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Fomes officinalis Extract Inhibits Sebum Secretion by Up-regulating MicroRNA-29

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1. Introduction

Oily-sensitive skin (OSS) is a dermatological condition combining characteristics of oily and sensitive skin. It presents a dual burden of sebum overproduction and cutaneous hyper-sensitivity, leading to compounded physiological and psychosocial impacts [1, 2]. Oily skin (OS) is a facial condition defined by enlarged pores and a shiny appearance, driven by excessive sebum production that causes the skin to feel greasy and heavy [3, 4]. Conversely, sensitive skin (SS), marked by neurosensory hyperactivity and impaired epidermal barrier function [5]. This condition manifests as unpleasant sensations such as stinging, burning, pain, and itching. Likewise, individuals with SS frequently report experiencing negative emotional impacts [6]. Intrinsic and environmental factors, such as UV irradiation, skincare habits, diet, sleep patterns, and psychological stress, can trigger or ag-gravate OSS [2]. Managing OSS is particularly challenging because conventional sebostatic agents, such as retinoids and salicylic acid, effectively reduce sebum but often disrupt epi-dermal homeostasis in SS [1]. This disruption exacerbates sensitivity by causing excessive desquamation, pH imbalance, and ceramide depletion.

The SCAP/SREBP-1 pathway functions as a central regulator of lipid biosynthesis, promoting sebum production by transcriptionally activating lipogenic enzymes, including fatty acid synthase (FASN) and acetyl-CoA carboxylase (ACC) [7, 8], with miR-29 identified as a key negative regulator of this pathway. The miR-29 family comprises three mature members: miR-29a, miR-29b, and miR-29c, encoded by two gene clusters [9, 10]. Subsequent studies have identified the role of miR-29 in the negative feedback regulation of lipid metabolism and immune inflammation in diabetes [11, 12]. However, its effects on sebocyte biology and its impact on oily and sensitive skin remain uncharacterized. Importantly, while natural compounds like berberine have demonstrated inhibitory effects on the SCAP/SREBP-1 pathway in colon cancer cells [13], no studies to date have explored the role of natural bioactives in miR-29-mediated regulation of this pathway in human sebocytes. With its dual activity in suppressing lipogenesis and inflammation [7, 11], miR-29 presents itself as a promising therapeutic target for managing OSS, which exhibits both excessive sebum production and impaired barrier function.

Fomes officinalis (FO) is a rare polypore fungus extensively employed in traditional Uighur medicine in China and is also recognized as one of the most widely used medicinal mushrooms in traditional European medicine owing to its notable therapeutic potential [14]. Recent studies have substantiated its diverse bioactivities. Traditional applications and chemical analyses have demonstrated that FO is rich in bioactive compounds, such as triterpenes, polysaccharides, and phenolics, which possess anti-inflammatory, antibacterial, and antioxidant properties [15, 16]. The synergistic effects of triterpenoids (which regulate lipids) and polysaccharides (which repair the skin barrier) also establish Fomes officinalis as a promising candidate for the treatment of oily and sensitive skin.

Therefore, this study hypothesized that FOEs may mitigate stress-induced excessive sebum secretion by epigenetically targeting miR-29 to inhibit SCAP/SREBP-1-mediated lipogenesis. Concurrently, the effects of FOEs on the skin barrier were explored. This work establishes a paradigm for the development of skincare ingredients that regulate miRNAs. These ingredients may simultaneously maintain lipid homeostasis and skin barrier stability.

2. Materials and Methods

2.1 Experimental materials

Human immortalized keratinocyte (HaCaT) and Human immortalized sebaceous gland cells (SZ95) were purchased from the Guangzhou Customs Technology Center (Guangzhou, China). Streptomycin-penicillin, fetal bovine serum (FBS) and DMEM were purchased from Gibco (NY, USA). Sebomed® Basal Medium, hydrocortisone (HC), retinoic acid (RA), Nile red was purchased from Sigma Aldrich (Shanghai, China). The recombinant human epidermal growth factor and TRIzol reagent were purchased from Invitrogen (Carlsbad, USA). CCK-8 kits was purchased from Neobioscience (Shenzhen, China). The cDNA synthesis kit, miRNA cDNA synthesis kit, and a reverse transcription kit were purchased from Takara Bio (Beijing, China).

