CeNT CENTRE OF NEW TECHNOLOGIES

UNIVERSITY OF WARSAW

invites to a seminar by

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What is a fit end for a viral RNA? Lessons from the IFIT proteins

26th April 2018 at 12 p.m.

Venue: Centre of New Technologies, Banacha 2C,
Lecture Hall 0142 (Ground floor)

Host: Joanna Kargul

Detection of viral infection in the cell often occurs by recognition of unusual RNA features in the cytosol that differ from the host transcripts. The interferon-induced proteins with tetratricopeptide repeats (IFITs) are potent innate immune effectors that bind non-self RNA, which results in the inhibition of translation of viral transcripts. The structure of IFIT5 monomer reveals the mode of recognition of the 5' triphosphate (PPP) group on RNA, whereas the dimeric IFIT1 preferentially binds cap 0 groups. IFIT1 interacts with IFIT3, which has no known RNA binding capability on its own, and for which the role in the larger multi-IFIT complex was elusive. We study the role of the higher-order IFIT complexes, in particular the IFIT1-IFIT3 interaction. In cells, IFIT1 and IFIT3 associated together, and re-distributed and co-localized together with PPP-RNA. The IFIT1-IFIT3 assembly is mediated by the last tetratricopeptide repeat motifs in both proteins, and results in reorganization of the RNA-binding site in IFIT1, so that the IFIT1-IFIT3 complex binds RNA with a higher affinity than IFIT1 alone. We propose a role for IFIT3 as a constitutive partner and an enhancer of IFIT1 activity. Regulation of the IFIT1-IFIT3 complex may provide additional possibility for signal integration in the antiviral response.

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