Research Article

Role of Vitamin E in Wound Healing of Rats Fed High-Fat Diet

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Abstract | Since 1990, adolescent obesity has increased fourfold, while adult obesity rates have more than doubled. Obesity can result in diabetes, a condition that promotes small vessel disease, leading to skin damage and impaired wound healing through a prolonged inflammatory phase and promoting oxidative stress. Although the potent antioxidant effects of vitamin E are known to enhance wound healing, its efficacy in obesity-impaired healing remains unexplored. This current study assessed the impact of vitamin E on wound healing in rats subjected to a high-fat diet. Twenty male Wistar rats (12 weeks old) were randomly allocated into four groups (5/each). Group I was administered a standard diet, Group II was given a high-fat diet, Group III received a standard diet with vitamin E supplements, and Group IV was provided a high-fat diet with vitamin E supplements. The total duration of treatment was eight weeks. Last not final, blood samples were collected for HDL, LDL, triglycerides, and cholesterol analysis, while body mass was also measured. Subsequently, the skin was excised. Seven days following skin excision, the wound surface area was measured. The wounded skin was obtained for histopathological analysis, and polymorphonuclear cells were counted. The findings indicated that a high-fat diet and vitamin E supplementation significantly impacted body weight, cholesterol, triglycerides, LDL, and HDL levels ($P<0.05$). The high-fat diet resulted in an increased wound surface area ($P<0.05$), whereas vitamin E treatment reduced polymorphonuclear cell counts ($P<0.05$). Histopathological analysis revealed incomplete wound closure in all groups, characterized by persistent scabs and dermal inflammatory cells. However, vitamin E-supplemented rats showed more extensive reepithelialization. Vitamin E supplementation enhanced lipid profiles and local wound healing in rats on a high-fat diet by reducing inflammation at the wound site, although complete wound closure was not yet achieved.

Keywords | Antioxidant, Lipid profile, Obesity, Oxidative stress, Vitamin E, Wound healing

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INTRODUCTION

Globally, while the prevalence of obese adolescents has increased fourfold since 1990, the number of obese adults has more than doubled. In 2022, over 890 million of the nearly 2.5 billion people who were deemed overweight were obese (WHO, 2024). Modern high-calorie diets and sedentary lifestyles are contributing to the global rise in obesity by causing an energy imbalance that eventually results in weight gain (Ahmed and Mohammed, 2025). Obesity, defined as the excessive accumulation of visceral and subcutaneous fat, leads to increased body weight and health problems (Dias *et al*., 2021). Epidemiological estimations indicate that over 5 million deaths globally in 2019 were caused by high body mass index, suggesting the significant mortality burden associated with obesity-related illnesses (WHO, 2024). Diets high in fat are known to cause obesity and a number of metabolic diseases in both people and animals, including diabetes and heart disease (Eastep and Chen, 2015). Alma *et al*. (2023) state

that these health issues also significantly inhibit the wound healing.

A wound is characterized as damage with disruption of the normal structure and tissue function (Liu *et al*., 2022). The damage can vary from a simple rupture of the epithelium to more severe lesions that extend into the subcutaneous tissue and might impact the muscles, organs, nerves, arteries, and bones (Okur *et al*., 2020). Inflammation, proliferation, and maturation are the three phases of wound healing that preserve tissue structure and function. Many cell types, cytokines, and growth factors interact during each phase (Shin *et al*., 2017; Kopcewicz *et al*., 2020).

