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POSTERS

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Indexing

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allowing the investigation of the G4 influence on the initial steps of *E. coli* MMR. Suggested DNA structure was verified via DNA footprinting and ^{1}H-NMR. The interaction of MutS and G4 in the conditions providing different MutS conformations was characterized with apparent dissociation constants calculated from EMSA data. The nearly full independence from nucleotide cofactor present was shown implying the G4 binding mode to differ from the one with GT-mismatch. To elucidate ATPase activity in presence of G4 DNA, we employed malachite green assay for phosphate detection. For the first time we investigated the binding of MutL protein to G4 DNA. The significantly higher affinity to G4 as compared to other DNA molecules was discovered. We further subjected model G4 to cleavage by MutH protein. The DNA hydrolysis efficiency by MutH-MutL-MutS complex was demonstrated to be not dependent on G4 presence in DNA duplex. Therefore, despite the efficient interaction between G4 and both MutS and MutL with the affinity higher than to DNA with a mismatch, G4 DNA does not serve as a substrate for MMR initiation, and actual cellular function of G4-MutS and G4-MutL complex formation is yet to be found. This work was supported by RFBR grant No. 18-34-00768.

P-27-086
The interdependence between amyloid formation and virulence of Proteobacteria

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Proteobacteria represents largest phylum of the gram-negative bacteria including as various pathogens of humans, mammals and plants as symbiotic species some of which perform beneficial for multicellular host functions, like nitrogen fixation. Recent data obtained by several research groups suggest that the virulence of different pathogenic Proteobacteria species are associated with formation of highly ordered protein fibrils by proteins acting as the virulence factors. Such fibrils called amyloids, represent important structural component of biofilms playing crucial role in virulence of various *Proteobacteria* species, and they are related to bacterial toxin formation. In addition, we have previously found that M60 mucin metalloprotease of *Escherichia coli* that is involved in the pathogenesis of the enterotoxigenic strains of these bacteria, is amyloidogenic. Thus, repertoire of functions of amyloid-forming virulence factors of Proteobacteria is apparently wider than we expected so far. We analyzed abundance of amyloidogenic regions in the proteomes of more than 80 species of the order Rhizobiales belonging to the class Alphaproteobacteria using different bioinformatic algorithms. We found these regions tended to be overrepresented in the proteins associated with virulence of these bacteria and comprising so-called beta-barrel structure typical for outer membrane proteins. Further experimental verification including analysis of detergent resistance, fibril formation in the C-DAG system and ability to bind amyloid-specific dyes confirmed amyloid properties of such outer membrane proteins of agriculturally important nitrogen-fixing species *Rhizobium leguminosarum*. Overall, these data demonstrate that amyloid formation by virulence factors is important for capability of different bacterial species to colonize multicellular host. The study of amyloid proteins of *R. leguminosarum* was supported by the Russian Science Foundation, grant 17-16-01100.

P-27-087
Structural instability of FGF1 R50E mutant restricts its mitogenic potential

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FGF1 has been shown to interact with integrin alphavbeta3 through a specific binding site, involving Arg50 residue. The FGF1 mutant (R50E) with impaired integrin binding was found to be defective in proliferative response, although it was still able to bind to FGFR and heparin and induce activation of downstream signaling pathways. Here we demonstrate that the lack of mitogenic potential of R50E mutant is directly caused by its decreased thermodynamic stability and susceptibility to degradation *via* proteolysis. Introduction of three stabilizing mutations into R50E variant compensated the effect of destabilizing mutation and restored the proliferation potential of FGF1, while remaining defective in binding to integrin alphavbeta3. Our results suggest that the thermodynamic stability and resistance to degradation rather than interaction with integrin are required for mitogenic response of FGF1. Acknowledgments: The work was supported by the National Science Centre, Poland (Sonata Bis 2015/18/E/NZ3/00501). *The authors marked with an asterisk equally contributed to the work.

P-27-088
Selective Hsp70-dependent docking of Hsp104 to protein aggregates protects the cell from the toxicity of the disaggregase

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Hsp104 is a yeast chaperone that rescues misfolded proteins from aggregates associated with proteotoxic stress and aging. Hsp104-dependent protein disaggregation involves extraction of a polypeptide from an aggregate and its translocation through the central channel of the Hsp104 hexamer. This process relies on Hsp104 cooperation with the Hsp70 chaperone, which also plays an important role in the regulation of the disaggregase. Although the protein-unfolding activity of Hsp104 enables cells to survive stress, when uncontrolled, it becomes toxic. There is a trade-off: on one hand, this protein-remodeling machine must be powerful enough to tackle protein aggregates, on the other hand, it needs to be restricted to prevent promiscuous unfolding of disordered proteins with important cellular functions. Here, we investigated how collaboration with Hsp70 at the initial stages of protein disaggregation allows to maintain this balance. Using the hyperactive, toxic Hsp104 variant with disrupted Hsp70-binding site we demonstrate that the cooperation with Hsp70 shifts Hsp104 substrate specificity from non-aggregated, disordered substrates towards protein aggregates. Our results show that the Hsp70-mediated, selective recruitment for disaggregation makes the Hsp104 unfoldase less toxic and more productive.

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