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Transcriptomic Insights into the Anti-Inflammatory Effects of Curcuma longa in Diabetic Endothelial Dysfunction

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Abstract

Endothelial dysfunction is a central mechanism underlying vascular complications in diabetes mellitus and is closely associated with chronic inflammation, oxidative stress, and metabolic dysregulation. Curcuma longa (curcumin) has been widely investigated for its anti-inflammatory and antioxidant effects; however, its molecular actions on diabetic endothelium are not yet fully understood. Recent advances in transcriptomic technologies, including RNA sequencing and single-cell RNA sequencing, have provided novel insights into the endothelial signaling pathways regulated by curcumin. Emerging evidence indicates that curcumin modulates not only classical inflammatory pathways such as nuclear factor kappa B signaling, but also gene networks involved in endothelial glycocalyx integrity, oxidative stress responses, advanced glycation end product–receptor for advanced glycation end product signaling, and epigenetic regulation. Transcriptomic analyses further suggest that curcumin influences non-coding RNA pathways and redox-inflammatory crosstalk through coordinated regulation of antioxidant and immune-related genes. Additionally, single-cell transcriptomics highlights the potential heterogeneity of endothelial responses across different vascular beds. Despite promising findings, limitations including small sample sizes, poor bioavailability, and lack of standardized dosing protocols remain important challenges. Overall, transcriptomic evidence supports the concept of curcumin as a multi-target modulator of diabetic endothelial inflammation and suggests its potential role as an adjunctive therapeutic strategy in diabetes-related vascular disease.

Keywords: Curcuma Longa, Curcumin, Diabetes Mellitus, Endothelial Dysfunction, Transcriptomics, Inflammation

Introduction

Diabetes mellitus is associated with chronic endothelial dysfunction characterized by inflammation, oxidative stress, impaired nitric oxide bioavailability, and vascular injury. Endothelial inflammation plays a critical role in the development of diabetic microvascular and macrovascular complications. Persistent hyperglycemia promotes activation of inflammatory signaling pathways, leukocyte adhesion, oxidative imbalance, and endothelial barrier disruption.

Curcuma longa, commonly known as turmeric, contains curcumin as its principal bioactive compound. Curcumin has attracted considerable attention because of its anti-inflammatory, antioxidant, and vasculoprotective properties. Previous studies demonstrated that curcumin suppresses pro-inflammatory cytokines and inhibits pathways associated with endothelial activation. However, many of these findings were obtained from hypothesis-driven approaches focusing on limited molecular targets.

Recent transcriptomic technologies now provide a systems-level understanding of endothelial responses in diabetes. RNA sequencing and single-cell transcriptomics have enabled identification of broader inflammatory and metabolic networks influenced by curcumin. This short communication summarizes recent transcriptomic evidence regarding the role of Curcuma longa in diabetic endothelial inflammation.

Materials and Methods

This study was designed as a narrative short communication based on current literature evaluating the transcriptomic effects of Curcuma longa and curcumin on diabetic endothelial inflammation. Relevant studies investigating endothelial signaling, RNA sequencing, single-cell transcriptomics, inflammatory pathways, oxidative stress responses, and epigenetic mechanisms were reviewed.

Published experimental and clinical studies focusing on diabetic endothelial dysfunction and transcriptomic alterations associated with curcumin treatment were included. Studies examining inflammatory mediators, endothelial glycocalyx integrity, non-coding RNA regulation, and redox-associated signaling pathways were particularly evaluated.

Results and Discussion

Traditional studies have primarily attributed the vascular effects of curcumin to inhibition of nuclear factor kappa B signaling and suppression of inflammatory cytokines such as interleukin-1 beta, interleukin-6, and tumor necrosis factor-alpha. Curcumin has also been shown to regulate endothelial adhesion molecules including intercellular adhesion molecule-1 and vascular cell adhesion molecule-1, thereby reducing leukocyte-endothelium interactions.

Recent transcriptomic analyses indicate that curcumin exerts broader regulatory effects extending beyond canonical inflammatory pathways. RNA sequencing studies demonstrate modulation of gene clusters associated with endothelial glycocalyx integrity, oxidative stress regulation, and metabolic homeostasis. Preservation of glycocalyx structure is particularly important because glycocalyx degradation is considered an early marker of diabetic vascular injury.

Transcriptomic profiling further suggests that curcumin modulates advanced glycation end product–receptor for advanced glycation end product signaling pathways. Chronic hyperglycemia promotes accumulation of advanced glycation end products, leading to activation of inflammatory transcription factors and endothelial dysfunction. Curcumin appears to attenuate these mechanisms through transcriptional regulation of upstream inflammatory mediators.

Emerging evidence also highlights the role of non-coding RNA pathways in diabetic endothelial inflammation. Curcuminoid derivatives have been associated with regulation of long non-coding RNAs and RNA methylation pathways involved in endothelial injury. These findings support the hypothesis that curcumin may exert epigenetic and post-transcriptional regulatory effects.

Another important observation involves the interaction between oxidative stress and inflammatory signaling. Curcumin activates antioxidant defense systems through the Keap1–Nrf2/antioxidant response element pathway while simultaneously suppressing inflammatory gene expression. This coordinated regulation suggests that curcumin functions as a network-modulating compound rather than a single-target therapeutic agent.

Single-cell transcriptomic approaches additionally reveal endothelial heterogeneity across vascular regions. Curcumin may therefore produce differential molecular responses depending on endothelial subtype and tissue microenvironment. Future integration of spatial transcriptomics and multi-omics approaches may further clarify these compartment-specific effects.

Despite promising results, several limitations remain. Transcriptomic studies evaluating curcumin in diabetic endothelial dysfunction are still limited in scale. Curcumin bioavailability remains low, and standardized therapeutic dosing protocols are lacking. Furthermore, transcriptional changes should be correlated with functional vascular outcomes through integrative proteomic and metabolomic investigations.

Conclusion

Recent transcriptomic evidence substantially expands the mechanistic understanding of Curcuma longa in diabetic endothelial inflammation. Beyond classical inflammatory inhibition, curcumin appears to regulate glycocalyx preservation, advanced glycation end product signaling, oxidative stress responses, and non-coding RNA pathways. These findings support the potential role of curcumin as a multi-target adjunctive therapy in diabetes-related vascular disease. Future large-scale transcriptomic and multi-omics studies are needed to clarify the clinical significance of these molecular mechanisms.

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Competing Interests

The author declares no conflicts of interest.

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