Department of Biology
February 20, 2024

Dear Interdisciplinary Research Origination Award Reviewers,

On behalf of our integrative team, I am pleased to share our IDR proposal titled "Kairn Solutions: Stacking Molecular Insights for Epilepsy Control." Like the *rock cairns* one might see on a trail in Moab that guide your path, we propose a novel way to explore the genetic landscape of epilepsy by staking the outputs of multiple spatial technologies. Here, we propose five core aims to provide our group with the preliminary data necessary to propel us to win external funding.

We have chosen a compelling disease with an unmet clinical need–epilepsy. The U.S. Center for Disease Control and Prevention estimates that 1.2% of the total US population has active epilepsy, with most instances occurring in the very young or very old. The epilepsy foundation also estimates that 1 in 26 persons will develop epilepsy in their lifetime. Of these, 10% will progress to an aggressive form of epilepsy called status epilepticus (SE), characterized by its extended duration. Mortality rates of individuals with SE are high–20%.

Furthermore, epilepsy remains a significant side effect of many drugs that treat cancer, bacterial infections, and even antidepressants. To date, there are few drugs to treat or provide long term remedies for SE, and little is known about how the body naturally (through genetics) responds to seizures (especially in space and time). We have assembled a team of talented researchers from across the university to address this challenging disease using cutting-edge technology leveraged by our labs. Investigator(s) of this project include the following:

Name (PI listed first)	**Department**	**College**
Dr. Matthew Bailey	Biology	College of Life Sciences
Dr. Ryley Parrish	Cell Biology	College of Life Sciences
Dr. Micah Shepherd	Physics and Astronomy	College of Physical and Mathematical Science
Dr. Kevin John	Communications	School of Communications

We propose for Track 1 funding for this BYU-IDR.

I anxiously await your assessment of our proposal.

All my best,

Matthew H. Bailey, Ph.D.
Assistant Professor
Department of Biology

Interdisciplinary Research (IDR) Origination Awards

Cover Page

Project Title

Kairn Solutions: Stacking Molecular Insights for Epilepsy Control.

Principal Investigator(s) (full-time faculty)

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Track

Track one

Abstract

Status Epilepticus (SE) is a prolonged seizure state lasting longer than five minutes and describes a medical emergency with a mortality rate of up to twenty percent. There is an unmet clinical need for such patients because of the limited number of treatments and the need for consensus in the clinical field on how to treat later stages of SE. Recently, our collaboration led to the development of a new approach to improve our understanding of SE. Our model demonstrates how the brain transitions from transient seizures that stop independently to SE in space and time. Using this recently developed technology, i.e., high-density multi-electrode array (HD-MEA), we can track where seizures occur and the electrophysiological nature of how they happen. Despite HD-MEA's strong spatial tracking capabilities, little can be learned from using HD-MEA results alone. Here, we propose to layer HD-MEA with Spatial Transcriptomics, a technology that can capture gene expression at thousands of spots on a small tissue slice. By combining Spatial Transcriptomics with HD-MEA technology, we aim to correlate seizure patterns in the brain with the gene expression changes at those sites. By adding location information to our layered experiment, we will capture a broad landscape of brain activity, thus providing even greater insights into how seizures function. The insights from this study may lead to new gene and drug treatments for SE.

Summary of Plans for External Funding

List target sources of external funding and proposed timeline for proposal submission.

- NIH-NINDS R21 (First attempt October 2024, re-submit June 2025)
- NIH-NINDS R01 (February 2026)
- CURES (January 2025 and January 2026)
- NSF (February 2026 – if all attempts fail because of its basic science aspects)

Introduction and Background

Epilepsy is a neurological disorder defined by recurrent, unprovoked seizures. Despite a long history of research, nearly 30% of people diagnosed with epilepsy do not obtain seizure freedom using today's available drugs[1]. Up to 10% of people worldwide will experience status epilepticus (SE), a prolonged seizure lasting more than five minutes that carries a very high mortality rate of 20%[2].

Due to the unethical nature of inducing seizures in humans, two major models exist to study epileptic activity in mouse brains: *in vivo* (live animals) and *ex vivo* (brain slices). *In vivo* models of epilepsy are difficult to study temporally and spatially because of the invasive nature of measuring live brains. Live brain slices, i.e., *ex vivo* models, on the other hand, provide a well-defined and controlled environment to explore the temporal and spatial progression of epilepsy. Dr. Parrish and colleagues have successfully implemented this brain slice technique, can induce seizures within these slices, and have observed the two distinct types of epilepsy. The first type is phasic seizures[3,4]. Defined by their periodicity, phasic seizures enter and exit states of high electrophysiological activity[5,6]. These events often return to what appears to be normal activity. The second type of seizure is status epilepticus (SE)[7–9]. This second type of seizure, SE, carries both a prolonged electrophysiological state (greater than 5 minutes) and high mortality rates for those who suffer from it[10]. Recently, Codadu et al. demonstrated that brain tissues progressively transition from phasic seizures to SE[7]. Our research team recently discovered that brain tissue sustains SE-like activity in some regions while remaining in phasic seizure-like activity in others. But why? Why would different brain regions stay in a phasic state, yet other regions enter SE? Is it because there are different concentrations of cell types in different regions? Is it because genes are protecting the brain cells? Is it random? Here, we outline several steps to address these questions, answers to which will provide molecular hypotheses that may one day lead to new therapeutics to treat and prevent epilepsy.

Our discovery that phasic and SE seizures are regional, can spread to different parts of the brain, or stay confined to specific brain sections was made possible by a sophisticated technology called high-density multi-electrode array (HD-MEA). These custom microchips are designed to capture the electrical activity of 4096 unique spots tiled across our ex-vivo brain slices. HD-MEA, while impressive, is limited to recording the electrical activity over time. HD-MEA cannot provide information about the gene activity within those spots nor what types of cells may be present or reacting to our seizure-inducing conditions. To resolve this, we plan to layer HD-MEA information with a groundbreaking spatial transcriptomics technology–10X Visium. Briefly, this technology can measure the gene expression of 18,000 genes in each of its 5,000 capture spots tiled across a tissue slice[11].

Below, we outline our research to "stack" the data we collect from HD-MEA and spatial transcriptomics to "visualize" the genes that differentially express during SE, phasic seizures, and regions with no epileptic activity, providing a roadmap of how different patterns of epileptiform activity alter gene expression. Layering both technologies is necessary to further our understanding of SE and provide greater insight into potential drug and gene therapies. We hypothesize that significant transcriptomic differences between progressive SE, phasic seizures, and different brain regions will provide novel insights into the etiology and progression of seizures. In turn, our findings may lead to developing a gene therapy or drug therapy capable of altering epileptiform activity on the transcriptomic landscape.

Research Plan

We have outlined an ambitious and novel strategy to characterize the electrophysiological and genetic components of epilepsy. To achieve our goal, we will integrate two cutting-edge technologies that can characterize live brain tissue in space and time. Below, we outline the steps and brief procedures we will implement to illuminate and improve our understanding of epilepsy.

