BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Kathrin Banach	Assistant Professor
eRA COMMONS USER NAME	
kbanach	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
University of Bochum, Bochum, Germany	Diplom	1987 - 1992	Biology
University of Berne, Switzerland	PhD	1992 - 1996	Physiology

A. Personal Statement

During my training and career I have obtained extensive experience in cardiac electrophysiology, which encompasses single cell as well as double whole cell voltage- and current-clamp measurements and field potential recordings with multi-electrode arrays from cardiomyocyte monolayers as well as from the epicardial surface of Langendorff perfused mouse hearts. This enables me to design and to analyze electrophysiological data on a cellular level as well as in multicellular and organ preparations. I received further training in intracellular Ca imaging that, as shown in my publications, I successfully implemented into my research projects. In my laboratory at Rush electro-physiological techniques (single, double whole cell voltage clamp and current clamp as well as multi-electrode array recordings) can be combined with imaging techniques and biochemical as well as molecular biological techniques (western blotting, immunofluorescence, subcellular fractionation etc.) are established to quantify and alter cellular protein levels. Using these techniques my laboratory has made substantial contributions in determining the mechanism of triggered cardiac activity during physiological pacemaker activity as well as during pathophysiological arrhythmic events e.g. during ischemia-reperfusion injury. A recent study describing the role of ROS dependent NCX regulation in ventricular arrhythmic activity was published early in 2014.

In the current research proposal we will test the exciting new hypothesis that Pak1 stimulation can provide cardioprotection during anthracycline therapy. The assembled research team is ideally suited for this project. With the expertise of Dr. L.A. Blatter and myself we are in the position to identify the relevant cellular mechanism of Pak1 induced cardioprotection. These will then lead to the targeted development of Pak1 mimetic peptides to avoid the necessity of systemic Pak1 stimulation. The integration of Dr. Feinstein (Professor/Medicine, Director/Echocardiography, Rush) into the study as an expert in contrast-enhanced ultrasound (CEUS) allows us to include a therapeutically relevant gene delivery mechanism and thereby enhance the translational relevance of our study. We believe that the research team assembled for this study brings together a unique set of expertise that is necessary for the successful completion of the current proposal.

Positions

05/91 - 07/92 Diploma Student, Dept. of Cellular Physiology, Univ. of Bochum, Germany.
08/92 - 08/95 Ph.D. Student, Dept. of Physiology, Univ. of Bern, Switzerland.
12/95 - 04/96 Postdoc, Dept. of Pharmacology, SUNY at Syracuse, NY, USA.
05/96 - 12/97 Postdoc, Dept. of Physiology and Biophysics, SUNY at Stony Brook, NY, USA.
01/98 - 07/99 Postdoc, Dept. of Physiology, Univ. of Bern, Switzerland.
08/99 - 12/01 Research Scientist (Habilitant), Dept. of Neurophysiology, Univ. of Koeln, Germany.
01/02 - 09/07 Research Assistant Professor, Dept. of Physiology, Loyola Univ. Chicago, IL, USA.
09/07 - 08/14 Assistant Professor, Dept. of Medicine/Cardiology, University of Illinois at Chicago.
08/14 - present Assistant Prof., Dept. of Molecular Biophysics and Physiology, Rush University Medical Center, Chicago, USA

Honors and Awards

12/95 -12/97 Post-Doc. Fellowship of the BASF, awarded by the "Studienstiftung des deutschen Volkes"

11/97 "Habilitation"-Fellowship funded by the Government of Nordrhein Westfalen (I returned the award because I accepted a position at the Department of Physiology in Bern)

05/00 -12/01 Lise Meitner "Habilitation"-Fellowship funded by the Government of Nordrhein Westfalen.

06/00 -12/01 Maria-Pesch Foundation; Research Grant; University of Koeln

05/01 -12/01 BMBF Research Grant in collaboration with MEDIGENE

B. Selected peer-reviewed publications

Relevant to the current application (15 out of 30)

