

**Proposal for the Establishment of a Board of Trustees Approved
Center or Institute**

Proposed name of Center or Institute:

Center for Healthy Air Water and Soil (CHAWS)

Physical Address/Location:

Second Floor, Cardiovascular Innovation Institute

University official to whom Center or Institute reports:

Executive Vice President for Health Affairs (EVPHA)
Executive Vice President for Research and Innovation (EVPRI)

Name and title of individual submitting this proposal:

Dr. Aruni Bhatnagar, Professor of Medicine

Anticipated Date of Initiation of this Center or Institute:

July 1, 2018

April 24, 2018

Robert Keynton, PhD
Interim Executive Vice President for Research and Innovation
University of Louisville

Dear Dr. Keynton,

I am writing to you to request the creation of the Center for Healthy Air Water and Soil (CHAWS). The Center will provide a multi-disciplinary umbrella for community-based environmental research.

The mission of the Center is to support and promote innovative, environmental research and projects focused on a health in all policies approach, using the city of Louisville as an urban laboratory. By empowering citizens and using inventive technology, the Center aspires to improve the health and harmony of our community and our world. The Center will gather people and data to encourage new conversations and find new solutions to health challenges in our community and our world.

Following is an overview of the purpose of the Center, its activities or outcomes as well as its overall objectives and goals.

1. Purpose of the Proposed Center: (indicate why a separate organizational structure is needed to fulfill this purpose)

The Center will bring together faculty with diverse interests and backgrounds as well as community members and leaders to collectively bear upon local environmental problems and issues. These range from the development of innovative measurements of environmental exposures; identifying, assessing, and characterizing environmental exposure, and finding unique, local solutions to environmental problems. These approaches will require input from a variety of disciplines, including health science, public health, toxicology, communication, law, business, psychology, economics and government. Hence, a unique and quasi-independent organizational structure is required to fulfill the overall mission of the Center. This independent organizational structure will also enable participation of community members and leaders, in ways not supported by conventional academic units and departments.

Relationship of the Center to the Mission of the University:

The mission of the University is to pursue excellence in its work to educate and serve its community through teaching; practicing and applying research, scholarship and creative activity; and notably, providing engaged service and outreach that improve the quality of

life for local and global communities. The Center will support and further this mission by facilitating and encouraging environmental research and scholarship, partnering with faculty across campus to incorporate environmental issues into graduate and undergraduate courses, and in facilitating engagement of university faculty and personnel with the local community and community leaders so as to enable bidirectional interactions and outreach programs focused on issues related to environmental health. This will support the University's mission of achieving preeminence as a nationally recognized metropolitan research university.

Organizational Relationships: (indicate linkage to the mission of each college, school, unit, department or program of which the Center will be a part)

Along with other Centers involved in environmental research, CHAWS will be nested within the existing Kentucky Institute for the Environment and Sustainable Development (KIESD). There is a forthcoming request to rename KIESD to the Envirome Institute to reflect new leadership and expansion of scope. This request is a Board of Trustees action item scheduled to be considered during the next Board meeting on May 17, 2018. Therefore, KIESD is henceforth referred to as the Envirome Institute. All Centers to be affiliated with the Envirome Institute are existing Centers, except for CHAWS, which will be the sole new Center within the Institute.

The mission of the Envirome Institute is to enable and support research on the effects of the environment on health and to promote an understanding of environmental health issues, both locally and globally. The CHAWS will facilitate the Institute in its community outreach and engagement activities. It will form an interface between the University and its community and using the city of Louisville as an urban laboratory, enable studies on how environmental differences within the city give rise to health disparities, and how, local social networks affect lifestyle choices, such as nutrition and physical activity that bear upon individual health and well-being within a community. The Center will support extensive community outreach activities for promoting interaction and collaboration with the community, which will involve specific community events for bidirectional exchange of information, recruitment of community participants into scientific studies, dissemination of environmental information to the community and community consultation in developing research programming of relevance to the environment and community health. To seek input on scientific, environmental and community research, the Center will support a visiting scholars program.

The Center will also be linked to research and educational activities supported by the newly established, Superfund Research Program, funded by the National Institutes of Health from 2017-2022. The Center will support the Superfund program in its Research Translational and Community Engagement activities. The overall goal of the Superfund program is to conduct leading-edge research on the cardiometabolic effects of environmental pollutants such as volatile organic chemicals; discover and validate biomarkers for exposure

assessment and create new technologies to detect Superfund chemicals in the environment. The CHAWS will support the Superfund program in its mission to educate and train new investigators in the field of environmental science; create collaborative networks; and facilitate the sharing of educational resources and research activities. CHAWS will be particularly helpful in accomplishing the Superfund program's mission of promoting awareness and participation of the local community. This will enhance mutual bidirectional understanding of exposure risk, health effects, and resilience to the toxicity of hazardous chemicals. Such activities will also enable the transfer of scientific understanding from the Program to affected or interested communities and end-users in the public and private sector and other stakeholders.

The CHAWS will also greatly facilitate the mission of the Diabetes and Obesity Center (DOC). The Diabetes and Obesity Center was established 10 years ago, with the mission of supporting a strong research program and developing infrastructure to promote diabetes and obesity research at the University of Louisville. While the overall goal of the DOC is to foster research in diabetes and obesity by providing support for innovative and impactful projects and to support basic and translational cardiovascular research and training, the Center also supports a range of translational studies as well as community-based research to promote a better understanding of diabetes and obesity and to identify environmental factors that contribute to metabolic disease. The CHAWS will facilitate the progression and continuation of studies related to the impact of the environment on the risk of developing diabetes and obesity by facilitating the recruitment and retention of community participants, organizing community events for evaluation of diabetes and obesity in our community and in identifying the underlying contributing factors. Such activities would particularly benefit studies focused on assessing community-wide risk burden and their underlying causes. By supporting students engaged in studying the environmental determinants of diabetes, obesity and cardiovascular disease, CHAWS will provide new and unique opportunities for innovative research in this area, by strengthening ties with the community and leveraging community resources for conducting community-participatory research. Support of students, educational activities, and community partnerships supported by CHAWS will greatly benefit junior DOC investigators in the development of new research programs that could attract additional federal and local funding.

Research activities, educational opportunities, and community engagement that are critical to the mission of the Envirome Institute, the Superfund Program and the Diabetes and Obesity Center, will not be possible without the creation and successful operation of CHAWS.

- 2. Unit and Unit Head to provide oversight to the Center** - Dr. Aruni Bhatnagar, Director, Diabetes and Obesity Center and the proposed Director of the Envirome Institute.

3. Center Administration

The Center will be directed by faculty with experience in academic research, community outreach and civic engagement. After a national search, the selection committee at the Diabetes and Obesity Center have selected Dr. Ted Smith as a potential candidate. Approval of his appointment within the School of Medicine is pending. A copy of his CV is attached.

In addition to the Director, the Center will have a 100% administrator for coordinating all administrative activities relevant to the Center. The Center will seek operational advice from an External Advisory Board comprised of leaders from the community engagement team at UofL and invited community members with recognized expertise in fields that will strengthen the CHAWS mission. The Board will be created following the formal establishment of the Center.

The following key faculty will be members of the Center and will devote approximately 5% FTE to Center related activities for the first three years of the Center's operation. These are –

- Dr. Aruni Bhatnagar, Professor of Medicine
- Dr. Sanjay Srivastava, Professor of Medicine
- Dr. Daniel Conklin, Professor of Medicine
- Dr. Lauren Heberle, Associate Professor, Sociology
- Dr. Petra Haberzettl, Assistant Professor of Medicine
- Dr. Alex Carll, Assistant Professor, Physiology and Biophysics
- Dr. Joy Hart, Professor in Arts and Sciences, Communication
- Dr. Kandi Walker, Professor in Arts and Sciences, Communication
- Dr. Rachel Keith, Assistant Professor of Medicine
- Dr. Timothy O'Toole, Assistant Professor of Medicine

As the mission of the Center dovetails well with existing research and scholarly activities supported by the Superfund Research Program and other externally funded projects, Center faculty will not be required to commit effort specific to Center activities. As an example, Dr. Lauren Heberle serves as the Director of the Community Engagement Core of the Superfund Research Program and therefore this effort aligns well with membership within CHAWS. Faculty effort committed to new programs developed by the Center will be budgeted in individual grant proposals. A copy of the NIH Biosketch for each of the proposed Center members is included. New members to the Center will be recruited as required. Should any of the Center members be unable to participate in the activities of the Center, the Center will appoint new members in consultation with the External Advisory Board of the Center.

Projected Financial Information

- 4. Anticipated amount and source of revenue:** The Center will be funded by a philanthropic gift of \$500,000 per year for the first 5 years by Christina Lee Brown. The gift is contingent upon institutional establishment of CHAWS, appointment of Center faculty and staff and allocation of appropriate space for Center operation. Conversely, operational start-up of the CHAWS is contingent upon receipt of the gift. No other sources of revenue have been identified at this time. The CHAWS will not receive any center research incentive funds (C-RIF).

It is expected that the Center will be fully supported after Year 5 by extramural grants and/or gifts/endowments.

- 5. Funds needed to operate the Center:** The following is the proposed budget of the Center per year for the first 5 years.

Proposed Annual Budget	
Personnel	\$420,000
Community Events	\$25,000
Travel	\$10,000
Office supplies, Miscellaneous	\$15,000
Guest Speakers	\$30,000
Total (per year)	\$500,000

The Center will operate with a defined budget of \$500,000 per year and funding will be redistributed across budget categories as necessary to address the changing needs of the Center. Any additional Center expenses will only be incurred if/when additional external funding is obtained.

- 6. Needs of the Center:** As indicated in the budget above, the Center will need a full time (100% FTE) Director as well as a 100% FTE Administrator. Both of these positions will be supported by CHAWS and there will be no reassignment or replacement of any classified staff or personnel currently working for UofL. A new administrator will be recruited for the Center.
- 7. Space:** The Center will need at least 2 offices (at least 150 sq. feet) for the Director and the Administrator, as well as space for meeting and consultation and access to an auditorium for presentation by invited visiting scholars. The offices will be located in the Cardiovascular Innovation Institute (CII), as recently pledged by Dr. Postel. Students and fellows supported by the Center will work in existing laboratory spaces of their mentors

who currently have space provided by the Diabetes and Obesity Center and the Superfund Research Program.

- 8. Equipment and Other Infrastructure Resources:** The Center does not require specialized dedicated equipment. The research supported by the Center will be conducted in existing laboratories within the DOC and the Superfund program.
- 9. Written Statement concerning adequacy of resources:** A written statement from the Dean, University Libraries regarding the adequacy of current resources is attached.
- 10. Written Statement verifying financial commitment:** A letter from the Offices of the Executive Vice President for Health Affairs (EVPHA) and Executive Vice President for Research and Innovation (EVPRI) attesting to the adequacy of resources is attached.
- 11. Evaluation of the Center:** The progress of the Center will be evaluated on the basis of the following measurable goals. These include:
 - (a) Acquisition of at least \$200,000 in new direct funding per year attributable to Center members after two years of creation of the Center;
 - (b) Graduation of at least 3 PhD or MD-PhD students, supported by the Center within 5 years of its inception;
 - (c) Publication of at least 5 full-length manuscripts authored by Center members over 5 years;
 - (d) Organization of at least 6 community events relevant to dissemination of environmental health information, study participation and retention;
 - (e) Outreach to more than 300 members of the community each year;
 - (f) At the end of the first 5 years of support, the Center will be expected to continue to be fully self-supporting, which means acquisition of extramural funding and/or commitment of philanthropic support of at least \$500,000 in direct costs per year for the next 5 year period.

The data relevant to the evaluation of the Center will be collected every year and presented to the External Advisory Board for their input, advice and suggestions at the Board's annual meeting. Recommendations of the Board will be implemented by the Center Director, in consultation with Center members.

Annual Financial and Programmatic Reports will be submitted to the EVPHA and EVPRI, who will provide institutional oversight to the new Center.

January, 2018

Theodore Smith, Ph.D.
410 Trinity Hills LN
Louisville, KY 40207
(502) 386-5821
tedsmithphd@gmail.com

EDUCATION

1984-1988 B.S. in Biology and Psychology, Allegheny College, Meadville, PA
1988-1990 M.S. in Experimental Psychology, Miami University, Oxford, OH
1990-1992 Ph.D. in Experimental Psychology, Miami University, Oxford, OH
1992-1994 Postdoctoral Fellow, Man-Vehicle Laboratory, Massachusetts Institute of Technology, Cambridge, MA

ACADEMIC APPOINTMENTS

1992-1993 Adjunct Professor
Department of Psychology
Thomas More College
Fort Mitchell, KY

1992-1994 Adjunct Professor
Department of Psychology
Suffolk University
Boston, MA

1993-1994 Adjunct Professor
Experimental College
Tufts University
Medford, MA

2011-2013 Executive-in-Residence and Administrator, Health Sector Management MBA
College of Business
University of Louisville
Louisville, KY

2016 Adjunct Professor
School of Education
Spalding University
Louisville, KY

OTHER POSITIONS AND EMPLOYMENT

1994-1999 Director of Technology Education
Andersen Consulting (Accenture) LLP

St Charles, IL

1999-2000	Chief Executive Officer Gartner Institute, Inc Eden Prairie, MN
2000-2001	Vice President of Community Research TechRepublic.com Louisville, KY
2001-2006	Senior Vice President Business Brands CNET Networks, Inc Louisville, KY
2006-2008	Presidents and Co-Founder MedTrackAlert LLC Louisville KY
2008-2010	Senior Vice President of Research Health Central Networks Inc Arlington, VA
2010-2011	Senior Policy Advisory Office of the National Coordinator of Health IT U.S. Dept. of Health and Human Services Washington, DC
2013- 2016	Executive Director, Co-Founder Institute for Healthy Air, Water, and Soil Louisville, KY
2011- 2016	Chief of Civic Innovation Office of the Mayor Louisville Metro Government Louisville, KY
2016-Present	Chief Executive Officer Revon Systems Crestwood, KY

CERTIFICATION AND LICENSURE

2011	HHS Initial Ethics Orientation U.S. Department of Health and Human Services
2011	Contracting Overview Defense Acquisition University

2011	Cost Estimating Defense Acquisition University
2011	Improved Statement of Work Defense Acquisition University
2011	Cost Estimating Defense Acquisition University
2017	Combating Medicare Parts C and D Fraud, Waste, and Abuse Centers for Medicare and Medicaid Services U.S. Department of Health and Human Services

PROFESSIONAL MEMBERSHIPS AND ACTIVITIES

Professional Memberships

Association for Talent Development
Consumer Technology Association
AdvaMed

Service

2011-2013 Member, NUCLEUS Community Advisory Board, University of Louisville

Grant Review

2011-2016 Reviewer: Space Medical and Related Technologies Commercialization Assistance Program (SMARTCAP), National Space Biomedical Research Institute, Houston, TX

2011 Reviewer: ONC Challenge Grant — Consumer-Mediated Information Exchange, U.S. Department of Health and Human Services, Washington DC_

HONORS AND AWARDS

1996 Best Paper, World Conference on Education and Hypermedia, Boston, MA

1996 GOLD CINDY – Graphics Animation Category, International Association of Audiovisual Communications

2007 Bronze Award – World Wide Web Health Award, Health Information Resource Center

2007-2009 Research Fellow, Interactive Media Studies Armstrong Interactive Media Studies Program
Miami University Oxford, OH

2012	Champion of Change – Local Innovation, The White House
2012	Smarter Cities Challenge Winner – Louisville Smart Inhaler Program, IBM
2014	Top 25 Doers, Dreamers & Drivers in Public Sector Innovation, Government Technology Magazine
2014-2016	Community Fellow for Energy and the Environment, Conn Center for Renewable Energy Research, University of Louisville, Louisville, KY
2015	LUNGFORCE Champion Award– American Lung Association, Midwestern States

COMMITTEE ASSIGNMENTS AND ADMINISTRATIVE SERVICES

University

2013	Member, Search Committee for Executive Vice President of Research Initiatives, University of Louisville
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Non-university

1999-2003	National IT Skills Standards Advisory Board, Northwest Center for Emerging Technologies, National Science Foundation
2003-2005	Member, Social Network Analysis Roundtable, University of Virginia, Charlottesville, VA
2006-2008	Member, Board of Directors, Louisville Science Center, Louisville, KY
May, 2011	Organizing Committee, 2011 Health DATAPALOOZA, Institute of Medicine and U.S. Department of Health and Human Services
2012-present	Board of Directors, Enterprise Corp, Greater Louisville Inc., Louisville, KY
2012-2013	Board Chair, Board of Directors, Parking Authority of the River City (PARC), Louisville, KY
2012-2015	Board Member, Board of Directors, KentuckianaWorks, Louisville, KY
2012-2016	Board Member, XLerate Health, Louisville, KY
2016-Present	Board Member, Board of Directors, Goodwill Industries of Kentucky, Louisville, KY
2016-Present	Board Member, Board of Directors, Association for Talent Development (ATD), Arlington, VA
March, 2017	Organizing Committee, Sharing Knowledge to Build a Culture of Health Conference, Robert Wood Johnson Foundation
2017-Present	Co-Chair of Board, Institute for Healthy Air, Water and Soil, Louisville, KY

Status: Completed

2. Spatial Orientation during Simulated GPS Instrument Approaches
U.S. Department of Transportation VNTSC-57-92-C-00054
10/1/93-9/30/94
- | | |
|--------------------|-----------|
| Annual Direct Cost | \$ 50,000 |
| Total Direct Costs | \$ 50,000 |

Status: Completed

3. Founder's Distillery: New Approaches for Vetting Start-Ups"
Ewing Kauffman Foundation
3/1/13-6/30/13
- | | |
|--------------------|-----------|
| Annual Direct Cost | \$ 26,000 |
| Total Direct Costs | \$ 26,000 |

Status: Completed

4. Identifying Regional Environmental Drivers of Asthma and
Chronic Obstructive Pulmonary Disease and Improving Outcomes
in Louisville, Ky
Robert Wood Johnson Foundation 71592/73423
10/15/14-7/14/17
- | | |
|--------------------|-------------|
| Annual Direct Cost | \$ 336,656 |
| Total Direct Costs | \$ 841, 640 |

Status: Completed

As Collaborator

1. Development and Implementation of Preflight Adaptation Training (P.I. Donald Parker)
National Aeronautics and Space Administration NAG-9-446
10/01/1992 - 9/30/1993
- | | |
|--------------------|------------|
| Annual Direct Cost | \$ 79,043 |
| Total Direct Cost | \$ 158,086 |

Status: Completed

2. Role of Visual Cues in Spatial Orientation (P.I. Charles Oman)
National Aeronautics and Space Administration NAS9-19536
10/1/1995-9/31/1999
- | | |
|--------------------|------------|
| Annual Direct Cost | \$ 235,135 |
| Total Direct Cost | \$ 940,540 |

Status: Completed

3. Inclusive and responsive methods to reduce blighted and vacant properties (P.I. Greg Fischer)
Living Cities
8/01/2015-7/30/2016
- | | |
|--------------------|-----------|
| Annual Direct Cost | \$ 45,000 |
| Total Direct Cost | \$ 45,000 |

Status: Completed

As Consultant

N/A

As Mentor

N/A

PATENTS

1. **Carpenter-Smith; Theodore R.**, Gombar; Michael John, Fisher; James B., Barfield; Thomas M. System and method for interactively transforming a system or process into a visual representation. U.S. 5,838,973 Issued Nov 17, 1998
2. **Smith, Theodore R.**, and Christopher Lavenson, Notifications for reducing overflows from combined sewer systems and sanitary sewer systems U.S. 62/135,592 Issued Mar 20, 2015

EDITORIAL WORK***Scientific Review***

N/A

Editorial Board

August, 1998-2002

Editorial Board Member: *Interactive Learning Environments***Invited Lectures at National/International Meetings**

1. **Carpenter-Smith, T.R.** The Age of Integrated Delivery: Content as Commodity. Invited Keynote: The 13th Annual Conference on Distance Teaching & Learning, Madison, WI, August, 1997.
2. **Carpenter-Smith, T.R.** Integrated Internet Delivery: Learner as Employee and Retail Consumer. Invited Keynote: The 10th Anniversary of the Rensselaer Polytechnic Institute Continuing Education Program. Troy, NY, November, 1997
- 3.

Invited Seminars (National/International)

N/A

PUBLICATIONS***Articles Published in Peer-Reviewed Journals***

1. Wall, C., **Smith, T.R.** and Furman, J.M. Plasticity of the human otolith-ocular reflex. *Acta Otolaryngol.* 112(3), 413-20, 1992
2. **Carpenter-Smith, T.R.**, Futamura, R.G. and Parker, D.E. Inertial Acceleration as a measure of linearvection: An alternative to magnitude estimation. *Perception & Psychophysics*, 1995, 57 (1), 35-42.
3. Oman, C. M., I. Howard, **Carpenter-Smith, T.**, Beall, A., Natapoff, A., Zacher, J. and Jenkin, H. Neurolab experiments on the role of visual cues in microgravity spatial orientation. *Aviat Space Environ Med*, 71(3): 283., 2000
4. **Smith, T.**, Coyle, J.R., Lightfoot, E. and Scott, A., Reconsidering Models of Influence: The relationship between consumer social networks and word-of-mouth effectiveness. *Journal of Advertising Research*, 47 (4), 387-395, 2007

5. Van Sickle, D., Su, J., Barrett, M., Humblet, O., Henderson, K., **Smith, T.** Impact of a mobile health pilot study on asthma rescue inhaler use, control and self-management, *European Respiratory Journal* 46 (59) 2528, 2015
6. Barrett, M.A., Henderson, K., Humblet, O., Hogg, C., Nesbitt, L., Su, J.G., **Smith, T.**, Sublett, J.W., Sickle, D.V., & Sublett, J.L. Feasibility of deploying inhaler sensors to identify the impacts of environmental triggers and built environment factors on asthma short-acting bronchodilator use. *Environmental Health Perspectives*. 125(2), 254-261, 2017
7. Swaminathan S., Qirko K., **Smith T.**, Corcoran E., Wysham N.G., Bazaz, G. A machine learning approach to triaging patients with chronic obstructive pulmonary disease. PLoS ONE 12(11): e0188532, 2017
<https://doi.org/10.1371/journal.pone.0188532>

Commentaries

N/A

Books and Book Chapters

1. Oman, C. M., I. Howard, **T. Smith**, A. Beall, A. Natapoff, J. Zacher and H. Jenkin. (2003). The Role of Visual Cues in Microgravity Spatial Orientation. The Neurolab Spacelab Mission: Neuroscience Research In Space. J. Buckey and J. Homick, Eds. Houston, TX, NASA Johnson Space Center. NASA SP-2003-535: 69-82.

