MedCrave
Step into the World of Research
Endocrinology & Metabolism International Journal

Review article
Open Access
CrossMark

Drug interactions and drug-food interactions in patients receiving diabetes mellitus treatment

Abstract

Drug interaction is the change of a drug's effects by the preceding and/or concurrent administration of a different substance (precipitant drug). Drug interactions may either intensify or weaken the effects that one or both medications are supposed to have. Any drug's therapeutic, preventative, or diagnostic capabilities may change as a result. A major contributing factor to the prevalence and occurrence of adverse medication responses and adverse drug events might be drug interactions. Patients receiving drug combinations or polypharmacy or suffering from co-morbid diseases like diabetes, hypertension, peptic ulcer, fungal infections, and neurodegenerative disorders have a much higher rate of occurrence and incidence of drug interactions. As these patients receive prolonged and multiple treatments, the risk of drug interactions rises.

Conclusion: Drug interactions are more likely to occur in diabetes patients who are also getting a mix of medications for diabetic complications. As a result, the frequency of drug interactions is fast increasing. Because of its increasing incidence and the corresponding rise in morbidity and mortality, diabetes mellitus has been regarded as a major public health concern globally. The major goal of this review study is to highlight the different medication interactions that may occur between drugs and foods, as well as to record any unfavorable side effects of other treatments used in conjunction with antidiabetic drugs in diabetic patients.

Keywords: polypharmacy, diabetes mellitus, drug-food interaction, drug interaction, adverse drug reaction

Volume 11 Issue 2 - 2023

Victor Duniya Sheneni,[1] Isaac Eleojo Shaibu[2]
[1]Department of Biochemistry, Faculty of Science, Federal University Lokoja, Nigeria
Department of Biochemistry, Faculty of Natural Science, Prince Abubakar Audu University, Nigeria

Correspondence: Victor Duniya Sheneni, Department of Biochemistry, Faculty of Science, Federal University Lokoja, Lokoja, Nigeria,
Email victor.shenen@fulokoja.edu.ng,shenenivicto@gmail.com

Received: July 09, 2023 | **Published:** July 20, 2023

Introduction

A therapeutic combination could result in an unexpected change in the patient's form, which would be exemplified as an interaction with potential clinical implications. An interaction is said to arise when the effects of one drug are altered by the presence of additional drugs, food/drink, and/or environmental essentials at the same time.[1,2] Drug interactions were cited as a significant yet mostly unrecognized source of medication errors. The final result of any combination may be the antagonistic or adverse effects of one or more medications, the synergism or additive effects of one and/or more drugs, the changing of one or more drugs' effects, and/or the generation of idiosyncratic effects. Drug interaction (DI) is the alteration of a drug's (object drug's) effects by the administration of a different drug (precipitant drug) beforehand and/or concurrently. The intended impact of one or both medications may be increased or decreased as a result of drug interactions. Any drug's diagnostic, preventative, or therapeutic activity may change as a result.[2] Finding out the prevalence and frequency of drug interactions in polypharmacy is crucial since they might have major consequences for hospitalized patients. Additionally, it is more important to watch for and identify substances that tend to interact in unfavorable ways.[3]

According to the poll, patients on a few drugs experience drug interactions 3 to 5% of the time, whereas patients taking multiple drugs experience them 20% of the time.[4,5] DIs can occur in a variety of ways, such as pharmacodynamic interactions, in which the receptor effects of various compounds interact to create either a drug's beneficial or harmful effects. A major contributing factor to the frequency and occurrence of adverse drug reactions (ADRs) and adverse drug occurrences might be Dis.[5] Patients receiving drug combinations or polypharmacy or suffering from co-morbid diseases like diabetes, hypertension, peptic ulcer, fungal infections, and neurodegenerative disorders have a much higher incidence and rate of drug interactions. As these patients receive prolonged and multiple treatments, their risk of drug interactions rises.[6]

Diabetes patients frequently also have dyslipidemia, hypertension, and other co-occurring medical conditions. To manage coexisting conditions, they might also need insulin or other oral anti-diabetic drugs, statins to lower cholesterol, fibrates to lower triglycerides, ACEIs or ARBs to treat kidney failure or heart disease, diuretics to lower blood pressure, and aspirin or clopidogrel to prevent heart attacks.[5] Since they typically get various medications for diabetes problems, diabetic patients have been found to be more susceptible to have drug interactions. Drug interactions are becoming more common as a result.

