BIOGRAPHICAL SKETCH

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NAME: Zeinomar, Nur

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

POSITION TITLE: Postdoctoral Research Scientist

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
University of Florida, Gainesville, FL	BS	12/2005	Microbiology and Cell Science
University of Florida, Gainesville, FL	MPH	05/2008	Public Health, Epidemiology
University at Albany, State University of New York, Albany, NY	PHD	05/2014	Chronic Disease Epidemiology
Mailman School of Public Health, Columbia University, New York, NY	Postdoctoral Fellow	08/2020	Cancer and Molecular Epidemiology, Precision Medicine

A. Personal Statement

Broadly, my research focuses on understanding the role of genetics, the environment, lifestyle factors, and their interplay on breast cancer etiology and prognosis. Currently, I am an Assistant Professor at the Cancer Institute of New Jersey (CINJ). In addition to my doctoral training in epidemiology, I have postdoctoral training in molecular epidemiology and precision medicine, as well as a foundation in microbiology and cell science from my undergraduate training. The ultimate aim of my research is to translate epidemiologic findings into clinical care through more accurate risk assessment and to optimize risk-reducing strategies for outcomes after a breast cancer diagnosis. My research is currently addressing ways to improve risk assessment after a breast cancer diagnosis through integration of genomic data and other relevant factors and is funded by a National Cancer Institute (NCI) K22 award to support this effort (CA251493-01A1). Specifically, I will investigate whether a polygenic risk score improves risk prediction of survival over and beyond standard clinical markers and tumor characteristics, utilizing a large international family-based cohort of over 6,000 high-risk women with breast cancer (PI: Terry) and the Women's Circle of Health Follow-Up Study (WCHFS), an ongoing population-based longitudinal study of over 1900 Black breast cancer survivors in New Jersey, based at Rutgers CINJ (PI: Bandera). More recently, the New Jersey Breast Cancer Survivors Study (NJBCSS) was initiated with the primary aim of demonstrating that we can recruit Black and Hispanic breast cancer survivors in New Jersey during the COVID-19 pandemic, with bridge funding from the New Jersey Commission on Cancer Research (PI: Bandera). I was awarded an internal Rutgers pilot award to leverage NJBCSS, which uses the same methodology and protocols as WCHFS, to add a microbiome component and examine the feasibility of collecting fecal/stool samples in Black and Hispanic breast cancer survivors in NJ. Given the plasticity of the gut microbiome and the fact that the environments that drive the composition of the gut microbiome may be modifiable through both therapeutic interventions and lifestyle modifications, the microbiome may have important clinical implications in terms of precision medicine. More recently, I will extend this microbiome research to an important and understudied population, South Asian breast cancer survivors, through funding from the New Jersey Health foundation. Overall, my academic training and research experience as a cancer epidemiologist to date provides a strong foundation for uniquely addressing ways to improve risk assessment after BC diagnosis through integration of genomic data and other relevant factors as well as plan future prevention programs that require empirical evidence from large studies of gene-environment interactions.

1. **Zeinomar N**, Qin B, Amin S, Lin Y, Xu B, Chanumolu D, Omene CO, Pawlish KS, Demissie K, Ambrosone CB, Hong CC, Bandera EV. Association of Cigarette Smoking and Alcohol Consumption With Subsequent Mortality Among Black Breast Cancer Survivors in New Jersey. JAMA Netw Open. 2023 Jan 3;6(1):e2252371. PubMed PMID: 36692882.

