SPARK

THE TULLIE AND RICKEY FAMILIES
SPARK AWARDS FOR
INNOVATIONS IN IMMUNOLOGY

Annual Report 2023

Many thanks to all donors for supporting our research.

Felix Nebgen

¡Muchas gracias! I am truly grateful for your belief in my research. Rosa Isela Galvan

Thanks to all the SPARK donors for joining hands to achieve our goal of 'Life Without Disease' & making this world a better place for next generations...

– GURUPREET SETHI

Thank you SPARK Donors for giving young researchers like me a chance to make an idea a reality!

– Erik Ehinger

SPARK | THE TULLIE AND RICKEY FAMILIES SPARK AWARDS FOR INNOVATIONS IN IMMUNOLOGY

Innovation doesn't come cheap. But without proof-of-concept to convince highly competitive granting agencies to fund a daring project, bold ideas are never put into action. This is
a particular challenge for younger scientists still establishing their careers. La Jolla Institute's Tullie and Rickey Families SPARK Awards for Innovations in Immunology is designed to overcome these hurdles.

This philanthropically-financed program provides seed funding that allows researchers to act on their ideas and help make discoveries that could lead to answers for the important questions surrounding how we can better treat and prevent diseases that afflict us today. Each Tullie and Rickey Families SPARK Award is $25,000 and must be spent within one year. The goal is to enable scientists to generate enough preliminary data through their projects to attract additional funding to further their research and careers.

In addition to funding cutting-edge research, the Tullie and Rickey Families SPARK program also trains and advances the careers of the next generation of researchers. Of course, SPARK Award winners ultimately gain experience in running an independent research project, an important career milestone. But equally as important, all SPARK finalists receive coaching in how to communicate their research to the public and how to present their ideas to funders.

Each year, LJI receives proposals from its many early-career scientists. A review panel (listed on page 21) narrows this pool down to the finalists who then have the opportunity to pitch their ideas to a donor panel with the hopes of securing funding to pursue their projects. Since 2017, more than 214 donors (listed on pages 28-29) have generously funded 39 projects, all of which have the power to transform human health and launch the careers of promising researchers.

We are incredibly grateful to all of the donors who are empowering young investigators to take risks that bridge the gap between imagination and ground-breaking discoveries, for The Tullie and Rickey Families SPARK Awards for Innovations in Immunology.

About the Tullie and Rickey Families

From left to right: Judy and Tom Tullie, Dave and Brenda Rickey

We'd like to thank and recognize the incredible support and generosity of LJI Board Director Tom Tullie and his wife Judy, as well as LJI Board Director Dave Rickey and his wife Brenda, and their families. Their joint commitments to the SPARK program made in 2019 meant LJI has the assurance that this program will have funding in place for several awards a year for a decade. That year, LJI honored their commitment by renaming the SPARK program to *The Tullie and Rickey Families SPARK Awards for Innovations in Immunology.*

Tom Tullie was one of the first SPARK pitch reviewers in 2018, and that was what drove him to get his family engaged in the program. "I've been an entrepreneur and involved in innovation my entire career, but this is the most exciting program I've seen, because the ideas these young scientists have are not only fascinating, they have the potential to generate breakthrough discoveries that will someday save lives," says Tom. Since then, Tom and his wife Judy and their daughters Jaqueline, Anna and Samantha have all taken part in the pitch review process.

For the Rickeys, involvement in the SPARK program was first spurred by an invitation from Tom, and also by the couple's belief in the Institute's mission itself. "Brenda and I love the idea of 'life without disease' because so many of our friends and family have suffered or died from cancer, heart problems, dementia, and other ailments," Dave says. The Rickeys were attracted to the SPARK program largely due to its structure and opportunity to drive research forward. "In the philanthropy world, we like to give money where there will be a return. This program is really tangible and it's good to see."

The Tullie and Rickey families hope that by ensuring the program's longevity, other donors will be inspired to contribute to the program to fund additional awards and get as many of these ideas off the ground as possible. "I'd love to see people continue to join us," shares Tom, "I guarantee you that it will be well worth our investment because these ideas are unbelievable and they need to get funded."

The Tullie and Rickey Families SPARK Awards program represents an opportunity for donors of all levels to have a profound impact on the future of early-career researchers and also help ignite the next big breakthroughs. You can donate online at donate.lji.org/SPARK or reach out to Clare Grotting at clare@lji.org or 858-752-6872 if you'd like to discuss other giving options.

SPARK

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SPARK AWARDS FOR
INNOVATIONS IN IMMUNOLOGY

It is our pleasure to present the 2023 Annual Report for the Tullie and Rickey Families SPARK Awards for Innovations in Immunology. In the following pages, we hope you enjoy reading the results of the 2022 SPARK winners' research projects and the scientist who won the very first "Progress Pitch" competition, allowing him to continue his project for an additional year.

Read on for progress updates from the 2023 SPARK winners, as well as highlights about former SPARK winners who have been promoted, earned follow-on funding, and published significant findings.

We are grateful to all of the donors, volunteers, and scientists who make this program possible!

Heather Callaway, Ph.D.

What if we could design longer-lasting vaccines?

FUNDED
JANUARY 2022

FUNDED BY
The generosity of the Rosemary Kraemer Raitt Foundation Trust

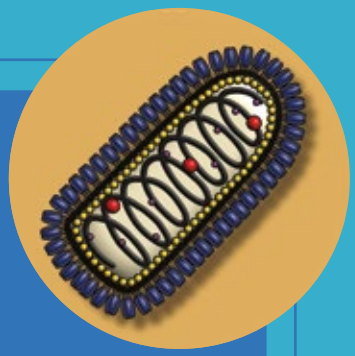
What was the goal of your SPARK project?

Rabies is nearly 100% lethal, and untreated rabies infections kill over 50,000 people every year. While rabies vaccines and antiviral antibody therapies exist, they both have fundamental flaws that prevent their universal use. Rabies antiviral antibody treatment consists of polyclonal human serum and is derived from the blood of vaccinated volunteers. Additionally, rabies vaccines are only effective for a relatively short time after administration, making revaccination necessary. Post-exposure rabies treatment also consists of multiple doses of rabies vaccine in combination with human rabies immunoglobulin (HRIG). This combination treatment can be prohibitively expensive in many countries, especially in low-income countries.

I had two aims: 1) to test a stabilized form of rabies virus glycoprotein (RABV-G) as a vaccine candidate to determine if stabilization improved the longevity of the anti-rabies immune response, and 2) to identify new anti-rabies monoclonal antibodies for structural and functional characterization, as well as later development as a therapeutic candidate to replace HRIG. My long-term goal of this research is to develop new rabies vaccines and anti-viral antibody therapeutics to prevent rabies deaths.

