
BIOGRAPHICAL SKETCH

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NAME: **Ronai, Ze'ev**

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

POSITION TITLE: Professor and Chief Scientific Advisor, Tumor Initiation and Maintenance Program

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
The Hebrew University, Jerusalem, Israel	B.S.	1980	Biology
The Hebrew University, Jerusalem, Israel	M.S.	1982	Microbiology
The Hebrew University, Jerusalem, Israel	Ph.D.	1985	Immunobiology
Columbia University, New York, New York	Postdoc	1988	Molecular Biology

A. Personal Statement

My passion for science is reflected in my dedication to solve critical unmet clinical problems in cancer, with a focus on epigenetic mechanisms that underlie tumor development, progression and resistance. Our studies often provided new fundamental understanding for transcription factor and ubiquitin ligase regulation and function in cancer, resulting in novel paradigms. In addition to the privilege of serving as a cancer researcher, I have had a number of administrative duties, including the establishment of the Signal Transduction Program, serving as Associate Director/Deputy Director to NCI Cancer Center, Scientific Director at Sanford Burnham Prebys Medical Discovery Institute (SBP) La Jolla, and over the past few years, parallel to serving as Chief Scientific Advisor I have established a new cancer center at the Technion, in Israel (Technion Integrated Cancer Center, with the Nobel Laureate Aaron Ciechanover). Initiated and led a number of large grant initiatives from P01 to T32 and led the SAC for the Melanoma Research Foundation. In addition to serving on a number of editorial boards, I served as editor-in-chief for *Pigment Cell Melanoma Research*, the formal journal for the melanoma society. Among my major scientific contributions are the development of sensitive PCR enabling detection of mutant Ras oncogene in normal appearing tissues, defining the early days of personal medicine, the appreciation of rewired signal transduction in cancer, and the fundamental understanding of ubiquitin ligases role in key cellular processes (i.e. hypoxia, UPR), which define cancer development, progression, and therapy resistance.

B. Positions and Honors

Research and/or Professional Appointments:

1982 – 1985 Ph.D., Lautenberg Center, Hebrew University, Jerusalem, Israel.
1985 – 1989 Postdoctoral Fellow, Columbia University, NY, Mentor I.B. Weinstein.
1988 – 1998 Head, Molecular Carcinogenesis Program, American Health Foundation.
1989 – 1997 Assistant/Associate Prof. Depts Microbiology Pathology, NY Med College, Valhalla, NY.
1993 – 1998 Consultant, Department of Medicine, Memorial Sloan Kettering, New York, NY.
1997 – 1999 Associate Professor, Rutenberg Cancer Center, Mount Sinai School of Medicine, NY.
1999 – 2005 Professor, Rutenberg Cancer Center, MSSM, New York, NY.
2004 – 2013 Director, and Professor, Signal Transduction Prog, Sanford-Burnham Medical Research Institute (SBMRI), La Jolla, CA.
2005 – 2016 Adjunct Professor, Molecular Pathology Program, UC San Diego, La Jolla, CA.
2008 – 2014 Associate/Deputy Director, NCI Cancer Center, SBMRI, La Jolla, CA.
2013 – Present Professor, Tumor Initiation and Maintenance Program, SBMRI, La Jolla, CA.
2013 – 2016 Scientific Director, Sanford-Burnham Medical Research Institute, La Jolla, CA.
2016 – 2018 Professor & Co-Director, Technion Integrated Cancer Center, Technion, Haifa, Israel.
2016 – Present Chief Scientific Advisor, Sanford Burnham Prebys Medical Discovery Institute, La Jolla, CA.

Professional Activities:

NIH grant review: 1991 NIH committee for RFA Design in Radiation Biology; 1994–1999 Member Cancer Study Section Tobacco Research Program, UCSF; 1995–1997 NCI Multidisciplinary Special Emphasis Study Section; 1995 NIH Review Committee for Program Project on Radiation Biology; 1996–2001 Member of NCI Chemical Pathology Study Section; 2001 NIH Review for PPG on Melanoma Biology; 2007–2009 NIH TPM Study Section; 2012 NCI Provocative Questions. 2012–present Ad Hoc NCI study sections CAMP, TCB, OIA/R35 and others.

