Metal Ions in Life Sciences

Dermatophyte Infections and the Role of Host Genetic Susceptibility in Disease Development: A Translational Mycogenetic Perspective

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Abstract

Because dermatophytes and their hosts are so different, infection susceptibility is most likely the consequence of changes on both sides, as well as reciprocal adaptation. In addition, some studies have revealed a role for host genetics in the development of illness, including possible Mendelian inheritance patterns for dermatophytosis tendency. This paper emphasizes the complexity of the genetic link between dermatophytes and their natural and accidental hosts. According to a literature study, different ideas and methodologies may lead to different interpretations of this connection. Selecting an appropriate model for analysis and reasoning is a critical step in better understanding these disorders. A significant portion of the research is focused on the host's genetic and immunological response to dermatophyte infection. Future studies will require a broader exploration of the dermatophyte genome in combination with analysis of large phenotypically well-characterized populations of various dermatophyte species in order to identify the main factors mediating infection risk that can be targeted to disrupt host–pathogen interactions and used in therapies. As a result, both conceptually and practically, extensive study on the interactions between dermatophytes and their specific hosts, which comprise intricate molecular pathways, is critical. However, it is undeniable that genetic predisposition plays a crucial role in the susceptibility to dermatophyte infection.

Introduction

Dermatophytosis is a common illness that affects 20–25 percent of the human population every year (1,2,3). Dermatophytes, filamentous fungi with a strong affinity for keratinized tissues such as skin, hair, and nails, are the main etiological agents of this illness (1, 4,5,6). Trichophyton, Microsporum, and Epidermophyton were previously designated as the causal agents of dermatophytosis in the order Onygenales (7). Molecular phylogenetic techniques, on the other hand, have changed dermatophyte taxonomy, proving that Trichophyton is a polyphyletic taxon and allowing for the introduction of new taxa such as Nannizzia, Arthroderma, Paraphyton, Lophophyton, Ctenomyces, and Guarromyces (8).

Over the last two decades, the global incidence of dermatophytosis has increased dramatically. Socioeconomic issues, large-scale worldwide travel, immigration from tropical countries, climate change, and regular interaction with animals, particularly pets, are all contributing factors (9,10,11,12). As a result of prolonged life and the unavoidable use of immunosuppressive medicines by many patients, dermatophytosis-induced morbidity has increased in humans (13,14,15). Furthermore, because the majority of dermatophytes are zoonotic, intimate contact with pets increases the risk of infection. As a result, it appears that only an interdisciplinary strategy comprising dermatologists, pediatricians, primary care physicians, mycologists, and veterinarians can assist to control the spread of dermatophytoses in today's world (16). The significance of genetic variables in the tendency to dermatophytosis and the possible inheritance of these tendencies is a fascinating topic.

We explain current results concerning the mechanism of dermatophyte infections in this review, with an emphasis on the disease's hereditary propensity in humans. The importance of heredity in families with a high incidence of dermatophyte infections, as well as unique host–pathogen interactions, are underlined in particular.

Determinants of Dermatophytosis Prevalence: Genetic Predisposition

Interestingly, data from several observational studies indicate that dermatophytes infect people of all ages, races, genders, and socioeconomic statuses at alarmingly high rates (17,18,19).The incidence of superficial fungal infections, however, varies greatly. The factors that influence dermatophyte infection susceptibility and frequency may be separated into three categories: (1) environmental factors that affect both the host and the pathogen, (2) host-specific factors, and (3) dermatophyte species and ecological group factors (1, 10, 20, 21). Climate conditions, such as humidity and temperature, are included in the first category (1, 5, 20,21,22,23). The host is largely dependent on genetic susceptibility to infectious diseases, as well as other factors such as age, sex, maceration of the epidermis, mechanical skin lesions, impairment of immunological barriers, and possible interactions with dermatophytes and their spores associated with socioeconomic status and profession (18, 23,24,25,26,27,28). Finally, the ecological niche filled by the fungus has a substantial influence on the course of infection (23, 24, 29,30,31).

The symptoms of dermatophyte infection are not limited to chronic or acute superficial lesions, but can range from individuals who are never infected to those who have inflammatory, non-inflammatory, or treatment-resistant symptoms to cases of invasive, disseminated, and life-threatening disease (14, 17, 32,33,34,35,36,37,38).

