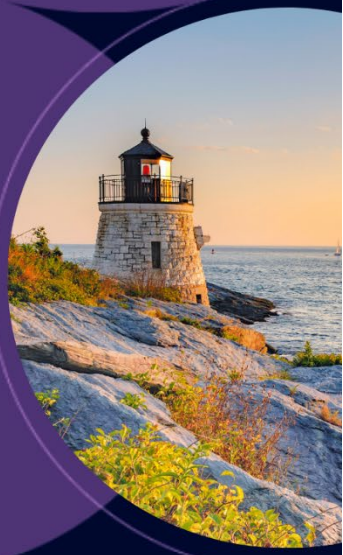
WIN Symposium 2026

PRECISION ONCOLOGY: INNOVATION AND EQUITY ACROSS THE GLOBE

OCTOBER 2 - 3 2026

RHODE ISLAND CONVENTION CENTER, PROVIDENCE, USA



WIN Symposium 2026

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KNOWLEDGE CONQUERS CANCER

WIN SYMPOSIUM 2026
Precision Oncology: Innovation and Equity Across the Globe

GENERAL INFORMATION AND PROGRAM

WIN Symposium 2026 is offered by the WIN Consortium

24, rue Albert Thuret, 94550 Chevilly-Larue, France
www.winconsortium.org

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WIN SYMPOSIUM 2026 – Chairs

Prof. Wafik S. El-Deiry, MD, PhD, FACP

Chair, Worldwide Innovative Network (WIN) Consortium for Personalized Cancer Medicine
American Cancer Society Professor
Mencoff Family University Professor of Medical Science
Professor of Pathology & Laboratory Medicine
Brown University
Attending Physician, Hematology/Oncology,
Brown University Health, USA

Prof. Razelle Kurzrock, MD, FACP

Chief Medical Officer, Worldwide Innovative Network (WIN) Consortium for Personalized Cancer Medicine
Professor of Medicine,
Director, Center for Precision Oncology and Rare Cancers
Linda T. and John A. Mellowes Endowed Chair of Precision Oncology
MCW Cancer Center and Linda T. And John A. Mellowes Center for Genomic Sciences and Precision Medicine
Founding Director
Michels Rare Cancers Research Laboratories
Froedtert and Medical College of Wisconsin, USA

WIN SYMPOSIUM 2026 – Welcome Messages

Welcome to Rhode Island

One of 13 original states, based on RI Freedom. Due to our unique size, we have one of the highest rates of Cancer per capita in the country.
Health Care has dominated our Legislative session for over a decade. We have started a process to form our own State Medical School in RI to compliment Brown Medical School and not compete.

Fall is the best season in RI, please take a break and take in some of our great natural sights and foliage.

Enjoy the best of RI on your visit here.

The Honorable K. Joseph Shekarchi
224th Speaker Emeritus of the Rhode Island House of Representatives, USA

WIN SYMPOSIUM 2026 – Welcome Messages

Welcome to 2026 WIN Symposium "Precision Oncology: Innovation and Equity Across the Globe" in Providence, Rhode Island, from October 2 – 3, 2026.

Dear Colleagues:

It is the best of times and the worst of times!

Precision Oncology is evolving rapidly. As a global community, we must address issues of access to molecular testing and therapeutics. We live in a time with expanding knowledge of the tumor microenvironment at the single cell level, drug mechanisms and resistance, novel AI algorithms and advances including daraxonrasib celebrated for addressing a target deemed undruggable for over 40 years. This drug costs nearly $40K per month and nearly $0.5 million per year while extending median overall survival to 13.7 months as monotherapy. This is very important progress but by no means a cure. Cooperation between stakeholders could improve this for both efficacy and access. Cooperation in prospective clinical trials will add important evidence with advanced drug combinations tailored precisely for individual patients. Demonstrating value and precision to treat the right patients with the right combinations at the right time will help people be healthier while saving money and unnecessary toxicities. WIN also has a focus on precision prevention as an emerging field.

The WIN Consortium was founded in 2010 by the late John Mendelsohn at MDACC and the late Thomas Tursz at Gustave Roussy with the premise that we can all do more working together through innovation and collaboration for global impact. Since then, WIN and its leaders have led the field of personalized cancer medicine through evidence generation for combinations of targeted and immunotherapeutics, as well as moving beyond genomics into multi-omics (transcriptomics and beyond) through trials such as WINTHER and iPREDICT published in Nature Medicine. The evidence has continued to show an overall survival benefit associated with a higher matching score for biomarker-matched therapy combinations.

In recent years, WIN has regularly convened an active truly global molecular tumor board that has been bringing the most up-to-date available prioritized options for individual patients for their physicians to consider. With establishment of WIN Consortium Disease Committees in the last couple of years, the WIN Precision Oncology Fellowship in 2026, educational activities through conferences and publications, collaborative outreach, WIN is continuing to lead and champion the future vision for oncology with precision.

The 2026 WIN Symposium has shaped up as one of the finest to date among WIN Symposia and other international venues in Precision Oncology with a record-breaking number of abstracts and a world-class line-up of amazing and inspiring colleagues. We invite you to learn from the presentations, both oral and poster, stop by exhibits, and take advantage of the opportunity to network for future impact. The sessions include Nobel Laureate Presentation, Keynote Presentations, Patient Perspective, N-of-1 trials, Fireside Chat on Regulatory Innovation for the future of Precision Oncology, AI, Industry Perspectives, Tumor Agnostic Oncology, Global Issues and Collaboration, Closing Access Gap, Screening, ctDNA, MTB,

WIN SYMPOSIUM 2026 – Welcome Messages

Microbiome, Immunotherapy, Germline Testing, Careers, Panel Discussions, WIN Fellows, and Poster Session.

Learn about Precision Oncology, appreciate the discipline and the practice as the future of oncology, and enjoy the beauty, splendor, culture, and cuisine of the Ocean State, Rhode Island. Network with leaders in Precision Oncology from around the world. Innovate and collaborate for global impact. For the 2026 WIN Symposium, registration fees were waived for all college students, medical students, graduate students, post-doctoral, and clinical trainees including fellows in the allied health professions.

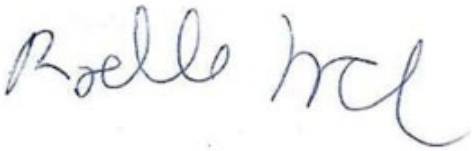
We welcome you to Providence and wish you a productive and enriching experience at the 2026 WIN Symposium.

Wafik S. El-Deiry, MD, PhD, FACP, Chair, WIN Consortium
2026 WIN Symposium Co-Chairs

WIN SYMPOSIUM 2026 – Welcome Messages

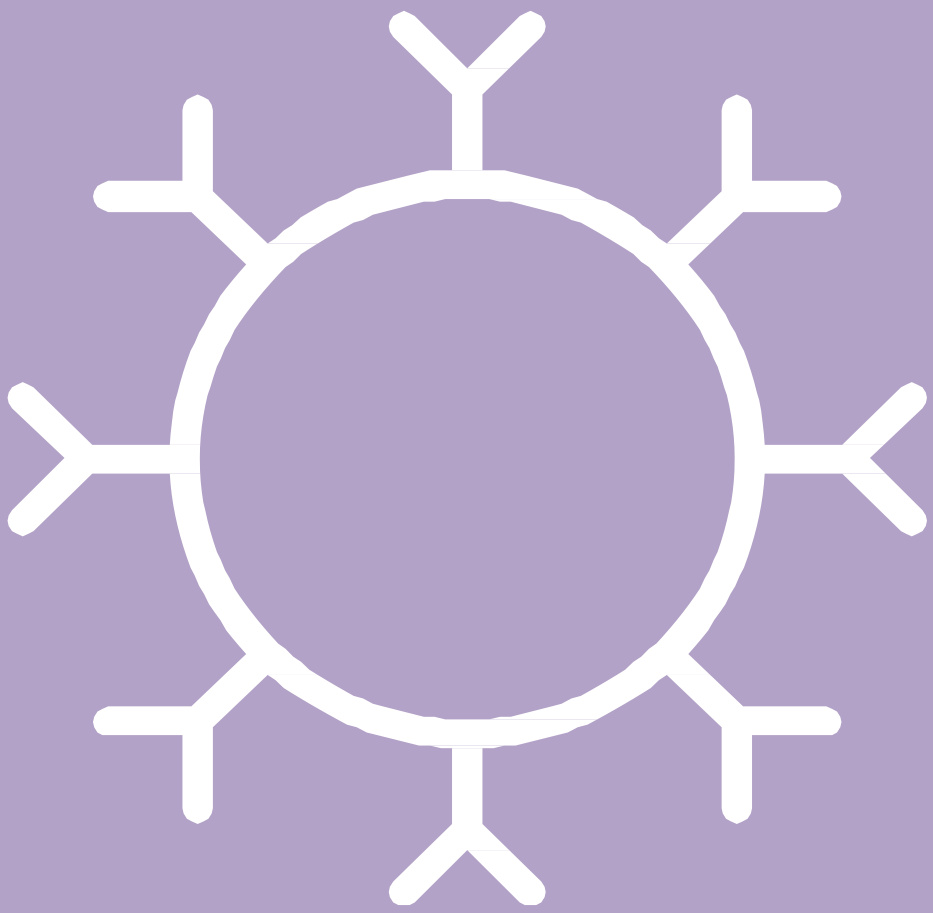
Welcome to the 2026 WIN Symposium

We are privileged to bring together global stakeholders and some of the foremost figures in oncology, united by a commitment to transforming cancer care. Precision Oncology is redefining cancer through molecular and immune reclassification, moving us beyond one-size-fits-all approaches toward truly individualized treatment. Over these two days, we hope bold science, diverse perspectives, and global collaboration will accelerate innovation and help revolutionize cancer care for patients everywhere.



Razelle Kurzrock, MD, CMO, WIN Consortium
2026 WIN Symposium Co-Chairs

WIN CONSORTIUM



WIN CONSORTIUM – About WIN Consortium

The Worldwide Innovative Networking (WIN) Consortium in personalized cancer medicine, a non-profit, non-governmental organization, headquartered in Paris, France was founded by the late Dr. John Mendelson, past Chairman, WIN Consortium, past President, The University of Texas, MD Anderson Cancer Center, Houston, USA and the late Professor Thomas Tursz, past Vice Chairman, WIN Consortium and General Director of Gustave Roussy, France.
Initiated in 2010 with leadership from leading cancer centers, WIN was formed on the premise that we can accomplish more together than each organization can achieve working alone. Our shared vision is delivering the promise of effective personalized cancer medicine to patients worldwide, to improve cancer patients' survival and quality of life.

WIN now includes 38 institutional members: 27 world-class academic medical centers, 8 research organizations/societies/patient advocates and 3 companies.
These stakeholders have come together covering 18 countries and 6 continents to address the challenge of increasing the efficacy of cancer diagnostics and therapeutics by understanding the genetics and biology of each individual's tumor and accounting for genetic differences across diverse populations—from North and South America, Europe, Asia, Middle East and Oceania.

WIN CONSORTIUM – WIN Members

Academic Institutions in Americas

Academic Institutions in Asia-Pacific

Academic Institutions in Middle East

WIN CONSORTIUM – WIN Members

Academic Institutions in Europe

Non-Profit Organizations

Industries

WIN CONSORTIUM – WIN Executive Committee

WIN Executive Committee

Prof. Wafik S. El-Deiry
Chair, WIN Consortium
Brown University Health
(USA)

Dr. Richard L. Schilsky
Chair Emeritus, WIN Consortium
Professor Emeritus,
University of Chicago
Past President & Chief Medical Officer, American Society of Clinical Oncology (ASCO)
(USA)

Prof. Razelle Kurzrock
Chief Medical Officer, WIN Consortium
Prof. of Medicine,
Director, Center for Precision Oncology and Rare Cancers,
MCW Cancer Center and Linda T. & John A. Mellowes Chair of Precision Oncology
(USA)

Dr. Sewanti Limaye
Vice-Chair, WIN Consortium
Director Medical & Precision Oncology, Director Clinical and Translational Oncology Research,
Sir HN Reliance Foundation Hospital and Research Centre
(India)

Prof. Marina Sekacheva
Chair, Membership Committee, WIN Consortium
Director of Institute for Personalized Oncology, I.M Sechenov First Medical State University,
(Russian Federation)

Prof. Apostolia M. Tsimberidou
Vice-Chair, WIN Consortium
Professor of Medicine in the Department of Investigational Cancer Therapeutics and a Katherine Russell Dixie Distinguished Endowed Professor at The University of Texas,
MD Anderson Cancer Center
(USA)

Dr. Jacques Raynaud
Senior Advisor,
WIN Consortium
(France)

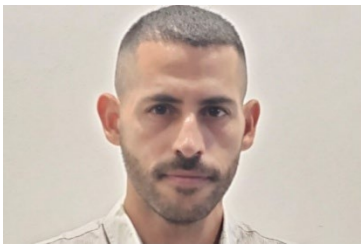
Dr. Fanny Wunder
Interim Chief Operating Officer
WIN Consortium
(France)

WIN CONSORTIUM – WIN Operational Team

WIN Operational Team

Fanny Wunder, PhD
Interim Chief Operating Officer

Layla El-Gharras
Operations and Administrative Manager



Shai Magidi
Clinical Data Manager & Research Data Analytics Specialist

Eitan Rubin
Head of Bioinformatics

WIN CONSORTIUM – WIN's Goals and Value

WIN's Mission

Significantly improve survival of cancer patients across the world through international collaboration.

WIN's Strategy

Advance Precision Oncology and early cancer diagnosis by developing and evaluating innovative strategies and tools through international collaborative research, including proof-of-concept clinical trials.

WIN's Value

Access to:

- Global thought leaders in Precision Oncology
- Platform of leading cancer centers across the world: rapid accrual/diversity of population

Expertise in genomics and transcriptomics

Experience and success with:

- Complex clinical trials (WINTHER and SPRING trials)
- Obtaining high-quality tissues and bloods for comprehensive omics
- Managing international multi-site trials
- International Molecular Tumor Board

Ability to partner with companies and philanthropic organizations or individuals to perform Precision Oncology studies to:

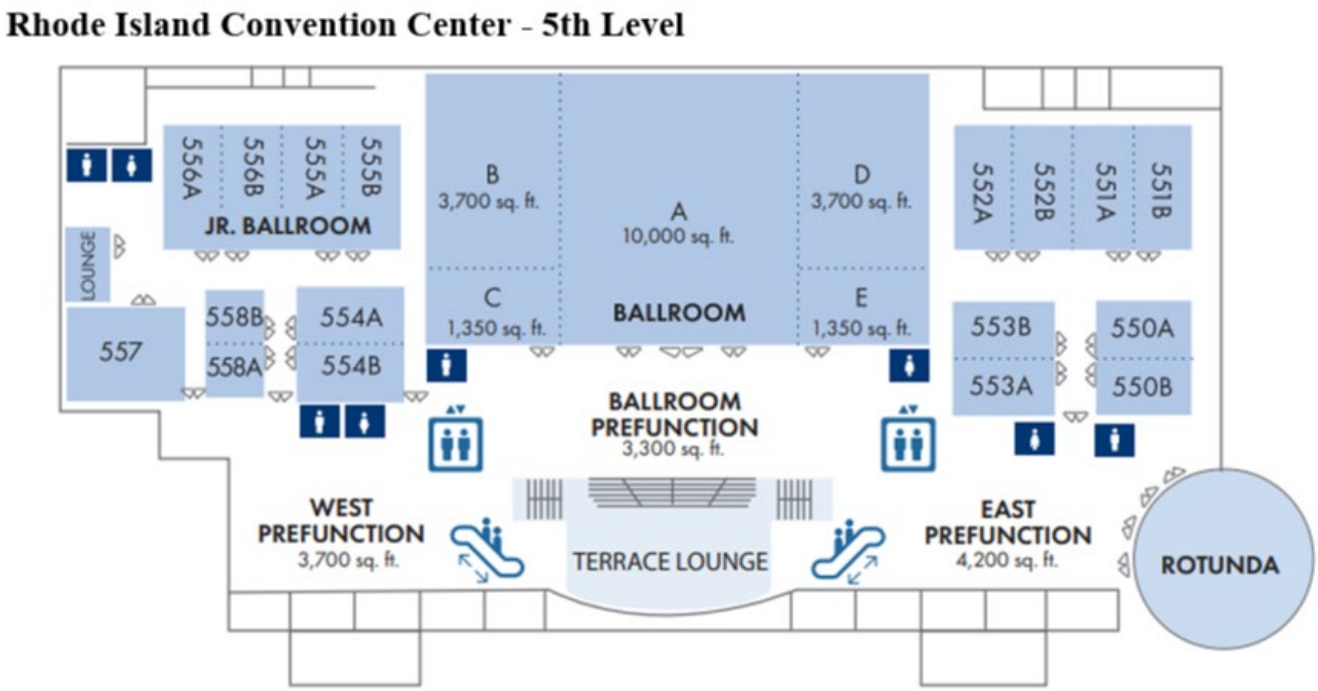
- Better identify treatment options for patients including optimal dosing of novel combination therapies
- Understand mechanisms of resistance to cancer therapy
- Improve patients' treatment follow-up and early diagnosis non-invasively
- Accelerate drug development through better patient selection

GENERAL INFORMATION

GENERAL INFORMATION – Symposium Information

Venue

WIN 2026 will be held in the 5th level of the Rhode Island Convention Center, 1 Sabin Street, Providence, RI 02903, United States.



Registration

The registration desks are located on the 5th floor of the Rhode Island Convention Center.

Registration Desk Opening hours:

Friday, October 2	**Saturday, October 3**
06.00am-5.30pm	06.00am-5.00pm

Your delegate registration includes:

- Morning Breakfast (2nd and 3rd October)
- Admissions to all scientific sessions and exhibitors'/posters' area
- Coffee and refreshments during morning and afternoon breaks
- Lunch (2nd and 3rd October)

GENERAL INFORMATION – Symposium Information

WIN Symposium App

Download the **Event App by EventsAir** to access the program, speaker information and this book from you device. Please use the code **WIN2026** to access the WIN Symposium App.

Google Play (Android)

App Store (Apple)

Badge

With your registration materials, you will receive a personalized name badge. During the symposium, this badge must be worn visibly at all times. It grants access to the scientific sessions, welcome reception & poster viewing. Only delegates wearing their badge will have access to these parts of the program.

Certificate of Attendance

Certificates of attendance will be emailed only to registered symposium participants who have completed the WIN 2026 online evaluation survey.
An invitation to participate in the evaluation survey will be sent via email to all registered participants, at the end of the symposium.

Disclosures

A print version of the disclosures of conflicts of interest by presenters and faculty of WIN 2026 is available from the registration desk during the Symposium.

Disclaimer

The Symposium Organizing Committee of WIN 2026, WIN Consortium and MCI do not accept any liability for injury or loss incurred by participants, accompanying persons and/or exhibitors, nor for loss of, or damage to their property and/or personal belongings.

Internet Access

Internet access is available throughout the Convention Center. Wi-Fi access codes will be displayed at the registration desk and in the rooms.

GENERAL INFORMATION – Symposium Information

Language
The official symposium language is English. No simultaneous translation is provided.

Parking
Location: North Garage, 99 West Exchange Street.
Parking is available at the North Garage. The garage is open 24 hours a day, 7 days a week and offers direct access to the Convention Center.
Drive-up parking rate is $25+7%tax. Symposium participants can pre-pay via this link: www.riconvention.com/plan-your-visit/directions-parking

Plenary Sessions
All plenary lecture sessions are held in Ballroom A on the 5th floor of the Rhode Island Convention Center.

Poster Sessions
All posters are located in the East Prefunction area on the 5th floor of the Rhode Island Convention Center.

Poster Viewing hours:

Friday, October 2
12.15pm-1.15pm (during lunch)
5.20pm-7.00pm (during Welcome Reception)

Saturday, October 3
1.10pm-2.10pm (during lunch)

Speaker Ready Room
The Speaker Ready room is located in Room 553 on the 5th floor of the Rhode Island Convention Center.

Speaker Service Center Opening hours:

Friday, October 2
07.00am-5.00pm

Saturday, October 3
07.00am-5.00pm

Welcome Reception
The Welcome Reception will take place in the Rotunda/East Prefunction area on the 5th floor of the Rhode Island Convention Center.

Friday, October 2: 5.20pm-7.00pm

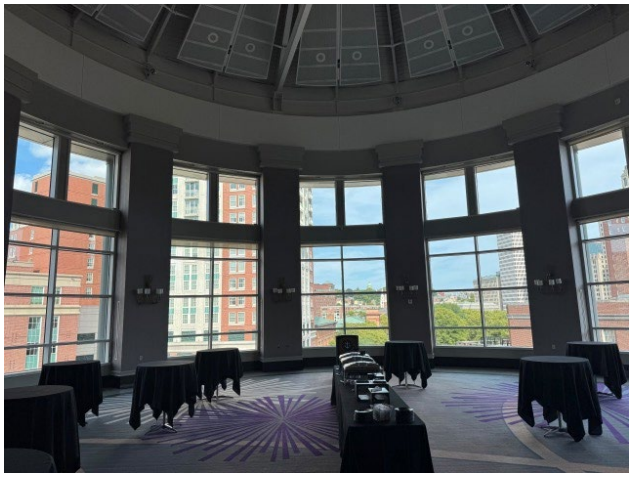
GENERAL INFORMATION – Social Events

Social Events

We warmly welcome all participants to our symposium to join us for a series of social events to foster connection, collaboration, and memorable moments together.

Thursday evening, October 1 – 6.00-8.30pm: Ocean State Labs – pre-WIN Symposium Reception.
Located in Providence's Innovation District, Ocean State Labs is a collaborative environment supporting life sciences innovation, entrepreneurship and scientific partnerships across Rhode Island.

Friday evening, October 6 – 5.20-7.00pm: Welcome Reception – Rotunda, Rhode Island Convention Center



GENERAL INFORMATION – Social Events

Saturday evening, October 3 – 6.00-9.00pm: 2026 WIN Symposium Banquet and Recognition Awards at the Hope Club in Providence.
Located on historic College Hill, East side of Providence, and established 150 years ago, the Hope Club is one of America's first private social clubs.

Sunday, October 4: Day Trip to Newport, Rhode Island.
Discover Newport, a charming coastal city renowned for its maritime heritage, picturesque waterfront, and grand Gilded Age mansions. We will have a stroll along the scenic Cliff Walk, admire historic estates, visit The Breakers, and enjoy the lively atmosphere of the harbor, lined with sailing boats, local shops, and restaurants. With its blend of history, architecture, and ocean views, Newport offers a memorable glimpse into New England's coastal charm.

GENERAL INFORMATION – Social Events

Discover Providence

While you attend the WIN Symposium, make the most of your time to discover the city of Providence. Known as Rhode Island's "Creative Capital," Providence offers a rich blend of history, culture, art, architecture, and renowned cuisine.

Explore the historic streets of College Hill and Benefit Street, home to beautifully preserved colonial buildings, Brown University, the Providence Athenaeum, and the RISD Museum. Stroll along the River Walk and Water place Park, admire the impressive Rhode Island State House, or enjoy panoramic city views from Prospect Terrace Park. Art lovers can discover vibrant murals, galleries, and cultural venues, while food enthusiasts can sample the city's acclaimed restaurants and explore the Italian heritage of Federal Hill.

Visit materials kindly offered by Go Providence can be found in your conference bag.

GENERAL INFORMATION – Social Media

Follow WIN Symposium on Social Media

We are using social media channels to inform you about the latest news on the WIN Symposium. If you are on X, LinkedIn or Facebook, join us and receive all WIN updates, ask questions to the organizers or connect with other participants of the WIN Symposium!
Please use the primary hashtag for the WIN Symposium 2026: #WINSympo2026 and don't hesitate to tag us @WIN_Consortium.

Facebook

If you have a Facebook account, search for WIN Symposium and follow us to receive the latest news on the WIN Consortium and WIN 2026 Symposium.

LinkedIn

If you have a profile on LinkedIn, join us on the WIN group:
Linkedin.com/company/win-symposium
Linkedin.com/company/win-consortium
Stay in touch with those you meet at the Symposium or reconnect with others from previous symposia or plan to meet at the next Symposium.

X

If you have an X profile, follow us @WIN_Consortium. We use X to announce the latest news on the WIN 2026 Symposium in real-time, discover updates about the program sessions, the speakers, announcements, photos and much more.

SCIENTIFIC/EDUCATIONAL INFORMATION

SCIENTIFIC/EDUCATIONAL INFORMATION – Learning Objectives

By the end of the Symposium, participants should be able to:

- Evaluate emerging advances in Precision Oncology and their clinical implications.
- Apply molecular profiling results to guide personalized treatment selection.
- Assess the role of tumor-agnostic therapies, basket trials, and innovative study designs.
- Integrate advances in immunotherapy, targeted therapies, radiation oncology, and translational research into patient management.
- Interpret the opportunities and limitations of AI, clinical informatics, and Real-World Data.
- Discuss regulatory approaches supporting innovation in precision medicine.
- Utilize Molecular Tumor Boards to optimize treatment planning and patient care.
- Identify strategies to improve access, equity, and implementation of Precision Oncology.
- Examine emerging approaches in neuro-oncology, pediatric oncology, microbiome science, and rare cancers.
- Foster international collaboration to accelerate innovation and expand access to Precision Oncology.

SCIENTIFIC/EDUCATIONAL INFORMATION – Symposium Goals

Scientific and Educational Goals

Present advances in genomics, translational science, immunotherapy, radiation oncology, AI, and cancer informatics; highlight novel therapeutic strategies and innovative clinical trial designs; disseminate best practices in biomarker-driven care; and promote multidisciplinary learning through lectures, panel discussions, and Molecular Tumor Boards. The meeting will also provide opportunities for trainees and investigators to present research and engage with international experts.

Strategic and Collaborative Goals

Advance equitable implementation of Precision Oncology worldwide; accelerate translation of scientific discoveries into improved patient outcomes; support innovation through genomics, AI, and emerging technologies; and strengthen collaboration among clinicians, researchers, regulators, industry representatives, patient advocates, and cancer centers to expand global access to Precision Oncology.

SCIENTIFIC/EDUCATIONAL INFORMATION – Educational Methods

The symposium will employ a variety of evidence-based educational formats designed to promote knowledge acquisition, critical thinking, multidisciplinary discussion, and application to clinical practice.

Expert Lectures and Keynote Presentations

- State-of-the-art presentations delivered by internationally recognized leaders in Precision Oncology, translational research, artificial intelligence, biomarker development, radiotherapy, and global oncology.
- Presentation of emerging scientific data, innovative technologies, and future directions in cancer care.

Fireside Chats

- Moderated conversations with distinguished leaders and policymakers, allowing exploration of complex and evolving topics through an interactive format.
- Opportunity for audience engagement and discussion of practical implications.

Panel Discussions

- Multidisciplinary panels bringing together experts from academia, industry, regulatory agencies, healthcare systems, and patient advocacy organizations.
- Exploration of controversies, implementation challenges, and opportunities for advancing Precision Oncology globally.
- Case-Based Discussions
- Integration of real-world clinical scenarios illustrating the application of Precision Oncology principles, biomarker-driven treatment decisions, and innovative therapeutic strategies.
- Interactive Question-and-Answer Sessions
- Dedicated discussion sessions following presentations and panels to facilitate audience participation, clarification of concepts, and exchange of experiences.

Patient Perspective Session

Presentation from a patient advocate and/or patient-scientist to highlight the patient experience, unmet needs, and the importance of patient-centered approaches in Precision Oncology.

Multidisciplinary Learning

Sessions designed to bring together medical oncologists, radiation oncologists, surgeons, pathologists, molecular biologists, bioinformaticians, data scientists, industry leaders, regulators, and patient representatives to foster cross disciplinary collaboration.

Networking and Collaborative Exchange

Opportunities for informal discussion and collaboration among participants from different regions and healthcare systems to promote the dissemination and implementation of best practices.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty Interviews

During WIN 2026, some faculty members will approach selected WIN Speakers to invite them to take part in brief, on-the-spot interviews. We greatly appreciate their willingness to share their expertise and insights with us, and we look forward to their valuable contributions.

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

Day 1: Friday, October 2, 2026

Plenary Keynotes, Regulatory Innovation, Genomics & Translational Science, Industry perspectives

06:30 07:30	**BREAKFAST** & EARLY NETWORKING — Registration Open	
OPENING SESSION **Wafik El-Deiry, MD, PhD, FACP** *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA* **Razelle Kurzrock, MD, FACP** *WIN Consortium, Chevilly-Larue, France & Medical College of Wisconsin, Milwaukee, WI, USA* **The Honorable Mia A. Ackerman** *Rhode Island State Representative for District 45* **The Honorable K. Joseph Shekarchi** *224th Speaker Emeritus of the Rhode Island House of Representatives*		
07:30 07:45	**OPENING** Welcome & Introduction to WIN Symposium 2026 | Symposium Directors	
07:45 08:15	**KEYNOTE** *30 min*	**Precision Oncology at an Inflection Point** **Wafik El-Deiry, MD, PhD, FACP** *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA*
08:15 08:40	**LECTURE** *25 min*	**Experiencing an Exceedingly Rare Cancer as a Scientist: From Existential Dread to Data at Scale** **Corrie Painter, PhD** *Even One Ventures, Boston, MA, USA*

SESSION 1 | NOBEL LAUREATE KEYNOTE		
08:40 09:15	**NOBEL LAUREATE KEYNOTE** *35 min*	**Precision Medicine for Brain Disorders** **Thomas C. Südhof, MD** *Stanford Medicine, Stanford, CA, USA* *— Nobel Laureate in Physiology or Medicine 2013*
09:15 09:35	**COFFEE BREAK**	
SESSION 2 | FROM DISCOVERY TO DELIVERY: ADVANCING PRECISION ONCOLOGY THROUGH TRANSLATIONAL AND REGULATORY INNOVATION **Chairs:** **Wafik El-Deiry, MD, PhD, FACP** *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA* **Razelle Kurzrock, MD, FACP** *WIN Consortium, Chevilly-Larue, France & Medical College of Wisconsin, Milwaukee, WI, USA*		
09:35 10:00	**KEYNOTE** *25 min*	**From Molecular Profiling to N-of-1 Oncology: Lessons from the I-PREDICT trial paradigm** **Razelle Kurzrock, MD, FACP** *WIN Consortium, Chevilly-Larue, France & Medical College of Wisconsin, Milwaukee, WI, USA*
10:00 10:25	**KEYNOTE** *25 min*	**Extending the Boundaries of Bedside-to-Bench Translational Research to Realize Rational Combination Therapies** **Keith T. Flaherty, MD, FAACR** *Massachusetts General Hospital / Harvard Medical School, USA | AACR President, Boston, MA, USA*
10:25 11:00	**FIRESIDE CHAT** *35 min*	**Regulatory Innovation and the Future of Precision Oncology** **Richard Pazdur, MD** *Former Director, FDA Oncology Center of Excellence, Washington, USA* In conversation with: Wafik El-Deiry • Razelle Kurzrock

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

SESSION 3 | INDUSTRY PERSPECTIVES

Chair: Bob Cormier JD *Rhode Island Life Sciences Hub (RILSH), RI, USA*

11:00 11:20 11.20 11.50	**LECTURE** *20 min* **PANEL 1** *30 min*	**AI and the Changing Frontier of Precision Medicine** **David Spetzler, MS, PhD, MBA** *Caris Life Sciences, Paradise Valley, AZ, USA* **Industry Perspectives: Genomic Profiling, Next-Generation Drugs & Precision Oncology** *Panelists:* **Zachary Boyd, MS, PhD** *J&J, Thousand Oaks, CA, USA* **John Tarantino, JD** *Iylon, USA* **David Spetzler, MS, PhD, MBA** *Caris Life Sciences, Paradise Valley, AZ, USA* **Navid Alipour, JD, MBA** *Curematch, USA* **Nesrin Erkol, PhD** *Vaccizone Biotechnology, Turkey* **Bradley Sander** *P53 Therapeutics, USA* **Emile Youssef, MD, PhD** *P53 Therapeutics, USA* *Genomic profiling in practice, novel drug targets, industry-academia partnerships*
11.50 12.15	**PANEL 2** *25 min*	**Pharma Managed Access Programs** *Moderator:* **Jeffrey R. Waldron, MBA** *Sherborn Consulting* *Panelists:* **Harpreet Ram, MBA** *EVR Consulting LLC, NJ, USA* **Lea Ann Browning-McNee, MS** *Reagan-Udall Foundation for the FDA, Frederick, MD, USA* **Annie Drelles, MPH** *Daiichi Sankyo, Stoughton, MA, USA*
12:15 13:15	**LUNCH -** BREAK & POSTER / Networking Session	

SESSION 4 | ADVANCING TUMOR-AGNOSTIC ONCOLOGY

Chairs:
Benedito A. Carneiro, MD, MS *Legorreta Cancer Center, Brown University, Providence, RI, USA*
Apostolia-Maria Tsimberidou, MD, PhD, MBA, FASCO, FAAAS *The University of Texas, MD Anderson Cancer Center, Houston, USA*
Jim Palma *TargetCancer Foundation, Cambridge, MA, USA*

13.15 13:45	**PANEL 2** *30 min*	**Tumor-Agnostic Therapy and Basket Trials** *Co-Chairs:* **Vivek Subbiah, MD** *Stanford Cancer Center, Palo Alto, CA, USA* **Keith T. Flaherty, MD, FAACR** *Massachusetts General Hospital / Harvard Medical School, USA | AACR President, Boston, MA, USA* *Panelists:* **Apostolia Maria Tsimberidou, MD, PhD, MBA, FASCO, FAAAS** *The University of Texas, MD Anderson Cancer Center, Houston, TX, USA* **Robyn Ward, MBBS, PhD, FRACP, FAHMS, FQA** *Monash University, Melbourne, Australia* **Sana Raoof, MD, PhD** *Brown University, Providence, RI, USA* **Jim Palma** *TargetCancer Foundation, Cambridge, MA, USA*
13:45 14:10	**LECTURE** *25 min*	**Closing the access gap: Universal Immunotherapy Reimbursement for Australians with Advanced and Metastatic Cancer** **Robyn Ward, MBBS, PhD, FRACP, FAHMS, FQA** *Monash University, Melbourne, Australia*
14:10 14:35	**LECTURE** *25 min*	**Precision Oncology Screening and Early Detection** **Sana Raoof, MD, PhD** *Brown University, Providence, RI, USA*
14:35 14:50	**COFFEE BREAK**	

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

SESSION 5 | TRANSLATIONAL ONCOLOGY

Chairs:
Sendurai A. Mani, MS, PhD *Legorreta Cancer Center, Brown University, Providence, RI, USA*
Attila A. Seyhan, PhD *Legorreta Cancer Center, Brown University, Providence, RI, USA*
Patrycja M. Dubielecka-Szczerba, PhD *Legorreta Cancer Center, Brown University, Providence, RI, USA*

14:50 15:15	**LECTURE** *25 min*	**Sequencing novel therapies in the modern era - implications for clinical trial design** **Giuseppe Curigliano, MD, PhD** *University of Milano / European Institute of Oncology, Milan, Italy | President-Elect, ESMO (2027 -2028)*
15:15 15:40	**LECTURE** *25 min*	**Defining the Frontier: Precision Oncology and The Next Generation of Prostate Cancer Therapeutics** **Benedito A. Carneiro, MD, MS** *Legorreta Cancer Center, Brown University, Providence, RI, USA*
15:40 16:05	**LECTURE** *25 min*	**Lessons Learned from a Career in Precision Oncology** **Jennifer Grandis, MD** *University of California San Francisco, San Francisco, CA, USA*
16:05 16:30	**LECTURE** *25 min*	**Heterogeneity in HER2+ Breast Cancer - Clinical and Functional Relevance** **Kornelia Polyak, MD, PhD** *Harvard Medical School / Dana-Farber Cancer Institute, Boston, MA, USA*

SESSION 6 | WIN INTERNATIONAL MOLECULAR TUMOR BOARD

Chairs:
Razelle Kurzrock, MD, FACP *WIN Consortium, Chevilly-Larue, France & Medical College of Wisconsin, Milwaukee, WI, USA*
Wafik El-Deiry, MD, PhD, FACP *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA*
Sewanti A. Limaye, MBBS, MD, MS *Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India*
Cara Mathews, MD, MS *Women & Infants Hospital of Rhode Island and Brown University, Providence, RI, USA*

16:30 17:15	**WIN MTB** *45 min*	**WIN International Molecular Tumor Board** *Chair:* **Razelle Kurzrock, MD, FACP** *WIN Consortium, Chevilly-Larue, France | Chair, WIN International MTB* *Co-Chairs:* **Wafik El-Deiry, MD, PhD, FACP** *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA* **Sewanti A. Limaye, MBBS, MD, MS** *Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India* *Panelists:* **Giuseppe Curigliano, MD, PhD** *University of Milano / European Institute of Oncology, Milan, Italy | President-Elect, ESMO (2027 -2028)* **Vivek Subbiah, MD** *Stanford Cancer Center, Palo Alto, CA, USA* **Giselle L. Saulnier Sholler, MD** *Penn State Health | Founder, Beat Childhood Cancer Research Consortium, Hershey, PA, USA* **Bicky Thapa, MD, MS, FACP** *Harvard Medical School / Dana-Farber Cancer Institute, Boston, MA, USA* *Multi-disciplinary live case discussions from WIN consortium institutions. Audience participation encouraged.*
17:15 17:20	**CLOSE** End of Day 1 — Closing Remarks by Symposium Directors	
17:20 19:00	**EVENING -** Welcome Reception *Rotunda, Rhode Island Convention Center*	