Scientific Advisory Boards: P01, University of Arizona, Tucson; P01, Wistar Institute, Philadelphia; SPORE, University of Colorado, Denver, MGH, Boston. **Conference Organization:** 1994 Chair, Symposium on Int. Cell Diff. Conf Hiroshima, Japan; 1996 Organizing Committee, Symposium on Cancer & Biotechnology, Nice, France; Organizing committee, 4th International Symposium Cancer Biotech., Geneva, Switzerland; 2000 Co-Chair, NIH workshop on Melanoma; 2004, 2006, 2010 Co-organizer, Ubiquitin Workshop, Hebrew University, Jerusalem; 2007 Co-organizer International Melanoma Conference. 2015, June, Ubiquitin Workshop, Southern China; June 2015 Melanoma workshop Iceland. **Editorial:** 1996–98 and 2001–2003 *Carcinogenesis*; 1997–2001 *Cancer Prevention and Detection*; 1999–2002 Associate Editor *Clinical Cancer Research*; 2001–present *Molecular Carcinogenesis*; 2002–present *Cancer Biology and Therapy*; 2002–2007 *Journal of Biological Chemistry*; 2002–present *Molecular and Cellular Biology*; 2007–2010 Associate Editor *Cancer Research*; 2007–2009 Executive Editor *Pigment Cell and Melanoma Research*; 2010–2012 Editor-in-Chief *Pigment Cell and Melanoma Research*. Frequent reviewer *Cell*, *Mol. Cell*, *Cancer Cell*, *Science*, *Science Sig.* *Nature*, *Nat. Cell Biol*, *Nat. Med.* *Nat Comm PNAS*, *EMBO J*, *PLoS journals*, and others.

Awards and Honors:

1982 Prize from the Hebrew University for MS. Outstanding Thesis.
1984 Awarded the Golda Meir Fellowship.
1992 JPCR Fellowship from National Cancer Center, Tokyo.
1995 Princess Takamatsu Symposium, Tokyo; Hiroshima Cancer Symposium.
2000 Dean lecture series – Mount Sinai School of Medicine.
2001 Karolinska Nobel Conference on Ubiquitin, Stockholm, Sweden.
2002, 2005 Banbury Conference CSH, Melanoma biology.
2004 Karolinska Nobel Conference on Hypoxia, Stockholm, Sweden.
2008 Keynote speaker, Melanoma Symposium, Penn State University.
2012 NCI Director Lecture Series.
2013 UCSD Cancer Center Director's Seminar Series, speaker.
2014 UCI Cancer Center Retreat, Keynote speaker.
2016 Frontiers in Cancer Research at the Nobel Forum, Karolinska Institutet, Stockholm, Sweden.
2016 Society Melanoma Research, Lifetime Achievement Award, Boston, MA.
2019 Keynote speaker, C3 (UCSD; Salk; SBP) cancer centers symposia.

C. Contributions to Science (over 21,000 citations; H-index >78; i10 index >196; >280 publications)

Significant discoveries from my lab include:

The early days (basis) of precision medicine. Identification of mutant oncogenes in pre-neoplastic lesions and bodily fluids. Established enriched PCR method and used the technology to identify mutant Ras oncogene alleles in normal appearing colonic tissues, and lavage/sputum from smokers.

