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In this presentation, I will discuss recent work towards a quantitative understanding of amoeboid motility. Amoeboid motility refers to the crawling-like movements that amoeba, zebrafish primordial germ cells and supposedly also neutrophils and cancer cells make use of to migrate. It is powered by variations actomyosin contractility that causes localized in cytosolic pressure. These in turn deform the plasma membrane, forming localized protrusions that drive crawling by cell shape changes. We have developed a computational model for this process, focusing on the mechanics involved. From our mechanobiological model, we delineate the range and scope of such protrusion-associated motility. Next, using neutrophil-like HL-60 cells, we tested some of the predictions of the model experimentally. In particular, we showed that the speed of amoeboid migration exhibits a biphasic relationship with the extent to which these cells are confined, suggesting that the pore size in extracellular matrices can be an important factor in regulating the invasion of cells by the amoeboid modality.