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“Mobile Field-Based Extraction of Fresh *Prunus speciosa* Flower: A Solvent-Free, Waterless Novel Approach for Anti-Hair Loss and Scalp Care Benefits”

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1. Introduction

Alopecia has emerged as a global health issue, significantly affecting the quality of life and psychosocial well-being of over 2.5 billion individuals worldwide. Among various types of alopecia, Androgenetic Alopecia (AGA) has garnered considerable clinical attention due to its high prevalence accounting for approximately 80%-90% of male hair loss cases) [1]. AGA is characterized by the miniaturization of hair follicles and the shortening of the anagen (growth) phase. Androgens, particularly dihydrotestosterone (DHT), play a central role in the pathogenesis of AGA. DHT, a more potent androgen converted from testosterone via 5 α -reductase, binds to androgen receptors in hair follicle cells, leading to follicle miniaturization and disruption of the hair growth cycle [2].

Current therapeutic strategies primarily focus on stimulating hair follicle growth and modulating the hair cycle to prevent further hair loss. However, this follicular-centric approach often overlooks the scalp, which serves as the fundamental "soil" for hair growth. In reality, daily scalp care plays a vital role in hair loss prevention. Common scalp-related issues—such as oxidative stress, blue light exposure and excessive sebum production—are significant contributors to hair loss.

Oxidative stress, defined as a state of imbalance between reactive oxygen species (ROS) and the body's antioxidant defense systems, leads to cellular damage, including hair follicle atrophy and dysfunction [3]. Studies have identified premature senescence linked to oxidative stress in the hair follicles of AGA patients [4]. Blue light has been shown to impair the viability and proliferation of hair follicle stem cells (HFSCs) and dermal papilla cells (DPCs)—both essential for hair regeneration and growth. As a component of visible light, blue

light can penetrate the skin and induce intracellular ROS production, thereby triggering oxidative stress. This mechanism helps explain the observed association between chronic blue light exposure and hair thinning and alopecia [5]. Although excessive scalp sebum production may not directly cause hair loss, it can lead to conditions such as seborrheic dermatitis and folliculitis. These inflammatory scalp disorders can damage hair follicles, disrupt the hair cycle, and ultimately contribute to hair shedding [6]. Addressing scalp health as a means to prevent hair loss is therefore a highly promising and forward-looking strategy.

Using an innovative, mobile, inhouse GMP (Good Manufacturing Practice) truck, the flowers were extracted on-site via a green, low-temperature-vacuum method that eliminates the need for additional water or solvents, as shown in Figure 1(a). We obtained a Fresh *Prunus speciosa* flowers (FPSFE) with antioxidant, anti-blue light, oil control and anti-hair loss effects in Shandong, shown in Figure 1(b). The main advantage of this extraction method is that the active ingredients in the plant are retained to the greatest extent, and the loss or degradation of ingredients caused by storage, transportation and drying are reduced[7]. LCMS analysis of FPSFE extract was mainly composed of Prunin, as shown in Figure 1(c).