- Kahn SM, Jiang W, Culbertson TA, Weinstein IB, Williams GM, Tomita N, Ronai Z. Rapid and sensitive nonradioactive detection of mutant K-ras genes via 'enriched' PCR amplification. **Oncogene**. 1991; 6(6):1079-83. PMID: 1676837.
- Tobi M, Luo FC, Ronai Z. Detection of K-ras mutation in colonic effluent samples from patients without evidence of colorectal carcinoma. **J Natl Cancer Inst**. 1994; 86(13):1007-10. PMID: 8007010.
- Minamoto T, Yamashita N, Ochiai A, Mai M, Sugimura T, Ronai Z, Esumi H. Mutant K-ras in apparently normal mucosa of colorectal cancer patients. Its potential as a biomarker of colorectal tumorigenesis. **Cancer**. 1995; 75(6 Suppl):1520-6. PMID: 7889485.

Defining pathways engaged in the immediate stress response. Demonstrated the activation of stress kinases by DNA breaks signals, the role of redox (GSTp) in the control of stress kinases and that JNK-based ubiquitination control stress-activated transcription factors.

- Adler V, Fuchs SY, Kim J, Kraft A, King MP, Pelling J, Ronai Z. jun-NH2-terminal kinase activation mediated by UV-induced DNA lesions in melanoma and fibroblast cells. **Cell Growth Differ.** 1995; 6(11):1437-46. PMID: 8562482.
- Fuchs SY, Dolan L, Davis RJ, Ronai Z. Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun N-kinase. **Oncogene.** 1996; 13(7):1531-5. PMID: 8875991.
- Fuchs SY, Adler V, Buschmann T, Yin Z, Wu X, Jones SN, Ronai Z. JNK targets p53 ubiquitination and degradation in nonstressed cells. **Genes Dev.** 1998; 12(17):2658-63. PMID: PMC317120.
- Adler V, Yin Z, Fuchs SY, Benezra M, Rosario L, Tew KD, Pincus MR, Sardana M, Henderson CJ, Wolf CR, Davis RJ, Ronai Z. Regulation of JNK signaling by GSTp. **EMBO J.** 1999; 18(5):1321-34. PMID: PMC1171222.

Rewired signaling in cancer. Demonstrated cross talk between ERK and JNK, STAT3 and c-Jun, RACK1 and JNK signaling in melanoma, and rewired metabolic signaling in melanoma.

- Ivanov VN, Bhoumik A, Krasilnikov M, Raz R, Owen-Schaub LB, Levy D, Horvath CM, Ronai Z. Cooperation between STAT3 and c-jun suppresses Fas transcription. **Mol Cell.** 2001; 7(3):517-28. PMID: 11463377.
- López-Bergami P, Habelhah H, Bhoumik A, Zhang W, Wang LH, Ronai Z. RACK1 mediates activation of JNK by protein kinase C [corrected]. **Mol Cell.** 2005; 19(3):309-20. PMID: PMC2953422.
- Lopez-Bergami P, Huang C, Goydos JS, Yip D, Bar-Eli M, Herlyn M, Smalley KS, Mahale A, Eroshkin A, Aaronson S, Ronai Z. Rewired ERK-JNK signaling pathways in melanoma. **Cancer Cell.** 2007; 11(5):447-60. PMID: PMC1978100.
- Pathria G, Scott DA, Feng Y, Sang Lee J, Fujita Y, Zhang G, Sahu AD, Ruppin E, Herlyn M, Osterman AL, Ronai ZA. Targeting the Warburg effect via LDHA inhibition engages ATF4 signaling for cancer cell survival. **EMBO J.** 2018; 37(20). pii: e99735. PMID: PMC6187221.
- Pathria, G., Lee, JS., Hasnis, E., Tandoc, K., Scott, DA., Verma, S., Feng, Y., Larue, Lionel., Sahu, AD., Topisirovic, I., Ruppin, **Ronai, ZA.** Translational Reprogramming Marks Adaptation to Asparagine Restriction in Cancer. **Nat. Cell Biol.** 21(12):1590-1603, 2019.

