Title: A B cell based immune therapy leads to a special mechanism incinerating triple negative breast cancers and conquering resistance to anti-PD1. Daniel Hollern PhD, Assistant Professor, Salk Institute

Significance In triple-negative breast cancer (TNBC), immunotherapy is not fully potentiated. While the majority of the immunotherapy field is focused on T cells, our lab has shown that responses to T cell checkpoint therapy requires B cells and antibody. Yet, the reliance on helper function from CD4+ T cells for B cell activation in T cell based therapy settings creates an **unsolved problem** for many cases of TNBC that lack tumor infiltrating B cells and T cells. This leaves many patients (particularly those whose TNBC lack the conditions needed for T cell activation) without the comprehensive therapy proven to incite immune responses capable of attacking their unique tumors. In developing **a novel concept** in tumor immune therapy that significantly **impacts these patients**, we have a found a way to directly elicit an anti-tumor B cell response that activates killer CD8+ T cells without the need for CD4+ T cell help. **This signifies a major innovation to therapy** as our results clearly show that CD40 agonist can replace helper CD4+ T cells and activate B cells capable of overcoming de novo and acquired resistance to anti-PD1 therapy. This is **significant to a large percentage of TNBC patients** whose tumors lack conditions for CD4+ T cells activation. We will reach this large group of un-aided patients and radicalize TNBC therapy by delivering therapeutic mechanisms explaining how B cell activation unleashes anti-tumor immunity.

HYPOTHESIS Our **overall hypothesis** is that CD40 agonist is mimicking CD4+ T cell CD40 ligand-mediated positive selection of B cells in the light zone of the tumor draining lymph node. We hypothesize a broadened B cell response that delivers anti-tumor immunity via multimodal activation of CD8+ T cells and B cell secreted antibodies that engage antibody dependent cellular phagocytosis. We posit that immune checkpoint blockade (ICB) synergizes with these responses by causing CD4+ T cell responses that work with CD40 agonist to further B cell cycling to dark zones and increase positive selection of B cell clones in the light zone of lymph nodes.

AIM 1. To determine function of CD40 activated B cell responses in mouse models of TNBC.
AIM 2. To determine how CD40 activated B cells incite CD8+ T cell responses during anti-tumor immunity to mouse TNBCs.
AIM 3. To determine the function of anti-histone antibodies in immune responses to mouse TNBCs.

STUDY DESIGN
In **aim 1**, we will test the **hypothesis** that CD40 agonist enforces positive selection of light zone B cells which function to organize anti-tumor lympho-myeloid aggregates in the tumor stroma. We posit that ICB broadens the positive selection of these B cell responses further and encourages dark zone and light zone activity, producing active GCs and TLS. A second new finding our lab has made is that these anti-tumor B cell responses have a direct role in promoting cytotoxic CD8+ T cell responses in the tumor. This raises the question as to how B cells activate CD8+ T cells in response to CD40 agonist without CD4+ T cell help. Based on our initial scSEQ data, we **hypothesize** that B cells activate CD8+ T cells multi-modally with antigen and activation factors. In **aim 2,** we will computationally and functionally test how B cell responses activate the CD8+ T cell response. We also found that CD40 agonist changes the profile of tumor reactive antibodies in the serum. Mapping mouse serum antibody responses to targets by TMT- Mass Spec and ELISA demonstrated that these antibodies recognize histone proteins. As these antibodies react to areas of cell death, we **hypothesize** they function in ADCP to help activate CD8+ T cells. We will define the function of histone specific antibodies in **Aim 3**.

IMPACT. **Aim 1** will have **immediate impact** by creating a basis to employ CD40 agonist tactically or broadly in TNBC to elicit anti-tumor B cell responses and synergy with anti-PD1 therapy. **Long term**, our results linking the B cells and BCR/antibody sequences tied to these anti-tumor responses will enable us to discover effective antigens to hard wire B cell or antibody responses by antibody cloning or B cell BCR engineering. **Aim 3 of this proposal will develop one such discovery**. **Aim 2** will deliver the first insights into how B cells activate killer CD8+ without CD4+ T cell help. This **will impact treatment** to use B cells to ensure CD8+ T cells attack TNBCs. This will **significantly impact** patients whose tumors lack MHC II associated antigens and cannot thus rely on CD4+ T cell help to induce anti-tumor responses. The **impact of Aim 3** will derive from delineation of how systemic antibodies to histones function in tumor immunity. Further **impact** of this aim will derive from defining the extent to which these antibodies and specific histone antigens are potentially important therapeutic targets. As such, we will deliver beneficial concepts with proven efficacy to patients and clinicians thus **accelerating progress** in developing significantly improved immunotherapy holding broader patient applicability and efficacy.

