

Original Research Article

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In Silico Investigation of the Role of Bacterial Metabolites in Vascular Remodeling

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ABSTRACT

Bacterial short-chain fatty acids (SCFAs) are increasingly recognized as microbial metabolites capable of modulating host cellular and inflammatory pathways. However, their potential interactions with proteins involved in vascular remodeling remain incompletely characterized. This study aimed to investigate the potential molecular interactions between selected bacterial SCFAs and key proteins associated with vascular remodeling using an integrated *in silico* approach. Vascular-remodeling- and immune-response-associated proteins were analyzed using the STRING database to construct a protein–protein interaction (PPI) network with a confidence score threshold of 0.700. Gene Ontology (GO) enrichment analysis was performed with an FDR threshold of <0.05 to identify significantly represented biological processes. Key vascular-remodeling-associated proteins identified from the network were subsequently subjected to molecular docking with acetate, propionate, butyrate, and valerate using the AutoDock Vina scoring function through the SwissDock interface. The PPI network revealed extensive functional interactions among proteins involved in inflammation, endothelial function, extracellular matrix organization, angiogenesis, and cytoskeletal dynamics. Gene ontology (GO) enrichment analysis predominantly highlighted biological processes related to inflammatory responses, leukocyte migration and extravasation, endothelial–leukocyte adhesion, and innate immune responses. Molecular docking demonstrated negative predicted binding energies for most SCFA–protein combinations. Butyrate and valerate generally exhibited more favorable docking scores than acetate and propionate. The strongest predicted interaction was observed between valerate and FLT1 (–4.178 kcal/mol), followed by valerate–MYH10 (–3.800 kcal/mol), valerate–ACVRL1 (–3.762 kcal/mol), and valerate–VEGFA (–3.706 kcal/mol). The findings suggest potential molecular interactions between bacterial SCFAs and proteins involved in vascular remodeling, particularly those associated with angiogenesis, extracellular matrix regulation, and cytoskeletal organization. These key findings could be further investigated through experimental studies to elucidate the potential mechanisms underlying the microbiota–metabolite–vascular axis.

Keywords

Bacterial metabolites;
Network
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Vascular remodeling;
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Introduction

Short-chain fatty acids (SCFAs) are microbial metabolites generated primarily through the fermentation of dietary and host-derived substrates. Among them, acetate, propionate, and butyrate are the predominant SCFAs, while other metabolites such as valerate may also function as important molecular signals in host physiology (Shibolet, O *et al.*, 2014). SCFAs influence host cellular processes through multiple mechanisms, including activation of G-protein-coupled receptors and modulation of intracellular enzymes such as histone deacetylases. Through these mechanisms, SCFAs can regulate inflammatory signaling, immune-cell activity, cellular metabolism, endothelial responses, and vascular homeostasis (Mann *et al.*, 2024; Li *et al.*, 2018).

The vascular endothelium represents an important interface between circulating microbial metabolites and the vascular wall. Under physiological conditions, endothelial cells regulate vascular permeability, vascular tone, leukocyte adhesion, platelet activity, and interactions between blood and the vessel wall. Disruption of endothelial homeostasis can promote the expression of adhesion molecules, inflammatory mediators, and signaling molecules involved in vascular remodeling (Caseiro A, *et al.*, 2022; McEver RP *et al.*, 2015). Microbial metabolites may influence endothelial function either through direct interactions with vascular cells or indirectly by altering systemic inflammatory and metabolic pathways (Li *et al.*, 2018). Consequently, changes in microbial composition or metabolite availability associated with intestinal dysbiosis may influence vascular homeostasis (Gao *et al.*, 2026). However, the biological effects of individual SCFAs can differ depending on their concentration, duration of exposure, host metabolic state, and specific molecular targets (Mann *et al.*, 2024).

