Abstract

Talin works as a docking site protein that consists of different focal adhesion protein binding site. It serves as one of the vital protein in focal adhesion formation through its ability in activating integrin. Talin equipt with a unique subunit within its head called F0 subunit. Up until now, this subunit is known by its ability to regulate integrin activation, but the molecular pathway behind it remains elusive. Another “underrated” domain that talin has is R11 which containing a similar protein binding site with the “famous” F3 domain. However, its mechanism to support focal adhesion formation remains unknown. This research project performed to study the important role of both F0 subdomain and R11 domain in focal adhesion formation by observing the deletion effect of each domain in MEF-RPTP and MEF-talin1-/- cell. This research project shows that the deletion of R11 significantly hinders the formation of focal adhesion. On the other hand, the deletion of F0 subdomain does not give any significant impact to focal adhesion and even actin formation.