**METHODS**

**ABSTRACT**

Animals: Eight-week-old, male mice with gp91phox+/- and gp91phox+/- gp91phox+/- (C57BL/6 strain, Jackson Laboratories) were used in the present study. These mice were either on the normal diet (ND) or folate-free diet (FF) (Dyets Inc). Hcy-induced renal injury was produced by feeding FF for 6 weeks in uninephrectomized mice. Total Hcy (Hcy) concentrations were measured by HPLC analysis and the kidneys were used for morphological examinations and biochemical analyses.

**RESULTS**

Electroretinographic Spin Response (ESR) Analysis of O2. Production in the Renal Cortex. Hematogenous renal cortical tissues were prepared by using sucrose buffer and reseeded with modified Krebs-Ringer bicarbonate containing different concentrations (200 U/ml) and nifedipine. NOX, NOX-mediated O2. production was examined by addition of 1 nM NaNO3 as a substrate in 95 μM probe in the presence or absence of NOD (200 U/ml), and then supplied with 1 mM O2- (Fig. 4). 2.5 3.0 3.5

**CONCLUSION**

Hyperhomocysteinemia (hHcys) has been reported to be a pathogenic factor to induce renal dysfunction and glomerular injury, which ultimately results in glomerular sclerotic and end-stage renal disease (ESRD).

Previous studies from our laboratory have indicated that the activation of Rac-NADPH oxidase (NOX) plays a crucial role in hHcys-induced glomerular sclerosis as shown in the following diagram:

![Diagram](image)

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