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PERSONALISED DRY EYE TREATMENT: THE USE OF GENETIC TESTING TO TAILOR TREATMENT PLANS TO INDIVIDUAL PATIENTS

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Abstract: **Dry eye disease is a chronic condition affecting millions of people worldwide, causing discomfort, visual impairment, and decreased quality of life. This research was designed as a clinical and laboratory based study for investigating the use of genetic testing to tailor treatment plans to individual patients. One hundred and twenty subjects, 58 (48.3%) males and 62 (51.7%) females with dry eye disease between the ages of 18-65 years were enrolled in the study. Tear assessment, Tear Break Up Time test (TBUT) using fluoresciene dye and tear flow rate test using schirmer strip without anesthesia were done clinically to confirm Dry Eye Disease (DED), tear film samples were collected using sterile swab and genetic investigations were carried out in the molecular laboratory to detect the enrichment of variants responsible for Dry Eye Disease (DED) and Ocular Surface Inflammation (OSI); DNAJB6 was detected in 59 subjects and absent in 61 subjects, MAML3 was detected in 47 and absent in 73 subjects, LINC02267 was detected in 13 subjects and absent in 107 subjects, MUC16 was detected in 14 subjects and absent in 106 subjects, SIRPB3P was detected in 48 subjects and absent in 72 subjects, GAS2L3 was detected in 13 subjects and absent in 107 subjects, KLF4 was detected in 81 subjects and absent in 89 subjects, and PAX6 was detected in 28 subjects and absent in 92 subjects. LINCO2267 and GAS2L3 were the least detected among genetic variants for OSI and DED respectively while KLF4 was the most detected among genetic variants for OSI and DED. The significant representation of DNAJB6, MAML3, LINC02267, and MUC16 variants shows molecular relationship between DED and OSI. From SPSS version 23 data output, data analysis using the Pearson Product Moment Correlation Coefficient at 0.05 level of significance revealed for DED genetic variants a p-value of 0.00. Since p(0.00) <0.05, there is an association between genetic variants and dry eye disease. For ocular surface inflammation, SPSS version 23 data output, data analysis using the Pearson Product Moment Correlation Coefficient at 0.05 level of significance revealed a p-value of 0.00. Since p(0.00) < 0.05, there is an association between genetic variants and ocular surface inflammation. In conclusion, the results of the study showed that dry eye disease and ocular surface inflammation were strongly influenced by genetic variation in epithelial and inflammatory pathways. The results also showed that the initiation and progression of dry eye were multifactorial. It is therefore recommended that eye care practitioners consider genetic testing as an alternative approach for the diagnosis and treatment of dry eye disease. Incorporating genetic testing alongside regular eye examinations can help eye care practitioners better understand each patient's condition.**

Keywords: **Discomfort, impairment, assessment, investigations, enrichment, inflammation, multifactorial.**

1. INTRODUCTION

Dry eyes, also known as dry eye syndrome (DES), dry eye disease (DED), ocular surface disease (OSD), dysfunctional tear syndrome (DTS), and keratoconjunctivitis sicca (KCS), are among the most common reasons for a visit to an eye doctor (Huang et al., 2022). The definition of a dry eye according to the Tear Film and Ocular Surface Society (TFOS) Dry Eye Workshop II (DEWS II) is, Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis

of the tear film and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and neurosensory abnormalities play etiologic roles (Craig et al., 2017).

Genetics is the study of heredity, and seeks to explain the mechanism of hereditary transmission, as well as the genetic basis of individual variation. Medical genetics is the science of genetically associated biologic variation relevant to human traits and diseases. In industrialized countries, improvements in public health and medical care have resulted in a marked reduction in mortality from infectious causes, or nutritional deficiencies; during this time, there has been a corresponding increase in awareness of the role of genetic factors in human diseases, including neurological disease (Armstrong, Shipkova, Von-Ahsen, & Oellerich, 2004).

Dry eye syndrome (DES), also referred to as dry eye disease (DED) or keratoconjunctivitis sicca (KCS), encompasses multifactorial ocular surface pathology causing discomfort and visual disturbances. Understanding the complexity of tear film composition and dysfunction is pivotal in assessing subjects presenting with dry eyes. This activity delves into the tear film's structure, comprising lipid, aqueous, and mucin layers, impacting tear stability and ocular surface health. It navigates the nomenclature nuances, differentiating between DES and DED, acknowledging their broader clinical implications and comprehensive categorizations (Chen et al., 2010).

Potential causes or factors associated with Dry Eye

Systemic medications: Antihistamines, Antihypertensive, Anxiolytics/benzodiazepines, Diuretics, Systemic hormones, Nonsteroidal antiinflammatory drugs, Systemic or inhaled corticosteroids, Anticholinergic medications, Isotretinoin (causes meibomian gland atrophy), Antidepressants (Paulsen et al., 2024).

Topical medications: Including glaucoma drops or preservative toxicity from eye drops containing preservatives (Andole & Senthil, 2023).