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

Day 2: Saturday, October 3, 2026

Global Equity, AI & Data Science, Translational Science

06:30 07:30	**BREAKFAST** & EARLY NETWORKING — Registration Open	
SESSION 7 | FUTURISTIC ONCOLOGY, NOVEL APPROACHES & TARGETS **Chairs:** **Yves Lussier, MD, FACMI, FIAHSI, FAIM, FAMIA** *The University of Utah, Salt Lake City, UT, USA* **Giselle L. Saulnier Sholler, MD** *Penn State Health | Founder, Beat Childhood Cancer Research Consortium, Hershey, PA, USA* **Robert W. Sobol, PhD** *Legorreta Cancer Center, Brown University, Providence, RI, USA*		
07:30 07:55	**LECTURE** *25 min*	**Next Generation Precision Oncology: Integrating Multi-Omics, ctDNA, Immunotherapy, and AI** **Apostolia Maria Tsimberidou, MD, PhD, MBA, FASCO, FAAAS** *The University of Texas, MD Anderson Cancer Center, Houston, TX, USA*
07:55 08:20	**LECTURE** *25 min*	**Microbiome and Cancer** **Sabine Hazan, MD** *Progenabiome / Ventura Clinical Trials, Ventura, CA, USA*
08:20 08:45	**LECTURE** *25 min*	**Precision Immunotherapy in Pediatric Oncology** **Giselle L. Saulnier Sholler, MD** *Penn State Health | Founder, Beat Childhood Cancer Research Consortium, Hershey, PA, USA*
08:45 09:25	**PANEL 3** *40 min*	**Cooperative Group with ACS Panel** *Co-Chair:* **Gevorg Tamamyan, MD, PhD, DSc,FASCO** *Oncodaily, USA* *Panelists:* **Wafik El-Deiry, MD, PhD, FACP** *WIN Consortium, Chevilly-Larue, France & Brown University Health, Providence, RI, USA* **Peter J. O'Dwyer, MD** *Group Co-Chair, ECOG-ACRIN Cancer Research Group | Perelman School of Medicine, University of Pennsylvania, USA* **Paul Campbell, PhD,** *American Cancer Society (ACS), USA* **Elvan Daniels, MD,** *American Cancer Society (ACS), USA* **Charles Balch, MD, FASCO, FACS** *International Society of Oncology Prevention and Treatments (ISOPT), Houston, TX, USA* **Razelle Kurzrock, MD, FACP** *Chair and Board of Governors Member, USA* **Ilyas Sahin, MD** *Mass General Brigham Cancer Institute, Harvard Medical School, Boston, MA, USA* **Stephen G. Chun, MD** *NRG Oncology Group, USA*
09:25 09:40	**COFFEE BREAK**	

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

SESSION 8 | GLOBAL PRECISION ONCOLOGY

Chairs:
Charles Balch, MD, FASCO, FACS *International Society of Oncology Prevention and Treatments (ISOPT), Houston, TX, USA*
Hesham Elghazaly, MD, MS *Egyptian Cancer Society, Cairo, Egypt*
Howard P. Safran, MD *Brown University Health Cancer Institute, Providence, RI, USA*

09:40 10:05	**LECTURE** *25 min*	**From Precision based Therapy to Precision Screening - a Global Fight Against Cancer!** **Sewanti A. Limaye, MBBS, MD, MS** *Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India*
10:05 10:30	**LECTURE** *25 min*	**Towards clinical utility for ctDNA in Breast Cancer** **Mark Basik, MD** *Segal Cancer Center, McGill University,Montreal, Quebec,Canada*
10:30 11:10	**PANEL 4** *40 min*	**Global Precision Oncology: Access, Equity & International Collaboration** *Co-Chairs:* **Charles Balch, MD, FASCO, FACS** *International Society of Oncology Prevention and Treatments (ISOPT), Houston, TX, USA* **Hesham ElGhazaly, MD, MS** *Egyptian Cancer Society, Cairo, Egypt* *Panelists:* **Gillian Lamoury, MD, FRANZCR** *University of Sydney, Australia* **Sewanti A. Limaye, MBBS, MD, MS** *Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India* **Hongxia Wang, MD, PhD** *Shanghai Cancer Center, Fudan University, Shanghai, China* **Ning Liao, MD, PhD** *Guangdong Provincial People's Hospital, Guangzhou, China* **Hikmat Abdel-Razeq, MD** *King Hussein Cancer Center, Amman, Jordan*
11:10 11:35	**LECTURE** *25 min*	**The Global Cancer Epidemic in the Working Age population** **Charles Balch, MD, FASCO, FACS** *International Society of Oncology Prevention and Treatments (ISOPT), Houston, TX, USA*
11:35 12:00	**LECTURE** *25 min*	**Building an Equitable Germline Genetic Testing Program in a Resource-Constrained Setting: Lessons from Jordan** **Hikmat Abdel-Razeq, MD** *King Hussein Cancer Center, Amman, Jordan*

SESSION 9 | RADIATION ONCOLOGY

Chairs:
David E. Wazer, MD *Brown University Health Cancer Institute, Providence, RI, USA*
Amir Onn, MD *Sheba Medical Center, Ramat Gan, ISRAEL*
Michelle R. Dawson, PhD *Brown University, Providence, RI, USA*

12:00 12:25	**LECTURE** *25 min*	**Lessons from the Integration of Checkpoint Immunotherapy with Radiation in Thoracic Malignancies** **Stephen G. Chun, MD** *The University of Texas, MD Anderson Cancer Center, Houston, TX, USA*
12:25 12:50	**LECTURE** *25 min*	**Bringing Radiation Oncology into the Era of Personalized Medicine** **Jacob Scott, MD, DPhil** *Cleveland Clinic / CWRU School of Medicine, Cleveland, OH, USA*
12:50 13:10	**WIN Consortium Fellows' posters** **The inaugural Class of 2026 WIN Fellows:** **Andre L. De Souza, MD** *The University of Oklahoma Health, OK, USA* **Karan Sood, MD, DNB, DrNB, DM** *TATA Memorial Hospital, Mumbai, India* **Daniel Sur, MD, PhD** *University of Medicine "I. Hatieganu" Cluj Napoca, Romania*	
13:10 14:10	**LUNCH -** BREAK & POSTER / Networking Session	

SCIENTIFIC/EDUCATIONAL INFORMATION – Program

SESSION 10 | ARTIFICIAL INTELLIGENCE & DATA SCIENCE IN PRECISION ONCOLOGY **Chairs:** **Matthew J. Hadfield, DO** *Brown University Health Cancer Institute, Providence, RI, USA* **Stephen G. Chun, MD** *The University of Texas, MD Anderson Cancer Center, Houston, TX, USA* **Brenda M. Rubenstein** *PhD Brown University, Providence, RI, USA*		
14:10 14:35	**LECTURE** *25 min*	**Towards Fast and Accessible Precision Oncology** **Eytan Ruppin, MD, PhD** *Cedars-Sinai, Los Angeles, CA, USA*
14:35 15:15	**PANEL 5** *40 min*	**Artificial Intelligence & Data Science in Precision Oncology** *Chair:* **Yves Lussier, MD, FACMI, FIAHSI, FAIM, FAMIA** *The University of Utah, Salt Lake City, UT, USA* *Panelists:* **Eytan Ruppin, MD, PhD** *Cedars-Sinai, Los Angeles, CA, USA* **Sean Khozin, MD, MPH** *CEO Roundtable on Cancer and Project Data Sphere, USA* *AI biomarker discovery, clinical decision support, equity in AI, real-world data*
SESSION 11 | PRECISION ONCOLOGY IN PRACTICE FOR RARE AND COMPLEX CANCERS **Chairs:** **Misty Shields, MD, PhD** *Indiana University School of Medicine, Indianapolis, IN, USA* **Jens Rueter, MD** *The Jackson Laboratory, Maine Cancer Genomics Initiative, Bar Harbor, ME, USA* **Sean Lawler, PhD** *Brown University, Providence, RI, USA*		
15:15 15:45	**LECTURE** *30 min*	**Rethinking the Rules of Cancer Drug Development: From Mutation to Tumor-agnostic Precision Medicine** **Vivek Subbiah, MD** *Stanford Cancer Center, Palo Alto, CA, USA*
15:45 16:10	**LECTURE** *25 min*	**Treating the Uncommon: Practical Strategies for Rare Genitourinary Cancers in the Precision Era** **Bicky Thapa, MD, MS, FACP** *Harvard Medical School / Dana-Farber Cancer Institute, Boston, MA, USA*
16:10 16:35	**LECTURE** *25 min*	**Cancer Cell Adaptation: From Mechanism to Precision Oncology** **Sendurai A. Mani, MS, PhD** *Legorreta Cancer Center, Brown University, Providence, RI, USA*
16:35 17:00	**LECTURE** *25 min*	**Precision Oncology in SCLC: From Molecular Substypes to Therapeutic Vulnerabilities** **Misty Shields, MD, PhD** *Indiana University School of Medicine, Indianapolis, IN, USA*
CLOSING CEREMONY		
17:00 17:10	**CLOSING**	**Closing Ceremony — Awards, Takeaways & Symposium Synthesis** **Symposium Directors** *WIN Consortium* *Awards, poster prizes, and announcement of WIN Symposium 2027*
18:00 21:00	**EVENING** 2026 WIN Symposium Banquet and Recognition Awards *Hope Club, 6 Benevolent Street, Providence, RI, 02906, USA*	

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

The WIN Symposium 2026 Organizers are honored by the presence of our distinguished speakers for opening the WIN Symposium 2026.

The Honorable Mia A. Ackerman

Rhode Island State Representative for District 45, USA

Rep. Mia A. Ackerman (D) has served the residents of Cumberland and Lincoln in District 45 since winning her first election in November 2012. She was named the first-ever female Deputy Majority Whip in January 2021 and is a member of the House Conduct Committee, the House Health and Human Services Committee, and the House Veterans' Affairs Committee.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

The Honorable K. Joseph Shekarchi

224th Speaker Emeritus of the Rhode Island House of Representatives, USA

After being elected by his House colleagues in 2021, K. Joseph Shekarchi recently completed nearly three terms as Speaker of the Rhode Island House of Representatives.

He previously served two terms as the House Majority Leader, and he was also the Chairman of the House Labor Committee. Speaker Shekarchi served the residents of District 23 in Warwick as state representative since first being elected in November 2012.

During his tenure as Speaker, Joe Shekarchi pledged to make housing issues the cornerstone of his legislative work and shepherded almost 60 housing bills into law. The bills, coupled with historic budget investments, were designed to tackle the state's housing crisis from multiple angles.

Joe's other legislative accomplishments include championing the development of the life science sector, elimination of the tangible tax for most small businesses, and the permanent phase-out of the car tax one year ahead of schedule.

Speaker Shekarchi also spearheaded the passage of landmark pay equity legislation, enacted strong environmental protection laws, and ensured that state aid for public education was significantly increased every year.

Joe is a graduate of Mount St. Charles Academy, Class of 1980, and graduated from Suffolk University in 1984. He returned to Suffolk to earn his law degree in 1990. In addition to his public service as a legislator, Speaker Shekarchi has been a successful attorney in private practice for over 30 years. Speaker Shekarchi is a lifelong Warwick resident.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

The WIN Symposium 2026 Organizers are very grateful to the Symposium faculty, whose names, affiliations and short biographies are listed below in alphabetical order.

Hikmat N. Abdel-Razeq, MD

Member, WIN Consortium
Chief Medical Officer and Deputy Director General
King Hussein Cancer Center, Jordan

Professor Hikmat Abdel-Razeq is Chief Medical Officer and Deputy Director General of the King Hussein Cancer Center, where he also serves as Chairman of the Department of Internal Medicine. He holds an academic appointment as Professor of Medicine at the University of Jordan School of Medicine.

He earned his medical degree from the University of Jordan in 1988 and completed his postgraduate training at New York Medical College and the Cleveland Clinic Foundation. Professor Abdel-Razeq is board-certified by the American Board of Internal Medicine in Internal Medicine, Medical Oncology, and Hematology.

A recognized leader in breast cancer, cancer genetics, and cancer-associated thrombosis, Professor Abdel-Razeq has made substantial contributions to clinical research and academic scholarship. He is the recipient of numerous national and international honors, including the King Hussein Cancer Research Award (2021). He serves on the editorial boards of several international peer-reviewed journals and has authored more than 200 scientific publications and book chapters in his fields of expertise.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Navid Alipour, JD, MBA
Co-Founder and CEO
Curematch, USA

Navid Alipour is the Co-Founder and CEO of CureMatch, a company focused on personalized medicine & combination therapy in oncology. The CureMatch Decision Support System guides the selection of cancer drugs customized for individual patients based on their molecular tumor profile. He is also the Co-founder/CEO of CureMetrix, which focuses on Women's health. CureMetrix has the first and only FDA cleared AI that detects & scores breast arterial calcification, from the same mammogram used to screen for breast cancer.

Through the founding of Analytics Ventures, an AI-focused VC fund & multiple companies, Navid focuses on the convergence of AI and the life sciences to prolong and save lives while reducing wasteful spending.

Navid serves on several Boards such as Tech San Diego, CureMetrix, CureMatch, Praxis Solutions, & the advisory Board at the University of San Diego's Applied AI and Applied Data Science program. He is a sought-after panelist, judge, speaker, and moderator on the topics of venture capital, artificial intelligence, healthcare, and entrepreneurship. Navid holds a BA from UC San Diego, and a JD/MBA from the University of San Diego.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Charles M. Balch, MD, FASCO, FACS
Member, Scientific Advisory Board, WIN Consortium
President
International Society of Oncology Prevention and Treatments (ISOPT), USA
Professor Emeritus and Past-Chair, Department of Surgical Oncology
The University of Texas MD Anderson Cancer Center, USA

Dr. Charles M. Balch has led a distinguished career as a clinical and academic surgical oncologist for the past 50 years, as a leading authority in both melanoma, breast cancer, and immunotherapy. He has also made significant contributions to laboratory research in tumor immunology and human T lymphocyte differentiation. He is author of over 848 publications, which have been cited over 38,000 times in the biomedical literature (h-index: 92). He was recently cited as "Top 5%" for all medical publications for the number of citations and citation index (Doximity March 2026). He has lectured in over 50 different countries in the world and most of the major academic centers in the United States. Dr Balch was the founding Editor-in-Chief of the Annals of Surgical Oncology, which is recognized as the leading journal in the world in its field. In past years, Dr. Balch has served as Executive Vice President and CEO of the American Society of Clinical Oncology, as President and CEO of the City of Hope National Medical Center, as Executive Vice President for Health Affairs, Vice President of Hospital and Clinics, and Surgeon-in-Chief and Head of the Division of Surgery, among other positions at the University of Texas M.D. Anderson Cancer Center. He has served on leadership positions at the International Society of Oncology Prevention and Treatments (President), Society of Surgical Oncology (President), Association of Academic Surgeons (President), the National Comprehensive Cancer Network (Founding Board of Directors), the Commission on Cancer (Chair, Board of Directors) and the American Joint Committee on Cancer (Executive Committee).

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Mark Basik, MDCM

Herbert Black Professor of Surgical Oncology Head, Cancer Genomics and Translational Research
Laboratory at the Lady Davis Institute for Medical Research
Segal Cancer Centre, McGill University, Canada

Dr. Mark Basik holds the Herbert Black Chair in Surgical Oncology at McGill University and is a surgical oncologist at the Jewish General Hospital. He directs the Cancer Genomics and Translational Research Laboratory at the Lady Davis Institute. He is also the principal investigator of several clinical trials. His fundamental research interests include understanding the causes of treatment resistance in breast cancer and developing novel blood tests for breast cancer diagnosis using circulating tumor DNA.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Zachary Boyd, MD, PhD

Vice President – Global Head, Oncology Precision Medicine & Diagnostics
Johnson & Johnson, USA

Zach Boyd, Vice President and Global Head of Precision Medicine and Diagnostics at Johnson & Johnson Innovative Medicines, leads a team of scientists and developers responsible for partnered diagnostic solutions and biomarker data delivery that enable the development and use of therapeutic products. With more than 20 years of experience across leading biopharmaceutical organizations, Zach is passionate about advancing precision medicine to deliver life-enabling options for patients. A strong collaborator, he actively partners across teams and organizations to drive more effective personalized medicines and diagnostic solutions.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Lea Ann Browning-McNee, MS
Director of Communication & Stakeholder Engagement
Reagan-Udall Foundation for the FDA, USA

Lea Ann Browning-McNee is director of communication and stakeholder engagement for the Reagan-Udall Foundation for the FDA, the nonprofit, non-government organization established by Congress to support the Food and Drug Administration. She manages multiple program portfolios at the Foundation including the Expanded Access Navigator, which includes the eRequest app developed in partnership with FDA. With more than 25 years of experience in communications, Lea Ann specializes in consumer insights, health education, and patient advocacy. Before joining the Foundation, she held senior leadership posts at BrainFutures, National Council for Behavioral Health, National Mental Health Association, and the Mental Health Association of Maryland and was adjunct faculty for sixteen years at the George Washington University Graduate School of Political Management. She received her B.S. and M.S. from Towson University.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Paul Campbell, PhD
Scientific Director - Cell Biology and Preclinical Cancer Research
American Cancer Society, USA

As Scientific Director of the Cell Biology & Preclinical Cancer Research Program in the Extramural Discovery Science department of ACS, Dr. Campbell oversees peer review and grant funding in areas related to cancer cell biology, tumor microenvironment, experimental therapeutics, biomarker discovery and translational science. Prior to joining ACS, Dr. Campbell was faculty in the Cancer Signaling and Microenvironment Program and The Marvin and Concetta Greenberg Pancreatic Cancer Institute at Fox Chase Cancer Center, and cross-appointed in the Department of Cancer and Cellular Biology at Temple University's Lewis Katz School of Medicine.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Benedito A. Carneiro, MD, MS

Member, WIN Consortium
Director of Clinical Research and Cancer Drug Development (Phase I)
Associate Director, Division of Hematology/Oncology Associate Professor of Medicine, Warren Alpert School of Medicine Co-Leader Cancer Therapeutics Program
Legorreta Cancer Center at Brown University, USA

Benedito A. Carneiro, MD, MS, is a medical oncologist and clinical investigator specializing in early drug development and first-in-human clinical trials of novel therapeutics for solid tumors. He is the founder and Director of the Cancer Drug Development Program (Phase I), Director of Clinical Research at the Brown University Health Cancer Institute, Associate Director of the Division of Hematology/Oncology, and Co-Leader of the Cancer Therapeutics Program at the Legorreta Cancer Center at Brown University. He has served as Principal Investigator or Co-Investigator on more than 100 clinical trials, including numerous first-in-human (phase I) studies across kidney, bladder, prostate, lung, pancreas, and colon cancers. His translational research spans innovative immunotherapies, genomics-driven treatment strategies, cellular therapies, and biomarkers of treatment response and resistance. He has authored more than 130 peer-reviewed publications. Dr. Carneiro is an active member of several professional societies, including the American Society of Clinical Oncology (ASCO), the European Society for Medical Oncology (ESMO), and the American Association for Cancer Research (AACR). He represents the Legorreta Cancer Center at the WIN (Worldwide Innovative Networking in Personalized Cancer Medicine) Consortium, serves as the Co-Chair of Genitourinary Disease Committee within the WIN Consortium, and has contributed to the ASCO Annual Meeting Scientific Program Committee and the AACR Annual Meeting Clinical Trials Committee.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Stephen G. Chun, MD
Member, Scientific Advisory Board, WIN Consortium
Associate Professor of Radiation Oncology
University of Texas MD Anderson Cancer Center, USA

Dr. Chun is an Associate Professor of Radiation Oncology at the MD Anderson Cancer Center. As Director of Radiation Oncology Clinical Research for the MD Anderson Houston Area Locations, Dr. Chun led the expansion of the radiation oncology clinical trial program across the MD Anderson integrated academic satellite network and is supported by grant funding from the National Institutes of Health as Principal Investigator. With the NRG Oncology group, Dr. Chun serves as national Vice Chair of the Protocol Operations Management and is co-host of the NRG Oncology Podcast. As an active clinical researcher, Dr. Chun has published in journals such as the Lancet, Journal of Clinical Oncology, Journal of Thoracic Oncology and JAMA Oncology.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Bob Cormier, JD

President and Chief Executive Officer
Rhode Island Life Science Hub, USA

Bob Cormier is President and Chief Executive Officer of the Rhode Island Life Science Hub (RILSH). A veteran medical-device executive with more than 25 years of leadership across startups, mid-size companies, and a Fortune 500 enterprise, he has built his career bringing new technology to the bedside — and building the companies that get it there.

Most recently, Cormier was CEO of Sentec, a global respiratory and critical-care monitoring company that he grew into a mid-size enterprise and whose technology became widely adopted in NICUs around the world. Over his career he has been part of dozens of company acquisitions and integrations, licensing and distribution deals, and new product introductions, and he co-founded Health Data Analytics Institute (HDAI), a digital health and AI startup. He has also held senior leadership roles at Covidien, Aspect Medical Systems, and Tyco Healthcare. Alongside his operating career, Cormier has worked on the investor side of the industry, including with family offices and private equity firms, giving him a dual perspective on both building life science companies and financing them.

A Rhode Islander for more than 30 years, Cormier holds a B.S. from the University of Rhode Island and a J.D. from Roger Williams University School of Law. At RILSH, he is focused on turning Rhode Island's research strength into a self-sustaining, nationally recognized life science economy — a place where companies choose to build, hire, and stay.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Giuseppe Curigliano, MD, PhD

Associate Professor of Medical Oncology
University of Milano Head, Division of Early Drug Development, European Institute of Oncology (IEO), Italy
President-Elect, 2027-2028, European Society for Medical Oncology (ESMO)

Giuseppe Curigliano, MD, PhD, is the director of the Early Drug Development Division at the European Institute of Oncology, a comprehensive cancer center in Milano, Italy. He is professor of Medical Oncology in the Department of Oncology and Hemato-Oncology at the University of Milano. In ESMO he served as Chair the ESMO Nomination Committee (2017-2019) and as Chair of the ESMO Clinical Practice Guideline Committee (2019-2023). He was one of the founding members and served as scientific Co-Chair of the "ESMO Breast Cancer Congress". He also served several years as scientific Co-chair of St Gallen Early Breast Consensus Conference. He contributed to launch the ESMO Living Guidelines and to the recognition of the Fellow of ESMO (FESMO). He organized and participated in many professional and educational congresses for oncologists all over the world. Professor Curigliano's research focuses on biomarkers and personalized therapies, with an emphasis on biomarker discovery, targeted agent development, and the integration of personalized medicine. He leads Phase I-III clinical trials on targeted therapies for breast cancer and oversees large-scale national studies on the implementation of high-throughput technologies in healthcare systems. Professor Curigliano was identified as Clarivate™ world's most influential researchers (the highly cited researchers) in 2022, 2023 and 2024. He has authored more than 900 papers in peer-reviewed journals (h-index 100) and many book chapters. He served as editorial board member for international journals in oncology (European Journal of Oncology, Journal of Clinical Oncology, Annals of Oncology). He served as Editor in Chief of ESMO Open from 2022 to 2025. He trained, mentored and sponsored dozens of medical oncologists from Latin America, Africa, Asia and Italy. Professor Curigliano was awarded with the first ESO Umberto Veronesi Award (2017) and with the Fellowship of the European Academy of Cancer Sciences in Paris (2017). He is ESMO President elect.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Elvan (Ellie) Daniels, MD, MPH

Senior Vice President, Center for Innovation in Cancer Research Training
American Cancer Society, USA

Elvan (Ellie) Daniels, MD, MPH, is Senior Vice President of the American Cancer Society Center for Innovation in Cancer Research Training, where she leads the development and implementation of training programs focused on the next generation of cancer research and care professionals.

A family physician by training, Dr. Daniels earned her medical degree at Wayne State University and completed her family medicine residency at Eastern Virginia Medical School's Ghent Family Practice. She went on to complete a clinical research fellowship at the Medical University of South Carolina and to earn a Master of Public Health from the Rollins School of Public Health at Emory University.

Dr. Daniels brings a career-long focus on community-engaged research, practice-based research networks, and workforce development to her current role. She previously served as medical director of two large federally qualified health centers and as director of a women's wellness center. At the Morehouse School of Medicine, she held a series of research and training leadership roles, including Associate Director for Community-Oriented Primary Care at the National Center for Primary Care; Director of the Southeast Regional Clinicians' Network (SERCN), a practice-based research network of federally qualified health centers; and Co-Leader of the Research Training and Career Development Core for the Morehouse School of Medicine/Tuskegee University/University of Alabama at Birmingham Cancer Partnership. She also served as a lead mentor for medical student learning communities.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Michelle R. Dawson, PhD
Associate Professor of Medical Science and Engineering, Department of Molecular Biology, Cell Biology, and Biochemistry
Associate Director for Cancer Research Training and Education Coordination
Legorreta Cancer Center, Brown University, USA

Dr. Michelle Dawson is a tenured Associate Professor of Molecular Biology, Cell Biology and Biochemistry at Brown University and Associate Director for Cancer Research Training and Education Coordination (CRTEC) at the Legorreta Cancer Center. With interdisciplinary training in Biomedical Engineering (BS), Chemical and Biomolecular Engineering (PhD), and Tumor Biology (postdoctoral training), she brings an engineering perspective to understanding how cancer cells adapt to and remodel their microenvironment. Her research integrates mechanobiology, metabolism, and quantitative cell biology to define mechanisms of tumor progression and therapeutic resistance. Her work on polyploid giant cancer cells (PGCCs) has identified distinct features of their cytoskeletal organization, metabolic adaptation, and chemoresistance, providing insight into how these stress-induced cell states persist and contribute to tumor recurrence. In complementary studies, her laboratory investigates how cellular senescence drives extracellular matrix (ECM) remodeling, altered tissue mechanics, and fibrosis, and how these changes create microenvironments that influence subsequent cellular behavior. Her research has been supported by both the National Science Foundation and National Institutes of Health, reflecting its contributions to fundamental cell biology and its relevance to cancer and human disease.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Andre de Souza, MD
Inaugural WIN Fellow 2026, WIN Consortium
Genitourinary Medical Oncology Director, Associate Professor, Phase I physician
Department of Internal Medicine, College of Medicine,
University of Oklahoma Health Campus, USA

I graduated from medical school in 2008, where I led a curriculum transformation group instituted by the Dean that won a 1-million-dollar Grant by the National Department of Health. I completed Internal Medicine Residency and Hematology/Oncology Fellowship training in Massachusetts and Rhode Island, United States. I completed training in early phase clinical trials at the Advanced Developmental Therapeutics Training program of the National Cancer Institute, National Institutes of Health, in Bethesda, Maryland. I am the Director of Genitourinary Medical Oncology at Oklahoma University, a co-PI at a Department of Defense Grant, a Faculty at a Masters in Foundations of Clinical Research and a World Innovation Network Fellow. I hold 2 provisional patents in drug development. I am affiliated to the rare malignancies and genitourinary malignancies committees at SWOG and ETCTN. I have been a member of IRBs and PRMCs, and proudly an author of the chapters on cancer biology and genitourinary cancers in Cecil Essentials. I have been a co-investigator in 150 trials and principal investigator in 11 trials, with 30 publications related to Oncology. I am married to a Brazilian woman with Italian roots and enjoy travelling, history and music. As an associate professor, I am mentoring 4 fellows, 4 medical students and collaborating with 5 labs, pathologists and biostatisticians in MTAP deleted tumors and peritoneal carcinomatosis. I see WIN as the future of Oncology, driving innovative approaches to cancer care across the planet.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Annie Drelles, MPH

Senior Director & Head, Office of Medical Access
Global Oncology Medical Affairs, Clinical Trial Management, Resources, Operations, & Governance
Daiichi Sankyo, Inc.

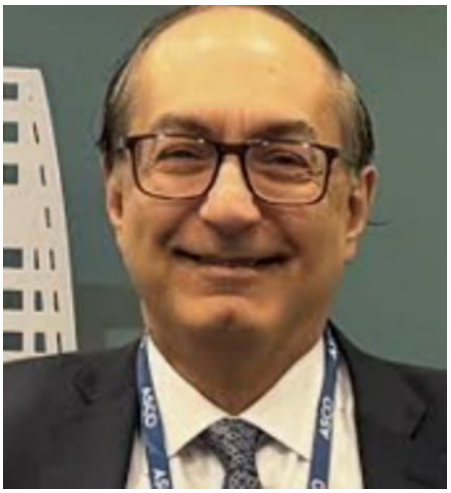
Annie Drelles is Head of the Office of Medical Access within Global Oncology Medical Affairs at Daiichi Sankyo. She leads the company's global strategy, governance, and operational oversight for Medical Access Programs, supporting patients with serious diseases who may have limited treatment options. Annie is also driving efforts to standardize medical access practices globally and strengthen cross-functional collaboration across regulatory, clinical, safety, commercial, and regional teams. She holds a Master of Public Health from George Washington University.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Patrycja Dubielecka-Szczerba, PhD
Associate Professor of Medicine (Research)
Warren Alpert Medical School of Brown University, USA

Dr. Dubielecka is an Associate Professor of Medicine at the Alpert Medical School of Brown University and Director of Translational Hematology within the Division of Hematology/Oncology in the Department of Medicine at the Rhode Island Hospital. Dr. Dubielecka is a Co-Director of Pathobiology Graduate Program at Brown University and a Co-Leader of the Cancer Biology Program at the Legorreta Cancer Center at Brown University. Dr. Dubielecka received her undergraduate (summa cum laude) and PhD degrees (with honors) from the University of Wroclaw, Poland. She was a scholar of the Socrates/Erasmus Program established by the European Commission, and trained at the University of Helsinki, Finland and at Charles University in Hradec Kralove, Czech Republic. Dr. Dubielecka did her postdoctoral training at the Lindsley F. Kimball Research Institute in New York and was a visiting scientist in training at the University of Michigan, Ann Arbor and Helmholtz Centre for Infection Research in Braunschweig, Germany. Dr. Dubielecka established her independent scientific program in 2012. Her program is focused on the biology of blood cancer-initiating stem cells and their role in the development, progression and relapse of myeloproliferative neoplasms with the translational goal of therapeutically targeting neoplasm-initiating stem cells.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty



Wafik El-Deiry, MD, PhD, FACP
Chair WIN Consortium
American Cancer Society Professor
Mencoff Family University Professor of Medical Science
Professor of Pathology and Laboratory Medicine
Brown University Health, USA

Dr. El-Deiry is an American Cancer Society Professor, Mencoff Family University Professor of Medical Science at Brown, Founding Director of the Legorreta Cancer Center (emeritus, July 2026), inaugural Associate Dean for Oncologic Sciences at Brown (emeritus, July 2026) and practicing physician-scientist in GI Oncology and Precision Oncology at Brown University Health.

El-Deiry's discoveries have shaped the fields of tumor suppression, cell death signaling, and cancer therapeutics. He elucidated the genomic DNA-binding consensus sequence for the p53 tumor suppressor, published in Nature Genetics in 1992, and went on to discover p21(WAF1) as a p53 target gene and cell cycle inhibitor — the first mammalian CDK inhibitor identified, and the work that opened the path to the CDK inhibitor drugs now in clinical use. That 1993 paper in Cell was the most-cited original article in the journal for over two decades. He discovered TRAIL receptor DR5 as a p53 target and identified ONC201/TIC10 (dordaviprone), a first-in-class small molecule that activates TRAIL-mediated cancer cell death. He founded Oncoceutics, the company that licensed the compound, and in August 2025, the drug received FDA approval for recurrent H3K27M-mutant diffuse glioma — over two decades from bench to bedside. His laboratory continues to develop agents that restore p53 pathway signaling in tumors carrying mutant p53. He is a Senior Member of the National Academy of Inventors.

He has published more than 500 peer-reviewed papers, edited eight books, and ranks among the most highly cited cancer researchers worldwide (121,426 citations; H-index 136 in September 2026 according to Google Scholar).

Dr. El-Deiry held leadership roles as co-program leader for Radiobiology & Imaging at the

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Abramson Comprehensive Cancer Center, Chief of Hematology/Oncology at Penn State University and Deputy Director for Translational Research at Fox Chase Cancer Center before being recruited to Brown by the end of 2018.

In the current phase of his career Dr. El-Deiry is leading the WIN Consortium in Precision Oncology, growing a Center of Excellence in Precision Oncology at Brown and is committed to supporting the biotechnology ecosystem in Rhode Island. In this regard he is advancing biotech startups p53-Therapeutics, SMURF-Therapeutics, Resurrect-Therapeutics and building a venture fund Wampanoag Ventures while earning a Physician MBA at Indiana University. He continues to conduct competitive funded research at Brown and support the careers of trainees and colleagues. Dr. El-Deiry is serving a term as the elected Secretary of the Faculty at Brown University for 2026-2027.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Hesham Elghazaly, MD, MS

Member, WIN Consortium
President, Egyptian Cancer Society (ECS), Egypt

Prof. Hesham Elghazaly, is a clinical oncology professor with an experience over 30 years, affiliated with different titles mainly as Executive Director of the Medical Research Center (MASRI) at Ain Shams University, and chairs the Scientific committee at the Presidential Initiative of Women's Health (PIWH) in Egypt, Also serves as a member of the early breast scientific committee in ESMO, member of the AORTIC research committee, and Union for International Cancer Control (UICC). A member of the International Agency for Research on Cancer (IARC/WHO) and the scientific committee and Advanced breast cancer (ABC) Global Alliance. His work focuses on Cancer care equity in LMICs and to enhance the global partnerships to develop the clinical research in Africa and the region.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Nesrin Erkol, PhD

Department Chair, Department of Molecular Biology and Genetics, Boğaziçi University
CEO/CSO, Vaccizone Biotechnology, Türkiye

With more than 30 years' experience in basic research on the topics of apoptosis, pyroptosis, inflammation, Behcet's disease and cancer. Her research team- Apoptosis and Cancer Immunology Laboratory is part of the Molecular Biology and Genetics Department at the legendary Boğaziçi University. With over 30 publications and 5,400 citations, Nesrin Erkol is holder of the first triadic patent of the country in the field of biotechnology and one of the two co-leaders on the first Turkish Genome Project.

One of the founders of the Molecular Biology Association of Türkiye (2011), its first vice president and president elect for 7 years (2014-2021). They established an ecosystem for sharing research, protocols and reagents in the country. During the COVID-19 pandemic, they cooperated with other Biology societies and were actively involved in dissemination of correct information about the qPCR tests. They recruited more than 3,000 volunteers to work at the rapidly growing numbers of qPCR testing centers that were urgently needed.

In 2020, Nesrin Erkol established Vaccizone Biotechnology Research, Development Consulting Industry and Commerce Inc. - a start-up company located at Boğaziçi University Teknopark Center to commercialize the first triadic patent of Türkiye, namely the ASC microparticle technology. Vaccizone was able to raise $1M USD in angel investor and government funds and grants. Currently Vaccizone is pursuing biomarker targeted immunotherapy for melanoma projects and VC funding is needed to bring the projects to human Phase I and II.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Keith T. Flaherty, MD, FAACR

Member, Scientific Advisory Board, WIN Consortium
Richard Saltonstall Chair in Oncology,
Massachusetts General Hospital, Professor of Medicine, Harvard Medical School, USA
President, American Association for Cancer Research (AACR)

Dr. Flaherty is the Richard Saltonstall Chair in Oncology at the Massachusetts General and Professor of Medicine at Harvard Medical School. As described in more than 350 peer reviewed primary research reports he has authored or co-authored, Dr. Flaherty and colleagues made several seminal observations that have defined the treatment of melanoma when they established the efficacy of BRAF, MEK and combined BRAF/MEK inhibition in patients with metastatic melanoma in a series of New England Journal of Medicine articles for which Dr. Flaherty was the first or senior author. He was the principal investigator of the NCI MATCH trial, the first NCI-sponsored trial assigning patients to targeted therapy independent of tumor type on the basis of DNA sequencing detection of oncogenes. Dr. Flaherty joined the NCI Board of Scientific Advisors in 2018 (chair 2021-23), AACR Board of Directors in 2019, and was elected President of AACR in 2025. Dr. Flaherty co-founded Loxo Oncology in 2013 and Scorpion Therapeutics in 2020, serving on the board of directors through the acquisitions of both by Eli Lilly in 2019 and 2025. He co-founded X4 Pharmaceuticals, privately held Strata Oncology (2015), Apricity Oncology (2017), Gyges Oncology (2020), Khora Therapeutics (2024), and Monimoi Therapeutics (2025).

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Jennifer R. Grandis, MD
Distinguished Professor & Principal Investigator
University of California San Francisco (UCSF), USA

Dr. Jennifer R. Grandis is a physician scientist who is interested in the impact of gender on career development in medicine and science. Her cancer research is focused on elucidating and targeting key signaling pathways and genomic alterations in head and neck cancer with the goals of enabling precision medicine studies. She has leveraged her access to head and neck cancer patients and their biospecimens to optimize translational research studies that include developing novel therapies in the laboratory for clinical application as well as generating and interrogating relevant preclinical models to determine the underlying mechanism of clinical findings. In her institutional roles at the University of Pittsburgh and since 2015, at UCSF, she has facilitated collaborations between clinicians and investigators with an emphasis of developing a robust research infrastructure to support clinical and translational cancer studies. She has published over 400 papers in the peer-reviewed literature and been continuously funded by the NIH since joining the faculty in 1993. Dr. Grandis is an elected member of the American Society for Clinical Investigation, the Association of American Physicians and the National Academy of Medicine. She is an American Cancer Society Research Professor.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Matthew J. Hadfield, DO
Associate Professor of Medicine at the Alpert School of Medicine
Brown University, USA

Matthew Hadfield, DO, is an Associate Professor of Medicine at the Alpert School of Medicine of Brown University and an attending physician at the Brown University/Legorreta Cancer Center specializing in cutaneous oncology and developmental therapeutics.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sabine Hazan, MD

CEO, Ventura Clinical Trials
CEO, Progenabiome

Dr. Hazan is the CEO of Ventura Clinical Trials, where she has 30+ years' experience leading clinical trials for cutting-edge research on various medical issues and has done over 300 clinical trials for pharmaceutical companies.

