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Mini Review Article

Imeglimin-A New Oral Antidiabetic Agent in the Armamentarium of Resistant type2 Diabetics Specifically Regarding t2d with Correlated Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)-A Minireview

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Abstract

Imeglimin delineates an innovative oral glucose- diminishing agent that possesses a tetrahydrotriazine structure, which in the beginning got recommended in Japan in September2021. Despite both effectiveness in addition to safety of imeglimin in Japanese persons with T2D at the dosage of 1000 mg twice /day got illustrated in phase 3 clinical trials, the Trials ofImeglimin for Efficacy and Safety (TIMES 1–3), it has not been picked up further for evaluation in either United States of America(USA) or European countries ,the former despite certain catastrophies with Ozempic are further trying to launch the other GLP1A- however no attempt has been made to pursue imeglimin despite its structural akinness to metformin-the firstline oral antidiabetic medicine s theexposition which is beyond my comprehension other than pharmaceutical companies dictating the outcomes in this materialistic world.Here we further emphasize why investigators need to pursue scientific research in variable ethnicities to corroborate their efficacy throughout geographical regions scientifically without any bias to accept it as the oral agent when needed in view of it not having the inimical sequelae of metformin-the firstline oral antidiabetic medicine namely lactic acidosis, as well as considerably lesser gastrointestinal(GI) sequelae.

Key Words; *Imeglimin; oral antidiabetic medicine; metformin; gastrointestinal sequelae.*

Introduction

The amount of subjects with type2 diabetes(T2D), is now determined to extend beyond 500 million throughout the world[1]. Insulin treatment is imperative for a considerable subset of such persons; nevertheless, its utilization in T2D might differ on the basis of geographic region. The percentage of patients that utilized insulin might reach 7.4% to 15.5% all overworld in 2030[2]. In Japan, where around 7.6% of people amongst ages 20 to 79 years possesses the disease[3], the consensus- dependent recommendations yielded by the Japan Diabetes Society advocated that patients with type 2 diabetes mellitus(T2DM), begin therapy with an oral hypoglycaemic drug or an injectable drug ,once lifestyle as well as diet modifications are insufficient in reference to sustenance of glycaemic regulation[4], in addition to highlighting the significance of personalized , patient-centric care. In such circumstances, the utilization of insulin has assumed substantial prevalence, with plausibly greater than 10% of T2D subjects delivering insulin either in the form of a monotherapy or in

combination with other oral antidiabetes agents[5]. Of considerable significance is the utilization of insulin in Japanese patients with T2D is greater common among elderly persons; the ones where diagnosis had been made, in considerably younger age, along with possesses greater time period of disease[5]. Despite combination treatments possess the capacity of improvement of glycaemic regulation, they might escalate the risk of inimical sequelae, specifically in the older population as well as those T2D individuals whose presentation was either in the form of T2D complications, that are usually carrying the load of extra treatments in order to treat associated comorbidities. Sequentially, newer alternative antidiabetic medicines was needed to get utilized in combination with insulin in reference to achieving maintained effectiveness in addition to attain greater safety as well as tolerability.

Imeglimin delineates an innovative oral glucose- diminishing agent that possesses a tetrahydrotriazine structure, which in the beginning got recommended in Japan in September2021. Its glucose- diminishing effectiveness got modulated via double mechanistic modes 1) escalation of glucose- based insulin liberation, as well as 2) improving hepatic glucose metabolism via mitochondrial working manipulation [6,7]. Owing to that it has been well acknowledged that type2 diabetes(T2D), in east Asians basically possesses the characteristics of dysfunctional insulin liberation [8], phase 3 clinical trials, the Trials ofImeglimin for Efficacy and Safety (TIMES 1–3), illustrated both effectiveness in addition to safety of imeglimin in Japanese persons with T2D at the dosage of 1000 mg twice /day[9-11].

Since Imeglimin possesses a structural akinness to metformin it is of significance to contrast the actions of the two -the mechanistic modes cited for imeglimin i)escalation of glucose- based insulin liberation,ii) targeting mitochondrial bioenergetics as well as improving mitochondrial working 9 by resulting in improvement of β-cell glucose reactiveness in T2D subjects iv) in addition to resulting in improvement of insulin sensitivity in a rodent model of DM, v)which assisted in glucose tolerance normalization vi) Furthermore , it causes avoidance of demise of human endothelial cells by hampering of the opening of the mitochondrial permeability transition pore.