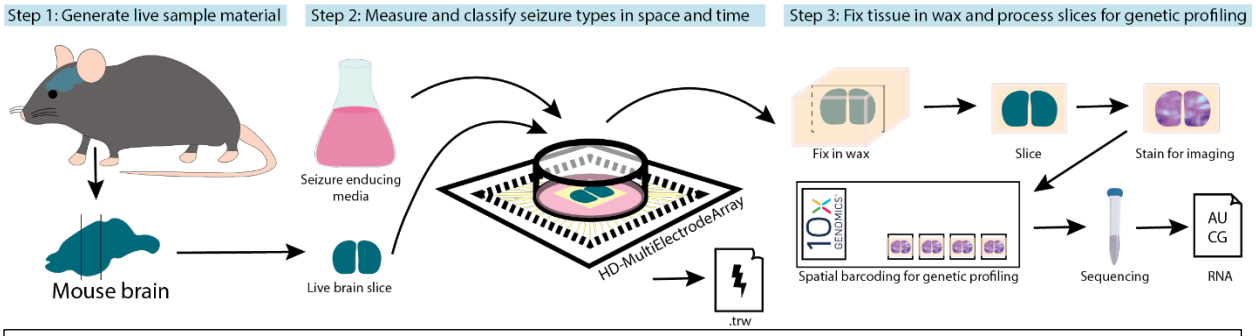


Figure 1: Overview of data generation. (Step 1) *Sample prep step.* Six-week-old mice will be euthanized. The whole brain will be removed and stored in a cold-cutting solution. After incubating, we will slice specific brain regions (350µm) containing the neocortex, entorhinal cortex, hippocampus, and subiculum. This slice will be kept 'alive' in artificial cerebrospinal fluid (ACSF). **(Step 2)** *Measure 4,096 "e-spots" on live brain slices.* Electrophysiological recordings will be performed on a 3Brain BioCAM DupleX system using 3Brain Accura High-Definition Multi-Electrode Array chips. These chips record 4,096 "e-spots" spatially distributed across the chip (See Figure 2). To induce seizures, we will use a zero Mg2+ artificial cerebrospinal fluid (ACSF) solution. E-spots will be recorded for 1-3 hours. **(Step 3)** *Measure 5000 "st-spots" to measure gene expression.* Following HD-MEA measurements, tissue slices will be stored, formalin-fixed, and set in paraffin (wax) in order to further slice the brain to 10-15µm. These smaller slices will be fixed to a glass slide. Using 10X's Cyassist platform, tissue slices will be transferred to special 10X Visium slides capable of capturing genetic material at 5000 "st-spots" (See Figure 4). This platform is capable of capturing ~90% of all possible genes. Genetic material will be sent externally for short-read sequencing.

Aim 1: Data Generation.

As outlined in **Figure 1**, we plan to capture electrical signal and genetic expression information from brain tissue that has experienced a seizure. We have outlined three essential steps to achieve this end. Step 1) Male and female mice (C57BL/6) will be used in this study and cared for under the laws and practices of the Institutional Animal Care and Use Committee (IACUC). Following euthanasia, six-week-old mice brains will be extracted and sliced to a thickness of 350µm and kept 'alive' in artificial cerebrospinal fluid. Meticulous brain slices will capture four critical regions of the mouse brain: the neocortex, entorhinal cortex, hippocampus, and subiculum. Step 2) Once established and thriving in the artificial cerebrospinal fluid, brain slides will be transferred to the high-density multi-electrode array (HD-MEA). The HD-MEA device is designed to capture electrical signals in space and time over its surface (**Figure 2**). Inducing a seizure requires artificial cerebrospinal fluid to be swapped with a similar solution lacking magnesium (Mg2+). This single change in media will dysregulate signal transduction pathways in the brain and induce seizure-like activity (done previously in Dr. Parrish's lab, **Figure 3**). We will record the electrophysiologic signal for 3 hours (max). The resulting experiment will produce a large file containing 10.8 million data points per spot. We refer to these spot-level electrical signals as "e-spots." Multiplied by 4096 spots on the chip, the resulting data file (.trw) will be approximately 44.2 billion bytes of information per sample. Step 3) Following the electrical recording, tissue slices will be formalin-fixed and set in paraffin (wax) to slice the brain smaller (~10-15µm). These smaller slices will be transferred to a standard glass slide for staining and imaging. Using 10X's Cyassist platform, tissue slices will be transferred to proprietary 10X Visium slides capable of capturing genetic material from 5000 spots (**Figure 4**). We call these genetic-based regions spatial transcriptomic spots or "st-spots." This platform can capture ~90% of all possible genes (spatial transcriptomic analysis as performed previously by Dr. Bailey, **Figure 5**). Once isolated and stabilized, genetic material will be sent externally for short-read sequencing because those capabilities are not locally supported at BYU.

Aim 2: Characterize electrophysiological spots (e-spots) to classify seizures.

Following data generation for HD-MEA, each e-spot (**Figure 2**) must be analyzed to determine the subtype of seizure observed (phasic or SE). Dr. Parrish's lab has successfully captured region-specific seizures (**Figure 3A**) using HD-MEA. As shown in **Figure 3B**, the varying seizure patterns are discernable and distinguished through millivolt (mV) patterns. Yet, to capture phasic and SE statistically/computationally will require a series of steps that we briefly outline here. First, HD-MEA will capture 10.8 million raw mV data points per spot for three hours (mV measures captured every millisecond). Second, data reduction

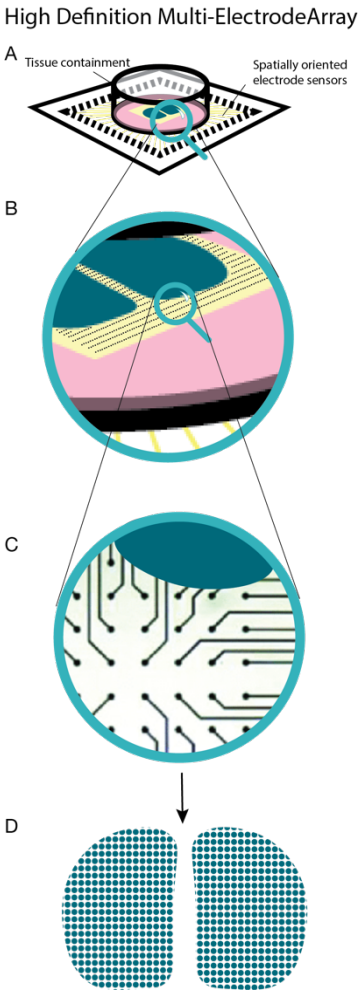


Figure 2: Defining the e-spot. A series of diagrams that progressively zoom in to illustrate the creation and generation of e-spots. **(A)** A cartoon of the HD-MEA apparatus. **(B)** An enlarged diagram of the HD-MEA chip where the tissue sample will be placed. **(C)** The final zoom-in provides the mind's eye with a glimpse at the recording circuits. **(D)** A final illustration of e-spots.

strategies will serially sample time course data to reduce data from ~11 million points to 100,000 points per e-spot. Third, we will label each e-spot using a consensus classification approach, i.e., the agreement of two or three classification algorithms. We have selected three strategies to algorithmically determine seizure status: i) Distance-based K-Nearest Neighbors with dynamic time warping[12], ii) Interval-based TimeSeriesForests[13], and iii) Shapelet Transform Classifier[14]. We will classify each spot as Phasic, Transitioning, or SE. Because of our experiments' high-throughput nature, e-spots from the same experiment can be used to train and test our approach for accuracy. We will rely on Dr. Shepard's experience in time course data to process these data.

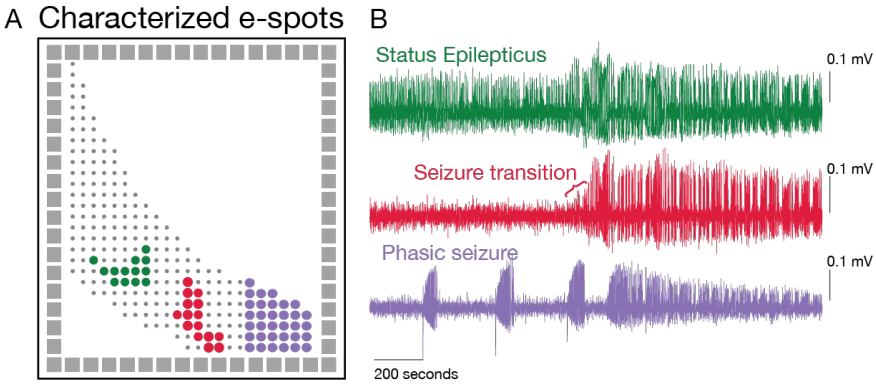


Figure 3: Neocortical tissue can be in disparate activity patterns. (A) Actual brain slice data is color-coded by seizure classification in separate e-spots. Selection of electrophysiology e-spots overlay subregions of the neocortex. **(B)** Representative electrophysiological traces from each group. The X-axis displays time, and the Y-axis displays activity in millivolts (mV). Colors are shared between panels A and B.

