

Stanford



Aly Elezaby

- Postdoctoral Medical Fellow, Cardiovascular Medicine
- Fellow in Medicine

Bio

BIO

Dr Aly Elezaby is a cardiologist fellow at Stanford University School of Medicine and a postdoctoral fellow in the lab of Dr Daria Mochly-Rosen. He attended college at the University of Arizona, where he studied molecular and cellular biology with a research focus on mechanisms of genome instability. He graduated from the MD-PhD program at Boston University, with a dissertation focus on the effects of nutrient excess on mitochondrial function and oxidative stress in the heart. He completed residency training in internal medicine at Stanford as part of the Translational Investigator Program, where he has continued in fellowship training in cardiovascular medicine. His current research focus is on the signaling pathways leading to cardiac ischemia-reperfusion injury, with particular focus on the interaction between contractile proteins and mitochondria. He intends to pursue a career studying cardiac metabolism in cardiomyopathy with a clinical focus on advanced heart failure and transplant cardiology.

PROFESSIONAL EDUCATION

- Postdoctoral Fellowship, Stanford University , Cardiovascular Medicine (2023)
- Fellowship, Stanford University , Cardiovascular Medicine (2022)
- Residency, Stanford University , Internal Medicine (2019)
- MD, PhD, Boston University , Molecular Medicine (2017)
- BS, University of Arizona , Molecular and Cellular Biology (2009)

Publications

PUBLICATIONS

- **A Selective Inhibitor of Cardiac Troponin I Phosphorylation by Delta Protein Kinase C (deltaPKC) as a Treatment for Ischemia-Reperfusion Injury.** *Pharmaceuticals (Basel, Switzerland)*
Qvit, N., Lin, A. J., Elezaby, A., Ostberg, N. P., Campos, J. C., Ferreira, J. C., Mochly-Rosen, D.
2022; 15 (3)
- **Cardiovascular effects of immunosuppression agents.** *Frontiers in cardiovascular medicine*
Elezaby, A., Dexheimer, R., Sallam, K.
2022; 9: 981838
- **Iron Deficiency as a Potential Modulator of Subclinical Deficiencies in Cardiac Performance and Exercise Capacity.** *Journal of cardiac failure*
Elezaby, A., Parikh, V. N., Nayor, M.
2021; 27 (7): 822-824
- **ABCB10 deletion in cardiomyocytes leads to mitochondrial dysfunction and early death**
Chambers, J., Elezaby, A., Croteau, D., Sverdlov, A., Liesa, M., Shirihai, O., Luptak, I., Pimentel, D., Siwik, D., Colucci, W.
ELSEVIER SCIENCE INC.2018: S22

- **Mitochondrial Reactive Oxygen Species Mediate Cardiac Structural, Functional, and Mitochondrial Consequences of Diet-Induced Metabolic Heart Disease.** *Journal of the American Heart Association*
Sverdlov, A. L., Elezaby, A. n., Qin, F. n., Behring, J. B., Luptak, I. n., Calamaras, T. D., Siwik, D. A., Miller, E. J., Liesa, M. n., Shirihai, O. S., Pimentel, D. R., Cohen, R. A., Bachschmid, et al
2016; 5 (1)
- **Mitochondrial remodeling in mice with cardiomyocyte-specific lipid overload.** *Journal of molecular and cellular cardiology*
Elezaby, A. n., Sverdlov, A. L., Tu, V. H., Soni, K. n., Luptak, I. n., Qin, F. n., Liesa, M. n., Shirihai, O. S., Rimer, J. n., Schaffer, J. E., Colucci, W. S., Miller, E. J.
2015; 79: 275–83
- **Partial Liver Kinase B1 (LKB1) Deficiency Promotes Diastolic Dysfunction, De Novo Systolic Dysfunction, Apoptosis, and Mitochondrial Dysfunction With Dietary Metabolic Challenge.** *Journal of the American Heart Association*
Miller, E. J., Calamaras, T. n., Elezaby, A. n., Sverdlov, A. n., Qin, F. n., Luptak, I. n., Wang, K. n., Sun, X. n., Vijay, A. n., Croteau, D. n., Bachschmid, M. n., Cohen, R. A., Walsh, et al
2015; 5 (1)
- **High fat, high sucrose diet causes cardiac mitochondrial dysfunction due in part to oxidative post-translational modification of mitochondrial complex II.** *Journal of molecular and cellular cardiology*
Sverdlov, A. L., Elezaby, A. n., Behring, J. B., Bachschmid, M. M., Luptak, I. n., Tu, V. H., Siwik, D. A., Miller, E. J., Liesa, M. n., Shirihai, O. S., Pimentel, D. R., Cohen, R. A., Colucci, et al
2015; 78: 165–73
- **Overexpression of Catalase Diminishes Oxidative Cysteine Modifications of Cardiac Proteins.** *PloS one*
Yao, C. n., Behring, J. B., Shao, D. n., Sverdlov, A. L., Whelan, S. A., Elezaby, A. n., Yin, X. n., Siwik, D. A., Seta, F. n., Costello, C. E., Cohen, R. A., Matsui, R. n., Colucci, et al
2015; 10 (12): e0144025
- **Impairment of the PPAR α /PGC1 α Axis Compromises Mitochondrial Biogenesis and Function in Hearts With Cardiomyocyte-Specific Fatty Acid Transport Protein 1 (FATP1) Overexpression**
Elezaby, A., Sverdlov, A., Tu, V., Soni, K., Liesa, M., Liesa, M., Shirihai, O., Colucci, W. S., Miller, E. J.
LIPPINCOTT WILLIAMS & WILKINS.2013
- **Cardiac-Specific Fatty Acid Transport Protein 1 (FATP1) Overexpression Causes Decreased Mitochondrial Respiration, Increased Oxidative Stress and Activation of AMPK**
Elezaby, A., Miller, E. J., Qi, F., Liesa, M., Shirihai, O. S., Colucci, W. S.
ELSEVIER SCIENCE INC.2012: S159
- **Fusion of nearby inverted repeats by a replication-based mechanism leads to formation of dicentric and acentric chromosomes that cause genome instability in budding yeast.** *Genes & development*
Paek, A. L., Kaochar, S. n., Jones, H. n., Elezaby, A. n., Shanks, L. n., Weinert, T. n.
2009; 23 (24): 2861–75