BIOGRAPHICAL SKETCH

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NAME: Alexander V. MAZIN, Ph.D.

eRA COMMONS USER NAME (credential, e.g., agency login): AVMAZIN

POSITION TITLE: Professor of Biochemistry and Structural Biology

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Institute of Cytology & Genetics, Novosibirsk, Russia	Ph.D.	1984	Genetics
University of California, CA, Davis	Postdoc	1994-99	Biochemistry

A. Personal Statement

My broad goal is to understand the mechanisms of homologous recombination and DNA repair in human cells and to translate the results of the basic studies into development of novel cancer therapies. I have more than 20 years of experience in the field of homologous recombination and DNA repair with specific expertise in biochemistry of homologous recombination proteins including RAD51, RAD52, and RAD54. Homologous recombination is responsible for the repair of the most harmful type of DNA damage DNA double-stranded breaks and inter-strand cross-links, faithful homologous chromosome segregation during meiosis, and telomere maintenance. As a postdoc at the University of California Davis, I studied several key homologous recombination proteins. As a faculty at Drexel University College of Medicine, I focus my research program on understanding the mechanisms of HR in humans and development of small-molecule inhibitors of the major HR proteins, RAD51, RAD52, and RAD52 for anticancer therapy.

As a PI or co-PI on several NIH, Leukemia and Lymphoma Society, KECK Foundation, Basser Foundation, Coulter program, and Drexel-funded grants I laid the groundwork for future research by developing the biochemical and cellular assays that are required for the analysis of the mechanisms of HR and development of small molecule inhibitors. We have produced multiple peer-reviewed publications from each project and have authored or co-authored 36 papers over the past 10 years, including those published in Nature, Molecular Cell, Nature Structural and Molecular Biology, Nature Communications, Genes and Development, and PNAS. I have trained 18 pre-doctoral and 9 postdoctoral fellows in my lab. I have an extensive experience in collaborative research. At UT Health San Antonio, I continue my nationally recognized program in understanding the mechanisms of DNA repair in human cells and development of specific inhibitors as cancer novel therapeutics. In these studies, I am currently expanding on using biophysical and structural approaches with the major emphasis on cryo-EM technology. In summary, I have the expertise, leadership, and motivation to conduct research in areas of DNA repair and tumorigenesis.

Ongoing and recently completed projects that I would like to highlight include:

R01 CA188347, NIH/NCI
Mazin (PI)
09/25/2015 – 08/31/2022
Small molecule inhibitors as a new approach to study human RAD51 recombinase

R01 GM136717 NIH/NIGMS

04/16/2020-02/29/2024
Mazin (PI)
Mechanisms of RNA-dependent DNA repair in humans
R01 CA23728, NIH/NCI
03/01/2020-01/31/2025
Skorsi and Mazin (PIs in MPI grant)
AML mutation-guided drugging of DNA repair

BC191160, Breast Cancer, Breakthrough Award Level 2, CDMRP/BCRP
08/01/2020-07/31/2023
Mazin and Du (Partnering PIs)
Development of inhibitors of RAD52 protein against BRCA-deficient breast cancer

RR210023, Recruitment of Established Investigators Award, CPRIT
04/01/21 – 03/31/26
Mazin (PI)

UT System Faculty STARs Award
01/10/2022-01/09/2025
Mazin(PI)

Citations:

1. Bugreev, D. V., and Mazin, A. V. (2004). Ca^{2+} activates human homologous recombination protein Rad51 by modulating its ATPase activity. Proc. Natl. Acad. Sci. USA 101, 9988-9993. PMCID: PMC454202
2. Bugreev, D. V., Mazina, O.M., and Mazin, A. V. (2006). Rad54 protein promotes branch migration of the Holliday junctions. Nature (London). v. 442: 590-593. PMID: 17545145
3. Bugreev, D.V., Yu, X., Egelman, E.H., Mazin, A.V. (2007). Novel pro- and anti-recombination activities of the Bloom's syndrome helicase. Genes & Development, 21 (23): 3085-3094. PMCID: PMC2081975
4. Rossi, M.J., Mazina, O.M., Bugreev, D.V., and Mazin, A.V. (2011). The RecA/RAD51 protein drives migration of Holliday junctions via polymerization on DNA. Proc Natl Acad Sci USA 108, 6432-6437. PMCID: PMC3080997
5. Huang, F., Goyal, N., Sullivan, K., Hanamshet, K., Patel, M., Mazina, O.M., Wang, C.X., An, W.F., Spoonamore, J., Metkar, S. Kyle A. Emmitte, Simon Cocklin, Tomasz Skorski, and Alexander V. Mazin (2016) Targeting BRCA1- and BRCA2-deficient cells with RAD52 small molecule inhibitors. Nucleic Acids Res. **44**, 4189-99. PMCID:PMC4872086
6. Mazina, O.M., Keskin, H., Hanamshet, K., and Storici, F., Mazin, A.V., (2017) Rad52-inverse strand exchange drives RNA-templated DNA double-strand break repair. Molecular Cell, 67, p 19-29 3e. PMCID: PMC5547995.

Patents:

1. Alexander V. Mazin and Huang Fei, Patent No.: US 9,750,742, September 5, 2017, Title: "Small Molecule Inhibitors of RAD51 Recombinase and Methods Thereof"
2. Alexander V. Mazin, Patent No.: US 10,738,061, Date of Patent: August 11, 2020. Title: "Inhibitors of RAD52 Recombination Protein and Methods Using Same".

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

1984-1991	Junior Investigator, then Investigator and Senior Investigator, Institute of Cytology and Genetics, Novosibirsk 630090, Russia
1991-1994	Visiting Scientist, Groupe d'Etude "Mutagénèse et cancérogénèse", Institut Curie-BIologie Centre Universitaire - Bâtiment 110, Mentor – Dr. R. Devoret, F-91405 Orsay, France
1994-1999	Postdoc, Section of Microbiology, University of California, Davis, Mentor – Dr. S. Kowalczykowski, CA 95616-8665 USA

1999-2001 Research Assistant Professor, Section of Microbiology, University of California, Davis, CA 95616-8665 USA
2001-2007 Assistant Professor, Department of Biochemistry and Molecular Biology, Drexel University College of Medicine, Philadelphia, PA 19102
2007-2012 Associate Professor, Department of Biochemistry and Molecular Biology, Drexel University College of Medicine, Philadelphia, PA 19102
2012-2021 Professor, Department of Biochemistry and Molecular Biology, Drexel University College of Medicine, Philadelphia, PA 19102
2021-current Professor, Department of Biochemistry and Structural Biology, University of Texas Health Science Center San Antonio, San Antonio, TX 78229

Honors

2004 10^6 Club Award at Drexel University
2008 The Leukemia and the Lymphoma Society Scholar Award
2012 Basic Research Scientist Award, Drexel University College of Medicine.
2018 Jefferson University, Department of Radiation Oncology Visiting Professor Honors
2021 Sidney Kimmel Cancer Center Achievement in Basic Research Award

C. Contribution to Science

1. Discovery of branch migration activity of Rad54 protein. The process of homologous recombination in the cell, a four-stranded DNA intermediate called the Holliday junction (HJ) arises. HJ can migrate along the DNA axis (branch migration) and its resolution or dissolution helps determine whether DNA recombinants with chromosome arm crossover or not are made. However, the identity of the proteins that catalyze branch migration of Holliday junctions in eukaryotes remained elusive for a long time. Our work has made major contributions in this area by showing that RAD54 is the main branch migration motor in eukaryotes and determined regulatory mechanisms that underpin this RAD54 attribute.

