ERRATUM

A nonessential role for Arg 55 in cyclophilin18 for catalysis of proline isomerization during protein folding

DOI: 10.1002/pro.129
Published online 26 May 2009 proteinscience.org

Moparthi SB, Hammarström P, Carlsson U. (2009) Protein Sci 18:475–479. In this article the cited comparison (Ref. 21) that the mitochondrial cyclophilin Cpr3 with a homologous active-site R73A mutation retained its prolyl isomerase activity towards a protein substrate[1] is not valid, since the activity has been reported to originate from a contamination of an E. coli protein SlyD[2]. Thus, human cyclophilin18 is so far the only cyclophilin that has been shown to be able to catalyze prolyl isomerization during protein folding without Arg55 that is essential for smaller peptide substrates.

1. Scholz C, Schindler T, Dolinski K, Heitman J, Schmid FX (1997) Cyclophilin active site mutants have native prolyl isomerase activity with a protein substrate. FEBS Lett 414:69–73.

2. Scholz C, Maier P, Dolinski K, Heitman J, Schmid FX (1999) R73A and H144Q mutants of the yeast mitochondrial cyclophilin Cpr3 exhibit a low prolyl isomerase activity in both peptide and protein-folding assays. FEBS Lett 443:367–369.

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