Kunal Sikder, PhD

Contact: +91-6289544240
Email: kunalsikder@gmail.com
ORCID No.: 0000-0002-4235-0936
University Webpage:
http://narendrapur.rkmvu.ac.in/laboratory-of-cardiology-and-metabolic-disorder/
http://sy.rkmvu.ac.in/dr-kunal-sikder/
LinkedIn Page: https://www.linkedin.com/in/kunal-sikder-9987b196/

Present Position: Assistant Professor, Dept. of Biomedical Science and Technology and Dept. of Sports Science and Yoga, Ramakrishna Mission Vivekananda Educational and Research Institute (RKMVERI), West Bengal, India

Professional Experience (Post PhD- 10 years):

Assistant Professor: June 2019- Present
(RKMVERI, West Bengal, India)

Post-Doctoral Scientist: March 2015- May 2019
(Dept. of Medicine, Thomas Jefferson University, Philadelphia, USA)

Assistant Professor: June 2012- February 2015
(Tripura Institute of Paramedical Sciences, Agartala, India)

Education:

Ph.D. (Physiology, University of Calcutta): Awarded November 2013*

M.Sc. (Physiology, University of Calcutta): July 2007 (1st class 3rd, 68%)

B.Sc. (Physiology, University of Calcutta): April 2005 (1st class, 62.5%)

* **Thesis submitted on May 2012:** Title of thesis- Antidyslipidemic and Antioxidative effect of some major active compounds of *Moringa oleifera* (Sajna) leaf extract

Journal Publications (Reverse chronological order):

1. Dastidar R and Sikder K. Diagnostic reliability of serum active vitamin B12 (holo-transcobalamin) in true evaluation of vitamin B12 deficiency: Relevance in current perspective. BMC Research Notes (accepted for publication, 28 September 2022). **(*corresponding author)**

2. Chakraborty A, Halder S, Kishore P, Saha D, Saha S, Sikder K, Basu A. The structure-function analysis of Obg like GTPase proteins along the evolutionary tree from bacteria to humans. Genes to Cells. 2022; Jul;27(7):469-481.
3. Talukdar D, Haldar AK, Dastidar R, Kumar S, Lodha M, **Sikder K*** et al. Inflammation-related Biomarkers Are Associated With Severity Of Idiopathic Dilated Cardiomyopathy (IDCM): A Brief Report On Patients Of West Bengal, India. J Cardiovasc Dis Res. 2022;13(1):1693-1701. **(*corresponding author)**
4. Chakraborty A, Halder S, Kishore P, Saha D, **Sikder K** and Basu A. The structure-function analysis of Obg-like GTPase proteins along the evolutionary tree from bacteria to humans. Genes Cells. 2022;27(7):469-481.
5. Kolpakov, A.K*, **Sikder, K.***, Sarkar, A., Chaki, S., Shukla, S.K., Guo, X., Qi, Z., Barbery, C., Sabri, A. & Rafiq, K. Inflammatory serine proteases play a critical role in the early pathogenesis of diabetic cardiomyopathy. Cellular Physiology and Biochemistry. 2019;53(6):982-998. doi:10.33594/000000190 **(*Combined first authorship).**
6. **Sikder, K**., Shukla, S.K, Patel, N., Singh, H. & Rafiq, K. High fat diet upregulates fatty acid oxidation and ketogenesis via intervention of PPAR-γ. Cellular Physiology and Biochemistry. 2018;48(3):1317-1331. doi: 10.1159/000492091
7. Shukla, S.K, **Sikder, K**., Sarkar, A., Addya, S. & Rafiq, K. Molecular network, pathway, and functional analysis of time-dependent gene changes related to cathepsin G exposure in neonatal rat cardiomyocytes. Gene. 2018 Sep 10;671:58-66. doi: 10.1016/j.gene.2018.05.110. Epub 2018 May 31
8. Shukla, S., K., Liu, W., **Sikder, K**., Addya, S., Sarkar, A., Wei, Y., & Rafiq, K. HMGCS2 is a key ketogenic enzyme potentially involved in type 1 diabetes with high cardiovascular risk. Scientific Reports. 2017 Jul 4;7(1):4590. doi: 10.1038/s41598-017-04469-z
9. Khan, A., **Sikder, K**., Dey, S. *et al*. Gossypetin ameliorates ionizing radiationinduced oxidative stress in mice liver-a molecular approach. Free Radical Research. 2015 Oct;49(10):1173-86. doi: 10.3109/10715762.2015.1053878. Epub 2015 Aug 14
10. **Sikder, K.**, Kesh, S. B., Das, N., Manna, K. & Dey, S. The high antioxidative power of quercetin (aglycone flavonoid) and its glycone (rutin) avert high cholesterol diet induced hepatotoxicity and inflammation in Swiss albino mice. Food & Function. 014 Jun;5(6):1294-303. doi: 10.1039/c3fo60526d. Epub 2014 Apr 17
11. **Sikder, K.**, Das, N., Kesh, S. B. & Dey, S. Quercetin and beta-sitosterol prevent high fat diet induced dyslipidemia and hepatotoxicity in Swiss albino mice. Indian Journal of Experimental Biology. 2014 Jan;52(1):60-66
12. Das, N., **Sikder, K.**, Dey, S. *et al*. Quercetin alleviates inflammation after short-term treatment in high-fat-fed mice. Food & Function. 2013 Jun;4(6):889-98. doi: 10.1039/c3fo30241e. Epub 2013 May 3
13. Kesh, S. B., **Sikder, K.**, Dey, S. *et al*. Promising role of ferulic acid, atorvastatin and their combination in ameliorating high fat diet-induced stress in mice. Life sciences. 2013 May 20;92(17-19):938-49. doi: 10.1016/j.lfs.2013.03.015. Epub 2013 Apr 6
14. **Sikder, K.**, Sinha, M., Das, N., Das, D.K., Datta, S. & Dey, S. Moringa oleifera leaf extract prevents in vitro oxidative DNA damage. Asian journal of Pharmaceutical and Clinical Research. 2013;6,92-96