1. Bugreev, D. V., Mazina, O.M., and Mazin, A. V. (2006). Rad54 protein promotes branch migration of the Holliday junctions. Nature (London). v. 442: 590-593. PMID: 17545145 *This article was rated by Faculty of 1000.*
2. Mazina, O.M., Mazin, A.V. (2008) Human Rad54 protein stimulates human Mus81/Eme1 endonuclease. Proc. Natl. Acad. Sci. USA**,** 105(47): p. 18249-54. PMCID: PMC2587595
3. Mazina, O.M., Rossi, M.J., Deakyne, J.S., Huang, F., and Mazin, A.V. (2012). Polarity and bypass of DNA heterology during branch migration of Holliday junctions by human RAD54, BLM, and RECQ1. J. Biol. Chem. 287, 11820-11832. PMCID: PMC3320930
4. Goyal, N., Rossi, M.J., Mazina, O.M., Chi, Y., Moritz, R.L., Clurman B.E., Mazin A.V. (2018) RAD54 N-terminal domain is a DNA sensor that couples ATP hydrolysis with branch migration of Holliday junctions. Nature Comm., 9, article number 34, doi:10.1038/s41467-017-02497-x. PMCID: PMC5750232.

2. Understanding of the role of the ATPase activity of human RAD51 and its homologs. Results from previous investigations showed that the ATP binding by RAD51, the major human recombinase, is crucial for DNA strand exchange, while ATP hydrolysis is not required for DNA strand exchange, the major RAD51 activity, and results in the dissociation of RAD51 protomers from DNA. Our studies made an important contribution to the field by demonstrating that RAD51 behaves as a self-inactivating ATPase in that, upon ATP hydrolysis, ADP remains bound to RAD51 and inhibits DNA strand exchange. We then found that Ca^{2+} or the HR factor HOP2-MND2 complex either attenuates ATP hydrolysis by RAD51 or modulates ATP binding by RAD51, respectively, to enhance DNA strand exchange. Furthermore, our studies also revealed the mechanism that links RAD51 ATP hydrolysis to branch migration promoted by this protein. The results showed that RAD51 and its bacterial homologue RecA drive branch migration of HJ by cycles of their ATP-dependent polymerization/dissociation on the HJ.

2. Bugreev, D. V., and Mazin, A. V. (2004). Ca^{2+} activates human homologous recombination protein Rad51 by modulating its ATPase activity. Proc. Natl. Acad. Sci. USA 101, 9988-9993. PMCID: PMC454202
3. Bugreev, D V., Golub, E I, Stasiak, A Z, Stasiak, A, and A, Mazin A V. (2005) Activation of human meiosis-specific recombinase Dmc1 by Ca^{2+}. J. Biol. Chem. 280(29): p. 26886-95. PMID: 16862129

4. Rossi, M.J., Mazina, O.M., Bugreev, D.V., and Mazin, A.V. (2011). The RecA/RAD51 protein drives migration of Holliday junctions via polymerization on DNA. Proc Natl Acad Sci USA 108, 6432-6437. PMCID: PMC3080997
5. Bugreev, D.V., Huang, F., Mazina, O.M., Pezza, R.J., Voloshin, O.N., Daniel Camerini-Otero, R., and Mazin, A.V. (2014). HOP2-MND1 modulates RAD51 binding to nucleotides and DNA. Nature Com. 5, 4198. PMCID: 4279451

3. Study on the function of BLM helicase. Inactivation of BLM helicase is responsible for human cancer predisposition syndrome, known as Bloom syndrome. However, the exact BLM function remains controversial. Mutations in the BLM cause hyperrecombination between sister chromatids indicating an anti-recombination role. Conversely, other data show that BLM is required for HR. We discovered two novel activities of BLM which may account for both these BLM functions. We found that BLM disrupts the Rad51-ssDNA filament, an active species of HR. However, this disruption occurs only if RAD51 is present in an inactive ADP-bound form. When the RAD51-ssDNA is present in an active ATP-bound form, BLM stimulates DNA strand exchange activity of RAD51. Our results demonstrate the important role of the RAD51 nucleoprotein filament conformation in regulation of HR by BLM. Interestingly, the nucleoprotein filaments formed by DMC1, a meiosis specific RAD51 homolog, resist BLM disruption, which may account of the role of DMC1 in meiosis.

