Block 1 :

Project 1 – « Neuronal excitability changes in early stages of Alzheimer`s disease »
Instructor : Senka Hadzibegovic (Neurocentre Magendie, France)

Alzheimer's disease (AD) is the most common progressive and fatal neurodegenerative disorder. Achieving early diagnosis is thus a crucial objective in both preclinical and clinical AD research, since it represents a vital therapeutic window during which interventions can be initiated before the onset of severe cognitive decline. The accumulation of amyloid-beta (Aß) peptide in the prefrontal cortex (PFC) is linked to rapid forgetting – an early symptom of this prodromal stage of AD. Our data suggest that Aß accumulation is linked to alterations in neuronal excitability in ACC neurons during the early stages of the disease. This suggests a potential mechanism through which Aß disrupts memory storage, contributing to accelerated forgetting in AD.
Based on these findings, we hypothesize that abnormal neuronal excitability in the ACC leads to dysfunction in network activity and crucial cellular memory mechanisms, ultimately disrupting long-term memory storage and accelerating forgetting. Our ongoing research aims to study ACC engram neurons excitability during different memory stages in early-phase AD and explore mechanism-based interventions to correct memory deficits, offering promising therapeutic avenues during this sensitive time window.
***Methodology**:*
We utilize two mouse models of AD: 3Tg mice-a genetic model associated with familial AD, and inducible model-microinfusion of soluble oAβ1-42 peptide. We perform the patch-clamp recordings in acute anterior cingulate cortex (ACC) slices to probe changes in the intrinsic excitability. To asses memory impairment we use contextual fear conditioning.

Project 2 – *« Activities of Basket and Axoaxonic cells of the BLA during anxiety assays and fear conditioning. »*
Instructor : Chloe Lhereaux (Neurocentre Magendie, France)

The survival of animals is determined by their ability to cope with danger. As such, mechanisms regulating emotions such as fear and anxiety are crucial. The basolateral amygdala (BLA) is a key structure involved in the regulation of those emotional processes. Within the BLA, excitatory principal neurons are responsible for the encoding and relay of emotionally relevant information to other brain structures, while GABAergic interneurons tightly regulate their activities. Among key interneuron classes, parvalbumin-expressing (PV) interneurons have been shown to be involved in the control of anxiety and fear-related behaviors. PV interneurons are comprised of two main cell types: basket cells (BCs) and axo-axonic cells (AACs). Their distinct post-synaptic targets and previous work in anesthetized rodents suggest that these cell types may play specialized roles. Here, we leverage the development of a novel transgenic mouse line that allows the selective genetic targeting of BLA AACs and BCs, to record these interneurons during fear and anxiety behaviors. We will perform stereotaxic surgery with optic fiber implantations, followed by calcium imaging fiber photometry to record the interneurons in a specific manner, combined with fear and anxiety behavioral assays. After mouse perfusion, we will perform brain slicing and immunohistochemistry and we will verify the virus expression and fiber placement under an epifluorescence microscope. Eventually, we will write custom scripts to analyse the activity of our cell types during behavior.
***Methodology**:*
During this week, we will first perform a surgery (viral injection and optic fiber implantation). Then, we will record mice during an elevated-plus maze, and a fear conditioning. Mice will either express the calcium indicator (GCAMP) in either BC or AAC. One day will be dedicated to histology (perfusion, brain slicing and histology control with an epifluorescence microscope). The last day will

consist in the analysis of the results using matlab (The students will design themselves the analyses, and I will write and explain the code needed to get them).

Project 3 - « *Examining Connectome of Major Depressive Disorder and Post-Stroke Depression.”*
Instructors: Suhrit Duttagupta (INCIA, France)

