Trichophyton mentagrophytes genotype VII: early detection and targeted resistance testing

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Article highlights:

- Routine laboratory methods may miss TMVII, so local spread can go unnoticed.
- Use genotype testing to identify linked cases and antifungal susceptibility testing when treatment fails.

Key words:
antifungal susceptibility testing; dermatophytosis; molecular diagnosis; sexual transmission; surveillance; *Trichophyton mentagrophytes* genotype VII

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Abstract

Trichophyton mentagrophytes genotype VII (TMVII) first gained attention in reports of genital tinea after travel. Recent case series, genomic studies, and public health alerts suggest that it is now spreading locally in parts of Europe and the United States. TMVII can cause painful, inflamed, or follicular tinea in the genital area and at other skin sites. Routine tests often identify only the *T. mentagrophytes/T. interdigitale* species complex, so TMVII may be missed. Its current pattern differs from that of *T. indotineae*, in which terbinafine resistance is common. In a New York laboratory series, all 39 TMVII isolates had low minimum inhibitory concentrations for terbinafine and itraconazole. A case with terbinafine resistance has also been reported. Two decisions are central to care: when to confirm the genotype and when to request antifungal susceptibility testing. Genotype testing is useful when clinical findings, contact history, or linked cases suggest local spread. Susceptibility testing is most useful when oral treatment fails, recurrent, or previous antifungal use raises concern about resistance. Sexual contact describes one route of exposure, it does not define who can be infected. In practice, clinicians should suspect TMVII in the right setting, confirm it when the result matters, and follow the response to treatment.

Why TMVII matters now

Early reports linked *Trichophyton mentagrophytes* genotype VII to genital tinea after sexual contact during travel. Later case series documented transmission among men who have sex with men (Luchsinger *et al.*, 2015; Kupsch *et al.*, 2019; Jabet *et al.*, 2023). A French study reported 13 cases diagnosed between 2021 and 2022 (Jabet *et al.*, 2023). A later Paris series reported 32 cases, including 17 linked to one massage setting (Jabet *et al.*, 2025). New York investigators identified 39 cases between 2022 and 2024. Most sequenced isolates were closely related, which was consistent with possible local spread (Todd *et al.*, 2026). In February 2026, a Minnesota health alert reported more than 30 confirmed or suspected cases in the metropolitan area (Minnesota Department of Health, 2026).

These reports cannot show how common TMVII is in the general population, but they confirm that local spread can occur and that cases may go unrecognized. Two questions matter in clinical practice. When should the genotype be confirmed to identify possible transmission? When should antifungal susceptibility testing be requested to investigate resistance? These tests answer different questions and should not be used interchangeably.

When should clinicians suspect TMVII?

No single clinical appearance is specific for TMVII. Consider it when tinea is painful, very inflamed, pustular, nodular, or follicular. Suspicion should also rise when lesions involve the genital or perianal area, buttocks, face, beard area, or skin around the mouth; persist or recur; or follow close skin contact with a person who has a similar rash. The Centers for Disease Control and Prevention also lists contaminated items as a possible route of spread, although their role in current clusters is unclear (CDC, 2026).

A German cohort of 51 patients shows why diagnosis may be delayed. The median time to diagnosis was 42.5 days. Purulent or infiltrated lesions were reported in 82.4 % of patients, and the median duration of systemic treatment was 83.5 days (Kämmerer *et al.*, 2025). The cohort came from a specialist setting and may include more severe cases than routine practice. Even so, limiting suspicion to a typical annular genital rash will miss patients.

The exposure history is as important as the skin findings. Ask about the sites of intimate skin contact, symptoms in partners, travel, shared towels or bedding, shaving, topical corticosteroids, and previous antifungal treatment. Most

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reported clusters have involved men who have sex with men, but anyone with relevant exposure can be infected. A recent report described TMVII with terbinafine resistance in a woman after sexual exposure during travel (Korbi *et al.*, 2026). Sexual contact is one route of exposure. It should not be used to define the type of patient at risk.

When genotype testing is useful

Direct microscopy and fungal culture can confirm dermatophytosis, but they usually cannot identify TMVII. Many routine methods identify only the *T. mentagrophytes/T. interdigitale* species complex. TMVII can be confirmed by sequencing the full internal transcribed spacer (ITS) region (Tang *et al.*, 2022; Todd *et al.*, 2026).

There is no need to sequence every uncomplicated tinea case. Genotype testing is most useful when the result may change patient care or public health action. Examples include anogenital or facial disease after intimate contact, marked inflammation, follicular lesions, linked cases, relevant travel, and infection that persists or returns after treatment. When possible, specimens for culture should be collected before or early in systemic treatment, and the isolate should be stored for further testing (Minnesota Department of Health, 2026).

Do not assume resistance

TMVII and *T. indotineae* belong to the same species complex, but their current resistance patterns differ. In the New York series, all 39 TMVII isolates had very low terbinafine minimum inhibitory concentrations (MICs) and low itraconazole MICs (Todd *et al.*, 2026). In an Italian series of three isolates, the squalene epoxidase gene (*SQLE*) had no substitutions known to cause terbinafine resistance (Gaspari *et al.*, 2026). These findings are reassuring, but the sample sizes remain small. There are no epidemiological cutoff values or clinical breakpoints for TMVII. A low MIC alone does not guarantee clinical success (Todd *et al.*, 2026).

Resistance can occur. In the 2026 case, four weeks of oral terbinafine did not clear the infection. Antifungal susceptibility testing confirmed terbinafine resistance, and the patient responded to itraconazole (Korbi *et al.*, 2026). Treatment failure does not always mean resistance. Other causes include an incorrect diagnosis, follicular disease, use of topical corticosteroids, an inadequate treatment course, poor adherence, reinfection, or continued contact with an infected person. Antifungal susceptibility testing is most useful when disease worsens, does not improve after an adequate systemic course, or returns. *SQLE* sequencing may help when the terbinafine MIC is high or the clinical course is difficult to explain. Results should be interpreted with the diagnosis, drug dose, treatment duration, adherence, exposure history, and clinical outcome.

A practical clinical approach

Suspect: Consider TMVII when tinea is painful, inflamed, follicular, persistent, or recurrent and involves the genital or perianal area, buttocks, face, beard area, or skin around the mouth. Suspicion is stronger after close skin contact or when linked cases are known.

Confirm: Perform direct microscopy and fungal culture. Request full ITS sequencing or another validated genotype assay when the result could help identify linked cases, guide assessment of contacts, or explain an unusual clinical course. Store the isolate when possible.

Follow up: Review adherence, treatment duration, topical corticosteroid use, follicular involvement, reinfection, and continued exposure. Request antifungal susceptibility testing when treatment fails or infection returns. Assess partners or close contacts who develop a rash. Involve public health teams when several linked cases are suspected. Current evidence supports assessment of symptomatic contacts, not routine testing or treatment of people without symptoms (CDC, 2026).

TMVII is not known to be common in the general population, but local spread can go unnoticed for weeks. Use genotype testing when clinical findings or exposure history suggest linked transmission. Use antifungal susceptibility testing when the response to treatment raises concern about resistance. Research should now measure prevalence in clearly defined populations, validate cheaper and faster tests, link MIC results with patient outcomes, and measure transmission before visible lesions appear.

Declarations

Conflict of interest: The authors declare no conflict of interest.

Data availability: No new data were generated or analysed for this Comment.

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