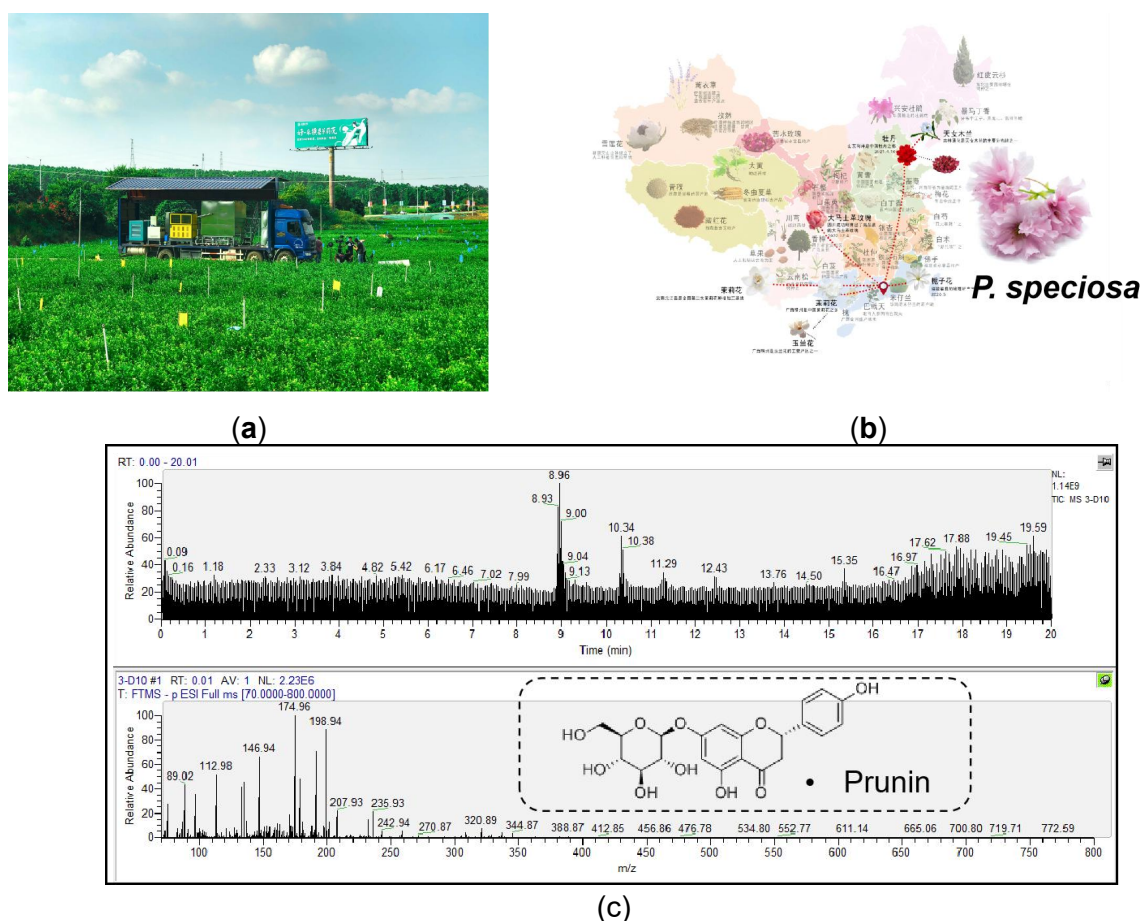


Figure 1. (a) The image of the mobile extraction workshop, (b) *Prunus speciosa* flowers are produced in Shandong, (c) LCMS analysis of FPSFE with the structure of Prunin.

2. Materials and Methods

2.1. FPSFE via Mobile Field-Based Extraction

A mobile low-temperature vacuum extraction unit was deployed to the fresh *Prunus speciosa* flower harvesting site, enabling on-site extraction of the raw water extract while simultaneously collecting fresh flowers. This immediate processing ensured the preservation of active compounds by minimizing delays and eliminating the need for storage or transportation prior to extraction. The residual flower material was collected, dried and stored, and then transported to the factory for further processing. Through subsequent extraction, purification, concentration, and drying steps, *Prunus speciosa* flower powder was obtained. The final formulation of FPSFE was produced by dissolving and combining the raw water extract with the flower powder.