The process of wound healing is influenced by numerous factors, frequently classified as local or systemic. Local factors, such as oxygen availability and infection, directly impact the wound site, while systemic factors relate to an individual's overall health and illness condition, including age, hormonal status, stress, diabetes, obesity, and drugs (Rosyid, 2022). Obesity has been associated with a higher risk of wound infection, dehiscence, and death in surgical patients; therefore, the detrimental impact of obesity on wound healing is an important aspect (Arnke *et al*., 2023). According to Yudhistira *et al*. (2022), obesity constitutes a risk factor for an impaired skin barrier. In obesity, hypertrophic adipocytes release pro-inflammatory cytokines that damage the stratum corneum, disrupting its lipid composition and leading to impaired skin barrier function, as evidenced by increased TEWL. A study demonstrated that obesity due to a high-fat diet prolongs the inflammatory phase, thereby impairing wound healing in a rat model (Pierpont *et al*., 2014). A high-fat diet inhibits wound contraction and reepithelialization, increases inflammatory cell migration, and delays myofibroblastic differentiation, collagen deposition, epithelial and connective cell proliferation, and angiogenesis. Short-term high-fat diet consumption can impede wound healing by delaying extracellular matrix deposition and wound closure due to inflammatory phase disruptions (Schaunel *et al*., 2020).

Vitamin E enhances wound healing through its vital antioxidant properties, with α-tocopherol considered the most effective form (Thompson *et al*., 2022). This vitamin also provides protection against metabolic disorders, such as diabetes, cancer, cardiovascular disease, and obesity (Shin *et al*., 2017). The function of vitamin E as the body's primary defense is protecting cell membranes from free radical damage by inhibiting lipid peroxidation (Joshi *et al*., 2023). According to Fatima *et al*. (2025), vitamin E affects skin health by providing antioxidant protection and improving skin structure. It accomplishes this by stabilizing phenoxyl radicals, neutralizing free radicals, facilitating enzymatic regeneration processes, and enhancing collagen synthesis, which collectively diminish oxidative stress and improve skin resilience (Figure 1). Thus, this study evaluated the therapeutic potential of vitamin E for wound healing in high-fat diet-fed rats, specifically assessing its effects on lipid profile, wound surface area, histopathologic figures, and polymorphonuclear cell count.

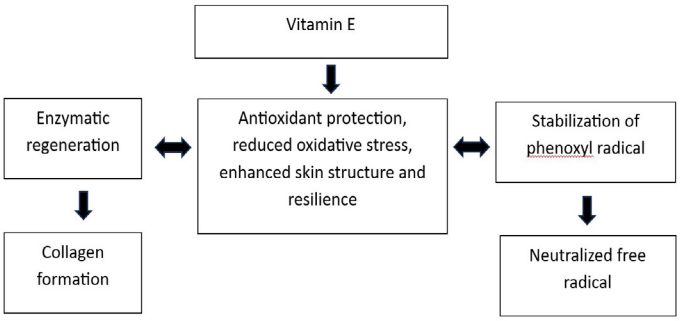

Figure 1: Impact of vitamin E on skin health. Through its ability to stabilize phenoxyl radicals, neutralize free radicals, enhance enzymatic regeneration processes, and encourage the synthesis of collagen, vitamin E boosts the health of the skin. Reduced oxidative stress, improved skin structure and resilience, and antioxidant protection are all facilitated by these activities (Fatima et al, 2025).

MATERIALS AND METHODS

Experimental design

This present study was carried out at the Center for Food and Nutrition Studies, Universitas Gadjah Mada, Yogyakarta, Indonesia. As in a study conducted by Chinko and Precious-Abraham (2024), this study utilized twenty male Wistar rats at twelve weeks old and weighing 200 g on average. They were exposed to a 12-hour light/dark cycle and kept at a steady temperature of 26 °C. The rats were randomly allocated to four groups (I, II, III, and IV) of five animals each. Each rat was housed individually and provided with unrestricted standard diet and water for a week. Upon completion of the adaptation period, the rats were assigned to the following treatment groups: Group I received a standard diet, Group II a high-fat diet, Group III a standard diet plus vitamin E supplementation (Ever E, Konimex, Indonesia) at 300 IU/kg, and Group IV a high-fat diet plus vitamin E supplementation (300 IU/kg). Vitamin E in oil-based form was administered orally using a feeding syringe. The treatment period was 8 weeks, following the previous study by Lasker *et al*. (2019). Table 1 lists the composition of standard and high-fat diets, modified from the AIN-93 diet formulation (Shirai *et al*., 2016).

