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

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

*1.2 Designation and Department/Division:*

***Principal Scientist***

***Division of Molecular Medicine***

***Scientist In-Charge, Centre for Translational Animal Research (CTAR)***

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

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***Cell: (+91)-9007042850/9007067720***

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

***Website: http://www.jcbose.ac.in/bose/faculty-details/kuladip-jana***

*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.***

***2006 Post-doctorate in Molecular Reproductive Medicine from Garrison Institute of Aging, Texas Tech University of Health Sciences Centre, Texas, USA.***

***2022 Pursuing D.Sc. in Science from Vidyasagar University, Midnapur, West Bengal., India***

***Professional Course: M.S. in Psychotherapy & Counselling from Institute of Psychotherapy& Management Sciences, Mumbai, 2005.***

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

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

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

***2009 Research Associate III, Crystallography & Molecular Biology Division, Saha Institute of Nuclear Physics, Kolkata.***

***2010 Scientist-C, Division of Molecular Medicine, Bose Institute, Kolkata, India.***

***2014 Senior Scientist, Division of Molecular Medicine, Bose Institute, Kolkata, India.***

***2018 Principal Scientist, Division of Molecular Medicine, Bose Institute, Kolkata, India.***

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

***Molecular & Cellular Endocrinology, Reproductive Medicine, Cancer Biology, Drug development against Cancer, Diabetes, Filaria, Tuberculosis and Leismania and Use of Bio-Polymer in Tissue Engineering as well as Nano-drug development.***

*As we know the multidrug resistance (MDR) is a major obstacle and it severely limits the efficacy of clinical chemotherapy in the treatment of cancer. We also identified some small molecules those have an anti-proliferation and anti-metastasis potential against doxorubicin (DOX) resistant breast cancer cell lines through the induction of apoptosis/autophagy. A novel sulforaphane derivative also identified which showed very promising anti-tumor potential which intermingle with IGF1R and block the PI3K/Akt pathway along with the induction of caspase independent apoptosis. Recently we started to successful implementation of CRISPR-Cas9 or conventional genome editing of cancer cell lines to determine the key players involved in disease pathogenesis with a long-term goal of drug designing. I have a vast expertise in working with different human disease models including cancer, diabetes and infectious diseases in mice, rat & hamsters. I intend to develop and implement successful genome editing in cancer cell lines using CRISPR-Cas9 with a long-term goal of using this tool in investigating the mechanism of existing drug resistance among different cancers. I am also keen in identification and characterization of biologically relevant pathways/components involved in disease pathology in response to parasite infection and would like to contribute in the field of drug development. Efficient identification and characterization of these components should serve as the basis of identifying novel inhibitors or re-purpose existing drugs in a rational drug designing approach for treatment of multidrug drug resistant cancers and parasitic infections.*

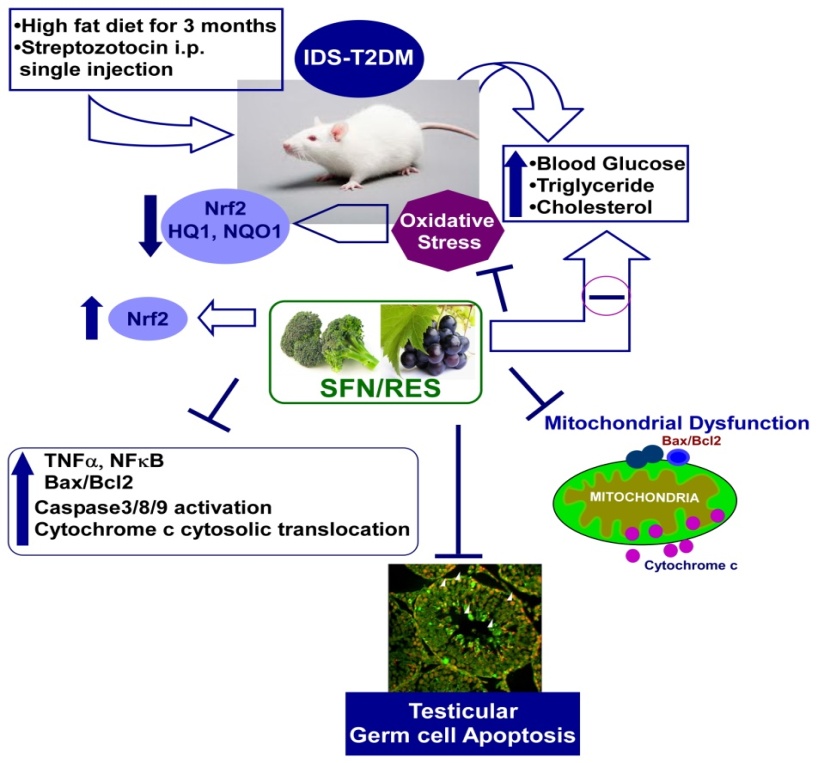
# *Presently, my laboratory focusing on Translational animal research in the diverse directions i.e. Molecular signalling of germ cell/ Leydig cell apoptosis by Benzo(a)pyrene and its protection by natural aryl hydrocarbon receptor (AhR) antagonist, Molecular Mechanisms of Germ-line Stem Cell Regulation: 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 Wntsignalling, 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, 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 officinalisin diabetic condition.*

