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Paracrine action of human mesenchymal stem cells for muscle diseases

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The role of Wharton's jelly-derived human mesenchymal stem cells (WJ-MSCs) in inhibiting muscle cell death has been elucidated in the present study. Apoptosis induced by serum-deprivation in mouse myoblast cell lines (C2C12) was significantly reduced when the cell lines were co-cultured with WJ-MSCs in a transwell system. Antibody arrays indicated high levels of chemokine (C motif) ligand (XCL1) secretion by co-cultured WJ-MSCs and XCL1 protein treatment resulted in complete inhibition of apoptosis in serum-starved C2C12 cells. Apoptosis of C2C12 cells and loss of differentiated C2C12 myotubes induced by lovastatin, another muscle cell death inducer, was also inhibited by XCL1 treatment. However, XCL1 treatment did not inhibit apoptosis of cell lines other than C2C12. When XCL1-siRNA pretreated WJ-MSCs were co-cultured with serum-starved C2C12 cells, apoptosis was not inhibited, thus confirming that XCL1 is a key factor in preventing C2C12 cell apoptosis. We demonstrated the therapeutic effect of XCL1 on the zebrafish myopathy model, generated by knock down of a causative gene ADSSL1 encoding a muscle isozyme of adenylosuccinate synthase. The exogenous expression of XCL1 resulted in significant recovery of the zebrafish skeletal muscle defects. These results suggest that human WJ-MSCs and XCL1 protein may act as pro-mixing and novel therapeutic agents for treatment of myopathies and other skeletal muscle diseases.

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