*1.1 Name****: Dr. Kuladip Jana***

***M.Sc., M.S., Ph.D., Postdoc. (U.S.A.)***

*1.2 Designation and Department/Division:*

***Senior Scientist***

***Division of Molecular Medicine***

***Scientist In-Charge, Central Animal House & Research Facility (Centre for Translational Animal Research)***

***Bose Institute, P 1/12, CIT Scheme VIIM, Kolkata-700 054, India.***

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[***kuladip.jana@gmail.com***](mailto:kuladip.jana@gmail.com/)

*2. Date of birth:* ***3rd day of April, 1975.***

*3. Full details of Academic qualifications: (Bachelor’s degree onwards with awarding University/Institute, year, specialization, if any):*

***1996 B.Sc. (Hons), Physiology (major), Zoology & Botany (minor), Vidyasagar University, Midnapur, West Bengal, India, 1st class.***

*1998 M.Sc., Human Physiology with Community Health (specialization: Immunology & Microbiology), Vidyasagar University, Midnapur, West Bengal, India, 1st class.*

***2004 Ph.D.,in Reproductive Medicine from Vidyasagar University, Midnapur, West***

***Bengal & W.B. University of Animal & Fishery Sciences, Kolkata, India.***

***2005 M.S. (Professional Award) in Psychotherapy and Counselling, from Institute of Psychotherapy & Management Sciences, Mumbai, India.***

***2008 Post-doctorate in Molecular Reproductive Medicine from Garrison Institute of Aging, Texas Tech University of Health Sciences, Texas, USA.***

*4. Details and nature of present and previous employment (Positions held, employer/place of work, duration, area/nature of work):*

***2005–2006 Research Associate in a NIH (U.S.A.) project, Immunology and Vaccine Development Unit, National Institute of Cholera & Enteric Diseases (NICED), Kolkata, India, Research & Development***

***2006-2008 Postdoctoral Research Associate in a NIH (U.S.A.) project, Garrison Institute on Aging, Texas Tech University Health Sciences Center, Lubbock, Texas, USA. Research & Development***

***2009-2010 Research Associate II, Crystallography & Molecular Biology Division, Saha Institute of Nuclear Physics, Kolkata. Research & Development***

***2010- 2014 Scientist-C, Division of Molecular Medicine, Bose Institute, Kolkata, India, Research & Development***

***2014-till-date Sr. Scientist, Division of Molecular Medicine, Bose Institute, Kolkata, India, Research & Development***

***5. Research specialization (Major scientific fields of interest):***

# *Presently, my interest focusing on Translational animal research in the diverse directions i.e. Molecular signalling of germ cell/ Leydig cell apoptosis by Benzo(a)pyrene and its protection by natural aryl hydrocarbon receptor (AhR) antagonist, Molecular Mechanisms of Germ-line Stem Cell Regulation: Special emphasis on Diabetes and aging, Ageing associated oxidative stress & testicular gametogenic and steroidogenic disorders: ameliorating potential of natural antioxidants, Resveratrol Induced Apoptosis in Cervical and Breast Cancer Cells: role of β- catenin and Wnt signalling, Sulforaphane inhibits Akt mediated GSK3β and FoxO3a signaling and triggers apoptosis in breast cancer. Development of nanoparticle mediated treatment strategy to target NOTCH1 in triple negative breast cancer (TNBC) Xenograft model, Testicular carcinogenesis in relation with steroidogenesis and StAR protein expression: role of MAPKs, Novel anti-filarial and anti-cancer drug development from natural resources and to search the link between Polycystic Ovary Syndrome and both Type 1 and Type 2 Diabetes Mellitus as well as mechanism of wound healing by Calendula officinalis in diabetic condition.*

***6. Research interest:***

***(A) Molecular signaling involved in patho-physiological action of common air pollutant “Benzo(a)pyrene” in Germ cell DNA damage and apoptosis in connection to male infertility : possible protection by natural aryl hydrocarbon receptor antagonists.***

