1.	Record Nr.	UNINA9910688341403321
	Autore	Giuseppe Rengo
	Titolo	The adrenergic system in cardiovascular physiology and pathophysiology [[electronic resource] /] / edited by: Giuseppe Rengo
	Pubbl/distr/stampa	Frontiers Media SA, 2015
		[Lausanne, Switzerland] : , : Frontiers Media SA, , 2015 ©2015
	Edizione	[Second edition.]
	Descrizione fisica	1 online resource (78 pages) : illustrations; digital, PDF file(s)
	Collana	Frontiers Research Topics Frontiers in Physiology
	Soggetti	Cardiovascular system - Diseases Cardiovascular system - Diseases - Pathogenesis Cardiovascular system - Diseases - Prevention - Research
	Lingua di pubblicazione	Inglese
	Formato	Materiale a stampa
	Livello bibliografico	Monografia
	Nota di bibliografia	Includes bibliographical references.
	Sommario/riassunto	Cardiovascular diseases pose an enormous clinical challenge, remaining the most common cause of death in the world adrenoceptors play an important role on cardiac, vascular and/or endothelial function at a cellular level with relevant applications in several cardiovascular diseases, such as heart failure and hypertension. G protein–coupled receptors (GPCRs), including -adrenergic receptors, constitute the most ubiquitous superfamily of plasma membrane receptors and represent the single most important type of therapeutic drug target. Sympathetic nervous system hyperactivity, which characterizes several cardiovascular diseases, such as heart failure and hypertension, as well as physiological ageing, has been proved to exert in the long-term detrimental effects in a wide range of cardiovascular diseases. Acutely, sympathetic hyperactivity represents the response to an insult to the myocardium, aiming to compensate for decreased cardiac output. This process involves the activation of beta-adrenergic receptors by catecholamine with consequent heart rate and cardiac contractility increase. However, long-term exposure of the heart to

elevated norepinephrine and epinephrine levels, originating from sympathetic nerve endings and chromaffin cells of the adrenal gland, results in further progressive deterioration in cardiac structure and function. At the molecular level, sustained sympathetic nervous system hyperactivity is responsible for several alterations including altered beta-adrenergic receptor signaling and function (downregulation/desensitization). Moreover, the detrimental effects of catecholamine affect also the function of different cell types including, but not limited to, endothelial cells, fibroblasts and smooth muscle cells. Thus, the success of beta-blocker therapy is due, at least in part, to the protection of the heart and the vasculature from the noxious effects of augmented catecholamine levels. The current research topic aims to support the progress towards understanding the role of sympathetic nervous system under physiological conditions, and the contribution of its hyperactivity in the pathogenesis and progression of cardiovascular diseases. The topic is open to original studies, descriptions of new methodologies, reviews and opinions.