ORIGINAL RESEARCH ORİJİNAL ARAŞTIRMA

DOI: 10.5336/ophthal.2025-109749

# The Impact of Androgenetic Alopecia on Ocular Health: An Investigation of Dry Eye and Meibomian Gland Dysfunction: Cross-Sectional study

## Androgenetik Alopesi ve Oküler Yüzey: Kuru Göz Hastalığı ve Meibomian Bezi Fonksiyonları Üzerine Etkileri: Kesitsel Çalışma

Sezer HACIAĞAOĞLU[a], Fahri Onur AYDIN[b], Ece ALTUN[c]

[a]Bahçeşehir University Faculty of Medicine, Department of Ophthalmology, İstanbul, Türkiye
[b]Başakşehir Çam and Sakura City Hospital, Clinic of Ophthalmology, İstanbul, Türkiye
[c]Medipol University Hospital, Clinic of Dermatology and Venereology, İstanbul, Türkiye

**ABSTRACT Objective:** To investigate the impact of androgenetic alopecia on ocular health, particularly focusing on dry eye disease and meibomian gland dysfunction in female patients. **Material and Methods:** A cross-sectional observational study was conducted involving 28 eyes of female patients diagnosed with androgenetic alopecia and 32 eyes of healthy controls. Participants underwent dry eye tests, including the Schirmer-I test, Oxford score, and Ocular Surface Disease Index. Meibomian gland morphology was evaluated using dynamic meibography, and tear film stability was assessed through non-invasive tear breakup time and tear meniscus height measurements. Meibomian gland quality and function were graded using standardized scoring systems. **Results:** Although patients with androgenetic alopecia exhibited higher Schirmer test values and Oxford scores compared to controls, the differences were not statistically significant. Similarly, no significant differences were found in Ocular Surface Disease Index, non-invasive tear breakup time, tear meniscus height, or blink rate. Meiboscores were comparable between both groups. However, meibum quality was significantly better in the androgenetic alopecia group (p=0.001). **Conclusion:** While androgenetic alopecia may influence meibomian gland function, this study found no significant differences in dry eye parameters between androgenetic alopecia patients and healthy controls. These findings suggest that the hormonal interplay between sex steroids and the ocular surface is complex, and further research with larger cohorts is needed to fully understand the ocular implications of androgenetic alopecia.

**Keywords:** Androgenetic alopecia; dry eye disease; meibomian gland dysfunction; sex steroids; female pattern hair loss

**ÖZET Amaç:** Bu çalışmanın amacı, kadın hastalarda androgenetik alopesinin oküler sağlık üzerindeki etkisini incelemek, özellikle kuru göz hastalığı ve meibomian bezi disfonksiyonu ile olan ilişkisini değerlendirmektir. **Gereç ve Yöntemler:** Androgenetik alopesi tanısı konmuş kadın hastalara ait 28 göz ve sağlıklı kontrol grubuna ait 32 gözün dâhil edildiği kesitsel gözlemsel bir çalışma yürütülmüştür. Katılımcılara Schirmer-I testi, Oxford skoru ve Oküler Yüzey Hastalık İndeksi gibi kuru göz testleri uygulanmıştır. Meibomian bezi morfolojisi, dinamik meibografi ile değerlendirilmiş, gözyaşı filmi stabilitesi, invaziv olmayan gözyaşı kırılma süresi ve gözyaşı menisküs yüksekliği ölçümleri ile analiz edilmiştir. Meibomian bezi kalitesi ve fonksiyonu, standart skorlamalar kullanılarak derecelendirilmiştir. **Bulgular:** Androgenetik alopesili hastalarda Schirmer testi değerleri ve Oxford skorları, kontrol grubuna kıyasla daha yüksek olmasına rağmen bu fark istatistiksel olarak anlamlı bulunmamıştır. Aynı şekilde Oküler Yüzey Hastalık İndeksi, invaziv olmayan gözyaşı kırılma süresi, gözyaşı menisküs yüksekliği ve göz kırpma hızı açısından da anlamlı bir farklılık saptanmamıştır. Meiboskor değerleri, her iki grupta benzer bulunmuştur. Ancak meibum kalitesi, androgenetik alopesi grubunda anlamlı derecede daha iyi olarak tespit edilmiştir (p=0,001). **Sonuç:** Androgenetik alopesi, meibomian bezi fonksiyonunu etkileyebilecek bir faktör olmakla birlikte bu çalışmada, androgenetik alopesi hastaları ile sağlıklı kontroller arasında kuru göz parametreleri açısından anlamlı bir fark bulunmamıştır. Bu bulgular, cinsiyet steroidleri ile oküler yüzey arasındaki hormonal etkileşimin karmaşık bir yapıya sahip olduğunu göstermekte olup, androgenetik alopesinin oküler etkilerini tam olarak anlayabilmek için daha geniş hasta grupları ile yapılacak ileri çalışmalara ihtiyaç duyulduğunu ortaya koymaktadır.

