Fig 1. FoxM1 expression levels are elevated in hypertensive lungs.

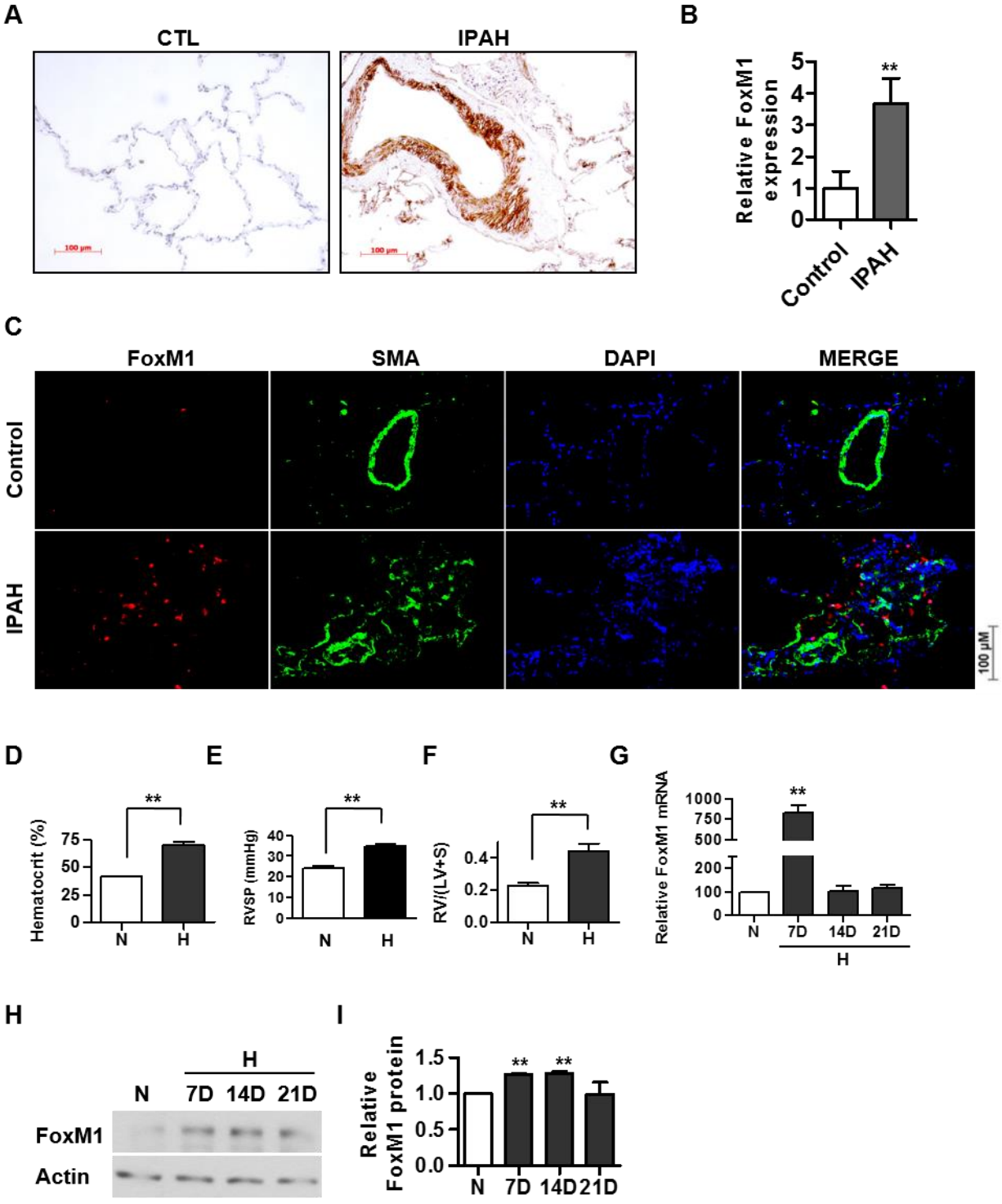
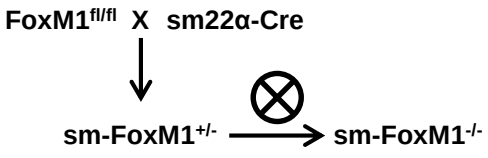
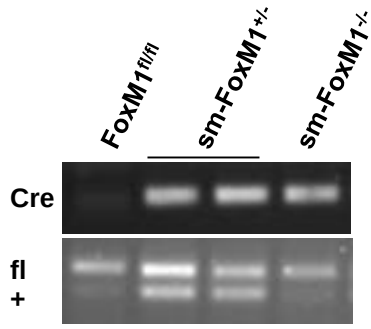