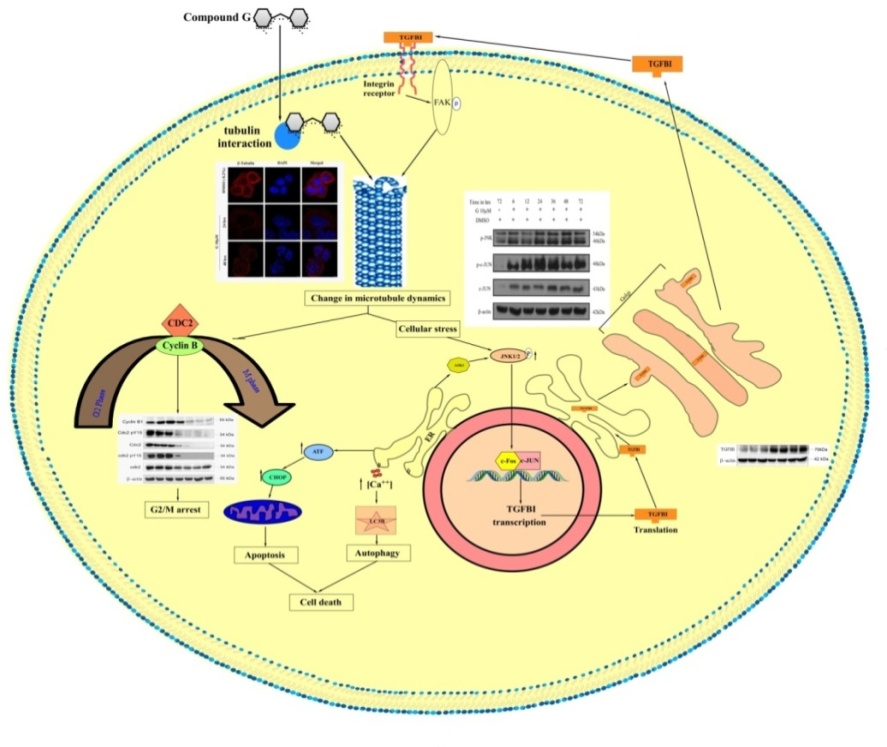
Benzo(a)pyrene (B(a)P) is an environmental toxicant that alters the steroidogenic profile of testis and induces testicular dysfunctions. In spite of the magnitude of the problem, the precise molecular and cellular mechanism of B(a)P mediated testicular damage and possible protective measures still remain unexplored. In the present study, we have investigated the molecular signaling of B(a)P and the ameliorative potential of the natural aryl hydrocarbon receptor (AhR) antagonist and antioxidant- 3,5,4'-trihydroxy-*trans*-stilbene (Resveratrol) on B(a)P induced male reproductive toxicity. Studies carried out in adult male Wistar rats significantly showed that B(a)P treatment resulted in p38 MAPK activation and increased iNOS production along with testicular apoptosis and steroidogenic dysfunction. Our study also highlighted that resveratrol co-treatment maintained testicular redox potential, increased serum testosterone level and enhanced major testicular steroidogenic proteins (CYPIIA1, StAR, 3β HSD,17β HSD) expression and subsequent onset of apoptosis. Resveratrol co-treatment also showed significant inhibition of protein and mRNA levels of testicular cytochrome P4501A1 (CYP1A1), which is the major B(a)P metabolizing agent for BPDE-DNA adduct formation. Resveratrol also significantly decreased the B(a)P-induced AhR protein levels, its nuclear translocation and subsequent promoter activation, thereby decreasing the expression of CYP1A1. Resveratrol down-regulates B(a)P-induced testicular iNOS production through suppressing the activation of p38 MAPK and ATF2 thereby improving the oxidative status of the testis and inhibiting apoptosis. Our findings thus cumulatively suggest that resveratrol exhibits both anti-initiating effects of B(a)P by modulating the transcriptional regulation of CYP1A1 and acting as an antioxidant thus preventing B(a)P-induced oxidative stress and testicular apoptosis.

