

Resource Summary Report

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Adaptive Poisson-Boltzmann Solver

RRID:SCR_008387

Type: Tool

Proper Citation

Adaptive Poisson-Boltzmann Solver (RRID:SCR_008387)

Resource Information

URL: <http://www.poissonboltzmann.org/apbs/>

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Description: APBS is a software package for modeling biomolecular solvation through solution of the Poisson-Boltzmann equation (PBE), one of the most popular continuum models for describing electrostatic interactions between molecular solutes in salty, aqueous media. APBS was designed to efficiently evaluate electrostatic properties for such simulations for a wide range of length scales to enable the investigation of molecules with tens to millions of atoms. It also provides implicit solvent models of nonpolar solvation which accurately account for both repulsive and attractive solute-solvent interactions. APBS uses FETk (the Finite Element ToolKit) to solve the Poisson-Boltzmann equation numerically. FETk is a portable collection of finite element modeling class libraries written in an object-oriented version of C. It is designed to solve general coupled systems of nonlinear partial differential equations using adaptive finite element methods, inexact Newton methods, and algebraic multilevel methods.

Abbreviations: APBS

Resource Type: software resource

Keywords: software package, modeling, biomolecular, electrostatic, molecular, dynamics, binding energy, equilibrium, protein, ligand, solvation, kinetics, simulation, finite element

Funding: IBM/American Chemical Society ;
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Resource Name: Adaptive Poisson-Boltzmann Solver

Resource ID: SCR_008387

Alternate IDs: nif-0000-30035

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Record Last Update: 20260725T190650+0000

Ratings and Alerts

No rating or validation information has been found for Adaptive Poisson-Boltzmann Solver.

No alerts have been found for Adaptive Poisson-Boltzmann Solver.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 50 mentions in open access literature.

Listed below are recent publications. The full list is available at [PRECISE-TBI](#) .

Lucaciu SA, et al. (2025) Skin disease-associated GJB4 variants differentially influence connexin stability, cell viability and channel function. The Journal of physiology, 603(15), 4383.

Mahana Y, et al. (2024) Structural evidence for protein-protein interaction between the non-canonical methyl-CpG-binding domain of SETDB proteins and C11orf46. Structure (London, England : 1993), 32(3), 304.

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Deák G, et al. (2022) Missense Variants Reveal Functional Insights Into the Human ARID Family of Gene Regulators. Journal of molecular biology, 434(9), 167529.

Zhao R, et al. (2022) Inhibition of histone H3-H4 chaperone pathways rescues C. elegans sterility by H2B loss. PLoS genetics, 18(6), e1010223.

de la Morena-Barrio ME, et al. (2022) Two SERPINC1 variants affecting N-glycosylation of Asn224 cause severe thrombophilia not detected by functional assays. Blood, 140(2), 140.

Jiang X, et al. (2022) DYF-5/MAK-dependent phosphorylation promotes ciliary tubulin unloading. Proceedings of the National Academy of Sciences of the United States of America, 119(34), e2207134119.

Strong LM, et al. (2021) Structural basis for membrane recruitment of ATG16L1 by WIPI2 in autophagy. eLife, 10.

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- Maharana J, et al. (2017) NOD1CARD Might Be Using Multiple Interfaces for RIP2-Mediated CARD-CARD Interaction: Insights from Molecular Dynamics Simulation. *PloS one*, 12(1), e0170232.
- Aguilar OA, et al. (2017) A Viral Immuno-evasin Controls Innate Immunity by Targeting the Prototypical Natural Killer Cell Receptor Family. *Cell*, 169(1), 58.
- Hardianto A, et al. (2017) Exploration of charge states of balanol analogues acting as ATP-competitive inhibitors in kinases. *BMC bioinformatics*, 18(Suppl 16), 572.