2.2. Antioxidant Assay by DPPH

To evaluate the antioxidant capacity, the DPPH radical scavenging assay was performed. A volume of 1 mL of the sample solution was mixed with 3 mL of 50.0 µg/mL DPPH solution prepared in absolute ethanol. The mixture was incubated at room temperature in the dark for 30 minutes and the absorbance was measured at 517 nm to obtain the value A₁. In parallel, 1 mL of the sample solution was mixed with 3 mL of absolute ethanol (without DPPH) and incubated under the same conditions to measure background absorbance, yielding A₂. Additionally, 1 mL of the sample solvent (without the sample) was mixed with 3 mL of DPPH solution and incubated similarly to obtain A₀ as the control. The DPPH radical scavenging activity was calculated using the following formula:

$$\text{DPPH scavenging rate (\%)} = [1 - (A_1 - A_2) / A_0] \times 100\%$$

2.3. 5α-Reductase Inhibition Assay

To assess 5α-reductase inhibitory activity, 1 mL of the sample working solution was mixed with 1 mL of enzyme solution, 1 mL of NADPH solution, and 1 mL of testosterone solution. The mixture was gently shaken, and 200 µL was transferred into a 96-well microplate. Absorbance at 340 nm was measured immediately ($\bar{A}_{\text{sample } 0}$), and again after incubation at 37 °C for 20 minutes ($\bar{A}_{\text{sample } 20}$). An enzyme control group was prepared using PBS instead of the sample solution, and absorbance was similarly recorded before ($\bar{A}_{\text{enzyme } 0}$) and after incubation ($\bar{A}_{\text{enzyme } 20}$). Each group was tested in triplicate. Blank (BP) and positive control (PC) groups were also included under the same conditions. The 5α-reductase inhibition rate was calculated using the formula:

$$\text{Rate of inhibition (\%)} = \left[1 - \frac{\bar{A}_{\text{sample } 0} - \bar{A}_{\text{sample } 20}}{\bar{A}_{\text{enzyme } 0} - \bar{A}_{\text{enzyme } 20}} \right] * 100\%$$

2.4. Apoptosis Assay

Human dermal fibroblasts (HDFs) in the logarithmic growth phase were cultured in DMEM complete medium supplemented with 10% fetal bovine serum and 1% penicillin–streptomycin in 25 cm² culture flasks at 37 °C in a 5% CO₂ atmosphere. When cell confluence reached approximately 60–70%, the medium was removed, and cells were washed twice with sterile

PBS to eliminate residual medium. For the experimental group, 2 mL of FPSFE (5%) was added and incubated for 24 h at 37 °C. For the control group, 2 mL of serum-free RPMI-1640 medium was added under the same conditions. After incubation, cells from both groups were digested with 0.25% trypsin (without EDTA), harvested into centrifuge tubes, and centrifuged at 1000 rpm for 5 minutes. The cell pellets were washed twice with ice-cold PBS, with centrifugation at 1000 rpm for 5 minutes between washes. Cells were then resuspended in 1× binding buffer, and the cell concentration was adjusted to 1.5×10^6 cells/mL. A 100 µL aliquot of cell suspension was stained with 5 µL Annexin V–FITC and 10 µL 7-AAD. After gentle mixing, samples were incubated in the dark for 15 minutes at room temperature. Subsequently, 380 µL of ice-cold 1× binding buffer was added to each tube. Samples were divided into three groups: Group 1 and Group 2 contained control cells, while Group 3 contained FPSFE-treated cells. Groups 2 and 3 were exposed to LED blue light ($160 \mu\text{W}/\text{cm}^2$) for 2 hours at room temperature, while Group 1 was kept in the dark as a blank control. Apoptosis was analyzed using an Attune NxT flow cytometer (Thermo Fisher Scientific).

