

Antihypertensive effect of *Lepechinia caulescens* extract on spontaneously hypertensive rats

Samuel Estrada-Soto^{1,*}, Gabriel Navarrete-Vázquez¹, Ismael León-Rivera², María Yolanda Ríos², Berenice Aguilar-Guadarrama², Patricia Castillo-España³, Rolffy Ortiz-Andrade⁴, Francisco Aguirre-Crespo⁵

¹Facultad de Farmacia, ²Centro de Investigaciones Químicas and ³Centro de Investigación en Biotecnología, Universidad Autónoma del Estado de Morelos, Avenida Universidad 1001, Colonia Chamilpa, 62209, Cuernavaca, Morelos, México.

⁴Facultad de Química, Universidad Autónoma de Yucatán, Mérida, Calle 421 No. 41 x 26 y 28 Col. Industrial, C.P. 97150 Mérida Yucatán 97150, México.

⁵División Ciencias de la Salud, Universidad de Quintana Roo, Boulevard Bahía s/n esq. Ignacio Comonfort, Col. del Bosque, 77019, Chetumal, Quintana Roo, México.

*Corresponding Author: enoch@uaem.mx

Received: 13 February 2012, **Revised:** 17 February 2012 **Accepted:** 18 February 2012

Abstract

The aim of the present study was to examine the antihypertensive effect of methanolic extract from *Lepechinia caulescens* (MELc) and to determine the thoracic aorta reactivity after long-term treatment with MELc. Results showed that MELc at 38 and 120 mg/Kg induced a significant decrease of heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) in comparison with control and similar than captopril (30 mg/Kg). Also, MELc (120 mg/Kg) induced a long-term antihypertensive activity when still down SBP and DBP from fifth day until the end of experiment. Vascular reactivity of vessels from extract-treated animals was improved when were stimulated with carbachol and sodium nitroprusside. However, treatment with noradrenaline enhanced contractile response on these preparations. In conclusion, MELc produced significant antihypertensive and bradycardic effects that may be related with an activation of NO/cGMP pathway.

Keywords: Antihypertensive agent; Lamiaceae; *Lepechinia caulescens*; Ursolic acid; Oleanolic acid; SHR rats

Introduction

The World Health Organization published in 2007 its *Guidelines for Assessment and Management of Cardiovascular Risk*, where reports an estimated of 58 million deaths globally in 2005, from them 30% was produced by Cardiovascular diseases (CVD) (WHO, 2011). CVD are most prevalent causes of death in Western population so far. Moreover, hyp-