Tom and Joan Steitz RNA Fellows

Yale Center for RNA Science and Medicine

2026 Fellows

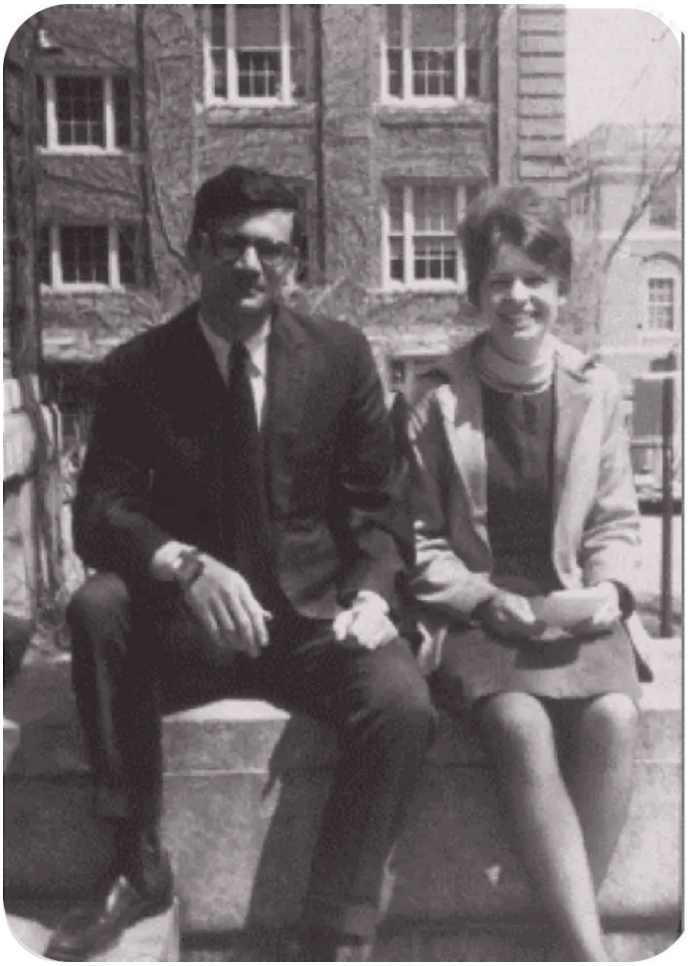
About the Fellowship

Joan (left) and Tom Steitz (right)

Joan A. Steitz, PhD, Sterling Professor of Molecular Biophysics and Biochemistry and alumna investigator of the Howard Hughes Medical Institute, donated her 2021 Wolf Prize to the Yale Center for RNA Science and Medicine. Her contribution led to the founding of the Tom and Joan Steitz RNA Fellows Program, which will honor Yale undergraduates, graduate students, and postdoctoral researchers who show promise as future leaders in RNA biology. The program also recognizes the scientific legacy of her late husband, Thomas A. Steitz, whose work profoundly shaped the field of structural biology.

The goal of the fellowship is to create an intergenerational community of RNA scholars that will foster each awardee's scientific excellence, career opportunities, and leadership potential through support from alums, peers, and faculty. The program is inspired by Joan Steitz's extraordinary history of mentorship.

Award



Tom (left) and Joan Steitz (right)

Each class of fellows will be recognized in February at a special Tom and Joan Steitz RNA Fellows Dinner. The fellows' mentors will also be invited. The RNA Fellows from previous years will be invited back each year for the Fellows Dinner honoring the next class of Tom and Joan Steitz RNA Fellows, thus growing the community of fellows. In addition, the winning essays of each fellow in the new class will be published in the Tom and Joan Steitz RNA Fellows Bulletin, which is disseminated annually to the community of fellows. The essays will also be publicly available on the Yale Center for RNA Science and Medicine website on the Fellows Program page.

The Yale Center for RNA Science and Medicine

Karla Neugebauer (left), Director of the Yale Center for RNA Science and Medicine, with the 2026 Fellows Srikar Gopinath (middle, back), Haejeong Lee (right), and Ryan Stanton (middle front).

