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: Gong, Jun

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

POSITION TITLE: Assistant 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
University of California, Los Angeles	B.S.	06/2007	Biology
New York Medical College	M.D.	05/2012	Medicine
Cedars-Sinai Medical Center	Residency	06/2015	Internal Medicine
City of Hope National Medical Center	Fellowship	06/2018	Hematology/Oncology

A. Personal Statement

I have had 10 years of basic science laboratory experience with familiarity in techniques such as embryo isolation, tissue sectioning, immunohistochemistry, H&E staining, cell counting, animal breeding and maintenance, cell collection and culture, RNA collection, RT-PCR, real-time PCR, Western blotting, and immunofluorescence staining through studies on fruit fly genetics, mice embryonic development, and mouse models of pancreatitis and pancreatic cancer. Since the beginning of my medical training, I have focused on translational and clinical cancer research in gastrointestinal (GI), hepatobiliary, and genitourinary (GU) cancers with experience in preclinical and early-phase clinical development of potential therapeutic targets and anticancer therapies. My long-term career goal is to become an independent translational researcher and clinical trialist focused on improving outcomes in GI and GU cancer patients through early therapeutic trials and biomarker development.

B. Positions and Honors

Positions and Employment

2004-2007 Researcher, Department of Molecular, Cell & Developmental Biology and Department of Psychiatry and Biobehavioral Sciences, UCLA, Los Angeles, CA
2005-2007 Laboratory Assistant, Pancreatic Research Group, West Los Angeles Veterans Affairs Medical Center, Los Angeles, CA
2007-2008 Laboratory Technician, Pancreatic Research Group, West Los Angeles Veterans Affairs Medical Center, Los Angeles, CA
2012-2014 Researcher, Basic and Translational Pancreas Research, Department of Medicine, Cedars-Sinai Medical Center, Los Angeles, CA
2012-2013 Intern, Department of Medicine, Cedars-Sinai Medical Center, Los Angeles, CA
2013-2015 Resident, Department of Medicine, Cedars-Sinai Medical Center, Los Angeles, CA
2015-2018 Fellow, Department of Hematology and Oncology, City of Hope National Medical Center/Harbor-UCLA, Duarte, CA
2018- Assistant Professor, Department of Medicine, Division of Hematology and Oncology, Cedars-Sinai Medical Center, Los Angeles, CA

Professional Memberships and Activities

2012- Member, American Medical Association
2013- Member, American College of Physicians
2013- Member, American Association for Cancer Research
2014- Editor, International Journal of Blood Research and Disorders
2015- Member, American Society of Clinical Oncology
2016- Member, European Society for Medical Oncology
2016- Ad hoc Reviewer, Oncotarget
2017- Ad hoc Reviewer, Kidney Cancer

Honors

2007 Best Abstract Award, University of Southern California Research Center for Alcoholic Liver and Pancreatic Diseases and Cirrhosis 9th Annual Symposium, Los Angeles, CA
2008 Merit Scholarship, New York Medical College, Valhalla, NY
2016 Travel Award, 15th International Kidney Cancer Symposium, Kidney Cancer Association, Miami, FL
2016 Young Investigator's Think Tank, 15th International Kidney Cancer Symposium, Kidney Cancer Association, Miami, FL
2017 Participant, 2017 NCCN Oncology Fellows Program
2017 Participant, ASCO/AACR Workshop on Methods in Clinical Cancer Research, Vail, CO

C. Contributions to Science

1. Mechanisms of Pancreatitis and Pancreatic Cancer: Our research involved understanding the molecular mechanisms of pancreatitis and mechanisms of risk factor promotion of pancreatic cancer. Here, I gained basic science laboratory experience with animal breeding and maintenance, RNA collection, RT-PCR, real-time PCR, immunofluorescence staining, isolation, culturing, and treatment of pancreatic stellate cells, gel electrophoresis, and Western blotting. We were able to characterize the role of the cholinergic system and unfolded protein response in mediating alcohol-induced pancreatitis in rodent models. We were also able to identify a putative role of leptin and the pancreatic stellate cell in facilitating risk factor (obesity) promotion of pancreatic cancer in rodents. Given the extremely poor prognosis of pancreatic cancer, methods of preventing this disease could serve as an alternative method to improve outcomes in this area. Our research contributed to understanding of molecular mechanisms tying obesity to pancreatic cancer promotion.
    a. Lugea, A., Gong, J., Nguyen, J., Nieto, J., French, S.W., & Pandol, S.J. (2010). Cholinergic mediation of alcohol-induced experimental pancreatitis. Alcoholism: Clinical and Experimental Research, 34(10), 1768-1781.
    b. b. Lugea, A., Tischler, D., Nguyen, J., Gong, J., Gukovsky, I., French, S.W., Gorelick, F.S., & Pandol, S.J. (2011). Adaptive unfolded protein response attenuates alcohol-induced pancreatic damage. Gastroenterology, 140(3), 987-997.
    c. c. Gong, J., Gong, R., Waldron, R.T., Lugea, A., & Pandol, S.J. (2014). Leptin regulates cell differentiation and protumorigenic responses in pancreatic stellate cells [abstract]. Cancer Research, 74(19 Suppl), abstract nr 5353. doi:10.1158/1538-7445.AM2014-5353.

