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Effect of *Moringa oleifera* leaf extract on blood pressure and vascular reactivity to adrenergic stimulation in L-NAME hypertensive rats

P. Tangsucharit[1], D. Aekthammarat[1], P. Pakdeechote[2], P. Pannangpetch[1]. [1]Pharmacology, Khon Kaen University, Khon Kaen, Thailand, [2]Physiology, Khon Kaen University, Khon Kaen, Thailand.

Introduction: Sympathetic overactivity resulting in increased total peripheral resistance plays a major role in pathogenesis of hypertension[1]. *Moringa oleifera* (MOE) (local name: Marum) is a tropical plant distributed in tropical/subtropical including Thailand. The leaf of *Moringa oleifera* is used in Thai folk medicine for the treatment of many ailments including cardiac and circulatory problems[2]. The present study aims to investigate the preventive effect of aqueous leaf extract of MOE on hemodynamic status and adrenergic reactivity in N^{ω}-nitro-L-arginine-methylester (L-NAME) induced hypertensive rats.

Methods: Male Wistar rats (n=7/each group) were administered daily with L-NAME (50 mg/kg/day) in drinking water for three weeks and concurrent treatment with MOE (30 or 60 mg/kg, p.o.) or captopril (5 mg/kg/day, p.o.). At the end of experiment, blood pressure and heart rate (HR) were measured under pentobarbital sodium (60 mg/kg, i.p.) anesthesia. Contractile responses to perivascular nerve stimulation (PNS; 2-16 Hz) and exogenous phenylephrine (Phe; 0.01-1.0 mmol) were tested in isolated mesenteric vascular bed[3]. Data were expressed as mean ± S.E.M. Statistical analysis was tested by one-way analysis of variance (ANOVA) and followed by Student Newman-Keul's test.

Results: Rats treated with L-NAME had higher BP and HR than the value in control rats. Concurrent treatment with MOE or captopril (5 mg/kg/day) prevented these alterations (Table 1, $p<0.05$). The hemodynamic disturbances were associated with augmented mesenteric reactivity to adrenergic stimulation including PNS and the selective α_1-adrenoceptor agonist in L-NAME rats. Daily MOE-treatment at both low and high doses significantly reduced the reactivity to PNS to the normal levels (Fig.2A, $p<0.05$). Moreover, oral administration of MOE at doses of 30 and 60 mg/kg resulted in dose-dependent recovery of contractile responses to Phe in L-NAME rats (Fig.2B, $p<0.05$).

Conclusion: MOE prevents L-NAME induced hypertension in rats, which might involve the suppression of adrenergic hyperactivity. The current results could suggest that MOE might be useful as a dietary supplement against hypertension associated with sympathoexcitation.

References:
1. Koyama T et al.(2010). *Hypertens Res* **33(5):** 485-91.
2. Randriamboavonjy JI et al. (2016). *Am J Hypertens* **29(7):** 873-81.
3. Tangsucharit P et al. (2012). *Am J Physiol Regul Integr Comp Physiol* **303(11):** R1147-56.

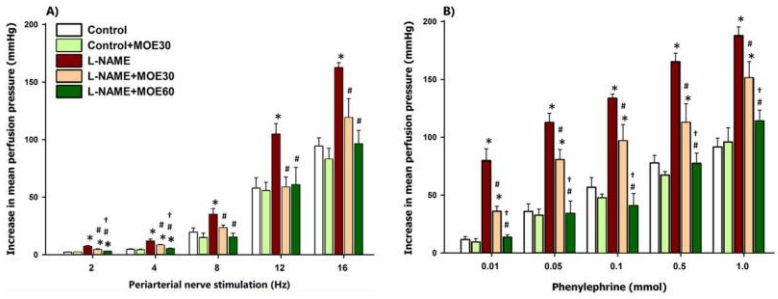

Figure 2 Effect of MOE on adrenergic reactivity to PNS (A) and Phe (B) in all experimental groups. Result are expressed as mean±SEM. n=7/ each group. *$P<0.05$ vs. control group, #$P<0.05$ vs. L-NAME group, †$P<0.05$ vs. L-NAME+MOE30 group

Table 1 Effect of MOE on blood pressure and heart rate in L-NAME hypertensive rats

Parameters	Control	Control+MOE	L-NAME	L-NAME+MOE30	L-NAME+MOE60	L-NAME+Cap
SBP (mmHg)	122.9 ± 1.4	121.9 ± 1.2	193.6 ± 1.6*	175.7 ± 1.9*,#	149.0 ± 1.8*,#,†	161.7 ± 1.2*,#
DBP (mmHg)	83.0 ± 1.6	76.9 ± 2.1	137.5 ± 2.8*	110.5 ± 3.0*,#	90.4 ± 1.4#,†	114.3 ± 4.7*,#
MAP (mmHg)	95.3 ± 1.1	91.8 ± 1.6	160.3 ± 1.8*	134.8 ± 3.6*,#	109 ± 1.4*,#,†	126.8 ± 2.7*,#
HR (beats/min)	380.9 ± 3.6	395.5 ± 13.4	435.0 ± 2.9*	386.2 ± 9.0#	339.0 ± 7.3*,#,†	373.3 ± 3.3#