2. Liu C*, **Zeinomar N***, Chung WK, Kiryluk K, Gharavi AG, Hripcsak G, Crew KD, Shang N, Khan A, Fasel D, Manolio TA, Jarvik GP, Rowley R, Justice AE, Rahm AK, Fullerton SM, Smoller JW, Larson EB, Crane PK, Dikilitas O, Wiesner GL, Bick AG, Terry MB, Weng C. Generalizability of Polygenic Risk Scores for Breast Cancer Among Women With European, African, and Latinx Ancestry. JAMA Netw Open. 2021 Aug 2;4(8):e2119084. PubMed Central PMCID: PMC8339934. ***co-first authors***
3. **Zeinomar N**, Chung WK. Cases in Precision Medicine: The Role of Polygenic Risk Scores in Breast Cancer Risk Assessment. Ann Intern Med. 2021 Mar;174(3):408-412. PubMed Central PMCID: PMC7965355.
4. **Zeinomar N**, Phillips KA, Daly MB, Milne RL, Dite GS, MacInnis RJ, Liao Y, Kehm RD, Knight JA, Southey MC, Chung WK, Giles GG, McLachlan SA, Friedlander ML, Weideman PC, Glendon G, Nesci S, Andrulis IL, Buys SS, John EM, Hopper JL, Terry MB. Benign breast disease increases breast cancer risk independent of underlying familial risk profile: Findings from a Prospective Family Study Cohort. Int J Cancer. 2019 Jul 15;145(2):370-379. PubMed Central PMCID: PMC6525034.

B. Positions, Scientific Appointments and Honors

Positions and Scientific Appointments

2022 – present	Assistant Professor of Medicine, Rutgers Cancer Institute of New Jersey (CINJ), Robert Wood Johnson Medical School, New Brunswick, NJ
2020 – present	Associate Member, Cancer Prevention and Control (CPC) Research Program, Rutgers CINJ
2020 – present	Member, Center for Cancer Health Equity, Rutgers CINJ
2021 – present	Member, Center of Excellence in Cancer Survivorship, Rutgers CINJ, New Brunswick, NJ
2020 – 2022	Instructor of Medicine, Rutgers Cancer Institute of New Jersey, Robert Wood Johnson Medical School, New Brunswick, NJ
2018 – 2020	TL1 Postdoctoral Research Fellow, Irving Institute for Clinical and Translational Research (CTSA), Columbia University Irving Medical Center, Mailman School of Public Health, Department of Epidemiology, New York, NY
2015 – 2018	R25/T32 Post-Doctoral Research Scientist in Molecular and Cancer Epidemiology, Department of Epidemiology, Columbia University Mailman School of Public Health, New York, NY
2010 – 2011	Graduate Teaching Assistant, University at Albany, Department of Epidemiology and Biostatistics
2009 – 2009	Graduate Research Assistant, New York State Department of Health, Bureau of Environmental and Occupational Epidemiology, Center for Environmental Health, Albany, NY
2007 – 2008	Graduate Research Assistant, University of Florida, Gainesville, FL
2007 – 2008	Research Intern, Southeastern National Tuberculosis Center, Gainesville, FL

Honors

2002 – 2005	Florida Bright Futures Scholarship, Florida Department of Education
2018	New Investigators Workshop Awardee, American Society for Preventative Oncology (ASPO)
2018	Award for Top-ranked Poster, Breast Cancer and the Environment Research Program (BCERP)
2017	Scholar in Training Travel Award to San Antonio Breast Cancer Symposium (SABCS), American Association for Cancer Research (AACR)
2013	David Axelrod Excellence in Research Award, University at Albany, SUNY
2013	Louise C. and Earl M. Applegate Award, Initiatives for Women, University at Albany, SUNY

C. Contribution to Science

1. Incorporating underlying susceptibility measures into cancer etiologic studies of modifiable factors

Both genetic and lifestyle factors are implicated in breast cancer (BC) etiology. While the discovery of high-penetrant genes such as BRCA1 and BRCA2 have had important implications on BC screening and treatment, they only explain a small portion of the familial risk for BC. This “missing heritability” in BC has