Excisional wound

After completing the 8-week treatment, all rats were weighed, and samples of blood were taken. Before drawing the blood, the rats were anesthetized by injecting ketamine 50 mg/kg (Ilium Ketamil, Troy Laboratories PTY LTD,

Australia) and xylazine 5 mg/kg (Xyla-Z 2%, BSHM Co., China) intramuscularly. Blood was drawn for triglyceride, cholesterol, LDL, and HDL analysis. Following the blood collection, the hair on the back area was clipped, and the skin was then rubbed with iodine before being excised. Skin excisions were made on the back using a biopsy punch with a diameter of 0.8 cm. Topical chloramphenicol (Ikamicetine, Ikapharmindo, Indonesia) was then applied to the wounds twice daily for 7 days. Seven days after wound excision, all rats were anesthetized, followed by wound surface area measurement.

Table 1: Composition of standard and high-fat diets

Diet	Standard (g/100g)	High-fat (g/100g)
Vitamins	5	5
Cellulose	5	5
Animal fat	4	20
Sucrose	10	10
Corn flour	62	46
Casein	14	14
Total	100	100

Hematoxylin-eosin (HE) staining procedure

The wounded skin was excised and preserved in 10% neutral buffered formalin (Leica Biosystems Richmond, US). Histopathologic examination was performed using hematoxylin and eosin staining. The staining procedure commenced with deparaffinization using three changes of xylene, each for 2 minutes. Subsequent rehydration was performed using a declining ethanol series (100%, 95%, 90%, 80%, and 70%) with 5-minute incubations at each concentration, after which the slides were rinsed in running tap water for 10 minutes. The tissues were subsequently stained with hematoxylin for 5 minutes, rinsed for another 10 minutes, and then stained with eosin for 2 minutes. The slides were then dehydrated in a graded alcohol series, cleared in xylene, and permanently mounted with mounted medium (Yudhika *et al*., 2021).

Wound surface area measurement

A millimeter-grid paper with a central hole was placed over the wound for scale calibration (Supplementary Figure 2). A digital image was captured, and the wound surface area was measured using Digimizer Image Analysis Software.

Polymorphonuclear cell counts

The counting of tissue polymorphonuclear cells was performed by capturing images of the skin histopathological section in four random fields directly beneath the scab, using a microscope equipped with a digital camera (Optilab, Miconos, Indonesia) at a magnification of 400x, as described by Lopez *et al*. (2012).

Statistical analysis

Cholesterol, triglyceride, LDL, HDL, body weight, wound surface area, and polymorphonuclear cell counts were statistically evaluated with two-way ANOVA, followed by Tukey's post hoc test to identify specific group differences, as described by Rosanto *et al*. (2021). A P-value less than 0.05 was considered to be statistically significant. Histopathological figures of the wounded skin were assessed descriptively.

RESULTS

Body weight

Body weight is one of the critical parameters related to the high-fat diet and vitamin E supplementation. The results indicate a significant effect of vitamin E and a high-fat diet on the body weight of rats. Rats maintained on a standard diet exhibited a significantly lower body weight (243.20 ± 7.28 g) than those subjected to a high-fat diet (284.40 ± 28.22 g; P<0.05). Additionally, the body weight of rats administered vitamin E (247.30 ± 11.51) was significantly lower than that of rats not receiving vitamin E (280.30 ± 32.48; P<0.05) (Table 2). This study demonstrated that vitamin E supplementation effectively reduced body weight in both rats on a standard diet and those on a high-fat diet.

Table 2: Mean body weight (in grams) of rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), after eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	249.6±2.70	236.80±3.11	$243.20±7.28^{b}$
High-fat diet	311.00±3.16	257.80±3.56	$284.40±28.22^{a}$
Total mean	$280.30±32.48^{x}$	$247.30±11.51^{y}$	

Different superscripts (a,b) within a column denote significant differences (P<0.05). Different superscripts (x,y) within a row denote significant differences (P<0.05).