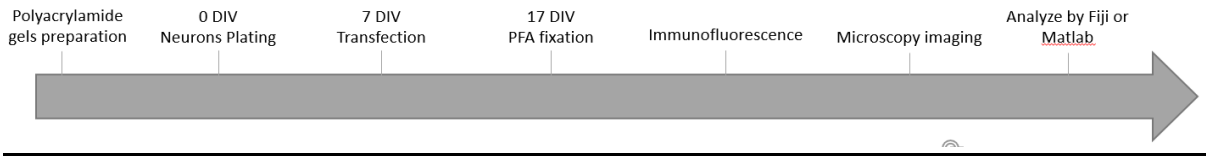
The ability to simultaneously monitor (calcium imaging) and control (optogenetic) neuronal activity opened the door to a new era of neuroscience. While such experiments are principally performed on restrained animals, studies have shown that whole body movement drastically affects neuronal processes. Developments in optical engineering are therefore needed to perform high resolution imaging and refined optogenetic control on freely moving animals. Our lab has developed a flexible two-photon microendoscope (2P-FENDO) capable of all-optical brain investigation at near cellular resolution in freely moving mice. The system performs fast two-photon (2P) functional imaging and 2P holographic photostimulation of single and/or multiple cells. This workshop will provide a complete overview and direct experience building and using this system.
Methodology :
It is planned to use resting-state functional MRI data from the Human Connectome Project and OpenNeuro and process them using the programming language Python.

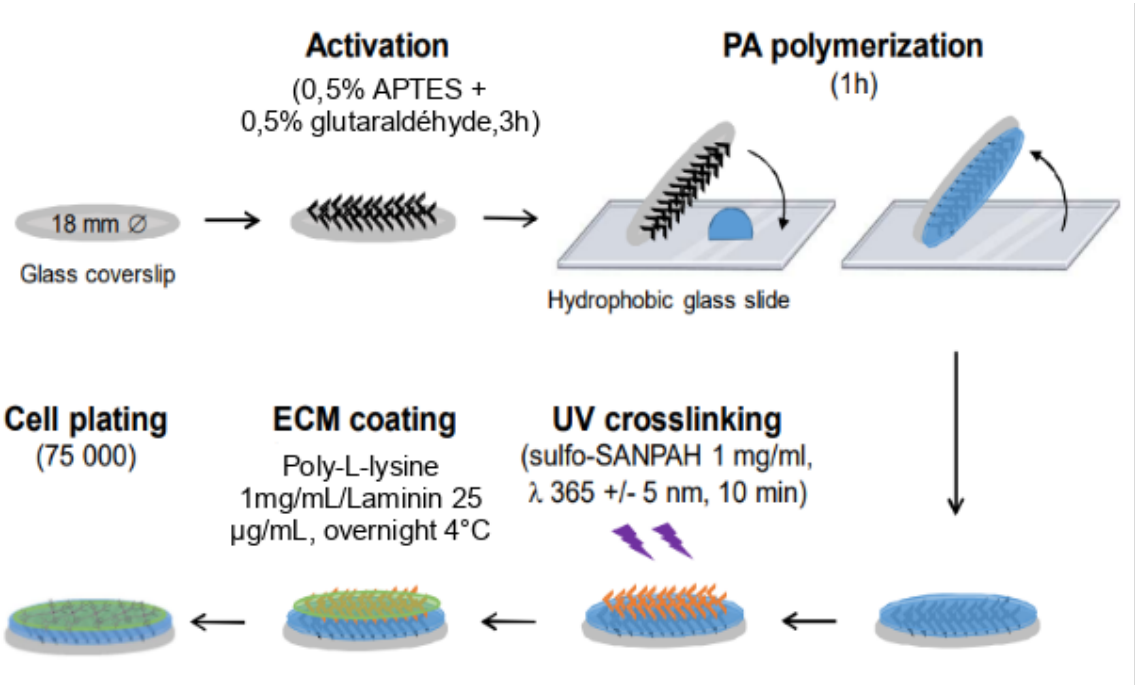
Project 4 - « *Functional connectome of a post-traumatic stress disorder-like memory.* »
Instructor : Flavia Simoes (IINS, France)

Post-traumatic stress disorder (PTSD) is a mental health condition that is triggered by a traumatic event. A cardinal symptom of PTSD is a paradoxical and long-lasting memory alteration including both an hypermnesia for some salient, but irrelevant, trauma-related cue and a contextual amnesia, that decreases the ability to restrict fear to the appropriate context. Previous studies from the lab found that glucocorticoids administration after fear conditioning induces PTSD-like memory impairments as mice become unable to identify the context as the right predictor of the threat and show fear responses for a discrete cue non predicting the threat in normal conditions.
With this project we aim to unravel the whole brain activation behind this maladaptive behaviour, hoping to shine light on the pathology of this disease. We will do this by generating a PTSD-like memory in mice, using a fear-conditioning task, followed by a brain clearing approach and whole brain imaging and quantification of an immediate early gene related to the behaviour.
Methodology:
Fear conditioning -Whole-brain clearing -Lightsheet microscopy

Project 5 – « *Impact of substrate rigidity on synapse and endocytosis dynamics.* »
Instructors: Domitille Des Robert (IINS, Bordeaux)

The main goal of this project is to characterize the environment of hippocampal neuronal culture to see the impact of the substrate rigidity in dynamics of endocytic zone of the spines and shaft dendrites. To do this, several steps are done as shown in the next graph.





