SARAT CENTENARY COLLEGE, DHANIAKHALI, HOOGHLY, WB
TEACHER PROFILE

Name: Dr. Moumita Adak	
Designation: ASSISTANT PROFESSOR STAGE II	
Department: ZOOLOGY	
Academic Qualifications: MSC, PHD	
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DATE OF JOINING	12.05.2026
SPECIALIZATION	FISH BIOLOGY
TEACHING INTEREST	BIOCHEMISTRY, IMMUNOLOGY, BIOTECHNOLOGY, CELL & MOLECULAR BIOLOGY
TEACHING EXPERIENCE	6.5 YEARS both UG & PG
AWARD/ FELLOWSHIP	UGC NET JRF , 2011 ; RET (A national level test)
MEMBERSHIP	

RESEARCH INTEREST: CELL & MOLECULAR BIOLOGY, IMMUNOLOGY, BIOCHEMISTRY

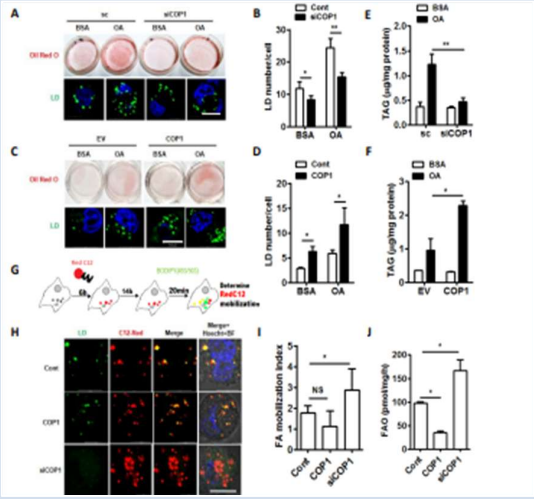
RESEARCH EXPERIENCE: 8 YEARS

RESEARCH GATE ACCOUNT URL: https://www.researchgate.net/profile/Moumita-Adak

SEMINAR/ WORKSHOP PARTICIPATION	PRESENTED PAPER		ATTENDED		CHAIRED SESSION	
	NATIONAL	INTERNATIONAL	NATIONAL	INTERNATIONAL	NATIONAL	INTERNATIONAL
	01		02			

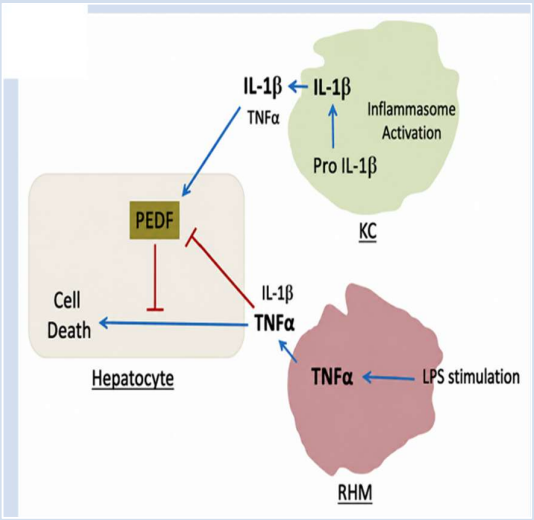
PUBLICATIONS	JOURNAL ARTICLES	BOOK/BOOK CHAPTERS
	8 REPUTED JOURNALS 3 BOOK CHAPTER	1.Ex Vivo Dual-Hit Method for Inflammasome Activation in Liver Chapter 20 (Springer Nature) 2455:255-265 2. Inflammasome Activation in Kupffer Cells Confers a Protective Response in NASH through PEDF Expression 3. Expression study of hepatic pigment epithelium-derived factor (PEDF) in non-alcoholic steatohepatitis (NASH)

SELECTED PUBLICATIONS:



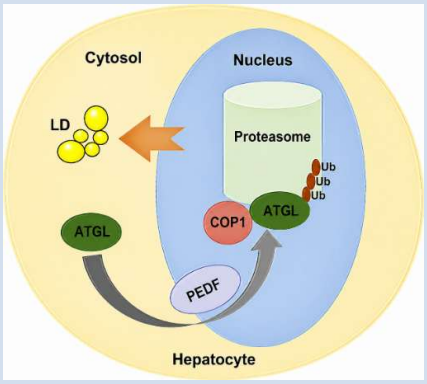
1. Ghosh, M., Niyogi, S., Bhattacharyya, M., Adak, M., Nayak, D. K., Chakrabarti, S., & Chakrabarti, P. (2016). Ubiquitin Ligase COP1 Controls Hepatic Fat Metabolism by Targeting ATGL for Degradation. *Diabetes*, *65*(12), 3561–3572. https://doi.org/10.2337/db16-0506

