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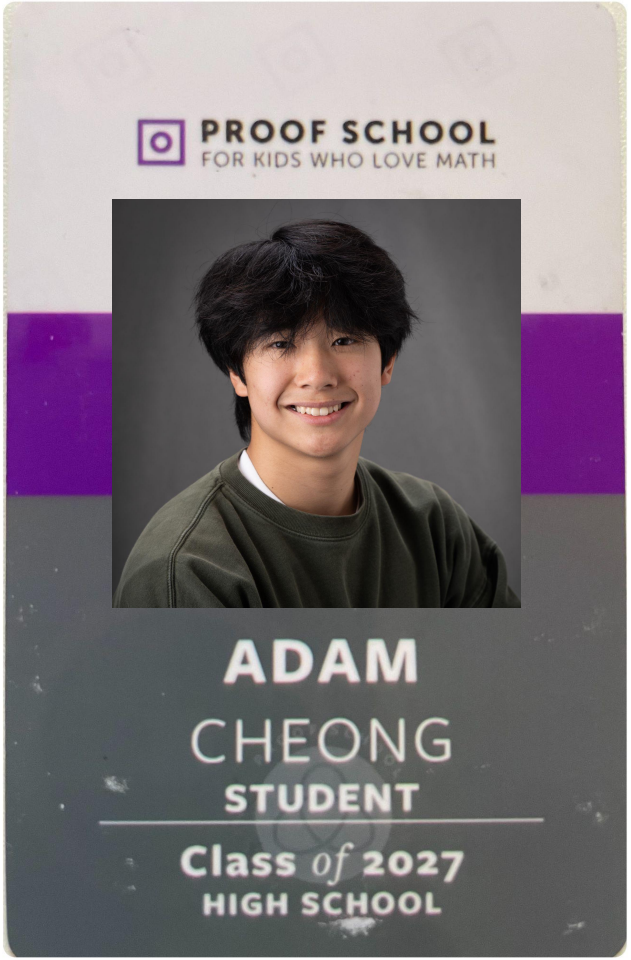
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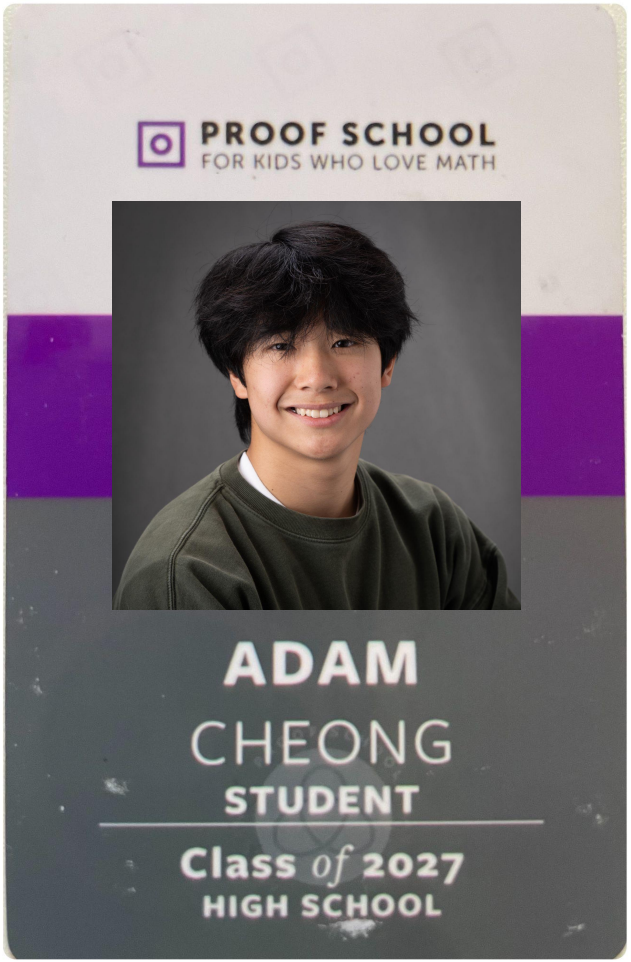

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- 11th: US History & the American Essay
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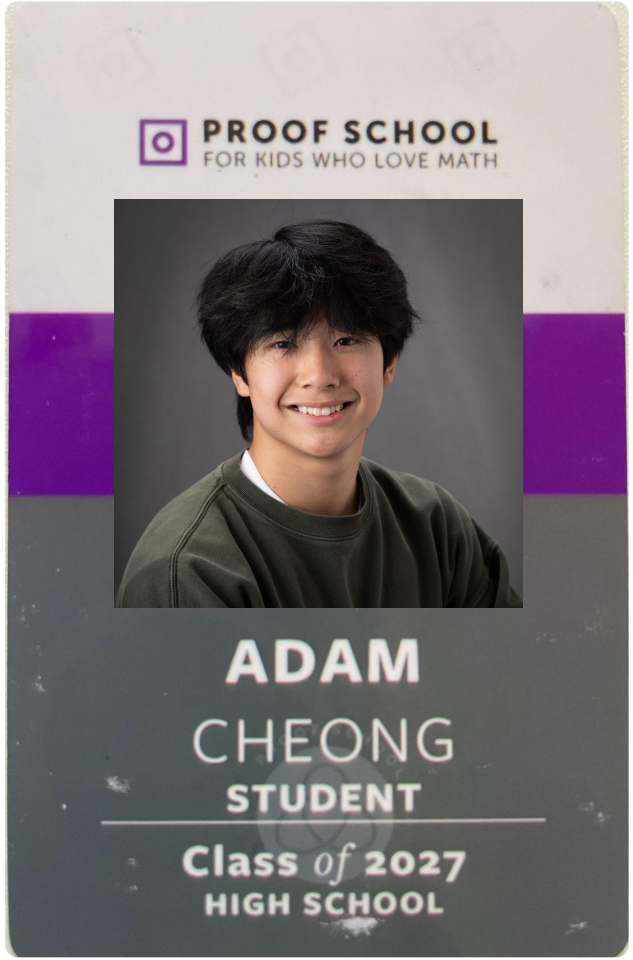
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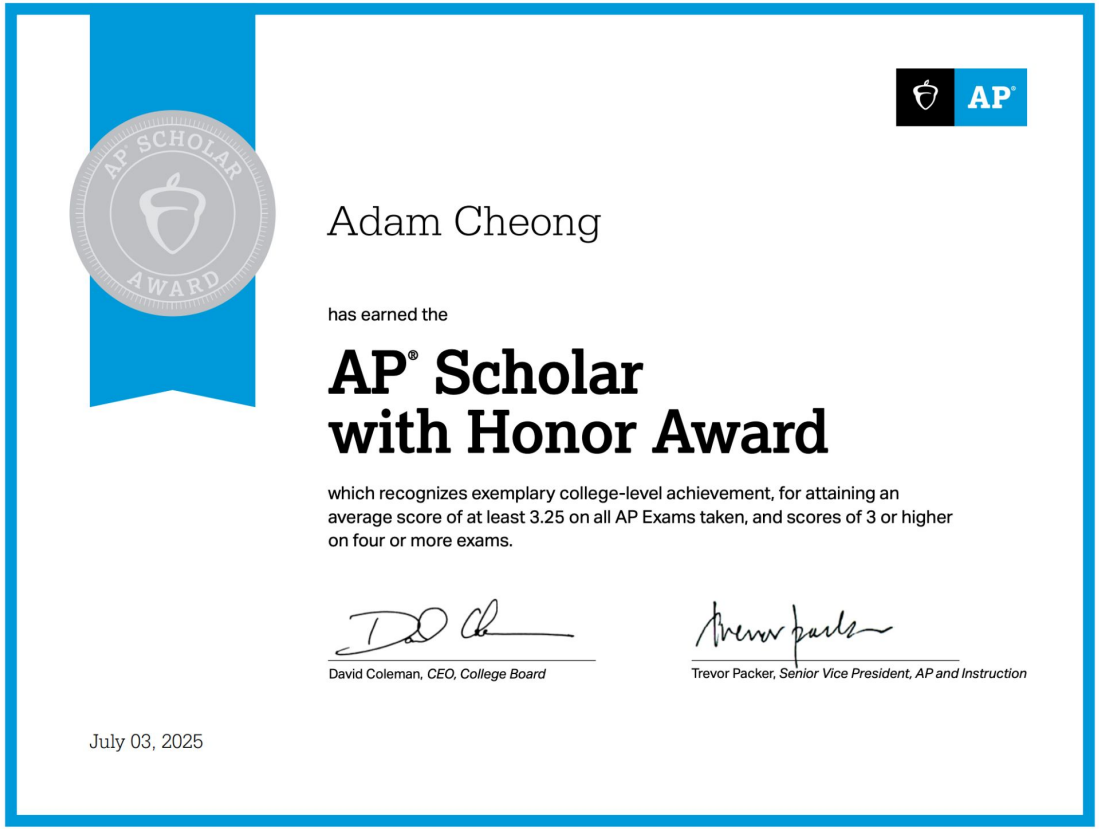
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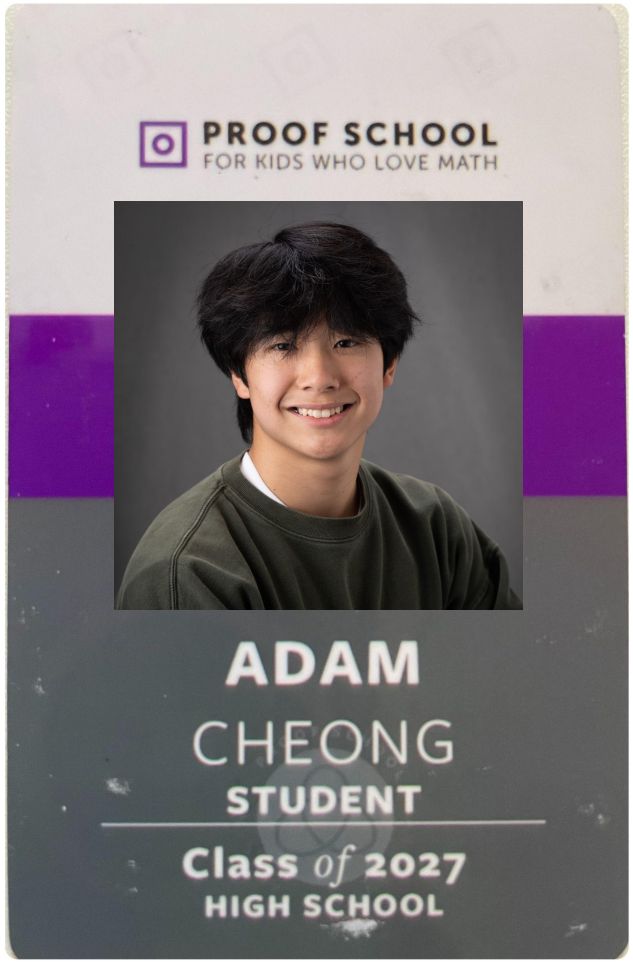

