Research Square

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Resveratrol Modulates Inflammatory Gene Expression in LPS Stimulated HMC3 Microglia

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Research Article

Keywords: Resveratrol, HMC3 microglia, neuroinflammation, lipopolysaccharide, TLR4, inflammatory gene expression

Posted Date: October 21st, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11164734/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Activated by LPS, the signaling pathway of TLR4 is triggered in microglial cells, leading to a dramatic increase in the production of inflammatory mediators (IL1B, TNF, CXCL10, and CCL2). Although resveratrol (RSV) has been reported to exert anti-inflammatory effects, its concentration- and time-dependent effects on multiple inflammatory transcripts in human HMC3 microglial cells remain incompletely characterized.

Objective

This study aimed to characterize the dose- and time-dependent effects of resveratrol on IL1B, TNF, CXCL10, and CCL2 expression in LPS-stimulated HMC3 human microglial cells.

Methods

We treated HMC3 cells with LPS (100 ng/mL) and then challenged them with RSV. Dose–response experiments employed 5, 10, and 20 μM RSV for 24 h. We performed time-course analysis using 10 μM RSV at 6, 12, and 24 h. We quantified mRNA levels by SYBR Green qRT-PCR using the $2^{-}\Delta\Delta Ct$ method, with GAPDH as a reference gene. Statistical comparisons were conducted by one-way ANOVA followed by Tukey's post hoc test ($p < 0.05$).

Results

RSV caused a concentration- and time-dependent inhibition of LPS-induced inflammatory gene expression. The highest inhibition was obtained at 10 μM RSV after 24 hours, with IL1B and TNF being inhibited to 50–60% ($p < 0.01$) and CXCL10 and CCL2 to 65–75% ($p < 0.05$) of LPS-induced levels. An additional increase to 20 μM RSV did not increase inhibition, indicating a plateau effect. The time-course experiment demonstrated a gradual inhibition of transcription, with maximal effects at 24 hours.

Conclusion

Resveratrol reduced LPS-induced expression of IL1B, TNF, CXCL10, and CCL2 in HMC3 human microglial cells, with the greatest transcriptional effects observed at 10 μM after 24 hours. These findings support a concentration- and time-associated anti-inflammatory effect of resveratrol at the transcriptional level. However, because we did not directly assess signaling pathways, protein expression, or cell viability, further studies are needed to establish the underlying mechanisms and determine the relationship between transcriptional suppression and cellular function.

Introduction

Several neurological and neuropsychiatric disorders include Alzheimer’s disease, Parkinson’s disease, multiple sclerosis, and major depressive disorder [1]. Microglia are the central nervous system's resident immune cells and play a critical role in maintaining neural homeostasis and responding to injury or infection. Microglia can alter their physiological state and biological functions in response to various stimuli, including infection, cellular injury, and protein aggregation[2].

While physiological microglial activation protects against injury or infection, chronic or unregulated activation can contribute to chronic neuroinflammation and further neuronal damage and pathology[3]. Lipopolysaccharide (LPS), a major component of the outer membrane of Gram-negative bacteria, is widely used as an experimental stimulus to induce inflammatory responses through Toll-like receptor 4 (TLR4) signaling[4]. TLR4 activation triggers intracellular signaling pathways that use both NF-κB and MAPKs. As a result, the cell secretes key molecules that trigger inflammation[5].

IL1B and TNF are key pro-inflammatory cytokines that initiate and amplify inflammatory responses, whereas CXCL10 and CCL2 are chemokines involved in immune-cell recruitment and neuroinflammatory signaling. Accordingly, we selected these four transcripts to provide complementary measures of cytokine- and chemokine-associated inflammatory responses in LPS-stimulated microglia. Uncontrolled regulation of these molecules triggers persistent inflammation in the brain and induces neurological disorders. [6, 7]. Resveratrol (RSV), a naturally occurring polyphenolic compound found in several plant-derived foods, has been reported to exhibit anti-inflammatory and antioxidant activities in experimental models. Previous studies suggest that resveratrol can modulate several inflammatory signaling pathways, including NF-κB, MAPK, and SIRT1-associated pathways. However, the present study did not directly assess these pathways[8].

