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Phosphoinositide 3-kinase (PI3K)γ and *Dictyostelium* PI3K are activated via G protein–coupled receptors through binding to the Gβγ subunit and Ras. However, the mechanistic role(s) of Gβγ and Ras in PI3K activation remains elusive. Furthermore, the dynamics and function of PI3K activation in the absence of extracellular stimuli have not been fully investigated. We report that *g*β null cells display PI3K and Ras activation, as well as the reciprocal localization of PI3K and PTEN, which lead to local accumulation of PI(3,4,5)P3. Simultaneous imaging analysis reveals that in the absence of extracellular stimuli, autonomous PI3K and Ras activation occur, concurrently, at the same sites where F-actin projection emerges. The loss of PI3K binding to Ras–guanosine triphosphate abolishes this PI3K activation, whereas prevention of PI3K activity suppresses autonomous Ras activation, suggesting that PI3K and Ras form a positive feedback circuit. This circuit is associated with both random cell migration and cytokinesis and may have initially evolved to control stochastic changes in the cytoskeleton.