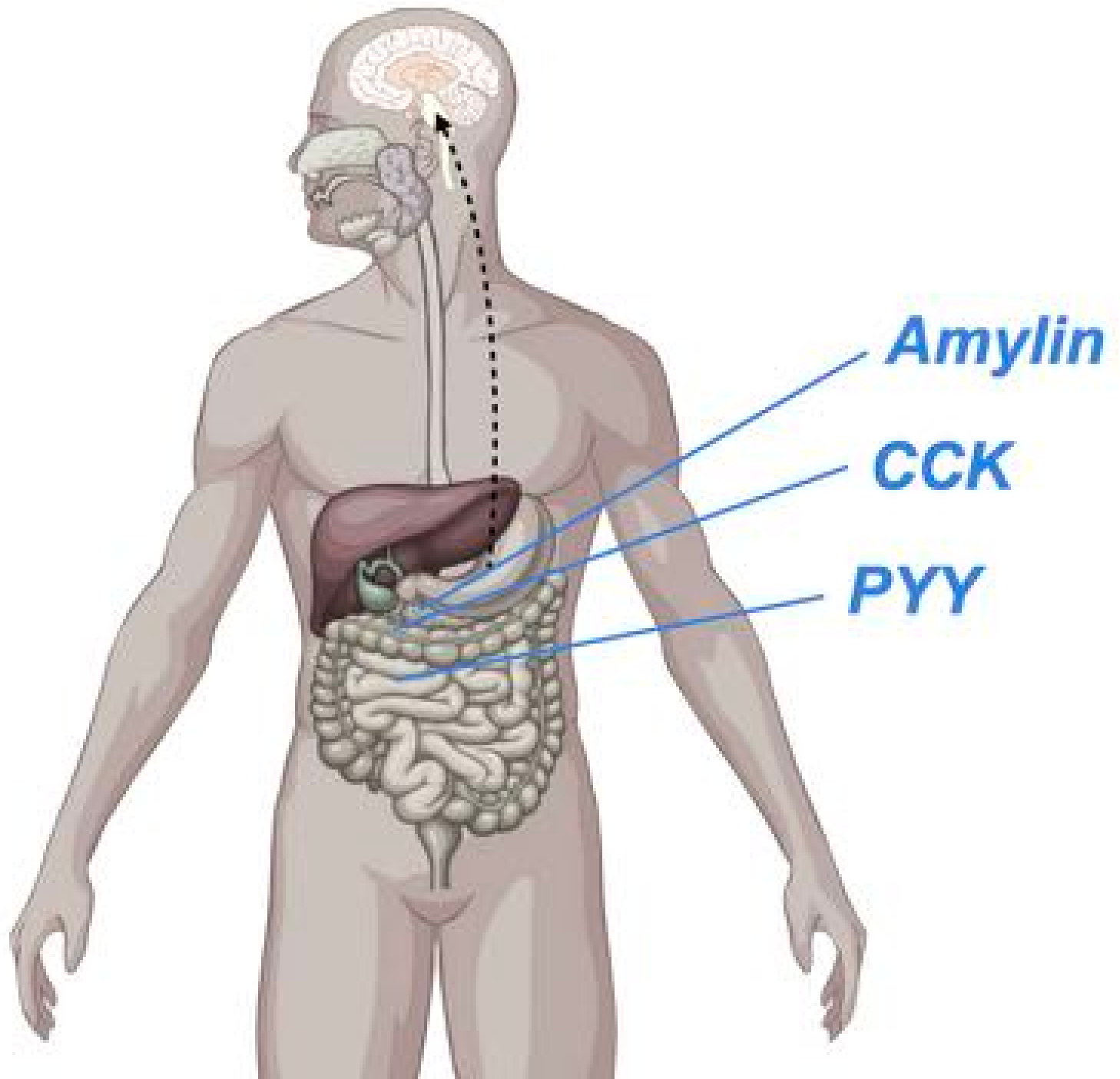
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