A study of the crosstalk between Unc-51 Like Autophagy Activating Kinase 1 and Protein Tyrosine Phosphatases

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Autophagy is a lysosomal catabolic process responsible for the degradation and recycling of cellular constituents. Autophagy has important roles in development, immune defense, programmed cell death, tumor suppression and prevention of neuron degeneration. The process is initiated by a serine/threonine kinase, Unc-51 like autophagy activating kinase 1 (ULK1). Due to the pivotal role that ULK1 plays in inducing autophagy and considering the role autophagy plays in disease, this research is aimed at understanding broader ULK1 signaling. The higher the signaling characterization of players within the autophagy pathway, especially its dominant inducer, ULK1, the better the chances to adequately address autophagy related questions. Recent published evidence shows a novel network of phosphatase complexes that are direct targets of ULK1. This study extends upon these findings and explores the functional crosstalk between ULK1, and protein tyrosine phosphatases implicated in this network. To answer these questions, we perform phospho-tyrosine immuno-precipitation mass spectrometry coupled with western blotting. We conclude on a novel role of ULK1 as an inhibitor of a protein tyrosine phosphatase that results in the upregulation of oncogenic kinases; EGFR, AKT, and ERK in a context-specific manner particular to distinct primary and cancer cell lines

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