Daniel P Hollern, Ph.D.

CONTACT INFORMATION

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RESEARCH SUMMARY

My research uses a multi-disciplinary approach to investigate mechanisms of response to therapy, immune cell dynamics in human cancers, and mechanisms controlling tumor metastasis. By integrating expertise in functional genomics with mouse models and experimental biology, the goal of my research is to improve treatment strategies for cancer patients.

EDUCATION and TRAINING

Assistant Professor, Salk Cancer Center, NOMIS Center for Immunobiology and Microbial Pathogenesis

The Salk Institute for Biological Studies, La Jolla, California

2021-Current Position

Postdoctoral Research Fellow, Cancer genetics and genomics, 2015-2020
University of North Carolina, Chapel Hill, NC
Mentor: Charles M Perou, Ph.D.
Areas of Study: Computational Genomics, Immunogenomics, Tumor Immunology, Breast Cancer, Testicular Cancer

Ph.D., Cell and Molecular Biology, 2015
Michigan State University, East Lansing
Mentor: Eran R. Andrechek, Ph.D.
Thesis: *A study of breast cancer heterogeneity and molecular mechanisms of metastasis*

Bachelors of Science, Biology, 2009
Grand Valley State University, Allendale, MI

31 PUBLICATIONS

1. Swiatnicki, M. R., Rennhack, J. P., Ortiz, M. M., **Hollern, D. P**., Perry, A. V., Kubiak, R., ... & Andrechek, E. R. (2022). Elevated phosphorylation of EGFR in NSCLC due to mutations in PTPRH. *PLoS Genetics*, *18*(9), e1010362.
2. Mara Gilardi, Monika Ramos, **Daniel Hollern.** B cells secrete GABA, which provokes a pro-tumor immune microenvironment. 2022. ***Cancer Cell***. https://doi.org/10.1016/j.ccell.2021.12.007
3. Celine Laumont , Allyson Banville, Mara Gilardi**, Daniel Hollern**, and Brad Nelson. B cells in cancer: clinical impact, immunological mechanisms and therapeutic opportunities. 2022. ***Nature Reviews Cancer*.** https://doi.org/10.1038/s41568-022-00466-1
4. Mara Gilardi, Robert Saddawi-Konefka, Victoria H Wu, Miguel Angel Lopez-Ramirez, Zhiyong Wang, Fernando Soto, Dana J Steffen, Marco Proietto, Zbigniew Mikulski, Haruka Miki, Andrew B Sharabi, Daniel Kupor, Ricardo Rueda, **Daniel Hollern**, Joseph Wang, and J. Silvio Gutkind. Microneedle-mediated intratumoral delivery of anti-CTLA-4 promotes cDC1-dependent eradication of oral squamous cell carcinoma with limited irAEs. 2022. ***Molecular Cancer Therapeutics***. https://doi.org/10.1158/1535-7163.MCT-21-0234
5. He, Y., Wang, L., Wei, T., Xiao, Y.T., Sheng, H., Su, H., **Hollern, D.P**., Zhang, X., Ma, J., Wen, S. and Xie, H., 2021. FOXA1 overexpression suppresses interferon signaling and immune response in cancer. ***The Journal of Clinical Investigation***
6. Garay, J.P., Smith, R., Devlin, K., **Hollern, D.P**., Liby, T., Liu, M., Boddapati, S., Watson, S.S., Esch, A., Zheng, T. and Thompson, W., 2021. Sensitivity to targeted therapy differs between HER2-amplified breast cancer cells harboring kinase and helical domain mutations in PIK3CA. ***Breast Cancer Research***, *23*(1), pp.1-17.