Did you face any challenges?

The immunization strategy I used elicited good levels of serum antibodies, but I was surprised to detect there were unexpectedly few cells in the spleen that secreted anti-rabies antibodies. This finding warrants further investigation and could yield insight into why existing rabies vaccines elicit short-lived immunity.



SPARK IMPACT STATEMENT:

"The SPARK award has been an incredible opportunity to learn how to communicate science to the community, design my own research project, and learn about the real logistics behind research grants."

SPARK project results:

I found that mice vaccinated with stabilized RABV-G produced a more uniform and longer-lasting anti-rabies antibody response, compared to mice vaccinated with unmodified RABV-G. Mice that received the unmodified RABV-G vaccine produced either very high or very low levels of antibodies, depending on the mouse. Mice that received the stabilized RABV-G vaccine, in contrast, produced a much more uniform immune response, with similar antibody levels from all mice.

I also isolated new anti-rabies antibodies from mice immunized with RABV-G and solved their protein structures in complex with RABV-G to determine where they bind. These antibodies bind to the side of RABV-G at slightly different angles. One antibody binds with relatively high strength, while the other antibody binds with intermediate strength.

These antibodies give me important information about what makes a strong anti-rabies antibody, and they are also potential therapeutics that can replace human rabies immunoglobulin as a rabies treatment.

What's next for this project?

The data I have collected from this study shows that stabilized RABV-G is promising and it will allow me to continue to develop stabilized RABV-G as a new, improved, and long-lasting rabies vaccine. For my next steps, I need to try new vaccine formulations (for example, protein vaccines instead of DNA vaccines) and vaccination schedules to see if I can elicit greater numbers of anti-rabies antibodies, and directly compare my experimental vaccine(s) to commercial vaccines.

The new anti-rabies antibodies I isolated are also promising as candidate therapeutics, and will yield great insight into how antibodies act to neutralize rabies virus. I can use the structures I solved to pick out specific protein/protein interactions between RABV-G and antibodies, and I can introduce changes to the antibodies to engineer improved antibody therapeutics that bind with even greater strength or that can bind to glycoproteins from viruses related to rabies virus.

What's next for Heather?

In August 2023, I began a position as Assistant Professor at Montana State University. As I establish my own lab, I also plan to write a paper on stabilized RABV-G and the results of my SPARK project. I also plan to analyze the polyclonal antibody response to vaccination with the wild-type and stabilized RABV-G vaccines using negative stain electron microscopy. Longer-term plans for this work include formulating the stabilized RABV-G vaccine as a protein, vectored, or live-attenuated vaccine and comparing the magnitude of the antibody response to that of commercial rabies vaccines.

Maria Inês Matias, Ph.D.

What if we could convert pro-tumor cells into tumor-fighting cells?

FUNDED

JANUARY 2022

FUNDED BY

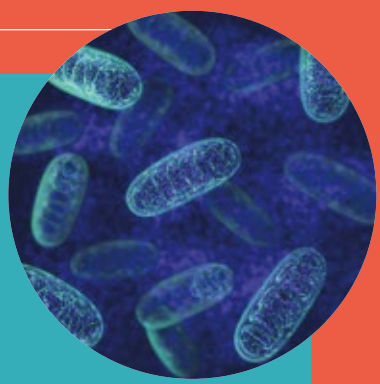
The generosity of LJI Board Director Emeritus Larry Spitcaufsky and Tiki Spitcaufsky

What was the goal of your SPARK project?

Anti-tumor immunotherapies that boost killer-cells represent a major breakthrough in cancer treatment. However, suppressive cells can enter the tumor and block tumor-killer cells. This is a major roadblock in the field of cancer immunotherapy. This unbalance between pro-tumoral and tumor-killer T cells in the tumor tissue is favored by the tumor's metabolic environment, which strongly inhibits the protective function of anti-tumor killer cells, while enhancing pro-tumor T cell function. My SPARK project aimed to uncover how nutrient availability in tumor tissue can impact how tumor-killing T cells differentiate and function. Ultimately, I want to know how we can transform pro-tumoral cells into tumor-fighting cells by modulating nutrient availability.

Did you face any challenges?

I needed to make several optimizations on the in vitro system that I was working on. As this system is the foundation of the entire project, it was important to make sure that everything was done correctly. This step delayed the proposed initial timeline but I was able to get back on track.



SPARK
IMPACT STATEMENT:

"My SPARK award gave me the confidence that I needed to believe in my scientific skills and opened the doors to new opportunities. It was great to be in close contact with several LJI donors who, through their passion and commitment, supported me throughout this year."

SPARK project results:

For this project, it was important to develop an in vitro model: a model where we could generate (in petri dishes) tumor-killer T cells similar to the ones that we find in our immune system. While the model to generate pro-tumoral T cells is well studied, how to generate tumor-killer T cells remains unexplored. Thus, most of my time was dedicated to characterizing and optimizing a differentiation protocol for these cells. I optimized a protocol that generates three different cell types that share common features with the killer T cells of our immune system and two cell types that share characteristics with pro-tumoral T cells. I then started work to optimize the necessary protocols for performing proteomics analysis. This technique will allow me to identify thousands of differentially expressed proteins between tumor-killer and pro-tumor cells. Proteomic analysis in T cells is challenging, and I tested several protocols. After proteomics optimization, I performed different independent experiments in order to have samples (tumor killer and pro-tumor T cells) for my proteomics screening. I am now performing proteomic analysis of the collected samples. The outcome of this screening will help me to choose interesting metabolic genes that could be targeted to skew the balance between tumor-killing and pro-tumor T cell development. I hope to identify a specific condition that turns pro-tumor T cells into tumor-fighting cells. This will be crucial for the improvement of current cancer immunotherapies.

What's next for this project?

I plan to pursue this study and identify metabolic factors that can be targeted to convert pro-tumor T cells into tumor-fighting cells. This project included several technical challenges that had to be overcome by months of protocol optimizations. Now that the important foundations of this project are ready, I can confidently move on for the next steps. I intend to publish this work once the next phase of experiments are completed.

What's next for Maria?

I recently applied for a grant from a private foundation using the preliminary data obtained from my SPARK project. If I win this grant, I'll have one year of salary guaranteed and funding for the next round of experiments. I would also be in close collaboration with Pfizer. In a few years I would like to be running my own independent laboratory.

Melissa Meyer, Ph.D.

What if we could target pro-tumoral immune cells while leaving infectious-disease fighting immune cells unharmed?

