The Effect of Topical Virgin Coconut Oil on Striae Gravidarum Prevention and Severity: A Randomized Controlled Trial

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1. Introduction

Striae gravidarum (SG), also known as stretch marks, is a common dermatological condition that affects a significant proportion of pregnant women. Characterized by linear atrophic streaks on the skin, SG primarily appears on the abdomen, breasts, thighs, and buttocks. These marks are a source of considerable distress for many women, impacting self-esteem, body image, and overall quality of life during and after pregnancy. The prevalence of SG varies depending on the population studied and the assessment methods used. However, estimates suggest that 50% to 90% of pregnant women develop SG at some point during their gestation. The prevalence tends to be higher among younger women, those with a higher pre-pregnancy body mass index (BMI), and those experiencing greater weight gain during pregnancy. Additionally, certain ethnic groups, such as those of African and Hispanic descent, appear to have a higher predisposition to developing SG. Pathophysiology. The precise pathophysiology of SG remains incompletely understood, but it is believed to involve a complex interplay of hormonal, mechanical, and genetic factors. During pregnancy, the skin undergoes significant stretching and hormonal fluctuations, particularly in cortisol and estrogen levels. These changes lead to alterations in the extracellular matrix, primarily affecting the production and degradation of collagen and elastin fibers. Collagen, the main structural protein of the skin,
provides strength and support, while elastin confers
elasticity. In SG, there is a decrease in collagen
synthesis and an increase in its breakdown, coupled
with a reduction in elastin content. This results in the
weakening and fragmentation of the connective tissue,
leading to the formation of visible scars.1,2

Several risk factors have been identified for the
development of SG. Family history of SG is a strong
predictor of their occurrence in subsequent
generations, suggesting a genetic component.
Excessive weight gain during pregnancy and a higher
pre-pregnancy BMI increase the risk of SG by placing
greater mechanical stress on the skin. Elevated
cortisol levels during pregnancy suppress collagen
synthesis, while increased estrogen levels may inhibit
fibroblast proliferation, contributing to the
development of SG. Younger women are more
susceptible to SG due to the relative immaturity of
their skin and its lower tolerance to stretching. Women
of African and Hispanic descent have a higher
prevalence of SG, likely due to differences in skin
composition and genetics. SG can have a profound
psychological impact on pregnant women. Studies
have shown that women with SG often experience
decreased self-esteem, body dissatisfaction, and
negative feelings about their appearance. These
psychological effects can persist postpartum and may
contribute to postpartum depression and anxiety.
Additionally, SG can interfere with intimate
relationships and sexual functioning.3,4

Virgin coconut oil (VCO), derived from the fresh
meat of mature coconuts, has been used for centuries
in traditional medicine for its various therapeutic
properties. In recent years, it has gained popularity as
a natural skincare product due to its potential benefits
for skin health. VCO is rich in medium-chain fatty
acids (MCFAs), including lauric acid, which has
antimicrobial and anti-inflammatory properties. It also
contains polyphenols and vitamin E, both of which
possess antioxidant activity. VCO has been shown to
have several potential mechanisms of action that may
contribute to its beneficial effects on skin health. The
polyphenols and vitamin E in VCO scavenge free
radicals, protecting the skin from oxidative damage
caused by environmental stressors. Lauric acid and
other MCFAs have been shown to reduce inflammation
in the skin, which may be beneficial in preventing and
treating various skin conditions. VCO has been shown
to promote wound healing by stimulating collagen
synthesis and reducing inflammation. VCO forms a
protective barrier on the skin, helping to retain
moisture and prevent dryness.5,6

While anecdotal evidence and traditional practices
suggest that VCO may be effective in preventing and
reducing the severity of SG, there is a lack of rigorous
scientific evidence to support these claims. Existing
studies on the use of VCO for SG are limited by small
sample sizes, methodological flaws, and the lack of a
control group. Therefore, this randomized controlled
trial (RCT) aims to provide high-quality evidence on the
efficacy and safety of topical VCO in preventing and
reducing the severity of SG among pregnant women.
This study is particularly relevant in Kerinci Regency,
Indonesia, where VCO is readily available and widely
used in traditional medicine. Conducting this RCT in
a culturally relevant setting will enhance the
generalizability and applicability of the findings to the
local population. Additionally, this study will
contribute to the growing body of evidence on the use
of natural products for skin health during pregnancy,
providing valuable information for both healthcare
providers and pregnant women.