The relationship between microbial metabolites and vascular remodelling is further influenced by immune signalling. Vascular tissues are continuously exposed to circulating immune cells and soluble inflammatory mediators, which can regulate endothelial function, vascular smooth muscle cell (VSMC) behaviour, and extracellular-matrix remodelling. SCFAs may modify this immune environment and thereby indirectly affect vascular cells and vascular remodelling processes (Li *et al.*, 2018). Conversely, inflammatory conditions and impaired intestinal barrier function can alter the composition and metabolic activity of the gut microbiota,

potentially changing the availability of circulating microbial metabolites. These bidirectional interactions suggest the existence of a microbiota–metabolite–immune–vascular axis linking microbial metabolism with vascular biology (Balint, D., and Brito, I. L., ss 2024).

Although the physiological effects of SCFAs have been investigated in several other biological pathways, their direct molecular interactions with proteins involved in vascular remodelling and immune responses remain insufficiently characterized. Identifying proteins that function at the intersection of vascular-remodelling and immunological pathways may therefore provide insights into potential molecular targets through which microbial metabolites influence vascular biology (Ma *et al.*, 2020; Extracellular Matrix Interactome in Modulating Vascular Homeostasis and Remodeling 2024).

In the present study, protein–protein interaction (PPI) analysis was performed to identify key interacting proteins connecting vascular-remodeling proteins with proteins associated with immune-response pathways. The identified key proteins were subsequently considered potential molecular targets for microbial metabolites. Furthermore, molecular docking was employed to investigate the interactions of four microbial SCFAs—acetate, propionate, butyrate, and valerate—with the identified vascular-remodeling-associated proteins. Molecular docking is a computational approach used to investigate the potential binding modes and interactions of small molecules within protein binding sites (McGann, 2011). This integrated *in silico* approach was used to explore potential molecular interactions underlying the microbiota–metabolite–immune–vascular axis and to provide insights into the possible role of SCFAs in vascular remodeling.

Materials and Methods

STRING-Based Protein–Protein Interaction Network Analysis

Protein–Protein interaction (PPI) network was constructed between the vascular remodelling proteins and the immune response proteins to investigate the functional relationships among genes. The input gene list was submitted to the STRING database, and the resulting interaction network was examined to identify functional associations among the encoded proteins. A specific interaction score of 0.700 was selected as confidence threshold. The experimentally supported and predicted

functional associations were also included during the analysis. (<https://string-db.org>.) (Szkarczyk D, *et al.*, 2021)

Gene Ontology Enrichment Analysis

Gene Ontology (GO) enrichment analysis was performed to identify biological processes that were significantly represented among the genes included in the PPI network. The relative enrichment or signal associated with individual GO terms was used to identify the predominant biological processes represented within the network. A significant cut off of FDR of less than 0.05 were set. The enriched terms were subsequently interpreted in relation to the known functions of the genes and proteins represented in the PPI network. (<https://string-db.org/cgi/network>) (Ashburner M, *et al.*, 2000)

Molecular Docking

The Key vascular proteins identified from protein protein networking were further screened against the Bacterial gut metabolites comprising short chain fatty acids. Molecular docking was performed using the AutoDock Vina docking engine through the SwissDock interface. Target protein structures were retrieved from the Protein Data Bank (PDB) and prepared by removing non-essential water molecules and heteroatoms. Hydrogen atoms were added, and the receptors were converted to PDBQT format. Ligand structures were obtained from suitable chemical databases and prepared in PDBQT format. The docking grid was defined around the reported or predicted active/binding site, with appropriate grid-centre coordinates and dimensions selected to encompass the binding pocket. Docking was performed using the AutoDock Vina scoring function, and the docking poses with highest score were ranked according to predicted binding affinity (kcal/mol). (<https://www.swissdock.ch/>) (Eberhardt J *et al.*, 2021; Trott O *et al.*, 2010)

Results and Discussion

A total of 41 proteins/nodes and 405 functional associations/edges were identified in the STRING-based protein-protein interaction (PPI) network, with an average node degree of 19.8, a local clustering coefficient of 0.758, and a PPI enrichment p-value $< 1.0 \times 10^{-16}$. The network demonstrated extensive functional connectivity among proteins associated with vascular

remodeling, extracellular-matrix organization, inflammatory signaling, endothelial activation, and immune responses.

