# Effect of Short-Term Oral Administration of Phikud Navakot in Rats

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***Background:*** *Phikud Navakot (PN), composed of nine herbs and used as a main component of Yahom Navakot, is used in traditional Thai medicine against dizziness and fainting.*
***Objective:*** *To investigate the effects of PN on blood pressure, heart rate (HR), and antioxidant properties on male Sprague Dawley rats.*
***Material and Method:*** *All rats were weighted everyday in the morning, after that, PN (10, 50, 100, 200 and 400 mg/kg BW) were given oroesophageal feeding for seven days. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and HR were measured once per two days. At the end of the experiment, the blood was taken for determination of biochemical and hematological parameters, and lipid peroxidation in serum. The heart was immediately removed for Western blot analysis.*
***Results:*** *SBP, DBP and MAP of rats were transiently increased after 1 day of PN (100 mg/kg BW) treatment. Meanwhile, HR did not change throughout the experiment. PN (400 mg/kg BW) significantly increased ($p<0.05$) the percentage of neutrophils in blood after 7 days of administration. PN treatment has no effect on biochemical parameters and peroxidation of lipid. In addition, ingestion of PN (100 mg/kg BW) significant increased ($p<0.05$) HO-1 expression, but did not change ERK1/2 and Bax/Bcl-2 ratio when compared with the control group.*
***Conclusion:*** *The results may possibly support the use of PN for prevention and/or alleviation of cardiovascular disorders, caused by reactive oxygen species. However, long-term treatment of PN has to be further studies.*

***Keywords:*** *Navakot, Blood pressure, Herbal remedy, Malondialdehyde, HO-1*

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Phikud Navakot (PN) is a major ingredient of "Yahom Navakot", a traditional Thai herbal remedy and an alternative medicine for treatment of cardiovascular symptoms in National List of Essential Medicines 2011, Thailand[1]. PN is composed of nine herbal medicines namely, *Angelica dahurica*, *Atractylodes lancea*, *Ligusticum chuanxiong*, *Angelica sinensis*, *Artemisia pallens*, *Saussurea costus*, *Picrorhiza kurrooa*, *Terminalia chebula* and *Nardostachys jatamansi*, as described in previous study[2]. Several studies have been investigated the properties of Yahom Navakot against cardiovascular symptoms, dizziness and fainting in both rats and human[3-5], supporting the used of Yahom in traditional medicine. Recently, the extracts of Yahom Navakot, PN, and four plants in PN (included *T. chebula*, *P. kurrooa*, *A. pallens* and *N. jatamansi*) have been demonstrated their potent antioxidant activities tested by cell-free systems[6]. In addition, PN attenuated intracellular reactive oxygen species (ROS) generation induced by hydrogen peroxide ($H_2O_2$), and showed no genotoxicity on human umbilical vein endothelial ECV304 cells. However, there has no evidence of PN on cardiovascular systems and its mechanisms in animal model. Thus, the objective of the present study was to evaluate blood pressure, heart rate (HR), and antioxidant properties on PN-administered rats.

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## Material and Method

### *Drugs and chemicals*

Bovine serum albumin (BSA), thiobarbituric acid (TBA) and 1,1,3,3-tetramethoxypropane were

purchased from Sigma-Aldrich Chemical Co. (St. Louis, USA). Halt protease and phosphatase inhibitor cocktail was obtained from Thermo Scientific, Pierce, USA. Primary antibodies, extracellular signal-regulated kinases (ERK) 1/2, Bax and Bcl-2 were obtained from Cell Signaling Technology (Beverly, MA, USA), and heme oxygenase (HO)-1 and beta-actin were from Santa Cruz Biotechnology (Santa Cruz, CA, USA). All other reagents were of analytical grade.

***Preparation of extracts***

An equal amount of nine crude plant materials in PN were purchased from a traditional Thai pharmacy in Bangkok, Thailand, on April 2011, and identified by Dr. Uthai Sotanaphun. Their voucher specimens (NVK01-09) have been deposited in the herbarium of the Department of Pharmacognosy, Faculty of Pharmacy, Silpakorn University, Nakhon Pathom, Thailand. The PN coarse powder was soaked in 10 times by weight of 80% ethanol for overnight and continuously extracted at 100°C, 3 h for 2 times. The two subsequent extracts were combined and concentrated under reduced pressure to give an ethanolic extract of PN (yield = 30% w/w). The total phenolic content (14.76±0.34% gallic acid equivalence) of the extract was determined by Folin-Ciocalteau method[7]. For oral administration, PN was homogenized with N-Lok (National Starch & Chemical, USA) and spray-dried by using an industrial scale spray-dryer (Thai-China Flavours and Fragrances Industry Co. Ltd., Thailand) operated at inlet temperature 200°C and outlet temperature 90°C. The water-soluble powder was obtained and concentration of PN in this preparation was 50%. Finally, PN was freshly suspended in water immediately before oral administration.