1. Bugreev, D.V., Yu, X., Egelman, E.H., Mazin, A.V. (2007). Novel pro- and anti-recombination activities of the Bloom's syndrome helicase. Genes & Development, 21 (23): 3085-3094. PMCID: PMC2081975
2. Bugreev, D.V., Mazina, O.M., and Mazin, A.V. (2009). Bloom syndrome helicase stimulates RAD51 DNA strand exchange activity through a novel mechanism. J. Biol. Chem. 284, 26349-26359. PMCID: PMC2786030
3. Bugreev, D. V., Pezza, R. J., Mazina, O. M., Voloshin, O. N., Camerini-Otero R. D., Mazin A. V. (2011) The resistance of DMC1 D-loops to dissociation may account for the DMC1 requirement in meiosis. Nature Struct. & Mol. Biol., 18, 56-60. PMCID: PMC3058924

4. Studies on RNA-dependent DNA repair: a) Discovery of the inverse strand exchange activity of RAD52 and its role in RNA-templated DNA repair and b) R-lop formation activity of RPA. RNA can serve as a template for DNA double-strand break repair in yeast cells, and Rad52, a member of the homologous recombination pathway, plays role in this process. However, the exact mechanism of how Rad52 contributes to RNA-dependent DSB repair remained unknown. We discovered a novel activity of yeast and human Rad52: inverse strand exchange, in which Rad52 forms a complex with dsDNA and promotes strand exchange with homologous ssRNA or ssDNA. In accord with our in vitro results, our experiments in budding yeast provide evidence that Rad52-inverse strand exchange plays an important role in RNA-templated DSB repair in vivo.

1. Keskin, H., Shen, Y., Huang, F., Patel, M., Yang, T., Ashley, K., Mazin, A.V., and Storici, F. (2014) Transcript RNA-templated DNA recombination and repair. Nature, *515*, 436-439.

2. Mazina, O.M., Keskin, H., Hanamshet, K., and Storici, F., Mazin, A.V., (2017) Rad52-inverse strand exchange drives RNA-templated DNA double-strand break repair. Molecular Cell, 67, p 19-29 3e. PMCID: PMC5547995.

Replication protein A (RPA), a major eukaryotic ssDNA-binding protein, is essential for all metabolic processes that involve ssDNA. While RPA is known to bind ssDNA tightly, it was presumed that it binds RNA weakly. However, recent data suggest that RPA may play a role in RNA metabolism. We investigated the RNA-binding properties of human RPA and found that RPA binds RNA with an unexpectedly high affinity (K_D ≈ 100 pM). Furthermore, RPA by forming a complex with RNA can promote R-loop formation with homologous dsDNA. We showed that human DNA polymerases can utilize RPA-generated R-loops for initiation of DNA synthesis mimicking the process of replication restart *in vivo*. These results support the role of RPA in RNA metabolism and suggest a mechanism of genome maintenance that depends on RPA-mediated DNA replication restart.

3. Mazina, O.M., Somarowthu, S., Kadyrova, L.Y., Baranovskiy, A.G., Tahirov, T.H., Kadyrov, F.A., and A.V. Mazin (2020). Replication protein A binds RNA and promotes R-loop formation (JBC, 295(41): p. 14203-14213) PMCID: PMC7549048.

