COLLOQUIA IN PHYSIOLOGY AND VASCULAR BIOLOGY

Venue: Medical University Vienna, Center for Physiology and Pharmacology, Institute of Pharmacology, Waehringerstrasse 13a, 1090 Vienna, "**Leseraum**"

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Friday 18.12.2015 10:00 s.t. **Ilaria Canobbio** (host: H. Schmid)

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"Platelets as a link between vascular inflammation and Alzheimer's disease"

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Abstract: Alzheimer's disease (AD) is the most invalidating neurological disorder in the elderly. It is characterized by abnormal deposition of amyloid beta peptides in the brain parenchyma that disrupt synaptic signaling. Amyloid deposition is also found in cerebral vessels together with chronic inflammation. Emerging evidence points to a central role of cerebrovascular dysfunctions in the onset of AD. According to the "vascular hypothesis", AD may be initiated by vascular dysfunctions that cause cerebral hypoperfusion and neuron suffering. Indeed, multiple bidirectional connections exist between AD and cerebrovascular dysfunctions. Vascular risks factors, such as diabetes, hypertension and hypercholesterolemia, predispose and accelerate AD and conversely AD patients show increased hemorragic or ischemic stroke risk.

In this new scenario, blood platelets play a crucial role. Circulating platelets are responsible for haemostasis and thrombosis but are also implicated in neuroinflammation. Interestingly, blood platelets contain a high concentration of amyloid precursor protein (APP) and generate amyloid beta peptides. Following activation, platelets release amyloid beta peptides and inflammatory molecules in the circulation that in turn stimulate cell activation and exacerbate vascular inflammation.

Altered morphology and metabolism and increased levels of activated platelets have been documented in AD patients and AD mouse models together with an augmented state of vascular inflammation. Therefore, targeting platelets in AD should aim at preventing their prothrombotic/proinflammatory action. In addition, platelets and platelet-related proteins may represent a potential peripheral easy-to-access biomarkers for AD. All these aspects of the complex interplay between blood platelets, vascular inflammation and AD will be discussed.