

CURRICULUM VITAE

Sagartirtha Sarkar

Professor

Elected Fellow: National Academy of Sciences, India.

Elected Fellow: West Bengal Academy of Science and Technology, India.

Elected Member: Guha Research Conference (GRC).

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Academic Qualifications:

1990: M.Sc.; University of Calcutta, Calcutta, India.

1998: Ph.D.; University of Calcutta, Calcutta, India

Research Experience:

1997: Postdoctoral fellowship in National Institute of Cholera and Enteric Diseases, Kolkata.

1998-2004: Postdoctoral fellow, Cleveland Clinic Foundation, Cleveland, Ohio, USA.

2004 - 2008: Lecturer, Genetics and Molecular Cardiology Laboratory, Dept. of Zoology, University of Calcutta, India.

2008 - 2011: Reader, Genetics and Molecular Cardiology Laboratory, Dept. of Zoology, University of Calcutta, India.

2008: Visiting Scientist, Department of Medicine, University of California, San Diego, USA.

2011- 2014: Associate Professor, Dept. of Zoology, University of Calcutta, India.

2014- till date: Professor, Dept. of Zoology, University of Calcutta, India.

Brief statement about area of research interest:

Prof. Sarkar's lab is one of the few laboratories in India which studies molecular mechanism of cardiac diseases (pathological hypertrophy and MI) with a view to understand disease pathogenesis and develop therapeutics. Apart from identifying major signaling cascades with novel therapeutic targets at the gene and protein levels and their cellular crosstalk, his work has successfully developed a nano-particle based targeted drug delivery system. This delivery system can successfully deliver the therapeutic molecules, siRNAs and DNA vectors directly to the diseased cardio-myocytes in vivo

resulting in improved efficacy of therapeutics with low bystander cytotoxic effects. This unique delivery system has the potential to change the therapeutic approach of diseased myocardium in near future. In addition, his laboratory has developed a method to regulate expression of protein of interest in cardio-myocytes in animal models. This has implications in assessing the functional role of that protein in the preclinical scenario. Prof. Sarkar's group is working towards establishing myocardial tissue engineering, with an aim to induce terminally differentiated resident myocyte cells from diseased heart to develop self-regeneration capacity.

Current research programs include:

- Mechanism of collagen upregulation during transition of hypertrophy to heart failure via cellular crosstalk
- Study of myocyte death, regeneration and migration of stem cells during heart failure
- Identification of candidate genes for etiologically different cardiac disease forms by analyzing the cardiac proteome during cardiac remodeling
- Development of therapeutic small molecules and targeted tissue engineering of diseased myocardium

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Selected Publications in last ten years:

1. Chatterjee, A., Mir, S.A., Dutta, D., Mitra, A., Pathak, K. and **Sarkar, S.**, 2011. Analysis of p53 and NF- κ B signaling in modulating the cardiomyocyte fate during hypertrophy. *Journal of Cellular Physiology*, 226(10), pp.2543-2554.
2. Mir, S.A., Chatterjee, A., Mitra, A., Pathak, K., Mahata, S.K. and **Sarkar, S.**, 2012. Inhibition of signal transducer and activator of transcription 3 (STAT3) attenuates interleukin-6 (IL-6)-induced collagen synthesis and resultant hypertrophy in rat heart. *Journal of Biological Chemistry*, 287(4), pp.2666-2677.
3. Mitra, A., Basak, T., Datta, K., Naskar, S., Sengupta, S. and **Sarkar, S.**, 2013. Role of α -crystallin B as a regulatory switch in modulating cardiomyocyte apoptosis by mitochondria or endoplasmic reticulum during cardiac hypertrophy and myocardial infarction. *Cell death & disease*, 4(4), p.e582.
4. Mitra, A., Ray, A., Datta, R., Sengupta, S. and **Sarkar, S.**, 2014. Cardioprotective Role of P38 MAPK During Myocardial Infarction Via Parallel Activation of α -Crystallin B and Nrf2. *Journal of Cellular Physiology*, 229(9), pp.1272-1282.
5. Nayak, M.K., Agrawal, A.S., Bose, S., Naskar, S., Bhowmick, R., Chakrabarti, S., **Sarkar, S.** and Chawla-Sarkar, M. 2014. Antiviral activity of baicalin against influenza virus H1N1-pdm09 is due to modulation of NS1-mediated cellular innate immune responses. *Journal of Antimicrobial Chemotherapy*, 69(5), pp.1298-1310.

