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EVOO Enhance the Expression of Endothelial Marker and Vascular Endothelial Growth Factor in Preeclampsia Placenta Rat Models

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Abstract
Introduction: *Preeclampsia is a pregnancy complication marked by hypertension and proteinuria after 20 weeks of gestation, often leading to maternal and fetal morbidity and mortality. The disorder arises from endothelial dysfunction, causing impaired vascularization, inflammation, and reduced placental perfusion.* ***Objective:*** *This study aimed to analyze the effect of Extra Virgin Olive Oil (EVOO) on endothelial markers CD31, CD34, and Vascular Endothelial Growth Factor (VEGF) expression in a preeclampsia rat model.* ***Method:*** *A post-test-only control group design was applied with five groups: negative control (normotensive pregnant rats), positive control (preeclamptic rats without EVOO), and three treatment groups receiving EVOO at doses of 0.5, 1, and 2 ml/day for 18 days. Immunohistochemical examination was used to assess the expression of CD31, CD34, and VEGF.* ***Result and Discussion:*** *EVOO significantly increased CD31, CD34, and VEGF expression ($p<0.05$) compared to the positive control, with the highest increase at a dose of 2 ml/day. The rise in angiogenic markers indicates endothelial recovery and improved placental vascularization.* ***Conclusions:*** *EVOO has potential as supportive therapy in preeclampsia by enhancing endothelial function and angiogenesis, leading to better placental blood flow*

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Introduction

Preeclampsia is a leading cause of maternal and neonatal morbidity and mortality (Gopman et al., 2024). Increased endothelial apoptosis and decreased regenerative potential may be part of its pathogenesis, as it is linked to systemic endothelial injury or impaired endothelial function (Jung et al., 2022). Normal maternal cardiovascular adaptations to pregnancy include enhanced endothelium-mediated relaxation and reduced vascular responsiveness to certain vasoconstrictors, contributing to a marked decrease in peripheral vascular resistance. Preeclampsia, a pregnancy-specific syndrome, is characterised by the failure of these adaptations, leading to the development of hypertension and proteinuria in the second half of pregnancy. Maternal endothelial cell dysfunction is a primary cause of the peripheral vasoconstriction typical of preeclampsia and the frequent multi-organ damage observed in severe cases (Ferreira et al., 2023; Hu & Zhang, 2021).

Endothelial progenitor cells (EPCs) are released into the bloodstream through nitric oxide (NO)-dependent pathways stimulated by vascular endothelial growth factor (VEGF), placental growth factor (PlGF), estrogen, and other mediators (Chen et al., 2022; Hu & Zhang, 2021). These cells contribute to placental vascular formation through vasculogenesis**.** Vasculogenesis is the formation of new vessels from endothelial precursors distinct from angiogenesis, which involves the growth of new capillaries from existing ones (Chambers et al., 2021; Hassanpour et al., 2023).

The CD34 antigen is expressed by hematopoietic stem cells (HSCs) and endothelial cells(Chambers et al., 2021). CD34 plays an important role in maintaining vascular homeostasis by supporting endothelial regeneration and neovascularization(Hassanpour et al., 2023). In pregnancy, CD34-positive cells contribute to the formation and repair of placental microvasculature, thereby supporting adequate oxygen and nutrient exchange between the mother and fetus (Boss et al., 2023). Reduced CD34 expression has been associated with endothelial dysfunction and impaired angiogenesis, conditions frequently observed in preeclampsia (Kwon & Maeng, 2024). CD31, an adhesion protein, has been widely used as an endothelial marker. It plays an important role in maintaining endothelial integrity, regulating nitric oxide (NO) bioavailability, and controlling vascular permeability and angiogenesis (Chen et al., 2022; Hu & Zhang, 2021). Experimental studies show that loss of CD31 increases vascular permeability and disrupts endothelial function (Türkmen & Akar İnan, 2024).

VEGF is a critical regulator of vascular development and function during pregnancy. It promotes placental angiogenesis and helps maintain maternal vascular stability and permeability (Kornacki et al., 2021). In preeclampsia, VEGF expression becomes dysregulated, leading to impaired placental perfusion and endothelial dysfunction. Recent studies indicate significantly reduced VEGF levels in maternal circulation in preeclampsia, diminishing the critical angiogenic support necessary for a healthypregnancy (Marrero et al., 2024).