More recently, RSV has been shown to act as an anti-inflammatory agent in microglial and other model systems. Still, its concentration and time-dependent effects on human microglial gene expression remain undetermined. The concentration- and time-dependent effects of resveratrol on various closely related inflammatory mediators remain poorly characterized under a cohesive experimental design. Given the diverse downstream effects that have been observed, such as cytokine expression, inflammasome activation, metabolic changes, and intracellular signaling, it is unclear if the concentration and time-dependence is the predominant characteristic of resveratrol regulation in human HMC3 microglial cells. Two particular cytokines (IL1B and TNF) and two chemokine mediators of inflammation and immune-cell trafficking (CXCL10 and CCL2) in the response have not had their concentration and time-dependence to RSV measured in concert under any unified experimental designs in an LPS-stimulated human microglial system to fully characterize an appropriate resveratrol concentration and dose-response under an LPS-stimulated regimen. Thus, this project aimed to characterize, systemically, how concentration- and time-dependency affects inflammation gene expression under stimulation of an LPS-driven human microglial model (HMC3). Specifically, we measured gene expression of the mediator genes IL1B, TNF, CXCL10, and CCL2 at varying

concentrations (5, 10, and 20 μM RSV) and over varied time courses at 10 μM RSV (6, 12, and 24 hours). We further determined which concentration and duration best decreased LPS-stimulated gene expression.

Materials and Methods

Cell Culture

We obtained human HMC3 microglial cells from the Iranian National Cell Bank (Tehran, Iran). We cultured cells in DMEM/F12 supplemented with 10% fetal bovine serum (FBS) and 1% penicillin–streptomycin, and maintained them at 37°C in a humidified incubator with 5% CO_2. We refreshed the medium every 2–3 days and passaged cells at 70–80% confluence.

Experimental Design

For dose–response and time-course experiments, we seeded HMC3 cells in 6-well plates and allowed them to attach for 16–18 hours. We assigned cells to the appropriate treatment groups for each experimental arm, including vehicle control, LPS-only, RSV-only, and LPS + RSV conditions.

- **Dose-response experiment**: Cells were treated with RSV at 5 μM, 10 μM, and 20 μM for 24 hours in the presence of LPS (100 ng/mL).
- **Time-course experiment**: Cells were treated with 10 μM RSV in the presence of LPS and harvested at 6, 12, and 24 hours. At each time point, we washed cells with PBS and processed them immediately for RNA extraction or stored them at − 80°C in lysis buffer.

RNA Extraction

We isolated total RNA using the High Pure RNA Isolation Kit (Roche, Germany) according to the manufacturer's instructions, including an on-column DNase treatment step to remove genomic DNA. We assessed RNA concentration and purity spectrophotometrically and used only samples with A260/A280 ratios between 1.8 and 2.0.

cDNA Synthesis

cDNA was synthesized from 1 μg total RNA using a commercial reverse transcription kit (SINACOLON, Iran). We performed reactions under the manufacturer's recommended thermal conditions. cDNA was diluted 1:5 and stored at − 20°C until qRT-PCR analysis.

Primer Sequences and Annealing Conditions

We designed gene-specific primers to target key inflammatory markers in microglia. Table 1 lists the sequences and melting temperatures.

Table 1
Primer sequences used in the study

Gene	Primer Sequence (5' → 3')	Tm (°C)
IL1β	F: AACCTCTTCGAGGCACAAGG	60.0
	R: AGATTCGTAGCTGGATGCCG	
TNF-α	F: GACAAGCCTGTAGCCCATGT	60.0
	R: GGAGGTTGACCTTGGTCTGG	
CXCL10	F: GCTTCCAAGGATGGACCACA	59.8
	R: GCAGGGTCAGAACATCCACT	
CCL2	F: CCCAAGCAGAAGTGGGTTCA	59.5
	R: GTGTCTGGGGAAAGCTAGGG	
GAPDH (housekeeping)	F: TTTTGCGTCGCCAGCC	60.0
	R: ATGGAATTTGCCATGGGTGGA	

qRT-PCR Procedure

qRT-PCR reactions were performed using SYBR Green Master Mix on a real-time PCR system. Each 20 µL reaction contained 10 µL of 2× SYBR Green Master Mix, 0.8 µL of each primer (0.4 µM final), 2 µL of cDNA, and 6.4 µL of nuclease-free water. Thermal cycling: 95°C for 3 min; 40 cycles of 95°C for 15 s, 60°C for 30 s. We performed melt curve analysis from 65°C to 95°C to confirm specificity.

Statistical Analysis

Relative gene expression was calculated using the $2^{-\Delta\Delta Ct}$ method. We set control gene expression to 1.0 and calculated fold changes for each treatment. Data are presented as mean ± SD of three independent biological replicates. One-way ANOVA followed by Tukey's post hoc test was used for multiple comparisons; unpaired Student's t-test was used for two-group comparisons. $P < 0.05$ was considered statistically significant.