We first prepare polyacrylamide gels of controlled stiffness. The protocol is composed of 4 steps depicted in Figure 1: (i) Glass activation for adhesion of the gel, (ii) Polyacrylamide gel preparation, (iii) Gel activation with sulfo-SANPAH and UV illumination, (iv) Coating with PLL and laminin. Gel stiffness is determined by the proportion of acrylamide and bis-acrylamide used to prepare it. We use 3 types of stiffness: 0.17 kPa (close to brain stiffness), 1 kPa (close to endothelial cells stiffness) and 32 kPa (close to muscle tissue stiffness). We compare the results with the ones obtained in classical cultures grown on glass coverslips (stiffness > 1 GPa).
Neurons are then plated in the coverslips. Two types of cultures are used: a mixed culture with the same number of neurons of glial cells and a “Banker” type culture to have 90% of neurons. These two types of culture are done in parallel.
To study neurons and dendrites morphology as well as spines density, a transfection is done at 7 Days In Vitro (DIV) with GFP.
At 17 DIV, neurons are fixed with a paraformaldehyde solution. To characterize cultures growing on different substrates, immunofluorescence experiments are performed:

- Quantification of astrocytes and neurons number with GFAP, an astrocyte marker and MAP2, a dendritic neuronal marker and their ratio.
- Quantification of synapse density with Shank2, a post-synaptic marker and Bassoon, a pre-synaptic marker as well the axon initial segment (AIS) with AnkyrinG.

Methodology:
Confocal and widefield microscopes are used to take pictures at different magnification to see the whole neuron or just synapses.
Macros and plugins in both Matlab and Fiji allow to quantify elements of neurons environment.

Project 6 - *« In vivo recording of glutamatergic neurons in the Fastigial nucleus during locomotion. »*
Instructor: Abderrahman Fettah (INCIA, France)

Data from the literature indicate that the fastigial nucleus (FN) in the cerebellum is likely to contribute to the expression of locomotor activities in rodents. Recent data showed that glutamatergic neurons

in the FN express the vesicular transporter VGluT2. In order to elucidate the contribution of this neuronal population in the context of locomotion, the mini-project will consist in recording this neuronal population during locomotor activities expressed spontaneously in an open field or in a moving treadmill.
We will perform fiber photometry recordings to reveal the activity of the FN glutamatergic neurons during locomotor behaviors. Using intra-cellular calcium fluctuation as the proxy of neuronal activity we will define whether this population increased tonically or phasically its activity during locomotor behavior.
Methodology:
Fiber photometry, movements tracking (DLC)

Project 7 – « *Deep brain structures, Neuroimaging Analysis, Neuromodulation Fundamentals* ».
Instructor : Nana Tchantchaleishvili (RebrAIn, UNIK, Univ Bordeaux)

My MSc project was a part of clinical trial focused on essential tremor targeting with a new software created in Bordeaux (OptimDBS, RebrAIn). But I'm planning to include anatomical background and clinical correlation, as different structures can be targeted for different diseases. I have been on this summer school as a student, and 1st week of my training (group fMRI analysis, GIFT, resting state) helped me a lot in choosing a correct internship project for myself and being more prepared and confident, as up to 30 % of software used are the same for fMRIs as well as lead DBS analysis. I hope my experience will also be helpful for the students interested in neuroradiological research. Reconstructed 3D structures will also help to better memorize the structures. I will use demo mode, MNI space, as well as patient data (with permit), there are multiple highest quality 3D atlases of incorporated in this softwares and it's a perfect learning tool for anatomy.