2.5. Determination of Reactive Oxygen Species (ROS) Content

HDFs in logarithmic growth phase were cultured in DMEM complete medium containing 10% (v/v) serum and 1% (v/v) streptomycin in 6-well plates at 37 °C with 5% CO₂. When the cell confluence reached approximately 60 – 70%, the medium was removed, and cells were washed twice with sterile PBS to eliminate residual medium. For the experimental group, 2 mL of cherry blossom extract solution (5% w/w, prepared from the water-soluble extract powder of *Prunus speciosa* flowers) was added to each well and incubated at 37 °C for 24 hours. For the control group, 2 mL of serum-free RPMI-1640 medium was added under the same conditions. After incubation, the treated cells were divided into four groups: Group 1 (experimental group), and Groups 2 – 4 (control group). Groups 1 and 2 were exposed to blue LED light ($160 \mu\text{W}/\text{cm}^2$) for 1 hour at room temperature, while Group 3 was kept in the dark at room temperature. Group 4 served as an untreated blank. Following blue light treatment, 500 µL of 5 µM H₂-DCFDA solution was added to each well, and cells were incubated at 37 °C for 10 minutes in the dark. After incubation, cells were washed with PBS to remove excess dye. ROS levels were determined by measuring fluorescence intensity using a fluorescence microplate reader, and data were analyzed accordingly.

2.6. Clinical Trials

A scalp serum formulation containing FPSFE was clinically evaluated in 30 participants with sensitive scalp conditions over a 3-month period to assess its real-world efficacy. Participants were instructed to apply the serum once daily directly onto the scalp. The assessment of scalp condition, including hair loss, sensitivity, and overall hair health, was performed at baseline and after 3 months of continuous use. Various objective measures, such as hair density and thickness, along with subjective assessments of scalp comfort and irritation, were recorded using both clinical grading scales and participant questionnaires. The efficacy of the formulation was compared to baseline data, with statistical analysis conducted to evaluate any significant improvements.

3. Results

3.1. Antioxidant Assay by DPPH

In this study, the DPPH assay was employed to evaluate the free radical scavenging ability of FPSFE, reflecting its antioxidant potential. As shown in Figure 2(a), a 1% Vitamin C (VC) solution ($50.3 \mu\text{g/mL}$) was used as a positive control, exhibiting a scavenging rate of 89.04%. FPSFE, at a concentration of 1% (w/w) active substance, demonstrated a comparable free radical scavenging rate of 83.22%, indicating that FPSFE possesses strong antioxidant activity.

3.2. 5 α reductase Inhibition

As shown in Table 1 and Figure 2(b), the positive control exhibited a 5 α -reductase inhibition rate of 77.50%, while FPSFE, at an active substance concentration of 0.2% (w/w) achieved an inhibition rate of 88.79%. This demonstrates that FPSFE possessed excellent 5 α -reductase inhibition potential. Additionally, a complex composition formulation (Composition) combining 0.2% (w/w) FPSFE, 0.0002% (w/w) Pisum sativum pea extract, 0.3% (w/w) niacinamide, and 0.15% (w/w) hydrogenated starch hydrolysate resulted in a remarkable 5 α -reductase inhibition rate of 99.71%. These findings suggest that FPSFE exhibits synergistic effects when combined with other ingredients, providing a strong foundation for further in-depth research.

Table 1. 5 α -reductase inhibition rate

Group	Inhibition rate of 5 α reductase [%]
Blank Control	0.00
Positive Control	77.50
0.2% FPSFE	88.79
Composition	99.71

