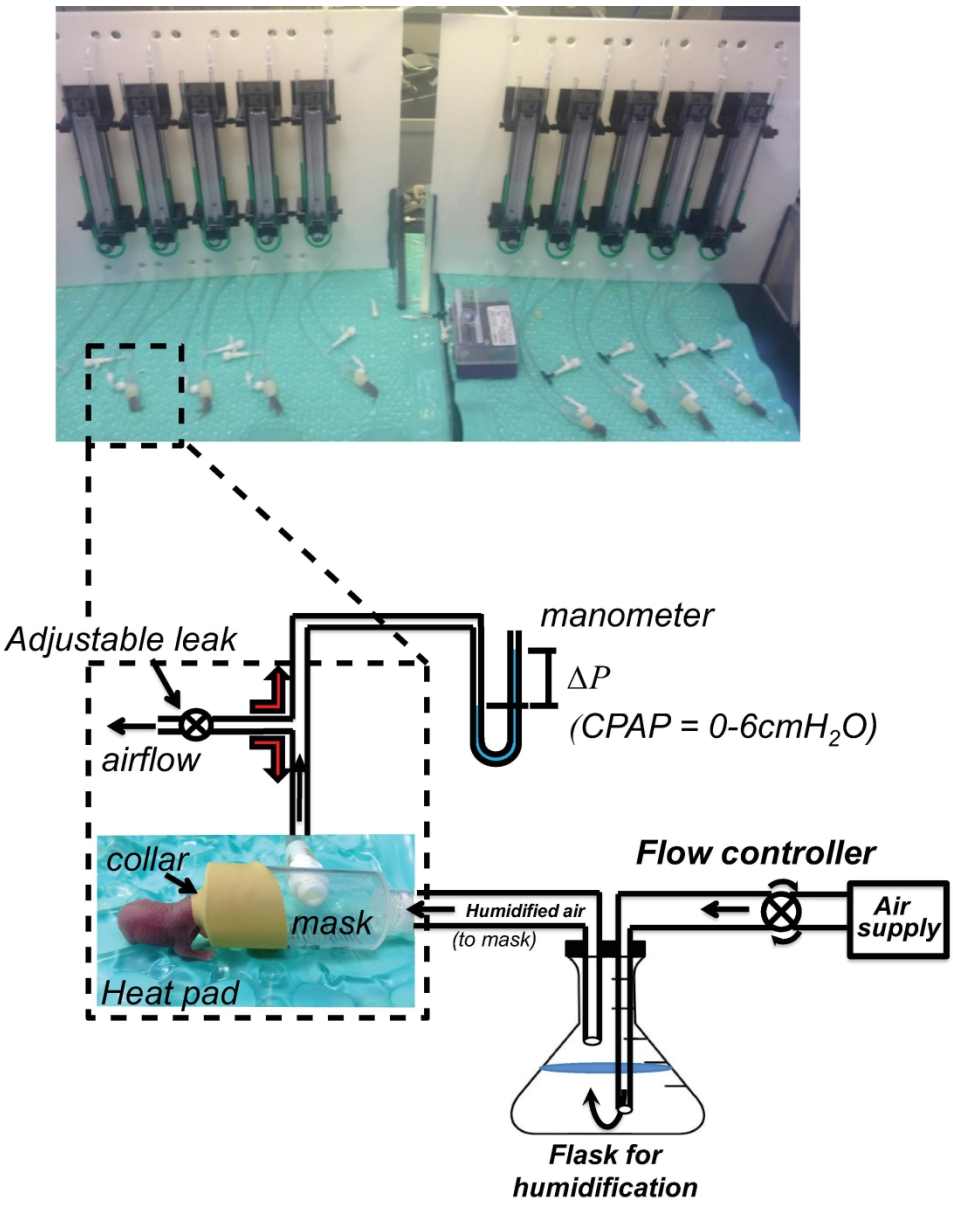
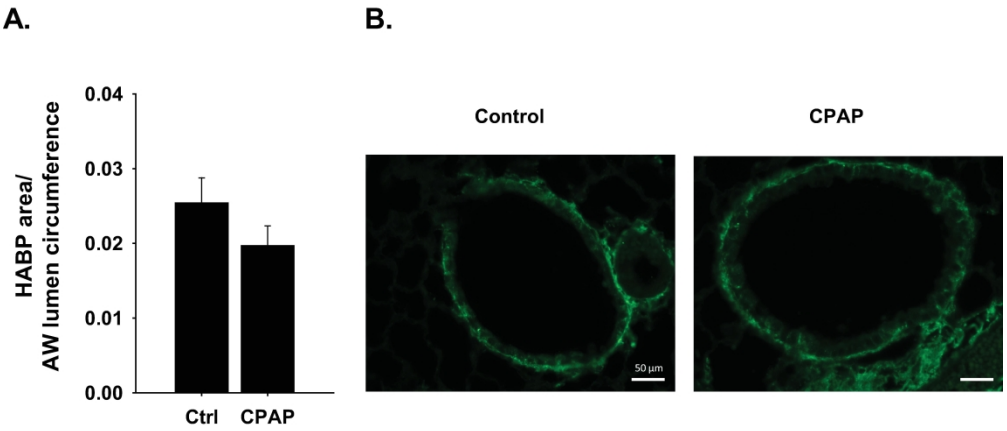