She is also the founder & CEO of Progenabiome, a genetic sequencing research laboratory, she leads 61+ studies investigating the role of the gut flora in various diseases.

Through their study exploring the role of the gut flora in COVID-19, Progenabiome became the first lab worldwide to detect SARS-CoV-2 from Patient Fecal Samples by Whole Genome Sequencing and the lab that showed a susceptibility marker for COVID in the microbiome.

Dr. Hazan is mastering Familial fecal transplant and Refloralization showing hope for kids with Autism. She created The Malibu Microbiome meeting and the biome squad where physicians and scientists who perform fecal transplant or deal with the microbiome can discuss their findings freely.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sean Khozin, MD, MPH

CEO
Roundtable on Cancer and Project Data Sphere, USA

Dr. Sean Khozin is a physician-scientist and oncologist, internationally recognized for his leadership in leveraging data science, AI, machine learning, and real-world evidence to drive innovation in biomedical research, with a particular emphasis on oncology drug development.

A founding member of the FDA Oncology Center of Excellence, Dr. Khozin escaped the revolution in Iran and found his way to the United States, harmonizing his passion for patients and data into a career spanning startups, the FDA, Johnson & Johnson, and pioneering AI ventures, all while pursuing his love of music.

His work bridges the gap between cutting-edge technology and clinical care, with a focus on harnessing advanced analytics and AI to accelerate drug discovery, optimize therapeutic development, and improve patient outcomes.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Razelle Kurzrock, MD, FACP

Chief Medical Officer, WIN Consortium
Professor, Medical College of Wisconsin, USA

Razelle (Ray) Kurzrock, MD is a world-renowned physician/scientist leader in precision medicine as well as in novel cancer therapeutics development.

She is recognized for founding, developing and chairing one of the largest Phase I clinical trial departments globally while at the University of Texas MD Anderson Cancer Center; the central theme of the department was a personalized medicine strategy. She led key early- or later-phase trials of 8 drugs that led to FDA approval. She is also one of the pioneering trialists of the WINTHER precision medicine international (five countries) trial focusing, for the first time, on transcriptomics in addition to genomics. This trial was the signature study of the Worldwide Innovative Network (WIN) consortium (published Nature Medicine, 2019). She also developed and pioneered some of the first tumor-agnostic cancer clinical trials and is recognized as having led the way for the molecular reclassification of cancer.

During her time at the University of California San Diego Health, Dr. Kurzrock's charge was founding and leading the Center for Personalized Cancer Therapy as well as the Experimental Therapeutics program, and she also founded a Rare Tumor Clinic focused on precision medicine. The signature study of the center was the I-PREDICT study (Nature Medicine, 2019) that gave, for the first time, individualized N-of-1 matched combination therapies to patients with lethal malignancies, potentially transforming the entire paradigm for cancer treatment. This work was presented at the Nobel Symposium, Karolinska Institute, Stockholm, Sweden in September 2023.

Dr. Kurzrock has performed key early-phase studies of 8 drugs that have gone on to FDA approval. Many of her studies have also been incorporated into NCCN guidelines for payors. Dr. Kurzrock also served on the Board of Directors and was elected to the Executive Committee for NCCN. She has also served as Chair of the Early Therapeutics and Rare Cancers Committee,

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

for NCI/SWOG, one of the largest clinical trial cooperative groups in the world; in this capacity, she was a PI of the DART trial that provided dual immunotherapy for patients with rare/ultra-rare cancers; the trial open at >1000 sites, accrued ~800 patients, resulted in multiple NCCN guideline changes and was recognized with the NCI Director's Award of Merit.

Dr. Kurzrock's strong record of funding is comprised of ~$125 million in lifetime funding, including ~$50 million in NIH/peer-related research support.

Ray Kurzrock is also an entrepreneur. She is co-founder of CureMatch, and on the Board of CureMetrix, CureMatch, and Xzom.

Dr. Kurzrock is the author of >1,000 peer-reviewed articles on PubMed and holds a highly exceptional Hirsch index (h-index) score of 161, with ~110,000 citations. She has been selected each year since 2019 on the Clarivate Web of Science list of most cited scientists worldwide.

She is listed by Scholar GPS as the #1 person worldwide in Precision Medicine.

She has four children and three dogs and lives with her husband Philip Cohen, MD.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Gillian Lamoury, MD, FRANZCR

Clinical Associate Professor, the University of Sydney
Senior Staff Specialist, Radiation Oncologist at Royal North Shore Hospital
VMO at GenesisCare, the Mater Hospital, Australia

Gillian is a Clinical Associate Professor at the University of Sydney and a Principal Investigator on numerous international, national, and investigator-led clinical trials, including pilot studies investigating novel biomarkers in breast, ovarian, and pancreatic cancer. She is dedicated to patient-centred, evidence-based and, increasingly, AI-augmented care, with interests in Precision Oncology, early cancer detection and screening, and the delivery of high-quality treatment through sophisticated radiation therapy techniques for breast cancer — including a strong interest in pre-operative radiotherapy using innovative technology.

A committed advocate for improved patient care, Gillian was the inaugural Chair of the Breast Interest Group – Faculty of Radiation Oncology Australia (BIG-FRO), is the recent past Chair of the Breast Cancer Advisory Group (Cancer Australia, 2018–2020) and serves on the Clinical Advisory Group for Breast Cancer Network Australia (BCNA). She was delighted to accept a membership position with the WIN Consortium Precision Oncology Screening and Prevention Committee in 2026.

In 2025, she was appointed to the Board of Pratham Australia, a volunteer-run, non-profit charitable organization whose vision is to end the cycle of poverty in India by providing access to education.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sean E. Lawler, PhD
Professor, Department of Pathology and Laboratory Medicine, Legorreta Cancer Center
Brown University, USA

Sean Lawler PhD is an experienced molecular biologist and leads the Brain Cancer Therapy Lab in the Department of Pathology and Laboratory Medicine at Brown University. He is also co-leader of the CNS Cancer Translational Disease Research Group at the Legorreta Cancer Center, and co-Director of the Therapeutic Sciences Graduate Program at Brown University.

Cancers of the brain such as glioblastoma are among the most challenging because of their location within the central nervous system and their highly aggressive nature. Brain tumors affect both adults and children and have a very poor prognosis. The Lawler Lab is developing new approaches to treat all brain cancers. Our major interests are 1) Drug delivery across the blood-brain barrier for cancer treatment in the brain, 2) Harnessing innate immune mechanisms to enable the effective application of immunotherapies for brain cancer, and 3) understanding the effects of a common pathogen, cytomegalovirus, on the growth of the malignant brain tumor glioblastoma.

Our goal is to design new and effective next generation therapeutic approaches for these extremely challenging diseases.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Ning Liao, MD, PhD
Director, Department of Breast Cancer, Cancer Center
Guangdong Provincial People's Hospital, China

Liao Ning holds a Doctor of Medicine degree and is a doctoral supervisor. She is the Administrative Director of the Breast Surgery Department at Guangdong Provincial People's Hospital and a member of the Oncology Drug Group of the Expert Committee on Rational Drug Use under the National Health Commission, with over 25 years of clinical experience in breast cancer. As the principal investigator, she has undertaken multiple projects funded by The National Natural Science Foundation of China and the National Natural Science Foundation for Young Scholars and has published numerous SCI-indexed publications in authoritative international journals.

In 2014, she furthered her studies at the MD Anderson Cancer Center in the United States (ranked first in the nation for ten consecutive years). She serves as the Chairperson of the Sino-American Breast Cancer Summit Forum and won the gold award for the "Health Communication Famous Medical Team" in 2021. In June 2023, she participated in the program "AI Future Night." In 2025, at the 10th Global Chinese Breast Cancer Organization Alliance Conference, she proposed aiming for the cure of breast cancer and emphasized the influence of genes on breast cancer. She specializes in minimally invasive surgery for breast tumors, as well as the early diagnosis and comprehensive treatment of breast cancer.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sewanti A. Limaye, MD, PhD
Vice Chair, WIN Consortium
Member, Scientific and Advisory Board, WIN Consortium
Director Medical & Precision Oncology, Director Clinical and Translational Oncology Research
Sir H. N. Reliance Foundation Hospital & Research Centre, Mumbai, India

Dr. Sewanti Limaye is a medical and precision oncologist with over 20 years of experience in the management of solid tumors, Precision Oncology, clinical research, and early drug development. She currently serves as Director of Medical & Precision Oncology and Director of Clinical & Translational Oncology Research at Sir H. N. Reliance Foundation Hospital & Research Centre, Mumbai, where she leads the Precision Cancer Centre and Initiative. Dr. Limaye has held academic and clinical positions at leading institutions in the United States, including Columbia University Medical Center/New York-Presbyterian Hospital and Dana-Farber Cancer Institute/Brigham and Women's Hospital. She has expertise across solid tumors, with particular interests in thoracic, head and neck, gastrointestinal oncology, Precision Oncology, and early-phase clinical trials. She holds a master's degree in Patient-Oriented Research with a focus on Cancer Genomics and Biostatistics from Columbia University's Mailman School of Public Health. A strong advocate for Precision Oncology and collaborative cancer research, Dr. Limaye has led several national and international clinical trials and contributed to multidisciplinary molecular tumor board initiatives. She is Vice-Chair of the WIN Consortium in Precision Oncology and Co-Chair of the WIN Molecular Tumor Board. She has also co-founded several national and international oncology initiatives, including the Precision Oncology Alliance of India, Clinical Trial Consortium of India, IMPACT India International Molecular Tumor Board, and Liquid Biopsy Foundation India (LIBRA), and is the Founder of Nova Precision AI. Dr. Limaye has been recognized nationally and internationally for her contributions to cancer care, precision medicine, clinical research, and patient care. Her recent recognitions include the 2026 Visionary Clinician Award in Precision Medicine at APT-2026 – International Conference & Workshops, IIT Bombay; the 2026 Indo-American Cancer Association (IACA) Award for Outstanding Contributions to Cancer Care in India and Pioneering Contributions to Clinical

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Cancer Research; and the 2026 National Oncologist of the Year Award at the Silver Financial Express Healthcare Summit & Awards. She was also honored as Oncologist of the Year – National at the ET Health World Healthcare Awards 2024 Dr. Limaye is an active contributor to the international oncology community, having been an invited speaker at the United Nations Headquarters in New York, ASCO, AACR, IIT Bombay, and other leading national and international forums. Her recent academic leadership includes presentations at ESMO, ELCC, WCLC, and IASLC, including a keynote on "Biomarkers for ADCs: IHC and Beyond" at the World Conference on Lung Cancer. With 68 PubMed-indexed peer-reviewed publications and contributions to multiple book chapters, including a 2026 Springer chapter on investigational biomarkers and future therapies in gastric cancer, Dr. Limaye continues to integrate Precision Oncology, cancer genomics, liquid biopsy, translational research, clinical trials, immunotherapy, targeted therapy, and artificial intelligence to advance personalized cancer care and accelerate the translation of innovative research into clinical practice. Dr. Limaye's overarching vision is to advance precision cancer care, clinical research, and innovation while building collaborative platforms that bring cutting-edge oncology closer to patients in India and globally.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Yves A. Lussier, MD, FAMIA, FACMI, FIAHSI
Chair, Scientific and Advisory Board, WIN Consortium
Professor and Chair, Department of Biomedical Informatics
University of Utah, USA

Dr. Yves A. Lussier, MD, FAMIA, FACMI, FIAHSI, is Professor and Chair of Biomedical Informatics at the University of Utah School of Medicine and an internationally recognized leader in translational bioinformatics, precision medicine, and AI-enabled healthcare transformation.

A physician-scientist and professional engineer, Dr. Lussier has held leadership roles at Columbia University, The University of Chicago, the University of Arizona, and the University of Illinois at Chicago. Across these institutions, he conceived and built foundational biomedical informatics infrastructures, including enterprise clinical data warehouses, translational informatics platforms, and clinical informatics teams. He also contributed to major Canadian academic hospital mergers integrating research, clinical care, and operational strategy.

His research spans computational genomics, systems biology, AI-driven precision medicine, and single-subject analytics. Dr. Lussier has authored more than 200 scientific publications, secured approximately $200 million in research funding, delivered 30 international keynote lectures, and mentored over 100 clinician-scientists, PhD trainees, and informatics leaders. His pioneering work includes genome-by-environment therapeutic classifiers, computational drug repurposing validated in clinical trials, and N-of-1 transcriptomic analytics for rare disease discovery and precision therapeutics. He was among a select group of U.S. academic leaders invited to the White House Precision Medicine Summit.

Dr. Lussier co-founded Purkinje.com, an AI-enabled pen-computing electronic medical record platform deployed in more than 4,000 clinics over three decades, and conceived Vigilens, a clinical decision support platform continuously operational at New York-Presbyterian/Columbia for over 20 years. He also co-founded and chaired major scientific conferences including the AMIA Summit and the Translational Bioinformatics Conference.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sendurai A. Mani, MS, PhD

Associate Director for Translational Oncology
Legorreta Cancer Center Dean's Chair of Translational Oncology Director
Translational Research Disease Groups Director, Stem Cell Center
Professor of Pathology and Laboratory Medicine
Legorreta Cancer Center, Brown University, USA

Sendurai A. Mani is a Professor in the Department of Pathology and Laboratory Medicine at Brown University. He is also the Associate Director of Translational Oncology at Brown University Legorreta Cancer Center. Dr. Mani earned a Ph.D. from The Indian Institute of Science, Bangalore, India and then did postdoctoral work with Dr. Robert A. Weinberg at the Whitehead Institute/Massachusetts Institute of Technology (MIT), Cambridge, Massachusetts, USA. He then joined the University of Texas MD Anderson Cancer Center, Houston, Texas as an Assistant Professor in December of 2007 and was promoted to Associate Professor with tenure in 2013. In late 2022, Dr. Mani joined Brown University Legorreta Cancer Center as a professor and Associate Director of Translational Oncology. Dr. Mani has received numerous prizes and awards for his research, including a Jimmy V foundation's V-Scholar Award and The American Cancer Society Research Scholar award. Dr. Mani's original finding demonstrating the cancer cells acquire stem cell properties by activating latent embryonic epithelial-mesenchymal transition (EMT) program provided the foundation and explanation for the presence of plasticity within the tumor as well as the development of resistance to various treatments.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Cara Mathews, MD, MS
Gynecology Oncologist
Women and Infants Hospital, Brown University, USA

Dr. Cara Mathews is an Associate Professor of Obstetrics and Gynecology at the Warren Alpert Medical School of Brown University and a practicing gynecologic oncologist at Women & Infants Hospital of Rhode Island and the Legorreta Cancer Center. She earned her undergraduate and medical degrees from Dartmouth College and Dartmouth Medical School, followed by residency training in Obstetrics and Gynecology at Women & Infants Hospital/Brown University and a fellowship in Gynecologic Oncology at the University of Oklahoma Health Sciences Center. Upon returning to Women & Infants, Dr. Mathews established an early-phase clinical trial program focused on gynecologic and breast cancers, incorporating both NCTN and industry-sponsored studies. She currently serves as Director of Oncology Research and the NRG Oncology Principal Investigator at Women and Infants Hospital.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Peter J. O'Dwyer, MD

Physician, Professor of Medicine
Perelman School of Medicine, University of Pennsylvania, USA
Group Co-Chair, ECOG-ACRIN Cancer Research Group

Peter J. O'Dwyer, MB, BCh, was appointed Group Co-Chair of the ECOG-ACRIN Cancer Research Group (ECOG-ACRIN) in May 2017. A medical oncologist with expertise in gastrointestinal cancers and the development of new cancer treatments, Dr. O'Dwyer is a physician at Penn Medicine in Philadelphia, and a professor of medicine in the Perelman School of Medicine, University of Pennsylvania. He is board-certified in Internal Medicine, Pediatrics, and Medical Oncology.

Within ECOG-ACRIN, Dr. O'Dwyer co-leads the organization's governance. He also co-chairs the landmark precision medicine cancer trial known as NCI-MATCH (National Cancer Institute-Molecular Analysis for Therapy Choice), its successor trial ComboMATCH, and is a member of several other scientific committees. He co-directs the Cancer Control and Outcomes Program and is co-principal investigator for two grants from the National Cancer Institute: the NCI Community Oncology Research Program (NCORP) Research Base and the National Clinical Trials Network (NCTN). He chaired the ECOG-ACRIN Gastrointestinal Cancer Committee until 2020. In addition, he is funded by FDA for ECOG-ACRIN Real-World Evidence studies in cancer. Dr. O'Dwyer has published more than 400 peer-reviewed scientific papers in the medical literature and participates in numerous national and international organizations. He is focused now in cancer research policy and implementation, especially as this relates to correlative science in association with clinical trials, novel approaches to genomic trials, and implementation of AI to facilitate this research. He serves as the Chair of the PrECOG, LLC Board of Managers, and as President of the ECOG Research and Education Foundation.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Amir Onn, MD

Member, WIN Consortium
Chief Medical Officer, Healthy Longevity Center
Sheba Medical Center, Tel Hashomer
Associate Professor of Medicine, Tel Aviv University, Israel

Dr. Amir Onn earned his medical degree from the Hadassah Hebrew University School of Medicine in Jerusalem, Israel. He completed his residency in internal medicine and fellowship in pulmonary medicine at Tel Aviv Sourasky Medical Center, affiliated with Tel Aviv University. He then completed post-doctoral and clinical fellowships in interventional pulmonary oncology at The University of Texas MD Anderson Cancer Center (MDACC) in Houston, Texas. Following his training, Dr. Onn joined the faculty of MDACC's Department of Pulmonary Medicine as an Assistant Professor, receiving a prestigious Physician-Scientist grant.

Upon returning to Israel in 2006, Dr. Onn established the Pulmonary Oncology Service at Sheba Medical Center—the largest medical center in Israel—and subsequently served as Head of the Institute of Pulmonary Medicine from 2014 to 2025. Under his leadership, the institute expanded significantly to include advanced clinical services in lung transplantation (including ECMO management during the COVID-19 pandemic), pulmonary hypertension diagnosis, management and research, interventional pulmonology, sleep and non-invasive ventilation, and bronchiectasis and airway diseases.

In 2026, he was appointed Chief Medical Officer of the Healthy Longevity Center at Sheba, where he leads medical screening initiatives and advances the center's growth.

In 2011, Dr. Onn founded the annual Israeli Multidisciplinary Conference on Lung Cancer, which he continues to chair as Israel's premier gathering for clinicians and researchers across all lung cancer specialties. He also serves as a mentor to medical students from underprivileged communities in Israel.

Dr. Onn is a co-founder of the Worldwide Innovative Networking (WIN) Consortium in

personalized cancer medicine, established in 2010. He previously served on its Executive Committee and as Global Coordinator for Tissue Procurement and Processing for WIN clinical trials.

Dr. Onn's primary research interests focus on lung cancer biomarkers and genomics. He has authored 128 scientific publications listed in the Tel Aviv University Research Portal, including over 100 peer-reviewed journal articles, review articles, and book chapters.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Corrie Painter, PhD

Venture Partner at Even One Ventures, USA

Corrie Painter, PhD, is a Partner at Even One Ventures, where she builds companies focused on personalized medicines and technologies. She brings experience as a cancer research scientist, biotech operator, and patient advocate. At the Broad Institute, she helped build a national patient-partnered clinico-genomic data platform and led efforts to openly share those data to accelerate discovery. She later helped build Precede Biosciences from its earliest days through its Series B financing, working across translational science, partnerships, and company building.

Painter earned her PhD in biochemistry and trained in cancer immunology. She is an angiosarcoma survivor and co-founder and President of Angiosarcoma Awareness, where she works to accelerate research and expand options for patients with rare cancers.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Jim Palma

Member, WIN Consortium
CEO, TargetCancer Foundation, USA

Jim Palma is Chief Executive Officer of TargetCancer Foundation. Since joining TargetCancer Foundation (TCF) in 2010, Jim has overseen its growth from a small, Boston-based start-up to a nationally recognized foundation driving comprehensive rare cancer research programs and patient support services.

Jim is a member of the Board of Directors of the National Organization for Rare Disorders (NORD) and is a founding Co-Chair of the NORD Rare Cancers Coalition. In addition, Jim serves on the Advisory Board of the Transformative Rare Cancer Initiative (TRACER) at the Fred Hutchinson Cancer Center and is a Steering Committee member at the Global Cholangiocarcinoma Alliance. Jim also serves as a member of the Programmatic Panel of the US Army Medical Research and Development Command Rare Cancers Research Program. Prior to joining TargetCancer Foundation, Jim spent eleven years at Fidelity Investments in Boston, MA. Jim completed studies at the Institute for Nonprofit Management and Leadership at the Questrom School of Business at Boston University and received his B.A. from Loyola University Maryland.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Richard Pazdur, MD
Former Director of the FDA Oncology Center of Excellence
Pazdur Direct, LLC, USA

Dr. Richard Pazdur is a leading figure in oncology drug regulation and the former Director of both the FDA's Oncology Center of Excellence and the Center for Drug Evaluation and Research. Over his 26-year FDA career, he played a transformative role in modern cancer drug development, working closely with academic oncologists, industry partners, and regulatory scientists to streamline and strengthen the approval of innovative therapies.

At the FDA, Dr. Pazdur built one of the most effective and nationally respected oncology regulatory centers. He championed an integrated review model that improved cross-center coordination and accelerated the evaluation of novel cancer treatments. His leadership not only advanced scientific rigor but also created clearer, more predictable pathways for oncology drug development. A mentor to generations of regulators, he significantly influenced how cancer therapies are reviewed and how regulatory science evolves to meet emerging medical needs.

Before joining the FDA in 1999, Dr. Pazdur was a professor of medicine at The University of Texas M.D. Anderson Cancer Center. His academic career included leadership roles on major hospital committees, service on national and international advisory groups, and contributions to several editorial boards.

Dr. Pazdur received his bachelor's degree from Northwestern University and his M.D. from Loyola Stritch School of Medicine, followed by clinical training at Rush-Presbyterian-St. Luke's Medical Center and the University of Chicago.

A highly published physician, he is the author of more than 800 articles, book chapters, and abstracts, as well as two oncology textbooks. His contributions have earned him recognition on Fortune's '50 World's Greatest Leaders,' The Bloomberg 50, OncLive's 'Giants of Cancer Care,' and numerous awards from major professional societies.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Kornelia Polyak, MD, PhD
Professor of Medicine
Dana-Farber Cancer Institute, Harvard Medical School, USA

Kornelia Polyak, MD, PhD, is Professor of Medicine at Dana-Farber Cancer Institute, Harvard Medical School, and a co-leader of the Harvard Cancer Consortium Cancer Cell Biology Program and SPORE in Breast Cancer. Dr. Polyak is an internationally recognized leader of breast cancer research. Her laboratory is dedicated to the study of human breast cancer with the goal of dissecting tumor evolution and using this information to improve the clinical management of breast cancer patients. Main areas of interests are: (1) breast cancer risk prediction and prevention, (2) drivers of tumor evolution from pre-invasive to metastatic treatment-resistant disease, and (3) novel therapeutic targets and rational combination therapies. Dr. Polyak has received numerous awards including the Paul Marks Prize for Cancer Research, the AACR Outstanding Investigator Award for Breast Cancer Research, the 14th Rosalind E. Franklin Award for Women in Science, and the 2023 AACR Distinguished Lectureship in Breast Cancer Research award. She is also an American Cancer Society Research Professor, NCI Outstanding Investigator, and a Weil-Cornell Distinguished Alumna Award recipient. Dr. Polyak is a fellow of the AAAS and the AACR Academy, member of the National Academy of Sciences, National Academy of Medicine, and American Academy of Arts and Sciences, and outside member of the Hungarian National Academy.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Harpreet Ram, MBA

Professor of Medicine
EVR Consulting LLC, USA

Harpreet Ram, MBA, is the President and Founder of EVR Consulting, a strategic consulting firm established in 2011 that helps biotechnology and pharmaceutical organizations strengthen their Global Medical Affairs capabilities and achieve operational excellence. She brings 25+ years of pharma experience supporting organizations of all sizes, with 15 years dedicated specifically to the managed access space.

Harpreet specializes in designing and implementing enterprise-wide organizational frameworks that enable proactive, cross-functional planning and execution of Managed Access Programs (MAPs). She has supported the development and implementation of global programs, helping organizations navigate the strategic, governance, operational, regulatory, and patient-access considerations required to deliver investigational and pre-approval therapies responsibly and sustainably.

Harpreet is also the Founder and Executive Director of GARDaccess, a multi-stakeholder initiative focused on expanding sustainable access to rare-disease therapies in low- and middle-income countries. Across her consulting and advocacy work, she is recognized for bringing together diverse stakeholders, translating complex access challenges into practical solutions, and building scalable frameworks that place patient needs at the center of organizational decision-making.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Sana Raoof, MD, PhD

Thoracic radiation oncologist
Co-director of the Legorreta Cancer Center's screening initiative
Legorreta Cancer Center at Brown University, USA

Sana Raoof, MD, PhD, is a thoracic radiation oncologist and co-director of the Legorreta Cancer Center's screening initiative. She is also an assistant professor in Biostatistics at the Brown School of Public Health. Dr. Raoof's research focuses on improving blood-based methods for detecting cancer earlier. She also leads a Brown University project to expand lung cancer screening for people who are not currently eligible and evaluate whether newer blood-based tools can detect cancer earlier in this group. Dr. Raoof serves on the American Cancer Society Guideline Development Group, where she contributes to national cancer screening recommendations, and the National Multi Cancer Early Detection Consortium. She was named Forbes 30 under 30 for Healthcare in 2020.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Brenda Rubenstein, PhD
Krieble Professor of Chemistry and Professor of Physics at Brown University, USA
Director of Data Science

I am a computational/theoretical chemist interested in three broad areas: 1) developing new quantum and statistical mechanics techniques for modeling strongly correlated molecules and quantum materials in the post-DFT era [Electronic Structure]; 2) designing new alternative (molecular, quantum, and cellular) computing techniques and algorithms [Alternative Computing]; and 3) using biophysical and statistical methods to predict the evolution of proteins, and by extension, drugs that can target them [Biophysics]. To date, my group has developed a wide variety of new, more accurate and efficient quantum Monte Carlo methods; demonstrated how simple organic molecules can be used to store information and compute; and designed physics- and ML-based computational tools for rapidly predicting the structures and resultant phenotypes of proteins of relevance to medicine and basic science. Other past and recurring research interests include problems in computational biology, neuroscience, data science, information theory, computational linear algebra, stochastics, and condensed matter physics. We frequently collaborate with experimentalists and industry to realize our theories regarding quantum materials, biophysics, and alternative computing strategies.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Jens Rueter, MD

Member, Scientific Advisory Board, WIN Consortium
Chief Medical Officer, The Jackson Laboratory
Medical Director for the Maine Cancer Genomics Initiative (MCGI), USA

Dr. Rueter is the Chief Medical Officer at The Jackson Laboratory, the Medical Director for the MCGI and the Associate Director for Translational Education at the JAX Cancer Center. In that role, he is particularly interested in enabling access to Precision Oncology in rural communities.

Since 2010, Dr. Rueter has practiced and continues to practice as a hematologist/oncologist at Northern Light Cancer Institute. He joined JAX in August 2016 to build and lead the Maine Cancer Genomics Initiative. After graduating from medical school in Berlin, Germany, Rueter completed his residency in internal medicine at Tulane University and fellowship training in hematology/oncology at the University of Pennsylvania.

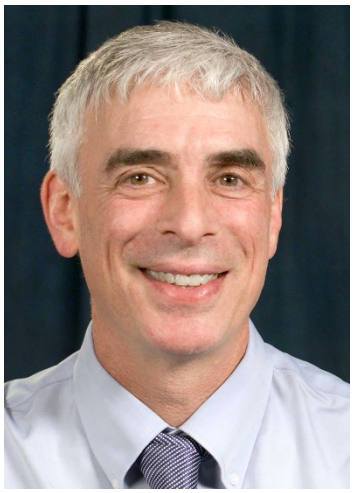
SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Eytan Ruppin, MD, PhD

Past Member, Scientific and Advisory Board, WIN Consortium
Deputy Director, Translational Research Institute Director, Integrative Data Sciences
Cedars-Sinai, USA

Eytan Ruppin received his M.D. and Ph.D. from Tel-Aviv University where he has served as a professor of Computer Science & Medicine since 1995, conducting computational multi-disciplinary research spanning computational neuroscience, natural language processing, machine learning and systems biology. In 2014 he joined the University of Maryland as director of its center for bioinformatics and computational biology (CBCB). In 2018 he moved to the NCI where he founded and is chief of its Cancer Data Science Lab (CDSL, ccr.cancer.gov/cancer-data-science-laboratory), before moving recently to the Translational Research Institute and the Department of Surgery in Cedars-Sinai. His research is focused on developing new computational approaches for advancing Precision Oncology, leading to a few ongoing clinical trials. Eytan is a fellow of the International Society for Computational Biology (ISCB), a recipient of the NCI Director award for his work on Precision Oncology (2022), the DeLano award for computational biosciences for his work on synthetic lethality (2023) and the NIH director award for developing new computational paradigms for Precision Oncology (2024). He is a member of GSK Oncology, ProCan and past WIN consortium scientific advisory boards and a co-founder of a few precision medicine startup companies.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty



Howard P. Safran, MD
Chief, Hematology Oncology
Brown University Health Cancer Institute, USA

Howard P. Safran, MD, is the chief of hematology/oncology at the Brown University Health Cancer Institute at Rhode Island, The Miriam, Newport, and Saint Anne's hospitals, and the medical director for the Brown University Oncology Group. Dr. Safran is a professor of medicine at The Warren Alpert Medical School of Brown University and a nationally recognized leader in the development of new treatments for upper GI cancers.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Ilyas Sahin, MD

Medical Oncologist
Massachusetts General Hospital, USA

Dr. Ilyas Sahin is a medical oncologist at Massachusetts General Hospital specializing in hepatocellular carcinoma and early-phase clinical trials. He is a member of the Center for Gastrointestinal Cancers and the Termeer Center for Targeted Therapies, where he serves as principal investigator for HCC studies ranging from first-in-human trials to later-phase therapeutic studies. His research focuses on targeted therapies, immunotherapy combinations, treatment resistance, and translational drug development in liver cancer.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Bradley J. Sander
Chief Executive Officer
p53-Therapeutics, Inc., USA

Bradley Sander is an accomplished executive, entrepreneur, and strategic operator with a proven track record of building and scaling companies, driving revenue growth, and maximizing enterprise value. He brings extensive experience across business development, product development, marketing, sales, and operations, with a strong ability to lead cross-functional teams and translate strategic vision into execution.

He is a senior commercial executive with experience building and scaling revenue platforms across technology and connected health. His background includes commercialization strategy, and operational execution, with a focus on translating growth initiatives into measurable financial outcomes. He has led large-scale go-to-market and channel transformations, including work at AT&T that contributed to more than $15 billion in revenue. At Disney, he built and scaled a national operating strategy that reduced costs by 75% while expanding market reach more than 40x. Earlier in his career, he led an international product launch across 22 countries, generating over $700 million in revenue and establishing repeatable commercialization processes.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Giselle L. Saulnier Sholler, MD, MSc

Division Chief, Pediatric Hematology/Oncology/BMT Penn State Hershey Children's Hospital
Professor, Pediatrics and Pharmacology, Penn State University, College of Medicine
Founder and chair of the Beat Childhood Cancer Research Consortium, USA

Dr. Sholler has focused on changing outcomes for children with cancer through creating new therapeutic options. Her lab's preclinical work focuses on precision medicine, understanding the biology of each cancer to designing novel therapeutic trials, overseeing regulatory development and working with the FDA and IRB's needed in drug development to initiate these studies as Chair of the Beat Childhood Cancer Consortium. As the Division Chief of Pediatric Hematology/Oncology/BMT at Penn State Health Children's Hospital she sees the impact of our research daily treating children on our clinical trials.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Jacob Scott, MD, DPhil

Principal Investigator,
Jacob Scott Laboratory Translational Hematology and Oncology Research and Radiation Oncology Dept. Associate Professor, School of Medicine, Case Western Reserve University
Associate Director for Data Science, Case Comprehensive Cancer Center
Adjunct Associate Professor, Department of Physics, Case Western Reserve University, USA

Jacob G. Scott, MD (MED '08), professor of medicine and physics at the College of Arts and Sciences and School of Medicine at CWRU; and a board-certified clinical (radiation) oncologist and established NIH investigator working at the Cleveland Clinic. He is a veteran of the US Navy submarine force turned academic physician-scientist. Scott is also co-director of the Center for AI Enabling Discovery in Disease Biology (AID2B) at CWRU and associate director for data sciences for the Case Comprehensive Cancer Center.

His lab pursues research decomposing the complexity of cancer using mathematical modeling and the biological and clinical validation of these models. With an educational background spanning physics, medicine, mathematics and engineering, Scott has a unique perspective on cancer and systems biology. He communicates and collaborates with professionals across many disciplines.

Scott also works in the space of mathematical radiation biology, where he's collaborated with partners to pioneer the concepts of the genomic adjusted radiation dose (GARD) and temporally feathered radiation therapy (TFRT). His commercialization efforts have been largely focused in this space. Most of his patents are in radiation planning/radiation genomics, which he's working to bring to the mainstream through industry partnerships.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Attila A. Seyhan, PhD

Director of Translational Oncology Operations & Director of Translational Oncology Shared Resources
Legorreta Cancer Center, Brown University, USA

Attila A. Seyhan, PhD, is the Director of Translational Oncology Operations at Cancer Center at Brown University, the Joint Program in Cancer Biology, Brown University and Lifespan Health System, Warren Alpert Medical School, Brown University.

He is also an Adjunct Associate Professor (Research), Laboratory of Translational Oncology and Experimental Cancer Therapeutics, Department of Pathology and Laboratory Medicine, Warren Alpert Medical School, Brown University, Providence, RI.

He manages and promotes multiple initiatives, including the development of investigator-initiated clinical trials and other translational protocols, protocol writing and manuscript preparation, organization of translational medicine events, support for translational requests for application (RFAs), and grant preparation and submissions.

He also works with industry to follow through on investigator-initiated basic and translational letters of intent (LOIs) and concepts. This work will involve collaboration with clinicians, scientists, regulatory personnel, administrators, tech transfer office staff, the grants management office, institutional review board (IRB), institutional advancement, and external entities.

His most recent position was Director of Translational Medicine Operations, Associate Research Professor, Translational Medicine, Fox Chase Cancer Center / Temple University.

Previously, he was Associate Professor at the Translational Research Institute for Metabolism and Diabetes at Florida Hospital in Orlando, FL, and Adjunct Associate Professor at Sanford Burnham Prebys Medical Discovery Institute, in Orlando (Lake Nona), FL. In addition, he served as a research affiliate in the Department of Chemical Engineering at the Massachusetts Institute of Technology in Boston, MA.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

In addition to academia, Dr. Seyhan has spent significant time in the pharmaceutical and biotechnology industries, having served in leading roles as Senior Principal Scientist at Pfizer Pharmaceuticals in Cambridge, MA., Principal Scientist and Head of Functional Genomics at Wyeth Pharmaceuticals in Collegeville, PA, Senior scientist II at Open Biosystems/Thermo Fisher in Huntsville, AL., Dharmacon/ GE Healthcare in Lafayette, CO., and SomaGenics, Inc., in Santa Cruz, CA.

He won several awards (a doctoral fellowship from NATO (North Atlantic Treaty Organization), Young Investigator Award at the Symposium on RNA Biology III, Pfizer Excellence Awards for contributions towards Biomarkers of Lupus Disease Study).

Dr. Seyhan did his postdoctoral fellowship with Professor Bernard Grandchamp and Carol Beaumont at Xavier Bichat Hospital, the University of Paris VII studying ferritin gene regulation during erythroid differentiation/proliferation on a postdoctoral fellowship from the National Institute of Health and Medical Research of France (INSERM). He did another fellowship with Professor John Burke at the University of Vermont studying hairpin ribozyme biochemistry and their use as RNA therapeutics.

Dr. Seyhan earned his PhD in Biological Sciences from Michigan Technological University (MTU), Houghton, MI on a NATO (North Atlantic Treaty Organization) Science Fellowship and BS degree in Biology from Hacettepe University, Ankara Turkey.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Misty D. Shields, MD, PhD
Assistant Professor of Medicine Adjunct Assistant, Professor of Medical & Molecular Genetics
Indiana University School of Medicine, USA

Misty Dawn Shields, MD, PhD, is a thoracic medical oncologist, assistant professor of clinical medicine, and associate member of experimental and developmental therapeutics at Indiana University School of Medicine and Indiana University Health Physicians.

For Dr. Shields, the fight against lung cancer is personal—she lost her father to small-cell lung cancer (SCLC) when she was 15 years old. As a result, she sought to better understand every piece of bench-to-bedside precision medicine for lung cancer.

Dr. Shields' graduate studies helped to identify predictive biomarkers to pemetrexed in non-SCLC. She also contributed to the discovery of NeuroD1 as a neuroendocrine subtype in SCLC.

Dr. Shields pursued her medical training at UT Houston, including rotations at MD Anderson Cancer Center, an internal medicine residency at Vanderbilt University, hematology/oncology fellowship at H. Lee Moffitt Cancer Center (as chief fellow), and through the FDA-AACR Oncology Educational fellowship.