**Anahtar Kelimeler:** Androgenetik alopesi; kuru göz hastalığı; meibomian bezi disfonksiyonu; cinsiyet steroidleri; kadın tipi saç dökülmesi

**Correspondence:** Sezer HACIAĞAOĞLU
Bahçeşehir University Faculty of Medicine, Department of Ophthalmology, İstanbul, Türkiye
**E-mail:** drsezerh@hotmail.com

Peer review under responsibility of Turkiye Klinikleri Journal of Ophthalmology.

***Received:*** 20 Feb 2025 ***Received in revised form:*** 05 Jul 2025 ***Accepted:*** 24 Jul 2025 ***Available online:*** 03 Sep 2025

Androgenetic alopecia (AGA) is characterized by progressive thinning and reduction of hair follicles in a specific pattern, affecting both men and women.[1] The condition can onset after puberty and tends to worsen with age, being more prevalent, especially among women during the postmenopausal period.[2] This condition is characterized by the miniaturization of hair follicles and a reduction in hair density, predominantly affecting the central and frontal scalp regions. It is hypothesized that AGA develops due to an increased number of androgen receptors and abnormal sensitivity of hair follicles to circulating androgens.[3] By changing testosterone into 5-alpha-dihydrotestosterone, the enzyme 5-alpha-reductase plays a vital role in causing hair follicle thinning, which in turn causes terminal hairs to become vellus hairs.[2,3]

Although traditionally associated with cosmetic and psychological concerns, emerging evidence suggests that AGA may also have systemic implications, including effects on sebaceous glands, such as meibomian glands, which are crucial for ocular surface homeostasis. The interplay between hormonal fluctuations, particularly androgens and estrogens, and meibomian gland function has been highlighted in several studies, suggesting that hormonal imbalances might contribute to dry eye disease (DED) and meibomian gland dysfunction (MGD).[4,5] Androgens also activate 25 distinct ontologies (with 5 genes) involved in lipid synthesis, homeostasis, transport, and binding, in addition to the dynamics of cholesterol, fatty acids, phospholipids, and steroids.[6] In the meibomian glands of both male and female mice, testosterone also significantly reduces keratinization.[7] The fact that topical androgens enhance the synthesis and secretion of meibomian gland lipids, improve meibum quality, extend the tear film breakdown time, and decrease evaporative DED in humans may be explained by these combined androgen actions that promote lipogenesis and decrease keratinization.

DED is a multifactorial condition, with endocrine abnormalities playing a significant role in its etiology.[8] Physiological factors such as menopause and menstrual cycle changes, as well as conditions like polycystic ovary syndrome and androgen resistance, along with iatrogenic factors such as contraceptive use and antiandrogen therapy, contribute to its development.[8] The ocular surface epithelium, including the meibomian glands responsible for the tear film's lipid layer, contains receptors for sex steroid hormones.[9] Androgens stimulate the secretion of both the quantity and quality of lipids from the meibomian glands, whereas estrogens and progesterones inhibit sebaceous gland function, leading to reduced lipid production.[9]

This study aimed to determine whether female patients with AGA were more likely than healthy people to experience MGD and DED.