2. Methods

This study employed a randomized, double-blind,
placebo-controlled trial design, the gold standard for
evaluating the efficacy of interventions. This design
minimizes bias and ensures the comparability of the
intervention and control groups, thereby
strengthening the validity of the study results. The
study was conducted in Kerinci Regency, Indonesia.
Kerinci Regency was chosen due to several factors.
First, it has a high prevalence of striae gravidarum
among pregnant women, making it a suitable setting
for studying this condition. Second, VCO is readily
available and widely used in traditional medicine in
this region, ensuring the cultural relevance and feasibility of the intervention. Third, the healthcare infrastructure in Kerinci Regency is well-developed, allowing for the recruitment of a representative sample of pregnant women and the reliable collection of data. Pregnant women in their second trimester (14–27 weeks of gestation) were recruited from antenatal clinics in Kerinci Regency. Inclusion criteria included: Age between 18 and 35 years; Singleton pregnancy; No prior history of SG; No known allergies to coconut or its derivatives and Willingness to provide informed consent and comply with study procedures. Exclusion criteria included: Multiple pregnancies; Pre-existing skin conditions (e.g., eczema, psoriasis); Use of topical medications on the abdomen, breasts, thighs, or buttocks; Any medical condition that could interfere with the study or pose a risk to the participant or fetus.

Participants were randomized to receive either topical VCO or a placebo (standard emollient cream without active ingredients). Both the VCO and placebo were packaged in identical, opaque containers to ensure blinding. Participants were instructed to apply a thin layer of the assigned product twice daily (morning and evening) to their abdomen, breasts, thighs, and buttocks, starting from the time of randomization until six weeks postpartum. VCO Application Protocol: Dose: 5 mL (approximately 1 teaspoon) of VCO per application; Frequency: Twice daily; Area of Application: Abdomen, breasts, thighs, and buttocks; Duration: From the time of randomization until six weeks postpartum. The placebo was a standard emollient cream without any active ingredients. It was similar in appearance, texture, and smell to the VCO to ensure blinding.

The primary outcome was the incidence and severity of SG, assessed using a modified striae gravidarum assessment scale (SGAS) at baseline, 32 weeks of gestation, and six weeks postpartum. The SGAS is a validated tool that assesses the severity of SG based on the number, length, width, color, and texture of the marks. Secondary outcomes included: Skin hydration: Measured using a corneometer at baseline, 32 weeks of gestation, and six weeks postpartum; Skin elasticity: Measured using a cutometer at baseline, 32 weeks of gestation, and six weeks postpartum. A sample size of 200 participants (100 per group) was calculated to detect a 20% difference in the incidence of SG between the VCO and placebo groups, with a power of 80% and a significance level of 0.05. This calculation was based on previous studies and assumed a dropout rate of 20%. Participants were randomized to the VCO or placebo group using a computer-generated randomization list. Allocation concealment was ensured by using sequentially numbered, opaque, sealed envelopes. Blinding of participants and assessors was maintained throughout the study. Data were analyzed using SPSS software. Descriptive statistics were used to summarize baseline characteristics and outcome measures. Chi-squared tests were used to compare the incidence of SG between the VCO and placebo groups. T-tests and analysis of variance (ANOVA) were used to compare continuous variables between the groups. A p-value of less than 0.05 was considered statistically significant.

3. Results and Discussion

Table 1 presents the baseline characteristics of the participants enrolled in the study, comparing the virgin coconut oil (VCO) group and the placebo group. The table includes demographic data such as age, parity (number of previous pregnancies), and pre-pregnancy body mass index (BMI), as well as the gestational age at enrollment. The two groups were similar in terms of age, parity, pre-pregnancy BMI, and gestational age at enrollment. This is indicated by the high p-values (>0.05) for all these variables. This similarity is important as it suggests that any observed differences in outcomes between the groups are likely due to the intervention (VCO or placebo) rather than pre-existing differences between the participants. The mean age of 26.5 years and the majority of participants being multigravida (62%) indicate that the sample is representative of the typical pregnant population in Kerinci Regency, Indonesia. This enhances the generalizability of the study findings to
the target population. The mean pre-pregnancy BMI of 23.5 kg/m² falls within the normal weight range. However, previous studies have shown that even within the normal BMI range, there is a risk of developing striae gravidarum (SG). This highlights the importance of investigating preventive measures like VCO for a wider range of pregnant women, not just those with high BMIs. The mean gestational age at enrollment of 20.5 weeks suggests that the intervention was initiated during the second trimester, a period when SG typically begins to appear. This is a crucial time for intervention, as preventing the onset of SG is likely easier than treating existing marks. Overall, Table 1 demonstrates the successful randomization of participants and the comparability of the VCO and placebo groups at baseline. This strengthens the internal validity of the study and increases confidence in the study results.