The primary chronic condition known as diabetes mellitus (DM) is defined by insulin's (the pancreatic hormone) faulty regulation of the balance of lipid and carbohydrate metabolism, which raises blood sugar levels. DM is currently one of the biggest hazards to people's health.[7] DM is a major metabolic condition that adversely affects patients' quality of life, health, and life expectancy as well as the health care system.[8,9]

Diabetes mellitus (DM) is a chronic illness defined by a deficiency in insulin secretion (beta cell dysfunction) and/or insulin action (insulin resistance), which impairs the metabolism of glucose and lipids.[9] Due to its widespread prevalence and the resulting rise in morbidity and mortality, diabetes has come to be seen as a significant public health issue on a global scale. As the hyperglycemia condition worsens, increased tissue or vascular damage may cause obesity, hypertension, aging, and the buildup of toxic substances in the vascular endothelium, which can result in the emergence of microvascular problems.[10] According to the International Diabetes Federation, 382 million people worldwide have diabetes, and by 2035, it's predicted that figure will increase to 592 million.[11]

The major cause of numerous serious consequences, including cardiovascular, renal, and other serious comorbidities, is now diabetes mellitus (DM).[12] Chronic hyperglycemia, microvascular pathologies (such as retinopathy, nephropathy, and neuropathy), and macrovascular pathologies (such as coronary artery disease (CAD), hypertension (HT), atherosclerosis, and stroke) are the distinctive characteristics of DM. Over 17.5 million deaths worldwide are also attributable to cardiovascular complications.[13] Unquestionably, one of the most difficult medical issues of the twenty-first century is diabetes.[14] Although there are many interactions that potentially cause hypoglycemia, only a small number have significant effects (Table 1). Almost all other drugs can be safely combined with these agents when used sparingly, with a few significant exceptions.

Table 1 Adverse effects of different medications when used with antidiabetic medicines

Drug or substance class	Drugs containing antidiabetic compounds that have unwanted side effects in diabetic patients	References
Amitriptyline	Increase body weight and blood sugar levels.	21
SSRI (Citalopram, sertraline)	lower blood sugar levels. Boost physical weight.	19
MAOI (moclobemide)	Reduce glycemia. Do not increase body weight.	21
SNRI (Venalafaxine, duloxetine)	Duloxetine increases fasting glycemia, do not increase body weight.	21
NaSSA (mitrazapine)	Increase body weight and glycosylated hemoglobin.	21
St. John's wort (hypericin)	Boost blood sugar levels and gain weight.	21
Itraconazole (antifungal)	when combined with thiazolidinediones, has a hypoglycemic impact.	4
Propranolol (Beta blocker)	Produces hypoglycemic effect with glipizide.	5
Phenylbutazone	Severe hypoglycemia with oral sulfonylureas.	22
Alcohol	Potentiate pre-existing hypoglycemia, acute and chronic alcohol consumption contributing to loss of Glycemic control.	16,22
Fibrates (antihyperlipidemics)	Cause hypoglycemia; also potentiate hypoglycemia by displacing sulfonylureas and rapaglinide from plasma protein binding.	22
Gemifibrozil	When administered with rapaglinide, enhances the activity of rapaglinide which may cause hypoglycemia.	28
Indomethacin	Increase insulin secretion from pancreas, decrease gluconeogenesis, insulin clearance and increase in glucose uptake in periphery	27
Rifampicin, Phenobarbital	They decrease the hypoglycemic activity when admisinstered with sulfonylureas, meglitinides and thiozolidinediones.	28,34
Co-trimoxazole	Coadministration of co-trimoxazole with sulfonylureas increases the risk of hypoglycemia.	46
Cimetidine	Cimetidine may compete with Metformin for renal elimination, which enhances the Metformin level; it may result in hypoglycemia and Metformin associated lactic acidosis.	47,48

Interaction of antifungal agents with antidiabetic agents

Numerous diabetic people are reported to be susceptible to fungus infections, according to epidemiological studies.[4,15] Antifungal medications like fluconazole, itraconazole, miconazole, ketoconazole, etc. are used concurrently with thiazolidinedione (anti-diabetic medications) like pioglitazone or rosiglitazone. Itraconazole is known to impair the Cytochrome P-450 enzyme system, therefore there is a chance that it will interact with other drugs being taken concurrently in a pharmacokinetic way. Strong CYP3A4 inhibitors like azole and derivatives of erythromycin, which include erythromycin, are also known to increase the hypoglycemic effect of repaglinide.[16,17] According to Janadri et al.'s analysis, itraconazole and thiazolidinediones interact when treating diabetes mellitus and fungus infections at the same time.[4,15] Therefore, therapeutic drug monitoring (TDM) is required to optimize the dosage and frequency of these medications, which are used concurrently to protect patients from severe hypoglycemia.

