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Localization of E-cadherin and Vimentin in Mucosal Ulcer Healing Treated by Vitis Vinifera Seeds Oil in Rat Model

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Abstract

Objective: The anti-inflammatory components generated by Vitis vinifera (Grape) seeds oil (GSO) help reduce inflammation and accelerate the healing process when applied topically to an ulcer. The current study aimed to assess the expression of E-cadherin and Vimentin during oral ulcer healing treated by GSO.

Materials and Methods: Twenty-four albino male rats (350–450 g) were used for this study. The rats were slaughtered on the 5th and 10th days after an ulcer induction on either their left or right cheek mucosa. The rats in the control group received sterile distilled water, whereas those in the experimental group received GSO for ulcers treatment. The tissue samples were subsequently utilized for histological and immunohistochemical investigations of E-cadherin and Vimentin.

Results: Compared with the control group, the GSO-treated ulcer group presented significantly higher expression of E-cadherin and Vimentin at days 5 and 10 than the control group, with some evidence of epithelial-mesenchymal transition (EMT) at 5 days in both groups.

Conclusion: With the limitation of this study, GS may accelerate the healing of mucosal ulcers, as demonstrated by increased expression of E-cadherin and Vimentin.

Clinical relevance: GSO can be used as a topical herbal modality for mucosal ulcer healing.

Keywords: Vitis vinifera seed oil (GSO), Mucosal ulcer, Vimentin, E-cadherin, Rats, Healing, EMT.

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Introduction

The development of the extracellular matrix (ECM), migration, adhesion, proliferation, and differentiation are all steps in the multicellular process that constitutes wound healing [1]. The complex connections among many cell types, cytokines, and the circulatory system are involved in the wound-healing process. The initial cascade of vasoconstriction and platelet aggregation stops bleeding, and an influx of inflammatory cells, led by neutrophils in the initial stages, occurs. To encourage angiogenesis and re-epithelialization, fibroblasts then construct a framework by laying down extracellular components [2].

Phenolic compounds are plant secondary metabolites found in fruits, vegetables and beverages, including grapes, The beneficial health effects of grape and grape products and the anti-inflammatory and protective effects of traditional grape preparations on the cardiovascular system are related to their bioactive phenolic compounds. The biological activities of grape phenolic compounds include among others anti-inflammatory, antioxidant and cardioprotective effects [3].

Proanthocyanidin in grape seeds can target human keratinocytes and cause them to produce the vascular endothelial growth factor VEFG [4]. Owing to the high concentrations of vitamins, fatty acids, and phenolic compounds, grape seed oil (GSO) is beneficial to the food, pharmaceutical, and esthetic sectors. Grape seed oil may interact with cellular and molecular pathways; it has anti-inflammatory, antibacterial, and anticancer properties [5]. It is

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composed of polyunsaturated fatty acids (PUFAs), with linoleic acid (C18:2) accounting for 58–78% of the total [6]. This may inhibit oxidative stress by preventing the release of proinflammatory mediators by oxidized low-density lipoprotein-stimulated macrophages [5]. The adhesion of cells in human epithelial tissues relies on adherent junctions, which are enhanced by the cell adhesion protein E-cadherin (E-cad) [7]. E-cadherin is crucial in the wound healing process, including mitosis, maturation, and migration. In vitro research revealed that the upregulation of keratinocyte migration leads to the downregulation of E-cadherin expression [8].

Vimentin is an intermediate filament protein that contributes to cellular processes such as migration, morphology, plasticity, and organelle anchoring. In addition to its structural and cytoskeletal functions. Lymphocytes, fibroblasts, endothelial cells, macrophages, and melanocytes are mesenchymal cells that express vimentin [9]. Vimentin located on the cell surface has several functions in cell–cell interactions, wound healing, migration, and immune system activation [10].

The epithelial–mesenchymal transition (EMT) is caused by metabolic alterations that enable polarized epithelial cells to adopt a phenotype like that of mesenchymal cells. During this process, the cell's basal surface interacts with the basement membrane [11]. Typically, studies related to EMT have investigated the expression of epithelial markers (e.g., E-cadherin) and mesenchymal markers (e.g., N-cadherin or vimentin) to characterize the process; however, conclusive experimental evidence for a real mesenchymal state as the endpoint is absent in several cases where the term EMT is employed [12]. The benefits of grape seed oil on the healing of oral ulcers have not been widely researched. Therefore, the purpose of this study is to investigate the curative process of an experimentally induced oral ulcer treated with grape seed oil in a rat by assessing the expression of E-cadherin and Vimentin monoclonal antibodies.