***6. Research interest:***

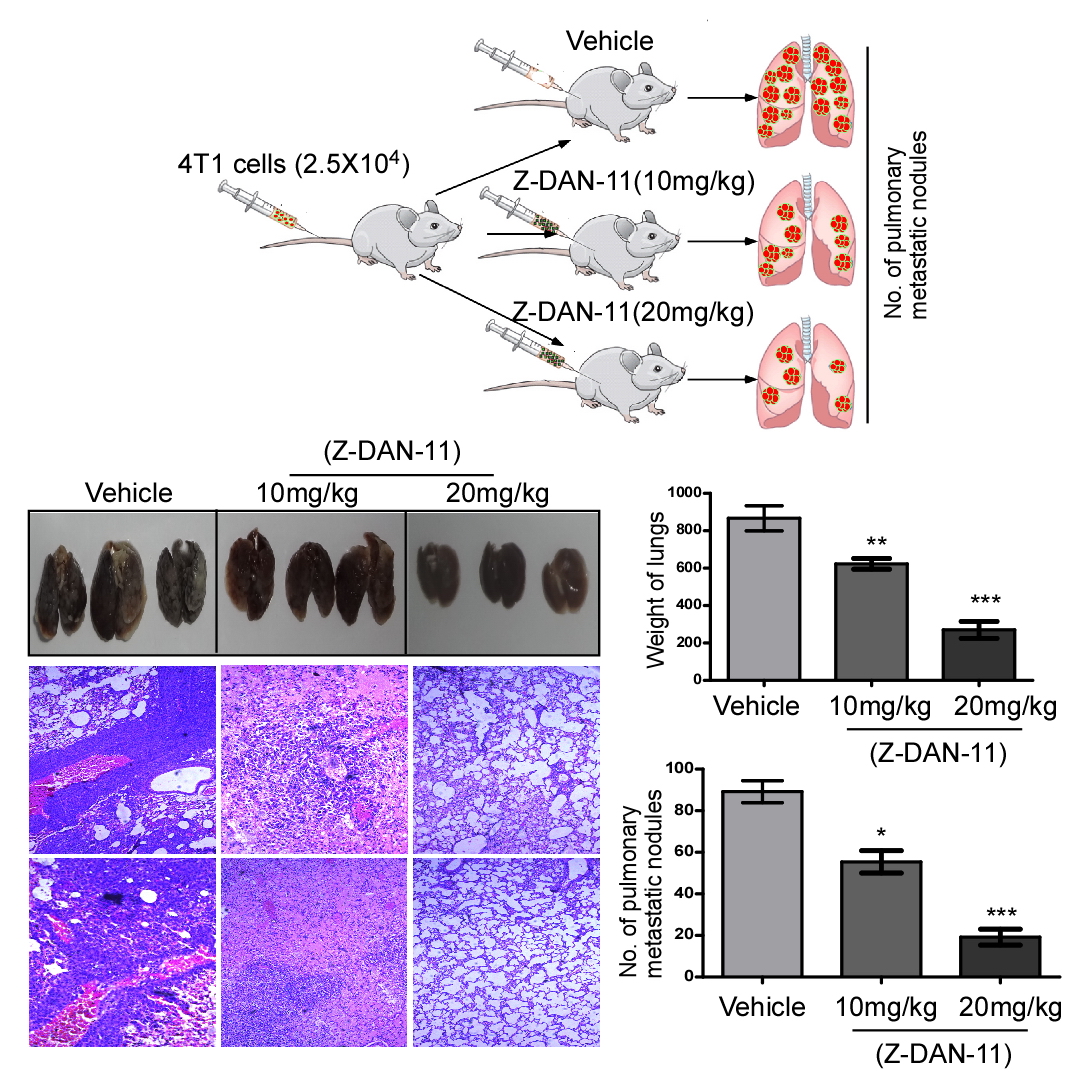
***(A) Molecular signaling involved in patho-physiological action of common air pollutant “Benzo(a)pyrene” in Germ cell DNA damage and apoptosis in connection to male infertility: possible protection by natural aryl hydrocarbon receptor antagonists***