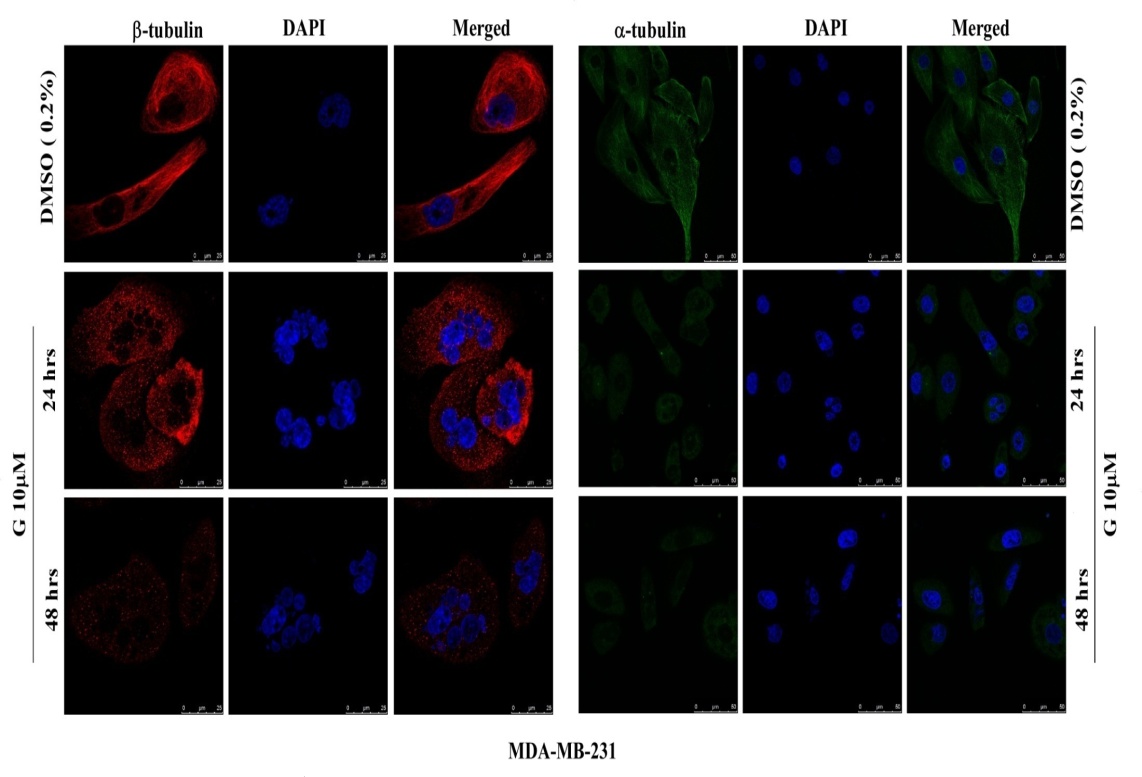


***(B) Molecular Mechanisms of Germ-line Stem Cell Regulation: Special emphasis on Diabetes and aging***

Germ-line stem cells (GSCs), which can self-renew and generate differentiated progeny, are unique stem cells in that they are solely dedicated to reproduction and transmit genetic information from generation to generation. Through the use of genetic techniques in Drosophila, Caenorhabditis elegans, and mouse, exciting progress has been made in understanding molecular mechanisms underlying interactions between stem cells and niches. The knowledge gained from studying GSCs has provided an intellectual framework for defining niches and molecular regulatory mechanisms for other adult stem cells. In most stem cell systems, one major GSC signaling pathway has been identified that relies on a signal provided by the niche and received by the germ cells. Mosaic analysis, tissue-specific gene expression assays, and transplantation experiments have determined the tissue dependence of the respective factors. Finally, knockout and overexpression or ectopic expression experiments have been used to examine whether a particular signaling pathway plays an instructive role. The major signaling pathways identified include BMP, JAK/STAT, Notch, and GDNF. More detailed analysis of these and additional signaling pathways has provided insight into intricate regulatory networks. As an increasing amount of information emerges, it is becoming clearer how a balance between self-renewal and differentiation is achieved at the molecular level. In the adult, niche signals, GSC-to-niche adhesion, GSC proliferation, and apoptosis are affected by both external and internal conditions. Diabetes and aging are associated with delay or complete failure in GSC self-renewal in both males and females. The effects of diabetes and aging can be overcome by the expression of niche factors, such as the ligands of the BMP and JAK/STAT pathways, the adhesion molecule E-cadherin, or the cell cycle regulator STRING, a Cdc25 homolog. Interestingly, with diabetes and aging the number of GSCs decreases less than expected, likely due to replacement of lost stem cells by symmetrical division, leading to clonal expansion of a subset of GSCs. If a similar process occurs in mammals, such a clonal expansion could contribute to the accumulation of genomic defects in the progeny of diabetes/aged parents.



***(D) Microtubular dynamics interfering agent causes apoptosis through Endoplasmic Reticulum stress pathway and sensitizes the breast and lung cancer cells through AP1 mediated TGFBI over- expression*.**