Whereas mechanistic modes of metformin, the manner summarized by An H, &He L [12], are inclusive of i) lesser metformin quantities (≤80 μM) by activating AMPK via its phosphorylation causes facilitation of the development of the AMPKαβγ heterotrimeric complex (seeFigure 1).

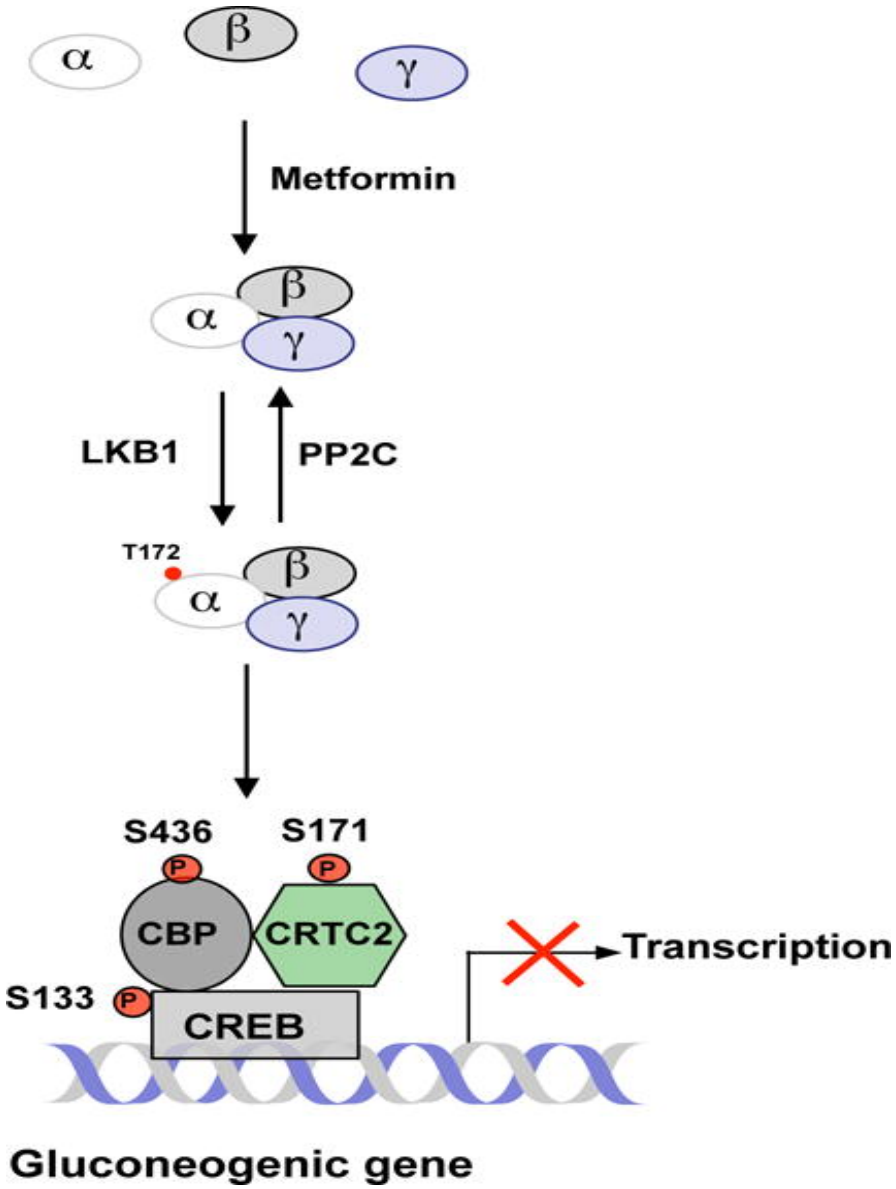


Figure 1 Courtesy ref no. 12- Metformin suppression of hepatic glucose production by promoting the formation of the AMPKαβγ heterotrimeric complex. Metformin increases the phosphorylation of AMPK by promoting the formation of the AMPK heterotrimeric complex. Activated AMPK leads to the phosphorylation of CREB co-activators and the inhibition of gluconeogenic gene expression.

ii) Metformin reppresses hepatic glucose generation via the hampering of mitochondrial respiratory chain complex 1 as well as AMP deaminase.