Aim 3: Characterize spatial transcriptomics spots (st-spots) to quantify.

Algorithms to process spatial transcript data are well established [15–17]. Briefly, out-of-the-box pipelines will perform the following operations. First, raw genetic information will be mapped to its st-spot (**Figure 4**). Second, each st-spot will be treated as an experiment of 5-10 cells and quantify how much a gene is present in a spot. Third st-spots can then be clustered according to gene expression similarities, labeled with custom tags, or analyzed gene-by-gene and spot-by-spot.

Anticipated results from this step will answer critical questions for this study. First, do st-spots that have experienced seizures cluster by brain region? Recall that our brain slices will capture four major brain regions: the neocortex, entorhinal cortex, hippocampus, and subiculum. From previous literature, we anticipate clear segregation of st-spots in brain components based on the gene expression[18–20], but we are naive regarding how the gene-expression profiles of spots will change due to seizure-like activity or seizure-like activity in proximity to a spot. Previous literature on the topic is limited, and we, to our knowledge, will be the first to explore the spatial gene-expression landscape of seizure-induced brain regions. The following aim will briefly outline the experiments necessary to determine the magnitude of the effect of seizures on gene expression in brain tissue. Dr. Bailey has ample experience analyzing spatial transcriptomic data, segregating st-spots into groups, and performing differential gene expression (**Figure 5**). He has a study (currently in review) that outlines a unique approach to classifying cancer tissue from non-cancer tissue and performs differential analysis using this technology (**Figure 5B & 5C**)[16].

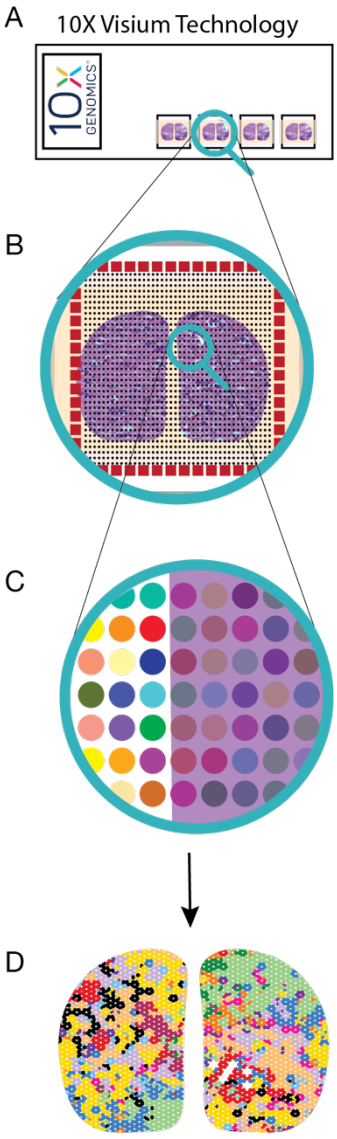


Figure 4: Defining the st-spot. A series of diagrams that progressively zoom in illustrates the generation of st-spots. **(A)** A cartoon of the 10X Visium slides with four capture regions. **(B)** Diagram zoom-in to highlight where the brain tissue slice will be placed. **(C)** The final zoom-in provides the mind's eye with a glimpse at unique genomic barcodes that will be released into the fixed tissue. These positional "barcodes" will then be used to map sequence data back to an (X, Y) coordinate plane. **(D)** Illustrated st-spots and their gene expression in space.

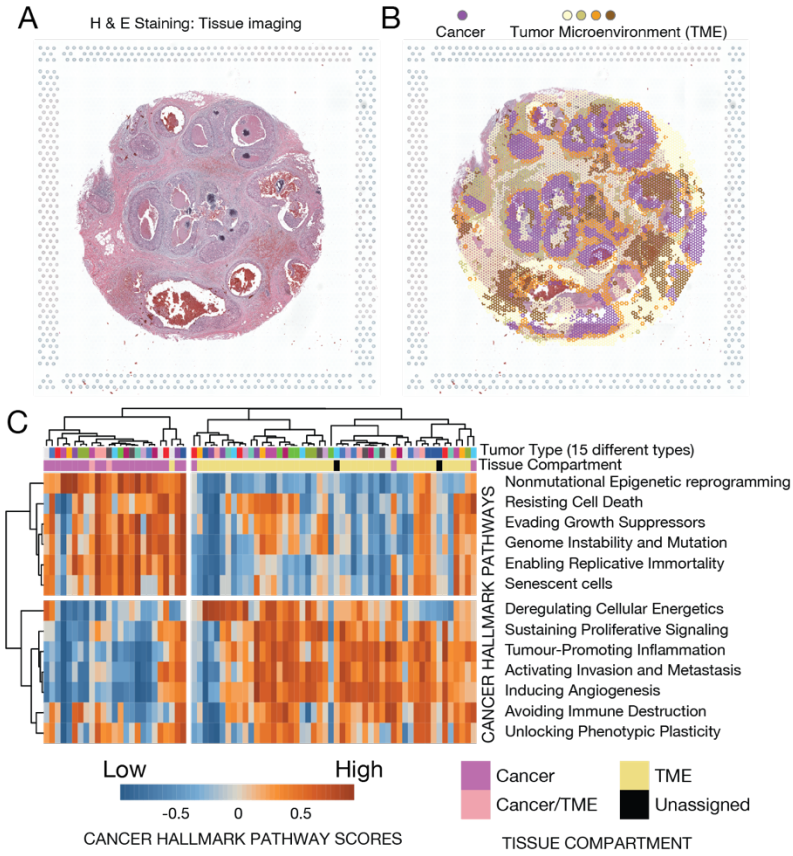


Figure 5: Actual data displays a breakdown of cancer tissue into cancer and non-cancer regions. (A) H&E staining images will be captured to ensure proper orientation of the tissue of interest. In this case, breast tissue. **(B)** Computational deconvolution of st-spots by their gene-expression profiles into tumor and non-tumor compartments. **(C)** A heatmap displays aggregated tumor and non-tumor st-spots from multiple cancer types (columns) and sets of cancer genes in cancer processes (rows). While all of these processes relevant to cancer, differential analysis clearly attributes some processes to the cancer regions and other processes to the tumor microenvironment.

Aim 4: Classify st-spots with e-spots to quantify gene expression. The final step of the research proposal will be to layer e-spots with st-spots from the same tissue. As outlined in **Figure 6**, we will rely heavily on recent advances in 3D spatial tumor modeling to "stack" e-spots with st-spots. Two tools, PASTE and PASTE2, are some of the most advanced algorithms in this space but require multiple slices from the same technology to "match" adjacent tissue slices[21,22]. Fortunately, because we will be using whole brain slices, we can implement a General Procrustes Analysis (GPA) that can leverage spatial landmarks and scale our (X, Y) coordinates to account for shrinkage or expansion throughout our experiment. While our team has little experience with this aim, we are confident we can implement this algorithm using the 'shapes' R-library[23]. Once confident in the overlay, st-spots will be classified by epilepsy type and used for multiple comparisons. First, SE-st-spots will be compared to st-spots that did not undergo seizure activity, i.e., wild time or wt-st-spots. This comparative analysis will prioritize differentially expressed genes that result from a seizure or may protect against seizure activity. Knowing these genes may provide a clear glimpse into the cellular response to seizures or genes

that protect against seizure activity. Because of the number of spots, we will conduct this same analysis in specific brain regions. Similar analyses can be performed in phasic-st-spots, across different brain regions, and eventually in different mouse strains.

