BIOGRAPHICAL SKETCH

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NAME: Parihar, Pankaj Singh

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

POSITION TITLE: Postdoctoral Research Associate

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)	END DATE MM/YYYY	FIELD OF STUDY
Kumaun University , Nanital	B.Sc.	06/2009	Life Science
GBPUAT University, Pantnagar	M.Sc.	06/2011	Biochemistry & Molecular Biology
Jawahar Lal Nehru University, Delhi	PHD	03/2021	Life Science
University Of Wisconsin , Madison	Postdoctoral Fellow	07/2024	

A. Personal Statement

I possess an extensive background in Biochemistry and Biotechnology, with specialized training and expertise in protein purification, protein biochemistry, and structural biology. Under the mentorship of Dr. J. V. Pratap during my doctoral studies, I focused on the structural and functional characterization of hypoxanthine-guanine phosphoribosyl transferase (HGPRT) and the cell cycle protein Mob1 of Leishmania donovani, as well as the coronin coiled coil domain of Trypanosoma brucei.

In my current postdoctoral fellowship in Dr. Yongna Xing's lab at the University of Wisconsin-Madison, I have had the remarkable opportunity to explore PP2A biology. During this tenure, I have acquired a suite of new skill sets, including insect cell-based protein production and cryo-EM techniques, specifically pertaining to the PP2A PR70 holoenzyme. Our research has illuminated a highly dynamic aspect of PP2A regulation that is pivotal for the stringent control of PP2A holoenzyme biogenesis, disassembly, and activity. Additionally, we have made significant strides in identifying short linear interaction motifs (SLiMs) in substrates or regulatory proteins for diverse PP2A holoenzymes and in characterizing the biochemical codes governing PP2A holoenzyme-substrate interactions.

With my extensive research experience in protein biochemistry and access to state-of-the-art cryo-EM facilities capable of achieving high-resolution structures, my research employs diverse multidisciplinary biophysical and biochemical methodologies. These approaches are meticulously designed to attain a profound mechanistic understanding and to facilitate the identification of novel therapeutic targets and strategies

B. Positions, Scientific Appointments and Honors

Positions and Scientific Appointments

2021 - 2024 Postdoctoral Research Associate, University of Wisconsin-Madison, Madison, WI
2020 - 2021 Research Assistant, CDRI Lucknow, Lucknow

Honors

2020 - 2021 Research Assistant, CSIR CDRI
2017 - 2020 Senior Research Fellowship, Council of Scientific & Industrial Research
2015 - 2017 Junior Research Fellowship, Council of Scientific & Industrial Research
2012 Uttarakhand State Eligibility Test (USET) for Assistant Professor, Kumaun University

C. Contribution to Science

1. Graduate Research during master's study: To devoted myself into science, I applied to a master's degree program at GB Pant University Pantnagar India. During two years in my master's study, I joined Dr. Himanshu Punetha lab to work on a project related to Alternaria Blight disease on Brassica Juncea. The investigation was carried out to know the biochemical basis of defense mechanism of seven Brassica juncea genotypes infected with Alternaria blight, under field condition. Biochemical analysis of infected genotype revealed an increase in PAL, PPO and peroxidase activity. EC-399299, EC-399296 and PHR-2 exhibited maximum increase in these parameters.

    a. Parihar P, Prakash O, Punetha H. Changes in metabolites of Brassica juncea (Indian mustard) during progressive infection of Alternaria brassicae. Nat Sci. 2012; 10:39-42.
    b. Parihar P, Prakash O, Punetha H. Investigation on defensive enzymes activity of Brassica juncea genotypes during pathogenesis of Alternaria blight. .

2. Graduate Research: During my graduate studies in molecular and structural biology under the supervision of Dr. J. V. Pratap, my research focused on the structural and functional characterization of key metabolic pathways and actin-binding proteins in pathogenic parasites, specifically Leishmania donovani and Trypanosoma brucei. My work encompassed the structural and functional elucidation of hypoxanthine-guanine phosphoribosyl transferase (HGPRT) and the cell cycle protein Mob1 in Leishmania donovani, as well as the coronin coiled coil domain in Trypanosoma brucei.

    HGPRT plays a central role in the salvage pathway by converting purine bases to their monophosphate products. To elucidate its function and facilitate drug development, we determined the crystal structure of L. donovani HGPRT at 2.76 Å resolution. This structure revealed differences in oligomer association compared to the human homolog, due to sequence insertions at the tetramer interface. These differences were exploited to identify small-molecule fragment inhibitors, which demonstrated significant effects on the enzyme's activity and the viability of parasite cells.