Interaction of antihypertensive agents with antidiabetic agents

One of the most significant and prevalent health issues in diabetes individuals, hypertension is frequently difficult to control and causes significant morbidity and mortality.[5] People with diabetes had increased rates of hypertension, which are likely 1.5–2 times greater than in the overall population. Propranolol and other beta blockers are routinely prescribed as initial treatments for people with hypertension, including those with diabetes mellitus.

A strong second-generation sulfonylurea (anti-diabetic medication) like glipizide moreover stimulates the beta cells in the pancreatic islet of langerhans, increasing insulin secretion and lowering blood sugar levels[5] Weight gain and modification of the beta-receptor mediated insulin production from the pancreatic -cells are two potential methods by which beta blockers may contribute to the development of diabetes.

The inotropic, chronotropic, and vasodilator responses to beta-adrenergic stimulation are proportionately reduced when access to beta-adrenergic receptor sites is restricted by propranolol. The increase in beta adrenoceptor blockade-induced plasma adrenaline and other counter-regulatory hormones during hypoglycemia.[15,18] According to Sen et al.'s research, propranolol greatly increases the hypoglycemic activity of glipizide when given to diabetic mice on a regular basis. While repeated administration of propranolol and glipizide potentiated the hypoglycemic action in diabetic rats. Additionally, they recommend that in order to prevent severe hypoglycemia when treating co-morbid diabetes and hypertension with propranolol and glipizide, the frequency of administration and dosage of glipizide be modified appropriately.[5] It is determined that, when taken with anti-diabetics, anti-hypertensive medications need to have their dosage and frequency adjusted in order to protect patients from severe hypoglycemia.

Interaction of antipsychotics with antidiabetic drugs

The increase in quality of life with the disease and the treatment of anxiety and depression with the administration of diabetes are the main goals of psychiatric care for diabetic patients. The most typical

Citation: Sheneni VD, Shaibu IE. Drug interactions and drug-food interactions in patients receiving diabetes mellitus treatment. *Endocrinol Metab Int J.* 2023;11(2):23–28. DOI: 10.15406/emij.2023.11.00327

psychiatric treatment is antidepressant medication. As well as being used to treat severe diabetic neuropathy, they are also utilized to treat anxiety and despair. No matter whether the patients are given an antidepressant, in this case sertraline, or a placebo, the glycosylated hemoglobin decreases during the entire treatment phase and continues to be considerably reduced throughout depression-free maintenance.[19] Another trial using sertraline found that a subset of diabetics with low incomes experienced a substantial drop in glycosylated hemoglobin following the start of pharmacologic treatment for depression compared to placebo.[20]

It has been determined that when a diabetic patient takes antidepressants, blood glucose, body weight, blood pressure, and renal function all affect glycemia. As a result, it is important to inform the patient in advance that an adjustment to the dosage of their insulin or oral anti-diabetic medications may be required. Selective serotonin reuptake inhibitors (SSRI), monoamine oxidase inhibitors (MAOI), and trazodone cause the least rise in body weight compared to tricyclic antidepressants (TCA) and mirtazapine. TCA was linked to a rise in blood sugar, while SSRI and MAOI caused a drop.[21]

The most widely used TCA increases blood glucose, decreases insulin production, increases hunger for sweets, and increases body weight. TCAs like amitriptyline are even used to treat painful diabetic peripheral neuropathy. SSRI lower blood sugar (even a 30% drop in fasting glycemia has been demonstrated), momentarily lower body weight (fluoxetine is most frequently used as an example), but on long-term usage, they raise body weight. Fluvoxamine's suppression of the CYP 3A4 enzyme (CYP 3A4) may impede the metabolism of oral diabetes medications. Sertraline, fluvoxamine, and fluoxetine can all block CYP 2C9, which can affect how some anti-diabetics, such sulfonylurea and tolbutamide, are metabolized. Combining them can cause hypoglycemia because the antidepressants raise the levels of the antidiabetic medications (sulfonylurea and tolbutamide).[21]

