CFTR Therapeutics

Heart failure (HF) and subarachnoid hemorrhage (SAH) chronically reduce cerebral perfusion, which negatively affects clinical outcome. A work of Lidington et al. demonstrates a strong relationship between cerebral artery cystic fibrosis transmembrane conductance regulator (CFTR) expression and altered cerebrovascular reactivity in HF and SAH. In HF and SAH, CFTR corrector compounds (C18 or lumacaftor) normalize pathological alterations in cerebral artery CFTR expression, vascular reactivity, and cerebral perfusion, without affecting systemic hemodynamic parameters. This normalization correlates with reduced neuronal injury. Therefore, CFTR therapeutics have emerged as valuable clinical tools to manage cerebrovascular dysfunction, impaired cerebral perfusion, and neuronal injury [1].

1)
Lidington D, Fares JC, Uhl FE, Dinh DD, Kroetsch JT, Sauvé M, Malik FA, Matthes F, Vanherle L, Adel A, Momen A, Zhang H, Aschar-Sobbi R, Foltz WD, Wan H, Sumiyoshi M, Macdonald RL, Husain M, Backx PH, Heximer SP, Meissner A, Bolz SS. CFTR Therapeutics Normalize Cerebral Perfusion Deficits in Mouse Models of Heart Failure and Subarachnoid Hemorrhage. JACC Basic Transl Sci. 2019 Nov 27;4(8):940-958. doi: 10.1016/j.jacbts.2019.07.004. eCollection 2019 Dec. PubMed PMID: 31909302; PubMed Central PMCID: PMC6939007.

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