Fig 2. Constitutive knockout of SMC-specific FoxM1 causes mice die early.

A



B



C

Genotypic ratios of heterozygous interbreeding

Viable mice (total No. 79)	Wt (number, ratio)	sm-FoxM1$^{+/-}$ (number, ratio)	sm-FoxM1$^{-/-}$ (number, ratio)
Expected distribution	34.6 (0.4375)	29.6 (0.375)	14.8 (0.1875)
Experimental distribution	47 (0.5949)	30 (0.3797)	2 (0.0253)

D

Genotypic ratios of FoxM1$^{fl/fl}$ and sm-FoxM1$^{+/-}$ interbreeding

Viable mice (total No. 89)	Wt (number, ratio)	sm-FoxM1$^{+/-}$ (number, ratio)	sm-FoxM1$^{-/-}$ (number, ratio)
Expected distribution	45 (0.50)	22.25 (0.25)	22.25 (0.25)
Experimental distribution	43 (0.483)	40 (0.459)	6 (0.067)

Fig 3. Constitutive knockdown of FoxM1 in SMC attenuates hypoxia-induced vascular remodeling and PH in mice.

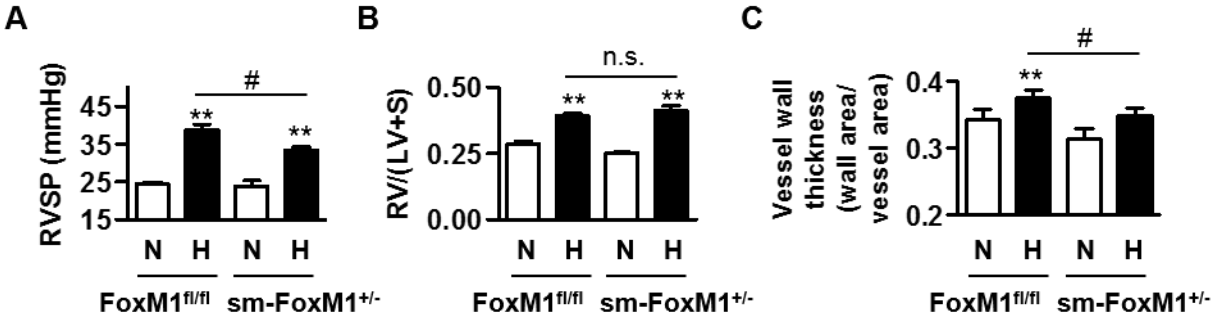


Fig 4. Inducible knockout of FoxM1 reverses existing vessel remodeling in hypoxic mice.

