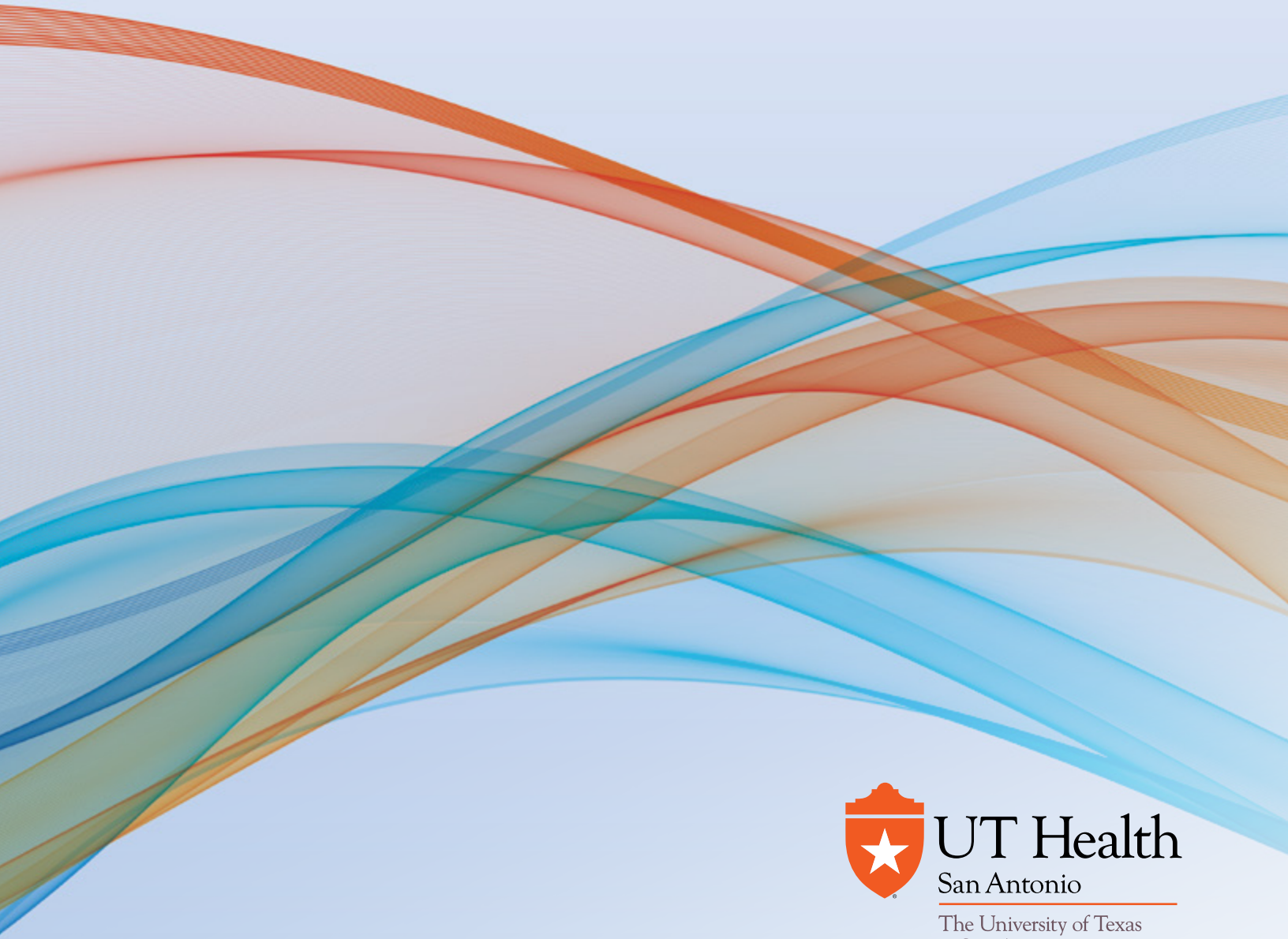
2025 ANNUAL REPORT

MAYS CANCER CENTER

Decreasing the burden of cancer in San Antonio and South Texas.



UT Health San Antonio

The University of Texas at San Antonio

OUR MISSION

Lei Zheng, MD, PhD

In 2025, the Mays Cancer Center at The University of Texas at San Antonio continued advancing its mission as the only National Cancer Institute-designated Cancer Center in South Texas, expanding access to research, clinical innovation and patient-centered care across the region.

This year marked significant momentum across our research and clinical enterprise. With more than 180 active clinical trials, including 54 newly opened studies, patients throughout South Texas gained greater access to investigational therapies and novel treatment approaches close to home.

A defining milestone in 2025 was the opening of the UT Health San Antonio Multispecialty and Research Hospital. The hospital strengthens our ability to provide inpatient and outpatient oncology services, clinical research and multidisciplinary care in a more connected environment for patients with complex cancer needs.

At the Mays Cancer Center and the UT Health San Antonio Multispecialty and Research Hospital, physicians and scientists are advancing a new era of medicine through groundbreaking therapies and highly specialized care once unavailable in South Texas. This year, clinical teams delivered the region's first ultrasound-based treatment for a brain tumor while also treating the first patients to receive advanced cellular therapies at the hospital. These milestones reflect more than individual achievements. They represent UT Health San Antonio's growing role as a leading academic health center where discovery, innovation and patient care come together to expand what is possible for patients across Texas and beyond.

Research growth continued to reflect the extraordinary momentum and scientific leadership emerging from the Mays Cancer Center in 2025. The cancer center secured a newly funded multi-project National Cancer Institute grant led by Reuben S. Harris, while overall research funding increased 11% and peer-reviewed funding rose 10%. The center also completed submission of its Cancer Center Support Grant renewal application to the National Cancer Institute, an important milestone expected to further affirm the groundbreaking science, translational discovery and lifesaving clinical innovation being advanced by Mays Cancer Center researchers and physicians on behalf of patients across Texas and the nation.

This year also represented a transformative moment for academic medicine and research in San Antonio with the announced merger of The University of Texas Health Science Center at San Antonio and The University of Texas at San Antonio to form UT San Antonio. Together, the unified institution is emerging as Texas' third-largest research university, creating unprecedented opportunities for scientific collaboration, innovation and discovery while strengthening the university's ability to advance healthcare, research and economic prosperity for Texas and the nation.

These accomplishments reflect the dedication of our faculty, researchers, clinicians, staff, donors and community partners, as well as the trust placed in us by patients and families across South Texas.

As you review this year's report, you will see a cancer center continuing to move forward with purpose and momentum while remaining deeply committed to improving the lives of the people and communities we serve.

With gratitude,

Lei Zheng, MD, PhD
Executive Director
Mays Cancer Center

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Cracking cancer's code

New research identifies novel drug target for acute myeloid leukemia treatment

By Claire Kowalick

Despite advances in cancer treatment in recent years, five-year survival rates for acute myeloid leukemia (AML) remain low at just 30% on average, according to the National Cancer Institute.

Factors in the development of AML are diverse and have many different drivers. A longtime goal for scientists in this field is to find a single drug that can treat all types of AML. A team led by scientists from The University of Texas at San Antonio and its academic health center, UT Health San Antonio, are closer than ever to this goal with their discovery of how a certain protein in the cell nucleus, paraspeckle component 1 (PSPC1), contributes to AML progression.

"The traditional AML treatment is the '3+7' chemotherapy regimen. However, AML is highly diverse, with more than 70 known driver mutations. Even targeted therapies like IDH inhibitors can only treat a small subset of AML patients. Given this diversity, we need a uniform, one-for-all drug target to effectively treat AML," said one of the study's primary investigators, Mingjiang Xu, PhD, professor in the Department of Molecular Medicine in the Joe R. and Teresa Lozano Long School of Medicine and CTRC Council Distinguished Chair in Oncology at Mays Cancer Center.

Xu's team published their findings online in Cell Stem Cell. Co-primary investigators include Feng-Chun Yang, MD, PhD, a tenured professor in the Department of Cell Systems and Anatomy, A.B. Alexander Distinguished Chair in Cancer Research, Mays Cancer Center and Jianlong Wang, PhD, Columbia University Irving Medical Center.

How PSPC1 fuels blood cancer

AML is an aggressive blood cancer that originates in the bone marrow and is known to impair the differentiation of myeloid cells, which leads to overproduction of immature cells. The condition inhibits normal blood cell formation and compromises the immune system. Successful treatment of AML is complicated for many reasons, said Xu. About a third of patients do not respond to chemotherapy and about half of the patients who achieved remission will relapse. Additionally, AML exhibits multiple mutations, often two or more in each cancer cell. Precision treatments may target one mutation but not be effective in other mutations.

THE DEVELOPMENT OF **PSPC1** INHIBITORS OR DEGRADERS WILL HAVE A HUGE IMPACT NOT JUST ON AML, BUT ALSO ON SOLID TUMORS LIKE LUNG AND PROSTATE CANCER, BY PREVENTING METASTASIS

PSPC1 is one of three proteins in a paraspeckle — tiny structures inside the cell nucleus believed to regulate gene activity by trapping and releasing RNA molecules. Research in solid tumors shows upregulation of PSPC1 is a strong factor for metastasis in many types of cancer. Upregulation of this protein is higher in AML than any other cancer.

Xu and his research team investigated the role of PSPC1 in AML using both human AML cells and mouse models. They discovered that PSPC1 is not required for normal blood cell formation but is essential for AML development.

In mouse models where PSPC1 was depleted, AML progression was significantly delayed and survival rates improved. Without PSPC1, myeloid cell differentiation resumed, effectively halting cancer progression. This suggests that PSPC1 plays a crucial role in maintaining AML characteristics. When tested in mouse models of different types of AML, knockdown of PSPC1 restored cell differentiation and stopped leukemic progression in each one.

"PSPC1 goes to specific chromatin regions and regulates important pro-leukemic genes through a non-canonical mechanism. When PSPC1 is knocked down, it can no longer promote these leukemic genes, leading to differentiation and apoptosis [cell death] of AML cells. We also found that PSPC1 alone regulates the leukemic gene transcription program consisting of many key leukemic genes," said Xu.

Concurrent role of PU.1

The mechanism behind PSPC1's oncogenic role was revealed through its interaction with PU.1, a transcription factor essential for blood and immune cell development. This interaction activated tumor-promoting genes, including NDC1, a gene not previously implicated in AML.

"PSPC1 and the transcription factor PU.1 work together to cooperatively drive the leukemic transcription program. This underlying mechanism makes PSPC1 a novel therapeutic target," said Xu.

What happens to AML without PSPC1?

Further experiments showed that knocking down PSPC1 led to reduced expression of AML-associated genes, increased myeloid differentiation and a decrease in cancer cell growth. Importantly, PSPC1 knockdown had no adverse effects on normal blood cell formation.

