CURRICULUM VITAE

DR. SAHELI ROY

Cancer Biology and Inflammatory Disorder Division, CSIR-IICB, Jadavpur-700032, West Bengal, India

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RESEARCH HIGHLIGHTS

I began my research journey in the Infectious Disease and Immunology division during my master's thesis. Following this, I joined Dr. Krishna Das Saha's lab at the Indian Institute of Chemical Biology, India, as a graduate research fellow. With over six years of experience, I have conducted extensive research in cancer biology, inflammatory disorders, and nanomedicine.

RESEARCH EXPERIENCE

Graduate Research Fellow (2016-2023):
Cancer Biology and Inflammatory Disorder Division, CSIR-IICB, Jadavpur-700032, West Bengal, India
Supervisor: **Dr. Krishna Das Saha**
Project: **Study on potential of chrysin and its nanoformulations in ameliorating pulmonary inflammatory disorders**
Research Trainee (2013-2014):
Division of Immunology, ICMR-RMRIMS, Agamkuan, Patna**,** Bihar, India
Supervisor: **Dr. Sanjiva Bimal**
Project**: Activation of endogenous CD2 pathway in immunomodulation and its relevance in adjunct chemotherapy for control of visceral leishmaniasis**

ACADEMIC EXPERIENCE

Assistant Professor (2021- ongoing) 3 years plus teaching experience for bachelors and masters pharmacy students at Adamas University, Kolkata **.**

Assistant Professor (2016) 6-month teaching experience for bachelors and diploma pharmacy students at Bihar college of Pharmacy, **BCP** Patna

QUALIFICATION

Ph.D. (2023): Jadavpur University and Cancer Biology and InflammatoryDisorder Division, CSIR-IICB, Jadavpur-700032, West Bengal, India
Supervisor: **Dr. Krishna Das Saha**
Project: **Study on potential of chrysin and its nanoformulations in ameliorating pulmonary inflammatory disorders**

M. S. Pharm (Biotechnology) 2014 : NIPER, Hajipur, Bihar. **(Awarded First Class)**
B.Pharm 2012 : MAKAUT(formerly WBUT),Kolkata. **(Awarded First Class)**

AWARDS AND SCHOLARSHIP

1) Awarded **ICMR-NET** fellowship (ICMR NET-2015, in Life Science to support my doctoral research given by the Indian Council of Medical Research, Government of India.

2) Qualified Graduate Aptitude Test (**GATE-2016** in Life Science) conducted by Indian Instituteof Technology (IIT)

3) Qualified TIFR **JGEEBILS**– 2016 in biological science.

4) Qualified **GPAT** (Graduate Pharmacy Aptitude Test) - 2012

5) Qualified **NIPER-JEE** (National Institute of Pharmaceutical Education and Research) entrance exam and availed fellowship for M. S. Pharm (Biotechnology) education from Ministry of chemicals and fertilizers, Government of India (2012)

6) Qualified **WBJEE AND AIEEE** - 2008

RESEARCH EXPERIENCE

My Ph.D. research focused on improving the pharmacokinetics and biomedical effectiveness of Chrysin (CHR), a natural flavonoid with potent anticancer, anti-inflammatory, anti-asthma, and antioxidant properties, but limited by poor water solubility. I developed polymeric and inorganic nanoformulations of CHR to enhance its therapeutic efficacy.

1. Anti-Asthmatic Efficacy of CHR-Loaded PLGA Nanoparticles

In this study, I formulated CHR-loaded PLGA nanoparticles (CHR-NP) to enhance CHR's anti-asthmatic properties in an OVA-induced allergic asthma mouse model. These nanoparticles demonstrated an average size of 99.034±9.494 nm, encapsulation efficiency of 91.45±1.4%, and drug loading of 8.37±0.12%. CHR-NP exhibited a sustained release profile and effective cellular uptake by A549 cells.

Mechanistic studies revealed that CHR-NP treatment led to a significant decrease in the expression of Toll-like receptors 2 and 4 (TLR-2/4), which are critical in recognizing allergens and initiating inflammatory responses. By downregulating these receptors, CHR-NP effectively disrupted the signaling pathways that lead to inflammation.

Additionally, CHR-NP inhibited the activation of the nuclear factor-kappa B (NF-κB) pathway, a key regulator of immune and inflammatory responses. Normally, the activation of TLR-2/4 triggers the translocation of NF-κB into the nucleus, where it promotes the transcription of pro-inflammatory cytokines. CHR-NP treatment prevented this translocation by stabilizing the inhibitor of kappa B (IκB), thereby reducing the levels of NF-κB in the nucleus and subsequently decreasing the production of inflammatory cytokines.

Furthermore, CHR-NP suppressed the activation of the NLRP3 inflammasome, a multiprotein complex involved in the maturation and release of pro-inflammatory cytokines like IL-1β and IL-18. The inflammasome's activation is crucial for the inflammatory response in allergic asthma. By inhibiting NLRP3 activation, CHR-NP reduced the release of these cytokines, leading to diminished inflammation and allergic symptoms.

These mechanistic insights demonstrate CHR-NP’s potential as a comprehensive anti-inflammatory treatment for allergic asthma by targeting and modulating multiple inflammatory pathways.

2. Synergistic Anticancer Effects of CHR-Functionalized Gold Nanoparticles with Paclitaxel in NSCLC

In another study, I investigated the synergistic anticancer effects of CHR-functionalized gold nanoparticles (CHR-AuNP) combined with paclitaxel (PTX) in non-small cell lung cancer (NSCLC). PTX, despite its efficacy, has significant side effects. The combination aimed to reduce toxicity and enhance therapeutic outcomes.

