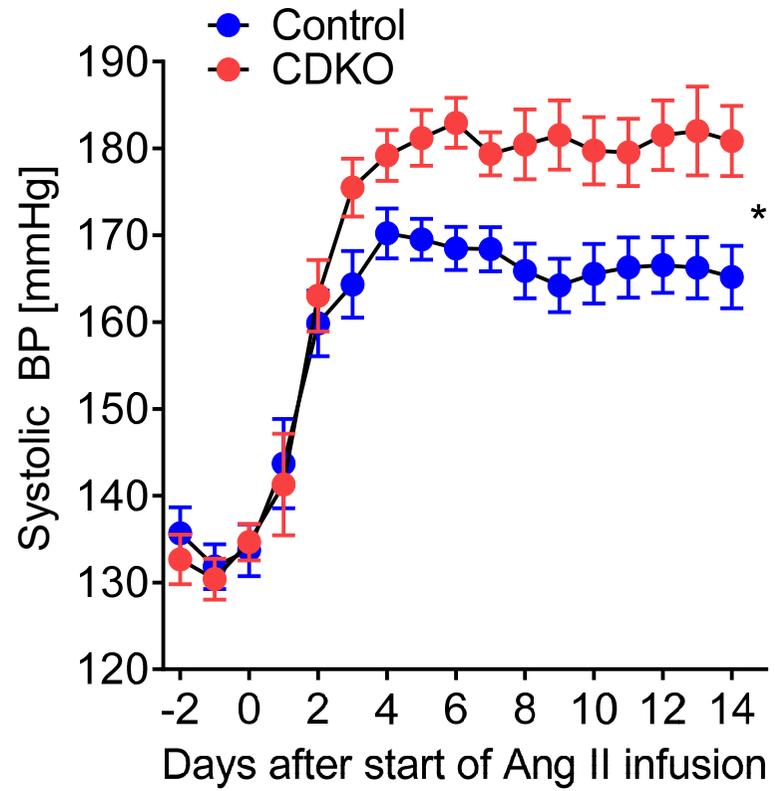
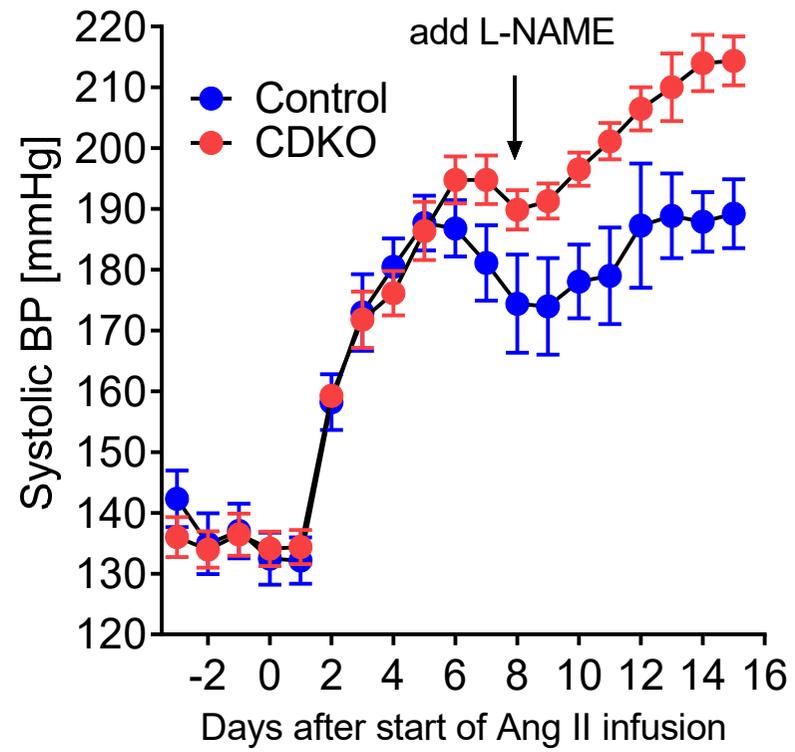