Lipid profile

Excessive fat consumption may contribute to an increase in body weight, which can affect blood triglyceride, HDL, LDL, and cholesterol levels. The current study demonstrated that a high-fat diet and the presence of vitamin E affected blood cholesterol levels. Higher cholesterol level (188.26 ± 45.93) was exhibited in rats on a high-fat diet compared to those on a standard diet (87.68 ± 4.38; P<0.05). However, vitamin E supplementation in rats effectively decreased cholesterol levels (114.38 ± 32.55) compared to those that did not receive it (161.57 ± 73.83; P<0.05) (Table 3).

This study demonstrated that both a high-fat diet and vitamin E influenced blood triglyceride levels. Rats subjected to a high-fat diet had markedly higher triglyceride levels (124.57 ± 12.62) compared to those on

a standard diet (70.56 ± 7.05; P<0.05). In contrast, rats administered vitamin E exhibited significantly reduced triglyceride levels (88.91 ± 26.40) compared to those not receiving vitamin E (106.22 ± 31.09; P<0.05) (Table 4).

Table 3: Mean blood cholesterol levels (mg/dL) of rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), after eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	91.57±1.59	83.80±1.71	87.68 ± 4.38^{b}
High-fat diet	231.57±3.71	144.96±6.63	188.26±45.93^{a}
Total mean	161.57±73.83^{x}	114.38±32.55^{y}	

Different superscripts (a,b) within a column denote significant differences (P<0.05).Different superscripts (x,y) within a row denote significant differences (P<0.05).

Table 4: Mean blood triglyceride levels (mg/dL) of rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), after eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	76.85 ± 2.88	64.27 ± 2.15	70.56 ± 7.05^{b}
High-fat diet	135.57±3.31	113.56±6.67	124.57±12.62^{a}
Total mean	106.22±31.09^{x}	88.91±26.4^{y}	

Different superscripts (a,b) within a column denote significant differences (P<0.05). Different superscripts (x,y) within a row denote significant differences (P<0.05).

Table 5: Mean Low-Density Lipoprotein (LDL) levels (mg/dL) of rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), after eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	29.34±1.43	26.16±1.33	27.75±2.12^{b}
High-fat diet	75.98±1.50	52.18±2.57	64.08±12.70^{a}
Total mean	52.66±24.62^{x}	39.17±13.85^{y}	

Different superscripts (a,b) within a column denote significant differences (P<0.05). Different superscripts (x,y) within a row denote significant differences (P<0.05).

Table 6: Mean High-Density Lipoprotein (HDL) levels (mg/dL) of rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), after eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	77.94 ± 2.49	84.71 ± 2.57	81.32 ± 4.29^{a}
High-fat diet	28.09±1.21	44.26 ± 1.41	36.18 ± 8.61^{b}
Total mean	53.02±26.33^{y}	64.49±21.4^{x}	

Different superscripts (a,b) within a column denote significant differences (P<0.05). Different superscripts (x,y) within a row denote significant differences (P<0.05).

The means of LDL and HDL levels are shown in Tables 5 and 6. Rats consuming a high-fat diet had the levels of LDL (64.08 ± 12.70) and HDL (36.18 ± 8.61) that were markedly different from those in rats on a standard diet (P<0.05). In this study, the high-fat diet increased LDL levels while decreasing HDL levels (P<0.05). Moreover, vitamin E supplementation affected LDL (39.17 ± 13.82) and HDL (64.49 ± 21.40) levels, whereas vitamin E decreased LDL levels and increased HDL levels significantly (P<0.05).

Wound surface area

On the seventh day following excision, wound surface areas were assessed. As indicated in Table 7, the wound surface areas in rats fed a high-fat diet were significantly larger (57.42 ± 11.02) than those in rats fed a standard diet (36.96 ± 9.31; P<0.05). Meanwhile, vitamin E treatment did not significantly affect wound surface area.

