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# Blood Pressure Regulation by $Mg^{2+}$ Transporter MagEx2

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Summary

Salt sensitive hypertension is associated with an increased risk of cardiovascular events, but the precise mechanisms underlying this disease are still largely unknown. We have previously found that magnesium reabsorption was impaired in mouse deficient of *MagEx2*, which encodes a $Mg^{2+}$ transporter. Also, we have shown by tail cuff method that the blood pressure was also significantly lower in *MagEx2*-deficient mice. In this study, we further analyzed this mouse strain to corroborate the importance of MagEx2 in blood pressure control.

First, we measured the blood pressure of *MagEx2*-deficient mice by radiotelemetry, another well-known method for blood pressure measurements in mouse studies. As expected, the blood pressure was significantly reduced in both systemic heterozygotes and kidney specific homozygotes of *MagEx2*-deficient allele. We next analyzed the blood pressure of *MagEx2*-deficient mice in hypertensive mouse model. For this purpose, we employed DOCA-salt method, which was frequently used to generate hypertensive mice. Under this condition, we observed a significant blood pressure increase in wild type mice, and the blood pressure was significantly lower in DOCA-salt treated *MagEx2*-deficient mouse strains.

Furthermore, we performed a comparative study with *MagEx4*-deficient mice, which encodes another MagEx family $Mg^{2+}$ transporter involved in intestinal magnesium absorption. While the blood magnesium level of *MagEx4*- and *MagEx2*-deficient mice were similarly low compared to wild type mice, the blood pressure of *MagEx4*-deficient mice was higher than wild type mice. Since the ratio of the renal magnesium reabsorption in *MagEx4*-deficient mice was higher than wild type mice, these experimental results strongly suggests that magnesium reabsorption mediated by MagEx2 is primarily important for the blood pressure regulation.