7. Bai, F., Wang, C., Liu, X. *et al.* Loss of function of BRCA1 promotes EMT in mammary tumors through activation of TGFβR2 signaling pathway. *Cell Death Dis* **13**, 195 (2022). https://doi.org/10.1038/s41419-022-04646-7
8. Bai, F., Liu, S., Liu, X., **Hollern, D.P**., Scott, A., Wang, C., Zhang, L., Fan, C., Fu, L., Perou, C.M. and Zhu, W.G., 2021. PDGFRβ is an essential therapeutic target for BRCA1-deficient mammary tumors. ***Breast Cancer Research***, *23*(1), pp.1-17.
9. Swarnima Singh, Nigel Lee, Igor Bado, Clark Hamor, Licheng Zhang, Sergio Aguirre, Jingyuan Hu, Yichao Shen, Yitian Xu, Yang Gao, Diego Pedroza, Na Zhao, Shu-Hsia Chen, Ying-Wooi Wan, Zhandong Liu, Jeffrey Chang, **Daniel Hollern**, Charles Perou, Xiang Zhang, and Jeffrey Rosen. Chemotherapy coupled to macrophage inhibition leads to B cell-mediated T cell memory activation and durable triple negative breast cancer regression. ***bioRxiv.*** https://www.biorxiv.org/content/10.1101/2021.02.22.432300v1.abstract
10. Swiatnicki MR, Rennhack JP, **Hollern D**, Perry AV, Kubiak R, Riveria SM, O'Reilly S, Andrechek ER. Elevated phosphorylation of EGFR in NSCLC due to mutations in PTPRH. ***bioRxiv***. 2021 Jan 1.
11. Briana To, Carson Broeker, Jing-Ru Jhan, Rachel Rempel, Jonathan P Rennhack, **Daniel Hollern**, Lauren Jackson, David Judah, Matthew Swiatnicki, Evan Bylett, Rachel Kubiak, Jordan Honeysett, Shams Reaz, Joseph R Nevins, Eran Robert Andrechek. 2021. Insight into mammary gland development and tumor prevention in a newly developed metastatic mouse model of breast cancer. ***bioRxiv***.https://www.biorxiv.org/content/10.1101/2021.09.24.461727v1.abstract
12. Crosby EJ, Acharya CR, Haddad A, Rabiola CA, Lei G, Wei J, Yang X, Wang T, Liu C, Wagner KU, Muller WJ, Chodosh LA, Broadwater G, Hyslop T, Shepherd JH, **Hollern D**, He X, Perou CM, Chai S, Ashby BK, Vincent BG, Snyder JC, Force J, Morse MA, Lyerly HK, Hartman ZC. 2020. Stimulation of Oncogene-Specific Tumor-Infiltrating T Cells through Combined Vaccine and αPD-1 Enable Sustained Antitumor Responses against Established HER2 Breast Cancer. ***Clinical Cancer Research***. https://doi.org/10.1158/1078-0432.CCR-20-0389
13. Garcia-Recio S, Thennavan A, East MP, Parker JS, Cejalvo JM, Garay JP, **Hollern DP**,He X ,Mott KR, Galván P, FanC, Selitsky SR, Coffey AR, Marron D, Brasó-Maristany F, Burgués O, Albanell J, Rojo F, Lluch A, Martinez de Dueñas E, Rosen JM, Johnson GL, Carey LA, Prat A, Perou CM. 2020. ***The Journal of Clinical Investigation***. https://doi.org/0.1172/JCI130323
14. Williams M, Liu X, Zhang Y, Reske J, Bahal D, Gohl T, **Hollern D,** Ensink E, Kiupel M, Luo R, Das R, and Xiao H. 2020. NCOA5 deficiency promotes a unique liver protumorigenic microenvironment through p21WAF1/CIP1 overexpression, which is reversed by metformin. ***Oncogene***. https://doi.org/10.1038/s41388-020-1256-x
15. Selitsky, S.R., Marron, D., **Hollern, D**., Mose, L.E., Hoadley, K.A., Jones, C., Parker, J.S., Dittmer, D.P. and Perou, C.M., 2020. Virus expression detection reveals RNA-sequencing contamination in TCGA. ***BMC genomics***, *21*(1), p.79.
16. **Hollern, D. P**. *et al.* Perou, C.M. 2019. B Cells and T Follicular Helper Cells Mediate Response to Checkpoint Inhibitors in High Mutation Burden Mouse Models of Breast Cancer. ***Cell*** 179,1191-1206.e1121, doi:10.1016/j.cell.2019.10.028
*** Immune Regulation News Top Story * Joseph S. Pagano Award**
17. **Hollern DP,** et al. Perou, C.M., 2019. A mouse model featuring tissue-specific deletion of p53 and Brca1 gives rise to mammary tumors with genomic and transcriptomic similarities to human basal-like breast cancer. ***Breast cancer research & treatment***, *174*(1), pp.143- 155.
18. **Hollern DP,** et al Andrechek, E.R., 2019. E2F1 Drives Breast Cancer Metastasis by Regulating the Target Gene FGF13 and Altering Cell Migration. ***Scientific Reports*** https://doi.org/9:10718.
19. An, Y., Adams, J.R., **Hollern DP (*Co-first author)**, Zhao, A., Chang, S.G., Gams, M.S., Chung, P.E., He, X., Jangra, R., Shah, J.S. and Yang, J., 2018. Cdh1 and Pik3ca Mutations Cooperate to Induce Immune-Related Invasive Lobular Carcinoma of the Breast. ***Cell reports***, *25*(3), pp.702-714.
20. Shen H*, Shi J*, **Hollern DP* (*Co-first author)** , TCGA working group. 2018.Integrated molecular characterization of testicular germ cell tumors. ***Cell reports*** 23.11 (2018): 3392-3406.
21. **Hollern DP,** Swiatnicki MR, Andrechek ER. 2018. Histological subtypes of mouse mammary tumors reveal conserved relationships to human cancers. ***Plos Genetics*** 14: 1 https://doi.org/10.1371/journal.pgen.1007135
22. Mukherjee, A., **Hollern DP**, Williams, O., Rayburn, T., Byrd, W., Yates, C., & Jones, J.