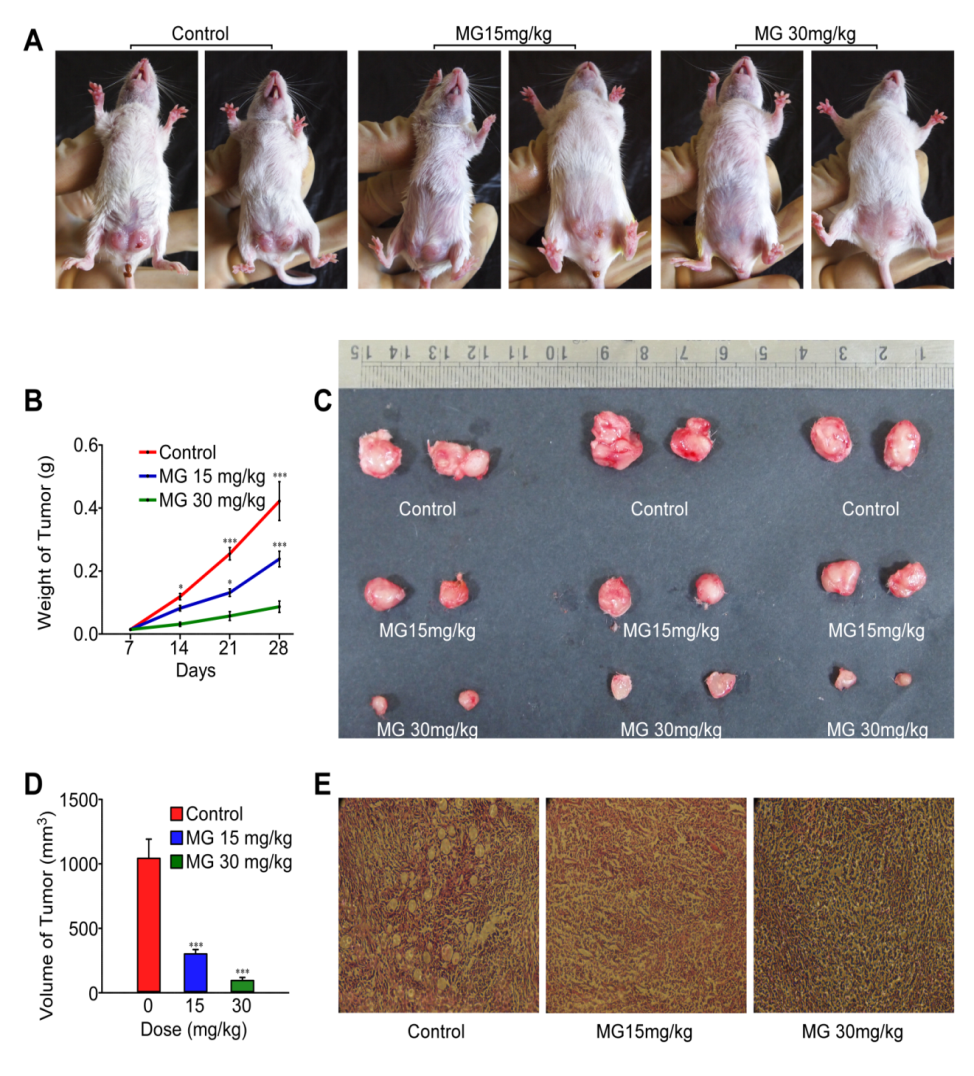
Microtubules play a critical role in the process of cell division, during which it helps in chromosomal segregation process. As cancer cells possess abnormal and uncontrolled cell division so targeting cell division through microtubule can be an important tool for cancer chemotherapy. We have identified a novel stilbene derivative (Z)-3-(3, 4-dimethoxyphenyl)-2-(3, 4, 5-trimethoxyphenyl) acrylonitrile (compound G) which causes microtubule depolymerization may leads to G2/M arrest. Several reports suggest that change in microtubular dynamics leads to cellular stress including endoplasmic reticulum stress. Microtubule dynamic interfering agents such as paclitaxel (taxol), docetaxel (taxotere), vinblastine, vincristine, nocodazole, and colchicines have been involved in activation of the c-Jun N-terminal kinase/stress-activated protein kinase (JNK/SAPK) signaling pathway in a variety of human cells actually requires interactions with microtubules. A like other microtubule dynamic interfering agents compound G also activates JNK/SAPK pathway. Preliminary experiment in compound G treated condition we have found higher expression level of CHOP/DDIT3/GADD153, an important biomarker for ER stress. ER stress has been reported to be linked with activation of JNK/SAPK pathway .Thus, compound G mediated activation of JNK/SAPK pathway may have link with ER stress. So, this project our first objective is to deduce whether the compound G mediated activation of JNK/SAPK is ER stress dependent or independent. Secondly, transforming growth factor-b-induced protein(TGFBI or keratoepithelin or βIg-h3), one of the ECM protein which has been implicated in a number of cellular disease processes including angiogenesis, tumor progression and metastasis has been found to be over-expressed at RNA and protein level in treated condition. TGFBI, a 68-kDa protein contains four conserved fasciclin-1 (FAS1) domains and a C-terminal RGD integrin-binding sequence. TGFBI mediates integrin binding to ECM proteins such as collagen, laminin and fibronectin. TGFBI binding to integrins has been related to the activation of cell proliferation, adhesion, migration and differentiation. This protein is up- regulated in human tumors of the colon, renal, pancreas, whereas it is down-regulated in breast cancer and lung cancer. TGFBI has conflicting roles in cancer progression. Depending on the tissue, TGFBI functions as an oncogene or tumor suppressor. Recent reports suggest TGFBI sensitizes ovarian cancers to paclitaxel via FAK- and Rho-dependent stabilization of microtubules by binding to integrin. Another interesting fact is that in benign to malignant breast cancer the expression of TGFBI decreases drastically and acts as a biomarker for breast cancer progression. So, now question arises does TGFBI over-expression sensitize the cancer cells to compound G through integrin-FAK pathway? If not what is the role of TGFBI over-expression in relation to treatment of compound G? How compound G induces the over expression of TGFBI happens? As reports suggests that JNK signaling pathway activates during TGFBI over-expression and in our system c-JUN part of an important transcription factor (AP1), is highly expressed in treated condition so our third objective is to establish the role of AP1 and JNK pathway in relation to TGFBI over-expression in compound G treated condition.

***(E) Oxidative stress plays major role in mediating apoptosis in filarial nematode Setaria cervi in the presence of trans-stilbene derivatives (In collaboration with Prof. S Sinhababu, Visva Bharati, Shantiniketan).***

