SMART TEAM SYMPOSIUM

MARCH 28, 2026 LONGMONT, CO

March 28, 2026

Innovation Center of St. Vrain Valley Schools
Longmont, Colorado

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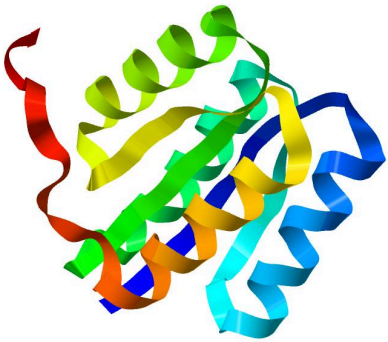
Transforming Science Learning

Welcome to the 2026 Colorado SMART Team Symposium!

About the SMART Teams Program

The SMART Teams (Students Modeling A Research Topic) program gives high school students hands-on experience with real scientific research. Teams explore protein structures using scientific literature and molecular visualization tools, then create 3D-printed models that highlight how a molecule's structure determines its function. These models help students tell the story of each protein, deepening their understanding of biology and chemistry while building critical research and communication skills.

Throughout the program, students develop abstracts and posters that showcase their findings. This symposium celebrates their curiosity, collaboration, and creativity — giving them a chance to share their discoveries with peers, mentors, and the community.



Agenda / Schedule of Events

Time	Event
9:00-9:10 AM	**Welcome & Brief Remarks** - Dr. Jackie Kapushion, Superintendent, St. Vrain Valley Schools
9:10-11:00 AM	**Poster Session** - Explore student protein modeling project posters
11:00-11:15 AM	**Break** - Refreshments and networking
11:15-11:45 AM	**Keynote Address** - Dr. Gayle Geschwind, Assessment Specialist, University of Colorado Anschutz
11:45-12:30 PM	**Lunch** - Networking and informal discussion
12:30-1:00 PM	**SWAP Meet** - Students exchange ideas, materials, and experiences
1:00-1:10 PM	**Closing Remarks**
1:10 PM	**Symposium Ends**

Keynote Speaker

Gayle Geschwind, PhD

Dr. Gayle Geschwind is an Assessment Specialist in the Office of Assessment, Evaluation, and Outcomes at the University of Colorado Anschutz. In this role, she applies item response theory methods of exam equating to ensure similar passing standards are maintained between different academic years, assists medical school faculty with exam generation by performing classical test theory analyses of exams and questions, and provides statistical expertise to various research endeavours. She graduated with her PhD in physics from the University of Colorado Boulder in 2024, where she focused on physics education research; her dissertation centered on quantitative analysis of assessment data surrounding undergraduate students' understanding of measurement uncertainty. During her time as a graduate student, she served as a science mentor for the Longmont High School SMART team for three years.

Acknowledgements

We would like to express our sincere gratitude to the following individuals and organizations for their invaluable support in making the 2026 Colorado SMART Team Symposium possible:

- **Teachers and Mentors** – for the countless hours spent working with students, guiding them through their research, and fostering a love of science.
- **Dr. Jackie Kapushion, Superintendent** – for her leadership of the St. Vrain Valley School District, her support of the SMART Team program, and for delivering the opening remarks.
- **Dr. Gayle Geschwind** – for delivering the keynote address and inspiring our students.
- **3D Molecular Designs** – for their sponsorship of the SMART Teams program and for printing the amazing 3D models of the proteins.
- **St. Vrain Valley School District** – for providing the facilities and sponsoring the symposium graphics.
- **Parents** – for their encouragement and support of our student researchers.

Your contributions have been essential in fostering a collaborative, innovative, and inspiring environment for our students.

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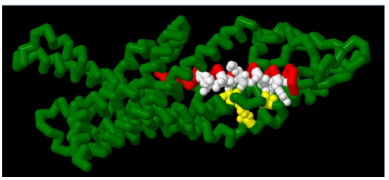
The Role of Membrane Bound Klotho-alpha in the Regulation of Phosphorus Absorption through FGF23 Pathways

Students: Kyle Borgemenke, Jacob Krah
Teacher: Daniel Shannon
School: Archbishop Moeller High School, Cincinnati, OH
PDB ID: 8TOH