***Animals***

Male Sprague Dawley rats having body weight (BW) 250-400 g were obtained from the National Laboratory Animal Center, Mahidol University, Thailand. The rats were housed in a temperature-controlled room at 25°C under 12 h light/dark cycle, and fed with a commercial standard food. All procedures involved in the use of animals for research were approved by the Animal Research Ethics Committee of the Faculty of Medicine, Srinakharinwirot University, Bangkok, Thailand (Approval No. 15/2553 and 12/2554).

Following seven days acclimatization, they were randomly divided into six groups. All rats were weighted daily and then given oroesophageal feeding of either distilled water (a negative control) or five doses of PN (10, 50, 100, 200 and 400 mg/kg BW, weight of herbal extract), namely as PN groups, for seven days. Control and PN groups were sacrificed 24 h after the last dose of administration.

***Measurement of cardiovascular parameters***

The systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), pulse pressure (PP), and HR were measured by a non-invasive tail-cuff blood pressure technology, the CODA™ non-invasive blood pressure (NIBP) system (Kent Scientific Co., USA), as described previously[8]. Rats were divided into 6 groups, n = 5. Conscious rats were restrained in individual holder, and then both an occlusion cuff and a volume pressure-recording cuff were placed close to the base of the tail. All parameters measured before the administration of PN were used as control. Following ingestion of indicated doses of PN, all parameters were measured on day 1, 3, 5 and 7. One week before the experiment, the rats were trained to become accustomed to the tail-cuff blood pressure recorder three times a week.

***Hematological and biochemical investigations***

On the eighth day of experiment, the 6 groups of rats (n = 5) were anesthetized by intramuscular injection of zoletil (50 mg/kg BW), and terminated by cervical dislocation. Blood was quickly collected from inferior vena cava for determination of hematological and biochemical analysis.

***Measurement of serum malondialdehyde (MDA) level***

Blood samples (n = 3-5) were centrifuged at 3,000 rpm for 10 min, and the serum samples were aliquoted and stored at -80°C until used. MDA, derived from polyunsaturated fatty acids, is worldwide used as a biomarker of oxidative stress and lipid peroxidation. The lipid peroxidation was determined by measurement of MDA content by using the TBA reaction as described previously[9] with a partial modification. Briefly, 0.1 mL of 8.1% sodium dodecyl sulfate, 1.5 mL of 20% acetic acid (pH 3.5), and 0.75 mL of 0.8% TBA were sequential added to 0.2 mL of serum. The reaction mixtures were heated at 100°C for 30 min and then cooling. After 1 mL of *n*-butanol: pyridine (15:1, v/v) solution was adding, the samples were vigorous mixing and centrifuged at 3,000 rpm for 15 min. Absorbance of the upper organic layer contained MDA-TBA complex was measured by Synergy™ HT, Multi Mode Microplate Reader, Program Gen5™ data analysis software (BioTek, USA.) at the excitation wavelength

of 485 nm and emission wavelength of 528 nm using a black 96-well microplate. The protein content of the samples was assessed by Bradford method, and a standard curve was obtained from various concentrations of BSA. The concentration of TBA reactive substances was calculated according to simultaneous calibration curves using the 1,1,3,3-tetraethoxypropane as MDA standard. Results were expressed as nanomoles per mg protein.

