

## 4 Discussion

Tea is widely consumed not only as a beverage but also for its numerous health benefits, which have been attributed to its abundance of polyphenols, catechins, and other antioxidant compounds (Musial et al., 2020). In the present study, we employed a well-established model of experimental hypertension induced by chronic administration of L-NAME, a non-selective nitric oxide synthase (NOS) inhibitor (Krasnylenko et al., 2019; Adedapo et al., 2020). By competitively inhibiting NOS, L-NAME reduces nitric oxide (NO) bioavailability, leading to endothelial dysfunction and elevated blood pressure (Zhao et al., 2015).

The co-administration of TSO alongside L-NAME appeared to mitigate the hypertensive effects caused by L-NAME. This normalization of blood pressure parameters may be attributed to the vasodilatory properties of TSO. This finding aligns with the report of Fuchs et al. (2014), who demonstrated enhanced microcirculation following catechin and theaflavin supplementation in healthy individuals, supporting the beneficial effects of tea-derived compounds on vascular function.

Hypertension is intricately associated with disturbances in lipid metabolism, with dyslipidemia frequently occurring as a coexisting condition. Elevated blood pressure is often accompanied by increased serum lipid levels, including TC, LDL-C, and TGs (Lee and Siddiqui, 2019). Among these lipid fractions, elevated LDL-C plays a major role in the development of atherosclerosis through the accumulation of cholesterol within arterial walls, thereby impairing vascular function and increasing cardiovascular risk. Conversely, HDL-C plays a protective role by mediating reverse cholesterol transport, facilitating the removal of cholesterol from peripheral tissues to the liver for excretion. Reduced HDL-C levels in hypertensive individuals may diminish this protective mechanism, thereby accelerating the progression of atherosclerotic disease (Ben-Aicha et al., 2020; Khatana et al., 2020). Furthermore, elevated triglyceride concentrations have been implicated in the development of cardiovascular complications and are considered an independent risk factor for cardiovascular disease (Packard et al., 2020). In the present study, L-NAME administration altered the lipid profile of the experimental animals, while concurrent treatment with TSO restored these parameters toward normal values. This finding is consistent with previous reports demonstrating the lipid-lowering effects of tea-derived products. For example, Samavat et al. (2016) reported significant reductions in TC and LDL-C following long-term supplementation with green tea catechin extract.

The growing recognition of the link between hypertension and hepatic dysfunction shows the importance of evaluating liver function in hypertensive models. Liver enzymes such as ALT, AST, GGT, ALP serve as established biomarkers of hepatic integrity and function (Rahman et al., 2020). Elevations in these enzymes are often associated with systemic inflammation and oxidative stress, which are known contributors to endothelial dysfunction and the pathogenesis of hypertension (Guzik and Touyz, 2017). The increased activities of these enzymes observed following L-NAME administration indicate hepatic stress, whereas their reduction in the TSO-treated groups suggests a hepatoprotective effect of TSO. The reduction in total protein observed in the hypertensive group may reflect impaired hepatic synthetic function or increased protein catabolism under conditions of oxidative stress (Li et al., 2020). A notable reduction in albumin (ALB) concentration in the Enalapril Maleate-treated group raises important considerations regarding the effects of this drug on hepatic protein metabolism. Although angiotensin-converting enzyme (ACE) inhibitors are generally regarded as hepatoprotective, the mechanism underlying this observed decrease warrants further investigation, particularly in the context of long-term administration or possible interaction with hypertensive states.

Oxidative stress is a key mechanism underlying L-NAME-induced hypertension (Tan et al., 2018). In the present study, elevated MDA levels together with reductions in antioxidant defenses confirmed the presence of oxidative imbalance following NOS inhibition. Also, decreased activities of CAT, SOD, GPx, and reduced levels of GSH in the L-NAME-treated group indicate impairment of endogenous antioxidant systems responsible for neutralizing reactive oxygen species and maintaining cellular redox homeostasis (Panday et al., 2020; Maurya and Namdeo, 2021; Vitolo, 2021). The improvement in these antioxidant markers following TSO administration suggests that TSO enhances endogenous antioxidant capacity and limits oxidative damage. These findings further support the