ABSTRACTS AND PRESENTATIONS

Abstracts

4. **Smith, T.** and Parker, D.E. The effects of unidirectional visual surround translation on detection of physical linear motion detection. *Annals of the New York Academy of Sciences*, 656, 817-819, 1992
5. **Carpenter-Smith, T.**, and Parker D.E. Development of a dependent variable to assess self-motion perception. 27th Meeting of the Barany Society, Prague, Czechoslovakia, June, 1992
6. **Carpenter-Smith, T.**, E. Katz, D. Merfeld, C. M. Oman and K. A. Polutchko. Perceived velocity in linear translation is affected by temporal frequency. Society for Neuroscience 23rd Annual Meeting. November, 1993
7. **Carpenter-Smith, T.R.**, and McDowell, M. A Virtual Reality Tool for Visualizing Technical Design Concepts. *Second International Conference on the Learning Sciences (ICLS 96)*, Evanston, IL, July, 1996
8. **Carpenter-Smith, T.R.** Scalable Distance Learning Approaches. *Distance Learning - The Strategic Revolution in Professional Development and Employee Education*, Massachusetts Institute of Technology, November 1996
9. **Carpenter-Smith, T.R.** Technologies that Redefine Professional Education. *Technology Assisted Learning*, February, 1997
10. Oman, C. M., Howard, I., **Carpenter-Smith, T.** and Beall, A. Neurolab Experiments on the Role of Visual Cues in Microgravity Spatial Orientation. *12th Man-In-Space Symposium*, "The Future of Humans in Space", Washington, DC, 1997
11. **Smith, T.R.** and Harkins, P. The Learning Organization: Managing and Developing Human Capital. *The 83rd Annual Conference of the University Continuing Education Association*, Chicago, IL, April, 1998.
12. Oman, C. M., Howard, I., **Carpenter-Smith, T.**, Beall, A., Natapoff, A., Zacher, J. and Jenkin, H. Role of visual cues in microgravit

spatial orientation. *NASA STS-90 Neurolab Spacelab Mission Symposium*, National Academy of Sciences, Washington, DC, 1999

13. Liu, A., Oman, C.M., Beall, A., Howard, I., **Smith, T.**, Young, L.R., Harris, L. and Jenkin, H. Human Orientation in Prolonged Weightlessness (ISS HRF-E085). *Conference on International Space Station Utilization*, American Institute for Aeronautics and Astronautics, Cape Canaveral, FL, 2001
14. Oman, C. M., Howard, I.P., **Smith, T.**, Beall, A., Natapoff, A., Zacher, J. and Jenkin, H. STS-90 Experiments on the Role of Visual Cues in Microgravity Spatial Orientation. *USRA Bioastronautics Investigators Workshop*, Universities Space Research Association, Division of Space Biomedicine. Galveston, TX, 2001
15. **Smith, T.**, How Technology-Purchasing Decisions Are Really Made (Social Network Analysis) *Network Roundtable of the University of Virginia Spring Meeting*, Boston, MA, April, 2006
16. **Smith, T.** How Context Helps Turn on a Mind. *AAAA/ARF Consumer Engagement Conference*, New York, NY, September, 2006
17. **Smith, T.** Why Consumers Cannot Resist Giving Advice – Social Networking Demystified. *Advertising Research Foundation Annual Meeting Re:think*, New York, NY, April, 2007.
18. Van Sickle, D., and **Smith, T.** Louisville Asthma Data Innovation Project. *Health Datapalooza IV*, Washington DC, June, 2013
19. Van Sickle, D., Su, J., Barrett, M., Humblet, O., Henderson, K., **Smith, T.** Identifying environmental drivers of asthma hotspots in Louisville, Kentucky, using sensors to capture spatially-explicit, real-time data on inhaler use: AIR Louisville program. *American Public Health Association (APHA) Annual Meeting*, Chicago, IL 2015

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: Bhatnagar, Aruni

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

POSITION TITLE: Professor of Medicine, University of Louisville

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 Lucknow, Lucknow, India	B.Sc.	1979	Chemistry, Biology, Zoology
University of Lucknow, Lucknow, India	M.Sc.	1981	Biochemistry
University of Kanpur, Kanpur, India	Ph.D.	1986	Chemistry
University of Texas Medical Branch, Galveston, TX	Post-Doc	1986-1990	Biological Chemistry

B. Positions and Honors**Positions and Employment**

1990 - 1995	Assistant Professor, Dept. of Human Biological Chemistry & Genetics and Dept. of Physiology and Biophysics, University of Texas Medical Branch, Galveston, TX
1995 - 1998	Associate Professor, Dept. of Human Biological Chemistry & Genetics and Dept. of Physiology and Biophysics, University of Texas Medical Branch, Galveston, TX
1998 - present	Professor, Division of Cardiology, Dept. of Medicine; Department of Biochemistry and Biophysics and the Department of Pharmacology and Toxicology, University of Louisville, Louisville, KY
2008 – present	Director, Diabetes and Obesity Center, University of Louisville, Louisville, KY.

Other Experience and Professional Memberships

1998 – 2003	Member, ALTX-4, Toxicology Study Section, National Institutes of Health
1999 – 2004	Member, Editorial Board, <i>Journal of Pharmacology and Experimental Therapeutics</i>
2004 – 2009	Member, Editorial Board, <i>Circulation Research</i>
2009 – present	Member, Editorial Board, <i>Journal of Molecular Cellular Cardiology</i>
2009 – present	Deputy Editor, <i>Circulation Research</i>
2008 – 2015	Guest Editor, Member Editorial Board, <i>Circulation</i>
2006 – 2013	Member, Louis M. Katz Young Investigator Award Committee, <i>American Heart Association</i>
2008 - 2014	Member, External Advisory Committee, <i>Center for Environmental Health Sciences</i> , University of Montana, Missoula, Montana
2008 – 2012	Scientific Advisor, NIEHS Discover Center on Cardiovascular Disease and Traffic Related Air Pollution, University of Washington, Seattle, WA.
2009	Member, Expert Panel Population and Prevention Science of the <i>American Heart Association (Air Pollution and Heart Disease)</i> .
2009	Member, <i>Institute of Medicine</i> Committee on Secondhand Smoke Exposure and Acute Coronary Events.
2010	Member, <i>Institute of Medicine</i> Committee on Long-term Health Consequences of

2013-2015	Exposure to Burn Pits in Iraq and Afganistan. Member, Clinical and Integrative Cardiovascular Sciences Study Section, Center for Scientific Review
2015-2017	Chair, Clinical and Integrative Cardiovascular Sciences Study Section, Center for Scientific Review

Honors

1976	National Science Talent Award, India
1998 – present	University Scholar, & Distinguished University Scholar, University of Louisville
2005 – present	Fellow, American Heart Association & Basic Cardiovascular Sciences
2007	President's Award for Outstanding Scholarship, Research and Creative Activity
2007	Partner in Healthcare Award – Contributing to Greater Louisville Healthcare Community
2014	Outstanding Mentor Award, Conference of Southern Graduate Schools (CSGS)
2016	Finalist, One Brave Idea, American Heart Association, Verily, AstraZeneca
2017	Research Exemplar Award, Washington University, St. Louis, MO

C. Contribution to Science

1. My early work was focused on the elucidation of the effects of free radicals and oxidative stress on myocardial excitability and conductance. I found that exposure of isolated cardiac myocytes to free radicals induces specific alterations in ion conductances, particularly the sodium channel. These changes led to alterations in cardiac action potential and intracellular calcium and sodium concentrations. These changes are expected to cause severe conductance defects in the heart and could account for the arrhythmogenic effects of conditions associated with oxidative stress, such as myocardial ischemia-reperfusion injury. This work led to detailed investigations on the mechanisms by which oxidative stress contributes to myocardial ischemic injury and heart failure.

- a. Conklin DJ, Guo Y, Jagatheesan G, Kilfoil PJ, Habertzettl P, Hill BG, Baba SP, Guo L, Wetzelberger K, Obal D, Rokosh DG, Prough RA, Prabhu SD, Velayutham M, Zweier JL, Hoetker JD, Riggs DW, Srivastava S, Bolli R, Bhatnagar A. Genetic Deficiency of Glutathione S-Transferase P Increases Myocardial Sensitivity to Ischemia-Reperfusion Injury. *Circ Res*. 2015 Aug 14;117(5):437-49. PubMed PMID: [26169370](#); PubMed Central PMCID: [PMC4854443](#).
- b. Srivastava S, Vladykovskaya E, Barski OA, Spite M, Kaiserova K, Petrash JM, Chung SS, Hunt G, Dawn B, Bhatnagar A. Aldose reductase protects against early atherosclerotic lesion formation in apolipoprotein E-null mice. *Circ Res*. 2009 Oct 9;105(8):793-802. PubMed PMID: [19729598](#); PubMed Central PMCID: [PMC3548455](#).
- c. West MB, Rokosh G, Obal D, Velayutham M, Xuan YT, Hill BG, Keith RJ, Schrader J, Guo Y, Conklin DJ, Prabhu SD, Zweier JL, Bolli R, Bhatnagar A. Cardiac myocyte-specific expression of inducible nitric oxide synthase protects against ischemia/reperfusion injury by preventing mitochondrial permeability transition. *Circulation*. 2008 Nov 4;118(19):1970-8. PubMed PMID: [18936326](#); PubMed Central PMCID: [PMC2763350](#).
- d. Bhatnagar, A., Srivastava, S.K. and Szabo, G. Oxidative stress alters specific membrane currents in isolated cardiac myocytes. *Circ Res* 1990, Sep; 67(3):535-49. PubMed PMID: [2168816](#); PubMed Central PMCID: [PMC4531836](#).

2. Along with the metabolism and detoxification of endogenously generated aldehydes generated during lipid peroxidation, I have also studied the metabolism and the toxicity of aldehydes that are generated as important toxic components of environmental pollutants. This line of enquiry led me to examine the cardiovascular toxicity of several environmental pollutants as well as tobacco smoke which contained several reactive aldehydes in high concentration. These studies have led to the development of a new field of environmental cardiology, focused on studying the link between exposure to environmental pollutants and cardiovascular disease.

- a. **Bhatnagar, A.** Environmental cardiology: studying mechanistic links between pollution and heart disease. *Circ Res* 2006, Sep 29; 99(7): 692-705. PubMed PMID: [17008598](#)
- b. **Bhatnagar, A.** Cardiovascular pathophysiology of environmental pollutants. *Am J Physiol*, 2004 Fed; 286(2): H479-H485. PubMed PMID: [14715496](#)

- c. Abplanalp W, DeJarnett N, Riggs DW, Conklin DJ, McCracken JP, Srivastava S, Xie Z, Rai S, **Bhatnagar A**, O'Toole TE. Benzene exposure is associated with cardiovascular disease risk. *PLoS One* 2017 Sep 8;12(9):e0183602. PubMed PMID [28886060](#)
- d. O'Toole TE, Hellmann J, Wheat L, Habertzettl P, Lee J, Conklin DJ, Bhatnagar A, **Pope CA** 3rd. Episodic exposure to fine particulate air pollution decreases circulating levels of endothelial progenitor cells. *Circ Res* 2010 Jul 23; 107 (2):200-203. PubMed PMID [20595651](#)

3. In addition to my work on basic mechanisms of cardiovascular disease and the effects of environmental pollutants in animal models, I have also participated in the design, execution and analysis of several clinical and translational studies to examine changes in cardiovascular disease risk and outcomes. As PI of the Louisville Healthy Heart Study, I have assembled a cohort of over 1000 participants to assess the effects of roadway proximity and acrolein on cardiovascular disease risk. In collaboration with Dr. DeFilippis, I have assembled a cohort of 150 participants to identify biomarkers of atherothrombotic myocardial infarction. In the AHA Tobacco Center, I oversee the assembly of a 400 participant cohort to study the cardiovascular effects of tobacco products. With Dr. Pope, I have worked on a 3 year cohort studies of 90 participants on the effects of air pollution on endothelial dysfunction in young adults. I have also participated in data analysis from several cohorts, including the Cardiovascular Cell Therapy Research Network, Multi-Ethnic Study of Atherosclerosis (MESA), Jackson Heart Study and the Brazilian Longitudinal Study of Adult Health.

- a. Keith RJ, Al Rifai M, Carruba C, De Jarnett N, McEvoy JW, **Bhatnagar A**, Blaha MJ, Defilippis AP. Tobacco use, insulin resistance and the risk of type 2 diabetes: Results from the Multi-Ethnic Study of Atherosclerosis. *PLoS One*, e0157592, 2016. PubMed PMID [27322410](#)
- b. DeFilippis, A.P., Chernyvaskiy, I, Amraotkar, A.R., Trainor, P.J., Kothari, S., Ismail, I., Hargis, C.W., Korley F.K., Leibundgut, G., Tsimikas, S., Rai, S.N., and **Bhatnagar, A**. Circulating levels of plasminogen and oxidized phospholipids bound to plasminogen distinguish between atherothrombotic and non-atherothrombotic myocardial infarction. *J. Thromb. Thrombolysis*. 42: 61-76, 2016. PubMed PMID [26510751](#)
- c. Hall M.E., Wang W., Okhominina V., Agarwal M., Hall J.E., Dreisbach A.W., Juncos L.A., Winniford M.D., Payne T.J., Robertson R.M., **Bhatnagar A**, Young B.A., Cigarette Smoking and Chronic Kidney Disease in African Americans in the Jackson Heart Study. *J Am Heart Assoc*. 5: pii:e003280, 2016. PubMed PMID [27225196](#)
- d. **Bhatnagar A**, Bolli R., Johnstone B.H., Traverse J.H., Henry T.D., Pepine C.J., Willerson J.T., Perin E.C., Ellis S.G., Zhao D.X., Yang P.C., Cooke J.P., Schutt R.C., Trachtenberg B.H., Orozco A., Resende M., Ebert R.F., Sayre S.L., Simari R.D., Moyé L., Cogle C.R., Taylor D.A., Bone marrow cell characteristics associated with patient profile and cardiac performance outcomes in the LateTIME-Cardiovascular Cell Therapy Research Network (CCTRN) trial. *Am Heart J*. 179: 142-50. 2016. PubMed PMID [27595689](#)

4. My interest in issues related to the health effects of air pollution and tobacco products has also provided me the opportunity to participate or lead several policy statements and position papers that provide guidance to policy makers and other stakeholders in regulating issues related to environmental exposures. From this experience, I have learned to address issues most relevant to public health and to design and conduct meaningful and rigorous studies that could have a direct and meaningful impact such as the **Green Heart Project**.

- a. Brook, R.D., Rajagopalan, S., Pope, A., Brook, J.R., **Bhatnagar, A**, Diez-Roux, A., Holguin, F., Hong, Y., Luepker, R.V., Mittleman, M., Peters, A., Siscovick, D., Smith, S.C., and Kaufman, J.D. Air pollution and cardiovascular disease: An update to the scientific statement for healthcare professionals from the interdisciplinary committee for prevention of the American Heart Association council on epidemiology and prevention. *Circulation* [121](#), 2331-2378, 2010. PubMed PMID: [20458016](#)
- b. **Bhatnagar, A**, Whitsel, L.P., Ribsil, K.M., Bullen, C., Chaloupka, F., Piano, M.R., Robertson, R.M., McAuley, T., Goff, D., Benowitz, N. on behalf of the American Heart Association Advocacy Coordinating Committee, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Quality of Care and Outcomes Research. Electronic Cigarettes: A Policy Statement from the American Heart Association. *Circulation* 2014, Oct 14;130(16)1418-36. PubMed PMID: [25156991](#).

- c. Whitsel, L., Benowitz, N., **Bhatnagar, A.**, Bullen, C., Goldstein, F., Matthias-Gray, L., Grossmeier, J., Harris, J., Isaac, F., Loeppke, R., Manley, M., Mosley, K., Niemiec, T., O'Brien, V., Palma-Davis, L., Pronk, N., Psnock, J., Richling, D., Stave, G.M., and Terry, P. Guidance to employers on integrating e-cigarettes/electronic nicotine delivery systems into tobacco worksite policy. *J. Occup. Environ. Med.* 2015 Mar;57(3):334-43. PubMed PMID: [25742539](#)
- d. Schick SF, Blount BC, Jacob P 3rd, Saliba NA, Bernert JT, El Hellani A, Jatlow P, Pappas RS, Wang L, Foulds J, Ghosh A, Hecht SS, Gomez JC, Martin JR, Mesaros C, Srivastava S, St Helen G, Tarran R, Lorkiewicz PK, Blair IA, Kimmel HL, Doerschuk CM, Benowitz NL, **Bhatnagar A.** *Am. J. Physiol.* 2017, Sep 1;313(3) L425-L452. PubMed PMID: [28522563](#).

D. Research Support

1P50 HL120163 09/19/2013 – 09/18/2018

Bhatnagar (PI; Robertson Co-PI)

NIH/Federal Drug Administration (FDA)

American Heart Association Tobacco Regulation and Addiction Center

The overall goal of this Center is to increase knowledge across the full spectrum of basic and applied research on tobacco and addiction. No overlap with current project.

1P20GM103492 (Bhatnagar, PI) 09/26/2008 - 06/30/2018

NIH

Center of Excellence in Diabetes and Obesity Research

The primary objective and the central focus of the Center are to enable, promote, and support scientific research related to the cardiovascular causes and consequences of diabetes and obesity. The second major aim of program is to provide mentoring and guidance to junior investigators in the Center, with the overall goal of discovering new therapies for the treatment and prevention of diabetes and obesity.

1R01 ES19217 (Bhatnagar, PI; O'Toole, PI) 09/23/2011 - 05/31/2018

NIH/NIEHS

Endothelial Progenitor Cells and Particulate Air Pollution

This project is to understand how particulate air pollution affects endothelial progenitor cells and whether particulate matter-induced cardiovascular injury is in part due to dysregulation of stem cell mediated repair. No overlap with the current project.

1P01 HL 78825 (PI, Project 4, Bolli, PD) 04/15/2005 - 05/31/2022

NIH/NHLBI

Protection of Ischemic Myocardium

The major goal of this program is to examine the mechanisms by which cardiac progenitor cells mediate myocardial repair and recovery in heart failure. Project 4 of the program is to assess how diabetes affects the efficacy of stem cell therapy and to identify diabetes-induced changes in stem cell competency that limit efficacy of cell therapy and to examine how the effects of diabetes could be overcome to optimize cardiac recovery after stem cell transplant.

No overlap with the current project.

Completed Research Support

2R01 HL55477 (Bhatnagar, PI) 12/01/1998 – 05/31/2017

NIH/NHLBI

Detoxification and Toxicity of 4-Hydroxyalkenals in Heart

The aim of this project is to examine how 4-hydroxyalkenals derived from lipid peroxidation are metabolized in the heart and whether they contribute to cardiac ischemia-reperfusion injury. No overlap with the current project.

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: Srivastava, Sanjay

eRA COMMONS USER NAME (credential, e.g., agency login): S0SRIV01

POSITION TITLE: Professor of Medicine and Distinguished University Scholar, University of Louisville

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 Lucknow, Lucknow, India	B.S.	1987	Chemistry, Physics, Mathematics
University of Lucknow, Lucknow, India	M.S.	1989	Chemistry
University of Lucknow, Lucknow, India	Ph.D.	1993	Chemistry
University of Texas Medical Branch, Galveston, TX	Post Doc	1994-1998	Biological Chemistry

B. Positions and Honors

Positions and Employment

1989-1999 Research Scholar, Industrial Toxicology Research Center, Lucknow, India
1994-1999 Post-Doctoral Fellow, Department of Human Biological Chemistry and Genetics, The University of Texas Medical Branch, Galveston, TX
1999-2005 Assistant Professor, Dept. of Medicine, Division of Cardiovascular Medicine, University of Louisville, KY
2005-2011 Associate Professor, Dept. of Medicine, Division of Cardiovascular Medicine, University of Louisville, KY
2011- Professor, Dept. of Medicine, Division of Cardiovascular Medicine, University of Louisville, KY
2017- Director, University of Louisville Superfund Research Center (funded by NIH).

Other Professional Activities

Reviewer: NIH Study Sections

Chair: Clinical Cardiovascular Sciences Special Emphasis Panel: 2012

Regular Member: Vascular Biology Standing Special Emphasis Panel ZRG1 CVS-Q 90 S: 2007-2010

Chartered Member: Atherosclerosis and Inflammation in Cardiovascular Systems (AICS) July 2012-June 2018

Ad-hoc member: Clinical and Integrative Cardiovascular Sciences - 2008, 2009; Research Challenge Grants (Panel 19) – 2009; Vascular Cell and Molecular Biology - 2009, 2010; Vascular Biology Ad-hoc Special Emphasis Panels: ZRG02 – 2010; VH-D3 - 2010; ZRG1; F10A - 2010; ZRG1 VH-D (02) – 2010; ZRG1 VH-F (02) – 2011; AICS -2011

Editorial Board: Circulation Research

Awards and Honors:

Distinguished University Scholar, University of Louisville, 2012

Elected Fellow of the American Heart Association, 2010

Professional Membership:

American Heart Association

C. Contribution to Science

1. **Protein Chemistry and Enzymology:** Some of my earlier work was focused on studying the kinetic properties of the enzymes involved in the reduction of aldehydes generated from the oxidation of lipids. We, for the first time elucidated the biochemical pathways which are involved in the metabolism and detoxification of lipid peroxidation derived aldehydes in cardiovascular tissues. *Using*, 4-hydroxy-trans-2-nonenal (HNE) and 1-palmitoyl-2-oxoveroyl phosphatidylcholine (POVPC) as model aldehydes, we showed that reduction, primarily catalyzed by aldose reductase was a novel metabolic pathway for the detoxification of these highly reactive aldehydes. We used mass spectrometric approach for the characterization and quantitation of several small molecular weight analytes and to study the mechanisms of protein modifications.

i. Srivastava, S., Harter, T.M., Chandra, A., Bhatnagar, A., Srivastava, S.K., and Petrash, J.M. Kinetic studies of a FR-1, a growth factor-inducible aldose reductase. *Biochemistry* 37, 12909-12917, 1998. PubMed PMID: [9737870](#).

ii. Srivastava, S., Chandra, A., Wang, L., Seifert, W.E. Jr., DaGue, B.B., Ansari, N.H., Srivastava, S.K., and Bhatnagar, A. Metabolism of lipid peroxidation product, 4-hydroxy trans-2-nonenal in isolated rat heart. *J. Biol. Chem.* 273, 10893-10899, 1998. PubMed Central PMCID: [PMC3522116](#)

iii. Srivastava S., Spite M., Trent JO, West M.B., Ahmed Y., Bhatnagar A. Aldose reductase-catalyzed reduction of aldehyde phospholipids. *J. Biol. Chem.* 279, 53395-53406, 2004. PubMed Central PMCID: [PMC3475326](#)

iv. Spite M., Baba S.P., Ahmed Y.O., Barski O., Nijhawan K., Petrash J.M., Bhatnagar A., and Srivastava S. Substrate specificity and catalytic efficiency of aldose reductases with phospholipid aldehydes. *Biochem J.* 405:95-105, 2007. PubMed Central PMCID: [PMC1925154](#)

2. **Diabetes:** Apart from metabolizing the oxidized lipids-derived aldehydes, aldose reductase also reduces glucose to sorbitol. Therefore, we examined the contribution of aldose reductase in vascular complications of diabetes. Our data suggest that although inhibition of aldose reductase diminishes smooth muscle cell proliferation, genetic ablation of aldose reductase exacerbates atherosclerosis, possibly by preventing the metabolism of the precursors of advanced glycation end products.

i. Ramana, K.V., Chandra, D., Srivastava, S., Bhatnagar, A., Aggarwal, B.B., and Srivastava, S.K. Aldose reductase mediates mitogenic signaling in vascular smooth muscle cells. *J. Biol. Chem.* 277, 32063-32070, 2002.

ii. Srivastava S., Ramana K.V., Tammali R., Srivastava S.K., and Bhatnagar A. Contribution of aldose reductase to diabetic hyperproliferation of vascular smooth muscle cells. *Diabetes* 55, 901-910, 2006. PubMed Central PMCID: [PMC3463958](#)

iii. Reddy AB, Ramana KV, Srivastava S, Bhatnagar A, Srivastava SK. Aldose reductase regulates high glucose-induced ectodomain shedding of tumor necrosis factor (TNF)-alpha via protein kinase C-delta and TNF-alpha converting enzyme in vascular smooth muscle cells. *Endocrinology* 150: 63-74, 2009. PMCID:[PMC2630901](#)

iv. Baba S.P., Barski O.A., Ahmed Y., O'Toole T.E., Conklin D.J., Bhatnagar A. and Srivastava S. Reductive metabolism of AGE precursors: a metabolic route for preventing AGE accumulation in cardiovascular tissue. *Diabetes* 58:2486-97, 2009. PubMed Central PMCID: [PMC2768164](#)

3. **Vascular Toxicology and Inflammation:** One of the major focus of my laboratory is to delineate the mechanisms by which environmental toxins, tobacco products and other xenobiotics exert cardiovascular toxicity. Over the years, we have examined the mechanisms by which these toxins affect plasma lipoproteins, endothelial function and vascular inflammation, which eventually exacerbates atherosclerosis and thrombosis. These experiments entail extensive phenotyping of mice after toxicants exposures, and mass spectrometric approaches for the analyses of several analytes.

i. Srivastava S., Vladykovskaya E.N., Haberzettl P., Sithu S.D., D'Souza S.E., and States J.C. Arsenic exacerbates atherosclerotic lesion formation in apoE^{-/-} mice. *Toxicol. Appl. Pharm.* 241:90-100, 2009. PMCID:[PMC4414341](#)

- ii. Srivastava S, Ramana KV, Bhatnagar A and Srivastava SK. Synthesis, quantification, characterization and signaling properties of glutathionyl conjugates of enals. **Methods Enzymology** 474:297-313, 2010. PMID:[PMC3049297](#)
- iii. Conklin DJ, Malovichko M, Zeller I, Das TP, Krivokhizhina TV, Lynch BL, Lorkiewicz P, Agarwal A, Wickramasinghe N, Haberzettl P, Sithu SD, Shah J, O'Toole, T, Rai SN, Bhatnagar A, and Srivastava S. Biomarkers of chronic acrolein inhalation exposure in mice: Implications of Tobacco products-Induced Toxicity. **Toxicological Sciences** 158:263-274, 2017. PMID:[PMC5837482](#)
- iv. Malovichko MV, Zeller I, Krivokhizhina TV, Xie Z, Lorkiewicz P, Agarwal A, Wickramasinghe N, Sithu SD, Shah J, O'Toole T, Rai SN, Bhatnagar A, Conklin DJ, and Srivastava S. Systemic toxicity of smokeless tobacco products in mice. **Nicotine Tobacco Research** (in press).