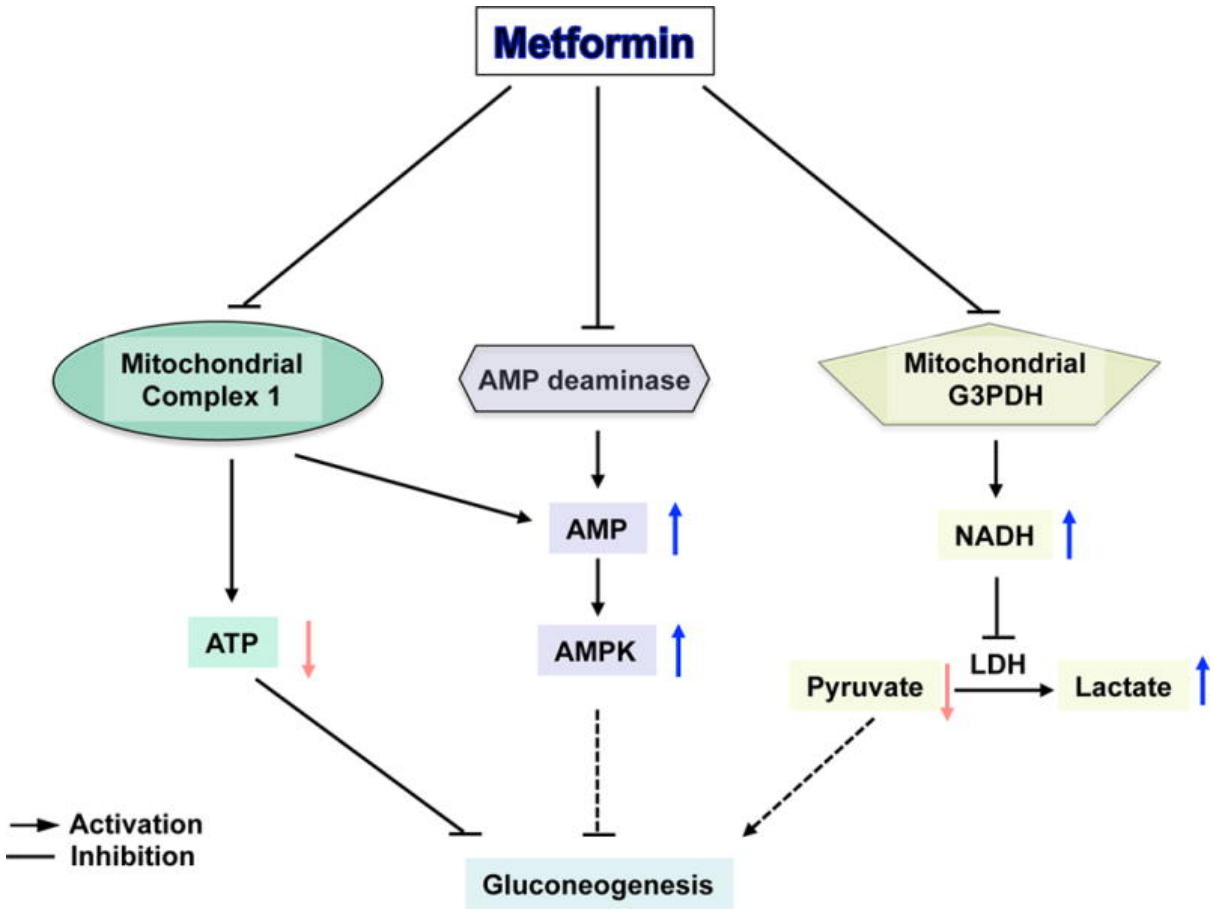


Figure 2 Courtesy ref no. 12- Direct suppression of hepatic glucose production by metformin through the inhibition of mitochondrial respiratory chain complex 1, AMP deaminase, and mitochondrial glycerol 3-phosphate dehydrogenase. Inhibition of mitochondrial complex 1 also leads to an increase in AMP levels or the AMP/ATP ratio, as does the inhibition of AMP deaminase, resulting in the activation of AMPK. However, high metformin concentrations are needed to inhibit the mitochondrial complex and AMP deaminase. Metformin inhibition of mitochondrial glycerol 3-phosphate dehydrogenase (G3PDH) will increase NADH levels in the cytoplasm and suppress the conversion of lactate from pyruvate. This mechanism of metformin action is important for patients with high levels of serum lactate.

