

***Zhang et al, “Clinical Effects of Antidiabetic Drugs on Psoriasis: The Perspective of
Evidence-based Medicine” (Manuscript Number: 63806)***

Dear Editor,

Thank you for your 30th March, 2021 letter with the referee comments. We thank the reviewer and the editor for useful comments and suggestions. We have substantially revised the paper as requested. The important issues raised by the reviewer have been clarified, corrected, and elaborated. We hope the correction of the revised manuscript is satisfactory and met the requirement of *World Journal of Diabetes*. Please find the revised manuscript and an outline of reply to the referee. We are happy to make any further change if required.

Yours Sincerely

Ching-Wen Chien (Corresponding author)

Reply to Reviewer #1

It was my pleasure to review this (invited) editorial describing the role of several antidiabetic drug classes in psoriasis, a common comorbidity of type 2 diabetes. The article is nicely written and shows the authors' concise effort to inform their readers about the potential antipsoriatic effects of TZDs, metformin and GLP1 receptor agonists, and the need for adequately powered and long-term studies. Although the editorial is presented in detail and with most of the relevant information, I would consider minor changes in a revised version: -The mechanisms of GLP1 receptor agonists that determine their antihyperglycemic and anti-inflammatory effects should be briefly mentioned and refereced (i.e. *Int J Environ Res Public Health*. 2020 May 22;17(10):3664. doi: 10.3390/ijerph17103664.) -"The microenvironment such as inflammation and insulin resistance, as well as genes and epigenetic changes may commonly contribute to the two diseases", this sentence should be properly rephrased. An altered adipose tissue microenvironment in obesity is responsible for insulin resistance and low-grade inflammation at either local or systemic level (e.g. skin). That said, diabetes can relate with a wide spectrum of skin conditions, including but not limited to psoriasis, due to hyperglycemia-induced microvascular damage, altered immune functions, infections, genetic predisposition, etc. In my opinion, this section would benefit of short but deeper insights into the etiological and pathophysiological mechanisms that specifically link diabetes to psoriasis (in all its variants).

Ans. Thanks for the reviewer's useful comments. We apologize for the inadequate descriptions. The descriptions have been corrected. The reference also has been corrected.