Chintala et al. showed that the transfection of the human glioma cell line SNB19 with antisense urokinase-type plasminogen activator (uPAR) resulted in downregulation of uPAR at both the mRNA and protein levels. In this study, we used SNB19 to determine how the presence or absence of uPAR promotes cell spreading and associated changes in cell morphology. Microscopic analysis of cell spreading revealed that antisense uPAR-transfected cells were larger, remained round, and did not spread efficiently over extracellular matrix substrate type IV collagen and fibronectin, unlike parental SNB19 cells, which were smaller and spindle shaped. Biochemical studies showed that antisense uPAR-transfected cells, in addition to not spreading, exhibited increased expression of alpha 3 beta 1 integrin but not alpha 5 beta 1 integrin. However, we could not find a change in the expression of extracellular matrix components or altered growth rate in these cells. Furthermore, despite the increased alpha 3 beta 1 integrin expression, antisense uPAR-transfected cells failed to form an organized actin cytoskeleton when plated on type IV collagen or fibronectin, unlike parental SNB19 cells, which displayed an organized cytoskeleton. These findings show that the absence of uPAR in human glioma cells leads to morphological changes associated with decreased spreading and a disorganized cytoskeleton resulting in altered cell morphology, suggesting that coordinated expression of uPAR and integrin may be involved in spreading of antisense uPAR-transfected glioma cells [1)].

1)
Chintala SK, Mohanam S, Go Y, Venkaiah B, Sawaya R, Gokaslan ZL, Rao JS. Altered in vitro spreading and cytoskeletal organization in human glioma cells by downregulation of urokinase receptor. Mol Carcinog. 1997 Dec;20(4):355-65. PubMed PMID: 9433480.