1. Kapur N, Mignery G & **Banach K.** (2006) Cell Cycle Dependent Calcium Oscillations in Mouse Embryonic Stem Cells. *Am J Physiol Cell Physiol.* 292(4):C1510-C1518.
2. Shang LL, Pfahnl AE, Sanyal S, Jiao Z, Allen J, **Banach K**, Fahrenbach J, Weiss D, Taylor WR, Zafari AM & Dudley SC, Jr. (2007). Human Heart Failure Is Associated With Abnormal C-Terminal Splicing Variants in the Cardiac Sodium Channel. *Circ Res.;*101(11):1146-54
3. Kapur N and **Banach K**. (2007) IP3 Mediated Ca Signaling Drives Pacemaker Activity in Early ES Cell Derived Cardiomyocytes. J. Physiol 58: 1113–1127. PMC2170837
4. Fahrenbach J, Mejia-Alvarez R and **Banach K**. (2007) The Relevance of Non-Excitable Cells for Cardiac Pacemaker Function. *J Physiol* ; 585(Pt 2):565-78. PMC2375482
5. Fahrenbach JP, Ai X & **Banach K.** (2008). Decreased intercellular coupling improves the function of cardiac pacemakers derived from mouse embryonic stem cells. *J Mol Cell Cardiol* **45,** 642-649. PMC2598737
6. Rinne A, **Banach K** & **Blatter LA**. (2009). Regulation of nuclear factor of activated T cells (NFAT) in vascular endothelial cells. *J Mol Cell Cardiol* **47,** 400-410. PMCID: PMC2779755
7. Grajales L, Garcia J, **Banach K** & Geenen DL. (2010). Delayed Enrichment of Mesenchymal Cells Promotes Cardiac Lineage and Calcium Transient Development. *J Mol Cell Cardiol* **48,** 735-745. PMCID: PMC2837799
8. Rinne A, Kapur N, Molkentin JD, Pogwizd SM, Bers DM, **Banach K** & **Blatter LA**. (2010). Isoform- and tissue-specific regulation of the Ca2+-sensitive transcription factor NFAT in cardiac myocytes and in heart failure. *Am J Physiol Heart Circ Physiol* **298,** H2001-2009. PMCID: PMC2886636
9. DeSantiago, JD, Bare D, Semenov I, Minshall RD, Geenen DL, Wolska BM, and **Banach K** (2012). Excitation-Contraction Coupling in Ventricular Myocytes is Enhanced by Paracrine Signaling from Mesenchymal Stem Cells. *J Mol Cell Cardiol* 52, 1249-1256. PMCID: PMC3570146
10. Mureli S, Gans CP, Bare DJ, Geenen DL, Kumar NM, and **Banach K** (2013). Mesenchymal Stem Cells Improve Cardiac Conduction by Up-Regulation of Connexin 43 Through Paracrine Signaling. *Am J Physiol Heart Circ Physiol* 304:H600-H609 (Podcast Featured Article) PMCID: PMC3566487
11. DeSantiago J, Bare DJ and **Banach K.** (2013) Protection from Ischemia Reperfusion Injury by Mitochondrial $I_{K,ATP}$ Activation Through Mesenchymal Stem Cell Derived Paracrine Factors. *Stem Cell and Development;* 22: 2497-2507; PMCID: PMC3760058
12. DeSantiago J, Bare DJ, Ke Y, Sheehan KA, Solaro RJ and **Banach K** (2013). Functional integrity of the t-tubular system in cardiomyocytes depends on p21-activated kinase 1. *J. Mol. Cell. Cardiol.* 60:121–128. PMCID: PMC3679655
13. Taglieri DM, Johnson KR, Burmeister BT, Monasky MM, Spindler MJ, Desantiago J, **Banach K**, Conklin BR, Carnegie GK. (2014)The C-terminus of the long AKAP13 isoform (AKAP-Lbc) is critical

for development of compensatory cardiac hypertrophy. *J. Mol. Cell. Cardiol.* 66: 27–40.

14. DeSantiago J, Bare DJ, Xiao L, Ke Y, Solaro RJ, **Banach K**. (2014) p21-Activated kinase1 (Pak1) is a negative regulator of NADPH-oxidase 2 in ventricular myocytes. *J. Mol. Cell. Cardiol.* 67: 77-85. PMCID: PMC3930036

15. Kapoor N, Maxwell JT, Mignery GA, Will D, **Blatter LA**, **Banach K**. (2014) Spatially Defined InsP3-Mediated Signaling in Embryonic Stem Cell-Derived Cardiomyocytes. *PLoS One* 9: e83715. PMCID: PMC3883750

D. Research Support

Completed Research Support:

NIH 1R01HL089617 07/2007-06/2013

Title: " Embryonic Stem Cell Derived Biological Pacemaker for the Heart"

Role: P.I.

The major goal of this grant is to determine the ability of embryonic stem cell derived cardiomyocyte aggregates to function as biological pacemaker for the heart and to determine how electrophysiological properties of the gap junction channels expressed in biological pacemakers influence their functional integration and their ability to gain and maintain pacemaker dominance.

NIH 3R01HL089617-03S

Title: " Embryonic Stem Cell Derived Biological Pacemaker for the Heart"

Role: P.I. 07/2009 - 06/2011

The supplement builds on the aims of the main grant. It extends the proposed projects by determining the gap junction expression in stem cells and the cardiomyocyte preparations used by quantitative real time PCR. Further we extended the characterization of the pacemaker mechanism in embryonic stem cell derived cardiomyocytes by taking advantage of a 2D confocal imaging system and flash release of caged IP_3 to identify functionally relevant IP_3 signaling domains in cardiomyocytes.

NIH 1PO1 HL080101-01 Program Project Grant (PI: DM Bers)

Title PPG: „CaMKII and IP_3-mediated signaling in cardiac myocytes“

Title Project II: “Ca and IP_3 signaling in cardiac myocytes” (PI: L.A. Blatter)

Role: Co-Project Leader, 12/2005 - 11/2010

NIH 1R01HL075115-01A1 (PI: Leanne Cribbs)

Title: "T-type Calcium Channels in Development and Cell Proliferation"

Role: Co-P.I. 07/2004 - 06/2007

American Heart Association ‘Predoctoral Fellowship’

Title: “The Use of Embryonic Stem Cell Derived Cardiomyocytes as Biological Pacemakers”

Role: Supervisor 01/2005 - 12/2007