An isoflavone compound Daidzein elicits myoblast differentiation and myotube growth

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The reduced regenerative capacity of muscle stem cells contributes to aging or disease-related skeletal muscle atrophy. Daidzein, a natural isoflavone from Leguminosae, improves insulin sensitivity in skeletal muscle and prevents TNF-α induced muscular atrophy. However, the molecular mechanisms by which daidzein exerts these beneficial effects are currently unknown. In this study, we determined the effect and molecular mechanisms by which daidzein might improve skeletal muscle function. Similarly to the results with TNF-α, daidzein treatment prevented myotube atrophy triggered by Dexamethasone (DEX) via Akt activation. Furthermore, daidzein promoted myoblast differentiation in a dose-dependent manner through activation of promyogenic kinases, Akt and p38MAPK. Daidzein treatment strengthened MyoD activation by enhancing its heterodimer formation with E protein. Additionally, daidzein induced myotube growth, through activation of Akt/mTOR/S6K pathway. Moreover, daidzein increased MyoD-dependent myogenic conversion of fibroblast and myogenic differentiation. Taken together, daidzein has a potential as a therapeutic or nutraceutical remedy to improve muscle function and to treat aging or disease-related muscle loss and weakness.

Biography
Gyu-Un Bae has completed his PhD from Sungkyunkwan University and Postdoctoral studies from Harvard University, School of Medicine. He has published more than 54 papers in reputed journals and has been serving as an Editorial Board Member of Archives of Pharmacal Research.

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