***Western blot analysis***

The heart was quickly removed and kept at -80°C until the time of analysis. The tissue (100 mg) was homogenized (IKA, Germany) in 1 mL of cold RIPA buffer supplement with protease and phosphatase inhibitor. The homogenate was centrifuged and supernatant was collected for protein determination using Bradford method[10]. Equal amount of proteins were separated on 12% SDS polyacrylamide gel electrophoresis, and electrically transferred onto PVDF membrane. After blocking with 5% non-fat milk in TBST (10 mM Tris pH 7.6, 0.1 M NaCl, and 0.05% Tween 20), the membranes were incubated overnight at 4°C with rabbit polyclonal antibody against HO-1 (1:200), ERK1/2 (1:500), Bax (1:1,000), and Bcl-2 (1:500) diluted in TBST containing 1-5% BSA. After washing, the membranes were further reacted with horse radish peroxidase-conjugated anti-rabbit secondary antibody (1:5,000). The protein bands labeled by Amersham ECL Prime Western Blotting Detection Reagent (GE Healthcare, Sweden) were then captured using Gel documentation system (GeneGnome5, Syngene, Cambridge, USA). After visualization of proteins on membranes, the blots were stripped and reprobed with anti beta-actin antibody (1:500). Density of each band was measured using Image J, and normalized with the density of beta-actin.

***Data and statistical analysis***

Data were expressed as mean $\pm$ standard error of mean (SEM), and statistical analyzed with SPSS software, version 21.0 using one-way ANOVA followed by Tukey's post hoc test. For testing of cardiovascular parameters, the repeated measurement analysis using mixed models was performed. Values of $p<0.05$ were considered statistically significant.

**Results**

***Effects of PN on BW***

The BW of the control and 5 groups administered PN at indicated doses for 7 days did not change significantly among groups. The percentage of BW gains was not significant different when compared with the control group, however, trended to be lower in rats administered with PN at the dose of 400 mg/kg BW as shown in Fig. 1.

***Effects of PN on cardiovascular parameters***

Short-term oral administration of PN at a dose of 100 mg/kg BW showed a significant increase in SBP, DBP, and MAP after 1 day of treatment (Fig. 2A, 2B, 2C). Meanwhile, ingestion of PN (10-400 mg/kg BW) for seven consecutive days did not cause any significant changes among BP, PP and HR (Fig. 2).

***Effect of PN on biochemical and hematological parameters***

Results showed no significant difference in biochemical parameters (Table 1) in rats after daily ingestion of PN (10-400 mg/kg BW) for 7 days, when compared to the control group. Regarding to hematological parameters, only white blood cell (WBC) neutrophil evaluation was significantly elevated in the 400 mg/kg PN group (22.50$\pm$3.50%) when compared with the control group (10.75$\pm$1.84%) as showed in Table 2.

***Effect of PN on serum MDA level***

MDA contents in the serum of rats pretreated with PN at the doses of 10, 50, 100, 200 and 400 mg/kg BW (0.088$\pm$0.019, 0.060$\pm$0.010, 0.076$\pm$0.013, 0.076$\pm$0.011 and 0.053$\pm$0.019 nmol/mg protein, respectively) for 7 days did not differ significantly from the control rats (0.082$\pm$0.012 nmol/mg protein).

***Effect of PN on cardiac expression of HO-1, ERK1/2, Bax/Bcl-2 ratio***

In the present study, changes of the expression of proteins HO-1, ERK1/2, Bax and Bcl-2 from cardiac tissues were investigated. The results showed treatment of PN at the dose of 100 mg/kg BW for 7 days significantly increased ($p<0.05$) the expression of HO-1 when compared with the control group (Fig. 3).

**Discussion**

The BW of rats treated with various doses of PN for 7 days did not change difference when compared with the control group. However, rats ingested PN (400 mg/kg BW) trended to be lower BW than the control group. Our data concurrent with previous results that the PN (100 and 1,000 mg/kg BW) for

90 days of male Sprague Dawley rats[11] when compared with corresponding group. Meanwhile, oral administration of Yahom Navakot extract (1,000 mg/kg BW) for 6 months did not change BW of male Wistar[12].

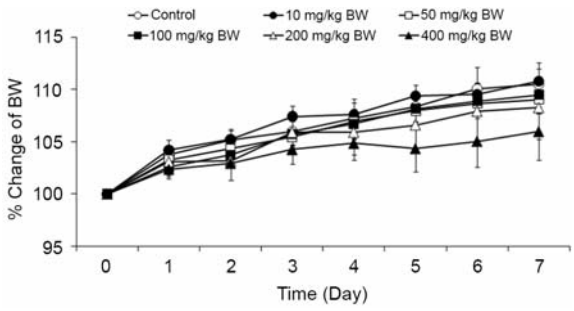


**Fig. 1** Effect of oroesophageal feeding of Phikud Navakot (PN) (10-400 mg/kg BW) once daily for 7 days on body weight (BW) of rats. The results were expressed as mean ± SEM (n = 5-8).