She joined faculty at Indiana University in August 2022 as a physician scientist with a focus in early phase trials, longitudinal liquid biopsies, and translational studies for novel therapeutics in acquired chemo-resistant SCLC as principal investigator of the Shields laboratory.

Dr. Shields serves on the guideline panel for SCLC with the National Comprehensive Cancer Network, the local leadership board for American Lung Association, and as the clinical trials advocate for IU Simon Comprehensive Cancer Center's End Lung Cancer Now. She is also the founder of Small Cell SMASHERS, a patient advocacy group for SCLC, in partnership with Lungevity Foundation.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Robert W. Sobol, PhD

Dean's Professor of Cancer Research, Department of Pathology and Laboratory Medicine
Warren Alpert Medical School, Brown University
Associate Director for Basic Research & Co-leader of the Cancer Biology Program Legorreta Cancer Center at Brown University
Associate Director, Joint Program in Cancer Biology, Brown Health Cancer Institute
Affiliate, Brown Institute for Translational Science, USA

Dr. Sobol is the Dean's Professor of Cancer Research, Department of Pathology and Laboratory Medicine, Warren Alpert Medical School, Brown University and is the Associate Director for Basic Research and the Co-leader of the Cancer Biology Program of the Legorreta Cancer Center at Brown University. Research in the Sobol lab focuses on the mechanism of base excision repair (BER), PARP and NAD+ metabolism in human cells and the convergent role of these enzymes and pathways in response to chemotherapy and environmental genotoxins/exposures.

Recent efforts have focused on the role of the BER pathway and NAD+ metabolism in the regulation of PARP1/2 activation in response to replication stress, identification of novel targets to overcome PARP-inhibitor resistance and the development of novel biological tools and assays to evaluate PARP-activation, genotoxic stress, and gene-environment interactions and the utilization of these novel tools for the analysis of tumor response, the response to industrial/environmental pollutants, and the discovery of molecules that inhibit cellular DNA repair.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Karan Sood, MBBS, MD, DNB, FIAPC
Inaugural WIN Fellow 2026, WIN Consortium
Assistant Professor
Tata Memorial Hospital, India

Dr. Karan Sood is an Assistant Professor of Medical Oncology at Tata Memorial Hospital, Mumbai. His research interests include cancer genomics, Precision Oncology, and translational research. He established his research foundation at the Johns Hopkins School of Medicine, where he studied the mechanisms of taxane resistance and the development of biomarkers in prostate cancer. Thoracic, genitourinary, hepatobiliary, neurooncology, and molecularly stratified therapies comprise his clinical and academic pursuits. He is a Junior Mentor with the European Hematology Association, served as Editor of the International Journal of Molecular and Immuno Oncology, and is a member of the ASCO TECAG committee. He established the Molecule online educational series to create a nationwide system of structured, practical learning for fellows and residents in medical oncology and chemotherapy. Additionally, he is dedicated to enhancing academic leadership and collaborative oncology networks in India and beyond, while advancing biomarker-driven cancer care.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

David Spetzler, MS, PhD, MBA
President
Caris Life Sciences, USA

Dr. Spetzler joined Caris Life Sciences in August 2009 and has led our R&D and laboratory operations in a multitude of roles. As President, he currently leads the company's Clinical Operations, Research and Development, Information Technology, Bioinformatics and Biopharma Services. As an innovator in molecular science and precision medicine, Dr. Spetzler has a relentless focus on improving patient care. He has led the development of each of the company's clinical offerings, including our launches of clinical Whole Transcriptome Sequencing and Whole Exome Sequencing. Dr. Spetzler oversaw the development of the company's exclusive and unique technology, ADAPT, which is able to measure thousands of protein aberrations and is being used to develop early cancer detection assays, discover novel drug targets and characterize protein differences in each patient's tumor. He also leads the ongoing development of the company's proprietary AI platform to create and validate dozens of machine learning signatures, called Next Generation Profiling™ (NGP), thus providing the most in-depth and exclusive analysis and interpretation using the most comprehensive suite of clinical offerings available to cancer patients today. The Company is preparing to launch and continues to develop dozens of unique proprietary, machine learning signatures unlocked from a decade of testing patients and accumulating outcomes data to improve cancer diagnosis and therapeutic guidance never before seen.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Vivek Subbiah, MD

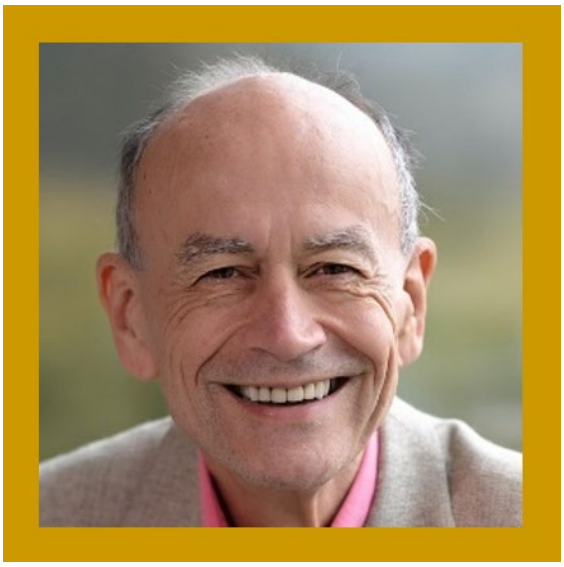
Member, Scientific and Advisory Board, WIN Consortium
Associate Director for Drug Development and Precision Oncology Jeffrey and Christina Bird Professor and Professor of Pediatrics (Hematology/Oncology)
Stanford Cancer Center, USA

Vivek Subbiah, MD, is a medical oncologist and early-phase clinical trialist with dual training in adult and pediatric oncology and 15+ years of leadership in oncology drug development. He holds the Jeffrey and Christina Bird Endowed Chair and is Professor of Medicine at Stanford University School of Medicine, Associate Director for Drug Development and Precision Oncology at Stanford Cancer Institute, and Executive Medical Director for Novel Therapies and the Clinical Trial Network at Stanford Health Care.

He has served as global Principal Investigator on trials contributing to 12+ FDA approvals across 15 cancer indications, including the tumor-agnostic approvals of dabrafenib/trametinib (ROAR) and selpercatinib and pralsetinib (LIBRETTO-001, ARROW). Across 150+ Phase I/II trials as principal investigator, his work spans targeted therapies, antibody-drug conjugates, immunotherapy, radioligand therapy, degraders, and cell therapies.

Previously held leadership positions at MD Anderson and Sarah Cannon Research Institute, he has published 450+ peer-reviewed articles and mentored 100+ early-career physician-scientists.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty



Thomas Südhof, MD

Avram Goldstein Professor in the School of Medicine, Professor of Neurosurgery and, by courtesy, of Neurology and Neurological Sciences and of Psychiatry and Behavioral Sciences
Nobel Laureate in Physiology or Medicine 2013
Stanford Medicine, USA

Thomas Christian Südhof received his M.D. and doctoral degrees from the Georgia Augusta Universität in Göttingen in 1982. He trained from 1983 to 1986 as a postdoctoral fellow with Drs. Mike Brown and Joe Goldstein at UT Southwestern in Dallas, TX, where he elucidated the domain structure, expression and cholesterol-dependent regulation of the LDL-receptor gene, defining among others the sterol-regulatory element (SRE) in cholesterol-regulated genes. Subsequently, Südhof joined the faculty of UT Southwestern in Dallas, where he was the founding chair of the Department of Neuroscience. Since 2008 Südhof serves as the Avram Goldstein Professor in the School of Medicine of Stanford University.

Südhof's research that was honored by the Nobel prize revealed the molecular mechanisms by which a presynaptic neuron releases neurotransmitters during synaptic transmission, thereby explaining the unparalleled speed and precision of information processing by synapses. Moreover, his studies provided a mechanistic understanding of synaptic plasticity and established a molecular foundation for an understanding of neuropsychiatric and neurodegenerative disorders involving synapses, such as schizophrenia and Alzheimer's disease. Many of the key molecules and mechanisms of action underlying the establishment and function of synapses, such as synaptotagmins, Munc13s, RIMs, complexins, neurexins, and neuroligins, were identified by Südhof's work.

In the last decade, Südhof's work changed directions to focus on the molecular logic guiding synapse formation in the design of neural circuits. Südhof's laboratory has identified key protein interaction networks that mediate synapse assembly and are regulated by neuronal activity. His

work revealed a molecular code in synapse assembly, dictated by gene expression and alternative splicing, that sheds light into how the mechanisms which determine how neural circuits are wired.

Südhof is an elected member of the National Academy of Sciences (NAS) and of Medicine (NAM) of the U.S. and of the Leopoldina Academy of Germany, and an elected foreign member of the Royal Society of the U.K., the Hungarian Academy of Sciences, and the Norwegian Academy of Sciences. Südhof's work was recognized by multiple awards, including the 1997 U.S. National Academy Award in Molecular Biology, the 2004 Bristol-Myers Squibb Award for Distinguished Achievement in Neuroscience Research, the 2013 Lasker~DeBakey Medical Basic Research Award, the 2013 Nobel Prize in Physiology or Medicine, and the 2022 Sherrington Prize Lecture Award.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Daniel Sur, MD, PhD

Inaugural WIN Fellow 2026, WIN Consortium
Consultant in Medical Oncology, Researcher in personalized medicine
Assistant Professor Department of Medical Oncology
Oncology Institute "Prof. Dr. Ion Chiricuță" (IOCN), Romania

Daniel Sur is a medical oncologist and academic at the Oncology Institute "Prof. Dr. Ion Chiricuță" (IOCN) in Cluj-Napoca, Romania, where he manages a broad spectrum of solid tumors with particular depth in gastrointestinal, neuro-, and sarcoma oncology. He also serves as Senior Lecturer at the "Iuliu Hațieganu" University of Medicine and Pharmacy.

His research program centers on early-onset colorectal cancer (EOCRC), the role of tumor-infiltrating lymphocytes (TILs) in colorectal cancer, spatial transcriptomics, and the application of artificial intelligence and machine learning to computational pathology. He directs and contributes to several nationally and internationally funded projects, and is engaged in developing a Precision Oncology program at IOCN.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Gevorg Tamamyan, MD, MSc, PhD, DSc, FASCO

Editor-in-chief, OncoDaily, USA
Chairman & Professor, Department of Hematology & Pediatric Oncology, YSMU
CEO, Immune Oncology Research Institute
President, International Society of Pediatric Oncology (SIOP) Asia Continental Branch
President, Pediatric Oncology East and Mediterranean (POEM) Group
Co-Chair, St. Jude Global EURO Regional Advisory Committee

Gevorg Tamamyan, MD, MSc, PhD, DSc, FASCO, is the Co-Founder and Editor-in-Chief of OncoDaily and CEO of P53 Inc. He is the CEO and co-founder of the Immune Oncology Research Institute (IMMONC). He is a global leader in oncology, health systems strengthening, and international health cooperation, recognized for building bridges between countries and advancing equitable cancer care in resource-limited settings.

Dr. Tamamyan was elected and currently serves as President of two major international pediatric oncology societies — the International Society of Pediatric Oncology (SIOP) Asia Continental Branch, representing pediatric oncology professionals across Asia, and the Pediatric Oncology East & Mediterranean (POEM) Group, uniting 28 countries and 110 cancer centers. He later became Co-Chair of the St. Jude EURO Regional Advisory Committee, encompassing 13 European countries.

At 32, Dr. Tamamyan was appointed Head of the Pediatric Cancer and Blood Disorders Center of Armenia, where he led the consolidation of previously fragmented pediatric oncology and hematology services into a single national center. Under his leadership, the center evolved into a regional reference point, with survival rates reaching up to 80%. He also co-founded a national pediatric cancer charity, City of Smile, ensuring universal coverage for diagnosis and treatment for every child and young adult in Armenia.

Dr. Tamamyan is a Professor and Chairman of Hematology and Pediatric Oncology at Yerevan State Medical University, Chairman of the Board of the Institute of Cancer and Crisis, and adviser to the University's Rector. He previously served as President of the Harvard Club of Armenia. Educated and trained across seven countries in Europe, North America, and Asia, his professional trajectory

spans frontline clinical practice, research, academic leadership, national center executive leadership, global health diplomacy, and institution building.

He is a recipient of numerous awards and distinctions, including Fellow of the American Society of Clinical Oncology (FASCO), ASCO LIFe and IDEA, UICC and IARC Young Leader Awards, the 2026 Global Health Catalyst Distinguished Leader Award, the SLACOM 2026 Award for Global Oncology Leadership by the Latin American and Caribbean Society of Medical Oncology, and the 40 under 40 in Cancer Award.

He is married and has two daughters: Charlize and Letizia.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

John Tarantino, JD

Chief Executive Officer
Iylon Precision Oncology LLC, USA

John Tarantino has been an attorney for 45 years. He served as President of the Rhode Island Bar Association, the Rhode Island Bar Foundation, and the New England Bar Association. He is also a Fellow of many prestigious organizations, including the American College of Trial Lawyers, the International Academy of Trial Lawyers, the International Society of Barristers, the American Board of Trial Advocates and Litigation Counsel of America. He is one of the few attorneys in America who holds a Star ranking in Chambers USA.

John is also the Managing Trustee of the Papitto Opportunity Connection, the Managing Director of the John and Pat Tarantino Charitable Foundation, the Managing Director of Iylon Foundation, and serves as CEO of Iylon Precision Oncology, LLC.

John is a UN Representative for the Economic and Social Council and a Global Advisory Board Member for Forttuna Health & Wellness Councils.

He has given talks in many states and countries, and he has achieved great success and popularity as a speaker.

John's talk, Redemption Stories, was the number one TEDx Talk in the world in 2023 and his talk, "You Only Have One Life… Until You Have Another", was the number one TEDx Talk in the world in 2025. John's most recent talk "The Things We Carry… and the Things We Hold" debuted in May 2026 on Think Network YouTube and has been watched by more than 2 million people in the first 8 weeks. More than 42 million people have watched John's talks.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Bicky Thapa, MD, MS, FACP
Instructor in Medicine
Harvard Medical School
Genitourinary Oncologist
Dana-Farber Cancer Institute, USA

Dr. Bicky Thapa is a faculty member at the Dana-Farber Cancer Institute and an Instructor in Medicine specializing in genitourinary oncology. He completed his medical degree at the Armed Forces Medical College in Pune, India, followed by service as a Medical Officer in the Indian Military. He subsequently trained in internal medicine at Cleveland Clinic Fairview Hospital and completed a hematology-oncology fellowship at the Medical College of Wisconsin, where he also earned a Master of Science in Clinical and Translational Science.

Dr. Thapa's research focuses on integrating genomics, transcriptomics, and epigenomics to advance Precision Oncology for patients with advanced genitourinary malignancies, including prostate, kidney, bladder, testicular, penile, and rare GU cancers. He is actively involved in early-phase clinical trials and is particularly interested in N-of-1 precision medicine approaches to develop personalized treatment strategies and inform novel combination therapies. His work also extends to cancer early detection and prevention, with a focus on bladder cancer and renal lesions.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Apostolia M. Tsimberidou, MD, PhD, MBA, FASCO, FAAAS
Vice Chair, WIN Consortium
Member, Scientific and Advisory Board, WIN Consortium
Professor of Medicine, Department of Investigational Cancer Therapeutics
Katherine Russell Dixie Distinguished Endowed Professor
The University of Texas MD Anderson Cancer Center, USA

Dr. Tsimberidou is a Professor of Medicine in the Department of Investigational Cancer Therapeutics, and a Katherine Russell Dixie Distinguished Endowed Professor at The University of Texas, MD Anderson Cancer Center. She has designed highly innovative clinical trials that have changed the standard of practice of oncology and improved the clinical outcomes of patients with solid tumors and hematologic malignancies. She pioneered the personalized medicine program across tumor types at M.D. Anderson. In 2007, she developed the IMPACT (Initiative for Personalized Medicine and Advanced Cancer Therapy) program and demonstrated that identifying tumor specific molecular aberrations and selecting therapy based on tumor abnormalities of individual patients with advanced cancer is associated with better overall outcomes compared with standard approaches. Her research accomplishments have garnered extensive media coverage by ASCO, The Economist, the Wall Street Journal, the Bloomberg report and the Boston Globe. She has published over 380 articles in high-impact peer-reviewed journals and book chapters and co-edited major textbooks in Oncology. She has been invited to present her work at numerous conferences. She has received prestigious awards, and she was appointed to serve on several committees at ASCO and the Worldwide Innovative Network (WIN) consortium in Precision Medicine. She is a board member of the Coalition for Personalized Medicine. Dr Tsimberidou is a Fellow of ASCO (FASCO) and a Fellow of the American Association for the Advancement of Science (AAAS) for "seminal contributions to instantiating and implementing Precision Oncology through innovative therapeutic approaches, patient care, and education aimed at improving clinical outcomes across tumor types".

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Jeffrey R. Waldron, MBA

Executive Director
Sherborn Consulting Group, USA

Jeff advises global healthcare organizations on precision medicine implementation. His forte is creating collaborative alliances to improve patient outcomes, implement advanced therapies, and lower healthcare costs . . . far more effectively than can be achieved by individual organizations.

In 2024, Jeff was recruited by Massachusetts General Hospital to head their newly launched Bipolar Action Network encompassing nine major U.S. academic medical centers. The model is a leaning health network with a state-of-the-art patient registry integrating real time measurement of patient outcomes to improve standard of care and support research on new therapies.

Previously, Jeff was Executive Director of the Personalized Medicine Connective, an oncology precision medicine start-up firm developing partnerships to drive the adoption of personalized medicine. In this role, he built a 700-collaborator including network research hospitals, pharmacy schools, patient foundations, clinical investigators; pharma and diagnostics firms, payors, and regulatory leaders from the FDA and NIH.

Prior to the PM Connective, Jeff worked for the President of Aetna heading strategic, operational, and financial planning for the enterprise. He subsequently led initiatives in three subsidiaries . . . Provider Contacting, Care Management, and Pharmacy . . . where he reported to the Clinical Head.

Jeff holds a BS from Brown University and an MBA from the Wharton School. He's on the Advisory Boards of two cancer foundations . . . Inspire2Live in Amsterdam and AnCan in Arizona.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Hongxia Wang, MD, PhD

Chief, Department of Medical Oncology
Fudan University Shanghai Cancer Center, China

Professor Hongxia Wang is a distinguished medical oncologist who serves as the Director of the Medical Oncology Department at Shanghai Cancer Center, Fudan University, and holds dual leadership roles as Deputy Director of the Phase I Clinical Research Center and Director of the Cell and Gene Therapy Center. Recognized for her significant contributions to precision medicine and oncology, Professor Wang has dedicated her career to the development of novel anti-tumor agents, translational research, and precision targeted therapies.

In her capacity as a key leader in early clinical trials, Professor Wang oversees a multidisciplinary team of over 120 specialists. Her research spans a broad spectrum of early-phase clinical development trials. As a principal investigator, she has directed numerous Phase I–III clinical trials and investigator-initiated studies across a range of solid tumors, evaluating new drugs and therapeutic technologies with the goal of translating scientific insights into clinical practice.

To advance the clinical translation of novel drugs and cell and gene therapies, Professor Wang has also spearheaded translational research initiatives. She has established state-of-the-art platforms including patient-derived organoids, liquid biopsy, and single-cell/multi-omics profiling, and built a translational research team of nearly 100 members. These efforts aim to address gaps in clinical management and foster therapeutic innovation. Her work focuses on understanding sensitivity and resistance to immunotherapies and targeted therapies, optimizing patient selection, refining combination strategies, and identifying predictive biomarkers for cancer recurrence and prognosis.

Professor Wang has authored more than 60 scientific publications in prestigious international journals such as The Lancet, Cell Research, Nature Cell Biology, Cancer Discovery, Science Translational Medicine, Nature Communications, Science Advances, and PNAS. She has filed 20 national patents and one PCT patent. In addition, she has led or participated in six expert consensus projects that provide guidance for Precision Oncology and serves as an editorial board member for several renowned peer-reviewed journals.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Robyn Ward, MBBS, PhD, FRACP, FAHMS, FQA

Deputy Vice-Chancellor (Research and Enterprise) and Senior Vice-President
Monash University, Australia

Professor Robyn Ward AM (MBBS, PhD, FRACP, FAHMS, FQA) is the current Chair of PBAC and is Deputy Vice-Chancellor (Research and Enterprise) and Senior Vice-President of Monash University. An academic leader, cancer researcher and medical oncologist, she has contributed immensely to the development of Commonwealth and NSW health policy, in addition to her pioneering work on the concept of epigenetic changes. Professor Ward was awarded a Member of the Order of Australia (AM) for significant service to medical research and patient care in the field of oncology. She is an ex-officio member of the PBAC Drug Utilisation Subcommittee (DUSC) and the PBAC Economic Subcommittee (ESC), as well as the former Chair of the Medical Services Advisory Committee (MSAC). Professor Ward has extensive experience in health technology assessment and provides expertise in the fields of medical oncology and cancer genetics.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

David E. Wazer, MD

Professor and Chairman of the Department of Radiation Oncology
Warren Alpert Medical School of Brown University, USA
Director
Brown University Health Cancer Institute, USA
Radiation Oncologist-in-Chief
Rhode Island Hospital, Women & Infants' Hospital, The Miriam Hospital, Newport Hospital, Hasbro Children's Hospital, and the Providence VA Medical Center, USA

Dr. David E. Wazer is Professor and Chairman of the Department of Radiation Oncology at the Warren Alpert Medical School of Brown University, Director of the Brown University Health Cancer Institute, and Radiation Oncologist-in-Chief for Rhode Island Hospital, Women & Infants' Hospital, The Miriam Hospital, Newport Hospital, Hasbro Children's Hospital, and the Providence VA Medical Center. Internationally recognized as one of the leading academic radiation oncologists, Dr. Wazer has combined scientific innovation, clinical excellence, educational leadership, and healthcare administration to help shape the evolution of modern radiation oncology over the past four decades. Dr. Wazer's research has focused on the clinical evaluation and implementation of emerging radiation technologies, particularly in breast cancer, brachytherapy, image-guided radiotherapy, stereotactic radiotherapy, health economics, and value-based cancer care. He has authored more than 300 peer-reviewed scientific publications, numerous invited reviews and book chapters, and several landmark textbooks that have become foundational references within the specialty. His scholarship has emphasized not only technological innovation but also rigorous evaluation of treatment efficacy, toxicity, quality of life, and cost-effectiveness, helping define evidence-based standards for contemporary radiation oncology.

SCIENTIFIC/EDUCATIONAL INFORMATION – Faculty

Emile Youssef, MD, PhD
Chief Medical Officer
p53 Therapeutics, USA

Emile Youssef, MD, PhD, is Chief Medical Officer of p53 Therapeutics and a physician-scientist with extensive experience across oncology drug development, translational medicine, medical affairs, and pharmacovigilance. His career spans academic oncology, global pharmaceutical companies, biotechnology, and CRO leadership, with prior roles involving oncology clinical development, medical monitoring, regulatory-facing strategy, and safety oversight. Dr. Youssef completed medical oncology training in Japan and hematology/oncology postdoctoral fellowship experience at The University of Texas MD Anderson Cancer Center, Houston, TX. His work focuses on translating cancer biology into practical clinical development strategies, including target rationale, biomarker thinking, patient selection, IND-enabling planning, and first-in-human execution. He has authored more than 40 peer-reviewed scientific works across oncology, precision medicine, cell and gene therapy, pharmacovigilance, and translational cancer research.

SCIENTIFIC/EDUCATIONAL INFORMATION – Posters Abstracts

1. Artificial Intelligence-Assisted Whole Exome Analysis Reveals Distinct Genomic Patterns Associated with Disease Expression in Familial Breast Cancer

Abdelkarim L and Baskys A
Genomic Medicine and Pharmacogenomics, Western University of Health Sciences, CA

Breast cancer development varies among individuals with familial susceptibility, suggesting that known genetic risk may not fully explain disease expression. We investigated whether phenotype- informed artificial intelligence (AI)-assisted whole-exome sequencing (WES) interpretation could complement algorithmic genomic analysis in identifying biological patterns associated with breast cancer expression.

Controlled-access WES data were obtained from an age-matched familial breast cancer case-control study (dbGaP: phs001044.v1.p1; protocol #24624). One of 35 available samples failed quality control; 34 women comprised breast cancer (n=18) and unaffected (n=16) cohorts. Reads were aligned to GRCh37/hg19 using BWA and variants called with FreeBayes and annotated using SnpEff/SnpSift, dbSNP, gnomAD v2.1.1, ClinVar, and dbNSFP v5.0. Benign/Likely Benign variants and those with allele frequency >5% were excluded. An in-house algorithm prioritized variants using population frequency, functional prediction, conservation, and clinical significance. Genes with DisGeNET gene-disease association (GDA) >0.40 were mapped through UMLS to 13 phenotype categories. A user- defined phenotype category ("Symptom") specified breast cancer, whereas the UMLS- derived Neoplasm category encompassed broader breast tumor associations. Separately, AI-assisted analysis integrated HPO, Ensembl, GenCC, CGD, UniProt, GWAS, gnomAD constraint, and inheritance evidence to generate a Global Disease Impact rank (1-10).

Algorithmic analysis identified two nominal differences. The breast cancer-specific phenotype category differed between cohorts (Welch's p=0.0087; BH q=0.113), whereas the broader Neoplasm category showed only a trend (p=0.09). Endocrine also differed nominally (p=0.0335; BH q=0.218); nonparametric analysis produced concordant results. Using the prespecified GDA >0.40 threshold, secondary analysis identified endocrine- associated findings in 11/18 (61.1%) affected versus 2/16 (12.5%) unaffected women (Fisher's exact p=0.0052). Broad Mental and narrowly defined Bipolar categories showed no differences.

AI-assisted (OpenAI, gpt-5.4) rule-based Global Disease Impact analysis showed that ranks 1-3 carried stronger established gene-disease evidence than ranks 8-10 (median GDA 0.80 vs. 0.455, p=0.0029, Mann-Whitney U test). The analysis was concordant with the endocrine signal and additionally concentrated lower-priority findings at ranks 8-10 in affected women, including *SLC26A2, POC1A, POP1, POLR3B, PDX1, KLHL3,* and *EIF2B5*. These exploratory findings suggest that phenotype-informed AI may complement constrained genomic algorithms by exposing broader gene- phenotype relationships.

Replication in larger independent cohorts and clinical validation are required.

2. Lipid nanoparticle delivery of SFK siRNA and STING agonists for the immune modulation of macrophages in breast cancer

Acosta González J [1], Pamudurthy A [2], Schorr A [1], Raimondo T [1,3-5]
[1] Center for Biomedical Engineering, School of Engineering, Brown University, Providence, RI, USA
[2] Department of Bioengineering, New York Institute of Technology, New York, NY, USA
[3] Division of Biology and Medicine, Brown University, Providence, RI, USA
[4] Legorreta Cancer Center, Brown University, Providence, RI, USA
[5] The Brown RNA Center, Brown University, Providence, RI, USA

The clinical success of immunotherapy in solid tumors has been limited by immunosuppressive tumor microenvironments (TMEs) that inhibit anti-tumor immune responses. In breast cancer, tumor-associated macrophages (TAMs) are abundantly present and constitute about 50% of the tumor mass. These TAMs play a key role in the promotion of tumor progression through the suppression of cytotoxic T cell activity, enhancement of tumor cell invasion, and aiding metastatic spread. In breast cancer, TAM infiltration is strongly associated with poor prognosis and resistance to therapy. This highlights the need for novel strategies that can modulate macrophage function to restore anti-tumor immunity.

Src-family kinases (SFK)play a central role in macrophage-mediated tumor promotion by regulating macrophage migration, focal adhesion dynamics, and cytoskeletal remodeling. Clinical breast cancer data demonstrate that SFK overexpression is correlated with increased macrophage infiltration and lower overall survival. Thus, SFK inhibition is a promising therapeutic strategy to limit macrophage-mediated tumor remodeling. Further, activation of the stimulator of interferon genes (STING)pathway presents a complementary strategy to stimulate anti-tumor macrophage immunity. STING agonists, however, are challenged by poor specificity and limited cellular uptake. Targeting these pathways requires precise intracellular delivery. Here we hypothesize that such delivery can be obtained through the implementation of lipid nanoparticles (LNPs).

In this project, we implemented a sequential lipid screening strategy to optimize an LNP formulation for efficient SFK siRNA delivery to macrophages. In optimized LNPs, SFK siRNA encapsulation (~86% encapsulation efficiency) enhanced macrophage transfection.

Notably, siRNA-LNPs achieved 86% SFK silencing at concentrations as low as 5nM siRNA, with peak silencing at 48 hours post-treatment. Further, LNP-mediated SFK silencing decreased macrophage migration rates by 1.2-fold, in in vitro scratch assays. Macrophage breast cancer co-culture spheroids were used to determine if SFK silencing could enhance LNP tumor penetration. In siRNA- LNP-treated spheroids, we observed enhanced LNP penetration and accumulation into the spheroid when compared to empty vehicle controls. This increase in LNP tumor penetration suggests that SFK silencing modulates the TME, allowing therapeutic penetration.

Dinucleotide and small molecule STING agonists were compared to determine the optimal immune stimulant, with diABZI providing the most robust STING activation in macrophages. STING agonists were also seen to repolarize IL-4-activated macrophages towards an inflammatory phenotype, as determined by increased CD80 expression. STING agonists were encapsulated in our optimized LNPs. STING-LNPs promoted T cell recruitment, accumulation, and penetration into breast cancer spheroids at significantly higher amounts compared to empty vehicles and free agonist conditions. This suggests that the LNP delivery of a STING agonist into tumors could serve as a potent immune activator in naturally immunosuppressive cancers.

3. Effects of the indirubin derivative 6-bromo-indirubin acetoxime (BIA) in pediatric glioma

Adewumi T, Clark J, Anand N, Vaughn-Beaucaire P, Lawler S
Department of Pathology and Laboratory Medicine, Legorreta Cancer Center, Brown University, RI, USA

Pediatric high grade gliomas (pHGG) are aggressive childhood brain tumors with no effective treatments and a median survival rate of just 10 months. These tumors extensively invade normal brain tissue making them impossible to remove surgically. Additionally, the major hurdle in effective drug administration for pHGG is the impenetrability of the blood-brain barrier (BBB) which protects the brain from most therapeutic agents. We previously showed that Bio- acetoxime (BIA), a derivative of the traditional Chinese medicine Indirubin, can cross the BBB and also has antimigratory effects in glioma cells. In this study we evaluated the effects of BIA on pediatric glioma cells using viability assays and flow cytometry to determine the effects of BIA on DIPG36 and SF8628 pHGG cell lines, both harboring the H3.3K27M mutation, in vitro. We also measured effects on barrier function using an in vitro 3D BBB model (BBB spheroids). BIA reduced viability in both DIPG36 and SF8628 cells (IC50= 1.2 μM DIPG36 and 1.8 μM SF8628). Additionally, BIA significantly reduced cell cycle progression and this was associated with arrest in G2/M. BIA treatment was also associated with disruption of endothelial barrier integrity by reducing tight junction protein expression in tumor vasculature. In our BBB spheroids we observed increased uptake of fluorescently labelled dextran indicating that BIA disrupts barrier integrity after 24 hour drug treatment. BIA treatment also reduced endothelial barrier integrity as measured by a decreased transendothelial electrical resistance (TEER).

These data show that BIA may be a promising approach for the treatment of pHGG. Multiple cancer hallmarks (invasion and proliferation) are directly affected by BIA and the fact that it can enter the brain suggests it could reach these invasive cancer cells in their native space.

Furthermore, the potential of BIA to disrupt endothelial cell tight junctions could improve the uptake of other drugs that could not normally enter that brain. This would potentially improve outcomes in pHGG by means of improved drug delivery. In future experiments we will develop an in vivo mouse model to investigate the translational potential of this approach.

4. Reassessing the USP5–OCT4 Axis in Glioblastoma: Multi-Omic Analysis Redirects to CDK4/6

Alfaro S [1] and Seyhan A [2-7]
[1] Brown University, Providence, RI, USA
[2] Department of Pathology and Laboratory Medicine, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[3] Legorreta Cancer Center at Brown University, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[4] The Warren Alpert Medical School, Brown University, Providence, RI, USA
[5] The Joint Program in Cancer Biology, Brown University and Brown University Health, Providence, RI, USA
[6] Laboratory of Translational Oncology and Experimental Cancer Therapeutics, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[7] Hematology-Oncology Division, Department of Medicine, Rhode Island Hospital, Brown University, Providence, RI, USA

Glioblastoma remains the most common malignant primary brain tumor in adults and is characterized by cellular plasticity, recurrence, and poor patient outcomes. Standard of care involves resection followed by radiotherapy and temozolomide.

Despite advances in treatment, prognosis remains poor. Recurrence is thought to be substantially influenced by glioblastoma stem cells (GSCs), a self-renewing, highly plastic subpopulation capable of surviving conventional therapy, with the mesenchymal (MES) transcriptional state particularly associated with treatment resistance. Recent evidence identifies the pluripotency factor OCT4 as a functional driver of MES GSC stemness and invasiveness, with its stability regulated in part post-translationally by the deubiquitinase USP5 through an E2F1–USP5–OCT4 regulatory axis. However, this proposed axis has not been independently validated. Our objective was to evaluate whether the USP5–OCT4 axis is MES-specific in independent datasets and identify potential therapeutic directions if it is not.

This study revealed no MES-specific USP5–OCT4 axis at the transcriptional level. It also showed that USP5 is broadly essential and not glioma-selective. Since the proposed mechanism operates at the post- translational level, these findings cannot independently refute the mechanistic claims of the original knockdown study. We additionally identified a correlation between USP5 and CCND1 mRNA, although its mechanistic relevance remains unclear. This relationship is of interest because CCND1 regulates CDK4/6 activity, an established therapeutic target with FDA-approved inhibitors in other cancer types.

Although USP5 did not exhibit the predicted MES-associated pattern, CDK4/6 inhibition represents a potential therapeutic direction warranting further investigation and direct functional validation.

5. Multi-omic AI Embeddings from Routine DNA Panels Stratify Mortality Risk in MAPK-activated Breast Cancer

Al-Shamsi H[1], Ghosheh M[2], Vlachavas E[2], Ghadeer Ghosheh G[2]
[1] Burjeel Cancer Institute, Abu Dhabi, UAE
[2] Nexomic, Dublin, Ireland

Background:
MAPK pathway activation is observed in approximately 20–30% of breast cancers, depending on the definition and genomic scope applied. MAPK pathway activation defines a potentially actionable breast cancer subgroup relevant to MEK-inhibitor and MAPK-targeted combination development. However, real-world enrichment cohorts are frequently panel-sequenced and lack transcriptomic or methylation profiling. We assessed whether embeddings learned through multi-omic pretraining retain prognostic value when transferred to panel-only MAPK- activated breast cancer.
Methods:
A frozen self-supervised foundation model pretrained on genomic, transcriptomic, and methylation data was applied to a panel-sequenced MAPK-activated breast cancer cohort (n=133; 58 death events) using mutation and copy-number inputs only. Embeddings were reduced using principal components and evaluated in Cox modelling, treating death and progression as competing first events. Five-fold stratified cross-validation was performed. TMB was assessed on identical splits.
Results:
Transferred multi-omic embeddings achieved a death C-index of 0.76, exceeding TMB (0.41) in the same patients consistent with no meaningful discriminative value in this cohort.
Conclusions:
Multi-omic-pretrained embeddings transferred to DNA panel-only MAPK-activated breast cancer and stratified mortality risk better than TMB. These results support further validation as a pragmatic enrichment biomarker using sequencing data already available in trial workflows.

6. Transcriptome-derived multi-omic AI signature predicts survival in anti-PD-1/PD-L1-treated melanoma

Al Shamsi H[1], Ghosheh M[2], Vlachavas E[2], Ghosheh G[2]
[1] Burjeel Cancer Institute, Abu Dhabi, UAE
[2] Nexomic, Dublin, Ireland

Background:
Durable benefit from anti-PD-1/PD-L1 therapy in melanoma remains difficult to predict. Established biomarkers, including TMB, PD-L1, and interferon-gamma signatures, explain only part of clinical variability. We evaluated whether transcriptomic profiles projected through a multi-omic- pretrained encoder predict overall survival across independent melanoma immunotherapy cohorts.
Methods:
Pretreatment transcriptomic data from 132 patients in three independent published anti-PD- 1/PD-L1-treated melanoma cohorts were analysed. A frozen self-supervised foundation model pretrained on genomic, transcriptomic, and methylation data generated embeddings from transcriptomic input. Embeddings were evaluated using PCA-aware logistic regression was fit within each training fold for overall survival event classification. Five-fold stratified cross-validation was used when event balance permitted; 70/30 holdout was used for the smallest cohort.
Results:
Cohort-level AUROC was 0.90 (95% CI 0.78-1.00) in the smallest cohort (n=27), 0.66 (SD 0.18) in a second cohort (n=55), and 0.64 (SD 0.09) in a third cohort (n=50).
Conclusions:
A transcriptome-derived multi-omic AI signature showed variable but reproducible survival discrimination in anti-PD-1/PD-L1-treated melanoma supporting further evaluation as a prognostic biomarker. Cross-cohort harmonisation and benchmarking against established immune signatures are required before clinical translation.

7. Unintended versus engineered nano/micro materials: microplastics and nanoparticles as modulators of the tumor microenvironment, a review

Contreras A, Travieso K
MS Biotechnology program at Brown University, Rhode Island, USA

The tumor microenvironment (TME) is a critical determinant of tumor initiation, progression and response to therapy, and is increasingly targeted by Precision Oncology and nanomedicine. At the same time, microplastics and nanoplastics (MPs) are emerging environmental contaminants that can accumulate in human tissues, including tumors, where experimental data suggest they may alter inflammation, stromal architecture, and vascular function in ways that resemble protumor TME remodeling. In contrast, engineered nanoparticles (NPs), including lipid-based platforms for RNA delivery and cancer immunotherapy, are explicitly designed to reprogram the same micro- environmental circuits to improve drug penetration and anti-tumor immunity.

