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Evaluating the Role of Antifungal Resistance in Treatment Failure of Dermatophytosis: A Systematic Review

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ABSTRACT

Background: The antifungal resistance during the treatment of dermatophytosis is becoming a global concern. Recently, the new dermatophyte strains, most significantly, *Trichophyton indotineae* (formerly T. mentagrophytes genotype VIII) has shown increased resistance against the antifungal medications in South Asia and other close regions. Henceforth, this review aims to systematically analyse current evidence regarding antifungal resistance in dermatophytes and evaluate its impact on treatment outcomes in patients with superficial fungal infections of skin.

Methodology: A systematic review was performed by searching PubMed/MEDLINE and Scopus using keywords 'dermatophytosis', 'antifungal-resistant dermatophytes', 'failed treatments' and 'recurrence'. During the search, 3,205 records were screened, and nine studies were included. Researchers extracted data on study designs, sample, fungal infections, resistance mechanisms, treatments and outcomes. The risk of bias of the included studies was assessed using Cochrane RoB2 and Newcastle–Ottawa Scales (NOS), and the findings were considered during the interpretation of the results.

Results: Terbinafine resistance was highest in *T. indotineae*, with 70% of isolates showing MICs of ≥0.5 mg/L. In North India, 60% of dermatophyte isolates were resistant to terbinafine and 85.6% to fluconazole, whereas itraconazole, ketoconazole and griseofulvin remained active. Higher terbinafine MICs were linked to slower cure and the need to switch to itraconazole, and itraconazole cured 92.5% of patients with terbinafine-resistant *T. indotineae* when serum levels exceeded 0.2 µg/mL. Where resistance was not tested, a two-week terbinafine course gave sustained cure in only 43% of patients. Most studies were observational, and few related MICs directly to clinical response.

Conclusion: Nevertheless, the preponderance of data suggests that antifungal resistance is indeed hindering effective therapy of dermatophytosis, especially where resistant *T. indotineae* dominates. Moreover, it is recommended that physicians should anticipate resistance in intractable cases, perform appropriate susceptibility testing, and consider strong antifungal options including itraconazole or other alternatives.

Keywords: *Antifungal Resistance, Dermatophytosis, SQLE Gene Mutations, Trichophyton Indotineae, Terbinafine Resistance.*

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INTRODUCTION

Dermatophytosis, or ringworm is a common and highly contagious superficial skin infection of keratinized tissues i.e., hair, nails, and skin which is mainly caused by *T. tonsurans, Trichophyton rubrum, Epidermophyton or Microsporum canis*[1]. The types of dermatophytoses are typically named for the area of the body that they affect: Tinea corporis (Body), Tinea pedis (Athlete's foot), Tinea cruris (Jock itch), Tinea capitis (Scalp) and Tinea unguium (Nails). It remains a significant public health concern worldwide, particularly in tropical and subtropical regions where heat and humidity favor fungal proliferation[2,3].

Cutaneous fungal infections are recounted to affect 20-25% of the global population, and in regions with high occurrences of fungal infections, the incidence can escalate to approximately 40-60%[4]. A 2013-2023 meta-analysis found onychomycosis was prevalent at 22.91% among tested samples with the highest prevalence in Southeast Asia[5]. Although fungal infections are rarely dangerous but recurrent

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and persistent infections lead to higher healthcare costs due to frequent checkups and extended treatment.

Globally, *Trichophyton rubrum* is leading cause of skin infections with highest prevalence among dermatophytes[6]. However, the epidemiology has seen a shift in last decade; Since 2014, Indian dermatologists have encountered a recurring dermatophytosis with poor response towards antifungal treatment, and *Trichophyton indotineae* (formerly *T. mentagrophytes* genotype VIII) surfaced as new dominant dermatophyte across India[7]. Since its emergence, it has spread in other regions with 1,215 confirmed cases in 36 different countries outside South Asia[8]. The global dissemination of *Trichophyton indotineae* is attributed to travel and migration across different corridors: intra-Asian, Asia–Europe, and Asia–America[8]. During 2022-2025, the cases of *Trichophyton indotineae* increased three folds from 237 in 2022 to 1,692 in 2025[9]. In the United Kingdom, UK National Mycology Reference Laboratory revealed that 38% of all dermatophytosis are caused by *T. indotineae*[10].

