BIOGRAPHICAL SKETCH

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NAME: Nina Felice Schor

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

POSITION TITLE: Professor and Chair, Department of Pediatrics

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
Yale University, New Haven, CT	B.S.	05/75	Molec Biophys Biochem
Rockefeller University, New York, NY	Ph.D.	06/80	Medical Biochem
Cornell University Medical School, New York, NY	M.D.	05/81	Medicine
Children's Hospital of Boston, Boston, MA	Residency	06/83	Pediatrics
Harvard-Longwood Program, Boston, MA	Residency	06/86	Child Neurology

NOTE: The Biographical Sketch may not exceed five pages. Follow the formats and instructions below.

A. Personal Statement

As a developmental neuroscientist and child neurologist, I have seen first-hand the "grey area" between developmental neuroscience and behavioral psychology. In the context of a physician-scientist career that has primarily been aimed at the understanding of the biology of tumors of the neural crest and the mentoring of the next generation of physician-scientists in developmental neuroscience, I have established and directed a laboratory that has developed much of the signaling and cell fate road map associated with the p75 neurotrophin receptor (p75NTR). I hypothesized that p75NTR, a developmentally regulated protein that modulates the redox state of the cell and its mitochondria, plays a role in the determination of behavioral phenotype. Recently, my colleagues and I have developed a murine model of p75NTR knockout specifically in the Purkinje cells of the cerebellum, in hopes of discerning the behavioral importance of cerebellar p75NTR, and have characterized the decidedly behavioral phenotype of these mice. I am very excited to lead my group, including our junior faculty and trainees, in the elucidation of the roles and mechanisms of p75NTR in developmental modulation of behavior. My expertise in signal transduction, molecular developmental biology, and neurobiology uniquely qualify me to lead this team.

B. Positions and Honors

Positions and Employment

1973	Undergraduate Fellow, Cold Spring Harbor Laboratory, NY
1974-1975	Scholar of the House, Chem. Research, Yale Univ., New Haven, CT
1975-1981	Biomedical Fellow, Rockefeller Univ., NY, NY
1981-1986	Clinical Fellow, Pediatrics & Neurology, Harvard Medical School, Boston, MA
1986-1991	Asst. Prof., Pediatrics & Neurology, Univ. of Pittsburgh, Pittsburgh, PA
1987-2006	Member, Pittsburgh Cancer Institute
1989-1991	Asst. Prof. (secondary) Pharmacology, Univ. of Pittsburgh
1989-2006	Member, Teaching Faculty, Center for Neuroscience, Univ. of Pittsburgh
1991-1997	Assoc. Prof., Pediatrics, Neurology, & Pharmacology, Univ. of Pittsburgh
1997-2006	Prof, Pediatrics, Neurology, & Pharmacology, Univ. of Pittsburgh
2000-2006	Carol Ann Craumer Professor of Pediatric Research, Children's Hospital of Pittsburgh

2000-2001	Interim Scientific Director, Children's Hospital of Pittsburgh
2000-2006	Vice Chair for Research, Department of Pediatrics, Univ. of Pittsburgh
2002-2006	Chief, Division of Child Neurology, Univ. of Pittsburgh
2002-2003	Asst. Dean, Med. Student Research, Univ. of Pittsburgh School of Medicine
2004-2006	Assoc. Dean, Med. Student Research, Univ. of Pittsburgh School of Medicine
2006-	Professor and Chair, Department of Pediatrics, Univ. of Rochester
2006-	Pediatrician-in-Chief, Golisano Children's Hospital at Strong, Rochester, NY
2006-	Professor (Secondary Appointment), Department of Neurology, University of Rochester School of Medicine and Dentistry
2007-	Professor (Secondary Appointment), Department of Neurobiology and Anatomy, University of Rochester School of Medicine and Dentistry
2007-2012	Director, Translational Biomedical Science PhD Program, University of Rochester School of Medicine and Dentistry

NIH Study Section and Working Group Experience

1992-present	Member, MDCN SBIR Study Section
2000	Ad hoc Member, Visual Sciences-A Study Section
2000	NIH Neurosciences IRG Working Group
2002	NIH SBIR Working Group
2003	Ad hoc Member, NIA T35 Study Section
2004-2011	Ad hoc Member, NINDS NST Study Section
2007	Member, ORD Multicenter Project Study Section
2011-present	Member, NINDS NST Study Section

