OMB No. 0925-0001 and 0925-0002 (Rev. 09/17 Approved Through 03/31/2020)

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:

eRA COMMONS USER NAME (credential, e.g., agency login): ILONA\_JASPERS

POSITION TITLE: 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 DateMM/YYYY | FIELD OF STUDY |
| --- | --- | --- | --- |
| Seton Hall University | BS | 05/1992 | Biology |
| New York University | MS | 05/1994 | Toxicology |
| New York University | PhD | 08/1997 | Toxicology |
| University of North Carolina at Chapel Hill | Postdoc | 06/1999 | Lung Biology & Environmental Health |
|  |  |  |  |

1. **Personal Statement**My research focuses on the adverse effects of e-cigarettes, especially how it pertains to adolescent populations. Together with my faculty, appointment in the Department of Pediatrics makes me well suited to support the studies described in this proposal. As outlined in more detail in the application, this project aims to provide evidence-based health communication approaches. In my capacity as an investigator whose research focuses on the toxicity and adverse health effects induced by vaping e-cigarettes, I will be well positioned to help integrate the health effects data with effective communication about e-cigarettes. Through NIEHS, NHLBI, EPA, and FDA/NIH funding, my lab examines how inhaled toxicants modify respiratory host defense responses using a number of translational research models. We are particularly interested in how inhaled toxicants, such as e-cigarettes or cigarette smoke modify respiratory innate immune responses resulting in enhanced susceptibility to microbial infections using human *in vivo* and *in vitro* experimental models. Because of my primary faculty appointment in the Department of Pediatrics here at UNC, I am especially interested in how environmental exposure modify respiratory immune responses in susceptible populations, like children and adolescents. I have broad experience in developing non-invasive techniques to obtain human clinical samples and immunophenotyping mucosal immune cells obtained from human volunteers, which have been incorporated into clinical studies examining effects of inhaled toxicants on respiratory health. I have extensively participated in cross-disciplinary translational research teams, providing expertise in toxicology and analyses of human clinical samples. During the past 5 years, I have met and communicated with Dr. Noar and received valuable input for my own studies. Hence, I am familiar with Dr. Noar’s investigative team and have been impressed by the kind of research conducted by this group of investigators. I look forward to expanding my existing collaborations with Dr. Noar in this application and apply my expertise on adverse health effects caused by inhalation of e-cigarette vapor in susceptible populations, such as children and adolescents.
2. Reidel B, Radicioni G, Clapp PW, Ford AA, Abdelwahab S, Rebuli ME, Haridass P, Alexis NE, Jaspers I, Kesimer M. E-Cigarette Use Causes a Unique Innate Immune Response in the Lung, Involving Increased Neutrophilic Activation and Altered Mucin Secretion. Am J Respir Crit Care Med. 2018 Feb 15;197(4):492-501. doi: 10.1164/rccm.201708-1590OC. PubMed PMID: 29053025; PubMed Central PMCID: PMC5821909.
3. Clapp PW, Jaspers I. Electronic Cigarettes: Their Constituents and Potential Links to Asthma. Curr Allergy Asthma Rep. 2017 Oct 5;17(11):79. doi: 10.1007/s11882-017-0747-5. Review. PubMed PMID: 28983782; PubMed Central PMCID: PMC5995565.
4. Clapp PW, Pawlak EA, Lackey JT, Keating JE, Reeber SL, Glish GL, Jaspers I. Flavored e-cigarette liquids and cinnamaldehyde impair respiratory innate immune cell function. Am J Physiol Lung Cell Mol Physiol. 2017 Aug 1;313(2):L278-L292. doi: 10.1152/ajplung.00452.2016. Epub 2017 May 11. PubMed PMID: 28495856; PubMed Central PMCID: PMC5582929.

