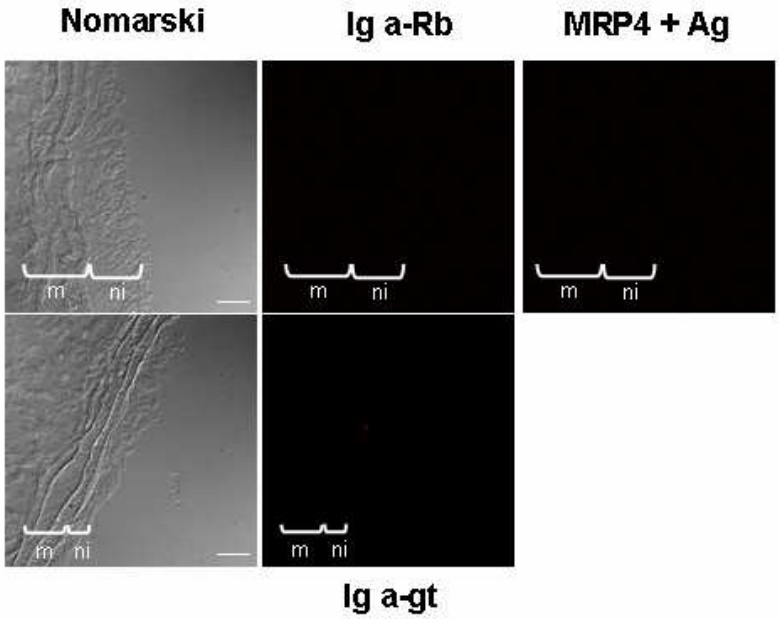
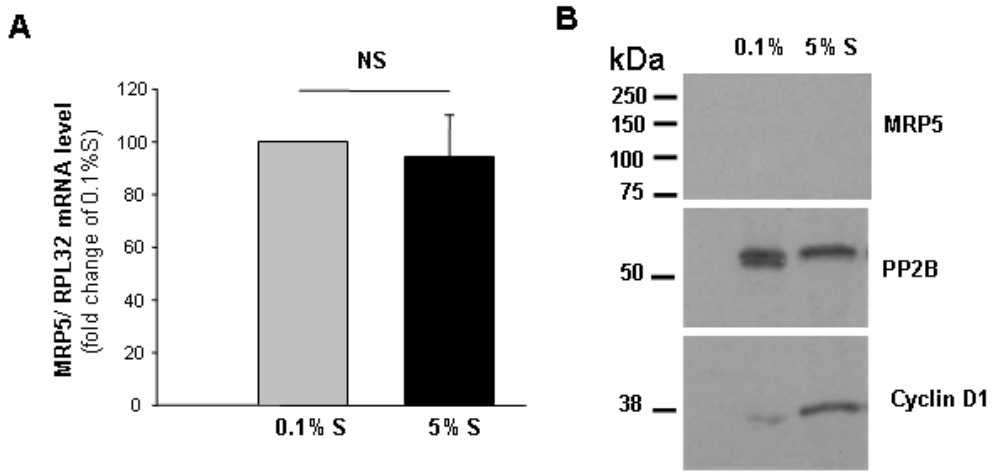
Supplemental Figure 1: Negative controls of immunofluorescence on coronary arteries. Proteins were incubated with the anti-species immunoglobulins alone (anti-rabbit-alexa-546 or anti-goat-alexa-546). No signal was observed in the absence of the primary antibodies. Furthermore, MRP4 signal was abolished by pre-incubation with the antigen. Scale bar: 50µm.

Supplemental Figure 2 : **(A)** MRP5 level quantified by quantitative RT-PCR in cultured hCASMC exposed for 72 hours to 5% or 0.1% S normalized to the value obtained in 0.1% S. (p= NS).**(B)** Western-blot analysis of MRP5 protein expression in the same conditions. PP2B was used as a loading control and cyclin D1 as a marker of proliferation.

