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Measuring the effect of common type II diabetes medications (DPP4 inhibitors and sulphonylureas) in combination with metformin on the gut microbiota

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Background: Type II diabetes (T2D) is a disorder characterized by elevated blood glucose levels due to insulin resistance and inadequate insulin secretion. It has become widespread throughout industrialised societies primarily due to the Western diet, which is high in saturated fat and low in dietary fibre. The range of pathways involved in insulin regulated glucose control have suggested a role for gut microbiota mediated mechanisms which represent an alternative target to combat the disease. Indeed, the mechanism of action of the most widely prescribed, first line antidiabetic therapy, metformin, has been linked to effects upon gut microbiota. Other commonly prescribed drugs include sulphonylurea and dipeptidyl peptidase 4 (DPP4) inhibitors (often prescribed in addition to metformin as second line therapies) which have been poorly studied in terms of potential microbiome impact.

Methods: A pilot study (n=20) on T2D patients, all of whom were prescribed metformin in addition to either a DPP4 inhibitor (n=6), a sulphonylurea (n=8) or a combination of the latter (n=6) was conducted. Faecal samples were collected and the gut microbiota profiled through 16S rRNA amplicon sequencing. Information on diet and metabolic blood markers was assessed.

Results: Individuals prescribed sulphonylurea had lower alpha-diversity (measure of species diversity) while this group also differed when comparing the log2-fold differences of the most abundant amplicon sequence variants (representing sequence-based bacterial divisions) in the study.

Conclusions: This provides evidence that these medications may exert an influence on the gut microbiota and the efficacy of these medications when used with first line therapy may depend on microbiome interactions.

Keywords: Type II diabetes (T2D); gut microbiota; sulphonylurea; metformin; dipeptidyl peptidase 4 inhibitor (DPP4 inhibitor)

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Footnote

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