**B. Positions and Honors**

Positions and Employment

1992-1994 N.I.E.H.S. Trainee in Inhalation Toxicology, New York University, Tuxedo, NY

1994-1997 Graduate Research Assistant, New York University, Tuxedo, NY

1994-1996 Assistant Instructor in Human Biology, William Paterson College, Wayne, NJ

1997-1999 Post-Doctoral Research Fellow, Center for Environmental Medicine and Lung Biology, UNC-CH

1999-2000 Research Associate, Center for Environmental Medicine and Lung Biology, UNC-CH

2000-2007 Assistant Professor, Department of Pediatrics, UNC-CH School of Medicine

2002-present Assistant/Associate/Full Professor, Curriculum in Toxicology, UNC-CH

2003-present Adjunct Assistant/Associate/Full Professor, Environmental Sciences and Engineering, UNC-CH

2005-present Deputy Director, Center for Environmental Medicine, Asthma and Lung Biology, UNC-CH

2007-present Associate/Full Professor, Department of Pediatrics, UNC-CH School of Medicine

2009-present Senior Investigator of Respiratory Biology, The Hamner Institutes of Health Sciences, RTP

2011-present Associate/Full Professor, Dept. of Microbiology & Immunology, UNC-CH School of Medicine

2011-present Director, Curriculum in Toxicology, UNC-CH

Other Experience and Professional Memberships

Member of the Society of Toxicology, and American Thoracic Society

2004-present “Inhalation Toxicology” Editorial Board

2009-present “American Journal of Respiratory Cell and Molecular Biology” Editorial Board

2011-present “American Journal of Physiology-Lung Cell and Molecular Biology” Editorial Board

2000-2002 Secretary/Treasurer of the Inhalation Specialty Section of the Society of Toxicology

2006, 2007 Ad Hoc Reviewer, LIRR Study Section, NIH

2006 Reviewer; Health Effects Institute; RFA 05-1A “Studies to Compare Characteristics of PM Associated with Health Effects”

2007 Reviewer, Special Emphasis Panel “Lung Cancer and Inflammation”, NCI/NIH

2008, 2009 Reviewer, Special Emphasis Panel “Systemic Injury of Environmental Exposure”, NIH

2009 Reviewer, Special Emphasis Panel, RC1 Grant Applications Review, “Respiratory Sciences”

2009 Reviewer, Special Emphasis Panel, R15 Grant Application review, ZRG1 CVRS-F

2010 Reviewer, Special Emphasis Panel, U19 Nanotoxicology Grant Applications, NIEHS/NIH, ZES1 SET-V 03

2014, 2015 Reviewer, Special Emphasis Panel, NIEHS R25/T32, K99/R00 Grant Review

2010-2014 Member of LIRR Study Section, NIH

2009-present External Advisory Committee; Swiss National Science Foundation Project; Project leader: Dr Marianne Geiser

2011-2013 External Scientific Advisory Board, Deepwater Horizon Disaster Research Consortia: Health Impacts and Community Resiliency (NIEHS/U19), PI: Maureen Lichtveld, MD

2010-present Reviewer for Flight Attendant Medical Research Institute

2015-present Gulf Long-term Follow-up Study (GuLF), NIEHS, Scientific Advisory Board

**C. Contributions to Science**

 **Effects of E-cigarettes on Respiratory Immune Health**

Comparing the toxicity of new and emerging tobacco products (NETP), such as e-cigarettes (e-cigs) and conventional cigarettes often focuses on toxicants known to be present in cigarette smoke (CS) (i.e. acrolein, formaldehyde, nitrosamines, etc.) and clinical endpoints associated with smoking, such as cancer, COPD, etc.. However, this approach disregards potential toxicity of components unique to NETPs, such as many different flavoring chemicals contained in e-cigs, which likely induce respiratory effects not usually observed in smokers. Our studies demonstrated that e-cig users have suppressed immune gene expression in their nasal mucosa and that specific flavoring chemicals have suppressive effects on innate immune cells. These studies are beginning to provide much needed data on the toxicity and adverse health effects induced by vaping-flavored e-cigs.