FUNDED
JANUARY 2022

FUNDED BY
The generosity of LJI Board Director Tom Tullie and the Tullie Family

What was the goal of your SPARK project?

My SPARK award supported work to understand how the immune cells that prevent infection differ from immune cells that support tumor progression in melanoma patients. To do this, I utilized a new technology, called CITE-seq, to understand the markers present on the outside of cells and associate them with an anti-bacterial or pro-tumoral phenotype.

I worked with a larger group of colleagues to design a custom reagent necessary for the proposed experiments. After a long delay, the custom reagent finally arrived and we performed the large-scale proposed experiment. The downstream analysis is now underway. We are working through the pipeline, striving to optimize the analysis for neutrophil detection. Once we clean up the data, we will look for the pro-tumoral and antibacterial neutrophils and interrogate how they change from healthy individuals to cancer patients.

Did you encounter any obstacles?

I experienced delays in the development of a custom reagent that was imperative to the experiments in my project. To adjust, I simply delayed the experiment until the arrival of the custom reagent. This means I am not as far along in the analysis as I had hoped I would be at this point. Because the custom reagent was critical to the novelty of the study, it was reasonable to delay results rather than pivoting to something more widely available.

SPARK IMPACT STATEMENT:

"My SPARK award has propelled my career forward. It has not only provided funds for me to pursue an important, clinically relevant question, but it has also given me the confidence to continue on my career path in pursuit of an independent academic position."

SPARK project results:

I used the new technology, CITE-seq, to profile immune cells, including neutrophils, in the blood of healthy human donors and melanoma patients. Samples from two healthy donors and three melanoma patients were collected as part of this project. From each donor, I collected 20,000 cells (40,000 from one melanoma patient), double what was previously possible with an older model of the cell separator. I then measured 201 molecular targets on the outside of the cells, a major advance compared to previous technology, which allowed us to detect 40-50 targets on the outside of the cell. Detection of the molecular signature on the outside of the cell is what makes CITE-seq unique and particularly poised to better detect neutrophils. I went on to perform a deeper-than-usual analysis on the molecular signature on the inside of the cell. Last, I combined the SPARK-funded samples with a similar analysis on human bone marrow. Because these data were collected and analyzed together, I can look across these samples to have a more integrated understanding of what is going on in the immune system during healthy periods and in cancer development. In short, I collected a large dataset.

I have a plan to tackle the data and come up with answers as well as more hypotheses and questions. I expect to find subsets of neutrophils that are new and identifiable as pro-tumoral or anti-bacterial. We will then identify unique molecular signatures on the surface of these subsets. The cell surface location of the molecular signatures makes them prime drug targets. In the end, I will devise strategies to target bad pro-tumoral neutrophils while leaving good antibacterial neutrophils intact.

What's next for this project?

The final analysis for my SPARK project will provide new drug targets that I can work to move towards the clinic. I will take the identified targets on pro-tumoral neutrophils and develop drugs that will work to deplete these harmful cells in cancer patients. If I find a marker that is specific to the anti-bacterial neutrophils, I will look for ways to engage that molecule to promote infection-fighting functions of neutrophils. I can then study mouse models to determine if this strategy might work to tackle cancers. I will test the effects of these drugs on tumor growth as well as whether the drugs leave an individual susceptible to infection. This preclinical work in a mouse model will pave the way to clinical application.

I will prepare a publication during the next year. The target journal and story will depend on the results found, but this research would likely serve as a resource paper in a journal such as *Immunity* or *Nature Immunology*.

What's next for Melissa?

I plan to launch my independent research career using the hypotheses I generate from this data-set. So, I plan to apply for early stage investigator grants and R01 or R21 using the data generate from this project. Those grant applications will begin later this year.

I hope to finish up my postdoctoral fellowship in the next year. This fall, I will go on the academic job market with hopes of landing an assistant professor position in the midwest studying aspects of targetable myeloid biology in disease.

Estefania Quesada-Masachs, M.D., Ph.D.

What if a person's insulin-producing beta cells are orchestrating their own demise?

FUNDED
JANUARY 2022

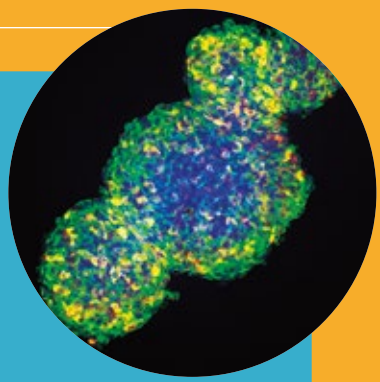
FUNDED BY
The generosity of LJI Board Director Barbara Donnell and Denny Sanford

What was the goal of your SPARK project?

Type 1 diabetes (T1D), an autoimmune disease that usually affects young people, is on the rise worldwide, and to date there is no cure. We still do not completely understand the underlying processes that drive the dysfunction and destruction of insulin-producing beta cells in the pancreas. One of the questions raised in the last few years is whether beta cells are orchestrating their own demise by causing a direct response in the immune cells around them. My hypothesis is that beta cells can affect the behavior of immune T cells around them.

Did you encounter any obstacles?

My experiments went really well. I was able to optimize most of the techniques and get preliminary data to ask for follow-on funding. The main obstacle I encountered is that I need more time and funding to perfect the staining experiments and the 3D image analysis strategy, and also to repeat the experiments to get more solid data (I need to verify that my findings are replicated in multiple donors).



SPARK
IMPACT STATEMENT:

"SPARK is a very valuable program. For a young investigator like myself, it was important to develop a project with autonomy, to test a risky hypothesis, and to practice managerial functions such as working with a relatively strict time frame and budget. I also learned a lot about public speaking and communicating science to a lay audience. I enjoyed my interactions with donors and it was very energizing to get to know people from the public who are interested in our science."

SPARK project results:

I successfully conducted the experiments, using both human pancreatic islet spheroids (a 3D in vitro platform of "perfect islets," that helps us to study and recreate some of the processes that happen in T1D) and human isolated native islets

I co-cultured human islets (spheroids and native) with immune cells (CD4+ T cells) under different conditions. I studied the interaction between the immune cells and the islets with a special focus on the insulin-producing beta cells using confocal microscopy and flow cytometry.

