

BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Zong, Wei-Xing

eRA COMMONS USERNAME (credential, e.g., agency login): WXZONG

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
Nankai University, Tianjin, China	B.S.	06/1987	Biology
Nankai University, Tianjin, China	M.S.	06/1991	Genetics
University of Medicine & Dentistry of New Jersey (UMDNJ), Piscataway, New Jersey	Ph.D.	12/1999	Biochemistry & Molecular Biology
University of Pennsylvania, Philadelphia, Pennsylvania	Postdoctoral	08/2005	Cancer Biology

A. Personal Statement

My laboratory studies cell metabolism and growth signaling during oncogenesis and in response to therapeutic stress. Our research focuses on several signaling pathways and biological processes including PI3 kinases, Myc/Ras/Akt proto-oncogenes, apoptosis, autophagy, and cell metabolism. I am fully committed to the training of next generation cancer researchers. There are currently four graduate students, one postdoctoral fellow, and two research assistants in my lab. I have graduated nine Ph.D. students and trained seven postdoctoral fellows; all have successfully advanced to the next stage of their professional careers. In addition, I have been actively involved in teaching/training/admissions activities. I have served on admissions committees for several graduate programs and on graduate thesis committees for more than 50 graduate students. I am Program Co-Leader of the Cancer Metabolism and Immunology Research Program at Rutgers Cancer Institute of New Jersey (CINJ). As Program CoLeader, I work to carefully select and mentor Program Members, and foster an interactive and cancer-focused environment that leads to impactful science.

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

R01CA224550 NIH-NCI

Zong (PI)

09/01/2018-08/31/2023

Glutamine metabolism in oncogenesis and cancer therapy

This project studies the regulation of glutamine anabolism and its role in cancer development and treatment.

R01CA232246 NIH-NCI

Zong (PI)

09/01/2018-08/31/2023

PI3-kinase PIK3CB (p110beta) in membrane trafficking and cell metabolism

This project studies how PIK3CB regulates endocytosis and autophagy and how it affects the stability of cell surface nutrient transporters.

R01CA129536 NIH-NCI

Zong (PI)

04/14/2008-06/30/2023

Protein and redox homeostasis in cancer development and therapy

This project studies the regulation of redox pathway by protein aggregation and its role in oncogenesis and cancer therapy.

T32CA257957-01 NIH-NCI

Zong (contact PI), Kang (co-PI)

04/01/2021-03/31/2026

Post-doctoral training program in cancer metabolism and tumor-host Interactions

Citations:

1. Pan JA, Sun Y, Jiang YP, Bott AJ, Jaber N, Dou Z, Yang B, Chen JS, Catanzaro JM, Du C, **Ding WX**, Diaz-Meco MT, Moscat J, Ozato K, Lin RZ, and **Zong WX**^{*}. 2016. TRIM21 ubiquitylates SQSTM1/p62 and suppresses protein sequestration to regulate redox homeostasis. *Mol Cell* 61: 720-733. "Featured Article". PMID: PMC4779181
2. Li Y, **Zong WX**, **Ding WX**. 2017. Recycling the danger via lipid droplet biogenesis after autophagy. *Autophagy* 13: 1995-1997. PMID: PMC5788485
3. Wang F[#], Zhang Y[#], Shen J, Yang B, Dai W, Yan J, Maimouni S, Daguplo HQ, Coppola S, Gao Y, Wang Y, Du Z, Peng K, Liu H, Zhang Q, Tang F, Wang P, Gao S, Wang Y, Ding WX, Guo G, Wang F, and **Zong WX**^{*}. 2021. The ubiquitin E3 ligase TRIM21 promotes hepatocarcinogenesis by suppressing the p62-Keap1-Nrf2 antioxidant pathway. *Cell Mol Gastroenterol Hepatol*. 11(5):1369-1385. PMID: PMC8024979
4. Hou K, Shen J, Yan J, Zhai C, Zhang J, Pan JA, Zhang Y, Jiang Y, Wang Y, Lin RZ, Cong H, Gao S, **Zong WX**^{*}. 2021. Loss of TRIM21 alleviates cardiotoxicity by suppressing ferroptosis induced by the chemotherapeutic agent doxorubicin. *EBioMedicine* 69:103456. PMID: PMC8261003