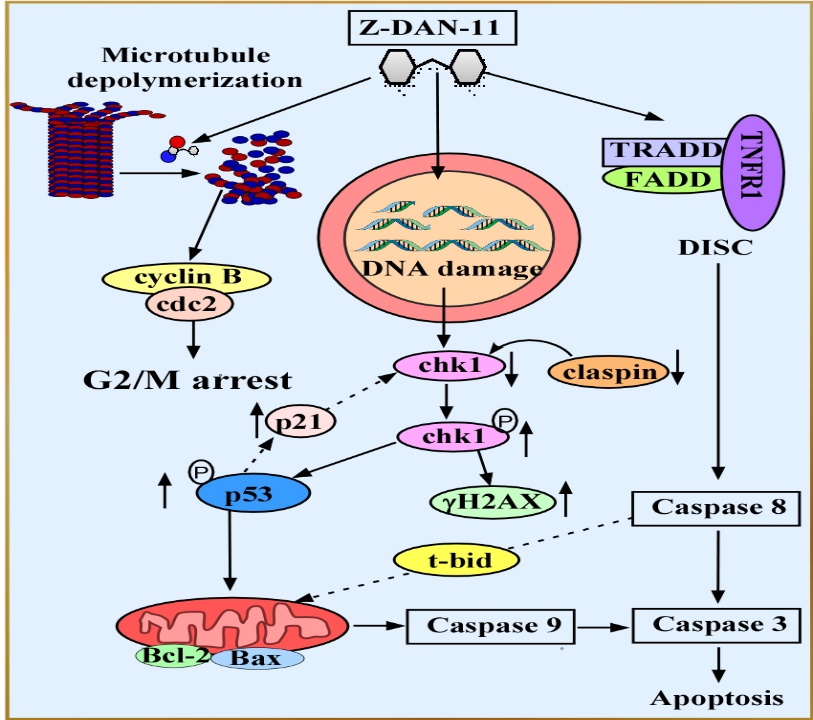
Benzo(a)pyrene (B(a)P) is an environmental toxicant that alters the steroidogenic profile of testis and induces testicular dysfunctions. In spite of the magnitude of the problem, the precise molecular and cellular mechanism of B(a)P mediated testicular damage and possible protective measures still remain unexplored. In the present study, we have investigated the molecular signalling 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) Insulin defective stage of type 2 Diabetes (IDS-T2DM) induced testicular Germ cell death/apoptosis in association with the up-regulation of Nrf2 expression: Ameliorative potential of sulforaphane (SFN) and resveratrol (RES)***

Diabetes-induced testicular celldeath is due predominantly to oxidative stress. Nuclear factor (erythroid-derived 2)-like 2 (Nrf2) is an important transcription factor incontrolling the anti-oxidative system and is inducible by sulforaphane(SFN) and resveratrol (RES). To test whether SFN/RES prevents diabetes-induced testicular germ celldeath/apoptosis, an insulin-defective stage of type 2 diabetes (IDS-T2DM) wasinduced in mice. This was accomplished by feeding them a high-fatdiet (HFD) for 3 months to induce insulin resistance and then giving oneintraperitoneal injection of streptozotocin to induce hyperglycemiawhile age-matched control mice were fed a normal diet (ND). IDS-T2DMand ND-fed control mice were then further subdivided intothose with or without 3-months SFN/RES treatment. IDS-T2DM inducedsignificant increases in testicular germ cell death/apoptosis presumably through receptorand mitochondrial pathways, shown by increased ratio ofBax/Bcl2 expression and cleavage of caspase-3 and caspase-8 withoutsignificant change of endoplasmic reticulum stress (GRP78/CHOP). Diabetes alsosignificantly increased testicular oxidative damage and inflammation (TNFα and NFκB).All of these diabetic effects were significantly prevented by SFN/REStreatment with up-regulated Nrf2 expression. These results suggest thatIDS-T2DM induces testicular germ cell death/apoptosis presumably throughcaspase-8 activation and mitochondria-mediated cell death pathwaysand also by significantly down-regulating testicular Nrf2 expressionand function. SFN/RES up-regulates testicular Nrf2 expression and its targetantioxidant expression, which was associated with significant protectionof the testis from IDS-T2DM-induced germ cell death.

