Dr. Anirban Chatterjee

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http://www.researchgate.net/profile/Anirban_Chatterjee9
http://scholar.google.co.in/citations?user=Crs1TVsAAAAJ&hl=en

Education

2014 **Ph.D.** Cellular and Molecular Biology, Cancer Biology;
CSIR-Indian Institute of Chemical Biology,
Department of Biotechnology,
University of Calcutta, India.

2008 **M.Sc.** Zoology (1st class/ 5.37 CGPA out of 6.0/89.00%);
University College of Science and Technology, Ballygaunge
University of Calcutta, India.

2006 **B.Sc.** Zoology, (1st class/ 71.00%);
Vivekananda College,
University of Calcutta, India.

Professional Experience

January, 2017– till date: **Assistant Professor (I)**

Department of Zoology,

Bolpur College
University of Burdwan
West Bengal, India

Research Experience

March, 2015–December, 2016: **Post-doctoral Research Associate**

Department of Human Oncology, Wisconsin Institute of Medical Research
Carbone Cancer Center
University of Wisconsin-Madison School of Medicine and Public Health
Madison, WI, USA

January, 2009–January, 2014: **Ph.D.**

Fellowship sponsored by: CSIR-Govtof India Division of Cancer Biology and Inflammatory Disorder
CSIR-Indian Institute of Chemical Biology
Advisor: Dr. Mrina l Kanti Ghosh, *Principal Scientist*
Registration Affiliation: Department of Biotechnology, University of Calcutta

Title of the Thesis:

Protein Kinase CK2 evokes premature degradation of PML in Cancer: Implicative study of some crucial pro-oncogenic signaling dynamics

PhD Research Summary*: The main focus of my Ph.D. dissertation was studying the molecular dynamics of signaling crosstalks and the mechanisms behind the perturbation of normal cellular physiology caused by the perturbations of some crucial signaling cascades that may eventually lead towards oncogenesis.My primary objective was to study the role of one of the prominent kinase playing the pivotal role in dismaying one of the crucial tumor suppressive – apoptosis signaling circuitry, by impeding the activity of a group of pivotal proteins and disengaging their normal association that was critical in the maintenance of normal cellular functioning and preventing oncogenic manifestations [for detail please see* ***Appendix-I****).*

Awards and Honors (Academic):

2010 **UGC-CSIR-Senior Research Fellowship**

2007 **UGC-CSIR-Junior Research Fellowship**

Awards and Honors (Research):

2013 Awarded among **the best five top scoring abstract**
atthe 10th Annual International Conference of Asian Society of Neuro-oncology (ASNO) jointly with the 5th Annual Conference of Indian Society of Neuro-oncology (ISNO)
Mumbai*March, 2013*

2011 **Best Performance Award at the Oral Presentation Category**
at the 2nd International Conference of "NEURON: DEGENERATION, REGENERATION & PROLIFERATION - NEUROCON-2011"
Kolkata, *January, 2011*

Invited Lecture:

2016 **Centenery Lecture**
Department of Zoology, Asutosh College, Kolkata, INDIA
On the occasion of Centenary Celebration along with 75th anniversary of Department of Zoology, February-2016

Membership:

International Society of Zoological Sciences (1413-I)

Indian Society of Chemists and Biologists (LF-800/2017)

ASSOCIATION OF TEACHERS IN BIOLOGICAL SCIENCES (LF/2018)

Publications:

Research/Review articles in international peer-reviewed journals

2015 **Modulation of therapeutic sensitivity by human papillomavirus** *(review article)*
Adam Swick, ***Anirban Chatterjee***, P Anna-Maria DeCosta, and Randall. J .Kimple.
Radiotherapy and Oncology ;September, 2015; 116(3): 342 -345
*PMID:26364887; PMCID: PMC4609293; ISSN:*0167-8140
URL:
http://www.sciencedirect.com/science/article/pii/S0167814015004600

2014 **Reduced phosphorylation of Stat3 at Ser-727 mediated by Casein Kinase 2 Protein phosphatase 2A enhances Stat3 Tyr-705 induced tumorigenic potential of glioma cells.**
*Mandal T, Bhowmik A,**Chatterjee A**, Chatterjee U, Chatterjee S, Ghosh MK.*
Cell Signal. 2014April 12**;** 26(8): 1725 - 1734.
doi:10.1016/j.cellsig.2014.04.003.
PMID: 24726840; ISSN: 0898-6568
URL:http://www.sciencedirect.com/science/article/pii/S0898656814001387