2.2 Cell culture

B16F10 and HaCaT cells were cultured in DDMEM, with 10% fetal bovine serum, and 1% mycilli. Cells were maintained in a 37°C incubator containing 5% carbon dioxide.

2.3 Cell viability assay

The viability of B16F10 cells and HaCaT cells were measured using a CCK-8 solution assay. The cell suspensions were inoculated into 96-well plates at 0.8-1.0×10^5 cells/well and incubated in a cell culture incubator for 24 h. After removing the culture medium, the cells were treated with FOEs at set concentration for 24 h. Then, 10μL of CCK-8 solution was added to each well, followed by incubation at 37°C for 1–2h. Absorbance was detected at 450nm with a Multiscan spectrum microplate reader.

2.4 Neutral lipid assay

Nile red fluorescent stain was employed to label neutral lipids for the quantitative assessment of lipid content in SZ95 sebocytes. Specifically, hydrocortisone (HC) at a 10 ng/mL cocentration was added to induce neutral lipid production. After treatment with or without FOEs for 48 h, and 0.3 ng/mL retinoic acid (RA) was used as a positive control. Subsequently, 0.1%

Nile red dye was added, followed by a 5-min incubation. The cell fluorescence was detected using a multifunctional microplate reader at 495 nm to analyze lipid content in SZ95 sebocytes.

2.5 Quantitative Real Time RT-PCR

Total RNA was extracted from each group of SZ95 sebocytes using the TRIzol method [17]. cDNA was synthesized by reverse transcription using a TaKaRa reverse transcription kit (the total system was 20 μL). miRNAs were reverse transcribed to cDNA using a miRNA cDNA synthesis kit.Real-time RT-PCR was carried out on a Real-Time PCR instrument (Bio-Rad, USA). The primers were shown in Table 1. Three replcicates were made for each sample, and the relative expression of each gene was calculated using the $2^{-\Delta\Delta CT}$ method.

Table 1. Primer sequences for real-time quantitative PCR

Gene	Primer Sequences
miR-29b F	ACACTCCAGCTGGGTAGCACCATTTGAAATCAG
miR-29b R	CTCAACTGGTGTCGTGGA
miR-29b RT	CTCAACTGGTGTCGTGGAGTCGGCAATTCAGTTGAGAACACTGAT
U6-F	CTCGCTTCGGCAGCACA
U6-R	AACGCTTCACGAATTTGCGT
U6-RT	AACGCTTCACGAATTTGCGT
SCAP-F	GCCTGTGGGATGTACTGACT
SCAP-R	GGTCCTGCTGAATGGAGTAG
ACC-F	GCGGAGTGGCTAGAGAAACA
ACC-R	TCCATGGCAACCTCTGGATT
18s-F	GGCCTCCAAGGAGTAAGAAA
18s-R	GCCCCTCCTGTTATTATGG

2.6 Cell migration assay

Cell migration experiments were carried out using scratch experiments. Specifically, HaCaT cells were seeded on 6-well plates, cultured overnight, and then treated with different concentrations of FOEs for 24 h and 48 h. After that, scratch the cell layer with a sterile plastic pipette tip to form a scratch. The detached cells were rinsed off with PBS. After that, the DMEM medium containing 1% fetal bovine serum was added, the cells were cultured for 24 h, photos were taken, and the cell migration rate was calculated accordingly.

