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Sommario/riassunto	The calcium-calmodulin dependent protein kinases (CaMKs) are a broadly expressed family of calcium-sensitive intracellular kinases, which are responsible for transducing cytosolic calcium signals into phosphorylation-based regulation of proteins and physiological functions. As the multifunctional member of the family, CaMKII has become the most prominent for its roles in the central nervous system and heart, where it controls a diverse range of calcium-dependent processes; from learning and memory at the neuronal synapse, to cellular growth and death in the myocardium. In the heart, CaMKII directly regulates many of the most important ion channels and calcium handling proteins, and controls the expression of an ever-increasing number of transcripts and their downstream products. Functionally, these actions are thought to orchestrate many of the electrophysiologic and contractile adaptations to common cardiac stressors, such as rapid pacing, chronic adrenergic stimulation, and oxidative challenge. In the context of disease, CaMKII has been shown to contribute to a remarkably wide variety of cardiac pathologies, of which Heart failure (HF) is the most conspicuous. Hyperactivity of CaMKII is an established contributor to pathological cardiac remodelling, and is widely thought

to directly promote arrhythmia and contractile dysfunction during HF. CaMKII is also ubiquitous in non-failing arrhythmia-susceptible phenotypes, several of which result from specific channelopathies that mimic constitutive channel phosphorylation. Because CaMKII contributes to both the acute and chronic manifestations of major cardiac diseases, but may be only minimally required for homeostasis in the absence of chronic stress, it has come to be one of the most promising therapeutic drug targets in cardiac biology. Thus, development of more specific and deliverable small molecule antagonists remains a key priority for the field. Here we provide a selection of articles to summarize the state of our knowledge regarding CaMKII in cardiac health and disease, with a particular view to highlighting recent developments in CaMKII activation, and new targets in CaMKII-mediated control of myocyte physiology.