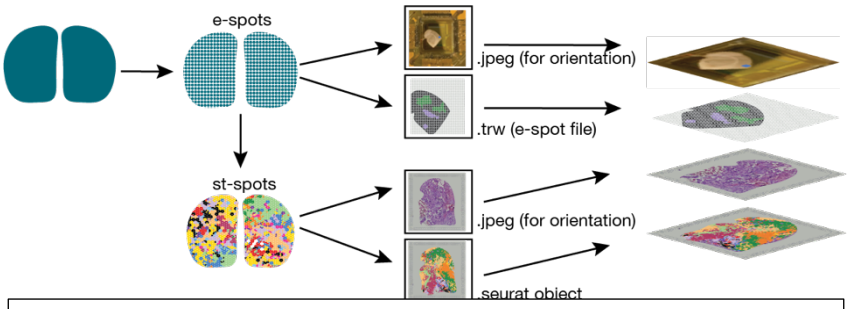


Figure 6: Spatial overlay of e-spots and st-spots. The final step of our analysis will be to integrate data from each of platform to realize our approach's integrative potential. Jpeg images (brain tissue on the HD-MEA and the H&E images) will be critical to layering the e-spots and st-spots.

Aim 5: Video production.
Due to the complex nature of this study, we have reserved $2,500 to generate a student-made video created by the Communications Department at BYU. Due to the complicated intricacies of our study, we will rely on them to generate content for general audiences. Dr. Kevin John will help us find a small team of undergraduate designers to create a short video that captures our findings.

Student Enrichment
In our minds, nothing is more exciting than working on the cutting edge of science and using the latest technology. In doing so, the students of this project will have ample opportunity to present their findings at major genomics conferences such as the American Society of Human Genetics, Biology of Genomes, and the International Epilepsy Congress Meeting (set to be held Aug. 30, 2025, in Portugal).

Limitations and future directions
Limitations are challenging to foresee with innovative and novel projects. Yet, some early attempts to fix brain slices in paraffin have resulted in noticeable tissue shrinkage. This may cause some challenges when stacking our technologies, but we continue to improve our data processing protocols and have already made considerable progress. Additionally, it is computationally feasible to shrink and stretch our spots using the Procrustes analysis. Looking forward, our study design and model system will integrate well with classic genetic models of disease, including gene-knockout and rescue experiments required for preclinical testing. We can also open many collaborative doors with the Department of Chemistry to stack additional technologies on top of the current design, such as spatial-proteomics and phospho-proteomics with Dr. Kelly or neurological drug testing with Dr. Sudweeks.

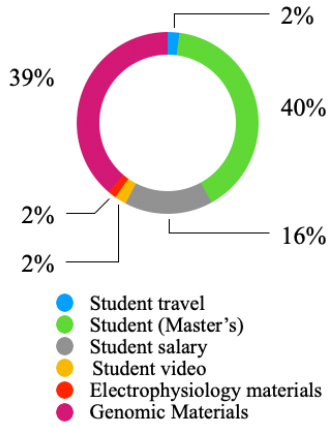
Timeline
We have created an ambitious timeline driven by research questions, grant proposal deadlines, and conference presentations (**Table 1**). This timeline accounts for possible setbacks in data generation.

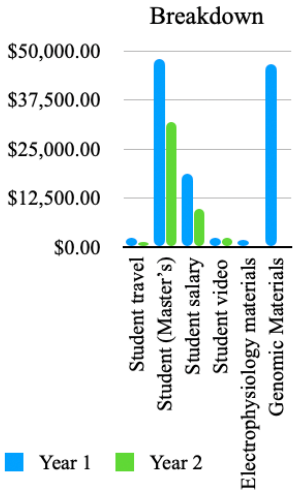
Table 1: Timeline and expected outcomes

Technology	*Aims and Outcomes*	3mo	6mo	9mo	*Timeline*	15mo	18mo	21mo
	Signals of seizures in different brain regions	✈			★			
	Signals of variable seizure inducing media				★			
	Differential expression of different brain regions				★ ✈			
	Genetics of seizure vs non-seizure regions						★	
	Bio-markers of phasic seizures							★
	Bio-markers of status epilepticus							★ ✈
	Aim 1: Data generation							
	Aim 2: e-spot classification							
	Aim 3: st-spot characterization							
	Aim 4: e-spot and st-spot integreation							
	Aim 5: Publicize							
$	Grant attempts	R21 Attempt		CURE Grant		Resubmit R21		Attempt R01

High Definition Multi-Electrode Array · Spatial Transcriptomics · $ Grant attempts · ★ Outcome completion goals · ✈ Present at conferences

Budget Summary and Justification





Category	Year 1	Year 2	Total	Notes
Student travel	$1,250.00	$1,250.00	$2,500.00	2 students per year for a domestic conference
Student (Master's)	$15,950.00	$31,900.00	$47,850.00	1/2 master's first year, 1 full master + salary
Student salary	$9,000.00	$9,850.00	$18,850.00	1 under graduate student for 2 full years
Student video	$0.00	$2,500.00	$2,500.00	Student-led team to publicize findings
Electrophysiology materials	$1,800.00	$0.00	$1,800.00	Hardware to record seizures in space & time
Genomic Materials	$46,500.00	$0.00	$46,500.00	Materials for spatial sequencing for 16 samples
Total	**$74,500.00**	**$45,500.00**	**$120,000.00**	

Overview: The majority of our funds from this proposal will go toward students. We want to provide them with enriching graduate and undergraduate experiences. Below, we outline the funds necessary to support our students and execute our experiments to study epilepsy.

Student Travel: We have set aside $2,500 over two years to support student travel. While we hope to take more than two students annually to academic conferences, these funds will help support travel, lodging, registration, and poster printing fees for conference attendance.

Graduate student: The largest share of these funds will go to the salary support and tuition for a master's student. We plan to cover half of a master's for the first year and fully support one year for a master's student. These numbers are based on costs provided by the Department of Biology for an LDS student.

Undergraduate student salaries: Undergraduate students will play a critical role in this project. These funds will subsidize each investigator to help pay for undergraduate students on this project.

Student-led video: We've set aside $2,500 to produce a short, student-led video to promote and publicize the science performed and the findings we discovered as a result of this IDR funding. We hope to share this piece at the college and university level.

Electrophysiology materials: The estimated cost for hardware to generate electrophysiological data on 16 brain samples will cost ~$1,800, which we will spend in the first year. This includes reagents, HD-MEA chips, and mice.

Genomic Materials: Our largest expense will be generating genomic data for the 16 proposed samples. We anticipate spending ~$2,900 per brain slice to capture, sequence, and analyze these samples. This includes sample prep and reagents at BYU, materials, shipping, and external sequencing costs (BYU does not have a short-read sequencer), and data analysis performed locally on lab laptops.

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Summary of Plans for External Funding

Our approach's novelty, overall potential, and cutting-edge nature will lead to external funding applications. We have a diverse plan for using the data from this application for external funding. Our first objective will be to apply for an NIH R21 (2 years, $250,000) not long following this internal IDR application. These are exploratory grants and do not require extensive preliminary data. This IDR will serve as the backbone of the R21 application. If our first application is unsuccessful, we will continue to use the evolving preliminary data we obtain from this project to reapply for the R21 during the two years of this IDR. Our first application for the R21 will be during the October 2024 NIH deadline. In January 2025, we will also submit a letter of intent to Citizen United for Research in Epilepsy for their CURE award (2 years, $250,000) (**Table 1**).

Furthermore, this exciting work will warrant an R01 application, which provides five years of funding at about $250,000 per year but does require significant preliminary data, which the funding from this IDR will allow us to obtain. We plan to apply for the R01 at the end of the second year following this IDR award, with continual applications if our first attempts are unsuccessful in the following years. These three grant mechanisms outlined above will be our first and primary attempts at external funding. If the above attempts are not successful, we also believe the data from this IDR will make a robust application for an NSF application. Understanding how gene expression changes in diverse brain regions during physiological activity and periods of extreme neuronal stress will be informative to our general understanding of science.