Table 7: Mean wound surface area (mm^2) after seven days of excision in rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), following eight weeks of treatment

	w/o vit E	w/vit E	Total mean
Standard diet	40.96 ± 9.93	32.96 ± 7.50	36.96 ± 9.31^{b}
High-fat diet	58.92 ± 11.04	55.92 ± 12.07	57.42 ± 11.02^{a}
Total mean	49.94 ± 13.69	44.44 ± 15.37	

Different superscripts (a,b) within a column denote significant differences (P<0.05)

Polymorphonuclear cell counts

According to the results, a high-fat diet had no effect on polymorphonuclear cell counts, while vitamin E supplementation significantly reduced them. Rats supplemented with vitamin E had significantly lower polymorphonuclear cell counts (53.17 ± 17.87) compared to those not supplemented with vitamin E (86.15 ± 18.60; P<0.05) (Table 8).

Table 8: Mean polymorphonuclear cell counts after seven days of excision in rats fed standard or high-fat diets, with or without vitamin E supplementation (300 IU/kg), following eight weeks of treatment.

	w/o vit E	w/vit E	Total mean
Standard diet	78.50 ± 21.53	47.55 ± 18.45	63.03 ± 24.97
High-fat diet	93.80 ± 12.93	58.80 ± 17.30	76.30 ± 23.41
Total mean	86.15± 18.6^{x}	53.17± 17.87^{y}	

Different superscripts (x,y) within a row denote significant differences (P<0.05).

Histopathologic figure of wound

Histopathological analysis of excision wounds (Figure 2) revealed incomplete wound closure beneath scabs in

all groups. However, the vitamin E-supplemented group exhibited more extensive re-epithelialization, despite the persistent presence of inflammatory cells in the dermis (Supplementary Figure 1)

DISCUSSION

This present study indicated a substantial rise in rat body weight following consumption of a high-fat diet, suggesting a disruption in fat metabolism. This aligns with established studies, as high-fat diets promote body fat mass accumulation and body weight in both humans and animals due to their high energy density (Eastep and Chen, 2015; Yang *et al*., 2018; Diaz *et al*., 2021; Nasution *et al*., 2022). The crucial regulator in this process is adiponectin, a hormone that stimulates fatty acid oxidation and glucose utilization to reduce body weight (Ma *et al*., 2016). Obesity is characterized by decreased adiponectin levels, resulting from adiponectin resistance and reduced receptor expression (Shen *et al*., 2010; Soliman *et al*., 2014). The reduction in body weight following vitamin E supplementation observed in this study suggests that α-tocopherol upregulates adiponectin, leading to weight reduction (Hendarto *et al*., 2019; Emami *et al*., 2021).

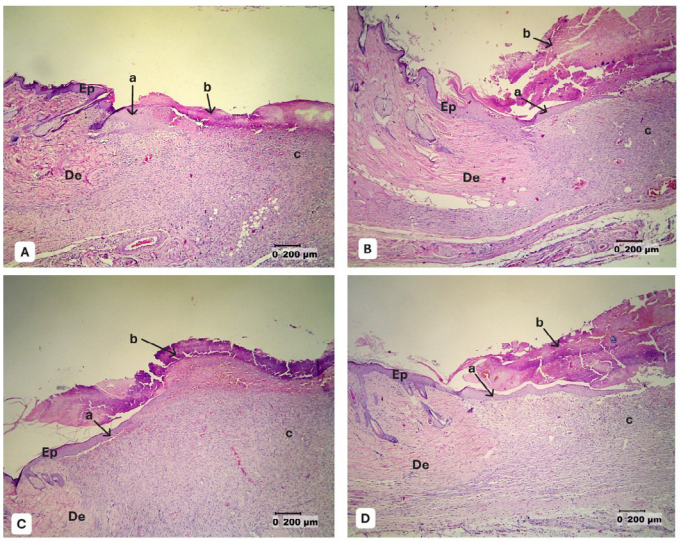

Figure 2: Histopathological figures of wounded skin on day 7 from rats fed a standard diet (A), a high-fat diet (B), a standard diet with vitamin E supplementation (C), and a high-fat diet with vitamin E supplementation (D). The epidermis (Ep) in the wound area had begun to regenerate, while reepithelialization (a) was more extensive in the vitamin E-supplemented rats. All wounds were still covered with scabs (b), and numerous inflammatory cells (c) remained in the dermis (De) (hematoxylin and eosin, 40×)