The acquisition of dermatophyte infectious components by the host's stratum corneum is not synonymous with the emergence of infection symptoms, according to conclusive data from observational studies (10, 39, 40). Furthermore, despite the harmful character of the dermatophytes, the frequency of cutaneous fungal infections is not as great as one might assume (1, 2, 23, 41, 42). Second, the relatively high incidence of dermatophytoses in specific communities or families may be a key indicator of genetic sensitivity to these fungi. In this context, an obvious issue arises: whatever genetic characteristics in the human/animal host cause some people to be immune to illness symptoms and even stay asymptomatic carriers, while others acquire severe dermatophytosis that, at worst, can be invasive and resistant to treatment?

Pathogen Virulence or Host Genetic Predisposition

The pathophysiology of infectious illnesses has been studied for about seven decades to determine if pedigrees, polymorphism, and other genetic alterations, particularly the genetics of immunity, underpin dermatophyte susceptibility or offer protection against these infections (10, 17, 19). Although the nature and severity of dermatophyte infections, their relapses despite treatment, and the receptivity of the host physicochemical barrier to the pathogen are all influenced by the host's genetics (43), the pathogenic potential of highly ecologically diverse dermatophytes is equally important in the disease's development (37, 44, 45). The presence of animals in the home is associated with the largest number of dermatophytosis cases, and their occurrence is increasing with the growing popularity of pets globally (9, 11, 26, 46). As a result, dermatophytes found in soil have a lower virulence than animal-related infections (47, 48), and many researchers and clinicians perceive geophilic dermatophytes as opportunistic pathogens (49, 50). Anthropophilic dermatophytes, which have evolved to thrive on keratinophilic substances of human origin, are at the heart of these harmful relationships, and their high transmission is confined to humans alone (10, 51, 52). However, even within the same ecological group, dermatophyte strains exhibit significant genomic and phenotypic variability, which is due to the profile of secreted exoenzymes influencing host response (5, 29, 37, 44, 53, 54). Furthermore, because of the diversity in enzyme profiles and activity, only a few or none of them can be sufficiently pathogenic to induce clinical illness (5, 55,56,57).

Interestingly, multiple infection patterns may be seen in the same genetic dermatophyte strain (26, 29, 35, 46, 58). Different clinical presentations of dermatophytosis have also been discovered in cognate persons living or working together in several circumstances (41, 59, 60). The type and degree of the interaction established with dermatophytes after exposure seems to be influenced by the host. The choice of a research approach is critical in the study of genetic predispositions to dermatophyte infections. Initially, variations in the prevalence of symptomatic infection between genetically related family members and spouses were used to infer the genetic basis of susceptibility to dermatophytes (61,62,63,64). Many scientists, however, saw this research as dubious, arguing that the variations detected based on medical history and mycological examination could not be meaningful. Because of the high frequency of daily routine interaction in the shared living environment, which increases infection rates, research among family members is unreliable. Humans who are not related by blood but have a shared working environment or daily activities, such as in the army, school, or hospital, have a comparable incidence of dermatophytoses (41, 42, 64, 65, 66, 67, 68, 69, 70). Abdel-Rahman et al. (58) suggested in their groundbreaking study that cross-sectional sampling strategies used in most epidemiologic studies are insufficient for describing the natural course of infection and fail to identify individuals who develop active disease; thus, a different strategy for testing infection predisposition should be used. In a two-year prospective longitudinal research (58), the authors analyzed preschool-aged children who attended the same child care center. Then, molecular strain typing was used to distinguish between those who had never been infected with the pathogen, those who had acquired and lost multiple fungal strain types on a regular basis, and those who had acquired and sustained infection with the same strain type for years. A total of 3541 scalp cultures were obtained from 446 youngsters for this investigation. Each month, 22% to 51% of the scalp cultures were positive, resulting in 1390 fungal cultures and 1048 typeable cultures. In children with multiple typeable isolates, 51% had the same strain exclusively, 37% had a single predominant strain with secondary strains acquired transiently, and 12% had a distinct strain of T. tonsurans in each typeable culture. It was approximately 90 percent likely that the same strain would survive in later months, which was unlikely to have occurred by accident. Exclusive, predominant, and temporary T. tonsurans carriers had significantly varied rates of symptomatic illness. Unlike dermatophyte infections in older people, where symptomatic disease appears to be a result of pathogen acquisition and asymptomatic carriers can be linked to the index case, the infection in the preschool-age population was endemic, and symptomatic disease appeared to be triggered by a single strain that remained on the scalp.