## MATERIAL AND METHODS

This cross-sectional observational study included 28 eyes of 28 patients diagnosed with AGA (Group 1) and 32 eyes of 32 healthy volunteers (Group 2). All female patients with AGA were assessed and diagnosed by the same dermatologist (EA) and had not received any prior treatment. Following a comprehensive ophthalmologic examination, participants underwent evaluations including corneal and conjunctival fluorescein staining (Oxford score), and the Schirmer-I test. Subjective symptoms were assessed using the Ocular Surface Disease Index (OSDI) scoring. Meibomian gland morphology was visualized using dynamic meibography (Lipiscan, TearScience Inc., Morrisville, NC, USA). The meibomian glands in each upper and lower eyelid were assessed by the same investigator (SH) using the grading system described by Arita et al., categorizing them into stages based on partial or total loss rates. A meiboscore ranging from 0 to 3 was assigned: Stage 0 (no loss), Stage 1 (less than 1/3 loss), Stage 2 (1/3 to 2/3 loss), and Stage 3 (more than 2/3 loss) (Figure 1).[10] The meiboscores from both upper and lower lids were combined to determine the total meiboscore for each eye. The quality of meibum produced by the glands [meibum quality score (MQS)] was graded as follows: Stage 0 (transparent), Stage 1 (cloudy), Stage 2 (containing debris), and Stage 3 (toothpaste consistency).[11] Additionally, non-invasive tear breakup time (NIBUT), tear meniscus height, and the number of blinks per minute were evaluated and recorded using MYAH equipment from Topcon Healthcare Inc., Tokyo, Japan, for all participants. All

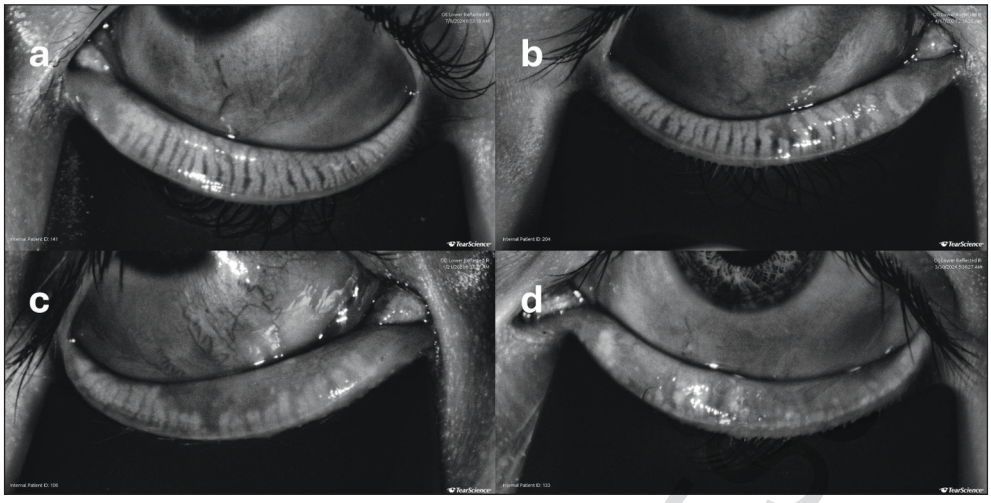


**FIGURE 1: a)** Stage 0 (no loss); **b)** Stage 1 (less than 1/3 loss); **c)** Stage 2 (1/3 to 2/3 loss); **d)** Stage 3 (more than 2/3 loss)

tests were conducted in the same examination room and under the same temperature setting.