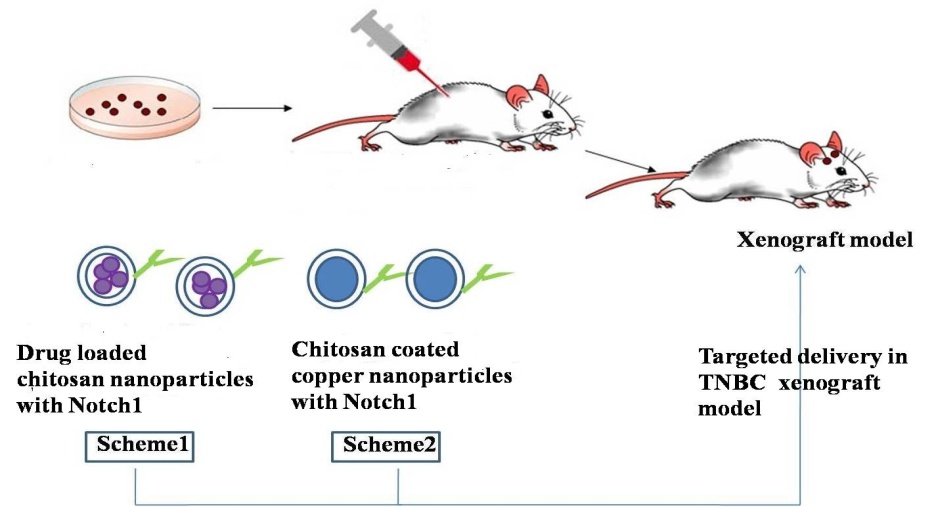
Lymphatic filariasis, affecting around 120 million people in 80 countries worldwide, is an extremely painful disease and caused permanent and long term disability. Owing to its alarming prevalence there is immediate need for development of new therapeutics. A series of *trans-*stilbene derivatives were synthesized using aqueous reaction condition showing potential as antifilarial agents demonstrated in vitro. MTT reduction assay and dye exclusion test were performed to evaluate the micro and macrofilaricidal potential of these compounds. Amid twenty *trans*-stilbene derivatives together with Resveratrol (RSV), a multifunctional natural product was screened; nine compounds have showed promising micro and macrofilaricidal activities and four of them showed better effectiveness than RSV. In the treated parasites apoptosis was established by DNA laddering, in situ DNA fragmentation and FACS analysis. The generation of ROS in the treated parasites was indicated by the depletion in the level of GSH,GR and GST activity and elevation of SOD, catalase, GPx activity and superoxide anion and H2O2 level. Along with the ROS generation and oxidative stress, the decreased expression of anti-apoptotic ced-9 gene and increased expression of nematode specific pro-apoptotic genes, egl-1, ced-4 and ced-3 at the level of transcription and translation level; the up-regulation of caspase-3 activity and involvement of caspase-8, 9, 3, cytochrome-c and PARP were also observed and which denotes the probable existence of both extrinsic and intrinsic pathways apoptosis in parasitic nematodes. This observation is reported first time and thus it confirms the mode of action and effectiveness of the *trans*-stilbene compounds.



**(F) *Induction of Mitochondrial Apoptotic Pathway in Triple Negative Breast Carcinoma Cells by Methylglyoxal via Generation of Reactive Oxygen Species(In collaboration with Prof Manju Ray, CSIR-Emeritus Scientist & Bhatnagar Awardees, Bose Institute, Kolkata)***

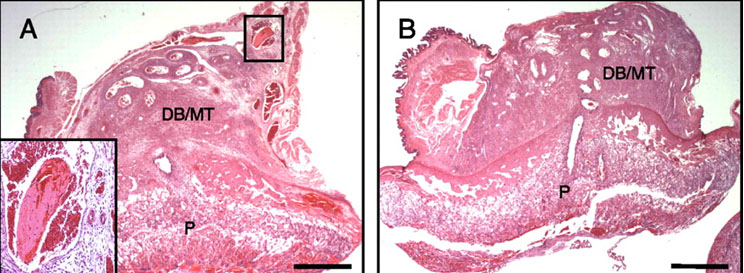
****Triple negative breast cancer (TNBC) tends to form aggressive tumors associated with high mortality and morbidity which urge the need for development of new therapeutic strategies. Recently the normal metabolite Methylglyoxal (MG) has been documented for its anti-proliferative activity against human breast cancer. However, the mode of action of MG against triple negative breast cancer remains open to question. In our study, we investigated the anticancer activity of MG in MDA MB 231 and 4T1 TNBC cell lines and elucidated the underlying mechanisms. MG dose-dependently caused cell death, induced apoptosis and generated ROS in both the TNBC cell lines. Furthermore, such effects were attenuated in presence of ROS scavenger N-Acetyl cysteine. MG triggered mitochondrial cytochrome c release in the cytosol and up-regulated Bax while down-regulated anti-apoptotic protein Bcl-2. Additionally, MG treatment down-regulated phospho-akt and inhibited the nuclear translocation of the p65 subunit of NF-κB. MG exhibited a tumor suppressive effect in BALB/c mouse 4T1 breast tumor model as well. The cytotoxic effect was studied using MTT assay. Apoptosis, ROS generation and mitochondrial dysfunction was evaluated by flow cytometry as well as fluorescence microscopy. Western blot assay was performed to analyse proteins responsible for apoptosis. This study demonstrated MG as a potent anticancer agent against TNBC both *in vitro* and *in vivo*. The findings will furnish fresh insights into the treatment of this subgroup of breast cancer.

