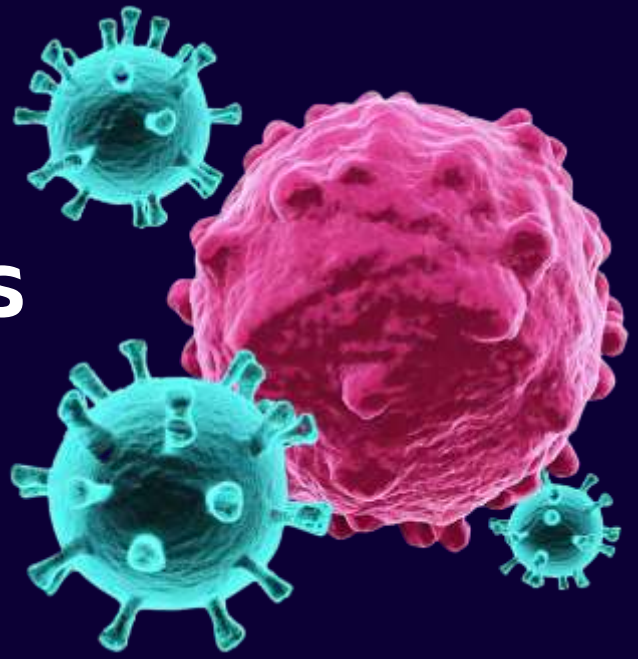
DeepOrigin

Simulating Life Processes Across Scales



Garegin Papoian

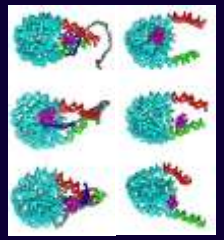
DeepOrigin's Simulation Stack

We build atomistic and coarse-grain models across **biological scales** to address every stage of drug discovery and find drugs faster.

Target Discovery

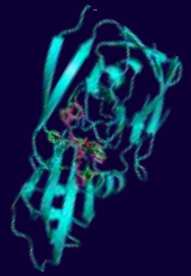
Dynamic Cell Models

Target Characterization



Structural Ensembles

Hit Discovery

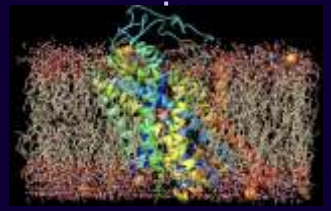


Virtual Screening

Hit Prioritization

Docking & Cheminformatics

Lead Optimization



Molecular Dynamics

Key:

- Available Now
- In Development

Outline

- Mesoscale modeling of the cytoskeleton and towards simulating eukaryotic cells
- Modeling protein complexes: For some important drug discovery tasks, AWSEM leaves AlphaFold2 in the dust
- Virtual Screening of Small Molecules

MEDYAN Work

- James Komianos

- Aravind Chandrasekaran

- Qin Ni

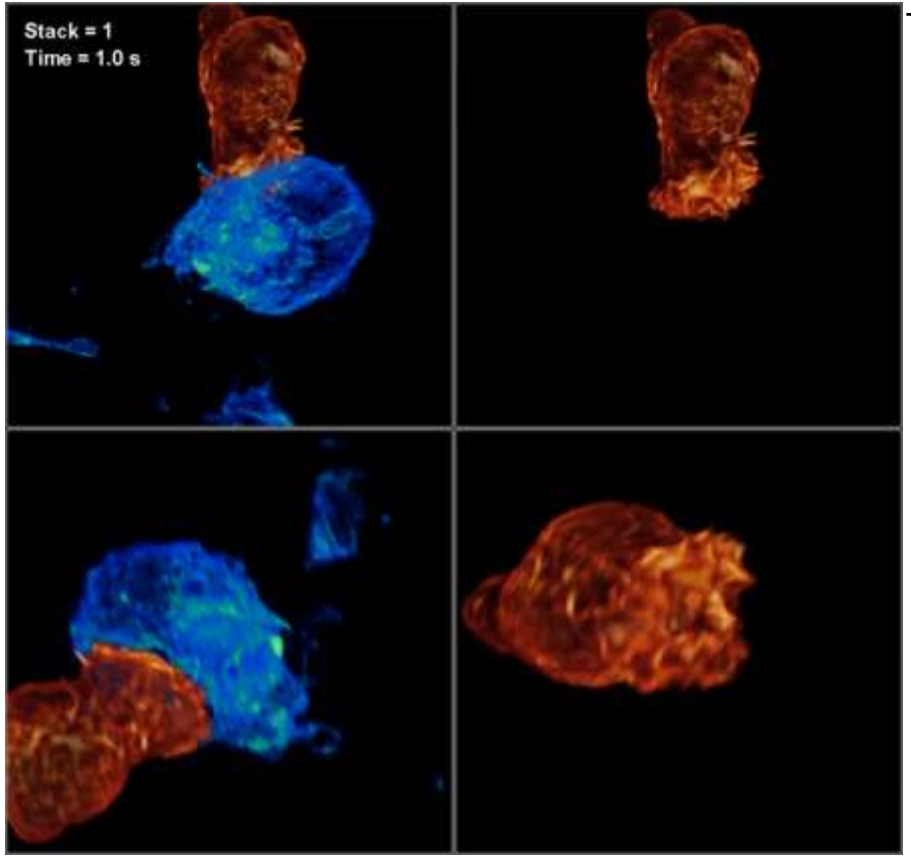
NSF CHEMISTRY: CTMC
NSF PHYSICS: POLS

- Haoran Ni

- Carlos Floyd

- Radek Erban

- Arpita Upadhyaya



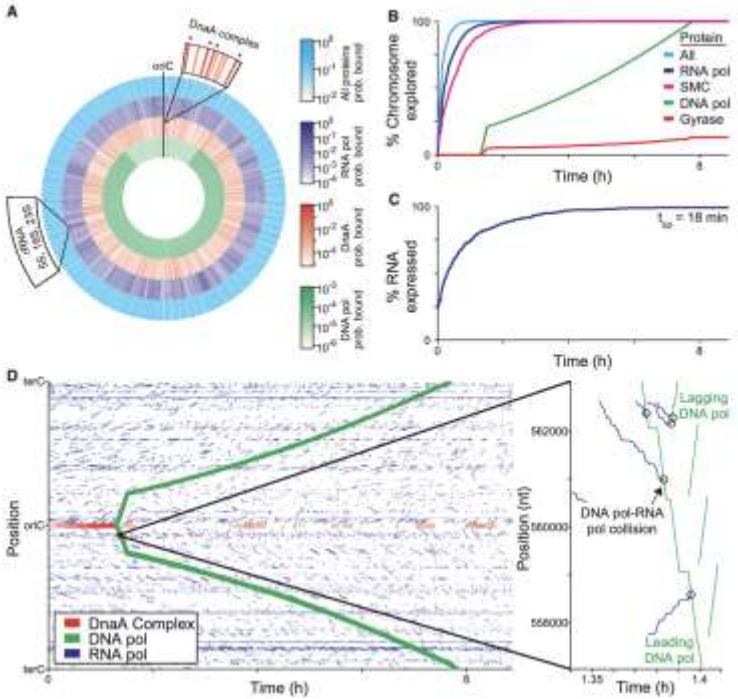
T-cell activation
Eric Betzig

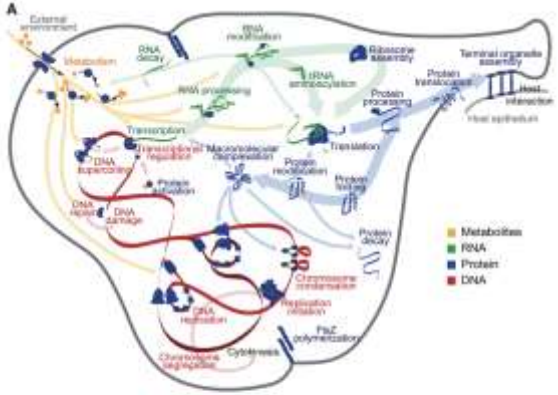
We would like to simulate cellular dynamics based on the microscopic laws of physics and chemistry

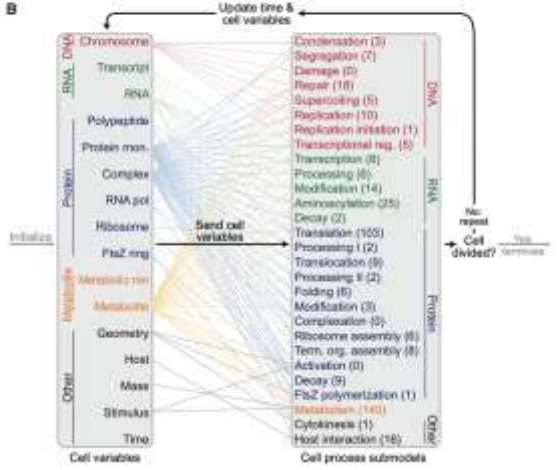
Cell

A Whole-Cell Computational Model Predicts Phenotype from Genotype

Jonathan R. Karr,[1,4] Jayodita C. Sanghvi,[2,4] Derek N. Macklin,[2] Miriam V. Gutschow,[2] Jared M. Benjamin Bolival, Jr.,[2] Nacyra Assad-Garcia,[3] John I. Glass,[3] and Markus W. Covert[2,*]
[1]Graduate Program in Biophysics







Dendritic nucleation/Array treadmilling model

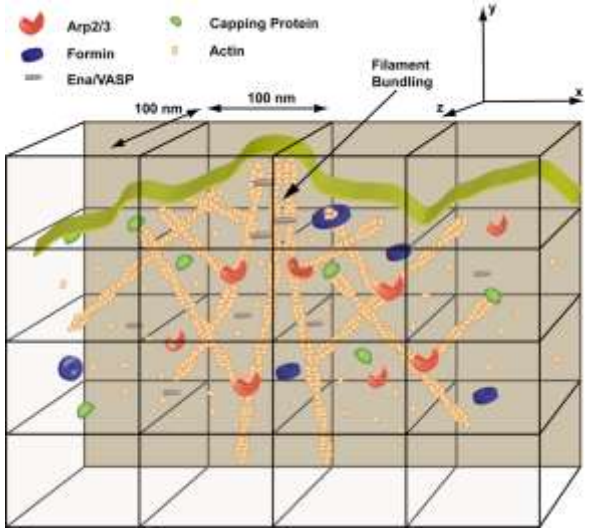
Regulation of force generation is complicated!

We would like to model all this!

- Pollard and Borisy, Cell 112, 453 (2003).
- T.D Pollard, L Blanchoin, R.D Mullins Annu. Rev. Biophys. Biomol. Struct., 545–576 (2000)

MEDYAN: Mechanochemical Dynamics of Active Networks

- 3D simulation region is divided into compartments.
- Diffusion (Actin, Capping protein, Arp2/3) between compartments.
- Chemical reactions in compartments:
  - Polymerization, Depolymerization, Capping, Branching...
- Monte Carlo algorithm to generate stochastic trajectories