Indeed, the form and degree of the interaction created with dermatophytes is influenced by the host's health, and the same genetic dermatophyte strain can cause distinct infection patterns in people sharing the same environment (2, 41, 58). Furthermore, various coexisting health disorders, such as eczema, psoriasis, ichthyosis, atopic dermatitis, and seborrheic dermatitis, may impact dermatophyte susceptibility (58, 71, 72, 73, 74, 75, 76). As a result, considering the genetic basis of susceptibility to dermatophyte infections without first determining the pathogenicity of the fungus does not appear to be supported by evidence. Finally, different strains of fungus with varying infective abilities may be involved, which, in combination with the host organism's genetic sensitivity, defines the sort of infection that occurs. As a result, the genetic predisposition of the host is just as significant as the pathogen's virulence and adaptability. Both aspects should be considered in clinical practice, and mycological tests should always be conducted and the findings correlated with the patient's state.

Susceptibility to Dermatophyte Infection Is Passed Down Through Generations

Regardless of the methodological flaws mentioned above, the study on the prevalence of dermatophyte infections in related persons raises questions. In the middle of the twentieth century, researchers came to conflicting findings on genetic tendencies in dermatophytoses. Genetically related family members, husbands/wives marrying into these households, and entirely unrelated persons living in the same environment all had different rates of dermatophyte infections (62, 63, 77,78,79). On the basis of questionnaires completed by over 100 dermatologists, Sulzberger et al. (80) examined the extent of family infections of the foot and groins. Their research found a relatively weak link between family members and the occurrence of dermatophytoses, with just four confirmed cases of familial infection among hundreds of thousands of patients evaluated; hence, such a high rate of infection was of no practical significance. The authors said that no familial infection could be established unless the fungus were isolated in culture and visually matched. The frequent interaction in a shared environment, which has been demonstrated to have an influence on infection rates in populations without genetic ties (66, 67, 78, 81), confused these associative investigations among family members. Skeptics are using the incidence of dermatophytoses in persons who live or work together as a criteria for further investigation into the genetic causes of vulnerability to these illnesses. Hopkins and Hillegas (78) observed that diverse species appeared at roughly the same proportions in most of the groups studied in their examination of fungal infections on the feet of soldiers at a military station. Individual vulnerability to a latent infection and immunity, rather than the possibility of cross-infection, were therefore prioritized. Nonetheless, the fact that three of the 26 groups evaluated in their study revealed significant variance in the major fungal species causing the illness, while others showed relatively little variation, raised questions about the correctness of the results. The inconsistencies in these results were due to the high turnover of personnel in military institutions, which did not always allow for enough time for any species to gain dominance (78).

Furthermore, contact sports have a high frequency of fungal skin infections, with tinea gladiatorum in wrestlers being a common example (70, 82). Dermatophytosis was shown to be more common among athletes under the age of 15 (82, 83). Despite continued wrestling activity, large levels of fungistatic effect of steroid hormones beyond puberty are likely to limit the occurrence of tinea gladiatorum in the older age range (83). Lewis and Lewis (64) identified healthcare staff in a rehabilitation center who had detectable physical contact and calculated that a quarter of them had a fungal infection. The significant contact group had a 33 percent infection rate and the moderate contact group had a 17 percent infection rate; the minimal contact group had no illnesses.

However, these historical data, together with examination of more refined pedigree results, give some support for a genetic link between dermatophyte infection susceptibility and genetics. Bonifaz et al. (84) came up with the first significant conclusion. The genetic predisposition to dermatophytosis was shown to be autosomal dominant in nine out of 16 family members, i.e., children with the same mother but different dads, in their investigation on confirmed instances of tinea imbricata caused by Trichophyton concentricum. Although Ravine et al. (85) reported autosomal recessive inheritance of tinea imbricata susceptibility in 1980, the authors firmly emphasize that the described family case clearly implies an autosomal dominant rather than recessive inheritance pattern of susceptibility. Furthermore, the genetic propensity to tinea imbricata was not entirely clarified by these contradicting data. According to Hay et al. (86), in addition to heritable vulnerability to this illness, heritable inadequate immune response to infection might also be a factor in the high recurrence rate and broad nature of clinical lesions seen in Tinea imbricata. Dey and Maplestone (87), Polunin (88), and Reid (89) found a significantly greater frequency of tinea imbricata in various races living in the same nation and under similar environmental conditions, indicating that racial traits play a role in disease susceptibility. The findings of racial disparities in infection rates might be the consequence of ethnically driven environmental variations between racial groups of persons living in close proximity.

