جمهورية العراق
وزارة التعليم العالي والبحث العلمي
دائرة البعثات والعلاقات الثقافية
قسم الدراسات خارج العراق
شعبة الزمالات الدراسية

بسم الله الرحمن الرحيم

العدد : ص ب/ ١٣/ ٣٠٩١٣

التاريخ : ١٠/٩/٢٠١٤

((معاً لمساندة قواتنا الباسلة لدحر الارهاب))

الجامعات كافة/ قسم العلاقات الثقافية

الهيئات كافة / قسم العلاقات الثقافية

م/ زمالات طبية من جامعة لوند السويدية (الوجبة الاولى)

تحية طيبة...

❖ أولاً / تتوفر لدى دائرة البعثات والعلاقات الثقافية (٦) منح دراسية ممنوحة إلى العـــراق في الاختصاصات الطبية من جامعة لوند السويدية (كوجبة اولى) للحصول على شهادة الدكتوراه في الاختصاصات الطبية وكما هـو مبين فـي الجدول (رقم ١) المرفق رقم (٢) ومفصله بالمرفق رقم (٣) ، ووفقاً للشروط والضوابط المعتمدة في الترشيح للزمالات الدراسية

❖ ثانياً / التفاصيل العلمية للزمالة :

- مدة الدراسة الاكاديمية (٤) سنوات .
- الدراسة باللغة الانكليزية وعلى الطالب الحصول على مستوى لغة كحد ادنى ما يعادل درجة ٥٠٠ توفل .
- حاصل على شهادة الماجستير .
- ان لا يزيد عمر المتقدم عن اربعون عاماً .

❖ ثالثاً / التفاصيل المالية :

- يتحمل الجانب السويدي نفقات الدراسة من اجور دراسية واجور المشرف واجور المختبرات والاجور المكتبية واجور الامتحانات .
- يتحمل الجانب العراقي دفع راتب االطالب طيلة فترة دراسته البالغة اربع سنوات .

❖ رابعاً / راجين تسمية مرشحيكم في ضوء الخلفية العلمية للمتقدمين وسيتم اعتماد مبدأ المنافسة في اختيار المرشحين وفق الضوابط المعتمدة في استمارة المفاضلة بين المتقدمين للزمالات الدراسية حيث سيتم ملئها من قبلنا مراعين أن يتضمن كتاب الترشيح المعلومات المطلوبة التالية (العنوان الوظيفي ، الخدمة الوظيفية بعد آخر شهادة، المعدل ، العمر) بالإضافة إلى تزويدهم بخلاصة خدمة .

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❖ خامساً / الشروط المطلوبة للدراسات العليا :-

- أن يكون المتقدم حاملاً للجنسية العراقية .
- أن لايكون منتسباً للدراسات العليا داخل العراق .
- أن لايقل معدل المتقدم من حملة شهادة الماجستير عن ٧٠% للحصول على شهادة الدكتوراه.
- أن لاتقل خدمة المتقدم عن سنتين بعد آخر شهادة باستثناء الحاصلين على شهادة الماجستير المشمولين بالقرار المرقم (٥١٨) لسنة ١٩٨٦ مشفوعاً بكتاب تأيد من جامعته .
- ان لايزيد عمر المتقدم من حملة شهادة الماجستير عن (٤٠) سنة .

❖ سادساً / المستمسكات المطلوبة بالنسبة للدراسات العليا :

- كتاب ترشيح مثبتاً فيه المعلومات التالية : (أسم المرشح ، المعدل، الخدمة الوظيفية بعد آخر شهادة، المواليد ، اللقب الوظيفي، خلاصة خدمة).
- وثيقتي البكالوريوس والماجستير مترجمتان ومصدقتان حسب الأصول .
- صورة ملونة من جواز السفر نوع (G) أو(A) .
- ملخص بحث الماجستير ومشروع أطروحة الدكتوراه .
- الشهادة الصحية الدولية مترجمة ومصدقة حسب الأصول .
- نسخة من شهادة الجنسية العراقية وهوية الأحوال المدنية مترجمة إلى اللغة الانكليزية ومصدقة من مكتب قانوني .
- السيرة الذاتية باللغة الانكليزية (CV) .
- توصيات عدد (٣) باللغة الانكليزية .
- عدم محكومية مصدقة من وزارة الخارجية .
- نسخة من كتاب المفاتحة للجنة المحلية للمساءلة والعدالة .
- صور حديثة عدد (٣) .

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- كفاءة اللغة الانكليزية (**TOEFL**) ، على ان لا يقل مستوى اللغة عن ٥٠٠ درجة وكما مشار اليه سابقاً .
- يقدم المرشح (٣) ملفات كل ملف يحتوي على المستمسكات المشار إليها آنفاً (حسب طلب الجانب المانح) .