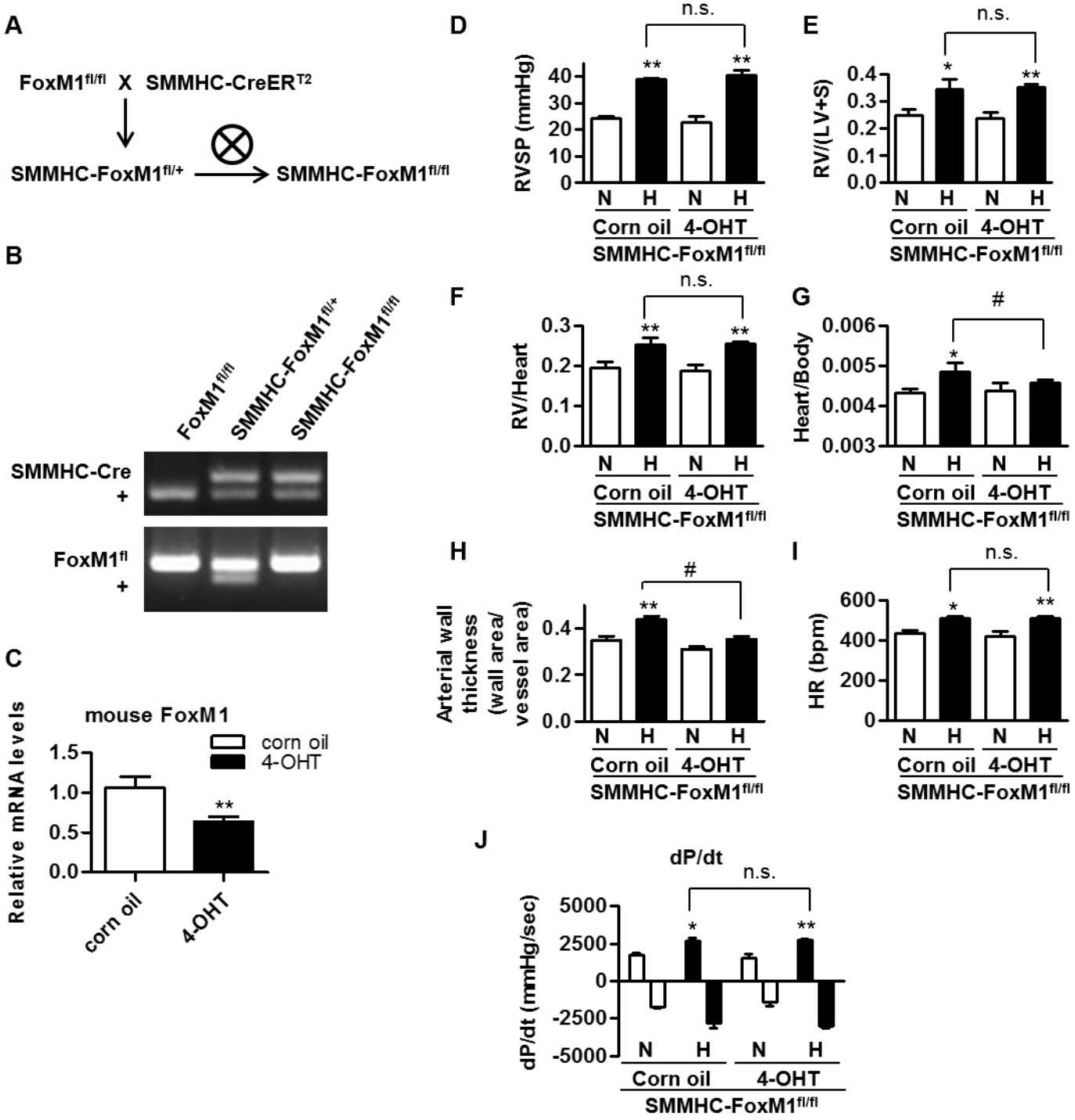


Fig 5. Overexpression of FoxM1 increases hypoxia-induced RV hypertrophy and vessel remodeling.

