



Francesca Briganti

Postdoctoral Research Fellow, Cardiovascular Institute

Bio

BIO

Dr. Francesca Briganti is a molecular biologist interested in understanding the molecular causes of human diseases and in using this knowledge to design targeted therapeutic strategies. She is a postdoctoral scholar in the Mercola lab. She is extending her training in the cardiac physiology, high-throughput screenings, and drug development.

Dr. Briganti received her PhD jointly from the European Molecular Biology Laboratory and the University of Heidelberg. During her PhD she studied new targeted therapeutic approaches for Dilated Cardiomyopathy. She identified a new potential therapeutic strategy that has been published and patented.

Dr. Briganti received her B.Sc and M. Sc in Genetics and Molecular Biology with honors from the University of Rome La Sapienza, Rome, Italy. During a three years internship in Prof. Bozzoni's lab she studied the role of alternative splicing modulation in the pathogenesis and treatment of Duchenne Muscular Dystrophy.

PROFESSIONAL EDUCATION

- Doctor of Philosophy, Ruprecht Karl Universitat Heidelberg (2018)
- Master of Science, Universita Degli Studi Di Roma (2014)
- Bachelor of Science, Universita Degli Studi Di Roma (2012)

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

One gene can lead to the production of many different RNA isoforms via mechanisms such as alternative promoter usage, splicing, and polyadenylation. The functional significance of many of these isoforms, their impact on cell physiology, and their regulation remain mostly controversial. Understanding the functional consequences of transcript heterogeneity will improve our understanding of gene expression regulation, broadening our ability to intervene when mutations that interfere with this regulation cause human disease.

My goal is to become an independent researcher leading an academic lab that focuses on better understanding human tissue-specific post-transcriptional regulation of gene expression and developing mechanism-based therapeutics. My general strategy is to study the function of regulatory genes and their deregulation in human disease. My specific approach is to understand the molecular mechanisms by which disease-causing mutations alter the gene function and lead to human disease. My hypothesis is that a detailed understanding of the relationship between the gene's molecular function and the disease mechanism will allow the development of first-in-class, personalized therapeutic strategies that target the disease mechanisms rather than manage symptoms independently of disease etiology.

Publications

PUBLICATIONS

- **Dysregulated ribonucleoprotein granules promote cardiomyopathy in RBM20 gene-edited pigs (vol 26, pg 1788, 2020) *NATURE MEDICINE***
Schneider, J. W., Oommen, S., Qureshi, M. Y., Goetsch, S. C., Pease, D. R., Sundsbak, R. S., Guo, W., Sun, M., Sun, H., Kuroyanagi, H., Webster, D. A., Coutts, A. W., Holst, et al
2021
- **Human iPSC modeling of heart disease for drug development. *Cell chemical biology***
Hnatiuk, A. P., Briganti, F. n., Staudt, D. W., Mercola, M. n.
2021; 28 (3): 271–82
- **Single-molecule, full-length transcript isoform sequencing reveals disease-associated RNA isoforms in cardiomyocytes. *Nature communications***
Zhu, C., Wu, J., Sun, H., Briganti, F., Meder, B., Wei, W., Steinmetz, L. M.
2021; 12 (1): 4203
- **Metabolic Maturation Media Improve Physiological Function of Human iPSC-Derived Cardiomyocytes. *Cell reports***
Feyen, D. A., McKeithan, W. L., Bruyneel, A. A., Spiering, S. n., Hörmann, L. n., Ulmer, B. n., Zhang, H. n., Briganti, F. n., Schweizer, M. n., Hegyi, B. n., Liao, Z. n., Pölonen, R. P., Ginsburg, et al
2020; 32 (3): 107925
- **Dysregulated ribonucleoprotein granules promote cardiomyopathy in RBM20 gene-edited pigs. *Nature medicine***
Schneider, J. W., Oommen, S. n., Qureshi, M. Y., Goetsch, S. C., Pease, D. R., Sundsbak, R. S., Guo, W. n., Sun, M. n., Sun, H. n., Kuroyanagi, H. n., Webster, D. A., Coutts, A. W., Holst, et al
2020
- **iPSC Modeling of RBM20-Deficient DCM Identifies Upregulation of RBM20 as a Therapeutic Strategy. *Cell reports***
Briganti, F. n., Sun, H. n., Wei, W. n., Wu, J. n., Zhu, C. n., Liss, M. n., Karakikes, I. n., Rego, S. n., Cipriano, A. n., Snyder, M. n., Meder, B. n., Xu, Z. n., Millat, et al
2020; 32 (10): 108117
- **Circ-ZNF609 Is a Circular RNA that Can Be Translated and Functions in Myogenesis *MOLECULAR CELL***
Legnini, I., Di Timoteo, G., Rossi, F., Morlando, M., Briganti, F., Sthandier, O., Fatica, A., Santini, T., Andronache, A., Wade, M., Laneve, P., Rajewsky, N., Bozzoni, et al
2017; 66 (1): 22–+
- **The lack of the Celf2a splicing factor converts a Duchenne genotype into a Becker phenotype *NATURE COMMUNICATIONS***
Martone, J., Briganti, F., Legnini, I., Morlando, M., Picillo, E., Sthandier, O., Politano, L., Bozzoni, I.
2016; 7: 10488