BIOGRAPHICAL SKETCH

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NAME: Waltz, Susan E

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

POSITION TITLE: Professor of Cancer Biology; Associate Director for Cancer Research Education and Coordination, University of Cincinnati Cancer Center

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
Youngstown State University, Youngstown, OH	BS	1989	Biology/Chemistry
Wright State University, Boonshoft School of Medicine, Dayton, OH	PhD	1994	Biomedical Sciences/Biochemistry
Cincinnati Children's Hospital, Cincinnati, OH	Fellowship	1997	Developmental Biology

A. Personal Statement

I am the Associate Director (AD) for Cancer Research Training and Education Coordination at the University of Cincinnati (UC) Cancer Center (UCCC) and a tenured Professor in the Department of Cancer Biology at the UC College of Medicine The goal of CRTEC is to coordinate a wide range of cancer related educational initiatives that cover the spectrum of academic and professional training. CRTEC includes efforts that stand the spectrum of learners from the pre-collegiate space through early-stage investigators. In addition to CRTEC, I am the director of the Graduate (PhD and MS) Program in Cancer and Cell Biology (CCB) at the University of Cincinnati. The mission of the CCB Program is to train the next generation of scientist and educators in basic mechanisms of molecular and cellular function with an emphasis on understanding mechanism of cancer. The program current has 51 trainees (45 PhD and 6 MS students) with 10 degrees (6 PhD and 4 MS) granted in FY24.

During my career as a faculty member, I have had numerous and substantive mentoring experiences. Specifically, with respect to training, I have been the primary mentor for 2 foreign exchange students, 10 high school students, 42 undergraduate students, 1 PREP scholar, 21 graduate students, 24 postdoctoral fellows and 8 junior faculty. I have also served on over 50 graduate student thesis committees and qualifying exam committees. With respect to mentorship on funded fellowship applications, I have served as a mentor for my pre and post-doctoral students on successful individual fellowship awards from the DOD (3), IGERT Award/NSF (1), NIH K Awards (4), NRSA F30/F31 (4), BIRCWH Scholar Awards/NIH (2), American Cancer Society (2), American Liver Foundation Postdoctoral Fellowship Award (1), VA Career Development Award (1) and the Children's Digestive Health and Nutrition Award (1). In addition to mentoring my trainees on these awards, I have also formally reviewed fellowship applications for the American Association for Cancer Research (AACR), the American Cancer Society (National Chapter), the National Cancer Institute (T32 and K awards) and for the Department of Defense Breast Cancer Research Program. Thus, I have a successful track record of both mentoring and reviewing pre and postdoctoral fellowship applicants. With respect to my trainees' success, recent predoctoral students who have graduated from my laboratory are in postdoctoral fellowship positions at Eli Lilly, Yale University, MedPace (a large clinical trial company) and at Cincinnati Children's Hospital Medical Center. The postdoctoral fellows that have worked in my laboratory have also gone on to successful scientific careers and have research related faculty positions at University of Alabama Birmingham (Department Chair), Thomas Jefferson University, Case Western Reserve University, Stony Brook University, UC Blue Ash/Raymond Walters College (Department Chair), Cincinnati Children's Medical Center, the University of Cincinnati (Dept. of Environmental Health; Dept Surgery; Dept of General Medicine-Surgery; Education Department) and one as the Clinical Research Medical Director at Amgen. I also had several trainees who obtained additional degrees: one is a certified professional scientific writer, one is an intellectual property lawyer for Amgen, and one obtained a Pharmacy degree and is working at a regional hospital.

