Title:	Comparison of RUN Domain-containing Proteins for binding to the Small GTPase Arl8b
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Abstract:	Cells have a dynamic environment which varies with space and time. In such a heterogeneous environment, homeostasis can only be maintained by the regulated trafficking of molecules in and out of the cell, a generalized process termed as endocytic and membrane trafficking. During endocytosis, vesicles that pinch off from the membrane are transported by motor proteins to different target organelles, and finally fuse with the destination to deliver the cargo molecules. The entire mechanism of vesicular fission and fusion is rather complex wherein a number of molecular players are involved. Small GTPases are active regulators in this process. They recruit different effector molecules that promote vesicular budding, transport, tethering and fusion. Arf-like (Arl) GTPase, Arl8b, is a lysosomal small GTPase that facilitates late endosome-lysosome fusion. SifA and kinesin-interacting protein (SKIP) is a well known effector of Arl8b that regulate the anterograde motility of lysosomes by binding to Arl8b via its RUN domain. Recently our lab has identified a new effector of Arl8b, PLEKHM1 (Pleckstrin homology domain-containing family M member 1), which also binds to Arl8b via its RUN-domain. So as a part of this thesis work, I have analyzed the interaction between Arl8b and PLEKHM1 that specifically localizes to late endosomes/lysosomes. Our preliminary results indicate that the RUN-domain of SKIP recruits Arl8b more strongly as compared to PLEKHM1.
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