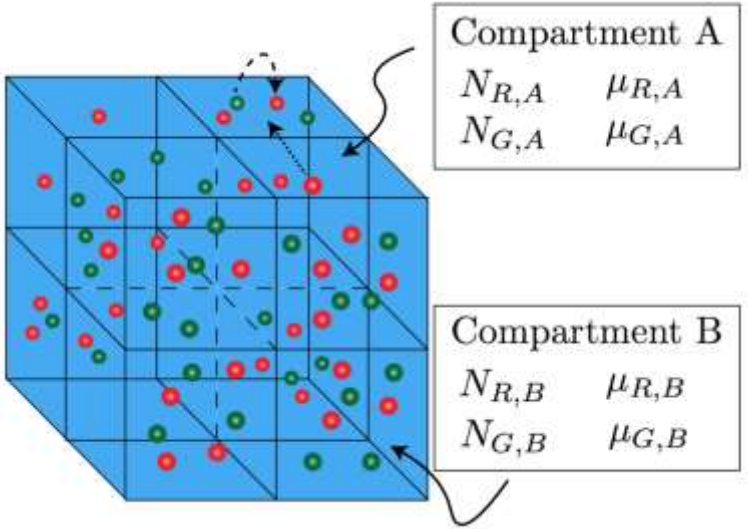


K. Popov, J. Komianos, G. A. Papoian, PLOS Comp Bio, *2016*, DOI:10.1371/journal.pcbi.1004877

L. Hu and G. A. Papoian, Biophys. J.; *2010*, 98,1375

L. Hu and G. A. Papoian, J. Phys.: Condens. Matter; *2011*, 23, 374101

The reaction-diffusion master equation

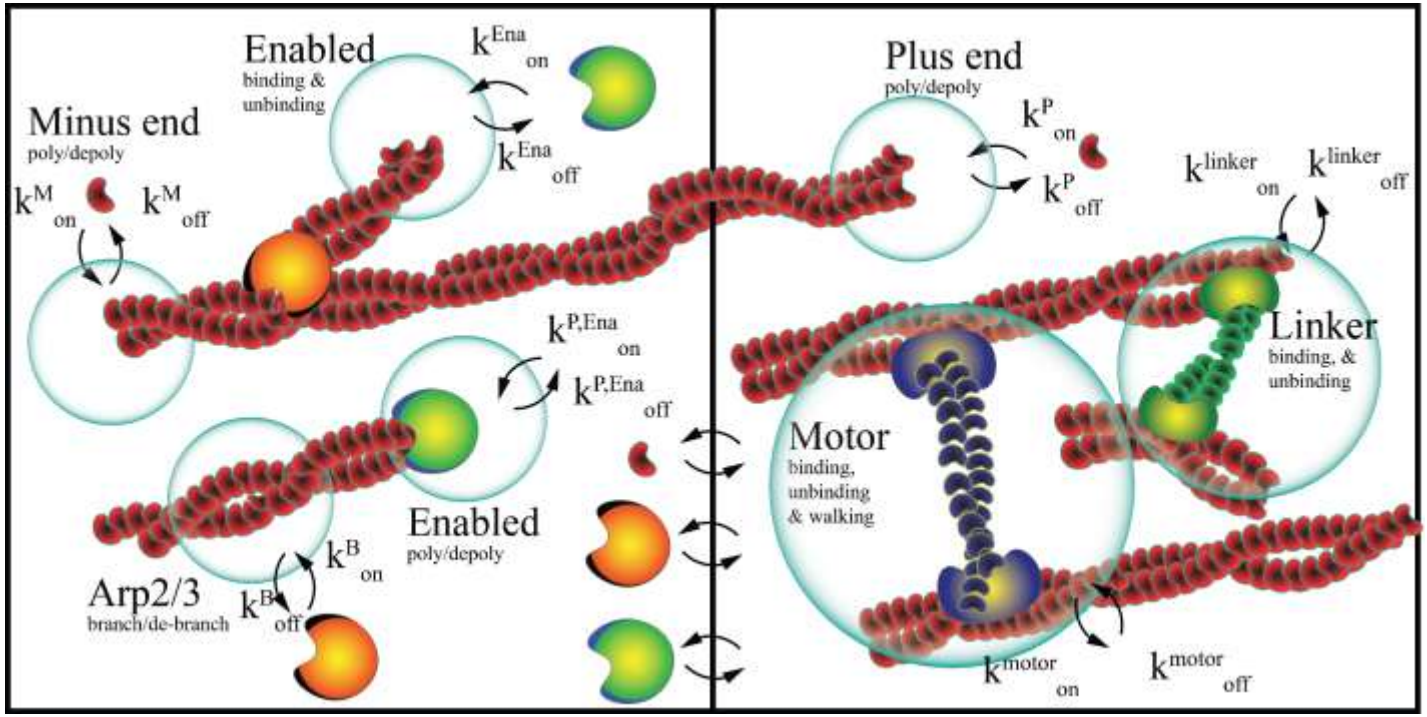


$$\frac{dP(\mathbf{N},t)}{dt} = \left(\hat{M} + \hat{D}\right) P(\mathbf{N},t)$$

$$\mathbf{N} = \{N_{i,A}\}_{i \in S, A \in E}$$

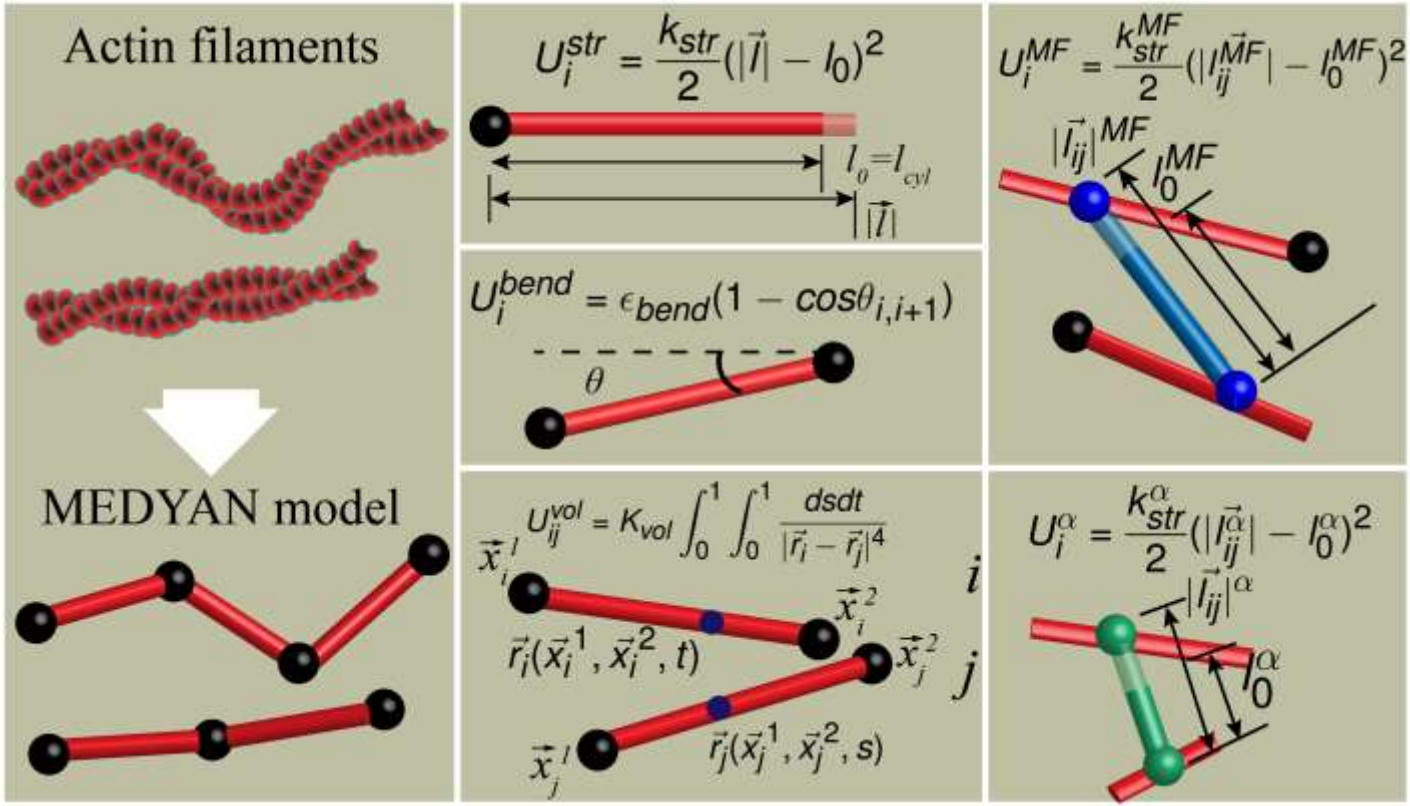
- **Discretize space into locally well-mixed compartments (mass-action kinetics)**
- **Includes both chemical reactions within compartments and diffusion between compartments**
- **Simulated using accelerated Gillespie algorithm variant (NRM)**

Spatially resolved chemistry



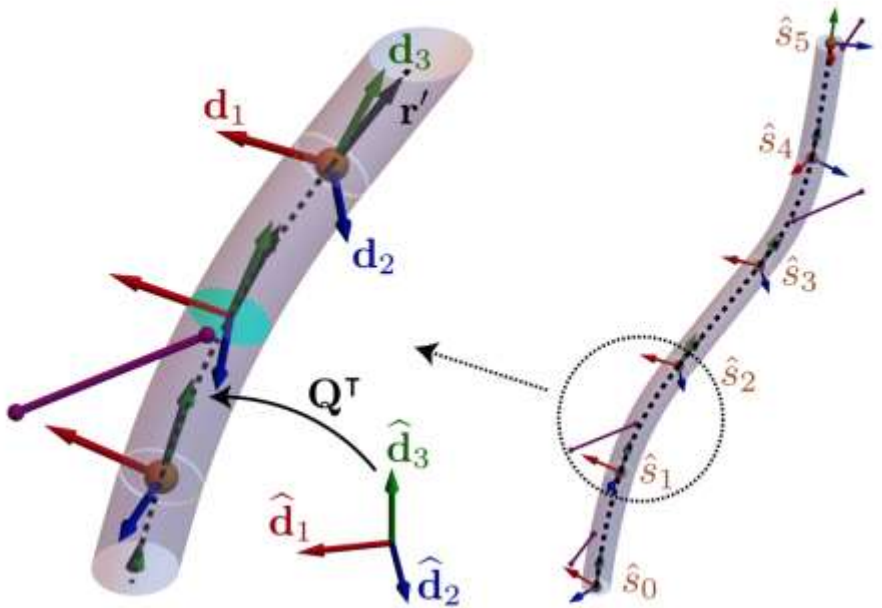
K. Popov, J. Komianos, G. A. Papoian, PLOS Comp Bio, 2016, DOI:10.1371/journal.pcbi.1004877

MEDYAN: Mechanics



Finite-width Filament Model

- Designed a filament model that includes **shearing, twisting, stretching, and bending**
- Built on the **Cosserat theory** of elastic rods
- **Spline functions** used to parameterize rod configuration



C. Floyd, H. Ni, R. Gunaratne, R. Erban, G. A. Papoian, "On Stretching, Bending, Shearing and Twisting of Actin Filaments I: Variational Models", J Chem Theor Comp, (2022), 18, 4865

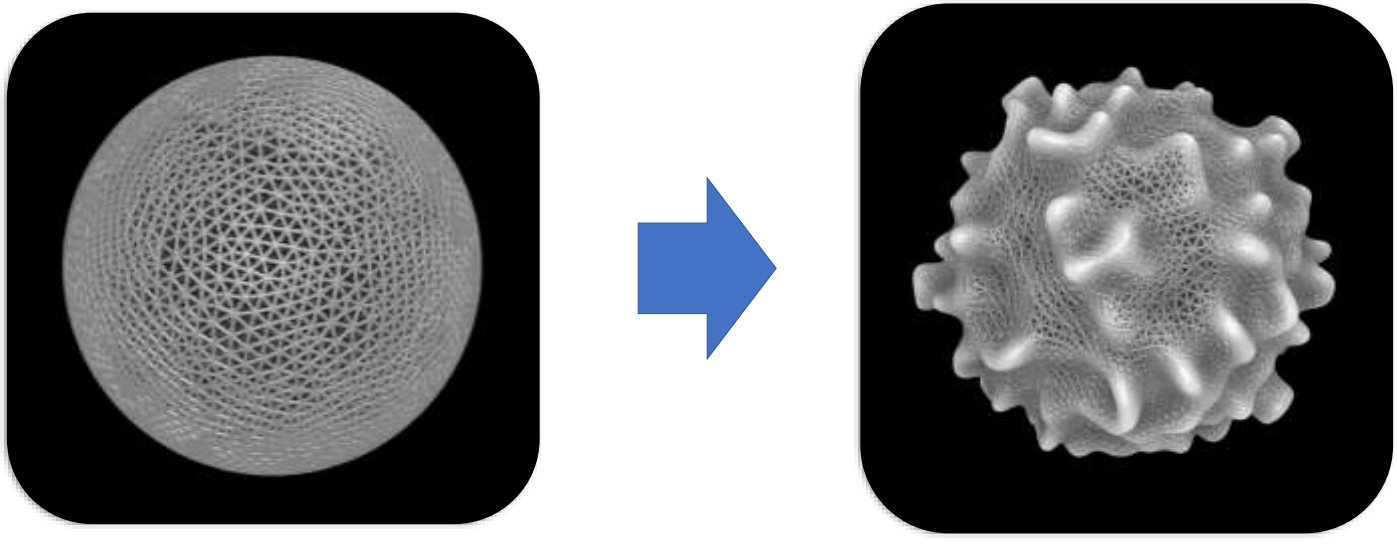

Carlos Floyd


Haoran Ni


Ravinda Gunaratne


Radek Erban

Membrane crumpling in hyperosmotic solution



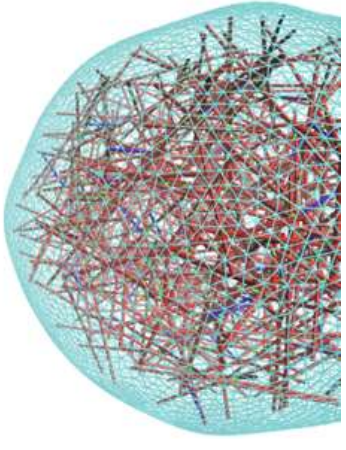
Tension: $F = k_s/(2A_0)(A - A_0)^2$

Bending: $F = \int 2k_b(H - c_0)^2 dA$

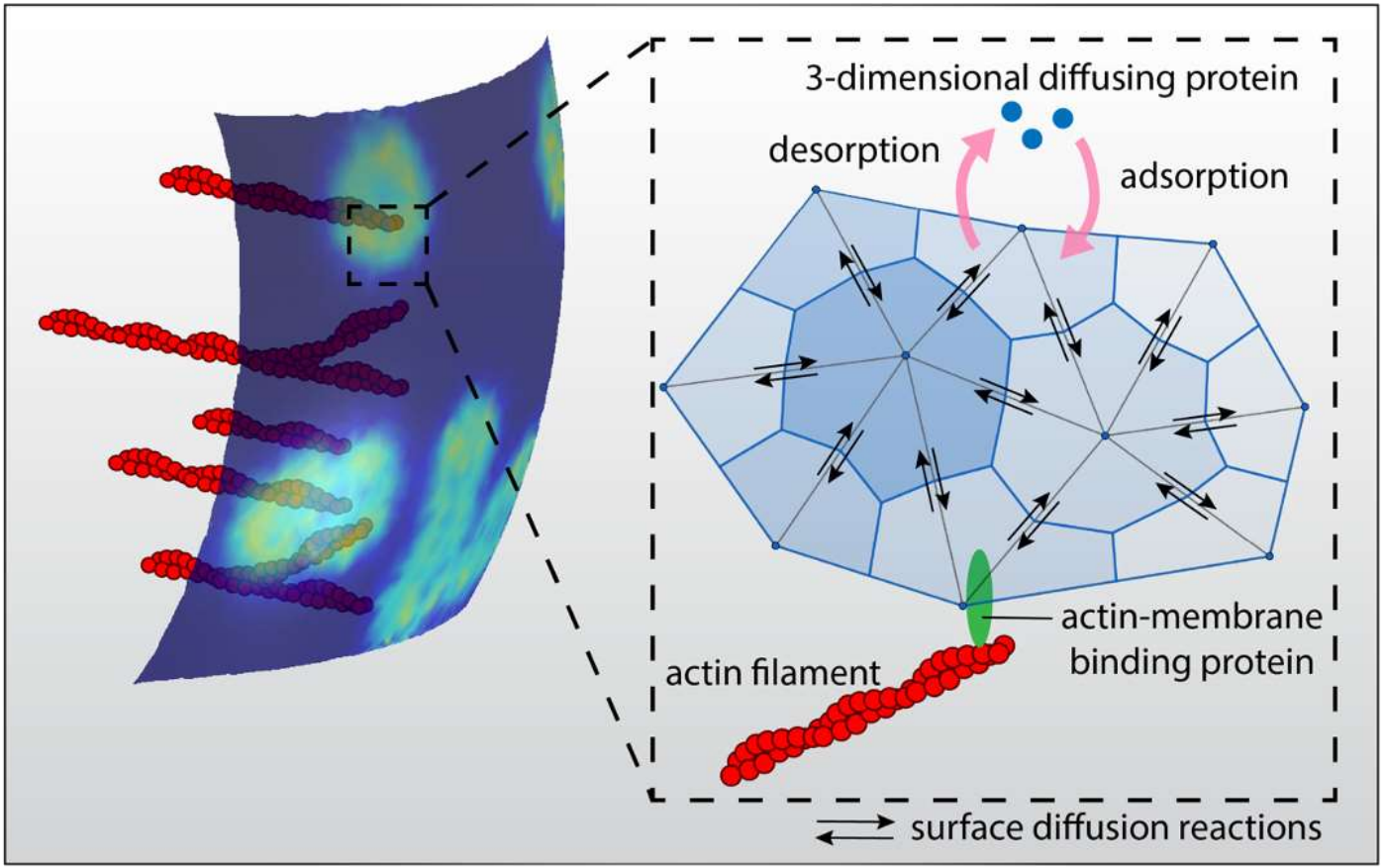
Volume Conservation: $F = k_V/(2V_0)(V - V_0)^2$