5. Development of small molecule inhibitors of the key Homologous recombination proteins. Using high throughput screening (HTS) we identified several small molecule inhibitors of RAD51. We demonstrated that one of them, named B02, inhibited HR and DSB repair *in vivo*. B02 was the first known biologically active inhibitor of a HR protein. Using mouse xenografts, we demonstrated that B02 increases the efficacy of cisplatin

in killing triple-negative breast cancer cells *in vivo*. We further improved the efficacy of B02 using medicinal chemistry approach

1. Huang, F., Mazina, O.M., Zentner, I J., Cocklin, S., and Mazin, A.V. (2012). Inhibition of homologous recombination in human cells by targeting RAD51 recombinase, J. Medicinal Chem., 55(7): p. 3011-20. PMID: 22380680 *Faculty of 1000 rated it twice as a "must read".*
2. Huang, F., and Mazin, A.V. (2014). A Small Molecule Inhibitor of Human RAD51 Potentiates Breast Cancer Cell Killing by Therapeutic Agents in Mouse Xenografts. PLoS ONE 9, e100993. PMCID: PMC4074124
3. Shkundina, I. S., A. A. Gall, A. Dick, S. Cocklin and A. V. Mazin (2021). "New RAD51 Inhibitors to Target Homologous Recombination in Human Cells." Genes (Basel) 12(6).

Previous studies demonstrated that while single RAD52 mutations show no significant phenotype in mice, their combination with mutations in Breast Cancer proteins 1&2 (BRCA1/2) are lethal. These observations defined RAD52 as a novel therapeutic target for BRCA1/2-deficient familial breast cancer and ovarian cancer. Recently we identified by HTS of ~400,000 compound library we identified small molecule inhibitors of RAD52. Several of these inhibitors show specific effect on BRCA1 and BRCA2-deficient cells and suppress RAD52-dependent recombination.

4. Huang, F., Goyal, N., Sullivan, K., Hanamshet, K., Patel, M., Mazina, O.M., Wang, C.X., An, W.F., Spoonamore, J., Metkar, S. Kyle A. Emmitte, Simon Cocklin, Tomasz Skorski, and Alexander V. Mazin (2016) Targeting BRCA1- and BRCA2-deficient cells with RAD52 small molecule inhibitors. Nucleic Acids Res. **44**, 4189-99. PMCID:PMC4872086

We also identified by HTS an inhibitor of RAD54 protein, streptonigrin. Using this inhibitor, we found that the ATPase activity of RAD54 is important for branch migration, but not for stimulation of RAD51

5. Deakyne J.S., Huang, F., Negri, J., Tolliday, N., Cocklin, S., and Mazin, A.V. (2013). Analysis of the activities of RAD54, a SWI2/SNF2 protein, using a specific small-molecule inhibitor, J. Biol. Chem. 288, 31567-31580. PMCID: PMC3814753

Complete List of Published Work in MyBibliography:

http://www.ncbi.nlm.nih.gov/sites/myncbi/alexander.mazin.1/bibliography/41139620/public/?sort=date&direction=ascending

BIOGRAPHICAL SKETCH

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NAME: Simranjeet Singh Sekhon

eRA COMMONS USER NAME (credential, e.g., agency login):

POSITION TITLE: Postdoctoral Research Fellow

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Guru Nanak Dev University Amritsar, India	MS	2006-2008	Bioinformatics
KRIBB School, University of Science & Technology Daejeon, South Korea	PHD	2009-2013	Bio-Analytical Science
Chungbuk National University, South Korea	Postdoctoral Fellow	2013-2016	Structural Biology / Systems Biology
University of Texas Health Science Center at San Antonio (UTHSCSA)	Postdoctoral Fellow	2022-present	Biochemistry / Structural Biology