We performed a focused evidence synthesis of recent preclinical and early human studies that examine how MPs and NPs afect five shared TME elements: exposure and localization, inflammation and macrophage function, fibroblasts and extracellular matrix, endothelial cells and angiogenesis, and tumor cell signaling. MPs have been linked to chronic macrophage activation and dysfunction, fibroblast activation and fibrosis-like matrix deposition, endothelial barrier disruption and aberrant neovascularization, and activation of oncogenic and stress-response pathways in cancer-relevant models. NPs, by comparison, have been shown to repolarize tumor-associated macrophages, modulate cancer-associated fibroblasts and matrix stifness, normalize disordered vasculature and relieve hypoxia, and reverse epithelial to mesenchymal transition while delivering cytotoxic or immunomodulatory payloads.

Our analysis supports MPs as biologically plausible, but not yet causally established, modifiers of the TME, and highlights engineered NPs as intentional tools for TME-targeted therapy. We identify key evidence gaps for MPs in exposure assessment, chronic low-dose modelling and longitudinal human data, and outline how TME assay systems and design principles from nanomedicine could be leveraged to better interrogate environmental nano/micro materials. This convergence between pollution biology and therapeutic nanotechnology suggests new opportunities for translational research and informs future strategies at the interface of cancer prevention and Precision Oncology.

8. SaGE DART (Sacituzumab Govitecan and Enfortumab vedotin Dual Antibody drug conjugates in Rare Tumors), an open-label, multi-institutional, single arm phase II basket study.

Andre Luiz Pitanga Bastos de Souza
Oklahoma University, USA

Introduction:
Sacituzumab govitecan (TROP2 target, TOP1 inhibitor payload) is approved in advanced breast cancers (1,2), while enfortumab vedotin (Nectin-4 target, MMAE payload) is approved in advanced urothelial carcinoma (3). *NECTIN4* amplifications are predictive of enfortumab vedotin response (4). Nectin-4 and TROP2 are co-overexpressed in germ cell tumors (5), squamous cell carcinoma of the bladder (6), HNSCC (7), anaplastic thyroid carcinoma (8), colorectal cancer (9), and pancreatic adenocarcinoma (10). A recent phase 1 study of enfortumab vedotin (EV) and Sacituzumab govitecan (SG) determined recommended phase 2 dose for this combination, achieving an objective response rate (ORR) of 70% (11).

Methods:
Rare tumors have an incidence of less than 6 in 100,000 per year (12). SaGE DART is an open-label, multi-institutional, single arm phase II basket study designed to evaluate the response of rare tumors to the combination of enfortumab vedotin and sacituzumab govitecan. We submitted the concept through Pfizer/Astellas, Gilead, SWOG and WIN, which will provide infrastructure to translate biomarker search harmonize access to trial slots worldwide. The primary endpoint is ORR by RECIST 1.1. Secondary endpoints include progression free survival, duration of response and safety. Exploratory endpoints comprise molecular response and mutational profile on progression in ctDNA, radiologic response in patients with baseline NECTIN4 amplification and in moderate or strong expression of NECTIN4 and TROP2 (H-score 100-300). A Simon-2 stage design incorporates an interim futility and final analysis based on H0 of ORR < 5% and H1 ORR> 40% planning to reach response in 1 out 6 patients in stage 1 and 2 out of 16 patients in stage 2 to refute H0, projecting a sample size between 36 to 96 patients depending on futility clearance for each one of 6 cohorts.

Protocol Schema:
Enfortumab vedotin 1.25 mg/kg D1 D8 (3 dose reductions allowed), Sacituzumab govitecan 8 mg/kg D1 (1 dose reduction allowed), 21-day cycles

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9. Molecular characterization of the immune checkpoint B7-H4 in squamous, MTAP-deleted, or MTAP-null tumors to inform a phase 1 clinical trial combining B7-H4 ADCs with PRMT5 /MTA cooperative inhibitors across rare and common tumors.

Andre Luiz Pitanga Bastos de Souza
Oklahoma University, USA

Introduction:
The MTAP gene is deleted in1 in 10 tumors, leading to defective salvage purine synthesis and sensitivity to pemetrexed [1-3]. MTAP deletion promotes MTA accumulation which binds to suppressive adenosine receptors in immune cells and inhibits PRMT5, responsible for spliceosome methylation, subsequent abnormal FANCA splicing variants, and sensitivity to taxanes [4]. After clearing safety in early phase trial, PRMT5 inhibitors are now combined with KRAS inhibitors and other drugs [5-11]. MTAP is co-deleted with the CDKN2A gene (p16 protein), which controls CDK4/6, and occasionally with type-I interferon genes, conferring immune resistance nature of MTAP deleted tumors; PRMT5 inhibition restores T cell function in these tumors [12-15]. Finally, aberrant expression of the immune checkpoints PD-L1 (CD274 gene), B7-H4 (VTCN1 gene), B7-H3 and TIGIT further define the immunosuppressed micro-environment of MTAP-deleted tumors (16). Currently, B7-H4 antibody drug conjugates, including Emiltatug Ledadotin (XMT-1660), Mocertatug Rezetecan, and AZD8205, are in advanced drug development (18,19). Our team aims to evaluate the predictive value of PD-L1/VTCN1 mRNA overexpression in various squamous and MTAP-deleted and null tumors.

Methods:
We hypothesize that patients with squamous histology (pure of differentiation) of various primary tumor sites, high/high and low/low VTCN1/CD274 mRNA expression predict survival to immune checkpoint inhibitors.

Results:
Preliminary data. At the CODEai database search phase, we assessed survival after defining 4 cohorts comprising over 8,000 patients with squamous non-small cell carcinoma (NSCLC) according to their low or high mRNA expression of the genes CD274 (PD-L1) and VTCN1 (B7-H4). The median survival in the high-high CD274/VTCN1 group (12 months) was numerically lower than the lower- lower CD274/VTCN1 upholding statistical significance as a prognostic biomarker. We also did not find an impact in MTAP deletion in this score, suggesting that B7H4 that the combination of these 2 biomarkers may have a stronger survival impact than prior the MTAP/PD-L1/CDKN2A score established previously by the MD Anderson group (17).

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10. Resurrect-Therapeutics AI-powered drug intelligence platform (AIDIP) to identify therapeutic opportunities and accelerate Precision Oncology to patients

El-Deiry, JM, El-Deiry JR, and El-Deiry W
Resurrect-Therapeutics, Barrington, RI, USA

Resurrect-Therapeutics core premise is simple: a terminated clinical trial does not mean a failed drug. Programs are discontinued for various reasons including inadequate patient accrual, financing constraints, strategic reprioritization, trial design, or operational challenges that may have little to do with a drug's underlying safety or efficacy. Resurrect Therapeutics AIDIP aggregates and analyzes vast amounts of fragmented biomedical and drug-development data from sources including ClinicalTrials.gov, US FDA, NIH, SEC filings, PubMed, scientific literature, patents and other public datasets. The Resurrect- Therapeutics AIDIP analytical engine connects information across clinical trials, compounds, mechanisms of action, indications, biomarkers, sponsors, regulatory history, scientific evidence, and commercial factors to uncover relationships and opportunities that may not be apparent through conventional research. Users can search specific abandoned drugs or clinical programs and receive a comprehensive dossier explaining its development history, scientific rationale, clinical evidence, reasons for discontinuation, regulatory considerations, potential development pathways, estimated difficulty, cost of advancement, and potential patient impact. Importantly, the platform proactively identifies overlooked, abandoned or shelved compounds that merit reconsideration thereby uncovering opportunities. Beyond drug intelligence, Resurrect-Therapeutics has designed an integrated operating platform for the life-sciences industry. A high-signal news and intelligence feed provides a continuously updated briefing on biotechnology, pharmaceuticals, clinical development, regulatory activity, and scientific advances. A professional network enables scientists, clinicians, researchers, investors, and industry professionals to exchange ideas and build relationships. An integrated AI workspace transforms the platform's research, intelligence, and professional knowledge into actionable work product, including analyses, reports, research summaries, strategic documents, and development materials. The platform is designed for researchers, academic medical centers, universities, biotechnology companies, pharmaceutical companies, investors, and other life-sciences professionals. Resurrect-Therapeutics organizes biomedical information, and uncovers medicines worth consideration for development, reduces wasted scientific innovation, accelerates drug development and regulatory approval, and gets potentially life-changing therapies to patients faster.

11. Adapting to a Pandemic: A Systematic Review of COVID-19 Consideration and Reporting in Phase 3 Oncology Trials

Erdozain C*, Gauri Bhat N*, Sanjay Mishra S, Warner J
Center for Clinical Cancer Informatics and Data Science, Legorreta Cancer Center, Brown University, Providence, RI, US; Rhode Island Hospital, Providence, RI, USA
**equal contribution*

Background:
The COVID-19 pandemic disrupted oncology clinical trials globally, resulting in decreased recruitment as well as increased trial suspensions and terminations. While prospective trials establish the evidentiary basis for drug approvals, the extent to which large, typically multinational, phase 3 randomized controlled trials (RCTs) incorporated and systematically reported COVID-19 considerations remains unclear.
Methods:
We analyzed oncology phase 3 RCTs initiated after November 2019 and identified in HemOncKB V53, with additional data extracted from ClinicalTrials.gov. Trials were evaluated using a researcher-developed COVID-19 "Preparedness Index" on a 0–100 scale based on protocol and statistical analysis plan documentation across four weighted domains: 1) toxicity recognition and management (40%); 2) operational mitigation (30%); 3) vaccination guidance (20%); and 4) statistical planning (10%). We also evaluated discrepancies between ClinicalTrials.gov and primary publications, as well as variation among trials investigating therapies supported by prior literature as potentially affecting COVID-19 severity.
Results:
Among 90 evaluable phase 3 RCTs, prospective preparedness varied widely across the 0–100 scale, with 24% of trials scoring below 30. Operational mitigation strategies were the most frequently documented preparedness measure (72.5%), whereas explicit statistical planning was rare (7.7%). A major planning gap was observed: 38.9% of trials reported COVID-19 adverse events (AEs) despite not prospectively identifying COVID-19 as a distinct toxicity or AE of interest. Higher preparedness scores were not associated with lower subsequently reported serious or non-serious COVID-19 AE rates.
Discrepancies in AE reporting were also observed between registry entries and primary publications. Trials investigating regimens with prior literature suggesting a potential impact on COVID-19 severity numerically demonstrated higher rates of toxicity management guidance (29% vs. 19%) and trial-specific operational planning (60% vs. 48%) compared with other trials, although these differences did not reach statistical significance.
Conclusions:
Oncology phase 3 RCTs appear to have adapted to the COVID-19 pandemic primarily through operational adjustments rather than prospective safety or statistical planning, leaving important gaps between trial preparedness and observed safety burdens. Inconsistent reporting across registries and primary publications further highlights the need for standardized reporting frameworks to improve protocol resilience and data integrity during future major external disruptions.

12. WIN Consortium: Building a Common Global Cancer Database

Farahnak F [1], El-Deiry W [2,3], Lussier Y [4], Kurzrock R [2,5], Magidi S [2], Bresson C [2], Enger S [1], Liu J [6,7,8], Bar J [9,10], Warner J [3,11,12], Meissner T 1[3], Rubin E [14], Rueter J [15], Gaddipati H [16], Kulkarni M [16], Chen Z [17], Limaye S [18], Elsey R [13], Rubenstein B [3], Joshua A [7,8], Al-Shamsi H [19,20,21], Musallam K [19], Wunder F [2], Raynaud J [2], Berchem G [22,23,24], Gantenbein M [24], Al Omari A [25], Dermime S [26], Abdel-Razeq H [25], Saintigny P [27,28], Cervantes A [29,30], Reddel R [6], Aref A [6], Martin-Liberal J [31], Lazaro C [31], Romera D [31], Sekacheva M [32], Berger R [9], Pramesh C [33], Berindan-Neagoe I [34,35], Girda E [36], Piris-Gimenez A [37,38], Farhangfar C [39], Salem M [39], Dienstmann R [37,40], Salazar R [41], Frankel N [42], Batist Z [43], Quintana Y [44,45,46,47], Batist G [1]

[1] McGill University Centre for Translational Research in Cancer and Segal Cancer Centre, Jewish General Hospital, Montreal QC, Canada
[2] Worldwide Innovative Network (WIN) Association—WIN Consortium, Chevilly-Larue,, France
[3] Legorreta Cancer Center at Brown University, Providence RI, USA
[4] Department of Biomedical Informatics, and Huntsman Cancer Center, University of Utah School of Medicine, Salt Lake City UT, USA
[5] Medical College of Wisconsin, Milwaukee WI, USA
[6] ProCan, Children's Medical Research Institute, The University of Sydney, Sydney NSW, Australia
[7] The Kinghorn Cancer Centre, St Vincent's Hospital, Darlinghurst NSW, Australia
[8] School of Clinical Medicine, Faculty of Medicine and Health, University of New South Wales, Sydney NSW, Australia
[9] Jusidman Cancer Center, Sheba Medical Center, Ramat Gan, Israel
[10] Gray Faculty of Medical and Health Sciences, Tel Aviv University, Tel Aviv, Israel
[11] Department of Medicine, Brown University, Providence RI, USA
[12] Department of Biostatistics, Brown University, Providence RI, USA
[13] Avera Cancer Institute, Sioux Falls SD, USA
[14] Ben-Gurion University of the Negev, Be'er Sheva, Israel
[15] The Jackson Laboratory, The Maine Cancer Genomics Initiative, Bar Harbor ME, USA
[16] Vyas Cancer Research (VCR Park), Visakhapatnam Andhra Pradesh, India
[17] Fudan University Shanghai Cancer Center, Shanghai, China
[18] Sir H.N. Reliance Foundation Hospital and Research Centre, Mumbai, India
[19] Burjeel Cancer Institute, Burjeel Medical City, Abu Dhabi, UAE
[20] Department of Medical Oncology, Dana-Farber Cancer Institute, Harvard Medical School, Boston MA, USA
[21] College of Medicine, Ras Al Khaimah Medical and Health Sciences University, Ras Al Khaimah, UAE
[22] Centre Hospitalier du Luxembourg, Luxembourg, Luxembourg
[23] University of Luxembourg, Luxembourg, Luxembourg
[24] Luxembourg Institute of Health, Luxembourg, Luxembourg
[25] King Hussein Cancer Center, Amman, Jordan
[26] National Center for Cancer Care and Research Hamad Medical Corporation, Doha, Qatar
[27] Department of Medical Oncology, Centre Léon Bérard, Lyon, France
[28] University of Lyon, Claude Bernard Lyon 1 University, INSERM 1052, CNRS 5286, Centre Léon Bérard, Cancer Research Center of Lyon, Lyon, France
[29] Department of Medical Oncology, INCLIVA Biomedical Research Institute, University of Valencia, Valencia, Spain
[30] CIBERONC, Instituto de Salud Carlos III, Madrid, Spain
[31] Catalan Institute of Oncology, Barcelona, Spain
[32] Federal State Autonomous Educational Institution of Higher Education I.M.Sechenov First Moscow State Medical University of the Ministry of Health of the Russian Federation, Sechenovskiy University, Moscow, Russian Federation
[33] Tata Memorial Centre, Affiliated to Homi Bhabha National Institute, National Cancer Grid, Mumbai, India
[34] University of Medicine and Pharmacy Iuliu Hatieganu, Cluj, Romania
[35] Academy of Medical Sciences, Bucharest, Romania

[36] Rutgers Cancer Institute, New Brunswick NJ, USA
[37] Vall d'Hebron Institute of Oncology (VHIO), Barcelona, Spain
[38] Vall d'Hebron Hospital Campus (VHIO), Barcelona, Spain
[39] Wake Forest University Health Sciences/Atrium Health (WFUHS), Winston-Salem NC, USA
[40] University of Vic—Central University of Catalonia, Vic, Spain
[41] Medical Oncology Department, Oncobell Program IDIBELL Institut Català d'Oncologia (ICO) Duran i Reynals Hospital, Clinical Sciences Department, Bellvitge Campus, University of Barcelona. CIBERONC, Barcelona, Spain
[42] CHAIM Medical Resource Organization, New York NY, US
[43] School of Population and Global Health McGill University, Montreal QC, Canada
[44] Beth Israel Deaconess Medical Center, Boston MA, USA
[45] Harvard Medical School, Boston MA, USA
[46] Homewood Research Institute, Guelph ON, Canada
[47] University of Victoria, Victoria BC, Canada

Precision Oncology increasingly depends on large, diverse, multidimensional datasets capable of capturing cancer complexity across populations. Yet current clinicogenomic resources remain limited by heterogeneous data structures, incomplete global representation, regulatory constraints, and limited interoperability. Individual institutions also often lack the statistical power needed to study rare genomic alterations, uncommon cancers, resistance mechanisms, and tumor-agnostic treatment strategies. To address these limitations, the Worldwide Innovative Network (WIN) Consortium outlined a strategic framework for a common global cancer database that can support Precision Oncology across diverse populations.

Rather than creating another isolated platform, WIN proposes aligning existing initiatives through a federated, consensus-driven ecosystem in which data remain locally governed while shared standards enable cross-site interoperability. Unlike initiatives focused primarily on standards development, clinicogenomic aggregation, data visualization, or national-scale data capture, WIN aims to connect multimodal and longitudinal cancer data with molecular tumor boards (MTBs), precision-oncology studies, and real-world clinical translation across an international network, while specifically promoting participation from underrepresented regions and low- and middle-income countries (LMICs).

The proposed framework includes a minimum viable dataset covering demographics, tumor characteristics, treatments, outcomes, biomarkers, and longitudinal follow-up. The Observational Medical Outcomes Partnership Common Data Model (OMOP CDM) is envisioned as the backbone for standardized clinical harmonization, with Fast Healthcare Interoperability Resources (FHIR) supporting data exchange across heterogeneous systems. Such an infrastructure could support quality assurance, real-world control cohorts, efficacy and toxicity/resistance prediction, rare-cancer research, global comparisons of standards of care, and artificial intelligence (AI)-driven decision support.

Feasibility is illustrated by existing initiatives. In Canada, the Exactis Personalized My Treatment (PMT) database integrates clinical, molecular, imaging, and pathology data across 16 centers and has captured more than 11,000 cancer trajectories, supported by automated extraction and natural language processing (NLP) and optical character recognition (OCR). In India, the Onco-Lumiseek/Vyas Cancer Research Park (VCR Park) platform links deidentified clinical, demographic, pathology, imaging, and genomic data from electronic and handwritten records through standardized mapping, harmonization, and hybrid automation adapted to low-resource settings.

WIN proposes a stepwise implementation pathway beginning with a shared metadata catalog and harmonized data dictionary, followed by federated pilots across selected sites and tumor types, and subsequent expansion of interoperability, analytics, and integration with molecular tumor boards and real-

world evidence workflows. By combining longitudinal, multimodal data from diverse populations while preserving local governance and enabling meaningful LMIC participation, a common global cancer database could reduce fragmentation, strengthen generalizability, and support more accurate and equitable precision-oncology research and care worldwide.

13. Transcriptomic Profiling of Residual Triple-Negative Breast Cancer

Fazyl F, Lafleur J, Lan C, Elebute O, Jenna S, MacNamara S, Pelmus M, Boileau JF, Brackstone M, Pezo R, Ng T, Aguilar-Mahecha A, Basik M.
Segal Cancer Center, Jewish Hospital, McGill University, Canada

Background:
Residual disease after neoadjuvant chemotherapy identifies triple-negative breast cancer (TNBC) patients at high risk of recurrence, but outcomes within this group are heterogeneous. The molecular determinants of early and late relapse in residual TNBC remain undefined. We applied a transcriptomic approach to characterize this heterogeneity and relate it to clinical outcome.
Methods:
Residual TNBC tumors from the TRICIA cohort were profiled by bulk RNA sequencing (n=82). Samples were annotated for PAM50 and Burstein molecular subtype, predicted homologous recombination deficiency (HRD), and immune composition (CIBERSORT, ESTIMATE). We applied unsupervised clustering to the transcriptomic profiles. Resulting clusters were characterized by pathway enrichment and clinical outcome, defined as poor prognosis and good prognosis.
Results:
Clustering resolved four transcriptomic subgroups with distinct molecular identities. C1 was luminal androgen receptor like -predominantly PAM50 luminal A and Burstein LAR, differentiated, and proliferation-low. C2, the largest cluster, was basal-like and BLIS- enriched, HRD-high, immune-depleted, and the most proliferative. Within C2, two subclusters were resolved. The first was mostly basal like and the most proliferative, characterized by enrichment of cell cycle programs and a predominance of poor prognosis cases. The second showed lower proliferation and was enriched for good prognosis cases. C3 was immune-activated, with the highest cytolytic and immune infiltration scores. C4 was a small mesenchymal-like group. Early relapse concentrated in the proliferative, immune cold C2 cluster, whereas good prognosis cases were more frequent in the LAR (C1) and immune-activated (C3) clusters.
Conclusion:
Residual TNBC comprises molecularly distinct transcriptomic subgroups, including differentiated LAR, immune-activated, and proliferative basal like/HRD-high phenotypes. This heterogeneity is structured primarily by proliferation and immune activity rather than by single subtype classification.

14. Exploring Molecular, Immune, and Microenvironmental Responses to Combined GSK3β Inhibition and Imipridone Therapy

Ghandali M [1–4], Zhang L[1–4], Carneiro B [2,3,5], El-Deiry W [1–5]
[1] Laboratory of Translational Oncology and Translational Cancer Therapeutics, Warren Alpert Medical School of Brown University, Providence, RI, USA
[2] Legorreta Cancer Center, Brown University, Providence, RI, USA
[3] Joint Program in Cancer Biology, Brown University and Lifespan Cancer Institute, Providence, RI, USA
[4] Department of Pathology and Laboratory Medicine, Brown University, Providence, RI, USA
[5] Hematology/Oncology Division, Department of Medicine, Lifespan and Brown University, Providence, RI, USA

Background:
Glycogen synthase kinase-3β (GSK3β) regulates multiple pathways involved in cancer cell survival, therapeutic resistance, metabolism, and immune regulation. Elraglusib (9-ING-41), a GSK3β inhibitor, has demonstrated antitumor activity across multiple cancer models.
Imipridones are a novel class of anticancer agents with activity across a range of tumor types. We previously demonstrated synergistic antitumor activity between elraglusib and the imipridone ONC206 across ovarian, glioblastoma, pancreatic, and colorectal cancer models. In this study, we further investigated the molecular mechanisms underlying this synergy, its effect on immune-mediated tumor cell killing, and the influence of hypoxia on treatment response.
Methods:
Cancer cell lines were treated with elraglusib, ONC206, or the combination. CellTiter-Glo viability assays were used to evaluate treatment response and synergy. Western blot analysis was performed to study pathways involved in the combination effect, including NF-κB signaling and downstream survival proteins. We also evaluated the effect of the combination on immune- mediated killing of cancer cells. Since hypoxia can change cancer cell metabolism and response to treatment, the effects of elraglusib and ONC206 are also being studied under hypoxic conditions.
Results:
Elraglusib and ONC206 showed synergy across multiple cancer models. Western blot analysis showed decreased NF-κB p65, MCL-1, and survivin following treatment, suggesting that inhibition of NF- κB signaling contributes to the combination effect. The combination also increased immune-mediated killing of cancer cells compared with either treatment alone. Studies examining the effect of hypoxia on sensitivity to elraglusib, ONC206, and the combination are ongoing.
Conclusion:
Elraglusib and ONC206 show synergistic anticancer activity that is associated with inhibition of NF-κB signaling and increased immune-mediated cancer cell killing. We are also investigating how hypoxia affects response to the combination, particularly given the effects of these treatments on pathways involved in cancer cell metabolism. Together, these studies may help better understand which tumor conditions influence response to elraglusib and ONC206.

15. Beyond Conventional Biomarkers: Multi-Omic Foundation Models Reveal Prognostic Patient States Across Solid Tumours

Ghosheh G[1], Vlachavas E[1], Ghosheh M[1], Al Shamsi H[2]
[1] Nexomic, Dublin, Ireland
[2] Burjeel Cancer Institute, Abu Dhabi, UAE

Background:
Molecularly defined treatment selection in oncology remains largely dependent on tumour type and a relatively small number of established biomarkers. However, patients assigned to the same clinical or molecular category often experience markedly different outcomes. We investigated whether DeepOmic, a self-supervised foundation model trained on multiple omic modalities, could derive patient representations that uncover additional clinically relevant heterogeneity across solid malignancies.
Methods:
DeepOmic was trained using integrated genomic, transcriptomic, and epigenomic profiles to generate patient-level latent representations without supervision from survival outcomes. Model representations were subsequently evaluated in seven independent cohorts comprising 5,447 patients with breast, non-small-cell lung, gastric, and renal cancers. For each cohort, frozen embeddings were grouped using consensus k-means across cluster solutions ranging from three to eight groups. Clinical outcomes were introduced only after clustering.
Associations between the resulting molecular subgroups and overall survival were evaluated using age-adjusted Cox proportional-hazards models.
Results:
Unsupervised organisation of patients in the DeepOmic latent space revealed survival- associated substructure across all evaluated tumour settings. In the breast cancer dataset containing 1,866 patients, cluster membership was strongly associated with overall survival across every clustering resolution tested, with global Cox p-values ranging from
5.95×10^{-33} to 2.55×10^{-29}. Differences in median overall survival between identified groups
reached 21 months. Similar patterns were observed across the remaining six independent datasets, where the separation between the most and least favourable survival groups ranged from 11 to 75 months.
Conclusions:
DeepOmic captures multidimensional molecular variation that translates into clinically relevant patient stratification despite the absence of outcome supervision. The recurrence of survival- associated structure across independent datasets and multiple tumour types indicates that multi-omic foundation-model representations may provide information beyond conventional disease labels and individual biomarkers. Future work will assess whether these latent patient states also predict progression, therapeutic response, and pathway-level vulnerabilities relevant to precision-oncology decision making.

16. In Vivo Generation of CAR Macrophages by Co-Optimizing RNA Modification and LNP Delivery Vehicle

Holmes-Farley S [1] and Raimondo T [1-4]
[1] Center for Biomedical Engineering, School of Engineering, Brown University, Providence, RI, USA
[2] Division of Biology and Medicine, Brown University, Providence, RI, USA
[3] Legorreta Cancer Center, Brown University, Providence, RI,
[4] The Brown RNA Center, Brown University, Providence, RI, USA

Clinical trials of CAR-M cell therapies have shown therapeutic promise against solid tumors, a notable distinction from currently used CAR-T therapies. Clinical strategies for CAR-M generation, predominantly adenoviral transduction, however, are hampered by safety concerns associated with viral transduction, immune cell exhaustion associated with permanent CAR expression, lymphodepletion requirements prior to adoptive CAR-cell therapy, and slow and costly manufacturing. The development of lipid nanoparticles (LNPs) for mRNA delivery to macrophages is of substantial interest, as this platform allows transient mRNA-driven CAR expression, eliminates risks associated with viral transduction and insertional mutagenesis, enables *in vivo* CAR-M generation and allows the encapsulation of synergistic therapeutics within the LNP vehicle. Chemical modifications to mRNA constructs, as well as lipids incorporated into LNPs, drive macrophage activation, mRNA transfection efficiency and thus mRNA- driven CAR expression level and therapeutic efficacy. Here we hypothesize that simultaneous tuning of mRNA chemistry and LNP composition will improve CAR expression and CAR-M therapeutic efficacy, enabling efficient in vivo CAR expression, and anti-tumor CAR-M activation within tumor microenvironments.

To test this, a library of 60 LNP formulations was generated by incorporating various ionizable lipid chemistries, cationic or anionic phospholipids, and RNA chemical modifications (including U and C methylations, and N1-methylpseudouridine). Optimized modified-mRNA LNP formulation enabled 56- fold higher CAR-expression per macrophage than the lowest performing LNP in our library, and approximately 2-fold higher CAR expression than currently FDA-approved LNP (Onpattro, formulated in- house as a control). Optimized mRNA-LNPs achieved 90% CAR-positive cells without decreasing macrophage viability in vitro; increased phagocytotic activity of antigen-positive breast cancer lines (HER2+ SKBR3 cells) by 10-fold in coculture assays, and decreased breast and ovarian cancer spheroid volume ~2-fold vs. a non-CAR mRNA LNP control in vitro. Loss of either optimal mRNA chemical modification or optimal LNP formulation eliminated CAR-M phagocytotic activity. Importantly, CAR-M activity was only observed against antigen-positive cancer (HER2+ breast-SKBR3 or ovarian-SKOV3 cells), with antigen-negative lines (HER2- MDA-MB-468) viability preserved. mRNA-LNP transfected macrophages remained active for at least six days post treatment.

In vivo experiments performed in a murine model of metastatic ovarian cancer assessed CAR-M generation 24 and 96 hours post systemic, intraperitoneal (IP) injection. Treatment groups included the optimal LNP formulation and RNA modification identified in vitro and the cross combinations of the worst modification-best LNP and best modification-worst LNP. CAR expression in the intraperitoneal lavage fluid found a significant nearly two fold increase in number of CAR-M between best and worst treatment groups which supported our in vitro findings that both the mRNA modification and LNP delivery vehicle are critical for sustained and active CAR macrophages. Tumor burden trended lower with the optimized formulation at both 24 and 96 hour time points. Together, these results demonstrate that combinatorial optimization of mRNA chemistry and LNP design is necessary for efficient in vivo generation of phagocytotic CAR-M cells; and critically advances the field towards the ultimate goal of developing a flexible, non-viral platform for in vivo generation of therapeutic CAR-M.

17. Evaluating a Brachyury-Overexpression Model for the Study of the Chordoma Epigenome in Brachyury- Deficient Chordoma Cells Lines

Huerta E [1], He K [1], Lopez A [1,2], Schuler A [1], Godinez C [1], Golaskan Z [1], Sullivan P [1], Martinez-Moreno M [1]
[1] Department of Neurosurgery, Warren Alpert School of Medicine at Brown University Collin Stevens, RI, USA
[2] Tufts University, MA, USA

Background:
Chordomas are rare, slow-growing tumors of the spine arising from notochordal remnants within intervertebral discs, resistant to conventional therapy, and displaying high local recurrence. Chordoma epigenomics seeks to identify master oncoregulators as potential therapeutic targets using commercially available cell lines and epigenetic laboratory techniques. Brachyury (*TBXT*) is a transcription factor and key diagnostic marker for chordoma linked to tumor proliferation and survival. Chordoma cell lines are addicted to brachyury and sustain stable expression over several passages. Unfortunately, rare brachyury loss hinders *in vitro* chordoma growth, leading to cell senescence and growth arrest, and ultimately undermining downstream epigenetic studies such as ChIP (chromatin immunoprecipitation)- seq (next-generation sequencing) due to poor signal enrichment. By overexpressing brachyury, we aim to enrich ChIP-seq data and improve downstream epigenetic studies in brachyury-deficient chordoma cells.

Objective:
To complement the use of ChIP-seq for *in vitro* chordoma epigenomics by inducing overexpression of brachyury in brachyury-deficient chordoma cells.

Methods:
Competent bacteria were transformed with commercially available plasmids: pCMV6-Entry Mammalian Expression Vector (PS100001, Origene) and T (NM_003181) Human Tagged Lenti ORF Clone (RC218530, Origene). Plasmids from overnight cultures were purified and transiently transfected into commercially available chordoma cell lines using the Transit-2X Dynamic Delivery System (MirusBio). After 48 hours, cells were harvested and lysed to perform downstream protein (western blot and immunohistochemistry) and RNA analyses (RT-qPCR). Untransfected cells were preliminarily fixed with formaldehyde and sonicated at different parameters for optimization. Fragments were validated using the TapeStation protocol and software.

Results:
Preliminary RT-qPCR analyses confirmed increased levels of brachyury expression in transfected UCH-1-N cells. Optimizing transfection of the JHC7 cell line is challenging due to growth arrest and early senescence, highlighting transfection challenges in chordoma cell lines. Sonication parameters varied across cell types, underscoring the challenges of chordoma epigenetic studies.

Conclusion:
Brachyury overexpression represents a potential rescue approach for brachyury loss in cultured chordoma cell lines with limited resources. An important consideration is the downstream effects of overexpressing brachyury, which could generate background noise in downstream applications. Regarding ChIP, the protocols for chordoma cell lines varied by cell type, requiring optimization and verification of chromatin fragmentation.

Future directions:
Future work includes optimization of other chordoma cell lines to evaluate the range of brachyury overexpression. We also look forward to assessing overexpression success by comparing ChIP– seq datasets from baseline versus transfected chordoma cell lines. Primary criteria would include normalized and relative strand cross-correlation coefficients (NSC and RSC), as well as strand-shift correlation scores per ENCODE 2012 ChIP-seq guidelines. Alternatively, we would evaluate using CUT&RUN to enrich brachyury-binding sites in brachyury-deficient cell cultures to elucidate endogenous transcription complexes.

18. Trials for Rare Cancers More Successful than those for Common Cancers

Kasibhatla V [1], Schäffer A [2], Sumeet Patiyal [2], Tian-Gen Chang [2], Padma Sheila Rajagopal [2,3], Eytan Ruppin [2]

[1] Warren Alpert School of Medicine, Brown University, Providence RI, USA
[2] Cancer Data Science Laboratory, Center for Cancer Research, National Cancer Institute, Bethesda, MD, USA
[3] Women's Malignancies Branch, Center for Cancer Research, National Cancer Institute, Bethesda, MD, USA

Background and Purpose:
Rare cancers are often difficult to study in clinical practice despite their potential to yield early efficacy signals and facilitate drug development for broader indications. We compare success rates between trials studying rare versus common cancers and identify contextual factors that may influence trial outcomes over time.

Materials and Methods:
We analyzed 32,145 completed trials from Trialtrove (https://citeline.informa.com/trials/results), comparing outcomes and common failure reasons between trials enrolling patients with only rare cancers, at least one rare cancer, or only common cancers. We stratified our results by treatment type, trial phase, and sponsor type. We defined rare cancers as those with incidence rates of 15 cases/100,000 person-years; our analyses were repeated using lower thresholds.

Results:
Trials enrolling only rare cancers had a 42.2% success rate, significantly higher common cancer trials (38.2%; $P=6.6x10^{-12}$). Trials including at least one rare cancer showed a similarly elevated success rate (42.5%; $P=8.1x10^{-15}$). In trials started in earlier years, "at least one rare" was more successful than "exclusively rare". However, among trials starting 2019-, the "at least one rare" and common groups have similar success rates, and "exclusively rare" trials have a higher success rate. Contrary to published assumption, poor enrollment was not a common cause of failure in rare cancer trials. Business-related failures increased substantially over the past decade, largely driven by industry-sponsored trials

Conclusion:
When a new therapy is applicable to both rare and common cancers, initiating trials in rare cancers may enhance rare cancer research and offer a higher probability of success.

19. Patient derived pancreatic tumor organoids as a surrogate to predict treatment response to radiation and chemotherapy in individual patients: A model for precision medicine.

Keepers Z, Roy S, Bhandary B, Molitoris J and Shukla H
Department of Radiation Oncology, University of Maryland School of Medicine, Baltimore, MD, USA

Pancreatic ductal adenocarcinoma (PDAC) remains one of the most lethal forms of cancer, largely due to the absence of effective screening methods and a high incidence of distant metastasis at the time of diagnosis. Regardless of eligibility for surgery, the majority of patients receive systemic chemo-radiation therapy, which includes nab-paclitaxel combined with radiation therapy (RT) or FOLFIRINOX with RT. Unfortunately, despite these interventions, patients' tumors often progress and develop resistance to therapies. In this study, we investigated the response of 10 patient-derived pancreatic tumor organoids (IDs: 8510, 7800, 11777, 982778, 242566, 671287, 868679, 987798, 7886, 8516) to gemcitabine, FOLFIRINOX, 5 FU, and radiation therapy (RT). The preliminary data has shown differential sensitivity of tumor organoids to drugs and radiation treatment as demonstrated by different IC50 doses. The tumor organoid 8510 exhibited a higher tolerance to 5-FU and FOLFIRINOX treatments compared to 7800 and 11777. We also observed the best treatment response and maximum organoids death when they treated with combination of chemotherapy and radiation. We also observed that 11777 showed resistance to high doses of RT and chemotherapy drugs. Notably, the genetic profile of this patient included PIM1 and ARID1b mutations, which could be associated with radiation resistance. The immunofluorescence and immunoblot data have shown an upregulation of OCT4 and SOX2 transcriptional factors in tumor organoids, indicating the presence of cancer stem cells that may play a role in treatment resistance. Thus, patient-derived pancreatic tumor organoids could be used as a surrogate to provide RT and chemotherapeutic drug sensitivity profiles in individual patients, which could bring us a step closer to precision medicine.