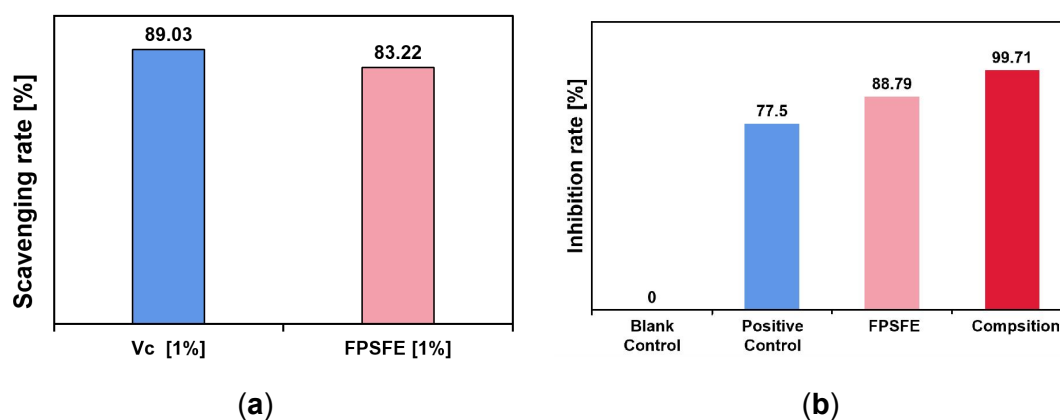


Figure 2. (a) DPPH radical scavenging activity of 1% VC (blue) and 1% FPSFE (pink). (b) 5 α -Reductase inhibition rate: Positive Control (blue), FPSFE (pink), and Composition (red).

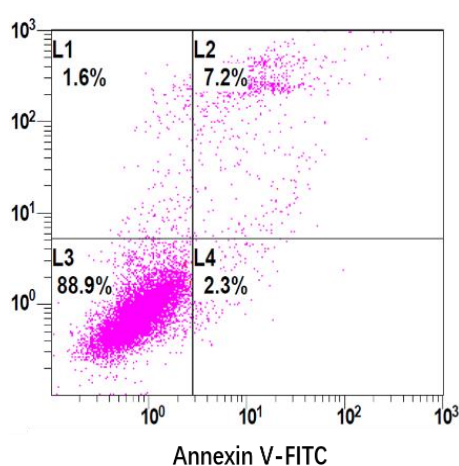
3.3. Anti-Blue Light

The anti-blue light ability of FPSFE has been evaluated through cell apoptosis analysis and ROS content measurement. As shown in Figure 3(b), cells without FPSFE were irradiated with blue light. The results revealed that 4.1% of the cells remained alive, 68.5% underwent apoptotic, and 27.5% were dead. These findings, compared to control cells, demonstrate that blue light irradiation caused significant damage to the cells, particularly increasing the proportion of apoptotic cells, as illustrated in Figure 3(a). In Figure 3(c), compared to the experimental group, the live cells percentage increased to 80.7%, apoptotic cells decreased to 68.5%, and dead cells dropped to 7.4%. This indicates that FPSFE can mitigate the damage of blue light at the cellular level, providing a better anti-blue light effects. Detailed results can be found in Table 2 and Figure 3.

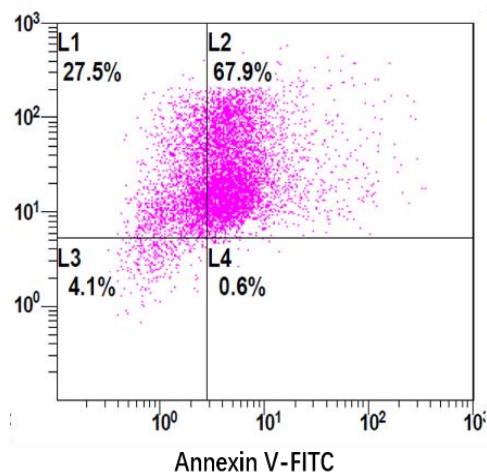
As shown in Figure 3(d), the ROS content in cells exposed to blue light was significantly higher than that in cells without blue light ($P < 0.05$). However, in cells treated with FPSFE after blue light exposure, the ROS content was significantly lower than in untreated cells. These results suggest that FPSFE reduces ROS production induced by blue light exposure, demonstrating its effective anti-blue light properties at the cellular level.