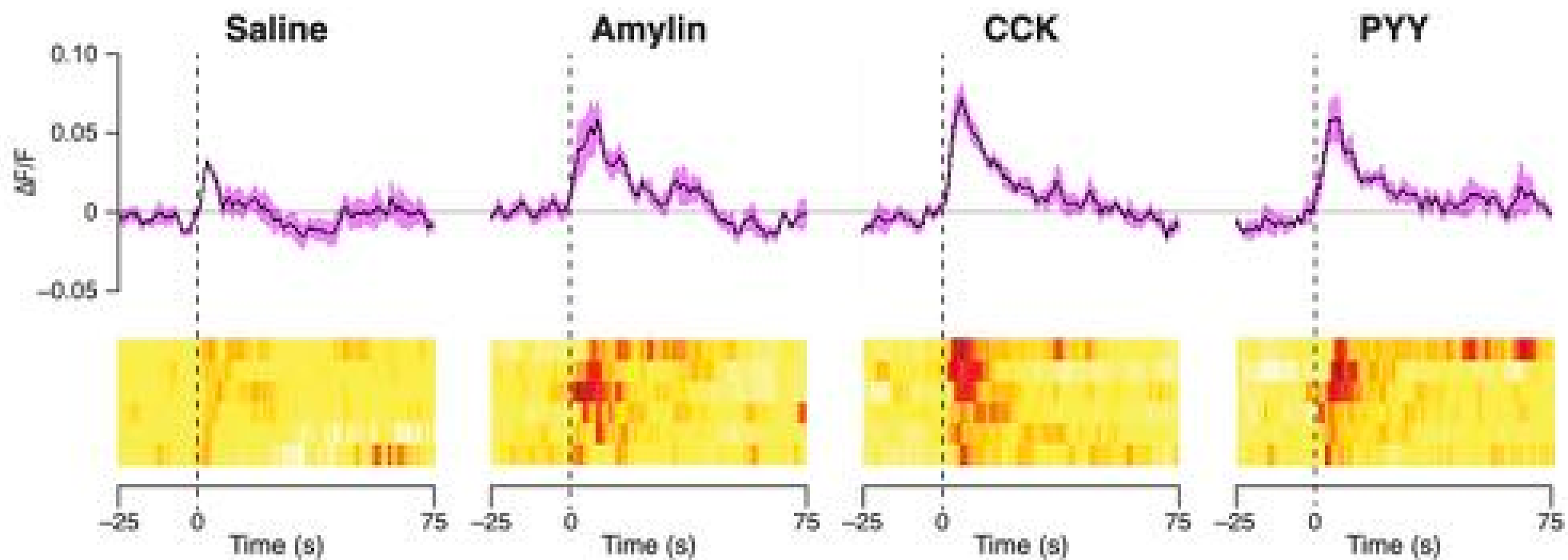