Klotho-α functions as a stabilizer to the tyrosine kinase receptor for fibroblast growth factor 23 (FGF23) in the kidneys and parathyroid gland. In the presence of Klotho-α, FGF23 activates downstream signaling components, an absence of klotho-α inhibits FGF23 ability to regulate these signaling components. These pathways result in the regulation of calcitriol through 1-alpha-hydroxylase and 24-hydroxylase respectively. The former converts 25-hydroxyvitamin into 1,25-hydroxyvitamin D, while the latter regulates vitamin D levels in the body through the catabolism of the active form. This regulation of active vitamin D relates to the regulation of phosphorus levels in the body as calcitriol signals to increase the phosphate absorption rate in the small intestines through the VDR. FGF23 also decreases the expression of the NaPi-2a, 2c cotransporter. FGF 23 signals for the stopping of this pump through a separate intracellular signaling pathway. Phosphorus is an important element in energy production. This improves energy efficiency which explains Klotho's role in limiting the effects of aging and stress.

The Role of Glucagon Receptor Agonism in the Metabolic Balance of Retatrutide in Treating Obesity and Related Disorders

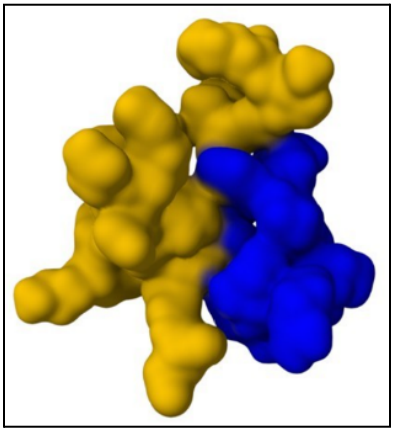
Students: Scott Kaplan, Giovanni Mikesell, Noah Pitz, Eli Umbaugh
Teacher: Daniel Shannon
School: Archbishop Moeller High School, Montgomery, OH
PDB ID: 8YW5



Obesity is one of the largest issues that researchers are attempting to effectively address through drug therapy. Many drugs, such as Ozempic and Mounjaro, employ semaglutides and tirzepatides, respectively, to facilitate weight loss using receptor agonists. These receptor agonists forcibly open receptors to further the absorption of incretin hormones, therefore signaling a decrease in hunger and stabilizing blood sugar levels. Receptor agonists currently used in medicine are primarily single or double receptor agonists. These are involved with the GLP-1 and GIP receptors; these hormones primarily involve the secretion of insulin, especially after eating meals. However, a recently discovered synthetic protein, retatrutide, involves the GLP-1 and GIP receptors in addition to the glucagon receptor (GCGR), giving it its status as a triple agonist. This additional pathway may allow for higher energy expenditure through increased thermogenetic activity, which in turn further progresses weight loss. GCGR signaling increases the breakdown of fatty acids in the brown adipose tissue (BAT), which is mitochondria dense. This metabolic pathway is responsible for removing energy in molecules. The increase in heat could potentially be responsible for the increase of heart rate during the human trials of retatrutide testing. GCGR is also responsible for the breakdown of glycogen into glucose using glucagon, which could then increase blood sugar levels. Although this may seem like an adverse effect, this ability to raise blood sugar levels could counter hypoglycemia.

A Hypothetical Explanation of Komodo Dragon Venom

Students: Adam Falci, Connor Connolly, Jack Bruns, Grant Collopy, Bentley Feihrer, Chase Gipson
School: Archbishop Moeller High School, Cincinnati, OH
Teacher: Daniel Shannon
PDB ID: 8S9Y



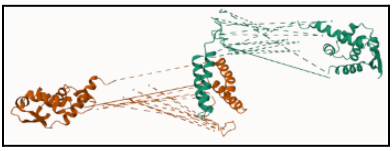
1-8, 26-39 The body protein 8S9Y (Yellow)

9-25 disulfide bond acts as a hook (Blue)