2.7 Clinical tral

A clinical and instrumental evaluation was conducted on 33 healthy Chinese subjects, 25 females and 8 males. Subjects in the study met the following inclusion criteria: sebum content of at least 120 μg/cm² on the forehead, transepidermal water loss (TEWL) ⩾ 20 g/m²h and a positive response to lactic acid-induced stinging. the study was conducted in Guangzhou, China, in September 2024. All experiments were carried out in accordance with the Declaration of Helsinki. All subjects were kept informed of all risks associated with the experiments before signing the informed consent form, and all participated voluntarily in this study. The subjects have agreed to publish the pictures involved in the paper. Thirty-three suubjects applied a 5% FOEs cream to their faces twice daily (morning and evening) for 2 weeks. Measurements were conducted at Weeks 0 and 2 to assess sebum content using the Sebumeter SM815, transepidermal water loss (TEWL) with the TewaMeter® TM Hex, skin redness (a* value) with the Colorimeter CL400. A lactic acid sting test was conducted on the participants' nasolabial folds on day 0 and after 2 weeks of cream application. Stinging scores were recorded to evaluate the effect of the 5% FOEs cream in reducing facial sensitivity to lactic acid.

2.8 Statistical analysis

GraphPad Prism 8 software was used to perform statistical analysis. Comparison between groups was made using ANOVA, and data were expressed as the means ±standard deviation. Statistically, differences were considered significant at $p<0.05$.

3. Results

3.1 T FOEs inhibited lipogenesis in SZ95 sebocytes

The MTT assay was performed to evaluate the effects of varying FOEs concentrations on the proliferation of SZ95 sebocytes. Results indicated that at FOEs concentrations below 0.12%, the relative cell viability of SZ95 sebocytes remained above 97% (Figure 2a). Compared to cells treated with HC alone, FOEs at concentrations ranging from 0.04% to 0.12% showed a dose-dependent inhibition of intracellular lipid accumulation (Figure 1b). The results suggested that FOEs could mitigate the HC-induced elevation in lipid levels in SZ95 sebocytes.

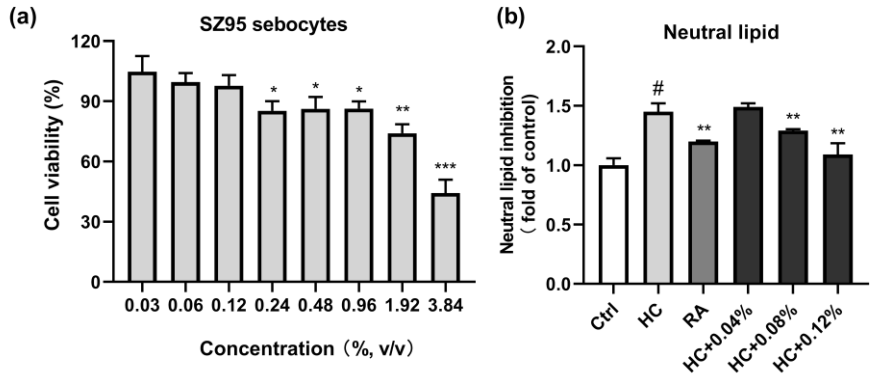

Figure 2. The effect of FOEs on neutral lipid secretion in HC-induced SZ95 sebocytes. (a) The cytotoxicity of FOEs in SZ95 sebocytes was determined by MTT assays. (b) Quantitative assessment of lipid synthesis after FOEs treatment (Nile Red staining).

3.2 FOEs inhibit miR-29-Mediated gene expression

Results revealed that HC stimulation and the positive control RA did not alter miR-29 expression, while FOEs significantly upregulated miR-29, with a particularly dose-dependent effect observed on miR-29b (Figure 2). On the other hand, HC significantly promoted the expression of key lipid synthesis genes, including SCAP and ACC, whereas FOE treatment effectively reversed this upregulation. These findings suggested that FOEs inhibited the miR-29-mediated lipid synthesis genes and may represent an underlying mechanism through which FOEs suppress lipid synthesis.