15. Das, N., **Sikder, K**., Ghosh, S., Fromenty, B. & Dey, S. Moringa oleifera Lam. leaf extract prevents early liver injury and restores antioxidant status in mice fed with high-fat diet. Indian Journal of Experimental Biology. 2012 Jun;50(6):404-412

***Total citation- 353**

h-index-9 (As on 19 September 2022, Scopus)

Major Conference Publications (Last 5 years):

1. **Kunal Sikder**, Elizabeth Phillips, Zhiju Zhong, Nadan Wang, Kenneth B. Margulies and Jason C. Choi. Celltype-Specific Functions in Dilated Cardiomyopathy Caused by the LMNA Gene Mutation. American heart association meeting, Philadelphia, USA. Circulation Research , 2019. Vol.125, Abstract no.730
2. **Kunal Sikder**, Amrita Sarkar, Sanket Kumar Shukla, Aimee Abbott, Domenica Carrier, Carlos Barbery, Richard Pestell, and Khadija Rafiq. Inflammatory Serine Protease Inhibition Attenuates Myocyte apoptosis and cardiac dysfunction via intervention of peroxisome proliferator-activated receptor gamma-induced lipotoxicity and inflammation in high fat diet-induced diabetic cardiomyopathy. American heart association meeting, New Orleans, USA. Circulation, 2018. Vol.134, Abstract no.17161
3. Sanket Shukla, **Kunal Sikder**, Amrita Sarkar, Weijing Liu and Khadija Rafiq. Activation of the E3 ligase Cbl by neutrophil cathepsin G impairs CXC chemokine receptor 4 signaling in cardiomyocyte degeneration. Conference on experimental biology, Chicago. FASEB journal, 2017. Abstract no. 614.19
4. **Kunal Sikder**, Amrita Sarkar, Sanket Kumar Shukla, Aimee Abbott, Domenica Carrier, Carlos Barbery, Richard Pestell, and Khadija Rafiq ISP Inhibition Attenuates myocyte apoptosis and cardiac dysfunction via intervention of PPAR-γ induced lipotoxicity and inflammation in HFD-induced DCM. 11th annual meeting for Post-doctoral researchers at Thomas Jefferson University, Philadelphia, USA May 2017
5. Sanket Shukla, **Kunal Sikder**, Amrita Sarkar, Weijing Liu, Aimee Abbott, Carlos Barbery, Christine Pham and Khadija Rafiq. Effect of ISP inhibition on CXCR4 expression during the development of DCM. American Physiological Society meeting, Westminster, Colorado, USA, 2016. Abstract 4.31

Research Interest:

1. Diabetic cardiomyopathy (DCM), Cardiac inflammation:

Diabetic cardiomyopathy (DCM) is characterized by structural and functional alterations that can lead to heart failure. Several mechanisms are known to be involved in the pathogenesis of DCM, however, only a few studies involved in

elucidating the molecular mechanism that links inflammation to DCM. We found that in diabetic heart there is surplus release of inflammatory serine proteases (ISP) from infiltrating neutrophils following early inflammation. By blocking dipeptidyl peptidase I (DPPI), an enzyme involved in the maturation of major ISPs, significantly decreased activation of ISPs, myocyte apoptosis, fibrosis and improve cardiac function in DPPI-KO mice. This also showed a decrease in overall systemic inflammatory status manifested by decreased production of pro-inflammatory cytokines like TNF-α, IL-1β and IL-6.

