Response to: 'In the idiopathic inflammatory myopathies, reactive oxygen species are at the crossroad between immune and non-immune cell mediated mechanisms' by Meyer *et al*

We welcome the comments from Meyer *et al*[1] regarding our recent article which hypothesised a potential role for reactive oxygen species (ROS) as a mediator of muscle weakness induction in the idiopathic inflammatory myopathies (IIM).[2] The focus of our article was specifically on the persistence of muscle weakness in the treated paucity or absence of inflammatory cell infiltrates, and the role that ROS may play as a mediator of this residual and non-inflammatory dysfunction.[2] In our article we did not try to imply that the mechanisms of actions of immune cells and ROS generation are mutually exclusive, as clearly inflammatory cell infiltrations are a cause of muscle cell injury and thus weakness. In another recent, and related, review we suggest that inflammatory cell infiltration is in fact a 'secondary' feature in IIM.[3] We agree that there is likely an interaction between immune cell-mediated and ROS-mediated muscle dysfunction. The interesting beneficial effects of using ROS-targeted therapies as described by Meyer *et al*[4] in the context of IIM thus appear timely and indeed fundamentally important. Based on their findings and our own postulates, it is perhaps logical to now consider the role of ROS in immune and non-immune settings in IIM. Moreover, and given the recent advances in ROS-targeted compounds, a new dawn may now be on the horizon regarding novel therapeutics to target weakness in IIM.

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