The study was approved by Medipol University's Institutional Review Board/Ethics Committee (date: September 14, 2022; no: E-10840098-772.02-5297) and strictly followed the Declaration of Helsinki's guidelines. Informed consent was obtained from all participants.

Patients with prior use of tear drops or contact lenses, history of eye surgery with dry eye as a side effect, systemic medications like isotretinoin and diuretics, systemic, ocular, and dermatologic conditions known to cause dry eye, as well as congenital or acquired ocular surface disorders were excluded from the study.

### STATISTICAL ANALYSIS

SPSS for Windows (Version 25.0, IBM Corp, NY, USA) was utilized for statistical analysis. Descriptive statistics included mean, standard deviation, and range (minimum-maximum). The Kolmogorov-Smirnov test and box plot graphs assessed the normality of variables. Pairwise comparisons of normally distributed variables employed the student t-test, while variables not following normal distribution were analyzed using the Mann-Whitney U test. Statistical significance was set at $p<0.05$. Sample size calculation was performed using G*Power software based on a preliminary power analysis from Kiyat et al., aiming for a type 1 error rate and power of 0.8, with an estimated sample size of 28 per group.[3]

## RESULTS

The mean ages of Group 1 and Group 2 were 29.5±5.1 and 31.2±9.2, respectively (p=0.527). Table 1 presents the dry eye test results for both groups. Compared to Group 2, Group 1 had higher Oxford score and Schirmer test values, although these differences were not statistically significant (p=0.457 and 0.201, respectively). There were no significant differences in OSDI score and NIBUT between the groups (p=0.857 and 0.773, respectively). Although Group 1 had a lower tear meniscus, this disparity was not statistically significant (p=0.072). Additionally, the number of blinks per minute did not differ significantly between the groups (p=0.923).

**TABLE 1:** Dry eye test measurement results

| | Group 1 $\bar{X}$±SD (range) | Group 2 $\bar{X}$±SD (range) | p value |
|---|---|---|---|
| Schirmer-I (mm) | 22.71±11.41 (2-35) | 19.58±10.32 (5-35) | 0.201 |
| Oxford score | 0.37±0.42 (0-1) | 0.24±0.41 (0-1) | 0.457 |
| OSDI score | 23.12±13.62 (4.16-45) | 22.01±13.32 (2.08-40) | 0.857 |
| NIBUT (sn) | 14.12±10.73 | 13.23±9.25 | 0.773 |
| Tear meniscus (mm) | 0.21±0.13 (0.09-0.63) | 0.25±0.11 (0.10-0.46) | 0.072 |
| Blink (n/min) | 17.32±14.43 (2-48) | 16.93±11.43 (3-38) | 0.923 |

SD: Standard deviation; OSDI: Ocular Surface Disease Index;
NIBUT: Non-invasive tear break-up time

**TABLE 2:** MQS scoring and meiboscore results

| | Group 1 $\bar{X}$±SD (range) | Group 2 $\bar{X}$±SD (range) | p value |
|---|---|---|---|
| MQS | 1.78±0.43 (1-2) | 2.43±0.48 (2-3) | 0.001* |
| Upper meiboscore | 0.38±0.48 (0-1) | 0.28±0.46 (0-1) | 0.532 |
| Lower meiboscore | 0.38±0.71 (0-2) | 0.25±0.53 (0-1) | 0.703 |
| Total meiboscore | 0.78±0.67 (0-2) | 0.56±0.72 (0-2) | 0.343 |

*Statistically significant (p<0.05); MQS: Meibum quality score; SD: Standard deviation

Table 2 summarizes the meibography results. Comparison of meiboscores between the groups revealed similar upper, lower, and total meiboscores (p=0.532, p=0.703, and p=0.343, respectively). However, the MGD score of Group 1 was significantly lower compared to Group 2 (p=0.001).

