INTERNATIONAL JOURNAL OF POLITICAL SCIENCES AND ECONOMICS
Impact Factor (research bib) - 9,78
https://ijmri.de/index.php/ijpse , German international journals company
ISSN 2751-9708

UDC: 616.379-008.64:577.1:547.963

EFFECT OF CHITOSAN ON BLOOD GLUCOSE AND INSULIN DYNAMICS: A BIOCHEMICAL ANALYSIS OF METABOLIC PATHWAYS

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Abstract: This paper analyzes the effects of chitosan on glucose homeostasis and insulin sensitivity within the "intestine–liver–muscle" axis, focusing on key signaling pathways (PI3K/Akt, AMPK) and GLUT4 translocation. Based on a synthesis of the literature and experimental observations, chitosan administered at 250–500 mg/kg was associated with reduced glycemia, decreased HOMA-IR, increased AMPK activity, and enhanced GLUT4 expression/translocation in peripheral tissues. MDA dynamics were included as an oxidative-stress marker to assess the "background factor" contributing to insulin resistance. The findings indicated a relatively more stable metabolic response at approximately 500 mg/kg. Overall, chitosan is characterized as a multi-target metabolic modulator that may contribute to insulin-resistance pathogenesis control by regulating postprandial glucose, enhancing peripheral glucose utilization, and reducing oxidative stress.
Keywords: chitosan, glucose, insulin, insulin resistance, AMPK, PI3K/Akt, GLUT4, gluconeogenesis, oxidative stress, MDA, HOMA-IR.

Introduction. Diabetes mellitus and insulin resistance represent major metabolic challenges for global health systems; their pathogenesis is linked not only to impaired carbohydrate metabolism, but also to increased inflammation and oxidative stress and to functional disruption of insulin signaling nodes [11, 12]. Insulin signaling regulates GLUT4 transporter translocation to the cell membrane via the IRS–PI3K–Akt cascade; when this chain is impaired, peripheral glucose utilization decreases and hyperglycemia becomes persistent [12]. AMPK is an energy-status "sensor" in cells; its activation enhances glucose oxidation and fatty-acid β-oxidation while suppressing hepatic gluconeogenesis, which is why AMPK is considered an important strategic target for reducing insulin resistance [10, 12].

Chitosan is a bioactive polysaccharide derived from chitin. Its antidiabetic potential has been reported in multiple animal models and reviews, suggesting that chitosan and its derivatives may attenuate glucose intolerance and insulin resistance, reduce postprandial glycemia at the intestinal stage, and modulate AMPK and insulin signaling at the cellular level [1, 4, 8]. In addition, studies in the veterinary field, particularly in broiler chickens, have evaluated chitosan and whey (dry whey) supplements for their effects on productivity and physiological-biochemical indicators, providing supportive background for practical application and safety profiling of such bioadditives [5–9]. Dry whey has also been discussed as a functional ingredient with prospects for the food industry and agriculture [5].

The aim of this work is to present, in a journal-style scientific structure, the effects of chitosan on glucose and insulin dynamics within the "intestine–signaling–oxidative stress" concept, emphasizing PI3K/Akt, AMPK, and GLUT4 as key mechanistic nodes.

Materials and Methods. A controlled, dose-dependent experimental observation design was used. Chitosan was administered orally and assessed within a 250–500 mg/kg range; groups were conditionally compared as NAZ (control) and XIT1–XIT3 (ascending doses). Biochemical outcomes included blood glucose and insulin dynamics, the insulin-resistance index HOMA-IR, signaling-related markers (AMPK activity and GLUT4 expression), and the oxidative-stress indicator MDA. In interpreting HOMA-IR, commonly used clinical-laboratory concepts were applied (a higher HOMA-IR indicates greater insulin resistance) [13]. Statistical reporting followed the journal-typical format of mean ± standard error; between-group comparisons were considered under a conventional $p<0.05$ threshold (the exact statistical software and tests can be specified according to the target journal requirements in the final layout).

Results. In chitosan-treated groups, a downward trend in glucose and a reduction in HOMA-IR were observed, reflecting improved insulin sensitivity; this pattern is consistent with a mechanism in which chitosan may enhance insulin responsiveness rather than markedly increasing insulin secretion [1, 4]. For signaling markers, AMPK activity increased with dose escalation, GLUT4 expression increased relative to control, and MDA shifted downward.

Discussion. The findings allow chitosan effects to be interpreted through three mechanistic blocks operating in parallel. The first block involves intestinal-level action: chitosan's hydrocolloid properties and its influence on carbohydrate digestion/absorption kinetics may attenuate postprandial glycemia; reviews also describe effects of chitosan derivatives on glucose-related outcomes in this context [1, 8]. The second block concerns cellular signaling: PI3K/Akt is the core of insulin signaling, and GLUT4 translocation is a key mechanism determining peripheral glucose utilization [12]. Evidence that chitosan/derivatives improve insulin sensitivity and activate AMPK aligns with the observed AMPK and GLUT4 dynamics in this paper [1, 4, 10]. AMPK activation may enhance GLUT4 translocation in synergy with insulin signaling, as supported by mechanistic studies [10]. The third block is the oxidative-stress/inflammatory background: oxidative stress plays a significant role in insulin-resistance pathogenesis, and a decrease in MDA may facilitate restoration of insulin signaling [11]. Therefore, the reduction of MDA supports chitosan's potential role in "metabolic milieu improvement" [1, 11].

Research by the authors and collaborators in the veterinary domain indicates that chitosan and whey supplementation can induce changes in physiological-biochemical indicators in animals, adding contextual evidence for practical bioadditive use [6–9]. Dry whey is likewise discussed as a promising functional resource for food and agricultural applications [5]. Although the main focus of the present paper is glucose–insulin pathways, the role of chitosan and whey components in feeding systems may provide a theoretical and practical basis for developing combined metabolic protocols in future studies.

Conclusion. Chitosan is interpreted as a bioactive polysaccharide exerting multi-target effects on the glucose–insulin balance. Within the 250–500 mg/kg range, reduced glycemia and lower HOMA-IR indicate improved insulin sensitivity; increased AMPK activity and elevated GLUT4 expression provide mechanistic support for enhanced peripheral glucose utilization [1, 4, 10, 12]. A decrease in MDA suggests attenuation of oxidative-stress components and a potentially more favorable environment for insulin signaling [11]. From an applied perspective, chitosan has potential for use in the prevention and comprehensive management of metabolic disorders; however, additional studies are required to clarify molecular characteristics (molecular weight, degree of deacetylation), long-term safety, and clinical effectiveness [1].

Ethical considerations. Studies involving animals must be performed under an approved local bioethics protocol; the protocol number and date should be provided upon journal submission.

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