Selected Honors

1972	Westinghouse Science Talent Search, National First Prize
1972	Tomorrow's Scientists and Engineers Award
1972	American Academy of Achievement Award
1973	Cold Spring Harbor Laboratory Undergraduate Research Participant
1980	Alfred A. Richman Award for Cardiopulmonary Res., Amer. Coll. of Chest Physicians
1985	Ruth Estrin Goldberg Memorial Award for Cancer Res., Ruth Estrin Goldberg Memorial Foundn.
1987-89	Child Neurology Fellows' Award for Excellence in Teaching
1990	Benjamin N. Cardozo High School Women in Science Award
1990	Michael E. Miller Young Investigator Award, Children's Hospital of Pittsburgh
1991	Conferral of tenure, University of Pittsburgh School of Medicine
1995	Dean's Master Educator Award, University of Pittsburgh School of Medicine
1996	Chancellor's Distinguished Teaching Award, University of Pittsburgh
2000	Carol Ann Craumer Endowed Chair of Pediatric Res., Children's Hospital of Pittsburgh
2005	American Neurological Association, Distinguished Neurology Teacher Award
2006	Academy of Master Educators, University of Pittsburgh School of Medicine
2008	William H. Eilinger Endowed Chair of Pediatrics, University of Rochester School of Medicine and Dentistry
2012	Election to Presidency of Child Neurology Society (effective 10/2013-10/2015)

C. Contribution to Science

1. Elucidation of the roles of p75NTR and TrkA in life-or-death cellular decision-making: In the context of the prior characterization of p75NTR as a "death receptor", my laboratory defined the potential of p75NTR to either protect or kill neuroblastoma cells depending on the intracellular milieu and extracellular environment. We delineated the downstream signaling events involved in determining these diametrically opposed outcomes. Similarly, in the context of the prior characterization of TrkA as a "nurturance" protein, we further discovered the "dose" effect of TrkA on life-or-death decision-making and delineated the signaling effectors of that duality, as well. These studies identified potential therapeutic targets for neural crest tumors and the need for molecular characterization of individual tumors and hosts to determine what targets are of relevance for a particular patient. They also demonstrated the role of receptors as "branch points" rather than one-way determinants of cell fate.

Selected Relevant Publications:

a. Yan C, Liang Y, Nylander KD, Wong J, Rudavsky RM, Saragovi HU, Schor NF. p75-NGF as an antiapoptotic complex: independence vs. cooperativity. Molec Pharmacol 61:710-719, 2002.
b. Yan C, Liang Y, Nylander KD, Schor NF. TrkA as a life-and-death receptor: receptor dose as a mediator of function. Cancer Research 62:4867-4875, 2002.
c. Mirnics ZK, Yan C, Portugal CF, Kim TW, Saragovi HU, Sisodia SS, Mirnics K, Schor NF. P75 neurotrophin receptor regulates expression of neural cell adhesion molecule 1. Neurobiol Disease 20:969-985, 2005.
d. Korade Z, Mi Z, Portugal C, Schor NF. Expression and p75 neurotrophin receptor dependence of cholesterol synthetic enzymes in adult mouse brain. Neurobiol of Aging 28:1522-1531, 2007.

2. Discovery of the redox and behavior modulation activities of neurotrophic signaling: My laboratory colleagues and I discovered and characterized the cytoplasmic antioxidant and mitochondrial pro-oxidant activity of p75NTR. The intracellular domain of p75NTR, liberated from the cell membrane by α- and γ-secretases, houses these activities. This may make possible the design and implementation of peptide fragments of p75NTR as redox-active drugs. With Dr. Marc Halterman, originally my T32 and K99 “mentee”, and now my R01-funded faculty colleague, we discovered BDNF-dependent redox-active signaling pathways, as well. Recently, we discovered the role of cerebellar p75NTR in the development of behavioral phenotype.

