

**SUSMITA BARMAN, Ph.D**

Postdoctoral Research Associate

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E-mail: [susmita.barman@unmc.edu](mailto:susmita.barman@unmc.edu)**Academic Qualifications**

Degree	Year	Organisation	Subjects studied
Post-Doc	2018-Present	UNMC, USA	Department of Biochemistry
Ph.D.	2012–2018	CSIR-CFTRI	Department of Biochemistry
M.Sc.	2009–2011	University of Calcutta	Food and Nutrition Science
B.Sc.	2006–2009	University of Calcutta	Food and Nutrition Science
P.G.Diploma	2010	Int. Diabetes Federation	Diabetes Education

**Awards/Recognitions**

1. **UGC-Research Fellowship** (2012–2017) funded by University Grants Commission, New Delhi (Govt. of India).
2. **National Scholarship** (2009–2011) for pursuing folk music funded by Ministry of Culture, New Delhi (Govt. of India).
3. **Gold Medal in Master of Music (2007).**

**Research Experience**

**Post-Doctoral Program** (Nov. 2018 – Present): University of Nebraska, Medical centre; Department of Biochemistry and molecular biology, Omaha, Nebraska, USA. Topic: role of greatwall kinase in the progression pancreatic cancer; FDA drug repurposing on colon cancer

**Ph.D.programme** (Nov. 2012 – Jan. 2018): UGC- Research Fellow, Department of Biochemistry, CSIR–Central Food Technological Research Institute, Mysore, India. Thesis topic: “**Animal studies on the attenuation of diabetic complications by Zinc supplementations**” (Awarded: 31/01/2018).

**Professional Experience**

1. **Internship Course** (Jan. 2009); Supervisor: Mrs. Mita Shukla, Sr. Executive, Clinical Nutrition and Dietetics Department, **WOCKHARDT**, Associated Hospital of HARVARD MEDICAL INTERNATIONAL, Kolkata.
2. **Nutrition Councilor** (Jan.–April. 2011) @ Apollo Gleneagles Nursing home, Kolkata.

3. **Diet Councilor** (Feb.-July 2012) @ a Private Clinic, Kolkata, India.

### **Professional background**

**Ph.D programme (Nov. 2012 – Jan. 2018)** entitled “*Animal studies on the attenuation of diabetic complications by Zinc supplementations*”.

Zinc dyshomeostasis and increased levels of oxidative stress have major roles in the pathogenesis of diabetes mellitus. Zinc concentrated in the islet cells is related to the synthesis, storage and secretion of insulin. Loss of large amounts of zinc from the body via urine and faeces is reported in diabetic situation. It is not clear if loss of zinc consequent hyperzincuria or decreased gastrointestinal absorption of zinc or both are responsible for diabetes and its' related complication. While it is clear that urinary excretion of zinc is markedly increased in diabetes, replacement with oral zinc supplementation should provide sufficient benefit. Hence, the investigation was intended to explore the beneficial modulatory potential of zinc supplementation with respect to: (a) Hyperglycemia and attendant abnormalities in experimental diabetes, (b) Beneficial modulation of compromised antioxidant status, (c) Renal lesions, cataract and risk of cardiovascular disease, and (d) gastrointestinal health in diabetic condition. Diabetes was experimentally induced with streptozotocin and diabetic rats were exposed for six weeks with supplemental zinc (5-times and 10-times of RDA). Dietary zinc supplementation had a significant beneficial effect in the control of zinc fluxes in diabetes induced Zn dyshomeostasis, through a protective influence on the modulation of expression of the tissue specific zinc transporters and along with protective influence on the oxidative stress induced metallothionein proliferation in these organs. Parameters related to metabolic abnormalities, nephropathy, cataract, and cardiovascular diseases were investigated at basic and molecular levels. Overall, this study added more insights into the mode of action of zinc supplementation in alleviating metabolic abnormalities in diabetes and its related secondary complications. Dietary zinc treatment showed a significant favorable effect on the restoration of structural integrity of small intestines in diabetic situation, and also exerted a promoting stimulus on the intestinal absorption of zinc, iron and calcium, which could encourage a dietary approach to counter the dyshomeostatic state of these trace elements prevalent in diabetes.

**Post-Doctoral Program (Nov. 2018 – Present):** Pancreatic ductal adenocarcinoma (PDAC) remains predominantly one of the fatal malignancies with a frightening resistance to chemotherapeutic and inept targeted options. Anti-cancer treatments or targeted therapy act primarily by damaging the DNA of cancer cells and consequently, inhibiting proliferation at cell cycle checkpoints. Hence, pharmacological inhibition of checkpoint kinases in combination with the DNA damaging anti-cancer therapies (chemotherapy or radiotherapy) is now emerging as promising cancer treatment strategy. In this regard, it is known to regulate pathways for DNA damage response and Cell Cycle and is recently found to be upregulated in different type of cancers. However, its role and regulation in PDAC, is currently unknown.

We used immunoblotting, immunohistochemistry, and TCGA database analysis to examine Great-wall kinase (GK) expression and its association with pancreatic cancer (PC) progression using pancreatic cancer cells, mouse models and patient samples. Genetically manipulated and inhibitor (GKI) treated cells were extensively monitored for its expression and kinase activity. Our results suggest a cross talk of Great-wall kinase/EGFR signalling pathway in PC progression. We hypothesize that combinatorial therapy targeting GK along with conventional chemotherapy (gemcitabine) would overcome the drug resistance in the PDAC. Overall, these observations account a novel therapeutic target GK, regulate PDAC through the regulation of EGFR expression.

