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Meeting abstract

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The PCH family member CIP4 is released from T cells upon activation-induced cell death

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The 'Pombe Cdc15 homology' (PCH) family of adaptor proteins gained much attention only very recently. PCH proteins link membrane trafficking events to the actin cytoskeleton. Structurally, a so-called F-BAR domain characterizes all members of the PCH family and recent studies indicate that this domain enables PCH proteins to bind to and deform cellular membranes resulting in membrane curvature and tubulation. Accordingly, PCH proteins have been functionally associated with endo-/exocytosis and the trafficking of vesicles. As several PCH proteins interact with the death factor FasL, we were interested in the expression and function of PCH proteins in T cells in this context.

Employing a new monoclonal anti-CIP4 antibody generated in our laboratory, we initially observed that the PCH family member CIP4 is expressed in T cells only after activation indicating a specific role in T cell maturation or effector function. Surprisingly, staining activated T cells for CIP4 followed by laser scanning microscopical inspection revealed that CIP4 seems to be somehow released from the cells in vesicular structures resembling exo- or ectosomes. The vesicle release can be visualized as a direct plasma membrane "budding". Morever, CIP4-microvesicle formation and release proved to be enhanced during the initial phase of activation-induced T cell death triggered e.g. by exposure to staphylococcal superantigen. To us this indicates that this process might be relevant during T cell death to provide additional danger signals to neighboring cells. We are currently analysing the molecular content of these vesicles, the role of CIP4 in their generation and their impact on T cell biology.

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