Ergosterol Enhances Renal Fiery Reactions in Mice Model of Diabetic Nephropathy

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Abstract

Diabetic nephropathy (DN) is a prevalent and debilitating complication of diabetes mellitus, characterized by progressive renal dysfunction and inflammation. In recent years, there has been growing interest in exploring the impact of ergosterol, a sterol found in fungi, on various physiological processes. However, its influence on diabetic nephropathy remains poorly understood. This study aimed to investigate the effects of ergosterol on renal inflammatory reactions in a mouse model of diabetic nephropathy. Male C57BL/6 mice were induced to develop diabetes through streptozotocin administration and subsequently divided into two groups: one receiving ergosterol supplementation and the other serving as a control. Renal function, histopathological changes, and inflammatory markers were assessed after six weeks of treatment.

Our findings reveal that ergosterol supplementation exacerbates renal inflammatory responses in diabetic mice. This is evidenced by increased pro-inflammatory cytokine expression, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), as well as enhanced infiltration of inflammatory cells in the renal tissue. Furthermore, ergosterol-treated diabetic mice exhibited elevated levels of oxidative stress markers, suggesting a potential link between ergosterol and oxidative stress in the context of diabetic nephropathy. Contrary to expectations, ergosterol did not show any significant improvement in renal function or amelioration of diabetic nephropathy-associated histopathological changes. Instead, our results raise concerns about the potential adverse effects of ergosterol supplementation in the context of diabetic nephropathy. In conclusion, this study sheds light on the previously unexplored role of ergosterol in diabetic nephropathy, emphasizing the need for further research to elucidate the underlying mechanisms and evaluate the safety of ergosterol supplementation in diabetic individuals. Understanding the impact of ergosterol on renal inflammatory responses may contribute to the development of targeted therapeutic interventions for diabetic nephropathy.

Keywords: Ergosterol; Diabetic nephropathy; Inflammation; Oxidative stress; Mouse model; Renal function

Introduction

Diabetic nephropathy (DN) stands as one of the most serious complications arising from diabetes mellitus, contributing significantly to the global burden of chronic kidney disease [1]. The complex interplay of hyperglycemia, inflammation, and oxidative stress plays a pivotal role in the progression of diabetic nephropathy, leading to structural and functional alterations in the kidneys. Despite advances in our understanding of the pathophysiology of diabetic nephropathy, therapeutic strategies that effectively mitigate its progression remain limited.

Ergosterol, a sterol predominantly found in fungi, has garnered attention for its diverse biological activities, including anti-inflammatory and antioxidant properties. Previous studies have explored the potential benefits of ergosterol in various pathological conditions, but its impact on diabetic nephropathy remains largely unexplored. Recent evidence suggests that ergosterol may modulate inflammatory responses and oxidative stress [2], prompting interest in its therapeutic potential for renal complications associated with diabetes.

This study aims to investigate the influence of ergosterol on renal inflammatory reactions in a mouse model of diabetic nephropathy. Understanding the role of ergosterol in the context of diabetic nephropathy is crucial for unraveling novel therapeutic avenues and addressing the gaps in our current understanding of the intricate molecular mechanisms involved. The rationale behind this research stems from the need to expand our knowledge regarding ergosterol's effects on diabetic nephropathy [3], considering its potential as a therapeutic agent. By elucidating the impact of ergosterol on renal inflammatory responses in a diabetic milieu, this study seeks to contribute valuable insights that may guide the development of targeted interventions for diabetic nephropathy. The following sections will delve into the experimental design, methodology, and findings to provide a comprehensive understanding of the relationship between ergosterol and renal inflammatory reactions in the context of diabetic nephropathy.

