ABSTRACT

Millions of people are affected by tendon overuse. Tendons transfer force from the muscle to the bone. Tenocytes have gap junctions, which are communication portals, and these gap junctions play a role during mechano-transduction. They can respond to mechano-transduction through cell-cell and cell-ECM interaction. Hereby tenocytes control ECM synthesis and degeneration, which maintains tissue homeostasis. If these junctions are disrupted, tissue homeostasis can no longer be maintained, which can result in ECM degeneration and loss of tissue anisotropy. Current treatment of injured tendons is often surgery, but this treatment associates with a lot of complications. A new approach could be to restore the loss of tendinopathy-induced gap junction functionality and thereby stimulate the regeneration of tendons. One question that remains is whether and how gap junctions influence functional remodelling of disorganized (isotropic) tissue. Therefore, the amount of tissue remodelling by tenocytes in in vitro (isotropic) tissue was measured in the presence or absence of functional gap junctions. Tenocytes were embedded in collagen gels and cultured on in vitro tissue platforms that enable tissue remodelling upon perturbation. Reduction in surface area of the tissues and the alignment of actin were taken as measures for remodelling. Findings suggest that inactivating gap junctions reduces remodelling capacity, as assessed by quantifying the change in tissue surface area and cellular stress fibre (f-actin) alignment.

GRAPHICAL ABSTRACT

The tissue platforms showing tissue contraction. In the left picture an unperturbed tissue can be seen. In the middle perturbed tissue with inhibition of active gap junctions can be seen and the picture on the right shows unperturbed tissue with active gap junctions.