4. Mechanisms of Vascular Pathophysiology and Inflammation: Our studies suggest that oxidized lipids-derived aldehydes cause endothelial cell and macrophage activation. Concentration of these aldehydes and their protein adducts are significantly increased in vascular pathologies, especially atherosclerosis. Our recent studies suggest that aldehyde-induced endoplasmic reticulum, could at least in parts, be involved in the pathogenesis of vascular complications. Our ongoing studies also suggest the oxidized lipids and reactive aldehyde induce the expression of micro RNA-21; and deficiency of micro RNA-21 affects macrophage apoptosis and necrosis. LC-MS/MS assays were used for the characterization of protein modification in JBC 2012 manuscript.

- i. Vladykovskaya E., Haberzettl P., Wickramasinghe N.S., Sithu S.D, Hill B.G., McCracken J., Dougherty S., D'Souza S.E., Barski O.A., O'Toole T., Bhatnagar A., and Srivastava S. The lipid peroxidation product, 4-hydroxy-trans-2-nonenal causes endothelial activation by inducing endoplasmic reticulum stress. **J. Biol Chem.** 287:11398-11409, 2012. PubMed Central PMID: [PMC3322871](#)
- ii. Zeller I and Srivastava S. Macrophage functions in atherosclerosis. **Circ Res** 115:e83-85, 2014. PMID: [PMC4414311](#)
- iii. Hellmann J, Tang Y, Zhang MJ, Hai T, Bhatnagar A, Srivastava S. and Spite M. Atf3 negatively regulates Ptg2/Cox-2 expression during acute inflammation. **Prostaglandins Other Lipid Mediat.** 116-117:49-56, 2015. PMID:[PMC4386069](#)
- iv. Ma X, Conklin DJ, Li F, Dai Z, Hua X, Li Y, Wysoczynski M, Sithu SD, Srivastava S, Bhatnagar A, and Yong Li. The Oncogenic MicroRNA miR-21 Enhances Programmed Necrotic Cell Death in Mice. **Nature Communications** 6:7151, 2015. PMID:[PMC4440243](#)

5. Molecular Mechanisms of Atherosclerosis: Because, our studies suggest that aldehydes generated from oxidized lipids, metabolism of VOCs, and as precursors of advanced glycation end products, affect endothelial cell and macrophage functions, we examined (a) how regulation of aldehyde metabolizing enzymes or exposure to reagent aldehydes exacerbate atherosclerosis, and (b) how does quenching of aldehydes by small molecular weight compounds prevents murine atherosclerosis. To examine the species independent affect of xenobiotic toxicity, recently, I established new LDL receptor-null rat models of atherosclerosis. The LDL receptor-null rats are also obese and glucose intolerant. Feeding the rats a western diet exacerbates obesity, glucose intolerance and atherosclerosis. We performed extensive phenotyping of experimental animals, and used mass spectroscopic approaches to examine protein modification.

- i. Srivastava S., Vladykovskaya E., Barski O.A., Spite M., Kaiserova K., Petrash J.M., Chung S.S., Hunt G., Dawn B., and Bhatnagar A. Aldose reductase protects against early atherosclerotic lesion formation in apolipoprotein E-null mice. **Circ. Res.** 105:793-802, 2009. PubMed Central PMID: [PMC3548455](#)
- ii. Srivastava S, Sithu SD, D'Souza SE, Vladykovskaya E, Haberzettl P, Hoetker DJ, Siddiqui MA, Conklin DJ, and Aruni Bhatnagar. Oral exposure to acrolein exacerbates atherosclerosis in apoE-null mice. **Atherosclerosis** 215: 301-308, 2011. PubMed Central PMID: [PMC3070047](#)
- iii. Barski OA, Xie Z, Baba SP, Sithu SD, Agarwal A, Cai J, Bhatnagar A, Srivastava S. Dietary Carnosine prevents early atherosclerotic lesion formation in apoE-null mice. **Arterioscl. Thromb. Vasc. Biol.** 33:1162-1170, 2013. PubMed Central PMID: [PMC3869200](#).
- iv. Sithu SD, Riggs KA, Winner MG, Agarwal A, Hamid-Berair RE, Kalani A., Riggs DW, Bhatnagar S, and Srivastava S. Atherogenesis and Metabolic Dysregulation in LDL Receptor Knockout Rats. **J. Clinical Investigation Insight** 2(9). pii: 86442. PMID: [PMC5414561](#)

D. Research Support

D. Research Support

Ongoing

P42 ES023716 (PI-Srivastava) 08/01/2017 – 03/31/2022

NIH/NIEHS

Environmental Exposure and Cardiometabolic Disease

Goal of the SUPERFUND Center is to examine the cellular and molecular mechanisms by which volatile organic compounds exert cardiometabolic toxicity. Our working hypothesis is that environmental toxins induce ER-stress in endothelial cells which leads to vascular insulin resistance, endothelial activation and atherogenesis.

1R01 HL120746 (PI: Srivastava, S.) 10/01/2013-9/30/2018

NIH/NHLBI

Tobacco Products and Atherosclerotic Disease

This project will examine the effect of tobacco smoke and its constituents on atherosclerosis.

1R01HL138992 (MPI: Fujise-Srivastava) 07/01/17-06/30/21

NIH/NIEHS

Using Fortilin Inhibitors to Block Atherosclerosis

This project will examine the mechanisms by which fortilins prevent macrophage activation and atherosclerosis.

1R01HL137229 (MPI: Chen-Srivastava) 07/01/17-06/30/21

NIH/NHLBI

Molecular Mechanisms Regulating Endothelial Dysfunction

This project will examine the mechanisms by which Epsins regulate endothelial functions and atherosclerosis.

T35 ES014559 (PI: McClain C.J., Prough R.A. and Srivastava S.) 04/01/11-03/31/21

NIH/NIEHS

Summer Environmental Health Sciences Training Program

Primary objective of the Summer Environmental Health Sciences Research Training Program is to provide environmental health science research experience to medical students.

P50 HL120163 (MPI- Robertson R and Bhatnagar A) 9/1/2013-6/30/2018

NIH/FDA

Role: (Project leader)

The goal of American Heart Association-Tobacco Regulation and Addiction Center is to identify the sensitivity of the cardiovascular system to harmful and potentially harmful constituents (HPHCs) in tobacco products.

1P20RR024489-01 (PI-Bhatnagar A) 07/01/2013-06/30/2018

NIH/NIGMS

Role: (Core Director-Bioanalytics and Pathology Core)

Center of Excellence in Diabetes and Obesity Research

The primary objective and the central focus of the Center are to enable, promote, and support scientific research related to the cardiovascular causes and consequences of diabetes and obesity.

Completed in last three years

R01 ES 17260 (PI: Srivastava, S.) 04/01/2009 – 03/31/2014

NIH/NIEHS

Atherogenic Mechanisms of Arsenic

The overall goal of this project was to understand the mechanisms by which exposure to arsenic accelerates or aggravates atherosclerosis.

1R01 HL-95593 (PI: Srivastava, S.) 03/15/2010 – 02/29/2016

NIH/NHLBI

Atherogenic Mechanisms of Lipid Peroxidation-Derived Aldehydes

The goal of this project is to understand the mechanisms by which lipid derived aldehydes affect atherogenesis and how the effects of these aldehydes could be prevented or therapeutically minimized to decrease atherosclerosis.

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: Conklin, Daniel J.

eRA COMMONS USER NAME (credential, e.g., agency login): D0CONK01

POSITION TITLE: Professor of Medicine

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
Univ. of Wisconsin-Whitewater, Whitewater, WI	B.S.	05/86	Biology, Chemistry
Univ. of Wisconsin-LaCrosse, LaCrosse, WI	M.S.	08/89	Biology
Univ. of Notre Dame, Notre Dame, IN	Ph.D.	12/95	Cardiovascular Phys.
Univ. of Texas Medical Branch, Galveston, TX	Post-Doc	01/96-07/98	Cardiovascular Toxicol.

B. Position and Honors**Positions**

1996-1998 National Institute of Environmental Health Sciences Toxicology Postdoctoral Fellow, University of Texas Medical Branch, Galveston, TX

1998-2002 Assistant Professor, Department of Biology, University of Wisconsin-Eau Claire, Eau Claire, WI

2003 Associate Professor, Department of Biology, University of Wisconsin-Eau Claire, Eau Claire, WI

2003-2009 Assistant Professor, Division of Cardiovascular Medicine, Department of Medicine, University of Louisville, Louisville, KY

2006-2014 Study Section Peer Reviewer, American Heart Association Great Rivers Affiliate (formerly Ohio Valley Affiliate); Vascular and Endothelium Biology 2: Co-Chair, 2011-2012; Chair, 2013-2014

2009 NIEHS R13 Study Section Ad Hoc Peer Reviewer

2009-present Director, Inhalation Facility, Division of Cardiovascular Medicine, Department of Medicine, University of Louisville, Louisville, KY

2009-2015 Associate Professor, Division of Cardiovascular Medicine, Department of Medicine, University of Louisville, Louisville, KY

2010-14 Councilor, Vice-President, President Cardiovascular Toxicology Specialty Section, Society of Toxicology

2011-present Director, Animal Core, Diabetes and Obesity Center, University of Louisville, Louisville, KY

2012-13 NIEHS Cardiovascular and Sleep Epidemiology (CASE) Study Section Peer Review (*ad hoc*)

2012-2016 Associate Editor, *Cardiovascular Toxicology*

2012-present Associate Editor, *Toxicology and Applied Pharmacology*

2013, 2015 NIEHS Systemic Injury of Environmental Exposure (SIEE) Study Section Peer Review (*ad hoc*)

2013-present Director, Tobacco Product Evaluation and Exposure Core, American Heart Association – Tobacco Regulation and Addiction Center (A-TRAC), University of Louisville, Louisville, KY

2014 Search Committee, Chair, Diabetes and Obesity Center, University of Louisville, Louisville, KY

2014 NIEHS Special Emphasis Panel (SEP) Study Section Peer Review (*ad hoc*)

2015 NIH Cancer, Cardiovascular and Sleep Epidemiology Study Section Peer Review (*ad hoc*)

2015-2017 Councilor, Inhalation and Respiratory Specialty Section, Society of Toxicology

2015 Professor, Division of Cardiovascular Medicine, Department of Medicine, University of Louisville, Louisville, KY

2015-present Editorial Board, *Circulation Research*

2016-2018 NIEHS Systemic Injury of Environmental Exposure (SIEE) Study Section Peer Review (permanent member)

Honors

1996 Eli J. and Helen Shaheen Graduate School Award In Science, University of Notre Dame
1998 National Research Service Award (NRSA), National Institutes of Environmental Health Sciences, Postdoctoral Fellowship, University of Texas Medical Branch, Galveston, TX
2003 Academic Research Enhancement Award (R15), National Institutes of Environmental Health Sciences, University of Wisconsin-Eau Claire, Eau Claire, WI
2011 "Top Reviewer" -- Toxicology and Applied Pharmacology
2014 Service Award, Cardiovascular Toxicology Specialty Section, Society of Toxicology

C. Contributions to Science (bolded authors indicate trainees)

1. *Comparative Physiology of Vascular Regulation*: Initially, I focused my research on mechanisms of vascular control in non-mammalian vertebrate models primarily fish because fish are the most diverse vertebrate class (>30,000 species), and thus, mechanisms of vaso-control (and ones operative in terrestrial vertebrates) likely evolved in fish. We assessed numerous mechanisms in a variety of species under physiological conditions, and identified vascular responses to classical mammalian vasoactive agents including angiotensin II (a.), arginine vasotocin (AVP in mammals; b.), atrial natriuretic peptide (ANP; c.), and localized vascular acclimation in snakes (spun head-in over weeks in a centrifuge at NASA; d.). Collectively, these data provide an evolutionary context for extant mammalian vaso-control mechanisms.

- a. Conklin, D.J. and K.R. Olson. 1994. Angiotensin II relaxation of rainbow trout vessels *in vitro*. *Am. J. Physiol.* 266:R1856-R1860. PMID: 8024039.
- b. Conklin, D.J., **N.W. Mick** and K.R. Olson. 1996. Arginine vasotocin relaxation of gar (*Lepisosteus spp.*) hepatic vein *in vitro*. *Gen. Comp. Endo.* 104:52-60. PMID: 8921355.
- c. Olson, K.R., D.J. Conklin, A.P. Farrell, J.E. Keen, Y. Takei, **L. Weaver, Jr.**, M.P. Smith and Y. Zhang. 1997. Effects of natriuretic peptides and nitroprusside on venous function in trout. *Am. J. Physiol.* 273:R527-R539. PMID: 9277535.
- d. Conklin, D.J., H.B. Lillywhite, K.R. Olson, R. Ballard and A.R. Hargens. 1996. Blood vessel adaptation to gravity in a semi-arboreal snake. *J. Comp. Physiol. B.* 196(7):425-432. PMID: 8617890.

2. *Role of Amine Metabolism in Vascular Control and Pathology*: Several primary amines stimulate vascular relaxation via a vascular-rich semicarbazide-sensitive amine oxidase (SSAO) enzyme that generates equal molar quantities of an amine-specific aldehyde, H₂O₂ and ammonia (NH₃). The enigmatic SSAO is highly expressed in the vasculature and in the presence of allylamine, an exogenous amine, there is a rapid onset of irreversible vasospasm in aorta (a.) and coronary arteries (b.). These studies highlight the potent dual action of amine metabolism mediated by vascular wall SSAO (c.), and in particular revealed that acrolein is a potent vascular toxin capable of inducing vasospasm in isolated human blood vessels (d.) – a highly relevant clinical cause of acute myocardial infarction and coronary artery bypass graft (CABG) failure.

- a. Conklin, D.J. and P.J. Boor. 1998. Allylamine cardiovascular toxicity: evidence for aberrant vasoreactivity. *Tox. Appl. Pharm.* 148(2):245-251. PMID: 9473532.
- b. Conklin, D.J., **C.L. Boyce**, M.B. Trent and P.J. Boor. 2001. Amine metabolism: a novel path to coronary artery vasospasm. *Tox. Appl. Pharm.* 175(2):149-159. PMID: 11543647.
- c. Conklin, D.J., **H.R. Cowley**, R.J. Wiechmann, G.H. Johnson, M.B. Trent and P.J. Boor. 2004. Vasoactive effects of methylamine in isolated human blood vessels: Role of semicarbazide-sensitive amine oxidase, formaldehyde, and hydrogen peroxide. *Am.J. Physiol. Heart Circ. Phys.* 286:H667-H676. PMID: 14715500.
- d. Conklin, D.J., A. Bhatnagar, **H. Cowley**, G.H. Johnson, R.J. Wiechmann, L.M. Sayre, M.B. Trent and P.J. Boor. 2006. Acrolein stimulates hypercontraction in isolated human blood vessels. *Tox. Appl. Pharm.* 217(3):277-88. PMID: 17095030; PMCID: PMC3487162.

3. *Environmental Cardiology*: Since 1993 and the publication of the 'Harvard Six Cities Study', the mechanism by which air pollution increases mortality and enhances cardiovascular disease risk has been an intriguing question. Upon joining Dr. Aruni Bhatnagar in 2003 at the Univ. of Louisville, my research has specifically addressed this larger question by focusing on particulate matter (a.) and aldehydes (b.) present in the environment. Uniquely, our group has identified novel and sensitive cardiovascular-specific biomarkers of

air pollution exposure in mice and humans. To this end, we were the first to show that elevated ambient air pollution decreased the level of circulating stem cells (i.e., endothelial progenitor cells, EPCs), important for cardiovascular health, in both young healthy humans (a.) and mice (a., b.). Moreover, we demonstrated that the mechanisms underlying this effect was a systemic 'VEGF resistance' (c.), and that a similar response is triggered by low-level exposure to acrolein (d.) indicating that air pollution at levels encountered in the real world impacts the physiological regulation of circulating stem cells perhaps depressing vascular repair, and thereby, enhancing cardiovascular disease risk.

- a. O'Toole, T.E., Hellmann, J., **Wheat, L.**, Haberzettl, P., **Lee, J.**, *Conklin, D.J.*, Bhatnagar, A. and Pope, C.A., 3rd. 2010. Episodic exposure to fine particulate air pollution decreases circulating levels of endothelial progenitor cells. *Circ. Res.* 107(2):200-203. PMID: 20595651; PMCID: PMC2943671.
 - b. *Conklin, D.J.*, Barski, O.A., **Lesgards, J-F.**, Juvan, P., Rezen, T., Rozman, D., Prough, R.A., Vladykovskaya, E., Liu, SQ., Srivastava, S. and A. Bhatnagar. 2010. Acrolein consumption induces systemic dyslipidemia and lipoprotein modification. *Toxicol. Appl. Pharm.* 243(1):1-12. PMID: 20034506; PMCID: PMC2922677.
 - c. **Wheat, L.A.**, Haberzettl, P., Hellmann, J., Baba, S.P., **Bertke, M.**, **Lee, J.**, McCracken, J., O'Toole, T.E., Bhatnagar, A. and *Conklin, D.J.* 2011. Acrolein inhalation prevents vascular endothelial growth factor-induced mobilization of Flk-1⁺/Sca-1⁺ cells in mice. *Arterioscler. Thromb. Vasc. Biol.* 31(7):1598-1606. PMID: 21415405; PMCID: PMC3182098.
 - d. Haberzettl, P., **Lee, J.**, **Duggineni, D.**, McCracken, J., Bolanowski, D., O'Toole, T.E., Bhatnagar, A. and *D. J. Conklin.* 2012. Exposure to ambient air fine particulate matter prevents VEGF-Induced mobilization of endothelial progenitor cells from the bone marrow. *Environ. Health Pers.* 120(6):848-856. PMID: 22418586; PMCID: PMC3385427.
4. *Role of Aldehyde Metabolism in Cardiovascular Toxicity and Disease Pathogenesis:* Over the last decade, we have documented the relatively high sensitivity of cardiovascular targets to exogenous and endogenous aldehyde exposure, especially responses to acrolein. Moreover, we have explored the metabolic basis of this exquisite cardiovascular sensitivity to aldehyde-induced injury by focusing on the specific contribution that aldehyde metabolism enzymes (aldose reductase, aldehyde dehydrogenase, GST, P450s, a.) makes to acute target organ injury and disease pathogenesis. For example, we study aldehyde-metabolizing enzymes that mediate GSH conjugation of unsaturated aldehydes, such as glutathione S-transferase P (GSTP). GSTP modulates aldehyde toxicity under environmentally- and clinically-relevant exposure conditions (i.e., inhaled acrolein, b. tobacco smoke, b.; CY, c.; ischemia-reperfusion, d.), thus, exemplifying the practical strength of our concept and its theoretical underpinnings. Moreover, we have in parallel showed in our animal models that these aldehyde metabolizing enzymes are also protective against the progression toward diabetes, which has important implications for this emerging epidemic.
- a. **Amunom, I.**, Stephens, L.J., Tamasi, V., *Conklin, D.J.*, Bhatnagar, A., Srivastava, S., Martin, M.V., Guengerich, F.P. and R.A. Prough. 2007. Cytochromes P450 catalyze oxidation of α,β -unsaturated aldehydes. *Archives of Biochemistry & Biophysics.* 464:187-96. PMID: 17599801; PMCID: PMC1994811.
 - b. *Conklin, D.J.*, Haberzettl, P., Prough, R.A. and A. Bhatnagar. 2009. Glutathione S-transferase P protects against endothelial dysfunction induced by exposure to tobacco smoke. *Am. J. Physiol. Heart Circ. Phys.* 296(5):H1586-97. PMID: 19270193; PMCID: PMC2685347.
 - c. *Conklin, D.J.*, Haberzettl, P., **Lesgards, J-F.**, Prough, R.A., Srivastava, S. and A. Bhatnagar. 2009. Increased sensitivity of glutathione S-transferase P-null mice to cyclophosphamide-induced urinary bladder toxicity. *Jrl. Pharmacol. Expt. Therap.* 331(2):456-69. PMID: 19696094; PMCID: PMC2775270.
 - d. *Conklin, D.J.*, Y. Guo, P. Haberzettl, B.G. Hill, S.P. Baba, L. Guo, **G. Jagatheesan, K. Wetzberger, D.** Obal, D.G. Rokosh, R.A. Prough, S.D. Prabhu, M. Velayutham, J.L. Zweier, D. Hoetker, D.W. Riggs, S. Srivastava, R. Bolli, and A. Bhatnagar. 2015. Genetic deficiency of glutathione S-transferase P increases myocardial sensitivity to ischemia-reperfusion injury. *Circ. Res.* 117(5):437-49. doi: 10.1161/CIRCRESAHA.114.305518. PMID: 26169370. PMCID in process.

