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2.	Record Nr.	UNINA9910136401003321
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	Sommario/riassunto	Stress proteins or heat-shock proteins (HSP) are evolutionary conserved proteins present in every prokaryotic and eukaryotic cell. Their main function is to protect cells and proteins from damage under stressful

circumstances. The latter circumstances do include the cell and protein damaging effects of inflammation. The discovery of mycobacterial HSP60 being a critical antigen in the model of adjuvant arthritis, has led to studies that showed the immuno-dominance of microbial HSP60 and the potential of the microbial HSP induced repertoire of antibodies and T cells to cross-recognize the self-HSP homologues of stressed cells. Since then, the research in the immunology of stress proteins started to comprise a widening spectrum of topics with potential medical relevance. Interestingly, since stress proteins have their activities in both innate and adaptive immunity, they are key elements in the cross-roads between both arms of the immune system. Stress proteins or HSP can be considered as functional 'biomarkers' of inflammation. They are up-regulated locally during inflammation and interestingly, they seem to function as targets for anti-inflammatory regulatory T cells. In experimental models of autoimmunity, mainly arthritis, administration of HSP peptides have been shown to suppress disease. First clinical trials have shown the anti-inflammatory nature of T cell responses to Hsp. In type I diabetes and in rheumatoid arthritis, parenteral and oral administration of Hsp peptides were shown to induce a bias in pro-inflammatory T cells, switching them in the direction of regulatory cytokine production (IL4, IL5 and IL10). In addition a raised level of a marker of natural T regulatory cells, the transcription factor FoxP3, was noted in the RA trial. Other inflammatory diseases or diseases with inflammatory components which feature the immune imprint of the up-regulated Hsp are atherosclerosis, inflammatory bowel diseases, multiple sclerosis and atopic diseases such atopic dermatitis and allergic asthma.
