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Corrigendum to "The chaperone action of bovine milk aS1- and aS2-caseins and their associated form aS-casein" [Arch. Biochem. Biophys. 510 (2011) 42–52]

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Corrigendum to "The chaperone action of bovine milk α_{S1}- and α_{S2}-caseins and their associated form α_S-casein" [Arch. Biochem. Biophys. 510 (2011) 42–52]

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The authors regret that there were typographical errors in the abstract of their paper. The correct abstract is inserted below.

Abstract

α_S-Casein, the major milk protein, comprises α_{S1}- and α_{S2}-casein and acts as a molecular chaperone, stabilizing an array of stressed target proteins against precipitation. Here, we report that α_S-casein acts in a similar manner to the unrelated small heat-shock proteins (sHsps) and clusterin in that it does not preserve the activity of stressed target enzymes. However, in contrast to sHsps and clusterin, α_S-casein does not bind target proteins in a state that facilitates refolding by Hsp70. α_S-Casein was also separated into α_{S1}- and α_{S2}-casein, and the chaperone abilities of each of these proteins were assessed with amorphously aggregating and fibril-forming target proteins. Under reduction stress, all α_S-casein species exhibited similar chaperone ability, whereas under heat stress, α_{S1}-casein was a poorer chaperone. Conversely, α_{S2}-casein was less effective at preventing fibril formation by modified κ-casein, whereas α_S- and α_{S1}-casein were comparably potent inhibitors. In the presence of added salt and heat stress, α_{S1}-, α_{S2}- and α_S-casein were all significantly less effective. We conclude that α_{S1}- and α_{S2}-casein stabilise each other to facilitate optimal chaperone activity of α_S-casein. In general, this work highlights the interdependency of casein proteins for their structural stability *in vivo* and the importance of casein–casein interactions, as present in the casein micelle, in the prevention of amyloid fibril formation.