Response to Oxidative Stress Promotes Hypertension and Albuminuria During the Autoimmune Disease Systemic Lupus Erythematosus

We thank Dhaun and Kluth\textsuperscript{1} for their interest in, and comments related to, our recent study designed to understand mechanisms that promote hypertension during the autoimmune disorder systemic lupus erythematosus (SLE)\textsuperscript{2}. Based on our previous work demonstrating that renal oxidative stress is associated with hypertension in a mouse model of SLE\textsuperscript{3}, the major purpose of our current study was to test whether oxidative stress has a causal role in the genesis of the elevated blood pressure. With regard to the reduction in albuminuria in SLE mice being attributed to reduced blood pressure, evidence in humans with SLE suggests that blood pressure and albuminuria are not necessarily linked\textsuperscript{4} and that albuminuria occurs independent of hypertension in other experimental mouse models of SLE\textsuperscript{5}. Nonetheless, we agree that blood pressure is an important determinant of urinary albumin excretion, and our laboratory is actively investigating the link between blood pressure and albuminuria in this model. An important component of this ongoing investigation includes careful examination of renal histological changes. We are also in agreement with the assessment by Dhaun and Kluth\textsuperscript{1} that a potentially important way in which oxidative stress promotes hypertension during SLE may be through a renal vascular mechanism. Indeed, we devoted a significant portion of the discussion and perspectives to this very idea.

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Disclosures

None.

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