B. Positions, Scientific Appointments, and Honors

Positions

2021-Present	Director, T32 postdoctoral training award on cancer metabolism tumor-host interaction, CINJ
2019-Present	John Colaizzi Endowed Professor, Department of Chemical Biology, Ernest Mario School of Pharmacy, Rutgers University, Piscataway, New Jersey
2015-Present	Professor, Department of Chemical Biology, Ernest Mario School of Pharmacy, Rutgers University, Piscataway, New Jersey
2015-Present	Co-Leader, Cell Metabolism and Growth Program, Cancer Institute of New Jersey (CINJ), New Brunswick, New Jersey
2013-2015	Professor, Department of Molecular Genetics & Microbiology, Stony Brook University, Stony Brook, New York
2011-2013	Associate Professor, Department of Molecular Genetics & Microbiology, Stony Brook University, Stony Brook, New York
2005-2011	Assistant Professor, Department of Molecular Genetics & Microbiology, Stony Brook University, Stony Brook, New York

Other appointments

06/2021	NIH, Mechanisms for Cancer Therapeutics-1 (MCT1) Study Section, Ad Hoc
11/2020, 03/2021, 11/2021	NIH, NCI F09A: Fellowships: Oncology Study Section
03/2020	NIH, NCI Special Emphasis ZRG1 CB-H (55) R study section
2015-2019	Standing Member, Tumor Cell Biology (TCB) study section, NIH
07/2016	NIH, NCI Special Emphasis ZRG1-OBT study section
01/2016	NIH, NCI P01 Special Emphasis Panel (SEP) study section
03/2015	DoD Study Section, Breast Cancer Research Program
01/2015	NIH, NCI P01 Special Emphasis Panel (SEP) study section
07/2014	DoD Study Section, Breast Cancer Research Program
06/2014	NIH, NCI P01 Special Emphasis Panel (SEP) study section
10/2013	NIH, Tumor Cell Biology (TCB) study section, Ad Hoc

03/2013	NIH, NRSA, Oncology study section
11/2012	NIH, NRSA, Oncology study section
10/2012	DoD Study Section, Breast Cancer Research Program
02/2012	NIH, Tumor Cell Biology (TCB) study section, Ad Hoc
06/2011	NIH, Tumor Cell Biology (TCB) study section, Ad Hoc
2008-2010	DoD Study Section, Breast Cancer Research Program

C. Contributions to Science

1. TRIM21 regulating ROS and proteostasis: My lab has demonstrated that the ubiquitin E3 ligase TRIM21 can regulate the p62-Keap1-Nrf2 redox axis, and plays an important role in liver cancer and oxidative heart injury:
 - a. Pan JA, Sun Y, Jiang YP, Bott AJ, Jaber N, Dou Z, Yang B, Chen JS, Catanzaro JM, Du C, Ding WX, Diaz-Meco MT, Moscat J, Ozato K, Lin RZ, and **Zong WX***. 2016. TRIM21 ubiquitylates SQSTM1/p62 and suppresses protein sequestration to regulate redox homeostasis. *Mol Cell* 61: 720-733. "Featured Article". PMID: PMC4779181
 - b. Wang F[#], Zhang Y[#], Shen J, Yang B, Dai W, Yan J, Maimouni S, Daguplo HQ, Coppola S, Gao Y, Wang Y, Du Z, Peng K, Liu H, Zhang Q, Tang F, Wang P, Gao S, Wang Y, Ding WX, Guo G, Wang F, and **Zong WX***. The ubiquitin E3 ligase TRIM21 promotes hepatocarcinogenesis by suppressing the p62-Keap1-Nrf2 antioxidant pathway. *Cell Mol Gastroenterol Hepatol*. 11(5):1369-1385 (2021). PMID: PMC8024979
 - c. Hou K, Shen J, Yan J, Zhai C, Zhang J, Pan JA, Zhang Y, Jiang Y, Wang Y, Lin RZ, Cong H, Gao S, **Zong WX***. Loss of TRIM21 alleviates cardiotoxicity by suppressing ferroptosis induced by the chemotherapeutic agent doxorubicin. *EBioMedicine* 69:103456 (2021). PMID: PMC8261003
2. SerpinB3/B4 (SCCA) in oncogenesis: My lab characterized the expression of the endogenous protease inhibitor SerpinB3/B4 (SCCA) in breast cancer patients and demonstrated that SerpinB3/B4 are a transcriptional target of oncogenic Ras, and that SerpinB3/B4 promotes tumorigenesis by inducing the unfolded protein response (UPR), IL-6 production, and epithelial-mesenchymal transition (EMT). We published the following papers:
 - a. Catanzaro JM, Guerriero JL, Liu J, Ullman E, Sheshadri N, Chen JJ, and **Zong WX***. 2011. Elevated expression of squamous cell carcinoma antigen (SCCA) is associated with human breast carcinoma. *PLoS One* 6: e19096. PMID: PMC3079753
 - b. Ullman E, Pan JA, and **Zong WX***. 2011. Squamous cell carcinoma antigen 1 (SCCA1) promotes caspase-8-mediated apoptosis in response to endoplasmic reticulum stress while inhibits necrosis induced by lysosomal injury. *Mol Cell Biol* 31:2902-2919. PMID: PMC3133401
 - c. Sheshadri N, Catanzaro JM, Bott A, Sun Y, Ullman E, Wu S, Pan JA, Chen E, Crawford HC, Zhang J, and **Zong WX***. 2014. Squamous cell carcinoma antigen 1 (SCCA1)/SerpinB3 promotes tumorigenesis through induction of unfolded protein response and IL-6 signaling. *Cancer Res* 74:6318-6329. PMID: PMC4216755
 - d. Catanzaro JM, Sheshadri N, Pan JA, Shi C, Li J, Powers RS, Crawford HC, and **Zong WX***. 2014. Oncogenic Ras induces cytokine production by up-regulating squamous cell carcinoma antigens SerpinB3/B4. *Nat Commun*. 5:3729. PMID: PMC4025922.
3. Oncogenic regulation of cell metabolism: Cell metabolism is often reprogrammed in cancer cells. We study how oncogenes regulate cell metabolism. We recently found that c-Myc can induce the expression of glutamine synthetase (GS) by promoting active demethylation of the GS promoter. GS catalyzes the de novo synthesis of glutamine to meet the increased demand for glutamine in cancer cells.
 - a. Fan Y, Dickman K, and **Zong WX***. 2010. Akt and c-Myc differentially activate cellular metabolic programs and prime cells to bioenergetic inhibition. *J Biol Chem* 285: 7324-7333. PMID: PMC2844180
 - b. Bott AJ, Peng IC (co-first author), Fan Y, Faubert B, Zhao L, Li J, Neidler S, Sun Y, Jaber N, Krokud D, Lu W, Rabinowitz J, Hatzoglou M, Powers S, Murphy DJ, Jones R, Wu S, Girmun G, and **Zong WX***. 2015. Myc induces expression of glutamine synthetase through thymine DNA glycosylase-mediated promoter demethylation. *Cell Metab* 22:1068-1077. PMID: PMC4670565
 - c. **Zong WX**, Rabinowitz JD, and White E. 2016. Mitochondria and cancer. *Mol Cell* 61: 667-676. PMID: PMC4779192