The Yale Center for RNA Science and Medicine, led by Director Karla M. Neugebauer, PhD, administers the Tom and Joan Steitz RNA Fellows Program.

RNA science is experiencing an extraordinary period of discovery. Over the past few decades, researchers have revealed that RNA molecules play critical roles across biology and biomedical science. More recently, scientists have found that much of the genome—once thought to be silent—produces RNA. Although much remains to be understood, many RNAs are already emerging as important diagnostic markers and therapeutic targets.

Mission

The mission of the Yale Center for RNA Science and Medicine is to build upon Yale's strengths in RNA biology, foster interdisciplinary collaboration, and apply collective expertise to understand disease processes and develop new treatments. The Center brings together members from more than a dozen Yale departments, with laboratories across all three campuses.

Community and Programs

Center-sponsored activities include the annual retreat, RNA Club, workshops, and seminars featuring leading researchers. These efforts foster a strong sense of community and encourage collaboration among trainees, faculty, and clinicians across Yale.

Class of 2023

Class of 2023 Fellows (pictured)

Kyrillos Abdallah
PhD student, Yale University

Amr Balabaki
PhD student, MIT Biology

Kevin Chen
Postdoctoral Associate, Yale University

Emily Sutton
Postdoctoral Associate, Yale University

Sudheesh Parambil
Associate Research Scientist, Yale University

Annsea Park
Medical Resident, Stanford

Gaëlle Talross
Assistant Professor, University of Rochester

Class of 2024

Class of 2023 and 2024 Fellows with Joan Steitz (seated)

Denethi Wijegunawardana
PhD student, Yale University

Lucille Tsao
PhD student, Yale University

Hannah Barsouk
PhD student, Stanford

Moreen Ng

Ethan Strayer
Postdoctoral Fellow, CU Anschutz

Ningning Zhang
Associate Research Scientist, Yale University

Leo Schärfen
Assistant Professor, UMass

Zhiliang Bai
Assistant Professor, MIT

Class of 2025

Class of 2023–2025 Fellows with Joan Steitz (center)

Kyle Robik
Undergraduate student, Yale University

Loren Wilson
PhD student, Yale University

Hannah Maul-Newby
Postdoctoral Associate, Yale University

Jessie Mohsen
Postdoctoral Fellow, The Rockefeller University

Michael Grome
Associate Research Scientist, Yale University

Leo Schärfen
Assistant Professor, UMass

Ling Xu
Assistant Professor, UF Scripps

Class of 2026

Tom (left) and Joan Steitz (right)

Kenny Cheng
Undergraduate student, Yale University

Haejeong Lee
PhD student, Yale University

Christopher G. King
PhD student, Yale University

Ryan Stanton
PhD student, Yale University

Dingyao Zhang
PhD student, Yale University

Srikar Gopinath
Postdoctoral Associate, Yale University

Chengtao Xu
Postdoctoral Associate, Yale University

Kenny Cheng
Undergraduate student, Breaker lab, Yale University

Kenny Cheng, Class of 2026

My childhood dream job was to be a novelist, captivated by the letters on a page that could conjure fantastical worlds. Yet, during the COVID-19 pandemic, I became fascinated by a different language—one written in cells. With only four letters (A, U, C, and G), RNA can act as sensor, scaffold, and regulator, functioning beyond the central dogma.

Joining the Breaker lab in my first year at Yale, I learned that the RNA molecules we discovered and characterized likely originated from the RNA World, serving as an exciting record of life's earliest problem-solving strategy. Here, I've had the opportunity to study some of the strangest RNAs discovered, each pushing the boundary of what we think RNA is capable of.

My first project focused on Ornate, Large, Extremophilic (OLE) RNAs, one of the largest and most widespread bacterial noncoding RNAs with an unresolved biochemical function. Previous work has shown that OLE RNAs form ribonucleoprotein complexes essential for environmental stress response in bacteria. I investigated the consequences of disrupting this complex by tracking changes in cell morphology under high magnesium, cold temperature, and ethanol stress conditions. The mystery has only deepened since, with recent structural evidence suggesting that OLE RNAs may span the lipid bilayer as integral membrane RNAs, once again challenging traditional boundaries of RNA function.