Public Bibliography URL:

<http://www.ncbi.nlm.nih.gov/sites/myncbi/daniel.conklin.1/bibliography/40915266/public/?sort=date&direction=ascending>.

D. Research Support

Ongoing

R01ES027881

Haberzettl (PI)

05/01/2017-04/30/2022

Air Pollution, Circadian Rhythm Disruption and Cardiometabolic Disease

The goal of this project is to test the novel hypothesis that air pollution exposure increases the risk of cardiovascular disease and diabetes via disruption of vascular circadian rhythm. This state of circadian dyssynchrony exacerbates cardiometabolic injury.

Role: Co-Investigator, **Conklin, D.J.**

R01 HL122676-01A1

Conklin (PI)

07/01/2015-04/30/2020

Cardiovascular Toxicity of Tobacco Products and Constituents

The goal of these studies is to identify biomarkers of cardiotoxicity in mice and humans associated with exposure to aldehydes present in conventional and electronic cigarettes.

FX-ATRAC-UL-01S

Conklin (PI)

09/01/2015-06/30/2017 (NCE)

Toxicity and Thermal Degradation of Flavors

(Supplement American Heart Association – Tobacco Regulation and Addiction Center; A-TRAC)

The goal of these studies is to identify toxicity in isolated human cardiovascular cells due to exposure to flavors and their thermal degradation products present in electronic cigarette aerosols.

R01 HL122581-01

Baba (PI)

04/01/2014-03/31/2019

Cardiac Pathophysiology of Histidyl Dipeptides

The goal of these studies is to identify the mechanism by which histidyl dipeptides such as carnosine act to protect against ischemia-reperfusion-induced cardiovascular injury in mice.

Role: Co-Investigator, **Conklin, D.J.**

R01 HL120746

Srivastava (PI)

10/01/2013-09/30/2018

Tobacco Products and Atherosclerotic Disease

The goal of these studies is to examine the effects of harmful and potentially harmful constituents (HPHC) of tobacco on atherosclerosis.

Role: Co-Investigator, **Conklin, D.J.**

P20 GM103492-07

Bhatnagar (PI)

09/01/2013-08/31/2018

Diabetes and Obesity Center -- Center of Biomedical Research Excellence (COBRE)

The goals of this center are to train the new generation of scientists, develop new models and pioneer new techniques to be used in obesity and diabetes research.

Role: Co-Investigator/Core Director, **Conklin, D.J.**

P50 HL120163-03

Bhatnagar and Robertson (co-PIs)

07/01/2013-06/30/2018

American Heart Association – Tobacco Regulation and Addiction Center (A-TRAC)

The goal of these studies is to identify biomarkers of cardiotoxicity associated with exposure to harmful and potentially harmful constituents (HPHC) of tobacco.

Role: Co-Investigator, **Conklin, D.J.**, Director, Tobacco Product Evaluation and Exposure Core (TPEE)

R01 ES019217-01A1

O'Toole (PI)

06/01/2011- 05/31/2017 (NCE)

Endothelial progenitor cells and particulate air pollution

In this study we characterize the quantitative and qualitative properties of stem cell populations in humans and mice exposed to particulate air pollution.

Role: Co-Investigator; **Conklin, D.J.**, Director, University of Louisville Inhalation Facility

Completed

R21HL120050-01A1

Li (PI)

05/01/2014-08/30/2016

MicroRNAs as Biomarkers for Tobacco Exposure and Heart Disease

The goal of these studies is to evaluate the utility of microRNAs in blood as biomarkers of tobacco exposure and tobacco-related cardiovascular injury in mice and humans.

Role: Co-Investigator, **Conklin, D.J.**

R21 ES024030

Conklin (PI)

09/01/2013-08/31/2015

Novel Treatments of Acrolein-induced Cardiotoxicity

The goal of these studies is to develop a post-exposure intervention to prevent acute cardiotoxicity of acrolein.

14ATRAC23640000

Conklin (PI)

09/01/2014-08/31/2015

AHA Tobacco Regulation and Addiction Center (ATRAC) Pilot Research Grants Program

Electronic Cigarette Aldehydes and Cardiovascular Toxicity

The goal of these studies is to identify biomarkers of cardiotoxicity associated with exposure to harmful and potentially harmful constituents (HPHC) of electronic cigarettes.

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: Heberle, Lauren

eRA COMMONS USER NAME (credential, e.g., agency login): LOHEBE01

POSITION TITLE: Associate Professor, Sociology and Director CEPM, University of Louisville

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
University of Chicago	BA	1989	Public Policy
Rutgers University	MA	1996	Sociology
Rutgers University	Ph.D.	2003	Sociology

B. Positions and Honors

Positions and Employment

UofL Center for Environmental Policy and Management

9/2000-12/2002 Research Associate, and Instructor
1/2003-1/2004 Post-Doctoral Fellow, and Instructor
1/2004- 6/2005 Assistant Director, and Instructor
7/2005-2013 Associate Director
2013-Present Director

EPA Region 4 @ UofL Environmental Finance Center

2005-2006 Co-Director
11/2006 – 2016 Director

- President of Environmental Finance Center Network 2012/2013

UofL Urban and Public Affairs

7/2006-6/2008 Assistant Professor (Term)

UofL Sociology Department

2008-2013 Assistant Professor
2013-Present Associate Professor

Federal Board/Committee Memberships

- Expert witness to U.S. EPA Environmental Finance Advisory Board, 2006-2016
- Network Member, U.S. EPA's Environmentally Responsible Redevelopment and Reuse Initiative (ER3). 2006 – 2015
- Team Member, U.S. Regional and Local Land Revitalization Planning Team (Part of Phase 4 of the EPA's US & German Bilateral Working Group on Redevelopment of Contaminated Sites). 2006 – 2015.

Other Professional Memberships

- American Sociological Association, 1995 – Present (Member of Environmental Tech section)
- Advisory member of the Center for Neighborhoods PAL Coalition, a project funded under the Office of National Drug Control Policy 2009-2012
- Urban Affairs Association, 2003 – 2008
- Committee Member, Urban Agriculture Steering Committee for the Jefferson County Extension Service
- Committee Member, Louisville Sustainability Council Workgroup Member

C. Contribution to Science

My research areas center on identifying and examining paths to community engagement in environmental decision making at a variety of levels – individual, local, state and federal. I began this work in the area of

brownfields redevelopment and broadened it to sustainable development and climate change planning. I have made substantive use of my Center's local research and technical assistance projects to inform my research agenda. My first major grant from the EPA Brownfields program was a project designed to increase community engagement in brownfields redevelopment in socio-economically challenged neighborhoods that ran from 2005 through 2009. This effort led to several other community engagement projects centered on brownfield redevelopment efforts in Louisville and has come full circle with a current EPA Brownfields research grant aimed at producing better methods for communities to project and document community benefits at a neighborhood level beyond those included in traditional economic development. This project will pilot the tools in a neighborhood in Louisville, KY over the next four years. The selected publications listed here demonstrate the trajectory of my research and the types of publications that typically result from technical assistance and applied social science research projects. A complete list of all my publications and technical reports can be found on my center website: <http://louisville.edu/cepm/staff-and-associates/lauren-heberle>.

Participatory Sustainable Development and Community Planning

- Heberle, L., Schilling, J., McReynolds, B., Sizemore, S. (2017). "HUD's Sustainable Communities Initiative: An Emerging Model of Place-Based Federal Policy and Collaborative Capacity Building." *Cityscape: A Journal of Policy Development and Research*. U.S. Department of Housing and Urban Development. Office of Policy Development and Research. Vol. 19. Number 3, pp. 9-37.
- Heberle, L., Merrill, S., Keeley, C. and Lloyd, S. (April 2014) "Local knowledge and participatory climate change planning in the northeastern U.S." in *International perspectives on climate change: Latin America and Beyond* (Editors Profs. Leal, Filho; Alves, Fátima; Caeiro, Sandra; and Azeiteiro; Ulisses). Springer. Cham Heidelberg, New York, Dordrecht, London, pp. 239-252.
- Heberle, L., Opp, S. Eds. (2008) *Local Sustainable Urban Development in a Globalized World*. Urban Planning and Environment Series, Ashgate Press, Hampshire, England.

Brownfields Policy

- Heberle, L. & Wernstedt, K. (2006) "Understanding Brownfields Regeneration in the US." *Local Environment: Special Edition Sustainability and Brownfields*. Vol. 11, No. 5, 479-497.
- Wernstedt, K., Meyer, P.B., Aberini, A., Heberle, L. (2006) "Incentives for Private Residential Brownfields Development in U.S. Urban Areas". *Journal of Environmental Planning and Management*. V49n1.pp. 101-119.

Climate Change Policy

- Heberle, L & Christensen, I., (2011) "US environmental governance and local climate change mitigation policies: California's story", *Management of Environmental Quality: An International Journal*, Vol. 22 Iss: 3, pp.317 – 329
- Meyer, P.B., Heberle, L. (2010) "Local Climate Change Initiatives in the United States: The Primacy of Short-Term Economic Returns." in *Local Governments and Climate Change: Sustainable Energy Planning and Implementation in Small and Medium Sized Communities*. van Staden, M. and Musco F (eds). *Advances in Global Change Research Volume #39*. Springer Dordrecht, Heidelberg, London, New York. Pp 181-190.
- Heberle, L (2008) "Local Strategies as Global Solutions" in *Local Sustainable Urban Development in a Globalized World*. Urban Planning and Environment Series, Ashgate Press, Hampshire, England. pp. 1-9.

D. Research Support

Ongoing Research Support

- NIH/NIEHS Superfund Research Center Community Engagement Core, 2017-2021, CEC Director. The CEC will support community and academic scientists/educators in transdisciplinary research that incorporates best practices in community engagement by facilitating beneficial and bidirectional interactions between residents, industry, policy makers, and Superfund Center investigators.
- U.S. Environmental Protection Agency, Brownfields Training, Research, and Technical Assistance Grant. (2014-2019). PI. Research grant sub-award through Virginia Tech, Metropolitan Institute. This project will develop methods and tools to be used by communities engaged in brownfields redevelopment to better measure community benefits at the neighborhood level beyond current traditional economic development models. It will develop models to systematically link publically available national data with local and "hyper-local" data using a web-based desktop portal and applications developed for hand-held electronic devices.

Completed in last three years

- U.S. Environmental Protection Agency, Training and Technical Assistance to Small Public Water Systems, 2012 to 2016. Co-PI. Sub-award via University of North Carolina, Chapel Hill. As part of the Environmental Finance Center Network (EFCN) our Center assists in this national technical assistance and training program.
- U.S. HUD Sustainable Communities Capacity Building Grant Phase II. 2013-2015. Co-PI Sub-award through Institute for Sustainable Communities.
- U.S. HUD Sustainable Communities Capacity Building Grant. 2011-2013 Phase I. PI. The U.S. HUD grants listed here provided technical assistance and capacity building to communities across the country engaged in regional and local planning efforts. Our team focused on assistance related to integrating water infrastructure, brownfields, local food system, and climate change planning into the regional and local planning efforts using an equity framework. As team leader for a group of four university based Environmental Finance Centers, I directed all activities.
- U.S. HUD "Market Analysis/Affordable Housing Needs Assessment." Co-PI. Sub-award through Louisville Metro Human Relations Commission, 2014-2015. This research report documented, through the use of focus groups and a descriptive demographic community profile, the challenges and obstacles to achieving safe, fair, and affordable housing faced by those in protected classes under the Fair Housing Act.
- U.S. Environmental Protection Agency, Area-wide Brownfields Planning Grant 2013-2016 PI. Sub-award via Louisville Metro Department of Economic Growth and Innovation. This grant funded community participation in an area-wide planning effort centered on sites located along a rail corridor in the Germantown/Shelby Park neighborhoods in Louisville, KY. We structured this effort around building community capacity related to neighborhood planning, brownfields, green infrastructure, and equitable development strategies.
- U.S. Center for Disease Control, Community Transformation Grant 2011 PI. Sub-award via Louisville Metro Public Health, 2012-2014. This project developed safe urban gardening guides translated into several languages, assisted local organizations in developing safe urban gardens and community gardening programs, produced a community garden guide for Louisville Metro Government, and hosted numerous community workshops across the Louisville area.
- U.S. Environmental Protection Agency, Environmental Finance Center Region 4, 2009-2015 PI. This is a technical assistance grant serving communities across EPA Region 4. Activities under this grant include the production of practice guides, technical reports, workshops, webinars, web based resources, and day-to-day one-on-one assistance to individuals and agencies across EPA Region 4.

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: Haberzettl, Petra

eRA COMMONS USER NAME (credential, e.g., agency login): POHABE02

POSITION TITLE: Assistant Professor of Medicine

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
Ruhr-University Bochum, Bochum, Germany	M.S.	02/2003	Biochemistry
Institute for Environmental Medicine, Heinrich-Heine University, Duesseldorf, Germany	Ph.D.	11/2006	Biology
University of Louisville, KY	Postdoctoral	03/2007-11/2011	Cardiovascular Toxicology

B. Position and Honors**Positions**

05/2003-03/2006	Scholar of the DFG International Graduate College, "Molecular Mechanisms of Food Toxicology", Institute for Environmental Medicine, Heinrich-Heine University, Duesseldorf, Germany
04/2006-11/2006	Research Assistant, Institute for Environmental Medicine, Heinrich-Heine University, Duesseldorf, Germany
03/2007-11/2011	Post-Doctoral fellow, Division of Cardiovascular Medicine, School of Medicine, University of Louisville, KY
12/2011-present	Assistant Professor, Division of Cardiovascular Medicine, School of Medicine, Center of Excellence in Diabetes and Obesity Research, University of Louisville, KY

Other Experience and Professional Memberships

2008-present	American Heart Association (AHA)
2009-present	Society of Toxicology (SOT)
2018-present	Society for Research on Biological Rhythms (SRBR)
05/2015-04/2017	Councilor of the Cardiovascular Toxicology Specialty Section (CVTSS) of the SOT
05/2018-04/2020	Councilor of the Stem Cell Specialty Section (SCSS) of the SOT

Honors

05/03-03/06	Scholar of the German Research Society (Deutsche Forschungsgesellschaft, DFG) International Graduate College, "Molecular Mechanisms of Food Toxicology", Institute of Environmental Medical Research (IUF), Heinrich-Heine University Duesseldorf, Duesseldorf, NRW, Germany
10/2005	<i>New Investigator Award</i> at the Conference of "Mechanisms of Action of Inhaled Fibers, Particles, and Nanoparticles in Lung and Cardiovascular Diseases", North Carolina
03/2011	<i>Postdoctoral Fellow Travel Award</i> , Cardiovascular Toxicology Specialty Section at the Meeting of the Society of Toxicology, Washington, DC
03/2013	<i>Best Postdoctoral Publication Award</i> , Society of Toxicology, San Antonio, TX

C. Contributions to Science

1. *Pulmonary effects of respirable particles*: The induction of pulmonary oxidative stress and inflammation is a key event in the development of particle induced lung disease such as fibrosis and cancer. Because, it has been indicated that particle clearance by alveolar macrophages plays a critical role in the pulmonary particle toxicity the goal of my doctoral thesis was to determine the mechanism of particle uptake and its consequences on pro-oxidant and pro-inflammatory responses. Results of these studies showed that the uptake of fine silica particles (DQ12) by alveolar macrophages is an actin dependent phagocytotic process that induces oxidative stress and inflammation. I also demonstrated by flow cytometry and by a unique investigation of the signaling cascade involved in receptor mediated phagocytosis that the uptake of DQ12 is mainly mediated by the FcγII-receptor. Activation of the FcγII-receptor in alveolar macrophages induced oxidative stress and inflammation that could contribute to the pulmonary toxicity of respirable particles.
 - a. Albrecht C, Knaapen AM, Becker A, Höhr D, **Haberzettl P**, van Schooten FJ, Borm PJ, Schins RP. The crucial role of particle surface reactivity in respirable quartz-induced reactive oxygen/nitrogen species formation and APE/Ref-1 induction in rat lung. *Respir Res.* 2005, 2; 6: 129; PMID: [16266428](#); PMCID: [PMC1291407](#)
 - b. **Haberzettl P**, Duffin R, Krämer U, Höhr D, Schins RP, Borm PJ, Albrecht C. Actin plays a crucial role in the phagocytosis and biological response to respirable quartz particles in macrophages. *Arch Toxicol.* 2007, 81(7): 459-70; PMID: [17375287](#)
 - c. **Haberzettl P**, Schins RPF, Höhr D, Wilhelmi V, Borm PJA, Albrecht C. Impact of the FcγII-receptor on quartz uptake and inflammatory response by alveolar macrophages. *Am J Physiol Lung Cell Mol Physiol.* 2008, 294: 1137-1148; PMID: [18390832](#)
 - d. Scherbart AM, Langer J, Bushmelev A, van Berlo D, **Haberzettl P**, van Schooten FJ, Schmidt AM, Rose CR, Schins RP, Albrecht C. Contrasting macrophage activation by fine and ultrafine titanium dioxide particles is associated with different uptake mechanisms. *Part Fibre Toxicol.* 2011, 8:31, PMID: [21995556](#); PMCID: [PMC3214143](#)
2. *Vascular effects of environmental air pollution*: Exposure to fine particulate matter (PM_{2.5}) air pollution is associated with an increased risk for cardiovascular disease (CVD). To investigate the mechanistic pathways involved in the induction of vascular injury by the inhalation of fine particulate matter (PM_{2.5}), we started inhalation exposure studies investigating the effects of inhaled concentrated PM_{2.5} on signaling pathways involved in endothelial progenitor cell (EPC) mobilization. Results of these studies showed for the first time that short-term inhalation of PM_{2.5} decreased circulating EPC levels in human and mice. Our data also demonstrated that exposure to PM_{2.5} impairs the VEGF-mediated mobilization of EPCs and that improving insulin sensitivity or preventing pulmonary oxidative stress could restore EPC homeostasis in mice. Preventing pulmonary oxidative stress by lung-specific overexpression of the antioxidant enzyme extracellular superoxide dismutase (ecSOD-Tg) also improved the vascular signaling response to insulin and VEGF as well as PM_{2.5}-induced EPC dysfunction. These results show that EPCs are early and sensitive targets of air pollution and indicate that PM_{2.5} by inducing insulin and VEGF resistance impairs the recruitment of EPCs. Because EPCs are implicated in vascular repair, oxidative stress dependent depletion of circulating EPCs and impairment of EPC function by air pollution exposure is likely to impair vascular repair and thus, increases vascular dysfunction and the risk for the development of cardiovascular disorders.
 - a. O'Toole TE, Hellmann J, Wheat L, **Haberzettl P**, Lee J, Conklin DJ, *Bhatnagar A*, Pope CA 3rd. Episodic Exposure to Fine Particulate Air Pollution Decreases Circulating Levels of Endothelial Progenitor Cells. *Circ Res.* 2010, 107(2):200-3; PMID: [20595651](#); PMCID: [PMC2943671](#)
 - b. **Haberzettl P**, Lee J, Duggineni D, McCracken J, Bolanowski D, O'Toole TE, *Bhatnagar A* and Conklin DJ. Exposure to ambient air fine particulate matter prevents VEGF-Induced mobilization of endothelial progenitor cells from the bone marrow. *Environ. Health Pers.* 2012, 120(6):848-856; PMID: [22418586](#); PMCID: [PMC3385427](#)
 - c. **Haberzettl P***, McCracken JP, *Bhatnagar A* and Conklin DJ. Insulin sensitizers prevent fine particulate matter-induced vascular insulin resistance and changes in endothelial progenitor cell

homeostasis. *Am. J. Physiol. Heart Circ. Physiol.*, 2016, 310(11):H1423-38; PMID: [27016579](#); PMCID: [PMC4971897](#) *corresponding author

- d. **Haberzettl P**, Conklin DJ, Abplanalp WT, Bhatnagar A, O'Toole TE. Inhalation of Fine Particulate Matter Impairs Endothelial Progenitor Cell Function Via Pulmonary Oxidative Stress. *Arterioscler Thromb Vasc Biol.* 2018, 38(1):131-142; PMID: [29191925](#); PMCID: [PMC5746456](#)
3. *Cardiometabolic effects of environmental air pollution*: Recent reports suggest that urbanization, accompanied by migration to more polluted areas (with light and air pollution) and our modern 24 h lifestyle (35% adults sleep less than the recommended 7-8 h, eating an unhealthy diet) are significant factors fueling the world-wide increase in CVD and type-2 diabetes (T2D). To examine the mechanisms contributing to the development of cardiometabolic disorders after air pollution exposure we examined vascular inflammation and insulin resistance indicated to contribute to diet-induced systemic insulin resistance. Results of this work uniquely demonstrated that acrolein and PM_{2.5} exposure induces early vascular insulin resistance. The diet-independent induction of vascular insulin resistance by PM_{2.5} occurred in the absence of other metabolic defects and was accompanied by vascular activation of the NF-κBα- and inflammasome pathway. Exposure to concentrated PM_{2.5} exacerbated diet-induced systemic insulin resistance and impaired insulin-induced eNOS phosphorylation in the skeletal muscle. Impeding PM_{2.5}-induced pulmonary oxidative stress or treatment with an insulin sensitizer preserves vascular insulin sensitivity and prevented vascular inflammation. These results support the idea that by inducing pulmonary oxidative stress PM_{2.5} exposure causes vascular inflammation and insulin resistance that could be an important and novel mechanism to accelerate the progression to T2D and CVD.
 - a. O'Toole TE, Abplanalp W, Li X, Cooper N, Conklin DJ, **Haberzettl P**, Bhatnagar A. *Acrolein decreases endothelial cell migration and insulin sensitivity through induction of Let-7a*. *Toxicol Sci*, 2014, 40(2): 271-83, PMID: [24812010](#), PMCID: [PMC4176051](#)
 - b. **Haberzettl P***, McCracken JP, *Bhatnagar A* and Conklin DJ. Insulin sensitizers prevent fine particulate matter-induced vascular insulin resistance and changes in endothelial progenitor cell homeostasis. *Am. J. Physiol. Heart Circ. Physiol.* 2016, 310(11):H1423-38; PMID: [27016579](#); PMCID: [PMC4971897](#) *corresponding author
 - c. **Haberzettl P***, *O'Toole TE*, *Bhatnagar A* and Conklin DJ. Exposure to fine particulate air pollution causes vascular insulin resistance by inducing pulmonary oxidative stress. *Environ Health Perspect.* 2016, in press, PMID: [27128347](#) *corresponding author
4. *Vascular Effects of Aldehydes*: My initial postdoctoral research and collaborative projects gave me the chance to work on different aspects of vascular effects induced by aldehydes. In these studies I was able to show that acrolein activates endothelial cells by the induction of the unfolded protein response (UPR) and endoplasmic reticulum stress and decreases the number of circulating EPCs by impairing their mobilization from the bone marrow. We also demonstrated that unsaturated lipid peroxidation-derived aldehydes such as 4-hydroxy-trans-2-nonenal (HNE) or 1-palmitoyl-2-oxovaleroyl phosphatidyl choline (POVPC) induce autophagy and ER-stress in vascular smooth muscle and endothelial cells. These processes could be imported contributors to the pathology of atherosclerosis.
 - a. **Haberzettl P**, Vladykovskaya E, Srivastava S, *Bhatnagar A*. Role of endoplasmic reticulum stress in acrolein-induced endothelial activation. *Toxicol Appl Pharmacol.* 2009, 1; 234(1):14-24; PMID: [18951912](#); PMCID: [PMC2936103](#)
 - b. Wheat LA*, **Haberzettl P***, Hellmann J*, Baba SP, Bertke M, Lee J, McCracken J, *O'Toole TE*, *Bhatnagar A* and Conklin DJ. Acrolein inhalation prevents vascular endothelial growth factor-induced mobilization of Flk-1⁺/Sca-1⁺ cells in mice. *Arterioscler. Thromb. Vasc. Biol.* 2011, 31(7):1598-1606; PMID: [21527748](#); PMCID: [PMC3182098](#); *Authors contributed equally to this manuscript.
 - c. Vladykovskaya E, Sithu SD, **Haberzettl P**, Wickramasinghe NS, Merchant ML, *Hill BG*, McCracken J, Agarwal A, Dougherty S, Gordon SA, Schuschke DA, Barski OA, *O'Toole T*, D'Souza SE, *Bhatnagar A*, Srivastava S. Lipid peroxidation product 4-hydroxy-trans-2-nonenal causes endothelial activation by inducing endoplasmic reticulum stress. *J Biol Chem.* 2012, 30;287(14):11398-409, PMID: [22228760](#); PMCID: [PMC3322871](#)
 - d. **Haberzettl P**, Hill BG. Oxidized lipids activate autophagy in a JNK-dependent manner by stimulating the endoplasmic reticulum stress response. *Redox Biology* 2013, 1:56-64; PMID: [24024137](#); PMCID: [PMC3757667](#)

