ACTC1

ACTC1 encodes cardiac muscle alpha actin. This isoform differs from the alpha actin that is expressed in skeletal muscle, ACTA1. Alpha cardiac actin is the major protein of the thin filament in cardiac sarcomeres, which are responsible for muscle contraction and generation of force to support the pump function of the heart.

Actins are highly conserved proteins that are involved in various types of cell motility. Polymerization of globular actin (G-actin) leads to a structural filament (F-actin) in the form of a two-stranded helix. Each actin can bind to four others. The protein encoded by this gene belongs to the actin family which is comprised of three main groups of actin isoforms, alpha, beta, and gamma. The alpha actins are found in muscle tissues and are a major constituent of the contractile apparatus. Defects in this gene have been associated with idiopathic dilated cardiomyopathy (IDC) and familial hypertrophic cardiomyopathy (FHC).

ACTC1, could function as a prognostic and predictive marker in clinical treatment of spinal cord injury (SCI) [1)].

ACTC1 may serve as a novel independent prognostic and invasion marker in glioblastoma Glioblastoma [2)].

A study of Wanibuchi et al., from the Department of Neurosurgery, Sapporo Medical University School of Medicine, Hokkaido Japan aimed to clarify whether the knockdown of highly expressed ACTC1 can inhibit the migratory capacity of cells in the Glioblastoma cell line.

ACTC1 expression was examined using immunocytochemistry and droplet digital polymerase chain reaction. The motility of Glioblastoma cells that were either treated with siRNA to knock down ACTC1 or untreated were investigated using a time-lapse study in vitro.

The relatively high ACTC1 expression was confirmed in a Glioblastoma cell line, i.e., U87MG. The ACTC1 expression in U87MG cells was significantly inhibited by ACTC1-siRNA ($p < 0.05$). A cell movement tracking assay using time-lapse imaging demonstrated the inhibition of U87MG cell migration by ACTC1 knockdown. The quantitative cell migration analysis demonstrated that the distance traversed during 72 h was 3607 ± 458 (median ± SD) μm by untreated U87MG cells and 3570 ± 748 μm by negative control siRNA-treated cells. However, the distance migrated by ACTC1-siRNA-treated cells during 72 h was significantly shorter (1265 ± 457 μm, $p < 0.01$) than the controls.

ACTC1 knockdown inhibits U87MG cell migration. [3)].

1)
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3)
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