The infection typically presents as annular, erythematous, scaly lesions and is primarily managed with topical anti-fungal agents; however, more extensive or recalcitrant cases often necessitate systemic therapy, including azoles and allylamines[11]. Topical azoles or allylamines are standardized treatment, with skin therapy involving oral terbinafine or itraconazole reserved for widespread or intractable infections. Traditionally, rate of cures was higher, while cases of relapses and/or persistent infections were very rare with adequate treatments[3].

However, the last decade witnessed a rise of stubborn, chronic and susceptible fungal infections, particularly in South Asia and the Middle East[1,12]. The widespread misuse of topical corticosteroid-antifungal combinations and over-the-counter availability of antifungal medications contribute to the development and spread of resistant strains[4,13]. A new type of species, *T. indotineae*, was found to carry point mutations in the squalene epoxidase (SQLE) gene that results in extensive terbinafine resistance[14,15].

Clinically, antifungal resistance may result in chronic and/or recurrent infection, delayed cure, and higher rates of recurrence, which consequently complicates standard treatment protocols[12,16]. Moreover, it leads to prolonged patient morbidity, as well as increased healthcare costs and more likelihood of transmitting the infection to other people[17]. Although clinicians recognize the potential contribution of antifungal resistance in treatment failure, the extent to which it directly hinders the effective treatment of dermatophytosis remains insufficiently clarified[13], with present research evidence reporting variable outcomes.

Therefore, this systematic review aims to synthesize current evidence regarding antifungal resistance in dermatophytes and evaluate its impact on treatment outcomes in dermatophytosis. By integrating findings from clinical and microbiological studies, this review seeks to determine whether resistance significantly compromises therapeutic efficacy and to identify key gaps in the literature for future investigation.

MATERIAL AND METHODS

Search Strategy and Eligibility Criteria

This systematic review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA 2020) guidelines. A thorough literature search was conducted in PubMed/MEDLINE, and Scopus between 21-March-2026 and 24-March-2026 to identify studies evaluating antifungal resistance and treatment outcomes in dermatophytosis. The search strategy was formulated which involved use of appropriate Boolean operators to combine terms. The databases were searched using terms related to dermatophytosis and tinea infections, antifungal resistance or susceptibility, and treatment outcomes. PICO framework was applied to decide eligibility criteria: The **population (P)** constitutes patients with dermatophytosis; the **intervention/exposure (I/E)** was antifungal resistance or reduced antifungal susceptibility; the **comparator (C)** involved susceptible infections and/or patients who received alternative antifungal treatment; and the **outcomes (O)** included treatment failure, cure, clinical or mycological response, or relapse. Original clinical studies offering relevant data on anti-fungal resistance and clinical outcomes were included for the study while clinical reports without relevant outcome data, animal studies, and systematic reviews were excluded.

Data extraction and Risk-of-bias assessment

From each study, researchers summarized author/year, country, study design, patient demographics, fungal species, identification methods for type of fungus (culture, ITS

sequencing), antifungal susceptibility data (MICs, SQLE mutations), dosage and duration of antifungal medications, clinical outcomes (clinical/mycological cure, time to cure, relapse, adverse events). To perform risk-of-bias assessment, Cochrane RoB2 was applied for appraisal of randomized trials while Newcastle–Ottawa Scales (NOS) was applied for observational studies. The RoB 2 was used to assess the five domains of bias: Randomization process, Deviations from intended interventions, Missing outcome data, Measurement of the outcome, and Selection of the reported result. The NOS assessment was applied for selection, comparability, and outcome/exposure ascertainment. Two reviewers independently extracted data and performed risk-of-bias assessment whereas mutual consent was taken to resolve any discrepancies.

RESULTS

Study selection

The database search returned 4,117 records (Scopus, n = 3,022; PubMed/MEDLINE, n = 1,095). After 912 duplicates were removed, 3,205 records were screened by title and abstract, and 3,013 were excluded. All 192 reports sought for retrieval were obtained and assessed in full text. Of these, 183 were excluded because they lacked relevant outcome data, were animal studies or were review articles, which left nine studies for the review (Figure 1).

Characteristics of included studies

The nine studies were published between 2016 and 2025 (Table 1). Eight were conducted in South Asia (six in India, one in Pakistan and one in Bangladesh), and one was a multinational genomic study. The designs comprised one prospective cohort[18], two randomized trials[19,20], one case report[21], one retrospective genomic epidemiology study[22], one prospective clinicomycological study[23] and three cross-sectional studies[24–26]. Sample sizes ranged from a single patient to 570 patients.