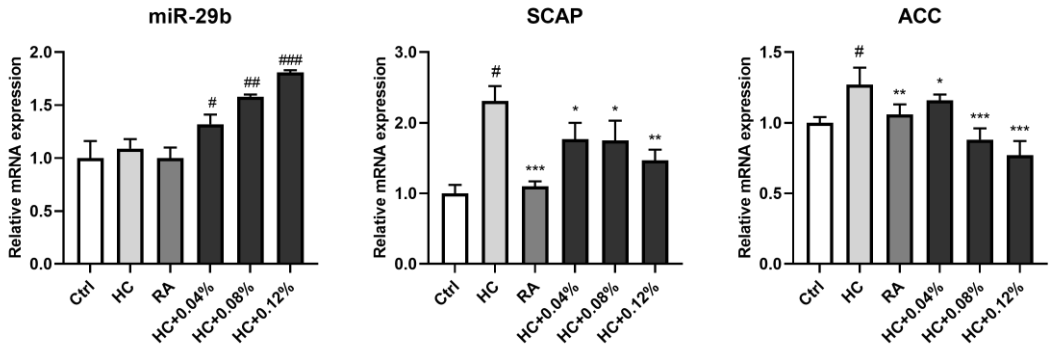

Figure 2. Inhibitory effects of FOEs on miR-29-mediated SCAP and ACC gene expression. # $P \leqslant 0.05$ vs. control; * $P \leqslant 0.05$, ** $P \leqslant 0.01$, *** $P \leqslant 0.001$ vs. HC group.

3.3 FOEs promoted HaCaT cell migration

The HaCaT cell viability assay revealed that FOEs at concentrations below 0.2% did not adversely affect the cells, with cell viability remaining above 90%. To determine the effects of FOEs on HaCaT cell migration, wound closure rates were measured to evaluate changes in migration ability. The results showed that FOEs significantly enhanced HaCaT cell wound healing compared to the untreated control group ($P \leqslant 0.01$), achieving a maximum healing rate of 4.29 times that of the control at 24 h (Figure 3). In summary, FOEs effectively promote cell migration, indicating their potential as a reparative agent.

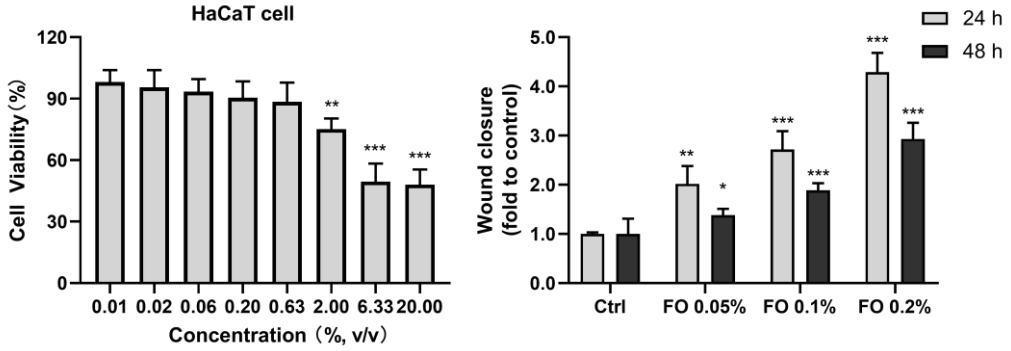

Figure 3. The effect of FOEs on the migration activity of HaCaT cells evaluated with the cell scratch test. * $P \leqslant 0.05$, ** $P \leqslant 0.01$, *** $P \leqslant 0.001$ vs. control.

3.4 FOEs improved oily sensitive skin condition

The results showed that after two weeks of applying a cream with 5% FOEs, facial sebum levels decreased by 42.5%, TEWL reduced by 25.1%, skin redness (a* value) decreased by 25.0%, and skin sensitivity to lactic acid diminished by 60.5% compared to baseline (all $P < 0.05$). No adverse reactions were observed among subjects.

