Publications Template

#	Research Title	Field	Abstract	Year of Publication Publishing	Publishing Link "URL"
1	Telmisartan and captopril ameliorate pregabalin-induced heart failure in rats	Pharmacology and Toxicology	Pregabalin (PRG) is highly effective in the treatment of epilepsy, neuropathic pain and anxiety disorders. Despite its potential benefits, PRG administration has been reported to induce or exacerbate heart failure (HF). It has been previously documented that overactivation of the renin angiotensin system (RAS) is involved in HF pathophysiological mechanism. The target of the current study was to examine the possible cardioprotective effect of telmisartan (Tel), an angiotensin II type 1 receptor (AT1R) blocker, compared with that of captopril (Cap), an angiotensin converting enzyme (ACE) inhibitor, in ameliorating PRG-induced HF in rats by assessing morphometric, echocardiographic and histopathological parameters. Furthermore, to investigate the role of RAS blockade by the two drugs in guarding against PRG-induced changes in cardiac angiotensin 1-7 (Ang 1-7) and angiotensin II (Ang II) levels, in addition to myocardial expression of ACE2, ACE, Mas receptor (MasR) and AT1R. Results showed that PRG administration induced morphometric, echocardiographic and histopathological deleterious alterations and significantly elevated cardiac Ang	2019	https://www.sciencedirect.com/science/article/abs/pii/S0300483X19302677

			II, ACE and AT1R levels, while reduced Ang 1-7, ACE2 and MasR cardiac levels. Concurrent treatment with either Tel or Cap reversed PRG-induced morphometric, echocardiographic and histopathological abnormalities and revealed prominent protection against PRG-induced HF via downregulation of ACE/Ang II/AT1R and upregulation of ACE2/Ang 1-7/MasR axes. These are the first findings to demonstrate that the potential benefits of Tel and Cap are mediated by counteracting the altered balance between the RAS axes induced by PRG. Hence; Tel and Cap may attenuate PRG-induced HF partially through stimulation of ACE2/Ang 1-7/MasR pathway.		
2	Assessment of pregabalin-induced cardiotoxicity in rats: mechanistic role of angiotensin 1-7	Cardiovascular Toxicology	Pregabalin (PRG) possesses great therapeutic benefits in the treatment of epilepsy, neuropathic pain and fibromyalgia. However, clinical data have reported incidence or exacerbation of heart failure following PRG administration. Experimental data exploring cardiac alterations and its underlying mechanisms are quite scarce. The aim of the present work was to investigate the effect of PRG on morphometric, echocardiographic, neurohumoral and histopathological parameters in rats. It was hypothesized that alterations in cardiac renin angiotensin system (RAS) might be involved in PRG-induced cardiotoxicity. To further emphasize the role of RAS in the mechanism of PRG-induced cardiotoxicity, the protective potential of diminazene aceturate (DIZE), an ACE2 activator, was investigated. Results showed 44% decrease in ejection	2020	https://link.springer.com/article/10.1007/s12012-019-09553-6

			fraction and 7-fold increase in plasma N-terminal pro-brain natriuretic peptide. Histopathological examination also showed prominent vacuolar changes and edema in cardiomyocytes. In addition, PRG significantly increased angiotensin II (Ang II), angiotensin converting enzyme (ACE) and angiotensin II type 1 receptor (AT1R) levels, while decreased angiotensin 1-7 (Ang 1-7), angiotensin converting enzyme 2 (ACE2) and Mas receptor (MasR) cardiac levels. DIZE co-administration showed prominent protection against PRG-induced echocardiographic, neurohumoral and histopathological alterations in rats. In addition, downregulation of ACE/Ang II/AT1R and upregulation of ACE2/Ang 1-7/MasR axes were noted in DIZE co-treated rats. These findings showed, for the first time, the detailed cardiac deleterious effects of PRG in rats. The underlying pathophysiological mechanism is probably mediated via altered balance between the RAS axes in favor to the ACE/Ang II/AT1R pathway. Accordingly; ACE2 activators might represent promising therapeutic agents for PRG-induced cardiotoxicity.		