6. Naskar, S., Datta, K., Mitra, A., Pathak, K., Datta, R., Bansal, T. and **Sarkar, S.**, 2014. Differential and conditional activation of PKC-isoforms dictates cardiac adaptation during physiological to pathological hypertrophy. *PloS one*, 9(8), p.e104711.
7. Mitra, A., Basak, T., Ahmad, S., Datta, K., Datta, R., Sengupta, S. and **Sarkar, S.**, 2015. Comparative proteome profiling during cardiac hypertrophy and myocardial infarction reveals altered glucose oxidation by differential activation of pyruvate dehydrogenase E1 component subunit β . *Journal of Molecular Biology*, 427(11), pp.2104-2120.
8. Ganguly, S., Mitra, A. and **Sarkar, S.**, 2014. Role of α -crystallin B in regulation of stress induced cardiomyocyte apoptosis. *Cardiovascular & Hematological Agents in Medicinal Chemistry (Formerly Current Medicinal Chemistry-Cardiovascular & Hematological Agents)*, 12(2), pp.60-65.
9. Chakrabarti, P., Rana, S., **Sarkar, S.**, Smith, B. and Basu, P., 2015. Pesticide-induced oxidative stress in laboratory and field populations of native honey bees along intensive agricultural landscapes in two Eastern Indian states. *Apidologie*, 46(1), pp.107-129.
10. Rana, S., Datta, K., Reddy, T.L., Chatterjee, E., Sen, P., Pal-Bhadra, M., Bhadra, U., Pramanik, A., Pramanik, P., Chawla-Sarkar, M. and **Sarkar, S.**, 2015. A spatio-temporal cardiomyocyte targeted vector system for efficient delivery of therapeutic payloads to regress cardiac hypertrophy abating bystander effect. *Journal of Controlled Release*, 200, pp.167-178.
11. Chakrabarti, P., Rana, S., Bandopadhyay, S., Naik, D.G., **Sarkar, S.** and Basu, P., 2015. Field populations of native Indian honey bees from pesticide intensive agricultural landscape show signs of impaired olfaction. *Scientific Reports*, 5, p.12504.
12. Datta, R., Bansal, T., Rana, S., Datta, K., Chattopadhyay, S., Chawla-Sarkar, M. and **Sarkar, S.**, 2015. Hsp90/Cdc37 assembly modulates TGF β receptor-II to act as a profibrotic regulator of TGF β signaling during cardiac hypertrophy. *Cellular Signalling*, 27(12), pp.2410-2424.
13. Datta, K. and **Sarkar, S.**, 2015. Proteomics as a Tool to Decipher Novel Mechanistic Candidates in Cardiac Pathophysiology. *Journal of Bioinformatics and Proteomics Review*. 1(3): 1-5.
14. Ganguly, S., Chatterjee, E. and **Sarkar, S.**, 2015. Targeting Insulin-Like Growth Factor-I receptor signaling pathways improve compromised function during cardiac hypertrophy. *Journal of Integrative Cardiology*, 1(6): 225-228.
15. Ray, A., Rana, S., Banerjee, D., Mitra, A., Datta, R., Naskar, S. and **Sarkar, S.**, 2016. Improved bioavailability of targeted curcumin delivery efficiently regressed cardiac hypertrophy by modulating apoptotic load within cardiac microenvironment. *Toxicology and Applied Pharmacology*, 290, 54-65.