"Targeting PSPC1 could offer a new avenue for AML treatment," said Xu. "By disrupting its binding to the chromatin and/or interaction with PU.1, we may be able to restore normal myeloid differentiation and inhibit cancer growth."

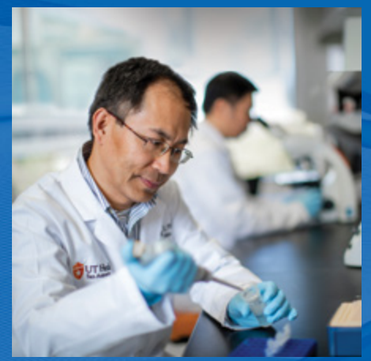
Beyond AML

Along with AML, PSPC1 has been identified in 21 different cancer cell lines, primarily in solid tumors where its overexpression contributes to disease progression. This raises the possibility that PSPC1-targeted therapies could have broader applications in oncology. Researchers are now exploring techniques to selectively inhibit PSPC1 in cancer cells while preserving its function in normal cells.

"We used PSPC1 knockout mice to show that PSPC1 inhibition is not toxic — the animals were completely normal even after knocking out PSPC1 and observing them for over two years. This makes PSPC1 an even more attractive drug target, as we can hit it [AML] hard without worrying about severe adverse effects," said Xu.

Xu said they are currently working with Daohong Zhou, MD, tenured professor of biochemistry and structural biology, associate director for drug development at Mays Cancer Center and director of The University of Texas at San Antonio's Center for Innovative Drug Discovery to begin testing potential drug candidates that may inhibit PSPC1.

"The development of PSPC1 inhibitors or degraders will have a huge impact not just on AML, but also on solid tumors like lung and prostate cancer, by preventing metastasis. This further increases the significance of our future drug development efforts targeting PSPC1," said Xu. ■



Mingjiang Xu, PhD, professor in the Department of Molecular Medicine in the Joe R. and Teresa Lozano Long School of Medicine and CTRC Council Distinguished Chair in Oncology at Mays Cancer Center

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Drug-discovery breakthrough for large and polar drugs

Discovery of chemical endocytosis could redefine precision medicine

By Claire Kowalick

A team of scientists, notably Zhengyu Wang, PhD, assistant professor in the department of pharmacology at The University of Texas at San Antonio and the Barshop Institute, led by Hong-yu Li, PhD, professor of medicinal chemistry and chemical biology with the department of pharmacology and the Barshop Institute, together with two other teams led by Hui-kuan Lin, PhD, from Duke University and Zhiqiang Qin, MD, PhD, from the University of Arkansas for Medical Sciences, uncovered the mechanism of cellular uptake for large and polar drugs and devised a novel strategy to optimize the capacity of drug delivery into these cells.

Published online in Cell, the study creates a strategy called chemical endocytic medicinal chemistry that may revolutionize how endocytic drugs in the future are designed and developed. Other contributors to this publication include Bo-Syong Pan, PhD, Rajesh Manne, PhD, and Che-Chia Hsu, PhD, (Duke); Jungang Chen, PhD, Phuc Tran, PhD, Tsigereda Weldemichael, PhD, and Jingwei Shao, PhD, (UAMS); and Dongwen Lv, PhD, Minmin Wang, PhD, Wei Yan, PhD, assistant professor, Hongfei Zhou, PhD, Gloria M. Martinez, PhD, Robert Hromas, MD, FACP, dean of the Joe R. and Teresa Lozano Long School of Medicine at UT San Antonio and Daohong Zhou, MD, professor, associate director for drug development at the Mays Cancer Center and director of the Center for Innovative Drug Discovery at UT San Antonio.

“Chemical endocytic medicinal chemistry has the potential to impact every aspect of endocytic drugs from drug discovery and development to clinical practice,” said Li.

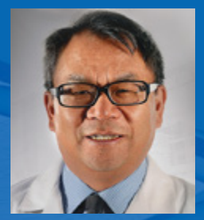
In this novel process, drug molecules are designed to better engage with CD36, a protein receptor found on the surface of many cells. By optimizing chemical interactions with CD36, the team was able to enhance the natural function of CD36, essentially escalating up the gateway for larger and polar drug compounds to enter the cell.

“This innovative chemical approach can potentially make any intravenous drug able to be taken orally. It can also promote any drug crossing the blood-brain barrier. This will remarkably broaden the number of agents we have to treat brain cancer or dementia,” said Robert A. Hromas, MD, FACP, dean of the Joe R. and Teresa Lozano Long School of Medicine at UT San Antonio.

Overcoming the ‘Rule of 5’ barrier for drug development

Small-molecule drugs have been limited due to the belief that passive diffusion was the primary mechanism of cell entry. One of the most promising developments in recent years in drug discovery is induced proximity. This drug discovery process utilizes molecules to bring proteins together to create a desired protein interaction and/or chemical reaction. Until now, molecules larger than 500 Daltons (Da) were believed to be practically unusable due to the challenges of cell access and bioavailability. This greatly restricted the kinds of compounds that could be developed as induced proximity drugs.

This new mechanistic discovery bypasses this limitation by chemically enhancing CD36-mediated uptake, amplifying the efficiency of larger and polar molecules to enter target cells. CD36 was known to play a role in lipid transport and metabolism, but the team found it also had unexpected potential for promoting cellular uptake of large and polar chemical drugs.



Hong-yu Li, PhD, professor of medicinal chemistry and chemical biology with the department of pharmacology and the Barshop Institute

“The Chemical endocytic medicinal chemistry has the potential to impact every aspect of endocytic drugs from drug discovery and development to clinical practice.”

“This discovery is important because it could rescue many drugs that were previously considered unusable due to poor absorption and turn them into clinically useful treatments for diseases,” said study author Hui-Kuan Lin, PhD, a cancer biology researcher and professor in the Department of Pathology at Duke University School of Medicine.

Provocative but well-validated results

In the study, the team first discovered and validated the CD36-mediated endocytic uptake of large and polar chemical compounds with sizes between 543 and 2,145 Da and then tested the efficacy of optimized CD36 action on the cellular uptake of proteolysis targeting chimeras (PROTACs), a class of large molecular compounds that includes an E3 ligase protein-binding domain, a binding domain for a target protein, and a linker. The team was astonished at the speed, effective uptake and potency of the compounds when utilizing the chemical endocytic medicinal chemistry strategy through CD36 interaction.

“This was completely unexpected in the research field,” said Li. “For decades, it was thought that molecules this large couldn’t cross membranes effectively, since the endocytic cellular uptake of chemicals was unknown. Through chemistry and biology, we identified CD36 as a protein for uptake and optimized chemicals better engaging with CD36 to internalize these drugs to more efficiently reach target proteins,” said Li.

The key experimental results were independently reproduced by each of the teams involved in the study.

“As the research conclusion is so provocative, we verified the key results multiple times,” said Li. “The implications of this for drug discovery and development are enormous.”

Rewriting the rules: Implications for drug development and the FDA

Traditional drug development is an extensive, expensive process focused on optimizing chemical compounds for passive diffusion into a cell by considering its contradictory characteristics of permeability, solubility and stability. This new process for endocytic drugs represents a paradigm shift that removes these challenges by using the membrane receptor-mediated cellular entry.

“This breakthrough discovery will force us to rethink how we approach efficacy and pharmacokinetics and toxicity,” said Li. “We believe it will also eventually change how regulatory agencies like the [Food and Drug Administration] FDA evaluate and approve new endocytic drugs.”

IN THE NEXT **10 TO 20** YEARS, THIS MAY BECOME A FOUNDATIONAL APPROACH IN DRUG DISCOVERY AND A NEW RESEARCH FIELD WITHIN MEDICINAL CHEMISTRY.

Patient stratification based on different CD36 expression

By analyzing tissue from prostate cancer patients, the team found CD36 expression levels varied widely. Li said this may explain why different patients respond differently to some cancer medications.

“By optimizing CD36 engagement through chemical endocytic medicinal chemistry, we may be able to target cancer and other diseases precisely through precision treatment based on the differential expression of CD36 in various tissues and different individuals,” said Li.

What comes next

Li said that, along with CD36, it is likely that there are additional cell receptors that could be targeted for chemical endocytosis, which Li’s laboratory continues to explore. He said the field of drug development may be significantly different in the next couple of decades due to this discovery and the potential it brings to induced proximity drugs. Li said there are high levels of CD36 receptors in intestine, brain and skin cells as well, so the chemical endocytosis strategy brings promise for better drug delivery that provides higher oral bioavailability, effectively bridges the blood-brain barrier or enters through the skin.

“In the next 10 to 20 years, this may become a foundational approach in drug discovery and a new research field within medicinal chemistry. We feel incredibly lucky to have made this discovery and opened the door to hope for previously untreatable diseases,” said Li. ■

Targeted drug offers hope for safe, effective senolytic treatment for liver disease, cancer

By Claire Kowalick

San Antonio has one of the highest rates of metabolic dysfunction-associated steatotic liver disease (MASLD) in the United States, largely driven by high rates of obesity and diabetes in the region. This chronic liver condition can lead to serious health conditions including severe liver fibrosis or cirrhosis and liver cancer, posing a significant public health challenge. Therapy that can slow the progression of MASLD and inhibit the development of cirrhosis and liver cancer remains an urgently unmet medical need.

A study published in Nature Aging highlights a promising new drug candidate that may safely and effectively eliminate harmful cells termed senescent cells from the liver to slow the progression of MASLD and inhibit the development of cirrhosis and liver cancer. The published studies were co-led by Daohong Zhou, MD, at The University of Texas at San Antonio and Liya Pi, PhD at The Tulane University and contributed by many other researchers from UT San Antonio (Yang Yang, PhD*, Araceli S. Huerta, LuZhe Sun, PhD, and Robert Hromas, MD, FACP), Tulane University (Natacha Jn-Simon*, Chunbao Sun, PhD, Tian Tian, PhD, Sreenivasulu Basha, PhD, and Xian-Ming Yin, MD, PhD), and the University of Florida (Yonghan He, PhD*, Peiyi Zhang, Wanyi Hu, Huadong Zeng, and Guangrong Zheng, PhD).

"Liver disease, particularly MASLD and hepatocellular carcinoma, disproportionately affects communities in San Antonio, where obesity and diabetes rates are high. Our study provides a promising path toward safer and more effective treatments for these diseases," said Zhou, tenured professor of biochemistry and structural biology, associate director for drug development at the Mays Cancer Center and director of the Center for Innovative Drug Discovery.

Daohong Zhou, MD, professor of biochemistry and structural biology, associate director for drug development at the Mays Cancer Center and director of the Center for Innovative Drug Discovery

"Liver disease, particularly MASLD and hepatocellular carcinoma, disproportionately affects communities in San Antonio, where obesity and diabetes rates are high. Our study provides a promising path toward safer and more effective treatments for these diseases."

