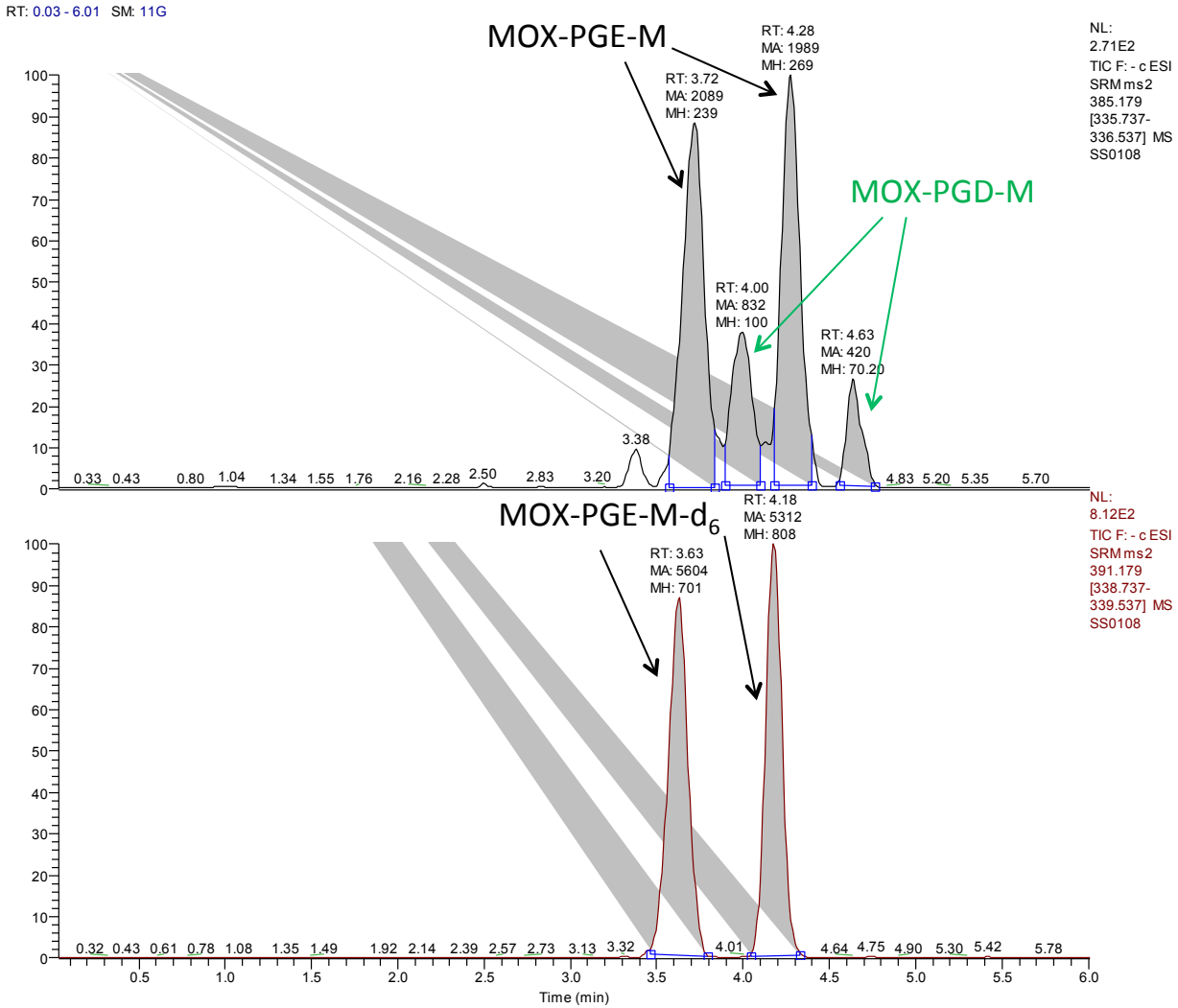
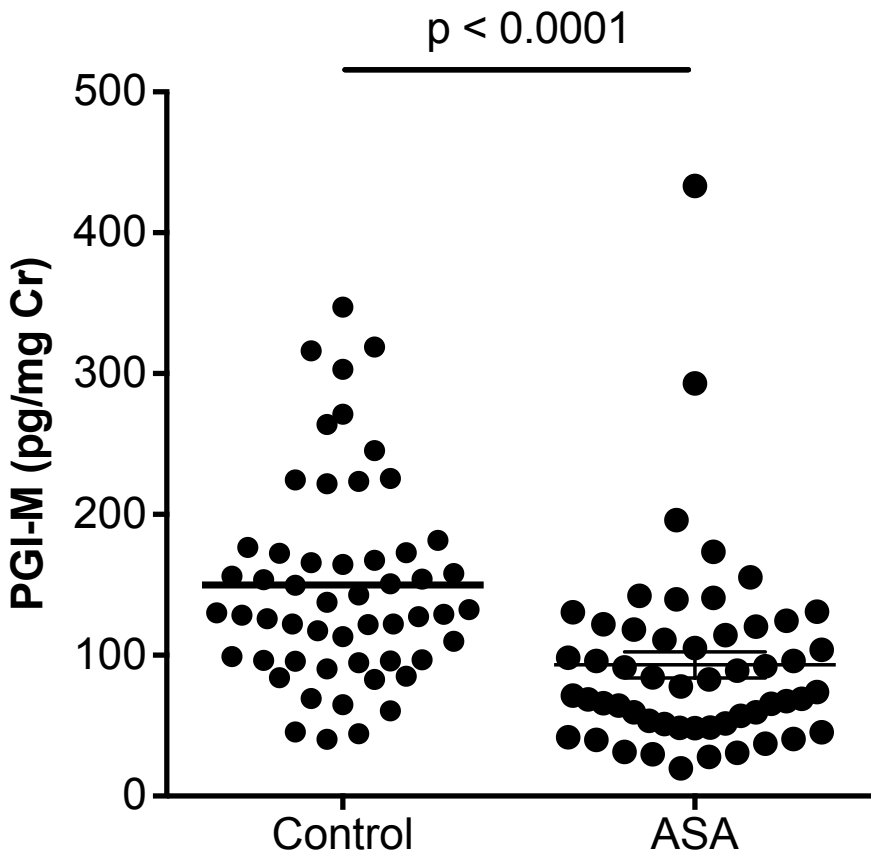
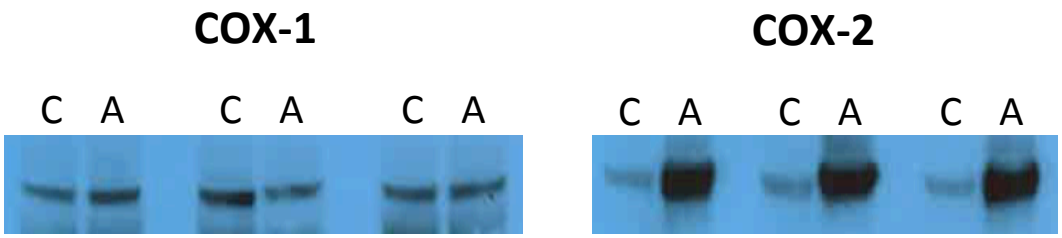