Material and Methods

The current study was approved by the Institutional Ethical Committee of Animal Experimentation of the (Blinded for review). The animals were kept under nursing and supervision from the staff of the animal house of the Medical Research Institute, City, for approximately 3 months, starting in November 2023 and ending in February 2024. In this study, the authors followed the Animal Research: Reporting in Vivo Experiments (ARRIVE) guidelines [13]. Grape seed extract has been approved as generally recognized as safe (GRAS) by the Food and Drug Administration (FDA) and is sold commercially as a dietary supplement listed in the Everything Added to Food in the United States (EAFUS) database. Bentivegna and Whitney reported that the no-observed- adverse-effect level (NOAEL) of grape seed extracts determined in rats was 1.78 g/kg body weight/day, and the normal amount of grape seed extract used in food applications was 0.01–1% [14].

The rats were housed in polypropylene enclosures within the experimental animal facility, maintaining consistent environmental conditions such as 12-hour light–dark cycles and a temperature of 24 ± 2 °C. The animals were given a rat chow pellet meal and were allowed free access to tap water. Before ulcer induction, the rats were allowed a one-week acclimation period to adjust to the laboratory environment.

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A total of twenty-four male albino rats, weighing between 250 and 300 grams and aged two to three months, were used as experimental models. Twelve rats were allocated for each study duration (five and ten days). The rats were then randomly divided into two groups, a control group and a group treated with GSO. Female rats were specifically excluded because of cyclic hormonal variations and the potential for pregnancy throughout the experimental study.

To determine the precise dosage of general anesthesia, the rats were weighed. Then, general anesthesia was induced via intraperitoneal injection of xylazine (5 mg/kg) and ketamine (50 mg/kg) [15]. After disinfection with 70% alcohol, ulcers were induced on both the right and left buccal mucosa via sterile round diamond burs, creating lesions 5 mm in diameter and 1 mm deep (Figure 1), which were measured via a digital Vernier caliper [16].

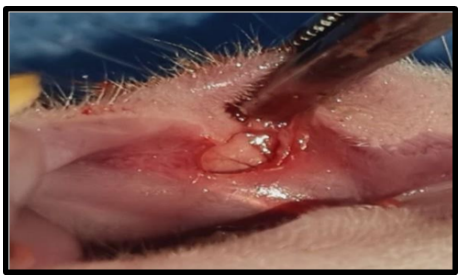

Figure 1. A photograph showing traumatic ulcer induction in the buccal mucosa.

The treatment involved daily application of 30 µl of GSO (US ORGANIC, Amazon)" USDA Certified Organic & 100% Pure: Cold-pressed for maximum purity and nutrient retention, sourced from premium quality grapes, antioxidant-Rich: Rich in essential fatty acids like Oleic and Linoleic" (Figure 2) on the right side via a micropipette, whereas the left side received distilled water as a control.

Figure 2. Vitis vinifera seed oil (GSO).

The animals were euthanized on days 5 and 10 through the administration of an excessive amount of anesthetic solution [17]. The ulcerated regions were excised and extended 5 mm beyond the ulcer edge. After fixation in 10% neutral buffered formalin for 24 hours, the samples were washed [18]. The samples were dehydrated by placing them in increasing alcohol concentrations, then clarified in xylene, and finally embedded in paraffin wax. Sections (4 µm) were then subjected to hematoxylin and eosin (H&E) staining (Figure 3) and immunohistochemical analysis, which was performed under a light microscope, to evaluate mucosal ulcer healing.

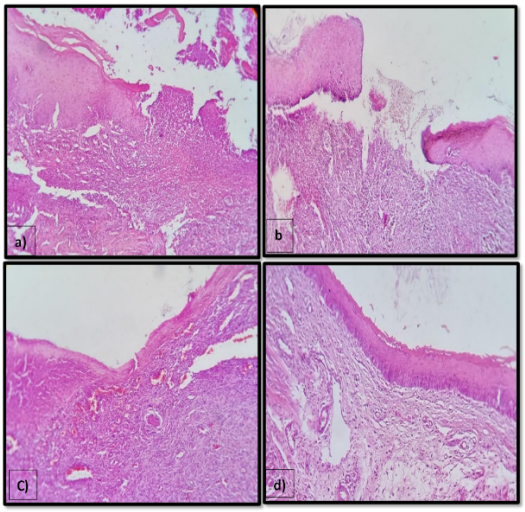

Figure 3. Histological progression of oral ulcers at two time points (day 5 and day 10) under 10X magnification. A) Control group on Day 5, B) GSO-treated group on Day 5, C) control group on Day 10, D) GSO-treated group on Day 10.