    In another study, we structurally characterized the cell cycle-interacting protein Mps One Binder (MOB1) from Leishmania donovani, with the crystal structure resolved at 1.3 Å resolution. MOB1 is known as a kinase-activating subunit involved in the regulation of the Hippo signaling pathway. Our ongoing interaction studies with the PK50 kinase aim to further understand its regulatory mechanisms.

    Additionally, we solved and analyzed the structures of the helical bundle-forming cytoskeleton-interacting protein coronin. This protein regulates actin dynamics through assembly and disassembly processes. The structure of the T. brucei coronin coiled coil domain revealed an asymmetric antiparallel tetramer, akin to its L. donovani homolog. In vivo co-localization studies have shown distinct differences, and my contributions aim to propose fundamental biological mechanisms underlying actin dynamics and its regulation.

    Overall, my research has provided valuable insights into the structural and functional aspects of these crucial proteins, contributing to a deeper understanding of parasite biology and potential therapeutic targets

    a. Parihar PS, Singh A, Karade SS, Sahasrabuddhe AA, Pratap JV. Structural insights into kinetoplastid coronin oligomerization domain and F-actin interaction. Curr Res Struct Biol. 2021;3:268-276. PubMed Central PMCID: PMC8554105.
    b. Parihar PS, Pratap JV. The L.donovani Hypoxanthine-guanine phosphoribosyl transferase (HGPRT) oligomer is distinct from the human homolog. Biochem Biophys Res Commun. 2020 Nov 19;532(4):499-504. PubMed PMID: 32873391.

3. Postdoctoral Research Study: Currently, I am working in the oncology division under the guidance of Dr. Yongna Xing at the University of Wisconsin-Madison. Dr. Xing's lab is a renowned structural biology laboratory dedicated to studying the structural basis of human Protein Phosphatase 2A (PP2A) function and the key mechanisms involved in PP2A biogenesis. Our primary research objective is to identify interaction motifs for PP2A-B' holoenzymes, aiming to elucidate the molecular mechanisms of substrate recognition for this class of PP2A holoenzymes. In collaboration with Dr. Y. Iva, we identified consensus

motifs recognized by PP2A-B’ holoenzymes, leading to the discovery of over 100 novel PP2A substrates within the human proteome. The discovery of interaction motifs for PP2A-B’ holoenzymes has provided profound insights for scientists in the field, significantly expanding the understanding of PP2A substrates and their associated cellular functions. This work not only advances the fundamental knowledge of PP2A biology but also has the potential to inform novel therapeutic strategies in oncology and beyond

a. Wu CG, Balakrishnan VK, Merrill RA, Parihar PS, Konovolov K, Chen YC, Xu Z, Wei H, Sundaresan R, Cui Q, Wadzinski BE, Swingle MR, Musiyenko A, Chung WK, Honkanen RE, Suzuki A, Huang X, Strack S, Xing Y. B56δ long-disordered arms form a dynamic PP2A regulation interface coupled with global allostery and Jordan's syndrome mutations. Proc Natl Acad Sci U S A. 2024 Jan 2;121(1):e2310727120. PubMed Central PMCID: PMC10769853.
b. Konovalov KA, Wu CG, Qiu Y, Balakrishnan VK, Parihar PS, O'Connor MS, Xing Y, Huang X. Disease mutations and phosphorylation alter the allosteric pathways involved in autoinhibition of protein phosphatase 2A. J Chem Phys. 2023 Jun 7;158(21) PubMed Central PMCID: PMC10238128.
c. Wu CG, Balakrishnan VK, Parihar PS, Konovolov K, Chen YC, Merrill RA, Wei H, Carragher B, Sundaresan R, Cui Q, Wadzinski BE, Swingle MR, Musiyenko A, Honkanen R, Chung WK, Suzuki A, Strack S, Huang X, Xing Y. Extended regulation interface coupled to the allosteric network and disease mutations in the PP2A-B56δ holoenzyme. bioRxiv. 2023 Apr 5; PubMed Central PMCID: PMC10103954.