HEPATOCELLULAR CARCINOMA IS THE MOST COMMON FORM OF LIVER CANCER WITH ABOUT

42K

PEOPLE DIAGNOSED EACH YEAR IN THE UNITED STATES, ACCORDING TO THE AMERICAN CANCER SOCIETY.

Senescent cells and liver disease

Senescent cells, sometimes called "zombie cells," are older cells that have stopped dividing but remain alive and secrete harmful toxins in the body. Accumulation of these cells has been linked to the onset and progression of MASLD, a chronic condition in which fat builds up in the liver that can lead to inflammation and damage. People with metabolic conditions like obesity and diabetes are more likely to develop MASLD.

Senolytics, a class of potential drugs designed to selectively remove senescent cells, may be an effective therapeutic for MASLD to reduce liver cancers. While senolytic therapies show promise, none are currently approved by the Food and Drug Administration for human use. This study highlights a senolytic developed in Zhou's lab and his collaborator Zheng that proved to be safer and more effective than many of the senolytics discovered previously.

A new approach

Zhou and his collaborators developed a drug candidate that works by degrading two proteins, BCL-xl and BCL-2, that help senescent cells avoid death and promote MASLD progression. Without these proteins, senescent cells self-destruct. These proteins also promote growth and survival of some tumors and without them, cancers that rely on them are weakened and often die. The new drug candidate successfully depleted both BCL-xl and BCL-2, leading to fewer senescent cells in the liver and a decline in MASLD progression and liver cancer development, all while avoiding toxic side effects of previous senolytics targeting these proteins.

Testing in liver disease model

Testing the drug candidate in cell culture and in a mouse model for MASLD developed by Pi demonstrated that it was a more powerful senolytic than predecessors and was able to target senescent cells more selectively in the liver. Notably, by targeting senescent liver cells it reduced fat buildup in the liver and scar tissue formation due to liver damage in the mouse model.

Potential to inhibit liver cancer

Hepatocellular carcinoma is the most common form of liver cancer with about 42,000 people diagnosed each year in the United States, according to the American Cancer Society. The study found that this novel drug candidate reduced the number and size of liver tumors in mice with MASLD, suggesting it could help inhibit liver cancer development. Importantly, they showed that the treatment was effective in mice even after the mice developed substantial MASLD and liver fibrosis but before cancer had fully developed via reducing senescent cell accumulation. Unfortunately, once cancer was established, the drug candidate did not stop tumor progression unless it was a BCL-xl/BCL-2-dependent tumor.

Advantages over existing treatments

Unlike broad-spectrum senolytics, which indiscriminately kill senescent cells (some of which may be beneficial for tissue repair), this new senolytic can selectively clear harmful senescent cells harming an organ, such as those in the liver. This selectivity is important, as indiscriminate removal of all senescent cells could disrupt wound healing and organ function, especially in older individuals.

Zhou said this senolytic's ability to selectively clear harmful senescent liver cells with reduced toxicity to platelets suggests it may offer a safer and more effective alternative to other senolytic therapies for MASLD.

"This breakthrough in targeted senolytic therapy opens the door to developing even more selective and less toxic drugs. Moving forward, we aim to refine these treatments to tackle a wider range of liver diseases and potentially other age-related conditions, ensuring broader clinical impact," said Zhou. ■

Drug found to double survival time for glioblastoma patients

By Steven Lee

A drug developed at The University of Texas at San Antonio has been shown to extend survival for patients with glioblastoma, the most common primary brain tumor in adults.

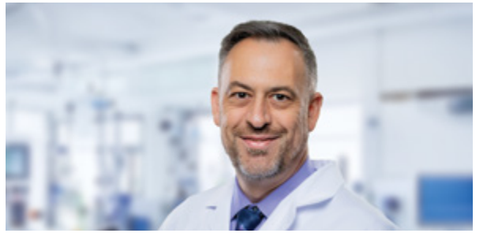
Results of a trial led by the university revealed that a unique investigational drug formulation called Rhenium Obisbemeda (186RNL) more than doubled median survival and progression-free time, compared with standard median survival and progression rates, and with no dose-limiting toxic effects.

"As a disease with a pattern of recurrence, resistance to chemotherapies and difficulty to treat, glioblastoma has needed durable treatments that can directly target the tumor while sparing healthy tissue," said Andrew J. Brenner, MD, PhD, professor and chair of neuro-oncology research with Mays Cancer Center. "This trial provides hope, with a second phase under way and planned for completion by the end of this year."

Brenner, who also is clinical investigator for the Institute for Drug Development at UT San Antonio and co-leader of its Experimental and Development Therapeutics Program, is lead author of the trial's study, titled, "Convection Enhanced Delivery of Rhenium (186Re) Obisbemeda (186RNL) in Recurrent Glioma: a multicenter, single arm, phase 1 clinical trial." It published in the journal Nature Communications.

Other authors also are with Mays Cancer Center, as well as UT Southwestern Medical Center of Dallas, Case Western Reserve University, University of Texas MD Anderson Cancer Center and trial sponsor Plus Therapeutics Inc., a clinical-stage pharmaceutical company receiving license to the trial technology to investigate the treatment of central nervous system cancers.

Brenner said that the median overall survival time for patients with glioblastoma after standard treatment fails with surgery, radiation and chemotherapy is only about eight months. More than 90% of patients have a recurrence of the disease at its original location.



Andrew J. Brenner, MD, PhD, professor and chair of neuro-oncology research with Mays Cancer Center

Rhenium Obisbemeda enables very high levels of a specific activity of rhenium-186 (186Re), a beta-emitting radioisotope, to be delivered by tiny liposomes, referring to artificial vesicles or sacs having at least one lipid bilayer. The researchers used a custom molecule known as BMEDA to chelate or attach 186Re and transport it into the interior of a liposome where it is irreversibly trapped.

In this trial, known as the phase 1 ReSPECT-GBM trial, scientists set out to determine the maximum tolerated dose of the drug, as well as safety, overall response rate, disease progression-free survival and overall survival.

After failing one to three therapies, 21 patients who were enrolled in the study between March 5, 2015, and April 22, 2021, were treated with the drug administered directly to the tumors using neuronavigation and convection catheters.

The researchers observed a significant improvement in survival compared with historical controls, especially in patients with the highest absorbed doses, with a median survival and progression-free time of 17 months and six months, respectively, for doses greater than 100 gray (Gy), referring to units of radiation.

Importantly, they did not observe any dose-limiting toxic effects, with most adverse effects deemed unrelated to the study treatment.

"The combination of a novel nanoliposome radiotherapeutic delivered by convection-enhanced delivery, facilitated by neuronavigational tools, catheter design and imaging solutions, can successfully and safely provide high absorbed radiation doses to tumors with minimal toxicity and potential survival benefit," Brenner concluded. ■

RESEARCHERS DISCOVER WAY TO SLOW OR BLOCK RECURRENCE OF GLIOBLASTOMA

By Steven Lee

Sandeep Burma, PhD, professor and vice chair (research) of neurosurgery with Mays Cancer Center

Researchers at The University of Texas at San Antonio have discovered a way to delay or even block recurrence of the deadliest brain cancer after radiation, bringing new hope for survival.

Ironically, the scientists found that the customary treatment for glioblastoma, ionizing radiation, can also cause tumors to recur, by generating senescent or aged cells that secrete molecules that can spur growth of neighboring cancer cells.

But they discovered that a new class of experimental "senolytic" drugs, given after radiation, can kill those senescent cells while sparing normal ones, thereby stemming recurrence. A senolytic refers to a novel class of small molecules thought to selectively induce death of senescent cells.

"These findings lend credence to the 'one-two punch' approach to radiation therapy, where radiation or other agents are first used to induce senescence in a tumor, and then the senescent cells are removed by a senolytic," said Sandeep Burma, PhD, professor and vice chair (research) of neurosurgery at UT San Antonio and Mays Cancer Center.

Burma and Bipasha Mukherjee, PhD, associate professor of neurosurgery at UT San Antonio, are lead authors of the study, "Targeting cIAP2 in a novel senolytic strategy prevents glioblastoma recurrence after radiotherapy," published Feb. 19 in the journal EMBO Molecular Medicine. Other authors also are with the departments of neurology, biochemistry and structural biology, and medicine at UT San Antonio, as well as the University of Texas Southwestern Medical Center in Dallas, and the Mayo Clinic of Rochester, Minnesota.

A double-edged sword

Recurrence of glioblastoma, the most common primary brain tumor in adults, is a major clinical problem as it occurs quickly and can be even more aggressive. Accordingly, Burma's lab has focused on understanding the forces driving recurrence and strategies to block the process.

Specifically, they are trying to understand whether senescence of cancer cells after radiation therapy – also called therapy-induced senescence, or TIS – might counterintuitively be driving recurrence.

Burma said that ionizing radiation, which is routinely and, in many cases, effectively used to treat cancer, is a double-edged sword since radiation also is a powerful carcinogen. For glioblastoma, radiation is still the most effective therapy. But radiation exposure also is the only known risk factor for its development, and could perhaps also drive recurrence.

When a tumor is radiated, a cancer cell can either die or remain alive but be permanently unable to divide further, a state called senescence, with both outcomes controlling tumor growth.

However, researchers in this study discovered that senescent glioblastoma cells secrete large amounts of growth factors and other molecules that can act on persisting cancer cells and encourage them to re-proliferate. What could be done about this problem?

End of senescence

Senolytic gets its name from the words "senescence" and "lytic," or destroying.

The researchers found that senescent glioblastoma cells rely on an anti-apoptotic protein, or one that slows or prevents cell death known as cIAP2, for survival. They also found that targeting cIAP2 with a senolytic drug called birinapant in mouse tumor models after radiation could kill senescent cells while sparing normal cells.

They tested their approach in multiple mouse models of glioblastoma and found that while the drug was not effective on its own, it was very effective at delaying or even preventing recurrence if given as an "adjuvant" after radiotherapy.

"These pre-clinical results highlighting a novel senolytic approach for glioblastoma are very exciting from a clinical standpoint as they clearly indicate that significant improvement in patient survival may become possible by eliminating senescent cells arising after radiotherapy," Burma concluded. ■

Uncovering how some cancers outsmart the immune system

By Claire Kowalick

For years, scientists have found the overexpression of a specific protein called FOXM1 in a wide range of cancers, including ovarian, breast and pediatric cancers. A recent study by scientists at The University of Texas at San Antonio has now led to understanding its exact role in cancer development and is the first study to define the cascade of events that FOXM1 initiates to help cancer cells escape immune detection. The study, published in Nature Communications, examines how this key protein allows several types of cancer to evade the immune system. The insight opens the door to more effective immunotherapies, personalized treatments and even therapies to prevent cancer recurrence.

"In many cancers, FOXM1 expression is high, but it was unclear how and whether it contributed to immune evasion," said Manjeet Rao, PhD, professor in the Department of Cell Systems and Anatomy, deputy director of the Greehey Children's Cancer Research Institute and Greehey Distinguished Chair in pediatric heme-malignancies at UT San Antonio. "We discovered that FOXM1 has an amazing ability to support cancer cell survival and progression by dampening the anti-tumor immune response."

