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BACKGROUND

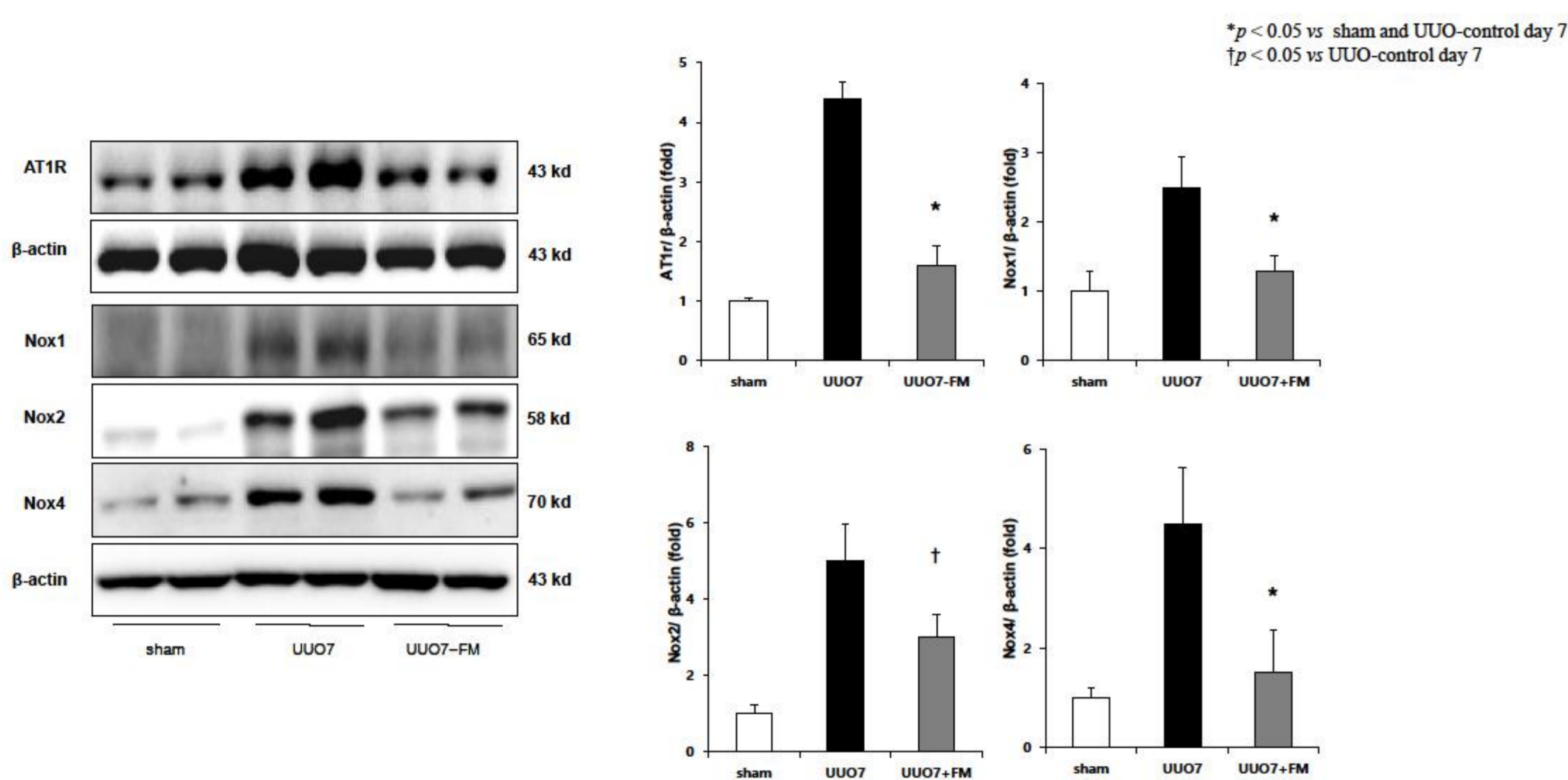
Renal interstitial fibrosis is a common final pathological process in the progression of kidney disease. This is primarily due to oxidative stress, which contributes to renal inflammation and fibrosis. This study is to evaluate the effect of the novel angiotensin receptor blocker, fimasartan, on the renal oxidative stress and fibrosis in an animal model.

METHODS

Mice received unilateral ureteral obstruction (UO) surgery with or without administration of fimasartan (FM). We investigated the effects of FM on renin-angiotensin system activation, the expressions of Nrf2 and its target molecules and renal inflammation & fibrosis on day 7 after UO.

