乾燥肌と敏感肌とオイリー肌の違い

-角質層の構造・外部刺激に対する反応・ターンオーバー-

1. 角質層の構造の違い:

- 乾燥肌は、皮膚バリアの要である細胞間脂質のラメラ構造が壊れ、角質細胞間に隙間ができ、バリア機能が低下しています。この隙間を埋めるために、角質細胞同士が物理的に近づき、デスモソームという接着因子の働きで強く結びついてしまいます。その結果、古い角質が剥がれにくくなり、角質肥厚を起こします。
- 敏感肌は免疫力のある肌の方に多い肌質で、微小な刺激にも免疫反応が起こりやすい肌です。そのため、角質肥厚を起こす前に免疫反応が起こり、炎症が生じます。乾燥肌の場合と同じように、皮膚バリアの要である細胞間脂質のラメラ構造が壊れ、角質細胞間に隙間ができ、バリア機能が低下しています。ただし、炎症が続くと、角質肥厚が起こります。敏感肌では、免疫反応が優先されます。
- オイリー肌は、角質層表面が、脂っぽい皮脂膜で覆われています。毛穴詰まり・アクネ菌の増殖・過酸化脂質による角質肥厚などの問題が生じます。これらの問題を解消するために、オイリー肌の人は、脂っぽい角質表面を嫌って、頻繁に洗顔料を使った洗顔を行います。これが逆効果になり、バリア機能のある皮膚バリアを壊し、角質水分保持力や過敏反応抑制力を低下させ、インナードライ(内側の乾燥肌)や敏感肌を悪化させます。

2. 外部刺激に対する反応の違い:

- 乾燥肌は免疫力が強い方ではないので、即座に角質肥厚で対応しようとします。
- 敏感肌は免疫力が強く、即座に免疫反応で対応しようと Lます。
- オイリー肌は、両方の反応(角質肥厚・免疫反応)で対応 しようとします。

3. **ターンオーバーの違い**:

- 乾燥肌は、角質水分保持力低下による水分不足が原因で起きる不全角化。未熟な角質細胞の生成・増殖。剥離過程で、角質細胞同士が強く結びついてしまい、古い角質が長く肌表面に留まりやすいです。角質肥厚を起こします。
- 敏感肌は、過敏反応抑制力低下による炎症が原因で起きる不全角化。未熟な角質細胞の生成・増殖。剥離過程で、過剰に剥がれるため、炎症を起こし、角質肥厚は起こさない。ただし、炎症が続くと角質肥厚を起こします。
- オイリー肌は、両方の状態(角質水分保持力低下による 水分不足が原因で起きる、過敏反応抑制力低下による 炎症が原因で起きる不全角化・過角化)が起こります。

参考文献: AI アシスタントとの情報作成協業

Differences Between Dry, Sensitive, and Oily Skin

-Structure of the Stratum Corneum Response to External Stimuli Turnover-

1. Differences in the Structure of the Stratum Corneum:

- **Dry Skin:** The lamellar structure of intercellular lipids, which is essential for the skin barrier, is broken down, creating gaps between corneocytes and resulting in decreased barrier function. To fill these gaps, corneocytes physically come closer together and, with the help of adhesion factors called desmosomes, they bind tightly. As a result, old corneocytes are less likely to shed, leading to hyperkeratosis.
- Sensitive Skin: This skin type is common among those with strong immune responses, and it easily triggers immune reactions even to minor stimuli. Therefore, immune reactions occur before hyperkeratosis develops, causing inflammation. Similar to dry skin, the lamellar structure of intercellular lipids is broken down, creating gaps between corneocytes and resulting in decreased barrier function. However, prolonged inflammation can lead to hyperkeratosis, and immune reactions are prioritized in sensitive skin.
- Oily Skin: The surface of the stratum corneum is covered by an oily lipid film. This results in issues such as clogged pores, proliferation of acne bacteria, and hyperkeratosis due to oxidized lipids. To solve these problems, individuals with oily skin often use cleansers frequently, which can have the opposite effect. Excessive cleansing can break down the skin barrier, reducing moisture retention and sensitivity suppression capabilities, worsening inner dryness (dry skin from within) and sensitivity.

2. Differences in Response to External Stimuli:

- **Dry Skin:** As it doesn't have strong immunity, it responds with immediate hyperkeratosis.
- **Sensitive Skin:** With strong immunity, it responds immediately with immune reactions.
- **Oily Skin:** It responds with both types of reactions (hyperkeratosis and immune reactions).

3. Differences in Turnover:

- Dry Skin: Insufficient keratinization occurs due to moisture deficiency caused by decreased moisture retention capabilities. This leads to the generation and proliferation of immature corneocytes. During the isolation process, corneocytes bind tightly, causing old corneocytes to stay on the skin surface longer, resulting in hyperkeratosis.
- Sensitive Skin: Insufficient keratinization occurs due to inflammation caused by decreased sensitivity suppression capabilities. This leads to the generation and proliferation of immature corneocytes. During the shedding process, excessive shedding occurs, leading to inflammation without causing hyperkeratosis. However, prolonged inflammation can lead to hyperkeratosis.
- Oily Skin: Both conditions (moisture deficiency due to decreased moisture retention capabilities and inflammation caused by decreased sensitivity suppression capabilities leading to insufficient and excessive keratinization) can occur.

References: Collaboration in Information Creation with Al Assistant