How cancer hides

As cancer cells develop, they mutate and divide uncontrollably, but also manipulate their environment to shield themselves from immune attack. Normally, immune cells such as T-cells detect and destroy abnormal cells, like cancer cells. However, tumors protect themselves by sending chemical signals that either exhaust immune cells or create an environment that prevents immune cells from functioning.

One way cancer evades immune attack is by suppressing cell-surface proteins called stress ligands, which alert the immune system to cancer cells. Rao's team found that FOXM1 directly manipulates this process. When FOXM1 levels are high, the expression of stress ligands is blocked, effectively making cancer cells invisible to immune surveillance.

Eye-opening discovery

Using CRISPR-Cas9 gene editing technology, scientists removed FOXM1 in cancer cells and tested their ability to form tumors in mouse models with intact immune systems. The results were dramatic — cancer cells without FOXM1 developed few or no tumors. In cases where small tumors developed, they regressed rapidly in most cases.
"It was eye-opening to see that a protein long known to be important for cancer cell growth also plays a central role in educating immune cells to not attack cancer cells," said Rao.

New path for immunotherapy

Current immunotherapies, such as immune checkpoint inhibitors, have revolutionized cancer treatment but remain ineffective for many patients. In breast and ovarian cancers, for instance, only a small number of patients respond to existing immunotherapy drugs. Rao believes that FOXM1 inhibition could dramatically improve these odds.

"FOXM1 is highly expressed not only in breast and ovarian cancers, but in many other types of cancer as well," he said. "By blocking FOXM1, we may be able to turn so-called 'cold tumors' into 'hot tumors'— tumors that attract immune cells and respond better to immunotherapy."

WE HAVE DISCOVERED THAT

FOXM1

HAS AN AMAZING ABILITY TO SUPPORT CANCER CELL SURVIVAL AND PROGRESSION BY DAMPENING THE ANTI-TUMOR IMMUNE RESPONSE.

Building immune memory

In another significant finding, Rao's team discovered that depleting FOXM1 not only enabled the immune system to attack tumors but also facilitated the creation of long-term immune memory. In mouse models where tumors were eliminated after FOXM1 depletion, reintroduction of cancer cells failed to generate new tumors.

"This tells us that immune memory has developed," said Rao. "If we can support that memory function, it could become a maintenance therapy to prevent cancer recurrence."

Toward personalized medicine

FOXM1 also holds promise as a biomarker to help guide treatment decisions. Rao's team found that patients whose tumors exhibit higher FOXM1 expression are less likely to respond to immunotherapy. Measuring FOXM1 levels could help physicians predict who might benefit from immunotherapy and who might require alternative therapies.

Promising early clinical results

While a specific FOXM1 inhibitor has yet to be developed, Rao and other UT San Antonio scientists have found early success in reducing the protein using imipramine, an antidepressant drug. In a small-scale clinical trial, breast cancer patients treated with an imipramine derivative showed promising response.

"Computer modeling revealed that this molecule may bind to FOXM1's DNA-binding domain, destabilizing the protein and causing it to degrade," said Rao. "The early clinical data are very encouraging."

The road ahead

Rao's team is also identifying peptides downstream of FOXM1 that may serve as targets for a multi-component preventative approach.

"The idea is to create a cocktail of these peptides and prime patients who have high FOXM1 expression, allowing the immune system to recognize and destroy cancer cells before they can establish new tumors," Rao said.

This approach has the potential to not only improve outcomes for current patients but may one day serve to prevent cancer recurrence.

"By targeting FOXM1, we are not just attacking the tumor directly but may also be able to retrain the immune system to identify and eliminate several types of cancer," Rao said. ■

"The idea is to create a cocktail of these peptides and prime patients who have high FOXM1 expression, allowing the immune system to recognize and destroy cancer cells before they can establish new tumors."

Manjeet Rao, PhD, professor in the Department of Cell Systems and Anatomy, deputy director of the Greehey Children's Cancer Research Institute and Greehey Distinguished Chair in pediatric heme-malignancies at UT San Antonio

Key DNA complex connected to PARP inhibitor cancer-drug resistance discovered

By Claire Kowalick

Scientists have made one of the most important discoveries to date in the study and treatment of BRCA1-deficient cancers and drug resistance.

Approximately one in 300 Americans have a pathogenic mutation in the BRCA1 or BRCA2 gene, placing them at an elevated risk of developing certain cancers, especially breast and ovarian cancer. Of those who develop these cancers and are treated with PARP inhibitors — a generally efficacious therapy that targets poly(ADP-ribose) polymerases that the cancer cells need for DNA repair and survival — most patients eventually develop resistance to the drug.

"This seminal discovery opens the door to understanding how some breast, prostate and ovarian cancers become resistant to an important class of cancer drugs called the PARP inhibitors. These drugs can generate tumor regressions lasting years, but ultimately almost everyone relapses due to resistance. The DNA repair complex studied in the report is a prime target for causing that resistance and, thus, is a novel target for therapy. Drugs against this complex could possibly extend the lives of cancer patients treated with PARP inhibitors," said Robert Hromas, MD, FACP, dean of the Joe R. and Teresa Long School of Medicine at UT San Antonio.

APPROXIMATELY

1 IN 300

AMERICANS HAVE A PATHOGENIC MUTATION IN THE BRCA1 OR BRCA2 GENE, PLACING THEM AT AN ELEVATED RISK OF DEVELOPING CERTAIN CANCERS, ESPECIALLY BREAST AND OVARIAN CANCER.

Pivotal understanding in PARP inhibitor resistance

Understanding the processes that cause cancer drug resistance is crucial for determining which treatment option would be most appropriate and for driving the development of new therapeutic approaches, said Patrick Sung, DPhil, director of the Greehey Children's Cancer Research Institute and associate dean for research in the Long School of Medicine.

A study published May 22 in Science, co-led by Sung, including scientists from the Dana-Farber Cancer Institute at Harvard Medical School, Columbia University Irving Medical Center and UT San Antonio, explains how dysfunction in a certain complex (CST complex) that is crucial to regulating how DNA breaks are repaired can lead to PARP inhibitor resistance in BRCA1-deficient cancer cells.

"Of all the things we have done, this one has the most impact on clinical practice over the long run. Everyone expects malfunction in these proteins to give rise to drug resistance, but they do not know why and how," said Sung.

What is the CST complex?

The CST complex is made up of three proteins and is a negative regulator of a type of DNA repair called homologous recombination. This means it suppresses that type of repair and cells must choose a different repair pathway. When this complex is working properly, PARP inhibitors target BRCA-deficient cancer cells and cause them to die because they cannot repair their DNA. Because this protein complex suppresses homologous recombination, if it is mutated or silenced, tumor cells can become resistant to PARP inhibitors and continue proliferation.

Patrick Sung, DPhil, director of the Greehey Children's Cancer Research Institute and associate dean for research in the Long School of Medicine at UT San Antonio

"We are going to take this project to a completely different level by solving high-resolution structures of different protein complexes to try to explain why activities are either enhanced or attenuated upon complex formation, what DNA binding is for in terms of function, and so on. This is only the beginning."

First author Cody Rogers, PhD, post-doctoral research fellow at the Greehey Children's Cancer Research Institute, said the CST complex is primarily recognized as a single-strand DNA binding protein involved in regulating telomere length. However, he said, this study highlights the different ways the CST complex works to suppress homologous recombination and that mutations in this complex readily cause PARP-inhibitor resistance in BRCA1-deficient cells.

"There are two different mechanisms. When CST restricts EXO1, it requires its DNA binding activity. But when it blocks the activity of BLM-DNA2, it acts by forming a complex with BLM-DNA2 that is non-functional," said co-first author Hardeep Kaur, PhD, senior research scientist at the Greehey Children's Cancer Research Institute.

Future investigation into CST complex dysfunction frequency

Sung said several investigations are underway that will continue to explore how mutations in this complex engender PARP-inhibitor resistance in BRCA1-deficient tumor cells.

"We are going to take this project to a completely different level by solving high-resolution structures of different protein complexes to try to explain why activities are either enhanced or attenuated upon complex formation, what DNA binding is for in terms of function, and so on. This is only the beginning," Sung said.

Another step in this research will include using epidemiological data to find out how often mutation in this complex occurs, which Sung believes could be a major reason for acquired resistance to PARP inhibitor therapy. For this research, colleagues at the Dana-Farber Cancer Institute — collaborators with UT San Antonio's $12.6 million project program from the National Cancer Institute to explore the biological mechanisms of BRCA1, BRCA2 and related tumor suppressors — will use their database of tissue samples from about 3,000 individuals who have ovarian cancer and many of whom became resistant to PARP-inhibitor treatment.

Molecular explanation for cancer-drug resistance

"This is a crucial first step to providing an intellectual framework to understanding why defects in this particular complex, and other proteins associated with this complex, can lead to cancer-drug resistance. We are well-poised to provide a molecular explanation, and this will also form the basis of finding compounds that interfere with protein function or enhance the stability of DNA-bound protein complexes to boost the therapeutic efficacy of PARP inhibitors and other cancer drugs. Imagine if we could create a way to have CST stay on DNA longer and make BRCA1-deficient tumor cells super sensitive to different therapeutic agents. It's a provocative concept," said Sung. ■

Sometimes it's something

By Claire Kowalick

Angel Inocencio never expected a routine dental cleaning to change his life. At 31, with no family history of cancer and no obvious symptoms, the last thing on his mind was a serious medical diagnosis. But thanks to his dentist's keen eye, he received the urgent care he needed in time.

During a routine appointment, Inocencio's dentist noticed a lesion on the far back of his tongue in an area that was difficult to see on his own. Though Inocencio said he never felt any pain, he later realized subtle signs had been present all along.

"When I would eat something, I was always having to clear my throat," Inocencio said. "Like every time for about 20 minutes after I ate anything. I was also having these stomach problems, but it never really dawned on me like, hey, this is not normal. I guess I just kind of adapted to that feeling."

Initially, Inocencio had dismissed the only visible sign — a small canker sore. So, when his dentist said it could be a sign of cancer and urged him to get the lesion checked out, the news came as a shock.

"To be told such drastic news, I was definitely blindsided and just very taken aback, considering that I am pretty young," he admitted. "But I was very glad that my dentist put that sense of urgency to get it looked at because it's super important."

An unexpected diagnosis

Inocencio recalls nervously waiting to be seen by a specialist after his dentist initially found the lesion.

"It was a very tough time for me," he said. "I didn't know what to think. Like, is it treatable? What if I do have it? What if I don't and I'm just psyching myself out? Am I terminal? What stage is it? Your mind just races through all kinds of things when you get told that you might have cancer."