Table 2. Detailed data on apoptosis between experimental groups and control groups

Group	Living cell L3 [%]	Apoptotic cells L2+L4 [%]	Dead cells L1 [%]
Control	88.9	9.5	1.6
Blue light exposure	4.1	68.5	27.5
Blue light exposure + FPSFE	80.7	11.9	7.4



(a)



(b)

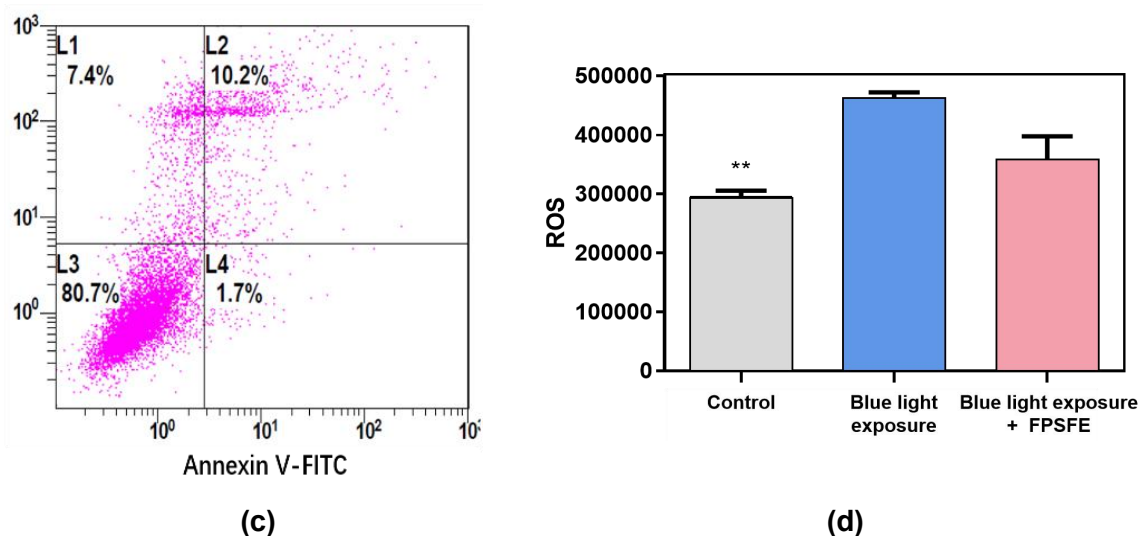


Figure 3. Annexin V-FITC/PI double staining assay: (a) Cells of the control group, (b) Cells exposed to blue light, (c) Cells exposed to blue light + FPSFE, (d) ROS content test: Cells of the control group (gray), Cells exposed to blue light (blue), Cells exposed to blue light + FPSFE (pink).

3.5. Clinical Trials

It is noteworthy that FPSFE was incorporated into a serum formulation for clinical testing of its anti-hair loss efficacy. After 12 weeks of treatment, a significant reduction in hair loss was observed compared to baseline ($p < 0.001$). The results demonstrated a 14.79% improvement in hair loss, which was 223.87% higher than the improvement observed in the control group after 12 weeks. Additionally, the overall hair density improvement rate reached 5.32%, which was 129.51% greater than that of the control group. Compared with the clinical picture before use, the hair density increased significantly after 12 months. Detailed data are presented in Table 3 and Figure 5.

Table 3. Clinical anti-hair loss data from a scalp serum formulation containing FPSFE

Test	Group	4 week	8 week	12 week
Hair loss count test	p	0.030	<0.001	<0.001
	Significance	*b	***b	***b
Improvement rate of hair loss	FPSFE	4.19%	8.17%	14.79%
	Control	-7.21%	-5.86%	-11.94%
Overall hair density test	p	0.317	0.034	0.004
	Significance	nsa	*b	**b
Improvement rate of Hair density	FPSFE	0.53%	3.19%	5.32%
	Control	-0.52%	-1.55%	-2.07%

a: indicates no significant difference compared with before use ($p > 0.050$)

b: indicates a significant difference compared with that before use, * ($0.010 < p < 0.050$), ** ($0.001 \leq p < 0.010$), *** ($p \leq 0.001$)

