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A Celluar Iron Receptor in Hypertensive Cardiovascular Remodeling

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Summary

Background: We have previously reported that dietary iron restriction prevents the development of hypertensive cardiovascular remodeling in rat models of hypertension. In addition, we have shown that a cellular iron transport protein, **transferrin receptor 1 (**TfR1) is highly expressed in cardiovascular tissues of hypertensive rats; however, the role of TfR1 in the pathophysiology of hypertension remains obscure. In this study, we investigate the role of TfR1 in cardiovascular tissues of hypertension and the effect of pair-feeding dietary iron restriction in rat models of hypertension.

Methods and Results: First, we infused angiotensin II via an osmotic minipump at the rate of 1.44 mg/kg/day for 2 weeks in wild-type (WT) and TfR1 hetero knockout mice. Systolic blood pressure was elevated in both WT and TfR1 hetero knockout mice after angiotensin II infusion; however, left ventricular hypertrophy was increased to a lesser extent in TfR1 hetero knockout mice compared with WT mice. Second, we assessed the effect of pair-feeding iron restriction in 5/6 nephrectomized rats. Pair-feeding iron restriction attenuated the development of renal damage and hypertension in 5/6 nephrectomized rats. Of interest, pair-feeding iron restriction attenuated renal expression of nuclear mineralocorticoid receptor in 5/6 nephrectomized rats.

Conclusions: These results indicate that TfR1 plays a role in the pathophysiology of hypertensive cardiac hypertrophy and iron might be possible therapeutic targets for hypertension.