Adding a bit of **color** and **light** to the brain

Neural Structure

Neural Activity

Neuromodulation



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Neural Structure

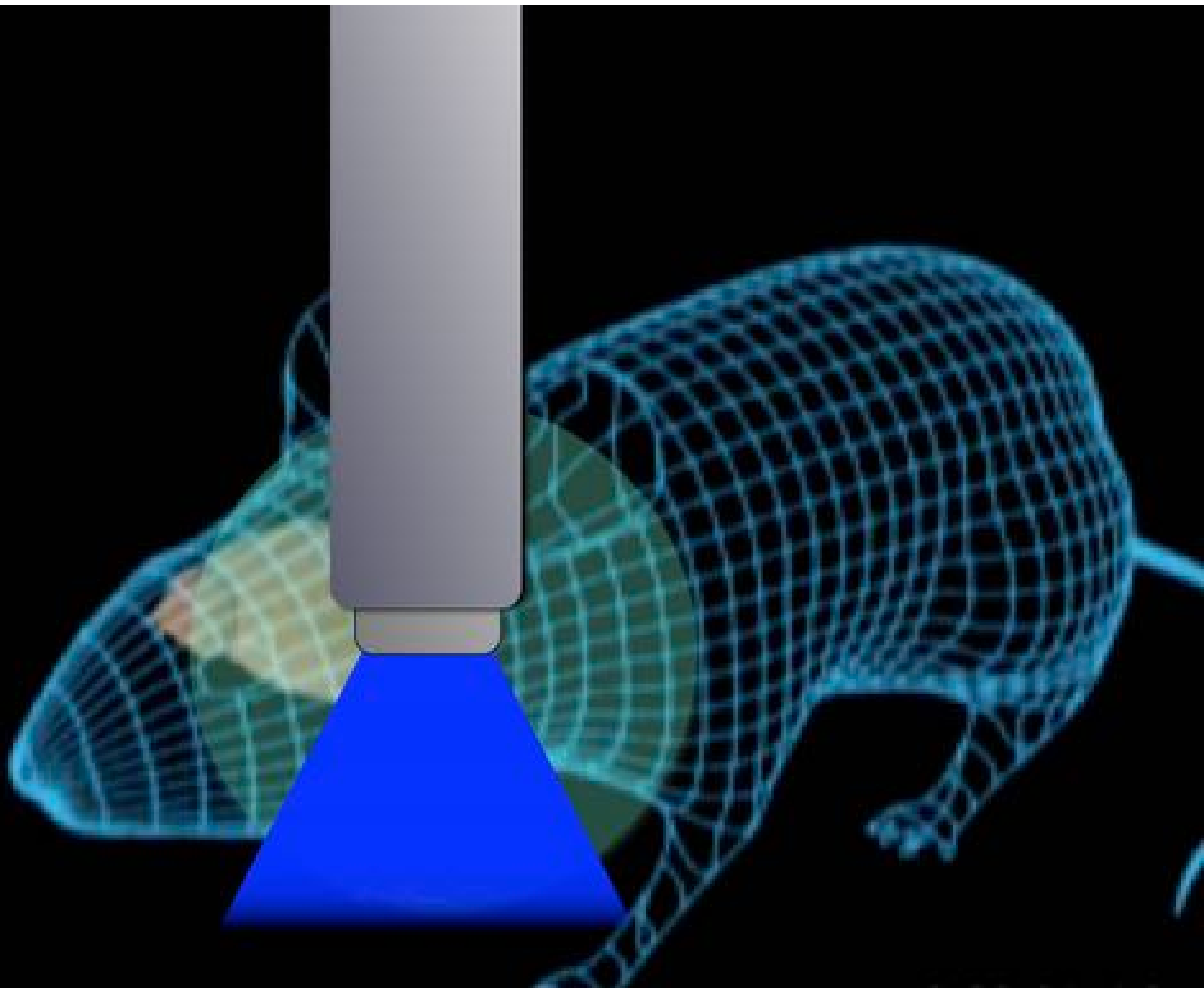
Neural Activity

Neuromodulation









Fiberoptic Control of Locomotion in ChR2 Mouse







A discrete parasubthalamic nucleus subpopulation plays a critical role in appetite suppression

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Abstract: Food intake behavior is regulated by a network of appetite-inducing and appetite-suppressing neuronal populations throughout the brain. The parasubthalamic nucleus (PSTN), a relatively unexplored population of neurons in the posterior hypothalamus, has been hypothesized to regulate appetite due to its connectivity with other anorexigenic neuronal populations and because these neurons express *Fox1*, a marker of neuronal activation following a meal. However, the individual cell types that make up the PSTN are not well characterized, nor are their functional roles in food intake behavior. Here, we identify and distinguish between two discrete PSTN subpopulations, those that express neuropeptide Y (PSTN^{NPY} neurons) and those that express corticotropin-releasing hormone (PSTN^{CRH} neurons), and use a panel of genetically encoded tools in mice to show that PSTN^{CRH} neurons play an important role in appetite suppression. Both subpopulations increase activity following a meal and in response to administration of the anorexigenic hormones amylin, cholecystikinin (CCK), and peptide YY (PYY). Interestingly, chemogenetic inhibition of PSTN^{CRH}, but not PSTN^{NPY} neurons, reduces the appetite-suppressing effects of these hormones. Consistently, optogenetic and chemogenetic stimulation of PSTN^{CRH} neurons, but not PSTN^{NPY} neurons, reduces food intake in hungry mice. PSTN^{CRH} and PSTN^{NPY} neurons project to distinct downstream brain regions, and stimulation of PSTN^{CRH} projections to individual anorexigenic populations reduces food consumption. Taken together, these results reveal the functional properties and projection patterns of distinct PSTN cell types and demonstrate an anorexigenic role for PSTN^{CRH} neurons in the hormonal and central regulation of appetite.