D. (2018). A Review of FOXI3 Regulation of Development and Possible Roles in Cancer Progression and Metastasis. ***Frontiers in cell and developmental biology***, 6, 69.

23. Tanioka, M., Mott, K.R., **Hollern, DP**, Fan, C., Darr, D.B. and Perou, C.M., 2018. Identification of Jun loss promotes resistance to histone deacetylase inhibitor entinostat through Myc signaling in luminal breast cancer. ***Genome medicine***, *10*(1), p.86.

24. Acosta, D., Bagchi, S., Broin, P.Ó., **Hollern, DP**, Racedo, S.E., Morrow, B., Sellers, R.S., Greally, J.M., Golden, A., Andrechek, E. and Wood, T., 2016. LPA receptor activity is basal specific and coincident with early pregnancy and involution during mammary gland postnatal development. ***Scientific reports***, *6*, p.35810.

25. Lunt SY, Muralidhar V, Hosios AM, Israelsen WJ, Gui DY, Newhouse L, Ogrodzinski M, Hecht V, Xu K, Acevedo PN, **Hollern DP**, Bellinger G, Dayton TL, Christen S, Elia I, Dinh AT, Stephanopoulos G, Manalis SR, Yaffe MB, Andrechek ER, Fendt SM, Vander Heiden MG. Pyruvate kinase isoform expression alters nucleotide synthesisto impact cell proliferation. ***Molecular Cell***. Volume 57, Issue 1, 8 January 2015, Pages 95–107

26. **Hollern DP,** Andrechek ER. 2014. A Genomic Analysis of Mouse Models of Breast Cancer Reveals Important Molecular Features of Individual Mouse Models and Relationships to Human Breast Cancer. ***Breast Cancer Research*** 16:R59
*** Editors pick ** Highly accessed *** Top 5 for most downloaded papers in 2014**

27. **Hollern DP,** Honeysett J,Cardiff RD, Andrechek ER. 2014. The E2F transcription factors regulate tumor development and metastasis in a mouse model ofmetastatic breast cancer. ***Molecular and Cellular Biology***.

28. Chen F, Li A, Gao S, **Hollern DP,** VanSickle EA, Williams M, Andrechek EA, Zhang C, Yang C, Luo R, and Xiao H. 2014. Tip30 controls differentiation of murine mammary luminal progenitor to estrogen receptor-positive luminal cell through regulating FoxA1 expression. ***Cell Death and Disease*** 5:e1242

29. Zhou X, **Hollern DP**, Liao J, Andrechek E, and Wang H. 2013. NMDA receptor- mediated excitotoxicity depends on the co-activation of synaptic andextrasynaptic receptors. *Cell Death and Disease* 4, e560

30. **Hollern DP**, Yuwanita I, Andrechek ER. 2012. A mouse model with T58A mutations in Myc reduces the dependence on KRas mutations and has similarities to claudin-low human breast cancer. *Oncogene*. 32(10):1296-304

31. Fujiwara K, Yuwanita I, **Hollern DP**, Andrechek ER. 2011. Prediction and Genetic Demonstration of a Role for Activator E2Fs in Myc-Induced Tumors. *CancerResearch* 71(5): 1924-1932.

