Functional and structural study of T5 bacteriophage immunity protein Llp

Résumé

Phage infection is triggered by host recognition thanks to the Receptor Binding Protein binding to its receptor at the surface of the cell: this interaction results in viral DNA delivery into the host cytoplasm. This first step of infection is followed by viral replication and eventually liberation of the new virions. During this vulnerable time, phages protect the new viral factory from over-infection. In coliphage T5, protection is mediated by a periplasmic lipoprotein, Llp (1), targeted to the inner leaflet of the outer-membrane, which binds the phage receptor FhuA (2), (3). Llp biological function is probably also to prevent the inactivation of progeny phage by active receptors present in outer-membrane debris of lysed cells, thereby increasing their chances of infecting a new host. We aim to decipher the mechanisms of T5 host inhibition by Llp at the molecular level. We over-expressed Llp in an acylated (Ac-Llp) and soluble (Sol-Llp) form in quantities compatible with biochemical and structural studies, and solved Sol-Llp (7.5 kDa) structure by NMR. We could show that Ac-Llp protects the overexpressing strain from T5 infection and we characterized the FhuA:Ac-Llp complex by several biochemical and biophysical methods. I will discuss my result in view of the structure of the FhuA-Llp complex reported by B Berg et al 2022(4).

Mots-Clés: bacteriophage, interaction host pathogen, phage T5