Consistent with established findings that a high-fat diet causes dyslipidemia (Du *et al*., 2021), this study confirmed that a high-fat diet elevated blood cholesterol, triglycerides, and LDL while lowering HDL. Vitamin E supplementation effectively counteracted these effects. The liver enzyme responsible for cholesterol production, HMG-CoA reductase, is inhibited by vitamin E, thereby preventing the accumulation of cholesterol and lowering cholesterol levels (Emami *et al*., 2021). According to Kim *et al*. (2013), a high-fat diet induces oxidative stress, leading to insulin resistance and elevated triglycerides. This occurs as insulin resistance fails to suppress free fatty acid release, boosting VLDL synthesis, which subsequently raises triglyceride levels. However, vitamin E improves this condition by upregulating PPAR, a receptor that enhances hepatic fat metabolism and reduces serum triglycerides (Megawati *et al*., 2021).

In order to evaluate the effects of a high-fat diet and vitamin E supplementation on the wound healing process, this study conducted an excisional wound model that healed by second intention. On the seventh day following the injury, the wound surface area was measured. The findings revealed that rats on a high-fat diet had significantly larger wound surface area. According to Pierpont *et al*. (2014), fat tissue becomes hypoxic in response to an inadequate blood supply, which is caused by disturbed angiogenesis and chronic inflammation due to excess body fat. Despite fibroblast survival in low oxygen, their function is compromised, leading to inadequate collagen synthesis. This limits collagen production, preventing the regeneration of a robust collagen matrix, reducing its mechanical strength, and impairing wound healing. Although the current study found no statistically significant effect of vitamin E on the wound surface area, a tendency toward reduction was observed. This aligns with established mechanisms whereby vitamin E facilitates wound healing by neutralizing free radicals, reducing proinflammatory cytokines (Emami *et al*., 2021), and enhancing immune function such as phagocytosis (Hobson, 2014; Lewis *et al*., 2019).

In this study, vitamin E significantly reduced tissue polymorphonuclear cell counts, likely due to its anti-inflammatory and antioxidant properties. This is consistent with findings that vitamin E suppresses proinflammatory cytokines like TNF-α, IL-1β, and IL-6 (Salinthone *et al*., 2013), countering the inflammatory state induced by a high-fat diet (Bae *et al*., 2023).

Histopathological examination of excision wounds demonstrated incomplete wound closure beneath scabs among all groups. Nevertheless, the group receiving vitamin E supplementation showed enhanced re-epithelialization, despite the ongoing presence of inflammatory cells in the dermis. Schaunel *et al*. (2020) have demonstrated that an obesity-induced high-fat diet adversely impacts wound healing by extending the inflammatory phase. A high-fat diet not only facilitates the migration of

inflammatory cells and disrupts wound contraction but also inhibits myofibroblast differentiation, diminishes collagen deposition, impairs the proliferation of epithelial and connective cells, and compromises angiogenesis. The degree and duration of obesity contribute to the levels of proinflammatory cytokines, mononuclear and polymorphonuclear cells, and macrophages, which lead to pathological alteration in inflammation (Pierpont *et al*., 2014; Cotterell *et al*., 2024).

The role of antioxidants is to protect cells from ROS-induced oxidative damage (Fatima *et al*., 2025). Vitamin E is a major fat-soluble antioxidant that protects cell membranes by scavenging free radicals and inhibiting lipid peroxidation. Additionally, it promotes skin health by maintaining the integrity of the skin barrier through regulated TEWL and enhanced lipid synthesis, as well as by modifying inflammatory pathways to lower pro-inflammatory cytokines (Fatima *et al*., 2025; Joshi *et al*., 2023). Even though all groups showed incomplete wound closure, the re-epithelialization was improved by vitamin E administration. This improvement is probably the result of vitamin E's dual function as an antioxidant that reduces oxidative stress caused by ROS and its anti-inflammatory properties that influence healing processes even when dermal inflammation is ongoing.