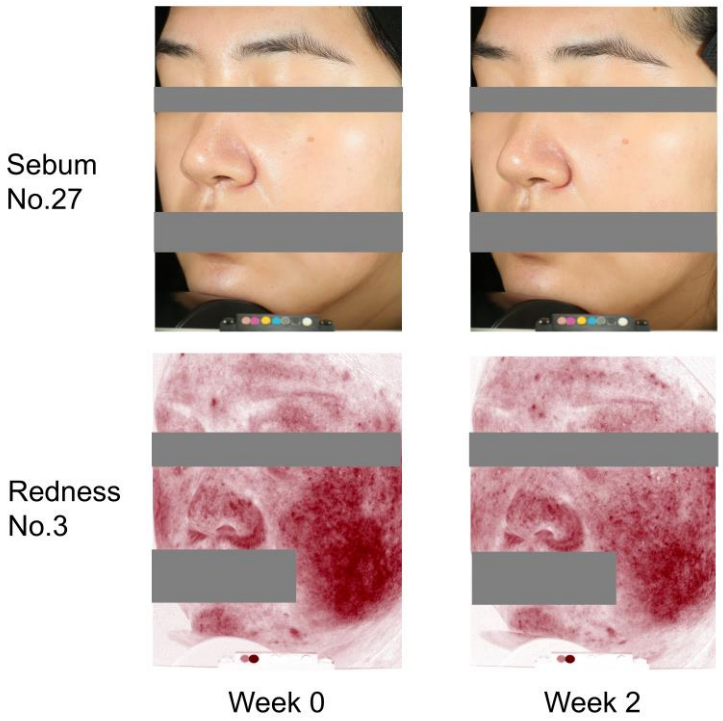

Figure 4. Typical images of subjects#3 and #27 after 2 weeks after using the cream.

4. Discussion

The stability of the levels and composition of lipids from sebaceous glands and intercellular lipids in the epidermis is crucial for the skin's ability to function as a barrier against harmful environmental threats [18]. Consequently, an imbalance in stratum corneum lipids may disrupt skin barrier function, potentially triggering various skin diseases and increasing the risk of skin cancer [19]. Notably, sebum produced by sebaceous gland cells exhibits antimicrobial activity, aiding in the control of the skin microbiome and preventing pathogen growth [20]; it acts as the first line of defense against external stimuli. However, sebaceous glands are more susceptible to hormonal and environmental influences than intercellular lipids, which can result in local microbial dysbiosis and the activation of antimicrobial peptides and pro-inflammatory cytokines [21]. This imbalance can lead to skin issues related to disrupted sebum secretion, such as oily sensitive skin (OSS). OSS is a distinctive skin type defined by excessive sebum production and increased sensitivity to external stimuli [22]. Recent advancements in technology, medicine, and the economy have highlighted the adverse emotional impacts associated with OSS, including negative self-perception, low selfesteem, depression, and distress, which have garnered increasing attention [4, 23]. Currently, most treatment protocols for oily sensitive skin tend to separate the goals of regulating sebum production from reducing skin sensitivity. However, it remains unclear whether addressing one aspect independently

may negatively impact the other. For example, many chemical exfoliants and surfactants can lower sebum production, but they may also compromise the sensitive skin barrier. Furthermore, some occlusive oils with skin barrier repairing properties may lead to increased sebum production, thereby exacerbating oily skin conditions. As Du suggests, the sensitivity of oily sensitive skin and sebum overproduction should not be treated in isolation [24]. This unmet clinical need emphasizes the urgency for natural therapeutics that can synchronize sebum suppression with reductions in skin sensitivity. *Fomes officinalis* (FO) is a fungus that has historically been utilized for its anti-inflammatory and wound-healing properties and is now emerging as a promising candidate for treating OSS. In this study, *in vitro* study indiciated the dual efficacy of *Fomes officinalis* extracts (FOEs) in inhibiting excessive sebum secretion and repairing the skin barrier.

