

# Department of Systems Biology

COLUMBIA UNIVERSITY IRVING MEDICAL CENTER

## Newsletter 2024–2025

- Single-cell breakthroughs, AI-driven discovery
- From microbiomes to brain tumors
- How data, biology, and engineering converge
- Next-generation tools for decoding disease
- Systems thinking for complex biology
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# New Technique for Single-Cell Sequencing Enhances Drug Screening Research

A new study by scientists at the Herbert Irving Comprehensive Cancer Center (HICCC) introduces a more cost-effective, scalable single-cell sequencing technique for co-sequencing RNA and DNA. The tool, called DEFND-seq (DNA and Expression Following Nucleosome Depletion sequencing), uses an existing microfluidics platform that can process cells in parallel, allowing cancer researchers to better explore questions related to disease progression, tumor evolution, and drug resistance.

Analyzing both RNA and DNA from several thousands of single cells within a tumor sample could help researchers understand how the genome and transcriptome interact to drive cancer growth. Current methods for simultaneous sequencing are limited by throughput, only processing a few thousand cells at once. They rely on manually manipulating individual cells in multi-well plates, flat plates embedded with many small test tubes.

“The use of multi-well plates requires more material, in terms of enzymes and other expensive ingredients, and puts a cap on the number of cells you can practically analyze from a single sample,” says Peter Sims, PhD, associate professor of systems biology at Vagelos College of Physicians & Surgeons (VP&S) and co-leader of the Precision Oncology and Systems Biology program at the HICCC. “With this new technique, we’re using a droplet microfluidic system that makes it really easy to process tens of thousands of cells in parallel.”

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**“We’ve got some really exciting work going on in our cancer center, including efforts to develop local delivery of drugs across the blood-brain barrier that we think are going to be really impactful”**

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## An Accidental Discovery Sparks a New Technique

DEFND-seq leverages an existing commercial droplet microfluidics system from 10x Genomics, a platform that is widely and routinely used in single-cell studies around the world. Reactions take place in tiny droplets, rather than wells, minimizing the amount of sample material and reagents needed for each reaction.

DEFND-seq originally came about from a mistake in the lab. As scientific director of the Sulzberger Columbia Genome Center, Sims observed many experiments where an existing test, called ATAC-seq, would fail. Normally, the



proteins act like spools of thread that wind up regions of DNA so they can’t be accessed, and ATAC-seq analyzes the regions that are left open. Sims realized that disrupting the chromatin packaging would enable sequencing to occur on the entire DNA strand.

“Some accidents in the lab led us to stumble into this possibility of basically doing ATAC-seq wrong on purpose,” he says. “Essentially, we’re messing up an ATAC-seq protocol so that all the protein falls off of the DNA, and that allows us to use very similar chemistry to profile the genome more uniformly.”