The combination therapy of CHR-functionalized gold nanoparticles (CHR-AuNP) and paclitaxel (PTX) exhibited potent anticancer effects on A549 non-small cell lung cancer (NSCLC) cells by inducing apoptosis. This was demonstrated by an increased Bax/Bcl-2 ratio, which is a hallmark of apoptosis, as well as the release of cytochrome c from mitochondria into the cytosol. The release of cytochrome c triggers the activation of caspases, crucial proteins involved in the execution of apoptosis. Additionally, elevated levels of reactive oxygen species (ROS) and phosphorylated p53 (p-p53) further exacerbated cellular stress, promoting apoptosis through the mitochondrial pathway.

Moreover, the combination therapy also targeted the Wnt/β-catenin signaling pathway, a critical regulator of cell proliferation, differentiation, and survival. This pathway is frequently dysregulated in cancers, including NSCLC, contributing to uncontrolled cell growth and resistance to apoptosis. CHR-AuNP and PTX treatment enhanced the expression of peroxisome proliferator-activated receptor gamma (PPAR-γ), a tumor suppressor that inhibits the Wnt/β-catenin pathway. PPAR-γ achieves this by facilitating the phosphorylation and subsequent degradation of β-catenin, thereby preventing its accumulation in the nucleus. In its active form, β-catenin can drive the transcription of genes responsible for cell proliferation and survival, promoting tumor growth.

By reducing β-catenin levels and activating PPAR-γ, the therapy effectively disrupted the Wnt/β-catenin signaling cascade. This resulted in diminished tumor cell proliferation and enhanced apoptosis. The enhanced PPAR-γ activity also provided additional anti-inflammatory and anti-proliferative effects, further strengthening the overall anticancer efficacy of the treatment. These combined mechanisms—induction of apoptosis through the mitochondrial pathway and inhibition of the Wnt/β-catenin signaling—highlight the therapeutic potential of CHR-AuNP and PTX as a synergistic anticancer treatment for NSCLC.

Overall, the combination of CHR-AuNP with PTX amplified cytotoxic effects against NSCLC, positioning CHR-AuNP as an effective chemosensitizer and potential anticancer agent.

My Ph.D. research demonstrated that nanoformulations of CHR significantly enhance its biomedical properties. CHR-loaded nanoparticles showed promising results in treating allergic asthma and lung cancer, paving the way for developing nanotechnology-based phytopharmaceuticals for various diseases.

SELECTED SYMPOSIUM/ CONFERENCE PRESENTATION

1) Bioterm 2019, IIT-Kanpur. Poster presented on “Chrysin loaded PLGA ameliorates OVA-induced allergic asthma by modulation of NF-κB/NLRP3 axis.”
2) ISNSCON 2018.Poster presentation on “Chrysin loaded PLGA ameliorates ova-induced allergic asthma by modulation of NF-κB pathway.”

3) XI global health care summit 2017: Health care, Commerce &Career: American Association of Physicians of Indian Origin. Poster presented on topic "PLGA encapsulated nano andrographolide ameliorates OVA-induced allergic asthma in mice by upregulating Th2 type cytokines and targeting 5-LOX pathway."

LIST OF PUBLICATIONS

1. **Roy, S.,** Kant, S., Das Saha, K., & Jha, T. (2024). Chrysin-functionalized gold nanoparticles and paclitaxel exhibit synergistic impact on lung cancer cell lines via regulating the AKT/PPAR-ϒ/β-Catenin pathway. *Drug Development and Industrial Pharmacy*, *50*(6), 1-27.

2. **Roy, S.,** Kant, S., & Saha, K. Das, (2024). Role Effectuated by Immune Microenvironment on the Prognosis of COVID-19 Infected Asthmatic Patients and the Potentialities of Repurposing Immunomodulatory Drugs. Coronaviruses, 5: p. 1-10.
3. Kant, S., Das, S., **Roy, S**., & Tripathy, S. (2024). Fungal cellulases: a comprehensive review. *The Nucleus*, 1-17. I.F – 2.1
4. **Roy, S.,** Manna, K., Jha, T., & Saha, K. D. (2020). Chrysin-loaded PLGA attenuates OVA-induced allergic asthma by modulating TLR/NF-κB/NLRP3 axis. *Nanomedicine : nanotechnology, biology, and medicine*, *30,* 102292.

5. Chakraborty, S., Ehsan, I., Mukherjee, B., Mondal, L., **Roy, S.,** Saha, K. D., Paul, B., Debnath, M. C., & Bera, T. (2019). Therapeutic potential of andrographolide-loaded nanoparticles on a murine asthma model. *Nanomedicine:nanotechnology,biology,andmedicine*, *20*, 102006.
6. Banerji, B., Chandrasekhar, K., Sreenath, K., **Roy, S**., Nag, S., & Saha, K. D. (2018). Synthesis of Triazole-Substituted Quinazoline Hybrids for Anticancer Activity and a Lead Compound as the EGFR Blocker and ROS Inducer Agent. *ACS omega*, *3*(11), 16134–16142.

BOOKS AUTHORED

1.A comprehensive text book for pharmaceutical microbiology.**ISBN-978-93-6087-485-8**
2.A laboratory manual of food chemistry**.(IIP publisher) ISBN-978-93-6252-831-5**

LIST OF REFEREES

Dr. (Mrs.) Krishna Das Saha
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