Table 1. Baseline characteristics of participants.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>VCO (n=100)</th>
<th>Placebo (n=100)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>26.4 (4.3)</td>
<td>26.6 (4.1)</td>
<td>0.78</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primigravida: 38</td>
<td></td>
<td>Primigravida: 36</td>
<td>0.72</td>
</tr>
<tr>
<td>Multigravida: 62</td>
<td></td>
<td>Multigravida: 64</td>
<td></td>
</tr>
<tr>
<td>Pre-pregnancy BMI</td>
<td>23.4 (3.7)</td>
<td>23.6 (3.9)</td>
<td>0.65</td>
</tr>
<tr>
<td>Gestational age</td>
<td>20.4 (3.2)</td>
<td>20.6 (3.0)</td>
<td>0.59</td>
</tr>
</tbody>
</table>

Table 2 presents the primary outcomes of the study, namely the incidence and severity of striae gravidarum (SG) in the virgin coconut oil (VCO) group and the placebo group at two key time points: 32 weeks of gestation and six weeks postpartum. The incidence of new SG was significantly lower in the VCO group compared to the placebo group at both 32 weeks (25% vs. 45%) and six weeks postpartum (35% vs. 55%). This indicates that topical application of VCO during pregnancy is associated with a reduced risk of developing new stretch marks. The p-values (<0.01 and <0.001) demonstrate the statistical significance of these findings, suggesting that the observed differences are unlikely due to chance. The severity of SG, as assessed by the modified striae gravidarum assessment scale (SGAS), was also significantly lower in the VCO group compared to the placebo group at both 32 weeks (1.8 vs. 2.6) and six weeks postpartum (1.4 vs. 2.2). This suggests that VCO not only prevents the formation of new SG but also reduces the severity of existing marks. Again, the p-values (<0.001) indicate the statistical significance of these results. The beneficial effects of VCO on SG incidence and severity were observed both during pregnancy (32 weeks) and postpartum (six weeks), suggesting a sustained effect of the intervention. This is an important finding as it indicates that VCO may be beneficial for both preventing and treating SG. The magnitude of the observed differences in SG incidence and severity between the VCO and placebo groups is clinically relevant. A 20% reduction in the incidence of new SG and a reduction of 0.8 to 0.8 points on the SGAS scale are meaningful improvements that could have a positive impact on the well-being of pregnant women. Overall, Table 2 provides compelling evidence of the efficacy of topical VCO in preventing and reducing the severity of SG during and after pregnancy. These findings support the traditional use of VCO for skin health and suggest a potential role for VCO in antenatal care.
Table 2. Incidence and severity of SG in the VCO and placebo groups.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>VCO (n=100)</th>
<th>Placebo (n=100)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence of SG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>32 weeks</td>
<td>25%</td>
<td>45%</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>35%</td>
<td>55%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Severity of SG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>32 weeks</td>
<td>1.8 (0.9)</td>
<td>2.6 (1.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>1.4 (0.7)</td>
<td>2.2 (1.1)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 3 presents the secondary outcomes of the study, specifically the changes in skin hydration and elasticity in both the virgin coconut oil (VCO) group and the placebo group at three-time points: baseline, 32 weeks gestation, and six weeks postpartum. The primary observation from Table 3 is the absence of statistically significant differences (all p-values > 0.05) in both skin hydration (measured in arbitrary units, AU) and skin elasticity (measured as R parameter) between the VCO and placebo groups at any of the assessed time points. This means that the application of VCO did not significantly alter skin hydration or elasticity compared to the placebo. The mean values and standard deviations (SD) for both skin hydration and elasticity are very similar between the VCO and placebo groups at each time point. This further reinforces the lack of significant group differences and suggests that VCO does not have a differential effect on these skin properties compared to the placebo. The slight variations observed in skin hydration and elasticity values across time points and between groups are likely due to normal physiological fluctuations during pregnancy and individual differences in skin properties. These variations are expected and do not indicate a significant effect of the intervention.

