

allowed to clot and subsequently centrifuged at 4,000 rpm for 10 minutes. The resulting serum was meticulously separated into another sterile plain tube and stored at 4°C until required. The heart and liver of each rat were then excised, rinsed with a saline solution at a low temperature. The tissues were immersed in liquid nitrogen and promptly preserved at -80°C for subsequent analyses.

2.6 Measurement of lipid profile

The collected sera were used to assess various lipid components, including total cholesterol (TC), triglycerides (TGs), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). The quantification of TC and TGs followed the methods of (Roeschlau et al., 1974), and (Biggs et al., 1975), respectively. The HDL-C level was determined using the method outlined by (Warnick et al., 2001), while the estimation of LDL-C employed the Friedewald formula (Krishnaveni and Gowda, 2015):

$$\text{LDL-C} = (\text{TC}) - (\text{HDL-C}) - (\text{TGs}/5)$$

2.7 Estimation of liver function indices

The activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were assessed following the procedure outlined by (Reitman and Frankel, 1957). Alkaline phosphatase (ALP) activity was determined using the method described by (Babson et al., 1966). Total protein (TP) was quantified using the biuret reaction, as described by (Linne and Ringsurd, 1979). The concentration of albumin (ALB) was determined using the bromocresol green dye-binding method (Doumas et al., 1971). Serum total and conjugated bilirubins were determined according to the method described by (Malloy and Evelyn, 1937) and modified by (Nwanjo and Alumanah, 2006).

2.8 Assay of tissue oxidative stress and antioxidant enzyme markers

The heart and liver were individually sectioned into smaller fragments with a sterile scalpel and homogenized in an aqueous solution of 0.1M potassium buffer (pH 7.4). Subsequently, the homogenates underwent centrifugation at 10,000 rpm (4°C) for 10 minutes, and the resulting supernatants were employed for the antioxidant assays. Catalase (CAT) activity was assessed using the method developed by (Goth, 1991). The activity of superoxide dismutase (SOD) was estimated through the pyrogallol autoxidation method as outlined by (Marklund and Marklund, 1974). Reduced glutathione (GSH) was determined using the 5,5'-dithiobis-2-nitrobenzoic acid (DTNB) recycling method described by (Banerjee et al., 1999). Glutathione peroxidase (GPx) activity was determined according to the method of Kinoshita et al. (1996). The level of malondialdehyde (MDA) was measured using the method described by (Okhawa et al., 1979).

2.9 Statistical analysis

Data were expressed as mean \pm standard deviation, using the statistical software SPSS version 27. The data were analysed by one way analysis of variance (ANOVA) followed by a post-hoc Tukey test at $P < 0.05$.

3 Results

As shown in Table 1, L-NAME administration significantly increased SBP and MAP compared with the control group. Treatment with TSO attenuated these elevations, with the higher dose producing effects comparable to those of enalapril. Significant changes in DBP were observed only in the high-dose TSO and enalapril-treated groups.

Table 1 Influence of TSO on blood pressure indices

Blood Pressure Parameters (mm Hg)	Group A (Control)	Group B	Group C	Group D	Group E
SBP	119.57 \pm 4.75	124.83 \pm 2.63 ^a	119.00 \pm 13.42 ^b	119.42 \pm 11.87 ^b	108.29 \pm 4.03 ^{a,b}
DBP	82.67 \pm 6.09	81.00 \pm 8.20	80.83 \pm 11.75	77.60 \pm 9.75 ^{a,b}	73.57 \pm 3.10 ^{a,b}
MAP	93.50 \pm 3.87	97.75 \pm 5.00 ^a	93.00 \pm 13.68	88.50 \pm 11.22	84.86 \pm 2.61 ^{a,b}

Values are expressed as mean \pm SD (n = 8). A, B, C, D and E represent control, 40 mg/kg L-NAME, 40 mg/kg L-NAME + 0.45 mL/kg TSO, 40 mg/kg L-NAME + 0.6 mL/kg TSO and 40 mg/kg L-NAME + 2 mg/kg Enalapril Maleate, respectively. ^aP < 0.05 significantly different compared with control (A); ^bP < 0.05 significantly different compared with group B