More recently, I have focused on the discovery and characterization of riboswitches—compact structured RNAs that bind diverse ligands to regulate gene expression, often by controlling transcription, translation, or alternative splicing. This work underscores that a single RNA molecule can recognize chemistry and enforce a regulatory decision without a protein intermediary. I biochemically validated that the NAD^+ class I riboswitch recognizes ADP-ribose as a signal of NAD^+ consumption rather than directly recognizing NAD^+, as previously reported. I also worked on a miniature NAD^+ class II riboswitch, among the smallest known riboswitches, with a minimal H-type pseudoknot fold that binds NMN to regulate de novo NAD^+ biosynthesis and nicotinamide salvage in bacteria.

Through a collaboration with the Ailong Ke lab, I've also begun building my structural biology toolkit, connecting RNA discovery to biochemistry to structure. Our mechanistic insights into RNA sensing and regulation can translate into real tools, from antimicrobial strategies to RNA devices that respond to cellular states. Continuing in RNA biology, I hope to pursue a PhD in Chemical Biology, "writing" newly discovered RNA capabilities into engineered systems that expand the biological world.

Haejeong Lee
PhD student, Giraldez lab, Yale University

Haejeong Lee, Class of 2026

One of the reasons I love RNA biology is the field's willingness to challenge and redefine established principles. The discovery of RNA's diverse regulatory functions stemmed from questioning the notion that it is merely a passive messenger. Likewise, RNA therapeutics emerged by reframing inherent RNA instability from a liability into a safety feature. In the Giraldez lab, my curiosity about RNA's dual roles as both messenger and regulator led me to take a fresh look at how codon identity influences mRNA stability.

Codons are not just codes for amino acids—they are also regulators of RNA stability. The prevailing dogma links optimal codons to high translation efficiency and RNA stability, and non-optimal codons to reduced translation and stability. However, this statement presumes that each codon acts independently. Moreover, the sequence complexity of endogenous transcripts has hindered our ability to disentangle codon-specific effects from other regulatory features influencing mRNA stability.

I decided to dissect how distinct coding sequence elements – not just individual codons but also combinations of codons– regulate mRNA stability. To achieve this, I conducted a massively parallel reporter assay with thousands of simplified codon repeats and systematically measured reporter half-lives. My data demonstrate that RNA stability cannot be fully explained by a simple summation of individual codon effects. Instead, I found that a classically defined "unstable" codon can, in fact, confer stability depending on the identity of its neighboring codons.

Based on these findings, I developed a computational method to design coding sequences optimized for stability. I generated a luciferase mRNA that is more stable than those designed using codon identity alone, and have also applied my algorithm to clinically relevant RNA therapeutics. Furthermore, in applying my findings to endogenous transcripts, I discovered that codon pair usage varies with gene function: stable codon pairs are enriched in genes involved in cellular maintenance and gene expression, while unstable codon pairs are associated with immune response, signaling, and lipid pathways. I am excited about the potential of these discoveries to advance RNA-based medicine.

I began with a question: what does an optimal or non-optimal codon truly mean? My discovery that codon pairs function differently from individual codons in determining RNA stability challenges a central assumption in the field and offers a new strategy for understanding and optimizing RNA stability. I am excited to continue tackling fundamental questions in RNA biology, questioning assumptions, and embracing the maverick spirit that defines this field.

Christopher G. King
PhD student, Breaker lab, Yale University

Christopher G King, Class of 2026

Even for RNA, the Medium is the Message

Some say life began in an RNA World. While I hesitate to ascribe an origin story to a distant primordial event, it is unquestionable that an RNA World, once begun, never actually ended. This hasn't always been apparent. Proteins execute much of the chemical activity in a cell; carbohydrates and lipids and other metabolites are the fundamental units of anabolism and catabolism. DNA, of course, serves as a stable repository of genetic information. The role of RNA at first appeared rather vague. We now know this vagueness stems from its versatility.

