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Abstract
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Investigating the Binding of Vasodilator-Stimulated Phosphoprotein (VASP) family of proteins and Arg Non-Receptor Tyrosine Kinase *in vitro*

Cell motility is the culmination of an array of metabolic processes, in particular the extension and turnover of cytoskeletal polymers of actin and tubulin. The reorganization of these structures is regulated in part by the cAMP-dependent protein kinase (PKA) and its interactions with the Mena/VASP family of proteins. VASP (vasodilator-stimulated phosphoprotein), and the related proteins Mena (mammalian *enabled*) and EVL (Ena-/VASP-like) constitute an evolutionarily conserved family of proteins that regulate actin dynamics and cell adhesion. The ability of VASP to regulate these cellular functions depends upon its interaction with the Abelson non-receptor tyrosine kinase (Abl). This interaction occurs directly between the proline-rich region of VASP and the SH3 domain of Abl. Arg (Abl-related gene) is a homologue of Abl and a proven constituent in cell movement. Both Abl and Arg have been implicated in human cancers, with an oncogenic form of Abl responsible for chronic myeloid leukemia and Arg related to acute myeloid leukemia. The structures of Arg and Abl are highly conserved with one exception that dictates the exclusive cytoplasmic localization of Arg and others that provide Arg additional domains for cytoskeletal interaction. These exceptions indicate that Arg may have a higher importance than Abl in regulating cytoskeletal dynamics. Importantly, the SH3 domain of Arg has been shown to have 90% amino acid identity with that of Abl. We hypothesize that Mena/VASP family members interact directly with Arg exactly as they do with Abl. To prove this postulate we are investigating the ability for complexes to form *in vitro* between all members of the Mena/VASP family and Abl or Arg using purified proteins. This research will provide insight into the relationships between these families of proteins and will allow for future studies of post-translational phosphoregulation by PKA and higher order regulation of interactions in intact human cells.