سيكون آخر موعد للتقديم ٢٠١٤/١١/٢ المصادف يوم الاحد ، وان موعد المقابلة في دائرة البعثات والعلاقات الثقافية ٣ /٢٠١٤/١١ المصادف يوم الاثنين و بخلافه يسقط حقه في الترشيح ، آملين أن تصل ترشيحاتكم بأسرع وقت ممكن.

...مع التقـــدير

المرفقات :
- جدول رقم ١ (المرفق رقم ٢) .
- مرفق رقم ٣

أ.م.د. صلاح هادي الفتلاوي
المدير العام لدائرة البعثات والعلاقات الثقافية وكالة
٢٠١٤/٩/١٠

نسخة منه إلى/
- مكتب معالي الوزير/ للتفضل بالاطلاع مع ....التقدير.
- مكتب السيد الوكيل الأقدم / للتفضل بالاطلاع ...مع التقدير .
- مكتب السيد وكيل الوزارة للشؤون العلمية والعلاقات الدولية للتفضل بالاطلاع... مع التقدير.
- مكتب السيد وكيل الوزارة لشؤون البحث العلمي/للتفضل بالاطلاع ..مع التقدير .
- مكتب مستشار الوزارة /للتفضل بالاطلاع ..مع التقدير .
- دوائر مركز الوزارة كافة ...مع التقدير
- قسم الأعلام راجين نشره في وسائل الإعلام المتاحة وسيتم نشره على موقع الوزارة على الانترنت(**www.mohesr gov.iq**). مع التقدير
- قسم الدراسات خارج العراق/الزمالات.
- البريد الدوار.

موقع دائرة البعثات والعلاقات : Website: www.Scrdiraq.com
الدائرة : E-mail: **generaldirector@mohesr.gov.iq**

LUNDS
UNIVERSITET
Department of Surgery
Skåne University Hospital
Professor Henrik Thorlacius

2014-09-13

المرفق رقم (١)

Professor Walled Ameen Mahmood
Iraqi Cultural Attaché
Drottningholmsvägen 26 (1tr)
11242 Stockholm

Dear Professor Mahmood,

It was a pleasure meeting you here in Malmö. I think that our collaboration will be very fruitful for all parties and look forward to work with you and academic institutions in Iraq.

Please find enclosed six different PhD projects (4 years) that I would be happy to accept six of your selected PhD candidates. I have had a long experience working with PhD students from Iraq and know that they will perform excellent in our academic environment. So far I had four completed PhD students and additional two will defend their PhD thesis next year from Iraq.

Looking forward to you response and fruitful collaboration

Yours Sincerely,

Correspondence to:
Henrik Thorlacius, MD, PhD
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Skåne University Hospital
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20502 Malmö
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المرفق رقم (2)

LUNDS UNIVERSITET

جدول رقم (1) الزمالات الطبية لجامعة لوند السويدية

الاختصاص	الجامعة	عنوان المشروع	ت
Medicine or Biology	**Lund**	Role of neutrophil extracellular traps in trypsin activation, inflammation and tissue damage in severe acute pancreatitis	1
Medicine or Biology	**Lund**	Ras signaling in the regulation of neutrophil infiltration and tissue damage in severe acute pancreatitis	2
Medicine or Biology	**Lund**	Monocyte regulation of systemic coagulation and inflammation in abdominal sepsis	3
Medicine or Biology	**Lund**	Rac1-dependent secretion of platelet-derived CCL5 and neutrophil recruitment in septic lung injury abdominal sepsis	4
Medicine or Biology	**Lund**	Streptococcal M1 protein triggers chemokine formation, neutrophil infiltration, and lung injury in an NFAT-dependent manner	5
Medicine or Biology	**Lund**	Histone Deacetylase regulation of trypsin activation, inflammation and tissue damage in acute pancreatitis in mice	6

(3) المرفق رقم 1-6

LUNDS UNIVERSITET

Role of neutrophil extracellular traps in trypsin activation, inflammation and tissue damage in severe acute pancreatitis

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University

Project description:

Background: Neutrophils play a pivotal role in local and systemic complications of acute pancreatitis, but the mechanisms regulating neutrophil-induced tissue damage in the inflamed pancreas is not fully understood. Recently, neutrophil extracellular traps have been demonstrated to contribute to organ dysfunction in both infective and non-infective diseases. In the present study, the potential role of neutrophil extracellular traps in acute pancreatitis will be investigated for the first time.