All cellular proteins are synthesized by the ribosome, a massive ribonucleoprotein complex in which RNA serves to catalyze the peptidyl transferase reaction and to ratchet the complex along an RNA template, usually copied from genomic DNA. This RNA template, or message, is translated by RNA adaptors into the appropriate amino acid sequence. Perhaps the best way to define a living system is to identify an autonomous loop wherein RNA messages direct macromolecular machines to synthesize more RNA messages. Modulation of the content and distribution of these messages allows the system to respond to the environment.

Marshall McLuhan observed that "The medium is the message." McLuhan's insight was a sociological concept, but can we not apply it to biochemical systems? Biological information is primarily perceived via RNA: the control and interpretation of these messages must occur via processes amenable to RNA. Riboswitches, RNA thermometers, and other cis-regulatory RNAs exemplify this, in which the message itself responds to its environment. So do the many RNA-guided systems, in which short RNAs guide dedicated protein complexes to target RNA or DNA via base pairing. Under this view the self-splicing and self-cleaving ribozymes provide a simple means of controlling content, and RNA-induced phase separation a simple means of controlling distribution. What hidden secrets do vault RNAs, RNA viroids, endogenous retroviruses, long non-coding RNAs and RNA therapeutics hold in store?

This perspective emphasizes the various selective forces that act on RNA as a medium. Given the limited nucleotide alphabet, all RNA sequences possess the capacity to fold. At one extreme, structured RNAs with well-defined biochemical functions exhibit strong signals of epistasis via negative selection against non-functional folds. At the other extreme, RNA guides exhibit supreme programmability and facilitate rapid adaptation. And in between, every other RNA must function within its folding landscape. Recognition of these subtle constraints facilitates the discovery and design of novel functionality.

Ryan Stanton
PhD student, Gilbert lab, Yale University

Ryan Stanton, Class of 2026

Taking a trip seems simple. You just go. But everyone knows organizing a vacation requires a lot of thinking. The right destination, time, landmarks—each step takes considerable planning. The life of messenger RNA seemed simple, as an in-between the genome and its proteins. Now, researchers like me are excited: we are starting to explain how mRNAs organize their own journeys.

mRNA modifications can send mRNAs to distinct fates. Pseudouridine (Ψ) is the second-most abundant modification in humans. Made by pseudouridine synthases (PUS), it can enhance splicing and reduce turnover. Still, Ψ is not known to broadly affect events in an mRNA's life. Unfortunately, limited transcriptome-wide coverage limits knowledge of Ψ. How might you know what a Ψ is doing if you're not sure it's there? Human 5' untranslated regions (UTRs) are particularly under-surveyed. Reanalyzing latest generation Ψ mapping data, I found 90 percent of 5' UTR uridines have insufficient coverage to show if Ψ is present. As the site of ribosome loading to mRNAs, the 5' UTR is a regulator of translation initiation and is ripe for mechanistic evaluation.

In the Gilbert lab, I leverage biochemistry to find out when and how Ψ directly changes ribosome recruitment. I found that human PUS I purified can installed hundreds of Ψ across thousands of human 5' UTRs. I established initiation on mRNAs with a Ψ is generally enhanced over those same mRNAs with uridines, but not without exceptions. Eukaryotic initiation factors or other proteins may be sensitive to 5' UTR Ψ or the structures these Ψ nucleate. With structural data, pulldowns, genetics, and analytical polysomes, I aim to find how Ψ exerts control over translation.

A favorite MBB301b lecture I TAed discussed the hypothesis that each gene encoded a ribosome for each protein. Although wrong, it acknowledged each gene's regulatory powers. We appreciate now that mRNAs encode these features, and now is the moment we can explore them. I study RNA biology because we are at a fascinating time where we can interrogate biology quantitatively. We might determine contexts where Ψ is functional with sequencing and synthetic libraries. Single-molecule fluorescence resonance energy transfer and solved structures can explain physics establishing our biology. Marrying systematic studies across libraries with mechanistic ones on exemplary RNAs will best explain the breath of mechanisms mRNAs use to control translation. From transcription to deadenylation, mRNAs take their own trips. Now we're learning how they plan them.