Obj1: Anatomy of deep brain structures
Obj2: Clinical Correlations and Future Research Perspectives
Obj3: Using Neuroradiological software for data analysis
Methodology:
lead DBS software (fmatlab version), MedInria for the final simplified 2D analysis

Project 8 – « *Introduction to the EEG methods in neurolinguistic research* ».
Instructor: Jane Aristia (Universidade de Lisboa, Portugal)

Language is our mean of communication; it helps us to deliver what is in our mind to the external word. On the surface, it seems we can process language effortlessly. However, behind this effortless process, our brain processes quite a lot of information, such as phonology, syntactic, and semantic information. Efficiently, our brain processes this information rapidly in milliseconds. To better understand what are the information that is used to process the language input such a word in a sentence, researchers employed Electroencephalography (EEG) techniques. It is one of the common methods in investigating language processing in the brain as it provides information about brain activities in milliseconds.
In this project, EEG method will be introduced particularly concerning the use of it in language processing research. For this reason, this proposed project will focus on event related potential (ERP), which is brain responses towards the targeted stimulus. This is a common approach to examine EEG data in language processing study. Hence, in this project participants will have the introduction of EEG data pre-processing and analysis. In this project, ERP components related to language processing, such as, LAN, N400, and P600 will also be discussed.
Methodology*:*

We will use open-source EEG data. For pre-processing purposes we will use MNE python, and for the analysis, a statistical software like R.

Block 2 :

Project 9 – « Decoding the perception of faces from patterns of brain activity. »
Instructor : Achille Gillig (IMN, France)

Among the different approaches to tackle the question of how objects are represented in the brain, multivariate pattern analysis (MVPA) applied to functional magnetic resonance imaging (fMRI) data has proven to be of great value. Instead of analyzing brain activations in response to task manipulations, MVPA inverses the problem and, instead, aims at decoding the stimulus or task condition from brain activity. This approach provides multiple advantages, including the ability to probe the way conditions are differentially represented within brain regions in the absence of differences in average activity. In the seminal MVPA experiment, this approach has been used to reveal that the perception of faces can be discriminated from the perception of objects on the basis of differentiated underlying representations in the fusiform face area (FFA).
The objective of this project is to perform multivariate pattern analysis to decode from brain activity the category of objects that were viewed by participants.
***Methodology**:*
All data analyses will be performed in python. We will use the openly available Midnight Scanner Club (MSC) dataset (https://www.openfmri.org/dataset/ds000224/) that contains extensive fMRI data from 10 subjects. The project will mostly rely on the nilearn package for handling and manipulation of brain volume files, and on the scikit-learn package for machine learning implementations. The data analysis pipeline will consist of 1) data importation and preprocessing, 2) first-level single-trial general linear model to extract parameters reflecting voxels' responses to each stimuli, 3) classification of faces vs scenes on the extracted statistics using nested k-fold cross-validation together with an appropriate classifier, 4) statistical inference using permutation testing (generative null hypothesis modeling).

Project 10 – *« Exploring the neural mechanisms of multisensory temporal integration in autism. »*
Instructors : Nikolett Zsigri and Ourania Semelidou (Neurocentre Magendie, France)

Altered sensory experience is one of the core features of autism spectrum condition (ASC), a neurodevelopmental condition also characterized by alterations in social communication and interaction and repetitive behaviors. These sensory alterations are observed across modalities and affect the majority of autistic individuals, exerting a strong negative influence on day-to-day life. Importantly, sensory perception alterations and contribute to the development of other core symptoms and medical conditions that co-occur with autism (eg anxiety). Sensory alterations can be detected already in the sixth month of life in infants with ASD, thus preceding other core symptoms, and they correlate with clinical diagnosis, an observation that suggests their predictive power. Importantly, alterations in the sensory domain have a strong neurophysiological basis, which shows great promise in our endeavor to identify the neurobiological mechanisms of autism. Amongst these alterations, multisensory integration is reported altered in autistic individuals, with changes in temporal processing leading to wider multisensory binding windows and altered perception.
In this project, we aim to explore the neurobiological mechanisms that underlie altered multisensory integration in autism. To this end, we will use a sophisticated toolset, combining a novel behavioral task with in vivo two photon microscopy in a genetic mouse model of autism. This genetic mouse model exhibits sensory alterations and changes in a wide range of behavioral phenotypes related to other ASD core symptoms. To study multisensory perception we will use a perceptual decision-making task. In this task, to mimic a multisensory environment the animal will report whether it perceived a

tactile and auditory stimulus synchronously (totally overlapping) or asynchronously occurring (with an onset delay). This task will allow us to study the temporal properties of stimulus integration and to define differences in the temporal binding window in the mouse model of autism. In parallel, we will study neuronal activity in the prefrontal cortex, to probe alterations in the activity of excitatory and inhibitory neurons during the detection of the audiotactile stimuli. Overall, in this project we will determine how multisensory integration is altered in autism and give us mechanistic insight into the neural correlates of these alterations.