Finally, we will aim for various smaller pots of money, like funds from the American Epilepsy Society and the Brain Research Foundation. While these funding bodies will not be our primary objectives, they provide quality alternatives if our primary objectives are not obtained.

BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. DO NOT EXCEED FIVE PAGES.

NAME: **Bailey, Matthew H.**

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

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE (if applicable)	END DATE MM/YYYY	FIELD OF STUDY
Brigham Young University, Provo, UT	BS	04/2013	Bioinformatics
Washington University in St. Louis, St. Louis, MO	PHD	10/2018	Human and Statistical Genetics
Huntsman Cancer Institute, UHealth, Salt Lake City, U	Postdoctoral Fellow	07/2021	Pre-clinical genomic modeling

A. Personal Statement

My role in this grant proposal is principal investigator. I have a strong track record in providing meaningful contributions to two aging phenotypes–Alzheimer's disease and adult cancer. I regularly analyze genomic datasets (whole genome, whole exome, GWAS, and pedigrees) with 10,000+ samples, which provided me with more than ten years of experience assisting and leading genomic collaborations (see section C. below). I am an early investigator at Brigham Young University (an undergraduate teaching institution in Provo, UT).

1. Li Y, Porta-Pardo E, Tokheim C, **Bailey MH**, Yaron TM, Stathias V, Geffen Y, Imbach KJ, Cao S, Anand S, Akiyama Y, Liu W, Wyczalkowski MA, Song Y, Storrs EP, Wendl MC, Zhang W, Sibai M, Ruiz-Serra V, Liang WW, Terekhanova NV, Rodrigues FM, Clauser KR, Heiman DI, Zhang Q, Aguet F, Calinawan AP, Dhanasekaran SM, Birger C, Satpathy S, Zhou DC, Wang LB, Baral J, Johnson JL, Huntsman EM, Pugliese P, Colaprico A, Iavarone A, Chheda MG, Ricketts CJ, Fenyö D, Payne SH, Rodriguez H, Robles AI, Gillette MA, Kumar-Sinha C, Lazar AJ, Cantley LC, Getz G, Ding L. Pan-cancer proteogenomics connects oncogenic drivers to functional states. Cell. 2023 Aug 31;186(18):3921-3944.e25. PubMed PMID: 37582357.

2. **Bailey MH**, Tokheim C, Porta-Pardo E, Sengupta S, Bertrand D, Weerasinghe A, Colaprico A, Wendl MC, Kim J, Reardon B, Kwok-Shing Ng P, Jeong KJ, Cao S, Wang Z, Gao J, Gao Q, Wang F, Liu EM, Mularoni L, Rubio-Perez C, Nagarajan N, Cortés-Ciriano I, Zhou DC, Liang WW, Hess JM, Yellapantula VD, Tamborero D, Gonzalez-Perez A, Suphavilai C, Ko JY, Khurana E, Park PJ, Van Allen EM, Liang H, Lawrence MS, Godzik A, Lopez-Bigas N, Stuart J, Wheeler D, Getz G, Chen K, Lazar AJ, Mills GB, Karchin R, Ding L. Comprehensive Characterization of Cancer Driver Genes and Mutations. Cell. 2018 Aug 9;174(4):1034-1035. PubMed Central PMCID: PMC8045146.

3. **Bailey MH**, Meyerson WU, Dursi LJ, Wang LB, Dong G, Liang WW, Weerasinghe A, Li S, Li Y, Kelso S, Saksena G, Ellrott K, Wendl MC, Wheeler DA, Getz G, Simpson JT, Gerstein MB, Ding L. Retrospective evaluation of whole exome and genome mutation calls in 746 cancer samples. Nat Commun. 2020 Sep 21;11(1):4748. PubMed Central PMCID: PMC7505971.

B. Positions, Scientific Appointments and Honors

Positions and Scientific Appointments

2021 -	Assistant Professor, Brigham Young University, Provo, UT
2018 - 2021	Postdoctoral Research Fellow, University of Utah, Huntsman Cancer Institute, Salt Lake City, UT
2013 - 2018	Graduate student, Department of Medicine and Oncology, Washington University in St. Louis, St. Louis, MO
2012 - 2012	Summer Research Assistant, University of Utah, Salt Lake City, UT
2011 - 2011	Summer Research Fellow, Simmons Center for Cancer Research, Brigham Young University, Provo, UT
2010 - 2013	Research Assistant, Brigham Young University, Provo, UT

Honors

2019 - 2021 T32 - Postdoctoral Training Fellew, University of Utah

C. Contribution to Science

1. Bookend to the TCGA cancer projects for the cancer driver gene group. Co-lead the driver gene discovery project for the PanCanAtlas consortia. Using 26 computational tools to catalog driver genes and mutations, we identified 299 cancer genes and >3,400 putative driver mutations from >9,000 samples using the TCGA dataset. 60-85% of our predictions were confirmed in 2 cell-lines. I also confirmed associations of microsatellite instability with higher expression of immune checkpoint blockers PD-1 and PD-L1. We also determined >50% of TCGA harbor a druggable mutation. To date, this study has been cited ~1600 times. It serves as a benchmark to the Pan-Cancer community and allows researchers to identify recurrent driver mutations in their respective studies regardless of sample size. This study also elevates the field by moving beyond drive gene analysis and focusing on the causal mutations within these genes.

a. **Bailey MH**, Tokheim C, Porta-Pardo E, Sengupta S, Bertrand D, Weerasinghe A, Colaprico A, Wendl MC, Kim J, Reardon B, Ng PK, Jeong KJ, Cao S, Wang Z, Gao J, Gao Q, Wang F, Liu EM, Mularoni L, Rubio-Perez C, Nagarajan N, Cortés-Ciriano I, Zhou DC, Liang WW, Hess JM, Yellapantula VD, Tamborero D, Gonzalez-Perez A, Suphavilai C, Ko JY, Khurana E, Park PJ, Van Allen EM, Liang H, Lawrence MS, Godzik A, Lopez-Bigas N, Stuart J, Wheeler D, Getz G, Chen K, Lazar AJ, Mills GB, Karchin R, Ding L. Comprehensive Characterization of Cancer Driver Genes and Mutations. Cell. 2018 Apr 5;173(2):371-385.e18. PubMed Central PMCID: PMC6029450.

2. Uniformly reprocessed the exomic mutations for The Cancer Genome Atlas (TCGA). I assisted in analyzing, decision-making, and writing the multicenter mutation calling in multiple cancer type (MC3) project. This project generated >400TB of raw sequencing data from over 10,000 samples across 33 cancer types. We overcame challenges inherent in the TCGA organization, where 33 cancer types were analyzed 33 different ways. Uniformly, we utilized seven mutation callers to identify single nucleotides and small indels on the TCGA data compendium. This study is the crux of the exomic mutation calling effort for TCGA and was publicly released to the entire cancer community.

a. Ellrott K, **Bailey MH**, Saksena G, Covington KR, Kandoth C, Stewart C, Hess J, Ma S, Chiotti KE, McLellan M, Sofia HJ, Hutter C, Getz G, Wheeler D, Ding L. Scalable Open Science Approach for Mutation Calling of Tumor Exomes Using Multiple Genomic Pipelines. Cell Syst. 2018 Mar 28;6(3):271-281.e7. PubMed Central PMCID: PMC6075717.

