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it can be shown that the activation of wnt/-catenin signaling with the specific gsk-3 inhibitor, ar-ao14418 (ar, [38, 42, 55]), is effective in protecting da neurons against neurotoxin-induced degeneration in vivo. the protection of da neurons from neurotoxin-induced degeneration by a pharmacological modulation of wnt/-catenin signaling with ar-ao14418 is correlated with a restoration of the neuroprotective phenotype in astrocytes in vitro and in vivo. the restoration of the neuroprotective phenotype in astrocytes in vitro is correlated with a restoration of the neuroprotective phenotype in da neurons in vivo. these results show that astrocytes are capable of switching from a neuroprotective to a neurodegenerative phenotype and back upon activation of wnt/-catenin signaling. thus, the da neurodegeneration can be reverted by modulating the wnt/-catenin signaling pathway (figure 13). the current study shows that wnt-1 exerts robust anti-inflammatory actions in the snpc astrocytes, which include a decrease in the release of two important proinflammatory cytokines, tnf-a and il-1b, and an increase in the release of anti-inflammatory il-10. interestingly, stimulation of cells with wnt-1 also leads to the activation of the non-canonical wnt pathway as evidenced by the increase in the nuclear translocation of β-catenin and the transcriptional activation of tcf/lef-dependent genes. importantly, the anti-inflammatory effects of wnt-1 were abolished in astrocytes from snpc dkk1 mice suggesting that these effects are mediated by the wnt-1-induced activation of non-canonical wnt signaling in snpc astrocytes. interestingly, the increase in wnt-1 levels in snpc astrocytes was accompanied by a significant decrease in the levels of inflammation-related factors, tnf-a and il-1b, and an increase in the anti-inflammatory factor, il-10. these results were confirmed in astrocytes from snpc dkk1 mice suggesting that non-canonical wnt signaling in astrocytes is required for the anti-inflammatory actions of wnt-1. of great interest, the overexpression of wnt-1 in astrocytes of the snpc by aav-wnt-1 delivery significantly reduced the levels of tnf-a, il-1b and increased the level of il-10 in the snpc. in addition, a single intranasal administration of wnt-1 to 6-ohda lesioned mice significantly increased the levels of wnt-1 in the snpc and significantly decreased the levels of tnf-a, il-1b and increased the level of il-10 in the snpc (figure 14). these results suggest that wnt-1 can directly act on astrocytes to decrease the production of pro-inflammatory cytokines and increase the level of the anti-inflammatory cytokine in the snpc. collectively, these data indicate that wnt-1 can directly act on astrocytes to decrease the production of pro-inflammatory cytokines and increase the level of the anti-inflammatory cytokine in the snpc.

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antithrombotic therapy is a well-recognized strategy to prevent and manage acute coronary syndromes (acs). indeed, data from clinical trials have shown that the addition of new oral antithrombotics to aspirin therapy has improved cardiovascular outcomes in acs. however, the administration of antiplatelet therapies (e.g. cilostazol, sarpogrelate, or prasugrel) is associated with an increased risk of bleeding, which increases the burden of antithrombotics in patients with acs. we reviewed the effect of platelet membrane protein gp iib/iiia receptor blockade on endothelial function and vascular remodeling after pci with or without cilostazol therapy in patients with st-segment elevation acs (stemi). this review outlines the potential and main challenges of the clinical use of cilostazol in stemi patients receiving platelet inhibition. cardiac transplantation is the definitive treatment for end-stage heart failure, although long-term survival remains suboptimal due to the persistent need for immunosuppression and its toxic side effects. the cumulative risk for cardiovascular complications and graft loss in heart transplant recipients is high and is the main cause of mortality. to assess the de-differentiation of mpp+ daergic neurons, their neurite outgrowth, glial scar formation, astrocytic activation and reactive microgliosis were analyzed by in vivo immuohistochemistry, and compared with the canonical wnt-/-catenin pathway activation. in snpc, cortical neurons were transiently transfected with 4 μg plasmid pda-egfp (1 μg/μl), expressing constitutively the enhanced green fluorescent protein (egfp), 3 days before cell transplantation. sections double-stained with egfp and tyrosine hydroxylase (th, the rate-limiting enzyme for da synthesis) were obtained from anaesthetized mice at 7 days post-transfection. results showed that egfp-positive cortical neurons were efficient to survive and extend processes in the snpc of the dkk1 group, while egfp-labeled cells appeared necrotic in the control group (figure 8a). 5ec8ef588b

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