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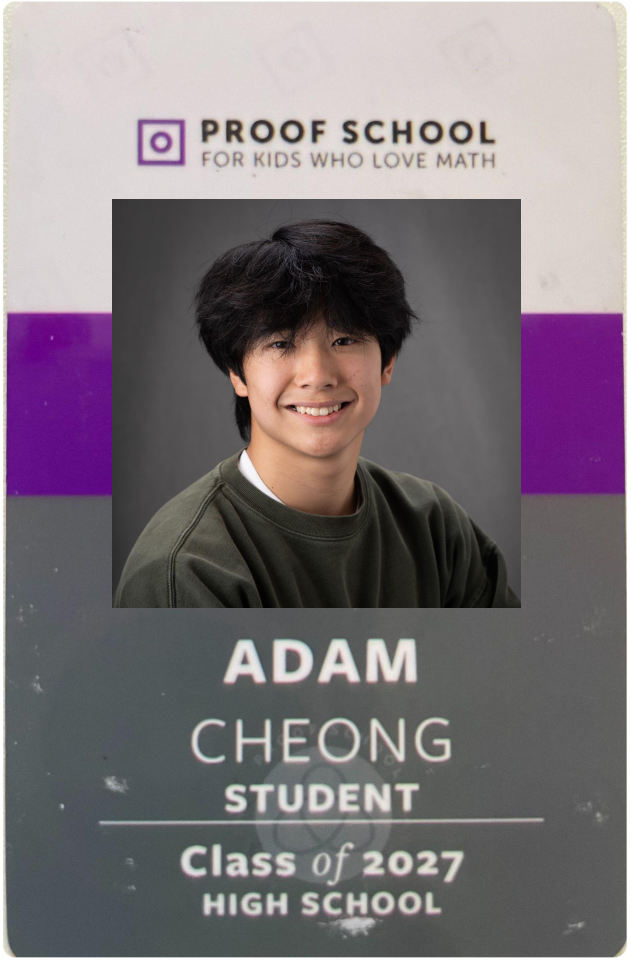
AMC 10 A	105	136.5	112.5
AMC 12 A	96	150	127.5
AMC 10 B	99	133.5	105
AMC 12 B	100.5	145.5	127.5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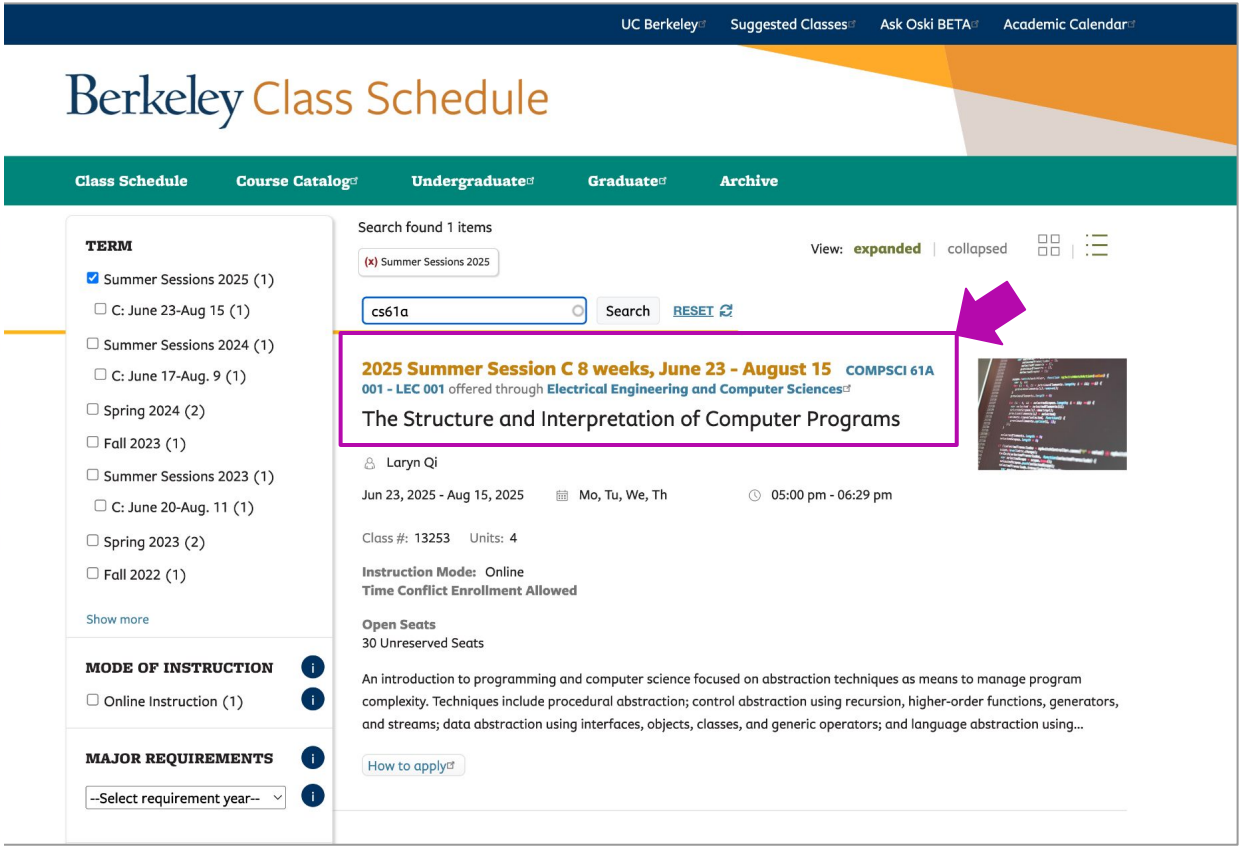
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The Structure and Interpretation of Computer Programs
Laryn Qi
Jun 23, 2025 - Aug 15, 2025
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05:00 pm - 06:29 pm
Class #: 13253 Units: 4
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An introduction to programming and computer science focused on abstraction techniques as means to manage program complexity. Techniques include procedural abstraction; control abstraction using recursion, higher-order functions, generators, and streams; data abstraction using interfaces, objects, classes, and generic operators; and language abstraction using...
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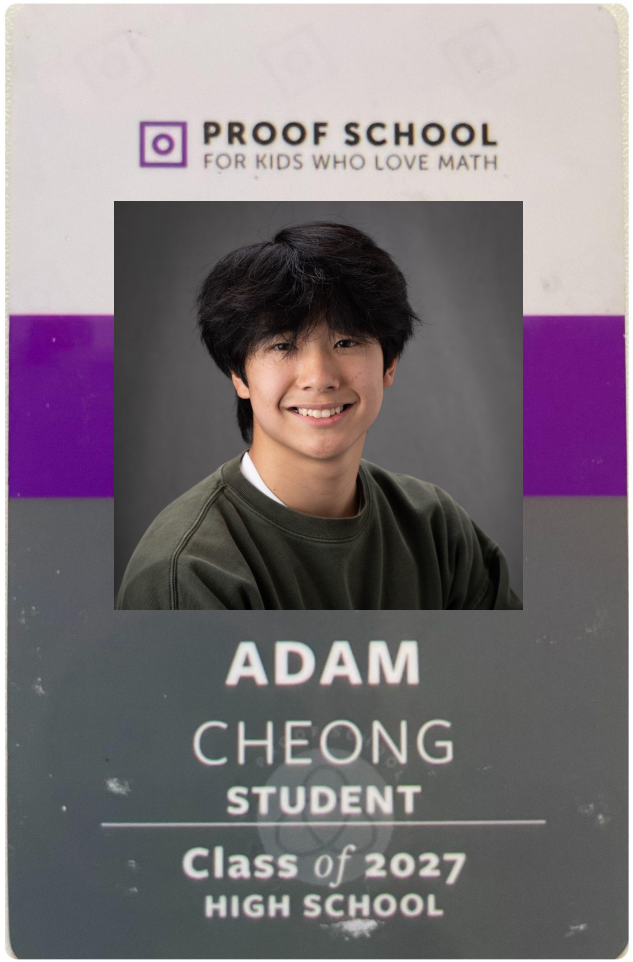

