Summer Research Fellowship (SRF) Sharing

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2020 SRF Participant, Major in Biochemistry

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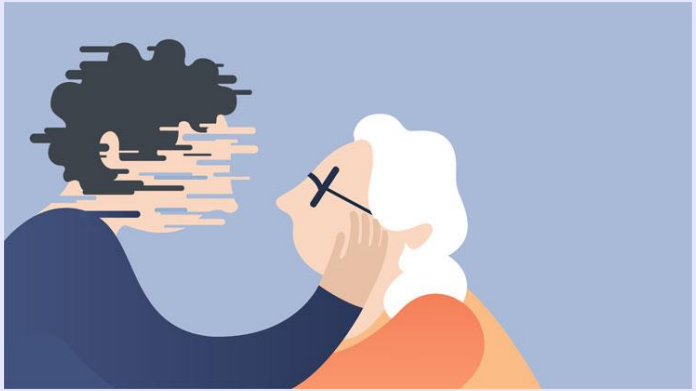

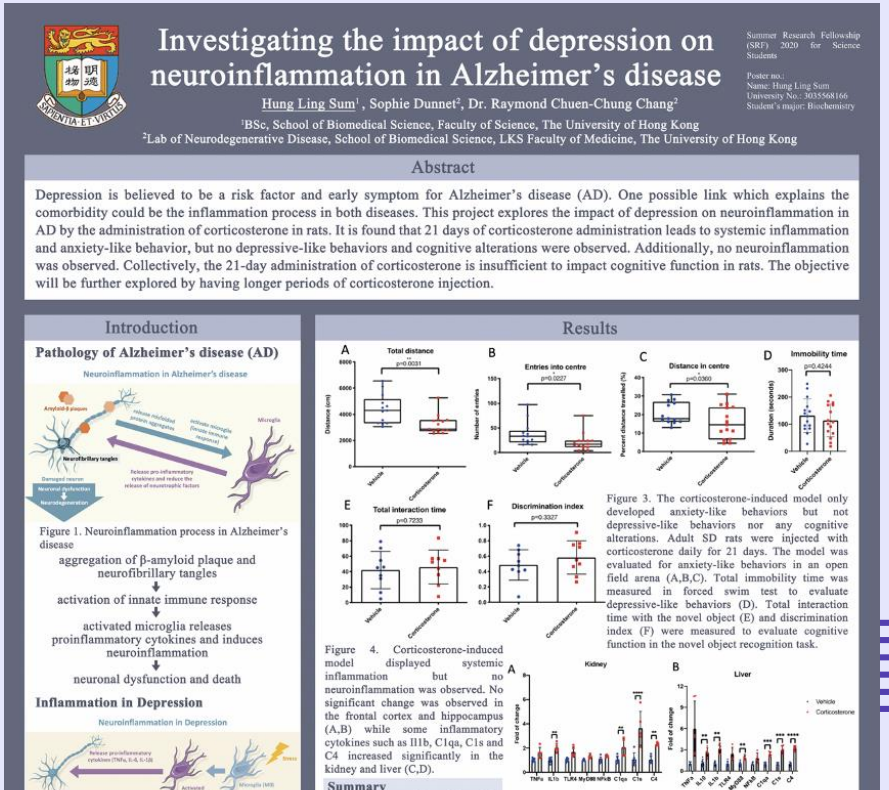

Investigating the impact of depression on neuroinflammation in Alzheimer's disease

Summer Research Fellowship (SRF) 2020 for Science Students

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Abstract

Depression is believed to be a risk factor and early symptom for Alzheimer's disease (AD). One possible link which explains the comorbidity could be the inflammation process in both diseases. This project explores the impact of depression on neuroinflammation in AD by the administration of corticosterone in rats. It is found that 21 days of corticosterone administration leads to systemic inflammation and anxiety-like behavior, but no depressive-like behaviors and cognitive alterations were observed. Additionally, no neuroinflammation was observed. Collectively, the 21-day administration of corticosterone is insufficient to impact cognitive function in rats. The objective will be further explored by having longer periods of corticosterone injection.

Introduction

Pathology of Alzheimer's disease (AD)

Figure 1. Neuroinflammation process in Alzheimer's disease

aggregation of β-amyloid plaque and neurofibrillary tangles
↓
activation of innate immune response
↓
activated microglia releases proinflammatory cytokines and induces neuroinflammation
↓
neuronal dysfunction and death