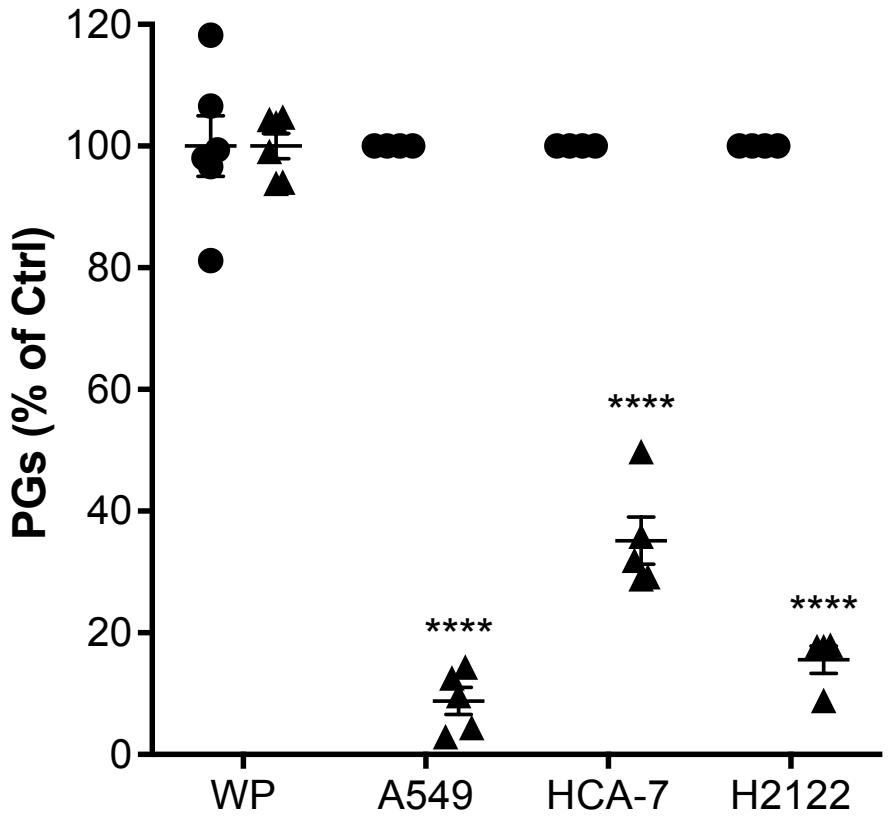
Supplementary Figure S1. LC/MS chromatograms of MOX-PGE-M, MOX-PGD-M and the internal standard MOX-PGE-M-d_6. MOX-PGE-M is eluted 6 seconds later than its internal standard. As can be seen in the upper panel, the two peaks representing MOX-PGD-M are eluting 17 seconds after those of MOX-PGE-M in this chromatographic system.