Enrollment

Summer 2025

Class	Title	Un.	Gr.	Pts.
COMPSCI 61A (Session C)	The Structure and Interpretation of Computer Programs	4	A	16

Summer 2026

Class	Title	Un.	Gr.	Pts.
COMPSCI 61BL (Session C)	Data Structures and Programming Methodology	4	—	—
DATA C8 (Session C)	Foundations of Data Science	4	—	—

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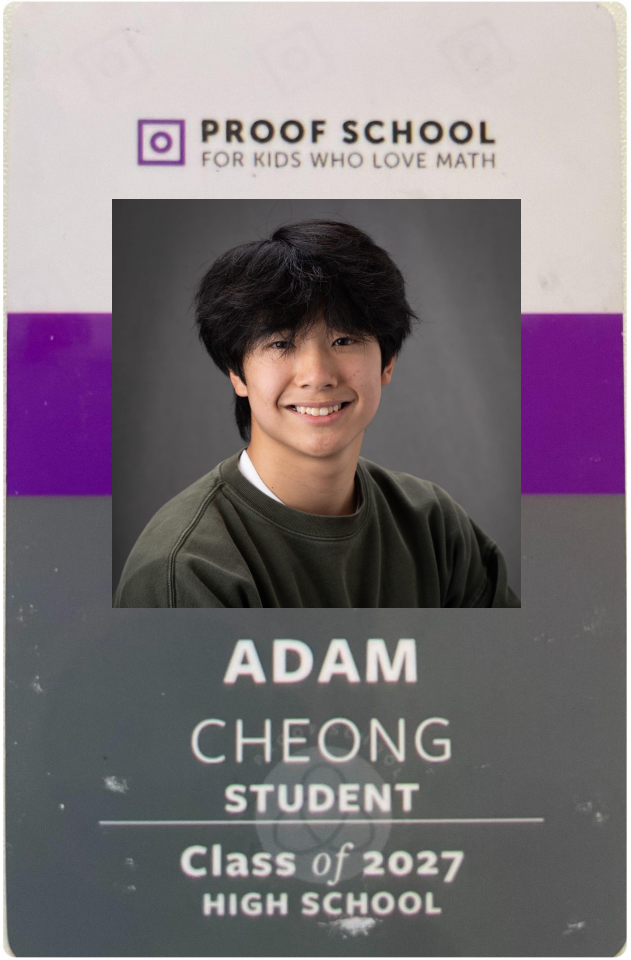
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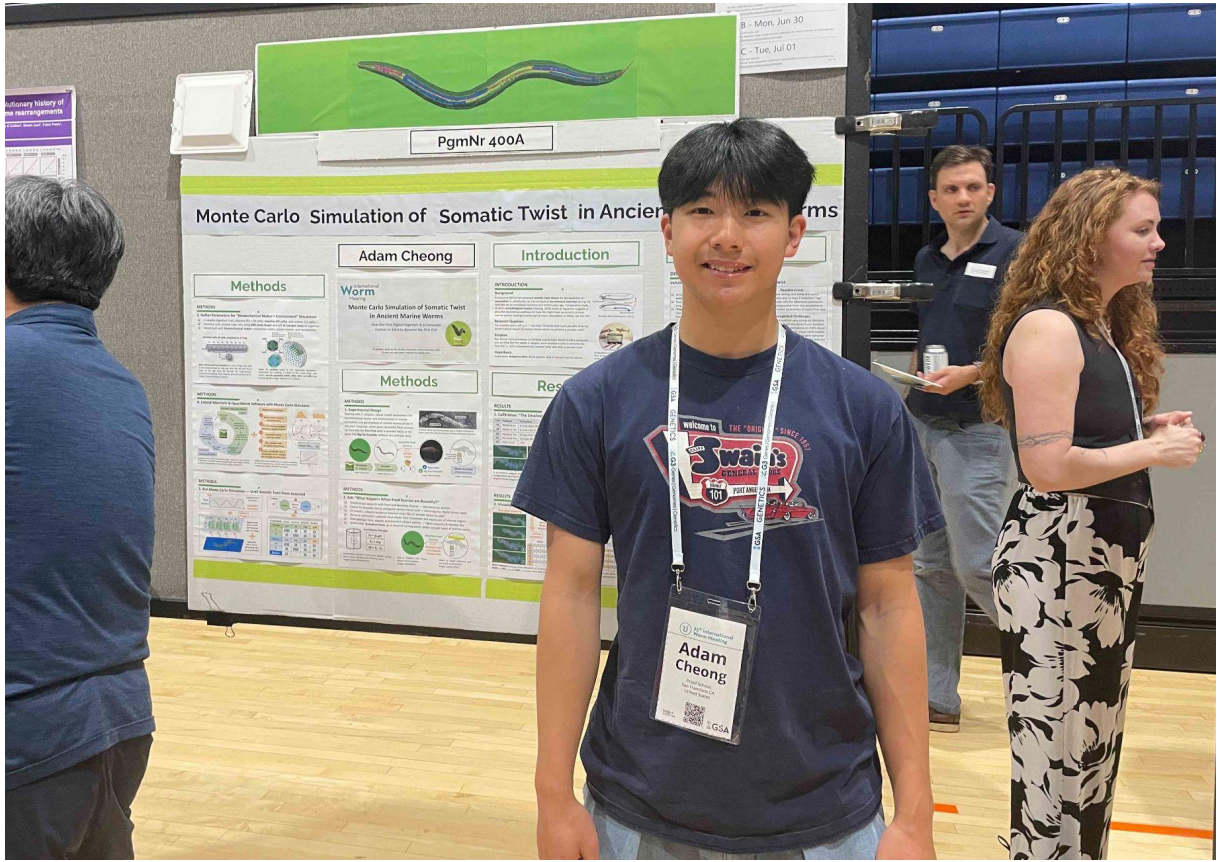
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How Cancer Cells Undergoing a Treatment Course Evolve to Survive Multiple Mass Extinctions

Adam Cheong[1,2], Mark Cheong[2], and Paul Fong[3]

[1]Proof School, [2]UC Berkeley, [3]Duke University

Project Summary

The vast diversity of genetic and phenotypic characteristics observed in human cancers, according to Hanahan (2026), can be understood in the context of how cancers arise via multistep pathways of tumorigenesis that lead to the formation of primary tumors, which subsequently evolve and progress due to selective advantage in the face of multifaceted barriers to continuing proliferative expansion, resulting in metastasis and adaptive resistance.

Here we elucidate the adversarial viewpoint; a press-pulse framework borrowed from paleobiology (Arens & West, 2008) for modeling therapeutic intervention targeted at the primary tumor, whose microenvironment undergoes terraforming and subsequently becomes unfit for cancer cells proliferation, leading to multiple *mass extinctions* over the course of cancer treatment, finally resulting in remission as the residual cancer cells turn quiescent.

The population dynamics driven by tumor growth vs. dose response is captured in our proposed model, as the cancer cells population follows a Gompertzian growth curve after metabolic reprogramming but shrinks in response to drug treatment. In particular, to shrink a tumor over multiple treatment cycles, dose responses are modeled as the outcome of catastrophic events decimating cancer cells as intravenous antibody-drug conjugate (Madhusoodanan, 2024) diffuses across blood vessel walls and penetrates the tumor, driven by concentration gradient according to Fick's laws.

Tumoral evolution within our framework offers mechanistic insights into the structure of a particular fitness landscape characterized by the selective pressure upon residual regrowth. We illustrate how subpopulations of residual cancer cells evolve to survive multiple mass extinctions originated from a prescribed course of antibody-drug conjugate targeting the primary tumor, potentially laying the seeds for future metastasis or adaptive resistance. We propose to construct a simulation testbed to further explore cancer population dynamics to better understand the mechanism of relapse.

Introduction

Antibody-Drug Conjugates Show Superior Response when Dosed at MTD

Fig. 1: Antibody-Drug Conjugate (ADC)[19] Image Credit: Drago & Norton (2021)

Over the last two decades, an increasing number of antibody-drug conjugates (ADCs) have been approved for cancer treatment, broadening therapeutic options for patients beyond traditional small molecule drugs. Through their unique mode of action as "magic bullets", these targeted therapies hold the promise of redressing the poor balance between *safety* and *efficacy* with traditional chemotherapy.[8, 9]

The "sweet spot" between the lowest concentration that produces a desired therapeutic effect (minimum effective dose or MED) and the concentration just before toxicity kicks in (maximum tolerated dose or MTD) is called the *therapeutic window*.