Volume Exclusion: $F = k_{vol} \int dA/|r_b - r_p|^4$

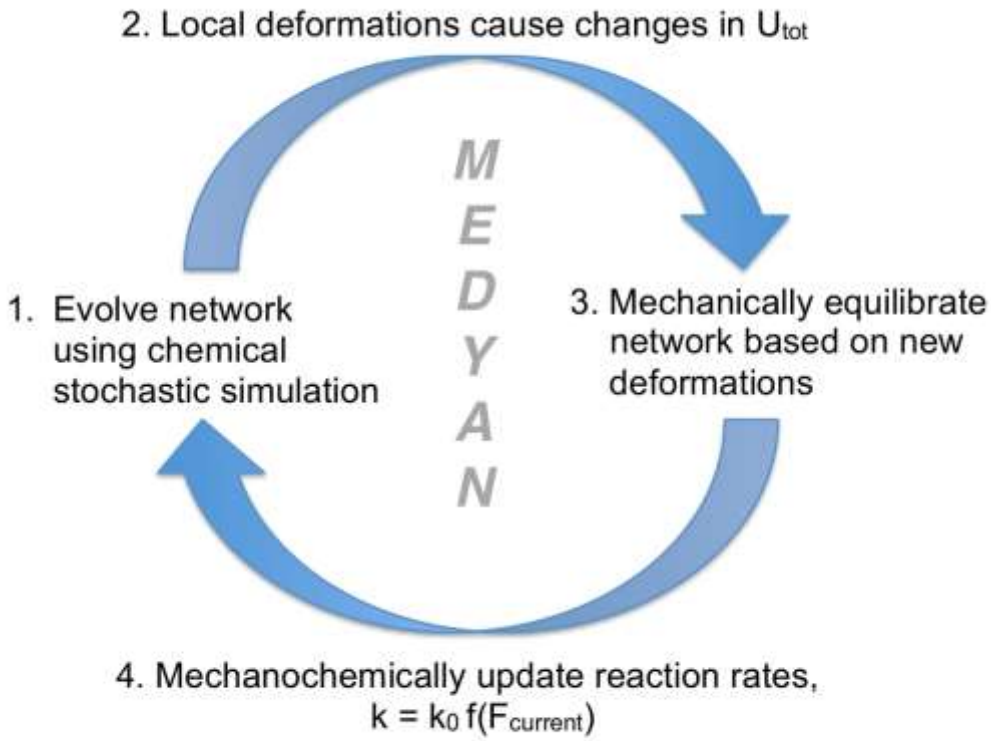
- Haoran Ni



Surface Reaction-Diffusion: Receptor Signaling & Clustering



Time Evolution: An Adiabatic Ansatz



Popov K, Komianos J, and Papoian GA, PLoS Comp. Biol. 2016

Timings on 1 core of 1 CPU

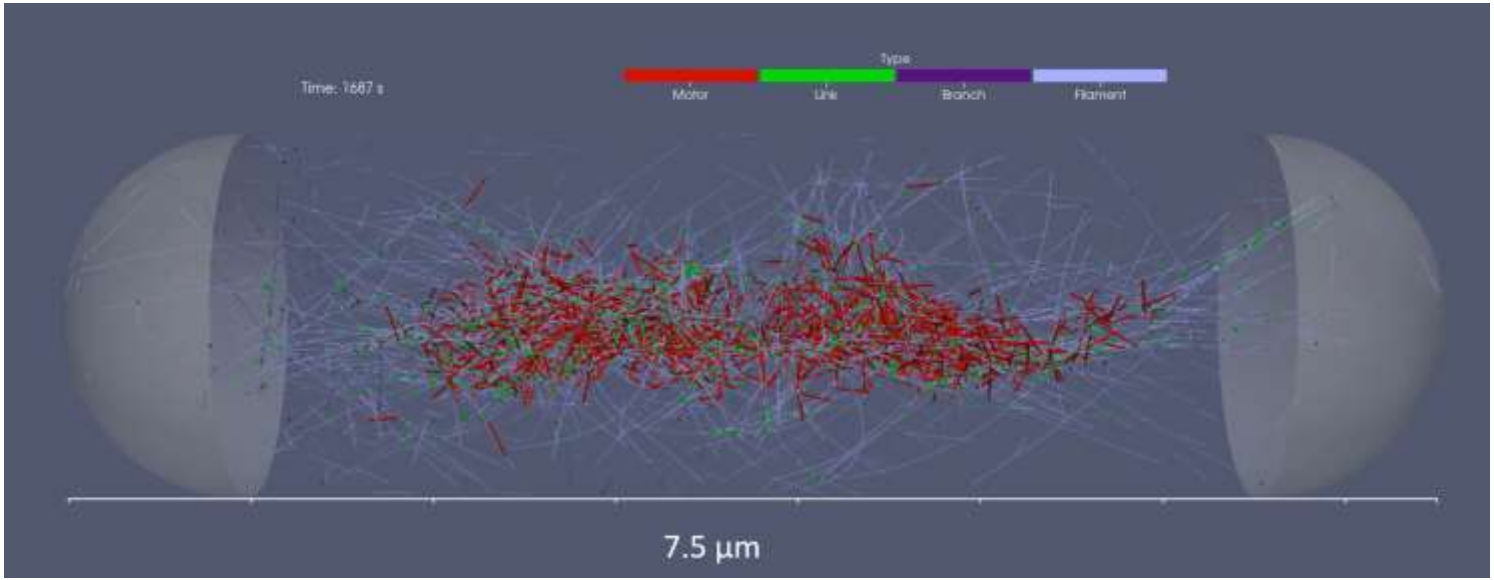
Wall times required to obtain **1000 seconds** long trajectories

Actin 20 muM a:A 0.01 M:A 0.05	# of actin monomers	# of polymer segments	MEDYAN 3.2	MEDYAN 4.0	MEDYAN 5.1	MEDYAN Julia
V = 1 μm^3	**12,000**	**300**	**12.5h**	**2.5h**		
V = 8 μm^3	**96,000**	**2,400**	**8d**	**1.5d**		
V = 27 μm^3	**270,000**				**12d**	**22h**
V = 125 μm^3	**1,500,000**	**38,000**	**360d**	**27d**		

Nathan Zimmerberg

Mouse embryonic fibroblasts have a volume of approximately 1000 μm^3

Axon Growth Cone Simulation



- 279283 actin monomers.
- The initial conditions and parameters for these simulations were ported from the 5 nM Arp2/3 simulations from:
  - Aravind Chandrasekaran et al., MBoC 33.11 (2022)
- A MEDYAN.jl simulation takes 3 days to run on a single core with 4 GB memory.
- Previously took multiple weeks using C++ version.

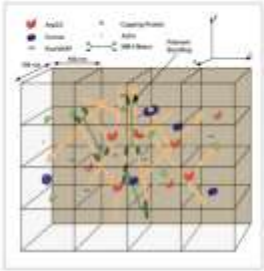
http://medyan.org

Home Documentation Download Publications Gallery Contact

MEDYAN - Mechanochemical Dynamics of Active Networks

Welcome to the webpage of the *MEDYAN*, an efficient and scalable computational model for mechanochemical simulations of active matter networks created by the Papoian lab at the University of Maryland. This webpage contains documentation and examples for the *MEDYAN* software package, which is implemented in C++. The source code for this package is downloadable for scientific use.

Background



The cell cytoskeleton plays a key role in human biology and disease, contributing ubiquitously to such important processes as embryonic development, wound repair and cancer metastasis. The Papoian laboratory is interested in gaining deeper understanding of the physical chemistry behind these complex, far-from-equilibrium mechanochemical processes.

Read more

Latest news

March 2022 - MEDYAN 5.4.0 is published. [Download]

March 2022 - MEDYAN 5.3.0 is published.

February 2022 - MEDYAN 5.2.1 is published.

December 2021 - MEDYAN 5.1.0 is published.

August 2021 - Our paper titled *Membrane-MEDYAN: Simulating Deformable Vesicles Containing Complex Cytoskeletal Networks* was published in JPCB and chosen as the cover.

July 2021 - MEDYAN 5.0.0 is published.

July 2021 - MEDYAN 4.3.0 is published.

July 2021 - MEDYAN 4.2.0 is published.

March 2021 - Our paper titled *Segmental Lennard-Jones interactions for semi-flexible polymer networks* was published in Molecular Physics.

Funding sources

Popov, Komianos, and Papoian, PLoS Comp. Biol. 2016

THE JOURNAL OF PHYSICAL CHEMISTRY B

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A JOURNAL OF THE AMERICAN CHEMICAL SOCIETY

September 30, 2021
Volume 125
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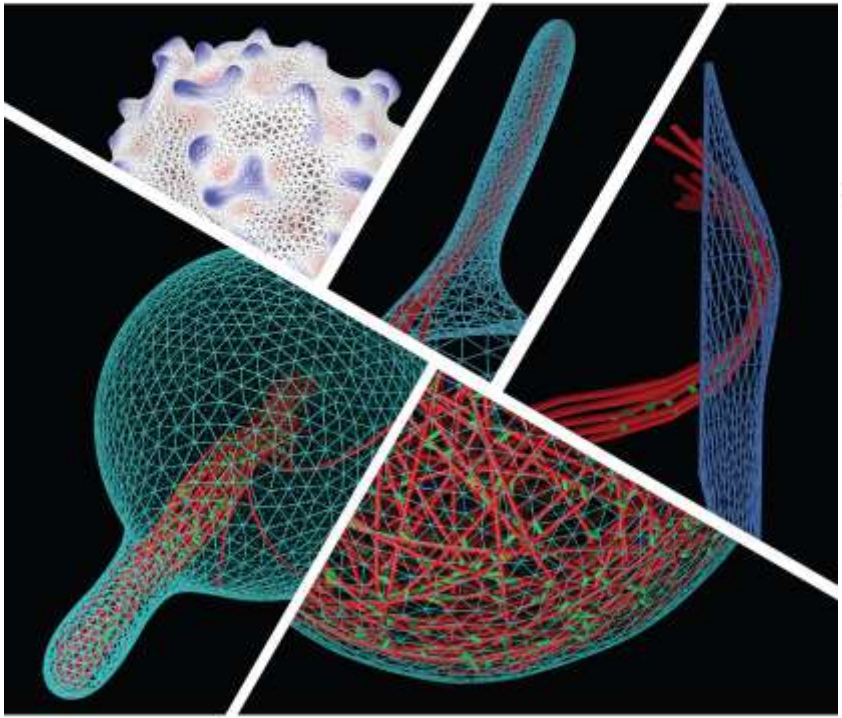

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THE JOURNAL OF PHYSICAL CHEMISTRY B

Membrane-MEDYAN: Simulating Deformable Vesicles Containing Complex Cytoskeletal Networks

Published as part of The Journal of Physical Chemistry virtual special issue "Dave Thirumalai Festschrift".

Haoran Ni and Garegin A. Papoian*

Cite This: J. Phys. Chem. B 2021, 125, 10118−10119

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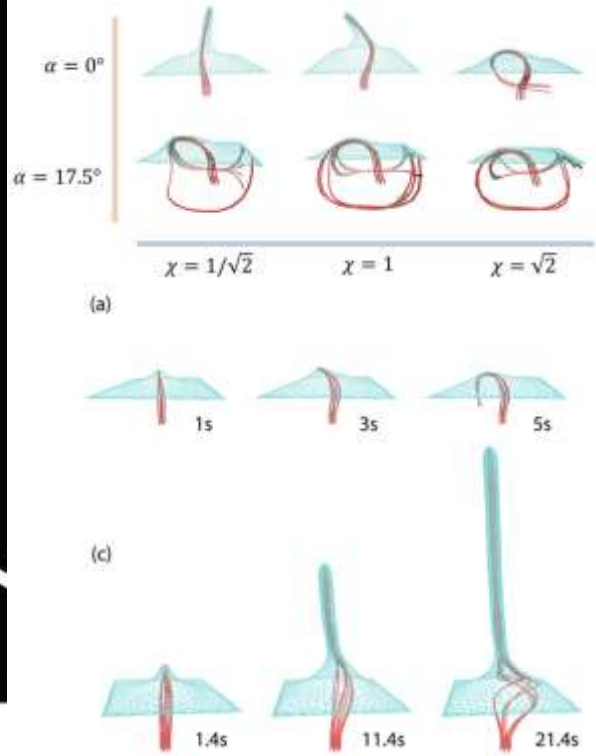
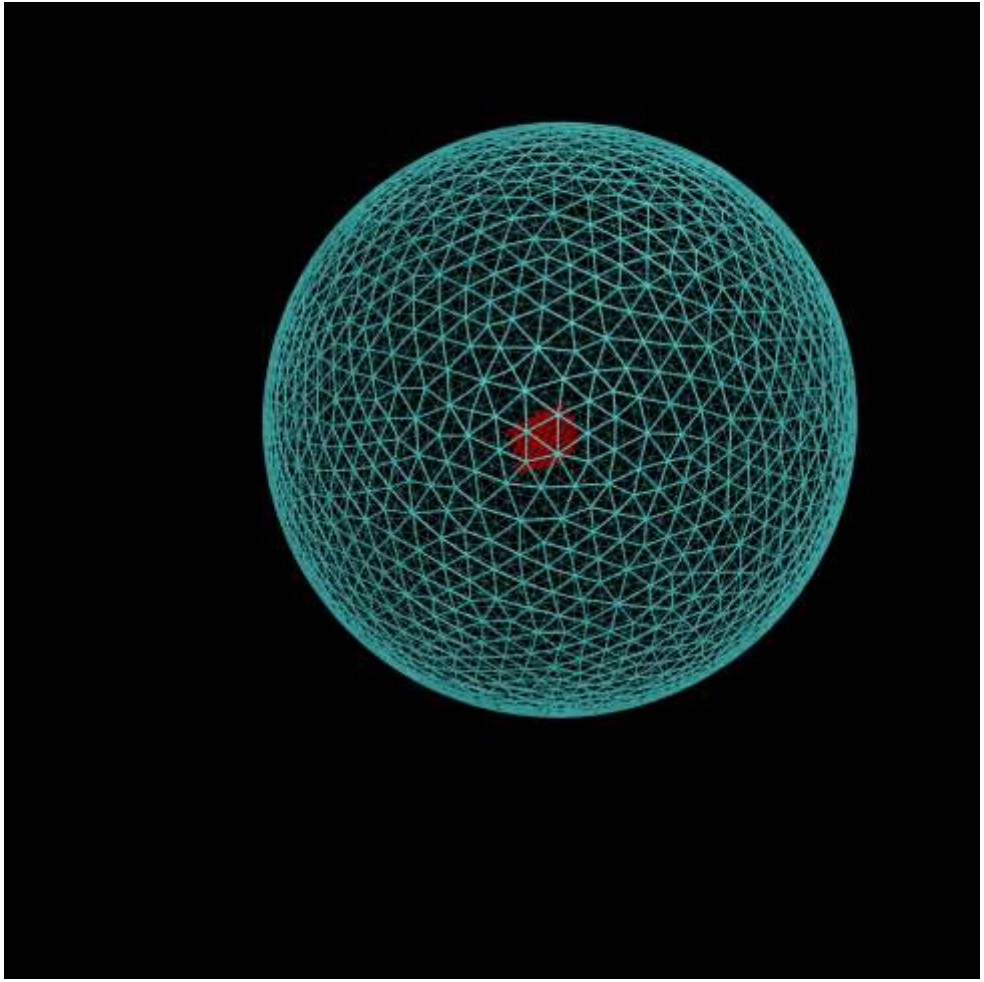
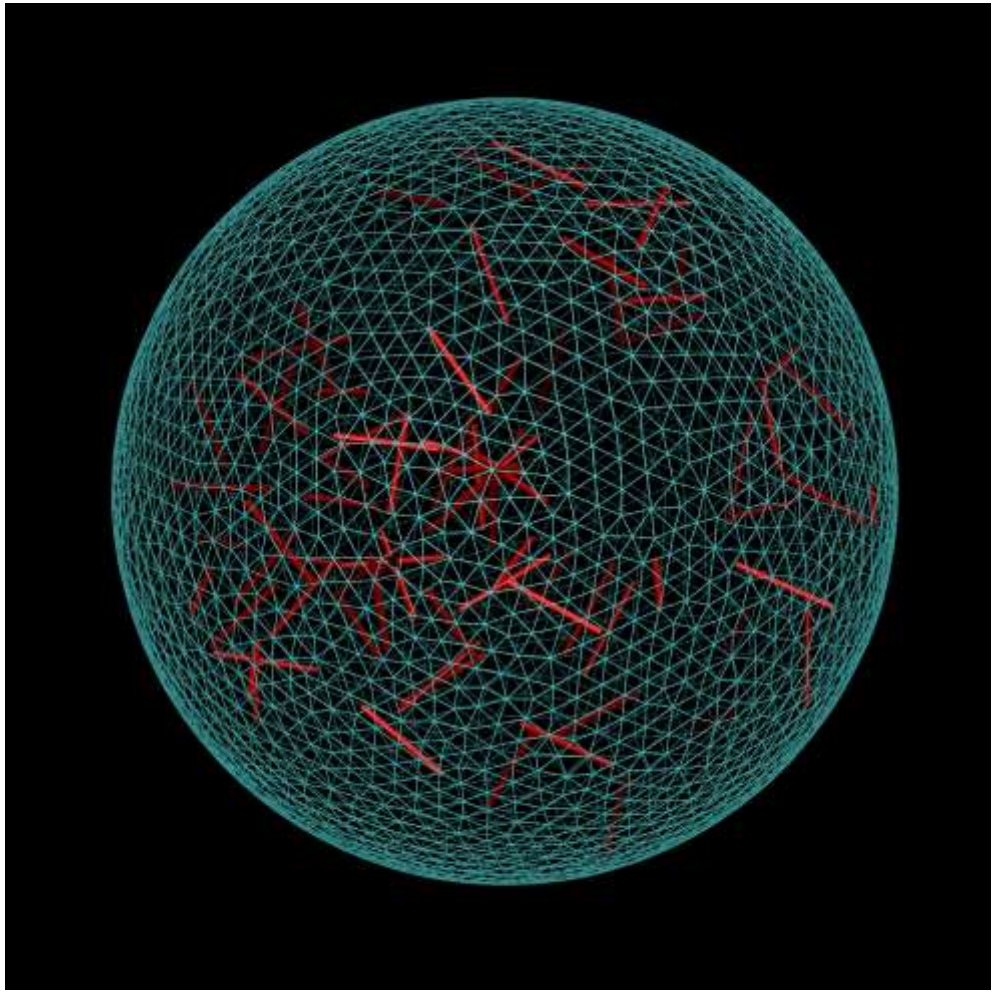