## DISCUSSION

Female androgenetic alopecia (FAA) is a type of non-scarring hair loss with a complex etiology that predominantly affects postmenopausal women.[12] The onset of FAA typically begins during the reproductive years, with a peak incidence observed during the menopausal period, generally occurring between the ages of 50 and 60.[12] Unlike male pattern baldness, which is well-understood to be influenced by androgens, the role of androgens in FAA remains uncertain. Surprisingly, low levels of androgens do not consistently correlate with FAA, leading to the adoption of the term female pattern hair loss (FPHL) for this condition in women.[13] This change reflects the unclear relationship between androgens and FPHL, as serum androgen levels may not be elevated in all cases of this condition.[14]

Sex steroids exert a complex influence on the tear film and meibomian glands. Current understanding suggests that low circulating androgen levels and high estrogen levels are generally recognized as risk factors for dry eye, either independently or in combination.[15] Meibomian glands have been found to include sex steroid receptors for both estrogen and androgens, which may have an impact on meibum production.[16]

Whereas estrogens result in a reduction in lipid production, androgens promote lipid synthesis and secretion from meibomian glands. Both a reduction in the quality of meibum secretion and alterations in the meibomian gland appearance have been noted in these patients.[17] Elevated blood estradiol levels are thought to raise the risk of dry eye by preventing the formation of lipids and encouraging their breakdown in the meibomian glands, even though the actions of estrogens on the ocular surface of the meibomian glands are less well-established.[4,16] In a study of women with premature ovarian failure aged 17-43, dry eye symptoms were more common than in an age-matched control group, indicating a positive influence of ovarian estrogen.[18] Three instances of an increased incidence of dry eye in postmenopausal women undergoing aromatase inhibitor therapy for breast cancer underscore the importance of peripheral estrogen production in preserving ocular surface homeostasis.[19] Dry eye is more common among postmenopausal women.[20] Lower levels of estrogen and testosterone are among the hormones that change as a result of menopause.[21] Therefore, these alterations in the levels of circulating sex steroids might be responsible for postmenopausal women's dry eye.[22] In addition, the duration of menopause and hormone replacement therapy have been shown to increase the incidence of dry eye.[23-25]

In a study conducted by Kiyat et al., dry eye tests and meibography results were compared between women with FPHL and age-matched healthy women. The study found that tear film breakup time, a measure of tear film stability, was significantly lower in the FPHL group, whereas the OSDI score, indicating ocular surface disease symptoms, was significantly higher. However, there were no significant differences between the groups in Schirmer-I test and Oxford scores. Subgroup analysis based on menopausal status (postmenopausal vs. non-menopausal) showed similar results in both groups.[3] In contrast, our study did not find significant differences in dry eye tests between FPHL patients and healthy subjects. Several factors may contribute to this disparity. Firstly, all participants in our study were in the non-menopausal period, potentially influencing tear production and inflammatory responses differently than in the follicular phase of the menstrual cycle. Subjective discomfort symptoms are notably associated with

hormonal fluctuations during the menstrual cycle.[26] Another contributing factor could be the significant impact of androgens on the meibomian gland structure, where they stimulate lipid synthesis and secretion.[16,27] Furthermore, androgens are known to enhance lacrimal gland function by boosting the production of transforming growth factor beta, which is linked to a decrease in interleukin-1b and tumor necrosis factor alpha levels. This has an anti-inflammatory effect.[28]

Androgens have been identified as key regulators of lipid synthesis in meibomian glands, enhancing the quality and quantity of lipid secretion, which is essential for tear film stability.[4] On the other hand, lipid production may be inhibited by estrogens, which could result in evaporative dry eye and unstable tear films. When compared to controls, meibomian glands in patients undergoing anti-androgen therapy show notable alterations, according to several studies. Orifice metaplasia, poorer secretion quality, a noticeable alteration in the meibum's neutral lipid balance, and a morphological appearance typical of severe illness are some of these alterations.[17,29]

Both men and women see a considerable decline in meibum quality as they age, and meibomian gland orifice metaplasia is far more common.[30,31] The polar and neutral lipid compositions of meibomian gland secretions also undergo significant alterations with aging.[30,31] These findings result from the sharp drop in androgen levels in both sexes.[32]

In a study comparing male AGA patients with healthy volunteers, statistically significant dry eye findings and MGD were observed in anrogenetic alopecia patients.[33] Dry eye and MGD are also significantly observed in patients with sex steroid imbalance such as polycystic ovary syndrome and post-menopausal women.