Komodo Dragons (*Varanus komodoensis*) are unique creatures in the animal kingdom. They are the largest monitor lizard alive today and the second largest ever. The Komodo Dragon is native to the Lesser Sunda Islands of Indonesia. Komodo Dragons have a specific venom that, rather than immediately killing the prey, slowly causes the animal to weaken and die, so the Komodo Dragon can then feast on the carrion. While Komodo Dragon venom is not widely researched, various snake species, such as the horned viper snake and Taipan, are hypothesized to have similar effects. Komodo Dragon venom is a cocktail of many protein groups found in snake venom; however, we researched CRISPS, Kallikrein, and natriuretic peptides. The Kallikrein group is responsible for its anticoagulation effects, which cause blood loss in its victims. The CRISPS proteins work to inhibit muscle contraction, which then weakens the prey. In Inland Taipan snakes (*Oxyuranus Microlepidotus*) one important part of their venom is natriuretic peptides, which are also found in Komodo Dragons. Natriuretic peptides are hormones that regulate blood pressure through normal vasodilation, but in venom, cause extreme vasodilation. This leads to shock from the loss of blood pressure and permeability due to the quick increase in vein size. As a result, we hypothesize that these same symptoms are also present in the prey of Komodo Dragons. In Taipan snakes, TNPc (Taipan Natriuretic Peptide C (8S9Y)) is an agonist for NPR-A (natriuretic peptide receptor A (A transmembrane guanylyl cyclase receptor protein for natriuretic peptides)), which causes the production of cGMP (cyclic guanosine monophosphate – a messenger molecule). cGMP activates cGMP-dependent protein kinases, which then mediate vasodilation. In Taipan venoms, the natriuretic peptides present are extremely potent and cause overstimulation in this pathway, leading to extreme vasodilation inciting further symptoms. This, combined with being a member of this venom cocktail, leads to the death of the prey, as the Komodo Dragon can easily track it down and consume it.

Lyssavirus P3: The Protein That Makes Rabies Deadly

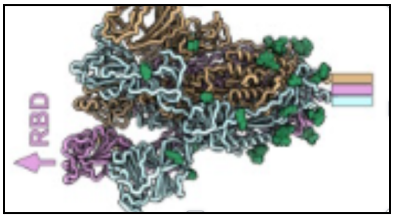
Students: Jake Allen
Teacher: Daniel Shannon
School: Archbishop Moeller High School, Cincinnati, Ohio
PDB ID (closest match): 8FUQ



The lyssavirus phosphoprotein (P) is one of the five proteins produced by the rabies lyssavirus. It exists in several forms, including the full-length P1 and the shorter P3. P1 mainly helps the virus replicate by working with the viral polymerase. P3, missing part of the N-terminal region, has different behaviors. P3, being smaller, travels more easily through the cytoplasm, and it diffuses easily in and out of the nucleus. It also can attach to a host cell's microtubules, which allows it to block immune system signals such as interferon-activated STAT1 more effectively. These changes make P3 an important factor in how lyssaviruses avoid the immune system.

Exploring the Past and the Future of the mRNA Vaccine

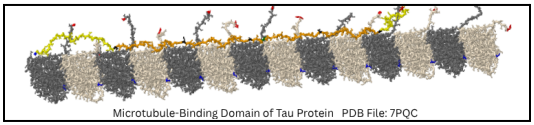
Students: Brynlyn Brockman, Norah Cassel, Anna Corkill
Teachers: Michele Palmgren
School: Blue Valley High School, Randolph, KS
Mentor: Inspired by Dr. Kataliin Kariko and her book, Breaking Through, My Life in Science; Ruth Hutson, Kansas State University
PDB ID: 6VSB



The mRNA vaccine is a preventative treatment that helps your body learn to fight diseases. The only available mRNA vaccines are for the prevention of the COVID-19 virus. While developed as a critical tool against SARS-CoV-2, mRNA technology has now emerged as a transformative breakthrough with connections beyond infectious disease prevention. This research summarizes current clinical data regarding the mechanism of mRNA and examines how lipid nanoparticles enable the intracellular delivery of synthetic mRNA to trigger an immune response. Unlike traditional vaccines that introduce a weak pathogen, mRNA technology uses the body's cellular machinery to produce the viral spike protein. Beyond COVID-19, this project analyzes emerging data on the application of mRNA platforms for non-infectious conditions. Current research indicates significant potential in programmable cancer immunotherapies, where mRNA is tailored to target patient-specific tumor antigens. As well as the treatment of genetic disorders through protein replacement therapies. Ultimately, the clinical and technical methodologies established during the pandemic allow for a new era of personalized medicine, provided that ongoing challenges in thermal stability and targeted delivery are resolved.

