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# Corrigendum: Persistent activation of autophagy after cisplatin nephrotoxicity promotes renal fibrosis and chronic kidney disease

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## KEYWORDS

autophagy, cisplatin, kidney injury and repair, renal fibrosis, profibrotic growth factor

## A Corrigendum on Persistent activation of autophagy after cisplatin nephrotoxicity promotes renal fibrosis and chronic kidney disease

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In the published article, there was an error in [Figure 2E](#) as published. The unit of measurement is incorrectly listed as “uL/min/100 g b.w.” and should be corrected to “mL/min/100 g b.w.”. The corrected [Figure 2](#) and its caption Pharmacologic inhibition of autophagy alleviates renal dysfunction and tubular damage in post-RLDC kidneys appear below.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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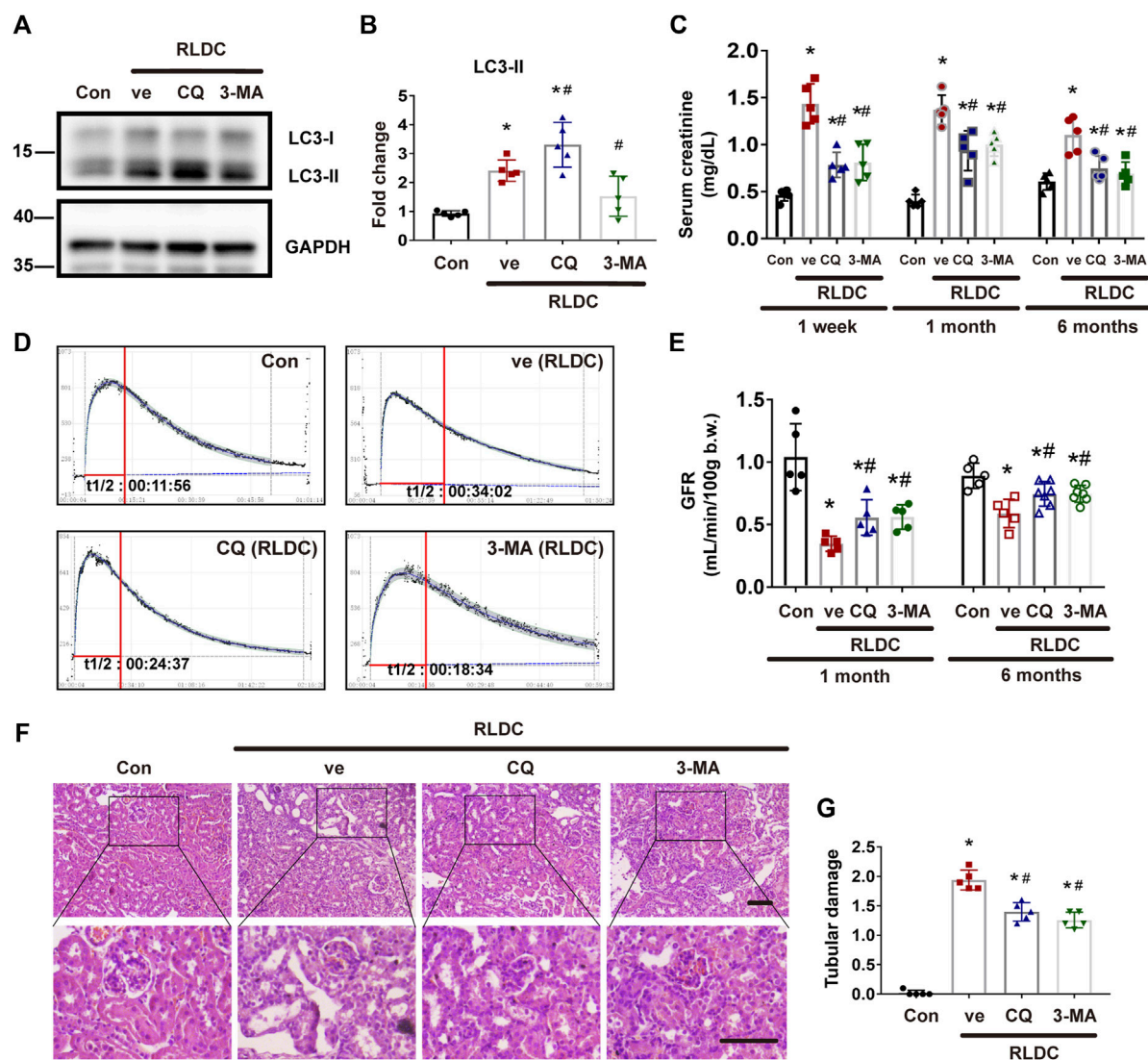


FIGURE 2

Pharmacologic inhibition of autophagy alleviates renal dysfunction and tubular damage in post-RLDC kidneys. Male C57BL/6 mice were injected weekly with 8 mg/kg cisplatin for 4 weeks (RLDC) or with saline as control (Con). After the final dose, the mice were injected with 60 mg/kg/day chloroquine (CQ), 20 mg/kg/day 3-methyladenine (3-MA), or saline as vehicle solution (ve) for 7 days. **(A)** Representative immunoblots of LC3-I, LC3-II and GAPDH (loading control) in kidney tissues ( $n = 5$ ). **(B)** Densitometry of LC3II. The experiments were normalized according to GAPDH expression. The protein level of control group (Con) was arbitrarily set as 1, and the signals of other conditions were normalized with the control group to indicate their protein fold changes. **(C)** Effect of autophagy inhibitor on serum creatinine at 1 week, 1 month and 6 months after RLDC treatment. ( $n = 5$ ). **(D)** Representative tracing curves of FITC-sinistrin clearance in mice. ( $n = 5$ ). **(E)** GFR measurement by transcutaneously monitoring FITC-sinistrin clearance ( $n = 5$ ). **(F)** Representative histology images of hematoxylin-eosin staining of kidney tissues in renal cortex and outer medulla. ( $n = 5$ , bar = 50  $\mu\text{m}$ ). **(G)** Pathological score of tubular damage. Quantitative data are expressed as mean  $\pm$  SEM. \* $p < 0.05$  vs the control group (Con), # $p < 0.05$  vs. (RLDC + vehicle) group.