In this context, the administration of EVOO or extra virgin olive oil, rich in antioxidants and phenolic compounds, is expected to increased VEGF expression, potentially restoring impaired vascular responses in preeclampsia (Massaro et al., 2020). EVOO is abundant in antioxidants, such as polyphenols, and monounsaturated fatty acids (MUFA), particularly oleic acid, which has demonstrated cardioprotective effects. Additionally, VEGF is known to reduce inflammation and oxidative stress, contributing to improved endothelial function(Alcaide-Hidalgo et al., 2020; Lu et al., 2024). According to a number of studies, regular EVOO consumption may increase the

expression of angiogenic factors and endothelial markers, such as VEGF, CD31, and CD34. Markers CD31 and CD34 play a role in endothelial cell activity essential for vascular tissue regeneration and repair (Njike et al., 2021). Consequently, EVOO administration is expected to not only increase the expression of these markers but also contribute to the maintenance of vascular function and the prevention of complications related to endothelial injury in preeclampsia (Lanza & Ninfali, 2020).

This study utilises endothelial cell markers CD31 and CD34 as well as the pro-angiogenic protein VEGF as indicators of placental vascular formation. The research objectives are 1) to evaluate vascular growth by examining VEGF expression and 2) to measure CD31 expression via immunohistochemistry (IHC). 3) to investigate placental stem cell activity, as indicated by CD34, in preeclampsia, hypothesised to exhibit incomplete spiral artery invasion and reduced endothelial regeneration.

Method

This study was experimental study using a post-test only control group design with in vivo laboratory procedures. The study population consisted of 20 pregnant Wistar strain rats divided into 5 groups. Each group included four replicates. The negative control group was comprised of normal pregnant rats, the positive control group included preeclampsia-induced pregnant rats (preeclampsia rat model); and treatment groups 1, 2, and 3 consisted of preeclampsia-induced pregnant rats receiving three different doses of EVOO at 0.5 ml/day, 1 ml/day, and 2 ml/day, respectively (Rahmawati et al., 2020; Silvani et al., 2020a).

The day following mating was considered as day 1 of pregnancy. Sacrifice of the animals occurred on day 19 of pregnancy. Samples collected for this study included placental tissue and plasma. This research was conducted at the Bioscience Laboratory, Physiology Laboratory, and Molecular Biochemistry Laboratory, Faculty of Medicine, University of Brawijaya, Malang.

Preeclampsia was induced using a NOS inhibitor, L-NAME ($C_7H_{15}N_5O_4 \cdot HCl$) obtained from Sigma-Aldrich (Merck KGaA, Darmstadt, Germany). L-NAME was administered intraperitoneally at a dose of 125 mg/kg body weight from day 13 to day 18 of pregnancy. EVOO "B" was administered orally via a gavage feeding tube from day 1 to day 18 of pregnancy.

Clinical assessments included non-invasive blood pressure measurements (CODA®, Kent Scientific Corporation), available in the Physiology Laboratory, Faculty of Medicine, University of Brawijaya, and proteinuria testing with dipstick strips (URISCAN® 3 GPH). Blood pressure and urinary protein measurements were conducted on days 12, 16, and 19 of pregnancy.

Histopathology preparation involved placental tissue dissection, tissue blocking and sectioning, deparaffinisation, staining (haematoxylin-eosin) with primary antibodies against VEGF, CD31, and CD34, graded alcohol dehydration, clearing, mounting, and coverslipping. Histological slide interpretation was conducted using an Olympus microscope at 400x magnification, focusing on the expression of VEGF, CD31, and CD34 in placental tissue.

Data analysis was conducted using SPSS 25.0. The Shapiro-Wilk test was applied to assess normality, and Levene's test was used for homogeneity of variances. An independent t-test was used to compare the negative and positive control groups, while one-way ANOVA was employed to describe differences across each group.

Result and Discussions

1. Result

EVOO Lowers Blood Pressure in Preeclampsia Rat Models

Table 1

Blood Pressure and Urinary Protein Measurements

Group	Blood Pressure (mmHG) Day 12 Systolic/diastolic	Day 15 Systolic/ Diastolic	Day 19 Systolic/ Diastolic
Negative Control (K–)	116.25/89.25	117/89.75	119.5/89.75
Positive Control (K+)	115.5/89	150.75/109	162.75/117.25
EVOO 0.5 ml/day (D1)	103.5/67.5	147/110.25	137.5/106
EVOO 1 ml/day (D2)	120.25/78	149.75/114.75	123.25/85.25
EVOO 2 ml/day (D3)	120.75/86.5	156.5/131	113.25/81.25

Source: Primary Data 2025

The blood pressure measurements in rats indicate that administration of EVOO in dose groups 1 (D1), 2 (D2), and 3 (D3) over 18 days demonstrates a potential effect in reducing both systolic ($P < 0.001$) and diastolic blood pressure ($P = 0.001$) by day 19.

In dose group 1, systolic blood pressure increased from day 12 to day 15, then decreased by day 19. Diastolic pressure in this group followed a similar pattern, rising on day 15 and slightly decreasing on day 19. In dose group 2, systolic pressure also rose on day 15 but showed a marked decline by day 19. Diastolic pressure in this group exhibited an increase on day 15 followed by a decrease on day 19. In dose group 3, systolic pressure increased on day 15 and then continued to rise by day 19, while diastolic pressure showed a steady decline from day 15 to day 19. Overall, EVOO administration appears effective in lowering blood pressure in certain dose groups after 18 days, with dose groups 2 and 3 demonstrating a significant reduction in both systolic and diastolic pressure by day 19.