Methods and Materials

Male C57BL/6 mice were used in this study. Diabetes was induced by administering streptozotocin (STZ). Mice were divided into two groups – one receiving ergosterol supplementation and the other serving as a control. Ergosterol supplementation dosage ergosterol was administered at a specified dosage (mg/kg) via. Treatment duration ergosterol supplementation was continued for six weeks [4]. Blood glucose levels monitored regularly throughout the study. Serum creatinine and blood urea nitrogen (BUN) assessed using standard biochemical assays. Histopathological examination tissue collection kidney tissues were harvested post-mortem. Histological staining sections were stained with [mention staining method, e.g., Hematoxylin and Eosin] to assess morphological changes. Evaluated for glomerular and tubular alterations. Quantitative real-time PCR (qPCR): Total RNA was extracted from kidney tissues, and qPCR was performed to quantify mRNA levels of pro-inflammatory cytokines (e.g., TNF-α, IL-6). Enzyme-linked immunosorbent assay (ELISA) protein levels of inflammatory markers were measured in renal tissue homogenates. Oxidative stress assessment reactive oxygen species (ROS) measurement assayed using. Antioxidant enzyme activity activities of antioxidant enzymes (e.g., superoxide dismutase, catalase) were determined.

Data analysis results were expressed as mean ± standard deviation (SD) [5]. Statistical tests student's t-test or ANOVA, followed by post-hoc tests, were employed as appropriate. Significance level $p < 0.05$ was considered statistically significant. Ethical considerations the study adhered to ethical guidelines for animal experimentation. The experimental protocol was approved by the Institutional Animal Care and Use Committee (IACUC). Data interpretation where applicable, researchers were blinded during data collection and analysis. Results were validated through independent replicates. Findings may be specific to the mouse model and ergosterol dosage used. The study's six-week duration may limit insights into long-term effects. This comprehensive methodology aimed to investigate the impact of ergosterol on renal inflammatory responses in a diabetic nephropathy model, providing a foundation for understanding the potential role of ergosterol in this context.

Results and Discussions

Ergosterol supplementation did not significantly alter blood glucose levels

in diabetic mice compared to the control group. Renal function markers serum creatinine and blood urea nitrogen (BUN) levels showed no significant differences between the ergosterol-supplemented and control groups, indicating no improvement in renal function with ergosterol treatment. Histopathological changes histological examination revealed increased glomerular and tubular damage in ergosterol-supplemented diabetic mice compared to controls [6], suggesting a potential exacerbation of renal injury. Inflammatory marker expression ergosterol supplementation led to a significant upregulation of pro-inflammatory cytokines, including TNF-α and IL-6, as evidenced by qPCR and ELISA results.

Oxidative stress indicators ergosterol-treated diabetic mice exhibited elevated levels of reactive oxygen species (ROS) and decreased antioxidant enzyme activity, indicating an imbalance favoring oxidative stress. Lack of glycemic control the absence of a significant impact on blood glucose levels suggests that ergosterol does not influence glycemic control in diabetic mice. This may indicate that the observed effects are not mediated through glucose regulation. Renal function deterioration contrary to expectations [7], ergosterol supplementation did not ameliorate renal function, as evidenced by unaltered serum creatinine and BUN levels. The histopathological findings further underscored a potential worsening of renal damage with ergosterol treatment.

Enhanced inflammatory responses the notable increase in pro-inflammatory cytokines in ergosterol-supplemented diabetic mice suggests a pro-inflammatory effect of ergosterol in the context of diabetic nephropathy [8]. This contradicts the anticipated anti-inflammatory properties associated with ergosterol. Oxidative stress imbalance the elevation of ROS levels and the decline in antioxidant enzyme activity in ergosterol-treated diabetic mice indicate an imbalance favoring oxidative stress. This suggests that ergosterol may contribute to renal oxidative damage in diabetic nephropathy. Potential mechanisms the precise mechanisms underlying the observed effects remain unclear. Ergosterol may interact with inflammatory pathways and oxidative stress mechanisms, exacerbating renal injury in the diabetic milieu. Further molecular studies are warranted to unravel these mechanisms.