20. Biomarker Loaded ASC Microparticles for Targeted Immunotherapy of Melanoma

Khalafkhany D, Tütüncü D and Erkol N
Boğaziçi University, Department of Molecular Biology and Genetics, Apoptosis and Cancer Immunology Laboratory (AKiL), and Center for Targeted Therapies and Technologies, Istanbul, Türkiye

We have developed an innovative patented antigen delivery platform based on extracellular ASC- speck microparticles, which harnesses natural human inflammasome activation and subsequent immune response. We successfully purified ASC-speck microparticles from transfected Hek293 cells. Furthermore, we expressed and purified chimeric ASC microparticles containing target biomarker/antigen for further assessment of antitumoral immune responses in a mouse model of melanoma. These ASC based microparticles can induce inflammatory responses in immune cells, both in vitro and ex vivo, which resembled the effects of traditional adjuvants. We observed that the stability, size, and immune-boosting properties of ASC specks make it a promising candidate as antigen carriers and a natural adjuvant/immune booster. We immunized mice with Tumor Associated Antigens (TAA) including human MageA3, and mouse MageA1 and mouse Mlana before inoculating them with tumor cells, enabling us to evaluate potential cancer treatments relying on the immune system. We used chimeric proteins of ASC and TAA antigens, Freund's adjuvanted recombinant antigens, or PBS as control. Antibody titers were measured by analyzing pre-immunization and pre- challenge sera samples for anti-MageA3 specific IgG. The immunized mice were inoculated subcutaneously with a mixture of Matrigel and 10e5 B16-hMageA3 cells, and tumor growth was monitored. Notably, the protection rate in vaccinated mice was 40% for MageA3, 50% for MageA1, 0% for Mlana in monovalent vaccines and 66% for trivalent vaccine in contrast, all mice in control groups developed tumors despite the high anti-MageA3 antibody titer in mice receiving Freunds adjuvanted antigen.

Further we tested the effectiveness of biomarker loaded ASC-microparticles in a combination therapy model using checkpoint blockade. The protective capacity of Bi-valent ASC- microparticles were demonstrated against B16-F10-hMAGE-A3 tumor model, with 77 percent tumor prevention rate in C57BL/6 mice. Ex vivo analysis revealed that splenic T cells of ASC- microparticle vaccinated mice can generate cytotoxicity to tumor cells in the presence of checkpoint blockade. In a therapeutic setting, combined treatment of ASC microparticles and anti-PD-1 produced pronounced tumor control and survival compared to control/single-treatment groups against tumor challenge. Also, correlation of survival with several intra- tumoral and blood associated parameters were demonstrated in this setting.

21. Redirecting pre-existing anti-pathogen T-cell immunity to overcome immune resistance in the tumor microenvironment

Riva Kovjazin MD, Lior Carmon PhD
Imyun Pharmaceuticals, CA, USA

Background:
Approximately 80% of patients with solid tumors fail to respond durably to available immunotherapy. Two mechanisms dominate: T-cell exhaustion within the tumor microenvironment (TME), and loss of MHC-I antigen presentation through TAP downregulation. Our group has developed signal peptide (SP) domains as multi-epitope antigen platforms. Work in animals and in cancer patients showed that SPs derived from human and bacterial particularly BCG antigens elicit robust CD8+ T-cell responses across MHC barriers through multiple MHC-I binding, and that SPs reach the ER by a TAP-independent route, allowing presentation in tumors that have lost TAP. We have now repurposed SPs as immune tags that mark tumor cells for destruction by pre-existing anti-pathogen T cells.

Methods:
ORION is a 33 kDa recombinant Immune-Tag Conjugate comprising a trastuzumab-derived anti- HER2 scFv joined by a furin-cleavable linker to a 24-mer SP from the BCG lipoprotein LpqH and a 9-mer CMV epitope. Binding and internalization were assessed in A431 and BT-474 cells. Killing was measured in HER2+/HLA-A2.1+ OVCAR-3 cells using human T cells primed against BCG or CMV, and in CT-26/hHER2 cells using splenocytes from BCG-vaccinated BALB/c mice. ORION was administered intratumorally to BCG- vaccinated, CT-26/hHER2-bearing BALB/c mice.

Results:
ORION showed specific HER2 binding with internalization sustained at least 72 hours. ORION- labeled OVCAR-3 cells underwent >60% specific killing by BCG- or CMV-primed human T cells. ORION-treated CT-26/hHER2 cells underwent approximately 50% specific killing by splenocytes from BCG-vaccinated mice. Intratumoral ORION produced significant anti-tumor activity ($P<0.00076$), accompanied by greater than 11-fold expansion of CD3+CD44+ T cells in the TME and a collapse of the PD-1+/LAG-3+ exhausted fraction from 30.5% to 1.1%.

Conclusions:
Tagging tumor cells with BCG-derived SPs redirects pre-existing, polyclonal, non-exhausted T- cell immunity against BCG and CMV to the TME. This Trojan horse strategy converts widely held anti-infective immunity into anti-tumor activity without CD3 engagement, without HLA typing, and without neoantigen sequencing. Because BCG coverage is large in the settings where Precision Oncology infrastructure is least available, the approach is intrinsically suited to global deployment.

22. Proteomic Analysis of Proximal Poly (ADP-Ribose)-Associated Protein Complexes in Response to DNA Damage and Replication Stress

Krystyniak H [1], Roos W [1], DaSilva N [2], Pearson C[1], Sobol R [1]
[1] Department of Pathology and Laboratory Medicine, Warren Alpert Medical School & Legorreta Cancer Center, Providence, RI, USA
[2] Department of Molecular Biology,Cell Biology, and Biochemistry; Brown University, Providence, RI, USA

Poly(ADP-ribose) (PAR) signaling, catalyzed by PARP1 and PARP2 in response to DNA damage or replication stress, serves as a critical sensor of base excision repair (BER) and DNA Repair protein complex networks. Beyond its role as a post-translational modifier, PAR has emerged as a driver for liquid-liquid phase separation (LLPS), functioning as a scaffold that nucleates biomolecular condensates at sites of DNA damage. The full interactome of proteins recruited to these PAR-driven condensates, and the dynamics of their assembly in response to PAR turnover, remain incompletely defined. Here, we developed a proximity biotinylation approach to investigate direct and proximal PAR-binding proteins. The PAR-selective RNF146(100-182) WWE PAR binding domain was fused to the proximity biotinylating enzyme TurboID, enabling biotin tagging of PAR-associated proteins under defined treatment conditions. Biotinylated proteins were captured and characterized by liquid chromatography mass spectrometry (LC-MS) following DNA damage induction, and PARG inhibition, and interaction networks were analyzed, revealing distinct, treatment-dependent enrichment and novel candidate PAR interactors. Three hits, PSPC1, CHD1L, and SFPQ, were further validated using EGFP fusion vectors and confocal microscopy. PSPC1 localized independently of DNA damage, CHD1L displayed transient recruitment kinetics, and SFPQ exhibited recruitment that was both DNA damage and PAR- dependent. Further characterization of SFPQ extended its known role to the repair of both single- strand and double-strand DNA breaks. Collectively, these findings nominate paraspeckle components as participants in PAR-driven formation at sites of DNA damage and establish a framework for investigating the intersection of RNA-binding protein biology with BER.

23. WIN International Molecular Tumor Board: From Real-World Molecular Evidence to Clinical Options— A Multi-Domain Analysis

Razelle Kurzrock [1,2], Shai Magidi [1], Catherine Bresson [1], Sewanti Limaye [3], Jia Liu [4], Alberto Hernando Calvo [5], Stephanie L. Graff [6], Hao-Wen Sim [4], Rishi R. Lulla [6], Liang Cheng [6], Jens Rueter [7], Matthew Hadfield [6], Fanny Wunder [1], Wafik S. El-Deiry [1,6]

[1] Worldwide Innovative Network (WIN) Association—WIN Consortium, Chevilly-Larue, France
[2] Medical College of Wisconsin, Milwaukee, WI, USA
[3] Sir HN Reliance Foundation Hospital and Research Centre, Mumbai, India
[4] Kinghorn Cancer Centre, St Vincent's Hospital, Darlinghurst, NSW, Australia
[5] Medical Oncology Department, Vall d'Hebron Hospital Campus and Vall d'Hebron Institute of Oncology (VHIO), IIS IR-HUVH, Barcelona, Spain
[6] Legorreta Cancer Center, Providence, RI, USA
[11] The Jackson Laboratory, Maine Cancer Genomics Initiative, Bar Harbor, ME, USA

Background:
Translating molecular findings into clinical options requires integrating tumor biology with evidence strength, clinical suitability and treatment access. The Worldwide Innovative Network (WIN) International Molecular Tumor Board (MTB) connects physicians and scientists from 19 cancer centers across 12 countries to review complex oncology cases. Cases were referred by patient-advocacy organizations seeking support or by investigators seeking professional support.

Methods:
Case presentations, molecular reports and MTB conclusion reports informed a retrospective analysis of 72 cases discussed since the board's 2023 launch, examining the available evidence, options and considerations accompanying those discussions. 19 cases (26.4%) were classified as rare cancers. Tissue-liquid comparisons were assessed using Jaccard similarity, calculated as shared gene- alteration features divided by all distinct features across tissue and liquid biopsies.

Results:
The international cohort spanned diverse cancer types, most commonly gastrointestinal, central nervous system, breast and thoracic cancers. It was substantially pretreated (median 3 prior major systemic treatment lines; range, 0–8), reflecting frequent referral for treatment-refractory or otherwise complex disease. Available evidence encompassed genomic alterations, gene-expression profiles, protein- expression findings and epigenetic markers, with testing varying across cases. Tissue–liquid comparisons illustrated the uneven molecular information availability and comparability: 23 cases (31.9%) had at least one qualifying somatic alteration reported from each specimen type. Median observed overlap was 10% (range, 0–50%). Findings were pooled within cases; differences in sampling dates, assay coverage and reporting limited interpretation. Discussions encompassed treatment options, further investigations and clinical management. Personalized/non-standard combinations outside trials were discussed for 69 cases (95.8%), clinical-trial options for 46 (63.9%), other non-standard therapies outside trials for 30 (41.7%), and standard treatments for 21 (29.2%). Further testing was discussed for 65 cases (90.3%) and monitoring/clinical management for 32 (44.4%). Categories overlapped and included alternatives. Among 273 therapeutic options with evaluable concurrent regimen size, 32.6% involved one agent, 31.5% two, 22.0% three, 9.5% four and 4.4% five or more; sequential strategies were excluded. The considerations accompanying these discussions varied by the type of option or clinical action discussed. Access concerns were documented for 60% of clinical-trial discussions, evidence or diagnostic uncertainty considerations for 40% of additional-testing discussions, and clinical-suitability concerns for 34% of personalized/non-standard combination discussions. Percentages were calculated within discussion type, and consideration domains could overlap.

Conclusion:
In this selected, clinically and molecularly heterogeneous cohort, therapeutic planning and further

investigation were prominent components of MTB discussions. The findings illustrate the breadth of molecular interpretation and the accompanying evidence, suitability and access considerations. They describe options discussed rather than treatment receipt or trial enrolment. Treatment outcomes were unavailable; follow-up linking discussions to implementation and outcomes is needed to establish clinical benefit.

24. PRISM-Pancreatic Ca: An International, Multi-Site, Risk-Stratified Cohort Study Evaluating a Blood-Based Multiomic Biomarker (Avantect) for Early Detection of Pancreatic Ductal Adenocarcinoma

Lamoury G
Northern Sydney Cancer Centre, Royal North Shore Hospital, Northern Sydney Local Health District; and University of Sydney, Sydney, Australia. *On behalf of the PRISM-Pancreatic Ca Study Group.*

Objectives:
To evaluate the performance and feasibility of serial testing with a blood-based multiomic biomarker (Avantect Pancreatic Cancer Test) added to risk-stratified surveillance for early detection of pancreatic ductal adenocarcinoma (PDAC) across three parallel risk populations: high genetic/familial risk, moderate risk (new-onset type 2 diabetes), and a pilot cohort with unexplained low-level CA19-9 elevation.
Methods:
PRISM-Pancreatic Ca is a prospective, multi-site, international, observational cohort study across sites in Australia and India, with ethics and site governance approvals obtained at each site prior to enrolment. Arm 1 enrols individuals with a pathogenic germline PDAC-susceptibility variant and/or familial pancreatic cancer, co-enrolled with the APRISE study, undergoing annual EUS/MRI surveillance with serial Avantect testing (target n~300-500). Arm 2 enrols adults aged 50 years or older with new-onset type 2 diabetes (absolute PDAC risk 0.4-1%), sub-stratified by metabolic syndrome status (Arm 2A/2B), using Avantect as an adjunct biomarker test alongside standard assessment (target n~1000-1500). Arm 3 is a hypothesis-generating pilot in individuals with unexplained, mildly elevated CA19-9 (above the upper limit of normal without an identified cause) (n~50-100).
Results:
PRISM-Pancreatic Ca is designed to sit alongside the UK SAFE-D study and the Global PRECEDE study, both currently recruiting. Unlike SAFE-D, Arm 2 sub-stratifies new-onset-diabetes participants by metabolic syndrome status. Arm 3 is the first prospective evaluation of a multiomic biomarker as a second-line discriminator for unexplained, low-level CA19-9 elevation. Given the Arm 1 sample size (n~300-500), the study is powered for specificity in Arm 1; other endpoints will be reported descriptively on completion.
Conclusion:
PRISM-Pancreatic Ca introduces a novel, internationally coordinated framework extending biomarker-integrated surveillance across a graded, metabolically and biochemically defined risk continuum not yet addressed elsewhere.
Disclosure:
Avantect is a laboratory-developed test performed in a CAP-accredited, CLIA-certified laboratory; it is evaluated as an adjunct to risk-stratified surveillance, not as a population screening test, and reported performance requires validation in the intended-use population.

25. OptiRanker: A Simulation and Optimization Framework for Efficient In Vivo Validation of Drug Prioritization Algorithms

Landau O [1] , Thangathurai K [1,2] , Magidi S [3] , Porgador A [1] , Rubin E [1]
[1] Ben Gurion University of the Negev, Be'er Sheva, Israel
[2] University of Vavuniya, Vavuniya, Sri Lanka
[3] Worldwide Innovative Network (win) Association - Win Consortium, Chevilly-Larue, France

Background & Objective:
Precision Oncology increasingly relies on computational drug-prioritization algorithms that use patient omics data to recommend therapies, yet few undergo rigorous in-vivo validation due to high costs, small cohorts, and ethical constraints of prospective trials. OptiRanker is a statistical simulation and optimization framework that identifies the minimal experimental cohort (number of individuals and drugs) required to reliably rank the performance of drug-prioritization algorithms prior to in-vivo testing.
Methods:
OptiRanker operates in two modes. In Simulation mode, a synthetic ground-truth ranking is generated and progressively perturbed with controlled noise to create predictors of decreasing accuracy, testing whether the framework correctly recovers the expected order. In Ranking mode, real transcriptomic and drug-sensitivity data are used: algorithm outputs are converted into per-individual drug ranks and scored against a ground truth using a position-biased weighted mean-squared error (WMSE) that gives higher penalty to errors involving higher ranked drugs. Spearman correlation between subset-derived and full-dataset rankings identifies the minimal informative subset of individuals and drugs, and a cost-effectiveness metric balances informativeness against experimental size. As proof of concept, three WIN-consortium drug-prioritization algorithms, Digital Display Precision Predictor (DDPP), Simplified Interventional Mapping System (SIMS), and WINTHER, were applied to Cancer Cell Line Encyclopedia (CCLE) transcriptomic data and Genomics of Drug Sensitivity in Cancer (GDSC) using half- maximal inhibitory concentration (IC50) values as proxy for effectiveness across 798 cell lines and 36 drugs.
Results:
Under controlled simulation (100 iterations; 20 predictors, 20 drugs, and 20 individuals), OptiRanker consistently recovered the expected ranking of progressively noisier predictors. Selecting individuals and drugs by their standard deviation across predictor outputs, rather than arbitrarily, substantially accelerated convergence of subset rankings toward the full-dataset ranking, achieving Spearman r greater than 0.8 with as few as 6-10 individuals and 5-8 drugs. In the in-silico proof of concept, DDPP, SIMS, and WINTHER were ranked best to worst (DDPP > SIMS > WINTHER) using the full 36 drug x 798 cell line dataset (approximately 29,000 measurements); this ranking was fully reconstructed using a minimal subset of only 6 individuals and 1 drug (6 measurements), consistent with principal component analysis of algorithm predictions. Shuffling experiments suggest this is not a coincidental result.
Conclusion:
OptiRanker provides a reproducible, data-driven approach to design minimal yet statistically robust experimental cohorts for validating drug-prioritization algorithms. It can substantially reduce the time, cost, and resource burden of prospective in-vivo trials. By enabling objective, quantitative comparison of algorithms before costly validation studies, OptiRanker addresses a key bottleneck in translating computational precision-oncology tools into clinical practice.

26. OncoAID: An Updated Open-Access Database of FDA-Approved Targeted Anti-Cancer Therapies for Precision Oncology

Landau O [1], Magidi S [2], Bresson C [2], Wunder F [2], Kurzrock R [2,3], El-Deiry W [2,4], Thangathurai K [1], Rubin E[1]
[1] Ben-Gurion University of the Negev, Be'er-Sheva, Israel
[2] Worldwide Innovative Network (WIN) Association - WIN Consortium, Chevilly-Larue, France
[3] Medical College of Wisconsin, Milwaukee WI, USA
[4] Legorreta Cancer Center at Brown University, Providence RI, USA

The rapid evolution of targeted therapies in oncology has revolutionized cancer treatment, offering new hope to patients through precision medicine by specifically inhibiting molecular targets that drive tumor growth. However, the expansion of these molecularly targeted cancer therapies has simultaneously created a deeply fragmented information landscape. Clinicians and researchers often need to integrate drug targets, genomic biomarkers, approved indications, clinical efficacy, regulatory status, and experimental drug-sensitivity information across multiple independent, isolated repositories. To resolve this disconnect and empower real-world decision-making, we developed OncoAID, an open-access interactive database designed as a practical, single resource for targeted oncology therapies.

At its foundation, OncoAID utilizes Food and Drug Administration (FDA)-approved targeted anti-cancer therapies as its core inclusion framework. The platform integrates manual curation with an automated pipeline that connects directly to major pharmacologic and genomic resources, including PharmGKB, DrugBank, Therapeutic Target Database (TTD), and GeneCards. To provide deeper mechanistic insights, OncoAID incorporates experimental cell-line sensitivity metrics from the Genomics of Drug Sensitivity in Cancer (GDSC) and the Cancer Cell Line Encyclopedia (CCLE), delivering median IC50 values alongside tissue-specific Z-score distributions. Recognizing the need to streamline information for time-constrained oncologists, the system employs a custom Named Entity Recognition (NER) model to systematically extract progression-free survival (PFS) and overall survival (OS) outcomes from clinical trial data. Furthermore, OncoAID leverages Large Language Models (LLMs) to generate standardized, high-level treatment overviews and bridge multi-lingual barriers to map global regulatory approval statuses across the European Medicines Agency (EMA), Pharmaceuticals and Medical Devices Agency (PMDA, Japan), and National Medical Products Administration (NMPA, China).

Following continuous updates, the OncoAID dataset now catalogs 657 approval entries comprising 375 unique treatments across 411 distinct indications, structured under 18 broad cancer-type categories with 179 target annotations. Longitudinal analysis reveals a marked increase in approved targeted therapies over time, with strong growth in combination regimens in recent years. Evaluating integrated clinical efficacy metrics demonstrates the practical impact of these strategies: combination regimens achieved a significantly longer average PFS compared to single-agent therapies (15.13 vs. 11.25 months; $p=1x10^{-4}$). Visualizing regional authorization differences highlights key international regulatory disparities, providing vital actionable insights for global tumor boards.

Ultimately, OncoAID bridges the gap between computational bioinformatics and bedside care by offering an open-access, actionable digital health resource for interactive exploration, data mining, and machine learning model development. By harmonizing trial outcomes, experimental pharmacology, and AI summarization, OncoAID serves as a practical tool for global Precision Oncology workflows.

27. Striking back: An integrated multiomic clinical decision support platform to restore treatment response after standard therapy failure

Limaye S [1]*, Patil D [2], Kumar P [2,3], Datta V [2], Akolkar D [2], Kataria P [1], Shah D [1], Patel R [1], Regmi S [1], Shah N [1], George I [2], Sambath J [2], Parab A [1], Singh S [1], Madre C [1], Deshpande R [1], Khatri M [3], Pardikar S [1], Srivastava N [3], Schuster S [2], Shreenivas A [4] , Crook T [5], Datar R [2], Kurzrock R [6]

[1] Sir H.N. Reliance Foundation Hospital and Medical Research Centre, India
[2] Datar Cancer Genetics, India
[3] R.D. Gardi Medical College, Ujjan, India
[4] City of Hope, USA
[5] Cromwell Hospital, UK
[6] Medical College of Wisconsin, USA

Background:
Patients with refractory metastatic cancer and exhausted standard-of-care options have limited actionable options from DNA-only comprehensive genomic profiling. Exacta, an integrated multiomic clinical decision support platform combining tissue and ctDNA genomics, transcriptomics, germline and pharmacogenetic profiling, and theranostic IHC-based markers, may identify additional therapeutic options in this setting.

Methods:
This single-practice, retrospective, real-world study evaluated outcomes in 138 patients with stage IV cancer treated on the basis of Exacta-informed therapy. Within-patient treatment response and PFS were compared between the most recent systemic therapy immediately preceding Exacta profiling (PFS1) and subsequent Exacta-guided therapy (PFS2), using the Von Hoff ratio (PFS2/PFS1); a ratio ≥1.3 was pre-specified as the benchmark for meaningful clinical benefit.

Results:
Von Hoff ratio was evaluable in 118/138 (85.5%) patients (34 tumor types; predominantly head and neck 16.1%, breast 14.4%, lung 9.3%). 75.4% (89/118) of these patients had received ≥2 prior systemic therapy lines, and 21.2% (25/118) had received ≥4 prior systemic therapy lines. PFS was longer with Exacta-guided therapy in 61.9% (73/118), unchanged in 2.5% (3/118), and shorter in 35.6% (42/118). The median Von Hoff ratio was 1.45. Overall, 55.9% (66/118) of patients met the pre-specified ≥1.3 benefit threshold, including 33.9% (40/118) with at least a two-fold PFS increase (Table 1).
Among evaluable patients, ORR was 13.2% with preceding therapy versus 29.4% with Exacta-guided therapy; DCR was 29.3% versus 66.1%, respectively. Exacta-guided therapy produced a complete response in 8 patients, a response category not observed with prior systemic therapy.

Conclusion:
This real-world, single-practice cohort study of heavily pretreated, refractory metastatic cancer demonstrates that Exacta-guided therapy, informed by an integrated multiomic clinical decision support platform, meaningfully restores treatment response after standard-of-care failure -including complete responses not seen with prior line of systemic therapy. This within-patient benefit was accompanied by a concordant increase in independently assessed radiological response, reinforcing the Von Hoff ratio as a meaningful measure of clinical benefit. These findings support a favourable shift in outcomes and warrant prospective validation in a larger, multi-site cohort.

Table 1: Distribution of patients according to Von Hoff PFS ratio

Von Hoff PFS ratio	Patients, % (n)
>1.0	64.4 (76/118)
>1.3	55.9 (66/118)
>1.5	48.3 (57/118)
>2.0	33.9 (40/118)

28. Decoding the Genomic Landscape of Gynaecological Cancers in India: Real-World Insights from 598 Tumors

Limaye S [1], Kumar P [2,3] *, Patil D [2], Chauhan R [4], Shankar A [5], Kataria P [1], Shah D [1], Patel R [1], Regmi S [1], George I [2], Sambath J [2], Parab A [1], Singh S [1], Madre C [1], Deshpande R [1], Pardikar S [1], Khatri M [3], Srivastava N [3], Dalmia J [3], Datar R [2], Mukhopadhyay A [6]

[1] Sir H.N. Reliance Foundation Hospital and Medical Research Centre, India
[2] Datar Cancer Genetics, India
[3] R.D. Gardi Medical College, Ujjan, India
[4] Mahavir Cancer Sansthan, Patna, India
[5] All India Institute of Medical Sciences, Delhi, India
[6] James Cook University Hospital and Northern Cancer Alliance, UK

Background:
Gynaecological malignancies represent a major cancer burden in India, yet genomic data characterizing these cancers in Indian populations remain limited. Understanding population- and histotype-specific molecular alterations may help identify clinically relevant biomarkers and opportunities for Precision Oncology.

Methods:
We performed targeted next-generation sequencing of 598 gynaecological malignancies, comprising ovarian (n=383), endometrial (n=121), cervical (n=66), uterine sarcoma (n=20), and rare gynaecological cancers (n=8). Somatic mutations and copy-number alterations were characterized, with tumor mutational burden (TMB), microsatellite instability (MSI), and PD-L1 expression evaluated in available subsets. Genomic findings were assessed across histological subtypes and compared with publicly available datasets, including The Cancer Genome Atlas (TCGA).

Results:
Ovarian cancers were characterized predominantly by TP53 alterations (59%), with distinct histotype-associated genomic patterns. PI3K/PTEN pathway alterations were enriched in endometrioid tumors, PIK3CA alterations were prominent in clear-cell carcinomas, and KRAS mutations were recurrent in mucinous tumors. CCNE1 amplification emerged as a recurrent copy- number alteration and frequently co-occurred with TP53 alterations, particularly in serous and endometrioid tumors. Endometrial cancers demonstrated frequent alterations in TP53 (55%), PTEN (39%), and PIK3CA (35%), with distinct genomic profiles between Type I and Type II tumors. Notably, MSI-high status was observed in 8.9% of ovarian cancers and 18.1% of endometrial cancers, with differences in prevalence compared with TCGA cohorts.

Conclusions:
This multi-cohort real-world analysis provides a comprehensive overview of the genomic landscape of gynaecological malignancies in an Indian population. The observed histotype-specific molecular patterns and differences in MSI prevalence compared with TCGA highlight the heterogeneity of gynaecological cancers and the potential value of region-specific genomic profiling. These findings support broader integration of molecular characterization into clinical research and Precision Oncology strategies for gynaecological cancers in India.

29. Cytoskeletal and Metabolic Reprogramming in Polyploid Giant Cancer Cells: Mechanisms Supporting HGSOC Progression

Lu X, Ghosh D, Dawson M
Brown University, Providence, RI, USA

High-Grade Serous Ovarian Cancer (HGSOC) is the most common, lethal, and aggressive subtype of epithelial ovarian cancer. Most patients are diagnosed with advanced stage disease, which is associated with a dramatic decline in five-year survival, from approximately 93% for early-stage to 30% for advanced-stage disease, and an increase in recurrence rates from 10% to approximately 90%. Polyploid giant cancer cells (PGCCs) are a treatment-resistant subpopulation of cancer cells. They resist chemotherapeutic stress due to their dormant state and can later exit dormancy and give rise to treatment-resistant and tumorigenic daughter cells through amitotic budding. PGCCs are more commonly identified in late-stage tumors and have been associated with increased recurrence and reduced survival. They are also enriched in circulating tumor cells, consistent with a potential role in tumor dissemination. Together these findings support the emerging role of PGCCs as a major driver of treatment resistance, metastasis, and tumor relapse.

Our lab successfully developed in vitro models of PGCCs that recapitulate key features of the PGCC phenotype, including chemotherapeutic resistance, dormancy, and the generation of proliferative daughter cells through amitotic budding. PGCCs are highly heterogeneous with markedly increased size and aberrant nuclear morphology. Through analysis of patient tissues, we identified epithelial and mesenchymal markers strongly associated with PGCCs and validated their differential expression using our in vitro cell models, supporting their utility as PGCC biomarkers.

Our studies further demonstrate that PGCC formation is accompanied by extensive cytoskeletal and metabolic reorganization. EMT-associated intermediate filaments, including cytokeratin and vimentin, exhibit distinct structural organization in PGCCs compared to non-PGCCs. We found that vimentin contributes to the enhanced migratory persistence of PGCCs, providing a potential mechanism underlying their increased metastatic capacity. Cytokeratin and vimentin may also contribute to the altered intracellular organization of mitochondria observed in PGCCs, linking cytoskeletal remodeling to changes in organelle organization and metabolic function.

To support their larger size and increased nutrient demands, PGCCs are known to undergo metabolic reprogramming. We found that PGCCs exhibit mitochondrial dysfunction and increased oxidative stress, accompanied by a shift toward a glycolysis-dependent metabolic phenotype. Collectively, our findings identify interconnected cytoskeletal, migratory, and metabolic adaptations that may enable PGCCs to survive adverse conditions and contribute to treatment resistance, metastasis, and tumor recurrence. These phenotypes also reveal potential vulnerabilities that could be exploited through strategies targeting cytoskeletal organization, metabolic dependencies, and dietary modifications.

30. CCCS001: A Prospective Real-World Observational Study Evaluating Functional Precision Oncology Approaches for Personalized Cancer Therapy

Nugent A [1,2], Bakker U [1,2], Pillai P [1], Reid C[1]
[1] Cancer Commons, Palo Alto, CA, USA
[2] Brain Cancer Support and Solutions Alliance, Palo Alto, CA, USA

Background:
Personalized oncology has expanded rapidly, yet limited real-world evidence exists comparing different treatment-selection approaches for identifying effective personalized therapies beyond standard clinical guidelines. CCCS001 is a prospective, decentralized observational study designed to evaluate the predictive performance of functional Precision Oncology assays for selecting FDA-approved drugs as off- guideline therapies in patients with advanced cancer.

Methods:
Patients with advanced cancers who undergo genomic profiling, functional drug sensitivity testing (DST), or other Precision Oncology assays are eligible for enrollment. Cancer Commons, a neutral nonprofit scientific organization, synthesizes testing results with published clinical evidence to identify evidence-based treatment options and facilitate discussions between patients and their treating oncologists. Treatment decisions remain entirely at the discretion of the treating oncologist. The registry prospectively captures patient characteristics, testing results, treatment recommendations, therapies received, implementation barriers, clinical response, progression-free survival, and overall survival.

Results:
CCCS001 establishes a real-world evidence platform for evaluating functional precision medicine alongside established molecular profiling approaches across diverse tumor types and practice settings. The primary objective is to determine the predictive accuracy of treatment selection tests in selecting off-guideline drugs. In addition to capturing testing results and treatment outcomes, this study also captures operational factors influencing adoption, including tissue availability, testing accessibility, physician acceptance of recommendations, treatment access, insurance barriers, and patient engagement. These longitudinal data will help characterize the clinical utility of DST, identify patient populations most likely to benefit, and evaluate how functional testing compares to genomic and transcriptomic profiling in guiding personalized therapy.

Conclusions:
CCCS001 provides a scalable real-world evidence framework for evaluating personalized oncology strategies while enabling patients to remain under the care of their local oncologists. By systematically capturing molecular profiling, drug sensitivity testing, treatment selection, and longitudinal outcomes, the study seeks to generate evidence on how functional precision medicine can identify new therapeutic opportunities beyond standard of care. In addition to evaluating clinical utility, CCCS001 aims to expand patient access to individualized treatment options by helping patients and their oncologists interpret and act on drug sensitivity testing results, ultimately informing the optimal integration of Precision Oncology into routine clinical practice.

31. GBM Precision Ecosystem, Closing the Glioblastoma Ignorance Gap - A patient-led framework to preserve personalized treatment options early enough to matter.

Rafael Palhinha Gomes, GBM patient

Global glioblastoma patients and caregivers face extreme time pressure while processing complex medical choices and profound emotional shock. The core challenge is the ignorance gap: doctors do not always offer, and families do not always know, which questions must be asked early to preserve treatment options. We propose redesigning the early workflow so that tissue, data, testing, and clinical decisions are coordinated from the start. By preserving living tumor tissue, organizing the data, and using AI- supported decision tools, families who want to engage more actively in their care can work with their physicians to co-design a more personalized treatment pathway.

32. Changes in PDL1 and VEGF expression after short- and long-term exposure to antibody-drug conjugates and payloads in human ovarian cancer cell lines

Pearce J, De la Cruz P, Buyungo S, Salaverria A, Bojko A, Mathews CA, Disilvestro P, James N
Program in Women's Oncology, Women and Infants Hospital, Providence, RI, USA

Introduction:
Antibody-drug conjugates (ADCs) are becoming more prevalent for treatment of gynecologic oncology malignancies. However, there is limited data regarding mechanisms of resistance in this landscape; furthermore, there is little knowledge regarding the impact of ADC and payload treatments on subsequent protein expression for other drug targets, such as bevacizumab (targets VEGF) and durvalumab (targets PDL1). The purpose of this study was to better understand the short- and long-term effects of ADC and payload treatment on PDL1 and VEGF protein expression in human ovarian cancer cell lines.

Methods:
Two commercially available human high grade serous ovarian cancer cell lines (PEA1 and PEA2) were each exposed separately to three commercially available payloads with corresponding, clinically-used ADCs [deruextecan (DXD)/trastuzumab deruxetecan (TDXD), SN38/sacituzumab govitecan (SG), and DM4/mirvetuximab soravtansine (MIRV)] for 48 hours. Western blot analyses were performed to evaluate for short term changes in PDL1 and VEGFA expression. Next, DXD- and DM4-resistant cell lines were created using long-term, sequential escalation of payload treatment exposure. Cell viability assays were performed to confirm and quantify resistance after completion of all treatments, and western blot analyses were performed to evaluate changes in PDL1 and VEGFA expression between payload-resistant cells and their corresponding payload-sensitive controls.

Results:
Short term treatment of chemosensitive PEA1 cells with TDXD ADC did not result in significant changes in PDL1 or VEGFA, but DXD payload treatment resulted in significantly decreased PDL1 expression. Chemoresistant PEA2 cells treated with TDXD expressed less VEGF compared to those treated with DXD, those there were not statistically different than controls. PEA1 cells treated with MIRV ADC expressed significantly more VEGFA compared to controls, while no difference was noted after DM4 payload exposure. Conversely, PEA2 cells treated with MIRV did not have a significant difference in VEGFA expression, while DM4 treatment resulted in significantly decreased VEGFA expression. While some trends in PDL1 expression were visualized, none were statistically significant.

Treatment of PEA1 cells with SG ADC resulted in significantly increased PDL1 expression compared to controls and a trend towards less VEGFA, though this was not statistically significant. VEGFA expression was significantly decreased after SN38 payload treatment. PEA2 cells treated with SG had no significant changes in PDL1 or VEGF, though SN38 payload treatment resulted in significantly decreased PDL1 expression.

Payload-resistant cell lines were created and validated; compared to payload-sensitive controls, PEA1 and PEA2 DXD- and DM4-resistant cells all displayed similar morphologic changes including increased surface area. All resistant cell lines demonstrated decreased VEGFA expression compared to payload-sensitive controls.

Conclusion:
This study demonstrates that ADC and payload treatments result in variable expression changes of PDL1 and VEGFA. Additionally, the differences in short- and long-term exposure results illustrate the importance of model selection in studies aimed towards understanding subsequent drug treatment implications. Future work is underway to expand these findings to other drug targets, and to further explore the implications of different drug sequencing on ovarian cancer treatment.

33. Biomarker-based efficacy of oncolytic herpes virus therapy for the treatment against NF1-associated malignant peripheral nerve sheath tumor.

Potti S, Stehn C, Largaespada D, Galvin R
University of Minnesota - Twin Cities, USA

Introduction:
Malignant peripheral nerve sheath tumors (MPNSTs) are the leading cause of death in individuals with Neurofibromatosis Type 1. The polycomb repressive complex 2 negatively regulates gene expression via trimethylation of histone 3.3 (H3K27me3), and its loss of function characterizes high- grade MPNST. Oncolytic herpes simplex virus (oHSV) represents a novel treatment avenue, but host cell repression of viral replication is a resistance mechanism. We hypothesize that PRC2 loss, characteristic of MPNST, enhances susceptibility to oHSV, as transcriptional silencing of viral replication may be dysregulated.
Methods:
MPNST cell lines were infected with the oncolytic virus HSV-G47Δ in vitro, with multiplicities of infection (MOI) ranging from 3 to 0. Cell viability was assessed via MTS assay after 6 days. The half- maximal inhibitory concentration was determined by non-linear regression in GraphPad Prism. All experiments included three biological replicates.
Results:
HSV-G47Δ demonstrated activity across all tested MPNST cell lines (S462-TY, ST88-14, MPNST724, and STS26T) at MOI < 1.0. Pharmacologic inhibition of PRC2 did not affect HSV-G47Δ activity in the PRC2-deficient S462-TY ($p = 0.8$) but enhanced susceptibility in PRC2-proficient MPNST724 cells ($p < 0.0001$). S462-TY was engineered to express wild-type SUZ12 to restore PRC2 function under a tetracycline-controlled transcriptional activation system. Following confirmation of H3K27me3 restoration via Western blot with doxycycline exposure, PRC2 proficiency in this add-back model increased resistance to HSV-G47Δ ($p < 0.0001$). Finally, cell viability assays were conducted in murine syngeneic MPNST models: 1) Nf1 loss of function (QmSC-N), 2) Nf1/Tp53 knock-out (JW16), and 3) Nf1/Cdkn2a/Cdkn2b/SUZ12 knock-out (QmSC-NCS). The PRC2 deficient QmSC-NCS model was most sensitive ($p < 0.0001$) while QmSC-N was resistant.
Conclusion:
Our results demonstrate that PRC2 status may serve as a biomarker for HSV- G47Δ sensitivity in MPNST, providing a therapeutic direction. Future experiments include mechanistic evaluation of our results with methylation assays and planned in vivo trials.

