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designed a method to foresee modeling accuracy for each individual protein of interest, with an average error of 7 percentage points, replacing previously available mean accuracy over a numerous set of different proteins with experimentally solved structures. The studies show that for modelling unknown protein structure 30% of correctly predicted contacts can be sufficient for acceptable model quality. Nevertheless, methods using residue-residue contact maps need methods to differentiate between models of native and mirror orientation. Ramachandran plots of the structures are not suitable for big data sets and difficult for proteins rich in beta-sheets. Total energies of structures are often not helpful. We analyzed structural protein models obtained from contact maps of 1 305 SCOP domains from 7 structural classes and proposed an automated method for differentiating model orientations, independent of their secondary structures. The best algorithm used k-means clustering with three common energy terms: probability of amino acid assuming certain values of dihedral angles, Ramachandran preferences and Coulomb interactions. The accuracies were in the range between 0.68 and 0.76, with sensitivity and selectivity in the range between 0.68 and 0.87, depending on the structural class. Modeling methods based on contact maps can be applied to all fully-automated tools for protein structure reconstruction, especially those analyzing big sets of models.

P-27-066
Investigation of disease-causing germline mutations in postsynaptic density

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The postsynaptic density (PSD) is a complex and dynamic network of interacting proteins involved in synaptic transmission. Polymorphisms in some of the proteins of the PSD are associated with neuronal diseases such as Autism Spectrum Disorder, schizophrenia or Parkinson disease. The rapidly growing data on human genomic variations enables large scale studies of the distribution of variations in PSD proteins. Disease-causing germline mutations (DCMs) represent a special class with relatively weak phenotypes. In this study we investigated the effect of single nucleotide variations on protein structures from the PSD. Our approach reveals how DCMs might contribute to functional changes, in ordered and intrinsically disordered regions. Our results also suggest that proteins in PSD are much more exposed to single nucleotide variations compared to those of other proteins in the human proteome. Associating DCMs with protein structure and disease groups may provide a better understanding of the underlying mechanisms that govern disease emergence.

P-27-067
Amyloidogenic properties of human protein NOS1AP

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The gene *NOS1AP* encodes a cytosolic protein that binds to the signaling cascade component, neuronal nitric oxide synthase (nNOS). Moreover, the *NOS1AP* gene is linked with disorders from schizophrenia, post-traumatic stress disorder, and autism to cardiovascular disorders and breast cancer. NOS1AP/CAPON protein mediates signaling between complex of NMDA receptor, PSD95 and nNOS. This adapter protein is involved in neuronal nitric-oxide (NO) synthesis regulation via its association with nNOS/NOS1. It mediates the indirect interaction between NOS1 and RASD1, leading to an increase NOS1 ability to activate RASD1. Resulting from our bioinformatics analysis, NOS1AP protein was predicted as one of the components of the amyloid network surrounding APP (amyloid-beta precursor protein). Extracellular amyloid aggregates of NOS1AP in the system C-DAG (curli-dependent amyloid generator) were detected by red colony color on the medium supplied with the amyloid binding dye Congo Red and "apple-green" birefringence in polarized light. Fluorescent microscopy revealed the aggregation of EGFP-NOS1AP in yeast *Saccharomyces cerevisiae*. SDD-AGE proved out these aggregates were resistant to the detergent (SDS) treatment. Mammalian cell line HEK293 transiently expressed the same construct also represented the local aggregation of the investigated protein. Thus, NOS1AP protein shows amyloid properties, that is of great interest considering the fact it can interact with APP, so there is a chance the molecular mechanisms of neurodegenerative diseases can be more complex. The authors acknowledge the Resource Center "Centre for Molecular and Cell Technologies" of the Research Park of St Petersburg State University. The research was supported by the Russian Science Foundation (17-74-10159).

P-27-068
Protein–lipid interactions in glycophorin-like dimerization motifs of transmembrane helices

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Receptor tyrosine kinases (RTK) are vital players in cell signaling governing growth and proliferation. These integral membrane proteins work only in dimeric states, so the conformation of transmembrane dimer determines the signal transferred into cell. Here, we used modern molecular modeling techniques to study details of protein-protein and protein-lipid interactions in model systems containing monomers and dimers of several receptor tyrosine kinases with glycophorin-like dimerization motifs. Comparison of structural and dynamic aspects of ErbB family members and glycophorin A (GpA) revealed similarities in their properties, especially, for ErbB1, ErbB2 and ErbB4 receptors utilizing the same GpA-like motif for dimerization in their basal state. We demonstrated that they all have similar organization of TM domain's molecular surface in terms of both relief, hydrophobic properties and lipid binding sites resembling GpA pattern studied before. All these RTKs strongly interact with lipid acyl chains, forming stable binding sites both in monomeric and dimeric states, and the most prominent binding areas are located in monomers on the future GpA-like dimerization interfaces. Then, lipids distribution changes upon dimer formation. This is not the case for alternative packing geometries observed for the second state of ErbB1 and, especially, ErbB3. We found higher numbers of immobilized lipids near C-terminus in ErbB1 and ErbB2 active dimers, thus assuming that the existing structure of ErbB3 is also active. However, there is non-functional GpA-like motif in ErbB3 with some bound lipids present near the N-terminus, suspecting

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