In view of the highest metformin dosage prescribed to T2D subjects is equivalent to2.5 g/day, such greater therapeutic dosage possesses the capability of influencing plethora of targets. In the form of an oral drug, i) metformin is capable of causing alteration of the constitution of gut microbiota(GM)[rev in12], ii) which gets followed by activating mucosal AMPK [13],which results in iii) sustenance of intestinal barrier coherence)[rev in12], Collectively, such metformin actions would diminish iv) LPS quantities in the circulation in addition to hepatic LPS quantities , since LPS gets delivered directly to the liver via the portal vein.

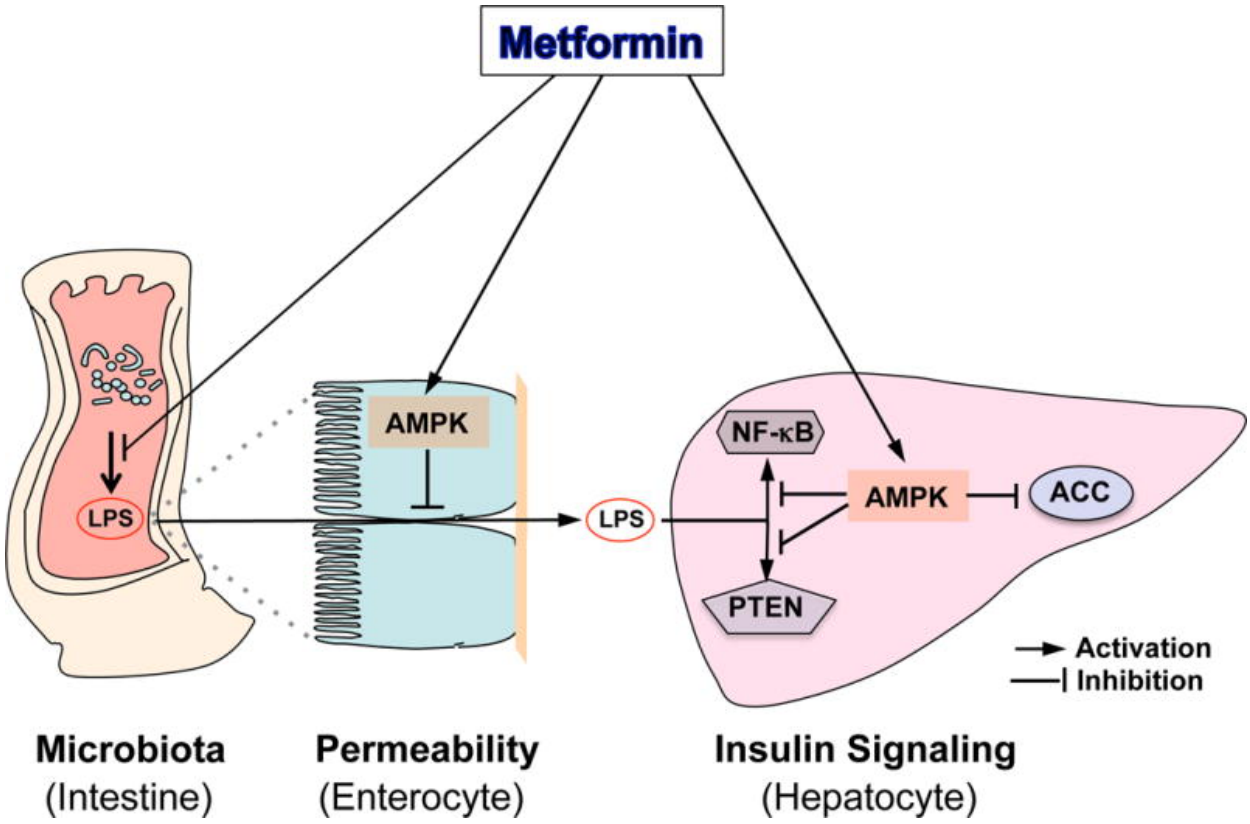


Figure 3 Courtesy ref no. 12- Metformin improves insulin signaling in the liver. Metformin can alter the microbiota in the intestine, resulting in a reduction in LPS production and translocation across the intestinal barrier. Activation of AMPK by metformin also blocks LPS-mediated activation of the NF-κB signaling pathway and PTEN induction.