Effect of EVOO on CD31 Marker Expression

Table 2

Mean ± SD Expression of CD31 in Placental Tissue of Preeclampsia Rat Models Treated with EVOO

Group	Mean ± SD (CD31)	95% Confidence Interval	F (df=4,15)	p-value
Negative Control (K–)	15.25 ± 3.20	10.16 – 20.34	8.265	0.001**
Positive Control (K+)	9.25 ± 2.06	5.97 – 12.53		
EVOO 0.5 ml/day (D1)	17.50 ± 1.73	14.74 – 20.26		
EVOO 1 ml/day (D2)	20.00 ± 1.83	17.09 – 22.91		
EVOO 2 ml/day (D3)	19.75 ± 5.12	11.60 – 27.90		

Source: Primary Data 2025

The average expression of CD31 shows a significant increase in the treatment groups receiving EVOO compared to the positive control group. Dose group D2 exhibited the highest average expression, followed by D3 and D1, each showing a gradual increase in expression relative to the positive control. The ANOVA analysis of CD31 expression revealed a significant difference between the treatment groups and the positive control group ($p<0.001$). This finding suggests that EVOO administration aids in enhancing CD31 expression in the placenta. Administration of EVOO significantly increased CD31 expression in the preeclampsia rat model, particularly in dose groups 2 and 3. Dose group D2 showed the highest increase in CD31 expression ($p=0.000$), followed by D3 ($p=0.000$), indicating repair of damaged endothelial cells and stimulation of angiogenesis. These results demonstrate that EVOO contributes to endothelial regeneration in preeclampsia conditions by enhancing vascular function in the placenta.

Effect of *EVOO* on CD34 Marker Expression

Table 3

Mean ± SD Expression of CD34 in Placental Tissue of Preeclampsia Rat Models Treated with EVOO

Group	Mean ± SD (CD34)	95% Confidence Interval	F (df=4,15)	p-value
Negative Control (K–)	4.25 ± 1.89	1.24 – 7.26	19.000	0.000***
Positive Control (K+)	3.00 ± 2.45	–0.90 – 6.90		
EVOO 0.5 ml/day (D1)	7.50 ± 1.73	4.74 – 10.26		
EVOO 1 ml/day (D2)	9.50 ± 0.58	8.58 – 10.42		
EVOO 2 ml/day (D3)	14.00 ± 2.71	9.69 – 18.31		

Source: Primary Data 2025

CD34 is a marker of endothelial progenitor cells (EPCs) responsible for vascular regeneration. The marked increase of CD34 expression in EVOO-treated groups suggests that EVOO stimulates endothelial progenitor activity and angiogenesis. The highest expression was seen in the D3 group, indicating that a 2 ml/day dose of EVOO maximally promotes endothelial repair and neovascularization in preeclamptic placentae. CD34 is a marker of endothelial progenitor cells (EPCs) responsible for vascular regeneration. The marked increase of CD34 expression in EVOO-treated groups suggests that EVOO stimulates endothelial progenitor activity and angiogenesis. The highest expression was seen in the D3 group, indicating that a 2 ml/day dose of EVOO maximally promotes endothelial repair and neovascularization in preeclamptic placentae.

Effect of EVOO on VEGF Expression

Table 3
Mean ± SD Expression of VEGF in Placental Tissue of Preeclampsia Rat Models Treated with EVOO

Group	Mean ± SD (VEGF)	95% Confidence Interval	F (df=4,15)	p-value
Negative Control (K–)	11.25 ± 1.71	8.53 – 13.97	37.640	0.000***
Positive Control (K+)	7.50 ± 3.11	2.55 – 12.45		
EVOO 0.5 ml/day (D1)	17.25 ± 1.71	14.53 – 19.97		
EVOO 1 ml/day (D2)	20.00 ± 1.83	17.09 – 22.91		
EVOO 2 ml/day (D3)	24.75 ± 2.50	20.77 – 28.73		

Source: Primary Data 2025

The average expression of VEGF increased significantly in the treatment groups, with the highest increase observed in the highest dose group (D3), followed by D2 and D1. This increase suggests that EVOO administration has the potential to stimulate angiogenesis in the placenta by enhancing VEGF expression, which is essential for improving placental blood flow under preeclampsia conditions.

The ANOVA results for VEGF expression demonstrated a significant difference among the treatment groups ($p<0.001$), with the highest increase in expression observed in group D3. Elevated VEGF expression indicates stimulation of angiogenesis, which can enhance blood perfusion to the placenta and support fetal growth in preeclampsia conditions. EVOO appears to enhance VEGF expression in the placental tissue of rats.

