

NIH BIOGRAPHICAL SKETCH COMMON FORM

Name: Toland, Amanda Ewart

Persistent Identifier (PID) of the Senior/Key Person: <https://orcid.org/0000-0002-0271-1792>

Position Title: Professor & Interim Chair

Organization and Location: The Ohio State University, Columbus, Ohio, United States

PROFESSIONAL PREPARATION

INSTITUTION AND LOCATION	DEGREE	Start Date	Completion Date	FIELD OF STUDY
University of California, San Francisco, San Francisco, CA, US	Postdoctoral Fellow	07/1999	06/2002	Cancer Genetics
University of California, San Francisco, San Francisco, CA, United States	Postdoctoral Fellow	07/1996	06/1999	Fellowship in PhD Medical Genetics
University of Utah, Salt Lake City, UT, United States	DOCTOR OF PHILOSOPHY	09/1990	05/1996	Human Genetics
Franklin & Marshall College, Lancaster, PA, USA	BACHELOR OF ARTS	08/1986	05/1990	Biology

Appointments and Positions

2020 - present	Professor & Interim Chair, The Ohio State University, Columbus, Ohio, United States
2024 - present	Interim Chair, Department of Cancer Biology and Genetics, The Ohio State University, Columbus, Ohio, United States
2024 - present	Director CAMELOT (Center for Cancer Mentoring, Education, Leadership and Oncology-Related Training), The Ohio State University Comprehensive Cancer Center, Columbus, Ohio, United States
2024 - present	Associate Director Cancer Research Training and Education Coordination (CRTEC), The Ohio State University Comprehensive Cancer Center, Columbus, Ohio, United States
2022 - 2025	Cancer and Hematological Disease Study Section Member, National Institutes of Health, Bethesda, Maryland, United States
2021 - 2022	ad hoc member SPORE Review Study Section, National Cancer Institute, Bethesda, Maryland, United States
2021 - 2021	Ad hoc member Secondary & Genomic Data Analysis ZDE1 NB03 study section, National Institute of Dental and Craniofacial Research, Bethesda, Maryland, United States
2020 - present	Professor, The Ohio State University, Department of Cancer Biology and Genetics and Department of Internal Medicine, Division of Human Genetics, Columbus, OH, USA
2020 - 2024	Vice Chair, Department of Cancer Biology and Genetics, The Ohio State University, Columbus, Ohio, United States
2020 - 2022	Cancer, Heart, and Sleep Epidemiology Panel B (CHSB) study section, National Institutes of Health, Bethesda, Maryland, United States
2020 - 2021	Reviewer Cancer Epidemiology & Prevention Award Selection Committee, American Association of Cancer Research, Philadelphia, Pennsylvania, United States
2019 - present	Member ClinVar Variant Expert Curation Panel BRCA1/BRCA2, National Institutes of Health, Bethesda, Maryland, United States
2018 - present	Data Gathering Group Member, BRCA Exchange, San Jose, California, United States
2018 - 2019	Provocative Questions Study Section, National Cancer Institute, Bethesda, Maryland, United States
2018 - 2018	Clinical Translational R21 Study Section ZCA1 SRB K 01, National Cancer Institute, Bethesda, Ohio, United States
2018 - 2018	Member Study Section ZES1 LWJ-S, National Institute of Environmental Health and Safety, Durham, North Carolina, United States