Alzheimer's Disease Pathology and Prevention

Students: Titus Cobb, Ben Klocke
Teacher: Michele Palmgren
School: Blue Valley High School, Randolph, KS
Mentor: Stephanie Hall, PhD, Kansas State Researcher and Asst. Professor
PDB ID: 7PQC


Microtubule-Binding Domain of Tau Protein PDB File: 7PQC

What is the role of the Tau protein in Alzheimer's Disease? The Blue Valley S.M.A.R.T. Team has been researching this question over the last year, through the partnership with Kansas State University's College of Veterinary Medicine. Shortly after, they expanded the focus of their research to include all domains of interest; the effects of the Tau Protein in mammalian brains were researched with the help of KSU.

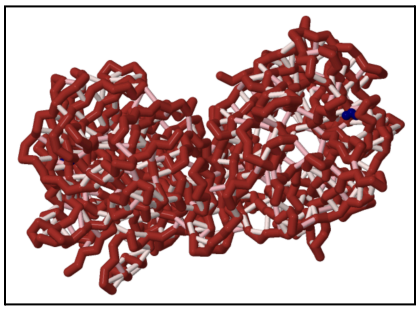
The Blue Valley S.M.A.R.T. Team initially aimed to research the role of hyperphosphorylation in the development of neurodegenerative diseases, specifically Alzheimer's Disease. Through extensive research, they reviewed the secondary characteristics of Tau in both normal and abnormal conditions. Utilizing a western blot technique, they quantified Tau in rat brains with Alzheimer's and without. The results showed a higher concentration of insoluble Tau in the brains with Alzheimer's Disease. After further research, they found that it was reported that exercise, increased levels of brain activity, and better sleep can all contribute to preventing onset or delaying effects associated with Alzheimer's disease. Increased glymphatic clearance, or the specialized waste removal process in the brain, is achieved through these three simple life-long activities, and can significantly reduce progression of neurodegenerative diseases. Through an increase of sleep quality and length, the flow of cerebrospinal fluid is greatly improved. Through exercise and increased levels of brain activity, glial cell function is also greatly improved. This results in a higher efficiency of waste clearance, primarily Tau and the Amyloid-beta protein, in the brain.

Tau is a significantly unstable protein, functionally important in the regulation of microtubule assembly and disassembly primarily found in cells of the nervous system. It is highly susceptible to coagulation due to a multitude of pathological secondary structures, resulting in neurofibrillary tangles associated with Alzheimer's disease. Phosphorylation and dephosphorylation are two primary post-translational modifications responsible for attachment and detachment of the microtubule-binding domain of the Tau molecule (amino acids 242-368) from the microtubule, along with acetylation. Hyperphosphorylation at different sites on serine, threonine, and tyrosine residues is an important component in repelling the protein from the microtubule. This also exposes both the PHF6 (VQIVYK) and PHF6* (VQIINK) hexapeptides, and is a key contributor to the formation of insoluble fibrillary Tau structures. These hexapeptides are extremely hydrophobic, leading to the insolubility and lipid binding properties of the Tau protein. When the hexapeptides become exposed, the Tau protein can form structures like β-sheets and develop into aggregates. The exposure eventually leads to formation of paired helical filaments (PHFs) and neurofibrillary tangles.

The next step in their research will be to continue exploring causes, consequences, and preventions of Alzheimer's Disease on a deeper level.

WaRPEd Into Darkness: The Gene That Can Change How You See the World

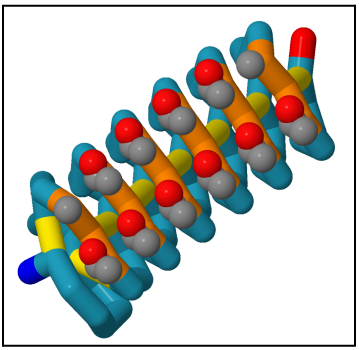
Students: D. Basurto-Sil, A. Diaz, C. Grams, S. Hughes, E. Jankowski, J. Krueger, M. Minessale, I. San Miguel, E. Wagner, O. Nencka, I. O'Brien, C. McBride, Z. Lavoe, L. Rodriguez, A. Toth, L. Tucker, E. Walker
Teachers: Megan Hahn, Stacey Strandberg
School: Divine Savior Holy Angels High School, Milwaukee, WI
Mentor: Hannah Follett, Dr. Joseph Carroll (Medical College of Wisconsin, Cell Biology, Neurobiology, & Anatomy, Ophthalmology and Visual Sciences, Joint Department of Biomedical Engineering)
PDB ID: 3FSN



