## Submit your application to Dr. Alex Galazyuk

Project Title: Neural mechanisms underlying age-related hearing loss

## PI Name: Alex Galazyuk

## Location: NEOMED

**Abstract:** Age-related hearing loss remains one of the most common chronic conditions of aging. It begins from the gradual loss or impairment of the inner and outer hair cells in the cochlea. This loss leads to the development of deficits in the central auditory system which eventually cause difficulties in processing temporally complex sounds such as speech, especially in noisy environments. Typically, individuals experience a notable decline in their hearing abilities after the age of 65, whereas cochlea degradation begins much earlier in life. Within the field, there exists a consensus that central plasticity, often referred to as central gain enhancement, serves as a compensatory mechanism to counterbalance the loss of input from the cochlea to the central auditory system due to aging. The postsynaptic mechanism underlying this compensation is largely unknown. It has been hypothesized that alterations in the balance between excitation and inhibition may play the key role. The goal of this project is to elucidate the postsynaptic mechanisms that contribute to the central gain and to identify pharmacological therapy to improve hearing performance in aged individuals.

**Significance:** Age-related hearing loss remains one of the most common chronic conditions of aging. Older listeners experience difficulties understanding speech, particularly in noisy environments. While audibility partially accounts for these functional deficits, elderly listeners with normal hearing and intact cognitive function still have poorer speech recognition ability in noise compared to young listeners. Central auditory processing has been shown to play a key role in compensation for the loss of cochlea function with age. This compensation has been hypothesized to rely on altered balance between excitation and inhibition and often referred as central gain enhancement. At present, little is known about postsynaptic mechanism(s) underlying central gain alteration. Deep knowledge about changes in both the excitatory and inhibitory components of this mechanism can help us to better understand the cellular basis for aging. The proposed study will improve our knowledge of the central mechanisms responsible for auditory aging and provide a foundation for the development of treatment strategies.

**Goals and Objectives:** The student will identify changes in sound-evoked responses of inferior colliculus neurons in different age groups of unanesthetized mice.

**Methods:** The student will learn how to record single neuron electrical activity of neurons in the inferior colliculus in response to different sound frequency and intensity. The student will learn to use electrophysiological setup to stimulate mice with sounds and record action potentials of auditory neurons in response to these stimuli. The student will also have an opportunity to observe other techniques in the lab, including

fabrication of glass recording microelectrodes and generation of automated sound stimulation protocol.

**Data Analysis:** Data collected by the student will be analyzed using custom-made software. For each recorded neuron its so-called frequency response area based on more than 3,000 sounds with different combinations of sound frequency and intensity will be constructed and analyzed.

**Anticipated Findings:** The anticipated findings from this project will identify the differences in sound processing with age.

## Student Fellow Training/Mentoring Plan

The student will have the opportunity to meet individually with the PI regularly. In addition to working directly with the PI, the student's training will be continuously monitored by a postdoctoral fellow. Research will be conducted at NEOMED. All resources necessary for the described experiments are available. We work as part of the NEOMED Hearing Research Group, and students in the lab will have the opportunity to interact with the other group members.