Furthermore, Zaias et al. (90) discovered that the autosomal dominant pattern of inheritance was significant in increasing sensitivity in pedigrees of families in Italy with distal subungual onychomycosis and associated tinea pedis produced in foot soles by T. rubrum. In addition, infections in family members with inherited predisposition were equally common in both sexes. Families in France made similar discoveries about onychomycosis tendencies being passed down over the generations. Onychomycosis is intriguing in another way, in terms of genetic proclivities: in almost half of cases identified in children, parents were also afflicted, implying hereditary predispositions (19).

A breakdown in the immune response has also been hypothesized as a reason for variation in dermatophyte susceptibility in various studies. CARD9 (caspase recruitment domain-containing protein 9) deficiency due to compound heterozygous mutations is a differential diagnosis for severe, deep, recurring cutaneous fungal infections in individuals who are non-immunosuppressed or do not receive immunosuppressive medication (91,92,93). CARD9 is an adapter protein that regulates macrophage and neutrophil antifungal activity in the skin (91).

This impairment is also inherited as an autosomal recessive trait (94). CARD9 is also required for the activation of T-helper 17 (Th17) cells, which is mediated mostly by dectin-2, but also by dectin-1 signaling, macrophage-inducible C-type lectin, and maybe additional immune-related receptors (95,96,97). Glocker et al. (98) conducted a pedigree study on a consanguineous family with multiple members suffering from chronic fungal infections and a CARD9 autosomal recessive inheritance mechanism.Recurrent fungal infections were identified clinically in eight family members in their investigation, three of whom died as children. There were no uncommon bacterial or viral illnesses in any of these individuals, indicating that the host's resistance against these pathogens was normal. Furthermore, Nazarian et al. (93) discovered a tinea profunda case in a 31-year-old male caused by Trichophyton rubrum and Trichophyton violaceum and linked to biallelic mutations in CARD9. Deep dermatophytosis accounted for 37.3 percent of all documented instances of fungal infections connected to CARD9 deficiency owing to autosomal recessive mutations, according to Vaezi et al. (96). These dermatophytoses were found to be caused by Trichophyton violaceum, Trichophyton rubrum, and Trichophyton mentagrophytes (93, 99,100,101). Surprisingly, analyses of the characteristics, distribution, frequency, and relationship between the genotype of the CARD9 gene mutations and fungal infections in the reported cases revealed that dermatophytosis caused by this factor accounted for up to 75% of African cases (96), which is likely reflected in the high prevalence of T. violaceum isolation on this continent (48, 102). These findings show that mutations may be particular to particular populations or geographic locations where numerous closed groups have a high percentage of consanguinity.

The adaptive immune response to dermatophytosis has been extensively examined in geographically separated groups of patients (75, 103, 104, 105, 106, 107). In the course of fungal infection, the major histocompatibility complex (MHC) and the HLA (Human Leukocyte Antigen) system are thought to be crucial for antigen presentation and activation of T cell-mediated responses (75). Zaitz et al. (103) discovered HLA-DR4 in 100 percent of persons without symptoms and 25 percent of cases in a Brazilian Ashkenazi Jewish community with T. rubrum onychomycosis, showing a preventive effect against disease susceptibility. In turn, Garca-Romero et al. (104) discovered a greater frequency of HLA-DR8 in families with onychomycosis caused by the same dermatophyte species in a Mexican mestizo community, suggesting that this haplotype may confer vulnerability. Carrillo-Meléndrez et al. (75) also discovered a link between HLA-DR8 and the genetic predisposition to onychomycosis in nail psoriasis patients. Their research also demonstrated a link between HLA-DR1 and a genetic tendency to develop onychomycosis.

The incidence of specific disease entities linked with this group of fungus is significantly higher when the genetic factor is implicated, according to this retrospective investigation of the hereditary determinants of dermatophytoses susceptibility. Furthermore, the spatial component of these partnerships involving human races living in close proximity to one another is beginning to emerge. In addition, identifying high-risk households will allow family members to be educated about the dangers of fungal diseases. As a result, in practical practice, the information gathered during the interview can help to speed up the diagnosis and motivate the dermatologist to prescribe the proper treatment.

An overly broad approach to dermatophytosis as a whole, as well as the use of approaches based only on infection frequency analysis, on the other hand, is inaccurate and leads to incorrect conclusions about genetic predisposition.