After 1 day of ingestion, PN at the dose 100 mg/kg BW significantly increased SBP, DBP and MAP, supporting the used of PN for treatment of fainting. However, PN did not cause the change of PP and HR in the present study. The effect of Yahom containing some herbs in PN on cardiovascular parameters has been still controversial reported. A study in human found that oral administration of Yahom powder and water extract of Yahom (3 g) increased DBP and MAP after approximately 50 min of ingestion[5]. Supernatant of Yahom powder (containing 1-2 herbs in PN) in hot water (1 mg/mL) caused a positive inotropic effect in isolated rat atrium[13]. Aqueous extraction of Yahom (0.83-16.67 mg/mL) increased aortic ring contraction in dose dependency.

The result shows that rats ingested PN (400 mg/kg BW) for 7 days significantly increased the percentage of WBC, neutrophil, of male Sprague Dawley rats when compared with the control group. Similarly,

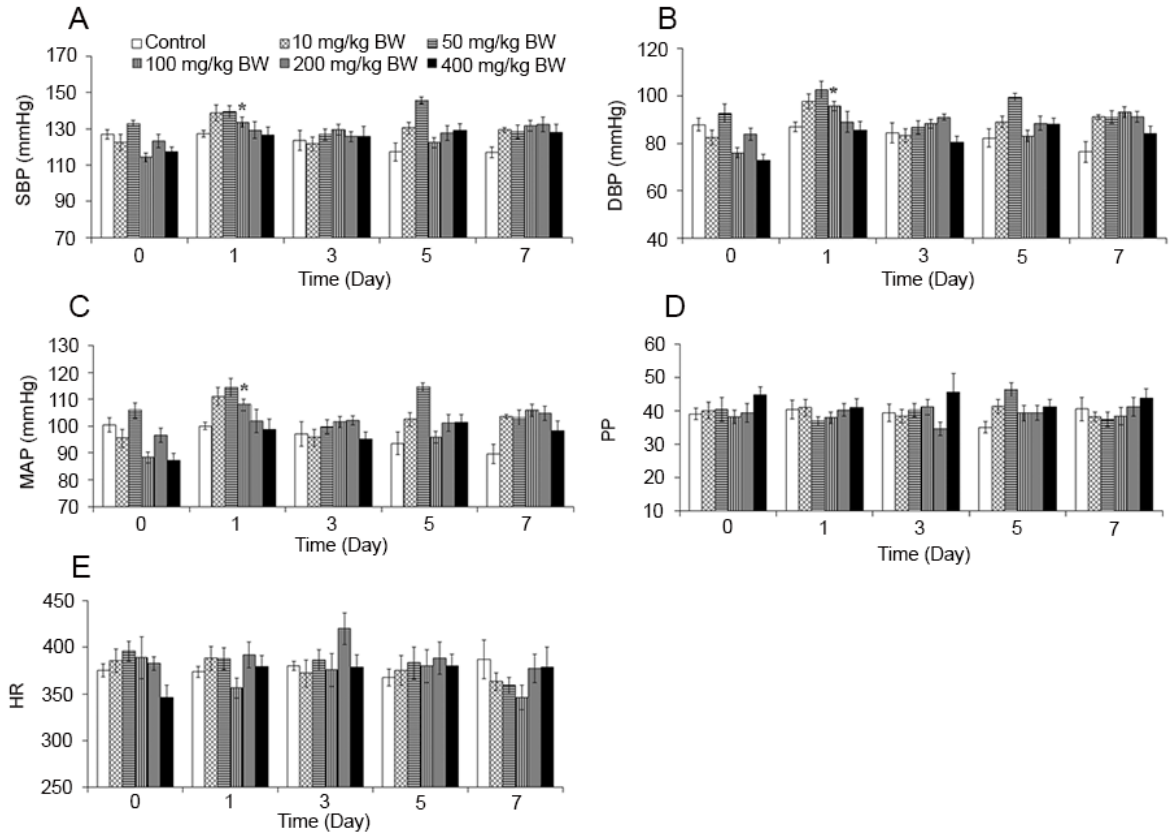


**Fig. 2** Effect of Phikud Navakot (PN) on cardiovascular parameters in rats. After daily oroesophageal feeding of PN (10-400 mg/kg) over 7 days, (A) systolic blood pressure: SBP, (B) diastolic blood pressure: DBP, (C) mean arterial pressure: MAP, (D) pulse pressure: PP, and (E) heart rate: HR of normotensive Sprague Dawley rats were measured by tail cuff every two days for 7 days. The results were expressed as mean ± SEM (n = 5), *$p$<0.05 versus control, using one-way ANOVA.