Results

In the dose-response experiment, RSV significantly modulated IL1B, TNF, CXCL10, and CCL2 expression in LPS-stimulated HMC3 cells. IL1B and TNF were markedly downregulated at 10 µM RSV (IL1B: 0.55 ± 0.07 fold, p = 0.003; TNF: 0.60 ± 0.06 fold, p = 0.004), while CXCL10 and CCL2 showed moderate

reductions (CXCL10: 0.72 ± 0.08 fold, p = 0.01; CCL2: 0.65 ± 0.05 fold, p = 0.005) compared to LPS-only controls (Fig. 1A).

(B)Time-course analysis of RSV (10 μM) treatment in LPS-stimulated HMC3 cells at 6, 12, and 24 h. Maximal suppression of inflammatory gene expression was observed between 12 and 24 h post-treatment. IL1B and TNF reached their lowest expression levels at 24 h (IL1B: 0.48 ± 0.05, p = 0.002; TNF: 0.52 ± 0.06, p = 0.003). CXCL10 and CCL2 remained consistently reduced throughout the experimental period, with the greatest suppression at 24 h (CXCL10: 0.68 ± 0.07, p = 0.01; CCL2: 0.60 ± 0.06, p = 0.004). Data are presented as mean ± SD (n = 3). Statistical significance was determined relative to LPS-only controls (p < 0.05, *p < 0.01)

Time-course analysis revealed maximal suppression at 12–24 hours post-treatment. IL1B and TNF reached lowest expression at 24 h (IL1B: 0.48 ± 0.05, p = 0.002; TNF: 0.52 ± 0.06, p = 0.003). CXCL10 and CCL2 were consistently reduced throughout 6–24 h, with the greatest suppression at 24 h (CXCL10: 0.68 ± 0.07, p = 0.01; CCL2: 0.60 ± 0.06, p = 0.004) (Fig. 1B). These results suggest that RSV effectively attenuates LPS-induced neuroinflammatory gene expression in human microglial cells.

DISCUSSION

This study systematically analyzed the modulatory effects of resveratrol (RSV) on LPS-stimulated inflammatory gene expression in human HMC3 microglial cells using combined dose-response and time-course analyses. The findings indicate that RSV suppresses LPS-induced expression of IL1B, TNF, CXCL10, and CCL2 in a dose- and time-dependent manner, with maximum potency at 10 μM after 24 hours. LPS stimulation creates a standard model of TLR4 activation by strongly inducing IL1B and TNF expression, indicating the early stages of the inflammatory pathway following NF-κB activation[5]. These cytokines play a crucial role as the main driver of microglial activation by establishing autocrine and paracrine mechanisms that facilitate the inflammatory response[2]. The two chemokines, CXCL10 and CCL2, are NF-κB responsive, but their primary role is to direct immune cell migration while also supporting neuroimmune activity [14]. At 10 μM, RSV more strongly suppressed IL1B and TNF than CXCL10 and CCL2, suggesting that RSV better regulates primary inflammatory transcription events than secondary chemokine responses. The dose-response analysis showed that 10 μM RSV produced the most consistent and significant reduction in inflammatory gene expression. The lack of an additional reduction at 20 μM may indicate a plateau within the tested concentration range.

Polyphenolic compounds show threshold-dependent biological activity because their active signaling components become inactive after reaching a specific concentration[15]. Higher concentrations will not produce additional transcriptional suppression because the primary signaling components have already been blocked[16]. The body uses compensatory signaling systems to control body functions, which limits the ability to further suppress them at higher doses [17]. The study did not conduct formal viability tests, so future research should include viability assays to determine the safe dosage range of RSV in this model. The 20 μM dose did not produce greater suppression because the plateau effect showed

that RSV-responsive regulatory mechanisms reached their maximum capacity. Polyphenolic compounds exhibit biological activity that depends on reaching specific threshold levels, while their primary signaling nodes become blocked, preventing further transcriptional suppression [18].

The body uses compensatory signaling feedback mechanisms to restrict the extent of downregulation that occurs when people use higher drug doses [19]. Cell viability was not formally assessed in the present study and therefore cannot be excluded as a potential contributor to the observed transcriptional changes. Still, future studies should include viability tests to establish RSV safety in this model. The time-course results showed that RSV suppression increased until it peaked after 24 hours. The progressive reduction in inflammatory gene expression over time suggests that RSV may affect transcriptional processes across different phases of the inflammatory response. However, this study did not directly assess the underlying signaling mechanisms[20]. The temporal regulation of IL1B and TNF may reflect secondary regulatory processes following LPS stimulation; however, the present study did not directly investigate these mechanisms. Between 6 and 24 hours, CXCL10 and CCL2 levels gradually decreased, indicating that RSV influences microglial activation in both early and late stages. The combined decrease in IL1B, TNF, CXCL10, and CCL2 suggests that shared upstream signaling pathways are affected [21]. NF-κB represents a plausible upstream regulatory pathway for the inflammatory genes examined in this study; however, its involvement was not directly assessed [22]. Previous studies have suggested that resveratrol may modulate MAPK, SIRT1, and NF-κB-related signaling pathways. Although these pathways may plausibly explain the transcriptional effects observed in the present study, we did not directly evaluate them here.

