AIM1 suppresses migration and metastatic dissemination in prostate cancer.

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Background:
A defining hallmark of primary and metastatic cancers is the migration and invasion of malignant cells. These invasive properties involve altered dynamics of the cytoskeleton and one of its major structural components β-actin. Here, we identify AIM1 (absent in melanoma 1) as an actin binding protein that suppresses pro-invasive properties in benign prostate epithelium.

Methods and Results:
Depletion of AIM1 in prostate epithelial cells increases cytoskeletal remodeling, intracellular traction forces, cell migration and invasion, and anchorage independent growth. In addition, decreased AIM1 expression results in increased metastatic dissemination *in vivo*. AIM1 strongly associates with the actin cytoskeleton in prostate epithelial cells in normal tissues, but not in prostate cancers. In addition to a mislocalization of AIM1 from the actin cytoskeleton in invasive cancers, advanced prostate cancers often harbor AIM1 deletion and reduced expression.

Conclusions:
These findings implicate AIM1 as a key suppressor of invasive phenotypes that becomes dysregulated in primary and metastatic prostate cancer.

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