Dr. SIDDIK SARKAR

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Job Objective

Research & Development in concurrent with teachings in Biological Sciences and Biotechnology

Key Research accomplishments

- Scientific contribution on **tyrosine kinase inhibitor ZD6474, Caprelsa; FDA approved** it for treatment for late-stage (metastatic) medullary thyroid cancer.
- Melanoma differentiation gene-7/ interleukin-24 (*mda*-7/IL-24): Phase I Clinical trial.
- Conditional replication-competent Adenovirus expressing mda-7/Il-24: Recruiting Phase II Clinical trial.
- **Use of a Truncated CCN1 Promoter for Cancer Diagnostics, Therapeutics and Theranostics. (Patent).**
- **Project Investigator (PI) for 2 year Post-doctoral research grant;** Department of Defense (DoD), Maryland, USA.

Area of Specialization

Cell and Molecular Biology, Biochemistry, Cancer Biology, Genetics, Protein Production and purification, Stem cell research, Immunology, Molecular-genetic imaging, Nuclear imaging modalities, Nanotechnology.

Academic Qualifications

PhD, Indian Institute of Technology Kharagpur, WB, INDIA	2011
MSc (Biotechnology), Indian Institute of Technology Roorkee, INDIA	2004
BSc (Bio-Chemistry), University of Delhi	2002

Division: All degrees obtained are 1st Class

Details of employment

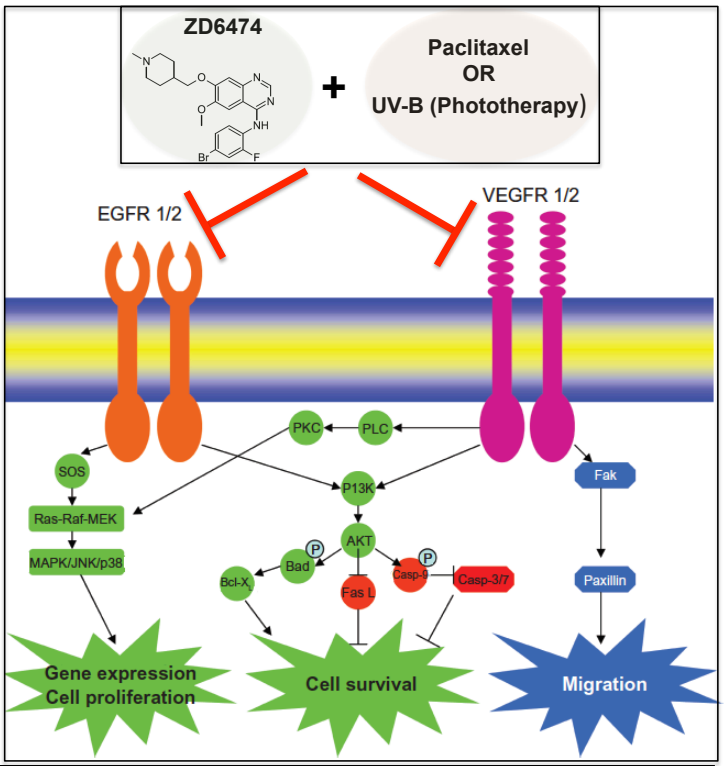
1. **Post-doctoral Associate:** Department of Pharmaceutics, Ernest Mario School of Pharmacy. **Rutgers**, The State University of New Jersey, NJ, USA. October 2015- ongoing.
2. **Post-doctoral research:** Department of Human & Molecular Genetics, School of Medicine, **Virginia Commonwealth University,** Richmond, VA, USA. Oct 2010- Sept 2015.

Research contribution

Ph.D Research (2006-2011)

Molecular & Cell Biology and Cancer Biology: **The molecular effect of ZD6474, a dual tyrosine kinase inhibitor of epidermal growth factor receptor and vascular endothelial growth factor receptor on breast cancer progression and treatment**.