As early as 2011, studies reported that miRNAs are involved in the regulation of lipid metabolism [25]. In 2016, Ru et al. first discovered that miR-29 regulates the SCAP/SREBP-1 pathway within the EGFR signaling pathway through a negative feedback mechanism, thus inhibiting lipid synthesis [10]. This process represents an essential self-regulatory mechanism in the body, helping to maintain lipid balance and stability. In subsequent years, miR-29 has emerged as a focal point of research within the miRNA family concerning lipid metabolism regulation. Research conducted by Julie et al. has demonstrated that miR-29 also inhibits insulin-stimulated lipid oxidation [9]. Additionally, miR-29 plays a role in regulating not only lipid metabolism but also immune modulation [38]. Recent research in 2024 indicated that miR-29 improves corneal damage by activating autophagy and exerting anti-inflammatory effects, suggesting its reparative properties [26]. Furthermore, miR-29 exhibits anti-photoaging properties by reducing collagen degradation in skin cells and decreasing the expression of matrix metalloproteinase MMP-2 [27]. Collectively, the studies provide compelling evidence that miR-29 is an ideal therapeutic target for skin issues related to sebaceous gland lipid imbalance, as it possesses dual functions of inhibiting sebum secretion and facilitating skin lesion repair. We established a model of excessive sebum secretion in SZ95 sebocytes using hydrocortisone (HC) to investigate the effects and mechanisms of FOEs on neutral lipid secretion. This investigation focused on the role of FOEs in regulating the miR-29 gene and its mediated key targets within the SCAP/SREBP-1 pathway, including SCAP and ACC. The results indicated that FOEs significantly inhibit neutral lipid production in a dose-dependent manner (Figure 1b). Gene expression analysis revealed that FOEs promote the upregulation of the miR-29 gene in a dose-dependent manner, particularly affecting miR-29b (Figure 2). Additionally, the expressions of SCAP and ACC showed dose-dependent suppression as well (Figure 2). Prior research has demonstrated that miR-29 negatively regulates lipid synthesis via the SCAP/SREBP-1 pathway. In cases of excess lipid secretion, miR-29 is upregulated, inhibiting the SCAP/SREBP-1 pathway and subsequently reducing lipid synthesis. However, following hydrocortisone (HC) treatment in SZ95 sebocytes, the SCAP/SREBP-1 pathway is activated while miR-29 expression remains unchanged, resulting in a significant increase in neutral lipid content. Conversely, FOEs treatment significantly upregulates miR-29, leading to a marked decrease in neutral lipid levels. This suggests that when the self-regulatory capacity of sebaceous glands is insufficient, FOEs can compensate for this deficiency by stimulating the miR-29-mediated negative feedback regulation of lipid synthesis pathways, thereby facilitating self-regulation of excessive sebum secretion by these glands, thereby restoring lipid homeostasis in the skin.

The epidermal barrier is recognized as the interface between the human body and the external environment. It serves to protect against mechanical, chemical, microbial, and environmental

factors, particularly ultraviolet (UV) radiation, while preserving skin softness and elasticity by preventing moisture loss from the dermis [28]. Another critical issue associated with OSS is barrier dysfunction; thus, repairing epidermal barrier damage represents a vital mechanism for alleviating OSS [29]. In this study, we examined the stimulating effects of FOEs on cell migration through a wound healing assay. Moreover, FOEs significantly enhanced cell migration, with the maximum healing rate at 24 h being 4.29 times that of the control group (Figure 3). This observation aligns with prior studies that indicated polysaccharides have significant barrier repair effects [28]. Based on previous findings, these results demonstrate that FOEs possess dual therapeutic effects *in vitro*, specifically inhibiting excess sebum secretion and repairing skin barrier dysfunction. Subsequently, we conducted a series of clinical trials to validate the results obtained from our *in vitro* experiments. We developed a cream containing 5% FOEs to evaluate the improvement of various clinical parameters in OSS participants in 2 weeks period. The results demonstrated that FOEs significantly improved the skin condition of OSS participants, particularly regarding sebum content, transepidermal water loss (TEWL) and a* value (Figure 4). Furthermore, FOEs significantly reduced participants' sensitivity to lactic acid, indicating that FOEs have the potential to improve the stress state of sensitive skin. In summary, FOEs represent a promising ingredient for managing OSS through inhibiting excessive sebum secretion and promoting barrier repair.

5. Conclusion

In conclusion, this study showed that FOEs simultaneously regulated lipid homeostasis and enhanced barrier repair, rendering them suitable natural cosmetic ingredients for managing OSS. Moreover, this study presented an innovative application of epigenetics in the skincare field, particularly regarding sebum control, and promotes further development of cosmetic ingredients targeting miRNAs.

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