Post-translational modification defines cancer progression. The role of protein glycosylation in tumor metastasis, the importance of PDK1 in melanoma progression, and altered protein methyl transferase (PRMT5) activity in melanoma

- Lau E, Feng Y, Claps G, Fukuda MN, Perlina A, Donn D, Jilaveanu L, Kluger H, Freeze HH, Ronai ZA. The transcription factor ATF2 promotes melanoma metastasis by suppressing protein fucosylation. **Sci Signal.** 2015; 8(406):ra124. PMID: PMC4818095.
- Scortegagna M, Ruller C, Feng Y, Lazova R, Kluger H, Li JL, De SK, Rickert R, Pellicchia M, Bosenberg M, Ronai ZA. Genetic inactivation or pharmacological inhibition of Pdk1 delays development and inhibits metastasis of Braf^{V600E}::Pten^{-/-} melanoma. **Oncogene.** 2014; 33(34):4330-9. PMID: PMC3955742.
- Tamiya H, Kim H, Klymenko O, Kim H, Feng Y, Zhang T, Han JY, Murao A, Snipas SJ, Jilaveanu L, Brown K, Kluger H, Zhang H, Iwai K, Ronai ZA. SHARPIN-mediated regulation of protein arginine methyltransferase 5 controls melanoma growth. **J Clin Invest.** 2018; 128(1):517-530. PMID: PMC5749505.

Ubiquitin ligases control of fundamental cellular processes. The role of Siah2 in hypoxia—control of prolyl hydroxylase stability (PHD1, PHD3); the role of Siah2 in UPR (unfolded protein response)—control of ATF4 signaling, implication to ischemia. The role of Siah2 in the control of mitochondrial fission, implications for heart infarct and aging and the role of Siah2 in controlling the duration of stress signaling (TRAF2/JNK). Discovered the regulation of I κ B and β -catenin stability by β -TRCP ubiquitin ligase

- Nakayama K, Frew IJ, Hagensen M, Skals M, Habelhah H, Bhoumik A, Kadoya T, Erdjument-Bromage H, Tempst P, Frappell PB, Bowtell DD, Ronai Z. Siah2 regulates stability of prolyl-hydroxylases, controls HIF1 α abundance, and modulates physiological responses to hypoxia. **Cell.** 2004; 117(7):941-52. PMID: 15210114.

- Scortegagna M, Kim H, Li JL, Yao H, Brill LM, Han J, Lau E, Bowtell D, Haddad G, Kaufman RJ, Ronai ZA. Fine tuning of the UPR by the ubiquitin ligases Siah1/2. **PLoS Genet.** 2014; 10(5):e1004348. PMID: PMC4014425.
- Kim H, Scimia MC, Wilkinson D, Trelles RD, Wood MR, Bowtell D, Dillin A, Mercola M, Ronai ZA. Fine-tuning of Drp1/Fis1 availability by AKAP121/Siah2 regulates mitochondrial adaptation to hypoxia. **Mol Cell.** 2011; 44(4):532-44. PMID: PMC3360955.
- Habelhah H, Frew IJ, Laine A, Janes PW, Relaix F, Sassoon D, Bowtell DD, Ronai Z. Stress-induced decrease in TRAF2 stability is mediated by Siah2. **EMBO J.** 2002; 21(21):5756-65. PMID: PMC131073.
- Fuchs SY, Chen A, Xiong Y, Pan ZQ, Ronai Z. HOS, a human homolog of Slimb, forms an SCF complex with Skp1 and Cullin1 and targets the phosphorylation-dependent degradation of I κ B and β -catenin. **Oncogene.** 1999; 18(12):2039-46. PMID: 10321728.

Ubiquitin ligases in cancer (development, therapy-resistance). Established the role of Siah2 in melanoma development and metastasis, and in the development of neuroendocrine prostate cancers through HIF signaling, and in the development of hormone refractory prostate via control of androgen receptor activity. Discovered the importance of RNF5 ubiquitin ligase in the control of glutamine carrier proteins, and its importance for BCa responsiveness taxenes, study that provided the basis for multicenter clinical trials. Demonstrated the importance of RNF125 ubiquitin ligase in melanoma resistance to BRAF inhibitor.