In the study by Kiyat et al., meiboscores were significantly higher in FPHL patients compared to healthy controls.[3] However, our study yielded different findings, with both groups showing similar meiboscores. The FPHL group in our study demonstrated significantly better MQS, potentially linked to differences in serum androgen levels between the groups. This finding may reflect the stimulatory effects of androgens on meibomian gland function, consistent with Mantelli et al., who highlighted the critical role of androgen imbalance in ocular surface pathophysiology.[5] Another possible scenario is androgen receptor upregulation. When there is an increased sensitivity to androgens in one area of the body or an imbalance in systemic androgen levels, other tissues may adapt to this situation in different ways. Cells in the meibomian glands may increase the number or sensitivity of androgen receptors as a compensatory mechanism to continue to benefit from the positive effects of androgens.[34] Finally, the meibomian glands have the capacity to synthesize and activate their own androgens locally. This allows the gland to function according to the needs of its microenvironment, partially independent of systemic circulating hormone levels. Increased local synthesis of androgens may also be one explanation.[35] However, the absence of serum hormone level measurements in our study limits the ability to establish a direct link between androgen levels and the observed differences.

However, our study has several limitations. These include a relatively small sample size, the potential influence of menstrual cycle variations among participants, and the absence of periodic measurements of serum hormone levels, which could fluctuate over time. In addition, not knowing the duration of the disease was one of the limitations.

## CONCLUSION

In conclusion, the dry eye parameters observed in FAA patients were similar to those in healthy volunteers, but meibum quality was found to be better. Future research should include larger cohorts and diverse populations, including postmenopausal women, to elucidate the complex interactions between hormonal changes and ocular health. This approach will provide a more comprehensive understanding of the systemic implications of FPHL and its potential role in ocular surface disorders.

***Source of Finance***

*During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that pro-*

*vides or produces medical instruments and materials which may negatively affect the evaluation process of this study.*

### *Conflict of Interest*

*No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.*

### *Authorship Contributions*

***Idea/Concept:*** *Sezer Hacıağaoğlu, Ece Altun;* ***Design:*** *Sezer Hacıağaoğlu, Fahri Onur Aydın;* ***Control/Supervision:*** *Sezer Hacıağaoğlu, Fahri Onur Aydın, Ece Altun;* ***Data Collection and/or Processing:*** *Sezer Hacıağaoğlu, Ece Altun;* ***Analysis and/or Interpretation:*** *Fahri Onur Aydın, Sezer Hacıağaoğlu;* ***Literature Review:*** *Sezer Hacıağaoğlu, Fahri Onur Aydın, Ece Altun;* ***Writing the Article:*** *Sezer Hacıağaoğlu, Fahri Onur Aydın, Ece Altun;* ***Critical Review:*** *Fahri Onur Aydın.*