Immunohistochemical analysis of E-cadherin and vimentin was carried out via monoclonal mouse anti-human E-cadherin, clone NCH-38 (Dako Company), England, and monoclonal mouse anti-vimentin, clone Vim 3B4 (Daco Company), England.

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Under a light microscope at x40, five fields were chosen from the epithelium area, and another five fields from the lamina propria from each tissue section were captured by a digital camera (5 pixels), and the images evaluated were imported to a computer.

The evaluation of the staining results was achieved by applying the Aperio positive pixel count algorithms program (from Aperio Image Scope software v11.1.2.760 developed by Aperio Technologies Inc., USA), and we neglected the yellow color, which represents a weak positive reading [1,19,20]. The average mean positive percentages for each of the five areas from the epithelium and lamina propria were obtained and considered the value of the expression of both E-cadherin and vimentin.

The data obtained from the immunohistochemical examination were assessed for normality via the Shapiro–Wilk test. Numeric values are presented as a range encompassing the minimum and maximum values, along with the mean, standard deviation, and median. When the variables exhibited a normal distribution, Student's t test was used to compare two distinct groups. The predetermined level of significance for the findings was established at P=0.05. The data were analyzed via the IBM SPSS software package version 20.0.

Results

The control group on days 5 and 10 presented mild to moderate membranous morphology with positive expression of E-cadherin in the newly formed basal, spinosum, and granulosum cells of the epithelium and negative expression in the keratinized layer. On the other hand, the endothelial cells and fibroblasts weakly positively expressed E-cadherin (Figure 4, A and B). The study group at both time points presented moderate to strong positive expression in all layers of the newly regenerated epithelium around the ulcer margin except the keratinized layer (Figure 3, C and D). In lamina propria, endothelial and fibroblasts presented moderate positive expression of E-cadherin, especially on day 5, in the control group (Figure 5). The expression levels of E-cadherin in the examine tissue samples are summarized in (Table 1).

The control group at two time points (day 5 and day 10) after ulcer induction presented moderate cytoplasmic expression of vimentin in fibroblasts, endothelial cells, and inflammatory cells (Figure 6, A and B). However, the study group exhibited strong cytoplasmic expression of vimentin in fibroblasts, endothelial cells, and inflammatory cells at both time points (Figure 6, C and D). On the other hand, moderate positive expression was detected in the cytoplasm of the granulosum epithelial cell layer, especially in both groups, on day 5 (Figure 7). The distribution and intensity of vimentin expression in the studied samples are presented in (Table 2).

A comparative analysis of E-cadherin and vimentin expression patterns is provided in (Table 3) demonstrate an inverse relationship between these markers.

The correlation analysis between E-cadherin and vimentin expression levels is summarized in (Table 4).

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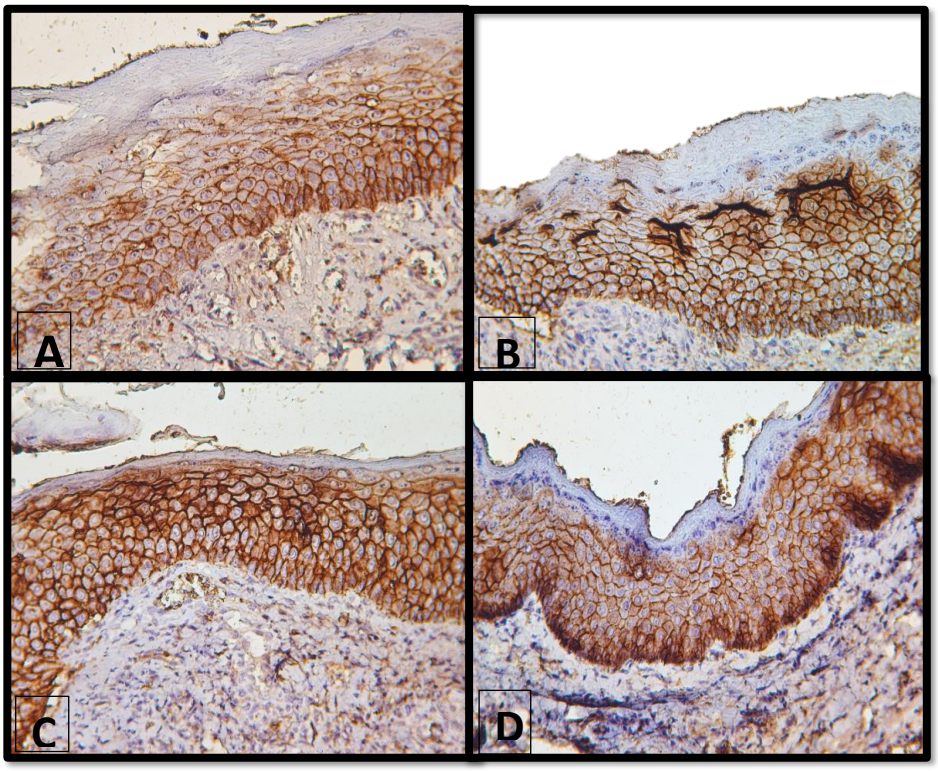