2017 - present	Associate Editor Translational Sciences, British Journal of Dermatology, Oxford, Not Applicable, N/A, United Kingdom
2017 - present	Director Genomics Shared Resource, The Ohio State University Comprehensive Cancer Center, Columbus, Ohio, United States
2016 - 2017	co-Chair Tumor Biology and Genomics Study Section, American Cancer Society, Atlanta, Georgia, United States
2012 - 2018	Breast Information Core (BIC) Steering Committee and Chair, NHGRI, Bethesda, Maryland, United States
2011 - 2020	Associate Professor, The Ohio State University, Departments of Cancer Biology and Genetics and the Department of Internal Medicine, Columbus, OH, USA
2010 - 2025	Member, PDQ Cancer Genetics Editorial Board, National Cancer Institute, Shady Grove, Maryland, United States
2008 - 2020	Academic Editor, PLoS One, San Francisco, California, United States
2005 - 2011	Assistant Professor, Assistant Professor, The Ohio State University, Department of Molecular Virology, Immunology and Medical Genetics, Division of Human Cancer Genetics, Columbus, OH, USA
2005 - 2011	Assistant Professor, The Ohio State University, Department of Internal Medicine, Division of Human Genetics, Columbus, OH, USA
2004 - 2005	Interim Laboratory Director, Genome Analysis Core, UCSF Comprehensive Cancer Center, San Francisco, CA, USA
2002 - 2005	Assistant Researcher, UCSF Comprehensive Cancer Center, San Francisco, CA, USA
2001 - 2005	Manager, Familial Risk Shared Resource, UCSF Comprehensive Cancer Center, San Francisco, CA, USA
2000 - 2001	Consultant, Genetic Health, San Francisco, California, United States
1999 - present	Fellow, American College of Medical Genetics, Bethesda, Maryland, United States
1999 - 2001	Postgraduate Researcher, UCSF Comprehensive Cancer Center, San Francisco, CA, USA
1998 - 2005	Medical Genetic Consultant, UCSF Cancer Risk Program, San Francisco, CA, USA
1996 - 1999	Fellow, UCSF Division of Medical Genetics, San Francisco, CA, USA
1996 - 1999	Postdoctoral Fellow, UCSF, Department of Laboratory Medicine, San Francisco, CA, USA

Products

Products Closely Related to the Proposed Project

1. Tjader NP, Beer AJ, Ramroop J, Tai MC, Ping J, Gandhi T, Dauch C, Neuhausen SL, Ziv E, Sotelo N, Ghanekar S, Meadows O, Paredes M, Gillespie JL, Aeilts AM, Hampel H, Zheng W, Jia G, Hu Q, Wei L, Liu S, Ambrosone CB, Palmer JR, Carpten JD, Yao S, Stevens P, Ho WK, Pan JW, Fadda P, Huo D, Teo SH, McElroy JP, Toland AE. Association of ESR1 Germline Variants with TP53 Somatic Variants in Breast Tumors in a Genome-wide Study. Cancer Res Commun. 2024 Jun 27;4(6):1597-1608. PubMed Central PMCID: [PMC11210444](#).
2. Pollock NC, Ramroop JR, Hampel H, Troester MA, Conway K, Hu JJ, Freudenheim JL, Olopade OI, Huo D, Ziv E, Neuhausen SL, Stevens P, McElroy JP, Toland AE. Differences in somatic TP53 mutation type in breast tumors by race and receptor status. Breast Cancer Res Treat. 2022 Apr;192(3):639-648. PubMed Central PMCID: [PMC8960361](#).
3. Ramroop JR, Gerber MM, Toland AE. Germline Variants Impact Somatic Events during Tumorigenesis. Trends Genet. 2019 Jul;35(7):515-526. PubMed Central PMCID: [PMC6571050](#).
4. Dworkin AM, Tseng SY, Allain DC, Iwenofu OH, Peters SB, Toland AE. Merkel cell polyomavirus in cutaneous squamous cell carcinoma of immunocompetent individuals. J Invest Dermatol. 2009 Dec;129(12):2868-74. PubMed PMID: [19554019](#).
5. Gerber MM, Hampel H, Zhou XP, Schulz NP, Suhy A, Deveci M, Çatalyürek ÜV, Ewart Toland A. Allele-specific imbalance mapping at human orthologs of mouse susceptibility to colon cancer (Scc) loci. Int J Cancer. 2015 Nov 15;137(10):2323-31. PubMed Central PMCID: [PMC6995280](#).

