The heat shock protein ClpC affects the intracellular survival capacity of *Staphylococcus aureus* in endothelial cells

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The ability of *Staphylococcus aureus* to invade non-professional phagocytic cells and to persist intracellularly for longer periods of time is thought to be one of the major reasons for the pathogens capacity to cause chronic and relapsing infections. Here we present data indicating that the Clp ATPase ClpC, a member of the heat shock protein 100 family, attenuates the intracellular survival capacity of *S. aureus*. Inactivation of *clpC* in the biofilm forming *S. aureus* isolate DSM20231 significantly enhanced the intracellular long-term survival capacity of mutant cells within human endothelial cells, without affecting the adhesion, invasion, and small colony variant formation rates. Transcriptional analyses of total RNAs isolated from intracellular DSM20231 and isogenic *clpC* mutant cells identified alterations in transcription of α-toxin (*hla*), protein A (*spa*), and *RNAIII* (the effector molecule of the *agr* locus), suggesting that ClpC negatively affects the intracellular survival capacity of *S. aureus* in endothelial cells via the transcriptional modulation of the virulon.