Table 3. Skin hydration and elasticity in VCO and placebo groups.

<table>
<thead>
<tr>
<th>Time point</th>
<th>Group</th>
<th>Mean skin hydration (AU)</th>
<th>SD</th>
<th>Mean skin elasticity (R)</th>
<th>SD</th>
<th>p-value (Hydration)</th>
<th>p-value (Elasticity)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>VCO</td>
<td>50.3</td>
<td>10.2</td>
<td>0.80</td>
<td>0.10</td>
<td>0.85</td>
<td>0.92</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>49.8</td>
<td>9.8</td>
<td>0.79</td>
<td>0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32 weeks gestation</td>
<td>VCO</td>
<td>45.5</td>
<td>8.4</td>
<td>0.75</td>
<td>0.08</td>
<td>0.73</td>
<td>0.67</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>46.1</td>
<td>7.9</td>
<td>0.76</td>
<td>0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 weeks postpartum</td>
<td>VCO</td>
<td>48.7</td>
<td>9.1</td>
<td>0.78</td>
<td>0.09</td>
<td>0.61</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>48.2</td>
<td>8.7</td>
<td>0.77</td>
<td>0.08</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The most significant observation from Table 4 is the complete absence of reported adverse events in both the VCO and placebo groups across all categories. This indicates that neither the topical application of VCO nor the placebo resulted in any adverse reactions or side effects in the participants. The absence of adverse events in the VCO group strongly supports the safety profile of topical VCO for use during pregnancy. This is consistent with the existing literature, which generally reports VCO as a safe and well-tolerated topical agent. While the absence of adverse events is encouraging, it is important to acknowledge the limitations of this study. The sample size was relatively small (100 participants per group), and the follow-up period was limited to six weeks postpartum. Therefore, while this study provides initial evidence of the safety of VCO, larger-scale studies with longer follow-up periods are needed to definitively confirm its long-term safety during pregnancy.
Table 4. Adverse events reported during the study.

<table>
<thead>
<tr>
<th>Adverse event category</th>
<th>VCO Group (n=100)</th>
<th>Placebo Group (n=100)</th>
<th>Total (n=200)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin irritation</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Allergic reactions</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other skin issues</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Systemic reactions</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total adverse events</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The skin, the body’s largest organ, serves as a vital barrier against the external environment. It protects us from physical damage, microbial invasion, and harmful substances, while also regulating body temperature and maintaining hydration. However, this constant exposure to the environment also subjects the skin to a variety of stressors, including ultraviolet (UV) radiation, pollution, and the byproducts of normal metabolic processes. These stressors can disrupt the delicate balance within the skin, leading to a state known as oxidative stress. Oxidative stress occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the body’s ability to neutralize them with antioxidants. ROS are highly reactive molecules that contain oxygen and have an unpaired electron. This unpaired electron makes them unstable and eager to react with other molecules in the body, including lipids, proteins, and DNA. While ROS are naturally produced during normal cellular metabolism, excessive amounts can overwhelm the body’s antioxidant defenses, leading to cellular damage and dysfunction. The skin is particularly vulnerable to oxidative stress due to its constant exposure to environmental factors like UV radiation and pollution. UV radiation, especially UVB rays, can directly damage cellular components and generate ROS within the skin. Air pollutants, such as particulate matter and ozone, can also induce oxidative stress by triggering inflammatory responses and generating ROS. Additionally, normal metabolic processes within the skin cells generate ROS as byproducts, contributing to the overall oxidative burden.\(^7,8\)

During pregnancy, the physiological demands on the body increase significantly. The growing fetus requires additional nutrients and oxygen, leading to an increase in metabolic rate and energy expenditure. This heightened metabolic activity, in turn, results in the production of more ROS, contributing to oxidative stress. Furthermore, pregnancy is characterized by significant hormonal fluctuations, particularly in estrogen and progesterone levels. These hormones play crucial roles in regulating various physiological processes, but they can also influence oxidative stress. Estrogen, for instance, has been shown to increase the production of ROS in certain cell types. Progesterone, on the other hand, can suppress the activity of antioxidant enzymes, further tipping the balance towards oxidative stress. The combination of increased metabolic demands and hormonal fluctuations during pregnancy creates a unique environment that amplifies the risk of oxidative stress in the skin. This heightened oxidative stress can have detrimental effects on skin health and integrity, contributing to the development of various skin conditions, including striae gravidarum (SG).\(^9,10\)

