In the idiopathic inflammatory myopathies, reactive oxygen species are at the crossroad between immune and non-immune cell-mediated mechanisms

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Dear editor,

We read with great interest the article by Lightfoot *et al*[1] who suggested that in the idiopathic inflammatory myopathies (IIM), reactive oxygen species (ROS) contribute to muscle weakness. The authors opposed immune and non-immune cell-mediated mechanisms in IIM and reviewed ROS as part of the latter. Here, we would like to point out recent data that reconcile this dichotomy and stress the interplay between ROS and immune cell-mediated processes during IIM.

In accordance with the authors' hypothesis, we recently observed high ROS formation, along with mitochondrial respiratory chain dysfunctions, in muscle of patients with dermatomyositis.[2] Furthermore, in muscle cells cultured in the absence of immune cells, interferon-β-induced mitochondrial dysfunctions in a ROS-dependent manner. Finally, in an antigen-induced mouse model of IIM, ROS scavenging with N-acetyl cysteine prevented muscle weakness and mitochondrial dysfunctions and immunize cell infiltrate in muscle.[2]

Thus, ROS and immune cells are not independent actors in IIM. Indeed, ROS participate in immune cell infiltrate, which in turn can further increase ROS formation and mediated damages in the muscle.[3] This definitely places ROS as central actors and potential therapeutic targets in IIM.

REFERENCES

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