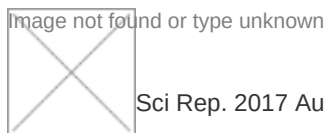


Nuovi risultati sperimentali



Un lavoro recente pubblicato sulla rivista *Scientific Reports* [1] e supportato dalla *Fondazione Telethon* [2] dimostra che alterazioni di meccanismi redox cellulari contribuiscono in maniera determinante all'aumento della permeabilità endoteliale in modelli sperimentali della malattia CCM, confermando ed estendendo in tal modo lavori precedenti che suggerivano che lo stress ossidativo svolge un ruolo importante nella patogenesi e nella severità di questa malattia.

Il lavoro è il frutto della collaborazione di due gruppi di ricerca italiani (S.F. Retta, Università di Torino, e L.Trabalzini, Università di Siena) con un gruppo americano (A.J.Glading, University of Rochester, NY).



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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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Marchi S, Trapani E, Corricelli M, Goitre L, Pinton P, Retta SF.

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Cytochrome P450 and matrix metalloproteinase genetic modifiers of disease severity in Cerebral Cavernous Malformation type 1 [9].

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

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Trapani E, Retta SF.

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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].

Goitre L, De Luca E, Braggion S, Trapani E, Guglielmotto M, Biasi F, Forni M, Moglia A, Trabalzini L, Retta SF.

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- [3] <https://www.ncbi.nlm.nih.gov/pubmed/28811547>
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- [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>
- [13] <https://www.ncbi.nlm.nih.gov/pubmed/24291398>
- [14] <http://www.ccmitalia.unito.it/it/etichette/aiacnews>