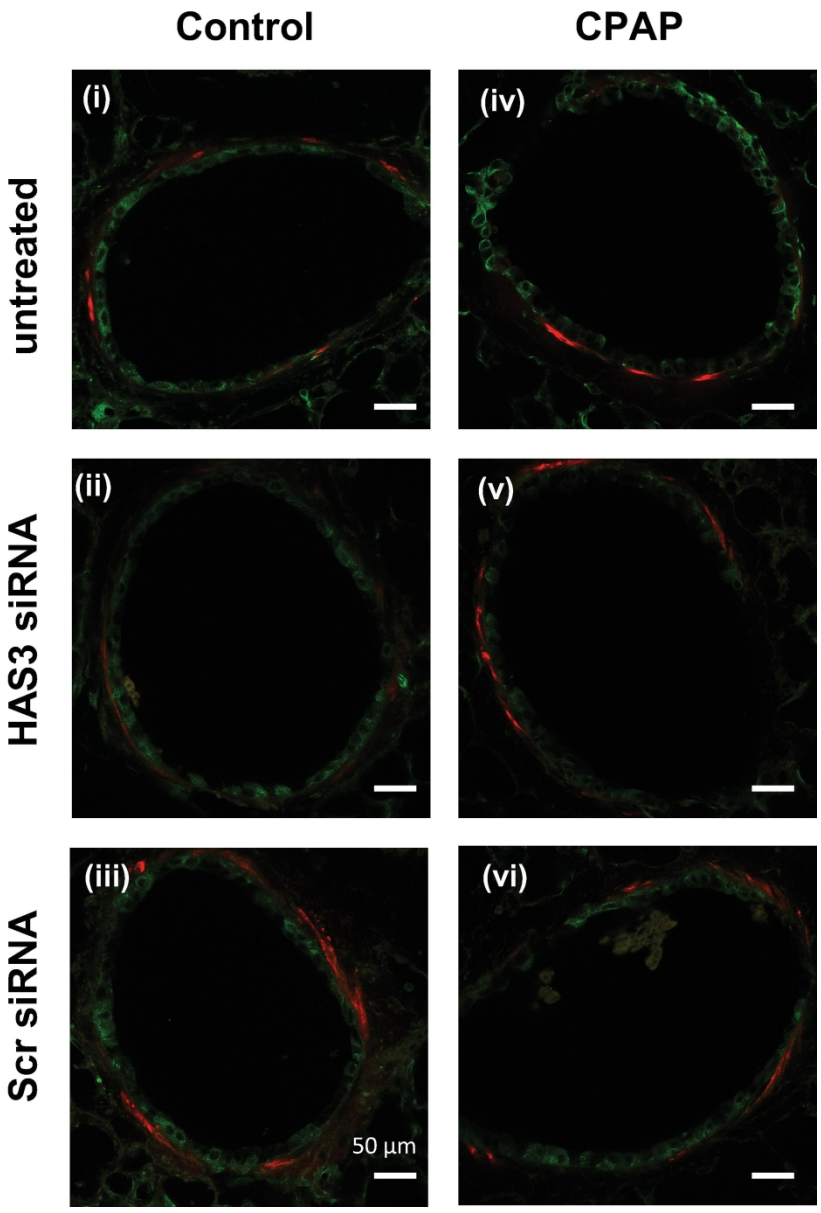
S1: Schematic of the setup used to administer CPAP non-invasively to unanesthetized neonatal mice. A collar is fitted over the face through which air flows to a downstream manometer. An adjustable leak allows optimization of the back-pressure (i.e. CPAP, red arrows) to be applied to the mask and pulmonary system of the mouse. The level of CPAP can be accurately monitored using the manometer (ΔP, pressure difference) and adjusted accordingly. Mice were assigned to receive either zero CPAP (i.e. control group) or 1, 3, or 6 cm H_2O, 3 hours/day (2 hours on the first day), for 7 consecutive days from postnatal age 1-7 days (P1-P7). A custom-designed system was constructed to permit delivery of CPAP to several mice simultaneously. Two weeks after CPAP treatment ended, mice were sacrificed for measurements of airway responses to methacholine challenge using the *in vitro* living lung slice preparation or lung tissue that was collected for rtPCR or IHC.

129x163mm (600 x 600 DPI)



S2: Immunohistochemical analysis using hyaluronan binding protein (HABP) in P21 day old mice following neonatal (P1-7) CPAP (6 cmH_2O). CPAP did not affect airway-specific HABP (green) compared to control mice (A). HABP is expressed as area of positive immunoreactivity/AW lumen circumference. Values are mean ± SEM. Representative images are also shown (B).

257x111mm (600 x 600 DPI)



S3. Representative images of airways from control, HAS3 siRNA, and scrambled siRNA (Scr siRNA) treated lungs in P21 day old control and CPAP treated male mice. Quantification (see Fig. 6) of epithelial HAS3 immunoreactivity (green) is shown double-labeled with airway α-smooth muscle actin (red). Note the increased HAS3 around airways following CPAP (iv) compared to control mice (i), which was significantly reduced following HAS3 siRNA treatment (v). Scrambled siRNA, however, had no effect on epithelial HAS3 expression in either control (iii) or CPAP (vi) treated mice.

102x151mm (600 x 600 DPI)