spurred extensive research examining the genetic predisposition to BC. We analyzed a database of more than 50,000 women who had undergone multi-gene hereditary cancer testing between 2013 and 2015 to examine the role of four Lynch syndrome genes MLH1, MSH2, MSH6, and PMS2 in BC risk. We found that women with a mutation in two specific Lynch syndrome genes, MSH6 and PMS2, had a two-fold higher risk of BC compared to women in the general population. This new study suggests MSH6 and PMS2 should be considered in future gene panels screening women with a family history of BC. This study also highlights the value of integrating epidemiologic methods and databases, like Surveillance Epidemiology and End Results (SEER) with clinical genetic databases. I also have used the prospective family study cohort (ProF-SC) to evaluate how risk associated with benign breast disease (BBD) is modified by underlying familial risk so as to guide clinical management and risk assessment of women with BBD. While relative risk of BC by BBD status did not differ by underlying familial risk profile, I still found absolute risk differences, with the largest absolute risk occurring in those at the highest end of the risk spectrum. In other words, the predicted cumulative BC risk to age 80 years is 14.8% for women with BBD at average population risk (12% lifetime risk) and increases to 37.2% for women with both BBD and high familial risk (30% lifetime risk). This study underscores the importance of also incorporating absolute risk estimates when examining two well-established risk factors and communicating risk with women across the familial risk spectrum. An abstract of these study findings was awarded an AACR Scholar in Training Award travel award and was recently presented at the San Antonio Breast Cancer Symposium (SABCS), and the final manuscript was published in the International Journal of Cancer.

a. **Zeinomar N**, Phillips KA, Daly MB, Milne RL, Dite GS, MacInnis RJ, Liao Y, Kehm RD, Knight JA, Southey MC, Chung WK, Giles GG, McLachlan SA, Friedlander ML, Weideman PC, Glendon G, Nesci S, Andrulis IL, Buys SS, John EM, Hopper JL, Terry MB. Benign breast disease increases breast cancer risk independent of underlying familial risk profile: Findings from a Prospective Family Study Cohort. Int J Cancer. 2019 Jul 15;145(2):370-379. PubMed Central PMCID: PMC6525034.
b. Terry MB, Liao Y, Whittemore AS, Leoce N, Buchsbaum R, **Zeinomar N**, Dite GS, Chung WK, Knight JA, Southey MC, Milne RL, Goldgar D, Giles GG, McLachlan SA, Friedlander ML, Weideman PC, Glendon G, Nesci S, Andrulis IL, John EM, Phillips KA, Daly MB, Buys SS, Hopper JL, MacInnis RJ. 10-year performance of four models of breast cancer risk: a validation study. Lancet Oncol. 2019 Apr;20(4):504-517. PubMed PMID: 30799262; NIHMSID: NIHMS1523036.
c. Roberts ME, Jackson SA, Susswein LR, **Zeinomar N**, Ma X, Marshall ML, Stettner AR, Milewski B, Xu Z, Solomon BD, Terry MB, Hruska KS, Klein RT, Chung WK. MSH6 and PMS2 germ-line pathogenic variants implicated in Lynch syndrome are associated with breast cancer. Genet Med. 2018 Oct;20(10):1167-1174. PubMed Central PMCID: PMC6051923.
d. **Zeinomar N,** Thai A, Cloud AJ, McDonald JA, Liao Y, Terry MB. Alcohol consumption and breast cancer-specific and all-cause mortality in women diagnosed with breast cancer at the New York site of the Breast Cancer Family Registry. PLoS One. 2017;12(12):e0189118. PubMed Central PMCID: PMC5731703.

2. Germline genetic variation

My research since my doctoral work and now through my currently funded NCI K22 project has examined germline genetic variation for breast cancer risk and prognosis. My early work used genome wide association studies (GWAS) to examine the association of post-menopausal BC and genetic variation (individual single nucleotide polymorphisms (SNPs), haplotypes, diplotypes, and diplotype-diplotype interactions, and SNP-sets) within chosen candidate DNA repair genes, identifying associations with breast cancer, particularly within the ERCC6 gene. During my postdoctoral fellowship at Columbia, I collaborated with the Electronic Medical Records and Genomics (eMERGE) project investigators in Columbia's Department of Bioinformatics where I co-lead a project examining the performance of validated European-based polygenic risk scores (PRS) for BC risk in individuals across different ancestries and in different settings. In this multicenter cohort study linking electronic medical records to genotyping data that including 39,591 women, PRSs were significantly associated with breast cancer risk in women of all ancestries, although the effect sizes were smaller in women with African ancestry. Our results highlighted the need to improve representation of diverse population groups, particularly African Ancestry women, in genomic research cohorts. Our study was one of the first to evaluate the performance of breast cancer PRSs using clinical data extracted from the electronic medical records.