1. *Cardiac Effects of Aldehydes*: Other collaborative projects gave me the opportunity to work on cardiac effects of aldehydes. Due to the involvement in studies examining aldehyde-induced cardiac injury I was able to extend my knowledge in aspects of enzyme metabolism and heart diseases. These studies have demonstrated that aldehyde metabolism enzymes (aldose reductase, AR and glutathione S-transferase P, GST) contribute to the cardiac sensitivity to endogenous and exogenous aldehyde exposure, and reveal promising therapeutic targets in cardiovascular disease.
 - a. Keith RJ, **Haberzettl P**, Vladykovskaya E, Hill BG, Kaiserova K, Srivastava S, Barski O, Bhatnagar A. Aldose reductase decreases endoplasmic reticulum stress in ischemic hearts. *Chem Biol Interact.* 2009, 16;178(1-3):242-9; PMID: [19041636](#); PMCID: [PMC3178409](#)
 - b. Ismahil MA, Hamid T, **Haberzettl P**, Gu Y, Chandrasekar B, Srivastava S, Bhatnagar A, Prabhu SD. Chronic oral exposure to the aldehyde pollutant acrolein induces dilated cardiomyopathy. *Am J Physiol Heart Circ Physiol.* 2011, 301(5):H2050-60; PMID: [21908791](#); PMCID: [PMC3213964](#)
 - c. Conklin DJ, **Haberzettl P**, Jagatheesan G, Baba S, Merchant ML, Prough RA, Williams JD, Prabhu SD, Bhatnagar A. Glutathione S-transferase P protects against cyclophosphamide-induced cardiotoxicity in mice. *Toxicol Appl Pharmacol.* 2015, 285(2): 136-48; PMID: [25868843](#); PMCID: [PMC4430434](#)
 - d. Conklin DJ, Guo Y, Jagatheesan G, Klifoil PJ, **Haberzettl P**, Hill BG, Baba SP, Guo L, Wetzelberger K, Obal D, Rokosh G, Prough RA, Prabhu SD, Velayutham M, Zweier JL, Hoetker JD, Riggs D, Srivastava S, Bolli R, Bhatnagar A. Genetic Deficiency of Glutathione S-Transferase P Increases Myocardial Sensitivity to Ischemia-Reperfusion Injury. *Circ Res.* 2015, 117(5):437-49; PMID: [26169370](#); PMCID: [PMC4854443](#)

The complete list of peer reviewed journal articles can be found in MyBibliography:

<http://www.ncbi.nlm.nih.gov/sites/myncbi/1zSQo7kOXOaAB/bibliography/48972106/public/?sort=date&direction=ascending>

D. Research Support

Ongoing Research Support

R01ES027881

Haberzettl, P. (PI)

05/2017-04/2022

The goal of this project is to test the novel hypothesis that air pollution exposure by disrupting vascular circadian rhythm exacerbates cardiometabolic injury in the new susceptibility state of circadian dyssynchrony. Results of this project will be of significance in understanding and preventing the adverse cardiometabolic effects originated from air pollution exposure.

2R01 ES019217-06

PI: O'Toole, Timothy, Co-I: Haberzettl, P.

05/2018-04/2023

This project will identify individual risk factors to determine cardiovascular susceptibility to PM_{2.5} and will test whether in the presence of cardiovascular risk factors exposure to PM_{2.5} induces vascular dysfunction that could be prevented by the treatment with carnosine. Results of this project will provide a better estimate of CVD burden due to PM_{2.5} exposures, identify specific risk factors that increase PM_{2.5} susceptibility and assess the efficiency of carnosine to attenuate PM_{2.5} toxicity.

Completed Research Support

P20GM103492 (COBRE)

PI: Bhatnagar, Junior Faculty, Project 2: Haberzettl, P. 12/2011-05/2017

The aim of this project is to test whether exposure to PM_{2.5} induces vascular inflammation and insulin resistance and impairs the repair capability of endothelial progenitor cells (EPCs) that lead to the progression of T2D and CVD. Completion of these studies will enable us to determine the specific molecular and cellular pathways that mediate the effects of PM_{2.5} on insulin resistance in diet-induced obesity.

AHA 12SDG9380000

Haberzettl, P. (PI)

01/2012-12/2015

The objective of this study is to test whether diet-induced obesity by inducing vascular insulin and VEGF resistance impairs endothelial progenitor cells (EPCs) mobilization and function leading to chronic deficits in vascular maintenance and repair. Results of this project will advance our basic understanding how obesity increases the risk for cardiovascular disease and may lead to the development of new diagnostic and therapeutic strategies for the treatment and management of vascular complications in obesity.

PENDING

1R01 ES028268-01A1 (PI's: Haberzettl, Petra and Hill, Bradford)

07/01/2018-06/30/2023

The goal of this project is to investigate whether PM_{2.5} exposure by inducing metabolic dysfunction impairs the capability of endothelial progenitor cells (EPCs) to promote vascular repair and maintenance. This new and innovative assessment of PM_{2.5} toxicity will lead to a novel understanding of the adverse cardiovascular effects of PM_{2.5} as well as move the field of stem/progenitor cell biology forward.

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: **Carll, Alex Perrow**

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

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
Duke University	A.B.	05/2004	Envir. Science & Policy
University of North Carolina, Chapel Hill	M.S.P.H.	05/2008	Public Health
University of North Carolina, Chapel Hill	Ph.D.	12/2012	Environmental Health
Harvard T.H. Chan School of Public Health	Post-Doc	10/2015	Environmental Health

B. Positions and Honors**Positions and Employment**

- 2004-05 Behavioral Therapist, CNC/Access, Inc., Boone, NC and Burlington, NC
 2005-06 Student Contractor, Environmental Toxicology Division, U.S. Environmental Protection Agency, Research Triangle Park, NC
 2015- Assistant Professor, Institute of Molecular Cardiology, University of Louisville, Louisville, KY

Honors

- 2006-12 Cooperative Fellow, Environmental Public Health Division, U.S. EPA, Research Triangle Park, NC
 2011 Graduate Student Travel Award, Cardiovascular Toxicology Specialty Section, Society of Toxicology (SOT) Annual Meeting
 2011 First Place Pre-Doctoral Trainee Poster Presentation, Research Triangle Visiting Pulmonary Scholar Series Annual Seminar
 2012 Student Travel Award, SOT Annual Meeting
 2012 Mary O. Amdur Award for Inhalation Toxicology, SOT Annual Meeting
 2013 Paper of the Year¹: Inhalation & Respiratory Toxicology, SOT Annual Meeting
 2014 Impact Paper of the Year²: Cardiovascular Toxicology, SOT Annual Meeting
 2015 Harvard Chan Post-Doctoral Association Travel Award
 2017 American Heart Association Tobacco Regulation & Addiction Center Fellowship

Other Experience and Professional Memberships

- 2006- Society of Toxicology, member
 2010- Sigma Xi, member
 2012 Chair, Cardiovascular Toxicity of Nanoparticles Platform Session, SOT Annual Meeting
 2012-13 Post-Doctoral Representative, Cardiovascular Toxicology Specialty Section, SOT
 2013- Delta Omega, Public Health Honor Society
 2015 American Thoracic Society, member
 2013-15 Grants and Research Strategy Chair, Harvard School of Public Health Post-Doctoral Association

C. Contributions to Science

1. My early research focused primarily on developing a reproducible rat model of cardiomyopathy to incorporate into air pollution exposure studies. Rodent models of heart failure are commonly complicated by technically difficult surgeries that often cause premature mortality and a variable phenotype while also precluding additional surgeries such as telemetry implantation. Yet epidemiologic studies indicate a

particularly strong link between air pollution exposure and cardiovascular morbidity and mortality among humans with preexisting heart failure. To facilitate toxicological investigations of the biological plausibility of these epidemiologic findings and their potential mechanisms, I developed rat models of noninvasively-induced cardiomyopathy using pharmacologic and dietary interventions. These models were particularly useful for air pollution investigations involving radiotelemetry, as demonstrated in my subsequent research.

- a. Carll AP, Haykal-Coates N, Winsett DW, Rowan WH 3rd, Hazari MS, Ledbetter AD, Nyska A, Cascio WE, Watkinson WP, Costa DL, Farraj AK. 2010. Particulate matter inhalation exacerbates cardiopulmonary injury in a rat model of isoproterenol-induced cardiomyopathy. *Inhal Toxicol* 22(5):355-68. PMID: 20121584.
 - b. Carll AP, Haykal-Coates N, Winsett DW, Hazari MS, Nyska A, Richards JH, Willis MS, Costa DL, Farraj AK. 2011. Dietary salt exacerbates isoproterenol-induced cardiomyopathy in rats. *Toxicol. Pathol.* 39(6):925-37. PMID: 21878552.
 - c. Carll AP, Willis MS, Lust RM, Costa DL, Farraj AK. 2011. Merits of Non-Invasive Rat Models of Left Ventricular Heart Failure. *Cardiovasc Toxicol.* 11(2):91-112. PMID: 21279739.
2. I applied rodent models of cardiomyopathy to reveal that underlying cardiac disease confers susceptibility to acute inhalation exposure to air pollutants. By applying a novel model of isoproterenol-induced cardiomyopathy in rats genetically predisposed to heart failure, I demonstrated that particulate matter (PM) inhalation transiently increases atrioventricular block arrhythmia and parasympathetic regulation of cardiovascular physiology solely in cardiomyopathic rats and not in their healthy counterparts. Similarly, I found that acutely inhaled diesel exhaust (DE) transiently increases parasympathetic influence, arrhythmia, LV diastolic volume, and heterogeneity of ventricular repolarization in heart failure-prone rats, with age imparting greater sensitivity. Additionally, I determined that DE filtered free of PM also similarly causes arrhythmia, parasympathetic dominance, and repolarization defects.
- a. **Carll AP**, Haykal-Coates N, Winsett DW, Hazari MS, Ledbetter AD, Richards JH, Cascio WE, Costa DL, Farraj AK. 2015. Cardiomyopathy confers susceptibility to particulate matter-induced oxidative stress, vagal dominance, arrhythmia, and pulmonary inflammation in heart failure-prone rats. *Inhal Toxicol* 27(2):100-12. PMID: 25600220.
 - b. **Carll AP**, Hazari MS, Perez CM, Krantz QT, King C, Winsett DW, Costa DL, Farraj AK. 2012. Whole and Particle-Free Diesel Exhausts Differentially Affect Cardiac Electrophysiology, Blood pressure, and Autonomic Balance in Heart Failure-Prone Rats. *Toxicol. Sci.* 128(2):490-9. PMID: 22543275.
 - c. **Carll AP**, Lust RM, Hazari MS, Perez CM, Krantz QT, King C, Winsett DW, Cascio WE, Costa DL, Farraj AK. 2013. Diesel Exhaust Inhalation Increases Cardiac Output, Bradyarrhythmias, and Parasympathetic Tone in Aged Heart Failure-Prone Rats. *Toxicol. Sci.* 131(2):583-95. PMID: 23047911.
3. At the outset of my subsequent research, it remained unclear how air pollution exposure might exacerbate heart failure. I thus tested the hypothesis that air pollution exposure impairs cardiac performance through ANS dysfunction. To address this I applied treadmill exercise stress tests, autonomic antagonists, and surgical vagotomy in heart failure-prone rats exposed acutely to DE. Treadmill exercise indicated DE exposure caused time-dependent changes in autonomic imbalance and depression in contractility, with likely sympathetic nervous system mediation. Measures of LV pressure and vagotomy suggested DE impaired cardiac performance and autonomic balance via sympathetic dominance, likely originating from impaired vagal function. This information elucidated a mechanism underlying air pollutant-induced acute exacerbation of heart failure and offers biological plausibility to epidemiological observations, thereby guiding health assessments and air pollution regulations. I followed these studies by continuously measuring LV pressure via telemetry in normal rats exposed subchronically to vehicular-derived PM. These findings indicated repeat PM exposure diminished LV performance and impaired baroreflexes while also enhancing sympathetic and arrhythmic responses to a psychosocial stress test.
- a. ***Carll AP**, Hazari MS, Perez CM, Krantz QT, King C, Haykal-Coates N, Cascio WE, Costa DL, Farraj AK 2013. An Autonomic Link between Inhaled Diesel Exhaust and Impaired Cardiac Performance: Insight from Treadmill and Dobutamine Challenges in Heart Failure-Prone Rats. *Toxicol. Sci.* 135(2):425-36. PMID: 23872579.
4. More recently I examined effects of repeated exposure to traffic-derived PM at ambient levels in a rat model of dietary fructose-induced metabolic syndrome. These low PM levels diminished baroreflex sensitivity and

caused sympathetic dominance and arrhythmia in metabolic syndrome rats, whereas normal healthy rats showed no such responses. These findings validate limited epidemiologic evidence that metabolic syndrome increases susceptibility to the adverse cardiovascular effects of PM, potentially through ANS imbalance.

- a. ***Carli AP**, Crespo SM, Zati DH, Filho MS, Zati DH, Coull BA, Diaz EA, Raimundo RD, Jaeger TNG, Ricci-Vitor AL, Papapostolou V, Lawrence JE, Garner DM, Perry BS, Harkema JR, Godleski JJ 2017. Inhaled ambient-level traffic-derived particulates decrease cardiac vagal influence and baroreflexes and increase arrhythmia in a rat model of metabolic syndrome. *Particle & Fibre Toxicology*. 14(1):16. PMID: 28545487.

5. I also recently authored an extensive review that details the many circumstances under which the ANS acts both as a mediator and byproduct of cardiovascular dysfunction due to disease and toxins.

- a. ***Carli AP**, Farraj AK, and Roberts AM, (2018), The Role of the Autonomic Nervous System in Cardiovascular Toxicity. In: McQueen, C.A. (ed.), *Comprehensive Toxicology*, 3e, Vol. 13, Oxford: Elsevier Ltd. <http://dx.doi.org/10.1016/B978-0-12-801238-3.64259-9>

*: corresponding author.

Complete list of references:

<http://www.ncbi.nlm.nih.gov/myncbi/browse/collection/40314864/?sort=date&direction=descending>

D. Research Support

Ongoing

NIH/NIEHS P42 ES023716 (PI: Srivastava) 04/01/17-03/31/22 1.2 Calendar Months

Environmental Exposure and Cardiometabolic Disease

This project will examine the cellular and molecular mechanisms by which volatile organic compounds exert cardiometabolic toxicity.

Role: Co-I

University of Louisville School of Medicine Basic Grant (Carli, PI) 9/2016-9/2018

Project: Air Pollution and Heart Failure – Role of Autonomic Nervous System in Cardiac Dysfunction and Decreased Regenerative Capacity

This project examines whether β -adrenergic receptor inhibition prevents ambient PM exposure-induced exacerbation of cardiac hypertrophy in a mouse model of transverse aortic constriction.

Role: PI

NIH/FDA 1P50 HL120163 (Bhatnagar, PI; Robertson, PI) 09/19/2013-08/31/2018 0.6 Calendar Months

American Heart Association-Tobacco Regulation and Addiction Center

Center investigators will study the cardiovascular toxicity of tobacco products and the relationship between tobacco product use and subclinical progression of cardiovascular disease to identify sensitive and robust biomarkers of cardiovascular injury related to tobacco product exposure.

Role: Project 1 Co-I

American Heart Association-Tobacco Regulation and Addiction Center Fellowship

7/2017-7/2018

Project: Acute Electrophysiologic and Hemodynamic Effects of ENDS Aerosols and Their Constituents

This project investigates the adverse effects of ENDS aerosols and their associated HPHCs, including carbonyls and nicotine, on cardiac electrophysiology and blood pressure.

Role: PI

Completed

EPA RD 83479801 P. Koutrakis (PI) 12/01/10 - 11/30/15

EPA Clean Air Research Center (CLARC): Air Pollution Mixtures: Health Effects Across Life Stages

The fundamental objectives of the Center are to investigate the effects of individual pollutants, pollution source types and multi-pollutant mixtures on cognitive and neuropsychological function, cardiovascular and

endothelial function, inflammation, birth weigh and growth, CVD hospitalization and mortality across life stages; and, to identify susceptibility and vulnerability factors that can modify these effects.

Role: Participating Investigator

Harvard NIEHS Center Pilot (P30ES000002) (Carll PI; Godleski PI) 4/2014-4/2015

Project: Does Inhalation of Traffic-Related Particulates Impair Cardiac Performance?

The goal of this project was to apply chronic LV pressure telemetry and stress tests in rats to determine if repeated exposure to traffic-derived PM impairs cardiac performance via autonomic imbalance.

Role: PI

SOUTH AMERICAN CONSORTIUM. P. Koutrakis (PI) 1/2012 - 12/2014

Health and Environmental Impacts of Exhaust from Biofuels

Role: Participating Investigator

NIH Individual T32 (HL007118). J. Fredberg (PI) 2/2013-10/2015

Training in Interdisciplinary Pulmonary Sciences.

Role: Research Fellow

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: Joy L. Hart

eRA COMMONS USER NAME (credential, e.g., agency login): JLHART01

POSITION TITLE: Professor, Dept. of Communication; Exec. Dir., University Honors Program

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 <i>(if applicable)</i>	Completion Date MM/YYYY	FIELD OF STUDY
University of Kentucky, Lexington, Kentucky, USA	B.A.	05/82	English
University of Kentucky, Lexington, Kentucky, USA	B.A.	12/82	Education
University of Kentucky, Lexington, Kentucky, USA	M.A.	12/84	Communication
University of Kentucky, Lexington, Kentucky, USA	Ph.D.	08/88	Communication

A. Positions and Honors

- 1987-1988 Research Associate, Survey Research Services, Council of State Governments, Lexington, KY.
- 1988-1990 Assistant Professor, Faculty of Communication, University of Tulsa, Tulsa, OK.
- 1990-1996 Assistant Professor, Department of Communication, University of Louisville, Louisville, KY.
- 1996-2004 Associate Professor, Department of Communication, University of Louisville, Louisville, KY.
- 2001 Visiting Associate Professor, Department of Management Communication, School of Management, University of Waikato, Hamilton, New Zealand.
- 2004-2007 Associate Appointment, Department of Epidemiology and Clinical Investigation Sciences, School of Public Health, University of Louisville, Louisville, KY.
- 2004-date Professor, Department of Communication, University of Louisville, Louisville, KY.
- 2014-date Executive Director, University Honors Program, University of Louisville, Louisville, KY.

Editor

- 2002-2005 *Southern Communication Journal*.
- 2003 *American Communication Journal*, special issue.

Key Offices/Positions Held

- 1998-2002 Board of Directors, American Communication Association.
- 2001-2002 President, American Communication Association.
- 2009-2010 President, Kentucky Communication Association.
- 2009-2012 Chair, Sustainability Council, University of Louisville. Founding member, 2008-present; Chair, Committee on Research and Education, Sustainability Council, 2008-2009 (member, 2009-present).
- 2009-2014 Co-Developer and Facilitator of Green Threads: Infusing Sustainability Across the Curriculum, Faculty Development Program.
- 2009-date Founding Member/Steering Committee, Peace, Justice, and Conflict Transformation Program.

2011-date Faculty Affiliate, Anne Braden Institute for Social Justice Research.
2010-date Faculty Advisor, RSO GRASS (Group Recycling and Sustainable Solutions).

C. Contribution to Science

Research Area: Tobacco rhetoric and tobacco control

In this line of work, my focus is examining the types of arguments made in favor of and against tobacco regulation. In particular, interest centers in the roots of arguments and how particular arguments can be challenged (i.e., identifying their vulnerabilities). Subsets of this work identify strategies for restricting access to tobacco and tobacco consumption.

Hart, J. L., Esrock, S. L., & Leichty, G. B. (2006). "Call 'em like we see 'em": Responding to unfair and unethical charges in tobacco control. In S. May (Ed.), *Cases in organizational communication: Ethical perspectives and practices* (pp. 305-315). Thousand Oaks, CA: Sage.

Esrock, S. L., **Hart, J. L.**, & Leichty, G. (2007). Smoking out the opposition: The rhetoric of reaction and the Kentucky cigarette excise tax. In L. Frey & K. Carragee (Eds.), *Communication and activism: Communication for social change* (pp. 385-410). Cresskill, NJ: Hampton Press. (Book won the National Communication Association, Applied Communication Division's 2008 Award.)

Leichty, G., Esrock, S. L., Seay, D., & **Hart, J. L.** (2008). Public health concerns or business peril?: Issues and arguments about smoking bans in Kentucky. *Kentucky Journal of Communication*, 27, 147-169.

Tompkins, L. K., Sears, C. G., **Hart, J. L.**, Walker, K. L., Lee, A. S., & Bhatnagar, A. (2017). "If you are old enough to die for your country, you should be able to get a pinch of snuff": Views of Tobacco 21 among Appalachian youth. *Journal of Applied Research on Children: Informing Policy for Children at Risk*, 8(2).