Other Significant Products Highlighting Contributions to Science

1. Ewart AK, Morris CA, Atkinson D, Jin W, Sternes K, Spallone P, Stock AD, Leppert M, Keating MT. Hemizyosity at the elastin locus in a developmental disorder, Williams syndrome. Nat Genet. 1993 Sep;5(1):11-6. PubMed PMID: [7693128](#).
2. Hanenberg H, Zhang F, Malev N, Wiek C, Klammer BG, Nassar N, Hesselbrock T, Hanenberg JH, Aeilts AM, Hentschel J, Faust U, Gehrig A, Engel C, Hauke J, Niederacher D, Toland AE, Andreassen PR. Reclassification of ATM Missense Variants of Uncertain Significance by Integrating Results from Systematic Functional Assays into an ACMG Points-Based Framework. Clin Cancer Res. 2025 Jun 13;31(12):2426-2440. PubMed Central PMCID: [PMC12234152](#).
3. Dworkin AM, Ridd K, Bautista D, Allain DC, Iwenofu OH, Roy R, Bastian BC, Toland AE. Germline variation controls the architecture of somatic alterations in tumors. PLoS Genet. 2010 Sep 23;6(9):e1001136. PubMed Central PMCID: [PMC2944791](#).
4. Yilmaz AS, Ozer HG, Gillespie JL, Allain DC, Bernhardt MN, Furlan KC, Castro LT, Peters SB, Nagarajan P, Kang SY,

Iwenofu OH, Olencki T, Teknos TN, Toland AE. Differential mutation frequencies in metastatic cutaneous squamous cell carcinomas versus primary tumors. Cancer. 2017 Apr 1;123(7):1184-1193. PubMed Central PMCID: [PMC5360561](#).

5. Wei L, Allain DC, Bernhardt MN, Gillespie JL, Peters SB, Iwenofu OH, Nelson HH, Arron ST, Toland AE. Variants at the OCA2/HERC2 locus affect time to first cutaneous squamous cell carcinoma in solid organ transplant recipients collected using two different study designs. Br J Dermatol. 2017 Oct;177(4):1066-1073. PubMed Central PMCID: [PMC5650498](#).

Certification:

I certify that the information provided is current, accurate, and complete. This includes, but is not limited to, information related to current, pending, and other support (both foreign and domestic) as defined in 42 U.S.C. § 6605.

In accordance with Section 10632 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19232), each individual identified as a senior/key person must certify that they are not a party to a malign foreign talent recruitment program.

Research Security Training Requirement for Federal Award Personnel: In accordance with Section 10634 of the CHIPS and Science Act of 2022 (42 U.S.C. § 19234), each individual identified as a senior/key person must certify that they have completed the requisite research security training that meets the requirements specified in Item 2 of Important Notice No. 149 within 12 months prior to proposal submission.

Misrepresentations and/or omissions may be subject to prosecution and liability pursuant to, but not limited to, 18 U.S.C. §§287, 1001, 1031 and 31 U.S.C. §§3729-3733 and 3802.

Certified by Toland, Amanda Ewart in SciENcv on 2026-05-21 14:29:20

NIH BIOGRAPHICAL SKETCH SUPPLEMENT

Name: Toland, Amanda Ewart

Persistent Identifier (PID) of the Senior/Key Person: <https://orcid.org/0000-0002-0271-1792>

Position Title: Professor & Interim Chair

Organization and Location: The Ohio State University, Columbus, Ohio, United States