a. Liu C*, **Zeinomar N***, Chung WK, Kiryluk K, Gharavi AG, Hripcsak G, Crew KD, Shang N, Khan A, Fasel D, Manolio TA, Jarvik GP, Rowley R, Justice AE, Rahm AK, Fullerton SM, Smoller JW, Larson EB, Crane PK, Dikilitas O, Wiesner GL, Bick AG, Terry MB, Weng C. Generalizability of Polygenic Risk Scores for Breast Cancer Among Women with European, African, and Latinx Ancestry. JAMA Netw Open. 2021 Aug 2;4(8):e2119084. PubMed Central PMCID: PMC8339934. ***co-first authors***

b. **Zeinomar N**, Chung WK. Cases in Precision Medicine: The Role of Polygenic Risk Scores in Breast Cancer Risk Assessment. Ann Intern Med. 2021 Mar;174(3):408-412. PubMed Central PMCID: PMC7965355.

c. Moslehi R, Tsao HS, **Zeinomar N**, Stagnar C, Fitzpatrick S, Dzutsev A. Integrative genomic analysis implicates ERCC6 and its interaction with ERCC8 in susceptibility to breast cancer. Sci Rep. 2020 Dec 4;10(1):21276. PubMed Central PMCID: PMC7718875.

3. Environmental carcinogenesis

It is well established that risk for cancer and cancer prognosis can be altered by different modifiable lifestyle factors, even in carriers of high-penetrant mutations. Therefore, understanding how environmental factors modify risks will allow us to provide more individualized risk estimates, improve risk stratification, and more effectively tailor primary prevention interventions and treatment. Additionally, the benefit in terms of absolute risk reduction that could be achieved by changing modifiable risk factors is expected to be larger for those who are at the highest end of the risk spectrum from non-modifiable factors, including family history and inherited genetic susceptibility. This is illustrated in a study conducted in a large prospective family based study (ProF-SC) of over 16,000 women that found that women at higher familial risk of breast cancer (BC) have a much larger difference in absolute risk depending on their body mass index than women at lower familial risk. Additionally in ProF-SC, I lead a study that examined whether the association of alcohol consumption, cigarette smoking and BC risk is modified by underlying absolute risk of BC. I found moderate alcohol intake to be associated with increased BC risk, particularly for those with estrogen receptor-positive tumors, but only for women at lower predicted absolute risk. On the other hand, for women at high predicted absolute risk who also consumed alcohol, being a current smoker was associated with increased BC risk. These findings can have implications in terms of absolute risk reduction, as alcohol and smoking are modifiable risk factors and present risk reduction opportunities for women across the spectrum of familial risk.

a. **Zeinomar N**, Qin B, Amin S, Lin Y, Xu B, Chanumolu D, Omene CO, Pawlish KS, Demissie K, Ambrosone CB, Hong CC, Bandera EV. Association of Cigarette Smoking and Alcohol Consumption With Subsequent Mortality Among Black Breast Cancer Survivors in New Jersey. JAMA Netw Open. 2023 Jan 3;6(1):e2252371. PubMed PMID: 36692882.

b. Kehm RD, Oskar S, Tehranifar P, **Zeinomar N**, Rundle AG, Herbstman JB, Perera F, Miller RL, Terry MB. Associations of prenatal exposure to polycyclic aromatic hydrocarbons with pubertal timing and body composition in adolescent girls: Implications for breast cancer risk. Environ Res. 2021 May;196:110369. PubMed Central PMCID: PMC8552520.