3. I was thrilled to be invited to assist as an analyst for the suite of CPTAC manuscripts published in August of this year. I have worked closely with this team in the past, and we have a collective mission to understand the functional consequences of cancer driver mutations, i.e., the mutations that occur most frequently across multiple cancer types. The proposed work extends portions of this work to identify the impact of pathogenic germline mutations on cancer and other phenotypes–especially concerning different U.S. demographics collected by All of Us.

a. Li Y, Porta-Pardo E, Tokheim C, **Bailey MH**, Yaron TM, Stathias V, Geffen Y, Imbach KJ, Cao S, Anand S, Akiyama Y, Liu W, Wyczalkowski MA, Song Y, Storrs EP, Wendl MC, Zhang W, Sibai M, Ruiz-Serra V, Liang WW, Terekhanova NV, Rodrigues FM, Clauser KR, Heiman DI, Zhang Q, Aguet F, Calinawan AP, Dhanasekaran SM, Birger C, Satpathy S, Zhou DC, Wang LB, Baral J, Johnson JL, Huntsman EM, Pugliese P, Colaprico A, Iavarone A, Chheda MG, Ricketts CJ, Fenyö D, Payne SH, Rodriguez H, Robles AI, Gillette MA, Kumar-Sinha C, Lazar AJ, Cantley LC, Getz G, Ding L. Pan-cancer proteogenomics connects oncogenic drivers to functional states. Cell. 2023 Aug 31;186(18):3921-3944.e25. PubMed PMID: 37582357.

4. This project is currently under review at Cancer Discovery. For this project, we created strong computational algorithms to address the Hallmarks of Cancer in one of the largest aggregated sets of Spatial Transcriptomic data to date. Our findings confirmed a reproducible split of Cancer Hallmarks between tumors and their microenvironments. We also show that the majority of the tumors are clonal in nature (as defined by predicted copy number) and that subclones (as defined by varying copy number) rarely shift away from their primary hallmarks. Finally, we highlight the neighbor transcriptomic spots cooccur and can be used to predict its neighbors. We published some of our preliminary findings on BioRxiv (cited below).

a. Sibai M, Cervilla S, Grases D, Musulen E, Lazcano R, Mo CK, Davalos V, Fortian A, Bernat A, Romeo M, Tokheim C, Grande E, Real F, Barretina J, Lazar AJ, Ding L, Esteller M, **Bailey MH**, Porta-Pardo E. The spatial landscape of Cancer Hallmarks reveals patterns of tumor ecology. bioRxiv, pp.2022-06.

BIOGRAPHICAL SKETCH – RESEARCH GRANT APPLICANT

Provide the following information for the Applicant and other key personnel. (Only required for the applicant for LOI submission.)
DO NOT EXCEED FIVE PAGES.

NAME: Robert Ryley Parrish

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Auburn University Montgomery (Alabama, USA)	B.S.	05/2007	Physical Science
University of Alabama Birmingham (Alabama, USA)	PhD.	04/2015	Neurobiology
Newcastle University (UK)	Post-doc	12/2019	Neurophysiology

A. Personal Statement.

I have been engaged in epilepsy research since starting my PhD in 2009. After completion of my PhD, I moved into the field of *in vivo* and *ex vivo* electrophysiology, using these techniques to continue study the pathophysiology of epilepsy. My experience includes patch-clamp electrophysiology, local field potential electrophysiology, wireless EEGs, and work with multi-electrode arrays (MEAs), including NeuroNexus probes, the Utah array, and the 3Brain CMOS arrays. The 3Brain arrays have 4,096 electrodes and are used with the 3Brain BioCam instrument. I have used the 3Brain BioCam DupleX for 4 years. My former objective involved the use of this equipment for drug discovery research at Xenon Pharmaceutical, focusing on compounds for the suppression of epileptiform activity. I obtained extensive experience using these large-scale MEAs while at Xenon and currently have two publications using this instrument[1,2] and another publication pending. The Mahadevan et. al., 2022 is of particular note, as it describes a graphical user interface we designed and are providing open source for visualization and analysis of local field recordings obtained from large-scale MEA recordings. In this graphical user interface, we outline a novel seizure tracking algorithm, which we used to analyze multiple seizure parameters from brain slices collected from a mouse model of Dravet syndrome. I have also worked directly in the past on the study of status epilepticus, where I contributed to a landmark paper outlining how alterations in E_{GABA} contribute to refractory status epilepticus[3]. Working with the 4,096 channel MEAs, creating a graphical user interface to analyze these MEA recordings, and working with others to study refractory status epilepticus have made me particularly well-suited to aid in the project outlined in this application. We are also very interested in how seizure activity impacts RNA transcriptional profiles[4].

1. Thouta S, Waldbrook MG, Lin S, Mahadevan A, Mezeyova J, Soriano M, Goodchild SJ, **Parrish RR**. Pharmacological determination of the fractional block of Nav channels required to impair neuronal excitability and ex vivo seizures. *Frontiers in Cellular Neuroscience*, 2022.
2. Mahadevan A, Codadu NK, **Parrish RR**. Xenon LFP Analysis Platform Is a Novel Graphical User Interface for Analysis of Local Field Potential From Large-Scale MEA Recordings. *Frontiers in Neuroscience*, 2022.
3. Burman RJ, Selfe JS, Lee JH, van den Burg M, Calin A, Codadu NK, Wright R, Newey S, **Parrish RR**, Katz AA, Wilmshurst JM, Akerman CJ, Trevelyan AJ, Raimondo JV. Activity-dependent chloride accumulation and excitatory GABAergic signaling contribute to benzodiazepine resistance in a model of status epilepticus. *Brain*, 2019.
4. Vaughan AJ, McMeekin LJ, Hine K, Stubbs IW, Codadu NK, Cockell S, Hill JT, Cowell R, Trevelyan AJ, **Parrish RR**. RNA sequencing demonstrates *ex vivo* neocortical transcriptomic changes induced by epileptiform activity in male and female mice. BioRxiv preprint: https://doi.org/10.1101/2023.11.20.567411, In revisions at eNeuro

B. Positions and Honors

2009-2014 Graduate Assistant, Advisor: Dr Farah Lubin, Neurobiology, UAB, Birmingham, AL
2014-2019 Postdoctoral Researcher, Advisor: Prof Andrew Trevelyan, Institute of Neuroscience, University of Newcastle, Newcastle upon Tyne, UK
2019-2021 Research Scientist II, In vitro Biology, Xenon Pharmaceuticals, Burnaby, British Columbia, Canada
2021-2022 Senior Research Scientist, In vitro Biology, Xenon Pharmaceuticals, Burnaby, British Columbia, Canada
2022-Present Assistant Professor, Department of Cell Biology and Physiology, Brigham Young University, Provo, Utah

C. Contribution to Science

Role of cortical inhibition in propagating epileptiform activity
Understanding how seizures propagate is critically important and a key step to development of therapy to prevent seizure generalization. Much of my work since the beginning of my postdoctoral training focused on understanding the endogenous mechanisms that prevent seizures and how these mechanisms fail, resulting in spreading seizures. I successfully characterized the cell classes involved in inhibitory restraint during spreading seizures, using imaging tools and patch-clamp electrophysiology[1,2]. I further worked to understand some of the key mechanisms related to failure of the inhibitory restraint during spreading seizures, such as pyramidal cell chloride loading and subsequent rises in extracellular potassium secondary to the elevated chloride levels in pyramidal cells[3,4]. This work has been critical to our understanding of how inhibition fails during spreading seizures and have opened up ideas for novel treatment options.

1. **Parrish RR**, Codadu NK, MacKenzie-Gray-Scott C, Trevelyan AJ. Feedforward inhibition ahead of ictal wavefronts is provided by both parvalbumin and somatostatin expressing interneurons. The Journal of Physiology, 2019.
2. Codadu NK, Graham R, Jackson-Tylor T, Burman RJ, Raimondo JV, Trevelyan AJ, **Parrish RR**. Divergent paths to seizure-like events. Physiological Reports, 2019.
3. **Parrish RR**, Jackson-Taylor T, Grundmann A, Codadu NK, Calin A, Alfonsa H, Wykes RC, Voipio J, Trevelyan AJ. Indirect effects of Halorhodopsin activation: potassium redistribution, non-specific inhibition and spreading depression. The Journal of Neuroscience, 2023.
4. Graham RT, **Parrish RR**, Alberio L, Johnson EL, Trevelyan AJ. Optogenetic stimulation reveals a latent tipping point in cortical networks during ictogenesis. Brain, 2022.