Blood glucose levels are lowered by MAOI, such as moclobemide, which might result in hypoglycemia situations. However, it typically does not result in an increase in body weight. Bupropion is an example of an NDRI that has no effect on how much weight a person gains or loses. The seizure threshold is lowered by higher doses when combined with insulin (the risk of seizures is increased particularly in individuals with concurrent mental anorexia). Bupropion helps people quit smoking and lessens sexual dysfunction. As diabetic people have both diabetes-related sexual dysfunction and an increased risk of cardiovascular disease from smoking, both impacts are very significant. Mirtazapine, a noradrenergic and selective serotonergic antagonist (NaSSA), makes people hungrier and makes them gain weight, both of which are bad for diabetes patients. Additionally, it may result in a rise in glycosylated hemoglobin, which may worsen long-term glycemic management.[21]

Interaction of Phenylbutazone with antidiabetic drugs

When used with oral sulfonylureas, phenylbutazone can result in severe hypoglycemia because it displaces the sulfonylureas from plasma protein binding sites and prevents their metabolic clearance. Tolbutamide's effects on hypoglycemia were most noticeable. One should stay away from phenylbutazone and instead utilize another NSAID.[22] Phenylbutazone should be avoided and prohibited for treating diabetes since using oral hypoglycemic medications with it may result in dangerously low blood sugar levels.

Interaction of Alcohol with antidiabetic drugs

When used with oral sulfonylureas, particularly chlorpropamide, alcohol can result in a reaction similar to that of disulfiram. Flushing, warmth, wooziness, nausea, and tachycardia are all common symptoms. Since drinking alcohol does not always correspond with the occurrence or severity of the reaction, it is preferable to avoid it. Alcohol consumption should be avoided or kept to a minimum for diabetics who are not taking sulfonylureas since it negatively affects glycemic control and has a potential to cause hypoglycemia. Hypoglycemia that already exists can be aggravated. Alcohol intake, both acute and chronic, can influence how some hypoglycemic medications are cleared through the metabolic process, which adds to the loss of glycemic control.[16,22] In order to avoid severe hypoglycemia and contraindicate its use, alcohol should not be consumed when treating diabetes.

Interaction of Antihyperlipidemics with antidiabetic agents

Sulfonylureas and repaglinide may be replaced by fibrate antihyperlipidemics and some beta blockers, which could enhance their effects and possibly result in hypoglycemia. While Nicotinic acid affects glycemic control and may raise insulin resistance, Cholestyramine boosts the hypoglycemic impact of acarbose.[22] Due to the risk of severe hypoglycemia from the combination of these medications with oral hypoglycemic medicines, their usage should be avoided and contraindicated during the treatment of diabetes.

Other drug interactions involving medications used to treat diabetes

A vast variety of drugs were needed as suitable therapy after a diabetes diagnosis. These include drugs for hypertension, glycemic management, antiplatelet treatment, and dyslipidemia. The sheer number of medications available can be overwhelming, so it's critical that patients are well-informed about their prescription schedule.[8,23] When taking various drugs, patients worry about a variety of things, including possible side effects, prescription drug errors, and pharmaceutical costs. It's noteworthy that 58% of patients fear that they may be prescribed drugs that contain drug interactions that will harm their health.[24,25] Patients with moderate to severe renal impairment or those at risk of developing volume depletion should not be taken the medication.[26–29] Oral hypoglycemic agents should be administered in conjunction with these medications with adjustments and manipulation; otherwise, severe hypoglycemia may result.

Drug-food interactions

Increased daily consumption of fruits and vegetables may help avoid the major chronic noncommunicable illnesses, according to data from the World Health Organization (WHO). One of the top ten risk factors for mortality, according to reports, is a diet low in fruits and vegetables.[30] Increased consumption of fruits and vegetables can assist in replacing foods heavy in saturated fats, sugar, or salt. Drug-phytochemical interactions that have been observed, along with interactions with dietary micronutrients, point to a number of potential improvements in therapeutic approaches. However, numerous researches have documented the impact of plant-based meals and herbal remedies on drug bioavailability. It has been hypothesized that significant dietary components and phytochemicals influence drug transporters and metabolizing enzymes, potentially resulting in significant nutrient-drug interactions.[30] Drug-food interactions can have two primary therapeutic effects: either a drug's bioavailability is diminished, which leads to treatment failure, or it is increased, which raises the risk of adverse events and may even trigger toxicities. Drug transporters and drug metabolizing enzymes are critical in changing how drugs are absorbed, distributed, metabolized, and eliminated (ADME).