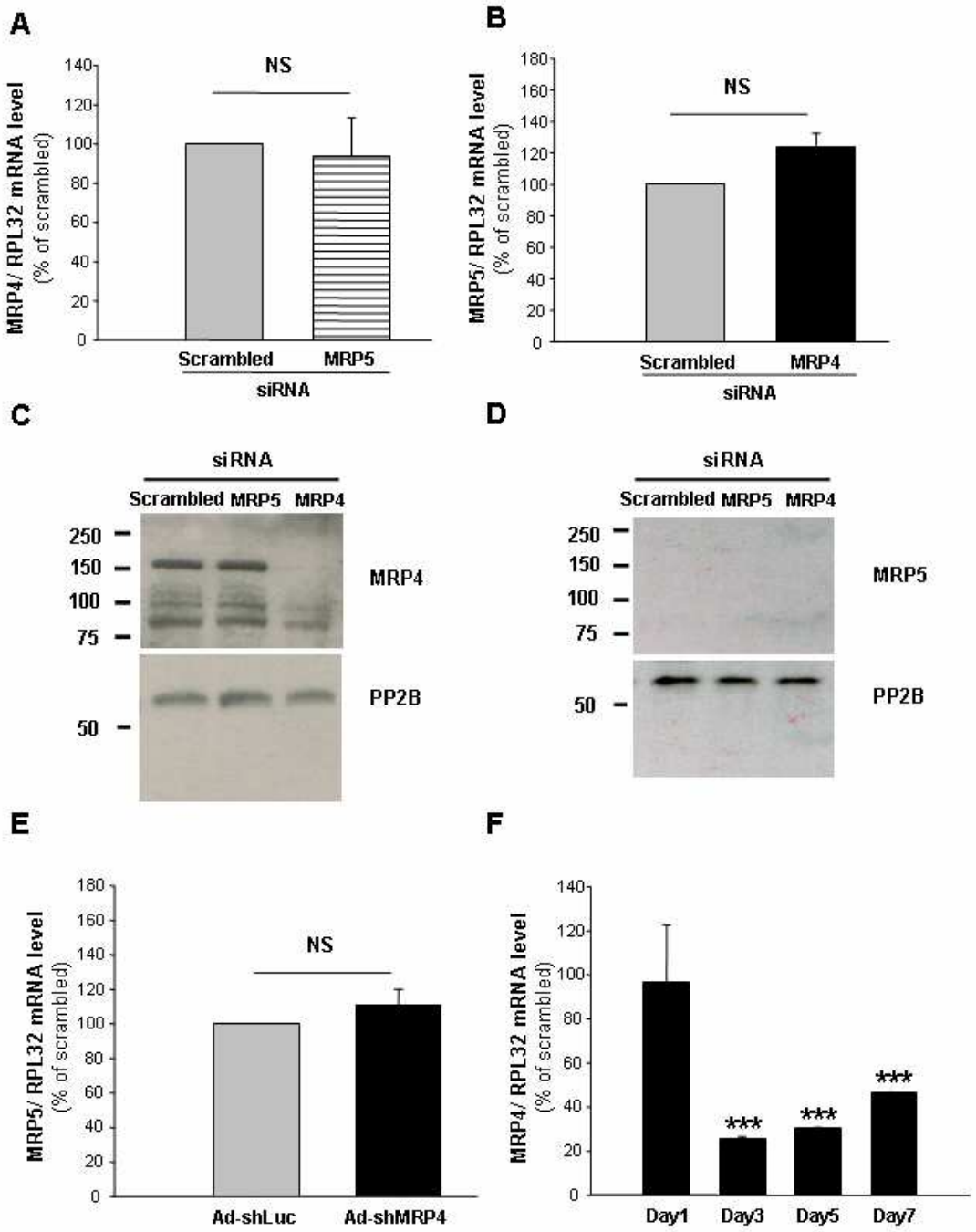
Supplemental Figure 3 : Quantitative real-time PCR with gene-specific primers of **(A)** MRP4 and **(B)** MRP5 in hCASMC transfected for 72h with MRP4, MRP5 or scrambled siRNA showing the selectivity of MRP4 or MRP5 siRNA. **(C,D)** Western-Blot analysis of total lysates of hCASMC transfected for 72 h with MRP4, MRP5 or scrambled siRNA. Proteins were incubated with anti-MRP4, anti-MRP5 or anti-PP2B antibodies. PP2B was used as a loading control. **(E)** Quantitative real-time PCR with gene-specific primers of MRP5 in rat smooth muscle cells infected for 72 h with Ad-shMRP4 or Ad-shLuc (n=3, p= NS). Viruses were used at a multiplicity of infection (MOI) of 30. **(F)** Quantitative real-time PCR with gene-specific primers of MRP4 in hCASMC transfected for 1, 3, 5 or 7 days with MRP4 siRNA (n=3, *** $p<0.001$).



Supplemental Fig. 1



Supplemental Fig. 2



Supplemental Fig. 3