Dingyao Zhang
PhD student, Lu lab, Yale University

Dingyao Zhang, Class of 2026

The field of RNA biology is undergoing a remarkable revolution. Advances in machine learning, coupled with new RNA sequencing technologies, are enabling new discoveries. Developing new computational tools in RNA biology holds strong promise for solving fascinating but previously challenging questions.

Since my undergraduate work on microRNAs and alternative polyadenylation of mRNAs, I have been passionate about RNA informatics. In 2021, with strong bioinformatics skills and deep biological understanding, I began my PhD at Yale, co-supervised by Dr. Mark Gerstein and Dr. Jun Lu. My graduate work has touched on three aspects of RNA biology: RNA expression, structure, and therapeutics. My PhD began during the pandemic. I therefore proposed that RNA-based therapeutic agents, such as siRNA and Cas13 CRISPR, could be designed before the emergence of a new viral strain that could cause an outbreak. Based on conserved regions in RNA viruses, I showed that anti-COVID-19 siRNAs and Cas13 gRNAs could have been developed years before the pandemic; I also designed sequences targeting potential future viruses (Zhang et al., Bioinformatics, 2022). Moreover, I am fascinated by the intricate spatial patterns of non-coding RNA expression, yet current technologies lack high-quality spatial profiling of such RNAs. Collaborating with the Fan lab on Patho-DBiT as the lead computational contributor, I developed the ASTRO pipeline to achieve first-of-its-kind spatial profiling of the entire transcriptome on clinically archived FFPE tissues (Bai, Zhang et al., Cell 2024; Zhang et al., Bioinformatics, 2026). Additionally, to address the challenge of predicting DROSHA-mediated microRNA processing efficiency, I employed statistical RNA folding and machine learning to develop a novel method to predict how RNA structure affects DROSHA processing.

I envision two areas for my future research. One of the most promising opportunities is to understand RNA "grammar and language" using novel machine-learning methods. Huge amounts of existing RNA-seq and CLIP-seq data provide essential input for models to encode and decode cis- and trans-regulation. I plan to leverage advanced machine learning methods to develop RNA sequence foundation models that uncover new insights into the RNA world. Additionally, with the rapid expansion of spatiomics methods, I envision building "Virtual Tissue" models based on spatial transcriptomics and other data to predict genetic and pharmacological impacts on tissues, thereby enabling in vivo-mimicking in silico experiments and drug testing. Ultimately, my goal is to both gain new knowledge in basic RNA biology and translate these insights into societal impact, from pandemic preparedness to precision medicine.

Srikar Gopinath
Postdoctoral Associate, Giraldez lab, Yale University

Srikar Gopinath, Class of 2026

At the inaugural Sid Altman Symposium, Craig Mello concluded his talk with a profound message: "RNAs never gave up control." A statement that deeply resonated with me. It emphasizes that RNAs are not merely middle men in the Central Dogma but persistent and active architects of life. The question that motivates me is simple: how do RNAs implement this control, and what are the consequences when it fails?

RNAs are remarkably diverse, each serving distinct regulatory functions. My postgraduate work focused on small RNAs in Hydra. My work revealed that miRNAs and piRNAs precisely control gene expression during regeneration. This discovery in an evolutionarily ancient organism demonstrates that RNA-based regulation is fundamental to biological processes and represents a deeply conserved feature of life. During my PhD, I studied a novel class of RNAs long thought to be degradation products.

Across multiple models of cell-state transition, I observed a consistent, temporal increase in RNAs mapping specifically to 5′ tRNA halves, indicating regulated biogenesis. I showed that these tRNA-derived small RNAs (tsRNAs) selectively modulate the translation and stability of defined mRNAs, shaping developmental and disease-relevant cell states. This work also highlights the versatility of RNAs; RNAs with established functions acquiring novel regulatory roles.