All studies identified the fungus by direct microscopy and/or culture, and two confirmed the species by internal transcribed spacer (ITS) sequencing[22,24]. Susceptibility was measured by minimum inhibitory concentration (MIC) testing in six studies[20–24,26] and by agar well diffusion in one[25], while two relied on clinical response alone[18,19]. Three studies also screened for squalene epoxidase (SQLE) gene mutations[21–23]. Terbinafine and itraconazole were the drugs evaluated most often, followed by fluconazole and ketoconazole; griseofulvin, voriconazole and amphotericin B were tested only in vitro[24,26].

Antifungal resistance prevalence

Resistance differed by species, drug and setting (Table 1). Terbinafine resistance was most marked in *T. indotineae*. In the multinational genomic study, 63 of 90 isolates (70%) had terbinafine MICs of ≥0.5 mg/L, and six isolates with raised MICs carried no SQLE mutation, which points to other resistance mechanisms[22]. Indian *T. indotineae* isolates in the trial by Khurana et al. likewise had uniformly high terbinafine MICs (8–32 µg/mL) but remained susceptible to itraconazole (0.06–0.5 µg/mL)[20]. In North India, 60% of dermatophyte isolates were resistant to terbinafine and 85.6% to fluconazole, whereas none were resistant to itraconazole, ketoconazole or griseofulvin[26]. Fluconazole was also the least active drug against isolates from Southern Punjab, Pakistan, where ketoconazole and voriconazole performed best and some *T. rubrum*, *T. interdigitale* and *Aspergillus versicolor* isolates resisted amphotericin B and/or fluconazole[24]. In Bangladesh, resistance rates were 70% for fluconazole, 55% for itraconazole and 20% for terbinafine[25].

Treatment outcomes by resistance status

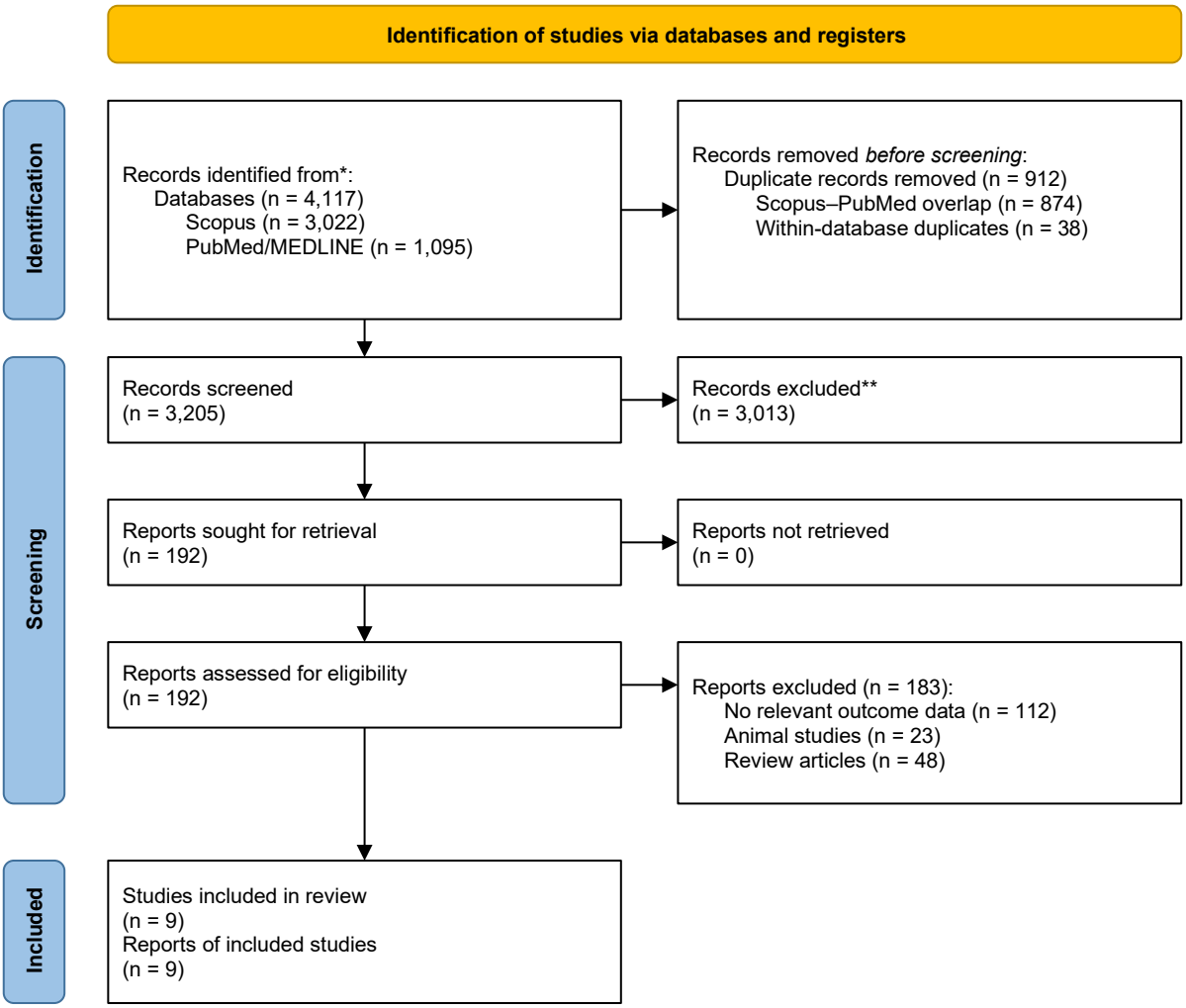
Two studies related laboratory findings directly to clinical response. Khurana et al.[23] followed 30 patients with *T. interdigitale* infection treated with terbinafine. Half were cured with the standard dose (mean 39 days), a second group needed a higher dose (mean 56 days), and a third group failed terbinafine and was cured after switching to itraconazole (mean 32 days). Higher terbinafine MICs went together with a slower response and the need to switch. In the dose-ranging trial by Khurana et al.[20], itraconazole cured 37 of 40 patients (92.5%) infected with terbinafine-resistant *T. indotineae*. Every patient with a serum itraconazole level above 0.2 µg/mL was cured, whereas failures occurred only below this level, so adequate drug exposure mattered as much as susceptibility.