34. Expression of co-MDM4-amplified chromosome 1q32 genes in high-grade gliomas supports precision therapy approaches

Raissi A [1], Horowitch B [2], Egg-Krings C [1], and El-Deiry W [1]
[1] Brown University Department of Pathology and Laboratory Medicine, Providence, Rhode Island, USA
[2] Department of Biology, Johns Hopkins University, Baltimore, Maryland, USA

Amplification of the p53 suppressors MDM2 and MDM4/MDMX is associated with poor outcomes across multiple tumor types and has been linked to the phenomenon of hyperprogression following immunotherapy. Arnoff & El-Deiry, 2022 (PMID: 35693093) previously described that approximately 10% of high-grade gliomas (HGG) have MDM4 amplification and such tumors lack specific precision targeted therapies. Additional studies by El-Deiry et al., 2022 (https://ascopubs.org/doi/10.1200/JCO.2022.40.16_suppl.2630) documented poor patient survival with MDM4 amplification across multiple tumor types. In exploring a Precision Oncology therapeutic approach for the subset of MDM4-amplified brain tumors, we investigated the correlation between gene amplification and mRNA expression of MDM4 and co-amplified genes. If the amplified genes are not expressed then therapeutic strategies using existing kinase and/or phosphatase inhibitors would not be expected to have efficacy due to lack of the mRNA or protein targets. Co-amplified genes within the MDM4 amplicon include the kinase PIK3C2B and the PP1 regulatory subunit PPP1R15B. Continuous copy-number–expression analyses were performed using downloaded segmented copy-number and RNA-seq TPM z-score data from the TCGA glioblastoma cohort (GDC 2025; gbm_tcga_gdc; accessed July 10, 2026). MDM4 copy number showed a modest monotonic association with mRNA expression (Spearman $\rho = 0.248$) and a stronger Pearson correlation ($r= 0.699$) in the TCGA GDC 2025 cohort. In the same cohort ($n = 519$ specimens with copy-number data), all 40 MDM4-amplified specimens also carried PIK3C2B amplification and 39 of 40 carried PPP1R15B amplification (log2 odds ratio > 3, $q < 0.001$ for both), identifying additional candidates for co- targeting on Chr 1q32.1. The correlation of PIK3C2B copy number and relative PIK3C2B mRNA expression (TPM z-score) in matched TCGA glioblastoma specimens was demonstrated (GDC 2025; $n = 277$; estimated copy-number range, 1–16; Spearman $\rho = 0.195$; Pearson $r = 0.606$; Least-squares fit). The correlation of PPP1R15B copy number and relative PPP1R15B mRNA expression (TPM z-score) in matched TCGA glioblastoma specimens was demonstrated (GDC 2025; $n = 277$; estimated copy-number range, 1–16; Spearman $\rho = 0.351$; Pearson $r = 0.875$; Least-squares fit). Co-MDM4 amplified genes are overexpressed at the mRNA level supporting the notion that they may be targetable with existing therapeutics. Further studies are required to advance this precision therapy concept towards translation in the clinic in the subset of aggressive high-grade gliomas with MDM4 amplification.

35. A Precision Oncology Approach to Geriatric Cancer Care: Real-World Experience with Comprehensive Genomic Profiling and Clinical Outcomes

Regmi S [1], Jain S [2], Parab P [1], Limaye S [1], Pardikar S [1], Khatri M [4], Bhatia G [1], Madre C[1], Patil D [3], Kumar P [3,4], George G [3], Sambath J [3], Kataria P [1], Patel R [1], Shah D [1], Jha S [1], Dubey S [1], Dalal R [1], Mazumder A [1], Bansal A [1], Wankhede S [1], Bharadwaj S [1], Chauhan P [1], Limaye S [1]*

[1] Sir H.N. Reliance Foundation Hospital and Medical Research Centre, India

[2] Dhirubhai Ambani International School, India

[3] Datar Cancer Genetics, India

[4] R.D. Gardi Medical College, Ujjan, India

Background:

Older adults with cancer are clinically heterogeneous, with varying degrees of frailty, comorbidity, and treatment tolerance. Although potentially actionable genomic alterations are present in this population, geriatric patients remain under-represented in Precision Oncology evidence. In real-world geriatric oncology practice, the clinical utility of comprehensive genomic profiling (CGP) and its impact on patient outcomes remain poorly characterized. We evaluated real-world CGP utilisation, clinical utility, and outcomes in geriatric patients with solid tumors managed in our Precision Oncology Clinic.

Methodology:

We conducted a retrospective observational study of 563 older adults (≥65 years) with solid malignancies. Based on the availability and performance of comprehensive genomic profiling (CGP) as part of routine clinical care, patients were classified into two cohorts: Cohort A (CGP cohort), who underwent CGP testing, and Cohort B (non-CGP cohort), patients in whom CGP testing was not performed. Clinical demographics, molecular landscape, treatment received, and outcome data were collected from medical records. Geriatric status was assessed using ECOG performance and the Charlson Comorbidity Index (CCI), and patients were categorised as fit, intermediate, or frail. In Cohort A, CGP findings were evaluated for clinically actionable genomic alterations in various cancer subtypes. Treatment response at 3 months and progression-free survival (PFS) were assessed from available clinical and radiological records for both the cohorts.

Results:

Among 563 older adults evaluated at the Precision Oncology Clinic, the median age was 70 years in Cohort A and 72 years in Cohort B; overall, 324 (57.5%) patients were male and 239 (42.5%) were female. Gastrointestinal and thoracic malignancies were the predominant tumour groups. Patients were divided on the basis of the availability of genomic report in two group: cohort A (CGP cohort) and cohort B (non-CGP Cohort). 405 (71.9%) comprised cohort A (CGP cohort), while 158 (28.1%) comprised cohort B (non-CGP cohort). Geriatric assessment incorporating ECOG performance status and Charlson Comorbidity Index (CCI) categorised patients as fit, intermediate, or frail. In Cohort A, 189 (46.7%) patients were fit, 143 (35.3%) were intermediate, and 73 (18.0%) were frail, compared with 80 (50.6%), 35 (22.2%), and 43 (27.2%), respectively, in Cohort B.

CGP identified clinically actionable genomic alterations in 294/405 (72.6%) patients in Cohort A TP53 (35%), EGFR (20%), PIK3CA (16%), KRAS (12%), ERBB2 (9%), and BRAF (7%) were the most frequently altered genes, with EGFR (n=64) alterations occurring predominantly in thoracic malignancies, PIK3CA (n=21) and ERBB2 (n=9) alterations predominantly in breast and gynaecological malignancies, KRAS (n=34) alterations primarily in thoracic and gastrointestinal malignancies, and BRAF (n=7) alterations distributed across thoracic, gynaecological, and CUP tumours. In addition, CGP identified immunotherapy-relevant biomarkers, including a high TMB-positive PD-L1 score (more than 1) (n=14), and MSI-H status (n=6), enabling biomarker-guided immunotherapy in selected patients. The median number of prior systemic therapies was 2 in both cohorts.

In the CGP Cohort , 221 patients received precision-guided treatment, 111 received standard care, 16 received best supportive care and 57 were lost to follow-up; in the Non-CGP Cohort , 134 received standard

clinical care and 24 received best supportive care. At 3 months, among response-evaluable patients, the CGP Cohort demonstrated 18 complete responses (CR), 139 partial responses (PR), 87 stable disease (SD), and 74 progressive disease (PD), compared with 2 CR, 71 PR, 45 SD, and 9 PD in the non-CGP cohort. Median PFS was 6.8 months in the CGP cohort and 2.6 months in the non-CGP cohort, with PFS assessed independently within each cohort, which was statistically significant.

Conclusion:

Comprehensive genomic profiling demonstrated meaningful clinical utility in geriatric oncology, with actionable genomic and biomarker findings translated into individualised treatment across a clinically heterogeneous older population. Importantly, treatment translation extended to patients with varying degrees of frailty and comorbidity, suggesting that geriatric vulnerability should not, by itself, preclude consideration of Precision Oncology. Integrating genomic profiling with geriatric assessment may enable biologically informed, individualised cancer care for older adults.

36. Rapid drug response profiling in malignant ascites through morphology-based predictive modeling

Ruehr A [1], Al-Jazrawe M [1,2], Rindtorff N [2], Hakimian D [1], Miglietta E [2], Ostrovsky N [1], Abeyta E [1,2],Nanda N [1], Barnes H [3], Simms C [3], Bannon S [3], Blum S [3], Park H [4], Cimini B [2], Klempner S [3], Boehm J [1,2]

[1] Koch Institute for Integrative Cancer Research, MA, USA
[2] Broad Institute of MIT and Harvard, MA, USA
[3] Massachusetts General Hospital, MA, USA
[4] Dana-Farber Cancer Institute, MA, USA

Malignant ascites is associated with particularly poor prognosis in gastroesophageal cancer. Most patients survive only weeks to months after onset due to treatment failure. Ascites contains a heterogeneous population of cancer, immune, and stromal cells, in addition to a cytokine-rich microenvironment, representing a potentially valuable source of biological and clinical information. Additionally, these samples are routinely collected yet discarded. We therefore see a scientific and clinical opportunity to enable the study of this rich resource.

Here we show the development of a platform for rapid functional profiling of cancer cells from patients with malignant ascites. We integrate viable sample processing, drug perturbation, imaging, and analysis to produce screening results in under 24 hours using automation and computer vision. First, we optimized the ex vivo culture conditions by incorporating microenvironmental factors by mixing ascites fluid with organoid media. Next, we hypothesized that label-free microscopy can provide a functional readout of drug response as a substitute for fluorescence staining which fades over time and causes cytotoxicity.

To test this hypothesis, we collected over 24 million images from over 40 samples and trained a gradient-boosted machine learning model to identify viable cancer cells using only brightfield morphology features. In unseen samples, we observed a precision score of 0.89 and recall score of 0.88, supporting our hypothesis. We then applied our label-free computer vision model to drug- perturbation experiments with 25 clinically and biologically relevant small molecules. Using this approach, our image-based functional platform was able to generate drug response profiles and identified diNerential response such as expected resistance pattern of a sample carrying TP53 R273H and R175H mutations to AMG-232, an MDM inhibitor, supporting that our ascites platform can recapitulate known genotype-phenotype relationships in the primary tumor. Moreover, we observed diNerential response to chemotherapeutics, suggesting that our platform can potentially link clinical information with functional response to guide treatment choice. Results from serially collected samples from the same patients ($N = 3$, $r > 0.85$) further support biological reproducibility.

Taken together, these findings support the use of label-free, morphology-based profiling as a rapid approach for functional characterization of malignant ascites. Since our platform couples the timing of perturbation with the preservation of cellular heterogeneity, it has the potential to serve as a strong foundation for preclinical studies aimed at understanding tumor resistance or tumor- immune interactions. Moreover, our automated workflow enables the potential of using early images to guide adaptive perturbation testing and rational drug combination nomination for each patient sample.

37. PPARD Is the PPAR Family Member Most Strongly Associated with Aggressive Breast Cancer Phenotypes and Poor Clinical Outcome

Ryspayeva D [1], Si K[1], Egg-Krings C [2], Wen B[1], Navaraj A [2], and El-Deiry W [1,2,3].
[1] Brown University, Legorreta Cancer Center, Providence, RI, USA
[2] The Warren Alpert Medical School, Brown University, Providence, RI, USA
[3] Brown University Health, Providence, RI, USA

Background:
Peroxisome proliferator-activated receptors (PPARs: PPARβα, PPARβ/δ, and PPARγ) are lipid-sensing nuclear receptors that regulate lipid metabolism, energy homeostasis, and cellular stress adaptation. Dysregulated PPAR signaling has been implicated in metabolic reprogramming, tumor progression, and adverse clinical outcomes across multiple cancer types. Although increasing evidence suggests that the PPAR family plays a role in tumor development, the relative clinical significance and isoform-specific contributions of individual family members in breast cancer remain incompletely defined.
Objective:
To compare the clinical and molecular associations of PPARs (encoded by PPARA, PPARD, and PPARG) in breast cancer and determine whether PPARD is preferentially linked to aggressive disease biology.
Methods:
Datasets: Integrated breast cancer cohorts from TCGA (n=975) and METABRIC (n= 1980; combined N=2,955).
Gene expression analysis: Evaluated PPARA, PPARD, and PPARG expression across breast cancer molecular subtypes.
Survival analysis: Kaplan–Meier overall survival (OS) analyses were performed using median cutoffs and quartile-based expression stratification.
Subtype analysis: Associations between PPAR isoform expression and PAM50 molecular subtypes were assessed in TCGA and validated in METABRIC.
Results
Prognostic impact:
Elevated PPARD correlated with significantly inferior OS in METABRIC (HR = 1.170, p=0.0082), showing a progressive OS decline across expression quartiles (p = 0.00027).
Subtype dynamics: PPARD expression was highest in Basal-like and HER2-enriched tumors and lowest in Luminal A/B subtypes. Increasing PPARD expression showed progressive enrichment of Basal-like disease alongside a corresponding reduction in luminal tumors.
Isoform divergence: In contrast to PPARD, PPARG expression was predominantly restricted to luminal subtypes.
Validation:
Subtype enrichment and survival trends for PPARD were independently validated across both TCGA and METABRIC cohorts (N = 2,955).
Conclusions:
Comparative analysis of the PPAR family identifies PPARβ/δ (encoded by PPARD) as the isoform most strongly associated with aggressive breast cancer biology. Elevated PPARD expression correlates with Basal-like/TNBC phenotypes and poor patient outcomes, highlighting PPARD as a promising prognostic biomarker and an actionable therapeutic target in high-risk disease.

38. Precise functionalization of DNA scaffold particles for enhanced NK cell antitumor efficacy

Sallam M, Jingge Chen, Deblin Jana, Tejal Desai
School of engineering, Brown University, RI, USA

Introduction:

Natural killer (NK) cells play a central role in innate anticancer immunity through their ability to recognize and kill cancer cells without prior antigen priming. Despite their therapeutic promise, clinical translation of NK cell–based immunotherapies has been hindered by limited persistence, functional exhaustion, and insufficient activation within the immunosuppressive tumor microenvironment. Strategies that enable precise and sustained engagement of activating receptors on NK cells, while simultaneously promoting tumor- specific targeting, are of significant interest.

Engineered NK cell engagers and biomaterial-based delivery platforms are promising approaches to address these challenges by improving spatial control and multivalency of immune activation. While Surface-functionalized polymeric particles offer opportunities to present immune ligands in defined densities and ratios, conventional conjugation strategies often lack the modularity and quantitative control needed to systematically optimize immune receptor engagement.

Here, we introduce a biocompatible DNA-scaffolded poly(lactic-co-glycolic acid) (PLGA) particle platform that enables ratiometric and programmable antibody loading. This system is designed to precisely control the presentation of NK cell–activating antibodies alongside tumor-targeting ligands, providing a platform to enhance NK cell mediated cytotoxicity against solid tumors.

Materials and Methods:

PLGA particles were fabricated using an emulsion-based technique and surface- functionalized with short synthetic DNA oligonucleotides via a PLGA–PEG–DNA conjugation strategy. Complementary DNA sequences were used as modular scaffolds to enable quantitative and ratiometric attachment of monoclonal antibodies (mAbs) through sequence-specific DNA hybridization. Selected mAbs targeting NK cell activating receptors, together with an anti-EGFR antibody, were conjugated to the particle surface at defined densities and ratios.

Particle size and antibody loading efficiency were characterized using electron microscopy, and fluorescence-based quantification. A series of formulations with varying antibody compositions were evaluated in vitro for their ability to promote NK cell activation and cytotoxicity against colorectal and glioblastoma cancer cell lines.

In vivo efficacy was evaluated in colorectal tumor–bearing mice following local administration of selected immune-engaging particle formulations, with tumor growth kinetics and survival monitored as study endpoints.

Results, Discussion and Conclusions:

DNA scaffolding enabled precise, reproducible, and ratiometric loading of multiple antibodies onto PLGA particles, overcoming key limitations of conventional surface conjugation approaches. Systematic modulation of surface antibody density and composition demonstrated that NK cell activity was strongly dependent on both the total antibody surface density and the relative stoichiometric ratios of the conjugated antibodies. An optimized ratio achieved maximal enhancement of NK-mediated cytotoxicity against cancer cells in vitro, compared to single-ligand or non-optimized controls across both colorectal and glioblastoma models.

In vivo study showed that intratumoral administration of immune-engaging particles resulted in enhanced NK cell antitumor activity and significantly prolonged survival compared to control treatments.

Together, these results indicate that quantitative control of immune ligand surface presentation is an important design consideration for engineering immune-engaging biomaterials to support NK cell–mediated immunotherapy. Additionally, this study demonstrates that DNA-scaffolded PLGA particles provide a tunable platform for controlled immune cell engagement and offer practical design guidance for the development of NK cell–based therapeutic strategies in solid tumor models.

39. Biomarker-Stratified First-Line Chemotherapy Outcomes in Pancreatic Ductal Adenocarcinoma Using CARIS CODEai: TACSTD2 (TROP2) and RRM1 Across FOLFIRINOX Versus Gemcitabine/nab-Paclitaxel

Samihah Ibrahim [1]; Wafik S. El-Deiry [1,2]
[1] Legorreta Cancer Center at Brown University, Providence, Rhode Island, USA.
[2] Laboratory of Translational Oncology and Experimental Cancer Therapeutics, Department of Pathology & Laboratory Medicine, The Warren Alpert Medical School of Brown University, The Joint Program in Cancer Biology, Brown University and Lifespan Health System, Division of Hematology/Oncology, The Warren Alpert Medical School of Brown University, Providence, RI,USA

Background:
First-line systemic therapy for pancreatic ductal adenocarcinoma (PDAC) commonly includes FOLFIRINOX (FFX) or gemcitabine/nab-paclitaxel (Gem/nab), but clinically useful biomarkers to refine regimen selection remain limited. We used CARIS CODEai to evaluate TACSTD2 (TROP2) and RRM1 transcript expression as biomarkers associated with outcome within each regimen and across biomarker-defined treatment strata.
Methods:
Using the CARIS CODEai platform, cohorts of PDAC patients treated with first-line FFX or Gem/nab were constructed. TACSTD2 and RRM1 were analyzed by RNA quartiles, comparing Q1 versus Q4. Kaplan-Meier analyses used the endpoint first of FOLFIRINOX, gemcitabine, nab-paclitaxel to last contact. Analyses included: (1) within-regimen comparisons of Q1 versus Q4, and (2) regimen-versus-regimen comparisons within the same biomarker stratum (FFX vs Gem/nab in Q1 and Q4).
Results:
Within-regimen analyses showed prognostic separation for TACSTD2 in both treatment groups. In FFX, TACSTD2 Q1 demonstrated longer median survival than Q4 (16.812 vs 13.982 months; HR 0.849, 95% CI 0.72- 1.001; p=0.05). In Gem/nab, TACSTD2 Q1 also outperformed Q4 (14.048 vs 11.350 months; HR 0.771, 95% CI 0.595-0.998; p=0.047). For RRM1, prognostic separation was observed in Gem/nab but not clearly in FFX. In Gem/nab, RRM1 Q1 had longer median survival than Q4 (15.364 vs 9.837 months; HR 0.641, 95% CI 0.492- 0.835; p<0.001), whereas in FFX the difference between RRM1 Q1 and Q4 was smaller and not statistically significant (16.582 vs 15.298 months; HR 0.929, 95% CI 0.790-1.094; p=0.378).
In biomarker-stratified regimen comparisons, FFX was associated with longer survival than Gem/nab in TACSTD2-defined strata. In TACSTD2 Q1, median survival was 16.812 months with FFX versus 14.048 months with Gem/nab (HR 0.765, 95% CI 0.618-0.947; p=0.014). In TACSTD2 Q4, median survival was 13.982 versus 11.350 months, respectively (HR 0.678, 95% CI 0.545-0.844; p<0.001). For RRM1-defined strata, no significant difference was observed in Q1 (16.582 vs 15.364 months; HR 0.890, 95% CI 0.713-1.110; p=0.300), while FFX was associated with longer survival in RRM1 Q4 (15.298 vs 9.837 months; HR 0.594, 95% CI 0.480-0.736; p<0.00001).
Conclusions:
In this CARIS CODEai analysis of first-line PDAC, TACSTD2 demonstrated consistent prognostic stratification within both FFX and Gem/nab, while RRM1 showed stronger prognostic separation within Gem/nab than FFX. In biomarker-stratified regimen comparisons, FFX was associated with superior survival in TACSTD2 Q1, TACSTD2 Q4, and RRM1 Q4, whereas outcomes were similar between regimens in RRM1 Q1. These findings support further evaluation of TACSTD2 and RRM1 as transcript-based biomarkers for treatment stratification in PDAC.

40. Understanding and targeting p53 impairment in advanced cancer - Transitioning into the clinic

Sarlis N [1] , Baar M [2,3] , Bouma E [2,3] , Putavet D [2,3] , Teifel M [2] , Madl T [4] , de Keizer P [2,3]
[1] CLEARA Biotech BV (based in Greenville DE, United States)
[2] CLEARA Biotech, BV, Utrecht, The Netherlands (Company HQ address)
[3] Center for Molecular Medicine, University Medical Center Utrecht, Utrecht, The Netherlands
[4] Research Unit Integrative Structural Biology, Medicinal Chemistry, Otto Loewi Research Center, Medical University of Graz, Graz, Austria

Background:
The inability to target distant metastases severely limits clinical outcomes, such as survival, symptom burden, and quality of life in patients with late- stage cancers. This is driven in part by a lack of optimized biologically targeted therapies and resistance to standard chemo(bio)/radiotherapy. In healthy, non-neoplastic cells, impairment in p53 function leads to transcription-independent apoptosis. In cancer (Ca), however, p53 impairment - through mutation and other mechanisms - fails to effectively halt cell division and induces resistance to cytotoxic drugs and various inhibitors of signaling cascades. Impaired p53 is highly prevalent in advanced malignancies, particularly within metastases, thus offering a promising therapeutic target. Nonetheless, direct pharmacological modulation of mutated or otherwise impaired p53 has historically proven difficult.

Methods & Results:
We previously demonstrated that interfering with p53 binding to the transcription factor Forkhead Box O4 (FOXO4) selectively eliminates p53 impaired apoptosis-resistant cells in fast aging mice (Baar…de Keizer, Cell, 2017). In Ca cells, we discovered and validated an impaired p53 "signature" defined by post-translational phosphorylation of this protein at highly specific residues, namely Ser46, Thr55, and Ser392. These changes cause p53 to localize into progressive multifocal leukemia (PML) nuclear bodies, preventing its translocation to the outer mitochondrial membrane, and, hence, are protecting Ca cells from transcription-independent apoptosis. This p53 impairment signature was universally present in p53-mutated metastatic breast, colon, and ovarian human Ca's, as well as in at least a quarter of such Ca's expressing wild- type (wt) p53, with an increase in prevalence of this phenotype as tumors advance (distant metastases>lymph nodes>>primary site).
To target this mechanism, we developed novel cell-penetrating peptides (CPPs) that selectively bind S46/T55- or S392-phosphorylated p53 in the low nanomolar range. We selected a clinical candidate, CL04183, which dissociates p53 from its PML scaffold with high selectivity, thus driving p53 nuclear exclusion and initiating mitochondrial apoptosis. In preclinical testing of Ca growth models across in vitro cultures, 3D organoids, and assorted in vivo models (cell line-derived xenografts, CDX; patient- derived xenografts, PDX; and spontaneous aged-mouse tumors), CL04183 showed potent antitumor efficacy, including against established distant metastases in vivo. In mice and non-human primates (NHPs), CL04183 demonstrated favorable pharmacokinetics and -dynamics (PK/PD) and a clean safety profile, while significantly reducing liver metastases at doses as low as 10-fold below the maximum tolerated dose (MTD) in mice. Based on these findings, a Phase 1 trial has been designed to evaluate safety and PK/PD parameters of CL04183 administration in patients with advanced,
late-line-refractory p53-mutated triple-negative breast, colorectal, and ovarian Ca's.

Conclusions:
These data define a new overarching paradigm of p53 impairment in Ca metastasis. Peptides like CL04183 release impaired p53 from nuclear structures in a targeted manner and with high therapeutic index to eliminate aggressive cancers via rapid apoptosis induction, thus offering an innovative strategy to overcome p53 "undruggability" in high-unmet-need malignancies. Validating these strategies in clinical settings is imminent and holds great promise.

41. Optimization of Chromatin Immunoprecipitation Coupled with Mass Spectrometry (ChIP-MS) to Identify Epigenetic Regulators of Brachyury in Chordoma

Schuler A[1], He K[1], Lopez A[1], Stevens C[2], Huerta Adrian E[1], Godinez C[1], Gokaslan Z[1], Sullivan P[1], Martinez-Moreno M[1]
[1] Department of Neurosurgery, Warren Alpert Medical School at Brown University, RI, USA
[2] Tufts University, MA, USA

Background:
Chordoma is a rare, slow-growing primary bone malignancy arising from notochordal remnants in the spine, marked by high local recurrence and resistance to conventional therapy. The T- box transcription factor brachyury (TBXT) is constitutively expressed in notochordal and chordoma cells, and is strongly implicated in chordoma pathogenesis. Although brachyury's DNA binding in chordoma has been separately studied, its protein interactions and epigenetic regulatory partners remain largely uncharacterized. We hypothesize that brachyury governs notochordal and chordoma cells through differential recruitment of epigenetic co-regulators, and that identifying these partners may reveal novel therapeutic targets.
Objective:
To establish a robust brachyury ChIP-MS workflow and integrate it with chromatin immunoprecipitation sequencing (ChIP-seq) to define differential epigenetic regulators between notochordal and chordoma cells.
Methods:
A primary chordoma line (JHC7), a recurrent chordoma line (UCH1), and a notochordal control (UCH1N) were cultured at early passage to preserve brachyury expression, which declines beyond passage 20–25. Chromatin was formaldehyde crosslinked, sonicated, and immunoprecipitated with a validated anti-brachyury antibody. The optimized ChIP-MS protocol incorporated Benzonase treatment for chromatin clearance, stringent washes, and elution in 5% sodium dodecyl sulfate plus triethylammonium bicarbonate with heat-mediated crosslink reversal. Protein concentration was quantified by a bicinchoninic acid (BCA) assay. Pulldown efficiency was assessed by quantitative polymerase chain reaction (qPCR) and immunoprecipitation confirmed by Western blot across cell lines and passages. Eluates will undergo liquid chromatography-tandem mass spectrometry (LC-MS/MS) analysis at the Brown University Proteomics Core.
Results:
Western blot confirmed brachyury expression in UCH1N at levels consistent with published data. Successful immunoprecipitation was validated by strong detection of brachyury at the expected
~48–50 kDa in the eluate fraction. Protocol optimization incorporating modified crosslink reversal conditions, elution buffer composition, and Benzonase treatment contributed to an improved ChIP-MS yield. Differential chromatin recovery was observed, with UCH1N and UCH1 exceeding JHC7, an effect attributed to cell number and sonication parameters now being refined. Quality assessments indicate that the UCH1N samples are in suitable condition for mass spectrometry analysis and are ready for submission to the Brown University Proteomics Core, pending completion of JHC7 and UCH1 sample preparation.
Significance:
To our knowledge, this is the first application of brachyury ChIP-MS in chordoma research. Integrating ChIP-MS interaction data with ChIP-seq binding data will yield a comprehensive view of the brachyury epigenetic regulatory network across primary chordoma, recurrent chordoma, and notochordal cells, nominating candidate targets for epigenetic therapy in this treatment-resistant cancer.

42. TProject "Enkh-Munkh": Closing the '120-Day Diagnostic Gap' in Nomadic Mongolia via a Mobile Aero- Medical Infrastructure

Shijirtuya G, Luvsanbayar N, Enkhjin Sh, Munkhjin Sh.
Regional Diagnostic Center, Umnugovi Province, Mongolia; Governor's Office of Dalanzadgad City; Mongolian National University of Medical Sciences; Mongolian University of Science and Technology.

Background:
Mongolia has the world's highest mortality rates for liver and gastric cancer. A critical barrier to survival is the "Geographic Penalty"—nomadic herders in remote provinces face an average 120-day diagnostic delay due to the lack of local advanced infrastructure. This delay results in 75-80% of cases being diagnosed at late stages (III- IV), leading to high mortality and catastrophic "financial toxicity" for families.
Objective:
To establish "Enkh-Munkh", a National Aero-Medical & Diagnostic Center (SOE), designed to deliver Level III specialized oncology care directly to underserved rural populations. This initiative is founded by a multidisciplinary family team—a Health Policy Expert/Surgeon and a Financial Economist (MBA)—to bridge the urban-rural health equity gap with professional governance.
Methods:
The project utilizes a Mobile Aviation Model, converting a C-130/Boeing Cargo aircraft into a fully equipped flying hospital featuring: Onboard Diagnostics: 1.5T Vibration-Dampened MRI, 64-Slice CT, and Digital Mammography for immediate screening. Intervention: A sterile Operating Theater and Angiography Suite for biopsies and TACE (Transarterial Chemoembolization).
Hub & Spoke Logistics:
Ground support via Land Cruiser Ambulances to retrieve patients from remote yurts to the airstrip. Operations are anchored by a sustainable "Township Model" in Khurmen Soum (Gobi Desert), managed under strict clinical and financial standards.
Expected Results:
We project a 20% reduction in cancer-specific mortality within the target provinces over 5 years. By shifting diagnosis from centralized hospitals to a mobile "Point-of-Care" system, we aim to achieve Stage Migration (detecting cancers at Stage I-II vs III-IV) and reduce out-of-pocket patient expenditures by 40%.
Conclusion:
Project Enkh-Munkh proposes a scalable, reproducible model for delivering precision
oncology to sparsely populated, nomadic nations. It demonstrates that geography should not dictate a patient's right to survival.

43. Biomarker-Guided Engineering of Armored CAR T Cells for Cholangiocarcinoma: From Tumor Microenvironment Biology to Clinical Translation

Si K [1,2], Seyhan A [1,2,3,4,5,6], Ryspayeva D [1,2], Clarke M [7], Almhanna K [3,6] , Lannigan L [1,2,4,5], Raufi A [1,2,3,4,5,6]
[1] Department of Pathology and Laboratory Medicine, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[2] Legorreta Cancer Center at Brown University, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[3] The Warren Alpert Medical School, Brown University, Providence, RI, USA
[4] The Joint Program in Cancer Biology, Brown University and Brown University Health, Providence, RI, USA
[5] Laboratory of Translational Oncology and Experimental Cancer Therapeutics, The Warren Alpert Medical School, Brown University, Providence, RI, USA
[6] Hematology-Oncology Division, Department of Medicine, Rhode Island Hospital, Brown University, Providence, RI, USA
[7] Icahn School of Medicine at Mount Sinai, New York City, NY, USA

Background:
Cholangiocarcinoma (CCA) is an aggressive, heterogeneous group of hepatobiliary malignancies arising from the biliary epithelium, with rising global incidence and persistently poor five- year survival across all anatomic subtypes. Only 20–35% of patients are surgical candidates at diagnosis. First-line therapy has moved beyond decades of gemcitabine and cisplatin (GemCis) with the addition of immune checkpoint inhibitors, but gains at the level of tumor response are still incremental. Resistance to immunotherapy in CCA is structural and receptor-mediated as much as antigenic, barriers that antibody-based checkpoint blockade cannot dismantle. Armored chimeric antigen receptor (CAR) T-cells are engineered lymphocytes that couple tumor-specific cytotoxicity with secreted or expressed payloads. They present a feasible mechanism to enter, persist within, and actively remodel precisely the CCA niche.
Gap:
Despite advances in CAR T-cell engineering and encouraging results in selected solid tumors, no armored CAR T-cell construct has entered a CCA-specific clinical trial. Key translational uncertainties include the selection of clinically actionable antigens, the identification of payloads matched to CCA- specific immunosuppressive niches, the ability to overcome stromal exclusion, and the incorporation of effective safety mechanisms
Rationale:
The biological complexity of CCA requires therapeutic strategies that extend beyond antigen recognition alone. Armored CAR T cells may be engineered to enhance persistence, remodel the immunosuppressive TME, counteract inhibitory signaling, and recruit endogenous antitumor immunity. Integrating tumor biology, antigen validation, immune engineering, and spatial profiling may provide a rational foundation for CCA-directed cellular therapies.
Hypothesis: A biomarker-informed, rationally stacked CAR T-cell platform can integrate a validated tumor-associated antigen, a TME-matched armoring payload, conditional activation, and an orthogonal safety switch together to potentially address key biological barriers to effective cellular immunotherapy in CCA while improving translational feasibility and safety.
Goal: To synthesize the current evidence supporting armored CAR T-cell therapy for CCA and establish a translational framework to guide target selection, payload design, preclinical validation, and future early-phase clinical development.
Approach:
We conducted a structured review of peer-reviewed literature via PubMed and of registered studies via ClinicalTrials.gov, accessed May–September 2026, restricted to
English-language publications, selected for mechanistic relevance to CCA-specific barriers. We synthesized 111 sources spanning CCA molecular pathology, antigen-target validation, armoring payload classes, active

clinical trials, and spatial-omics profiling, and organized them into four convergent translational strategies.

Translational Impact:

Prospective integration of spatial profiling and pharmacodynamic monitoring could enable patient selection, guide payload matching, and establish measurable indicators of TME remodeling during early-phase clinical studies.

Limitations of the Current Approaches:

Current evidence is limited by the predominance of preclinical studies and the absence of dedicated clinical trials evaluating armored CAR T cells in CCA. Furthermore, evidence from other solid tumors may not fully translate to the unique biological and anatomical context of CCA.

Perspectives and Future Directions, Including Wet-Lab Validation and Pilot Grants: Near-term priorities include wet-lab validation of candidate antigens and payload combinations in CCA cell lines, patient-derived organoids, three-dimensional co-culture systems, and preclinical in vivo models. Four strategies define this translational path. First, spatial multi-omics for payload matching. Single-cell and spatial atlases of intrahepatic CCA have resolved PD-L1^{+}-TAM exclusion zones, DEFB1^{+} cholangiocyte–IFI30^{+} macrophage signaling pairs, and a POSTN^{+}FAP^{+} CAF/SPP1^{+} macrophage/MAIT-cell triad at the invasive margin. Prospectively embedding this profiling in early-phase trials would enable patient endotyping and provide pharmacodynamic readouts of niche remodeling. Second, conditional activation.

Hypoxia-responsive promoters, NFAT-gated cytokine cassettes, and protease-cleavable masked payloads confine activity to the tumor. Third, targeting the stroma rather than the epithelium.

IL-12-armored CAR T-cells directed at macrophage markers have cleared metastatic tumors in immunocompetent models by mobilizing endogenous CD8^{+} T-cells. Fourth, safety architecture as a design requirement. Program-wide halts of autoimmune CAR T-cell trials in late 2026, following fatal hyperinflammatory events in unarmored constructs under minimal antigen burden, establish that orthogonal safety switches belong in first-in-human CCA constructs rather than retrofitted after a signal emerges.

Conclusion:

Armored CAR T-cell therapy addresses the specific mechanisms that make CCA refractory to checkpoint blockade, yet no armored construct has been tested in this disease.

The bridge is not a single better payload but a rationally stacked design. Prospective integration of spatial profiling into early-phase trial design offers the most immediate opportunity to convert a compelling preclinical literature into a deliberate, safety-conscious clinical program.

44. Novel Biomarkers and Drug Therapeutics: A Multi-Omic Investigation of genes PPP4R2, TBPL1, and SH3D21 in Glioblastoma

Chloe Smith [1] and Attlia A. Seyhan [1,2,3,4,5,6,7]
[1] Brown University, Providence, RI, USA.
[2] Department of Pathology and Laboratory Medicine, The Warren Alpert Medical School, Brown University, Providence, USA
[3] Legorreta Cancer Center at Brown University, The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[4] The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[5] The Joint Program in Cancer Biology, Brown University and Brown University Health, Providence, RI, USA.
[6] Laboratory of Translational Oncology and Experimental Cancer Therapeutics, The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[7] Hematology-Oncology Division, Department of Medicine, Rhode Island Hospital, Brown University, Providence, RI, USA.

Glioblastoma (GBM) is one of the two most rapid and aggressive forms of cancer, originating from astrocytes in the central nervous system. The current standard of treatment, the Stupp Protocol, involves surgical resection, followed by radiation therapy and administration of temozolomide, extending overall patient survival by only two months. Many known biomarkers of GBM fail to offer a reliable way of tracking tumor progression and frequently do not align with new avenues of cancer treatment. This study addresses the need for novel biomarkers through a multi-omics examination of three potential biomarker genes, PPP4R2, TBPL1, and SH3D21. The aim of the study is to identify if each gene, PPP4R2, TBPL1, and SH3D21 offers indicative data on tumor progression, patient prognosis, GBM subtype, novel therapeutic strategies or fulfills more than one category. This study examines 3 combined studies on CBioPortal (495 patients and 491 samples), with inclusion of GBM IDH wild type only, alongside the databases DepMap, STRING, Q-omics, and Gene Cards were utilized to investigated the mutual exclusivity/co-occurrence, mRNA co-expressions, copy number mutations, mutation frequency, overall survival (OS) effects, gene function information, druggable targets, and the associated networks/pathways. We found PPP4R2 to have genetic interactions with PIK3CB, PIK3R1, PIK3R3, RB1, HIF1a, MDM2, and GLIS2 including with a strong Spearman correlation of 0.662 with PIK3CA in mRNA expression. TBPL1 exhibited co- expressional connections to PIK3CB, RB1, and PTEN; physical interactions with TP53 proteins; and genetic relations with PDGFRa; including a Spearman's correlation of 0.613 with ERBB2.

Finally, SH3D12 was observed to have co-expressional relations with GLIS2, physical interactions with EGFR-associated proteins, and a Spearman's correlation of 0.700 with VEGFa in mRNA expression. Commentary surrounding these genes' effects on OS or whether they have aberrant expression patterns remains inconclusive due to insufficient sample sizes. Current findings suggest novel dual drug therapeutic strategies that target pivotal processes such as p53 regulation, cell cycle progression, and growth factor expression with additional emphasis for immediate wet-lab validation studies, preclinical models, and overall, a broader focus on biomarker/therapeutic target identification and development towards translational studies and clinical application.