Editor's evaluation

This work has identified a previously unexplored role of the parasubthalamic nucleus in the regulation of feeding behavior. The combination of genetic and pharmacological approaches nicely demonstrates the physiological role of this group of neurons in regulating appetite. These studies will be of interest to the field and more broadly to the readers of eLife.

Introduction

The brain regulates food intake behavior through the coordinated activity of several distinct neuronal populations (Anderson and Lowell, 2012; Stevenson and Bell, 2012). Activity in anorexigenic



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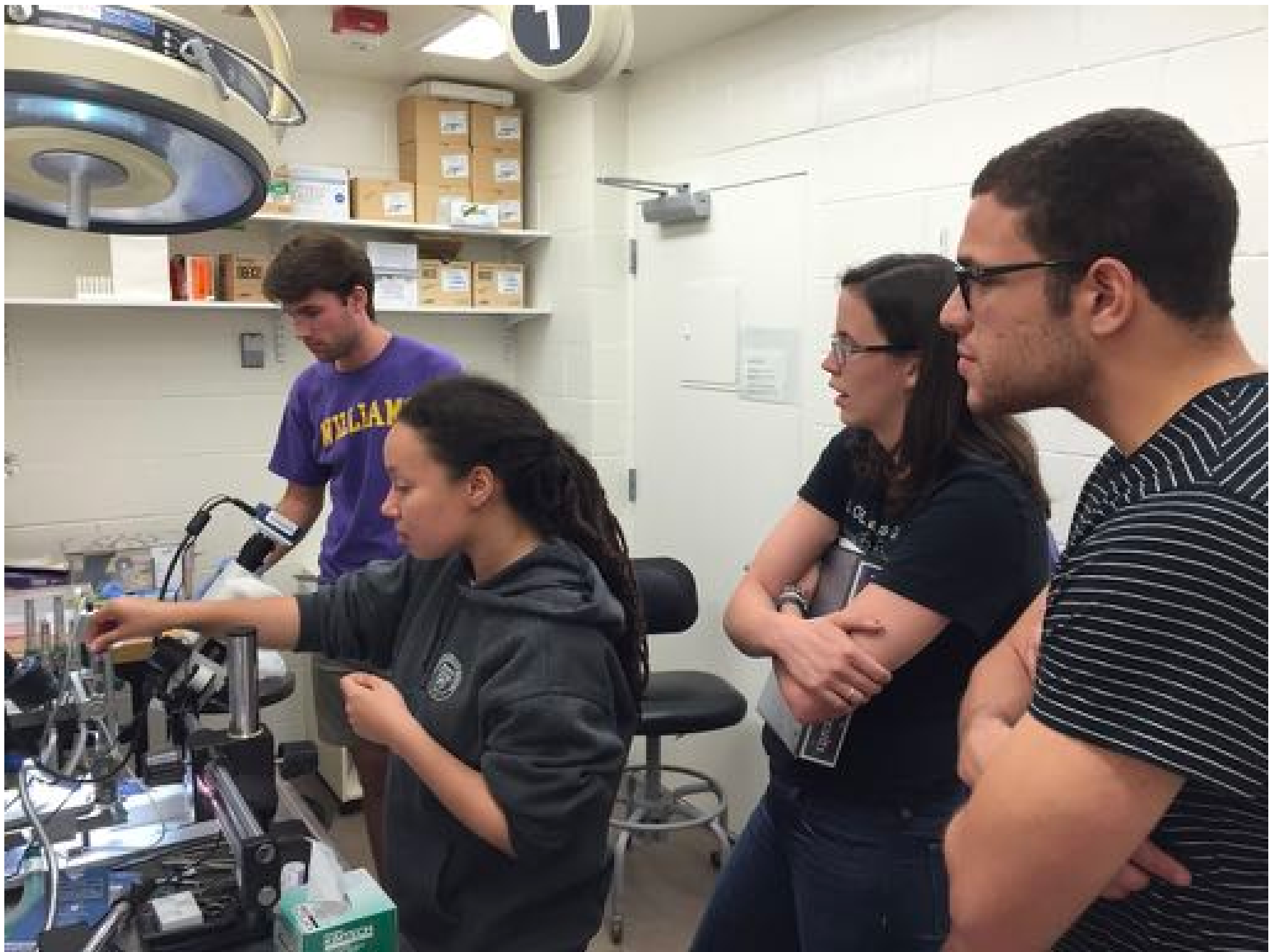
Neural Structure