**Table 1.** The effects of Phikud Navakot (PN) on the biochemical parameters in serum of male Sprague Dawley rats

| Treatment (mg/kg BW) | GLU (mg/dL) | BUN (mg/dL) | CREA (mg/dL) | CHOL (mg/dL) | TG (mg/dL) | URIC (mg/dL) | TP (g/dL) | ALB (g/dL) | GLOB (g/dL) | Bili-T (mg/dL) | AST (U/L) | ALT (U/L) | ALP (U/L) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Control | 211.20±15.96 | 22.60±1.40 | 0.54±0.02 | 77.60±3.17 | 92.80±7.40 | 1.60±0.12 | 6.08±0.10 | 4.10±0.05 | 1.98±0.09 | 0.06±0.02 | 148.00±32.39 | 52.80±5.49 | 135.00±14.40 |
| 10 | 235.00±24.75 | 20.20±0.92 | 0.52±0.02 | 79.20±4.02 | 75.40±13.38 | 1.52±0.18 | 6.12±0.20 | 4.06±0.08 | 2.06±0.15 | 0.08±0.04 | 225.00±81.57 | 66.40±18.04 | 139.20±9.66 |
| 50 | 215.60±13.07 | 19.60±0.93 | 0.54±0.02 | 78.40±2.71 | 113.60±23.98 | 1.86±0.33 | 6.20±0.16 | 4.18±0.06 | 2.02±0.11 | 0.06±0.02 | 117.20±12.13 | 46.60±2.94 | 140.40±7.91 |
| 100 | 208.80±33.64 | 22.20±1.16 | 0.60±0.03 | 81.20±1.07 | 95.80±7.81 | 2.00±0.25 | 6.26±0.07 | 4.26±0.05 | 2.00±0.08 | 0.06±0.02 | 177.00±21.72 | 84.80±12.28 | 148.80±3.35 |
| 200 | 188.20±6.06 | 20.40±1.33 | 0.54±0.02 | 79.80±2.75 | 75.80±7.02 | 1.76±0.09 | 6.36±0.19 | 4.20±0.04 | 2.16±0.15 | 0.03±0.03 | 136.80±17.80 | 47.40±2.94 | 139.00±9.70 |
| 400 | 174.00±10.96 | 22.60±0.81 | 0.62±0.02 | 83.40±4.17 | 68.20±14.56 | 2.18±0.36 | 6.32±0.17 | 3.68±0.43 | 2.64±0.36 | 0.08±0.02 | 259.00±91.57 | 73.40±19.03 | 111.80±30.39 |

The results were expressed as mean ± SEM (n = 5).
GLU = glucose; BUN = blood urea nitrogen; CREA = creatinine; CHOL = cholesterol; TG = triglyceride; TP = total protein; ALB = albumin; GLOB = globulin; Bili-T = total bilirubin; AST = aspartate aminotransferase; ALT = alanine transaminase; ALP = alkaline phosphatase