Figure 4. A: Photomicrograph showing membranous positive expression of E-cadherin in the epithelial layer, DAB stain (40x), A) Control Day 5 B) Control day 10, C) Study day 5, D) Study day 10.

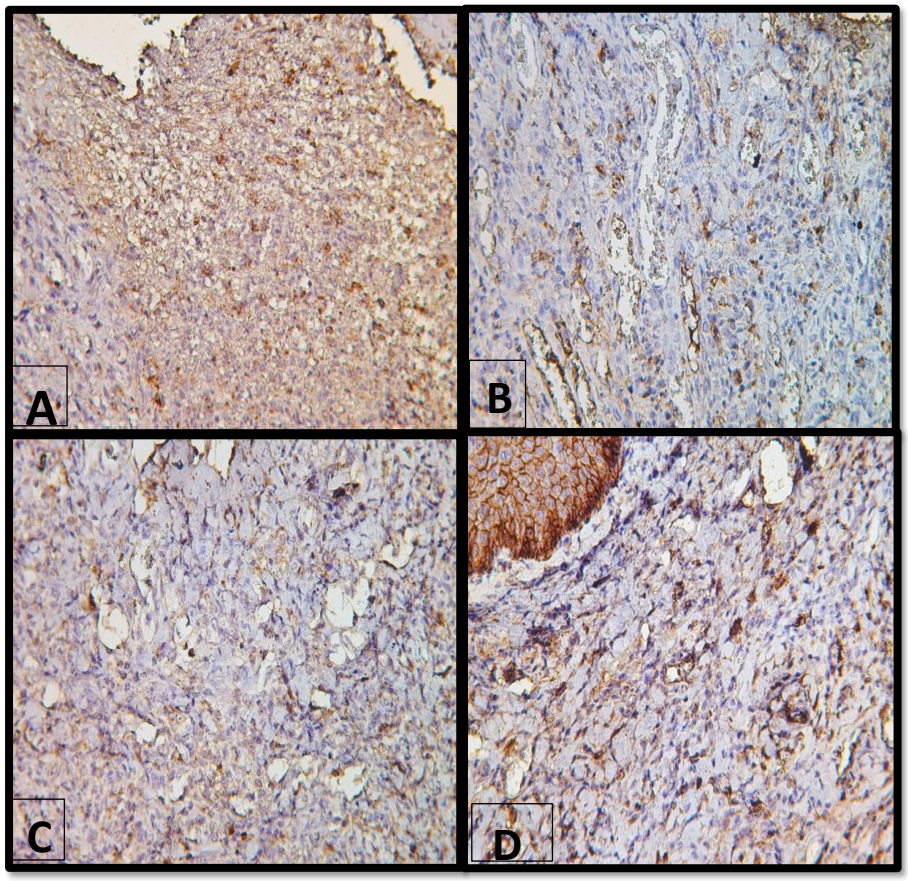

Figure 5. Photomicrograph of the lamina propria of the rat oral mucosa showing moderate positive expression of E-cadherin in A and weak positive expression in B, C, and D. DAB staining at 40x.

A) Control day 5. B) Control day 10. C) Study day 5. D) Study day 10.

