Aquaculture is an expanding worldwide industry producing an increasing amount of fish every year. The quality of the fish meat is dependent upon many biological and non-biological factors. Infectious diseases are known to cause bleedings and damage of the muscle tissue that may lead to scarring after recovery, which possibly will affect some quality parameters. However, until now it has not been examined if previous infections have an impact on the sensory characteristics of the meat after slaughter. Further, the underlying molecular mechanisms involved in regeneration of muscle tissue are poorly described in fish. The present work in this thesis focused on: 1) examination of potential changes in the quality regarding texture of the muscle tissue in rainbow trout (Oncorhynchus mykiss) after previous infection with the bacterial pathogens Yersinia ruckeri and Vibrio anguillarum; 2) characterisation of potential immune functions of fibroblasts and the importance of this in relation to tissue regeneration; 3) creation of a model to study local, sterile tissue damage in the muscle tissue of rainbow trout and comparison of this to infection of Atlantic salmon (Salmo salar) by the bacterium Moritella viscosa, the causative agent of ‘winter ulcer’ in Norway. In order to reach these objectives, sensory analysis and ELISA was used. Further, quantitative real-time RT-PCR was used in order to measure the expression of genes coding for immunological factors and tissue regeneration. The results of these studies showed that previous infections by Yersinia ruckeri and Vibrio anguillarum gave rise to subsequent changes regarding textural quality parameters in fresh fish meat, while no differences were seen for cold-smoked meat from the same fish. The texture in previous infected fish was less flaky and less oily, but had a higher toughness and fibrousness in comparison to control fish. These observations could be explained from the molecular studies. Herein, local inflammatory responses characterised by up-regulation of IL-1β and IL-8 was seen in mechanically damaged and M. viscosa infected fish. However, this response was much stronger in infected versus damaged fish, indicating that damaged cells initiate an immune response, but pathogenic triggering was much more potent. A further activation of the genes TGF-β, MMP-2, CTGF and myostatin-1αβ was then seen in both groups, indicating initiation of tissue regeneration. Likely as a consequence of heavier tissue damage in infected fish, the collagen-1α gene was induced in this group. Scarring or fibrosis is equal to deposition of collagen in repaired tissue. Hence, these data may explain the sensory observations from previously infected fish. Finally, the fibroblast cell-line RTHDF from rainbow trout was found to be an immune competent cell type. This was examined since a fast up-regulation of IL-1β and IL-8 shortly after tissue damage was observed in muscle tissue from rainbow trout. These observations led to the suggestion that local resident cells in the muscle tissue are the first to initiate an inflammatory response following tissue damage. The RTHDF cell-line was found to be responsive to LPS from the surface of gram-negative bacteria, but also from damaged RTHDF cells. Hence, the data supported that theory.