We previously showed that L-lysine (Lys) and a metabolite of Lys, L-saccharopine, suppressed autophagic proteolysis in C2C12 myotubes. However, the effects of other metabolites of Lys on protein turnover were unknown. We here investigated the effect of the Lys metabolites, L-2-aminoadipic acid (2-AA) and L-pipecolic acid (Pip), on protein turnover in C2C12 myotubes. 2-AA suppressed myofibrillar protein degradation evaluated by the 3-methylhistidine and autophagy activity evaluated by light chain 3-II at lower concentration (100 μM) than did Lys. On the other hand, Pip stimulated the mammalian target of rapamycin signaling activity. Additionally, 100 μM Pip significantly increased the rates of protein synthesis whereas 100 μM Lys had no effect. These results indicate that in C2C12 myotubes, 2-AA could suppress autophagy and Pip could stimulate the rates of protein synthesis, and these metabolites may contribute to exert effect of Lys on protein turnover.

L-lysine metabolites, L-2-aminoadipic acid and L-pipecolic acid, have regulatory effects on proteolysis and protein synthesis.