Personal Statement

The major interest of my laboratory is to better understand genetic susceptibility of cancers using data from mouse models and human populations in the context of somatic events in tumors. I have a specific interest in understanding biological differences in germline genetic differences that impact cancer health disparities. My lab has long been interested in the interplay between germline genetics and somatic events in tumors beginning as a postdoctoral fellow when I discovered that a putative susceptibility gene showed allele-specific gains in colon tumors. My doctoral training was in the identification of Mendelian disease genes and led to the identification of a microdeletion on chromosome 7q which causes Williams syndrome; work that still frequently cited. I began working in the field of susceptibility genes and using mouse models for mapping disease genes during my postdoctoral training. I became board certified through the American Board of Medical Genetics in both Clinical Molecular Genetics and PhD Medical Genetics. In my tenured faculty position, my lab focuses on the identification of germline variants that associate with somatic mutations that vary in frequency between different ancestral populations. In our NIH funded work, we use multiple strategies to identify and characterize the mechanisms underlying association between key variants and specific somatic events in multiple tumor types.

Trainees are a critical component of my laboratory, and I am highly committed to mentoring and their success. One of the characteristics on my laboratory is a collaborative, inclusive environment. I have taken mentor training through the NRMN Research Network and employ tools that I have learned to foster a welcoming training environment for my students. I work closely with all my trainees to personalize their training experience to best fit their needs, experiences, and interests to have a successful experience and enter the biomedical workforce or their next step in academic training in a timely manner.

Success of early career faculty in all academic aspects results in a thriving department and institution, increased work satisfaction, and enriches the science for all of us. In my previous role as Vice Chair of the Department of Cancer Biology & Genetics, I established a faculty mentoring program. In my current role as Interim Chair, success of our junior faculty is essential. I currently serve on mentoring committees for multiple faculty. Due to my strong commitment to mentoring at all levels, I was recently appointed as Associate Director for Cancer Research Training and Education Coordination (CRTEC). The mission of CRTEC aligns with my passion for mentoring of cancer scientists across the training and career trajectory. From my experience in mentoring trainees and junior faculty, my scientific expertise in cancer genetics and genomics, and my leadership roles that focus on mentoring and career development, I have the necessary background and leadership to oversee mentoring and career development for students in this T32 program.

Pertinent funding includes: 1R01CA265975-01A1, Role MPI, Racial differences in immunogenetic tumorigenesis of head and neck squamous cell carcinoma; ACS DICR POST-BACC-24-1342575-01-DPBACC, Role: PI, THE OSUCCC ACS DICR Post-Baccalaureate Fellows Program; DICR ACS SHE-24-1272435-01-SHE, Role: PI. Summer health experience in oncology – The Ohio State University CCC.

Honors

2024	Mentor of the Year Award, Comprehensive Cancer Center, The Ohio State University
2010	NIH Merit Award (Group award to PDQ Cancer Genetics Editorial Board), National Institutes of Health
2007	American Cancer Society Research Scholar, American Cancer Society
1999	Melvin Grumbach Award for Excellence in Research, University of California, San Francisco
1990	Phi Beta Kappa, Franklin & Marshall College

