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Author's Response to Burgess

Hermann Feldmeier

Sir,

I thank Dr. Ian F. Burgess for critically commenting on the new concept for the treatment of epidermal parasitic skin diseases (EPSD), a group of infectious diseases common in the tropical world. However, I do not think that the article "is potentially misleading and likely to cause confusion and distress in some circumstances." Please let me address the points raised by Dr. Burgess one by one.

In the article, EPSD were defined as parasitic diseases caused by mites, lice and other blood-sucking insects such as fleas. The potential of dimeticones with appropriate physico-chemical characteristics to kill such ectoparasites in vivo was discussed. Ticks were not included as a target for the new treatment concept, because they can be removed effectively with simple devices.

The effectiveness of dimeticones with defined physico-chemical characteristics for the treatment of tungiasis has been clearly demonstrated. First, the cited study was sufficient to obtain valid conclusions, and an efficacy of 78% is substantial, taking into account that current treatments do more harm than good [1, 2]. Second, a recent study showed that treatment targeted to the site where sand fleas are embedded increased the efficacy to 97%. Besides, the targeted approach reduced the amount of the dimeticone needed by a factor of 20, thereby increasing the cost-effectiveness of this approach [3].

The worry by Dr. Burgess that "… killing a large cluster of fleas all at once could have the same potentially hazardous effect as using a conventional insecticide for the same purpose" seems inappropriate. First, we are not aware of any study showing that the death of ectoparasites can cause a "Jarisch-Herxheimer-like reaction." By definition, the Jarisch-Herxheimer reaction is an inflammatory response developing when a high number of certain bacteria species present in the blood or in internal organs are killed by an antibiotic. Second, it is difficult to see how "elimination of all the [ectoparasitic] arthropods at one time potentially releases a large amount of immunogenic material into the body." In fact not a single case of a pronounced inflammatory reaction was observed in more than 20,000 Kenyan children with tungiasis who were treated topically with a combination of neem extract (an insecticide) and coconut oil (a suffocating compound), even if the patients had hundreds of embedded sand fleas (Dr. Lynne Elson, unpublished observation, 2014). Instead, as soon as the embedded sand fleas are killed, the already existing local inflammation regresses rapidly [4, 5].

With regard to scabies, Dr. Burgess excludes the possibility that dimeticones are effective against Sarcoptes mites. He states that "mites have no problem surviving because dimeticone is highly oxygen permeable and so the mites simply swim around in the fluid if they are immersed." First, such a statement has to be substantiated by an appropriate reference. Second, the dimeticones hitherto used will immobilize mites and thereby prevent them from continuing to feed; eventually this will result in the death of the ectoparasites. Besides, the argument that adult mites "which are the primary target of treatment are all hidden in burrows in the epidermis and that there is no direct exposure access anyway to a dimeticone treatment" is not valid. The tunnels are comparatively large openings in the skin into which an appropriate dimeticone can easily seep and eventually cover the mite (In fact, the diameter of the burrow is so large that even water penetrates, a fact used for diagnosis in the burrow-ink-test). Moreover, whereas Sarcoptes mites are burrowed in tunnels in superficial layers of the stratum corneum. *T. penetrans* is embedded in lower strata almost completely surrounded by host tissue. Nonetheless, appropriate dimeticones kill embedded sand fleas.

References

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Institute of Microbiology and Hygiene, Charite University Medicine, Berlin Campus Benjamin Franklin, Hindenburgdamm 30, 12203 Berlin, Germany
Tel: +49-163-674 37 07
E-mail: Feldmeier.FUBerlin@t-online.de

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