Complete List of Published work in MyBibliography

https://www.ncbi.nlm.nih.gov/myncbi/pankaj%20singh.parihar.1/bibliography/public/

BIOGRAPHICAL SKETCH

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NAME: Xing, Yongna

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

POSITION TITLE: Professor

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
Fudan University	BS	06/1995	Biochemistry
Fudan University	MS	06/1997	Genetics
Rutgers/UMDNJ	PHD	05/2002	Molecular Genetics & Microbiology
UMDNJ	Postdoctoral Fellow	2004	Molecular and Cell Biology
Princeton University, Princeton, NJ	Postdoctoral Fellow	2008	Structural Biology

A. Personal Statement

Our lab utilizes highly multidisciplinary research approaches, including x-ray crystallography, cryo-EM, biochemistry, chemistry, biophysics, proteomics, system biology, bioinformatics, cell biology and state-of-the-art imaging approaches, to elucidate molecular mechanisms for rationale targeting of cellular signaling and molecular machinery for cancer, toxicity, and neurodegenerative disorders. We have long-standing expertise and interest in understanding complex protein phosphatase 2A (PP2A) regulation and have made key mechanistic understanding to cancer-promoting and immune disease-related signaling such as those mediated by aryl hydrocarbon receptor (AHR) and by metadherin (MTDH) and staphylococcus nuclease domain 1 (SND1).

To tackle complex PP2A regulation and function, we have developed a plethora of research tools that are uniquely developed in our lab that allow us to reconstitute and dissect biochemical processes involved in diverse aspects of PP2A regulation and substrate recognition. Our study elucidated a highly dynamic aspect of PP2A regulation that is crucial for tight control of PP2A holoenzyme biogenesis, disassembly, and activity. We also made important advances in identifying short interaction motifs (SLiMs) in substrates or regulatory proteins for diverse PP2A holoenzymes and characterizing biochemical codes for PP2A holoenzyme-substrate interactions. Using these biochemical codes, we build bioinformatic tools for rapid search and prediction of phosphatase action in signaling networks and human diseases. In combination with the cryo-EM expertise we built in the recent three years, we are illuminating high-resolution structures that couple SLiMs to diverse PP2A signaling and regulation core complexes to create versatile function and regulation. These tools facilitate personalized medicine research on phosphatase diseases to tackle and target disease mutations or dysregulations that alter phosphatase function in cancer and neurological disorders.

In recent years we also made key breakthroughs for both structural biology and ligand chemistry of AHR signaling and cancer-promoting MTDH-SND1. AHR signaling is notoriously difficult to study due to its ability to respond to diverse compounds that mediate broad and even opposing biological endpoints. We recently determined the crystal structure of an AHR transcriptional complex. In addition, we discovered that kynurenine, a long-known endogenous AHR ligand, activates AHR by the formation of trace extended aromatic condensation products (TEACOPs). Our studies provide key insights into AHR signaling ramifications mediated by endogenous ligands versus environmental toxicants. Building on the structural basis of MTDH-SND1 binding, lead compounds have been identified that effectively suppress breast cancer metastasis (https://cdmrp.health.mil/bcrp/research_highlights/23Kang_Xing_highlight.aspx).

Training, Mentoring and Outreach activities.
Our vigorous research program provides a highly multidisciplinary research environment. It allows me to make strong commitments to identifying and training the next generation of scientists for personalized medicine. I have taught “Purification and Characterization of Protein and Protein Complexes” and Structural Toxicology (a session of lectures in Toxicology) for over ten years. I have trained more than 50 postdoctoral fellows, graduate students, and undergraduate students. Our graduate students have contributed to more than 50% of publications in the lab. The majority of our trainees have attended and received awards from national scientific meetings and moved on to faculty, postdoctoral positions, or take other key research positions in academia or pharmaceutical companies. I have also served as consultant on scientific advisory board of Biotech companies to facilitate mechanistic understanding of drugs and translation and commercialization of our research results. My group also interacts with and presents our results to patient families related to phosphatase disease mutations on a quarterly basis.

