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CORRESPONDENCE

Reply to 'Iron restriction in renovascular hypertension'

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We thank Dr Tanemoto for his critical reading of our recently published manuscript in *Hypertension Research*.[1] We would like to point out that the aim of our study was to assess the effect of dietary iron restriction (IR) on hypertension and renal damage in a rat model of two-kidney one-clip (2K1C) renovascular hypertension.

2K1C model is widely used as an experimental model that in many respects resembles human renovascular hypertension (RVHT).[2] Stenosis of the unilateral renal artery develops progressive atrophy of the clipped kidney and compensatory hypertrophy of the nonclipped kidney.[3] In 2K1C model, the degree of renal artery stenosis, atrophy of the clipped kidney and compensatory hypertrophy of the nonclipped kidney depend on the degree of the clipping procedure.[4] Since progressive atrophy of the clipped kidney was observed in our study, the degree of our clipping procedure might be severe. We agree with Dr Tanemoto's opinion that activation of the renin-angiotensin system (RAS) compensates for the ischemia of the clipped kidney by increasing blood pressure, but we could not assess the RAS activity in our study. Since the degree of our clipping procedure was essentially constant and unvarying in the present study, the effect of IR on our 2K1C model is considered to be evaluated accurately.

As Dr Tanemoto pointed out, the reduction in compensatory hypertrophy of the nonclipped kidney may reflect body weight (BW) of the animals because BW was lower in the 2K1C+IR group compared with the other groups. However, there was no difference in the clipped kidney weights between the 2K1C and 2K1C+IR groups. Additionally, a previous study reported that kidney weights are not affected by IR despite of BW reduction.[5] Thus, there may be little association between compensatory renal hypertrophy and BW in our study. Moreover, tibia lengths did not differ among the experimental groups. We thus compared kidney weight to tibia length ratios but not kidney weight to BW ratios among the groups.

We and others have previously reported that long-term IR reduced BW,[5,6] suggesting an association between IR and reduced food intake. As Dr Tanemoto pointed out, reduced food intake may decrease potassium intake, which might lead to reduced urinary potassium excretion and reduced mineralocorticoid receptor activity in the 2K1C+IR group. If so, it should also decrease sodium and protein intake. However, we did not observe reduced urinary sodium or protein excretion in the 2K1C+IR group compared with the control group. On the other hand, reduced food intake might contribute to blood pressure reduction. In this regard, experiments need to be controlled for caloric intake. Therefore, we would like to investigate the effect of IR on hypertension and renal damage using pair-feeding experiments in the near future.

We hope these responses are helpful in providing additional understanding of our study.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

Yoshiro Naito, Makiko Oboshi and Tohru Masuyama

Cardiovascular Division, Department of Internal Medicine, Hyogo College of Medicine, Nishinomiya, Japan
E-mail: ynaito@hyo-med.ac.jp

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