A. Personal Statement

My research focuses on structural biology, which is the integration of biochemistry and biophysics concerned with the structure of biological macromolecules. Characterization of molecular structure of proteins has become critical, as it can provide insights into the way proteins function and interact with other molecules. As a part of my Ph.D. work, the Structure of Wheat Cyclophilin (wTaCypA1) and its complex with its inhibitor Cyclosporin A (wTaCypA1-CsA) was determined by X-ray Crystallography and it was the first report on the structure of a Cyclophilin derived from Plants. The structure of Cyclophilin from wheat played an important part in understanding the role of TaCypA1 in its interactions with other proteins. Later on as a Postdoctoral Fellow, I studied aptamers and their microbial and biomedical applications with an emphasis on the aptamer structural features involved in aptamer-target specific interactions. The aptamer technology is an integral part of systems biology and the structural studies help converge computational approaches with biology for better understanding of its complex mechanisms. My research on aptamers is focused on various targets ranging from proteins, microbial pathogens, heavy metals, and marine environmental bio-indicators. Some of the detection techniques successfully developed include biosensors, aptamer based strip sensors, aptabody (aptamer as antibody), and aptamer based sandwich assays. I have authored and co-authored multiple peer-reviewed publications, including those published in NPJ 2D Materials and Applications, Nanoscale, Journal of Nanobiotechnology, Acta Cryst. D, Journal of Biomedical Nanotechnology, Biosensors and biotechnology, Scientific Reports. As a trained protein biochemist and X-ray crystallographer, I have significant appreciation for how high-quality sample preparation influences successful structure determination. I am excited to work on RAD52 protein target biological system that my current laboratory at UTHSCSA already has a prior experience in biochemical and biophysical characterization. My aim is to determine the RAD52 structural framework using cryo-EM analysis to understand the mechanism of RAD52 interactions with its inhibitors.

Completed Projects
2017R1D1A1B03033366, National Research Foundation Korea (NRF)
Sekhon Simranjeet Singh (PI)
07/2017-06/2019
Development of LAMP & Aptamer based highly sensitive GM crop coupled detection system

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

2008 – 2009	Project Assistant, Molecular Biophysics Unit, Indian Institute of Science Bengaluru, India. (Mentor – Prof. Manju Bansal)
2009 – 2009	Post Masters, Korea Research Institute of Bioscience & Biotechnology, South Korea. (Mentor – Dr. Tae-Sung Yoon)
2009 – 2013	PhD student, KRIBB School, University of Science & Technology, Daejeon, South Korea. (Mentor – Dr. Tae-Sung Yoon)
2013 – 2016	Postdoctoral Fellow, School of Biological Sciences, Chungbuk National University, South Korea. (Mentor – Dr. Yang-Hoon Kim)
2016 – 2021	Visiting Assistant Professor (Research), Chungbuk National University, South Korea.
2021 – 2022	Visiting Associate Professor (Research), Chungbuk National University, South Korea
2022 – Present	Postdoctoral Research Fellow, Department of Biochemistry & Structural Biology, University of Texas Health Science Center at San Antonio, Texas, USA. (Mentor – Prof. Alexander Mazin)

Honors

2009	Summer Research Fellowship at Indian Institute of Science Bengaluru, awarded by The Indian Academy of Science Bengaluru, India.
2012	KRIBB Best Researcher Award, awarded by the President of KRIBB on 28th Annual Day ceremony
2013	UST Excellence Award, presented by the President of University of Science & Technology, Daejeon, South Korea at Spring Commencement Graduation Ceremony.
2014	UST Research Paper Award, in recognition of contribution to the University and the progress of science and technology through outstanding research achievements.

C. Contributions to Science

1. **Structure of wheat cyclophilin TaCypA-1:** The wheat cyclophilin TaCypA-1 structure is the first structure of a plant cyclophilin that shows peptidyl-prolyl isomerase (PPIase) activity. This study was the first to elucidate the crystal structures of TaCypA-1 and its complex with CsA. The structures of apo TaCypA-1 and the TaCypA-1– CsA complex were determined at 1.25 and 1.20 A˚ resolution, respectively, using X-ray diffraction. The TaCypA-1 structure revealed the presence of a divergent loop of seven amino acids ^{48}KSGKPLH^{54} which is a characteristic feature of plant cyclophilins, is absent in human cyclophilins (hCypA and hCypD). The presence of the divergent loop region in TaCypA-1 suggests an important role in protein–protein interactions.

    a. **Sekhon, S. S.**, Kaur, H., Dutta, T., Singh, K., Kumari, S., Kang, S., Park, S. G., Park, B. C., Jeong, D. G., Pareek,A., Woo, E. J., Singh. P. and Yoon, T. S. Structural and biochemical characterization of the cytosolic wheat cyclophilin TaCypA-1. Acta Cryst.D. 2013; 69:555-563.

