The Robert Larner, M.D. College of Medicine
THE UNIVERSITY OF VERMONT

Steven A. Roberts

CURRICULUM VITAE

Position: Professor
Department of Microbiology and Molecular Genetics
Larner College of Medicine
University of Vermont Cancer Center
University of Vermont

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EDUCATION

2008	University of North Carolina	Ph.D.	Biochemistry
2003	Bowling Green State University	B.S.	Chemistry, Biology Graduated Summa Cum Laude, GPA 4.0

LICENSES, CERTIFICATION

None

FACULTY POSITIONS HELD

2025-present	University of Vermont	Professor	Department of Microbiology and Molecular Genetics
2023-2025	University of Vermont	Associate Professor	Department of Microbiology and Molecular Genetics
2025-present	University of Vermont	Cancer Cell Program Co-leader	University of Vermont Cancer Center; Cancer Cell Program

2023-present	University of Vermont	Full Member	University of Vermont Cancer Center; Cancer Cell Program
2023-2024	Washington State University	Affiliate Faculty	School of Molecular Biosciences
2019-2023	Washington State University	Associate Professor	School of Molecular Biosciences
2014-2019	Washington State University	Assistant Professor	School of Molecular Biosciences

OTHER POSITIONS AND MAJOR ADMINISTRATIVE POSITIONS HELD

[optional-add duties/description]

2009-2014	Laboratory of Molecular Genetics, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, NC 27709	Postdoctoral Research Associate
2008-2009	Department of Biochemistry and Biophysics, University of North Carolina, Chapel Hill NC 27599	Postdoctoral Research Associate

HONORS AND AWARDS [*All inclusive, most recent last]*

2002	Barry M. Goldwater National Scholarship in Science
2003	Phi Beta Kappa Honor Society
2003	Irving Scholarship, Department of Biochemistry and Biophysics, UNC-CH
2004	National Science Foundation Pre-doctoral Fellowship: Honorable Mention
2010	Best Oral Presentation, Genetics and Environmental Mutagenesis Society Fall Meeting
2011	Best Poster Presentation, Gordon Research Conference in Genetic Toxicology
2012	NIH Fellows Award for Research Excellence
2012	NIEHS Paper of the Year (Roberts, et al. Molecular Cell, 2012)
2013	Best Poster Presentation, NIEHS Science Day
2013	NIEHS Fellow of the Year
2017	WSU CVM Dean's Outstanding Junior Faculty Research Award
2018	WSU Pacesetter Award (from the Office of Research)
2019	SMB Fall Conference Outstanding Faculty Research Award

KEYWORDS/AREAS OF INTEREST *Please provide a set of MESH indexing terms to describe your research and/or clinical interests.]*

- Genome dynamics: mechanisms of mutation and chromosome alteration that contribute to human disease.
- Roles of APOBEC cytidine deaminases in cancer mutagenesis
- Genome-wide mapping of DNA lesions
- DNA damage-induced mutation
- Mutagenic DNA double-strand break repair

SUMMARY OF PROFESSIONAL ACTIVITIES- OVERALL

My primary responsibility as faculty at Washington State University (WSU) and the University of Vermont (UVM) has been conducting research with secondary efforts in contributing to the undergraduate/graduate student teaching missions and providing departmental, college, university and external service.
As a principal investigator, my research program has been continually funded from external agencies (the National Institutes of Health (NIH) and the Department of Defense (DOD)) since 2014, usually with multiple simultaneously funded projects. These research efforts have focused on two general areas: 1) characterizing how dysregulation of the APOBEC family of enzymes leads to genetic instability and cancer development; 2) utilizing genome-wide DNA lesion mapping and sequencing methods to evaluate the distribution of DNA lesion formation, repair, and mutation in yeast, human cells, and tumors. Since establishing my independent research program, these efforts have resulted in 34 primary research articles (published in journals like *Nature Genetics*, *Nature Communications*, *Cell Reports*, *PLoS Genetics*, *PNAS*, etc.) and 10 review articles. Additional primary research articles have also been published associated with my membership in several working groups of the International Cancer Genome Consortium.

As an educator, my teaching responsibilities have focused on team-teaching undergraduate and graduate Bioinformatics and an undergraduate Genetics Laboratory at WSU. These courses have ~60 and ~25 students a semester, respectively. I also taught in the Cancer Genetics course and am teaching a graduate level Genetics and Genomics course at UVM. I additionally supplement these formal teaching responsibilities with informal independent undergraduate research, where I teach students in a one-on-one setting about scientific research, experimental design, and the scientific method.

My service efforts primarily supported the graduate education mission within the School of Molecular Biosciences (SMB) and to advance the research capacity and portfolio of the College of Veterinary Medicine (CVM) at WSU. I served and co-chaired committees to recruit and advise graduate students in SMB, while also serving on steering committees of the Center of Reproductive Biology and the NIH-funded T32 Protein Biotechnology training grant. My efforts in leading the CVM scientific mission included serving on the CVM Research Council and CVM Research Committee. At UVM, I serve as the co-chair of the Graduate Students and Academic Programming (GAP) committee within the Department of Microbiology and Molecular Genetics (MMG) and have participated in out-reach efforts for the UVM Cancer Center. External to WSU and UVM, I have also continuously served on

National Institute of Health and Department of Defense grant review panels as well as an adhoc reviewer for multiple high profile scientific journals (e.g. *Nature*, *Nature Genetics*, *Nature Communications*, *PNAS*, etc.). I also was a member of multiple working groups of The Cancer Genome Atlas (TCGA) and International Cancer Genome Consortium (ICGC).

SUMMARY OF ACCOMPLISHMENTS

Career Discoveries:

- APOBEC cytidine deaminases mutate the lagging strand template.
- APOBEC3A is a primary breast cancer mutator.
- APOBEC-induced mutation is avoided by error-free lesion bypass mechanisms.
- Nucleosomes and Ets family transcription factors modulate UV-induced mutagenesis.
- UV light produces atypical UV photoproducts that cause common *BRAF* oncogenic mutations.

External Funding Success:

- My lab has been externally funded continuously since its establishment in 2014.
- Funded grants include the following NIH awards: 1 R00, 3 R01, 2 R21.

Mentoring Success:

- In 10 years, I have mentored 13 graduate students, already graduating 7 PhDs and 1 MS student. 1 more is likely to graduate with a PhD in the next year. I have also had over 30 undergraduate students work in my lab, with multiple being co-authors on publications and progressing on to MD, PhD, and MD/PhD programs.

PROFESSIONAL SERVICE

DEPARTMENTAL SERVICE

At WSU

2014	School of Molecular Biosciences	Ad hoc Faculty Recruitment Committee (Bret Freudenthal)	Member
2014	School of Molecular Biosciences	Graduate Recruiting Committee	Member
2014	School of Molecular Biosciences	Retreat Committee	Co-Chair
2015	School of Molecular Biosciences	Retreat Committee	Member
2015-2018	School of Molecular Biosciences	Graduate Affairs Committee	Member
2015-2018	School of Molecular Biosciences	Graduate Studies Committee	Member
2015-2018	School of Molecular Biosciences	Graduate Recruiting Committee	Co-Chair

2016	School of Molecular Biosciences	FGI Faculty Search Committee	Member
2017	School of Molecular Biosciences	FGI Faculty Search Committee	Member
2015-2021	School of Molecular Biosciences	Graduate Recruiting Committee	Member
2019-2023	School of Molecular Biosciences	Graduate Studies Committee	Member
2019-2020	School of Molecular Biosciences	SMB Director Search Committee	Member
2021-2023	School of Molecular Biosciences	SMB Tenure and Promotion Committee	Member
2021-2023	School of Molecular Biosciences	Graduate Affairs Committee	Member

At UVM

2024-current	Department of Microbiology and Molecular Genetics	GAP Committee	Co-Chair
2024-2025	Department of Microbiology and Molecular Genetics	2024-2025 MMG seminar series	Organizer

COLLEGE SERVICES

At WSU

2015	Poncin Scholar Grant	Reviewer
2015	IPN Faculty Search Committee	Member
2019	CVM WSU Intramural Seed Grant Review Committee	Member
2019-2023	Center for Reproductive Biology Steering Committee	Member
2019	IPN Faculty Search Committee	Member
2020-2022	CVM Research Committee	Member
2020	Alcohol and Drug Addiction Research Program	Reviewer

2020	Associate Dean of Research Search Committee	Member
2021-2023	CVM Research Council	Member
2022	Alcohol and Drug Addiction Research Program	Reviewer

At UVM

2025-current	UVMCC Cancer Cell Program	Co-Leader

MEDICAL CENTER SERVICE

Not Applicable

UNIVERSITY SERVICE

At WSU

2018-2023	LBB1 sequencing core user group	Leader
2019-2022	Executive Steering Committee; Biotechnology Training Grant	Member
2020-2023	Goldwater Scholarship Selection Committee	Member

At UVM

2024-2025	CMB 2.0 Task Force	Member
2025-present	CMB Student Progress Committee	Chair
2025-present	CMB Education Committee	Member
2025-present	CMB Steering Committee	Member
2025-present	Graduate Executive Committee	Member

GOVERNMENT

2015	Medical Research Council UK (New Investigator Research Grant)	Reviewer
2017	National Cancer Institute Special Emphasis Panel: NATIONAL CANCER INSTITUTE ZCA1 RPRB-L (J1) NCI Program Project III (P01)	Member
2018	National Cancer Institute Special Emphasis Panel: NATIONAL CANCER INSTITUTE 2018/10 ZCA1 RTRB-E (O1) P NCI Program Project V (P01).	Member
2019	NCI Program Project II (P01) Review Committee	Member

