Role of membrane bending in ASAP1 protein activity BEATRIZ E. BURROLA GABILONDO, University of Maryland, RUIBAI LUYO, National Cancer Institute, WOLFGANG LOSERT, University of Maryland, PAUL A. RANDAZZO, National Cancer Institute — ASAP1 is part of the protein machinery that alters membranes and the actin cytoskeleton in cellular structures, called invadopodia, that mediate invasion of mammary cell carcinoma and uveal melanoma. The molecular mechanism by which ASAP1 contributes to these structures is not well defined. ASAP1 induces the hydrolysis of GTP that is bound to the protein Arf. Another activity is to deform lipid bilayers into tubules. We have set out to test the hypothesis that the enzymatic GAP activity is related to the mechanical activity. We contrast several reaction schemes for GAP activity, including steps that would be sensitive to physical changes in the membrane. We compare the numerical model predictions to data obtained from kinetics experiments. We are also developing assays such as FRET and tools like laser tweezer forcing of vesicle deformations to be used to determine the effect of ASAP1 and mutants with defects in enzymatic activity on the physical state of lipid vesicles. The ramifications of the results to the role of ASAP1 in invadopodia formation will be discussed.