CONCLUSION

This study demonstrated that vitamin E supplementation decreased the body weight of rats on both standard and high-fat diets. In addition to its effects on weight, vitamin E positively improved the lipid profile, as shown by reduced cholesterol, triglycerides, and LDL levels, and increased HDL levels. At the wound site, the healing process showed marked improvement, as indicated by a reduced number of polymorphonuclear cells, highlighting the beneficial anti-inflammatory and antioxidant properties of vitamin E. Although complete wound closure was not achieved during the observation period, the anti-inflammatory properties of vitamin E indicate a potential mechanism to alleviate obesity-related healing problems. Accordingly, further research is needed to investigate its clinical potential.

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NOVELTY STATEMENT

The efficacy of vitamin E in the context of obesity-impaired healing remains unexplored, presenting a clear gap in knowledge that this study aims to address by testing this established agent under compromised conditions

AUTHOR'S CONTRIBUTION

DA conceptualized the study, drafted the manuscript, and performed data analysis. AP contributed to the investigation, data interpretation, and reviewed the manuscript. MER participated in validation and editing the manuscript. All authors reviewed and approved the final manuscript.

Ethical approval

The Ethical Clearance Commission of Universitas Gadjah Mada's Integrated Research and Testing Laboratory approved this study (No. 00019/04/LPPT/VII/2022), and all activities were performed in compliance with relevant regulations and recommendations.

Abbreviations

ANOVA, Analysis of variance; HDL, High-density lipoprotein; HMG-CoA, 3-hydroxy-3-methylglutaryl coenzyme A; IL-1β, Interleukin-1β; IL-6, Interleukin-6; LDL, Low-density lipoprotein; PPAR, Peroxisome proliferator-activated receptors; ROS, Reactive oxygen species; TEWL, Transepidermal Water Loss; TNF-α, Tumor necrosis factor-α.

Generative AI and AI-assisted technology statement

Generative AI and AI-assisted technologies were utilized for grammar and style checking, as well as to refine author-written paragraphs for improved clarity. The authors thoroughly reviewed, verified, and edited all AI-generated output to ensure the accuracy and integrity of the scientific content.

Conflict of interest

The authors have declared no conflict of interest.