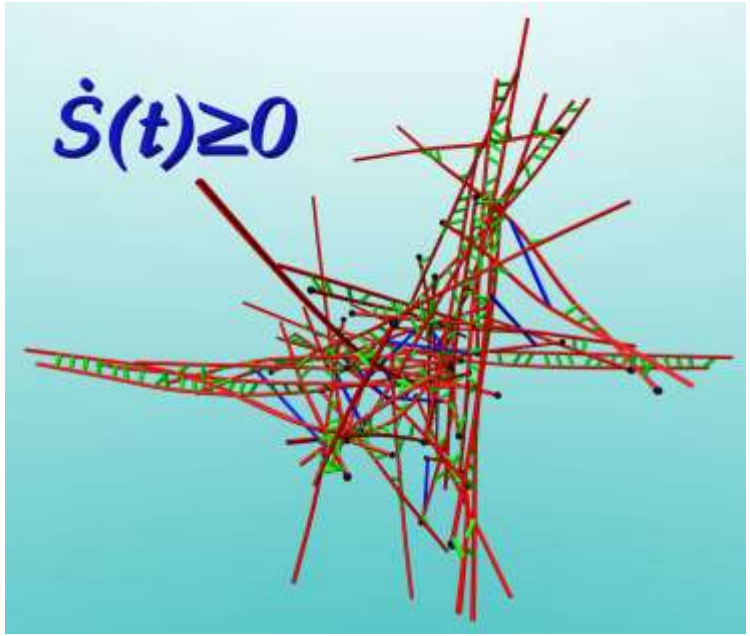
Towards Simulating a Whole Cell



Towards Simulating a Whole Cell

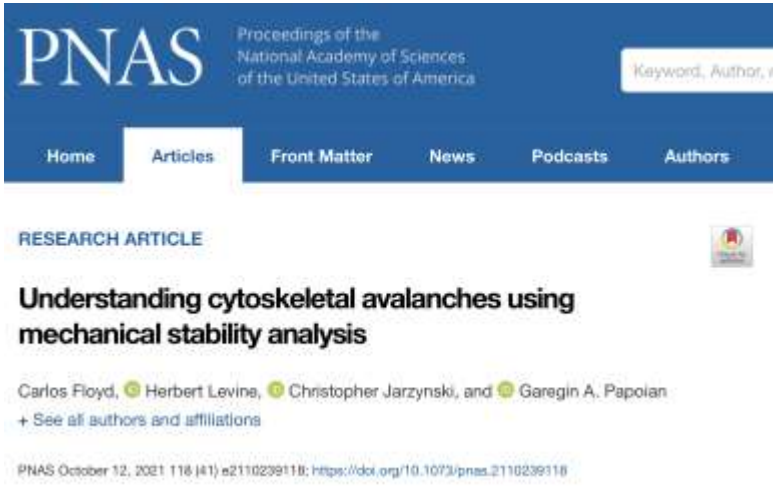


Entropy Production and Avalanches in Actomyosin Networks



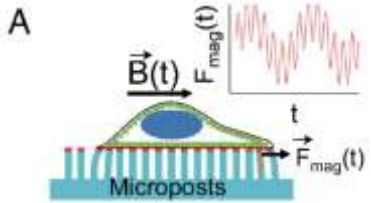
- **Measuring entropy production in active matter phases is a necessary step to understand their self-organization**
- **Experimental measurements of forces produced by migrating cells suggests that dissipation of mechanical energy during migration is poorly understood**
- **We developed an algorithm in MEDYAN to quantify dissipation rates**

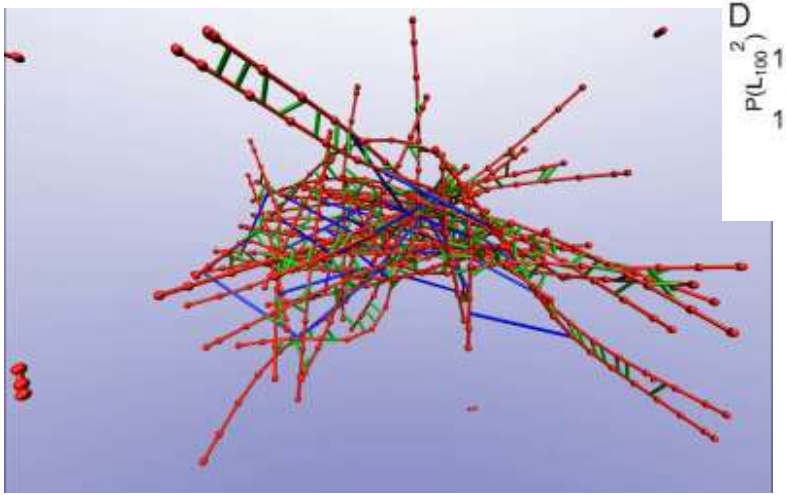
- *Floyd C, Papoian GA, Jarzynski C, Interface Focus, 10.1098/rsfs.2018.0078, (2019)*
- *Liman, …, Wolynes, Levine, Cheung, PNAS, v 117, 10825, (2020)*

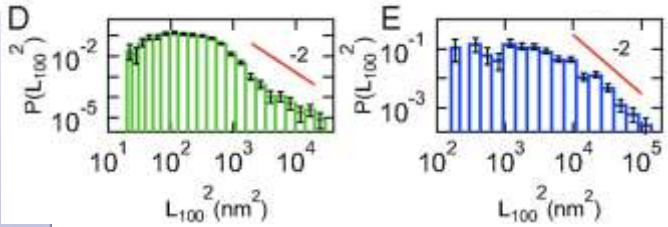


- *In vivo* studies of cytoskeletal motions reveal **heavy-tailed distributions of event sizes** - similar to Gutenberg-Richter law

- "Cytoquakes" have been introduced as **large, sudden events** in cytoskeletal dynamics





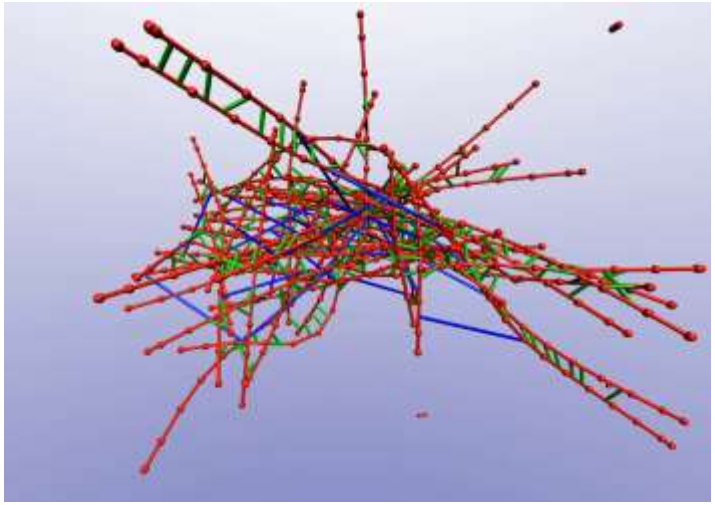


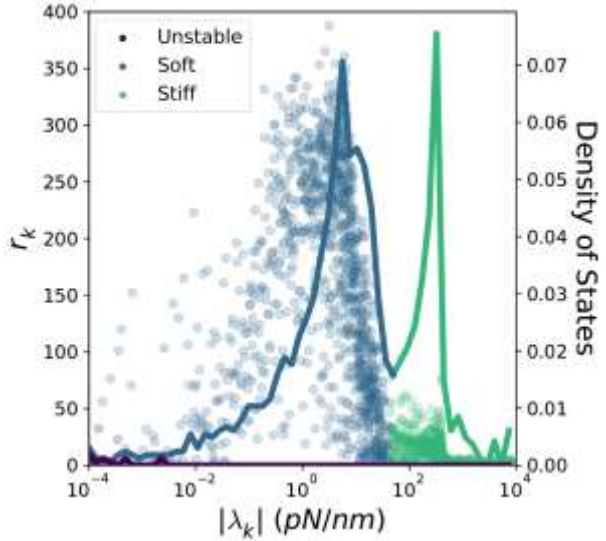
Shi, Y., et al., PNAS (2019)

Cal Floyd

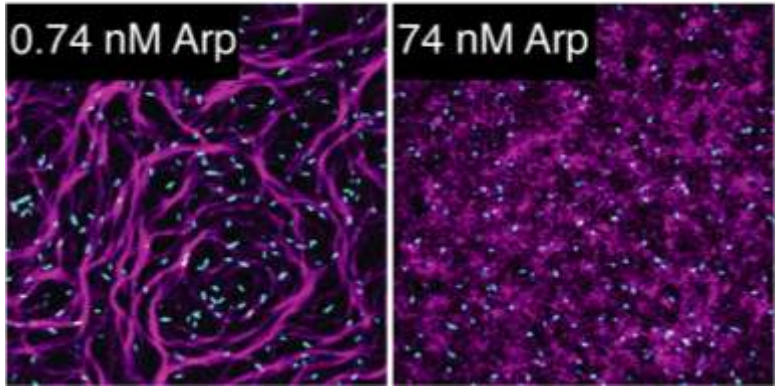
Soft and Stiff Vibrational Modes

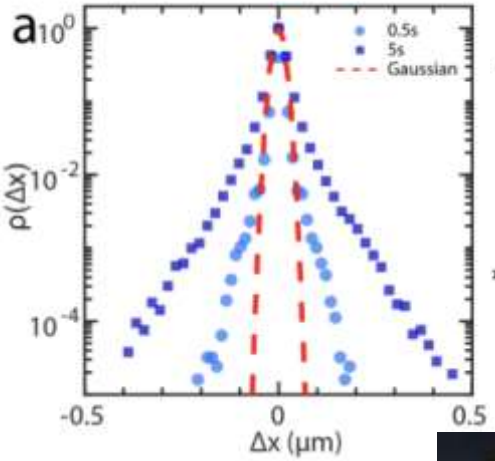
- **Numerically constructed Hessian matrix of U to find the vibrational modes $\mathbf{v}_k$ with stiffness λ_k and delocalization r_k**
- **Soft modes more spatially spread out than stiff modes**

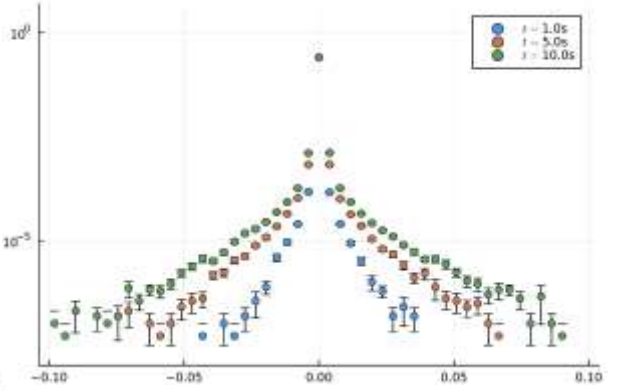




Avalanches in in vitro Actomyosin Systems







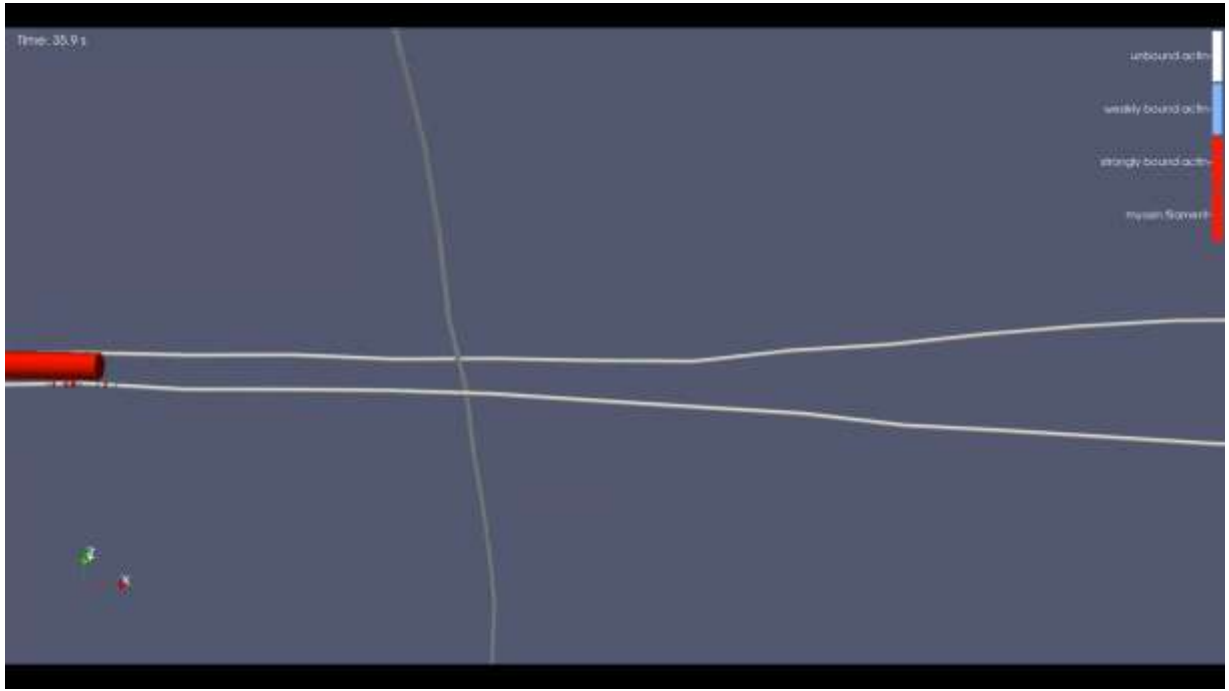
The self-part of the van Hove function, $G_s(x,t)$, for actomyosin system with 0.74nM Arp 2/3. (Murrell Lab) (left). $G_s(x,t)$ for system approximately equivalent to that with MEDYAN. (Papoian Lab) (right).

