

Integrin beta 1

Integrin beta-1 (ITGB1), also known as [CD29](#), is a cell surface receptor that in humans is encoded by the ITGB1 gene. This integrin associates with integrin alpha 1 and integrin alpha 2 to form integrin complexes which function as collagen receptors. It also forms dimers with integrin alpha 3 to form integrin receptors for netrin 1 and reelin. These and other integrin beta 1 complexes have been historically known as very late activation (VLA) antigens.

[Stretch injury](#) of the facet capsular [ligament](#) is a cause of [neck pain](#), inducing [axonal injury](#), [neuronal hyperexcitability](#), and upregulation of pain [neuromodulators](#). Although thresholds for [pain](#) and [collagen](#) reorganization have been defined and [integrins](#) can modulate [pain](#) signaling with [joint trauma](#), little is known about the role of integrin signaling in neuronal [dysfunction](#) from tensile loading of the innervated capsular ligament. Using a well-characterized biomimetic collagen gel model of the capsular ligament's microstructure and [innervation](#), a study evaluated extrasynaptic expression of N-Methyl-D-Aspartate receptor subtype 2B ([GRIN2B](#)) as a measure of neuronal dysfunction following tensile loading and determined mechanical thresholds for its [upregulation](#) in primary sensory [neurons](#), with and without integrin inhibition. [Collagen gels](#) with dissociated dorsal root ganglion neurons (n = 16) were fabricated; a subset of [gels](#) (n = 8) was treated with the $\beta 1$ integrin subunit inhibitor, TC-I15. Gels were stretched to failure in tension and then immunolabeled for axonal NR2B. Inhibiting the integrin subunit does not change the failure force (p = 0.12) or displacement (p = 0.44) but does reduce expression of the $\beta 1$ subunit by 41% (p < 0.001) and decrease axonal NR2B expression after stretch (p = 0.018). [Logistic regressions](#) estimating the maximum principal strain threshold for neuronal dysfunction as evaluated by Analysis of Covariance determine that integrin inhibition increases (p = 0.029) the 50th percentile strain threshold (7.1%) above the threshold for upregulation in untreated gels (6.2%). These results suggest that [integrin](#) contributes to stretch-induced neuronal dysfunction via neuron-integrin-collagen interactions ¹⁾

Combined inhibition of the adhesion receptor $\beta 1$ integrin and the stress-mediator c-Jun N-terminal kinase (JNK) induces radiosensitization and blocks invasion in stem-like and patient-derived Glioblastoma cultures as well as in Glioblastoma cell lines. In vivo, this treatment approach not only significantly delays tumor growth but also increases median survival of orthotopic, radiochemotherapy-treated Glioblastoma mice. Both, in vitro and in vivo, effects seen with $\beta 1$ integrin/JNK co-inhibition are superior to the monotherapy. Mechanistically, the in vitro radiosensitization provoked by $\beta 1$ integrin/JNK targeting is caused by defective DNA repair associated with chromatin changes, enhanced ATM phosphorylation and prolonged G2/M cell cycle arrest. Our findings identify a $\beta 1$ integrin/JNK co-dependent bypass signaling for Glioblastoma therapy resistance, which might be therapeutically exploitable ²⁾.

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Singh S, Winkelstein BA. Inhibiting the $\beta 1$ integrin subunit increases the strain threshold for neuronal dysfunction under tensile loading in collagen gels mimicking innervated ligaments. Biomech Model Mechanobiol. 2022 Mar 12. doi: 10.1007/s10237-022-01565-6. Epub ahead of print. PMID: 35279768.

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Vehlow A, Klapproth E, Storch K, Dickreuter E, Seifert M, Dietrich A, Bütof R, Temme A, Cordes N. Adhesion- and stress-related adaptation of glioma radiochemoresistance is circumvented by $\beta 1$ integrin/JNK co-targeting. Oncotarget. 2017 Apr 27. doi: 10.18632/oncotarget.17480. [Epub ahead of

print] PubMed PMID: 28514757.

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