Therefore, further studies incorporating phosphoprotein analysis, NF-κB nuclear translocation assays, and pathway-specific inhibitors are required to determine whether these mechanisms contribute to the observed effect [23, 24]. The concomitant modulation of four inflammatory transcripts suggests that RSV may exert effects beyond a single inflammatory marker[25]. The research will need to use phospho-protein analysis, NF-κB nuclear translocation assays, and pathway-specific inhibitors. The study shows that concomitant suppression of IL1B, TNF, CXCL10, and CCL2 has significant implications for translational research. Neurodegenerative disorders show a positive association between IL1B and TNF overexpression and synaptic dysfunction and neuronal damage, whereas CXCL10 and CCL2 induce leukocyte infiltration and chronic dissemination of neuroinflammation[26]. RSV has broad-spectrum regulatory effects on inflammatory conditions in the central nervous system because it can suppress the transcription of both cytokine and chemokine genes[27].

Scientists must be cautious when making inferences about living systems because resveratrol has low bioavailability and a short half-life[28]. Several limitations warrant consideration. First, the researchers used an immortalized HMC3 cell line, which cannot accurately reproduce primary human microglia characteristics or the complexities of the central nervous system microenvironment. Second, the researchers measured inflammatory changes through mRNA assessment but did not assess protein levels or functional outcomes. The third point notes that transcriptional strength depends on using a single housekeeping gene, GAPDH, and that additional reference genes would strengthen the

transcriptional analysis. The researchers need to conduct viability and cytotoxicity tests because these tests remain essential for upcoming research to establish safe concentration limits. The results show that resveratrol suppresses the transcription of the IL1B, TNF, CXCL10, and CCL2 genes in human HMC3 microglial cells in response to LPS, in a time- and dose-dependent manner, with the strongest suppression observed at 10 μM after prolonged treatment. These findings provide a basis for further investigation of the molecular mechanisms underlying resveratrol's effects on inflammatory gene expression in human microglial models.

Study Limitations

Several limitations must be taken into account while interpreting the results presented herein. First, the study used the immortalized cell line HMC3. While HMC3 cells are a useful human-derived model, immortalized microglia imperfectly replicate the phenotype, functional diversity, and complex interactions of primary human microglia in the central nervous system. Future studies validating these findings in human microglia or more physiologically relevant systems will be needed to confirm their translational significance, a concern previously noted for HMC3 and other cultured microglial models. Second, only three independent experiments were performed, reducing statistical power and possibly limiting the generalizability of the transcriptomic results. More biological replicates would provide a more robust estimate of variability. Third, changes observed at the transcriptional level do not necessarily reflect decreased protein synthesis of IL1B, TNF, CXCL10, and CCL2 or a functional effect of transcriptomic suppression of these inflammatory mediators, which future protein-level assessments and, where applicable, functional assays could test. Fourth, we did not rigorously investigate cell viability and potential toxicity. As a result, while observed inflammatory gene downregulation could reflect genuine anti-inflammatory activity, we cannot rule out contributions from concentration- and time-dependent effects on cellular viability. Therefore, validation with investigation of cellular viability is necessary when examining dose-responses hereafter, especially given the known effects of resveratrol (concentration- and experimental system-dependent). Lastly, we measured transcripts relative to a single reference gene (GAPDH) and did not directly investigate the signaling pathways responsible for the observed transcriptomic alterations. While we speculate activation of NF-B, MAPK, and SIRT1-related pathways based on prior literature, we did not verify these in the current study. Multiple validated reference genes and further experiments using pathway-specific analyses, including phosphorylation studies, NF-B nuclear translocation, and pathway inhibition, will strengthen future findings, as shown in prior literature.

Declarations

Consent for participation: Not Applicable

Ethics declaration: Not Applicable

Consent for publication: Not Applicable

Data Availability: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Conflict of Interest/ competing interests: The authors declare that they have no known conflict of interest/competing interests.

Funding: No funding was received to assist with the preparation of this manuscript.