Additionally, the duodenal AMPK activation by acute metformin therapy would induce the gut–brain–liver axis to regulate hepatic glucose generation [13]. Subsequent to getting delivered to the liver from the intestines, metformin possesses the capability of hampering gluconeogenesis via variable mechanistic modes.

i) Firstly, it has the capability of activating hepatic AMPK, that gets followed by hampering gluconeogenesis by phosphorylating the pivotal co-activators CBP at serine 436 via PKCι/λCBP and (cAMP response element-binding protein (CREB)-regulated transcription coactivator 2) (CRTC2) [14, Figure. 1].

ii) Secondly, greater metformin quantities possess the capacity of repressing hepatic gluconeogenesis by hampering mitochondrial respiratory chain complex 1 to diminish cellular ATP quantities as well asescalating the AMP/ATP ratio [rev in12], owing to the greater -energy needs of gluconeogenesis. ii) Thirdly, hampering of mG3PDH enzymatic activity by metformin would be influencing transportation of NADH from the cytoplasm into mitochondria [rev in12], reppressing gluconeogenesis event from lactate.

Thereby diminishing LPS leakage from the gut along with barricading the activation of the NF-κB pathway, in collaboration with down-regulating of phosphatase as well as tension homolog(PTEN) expression, would lead to an escalation of insulin sensitivity (Figure3). Additionally, metformin delivery would activate AMPK results in escalated fatty acid oxidation by phosphorylating acetyl-CoA carboxylase(ACC) therefore sequentially leading to improvementof insulin sensitivity in the liver [15]. Taken together, gluconeogenesis reppression by metformin's direct actions as well as the indirect improvement of insulin signaling in the liver would result in the mitigation of hyperglycemia in T2D patients. Despite, their studies illustrate that reppression of gluconeogenesis by lesser metformin quantities is via the activation of AMPK by facilitating the generation of the AMPKαβγ heterotrimeric complex in both in vivo as well as in vitro assays[rev in12], in addition to the γ subunit possesses a pivotal part in the phosphorylation of the α subunit at T172 by upstream kinase Liver kinaseB (LKB1). Nevertheless, the exactitude metformin binding site(s) on AMPK subunits along with and the detailed mechanistic modes resulting in the improvement of insulin signaling by metformin continues to beuncharted with need to be elucidated in future studies

The advantages of Imeglimin over metformin is Imeglimin does not lead to fatal complications of lactic acidosis which in rodent models Vial G etal. [16], Illustrated.