Subba et al.[19] found significantly higher cure rates with itraconazole-containing regimens than with terbinafine alone, and standard-dose terbinafine was associated with more failures and relapses. Resistance in that trial, however, was inferred from clinical response because no MIC testing was done. Sharath et al.[21] described a patient with *T. mentagrophytes*/*T. interdigitale* infection who failed terbinafine, conventional and super-bioavailable itraconazole and fluconazole over several months, and whose lesions finally cleared with oral ketoconazole.

The Bangladeshi study pointed the other way: one month of terbinafine 250 mg daily cured 82% of patients (18% relapsed), compared with 56% for itraconazole and 45% for fluconazole[25]. In Kashmir, Majid et al.[18] treated 100 patients with terbinafine 250 mg daily for two weeks. Initial clinical and mycological cure was seen in 65%, but 22 patients relapsed within 12 weeks, leaving a sustained cure rate of 43%. The infections were caused mainly by *T. rubrum* (55%) and *T. tonsurans* (20%), and as no susceptibility testing was performed, these failures cannot be attributed to resistance; the short two-week course is the more likely explanation. The remaining three studies reported laboratory data only[22,24,26].

Risk-of-bias assessment of studies

Methodological quality varied considerably across the nine studies. The two randomized trials[19,20] were appraised with the Cochrane Risk of Bias 2 (RoB 2) tool, and both raised some concerns (Table 2a). The six observational studies were appraised with the Newcastle–Ottawa Scale (NOS) (Table 2b): three were judged at moderate risk of bias, one at moderate-to-high risk and two at high risk, mostly because of weak comparability between groups. The case report by Sharath et al.[21] was not scored, as a single-patient observation cannot be compared with cohort or case-control designs and offers the lowest level of evidence.



*Records identified from each database are shown separately. **All exclusions at screening were made by the reviewers; no automation tools were used.