- d. Bott AJ, Shen J, Tonelli C, Zhan L, Sivaram N, Jiang YP, Yu X, Bhatt V, Chiles E, Zhong H, Maimouni S, Dai W, Velasquez S, Pan JA, Muthalagu N, Morton J, Anthony TG, Feng H, Lamers WH, Murphy D, Guo JY, Jin J, Crawford HC, Zhang L, White E, Lin RZ, Su X, Tuveson D, and **Zong WX***. 2019. Glutamine anabolism plays a critical role in pancreatic cancer by coupling carbon and nitrogen metabolism. Cell Rep 29:1287-1298. PMID: PMC6886125
4. Regulation of autophagy and endocytic trafficking by PI3 kinases: My lab demonstrated that the Class III PI3 kinase Vps34 is essential for autophagy by creating a Vps34 conditional knockout mouse strain. We also demonstrated that the p110beta isoform of the Class I PI3 kinase is a positive regulator of autophagy by activating Rab5 small GTPase.
- a. Dou Z, Chattopadhyay M, Pan JA, Guerriero JL, Jiang YP, Ballou LM, Yue Z, Lin RZ, and **Zong WX***. 2010. The Class IA phosphatidylinositol 3-kinase p110 β subunit is a positive regulator of autophagy. J Cell Biol 191:827-843. PMID: PMC2983054
- b. Jaber N, Dou Z, Chen JS, Catanzaro JM, Jiang YP, Ballou LM, Selinger E, Ouyang X, Lin RZ, Zhang J*, and **Zong WX***. 2012. Class III PI3K Vps34 plays an essential role in autophagy and in heart and liver function. Proc Nat Acad Sci, USA 109:2003-2008. PMID: PMC3277541
- c. Dou Z, Pan JA, Dbouk HA, Ballou LM, DeLeon JL, Fan YJ, Chen JS, Liang Z, Li G, Backer JM, Lin RZ, and **Zong WX***. 2013. Class IA PI3-kinase p110 β subunit promotes autophagy through Rab5 small GTPase in response to trophic factor limitation. Mol Cell 50:29-42. PMID: PMC3628298
- d. Jaber N, Mohd-Naim N, Wang Z, DeLeon JL, Kim S, Zhong H, Sheshadri N, Dou Z, Edinger AL, Du G, Braga VMM, and **Zong WX***. 2016. Vps34 regulates Rab7 and late endocytic trafficking through recruitment of the GTPase activating protein Armus. J Cell Sci 129:4424-4435. PMID: PMC5201010

Complete List of Published Work in PubMed: <http://www.ncbi.nlm.nih.gov/pubmed/?term=zong+wx>