Building on my work in trans regulation, I joined the Giraldez lab as a postdoc to study cis control within mRNAs. 5'UTRs are dense regulatory modules that dictate translation efficiency, and mutations in these regions can alter protein output without changing protein identity. Using MPRAs, I analyzed over 1 million 5'UTR mutations observed across population and identified more than 100,000 that significantly alter translation, including mutations in key disease-relevant genes. These variants disrupt both canonical and novel regulatory elements, highlighting that the RNA encoded control is essential for biology and that its perturbation can drive disease.

Several studies, including my own, show that RNA-mediated regulation is pervasive and precise. Yet, we still lack a comprehensive understanding of post-transcriptional control. Many functional RNAs likely remain undiscovered, and we do not entirely know how cis- and trans-acting elements coordinate across various cellular contexts. Emerging technologies enable probing these mechanisms at unprecedented resolution, revealing regulatory layers invisible just a decade ago. I am excited to leverage these advances to systematically map the functional landscape of RNA and define principles governing post-transcriptional gene regulation. Because RNAs never relinquished control, deciphering their sophisticated regulatory architecture is essential for understanding life and advancing human health.

Chengtao Xu
Postdoctoral Associate, Ke lab, Yale University

Chengtao Xu, Class of 2026

My fascination with nucleic acids began during my doctoral work on DNA-based data storage, where I learned to encode, retrieve, and manipulate information using the precise base-pairing logic of DNA. It was intellectually intriguing work, but it was also confined to the test tube. The deeper question that began to occupy me was whether similar principles of information storage and control could operate inside living cells. That question drew me toward RNA, which is an active participant in cellular decision-making, capable of regulating gene expression, modulating protein function, and responding dynamically to the cell's needs. Redirecting that same engineering logic toward RNA felt like a natural next step, one that led me to join Dr. Ailong Ke's lab.

It was there that a casual weekend conversation set the course of my postdoctoral research. Cas9 is celebrated for its DNA-targeting precision, but it also harbors a powerful but hidden RNA-binding capacity. This observation prompted a provocative question: could IscB, the evolutionary ancestor of Cas9, be similarly redirected to target RNA rather than DNA?

The idea was conceptually appealing, but technically treacherous. IscB naturally binds dsDNA with high affinity, which would overwhelm any RNA-targeting activity. After considerable deliberation, we made a bold decision: remove the Target Adjacent Motif (TAM) recognition domain. There was no guarantee this would work, since eliminating a structural domain from an already compact protein risks destabilizing the whole complex. But the logic was sound, and the result was exciting. Without its TAM domain, IscB not only retained its RNA-binding capability but became a remarkably versatile RNA-targeting platform.

We named this repurposed system R-IscB, and its functional range exceeded our initial expectations. In human cells, R-IscB can be deployed for binding-based splicing perturbation, cleavage-based mRNA knockdown, trans-splicing to replace disease-causing exons, and ADAR2-facilitated A-to-I editing to correct genetic mutations at the mRNA level. All of this happens within a single programmable scaffold. Critically, R-IscB outperforms conventional Cas13-based platforms in several of genes, suggesting that evolutionary ancestors of modern CRISPR proteins may harbor untapped capabilities that predate the specialization of their descendants.

This discovery reinforced a conviction that has guided my research trajectory: the most transformative tools in molecular biology are often hiding within biological processes we thought we already understood. RNA-guided systems like R-IscB represent only the surface of what nature has invented. Beneath that surface lies a vast landscape of RNA-protein complexes with yet-to-be-characterized functions, embedded in cellular metabolism, cell cycle regulation, and defense mechanisms.

My long-term scientific goal is to explore this landscape systematically. By mapping the structural basis of RNA-protein interactions and connecting those structures to downstream biological functions, I will continue my journey to both deepen our fundamental understanding of RNA biology and develop the next generation of RNA-guided tools for cellular regulation, genome editing, and therapeutic intervention.

Eligibility for the Program

All current Yale undergraduate, graduate, and Postdoctoral Researchers are eligible.

Selection for the Program

An annual call for applications is issued in January. A committee of RNA Center Faculty members evaluates and selects awardees based on their personal essay, CV, and two letters of recommendation.

Please direct questions about the application to karla.neugebauer@yale.edu (*not a member of the selection committee*).

YALE CENTER FOR RNA
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