Figure 1: PRISMA 2020 flow diagram of study selection

Table 1. Study Characteristics

Research papers	Country [a]	Design [b]	n (cases) [c]	Species (major) [d]	Resistance test [e]	Antifungal regimen and outcome [f]
Majid et al.[18]	India (Kashmir)	Prospective cohort	100 patients with tinea corporis and/or tinea cruris	55% cases of *Trichophyton rubrum* while 20% *Trichophyton tonsurans*; Few other dermatophytes	Clinical examination, phenotypic identification methods and fungal culture; No molecular resistance testing or MIC antifungal susceptibility testing	Oral Terbinafine 250 mg once daily for 2 weeks. Outcome: Only 65% achieved initial clinical and mycological cure; during 12-week follow-up, 22 patients relapsed, while long-term cure was seen in merely 43% of the patients
Subba et al.[19]	India	Randomized controlled trial (RCT)	275 patients with tinea corporis and/or tinea cruris	Majorly *Trichophyton* mentagrophytes complex; some *Trichophyton rubrum* species were also present.	KOH microscopy and fungal culture for diagnosis; Antifungal susceptibility/resistance was checked via clinical response and rates of relapses; no detailed molecular resistance testing	Oral terbinafine and itraconazole in four regimens: a) standard-dose terbinafine, b) high-dose terbinafine, c) itraconazole and d) terbinafine plus itraconazole. Outcome: Itraconazole-containing regimens showed significantly better cure rates than terbinafine alone. Standard-dose terbinafine had poor response and higher treatment failure/relapses
Khurana et al.[20]	India	Randomized dose-ranging trial	40 patients with tinea corporis/ cruris	*Trichophyton indotineae*	KOH microscopy and fungal culture; antifungal susceptibility evaluated via Minimum Inhibitory Concentration (MIC) testing for itraconazole and terbinafine	Itraconazole 100, 200 or 400 mg/day. Outcome: 37 of 40 (92.5%) cured. Isolates were itraconazole-susceptible (MIC 0.06–0.5 µg/mL) with high terbinafine MICs (8–32 µg/mL). All patients with serum itraconazole >0.2 µg/mL were cured; 2 of 10 below this level failed
Sharath et al.[21]	India	Case report	1 patient	*T. mentagrophytes* /*T. interdigitale* species complex	CLSI MIC, SQLE PCR	Failed terbinafine 250 mg BD (8 weeks), itraconazole 100 mg BD (8 weeks), super-bioavailable itraconazole 130 mg OD (12 weeks) and fluconazole 50 mg OD (4 weeks). Outcome: cleared with oral ketoconazole 200 mg BD plus topical ciclopirox
Rhodes et al.[22]	Multinational; India, United Kingdom, Ireland, France, Canada, and type strain from Japan	Retrospective genomic epidemiology study (isolates 2014–2023; cases 2018–2023)	103 cases of severe dermatophytosis; 90 isolates confirmed as *T. indotineae*	*T. indotineae*	Internal transcribed spacer (ITS) sequencing and Minimum Inhibitory Concentration (MIC) to test antifungal susceptibility	Effects of terbinafine were evaluated in patients. Outcome: Out of ninety *T. indotineae* isolates, 63 (70%) showed resistance to terbinafine with MIC ≥ 0.5 mg/L. 6 isolates showed high MIC values without SQLE mutations showing existence of alternative resistance