Research Area: Tobacco campaigns

Across time, I have explored methods of designing campaigns and other messaging to reduce smoking and its health consequences.

Esrock, S. L., **Hart, J. L.**, & Leichty, G. (2004). Smudging the 'golden leaf': A communication campaign to reduce the burden of tobacco in Kentucky. *Electronic Journal of Communication/La Revue Electronique de Communication*, 13(4).

Hart, J. L., Esrock, S. L., & Leichty, G. (2008). Blowing smoke and billowing logic: Consulting on cigarette excise tax increase messages. *Communication Education*, 57, 434-442.

Ridner, S. L., Walker, K. L., **Hart, J. L.**, & Myers, J. A. (2010). Smoking identities and behavior: Evidence of discrepancies, issues for measurement and intervention. *Western Journal of Nursing Research*, 32, 434-446. [Reprinted in D. Loseke's *Principles of social research design*, 2012, Sage.]

Esrock, S. L., Walker, K. L., & **Hart, J. L.** (Eds.). (2014). *Talking tobacco: Interpersonal, organizational, and mediated messages*. New York: Peter Lang.

Research Area: Workplace/organizational communication

My work in organizations seeks to understand the experiences of the average employee. In particular, I am interested in how employees interpret workplace messages and how they make sense of their organizational experiences. I have also examined how communication shapes the types of groups and workplaces that are created (e.g., individuals' orientations to and skills in communicating, organizational culture, team cooperation).

Allen, M. W., Coopman, S., **Hart, J. L.**, & Walker, K. L. (2007). Workplace surveillance and managing privacy boundaries. *Management Communication Quarterly*, 21, 172-200.

K'Meyer, T. E., & **Hart, J. L.** (2009). *"I saw it coming": Worker narratives of plant closings and job loss*. New York: Palgrave Macmillan.

Hart, J. L., Walker, K. L., Sears, C. G., Lee, A. S., Smith, C., Siu, A., Keith, R., & Ridner S. L. (2017). Vape shop employees: Public health advocates? *Tobacco Prevention and Cessation*, 2(Suppl).

Ridner, S. L., Keith, R. J., Walker, K. L., **Hart, J. L.**, & Robertson, S. E. (2017). Primary care nurse practitioners' perceptions of the use of e-cigarettes. *Journal for Nurse Practitioners*. 13, e283–e286.

Research Area: Health beliefs and practices

In this research area, work centers in identifying and examining health beliefs and practices. More specifically, the intersections between how we produce and process health-related information are explored as well as how such information translates to and also evidences our health beliefs and results in specific practices.

Hart, J. L., & Walker, K. L. (2008). Communicating health beliefs and practices. In K. B. Wright & S. D. Moore (Eds.), *Applied health communication* (pp. 125-142). Cresskill, NJ: Hampton Press.

Walker, K. L., **Hart, J. L.**, Gregg, J. L., & LaJoie, A. S. (2009). Undressing "health fashion": An examination of health-cause clothing and accessories. *Health Promotion Practice*, 10, 1-10.

Hart, J. L., & Walker, K. L. (2011). Health education and health communication: Projects in two rural villages in Belize. In G. Steele (Ed.), *Health communication in the Caribbean and beyond: A reader* (pp. 212-226). Kingston, Jamaica: University of the West Indies Press.

Hart, E. P., Sears, C. G., **Hart, J. L.**, & Walker, K. L. (2017). Electronic cigarettes and communication: An examination of college students' perceptions of safety and use. *Kentucky Journal of Communication*, 36.

Sears, C. G., **Hart, J. L.**, Walker, K. L., & Robertson, R. M. (2017). Generally recognized as safe: Uncertainty surrounding e-cigarette flavoring safety. *International Journal of Environmental Research and Public Health*, 14, 1274-1278.

Hart, J. L., Walker, K. L., Sears, C. G., Tompkins, L. K., Lee, A. S., Mattingly, D. T., Groom, A., Landry, R., Giachello, A. L., Payne, T. J., Kesh, A., Siu, A., Smith, C., & Robertson, R. M. (2018). The "state" of tobacco: Perceptions of tobacco among Appalachian youth in Kentucky. *Tobacco Prevention and Cessation*, 4.

Research Area: Health communication

This work examines how individuals communicate about health. Across these studies, foci have included acute health events, cancer diagnoses, environmental issues, HIV and AIDS, and communication strategies to raise awareness.

Walker, K. L., **Hart, J. L.**, Della, L., Ashlock, M. Z., & Hoag, A. (2012). Mom is no longer mom: Adult children discuss their parents' acute health events. In F. C. Dickson and L. M. Webb (Eds.), *Communication for families in crisis: Theories, methods, strategies* (pp. 249-277). New York, NY: Peter Lang.

Walker, K. L., **Hart, J. L.**, & D'Silva, M. U. (Eds.). (2012). *Communicating about HIV/AIDS: Taboo topics and difficult conversations*. Cresskill, NJ: Hampton Press.

Hart, J. L., & Walker, K. L. (2014). International health communication activism: A service-learning pedagogy to promote global understanding. In L. R. Frey & D. L. Palmer (Eds.), *Teaching communication activism: Communication education for social justice* (pp. 293-320). New York, NY: Hampton Press. (Book won the National Communication Association, Applied Communication Division's 2015 Award.)

Sears, C. G., **Hart, J. L.**, Walker, K. L., Lee, A. S., Keith, R., & Ridner, S. L. (2016). A dollars and "sense" exploration of vape shop spending and e-cigarette use. *Tobacco Prevention and Cessation*, 2(Suppl), 1-7.

Lee, A. S., **Hart, J. L.**, Sears, C. G., Walker, K. L., Siu, A., & Smith, C. (2017). A picture is worth a thousand words: Electronic cigarette content on Instagram and Pinterest. *Tobacco Prevention and Cessation*, 3.

Sears, C. G., Walker, K. L., **Hart, J. L.**, Lee, A. S., Siu, A., & Smith, C. (2017). Clean, cheap, convenient: Promotion of electronic cigarettes on YouTube. *Tobacco Prevention and Cessation*, 3.

D. Research Support

Robertson, R. M., Bhatnagar, A. (PIs), et al.; AHA Tobacco Regulation and Addiction Center (A-TRAC) (three project areas—1. Cardiovascular Toxicity of Tobacco Products, 2. Cardiovascular Injury Due to Tobacco Use, and 3. Perception of Tobacco Use in Vulnerable Populations). Co-PI on Project 3. National Institutes of Health (NIH) and Food and Drug Administration (FDA), 2013-2018.

Srivastava, S. (PI), Bhatnagar, A. (Co-I), et al. Environmental Exposure and Cardiometabolic Disease. Hart Co-I on Research Translation Core. National Institutes of Health (NIH)/National Institute of Environmental Health Sciences (NIEHS) [Superfund Hazardous Substance Research and Training Program (P42); Tracking Number: GRANT12141117], 2017-2022.

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: Walker, Kandi L.

eRA COMMONS USER NAME (credential, e.g., agency login): KLWALK03

POSITION TITLE: Professor, Department of Communication, College of Arts and Sciences, University of Louisville

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 Tennessee, Knoxville, Tennessee, USA	B.A.	05/94	Speech Communication
University of Memphis, Memphis, Tennessee, USA	M.A.	5/96	Communication
University of Denver, Denver, Colorado, USA	Ph.D.	05/99	Communication

B. Positions and Honors

Key Positions and Employment

- 1999-2004 Assistant Professor, Department of Communication, University of Louisville, Louisville, KY.
- 2004-2012 Associate Professor, Department of Communication, University of Louisville, Louisville, KY.
- 2007-date Co-Director of Interdisciplinary Programs, International Service Learning Program.
- 2012-date Professor, Department of Communication, University of Louisville, Louisville, KY.
- 2016-date Director of Undergraduate Studies, Department of Communication, University of Louisville, Louisville, KY.
- 2016-date Vice Chair, Department of Communication, University of Louisville, Louisville, KY.

Other Experience and Professional Memberships

- Member, Editorial Boards
- 2002-date *Southern Communication Journal*
- 2010-date *Kentucky Journal of Communication*
- 2012-2016 *Partnerships: A Journal of Service-Learning and Civic Engagement*

Honors (last 6 years)

- 2011 Distinguished Teaching Award, College of Arts and Sciences, University of Louisville.
- 2011 President’s Award for Distinguished Teaching, University of Louisville.
- 2011 Outstanding Advisor, University of Louisville.
- 2012 Outstanding Faculty Advisor, State of Kentucky, National Academic Advising Association.
- 2012 Outstanding Faculty Advisor, National, National Academic Advising Association.
- 2014 Outstanding Community Engagement Award, University of Louisville.
- 2016 Excellence in the Practice of Communication Arts Award, Kentucky Communication Association.

C. Contributions to Science

1. Research Area: Health communication in applied settings. My early publication and research interests explored how health communication pattern emerged in interpersonal and media contexts.
 - a. Hughes, P., **Walker, K. L.**, & Scholl, J. (2005). The influence of expectations for health-related talk on reports of marital satisfaction. *Communication Research Reports*, 22, 167-174. <http://dx.doi.org/10.1080/00036810500206925>
 - b. **Walker, K. L.**, Hart, J. L., Gregg, J. L., LaJoie, A. S. (2010). Undressing “health fashion”: An examination of health-cause clothing and accessories. *Health Promotion Practice*, 11(5), 665-74. PMID: 19321886.
 - c. Jorayeva, A., Ridner, S.A., Staten, R., **Walker, K.L.** (2017). A novel text message-based motivational interviewing intervention for college students who smoke cigarettes. *Tobacco Prevention and Cessation*, 3(129). 1-10. DOI: <https://doi.org/10.18332/tpc/78509>
2. Research Area: Health messages across relationships. My research interests explored how health messages can affect personal and interpersonal relationships.
 - a. **Walker, K. L.**, & Dickson, F. C. (2004). An exploration of illness-related narratives in marriage: The identification of illness-identity scripts. *Journal of Social and Personal Relationships*, 21(4), 527-544. <https://doi.org/10.1177/0265407504044846>
 - b. **Walker, K. L.**, Hart, J. L., Della, L., Ashlock, M. Z., & Hoag, A. (2012). “My mom is no longer my mom”: How adult children caregivers talk about aging parents’ acute health events and the associated life and role changes. In L. M. Webb and F. C. Dickson (Eds.), *Communication for families in crisis: Theories, methods, strategies*, 249-278 Cresskill, NJ: Hampton Press.
 - c. Tompkins, L. K., Sears, C. G., Hart, J. L., **Walker, K. L.**, Lee, A. S., & Bhatnagar, A. (2017). “If you are old enough to die for your country, you should be able to get a pinch of snuff”: Views of Tobacco 21 among Appalachian youth. *Journal of Applied Research on Children*, 8(2), Article 2.
3. Research Area: Culturally diverse health messages. In addition to the above work, I have focused research attention to health messages across culturally diverse settings. I served as the primary investigator, co-investigator, or team collaborator in all of these studies.
 - a. Hart, J. L., & **Walker, K. L.** (2014). International health communication activism: A service-learning pedagogy to promote global understanding. In L. R. Frey & D. L. Palmer (Eds.), *Teaching communication activism: Communication education for social justice* (pp. 293-320). New York, NY: Hampton Press. **Chapter (International health communication activism: A service-learning pedagogy to promote global understanding) in book that won the National Communication Association, 2015 Award for Edited Volume.
 - b. Giachello, A. L., Robertson, R. M., Payne, T. J., Rodriguez, C. J., **Walker, K. L.**, Hart, J. L., Groom, A., Quiroz, C., Navas, E., & Kesh, A. (2015). Hispanic/Latino knowledge, attitudes, and behaviors about tobacco use: Results of focus groups in four U.S. cities. *Circulation*, 132, A19070.
 - c. **Walker, K. L.**, Hart, J. L., Sears, C., Lee, A., Smith, C., & Siu, A. (2015). Appalachian youth and tobacco products: Perceptions, attitudes, and behaviors. *Circulation*, 132, A18094.
 - d. Ridner, S. L., Keith, R. J., **Walker, K. L.**, Hart, J. L., Robertson, S. E. (2017). Primary care nurse practitioners’ perceptions of the use of e-cigarettes for smoking cessation. *Journal of the American Association of Nurse Practitioners*, 13(6), 3283-e286. <https://doi.org/10.1016/j.nurpra.2017.01.019>
4. Research Area: Tobacco communication and health messages. My research focus explores the intersection between health and interpersonal and social communication. This line of work examines how people perceive the social world surrounding health issues. Specifically, my research examines how people talk and communicate about risky health behaviors. I served as the primary investigator or co-investigator in all of these studies
 - a. Ridner, S. L., **Walker, K. L.**, & Hahn, E. J. (2008). College students’ knowledge, exposure, and the pervasiveness of tobacco marketing in two Kentucky cities. *Kentucky Journal of Communication*, 27, 107-124.

- b. Ridner, S. L., **Walker, K. L.**, Hart, J. L., & Myers, J. A. (2010). Smoking identities and behavior: Evidence of discrepancies, issues for measurement and intervention. *Western Journal of Nursing Research*, 32, 434-446. PMID: 20685903 [Reprinted in D. Loseke's *Principles of social research design*, 2012, Sage.]
 - c. Esrock, S. L., **Walker, K. L.**, & Hart, J. L. (Eds.). (2014). *Talking tobacco: Interpersonal, organizational, and mediated messages*. New York, NY: Peter Lang.
 - d. Sears, C. G., **Walker, K. L.**, Hart, J. L., Lee, A. S., Siu, A., Smith, C., Bhatnagar, A., & Robertson, R. M. (2016). Perceptions and use of electronic cigarettes among middle and high school students in Appalachia. *Circulation*, 134, A14605.
5. Research Area: Tobacco perceptions. Important to understanding tobacco communication is to better understand current perceptions of tobacco use and behaviors. This line of work has focused on perceptions across different populations. I served as the primary investigator or co-investigator in all of these studies
- a. Hart, J. L., Esrock, S. L., & **Walker, K. L.** (2014). What's all the talk about?: Communication perspectives on tobacco issues. In S. L. Esrock, **K. L. Walker**, & J. L. Hart (Eds.), *Talking tobacco: Interpersonal, organizational, and mediated messages* (pp. 1-8). New York, NY: Peter Lang.
 - Hart, J. L., **Walker, K. L.**, Sears, C. G., Lee, A. S., Smith, C., Siu, A., Keith, R., & Ridner, S. L. (2017). Vape shop employees: Public health advocates? *Tobacco Prevention and Cessation*, 2(Suppl). PMID: PMC5512600.
 - b. Sears, C. G., **Walker, K. L.**, Hart, J. H., Lee, A. S., Siu, A., & Smith, C. (2017). Clean, cheap, convenient: Promotion of electronic cigarettes on YouTube. *Tobacco Prevention and Cessation*. PMID: 28725876.
 - c. Hart, J. L., Walker, K. L., Sears, C. G., Tompkins, L. K., Lee, A. S., Mattingly, D. T., Groom, A., Landry, R., Giachello, A. L., Payne, T. J., Kesh, A., Siu, A., Smith, C., & Robertson, R. M. (2018) The "state" of tobacco: Perceptions of tobacco among Appalachian youth in Kentucky. *Tobacco Prevention and Cessation*, 4, 1-7. DOI: 10.18332/tpc/81857

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

Robertson, R. M., Bhatnagar, A. (PIs), et al.; AHA Tobacco Regulation and Addiction Center (A-TRAC) (three project areas—1. Cardiovascular Toxicity of Tobacco Products, 2. Cardiovascular Injury Due to Tobacco Use, and 3. Perception of Tobacco Use in Vulnerable Populations). Co-PI on Project 3. National Institutes of Health (NIH) and Food and Drug Administration (FDA), \$19.6 million, 2013-2018.

Robertson, R. M., Payne, T. J., Walker, K. L., Hart, J. L., Giachello, A. L., et al. Vaping Among Ethnoracial Subgroups: Patterns of Use and Perceptions of Risk. National Institutes of Health (NIH) and Food and Drug Administration (FDA) Pilot Research Grants Program (5P50HL120163-03); pilot funds approved; funded through A-TRAC Project 3, \$50,000, March-June 2016.

Srivastava, S. (PI), Bhatnagar, A. (Co-I), et al. Environmental exposure and cardiometabolic disease. Walker on Research Translation Core. National Institutes of Health (NIH)/National Institute of Environmental Health Sciences (NIEHS) [Superfund Hazardous Substance Research and Training Program (P42); Tracking Number: GRANT12141117], \$9.2 million, 2017-2022.

Walker, K. L. Dual credit education/communication program. Commonwealth of Kentucky, 2017-2020.

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: Rachel J. Keith

eRA COMMONS USER NAME (credential, e.g., agency login): R.J.Keith

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 <i>(if applicable)</i>	Completion Date MM/YYYY	FIELD OF STUDY
University of Louisville	BA	05/00	Biology
University of Louisville	BSN	08/11	Nursing
University of Louisville	MS	05/04	Physiology and Biophysics
University of Louisville	MSN	05/13	Adult Nurse Practitioner
University of Louisville	PhD	05/09	Physiology and Biophysics

B. Positions and Honors

Positions and Employment

- 2017- Director University of Louisville Tobacco treatment clinic
- 2015- Assistant Professor of Medicine University of Louisville
- 2015- Adjunct Professor Physiology University of Louisville
- 2014 Assistant Professor, University of Louisville School of Nursing, Louisville, KY
- 2014-2016 Fellow AHA-Tobacco Regulation and Addiction Center
- 2011-2013 Research Associate- Neonatal Associates, Louisville, KY
- 2012-2013 Nurse, Floyd Memorial Hospital
- 2011-2012 Nurse, University of Louisville Hospital
- 2009-2011 Post-doctoral Fellow Diabetes and Obesity Center, University of Louisville
- 2004-2009 Pre-doctoral Fellow Physiology and Biophysics, University of Louisville

Other Experience and Professional Memberships

- 2017- Certified Tobacco Treatment Specialist
- 2013- American Academy of Nurse Practitioners
- 2012- Kentucky Coalition of Nurse Practitioners and Nurse Midwives
- 2011- Sigma Theta Tau International membership
- 2009- American Heart Association
- 2008-2010 American Society for Biochemistry and Molecular Biology

Honors

- 2014-2016 AHA- Tobacco Regulation and Addiction Center Fellowship
- 2016 AHA Research Leaders Academy
- 2011-2013 Graduate School Scholarship
- 2011 Dean’s List University of Louisville, School of Nursing
- 2009-2011 Post-doctoral Fellow- Diabetes and Obesity Center, Louisville, KY
- 2007-2009 Graduate Student Fellowship Award
- 2004-2009 Graduate Research Associate- Center for Molecular Cardiology, Louisville KY

C. Contribution to Science

1. My early work addresses mechanisms of cardiovascular disease, with a focus on the role of aldehydes, such as HNE. Many aldehydes are found as part of environmental exposures, and these highly reactive substances can cause damage through a variety of mechanisms. Specifically aldehydes can increase inflammation, mitochondrial damage, and lipid peroxidation. Epidemiological studies suggest that aldehydes may contribute to cardiovascular disease as well as evidenced by to adverse cardiovascular outcomes. Interestingly, there are ways to prevent these effects. Mechanisms that promote detoxification of the reactive aldehydes or the metabolites associated with them can decrease damage to the heart or vasculature. Mitochondria may play an important role in protecting the cardiovascular system from the toxicity of aldehydes.

A. West MB., Rokosh G., Obal D., Velayutham M., Xuan YT., Hill BG., Keith RJ., Schrader J., Guo Y., Conklin DJ., Prabhu SD., Zweier JL., Bolli R., Bhatnagar A. Cardiac myocyte-specific expression of inducible nitric oxide synthase protects against ischemia/reperfusion injury by preventing mitochondrial permeability transition. *Circulation*. 118(19):1970-8, 2008 Nov 4.

B. Keith RJ., Haberzettl P., Vladykovskaya E., Hill BG., Kaiserova K., Srivastava S., Barski O., Bhatnagar A. Aldose reductase decreases endoplasmic reticulum stress in ischemic hearts. *Chemico-Biological Interactions*. 178(1-3):242-9, 2009 Mar 16.

C. Keith RJ., Bhatnagar A. Cardioprotection and the mitochondrial permeability transition. *Minerva Cardioangiologica*. 58(2):241-251, 2010 Apr.

2. In addition to the contribution described above, with a multi-institution and multi-disciplinary team, I have recruited and completed study visits on hundreds of participants to look at toxic exposures and the effect on biomarkers of injury. Specifically we are interested in identifying early indicators of cardiovascular damage that may help predict long term cardiovascular outcomes or mortality. We are pursuing both traditional markers of cardiovascular disease and unique markers as well. Furthermore, pilot studies I have recruited participants in multiple studies looking at the effects of greenness that will be used to explore the effects of greenness on chronic disease risk, including cardiovascular disease and diabetes.

A. Keith RJ., Al Rifai M, Carruba C, DeJarnett N, McEvoy JW, Bhatnagar A, et al. Tobacco Use, Insulin Resistance, and Risk of Type 2 Diabetes: Results from the Multi-Ethnic Study of Atherosclerosis. *PLoS ONE* 2016; 11(6): e0157592. doi:10.1371/journal.pone.0157592

B. Al-Rifai, M., DeFilippis, AP., McEvoy, JW., Hall, ME., Acien, AN., Jones, MR., Keith RJ., Magid, HS., Rodriguez, CJ., Barr, GR., Benjamin, EJ., Robertson, RM., Bhatnagar, A. and Blaha, MJ. The Relationship of Smoking Intensity and Subclinical Cardiovascular Injury: The Multi-Ethnic Study of Atherosclerosis (MESA). *Atherosclerosis*. 2017;258:119-130. doi:10.1016/j.atherosclerosis.2017.01.021.

C. Keith RJ., Fetterman JL, Riggs DW, O'Toole T, Nystoriak JL, Holbrook M, Lorkiewicz P, Bhatnagar A, DeFilippis AP, Hamburg NM. Protocol to Assess the Impact of Tobacco-Induced Volatile Organic Compounds on Cardiovascular Risk in a Cross-Sectional Cohort: Cardiovascular Injury Due to Tobacco Study. *BMJ open* (In Review)

3. In combination with a multi-disciplinary team I am establishing how the environment influences health and perception of risk in the community. The environment can be made up of many things including lifestyle choices such as physical activity, tobacco use, and diet choices as well as exposure to toxic compounds such as air pollution. Though genetics plays a role in susceptibility of individuals to disease, a larger portion likely comes from the environment. Establishing how the environment influences both disease and the perceptions of risk has many policy and regulatory implications.

A. Sears CG, Hart JL, Walker KL, Lee AS, Keith RJ., Ridner SL. A dollars and "sense" exploration of vape shop spending and e-cigarette use. *Tob Prev Cessation*; 2016; 2(Supplement):7. doi: 10.18332/tpc/67435.