Several matrix metalloproteinase, including MMP1, MMP3, and MMP9, occupied a highly interconnected region of the network and showed associations with multiple extracellular-matrix and inflammatory proteins. The collagen-associated proteins, including COL1A1, COL1A2, COL3A1, COL4A2, and COL10A1, formed another prominent interaction group, indicating the involvement of extracellular-matrix components in the identified network and also as direct interactors with different Matrix metallo proteinases.

Inflammatory and immune-response proteins, including IL6, IL1A, IL18, IL33, TNF, CCL2, CCL21, CXCL1, and CXCL3, were extensively connected with proteins involved in vascular and endothelial functions. In addition, TLR2 and TLR4 showed associations within the inflammatory signaling component of the network, suggesting functional links between innate immune signaling and vascular remodeling-related proteins. Endothelial and cell-adhesion-associated proteins, including ICAM1, VCAM1, SELE, SELP, ENG, and FLT1, were also interconnected with inflammatory mediators and extracellular-matrix proteins, highlighting the relationship between endothelial activation, immune signaling, and vascular remodeling.

Furthermore, proteins associated with cytoskeletal organization and vascular smooth-muscle functions, such as ACTA2, MYH10, PXN, and RHOA, were incorporated into the network and displayed functional associations with other vascular-remodeling proteins. FN1 also occupied an interconnected position between extracellular-matrix and vascular-associated proteins. The presence of TIMP3 together with MMP proteins further indicates the involvement of the matrix metalloproteinase-tissue inhibitor system in extracellular-matrix regulation. The highly interconnected proteins identified from this network were subsequently considered potential key interactors and candidate targets for further molecular docking analysis with the selected microbial short-chain fatty acids.

Each node represents an individual protein, while edges represent protein-protein interactions. The different edge colors represent distinct sources of interaction evidence: light blue, curated databases; pink, experimentally determined interactions; green, gene neighborhood

evidence; red, gene fusion evidence; dark blue, gene co-occurrence evidence; light green, text-mining evidence; black, co-expression evidence; and purple, protein homology evidence.

GO Biological Process enrichment analysis demonstrated significant representation of biological processes associated predominantly with inflammatory responses, leukocyte trafficking, endothelial interactions, and vascular inflammatory regulation. Among the enriched terms, regulation of neuroinflammatory response showed the highest reported signal value (≈ 3.3), followed by cellular extravasation (≈ 3.0) and leukocyte migration (≈ 2.8). These findings indicated that inflammatory-cell movement and migration across vascular barriers represented major functional themes within the analysed gene set.

Enrichment was also observed for regulation of leukocyte adhesion to vascular endothelial cells and positive regulation of leukocyte adhesion to vascular endothelial cells. These processes are consistent with endothelial activation and the subsequent attachment of circulating leukocytes to the vascular endothelium. The enrichment of regulation of leukocyte migration and positive regulation of leukocyte migration further supported the involvement of chemotactic and adhesion-related mechanisms. The presence of SELE, SELP, ICAM1, VCAM1, CXCL1, CXCR2, CCL2, and CX3CL1 within the analyzed gene set provided a functional basis for the observed enrichment of leukocyte trafficking and cellular extravasation processes.

The GO analysis also identified enrichment for positive regulation of nitric oxide biosynthetic process, suggesting an association between the analyzed genes and pathways involved in endothelial nitric oxide signaling. This finding indicates potential functional involvement in processes related to endothelial vascular regulation. Enrichment for response to lipopolysaccharide was also observed, supporting the representation of innate immune and inflammatory-response pathways within the gene set. This enrichment was consistent with the presence of inflammatory and innate immune regulators such as TLR2, TLR4, TNF, IL1A, IL1B, and IL6.

Although “response to amyloid-beta” and “neuroinflammatory response” were enriched, these findings were not prioritized in the present study, which focused primarily on vascular remodeling and

inflammatory processes. Their enrichment may reflect shared functional annotations rather than direct involvement in amyloid-beta-associated vascular pathology. Collectively, the GO enrichment analysis supported the STRING network findings and highlighted inflammation, leukocyte recruitment and migration, endothelial-leukocyte interactions, cellular extravasation, innate immune responses, and endothelial signaling as major biological processes represented within the analyzed gene set.