16. Ganguly, M., **Sarkar, S.**, Ghosh, P., Sarkar, A., Alam, J., Karmakar, B.C., De, R., Saha, D.R. and Mukhopadhyay, A.K., 2016. Helicobacter pylori plasticity region genes are associated with the gastroduodenal diseases manifestation in India. *Gut Pathogens*, 8(1), 1-10.
17. Datta, K., Basak, T., Varshney, S., Sengupta, S. and **Sarkar, S.**, 2017. Quantitative proteomic changes during post myocardial infarction remodeling reveals altered cardiac metabolism and Desmin aggregation in the infarct region. *Journal of Proteomics*, 152, pp.283-299.
18. Datta, R., Bansal, T., Rana, S., Datta, K., Chaudhuri, R.D., Chawla-Sarkar, M. and **Sarkar, S.**, 2017. Myocyte-derived Hsp90 modulates collagen upregulation via biphasic activation of STAT-3 in fibroblasts during cardiac hypertrophy. *Molecular and Cellular Biology*, 37(6), pp.e00611-16.
19. Bansal, T., Chatterjee, E., Singh, J., Ray, A., Kundu, B., Thankamani, V., Sengupta, S. and **Sarkar, S.**, 2017. Arjunolic acid, a peroxisome proliferator-activated receptor α agonist, regresses cardiac fibrosis by inhibiting non-canonical TGF- β signaling. *Journal of Biological Chemistry*, 292(40), pp.16440-16462.
20. Mitra, A., Datta, R., Rana, S. and **Sarkar, S.**, 2018. Modulation of NF κ B1/p50 by ROS leads to impaired ATP production during MI compared to cardiac hypertrophy. *Journal of Cellular Biochemistry*, 119(2), 1575-1590.
21. Chakrabarti, P., Sarkar, S. and Basu, P., 2018. Field populations of wild *Apis cerana* honey bees exhibit increased genetic diversity under pesticide stress along an agricultural intensification gradient in eastern India. *Journal of Insect Science*, 18(3), p.3.
22. Rana, S., Datta, R., Chaudhuri, R.D., Chatterjee, E., Chawla-Sarkar, M. and **Sarkar, S.**, 2019. Nanotized PPAR α Overexpression Targeted to Hypertrophied Myocardium Improves Cardiac Function by Attenuating the p53-GSK3 β -Mediated Mitochondrial Death Pathway. *Antioxidants & Redox signaling*, 30(5), 713-732.
23. Mittal, A., Rana, S., Sharma, R., Kumar, A., Prasad, R., Raut, S.K., **Sarkar, S.**, Saikia, U.N., Bahl, A., Dhandapany, P.S. and Khullar, M., 2019. Myocardial ablation in a cardiac-renal rat model. *Scientific Reports*, 9(1), p.5872.
24. Chakrabarti, P., **Sarkar, S.** and Basu, P., 2019. Pesticide induced visual abnormalities in Asian honey bees (*Apis cerana* L.) in intensive agricultural landscapes. *Chemosphere*, 230, 51-58.
25. Chatterjee, E., Chaudhuri, R.D. and **Sarkar, S.**, 2019. Cardiomyocyte targeted overexpression of IGF1 during detraining restores compromised cardiac condition via mTORC2 mediated switching of PKC δ to PKC α . *Biochimica et Biophysica Acta (BBA)- Molecular Basis of Disease*, 1865(10), 2736-2752.

26. Banerjee, D., Datta Chaudhuri, R., Niyogi, S., Roy Chowdhuri, S., Poddar Sarkar, M., Chatterjee, R., Chakrabarti, P., **Sarkar, S.** 2020. Metabolic impairment in response to early induction of C/EBP β leads to compromised cardiac function during pathological hypertrophy. *J Mol Cell Cardiol.* 139:148-163.
27. Datta Chaudhuri R, Banerjee D, Banik A, **Sarkar S.** 2020. Severity and duration of hypoxic stress differentially regulates HIF-1 α -mediated cardiomyocyte apoptotic signaling milieu during myocardial infarction. *Arch Biochem Biophys.* Sep 15; 690:108430.

Book:

1. Guha, S.K., **Sarkar Sagartirtha.** 2012. Growth Factors vis-a-vis healing of bones. LAP Lambert Academic Publishing; ISBN 978-3-8484-3152-6.

Book Chapter:

1. Ratul Datta Chaudhuri, Santanu Rana, Kaberi Datta and **Sagartirtha Sarkar.** 2019. Key Cellular Effectors in ROS-Mediated Cardiac Diseases. pp 151-195. Modulation of Oxidative Stress in Heart Disease, <https://doi.org/10.1007/978-981-13-8946-7>. Springer Nature Singapore Pte Ltd.. ISBN 978-981-13-8945-0

Patents:

“A nanovehicle for targeted and controlled delivery of a therapeutic molecule” - **Indian Patent No.: 335668** dated July 11, 2013.

Memberships of Societies:

- 1) Life member: Zoological Society, Kolkata, India.
- 2) Life member: Society of Cell Biologists, India
- 3) Life member: Society of Biological Chemists, India
- 4) Life member: Indian Science Congress Association, India.
- 5) Member: British Society of Cardiovascular Research, UK.
- 6) Life member: Indian Society for Translational Research
- 7) Life Member: West Bengal Academy of Science and Technology.
- 8) Member: Guha Research Conference

Research Collaborations:

1. Dr. Shantanu Sengupta, Institute of Genomics and Integrative Biology, New Delhi, India
2. Dr. Raja Mazumder, University of Georgetown Medical Center, Washington DC, USA.
3. Prof. Derek Hausenloy, Cardiovascular Disorder Program, Duke-NUS Medical School, Singapore
4. Prof. S.K.Mahata, Department of Medicine, University of California, San Diego, USA.
5. Prof. N. Maulik, Department of Surgery, UCONN Health. USA.