Current Activities Include

• Characterizing immune cell phenotypes and functions in decompensated cirrhotic patients with and without infection

• Investigating how SBP related bacteria influence macrophage, neutrophil and T cell responses and contribute to immune evasion

• Developing a preclinical animal model of SBP to study infection dynamics and host responses

• Performing proteomic analysis of ascitic fluid samples from SBP and non SBP patients to identify pathways and biomarkers linked to infection and immune dysfunction

• Examining mitochondrial adaptations and oxidative stress and their contribution to fibrosis and liver failure

• Investigating exosomal microRNAs from injured hepatocytes and their role in hepatic stellate cell activation, immune modulation and fibrotic progression during drug induced liver injury (DILI)

• Identifying cell free DNA and miRNA signatures as biomarkers for liver disease progression and antitubercular drug resistance

Research

Our lab focuses on understanding the immune landscape of decompensated cirrhotic patients, on uncovering the cellular and molecular mechanisms that maintain liver homeostasis but become disrupted in advanced liver disease. We study how decompensated cirrhosis drives immune dysfunction, shaping responses to infection in conditions such as spontaneous bacterial peritonitis (SBP). A key area of interest is how immune cells including macrophages, neutrophils, and T cells respond to SBP causing bacteria, undergoing phenotypic and functional changes that allow pathogens to evade host defense. These investigations use human samples and animal models to bridge clinical observations with mechanistic insights. We also explore how mitochondrial alterations and oxidative stress contribute to cirrhosis progression and its complications.

In parallel, we investigate drug induced liver injury (DILI), focusing on exosomal microRNAs released from injured hepatocytes and their roles in activating hepatic stellate cells, modulating immune responses, and driving fibrotic progression. We are also identifying biomarkers such as cell free DNA and miRNA signatures for early detection and risk prediction in liver diseases, including antitubercular drug resistance. Together, these efforts aim to connect fundamental cellular processes with clinical outcomes, ultimately informing improved diagnostic and therapeutic strategies for liver disease.

Publications:

- Santra A, Bishnu D, Santra S, Ghatak S, Mukherjee PS, Dhali GK, Chowdhury A. Arsenic-Induced Injury of Mouse Hepatocytes through Lysosome and Mitochondria: An In Vitro Study. Int J Hepatol. 2022 Sep 8;2022:1546297. doi: 10.1155/2022/1546297. PMID: 36117518; PMCID: PMC9477643.
- Santra S, BishnuD,Dhali GK, Santra A, Chowdhury A. Expression of type I collagen in response to Isoniazid exposure is indirect and is facilitated by collateral induction of Cytochrome P450 2E1: An in-vitro study. Plos One 2020; 15(7): e0236992. Published online 2020 Jul 31. doi: 10.1371/journal.pone.0236992; PMCID: 7394448
- Roy S, Santra S, Das A , Dixith S, Sinha M, Ghatak S, Ghosh N, Banerjee P, Khanna S, Mathew-Steiner S, Blackstone B, Powell H, Bergdall V, Wozniak D J, Sen C. Staphylococcus aureus Biofilm infection Compromises Wound Healing by Causing Deficiencies in Granulation Tissue Collagen. Annals of surgery AnnSurg 2020;271:1174–1185)
- BISWAS A*, SANTRA S*, DhaliGK,Chowdhury A, Santra A. Isoniazid and Rifampicin, produce hepatic fibrosis through a oxidative stress dependent mechanism. Int J Hepatolonnals of Hepatolo 2020; 2020: 6987295. Published online 2020 Apr 23. doi: 10.1155/2020/6987295 PMCID: 7195633 (*Authors contributed equally)

- Chakraborty BC, Bishnu D, Santra S, Dhali GK, Chowdhury A, Santra A. Pentoxifilline Prevents Oxidative Stress Mediated Acute Liver Injury in Carbon Tetrachloride Treated Mice. International Journal Of Clinical Medicine Research 2018;5(2):20-26
- Chakraborty BC, Bishnu D, Santra S, Dhali GK, Chowdhury A, Santra A. Inhibition Of NADPH Oxidase Attenuates Inflammation and Cellular Senescence At Early Phase of Liver Injury in Mice. International Journal of Current Advanced Research. 2018;7(3):11174-11182
- BISHNU D*, SANTRA S*, Dhali GK, Chowdhury A, Santra A. Induction of Hemeoxygenase 1 Protects Hepatocytes from Isoniazid-Rifampicin Induced Cell Death: an invitro study. Tropical Gastroenterology 2016;37(1): 27-36

Book Chapters

- Chaffee S, Das A, SantraS, Roy S.Diabetic Wound Inflammation. In (D Bagchi and S Nair eds) Nutritional and Therapeutic Interventions for Diabetes and Metabolic Syndrome (Academic Press, Elsevier) 2018; p:269-278.
- Santra S, Rawat A, Pattarayan D, Roy S, Chronic infection and inflammation: Hallmarks of diabetic foot ulcers. In (D. Bagchi; A Das and S Roy eds) Wound Healing, Tissue Repair and Regeneration in Diabetes (Academic Press, Elsevier) 2019; p 39-44.
- Madeshiya A, Banerjee P, Santra S, Ghosh N, Karmakar S, Bagchi D, Roy S. Role of MetalsandMetaloids in Redox Biology. In (Bagchi D, BagchiM eds) Metal Toxicology Handbook. 2020 in Press.