Publications Template

#	Research Title	Field	Abstract	Year of Publication Publishing	Publishing Link "URL"
1	Potential cardioprotective effect of octreotide via NOXs mitigation, mitochondrial biogenesis and MAPK/Erk1/2/STAT3/NF-kβ pathway attenuation in isoproterenol-induced myocardial infarction in rats. European journal of Pharmacology.	Pharmacology	Myocardial infarction (MI) is a global health care problem, which instigates irreversible cardiac tissue damage and sudden death, necessitating new prevention and management strategies. Hence, the cardioprotective effect of octreotide in MI was scrutinized by tackling the possible underlying trajectories involved. Isoproterenol (ISO)-induced acute MI model was adopted using ISO (85 mg/kg/day, S.C.) for 2 days. Rats in octreotide groups were pretreated with 20 or 40 μg/kg/day S.C. for 8 days and ISO was given on the 7th and 8th days. Octreotide showed a restoration of ECG changes, cardiac hemodynamics abnormalities, serum cardiac markers elevation (creatine kinase MB, troponin I, lactate dehydrogenase, and aspartate aminotransferase), and cardiac histoarchitecture abnormalities. In addition, octreotide pretreatment showed a significant increase in the cardiac and serum level of the diagnostic microRNA-133a. Octreotide attenuates oxidative stress indices (MDA, GSH, SOD, TAC, and HIF-1α), besides a better adjustment of NOX-1/-2/-4 expression and protein levels. Mitochondrial morphology and mtDNA copy number were preserved following the pre-treatment of Octreotide. The inflammatory pathway p38 MAPK/Erk-1/-2/p-STAT3/NF-κB pathway and the proinflammatory cytokines (TNF- α, IL-6, and IL- 1β) were attenuated. The proapoptotic markers (cyt *c*, caspase-3/-9, and Bax) were also attenuated and the antiapoptotic Bcl2 marker was increased by the preadministration of octreotide. In almost all parameters, Octreotide 40 μg/kg/day was more prominent than its lower dose. Octreotide possesses dose-dependent cardioprotective properties via its antioxidant, anti-inflammatory, and anti-apoptotic capabilities.	2022	https://doi.org/10.1016/j.ejphar.2022.174978

2	Thymoquinone dose-dependently attenuates myocardial injury induced by isoproterenol in rats via integrated modulations of oxidative stress, inflammation, apoptosis, autophagy, and fibrosis.	Pharmacology	As rats develop myocardial infarction (MI) like lesions when injected with large doses of isoproterenol (ISO), this investigation was designed to evaluate the dose-dependent effects of thymoquinone (TQ) on ISO-induced myocardial injury in rats. Adult male rats were divided into negative control, TQ20 (20 mg/kg/day), TQ50 (50 mg/kg/day), ISO positive control, TQ20 + ISO, and TQ50 + ISO groups. In these rats, biochemical, immunobiochemical, and histopathological studies were carried out to evaluate myocardial oxidative stress, inflammation, apoptosis, fibrosis, and autophagy, and the changes in serum cardiac biomarkers. The results showed that TQ pretreatment in ISO-administered rats produced a dose-dependent significant reduction of the myocardial infarct size, markedly reduced the ISO-induced elevation in serum cardiac markers and demonstrated several other important findings related to the cardioprotective efficacy of TQ. First, this study is the first reported research work showing that TQ treatment could increase the myocardial reduced glutathione baseline level, adding an indirect antioxidant effect to its known direct free radical scavenging effect. Second, pretreatment with TQ significantly reduced the markers of myocardial oxidative stress, inflammation, fibrosis, and apoptosis. Third, TQ acted as an autophagy enhancer ameliorating myocardial cell damage and dysfunction. Thus, the morphological and biochemical changes associated with ISO-induced myocardial injury were ameliorated with TQ pretreatment. The extent of this improvement was significantly greater in the TQ50 + ISO group than in the TQ20 + ISO group. The present study, for the first time, demonstrates these dose-dependent effects of TQ in experimentally induced myocardial injury. These findings raise the possibility that TQ may serve as a promising prophylactic cardioprotective therapy for patients who are at risk of developing myocardial injury and against the progression of existent myocardial injury as in cases of MI	2021	https://pubmed.ncbi.nlm.nih.gov/34216225/
	The Role Of Anticoagulants And Antiplatelets In Prophylaxis And/Or Treatment Of Severe SARS-COV-2 Infection..	Pharmacology	Severe acute respiratory syndrome coronavirus-2(SARAS-COV-2) was reported firstly in China by the end of 2019 then disseminated vigorously worldwide and in 2020 reported by WHO as pandemic disease. It is associated by many symptoms, however; high incidence of thrombotic events was strongly correlated with SARAS-COV-2. Exploring anticoagulants to be added as thromboprophylaxis for Covid 19 patients	2021	https://doi.org/10.22270/ujpr.v6i1.540

