Bruno B. Queliconi, PhD

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Education

University of São Paulo, Chemistry Institute, Department of Biochemistry, 2009-14
PhD in Biochemistry and Molecular Biology, scholarship (~US$100k) and research grant (~US$25k)

University of São Paulo, Biology Institute 2005-09
B.Sc. in Biological Sciences, national level scholarship renewed for 3 years (R$4K/~US$2.6k)
Best South American University, accepted on the top 3% classification

Leadership & Accomplishments

13 international publication *(+400 citations /* 4 oral presentations and 9 posters*)* 2007-present
- 4 publications as 1st and 5 as 2nd author, with a 1st author publication as an undergraduate

Consultant for the Metabolism and Mitochondrial Research Core at Cedars-Sinai 2014
- Worked to identify the relevant questions and created the appropriated protocols

Founder and President of the PhD students Association (Team of 12, representing 300 students) 2009-12
- Created new tools for collaboration and efficient resource use, with +60% adoption and recurrent use
- Worked with students and professors to create a framework to produce solutions to problematic disciplines

Brazilian stock portfolio, producing 2x+ the local index (IBOV) 2005-present
- Developed an investment strategy focusing on company growth stage, finance statement and its market fit
- Successfully implemented on variety of family reserves, while carefully selecting REITs to diversify risks

Work experience

➢ ***Postdoctoral Fellow*** at Stanford University, School of Medicine, June 2018-Present
Department of Chemical and Systems Biology, Stanford, CA, USA

Consultant about Mitochondrial and protein interaction topics. Developing a new research line exploring ALDH2 and neurodegeneratives disease

➢ ***Postdoctoral Fellow*** at Tokyo Metropolitan Institute of Medical Science Nov. 2014- 2018
Department of Advanced Science for Biomolecules, Tokyo, Japan

Discovered a new mitochondrial import mechanism that affects disease linked proteins (1st author work sent to *Nature* for review), improved the statistical analysis and created high throughput assays to measure protein function for the whole laboratory

➢ ***Visiting PhD Student*** at Cedars-Sinai Hospital June 2013-2014
Heart Institute, Los Angeles, CA, USA

Served as the point of contact on mitochondrial metabolism, characterized a new factor affecting ischemia damage and validated the function of drug candidate proteins in Pig hearts

➢ ***Visiting PhD Student*** at University of Rochester Medical Center June-Oct. 2011
Department of Medicine, Rochester, NY, USA

Developed a new ischemia-reperfusion model in *C. elegans*. This model was essential for at least 3 additional publications and is still in use producing results

➢ ***Undergraduate researcher and PhD candidate*** at University of São Paulo, 2005-2014
Chemistry Institute, Department of Biochemistry, São Paulo, Brazil

Worked with mitochondrial and heart physiology, starting an independent research line as an undergraduate. In addition to the scientific works, coordinated national and international collaborations that still produce results.

Selected Awards and Honors (4 of 8)

- JSPS Postdoctoral Fellow - successful rate 10% - Stipend US$86k + Research US$25k 2016
- Young Scientist Program Award from The International Union of Biochemistry and Molecular Biology (successful rate 15%, +300 contenders) 2015
- American Society for Biochemistry and Molecular Biology PROLAB Prize - US$4.5k grant 2011
- Undergraduate selected for oral presentation at the International Cone Sul Symposia 2008

Selected Publications (4 of 15)

➢ Redox regulation of the mitochondrial K(ATP) channel in cardioprotection

Biochim. Biophys. Acta. – Molecular Cell Resarch (80 years old, traditional basic research journal) 2011

Queliconi, B.B., Wojtovich, A.P., Nadtochiy, S.M., Kowaltowski, A.J., Brookes, P.S.

Mitochondrial physiology is highly regulated, and reactive oxygen species (ROS) is a component that regulate and damage the cell, but with no negative feedback loop to control its production. I coordinated a collaboration of our Brazilian group with an American group to publish a comprehensive analysis of a mitochondrial channel has a negative feedback loop to control ROS. This worked consolidated an essential mechanism of cell protection.

➢ Bicarbonate increases ischemia-reperfusion damage by inhibiting mitophagy

PLoS One (Biggest open science journal) 2016

Queliconi, B.B., Kowaltowski, A.J., Gottlieb, R.A.

We saw in the literature that bicarbonate derived radicals were imported in redox reactions, but there was a lack of knowledge on its role on reperfusion damage, where it is widely used in clinical settings. We showed that bicarbonate increases damage during reperfusion, being a new source of damage. In this work I was responsible for the conception, design, and data collection, while the other authors provided financial and intellectual support.

➢ Exercise reestablishes autophagic flux and mitochondrial quality control in heart failure.

Autophagy (Most relevant journal in the field) 2017

Campos, J.C., **Queliconi, B.B.**, Bozi, L.H.M., Bechara, L.R.G., Dourado, P.M.M., Andres, A.M., Jannig, P.R., Gomes, K.M.S., Zambelli, V.O., Rocha-Resende, C., Guatimosim, S., Brum, P.C., Mochly-Rosen, D., Gottlieb, R.A., Kowaltowski, A.J., Ferreira, J.C.B.

Exercise is known to be beneficial in several conditions, but with no molecular mechanism explanation. In this work we showed how exercise affects cell signalling and induces autophagy improving heart failure recovery. This paper was a collaboration of 5 different teams, where I worked as a mitochondrial specialist helping to design questions and experiments, interpret results and created new collaborations to solve the complicated problems we faced.

➢ Parkinson's disease-related DJ-1 functions in thiol quality control against aldehyde attack in vitro

Scientific Reports (Nature Group open science journal) 2017

Matsuda N., Kimura M., **Queliconi B.B.**, Kojima W., Mishima M., Takagi K., Koyano F., Yamano K., Mizushima T., Ito Y., Tanaka K.

The enzymatic activity of the Parkinson's related protein DJ-1 was recently described with a lack of relevant physiological substrate, making the protein function unclear. Our work characterized new substrates and helped explain its function. I was responsible for protocol and experimental design, results interpretation and helped to organize the final publication.

Skills and Information

- **Languages –** Portuguese (Proficient), English (Proficient), Japanese (Basic Proficiency)
- **Work Permit Countries –** Brazil, Europe and Japan
- **Sports –** Tennis, Triathlon
- **Project Development** – Budgeting, Project planning, Priorities determination, Team management