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ANTIGLYCATION POTENTIAL OF GANODERMA LUCIDUM MYCELIUM IN FRUCTOSE-INDUCED METABOLIC SYNDROME

Background. Metabolic syndrome (MetS) currently affects over one-fifth of the global population. Chronic hyperglycemia, inherent to MetS, acts as a triggering factor for the non-enzymatic glycation of proteins, initiated by an excess of reducing sugars in the bloodstream, with the subsequent formation of stable advanced glycation end-products (AGEs). Accumulated glycation products induce structural and functional modifications of proteins, particularly receptors and enzymes, leading to the inhibition of their physiological activity [1]. Consequently, searching for effective agents to correct these alterations is highly relevant, with *Ganoderma lucidum* being of particular interest due to its combined hypoglycemic and antioxidant potential. **Methods.** MetS was modelled in male Wistar rats via 42-day high-carbohydrate intake (10% fructose). Animals were randomized into four groups ($n = 8$): a control group, a MetS group, and two corresponding groups treated with the extract. *G. lucidum* mycelium extract was administered per os (1 g/kg) during the final 14 days of the experiment. Quantitative assessment of AGEs and their fluorescent derivatives (pentosidine, vesperlysine, argpyrimidine) in blood plasma was performed via spectrofluorometry using a Shimadzu RF-5000 spectrofluorophotometer (Shimadzu, Japan). **Results.** In rats with MetS, AGEs content increased by 124.6%, argpyrimidine by 104.1%, while pentosidine and vesperlysines nearly doubled. Extract administration reduced the levels of AGEs by 46.4%, argpyrimidine by 52.4%, pentosidine by 41.1%, and vesperlysine A/B by 41.8% compared to the MetS group. Vesperlysine C levels showed only a downward trend. In the control group, the extract did not cause any statistically significant changes. **Conclusions.** *G. lucidum* mycelium extract exhibits a pronounced antiglycation potential under experimental MetS conditions. The extract administration significantly reduces blood plasma AGEs accumulation, with argpyrimidine being the most sensitive marker to correction. This effect is driven by specific bioactive components: apigenin, luteolin, polysaccharides, and proteoglycans provide a systemic hypoglycemic effect that limits early glycation product formation, while the synergistic antioxidant action of the extract's polysaccharides and phenolic compounds (gallic and ellagic acids, rutin) prevents late-stage biomolecule modification [2].

Keywords: metabolic syndrome, *Ganoderma lucidum*, AGEs.

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