***Methodology**:*

behavioral experiments (reward-based decision-making task), in vivo calcium imaging, stereotactic surgeries

Project 11 - *« Study microglia phagocytosis during AD pathogenesis. »*
Instructor: Wenqiang Chen (Harvard Medical School; Steno Diabetes Center Copenhagen)

Alzheimer's disease (AD), characterized by accumulation of abnormal plaques and neurofibrillary tangles, is a devastating disease that is still unsolved, despite many progresses have been made in the past years. Although exact pathogeneses of this disease are still not fully understood, neuroinflammation is a well-characterized hallmark of AD pathogenesis and is believed to be mainly triggered by uncontrolled activation of microglia and impairment of phagocytosis at advanced disease stages. Thus, studying microglia phagocytosis is a key step to address the roles of this critical cell type in disease progression.

In this project, I will teach how to explore the basic concepts of neuroinflammation and to apply this knowledge to address potential scientific questions using rodent models. I will bring what I establish during my second postdoc training to teach the students how to work as a team to study phagocytosis at different levels, including immunohistochemistry of brain sections, FACS sorting of isolated primary microglia, and immortalized microglia cell line (BV2 or SIM-A9).

Methodology :

I will teach the students the following cell biology experiments: Cell culture of microglia cells (primary or immortalized cells) Cell sorting and FACS (cell isolation, in vitro phagocytosis of fluorescently labeled Abeta) Immunohistochemistry staining of microglia and beta-amyloid in brain sections of 5xFAD mice High-content live cell imaging (IncuCyte S3).

Project 12 - « *Study of neuropathological lesions of Alzheimer's disease in a non-human primate model: focus on the brainstem.* »
Instructor : Morgane Darricau (IMN, France)

Tauopathies are a group of neurodegenerative diseases characterized by the aggregation and accumulation of misfolded tau proteins in the brain. Alzheimer's disease (AD) is the most prevalent tauopathy in the population; 1/10 people aged 65 and older are affected. AD is characterized by the presence of β-amyloid plaques and neurofibrillary tangles (NFTs), which result from the aggregation of abnormally hyperphosphorylated tau protein. Several studies demonstrated a causal link between β-amyloid, tau, neurodegeneration, and cognitive decline. However, the mechanisms of initiation and progression of tauopathies remain poorly understood for these pathologies. Recent studies have suggested that the development of tauopathies is based on "prion-like" features, where a single tau proteopathic seed would have the ability to transmit pathogenic information to "contaminate" non-pathological soluble tau proteins, leading to neurotoxicity. Tau aggregates would be transmitted from cell to cell, thus explaining the spreading of tauopathy in different connected brain regions. However, this hypothesis is based only on in vitro and in vivo rodent experiments. These experimental models offer many advantages for the study of tauopathies, but they do not fully reproduce human pathologies. To develop relevant pre-clinical studies of these pathologies

including AD, it seems necessary today to work on experimental models as close as possible to human models. Thus, in my PhD project, we modeled Alzheimer's disease in rhesus macaques (Macaca mulatta), an animal phylogenetically closest to humans. The main aim was to demonstrate the "prion-like" propagation of AD tauopathy in non-human primates. We induced the pathology using intracerebral injections of tau aggregates extracted and purified from AD patients. Indeed, we could observe in these primates the presence of typical lesions of AD: lesions of NFTs and neuropils threads. The observation of these lesions around injection sites (entorhinal cortex) and within connected structures (hippocampal trisynaptic loop and cingulate cortex) suggests the "prion-like" feature of tau protein, where the simple injection of pathogenic material allows the formation of new aggregates progressing from cell to cell. These works have enabled us to understand better the pathophysiology and mechanisms involved in the cognitive disorders of AD.
Thanks to my PhD projects, we have in the lab brain tissue of AD non-human primate models that we developed by bilateral injections of tau aggregates extracted from AD patients, and one group of macaques also received injections of recombinant oligomeric Aß peptides into lateral ventricles. In these previous works, we assessed the neuropathology of AD around the injection sites (entorhinal cortex) and closed connected areas. For this project, I would like to investigate regions more distant from the injection sites: the brainstem, more specifically the pons in which the locus coeruleus is located Indeed, AD human post-mortem studies demonstrated that the locus coeruleus is a more vulnerable nuclei to degenerate and accumulate intraneuronal phosphorylated tau before the entorhinal cortex. It has also been shown that 80% of locus coeruleus neurons are lost in AD. But, here again, we didn't know the mechanisms of initiation and spreading of pathological tau proteins: from the entorhinal cortex to the brainstem? Or with a bidirectional spreading? In that way, investigating this area could be interesting for a better understanding of the pathophysiology and mechanisms of AD. Thus, in this project, I propose to study neuropathological lesions of AD in the brainstem, mainly focusing on the locus coeruleus.

