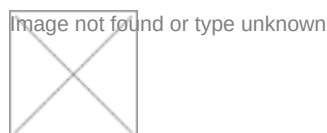


Altered redox signaling contributes to CCM pathogenesis



A recent research work published in **Scientific Reports** ^[1] and supported by the **Telethon Foundation** ^[2] demonstrate that altered redox signaling is a major contributor to increased endothelial permeability in experimental models of CCM disease, thus confirming and extending previous reports suggesting that oxidative stress plays an important role in CCM pathogenesis and severity.



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Up-regulation of NADPH oxidase-mediated redox signaling contributes to the loss of barrier function in KRIT1 deficient endothelium.

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[Abstract](#) ^[3]

[Full article](#) ^[4] ([PDF](#) ^[5])

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Int J Biochem Cell Biol. 2016 Dec;81(Pt B):254-270.

Oxidative stress and inflammation in cerebral cavernous malformation disease pathogenesis: Two sides of the same coin ^[6].

Retta SF, Glading AJ.

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Beyond multiple mechanisms and a unique drug: Defective autophagy as pivotal player in cerebral cavernous malformation pathogenesis and implications for targeted therapies [7].

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Defective autophagy is a key feature of cerebral cavernous malformations [10].

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Strategy for identifying repurposed drugs for the treatment of cerebral cavernous malformation [12].

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KRIT1 loss of function causes a ROS-dependent upregulation of c-Jun [13].

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Lingua Italiano

Source URL: <http://www.ccmitalia.unito.it/it/content/altered-redox-signaling-contributes-ccm-pathogenesis>

Links

- [1] <https://www.nature.com/srep/>
- [2] <http://www.telethon.it/en>
- [3] <https://www.ncbi.nlm.nih.gov/pubmed/28811547>
- [4] <https://www.nature.com/articles/s41598-017-08373-4>
- [5] <https://www.nature.com/articles/s41598-017-08373-4.pdf>
- [6] <https://www.ncbi.nlm.nih.gov/pubmed/27639680>
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- [10] <https://www.ncbi.nlm.nih.gov/pubmed/26417067>
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- [12] <https://www.ncbi.nlm.nih.gov/pubmed/25486933>
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