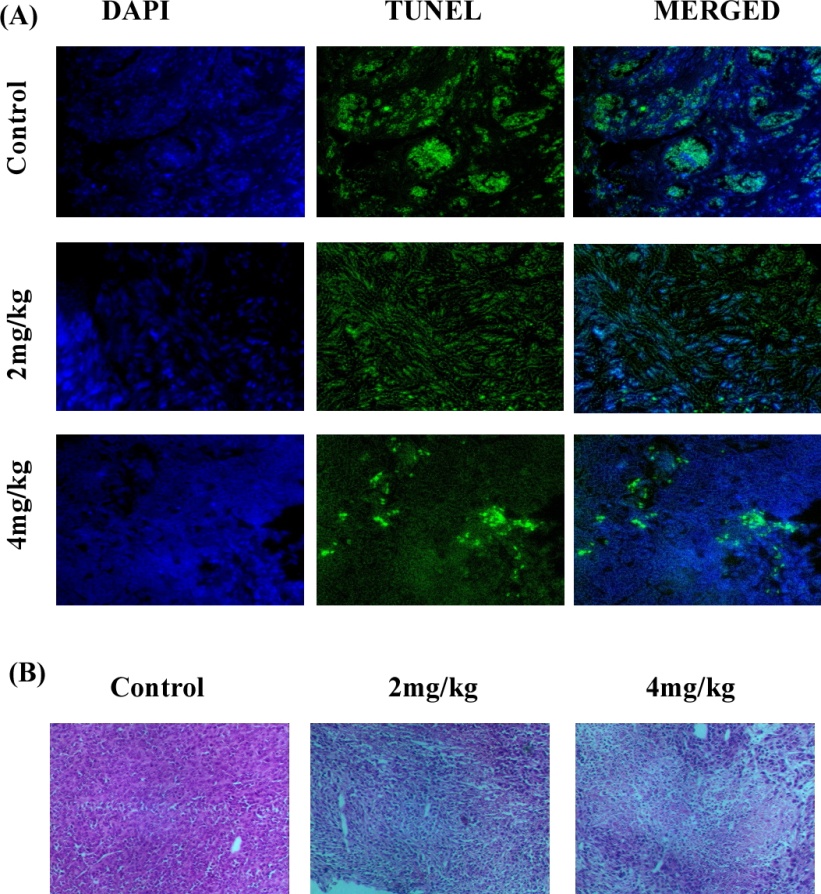
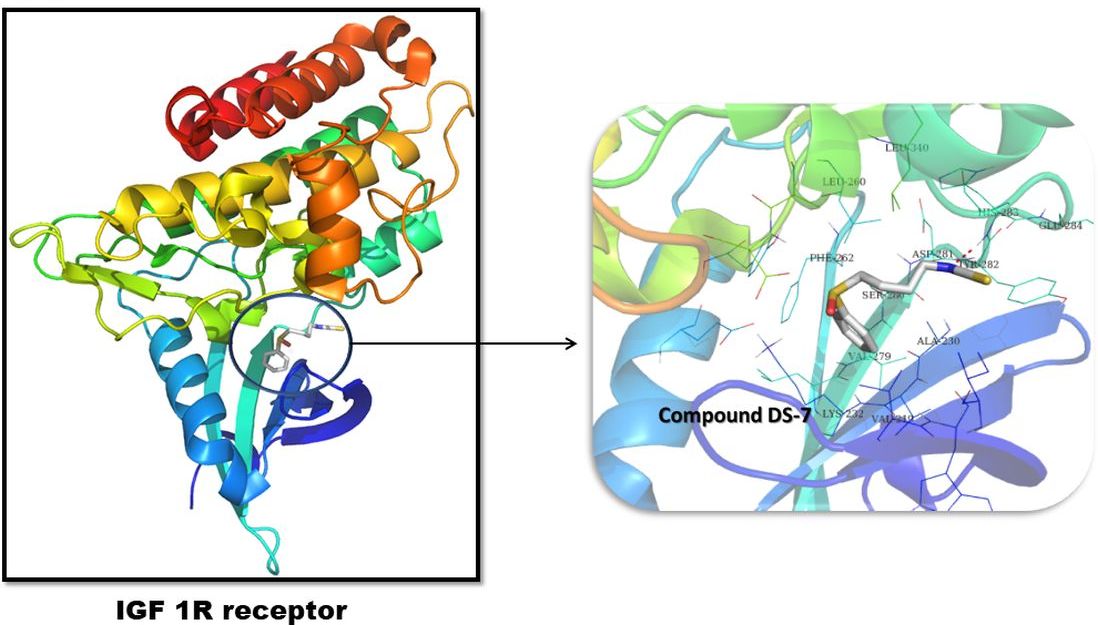
***(C) A microtubular dynamics interfering trans-stilbene derivative compound G drives G2/M arrest, apoptosis and impedes cancer progression.***

Resveratrol, a *trans* –stilbene polyphenolic compound and its synthetic analogs have been widely used bioactive molecules due to their remarkable chemopreventive potential. Here, we have identified a novel resveratrol analog, compound G ((Z)-3-(3, 4-dimethoxyphenyl)-2-(3, 4, 5-trimethoxyphenyl) acrylonitrile (Z-DAN-11)which inhibits proliferation of several cancer cell lines*in vitro*through microtubule depolymerization that induced G2Marrest and consequently leads to apoptotic cell death. Importantly, compound G shows limited cytotoxicity to normal cells as compared to cancer cells. Moreover, insight into the molecular and mechanistic detailed studies we reasoned that compound G induces increase in the expression of pro-apoptotic proteins and decrease in the expression anti-apoptotic proteins that decisively helps the activation of caspase 8, caspase 9, caspase 3, leading to PARP-1 and cell death via intrinsic and extrinsic pathways of apoptosis. More importantly, we also have established the crucial contribution of tumor suppressor protein p53 in compound G mediated apoptosis. Interestingly, the compound G also imparts its anti tumorigenic effect by inhibiting clonogenic property and anchorage independent growth potential of cancer cells. Finally, *in vivo* study with immune-competent syngeneic mice tumor model shows thatadministration of compound G is able to impede tumor progression without any side effects. So, our presently studied novel *trans*-stilbene derivative compound G has tremendous anti-tumorigenic potential and can be added to the current regimes of chemotherapy.