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

Shaw Scientist Award
Xing (PI)
07/01/2011 – 06/30/2031
Exploring Novel Approaches of Targeting PP2A Phosphatase Activator

R01 GM145811
Xing (PI)
09/01/2023 – 06/30/2027
The Structural Bases of PP2A Phosphatase Diseases

R01 GM137090
Xing (PI)
04/01/2021 – 02/28/2025
Structural and Cellular Choreography for Decommissioning and Recycling of PP2A Holoenzymes

A19-3376-S007
Xing (PI)
07/01/2018 – 12/31/2022
Mechanistic Understanding and Targeting of PPP2R5D Disease Mutations (Jordan’s Syndrome)

MSN223974
Xing (PI)
09/01/2018 – 12/31/2021
Developing Novel Kynurenine Derivatives for Mitigating Inflammatory Human Diseases

MSN242677
Xing (PI)
07/01/2020 – 06/30/2021
VILAS Associate Year 1: Biological Sciences 2020-2021

PRJAAC9577
Bradfield, Xing (PIs)
01/10/2018 – 01/09/2020
Research project for Ah receptor research

BC151403P1
Xing, Kang (PIs)
08/15/2016 – 08/14/2019
Functional Mechanism and Targeting of Metadherin in Breast Cancer

MSN211614
Xing (PI)
04/01/2018 – 03/31/2019
PPP2R5D Gene Variation (Jordan’s Syndrome)

R01 GM096060
Xing (PI)

01/15/2012 – 08/31/2018
Structural and biochemical insights into PP2A holoenzyme biogenesis

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

2020-present Professor, McArdle Laboratory for Cancer Research, Department of Oncology, University of Wisconsin-Madison, Madison, WI
2014-2020 Associate Professor, McArdle Laboratory for Cancer Research, Department of Oncology, University of Wisconsin-Madison, Madison, WI
2008-2014 Assistant professor, UNIVERSITY OF WISCONSIN-MADISON, Madison, WI
2004-2008 Postdoctoral research associate, PRINCETON UNIVERSITY, Princeton, NJ

Other Experience and Professional Memberships

2014-present Member, American Society for Cell Biology
2011-present Member, American Society for Biochemistry and Molecular Biology
2004-present Member, Biophysical Society
2007-2012 Member, American Association of Crystallography
2003-2004 Member, The Society of Study of Reproduction
2001-2003 Member, Protein Science Society

Professional Services

2021 SMPH Computing & Storage Steering Committee, UW-Madison
2020- Executive Editor, Journal of Protein Expression and Purification
2019 Session Chair, SOT annual meeting
2018 Session Chair, FASEB summer conference on Protein Phosphatases
2017-2018 Cancer Biology Faculty Search Committee, Search Committee for Chair of Dermatology
2017 Chair of Advisory Committee for UWCCC drug discovery cores
2017 SMPH Security Policy Committee
2017 Editorial Board, Nature Cell Discovery
2017 Chair of McArdle Seminar Committee
2015 Molecular Medicine Center Project Prioritization Committee, University of Wisconsin-Madison, Madison, WI
2015 Faculty Senate, University of Wisconsin-Madison, WI
2015 Scientific Advisory Board for Firebrand for development of novel cancer therapeutics
2014 Consultant for Signum Biosciences, Inc. on PP2A-targeting compounds for control of neurodegenerative disorders.
2010 Associate Editorial Board, International Journal of Biochemistry and Molecular Biology
2010 Admission Committee of the Biophysics Program, University of Wisconsin-Madison, Madison, WI

Ad-hoc grant reviewer for: Breast Cancer Research Program (BCRP) for the Department of Defense (DoD), Canada Research Chairs, National Science Foundation of China, Research Grants Council (RGC) of Hong Kong, French Research Council, Research Council of KU Leuven, China National Natural Science Foundation, UWCCC pilot grant, UW ICTR pilot grant, UW 2020, UW research core revitalization

Honors

2020 Vilas Associate Award, University of Wisconsin-Madison
2017 Society of Toxicology, Young Investigator Award
2011 Shaw Scientist Award, Greater Milwaukee Foundation
2010 Research Scholar Award, American Cancer Society
2007 NCI K01 Howard Temin Cancer Development Award, UW-Madison
1997 Molecular Biology Graduate School Fellowship, Rutgers/UMDNJ Joint Program
1994 Institute of Genetics Fellowship, Fudan University
1991 People's Scholarship, Fudan University
1990 Second prize, China National High School Student Mathematics Competition
1990 Second prize, China National High School Student Chemistry Competition