The enriched GO terms represent significantly overrepresented biological processes among the analyzed proteins. Dot size represents the number of proteins associated with each GO term, while the color gradient indicates the statistical significance of enrichment based on the adjusted *p*-value

Molecular docking analysis demonstrated a progressive increase in the magnitude of negative binding energies from acetate and propionate to butyrate and valerate for most of the evaluated protein targets. Longer-chain SCFAs, particularly butyrate and valerate, may have a greater propensity to interact with several proteins involved in tissue remodeling and vascular signaling.

Among the extracellular matrix-associated proteins, COL1A1, COL1A2, COL3A1, and COL4A2 exhibited negative binding energies with all four SCFAs. Valerate showed the most favorable docking scores for these targets, with binding energies of -2.370 , -2.623 , -1.856 , and -3.189 kcal/mol, respectively. A similar pattern was observed for FN1, for which the binding energy increased in magnitude from -2.362 kcal/mol with acetate to -3.181 kcal/mol with valerate. These interactions are of potential biological interest because collagen and fibronectin are major structural components of the extracellular matrix and contribute to cellular adhesion, tissue architecture, and remodeling.

Matrix metalloproteinase (MMPs) exhibited a consistent trend toward increasingly favorable docking energies with increasing SCFA chain length. MMP1, MMP3, and MMP9 showed particularly favorable interactions with butyrate and valerate. MMP3 docking energy decreased from -2.307 kcal/mol with acetate to -3.394 kcal/mol with valerate, while MMP9 showed a similar shift from -2.167 to -3.138 kcal/mol. TIMP3 demonstrated an even stronger interaction profile, with docking energies ranging from -2.508 kcal/mol for acetate to -3.503 kcal/mol for valerate.

Table.1 Molecular docking of different protein targets involved in Vascular remodeling against the Short chain fatty acids.

S. No	Protein Targets	Acetate Kcal /Mol	Propionate	Butyrate	Valerate
1	COL1A1	-1.533	-1.930	-2.283	-2.370
2	COL1A2	-2.200	-2.343	-2.506	-2.623
3	COL3A1	-1.452	-1.682	-1.831	-1.856
4	COL4A2	-2.267	-2.679	-2.878	-3.189
5	FN1	-2.362	-2.575	-2.991	-3.181
6	MMP1	-2.003	-2.455	-2.736	-
7	MMP3	-2.307	-2.673	-3.053	-3.394
8	MMP9	-2.167	-2.560	-2.957	-3.138
9	TIMP3	-2.508	-2.740	-3.301	-3.503
10	VEGFA	-2.408	-2.917	-3.362	-3.706
11	FLT1	-2.895	-3.221	-3.739	-4.178
12	KDR	-1.910	-2.333	-2.628	-2.805
13	ENG	-2.475	-2.745	-3.007	-3.115
14	ACVRL1	-2.489	-2.854	-3.385	-3.762
15	ACTA2	-	-	-	-
16	MYH10	-2.543	-2.957	-3.382	-3.800
17	PXN	-1.997	-2.587	-2.610	-2.821
18	RHOA	-2.359	-2.832	-3.183	-3.479

Figure.1 Protein–Protein Interaction Network Revealing Functional Associations among Vascular Remodeling- and Immune Response-Associated Proteins