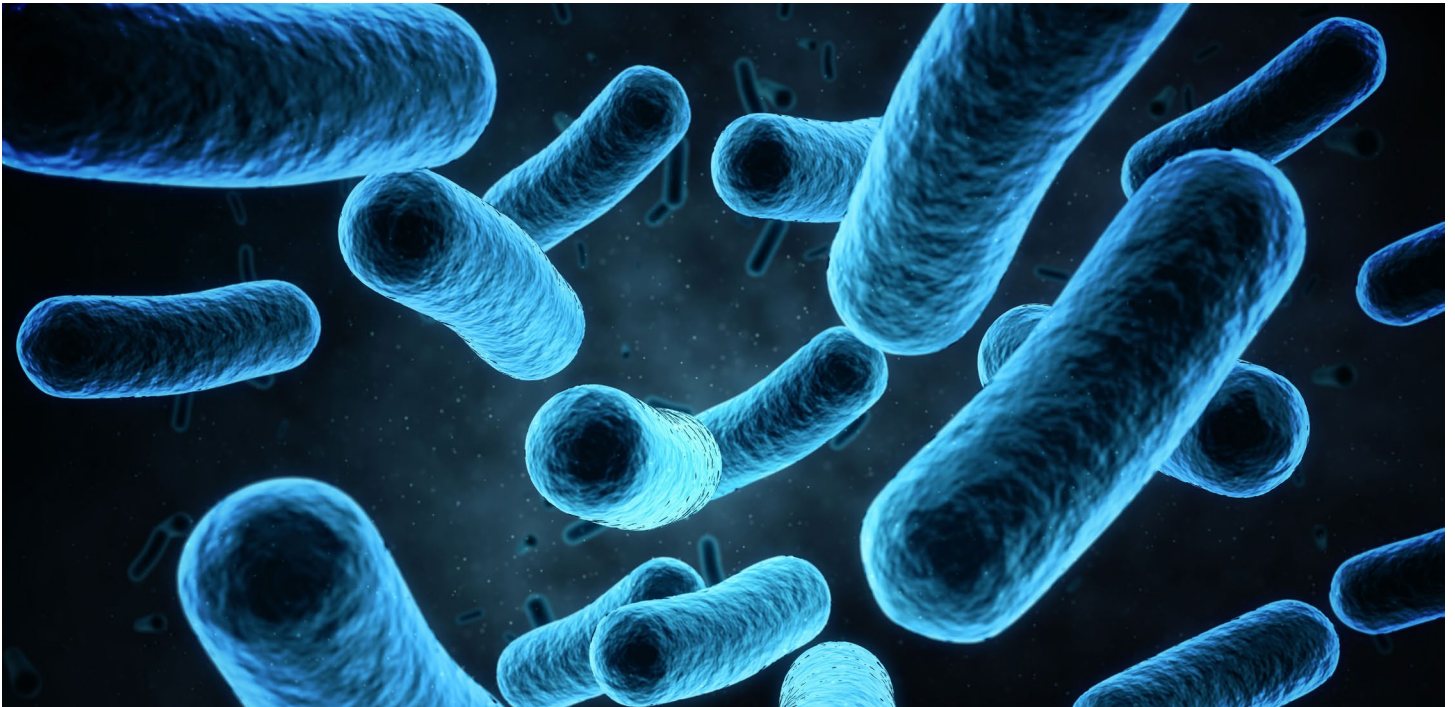
## Enhancing Drug Screening in Brain Tumors

Tim Olsen, a postdoctoral researcher in the lab, performed a series of benchmarking experiments where they applied DEFND-seq to various glioblastoma samples, including archived surgical specimens. Glioblastoma is an aggressive primary brain tumor with a median survival of 15 months. Sims, whose lab focuses on glioblastoma and other neurological disorders, plans to use DEFND-seq to help analyze cell type-specific drug responses.

“We can take a surgical resection from a human tumor and perform a drug screen directly on the intact tumor tissue, including with drug combinations,” says Sims. “Historically, we’ve used single-cell RNA sequencing as our primary readout for these screens and that allows us to identify the cell types that are impacted by each drug, including non-tumor cell types.”

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# Supercharging Bacteria to Fight Food Poisoning



When food poisoning hits, the idea of consuming anything, much less a concoction of bacteria, probably turns your stomach.

But for the microbiologists and genetic engineers in Harris Wang's Columbia University laboratory, a therapeutic bacterial brew could be the only way to disarm some of the most dangerous intestinal pathogens.

The idea to use good bacteria to thwart bad bacteria in the gut microbiome isn't a new one. But probiotics often fail to colonize the gut. "That's one of the challenges with live bacterial therapy. Because they have such a transient nature, their effects are also transient," says Wang, who is interim chair of the Department of Systems Biology at Columbia University's Vagelos College of Physicians and Surgeons.

To make a more long-lasting and effective treatment, Wang has taken a cue from the way bacteria naturally swap genetic material with members of their own species and even unrelated ones.

## Horizontal gene swaps

This process, known as horizontal gene transfer, occurs frequently between microbes in the gut, often with negative consequences for human health. "This is how genes that confer multi-drug resistance and virulence spread," Wang says. "But it's also a driver for genetic innovation and allows for microbes to evolve much more quickly."

Wang's idea was to engineer bacteria to transfer genes into harmful bacteria that render them harmless. The transferred cargo includes a genetic editor that locates and dis-

ables toxic genes, essentially disarming harmful bacteria by editing their genomes.

Because the targeted bacteria don't die, the therapy could sidestep a major issue with today's antibiotics, which are designed to kill pathogens rather than disable them. "With antibiotics, pathogen elimination is never 100% effective, and variants inevitably emerge that are even less treatable,"

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**"We see this work establishing a versatile platform for targeting a broad range of pathogens and advancing the delivery and expression of novel therapeutic payloads."**

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Wang says. "Leveraging natural gene transfer could be a more effective approach without generating resistance."