			become a must. Many options for thromboprophylaxis are available including anticoagulants, antiplatelets and fibrinolytics which were illustrated in this mini review.		
3	Covid-19: Pharmacological and Therapeutic Approaches	Pharmacology	In the end of 2019, SARS-CoV-2, a new virus from Corona viruses family, has been detected in China and was responsible for COVID-19 disease. This disease has been suddenly and vigorously disseminated among individuals all over the world. Based on genetic vicinity, this novel virus is similar to SARS-CoV and MERS-CoV and it can spread from an unknown animal host to individuals. Many published clinical data and *in vitro* studies may offer treatment strategies of some effective antiviral and repurposed drugs, including remdesivir, favipiravir, lopinavir/ritonavir, corticosteroids, etc. This narrative review describes current pharmacological proposed treatments for COVID-19 patients and available experimental and clinical studies for these drugs. Eventually, these data may help to explain the most preferable way to treat COVID-19 and lessen the accompanied symptoms and complications.	2021	http://ujpr.org/index.php/journal/article/view/514
4	Effect of Thymoquinone on Mitochondrial DNA Together With Oxidative Stress, Inflammation and Apoptosis in Isoproterenol-Induced Myocardial Infarction Model**.**	Pharmacology	Myocardial infarction (MI) is an ischemic life-threatening disease with exaggerated oxidative stress state that vigorously damages the cardiomyocyte membrane and subcellular structures, including the vital mitochondrial DNA (mtDNA). The mtDNA is responsible for the proper functionality of the mitochondria, which are abundant in cardiomyocytes due to their dynamic nature and energy production requirements. Furthermore, oxidative stress triggers an inflammatory cascade and eventual apoptosis, which exacerbates cardiac injuries and dysfunction.The present study used an isoproterenol (ISP)-induced MI rat model to investigate the role of the main active constituent of Nigella Sativa seeds, thymoquinone (TQ), in preserving the cardiac mtDNA content and ameliorating oxidative stress, inflammation, and apoptosis.Rats in the (TQ + ISP) group were pre-treated with TQ (20 mg/kg/day) for 21 days before the MI induction using ISP (85 mg/kg/day). In addition, negative control and ISP groups were included in the study for comparison. A histopathological examination was performed and serum cardiac parameters (cTnI and LDH) were assessed. In addition, mtDNA content, oxidative stress parameters (MDA, GSH, SOD, GPx, and CAT), inflammatory mediators (IL-6, IL-1β, and TNF-α), and apoptosis markers (BAX, Bcl2, and caspase-3) were detected.The results	2020	https://academic.oup.com/eurheartjsupp/issue/22/Supplement_Q

			showed that pre- and co-treatment with TQ in the (TQ + ISP) group reversed the histoarchitecture changes, caused a significant decrease in serum cardiac markers, oxidative stress markers, inflammatory cytokines, the apoptosis process, and preserved the cardiac mtDNA content.TQ is a cardioprotective agent with an extended effect on preserving the cardiac mtDNA content, in addition to its powerful antioxidant, anti-inflammatory, and anti-apoptotic action.		
5	Most Important Cellular Changes Involved In Renal Ischemia Reperfusion Injury And The Consequent Impact On Selected Remote Organs	Pharmacology	Because of the high rate of baseline oxygen use by renal cells, kidney is highly influenced by obstruction of arterial blood inflow and subsequent shortage of the received oxygen, this condition is known as Ischemic injury. There are many clinical settings associated with unavoidable ischemic state such as kidney transplantation, partial nephrectomy or suprarenal procedures of the aorta. During ischemia many cellular changes occur including vascular congestion and adhesion of inflammatory cells to the endothelium with subsequent infiltration into the kidney tissue. Following ischemia, a phase known as Reperfusion begins and involves a return of blood and oxygen supply to micro vessels. Reperfusion was expected to restore the damage occurred during the ischemic phase, paradoxically, reperfusion leads to more congestion, red cells trapping and excessive generation of reactive oxygen species (ROS), which can oxidatively modify significantly every type of biomolecule, thereby inducing cell dysfunction and induce reperfusion injury.	2018	http://ujpr.org/index.php/journal/article/view/3
6	Hepatorenal protection in renal ischemia/reperfusion by celecoxib and pentoxifylline	Pharmacology	Renal ischemia/reperfusion (I/R) is a major clinical problem. Its pathogenesis is multifactorial involving oxidative stress, cytokine overproduction, and inflammatory responses in the kidney and remote organs. This study was performed to evaluate the effects of celecoxib (CEB) and pentoxifylline (PTX) on kidney and liver changes after renal I/R in rats. Renal I/R caused changes in kidney and liver histology with a significant reduction in the function of both organs. An increase in tumor necrosis factor-alpha, myeloperoxidase …	2016	https://www.journalofsurgicalresearch.com/article/S0022-4804(16)30065-8/fulltext