External Seminar

Speaker

Prof. Hugues Abriel, MD PhD,
Director,
Department of Clinical Research (DCR) and
Groupleader Ion Channels and Channelopathies
Research Group DCR,
University of Bern, Switzerland

Place: Auditorium-Hörsaal
(INF 410)

Date: Tuesday, October 27th, 2015

Time: 5.00 pm

Title: “Cardiac sodium channel Nav1.5 and interacting proteins: roles in cardiac channelopathies”

The field of molecular arrhythmology has progressed at an impressive pace during the past 20 years. Throughout the years, we have learned more and more about the genetic factors and molecular mechanisms underlying electrical abnormalities of the heart such as congenital long QT syndrome (LQTS) and Brugada syndrome. Since in most cases, the genes that are found to be mutated are encoding either the pore-forming subunit of cardiac ion channels or of ion channel regulatory proteins, the term “genetic cardiac channelopathies” has been used to define these disorders. Among the still-growing list of genes that lead to genetic cardiac channelopathies, the role of the gene, SCN5A, is truly unique. The gene SCN5A encodes the pore-forming subunit of the cardiac sodium channel, Nav1.5, which is the main channel responsible for the cardiac sodium current. Hundreds of genetic variants have been reported. The striking point here is that these variants were found in patients with a long list of distinct clinical manifestations ranging from delayed repolarization (in LQTS) to structural abnormalities (in the case of patients with dilated cardiomyopathies). During this research seminar, new findings related to the roles and regulation of Nav1.5 in cardiac cells will be presented. In particular, most of the recent results are coming from the investigation of genetically-modified mouse models.

Host: Prof. Dr. med. Patrick Most
Chair, Molecular and Translational Cardiology Section
Department of Internal Medicine III
University of Heidelberg