High on Wang's list of harmful bacteria to target are those that produce the Shiga toxin, which spread through contaminated water, food, and surfaces, causing severe food poisoning in nearly 1 million people each year around the world.

Shiga-producing bacteria represent a unique challenge for medicine. Antibiotics can kill the bacteria but also cause a massive outpouring of toxins from the dying cells. "Giving antibiotics to these patients only worsens their situation," Wang says. "There's really nothing other than keeping people hydrated and providing supportive care."

Because of the heightened risk, Wang's team took extra care

to design therapeutic bacteria that didn't accidentally kill the pathogens while editing them. The CRISPR gene editor was deemed too risky, because DNA cleavage caused by the editor can result in bacterial death and toxin release. Instead, the Wang team deployed a gene editor recently developed by Columbia's Samuel Sternberg that works without cleaving DNA.

The therapeutic bacteria—dubbed BACTRINS—also transfer a gene encoding a synthetic antibody designed to interfere with the pathogen's attachment to the gut.

### Defanging harmful gut bugs

In tests with mice, the results were striking.

As the team reports in *Nature Biomedical Engineering*, BACTRINS transferred its cargo to Shiga-producing bacteria with 100% efficiency, sharply reduced levels of the toxin in the mice, and increased survival. The treatment also acted like a vaccine, fully protecting mice from subsequent infections.

“Programmable microbiome editing gives us surgical precision, defanging pathogens like Shiga-toxin bacteria and turning harmful gut bugs into protective allies,” says Carlotta Ronda, one of the study's authors, now a principal investigator at the University of California, Berkeley.

In addition to disabling “bad” bacteria, BACTRINS could also be used to deliver new capabilities to the microbiome's “good” bacteria, including the production of drugs that could boost the immune system or control metabolic diseases like diabetes.

“We see this work establishing a versatile platform for targeting a broad range of pathogens and advancing the delivery and expression of novel therapeutic payloads,” says study author Tyler Perdue, who conducted much of the research as a graduate student in the Wang lab. The system needs some fine-tuning before it can be deployed in people, but it “opens the door to next generation living therapeutics,” Ronda says.

Source/Credit: The Herbert Irving Comprehensive Cancer Center. [Link: https://www.cuimc.columbia.edu/news/supercharging-bacteria-fight-food-poisoning](https://www.cuimc.columbia.edu/news/supercharging-bacteria-fight-food-poisoning)

*Continued from page 3*

Scalable co-sequencing of RNA and DNA from individual nuclei was published Feb. 12, 2025, in *Nature*.

With the addition of high-resolution DNA data provided by DEFND-seq, Sims and his colleagues can easily map the drug responses for subclones, defined as cells with distinct genetic lineages, within heterogeneous tumors. In particular, the technology can identify specific subclones that drive mechanisms of drug resistance.

“We've got some really exciting work going on in our cancer center, including efforts to develop local delivery of drugs across the blood-brain barrier that we think are going to be really impactful,” he says. “We're trying to make this drug screen platform as sophisticated as possible so that we can look on a cell state-by-cell state basis at what drugs we can combine together to maximally ablate the tumor.”

Source/Credit: The Herbert Irving Comprehensive Cancer Center. [LINK: https://www.cancer.columbia.edu/news/new-technique-single-cell-sequencing-enhances-drug-screening-research](https://www.cancer.columbia.edu/news/new-technique-single-cell-sequencing-enhances-drug-screening-research)

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## DSB Welcomes Dig Bijay Mahat, PhD

Dig Bijay Mahat earned his BS in Biochemistry, Molecular Biology, and Bioinformatics from Towson University, completed his PhD in Molecular Biology and Genetics at Cornell University with John Lis, and conducted postdoctoral training in Phillip Sharp's laboratory at the Koch Institute for Integrative Cancer Research at MIT.

His lab investigates how non-coding DNA controls gene expression in health and disease. Focusing on enhancers that shape the tumor immune microenvironment in gastrointestinal cancers and those harboring genetic risk variants for autoimmune disorders, his group develops and applies single-cell transcriptomic assays, integrating them with AI and machine-learning frameworks to map gene-regulatory programs at high temporal resolution. Their goal is to identify and target disease-driving enhancers to advance new therapeutic strategies for immune dysfunction diseases.