Administration of EVOO significantly increased VEGF expression in the preeclampsia rat model, with the most significant effect observed at the highest dose (D3, $p=0.000$). This increase in VEGF expression suggests stimulation of angiogenesis and vasculogenesis, crucial for improving blood flow to the placenta and supporting fetal development. These results indicate that EVOO has potential in enhancing placental blood flow, an essential factor in managing preeclampsia.

2. Discussion

Effect of EVOO on Blood Pressure Improvement

EVOO contains oleic acid, which increases the production of nitric oxide (NO), a natural vasodilator that helps to widen blood vessels and lower blood pressure in preeclampsia. (Sayec et al., 2021). In preeclampsia, NO levels are reduced, leading to vasoconstriction. EVOO assists in increasing NO bioavailability and reducing vascular resistance. The polyphenols in EVOO, such as hydroxytyrosol and oleuropein, also exhibit strong antioxidant and anti-inflammatory properties. These compounds help neutralise free radicals and reduce inflammation, thus protecting endothelial cells from damage, an essential factor for blood pressure stability (Massaro et al., 2020). Furthermore, EVOO increases the expression of endothelial markers like CD31 and CD34, which suggests better vascular elasticity and endothelial cell regeneration. In preeclampsia, this promotes angiogenesis and helps to lower blood pressure (Lu et al., 2024). Additionally, EVOO supports kidney function, lowers fluid retention, and inhibits

the renin-angiotensin-aldosterone system (RAAS), all of which help to regulate blood pressure and lower proteinuria (Alcaide-Hidalgo et al., 2020).

Effect of EVOO on the Increased Expression of Endothelial Markers

The increased expression of endothelial markers, such as CD31 and CD34, indicates stimulation of angiogenesis, crucial for the formation of new blood vessels and tissue repair in preeclampsia. CD31, also known as platelet endothelial cell adhesion molecule (PECAM-1), is involved in endothelial cell interactions and maintains vascular wall integrity(Türkmen & Akar İnan, 2024). CD34, as a marker of endothelial progenitor cells, signifies endothelial regenerative activity required to restore vascular function compromised by preeclampsia (Marrero et al., 2024). The enhanced expression of these markers with EVOO administration suggests that EVOO can improve damaged endothelial repair, reduce vasoconstriction effects, and support angiogenesis (Sharma et al., 2021).

Effect of EVOO on the Increased Expression of VEGF

VEGF is a key mediator in angiogenesis, playing a critical role in preeclampsia. The increased VEGF expression in the placenta treated with EVOO indicates stimulation of angiogenesis and vasculogenesis processes, vital for supporting placental blood flow and fetal health (Sharma et al., 2021). In preeclampsia, low VEGF levels often lead to tissue damage due to inadequate blood supply. Therefore, increased VEGF expression through *EVOO* administration suggests therapeutic potential for repairing damaged endothelial conditions and enhancing placental perfusion, which may positively impact fetal growth and prevent related complications (Jiwani et al., 2020).

EVOO and Preeclampsia Improvement

Numerous health benefits, including notable cardioprotective effects, have been established by the Mediterranean Diet (MD), which is high in EVOO. By improving endothelial function, the Mediterranean diet, which places a strong emphasis on eating foods high in antioxidants and monounsaturated fats, especially from EVOO, has been demonstrated to lower the incidence of preeclampsia during pregnancy (Silvani et al., 2020). The phenolic content in EVOO acts as a potent anti-inflammatory and antioxidant agent, helping to reduce blood pressure and promoting the development of angiogenic factors like VEGF and CD34, which are crucial for vascular health during pregnancy (Alcaide-Hidalgo et al., 2020; Lu et al., 2024)

Study Limitations and Considerations

Although the findings suggest a beneficial effect of EVOO on vascular improvement and preeclampsia outcomes, several limitations should be acknowledged. First, this study used an animal model (pregnant Wistar rats), and physiological differences between species limit the direct generalization of results to humans. Variations in rat strain response to preeclampsia induction or EVOO dosage may also influence the outcomes, introducing potential bias. Future studies should include human clinical trials to validate these findings, determine optimal dosing, and evaluate long-term safety profiles of EVOO use during pregnancy.

Conclusion

This study demonstrates that EVOO administration in a preeclampsia rat model can enhance the expression of endothelial markers (CD31 and CD34) and VEGF, significantly supporting placental vascular function improvement. The increased expression of these markers indicates strengthened endothelial regeneration, contributing to improved blood flow to the placenta and enhanced fetal health. Therefore, EVOO shows potential as a beneficial adjunct intervention to prevent or mitigate the impact of preeclampsia in high-risk pregnancies.

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