Nuovi risultati sperimentali

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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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Sci Rep. 2017 Aug 15;7(1):8296.

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

Goitre L, DiStefano PV, Moglia A, Nobiletti N, Baldini E, Trabalzini L, Keubel J, Trapani E, Shuvaev VV, Muzykantov VR, Sarelius IH, Retta SF & Glading AJ.

Abstract [3]

Full article [4] (PDF [5])

Articoli correlati:

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.

Rare Dis. 2016 Jan 25;4(1):e1142640.

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

[7].

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

Autophagy. 2016;12(2):424-5.

Cellular processes underlying cerebral cavernous malformations: Autophagy as another point of view [8].

Marchi S, Retta SF, Pinton P.

Free Radic Biol Med. 2016 Mar;92:100-9.

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

Choquet H, Trapani E, Goitre L, Trabalzini L, Akers A, Fontanella M, Hart BL, Morrison LA, Pawlikowska L, Kim H, Retta SF.

EMBO Mol Med. 2015 Nov;7(11):1403-17.

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

Marchi S, Corricelli M, Trapani E, Bravi L, Pittaro A, Delle Monache S, Ferroni L, Patergnani S, Missiroli S, Goitre L, Trabalzini L, Rimessi A, Giorgi C, Zavan B, Cassoni P, Dejana E, Retta SF, Pinton P.

J Neurosurg Sci. 2015 Sep;59(3):201-9.

Cerebral cavernous malformation (CCM) disease: from monogenic forms to genetic susceptibility factors [11].

Trapani E, Retta SF.

Circulation. 2015 Jan 20;131(3):289-99.

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

Gibson CC, Zhu W, Davis CT, Bowman-Kirigin JA, Chan AC, Ling J, Walker AE, Goitre L, Delle Monache S, Retta SF, Shiu YT, Grossmann AH, Thomas KR, Donato AJ, Lesniewski LA, Whitehead KJ, Li DY.

Free Radic Biol Med. 2014 Mar;68:134-47.

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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