Identification of Susceptibility-Related Genes

The assertion that genetic vulnerability to dermatophytosis is a monogenic trait can be just as misleading as the current tendency of emphasizing the importance of genetic determinants while ignoring other host, pathogen, or environmental variables. The search for genetic dependencies while neglecting non-inherited variables is one of the major shortcomings of investigations investigating genetic predictors of susceptibility to anthropophilic dermatophytes (17). As a result, finding genes that cause higher susceptibility to dermatophytosis is difficult, because the pathogen–host interaction should be addressed holistically (10, 28).

Abdel-Rahman and Preuett (108) used successful approaches for identifying genes involved in host–pathogen interactions and hence linked with greater susceptibility to dermatophyte infections. An exhaustive search for genes that may be connected to infection was carried out as part of a genome-wide association research in a cohort of children whose tinea capitis infection was tracked longitudinally over several years. The study included 20 children with T. tonsurans > 90% of the time and 20 children with the fungus less than 10% of the time. In general, the scientists discovered 21 genes with a genotype linked to fungal carriage, albeit they did not investigate whether this was linked to tinea capitis symptoms. The genes identified in this study were linked to a variety of tasks, including leukocyte activity, extracellular matrix remodeling, wound healing, and cutaneous permeability. Over 60% of the diversity in infection rate was accounted for by the risk score given to the genotypes in these 21 genes, and eight of the investigated genes appeared to account for the bulk of the observed heterogeneity in susceptibility to dermatophyte infections (108). These data suggest that a genetically defined impairment in adaptive immune responses may influence dermatophyte infection susceptibility. Furthermore, research findings have suggested probable interactions in prevalence, i.e., a failure in the innate response may impede the adaptive response, increasing vulnerability (19, 43, 109, 110).

T. rubrum dermatophytosis was linked to the expression of genes encoding IL-22, human-defensin 2 (hBD-2), and 4-defensin, according to Jaradat et al. (109). (DEFB4). The researchers discovered a link between the number of copies of DEFB4 mRNA and the incidence of T. rubrum–induced superficial dermatophytosis. The authors postulated that a low DEFB4 copy number, in combination with high IL-22 levels, was a risk factor for dermatophytosis pathogenesis. Other dermatophytosis candidate genes, such as the Fc receptor gamma, which is exploited by the pattern recognition receptor dectin-2 to promote innate immune responses against T. rubrum (111, 112), have been discovered in other research. This gene has been shown to have a varied number of copies in persons with and without infections, suggesting that it may play a role in dermatophyte infection pathogenesis.

CLEC7A-Y238X, an early stop codon variation that alters the detection of fungal-glucan by the receptor dectin-1, was identified in other investigations of a potential gene for susceptibility in individuals suffering from superficial dermatophytosis (113). Dectin-1 surface expression defects caused by the Tyr238X polymorphism resulted in-glucan recognition failure and cytokine response impairment in monocytes and macrophages (114). Because this polymorphism was found in all African communities studied, it is most likely an old mutation that first appeared more than 60,000 years ago, before the separation of modern human lineages in the late Paleolithic (115). In Europe, a genetic abnormality was discovered in a Dutch family with onychomycosis affecting all members (113).

The Human Genome Diversity Project (HGDP) can help elucidate the genetic susceptibility to dermatophytoses and give further information on the frequency and global distribution of genomic polymorphisms linked to dermatophytosis prevalence (116). By investigating genome-wide polymorphism databases, such investigations of haplotype diversity within the white population and other races, families, and populations will undoubtedly discover more genes that are crucial to enhanced sensitivity (43-267).

Conclusion

The heterogeneity of dermatophytes and their hosts suggests that infection susceptibility is most likely the result of alterations on both sides and reciprocal adaptation. Furthermore, a role for host genetics in the development of sickness has been discovered in several investigations, including probable Mendelian inheritance patterns for dermatophytosis propensity. The intricacy of the genetic connection between dermatophytes and their natural and unintentional hosts is underlined in this review. Different theories and approaches may lead to diverse interpretations of this link, according to a literature review. A vital step in better understanding these diseases is selecting an appropriate model for analysis and reasoning. There is also a large sector dedicated to studying the host's genetic and immunological response to dermatophyte infection.

In order to identify the main factors mediating infection risk that can be targeted to disrupt host–pathogen interactions and used in therapies, future studies will require a broader exploration of the dermatophyte genome in combination with analysis of large phenotypically well-characterized populations of various dermatophyte species. As a result, significant research on the relationships between dermatophytes and their unique hosts, which include complicated molecular pathways, is important both theoretically and practically. However, it is certain that genetic predisposition plays a significant role in dermatophyte infection susceptibility.

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