RESULTS

Fig 1. Effects of FM on the expressions of AT1R and Nox in UO mice



UUO7, UUO-control day 7; UUO7-FM, UUO-fimasartan day 7; Values are expressed as means ± SE.

Fig 2. Effects of FM on the expressions of Keap1 & Nrf2 in UO mice

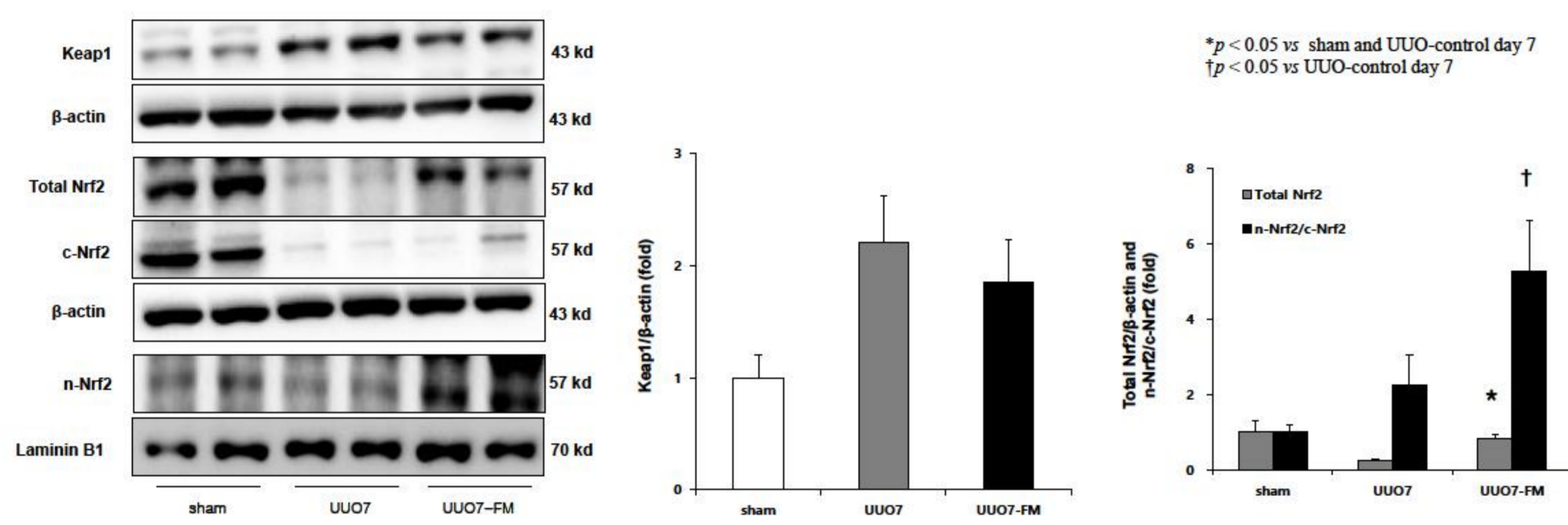
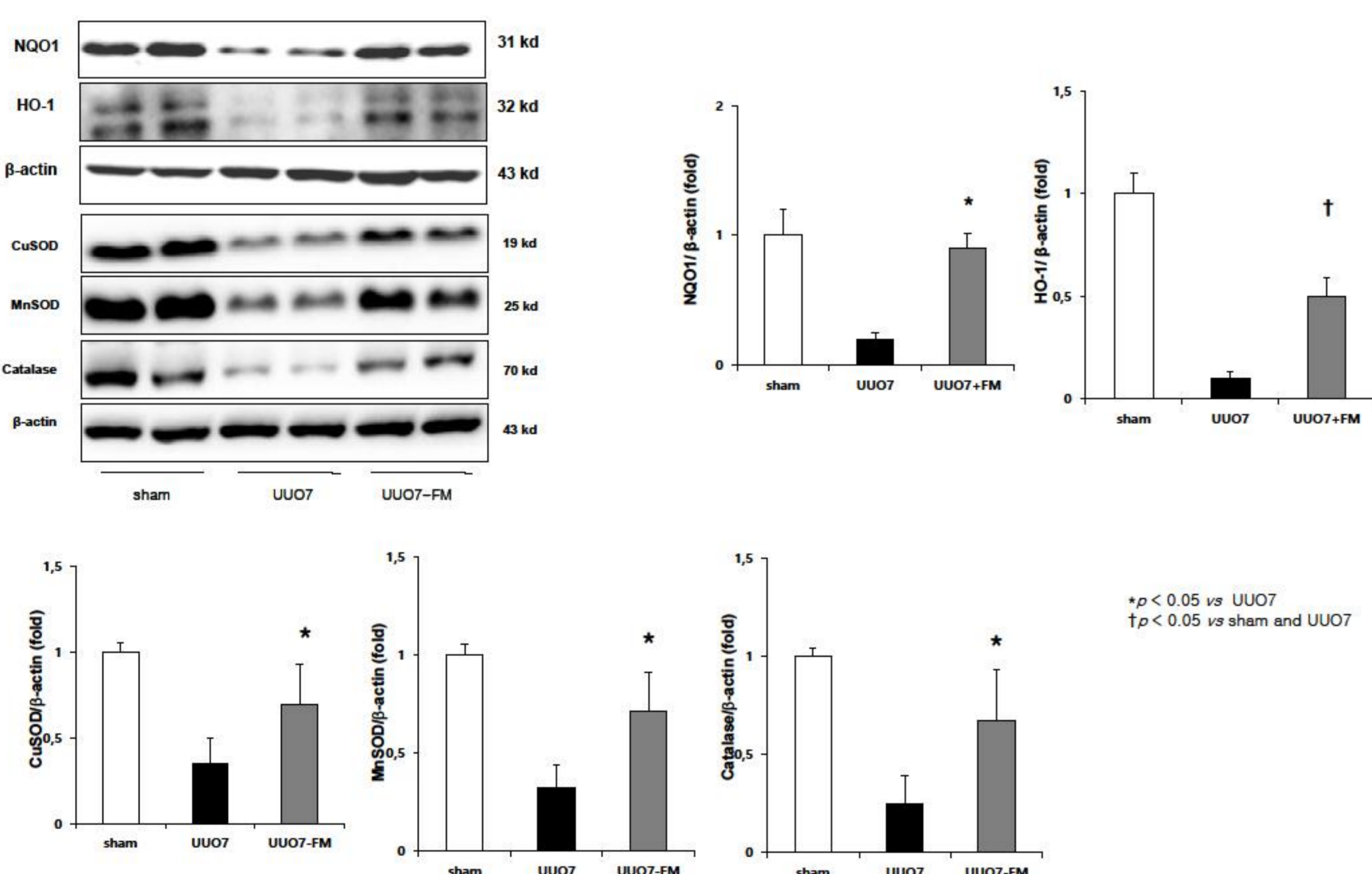