***(G) Development of nanoparticle mediated treatment strategy to target NOTCH1 in triple negative breast cancer ( TNBC) Xenograft model (In collaboration with Prof. Parimal Karmakar, Jadavpur University).***

The tumors with estrogen negative, progesterone negative and HER2-negative are known as triple-negative (TN) tumors and account for about 15% of breast tumors. These cells have poor prognostic outcome compared with other types of breast cancer. Treatment of TNBC limited due to the lack of a therapeutic target and as a result, these cells are chemo-resistance. As a matter of fact, Notch signaling pathway also have great role for chemo-resistance. These signaling pathway is a conserved pathway that has been involved in the determination of cell fate and self-renewal of a variety of cancer cells. This possible link between Notch signaling and breast cancer was established and Notch-1 exert an influence in tumor metastasis and proliferation in vivo. It was also shown that the expression of Notch 1 was enriched in triple negative breast cancer cells, where as normal breast cells exhibited very low or no dateable noth1 expression. However, the increased expression of Notch-1 in triple negative breast cancer has been associated with malignant tumor behavior and poor prognosis.

To overcome this problem, nanoparticles may be used to deliver the drug in different cancer cells both in vitro and in vivo system. our focus is to delivery of drug loaded chitosan nanoparticles and copper coated chitosan nanoparticles tagged with Notch1 antibody for TNBC therapy. Due to its unique polymeric cationic character, chitosan has been extensively examined for the delivery system. We tried to load several anti-cancer drug such as doxorubicin and methoxtrate with chitosan nanoparticles or metal based nanoparticles such as Cu or Zn coated with chitosan. As mentioned before, there is very good strong correlation between the expression of Noth1 and TNBC. So, our target is to conjugate the Noth1 antibody with these nanoparticles. Initially, we examined the effect of these nanoparticles (both Noth1 conjugated or without Notch1 conjugated) on different sub type of breast cancer cells such as MDA-MB-231 (ER–, PR–, HER2–), MCF-7 (ER+, PR+/–, HER2–), BT-474 (ER+, PR+, HER2+), MDA-MB-453(ER–, PR–, HER2+). Next, we will try to focus on the exact molecular mechanism of cell death. The mouse models of human cancer are valuable tools for cancer research. So, we will also develop the TNBC xenograft model in immunodeficient mouse. The targeted drug or nanodrug delivery through chitosan nanoparticles tagged with Notch1 antibody will be therapeutic approach for treatment of chemo resistant triple negative breast cancer cells. Successful development of these kind of strategy for targeted delivery in TNBC xenograft model has tremendous application for overcome chemo-resistance of TNBC.

***(H) Exploration of molecular cross-talk involved in the roadmap to hyperhomocysteinemia- induced pregnancy loss in Polycystic Ovarian Syndrome (PCOS) (In collaboration with Prof. Baidyanath Chakravorty, Institute of Reproductive Medicine, Kolkata).***

 Women with pregnancy wastage encompass a wide range of phenotypes since list of candidate causes have grown rapidly in last few years. Obesity and insulin resistance (IR) remain as the most talked about features in pregnancy loss etiology. Recent evidence suggests a causal association between hyperhomocysteinemia (HHcy) and pregnancy loss; with mild-to-moderate degree of HHcy leads to a 3-fold increase in the risk of early pregnancy wastage. Moreover, over the last decade, evidence has accumulated to suggest that some cases of pregnancy wastage and later pregnancy complications are due to an exaggerated haemostatic response during pregnancy leading to placental thrombosis and infarction. Therefore, proper understanding of the underlying pathogenesis is an absolute necessity. Bone morphogenetic proteins (BMP) are the principal players in the maturation phase of angiogenesis. Recent reports have documented inhibition of neo-angiogenesis by BMP-9 and therefore have a greater possibility to contribute in the arena of pregnancy loss. The current project may cue towards the mechanism through which hyperhomocysteinemia brings about pregnancy loss which may bring the advent of newer drugs against it. Furthermore, to substantiate a cause and effect relationship between HHcy and pregnancy wastage elucidation of candidate genes associated with the disease are conducting by qRT-PCR and microarray studies to investigate global placental gene expression.

***7. Experience in Translational Animal Research works:***

***More than 20 years of experience in animal handling, breeding, experimentation and treatment as well as more than 50 International publications related to small and large animals. Worked with StAR and COX-2 knockout mouse as well as COX-2 over expressed Transgenic mouse in Garrison Institute on Aging, Texas Tech University of Health Sciences Centre, Texas, USA. Attended, participated and organized several rat and mouse Bio-methodology workshops.***

***I am established myself as a highly motivated and vibrant Animal Research Scientist as well as Molecular Reproductive Biologist at Div. of Mol. Medicine, Bose Institute, Kolkata. I have taken a pioneer role to establish the State of the Art and world class Centre for Translational Animal Research (CTAR), a Central Animal House and Research Facility in North Eastern Part of India and did innovative research in the areas of Modern Laboratory Animal Sciences. I have a significant and unique experience in the maintenance and management of knockout/transgenic & Xenograft mouse facility. I am well versed with the L***[***aws***](http://en.wikipedia.org/wiki/Laws) ***or*** [***guidelines***](http://en.wikipedia.org/wiki/Guideline) ***that permit and control the use of non-human animals for*** [***scientific experimentation***](http://en.wikipedia.org/wiki/Scientific_experimentation)***. I worked several years in USA in close association with Jackson Laboratories. I was participated in several seminars, workshops related with the experiments on animals and drug testing. I am the member in several National & International Committees related with animal welfare. I am well versed with the Guide for the Care and Use of Laboratory Animals in Bio-medical Research in USA, UK, and Canada etc. I am strongly with the activities of CPCSEA, AAALAC, CCAC, FELASA, ASPA, & ICLAS etc. I have more than 20 years of experience on Standard Operating Procedures (SOP) for Institutional Animal Ethics Committee (IAEC) of the CPCSEA.***

