BIOGRAPHICAL SKETCH

NAME: Fernandez-Becker, Nielsen Quimaira

ORCID ID: 0000-0002-9562-868X

POSITION TITLE: Clinical Professor

ORGANIZATION NAME: Stanford University

ORGANIZATION LOCATION: Stanford California USA

PROFESSIONAL PREPARATION:

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
City University of New York, Queens, NY	B.S.	1989-1994	Biology
Albert Einstein College of Medicine, Bronx, NY	M.S.	1997	Biological Sciences
Albert Einstein College of Medicine, Bronx, NY	M.D.	1994-2002	Medicine
Albert Einstein College of Medicine, Bronx, NY	Ph.D.	1994-2002	Molecular Genetics

APPOINTMENTS AND POSITIONS:

START/END DATE	APPOINTMENT	ORGANIZATION	LOCATION
2024-present	Clinical Professor of Medicine	Stanford University	Stanford, CA
2012- present	Director, Celiac Disease Program	Stanford University	Stanford, CA
2019-2024	Clinical Associate Professor of Medicine	Stanford University	Stanford, CA
2012- 2019	Clinical Assistant Professor of Medicine	Stanford University	Stanford, CA
2009-2012	Instructor Division of Gastroenterology and Hepatology	Stanford University	Stanford, CA
2005-2009	Clinical and Research Fellow in Gastroenterology	Beth Israel Deaconess Medical Center/Harvard Medical School	Boston, MA
2002-2005	Clinical Fellow in Medicine	Massachusetts General Hospital	Boston, MA

PRODUCTS:

Overview

I am a Clinical Professor of Medicine in the Division of Gastroenterology and the Director of the Celiac Disease Program at Stanford University. My focus is on diagnosing and managing celiac disease (CeD) and gluten-related disorders, where I aim to provide high-quality patient care while advancing our understanding of these complex diseases. I oversee clinical and research initiatives, maintain a biorepository of clinical specimens, and collaborate with leading scientists at Stanford, including Drs. Chaitan Khosla, David Relman, Calvin Kuo, and Mark Davis. As a testament to the success of our collaborative team, we developed organoid cultures from endoscopic biopsies of celiac patients and controls that recapitulate the disease in vitro (Nature, 2024). Recently, we have focused on elucidating the role of dendritic cells in presenting gluten to the immune system via transglutaminase 2, shedding light on how gluten triggers the autoimmune response in celiac disease (Yang et al., 2025).

I have collaborated closely with Dr. Vanessa El Kamari to investigate the role of microbiota in celiac disease pathogenesis. Dr. El Kamari has prospectively analyzed the microbiota in the small intestine of CeD patients at diagnosis and at various time points following treatment with a gluten-free diet. Together, we have established a workflow for identifying appropriate candidates for this research. I have built a strong referral base for new patients while maintaining a large cohort of established patients. My clinical expertise, combined with a solid foundation in basic science, enables me to analyze clinical data and correlate it with experimental findings, effectively bridging the gap between clinical practice and research.

In summary, I am well-positioned to collaborate with Dr. El Kamari in studying the role of microbiota in celiac disease pathogenesis, leveraging my background in clinical gastroenterology, a strong network of interdisciplinary collaborations, and a commitment to advancing the understanding and treatment of celiac disease.

Citations:

Santos AJM, Van Unen V, Lin Z, Ha Nhi, Batish A, Chan JE, Cedano J, Chirieleison SM, Mu Q, Guh-Siesel A, Tomaske M, Zhang ET, Choic SS, Asbjørn C, Baghdasaryan , A, Yost, KE, Karlsson K, Ha A, Li J, Dai H, Sellers ZM, Chang HY, Dunn JCY, Zhang BM, Mellins ED, Sollid LM, **Fernandez-Becker NQ,** Davis MM, Kuo CJ. A human autoimmune organoid model reveals IL-7 function in celiac disease. 2024 Nature **632**, 401–410 (2024) PMID: **39048815** PMC11747932

Yang FC, Besser HA, Chun HR, Albertelli M, **Fernandez-Becker NQ**, Jabri B, Khosla C A dendritic cell population responsible for transglutaminase 2-mediated gluten antigen presentation in celiac disease. bioRxiv [Preprint]. 2025 Jun 5:2025.06.02.657527. doi: 10.1101/2025.06.02.657527. PMID: **40502135** PMC12157571

Honors and Awards:

