



**THE SAHLGRENKA ACADEMY
INSTITUTE OF NEUROSCIENCE AND PHYSIOLOGY**

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Announcement - scholarship at undergraduate/advanced level

The Department of Pharmacology, Institute of Neuroscience and Physiology, hereby announces a vacant scholarship at undergraduate/advanced level in psychopharmacology.

Training plan

Subject

Electroconvulsive therapy and rapid-acting antidepressants – shared mechanisms of action

Background

Depression is a debilitating mental disorder that affects more than 300 million people worldwide and causes significant suffering. Symptom management with traditional pharmacotherapies such as SSRI/SNRIs can take weeks to reach full effect and are ineffective in approximately a third of the patient population, highlighting a need for faster and more efficient treatment strategies.

Electroconvulsive therapy (ECT) has been used to combat treatment-resistant depression and related affective disorders since the 1930s, but its underlying antidepressant mechanisms remain elusive. Recent advances within the field of psychopharmacology have shown rapid-acting antidepressants such as ketamine and tentatively the serotonergic 5-HT_{2A} agonist psilocybin to represent treatment alternatives with mechanisms of action that differ from the traditional monoaminergic therapies. In rodents, depressive-like behavior has been associated with reduced synaptic plasticity in brain regions such as the prefrontal cortex (PFC) and hippocampus, and rapid-acting antidepressants have been shown to promote restoration of dendritic spine density in these regions. In the case of ketamine, this seems to occur downstream of its primary pharmacological target. Ketamine-induced blockade of NMDA receptors located on GABAergic interneurons seems to cause a compensatory glutamatergic signaling surge that activates AMPA receptors and thus increases signaling of intracellular messengers important for synaptic plasticity such as mTOR.

As our preclinical findings have indicated convergent antidepressant mechanisms of action between ketamine, psilocybin and ECT, this project will investigate how these mechanisms impact the dendritic spine density in the PFC and hippocampus of mice.

Purpose

The project aims to identify shared mechanisms of action between rapid-acting antidepressants and ECT by investigating the impact of mTOR and 5-HT_{2A} receptor signaling on the antidepressant-like response to ECT in mice.

Method

Mice are subjected to several weeks of chronic mild stress to induce depressive-like behavior and are then treated with electroconvulsive therapy (ECT) or sham-ECT (placebo) for a period of one week. Prior to each ECT/sham treatment, the mice are injected with either a 5-HT_{2A} receptor antagonist, an mTOR blocker or a vehicle solution. Following treatment, antidepressant-like response is measured by observing how long the mice spend swimming in the forced swim test – a behavioral measure indicative of clinical antidepressant effect. Mice are euthanized shortly following the behavioral test. Brains are extracted and processed for evaluation of dendritic spine density using Golgi-Cox histological staining, and for protein detection using the Jess automated Western blot system.

Time plan

Month 1-2: In vivo preparations, behavioral experiment, tissue collection and processing

Month 3: Data analysis, group presentation, manuscript preparation

Learning outcomes

The student is expected to develop an understanding for the preclinical research process as well as to gain practical experience in experimental animal research, tissue staining and data analysis. The student will partake in the research group's meetings and seminars of preclinical and clinical psychopharmacology.

The scholarship covers living expenses and is not covered for work conducted at the University of Gothenburg.

Period

2026-11-09 to 2027-01-31

Financing

1 payment of 60.000 SEK for the whole period.

If you require any further information, please contact Elias Eriksson, elias.eriksson@neuro.gu.se, supervisor.

Application

To apply please fill out the form "Scholarship application" and send it to Elias Eriksson, elias.eriksson@neuro.gu.se, supervisor.

Please attach a copy of:

- CV
- Letter of motivation

Closing date is 2026-11-06.