2. Role of cardiac autoimmunity in idiopathic dilated cardiomyopathies:

Dilated cardiomyopathy (DCM) is the most lethal form of all CVDs that primarily affect the left ventricle (LV). There is a major form of DCM present without any identified etiology, known as idiopathic dilated cardiomyopathy (IDCM). Based on evidences that suggest a faulty central immune-tolerance leads to persistent T-cell mediated autoimmune responses in myocarditis, all of the IDCM cases may be connected with an autoimmune origin. People who develop autoimmunity against cardiac proteins like alfa-MHC, Tni, Beta-1 receptor etc due to presence of certain HLA alleles are at high risk of developing IDCM irrespective of their lifestyle and age.

3. PPAR-γ mediated upsurge in mitochondrial oxidation and ketogenesis in diabetic heart:

Systemic hyperlipidemia and intracellular lipid accumulation induced by chronic high fat diet (HFD) leads to enhanced fatty acid oxidation (FAO) and ketogenesis. We found that increased PPAR-γ expression in diabetic heart by chronic hyperlipidemia is responsible for cardiac dysfunction via upregulation of mitochondrial ketogenic enzymes HMGCS2, BDH1 and PDK4. Targeting PPAR-γ and its downstream mitochondrial enzymes will provide novel strategies in preventing metabolic and myocardial dysfunction in diabetes mellitus.

4. Cell type-specific functions in dilated cardiomyopathy caused by the *LMNA* gene mutation:

Mutations in the *LMNA* gene (lamin A/C) cause a diverse group of human diseases termed laminopathies. The most prevalent laminopathy is dilated cardiomyopathy (*LMNA* cardiomyopathy) characterized by variable onset of fibrosis/pathological remodeling that always progresses to heart failure. Despite recent progress, how cell type-specific effects of *LMNA* mutations are integrated at the tissue level to engender complex pathologies in a heterocellular organ such as the heart are not well understood. Our results suggest lamin A/C-depleted cardiac fibroblasts (CF) mediate a brake on cardiomyopathy development and interactions between CFs and cardiac myocytes (CMs) are important determinants of the rate of progression and the severity of *LMNA* cardiomyopathy. Therefore, strategies targeting lamin A/C function in CFs may hold therapeutic potential for patients with *LMNA* cardiomyopathy as well as for other forms of cardiomyopathy in which fibrosis is integral to disease pathogenesis.

Professional Memberships:

1. Reviewer and editorial board member of 'Food and Nutrition Research' since 2015 (Taylor & Francis).
2. Reviewer of 'Life Sciences' since 2019 (Elsevier)
3. Former Member of American Heart Association (AHA)
4. Former Member of American Physiological Society (APS) since 2016

Research Expertise:

- Extensive research experience with small animals: breeding, genotyping, dissection, tissue processing for histopathology etc
- Specialization with primary rat and mouse cardiomyocytes, cardiac fibroblasts. Expertise in co-culture in conventional and 3D culture. Culture and differentiation of human stem cells (h-iPSC)
- *In vitro* genetic manipulation by means of viral transduction, transfection and molecular cloning
- Immunohistochemistry, immunocytochemistry, confocal microscopy, live cell microscopy and bright field microscopy.
- Nucleic acid and protein works, specialization in designing PCR and cloning primers by means of NCBI blast, gene runner and primer3 software

Teaching Statement:

Over 6 years teaching experience as Assistant Professor at undergraduate, post-graduate and pre-clinical levels enable me to understand the student psyche very well. I believe in mixing up my teaching with materials and practical implications. In each class, at the very least, I try to engage students in variety of ways, with visuals, sounds, and words. Each class I focus on a particular goal and each discussion is the result of careful preparation. I do not want my students to leave class with their heads spinning from over burden or something. I try to engage them with the topic and generate love and interest for the subject. I also try to end each class with summation of the topic taught and begin the next class by recapitulating the previous topic to ease the students in.