I found that when immune CD4+ T cells are in a resting state (not activated), they are less prone to infiltrate the islets, and they do not affect the capacity of the islet beta cells to secrete insulin. Instead, when immune (CD4+ T cells) were activated, they infiltrated the islet and caused some impact in the islet function. On the other hand, once again I confirmed our previous findings showing that pro-inflammatory cytokines (proteins secreted after infections or during local injuries, that can stress the islets) induce the expression of proteins in the islets such as HLA class I and HLA class II. These proteins are important because, in principle, they allow the islet beta cells to communicate directly with the immune system. Activated CD4+ T cells increase the expression of HLA class I by the islets co-cultured with these activated CD4+ T cells. This is relevant, because other immune cell types (like CD8+ T cells) can detect these HLA class I proteins and launch an additional immune response. Those findings suggest that the immune cells on the pancreas of patients with T1D are probably locally activated, and for that reason are more prone to invade the islet and make it dysfunctional.

Even one specific immune cell type seems to be able to induce changes in the beta cells, which can culminate in an orchestrated immune response (by implicating other immune cell types around it). In our field, it is critical to understand whether type 1 diabetes is primarily triggered by the beta cell itself or by the immune system—and to understand the chronology of the events in the human pancreas. This knowledge will drastically change future therapeutic approaches.

What's next for this project?

I plan to continue this project. With my SPARK award, I learned that CD4+ T cells are able to infiltrate the islet when activated and that having enough of these cells can induce HLA class I expression in the islet, which can have important implications in T1D. Additionally, I improved some technical aspects that are innovative and relevant when asking for further funding. With early preliminary data, I submitted a career award from Juvenile Diabetes Research Foundation and an R01 grant to continue with the project. Unfortunately, I didn't get funded in the first cycle, but I received constructive feedback and now I have more data, so I plan to apply for those grants again.

What's next for Estefania?

I recently got an R01 from the NIH and I am launching my independent research career in academia. I will need more funding in order to be successful in academia, so I plan to continue applying for grants. My next career step is to become an Assistant Professor.

Priyanka Saminathan, Ph.D.

What if we could uncover the biological mechanism that makes men more vulnerable to COVID-19?

FUNDED
JANUARY 2022

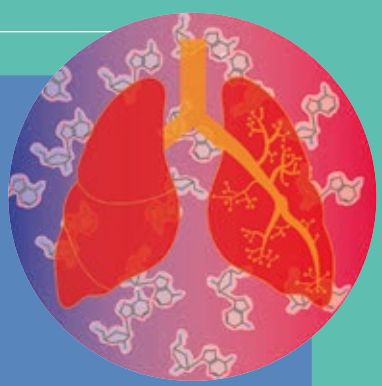
FUNDED BY
The generosity of Bill Passey and Maria Silva, and various 2021 donors

What was the goal of your SPARK project?

I set out to discover why men are more vulnerable to COVID infectivity. We have so far found that men tend to have increased adenosine deaminase (ADA) activity in their serum. Previous studies in the Sharma lab have shown that increased ADA activity can suppress the expression of Interferon Beta (IFNβ), a key antiviral cytokine important in early elimination of COVID infectivity. Thus, I hypothesized in this study that increased ADA activity in men could contribute to their increased vulnerability to COVID. To test this hypothesis, I employed the use of several in vitro, in vivo, and bioinformatic approaches.

Did you encounter any obstacles?

I was initially interested in validating our preliminary results in mice. But due to some technical difficulties (a long timeline for biosafety lab training), I decided to utilize this study to further strengthen our hypothesis using data from human patient groups. This shift actually allowed us to uncover further key possible regulars of ADA2 activity in men and formulate a working hypothesis, which we are now testing using human bronchoalveolar epithelial cells.



SPARK
IMPACT STATEMENT:

"Securing SPARK funding and learning how to communicate science to a lay audience has given me the confidence to believe in myself and my science, and connect with people who are looking for an opportunity to help make research possible.

I have learned how to manage a small project, train and mentor project assistants, and even provided high school students with the opportunity to learn about sex biases in immunology. I believe my SPARK project has ignited years worth of research that may not have otherwise been explored."

SPARK project results:

To better understand vulnerability to SARS-CoV-2, we wondered if measuring molecular players called Endogenous Retroviral elements (ERVs) could be used as a proxy for measuring ADA activity in the blood and tissues in humans. Using several publicly available data sets, we analyzed sexual dimorphism of ERV expression in blood and serum samples. Interestingly, we observed a significant increase in ERV expression in the lung tissue of females, compared with males, from the same cohort. Given that immune cells called monocytes and macrophages are the major contributors on ADA activity in the serum, we hypothesize that in the lungs, where monocytes can become long-living differentiated macrophages, the increased ADA activity could contribute to the decreased ERV expression in men, thus subjecting them to lowered tissue IFNβ during acute infections.

Recent research has identified that epithelial cells of the upper and lower respiratory system distal lung are primary targets of COVID infectivity. To understand how this decreased ERV expression and increased ADA activity in the lungs can contribute to lowered IFNβ response in men, I collaborated with Dr. Ian Mathews ('18 SPARK winner) and the Hedrick Lab to mine publicly available data from COVID patients. We analyzed bronchoalveolar lavage fluid and sputum samples for COVID infectivity, ADA activity and ERV expression, and also IFNβ and other cytokine expression. We identified a strong sex-bias associated with the expression of IFN stimulatory genes. This discovery suggests females are better poised to have a strong IFN response to COVID infection, compared with their male counterparts.

What's next for this project?

My SPARK award allowed this research to come much farther than I expected, and we are now on the way to publishing the results of this study in a reputable journal. I also hope to apply for NIH funding to continue this work.

In the meantime, we are taking steps to identify key immune cell populations that express ADA, and learn how these levels may change during COVID infection between males and females. To further validate the effect of ADA expression on COVID infectivity, we will be treating primary Bronchial Epithelial cells with exogenous ADA in vitro and watching for changes to IFNβ expression.

Our next goal is to identify how this translates to levels of IFNβ at the tissue level, which we believe is one step closer to identifying why men are more vulnerable to not only COVID, but to several other respiratory tract Infections such as influenza, pneumococcal disease, and the common cold.

What's next for Priyanka?

I see myself as a mentor and a project leader, either in industry or an academic setting.

Gurupreet S. Sethi, Ph.D.

What if we could cure asthma by erasing the immune system's memory of allergens?

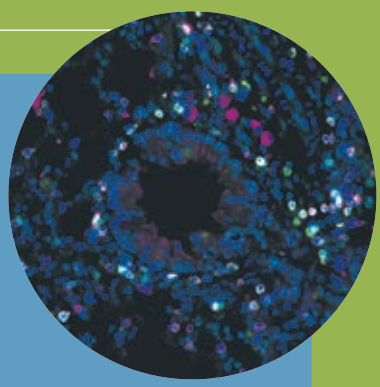
FUNDED
JANUARY 2022

FUNDED BY
The generosity of LJI Board Director Dave Rickey and the Rickey Family

What was the goal of your SPARK project?

