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Salute

# PARTNER SEARCH HEALTH-EU-LCP-8

*01 dicembre 2017*

PARTNER SEARCH HEALTH-EU-LCP-8

Richiesta di un istituto di ricerca francese alla ricerca di partner da includere in un loro progetto da presentare nel programma COOPERATION tematica SALUTE.
Per maggiori informazioni sulla Ricerca Partner e per conoscere i contatti del proponente, potete consultare il seguente indirizzo web http://www.apre.it/formaAssist/scheda.asp?id=1040

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------- PARTNER SEARCH HEALTH-EU-LCP-8 --------
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<Reference n.: HEALTH-EU-LCP-8>
<Deadline: 30/09/2008>
<Programme: >
<Project Title: Role of inflammation in the development of chronic kidney disease (CKD).>
<Financial Scheme: Progetti in collaborazione - Large>
<Description: Priorities' Main Research Areas:
Background: Endothelium dysfunction and vasculitis
Vasculitis may be the outcome of a number of pathological events in which inflammatory damage to the endothelium leads to necrosis of the vessel wall and often to vessel destruction. Although vasculitis may be associated with a number of clinical conditions, it is the predominant disease manifestation in syndromes such as Wegener's granulomatosis (WG) and microscopic polyangiitis (MPA), with which the presence of anti-neutrophil circulating antibodies (ANCA) is associated.
The aetiology of the primary systemic vasculitis remains obscure. Recent years have seen significant advances in the understanding of inflammation and in particular the role of and interaction between the vascular endothelium, mediators and immune effector cells. The specific processes relevant to vasculitis which result in EC damage include factors such as cytokines and circulating autoantibodies, immune complexes, neutrophil and EC activation and proteinase 3 expression and regulation play a role. Nevertheless, the mechanisms that

drive systemic vascular endothelial dysfunction are still unclear.
Vascular endothelial dysfunction is common to a range of immune-mediated inflammatory diseases, seen in multiple vascular beds, and could be reversible upon disease remission. However, persistence of endothelial dysfunction in systemic vasculitis appears to have long-term consequences, leading to the acceleration of atherosclerosis. Moreover, vascular damage in critical vascular beds such as in the kidney, lung and brain can very rapidly lead to tissue destruction and organ failure. Therefore a rapid diagnostic test for the presence and outcome of vasculitis is needed. Indeed, vasculitis often pose difficulties with regard to diagnosis and monitoring of disease activity, both at the initial presentation and during follow-up. Although ANCA positivity was associated with relapse, discordance between cytoplasmic ANCA and disease activity was not unusual.Therefore, novel markers of disease activity are therefore awaited. Recently, some potential markers have emerged such as circulating endothelial cells but serological marker of disease activity and progression in active limited and generalized WG and other vasculitis reflecting the degree of endothelial cell damage remains to be found.

Project description
Hypothesis:
We hypothesize that the endothelial-restricted surface expression and release of soluble non classical MHC class I molecules (including HLA-E, MICA and ULBPs) confer new immunoregulatory functions to vascular endothelial cells that we want to characterize in the present proposal.
We also postulate that the recent identification of soluble HLA-E molecules by our group (INSERM patents pending) has important implications for the understanding of immune-mediated and vascular diseases and for the diagnosis and monitoring of patients.

Objectives:
The basic aim of the proposal is to establish the roles that non classical MHC class I molecules molecules expressed on and released by vascular EC may play in vascular cytoprotection processes and to determine the molecular mechanisms involved in their expression, regulation and shedding. Another important aim of this proposal is to evaluate whether the soluble HLA-E molecules and other non classical MHC class I molecules produced by damaged EC could be useful for the diagnosis and to predict vasculitis outcome either alone or in addition to other molecular markers of the disease.

Keywords:
Chronic Kidney Disease, inflammation, anti-neutrophil cytoplasmic antibody (ANCA)-associated systemic vasculitis, endothelium, MHC, Vascular injury and cytoprotection, molecular targets, biomarkers, immunotherapy

TOPICS DRAFT:
Cellular and molecular mechanisms of development of chronic kidney disease (CKD). FP7-HEALTH-2009-single-stage>

<Organisation Type: Centro di Ricerca>
<Partner Sought: Role: technology development, research, training, dissemination, demonstration
Country /region: Europe
Start of partnership: start-up phase
Expertise required: Any expertise in the field of inflammation pertaining to the development of chronic kidney disease

For further information about this Partner Search, including Contact Person's details, please consult this web address: http://www.apre.it/formaAssist/scheda.asp?id=1040