In Reply — Comparison of Outcomes With Metformin and Sulfonylureas in Chronic Kidney Disease

To the Editor: We thank Weinrauch et al1 for their letter in response to our article. The authors raise an interesting point regarding the absence of glipizide from the sulfonylurea group in our study and in the other studies they cited in their letter. Unfortunately, there were no prescriptions of glipizide in Manitoba during our study period. We agree that the safety of glipizide in individuals with an estimated glomerular filtration rate (eGFR) less than 30 mL/min per 1.73 m² is an important evidence gap that should be addressed given that, unlike other sulfonylureas, they are primarily excreted through the liver rather than the kidneys. We also agree with the authors that metformin use is associated with a higher risk of lactic acidosis in patients with an eGFR less than 30 mL/min per 1.73 m² compared with alternative diabetes management.2

However, it is also the case that sulfonylureas, including glipizide, are associated with a higher absolute risk of major hypoglycemic episodes compared with other oral agents.3,4 Furthermore, current guidelines suggest that newer therapies with a more favorable safety profile such as sodium/glucose cotransporter 2 inhibitors and glucagon-like peptide 1 receptor agonists should not be initiated in patients in later stages of chronic kidney disease.5,6 This leaves clinicians with very few options in this population when starting a diabetes treatment regimen. As such, we recommend an individualized approach which balances the more frequent risks of cardiovascular events and major hypoglycemic episodes of sulfonylureas versus the rare but likely real risk of lactic acidosis with metformin in patients with an eGFR less than 30 mL/min per 1.73 m².

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Angiotensin-Converting Enzyme 2 and the Resolution of Inflammation: In Support of Continuation of Prescribed Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers

To the Editor: The coronavirus disease 2019 (COVID-19) pandemic has resulted in a debate on potential risks vs benefits of certain pharmaceuticals—angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs). A recent letter to the editor, published in the Journal of Travel Medicine,1 outlined a hypothesis stating that the use of ACEIs and ARBs may increase the risk of severe disease in patients with COVID-19. Data suggested that the intravenous infusion of ACEIs and ARBs in experimental animals increased the numbers of angiotensin-converting enzyme 2 (ACE2) receptors in the cardiopulmonary circulation. It is acknowledged that ACE2 receptors are widely dispersed, including lung alveolar epithelial cells, gastrointestinal tract enterocytes, the vascular system, the nasopharynx, kidney, and brain.2 Based on those preclinical data, recognizing that ACE2 receptors are binding sites for coronavirus entry into the body, an increased number of ACE2 receptors was hypothesized to increase the risk of severe