2020	Congressionally Directed Medical Research Program (CDMRP) Grant review panel. Department of Defense	Member
2020	Congressionally Directed Medical Research Program (CDMRP) Grant review panel. Department of Defense	Member
2020	UK Research and Innovation-BBSRC Grant review	Reviewer
2021	Congressionally Directed Medical Research Program (CDMRP) Grant review panel. Department of Defense	Member
2022	Medical Research Council UK Program Reviewer	Reviewer
2023 (July)	NIH F09A Study Section	Ad hoc Member
2023 (October)	NIH F09A Study Section	Ad hoc Member
2024 (June)	2024/10 ZCA1 RPRB-M (O1) NCI Program Project (P01) Review SEP- D	Member
2024 (October)	UK Research and Innovation-Future Leaders Fellowships	Reviewer
2025 (Feb)	2025/02 ZCA1 RPRB-T (M1), NCI Program Project (P01) Review SEP-B	Member
2025 (Feb)	2025/02 NCI P50 Research Center Grants for Specialized Programs of Research Excellence (SPORE) review	Member

SOCIETY MEMBERSHIPS

None

SERVICE TO PROFESSIONAL ORGANIZATIONS

2019	FASEB Genetic Recombination and Genome Rearrangements. Steamboat Springs, CO	Discussion Leader
2014-2016	The Cancer Genome Atlas Working Groups for Bladder, Cervical and Kidney Chromaphobe cancers	Member

2014-2020	International Cancer Genome Consortium Pan-cancer working groups for Structural Rearrangements (PCAWG-6), Mutation Signatures (PCAWG-7), and Germline (PCAWG-8)	Member

SERVICE TO PROFESSIONAL PUBLICATIONS

2013-present	Adhoc Manuscript Review: >50 manuscripts reviewed total for the journals *Science Advances*, *Communications Biology*, *PLOS Genetics, Scientific Reports, Nature, Nature Genetics, eLife, EMBO, EMBO Reports, Journal of Experimental Medicine, PNAS, Nature Communications, Breast Cancer Research, The FEBS Journal, Physical Reviews X, Genome Research, Trends in Biochemical Sciences, Chemical Reviews, Nucleic Acids Research, DNA Repair, Frontiers in Genetics: Genomic Assay Technology, Oral Oncology, Cell Division, PLOS One, Genetics, NAR Cancer, Viruses*
2016-2021	Review Editor: Frontiers in Genetics: Genomic Assay Technology
2019-present	Editorial Board Member: Mutation Research – Reviews in Mutation Research
2021-present	Associate Editor: Frontiers in Genetics: Genomic Assay Technology

PUBLIC SERVICE

None

SUMMARY OF SERVICE ACTIVITIES

Most of my service activities have centered on the review and strategic deployment of funds to support scientific research.

I contributed extensively to service activities within Washington State University (WSU) at the Department, College, and University levels. At the Department level, I was a member of the Tenure and Promotion Committee from 2021-2023. I was also co-Chair of the Graduate Student Recruiting committee from 2015-2018. In this role, I was responsible for overseeing the selection of our graduate student applicants and recruiting them into our graduate program. As co-chair of this committee, I also served as a member of the graduate curriculum committee and graduate affairs committee that established the student curriculum and advised enrolled graduate students. As extensions of these activities, I was a member of the Executive Steering Committee of WSU's Protein Biotechnology T32 training grant funded by NIH. At a College and University level, I served on the Executive Steering Committee of the Center for Reproductive Biology to determine the research direction of the Center and the faculty it supports. I was a member of the College of Veterinary Medicine's Research Committee to evaluate and review internal CVM grant submissions. I was also on the CVM Research Council, which functions to provide a strategic mission for supporting research in the College.

I was the faculty leader of the Genomics Core user group at WSU. At UVM, my service commitments are just beginning. Within my first year at UVM, I have already begun serving as co-chair of the Graduate Students and Academic Programming (GAP) committee within the Department of Microbiology and Molecular Genetics (MMG). I have also taken over the organization of the 2024-2025 MMG seminar series. I am a member of the CMB 2.0 task force to re-envision the organization of the graduate program in Cellular, Molecular, and Biomedical Sciences at UVM.

Outside of WSU and UVM, I have participated on multiple review panels for the National Institutes of Health (reviewing F30, F31, F32, and Program Project Grants) and Congressionally Directed Medical Research Program (CDMRP) Grant review panels of the Department of Defense. I have also reviewed multiple grants from the Medical Research Council in the United Kingdom. I have served as an adhoc reviewer for many peer review journals, including prestigious journals such as *Nature*, *Nature Genetics*, *Nature Communications*, *Proceedings of the National Academy of Sciences*, *EMBO*, *Science Advances*, *etc*. I am currently on the editorial boards of *Frontiers in Genetics: Genomic Assay Technology* and *Mutation Research – Reviews in Mutation Research*.

TEACHING

FORMAL SCHEDULED COURSES

Year	Course Title	Course R	Course E	Hours	Number of Learner	Learner Level
2017	Bioinformatics (at WSU)		x	10	48	Undergrad
2017	Bioinformatics (at WSU)		x	10		G
2018	Bioinformatics (at WSU)		x	10	34	Undergrad
2018	Bioinformatics (at WSU)		x	10		G
2019	Bioinformatics (at WSU)		x	10	74	Undergrad
2019	Bioinformatics (at WSU)		x	10		G
2019	Genetics Laboratory (at WSU)	x		10	18	Undergrad
2020	Bioinformatics (at WSU)		x	10	51	Undergrad
2020	Bioinformatics (at WSU)		x	10		G
2020	Genetics Laboratory (at WSU)	x		10	35	Undergrad
2021	Genetics Laboratory (at WSU)	x		10	24	Undergrad
2022 - Spring	Bioinformatics (at WSU)		x	10	56	Undergrad
2022 - Spring	Bioinformatics (at WSU)		x	10		G
2022 - Spring	Genetics Laboratory (at WSU)	x		10	32	Undergrad
2022 - Fall	Bioinformatics (at WSU)		x	10	39	Undergrad
2022 - Fall	Bioinformatics (at WSU)		x	10		G
2023 - Spring	Genetics Laboratory (at WSU)	x		10	25	Undergrad
2024 - Spring	Cancer Genetics (at UVM)		x	7	25	Undergrad
2024 - Spring	Cancer Genetics (at UVM)		x	7	4	G
2024 – Fall	Genetics and Genomics (at UVM)	x		30	15	G
2025 – Fall	Genetics and Genomics (at UVM)	x		30	23	G
2026 – Spring	Genetics and Genomics (at UVM)	x		30	19	G

R-required; E-elective; Hours-approx. per semester; G-graduate studies (instruction as per the LCOM Teaching Academy Portfolio)

CURRICULUM DEVELOPMENT

In all the courses I have taught, I have made efforts to provide my students with a first-hand scientific research experience. I have done this by incorporating primary research literature into the coursework as well as active-learning laboratory projects that mirror real scientific studies. Primary research papers were used to illustrate the usefulness of specific bioinformatics analyses in my section of Bioinformatics at WSU. To additionally provide advanced hands-on experience in bioinformatics analyses, I created lab projects in the course that involved using publicly available raw whole genome sequencing data, which the students then process using published bioinformatics tools, and subsequently tabulate to describe a biological phenomenon occurring in the test samples. In most cases, the raw data and analysis closely resembled the processes used in published primary literature. In Genetics Laboratory, I developed a multi-step laboratory for students to design CRISPR-Cas9 guide RNA sequences and Donor repair templates to insert a novel mutation in an essential gene of yeast, most recently *POL3*. Students chose the site they want to mutate, design the appropriate guide RNA sequence, and repair template to make the mutation. They then clone DNA oligonucleotides into an appropriate expression vector to create the functional CRISPR-Cas9 with guide RNA and transform this vector into yeast with the repair template to make the mutation in the *POL3* gene. Mutations were confirmed by DNA sequencing and the impact of the mutation on function is tested by evaluating growth of the

mutant yeast on media containing hydroxyurea or methyl methanesulfonate. Similarly, I led a student discussion of a primary research article on the immune therapies targeting cancer neoantigens in Cancer Genetics at UVM and presented original research from my lab illustrating how cancer therapies can influence mutagenic mechanisms occurring during cancer progression, leading to therapeutic resistance. I have independently developed a graduate course in Genetics and Genomics (MMG6990C) currently being taught to 15 students. This course focuses on advanced Genetics and Genomics concepts and methodologies, separating graduate level students from a previously conjoined class structure. Topics covered in this course include: 1) DNA replication, repair, and packaging 2) chromosomal organization 3) mitotic and meiotic cell division 4) inheritance 5) transcription and gene expression 6) epigenetics 7) next-generation sequencing technologies and 8) genomics techniques like whole genome sequencing, RNA-seq, ChIP-seq, etc. The objectives of the course are for students to gain a solid foundation in the fundamentals of Genetics and Genomics and to learn to analyze and interpret experiments using genetics and genomics methodologies. Consequently, the course is structured in a hybrid format, consisting of lectures, student journal club presentations, and hands on computational work to analyze raw genomics data. The course places a particular emphasis on experimental design.