2. Biomarker Development: Immunotherapies such as nivolumab and pembrolizumab have been shown to benefit patients with metastatic colorectal cancer (mCRC) that are microsatellite instability-high (MSI-H) or have high tumor mutational burden (TMB). Interrogation of City of Hope's (COH) next-generation sequencing (NGS) database of CRC tissues allowed us to describe molecular signatures that may have potential prognostic and therapeutic implications. Genomic alterations of potential clinical significance, MSI, and TMB were correlated according to patient clinicopathologic features to identify clinically actionable subsets of CRC patients. We have since performed immune profiling of COH's cohort of MSI and *POLE*-mutated mCRC patients to describe potential predictors of response to checkpoint inhibitors. The gut microbiome may also represent a predictive biomarker for checkpoint blockade and other systemic therapies. We performed preliminary investigations at COH into the relationship between the stool microbiome and response to sunitinib in metastatic renal cell carcinoma (mRCC) patients. Hyaluronic acid

(HA) expression has shown to predict response to HA-targeted therapy and gemcitabine/nab-paclitaxel in advanced pancreatic cancer. We performed an initial investigation at Cedars-Sinai Medical Center (CSMC) assessing the potential of HA expression as a biomarker in a large, predominantly metastatic lung cancer cohort with hypothesis-generating findings.

a. Gong, J., Sy, M., & Fakih, M. (2016). The genomic complexity of metastatic colorectal tumors by age, gender, race, and location of primary tumor using next-generation sequencing [abstract]. Annals of Oncology, 27(6 Suppl), abstract nr 538P. doi:10.1093/annonc/mdw370.86.
b. Gong, J., Cho, M., Sy, M., Salgia, R., & Fakih, M. (2017). Molecular profiling of metastatic colorectal tumors using next-generation sequencing: A single-institution experience. Oncotarget, doi:10.18632/oncotarget.15030.
c. Gong, J., Sy, M., & Fakih, M. (2017). Tumor mutational burden of microsatellite stable metastatic colorectal tumors by patient factors and KRAS, BRAF, and PIK3CA mutation status [abstract]. Journal of Clinical Oncology, 35(4S Suppl), abstract nr 627.
d. Gong, J., Dizman, N., Poroyko, V., Won, H., Bergerot, C.D., Bergerot, P.G., Maia, M.C., Hsu, J., Frankel, P.H., Jones, J., Salgia, R., & Pal, S.K. (2018). Gut microbiome composition and response to sunitinib in metastatic renal cell carcinoma (mRCC) [abstract]. Journal of Clinical Oncology, 36(6S Suppl), abstract nr 657.
e. Gong, J., Guan, M., Cook-Wiens, G., Larson, B.K., Zhou, J., Patel, R., Lapite, I., Tuli, R., Natale, R.B., & Hendifar, A. (2018). Tumor hyaluronan (HA) as a novel biomarker in non-small cell lung cancer (NSCLC) [abstract]. Journal of Clinical Oncology, 36(15 Suppl), abstract nr e24280.
f. Wang, C., Gong, J., Lee, P.P., & Fakih, M. (2018). Immune profiling of microsatellite instability-high and polymerase ε (POLE)-mutated metastatic colorectal tumors identifies predictors of response to anti-PD-1 therapy. Journal of Gastrointestinal Oncology, doi:10.21037/jgo.2018.01.09.

3. Early-Phase Therapeutic Development: In recognizing that *RAS*-mutated colorectal tumors represent a large population of patients in need of further treatment options, particularly in chemorefractory settings, we conducted preclinical studies in *RAS*-mutated CRC cells and demonstrated evidence of synergism for the combination of MEK inhibitor and TAS-102. Our findings provided the rationale for initiation of a phase I trial entitled: “A phase I clinical trial of trametinib in combination with TAS-102 in patients with chemotherapy-resistant RAS-mutated (PIK3CA/PTEN-wild-type) metastatic colorectal cancer” that is IRB-approved and actively enrolling as of April 2018 at COH. Preclinical data has also shown that MEK inhibition could reverse platinum resistance in CRC models supporting the rational combination of oxaliplatin-based therapy and MEK inhibition. We conducted a phase I trial investigating the combination of FOLFOX and binimetinib (MEK1/2 inhibitor) in chemorefractory mCRC and showed that this combination was feasible and had promising efficacy in an unselected population of mCRC. Advancement of these combinations could serve an unmet need in mCRC patients in the treatment-refractory settings. I was also involved in an early therapeutics trial in bladder cancer for which therapies in gemcitabine-resistant metastatic patients are sorely needed. We were able to demonstrate the safety and tolerability and preliminary efficacy of RX-3117, an oral antimetabolite nucleoside, in patients with metastatic bladder cancer resistant to gemcitabine. Continued development of this agent could potentially serve an unmet need in the second-line and beyond treatment of metastatic bladder cancer.

