Author's reply

Sir,

It is a pleasure to reply to the comments on our article[1] and I thank you for the opportunity to make this humble submission.

What did we do?

As mentioned,[1] the study was performed to find evidence for or against the perceived loss of effectiveness of terbinafine in tinea infections in India. We presented the first data and said that terbinafine is not working in real-world setting. That is all.

Did we speculate about the causes of ineffectiveness of terbinafine?

We did not speculate about the causes, which may be many, nor did we mention that the cause of abysmal effectiveness is drug resistance. In fact, the word "resistance" is missing from the article. And would it not be in the fitness of things to wait for future research to find the answers?

Comparison with commentators' onychomycosis study

Commentators say that in their onychomycosis study, "patients were normally treated according to the British guidelines". The guidelines recommend oral terbinafine for 6 weeks in fingernail and for 12–16 weeks in toenail infection.[2] Two other sentences cloud the issue of how long the treatment was given, these are, "only 24.3% of the patients, responded to treatment after 6 months" and "21% of them said they only received a treatment for <4 months". Was the duration of treatment 6 months? In any case, the duration of treatment was much longer than 4 weeks.

Comparing the commentators' onychomycosis study with ours is comparing dissimilar studies. Cure rates of tinea corporis/cruris/faciei and onychomycosis cannot be compared. Chances of poor compliance occurring in their study of long treatment duration are much higher than in our study of 4 weeks. Also, the causes of poor compliance found in their study (cost of drug and fear of adverse effects), being dependent on duration of treatment, would be minimal and inconsequential in our study. Furthermore, it is hard to believe that the patients of our study were coming for follow-ups but were not taking treatment. I refrain from commenting further because (a) the data provided are thin and (b) reference to the study is not available.

Does poor compliance as *the* cause of ineffectiveness of terbinafine really stand up to scrutiny?

A mini thought experiment, comprising two invariables (excellent effectiveness of terbinafine until recent past and its dramatic decline now) and a hypothetical antecedent to the second event (poor compliance), will shed some light on the issue. To accept that the hypothetical antecedent happened and caused the second event, we must accept that a couple of years ago, almost precipitously, profound unidirectional change in treatment-taking behavior of patients living over a large land mass occurred. Now, you see the problem, and the implausibility of poor compliance explaining the lack of effectiveness of terbinafine, as of now and pending new evidence, becomes apparent. Poor compliance may have contributed, but the chances of it being the sole or main cause are slim.

"End of the road?" or "End of the road"?

The only place where we used the phrase "end of the road" in the article is the title. There too it was "End of the road for terbinafine?" (note the question mark, which makes the phrase open-ended, interrogative and in the spirit of enquiry), and *not* "End of the road for terbinafine" (which would be affirmative). The difference between the two may be appreciated.

The big question

In the last analysis, following questions present themselves: Should a drug, which has been shown to be ineffective in clinical trial, be considered effective based on *in vitro* studies (in case the fungus is susceptible)? Is it not the clinical trial that is *the final arbiter* on the effectiveness of a drug? This is the question.

Let us go where the data take us.

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Conflicts of interest

There are no conflicts of interest.

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