Neural Activity

Neuromodulation













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Stimulation of AgRP neurons eliminates the effects of appetite suppressing compounds

Rachel Eason and Brad Carter | Department of Biology, Williams College, Williamstown, MA | 2018 Annual Meeting of the Society for Neuroscience, November 11, 2018

Background

AgRP neurons in the hypothalamus regulate feeding behavior. Stimulation of these neurons increases feeding, while inhibition decreases it. We investigated whether stimulation of AgRP neurons could overcome the effects of appetite-suppressing compounds.



Methods

AgRP neurons were stimulated using optogenetics (ChR2). Mice were treated with appetite suppressants (amprolium, CCK, and Lorcaserin) and then stimulated. Feeding was measured by weight gain and food intake.

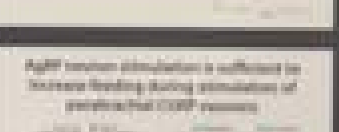
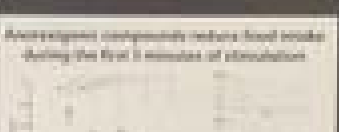
Validation of gene expression and AgRP neuron activation



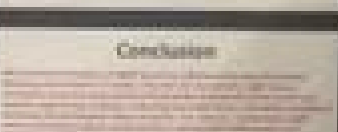
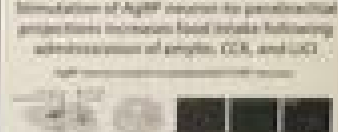
AgRP neuron stimulation is sufficient to increase feeding following administration of amprolium, CCK, and Lorcaserin (LPS)



AgRP neuron stimulation eliminates the appetite-suppressing effects of amprolium, CCK, and Lorcaserin (LPS)



AgRP neuron stimulation reduces expression of Fos protein in paraventricular (PV) neurons



Appetite-suppressant returns food intake during the first 3 minutes of stimulation



Stimulation of AgRP neurons in paraventricular projections increases food intake following administration of amprolium, CCK, and Lorcaserin



AgRP neuron stimulation is sufficient to increase feeding during stimulation of paraventricular (PV) neurons



Conclusion

Stimulation of AgRP neurons in the hypothalamus is sufficient to increase feeding and overcome the effects of appetite-suppressing compounds. This suggests that AgRP neurons act as a central hub for feeding regulation.



The Effects of AgRP Neuron Stimulation During Appetite Suppression

Anna Smith and Matt Carter, Williams College, Williamstown, MA | 2016 Winter Conference on Brain Research

Background

The regulation of food intake depends on a balance of anorexigenic (appetite-suppressing) and orexigenic (appetite-stimulating) signals. The hypothalamus integrates these signals to determine appropriate feeding behavior.