## REFERENCES


1. Olsen EA. Current and novel methods for assessing efficacy of hair growth promoters in pattern hair loss. J Am Acad Dermatol. 2003;48(2):253-62. PMID: 12582397
2. Nestor MS, Ablon G, Gade A, Han H, Fischer DL. Treatment options for androgenetic alopecia: efficacy, side effects, compliance, financial considerations, and ethics. J Cosmet Dermatol. 2021;20(12):3759-81. PMID: 34741573; PMCID: PMC9298335
3. Kiyat P, Palamar M, Gerceker Turk B, Yagci A. Evaluation of dry eye and Meibomian gland dysfunction in female androgenetic alopecia patients. Int Ophthalmol. 2021;41(11):3749-57. PMID: 34241758
4. Versura P, Giannaccare G, Campos EC. Sex-steroid imbalance in females and dry eye. Curr Eye Res. 2015;40(2):162-75. PMID: 25290221
5. Mantelli F, Moretti C, Macchi I, Massaro-Giordano G, Cozzupoli GM, Lambiase A, et al. Effects of sex hormones on ocular surface epithelia: lessons learned from polycystic ovary syndrome. J Cell Physiol. 2016;231(5):971-5. PMID: 26491950
6. Liu S, Richards SM, Lo K, Hatton M, Fay A, Sullivan DA. Changes in gene expression in human meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2011;52(5):2727-40. PMID: 21372006; PMCID: PMC3088560
7. Sullivan DA, Jensen RV, Suzuki T, Richards SM. Do sex steroids exert sex-specific and/or opposite effects on gene expression in lacrimal and meibomian glands? Mol Vis. 2009;15:1553-72. PMID: 19693291; PMCID: PMC2728565
8. Gorimanipalli B, Khamar P, Sethu S, Shetty R. Hormones and dry eye disease. Indian J Ophthalmol. 2023;71(4):1276-84. PMID: 37026259; PMCID: PMC10276676
9. Truong S, Cole N, Stapleton F, Golebiowski B. Sex hormones and the dry eye. Clin Exp Optom. 2014;97(4):324-36. PMID: 24689906
10. Arita R, Itoh K, Inoue K, Amano S. Noncontact infrared meibography to document age-related changes of the meibomian glands in a normal population. Ophthalmology. 2008;115(5):911-5. PMID: 18452765
11. Gutgesell VJ, Stern GA, Hood CI. Histopathology of meibomian gland dysfunction. Am J Ophthalmol. 1982;94(3):383-7. PMID: 7124880
12. Ramos PM, Miot HA. Female pattern hair loss: a clinical and pathophysiological review. An Bras Dermatol. 2015;90(4):529-43. PMID: 26375223; PMCID: PMC4560543
13. Olsen EA. Female pattern hair loss. J Am Acad Dermatol. 2001;45(3 Suppl):S70-80. PMID: 11511856
14. Fabbrocini G, Cantelli M, Masarà A, Annunziata MC, Marasca C, Cacciapuoti S. Female pattern hair loss: a clinical, pathophysiologic, and therapeutic review. Int J Womens Dermatol. 2018;4(4):203-11. PMID: 30627618; PMCID: PMC6322157
15. Ablamowicz AF, Nichols JJ, Nichols KK. Association between serum levels of testosterone and estradiol with meibomian gland assessments in postmenopausal women. Invest Ophthalmol Vis Sci. 2016;57(2):295-300. PMID: 26830366; PMCID: PMC4736742
16. Suzuki T, Schirra F, Richards SM, Jensen RV, Sullivan DA. Estrogen and progesterone control of gene expression in the mouse meibomian gland. Invest Ophthalmol Vis Sci. 2008;49(5):1797-808. PMID: 18436814
17. Krenzer KL, Dana MR, Ullman MD, Cermak JM, Tolls DB, Evans JE, et al. Effect of androgen deficiency on the human meibomian gland and ocular surface. J Clin Endocrinol Metab. 2000;85(12):4874-82. PMID: 11134156
18. Smith JA, Vitale S, Reed GF, Grieshaber SA, Goodman LA, Vanderhoof VH, et al. Dry eye signs and symptoms in women with premature ovarian failure. Arch Ophthalmol. 2004;122(2):151-6. PMID: 14769589
19. Cuzick J, Sestak I, Forbes JF, Dowsett M, Knox J, Cawthorn S, et al; IBIS-II investigators. Anastrozole for prevention of breast cancer in high-risk postmenopausal women (IBIS-II): an international, double-blind, randomised placebo-controlled trial. Lancet. 2014;383(9922):1041-8. Erratum in: Lancet. 2014;383(9922):1040. Erratum in: Lancet. 2017;389(10073):1010. PMID: 24333009
20. Schaumberg DA, Sullivan DA, Buring JE, Dana MR. Prevalence of dry eye syndrome among US women. Am J Ophthalmol. 2003;136(2):318-26. PMID: 12888056
21. Versura P, Campos EC. Menopause and dry eye. A possible relationship. Gynecol Endocrinol. 2005;20(5):289-98. PMID: 16019376
22. Turgut FG, Turgut A, Dolgun ZN, Köroğlu N. Effects of menopause on corneal topography and dry eye. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2017;6(2):461-5. https://www.ijrcog.org/index.php/ijrcog/article/view/1558
23. Erdem U, Ozdegirmenci O, Sobaci E, Sobaci G, Göktolga U, Dagli S. Dry eye in post-menopausal women using hormone replacement therapy. Maturitas. 2007;56(3):257-62. PMID: 17030104
24. Schaumberg DA, Buring JE, Sullivan DA, Dana MR. Hormone replacement therapy and dry eye syndrome. JAMA. 2001;286(17):2114-9. PMID: 11694152
25. AlAwlaqi A, Hammadeh M. Examining the relationship between hormone therapy and dry-eye syndrome in postmenopausal women: a cross-sectional comparison study. Menopause. 2016;23(5):550-5. PMID: 27003590
26. Versura P, Fresina M, Campos EC. Ocular surface changes over the menstrual cycle in women with and without dry eye. Gynecol Endocrinol. 2007;23(7):385-90. PMID: 17701769
27. Schirra F, Richards SM, Liu M, Suzuki T, Yamagami H, Sullivan DA. Androgen regulation of lipogenic pathways in the mouse meibomian gland. Exp Eye Res. 2006;83(2):291-6. PMID: 16579987