2013 **Current Understanding on EGFR and Wnt/β-Catenin Signaling in Glioma and Their Possible Crosstalk** *(review article)*
Indranil Paul, Seemana Bhattacharya,* **Anirban Chatterjee**, Mrinal K. Ghosh *equal contribution*
Genes & Cancer November/December 2013 4: 427-446
PMCID: PMC3877660; ISSN: 1947-6019
URL: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3877660/

2013 **Activation of protein kinase CK2 attenuates FOXO3a functioning in a PML-dependent manner: implications in human prostate cancer.**
***Chatterjee A**, Chatterjee U, Ghosh MK.*
Cell Death Dis. 2013 Mar 14; 4:e543. doi: 10.1038/cddis.2013.63.
PMID: 23492774; PMCID: PMC361384; ISSN: 2041-4889
URL:http://www.nature.com/cddis/journal/v4/n3/full/cddis201363a.html

2012 **Mechanism of β-catenin-mediated transcriptional regulation of epidermal growth factor receptor expression in glycogen synthase kinase 3 β-inactivated prostate cancer cells.**
*Guturi KK, Mandal T, **Chatterjee A**, Sarkar M, Bhattacharya S, Chatterjee U, Ghosh MK.*
J Biol Chem. 2012 May 25; 287(22):18287-18296.
doi: 10.1074/jbc.M111.324798.
PMID: 22493441; PMCID: PMC3365735; ISSN: 0021-9258

URL:http://www.jbc.org/content/287/22/18287.long

2012 **The chaperone-assisted E3 ligase C terminus of Hsc70-interacting protein (CHIP) targets PTEN for proteasomal degradation.**

Ahmed SF, Deb S, Paul I, ***Chatterjee A****, Mandal T, Chatterjee U, Ghosh MK.*

J Biol Chem. 2012 May 4; 287(19):15996-16006.
doi: 10.1074/jbc.M111.321083
PMID: 22427670; PMCID: PMC3346122; ISSN: 0021-9258
URL:http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3877660/

Conferences/symposiums attended along with abstracts presented:

(either in form of poster or platform presentations, or being the principal or the co-author)

2016 **107th ANNUAL MEETING of AMERICAN ASSOCIATION OF CANCER RESEARCH (AACR)**

Duration: 16th to 20th April, 2016, New Orleans, LA

(presented poster as the principa lauthor of own research work)

2013 **10th MEETING OF ASIAN SOCIETY FOR NEURO-ONCOLOGY (ASNO)** jointly with the **5th ANNUAL MEETING OF INDIAN SOCIETY OF NEURO ONCOLOGY (ISNO)** Organized by **ASIAN SOCIETY FOR NEURO-ONCOLOGY** and **INDIAN SOCIETY FOR NEURO-ONCOLOGY**

Duration: 21st to 24th March, 2013; Mumbai

(Also presented a poster and delivered an oral presentation as the principal author of own research work)

2013 **32nd ANNUAL CONVENTION OF INDIAN ASSOCIATION FOR CANCER RESEARCH (IACR)** and **One Day International Symposium on "INFECTION AND CANCER"** Organized by **INDIAN ASSOCIATION FOR CANCER RESEARCH (IACR) and DR. B R AMBEDKAR CENTER FOR BIO-MEDICAL RESEARCH (ACBR), UNIVERSITY OF DELHI**

Duration:13th to 16th February, 2013; New-Delhi.

(Also presented a poster as the principal author of own research work)

2012 Two day National Conference on **ADVANCEMENT OF SCIENCE AND TECHNOLOGY AND SUSTAINABLE DEVELOPMENT FOLLOWING THE IDEALS OF SWAMI VIVEKANANDA** Organized by **VIVEKANANDA INSTITUTE OF ENVIRONMENT & MANAGEMENT, KOLKATA & HERITAGE INSTITUTE OF TECHNOLOGY, KOLKATA**

Duration: 16th and 17th November, 2012; IACS, Kolkata.

2011 **3rdANNUAL A CONFERENCE OF THE INDIAN SOCIETY OF NEURO-ONCOLOGY**, ISNOCON-2011- and **Three Day International Symposium on "SELLAR TUMOUR"** Organized by **INDIAN SOCIETY OF NEURO-ONCOLOGY (ISNO)-WEST BENGAL CHAPTER** and **PARK CLINIC, KOLKATA**
Duration: 25th to 27th March, 2011; Kolkata.
(Also presented an abstract as co-author)

2011 **30thANNUAL CONVENTION OF INDIAN ASSOCIATION FOR CANCER RESEARCH (IACR)** and Four Day International Symposium on "**SIGNALING NETWORK AND CANCER**" Organized by **INDIAN ASSOCIATION FOR CANCER RESEARCH (IACR)** and **INDIAN INSTITUTE OF CHEMICAL BIOLOGY (IICB)-CSIR**
Duration: 6th to 9th February, 2011; CSIR-IICB, Kolkata