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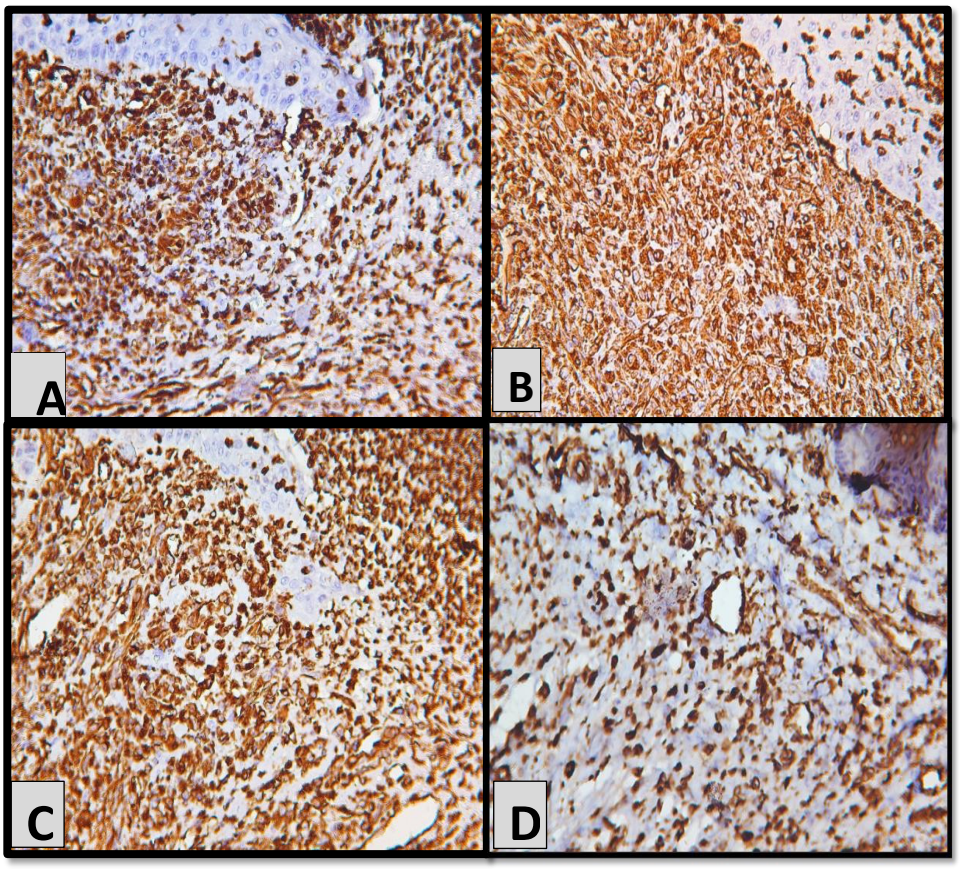

Figure 6. Immunohistochemical image showing strong positive cytoplasmic expression of vimentin in inflammatory cells, fibroblasts, and blood vessels in the lamina propria; vimentin DAB stain (40x). A) Control day 5, B) control day 10, C) study day 5, D) study day 10.