Abnormalities in gene expression and signaling pathways downstream of the epidermal growth factor receptor (EGFR) and vascular endothelial growth factor receptor (VEGFR) contribute to the progression, invasion, and maintenance of malignant phenotype in human cancers, including breast. We have shown that breast cancer cells can be selectively programmed to cell death or decreased cell proliferation by targeting dual tyrosine kinase EGFR and VEGFR by tyrosine kinase inhibitor-ZD6474. In combination therapy, ZD6474 enhances the efficacy of chemotherapy (paclitaxel) and radiotherapy (UV-B) by inhibiting cell proliferation, migration and metastasis. The combination of ZD6474 with paclitaxel or UV-B versus either agent alone also more potently down-regulated the anti-apoptotic bcl-2 protein, up-regulated pro-apoptotic signaling events involving the expression of bax, activation of caspase-3 and caspase-7 proteins, and induced poly(ADP-ribose) polymerase resulting in apoptosis. These observations have considerable potential clinical relevance for patients with locally advanced metastatic breast cancer, where clinical studies of dose-intensive paclitaxel therapy and radiotherapy are currently in progress. Moreover, the local administration of chemotherapeutic drugs often leads to nonspecific distribution in the body via circulatory system. This resulted in cytotoxicities to normal cells other than tumor nest. Nanoparticle (AuNp) assisted drug (ZD6474) delivery system will play a crucial role in tissue and site specific drug delivery with lesser cytotoxicity and greater efficiency. The recurring problem of cancer therapies is the development of chemo-resistance. Thus, the development of chemo (paclitaxel)-resistant breast cancer cell line will help in elucidation of molecules that may be targeted to sensitize and overcome chemo-resistance to conventional therapies.



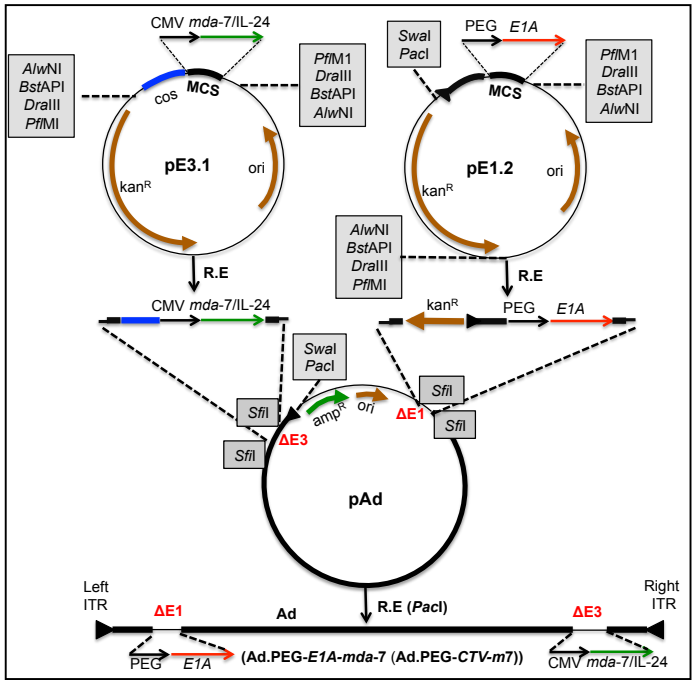
ZD6474, a dual kinase inhibitor of both EGFR and VEGFR in combination with Paclitaxel or radiation (UV-B) inhibits proliferation, cell survival, migration and induces apoptosis in breast cancer cells.

Post-doctoral Research (2010-2015):

Genetics, oncology, molecular biology, transgenic animal models of cancer, Molecular imaging, targeted delivery and chemistry:

Gene therapy in pancreatic cancer in combination with chemoprevention agent or small molecule inhibitor: Pancreatic cancer remains one of the deadliest of all cancers despite aggressive surgical treatment combined with adjuvant radiotherapy and chemotherapy. Chemo-resistance and radio-resistance are the principal causes of failure of pancreatic cancer patients to respond to therapy. Conditionally replication