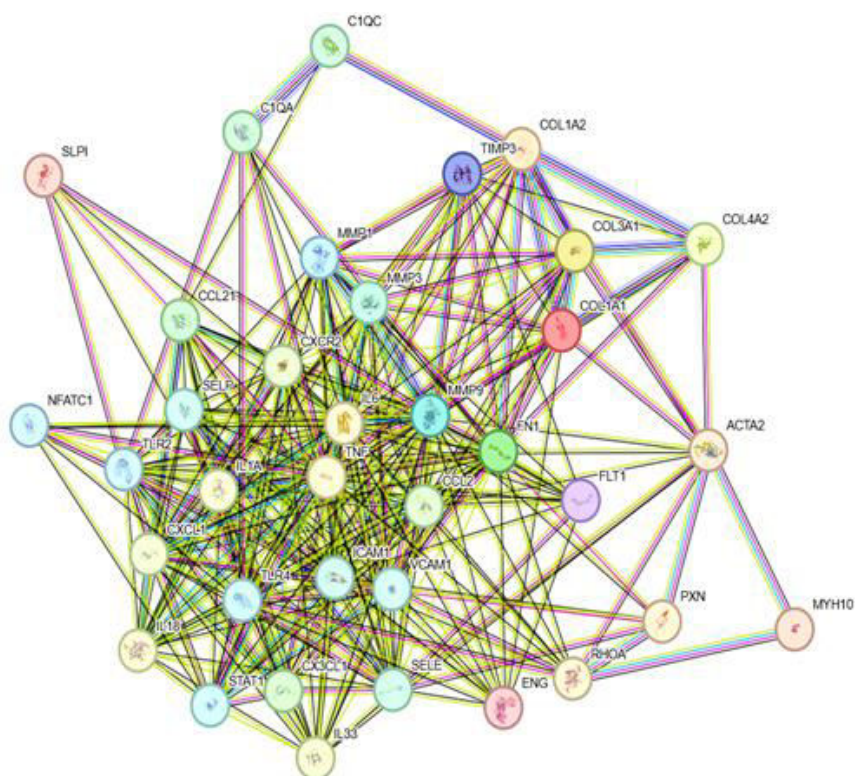
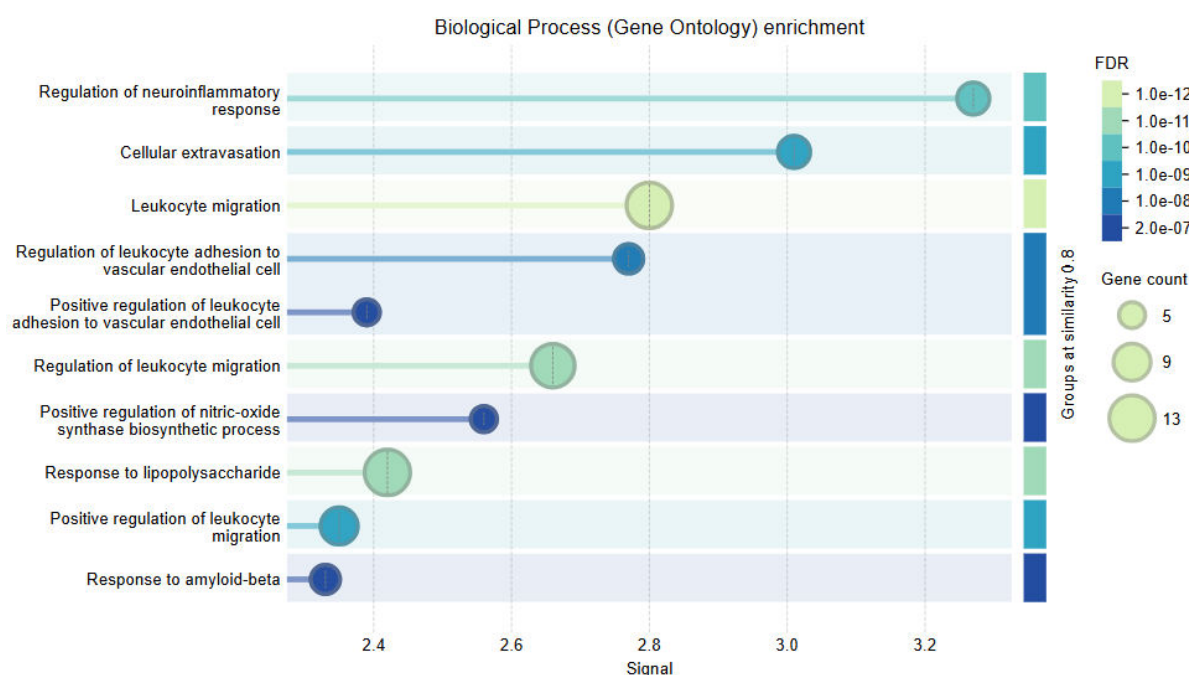


Figure.2 Gene Ontology (GO) enrichment analysis of proteins associated with vascular remodeling and immune responses.