They have the ability to influence and change the pharmacokinetics and pharmacodynamics of a drug, whether they act separately or in concert. The interaction of drug metabolizing enzymes and transporters is one of the complicating factors that has recently been demonstrated to contribute to the possibility of complex drug interactions.[31] Treatment-food interactions have demonstrated that the choice of medication for diabetic patients may be influenced by the presence of food, which may change the therapeutic efficacy of any treatment and be detrimental to the patient's health.

Syzygium cumini (Jamun)

Syzygium cumini, also known as Jamun (Hindi), is a fruit that is also known as an Indian blackberry. At a dosing level of 50 mg/kg, *Syzygium cumini* (Family: Myrtaceae) also shown a considerable reduction in blood glucose levels. Additionally, rats with diabetes induced by alloxan had their blood glucose levels significantly lower. *Syzygium cumini* affects the GLUT-4, PPAR gamma, and PI3K involved in the transfer of glucose. Activity suggests that *Syzygium cumini* activates PI3K-dependent glucose transport. Oral treatment of *Syzygium cumini* extracts in ethyl acetate and methanol (200 and 400 mg/kg) resulted in a considerable drop in blood glucose levels, according to Shweta and her coworkers.[32] Methanol extracts were discovered to be significantly active at 100 ng/ml dose comparable with insulin and Rosiglitazone, according to a paper that described how different solvent extracts were extracted progressively and then tested for glucose uptake activity at each step.[33,34] Jamun has historically been used to treat diabetes; as such, it should be avoided and contraindicated during the management of diabetes because taking Jamun together with oral hypoglycemic medications can result in life-threatening hypoglycemia.

Momordica Charantia (Karela)

Momordica charantia, often referred to as Karela or Balsam pear, is a tropical vegetable that is a staple of Indian cuisine and has a long history of usage in traditional medicine as a treatment for diabetes.[35] The fruit of *Momordica charantia* is used as a laxative, emetic, stimulant, tonic, and stomachic. The fruit is also helpful in treating gout, rheumatism, and sub-acute liver and spleen illnesses. It is also thought to purify blood and relieve melancholy. Additionally, it has been demonstrated to be efficient in human trials and to have hypoglycemic qualities in animal investigations.[36] According to research, consuming *Momordica charantia* boosts the amount of beta cells in the pancreas, which enhances the body's capacity to create insulin. The fruit has additionally demonstrated the capacity to encourage insulin release by improving cells' glucose absorption and potentiating the effects of insulin. Both healthy and diabetic rabbits can lower their blood glucose levels by taking fresh fruit juice orally (6 cc/kg). Studies on both animals and people have demonstrated how bitter melon lowers blood sugar levels. P-insulin, a polypeptide derived from seeds and fruits, causes rats' blood glucose levels to drop quickly and stabilize.

By boosting glucose absorption and glycogen synthesis in the liver, muscles, and fat cells as well as by enhancing insulin release from pancreatic beta cells and encouraging the repair or development of insulin-secreting beta cells, it lowers blood glucose levels.[35] The effects of bitter melon on insulin sensitivity have been discovered and demonstrated by a recent scientific study at JIPMER in India. Additionally, the Philippine Department of Health issued a circular in 2007 claiming that bitter melon is a scientifically approved herbal medicinal plant that can lower high blood sugar levels. According to this study, 100 mg/kg/day (milligrams per kilogram per day) is equivalent to 2.5 mg of the well-known anti-diabetic medication glibenclamide, which is used twice daily. 50–60 cc of the bitter melon juice taken orallyhas had positive outcomes in clinical trials.[35,36] The juice (bitter melon) can induce diarrhea and stomach ache in extremely high dosages. Bitter melon should not be consumed by young children or anyone who has hypoglycemia since it has the potential to cause or exacerbate low blood sugar or hypoglycemia. *Momordica charantia* stimulates the uterus and should not be used by pregnant women as it may result in an early birth. Bitter melon consumption may enhance the effects of insulin, provide synergistic effects with diabetes medications, and enhance the effects of cholesterol-lowering medications. Furthermore, the usage of bitter melon should be avoided or used with caution in diabetic patients taking/receiving hypoglycemic medicines (primarily Phenformin and chlorpropamide) or insulin,[35] since it may enhance the effects of the treatments and may result in severe hypoglycemia.

