

Correction

Correction: Lu et al. Autophagy Alters Bladder Angiogenesis and Improves Bladder Hyperactivity in the Pathogenesis of Ketamine-Induced Cystitis in a Rat Model. *Biology* 2021, 10, 488

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Error in Figures 6 and 8

In the original publication [1], there was a mistake in Figure 6C (Ketamine + Rapamycin group, immunofluorescence analysis of laminin) and Figure 8A (Western blot of Akt) as published. Image panels were mistakenly duplicated or misplaced during figure preparation. The corrected Figures 6C and 8A appears below. The authors state that the scientific conclusions are unaffected. This correction was approved by the Academic Editor. The original publication has also been updated.

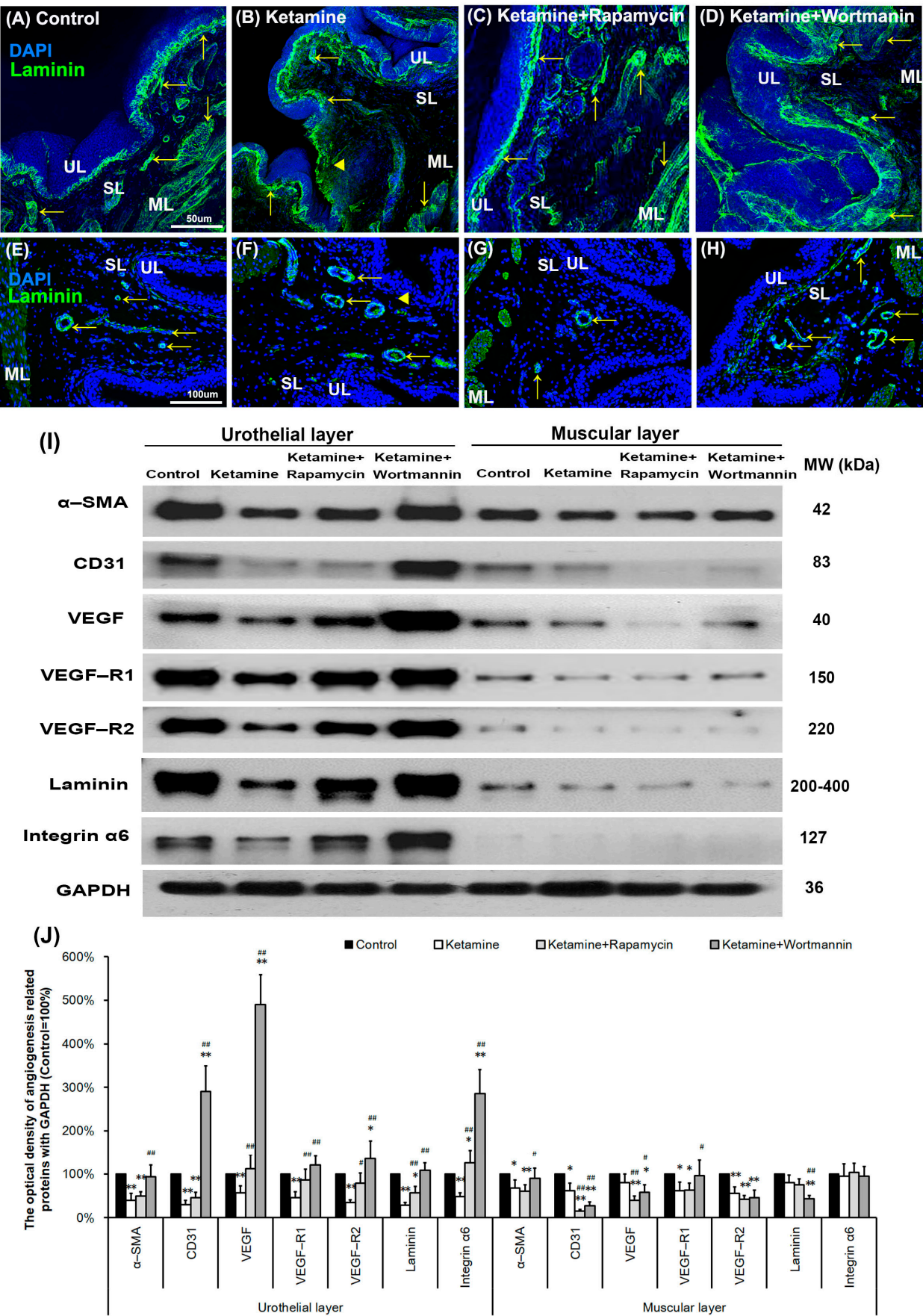


Figure 6. Autophagy altered bladder angiogenesis in the pathogenesis of KIC. (A–H) Immunofluorescence analysis of laminin, and α -SMA (E–H) (angiogenesis marker, arrows) expression after treatment

with saline (A,E), ketamine (B,F), ketamine+rapamycin (C,G), and ketamine+wortmannin (D,H) in urothelial layer (UL), suburothelial layer (SL) and muscular layer (ML) of bladder tissue. In the ketamine+rapamycin group, bladder angiogenesis was mainly expressed in the marginal zone of lamina propria (suburothelial layer) near the urothelial basement membrane. The ketamine+wortmannin group significantly intensified the expression levels in the urothelial layer as compared to the other groups. The nuclei were counterstained by DAPI (blue). Yellow arrowheads indicate the denuded urothelial mucosa. Scale bar = 50 μm (Magnification $\times 200$) and 100 μm (Magnification $\times 400$). (I) Western blots of angiogenesis markers, including α -SMA, CD31 (endothelial marker), VEGF, VEGF-R1, VEGF-R2 (VEGF receptor), laminin, and integrin- $\alpha 6$ (laminin receptor), were quantified against glyceraldehyde-3-phosphate dehydrogenase (GAPDH). (J) Results were normalized to the control group (equal to 100%). α -SMA, alpha-smooth muscle actin; CD31, cluster of differentiation 31; VEGF, vascular endothelial growth factor. Data are expressed as means $\pm$ SD for $n = 6$, * $p < 0.05$, ** $p < 0.01$ versus the control group; # $p < 0.05$, ## $p < 0.01$ versus the ketamine group.

