# Aggravation of adriamycin-induced nephropathy in SLC41A2 knockout mice

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$Mg^{2+}$ is an essential divalent cation, which plays pivotal roles in fundamental cellular functions. $Mg^{2+}$ deficiency or abnormal $Mg^{2+}$ metabolism is related to various cardiovascular and neuromuscular diseases. In recent years, several candidate genes of $Mg^{2+}$ transporters including SLC41A1/A2 have been reported. We have been studying their physiological roles and pathological significances using genetic altered mice targeting each candidate gene. Recently, we generated and characterized SLC41A1/A2 mice, and found that these mice exhibited dysregulation of serum $Mg^{2+}$ levels and urinary $Mg^{2+}$ excretion. Here we show that adriamycin-induced nephropathy (albuminuria, renal glomerular degeneration) was markedly aggravated in SLC41A2 knockout mice, but not in SLC41A1 knockout mice, compared to wild-type mice. On the other hand, cisplatin-induced nephrotoxicity was observed to the similar degree among SLC41A1/A2 knockout mice and wild-type mice.These results suggest that SLC41A2 is involved in the development and progression of adriamycin-induced nephropathy.