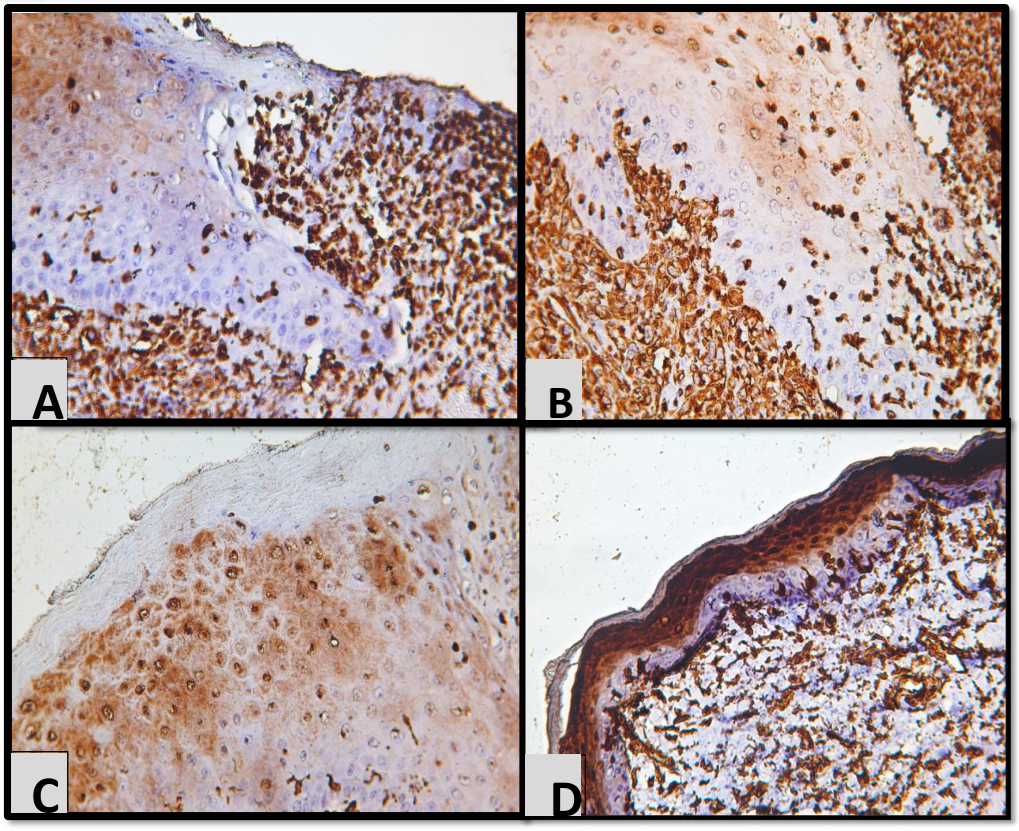

Figure 7. Photomicrograph showing weakly positive expression of Vimentin in the granulosa layer of the epithelium in A, B, and C and strongly positive expression in D. DAB staining (40x): A) Control Day 5 B) Control Day 10 C) Study Day 5 D) Study day 10.

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Table 1. Comparison of E-cadherin expression in the epithelium and lamina propria between the studied groups.

day	site	control		study		T test	P value
		Mean	SD	Mean	SD		
5th	Epithelium	56.95	3.26	71.31	3.57	-10.291	0.000*[2]
	Lamina properia	26.71	4.73	19.10	4.53	3.292	0.000*[1]
10th	Epithelium	60.74	2.28	80.37	1.79	-23.434	0.000*[2]
	Lamina properia	12.36	1.66	13.63	2.22	-1.585	0.127*[2]

Table 2. Comparison of the expression of vimentin in the epithelium and lamina properties between the studied groups.

day	site	control		study		T test	P-value
		Mean	SD	Mean	SD		
5th	Epithelium	27.29	3.5	49.05	4.32	-13.56	0.000*[1]
	Lamina properia	41.09	2.93	71.09	3.17	-24.059	0.000*[1]
10th	Epithelium	36.70	1.83	56.51	3.55	-4.158	0.000*[2]
	Lamina properia	54.93	3.27	82.77	1.67	-26.233	0.000*[2]

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Table 3. Comparison of E-cadherin and vimentin markers.

Day	Group	Layer	Mean differnce±SE	T test	P value
Day 5	Control	Epithelium	29.66±1.38	21.495	0.000*[1]
		Lamina properia	-14.38±1.61	-3.927	0.000*[2]
	Study	Epithelium	22.27±1.62	13.768	0.000*[1]
		Lamina properia	-51.99±1.60	-32.569	0.000*[1]
Day 10	Control	Epithelium	24.04±0.85	28.446	0.000*[1]
		Lamina properia	-42.57±1.06	-40.187	0.000*[1]
	Study	Epithelium	23.86±1.15	4.158	0.000*[2]
		Lamina properia	-69.15±0.8	-86.141	0.000*[1]

Table 4. Correlations between E-cadherin and vimentin marker.

Day	Group	Layer	r	P value
Day 5	Control	Epithelium	0.035	0.915[1]
		Lamina properia	-0.165	0.609[2]
	Study	Epithelium	0.148	0.646[1]
		Lamina properia	0.031	0.925[1]
Day 10	Control	Epithelium	0.028	0.932[1]
		Lamina properia	0.241	0.451[1]
	Study	Epithelium	0.329	0.296[2]
		Lamina properia	0.417	0.177[1]

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Discussion

The current study chose laboratory Albino rats because of their physiological similarity to humans, making them a practical model for evaluating the dynamics of the mucosal healing process [21]. Traumatic ulcers were induced with a diamond bur 5 mm in diameter, which reflects the common clinical similarity with mucosal injury [22]. The development of mucosal inflammation has frequently been suggested to be influenced by a loss of epithelial barrier function [23]. Currently, herbal medicine represents a significant aspect of complementary and alternative medical practices. The therapeutic use of herbs in wound and injury management has been well-established since the dawn of ancient civilizations [24].