Methods: Acute pancreatitis will be induced in male C57BL/6 mice by infusion of taurocholate into the pancreatic duct. Extracellular DNA will be stained by Sytox green and neutrophil extracellular trap formation quantified by confocal microscopy and cell-free DNA in plasma. Pancreatic levels of CXC chemokine and histones as well as cytokines and chemokine's in plasma will be determined by ELISA. Neutrophil expression of macrophage-1 antigen will be determined by flow cytometry. To analyze the impact of neutrophil extracellular trap formation in acute pancreatitis, neutrophil extracellular trap depletion will be induced by DNAse I administration. In separate experiments, signal transducer and activator of transcription 3 phophorylation and trypsin activation will be analyzed in isolated acinar cells exposed to neutrophil extracellular traps and histones.

Importance: This project will not only increase the understanding of the pathophysiology behind acute severe pancreatitis but might also open new ways to treat patients with severe acute pancreatitis.

Optimal background of candidate: Medicine or Biology

Project start: As soon as possible

Ras signaling in the regulation of neutrophil infiltration and tissue damage in severe acute pancreatitis

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University

henrik.thorlacius@med.lu.se

Project description:

Background: Neutrophil recruitment is a rate-limiting step in mediating tissue injury in severe acute pancreatitis. However, the signaling mechanisms controlling inflammation and organ damage acute pancreatitis remain elusive. We have recently shown that Rho-kinase regulates trypsin activation and neutrophil recruitment in severe acute pancreatitis. Rho-kinase is one of many effector molecules acting downstream of the Ras superfamily, including more than 50 different small GTPases. In this project we will examine the role of Ras signaling in acute pancreatitis.

Methods: Male C57BL/6 mice will be treated with the Ras inhibitor (farnesylthiosalicylic acid) before to infusion of taurocholate into the pancreatic duct. Pancreatic and lung tissues as well as blood will be collected 24h after pancreatitis induction. Activity of Ras signaling will be examined by use of western blot and immunoprecipitation. Pancreatic levels of CXC chemokines and histones as well as cytokines and chemokines in plasma will be determined by ELISA. Neutrophil expression of macrophage-1 antigen will be determined by flow cytometry.

Importance: If Ras signaling regulates inflammation and tissue damage in severe acute pancreatitis new possibilities to use already existing drugs to target Ras signaling in acute pancreatitis emerge. Thus, this work will not only highlight a potential new signaling mechanism in AP but also indicate whether targeting Ras signaling could be effective in order to attenuate local and systemic inflammation in severe acute pancreatitis.

Optimal background of candidate: Medicine or Biology

2-6

Project start: As soon as possible

Monocyte regulation of systemic coagulation and inflammation in abdominal sepsis

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University
henrik.thorlacius@med.lu.se

Project description:

Background: Hemostatic dysfunction is one of the most prominent features in sepsis. Hemostatic cascades are complex processes consisting of a dynamic interplay between several discrete elements. Individual quantification of these elements by classical assays including the ones used to measure coagulation factors and inhibitors, do not capture the global effect of all these elements in hemostasis. Instead, global hemostasis assays, including thrombin generation tests and thromboelastometry have emerged as effective tools to obtain more comprehensive evaluations of hemostasis. Thrombin generation has been shown to be useful in the evaluation of diseases with complex changes in hemostasis, such as chronic liver disease and trauma-induced coagulopathy. Herein, we hypothesized that monocytes might play a role in regulating thrombin generation and coagulation factor consumption in sepsis.

Methods: This project will include setting up useful assays for determining thrombin generation via both extrinsic and intrinsic pathways. Abdominal sepsis will be induced by cecal ligation and puncture in C57/Bl6 mice. Plasma and lung levels of interleukin-6 (IL-6), CXC chemokines and pulmonary activity of myeloperoxidase, thrombin generation and coagulation factors will be determined after CLP induction. Administration of clodronate liposomes is used to deplete animals of monocytes.

Importance: This project will help to elucidate the role of monocytes in the pathophysiology of sepsis and encourage further attempts to target monocytes in order to ameliorate hemostatic dysfunction and pathological inflammation in abdominal sepsis.