When he was examined by a head and neck oncologist at Mays Cancer Center, Inocencio was diagnosed with Stage 2 oral cavity squamous cell carcinoma, a relatively rare occurrence for someone his age.

"Angel definitely doesn't fit the stereotype of a head and neck cancer patient," said Inocencio's surgeon, Jay Ferrell, MD, associate professor of head and neck surgery in the Department of Otolaryngology and director of the Division of Head and Neck Surgery. "But our understanding of who develops this type of cancer is starting to change."

"To be told such drastic news, I was definitely blindsided and just very taken aback, considering that I am pretty young."

Angel Inocencio, Otolaryngology patient at Mays Cancer Center

No prototypical patient

Head and neck cancers are the sixth most prevalent form of cancer in the United States, and squamous cell carcinoma accounts for over 90% of oral cavity malignancies.

Ferrell explained that, historically, head and neck cancers were viewed as a lifestyle disease. The condition has typically been linked to heavy tobacco use and drinking and is most often seen in men over the age of 55.

"It may not be the first thought in a healthy 30-something-year-old that a lesion in the mouth could be cancer. But we're actually seeing increased rates of oral cavity cancers even in nonsmokers and younger people, and we don't entirely know yet why that is," Ferrell said.

"It's important to recognize that there is no prototypical head and neck cancer patient. This is no longer just relegated to elderly people who smoke; it can happen in people in the prime of their lives who don't have those traditional risk factors."

What to watch

Oral cancers usually present as a sore or lesion on the tongue or other part of the oral cavity that doesn't heal and becomes painful as it grows deeper into the nerves, Ferrell explained. However, sometimes it can be as subtle as a discoloration of the tongue, like a raised white or red patch.

"Interestingly, Angel didn't experience much pain, which was overall good for him because this type of cancer is notoriously painful due to the tongue's rich nerve supply," Ferrell said. "So, if you notice discoloration or a sore that doesn't heal over a few weeks and keeps getting bigger, or there's a spot in your mouth that bleeds repeatedly when brushing, you should bring that to your physician's attention," he advised.

Surgery and reconstruction

Surgery is the mainstay of treatment for most oral cavity cancers. Inocencio underwent what is known as a partial glossectomy, a removal of the tumor with a sizeable resection from his tongue. During the procedure, Ferrell also removed several lymph nodes from Angel's neck, which were clear of cancer.

For reconstruction, skin and fascial tissue from the undersurface of Inocencio's forearm, along with the tissue's associated vascular supply, were transplanted and grafted to replace the removed section of his tongue. Forearm skin was chosen because its thin and pliable texture closely resembles that of the tongue, explained Ferrell.

Next, a skin graft from Inocencio's thigh was used to repair the forearm.

As with any cancer, early detection is key, said Ferrell. He notes that treating an early-stage cancer like Angel's that hasn't moved into lymph nodes or into other parts of the body provides about a 50% higher survival rate at two to five years after treatment compared to someone who finds the cancer later.

Advocating for awareness

Four months after surgery, Angel was back at work and grateful for a return to normal life. The likelihood of his cancer returning is now very low.

"Even in Angel's case, which thankfully has a very favorable prognosis, we monitor him closely with in-person head and neck examinations and imaging every three months," said Ferrell.

While head and neck cancer is notorious for recurrence and can be challenging to treat long-term, Ferrell said Inocencio's risk of recurrence is relatively low compared to patients diagnosed at more advanced stages with concurrent spread of the cancer to lymph nodes in the neck.

"With these more advanced cases, it is still possible to effect a durable cure, but it often requires more aggressive treatment, including radiation and chemotherapy after major surgical resections," Ferrell said.

Inocencio now advocates for regular dental visits and encourages others not to ignore unusual symptoms, no matter how minor they may seem.

"Don't be afraid to get seen by a professional," Inocencio urged. "Let them do their job, and they'll tell you if something is unusual. Be on top of your oral health and get it checked out because you just never know. It's worth the little bit of stress of going to the dentist. I'd rather know than not know because they said that it could have been there for about a year, and I had no idea. It's not always nothing, right? Sometimes it's something." ■

"With these more advanced cases, it is still possible to effect a durable cure, but it often requires more aggressive treatment, including radiation and chemotherapy after major surgical resections."

Jay Farrell, MD, associate professor of head and neck surgery in the Department of Otolaryngology and director of the Division of Head and Neck Surgery at UT San Antonio

Siblings race to honor everyday cancer heroes — and each other

By Susan Anasagasti

For Kathy Lozano and her brother, Larry Young, taking part in the 2025 Mays Cancer Center's Give Cancer the Boot Survivorship 5K race and 1-mile walk was a celebration of their remission.

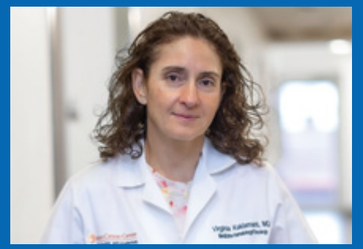
The event, dedicated to honoring survivors and supporting those still in treatment, drew more than 1,300 participants and raised more than $80,000 to support the center's Patient and Family Assistance Fund — covering practical needs, including transportation to appointments temporary lodging assistance for eligible patients.

In 2022, cancer had upended the lives of both Lozano and Young, and behind their finish-line smiles were months of setbacks and uncertainty.

Young's diagnosis came first: throat cancer. The treatment was grueling — 35 rounds of radiation, six rounds of chemotherapy and a feeding tube that remained in place for nearly two years. Lozano became a steady presence throughout her brother's treatments, but just as he began to recover, Lozano got her own cancer diagnosis — an aggressive lymphoma, which required swift, intensive treatment, including infusions, a port and a bone marrow biopsy followed by radiation.

"To be here, to be joyful, to be with my family and to be able to encourage others who are still going through it — that means everything," Lozano said of the event.

"This event is a powerful reflection of what makes this community so special," said Virginia Kaklamani, MD, leader of the cancer center's breast oncology program. "It's families supporting one another, volunteers giving their time and friends and community sponsors coming together with one shared purpose — to uplift and care for those affected by cancer." ■



"This event is a powerful reflection of what makes this community so special. It's families supporting one another, volunteers giving their time and friends and community sponsors coming together with one shared purpose — to uplift and care for those affected by cancer."

Virginia Kaklamani, MD, professor of medicine in the division of hematology/oncology and is the leader of the Breast Cancer Program at Mays Cancer Center.

Larry Young and his Sister Kathy Lozano, at the 2025 Mays Cancer Center's Give Cancer the Boot Survivorship 5K race and 1-mile walk

GIVE CANCER THE BOOT

CPRIT awards UT Health San Antonio $6.6 million to accelerate cancer research

By Jane Alvarez-Hernandez

The state agency, Cancer Research Prevention Institute of Texas (CPRIT), which focuses on the fight against cancer, has now awarded $167 million to UT Health San Antonio since 2010.

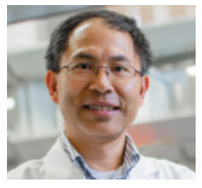
UNDERSTANDING OBESITY'S ROLE IN INCREASING LIVER CANCER RISK

Masahiro Morita, PhD, associate professor in the Department of Molecular Medicine and a researcher with the Sam and Ann Barshop Institute for Longevity and Aging Studies at UT Health San Antonio, received a $900,000 individual investigator award from CPRIT to study how obesity increases the risk of liver cancer by investigating the mechanisms by which excess nutrients lead to fatty liver and subsequently liver cancer.

"This understanding could lead to novel strategies for early detection, prevention and treatment of liver cancer, ultimately reducing the burden of obesity-associated cancer and improving patient outcomes," Morita said.

The team, including co-investigators Francisco Cigarroa, MD, senior executive vice president for health affairs and health system at UT Health San Antonio, Blake Rasmussen, PhD, professor and chair in cellular and integrative physiology, who is affiliated with the Barshop Institute, Madesh Muniswamy, PhD, tenured professor of medicine in the Division of Cardiology, and Sakie Katsumura, DDS, PhD, assistant professor of molecular medicine, believe the findings will be particularly significant in regions like South Texas, where the incidence of liver cancer among obese individuals is notably high and could provide a foundation for targeted therapeutic interventions. ■

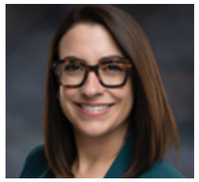
ADVANCING THE FIGHT AGAINST LEUKEMIA



A $900,000 individual investigator award from CPRIT to **Mingjiang Xu**, PhD, professor in the Department of Molecular Medicine in the university's Joe R. and Teresa Lozano Long School of Medicine, and distinguished chair in oncology at Mays Cancer Center, will support his research into identifying the role of TET2 gene mutations to develop future therapies targeting TET2-mutated myeloid malignancies.

"Along our investigation, we will identify potential specific therapeutic targets for the eradication of TET2-mutated cells," Xu said. "Success of the proposed studies will fill a critical knowledge gap on how TET2 mutation leads to adult myeloid malignancies and clonal hematopoiesis and lay a solid foundation for future precision therapy developments targeting TET2-mutated myeloid malignancies and normal-aged individuals." ■

COMMUNITY-BASED INTERVENTION TO INCREASE HPV VACCINATION RATES

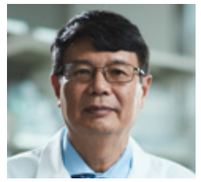


Erika L. Thompson, PhD, MPH, associate professor in the Department of Quantitative and Qualitative Health Sciences at the university's Kate Marmion School of Public Health received a CPRIT prevention grant of $975,000 to fund a community-based intervention program to increase vaccination rates for the human papillomavirus (HPV) among adolescents in Bexar County. HPV can lead to several different types of cancers in men and women. Therefore, improving vaccination rates is an effective way to reduce cancer risk and help prevent HPV-related cancers.

The project will integrate and adapt the All for Them vaccination program, an evidence-based, bilingual initiative run by Paula Cuccaro, PhD, at UTHealth Houston School of Public Health for the delivery and coordination of the social marketing campaign. The university's School of Nursing will deliver the HPV vaccines. The team will collaborate with community organizations and community health workers who work closely with underserved communities to administer community-wide vaccination services, including in after-school programs.

"The project's focus on reaching underserved populations ensures that it addresses the most pressing barriers to vaccination," Thompson said. "By increasing community demand for the HPV vaccine, improving access to vaccination services, and building trust in vaccine safety and efficacy, this intervention aims to advance efforts to reduce HPV-related cancer incidences and mortality in Texas." ■

DEVELOPING TECHNOLOGIES THAT TARGET DRUG-RESISTANT CANCERS



Daohong Zhou, MD, tenured professor in the Department of Biochemistry and Structural Biology in the Long School of Medicine, received CPRIT's academic research award of over $2.4 million to expand UT Health San Antonio's core facilities laboratories to increase researchers' capabilities to better identify therapeutic targets and develop technologies to target hard-to-treat cancers.