Nathan Zimmerberg

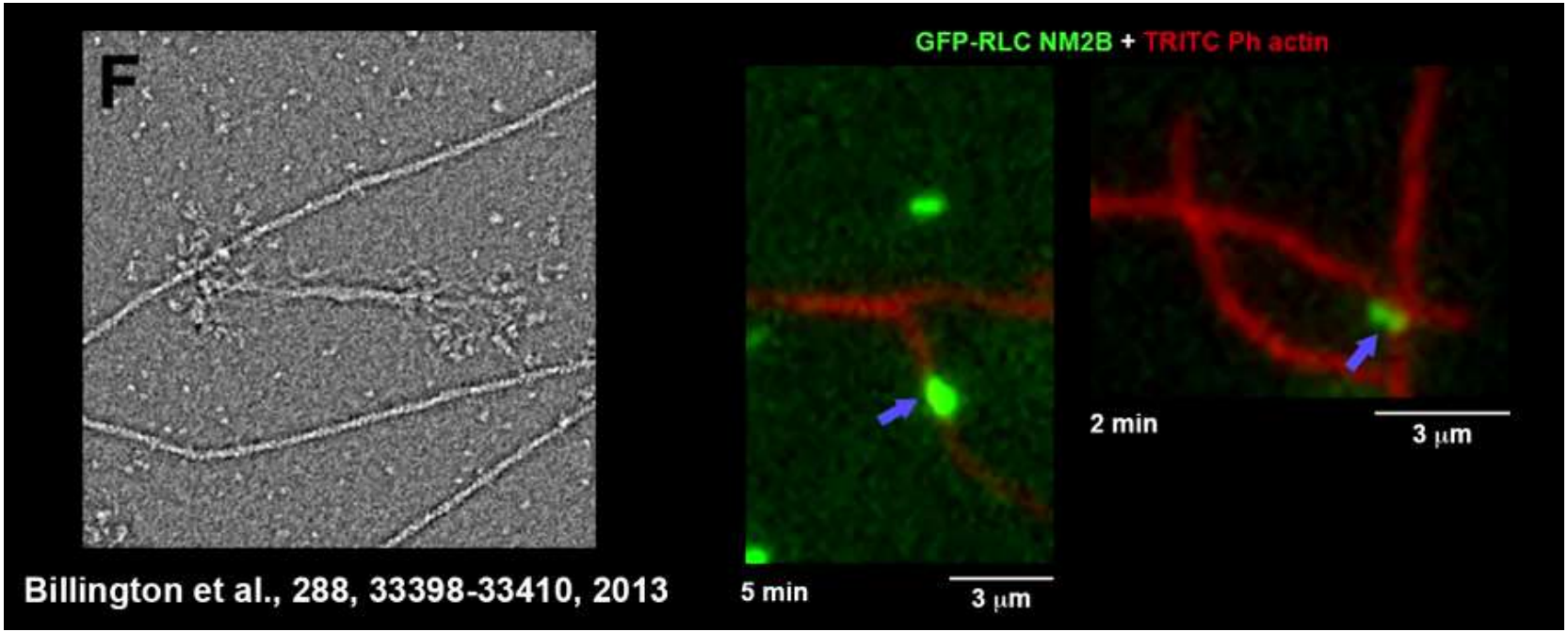
Patrick Kelly

Michael Murrell

Myosin walking on two parallel filaments with a third perpendicular filament blocking its path leads to motor stalling



This kind of motor stalling has been observed experimentally

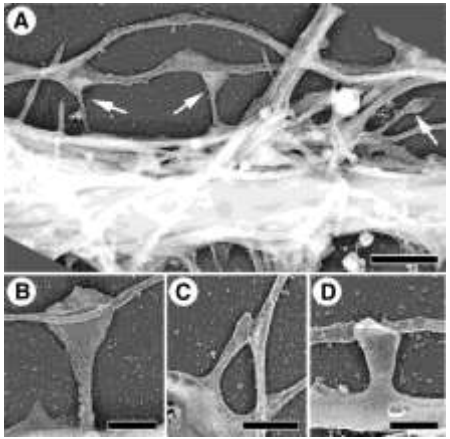


Melli, L., et al. (2018) https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5829915/#video3

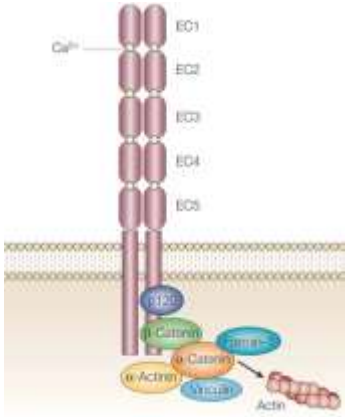
MEDYAN Application: Dendritic Spine

Mengxin Gu

Dendritic Spine: A small protrusion on a neuron's dendrite. Most spines have a bulbous head (the spine head), and a thin neck. Our study concentrated on the post-synaptic spine consisting of branched F-actin, membrane and membrane proteins.



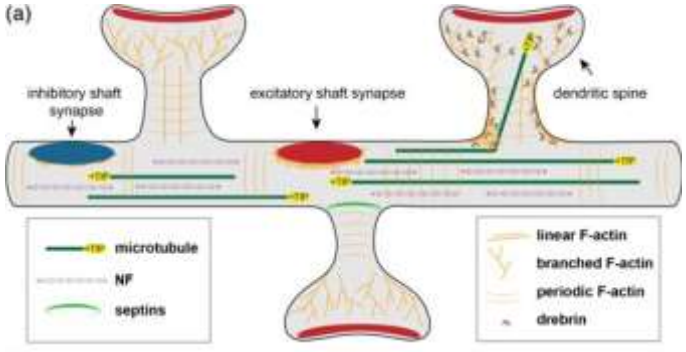
Korobova, F. and Svitkina, T., 2010. Molecular architecture of synaptic actin cytoskeleton in hippocampal neurons reveals a mechanism of dendritic spine morphogenesis. *Molecular biology of the cell*, *21*(1), pp.165-176.



Cadherin: an intercellular protein anchored to F-actin via the catenin proteins, positioned to dynamically regulate spine actin cytoskeleton and is required for the growth and persistence of a spine.

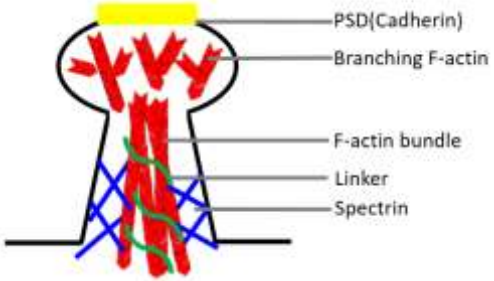
[Bozdagi, O., 2010. Persistence of coordinated long-term potentiation and dendritic spine enlargement at mature hippocampal CA1 synapses requires N-cadherin. *Journal of Neuroscience*, *30*(30), pp.9984-9989.]

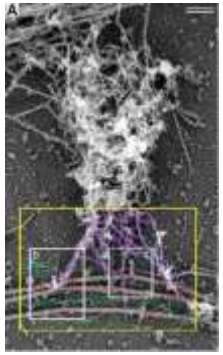
[Gumbiner, B.M., 2005. Regulation of cadherin-mediated adhesion in morphogenesis. *Nature reviews Molecular cell biology*, *6*(8), pp.622-634.]

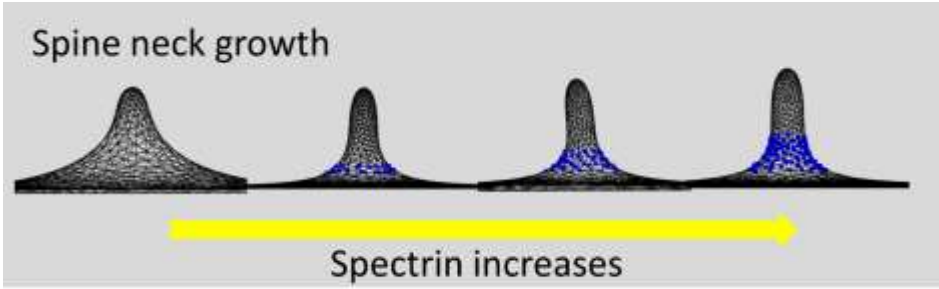


[Bucher, M., Fanutza, T. and Mikhaylova, M., 2020. Cytoskeletal makeup of the synapse: Shaft versus spine. *Cytoskeleton*, *77*(3-4), pp.55-64.]

Simplified spine model

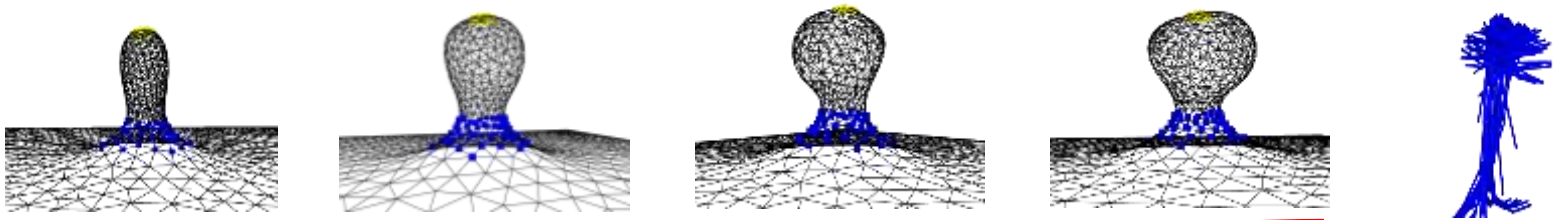




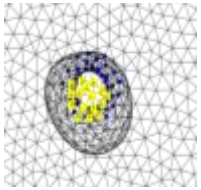


[Efimova, N., 2017. βIII spectrin is necessary for formation of the constricted neck of dendritic spines and regulation of synaptic activity in neurons. *Journal of Neuroscience*, *37*(27), pp.6442-6459.]

Spectrin is model as slip bond. Spectrin distributes from the base of the neck to the base of the head.



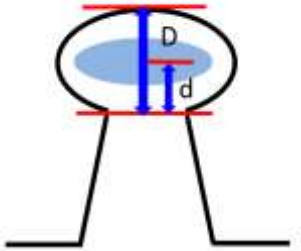
Time Evolution of A Spine: with both spectrin and cadherin, a mushroom-like shape is achieved

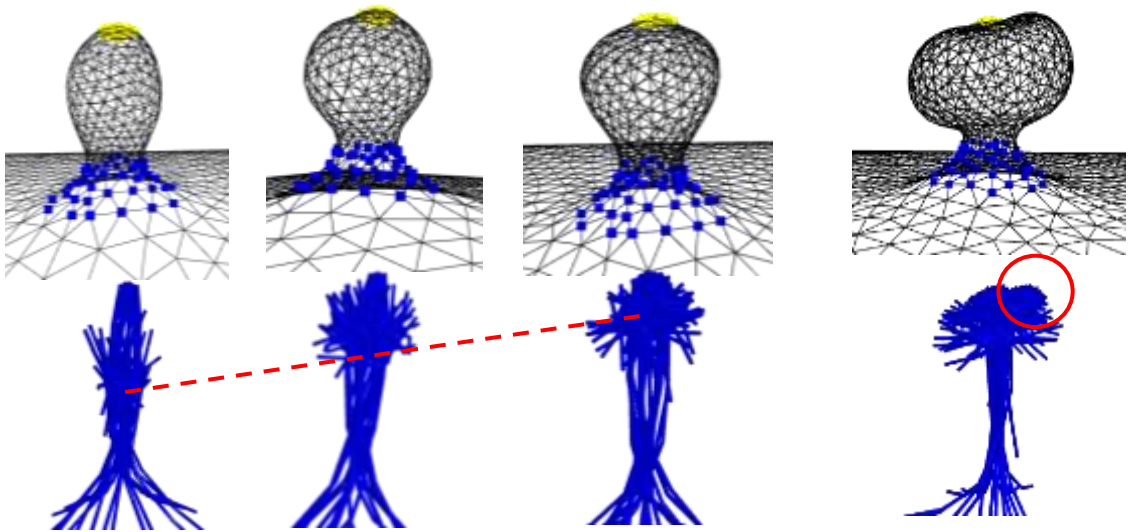


Cadherin distribution

Actin filaments

Effects of Arp2/3 Distribution



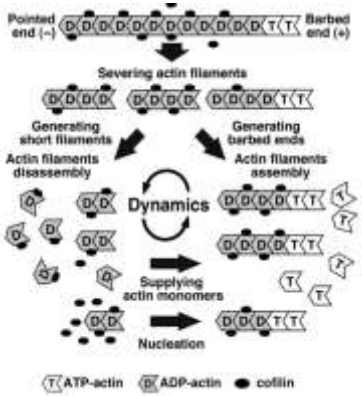


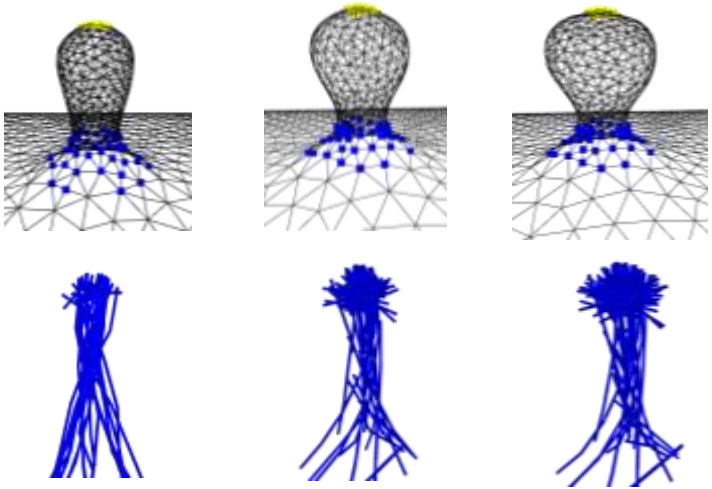
Arp2/3 Distribution ratio = d/D:	0.4	0.6	0.8

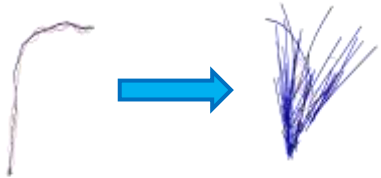
The closer the Arp2/3 area to the PSD, the larger the spine head is. And the upwards branching is one of the reason for a expansion of spine upper surface and contributes to the negative curvature of the post-synaptic membrane.

Filament Severing by Cofilins

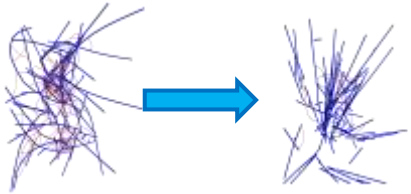
Cofilins sever actin filaments by depolymerizing sequences to free monomers. The structure of cytoskeleton can be obviously changed.





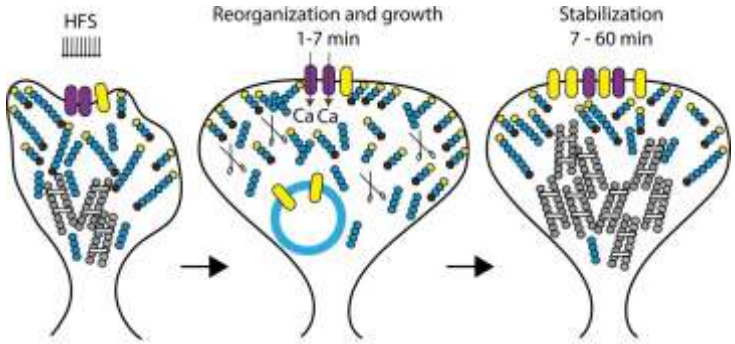


Single F-actin to F-actin bundle



Cytoskeleton is discreted

Cofilins play a key role in Long-term Potentiation (LTP)

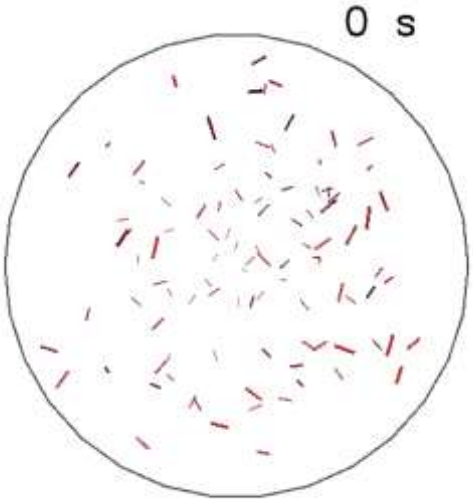


[Hlushchenko, I., Koskinen, M. and Hotulainen, P., 2016. Dendritic spine actin dynamics in neuronal maturation and synaptic plasticity. *Cytoskeleton*, *73*(9), pp.435-441.]

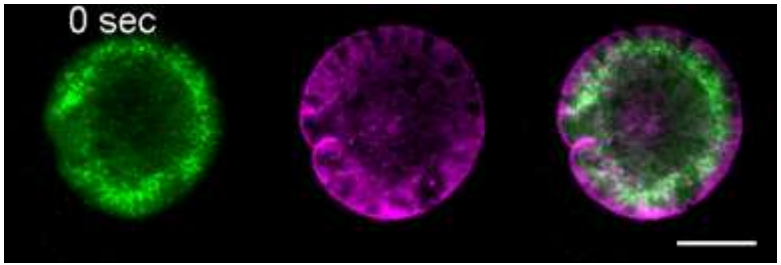
[Ohashi, K., 2015. Roles of cofilin in development and its mechanisms of regulation. *Development, growth & differentiation*, *57*(4), pp.275-290.]