POSTGRADUATE AND OTHER COURSES

PREDOCTORAL STUDENTS SUPERVISED OR MENTORED

Dates	Name	Program School	Role	Current Position
Jan. 2015 - Dec. 2019	James Hoopes	Ph.D. Student	Research Advisor	Sales Representative Thermo-Fisher Scientific
Jan. 2015 - Sept. 2018	Alexander Brown	Ph.D. Student	Research Advisor	Scientist, NGS Data Analyst, AbSci, Vancouver WA
Jan. 2016 - May 2020	Amber Brown	Ph.D. Student	Research Advisor	Scientist, High Throughput Screening Group, AbSci, Vancouver WA
March 2019 – May 2023	Madeline Dennis	Ph.D. Student	Research Advisor	Post-Doc, Cancer Vaccine Institute, University of Washington, Seattle WA
March 2019 – June 2024	Christopher Collins	Ph.D. Student	Research Advisor	Graduated with PhD
March 2020 - Aug. 2022	Nicolas Bray	M.S. Student	Research Advisor	MD-PhD student University of Texas Health Science Center-San Antonio
March 2020 – Aug. 2024	Brittany Vandenberg	Ph.D. Student	Research Advisor	Current Post-Doc in the Billon Lab, University of Calgary
Aug. 2021 – Aug. 2024	Margo Coxon	Ph.D. Student	Research Advisor	Finishing DVM portion of DVM/PhD program at Washington State University
March 2022 – Dec. 2025	Cameron Cordero	Ph.D. Student	Research Advisor	Bioinformatics Developer, Naveris, Inc. Waltham, MA

March 2022 - present	Vanessa Lopez	Ph.D. Student	Research Advisor	Current graduate student in the Roberts Lab
Nov 2023 – present	Alyssa Hurley	Ph.D. Student	Research Advisor	Current graduate student in the Roberts Lab
Sept 2024 – present	Erin Gaston	Ph.D. Student	Research Advisor	Current graduate student in the Roberts Lab
June 2024 – present	Atri Raval	Ph.D. Student	Research Advisor	Current graduate student in the Roberts Lab
June 2024 – present	John Ball	Ph.D. Student	Research Advisor	Current graduate student in the Roberts Lab
Oct. 2014 - July 2018	Luis Cortez	Undergraduate	Research Advisor	MD-Ph.D. student at University of Kansas Medical Center
Jan. 2015 - Jan. 2016	Stephen Mather	Undergraduate	Research Advisor	Ph.D. student in Mathematics at University of Tennessee-Knoxville
Jan. 2015 - Dec. 2016	Anthony Sader	Undergraduate	Research Advisor	employee at Seattle Cancer Care Alliance, University of Washington, Seattle WA
Jan. 2015 - June 2015	Austin Jensen	Undergraduate	Research Advisor	Genomics Data Scientist, Omics, Seattle WA
Jan. 2016 - Jan. 2017	Lauren Hobson	Undergraduate	Research Advisor	Employee at EcoAnalytics, Moscow, ID
Jan. 2016 - Jan. 2017	Anna Wood	Undergraduate	Research Advisor	Ph.D. program, Cornell University
Aug. 2016 - Sept. 2017	Elizabeth Rice-Reynolds	Undergraduate	Research Advisor	Patient Care Technician at the University of Washington
Jan. 2017 - Dec. 2017	Ly Nguyen	Undergraduate	Research Advisor	graduated from WSU
Jan. 2017 - May 2019	Ellen MacNary	Undergraduate	Research Advisor	AMR EMT
Jan. 2017 - May 2018	Ellie Lashman	Undergraduate	Research Advisor	Masters in Teaching student at WSU-Spokane
Jan. 2017 - June 2017	Samuel Peterson	Undergraduate	Research Advisor	Transferred to Boise State University
May 2017 - May 2019	Sierra Roy	Undergraduate	Research Advisor	graduated from WSU
May 2017 - May 2018	Mikayla Engstrom	Undergraduate	Research Advisor	MD program, WSU School of Medicine
Oct 2017 - Aug 2018	Mary Lalone	Undergraduate	Research Advisor	graduated from WSU
May 2018 - May 2019	Charles McElroy	Undergraduate	Research Advisor	M.D. program; University of Washington School of Medicine
May 2017 - 2021	Secily Thompson	Undergraduate	Research Advisor	EMT-B at Bayview Medical Associates

June 2019 - 2020	Nico Logerfo	Undergraduate	Research Advisor	Rocky Vista University School of Osteopathic Medicine
Jan. 2020 - Dec. 2020	Deandre Richie	Undergraduate	Research Advisor	graduated from WSU
Jan. 2020 - May 2021	Rachel Burstein	Undergraduate	Research Advisor	Production Associate at Blue Heron Biotech
Jan. 2020 - May 2021	Bayley McDonald	Undergraduate	Research Advisor	Ph.D. student, School of Molecular Biosciences, WSU
Jan. 2020 - 2023	Jordan Kreyenhagen	Undergraduate	Research Advisor	currently enrolled in WSU
2021-2021	Jacob Satake	Undergraduate	Research Advisor	currently enrolled in WSU
Jan. 2021 - May 2022	Alexandra Puzon	Undergraduate	Research Advisor	Enrolled in Pacific Northwest University College of Osteopathic Medicine
2021 - May 2022	Rudri Vyas	Undergraduate	Research Advisor	CMB Ph.D. program, University of Vermont
2021-2023	Jose Ilagan	Undergraduate	Research Advisor	MD student, University of North Carolina
Aug. 2021 - Dec. 2021	Daniel Ng	Undergraduate	Research Advisor	graduated from WSU
2021 - 2022	Lauren Eberhardt	Undergraduate	Research Advisor	currently enrolled in WSU
2021 - 2023	Sydney Croasdell	Undergraduate	Research Advisor	Attending M.S. in forensics; King's College London
Sept. 2023 – present	Daniel Schiefen	Undergraduate	Research Advisor	Currently enrolled at UVM
Sept. 2023 – present	Sophie Venman	Undergraduate	Research Advisor	Currently enrolled at UVM
Jan. 2024 – May 2025	Jordan Murch	Undergraduate	Research Advisor	M.S. student, Syracuse
Mar. 2024 – present	Laura Brooks	Undergraduate	Research Advisor	Currently enrolled at UVM
Mar. 2024 Sept. 2024	Wyatt Washko	Undergraduate	Research Advisor	Currently enrolled at UVM
Apr. 2024 – May 2025	Sylvia Snow	Undergraduate	Research Advisor	Graduated from UVM
Apr. 2024 – May 2025	Delaney Lima	Undergraduate	Research Advisor	Graduated from UVM; DVM student University of Alaska Fairbanks
Sept. 2024 - present	Kate Gibbemeyer	Undergraduate	Research Advisor	Currently enrolled at UVM
Jan. 2025 - present	Dana Burrington	Undergraduate	Research Advisor	Currently enrolled at UVM
April 2025 - present	Emma Cibulskis	Undergraduate	Research Advisor	Currently enrolled at UVM

Jan. 2026	Ashley Sanders	Undergraduate	Research Advisor	Currently enrolled at UVM
Jan. 2026	Emma McBournie	Undergraduate	Research Advisor	Currently enrolled at UVM
Jan. 2026	Emma Coupe	Undergraduate	Research Advisor	Currently enrolled at UVM
Jan. 2026	Madeliene Bolno	Undergraduate	Research Advisor	Currently enrolled at UVM

DISSERTATION/THESIS COMMITTEE MEMBERSHIP

Graduate Student Committees

1. Atri Raval, Ph.D. Student, CMB UVM, 2025-present
2. John Ball, Ph.D. Student, CMB UVM, 2025-present
3. Erin Gaston, Ph.D. Student, CMB UVM, 2024-present
4. Brailey Buntin, Masters Student, UVM, 2025-present
5. Jessica Reich, AMP Student, UVM, 2025-present
6. Ignatius Hastomo, Masters Student, UVM, 2025-2025
7. Tylar Kirch, Ph.D. Student, CMB UVM, 2024-present
8. Alyssa Hurley, Ph.D. Student, CMB UVM, 2023-present
9. Cameron Cordero, Ph.D. Student, CMB UVM, 2022-present
10. Vanessa Lopez, Ph.D. Student, CMB UVM, 2022-present
11. Margo Coxon, Ph.D. Student, SMB WSU, 2021-2024
12. Marian Laughery, Ph.D. Student, SMB WSU, 2021-2024
13. Hannah Wilson, Ph.D. Student, SMB WSU, 2021-2025
14. Nuha Haque, Ph.D. Student, SMB WSU, 2021-2023
15. Nicolas Bray, Masters Student, SMB WSU, 2020-2022
16. Brittany Vandenberg, Ph.D. Student, SMB WSU, 2020-2024
17. Ben Morledge-Hampton, Ph.D. Student, SMB WSU, 2020-2024
18. Bastian Stark, Ph.D. Student, SMB WSU, 2020-2023
19. Madeline Dennis, Ph.D. Student, SMB WSU, 2019-2023
20. Christopher Collins, Ph.D. Student, SMB WSU, 2019-2024
21. Kaitlynne Bohm, Ph.D. Student, SMB WSU, 2018-2022
22. Nick Negretti, Ph.D. Student, SMB WSU, 2018-2020
23. Dalton Plummer, Ph.D. Student, SMB WSU, 2017-2022
24. Rachel Gewiss, Ph.D. Student, SMB WSU, 2017-2021
25. Aneesa Al Soodani, Ph.D. Student, SMB WSU, 2015-2017
26. Amber (Hughes) Brown, Ph.D. Student, SMB WSU, 2015-2020
27. James Hoopes, Ph.D. Student, SMB WSU, 2014-2019
28. Alexander Brown, Ph.D. Student, SMB WSU, 2014-2018

PSM Student Committees

1. Chair, Charisma Jenkins, PSM WSU, 2015-2016
2. Heather Maylor-Hagen, PSM WSU, 2016-2018
3. Edith Orozco, PSM WSU, 2016-2019
4. Kelly Husted, PSM WSU, 2016-2018
5. Jessica Watkins, PSM WSU, 2020-2023
6. Chair, Bhim Thapa, PSM WSU, 2020-2022

Undergraduate Honors Thesis Committees

1. Tierra Hunter, WSU, 2015
2. Advisor, Joshua Wilkes, WSU, 2016-2018
3. Advisor, Elizabeth Rice-Reynolds, WSU, 2016-2018, pass with distinction.
4. Advisor, Alexandra Puzon, WSU, 2021-2022
5. Advisor, Dan Schiefen, UVM, 2023-present
6. Advisor, Laura Brooks, UVM, 2024-present
7. Advisor, Sylvia Snow, UVM, 2024-2025

POSTDOCTORAL FELLOWS AND RESIDENTS DIRECTLY SUPERVISED OR MENTORED

Dates	Name	Fellow	Faculty Role	Current Position
2016-2019	Peng Mao	Research Assistant Professor in the Wyrick Lab	Personal advisor	Associate Professor, Stony Brook University
2016-2019	Lisa Victoria Harcy	Post-doctoral Fellow	Research Advisor	Scientist III - Assistant Manager at Idaho Bureau of Laboratories
Jan. 2016 - 2023	Tony Mertz	Post-doctoral Fellow	Research Advisor	Currently Assistant Professor in the Roberts Lab
Sept. 2024 – June 2025	Brittany Vandenberg	Post-doctoral Fellow	Research Advisor	Currently in the Billon Lab, University of Calgary
Nov. 2025 - present	Shannon Prior	Post-doctoral Fellow	Research Advisor	Currently in the Roberts Lab

INFORMAL TEACHING

2014-present Undergraduate research instruction involving the development of small research projects.