When dosed at or near MTD, ADCs display improved efficacy over small molecules in trials. Across the board, ADCs have already achieved greater beneficial outcomes for patients compared with traditional chemotherapy. The *objective response rates* (ORR) of ADC treatments frequently score above 40 percent in breast, gastric, urothelial, and lung cancer trials where small molecules rarely exceed 25 percent.[2, 8]

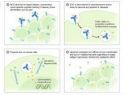

Fig. 2: Mechanism of Action for Antibody-Drug Conjugates[19]

Research Question

How to Make a Tumor Microenvironment "Most Unfit" Under Press-Pulse?

Four forces that drive cancer cells population dynamics:

(a) **Tumor Growth**: proliferative cancer cells harness aerobic glycolysis to generate energy rapidly to drive tumor growth;

(b) **Pulse Therapy**: dose responses are the outcome of catastrophic events decimating cancer cells as intravenous drug diffusing across blood vessel walls penetrates the tumor;

(c) **Somatic Mutation**: somatic mutation during residual regrowth in between treatment cycles selects for drug-resistant cells that drive future cancer recurrence; and

(d) **Press Therapy**: chronic microenvironmental stress (e.g., energy deprivation from starvation of glucose) slows down cell cycles and reduces cancer cells population resistance.

Note: *Cancer cells compete for resources in order to grow rapidly, while evading counteractacks.* Mathematical models of *tumoral evolution* describe how cancer cell populations grow with aerobic glycolysis but shrink under drug treatment, just as Lotka-Volterra equations describe the population dynamics of snowshoe hares and the lynx that feed upon them. New complexity in population dynamics arises when somatic mutation across treatment cycles and metabolic intervention are introduced and added to the model.

Fig. 3: Terraforming a tumor microenvironment under press-pulse.

Methods — Growth

Modeling Tumor Growth with a Gompertzian Curve

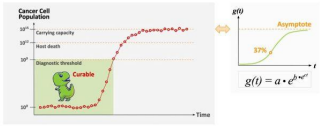

Fig. 4: **Gompertzian tumor growth**: initial growth at the inflection point appeared exponential, before slowing to approach the asymptote (i.e., asymmetric sigmoid curve).

Methods — Pulse

1 Dose at Maximal Effective Concentration — Dependent on Size of Tumor

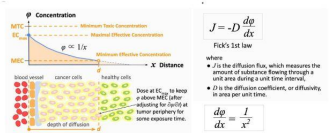

Fig. 5: **Fick's laws** describe diffusion driven by a concentration gradient, from high-concentration intravenous space to low-concentration intercellular space.

$$J = -D\frac{d\varphi}{dx}$$

Fick's 1st law

where

- J is the diffusion flux, which measures the amount of substance flowing through a unit area during a unit time interval,
- D is the diffusion coefficient, or diffusivity, in area per unit time.

$$\frac{d\varphi}{dx} \propto \frac{1}{x^2}$$

Concentration gradient formula

2 Compute Dose Response Curve — for Each Tumor Size and Shape

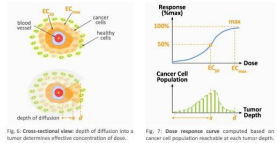

Fig. 6: **Cross-sectional view**: depth of diffusion into a tumor determines effective concentration of dose.

Fig. 7: **Dose response curve** computed based on cancer cell population reachable at each tumor depth.

3 Adjust Dose Response Model — as Concentration Decreases Over Time

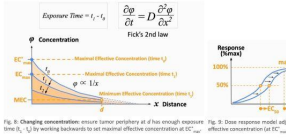

Fig. 8: **Changing concentration**: ensure tumor periphery at x has enough exposure time ($t_i - t_\delta$) by working backwards to set maximal effective concentration at EC_{max}.

Fig. 9: Dose response model adjusted for maximal effective concentration (at EC_{max}).

Discussion — Mutation

Cancer Cells Evolve in Between Treatment Cycles to Survive Mass Extinctions

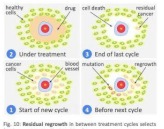

Fig. 10: **Residual regrowth** in between treatment cycles selects for drug-resistant cells that drive future cancer recurrence.

Would starving *proliferative* cancer cells of glucose improve dose response for cancer patients?

Could one switch to a *reduced dose* regimen without compromising dose response by going on a calorie-restricted diet to bring down the glucose level, which slows down the cell cycle and makes proliferative cancer cells more vulnerable?

It follows that one could then apply a *denser* dose schedule to suppress residual regrowth while also shortening treatment cycles.

Reduced heterogeneity in the tumor would then make relapse less likely, thus maximizing disease-free survival (DFS) for cancer patients.

Discussion — Press

Making Proliferative Cancer Cells Vulnerable Through Bioenergetic Intervention

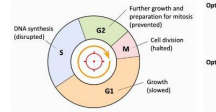

Fig. 11: **Proliferative cancer cell cycle**: glucose restriction can cause cancer cells to stay in G1 phase longer (or become quiescent), so cell-cycle (S/G2/M) targeting drug has ample time to enter cancer cells before the start of its targeted phase (e.g., to disrupt DNA synthesis), thus improving dose responses.

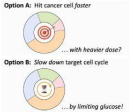

Fig. 12: **Epiphany**: either: (A) magic bullet moves faster to hit cancer cells with heavier dose of ADC at a higher concentration gradient; or (B) we slow down target cell cycle awaiting magic bullet arrival.

Conclusions

The Dose Makes the Poison: "Pulse Train" Designed to Suppress Regrowth

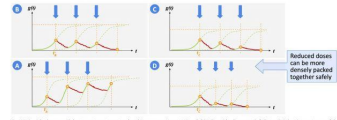

Fig. 13: Residual regrowth between treatment cycles drives somatic mutation: (A) high residual regrowth fails to shrink primary tumor, (B) baseline residual regrowth shrinks primary tumor but neglects somatic mutation, (C) low residual regrowth under press therapy reduces somatic mutation, and (D) least residual regrowth under press therapy minimizes somatic mutation with a denser dose schedule.

Future Work

Safer ADC Regimens That Also Maximize Disease-Free Survival?

Fig. 14: *Chain-of-thought reasoning* for our research design.

We illustrated how tumor size and bioenergetics together determine dose response, which we see as multifaceted and not simply a static curve as commonly believed. Importantly, a cancer patient can improve dose response when combined with bioenergetic intervention.

Our ongoing research has therapeutic implications: a *safer* ADC regimen realized through improved dose response modeling benefits not just late-stage cancer patients considering palliative care, but also early-stage cancer patients seeking alternative treatment paths that maximize long-term *disease-free survival*[13].

By taking a longer view of DFS as an alternate endpoint, we hope to optimize *safety and efficacy* together by limiting residual regrowth during treatment — unlike traditional chemotherapy preoccupied with maximizing cell kill in the primary tumor, sacrificing safety.

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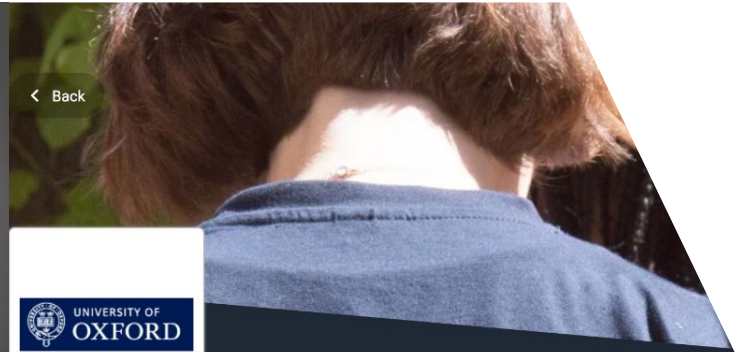

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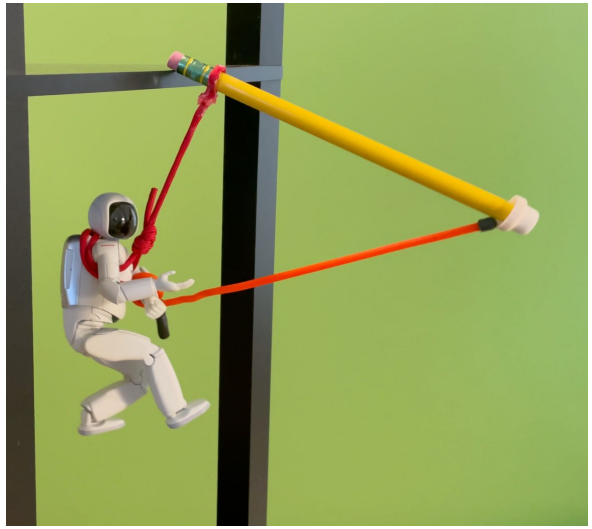