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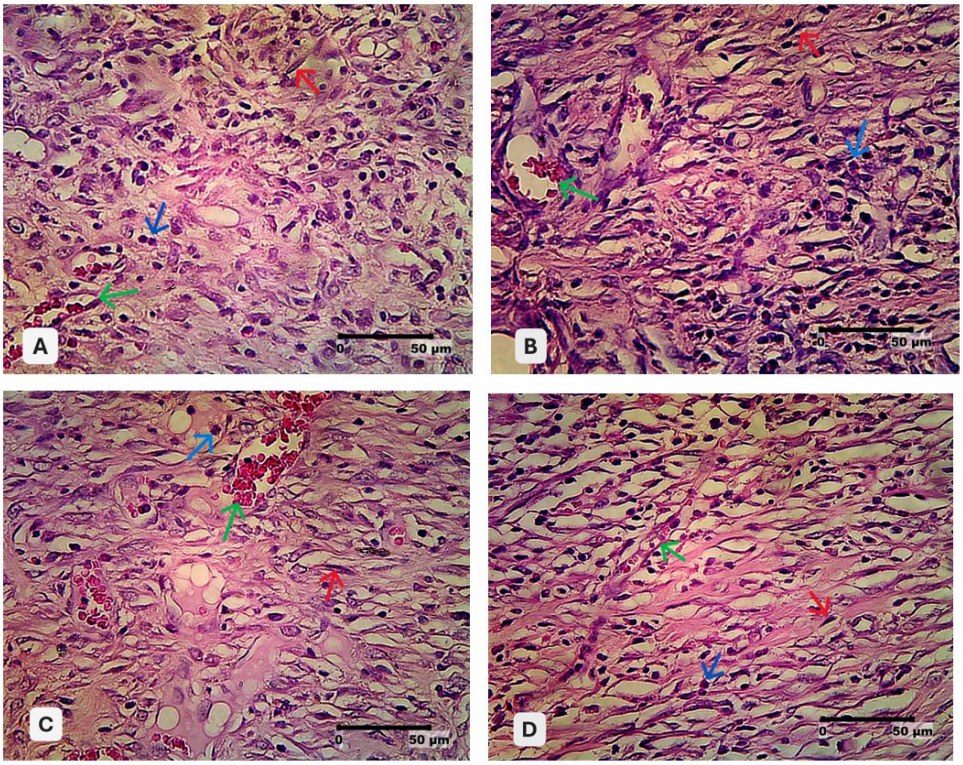
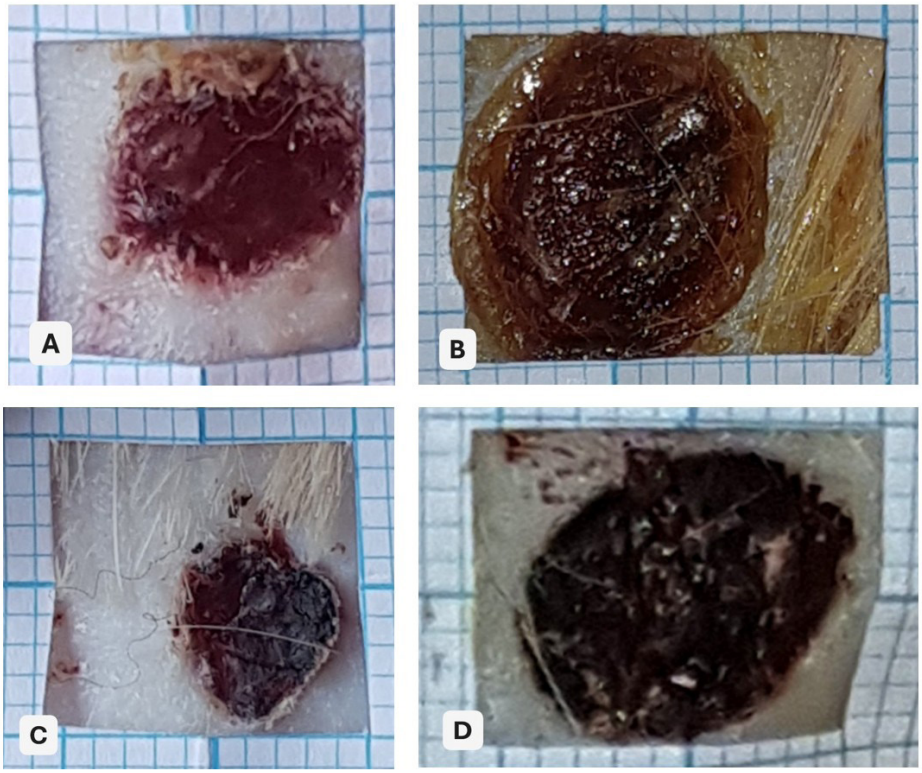


Supplementary Figure 1: Histopathological figures of wounded skin on day 7 from rats fed a standard diet (A), a high-fat diet (B), a standard diet with vitamin E supplementation (C), and a high-fat diet with vitamin E supplementation (D). The dermis of the wound area showed granulation tissue that was composed of polymorphonuclear cells (blue arrow), blood vessels (green arrow), and fibroblasts (red arrow). The number of polymorphonuclear cells was fewer in rats supplemented with vitamin E.



Supplementary Figure 2: Macroscopic figures of wounded skin on day 7 from rats fed a standard diet (A), a high-fat diet (B), a standard diet with vitamin E supplementation (C), and a high-fat diet with vitamin E supplementation (D). The wound surface area of rats fed a high diet was larger than that of rats fed a standard diet; however, the wound surface area tends to be smaller in rats supplemented with vitamin E.