

Big Shift Seen in Prescribing Patterns for Type 2

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SAN DIEGO – Since the mid-2000s clinicians dramatically shifted away from prescribing rosiglitazone and pioglitazone in favor of other novel type 2 diabetes medications, results from a large, single-center study suggest.

At the meeting, Dr. Sanjeev N. Mehta presented findings from a study of electronic medical records at Joslin Diabetes Center, Boston, that evaluated 10-year prescribing patterns for rosiglitazone and pioglitazone, as well as uptake of novel drug classes (categorized as “other”) approved between 2001 and 2010. The “other” group consisted of amylin analogs, GLP-1 analogues, DPP-IV inhibitors, and bile acid sequestrants. Insulin use was not studied, nor were medications introduced prior to 2001.

The analysis aimed to measure

After 2007, new prescriptions for rosiglitazone and pioglitazone declined dramatically; by 2010, new prescriptions were at 7% and 47%, respectively, of peak levels.

provider response to the 2007 FDA boxed warning for rosiglitazone, which was implemented due to the drug’s association with adverse cardiovascular outcomes.

“Given the availability of these new drugs, there’s a need to better understand provider patterns reading the use of new and established medications, [including] responsiveness to FDA indications and safety warnings,” said Dr. Mehta, a staff physician at Joslin Diabetes Center.

Electronic health records with integrated prescribing functionality “may best describe provider behaviors, as their content is not limited to patient claims,” he added. “Further, the detailed clinical information may provide the information necessary to validate both patient conditions and health outcomes.”

Eligible patients had a diagnosis of type 2 diabetes based on an algorithm that used ICD-9 codes and a field that specified diabetes type. If a medication was used and later resumed, he and his associates used the earliest start date. For validation they conducted a manual review of 60 random electronic medical records.

Over the 10-year study period, 7,846 patients with type 2 diabetes had 9,178 new prescriptions for rosiglitazone, pioglitazone, and other agents. After 2007, the number of new prescriptions for rosiglitazone and pioglitazone declined dramatically.

By 2010, new prescriptions for rosiglitazone and pioglitazone were at 7% and 47%, respectively, of peak levels. The relative proportion of prescriptions for rosiglitazone, pioglitazone, and other medications was 44%, 50%, and 6% in

2005 and 2%, 18%, and 80% in 2010. “Prescribing patterns may be described using EHR-based clinical data,” Dr. Mehta said.

“The data suggest provider responsiveness to an FDA warning for an established medication, as well as rapid adoption of new drugs and drug classes during this period. However, the explanation for provider behav-

iors cannot be fully determined in the present analysis, but merit further investigation.” ■

To view an interview with Dr. Mehta, scan this QR code using your smartphone.

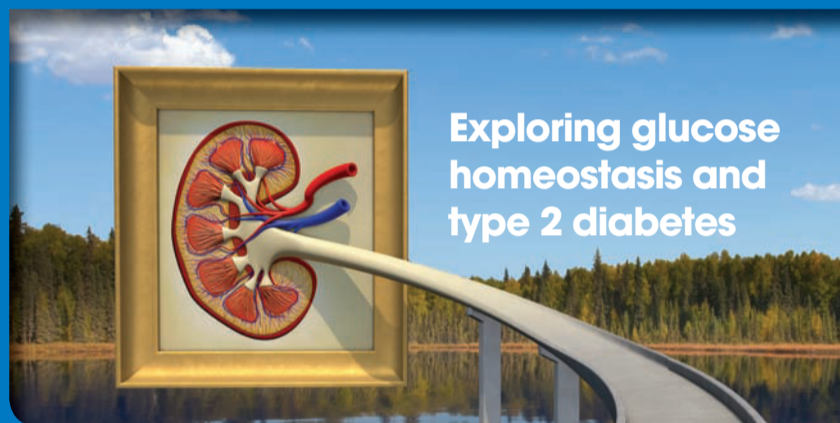


VITALS

Major Finding: The relative proportion of prescriptions for rosiglitazone, pioglitazone, and other medications was 44%, 50%, and 6% in 2005 and 2%, 18%, and 80% in 2010.

Data Source: A study of 7,846 patients with type 2 diabetes at the Joslin Diabetes Center, Boston, who had 9,178 new prescriptions for rosiglitazone, pioglitazone, and other agents from 2001 to 2010.

Disclosures: Dr. Mehta said that he had no relevant financial conflicts to disclose.



Many organs play a role in glucose homeostasis

Fasting glucose levels are controlled by the body within a range of 70-110 mg/dL.¹ Lifestyle choices, including diet and exercise, are essential to help manage glucose levels.^{2,4} Maintaining glucose homeostasis is a multiorgan process involving the muscle, adipose tissue, liver, gastrointestinal (GI) tract, pancreas, brain, and kidney.^{5,6}

The body handles glucose through both insulin-dependent and insulin-independent pathways⁵

Insulin-dependent pathways located in the liver, muscle, and adipose tissue, and insulin-independent pathways, found mostly in the brain, kidney, GI tract, and liver, help create a complex interplay of processes essential for glucose management.^{5,6}

Type 2 diabetes mellitus (T2DM) is characterized by core defects of impaired insulin secretion from the pancreas and increased insulin resistance in the muscle, liver, and adipose tissue.^{5,7} These defects contribute to chronically elevated glucose levels.⁷

Because type 2 diabetes is the leading cause of kidney failure, the kidney is often viewed as a victim of the disease.⁸ But emerging understanding of renal-glucose transporters helps illustrate the ways in which the kidney is an active contributor to the disease as well.^{9,10}

- Sodium-glucose cotransporters (SGLTs) 1 and 2 are expressed in the kidneys, along with facilitative glucose transporters (GLUTs) 1 and 2, where they promote reabsorption of filtered glucose from the renal tubules back into the bloodstream in an insulin-independent process.^{9,10}
- In type 2 diabetes, the renal glucose transport system continues to reabsorb glucose even in the presence of high blood glucose, further worsening hyperglycemia.^{9,11}

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