* 1. Clapp PW, Pawlak EA, Lackey JT, Keating JE, Reeber SL, Glish GL, Jaspers I. Flavored E-cigarette Liquids and Cinnamaldehyde Impair Respiratory Innate Immune Cell Function. Am J Physiol Lung Cell Mol Physiol. 2017 May 11:ajplung.00452.2016. doi: 10.1152/ajplung.00452.2016. [Epub ahead of print] PubMed PMID: 28495856.
	2. Carson JL, Zhou L, Brighton L, Mills KH, Zhou H, Jaspers I, Hazucha M. Temporal structure/function variation in cultured differentiated human nasal epithelium associated with acute single exposure to tobacco smoke or E-cigarette vapor. Inhal Toxicol. 2017 Feb;29(3):137-144. doi: 10.1080/08958378.2017.1318985. PubMed PMID: 28470140.
	3. Rebuli ME, Speen AM, Clapp PW, Jaspers I. Novel applications for a noninvasive sampling method of the nasal mucosa. Am J Physiol Lung Cell Mol Physiol. 2017 Feb1;312(2):L288-L296. doi: 10.1152/ajplung.00476.2016. Epub 2016 Dec 23. PubMed PMID: 28011618; PubMed Central PMCID: PMC5336583.
	4. Martin EM, Clapp PW, Rebuli ME, Pawlak EA, Glista-Baker E, Benowitz NL, Fry RC, Jaspers I. E-cigarette use results in suppression of immune and inflammatory-response genes in nasal epithelial cells similar to cigarette smoke. Am J Physiol Lung Cell Mol Physiol. 2016 Jul 1;311(1):L135-44. doi: 10.1152/ajplung.00170.2016. Epub 2016 Jun 10. PubMed PMID: 27288488; PubMed Central PMCID: PMC4967187.

**Effects of Smoking on Respiratory Innate Immune Responses**

Over the past 7 years, a large part of my effort has been dedicated to identifying mechanisms through which smoking alters respiratory immune defense responses. Using linked human in vitro and in vivo models we have demonstrated that smoking induces epigenetic changes in genes directly related to antiviral host defense responses. In the context of these studies, we have developed a human in vivo model of influenza virus infections using the live-attenuated influenza virus (LAIV) vaccine. Using this model we have uncovered important roles for NK cells and gd T cells in the nasal mucosa, whose level and function are altered in smoking. These studies have demonstrated that smoking-induced epigenetic changes at the level of the epithelium modify antiviral immune responses and identified novel roles for NK cells and gd T cells in nasal mucosal immune responses to influenza virus in humans *in vivo*. I was the PI or Co-PI on all of these studies and have mentored several trainees affiliated with these publications.

1. Rager JE, Bauer RN, Müller LL, Smeester L, Carson JL, Brighton LE, Fry RC, Jaspers I. DNA methylation in nasal epithelial cells from smokers: identification of ULBP3-related effects. Am J Physiol Lung Cell Mol Physiol. 2013 Sep 15;305(6):L432-8. PMCID: PMC3763036.
2. Horvath KM, Brighton LE, Herbst M, Noah TL, Jaspers I. Live attenuated influenza virus (LAIV) induces different mucosal T cell function in nonsmokers and smokers. Clin Immunol. 2012 Mar;142(3):232-6. PMCID: PMC3288450.
3. Horvath KM, Herbst M, Zhou H, Zhang H, Noah TL, Jaspers I. Nasal lavage natural killer cell function is suppressed in smokers after live attenuated influenza virus. Respir Res. 2011 Aug 4;12:102. PMCID: PMC3163542.
4. Noah TL, Zhou H, Monaco J, Horvath K, Herbst M, Jaspers I. Tobacco smoke exposure and altered nasal responses to live attenuated influenza virus. Environ Health Perspect. 2011 Jan;119(1):78-83. PMCID: PMC3018504.