a. Cho, M., Gong, J., Frankel, P., Synold, T., Lim, D., Chung, V., Chao, J., Li, D., Chen, Y., Sentovich, S., Melstrom, K., Singh, G., Luevanos, E., & Fakih, M. (2017). A phase I clinical trial of binimetinib in combination with FOLFOX in patients with advanced metastatic colorectal cancer who failed prior standard therapy. Oncotarget, https://doi.org/10.18632/oncotarget.19336.
b. Gong, J., Chen, Y., Yang, L., Pillai, R., Shirasawa, S., & Fakih, M. (2017). MEK162 enhances 5-fluorouracil or trifluridine antitumor activity in KRAS-mutated human colorectal cancer cell lines. Anticancer Research, 37(6), 2831-2838.
c. Gong, J., Chung, V.M., Picus, J., Tagawa, S.T., Gupta, S., Poore, J., Peterson, C., Benaim, E., & Pal, S.K. (2017). Activity of RX-3117, an oral antimetabolite nucleoside, in subjects with metastatatic bladder cancer resistant to gemcitabine: Preliminary results of a phase Ia/IIb study [abstract]. Journal of Clinical Oncology, 35(15S Suppl), abstract nr 4544.

4. Renal Cell Carcinoma and Prostate Cancer Outcomes Research: Vascular endothelial growth factor (VEGF) tyrosine kinase inhibitors (TKIs) have seen widespread implementation in the treatment of mRCC. However, real-world data describing toxicities and provider preferences are relatively limited. We

interrogated a large U.S. claims-based database and performed a longitudinal analysis to describe provider preferences, toxicities, and cost analyses related to VEGF-TKIs in mRCC. We identified that comorbidities including heart failure and diabetes independently affected provider prescriber patterns for VEGF-TKIs and mTOR inhibitors, described costs and health resource utilization for VEGF-TKIs and mTOR inhibitors, and identified substantial latency observed with the onset of toxicities typically observed with initiation of anti-angiogenesis therapies. Our findings provide real-world context that inform ongoing trials in mRCC involving anti-angiogenesis therapies and combination therapies with immunotherapy. The Advanced Prostate Cancer Consensus Conference (APCCC) 2018 convened an international panel of leading prostate cancer experts for consensus voting on a series of controversial topics in advanced prostate cancer (APC). We performed an analysis of attendee voting patterns before and after APCCC and reported interesting changes in provider preferences in APC management following the conference.

a. Gong, J., George, D., Mhatre, S., Lin, S-W., Surinach, A., Wallen, H., Vohra, R., Simpson, J., Ogale, S., & Pal, S.K. (2016). Influence of diabetes and congestive heart failure (CHF) on selection of first-line (1L) treatment for metastatic renal cell carcinoma (mRCC) [abstract]. Annals of Oncology, 27(6 Suppl), abstract nr 810P. doi:10.1093/annonc/mdw373.37.
b. Gong, J., George, D., Wallen, H., Mhatre, S., Lin, S.W., Ogale, S., Vohra, R., Surinach, A., Simpson, J., & Pal, S.K. (2016, November). Latency of potential treatment-related adverse events among patients treated with TKI/VEGF-directed therapy for metastatic renal carcinoma (mRCC). Oral presentation at the 15th International Kidney Cancer Symposium, Miami, FL.
c. Mhatre, S., Lin, S., Surinach, A., Vohra, R., Satram-Hoang, S., Simpson, J., Wallen, H., Ogale, S., Gong, J., Pal, S.K., & George, D.J. (2016). Health resource utilization (HRU) and costs for metastatic renal cell carcinoma (MRCC) patients treated with first-line (1L) systemic therapy in the United States [abstract]. Value in Health, 19(7), A755.
d. Gong, J., Omlin, A., Pal, S.K., Hsu, J., Tombal, B., Sydes, M.R., & Gillessen, S. (2018). Influence of an international consensus conference on practice patterns in advanced prostate cancer. European Urology, 74(2), 239-240. doi:10.1016/j.eururo.2018.04.024.

D. Additional Information: Research Support and/or Scholastic Performance

Complete List of Published Work in MyBibliography:
https://www.ncbi.nlm.nih.gov/sites/myncbi/18ahsxPnqtrAj/bibliography/53258625/public/?sort=date&direction=descending