2011 Three Day International Conference on "**NEURON: DEGENERATION, REGENERATION & PROLIFERATION - NEUROCON-2011**" Organized by **DEPARTMENT OF BIOCHEMISTRY, INSTITUTE OF POST-GRADUATE MEDICAL EDUCATION AND RESEARCH AND SETH SUKHLAL KARNANI MEMORIAL HOSPITAL (IPGMER-SSKM), KOLKATA**"
Duration: 29th to 31st January, 2011, Kolkata.
(Also delivered an oral presentation of own research work and have secured the FIRST prize)

2010 One Day International Symposium on "**CHALLENGES IN MODERN BIOLOGY**" Organized by **CENTRE FOR MODERN BIOLOGY AND DR B C GUHA CENTRE FOR GENETIC ENGEERING AND BIOTECHNOLOGY (GCGEB), UNIVERSITY OF CALCUTTA**"
On: 28th December, 2010 at Department of Biotechnology, University of Calcutta, Kolkata.

2010 Three Day Regional Symposium on "**CURRENT TRENDS IN BIOLOGICAL SCIENCES**" Organized by **SOCIETY OF BIOLOGICAL CHEMISTS-KOLKATA CHAPTER**
Duration: 4th to 6th September, 2009; Digha, West Bengal.
(Also presented an abstract as co-author)

Professional Experiences:

- **Guest Lecturer**: Department of Microbiology,
  RashtraguruSurendranath College, Barrackpore
  (Special UGC-CSIR NET mentoring class)

- **Guest Lecturer:** Department of Zoology,
  Vivekananda College, Thakurpukur
  (externel Post-graduate course of Vidyasagar University)

- **Assistant Professor (I)**
  Department of Zoology,
  Bolpur College
  Also serving as **Head** (from 01.07.2019 – till 30.06.2021)

Disclosures and Declaration:

There haven't been any breaks in my academic career all throughout.

I haven't been punished/debarred in from any examinations for adopting unfair means or any other punishable offences.

I have never been convicted by a court for any criminal offence.

I hereby declare that all the above mentioned credential facts and informations are correct and true to the best of my knowledge.

Sd/-

Anirban Chatterjee

Date: *24.01.2020*

Place: Kolkata

REFEREES:

1. **DR. Mrinal Kanti Ghosh**; *Senior Principal Scientist cum Assoc. Profess.*
   Email: ***mrinalghosh@iicb.res.in/mrinal.res@gmail.com***

   Division of Cancer Biology and Inflammatory Disorder
   CSIR-Indian Institute of Chemical Biology (also AcCSIR)
   CN-06, CN Block, Sector V, Bidhannagar, Kolkata, WB 700091
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   ☎ (O): +91 33 2499 5700

2. **Dr. Randall J Kimple**; *Associate Professor*
   Email: ***rkimple@humonc.wisc.edu***

   Department of Human Oncology
   Carbone Cancer Center
   University of Wisconsin-Madison School of Medicine and Public Health
   1111, Highland Avenue, Madison 53705, WI, USA
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3. **Dr. Sandip Chatterjee;** *Professor*
   Consultant Neurosurgeon and Academic Coordinator, Park Clinic Kolkata
   Hony. Professor and Head of Neurosurgery, Vivekananda Institute of Medical Sciences, Kolkata
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4. **Dr Uttara Chatterjee;** *Professor*
   Consultant Pathologist, Park Clinic Kolkata
   Reader, Department of Pathology, IPGMER SSKM Hospital, Kolkata
   Email:*uttarac1@gmail.com*

   244 A.J.C Bose Road,
   Kolkata - 700 020
   ☎ (M): +91 9433 763528

*** Please Note: Referees [3] and [4] are not in any way my relatives, as it could appear from the family names/surnames. I absolutely do not possess any blood relation with them. They are just my research co-investigators and of course co-mentor too.**

Appendix: I

Ph.D. Research Summary:

My doctoral research has been aimed towards unraveling the molecular details and dynamics of some crucial signaling cascades and their crosstalks in the context of cancer.
Though in my earlier days of research study, I focused up on Glioma as my main model of cancer and was actively engaged in discerning one of the crucial signaling perturbation in it –the EGFR signaling and its related dysregulations.Glioblastoma (GBM) is the most frequent primary brain tumor in the adult and one of the most deadly among all human tumors. Despite current aggressive treatment at diagnosis, the tumor almost invariably recurs or progresses, with a median survival of 14.6 month. GBM is also considered to be one of the highly angiogenic and vascular tumors with prolific rate of proliferation and they are extensively heterogeneous at both cellular and molecular levels. In spite of all efforts, the prognosis of glioma patients remains dismal. Therefore towards the goal of proper understanding of individual molecular pathways responsible for the progression of GBM I had spent significant portion of my initial graduate study which continued even to the last days along with my principle objective.