FACULTY MENTORED

Dates	Name	Position while Mentored	Faculty Role	Current Position
2023	Alan Goodman	Associate Professor	Research grant review	Associate Professor
2022	Sascha Duttke	Assistant Professor	Research grant review	Assistant Professor
2021	Eric Shelden	Associate Professor	Research grant review	Associate Professor

OTHER VISITING FACULTY SUPERVISED

None

TEACHING AIDS

1. None

TEACHING AWARDS AND NOMINATIONS

None

Teaching portfolio available on request

SUMMARY OF TEACHING ACTIVITIES

At WSU, my primary teaching responsibilities include team teaching a conjoined undergraduate and graduate course in Bioinformatics and an undergraduate Genetics laboratory course. In Bioinformatics, I taught next generation sequencing technology and analysis. This consisted of eight lectures that described how different next generation sequencing methods work, what data they produce, how this data is processed, and how it can be applied to address important biological questions. These analyses are focused on whole genome sequencing to identify sequence variants in samples and RNA-seq analysis to determine gene expression. In Genetics Laboratory, I taught CRISPR/Cas9 gene editing by having students introduce a single nucleotide substitution into an essential gene in yeast (most recently *POL3*) and measuring phenotypes associated with this change. In my first year at UVM, I taught 7 lectures of a conjoined undergraduate and graduate course in Cancer Genetics. The topics I covered were p53 tumor suppressor function, apoptosis, multi-step tumorigenesis, tumor immunology, and cancer immunotherapy. I am currently creating a graduate level course in Genetics and Genomics to be taught in Fall 2024.

I have also placed a great deal of effort teaching and training both undergraduates and graduate students as researchers in my laboratory at both WSU and UVM. To date, I have mentored 35 undergraduates and 13 graduate students. I currently mentor 7 UVM undergrads and 5 UVM PhD students. The major aim for undergraduate researchers in my laboratory is to gain a realistic experience for what working in an academic laboratory environment is like, to learn to develop a hypothesis-driven research project, and to develop a technical skillset that will help the students when they transition either to graduate school or a professional laboratory position. To accomplish this goal, each undergraduate works on a small, self-contained project that fits within a larger research project ongoing in the lab. The student is responsible for interacting with the lab member that is the lead researcher on the larger project and myself to develop their research question and to learn the technical skills required to complete the project. As the student begins to mature both technically conducting experiments and in developing the rationale for individual experiments, they are encouraged to transition towards leading an extension of their research project.

Thus far, this approach towards organizing undergraduate research experiences has been very effective. 13 undergraduate trainees have made sufficient research advances to contribute to peer-reviewed manuscripts published in *Nucleic Acids Research*, *Nature Communications*, *PLoS Genetics*, *Cell Reports*, *Genome Research*, *NAR Cancer*, and *eLife*. Multiple other undergraduate trainees have contributed significantly to research projects within my laboratory and will also be included as authors on up-coming manuscripts. I believe the documentation of their research achievements will greatly aid these students as many of them transition to graduate and medical school studies.

My approach towards graduate student training is likewise to foster the development of independent, critical thinking scientists. To accomplish this, my 11 graduate students have

been responsible for leading their own research projects. While I have been involved in the initial organization of the research question and experimental training, I attempt to quickly develop my students' ability to accurately interpret their data, develop a hypothesis for the next logical extension of their research, and design appropriate, well controlled experiments to test this hypothesis. I facilitate my student's development by providing general direction for the project, but more importantly, by providing support and serving as a critical sounding board for my students' ideas, whether they are for a new direction to advance a project or for troubleshooting difficult experiments. My graduate students have produced 10 first or co-first author publications (in *NAR Cancer*, *Nature Communications*, *Cell Reports, Nucleic Acids Research, Genome Research*, 3 in *PLoS Genetics, Methods of Enzymology*, and *Scientific Reports*), with more nearing submission and publication. 7 of my graduate students have already defended their PhDs (two in four years or less) and one graduated with a MS. The five remaining students are in various stages of completing their degrees.

RESEARCH AND SCHOLARLY ACTIVITIES

RESEARCH AWARDS AND GRANTS

Ongoing Research Support

R01 ES030335-07 (PI: Bernstein) 4/01/2024 – 12/31/2028
Agency: NIH/NIEHS
Replication fork dynamics and repair by Rad51 paralogs after DNA alkylation
This application is to characterize the function of RAD51 paralogs in processing and tolerance of alkylation-induced DNA damage. I am a Co-Investigator on this study. We will assist in this project by determining MMS-induced mutation signatures in RAD51 paralog deficient cells via whole exome sequencing. I will also aid in the analysis of mutation signatures from primary tumors.
Role: Co-I
Direct Costs Year 1: $50,602
Total Period: $463,606 (UVM)

R01 CA269784 (PI: Roberts) 2/06/2023 – 01/31/2028
Agency: NIH/NCI
Regulation of APOBEC3 cytidine deaminase-induced mutation during cancer development
This proposal consists of three major goals: 1) to characterize the processes leading to APOBEC-induced insertion/deletion mutations, 2) to characterize the transcriptional and proteasomal regulation of APOBEC3A during breast cancer development, and 3) to assess whether BRCA1 and BRCA2 dependent homologous recombination limits APOBEC3A induced mutagenesis in breast cancers.
Role: PI
Direct Costs Year 1: $335,273
Total Period: $2,358,840

R01 ES032814 (MPI: Roberts and Wyrick) 4/01/2021 - 1/31/2026
Agency: NIH/NIEHS
Genome-wide analysis of the formation and mutagenesis of atypical UV photoproducts in skin cancer
This proposal attempts to characterize the formation of non-canonical photoproducts in response to UVB irradiation and assess their potential importance to causing driver mutations in skin cancers.

Role: MPI
Direct Costs Year 1: $272,461
Total Period: $2,084,325

Completed Research Support

R21 CA264086 (PI: Roberts) 9/11/2020 – 6/30/2024
Agency: NIH/NCI
Characterizing the contribution of transcription-associated DNA-topoisomerase adducts to mutagenesis in cancer
This project will characterize the types of mutations induced by high transcription which causes the formation of DNA-topoisomerase adducts. We will develop a method to map the position of DNA-Top1 adducts at single nucleotide resolution across the genome of human cancers. It will also investigate a potential link between DNA-Top1 adducts and the mutational signature ID4 that is common in multiple sequenced human cancers.
Role: PI
Direct Costs Year 1: $140,250
Total Period: $381,013

R01 HD101223 (PI: Oatley) 9/11/2020 – 6/30/2024
Agency: NIH/NIMHD
Mechanisms of stem cell specification in the male germline
The goal of this proposal is to investigate mechanisms that contribute to spermatogonial stem cell differentiation. I am a Co-Investigator on this proposal and will solely aid in the bioinformatics analysis of ChIP-seq data.
Role: Co-I
Direct Costs Year 1: $308,338
Total Period: $1,968,450

R01 CA218112 (PI: Roberts) 6/15/2017 – 5/31/2023 (NCE)
Agency: NIH/NCI
Mechanisms of genome instability induced by APOBEC Cytidine Deaminases and its impacts during cancer development.
The major goal of this proposal is to determine how the changes in cellular metabolism that occur during cancer development impact APOBEC-induced mutagenesis and whether these enzymes may be responsible for chromosomal rearrangements in addition to their role in causing base substitutions.
Role: PI
Direct Costs Year 1: $239,693
Total Period: $1,924,210

R01 CA218112- (PI: Roberts) 8/1/2020 - 7/31/2022
Diversity Supplement
Agency: NIH/NCI
Mechanisms of genome instability induced by APOBEC Cytidine Deaminases and its impacts during cancer development.
The major goal of this supplement to R01CA218112 is to support the Ph.D. training of Christopher Collins, who is a member of an under-represented demographic among biomedical scientists. He will be extending the research proposed in R01CA218112 by identifying and characterizing post-translational modifications (primarily acetylation) and a

protein inhibitor of APOBEC3A. This role of these factors in regulating APOBEC3A activity during breast cancer development will be determined.
Role: PI
Direct Costs Year 1: $45,612
Total Period: $134,773

R01 CA218112- (PI: Roberts) 9/30/2018 – 5/31/2021
Administrative
Supplement
Agency: NIH/NCI
Mechanisms of genome instability induced by APOBEC Cytidine Deaminases and its impacts during cancer development.
The major goal of this supplement to R01CA218112 is to establish a collaboration with Dr. Kara Bernstein at the University of Pittsburgh to investigate the role of the Shu complex and Rad51 paralogs in bypassing APOBEC-induced DNA damage. Whether inhibiting Shu complex function in APOBEC mutated cancer cells could provide a potential therapeutic advantage will also be evaluated.
Role: PI
Direct Costs Year 1: $114,401
Total Period: $314,759

R21 ES027937 (PI: Roberts and Wyrick) 8/1/2017 - 6/30/2020
Agency: NIH/NIEHS
Genome-wide analysis of UV damage formation, repair, and mutagenesis
The goals of the project are to determine how chromosomal features influence the distribution of UV damage formation, repair, and mutagenesis using yeast and human cell models.
Role: MPI
Direct Costs Year 1: $150,000
Total Period: $419,167

BC141727 (PI: Roberts) 9/30/2015 – 09/29/2019
Agency: Department of Defense
Synthetic lethality between APOBEC-induced DNA damage and base excision repair inhibition as a treatment strategy for breast cancer
The major goals of this proposal are to develop a robust method to screen breast cancers for elevated amounts of APOBEC-induced deoxyuridine and to determine whether such tumors are sensitive to inhibition of the base excision repair (BER) pathway.
Role: PI
Direct Costs Year 1: $350,000 provided at once
Total Period: $528,500