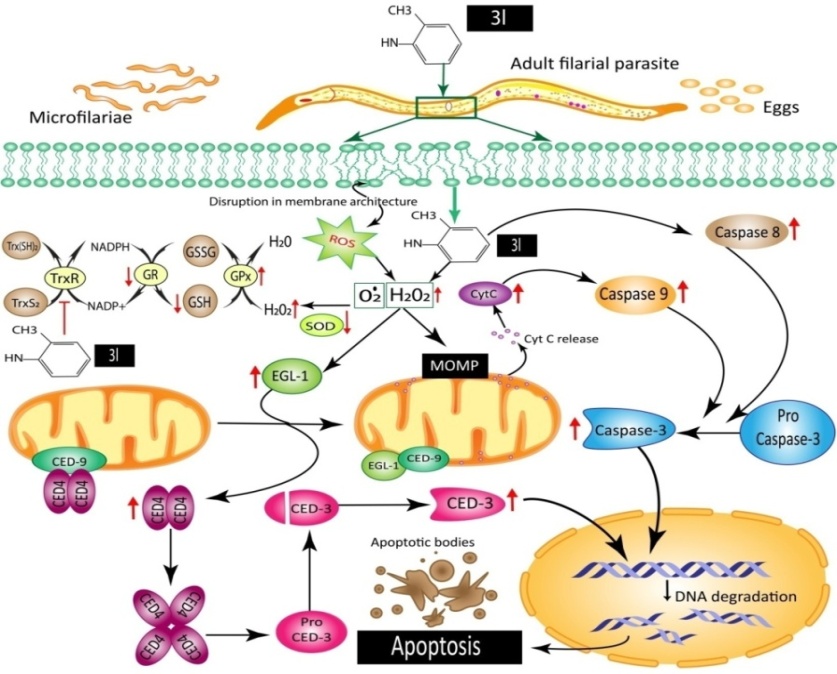


**ROS**

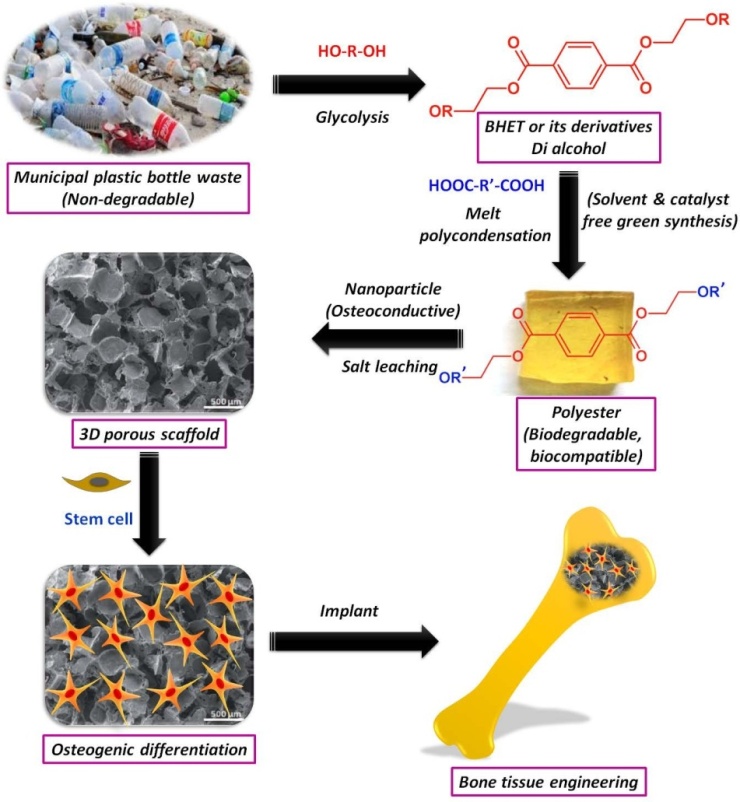
***(D) A novel sulforaphane derivative impedes cancer cell growth progression through G2/M arrest and ROS mediated caspase independent apoptosis.***

An array of suforaphane derivatives were synthesized and evaluated for their cytotoxic potentials on various murine and human malignant cell lines. One such derivative was effectively opt for out as most potent with high cytotoxic effect against cancer cells and less toxicity against normal cell lines. Our present study established ROS induced mitochondrial dysfunction and finally apoptosis induction by the lead compound. The inhibition of cell propagation was linked to the data confirming G2/M phase arrest. The mitochondrial dysfunction was confirmed by the Bax/Bcl-2 ratio. The lead compound suppressed the NRF-2 protein expression thus increasing the free radicals in the tumor cells. The derivative induced a ROS mediated caspase-independent apoptosis as the western blot data confirmed no caspase activation. The compound showed to intermingle with the IGF1R and block the PI3K/Akt pathway. The augmentation inhibitory outcome of the lead compound was confirmed by the 4T1 injected Balb/c tumor model.