I set out to develop a mouse model to study how the immune system remembers allergens. I worked with Foxp3 GFP reporter mice subjected to the multiple exposures to house dust mites extract (human allergen), which leads to the development of memory T cells in their lungs. By staining their lung tissue with fluorochrome antibodies, I tracked down a particular subset of lung-localized memory T cells. These memory T cells were sorted out and further processed for RNA extraction and single-cell RNA sequencing.

The next half of the project was critical. I aimed to analyze the single-cell RNA sequence data from these T cells using different bioinformatics tools to help us understand the allergic memory T cells. By comparing the allergic memory T cells with memory T cells of naïve mice, I hoped to shed light on the differentially expressed genes responsible for the long-lasting persistence of these allergic memory T cells.



Did you encounter any obstacles?

Life never goes as we plan, and neither does science. Unfortunately, due to some technical issues, all the samples of naïve mice were not of good quality, so we were left with no control samples to compare our lung-localized bad memory T cells of allergic mice. However, my plan B saved the project. I managed to sort out and sequence all the memory T cells in circulation, along with lung-localized memory cells of allergic mice. Furthermore, I realized that the circulatory memory T cells of allergic mice were an excellent internal control to better understand the long-lasting behavior of tissue-resident memory T cells of allergic lungs.

SPARK
IMPACT STATEMENT:

"Recieving a SPARK award was a very special event in my early scientific career. It not only gave me the confidence to think of bold ideas but also trained me to execute them within a definite time frame. I am honored to say that the SPARK award prepared me in so many ways for the next step in my scientific journey."

SPARK project results:

My SPARK award led to the development of a mouse model for studying allergic asthma—and the discovery of this dangerous group of "memory" T cells. I used two experimental mouse model groups, a naïve group and the allergic asthma group, to track down the lung-localized T cells carrying the markers of memory T cells.

By harnessing a technique called scRNA seq and using different bioinformatic tools, I found that the pool of memory T cells is not a single population but a heterogenous pool of different subsets of T cells. Interestingly, my results indicated that the pool of lung-localized memory T cells comprises more than ten different sub-population. It is essential to mention that all of these members are unique and carry different gene signatures.

What's next for this project?

I plan to explore which subset(s) of the long-lasting memory T cells in this study would be the most notorious member(s) of this extended family. We need to figure out which of these cells has the potential to survive and thus to be responsible for therapy-resistant asthma, a severe clinical situation that needs to be addressed on an urgent basis. I am honored to have received funding for one more year to test this extended SPARK idea.

What's next for Gurupreet?

I have been working in the field of asthma immunology for the last nine years, and the next step for me is to establish my lab in this direction. Notably, the SPARK award helped me to explore a very less-traveled path in the field of asthma immunology; to examine lung-localized, long-lasting bad memory T cells against allergens.

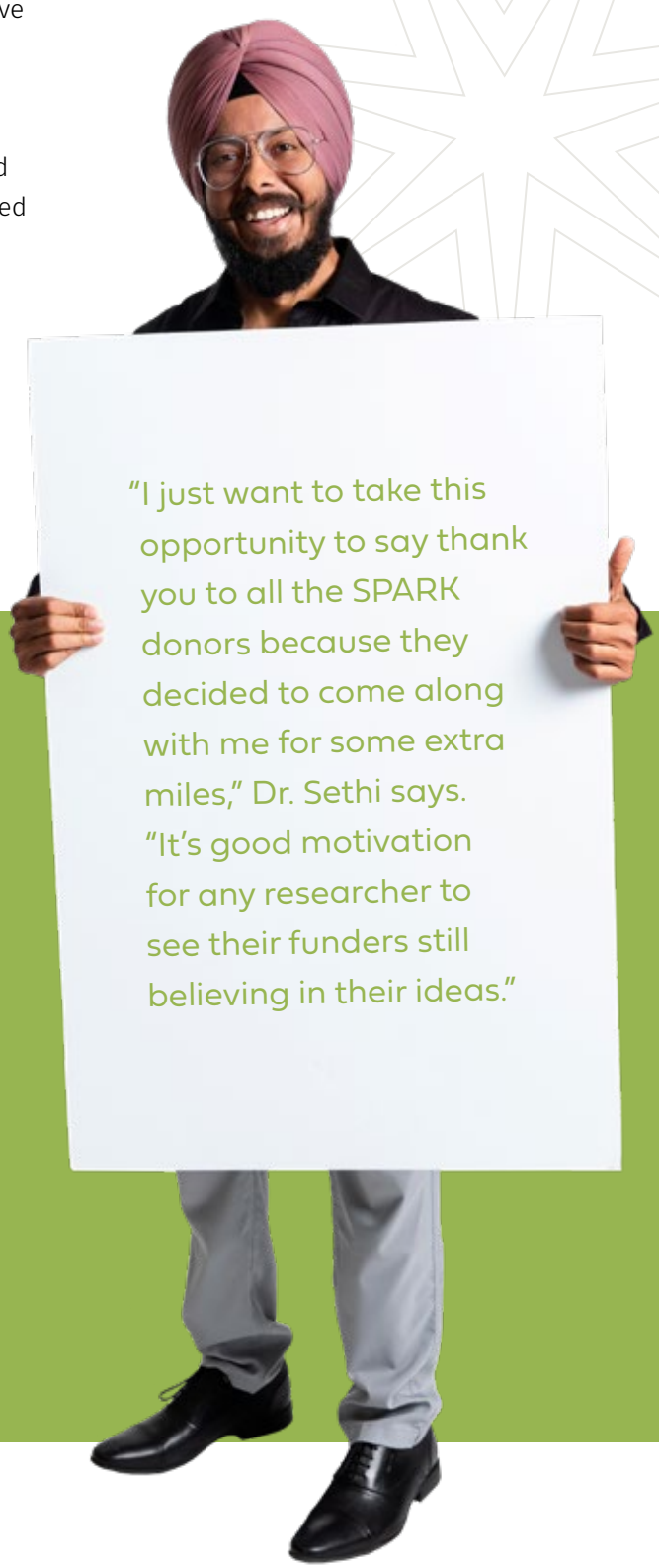
I strongly believe this project will help me to open more avenues in this area. In the near future, this information might help scientists identify possible targets to erase the immune system's long-lived allergic memory and help asthma patients live healthier lives.

