

**Harvard Medical School Department of
Continuing Education and the Renal Division
of Brigham and Women's Hospital**



Nephrology Rounds
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Molecular Mechanisms Underlying Diabetic Nephropathy

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Objectives

This issue of *Nephrology Rounds* will help readers to review:

- intracellular signaling that is activated by increased intracellular glucose in mesangial cells
- the current understanding of the association between glomerular hemodynamic changes and mesangial structural lesions
- the contribution of glucose uptake to altered intracellular signaling.

Questions:

1. The deterioration in glomerular filtration rate (GFR) that occurs as a result of diabetic nephropathy is likely unrelated to the structural changes that occur in the mesangium.

True False

2. Increased glucose uptake by mesangial cells underlies many of the pathogenetic effects of hyperglycemia.

True False

3. Which of the following contributes to diabetic structural lesions:

- a. advanced glycosylation end-products
- b. hexosamines
- c. reactive oxygen species
- d. all of the above

4. According to current data, activation of TGF-, underlies all of the manifestations of diabetic nephropathy.

True False

5. Increased transglomerular pressure is easily explained by the effect of increased angiotensin II on the efferent arteriole.

True False

6. Glomerular hemodynamic changes and mesangial structural lesions both contribute to the decline in GFR from diabetic nephropathy, but are unrelated to each other.

True False

7. Reactive oxygen species are primarily generated as an inevitable byproduct of oxidative phosphorylation.

True False

8. All of the effects of advanced glycosylation end-products are receptor-independent.

True False

9. All cell types carefully regulate the uptake of glucose.

True False

10. TGF- β expression is invariably detrimental.

True False

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