Methodology :

We will practice immunohistochemistry techniques on AD non-human primate tissue for this project. After selection slices, we will do AT8 staining (specific staining of the tau protein phosphorylated at specific sites), β-amyloid staining, and Thioflavin S staining to visualize β-amyloid plaques and mature lesions of AD tauopathy. These immunohistochemistries will be performed on some slices of primate tissue at different levels of the brainstem containing locus coeruleus. At the end, we will observe the different staining with an optic microscope and discuss the results analysis and possible statistical tests.

Project 13 – « *Effects of a food odour on hypothalamic food intake regulating neurons.* »
Instructor: Louise Eygret (NutriNeuro, France)

The olfactory system plays an important role in food intake, thus it could offer safer alternative strategies to regulate eating behaviour. Food intake is essentially regulated by the hypothalamic neurons of the melanocortin network: AgRP (Agouti Related Peptide) and POMC (Pro-opiomelanocortin) neurons, respectively orexigenic and anorexigenic. Previous studies showed that the presentation of food rapidly modulates the activity of these neurons in mice even before eating suggesting the involvement of sensory cues in this modulation. However, the determinants of this modulation have not been identified.
This project studies the modulatory effects of a vanilla odour molecule on hypothalamic POMC neurons involved in the control of food intake. Students will discover how to study this question from the behavioural experiments (observing food intake behaviour) to the cellular experiments (electrophysiological recordings). Food intake will be assessed using automated food intake monitoring cages in two groups of C57Bl6 mice exposed to either vanilla-smelling chow or standard chow. In parallel, patch-clamp recordings will be performed on POMC-GFP mice previously exposed

to vanilla odour (compared to solvent-exposed mice). Synaptic inputs will be monitored in voltage clamp mode and spontaneous activity and IV curves will be assessed in current clamp mode.

Methodology:

Animal handling Behavioural experiments with automated food-intake measurement cages Brain slices preparation (dissection + vibratome) Whole-cell patch clamp on POMC neurons (thanks to POMC-GFP mice) Analysis (Easy Electrophysiology software + Clampfit software)

Project 14 - ***« Prdm2 role in cocaine self-administratio. »***
Instructor : Giulia De Maio (Neurocentre Magendie, France)

To better understand the neurobiological mechanisms that cause some individuals to lose control of their cocaine use, we are using a rat model that allows longitudinal study of pathology. This model reproduces the diagnostic criteria of human pathology as defined in the DSMV(1). After prolonged cocaine self-administration, approximately 15% of the rats exhibit the three main criteria of human addiction: i. Strong motivation to take the drug, ii. Use despite harmful consequences, iii. Loss of control over drug-seeking behavior. Regarding this last criterion, drug-seeking rats are unable to inhibit their search when the drug is signaled as unavailable (drug-free periods). In a pre-addiction state, rats vulnerable to addiction already show a reduced ability to inhibit seeking until the end of drug-free periods (2), a behavior considered an early marker of vulnerability to addiction (3). My PhD work focuses on Prdm2, a repressive histone methyltransferase that uniquely and specifically methylates histone H3 on lysine 9 (H3K9me1) and is particularly expressed in the medial prefrontal cortex. More importantly, in rats, decreased Prdm2 expression in the dmPFC is a marker of vulnerability to alcohol dependence (4). 1. Deroche-Gamonet V. et al. Evidence for addiction-like behavior in the rat. Science 305, 1014–1017 (2004) 2. Belin D. et al. Pattern of intake and drug craving predict the development of cocaine addiction-like behavior in rats. Biol. Psychiatry 65, 863–868 (2009). 3. Kasanetz F. et al. Transition to addiction is associated with a persistent impairment in synaptic plasticity. Science 328, 1709–1712 (2010). 4. Barbier E, et al. Dependence-induced increase of alcohol self-administration and compulsive drinking mediated by the histone methyltransferase PRDM2. Mol. Psychiatry 22, 1746–1758 (2017).
The project involves rats injected with a shPrdm2 virus and others injected with a control virus. After viral expression, cocaine self-administration is started and the animals' behavior is studied in a cocaine pre-addiction state. The prelimbic (PL) is then collected and RNA is extracted. RNA sequencing will be performed and bioinformatic analysis will reveal genes expression differences between the two groups.