R00 ES022633 (PI: Roberts) 5/31/2016 – 5/31/2018
Diversity Supplement
Agency: NIH/NIEHS
Environmentally-modulated cytosine deamination in genome instability and cancer
The major goals of this proposal are to support the scientific development of Luis Cortez. He will continue to investigate the substrates of APOBEC-induced mutation in yeast and investigate the role of APOBEC3A in causing cancer mutations.
Role: PI
Direct Costs Year 1: $46,163

Total Period: $121,342

R00 ES022633 (PI: Roberts) 1/01/2015 – 12/31/2017
Agency: NIH/NIEHS
Environmentally-modulated cytosine deamination in genome instability and cancer
The major goals of this proposal are to investigate the impact of APOBEC cytidine deaminases in causing DNA damage and mutation in human cancers by understanding how environmental factors can modulate the expression of these enzymes as well as the amount of their ssDNA substrate.
Role: PI
Direct Costs Year 1: $198,227
Total Period: $746,999

Pending

R01 ES032814-06 (MPI: Roberts and Wyrick)
Agency: NIH/NIEHS
Genome-wide analysis of the formation and mutagenesis of atypical UV photoproducts in skin cancer
This proposal attempts to characterize the formation of non-canonical photoproducts in response to UV irradiation and assess their potential importance to causing driver mutations in skin cancers. The proposal aims to determine the impacts of different exposures and molecular sensitizers on atypical UV photoproduct formation.
Role: MPI
Direct Costs Year 1: $181,662 (UVM)
Total Period: $3,749,951

R01 CA304293-01 (PI: Mertz) 7/01/2025 – 6/30/2030
Agency: NIH/NCI
Multidimensional reciprocity between transcription-associated mutagenesis and cancer drivers and therapeutics
This proposal aims to characterize the mechanisms that modulate transcription-associated mutagenesis in human cells, demonstrate that increased transcription-associated mutagenesis is a common trait of tumor cells and a major source of intragenic insertion and deletion mutations, and directly show that this process contributes to cancer therapeutic resistance. Completion of this proposal will significantly increase our knowledge of the pathways that modulate transcription-associated mutagenesis, help define the molecular etiology of intragenic mutations and enable the improved use of cancer therapeutics.
Role: Co-I
Direct Costs Year 1: $404,738
Total Period: $3,480,140

3R01CA269784-04 (PI: Roberts) 8/01/2025 – 7/31/2027
Agency: NIH/NCI
The APOBEC3A/APOBEC3B hybrid polymorphism as a genetic factor determining HPV associated cancer health disparities
This proposal aims to characterize the impact of the APOBEC3A/APOBEC3B hybrid polymorphism on HPV-associated cancer incidence and cancer health disparities. We will also determine the mechanism by which this allele activates APOBEC3A.
Role: PI
Direct Costs Year 1: $150,000

Total Period: $451,057

2025177 (PI: Covo and Roberts) 10/01/2026 – 9/30/2030
Agency: U.S. – Israel Binational Science Foundation
The mutagenic consequences of DNA damage tolerant pathogenic fungi
This application will evaluate differences in UV light DNA damage responses in subpopulations of the fungal plant pathogen Fusarium oxysporum. Upon UV irradiation, this yeast generates two subpopulations, one that arrests and re-initiates growth quickly and a second that has a prolonged arrest. These two populations can be separated by size. We will utilize UV lesion mapping technologies and whole genome sequencing to evaluate differences in the UV damage response between these groups to determine the mechanism of prolonged arrest.
Role: US PI
Direct Costs Year 1: $27,450 (UVM)
Total Period: $127,500 (UVM)

Selected Unfunded Grant Submissions
R01 CA251241 (PI: Roberts) Submitted: 11/03/20 Score: 35 (21%)
Agency: NIH/NCI
Regulation of APOBEC3A during cancer development
This proposal consists of four major goals: 1) to determine the types of cancers in which APOBEC3A contributes to mutagenesis, 2) to characterize the transcriptional regulation of APOBEC3A during breast cancer development, 3) identify and characterize post-translational modifications and protein interactors that regulate APOBEC3A activity, and 4) to assess whether BRCA1 and BRCA2 dependent homologous recombination limits APOBEC3A induced mutagenesis in breast cancers.
Role: PI

R03 CA259657 (PI: Roberts) Submitted: 06/23/20 Score: 41
Agency: NIH/NCI
Investigating mechanisms of transcription-associated mutagenesis in human cells utilizing novel mutation reporters
This proposal will develop novel mutation reporters for use in human cells and investigate whether transcription-associated mutagenesis is a significant source of mutation in human cells and tumors.
Role: PI

SCHOLARSHIP

Peer Reviewed Publications

Original Research
1. **Roberts SA**, and Ramsden DA (2007). Binding of the Non-Homologous End Joining Factor, Ku, to protein-occluded ends. ***Journal of Biological Chemistry***. 282(14): 10605-13. PMID: 17289670
2. Burkhalter MD, **Roberts SA**, Havener JM, and Ramsden DA (2009) Availability of nucleotides helps determine how cells repair double strand breaks. ***DNA Repair (Amst)***. 8(11):1258-63. PMCID: PMC2763971
3. **Roberts SA**, Strande N, Burkhalter MD, Strom C, Havener JM, Hasty P, and Ramsden

DA (2010). Ku is a 5'-dRP/AP lyase that excises nucleotide damage near broken ends. ***Nature***. 464(7292):1214-7. PMCID: PMC2859099

4. Burch LH, Yang Y, Sterling JF, **Roberts SA**, Chao FG, Xu H, Zhang L, Walsh J, Resnick MA, Mieczkowski PA, and Gordenin DA (2011) Damage-induced localized hypermutability. ***Cell Cycle***. Apr 1;10(7):1073-85. PMCID: PMC3100884
5. **Roberts SA**, Sterling J, Thompson C, Harris S, Mav D, Shah R, Klimczak LJ, Kryukov GV, Malc E, Mieczkowski PA, Resnick MA, and Gordenin DA (2012) Clustered Mutations in Yeast and in Human Cancers Can Arise from Damaged Long Single-Strand DNA Regions. ***Molecular Cell***. 46(4):424-35. PMCID: PMC3361558
6. Strande N, **Roberts SA**, Oh S, Hendrickson EA, and Ramsden DA (2012) Specificity of Ku's 5'dRP/AP lyase promotes Nonhomologous end joining (NHEJ) fidelity at damaged ends. ***Journal of Biological Chemistry***. 287(17):13686-93. PMCID: PMC3340204
7. Chan K, Sterling JF, **Roberts SA**, Bhagwat AS, Resnick MA, and Gordenin DA (2012) Base damage within single-strand DNA underlies in vivo hypermutability induced by a ubiquitous environmental agent. ***PLoS Genetics***. 8(12):e1003149. PMID: 23271983
8. **Roberts SA**, Lawrence MS, Klimczak LJ, Grimm SA, Fargo D, Stojanov P, Kiezun A, Kryukov GV, Carter SL, Saksena G, Harris S, Shah RR, Resnick MA, Getz G, and Gordenin DA (2013) An APOBEC Cytidine Deaminase Mutagenesis Pattern is Widespread in Human Cancers. ***Nature Genetics***. 45(9):970-6. doi:10.1038/ng.2702. PMID: 23852170
9. Lawrence MS, Stojanov P, Polak P, Kryukov GV, Cibulskis K, Sivachenko A, Carter SL, Stewart C, Mermel CH, **Roberts SA**, Kiezun A, Hammerman PS, McKenna A, Drier Y, Zou L, Ramos AH, Pugh TJ, Stransky N, Helman E, Kim J, Sougnez C, Ambrogio L, Nickerson E, Shefler E, Cortés ML, Auclair D, Saksena G, Voet D, Noble M, DiCara D, Lin P, Lichtenstein L, Heiman DI, Fennell T, Imielinski M, Hernandez B, Hodis E, Baca S, Dulak AM, Lohr J, Landau D, Wu CJ, Melendez-Zajgla J, Hidalgo-Miranda A, Koren A, McCarroll SA, Mora J, Crompton B, Onofrio R, Parkin M, Winckler W, Ardlie K, Gabriel SB, Roberts CWM, Biegel JA, Stegmaier K, Bass AJ, Garraway LA, Meyerson M, Golub TR, Gordenin DA, Sunyaev S, Lander ES, Getz G (2013) Mutational heterogeneity in cancer and the search for new cancer genes. ***Nature***. 499(7457):214-8. PMID: 23770567
10. The Cancer Genome Atlas Research Network (2014) Comprehensive molecular characterization of urothelial carcinoma of the bladder. ***Nature***. 20;507(7492):315-22. doi: 10.1038/nature12965. PMID:24476821
11. The Cancer Genome Atlas Research Network (2014) The somatic genomic landscape of chromophobe renal cell carcinoma. ***Cancer Cell***. 26(3):319-30. PMCID: PMC4160352
12. Strande N, Carvajal-Garcia J, Hallett R, Waters C, **Roberts SA**, Strom C, Kuhlman B, and Ramsden DA (2014) Requirements for 5'dRP/AP lyase activity in Ku. ***Nucleic Acids Research***. 42(17):11136-43. PMID: 25200085
13. Sakofsky CJ, **Roberts SA***, Malc E, Mieczkowski PA, Resnick MA, Gordenin DA, and Malkova A (2014) Break-Induced Replication Is a Source of Mutation Clusters Underlying Kataegis. ***Cell Reports***, 7(5):1640-8. PMID:24882007. (* co-first author)
14. Chan K, **Roberts SA**, Klimczak LJ, Sterling JF, Saini N, Malc EP, Kim J, Kwiatkowski DJ, Fargo D, Mieczkowski PA, Getz G, and Gordenin DA, (2015) An APOBEC3A hypermutation signature is distinguishable from the signature of background mutagenesis by APOBEC3B in human cancers. ***Nature Genetics***. 47(9):1067-72. doi: 10.1038/ng.3378. Epub 2015 Aug 10. PMID: 26258849
15. Kazanov MD, **Roberts SA**, Polak P, Stamatoyannopoulos J, Klimczak LJ, Gordenin DA, Sunyaev SR (2015) APOBEC-Induced Cancer Mutations Are Uniquely Enriched in Early-Replicating, Gene-Dense, and Active Chromatin Regions. ***Cell Reports***.