						mechanisms. It also showed rapid transcontinental spread and significant terbinafine resistance
Khurana et al.[23]	India	Prospective observational clinicomycological study with antifungal susceptibility testing and treatment response correlation	85 enrolled; 64 culture-positive; 30 with complete follow-up for outcome analysis	*T. interdigitale* (64 isolates)	CLSI M38-A2 broth microdilution antifungal susceptibility testing + SQLE gene sequencing (mutation analysis of terbinafine resistance)	Terbinafine (TRB) 250mg OD then BD; Itraconazole (ITR) 100 mg BD for non-responders. Outcomes: Group 1 (50%) responded to TRB (mean cure 39 days), Group 2 required higher TRB dose (mean 56 days), Group 3 failed TRB and required ITR with cure in 32 days. Higher TRB MIC correlated with poorer response and need for ITR
Ismail et al.[24]	Pakistan	Laboratory-based cross-sectional observational study	74 clinically diagnosed dermatophytosis cases	Nine species: *T. interdigitale*, *T. mentagrophytes* and *T. rubrum*, plus non-dermatophytes *Aspergillus terreus*, *A. versicolor*, *A. niger*, *Alternaria alternata*, *Acremonium sordidulum* and *A. sclerotigenum*	Culture on Sabouraud dextrose agar (SDA) and phenotypic microscopy; species confirmed by ITS1/ITS4 PCR with DNA sequencing; antifungal susceptibility by MIC	Antifungals tested: amphotericin B, fluconazole, ketoconazole, voriconazole. Outcome: fluconazole showed the highest resistance and was least effective; ketoconazole and voriconazole were most effective. Some *T. rubrum*, *T. interdigitale* and *A. versicolor* isolates were resistant to amphotericin B and/or fluconazole
Chowdhury et al.[25]	Bangladesh	Cross-sectional study	570 consecutive patients with superficial fungal infection (360 males, 210 females)	Mixed dermatophyte species causing SFI	Agar well diffusion antifungal susceptibility testing (zone of inhibition measurement) + culture on DTM; resistance defined clinically as relapse or no response; MIC not used	Fluconazole 50 mg OD (6 weeks): 45% cured, 55% relapse/ineffective. Itraconazole 100 mg BD (1 month): 56% cured, 44% relapse. Terbinafine 250 mg OD (1 month): best response with 82% cure, 18% relapse. Resistance rates: 70% (fluconazole), 55% (itraconazole), 20% (terbinafine)
Lohariwala et al.[26]	India	Descriptive cross-sectional study	130 enrolled; 115 KOH-positive; 90 culture-positive dermatophyte isolates analyzed	*Trichophyton mentagrophytes* (predominant 75.7%), *T. rubrum* (2.6%), rare *Aspergillus spp.* (4.3%)	CLSI M38-A2 broth microdilution antifungal susceptibility testing (MIC determination); KOH microscopy and culture (SDA, DTM, LPCB, urease test) for identification	Fluconazole: 85.6% resistant, 14.4% sensitive (poor effectiveness). Terbinafine: 60% resistant, 40% sensitive (reduced efficacy). Itraconazole, ketoconazole, griseofulvin: 100% sensitive (no resistance observed)

Table 1. a: country of study, b: study design, c: number of cases/ patients, d: type of dermatophyte, e: test performed to identify resistance, f: antifungal used for treatment and its outcome.

Table 2a Risk of bias in randomized studies using Cochrane RoB 2

Study	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of outcome	Selection of reported result	Overall risk of bias
Subba et al.[19]	Some concerns	Some concerns	Low risk	Some concerns	Some concerns	Some concerns
Khurana et al.[20]	Some concerns	Some concerns	Some concerns	Low risk	Some concerns	Some concerns

Table 2b Risk of bias in observational studies using the Newcastle–Ottawa Scale

Study	Selection	Comparability	Outcome/Exposure	Overall assessment
Majid et al.[18]	Moderate concern	High concern	Low concern	**Moderate risk of bias**
Rhodes et al.[22]	Moderate concern	Moderate-high concern	Low concern	**Moderate risk of bias**
Khurana et al.[23]	Moderate concern	High concern	Low concern	**Moderate-high risk of bias**
Ismail et al.[24]	Moderate-high concern	High concern	Moderate concern	**High risk of bias**
Chowdhury et al.[25]	Moderate-high concern	High concern	Moderate concern	**High risk of bias**
Lohariwala et al.[26]	Moderate concern	High concern	Low–moderate concern	**Moderate risk of bias**

Note: The NOS was originally developed for cohort and case-control studies. Therefore, for studies with cross-sectional, descriptive, or other non-comparative designs, the NOS domains were interpreted cautiously and were not used to imply that these studies had the same evidentiary strength as comparative cohort or case-control studies. The overall judgments reflect the methodological limitations relevant to the review question.

Overall, the risk-of-bias assessment indicates that the available evidence should be interpreted cautiously. Although several studies demonstrate an association between antifungal resistance, elevated minimum inhibitory concentrations, resistance-associated mutations, and poor therapeutic response, the heterogeneity of study designs and methodological limitations prevent definitive causal inference regarding the role of antifungal resistance in treatment failure.

DISCUSSION

This systematic review indicates that antifungal resistance is an important contributor to treatment failure in dermatophytosis, although the strength of evidence varies according to the dermatophyte species, antifungal agent, and study design. The most consistent evidence was observed in infections caused by *T. indotineae*, in which elevated terbinafine MICs and resistance-associated SQLE mutations were frequently accompanied by poor clinical response to terbinafine[27,28]. Mechanistic studies showed common SQLE mutations associated with resistance[29]: in *T. indotineae*, Phe397Leu (33%) and Ala448Thr (25%) were frequent[28]; in *T. rubrum*, Phe397Leu/Ile (41.6%) and Leu393Phe (20.8%) were documented in resistant isolates[29]. Other factors (efflux pumps, biofilm formation) may also contribute[30–32].