2022 PROGRESS PITCH COMPETITION AND WINNER PROGRESS

Last August, the 2022 Tullie and Rickey Families SPARK Awards winners participated in an opportunity to win one additional $25,000 SPARK award to continue their research. This was the pilot year for the "Progress Pitches," the goal of which is to give an extra boost to some of the most promising early research funded by a SPARK award, helping the scientist to continue their momentum in establishing proof-of-concept and avoid a funding gap. Each scientist presented the progress they had made with their initial SPARK award, and proposed a continued line of research. A panel of SPARK program stakeholders and LJI Board Directors selected the winner.

2022 PROGRESS PITCH WINNER:

GURUPREET S. SETHI, PH.D.



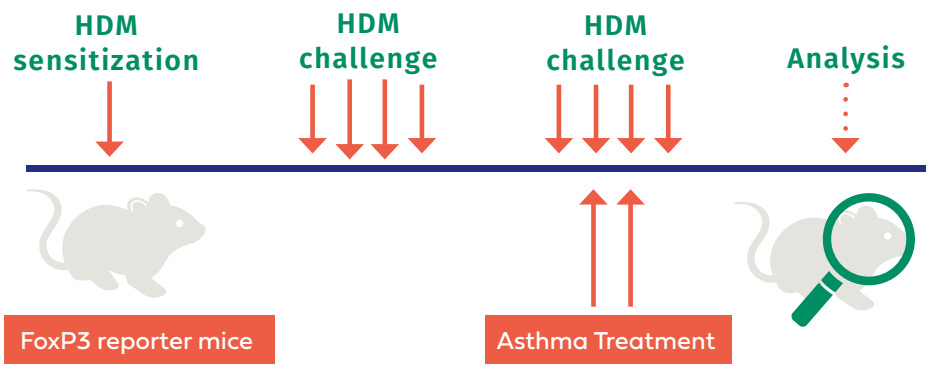
Dr. Sethi's initial SPARK Award allowed him to make progress in understanding asthma, leading to the development of a mouse model for studying allergic asthma—and the discovery of a dangerous group of bad "memory" T cells.

As winner of the 2022 SPARK Progress Pitches, Dr. Sethi proposed the additional funding to figure out which memory T cells in allergic lungs are the worst. These mysterious cells can survive asthma treatments and may be responsible for the reappearance of disease symptoms—a condition referred to as therapy-resistant asthma.

Scan this QR code to watch a video and to read more about Dr. Sethi's continued SPARK project.

What is the goal of this next phase of your SPARK Project?

Recently in the Croft lab we replicated the clinical situation of therapy-resistance in a mouse model. Now the plan is to uncover and understand the role of bad memory T cells under these complex asthma settings. Given the heterogeneous nature and long-lasting persistence of these memory T cells, I hypothesized their close connection with the reappearance of disease symptoms after stopping the therapy in severe asthma patients. I firmly believe that studying the gene profile of bad memory T cells that survived asthma treatment, and their comparison to that of the therapy-sensitive memory T cells, will take us one step closer to developing long-lasting therapies for asthma patients.



What progress have you made so far and what comes next?

So far, we have performed the main experiment by subjecting specialized mice, called "FoxP3 Reporter Mice," to multiple exposures of a human allergen, standardized house dust mites, in order to develop the pool of bad memory T cells in their lungs (see the figure above). We have already demonstrated that modulating bad memory T cells by targeting their surface molecules, namely CD3 co-receptors, provides temporary relief to disease symptoms. Thus, I decided to treat some allergic mice with anti-CD3 F(ab')2 antibodies to mimic the therapy-resistant-like settings in lab conditions. We then collected lung tissue, isolated the bad memory T cells, and processed them for RNA extraction. Finally, we obtained the single-cell RNA sequence data of allergic mice.

My next step is to analyze the RNA sequencing data to understand what markers of these bad memory T cells might be responsible for their long-lasting survival, even after receiving the asthma therapies. Understanding this will be helpful in managing, and perhaps even curing, severe asthma.

2023 SPARK Award winners from left to right: Felix Nettersheim, M.D., Erik Ehinger, Dawid Zyla, Ph.D., Sloan Lewis, Ph.D., Gregory Williams, Ph.D. (not pictured: Rosa Isela Gálvez, Ph.D.) Photo credit: Melissa Jacobs

SPARK AWARDS CELEBRATION

La Jolla Institute for Immunology welcomed guests to an outdoor reception on June 1, 2023 in celebration of the six winners of the 2023 Tullie and Rickey Families SPARK Awards. The reception was a chance for LJI to recognize both the supporters of the Tullie and Rickey Families SPARK Awards program and honor the newest cohort of SPARK winners.

Guests had the opportunity to hear remarks from two former SPARK winners, **Tom Riffelmacher, Ph.D.** ('20 SPARK Award winner) and **Melissa Meyer, Ph.D.** ('22 SPARK Award winner), who spoke about the impact the program has had on their careers and research.

SPARK supporters Carol Kearney, Maria Silva, and Barbara Donnell together with Maria Matias Ph.D., '22 SPARK winner.

Judy, Anna and Tom Tullie, foundational donors of the Tullie and Rickey SPARK Awards for Innovations in Immunology.

Tom Riffelmacher, Ph.D., '20 SPARK winner.

THANK YOU, REVIEWERS

We are extremely grateful for the time and support of the application and pitch reviewers for the 2023 Tullie and Rickey Families SPARK Awards for Innovations in Immunology:

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Judy Tullie
Tom Tullie
David Webb, Ph.D.

2023 WINNER PROGRESS UPDATES

Erik Ehinger

WHAT IF WE CAN HIJACK THE IMMUNE SYSTEM'S RUBBISH-DISPOSAL SYSTEM TO TOUGHEN T CELLS AGAINST CANCER?

Although immunotherapies have been successful in treating cancer patients, they are limited, especially in solid cancers, by a phenomenon known as T cell "exhaustion." This term refers to the fact that tumor-infiltrating T cells are initially effective in destroying cancer cells, but later lose function and fail to destroy the tumor. Exhausted T cells upregulate inhibitory surface receptors, including PD-1. Treatment with antibodies to these receptors forms the basis for current approaches to cancer immunotherapy; however, blocking individual inhibitory receptors, or even combinations of inhibitory receptors, rarely achieves a complete cure.

In a quest to develop the next generation of cancer therapies, we are harnessing cells' rubbish-disposal system to target the root cause of immune cell exhaustion, rather than current approaches that only target the peripheral receptors (expressed as a consequence of the exhaustion program).

We set up a mouse proof-of-concept system to show whether degrading exhaustion-causing proteins could counter T cell exhaustion. We found that by degrading each of our targets, we are able to substantially decrease the number of "terminally exhausted" T cells. These T cells regained their ability to secrete cytokines, which are paramount to their function in tumors. The remaining phase of this project will show whether our approach directly improves T cell tumor killing, and whether these targets are potent in humanized systems. In parallel, we are embarking on the beginning discovery stages of a lead compound that will unlock the totality of the entire immune system against solid cancers by using the rubbish-disposal system.