13(6):1103-9. doi: 10.1016/j.celrep.2015.09.077. Epub 2015 Oct 29. PMID: 26527001
16. Hoopes JI, Cortez LM, Mertz TM, Malc EP, Mieczkowski PA, and **Roberts SA**, (2016) APOBEC3A and APOBEC3B Preferentially Deaminate the Lagging Strand Template during DNA Replication. ***Cell Reports***. 14(6):1273-82. PMID:26832400.
17. Wyatt DW, Feng W, Conlin MP, Yousefzadeh MJ, **Roberts SA**, Mieczkowski P, Wood RD, Gupta GP, and Ramsden DA (2016) Essential roles for Polymerase θ mediated end-joining in repair of chromosome breaks. ***Molecular Cell***. 63(4):662-673. doi:http://dx.doi.org/10.1016/j.molcel.2016.06.020.
18. Mao P, Smerdon MJ, **Roberts SA**, and Wyrick JJ (2016) Chromosomal Landscape of UV Damage Formation and Repair at Single Nucleotide Resolution. ***PNAS***. 113(32):9057-62. doi: 10.1073/pnas.1606667113. Epub 2016 Jul 25.
19. Saini N, **Roberts SA**, Klimczak LJ, Chan K, Grimm SA, Dai S, Fargo DC, Boyer JC, Kaufmann WK, Taylor JA, Lee E, Cortes-Ciriano I, Park PJ, Schurman SH, Malc EP, Mieczkowski PA, and Gordenin DA (2016) The impact of environmental and endogenous damage on human somatic mutation load. ***PLoS Genetics***. 12(10):e1006385. doi: 10.1371/journal.pgen.1006385. PMID:27788131
20. The Cancer Genome Atlas Research Network (2017) Integrated genomic and molecular characterization of cervical cancer. ***Nature***. 543:378-384. doi:10.1038/nature21386.
21. Saini A, **Roberts SA**, Sterling JF, Malc EP, Mieczkowski PA, and Gordenin DA (2017) APOBEC3B cytidine deaminase targets the non-transcribed strand of tRNA genes in yeast. ***DNA Repair***. 53:4-14. doi: 10.1016/j.dnarep.2017.03.003. Epub 2017 Mar 21. PubMed PMID: 28351647
22. Hoopes JI, Hughes AL, Hobson LA, Cortez LM, Brown AJ, and **Roberts SA** (2017) Avoidance of APOBEC3B-induced mutation by error-free lesion bypass. ***Nucleic Acids Research***. 45(9):5243-5254. doi: 10.1093/nar/gkx169. PubMed PMID: 28334887.
23. Mao P, Brown AJ, Malc EP, Mieczkowski PA, Smerdon MJ, **Roberts SA***, and Wyrick JJ (2017) Genome-wide Maps of Alkylation Damage, Repair, and Mutagenesis Reveal Mechanisms of Mutational Heterogeneity. ***Genome Research***. 27: 1674-1684; Published in Advance September 14, 2017, doi:10.1101/gr.225771.117. PMID: 28912372 PMCID: PMC5630031. (* co-corresponding author)
24. Khodaverdian V, Hanscom T, Yu A, Yu T, Mak V, Brown AJ, **Roberts SA**, and McVey M (2017) Secondary-structure forming sequences drive SD-MMEJ repair of DNA double-strand breaks. ***Nucleic Acids Research***. Published Nov. 7. https://doi.org/10.1093/nar/gkx1056
25. Martin M, Hiroyasu A, Guzman RM, **Roberts SA**, Goodman AG. (2018) Analysis of Drosophila STING Reveals and Evolutionarily Conserved Antimicrobial Function. ***Cell Reports***. 23(12):3537-3550.e6. doi: 10.1016/j.celrep.2018.05.029. PMID:29924997
26. Mao P, Brown AJ, Esaki S, Lockwood S, Poon GMK, Smerdon MJ, **Roberts SA***, and Wyrick JJ (2018) ETS transcription factors induce a unique UV damage signature that drives recurrent mutagenesis in melanoma. ***Nature Communications***. 9, Article number: 2626. (* co-corresponding author)
27. Layer JV, J. Cleary P, Brown AJ, Stevenson KE, Morrow SN, Van Scoyk A, Blasco RB, Karaca E, Meng F, Frock RL, Tivey T, Kim S, Fuchs H, Chiarle R, Alt FW, **Roberts SA**, Weinstock DM, and Day TA (2018) Parp3 promotes long-range end-joining in murine cells. ***PNAS***. 115(40):10076-10081. doi: 10.1073/pnas.1801591115. Epub 2018 Sep 13. PMID:30213852.
28. Brown AJ, Mao P, Smerdon MJ, Wyrick JJ, and **Roberts SA** (2018) Nucleosome positions establish an extended mutation signature in melanoma. ***PLoS Genet*** 14(11): e1007823. https://doi.org/10.1371/journal.pgen.1007823. PMCID:PMC6287878
29. Rosenbaum JC, Bonilla B, Herken BW, Mertz TM, Kazemier HG, Pressimone CA,

Ratterman TC, MacNary E, Godin SK, Van Houten B, Normolle DP, Das SR, Paeschke K, **Roberts SA**, Van Bemark AP, and Bernstein KA (2019) The Rad51 paralogs facilitate a novel DNA strand specific damage tolerance pathway. ***Nature Communications***. Aug 5;10(1):3515. doi: 10.1038/s41467-019-11374-8. PMID:31383866

30. Elango R, Osia B, Harcy V, Malc E, Mieczkowski P, **Roberts SA***, and Malkova A* (2019) Repair of base damage within Break-Induced Replication intermediates promotes kataegis-associated with chromosome rearrangements. ***Nucleic Acids Research***. Aug 8. pii: gkz651. doi: 10.1093/nar/gkz651. PMID:28334887. (* co-corresponding author)
31. Cortez LM, Brown AL, Dennis MA, Collins CD, Brown AJ, Mitchell D, Mertz TM*, and **Roberts SA***. APOBEC3A is a prominent cytidine deaminase in breast cancer. (2019) ***PLoS Genet***. published 16 Dec 2019. https://doi.org/10.1371/journal.pgen.1008545. PMID:31841499. (* co-corresponding author)
32. Mao P, Smerdon MJ, **Roberts SA**, and Wyrick JJ. Asymmetric Repair of UV Damage in Nucleosomes Induces a DNA Strand Polarity to Somatic Mutations in Skin Cancer. (2020) ***Genome Res***. published Jan;30(1):12-21. doi: 10.1101/gr.253146.119. Epub 2019 Dec 23. PMID:31871068.
33. Carvajal-Garcia J, Cho J, Carvajal-Garcia P, Feng W, Wood RD, Sekelsky J, Gupta GP, **Roberts SA**, and Ramsden DA. Mechanistic Basis for Microhomology Identification and Genome Scarring by Polymerase Theta. (2020) ***PNAS***. 117(15):8476-8485. doi: 10.1073/pnas.1921791117. Epub 2020 Mar 31. PMID:32234782.
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35. Laughery MF, Brown AJ, Bohm KA, Sivapragasam S, Morris HS, Tchmola M, Washington AD, Mitchell D, Mather S, Malc EP, Mieczkowski PA, **Roberts SA***, and Wyrick JJ*. Atypical UV photoproducts induce novel mutation signatures that promote driver mutations in melanoma. (2020) ***Cell Reports***. Nov 17;33(7):108401. doi: 10.1016/j.celrep.2020.108401. PMID: 33207206. (* co-corresponding author)
36. Sivapragasam S, Stark B, Albrecht AV, Mao P, Emehiser RG, **Roberts SA**, Hrdlicka, PJ, Poon GMK, and Wyrick JJ (2021) CTCF binding modulates UV damage formation to promote mutation hotspots in melanoma. ***EMBO J***. Sep 6;e107795. doi: 10.15252/embj.2021107795.
37. Brown AL, Collins CD, Thompson S, Coxon M, Mertz TM, and **Roberts SA** (2021) Single-stranded DNA binding proteins influence APOBEC3A substrate preference. ***Sci Rep***. Oct 25;11(1):21008. doi: 10.1038/s41598-021-00435-y. PMID: 34697369
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39. Antony J, **Roberts SA**, Wyrick J, and Hinz J (2021) Cas9 binding inhibits the initiation of base excision repair in vitro. ***DNA Repair (Amst)***. Nov 20;109:103257. doi: 10.1016/j.dnarep.2021.103257. PMID: 34847381.
40. Al Soodani A, Wu X, Her S, Saul M, Kelp N, Xu Y, Brown AJ, **Roberts SA**, and Her C (2022) hMSH5 Regulates NHEJ and Averts Excessive Nucleotide Alterations at Repair Joints. ***Genes (Basel)***. Apr 11;13(4):673. doi: 10.3390/genes13040673. PMID: 35456479 PMCID: PMC9026759

41. Hanscom T, Woodward N, Batorsky R, Brown AJ, **Roberts SA**, and McVey M (2022) Characterization of sequence contexts that favor alternative end joining at Cas9-induced double-strand breaks. ***Nucleic Acids Res***. 2022 Jul 12;gkac575. doi: 10.1093/nar/gkac575. PMID: 35819195.
42. Talukdar PK, Crockett TM, Gloss LM, Huynh S, **Roberts SA**, Turner KL, Lewis STE, Herup-Wheeler TL, Parker CT, Konkel ME. (2022) The bile salt deoxycholate induces Campylobacter jejuni genetic point mutations that promote increased antibiotic resistance and fitness. ***Front Microbiol***. 2022 Dec 21;13:1062464. doi: 10.3389/fmicb.2022.1062464. eCollection 2022. PMID: 36619995
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<u>In Review</u>

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In Preparation

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Non-Peer Reviewed Publications

Review Articles

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Books and Chapters

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2. Mertz TM, Kockler ZW, Coxon M, Cordero C, Raval AK, Brown AJ, Harcy V, Gordenin DA and **Roberts SA**. Defining APOBEC-induced mutation signatures and modifying activities in yeast. ***Methods in Enzymology***. https://doi.org/10.1016/bs.mie.2024.11.041.