competent adenovirus (CRCA)-based cancer gene therapy is an innovative strategy for treating cancers displaying inherent resistance to treatment. Limitations of current adenovirus (Ad)-based gene therapies for malignant tumors include lack of cancer-specificity, and effective and targeted delivery. To remedy this situation, CRCAs have been designed that express *E1A*, necessary for Ad replication, under the control of a cancer-specific progression elevated gene-3 promoter (PEG-Prom) with concomitant expression of an immunomodulatory cytokine, such as *mda*-7/IL-24 under the control of a ubiquitous and strong cytomegalovirus promoter (CMV-Prom) from the E3 region. These bipartite CRCAs, when armed with a transgene, are called cancer terminator viruses (CTVs), i.e., Ad.PEG-E1A-CMV-*mda*-7 (Ad.PEG-CTV-*m*7) because of their universal effectiveness in cancer treatment irrespective of genetic alterations in tumor cells. In addition to their selective oncolytic effects in tumor cells, the potent 'bystander antitumor' properties of MDA-7/IL-24 embody the CTVs with expanded treatment properties for both primary and distant cancers. Pancreatic cancer cells display a "translational block" of mda-7/IL-24 mRNA, limiting production of MDA-7/IL-24 protein and cancer-specific apoptosis. Specific chemopreventive agents inducing reactive oxygen species abrogate this "translational block" resulting in pancreatic cancer-specific killing. This combination synergistically induces *mda*-7/IL-24-mediated cancer-specific apoptosis by inhibiting anti-apoptotic Bcl-xL and Bcl-2 protein expression and inducing an endoplasmic reticulum (ER) stress response through induction of BiP/GRP-78, which is most evident in chimeric-modified non-replicating Ad.5/3-*mda*-7- and replicating Ad.5/3-PEG-CTV-*m*7 infected pancreatic cancer cells. Moreover, Ad.5/3-PEG-CTV-*m*7 in combination with POH sensitizes therapy-resistant MIAPaCa-2 cell lines over-expressing either Bcl-2 or Bcl-xL to *mda*-7/IL-24-mediated apoptosis. Ad.5/3-PEG-CTV-m7 plus POH also exerts a significant antitumor 'bystander' effect in vivo suppressing both primary and distant site tumor growth. This novel chemoprevention gene therapy (CGT) strategy holds promise for both prevention and treatment of pancreatic cancers where all other strategies have proven ineffective.

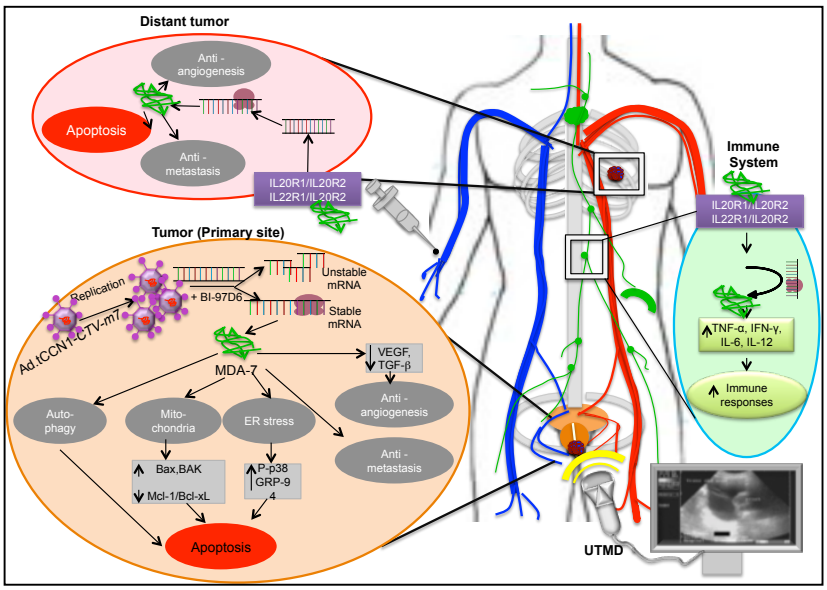


Cloning of conditionally replication competent Adenovirus; Ad.PEG-*E1A*-*mda*-7 (Ad.PEG-CTV-*m*7). Cancer selective PEG-prom regulating the expression of *E1A* is inserted into the deleted *E1A* (Δ*E1A*) and CMV regulating *mda*-7 /IL-24 gene in the deleted E3 (ΔE3) of Adenovirus plasmid (pAd).