# Bianca Dumitrascu Uses Statistics and AI to Understand the Mysterious Lives of Cells

Growing up in Romania, Bianca Dumitrascu liked humanities—literature, history, philosophy. But, like other children in her country, she also studied math. “I grew up with math competitions and Olympiads, there’s a big history of that in Romania,” she says. “It’s a rite of passage for every kid.” At the time, biology was just another school subject where one primarily memorized facts.

When Dumitrascu started college at MIT, the curriculum required taking a variety of courses—introductory chemistry, physics, linguistics, anthropology, economics and biology—aimed to expose students to different ideas. She took a biology class with Eric Lander, a big proponent for using technologies to interpret and learn from the data collected from genome sequencing and other sources. “He suggested that mathematics, computer science, and statistics could be employed to help us analyze this ever-increasing amount of data,” she says. “Before that I never considered getting into biology, but after this class, I was mesmerized by the field.” As an undergraduate student, she joined the labs of Bonnie Berger and Aviv Regev, and later studied biology and machine learning under the guidance of Barbara Engelhardt eventually earning her PhD in computational biology at Princeton University.

Since then, Dumitrascu had been a member in the School of Mathematics at the Institute for Advanced Study, attended a program in deep learning at the Statistical and Applied Mathematical Sciences Institute and was a Departmental Early Career Fellow in the Department of Computer Science and Technology at the University of Cambridge. In 2023, she joined Columbia University to lead the Computational Morphogenesis Lab. “Part of what we do in my lab is we design interpretable machine learning tools to understand self-organizing systems, which includes wound healing, development and regeneration,” Dumitrascu says. “And we also use biology as an inspiration for thinking about new mathematical models. So the hope is to try to understand biology, but sometimes we also use biology as an excuse to come up with new mathematical formalisms.”

In her work, Dumitrascu studies how different species heal themselves from injuries, what cells are involved and how they interact with each other. “I’m very interested in how cells know where to go, how they navigate the environments in which they reside, and how they use information from their neighbors to make better decisions as they build organs, heal wounds or regenerate full organisms,” she says.

To understand how cells self-organize to rebuild tissues, Dumitrascu partners closely with biological collaborators across Columbia’s medical campus. In the context of regeneration, she works with Dr. Joanna Smeeton—an As-



sistant Professor in the Departments of Rehabilitation & Regenerative Medicine and Genetics & Development and a member of the Columbia Stem Cell Initiative. In the context of wound healing and tissue repair, she collaborates with Dr. Yvon Woappi, who directs the Synthetic Regeneration and Systems Physiology Laboratory at Columbia’s Vagelos College of Physicians and Surgeons. Although the systems differ, these collaborations are united by a common statistical question: what aspects of tissue repair are shared across conditions or species, and what aspects depend on age, treatment or environment?

For example, humans cannot regrow their injured joints well. When injury occurs, the body often replaces damaged tissue with scar-like material that lacks strength and flexibility. Meanwhile, zebrafish can rebuild an entirely functional jaw after its complete removal. “How do their

cells accomplish that? How do they know the blueprint of a jaw?” asks Dumitrascu. “The cells have to start growing from two different sides, and they have to work together in order to create a functional jaw. How do they know to end up in the right place?”

To investigate this phenomenon, Dumitrascu partnered with Smeeton’s lab. In this collaboration led by Smeeton’s team, Dumitrascu’s group contributed the statistical analyses that supported and contextualized the experimental findings. “We wanted to understand whether the presence of certain cells makes healing more efficient. To answer that, you need to know which cells are present—are there chondrocytes, fibroblasts, osteoblasts—and what they do,” she explains. The team discovered that multipotent neural crest–lineage cells were responsible for zebrafish jaw regrowth, findings reported in their paper *Dynamic cell fate plasticity and tissue reintegration drive functional adult synovial joint regeneration after complete resection*,

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**“How do cells detect the changes in the mechanics of the environment so that some become more rigid and stick to one another, and some become more fluid, so they flow past each other?”**

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published in *Nature Communications* in September 2025. “These findings suggest that even adult bones contain hidden biological programs that, if activated, could one day help humans regenerate healthy, fully functional joints,” Dumitrascu says.