Other Scholarly Publications

Abstracts

Patents Issued or Pending: None

Other Creative Activities

Quality Improvement and Patient Safety Activities

Not Applicable

SUMMARY OF SCHOLARLY ACTIVITIES

Program in somatic cell mutagenesis during cancer development: Mutation and recombination of DNA sequences drive evolution and contribute to human diseases including cancer. My research program combines biochemistry, genetics, cell biology, and genomics to understand how DNA damage and DNA repair mechanisms sculpt the mutational landscape across cancer genomes. My current research activities focus on the following areas.

APOBEC-induced mutation in human cancers: APOBECs are cytidine deaminases that catalyze the conversion of cytidine to deoxyuridine in single stranded (ss) DNA and have normal roles in adaptive and innate immunity[1]. My work, as well as that of others, has demonstrated these enzymes also promote somatic cell mutagenesis[2-9], which underlies tumorigenesis. APOBEC-induced mutations are prominent in ~20% of sequenced human tumors[2] and contribute to both cancer progression through *PIK3CA* mutation[10] and tumor relapse by increasing genetic heterogeneity[11]. We are investigating the following topics to better understand the causes and consequences of dysregulated APOBEC activity during cancer development. The following work has been supported by NIH funded R00, two R01 awards, and a DOD Breast Cancer Break-through award.

Defining targets of APOBEC-mediated genome instability: My research group determined that in proliferating cells, the lagging strand template during DNA replication is the major substrate for mutagenic APOBEC-activity by examining patterns of mutations in sequenced genomes and genetic reporters in model systems[12]. Similar mutation patterns indicate that the lagging strand template is also the primary APOBEC substrate in human cancers[11,13] likely due

to high levels of replication in these cells. However, in pre-cancerous cells other ssDNA targets are likely. We are applying yeast and human cell genomics, genetic reporters, and biochemical techniques to further quantify the contribution of different sources of ssDNA and DNA secondary structures to APOBEC-induced mutations[12,14,15].

Determining the identity of APOBEC genome mutators: The human genome encodes 11 APOBEC family members[1]. We have accumulated substantial evidence indicating that APOBEC3A (A3A) is the primary APOBEC that causes genome instability in breast cancer[4,5]. Part of this effort has involved developing methods to map deoxyuridine lesions genome-wide at single nucleotide resolution and measuring mutation rates in cell lines with CRISPR-mediated APOBEC knockouts and/or overexpressing individual APOBECs. We are completing these experiments with a novel high-throughput mutation reporter my group has developed. This reporter has the potential to revolutionize the study of mutagenesis in mammalian cells and to be widely used in studies outside of the scope of APOBEC biology. Additionally, we are utilizing genomics, biochemical, and targeted cellular biology approaches to determine why A3A is transcriptionally upregulated in breast cancers as well as how A3A is regulated by post-translational modifications (K30 acetylation) and protein-protein interactions that we have identified. We have determined that in human epithelial cells and breast cancer cells, A3A is regulated by the proteosome[16]. We are particulary interested in understanding how this process occurs and whether its dysregulation in cancer development or by anti-cancer proteosome inhibitor therapeutics enhances A3A mutagenesis of cancer genomes.

Characterizing modulators of APOBEC activity: Due to their highly mutagenic nature, cells employ multiple mechanisms to protect the genome from detrimental deamination by APOBECs. We have begun characterizing factors that influence APOBEC mutagenesis in yeast by screening to identify gene deletions that specifically elevate APOBEC-induced mutation at the *CAN1* gene or by testing chemical exposures that augment APOBEC-induced mutation[17]. Using these methods, we have identified key factors that modulate APOBEC-induced mutagenesis: 1) a novel homologous recombination mediated repair mechanism for APOBEC-induced damage in front of nascent lagging strand synthesis[18,19] 2) a set of chromatin remodeling factors that limit APOBEC mutagenesis, 3) replication fork stability factors, and 4) chemical or genetic induction of replication stress[12].

Characterizing the spectrum of APOBEC-induced mutation: When editing genomes, APOBECs can induce tens of thousands of base substitutions over the course of a tumor's development [20]. A subset of these mutations form discrete groups of localized hypermutation, termed "kataegis" [6] or "mutation clusters[9]." Mutations in kataegis events are "strand-coordinated" (*i.e.* involving cytidines only on one DNA strand) and are frequently located near chromosomal translocation break points, which indicates that the mutations were likely induced simultaneously along with a recombination event[6,9]. While kataegis appears to occur by APOBECs mutagenizing ssDNA formed during complex recombination events, what initiates these events is unclear. We have found that activity of A3A on break-induced replication intermediates creates kataegis-like events and induces further recombination[21,22]. Additionally, we have recently discovered that A3A and APOBEC3B induce insertion/deletion mutations in addition to their canonical base substation signature. We have determined in yeast that these mutations involve recruitment of trans-lesion synthesis (TLS) polymerases to abasic sites derived from Ung1 glycolytic cleavage of APOBEC-induced deoxyuridine lesions. Once recruited, these TLS polymerases either extend from a primer slipped across from the abasic site or incorrectly insert across from the abasic site and subsequently delete a base in a neighboring homopolymer run. We are currently assessing whether similar APOBEC-induced insertion/deletion mutation occur in human cells and tumors. If observed in tumors, we will assess if these particularly deleterious mutations contribute to tumor suppressor inactivation during cancer development.

UV-induced mutagenesis in skin cancer: My group has expanded our efforts to understand the determents of cancer genome evolution beyond those related to dysregulation of APOBECs. In collaboration with Dr. John Wyrick's lab at WSU, we have developed methods to map, genome-wide, the position of ultraviolet (UV) light photoproducts (CPDs, 6-4 photoproducts, and atypical photoproducts) [23-25] in yeast and cultured human cells and correlate regional densities of lesion formation and repair to the density of mutation. Using these methods, we have found that the structure of the nucleosome and some histone modifications significantly impact UV-induced damage formation, repair, and mutagenesis[23-26]. Moreover, we surprisingly found that Ets family transcription factors dramatically elevate UV lesion formation at their binding sites, resulting in extremely frequent mutation of these sites in human melanomas[23]. Some of these specific mutations are highly recurrent and may impact expression of cancer-related genes, suggesting these events are important for promoting disease progression. Utilizing whole genome sequencing of UV irradiated yeast and a novel method for mapping non-CPD UV lesions, we have identified non-canonical UV lesions that occur at TA and AC dinucleotides that are likely the source of some skin cancer specific *BRAF* driver mutations[27]. We are currently investigating the identity of these non-canonical photoproducts and assessing how mutagenic TLS bypass of these UV lesion occurs in yeast[28] and human cancer cells. This work is supported by an NIEHS-funded multiple PI R01 award.

Alkylation-induced mutagenesis: Also in collaboration with the Wyrick lab, we have modified our methods for mapping the sites of UV lesions to identify where DNA alkylation lesions[26] are most likely to occur and are most efficiently repaired by base excision repair. We additionally utilized whole genome sequencing of methyl methanesulfonate treated yeast to determine how these regional differences in alkylation damage and repair translate into mutation distributions. We determined that like UV lesions, alkylation repair is modified by nucleosome structure and histone modification. We determined that in the absence of base excision repair, transcription coupled nucleotide excision repair serves as a backup repair pathway for removal of lesions on the transcribed strand of yeast genes, resulting in strong transcriptional asymmetries of alkylation-induced mutations. These asymmetries are also observed in the mutations of telomazolamide (TMZ) treated cancers. Additionally, we discovered that Shu complex-mediated homology directed bypass is the primary mechanism for repair of single strand specific alkylation lesions in yeast[29]. This is likely because yeast lack AlkB mediated direct-reversal mechanisms present in many other organisms. This suggests that recombination many be an important survival mediator in tumors treated with TMZ that lack direct reversal like Mgt1 or AlkB. We will also begin to modify these methods to determine what factors control the distribution of oxidative damage and its associated mutations.

Transcription-associated mutagenesis: In human cells, perturbing transcription and inducing DNA-topoisomerase adducts cause DNA damage. However, the mechanisms generating transcription- and DNA-topoisomerase adduct-associated mutations in human cells, the types of mutations caused by these processes, and their contribution to mutations in cancer are poorly understood. Using our novel mutation reporter in human cells, we have observed a mutation spectrum dominated by 2- to 5-base pair (bp) deletions and distinct larger deletions that mirrors DNA topoisomerase 1-dependent, transcription-associated mutagenesis in yeast. This spectrum indicates transcription-induced DNA-topoisomerase adducts produce these mutations in human cells. We additionally found that 2- to 5-bp deletions are enriched within highly expressed genes in primary breast cancers, suggesting DNA-topoisomerase adducts are important contributors to cancer etiology as deletions of ≥ 2 bp constitute between 3% and 12% of all inactivating mutations in tumor suppressor genes. We are characterizing the basic determinants of transcription-associated, DNA-topoisomerase adduct-induced mutagenesis in

human cells and assessing its contribution to mutagenesis in cancer, using mutational analyses (both with reporters and whole genome sequencing) and a novel method to map DNA-TOP1 adducts genome-wide at single nucleotide resolution. The distributions of these adducts and mutations will be further compared to the distribution DNA-TOP1 adduct signature mutations in sequenced human tumors to estimate the contribution of DNA-TOP1 adducts to mutagenesis in cancer. This activity is supported by a NCI-funded R21 grant.