**(E)Evaluation of anti-filarial efficacy of the novel piperine derivative through disruption of redox homeostasis with synchronized activation of apoptosis.**

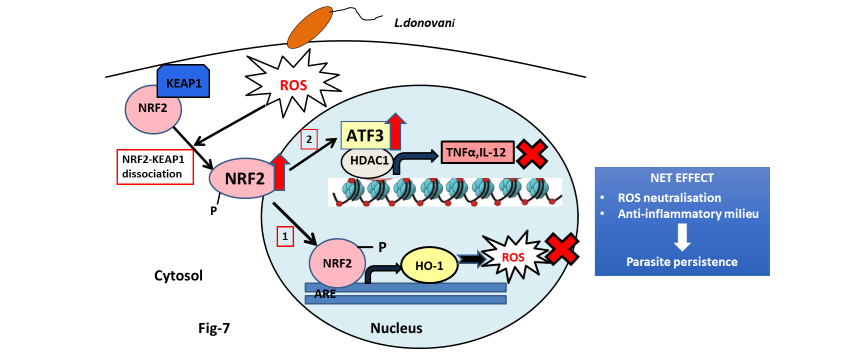
A series of novel piperine derivatives were synthesized and were evaluated for its antifilarial potential against the bovine filarial parasite *Setaria cervi*. One of the piperine derivative (3l**)**showed the highest efficacyamong the selected other compounds. The antifilarial activities its exhibited were clearly fuelled through disparity of the internal redox homeostasis as evidenced from the alterations in the enzymatic and non-enzymatic antioxidants level which ultimately shifted towards activation of pro-apoptotic signaling cascade eventually leading to the death of the parasites. The ability of the compound **3l** to bind thioredoxin reductase (TrxR) and CED-3 protein are the key findings of this study. The present study supported with several biological experiments is therefore a maiden report on the antifilarial effectiveness of these novel piperine derivatives.

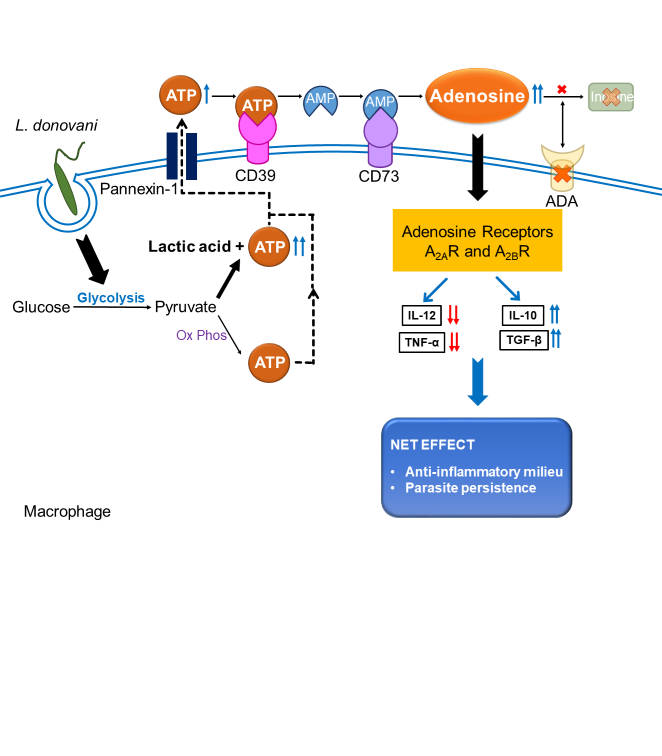
***(F) Plastic Waste Derived Biodegradable and Biocompatible Polyester for Bone Tissue Engineering (In collaboration with Dr. K. Sarkar, Dept. of Polymer Sciences, Univ. Of Calcutta)***

It is widely believed that engineered tissues could revolutionize medicine and offer promising  
new treatments for many debilitating diseases. In tissue engineering, a scaffold should ideally be  
non-toxic, biodegradable, and highly porous with interconnected architecture to support cell  
attachment, proliferation and extra-cellular matrix (ECM) production to ultimately facilitate  
tissue generation. In addition, it should possess optimal mechanical and physical properties. The  
currently available library of such materials is limited and expensive adding to the already high  
costs of these emerging therapies. There is a need for biomedical polymers derived from low  
cost, renewable sources and recycled waste that could offer cost effective solutions.  
The use of polyethylene terephthalate (PET) has greatly increased because of its widespread  
application in packaging of food and drinks. This has resulted in an ever-growing volume of post-  
consumer municipal PET waste. Therefore, recycling of waste PET has become a significant global  
challenge. PET can be recycled by various techniques such as glycolysis, aminolysis and hydrolysis  
to get useful monomer such as bis-hydroxyethylene terephthalate (BHET) or its derivatives which  
may be used as one of the good source of monomer for the synthesis of polyester. Therefore, a  
large volume of PET municipal waste may be a potential source of low cost polymers. The  
hypothesis of the proposed proposal consists of the following points (Scheme 1):

* Recycling of municipal plastic PET waste by glycolysis technique to get BHET or its  
  derivatives.
* Synthesis of a library of biodegradable and biocompatible polyester by solvent and  
  catalyst free melt polycondensation technique by using recycled BHET and its derivatives.
* Fabrication of 3D porous scaffold by salt leaching technique containing inorganic  
  nanoparticle as osteoconductive and reinforcing material.
* In vitro biocompatibility and bone formation study by seeding human mesenchymal stem  
  cell (hMSC).
* In vivo implantation for bone formation.