Virgin coconut oil (VCO), derived from the fresh meat of mature coconuts, has emerged as a promising natural agent for combating oxidative stress in the skin. VCO is rich in bioactive compounds, including medium-chain fatty acids (MCFAs), polyphenols, and vitamin E. These constituents work synergistically to provide potent antioxidant protection and mitigate the damaging effects of ROS. MCFAs, particularly lauric acid, have been shown to exhibit antioxidant activity by scavenging free radicals and inhibiting lipid peroxidation, a process by which ROS damage cell membranes. Polyphenols, such as phenolic acids and flavonoids, are potent antioxidants that neutralize ROS and protect cellular components from oxidative stress.\(^7,8\)
damage. Vitamin E, a fat-soluble vitamin found in VCO, is a well-known antioxidant that protects cell membranes from lipid peroxidation and helps maintain skin integrity. The combined antioxidant activity of these compounds in VCO creates a protective shield against oxidative stress in the skin. By neutralizing ROS and preventing oxidative damage, VCO helps to maintain the integrity of collagen and elastin fibers, the essential building blocks of healthy skin. This protective effect is particularly important during pregnancy when the skin is subjected to increased oxidative stress due to metabolic demands and hormonal fluctuations.\textsuperscript{11,12}

Collagen and elastin are two key structural proteins that provide the skin with strength, elasticity, and resilience. Collagen fibers form a dense network that provides structural support, while elastin fibers confer elasticity, allowing the skin to stretch and recoil. The integrity of these fibers is crucial for maintaining skin health and preventing the formation of stretch marks. During pregnancy, the rapid expansion of the abdomen and other areas puts significant stress on the skin. This stretching, combined with the hormonal changes of pregnancy, can disrupt the balance of collagen and elastin production and degradation. The increased levels of cortisol, a stress hormone, can suppress collagen synthesis and promote its breakdown. Additionally, the elevated estrogen levels can inhibit the proliferation of fibroblasts, the cells responsible for producing collagen and elastin. The resulting decrease in collagen and elastin content weakens the skin’s connective tissue, making it more susceptible to tearing and the formation of striae gravidarum. The antioxidant properties of VCO can help to mitigate this process by protecting collagen and elastin fibers from oxidative damage. By neutralizing ROS and preventing the breakdown of these proteins, VCO helps to maintain the skin’s structural integrity and reduce the risk of stretch marks. Furthermore, VCO has been shown to stimulate collagen synthesis and promote wound healing, which are essential for repairing damaged skin tissue. The MCFAs in VCO have been found to increase the production of collagen by fibroblasts, leading to improved skin thickness and elasticity. VCO also enhances angiogenesis, the formation of new blood vessels, which is crucial for delivering nutrients and oxygen to the skin and promoting healing.\textsuperscript{13,14}

The emollient properties of VCO also contribute to maintaining skin integrity during pregnancy. The fatty acids in VCO form a protective barrier on the skin, preventing moisture loss and improving hydration. This helps to keep the skin supple and elastic, allowing it to stretch without tearing. VCO’s multifaceted actions, including its antioxidant, anti-inflammatory, wound-healing, and emollient properties, work synergistically to protect the skin from oxidative stress and maintain its integrity during pregnancy. By preserving the balance of collagen and elastin fibers and promoting skin repair, VCO can effectively prevent and reduce the severity of striae gravidarum, a common concern for pregnant women. Further research is needed to fully elucidate the molecular mechanisms underlying VCO’s beneficial effects, but the existing evidence strongly supports its potential as a safe and effective intervention for maintaining skin health during pregnancy.\textsuperscript{13,15}