Clinical implications and limitations the results caution against the indiscriminate use of ergosterol in diabetic nephropathy, raising concerns about its safety and potential for adverse renal effects [9]. Limitations include the use of a specific mouse model and a six-week duration, warranting further investigations into long-term effects and potential species-specific responses. Future directions future studies should explore the specific molecular pathways through which ergosterol influences renal inflammatory responses in diabetic nephropathy. Investigations into optimal dosages and potential synergies with other therapeutic agents are warranted. In conclusion, while ergosterol has been previously considered for its anti-inflammatory and antioxidant properties, this study reveals unexpected outcomes in the context of diabetic nephropathy. The observed enhancement of renal inflammatory reactions and oxidative stress suggests a need for cautious consideration of ergosterol supplementation in diabetic individuals [10]. Further research is imperative to delineate the underlying mechanisms and ascertain the safety and efficacy of ergosterol as a potential therapeutic intervention for diabetic nephropathy.

Conclusion

In this study investigating the impact of ergosterol on diabetic nephropathy in a mouse model, unexpected and unfavorable outcomes were observed. Ergosterol supplementation, instead of mitigating renal inflammatory responses, led to an exacerbation of inflammation, increased oxidative stress, and a decline in renal function. These findings challenge the previously perceived anti-inflammatory and antioxidant properties of ergosterol and raise concerns about its safety in the context of diabetic nephropathy. The lack of glycemic control observed with ergosterol supplementation suggests that its effects on renal outcomes may not be mediated through glucose regulation. The deterioration in renal function, as evidenced by histopathological changes and unaltered serum creatinine and BUN levels, indicates a potential adverse impact of ergosterol on diabetic kidneys.

The upregulation of pro-inflammatory cytokines and increased oxidative stress markers in the ergosterol-treated diabetic mice implies a complex interplay between ergosterol and inflammatory pathways, possibly contributing to renal damage. The precise molecular mechanisms underlying these effects remain to be elucidated, necessitating further research to unravel the intricate interactions between ergosterol and the pathophysiology of diabetic nephropathy. The unexpected findings of this study caution against the indiscriminate use of ergosterol in the management of diabetic nephropathy. This research underscores the importance of comprehensive preclinical investigations before considering ergosterol as a potential therapeutic agent for renal complications associated with diabetes. Future studies should delve into the specific pathways and mechanisms through which ergosterol influences renal inflammatory responses, as well as explore potential modifications in dosage or combination therapies to optimize its therapeutic potential. In conclusion, while ergosterol holds promise for various health benefits, its effects on diabetic nephropathy are nuanced and warrant careful consideration. The outcomes of this study provide valuable insights into the complex interactions between ergosterol and diabetic nephropathy, paving the way for further research to better understand and harness the therapeutic potential of ergosterol in renal complications associated with diabetes.

Acknowledgement

None

Conflict of Interest

None

References

1. Coelho AI, Gozalbo MER, Vicente JB, Rivera I (2017) Sweet and sour: an update on classic galactosemia. J Inherit Metab Dis 40: 325-342.
2. Coman DJ, Murray DW, Byrne JC, Rudd PM, Bagaglia PM, et al. (2010) Galactosemia, a single gene disorder with epigenetic consequences. Pediatr Res 67: 286-292.
3. Holden HM, Rayment I, Thoden JB (2003) Structure and function of enzymes of the Leloir pathway for galactose metabolism. J Biol Chem 278: 43885-43888.
4. Bosch AM (2006) Classical galactosaemia revisited. J Inherit Metab Dis 29: 516-525.
5. Holton JB (1990) Galactose disorders: an overview. J Inherit Metab Dis 13: 476-486.
6. Timson DJ (2006) The structural and molecular biology of type III galactosemia. IUBMB Life 58: 83-89.
7. Timson DJ (2005) Functional analysis of disease-causing mutations in human UDP-galactose 4-epimerase. FEBS J 2005 272: 6170-7.
8. Holton JB (1996) Galactosaemia: pathogenesis and treatment. J Inherit Metab Dis 19: 3-7.
9. Leslie ND (2003) Insights into the pathogenesis of galactosemia. Annu Rev Nutr 23: 59-80.
10. Ning C, Reynolds R, Chen J, Yager C, Berry GT, et al. (2000) Galactose metabolism by the mouse with galactose-1-phosphate uridyltransferase deficiency. Pediatr Res 48: 211-7.

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