Zhou, who serves as the director of the Target Discovery Core at the Greehey Children's Cancer Research Institute at UT Health San Antonio, associate director for drug development at the Mays Cancer Center and director of the institution's Center for Innovative Drug Discovery, shared that while there has been significant progress in treating cancers, we are still facing big challenges, especially with certain tough-to-treat pediatric and adult cancers like brain tumors, soft tissue sarcomas and those that do not respond well to current therapies.

"However, the absence of core facilities for target identification and validation (TIV) in Texas has limited the success in discovering and developing new cancer therapeutics because TIV is essential for drug discovery and development," Zhou said. "In addition, the CPRIT award will allow us to acquire and develop new TIV technologies, including the state-of-the-art arrayed CRISPR knockout screening for TIV and groundbreaking small molecule degrader (SMD) discovery platform for screening SMDs to target undruggable proteins, which are not available in any other core facilities in Texas." ■

Robert A. Hromas, MD, FACP, dean of the Long School of Medicine at UT San Antonio

"Advancing research to find cures for cancers, particularly those that disproportionately affect the populations of San Antonio and South Texas, is not only our mission, it is the passion that fuels our daily work to make lives better."

Research impact in South Texas and beyond

By Jane Alvarez-Hernandez

"Our researchers receiving highly competitive awards, such as CPRIT, to improve the lives of Texans, is a testament to the excellence of UT Health San Antonio's research enterprise and demonstrates the extraordinary science conducted here," said Jennifer Sharpe Potter, PhD, MPH, senior executive vice president for research and innovation. "CPRIT is the largest state cancer research investment in the United States and the second largest cancer research and prevention program in the world. This recognition spotlights UT Health San Antonio's ability to attract top-tier talent, which drives pioneering discoveries and advances health innovation on an international scale."

"Advancing research to find cures for cancers, particularly those that disproportionately affect the populations of San Antonio and South Texas, is not only our mission, it is the passion that fuels our daily work to make lives better," said Robert A. Hromas, MD, FACP, dean of the Long School of Medicine.

"I am excited that CPRIT continues to invest in cancer research in South Texas. This investment will allow us to continue to build on the Mays Cancer Center's legacy of excellence and to explore new approaches in cancer care that will benefit San Antonio and beyond," said Lei Zheng, MD, PhD, executive director of the Mays Cancer Center and a world-renowned expert in pancreatic cancer.

Manzoor Bhat, PhD, MS, vice dean for research and distinguished chair in neurosciences at the university's Long School of Medicine, added, "Our investigators are making groundbreaking discoveries that extend beyond South Texas and have a global impact on advancing scientific knowledge, which will lead to developing novel solutions for human diseases that currently have no cures. These new CPRIT awards will undoubtedly advance cancer therapeutics leading to cures in the future." ■

Jennifer Sharpe Potter, PhD, MPH, senior executive vice president for research and innovation

"Our researchers receiving highly competitive awards, such as CPRIT, to improve the lives of Texans, is a testament to the excellence of UT Health San Antonio's research enterprise and demonstrates the extraordinary science conducted here."

Hyundai Hope on Wheels® awards $100K to continue its fight against childhood cancer

By Eileen Teves

In the United States, 43 children are diagnosed with cancer every day. Pediatric cancer is the leading cause of death by disease for children under the age of 14. Worldwide, about 400,000 children and adolescents develop cancer each year, only half of whom are diagnosed.

Since 1998, Hyundai Hope on Wheels® has been on a quest to end childhood cancer and has become one of the nation's leading funders of pediatric cancer research.

To help in the fight against childhood cancer, Hyundai Hope on Wheels® presented the Hyundai Impact Grant Award for $100,000 to three UT Health San Antonio recipients: Gail Tomlinson, MD, PhD, professor of pediatrics and division chief of pediatric hematology-oncology in the Department of Pediatrics; Shafqat Shah, MD, professor of pediatric hematology-oncology with expertise in neuro-oncology and cancer survivorship; and Debra Kent, DNP, MSN, APRN-PC, RN, associate professor, pediatric nurse practitioner and director of the adult survivorship program at Mays Cancer Center.

The award supports their nutrition education program called Hope in Every Bite. The program aims to improve the lives of patients and their families by highlighting the role of nutrition in prevention, treatment and survivorship. The support of Hyundai Hope on Wheels® helps the team reach their goal of educating and promoting healthy habits and improve the overall outcomes of pediatric cancer survivors.

With the latest grants, UT Health San Antonio has received more than $2.1 million in total grants since 2007 from Hyundai Hope on Wheels®. ■

Gail Tomlinson, MD, PhD, professor of pediatrics and division chief of pediatric hematology-oncology in the Department of Pediatrics at UT San Antonio

Shafqat Shah, MD, professor of pediatric hematology-oncology with expertise in neuro-oncology and cancer survivorship at Mays Cancer Center

Debra Kent, DNP, MSN, APRN-PC, RN, associate professor, pediatric nurse practitioner and director of the adult survivorship program, Mays Cancer Center.

Kurmasheva awarded CURE foundation grant for childhood cancer research

By Claire Kowalick

Raushan Kurmasheva, PhD, assistant professor at the Greehey Children's Cancer Research Institute at UT Health San Antonio and Department of Molecular Medicine at The University of Texas at San Antonio, was awarded a $330,000 Translation to Cure (T2C) award from the CURE Childhood Cancer Foundation.

The foundation prioritizes research that is on the fast track to discovering new treatments and research into hard-to-treat childhood cancers. The two-year grant awarded to Kurmasheva's team will fund her research project titled "Advancing Innovative and Effective Therapies for Children with Malignant Rhabdoid Tumors."

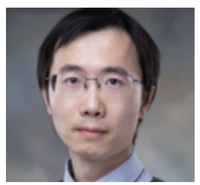
Malignant rhabdoid tumors, or MRT, are a rare, fast-growing cancer that is most common in infants and toddlers. This cancer often begins in the kidneys but can also occur in soft tissues and the brain. Five-year survival rates for this type of cancer are 20% to 25%. Current treatments for this cancer are surgery, aggressive chemotherapy, stem cell transplant and radiation therapy if the child is over six months old.

The project will explore a novel combination therapy using next-generation PARP1 inhibitors — a class of anti-cancer drugs — and DNA-damaging agents to target tumors.

Through this project, her team will establish preclinical data that will support future clinical trials with an ultimate goal of improving outcomes for children with aggressive malignancies.

"Malignant rhabdoid tumors mostly affect babies and toddlers under the age of 3," Kurmasheva said. "These very young patients have very few treatment options. With support from the CURE Childhood Cancer Foundation, we will develop therapies that not only shrink tumors but also avoid toxic side effects, ensuring treatments are safe and help preserve the children's long-term health. Our goal is to bring new hope to children and families facing this heartbreaking diagnosis." ■

Lung Cancer Research Foundation expands scientific advisory board



In August 2025, The Lung Cancer Research Foundation expanded its scientific advisory board by five new members and added its first cohort of five junior members, whose role is to review, score and provide commentary on grant applications as well as serve as volunteer spokespersons for the foundation and its research program.

Among the five new junior members is **Lingtao Jin,** PhD, associate professor in the Department of Molecular Medicine at UT Health San Antonio, the academic health center of The University of Texas at San Antonio.

"By welcoming junior members to our scientific advisory board, [the foundation] is investing in the future leaders of lung cancer research," said Dhruba Deb, PhD, the foundation's senior director of research programs. "These rising scientists will have the opportunity to learn directly from some of the most accomplished experts in the field, gaining insights and mentorship that will shape their careers. This is a critical step in ensuring that bold, innovative research continues to thrive, ultimately accelerating progress for people living with lung cancer."

Aubrey Rhodes, the foundation's executive director, said: "Our scientific advisory board is essential in identifying the best and brightest investigators whose creative ideas have the potential to uncover novel, innovative solutions for people living with lung cancer. The addition of these new members strengthens our ability to discover and support groundbreaking research that will truly make an impact." ■

Ramirez awarded Sedgwick Memorial Medal for Distinguished Service in Public Health

Amelie G. Ramirez, DrPH, MPH, has received the 2025 Sedgwick Memorial Medal for Distinguished Service in Public Health from the American Public Health Association. Ramirez is director of the Institute for Health Promotion Research, chair of the Department of Population Health Sciences and associate director of cancer outreach and engagement at Mays Cancer Center.

The award recognizes Ramirez's work in public health, which spans more than 30 years. An internationally recognized health researcher, Ramirez also launched the "Avanzando Salud Center," supported by a 4-year, $4.08-million grant from the American Cancer Society. The center is a response to the cancer burden facing South Texas. Center research scholars and the community are teaming up to address health across the cancer care continuum by targeting non-medical drivers of health that prevent people from obtaining care.

In addition, Ramirez is among a team of researchers at UT Health San Antonio selected to launch a community engagement resource center on heart health, thanks to a generous contribution from the American Heart Association and the Robert Wood Johnson Foundation. From a total gift of $20 million to support four teams of scientists and community leaders from across the United States to engage the community in research to improve health, UT Health San Antonio received $3.5 million for its CONNECTOR initiative. The initiative aims to identify, evaluate and manage community-based solutions to fight precursors of heart and vascular disease, including upstream factors such as nutrition insecurity and other related non-medical drivers of health. ■

Owens Medical Research Foundation awards investigators $750K to advance cancer research

By Jane Alvarez-Hernandez

In 2025, the William and Ella Owens Medical Research Foundation awarded $250K to three UT Health San Antonio research projects on pancreatic and childhood cancers.

DRUG TARGETING IN EARLY-STAGE MODELS OF PANCREATIC CANCER

INVESTIGATOR: Simon Gayther, PhD, professor in the Division of Hematology and Oncology, and founding director, Center for Inherited Oncogenesis. Co-PI: Patrick Sung, DPhil, professor in the Department of Biochemistry and Structural Biology and Robert A. Welch Distinguished Chair in Chemistry.

SYNOPSIS: Five-year survival rates for pancreatic ductal adenocarcinoma, PDAC, are the worst of all tumor types. Globally, the incidence of the disease is rising. In South Texas, PDAC represents a major cancer burden. During 2011-2015, PDAC incidence was 12.3 per 100,000, higher than the national average.

One main barrier to effective prevention and treatment is the difficulty in modeling PDAC. Its underlying pathology remains poorly understood at the mechanistic level.

In Aim 1, researchers will establish induced pluripotent stem cell (iPSC) models of normal pancreatic tissues with and without BRCA2 mutations, the strongest known genetic risk factor for PDAC. To these iPSC models, researchers will layer on additional mutations frequently occurring in PDAC to mimic both the early and later stages of PDAC to identify precision therapies that could improve outcomes.