Emergence of anorexigenic compounds of AgRP neurons during starvation... (text partially obscured)

Experimental design

We used a... (text partially obscured)



During starvation... (text partially obscured)

1. Starvation
2. AgRP stimulation
3. Food intake measurement



AgRP neuron stimulation is insufficient to increase food intake under anorexic conditions



AgRP stimulation does not increase food intake under anorexic conditions.



AgRP stimulation does not increase food intake under anorexic conditions.



AgRP stimulation does not increase food intake under anorexic conditions.



AgRP stimulation does not increase food intake under anorexic conditions.

Validation that photostimulation increases AgRP neuron activity

Calcium imaging of AgRP neurons during photostimulation.



Effect of anorexic compounds on an individual mouse



Conclusions and future directions

Our results show that AgRP stimulation is insufficient to increase food intake under anorexic conditions. This suggests that other factors, such as leptin, may play a role in regulating food intake during starvation.

Future directions include investigating the role of other hypothalamic neurons in regulating food intake during starvation.



AgRP Neurons Can Disrupt Sleep/Wake Architecture and Cause Deficits in Rapid Eye Movement (REM) Sleep

Ashley Lee, Neeraj Kulkarni, and Brad Carter - Williams College, Williamstown, MA - 2018 Student Conference on Brain Research

Background

These studies in *Drosophila* are the first to demonstrate that AgRP neurons can disrupt sleep/wake architecture and cause deficits in REM sleep. We found that AgRP neurons are necessary for normal sleep/wake architecture and that their activation during sleep causes a shift in sleep/wake architecture, including a decrease in REM sleep.

AgRP neurons are known to be involved in feeding behavior and energy balance. We hypothesized that AgRP neurons might also be involved in sleep/wake architecture. We tested this hypothesis by activating AgRP neurons during sleep and measuring the effects on sleep/wake architecture.



Our results show that AgRP neuron activation during sleep causes a decrease in REM sleep and a shift in sleep/wake architecture. These findings suggest that AgRP neurons may be involved in the regulation of sleep/wake architecture.

Experimental Design

We used a genetic approach to activate AgRP neurons during sleep. We used a temperature-sensitive Gal4 driver to express a temperature-sensitive Cre recombinase in AgRP neurons. We then used a temperature-sensitive Cre recombinase to activate AgRP neurons during sleep.

We measured sleep/wake architecture using a sleep/wake monitoring system. We found that AgRP neuron activation during sleep causes a decrease in REM sleep and a shift in sleep/wake architecture.

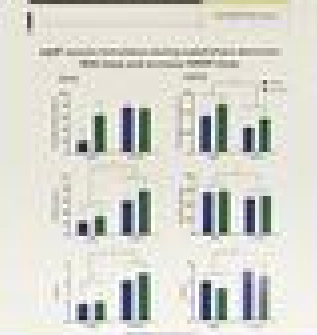
Validation of AgRP photostimulation

We first validated our AgRP photostimulation protocol by measuring the effects of AgRP photostimulation on feeding behavior. We found that AgRP photostimulation causes a decrease in feeding behavior.



AgRP neuron photostimulation during wakefulness affects subsequent sleep

We next tested whether AgRP neuron photostimulation during wakefulness affects subsequent sleep. We found that AgRP neuron photostimulation during wakefulness causes a decrease in REM sleep and a shift in sleep/wake architecture.



AgRP neuron photostimulation during sleep affects sleep/wake architecture

We then tested whether AgRP neuron photostimulation during sleep affects sleep/wake architecture. We found that AgRP neuron photostimulation during sleep causes a decrease in REM sleep and a shift in sleep/wake architecture.



Conclusion

Our results show that AgRP neurons are necessary for normal sleep/wake architecture and that their activation during sleep causes a decrease in REM sleep and a shift in sleep/wake architecture. These findings suggest that AgRP neurons may be involved in the regulation of sleep/wake architecture.