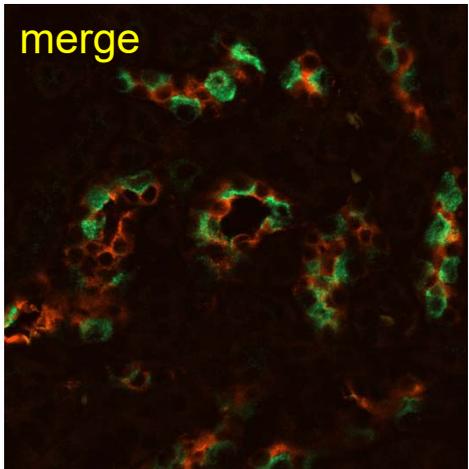
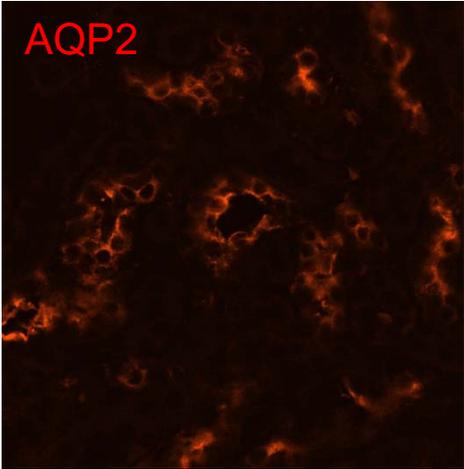
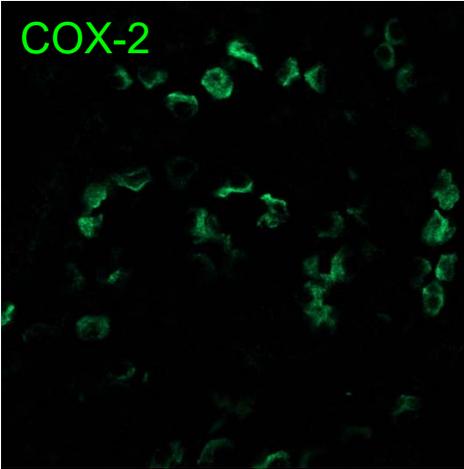
Supplementary Figure 1