***8. Development of the Animal Research Facility at Bose Institute.***

I am serving as a Member Secretary/Scientist- In-Charge, Central Animal House & Research Facility as well as in "**Institutional Animal Ethics Committee (IAEC)"** & takes active participation to establish and maintenance of “**Central Animal House & Research Facility"** at Bose Institute.This is a *state of the art* world class environmentally controlled "Central Animal House and Research Facility” for Developmental and Toxicology Research" with all facilities for breeding, maintenance, experimentation on small laboratory animals and it has been exclusively developed in a plinth area of 15,000 sq. ft. This Animal facility will be utilized for experimental research in accordance with the principles of good laboratory practices and CPCSEA, Ministry of Environment, Forest & Climate Change, Government of India guidelines. Further, it facilitates research and development in partnership with academic Institutions, Industries and funding agencies for drug discovery-cum-validation and devices of translational medical research with the basic objective of advancement of biological knowledge which is useful for saving the lives and /or alleviating the suffering of human being, animals and plants. However such experiments are performed with due care so as to minimize the pain inflicted on animals. The Centre is also involved in skilled manpower development through education and training in laboratory animal care and experimental techniques. The main objective of the Centre is to supply defined strains of laboratory animals like mice, rats, guinea pigs, hamsters and rabbits for Bio-medical Research to the scientific community of the Eastern and North eastern part of India.

**Future Planning:** In view of global competitiveness, there is an urgent as well as strong need to synthesise molecules as new chemical entities which may be considered for IPR protections provided data on these entities can be generated in specific genetically engineered strains, species and animal models for disease like diabetes, hyperlipidaemia, hypertension, immunodeficiency and cancer etc. It becomes crucial for the laboratories to develop facilities where these activities are thoroughly evaluated and labs are able to provide data, which is acceptable to regulatory authorities. Unless we able to get these opportunities within the Country, it would be extremely difficult for the Scientists as well as institutions to obtain global marketing rights for drugs**. Hence, it is an utmost need to set up here a state-of-the-art well-equipped transgenic/ knockout/ Xenograft mouse laboratory (Centre for Translational Animal Research) for the Scientists of Eastern & North Eastern part of India. (A MOU already signed with TATA Medical Centre, Kolkata to develop this kind of facility with the help of DBT funding)**

***9. Collaborators:***

***1. Prof. Sanghamitra Raha, Visva Bharati, Santiniketan, India.***

***2. Prof. Parimal C. Sen, Bose Institute, Kolkata***

***3. Prof. Sujoy Guha, Indian Institute of Technology, Kharagpur***

***4. Prof. Narayan Jana, Institute of Post-Graduate Medical Education & Research, Kolkata***

***5. Prof. Parimal Karmakar, Dept. of Life Sciences & Biotech, Jadavpur University, Kolkata.***

***6. Prof. Baidyanath Chakraborty, Institute of Reproductive Medicine, Kolkata.***

***7. Dr. Debasish Bandopadhyay, University of Calcutta, Kolkata.***

***8. Prof. Manikuntala Kundu and Prof. Manju Ray, Bose Institute, Kolkata***

***9. Dr. Anup Kr. Misra & Dr. Kaushik Biswas, Bose Institute, Kolkata.***

***10. Awards/Honor Received:***

1. ***The Biography included in Americas “Who’s Who in Medicine & Healthcare”, 2008-2009.***
2. ***DST-Fast-Track Young Scientist Scheme Award, 2010.***
3. ***Young Scientist Travel Award, 2010 from ACC&D (Portland, Oregon, U.S.A.)***
4. ***Young Investigator Award, 2011 from Parsemus Foundation (San Francisco, California, USA).***
5. ***Nominated as “International Scientist of the Year, 2011” by International Biographical Centre, Cambridge, U.K.***
6. ***Scientific Award of Excellence for 2011" by American Biographical Institute, San Francisco, California, U.S.A.***
7. ***Young Scientist Travel Award, 2012, from International Congress on Animal Reproduction (ICAR)”, Vancouver, British Columbia, Canada.***
8. ***Young Scientist (Clinical) Award-2012” from Parsemus Foundation, Berkley, California, U.S.A.***
9. ***Selected and Trained as an Expert Consultant of CPCSEA, Ministry of Environment & Forests, Govt. of India.***
10. ***Young Scientist Travel Award, 2013 from ACC&D (Portland, Oregon, U.S.A.)***