In Aim 2, researchers will examine functional interactions between BRCA2 and mutated versions in the iPSC models. They will study how these interactions affect the DNA damage response, specifically DNA damage repair and replication fork preservation. The goal is to define how BRCA2 physically and functionally interacts with the co-occurring mutations to impair DNA damage repair and replication functions of BRCA2. ■

IN SOUTH TEXAS, PDAC REPRESENTS A MAJOR CANCER BURDEN. DURING 2011-2015, PDAC INCIDENCE WAS

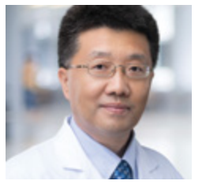
12.3

PER

100K,

HIGHER THAN THE NATIONAL AVERAGE.

THE ROLE OF THE WARBURG EFFECT ON CELLULAR AND SYSTEMIC ENERGY METABOLISM IN PDAC CACHEXIA



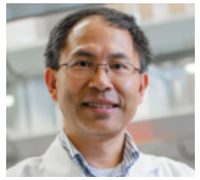
INVESTIGATOR: Gang Huang, PhD, professor in the Departments of Cell Systems and Anatomy and Pathology and Laboratory Medicine, and holder of the Kathryn Mays Johnson Distinguished Chair in Oncology; associated with the Mays Cancer Center.

SYNOPSIS: Pancreatic ductal adenocarcinoma, or PDAC, is one of the deadliest types of cancer and is often diagnosed in its later stages. A common and very serious problem that the majority of PDAC patients face is called cancer-associated cachexia, or CAC — a profound weight loss and muscle-wasting condition that not only weakens patients but also makes treatments less effective. CAC-induced organ failure is the primary cause of death in patients with advanced cancer, and unfortunately, there are no FDA-approved treatments for the condition.

This project investigates how the loss of a key regulator gene, called LKB1 or STK11, in PDAC cells leads to a chain reaction in the body's energy use, known as the Warburg effect. In healthy cells, LKB1 helps maintain balanced energy production by activating AMPK. When LKB1 is missing, cancer cells accelerate their sugar-burning processes while reducing fat-burning processes like fatty acid oxidation and other energy-generating pathways. This shift forces the tumor to use up large amounts of glucose, lowering blood sugar levels throughout the body. As a result, the patient's muscles and fat stores are broken down to compensate, contributing to the severe weight loss seen in cachexia. Eventually, this unintentional weight loss leads to weakness, fatigue, anorexia, pain, depression and systemic organ failure.

By pinpointing the exact molecular events that drive this imbalance in both cells and animal models, researchers aim to develop new therapies that block the tumor's hijacking of the body's energy stores. ■

DEVELOP PSPC1 DEGRADER FOR NOVEL PEDIATRIC AML THERAPEUTICS



INVESTIGATOR: Mingjiang Xu, PhD, professor in the Department of Molecular Medicine and CTRC Council Distinguished Chair in Oncology; associated with the Mays Cancer Center. Co-PI: Yaxia Yuan, PhD, assistant professor in the Department of Biochemistry and Structural Biology.

SYNOPSIS: The most common pediatric cancer is acute leukemia, of which acute myeloid leukemia, or AML, accounts for about 20% of cases. The current treatment approaches for pediatric AML primarily rely on intensive chemotherapy, followed by stem cell transplantation for high-risk or relapsing patients. While there have been some recent successes with genetically targeted therapies for adults, pediatric AML has been slower to advance in children due to its distinct genetic features. Therefore, it is crucial to discover common dependencies for both pediatric and adult AMLs, despite their diverse genetic drivers. Targeted therapies based on such common dependency could offer a "one-size-fits-all" therapy, especially pediatric AMLs with distinct genetic features.

Recent studies (published in Cell Stem Cell, 2025) have discovered paraspeckle protein component 1 (PSPC1) as a novel pan-AML target. PSPC1 is a protein implicated in the development and spread of multiple solid cancers. Preliminary screening has identified a few small molecules that can induce rapid PSPC1 degradation in AML cells. These compounds exhibit potent anti-leukemic activity, while having minimal effect on normal cells.

In this project, we will optimize our PSPC1 small molecule degraders in a timely and cost-effective manner. We will then perform validation of the best degrader by evaluating its specificity, anti-leukemic potency and toxicity. Our study could lead to a breakthrough in AML treatment by developing novel PSPC1 small molecule degraders as a safe and effective therapeutic for pan-AMLs, especially pediatric AMLs. The team aims to develop PSPC1 degraders, a small molecule that could have broad applications for cancer treatment far beyond pediatric and adult AMLs. ■

Promising cancer researcher receives UT System Rising STARs, Voelcker Fund Young Investigator awards

By Jane Alvarez-Hernandez

Maria E. Falzone, PhD, assistant professor in the Department of Biochemistry and Structural Biology in the Joe R. and Teresa Lozano Long School of Medicine, has received a $150,000 UT Science and Technology Acquisition and Retention (STARs) award from the University of Texas System to help advance research into the molecular mechanisms underlying signaling-dependent regulation of lipid-editing enzymes and their dysregulation in cancer.

Falzone, who joined UT Health San Antonio in the fall of 2024 as part of a $52 million-recruitment effort from the Cancer Prevention and Research Institute of Texas announced earlier that year, is cross-appointed as a full member of the Greehey Children's Cancer Research Institute and Mays Cancer Center.

"One of my main research goals is to contribute to an increased understanding of cellular signaling and its dysregulation in cancer. I hope our work can make a small contribution to something that will eventually help people in our community, whether it is through therapeutic development or an increased understanding of the underlying cellular processes," Falzone said. "Another is always to keep pushing the boundaries of what is experimentally possible. If we are going to understand new biological questions, we must keep developing new and creative approaches to push our research forward."

Separately, Falzone was among three UT Health San Antonio researchers to receive a 2025 Voelcker Fund Young Investigator Award from the Max and Minie Tomerlin Voelcker Fund supporting biomedical research from early-career investigators. Falzone's project is focused on identifying compounds that disrupt phospholipase D signaling and to assess the effects of disrupting these pathways on breast cancer proliferation and progression. ■

IN MAY 2024, THE CANCER PREVENTION AND RESEARCH INSTITUTE OF TEXAS (CPRIT) INITIATED A

$52M

RECRUITMENT INITIATIVE—TO STRENGTHEN THE STATE'S FIGHT AGAINST CANCER, WITH A SUBSTANTIAL SHARE DIRECTED TOWARD ATTRACTING LEADING RESEARCHERS TO TEXAS INSTITUTIONS LIKE UT HEALTH SAN ANTONIO

AS PEOPLE AGE AND HORMONE LEVELS DROP, SEX STEROID HORMONE RECEPTORS BECOME THE DRIVERS OF MORE THAN

40%

OF CANCERS

Wasmuth named Howard Hughes Medical Institute Freeman Hrabowski Scholar

By Claire Kolalick

Elizabeth Wasmuth, PhD, assistant professor in the Department of Biochemistry and Structural Biology at the Joe R. and Teresa Lozano Long School of Medicine and co-director of the cryo-EM facility at The University of Texas at San Antonio, has been selected by the Howard Hughes Medical Institute as a Freeman Hrabowski Scholar.

The five-year appointment provides up to $8.6 million over a 10-year period. The allotment includes salary, benefits, research budget and scientific equipment. Wasmuth was one of 30 scholars chosen from nearly 900 applicants. Along with achievements in biomedical research, Freeman Hrabowski Scholars are expected to make strides in becoming strong mentors and creating a supportive laboratory environment that provides a strong foundation for trainees.

Under this program, Wasmuth's lab will conduct research on sex steroid hormone receptors to develop more-effective treatments for prostate cancer and other health conditions related to these receptors.

"As people age and hormone levels drop, these receptors become the drivers of more than 40% of cancers. Since the 1940s, we have been drugging these receptors basically the same way, and there is inevitable resistance. Newer versions of these drugs work for a period of time, but there is resistance to these drugs as well. I'm taking a multidisciplinary approach, from the lens of a cancer biologist, but also the lens of the structural biologist, and integrating these fields to get answers about how these receptors are activated, turned off or kept quiescent," said Wasmuth.

Wasmuth joined the university in 2022, where her laboratory uses a multidisciplinary approach of reconstitution biochemistry, biophysics, proteomics and structural biology to investigate mechanisms of androgen-receptor function and regulation with the goal of discovering more-effective prostate cancer therapies.

Wasmuth's efforts, together with Shaun K. Olsen, PhD, professor in the Department of Biochemistry and Structural Biology and director of the structural biology cores, and Patrick Sung, DPhil, director of the Greehey Children's Cancer Research Institute, associate dean for research at the Long School of Medicine, professor of biochemistry and structural biology and Robert A. Welch Distinguished Chair in Chemistry, were pivotal in the acquisition and widespread use of cryo-EM microscopy technology, which has benefited many fields of research at the university. ■

Max and Minnie Tomerlin Voelcker Fund grants $1.25 million to support research from early-career investigators

By Jane Alvarez-Hernandez

UT Health San Antonio, the academic health center of The University of Texas at San Antonio, has been awarded $1.25 million by the Max and Minnie Tomerlin Voelcker Fund to support early-career investigators conducting biomedical research. The Voelcker Fund Young Investigator Awards will be allocated over three years.

ATPASE IN STRUCTURE-BASED DRUG DISCOVERY AGAINST HEART ATTACK EFFECTS

INVESTIGATOR: Oleg Klykov, PhD, assistant professor, Department of Biochemistry and Structural Biology

SYNOPSIS: Ischemia involves a lack of blood supply to an organ, such as during a heart attack. While restoring blood flow, known as reperfusion, is essential, it can also cause cell death and mitochondrial dysfunction, leading to ischemia-reperfusion injury. Mitochondria are critical for the production of adenosine triphosphate, or ATP, the primary energy currency of cells, and disruptions in their function can hinder recovery.

This project aims to explore mitochondrial dysfunction related to ATP synthase during myocardial ischemia and reperfusion using advanced biophysical and structural biology techniques. It will map mitochondrial membrane protein interactions and visualize changes in protein architecture under these conditions. The goal is to generate insights for developing innovative cardioprotective therapies. ■

FUNCTIONS OF SMALL MAF PROTEINS IN THERAPY-INDUCED SENESCENCE

INVESTIGATOR: Lu Wang, PhD, assistant professor, Department of Biochemistry and Structural Biology; member of the UT Health San Antonio Mays Cancer Center and the Sam and Ann Barshop for Longevity and Aging Studies

SYNOPSIS: Hepatocellular carcinoma accounts for 90% of liver cancer cases and is a leading cause of cancer-related death worldwide. Early-stage patients can benefit from surgical treatments, but many are diagnosed late and miss these options. Systemic chemotherapy can induce cellular senescence as a tumor-suppressing mechanism, but it may also promote tumor growth. This research focuses on small Maf proteins and their roles in therapy-induced senescence and evasion, potentially contributing to cancer recurrence. The ultimate goal is to identify potential therapeutic targets for enhancing patient outcomes. ■

San Antonio Firefighters Cancer Prevention Program launches at UT Health San Antonio

By Steven Lee

Firefighting is classified as a known human carcinogen by the International Agency for Research on Cancer. Occupational cancer has become the leading cause of death among firefighters. Studies have shown that firefighters face a 9% higher risk of being diagnosed with cancer and a 14% higher risk of dying from cancer compared to the general U.S. population.