The established roles of MMPs and their endogenous inhibitors in extracellular matrix turnover indicates a potential modulation of matrix-degrading processes by the SCFAs.

The angiogenesis-related targets exhibited some of the most favorable docking scores in the dataset. VEGFA showed progressively stronger predicted interactions from acetate (−2.408 kcal/mol) and propionate (−2.917 kcal/mol) to butyrate (−3.362 kcal/mol) and valerate (−3.706 kcal/mol). Notably, FLT1 displayed the most negative docking energy among all evaluated protein–ligand combinations, reaching −4.178 kcal/mol with valerate.

The concurrent interaction of SCFAs with VEGFA and multiple components of the VEGF receptor signaling axis is noteworthy because these proteins are central regulators of angiogenic signaling. The docking results therefore provide a structural basis for investigating whether SCFAs can influence angiogenesis-associated pathways through direct protein interactions.

The cytoskeletal and contractile-associated targets also displayed differential docking behaviour. MYH10 showed a marked progression in binding energy from

−2.543 kcal/mol with acetate to −3.800 kcal/mol with valerate. The strongest interaction observed in the present dataset was between valerate and FLT1 (−4.178 kcal/mol), followed by valerate–MYH10 (−3.800 kcal/mol), valerate–ACVRL1 (−3.762 kcal/mol), and valerate–VEGFA (−3.706 kcal/mol). This consistent pattern may indicate that increasing carbon-chain length is associated with more favorable interactions within the binding sites examined.

Overall, these findings suggest that the proteins, particularly FLT1, ACVRL1, VEGFA, MYH10, TIMP3, MMP3, MMP9, and FN1 could act as potential molecular interaction partners for SCFAs.

Experimental studies have demonstrated that butyrate and other SCFAs can modulate endothelial inflammatory responses, vascular barrier integrity, vascular remodeling, and extracellular-matrix-related processes, supporting their potential role in vascular homeostasis (Pender SLF *et al.*, 2007; Andrade-Oliveira *et al.*, 2021; Gua *et al.*, 2023; Li *et al.*, 2018; Dardi *et al.*, 2022; Quinqin Tian *et al.*, 2021). Consistent with these observations, the present docking analysis identified several proteins involved in vascular inflammation, angiogenic signaling, extracellular-matrix remodeling,

and cytoskeletal regulation as potential SCFA interaction partners. The predominance of more negative docking energies observed with butyrate and valerate, particularly for MMP3, MMP9, TIMP3, and other vascular-associated proteins, suggests that these metabolites warrant further investigation as potential modulators of vascular remodeling. However, the predicted interactions, particularly those involving FLT1, ACVRL1, MYH10, and TIMP3 requires experimental validation. Further studies incorporating direct binding assays, enzyme or receptor activity assays, and relevant cellular models will be necessary to determine whether these computationally predicted interactions translate into functional effects on vascular remodeling.

In conclusion, the integrated PPI and molecular docking analyses highlight a potential role for SCFAs in modulating molecular networks associated with vascular remodeling. The identified interactions involving MMP1, MMP3, MMP9, TIMP3, FN1, FLT1, MYH10, ACVRL1, and VEGFA suggest potential links between SCFA activity and extracellular-matrix turnover, angiogenic signaling, endothelial function, inflammation, and cytoskeletal organization. The more favorable docking profiles observed for butyrate and valerate further support their potential relevance to these vascular processes. Although these findings provide valuable computational insights, the predicted interactions requires further experimental validation through biochemical and cellular approaches.

Author Contributions

S. Fathima: Conceived the original idea and designed the model; Written, Reviewed and Edited the manuscript. K. Sangeetha: Designed the model and the computational framework and analysed the data; Written, Reviewed and Edited the manuscript.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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