Fig 3. Effects of FM on the expressions of anti-oxidative enzymes in UO mice



RESULTS (continued)

Fig 4. Effects of FM on Nrf2 downstream gene expressions in UO mice

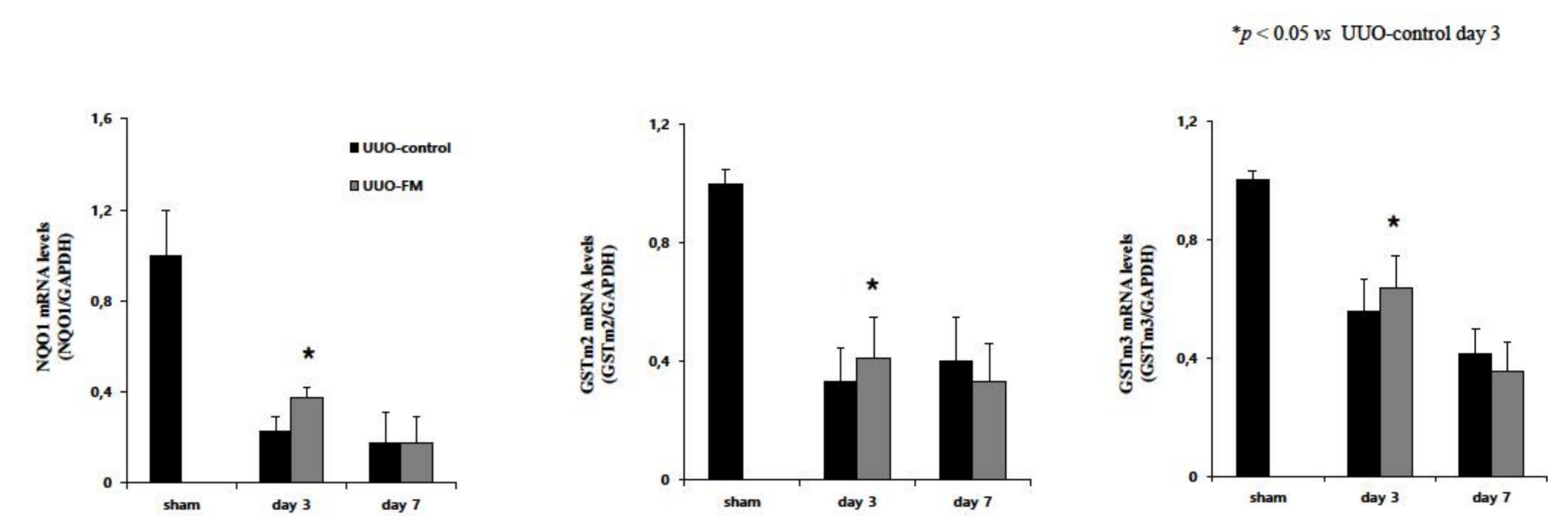


Fig 5. Effects of FM on apoptosis in UO mice

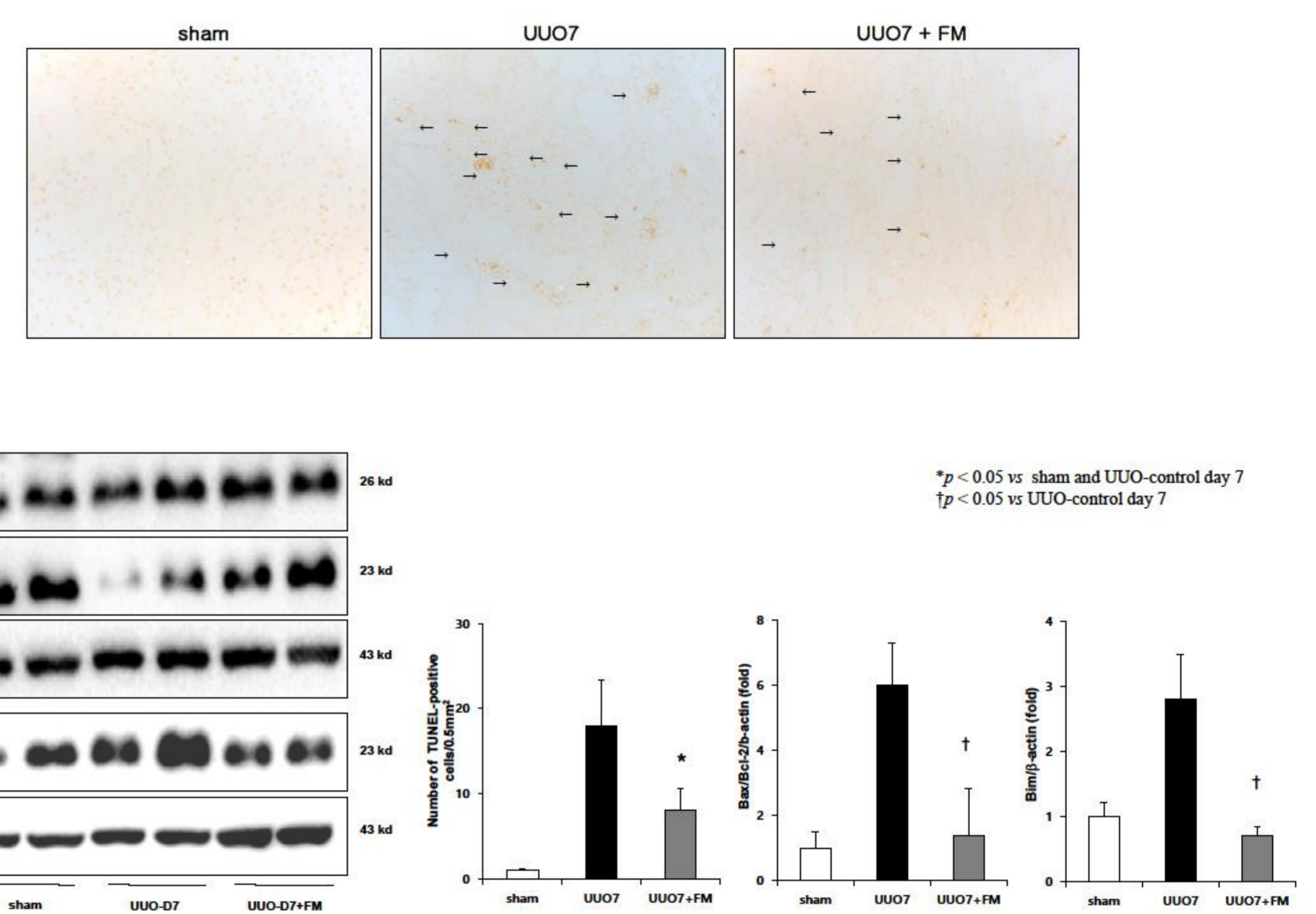
