Development of muscle regeneration strategies targeting novel immune cell populations directing muscle repair

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Musculoskeletal diseases such as fractures, sarcopenia and joint diseases account for 25% of the causes of need for assistance and care for the elderly. Age-related deterioration of musculoskeletal functions increases the frequency of skeletal muscle injuries in daily life. Thus, the development of medical technology to enhance the tissue repair capability of the components of locomotory organs such as bones, joints and muscles is an important issue. We have previously found that IL-17-producing $\gamma\delta$ T cells accumulating around injured bone tissue trigger bone regeneration by promoting the proliferation of mesenchymal progenitor cells and their osteogenesis (Ono, Okamoto, et al, Nat Commun, 2016). We have recently found that a novel macrophage subpopulation is capable of inducing skeletal muscle regeneration. It is suggested that an approach targeting the newly indentified macrophage subset that induce skeletal muscle repair may be effective for skeletal muscle regeneration. In this study, we sought to elucidate the mechanisms how the macrophage subset develops after skeletal muscle injury, and how the macrophage subset regulates skeletal muscle regeneration, to provide a molecular basis for the development of novel skeletal muscle regeneration therapies.