And cancer is a big concern for firefighters across San Antonio. Within the San Antonio Fire Department (SAFD), more than 70 firefighters have been diagnosed with cancer in the past 10 years — from leukemia to multiple myeloma and cancers of the brain, thyroid, colon, prostate and testicles. Many cases also go unreported.

To help reduce the cancer risk among firefighters and emergency medical services personnel, SAFD, UT Health San Antonio and Sylvester's Firefighter Cancer Initiative at the University of Miami have partnered to launch the San Antonio Firefighters Cancer Prevention Program. The program aims to better understand and reduce the burden of cancer among local first responders.

SAFD, which includes more than 1,800 fire and emergency personnel serving a population of over 1.4 million residents, created its Occupational Cancer Committee to address the issue. In 2024, the committee began working with UT Health San Antonio's Institute for Health Promotion Research and its Mays Cancer Center's community outreach and engagement team. Together, they also partnered with Sylvester's Firefighter Cancer Initiative, which began in 2015. Now, the San Antonio Firefighters Cancer Prevention Program has started work.

"Our UT Health San Antonio team is already connecting with firefighters by sharing educational resources at SAFD health fairs," said Amelie G. Ramirez, DrPH, MPH, director of the Institute for Health Promotion Research and associate director of community outreach and engagement at Mays Cancer Center. The program has also initiated an immediate response system with the Mays Cancer Center to provide cancer care referrals to firefighters who receive a cancer diagnosis.

"Our goal is to strengthen cancer prevention, education and expert care for the incredible firefighters and emergency responders who dedicate their lives to protecting our community," Ramirez said.

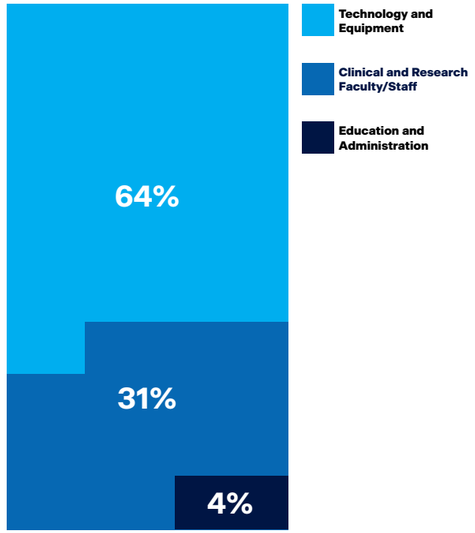
Among the activities being explored through the program:

- Launching an annual cancer risk assessment study based on the national firefighter annual cancer cohort study of Sylvester's Comprehensive Cancer Center.
- Tailoring Miami's Firefighter Cancer Initiative educational resources for San Antonio firefighters and hosting educational sessions with the SAFD.
- Developing an additional referral and navigation system to connect SAFD firefighters to UT Health San Antonio and Mays Cancer Center providers for early cancer screening, diagnosis and survivorship programs.
- Connecting SAFD firefighters to cancer clinical trials and research studies at Mays Cancer Center.
- Identifying opportunities to advise the National Institute for Occupational Safety and Health (NIOSH) and other relevant stakeholders on a strategy to collect Firefighter Cancer Registry data.
- Collaborating with SAFD leaders to train UT Health San Antonio primary care providers on the unique screening needs and potential occupational or environmental exposures relevant to firefighters or emergency service personnel.

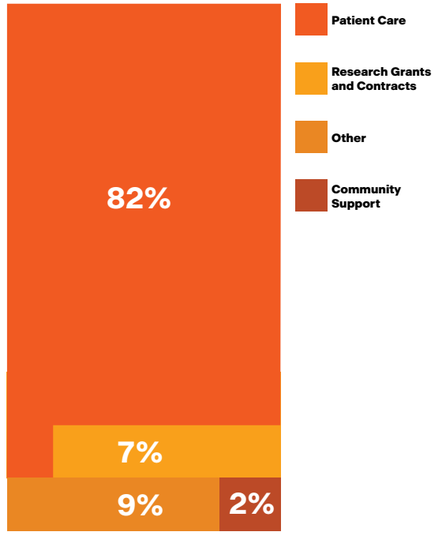
Ramirez said collaborators are unified in the goal to boost the well-being of local safety personnel.

"Firefighters dedicate their service and their lives to protect us. We should work just as hard to reduce and prevent cancer within the firefighter community," Ramirez said. ■

USE OF FUNDS BY TYPE



FUNDING SOURCE



144,214
TOTAL PATIENT ENCOUNTERS

4,917
NEWLY DIAGNOSED PATIENTS

2,918
CANCER SURGERIES

11%
INCREASE IN OVERALL FUNDING
and a 10% increase in peer-reviewed funding from the National Cancer Institute

325
ACTIVE RESEARCH PROJECTS,
including 184 peer-reviewed grants and 141 non-peer-reviewed grants and contracts

183+
CLINICAL STUDIES
Prevention, treatment and survivorship clinical studies, including 54 new clinical research studies opened in FY25

IN FY25, MAYS CANCER CENTER GENERATED AN ESTIMATED

$160M

ECONOMIC IMPACT ACROSS SAN ANTONIO AND THE BROADER SOUTH TEXAS REGION

MAYS CANCER CENTER **SENIOR LEADERSHIP**

Lei Zheng, MD, PhD
Executive Director, Mays Cancer Center
Vice President for Oncology, UT Health San Antonio

Patrick Sung, DPhil
Director, Greehey Children's Cancer Research Institute

Tim H.M. Huang, PhD
Deputy Director, Mays Cancer Center
Associate Director, Basic Science

Nicholas DeLapo, MPH
Chief Operating Officer, Mays Cancer Center
Associate Vice President for Oncology, UT Health San Antonio

Richard Tuli, MD, PhD
Oncology Service Line Director, Mays Cancer Center
Associate Chief Medical Officer, UT Health
San Antonio Multispecialty and Research Hospital

Gwen Tate, MBA, MSN, RN, NEA-BC
Sr. Director of Nursing

Travis Corwin, MBA
Sr. Administrator, Finance and Administration,
Radiation Oncology

Amelie Ramirez, DrPH
Associate Director, Community Outreach and Engagement

Susan Padalecki, PhD
Associate Director, Research Administration

David Gius, MD, PhD
Associate Director, Translational Research

Daruka Mahadevan, MD, PhD
Associate Director, Clinical Research

Michael Halpern, MD, PhD, MPH
Associate Director, Population Science

Robin Leach, PhD
Associate Director, Education

Teresa Johnson-Pais, PhD
Assistant Director, Education

Luzhe Sun, PhD
Associate Director, Shared Resources

Gang Huang, PhD
Assistant Director, Shared Resources

Daohong Zhou, MD
Associate Director, Drug Development

Daniel Dent, MD
Associate Director, Workforce Development

Angel Gonzalez, PhD
Assistant Director, Workforce Development

Anand Karnad, MD
Chief, Division of Hematology and Medical Oncology

Elizabeth Bowhay, MD
Vice Chief, Hematology

Sukeshi Arora, MD
Vice Chief, Medical Oncology

Danielle Fritze, MD
Interim Chief, Division of Surgical Oncology
and Endocrine Surgery

SCIENTIFIC **PROGRAM LEADERS**

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Co-leader, Cancer Development and Progression

Sandeep Burma, PhD
Co-leader, Cancer Development and Progression

Patricia Dahia, MD, PhD
Co-leader, Cancer Development and Progression

Andrew Brenner, MD, PhD
Co-leader, Experimental and Developmental Therapeutics

Manjeet Rao, PhD
Co-leader, Experimental and Developmental Therapeutics

Gail Tomlinson, MD, PhD
Co-leader, Population Science and Prevention

Simon Gayther, PhD
Co-leader, Population Science and Prevention

CLINICAL AND RESEARCH **OPERATIONS**

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Chief Executive Officer,
UT Health San Antonio
Multispecialty and Research Hospital

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Chief Medical Officer,
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Director, Clinical Trials Office

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Nursing Director of Operations, Infusion
and Radiation Oncology

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Lydia Herrera, PharmD, MBA, BCPPS
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Roseanna Martinez, MPH
Director, Practice Operations, Patient Access

Melissa Nashawati, MPA
Director, Quality Assurance and Training

Jeanie Paradise, MA, LPC-S
Director, Clinical Operations, Social Services

Lixin Song, PhD, RN, FAAN
Director, Caregiver Research

Elizabeth Thompson, MLS
Director, Laboratory Operations

SHARED **RESOURCES**

BIOSTATISTICS AND BIOINFORMATICS

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Co-director

Jonathan Gelfond, MD, PhD
Co-director

DRUG DISCOVERY AND STRUCTURAL BIOLOGY

Sean Olsen, PhD
Co-director,
Structural Biology

Daohong Zhou, MD
Co-director, Drug Discovery

FLOW CYTOMETRY

Michael Berton, PhD
Scientific Director

Yue Li, PhD
Director

NEXT-GENERATION SEQUENCING

Zhao Lai, PhD
Director

OPTICAL IMAGING

James Lechleiter, PhD
Director

Exing Wang, PhD
Co-director

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Andrew Brenner, MD, PhD
Neuro-Oncology

Adolfo Enrique Diaz Duque, MD
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Virginia Kaklamani, MD, DSc
Breast Oncology

Usha Perepu, MD
Classical Hematology and Blood Disorders

Debra Kent, DNP
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Daruka Mahadevan, MD, PhD
Phase I Oncology
Director, Institute for Drug Development

Georgia McCann, MD
Gynecologic Oncology

Prince Otchere, MD
Cardio-Oncology

Montaser Shaheen, MD
Melanoma and Immunotherapy

Deepak Pruthi, MD
Urologic Oncology

Gail Tomlinson, MD, PhD
Pediatric Hematology Oncology

DEPARTMENT CHAIRS/ **DIVISION CHIEFS**

Richard Tuli, MD
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Lacy Harville
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Cardiothoracic Surgery

Daniel DeArmond, MD
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John Floyd, II, MD
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Russell Higgins, MD
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Georgia McCann, MD
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Ronald Stewart, MD
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Anton Fries, MD, PhD
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structive Surgery

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NEW MAYS CANCER CENTER **MEMBERS**

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Syed Usama, BS, PhD
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Lei Zheng, MD, PhD
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Experimental and Developmental Therapeutics Program

MAYS CANCER CENTER ANNUAL REPORT 2025

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San Antonio
Mays Cancer Center

THE UNIVERSITY OF TEXAS AT SAN ANTONIO