Stanford

Axel Brunger

Professor of Molecular and Cellular Physiology, of Neurology and Neurological Sciences, of Photon Science and, by courtesy, of Structural Biology

Bio

BIO

Axel Brunger, Ph.D., is at Stanford University, where he is a Professor in the Department of Molecular and Cellular Physiology. He is also an Investigator of the Howard Hughes Medical Institute. Brunger is a member of the National Academy of Sciences and the American Academy of Arts & Sciences.
Early in his career, Brunger developed tools for interpreting X-ray crystallography diffraction data that revolutionized structural calculation. In his current research, Brunger applies his expertise in structural biology and biophysics to study the molecular mechanisms of synaptic proteins that enable nerve cell communication. Brunger and his collaborators use biochemical reconstitution, biophysical analyses, and structural biology methods to study the molecular mechanism of neuronal SNAREs, complexin, and synaptotagmin, as well as other factors involved in priming and pre-synaptic plasticity.
His group used single-particle electron cryo-microscopy to determine structures of the supercomplex of SNAREs, the ATPase NSF, and the adapter protein α-SNAP. This subnanometer-resolution structure and functional studies revealed first glimpses of the molecular mechanism of NSF-mediated SNARE complex disassembly, which allows SNARE to be recycled for the next round of synaptic vesicle fusion. Recently, Brunger studied the molecular architecture of proteins and protein complexes at the synapse and in synaptic vesicles using cryo-electron tomography, which led to the discovery of new protein-protein interactions.

ACADEMIC APPOINTMENTS

- Professor, Molecular and Cellular Physiology
- Professor, Photon Science Directorate
- Professor, Neurology and Neurological Sciences
- Professor (By courtesy), Structural Biology
- Member, Bio-X
- Member, Wu Tsai Neurosciences Institute

ADMINISTRATIVE APPOINTMENTS

- Chair, Department of Molecular and Cellular Physioogy, (2013-2017)

HONORS AND AWARDS

- Elected Member, American Academy of Arts & Sciences (2021)
- Trueblood Award, American Crystallographic Association (2016)
- Carl Hermann Medal, German Crystallographic Society (DGK) (2014)
- Katz Award, Exocytosis & Endocytosis Group, Biophysical Society (2014)
- DeLano Award, American Society for Biochemistry and Molecular Biology (2011)

- Elected Member, National Academy of Sciences (2005)
- Gregori Aminoff Prize, The Royal Swedish Academy of Sciences (2003)
- Röntgen Prize in Biosciences, University of Würzburg, Germany (1995)

BOARDS, ADVISORY COMMITTEES, PROFESSIONAL ORGANIZATIONS

- Investigator, Howard Hughes Medical Institute (1987 - present)

PROFESSIONAL EDUCATION

- Diplom, University of Hamburg , Physics (1980)
- Ph.D., Technical Univ. of Munich , Biophysics (1982)

LINKS

- My Lab Site: http://atbweb.stanford.edu

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

My current research primarily focuses on the molecular mechanisms of synaptic neurotransmission. in 1998, my laboratory performed several pioneering structural studies: We determined the first X-ray crystal structure of the neuronal SNARE complex, setting the framework for subsequent functional and mechanistic studies. My laboratory also determined X-ray crystal structures of other key components of the synaptic release machinery, including parts of the ATPase NSF (N-ethylmaleimide sensitive factor), Sec17 (an α-SNAP homolog), synaptotagmin's C2 domains, and Rab proteins and their effectors. Moreover, we determined the complex between the catalytic domain of botulinum neurotoxin A and its target SNARE (SNAP-25).

In 2015 and 2017, my lab determined crystal structures of the SNARE complex bound to the Ca2+-sensor synaptotagmin-1 and the regulator complexin at atomic resolution, revealing two interfaces, with at least one of the two being essential for fast synchronous release of neurotransmitters. The structures of these complexes suggest, along with functional studies in vitro and in neuronal cultures, that it is in a primed and locked state. Action-potential-driven Ca2+ ions bind to the synaptotagmin proteins, unlock the complex, and trigger membrane fusion on a sub-millisecond timescale. We used the structures of one of these two interfaces, the so-called primary interface, to develop an inhibitor of Ca2+-triggered exocytosis and mucin hypersecretion that could apply to controlling mucin-related diseases.

To complement our structural studies, we developed reconstituted systems with synaptic proteins and isolated synaptic vesicles to study synaptic vesicle fusion at the single-vesicle and single-molecule levels. This system revealed new insights about fusion pathways and the molecular mechanisms of synaptic vesicle priming and fusion.