Research grant:

Developing an effective therapy for prostate cancer. (2011-2013):

Despite recent advances, treatment options for advanced prostate cancer (CaP) remain limited. We are pioneering approaches to treat advanced CaP that employ conditionally replication-competent oncolytic adenoviruses that simultaneously produce a systemically active cancer-specific therapeutic cytokine, *mda*-7/IL-24, Cancer Terminator Viruses (CTV). A truncated version of the CCN1/CYR61 gene promoter, tCCN1-Prom, was more active than progression elevated gene-3 promoter (PEG-Prom) in regulating transformation-selective transgene expression in CaP and oncogene-transformed rat embryo cells. Accordingly, we developed a new CTV, Ad.tCCN1-CTV-*m*7, which displayed dose-dependent killing of CaP without harming normal prostate epithelial cells *in vitro* with significant anti-cancer activity *in vivo* in both nude mouse CaP xenograft and transgenic Hi-*myc* mice (using ultrasound targeted microbubble (MB)-destruction, UTMD, with decorated MBs). Resistance to *mda*-7/IL-24-induced cell death is correlated with overexpression of Bcl-2 family proteins. Inhibiting Mcl-1 using an enhanced BH3 mimetic, BI-97D6, sensitized CaP cell lines to mda-7/IL-24-induced



Ad.tCCN1-*CTV-m7* and BI-97D6 eradicates prostate cancer: Ad.tCCN1-*CTV-m7* was mixed with targeted microbubble (MB) specific to bind with prostate tumor vasculature overexpressing VCAM, and injected i.v followed by delivery in the prostate region by ultrasound targeted microbubble destruction (UTMD) approach. Following infection, Ad.tCCN1-*CTV-m7* selectively replicates in CaP cells with surplus production of *mda*-7/IL-24, which on translation produced secretory cytokine MDA-7/IL-24. MDA-7 induced apoptosis via Bcl-2 dependent (mitochondria), ER stress or toxic autophagy in prostate tumor (primary site of infection). BI-97D6 synergistically co-operates with Ad.tCCN1-*CTV-m7* in inducing cancer specific apoptosis. BI-97D6 also stabilized *mda*-7/IL-24 mRNA further enhancing the activity of *mda*-7/IL-24. The secreted MDA-7/IL-24 acts on adjacent uninfected CaP cells or tumor located distantly (lung, bone metastasis) via receptor dimerization and signal transduction culminating into cancer-specific apoptosis. It can also activate anti-tumor-immune responses in further exhilarating the antitumor effect of MDA-7/IL-24, and thus eradicates CaP including metastasis

apoptosis. Combining BI-97D6 with Ads expressingmda-7/IL-24 promoted ER stress, decreased anti-apoptotic Mcl-1 expression and enhanced *mda*-7/IL-24 expression through mRNA stabilization selectively in CaP cells. In Hi-*myc* mice, the combination induced enhanced apoptosis and tumor growth suppression. These studies highlight therapeutic efficacy of combining a BH3 mimetic with a novel CTV, supporting potential clinical applications for treating advanced CaP.

Patent:

Use of a truncated CCN1 promoter for cancer diagnostics, therapeutics and theranostics:

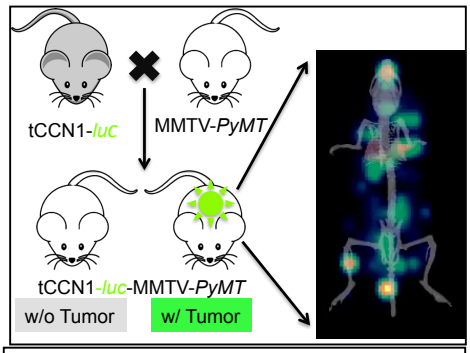


Figure 1: Genetically modified (Can-met-view) mouse to view cancer and metastases. Transgenic mouse containing tCCN1-*luc* vector crossed with breast cancer MMTV-*PyMT* mouse to generate tCCN1-*luc*-MMTV-*PyMT* mouse that upon transformation yield luminescence signal as observed via Bioluminescence imaging system.