Methodology:

Behaviour (cocaine self-administration and behavioral tests) RNA extraction and sequencing Analysis of data

Project 15 – ***"Establishment of two-dimensional static contact based human blood brain barrier using Transwell apparatus"***
Instructor: Guneet Kaur (National Brain Research Centre, India)

The mini project will focus on assembly and co-culture of two BBB cells: human brain microvascular endothelial cells (BMECs) and astrocytes. Transwell inserts with semipermeable polyester (PET) membrane will be used to culture the cells on its either side: luminal (towards blood) and abluminal (towards brain), as described in Kaur et.al 2023. To closely mimic the in vivo human physiology, the cells will be cultured in contact with each other. Firstly, astrocytes will be seeded onto the abluminal side by creating an external well using a sterile silicon tubing by inverting the transwell and will be allowed to adhere for 5-6 hours. After that the inserts will be inverted back to its upright position and cells will be allowed to grow for 24 hours. Then, BMECs will be seeded on to the luminal side of the membrane and cells will be allowed to grown in contact with each other for another 24 hours.

To assess the BBB integrity, endothelial resistance will be measured using a TEER meter and a high molecular weight dye (dextran-FITC) will also be used to assess the endothelial permeability. To visualize the contact co-culture of BBB cells, immunocytochemistry will be performed on the PET membrane, cells will be immunostained with their respective characteristic functional markers- VE-Cadherin for BMECs and GFAP for astrocytes. The cells will then be imaged using a fluorescence microscope.

Project 16 – *"Exploring Neuronal Development: A Simulation Study on Neuron Cells using NEURON Simulator"*
Instructor: Eslin Ustun Karatop (University of Ottawa, Canada)

NEURON Simulator is a powerful tool that allows for the simulation of the electrical and chemical processes that occur within neurons. It enables the modeling of individual neurons as well as networks of neurons and provides a platform for testing various interventions that can modulate neuronal function. In this project, we will use NEURON Simulator to simulate the effect the electrical and chemical properties of neuron cells. Specifically, we will investigate how changes in modifications affect the ion channels and receptors that regulate neuronal activity. By simulating the neuron cells, this project aim to identify potential targets for modulating neuronal function and improving communication between neurons. This could have implications for the treatment of neurological disorders, such as Alzheimer's disease, Parkinson's disease, and epilepsy.
Optogenetic material refers to the genetic information that is involved in the development of an organism. It plays a crucial role in the formation and function of the nervous system, including the development and maintenance of neuron cells. By simulating the effects of optogenetic material on neuron cells, we can gain insight into the underlying mechanisms of neuronal development and function. NEURON Simulator is a powerful tool that allows for the simulation of the electrical and chemical processes that occur within neurons. It enables the modeling of individual neurons as well as networks of neurons and provides a platform for testing various interventions that can modulate neuronal function. In this project, we will use NEURON Simulator to simulate the effects of optogenetic material on the electrical and chemical properties of neuron cells. Specifically, we will investigate how changes in gene expression and epigenetic modifications affect the ion channels and receptors that regulate neuronal activity. By simulating the effects of optogenetic material on neuron cells, we aim to identify potential targets for modulating neuronal function and improving communication between neurons. This could have implications for the treatment of neurological disorders, such as Alzheimer's disease, Parkinson's disease, and epilepsy. Overall, this simulation study of optogenetic material on neuron cells using NEURON Simulator has the potential to deepen our understanding of the mechanisms of neuronal development and function and could lead to the development of new treatments for neurological disorders.

Methodology:

PC is needed. NEURON simulator will be download. Phyton coding environment is needed. First, we will learn to usage of NEURON simulator. Secondly, we will create our cell environment, integrated the ChR2. Thirdly, we will stimulate with light and will analyze the results. Finally, we will learn how to implement disease model and solve a problem for neuronal diseases.