Previous studies have shown that the therapeutic efficacy of the GSO accelerates mucosal healing by enhancing wound closure, reducing inflammation, and accelerating the restoration of epithelial integrity [25]. This finding is consistent with our finding that ulcers treated topically with GSO accelerated wound healing better than that in the control one.

Moreover, GSO significantly increased the expression of E-cadherin and vimentin compared with the control group. This may be because of the phenolic compounds, fatty acids, and vitamins present in the oil, which act as anti-inflammatory and antioxidant agents [26].

The present study revealed strong membranous expression of E-cadherin in almost all the epithelial layers except the keratinized layer.

This finding coincided with previously observed moderate to strong (2+/3+) membranous E-cadherin staining observed in the epithelial cells of the oral cavity [27]. In a previous study [28], E-cadherin was highly expressed in normal mucosal tissue. Intense staining of E- cadherin was observed in the spinous and basal cell layers, this finding correlates with our results.

Vimentin is a type III intermediate filament protein exclusively expressed in mesenchymal cells. Because Vimentin is naturally observed in the lamina propria, Vimentin facilitates the integration, response, and perception of microenvironmental stimuli by cells [29]. Greater expression was observed in the GSO group than in the control group, which was confirmed previously [30]. The immortalized keratinocyte cell line exhibited significantly elevated levels of vimentin when cocultured with both normal and keloid-derived fibroblasts in the extracellular matrix (ECM).

However, several investigations have recorded the temporary stimulation of vimentin expression in epithelial cells in response to certain pathological or physiological situations that require epithelial cell motility. Consequently, vimentin has been used as an indicator of the migratory state of epithelial cells [31]. Our results demonstrate that vimentin is expressed in the granulosum layer of the epithelium, which matches previous findings [30], with epidermal cells not differentiating into fibroblasts but having a fibroblast-like phenotype. Additionally, abnormal vimentin expression in the oral epithelium of premalignant lesions and weak vimentin immunoreactivity in the cytoplasm of suprabasal epithelial cells was previously reported [32]. The production of vimentin in epithelial cells may induce early molecular alterations in both the epithelial and mesenchymal compartments.

The expression of vimentin in the epithelial layer may be linked to

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the appearance of cells exhibiting a mesenchymal phenotype inside an epithelial sheet, which is generally referred to as epithelial-to-mesenchymal transition (EMT) which results from the transformation of endogenous epithelial cells [33,34].

The epithelial-mesenchymal transition (EMT) is a crucial process requiring two key proteins, E- cadherin and vimentin, known for their varied expression and unique roles in this transition. The cytoplasm was positively stained with vimentin, whereas the cell membrane was positively stained with E-cadherin. During EMT, cellular phenotypes change quickly and may be reversed. In contrast, epithelial cells change their polarity and cytoskeleton by losing adhesion junctions. E-cadherin is recognized for maintaining the integrity of epithelial cells and plays a crucial role in cell differentiation. Vimentin is a cytoskeletal protein present in endothelial cells, fibroblasts, and leukocytes, among other cell types. It is absent in normal epithelial cells and contributes to EMT [35].

In the present study, after re-epithelization, complete ulcer healing, and strong expression of Vimentin in the epithelial layers on day 10 in the GSO group may be due to mesenchymal-to- epithelial transition (MET) may occur to help in wound healing and restores epithelial layers. As an organism's body plan develops, the MET plays a role in several stages of morphogenesis and organogenesis. It occurs in intermittent alternation with the EMT. MET controls epithelial creation throughout embryonic development, whereas mesenchymal cells gradually achieve apicobasal polarity [36].

Conclusion

The topical application of GSO effectively treated experimentally induced buccal mucosal ulcers in rats by promoting their proliferation, as shown by alterations in the expression of E-cadherin and vimentin. Continuous treatment for 10 days increased the expression of E-cadherin and vimentin, with significant differences observed between the control and study groups. In the current study, the expression of the epithelial marker E-cadherin was detected strongly in epithelial cells with weak intensity in the lamina properia. In contrast, the mesenchymal marker vimentin was identified in lamina properia with weak intensity in epithelial cells. EMT and MET may explain this; however, this finding requires validation via several future investigations.

Conflicts of interest

The authors declare no competing interest.

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