Recombinant vectors in which expression of one or more elements (e.g. genes required for viral replication, detectable imaging agents, therapeutic agents, etc.) are driven by a truncated CCN1 cancer selective promoter (tCCN1-Prom) are provided, as are cells and transgenic animals that contain such vectors. The vectors are used in cancer therapy and/or diagnostics, and the transgenic mice are used to monitor cancer progression, e.g. in screening assays. As a proof-of-principal we have developed a double transgenic mice (tCCN1-*luc*-Hi-*myc* and tCCN1-*luc*-PyMT-*MMTV*) by crossing transgenic tCCN1-*luc* mice with established transgenic cancer mice (Hi-*myc* and PyMT-*MMTV*) mice of various origin that upon transformation (development of cancer) activated the CCN1-prom leading to the subsequent expression of the linked luciferase transgene as detected by non-invasive bioluminescence imaging approach to monitor tumor kinetics. We have also shown successfully that these double transgenic cancer mice could be explored and used in clinical settings in observing the subsequent treatment outcome of various anti-cancer agents.

Professional Recognitions, Awards, and Fellowships received

- **Project Investigator** (PI) for 2 year Post-doctoral fellowship grant (W81XWH-11-1-018) (Developing an Effective Therapy for Prostate Cancer) from Department of Defense, U.S. Army Medical Research and Material Command.
- Received Travel grant from Department of Defense, U.S. Army Medical Research and Material Command to defend my Late breaking abstract and to attend the Annual Meeting 2013 held at Washington DC organized by American Association for Cancer Research.
- Received Travel grant from Department of Biotechnology, India and also from Indian Institute of Technology Kharagpur, India to attend the international symposium " VIII Madrid Breast Cancer Conference", Madrid, Spain (June 2009)
- Won first prize in poster presentation in International Conference "Emerging Trends in Biological Sciences", KIIT Bhubaneswar, Bhubaneswar, India. Oct 2008.
- Won **first prize in National Level Technical Festiva**l "Cognizance-2004" held at IIT Roorkee for model making competition "The Motifs".
- Won **first prize in National Level Technical Festival** "Cognizance-2004" held at IIT Roorkee for Poster making competition.
- Awarded CSIR-JRF (Council of Scientific and Industrial Research- Junior Research Scholar) and screened for SPM (Shyma Prasad Mukherjee) award on the merit basis for NET Examination (June 2004).
- Awarded scholarship for Masters programme (**Rank 3** in admission exam for PG (Biotechnology), IIT Roorkee) in Biotechnology on Merit basis sponsored by DBT (Department of Biotechnology, India) (June 2002).

Publications (Chronological order)

Patent

1) Fisher PB, **Sarkar S**, Das SK, Dash R, Sarkar D, Windle J. **Use of a Truncated CCN1 Promoter for Cancer Diagnostics, Therapeutics and Theranostics**" assigned International Application No.**PCT/US2014/40796** which includes and claims priority of U.S. Provisional Application Serial No. 61/830,837 filed in the U.S. Patent and Trademark Office on June 04, 2013. This patent was approved and published on 31.12.2014. Pub No. WO/2014/209553.