**Research Publications** ([Google Scholar](#), [PubMed](#))

**Published:**

SI No	Title, Authors, Journal name, Year, Volume, Page Nos.	Impact Factor
1.	MASTL Regulates EGFR Signalling to Impact Pancreatic Cancer Progression., Fatima, I.*, <b>Barman, S.*</b> , Uppada, J., Chauhan, S., Rauth, S., Rachagani, S., ... & Dhawan, P. <i>Oncogene</i> , <b>2021</b> : 1-14	<b>9.9</b>
2.	Pancreatic Cancer and Therapy: Role and Regulation of Cancer Stem Cells. <b>Barman, S.</b> , Fatima, I., Singh, A.B. and Dhawan, P., <i>International Journal of Molecular Sciences</i> , <b>2021</b> : 22(9), p.4765.	<b>5.9</b>
3.	Targeting Wnt Signaling in Endometrial Cancer. Fatima, I*., <b>Barman, S*</b> ., Rai, R., Thiel, K.W. and Chandra, V. <i>Cancers</i> , <b>2021</b> : 13(10), p.2351.	<b>6.6</b>
4.	Diabetes and zinc dyshomeostasis: Can zinc supplementation mitigate diabetic complications? <b>Barman, S</b> & Srinivasan, K. <i>Critical Reviews in Food Science and Nutrition</i> , <b>2020</b> : 1-16	<b>11.18</b>
5.	Attenuation of hyperglycemia-mediated renal lesions by dietary fenugreek seeds ( <i>Trigonella foenum-graecum</i> L.) and onion ( <i>Allium cepa</i> L.) via blockade of glucose translocation and renin-angiotensin system. Pradeep, S.R. <b>Barman, S</b> & Srinivasan, K. <i>Nutrition</i> , <b>2019</b> : 67-68: 110543	<b>4.01</b>
6.	Ameliorative effect of zinc supplementation on compromised small intestinal health in streptozotocin-induced diabetic rats. <b>Barman S</b> , Srinivasan K. <i>Chemico-Biological Interaction</i> . <b>2019</b> Jul 1;307:37-50.	<b>5.19</b>
7.	Zinc Supplementation Ameliorates Diabetic Cataract Through Modulation of Crystallin Proteins and Polyol Pathway in Experimental Rats. <b>Barman S</b> , Srinivasan K. <i>Biological Trace Element Research</i> . <b>2019</b> Jan;187(1):212-223.	<b>3.74</b>
8.	Enhanced intestinal absorption of micronutrients in streptozotocin-induced diabetic rats maintained on zinc supplementation. <b>Barman S</b> , Srinivasan K. <i>Journal of Trace Element Medicinal Biology</i> . <b>2018</b> Dec;50:182-187.	<b>3.85</b>
9.	Zinc supplementation alleviates the progression of diabetic nephropathy by inhibiting the overexpression of oxidative stress- mediated molecular markers in streptozotocin-induced experimental rats. <b>Barman, S.</b> , Pradeep, S.R. & Srinivasan, K. <i>The Journal of Nutritional Biochemistry</i> , <b>2018</b> , 54: 113–129.	<b>6.048</b>
10.	Zinc supplementation mitigates its dyshomeostatsis in experimental diabetic rats by regulating the expression of zinc transporters and metallothionein. <b>Barman, S.</b> , Pradeep, S.R. & Srinivasan, K. <i>Metallomics</i> , <b>2017</b> , <b>9</b> : 1765-1777.	<b>4.526</b>
11.	Attenuation of oxidative stress and cardioprotective effects of zinc supplementation in experimental diabetic rats. <b>Barman S</b> , Srinivasan K. <i>British Journal of Nutrition</i> . <b>2017</b> Feb;117(3):335-350.	<b>3.6</b>
12.	Zinc supplementation alleviates hyperglycemia and associated metabolic abnormalities in streptozotocin-induced diabetic rats. <b>Barman S</b> , Srinivasan K. <i>Canadian Journal of Physiology and Pharmacology</i> . <b>2016</b> Dec;94(12):1356-1365.	<b>2.27</b>
13.	Zinc: the essential micronutrient. – <b>Susmita Barman</b> & K. Srinivasan ( <i>Vigyan Prasar's News Letter</i> , <b>July 2019</b> ; 21(10): 28-31	
14.	<b>Barman, S.</b> , I Fatima, AB Singh, SK Batra, P Dhawan ( <b>2020</b> ) MASTL Regulates EGFR Signaling to Promote Pancreatic Cancer Progression. <i>Pancreas</i> <b>49 (10)</b> , 1399-1442	<b>2.9</b>

**Communicated:**

- Barman, S.**, Pradeep, S.R. and Srinivasan, K. Exogenous zinc mitigates high glucose induced EMT by regulating the zinc transporters/metallothionein and oxidative stress in rat primary intestinal epithelial cell and HIEC-6 cell line. (Being communicated).

### **Conference participated**

1. **Barman, S**, Pradeep S.R., and Srinivasan, K. Zinc supplementation alleviates zinc dyshomeostasis by metallothionein and zinc transporter changes in tissue of streptozotocin–induced diabetic rats. 85<sup>th</sup> Annual meeting of Society of Biological Chemists (India), @ Mysore, India (Nov. 2016).
2. I Fatima, **S Barman**, S Chauhan, JP Uppada, G Talmon, AB Singh, ...MASTL, a Novel Therapeutic Target for Pancreatic Cancer Progression LIPPINCOTT WILLIAMS & WILKINS 48 (10), 1426-1427
3. **Barman, S.**, I Fatima, AB Singh, SK Batra, P Dhawan (**2020**) MASTL Regulates EGFR Signalling to Promote Pancreatic Cancer Progression, APA, 50<sup>th</sup> Annual meeting (Virtual).

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