Skin diseases: On or around the eyelids, such as rosacea or eczema (Sobolewska, Schaller, & Zierhut, 2022).

Meibomian gland dysfunction: A common comorbidity with thickening and erythema of the eyelids and inadequate or altered secretions of meibomian glands (Bilgic et al., 2023).

Ophthalmic surgery: Refractive surgery, Cataract surgery, Keratoplasty, Lid surgery (Sabti Zechevikj & Raizada, 2022)

Chemical or thermal burns: Those that scar the conjunctiva (Napoli et al., 2019)

Ocular allergies and decreased androgen levels: Occurring with menopause and other conditions.

Computer or device usage: This may lead to decreased blinking when looking at the screen.

Excess or insufficient dosages of vitamins: Particularly vitamin A deficiency; can lead to xerophthalmia and the appearance of Bitot spots on the conjunctiva in severe cases.

Decreased sensation in the cornea: Due to: Long-term contact lens wear, Herpes virus infections.

Other causes of a neurotrophic cornea (Altinbas, Elibol, Fıratlı, Ayhan & Celebi, 2023)

Graft-versus-host disease, Autoimmune systemic diseases: Sjogren syndrome, Graves' ophthalmopathy.

Connective tissue disorders: Rheumatoid arthritis, Lupus, Thyroid disease (Choudhry et al., 2022)

Systemic conditions: Diabetes, Xerophthalmia, Hereditary disease, Gut dysbiosis, Multiple sclerosis and Nerve damage pathologies (García-Marqués Talens-Estarelles, C., García-Lázar, Wolffsohn & Cerviño, 2022)

Environmental factors: Including exposure to irritants such as: Chemical fumes, Cigarette smoke, Strong winds, High temperature, Pollution, High altitude and Low humidity (Tandon et al., 2020)

Behavioral habits: Smoking, Alcohol, Poor sleep, Unhealthy diet, Dislipidemia (Choi, Talens-Estarelles, García-Lázaro, S., Wolffsohn & Cerviño, 2020).

2. RESULTS

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Gender Distribution of Subjects

Table 1 below shows the gender distribution of subjects used in the research. A total number of 120 Dry eye disease subjects participated in the research. There were 58 (48.3%) males and 62 (51.7%) females clinically diagnosed with Dry eye disease.

Table 1: Showing Gender Distribution of Subjects

	Frequency (n)	Percentage (%)
Male	58	48.3
Female	62	51.7
Total	120	100

n=number of subjects, %=percentage of subjects

Age Distribution of Dry Eye Disease Subjects

Table 2 below shows the age distribution of subjects that were used in the research. Subjects between 50-65 (36.7%) years of age were higher in number than other subjects. The least number of subjects fall within 18-29 (5%).

Table 2: Showing Age Distribution of Dry Eye Disease Subjects

Age group	Frequency (n)	Percentage (%)
18-29	6	5
30-39	36	30
40-49	34	28.3
50-65	44	36.7
Total	120	100

n=number of subjects, %=percentage of subjects

Clinical Diagnosis of Dry Eye Disease (DED Clinical) and Ocular Surface Inflammation (OSI Clinical)

Table 3 below shows clinical diagnosis of Dry eye disease and Ocular surface inflammation among subjects. A total number of 120 (100%) subjects were diagnosed with Dry eye disease, 72 (60%) were clinically diagnosed with ocular surface inflammation and 48 (40%) were clinically diagnosed of no ocular surface inflammation.

Table 3: Showing Clinical Diagnosis of Dry Eye Disease (DED Clinical) and Ocular Surface Inflammation (OSI Clinical)

Condition	Present n (%)	Absent n (%)
Dry eye disease (clinical)	120 (100)	0 (0)
Ocular surface inflammation (clinical)	72 (60)	48 (40)

n=number of subjects, %=percentage of subjects

Genetic Diagnosis of Dry Eye Disease (DED Genetic) and Ocular Surface Inflammation (OSI Genetic)

Table 4 below shows genetic diagnosis of Dry eye disease and Ocular surface inflammation among subjects. A total number of 120 (100%) subjects were diagnosed with Dry eye disease, 72 (60%) were genetically diagnosed with ocular surface inflammation and 48 (40%) were clinically diagnosed of no ocular surface inflammation.

Table 4: Showing Genetic Diagnosis of Dry Eye Disease (DED Genetic) and Ocular Surface Inflammation (OSI Genetic)

Condition	Present n (%)	Absent n (%)
Dry eye disease (genetic)	120 (100)	0 (0)
Ocular surface inflammation (genetic)	72 (60)	48 (40)

n=number of subjects, %=percentage of subjects

Comparison between DED (Clinical) and DED (Genetic)

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Table 5 below shows the comparison between DED (clinical) and DED (genetic). All 120 (0%) subjects clinically diagnosed with Dry eye disease were also genetically diagnosed with Dry eye disease.

