TRIM32 was identified as a p53-regulated gene and an E3 ubiquitin ligase of p53. Thus, TRIM32 and p53 form a novel auto-regulatory negative feedback loop for p53 regulation in cells. [1].

In vitro studies on SH-SY5Y cells indicate that oxidative stress triggers proteasomal degradation of dysbindin-1A, and that this requires interactions with its PEST domain, which may be a TRIM32 target.

Fu et al.identify tripartite motif protein 32 (TRIM32), an E3 ubiquitin ligase, which is barely detected in glial cells in the normal uninjured spinal cord, exhibits strong expression in both astrocytes and microglia following SCI.

They further observe that deficiency of TRIM32 results in increased numbers of astrocytes and microglia, which is accompanied by enhanced proliferation of both cells and increased secretion of interleukin (IL)-1 and IL-10. The Axon regeneration is impaired in the spinal cord of TRIM32-/- mice following SCI, which is indicated by increased distances of the corticospinal tracts (CST) fiber to the lesion site and less axonal sprouting. We further show that deficiency of TRIM32 results in delay motor recovery following SCI. Therefore, TRIM32 is a novel essential positive factor modulating Axon regeneration and the recovery of motor function following SCI, possibly through suppressing proliferation of glial cells [2].

1)
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2)
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