Dumitrascu also works with spatial transcriptomics, a technology that measures which genes are active in specific locations within a tissue sample. “If you want to understand how a cell makes decisions, you must understand the context in which that cell lives, where it sits in the tissue and what its neighbors are.” For example, in embryonic development, cells sort themselves—some become placenta and others form the embryo. “How do cells detect the changes in the mechanics of the environment so that some become more rigid and stick to one another, and some become more fluid, so they flow past each other?” Dumitrascu wonders. “So we’ve been using spatial transcriptomics and morphology of the cells to understand how tense they are, how packed they are, and whether that has an effect on what the cells’ genes will do.”

Using data from developing mouse embryos, Dumitrascu’s team estimated the physical forces acting on each cell and also identified patterns of cell shape, mechanical stress, and gene expression that define where different tissue regions form. They outlined their method in the study titled [A computational pipeline for spatial mechano-transcrip-](#)

[tomics](#), published in *Nature Methods* in April 2025. This framework can be applied to many different spatial datasets, so it offers a way to study how chemical and physical signals work together to shape tissues during development or disease, Dumitrascu explains.

In an upcoming study developed in close collaboration with Dr. Yvon Woappi’s group, which Dumitrascu is excited about, her team developed a new method called Patches, which uses machine learning to analyze cells’ responses to various conditions. Single-cell genomics lets one study how individual cells behave and change under different conditions—such as during aging, after taking a drug, or following an injury. But analyzing this data is complicated. Many existing tools struggle to separate what is shared among all cells from what is unique to a specific condition, especially when data is incomplete or when cell types don’t perfectly match between experiments. The Patches method helps identify both the common genetic activity that all cells share and the specific changes that occur in response to the environment or treatment. And, unlike older methods, Patches can handle complex experiments—for example, studies that look at how cells respond over time or compare young and old tissues treated with drugs. When the team tested Patches on data from skin injury studies, it successfully revealed both the universal patterns of wound healing and the unique differences caused by aging or treatment.

Dumitrascu is also interested in multi-scale biology, an approach that investigates biological systems across multiple levels of organization and across various spatial and temporal scales. To do this, her team uses light sheet microscopy, a technology that allows one to peek inside a transparent organism to see and record how the cells move through space and time to understand how organisms develop and organs form. “I’m very interested in developing mathematical tools that integrate all these biological scales,” Dumitrascu says. “Biology is very complicated, there’s a lot of exceptions to rules,” she adds. “Math may not always be the right language for biology, but it lets us simplify things and helps us crystallize knowledge. And I am very interested in how knowledge is organized and how it makes an impact on humans and their health.”

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Hallou A, He R, Simons BD, Dumitrascu B. A computational pipeline for spatial mechano-transcriptomics. *Nat Methods*. 2025 Apr;22(4):737-750. doi: 10.1038/s41592-025-02618-1. Epub 2025 Mar 17. PMID: 40097810; PMCID: PMC11978512.

Blumenkrantz, M., Woron, F, Gagarin, E. et al. Dynamic cell fate plasticity and tissue reintegration drive functional adult synovial joint regeneration after complete resection. *Nat Commun* 16, 8570 (2025). <https://doi.org/10.1038/s41467-025-63596-8>.

# Tackling Rare Diseases One Gene at a Time

As an undergraduate electrical engineering student at Tsinghua University in Beijing, Chaolin Zhang, PhD, couldn't imagine running a wet lab. He anticipated a career in building inanimate objects like robots, yet here he is, an associate professor of systems biology, biochemistry and molecular biophysics at Columbia University, investigating some of the most tantalizing questions in basic biology—the RNA splicing mechanisms. “Initially, I was into robotics, but then I got interested in machine learning and computational biology,” says Zhang, who received his tenure in 2019. “And now I'm at the point of no return,” he quips.

Zhang studies the regulation of alternative splicing, a molecular process that removes introns and rejoins exons in precursor mRNA, particularly during neurodevelopment. More specifically, his lab focuses on mapping the binding sites of RNA-binding proteins (RBPs) and building new, state-of-the-art tools and technologies to facilitate that research. “The core of our work is to understand the interactions between the RNA-binding proteins, how they recognize their specific targets and how this interaction affects the expression of the gene,” Zhang says. “In our latest work, we frequently start from a target, a gene that's already known to play a key role in a disease and try to understand how the gene is regulated.”

