An emerging concept in biology is that extracellular matrix composition and stiffness contribute to cellular development and activities such as locomotion and transmission of signals. However, the exact molecular mechanisms remain largely unknown. Integrin is a heterodimeric molecule expressed on cell membranes, and is consisted of extracellular head and leg domain, transmembrane domain, and cytoplasmic domain. Interacting with both extracellular matrix and intracellular actin cytoskeleton, integrin has been proposed to be the mechanotransducer involved in stiffness sensing. The action of integrin comprises the dynamic equilibrium of low or high affinity conformation state through various combinations of each subunit domain position: closed or open headpiece, and bent or extended leg. Fibrinogen, together with its polymerized form fibrin, regulates cellular responses during wound repair and tissue remodeling by helping aggregate platelets and form blood clots. This process requires mechanical forces generated by platelets and direct interactions with integrin receptors. Integrin β3 is a widely distributed fibrinogen receptor expressed on platelets. In this research, specific domains of β3 integrins crucial for the conformation change are mutated and their abilities to sense matrices of different stiffness will be examined after the transfected cells attaching to engineered matrix of different stiffness. In order to do so, matrices of different stiffness will be generated by varying the ratio of bis-crosslinker and acrylamide. An immortalized endothelial cell line transduced with wild-type integrin β3 or mutant integrin β3 with rationally designed single mutation will be used. Lastly, the intracellular signaling events after seeding these cells onto matrices of different stiffness will be examined.