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Y29

Identification of Discrete, Intermingled Hypocretin Neuronal Populations

Blanchard and Stern Center, Department of Biology, Williams College, Williamstown, MA 01267 Annual Meeting of the Society for Neuroscience, October 21, 2015

Background

Hypocretin (Hcrt) neurons in the lateral hypothalamus (LH) project to arousal and reward centers in the brain. Hcrt neurons are thought to be involved in the regulation of arousal and reward. However, the precise organization of Hcrt neurons and their projections to downstream targets remains unclear.



To address this question, we performed a detailed analysis of Hcrt neurons and their projections to arousal and reward centers. We used a combination of genetic and anatomical approaches to identify discrete populations of Hcrt neurons and their projections. We found that Hcrt neurons project to arousal centers with high fidelity, but project to reward centers with lower fidelity. This suggests that Hcrt neurons are organized into discrete populations that project to different downstream targets.

Approach

We used a combination of genetic and anatomical approaches to identify discrete populations of Hcrt neurons and their projections. We used Cre-loxP recombination to generate Hcrt-Cre mice, which allowed us to trace the projections of Hcrt neurons to downstream targets. We also used immunohistochemistry to identify Hcrt neurons and their projections.

Hypocretin neurons that project to arousal centers are distinct from hypocretin neurons that project to reward centers



Hypocretin neurons project to downstream arousal or reward centers with high fidelity



Hypocretin neuronal populations are not anatomically segregated into medial and lateral groups

We performed a detailed analysis of Hcrt neurons and their projections to arousal and reward centers. We found that Hcrt neurons are not anatomically segregated into medial and lateral groups. Instead, Hcrt neurons are organized into discrete populations that project to different downstream targets.