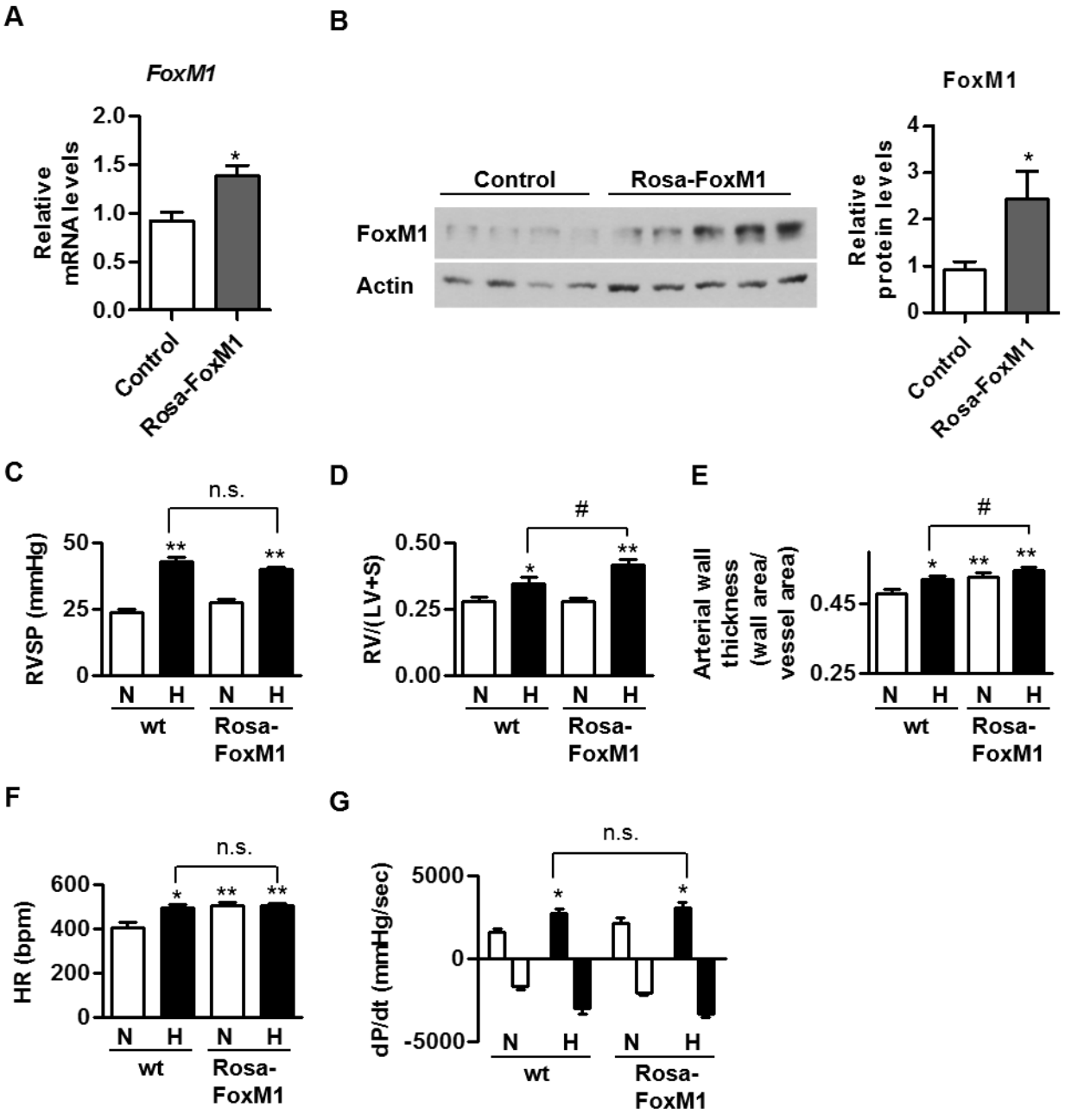


Fig 6. Knockdown of FoxM1 does not affect hypoxia-induced HIF activation.

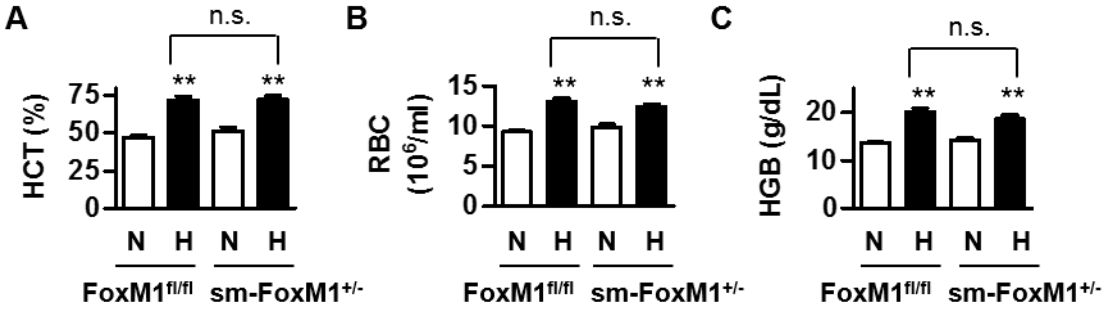


Fig 7. Loss of FoxM1 in SMC decreases PASMC proliferation.