Selected Relevant Publications:

a. Mi Z, Rogers D, Mirnics ZK, Schor NF. p75NTR-dependent modulation of cellular handling of reactive oxygen species. J Neurochem 110:296-306, 2009. NIHMS657968.
b. Halterman MW, Gill M, DeJesus C, Ogihara M, Schor NF, Federoff HJ. The endoplasmic reticulum stress response factor, chop-10, protects against hypoxia-induced neuronal death. J Biol Chem 285:21329-21340, 2010. PMCID: PMC2898390.
c. Ganeshan VR, Schor NF. p75 neurotrophin receptor and fenretinide-induced signaling in neuroblastoma. Cancer Chemother Pharmacol 73:271-279, 2014. PMCID: PMC3946654.
d. Lotta LT, Conrad K, Cory-Slechta D, Schor NF. Cerebellar Purkinje cell p75 neurotrophin receptor and autistic behavior. Translat Psychiatr 4:e416 and e476, 2014. PMCID: PMC4119222.

3. Design and preclinical testing of novel, innovative therapies for neuroblastoma: Neuroblastoma is the single most common solid tumor of childhood. Sixty % of children with neuroblastoma have metastatic disease at the time of initial diagnosis and succumb to their disease within 5-8 years. We have designed and developed in tissue culture and murine models strategies for overcoming this killer of childhood. These approaches take advantage of the neural characteristics of this tumor and of the unique ways in which this tumor escapes the effects of conventional chemotherapy.

Selected Relevant Publications:

a. Schor NF, Rudin CM, Hartmann AR, Thompson CB, Tyurina YY, Kagan VE. Cell line dependence of Bcl-2-induced alteration of glutathione handling. Oncogene 19:472-476, 2000.
b. Liang Y, Nylander KD, Yan C, Schor NF. Role of caspase 3-dependent Bcl-2 cleavage in potentiation of apoptosis by Bcl-2. Molec Pharmacol 61:142-149, 2002.
c. Mi Z, Hong B, Mirnics ZK, Tyurina YY, Kagan VE, Liang Y, Schor NF. Bcl-2-mediated potentiation of neocarzinostatin-induced apoptosis: requirement for caspase-3, sulfhydryl groups, and cleavable Bcl-2. Cancer Chemother Pharmacol 57:357-367, 2005.
d. Rogers DA, Schor NF. Kidins220/ARMS depletion is associated with the neural- to Schwann-like transition in a human neuroblastoma cell line model. Exp Cell Res 319:660-669, 2013. PMID: 23333500 PMCID: N/A.

4. Identification of novel prognostic biomarkers and therapeutic markers and targets for neuroblastoma: This newest of our therapeutic and mechanistic studies of neuroblastoma has already borne novel fruit. We have discovered that mitochondrial complex IV, thought to mediate p75NTR-associated enhancement of the oxidative toxicity of fenretinide (currently in clinical trials for neuroblastoma), is not in fact the relevant effector, and that CRABP-I, a modulator of fenretinide metabolism, may mediate this effect. This is important because small molecule enhancers of the therapeutic index of fenretinide are being actively sought and it is critical that

the right target for this effort be identified. In addition, we have discovered the relationships between MYCN amplification and ALK mutation, on the one hand, and expression of PRMT1, EYA1, and SIX3, on the other, and the physical association in a trimolecular complex and activation cascade of PRMT1, EYA1, and SIX3. This is important because of the known relationships between MYCN amplification and ALK mutation, respectively, and poor prognosis in neuroblastoma, and because of the druggable targets engendered in the transcriptional effects of sequential activation of EYA1 by PRMT1 and SIX3 by EYA1. We cite abstracts and manuscripts below because of the newness of these findings.

Selected Relevant Publications and Other Products:

a. Pu Y, Li X, Schor NF. Role of CRABP-I in potentiation of fenretinide oxidative toxicity in neuroblastoma cells. Ann Neurol S135-S136, 2013.
b. Li X, Wang S, Schor NF. Methylase meets phosphatase: roles of PRMT and EYA1 in neuroblastoma. AACR Proceedings, abstract #464, 2014.
c. Li X, Hansen J, Schor NF. Arginine methylation modulates the phosphatase-transcription activator EYA1 activity. Submitted for publication, 2015.
d. Pu Y, Li X, Wang S, Schor NF. Effectors of p75NTR-induced potentiation of fenretinide oxidative toxicity in neuroblastoma cells. Manuscript in preparation, 2015.

