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Nerolidol improves cardiac function in spontaneously hypertensive rats by inhibiting cardiac inflammation and remodelling associated TLR4/NF-κB signalling cascade

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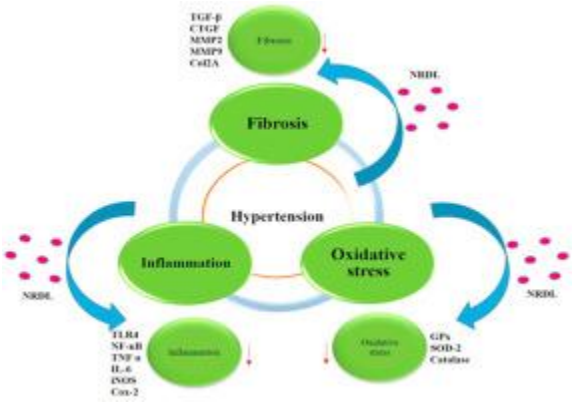
Abstract

Toll-like receptor 4 (TLR4) is an important mediator of hypertension and AngII induced cardiac inflammation and remodelling. In this study, the potential of nerolidol to ameliorate hypertension induced cardiac injuries and the underlying mechanism of action was explored by using *in vitro* and *in vivo* models. The *in vitro* analysis was performed on AngII challenged H9c2 cells and their ability to overcome cardiac inflammation and cardiac remodelling effects was determined by evaluating TLR4/NF-κB signalling cascade using Western blot analysis and immunofluorescence. The results were further ascertained using *in vivo* experiments. Eighteen week old male rats were randomly allocated into different groups i.e. Wistar Kyoto (WKY) rats, hypertensive SHRs, SHRs treated with a low-dose (75 mg/kg b.w) and high-dose of nerolidol (150 mg/kg b.w) and SHRs treated with captopril (50 mg/kg b.w) through oral gauge and finally analysed through echocardiography, histopathological techniques and molecular analysis. The results show that nerilodol target TLR4/NF-κB signalling and thereby attenuate hypertension associated inflammation and oxidative stress thereby provides effective cardioprotection. Echocardiography analysis showed that nerolidol improved cardiac functional characteristics including Ejection Fraction and Fractional Shortening in the SHRs. Collectively, the data of the study demonstrates nerolidol as a cardio-protective agent against hypertension induced cardiac remodelling.

Graphical abstract



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Keywords

Nerolidol; Inflammation; Oxidative stress; Fibrosis; Hypertension

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