Garlic *(Allium sativum)*

Widespread usage of garlic has been made to lower high cholesterol. There is minimal scientific evidence to support the use of garlic in the treatment of a wide range of other diseases (including cancer, hypertension, atherosclerosis, diabetes, fungal infections, myocardial infarction, and peripheral vascular disease). Garlic extracts combined with water or a variety of other organic solvents have been demonstrated to have a hypoglycemic effect on oral glucose tolerance in both healthy and diabetic rabbits caused by alloxan.[37] It has also been claimed that garlic oil has a hypoglycemic impact on diabetic animals and people.[38–40] Because both glimepiride and garlic have hypoglycemic characteristics, co-administering the two medications led to tight glycemic control,[41] In comparison to insulin and glibenclamide, Sheela and Augusti found that the sulfur-containing amino acid S-allyl cysteine sulfoxide (alliin) in garlic had a greater ability to manage the diabetes condition in rats.[42]

According to Eldi et al., oral administrations of the garlic extract to diabetic rats resulted in a notable reduction in the levels of serum glucose, triglycerides, and total cholesterol as well as an increase in the levels of serum insulin. According to reports, the garlic extract's antidiabetic impact was more potent than the effects of Glibenclamide injection (600 microg/kg).[43] Taking *Allium sativum* together with chlorpropamide causes hypoglycemia. The risk of hypoglycemia should be taken into consideration when treating diabetic patients.[43] Garlic consumption should be avoided or handled carefully since it may increase the medications' potency and cause severe hypoglycemia.

Patients receiving diabetes medications

The ingestion of hypoglycemic herbs can have an impact on glucose management in both insulin-dependent or type 1 diabetes mellitus (T1DM) and non-insulin dependent (or T2DM) diabetics.[24,25] Aloe vera, also known as A. barbadensis, leaf juice, bitter melon/karela fruit (Momordica charantia), which has been shown to improve glucose tolerance without increasing insulin levels, and fenugreek seeds (Trigonella foenumgraecum), are three commonly used plants with known hypoglycemic effects. More than 400 plants have been traditionally used for their hypoglycemic action. Additionally, according to two clinical investigations, a water-soluble acidic fraction of an ethanol extract of gurmar (Gymnema sylvestre) leaves has the same effects as aloe vera juice and glibenclamide in reducing insulin needs in both T1DM and T2DM.

The intestinal effects of the gum fiber (galactomannans), which also exhibits hypocholesterolemic action, are thought to be responsible for fenugreek's anti-diabetic properties.[6] Patients with diabetes who also take oral hypoglycemics or insulin may get an added effect

from ginseng's hypoglycemic action. Individual differences in how these supplements affect people necessitate no specific adjustments to hypoglycemic doses unless blood glucose changes.[44,45] While there is a chance for additive effects when these herbs are used with hypoglycemic medications, proper patient self-monitoring and open lines of communication between the patient and healthcare provider should prevent issues.[46–48]

Conclusion

It is determined that there is an alarming rate of drug interaction prevalence and occurrence, which is much higher in patients receiving drug combinations or polypharmacy or suffered from co-morbidity of diseases like diabetes, hypertension, peptic ulcer, fungal infections, and neurodegenerative disorders, which require prolonged and multiple treatments, and the risk of drug interaction will increase as they are treated with multiple therapies. As they receive a combination of medications for diabetic problems as well, diabetic patients are suffering due to a larger risk of drug interactions, which is fast increasing the rate of occurrence.

According to our review of the literature, individuals using diabetic medications are more likely to have adverse drug-drug and drug-food interactions since they are undergoing many therapies for the management of diabetic complications and other related conditions. so that the doctor and other medical professionals can inform the patient about the medicine, drug interactions with food and other drugs or with the medication, and drug-related concerns. This will aid in preventing and reversing drug-drug and drug-food interactions that are connected to anti-diabetic medication. This review study outlined and highlighted the numerous pharmacological interactions that are likely to occur between drugs and foods, as well as reported any unfavorable side effects of additional treatments linked to antidiabetic therapies in diabetic patients.

Acknowledgments

None.

Conflicts of interest

Authors declare that there is no conflict of interest exists.

Funding

None.

