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The ubiquitin proteasome system (UPS) plays a crucial role in many critical cellular processes, frequently by not only mediating the selective degradation of constituent proteins but also by playing non-degradative role especially important as in many signalling pathways. Over the last three decades mainly, accumulated evidence indicated that UPS proteins are primal modulators of cell cycle progression, DNA replication and repair, transcription, immune responses, and apoptosis.

Comparatively, more recently involvement of UPS proteins and its impairment in cardiovascular physiology and pathophysiology including ischemia, hypertrophy and heart failure has been reported. Recent studies have demonstrated a remarkable level of complexity in the regulation of the UPS in the heart and have identified various UPS proteins playing a part in the development of cardiomyopathies. Recent emergence of the roles of TNF receptor-associated factor (TRAF) subfamily of proteins and the proteins of Ubiquitin Specific Proteases (USP or DUBs) superfamily has increased our repertoire.

However, our understanding of how UPS dysfunction might contribute to the pathophysiology of and the complete list of UPS component proteins regulating such a wide range of cardiac afflictions is still very limited. This review focuses on the addition of TRAF proteins and USP proteins to the list of other known E3 ligases likes of MURF1, Atrogin-I and MDM2 that are specific to cardiac hypertrophy and highlights the expression profile of these proteins in cardiac hypertrophy that can serve as opportunity for the development of novel therapies.