**Effects of ambient air pollutants on antiviral host defense responses**

For the past 10 years, a major research effort in my lab has been dedicated to understanding how exposure to air pollutants, such as ozone or diesel exhaust affects antiviral host defense responses. Using linked human in vitro and in vivo as well as mouse in vivo experimental models my lab has demonstrated that exposure to air pollutants increases susceptibility to influenza virus infection and that these effects are associated with an increased ability of the virus to enter epithelial cells and suppression of NK cell function. In addition, we have shown that in the context of allergy, exposure to air pollutants prior to infection with influenza virus significantly enhances markers of allergic inflammation in mice and humans in vivo. Observations made in these studies proved a novel paradigm, suggesting that ambient air pollutant exposure enhance the susceptibility to common respiratory infections, which may synergize to exacerbate allergic inflammation in asthmatics. I was the PI or Co-PI on all of these studies and have mentored several trainees affiliated with these publications.

1. Müller L, Chehrazi CV, Henderson MW, Noah TL, Jaspers I. Diesel exhaust particles modify natural killer cell function and cytokine release. Part Fibre Toxicol. 2013 Apr 24;10:16. PMCID: PMC3637383.
2. Kesic MJ, Meyer M, Bauer R, Jaspers I. Exposure to ozone modulates human airway protease/antiprotease balance contributing to increased influenza A infection. PLoS One. 2012;7(4):e35108. PMCID: PMC3322171.
3. Noah TL, Zhou H, Zhang H, Horvath K, Robinette C, Kesic M, Meyer M, Diaz-Sanchez D, Jaspers I. Diesel exhaust exposure and nasal response to attenuated influenza in normal and allergic volunteers. Am J Respir Crit Care Med. 2012 Jan 15;185(2):179-85. PMCID: PMC3297091.
4. Jaspers I, Sheridan PA, Zhang W, Brighton LE, Chason KD, Hua X, Tilley SL. Exacerbation of allergic inflammation in mice exposed to diesel exhaust particles prior to viral infection. Part Fibre Toxicol. 2009 Aug 14;6:22. PMCID: PMC2739151.

**Methods to examine the adverse health effects of air pollution mixtures**

In collaboration with atmospheric chemists from the Gillings School of Global Public Health at UNC-CH, I have investigated the toxicity of ambient air pollution mixtures for the past 12 years. Using “smog chambers”, we have been able to compare the toxicity of primary pollutants, such as ozone, to complex urban air pollution mixtures containing ozone and many other photochemically-derived products. These studies have developed and used innovative exposure systems to understand the toxicity derived from photochemically transformed air pollution mixtures. Observations made in these studies have yielded new concepts related to the differences in toxicity of primary and photochemically altered air pollution mixtures. I was PI and Co-I on all of these studies, have mentored, and co-mentored several trainees affiliated with these publications.

1. Gutierrez ER, Kamens RM, Tolocka M, Sexton K, Jaspers I. A comparison of three dispersion media on the physicochemical and toxicological behavior of TiO(2) and NiO nanoparticles. Chem Biol Interact. 2015 May 9;236:74-81. PMID: 25964212.
2. Zavala J, Lichtveld K, Ebersviller S, Carson JL, Walters GW, Jaspers I, Jeffries HE, Sexton KG, Vizuete W. The Gillings Sampler--an electrostatic air sampler as an alternative method for aerosol in vitro exposure studies. Chem Biol Interact. 2014 Sep 5;220:158-68. PMCID: PMC4252865.
3. McIntosh-Kastrinsky R, Diaz-Sanchez D, Sexton KG, Jania CM, Zavala J, Tilley SL, Jaspers I, Gilmour MI, Devlin RB, Cascio WE, Tong H. Photochemically altered air pollution mixtures and contractile parameters in isolated murine hearts before and after ischemia. Environ Health Perspect. 2013 Nov-Dec;121(11-12):1344-8. PMCID: PMC3855513.
4. Rager JE, Lichtveld K, Ebersviller S, Smeester L, Jaspers I, Sexton KG, Fry RC. A toxicogenomic comparison of primary and photochemically altered air pollutant mixtures. Environ Health Perspect. 2011 Nov;119(11):1583-9. PMCID: PMC3226493.