A tug of war between filament treadmilling and myosin induced contractility generates actin ring

eLife, 2022, v11, e82658



T cell activation



- Qin Ni

- Kaustubh Wagh

- Vishavdeep Vashisht

- Arpita Upadhyaya

Thanking colleagues at DeepOrigin and Peter

Michael Antonov
Co-Founder and CEO

Aram Davtyan
Scientific Development

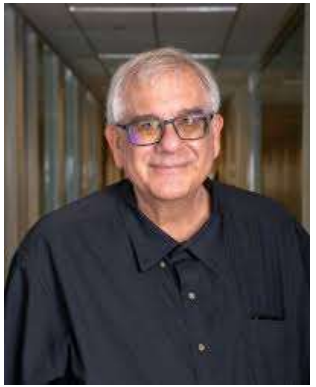
Garik Petrosyan
Scientific Development

Grigor Arakelov
Partnerships

Hayk Saribekyan
Scientific Development

Natalie Ma
Business Development

Ashot Papoyan
COO-Scientific Development



Peter Wolynes (Rice U)

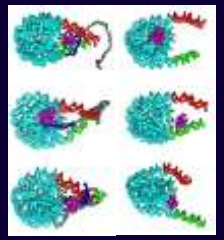
DeepOrigin's Simulation Stack

We build atomistic and coarse-grain models across **biological scales** to address every stage of drug discovery and find drugs faster.

Target Discovery

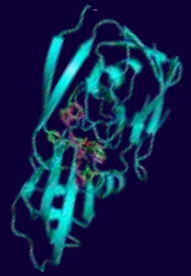
Dynamic Cell Models

Target Characterization



Structural Ensembles

Hit Discovery

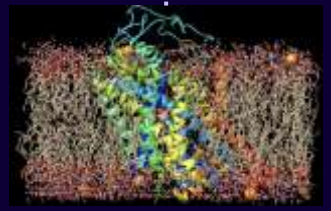


Virtual Screening

Hit Prioritization

Docking & Cheminformatics

Lead Optimization



Molecular Dynamics

Key:

- Available Now
- In Development

ML models have not solved key challenges in the study of proteins

- Can predict:
  - Static folded structure
  - Some structural changes due to mutation (e.g., single amino acid substitution)

- Cannot predict very well:
  - Protein-protein interactions (e.g., dimer and multimer formation, antibody interactions)
  - Interaction with other macromolecules (e.g., DNA, within a lipid membrane)
  - Actual protein folding pathways and kinetics
  - Protein dynamics

AlphaFold, Artificial Intelligence (AI), and Allostery

Published as part of The Journal of Physical Chemistry virtual special issue "Protein Folding and Dynamics—An Overview on the Occasion of Harold Scheraga's 100th Birthday".

Ruth Nussinov,* Mingzhen Zhang, Yonglan Liu, and Hyunbum Jang

Structural bioinformatics

Current structure predictors are not learning the physics of protein folding

Carlos Outeiral, Daniel A. Nissley and Charlotte M. Deane *

Department of Statistics, University of Oxford, Oxford OX1 3PS, UK

*To whom correspondence should be addressed.

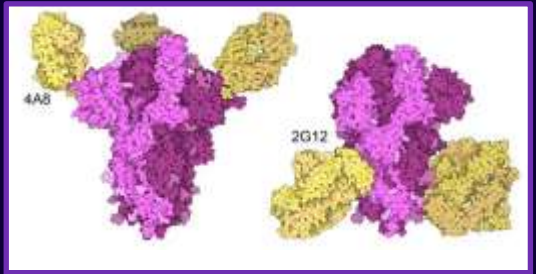
Associate Editor: Lenore Cowen

Abstract

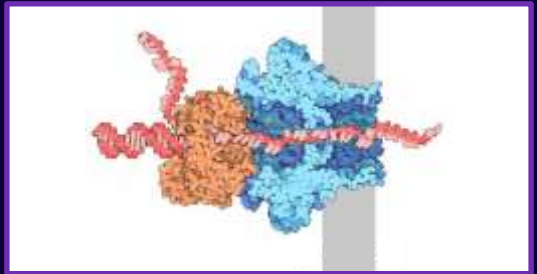
Summary: Motivation. Predicting the native state of a protein has long been considered a gateway problem for understanding protein folding. Recent advances in structural modeling driven by deep learning have achieved unprecedented success at predicting a protein's crystal structure, but it is not clear if these models are learning the physics of how proteins dynamically fold into their equilibrium structure or are just accurate knowledge-based predictors of the final state. **Results.** In this work, we compare the pathways generated by state-of-the-art protein structure prediction methods to experimental data about protein folding pathways. The methods considered were AlphaFold 2, RoseTTAFold, trRosetta, RaptorX, DMPfold, EVfold, SAINT2 and Rosetta. We find evidence that their simulated dynamics capture some information about the folding pathway, but their predictive ability is worse than a trivial classifier using sequence-agnostic features like chain length. The folding trajectories produced are also uncorrelated with experimental observables such as intermediate structures and the folding rate constant. These results suggest that recent advances in structure prediction do not yet provide an enhanced understanding of protein folding.

ML models are incredibly useful, but not for every problem*

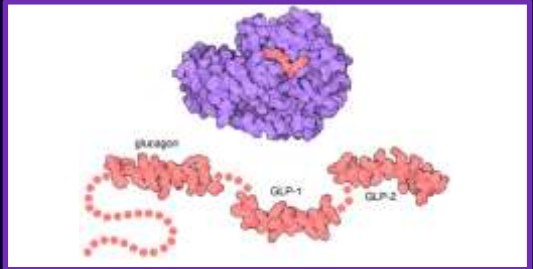
What ML cannot predict is critical to many R&D problems



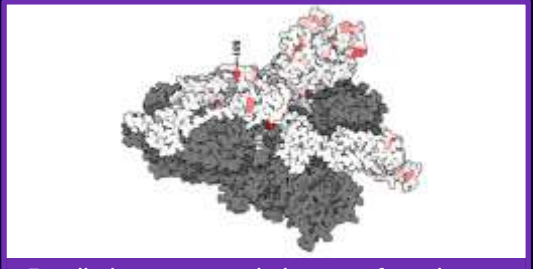
Optimization of protein-protein interactions (e.g., antibody design)



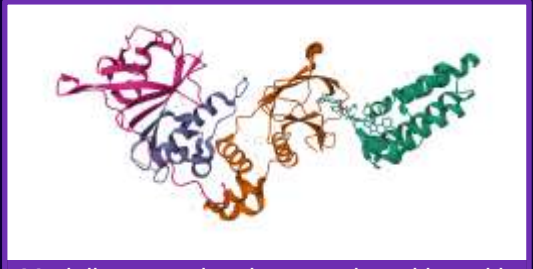
Optimizing macromolecular interactions (e.g., nanopore sequencing)



Predicting changes in conformation from binding (e.g., peptide drug design)



Predicting structural changes from larger or multiple mutations (e.g., target discovery, drug design)



Modeling complex therapeutics with multi-step rate constants (e.g., PROTACs, molecular glues)

Current Landscape in Protein Structure Prediction

Multiple sequence alignment (MSA)-based approaches

- AlphaFold2/3 (Google)
- RoseTTAFold (Baker lab)

Pros:

- High accuracy for natural proteins that have many analogs in sequence databases (MSA data)
- Use both MSA and structural data for training
- Database of ~200 million predicted structures available

Cons:

- Costly to train and run predictions
- Provide single or small number of conformation
- Conformations may not be biologically relevant
- Do not work well for sequences with no MSA data (antibodies, orphan and synthetic proteins).
- Not well applicable to sequences with mutations
- Not well suited for prediction of protein-protein binding and multi-protein assembly

Large language model (LLM)-based approaches

- ESMFold (Facebook)
- OmegaFold (Helixon)

Pros:

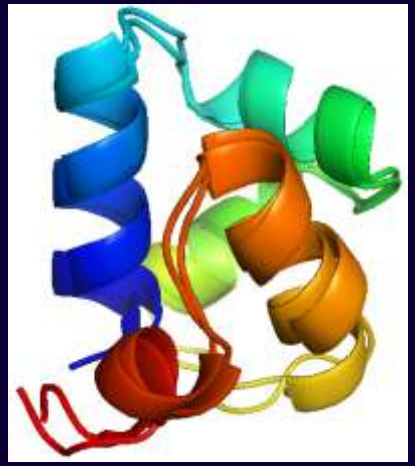
- Predictions are an order of magnitude faster to run
- Work better for synthetic, mutant sequences and orphan proteins due to using single sequence input for training
- Database of ~600 million predicted structures available

Cons:

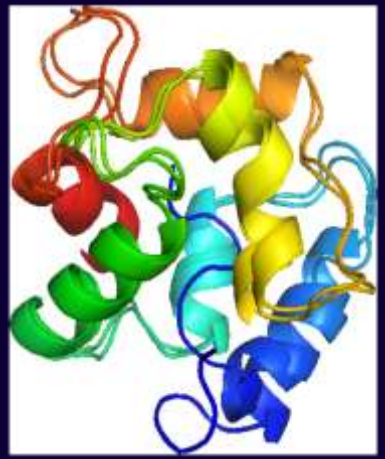
- Training is very costly (15 billion parameters)
- Provide single or small number of conformation
- Conformations may not be biologically relevant
- Lower accuracy for sequences with MSA data
- Not well suited for prediction of protein-protein binding and multi-protein assembly
- Only 1/3 of the database is considered of “high accuracy”

The combination of physical and bioinformatics potentials allows for *de novo* prediction of protein structure

1R69
(RMSD 1.6 Å)



4CPV
(RMSD 1.3 Å)

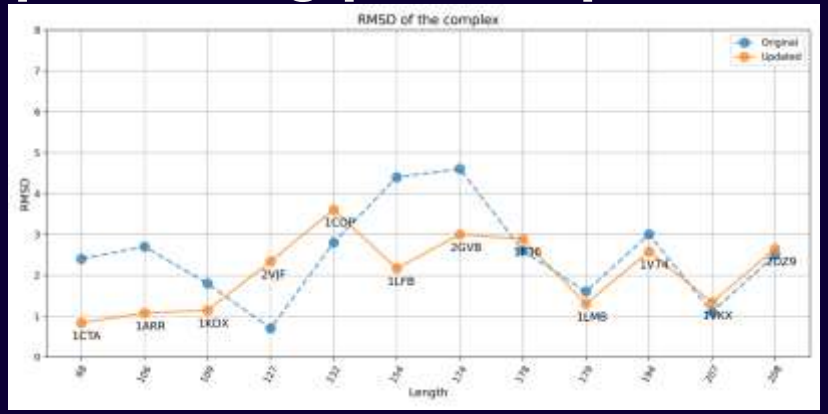


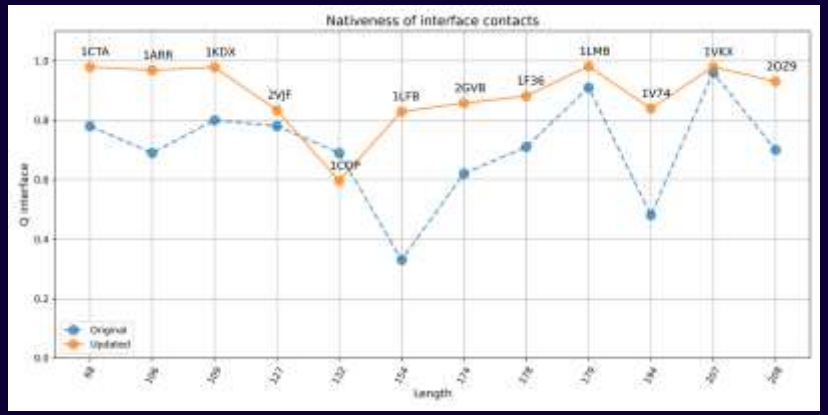
3TMS
(RMSD 1.34 Å)

The superposition of native and predicted structures

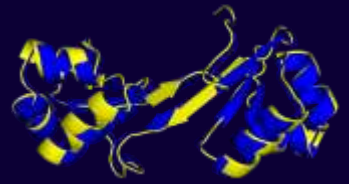
Davtyan, Schafer, Zheng, Clementi, Wolynes, Papoian, **J. Chem Phys B**, 116, (**2012**), 1709–1715

AWSEM demonstrates exceptional performance in predicting protein-protein interactions



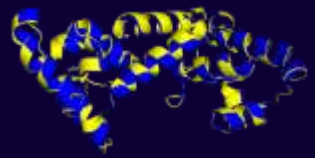


Arc repressor
1ARR N=106

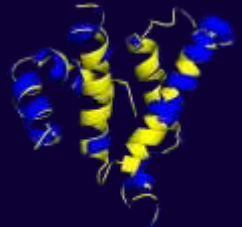


Lambda Cro repressor
1COP N=132

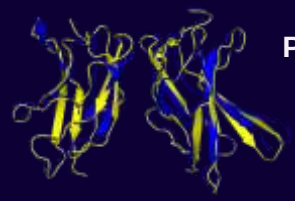
Factor for inversion stimulation
1F36 N=178



Lambda repressor
1LMB N=179



KIX-PKID
1KDX N=109



NFkB P50/P65
1VKX N=207

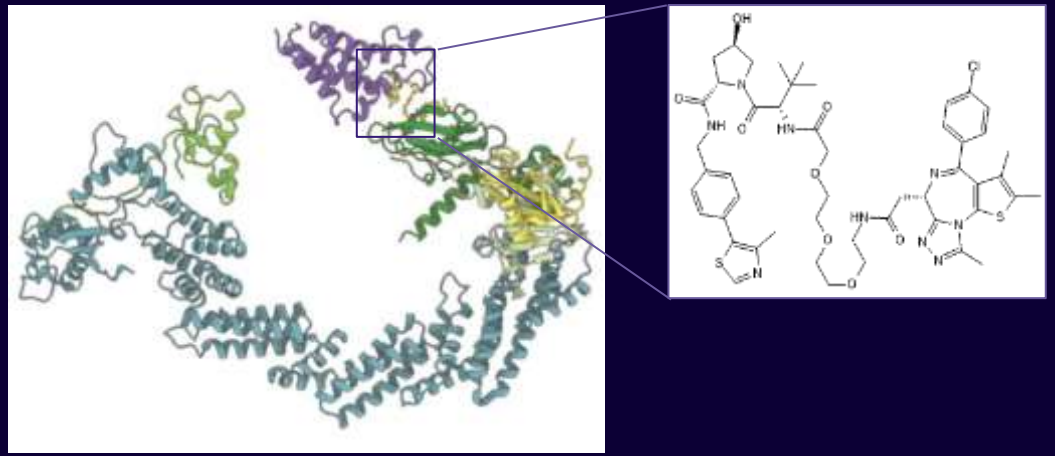
Following Zheng, Schafer, Davtyan, Papoian, Wolynes, **PNAS**, 109, (**2012**), 19244-19249

Crystal structure

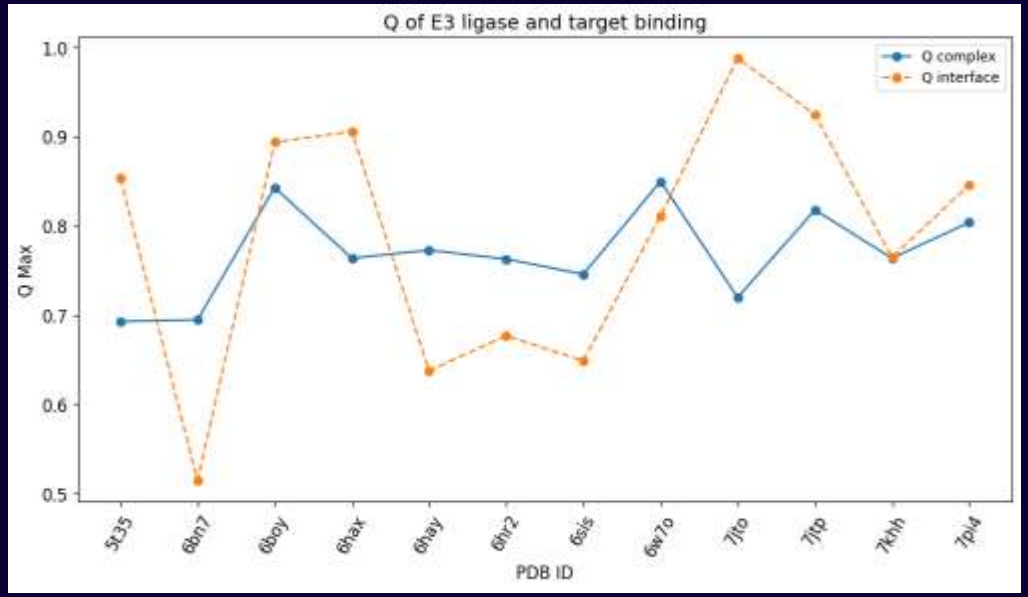
Predicted

Application of AWSEM to complex problems: Design of PROTACs and molecular glues

- Targeted protein degradation and modification is the fastest growing area in drug discovery with over 25 candidates in clinical trials for various diseases.
- However, development and optimization of PROteolysis Targeting Chimeras (PROTACs) and molecular glues remains challenging, especially in absence of structural data.
- Accurate binding simulations with AWSEM can enable rational development and optimization of PROTACs and molecular glues.
- The ability to sample dynamics and assembly of large protein complexes is key to address unsolved problems similar to this.