Scientific Research articles

1. **Sarkar S**, Pradhan A, Das S, Emdad L, Sarkar D, Pellecchia M, Fisher P. Novel therapy of prostate cancer employing a combination of viral-based immunotherapy and a small molecule BH3 mimetic. **Oncoimmunology**. 2015 September. (doi: 10.1080/2162402X.2015.1078059) (Impact factor 6.3).
2. Rajput S, Puvvada N, Kumar BN, **Sarkar S**, Konar S, Bharti R, Dey G, Mazumdar A, Pathak A, Fisher PB, Mandal M. Overcoming Akt induced therapeutic resistance in breast cancer through siRNA and thymoquinone encapsulated multilamellar gold niosomes. **Mol Pharm**. 2015 Oct 27. [Epub ahead of print] PMID: 26505213.
3. Dey KK, **Sarkar S**, Pal I, Das S, Dey G, Bharti R, Banik P, Ray JG, Maity S, Kulavi I, Mandal M. Mechanistic attributes of S100A7 (psoriasin) in resistance of anoikis resulting tumor progression in squamous cell carcinoma of the oral cavity. **Cancer Cell Int.** 2015 Oct 8;15: 74. (Impact factor 2.8)
4. Quinn BA, Dash R, **Sarkar S**, Azab B, Bhoopathi P, Das SK, Emdad L, Wei J, Pellecchia M, Sarkar D, Fisher PB. Pancreatic Cancer Combination Therapy Using a BH3 Mimetic and a Synthetic Tetracycline. **Cancer Res**. 2015 Jun 1; 75(11): 2305-2315. (Impact factor 9.3).
5. PuvvadaN, Rajput S, Kumar BN, **Sarkar S**, Konar S, Brunt KR, Rao R, Mazumdar A, Das, SK, Basu R, Fisher PB, Mandal M, Pathak A. Novel ZnO hollow-nanocarriers containing paclitaxel targeting folate-receptors in a malignant pH-microenvironment for effective monitoring and promoting breast tumor regression. **Sci Rep**. 2015 Jul 6; 5:11760. (Impact factor 5.07)
6. **Sarkar S**, Quinn BA, Shen X-N, Dash R, Das SK, Emdad L, Klibanov AL, Wang X-Y, Pellecchia M, Sarkar D, Fisher PB. Therapy of prostate cancer using a novel cancer terminator virus and a small molecule BH-3 mimetic. **Oncotarget** 2015 Mach; 6(13): 10712-10727. (Impact factor 6.63).
7. **Sarkar S,** Quinn BA, Shen X, Dent P, Das SK, Emdad L, Sarkar D, Fisher PB**.** Reversing translational suppression and induction of toxicity in pancreatic cancer cells using a chemoprevention gene therapy (CGT) approach. **Mol Phramacol**. 2015 Feb; 87(2):286-295. (Impact factor 4.5).
8. Kumar BN, Puvvada N, Rajput S, **Sarkar S**, Venkatesan P, Pal I, Dey G, Konar S, Mazumdar A, Kundu SC, Pathak A, Fisher PB, Mandal. M Sequential release of tamoxifen and diosgenin from hollow manganese ferrate nanocarriers for breast cancer treatment. **J. Mater. Chem. B**, 2015; 3: 90-101 (Impact factor 6.5).
9. Bhatnagar A, Wang Y, Mease R, Gabrielson M, Sysa P, Minn I, Green G, Simmons B, Gabrielson K, **Sarkar S**, Fisher PB, Pomper M. AEG-1 promoter-mediated imaging of prostate cancer. **Cancer Res.** 2014; 74(20):5772-5781 (Impact factor 9.3).
10. Pal I, **Sarkar S**, Rajput S, Dey KK, Chakraborty S, Dash R, Das SK, Sarkar D, Barile E, De S, Pellecchia M, Fisher PB, Mandal M. BI-69A11 enhances susceptibility of colon cancer cells to mda-7/IL-24-induced growth inhibition by targeting Akt. **Br J Cancer.** 2014 Jul 8;111(1):101-11. (Impact factor 5.2).
11. Azab BM, Dash R, Das SK, Bhutia SK, **Sarkar S**, Shen XN, Quinn BA, Dent P, Dmitriev IP, Wang XY, Curiel DT, Pellecchia M, Reed JC, Sarkar D, Fisher PB. Enhanced prostate cancer gene transfer and therapy using a novel serotype chimera cancer terminator virus (Ad.5/3-CTV). **J Cell Physiol**. 2014 Jan; 229(1): 34-43. (Impact factor 4.3)
12. Dash R, Bhoopathi P, Das SK, **Sarkar S**, Emdad L, Dasgupta S, Sarkar D, Fisher PB. Novel mechanism of MDA-7/IL-24 cancer-specific apoptosis through SARI induction. **Cancer Res**. 2014; 74 (2), 563-574. (Impact factor 9.3)