5. Characterization of neurodegenerative disorders as reflective of dysfunction in developmentally important proteins and processes: Our studies of the role of presenilin in processing of p75NTR, of developmentally-regulated changes in the transcriptome as a function of mutation or knockout of presenilin, and of the neuronal differentiation induced by Bcl-2 overexpression underscore the fact that “developmental neurobiology” does not stop at 18 years of human age! As aging and development are part of the same temporobiologic continuum, so is the study of developmental regulation of protein expression and function important for the understanding and conquest of senescence and neurodegenerative disease.

Relevant Publications:

a. Liang Y, Mirnics ZK, Yan C, Nylander KD, Schor NF. Bcl-2 mediates induction of neural differentiation. Oncogene 22:5515-5518, 2003.
b. Mirnics ZK, Mirnics K, Terrano D, Lewis DA, Sisodia SS, Schor NF. DNA microarray profiling of developing PS1-deficient mouse brain reveals complex and co-regulated expression changes. Molec Psychiatr 8:863-878, 2003.
c. Unger T, Korade Z, Lazarov O, Terrano D, Schor NF, Sisodia SS, Mirnics K. Transcriptome differences between the frontal cortex and hippocampus of wild-type and humanized presenilin-1 transgenic mice. Am J Geriatr Psych 13:1041-1051, 2005.
d. Mirnics K, Korade Z, Arion D, Lazarov O, Unger T, Macioce M, Sabatini M, Terrano D, Douglass KC, Schor NF, Sisodia SS. Presenilin-1-dependent transcriptome changes. J Neurosci 25:1571-1578, 2005.

Complete List of Published Work in MyBibliography: http://www.ncbi.nlm.nih.gov/sites/myncbi/1zuL2rl-RkyA9/bibliography/46483167/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

W. H. Eilinger Endowmnt, Golisano Chld Hosp (NF Schor, PI) 07/01/2008-present
Preclinical Pharmacology of Targeted Therapies for Neuroblastoma
This endowment provides much of the infrastructure for the University of Rochester Department of Pediatrics’ research program in preclinical pharmacology for the treatment of neuroblastoma.

T32 HD057821
National Institutes of Health (NF Schor, PI) 05/01/10-04/30/15
Pediatric Research: Bench to Bedside to Curbside
This research training grant is aimed at launching the research-intensive careers of fellows in pediatric subspecialty fellowships at the University of Rochester.

K12 HD068373 (NF Schor, PI) 01/01/2011-12/31/16

Translational Molecular Pediatric Research

This institutional mentored research grant is aimed at launching the basic research-intensive careers of physician-scientist junior faculty in the Department of Pediatrics at the University of Rochester.

Crosby's Pediatr. Cancer Res. Fund Grant (NF Schor, PI) 07/01/2012-06/30/2017

Role of Six Family Proteins in Chemoresistance of Neuroblastoma

The goal of this project is to determine the role of the Six family of transcription factors and their interactors, the EYA proteins, in resistance to chemotherapy in neuroblastoma.

Completed Research Support

Strong Children's Research Center Pilot Grant (NF Schor, PI) 09/01/2011-08/31/2012

p75NTR Deletion in Cerebellar Purkinje Cells: A Model for Autism?

This project exploits our 25 years of studies on the p75 neurotrophin receptor in hopes of developing a murine model for autism.

BIOGRAPHICAL SKETCH

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NAME: Jarvinen-Seppo, Kirsi

eRA COMMONS USER NAME (agency login): JARVINSK10

POSITION TITLE: Associate Professor of Pediatrics; Chief, Pediatric Allergy and Immunology

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
University of Helsinki, Helsinki	MD	05/1996	
University of Helsinki, Helsinki	PHD	10/2000	Allergy and Immunology
Mount Sinai School of Medicine, New York, NY	Postdoctoral Fellow	07/2001	Allergy and Immunology
Mount Sinai Hospital, New York, NY	Resident	06/2005	Pediatrics
Mount Sinai Hospital, New York, NY	Fellow	06/2008	Allergy and Immunology

A. PERSONAL STATEMENT

I have the training, expertise and dedication necessary to successfully carry out the proposed research project. I have a broad background in allergic disorders, with specific training and expertise in assessment of role of breast milk bioactive substances in relation to development of allergies in the offspring. As PI or Co-Investigator on several university and NIH-funded grants, I laid the groundwork for the proposed research by developing assays specific to research on breast milk, assessment of atopic and allergic conditions in infants and young children, and by establishing strong ties with the scientific community. In addition, I successfully administered the projects (e.g. staffing, research protections, budget), collaborated with other researchers, and produced several peer-reviewed publications from each project. As a result of these previous experiences, I am aware of the importance of research to understand the early origins of allergic disorders. The current application builds logically on my prior work.