Pregnancy, while a physiological process, is characterized by a complex interplay of hormonal and immunological changes that result in a state of mild systemic inflammation. This inflammatory response is essential for various physiological processes, including implantation, placental development, and fetal growth. However, an excessive or prolonged inflammatory state can have detrimental effects on maternal health and may contribute to the development of pregnancy complications, including striae gravidarum (SG). The systemic inflammatory response during pregnancy is primarily driven by the placenta, which releases various pro-inflammatory cytokines and chemokines into the maternal circulation. These molecules, including tumor necrosis factor-alpha (TNF-\(\alpha\)), interleukin-6 (IL-6), and C-reactive protein (CRP), play critical roles in immune regulation, tissue remodeling, and angiogenesis. However, their excessive production...
can lead to a chronic low-grade inflammatory state that has been linked to several pregnancy complications, such as preeclampsia, gestational diabetes, and preterm birth. The inflammatory response during pregnancy is not limited to systemic circulation but also affects various tissues, including the skin. The increased levels of pro-inflammatory cytokines can disrupt the delicate balance of collagen and elastin synthesis and degradation in the skin, leading to the weakening and fragmentation of connective tissue. This process, coupled with the mechanical stress of stretching, culminates in the formation of SG. Several studies have reported elevated levels of pro-inflammatory cytokines, such as TNF-α and IL-6, in the skin of pregnant women with SG. These cytokines have been shown to stimulate the production of matrix metalloproteinases (MMPs), enzymes that degrade collagen and elastin. Additionally, they can inhibit the activity of tissue inhibitors of metalloproteinases (TIMPs), which normally regulate MMP activity. This imbalance in MMP and TIMP activity can lead to the excessive breakdown of collagen and elastin, contributing to the development of SG. Virgin coconut oil (VCO) has emerged as a promising natural agent for mitigating inflammation and its associated complications, including SG. VCO is rich in medium-chain fatty acids (MCFAs), primarily lauric acid, which has been shown to possess potent anti-inflammatory properties. Lauric acid, the most abundant MCFA in VCO, exerts its anti-inflammatory effects through multiple mechanisms. It has been shown to inhibit the production of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6, by modulating the activity of nuclear factor-kappa B (NF-κB), a key transcription factor involved in the inflammatory response. Additionally, lauric acid can activate peroxisome proliferator-activated receptor gamma (PPARγ), a nuclear receptor that plays a crucial role in anti-inflammatory and immune regulatory processes. Furthermore, lauric acid has been shown to inhibit the activity of cyclooxygenase-2 (COX-2), an enzyme involved in the production of prostaglandins, which are potent mediators of inflammation. By suppressing COX-2 activity, lauric acid can reduce the synthesis of prostaglandins and mitigate the inflammatory response. In addition to lauric acid, VCO contains other MCFAs, such as capric acid and caprylic acid, which also exhibit anti-inflammatory properties. These MCFAs have been shown to inhibit the production of pro-inflammatory cytokines and chemokines, as well as the activity of enzymes involved in the inflammatory cascade. Moreover, they can modulate the balance of T helper (Th) cell subsets, promoting a shift from a pro-inflammatory Th1 response to an anti-inflammatory Th2 response. This shift in immune response can help to dampen the inflammatory process and protect against tissue damage.\textsuperscript{15,16}

The anti-inflammatory properties of VCO make it a promising agent for preventing and reducing the severity of SG. By inhibiting the production of pro-inflammatory cytokines and modulating the activity of inflammatory enzymes, VCO can help to protect the skin from damage caused by excessive inflammation. This can preserve the integrity of collagen and elastin fibers, maintain skin elasticity, and reduce the risk of SG formation. Furthermore, VCO’s wound-healing properties can contribute to the reduction of SG severity. By stimulating collagen synthesis, enhancing angiogenesis, and modulating the inflammatory response, VCO can promote the healing and remodeling of damaged skin tissue, leading to a reduction in the appearance of stretch marks. Preclinical studies have demonstrated the anti-inflammatory effects of VCO in various animal models of inflammation. For example, a study by Intahphuak et al. (2010) showed that VCO reduced paw edema and inflammatory cell infiltration in rats with carrageenan-induced paw edema, a model of acute inflammation. Another study by Yeap et al. (2015) found that VCO attenuated inflammation and oxidative stress in the liver of rats with high-fat diet-induced nonalcoholic steatohepatitis, a model of chronic inflammation. Clinical studies have also provided evidence for the anti-inflammatory effects of VCO in humans. A study by Assunção et al. (2018) found that VCO
supplementation reduced serum levels of CRP and IL-6 in healthy adults. Another study by Liau et al. (2016) reported that VCO improved skin barrier function and reduced inflammation in patients with atopic dermatitis. While these studies did not specifically focus on SG, they support the potential of VCO as an anti-inflammatory agent.\textsuperscript{16,17}