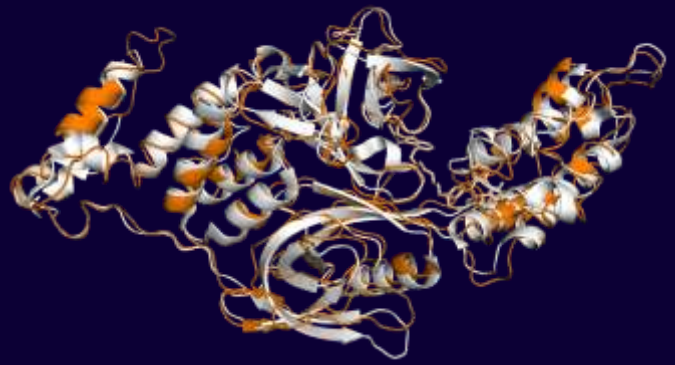


Preliminary results of binding prediction between E3 ligase and target proteins



Q over 0.6 indicates high degree of similarity to the native structure

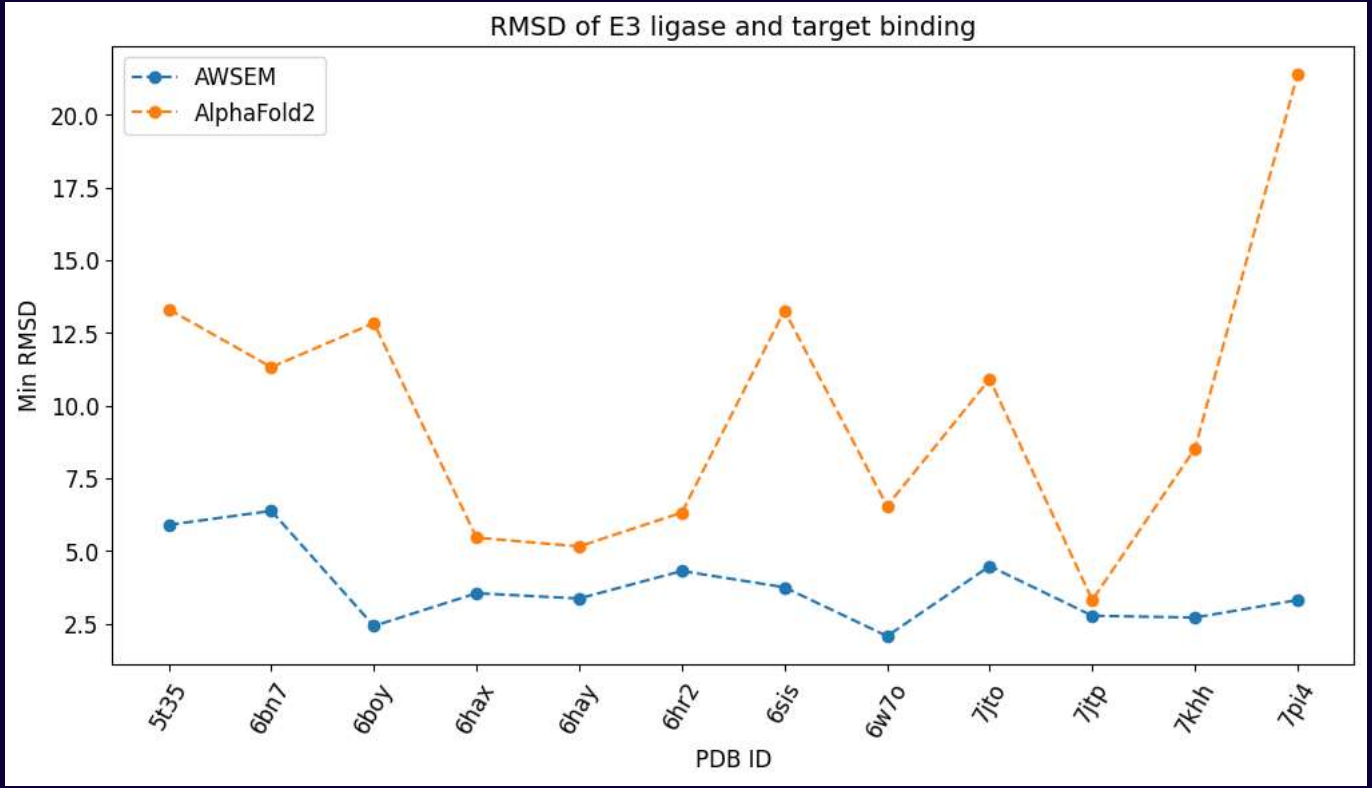
Overlap with native complex of Cereblon and $BRD4^{BD1}$ mediated by dBET6 PROTAC
PDB ID: 6BOY



RMSD: 1.5 Å

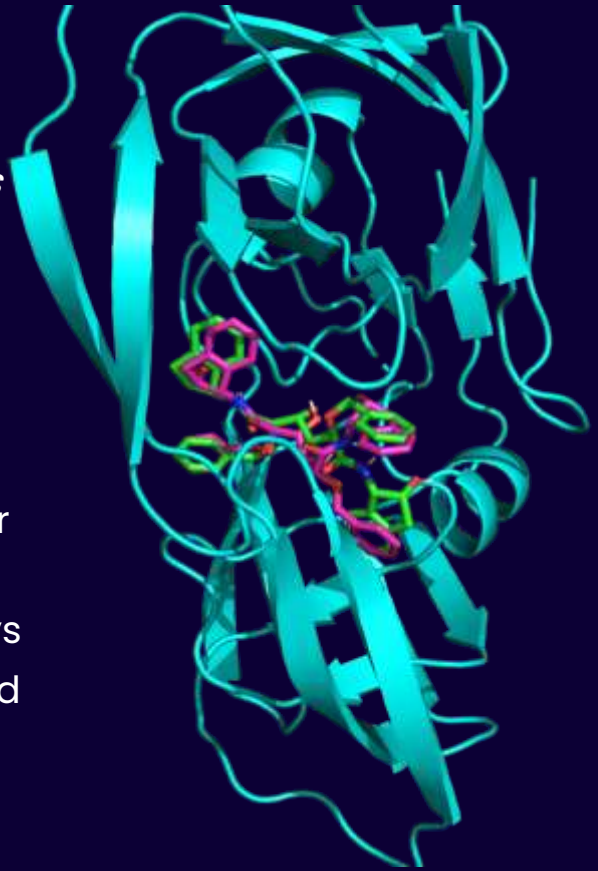
The native structure is shown in white and prediction in orange

E3 ligase/target complex predictions: AWSEM outperforms AlphaFold2



Virtual Screening

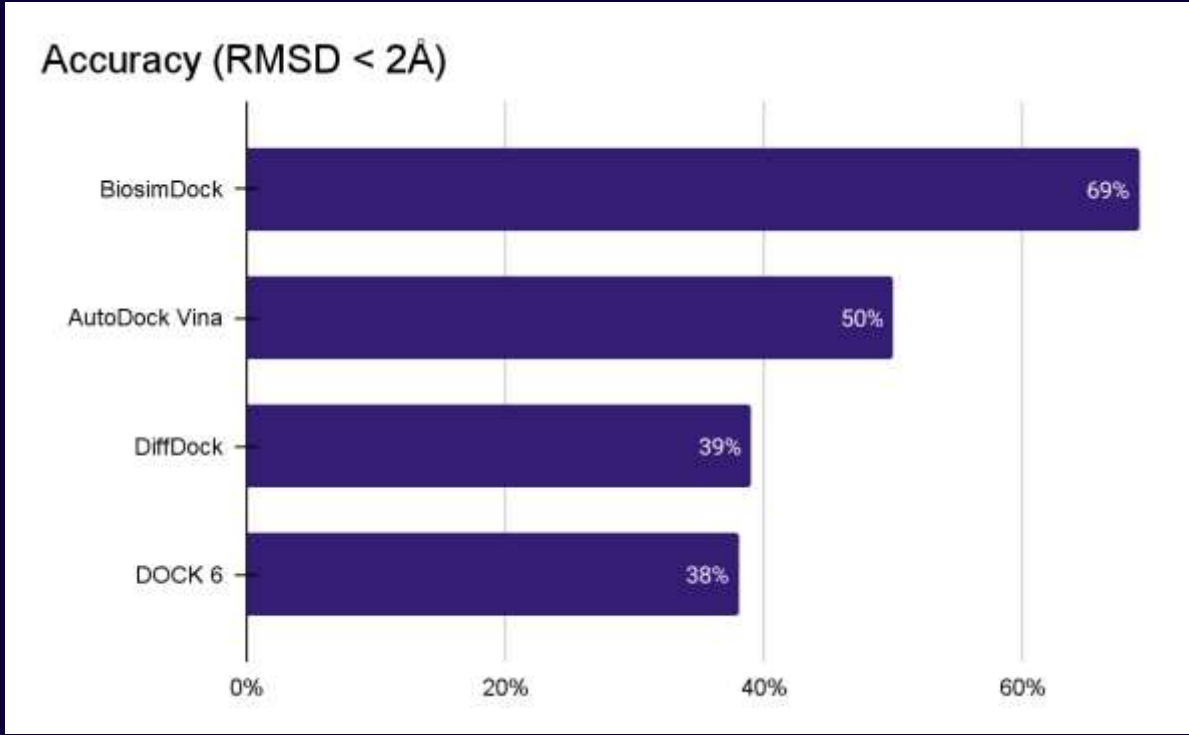
- *Problem: Current VS tools produce mostly false positives and likely miss highest quality binders*
- **How BiosimVS addresses this problem:**
  - Provides new algorithms with significantly better accuracy than then current SOTA
  - Efficiently screens multibillion ligand databases
    - Optimizes for binding affinity and desirable molecular properties
    - Highly efficient: Screening of 5B ligand library in 3 days
    - SOTA property predictors for logS, logP, logD, hERG and other molecular properties
  - Novel Molecular Generative AI



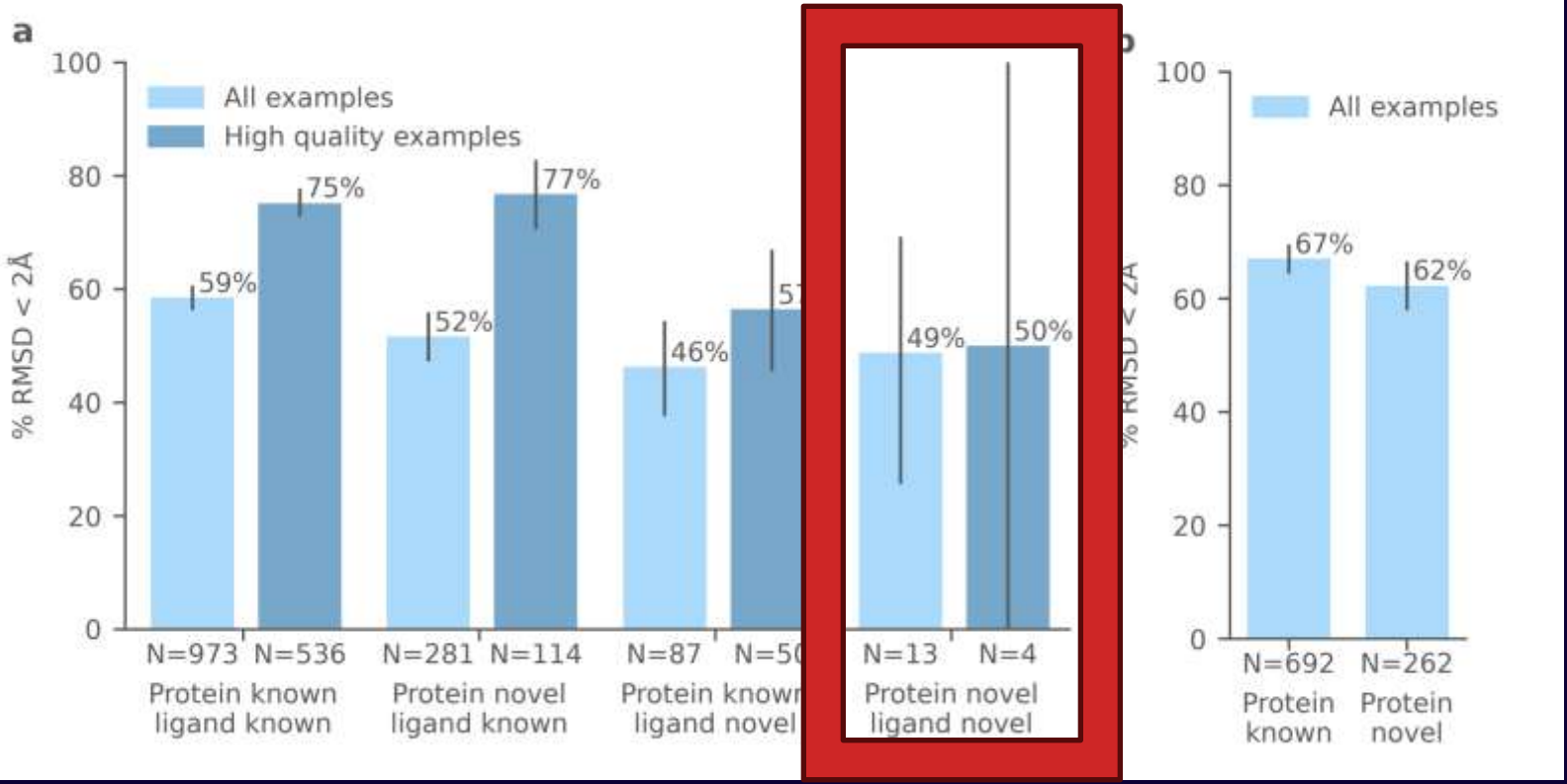
Docking

The test dataset is PDBBind 2020 core set 285 complexes

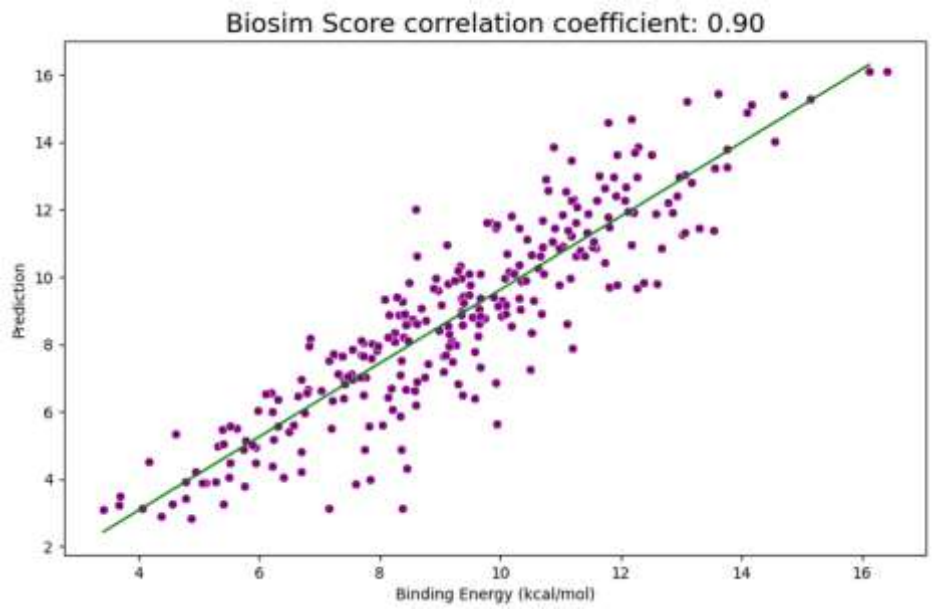
AutodockVina https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3041641/ (25K citations)
DiffDock: https://arxiv.org/abs/2210.01776
DOCK 6: https://onlinelibrary.wiley.com/doi/abs/10.1002/jcc.23905