GRANTS AWARDED

2022	Discovering modes of B cell activation to enhance the treatment of metastasis. Susan G Komen Career Catalyst Award
2021	Using B cells to eliminate TNBC liver metastases. Metavivor Early Career Investigator Award
2021	Crosstalk between EMT & immune cells in breast cancer immunotherapy resistance. Pedal the Cause.
2021	Identifying the Role of B cells and Inflammation in Hepatocellular Carcinoma Initiation. Salk P30
2020	Uncovering pathways that control response and resistance of TNBCs to immunotherapy. Salk P30
2015	Ruth L. Kirschstein National Research Service Award (NRSA) Post-doctoral fellowship F32 National Cancer Institute CA210427
2014	Ruth L. Kirschstein National Research Service Award (NRSA) Post-doctoral fellowship F31 National Cancer Institute CA183272
2014	Aitch Foundation Fellow
2013	College of Natural Science Dissertation Continuation Fellowship
2010	Michigan State University, The Graduate School Summer Fellowship

SELECT PUBLIC PRESENTATIONS OF RESEARCH

1. Investigating B cell - T cell interactions to identify therapeutic opportunities for cancer patients. World Vaccine and Immunotherapy Congress. 2021.
2. B Cells and T Follicular Helper Cells Mediate Response to Checkpoint Inhibitors in High Mutation Burden Mouse Models of Breast Cancer. Virtual oral presentation. Deeley Research Centre Seminar Series. 2020.
3. B Cells and T Follicular Helper Cells Mediate Response to Checkpoint Inhibitors in High Mutation Burden Mouse Models of Breast Cancer. Virtual oral presentation. B cells in Cancer UNC-Kings College of London Virtual Symposium. 2020.
4. Apobec3 induced mutagenesis sensitizes murine models of triple negative breast cancer to immunotherapy by activating B-cells and CD4+ T-cells. **Oral Presentation, General assembly, San Antonio Breast Cancer Symposium**, December 2018
5. Gene Expression features of Seminomas, Mixed Tumors, and Kit Mutant Testicular Germ Cell Tumors, Oral Presentation, TCGA Testicular Germ Cell Data Analysis Workshop, November 2015
6. Identifying Genes That Control Breast Cancer Metastasis, Oral Presentation, Aitch Foundation Hidden Key Fashion Show Fundraiser, June 2014
7. Human breast cancer and mouse models; similarities and constraints identified through gene expression patterns. Oral Presentation Michigan State University, Biomolecular Sciences Annual Retreat, August 2013
8. Human breast cancer and mouse models; similarities and constraints identified through gene expression patterns. Oral Presentation, Mammary Gland Biology, Gordon Research Conferences, June 2013

HONORS and AWARDS

2023	Invited Speaker. La Jolla Immunology Conference
2023	Organizer and Invited Speaker. Keystone Symposia on Cell & Molecular Biology
2022	Organizer. SITC Deep Dive Webinar.
2021	Invited Speaker. U Mass.
2021	Invited Speaker. World Vaccine and Immunotherapy Congress.
2021	Invited Speaker. Salk Cancer Center Summit
2020	Joseph S Pagano Award. University of North Carolina
2020	Invited Speaker. Deeley Research Centre Seminar Series
2020	Invited Speaker. B cells in Cancer UNC-Kings College of London Virtual Symposium
2018	Invited Speaker. San Antonio Breast Cancer Symposium.
2014	The Aitch Foundation Award
2013	Invited Speaker. Mammary Gland Biology, Gordon Research Conferences
2013	Gordon Research Conference Travel Fellowship
2013	The Graduate School Travel Fellowship
2013	Cell and Molecular Biology Travel Fellowship
2007-2009	Dean's List, Grand Valley State University
2003-2004	Dean's List, Grand Rapids Community College