45. Dissociation Between Bulk Tumor-Tissue Interleukin-15 Expression and Peripheral Absolute Lymphocyte Count in Advanced Solid Tumors

Sood K [1,5], Nishizaki D [2], Kato S [2]*, El-Deiry W [3,5], Kurzrock R [4,5]*
** These authors contributed equally.*
[1] Department of Medical Oncology, Tata Memorial Hospital, Mumbai, Maharashtra, India
[2] University of California San Diego, San Diego, California, USA
[3] Brown University, Providence, Rhode Island, USA
[4] Medical College of Wisconsin, Milwaukee, Wisconsin, USA
[5] Worldwide Innovative Network (WIN) Consortium, USA

Background:
Interleukin-15 (IL-15) supports natural killer and memory CD8+ T-cell survival and may define an immune-active tissue state. We examined whether peripheral absolute lymphocyte count (ALC) reflects tissue IL-15 expression or modifies clinical outcomes with immune checkpoint inhibitor (ICI) therapy.
Methods:
IL-15 RNA expression was assessed from bulk RNA profiling of tumor biopsy tissue in a real- world pan- cancer cohort (n=514) using a 395-transcript panel. High IL-15 was defined as >=75th percentile RNA rank. Correlation between IL-15 RNA rank and ALC measured near tumor specimen procurement was assessed. Among ICI- treated patients, pre-ICI ALC was evaluated as a continuous variable and at thresholds of 1000 and 2000 cells/uL. Survival stratification by IL-15 and ALC was available in 175 ICI-treated patients. Overall survival (OS) from ICI initiation was compared across IL-15/ALC groups and modeled using multivariable Cox regression incorporating IL- 15, ALC, their interaction, immune transcripts, and clinical/molecular covariates.
Results:
Peri-specimen ALC showed no apparent association with bulk tissue IL-15 RNA rank. Combined stratification by IL-15 status and pre-ICI ALC did not separate OS using either the 1000 cells/uL threshold (log-rank p=0.71) or the 2000 cells/uL threshold (p=0.85). In multivariable analysis using the 1000 cells/uL threshold, neither high IL-15 (hazard ratio [HR] 0.74, 95% confidence interval [CI] 0.27-2.03; p=0.56), preserved ALC (HR 0.76, 95% CI 0.44-1.32; p=0.33), nor the IL-15-by-ALC interaction (HR 1.41, 95% CI 0.41-4.88; p=0.586) was associated with OS. Findings were similarly negative with the 2000 cells/uL threshold and when IL-15 and ALC were modeled continuously.
Conclusions:
Peripheral ALC did not mirror bulk tissue IL-15 transcriptional activity and did not modify the association between IL-15 and ICI outcomes. A key limitation is that IL-15 was measured in bulk biopsy RNA, precluding attribution to specific cellular compartments. Because IL-15 may derive from myeloid- lineage, stromal, or epithelial cells, future single-cell and spatial transcriptomic studies should define the cellular source of the IL-15 signal and determine whether compartment-specific expression has distinct relationships with systemic lymphocyte abundance or immunotherapy outcomes.

46. Pan-Cancer Transcriptomic and Clinical Correlates of Interleukin-15 in Advanced Solid Tumors

Sood K [1,2]*, Nishizaki D [3]*, Kato K [3]*, El-Deiry W [1,4], Kurzrock R [1,5]
These authors contributed equally.
[1] Worldwide Innovative Network (WIN) Consortium, USA
[2] Department of Medical Oncology, Tata Memorial Hospital, Mumbai, Maharashtra, India
[3] University of California San Diego, San Diego, California, USA
[4] Brown University, Providence, Rhode Island, USA
[5] Medical College of Wisconsin, Milwaukee, Wisconsin, USA

Background:
Interleukin-15 (IL-15) sustains natural killer cells and memory CD8+ T cells. The IL-15 receptor agonist nogapendekin alfa inbakicept (Anktiva) with bacillus Calmette- Guerin (BCG) is approved for BCG-unresponsive non-muscle-invasive bladder cancer. We explored the association of IL-15 transcripts with other immunoregulatory molecules as well as with outcome in advanced cancer patients.

Methods:
IL-15 RNA expression was analysed in a real-world pan-cancer cohort (n=514) profiled by a 395-transcript panel reporting percentile rank scores; the study was conducted under the IRB- approved PREDICT protocol (NCT02478931) with patients consenting to investigational interventions. High expression was defined as ≥75th percentile RNA rank (735 tumor control group). Overall survival (OS) was measured from advanced disease by Kapan Meier; progression-free survival (PFS) and OS in immune checkpoint inhibitor (ICI) treated patients (N=217) was assessed from ICI start.

Results:
IL-15 transcripts were high in 29.2% of cancers (150/514), varying by histology. High IL-15 was independently associated with high IL2RB (odds ratio [OR] 4.05, 95% confidence interval [CI] 2.44-6.70, p<0.001) but not IL2RG, IL2 or IL2RA. In a multivariate model, high IL-15 was associated with high VISTA checkpoint (V-domain immunoglobulin suppressor of T cell activation; OR 2.60, CI 1.54-4.38, p<0.001), high TNFRSF14 (tumor necrosis factor receptor superfamily member 14 ((HVEM; BTLA ligand)); OR 3.83, CI 2.19- 6.69, p<0.001), high granulysin (GNLY) (cytotoxic protein released primarily by activated natural killer (NK) cells and cytotoxic T cells; IL-15 promotes the activation, proliferation, and survival of these cells). (OR 2.04, CI 1.07-3.89, p=0.031), low BTLA (B and T lymphocyte attenuator; OR 0.46, CI 0.22-0.97, p=0.043), as well as liver (OR 3.16, p=0.039) and ovarian (OR 3.19, p=0.003) cancer primaries. Among 489 patients with advanced disease with clinical correlative data available, high IL-15 correlated with longer median OS (log-rank p=0.022). This correlation did not persist among patients receiving immune checkpoint inhibitors (ICIs) (n=217; OS p=0.53, PFS p=0.88).

Conclusions:
High tumor IL-15 RNA expression marks an inflamed phenotype co- expressing IL2RB, GNLY and alternative checkpoints. This is consistent with trans- presentation biology with GNLY as a proximate effector. High IL-15 correlates with longer OS in our cohort of advanced cancer patients, consistent with the role of IL-15 and IL-15 receptor agonists such as Anktiva as immune enhancers.

47. PRIME-15: A Hypothetic Framework for Interleukin-15 Priming Before Programmed Cell Death Protein 1 Blockade in Immune-Excluded Head and Neck Cancer

Sood K [1,2], El-Deiry [1,3]*, Kurzrock R [1,4]
[1] Worldwide Innovative Network (WIN) Consortium, USA
[2] Department of Medical Oncology, Tata Memorial Hospital, Mumbai, Maharashtra, India
[3] Brown University, Providence, Rhode Island, USA
[4] Medical College of Wisconsin, Milwaukee, Wisconsin, USA

Background:
Programmed cell death protein 1 (PD-1) blockade depends on a viable intratumoral effector-cell pool, and immune-excluded tumors may fail to respond because cytotoxic lymphocytes remain sparse within tumor parenchyma. Nogapendekin alfa inbakicept (NAI), an interleukin-15 receptor agonist, expands natural killer (NK) and memory CD8+ T-cell compartments. We hypothesize that short-course NAI priming before PD-1 blockade can convert immune exclusion into an inflamed phenotype and improve response.

Methods:
PRIME-15 is a multicenter, open-label, randomized phase II signal-seeking trial in 96 adults with untreated recurrent/metastatic head and neck squamous cell carcinoma, programmed death-ligand 1 combined positive score >=1, no prior PD-1/PD-L1 therapy in the recurrent/metastatic setting, and eligibility for pembrolizumab monotherapy. A mandatory baseline biopsy must demonstrate a centrally confirmed immune- excluded phenotype using a prespecified digital CD8 spatial score. Patients will be randomized 1:1 to pembrolizumab alone or NAI lead-in on days -14 and -7 followed by pembrolizumab plus NAI. A safety run-in will precede randomized enrollment. Randomization will be stratified by PD-L1 combined positive score (1-19 vs >=20), human papillomavirus/p16 status, and primary site. A paired biopsy immediately after NAI priming but before the first pembrolizumab dose will isolate the biologic effect of priming; a matched time-point biopsy will be obtained in controls.

Endpoints:
The primary endpoint is objective response rate by Response Evaluation Criteria in Solid Tumors version 1.1, assessed by blinded central review. Secondary endpoints include progression-free survival, overall survival, duration of response, disease control, safety, and quality of life. Prespecified translational endpoints include changes in intratumoral and stromal CD8+ and NK-cell density, immune-excluded-to-inflamed conversion, conventional type 1 dendritic cell-associated CXCL9/CXCL10 signaling, IL-15/IL2RB pathway activity, absolute lymphocyte count, NK-cell expansion, and CD8+ memory subsets.

Statistics and significance:
With 48 patients per arm, the trial has approximately 80% power at one-sided alpha 0.10 to detect an objective response rate increase from 20% to 40%. The primary analysis will use stratified logistic regression; time-to-event endpoints will use Cox models. PRIME-15 directly tests whether restoring effector substrate before checkpoint blockade improves clinical response while linking spatial immune conversion to outcome. A positive signal would support biomarker-directed expansion into other immune-excluded solid tumors.

48. Allosteric phosphoinositide 3-kinase alpha inhibition in PIK3CA-mutant glioblastoma: a biomarker- selected phase 0/2 window-of-opportunity trial

Sur D
Department of Medical Oncology, University of Medicine and Pharmacy "Iuliu Hatieganu", Oncology Institute "Prof.dr.Ion Chiricuta" Cluj-Napoca, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, Romania

Background and rationale:
The phosphoinositide 3-kinase (PI3K)/AKT/mechanistic target of rapamycin (mTOR) pathway is a leading oncogenic driver in glioblastoma, yet no PI3K-directed agent is approved. Activating PIK3CA mutations occur in 6 to 15% of glioblastomas as early clonal events retained at recurrence. Prior trials failed because enrolment used generic pathway activation rather than PIK3CA genotype, and target inhibition was incomplete at doses capped by wild-type PI3K alpha toxicity (hyperglycaemia). Zovegalisib, an oral mutant-selective allosteric PI3K alpha inhibitor, suppresses helical and kinase-domain hotspots while sparing wild-type enzyme, widening the therapeutic window; its central nervous system exposure in glioma is unproven.
Study design:
This academic, multicentre, biomarker-selected trial, enrols isocitrate dehydrogenase- wildtype, PIK3CA-mutant glioblastoma (2021 World Health Organization classification). Part A is a phase 0 surgical window-of-opportunity: patients with recurrent disease receive drug about 7 to 10 days before scheduled re-resection, and resected tumour is assayed for drug concentration and pathway suppression. A predefined pharmacodynamic suppression threshold must be met to open the efficacy phase; this target-engagement gate is the proof of concept. Part B confirms the recommended phase 2 dose in glioblastoma. Part C is a single-arm phase 2 efficacy cohort in recurrent disease, with optional newly diagnosed maintenance expansion. Paxalisib, a brain-penetrant inhibitor, may substitute if the primary agent fails the Part A gate.
Population:
Adults with a confirmed activating PIK3CA mutation (hotspot-preferred; tissue next- generation sequencing or plasma/cerebrospinal fluid circulating tumour DNA) and Karnofsky performance status of at least 60. The primary cohort is biomarker-pure (PIK3CA-mutant, PTEN and AKT co-alteration absent); an exploratory PTEN/AKT co-altered cohort tests the combination question. Stratification is by O6-methylguanine-DNA methyltransferase promoter status and mutation domain (helical versus kinase). Key exclusions are uncontrolled diabetes or baseline hyperglycaemia, enzyme- inducing antiepileptics without washout, and prior PI3K/AKT/mTOR inhibitor exposure.
Statistical considerations:
Part A enrols about 12 evaluable patients for a descriptive pharmacodynamic gating decision. Part C uses a Simon two-stage design against a historical recurrent-glioblastoma 6-month progression-free survival of 15% ($p0 = 0.15$, $p1 = 0.35$, $alpha = 0.05$, $power = 0.80$): about 19 patients in stage 1 and 39 in total. The biomarker-pure cohort is the primary efficacy population.
Correlative programme:
Paired pre- and post-treatment tissue supports spatial transcriptomics of PI3K and compensatory signalling, immune profiling, longitudinal circulating tumour DNA tracking for resistance, and an image-based classifier for sequencing-independent selection.
Ethics and feasibility:
The investigational agent is sponsor-supplied, so reimbursement is not rate- limiting. National regulatory and multicentre ethics approval are required, under Good Clinical Practice with written informed consent. Because PIK3CA-mutant, PTEN-intact glioblastoma is uncommon, accrual depends on a centralised molecular pre-screening network across European sites. The Part A gate de-risks the programme before any efficacy commitment.

49. Breast Cancer in Very Young, Young, and Older Women: A Comparative Bioinformatic Study of Protein- Protein Interactions Identifying Distinct Age-Related Proteomic Networks

Verovkina N [1,2,3], Chang E [4], Grether V [5], Ryspaeva D [1,2,3], Seyhan A [1,2,3,4,9], El-Deiry W [1,2,3,4,9,10], Uzun A [2,3,6,7,8]

[1] Laboratory of Translational Oncology and Experimental Cancer Therapeutics, The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[2] Department of Pathology and Laboratory Medicine, The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[3] Legorreta Cancer Center at Brown University, The Warren Alpert Medical School, Brown University, Providence, RI, USA.
[4] Warren Alpert Medical School, Brown University, Providence, RI, USA
[5] Department of Biomedical Engineering, Brown University, RI, USA
[6] Center for Computational Molecular Biology, Brown University, Providence, RI, USA
[7] Center for Clinical Cancer Informatics and Data Science (CCIDS), Brown, Providence, RI, USA
[8] Department of Pediatrics, Warren Alpert Medical School, Brown University, Providence, RI, USA
[9] The Joint Program in Cancer Biology, Brown University and Brown University Health, Providence, RI, USA.
[10] Hematology-Oncology Division, Department of Medicine, Rhode Island Hospital, Brown University, Providence, RI, USA.

Background:
Breast cancer (BC) remains the most frequently diagnosed female cancer and predominantly occurs in middle-aged and older women. Recent trends in BC incidence rates over the past ten years show an increasing burden among younger age groups across regions worldwide.
Younger women have worse outcomes than their older counterparts due to reasons that remain mostly unexplained. In this bioinformatic study, we used a systems biology approach to explore age- stratified transcriptional and proteomic profiles to better understand the age-related biology of BC.
Purpose: This study aims to explore how genetic alterations disrupt the function of key protein- protein interaction (PPI) networks across three age cohorts of breast cancer patients (<35 vs 36-44 vs≥45) and relate our results to clinicopathologic data

Methods:
We analyzed RNA sequencing data from publicly available gene expression datasets for primary BC, including the combined METABRIC and MSK dataset (n = 2182) and the TCGA Firehose Legacy Dataset (n = 1096). Here, we analyzed the data using a multi-sample protein-protein interaction (PPI) analysis tool, Proteinarium, across the three groups: Group A, very young patients (≤ 35 years); Group B, young patients (36-44 years); and Group C, older patients (≥ 45 years) at BC diagnosis, which were also intrinsic subtype-stratified.

Results:
Here, we run comparative analysis of PPI Networks and showed a distinct separation between networks in the three groups: Group 1 (A) Very Young patients (≤35 years of age), Group 2 (B) Young patients (36-44 years of age), and Group 3 (C) older patients (≥45 years of age) at breast cancer diagnosis. Proteomic analysis of chosen clusters shows a significant degree of heterogeneity within hormone receptor-positive breast cancer in very young (☐ 35y.o.) and young (36-44 y.o.) women compared to older patients with luminal subtypes of breast cancer. Our analysis did not reveal this pattern in more aggressive triple-negative and HER2neu-positive breast cancer subtypes.

Conclusions:
Our primary finding shows that even though most tumors in young and very young patients belong to aggressive subtypes of breast cancer, the highest degree of variability was observed in young and very young patients with luminal types of breast cancer. Differences across PPI networks by age group may reflect unique protein interactions and dynamics driving disease progression in each cohort and partly

explain the more aggressive behavior and worse prognosis observed in younger BC patients compared with older counterparts in prognostically favorable luminal BC.

50. Peroxisomal Fatty Acid Oxidation and Lipid Metabolic Remodeling Drive ABCB1-Associated Paclitaxel Resistance in High-Grade Serous Ovarian Carcinoma

White B, Park P, Lai Y, Doroshenko S, Ghosh D, Dawson M
Brown University, Providence, Rhode Island, USA

Resistance to paclitaxel (PTX), a frontline chemotherapeutic for high-grade serous ovarian carcinoma (HGSOC), frequently arises through upregulation of ATP-binding cassette (ABC) transporters that promote drug efflux. However, disseminated HGSOC cells preferentially metastasize to the lipid-rich omentum, where abundant extracellular lipids may provide metabolic substrates that support tumor cell survival and drug resistance. Lipid droplets (LDs), intracellular organelles that store neutral lipids, are increasingly associated with aggressive disease and chemoresistance, yet the metabolic adaptations that enable PTX-resistant cells to survive within this lipid-rich microenvironment remain poorly understood. We hypothesized that lipid metabolic reprogramming enables HGSOC PTX-resistant (TR) cells to meet the energetic demands of chemoresistance and cooperates with ABCB1-mediated drug efflux to promote survival. We investigated ABCB1, lipid metabolism, fatty acid oxidation (FAO), and LD dynamics in PTX-sensitive (TS) and TR HGSOC cells under basal, lipid-supplemented, and nutrient-restricted conditions. TR cells exhibited increased LD accumulation and elevated expression of FAO-associated genes, including carnitine palmitoyltransferase 1A (CPT1A) and carnitine O- octanoyltransferase (CROT). Inhibition of ABCB1 with verapamil increased LD accumulation without reducing TR cell viability, whereas combined verapamil/PTX treatment significantly resensitized TR cells to PTX. Genetic depletion of CROT altered respiratory metabolism, increasing basal and maximal respiration under normal culture conditions but reducing spare respiratory capacity during glucose restriction, consistent with impaired metabolic flexibility. CROT depletion also reduced LD number while increasing LD area, linking peroxisomal FAO to lipid homeostasis. Importantly, genetic and pharmacologic inhibition of CROT sensitized TR cells to PTX, supporting a functional role for CROT-dependent lipid metabolism in chemoresistance. Analysis of HGSOC tissue further demonstrated spatially distinct expression of resistance and metabolic markers. While ABCB1 was enriched primarily within tumor-associated stromal/macrophage regions, CPT1A expression was predominantly localized to malignant tumor cells, and CROT was broadly distributed across both stromal and tumor compartments. Notably, CPT1A and CROT expression were consistently elevated in tumor-associated and metastatic tissues, supporting the clinical relevance of FAO-associated metabolic remodeling during HGSOC progression. Together, these findings identify CROT-dependent peroxisomal lipid metabolism as a previously underexplored mechanism supporting metabolic adaptation and PTX resistance in HGSOC. Our results suggest that chemoresistance is sustained not only through drug efflux, but also through metabolic flexibility within the lipid-rich tumor microenvironment, highlighting CROT as a potential therapeutic vulnerability for overcoming PTX resistance.

51.Translating NCI-8 into Precision Oncology: Biomarker-Guided p53-Pathway Restoration Through p73 in TP53-Altered Solid Tumors

Youssef E [1], Xiaobing Tian X [2], El-Deiry W [1,2]
[1] p53-Therapeutics, Inc., Providence, RI, USA
[2] Legorreta Cancer Center at Brown University, Warren Alpert Medical School, Providence, RI, USA

Background:
TP53 is the most frequently mutated gene in human cancer, and p53-pathway output is additionally lost in tumors with wild-type TP53, most notably through HPV E6-mediated p53 degradation in HPV-associated malignancies. Allele-selective p53 reactivators, while clinically validating the target, address narrow molecular subsets; Y220C accounts for approximately 1.8% of TP53 mutations. NCI-8 (NSC59984) was developed to address this breadth gap by restoring p53- pathway signaling through p73, a p53-family transcription factor that is rarely mutated in human tumors and may remain available as an effector across TP53 alteration classes.
Previously published preclinical work has defined the mechanism and provides a translatable pharmacodynamic framework. NSC59984 induces ROS-dependent sustained ERK2 signaling and MDM2 Ser166 phosphorylation, leading to ubiquitin-proteasome-mediated degradation of gain- of-function mutant p53 and restoration of p53-pathway output through p73-associated activation of the CDKN1A/p21 and PMAIP1/NOXA promoters. Reported activity is p73-dependent in cancer- cell death assays and mutant-p53 xenograft models, with minimal genotoxicity and no evident toxicity in tested normal-cell models at active doses. Preclinical activity is enhanced under high- ROS conditions, nominating baseline tumor redox state as a candidate response biomarker. NSC59984 also synergizes with irinotecan (CPT-11) in mutant-p53 colorectal cancer models, establishing a combination rationale. Together, these data define a mechanism-linked biomarker set—mutant-p53 depletion and p73-associated p21/NOXA induction—that can be measured directly in patient tumors and support first-in-human evaluation.
Trial design:
This proposed Phase 1 study will enroll patients with advanced solid tumors harboring documented TP53 alterations, with a prospectively defined HPV-associated, TP53-wild- type HNSCC cohort permitted in expansion based on evidence of E6-mediated p53 suppression. Prospective stratification will include tumor lineage, TP53 alteration class, p53 immunohistochemistry, and baseline molecular context. Dose escalation will characterize safety and pharmacokinetics; serial ctDNA and paired pre-/on-treatment biopsies will interrogate target engagement and pathway restoration, with mutant-p53 depletion and p73-associated p21/NOXA induction as prespecified mechanism-linked pharmacodynamic endpoints. The objective is to identify a biologically active dose defined by mechanism rather than toxicity alone. Colorectal cancer will serve as the proof-of-mechanism entry cohort, anchored by the published mutant-p53 colorectal dataset and the irinotecan combination rationale. HNSCC will constitute a hypothesis- driven, evidence-gated expansion encompassing HPV-negative/TP53-mutant disease and selected HPV-positive/TP53-wild-type tumors, testing whether E6-mediated p53 suppression permits p73- directed pathway restoration.
Conclusions:
NCI-8 translates p53-family biology into a staged, biomarker-guided development plan advancing sequentially through safety, exposure, mechanism-linked tumor pharmacodynamics, biologically active dose, and evidence-gated expansion. By acting through p73 rather than a single mutant conformation, NCI-8 is designed to address genetically and functionally p53-suppressed tumors beyond the scope of allele-specific reactivators and to support biomarker-defined clinical development.

52. The genomic landscape of H3K27M and H3G34R-mutated gliomas suggests divergent Precision Oncology treatment strategies

Zayan K.M. Islam and Wafik S. El-Deiry
Brown University, Rhode Island, USA

Histone H3.3-G34R and K27M mutations define distinct subtypes of pediatric diffuse intrinsic pontine glioma (DIPG) and young adult diffuse high-grade glioma, yet the mutation-specific comparative genetic and epigenetic landscapes remain incompletely characterized. We are interested in comparing these epigenetically mutated tumors due to the previous discovery of ONC201/TIC10 in our lab ultimately leading to its approval as dordaviprone by the FDA in 2025 for recurrent H3K27M-mutated diffuse gliomas. Using publicly available genomic data from cBioPortal and PedcBioPortal, we analyzed mutation frequencies, copy number alterations, and structural variant data across H3G34R (N=102) and H3K27M (N=702) glioma samples.

H3G34R tumors were characterized by a near-universal TP53 and ATRX co-mutation (95.2% and 79.8%, respectively), recurrent PDGFRA alteration within the 4q12 amplicon, and pathway enrichment converging on cell cycle checkpoint and epigenetic gene regulation. By contrast, H3K27M-mtated tumors displayed a more diffuse and common co-mutational profile characterized by PI3K/AKT pathway activation (PTEN, 5.7%; PIK3CA, 14.7%; PIK3R1, 7.8%; NF1, 12.4%), ATRX co-mutation (20.5%) and the less common co-mutations involving oligodendrocyte lineage regulators (SOX10, 5.1%; OLIG2, 1.3%) associated with a proposed oligodendrocyte precursor cell-of-origin model for this tumor. Co-mutation of either H3K37M or H3G34R with ATRX is associated with PARP inhibitor sensitivity. MGMT promoter methylation analysis from subsets with available data found that MGMT hypermethylation was markedly more prevalent in H3G34R (65.2%, 15/23) than H3K27M tumors (5.6%, 4/72), despite the known genome-wide hypomethylation characteristic of H3G34R. Lack of MGMT methylation in most H3K27M- mutated gliomas is associated with resistance to temozolomide. Our laboratory has reported a preclinical effect of the imipridone drug class that includes ONC201 and ONC206 towards reduction of MGMT, EZH1 and EZH2 protein expression in H3K27M-mutated tumor cells. Subgroup analysis stratified by MGMT status revealed a notable divergence in PDGFRA mutation frequency between MGMT- methylated and unmethylated H3G34R tumors, suggesting a possible relationship between PDGFRA- driven signaling and epigenetic regulation that warrants further investigation. H3G34R-mutated tumors have an associated CDK6- dependency with reported benefit from CDK4/6 inhibition, and immune responsive signatures. Together, these findings indicate distinct co-mutational and pathway-level architectures underlying H3G34R and H3K27M glioma that predict divergent Precision Oncology therapeutic strategies that may exploit unique vulnerabilities.

53. WikiMed Article Recommender: An Open-Source Dashboard for Data-Driven Prioritization of Cancer-Related Wikipedia Article Editing

Schneider-Lynch, Z, Warner J

Warren Alpert Medical School Of Brown University, Rhode Island, USA

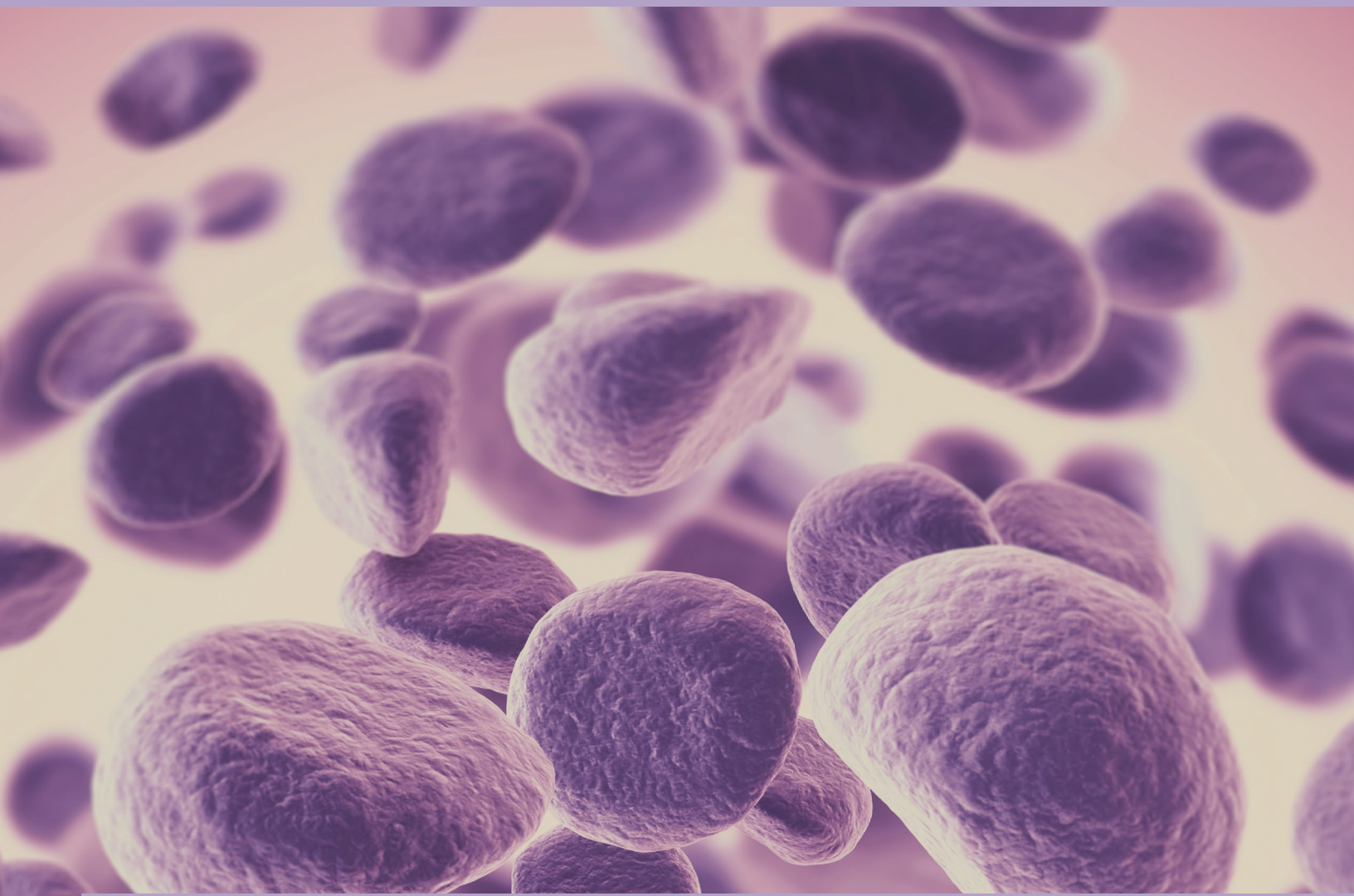
Background: Wikipedia is the most accessed online health information resource and depends entirely upon volunteer editors. For patients and clinicians outside academic cancer centres it is frequently one of the first freely available explanations of a biomarker report, a targeted agent, or an inherited cancer risk. General cancer content on Wikipedia has been characterized, but the precision oncology (PO) corpus specifically has never been separately characterised, even as biomarker-driven care enters routine practice. We developed a customized algorithm and dashboard that addresses this unmet need and provides a tool to help volunteer student editors select articles to edit.

Methods: An open-source web application was developed featuring dedicated Cancer and Precision Oncology dashboards. Medical Subject Headings (MeSH) descriptors were assigned to WikiProject Medicine-flagged articles through a custom seven-layer pipeline, and cancer-specific articles identified by filtering assigned descriptors against MeSH subtrees. PO articles were isolated using 128 descriptors spanning tumour biomarkers, molecular diagnostics, targeted and immunological antineoplastic agents, biomarker-directed endocrine therapy, and hereditary cancer predisposition, with drug capture requiring both an antineoplastic action and a molecularly targeted mechanism. Each article was scored using a normalized Impact-Need Score (0-100) combining pageview reach, editorial importance labels, quality deficit, editor scarcity, and clickstream-derived search intent. Readability was estimated by Flesch-Kincaid (FK) grade level, and PO articles compared against the remaining cancer corpus (Mann-Whitney U).

Results: Among >53,000 WikiProject Medicine-tracked articles, 1,859 cancer-specific articles were identified, of which 129 were PO-specific. PO articles drew a median 9,559 annual pageviews versus 2,432 for other cancer articles ($p<0.001$; 2.96 million views in total), yet were written at a significantly higher median FK grade level of 14.4 (IQR 12.3–16.1) versus 13.6 ($p=0.020$). Only 1 of 128 PO articles (0.8%) was readable at or below the eighth-grade level of the average US adult, while 78.9% exceeded twelfth-grade level. Median Impact-Need Score was 66.1 (IQR 59.4–74.0) versus 63.5 ($p=0.008$); 49.6% were rated Stub or Start quality, with one Good Article and none Featured. Rare cancers showed the widest gap, drawing a median 15,497 annual views with 90.9% written above twelfth-grade level. The cancer-wide median of 13.6 closely matches the grade 14.1 reported for Wikipedia cancer content in 2011, indicating no readability improvement over fifteen years.

Conclusions: Wikipedia is an ideal forum for widespread and unfettered information sharing. By repurposing open data standards such as MeSH, our tool provides a scalable, transparent pathway for directing medical volunteer editors toward the highest-impact gaps in publicly accessible precision oncology information. The molecular concepts that increasingly determine treatment selection are among the most read and least readable cancer content on Wikipedia — a barrier that falls hardest on those already furthest from specialist care, and one that propagates into the large language models now trained on this corpus. The dashboard is available for public use at wikimed-dashboard.com, licensed CC BY-NC-SA 4.0, with all code, MeSH mapping files, and pipelines open-source on GitHub.

ACKNOWLEDGEMENTS



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Medical Oncologist,
Avera Cancer Institute
(USA)

Dr. Carol Farhangfar
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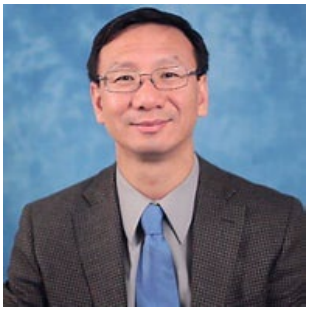

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Department of Pathology and Laboratory Medicine
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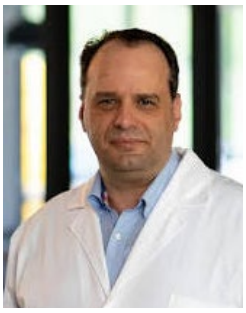

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- Prepare scientific program

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- Plan new publications in Precision Oncology as well as trials WIN is involved with
- Plans for meeting abstracts

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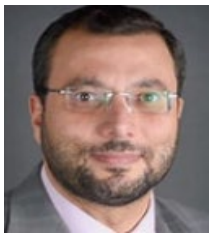

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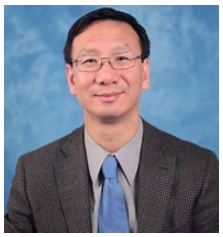
Dr. Mandar Kulkarni (VCR Park, India) - Co-Chair



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WIN International Molecular Tumor Board

The objective of the Molecular Tumor Board (MTB) is to allow the physicians who participate to:

- Discuss cancer cases they encounter in their professional practice, in particularly on rare cases,
- Identify treatment options that the physician who presented the case to the MTB could consider but is not required to follow,
- Educate and improve the knowledge of physicians on the diagnosis and treatment of different cancer cases,
- Provide their expertise and hence input into the scientific knowledge regarding possible therapy and related matters.

#	Institutions	#	Institutions
1	Avera Cancer Institute, Sioux Falls, USA	12	University of Medicine "Iuliu Haţieganu" Cluj-Napoca, ROMANIA
2	Legorreta Cancer Center, Brown Health, Providence, USA	13	Acibadem Adana Hospital, Adana, TURKEY
3	Chaim Medical Resource, New York, USA	14	Medicana Health Group Demiray Precision Oncology Center, Istanbul, TURKEY
4	The Jackson Laboratory, Maine Cancer Genomics Initiative, Farmington, USA	15	Sheba Medical Center, Tel Aviv, ISRAEL
5	Rutgers Cancer Institute, New Brunswick, USA	16	King Hussein Cancer Center, Amman, JORDAN
6	Segal Cancer Centre, McGill University, Montreal, CANADA	17	Burjeel Medical City Hospital, Abou Dabi, UAE
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9	Centre Hospitalier de Luxembourg, LUXEMBOURG	20	Fudan University Shanghai Cancer Center, Shanghai, CHINA
10	Catalan Institute of Oncology, Barcelona, SPAIN	21	Institute of Science Tokyo Hospital, Tokyo, JAPAN
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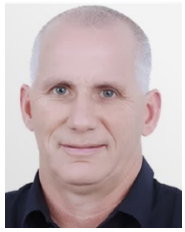

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WIN MTB Breast Cancer Co-Moderator: Dr. Stephanie Graff, Legorreta Cancer Center, USA
WIN MTB Clinical Trials Co-Moderator: Dr. Alberto Hernando Calvo, Vall d' Hebron Institute of Oncology, Spain
WIN MTB Early-Phase Trials Co-Moderator: Dr. Benedito Carneiro, Legorreta Cancer Center, Lifespan, USA
WIN MTB Endometrial Cancer: Dr. Eugenia Girda, Rutgers Cancer Institute, USA
WIN MTB Lung Cancer Co-Moderator: Dr. Christopher Azzoli, Legorreta Cancer Center, Lifespan, USA
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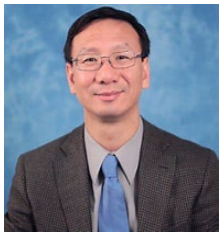
WIN MTB Molecular Pathology Co-Moderator: Dr. Liang Cheng, Legorreta Cancer Center, USA
WIN MTB Radiation Oncology Co-Moderator: Dr. Ibrahim Abu-Gheida, Burjeel Medical City, UAE



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WIN CONSORTIUM – WIN Precision Oncology Fellowship Program

WIN Precision Oncology Fellowship Program

The aim of the WIN Precision Oncology Fellowship Program is to bring together faculty fellows from around the world with WIN Consortium leaders in oncology and WIN member institutions, in order to train, mentor, and develop the next generation of global leaders in precision cancer medicine.

The objective is to support the career development of faculty fellows in Precision Oncology through structured research, mentorship, and leadership opportunities within the WIN Consortium.

Fellows will engage in the following activities during the fellowship:

- Develop and initiate one study protocol within the first four months of the fellowship.
- Conduct outreach to industry collaborators and participate in grant application processes.
- Prepare and submit at least one manuscript for publication.
- Present a WIN abstract at national and/or international meetings.
- Contribute to social media activities, interviews, and news updates to promote Precision Oncology initiatives.
- Participate regularly in the WIN International Molecular Tumor Board.
- Take an active role in WIN committees to support consortium-wide projects and governance.

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Dr. Karan Sood, MD
Assistant Professor, Medical Oncology, Advanced Centre for Treatment, Research and Education in Cancer ACTREC- Tata Memorial Hospital (India)

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