2018	Endowed Fellow, Sean N Parker Center for Allergy and Asthma Research, Stanford University
2014-2016	Councilor on the Neurogastroenterology and Motility Section of the American Gastroenterological Association Institute Council
2014	Hispanic Center of Excellence Fellowship Grant, Stanford University
2002	Dean's List, Albert Einstein College of Medicine
2000-2002	Samuel and May Rudin Scholarship, Albert Einstein College of Medicine
1995-2000	NIH NIGMS MARC Predoctoral Fellowship F31-GM17568, Albert Einstein College of Medicine
1994	William Stratford Prize for Academic Excellence, City University of New York
1994	Edmund Baerman Award for Excellence in Biology, City University of New York
1994	Jonas Salk Award, Honorable Mention, City University of New York
1992-1994	NIH MARC/MBRS Undergraduate Program

Contributions to Science

1. Early studies focused on understanding molecular mechanisms that control embryonic dorsal-ventral polarity in Drosophila. We discovered that Cactus protein undergoes regulated degradation in gradient fashion which results in graded activation of the NF-KB analogue protein dorsal protein that governs the Drosophila dorso-ventral body plan. We reported on novel separable redundant regulatory determinants in the Cactus protein.

    a. Bergmann A, Stein D, Geisler R, Hagenmaier S., Schmid B, **Fernandez N**, Schnell B, Nusslein-Volhard C. A gradient of cytoplasmic Cactus degradation establishes the nuclear localization gradient of the dorsal morphogen in Drosophila. *Mechanisms of Development*. 1996; 60(1)109-23. PMID: **9025065**

    b. **Fernandez NQ,** Grosshans J, Goltz JS, Stein D. Separable and redundant regulatory determinants in Cactus mediate its dorsal group dependent degradation. *Development*. 2001; 128(15) 2963-74. PMID: **11532919**

    c. **Fernandez NQ**. Control of *drosophila* Embryonic Dorsal-Ventral Polarity by the Cactus Protein. Thesis Dissertation, 2002.

2. Later studies focused on understanding clinical predictors of response to infliximab in patients with Crohn's disease. Our findings indicated that infliximab infusion reactions led to interruption of therapy and that concomitant use of an immunomodulator had a modest effect at preventing infusion reactions.

    a. Moss AC, **Fernandez-Becker N**, Jo Kim KJ, Cury D, Cheifetz AS. The impact of infliximab infusion reactions on long-term outcomes in patients with Crohn's disease. *Alimentary Pharmacology Therapeutics*. 2008; 28(2):221-7. PMID: **18485127**

    b. Moss AC, Kim KJ, **Fernandez-Becker N,** Cury D, Cheifetz AS. Impact of concomitant Immunomodulator use on long-term outcomes in patients receiving scheduled maintenance infliximab. *Digestive Disease Science*. 2010; 55(5): 1413-20. PMID: **19533357**

    c. **Fernandez-Becker NQ**, Moss AC. *In silico* analysis of T-bet activity in peripheral blood mononuclear cells in patients with inflammatory bowel disease (IBD). *In silico Biology*. 2009; 9 (5-6):355-63. PMID: **22430437**

3. Current studies focus on molecular mechanisms underlying the pathogenesis of celiac disease. Celiac disease results when dietary gluten and MHC class II alleles, HLA-D02 or HLA-DQ8, trigger CD4+ T cell-dependent mucosal injury to the small bowel. Epithelial and immune cell interactions are critical for development of celiac disease. Our work supports a role for gut-homing gluten responsive CD8+ and γδ T cells following gluten exposure in celiac disease pathogenesis. To further understand celiac pathogenesis, our collaborative team (Chaitan Khosla, Calvin Kuo, Mark Davis, Elizabeth Mellins and myself) have taken multiple approaches to understand celiac disease pathogenesis. In an effort led primarily Dr. Kuo, we created a 3-D organoid culture model derived from celiac disease patient biopsies, which faithfully recapitulates the disease *ex vivo* to define crosstalk between gluten-specific T cells and epithelium. Our work has resulted in the elucidation of a role for $CD8^+$ T cells in intestinal inflammation in celiac disease (CeD) (*PNAS*; 110(32):13073-8 & *Science* 376(6590) as well as a novel platform for the identification of gluten-derived peptides in the urine of CeD patients (*Nat Commun*. 2022 16;13(1)) and identified a novel role for IL-7 in celiac disease pathogenesis (*Nature.* 2024 632, 401–410). Most recently through collaboration with Dr. Khosla, our group identified a specific dendritic cell population that mediates gluten antigen presentation via transglutaminase 2, further enhancing our understanding of gluten's role in triggering the autoimmune response and highlighting potential therapeutic targets for managing celiac disease (bioRxiv [Preprint]. 2025 Jun 5:2025.06.02.657527).

