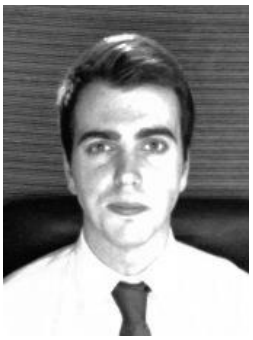
Curriculum vitae



Name: Mihály Ruppert
Nationality: Hungarian
Date of birth: 24.05.1990.
Address: 9024, Győr Bem tér 13.
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Education:
Czuczor Gergely Benedictine High School, Győr, 2005-2009
Whitgift School London, United Kingdom, 2008
*Semmelweis University, Faculty of Medicine, B*udapest, 2009-
Buffalo School of Medicine and Biomedical Sciences, Buffalo, New York State, USA, 2014
Ruprecht-Karls-Universität Heidelberg, Germany, 2014-2015

Academic Competitions:
2011 Semmelweis University, Medical Biochemistry Academic Competition III. Place
2012 Semmelweis University, Microbiology Academic Competition IV. Place
2013 Semmelweis University, Pharmacology Academic Competition II. Place
2013 Semmelweis University, Cardiology Academic Competition I. Place

Languages: English (advanced, C1), Italian (intermediate, B2)

Research experience:

Research Institutes:

1. / Semmelweis University, Heart Centre Research Laboratory, Budapest
2. / Ruprecht-Karls-Universität, Experimental Heart Surgery laboratory, Heidelberg

Tutor: Tamás Radovits MD, PhD

Scientific Students' Conferences::
2014 Annual Scientific Students' Associations Conference, Budapest I. Prize
2014 Annual Scientific Students' Associations Conference, Budapest II. Prize
2014 Scientific Forum of Korányi Frigyes, Budapest I. Prize
2014 International Student's Conference at Marosvásárhely (Romania) Special Prize
2015 Annual Scientific TDK Conference, Budapest I. Prize
2015 Semmelweis International Students' Conference, Budapest II. Prize
2015 XXXII. National Scientific Students' Associations Conference, Budapest I. Prize
2015 XXXII. National Scientific Students' Associations Conference, Budapest I. Prize
2015 XXXII. National Scientific Students' Associations Conference I. Prize

Awards:
2014-2015 Republican Scholarship
2014 Hungarian Medical Association Of America (HMAA) Scholarship - Buffalo School of Medicine and Biomedical Sciences
2014 Scientific Scholarship of the Order of Malta, Hungarian Association's North American Delegation
2014-2015 Jellinek-Harry Research Scholarship
2015 Semmelweis University Scientific Scholarship
2015 Stephen W. Kuffler Research Scholarship

Publications

1. Publications

Attila Oláh, Balázs Tamás Németh, Csaba Mátyás, Eszter Mária Horváth, László Hidi, Ede Birtalan, Dalma Kellermayer, ***Mihály Ruppert***, Gergő Merkely, Gábor Szabó, Béla Merkely, Tamás Radovits.
Cardiac effects of acute exhaustive exercise in a rat model.
Int J Cardiol. 2014; 182:258-266
IF: 6.175

Attila Kovács, Attila Oláh, Árpád Lux, Csaba Mátyás, Balázs Tamás Németh, Dalma Kellermayer, ***Mihály Ruppert***, Marianna Török, Lilla Szabo, Anna Meltzer, Alexandra Assabiny, Ede Birtalan, Béla Merkely, Tamás Radovits.
Strain and strain rate by speckle tracking echocardiography correlate with pressure-volume loop derived contractility indices in a rat model of athlete's heart.
Am J Physiol Heart Circ Physiol. 2015; 308:743-8.
IF: 4.012

Sevil Korkmaz-Icöz,Alice Lehner, Shiliang Li,Adrian Vater, Tamás Radovits,Péter Hegedűs, ***Mihály Ruppert***, Paige Brlecic, Markus Zorn, Matthias Karck, Gábor Szabó.
Mild Type 2 Diabetes Mellitus Reduces the Susceptibility of the Heart to Ischemia/Reperfusion Injury: Identification of Underlying Gene Expression Changes.
Journal of Diabetes Research 2015 doi: 10.1155/2015/396414
IF: 2,2