- Qi J, Nakayama K, Gaitonde S, Goydos JS, Krajewski S, Eroshkin A, Bar-Sagi D, Bowtell D, Ronai Z. The ubiquitin ligase Siah2 regulates tumorigenesis and metastasis by HIF-dependent and -independent pathways. **Proc Natl Acad Sci USA.** 2008; 105(43):16713-8. PMID: PMC2575485.
- Qi J, Nakayama K, Cardiff RD, Borowsky AD, Kaul K, Williams R, Krajewski S, Mercola D, Carpenter PM, Bowtell D, Ronai ZA. Siah2-dependent concerted activity of HIF and FoxA2 regulates formation of neuroendocrine phenotype and neuroendocrine prostate tumors. **Cancer Cell.** 2010; 18(1):23-38. PMID: PMC2919332.
- Qi J, Tripathi M, Mishra R, Sahgal N, Fazli L, Ettinger S, Placzek WJ, Claps G, Chung LW, Bowtell D, Gleave M, Bhowmick N, Ronai ZA. The E3 ubiquitin ligase Siah2 contributes to castration-resistant prostate cancer by regulation of androgen receptor transcriptional activity. **Cancer Cell.** 2013; 23(3):332-46. PMID: PMC3750989.
- Jeon YJ, Khelifa S, Ratnikov B, Scott DA, Feng Y, Parisi F, Ruller C, Lau E, Kim H, Brill LM, Jiang T, Rimm DL, Cardiff RD, Mills GB, Smith JW, Osterman AL, Kluger Y, Ronai ZA. Regulation of glutamine carrier proteins by RNF5 determines breast cancer response to ER stress-inducing chemotherapies. **Cancer Cell.** 2015; 27(3):354-69. PMID: PMC4356903.
- Kim H, Frederick DT, Levesque MP, Cooper ZA, Feng Y, Krepler C, Brill L, Samuels Y, Hayward NK, Perlina A, Piris A, Zhang T, Halaban R, Herlyn MM, Brown KM, Wargo JA, Dummer R, Flaherty KT, Ronai ZA. Downregulation of the ubiquitin ligase RNF125 underlies resistance of melanoma cells to BRAF inhibitors via JAK1 deregulation. **Cell Rep.** 2015; 11(9):1458-73. PMID: PMC4681438.
- Scortegagna M, Hockemeyer K, Dolgalev I, Poźniak J, Rambow F, Li Y, Feng Y, Tinoco R, Otero DC, Zhang T, Brown K, Bosenberg M, Bradley LM, Marine JC, Aifantis I, Ronai ZA. Siah2 control of T-regulatory cells limits anti-tumor immunity. **Nat Commun.** 2020 Jan 7;11(1):99. doi: 10.1038/s41467-019-13826-7

Subcellular localization/genomic organization define oncogenic/tumor suppressor signaling. Carved the paradigm of subcellular localization importance in defining oncogenic or tumor suppressor activity by demonstrating that PKC ϵ defines ATF2 subcellular localization and activity as oncogene (nuclear) or tumor suppressor (cytosolic). Identified ATF2 splice variant, lacking transcriptional capabilities, as a super-oncogene in melanoma and ERK-dependent cytoplasmic accumulation of hnRNP-K limits mRNA translation

- Habelhah H, Shah K, Huang L, Ostareck-Lederer A, Burlingame AL, Shokat KM, Hentze MW, Ronai Z. ERK phosphorylation drives cytoplasmic accumulation of hnRNP-K and inhibition of mRNA translation. **Nat Cell Biol.** 2001; 3(3):325-30. PMID: 11231586.
- Lau E, Kluger H, Varsano T, Lee K, Scheffler I, Rimm DL, Ideker T, Ronai ZA. PKC ϵ promotes oncogenic functions of ATF2 in the nucleus while blocking its apoptotic function at mitochondria. **Cell.** 2012; 148(3):543-55. PMID: PMC3615433.