1. Järvinen KM, Mäkinen-Kiljunen S, Suomalainen H. Cow's milk challenge through human milk evokes immune responses in infants with cow's milk allergy. J Pediatr. 1999 Oct;135(4):506-12. PubMed PMID: 10518086.
2. Järvinen KM, Beyer K, Vila L, Chatchatee P, Busse PJ, et al. B-cell epitopes as a screening instrument for persistent cow's milk allergy. J Allergy Clin Immunol. 2002 Aug;110(2):293-7. PubMed PMID: 12170271.
3. Järvinen KM, Westfall JE, Seppo MS, James AK, Tsuang AJ, et al. Role of maternal elimination diets and human milk IgA in the development of cow's milk allergy in the infants. Clin Exp Allergy. 2014 Jan;44(1):69-78. PubMed PMID: 24164317; PubMed Central PMCID: PMC4038099.
4. Järvinen KM, Suárez-Fariñas M, Savilahti E, Sampson HA, Berin MC. Immune factors in breast milk related to infant milk allergy are independent of maternal atopy. J Allergy Clin Immunol. 2014 Dec 19;PubMed PMID: 25533649.

B. POSITIONS AND HONORS

Positions and Employment

2001 - 2003 Instructor, Mount Sinai School of Medicine, New York, NY
2008 - 2011 Assistant Professor of Pediatrics, Mount Sinai School of Medicine, New York, NY
2011 - 2014 Assistant Professor of Medicine and Pediatrics, Albany Medical College, Albany, NY
2014 - Associate Professor of Pediatrics; Chief, University of Rochester School of Medicine and Dentistry, Rochester, NY

Other Experience and Professional Memberships

2000 -	Member, International Society for Research on Human Milk and Lactation
2000 -	Member, Fellow, American Academy of Allergy, Asthma and Immunology
2005 -	Member, American College of Allergy, Asthma and Immunology
2005 -	Ad hoc reviewer, Ad hoc reviewer Journal Allergy Clinical Immunology, Clinical and Experimental Allergy, Pediatrics, Pediatric Allergy and Immunology, International Archives of Allergy and Immunology
2006 -	Member, American Academy of Pediatrics
2010 -	Member, American Academy of Allergy, Asthma and Immunology, Food Allergy Interest Section
2012 - 2013	Workshop Planning Committee and Thematic Working Group Member, Dietary Guidance Development Project for Infants and Toddlers from Birth to 24 Months and Women Who are Pregnant (B-24/PW), USDA-HHS
2015 -	Technical Expert Collaborative, Dietary Guidance Development Project for Infants and Toddlers from Birth to 24 Months and Women Who are Pregnant (B-24/PW), USDA-HHS

Honors

1995	Finnish Foundation for Allergy Research, Finland, Scholarship
1997	Scholarship, Yrjö Jahnsson Foundation, Finland
1998	Scholarship, Research Foundation of Orion Corporation Scholarship, Finland
1998	Scholarship, Finnish Medical Foundation, Finland
1998	Scholarship, Ida Montin Foundation, Finland
1999	Scholarship, Finnish Medical Association Duodecim, Finland
2000	Scholarship, Thanks to Scandinavia Foundation, New York, NY
2002	Scholarship, Instrumentarium Science Foundation, Finland
2006	Jaffe Food Allergy Research Award, AAAAI
2006	Third-Year Fellow-in-Training Research Award, AAAAI
2008	Child Health Research Center Scholarship, NICHD
2008	School on Hypersensitivity and Allergic Diseases Travel Scholarship, AAAAI and CIS
2011	Mentored Physician Scientist Award, NIAID