Pyramidal cell chloride loading results in cortical spreading depolarizations
While studying how pyramidal cell chloride loading contributes to the failure of inhibitory restraint, I made a very exciting discovery related to the induction of cortical spreading depolarizations (CSDs). CSDs were first described in the 1940s and much work has been done to try and understand the mechanism of induction of these events. The current leading hypothesis involves a critical threshold of extracellular potassium that must be reached, some reporting this to be about 12mM, to induce a CSD. Using long stimulations of halorhodopsin, I have discovered that CSDs can be induced during periods of cellular inhibition, when extracellular potassium is at or below physiological levels[1]. This data might, quite literally, completely transform the way people think about CSD induction, suggesting that potassium may not be a necessary critical trigger for CSD induction. This is, therefore, a very exciting finding and is likely a key advancement in our understanding of the mechanisms involved in CSD induction.

1. **Parrish RR**, Jackson-Taylor T, Grundmann A, Codadu NK, Calin A, Alfonsa H, Wykes RC, Voipio J, Trevelyan AJ. Indirect effects of Halorhodopsin activation: potassium redistribution, non-specific inhibition and spreading depression. The Journal of Neuroscience, 2023.
2. **Parrish RR**, Jackson-Taylor T, Voipio J, Trevelyan AJ. Optogenetic ion pumps differ with respect to the secondary pattern of K+ redistribution. Physiological Reports, 2023

Link to Robert Ryley Parrish's full list of published works: R Ryley Parrish (0000-0001-6798-6140) (orcid.org)

BIOGRAPHICAL SKETCH – RESEARCH GRANT APPLICANT

NAME: Micah Shepherd

POSITION TITLE: Associate Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Brigham Young University	B.S.	08/2005	Applied Physics
Brigham Young University	M.S.	12/2007	Physics
The Pennsylvania State University	Ph.D.	5/2014	Acoustics

A. Personal Statement

I have extensive experience using signal processing to characterize the temporal and spectral characteristics of wave signals in fluids and solids. While my work has been primarily related to acoustics and vibration, I have also been applying signal processing techniques to identify and characterize neural activity in the brain.

B. Positions and Honors

Academic Positions

Research Assistant, The Pennsylvania State University — *Aug 2007 – Dec 2010*
Senior Research Assistant, The Pennsylvania State University — *Jan 2011 – April 2014*
Assistant Research Professor, The Pennsylvania State University — *May 2014 – June 2021*
Associate Research Professor, The Pennsylvania State University — *July 2021 – July 2022*
Associate Professor, Brigham Young University — *Aug 2022 – present*

C. Contribution to Science

Advancements in understanding vibration and acoustic radiation

Numerical and experimental studies were performed to better understand the relationship of complex vibrating structures and the noise they generate. Advanced signal processing methods were used for the measurements. Roles: Graduate student advisor, principal investigator, graduate student

- **M.R. Shepherd**, E.M. Stimson and A.D. Hanford, "Local and global modes of single and multi-grid acoustic black hole panels," JASA Express Lett. **3** (2023)
- T.W. Jerome, **M.R. Shepherd**, and S.A. Hambric "Ultrasonic Investigation of the Pressure Profile on the Faying Surface of Fastened Aluminum Plates," J. Mech. Syst. Sig. Proc. **150** (2021).

- G.L. Rhoades, **M.R. Shepherd**, and J.R. Harris, “Measuring plate vibration using deflectometry: The advantages and limitations of add-on reflective material,” Proc. Mtgs. Acoust. **42** (2021).
- **M.R. Shepherd**, M.T. Rose, S.D. Sommerfeldt, “On the physical meaning of wavenumber truncation as applied to source identification on vibrating structures,” Proc. Mtgs. Acoust. **35** (2019).

Applications of structural-acoustic optimization

Mathematical optimization was used to study and improve the structural acoustic behavior of systems including shaped concrete flooring, the acoustic black hole effect and panels excited by turbulent flow. This led to improved understanding of how to design these systems and the trade-off relationships which exist with other parameters. Roles: Graduate student advisor, principal investigator, graduate student

- J.M. Broyles, **M.R. Shepherd**, and N.C. Brown, “Modified acoustic transmission metrics for early-stage design exploration using a computational case study of heavyweight floors,” Ap. Acoust. **196** (2022).
- J.M. Broyles, **M.R. Shepherd**, and N.C. Brown, “Design optimization of structural-acoustic spanning concrete elements in buildings,” J. Archit. Eng. **28**(1) (2022).
- C.A. McCormick and **M.R. Shepherd**, “Design optimization and performance comparison of three styles one-dimensional acoustic black hole vibration absorbers,” J. Sound Vib. **470** (2020)
- **M.R. Shepherd**, R.L. Campbell and S.A. Hambric, “A parallel computing framework for performing structural-acoustic optimization,” Struct. MultiDisp. Opt. (2019)

Physics of the sound production of musical instruments

The physics underlying the production of sound by two musical instruments (acoustic guitar and cello) was studied. Specially, the role of vibration was examined and compared with mathematical models. Role: Graduate student advisor, principal investigator

- S.D. Bellows, D.T. Harwood, K.L. Gee and **M.R. Shepherd**, “Directional characteristics of two gamelan gongs,” J. Acoust. Soc. Am. 154, 1921-1931 (2023).
- E. Rokni, M. Smallcomb, T.E. Blanford and **M.R. Shepherd**, “Noninvasive methods for quantifying sounds post placement in a cello,” Proc. Mtgs. Acoust. **42** (2021).
- T.E. Blanford, **M.R. Shepherd**, T.W. Jerome, “A comparison of fractional-sized to full-sized cellos,” Proc. Mtgs. Acoust. **33** (2018).
- **M.R. Shepherd**, S.A. Hambric, D.B. Wess “The effects of wood variability on the free vibration of an acoustic guitar top plate,” J. Acoust. Soc. Am. **136**(5), EL357-EL361 (2014).

Dr. Shepherd’s full publication list is available here:
https://physics.byu.edu/department/publications/shepherd.

BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: John, Kevin K.

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

POSITION TITLE: Assistant Professor, Brigham Young University

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Brigham Young University, Provo, Utah	B.A.	08/07	Public Relations
Brigham Young University, Provo, Utah	M.A.	12/09	Communication
University of Utah, Salt Lake City, Utah	Ph.D.	05/15	Communication

A. Personal Statement

I specialize in health communication, with past research covering topics from skin cancer and body image, to alcohol, tobacco, hospice, and narrative transportability. For the past 12 years I have served as the Director of the BioComms Lab in the Brigham Young University School of Communication, and I am proficient in visual attention, cognition, and the impact of message features and strategies on visual processing and affect. I offer 5 years of experience working in professional strategic communications positions, and nearly 20 years of experience with an assortment of eye-tracking hardware and software platforms. Many of my publications focus on how individuals visually process messages, including advertisements, health campaigns, and other persuasive communications—and several of these focused specifically on scientific and medical communications. This experience has direct bearing on my role in the current project, where my primary function will be the development of messaging targeted to lay audiences.