A long-term goal of my research program has been to investigate the functional roles of cell surface receptor tyrosine kinases and their ligands in hormonally regulated cancers. Specifically, my laboratory has focused on the involvement of the RON receptor tyrosine kinase and its ligand hepatocyte growth factor-like protein (HGFL) in inflammation and cancer. Our laboratory was the first to demonstrate that the RON receptor plays an important role in breast cancer and were the first to show that 1) RON overexpression is sufficient to promote the development of aggressive breast cancers in mice, 2) RON is highly expressed in primary breast cancers and is highly associated with metastasis, 3) RON promotes tumor vascularization though the production of angiogenic chemokines, 4) loss of RON in a murine model of breast cancer leads to diminished tumor growth, and 5) RON expression in tumor-associated macrophages promotes tumor growth in mice. In the process of these studies, we have developed novel reagents including conditional RON and HGFL deficient mice, overexpressing mice, and modulated cell lines, as well as have utilized a variety of model systems including orthotopic transplantation into the breasts of syngeneic mice, xenografts of human cell lines, and human tissue research. I have the expertise, leadership, and motivation necessary to successfully carry out funded studies. I have a broad background in biochemistry and molecular biology. As a PI on several NIH, VA, and other funded research projects, my laboratory has published hallmark papers demonstrating that RON overexpression is sufficient to induce tumor formation in murine models and we have translated these studies to analyses on human tumor specimens. In addition, I have successfully administered projects (i.e. staffing, research protections, ethics considerations, and budgets), collaborated with other investigators, and produced several peer-reviewed publications from each project.

I also recently completed an 8-hr mentor training based on based on the evidence-based Entering Mentoring curriculum from the Center for the Improvement of Mentored Experiences in Research (CIMER), which has been shown to positively impact mentorship skills and knowledge. Training facilitated by the University of Cincinnati College of Medicine, Office of Graduate Education, on April 29, 2025.

Ongoing and recently completed projects that I would like to highlight include:

2I01BX000803-11A1
Waltz (PI)
10/01/2010 - 09/30/2027
Ron Receptor Tyrosine Kinase Signaling in Breast Cancer

R01 CA239696
Waltz (M-PI)
04/01/20 - 08/31/2026; Renewal under review
Defining genetic and metabolic requirements of aggressive breast cancer

NCI T32CA298951-01
Waltz (M-PI)
07/01/2025 - 06/31/2030
Training Program in Basic and Translational Oncology

NCI T32CA117846
Waltz (M-PI)
09/01/2006 - 08/31/2024
Training Program in Cancer Therapeutics

Citations

1. Ruiz-Torres SJ, Bourn JR, Benight NM, Hunt BG, Lester C, **Waltz SE**. Macrophage-mediated RON signaling supports breast cancer growth and progression through modulation of IL-35. *Oncogene*. 2022; 41 (3): 321-333. PMCID: PMC8758553.
2. Oropeza E, Seker S, Carrel S, Mazumder A, Lozano D, Jimenez A, VandenHeuvel SN, Noltensmeyer DA, Punturi NB, Lei JT, Lim B, **Waltz SE**, Raghavan SA, Bainbridge MN, Haricharan S. Molecular portraits of cell cycle checkpoint kinases in cancer evolution, progression, and treatment responsiveness. *Sci Adv*. 2023; 9 (26): eadf2860. PMCID: PMC10313178.