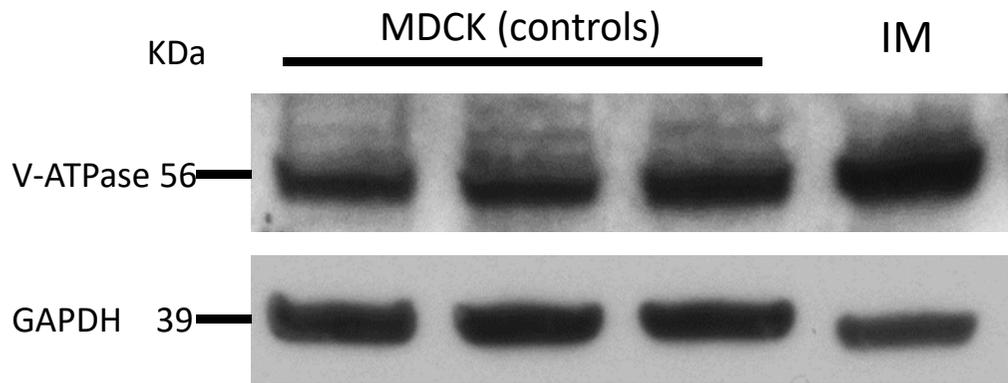
Supplementary Figure 2



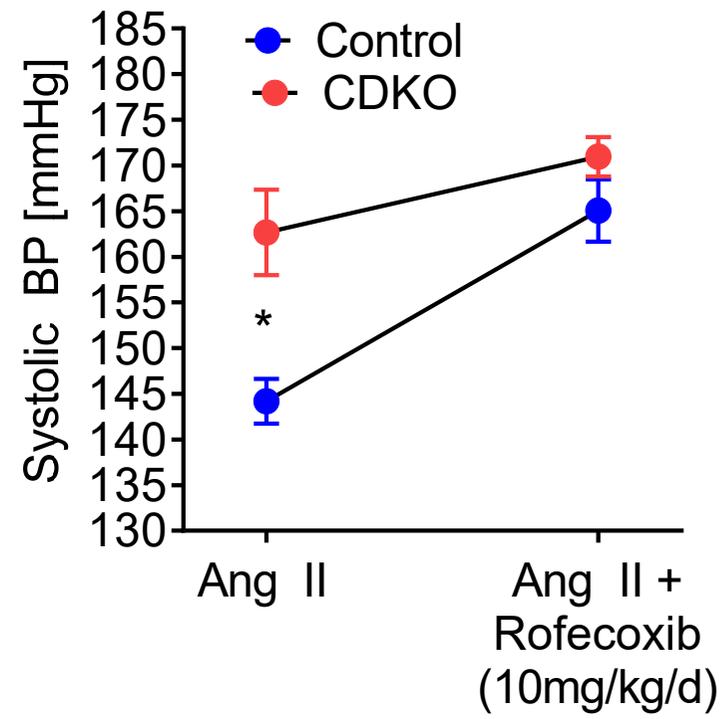
Supplementary Figure 3



Supplementary Figure 4

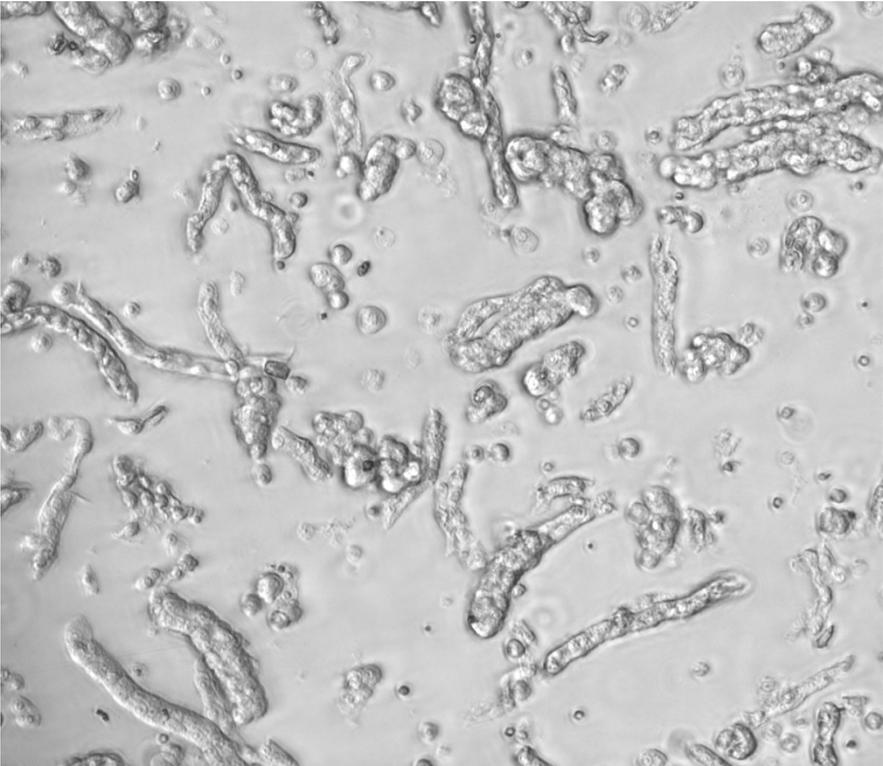


Supplementary Figure 5



Supplementary Figure 6

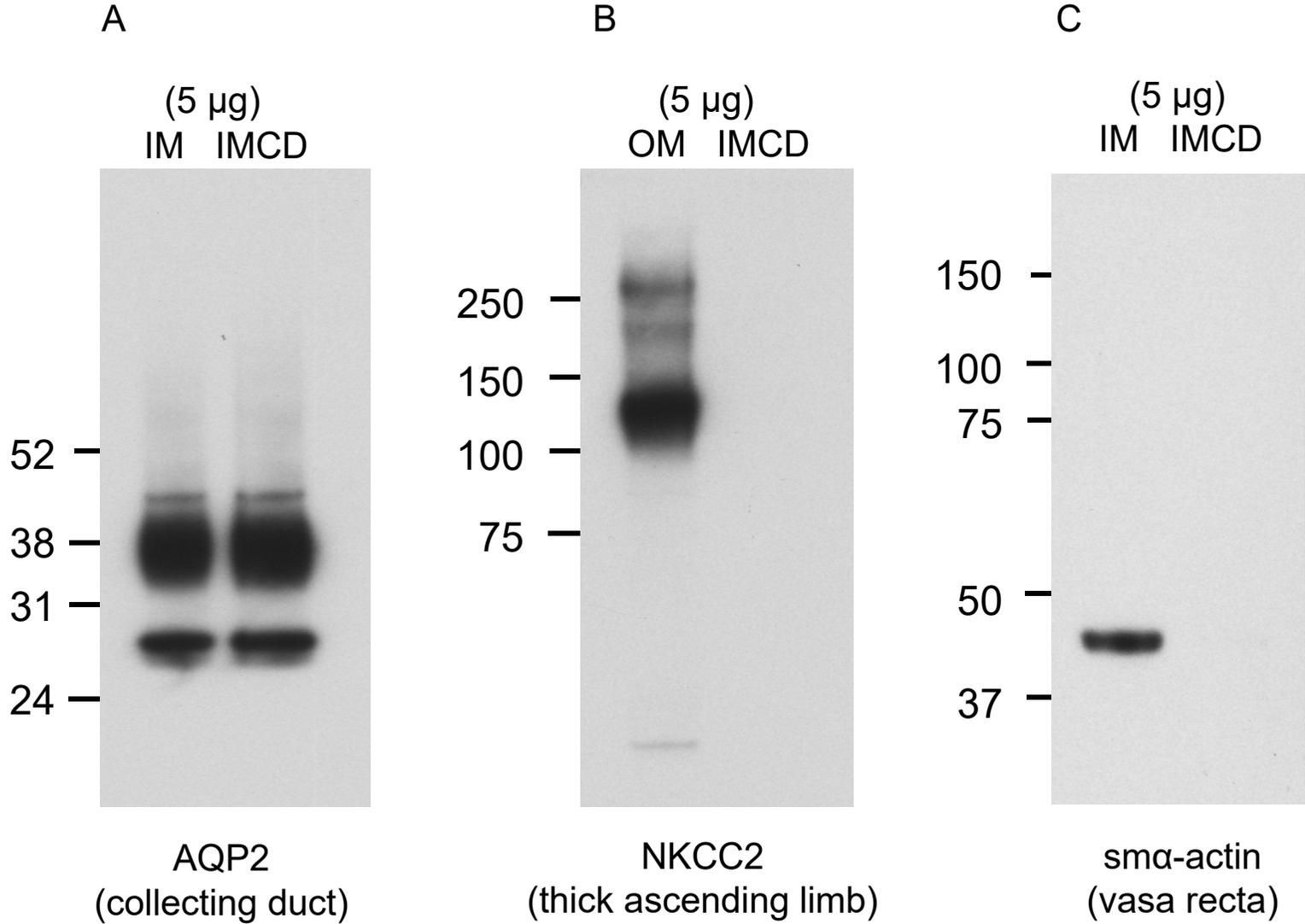
A



B



Supplementary Figure 7



Supplementary Figure Legends

Supplementary Figure 1. Exaggerated blood pressure response to angiotensin II in mice lacking AT_{1A} receptors from the collecting duct. Chronic angiotensin II infusion (1000ng/kg/min) caused a greater hypertensive blood pressure response in CDKO compared to control mice (systolic BP: 180 ± 3 vs. 166 ± 2 mmHg, * $p < 0.001$; $n=16$). ANOVA followed by Bonferroni's multiple comparison post-hoc test was used to compare the differences between the groups.

Supplementary Figure 2. Nitric oxide does not modify the exaggerated blood pressure response to angiotensin II in CDKO mice. The exaggerated blood pressure response to angiotensin II is not due to impaired NO generation as chronic administration of L-NAME (20mg/kg/day) did not decrease the systolic blood pressure difference between CDKO and control mice ($n=6$). ANOVA followed by Bonferroni's multiple comparison post-hoc test was used to compare the differences between the groups.

Supplementary Figure 3. Expression of AQP2 and COX-2 in the collecting duct. In angiotensin II treated control mice, AQP2 a specific marker for principal cells within the collecting duct did not co-localized with COX-2 (original magnification, $\times 20$).

Supplementary Figure 4. Representative western blots showing robust V-ATPase expression, a marker for intercalated cells in MDCK C-11 cells.