Research Experience

University of North Carolina, Chapel Hill, *Post-doctoral Research Associate,* 2015 to Present
Breast cancer genetics and genomics
Mentor: Dr. Charles M. Perou, Ph.D., May Goldman Shaw Distinguished Professor of Molecular Oncology

- **Identification of predictive gene signatures and mechanisms of response to immune checkpoint inhibitors**

- Lead study identifying predictors and mechanisms of response to immune checkpoint inhibitors using preclinical models of triple negative breast cancer.
- Developed new mouse mammary tumor models to study the immune cell dynamics in triple negative breast cancer.
- Discovered the necessity of B cells, antibody secretion, and T follicular helper cell function to sustained anti-tumor responses to immune checkpoint inhibitors.

- **The Cancer Genome Atlas (TCGA) project – Testicular Germ Cell Tumors analysis working group**
  - Co-lead author on Integrated Molecular Characterization of Testicular Germ Cell Tumors.
  - Analyzed the immune landscape of testicular germ cell tumors.
  - Identified the BCR and TCR repertoire of testicular germ cell tumors.
  - Identified histological specific mRNA expression (mRNAseq) patterns.
  - Identified elevated kit signaling pathway components in testicular germ cell tumors.

- **Credentialing mouse models for the study breast cancer and the immune microenvironment**
  - Led genomic analyses to characterize mouse models of invasive lobular carcinoma.
  - Led genomic analyses to establish mouse models of basal-like breast cancer
  - Established relationships to human breast cancer
  - Led genomic studies to identify key functional pathways and immune cell subsets

Michigan State University, *Graduate Research Assistant,* 2009 - 2015
Cancer genetics and genomics
Advisor: Dr. Eran Andrechek, Ph.D., Associate Professor with Tenure

- **Development of gene expression signatures of mouse mammary tumor histology**
  - Developed and experimentally validated a series of gene expression signatures capable of predicting mouse mammary tumor histology.
  - Identified relationship of mouse model tumor histologies to activation of key oncogenic pathways.
  - Identified relationship of mouse model tumor histologies to human cancer histologies and molecular subtypes on the basis of transcriptomes and oncogenic pathway activity.

- **Dissecting tumor heterogeneity of breast cancer using mouse models**
  - Developed an expansive database of 26 major mouse models of breast cancer.
  - Determined optimal procedures for adjusting and monitoring batch effects.
  - Identified tumor heterogeneity within models on the basis of gene expression profiles and pathway activity.
  - Identified mouse models that parallel human breast cancer.
- **Discovery of novel mechanisms of metastasis using computational analysis of mouse model and breast cancer transcriptomes**
  - Predicted and validated a role for the E2F transcription factors in breast cancer metastasis using a computational analysis of mouse and human breast tumors and genetic tests.
  - Discovered that loss of E2F1 leads to a reduction in circulating tumor cells
  - Identified that loss of E2F1 reduces tumor angiogenesis concomitant with reduced expression of multiple E2F target genes involved genes involved in the hypoxia response.
  - Used CRISPR gene editing to knockout FGF13 and validate a role for FGF13 in metastasis.

Grand Valley State University, *Undergraduate Research Assistant,* 1998-2000
Plant Cell & Developmental Biology
Advisor: Dr. Sheila Blackman, Ph.D., Professor
- Performed immunofluorescence staining to assess cytoskeleton structure of cell cultures of *Daucus carota* in response to integrin-binding peptide, RGD.

SERVICE and LEADERSHIP

B Cells in Cancer Consortium Co-Founder

2021 DOD Study Section Reviewer CON-GC-BC

2021 DOD Study Section Reviewer PB-6

Ad hoc reviewer: Scientific Reports, Nature, Nature Medicine, Cell,

Journal of Immunology , Clinical Cancer Research
The Cancer Genome Atlas (TCGA, Testicular Germ Cell Tumors 2015-2018)
AACR

MANAGEMENT EXPERIENCE and TRAINING

- Principle investigator
- Mentored 1 MD / post doc to independence
- Mentored 1 post doc to industry
- Mentor 2 Grad Students
- Supervise 1 Research Assistant
- Supervise 1 Research Scientist
- 1 part time administrator
- Completed Mentor Training Group for Junior Faculty
- Completed PiBS T32 Mentorship Training