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

2024	Ad Hoc Reviewer, NCI Transition Career Development Award, Institutional Research Training Grants and Cancer Research Education Grants Program Peer-Review Study Section
2023 - Present	Associate Director, Cancer Research Training Education Coordination, University of Cincinnati Cancer Center, Cincinnati, OH
2023 - 2024	Reviewer, VA Merit Review, ONCA Subcommittee Study Sections
2021 - Present	Member, University of Cincinnati Cancer Center, Cincinnati, OH
2021 - 2024	Ad Hoc Reviewer, NIH/NIGMS SuRE R16 Program Review Study Section
2018 - Present	Director, Graduate Program in Cancer and Cell Biology, University of Cincinnati, Cincinnati, OH
2014 - 2020	Standing Member, NIH Cancer Molecular Pathobiology (CAMP) Study Section
2013 - 2016	Standing Member, American Association for Cancer Research (AACR) Postdoctoral Fellowship Award Review Committee
2010 - Present	Investigator, Research Service, Cincinnati Veteran's Administration Medical Center
2009 - Present	Professor with Tenure, Department of Cancer Biology, University of Cincinnati College of Medicine, Cincinnati, OH
2008 - 2012	Director, Graduate Program in Cancer and Cell Biology, University of Cincinnati, Cincinnati, OH
2008 - 2012	Standing Member, NIH Hepatobiliary Pathophysiology (HBPP) Study Section
2007 - 2009	Interim Scientific Director, University of Cincinnati Barrett Cancer Center/Joint Cancer Program, Cincinnati, OH
2006 - 2020	Ad Hoc Reviewer, NIH HBPP; NIH CAMP; NIH Tumor Progression and Metastasis (TPM); NIH Provocative Questions, and Cell Biology Special Emphasis Panel Study Sections; Health Research Board Ireland, Women's Research Institute
2005 - 2008	Faculty Consultant, Shriners Hospital for Children, Cincinnati, OH
2005 - 2008	Director, Oncology Research Program, Department of Surgery, University of Cincinnati, Cincinnati, OH
2004 - 2007	Member/Reviewer, 2004 Congressionally Directed Medical Research Programs (CDMRP) 2004 Breast Cancer Research Program - Pathobiology #3; 2005 CDMRP Concept Award, Peer Review Panel, Pathobiology #1; 2006 CDMRP Cell Biology Concept Review Panel #3; 2007 CDM
2003 - 2009	Associate Professor with Tenure, Departments of Surgery and Cancer and Cell Biology, University of Cincinnati College of Medicine, Cincinnati, OH
2002 - Present	Faculty Member, Graduate Program in Cancer and Cell Biology
2000 - Present	Faculty Member, Physician Scientist Training Program
1999 - 2004	Standing Member, National American Cancer Society (ACS), Cell Cycle and Growth Control Study Section
1997 - 2003	Assistant Professor of Pediatrics, Division of Developmental Biology, Children's Hospital Medical Center, Cincinnati, OH

Honors

2021	University of Cincinnati College of Medicine Gratitude Award Recipient
2020	Chair, Symposium on the Tumor Microenvironment (Virtual), Co-hosted by the Departments of Cancer Biology and Hematology/Oncology, University of Cincinnati
2016	Co-Chair, National Jensen Symposium on Breast Cancer, University of Cincinnati
2016	Career Research Service Award, University of Cincinnati

2015	University of Cincinnati, Research Cabinet Member
2014	Cincinnati Cancer Center, Internal Advisory Board Member
2011	US Chamber of Commerce Life Sciences Award Finalist
2011	Session Chair, Molecular Oncology on Breast Cancer, BIT's 4th Annual World Cancer Congress 2011
2010 - Present	Elected Member, Graduate Faculty Fellow, University of Cincinnati
2008	Chairperson, AACR Mini symposium on Cell surface receptors in cancer initiation and progression, AACR Annual Meeting 2008, San Diego, CA
2002 - 2007	Career Development Award, Department of Defense, October 2002-2007
1999	Basil O'Connor Scholar Research Award, March of Dimes.
1998	Scientist Development Award, Career Development Award, American Heart Association-National Center
1996 - 1999	National Research Service Award (NRSA), Postdoctoral Fellowship, National Institutes of Health (DK09466). (Returned 6/1/97 due to Faculty Appointment).