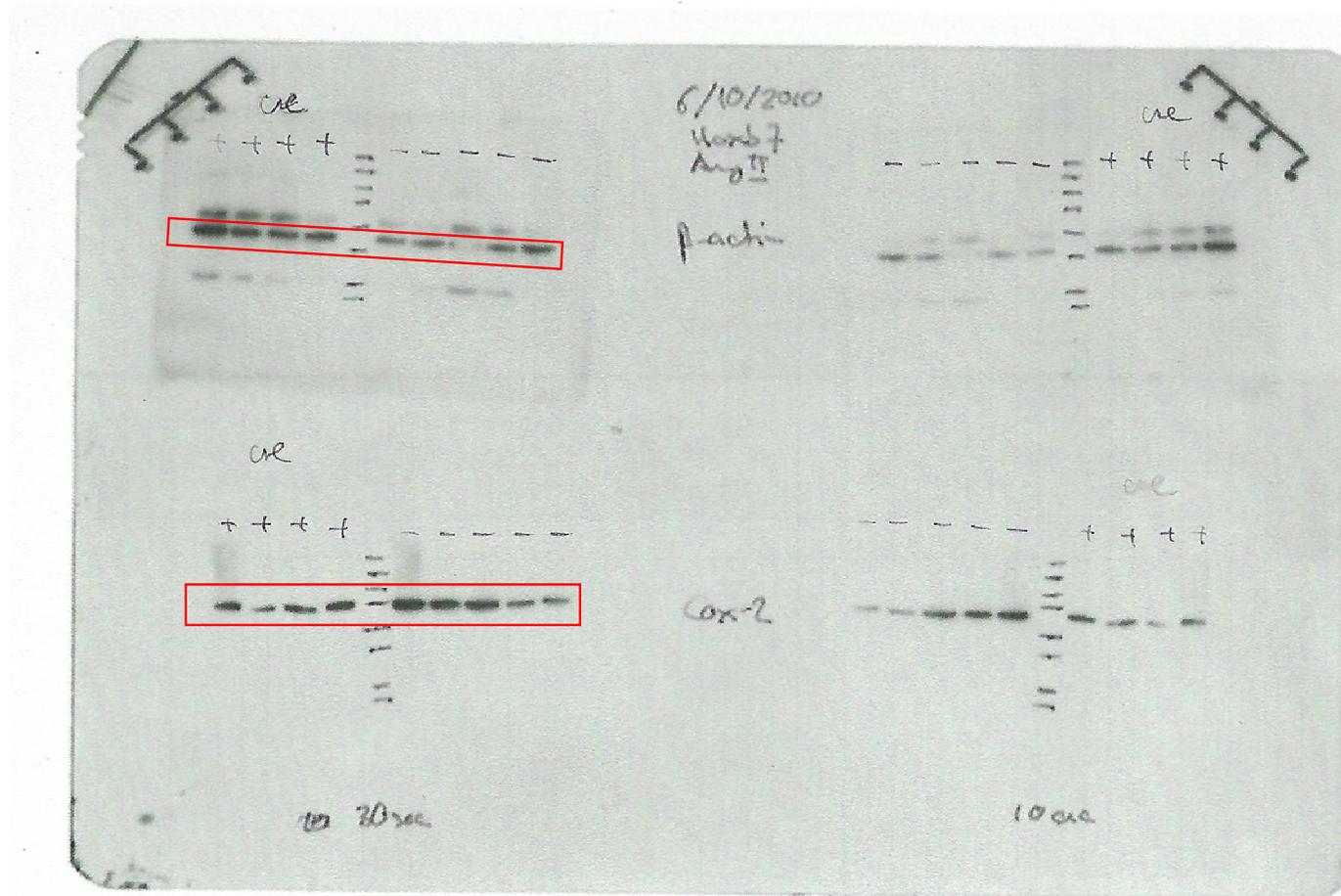
Supplementary Figure 5. Effects of COX-2 inhibition on angiotensin II-dependent hypertension. After one week of chronic angiotensin II treatment, CDKO and control mice were co-treated with a specific COX-2 inhibitor (rofecoxib 10mg/kg/day). After one week of

angiotensin II infusion, systolic BP was significantly higher in CDKOs compared to controls (162 ± 4 vs. 144 ± 2 mmHg; $*p<0.05$; $n=10$). Co-treatment with rofecoxib (10mg/kg) caused a significant increase in systolic blood pressures in both groups and abolished the systolic blood pressure difference between CDKO and control mice (171 ± 2 vs. 165 ± 3 mmHg; $n=10$).

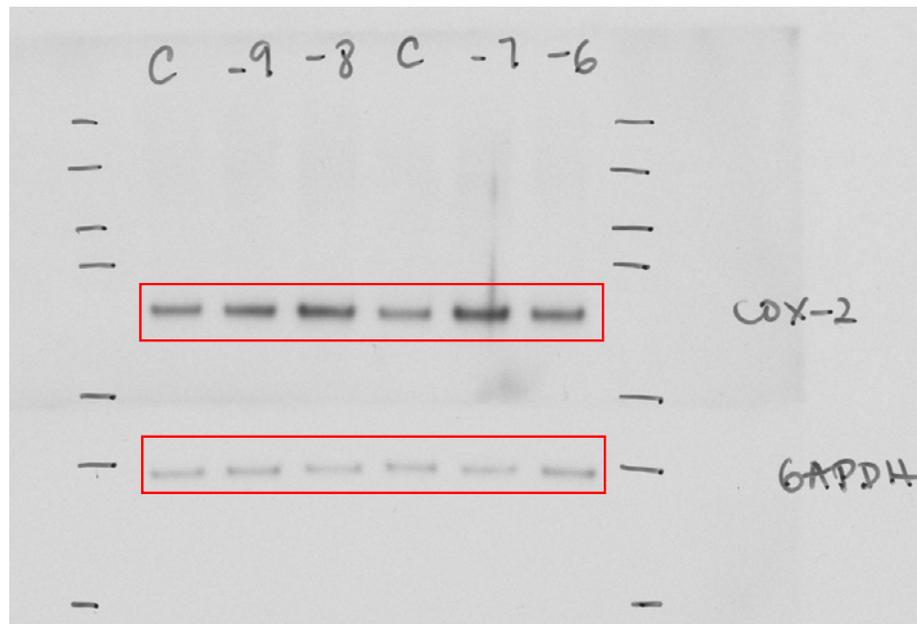
Supplementary Figure 6. Representative transmitted light images [(A)10 x and (B)40 x] of a mouse inner medullary collecting duct (IMCD) suspension.

