論 文 要 旨

Chorein interacts with α-tubulin and histone deacetylase 6, and overexpression preserves cell viability during nutrient deprivation in human embryonic kidney 293 cells

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The autophagy pathway has recently been implicated in several neurodegenerative diseases. Recently, it was reported that chorein-depleted cells showed accumulation of autophagic markers and impaired autophagic flux. Here, we demonstrate that chorein overexpression preserves cell viability from starvation-induced cell death in human embryonic kidney 293 (HEK293) cells. Subsequent coimmunoprecipitation and reverse coimmunoprecipitation assays using extracts from chorein that stably overexpressed HEK293 cells revealed that chorein interacts with α-tubulin and histone deacetylase 6, a known α-tubulin deacetylater and central component of basal autophagy. Indeed, acetylated α-tubulin immunoreactivity was significantly decreased in chorein that stably overexpressed HEK293 cells. These results suggest that chorein/histone deacetylase 6/ α-tubulin interactions may play an important role in starvation-induced cell stress, and their disruption may be one of the molecular pathogenic mechanisms of chorea-acanthocytosis.