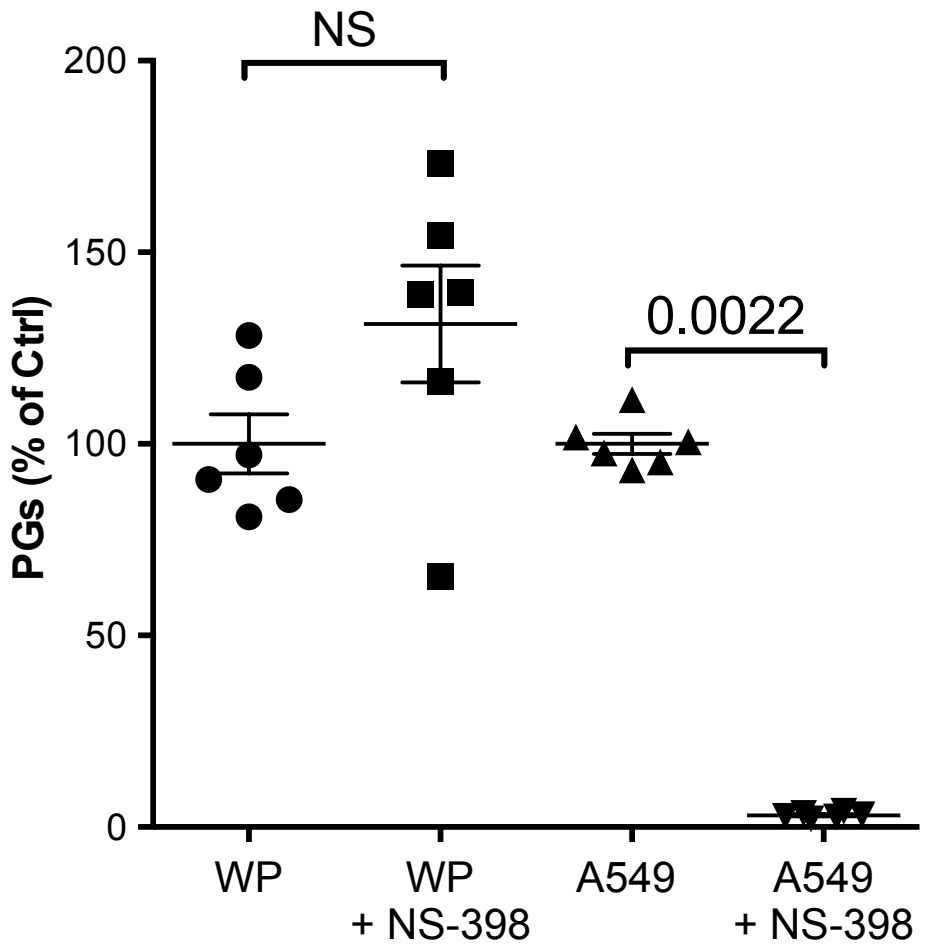
Supplemental Fig S2. Daily low dose aspirin inhibits prostacyclin biosynthesis in humans. 81 mg of aspirin was administered daily to healthy participants for 2 weeks. PGI-M, the urinary metabolite of prostacyclin, was measured by GC/MS before and after aspirin therapy. Paired analysis comparisons were performed using Wilcoxon-ranked tests. Low dose aspirin inhibited the biosynthesis of prostacyclin by 37% ($p<0.0001$, $n=64$).



Supplemental Fig S3. Western blots analysis of COX-1 and COX-2 expression in A549 cells before and after cell activation with IL-1b 1 ng/ml. n = 3



Supplemental Fig S4. Prostanoid inhibition by the COX-2 specific inhibitor valdecoxib. Washed platelets (WP), A549 and H2122 lung adenocarcinoma or HCA-7 colon adenocarcinoma cells were preincubated for 30 min with 100 nM valdecoxib (triangle) or vehicle (circle) prior addition of 2 µM arachidonic acid. After 15 min, prostanoid production was measured by GC/MS. Values represent means ± S.E.M., comparison between treatment and vehicle was performed using Holm-Sidak method (****, $P \leq 0.0001$ vs vehicle, $n = 4$). Valdecoxib effect is non significant in washed platelets (WP).



Supplemental Fig S5. Prostanoid inhibition by the COX-2 specific inhibitor NS-398. Cells were preincubated for 30 min with 100 nM NS-398 or vehicle prior addition of 2 µM arachidonic acid. After 15 min, prostanoid production was measured by GC/MS. Values represent means ± S.E.M., comparison between treatment and vehicle was performed using t-test.