***11. Young Scientist Award, 2014 from Parsemus Foundation, Berkley, California, U.S.A.***

***12. Outstanding Scientist Award, 2015 from Venus International Foundation, India.***

***11. Editors/Editorial Board:***

1. ***Translational Medicine***
2. ***International Journal of Plant Physiology & Biochemistry***
3. ***Journal of Diabetes & Endocrinology***
4. ***International Journal of Clinical Therapeutics and Diagnosis (IJCTD)***
5. ***American Journal of Medical & Biological Research (AJMBR)***
6. ***Journal of Cell and Molecular Biology Research (JCMBR)***
7. ***Signpost Open Access Journal Of Animal Reproduction Studies***
8. ***Journal of Translational Medicine and Epidemiology.***
9. ***Journal of Preventive Medicine***
10. ***Reproductive Biology Insights***
11. ***Journal of Preventive Medicine***
12. ***Journal of Cancer Research and Molecular Medicine***
13. ***Peak Journal of Medicine and Medical Sciences***
14. ***Annals of Translational Medicine & Epidemiology***
15. ***Frontiers in Endocrinology***
16. ***Journal of Collaborative Heath Care & Translational Medicine***
17. ***International* Journal *of Clinical Endocrinology and Metabolism***
18. ***International Journal of Obstetrics and Gynecology***
19. ***Advances in Applied Physiology***
20. ***Austin Endocrinology & Diabetes case Reports.***

***12. Reviewer:***

**1. Journal of Endocrinology/Endocrinology 2. Molecular Cellular Endocrinology 3.Toxicological Sciences 4. Andrology 5. Asian Journal of Andrology 6. Food & Chemical Toxicology 7. Ecotoxicology & Environmental Safety 8. Fertility & Sterility 9. Contraception 10. Reproductive Toxicology 11. Molecular Cellular Biochemistry 12. Molecular Reproduction & Development 13. Journal of Trace Elements in Medicine & Biology 14. Molecular Human Reproduction 15. Life Sciences and Reproduction etc.**

***13. Member of Scientific Bodies:***

|  |
| --- |
| **1. The Science Advisory Board, U.S.A.** |

**2. Society for the Study of Reproduction and Fertility**

**3. Society for Reproductive Biology and Comparative Endocrinology.**

**4. The Society of Physiology.**

**5. Indian Society for Translational Research**

**6. DNA Society of India**

***14. Ph.D. guidance*:**

**Two are already awarded and Four students (SRFs) have already been registered under the Ph.D. Programme in the University of Calcutta where I am acting as a Supervisor/Co-supervisor. Three JRFs will be registered soon in different Departments of Calcutta University. One DBT-RA and one CSIR RA are also working under my supervision.**

***15. Teaching activities:***

**Taught and taken special paper classes in Biochemistry (Cell signalling, hormone action, cancer signalling and Immunology) and Environmental Science (Toxicology and pharmacology, Reproductive and endocrine disorders etc.) of M.Sc. (Physiology) students of University of Calcutta as well as Integrated M.Sc.-Ph.D. classes at Bose Institute.**

***16. CPCSEA, Ministry of Environment, Forest & Climate Change, Govt. Of India Nominee:***

***Serving as a "CPCSEA Nominee" in different Institutes like Visva Bharati (a Central University), Chittaranjan National Cancer Institute, Indian Institute of Chemical Biology (IICB), Vidyasagar University, GN Inst. of Pharmaceutical & Dental Sciences, Institute of Reproductive Medicine, IQ Medical College, Cris Biotech etc.***

***17. List of Extramural Funded Projects:***

*1. SERC Fast Track Proposals for Young Scientist Research Scheme Entitled " Molecular Signalling Involved in patho-physiological Action of Common Air Pollutant Benzo(a)pyrene in Testicular DNA Damage and Apoptosis in Connection to Male Infertility: Possible Protection by Natural Aryl Hydrocarbon Receptor antagonists" from DST, Govt. of India. (PI: Dr. Kuladip Jana). (Completed on 31st March, 2015)*

*2. “Targeted delivery of nano-conjugated Methylglyoxal to cancer cells and understanding the bioenergetics difference between normal and malignant cells at molecular level” from DST, Govt. of India. (PI: Prof. Manju Ray; Co-PI: Dr. Kuladip Jana), (Project started from 01 January, 2014 with an appointment of a JRF and a R.A.)*

*3. “Assessment of the anti-cancer effect of Methylglyoxal in combination with conventional anti-cancer drugs at metronomic doses with special reference to cancer stem cells” from CSIR, Govt. of India (PI: Prof. Manju Ray; Co-PI: Dr. Kuladip Jana), (Project funded on 10/05/2016).*

***18. Attended National & International Conferences****:*

*Attended several National and International conferences and delivered lectures and chaired sessions.*

19. List of Important research publications:

1. Roy A, Ahir M, Bhattacharya S, Parida PK, Adhikary A, Jana K, Ray M. [Induction of Mitochondrial Apoptotic Pathway in Triple Negative Breast Carcinoma Cells by Methylglyoxal via Generation of Reactive Oxygen Species.](https://www.ncbi.nlm.nih.gov/pubmed/28418078) **Molecular Carcinogenesis** 2017 Apr 18. doi: 10.1002/mc.22665.
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