Mutations in specific genes, such as the microtubule-associated protein tau (MAPT) gene, can cause RNA splicing errors. That leads to disruptions in the normal production of tau protein and the development of neurodegenerative disorders like tauopathies, including frontotemporal dementia (FTD). The MAPT gene was the subject of Zhang's recent study. “We found a major regulator that determines the splicing level of exon 10 of the tau protein,” says Zhang. “That switch determines why this exon is completely skipped in the early stage during embryonic brain development but is highly included in the adult brain. Now that we know this pathway, we can try to inhibit it to normalize the exon inclusion level to potentially prevent disease.”

At the RNA level, this intervention can be done by using an antisense oligonucleotide (ASO), a short, synthetic strand of nucleic acid designed to bind to an RNA molecule to control how it works. However, zeroing-in on the right type of ASO requires properly mapping splicing-regulatory elements (SREs), which is time-consuming and expensive. “You can do it with brute force, but we're trying to develop better methods,” says Zhang.

To speed up the process, his team developed SpliceRUSH, a high-throughput screening method, which they outlined in another study. When applied to SMN2, an already-known therapeutic target for spinal muscular atrophy, SpliceRUSH identified not only known splicing-regulatory elements but also a previously unknown one, proving that it could be an efficient way to screen for drug targets.

Zhang's team is also working on a computational algo-



rithm that can predict alternative splicing from genomic sequences. Called DeltaSplice, it's an AI system trained on deep neural network—akin to Google's AlphaFold that predicts the 3D structure of proteins from amino acid sequences. “DeltaSplice isn't at the level of AlphaFold yet, we're in early stages,” clarifies Zhang, explaining that one big challenge is data scarcity. The more data you have, the more precise your AI model is. The reason why computer vision is so successful is because one can get millions of pictures to train the algorithm. “But for genomic application we have only 20,000 genes,” he notes. To circumvent this problem, his team has been expanding the model to include related mammalian species because many genetic rules still apply, and AI can use this info to improve its performance.

Zhang hopes that the system will eventually provide insight into gene-regulatory mechanisms and help identify disease-causing mutations, such as for example, in autism—as well as the corresponding RNA-based drug targets. “Once we further improve the accuracy, it will be a real game changer,” he says. “We'll be able to make sense of how these mutations cause a disease and screen for therapeutics.”

Zhang knows the value of RNA-based therapeutics firsthand. As a PhD candidate at Cold Spring Harbor Laboratory, Zhang studied with advisor Adrian R. Krainer, whose RNA splicing research led to the development of Spinraza®, the first FDA-approved drug for spinal muscular atrophy. A genetic mutation that affects one in 10,000 kids, spinal muscular atrophy used to cut their lives short—many didn't live past their second birthday. “People say these diseases are rare, but they aren't actually that rare,” Zhang points out, especially given the fact that over 600 of rare neuromuscular disorders have been identified so far. “They affect a lot of families.”

With genetic sequencing techniques becoming widely available, genetic testing is now within reach for patients, and once you find the mutated gene, the next question is—how can we fix it? Zhang’s lab tries to fill this gap one gene at a time, zeroing-in on problem-causing genes to understand how each one is regulated and then translate that knowledge to help develop a potential therapeutic. With rare disorders no longer being that rare, a lot of patients can benefit from RNA-based drugs.

“That’s why in my lab, we’ve been focusing more and more on using our knowledge and our technologies on things that can make a real impact,” Zhang says. “We’ve been developing a kind of a pipeline that can be applied to many genetic diseases and lead to developing therapeutics for affected families. I hope that in my career I will see some of our efforts bearing fruit.”

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Xu C, Bao S, Wang Y, Li W, Chen H, Shen Y, Jiang T, Zhang C. Reference-informed prediction of alternative splicing and splicing-altering mutations from sequences. *Genome Res.* 2024 Aug 20. <https://pmc.ncbi.nlm.nih.gov/articles/PMC11368187/>

## Annual Retreat

The Department of Systems Biology’s 2025 annual retreat was held in October at the Roy and Diana Vagelos Education Center and it provided DSB faculty, post-docs, and students a chance to share and learn about their peers’ research, and network.