C. **Contribution to Science** (BBP trainees and collaborators underlined)

1. Our studies on PP2A regulation addressed a highly challenging, dynamic aspect of PP2A regulation – biogenesis of substrate-specific holoenzymes from the newly synthesized PP2A catalytic subunit (C or PP2Ac). Four exciting breakthroughs had been made. We uncovered that a PP2A inhibitory protein α4 binds only to the partially folded C, underlying its function and mechanism in stabilizing the newly synthesized C in an inactive form. Next, our studies reveal that PP2A phosphatase activator (PTPA) is a unique chaperone to the PP2A active site and orients ATP for altering metal chelation chemistry at the PP2A active site that enhances the binding of authentic catalytic metal ions by 10,000-fold. Furthermore, PP2A activation stimulates the activity of PP2A methyltransferase (LCMT-1). Prior to these studies, we elucidated a striking mechanism of PP2A demethylation that involves a large conformational change in PP2A methylesterase (PME-1) essential for activation of the enzyme and revealed an unexpected role of PME-1 in PP2A inactivation by eviction of catalytic metal ions. These observations link two seemingly separate PP2A regulations – formation of oligomeric holoenzymes, and modulation of the PP2A active site, into hierarchical controls of the molecular events en route to the biogenesis of substrate-specific holoenzymes.
    a. Stanevich V, Jiang L, Satyshur KA, Li Y, Jeffrey PD, Li Z, Menden P, Semmelhack MF, **Xing Y**. The structural basis for tight control of PP2A methylation and function by LCMT-1. Mol Cell. 2011 Feb 4;41(3):331-42. PubMed Central PMCID: PMC3060061.
    b. Jiang L, Stanevich V, Satyshur KA, Kong M, Watkins GR, Wadzinski BE, Sengupta R, **Xing Y**. Structural basis of protein phosphatase 2A stable latency. Nat Commun. 2013;4:1699. PubMed Central PMCID: PMC3644067.
    c. Guo F, Stanevich V, Wlodarchak N, Sengupta R, Jiang L, Satyshur KA, **Xing Y**. Structural basis of PP2A activation by PTPA, an ATP-dependent activation chaperone. Cell Res. 2014 Feb;24(2):190-203. PubMed Central PMCID: PMC3915903.
    d. Wlodarchak N, **Xing Y**. PP2A as a master regulator of cell cycle. Crit Rev Biochem Mol Biol. 2016 Feb 24:1-23. PMID:26906453.

2. Taking advantage of our unique strength in PP2A biochemistry, we recently opened a new direction of research in collaboration with Dr. YIva Ivarsson to identify interaction motifs for diverse PP2A holoenzymes globally using powerful proteomic peptide phage display (ProP-PD) technology. By acquiring advanced biolayer interferometry (BLI) and live cell time-lapse imaging systems, we developed high throughput PPIs and PP2A cell biology for rapid validation of PP2A interaction motifs and exploration of their cellular functions. By developing powerful bioinformatic tools, we identified many proteins at centrosome and midbody that harbor intrinsic or phosphorylation-responsive interaction motifs for PP2A-B56/B' holoenzymes, underlying diverse signaling loops formed by mitotic kinases and PP2A-B' holoenzymes at these supercomplexes for precise control of mitosis and cytokinesis. Currently, we are developing bioinformatic tools for prediction and guiding investigation of phosphatase action in diverse cellular signaling and under pathological conditions.
    a. Wlodarchak N, Guo F, Satyshur KA, Jiang L, Jeffrey PD, Sun T, Stanevich V, Mumby MC, **Xing Y**. Structure of the Ca2+-dependent PP2A heterotrimer and insights into Cdc6 dephosphorylation. Cell Res. 2013 Jul;23(7):931-46. PubMed Central PMCID: PMC3698643.
    b. Wu CG*, Chen H*, Guo F*, Kyaiims V*, Rowse M, Mcilwain SJ, Ong IM, Li Y[1], Gu T, Zheng A, Lee W, Johnson B, Ge Y, Burkard E, Ivarsson Y, **Xing Y**. PP2A-B' holoenzyme substrate recognition, regulation, and role in cytokinesis. Nature Cell Discovery, (2017) 3, 17027; doi:10.1038.
    c. Konovolov KA, Wu CG, Balakrishnan VK, Parihar PS, O'Connor MS, **Xing Y***, Huang X*. Disease mutations and phosphorylation alter the allosteric pathways involved in autoinhibition of protein phosphatase 2A. J Chem Phys 2023; 158(21): 215101. (*Shared corresponding authors).
    d. Wu CG, Balakrishnan VK, Parihar PS, Konovolov KA, Qiu Y, Chen Y, Merrill RA, Wei H, Sundaresan R, Cui Q, Wadzinski BE, Swingle MR, Musiyenko A, Honkanen R, Chung WK, Suzuki A, Strack S, Huang X, **Xing Y**. B56δ long-disordered arms form a dynamic PP2A regulation interface coupled with global allostery and Jordan's syndrome mutations. Proceedings of the National Academy of Sciences of the United States of America. 2024 January 2;121(1):e2310727120. PubMed PMID: 38150499; PubMed Central PMCID: PMC10769853; DOI: 10.1073/pnas.2310727120.