C. Contributions to Science

1. **Breast Tumorigenesis** Our contribution to HGFL-RON signaling in breast cancer include seminal work wherein we were the first to demonstrate that RON participates in breast tumorigenesis and overexpression is sufficient for the generation of aggressive and metastatic breast cancer in mice. We further demonstrated that RON targets β-catenin signaling, Dek signaling and immune sensing pathways as important downstream effectors of breast tumorigenesis. We also provided data demonstrating that RON activation promotes chemotherapeutic resistance.
    a. Bourn JR, Ruiz-Torres SJ, Hunt BG, Benight NM, **Waltz SE**. Tumor cell intrinsic RON signaling suppresses innate immune responses in breast cancer through inhibition of IRAK4 signaling. *Cancer Lett*. 2021; 50375-90. PMCID: PMC7981256.
    b. Ruiz-Torres SJ, Bourn JR, Benight NM, Hunt BG, Lester C, **Waltz SE**. Macrophage-mediated RON signaling supports breast cancer growth and progression through modulation of IL-35. *Oncogene*. 2022; 41 (3): 321-333. PMCID: PMC8758553.
    c. Hunt BG, Wicker CA, Bourn JR, Lower EE, Takiar V, **Waltz SE**. MST1R (RON) expression is a novel prognostic biomarker for metastatic progression in breast cancer patients. *Breast Cancer Res Treat*. 2020; 181 (3): 529-540. PMCID: PMC7255851.
    d. Hunt BG, Davis JC, Fox LH, Vicente-Muñoz S, Lester C, Wells SI, **Waltz SE.** Oncogene. 2023 RON-augmented cholesterol biosynthesis in breast cancer metastatic progression and recurrence.May;42(21):1716-1727. doi: 10.1038/s41388-023-02688-5. Epub 2023 Apr 7. PMID: 37029299.
2. **Breast Development** In addition to breast cancer, my laboratory has established expertise in the study of mammary gland development. We were the first to identify the expression and function of RON and HGFL in the mammary gland and on functions in mammary gland branching morphogenesis. We have also published work on examination of vitamin D receptor functions in both the mammary epithelium as well as in adipose tissue using clinically relevant murine models.
    a. Oropeza E, Seker S, Carrel S, Mazumder A, Lozano D, Jimenez A, VandenHeuvel SN, Noltensmeyer DA, Punturi NB, Lei JT, Lim B, **Waltz SE**, Raghavan SA, Bainbridge MN, Haricharan S. Molecular portraits of cell cycle checkpoint kinases in cancer evolution, progression, and treatment responsiveness. Sci Adv. 2023 Jun 30;9(26):eadf2860. doi: 10.1126/sciadv.adf2860. Epub 2023 Jun 30. PMID: 37390209
    b. Welsh J, Zinser LN, Mianecki-Morton L, Martin J, **Waltz SE**, James H, Zinser GM. Age-related changes in the epithelial and stromal compartments of the mammary gland in normocalcemic mice lacking the vitamin D3 receptor. *PLoS One*. 2011; 6 (1): e16479. PMCID: PMC3027678.
    c. Gurusamy D, Ruiz-Torres SJ, Johnson AL, Smith DA, **Waltz SE**. Hepatocyte growth factor-like protein is a positive regulator of early mammary gland ductal morphogenesis. *Mech Dev*. 2014; 13311-22. PMCID: PMC4177297.
    d. Johnson AL, Zinser GM, **Waltz SE**. Loss of vitamin D receptor signaling from the mammary epithelium or adipose tissue alters pubertal glandular development. *Am J Physiol Endocrinol Metab*. 2014; 307 (8): E674-85. PMCID: PMC4200307.

3. **Microenvironmental Factors and Tumorigenesis** A central finding my laboratory demonstrated was the critical importance of RON signaling in the tumor microenvironment. Our studies showed that myeloid RON signaling is sufficient to dramatically inhibit tumor growth though mechanisms that are associated with activation of the adaptive immune response. This line of research has opened exciting new avenues of research into RON signaling as a key modulator of tumor immunity with significant implications for future tumor targeting.
    a. Eyob H, Ekiz HA, Derose YS, **Waltz SE**, Williams MA, Welm AL. Inhibition of ron kinase blocks conversion of micrometastases to overt metastases by boosting antitumor immunity. *Cancer Discov*. 2013; 3 (7): 751-60. PMCID: PMC3710539.
    b. Gurusamy D, Ruiz-Torres SJ, Johnson AL, Smith DA, **Waltz SE**. Hepatocyte growth factor-like protein is a positive regulator of early mammary gland ductal morphogenesis. *Mech Dev*. 2014; 13311-22. PMCID: PMC4177297.
    c. Benight NM, Wagh PK, Zinser GM, Peace BE, Stuart WD, Vasiliauskas J, Pathrose P, Starnes SL, **Waltz SE**. HGFL supports mammary tumorigenesis by enhancing tumor cell intrinsic survival and influencing macrophage and T-cell responses. *Oncotarget*. 2015; 6 (19): 17445-61. PMCID: PMC4627320.
    d. Pease NA, Shephard MS, Sertorio M, **Waltz SE**, Vinnedge LMP. DEK Expression in Breast Cancer Cells Leads to the Alternative Activation of Tumor Associated Macrophages. *Cancers (Basel)*. 2020; 12 (7): PMCID: PMC7409092.

