List of 10 best papers and their importance

Publications as corresponding author

1. Tripathi V, Agarwal H, Priya S, Batra H, Modi P, Pandey M, Saha D, Raghavan SC, **Sengupta S** (2018). MRN complex-dependent recruitment of ubiquitylated BLM helicase to DSBs negatively regulates DNA repair pathways. Nat Commun. 9(1):1016. PMID: 29523790.

Importance: This work gave a an idea about how signalling happen in cells after exposure to DNA damage. It also provides a frame work to design therapeutic interventions to artificially accelerate the process by which the integrity of the genome can be identified and repaired.

2. Hussain M, Mohammed A, Saifi S, Khan A, Kaur E, Priya S, Agarwal H, **Sengupta S** (2021). MITOL-dependent ubiquitylation negatively regulates the entry of Pol□A into mitochondria. PLoS Biol. 19(3): e3001139. PMID: 33657094

Importance: This publication was a paradigm shifting work which indicated that inspite of the presence of Mitochondrial Localization Signal (MLS), a key protein for mitochondrial replication, Polymerase gamma A (PolgA), can enter into mitochondria only if it non-ubiquitylated. The work has pathological significance as PolgA mutants in progressive external ophthalmoplegia (PEO) are hyper-ubiquitylated and hence cannot enter mitochondria.

3. Tripathi V, Nagarjuna T, **Sengupta S** (2007) BLM helicase-dependent and independent roles of 53BP1 during replication stress mediated homologous recombination. J. Cell Biol. 178(1): 9-14. PMID: 17591918

Importance: This work indicated how DNA repair by homologous recombination happens in the cells via functional interaction between two key players which were previously thought to have functions in completely different pathways. Gave an early clue about how cell death using synthetic lethality can be carried out in cancer cells.

4. Tikoo S, Madhavan V, Hussain M, Miller ES, Arora P, Zlatanou A, Modi P, Townsend K, Stewart GS, **Sengupta S** (2013) Ubiquitin-dependent recruitment of the Bloom Syndrome helicase in response to replication stress is required to suppress homologous recombination. EMBO J. 32(12): 1778-1792. PMID: 23708797

Importance: This elegant study mapped the complete mechanism by which a key protein involved in DNA repair remains in normal resting phase and how that protein becomes activated by ubiquitylation after exposing the cells to DNA damage. Biochemical, cell biology and imaging techniques were used to put across the results.

5. De S, Kumari J, Mudgal R, Modi P, Gupta S, Futami K, Goto H, Lindor NM, Furuichi Y, Mohanty D, **Sengupta S** (2012) RECQL4 is essential for the transport of p53 to mitochondria in normal human cells in the absence of exogenous stress. J Cell Sci. 125(Pt 10): 2509-2522. PMID: 22357944

Importance: This study identified a new mitochondrial helicase, RECQL4. At the time of its publication - it was the second known mitochondrial helicase. The study also shed light how RECQL4 was involved in regulation of mitochondrial replication in a cell cycle specific

manner. Patents in India and European Union have been granted based on the work described in this study. Patent application has also been filed in USA.

6. Priyadarshini R, Hussain M, Attri P, Kaur E, Tripathi V, Priya S, Dhapola P, Saha D, Madhavan V, Chowdhury S, **Sengupta S** (2018). BLM potentiates c-Jun degradation and alters its function as an oncogenic transcription factor. Cell Rep. 24(4):947-961. PMID: 30044990.

Importance: This work described the discovery of a potential therapeutic intervention for a significant fraction of colon cancer patients whereby preferential degradation of c-Jun protooncogene can lead to reduction in tumour development. A PCT application has also been filed based on this study.

7. Hussain M, Mohammed A, Saifi S, Priya S, **Sengupta S** (2023). Hyper-ubiquitylation of DNA helicase RECQL4 by E3 ligase MITOL prevents mitochondrial entry and potentiates mitophagy. J. Biol Chem. 299(9):105087. PMID: 37495109

Importance: This publication indicated that inspite of the presence of Mitochondrial Localization Signal (MLS), a key protein for mitochondrial replication, RECQL4, can enter into mitochondria only if it non-ubiquitylated. The work has pathological significance as RECQL4 mutants in Rothmund Thomson Syndrome (RTS) are hyper-ubiquitylated and hence cannot enter mitochondria.

8. Priya S, Kaur E, Kulshrestha S, Pandit A, Gross I, Kumar N, Agarwal H, Khan A, Shyam R, Bhagat P, Prabhu JS, Nagarajan P, Deo SVS, Bajaj A, Freund JN, Mukhopadhyay A, **Sengupta S** (2021). CDX2 inducible microRNAs sustain colon cancer by targeting multiple DNA damage response pathway factors. J Cell Sci. 134(15): jcs258601. PMID: 34369561

Importance: This study identified and validated in a AIIMS, New Delhi cohort a group of six DNA Damage Sensitive MicroRNAs (DDSMs) which were capable to identifying even Stage I and Stage II colon cancer patients. The common transcription factor regulating the DDSMS, the downstream targets of the microRNAs were identified and validated. This work led to the filing of US and Indian patent applications.

9. Tripathi V, Kaur E, Kharat SS, Hussain M, Damodaran AP, Kulshrestha S, **Sengupta S** (2019). Abrogation of FBW7α-dependent p53 degradation enhances p53's function as a tumor suppressor. J Biol Chem. 294(36):13224-13232. PMID: 31346036.

Importance: This study indicated how the turnover of a key tumour suppressor like p53 can be decreased so that it's in vivo efficacy is increased. These results have immense possibility for potential gene therapy in colon cancer utilizing a "super long-lasting p53" molecule.

10. Kharat SS, Tripathi V, Damodaran AP, Priyadarshini R, Chandra S, Tikoo S, Nandhakumar R, Srivastava V, Priya S, Hussain M, Kaur S, Fishman JB, **Sengupta S** (2016). Mitotic phosphorylation of Bloom helicase at Thr182 is required for its proteasomal degradation and maintenance of chromosomal stability. Oncogene 35(8): 1025-1038. PMID: 26028025

Importance: This study involved understanding of ubiquitination in cancer cells. In this study a very specific phospho-antibody for a protein which gets phosphorylated during mitosis was generated, which can potentially serve as a marker for cancer progression.