3. Built on our knowledge on PP2A holoenzyme biogenesis, our recent advance on PP2A structural biology and functional studies reveal striking mechanisms for disassembly of PP2A holoenzymes and for recycling PP2Ac back to the latent form. Altogether, PP2A holoenzyme biogenesis and recycling form a novel signaling loop for dynamic and precise control of PP2A holoenzyme function.

a. Wu CG*, Zheng A*, Jiang L, Rowse M, Stanevich V, Chen H, Li Y, Satyshur KA, Johnson B, Gu T, Liu Z, **Xing Y**. (2017) Methylation-regulated decommissioning of multimeric PP2A complexes. Nature Communications, 8: 2272 | DOI: 10.1038/s41467-017-02405-3.
b. Guo F, Wlodarchak N, Menden P, Xing Y. Purification of Target Proteins from Native Tissues: CCT Complex from Bovine Testes and PP2Ac from Porcine Brains. Methods Mol Biol. 2017 Dec 17. doi: 10.1007/7651_2017_89.
c. Li Y, Rowse M, Stanevich V, Chen H, Satyshur KA, Johnson B, Gu T, Liu Z, **Xing Y**. (2022) Coupling to short linear motifs creates versatile PME-1 activities in PP2A holoenzyme demethylation and inhibition. eLife 2022;11:e79736.
d. Polubothu S, Zecchin D, et al, **Xing Y**, Healy E., Moore GE, Di W, Newton-Bishop J, Downward J, Kinsler VA, (2021) Inherited duplications of PPP2R3B predispose to naevi and melanoma via a C21orf91-driven proliferative phenotype. Genet Med. 2021; 23(9): 1636–1647.

4. We made important advances on the structural biology of aryl hydrocarbon receptor (AHR) signaling and MTDH-SND1 cancer signaling that promote cancer metastasis. We built the homology models of AHR ligand binding domain bound to its diverse environmental and endogenous ligands and explained the binding of different toxins to AHR with genetic variations. We made exciting breakthrough toward elucidating the high-resolution crystal structure of AHR in complex with its transcription partner, ARNT, and the DNA enhancer sequences targeted by AHR/ARNT. We also made key discovery of novel derivatives of kynurenine, a key cellular metabolite, that serve as potent ligands for AHR, underlying key structural and chemical basis of kynurenine in AHR activation. Our work also illuminated the structural basis of MTDH-SND1 cancer cell signaling and facilitated drug discovery targeting MTDH to reduce cancer metastasis.
   a. Seok S, Lee W, Jiang L, Molugu K, Erickson SS, Zheng A, Li Y, Park S, Bradfield CA, **Xing Y**. (2017) Structural Hierarchy Controlling Dimerization and Target DNA Recognition in AHR Transcriptional Complex, PNAS, 114(21):5431-5436.
   b. Seok S*, Ma Z*, Feltenberger JB, Chen H, Chen H, Scarlett C, Lin Z, Satyshur KA, Cortopassi M, Jefcoate CR, Ge Y, Tang W, Bradfield, CA, **Xing Y***. (2018) Kynurenine condensation products potently activate the aryl hydrocarbon receptor (AHR), JBC, 293(6):1994-2005.
   c. Guo F*, Wan L*, Zheng A, Stanevich V, Chen H, Wei Y, Satyshur KA, Shen M, Lee W, Kang Y and **Xing Y.** (2014) The structure of MTDH-SND1 complex reveals novel cancer-promoting interactions. Cell Report, 8(6): 1704-13. PMID: 25242325.
   d. Shen M, Wei Y, et al, **Xing Y**, Shao Z, Kang Y. (2022) Small Molecule Inhibitors that Disrupt the MTDH-SND1 Complex Suppress Breast Cancer Progression and Metastasis. Nature Cancer 3:43-59.

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