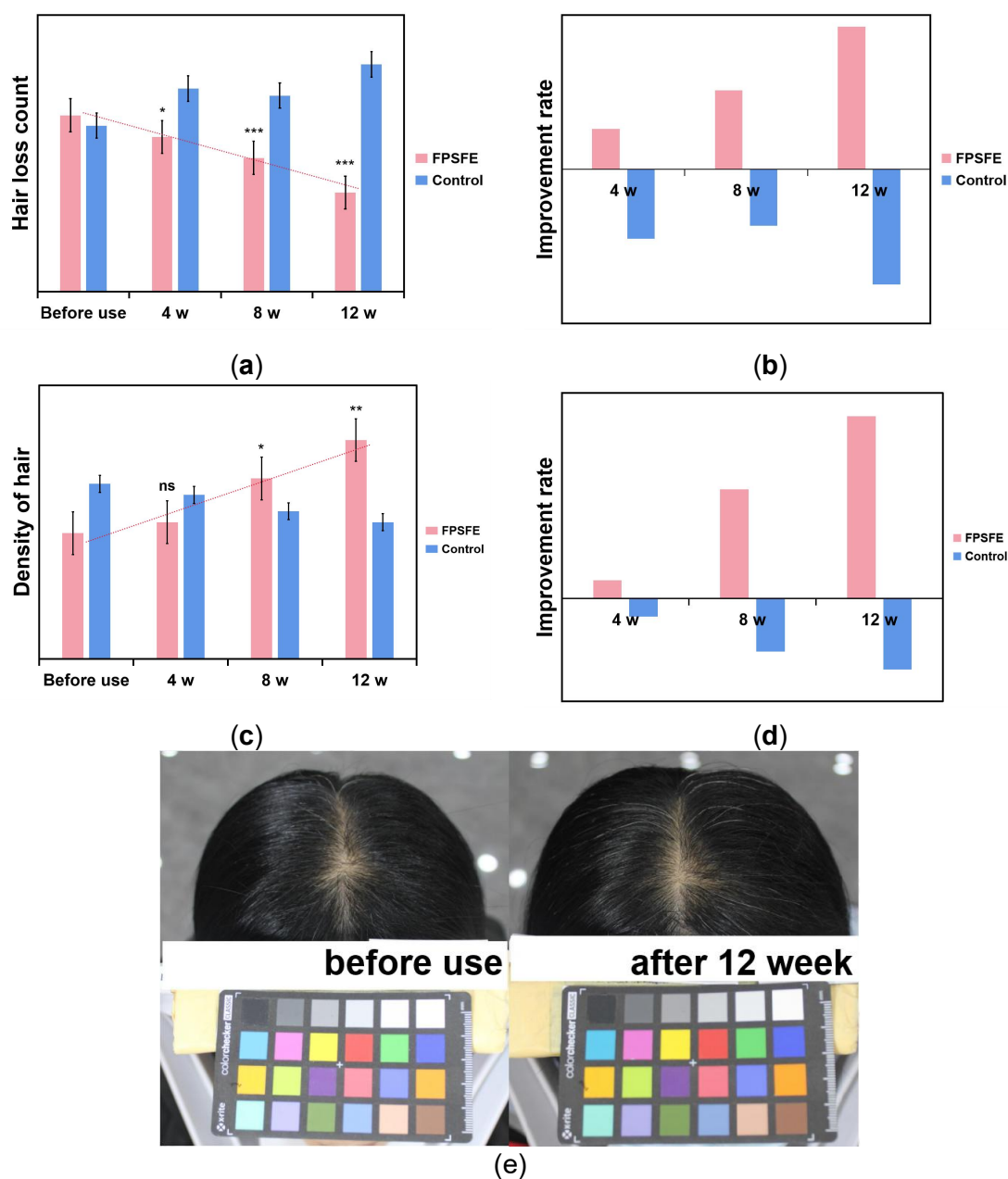


Figure 5. Clinical anti-hair loss data from a scalp serum formulation containing FPSFE: (a) Hair loss count test results, (b) Improvement rate of hair loss count relative to the mean initial value, (c) Overall hair density test results, (d) Improvement rate in overall hair density relative to the mean initial value, (e) Overall hair density of volunteers before use compared with 12 weeks later.