4. **Prostate Tumorigenesis** Our contributions to prostate cancer include the first demonstration of RON and HGFL overexpression in human and murine prostate cancer as well as the demonstration that this signaling cascade is functionally important for prostate tumor development. We also showed that epithelial RON signaling is required to promote tumor angiogenesis through the production of angiogenic chemokines which stimulate CXCR2 mediated endothelial cell migration.
    a. Vasiliauskas J, Nashu MA, Pathrose P, Starnes SL, **Waltz SE**. Hepatocyte growth factor-like protein is required for prostate tumor growth in the TRAMP mouse model. *Oncotarget*. 2014; 5 (14): 5547-58. PMCID: PMC4170603.
    b. Brown NE, Paluch AM, Nashu MA, Komurov K, **Waltz SE**. Tumor Cell Autonomous RON Receptor Expression Promotes Prostate Cancer Growth Under Conditions of Androgen Deprivation. *Neoplasia*. 2018; 20 (9): 917-929. PMCID: PMC6098205.
    c. Sullivan C, Brown NE, Vasiliauskas J, Pathrose P, Starnes SL, **Waltz SE**. Prostate Epithelial RON Signaling Promotes M2 Macrophage Activation to Drive Prostate Tumor Growth and Progression. *Mol Cancer Res*. 2020; 18 (8): 1244-1254. PMCID: PMC7415572.
    d. Brown NE, Jones A, Hunt BG, **Waltz SE**. Prostate tumor RON receptor signaling mediates macrophage recruitment to drive androgen deprivation therapy resistance through Gas6-mediated Axl and RON signaling. *Prostate*. 2022; 82 (15): 1422-1437. PMCID: PMC9492645.

5. **Microenvironment Interactions, Inflammation and Acute Injury** Our early studies on RON receptor signaling showed that a major function of RON and HGFL are to suppress immune responses following acute and chronic insults. These studies paved the way to our understanding of RON signaling as mediator of macrophage responses *in vivo*.
    a. Stuart WD, Kulkarni RM, Gray JK, Vasiliauskas J, Leonis MA, **Waltz SE**. Ron receptor regulates Kupffer cell-dependent cytokine production and hepatocyte survival following endotoxin exposure in mice. *Hepatology*. 2011; 53 (5): 1618-28. PMCID: PMC3082400.
    b. Hunt BG, Jones A, Lester C, Davis JC, Benight NM, **Waltz SE.** RON (MST1R) and HGFL (MST1) Co-Overexpression Supports Breast Tumorigenesis through Autocrine and Paracrine Cellular Crosstalk. Cancers (Basel). 2022 May 19;14(10):2493. doi:10.3390/cancers14102493.
    c. Kulkarni RM, Stuart WD, Gurusamy D, **Waltz SE**. Ron receptor signaling is protective against DSS-induced colitis in mice. *Am J Physiol Gastrointest Liver Physiol*. 2014; 306 (12): G1065-74. PMCID: PMC4059975.
    d. Stuart WD, Brown NE, Paluch AM, **Waltz SE**. Loss of Ron receptor signaling leads to reduced obesity, diabetic phenotypes and hepatic steatosis in response to high-fat diet in mice. *Am J Physiol Endocrinol Metab*. 2015; 308 (7): E562-72. PMCID: PMC4385874.

Complete List of Published Work in MyBibliography:
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