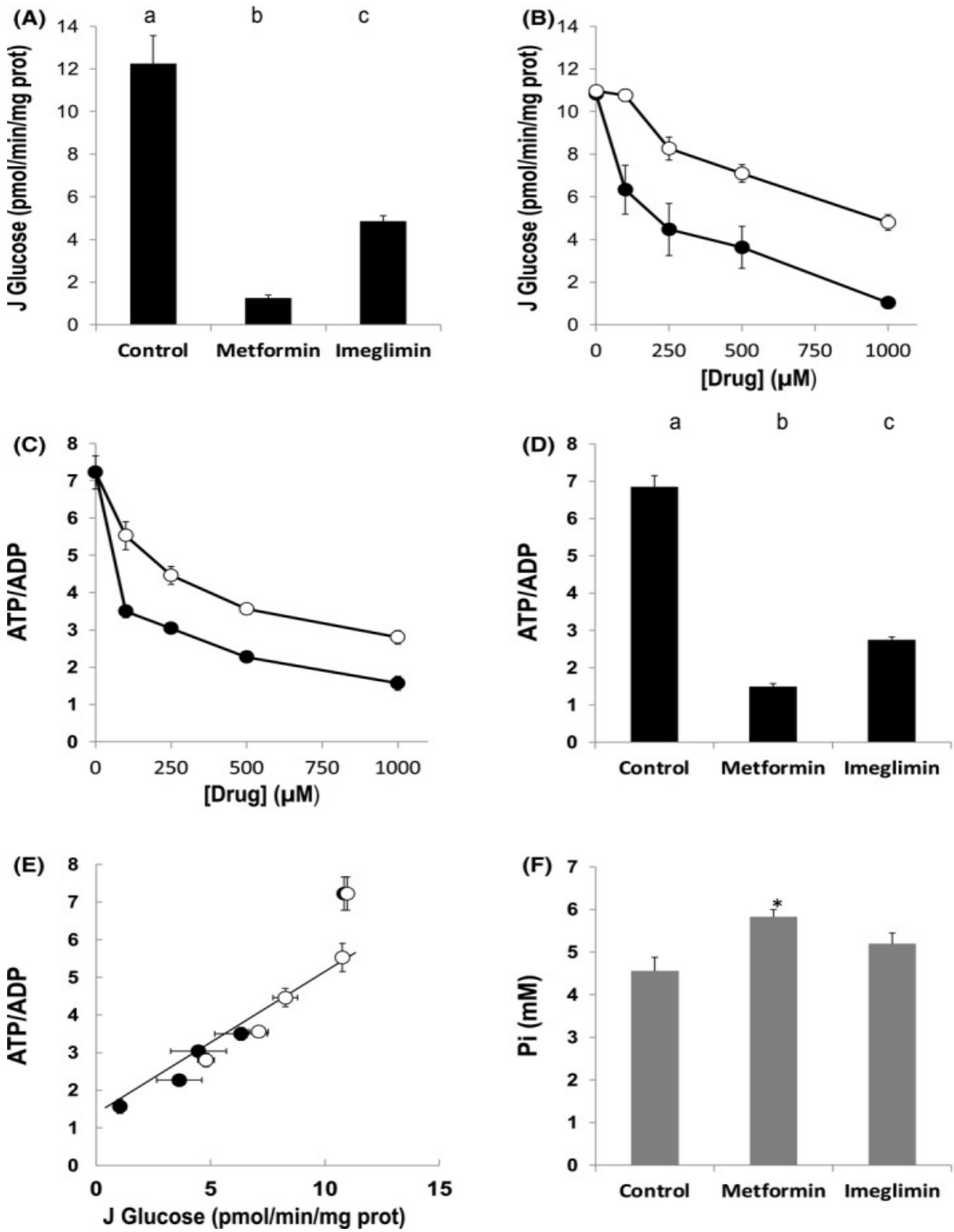
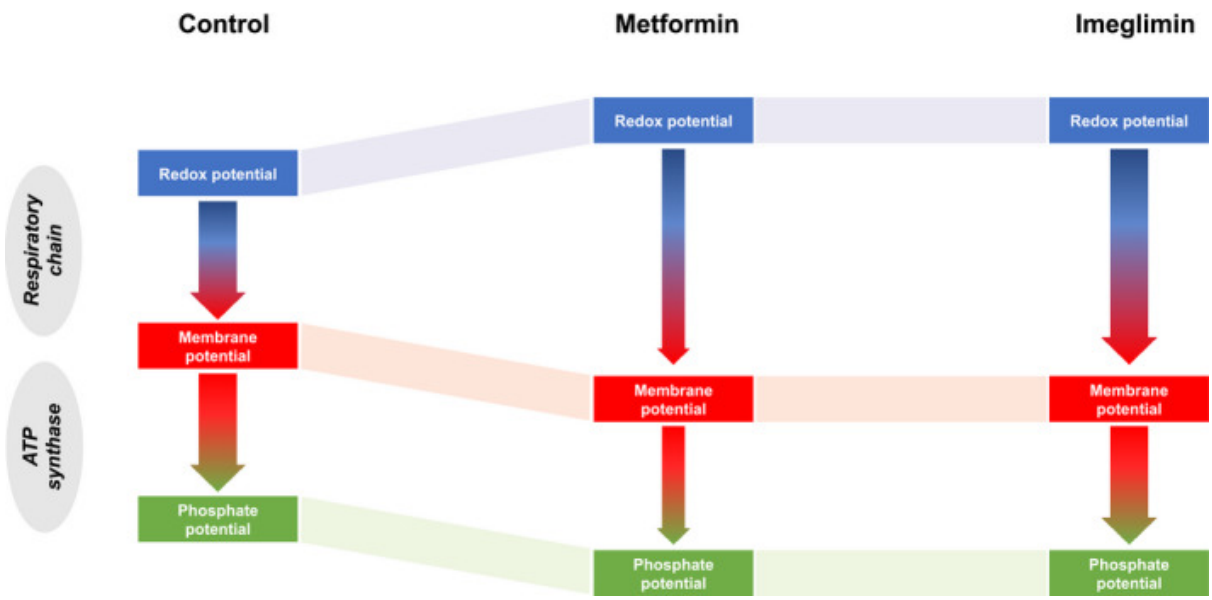