Working on the basic platform of elucidating mechanisms of signalling crosstalks and perturbations, later on I focused upon Casein Kinase 2 (CKII), one of the ubiquitous Ser/Thr protein kinase present in both the nucleus and cytoplasm of cells, targeting several key enzymes, growth factor receptors, transcription factors and cytoskeletal proteins. It is not only a key player in regulating cellular growth and proliferation, but also behaves as a potent suppressor of apoptosis. CK2 has been frequently found to be deregulated (mostly hyper-activated) in all cancers, and prostate cancer being prominent of them, I chose to work on it as my model system and concentrating on the less explored nuclear role of CK2 towards onset of oncogenesis and its progress. In the recent past, tumor suppressor PML (promyelocytic leukemia) has been shown to be a target of phosphorylation by CK2 and thereby bereaved of its normal cellular functioning.

I have depicted existence of a novel signaling axis apexed by deregulated/hyper activated(expressed) CK2, dismantling the association of PML and PHLPP2 (incidentally, I have illustrated the phosphatase PHLPP2 to be a novel interacting partner of PML inside the nucleus), ultimately leading to the inactivation and nuclear exclusion of FOXO3a, thereby down regulating p21/p27/Bim in which degradation of PML and the concomitant stabilization of active AKT (pAKT) plays a cardinal part.

Similarly, I had also progresses up to a significant level in demonstrating the existence of an identical crusading effect of hyper-expresses CK2 upon PML especially in advanced grade of Glioma (GBM-IV),

whereby the PML protein level is depicted to significantly curbed, thus deprived of its critical tumor suppressive functioning in checking oncogenesis or its progress.

Post-doctoral Research Summary

Squamous cell carcinoma of head and neck (HNSCC) is a heterogeneous disease. Although recent advances in treatment have improved quality of life, overall 5 year survival rates have not improved significantly. HNSCC frequently shows local recurrence and metastasis after the initial treatment of the primary tumor. Mortality from this disease remains high because of the development of metastases and therapy-resistant local and regional recurrences. Progress in treatment and prognosis for HNSCC has been limited and the molecular mechanisms of HNSCC escape from chemo- and/or radiation therapies remain mostly unknown.Research indicates that a small population of cancer cells is highly tumorigenic, endowed with the capacity for self-renewal, and has the ability to differentiate into cells that constitute the bulk of tumors. These cells are considered the ‘‘drivers’’ of the tumorigenic process in some tumor types, and have been named cancer stem cells (CSC). There is increasing evidence that the growth and spread of cancers is driven by these small subpopulation of cancer stem cells (CSCs)— the only cells that are capable of long-term self-renewal and generation of the phenotypically diverse tumor cell population. CSCs have been identified and isolated in a variety of human cancers including head and neck squamous cell carcinoma (HNSCC). The concept of cancer stem cells may have profound implications for our understanding of tumor biology and for the design of novel treatments targeted toward these cells. CSCs have also been identified as one of the kye player in Epithelial-mesenchymal transition (EMT). Through this process, cells acquire an invasive phenotype that may contribute to tumor recurrence and metastasis. CSC are identified on the basis of specific markers, including membrane proteins or cell enzymes, or by using their self-renewal properties. As their resistance to standard HNSCC treatment may eventually lead to the lack of treatment success, there is an urgent need to better understanding CSC biology and identify them as potential target new treatment modality.

Wnt/Beta Catenin signalling have long been held as one of the crucial player in maintaining and progress of this CSC phenotype and manifestations of different characyeristic functionalities. Our initial investigations have shown that Wnt/Beta catenin signalling is responsible for the expression og various ‘stemness’ genes including Sox2. It controls various CSC phenotypes and expression of CSC specific markers. Our in vivo study in nude mice have depicted that H&N SCC cells lacking Beta catenin are severly affected in their tumor forming ability through the SOX2 gene.

Personal Informations:

Date of Birth:	**April 22nd 1984**
Place of Birth:	**Kolkata**
Citizenship:	**Indian**
Marital Status:	**Unmarried**

Languages known:

To speak:	Bengali, Hindi, English
To read:	Bengali, Hindi, English
To write:	Bengali, English

IT skills:

MS-Office,

Art Board, Adobe Illustrator,

Corel Draw, ImageJ

GraphPad Prism