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LETTER

Response to Article "Autophagy Inhibits Inflammation via Down-Regulation of p38 MAPK/mTOR Signaling Cascade in Endothelial Cells" [Letter]

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Dear editor

The work performed by Zhou et al regarding the regulative mechanism of autophagy that inhibits inflammation via down-regulation of p38 MAPK/mTOR signaling cascade in endothelial cells has been reviewed and much appreciated.[1] In their study, rapamycin-induced autophagy was shown and proven to be able to improve the function of endothelial cells in psoriasis, the chronic immune-mediated skin disease. Their results suggested that approaches to induce autophagy could be used to ameliorate psoriasis. This work supplied novelty and promising ideas in the autophagy and psoriasis fields. However, a few suggestions and recommendations for improving the further study in the fields are provided.

Autophagy is a key process by which cells degrade their components to maintain homeostasis. There are some previous studies reporting the role of autophagy and its regulation in skin diseases, including skin pigmentation and skin inflammation. Previously reported data suggested that modulation of autophagy or activation of aryl hydrocarbon receptors (AHRs) could affect processes involved in epidermal differentiation and contribute to the pathogenesis of chronic inflammatory skin diseases with skin barrier abnormalities such as psoriasis. This previous study had unraveled the role of autophagy contribution in improving psoriasis by treating the polycytokine-stimulated human keratinocytes and psoriasis skin biopsies tested in their in vitro and in vivo studies.[2]

In their study, Zhou et al reported that the induction of autophagy by rapamycin could improve the function of M5 cytokines-induced inflamed endothelial cells proven by a significant declined NO content and NOS activity in HUVECs compared with that in controls.[1] However, a negative control, a control treated with autophagy inhibitor, such as PI3K/mTOR inhibitor, was not shown or performed in this study. A comprehensive study which included both a positive and negative control could be used as more fundamental information regarding a novelty in a particular objective.[3] Therefore, an appropriate negative control is much recommended to be included in further study.

There are also other approaches to induce autophagy in in vitro and in vivo studies. The induction of autophagy by an autophagy inducer, Synthesized Aquatide^{TM}, has been recently reported to play an important role in maintaining overall health and skin health in animal model dogs with underlying diseases.[4] Therefore, an in vivo study is recommended to be performed as part of the further study.

Regardless, we appreciated the outcomes reported on the mechanism involved in this study, that autophagy could inhibit M5 cytokines-induced inflammation through the p38-MAPK/mTOR pathway in HUVEC cells. Their data confirmed and added more information on a previously reported study which stated that the inhibition of autophagy was associated with PI3K/AKT/mTOR and eNOS signaling pathways in HUVEC cells.[5]

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Author Contributions

Both authors have equal contribution in conceiving the critical design and thoughts written and discussed in the letter manuscript, writing and revising the manuscript. Both authors agreed the final form of the manuscript.

Disclosure

All authors stated that there is no conflict of interest regarding this communication.

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