***(G) Leishmania donovani targets host transcription factor NRF2 to activate anti-oxidant enzyme HO-1 and transcriptional repressor ATF3 for establishing infection (In collaboration with Dr. A. Ukil, Dept. of Biochemistry, University of Calcutta)***



In order to gain the knowledge about exploitation of host defence by infectious organisms, we selected a model macrophage associated disease, visceral leishmaniasis. Macrophages play an important role in the first line of defense against various pathogens through generation of ROS and production of various pro-inflammatory cytokines. Thus, they serve as a very powerful guard protecting our body from pathogens. *Leishmania donovani*, the causative agent of fatal visceral leishmaniasis, is an obligate intracellular parasite inoculated into mammals by the bite of its vector sand fly. Once transmitted to the host, the infective promastigote forms are rapidly phagocytosed by these macrophages. Within these cells, the parasite along with combating against the anti-microbial defence arsenals of the macrophages has to survive within the harsh environment of the phagolysosomes. Thus, the parasite upon infecting a macrophage must subvert or impair the defence arsenals in order to successfully survive and propagate infection.Generation of Reactive oxygen species (ROS) is one of the primary anti-microbial activities of a macrophage, when it encounters a pathogen. ROS can even ultimately result in apoptosis of the host harboring the pathogen, and thereby results into parasite clearance. Thus, the parasite has to suppress production of reactive oxygen species. Another important defence arsenal put forward by an infected macrophage is both membrane and cytosolic receptor mediated pro-inflammatory responses. The initial innate immune response of generation of pro-inflammatory cytokines like IL-12 and TNF-α may recruit inflammatory cells and T cells necessary for resolution of infection.In order to succeed against host cell, *Leishmania* requires substantially high energy. Now, as the intracellular parasite andthe host cell utilise the same host energy sources, therefore knowledge of metabolic status of infected macrophage leading to energy production i.e. ATP generation, also seems extremely important to address molecular signalling of infection. Adenosine 5’-triphosphate (ATP), the universal energy currency of a cell, fuels for variousvital processes necessary for maintenance of a cell and also act as an intracellular andextracellular messenger. The main source of ATP is the catabolic part ofcarbohydrate metabolism where glycolysis is followed by TCA cycle and oxidative phosphorylation. ATP generated within a cell, apart from acting as a source of energy, may act as a signalling molecule both intra-cellularly and even extracellularly.Various stimuli like stress, hypoxia, inflammation and ROS are found to induce ATP release from cells with the help of various ATP release channels and even via vesicular exocytosis in a non-lytic mechanism.Released extracellular ATP (eATP) often act as a signalling molecule and regulate various cellular processes. Thus, extracellular ATP concentration is also very crucial. High extracellular ATP acting as a ‘danger signal’ can even promote inflammation through the P2X7-receptor dependent activation of the NLRP3 inflammasome, leading to production of pro-inflammatory cytokine IL-1β. So, ATP level in the extracellular environmentis usually maintained with the help of various ATP hydrolysing enzymes of macrophage like ecto-apyrase, pyrophosphatases, phosphodiesterases and ecto-adenosine triphosphatases (eATPase). So, *Leishmania* must be utilising these enzymes to maintain eATP concentration. Moreover, after evasion of first line of defence, parasite induces the host to generate immunosuppressive molecules, which favor their intracellular survival. Since apoptosis is the last resort for an infected cellto get rid of pathogens, *Leishmania* finally have to inhibit host cell apoptosis to ensure their persistent survival.

***Translational Animal Research works:***

***More than 20 years of experience in animal handling, breeding, experimentation 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 and Endocrinologist as well as Cancer 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 onanimals 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 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 **Scientist- In-Charge, *Centre for Translational Animal Research (CTAR)***as well as in "**Institutional Animal Ethics Committee (IAEC)"**&takes prime role to establish and maintenance of “**Central Animal House & Research Facility"(CTAR)** 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:**

* ***Maintenance of Knockout mouse, Xenograft mouse for biological research***
* ***Maintenance of NOG (NOD/Shi-Scid/IL-2Rγnull)/NSG (NOD.Cg-Prkdcscid IL2rgtm1Wjl/SzJ) Mouse***



* ***Humanized mouse model will be maintained for biological & medical research for human therapeutics***

