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Heat shock protein 70 is translocated to lipid droplets in rat adipocytes upon heat stimulation.

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Abstract

In mammalian cells, lipid storage droplets contain a triacylglycerol and cholesterol ester core surrounded by a phospholipid monolayer into which a number of proteins are imbedded. These proteins are thought to be involved in modulating the formation and metabolic functions of the lipid droplet. In this study, we show that heat stress upregulates several heat shock proteins (Hsps), including Hsp27, Hsp60, Hsp70, Hsp90, and Grp78, in primary and differentiated adipocytes. Immunostaining and immunoblotting data indicate that among the Hsps examined, only Hsp70 is induced to redirect to the lipid droplet surface in heat-stressed adipocytes. The thermal induction of Hsp70 translocation to lipid droplet does not typically happen in a temperature- or time-dependent manner and occurs abruptly at 30-40 min and rapidly achieves a steady state within 60 min after 40 degrees C stress of adipocytes. Though Hsp70 is co-localized with perilipin on the lipid droplets in stressed adipocytes, immunoprecipitation experiments suggest that Hsp70 does not directly interact with perilipin. Alkaline treatments indicate that Hsp70 associates with the droplet surface through non-hydrophobic interactions. We speculate that Hsp70 might noncovalently associate with monolayer microdomains of the lipid droplet in a manner similar to its interaction with lipid bilayer moieties composed of specific fatty acids. As an acute and specific cellular response to the heat stimulation, accumulation of Hsp70 on adipocytes lipid droplets might be involved in stabilizing the droplet monolayer, transferring nascent proteins to the lipid droplets, or chaperoning denatured proteins on the droplet for subsequent refolding.

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