**Development of co-cultures to assess mechanisms of pollutant-induced toxicity**

To assess mechanisms underlying pollutant-induced adverse health effects, my lab has developed several co-culture methods. Specifically, we have developed innovative co-culture models of respiratory epithelial cells with dendritic cells, NK cells, or macrophages. These studies have demonstrated key roles of respiratory epithelial cells in orchestrating immune responses in the lung and communicating with immune cells in the context of pollutant exposures. Observations made in these projects highlight the importance of cell-cell communication and the critical role structural cells, such as epithelial cells, play in the activation of immune cells. I was the PI on all of these studies and have mentored several trainees affiliated with these publications.

1. Bauer RN, Müller L, Brighton LE, Duncan KE, Jaspers I. Interaction with epithelial cells modifies airway macrophage response to ozone. Am J Respir Cell Mol Biol. 2015 Mar;52(3):285-94. PMCID: PMC4370258.
2. Müller L, Brighton LE, Jaspers I. Ozone exposed epithelial cells modify cocultured natural killer cells. Am J Physiol Lung Cell Mol Physiol. 2013 Mar 1;304(5):L332-41. PMCID: PMC3602740.
3. Horvath KM, Brighton LE, Zhang W, Carson JL, Jaspers I. Epithelial cells from smokers modify dendritic cell responses in the context of influenza infection. Am J Respir Cell Mol Biol. 2011 Aug;45(2):237-45. PMCID: PMC3175553.

**D. Additional Information: Research Support**

**On-going Research Support**

1R01ES028269 Jaspers/Porter (MPI) 04/01/2018 – 03/31/2023

NIEHS/NIH

**Ozone, Oxysterols and Lung Inflammation**

The overall objective of this application is to determine how formation of oxysterols and oxysterol-protein adducts link O3-induced chemical reactions with biological effects.

Role: MPI

R01HL139369 Jaspers (PI) 09/01/2017 to 08/81/2021

NIH/NHLBI

**E-cig flavors and their effects on respiratory immune responses**

This application proposes highly integrated translational studies aimed at identifying mechanisms by which e-cigarette flavorings such as cinnamaldehyde affect respiratory innate immune responses in humans.

Role: PI

FAMRI CIA #160016 Jaspers (PI) 07/01/2017 - 06/30/2020

Flight Attendant Medical Research Institute

**Novel Approach to Overcome CRS-Induced Immune Dysfunction**

The overarching hypothesis of this proposal is that teaching socially meaningful positive emotions through LKM shifts nasal mucosal and systemic immune responses towards less inflammation and greater NK cell function, thus providing a novel approach to counterbalance the pathophysiology associated with CRS.

Role: PI

5R01ES025198 Tilley (PI) 05/01/2015 to 01/31/2020

NIH/NIEHS

**Pathogenesis of Ozone-Induced Asthma Exacerbation**

The objective of this proposal is to elucidate the cellular and molecular mechanisms by which zone causes asthma exacerbations, with the overarching goal of informing the rationale design of clinical trials targeting pathways identified by this investigation.

Role: Co-investigator

P50 HL120100-05 Tarran (PI) 9/19/2013 – 6/30/2019

FDA/NIH/HNLBI

**The Impact of Tobacco Exposure on the Lung’s Innate Defense System**

This project will describe the effects of tobacco alternatives on lung health using in vitro and in vivo model systems as well as obtaining biomarker samples from smokers of tobacco alternatives such as Hookah and Little Cigars.

Role: Principal Investigator: Project 4

Role: Principal Investigator: Training Core

CR83578501 Peden (PI) 02/01/2015-03/31/2022

U.S. Environmental Protection Agency

**Translational Research Center for Environmental Medicine and Toxicology-Admin Core**.

This core will provide oversight, coordination and integration of Center activities and interfaces with EPA management and key investigators.

Role: Investigator

CR83578501 Peden (PI) 02/01/2015-03/31/2022

U.S. Environmental Protection Agency

**Translational Research Center for Environmental Medicine and Toxicology-Project 2-Health Effects Associated with Inhaled Particular Pollutants Derived from Specific Sources**.