**Table 2.** The effects of Phikud Navakot (PN) on the hematological parameters in serum of male Sprague Dawley rats

| Treatment (mg/kg BW) | WBC ($10^3$/mL) | RBC ($10^6$/mL) | HBG (g/dL) | HCT (%) | MCV (fL) | MCH (pg) | MCHC (g/dL) | PLT ($10^5$/mL) | Differential count (%) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | NEU | LYMPH | EO | BASO | MONO |
| Control | 5.61±0.95 | 8.94±0.16 | 16.80±0.07 | 49.25±0.48 | 55.18±1.03 | 18.80±0.30 | 34.15±0.42 | 781.00±92.93 | 10.75±1.84 | 84.50±1.66 | 1.50±0.65 | 0.50±0.29 | 2.75±1.18 |
| 10 | 4.63±0.83 | 8.75±0.09 | 16.34±0.18 | 47.60±0.68 | 54.54±0.58 | 18.70±0.15 | 34.26±0.22 | 802.0±196.48 | 14.40±1.96 | 78.80±3.22 | 3.20±2.18 | 0.80±0.49 | 2.80±0.92 |
| 50 | 4.61±0.34 | 8.79±0.06 | 16.48±0.24 | 47.60±0.68 | 54.02±0.82 | 18.78±0.27 | 34.72±0.35 | 872.80±69.75 | 13.20±0.80 | 82.60±0.51 | 1.80±0.66 | 0.40±0.24 | 2.00±0.95 |
| 100 | 5.16±0.65 | 8.95±0.06 | 16.78±0.12 | 49.20±0.58 | 54.94±0.54 | 18.74±0.16 | 34.14±0.28 | 791.40±83.66 | 12.80±1.39 | 82.40±1.40 | 2.80±1.11 | 0.40±0.24 | 1.60±0.75 |
| 200 | 4.65±0.71 | 8.92±0.07 | 16.52±0.12 | 48.60±0.81 | 54.34±0.86 | 18.54±0.21 | 34.12±0.41 | 816.40±71.02 | 19.00±2.47 | 75.20±3.22 | 2.40±1.03 | 0.40±0.24 | 3.00±1.30 |
| 400 | 5.34±1.34 | 9.28±0.18 | 17.08±0.31 | 50.25±1.31 | 54.00±0.84 | 18.43±0.15 | 34.10±0.39 | 726.00±163.40 | 22.50±3.50* | 72.75±4.33 | 1.75±1.44 | 0.25±0.25 | 2.75±1.31 |

The results were expressed as mean ± SEM (n = 3-5).
WBC = white blood cell; RBC = red blood cell; HBG = hemoglobin; HCT = hematocrit; MCV = mean corpuscular volume; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; PLT = platelet; NEU = neutrophil; LYMPH = lymphocyte; EO = eosinophil; BASO = basophil; MONO = monocyte