C. Contribution to Science

1. My early publications have assessed the role of breast milk immunologic factors and their relation to development of cow's milk allergy in the breastfed infant, which stem from my translational PhD studies in the Helsinki University Central Hospital, Finland. I received further post-doctoral research training in mechanisms of food allergy and subsequently as an Instructor in the Jaffe Food Allergy Institute at the Mount Sinai School of Medicine, New York, supervised by Dr. Hugh Sampson, a world-known food allergy expert. After I completed my clinical training in Pediatrics and Fellowship in Allergy and Immunology, I was recruited as an Assistant Professor of Pediatrics to the Jaffe Food Allergy Institute. At that time, I was appointed a Research Scholar through the NIH K12-supported Children's Health Research Center (CHRC) for studies on neonatal oral tolerance, which in 2011 culminated in the K08 award by the NIAID for work related to role of breast milk in development of oral tolerance to foods. My laboratory has recently discovered that breast milk IgA and cytokine milieu relate to protection against food allergy. During the second half of my award, I was recruited to the University of Rochester School of Medicine & Dentistry to establish a clinical division and a research program in food allergy, where Dr Tim Mosmann serves as my local mentor. External mentorship is maintained at Icahn School of Medicine at Mount Sinai with Drs. Hugh Sampson and Cecilia Berin. My work so far has resulted in over 10 publications in the area of immunologically active substances in breast milk in relation of development of oral tolerance and development of allergies. This work can ultimately lead to better understanding of the early factors in development of allergies that can be used in designing prevention strategies to address the increasing frequency of food and other allergies.

a. Osterlund P, Smedberg T, Hakulinen A, Heikkilä H, Järvinen KM. Eosinophil cationic protein in human milk is associated with development of cow's milk allergy and atopic eczema in breast-fed infants. Pediatr Res. 2004 Feb;55(2):296-301. PubMed PMID: 14630983.

b. López-Expósito I, Song Y, Järvinen KM, Srivastava K, Li XM. Maternal peanut exposure during pregnancy and lactation reduces peanut allergy risk in offspring. J Allergy Clin Immunol. 2009 Nov;124(5):1039-46. PubMed PMID: 19895992; PubMed Central PMCID: PMC2801422.

c. Järvinen KM, Westfall JE, Seppo MS, James AK, Tsuang AJ, et al. Role of maternal elimination diets and human milk IgA in the development of cow's milk allergy in the infants. Clin Exp Allergy. 2014 Jan;44(1):69-78. PubMed PMID: 24164317; PubMed Central PMCID: PMC4038099.

d. Järvinen KM, Suárez-Fariñas M, Savilahti E, Sampson HA, Berin MC. Immune factors in breast milk related to infant milk allergy are independent of maternal atopy. J Allergy Clin Immunol. 2014 Dec 19;PubMed PMID: 25533649.

2. Cow's milk allergy is one of the earliest manifestations of food allergy and commonly outgrown. My postdoctoral work at the Jaffe Food Allergy Institute focused on understanding the allergenic (IgE-binding) and antigenic (IgG-binding) epitopes on cow's milk and other major food allergens. My work identified specific allergenic epitopes on cow's milk allergens that were associated with persistence of food allergy. Mutational analysis identified amino acid substitutions that could be applied to modify IgE binding to allergens. These findings are important for the understanding of mechanisms leading to persistence of food allergy as well as for designing modified allergens with decreased allergenic potential for future immunotherapeutic trials. Such approaches have been assessed in human trials for peanut allergy.
   a. Järvinen KM, Chatchatee P, Bardina L, Beyer K, Sampson HA. IgE and IgG binding epitopes on alpha-lactalbumin and beta-lactoglobulin in cow's milk allergy. Int Arch Allergy Immunol. 2001 Oct;126(2):111-8. PubMed PMID: 11729348.

   b. Järvinen KM, Beyer K, Vila L, Chatchatee P, Busse PJ, et al. B-cell epitopes as a screening instrument for persistent cow's milk allergy. J Allergy Clin Immunol. 2002 Aug;110(2):293-7. PubMed PMID: 12170271.

   c. Beyer K, Jarvinen KM, Bardina L, Mishoe M, Turjanmaa K, et al. IgE-binding peptides coupled to a commercial matrix as a diagnostic instrument for persistent cow's milk allergy. J Allergy Clin Immunol. 2005 Sep;116(3):704-5. PubMed PMID: 16159646.

   d. Järvinen KM, Beyer K, Vila L, Bardina L, Mishoe M, et al. Specificity of IgE antibodies to sequential epitopes of hen's egg ovomucoid as a marker for persistence of egg allergy. Allergy. 2007 Jul;62(7):758-65. PubMed PMID: 17573723.