Contributions to Science

1. My early work focused on the identification of the genetic etiology of an autosomal dominant disease, supravalvular aortic stenosis, and a related syndrome, Williams syndrome. Through linkage studies, I discovered that elastin (ELN) contributed to hereditary SVAS (published in PNAS in 1993). Subsequent studies looking for mutations in individuals with SVAS and Williams syndrome led to the identification of a microdeletion on chromosome 7q23.1, including ELN, that caused Williams syndrome (Ewart AK et al. Hemizygosity at the elastin locus in a developmental disorder, Williams syndrome. Nat Genet. 1993). This work led to the first clinical test using FISH probes at this locus, for Williams syndrome which enabled further characterization and understanding of the different features of this disorder. This work was completed when I was a graduate student in the laboratory of Dr. Mark Keating, but I played a critical role in the experimental design, data generation and data interpretation of these studies.
2. During my clinical genetics training, I realized that variants of uncertain significance were a major clinical problem for interpretation of clinical gene sequencing. This led to an ongoing focus in the laboratory to identify new means of characterizing VUS in BRCA1, BRCA2 and other breast cancer susceptibility genes such as BARD1 and ATM. I developed a new multifactorial algorithm for classification of variants that did not depend on segregation analyses. This work led to the classification of several VUS which was published in the Journal of Clinical Oncology in 2008. I also contribute to the VUS classification through my participation in ENIGMA, an international consortium whose goal is to classify VUS in hereditary breast and ovarian cancer genes. Ongoing collaborative work with the ENIGMA consortium has led to the identification of some moderately penetrant missense changes in BRCA1. Currently I am integrating functional assays developed by Paul Andreassen into an ACMG rules-based platform for variant classification which resulted in a recent paper published in Clinical Cancer Research (Hanenberg H, Zhang F, Malev N, Wiek C, Klamer BG, Nassar N, Hesselbrock T, Hanenberg JH, Aeilts AM, Hentschel J, Faust U, Gehrig A, Engel C, Hauke J, Niederacher D, Toland AE, Andreassen PR. Reclassification of ATM Missense Variants of Uncertain Significance by Integrating Results from Systematic Functional Assays into an ACMG Points-Based Framework, 2025).
3. My laboratory has long been interested in the connection between germline variants and somatic events in tumors. Based on data from mouse models, we hypothesized that variants conferring cancer susceptibility would be preferentially gained in tumors and those conferring resistance would show relative loss. We were the first to show preferential allelic gains in losses in independent tumors occurring in the same individual, suggesting that germline differences were driving somatic changes. These studies have led to the identification of candidate genes important in colon and squamous cell carcinoma (Dworkin et al. Germline variation controls the architecture of somatic alterations in tumors. PLoS Genetics 2010). This led to current studies evaluating the association between germline variants and somatic mutations that help to explain differences in somatic mutation frequencies between Association of ESR1 Germline Variants with TP53 Somatic Variants in Breast Tumors in a Genome-wide Study, populations which impact cancer prognoses and outcomes such as TP53 in breast cancer and HNSCC and KRAS in colorectal cancer (Tjader NP et al. Cancer Research Communication 2024).
4. Following from work done as a postdoctoral fellow, my laboratory has identified microRNAs and somatic mutations that contribute to SCC tumorigenesis and/or metastasis, including those important in solid organ transplant recipients. My laboratory performed multiple studies which identified and functionally characterized microRNAs and mutations associated with cSCC development and/or aggression in both mouse and humans. In addition, we identified variants in the 3'UTRs of candidate cancer susceptibility genes in the mouse that affected miRNA binding and gene expression. From exome studies of paired primary and metastatic cSCCs we determined that somatic mutations in KMT2D were more common in aggressive tumors (Yilmaz et al Differential mutation frequencies in metastatic cutaneous squamous cell carcinomas versus primary tumors, Cancer, 2017). We furthermore showed in xenograft models of isogenic KMT2D knock-out and wildtype cell lines that somatic mutation led to characteristics of more aggressive tumors (Dauch et al. KMT2D loss drives aggressive tumor phenotypes in cutaneous squamous cell carcinoma. American Journal of Cancer Research, 2022).
5. In addition to contributing to consortia (CIMBA, GECCO and others) performing large genome-wide association studies for cancer risk of breast cancer, ovarian cancer, colorectal cancer and cutaneous squamous cell carcinoma, we have led studies identifying variants in AURKA as important for cancer risk of breast and colorectal cancer (Ewart-Toland et al. Aurora-A/STK15 T+91A is a general low penetrance cancer susceptibility gene: a meta-analysis of multiple cancer types. Carcinogenesis. 2005) as well as identifying genetic risk factors for cutaneous squamous cell cancer risk in the general population and high risk solid organ transplant recipients (Wei et al. Variants at the OCA2/HERC2 locus affect time to first cutaneous squamous cell carcinoma in solid organ transplant recipients collected using two different study designs. British Journal of Dermatology, 2017)

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