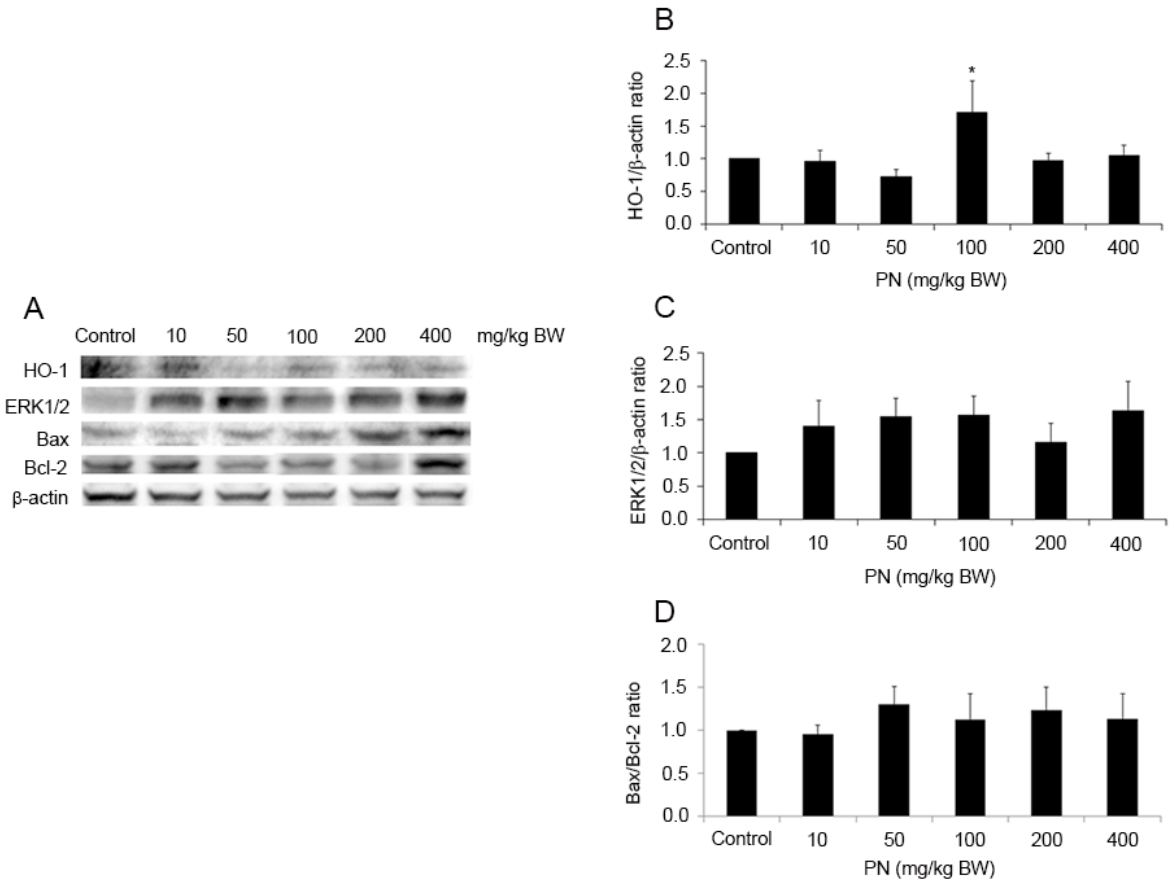


**Fig. 3** The cardiac protein expression of HO-1, ERK1/2, Bax and Bcl-2 in rats after 7-day oroesophageal feeding of Phikud Navakot (PN) (10-400 mg/kg BW) once daily. (A) Representative immunoblotting with anti-HO-1, anti-ERK1/2, anti-Bax, and anti-Bcl-2, and densitometric quantification: (B) HO-1 and (C) ERK1/2 normalized to beta-actin, and (D) Bax/Bcl-2 ratio were shown. Values are presented as the mean ± SEM (n = 4). * $p<0.05$ versus control.

male Wistar rats orally administrated of Yahom Navakot extract (1,000 mg/kg BW) for 6 months also found a significant increase in WBC count, and a decrease in the percentage of eosinophil[12]. Previous studies showed no changes in hematological profiles in male Sprague Dawley rats treated orally with the ethanolic PN at the doses up to 1,000 mg/kg BW for 90 days. However, an increase in blood urea nitrogen, triglyceride, uric acid, globulin and aspartate aminotransferase was demonstrated[11]. The increase in neutrophil count in PN-treated rats may suggest the partial role of PN as an antibacterial function, as demonstrated in the 4-week study of *Ocimum gratissimum* extract[14]. Neutrophils are the major granulocytes which play a major role in defensive mechanism against invading microorganisms through a massive generation of ROS causing lipid peroxidation[15]. However, the present study showed that serum MDA level did not significantly change from the control group after 7-day treatment of PN suggesting the antioxidative properties of PN. It has been reported the antioxidant properties of some herbal compositions and its own active compounds in PN. For instance, methanolic extract of *T. chebula* possessed antioxidant and radical scavenging abilities measured by in vitro antioxidant activities[16]. Ligustilide, a major ingredient of *A. sinensis*, suppressed the production of inflammatory mediators from RAW 264.7 macrophages by down-regulation the intracellular ROS production[17]. Apocynin, isolated from *P. kurroa*, showed powerful antioxidant and anti-inflammatory activities by inhibition of the NADPH oxidase activity on neutrophils and eosinophils[18]. Moreover, an aqueous root extract from *N. jatamansi* was able to

restore haloperidol-induced TBARS production in rats to normal level[19].

Our results showed that PN caused a significant increase in myocardial expression of HO-1 in male Sprague Dawley rats. HO-1 and ERK1/2 are known to modulate multiples pro-survival genes, which play an important role in protection of apoptotic cell deaths against ROS[20]. HO-1 has been demonstrated to improve liver function from ischemia/reperfusion injury by reduction of apoptotic cell death and TBA reactive substances[21]. Some herbs in PN have been shown to up-regulate HO-1 expression as well as antioxidant enzymes regulated by HO-1, as the follows. The ethanolic extract of *A. dahurica* has showed anti-inflammatory action via up-regulation of HO-1 on RAW264.7 cells[22]. *L. chuanxiong* and *A. sinensis*, prepared by ethyl ether, protected ECV304 cells damage against $H_2O_2$ via increased activities of superoxide dismutase, catalase, and glutathione peroxidase as well as activation of ERK and eNOS pathway[23]. Eupatilin, derived from *Artemisia* plants, inhibited indomethacin-induced cell damage by up-regulation of HO-1 expression lead to activation of ERK and the nuclear transcription factor E2-related factor 2 signaling in cultured feline ileal smooth muscle cells[24]. In the present study, a significant increase in the expression of HO-1 in normal rats received PN, possibly suggests a potential role of PN in protection of cell death from lipid peroxidation at least via producing antioxidant enzymes.

In conclusion, PN caused a transient increase in SBP, DBP, and MAP after 24 h administration. Serum MDA had no significant change. An increment in expression of HO-1 was also identified suggesting an antioxidative property of PN. Thus, this scientific evidence suggests that PN may be useful for protection of cardiovascular disorders caused by ROS generation. However, the mechanism of long-term administration of PN has to be further elucidated.

