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域、中央的 Rho 结合的卷曲螺旋结构域和羧基端 pH 结构域。
在静息状

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还可通过介导缺氧或凝血酶引起的内皮型 NOS (endo helial NOS,eNOS)表达下调^[21],使 NO 扩张血管增加脑缺血区血流量、抑制血小板聚集、抑制小血管缺血后炎症反应的脑保护作用下降。有研究发现大鼠大脑中动脉阻塞后,出现三磷酸腺苷(ATP)耗竭,缺血造成细胞内钙离子增多,引发级联反应,与迟发性细胞死亡或凋亡有关,应用 Fausdil 在各时段增加局部脑血流量,可延缓缺血脑组织进入不可逆的细胞死亡过程,体现脑保护作用^[22]。Rikitake 等^[23]在大脑中动脉闭塞大鼠中应用不同剂量 Fasudil 抑制 Rho 激酶活性,结果显示,内皮型一氧化氮酶 mRNA 及其蛋白表达随 Fausdil 浓度的增加而增加,相应的 eNOS 活性和 NO 含量也增加,从而使脑梗死体积较未治疗组缩小 33 %,神经功能评分提高 37 %。而在 eNOS 缺陷小鼠中未能观察到神经保护作用。



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