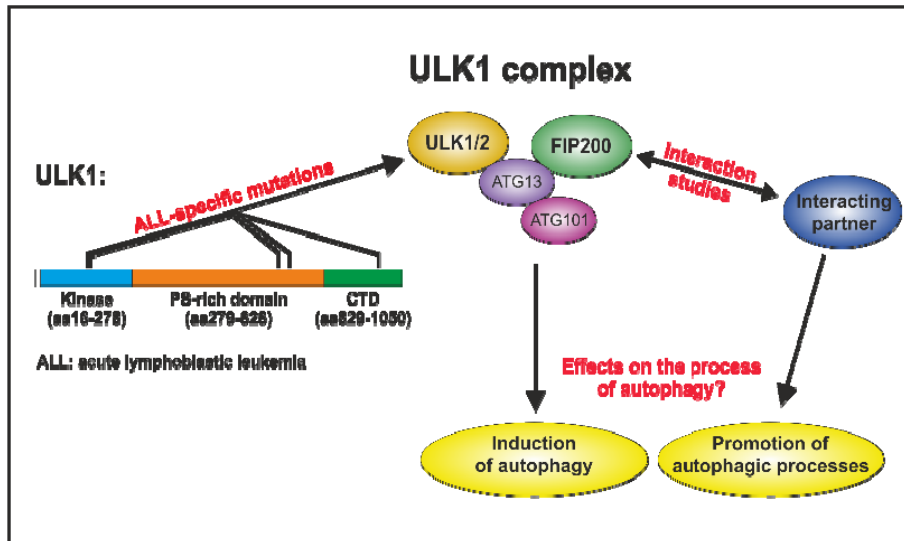


Analysis of the influence of ALL-specific ULK1 mutations and novel FIP200 interaction partners on the process of autophagy



Autophagy, an intracellular recycling process for damaged or long-lived proteins and organelles, has a dual role for cancer therapy, since it impedes cancer development in earlier and promotes it in later stages. The ULK1 complex, consisting of ULK1, FIP200, ATG13 and ATG101, is the main complex for initiating autophagy. In cooperation with the Department of Pediatric Oncology, Hematology and Immunology, we found several ULK1 mutations in patients with relapsed acute lymphoblastic leukemia (ALL). Therefore, these mutations will be characterized regarding autophagy signal transduction, sensitization toward chemotherapy and changes in the ULK1 interactome. Furthermore, we identified a novel interacting partner of FIP200. In this project the interaction area between these two partners will be characterized. Additionally, due to the importance of this novel interacting partner for specific autophagic processes, this interaction will be analyzed regarding its influence on autophagy.

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