Sevil Korkmaz-Icöz, Adrian Vater, Shiliang Li, Alice Lehner, Tamás Radovits, Peter Hegedűs, ***Mihály Ruppert***, Paige Brlecic, Markus Zorn, Matthias Karck, Gábor Szabó.
Mild type 2 diabetes mellitus improves remote endothelial dysfunction after acute myocardial infarction.
Journal of Diabetes and its Complications 2015 doi : 10.1016/j.jdiacomp.2015.06.012
IF: 3,1

Oláh Attila, Lux Árpád, Németh Balázs Tamás, Hidi László, Birtalan Ede, Kellermayer Dalma, Mátyás Csaba, ***Ruppert Mihály***, Merkely Gergő, Szabó Gábor, Merkely Béla, Radovits Tamás. A sportszív részletes hemodinamikai jellemzése bal kamrai nyomás-térfogat analízis segítségével
Cardiol Hung. 2013; 43:224-32

2. Presentations

Németh BT, Mátyás C, Oláh A, Hidi L, ***Ruppert M***, Lux, Á, Kellermayer D, Birtalan E, Merkely G, Merkely B, Radovits T.
Pharmacological activation of the soluble guanylate cyclase inhibits pressure overload-induced cardiac hypertrophy.
Semmelweis University PhD Scientific Days 2014, Budapest

Mátyás C, Oláh A, Németh BT, Hidi L, ***Ruppert M***, Lux Á, Kellermayer D, Merkely B, Radovits T A szolubilis guanilát-cikláz gyógyszeres aktiválása meggátolja a nyomás-indukált szívizom-hipertrófia kialakulását
Cardiologia Hungarica 2014; 44 (Suppl. E): 31
Annual meeting of the Hungarian Society of Cardiology, Balatonfured, 2014

Németh BT, Mátyás C, Oláh A, Hidi L, ***Ruppert M***, Lux Á, Kellermayer D, Merkely G, Merkely B, Radovits T.
Pharmacological activation of the soluble guanylate cyclase inhibits pressure overload-induced cardiac hypertrophy.
Cardio Croat 2014;9:245
22nd Annual Meeting of the Alpe-Adria Cardiology Association, Opatija, Croatia, 2014

3. Posters

Kellermayer D, Oláh A, Lux Á, Németh BT, Hidi L, Birtalan E, ***Ruppert M***, Mátyás C, Merkely B, Radovits T.
Detailed Hemodynamic Characterization of Athlete's Heart using Left Ventricular Pressure-Volume Analysis in a Rat Model.
58th Annual Meeting of the Biophysical Society, San Francisco, 2014
Biophysical Journal 2014; 106: 344a.

Radovits T, Mátyás C, Oláh A, Németh BT, Hidi L, ***Ruppert M***, Lux Á, Merkely G, Kellermayer D, Merkely B.
Pharmacological activation of the soluble guanylate cyclase inhibits pressure overload-induced cardiac hypertrophy.
Frontiers in Cardiovascular Biology 2014, Barcelona, 4th-6th July 2014.
Cardiovasc Res 2014; 103 (Suppl.1): S93

Area of research interest

In our research we try to indentify novel signaling pathways in pathological myocardial hypertrophy and gain a deeper understanding about the mechanisms that take place during the transition from compensated hypertrophy to congestive heart failure.

Cardiac hypertrophy is a well-known response reaction of the heart to sustained pressure-overload. Although it contains initial salutary components, in a long term it leads to chronic heart failure and sudden death.

Despite the fact that the signaling pathways involved in pathological hypertrophy offer many opportunities to intervene, the drugs that are currently in clinical use are not sufficiently effective. However the nitric oxide (NO) – soluble guanylate cyclase (sGC) – cyclic guanosine monophosphate (cGMP) - cGMP-dependent protein kinase G pathway has been increasingly identified as an effective anti-hypertrophic signaling.

Therefore we investigate the effects of chronic activation of the cGMP producing enzyme, the soluble guanylate cyclase (sGC) by cinaciguat in rat models of pressure overload-induced cardiac hypertrophy.