**A Dipeptidyl Peptidase-4 Inhibitor, Alogliptin, Attenuates Arterial Inflammation and Neointimal Formation After Injury**

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**Background:** Recent studies suggest dipeptidyl-peptidase 4 (DPP-4) inhibitor attenuates inflammation and possibly reduces atherosclerosis. However, it remains undetermined whether a DPP-4 inhibitor suppresses arterial inflammation and intimal hyperplasia after injury. The aim of this study was to investigate the anti-inflammatory effects of the DPP-4 inhibitor, alogliptin (AGP), in injured artery.

**Methods:** We investigated intimal hyperplasia in LDL receptor-deficient (LDLr-/-) mice (that could be induced more neointimal formation compared with wild-type mice) two weeks after inducing femoral artery injury with an external vascular cuff model. All mice received oral injection of AGP (20mg/kg/day) or normal saline once daily for 14 days.

**Results:** AGP treatment yielded no adverse systemic effects, and we observed no significant differences in fasting blood sugar levels, serum cholesterol levels, or blood pressure in either group. Compared to saline treatment (n=8), however, AGP treatment (n=9) significantly reduced intimal hyperplasia (1,087 ± 127 vs. 1,896 ± 140 μm²; P <0.001) and intima/media ratio (0.08 ± 0.01 vs. 0.16 ± 0.02; P<0.001). Immunostaining revealed AGP treatment reduced proliferating cells (PCNA positive nuclei) (P<0.01) and suppressed smooth muscle cell proliferation (α-SMA positive cells) (P=0.029) compared to control group. Furthermore, Immunostaining for Mac3 and Ly6G revealed significant fewer inflammatory cells in the neointima of AGP treated mice compared with controls (P<0.05 and P<0.01, respectively). These results indicated that AGP decreased inflammation and intimal hyperplasia in injured arteries of LDLr-/- mice. Importantly, we also detected similar findings using wild-type mice with (n=10) or without AGP (n=10).

**Conclusion:** Our data suggest that DPP-4 inhibitor, AGP, suppresses neointimal formation by inhibiting inflammation without relation to its effects for glucose or cholesterol metabolisms in both atherogenic and wild-type mice. This study highlights the potential therapeutic benefit of AGP therapy in preventing intimal hyperplasia and arterial inflammation.