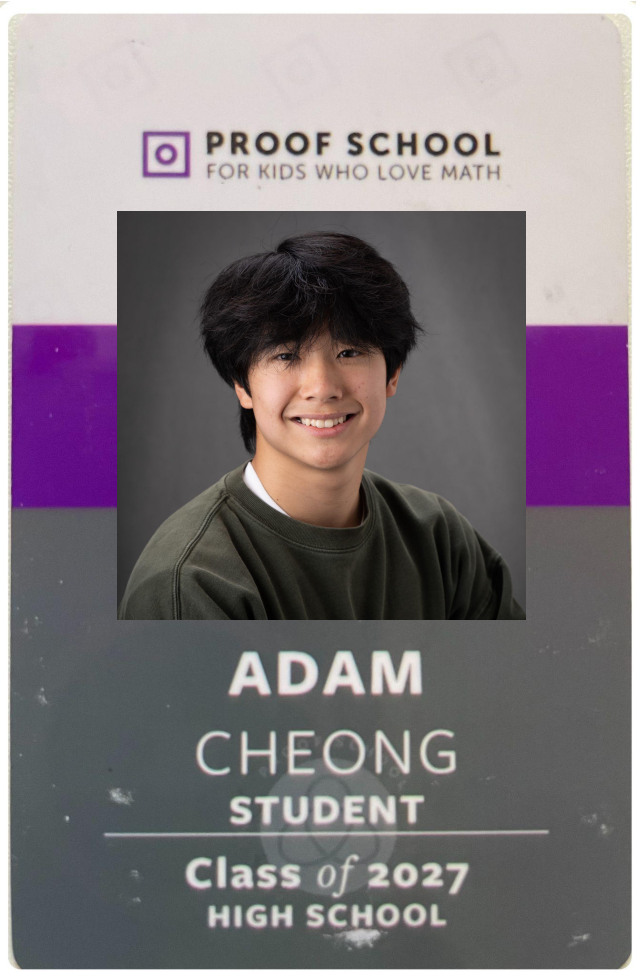
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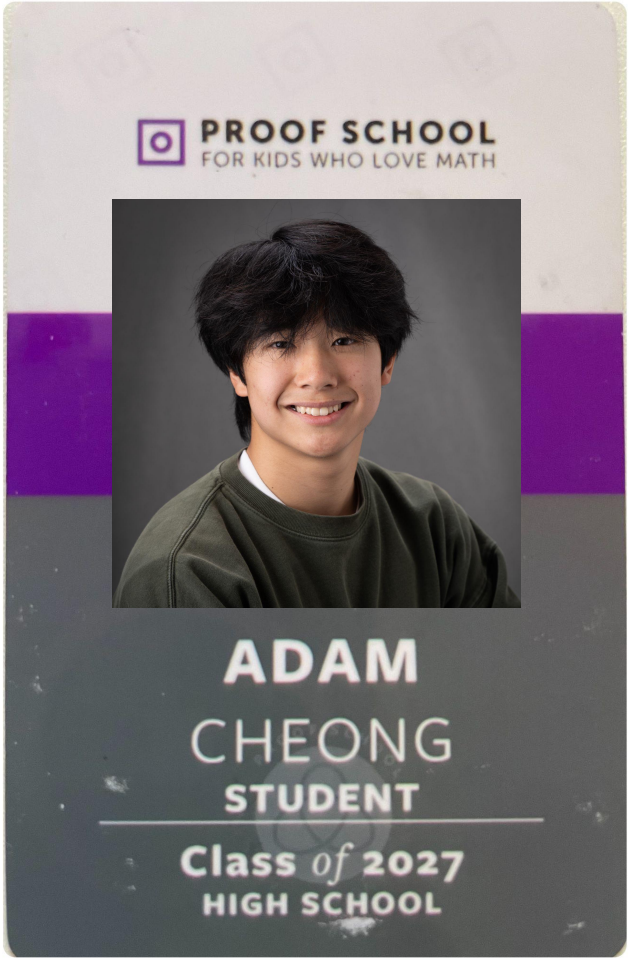
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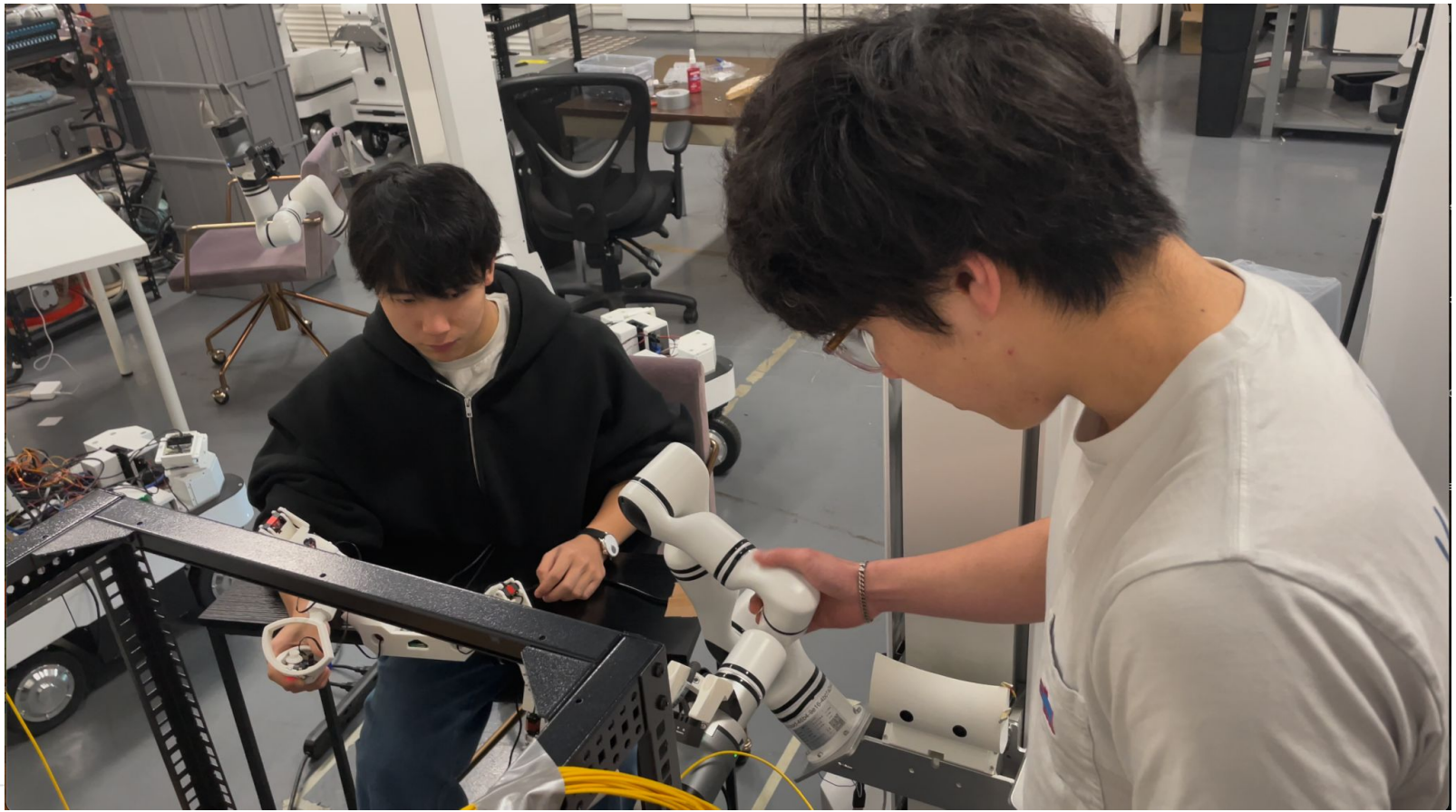

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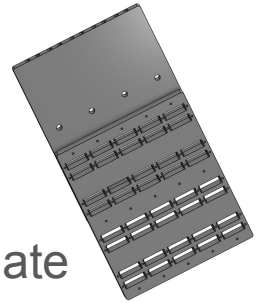

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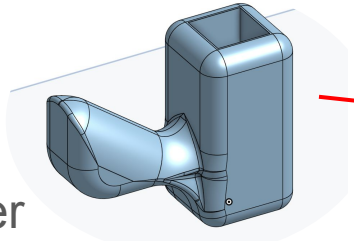

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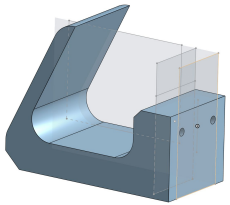

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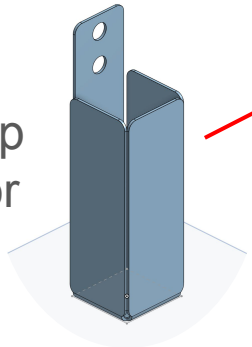

