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Modeling Pharmaceutical Inhibition of Glucose-Stimulated Renin-Angiotensin System in Kidneys

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Diabetic hyperglycemia is known to induce kidney damage by causing injury to renal cells known as podocytes. The cells are critical in maintaining the filtration function of kidney. These cells express hormones of the renin-angiotensin system (RAS) intracellularly in a manner that is altered in hyperglycemic conditions. The key hormone that is overexpressed and can lead to kidney damage is angiotensin II (ANG II). The enzyme responsible for production of ANG II from precursors is angiotensin converting enzyme (ACE). A pharmacokinetic/pharmacodynamic model is developed for localized intracellular RAS to predict the effect of an ACE inhibitor pharmaceutical to suppress elevated ANG II levels. The results of this model are compared to a previously developed model for the RAS expressed systemically.