4. Discussion

Scalp care has emerged as a significant trend, with the scalp no longer being viewed merely as an appendage of the hair but as an integral part of the skin that requires dedicated attention. The health of the scalp directly influences hair growth and appearance., as healthy scalp forms the foundation for healthy hair growth. Effective scalp care focuses on reducing

inflammation and oxidative stress, thereby fostering an environment conducive to optimal hair growth.

Common triggers of scalp damage, such as air pollution particles and visible light, are frequently overlooked. Airborne pollutants like PM_{2.5} can settle on the scalp, causing inflammation and oxidative stress. These particles directly irritate the scalp, disrupt the scalp barrier, and lead to moisture loss and dryness. In this study, the antioxidant capacity of FPSFE was confirmed, with DPPH free radical scavenging rate reaching 83.22%. This suggests that FPSFE may help alleviate hair loss and support healthy hair growth by mitigating oxidative stress, protecting the scalp, and improving its overall health. Visible blue light, whether from sunlight or electronic devices, is another source of oxidative stress and inflammation on the scalp. Excessive intracellular ROS can overwhelm the antioxidant system, leading to cellular damage. In the scalp, blue light-induced oxidative stress can harm hair follicle cells, impair their function, and eventually result in scalp and hair damage. Additionally, direct exposure to blue light can reduce cell viability and even trigger apoptosis. In this study, cells exposed to blue light showed a significantly improved survival rate (80.7%), when treated with FPSFE, a 1868% increase compared to untreated cells (4.1%). This demonstrates FPSFE's ability to protect the scalp from blue light-induced damage. Furthermore, FPSFE treatment significantly reduced intracellular ROS content, supporting its antioxidant effect.

AGA is the most prevalent form of hair loss. The enzyme 5 α -reductase plays a crucial role in converting testosterone into the more potent androgen DHT, which is a major cause of AGA. DHT also contributes to increased scalp sebum production by stimulating sebaceous glands. Therefore, inhibition of 5 α -reductase activity can reduce DHT levels, lowering the risk of AGA and controlling excess sebum secretion, ultimately preventing hair loss and managing oily scalp. In this study, FPSFE exhibited 5 α -reductase inhibition rate of 88.79%, demonstrating its potential to prevent hair loss and regulate scalp oiliness. Additionally, when FPSFE was combined with four other components, the inhibition rate increased to 99.71%, highlighting the synergistic potential of FPSFE in combination therapies. This finding warrants further exploration of FPSFE in combination with other agents for enhanced efficacy.

Furthermore, a clinical trial involving 30 participants evaluated the efficacy of a scalp serum formulation containing FPSFE over a 3-month period. After 12 weeks, the clinical data showed that the FPSFE-containing serum significantly reduced hair loss and increased overall hair density, demonstrating its effectiveness in promoting hair growth. These results underscore the potential of FPSFE as an effective anti-hair loss agent when incorporated into topical formulations.

5. Conclusion

This study presents an innovative Mobile-Field-Based Extraction system for directly obtaining raw water from fresh *Prunus speciosa* flowers. The effective recycling of flower residue leads to the production of FPSFE, which offers various scalp and hair care benefits, including

anti-hair loss properties. Experimental results demonstrated FPSFE's strong antioxidant and anti-blue light effects, providing protection against environmental pollutants and blue light exposure. Additionally, FPSFE exhibited a high 5 α -reductase inhibition rate and promising clinical outcomes, highlighting its potential to control scalp oil, prevent hair loss, and enhance hair density. These findings suggest FPSFE as a promising ingredient for future hair care formulations. The solvent-free, waterless extraction process is eco-friendly and preserves bioactive potency, making FPSFE a natural solution for hair loss. Future research will explore its potential for scalp relief, hydration, oil control, and damage repair.

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