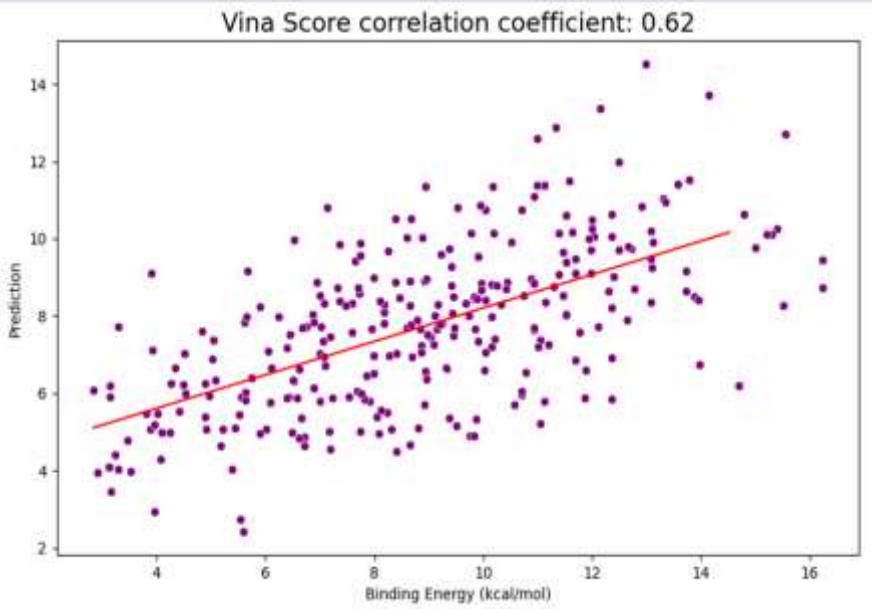


Docking: AF3 Results

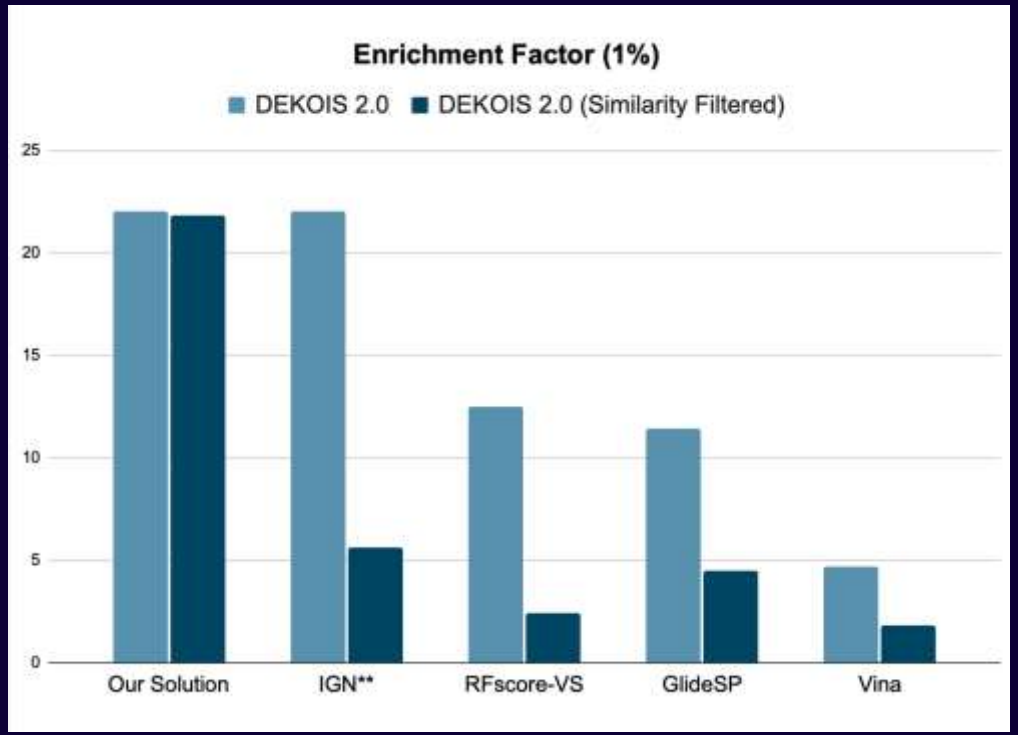


Scoring versus Binding Energies





Virtual Screening



- Significantly outperforms the rest on new target proteins not seen during the training
- IGN was trained on DUD-E dataset, which contained major overlap with the target proteins of DEKOIS2.0 benchmark

Results as reported in "*Jiang, Dejun, et al. "Interactiongraphnet: A novel and efficient deep graph representation learning framework for accurate protein–ligand interaction predictions." Journal of medicinal chemistry 64.24 (2021)*"

Property predictors

logS		
Rank	Model	**MAE**
1	**Biosim Props**	**0.525**
2	Chemprop-RDKit	0.762
3	AttentiveFP	0.776
4	Chemprop	0.818
5	RDKit2D + MLP (DeepPurpose)	0.827
6	Basic ML	0.828
7	GCN	0.907
8	NeuralFP	0.947
9	CNN (DeepPurpose)	1.023

logD		
Rank	Model	**MAE**
1	**Biosim Props**	**0.425**
2	Chemprop-RDKit	0.466
3	Chemprop	0.469
4	BaseBoosting	0.479
5	ContextPred	0.535
6	GCN	0.541
7	AttrMasking	0.547
8	NeuralFP	0.563
9	AttentiveFP	0.572

logP			
Rank	Model	**RMSE**	**SAMPLE6**
1	OCHEM	0.34	0.49
2	**Biosim Props**	**0.449**	**0.421**
3	DNN(taut)	0.47	0.33
4	DNN(mono)	0.50	0.31
5	ACD/GALAS	0.50	0.51
6	ALOGPS	0.50	0.45
8	KOWWIN	0.65	0.53
9	JChem	0.72	0.39

1. TDC.Solubility_AqSolDB
2. Ulrich N., Goss K. U., Ebert A. Exploring the octanol–water partition coefficient dataset using deep learning techniques and data augmentation // Communications Chemistry. – 2021. – Т. 4. – №. 1. – С. 90.
3. TDC.Lipophilicity_AstraZeneca

Case Studies

Tests with small scale virtual screens to answer the following questions:

Can we rediscover known binders and drugs?

How do we perform compared to other tools?

JAK2 (PK Domain)

A comparison to Schrödinger's GLIDE

CD73

Cell surface-anchored nucleotidase implicated in cancer

KRAS (G12D)

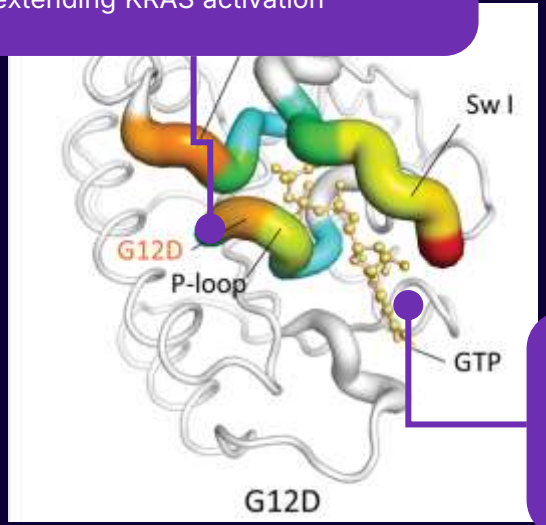
GTPase and classical challenging target

Beyond nucleotide binders: DPP4

Undruggables: targeting KRAS G12D

The GTPase mutated in 15-25% of all cancers and classical challenging target

Mutation of G12D increases P-loop flexibility, lowering binding to GAP effectors prolonging GTP hydrolysis, and extending KRAS activation

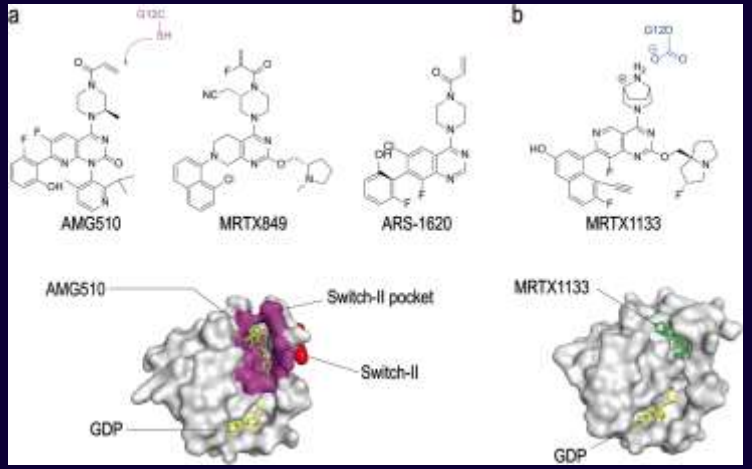


Targeting KRAS is challenging due to lack of apparent binding interfaces

Source: Chen et al, 2013

Given the critical functions of KRAS, drugs must target only **mutant form**

Only a handful are in development:



Source: Zhu et al, 2022

Our performance persists on KRAS G12D

Methods:

- Dock a larger library:
  - 100,000 random molecules from Enamine's virtual library
  - 16 experimentally-validated binders
- Rank compounds based on score of top docking pose
- Top 100 enrichment factors
  - **BiosimAI: 813x**
  - Autodock Vina: 313x

BiosimAI:

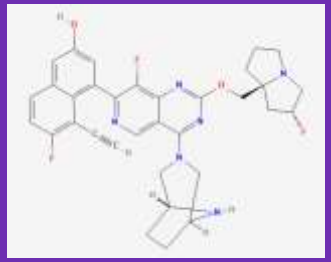
	Molecule ID	BiosimAI Score
1	CHEMBL5081048	-12.07850075
2	CHEMBL4863371	-11.93155384
3	CHEMBL4867851	-11.54409218
4	CHEMBL4876243	-11.39323235
5	CHEMBL4874297	-11.37298679
6	CHEMBL4859236	-11.35258293
7	CHEMBL4855757	-11.30750275
8	CHEMBL4858364	-11.23495483
9	CHEMBL4857719	-11.22687531
10	CHEMBL4872788	-11.02645397
11	CHEMBL4876040	-10.9985075
12	CHEMBL4857438	-10.8617363
13	CHEMBL4863339	-10.62152958
14	Z2518863178	-10.33911133
15	PV-010050840864	-10.19663048
16	PV-010058741110	-10.18711281
17	PV-007134722727	-10.16457748
18	PV-006134781118	-10.15623856
19	PV-008998021938	-10.09503746
20	Z2127055580	-10.08419609

Autodock Vina:

	MoleculeID	Vina Score
1	PV-009984343122	-11.90
2	Z5588179967	-11.90
3	Z3000099585	-11.80
4	CHEMBL4867851	-11.70
5	Z7603912964	-11.70
6	CHEMBL4863371	-11.70
7	Z4564240967	-11.60
8	CHEMBL4863339	-11.60
9	PV-009757158782	-11.60
10	Z2225400372	-11.60
11	Z1067622164	-11.50
12	Z6297285602	-11.50
13	PV-009970661431	-11.50
14	PV-009091048644	-11.50
15	PV-006906778392	-11.40
16	Z2980606341	-11.40
17	PV-006255799432	-11.40
18	Z7614130814	-11.30
19	PV-008670150797	-11.30
20	PV-006151853477	-11.30

We rediscover assets in development for KRAS G12D

The top hit is MRTX1133, a Phase I asset from Mirati Therapeutics



These compounds are patented

BiosimAI:

	Molecule ID	BiosimAI Score
1	CHEMBL5081048	-12.07850075
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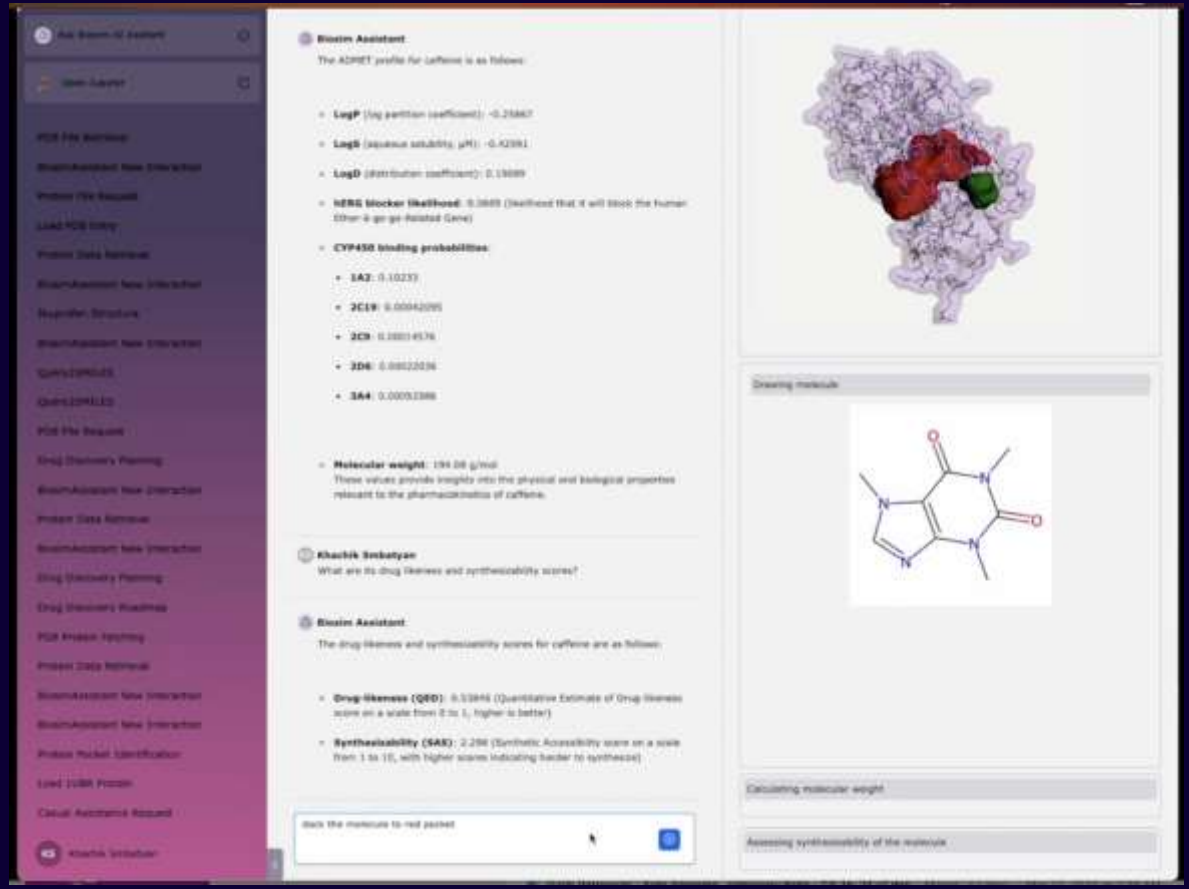
An AI Assistant Interface to Democratize Access



How can I help you today?

Find pockets of 1eby protein

How similar are caffeine and aspirin molecules ?

Show me functional groups of Aspirin

Which of these molecules is more drug-like ? [C=CCc...

can

Workspace