Dawid Zyla, Ph.D.

WHAT IF WE CAN USE VIRUSES TO MONITOR THE EFFICACY OF OUR IMMUNE SYSTEM?

The human body is host to many viruses that stay with us throughout our lives. Under normal conditions, equilibrium is maintained, keeping non-pathogenic viruses suppressed. Disease or medication can weaken the immune response, escalating virus levels, a condition known as viremia. Latent viruses might serve as immune function indicators, enabling the prediction of infections and rejection of transplanted organs. With my SPARK award, I aim to develop an antigen-based method to measure virus concentrations in the blood, effectively controlling immunosuppression medication, and decreasing the likelihood of viremia. So far, in collaboration with LJI's Bioinformatics Core, we have analyzed numerous sequences from the GeneBank database. Parts of the sequences did not correspond to existing Anelloviridae family categories, requiring the creation of a tool for uncategorized virus classification. These sequences provide an improved approach to virus surveillance and control, critical for patients undergoing immunosuppressive treatment due to an organ transplant or cancer, as they exhibit increased Anellovirus concentrations. Our main objective remains the development of an Anellovirus detection assay to evaluate immune competency. To achieve this, we need to generate antibodies that recognize Anelloviruses, and determine the most common species of these viruses in the blood of immunocompromised patients.

Felix Nettersheim, M.D.

WHAT IF WE CAN ENHANCE A HEART DISEASE VACCINE BY BLOCKING REGULATORY MOLECULES?

Worldwide, almost 18 million people die each year from cardiovascular diseases. A major cause of heart disease is an uncontrolled autoimmune attack against the blood vessels. Accumulating evidence suggests that a vaccine with cholesterol particles can dampen this autoimmune process. However, such vaccines elicit a considerably weaker response compared to vaccines against bacteria or viruses. I aim to investigate whether blocking two regulatory molecules can increase the effectiveness of a heart disease vaccine.

My first experiment was to analyze the vaccine response in mice lacking one of the two regulatory molecules. While a lack of the first molecule did not make a difference, the vaccine response in mice lacking the second molecule was more than doubled compared to control mice.

Next, I tested whether pharmacological blockade of both molecules simultaneously enhances the vaccine response. Consistent with the first experiment, blocking one molecule approximately doubled the number of responding cells, whereas blocking the other had no relevant effect. Strikingly, blocking both molecules together enabled a more than six-fold higher vaccine response compared to control mice.

My third experiment, which I have yet to perform, will provide evidence as to whether the blocking strategy can enhance the clinical efficacy of a heart disease vaccine.

Rosa Isela Gálvez, Ph.D.

WHAT IF IMMUNE "CROSSTALK" COULD MAKE MALARIA PARTIALLY PROTECTIVE AGAINST DENGUE FEVER?

In tropical regions, where forty percent of the global population lives, the combination of poverty and numerous mosquito-borne pathogens present a unique challenge for the immune system. The immune system faces the task of dealing with multiple infections simultaneously, making it even more complex to study individual immune responses. My SPARK project focuses on T cell responses to two deadly infectious diseases in tropical regions: malaria and dengue fever. By analyzing samples from Ghanaian children, I aim to understand the immunological interplay between these infections and how T cell variations might impact dengue fever's severity.

My central hypothesis suggests that recurrent malaria in early childhood leads to the development of cross-reactive T cells, offering initial protection against subsequent Flavivirus infections, and potentially reducing severe dengue disease.

So far, through my collaboration with the Bernhard Nocht Institute for Tropical Medicine in Hamburg, Germany, I have examined 410 samples from children who were diagnosed with severe malaria, seeking possible undetected and asymptomatic dengue infections. As hypothesized, 17.3% of the tested sera had prior exposure to dengue virus without a previous dengue fever diagnosis. We are currently investigating whether T cells from these children show altered responses to dengue virus re-stimulation peptide pools, providing insights into the observed cross-reactivity mechanisms.

Sloan Lewis, Ph.D.

WHAT IF WE CAN IDENTIFY NEW WAYS OF DIAGNOSING AND TREATING MILK ALLERGY IN CHILDREN?

Cow's milk allergy is the most common food allergy in infants and young children, and the prevalence is increasing. There are no approved treatments except avoidance, which is dangerous because of the widespread use of cow's milk in food products. To make things more challenging, we do not currently have reliable ways of diagnosing food allergy, making this research critically important. A specific immune cell, the monocyte, is known to be involved in inflammatory diseases, but the role these cells play in the causes and effects of food allergy has yet to be explored. For my SPARK project, I am analyzing monocytes in a cohort of children with cow's milk allergy to try to discover an "immune signature," or a special signal from the cell that we can detect. We could then try to use that immune signature for diagnosing cow's milk allergy or potentially try to target these cells in treatment.

I have performed some preliminary experiments to determine the percentages of monocytes within total blood immune cells in pediatric donors. I am now optimizing the stimulation of immune cell populations with cow milk extract and a cow milk peptide pool to see how monocytes respond. This will lead to the main experiment for my project, which will allow me to assess monocyte immune signatures of cow's milk allergy.

Gregory Williams, Ph.D.

WHAT IF WE CAN DETECT DANGEROUS T CELL RESPONSES IN THE BLOOD OF ALS PATIENTS?

Amyotrophic lateral sclerosis (ALS, formerly Lou Gehrig's disease) is a fatal neurodegenerative disorder that affects thousands of individuals each year. The exact cause(s) of ALS is unclear; however, we do know that several brain proteins become misfolded and neurotoxic. One potentially promising area of research on ALS involves the immune system. Through my SPARK project, I intend to answer key questions about how T cells may be contributing to ALS. These answers about the immune system could prove critical in the design of immunotherapies for ALS.

A major portion of my project involves the collection of blood from patients with ALS and age-matched healthy donors. Thus far, we have established collaborations with three exceptional clinical sites that provided blood samples that were then processed by LJI's John and Susan Major Center for Clinical Investigation.

To date, we have received 20 ALS samples, surpassing the intended goal of 15 recruitments. We are still actively receiving healthy control samples. I have performed quality control analysis on a subset of the samples and determined that they are of good quality for our intended assays.

Once both the donor blood samples and the proteins are ready, I will run assays to measure and compare the inflammatory response towards ALS-related proteins.