Adipose Triglyceride Lipase is a key regulator of hepatic triglyceride turnover and lipid homeostasis. This study identifies COP1 as an E3 ubiquitin ligase that targets ATGL for K48-linked proteasomal degradation, thereby suppressing fatty acid mobilization and oxidation. Loss of COP1 stabilizes ATGL, reduces hepatic steatosis, and improves liver function high-fat diet-fed mice. These findings establish the ubiquitin–proteasome system as an important regulator of hepatic lipid metabolism and highlight COP1 as a potential therapeutic target for Nonalcoholic Fatty Liver Disease.



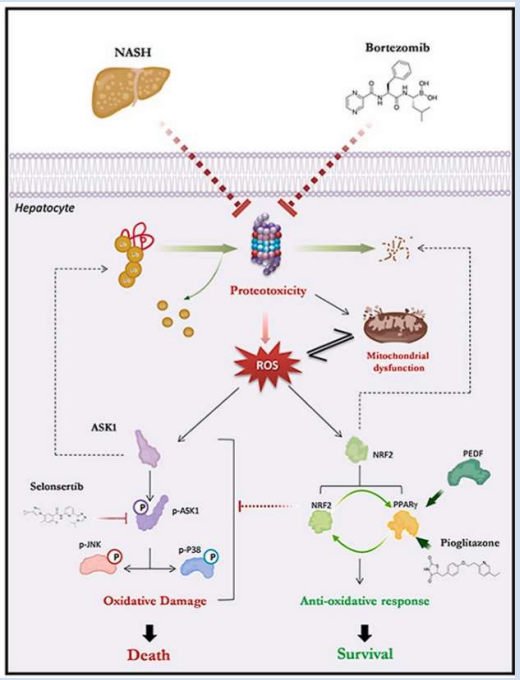
2. Adak, M., Das, D., Niyogi, S., Nagalakshmi, C., Ray, D., & Chakrabarti, P. (2018). Inflammasome activation in Kupffer cells confers a protective response in nonalcoholic steatohepatitis through pigment epithelium-derived factor expression. *FASEB journal : official publication of the Federation of American Societies for Experimental Biology*, fj201800190. Advance online publication. https://doi.org/10.1096/fj.201800190

Pigment Epithelium-Derived Factor was identified as a hepatoprotective hepatokine that limits hepatocyte death during progression of Nonalcoholic Steatohepatitis. The study showed that inflammasome-driven Interleukin 1 beta production from Kupffer cells induces hepatic PEDF expression, whereas Tumor Necrosis Factor alpha secreted by recruited hepatic macrophages suppresses it through differential NF-κB signaling. Functionally, PEDF protects hepatocytes by inhibiting TNF-α-mediated extrinsic apoptosis pathways. These findings establish a mechanistic link between inflammasome activation, macrophage signaling, and hepatocellular survival during NASH progression.



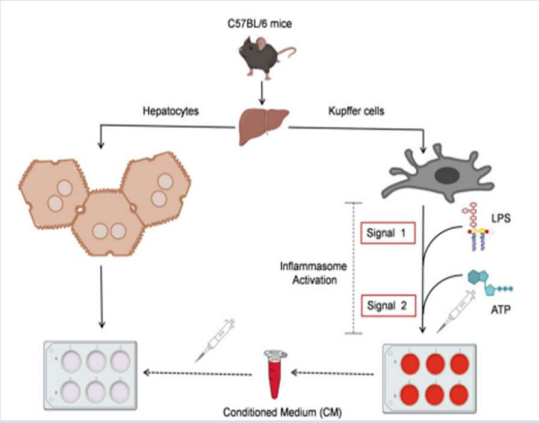
3.Niyogi, S., Ghosh, M., Adak, M., & Chakrabarti, P. (2019). PEDF promotes nuclear degradation of ATGL through COP1. *Biochemical and biophysical research communications*, *512*(4), 806–811. https://doi.org/10.1016/j.bbrc.2019.03.111

Pigment Epithelium-Derived Factor regulates hepatic lipid metabolism by promoting Adipose Triglyceride Lipase degradation through the E3 ubiquitin ligase COP1. The study shows that PEDF enhances nuclear translocation of ATGL, enabling its COP1-dependent proteasomal degradation within the nucleus. Through this mechanism, PEDF modulates hepatocyte lipid accumulation and fatty acid mobilization in a cell-autonomous manner. These findings uncover a previously unrecognized PEDF–COP1–ATGL regulatory axis controlling hepatic lipid turnover.