Inflammation in Depression

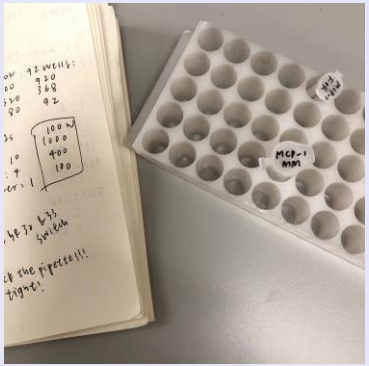
Results

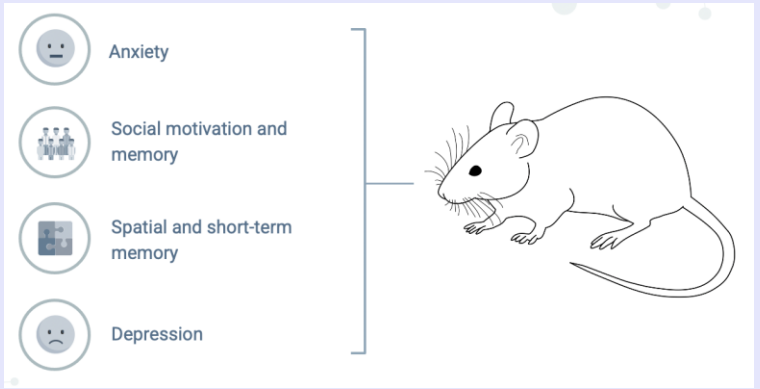
Figure 3. The corticosterone-induced model only developed anxiety-like behaviors but not depressive-like behaviors nor any cognitive alterations. Adult SD rats were injected with corticosterone daily for 21 days. The model was evaluated for anxiety-like behaviors in an open field arena (A,B,C). Total immobility time was measured in forced swim test to evaluate depressive-like behaviors (D). Total interaction time with the novel object (E) and discrimination index (F) were measured to evaluate cognitive function in the novel object recognition task.

Figure 4. Corticosterone-induced model displayed systemic inflammation but no neuroinflammation was observed. No significant change was observed in the frontal cortex and hippocampus (A,B) while some inflammatory cytokines such as Il1b, C1qa, C1s and C4 increased significantly in the kidney and liver (C,D).

Summary

My SRF Research Project



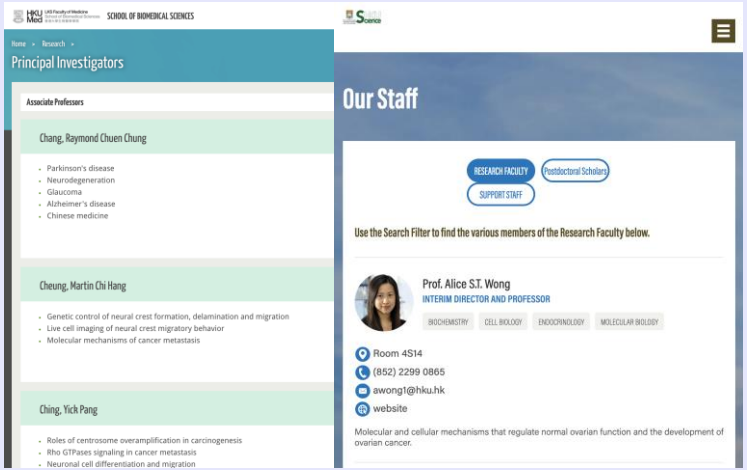


- RNA extraction from animal tissue
- Reverse transcription
- qPCR to examine expression of different inflammatory markers
- Analysis of behavioural tests to evaluate the behavioural changes of animal

Things to consider before application

Research Interest

Or just search through school websites and have a look on their research topics!



Expectation on SRF

Research experience?
Preparation for FYP?

Time commitment

Any other internship/ part-time jobs arranged in Summer?

Approaching Supervisor

1

Understand their research objectives

Read through their publications

2

Brainstorm any questions towards their research

Helpful for coming up with potential research topics

3

Email them and express your interest

May also include unofficial transcript for their reference

4

Meet them f2f to further discuss on potential research opportunities

May give you an idea of your research direction

Writing Research Proposal

- Read through **literature reviews** and give a brief background of your research topic
- Highlight the **significance** of your research topic
- Ask for your supervisor advice on the **project topic/ research direction**
- Request for **supervisor's advice** before submission

Investigating the impact of depression on tau pathology in Alzheimer's disease

Abstract

This research project aims to explore the mechanism on how depression could be the risk factor for Alzheimer's disease, by focusing on the impact of increased corticosterone on tau pathology in corticosterone-induced rats.

Introduction

Alzheimer's disease (AD) is a common devasting neurodegenerative disorders among elderly. It affects 53 million people around the globe in 2018[1]. The pathological hallmarks for AD are β-amyloid plaque deposition and neurofibrillary tangles of hyperphosphorylated tau[2]. However, the understanding on the mechanisms of initiation of AD is still limited.

Depression is believed to be a risk factor for AD and depression may be an early symptom of AD as it is highly co-morbid with AD[3]. The research conducted by Dr. Raymond Chang focused on the impact of depression on tau pathology.

Tau protein exerts pathological effects on cognitive functions by reducing tau affinity

Concerns on application requirements

Research topics

Just choose what you are **interested in**!

For most of the time, **no prior knowledge or lab skills** are required!

You learn them during SRF!

Academic merits

To be honest, my cGPA is below 3.0 when I applied for SRF so... just try it first! XD

Having a low GPA does not mean that you are not suitable to be a researcher!

SRF: A valuable research experience

- Weekly **lab meetings and journal clubs**
  - learn more about other research topics in the lab and in this field of research (neuroscience)
- Get to know **friendly lab members** all over the world
  - provide detailed explanation on different experiments and laboratory skills
  - Learn about their life as researchers
- **Self-improvement**
  - Conduct experiments **independently**
  - Enhanced **organizational** skills
  - Gain courage to **reach out and ask for help**

Thank you & Good luck!