- Claps G, Cheli Y, Zhang T, Scortegagna M, Lau E, Kim H, Qi J, Li JL, James B, Dzung A, Levesque MP, Dummer R, Hayward NK, Bosenberg M, Brown KM, Ronai ZA. A transcriptionally inactive ATF2 variant drives melanomagenesis. *Cell Rep.* 2016; 15(9):1884-92. PMID: PMC4889472.

Ubiquitin ligase in the interplay between autoimmunity and anti-tumor immunity. *Demonstrated that RNF5 defines the gut microbiota's ability to induce anti-tumor immunity and tumor inhibition while the same ubiquitin ligase defines susceptibility to intestinal inflammation (IBD like phenotypes).*

- Li Y, Tinoco R, Elmén L, Segota I, Xian Y, Fujita Y, Sahu A, Zarecki R, Marie K, Feng Y, Khateb A, Frederick DT, Ashkenazi SK, Kim H, Perez EG, Day CP, Segura Muñoz RS, Schmaltz R, Yooseph S, Tam MA, Zhang T, Avitan-Hersh E, Tzur L, Roizman S, Boyango I, Bar-Sela G, Orian A, Kaufman RJ, Bosenberg M, Goding CR, Baaten B, Levesque MP, Dummer R, Brown K, Merlino G, Ruppin E, Flaherty K, Ramer-Tait A, Long T, Peterson SN, Bradley LM, Ronai ZA. Gut microbiota dependent anti-tumor immunity restricts melanoma growth in Rnf5^{-/-} mice. *Nat Commun.* 2019; 10(1):1492. PMID: PMC6445090.
- Fujita Y, Khateb A, Li Y, Tinoco R, Zhang T, Bar-Yoseph H, Tam MA, Chowers Y, Sabo E, Gerassy-Vainberg S, Starosvetsky E, James B, Brown K, Shen-Orr SS, Bradley LM, Tessier PA, Ronai ZA. Regulation of S100A8 stability by RNF5 in intestinal epithelial cells determines intestinal inflammation and severity of colitis. *Cell Rep.* 2018; 24(12):3296-3311.e6. PMID: PMC6185744.
- Li Y, Elmén L, Segota I, Xian Y, Tinoco R, Feng Y, Fujita Y, Segura Muñoz RR, Schmaltz R, Bradley LM, Ramer-Tait A, Zarecki R, Long T, Peterson SN, **Ronai ZA.** Prebiotic-Induced Anti-tumor Immunity Attenuates Tumor Growth. *Cell Rep.* 2020 Feb 11;30(6):1753-1766.

Targeting the undruggable. *Developing inhibitors to transcription factor ATF2, altering its subcellular localization, developing inhibitors against the ubiquitin ligase Siah2 and novel class inhibitors for the translation initiation complex.*

- Bhoumik A, Huang TG, Ivanov V, Gangi L, Qiao RF, Woo SL, Chen SH, Ronai Z. An ATF2-derived peptide sensitizes melanomas to apoptosis and inhibits their growth and metastasis. *J Clin Invest.* 2002; 110(5):643-50. PMID: PMC151112.
- Varsano T, Lau E, Feng Y, Garrido M, Milan L, Heynen-Genel S, Hassig CA, Ronai ZA. Inhibition of melanoma growth by small molecules that promote the mitochondrial localization of ATF2. *Clin Cancer Res.* 2013; 19(10):2710-22. PMID: PMC3690798.
- Feng Y, Pinkerton AB, Hulea L, Zhang T, Davies MA, Grotegut S, Cheli Y, Yin H, Lau E, Kim H, De SK, Barile E, Pellecchia M, Bosenberg M, Li JL, James B, Hassig CA, Brown KM, Topisirovic I, Ronai ZA. SBI-0640756 attenuates the growth of clinically unresponsive melanomas by disrupting the eIF4F translation initiation complex. *Cancer Res.* 2015; 75(24):5211-8. PMID: PMC4681635.
- Stebbins JL, Santelli E, Feng Y, De SK, Purves A, Motamedchaboki K, Wu B, Ronai ZA, Liddington RC, Pellecchia M. Structure-based design of covalent Siah inhibitors. *Chem Biol.* 2013; 20(8):973-82. PMID: PMC3763817.
- Feng Y, Sessions EH, Zhang F, Ban F, Placencio-Hickok V, Ma CT, Zeng FY, Pass I, Terry DB, Cadwell G, Bankston LA, Liddington RC, Chung TDY, Pinkerton AB, Sergienko E, Gleave M, Bhowmick NA, Jackson MR, Cherkasov A, Ronai ZA. Identification and characterization of small molecule inhibitors of the ubiquitin ligases Siah1/2 in melanoma and prostate cancer cells. *Cancer Lett.* 2019; 449:145-162. PMID: PMC6411447.