Striae gravidarum (SG) can indeed be viewed as a form of micro-trauma to the skin’s connective tissue. The rapid stretching of the skin during pregnancy, coupled with hormonal changes, disrupts the delicate balance of collagen and elastin fibers, leading to the characteristic appearance of stretch marks. This disruption can be likened to a wound-healing process, albeit one that often results in imperfect repair due to the unique physiological environment of pregnancy. Virgin coconut oil (VCO), with its diverse array of bioactive components, has demonstrated remarkable potential in promoting wound healing through various mechanisms. These properties may explain its observed efficacy in reducing the severity of SG in the present study. Collagen is the primary structural protein of the skin, providing strength and resilience. During the formation of SG, the normal collagen architecture is disrupted, leading to a loss of tensile strength and the formation of visible scars. This major component of VCO has been found to increase the expression of collagen genes and promote the proliferation of fibroblasts, the cells responsible for collagen production. This potent antioxidant protects fibroblasts from oxidative damage, ensuring their optimal function in collagen synthesis. These compounds, found in smaller amounts in VCO, have also been shown to stimulate collagen production and improve the organization of collagen fibers. By promoting collagen synthesis, VCO may help to repair the damaged connective tissue in SG, leading to a reduction in the depth and width of the stretch marks.\textsuperscript{17,18}

Angiogenesis, the formation of new blood vessels, is a crucial step in wound healing. It provides the necessary oxygen and nutrients to the damaged tissue, facilitating repair and regeneration. VCO has been shown to enhance angiogenesis by promoting the expression of vascular endothelial growth factor (VEGF), a key signaling molecule involved in blood vessel formation. In the context of SG, enhanced angiogenesis could improve blood flow to the affected area, delivering essential nutrients and growth factors that support the repair of damaged collagen and elastin fibers. This may contribute to the reduction of SG severity observed in the VCO group. Inflammation is a natural part of the wound-healing process. However, excessive or prolonged inflammation can hinder tissue repair and lead to scarring. VCO has been shown to modulate the inflammatory response by inhibiting the production of pro-inflammatory cytokines and chemokines. This anti-inflammatory effect may help to create a more favorable environment for wound healing, leading to a reduction in the intensity of SG. The lauric acid in VCO is a potent antimicrobial agent, which may also contribute to its anti-inflammatory properties. By preventing infection, VCO may further promote wound healing and reduce the risk of complications associated with SG. The fatty acids in VCO, particularly the medium-chain fatty acids (MCFAs), have excellent emollient properties. They form a protective barrier on the skin’s surface, preventing moisture loss and improving hydration. This is particularly important during pregnancy when the skin is under increased stress due to stretching. Maintaining adequate skin hydration is essential for preserving skin elasticity and preventing the formation of SG. By improving skin hydration, VCO may enhance the skin’s ability to withstand stretching, thereby reducing the risk of developing new stretch marks. In addition to the mechanisms discussed above, VCO may also influence other factors involved in the development and severity of SG. For example, VCO has been shown to modulate the expression of matrix metalloproteinases (MMPs), enzymes that break down collagen and elastin. By inhibiting MMP activity, VCO may further protect the skin’s connective tissue and reduce the risk of SG. VCO also contains antioxidants that may protect against oxidative damage to the skin. Oxidative stress is known to contribute to the aging
process and may play a role in the development of SG. By scavenging free radicals, VCO may help to preserve the integrity of the skin's extracellular matrix and reduce the likelihood of stretch mark formation.\(^{19,20}\)

4. Conclusion

This randomized controlled trial provides robust evidence that topical virgin coconut oil (VCO) is a safe and effective intervention for the prevention and reduction of striae gravidarum (SG) severity in pregnant women. Our findings demonstrate that VCO significantly reduces both the incidence of new SG and the severity of existing SG, with no reported adverse events.

5. References

15. Natarajan V, Venugopal M, Ramachandran A. Effect of virgin coconut oil on wound healing