**The CTAR maintains animal under pathogen-free conditions**

***9. Collaborators:***

***1. Prof. Santi P. Sinhababu, Visva Bharati, Santiniketan***

***2. Prof. Jayati Basu & Manikuntala Kundu, Bose Institute***

***3. Prof. ParimalKarmakar, Jadavpur University.***

***4. Prof. Baidyanath Chakraborty, Institute of Reproductive Medicine***

***5. Prof. Gaurisankar Sa,Bose Institute***

***6. Prof. Tanya Das, Bose Institute***

***7. Prof. Chittaranjan Sinha, Jadavpur University***

***8. Prof. Debasish Das, University of Calcutta***

***9. Prof. Anup Kumar Misra, Bose Institute***

***10. Prof. Debapratim Das, IIT, Guwahati***

***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. ***Selected and Trained as a CPCSEA Nominee, Ministry of Environment & Forests, Govt. of India.***
9. ***Young Scientist Travel Award, 2013 from ACC&D (Portland, Oregon, U.S.A.)***

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

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

***12. Bharat Bikas Award, 2017 for outstanding contribution in Molecular Medicines.***

***13. Rastriya Gaurav Award, 2018***

***14. Fellow of “Scholars Academic and Scientific Society” (FSASS).***

***15. Honorary Rosalind Member of London Journal Press***

***16. Honorary Member, Society for Redox Biology & Medicine, USA***

***17. Fellow of Reproduction & Endocrinology (FRE) from SRBCE***

***18. Fellow of National Environmental Science Academy (FNESA)***

***19. Outstanding Scientist 2022, INSO International Scientist Award on Engineering, Science & Medicines.***

***20. Eminent Scientist of the Year Award 2022 from National Environmental Science Academy***

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

1. ***Translational Medicine***
2. ***Journal of Translational Medicine and Epidemiology.***
3. ***Current Medicine***
4. ***Journal of Cancer Research and Molecular Medicine***
5. ***Annals of Translational Medicine & Epidemiology***
6. ***Frontiers in Endocrinology***
7. ***International Journal of Obstetrics and Gynecology***
8. ***Austin Endocrinology & Diabetes case Reports.***
9. ***American Journal of Medical & biological research***
10. ***Journal of Molecular Genetics & Medicine***
11. ***Current Clinical Pharmacology***
12. ***World Journal of Preventive Medicine***
13. ***Advances in Pharmaceutical Sciences***
14. ***Journal of Biomedical Research & Environmental Sciences***
15. ***Current Reviews in Clinical and Experimental Pharmacology***
16. ***Journal of Cancer Biology***
17. ***Current Indian Science (Section Editor, Biochemistry)***
18. ***Exploration of Biomat-X***

***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 Sciences16.Reproduction 17. Frontiers in Nutrition 18. Frontiers in Immunology 19. Frontiers in Endocrinology and 20. Cell Death & Disease etc.***

***13. Membership of Scientific Bodies:***

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***1. The Science Advisory Board, USA***

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

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

***4. The Physiological Society of India.***

***5. Indian Society for Translational Research***

***6. DNA Society of India***

***7. Scholars Academic and Scientific Society***

***8. National Environmental Science Academy***

***9. National Academy of Biological Sciences***

***10. Honorary Rosalind Member of London Journal Press 2021***

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

**Eight (8) are already awarded and Six students (SRFs) have already been registered under the Ph.D. Programme in the University of Calcutta/Jadavpur University where I am acting as a Supervisor/Joint-Supervisor. One DBT-RA and one CSIR-Project RA were also worked under my supervision.**

***15. Teaching activities:***

**Taught special paper classes in Biochemistry (Cell signalling, hormone action, cancer signalling and Immunology) and Reproductive Biology (Reproduction, Endocrinology, Reproductive and endocrine disorders etc.) of M.Sc. (Physiology) students of University of Calcutta and Ram Mohan College 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, Chittaranjan National Cancer Institute, Indian Institute of Chemical Biology (IICB),Indian Association for the Cultivation of Science (IACS), Jadavpur University, Vidyasagar University, Institute of Post-Graduate Medical Education & Research (IPGMER) , Institute of Reproductive Medicine, School of Tropical Medicine, All India Institute of Hygiene & Public Health, IQ City Medical College, Krish Biotech Research Pvt. Ltd. etc.***

***17. Advisor (Honorary.)***

***Serving as an Advisor (Honorary) at WBLDCL, Govt. of West Bengal for the Establishment of a State-of-the-art Preclinical Animal Research Centre (CRO/State Centre for laboratory Animal sciences (SCLAS)) funded by Govt. of India.***

***18. 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, 17.14 lakh)***

***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., 39.85 lakh).***

***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, 21.78 lakh).***

***4. “Development of Recombinant Vaccine for Aspergillus-related Asthma and Allergic Broncho-Pulmonary Aspergillosis” from The Wellcome Trust/DBT India Alliance (PI: Dr. Gaurab Sirkar; Co-Investigator: Dr. Kuladip Jana), (Project funded on 01 April, 2019, Rs. 1, 69, 16,460/- at Visva Bharati University).***

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

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

20. List of Important research publications: (Citations: 3500; H index: 30)

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**Editor: Dr. Kuladip Jana**

1. **The Role of Reactive Oxygen Species in Health and Disease by NOVA Publishers, USA, 2022 (In Press)**

## **Apoptosis and Human Health: Understanding Mechanistic and Therapeutic Potential by Springer Nature, 2023. (Manuscript under preparation)**