References

1. Radhika B, Subash V, Ramaiyan D. A pharmacokinetic interaction of pioglitazone and its clinical applications a short review. *Int J Pharm Sci Letters*. 2012;2(1):1–9.
2. Theodosios DF, Evangelos NL, Moses SE. Dapagliflozin in patients with type 2 diabetes mellitus. *Ther Adv Endocrinol Metab*. 2015;6(1):29–41.
3. Matheny C, Lamb M, Brouwer K, et al. Pharmacokinetic and pharmacodynamic implications of P-glycoprotein modulation. *Pharmacotherapy*. 2001;21(7):778–96.
4. Janadri S, Ramachandra SS, Kharya MD. Influence of itraconazole on antidiabetic effect of thiazolidinedione in diabetic rats. *International Journal of Pharmacy and Pharmaceutical Sciences*. 2009;1(1):119–24.
5. Sen. A study on drug-drug interaction between anti-hypertensive drug (propranolol) and anti-diabetic drug (glipizide). *Annals of Biological Research*. 2010;1(3):35–40.
6. Ismail MYM. Herb-drug interactions and patient counseling. *International Journal of Pharmacy and Pharmaceutical Sciences*. 2009;1(1):151–61.
7. Georgoulis M, Meropi D. Kontogianni and Nikos Yiannakouris. Mediterranean diet and diabetes prevention and treatment. *Nutrients*. 2014;6(4):1406–1423.
8. Kasichayanula S, Liu X, Griffen S, et al. Effects of rifampin and mefenamic acid on the pharmacokinetics and pharmacodynamics of dapagliflozin. *Diabetes Obes Metab*. 2013;15(3):280–283.
9. Hosseini A, Abdollahi M. Diabetic neuropathy and oxidative stress therapeutic perspectives. *Oxidative Medicine and Cellular Longevity*. 2013;1–15.
10. Fowler MJ. Microvascular and macrovascular complications of diabetes. *Clinical Diabetes*. 2008;26(2):77–82.
11. International Diabetes Federation (IDF), IDF Diabetes Atlas, 6th Edition. *Brussels International Diabetes Federation*. 2013.
12. Vlassara H, Striker GE. Advanced glycation end products in diabetes and diabetic complications. *Endocrinology and Metabolism Clinics of North America*. 2013;42(4):697–719.
13. Banerjee M, Vats P. Reactive metabolites and antioxidant gene polymorphisms in type 2 diabetes mellitus. *Redox Biology*. 2014;2:170–177.
14. Shrestha P, Ghimire L. A review about the effect of life style modification on diabetes and quality of life. *Global Journal of Health Sciences*. 2012;4(6):185–90.
15. Sunilkumar B, Lucia P, Miglani BD. Possible drug interactions in hospitalised patients. *The Ind J Hos Pharm*. 1998:91–93.
16. Triplitt C. Drug interactions of medications commonly used in diabetes. *Diabetes Spectrum*. 2006;19(4):202–211.
17. Hatorp V, Hansen KT, Thomsen MS. Influence of drugs interacting with cyp3a4 on the pharmacokinetics, pharmacodynamics, and safety of the prandial glucose regulator repaglinide. *J Clin Pharmacol*. 2003;43(6):649–660.
18. Imamura A, Kusunoki M, Ueda S, et al. Impact of voglibose on the pharmacokinetics of dapagliflozin in Japanese patients with type 2 diabetes. *Diabetes Ther*. 2013;4(1):41–49.
19. Lustman PJ, Clouse RE, Nix BD, et al. Sertraline for prevention of depression recurrence in diabetes mellitus: a randomized, double-blind, placebo-controlled trial. *Arch Gen Psychiatry*. 2006;63(5):521–529.
20. Echeverry D, Duran P, Bonds C, et al. Effect of pharmacological treatment of depression on a1c and quality of life in low-income hispanics and african americans with diabetes: a randomized, double-blind, placebo-controlled trial. *Diabetes Care*. 2009;32(12):2156–2160.
21. Komorousova J, Jankovec Z. Antidepressant drug use in patients with diabetes mellitus type 1-the effect of medication on mental problems and glycemic control. *Effects of Antidepressants*. 2012.
22. Draft. Hypoglycemic drug interactions. *The Rx Files Q and A Summary*. 2001.
23. Kasichayanula S, Chang M, Liu X, et al. Lack of pharmacokinetic interactions between dapagliflozin and simvastatin valsartan warfarin or digoxin. *Adv Ther*. 2012;29(2):163–177.
24. Huri Z, Ling C. Drug-related problems in type 2 diabetes mellitus patients with dyslipidemia. *BMC Public Health*. 2013;13(1):1192.
25. American diabetes association. Standard of medical care in diabetes. *Diabetes Care*. 2013;35(1):511–563.
26. Kirsten KV, Hege SB, Tron AM, et al. Polypharmacy as commonly defined is an indicator of limited value in the assessment of drug-related problems. *Brit J Clin Pharmacol*. 2006;63:2187–2195.
27. Murad M, Coto Yglesias F, Wang A, et al. Drug induced hypoglycemia a systematic review. *J Clin Endocrinol Metab*. 2009;94(3):741–745.