SPARK STARS

In addition to providing seed funding for innovative ideas, the Tullie and Rickey Families SPARK Awards program also aims to train and support the most promising early-career scientists so that they are equipped and encouraged to stay dedicated to medical research. Throughout 2023 we've shared some of the successes of our "SPARK Stars" who are demonstrating that donor investment in SPARK is helping to ignite discoveries and field the next generation of researchers. In case you missed them, here are a few of those updates and links to read more:

DANIELA WEISKOPF, PH.D.,

'18 SPARK WINNER

In November 2022, Research Assistant Professor Daniela Weiskopf, Ph.D., was among seven LJI scientists named to the 2022 Highly Cited List, released by Clarivate. This distinction recognizes scientists whose papers have ranked in the top 1% of citations for their field of study between 2010 and 2020. For Dr. Weiskopf, being named to the Highly Cited List reflects the global impact of her COVID-19 research.

ANNIE ELONG NGONO, PH.D.,

'21 SPARK WINNER

In December 2022, Instructor Annie Elong Ngono, Ph.D., was recognized for her infectious disease research spearheading important studies into the human body's response to deadly pathogens such as dengue virus. Her dedication to global health and virology earned her acceptance to the Global Virus Network's (GVN) highly selective Rising Star Mentorship Program. The program offers early career virologists unique opportunities for close collaboration with more senior scientists and clinicians. Dr. Elong Ngono will also get to participate in exclusive GVN meetings and other professional development opportunities in virology.

CECILIA LINDESTAM ARLEHAMN, PH.D.,

'20 SPARK WINNER

In October 2022, Research Assistant Professor Cecilia Lindestam Arlehamn, Ph.D., was granted more than $25,000 through a Women's Health Access Matters (WHAM) Edge Award to support new research into Parkinson's and Alzheimer's disease. Dr. Lindestam Arlehamn aims to shed light on how sex-based immune system differences may affect the development and progression of these neurodegenerative diseases in men versus women.

MELISSA MEYER, PH.D., '22 SPARK WINNER & ERIK EHINGER, '23 SPARK WINNER

In 2022, Postdoctoral Fellow Melissa Meyer, Ph.D., and Graduate Student Researcher Erik Ehinger, each received $100,000 in Primer Phase funding from the Accelerator Primer Program, supported by LJI's longtime partner Kyowa Kirin. Earlier this year, both researchers each went on to receive an additional $200,000 in booster funding to continue their research from the same program.

TOM RIFFELMACHER, PH.D.,

'20 SPARK WINNER AND '24 FINALIST

In 2022, Tom Riffelmacher, Ph.D., was named Director of the Immunometabolism Core, LJI's newest research core facility. As Core Director, Dr. Riffelmacher uses cutting-edge techniques to study how immune cells work—and how we might boost cellular metabolism to better fight disease.

In May 2023, Dr. Riffelmacher published a new study in *Nature Cell Biology*, exploring the location, function, gene expression, and metabolism of mucosal-associated invariant T (MAIT) cells, in the mouse lung. The findings may now inspire novel vaccines and cell therapies that shift the balance between these two cell groups to help individuals fight specific pathogens.

ESTEFANIA QUESADA-MASACHS, M.D., PH.D.,

'20 AND '22 SPARK WINNER

In April 2023, Instructor Estefania Quesada-Masachs, M.D., Ph.D., won the 2023 Young Investigator of the Year Award from the Network for Pancreatic Organ donors with Diabetes (nPOD). This prestigious award recognizes Dr. Quesada-Masachs' groundbreaking research in type 1 diabetes.

In May 2023, Dr. Quesada-Masachs won a $2.7M R01 grant from the NIH which will allow her to study the role of killer T cells in the development of type 1 diabetes, and determine how these cells contribute to the destruction of insulin-producing cells in the pancreas. This accomplishment also earned her a promotion to Instructor at LJI.

GURUPREET S. SETHI, PH.D.,

'22 SPARK WINNER

In December 2022, Dr. Sethi earned a promotion to Instructor in the Croft Lab.

PRIYANKA SAMINATHAN, PH.D.,

'22 SPARK WINNER

In July 2023, Dr. Saminathan won $100,000 in funding from Gary and Shuko Clouse to support her research on Alzheimer's disease in the Sharma Lab. With this postdoctoral fellowship, Dr. Saminathan will continue to spearhead a humans-first approach to deciphering the unique metabolic/metabolite differences in women and men who develop Alzheimer's disease.

NEW FACULTY POSITIONS

HEATHER CALLAWAY, PH.D.,

'22 SPARK WINNER

In August 2023, Postdoctoral Fellow Heather Callaway, Ph.D., accepted a position as Assistant Professor at Montana State University where her lab will focus on understanding how antibodies bind to and neutralize rabies virus, and designing a long-lasting pan-lyssavirus vaccine.

SARA LANDERAS BUENO, PH.D.,

'20 SPARK WINNER

In July 2023, Instructor Sara Landeras Bueno, Ph.D., accepted a position as Research Professor at the University Cardenal Herrera in Valencia, Spain, where she will establish her own independent lab focused on pandemic preparedness research, integrating her knowledge of influenza virus, Ebola virus, and coronavirus to gain a comprehensive understanding of RNA viruses with pandemic potential, and to find broad-spectrum targets for therapeutic intervention.

DONOR HONOR ROLL

PHILANTHROPY FUELS THE NEXT GENERATION OF RESEARCH

Thank you to all of the generous donors to the Tullie and Rickey Families SPARK Awards for Innovations in Immunology. The donor list below represents all donors to the Tullie and Rickey Families SPARK Awards for Innovations in Immunology since the beginning of the program in fall 2017 through June 2023. Any donor with a SPARK icon after their name denotes they've given $25,000+ the equivalent of at least one whole SPARK Award.

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This year, La Jolla Institute for Immunology is participating in

San Diego GIVES,

a local day of giving, on

September 7, 2023,

for the first time.

Our goal for this day celebrating good will in our community is to fundraise $25,000 for one full SPARK Award.

With your help, we believe that we can reach this goal and enable one early career scientist at LJI to launch their first independent research project in 2024.

To learn more about **San Diego GIVES** and make a gift towards our goal, please visit the link below or use the QR code to access our fundraising page.

sandiegogives.org/organization/LJI

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SPARK ignited my curiosity,
Steered my career!
Thanks to all the donors,
for allowing me to come out of fear.

– Abhijit

You've SPARKed
an idea.
a career.
and hope for patients.
Thank you!
Melissa Mayer

Thanks to the SPARK award, I can develop a project entirely independent and focus on a disease (ALS) that is in need of more study.

– Greg Williams

Thank you for allowing
LJI's young scientists
to make their Spark!

Julie

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