Supplementary Figure 7. Detection of specific markers in mouse inner medullary collecting duct suspensions by western blotting. (A) aquaporin 2, specific for collecting ducts; (B) NKCC2, only expressed in thick ascending limbs; and (C) α -actin, specific marker of vascular structures. ANOVA followed by Bonferroni's multiple comparison post-hoc test was used to compare the differences between the groups.

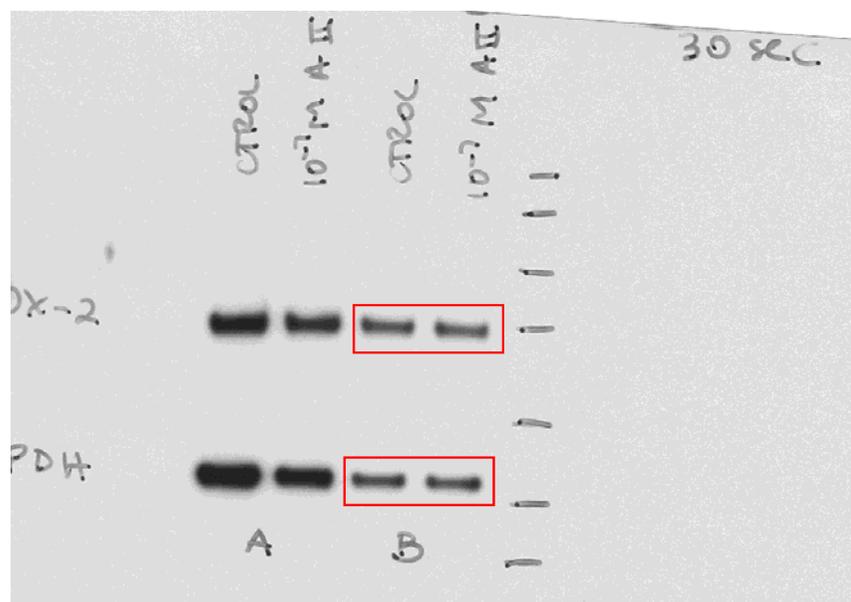
Full unedited gel for Figure 4B:



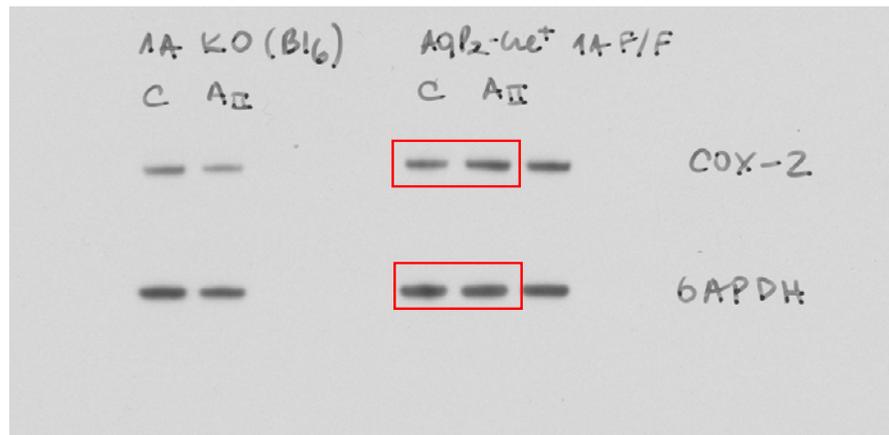
Full unedited gel for Figure 5A:



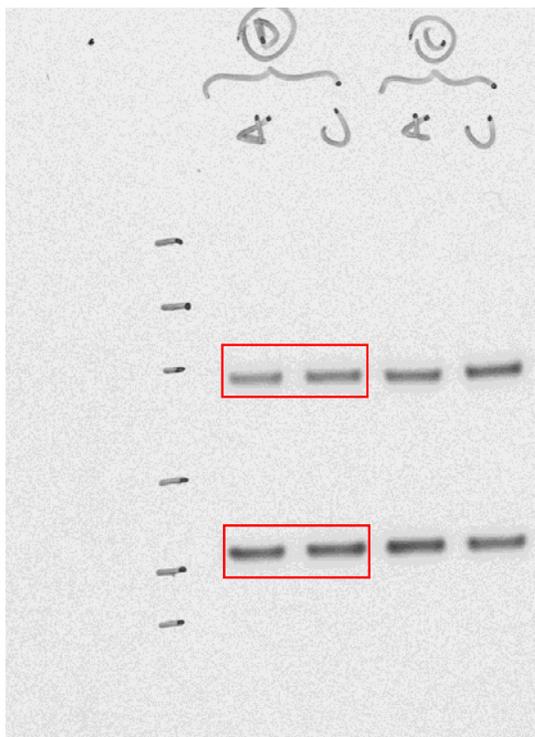
Full unedited gel for Figure 5B:



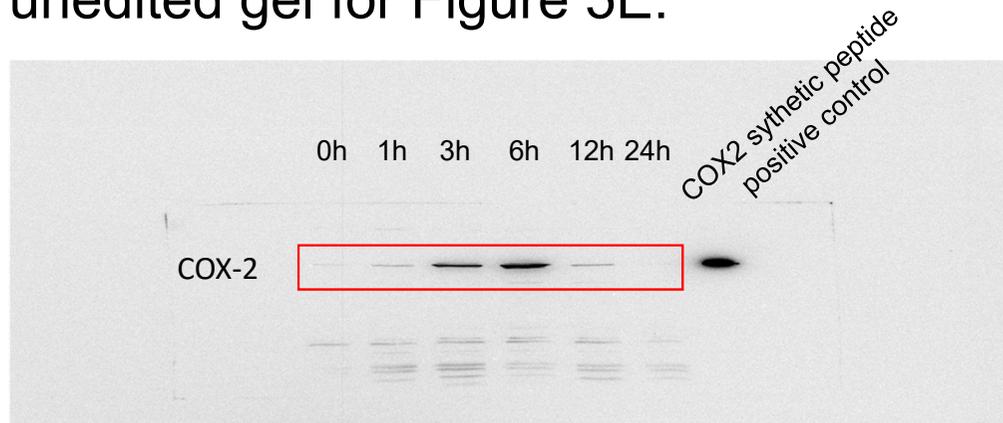
Full unedited gel for Figure 5C:



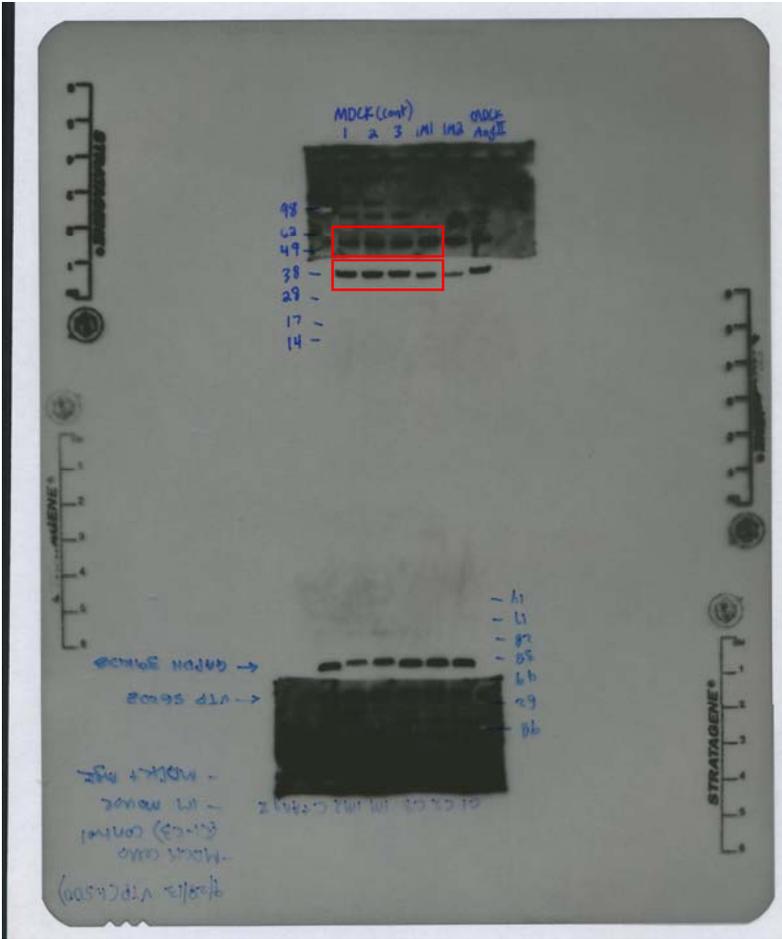
Full unedited gel for Figure 5D:



