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Association of a Novel Hsp70 Species with Brain Aging and Proteasome Dysfunction

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Abstract

Most neurological diseases are characterized by the presence of protein aggregates, suggesting that aberrations in protein homeostasis are associated with neuronal demise. In eukaryotic cells, protein homeostasis is maintained by the chaperone, ubiquitin proteasome (UPS) and autophagy systems. As age is a risk factor for several types of neurodegenerative diseases, the function of these various protein homeostatic systems could become compromised with age. To understand the events that occur during normal aging, we examined the expression of key markers associated with the aforementioned systems in mice aged 1, 3, and >18 months. We found that proteasome activity and the amount of proteasome-related structures remained unaffected with age. Interestingly though, an age-related increase of a novel Hsp70 chaperone protein species (herein designated Hsp70*) was observed. The expression of Hsp70* is also increased markedly in cells treated with pharmacological agents that promote proteasome inhibition, suggesting a functional interaction between the chaperone system and the UPS. Taken together, our results suggest that there is some form of crosstalk between the chaperone system and the UPS involving the observed HSP70 species.