**What is already known on this topic?**

The present study is the first time to evaluate the effects of short-term administration of PN in rats.

**What this study adds ?**

Short-term oral administration of PN increased HO-1 expression in cardiac tissues, and increased blood level of neutrophils in rats.

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**Potential conflicts of interest**

The authors have declared that there is no conflict of interest.

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## ผลของการให้สารสกัดพิกัดนวโกฐทางปากระยะสั้นในหนูแรท

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***ภูมิหลัง:*** *พิกัดนวโกฐประกอบด้วยสมุนไพร 9 ชนิด เป็นส่วนประกอบหลักในตำรับยาหอมนวโกฐ ซึ่งใช้ในการแพทย์ไทยแผนโบราณสำหรับต้านอาการหน้ามืดเป็นลม*

***วัตถุประสงค์:*** *เพื่อศึกษาผลของสารสกัดพิกัดนวโกฐต่อความดันเลือด อัตราการเต้นของหัวใจ และคุณสมบัติในการต้านอนุมูลอิสระในหนูแรทเพศผู้สายพันธุ์ Sprague Dawley*

***วัสดุและวิธีการ:*** *หนูแรททุกตัวได้รับการชั่งน้ำหนักวันละครั้งในตอนเช้าก่อนได้รับสารสกัดพิกัดนวโกฐขนาด 10, 50, 100, 200 และ 400 มิลลิกรัมต่อน้ำหนักตัว 1 กิโลกรัม โดยการป้อนทางปากเป็นเวลา 7 วัน ทำการวัดค่าความดันเลือดช่วงหัวใจบีบตัว ความดันเลือดช่วงหัวใจคลายตัว ค่าเฉลี่ยความดันเลือด และอัตราการเต้นของหัวใจ ทุก 2 วัน เมื่อครบกำหนดเวลา เก็บตัวอย่างเลือด เพื่อตรวจวัดค่าทางเคมีและค่าทางโลหิตวิทยา วัดระดับปฏิกิริยาออกซิเดชันของลิพิดในซีรัม และเก็บหัวใจเพื่อดูการแสดงออกของโปรตีนโดยวิธี western blot*

***ผลการศึกษา:*** *การให้สารสกัดพิกัดนวโกฐ (100 มิลลิกรัมต่อน้ำหนักตัว 1 กิโลกรัม) เป็นเวลา 1 วัน มีผลเพิ่มค่าความดันเลือดช่วงหัวใจบีบตัว ความดันเลือดช่วงหัวใจคลายตัว และค่าเฉลี่ยความดันเลือดไม่มีผลต่ออัตราการเต้นของหัวใจตลอดการทดลอง หนูแรทที่ได้รับสารสกัดพิกัดนวโกฐขนาด 400 มิลลิกรัมต่อน้ำหนักตัว 1 กิโลกรัม เป็นเวลา 7 วัน พบเปอร์เซ็นต์ของเม็ดเลือดขาวนิวโทรฟิลเพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติ ($p<0.05$) สารสกัดพิกัดนวโกฐไม่มีผลเปลี่ยนแปลงค่าทางเคมีในเลือดและปฏิกิริยาออกซิเดชันของลิพิดในซีรัม สารสกัดพิกัดนวโกฐขนาด 100 มิลลิกรัมต่อน้ำหนักตัว 1 กิโลกรัม เพิ่มการแสดงออกของ HO-1 ในเนื้อเยื่อหัวใจอย่างมีนัยสำคัญทางสถิติ ($p<0.05$) เมื่อเปรียบเทียบกับหนูแรทกลุ่มควบคุมแต่ไม่มีผลเปลี่ยนแปลงการแสดงออกของ ERK1/2 และอัตราส่วนของ Bax ต่อ Bcl-2*

***สรุป:*** *จากผลการศึกษา มีความเป็นไปได้ที่จะนำสารสกัดพิกัดนวโกฐไปใช้ประโยชน์เพื่อป้องกัน และ/หรือบรรเทาความผิดปกติทางระบบหัวใจ และหลอดเลือดที่เกิดจากอนุมูลอิสระ อย่างไรก็ตามจำเป็นต้องมีการศึกษาผลระยะยาวของสารสกัดพิกัดนวโกฐต่อไป*