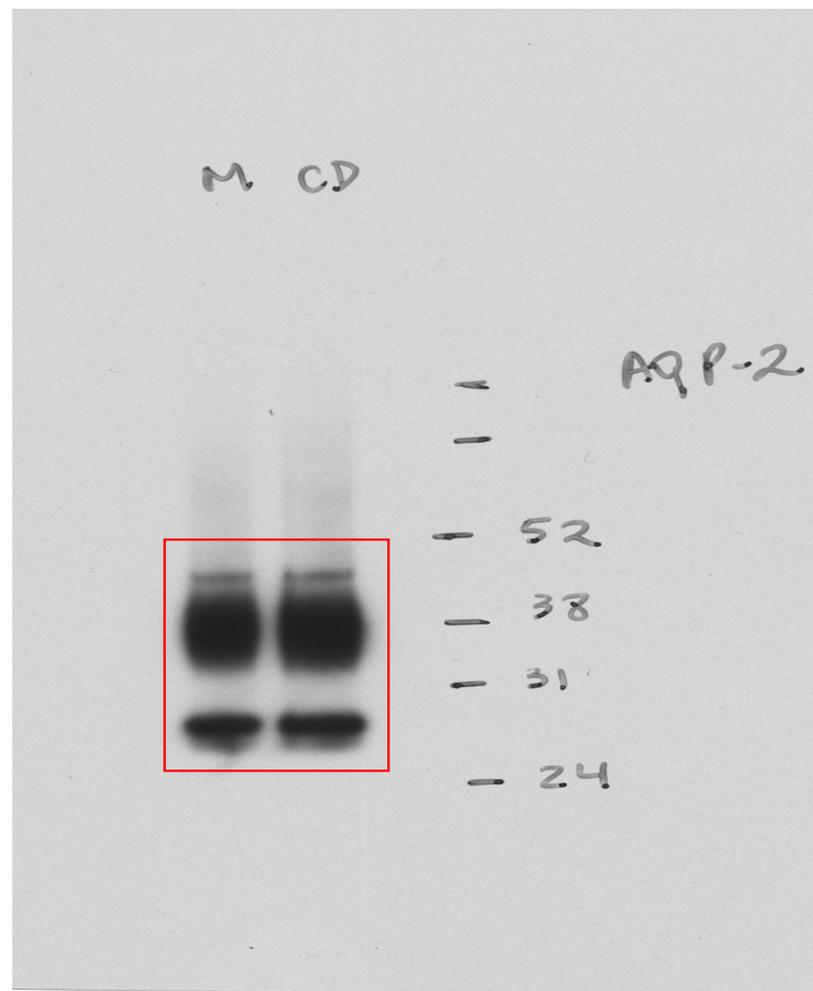
Full unedited gel for Figure 5E:



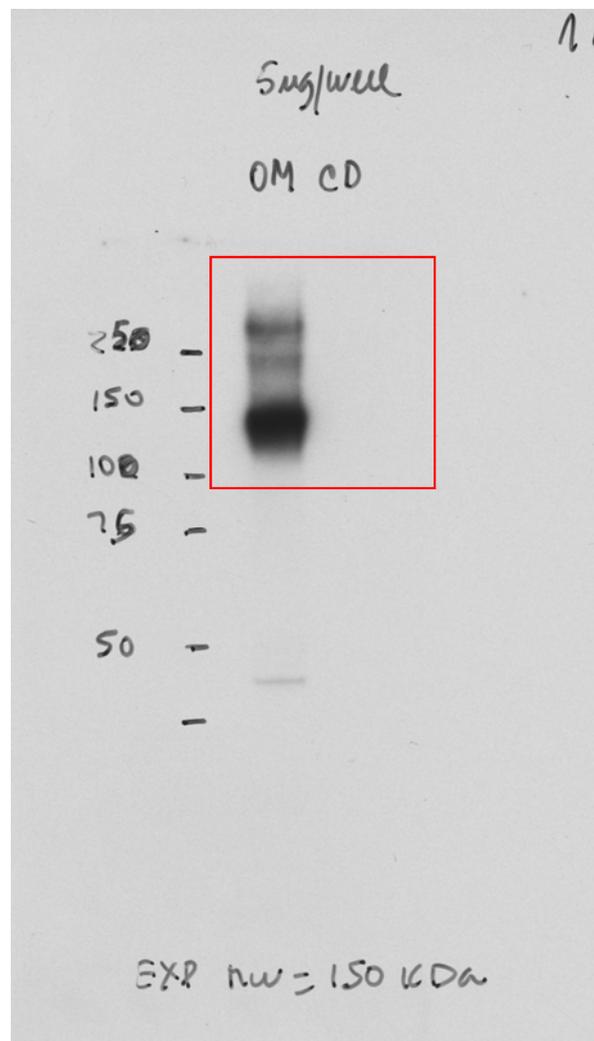
Full unedited gel for Supplementary Figure 4:



Full unedited gel for Supplementary Figure 5A:



Full unedited gel for Supplementary Figure 5B:



Full unedited gel for Supplementary Figure 5C:

