EDITAL INTERNO 05/2018
PARCERIA FMRP
E
UNIVERSITY OF BRADFORD (REINO UNIDO)

Público Alvo: Alunos de Mestrado

ATENÇÃO ÀS DATAS

Inscrições:
26/11 /2018 a 4/2/2019

Banca entrevistadora:
25 a 28/2/2019

Resultado:
até 1/3/2019

Período do intercâmbio:
1/4/2019 a 31/7/2019

SERÃO OFERECIDAS 4 BOLSAS PARA ALUNOS REGULARMENTE MATRICULADOS EM CURSOS DE PÓS-GRADUAÇÃO DA FMRP. AS QUATRO BOLSAS TERÃO O VALOR GLOBAL DE DE €4,020 (3,200 EUROS MAIS 820 EUROS PARA CUSTOS RELATIVOS À VIAGEM).

Documentação necesssária para inscrição:

- Histórico escolar da graduação e pós-graduação em inglês
- Carta em inglês do orientador atual aceitando o intercâmbio
- Cartas de referência do atual orientador e do orientador da iniciação científica (se aplicável) também em inglês
- Comprovante de proficiência em inglês (nível AVANÇADO): Certificados aceitos pela University of Bradford ou declaração emitida pela Comissão de Relações Internacionais da FMRP (agende pelo e-mail crint@fmrp.usp.br)
-Desejavel: curso de neurobiologia.

Composição da banca entrevistadora

Prof. Drª Sonia AL Correa (University of Bradford)
Prof. Dr. Luis L. P. Silva (Departamento Biocel)
Prof. Dr. Rodrigo T. Calado (Presidente CRint)
A entrevista será realizada em língua inglesa

INSCRIÇÃO:
PREENCHA O FORMULÁRIO CLICANDO AQUI

ABAIXO ESTÃO OS PROJETOS DE PESQUISA E NÚMERO DE VAGAS DISPONÍVEIS:

Title: Development of novel blood brain barrier permeable peptides to reduce neuro-inflammation in Alzheimer disease

Supervision Drs Sonia AL CORRÊA and Jürgen MÜLLER
School of Pharmacy and Medical Sciences
University of Bradford
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One place for this project

The great challenge in Alzheimer's disease (AD) research is to establish the underlying process(es) and identify the key molecules that initiate pathogenesis. Recently, compelling evidence has linked the development of AD with activation of the immune cell microglia, which then overproduces toxic pro-inflammatory cytokines. Supporting this hypothesis is the observation that deposition of amyloid beta (Aβ) is associated with increased levels of pro-inflammatory factors in AD. It has been shown that release of pro-inflammatory cytokines such as TNFα alters synaptic function and effects cognitive abilities. Despite this, not much information is available to demonstrate whether inhibition of pro-inflammatory cytokines is beneficial for the treatment of AD. The aim of this project is to develop and test cell permeable peptides that are able to pass through the blood brain barrier (BBB) to inhibit the production of pro-inflammatory factors specifically in the brain. During the research placement at the University of Bradford, the student will characterise the already designed peptides in mammalian cultured cells that mimics the properties of mouse and human BBB and primary neurons to test whether the novel peptides permeability.

Title: Characterization of the Arc/Arg3.1 interaction with adaptor proteins and its relevance in Alzheimer disease

Supervision Drs Sonia AL CORRÊA and Jürgen MÜLLER
School of Pharmacy and Medical Sciences
University of Bradford
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Two places for this project

The activity-regulated protein Arc/Arg3.1 is a key coordinator of cognition. In recent years, an extraordinary amount of studies showed that Arc/Arg3.1 mediates different forms of synaptic plasticity, including long-term depression, by directly regulating the trafficking of AMPA receptors (for review Semin Cell Dev Biol, (2018) Volume 77,pages 1-78; Wall et al., 2018). Recently, we showed that Arc/Arg3.1 directly interacts with the clathrin-mediated endocytosis machinery adaptor protein 2 to recruit AMPA receptors for endocytosis (DaSilva et al., 2016) and mediates synaptic activity. Interestingly, increase in neuronal activity has also been associated with an enhanced release of beta amyloid peptides from neurons. The production of beta amyloid is closely dependent on the itinerary of the transmembrane amyloid precursor protein (APP) through the secretory pathway, suggesting that activity may regulate APP trafficking. The aim of this proposal is to establish whether Arc is a mechanistic link between changes in neuronal activity and the machinery that controls protein sorting. During the research placement at the University of Bradford, the student will use hippocampal primary cultures combined with immunocytochemistry and confocal imaging to characterise Arc's function in the secretory pathways and its implication in Alzheimer disease.

References:

Wall MJ, Collins DR, Chery SL, Allen ZD, Pastuzyn ED, George AJ, Nikolova VD, Moy SS, Philpot BD, Shepherd JD, Müller J, Ehlers MD, Mabb AM, Corrêa SAL (2018) The temporal dynamics of Arc expression regulate cognitive flexibility Neuron, 98:1-9.

DaSilva LL, Wall M, deAlmeida LP,Wauters SC, Januário YC, Müller J, Corrêa SAL (2016) Arc controls AMPAR endocytosis through a direct interaction with clathrin-adaptor protein 2. eNEURO DOI: 10.1523/ENEURO.0144-15.2016.

This project is in collaboration with:

Dr Luis Lamberti P da Silva (Faculdade de Medicina de Ribeirão Preto)
Email: lldasilva@fmrp.usp.br

TITLE: The ERK5 cell signalling pathway as a potential neuroprotective mechanism in Alzheimer's Disease

SUPERVISORS: Dr Jürgen MÜLLER, Dr Sonia AL CORRÊA
School of Pharmacy and Medical Sciences
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E-mail: j.muller@bradford.ac.uk

One place for this project

Neurodegenerative diseases have devastating effects on individuals' lives and our society, with no effective treatments available. This is particularly true for Alzheimer's disease, which is multifactorial and develops over a relatively long time. The aetiology of the disease is poorly understood and is likely a combination of causative events and failure of neuroprotective mechanisms. However, relatively little research has focussed on the loss of neuroprotective mechanisms, which could be exploited to slow down or prevent the cognitive decline associated with Alzheimer's disease.

Cellular signal transduction pathways are key regulators of learning in the healthy brain and are frequently mis-regulated in neurodegenerative disorders. Based on previous results and published evidence we hypothesise that disease progression involves the loss of neuroprotective mechanisms and that neuroprotection can be provided by the MAPK (mitogen-activated protein kinase) ERK5. In this project, we will therefore investigate the role of the ERK5 MAPK pathway in the brain using a number of innovative technologies, including biological model systems, biochemistry, molecular biology, modern imaging systems as well as proteomic technologies. This programme of work aims to establish ERK5 as a neuroprotective protein that can be exploited as a novel target for the treatment of Alzheimer's disease and other neurodegenerative disorders.

PARA OUTRAS INFORMAÇÕES SOBRE OS PROJETOS DE PESQUISA, ENTRE EM CONTATO DIRETAMENTE COM OS SUPERVISORES

Erasmus+

USP FACULDADE DE MEDICINA DE RIBEIRÃO PRETO PÓS GRADUAÇÃO

UNIVERSITY of BRADFORD School of Pharmacy & Medical Sciences

CRInt FMRP-USP

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CANDIDATOS APROVADOS:

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Nota: Devido a fatores externos, o professor Rodrigo T. Calado foi substituído pelo professor Eurico de Arruda Neto na banca examinadora.