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## *How bacterial effectors hijack small GTPases to divert membrane traffic*

J. Cherfils[1], M. Folly-Klan[1], V. Campanacci[1]
[1]*Laboratoire d'Enzymologie et Biochimie Structurales, CNRS, Gif-sur-Yvette, France*

Membrane traffic, which is the "cellular postal service" that shuttles biomolecules around the cell and organizes the structure of organelles, is among the primary targets of effectors injected by intracellular pathogenic bacteria to invade their host and avoid from being destroyed. I will present our recent structural and biochemical studies of effectors from Legionella pneumophila (the bacteria that causes the legionnaire's disease, a severe pneumonia) that divert membrane traffic to generate a membrane-bound vacuole where the pathogen hides and replicates. One of these effectors, AnkX, is a FIC domain-containing toxin that alters the functions of a Rab GTPase involved in vesicular traffic at the endoplasmic reticulum, by covalent attachment of a phosphocholine molecule. The other one, RalF, functions as an illegitimate guanine nucleotide exchange factor to activate an Arf GTPase on the vacuole. Our studies showed how AnkX binds and processes CDP-choline to transfer phosphocholine onto Rab1 [1], and uncover a novel membrane sensor in RalF that controls its localization and activity [2]

***[1]*** *M. Folly-Klan, E. Alix , D. Stalder, P. Ray, L.V. Duarte, A. Delprato, M. Zeghouf, B. Antonny, V. Campanacci, C.R. Roy, J. Cherfils PLoS Pathog. , 2013, 9(11):e1003747,* ***[2]*** *V. Campanacci, S. Mukherjee, C. R. Roy, J. Cherfils. EMBO J., 2013, 32:1469-77.*

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