After fusion, SNARE complexes are recycled by the ATPase NSF, which breaks down the SNARE complex into its components. This disassembly process is also essential for quality control for fusogenic SNARE complex formation in cooperation with Munc13 and Munc18. We determined structures of the so-called 20S complex consisting of the SNARE complex, α-SNAP, and NSF by single particle electron cryo-microscopy; this complex has provided first glimpses of the mechanism of this molecular recycling machine. The SNARE complex resembles a rope with a left-handed twist, and NSF uses adapter proteins called SNAPs to grasp the "rope" in multiple places. The SNAPs wrap around the SNARE complex with a right-handed twist. We recently discovered how the SNARE complex is loaded into NSF via side-loading and engagement of the N-terminal residues of one of the SNARE proteins (syntaxin or SNAP-25), mediated by conserved tyrosine residues in the pore of the NSF D1 ring.

Recently, we studied the molecular architecture of synapses by cryo-electron tomography. We showed that the presynaptic machinery leads to distinct inter-membrane proteinaceous interfaces in the resting state through 3D reconstructions of isolated synaptic vesicles bound to reconstituted acceptor liposomes and reconstructions of entire synapses of neural cultures. We thus uncovered that the neurotransmitter release machinery establishes stable prefusion inter-membrane complexes that facilitate fast fusion upon calcium triggering. We also discovered new interactions between macromolecules in situ: (1) Complexes of AMPA receptors with postsynaptic density scaffolding proteins (PSD). This work revealed that these molecules participate in a well-defined network that likely contributes to AMPAR stabilization and clustering. (2) A new interaction between the synaptic vesicle proteins synaptophysin and the V-ATPase. This work suggests that synaptophysin is involved in the biogenesis of synaptic vesicles by controlling the copy number of V-ATPases and possibly other molecules, such as synaptobrevin, that also interact with synaptophysin.

Teaching

STANFORD ADVISEES

Doctoral Dissertation Reader (AC)

Ipsita Krishnamurthy

Postdoctoral Faculty Sponsor

Liv Jensen

Doctoral Dissertation Advisor (AC)

Jiahao Liang

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Biophysics (Phd Program)
- Molecular and Cellular Physiology (Phd Program)
- Neurosciences (Phd Program)
- Structural Biology (Phd Program)

Publications

PUBLICATIONS

- **The scientific legacy of Martin Karplus from the perspective of his collaborators.** *Biophysical journal*
  Andricioaei, I., Best, R. B., Birge, R. R., Boresch, S., Brunger, A., Brooks, B. R., Buck, M., Brüschweiler, R., Caflisch, A., Case, D. A., Cui, Q., Dejaegere, A., Dinner, et al
  2026
- **Editorial overview: Cryo-electron microscopy (2025).** *Current opinion in structural biology*
  Brunger, A. T., Frank, G. A.
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- **Recent Structural Insights into the Molecular Architecture of Synapses.** *Advances in neurobiology*
  Brunger, A. T., Held, R. G., Khan, Y. A., Leitz, J., Liang, J., Wang, C., White, K. I.
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- **Synaptobrevin-2 disease variants reveal spatial constraints within the presynaptic active zone.** *Proceedings of the National Academy of Sciences of the United States of America*
  Guzikowski, N. J., Bagatelas, E. D., Shin, O. H., Khan, Y. A., Esquivies, L., Alten, B., Brunger, A. T., Kavalali, E. T.
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- **Structural remodeling of target-SNARE protein complexes by NSF enables synaptic transmission.** *Nature communications*
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- **SNARE disassembly requires Sec18/NSF side loading.** *Nature structural & molecular biology*
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  2024
- **Pre-fusion AAA+ remodeling of target-SNARE protein complexes enables synaptic transmission.** *bioRxiv : the preprint server for biology*
  White, K. I., Khan, Y. A., Qiu, K., Balaji, A., Couoh-Cardel, S., Esquivies, L., Pfuetzner, R. A., Diao, J., Brunger, A. T.
  2024
- **Sec18 side-loading is essential for universal SNARE recycling across cellular contexts.** *bioRxiv : the preprint server for biology*
  Khan, Y. A., Ian White, K., Pfuetzner, R. A., Singal, B., Esquivies, L., Mckenzie, G., Liu, F., DeLong, K., Choi, U. B., Montabana, E., Mclaughlin, T., Wickner, W. T., Brunger, et al
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