13. **Sarkar S**, Azab B, Quinn BA, Shen X, Dent P, Klibanov AL, Emdad L, Das SK, Sarkar D, Fisher PB. Chemoprevention gene therapy (CGT) of pancreatic cancer using perillyl alcohol and a novel chimeric serotype Cancer Terminator Virus. **Curr Mol Med**. 2014; 14(1): 125-140. (Impact factor 4.6)
14. **Sarkar S**, Rajput S, Tripathi AK, Mandal M. Targeted therapy against EGFR and VEGFR using ZD6474 enhances the therapeutic potential of UV-B phototherapy in breast cancer cells. **Mol Cancer**. 2013 Oct 20; 12(1): 122. (Impact factor 5.4)
15. **Sarkar S**, Azab BM, Das SK, Quinn BA, Shen X, Dash R, Emdad L, Thomas S, Dasgupta S, Su ZZ, Wang XY, Sarkar D, Fisher PB. Chemoprevention gene therapy (CGT): novel combinatorial approach for preventing and treating pancreatic cancer. **Curr Mol Med**. 2013 Aug; 13(7): 1140-1159. Review (Impact factor 4.6)
16. Rajput S, Kumar BN, **Sarkar S**, Das S, Azab B, Santhekadur PK, Das SK, Emdad L, Sarkar D, Fisher PB, Mandal M. Targeted apoptotic effects of thymoquinone and tamoxifen on XIAP mediated Akt regulation in breast cancer. **PLoS One**. 2013 Apr 17;8(4): e61342. (Impact factor 4.6)
17. Hedvat M, Emdad L, Das SK, Kim K, Dasgupta S, Thomas S, Hu B, Zhu S, Dash R, Quinn BA, Oyesanya RA, Kegelman TP, Sokhi UK, **Sarkar S**, Erdogan E, Menezes ME, Bhoopathi P, Wang XY, Pomper MG, Wei J, Wu B, Stebbins JL, Diaz PW, Reed JC, Pellecchia M, Sarkar D, Fisher PB. Selected approaches for rational drug design and high throughput screening to identify anti-cancer molecules. **Anticancer Agents Med Chem**. 2012 Nov; 12(9): 1143-1155. Review. (Impact factor 2.6)
18. Azab BM, Dash R, Das SK, Bhutia SK, Shen XN, Quinn BA, **Sarkar S**, Wang XY, Hedvat M, Dmitriev IP, Curiel DT, Grant S, Dent P, Reed JC, Pellecchia M, Sarkar D, Fisher PB. Enhanced delivery of mda-7/IL-24 using a serotype chimeric adenovirus (Ad.5/3) in combination with the Apogossypol derivative BI-97C1 (Sabutoclax) improves therapeutic efficacy in low CAR colorectal cancer cells. **J Cell Physiol**. 2012 May; 227(5): 2145-2153. (Impact factor 4.6)
19. Quinn BA, Dash R, Azab B, **Sarkar S**, Das SK, Kumar S, Oyesanya RA, Dasgupta S, Dent P, Grant S, Rahmani M, Curiel DT, Dmitriev I, Hedvat M, Wei J, Wu B, Stebbins JL, Reed JC, Pellecchia M, Sarkar D, Fisher PB. Targeting Mcl-1 for the therapy of cancer. **Expert Opin Investig Drugs**. 2011 Oct; 20(10): 1397-1411. (Impact factor 4.7)
20. Ramachandran S, **Sarkar S,** Mazumadar A, Mandal M. Azurin Synthesis from Pseudomonas Aeruginosa MTCC 2453, Properties, Induction of Reactive Oxygen Species, and p53 Stimulated Apoptosis in Breast Carcinoma Cells. **Journal of Cancer Science & Therapy.** 2011 Sept; 3 (5): 104-111. (Impact factor 5.8)
21. **Sarkar S**, Mazumdar A, Dash R, Sarkar D, Fisher PB, Mandal M. ZD6474 enhances paclitaxel antiproliferative and apoptotic effects in breast carcinoma cells. **J Cell Physiol**. 2011 Feb; 226(2): 375-384. (Impact factor 4.6).
22. Sethi K, **Sarkar S**, Das S, Rajput S, Mazumder A, Roy B, Patra S, Mohanty B, El-Naggar AK, Mandal M. Expressions of CK-19, NF-kappaB, E-cadherin, beta-catenin and EGFR as diagnostic and prognostic markers by immunohistochemical analysis in thyroid carcinoma. **J Exp Ther Oncol**. 2011;9(3):187-199.
23. Dash R, Azab B, Shen XN, Sokhi UK, **Sarkar S**, Su ZZ, Wang XY, Claudio PP, Dent P, Dmitriev IP, Curiel DT, Grant S, Sarkar D, Fisher PB. Developing an effective gene therapy for prostate cancer: New technologies with potential to translate from the laboratory into the clinic. **Discov Med**. 2011 Jan;11(56):46-56. Review. (Impact factor 3.0)
24. Thakur G, Mitra A, Rousseau D, Basak A, **Sarkar S**, Pal K. Crosslinking of gelatin-based drug carriers by genipin induces changes in drug kinetic profiles in vitro. **J Mater Sci Mater Med**. 2011 Jan; 22(1):115-123. (Impact factor 2.5)
25. **Sarkar S**, Mazumdar A, Dash R, Sarkar D, Fisher PB, Mandal M. ZD6474, a dual tyrosine kinase inhibitor of EGFR and VEGFR-2, inhibits MAPK/ERK and AKT/PI3-K and induces apoptosis in breast cancer cells. **Cancer Biol Ther**. 2010 Apr 15;9(8):592-603. (Impact factor 3.3)
26. Sethi K, **Sarkar S**, Das S, Mohanty B, Mandal M. Biomarkers for the diagnosis of thyroid cancer. **J Exp Ther Oncol**. 2010;8(4): 341-352. Review.