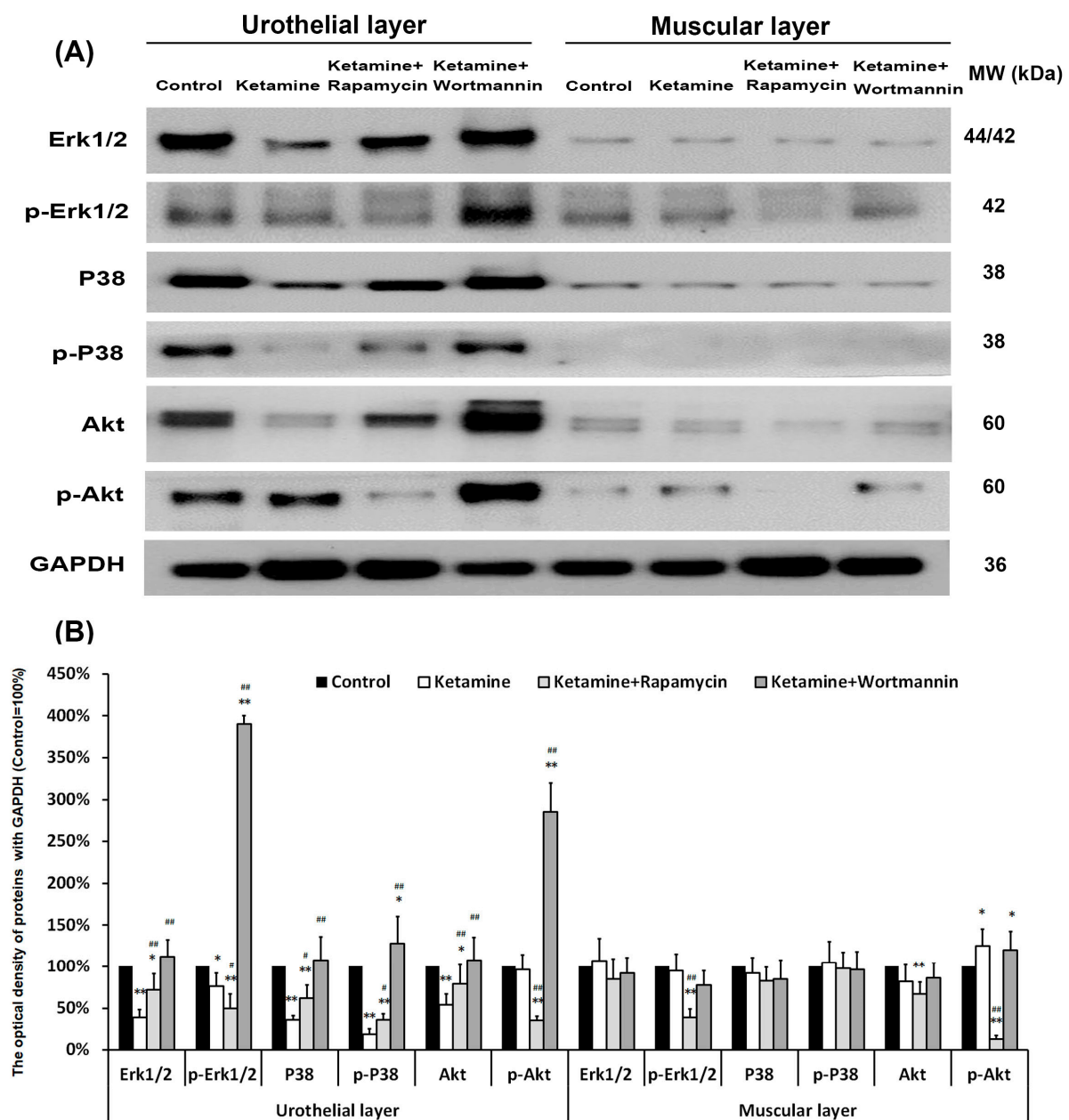


Figure 8. Western blots of signaling pathway kinase, including Erk1/2, p-Erk1/2, P38, p-P38, Akt, and p-Akt, were quantified against GAPDH (A). (B) Results were normalized to the control group (equal to 100%). The data revealed that treatment with ketamine significantly increased the Akt phosphorylation,

but reduced the phosphorylation of Erk1/2 and p38 as compared to the control group. However, treatment with rapamycin increased the p38 phosphorylation, but suppressed the expression of p-Erk1/2 and p-Akt. Data were expressed as means $\pm$ SD for $n = 6$, * $p < 0.05$, ** $p < 0.01$ versus the control group; # $p < 0.05$, ## $p < 0.01$ versus the ketamine group.

Reference

1. Lu, J.-H.; Wu, Y.-H.; Juan, T.-J.; Lin, H.-Y.; Lin, R.-J.; Chueh, K.-S.; Lee, Y.-C.; Chang, C.-Y.; Juan, Y.-S. Autophagy Alters Bladder Angiogenesis and Improves Bladder Hyperactivity in the Pathogenesis of Ketamine-Induced Cystitis in a Rat Model. *Biology* **2021**, *10*, 488. [[CrossRef](#)] [[PubMed](#)]

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