c. **Zeinomar N**, Oskar S, Kehm RD, Sahebzeda S, Terry MB. Environmental exposures and breast cancer risk in the context of underlying susceptibility: A systematic review of the epidemiological literature. Environ Res. 2020 Aug;187:109346. PubMed Central PMCID: PMC7314105.

d. **Zeinomar N**, Knight JA, Genkinger JM, Phillips KA, Daly MB, Milne RL, Dite GS, Kehm RD, Liao Y, Southey MC, Chung WK, Giles GG, McLachlan SA, Friedlander ML, Weideman PC, Glendon G, Nesci S, Andrulis IL, Buys SS, John EM, MacInnis RJ, Hopper JL, Terry MB. Alcohol consumption, cigarette smoking, and familial breast cancer risk: findings from the Prospective Family Study Cohort (ProF-SC). Breast Cancer Res. 2019 Nov 28;21(1):128. PubMed Central PMCID: PMC6883541.

4. Community translation and impact

While the past several decades were filled with remarkable scientific discoveries and development of powerful new technologies including genomic medicine, the translation of these findings into effective clinical and public health interventions to treat and prevent disease has been a major challenge. It is estimated that cancer outcomes can be improved by 30% with full application of what is currently known. One method for translation is knowledge translation and dissemination of research findings to the

appropriate audiences using the appropriate mediums. My doctoral work used a community-based educational intervention to move results of epidemiological and genomic studies into practice. I worked with local community partners to synthesize the existing breast cancer etiology and risk factor literature, identify key messages for primary and secondary prevention of breast cancer, and tailor those messages for two target populations: college students and community-based participants. I developed assessment questionnaires and took the lead on dissemination of the education, assessment, and analysis of over 500 questionnaires. We found low baseline breast cancer knowledge in both groups, particularly about basic cancer biology and risk factors, and an improvement in knowledge post-education. I continued research in knowledge translation while a post-doctoral fellow at Columbia through the Columbia Center for Children's Environmental Health (CCCEH), which for nearly two decades has worked to translate research findings and educate the community on a variety of environmental exposures, particularly during vulnerable windows of susceptibility. Through a series of focus groups with adolescents in the CCCEH cohort assessing health priorities and modes of communication, we found that environmental health and primary prevention of chronic disease was not a priority and that adolescents preferred interactive modes of communication, including social media and videos, to deliver health messages. We also developed a cancer risk-reduction educational tool tailored for adolescents that focused on five modifiable cancer risk factors, which we administered to high school and college students in New York City. These studies underscore the importance of understanding the target audience when developing targeted public health prevention campaigns.

a. Grant-Alfieri A, Burke K, **Zeinomar N**, Delgado ML, Terry MB. Cancer Education Interventions in Adolescents: A Systematic Review of Scope and Content. Health Educ Behav. 2022 Dec;49(6):993-1003. PubMed PMID: 35898117.
b. **Zeinomar N**, Grant-Alfieri A, Burke KR, de Hoz M, Tehranifar P, Walker DAH, Morton T, Shepard P, Herbstman JB, Miller RL, Perera F, Terry MB. Cancer Risk Reduction Through Education of Adolescents: Development of a Tailored Cancer Risk-Reduction Educational Tool. J Cancer Educ. 2022 Aug;37(4):1220-1227. PubMed PMID: 33523407.
c. **Zeinomar N**, Moslehi R. The effectiveness of a community-based breast cancer education intervention in the New York State Capital Region. J Cancer Educ. 2013 Sep;28(3):466-73. PubMed Central PMCID: PMC3776602.
d. **Zeinomar N**, Burke K, Cole A, Diaz D, Evans D, Steele T, Miller RL, Sahay D, Tehranifar P, Perera F, Terry MB. Understanding how to communicate health information to adolescents in Northern Manhattan and the South Bronx.. National Institute of Environmental Health Sciences (NIEHS) The Environmental Health Science FEST; 2016 December 06; Durham, NC, USA.

Complete List of Published Work in MyBibliography:

https://www.ncbi.nlm.nih.gov/myncbi/nur.zeinomar.1/bibliography/public/