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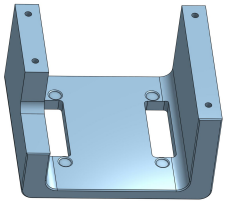
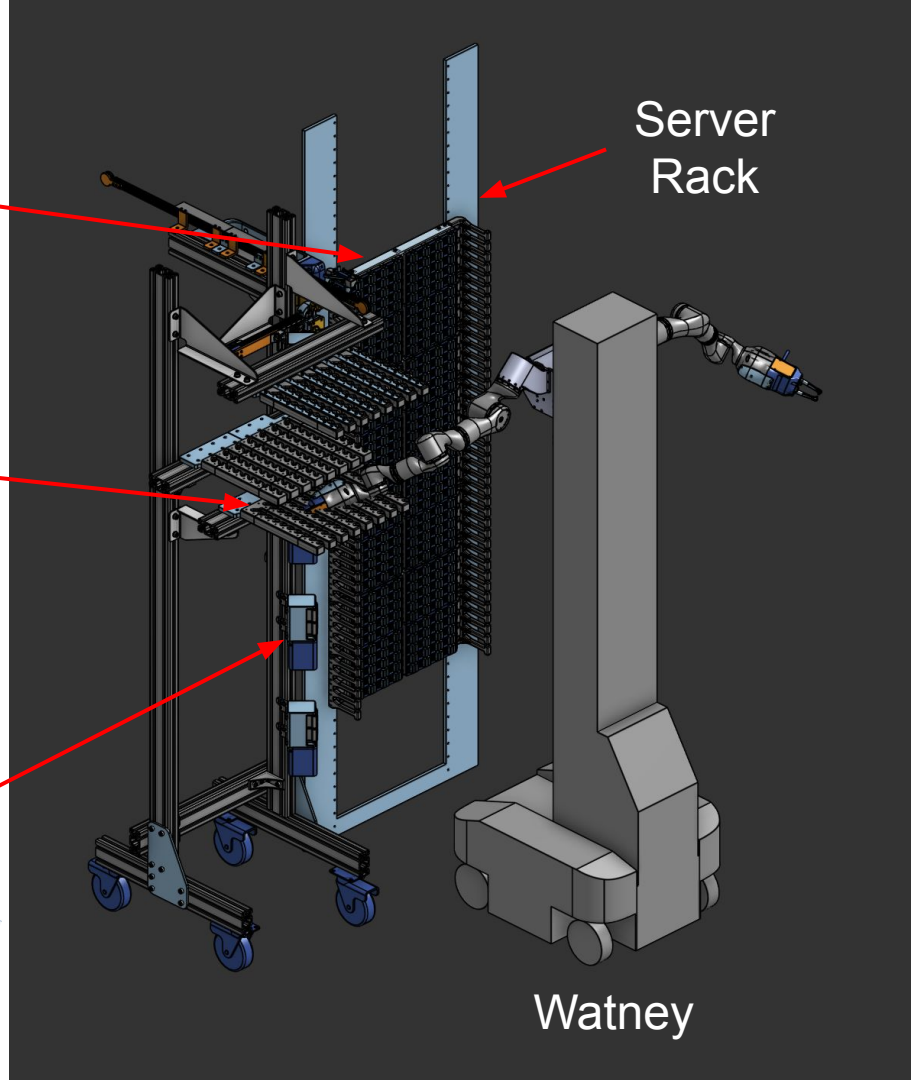
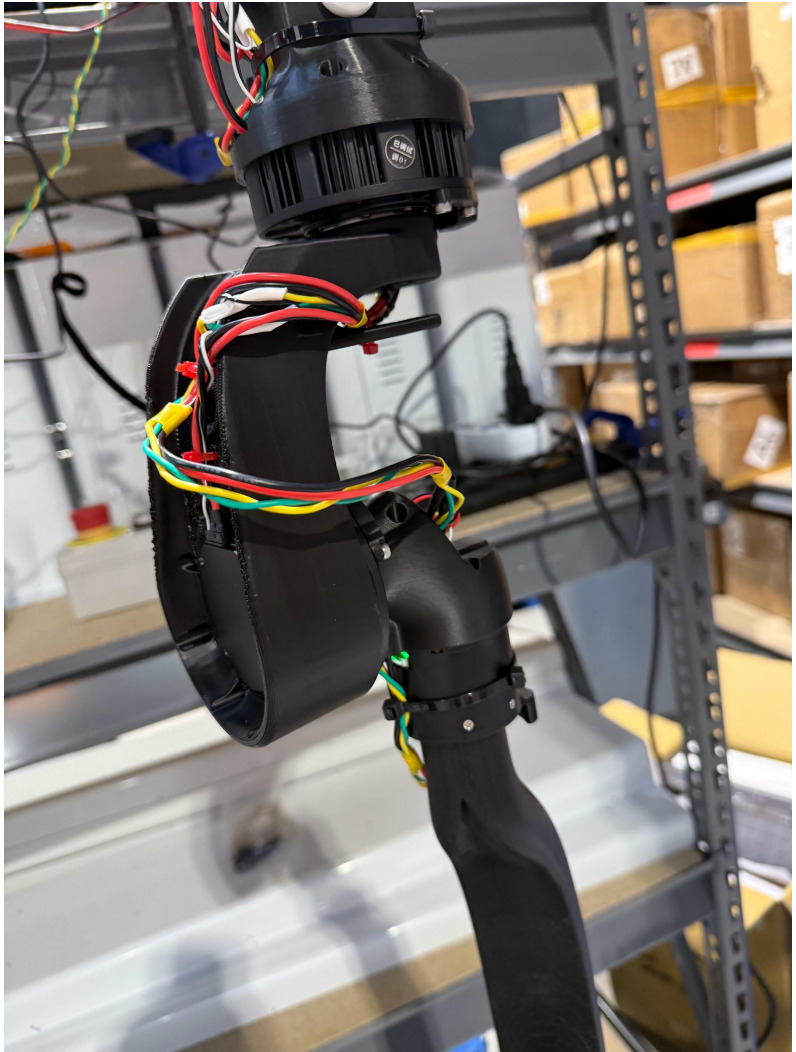
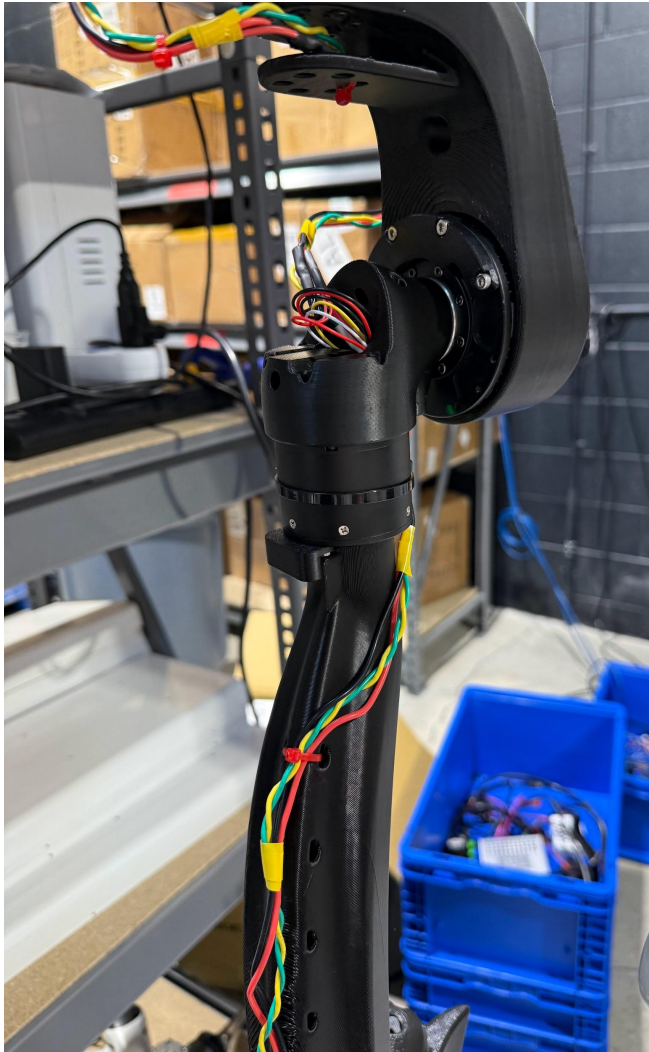
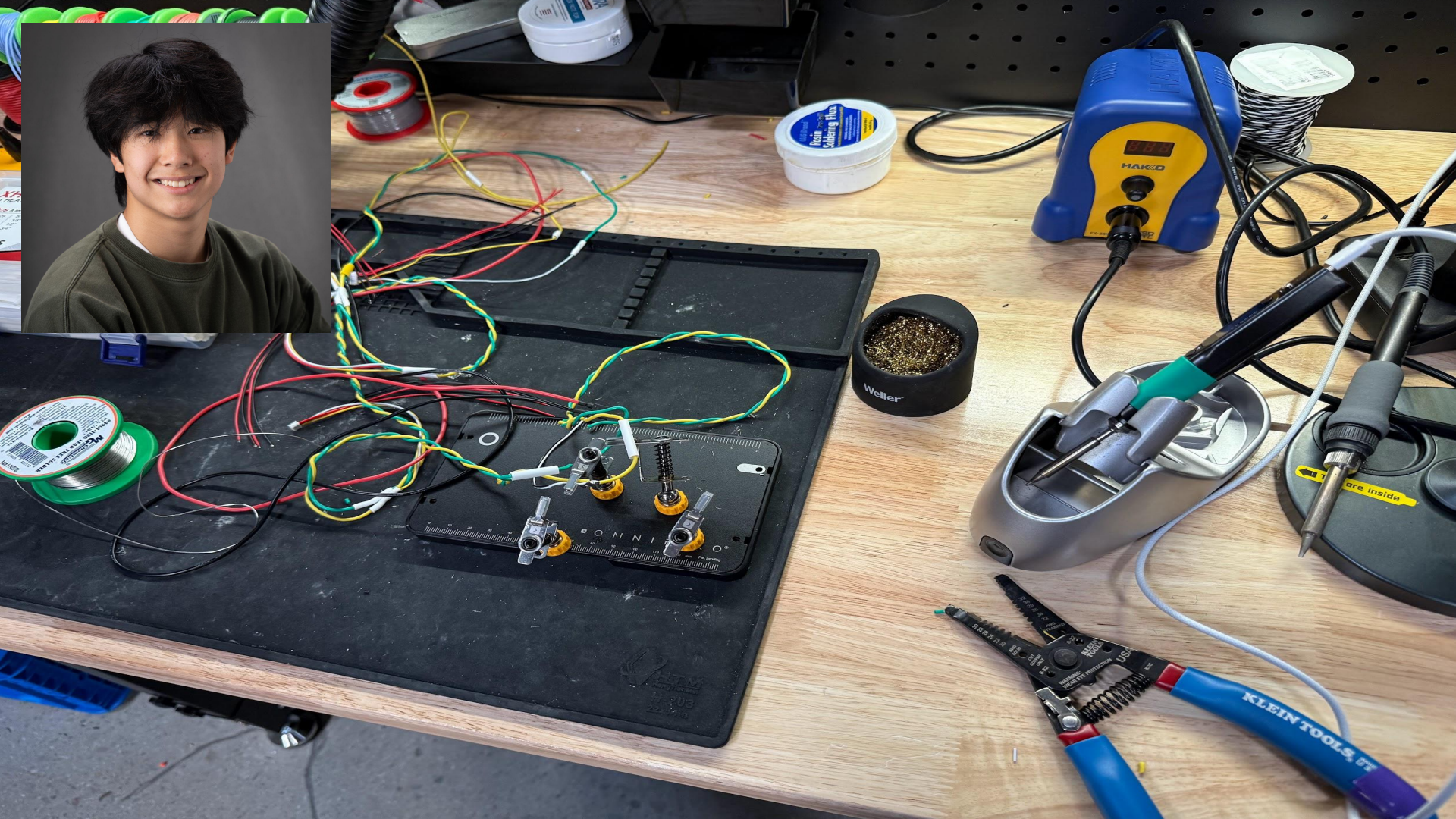
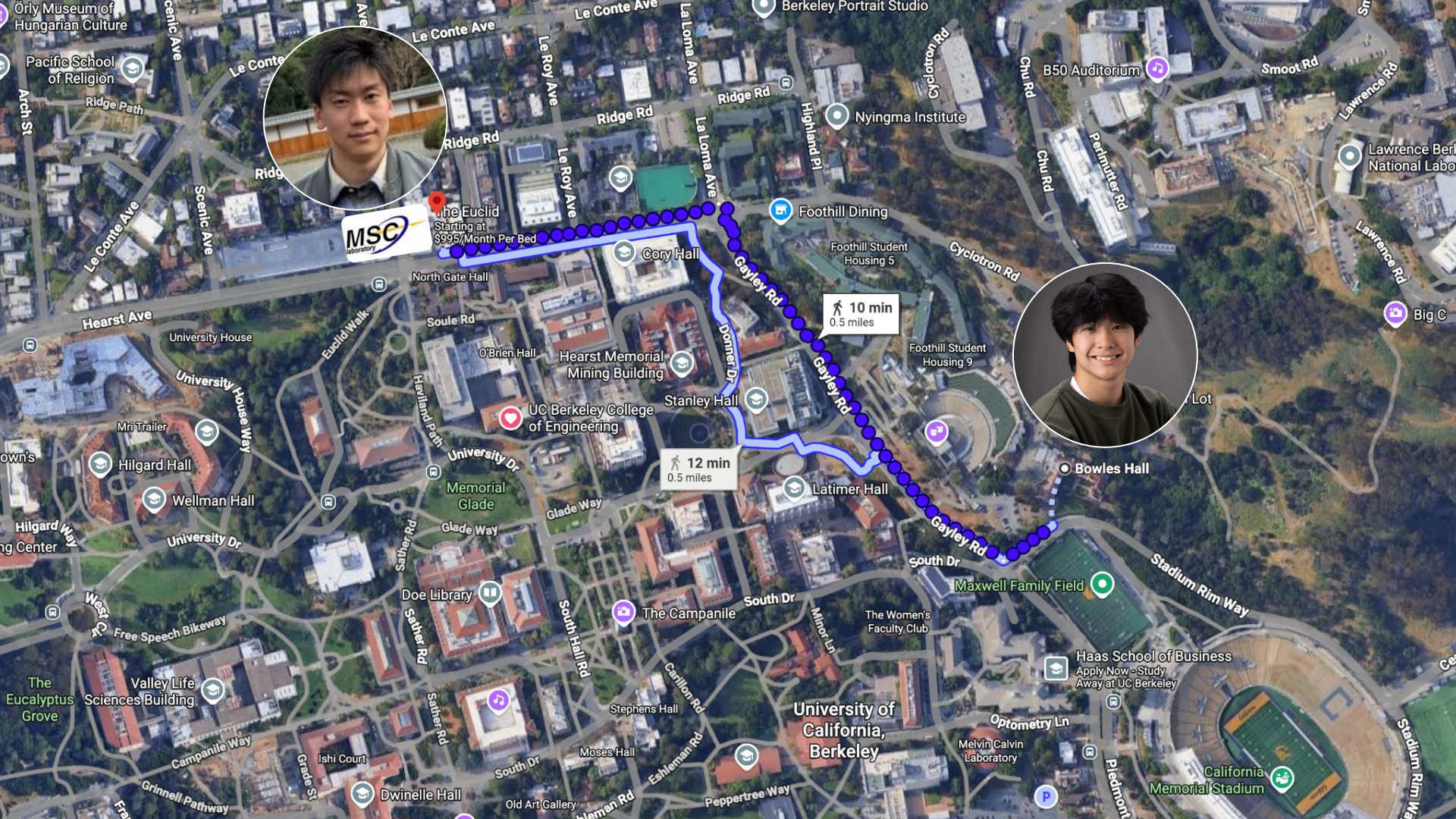