    a. Palanski B, Weng N, Zhang L, Hilmer A, Fall L, Swaminathan K, Jabri B, Sousa C, **Fernandez-Becker NQ,** Khosla C, Elias J. Identification of Chemically Defined Gluten Peptides in the Urine of Patients with Celiac Disease. Nat Commun. 2022 Feb 16;13(1):888. doi: 10.1038/s41467-022-28353-1. PMID: **35173144** PMC8850430

    b. Li J, Zaslavsky M, Su Y, Guo J ,Sikora MJ, van Unen V, Christophersen A, Chiou S, Chen L, Ji X, McSween AM, Palanski BA, Mallajosyula VVA, Bracey NA Dhondalay GKR, Bhamidipati K, Pai J, Kipp LB, Dunn JE, Hauser SL, Oksenberg JR, Satpathy AT, Robinson WH, Steinmetz LM, Khosla C, Utz PJ, Sollid LM,Chien YH, Heath JR, **Fernandez-Becker NQ**, Nadeau KC, Saligrama N, Davis MM. Human KIR^+ $CD8^+$ T cells target pathogenic T cells in Celiac disease and are active in other autoimmune disorders and COVID-19 Science**.** 2022 Apr 15;376(6590):PMID: **35258337** PMC8995031

    c. Santos AJM, Van Unen V, Lin Z, Ha Nhi, Batish A, Chan JE, Cedano J, Chirieleison SM, Mu Q, Guh-Siesel A, Tomaske M, Zhang ET, Choic SS, Asbjørn C, Baghdasaryan , A, Yost, KE, Karlsson K, Ha A, Li J, Dai H, Sellers ZM, Chang HY, Dunn JCY, Zhang BM, Mellins ED, Sollid LM, **Fernandez-Becker NQ,** Davis MM, Kuo CJ. A human autoimmune organoid model reveals IL-7 function in celiac disease. 2024 Nature **632**, 401–410 (2024) PMID: **39048815** PMC11747932

d. Yang FC, Besser HA, Chun HR, Albertelli M, **Fernandez-Becker NQ**, Jabri B, Khosla C A dendritic cell population responsible for transglutaminase 2-mediated gluten antigen presentation in celiac disease. bioRxiv [Preprint]. 2025 Jun 5:2025.06.02.657527. doi: 10.1101/2025.06.02.657527. PMID: **40502135** PMC12157571

4. Food allergies have increased in prevalence in last decade as have eosinophilic disorders of GI tract namely eosinophilic esophagitis (EoE). The goal of our research is to understand molecular mechanisms for food allergy. Using biopsies GI tract of peanut allergic undergoing oral-immunotherapy (OIT) we found that peanut allergic patients have gastrointestinal eosinophilia at baseline. Subsequent studies will focus on development of tissue eosinophilia longitudinally in our OIT cohort and role of B and T cells in development of food allergies.

a. Wright BL, **Fernandez-Becker NQ,** Kambham N, Purington N, Cao S, Tupa D, Zhang, W, Sindher SB, Rank MA, Kita H, Katzka DA, Shim KP, Bunning BJ, Doyle AD, Jacobsen EA, Tsai M, Boyd SD, Manohar M, Chinthrajah RS. Gastrointestinal Eosinophil Responses in a Longitudinal, Randomized Trial of Peanut Oral Immuno Therapy. Clin Gastroenterol Hepatol 2021 Jun;19(6):1151-1159 PMID: **32434067** PMC8445108

b. Serling-Boyd, N, Chung M, Li SHufeng, Becker L, **Fernandez-Becker N,** Clarke J, Fiorentino Chung L. Gastric Antral Vascular Ectasia in Systemic Sclerosis: Association with Anti-RNA Polymerase III and Negative Anti-Nuclear Antibodies. Semin Arthritis Rheum 2020 Jul 6;50(5):938-942. PMID: **32906028** PMC7584748

c. Zhang W, Dhondalay GK, Liu TA, Kaushik A, Hoh R, Kwok S, Kambham N, **Fernandez-Becker NQ**, Andorf S, Deesai M, Galli SJ, Boyd SD, Nadeau KC, Manohar M, DeKruyff RH, Chinthrajah RSGastrointestinal γδ T cells reveal differentially expressed transcripts and enriched pathways during peanut oral immunotherapy. 2022 May;77(5):1606-1610. PMID: **35143054** PMC9581524