Recent reviews related to our fields of interest (out of over 50):

- UPR, autophagy, and mitochondria crosstalk underlies the ER stress response. *Trends Biochem Sci.* 2015; 40(3):141-8 PMID: PMC4340752.
- Adaptive stress responses during tumor metastasis and dormancy. *Trends Cancer.* 2016 Aug;2(8):429-442. PMID: PMC5114008.
- Precision oncology: the road ahead. *Trends Mol Med.* 2017; 23(10):874-898. PMID: PMC5718207.
- Ubiquitin ligases in oncogenic transformation and cancer therapy. *Nat Rev Cancer.* 2018; 18(2):69-88. PMID: PMC6054770.
- Ubiquitin ligases in cancer immunotherapy - balancing antitumor and autoimmunity. *Trends Mol Med.* 2019; 25(5):428-443. PMID: PMC6488401.

Complete List of Published Work in My Bibliography:

<https://www.ncbi.nlm.nih.gov/sites/myncbi/ze'ev.ronai.1/bibliography/47757767/public/?sort=date&direction=descending>

Lab web page – www.ronailab.net

Research Support

Ongoing Research Support

5 P01 CA128814-09 05/05/2016 – 04/30/2021

NIH/NCI

PI Project 1: Ronai, Z.

PI Core A: Ronai, Z.

ER Stress and Mitochondrial Biogenesis in Melanoma

Goals: To identify new therapeutic strategies for overcoming drug resistance and metastatic disease in melanoma. CO-PI, Drs. Karin (UCSD), Bosenberg (Yale), Kelly (Penn), Goding (Oxford), Koumenis (Penn).

5 R35 CA197465-04 (PI: Ronai, Z.) 02/02/2016 – 01/31/2023

NIH/NCI

Rewired Signaling at the Nexus Melanoma Metastasis and Resistance

Goal: To establish novel mechanisms underlying tumor plasticity, enabling the development of novel agents for predicting, monitoring, and preventing tumor metastasis and resistance.

5 R01 CA202021-04 (PI: Ronai, Z.) 07/01/2016 – 06/30/2021

NIH/NCI

Control of Protein Synthesis by the UPS Under Stress

Goal: The major goal of this project is to establish the importance and significance of select UPS components in a novel regulatory network that controls protein synthesis during cellular stress. Collaborators: Drs. Sonenberg (McGill) and Topisirovic (Lady Davis).

W81XWH-18-1-0216 (PI: Ronai, Z) 08/15/2018 – 08/14/2020

DOD

Siah2 Ubiquitin Ligase in Immune Checkpoint and Melanomagenesis

Goal: characterize the regulation of anti-tumor immunity by the ubiquitin ligase Siah2, using melanoma models.

T3OIP0941 (PI: Ronai, Z) 10/22/2019 – 10/21/2021

TRDRP

Role of the E3 ubiquitin ligase RNF125 in pancreatic cancer

Goal: define the role of RNF125 in pancreatic cancer development and progression.