HAKKO
Weller
KLEIN TOOLS


Örly Museum of Hungarian Culture
Berkeley Portrait Studio
Pacific School of Religion
Ridge Path
Le Conte Ave
Scenic Ave
Arch St
Le Roy Ave
Ridge Rd
La Loma Ave
Highland Pl
Nyingma Institute
Cyclotron Rd
Chu Rd
B50 Auditorium
Smoot Rd
Lawrence Rd
Perimutter Rd
Lawrence Berkeley National Labo
MSC laboratory
The Euclid
Starting at $995/Month Per Bed
Foothill Dining
Foothill Student Housing 5
Cory Hall
North Gate Hall
Gayley Rd
10 min
0.5 miles
Big C
Hearst Ave
University House
Euclid Walk
Soule Rd
O'Brien Hall
Hearst Memorial Mining Building
Donner Dr
Foothill Student Housing 9
Stanley Hall
UC Berkeley College of Engineering
University House Way
Haviland Path
University Dr
Mri Trailer
Hilgard Hall
Wellman Hall
12 min
0.5 miles
Bowles Hall
Latimer Hall
Memorial Glade
Glade Way
Hilgard Way
Sather Rd
South Dr
Maxwell Family Field
Stadium Rim Way
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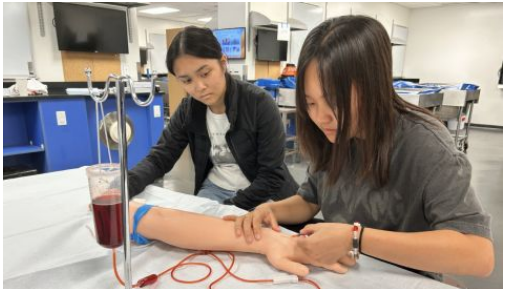
South Hall Rd
Minor Ln
University of California, Berkeley
Optometry Ln
Melvin Calvin Laboratory
Campanile Way
Ishi Court
Grade St
Moses Hall
Eshleman Rd
Piedmont
California Memorial Stadium
Grinnell Pathway
Dwinelle Hall
Old Art Gallery
Peppertree Way