DSB researchers and graduate students participated in a poster competition held the first evening and reviewed by Systems Biology faculty judges. The three poster winners are clockwise from left:

- **Joseph Brian**  
*Engineering splicing-regulatory networks to mature stem cell-derived neurons*
- **Haoyu Wang**  
*Spatiotemporal Lineage Tracing of Lung Cancer Evolution*
- **Lei Chang**  
*Droplet Hi-C for Fast and Scalable Profiling of Chromatin Architecture in Single Cells*



## Welcome to Our New Cross-Appointed and Affiliated Faculty

### **Bianca Dumitrascu, PhD**

*Assistant Professor of Statistics and Herbert and Florence Irving Assistant Professor of Cancer Data Research (DSB Affiliate).*

Dumitrascu works at the intersection of machine learning and genetics. Her main research interest is understanding how local molecular rules give rise to emergent spatial patterns in the context of biological dynamical systems.

### **Bing Ren, PhD**

*Professor of Genetics and Development, Biochemistry and Molecular Biophysics, and Systems Biology.*

As a pioneer in the field of epigenomics, Dr. Bing Ren has devoted his career to unraveling the complexities of gene regulation and chromatin architecture.

### **Parisa Yousefpour, PhD**

*Assistant Professor of Biomedical Engineering (DSB Affiliate).*

Yousefpour designs and engineers RNA- and protein-based platforms to finely modulate immune responses, enhancing therapeutic efficacy while minimizing adverse effects.

## Around the Department, 2024-2025

### Grants, Awards, and More

**Barry Honig, PhD** and Raul Rabadan, PhD received a three year grant from ARPA-H for *Closing the Doors to all Pathogens*.

**Cory Abate-Shen, PhD** received a five-year grant from NIH for *Nuclear and non-nuclear functions of NKX3.1 in suppression of prostate cancer*.

**Cory Abate-Shen, PhD** received a five-year grant from Mark Foundation Center Award for *Mechanisms and therapeutic targeting of lineage plasticity and tumor progression in bladder and esophageal cancer*.

**Chaolin Zhang, PhD** and Harris Wang, PhD received a three-year grant from NSF for *Programmable RNA targeting and engineering using pentatricopeptide repeat proteins*.

**Chaolin Zhang, PhD** received a four-year grant from NIH/NINDS for *Engineering alternative splicing programs to accelerate maturation of stem cell-derived neurons*.

**Chaolin Zhang, PhD** received a one-year grant from Million Dollar Bike Ride (“MDBR”) for *A deep screen of RNA-based therapeutics for STXBPI syndrome*.

**Haris Wang, PhD** received a three-year award from NSF for *Elucidating principles of microbiome spatial organization using synthetic gut*.

**Haris Wang, PhD** and Virginia Cornish, PhD received a one-year award from NSF for *NSF Convergence Accelerator Phase 2*.

**Harris Wang, PhD** received a one-year award from Crohn’s & Colitis Foundation for *Stool-based RNA analysis to assess IBD status and drug response*.

**Jeremy Worley** was among Columbia scientists named Chan Zuckerberg Investigators to Advance Cell Therapies; he received a three-year grant to support his research.

**Michael Shen, PhD** received a five-year grant from NIH for *Molecular analysis of castration sensitivity in the prostate epithelium*.

**Mohammed AlQuraishi, PhD** received a two-year grant from the Michelson Found Animals Foundation, for *Design of protein- and peptide-based inhibitors of GnRH and GnRHR*.

**Mohammed AlQuraishi, PhD** received a six-month grant from the Dimond Light for *Accelerating structure-based drug discovery for the twenty-first century*.

**Mohammed AlQuraishi, PhD** received a three-year grant from NSF for *ANASBFU Convergence Accelerator Phase 2*.

**Mohammed AlQuraishi, PhD** received a combined grant from Astex, BMS and Takeda for *AISB-1: A Federated Approach Using Industry Data to Evaluate and Improve Performance of Co-Folding Prediction Model OpenFold3*.

**Mohammed AlQuraishi, PhD** received a four-year R01 from NIH for *Fast and slow prediction of stable and transient protein-protein interactions*.

**Peter Sims, PhD** and Chaolin Zhang, were awarded from NIH/NIGMS a T32 grant: “Columbia University Graduate Training Program in Computational and Systems Biology”.