The most common cause of inherited blindness in childhood is Leber Congenital Amaurosis (LCA): an autosomal recessive disease that causes vision impairment in babies and worsens as the child ages. LCA is caused by mutations in the Retinal Pigment Epithelium 65 (RPE65) gene which provides instructions to make the RPE65 protein. RPE65 is produced by the single layer of pigmented RPE cells located in the back-interior of the eye between the photoreceptors and the choroidal blood supply. The RPE65 is a critical enzyme within the visual cycle, regenerating the 11-cis retinal chromophore to its inactive form to absorb more light. The 11-cis-retinal is a derivative of Vitamin A that captures a photon of light, which is converted into a signal that is sent to the brain to process an image. As 11-cis retinal performs this process, it converts into 11-trans retinal, which then interacts with RPE65 so it can be recycled. The cycle continues by returning 11-cis retinal to the photoreceptors. However, mutations in RPE65 result in a deficiency of 11-cis retinal. Congenital loss of chromophore production due to RPE65-deficiency causes progressive photoreceptor degeneration and ongoing vision loss for individuals with LCA. Luxturna is the first FDA approved gene therapy for LCA. While vision improvements through this therapy are mostly permanent, the means of delivering the therapy can lead to retinal damage as gene therapy is injected directly to the eyes. Research continues to develop in order to improve gene therapy.

The Cold Never Bothered Us Anyway: How Antifreeze Proteins Enable Sub-Zero Survival

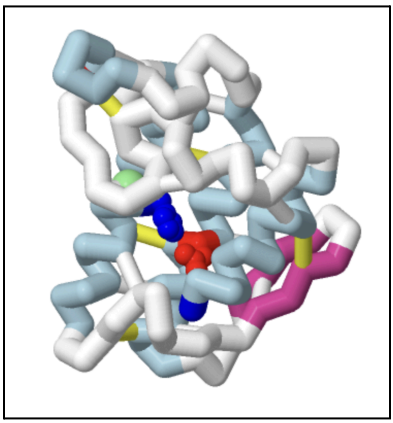
Students: Kiran Davis, Dominic Herrera, Quang Nguyen, Anya Storey-McFadden, Adam Wegner, Reese Wieder
Teachers: Chris Chou, Dick Martyr
School: Longmont High School, Longmont, CO
Mentor: Frederick Longshore-Neate, Ph.D. Candidate, Department of Biochemistry, University of Colorado Boulder
PDB: 1EZG



When ice crystals form within a cell they can puncture the cell membrane, ultimately causing the death of an organism. To adapt to frigid environments and avoid the detrimental effects of ice nucleation, some organisms have evolved to produce antifreeze proteins (AFPs), which inhibit ice recrystallization and nucleation. During the Cenozoic Era, organisms faced a rapidly cooling earth. To survive freezing temperatures, diverse organisms - including fish, insects, plants, fungi, and bacteria - independently evolved proteins that inhibit ice crystal growth. AFPs function by lowering the freezing point of water within cells without affecting its melting point, a phenomenon known as thermal hysteresis, and by ice recrystallization inhibition. Many AFPs bind to ice crystals through threonine (Thr) residues and have hydrophobic binding domains. In the yellow mealworm, *Tenebrio molitor*, its antifreeze protein (TmAFP) consists of 84 amino acids, including seven tandem repeats of 12 amino acid groups stabilized by cysteine (Cys) disulfide bonds. On one surface of the protein, Thr-Cys-Thr motifs form a flat, right-handed beta helix that creates a superhelix capable of binding to the surface of an ice crystal. TmAFP binds to ice crystals on the basal and prism planes, adsorbing to the surface to produce a lemon-shaped ice crystal morphology. Traditional antifreeze chemicals, such as ethylene glycol, used to inhibit ice formation can pose environmental threats. As a result, researchers are exploring AFPs as a less harmful alternative. However, large-scale production of AFPs can be cost-prohibitive. Instead, scientists can use insights from naturally occurring AFPs to design new, nontoxic antifreeze materials that prevent ice nucleation. Additional research may also focus on genetically modifying plants to produce AFPs, enhancing their resistance to freezing. The Longmont High School SMART (Students Modeling A Research Topic) Team has constructed a 3D-printed model of a *T. molitor* antifreeze protein to investigate its structure-function relationships.

