Oxidative stress in children with nephrotic syndrome

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Received: 17 September 2011 / Accepted: 19 September 2011 / Published online: 8 October 2011
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Sir,

The letter to the editor by Kaneko and colleagues reporting changes in urinary 8-hydroxydeoxyguanosine (8-OH-DG) in idiopathic nephrotic syndrome adds further important information to the role played by oxidative stress in acute glomerular disease [1].

Free radicals are extremely reactive compounds that interact with lipids, proteins, and nucleic acids. The kidney physiologically generates a small amount of reactive oxygen species (ROS) as part of oxidative metabolism, which is well tolerated. If generated in larger amounts locally, ROS may conceivably cause or contribute to the damage of glomerular structures, leading to altered glomerular permselectivity.

Whereas the increased plasma levels of malonyldialdehyde and nitrite observed by us and others in acute nephrotic syndrome signal increased lipid and protein peroxidation [2, 3], the increased urinary excretion of 8-OH-DG as observed by Kaneko et al. reflects the interaction of ROS with cellular DNA [1]. Hence, their finding nicely complements the concept of an imbalance of ROS

induction and impaired antioxidant mechanisms in acute nephrotic syndrome [2, 3]. With respect to the specificity of this potential pathophysiological mechanism, it is of note that no increase in 8-OH-DG occurred in three patients with acute poststreptococcal glomerulonephritis. If this preliminary observation is confirmed, it would lend support to a specific role of oxidative stress in idiopathic nephrotic syndrome.

References

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