1. **John, K. K.**, Freeman, J. R., Zurcher, J. D., & Thomsen, S. R. (2022). Eye-tracking and adolescent interest in alcohol responsibility messages: The message matters. *Journal of Studies on Alcohol and Drugs*, *83*(3), 412-419.
2. Pokharel, M., Jensen, J. D., **John, K. K.**, & Christy. K. R. (2019). Visual communication & skin cancer prevention: A message experiment comparing personalized, stock, & non-ultraviolet images. *Annals of Behavioral Medicine*, *53*, S602-S602.
3. **John, K. K.**, Jensen, J. D., King, A. J., Pokharel, M., & Grossman, D. (2018). Emerging applications of eye-tracking technology in dermatology. *Journal of dermatological science*, *91*(2), 117-123.
4. **John, K. K.**, Jensen, J. D., King, A. J., Ratcliff, C. L., & Grossman, D. (2017). Do Pattern-Focused Visuals Improve Skin Self-Examination Performance? Explicating the Visual Skill Acquisition Model (VSAM). *Journal of health communication*, *22*(9), 732-742.

B. Positions and Honors

Positions

2021-present **Fellow**, Simmons Center for Cancer Research, Brigham Young University
2021-present **Associate Professor**, School of Communication, Brigham Young University
2015-2021 **Assistant Professor**, School of Communication, Brigham Young University
2012-present **Director of the BioComms Lab**, School of Communication, Brigham Young University
2013-2015 **Project Coordinator:** Partners in Hospice Care project (NIH-funded, P01CA138317), University of Utah
2012-2013 **Coder:** Partners in Hospice Care project (NIH-funded, P01CA138317), University of Utah
2012-2015 **Teaching Assistant**, University of Utah
2011-2015 **Adjunct Professor**, Brigham Young University
2006-2009 **Research Assistant**, Brigham Young University

Honors

2023-2026 **David P. Forsyth Memorial Research Award:** BYU School of Communications
2021 **Top Paper:** Health Communication Division, AEJMC Conference
2021 **Top Paper:** Entertainment Studies Interest Group, AEJMC Conference
2020 **Top Paper:** Visual Communication Division, AEJMC Conference
2020 **Faculty Teaching Award:** BYU Division of Continuing Education
2020 **Ruth S. Silver Fellowship:** BYU School of Communications
2019 **Faith + Works Scholar:** BYU College of Fine Arts & Communications
2018 **Raymond E. and Ida Lee Beckham Lecturer:** BYU School of Communications
2018 **Top Paper:** Visual Communication Division, National Communication Association
2018 **Excellent in Teaching Award:** BYU College of Fine Arts & Communications
2014 **Top Student Paper**: 38th Annual Meeting, American Society of Preventive Oncology

C. Research Support

Ongoing Research Support

(John, K., co-PI)
Utah Grand Challenges Initiative 02/1/2022 – Present
Using Rapid Exposure to Mole Imagery to Promote the Early Detection of Melanoma. Co-Principal Investigator with Tawnya Bowles (IHC) and Jakob D. Jensen (Huntsman Cancer Institute). ($100,000)

(John, K., PI)
Wendell J. Ashton Award 02/1/2022 – Present
Project #1: Recall and Emotional Reactions to Holographic vs. Traditional Video, Project #2: Using Biometrics to Assess the Impact of Social Media Exposure on Female Cosmetic Surgery Normative Beliefs, Project #3: The Impact of Positive and Negative Chat Feeds on Perceptions of Player Performance During Twitch Streams, Project #4: Examining the Effects of a Plant-Based Diet on Individuals with Eating Disorders, Project #5: The Efficacy of Virtual Reality in Active Shooter Training Scenarios, Project #6: Comparing Markers of Authenticity in Online Communicators Between Older and Younger Audiences. ($12,000)

(John, K., PI)
Ruth S. Silver Award 12/01/2019 – 12/31/2020
This funding supports data collection for a project entitled “Changes in Discrete Emotional State Following Social Media Exposure,” which seeks to measure the impact of social media use on the emotions of users directly following use. ($3,500)

Completed Research Support

(John, K., PI)
BioComms Lab Development and Support 08/01/2015 – 12/15/2018
This funding supported the upgrading of the BioComms Lab equipment to facilitate several projects related to my SSE research program including “Emerging applications of eye-tracking technology in dermatology” and “Do Pattern-Focused Visuals Improve Skin Self-Examination Performance? Explicating the Visual Skill Acquisition Model (VSAM),” among others. ($245,000)

(John, K., PI) 08/01/2016 – 12/31/2017
Ashton Fund Grant
“Male body image: Using eye-tracking to examine moderators of regional visual attention”
This grant was awarded to support the expansion of a female body image study to cover a male sample, and to examine moderating factors for visual attention on specific regions of male model bodied in both video-based and still-image advertisements. ($7,000)

(Thomsen, S., PI) 08/01/2007 – 12/31/2009
Ashton Fund Grant
“Using eye-tracking to investigate body image among females, and alcohol warning messages for adolescents”
This grant provided funds to expand use of the eye-tracking lab to support faculty research interests. ($5,000)

Current and pending funding

PI: Matthew Bailey

M.H. Bailey (PI) August 2021 – July 2024. BYU faculty start-up funds. This includes funds for equipment, consumables, and research technician pay. This money is not tied to a particular project.

M.H. Bailey (PI) "Establishing the Polynesian Utah Kancer Alliance (PUKA) for Comprehensive Molecular Characterization in Many Cancers." Simmons Center for Cancer Research IDR. (February 2024 – February 2026; $40,000)

M.H. Bailey (Sub-award) "Phosphoproteomics in tumor organoids." Awarded privately by Coulter Pharmaceutical Inc. through the University of Virginia. (January 2024 – November 2024; $50,000)

*M.H. Bailey (PI) "Genetic predisposition and misdiagnosis of cancer in All of Us participants." This grant is **pending** from NIH-R15. ($150,000 June 2024- June 2027)*

PI: Ryley Parrish

R.R. Parrish (PI) August 2022 – July 2025. BYU faculty start-up funds. This includes funds for equipment, consumables, and research technician pay. This money is not tied to a particular project.

R.R. Parrish (PI), M.R. Shepherd (co-I), "Novel model to study refractory status epilepticus," funded by American Epilepsy Society. (Sept 2023 – Aug 2024; $50,000)

PI: Micah Shepherd

M.R. Shepherd (PI), "Optimization of Anechoic Musical Instrument Recordings," funded by the Institute of Scientific Research in Music (Dec 2022 – Nov 2024; $75,000)

M.R. Shepherd (PI), "Advanced damping system for broadband vibration and interior noise control of composite airframes of transport rotorcraft," funded by the Office of Naval Research as a subaward under Penn State University. (Jan 2023 – Dec 2024; $18,961)

M.R. Shepherd (PI), K.L. Gee (co-I), "Rocket noise source characterization using large bandwidth intensity," funded by Northrup Grumman Corporation. (March 2023 – June 2024; $107,000)

R.R. Parrish (PI), M.R. Shepherd (co-I), "Novel model to study refractory status epilepticus," funded by American Epilepsy Society. (Sept 2023 – Aug 2024; $50,000)

PI: Kevin John

K. John (co-PI) "Using Rapid Exposure to Mole Imagery to Promote the Early Detection of Melanoma." Awarded by the Utah Grand Challenges Initiative (Feb 2022 - Present; $100,000)

K. John (PI) "Project #1: Recall and Emotional Reactions to Holographic vs. Traditional Video, Project #2: Using Biometrics to Assess the Impact of Social Media Exposure on Female Cosmetic Surgery Normative Beliefs, Project #3: The Impact of Positive and Negative Chat Feeds on Perceptions of Player Performance During Twitch Streams, Project #4: Examining the Effects of a Plant-Based Diet on Individuals with Eating Disorders, Project #5: The Efficacy of Virtual Reality in Active Shooter Training Scenarios, Project #6: Comparing Markers of Authenticity in Online Communicators Between Older and Younger Audiences." Wendell J. Ashton Award (02/1/2022 – Present; $12,000)

K. John (PI) "Changes in Discrete Emotional State Following Social Media Exposure," which seeks to measure the impact of social media use on the emotions of users directly following use. Ruth S. Silver Award. (12/01/2019 – 12/31/2020; $3,500)