B. Engle, WA., West, KW., Hocutt, GA., Pallotta, EK., Haney, B., Keith, RJ. Stewart, DL., Knodel, E., Suttner, D., Chapman, R., Bartlett, R., Thomas, A., Schwerin, B., Stork, E., Crowley, M., Gebregziabher, N., Fadel, W. Adult Outcomes After Newborn Respiratory Failure Treated with Extracorporeal Membrane Oxygenation. Pediatric Crit Care Med. 2017;18(1):73-79. doi: 10.1097/PCC.0000000000001018.

C. White, WB, Cain, LR, Benjamin, EJ, DeFilippis, A, Blaha, MJ, Wang, W, Okhomina, V, Keith, RJ, Al Rifai, M, Kianoush S, Winniford, MD, Robertson, RM, Bhatnagar, A; Correa, A, Hall, ME. High-Intensity Cigarette Smoking is Associated with Incident Diabetes: The Jackson Heart Study. JAMA (In press)

Complete List of Published Work in MyBibliography: <http://www.ncbi.nlm.nih.gov/sites/myncbi/1RIKe-ynEFgQ-/bibliography/48227193/public/?sort=date&direction=ascending>

D. Research Support

Ongoing Research Support

2P20GM103492 (Bhatnagar, Aruni PI) 08/01/13-06/30/18 6.0 Calendar
NIH *

Center of Biomedical Research Excellence (COBRE) in Diabetes and Obesity Research

The overall goal of this COBRE is to establish an internationally recognized center of research aimed at understanding the cardiovascular causes and consequences of diabetes and obesity.

Role: Project PI

R01HL122676-01A1 (Conklin, D.J., PI) 04/01/2015-03/31/2020 0.6 Calendar
NIH/NHLBI

Cardiovascular Toxicity of Tobacco Products and Constituents

The goal of these studies is to identify the sensitivity of the cardiovascular system to harmful and potentially harmful constituents (HPHCs) in tobacco products. Hence, we will use an animal model and determine the relative toxicity of major HPHCs in cigarette smoke and smokeless tobacco, determine the toxicity profile of individual HPHCs and assess how the toxicity of an individual HPHC is modified by other HPHCs. To validate this toxicity profile in humans, we will examine the relationship between exposure to different HPHCs and cardiovascular toxicity in a well-characterized cohort.

P42 ES023716 (PI: Srivastava) 07/01/17-06/31/23 0.6 Calendar
NIH/NIEHS

Environmental Exposure and Cardiometabolic Disease

This project will examine the cellular and molecular mechanisms by which volatile organic compounds exert cardiometabolic toxicity.

Role: Co-investigator

BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME Timothy E. O'Toole, Ph.D.		POSITION TITLE Assistant Professor; Department of Cardiology	
eRA COMMONS USER NAME Teetoo01			
EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)			
INSTITUTION AND LOCATION	DEGREE (if applicable)	YEAR(s)	FIELD OF STUDY
University of Michigan, Ann Arbor, Michigan	B.S.	1975-1979	Chemistry, Cell Biology
University of Michigan, Ann Arbor, Michigan	Ph.D.	1980-1987	Biological Chemistry
Scripps Research Institute, LaJolla, California	Post-doctoral	1987-1991	

B. Positions and Honors**Positions and Employment:**

1991-1993 Senior Research Associate, Committee for the Study of Vascular Biology, Scripps Research Institute
 1993-2000 Assistant Professor, Dept. of Vascular Biology, Scripps Research Institute
 2001 Scientific Curator, Incyte Genomics
 2001-2006 Project Scientist, Dept. of Molecular Cardiology, Cleveland Clinic Foundation
 2006-present Assistant Professor, Dept. of Cardiology, University of Louisville

Other Experience and Professional Memberships:

1993- Member, American Association for the Advancement of Science
 1996- Member, American Society for Cell Biology
 1997-1998 Arthritis Foundation Inflammation Study Section
 1997 NASA Cellular and Molecular Biology Study Section
 2009- Member, American Heart Association
 2011- American Heart Association, Basic Cell Structure and Survival Study Section
 2012- Member, Society of Toxicology
 2016- Editorial Board, *Circulation Research*

Honors and Awards:

1989-1992 Arthritis Foundation Postdoctoral Fellowship
 1991 Sanofi Foundation Prize for Thrombosis Research
 1994-1999 Established Investigator, American Heart Association

C. Contributions to Science

1. Endothelial progenitor cells (EPCs) are stem cell type that assist in vascular repair through terminal differentiation into mature endothelial cells or through paracrine stimulation of existing cells. Epidemiological and laboratory studies indicate that the levels of these cells are reflective of vascular health and are inversely correlated with cardiovascular disease risk. Furthermore, the levels and functionality of these cells are particularly sensitive to toxicological stimuli or external influences such as hyperglycemia. Listed below are a series of manuscripts which characterize EPCs quantitatively and qualitatively in response to air pollution exposure or diabetes. Ongoing, mechanistic studies seek to identify the means to reverse these effects and improve vascular health after exposures and in disease states.

- a. **O'Toole T.E.**, Hellmann, J., Wheat, L., Haberzettl, P., Lee, J., Conklin, D.J., Bhatnagar, A., Pope, C.A.III. Episodic exposure to fine particulate air pollution decreases circulating levels of endothelial progenitor cells. *Circ. Res.* 107: 200-203, 2010. PMID: PMC2943671
 - b. Haberzettl, P., Lee, J., Duggineni, D., McCracken, J., **O'Toole, T.E.**, Bhatnagar, A., Conklin, D.J. Exposure to fine air particulates prevents mobilization of endothelial progenitor cells from the bone marrow. *Environmental Health Perspectives* 120: 848-856, 2012. PMID: PMC3385427
 - c. DeJarnett, N., Yeager, R., Conklin, D.J., Lee, J., **O'Toole, T.E.**, McCracken, J., Abplanalp, W., Srivastava, S., Riggs, D.W., Hamzeh, I., Wagner, S., Chugh, A., DeFilippis, A., Ciszewski, T., Wyatt, B., Becher, C., Higdon, D., Ramos, K.S., Tollerud, D.J., Myers, J.A., Rai, S.N., Shah, J., Prabhu, S.D., and Bhatnagar, A.: Residential proximity to major roadways is associated with AC133⁺ circulating angiogenic cells. *Arter. Throm. Vasc. Biol.* 35: 2468-77, 2015. PMID: 26293462
 - d. Abplanalp, W.T., Conklin, D.J., Cantor, J.M., Ginsberg, M.H., Wysoczynski, M., Bhatnagar, A., and **O'Toole, T.E.** Enhanced integrin $\alpha 4\beta 1$ -mediated adhesion contributes to a mobilization defect of endothelial progenitor cells in diabetes. *Diabetes*. In press 2016. PMID: 27495221
2. Acrolein is a common environmental pollutant found in automobile exhaust, factory emissions, cigarette smoke, fried foods and some beverages, and even water supplies. It is also generated endogenously as a byproduct of the myeloperoxidase reaction and from the metabolism of certain pharmaceuticals. Exposure to soluble or volatile acrolein is associated with cardiovascular dysfunction but the basis for these associations is unclear. In the following series of papers, we have examined the epidemiological, biochemical, and cellular consequences of exposure to acrolein. Understanding the basis of acrolein toxicity may identify means to mitigate the pathologies resulting from exposure.
- a. **O'Toole, T.E.**, Zheng, Y.-T., Hellmann, J., Conklin, D.J., Barski, O., and Bhatnagar, A.: Acrolein activates matrix metalloproteinases by increasing reactive oxygen species in macrophages. *Tox. App. Pharm.* 236: 194-201, 2009. PMID: PMC2677519
 - b. Sithu, S.D., Srivastava, S., Siddiqui, M.A., Vladykovskaya, E., Riggs, D.W., Conklin, D.J., Haberzettl, P., **O'Toole, T.**, Bhatnagar, A., D'Souza, S.E. Exposure to acrolein by inhalation causes platelet activation. *Tox. App. Pharm.* 248: 100-110, 2010. PMID: PMC2946419
 - c. **O'Toole, T.E.**, Abplanalp, W., Li, X., Cooper, N., Conklin, D.J., Haberzettl, P., and Bhatnagar, A. Acrolein decreases endothelial cell migration and insulin sensitivity through induction of let-7a. *Toxicol. Sci.* 140: 271-82, 2014. PMID: 24812010 PMID: PMC4176051
 - d. DeJarnett, N., Conklin, D.J., Riggs, D.W., Myers, J.A., **O'Toole, T.E.**, Hamzeh, I., Wagner, S., Chugh, A., Ramos, K.S., Srivastava, S., Higdon, D., Tollerud, D.J., DeFilippis, A., Becher, C., Wyatt, B., McCracken, J., Abplanalp, W., Rai, S.N., Ciszewski, T., Xie, Z., Yeager, R., Prabhu, S.D., and Bhatnagar, A.: Acrolein Exposure is Associated with Increased Cardiovascular Disease Risk. *Journal of the American Heart Association* 3: e000934, 2014. PMID: PMC4310380
3. The integrin adhesion receptors are unique in their ability to regulate their ligand binding affinity. In a process variably referred to as integrin activation, integrin affinity modulation, or integrin inside-out signaling, cell surface integrins respond to external stimuli by changing their extracellular alignment from a low affinity binding conformation to one with a high affinity binding conformation. The molecular basis of these responses were unknown for a long time, however this series of papers outlines my involvement in studies identifying a key role for the unique sequences of the integrin α and β subunit cytoplasmic domains in this process. These studies have contributed an understanding to the regulation of integrin outside-in signaling and how this is disrupted in various disease states.
- a. **O'Toole, T.E.**, Loftus, J.C., Du, X., Glass, A.A., Ruggeri, Z.M., Shattil, S.J., Plow, E.F. and Ginsberg, M.H.: Affinity modulation of the $\alpha_{IIb}\beta_3$ integrin (platelet GPIIb-IIIa) is an intrinsic property of the receptors. *Cell Regulation* 1: 883-893, 1990. PMID: PMC362859
 - b. **O'Toole, T.E.**, Mandelman, D., Forsyth, J., Shattil, S.J., Plow, E.F. and Ginsberg, M.H.: Modulation of the affinity of integrin $\alpha_{IIb}\beta_3$ (GPIIb-IIIa) by the cytoplasmic domain of α_{IIb} . *Science* 254: 845-847, 1991. PMID: 1948065

- c. **O'Toole, T.E.**, Katagiri, Y., Faull, R.J., Peter, K., Tamura, R., Quaranta, V., Loftus, J.C., Shattil, S.J., and Ginsberg, M.H.: Integrin cytoplasmic domains mediate inside-out signal transduction. *J. Cell Biol.* 124: 1047-1059, 1994. PMC2119979
 - d. **O'Toole, T.E.**, Yläne, J., and Culley B.C.: Regulation of integrin affinity states through an NPXY motif in the β subunit cytoplasmic domain. *J. Biol. Chem.* 270: 8553-8558, 1995. PMID: 7721755
4. In addition to inside-out signaling, integrins participate in outside-in signaling. That is, upon ligand binding, integrins bind to or recruit other intracellular molecules to build signaling networks. These signals affect various aspects of cell physiology including adhesion, growth, and migration. This series of papers describes studies identifying integrin domains and/or integrin-binding partners and how these impact inside-out signaling. These studies have clarified the sequence of events leading from ligand binding to altered cellular function.
- a. Yläne, J., Huuskonen J., **O'Toole, T.E.**, Ginsberg, M.H., Virtanen, I., and Gahmberg, C.G.: Mutation of the cytoplasmic domain of the integrin β_3 subunit: differential effects on cell spreading, recruitment to adhesion plaques, endocytosis and phagocytosis. *J. Biol. Chem.* 270: 9550-9557, 1995. PMID: 7721884
 - b. Shattil, S. J., **O'Toole T. E.**, Eigenthaler, M., Thon, V., Williams, M., Babior, B. M., and Ginsberg, M. H.: β_3 -endonexin, a novel polypeptide that interacts specifically with the cytoplasmic tail of the integrin β_3 subunit. *J. Cell Biol.* 131: 807-816, 1995. PMCID: PMC2120613
 - c. Buensuceso, C.B., Woodside, D., Huff, J.L., Plopper, G.E., and **O'Toole, T.E.**: The WD protein Rack1 mediates PKC and integrin dependent cell migration. *J. Cell Sci.* 114: 1691-1698, 2001. PMID: 11309199
 - d. **O'Toole, T.E.**, Bialkowska, K. Li, X., Fox, J.E.B. Tiam1 is recruited to β_1 -integrin complexes by 14-3-3 ζ where it mediates integrin-induced Rac1 activation and motility. *J. Cellular Phys.* 226: 2695-2978, 2011. PMID: 21302295
5. Angiogenesis is essential for tumor growth and metastasis. Several integrins are involved in angiogenesis, with the most crucial being $\alpha v\beta_3$. This series of papers outlines studies detailing the essential role $\alpha v\beta_3$ plays in tumor metastasis. These studies are important with regards to the development of anti-angiogenic strategies to combat tumorigenesis.
- a. Felding-Habermann, B., **O'Toole, T. E.**, Smith, J. W., Fransvea, E., Ruggeri, Z. M., Ginsberg, M. H., Hughes, P. E., Pampori, N., Shattil, S.J., Saven, J., and Mueller, B. M.: Integrin activation controls metastasis in human breast cancer. *Proc. Natl. Acad USA.* 98: 1853-1858, 2001. PMCID: PMC29346
 - b. Felding-Habermann, B., Fransvea, E., **O'Toole, T.E.**, Manzuk, L., Faha, B., and Hensler, M.: Involvement of tumor cell integrin $\alpha v\beta_3$ in hematogenous metastasis of human melanoma cells. *Clin. Exp. Metastasis* 19: 427-436, 2002. PMID: 12198771
 - c. De, S., Razorenova, O., McCabe, N.P., **O'Toole, T.**, Qin, J., and Byzova, T.V.: VEGF- integrin interplay controls tumor growth and vascularization. *PNAS* 102: 7589-7594, 2005. PMCID: PMC1129024

Complete List of Published Work in MyBibliography:

<https://www.ncbi.nlm.nih.gov/sites/myncbi/timothy.o'toole.1/bibliography/41166499/public/?sort=date&direction=ascending>

D. Research Support

Ongoing support

NIH 2RO1 ES019217-06 4/1/18- 3/31/23 O'Toole (PI)

"Endothelial progenitor cells and particulate air pollution"

In this study we will characterize the effects of exposure to particulate matter air pollution in at risk populations and assess the efficacy of an anti-oxidant intervention in mitigating these outcomes.

NIH 1-P50 HL120163-01 9/19/13 - 8/31/18 Bhatnagar (PI)
"AHA Tobacco Regulation and Addiction Center"

Role: Core C Director

As Director of this Cardiovascular Pathology Core, I will oversee a team responsible for the generation of all biochemical and cellular endpoints.

NIH P20 RR024489 9/26/13 - 6/30/18 Bhatnagar (PI)
"Center of Biomedical Research Excellence"

Role: Core B Director

As Director of the Flow Cytometry Core, I am responsible for all operations of existing flow cytometry equipment within the Diabetes and Obesity Center.

NIH 1-RO1 ES027882-01 5/1/17- 4/30/22 Haberzettl (PI)
"Air pollution, circadian rhythm disruption and cardiometabolic disease"

Role: Collaborator

I will assist in these studies on exposure to particulate matter air pollution and circadian disruptions in mice.

NIH 1-R01 HL122676-01 04/01/15 - 03/31/20 Conklin (PI)
"Cardiovascular Toxicity of Tobacco Products and Constituents"

Role: Collaborator,

I will assist in these studies on the cardiovascular toxicology of tobacco products and their constituents.

NIH P01 HL078825 06/01/17-05/31/22 Bolli (PI)
"Protection of Ischemic Myocardium"

Role: Core Director

As Pathology Core Director, I will oversee a team responsible for the collection and analysis of flow cytometry-based endpoints.

NIH P42 ES023716 4/1/17- 3/31/22 Srivastava (PI)
"Environmental Exposure and Cardiometabolic Disease"

Role: Core Director

As Metabolism and Toxicity Core Director, I will oversee a team responsible for the collection and analysis of all flow cytometry-based endpoints.

Completed support

NIH 1RO1 ES019217-05 6/1/11- 10/31/17 O'Toole (PI)
"Endothelial progenitor cells and particulate air pollution"

In this study we characterized the quantitative and qualitative properties of stem cell populations in humans and mice exposed to particulate air pollution.

UofL Center for Environmental Genomics and Integrative Biology 6/1/10-5/31/11 O'Toole (PI)
"Toxic air pollutants and endothelial progenitor cells"

In this one year intramural pilot project, we will quantify levels of antigenically defined EPC populations in mice exposed to particulate air pollution and identify interventions to enhance their mobilization.

NIH-RO1 HL53620 7/1/95 - 6/30/00 O'Toole (PI)
"Physiological mechanisms of integrin affinity modulation"

In this study we examined the molecular mechanisms of integrin function using cell-based models.

American Heart Association - Established Investigator Award 7/1/94 - 6/30/99 O'Toole (PI)
"The role of α and β cytoplasmic domains in affinity modulation of the platelet integrin α IIb β 3"

Principal Investigator/Program Director (Last, First, Middle): Timothy E. O'Toole Ph.D.

In this study we examined the molecular mechanisms of integrin function using cell-based models.

State of California - Tobacco Related Disease Research Program 7/1/92 - 6/30/95 O'Toole (PI)
"Integrin cytoplasmic domains and ligand affinity modulation"

In this study we examined the molecular mechanisms of integrin function using cell-based models.

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: 4-23-18

Name (first, middle, last):

 Aruni Bhatnagar

Degree(s): Faculty Appointment:

 PhD: Professor of Medicine

Admin Titles (if applicable):

 Director, Diabetes and Obesity Center; Co-Director, *American Heart Association Tobacco Regulation and Addiction Center* (ATRAC)

Research Expertise/Field:

 Environmental Cardiology, Diabetes
Obesity, Tobacco exposure

Campus address:

 580 S. Preston, Baxter II 421

Telephone number

 852-5966

Fax number:

 852-3663

e-mail address:

 Aruni@louisville.edu



Signature

Please describe your current Research Interests:

I have studied cardiovascular dysfunction induced by endogenous and xenobiotic oxidants, and pollutants. This work has led to the identification of several biochemical pathways for pollutant metabolism and detoxification, as well as a better understanding of how pollutants affect cardiovascular function and accelerate cardiovascular disease in both humans and animals. During the course of this work, I have acquired extensive experience in cardiovascular biochemistry, physiology and pathology. I have planned, designed, directed, conducted and reported experiments relating to the cardiovascular toxicity of environmental exposures, tobacco smoke, and endogenous and xenobiotic VOCs. I led and directed a large team of toxicologists studying how aldehydes such as acrolein and crotonaldehyde affect atherogenesis, thrombosis and myocardial ischemic injury.

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: 04/23/18

Name (first, middle, last):	Sanjay Srivastava
Degree(s): Faculty Appointment:	Ph.D., Professor of Medicine
Admin Titles (if applicable):	Director, Superfund Research Center
Research Expertise/Field:	Vascular Biology/Toxicology
Campus address:	204 F, Baxter II
Telephone number	852-5834
Fax number:	852-3663
e-mail address:	sanjay@louisville.edu



Signature

Please describe your current Research Interests: My current research interest is focused on delineating the mechanisms by which environmental pollutants such as volatile organic compounds (VOCs) cause endothelial activation, vascular inflammation, insulin resistance and atherosclerosis. My recent studies suggest that several of these toxicants induce endothelial activation and vascular inflammation by triggering endoplasmic reticulum (ER) stress. Studies are ongoing to examine which components of ER-stress affect vascular inflammation and atherosclerosis.

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: 04/23/18

Name (first, middle, last): Daniel J. Conklin

Degree(s): Faculty Appointment: Ph.D.; Professor of Medicine

Admin Titles (if applicable): Director, Inhalation Facility; Director,
Diabetes and Obesity Center Animal Phenotyping Core

Research Expertise/Field: Cardiovascular Toxicity /
Environmental Cardiology

Campus address: Delia Baxter, Rm. 404E

Telephone number (502) 852-5836

Fax number: (502) 852-3663

e-mail address: dj.conklin@louisville.edu

Signature



Please describe your current Research Interests:

How the environment influences cardiovascular disease is a critical question, and over the last 2 decades, we have documented the relatively high sensitivity of cardiovascular targets to exposures to exogenous and endogenous aldehydes – known toxic contaminants of the environment. Moreover, we have tested the metabolic basis of this cardiovascular sensitivity to aldehyde-induced injury by focusing on the specific contribution that aldehyde metabolism makes to target organ injury and disease pathogenesis. For example, we have studied aldehyde-metabolizing enzymes that mediate detoxification (via GSH conjugation) of unsaturated aldehydes, such as glutathione S-transferase P (GSTP). Importantly, GSTP modulates aldehyde toxicity under environmentally- and clinically-relevant exposure conditions (e.g., tobacco smoke, CY) in animal models, thus, exemplifying the practical strength of our approach and its theoretical underpinnings. Similarly, I have explored the relationship between inhaled pollutants and cardiopulmonary injury through mediators such as the irritant and pain receptor, TRPA1. By better understanding both targets and metabolism of aldehydes, we can develop interventional approaches that may lessen the burden of environmentally-induced cardiovascular disease.

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: _____ 4/23/2018 _____

Name (first, middle, last): Dr. Lauren C. Heberle
Degree(s): Faculty Appointment: PhD. Associate Professor, Sociology
Admin Titles (if applicable): Director, Center for Environmental Policy and Management
Research Expertise/Field: Environmental Policy/Sociology
Campus address: Lutz Hall, Sociology, 118
Telephone number 502-852-4749
Fax number:
e-mail address: Lauren.Heberle@louisville.edu

Signature



Please describe your current Research Interests:

Dr. Lauren C. Heberle is the Director of the Center for Environmental Policy and Management and Associate Professor of Sociology. She teaches environmental and social policy courses. Her expertise is in community participation in environmental decision making. Her areas of research include urban redevelopment, environmental policy, environmental justice, community engagement, and sustainable development. She serves as a member of or expert witness to a variety of local and federal task forces and committees.

Center for Healthy Air Water and Soil (CHAWS)

Membership Application Form

(Please submit Electronically)

General Profile

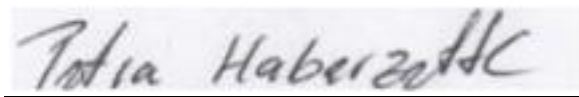
Date of Application: 04-24-2018

Name (first, middle, last):	Petra Haberzettl
Degree(s): Faculty Appointment:	PhD, Assistant Professor
Admin Titles (if applicable):	NA
Research Expertise/Field:	Cardiovascular Toxicology
Campus address:	Delia Baxter II, 404F University of Louisville 580 South Preston Street Louisville, KY, 40202,USA
Telephone number	502-852-4235
Fax number:	502-852-3663
e-mail address:	p0habe02@louisville.edu

Please describe your current Research Interests:

My current research examines how the exposure to fine particulate matter (PM_{2.5}) affects vascular circadian rhythms and endothelial progenitor cells health and how this contributes to the development of vascular and cardiometabolic injury.