Figure 04 Courtesy ref no. 16- Effect of metformin and imeglimin on glucose production and energy status of primary isolated rat hepatocytes. Hepatocytes incubated overnight in the presence or not of 1 mM metformin or 1 mM imeglimin (Panels A, D and F) or the indicated concentration of metformin or imeglimin (Panels B and C) were washed with PBS and incubated at 37°C for 1 h in a Krebs-Ringer-bicarbonate-calcium buffer

(glucose free) containing 2 mM sodium pyruvate and 20 mM sodium lactate, saturated with O2 : CO2 (19:1). Glucose production, adenine nucleotide and phosphate contents were measured as indicated in Material and Methods. Panel E: data from Panel B and D were plotted. Open circles, imeglimin. Closed circles, metformin. The best correlation (R 2 > 0.97) calculated by computed analysis (Excel®) was obtained when excluding the control condition (data obtained in the absence of metformin or imeglimin). Results are presented as mean ± SEM, n = at least 9 (Panel A), 3 (Panel B), at least 6 (Panel C) at least 15 (Panel D) and 5 (Panel F). Values not connected by the same letter in Panel A and B are significantly different (Tukey-Kramer HSD post hoc test, $p < .05$). Panel F, * significantly different from the proper control (Dunnett's test, $p < .05$)

Figure 4 pointed that that metformin as well as imeglimin hamper gluconeogenesis owing to its lowering of driving force (the ATP/ADP ratio). The variations amongst the two agents apparently once their evaluation are performed from a kinetic perspective. Metformin stimulates a non competing hampering of the respiratory chain implicated for a diminishing in cellular oxygen utilization rate which might result in lactic acidosis regarding metformin intoxication. Conversely, imeglimin stimulates a competing hampering of the respiratory chain involved in an akin fashion thermodynamic consequences without affecting the oxygen utilization rate. In other words, the analogous outcomes is derived by diminishing flux in addition to, force (metformin) or by lowering just force (imeglimin). in reference to a theoretical perspective, such considerably characteristics wouldddiminish the risk to the at least of imeglimin stimulating lactic acidosis. however, that Vial G etal. [16], earlier documented that imeglimin hampers ROS generation stimulated by reverse electron flux via Complex I[rev in 16], pointing that imeglimin does impact Complex I either directly or indirectly.



Courtesy ref no. 16- Since theplausible span amongst the membrane potential(MP) as well as the phosphate potential(PP) is the guiding force for ATP generation, the diminishing in MP stimulated by imeglimin is presumed to be resulting in reduction of ATP synthase activity. Sequentially, ATP generation diminishes untill further reduction kindred to attain the plausible l span required in reference to sustenance of a parallel rate of attainingATP generation, the manner found in control situations. Noticeably, that imeglimin did not affect the ATP synthase activity (the manner evaluated by the oligomycin-sensitive oxygen consumption rate(OCR)—Figure 2B(see ref 16), however diminished both electrical (Figure 3(see ref 16),) along with PP (Figure 1). Thereby Vial G etal. [16], posited that the reduction in the ATP/ADP ratio stimulated by imeglimin is not owing to a reduction in ATP generation, however conversely is an absolutely imperative situation for conservation of a normal rate of ATP generation once the MP diminishes (Figure 5).