Table 5: Showing Comparison between DED (Clinical) and DED (Genetic)

Condition	Present n (%)	Absent n (%)
Dry eye disease (clinical)	120 (100)	0 (0)
Dry eye disease (genetic)	120 (100)	0 (0)

n=number of subjects, %=percentage of subjects

Comparison between OSI (Clinical) and OSI (Genetic)

The table 6 below shows the comparison between OSI (clinical) and OSI (genetic). All 72 (60) subjects clinically diagnosed with Ocular surface inflammation were also genetically diagnosed with Ocular surface inflammation while 48 subjects clinically diagnosed with no Ocular surface inflammation were genetically diagnosed with no Ocular surface inflammation.

Table 6: Showing Comparison between OSI (Clinical) and OSI (Genetic)

Condition (Genetic)	Present n (%)	Absent n (%)
Ocular surface inflammation (clinical)	72 (60)	48 (40)
Ocular surface inflammation (genetic)	72 (60)	48 (40)

n=number of subjects, %=percentage of subjects

Frequency of Detected Genetic Variants

Figure 1 below shows the frequency of detected genetic variants among DED subjects. KLF4 genetic variant was present in 81 (67.5%) subjects and absent in 39 (32.5%) subjects making it the highest genetic variant found in DED subjects. LINCCO2267 and GAS2L3 genetic variants were equally present in 13 (10.8%) subjects and absent in 107 (89.2%) subjects making them the least genetic variant present among the DED subjects.

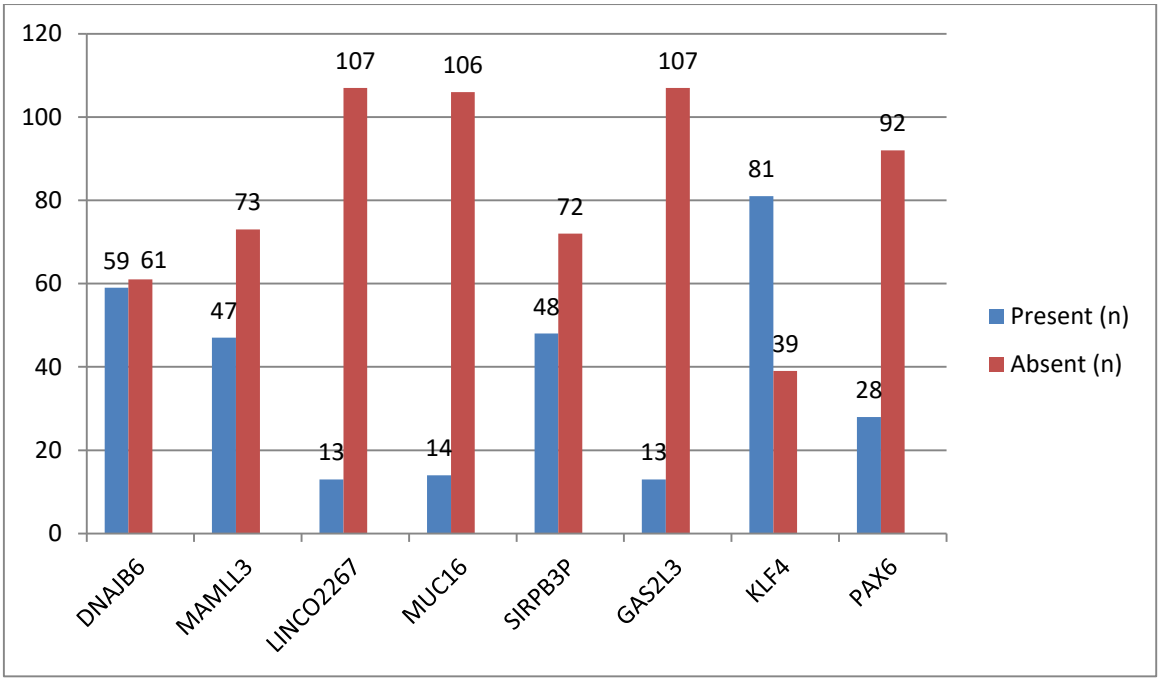

Fig. 1: Showing Frequency of Detected Genetic Variants

3. CONCLUSION

In conclusion, the results of the study showed that dry eye disease and ocular surface inflammation were strongly influenced by genetic variation in epithelial and inflammatory pathways. The results also showed that the initiation and progression of dry eye were multifactorial. These findings provide a molecular foundation for personalised dry eye treatment.

Consequently, integrating genetic testing into clinical practice offers the potential to improve diagnostic accuracy, facilitate early identification of at-risk individuals, and support the selection of targeted treatment strategies that are more effective.

The findings further demonstrate that personalised approach has the potential to transform the management of dry eye disease by improving treatment outcomes, reducing unnecessary therapeutic interventions, and enhancing patients' quality of life.

Although genetic testing has not yet become a standard component of dry eye management, this work suggests that it holds considerable promise for advancing personalised patient care. As research, technology and accessibility continue to improve; the integration of genetic information into clinical decision-making in the management of dry eye disease is will become an important aspect of future dry eye management.

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