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RESPONSE TO LETTER

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Awareness of Genetic Polymorphism in Drug Metabolizing Enzymes and Transporters May Promote Personalized Type 2 Diabetes Management [Response to Letter]

David M Williams ¹, Jeffrey W Stephens ^{1,2}

¹Department of Diabetes and Endocrinology, Morriston Hospital, Swansea Bay University Health Board, Swansea, SA6 8NL, UK; ²Diabetes Research Group, Swansea University Medical School, Swansea University, Swansea, SA2 8PP, UK

Correspondence: David M Williams, Diabetes Centre, Morriston Hospital, Swansea, SA6 6NL, UK, Tel +441792704078, Email david.williams@doctors.org.uk

Dear editor

We have read the letter to the editor by Zhu and Zhou and would like to thank the authors for their interesting comments. We agree with the authors' observations, which highlight some of the complexities associated with the personalized management of patients with type 2 diabetes (T2D) raised in our recently published review.¹

We concur that further work needs to be undertaken to explore the importance of pharmacogenomics in people with T2D. The authors summarize the implications of pharmacogenomics on the metabolism and therefore the efficacy and sideeffect profiles of older classes of diabetes medications including metformin, sulphonylureas, and thiazolidinediones. However, the implications of pharmacogenomics in the response to newer therapies for T2D remains relatively unknown.^{2,3} Interestingly, DNA and plasma are increasingly obtained prospectively in Phase III clinical trials evaluating newer diabetes pharmacotherapies to determine genetic and biochemical variables which associate with therapy efficacy.^{4,5} Naturally, this has the potential to provide a major opportunity to profile genetic variants which affect the pharmacokinetics of current drug therapies utilised for T2D. This may develop our understanding of the role for pharmacogenomics for both glycemic and non-glycemic benefits (eg, cardiovascular, renal, and hepatic outcomes) and adverse effects associated with various drug treatments for T2D.

Regrettably, the routine use of genetic profiling in people with T2D is not yet readily available to permit these findings a role in the modern treatment of people with T2D. Clearly for there to be a role for pharmacogenomics in a personalized treatment approach, such genomic testing would at least need to be readily available for most patients.³ Clearly, in order to adopt such a personalized approach, a greater evidence base must be developed, and well-designed prospective trials examining gene-drug interactions are needed. We trust that with our developing understanding of the use of pharmacogenomics in the treatment of people with T2D, that easily accessible technology to support clinicians will be developed in future. Indeed, such an approach has the potential to not only improve treatment choices for people with T2D but also to minimise the burden associated with failed drug treatments on healthcare services and the economic costs associated with the prescription of inappropriate medicines.

Again, we would like to thank Zhu and Zhou for their comments in response to our review article and for highlighting the importance of genetic polymorphisms in the treatment response in people with T2D to currently available pharmacotherapies.

Disclosure

The authors report no conflicts of interest in this communication.

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