Our muscles do more than help us move around. Because they are a main engine for the metabolism of glucose, protein and lipids, alterations to muscle metabolism have implications for various metabolic diseases. Defects in skeletal muscle metabolism and/mass underlie or worsen diseases like diabetes, cancer and HIV. In fact, for some of these diseases, skeletal muscle mass is an independent predictor of length of stay in hospital and survival. It is not surprising therefore that various mechanisms and pathways regulate the mass and metabolism of this tissue.

In this paper, Adegoke et al reviewed recent findings on a protein complex, mammalian target of rapamycin 1 (mTORC1). This complex regulates muscle mass, in part by regulating muscle protein synthesis. However, it has a dark side: activation of the complex is implicated in cancer and insulin resistance, a condition that predisposes to type 2 diabetes and cardiovascular disease. This presents an interesting conundrum, as extensive muscle wasting or cancer cachexia seen in many cancers may be improved by stimulating anabolic pathways mediated by mTORC1. This review discusses recent findings on how mTORC1 is regulated. It also examines possible approaches, including resistance exercise, customized nutrition and tissue specific drug targeting, to harness its anabolic effects in muscle while limiting undesirable effects in other tissues.

Reference: **Adegoke OA**, Abdullahi A, Tavajohi-Fini P. mTORC1 and the regulation of skeletal muscle anabolism and mass. Appl Physiol Nutr Metab. 2012 Jun;37(3):395-406.

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