Signature



Petra Haberzettl

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

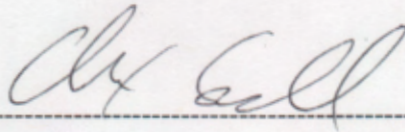
(Please submit Electronically)

General Profile

Date of Application: 4/23/18

Name (first, middle, last):	Alex Perrow Carl
Degree(s): Faculty Appointment:	Ph.D., M.S.P.H., Assistant Professor
Admin Titles (if applicable):	
Research Expertise/Field:	Cardiopulmonary Toxicology & Physiology
Campus address:	580 S. Preston St.
Telephone number	502-852-4243
Fax number:	502-852-3663
e-mail address:	alex.carll@louisville.edu

Signature



Please describe your current Research Interests:

I assess rodent and human hearts for adverse changes in electrical activity, mechanical performance, and neural regulation following exposure to multiple pollutants, including particulate matter from highways, office printers, and ambient urban airsheds, as well as diesel exhaust, volatile organic compounds (VOCs), mainstream cigarette smoke, and electronic cigarette aerosols. My lab conducts basic biochemical and molecular toxicologic assays while analyzing physiologic signals, including the electrocardiogram (ECG), arterial and left ventricular pressure waveforms, and echocardiogram. Currently we are investigating in both rodents and humans several emerging questions in environmental health sciences, including:

1. What are the neural and cellular pathways underlying pollutant-induced myocardial dysfunction and remodeling?
2. How do e-cigarette and office printer aerosols adversely affect cardiac function, intracellular signaling, and neuroregulation?
3. How do VOCs contribute to the cardiac risks of exposure to pollutant aerosols?

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: 4/23/18

Name (first, middle, last): Joy L. Hart
Degree(s): Faculty Appointment: Ph.D; Professor
Admin Titles (if applicable): _____
Research Expertise/Field: Health Communication; Environmental
Communication; Organizational
Communication
Campus address: Dept. of Communication,
310 Strickler Hall
Telephone number 502-852-6293
Fax number: _____
e-mail address: joy.hart@louisville.edu



Signature _____

Please describe your current Research Interests:

Health communication; environmental communication; links between the environment and health; vulnerable populations and health; health equity; perceptions and use of tobacco products; organizational communication

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**
(Please submit Electronically)

General Profile

Date of Application: _____

Name (first, middle, last):	Kandi L. Walker
Degree(s): Faculty Appointment:	B.A., M.A., Ph.D.; Professor of Communication
Admin Titles (if applicable):	Vice Chair – Dept of Communication; Director of Undergraduate Education – Dept of Communication
Research Expertise/Field:	Health Communication, Interpersonal Communication, Environmental Communication
Campus address:	310 Strickler Hall; Louisville, KY 40292
Telephone number	502-852-6976
Fax number:	502-852-8166
e-mail address:	Kandi.walker@louisville.edu

Signature



Please describe your current research interests:

Kandi L. Walker, Ph.D., is Professor in the Department of Communication at the University of Louisville. Her research primarily focuses on health, interpersonal, and environmental communication; specifically, her research examines how people perceive and communicate about health and environmental issues and how people perceive and communicate about risky health behaviors (i.e., smoking). Currently, she is working on several health and environmental projects with the American Heart Association, the Green Heart Project, UofL Superfund Research Center, and ACORNS/TREE research translation groups.

Center for Healthy Air, Water and Soil
(CHAWS) Membership Application Form
(Please submit Electronically)

General Profile

Date of Application: 4/23/2018

Name (first, middle, last):

Rachel J Keith

Degree(s): Faculty Appointment:
Medicine

PhD, MSN, ANP-C. Assistant Professor of

Admin Titles (if applicable):

Research Expertise/Field:
exposures and heart disease

Clinician scientist in environmental

Campus address:
Louisville, KY 40202

580 S. Preston St. Delia Baxter Rm 321C

Telephone number

502-852-4211

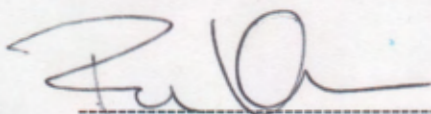
Fax number:

502-852-3663

e-mail address:

Rachel.keith@louisville.edu

Signature



Please describe your current Research Interests:

My specific training as a scientist and a nurse practitioner are ideal for correlating environmental exposures and health outcomes. My research and clinical experience has looked at prevention of cardiovascular disease and novel identifiers of early disease. I have participated in numerous clinical trials and NIH or industry sponsored investigator initiated human studies as well. As a new investigator or coinvestigator on several cross-sectional human studies I have developed and compiled effective tools for evaluating perceptions of health, cardiovascular health, exposure to hazardous chemicals, greenness exposure, e-cigarettes, and biomarkers of injury. I have demonstrated my ability to recruit participants for a wide range of studies. Furthermore, I have successfully managed studies including staffing, budget, ordering, testing, regulatory oversight, and data collection. I have collaborated with multi-disciplinary researchers to measure a wide variety of biomarkers. I am highly motivated to pursue human studies and took off almost 3 years from traditional research to train in clinical care and studies.

**Center for Healthy Air Water and Soil
(CHAWS) Membership Application Form**

(Please submit Electronically)

General Profile

Date of Application: 4/24/18

Name (first, middle, last): Timothy E. O'Toole _____

Degree(s): Faculty Appointment: Ph.D. Assistant Professor _____

Admin Titles (if applicable): _____

Research Expertise/Field: Environmental Cardiology _____

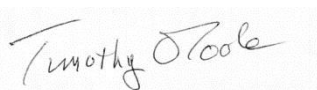
Campus address: 207E Baxter II _____

Telephone number: 502-852-5886 _____

Fax number: 502-852-3663 _____

e-mail address: tim.otoole@louisville.edu

Signature



Please describe your current Research Interests:

In my research h career, I have used a variety of cellular and molecular approaches, and both human and animal models, to study mechanisms underlying cardiovascular disease. My current interests focus on the under-studied and unappreciated role of environmental exposures on cardiovascular outcomes. In prior studies, we characterized the effects of exposure to airborne particulate matter (PM_{2.5}) in humans and mice. We found that exposure led to alterations in circulating pro-angiogenic cells, increases in endothelial cell death, increases in inflammatory cell types, increases in anti-angiogenic cytokines, and a decrease in growth factors and pro-angiogenic cytokines. Similarly, mice exposed to concentrated ambient particles (CAPs) also had altered immune cell populations and demonstrated additional defects in hematopoiesis, insulin signaling and stem cell function as well. As PM_{2.5} exposures do not occur in isolation but are often in combination with volatile organics (VOCs), we have also extended our studies to examine effects of inhalation exposure to acrolein and benzene in mice. Again, we have observed several adverse outcomes resulting from direct exposure to these two VOCs. Ongoing, studies will attempt to identify a mechanistic basis for these outcomes and explore the efficacy of anti-oxidant interventions in mitigating the effects of PM_{2.5} and VOC exposure. Thus my background and research interests blend nicely with the overall goals CHAWS.

April 27, 2018

Connie Shumake
Office of the Provost
University of Louisville
Louisville, KY 40292

Connie,

We have been asked to provide a letter of support for the proposed Center for Healthy Air Water and Soil (CHAWS). I am attaching the review completed by the Kornhauser Health Sciences Library for this purpose.

The review indicates that no additional resources or library materials expenditures should be required to support the Center unless the program requires that specific resources be added to the library collections. We welcome any suggestions to help develop library collections and services. Please contact us if you have any questions or need additional information.

Sincerely,



Robert E. Fox, Jr.
Dean and Professor
University Libraries

Cc: Kevyn Merten
Neal Nixon

Review of Library Materials and Support for the proposed Center for Healthy Air Water and Soil (CHAWS)

April 27, 2018

Over the years, the Kornhauser Health Sciences Library (KHSL) staff has developed a collection of full-text journals, books, databases and evidence-based resources to support environmental research forming a core of information resources that will also support this program.

UL Libraries subscribes to a very diverse collection of both print and e-resources which includes thousands of books and journals. Since the Center's demands cover a broad spectrum of topics, resources from both the KHSL and the Ekstrom Library would need to be used. UL Libraries has access to approximately 8,752 ecology titles, 5,000 climate titles, 2,089 titles dealing with water quality, 4,422 titles related to pollution, and 1,089 titles focusing on environmental protection. *Clean: Soil, Air, Water* is an example of an electronic journal title that is an available library resource. UL Libraries also host 327 databases, approximately 35 of those databases are related to the various areas that CHAWS focuses on, including health sciences, communications, business, economics, psychology, government, law, toxicology, and public health. Some available databases include: Agricola, GreenFILE, Environmental Engineering Abstracts, Emerald Library, American Chemical Society Web Edition, BIOSIS, Health and Safety Sciences Abstracts, IEEE Xplore, Web of Science, JSTOR, EBSCO Web, Toxnet, MEDLINE/PubMed, Nature Protocols and SciFinder.

All University faculty, students, and researchers may access any electronic resources 24/7 from their home or office. If physically coming to one of the UL Libraries is not an option, the Document Delivery Service can electronically send an article or book chapter from a library-owned resource to a requestor's office or home. For resources not held by any of the UL Libraries, interlibrary loan (ILL) services are available free-of-charge to any University student, staff, or faculty. The ILL office can borrow almost any item published from any library in the world, either in the original format, or as a photocopy.

The staff of the UL Libraries also facilitate the understanding and use of the resources. To foster a supportive learning environment, the staff offers extensive reference assistance, helping users to formulate online search strategies, validate citations, and locate materials. KHSL librarian liaisons provide in-depth consultation and training sessions by appointment for faculty, students, and residents; classroom instruction is available by request. Additionally, Ekstrom Library's STEM librarian and liaison librarians to the Communications, Social Work, and Business departments may also serve as a resource to the CHAWS program.

April 24, 2018

Aruni Bhatnagar, Ph.D., FAHA
Professor of Medicine
Smith and Lucille Gibson Chair in Medicine
Director, Diabetes and Obesity Center
University of Louisville

Re: Creation of the Center for Healthy Air, Water and Soil (CHAWS)

Dear Dr. Bhatnagar:

We are delighted to strongly support your proposal for the creation of the Center for Healthy Air, Water and Soil (CHAWS), which will be housed within the University of Louisville Envirome Institute and under the oversight of the Office of the Executive Vice President for Research and Innovation (EVPRI) and the Executive Vice President for Health Affairs. Environmental health is becoming a major research priority of the University of Louisville and you have created a strong foundation with the development of the Diabetes and Obesity Center and the Superfund Research Program, which are both supported with substantial extramural funding from the National Institutes of Health. You are also now creating the multi-disciplinary Envirome Institute to coordinate environmental health related research at the University, and the CHAWS will play a pivotal role in enhancing research, educational and community engagement activities that are essential to the success of these endeavors.

As you note in your Center proposal, the CHAWS will be supported by a \$2.5M philanthropic gift (\$500,000/year for 5 years). This gift will be used to support programming and personnel focused on community outreach and engagement that will connect UofL faculty with the local community and help to advance the research mission and international reputation of the University. Funding will also support the educational activities of UofL students and provide for new innovative research opportunities in the area of environmental health and welfare. These activities will be supported by the outstanding multi-disciplinary team of faculty that you have assembled to help seed and propel the mission of the CHAWS into the future.

We are very pleased to offer our strong and enthusiastic support to the CHAWS and to your proposal. We wish you great success with this Center and look forward to the contributions that CHAWS will make to environmental health in both our local community and the nation.

Sincerely yours,



Robert S. Keynton, Ph.D.
Interim Executive VP for Research and Innovation
Professor and Lutz Endowed Chair
Department of Bioengineering
Fellow – AIMBE
Fellow – National Academy of Inventors



Gregory C. Postel, M.D.
Executive VP for Health Affairs

List all current employees paid from the center/institute budget (faculty, staff, graduate asst. or other temp. employees.)				
1.Full-time Faculty (FTE)				
Name	Full-time Equivalent (FTE)	<u>Status</u> Perm. or Temp.	Total Salary	Salary paid by the program/center/institute
N/A				
2. Part-Time Faculty (PTE)				
Name	Full-time Equivalent (FTE)	<u>Status</u> Perm. or Temp.	Total Salary	Salary paid by the program/center/institute
N/A				
Fellows				
Name	Full-time Equivalent (FTE)	<u>Status</u> Perm. or Temp.	Total Salary	Salary paid by the program/center/institute
N/A				
4. Staff Support (SS)				
Name	Full-time Equivalent (FTE)	<u>Status</u> Perm. or Temp.	Total Salary	Salary paid by the program/center/institute
N/A				
Note: the total FTE and salary amounts should be equal to the personnel cost information listed in the departmental expenditures.				



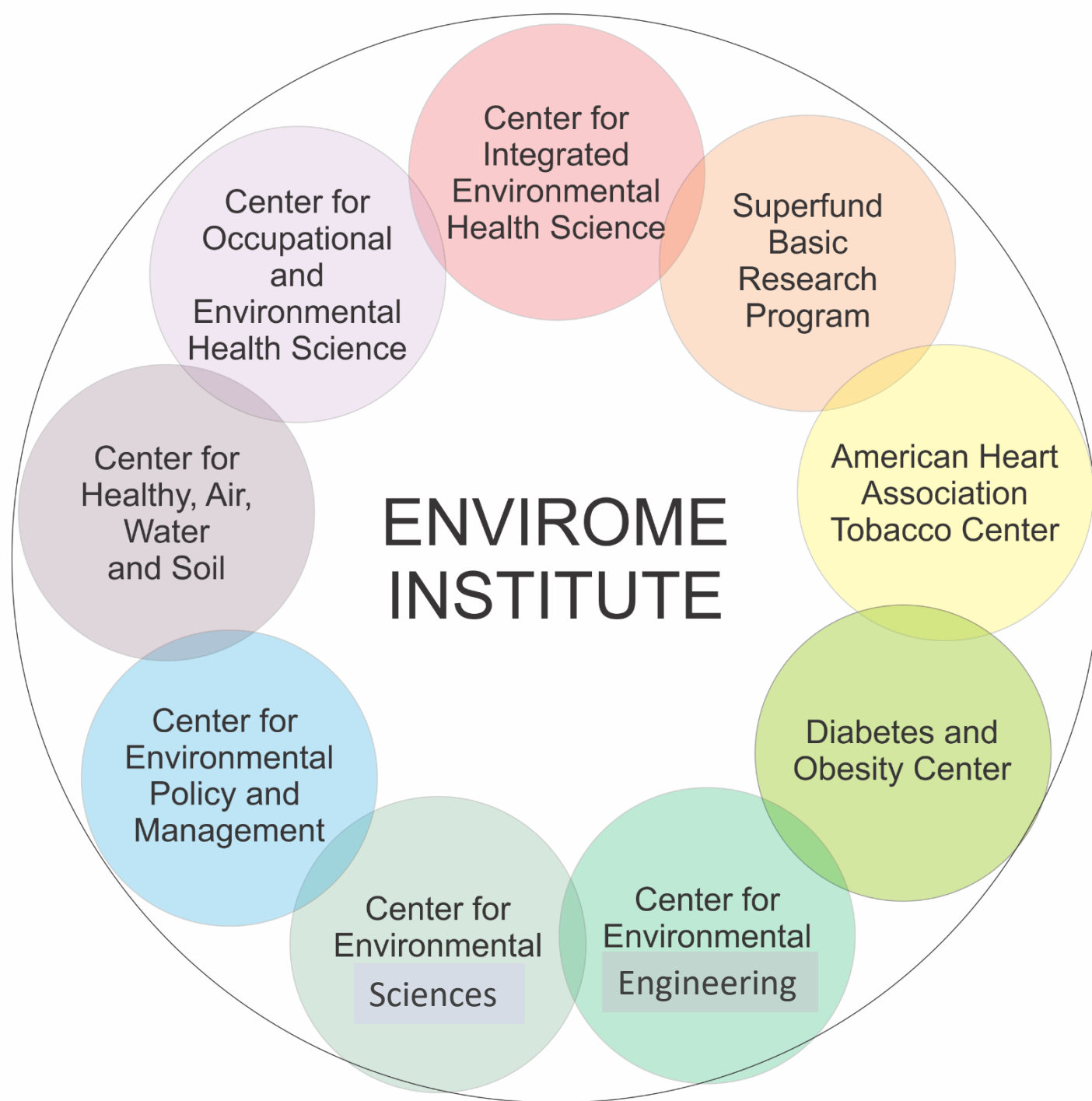
**CENTER FOR HEALTHY
AIR WATER & SOIL**

MISSION

To support and promote innovative, environmental research and project focused on health in all policies using the city of Louisville as an urban laboratory

To improve the health and harmony of our community by empowering citizens and using inventive technology

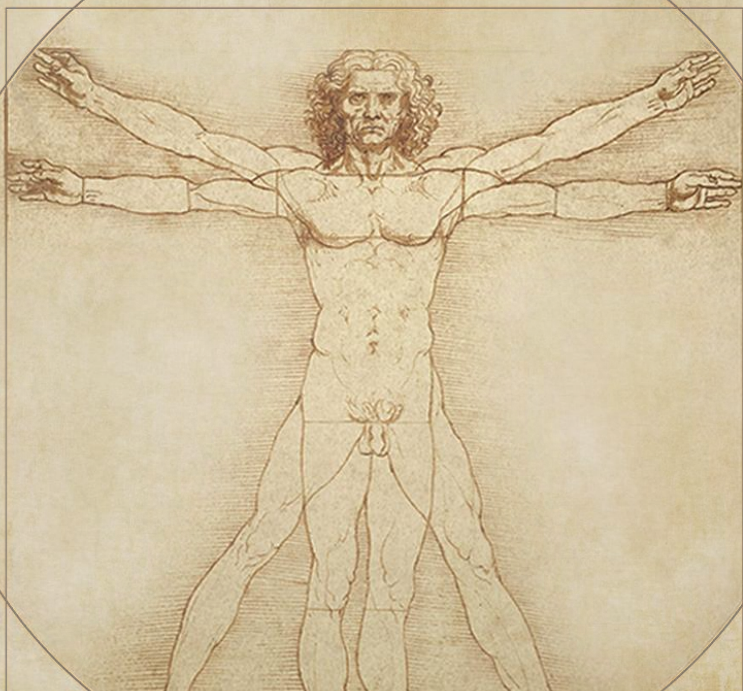
To encourage new conversations and find new solutions to health challenges in our community and our world




NATURAL ENVIRONMENT

SOCIAL ENVIRONMENT

PERSONAL ENVIRONMENT

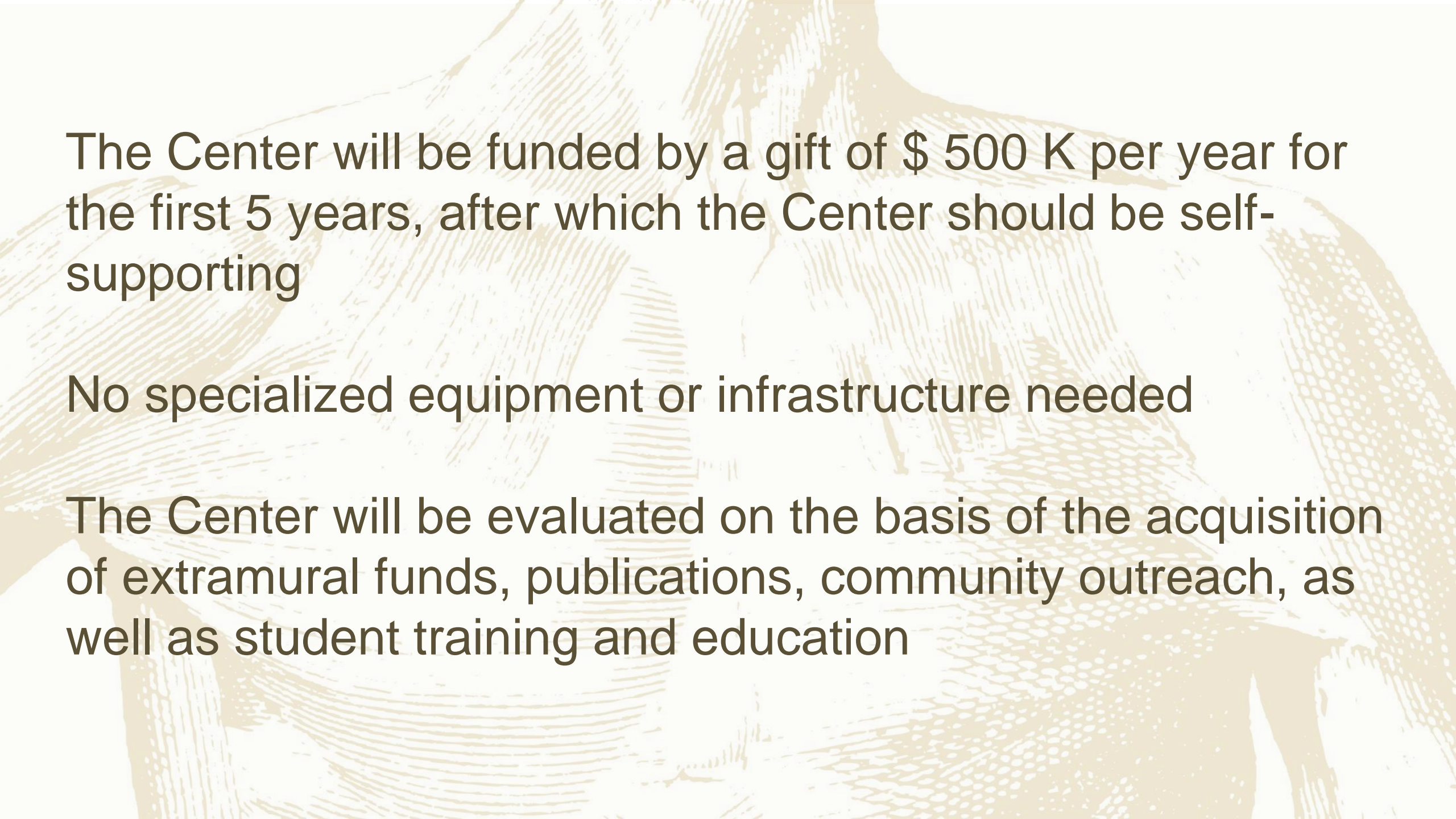


The background features a detailed anatomical illustration of a human torso, showing the ribcage, spine, and internal organs. Overlaid on this illustration are various handwritten-style letters and numbers, such as '72', '9', 'd', '10', '7', and 'A', which appear to be part of a scientific or medical study. The overall color palette is a warm, golden-brown hue.

The Center will bring together faculty with diverse interests and backgrounds as well as community members and leaders to collectively bear upon local environmental problems and issues

The Center will facilitate and encourage environmental research and scholarship by partnering with faculty and students across campus and members of the local community

It will support the University's mission of achieving preeminence as a nationally recognized metropolitan research university



The Center will be funded by a gift of \$ 500 K per year for the first 5 years, after which the Center should be self-supporting

No specialized equipment or infrastructure needed

The Center will be evaluated on the basis of the acquisition of extramural funds, publications, community outreach, as well as student training and education