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Figures

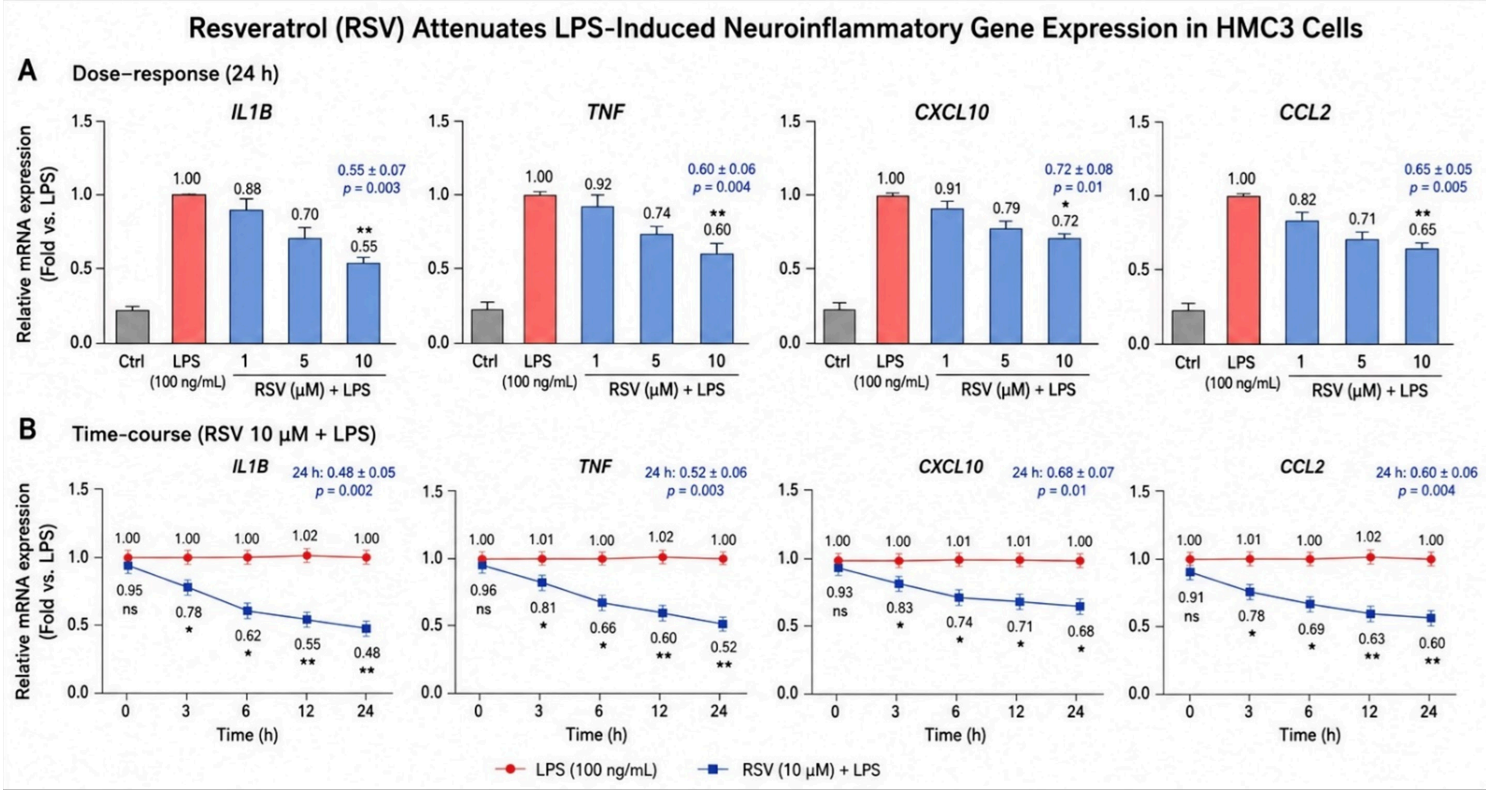


Figure 1

Resveratrol (RSV) attenuates LPS-induced neuroinflammatory gene expression in HMC3 microglial cells. (A) Dose–response analysis of RSV treatment in LPS-stimulated HMC3 cells after 24 h. Relative mRNA expression levels of IL1B, TNF, CXCL10, and CCL2 were measured following treatment with increasing concentrations of RSV (1–10 μM) in the presence of LPS (100 ng/mL). RSV significantly reduced expression of IL1B and TNF at 10 μM (IL1B: 0.55 ± 0.07 fold, p = 0.003; TNF: 0.60 ± 0.06 fold, p = 0.004), while CXCL10 and CCL2 showed moderate but significant reductions (CXCL10: 0.72 ± 0.08 fold, p = 0.01; CCL2: 0.65 ± 0.05 fold, p = 0.005) relative to LPS-only controls.