28. Beauregard C, Brandt P. Down regulation of interleukin-1beta-induced nitric oxide production in lacrimal gland acinar cells by sex steroids. Curr Eye Res. 2004;29(1):59-66. PMID: 15370368

29. Sullivan BD, Evans JE, Krenzer KL, Reza Dana M, Sullivan DA. Impact of antiandrogen treatment on the fatty acid profile of neutral lipids in human meibomian gland secretions. J Clin Endocrinol Metab. 2000;85(12):4866-73. PMID: 11134155

30. Sullivan BD, Evans JE, Dana MR, Sullivan DA. Influence of aging on the polar and neutral lipid profiles in human meibomian gland secretions. Arch Ophthalmol. 2006;124(9):1286-92. PMID: 16966624

31. Sullivan BD, Evans JE, Dana MR, Sullivan DA. Impact of androgen deficiency on the lipid profiles in human meibomian gland secretions. Adv Exp Med Biol. 2002;506(Pt A):449-58. PMID: 12613945

32. Labrie F, Bélanger A, Cusan L, Gomez JL, Candas B. Marked decline in serum concentrations of adrenal C19 sex steroid precursors and conjugated androgen metabolites during aging. J Clin Endocrinol Metab. 1997;82(8):2396-402. PMID: 9253307

33. Koc HA, Koc H. Evaluation of dry eye parameters and meibomian gland function using the Lipiscan system in male androgenetic alopecia patients. Clin Exp Optom. 2025:1-7. PMID: 39864098

34. Wang LX, Deng YP. Androgen and meibomian gland dysfunction: from basic molecular biology to clinical applications. Int J Ophthalmol. 2021;14(6):915-22. PMID: 34150548; PMCID: PMC8165631

35. Khandelwal P, Liu S, Sullivan DA. Androgen regulation of gene expression in human meibomian gland and conjunctival epithelial cells. Mol Vis. 2012;18:1055-67. PMID: 22605918; PMCID: PMC3351406