The specific aims of this project are: SA1: Determine the Effects of Controlled Woodsmoke exposures on markers of viral replication and mucosal immune responses in healthy human volunteers. SA2: Using *in vitro* models of differentiated human nasal epithelial cells assess mechanisms of interactions between woodsmoke particles and influenza virus at the level of the epithelium SA3: Using co-culture models, determine effects of woodsmoke on mucosal cell-cell communication in the context of viral infections.

Role: Project Lead

CR 83591401 Jaspers (PI) 12/01/2015 – 11/30/2018

U.S. Environmental Protection Agency

**Cooperative Training Partnership between the U.S. EPA and the UNC-CH Training Collaboration in Toxicology and Environmental Sciences (TC-ToxES)**

The objective of this project is to provide research-training opportunities to Predoctoral and postdoctoral trainees in the environmental sciences through a cooperative agreement between the University of North Carolina (UNC-CH) and the U.S. EPA Office of Research and Development (ORD).

Role: PI

P30ES010126 Troester 02/01/2000 – 03/31/2021

NIH/NIEHS

**UNC Center for Environmental Health and Susceptibility**

**The overall goals of the UNC-CEHS are: Goal 1. To disseminate knowledge and technology across interdisciplinary groups of researchers in environmental health. Goal 2. To foster outstanding translational research and outreach in three focus areas: Environmental Cancer, Cardiopulmonary Disease and Developmental Disease. Goal 3. To respond to emerging environmental threats facing North Carolinians and the nation. The Administrative Core efficiently administers resources, ensures connectivity across UNC-CEHS via multiple enrichment activities, ensures engagement of members in stakeholder and community discussions and strategically responds to North Carolina environmental health needs.**

**Role: Co-Investigator, Admin Core**

**Completed Research Support (list projects completed WITHIN the last 3 years ONLY)**

Society of Toxicology Jaspers                                05/1/2018-07/31/2018

**2018 SOT Summer Undergraduate Internship Program (Summer of Learning and Research-SOLAR)**

Our goal is to provide opportunities for undergraduate students (rising juniors and seniors) from under-represented backgrounds who are interested in biomedical research careers to gain hands-on research experience under the guidance of a UNC faculty mentor. In addition to the lab experience, students participate in weekly journal clubs weekly professional development workshops aimed at helping them prepare competitive applications for the PhD programs, and attend a weekly GRE preparation course.

Role: PI

**Ozone, Lipid-Protein Adducts, and Biological Effects**

This project is aimed to develop new tools and methods to examine the role of ozone-derived lipid-protein adducts in causing adverse health effects in the lung.

Role: Principal Investigator

NIH/NIEHS R01ES013611                        7/1/06-6/30/17

**Diesel-Induced Alterations of Influenza Infectivity**

The purpose of this project is to investigate the effects of diesel exhaust particle exposure on epithelial antiviral and inflammatory pathways in the setting of influenza.

NIH/NIEHS R01ES013611-08S2              9/12/13-6/30/17

**Diesel-Induced Alterations of Influenza Infectivity ViCTER Supplement**

This will be a randomized, prospective comparison study comparing cohorts of normal or AR subjects randomized to receive either DE (100-300 ug/m3 x 2 hr at rest) or placebo (clean air), followed by a standard dose of LAIV. Nasal lavage fluids and biopsies will be sampled at intervals during the resulting self-limited infection.

FAMRI            , Inc. CIA 12 #123009                                 7/1/13-6/30/17

**Cigarette Smoke, Viral infections and NK Cells**

This project will build on published and preliminary data regarding the mechanisms of Cigarette Smoke –induced alterations of NK cell function in the context of viral infection, to use an experimental co-culture model to assess the role of epithelial cells in these responses, and to explore whether EGCG could be used as a therapeutic strategy in upper respiratory tract viral infections

**Cigarette Smoke and Susceptibility to Influenza Infection**

NIH/NHLBI R01 HL095163

8/12/09-7/31/15

This proposal is designed to test the hypothesis that chronic exposure to cigarette smoke alters epithelial antiviral and inflammatory responses to influenza virus infection via two potentially related mechanisms: decreased expression of phase II (antioxidant) enzymes and suppression of type 1 interferon (antiviral) pathways.