$sPLA_2$: The Ssss-cience of Snake Bites

Students: Chace Johnston, Karina Madinger, Tristan Perry, Owen Sage, Noah Kline
Teacher: Kelly Lubkeman
Mentors: Aiden Wegner, Ph.D. Candidate, Department of Chemical and Biological Engineering, and Isabel Crooker, Ph.D. Candidate, Department of Biochemistry, University of Colorado Boulder
PDB ID: 1POA



The Phospholipase A_2 (PLA_2) superfamily contains hundreds of enzymes that all fulfill the same function of breaking down phospholipids by cleaving off their sn-2 hydrocarbon tails. Inside human cells, PLA_2 performs several key cellular functions including the production of signaling molecules and the digestion of dietary fats. However, PLA_2 can also enter the human body unexpectedly through animal toxins – such as snake venom – where it causes disruption of the cell membrane and eventually leads to tissue death. Due to PLA_2 and other toxins, the World Health Organization classifies snake bites as the "deadliest neglected tropical disease." (World Health Organization, 2023) PLA_2 proteins range in size from 14 to 18 kDa, including three conserved alpha helices, and six to eight disulfide bridges stabilizing the molecule. The enzyme's catalytic dyad is nestled between the α2 and α3 helices and consists of a histidine (His47) and aspartate (Asp93), where the lipid cleavage takes place. In order for the protein to react, a Ca^{2+} ion cofactor must be present, stabilized by a glycine (Gly30). In addition, a flexible C terminal loop allows PLA_2 to change conformation in order to interact with a number of natural lipids. As one of the main components of highly toxic snake venoms, research on PLA_2 aims to develop better antivenoms, with the hope of saving people from death (81,000 annually) or permanent injury (400,000 annually). (World Health Organization, 2023) Other intriguing directions for future studies of the PLA_2 superfamily include cancer research, degenerative joint disease, and cardiovascular disease.

AI-Assisted Prediction Models for Primary Anterior Cruciate Ligament Injury Risk and Recovery Time using Basketball Athlete Performance Data

Student: Ben Zhang

Anterior Cruciate Ligament (ACL) injuries are among the most common and debilitating injuries in basketball athletes. Traditional clinical approaches and existing studies often rely on observational data or systematic reviews, limiting predictive accuracy. This project develops AI-assisted machine learning models to predict both primary ACL injury risk and recovery time, using basketball athlete demographics, training patterns, and performance metrics.

Two datasets were analyzed, capturing variables such as age, sex, height, weight, training intensity, training hours, rest days, jump height, speed, and rehabilitation efficiency. Injury risk was classified using Random Forest and Logistic Regression, while recovery time was predicted with Random Forest, Gradient Boosting, and Linear Regression. Model performance was evaluated using accuracy, precision, recall, ROC-AUC, RMSE, MAE, and R2. The Random Forest models consistently achieved the best performance, accurately classifying injury risk and closely predicting recovery time.

Feature importance analysis revealed that rest days and training load were the strongest contributors to injury risk, while rehabilitation efficiency, jump height, and speed were most predictive of recovery time. These findings demonstrate how machine learning models can serve as interpretable frameworks, representing complex relationships between athlete characteristics, training behaviors, and injury outcomes.

This work highlights the potential of AI and predictive modeling in sports medicine, providing a data-driven approach for injury prevention and rehabilitation planning. The project establishes a reproducible framework that can be adapted for clinical and athletic settings, showing how computational models can inform real-world decision-making in athlete care.

Thank You for Attending the 2026 Colorado SMART Team Symposium!

We are thrilled to celebrate the incredible work of our student researchers and the dedication of their mentors, teachers, and families. Your curiosity, collaboration, and passion for science have made this symposium a truly inspiring event.

For a closer look at all of this year's SMART Team projects, including **abstracts**, **posters**, and **3D-printed protein models**, scan the QR code below. Explore the creativity and discovery of our **SMART Teams**, and see how students are bringing molecular science to life!

Thank you for supporting the next generation of scientists!