Optimal background of candidate: Medicine or Biology

3-6

Project start: As soon as possible

Project number 4 between Iraq Cultural Attache and Lund University, Henrik Thorlacius

Rac1-dependent secretion of platelet-derived CCL5 and neutrophil recruitment in septic lung injury abdominal sepsis

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University
henrik.thorlacius@med.lu.se

Project description:

Background: A growing body of evidence suggests that platelets exert pro-inflammatory actions. Platelets contain a plethora of potential mediators, including chemokines, capable of stimulating leukocyte activation and recruitment. One of the most prevalent chemokine in platelets is CCL5, which belong to the CC chemokine family and is a potent stimulator of leukocytes. The intracellular signaling cascades triggering platelet secretion of CCL5 are not well understood. It has been reported that Rac1 is expressed in platelets and that Rac1 is essential for granule secretion, clot retraction, and phospholipase Cγ2 activation in platelets. This project will focus on the role of Rac-1 in regulating platelet secretion of CCL5 as well as the function of CCL5 in controlling neutrophil recruitment and lung damage in abdominal sepsis.

Methods: Abdominal sepsis is induced by cecal ligation and puncture in C57/Bl6 mice. Platelet secretion of CCL5 will be examined by use of confocal microscopy. Levels of CCL5 in plasma and lungs will be quantified by ELISA. Flow cytometry will be used to determine surface expression of Mac-1, CCR1 and CCR5 on neutrophils and alveolar macrophages. Antibodies against CCL5 and CCL5-deficient animals will be used.

Importance: These studies will define the role Rac1 in regulating platelet secretion of CCL5. Also, the direct role of CCL5 in sepsis-induced neutrophil recruitment in the lung will be clarified. Thus, this project can be useful to identify new ways to ameliorate lung damage in sepsis.

Optimal background of candidate: Medicine or Biology

4-6

Project start: As soon as possible

Project number 5 between Iraq Cultural Attache and Lund University, Henrik Thorlacius

Streptococcal M1 protein triggers chemokine formation, neutrophil infiltration, and lung injury in an NFAT-dependent manner

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University
henrik.thorlacius@med.lu.se

Project description:

Background: The microbial causes of septic shock have been dominated by Gram-negative bacteria but a recent resurgence of Gram-positive bacterial infections has changed the microbial etiology in septic patients. *Streptococcus pyogenes*, especially the M1 serotype, is a common cause of Gram-positive infections presenting with a severe and fatal condition, i.e. streptococcal toxic shock syndrome. The purpose of the present study was to examine the role of nuclear factors of activated T cells (NFAT) signaling in streptococcal M1 protein-induced lung injury.

Methods: NFAT-luciferase reporter mice will be treated with the NFAT inhibitor A-285222 prior to administration of M1 protein. Neutrophil infiltration, edema, and CXC chemokines will be quantified in the lung 4h after challenge with M1 protein. Flow cytometry was used to determine Mac-1 expression on neutrophils. Alveolar macrophages will be isolated to study the role of NFAT signaling for cytokine secretion.

Importance: These studies will demonstrate if inhibition of NFAT can attenuate CXC chemokine generation, neutrophil activation and recruitment in streptococcal M1 protein-induced lung edema formation and tissue injury. Thus, our might identify a novel NFAT-dependent signaling pathway which can be used to protect against pulmonary damage in severe infections caused by *Streptococcus pyogenes*.

Optimal background of candidate: Medicine or Biology

Project start: As soon as possible

5-6

Histone deacetylase regulation of trypsin activation, inflammation and tissue damage in acute pancreatitis in mice

Main Supervisor: Henrik Thorlacius, Department of Surgery, Lund University
henrik.thorlacius@med.lu.se

Project description:

Background: Acetylation and deacetylation near the N-terminal end of core histones is believed to regulate the accessibility of the chromatin to transcription factors and thereby controlling gene transcription. This dynamic process is coordinated by histone acetylases and a family of histone deacetylases (HDAC). The aim of this study is to investigate the potential role of HDAC in regulating trypsin activation, inflammation and tissue damage in severe acute pancreatitis.

Methods: Male C57Bl/6 mice will be treated i.p. with the HDAC inhibitor trichostatin A (2 mg/kg) prior to retrograde infusion of taurocholic acid (5%) into the pancreatic duct. Activity of different subtypes of HDACs will be examined. Tissue morphology, serum levels of amylase, pancreatic levels of macrophage inflammatory protein-2 as well as myeloperoxidase activity in the pancreas and lung will be determined 24h after taurocholate challenge. The role of HDAC for trypsin activation will also be analysed in isolated acinar cells. Quantitative RT-PCR is used to examine gene expression of pro-inflammatory mediators in the pancreas.

Importance: This project will define whether HDAC activity plays an important role in acute pancreatitis by regulating trypsin activation, formation of pro-inflammatory mediators or leukocyte recruitment in the inflamed pancreas. Thus, these efforts will not only illustrates the significance of acetylation in the development of pancreatitis but also reveal if HDAC activity might serve as a useful target to ameliorate tissue injury in AP.

Optimal background of candidate: Medicine or Biology

Project start: As soon as possible

6-6