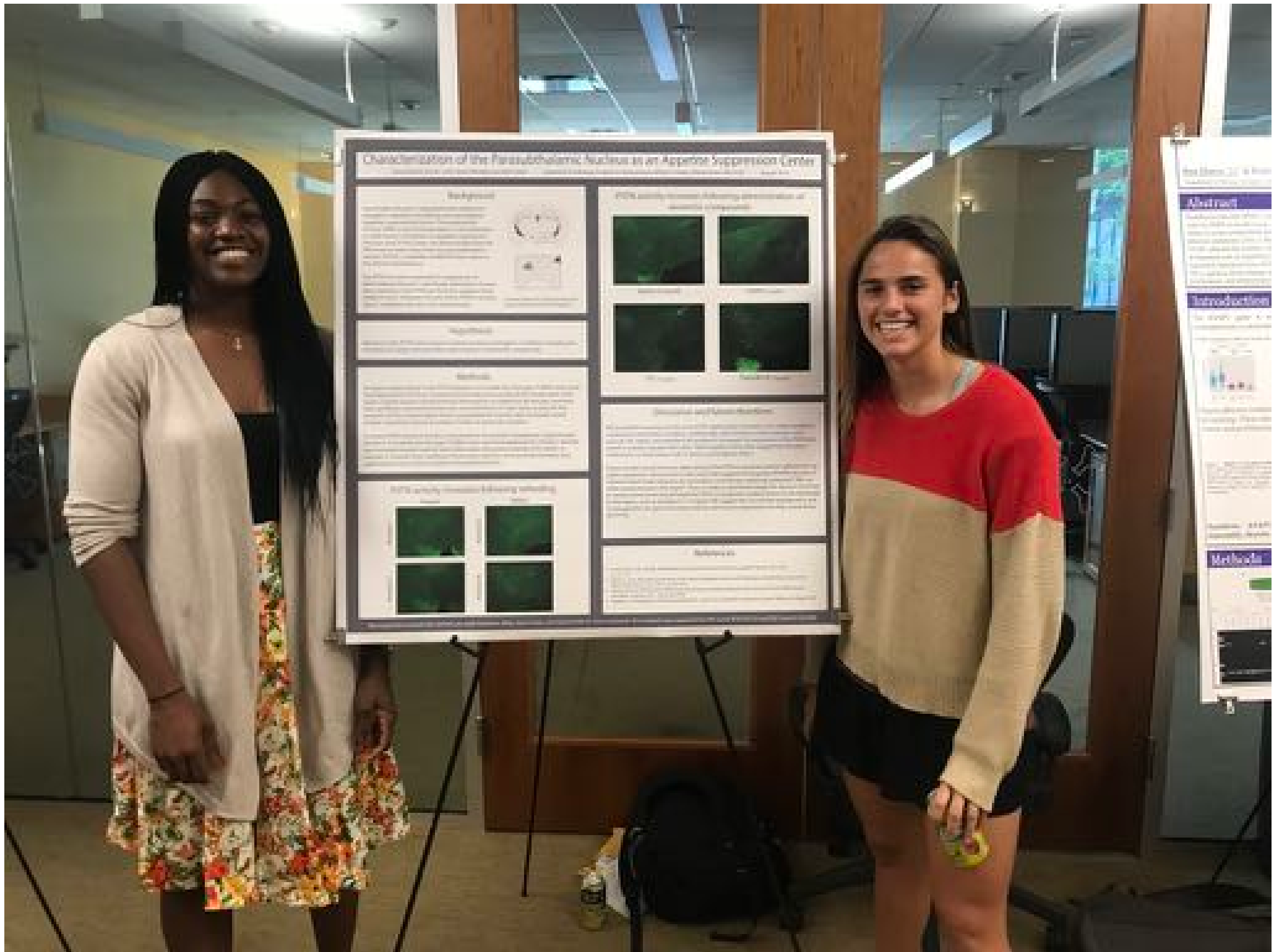
Conclusion

We found that Hcrt neurons project to arousal centers with high fidelity, but project to reward centers with lower fidelity. This suggests that Hcrt neurons are organized into discrete populations that project to different downstream targets. We also found that Hcrt neurons are not anatomically segregated into medial and lateral groups. Instead, Hcrt neurons are organized into discrete populations that project to different downstream targets.

References

- Blanchard, R. L., & Stern, R. M. (2015). Identification of discrete, intermingled hypocretin neuronal populations. *Journal of Neuroscience*, 35(42), 14345-14355.





Characterization of the Parasympathetic Nucleus as an Appetite Suppression Center

Background
The parasympathetic nucleus (PN) is a brain region that has been implicated in the regulation of appetite and energy balance. It is located in the brainstem and is part of the parasympathetic nervous system. The PN is thought to play a role in the regulation of the autonomic nervous system, which controls the body's internal organs and functions. The PN is also thought to be involved in the regulation of the hypothalamic-pituitary-adrenal axis, which is a key component of the stress response. The PN is a complex structure and its function is not fully understood. This study aims to characterize the PN as an appetite suppression center.



Hypothesis
The PN is a key component of the appetite suppression center and its activation leads to a decrease in food intake.

Methods
The study used a combination of behavioral, electrophysiological, and molecular biology techniques to investigate the role of the PN in appetite suppression. Behavioral studies were conducted to measure food intake and energy expenditure. Electrophysiological recordings were used to measure the activity of neurons in the PN. Molecular biology techniques were used to identify the genes and proteins involved in the PN's function.



Behavioral and Electrophysiological Results
The study found that activation of the PN leads to a significant decrease in food intake and an increase in energy expenditure. This effect was observed in both acute and chronic studies. Electrophysiological recordings showed that the PN is highly active during periods of food restriction and that this activity is modulated by anorectic agents.

Conclusions
The PN is a key component of the appetite suppression center and its activation leads to a decrease in food intake. This study provides evidence for the role of the PN in the regulation of appetite and energy balance.

Abstract

The abstract section of the poster on the right, which is partially visible, contains a summary of the research findings.

Introduction

The introduction section of the poster on the right provides background information on the research topic.

Methods

The methods section of the poster on the right describes the experimental procedures used in the study.

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Matt Carter
Department of Biology
Williams College

August 8, 2022