High-throughput characterization of mutagenic DNA double strand break repair: Errors made during DNA double strand break repair are another common source of mutation observed in cancer genomes. These types of mutations are usually insertion/deletion mutations generated during end joining processes and occur in higher amounts in tumors deficient in homologous recombination (e.g. BRCA1/2 deficient breast or ovarian cancers). Despite the general understanding that many deletions are mediated by microhomologies in the DNA flanking break sites, little is known about the specific proteins that cause these errors and why their utilization can be elevated in cancers. To help address these gaps in knowledge, we have leveraged our expertise in mechanisms of DSB repair and next generation sequencing analysis to create a novel set of scripts to identify the specific sequences generated by DSB repair at site specific breaks[30]. These scripts enable the analysis of tens of thousands of repair events from next-generation sequencing data and classify specific types of errors made during these processes[31-33]. Utilizing this software, we have contributed significantly to the understanding of how DNA pol theta functions in a novel DSB repair pathway[31,34,35], leading to characteristic insertion/deletion mutations in cancers with recombination deficiencies[34]. We are currently increasing the throughput of this process to evaluate large numbers of different sequence contexts that breaks could occur in to establish rules for pol theta-based errors based on the surrounding sequence.

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31. **Khodaverdian, V.Y., Hanscom, T., Yu, A.M., Yu, T.L., Mak, V., Brown, A.J., Roberts, S.A., and McVey, M. (2017). Secondary structure forming sequences drive SD-MMEJ repair of DNA double-strand breaks. Nucleic Acids Res *45*, 12848-12861. 10.1093/nar/gkx1056.**

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INVITED PRESENTATIONS

Regional

2010	Genetics and Environmental Mutagenesis Society Fall Meeting "Localized Hypermutability Caused by Chronic Alkylation Damage to a Eukaryotic Genome"	Durham, NC

2011	Small Eukaryotes Meeting "Pathways of Damage-Induced Localized Hypermutability"	Research Triangle Park, NC
2011	NIEHS Genomics Day, NIEHS "Permanent Records of Transient Hyper-mutation in Yeast and Human Genomes"	Research Triangle Park, NC
2012	Southeast Regional Yeast Meeting "Permanent Records of Transient Hyper-mutation in Yeast and Human Genomes"	Atlanta, GA
2012	Next Generation Sequencing Group, North Carolina Biotechnology Center "A record of transient hyper-mutation in yeast and in human cancers revealed through next-generation sequencing"	Research Triangle Park, NC
2012	DNA Repair and Replication Focus Group, University of North Carolina "Permanent Records of Transient Hyper-mutation in Yeast and Human Genomes"	Chapel Hill, NC
2013	The Institute of Marine and Environmental Technology (IMET), University of Maryland "Dissecting sources of genome instability in cancer"	Baltimore, MD
2013	Department of Pharmacology & Toxicology, University of Texas "Dissecting sources of genome instability in cancer"	Austin, TX
2013	Center for Research on Environmental Disease, University of Kentucky "Dissecting sources of genome instability in cancer"	Lexington, KY
2013	Duke Center for DNA and Genome Stability, Duke University "Hyper-mutation of single stranded DNA across yeast and cancer genomes"	Durham, NC
2014	Department of Biochemistry and Molecular Biology, Johns Hopkins University "Dissecting sources of genome instability in cancer"	Baltimore, MD
2014	The Department of Biological Sciences, Clemson University "Dissecting sources of genome instability in cancer"	Clemson, SC

2014	Department of Pharmacological Sciences, Stony Brook University "Dissecting sources of genome instability in cancer"	Stony Brook, NY
2014	Department of Molecular Genetics & Microbiology, University of New Mexico "Dissecting sources of genome instability in cancer"	Albuquerque, NM
2014	Department of Biochemistry and Molecular Biology, Penn State University "Dissecting sources of genome instability in cancer"	Hershey, PA
2014	School of Molecular Biosciences, Washington State University "Dissecting sources of genome instability in cancer"	Pullman, WA
2015	Cancer and Aging Focus Group, Washington State University "Genome Editing by APOBEC Cytidine Deaminases"	Pullman, WA
2015	DNA Repair in Chromatin Symposium: The First 40 years (and Beyond), Washington State University "Inhibition of DNA repair in ssDNA sculpts mutation landscapes in human cancers"	Pullman, WA
2015	Department of Molecular and Computational Biology, University of Southern California "Dissecting mechanisms of cancer genome instability through analysis of clustered mutations"	Los Angeles, CA
2017	Department of Biochemistry and Molecular Biology, University of Kansas Medical Center "Landscape of APOBEC Mutagenesis in cancer"	Kansas City, KS
2018	School of Molecular Biosciences, Washington State University "Mutation Signatures, Distributions, and Hot Spots: Causes and Consequences of Genetic Instability of Cancer"	Pullman, WA
2018	Center for Reproductive Biology Annual Retreat, Washington State University "APOBEC-induced mutation in breast cancers"	Orofino, ID
2018	Eppley Institute, University of Nebraska Medical Center "Cancer Genome Editing by APOBEC Cytidine Deaminases"	Omaha, NE

2018	Department of Biology, Tufts University "Protein-DNA interactions modulate UV lesion formation and mutagenesis in melanoma"	Medford, MA
2018	School of Molecular Biosciences, Washington State University "Transformers: Mutagenic Effects of APOBEC Cytidine Deaminases"	Pullman, WA
2019	Basser Center for BRCA, University of Pennsylvania "Dissecting Mechanisms of Mutation in Breast Cancers"	Philadelphia, PA
2019	Northwest Reproductive Sciences Symposium "APOBEC3A induces genetic instability during breast cancer development"	Astoria, OR
2021	Gordenin 70th Birthday Symposium, NIEHS "Atypical UV photoproducts are associated with melanoma driver mutations"	Virtual Presentation
2021	Petes-Jinks-Robertson Lab Meeting, Duke University "Non-canonical UV-induced mutations are associated with melanoma driver mutations"	Virtual Presentation
2022	WSU Emeritus Society, Washington State University "Understanding cancer through mutation signatures"	Virtual Presentation
2022	Department of Biological Sciences, University of North Carolina - Charlotte "Fingerprints in DNA: mutation patterns reveal impacts of genome instability on cancer"	Charlotte, NC
2022	Center for Environmental Toxicology, North Carolina State University "APOBEC-induced mutation during cancer development and treatment"	Raleigh, NC
2022	Department of Microbiology and Molecular Genetics, University of Vermont "Fingerprints in DNA: mutation patterns reveal impacts of genome instability on cancer"	Burlington, VT
2022	Department of Biochemistry and Molecular Biology, Medical University of South Carolina "Fingerprints in DNA: mutation patterns reveal impacts of genome instability on cancer"	Virtual Presentation

2022	University of Vermont Cancer Center Retreat, University of Vermont "Insertion and deletion mutagenesis induced by APOBEC3A and APOBEC3B"	Burlington, VT
2023	Genome Integrity & Structural Biology Lab, NIEHS, "APOBEC-induced mutagenesis from tumor initiation through cancer therapy"	Research Triangle Park, NC
2024	University of Vermont Redox Biology and Pathology (RBP) Seminar Series "An expanded GO network in human cells for removal of oxidative DNA damage."	Burlington, VT
2024	Smerdon-Reeves Symposium, Washington State University "Cartography of oxidative DNA damage and mutation"	Virtual Presentation
2024	MMG Faculty Seminar, University of Vermont "Cartography of oxidative DNA damage and mutation"	Burlington, VT
2025	UVMCC Cancer Cell Meeting "Mutagenic Consequences of Atypical UV Photoproducts"	Burlington, VT
2025	Department of Biochemistry and Structural Biology, University of Texas Health Science Center – San Antonio "Responses of APOBEC3A to cancer therapeutics"	San Antonio, TX

National

International

2010	Gordon Research Conference in Mutagenesis "Localized Hypermutability Caused by Chronic Alkylation Damage to a Eukaryotic Genome"	Colby College, ME
2013	FASEB Science Research Conferences Genetic Recombination & Genome Rearrangements "Mutagenesis by single strand DNA-specific APOBEC cytidine deaminases associates with chromosome rearrangements and pervades the genomes of multiple human cancer types"	Steamboat, CO

2014	Gordon Research Conference in Mutagenesis "Hypermutation of ssDNA in yeast and human cancers"	Girona, Spain
2016	Environmental Mutagenesis and Genomics Society Annual Meeting "Landscapes of DNA Damage and Mutagenesis"	Kansas City, MO
2020	Gordon Research Conference in DNA Damage, Mutation, and Cancer "APOBEC3A is a prominent cytidine deaminase active in human cancers"	Ventura, CA
2020	1st International Conference on Base Editing – Enzymes and Applications "APOBEC3A is a prominent cytidine deaminase active in human cancers"	Palm Springs, CA
2021	APOBEC Translational Working Group "Genetic Modifiers of APOBEC Mutagenesis"	Virtual Presentation
2021	2nd International Conference on Base Editing – Enzymes and Applications "Genetic Modifiers of APOBEC Mutagenesis"	Virtual Meeting
2022	APOBEC Translational Working Group "Impacts of Proteasome Dysfunction on APOBEC3A"	Virtual Presentation
2022	Gordon Research Conference in Mutagenesis "Insertion and deletion mutagenesis induced by APOBEC3A and APOBEC3B"	Newry, ME
2024	AACR Special Conference on DNA Damage Repair "Polymerase-based bypass of atypical UV photoproducts"	Washington DC
2024	Gordon Research Conference in Mutagenesis "A dysfunctional ubiquitin-proteosome system influences APOBEC3A expression"	Waterville Valley, NH
2024	American Society for Photobiology "Mutagenic Bypass of Atypical UV Photoproducts"	Chicago, IL
2025	6th International Conference on Base Editing – Enzymes and Applications "Mutagenic Potential of APOBEC3A orthologs"	Palm Springs, CA
2025	APOBEC Translational Working Group "A machine learning approach to distinguish APOBEC3A and APOBEC3B mutated cancer cells"	Virtual Presentation