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Mechanism of the Glomerulus and Tubules in the Diabetic Kidney

Hamilton Dare*

Department of Cancer Biology, 4500 San Pablo Road, Jacksonville, USA

Introduction

The most common cause of ESRD in the West is diabetic nephropathy. Structure and function of the kidney begin to alter in the early stages of diabetes, and these changes have a long-lasting impact on future renal function as well as the onset and progression of overt diabetic nephropathy. These early anatomical and functional alterations, such as kidney hypertrophy and glomerular hyperfiltration, have been thoroughly studied in animal models of diabetes. Both are thought to be the starting points for glomerulosclerosis and the following decline in kidney function.

A fraction of diabetic individuals are found to have kidney hypertrophy in the clinical setting, and this finding is linked to the progression of diabetic nephropathy later on. Similar to early hyperfiltration, early hyperfiltration has been seen in a minority of diabetic patients in the early stages. These patients appear to be more prone to later develop overt diabetic nephropathy, which affects about one-third of diabetic patients. These and other related early pathophysiological alterations in the diabetic kidney, as well as their underlying processes, are the main topics of this review [1-3].

Hypertrophy

Kidney development has been noticed relatively early in streptozotocin-induced experimental diabetes. For the first four days of the first phase, there is hyperplasia, which is followed by a change to hypertrophy, which is mostly mediated by transforming growth factor-β1. The transition from hyperplasia to hypertrophy appears to be dependent on protein kinase C β1, and the initial hyperplasia may require stimulation of the renin-angiotensin system (PKC β 1). After streptozotocin diabetes is induced, PKC β 1 is expressed in the proximal tubule and can trigger transforming growth factor- β.

Angiotensin-converting enzyme inhibition prevents the diabetes-induced activation of PKCb in the kidney, which may link the positive effects of angiotensin inhibition to tubular reabsorption and renal growth in the diabetic kidney. Increased mTOR activity seems to be made possible in the diabetic kidney by reduced AMPK phosphorylation, and decreasing AMPK phosphorylation reduces kidney development without changing hyperglycemia [4].

Hyperfiltration

Early hyperfiltration has been seen in rat models of experimental diabetes utilising streptozotocin, a model of type 1 diabetes mellitus (T1DM) caused by pancreatic beta-cell damage. If an animal is hyperglycemic, hyperfiltration can also happen even in the presence of insulin, but insulin-intensive blood sugar management consistently delays the development of glomerular hyperfiltration.

Based on the material that is currently available, it is not possible to draw any definitive conclusions about the precise contribution of changes in known hormonal vasoconstrictors or vasodilators as the cause of renal vasodilation. Our own lab is one of many that have looked into the function of the tubuloglomerular feedback (TGF) system in early diabetes. Whether the glomerular hyperfiltration, which is predominantly caused by renal vasodilation, is mediated by factors external to the kidney or whether it is secondary to some internal kidney mechanism is a crucial topic that has to be answered [5].

The inactivation of the TGF system and a significant increase in tubular reabsorption close to the MD, which leads to secondary vasodilation and an increase in SNGFR, are implied if luminal Na and Cl are lowered below normal. In diabetic rats, micropuncture investigations have shown an increase in absolute and fractional proximal tubular reabsorption, as well as a decrease in the concentration of MD NaCl and an increase in SNGFR.

Hyperreabsorption

An explanation of the mechanisms behind the primary increase in proximal reabsorption is necessary to comprehend the pathophysiology of the early diabetic kidney. On the basis of the literature that is currently accessible, three factors seem to be involved in the rise in proximal reabsorption.

The first is proximal tubular hyperplasia and hypertrophy, the second is increased proximal reabsorption brought on by amplified Na-glucose cotransport, which is mediated by SGLT2 and SGLT1, and the third is an exaggerated and adverse reabsorption effect of NaCl intake on the proximal tubule in the diabetic kidney. Hypertrophy was covered in the part before, and now we'll go into more detail about the other 2 variables.

Conflict of Interest

There are no conflicts of interest by author.

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***Address for Correspondence:** Hamilton Dare, Department of Cancer Biology, 4500 San Pablo Road, Jacksonville, USA; E-mail: dare.hamilton@gmail.com*

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