A recent individual patient data meta-analysis[33] indicated that 85.3% of cases were terbinafine-resistant, and 21.9% showed itraconazole-resistance. Median time to complete cure was 11.5 weeks, and relapse occurred in 19.7%. Crucially, initial therapy with itraconazole was far more effective than terbinafine: among patients receiving oral itraconazole (without switching), 75.8% improved compared to only 18.2% on terbinafine ($p<0.001$)[33]. In other words, these findings suggest that terbinafine resistance has a higher probability of treatment failure compared to susceptible infections.

Previous clinical trials and susceptibility studies reinforce that terbinafine-resistant infections require alternative therapy. An RCT of 275 Indian patients found significantly higher cure with itraconazole-based regimens than terbinafine alone[19]. In vitro susceptibility data corroborate clinical outcomes: *T. indotineae* isolates have mean terbinafine MIC 12 µg/mL (MIC_{50}=4), versus itraconazole MIC_{50}=0.06 µg/mL[33]. In practice, switching to itraconazole (usually 200 mg daily) or using combination therapy often saves treatment. The MMWR reported two US cases of *T. indotineae*: both failed 4–6-week terbinafine therapy but improved with itraconazole (or griseofulvin)[34].
Due to limited comparative studies, the analysis assumed that research on *T. indotineae* generally indicates “resistant exposure”,

whereas earlier clinical trials represented "susceptible comparators". Systematic analysis revealed strong evidence of antifungal resistance hampering treatment of tinea corporis. In areas with endemic and resistant *T. indotineae*, standard terbinafine course is ineffective. In cases where dermatophytes were not sequenced, the rate of clinical failure was high inspite of treatments. The laboratory data associates these failures with high MICs and SQLE mutations[31].

Moreover, antifungal resistance affects the clinical outcomes as resistant dermatophytes remain incurable with standard topical and/or oral therapies as cure rate falls with standard terbinafine treatment[19,33], resulting in prolonged and combined courses[34,35]. It also extends the resolution time, resulting in prolonged treatment courses with high probability of a relapse. Nevertheless, studies reported effectiveness of alternative treatment course particularly itraconazole and voriconazole[24,36,37] to treat dermatophyte infections, although evidence on voriconazole remains insufficient. Notably, steroid misuse (common in India) was associated with non-improvement, indicating that factors other than antifungal resistance may also contribute to poor clinical outcomes.
Similarly, this present review underlines that occurrence of antifungal resistance is higher in developing countries. Terbinafine-resistant isolates carrying SQLE mutations have been documented across India and in Iran[16,30]. On contrary, *T. rubrum* resisted clinical therapy in regions outside South Asia[35], whereas in the United States, *Trichophyton indotineae* was identified as highly resistant to terbinafine[38]. Hence, it is imperative that clinicians suspect anti-fungal resistance while handling chronic or extensive tinea corporis.

Limitations

Although this review presents a strong evaluation on the antifungal resistance in tinea corporis, there are some limitations which should also be considered as well. The existing data mostly includes case studies from clinical centers while there is limited or rare data from randomized trials exclusively researching on resistance. Similarly, there were no extensive cohort comparisons to evaluate the different outcomes. Hence, it is recommended that more research is needed on ideal treatments for antifungal resistance and on investigation of resistance trends. Moreover, clinicians need to update their first-line management against the dermatophytes, and regions with higher prevalence of resistance should consider itraconazole or other alternatives topical and/or oral antifungals in earlier stages of the infection.

CONCLUSION

In conclusion, evaluated evidence indicates that antifungal resistance does hinder the treatment of tinea corporis, particularly in areas with emergent resistant Trichophyton strains. It is crucial to recognize this challenge in order to adjust and/or upgrade treatment strategies and regulate public health surveillance. Clinicians should be attentive to chronic and extensive antifungal resistance in tinea corporis, and it should be reasonable to empirically switch from terbinafine to itraconazole (or combination therapy) if the symptoms are not improving in 2-3 weeks. In future, research studies should target new antifungals and effective management for preventing further spread of resistant dermatophytes.

Author Contributions
KA: Conception and design, data analysis, and manuscript writing
WH: Literature search, study selection, and data extraction
HS: Study selection, data extraction, and risk-of-bias assessment
HT: Results synthesis and drafting of the discussion
IN: Results synthesis and critical review
MH: Proofreading, critical review, and final approval

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Data availability: No new data were generated or analyzed in this review.

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