2. **Structure targeted aptamer interactions**: Aptamers are oligonucleotides that can mimic antibodies by folding into complex three-dimensional structures and bind to specific targets. Their easy synthesis, low-cost, low immunogenicity, and low variability empower them as an ideal alternative to antibodies. The aptamers bind to the molecules of interest through the specific shape induced by their sequence and complementary functioning. The interaction between the specific region of the aptamer and its counterparts is not well understood, which is partially because of the relatively small number of 3-D structures that have been characterized to date. Using a combination of complementary techniques (bioinformatics and experimental approaches) can help in understanding the interaction between the aptamer and its target molecules, which is a key step towards improving the efficiency of aptamer-based diagnostic platform technology.

a. **Sekhon, S. S.,** Um, H. J., Shin, W-R., Lee, S. H., Min, J., Ahn, J-Y. and Kim, Y-H. Aptabody–aptatope interactions in aptablotting assays. Nanoscale. 2017; 9:7464-7475.

b. Shin, W-R., Park, D-Y, Kim. J. H., Lee, J-P., Thai, N. Q., Oh, I. H., **Sekhon, S. S.**, Choi, W., Kim, S. Y., Cho, B-K., Kim, S. C., Min, J., Ahn, J-Y., Kim, Y-H. Structure based innovative approach to analyze aptaprobe–GPC3 complexes in hepatocellular carcinoma. Journal of Nanobiotechnology. 2022; 20:204.

3. **Aptamer based detection system**: Aptamers are oligonucleotides having excellent chemical stability and low immunogenicity that can be easily integrated with other technologies involving nucleic acid-based systems. Their small size increases the accessibility to most biological areas that are beyond the reach of antibodies. The real significance of aptamers lies in the ease by which they can be engineered into biosensors and other devices that are often vital to emerging technologies. Their low-cost synthesis and high stability, can be used to generate multiple aptamers against different targets, such as small molecules, proteins, intact viruses, and even cells. Aptamers can be easily functionalized multiple times within their backbone or at the termini with a number of functional groups for diagnostic and therapeutic objectives, without any compromise in their activity. The use of aptamers in cancer research can lead to significant advances in cancer detection, diagnosis, and target therapy.

   a. **Sekhon, S. S**., Kaur, P., Kim, Y-H. & Sekhon, S. S. 2D Graphene oxide-aptamer conjugate materials for cancer diagnosis. NPJ 2D Materials and Applications. 2021; 5:21.

   b. **Sekhon, S. S**., Lee, S. H., Lee, K. A., Min, J., Lee, B. T., Kim, K. W., Ahn, J-Y. and Kim, Y-H. Defining the copper binding aptamotif and aptamer integrated recovery platform (AIRP). Nanoscale. 2017; 9:2883-2894.

   c. Shin, W-R., **Sekhon, S. S.*,** Kim, S. G., Won, K., Rhee, S. K., Ryu, H., Kim, K., Min, J., Ahn, J-Y. & Kim, Y-H. Aptamer-based Pathogen Monitoring for Salmonella enterica ser. Typhimurium. J Biomed Nanotechnol. 2018; 14:1991–2001. (*Co-First Author)

   d. Shin, W- R., **Sekhon, S. S.**, Rhee, S. K., Ko, J. H., Ahn, J-Y., Min, J. and Kim, Y-H. Aptamer Based Paper Strip Sensor for Detecting Vibrio fischeri. ACS Combinatorial Science 2018; DOI: 10.1021/acscombsci.7b00190.

   e. Song, M. S., **Sekhon, S. S.*,** Shin, W-R., Kim, H. C., Min, J., Ahn, J-Y. & Kim, Y-H. Detecting and Discriminating Shigella sonnei Using an Aptamer-Based Fluorescent Biosensor Platform. Molecules. 2017; 22:825. (*Co-First Author)