Diabetes has a wide range of negative effects on skeletal muscle, including a loss of muscle mass for a number of reasons. This study demonstrated that diabetes does not affect all the muscles in your body equally. In fact, some muscles, such as the soleus muscle (which is used for postural support and is composed of slow-twitch fibers) appears to be resistant to many of the negative effects of diabetes (e.g. impaired repair from damage) while other muscles are badly affected. Muscles affected included the tibialis anterior and gastrocnemius, both composed of fast-twitch fibers. We then went on to demonstrate that the reason for the impaired ability to regenerate from injury was the result of poor inflammatory cell influx. Normally, inflammatory cells should arrive in high number following injury, but this was limited in muscles from diabetic mice. Interestingly, a hormone that is highly elevated in diabetes, Plasminogen Activator Inhibitor (PAI)-1, appears to be a master regulator of this poor injury/inflammatory response. This is exciting because PAI-1 has also been found to be part of the pathophysiology of diabetes-related kidney and cardiovascular complications by much the same mechanism.

Reference: Krause MP, Al-Sajee D, D'Souza DM, Rebalka IA, Moradi J, Riddell MC, Hawke TJ. Impaired macrophage and satellite cell infiltration occurs in a muscle-specific fashion following injury in diabetic skeletal muscle. PLoS One. 2013 Aug 12;8(8):e70971.

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