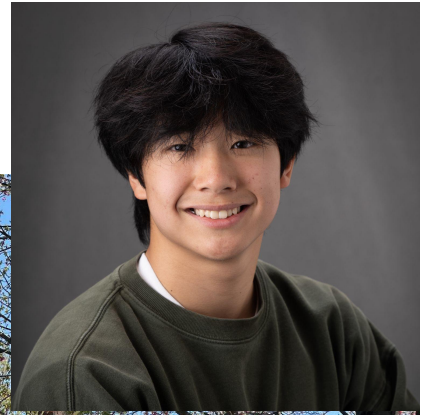


Stanford MEDICINE

Clinical Anatomy

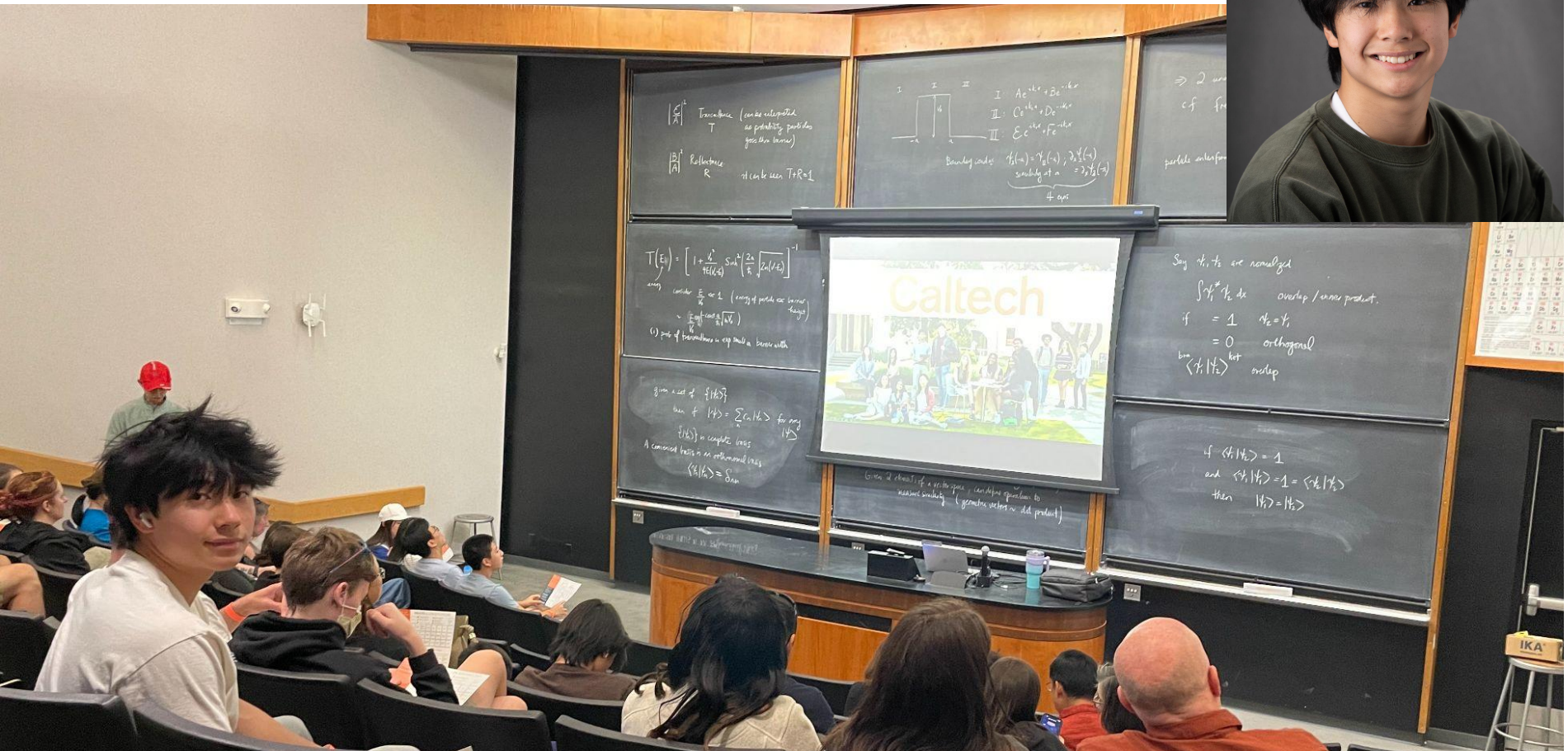
1.	Stanford University	Biomechanical Engineering	
2.	Massachusetts Institute of Technology	Mechanical Engineering	
3.	Berkeley UNIVERSITY OF CALIFORNIA	Mechanical Engineering + Bioengineering + [Business]	
4.	UNIVERSITY OF OXFORD	Engineering Science	EIT Internship
5.	REED COLLEGE	Physics + Bioengineering	Dual Degree Program
6.	Caltech		
7.	JOHNS HOPKINS UNIVERSITY	Biophysics	Top Program

JOHNS HOPKINS
UNIVERSITY



REED COLLEGE

Caltech



UNIVERSITY OF
OXFORD

Berkeley
UNIVERSITY OF CALIFORNIA

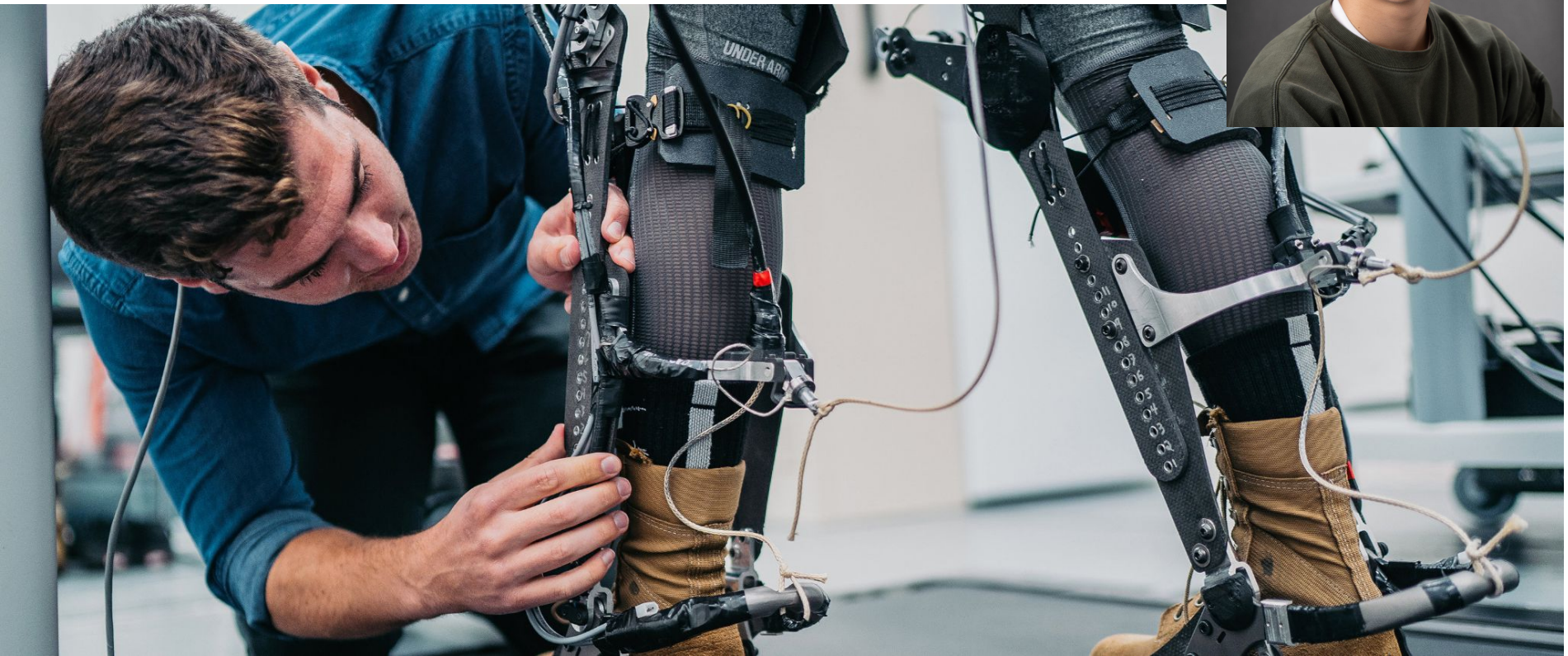
Berkeley
UNIVERSITY OF CALIFORNIA

Berkeley
UNIVERSITY OF CALIFORNIA

Massachusetts Institute of Technology

MITMECHE

Stanford University | Biomechatronics Laboratory



【2023~2027】

1.	Experience「経験」	Technical Skills「技術力」
A.	**Watney Robotics** (Silicon Valley Startup)	Robot Arm Controller Assembly, Teleoperation, ML Models Training, PCB Soldering, CAD Design, Prototyping
B.	**Tumoral Evolution** (Mathematical Oncology)	Cancer Population Dynamics, Press-Pulse, Mass Extinction, Dose Optimization, Gompertzian Growth, Fick's Laws
C.	**Monte Carlo Simulation** (Computational Biology)	OpenWorm, Sibernetic, *C. Elegans*, Monte Carlo Method, Decussation, Somatic Twist, Evolutionary Biology

2.	Talented「異能」Outstanding「優れている」
a.	2 International Posters: 25th IWM & PEQG 2026
b.	5 National Contest Awards:
c.	8 STEM Fair Prizes:
d.	SAT Score: 1550 (760 EBRW, 790 Math)
e.	WISC-V: Extremely High (Full Scale IQ: 138)

3.	Wish to Achieve「これから成し遂げたいこと」
a.	18 Math Courses on High School Transcript (Jun 2027)
b.	Participate in iGEM Jamboree in Paris (Nov 2026)
c.	Research Paper for Regeneron STS (Jun~Oct 2026)
d.	12 Credits @ Berkeley: 3 CS/Data Courses (Aug 2026)
e.	8 AP Exams: 4 Science, 2 Math, 2 Econ (Jun 2026)

Generation 9

ADAM CHEONG

BORN IN 2009

READ MORE

#robotics
#engineering
#mathematics