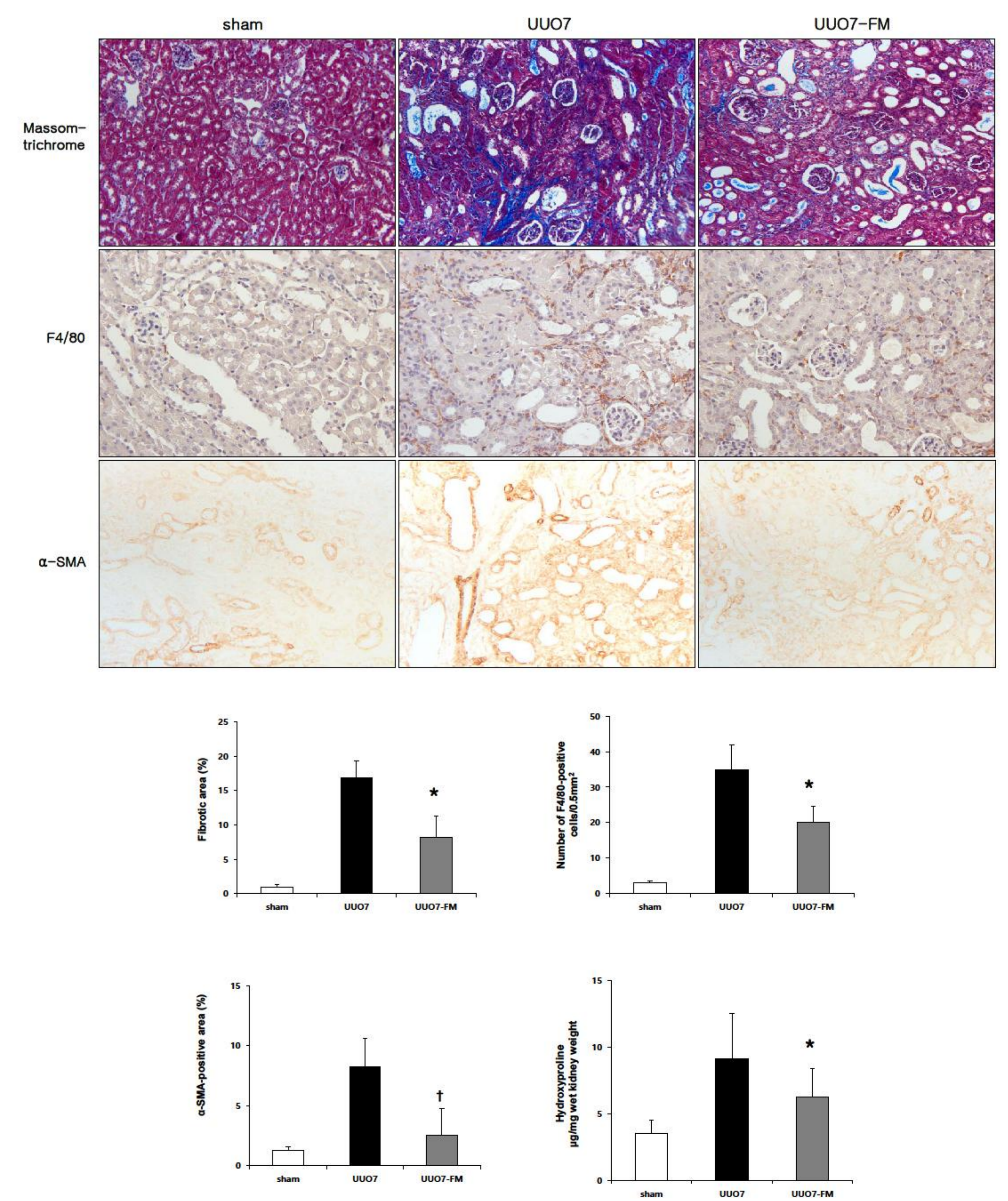


Fig 5. Effects of FM on renal inflammation & fibrosis in UO mice



CONCLUSION

These results suggest that fimasartan may exert beneficial effects on renal damage induced by UO via increasing nuclear translocation of Nrf2 and subsequently reducing renal oxidative stress, inflammation and fibrosis.