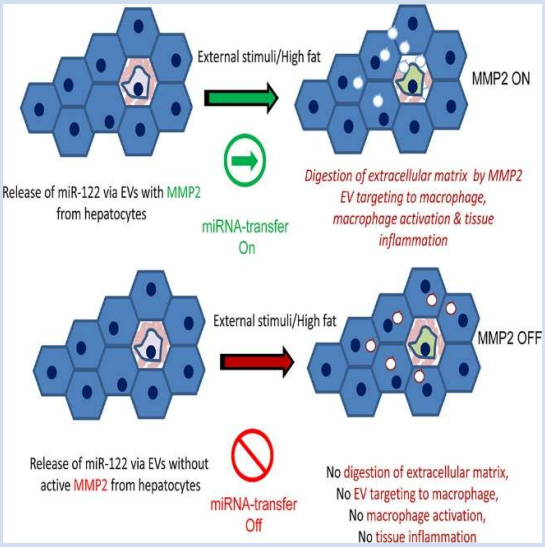
4. Das, D., Paul, A., Lahiri, A., Adak, M., Maity, S. K., Sarkar, A., Paul, S., & Chakrabarti, P. (2021). Proteasome dysfunction under compromised redox metabolism dictates liver injury in NASH through ASK1/PPARγ binodal complementary modules. *Redox biology*, *45*, 102043. https://doi.org/10.1016/j.redox.2021.102043

Proteasome dysfunction in hepatocytes induces severe oxidative and metabolic stress through activation of the Apoptosis Signal-regulating Kinase 1–JNK/p38 pathway alongside insufficient Peroxisome Proliferator-Activated Receptor Gamma–Nuclear Factor Erythroid 2-Related Factor 2-mediated antioxidant defense. The study demonstrates that endogenous PPARγ activation by Pigment Epithelium-Derived Factor is essential for protection against proteotoxic liver injury and for effective therapeutic response to ASK1 inhibition. Combined pharmacological activation of PPARγ with ASK1 inhibition synergistically ameliorated hepatotoxicity, steatosis, fibrosis, and hepatocellular death in preclinical Nonalcoholic Steatohepatitis models. These findings uncover an interdependent ASK1–PPARγ molecular circuit linking proteostasis failure to oxidative liver injury and identify a therapeutically targetable pathway in NASH.



5. Das, D., Adak, M., & Chakrabarti, P. (2022). Ex Vivo Dual-Hit Method for Inflammasome Activation in Liver. *Methods in molecular biology (Clifton, N.J.)*, *2455*, 255–265. https://doi.org/10.1007/978-1-0716-2128-8_20

This study describes an ex vivo platform to investigate inflammasome-mediated crosstalk between hepatic macrophages and hepatocytes in the context of Nonalcoholic Steatohepatitis. Primary Kupffer Cells were activated using LPS and ATP to induce NLR Family Pyrin Domain Containing 3 signaling and robust Interleukin 1 beta secretion, after which conditioned media were used to assess hepatocyte responses. The protocol enables mechanistic evaluation of cytokine-dependent hepatocellular injury and inflammatory signaling using isolated primary liver cells. This approach provides a robust experimental framework to study inflammasome activation and macrophage–hepatocyte communication during liver disease progression.



6. Das, A., Basu, S., Bandyopadhyay, D., Mukherjee, K., Datta, D., Chakraborty, S., Jana, S., Adak, M., Bose, S., Chakrabarti, S., Swarnakar, S., Chakrabarti, P., & Bhattacharyya, S. N. (2021). Inhibition of extracellular vesicle-associated MMP2 abrogates intercellular hepatic miR-122 transfer to liver macrophages and curtails inflammation. *iScience*, *24*(12), 103428. https://doi.org/10.1016/j.isci.2021.103428

Hepatic MIR122 promotes liver inflammation through extracellular vesicle-mediated transfer from lipid-stressed hepatocytes to tissue macrophages, leading to inflammatory cytokine production. The study identifies Matrix Metallopeptidase 2 as a critical regulator of extracellular vesicle movement and uptake by facilitating their transport across the extracellular matrix. Inhibition of MMP2 impaired intercellular transfer of miR-122 and attenuated hepatic innate immune activation and metaflammation. These findings establish MMP2-dependent extracellular vesicle trafficking as a key mechanism linking hepatic lipid stress to obesity-associated liver inflammation.