Fasudil is a rho-associated kinase (ROCK) inhibitor that exhibits cardioprotective, vasodilatory, neuroprotective, anti-inflammatory, and anti-angiogenic activities. In animal models of myocardial ischemia/reperfusion, fasudil increases expression of Bcl-2 and p-Akt and decreases expression of Bax and caspase 3, decreasing myocardial infarction size. In other heart failure models, fasudil decreases activation of JNK, translocation of ERK, and expression of c-fos and c-jun. Fasudil also decreases activity of matrix metalloproteinase 9 (MMP9). This compound decreases aneurysm size in models of abdominal aortic aneurysm and also inhibits progression of existing aneurysms in vivo. In animal models of amyotrophic lateral sclerosis (ALS), fasudil decreases motor neuron loss, slowing disease progression and increasing survival time. In animal models of experimental autoimmune encephalitis (EAE), this compound decreases production of toll-like receptor 4 (TLR4), NF-κB, IL-1β, IL-6, and TNF-α and increases production of IL-10 and cannabinoid receptor 2 (CBR2). Additionally, fasudil decreases expression of fibrotic mediators and upregulates expression of prolyl hydroxylase 2, downregulating expression of HIF-1α in diabetic mice.

References


Raja SG. Evaluation of clinical efficacy of fasudil for the treatment of pulmonary arterial hypertension. Recent Pat Cardiovasc

Caution: This product is intended for laboratory and research use only. It is not for human or drug use.