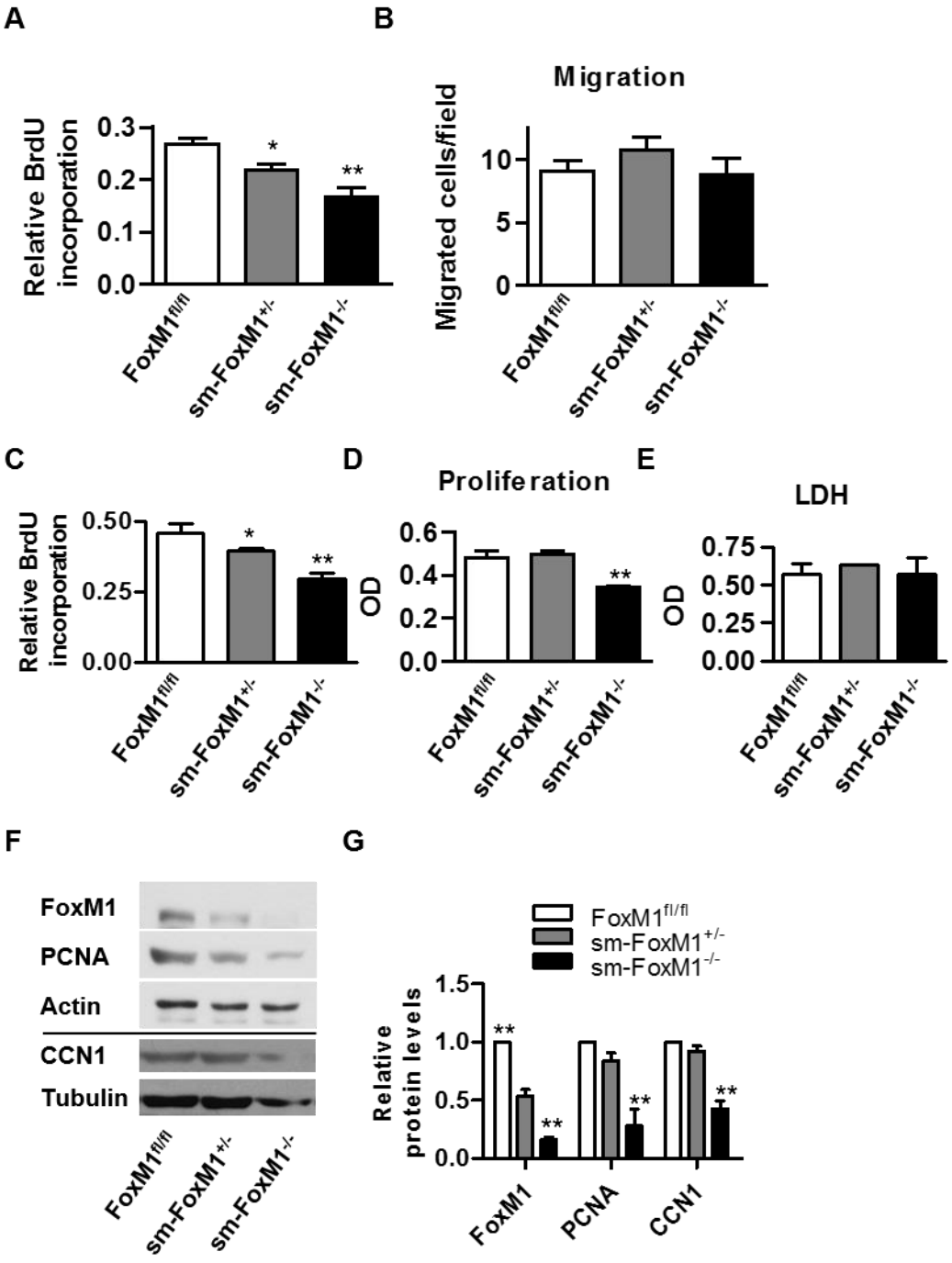


Fig 8. Loss of FoxM1 increases Smad3 signaling and SMC contractile protein expression.