**Peter Sims, PhD** received a four-year grant for *Generating Synthetic Lethality in Glioblastoma with a First-In-Class Non-Muscle Myosin. (Co-PI)*

**Peter Sims, PhD** received a four-year grant from NIH for *Preventing Fatal Infections in Children with Down Syndrome During Treatment. (Co-PI)*

**Peter Sims, PhD** received a five-year grant from NIH for *Bone as a Sensor of and Responder to Stress During Aging. (Co-PI)*

**Peter Sims, PhD** received a three-year grant from Allen Institute for *Mapping Immune Cell Niches in the Human GI Tract*

**Tal Korem, PhD** received a one year grant from Origyn Solutions to advance algorithm development for prediction of adverse pregnancy outcomes.

**Tal Korem**, PhD received a two year grant from the March of Dimes for *The Vaginal Immune-Microbiome-Metabolome Axis in Spontaneous Preterm Birth*.

**Tal Korem**, Harris Wang, Jeremy Worley, Mohammed AIQuraishi, Peter Sims and Dennis Vitkup were all awarded Research Stabilization Funds.

## Awards and Promotions

**Andrea Califano** was recognized by the Fox Chase Cancer Center with the Stanley P. Reimann Honor Award, the center's highest distinction.

**Chaolin Zhang**, PhD and Tal Korem, PhD were named Directors of Graduate Studies of the Systems Biology track of VIBRE effective on 10/1/2025.

**Cory Abate-Shen** was elected to National Academy of Sciences.

**Harris Wang** was promoted to Professor, effective July 2025.

**Itsik Pe'er**, PhD was named 2025 ISCB Fellow.

**Mohammed AIQuraishi**, PhD received The Harold and Golden Lampert Award for Excellence in Basic Science research.

**Tal Korem**, PhD was named Clarivate Highly Cited Researcher for 2025.

**Tim Olsen** (Sims Lab) - Group Leader, CZ Biohub NY

## Post-Docs and GRAs News

**Armer Chase**, GRA (AIQuraishi Lab) received a one-year NSF grant for I-Corps: Translation Potential of a Cloud Laboratory Platform for Laboratory Automation.

**Julia Rogers**, Post-Doc (AIQuraishi Lab) received a five-year grant from Burroughs Wellcome Fund for Learning the biophysical logic of cell signaling.

**Julia Urban**, GRA (Korem Lab) received a three year F31 grant from NIH for Deciphering the role of the vaginal microbiome in spontaneous preterm birth via consideration of host genetics.

**Maya Venkatraman** was named a 2025 recipient of the National Science Foundation (NSF) Computer and Information Science and Engineering Graduate Fellowship (CSGrad4US).

**Shahd EINaggar**, Analyst (Korem Lab) was selected a finalist for the Simons Graduate Fellowships in Ecology and Evolution.

**Wenxing Li** (Yufeng Shen lab) was named a Cardiovascular Development Data Resource Center (CDDRC) Fellow.

## Service Core News

**Information Technology Service Core.** For over 15 years, C2B2/DSBIT has provided essential IT services including co-location, big data storage, high-performance computing (HPC), and desktop administration. These services have been widely utilized by the Department of Systems Biology, various departments and centers, as well as individual research labs. During FY26 we completed the transition to the CUIMC IT, with all services and personnel transitioning as well.

**GECO, HTS, and SCC** are collaborating on bi[o]hub-funded projects led by PIs Andrea Califano and Peter Sims. In 2025, CRISPRi Perturb-seq screens were performed on tens of millions of cells across multiple immune cell contexts. HTS also developed single-cell PLATE-seq, enabling chemical genomic screens with single-cell RNA-seq readouts on over 50 million cells in 2026. This represents one of the largest publicly disclosed efforts to generate perturbational single-cell data for AI model training to date.

## Congratulations to the New Graduate Students

**Mick Aitken** (Rabadan Lab)

**Xi Fu** (Rabadan lab)

**Karin (Karen) Isaev** (Knowles lab)

**Logan Schwanz** (Wang lab)

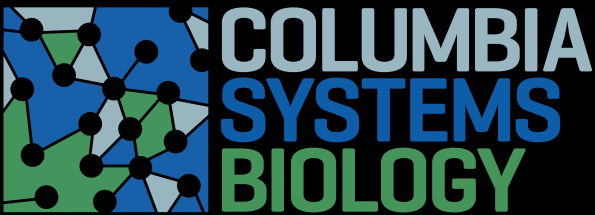
**Yiwei Sun** (Wang lab)

**Jiao Wang** (Vitkup lab)





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**IRVING MEDICAL CENTER**



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