Animal studies have further illustrated that imeglimin changes the GM in a fashion analogous to metformin[17], plausibly aggravating gastrointestinal(GI) disruptions.

Fujisawa etal [18], conducted a single-center retrospective cohort study of real-world effectiveness and safety of imeglimin in Japan where in their stratified analysis,HbA1c lowering was meaningfully greater in persons who persisted with biguanides without dose diminishing in contrast with the ones who had undergone dosage diminishing / total cessation of biguanides. While glycemic target attainment rates were comparable amongst groups, baseline achievement was greater in the diminishing group, pointing a greater prominent germane improvement in the non- diminishing group.

In reference to safety, GI inimical sequelae did not vary amongst groups even in pts>74 yrs of age . Nevertheless, further stratification by metformin dosage documented a meaningfully greater incidence of GI processes amongst the ones getting ≥1,000 mg/day. Such observations pointed that imeglimin might safely get combined with biguanides at <1,000 mg/day,whereas greater doses might escalate GI risk.

Further than glycemic control, extra advantages of diabetes treatments are escalatingly highlighted.

Imeglimin, in the form of a first-in-class agent, has been evaluated regarding its plausible actions further than glucose diminishing.

Of the comorbidities, metabolic dysfunction-associated steatotic liverdisease (MASLD) is specifically meaningful, as well as recent therapy algorithms from both the American Diabetes Association/theEuropean Association for the Study of Diabetes in addition to the Japan Diabetes Society advocate this MASLD to be taken into account when making a choice of antidiabetic treatments [19-21].

Furthermore,the biggest limitations continues to be, total studies have been performed on Japanese subjects of diabetes,thereby need for performance as well as corroboration of outcomes is needed.

Satheesan et al[22], recently had attempted to evaluated the efficacy of Imeglimin-based therapies on glycemic control, mitochondrial stress (circulating cell-free mitochondrial DNA [ccf-mtDNA]), and inflammation in view of structural akinness to metformin with pyroptosis effects of metformin cited T2DM -although low number-71 cases[22].

Conclusions

Hereby, we have illustrated, the manner imeglimin- in the form of an innovative oral hypoglycaemic drug might be utilized in T2D therapy. This is particularly meaningful in reference to subjects T2DM who fail to respond to anyof the currently accessible hypoglycaemic drugs inclusive of old first line metformin, orseparate classes of 2nd line antihyperglycemic agents that have been advocated by ADA in addition to EASD; i) DPP-4 inhibitors, glucagon like peptide 1(GLP-1)-RA, Sodium –glucose cotransporter 2(SGLT2) inhibitors, Sulfonylureas, thiazolidenediones as well as the utilization of insulin. Sulphonylureas do not possess any action on insulin sensitivity,but to start with will enhance insulin function[rev by us in extensive details in ref 23, as well as the SGLT2 inhibitors, [rev by us in ref 24,25]GLP-1)-RA , recently we updated GLP-1)-RA in Heart Failure from Liraglutide uptill tirzepatide ,latest triple receptor agonist (retatrutide) that targets glucagon receptor (GCGR), glucose dependent insulin tropic peptide receptor (GIPR) as well as GLP-1R[rev by us in ref 26],nevertheless, certain elderly T2D individuals possess T2D of long time duration illustrated resistance to all treatments insulin, which need greater interventions for which Imeglimin apparently is considerably good in combo with metformin but for GI inimical sequelae,however recently we encountered a T2D individual who generated IBS with metformin as well as was considerably reluctant to take insulin despite addition of SGLT2 inhibitor Empagliflozin greater than dose(25mg) recommendations as without metformin(normal combo is empagliflozin 12.5+ 1gmmetformin) with Dipeptidyl peptidase -4 inhibitor linagliptin 5mg bd where she did not rerspond with fasting blood sugar(FBS) ≥ 250-300mg when she was forced to start actrapid insulin 20mg tid BBF,BL along with BD. Despite, FBS got controlled in fbeginning in comb empagliflozin as in addition to, linagliptin 5mg bdthere was repeated fluctuation owing to not following lifestyle modifications. This case might be fit for insulin with Imeglimin as right now she has been switched to mixtard.

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