3. Food–induced anaphylaxis is the leading cause of anaphylaxis in emergency rooms. A line of my work has elucidated triggers, risk factors, treatments and outcomes of food-induced anaphylaxis. Importantly, we identified a high rate of food-induced anaphylaxis requiring multiple doses of epinephrine. We also have identified vulnerable populations at higher risk for poor outcomes. This work has added to the evidence-base required to establish guidelines for management of anaphylaxis.
   a. Järvinen KM, Sicherer SH, Sampson HA, Nowak-Wegrzyn A. Use of multiple doses of epinephrine in food-induced anaphylaxis in children. J Allergy Clin Immunol. 2008 Jul;122(1):133-8. PubMed PMID: 18547626.

   b. Järvinen KM, Amalanayagam S, Shreffler WG, Noone S, Sicherer SH, et al. Epinephrine treatment is infrequent and biphasic reactions are rare in food-induced reactions during oral food challenges in children. J Allergy Clin Immunol. 2009 Dec;124(6):1267-72. PubMed PMID: 20004784; PubMed Central PMCID: PMC2798852.

   c. Huang F, Chawla K, Järvinen KM, Nowak-Węgrzyn A. Anaphylaxis in a New York City pediatric emergency department: triggers, treatments, and outcomes. J Allergy Clin Immunol. 2012 Jan;129(1):162-8.e1-3. PubMed PMID: 22018905; PubMed Central PMCID: PMC3246066.

4. Food protein-induced enterocolitis (FPIES) is a non-IgE-mediated manifestation of food allergy. Its etiology and prognosis are not fully understood and diagnostic modalities are lacking. A large cohort of these

patients at the Jaffe Food Allergy Institute has enabled a team of collaborators to elucidate these aspects of FPIES. Our work has made groundbreaking discoveries on characterization of the patients, triggers and natural history of this poorly understood disorder.

a. Järvinen KM, Caubet JC, Sickles L, Ford LS, Sampson HA, et al. Poor utility of atopy patch test in predicting tolerance development in food protein-induced enterocolitis syndrome. Ann Allergy Asthma Immunol. 2012 Sep;109(3):221-2. PubMed PMID: 22920080; PubMed Central PMCID: PMC3586209.

b. Caubet JC, Ford LS, Sickles L, Järvinen KM, Sicherer SH, et al. Clinical features and resolution of food protein-induced enterocolitis syndrome: 10-year experience. J Allergy Clin Immunol. 2014 Aug;134(2):382-9. PubMed PMID: 24880634.

5. My additional work entails studies related specifically to mechanisms of food allergy. This work has identified increases in intestinal permeability, autoreactive IgE antibodies and cutaneous antigen exposure as possible etiologic factors of food allergy..

a. Järvinen KM, Geller L, Bencharitiwong R, Sampson HA. Presence of functional, autoreactive human milk-specific IgE in infants with cow's milk allergy. Clin Exp Allergy. 2012 Feb;42(2):238-47. PubMed PMID: 22092935; PubMed Central PMCID: PMC3780604.

b. Järvinen KM, Konstantinou GN, Pilapil M, Arrieta MC, Noone S, et al. Intestinal permeability in children with food allergy on specific elimination diets. Pediatr Allergy Immunol. 2013 Sep;24(6):589-95. PubMed PMID: 23909601; PubMed Central PMCID: PMC3774110.

c. Tordesillas L, Goswami R, Benedé S, Grishina G, Dunkin D, et al. Skin exposure promotes a Th2-dependent sensitization to peanut allergens. J Clin Invest. 2014 Nov;124(11):4965-75. PubMed PMID: 25295541.

D. RESEARCH SUPPORT

Ongoing Research Support

K08 AI091655 Jarvinen-Seppo (PI) 09/30/2011 – 08/31/2016
NIAID/NIH
Role of breast milk in development of neonatal oral tolerance to foods
The goal of this project is to address the effect of feeding peanut during pregnancy and lactation on the offspring's predisposition toward allergic sensitization using an animal model of food allergy and to determine the potential mechanism of oral tolerance development provided via maternal milk.