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28. Mandal SM, Dey S, Mandal M, **Sarkar S**, Maria-Neto S, Franco OL. Identification and structural insights of three novel antimicrobial peptides isolated from green coconut water. **Peptides**. 2009 Apr; 30(4): 633-637. (Impact factor 2.5).
29. Ghosh K, Chandra K, Ojha AK, **Sarkar S**, Islam SS. Structural identification and cytotoxic activity of a polysaccharide from the fruits of Lagenaria siceraria (Lau). **Carbohydr Res**. 2009 Mar 31; 3 44(5): 693-698. (Impact factor 2.1).

In-Preparation

1. **Sarkar S,** Shen X, Dash R, Al-Zubi M, Windle J, Das SK, Sarkar D, Fisher PB. Theranostic application of cancer-specific promoter CCN1/Cyr61 in breast cancer detection and management. **Nature Medicine** (2014) (in-preparation) (Impact factor 28.05).

Books and Chapters

1. Min, I., Menezes ME, **Sarkar S**, Yarlagadda K, Das SK, Emdad L, Sarkar D, Fisher PB, Pomper M. 2014. Molecular-genetic imaging of cancer. **Adv. Cancer Res** 2014**;** 124:131-169 (Impact factor 6.4).
2. Das SK, **Sarkar S**, Dash R, Dent P, Wang XY, Sarkar D, Fisher PB. Chapter One---Cancer terminator viruses and approaches for enhancing therapeutic outcomes. **Adv Cancer Res**. 2012; 115:1-38.
3. **Sarkar S**, Mandal M. Breast Cancer - Focusing Tumor Microenvironment, Stem cells and Metastasis. Gunduz M editor. Rijeka, Croatia: InTech; 2011. Chapter 4, **Breast Cancer: Classification Based on Molecular Etiology Influencing Prognosis and Prediction**; p.1-17.

Conferences and Proceedings

1. **Sarkar S,** Azab B, Quinn BA, Shen X, Dent P, Klibanov AL, Emdad L, Das SK, Sarkar D, Fisher PB**.** Chemoprevention gene therapy (CGT) approach for pancreatic cancer. (Proceedings Supplement: **Late-Breaking Abstracts at Annual Meeting 2013 American Association for Cancer Research (AACR),** Washington, DC 6-10 April 2013).
2. **Sarkar S,** Mandal M. ZD6474 in combination with paclitaxel and UVB irradiation enhances the antiproliferative effect and apoptosis on breast carcinoma. **Breast Cancer Research** 2009, 11(Suppl 1):P28 (Poster presentation at **VIII Madrid Breast Cancer Conference**: Latest Advances in Breast Cancer Madrid, Spain 24-26 June 2009).
3. **Sarkar S,** Mandal M. The molecular effect of ZD6474, a dual tyrosine kinase inhibitor of EGFR and VEGFR in combating breast cancer. (International Conference: Emerging Trends in Biological Sciences Organized by School of Biotechnology, KIIT University, Bhubaneswar, Orissa, INDIA 24-25 Oct 2008).

References

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Declaration: It is certified that all information given by me above is true to the best of my knowledge. If any information were found false, I am aware that my application is liable for cancellation.

Date: November 20, 2015.
Place: Highland Park, NJ, USA

Signature: 
Name: Siddik Sarkar