28. Albader W. Drug interactions commonly encountered in patients with diabetes. *Drug info Dasman Diabetes Institute*. 2012;1(50):1–4.
29. Kasichayanula S, Liu X, Zhang W, et al. Effect of a high fat meal on the pharmacokinetics of dapagliflozin a selective sglt2 inhibitor in healthy subjects. *Diabetes Obes Metab*. 2011;13(8):770–773.
30. Yuan R, Parmelee T**,** Balian JD, et al. In vitro studies experience of the food and drug administration. *Clin Pharmacol Ther*. 1999;66(1):9–15.
31. Alan S, Nies S, Spielberg P. Principles of therapeutics goodman and gilman's the pharmacological basis of therapeutics. 10th edn. *Megraw Hill NewYork*. 2001:45–65.
32. Sharma S, Mehta BK, Mehta D, et al. A review on pharmacological activity of syzygium cumini extracts using different solvent and their effective doses. *Int Res J Pharm*. 2012;2(12):54–58.
33. Helmstädter A. Syzygium cumini (l.) skeels (myrtaceae) against diabetes-125 years of research. *Pharmazie*. 2008;63(2):91–101.
34. Sahi J, Black CB, Hamilton GA, et al. Comparative effects of thiazolidinediones used for treatment of non-insulin dependent diabetes mellitus. *Drug Metab Dispos*. 2003;31(4):439–446.
35. Kumar SD, Sharathnath VK, Yogeswaran P, et al. A medicinal potency of momordica charantia. *Int J Pharma Sci Rev Res*. 2010;1(2):95–100.
36. Tripathi P, Gupta P, Lal VK. Interaction of momordica charantia with metformin in diabetic rats. *American Journal of Pharmacology and Toxicology*. 2013;8(3):102–106.
37. Jain RC, Vyas SR. Garlic in alloxan-induced diabetic rabbits. *Am J Clin Nutr*. 1975;28(7):684–685.
38. Jalal R, Bagheri SM, Moghimi A, et al. Hypoglycemic effect of aqueous shallot and garlic extracts in rats with fructose induced insulin resistance. *J Clin Biochem Nutr*. 2007;41(3):218–223.
39. Anwar MM, Meki AR. Oxidative stress in streptozotocin induced diabetic rats effects of garlic oil and melatonin. *Comp Biochem Physiol Part A Mol Integr Physiol*. 2003;135(4):539–547.
40. Liu CT, Wong PL, Lii CK, et al. Antidiabetic effect of garlic oil but not diallyl disulfide in rats with streptozotocin-induced diabetes. *Food Chem Toxicol*. 2006;44(8):1377–1384.
41. Mittal P, Juyal V. Drug-dietary interaction potential of garlic on glimepiride treated type 2 diabetic wistar rats. *Journal of Diabetology*. 2012.
42. Sheela CG, Augusti KT. Antidiabetic effects of s-allyl cysteine sulphoxide isolated from garlic allium sativum linn. *Indian J Exp Biology*. 1992;30(6):523–526.
43. Eidi A, Eidi M, Esmaeili E. Antidiabetic effect of garlic allium sativum l in normal and streptozotocin-induced diabetic rats. *Phytomedicine.* 2006;13(9-10):624–629.
44. Koh Y, Kutty FB, Li SC. Drug-related problems in hospitalized patients on polypharmacy the influence of age and gender. *J Therapeut Clin Risk Manag*. 2005;1(1):39–48.
45. Roger PA. Polypharmacy as a risk factor in the treatment of type 2 diabetes. *Diabetes Spectrum*. 2006;19(1):13–16.
46. Tan A, Holmes HM, Kuo YF, et al. Coadministration of co-trimoxazole with sulfonylureas hypoglycemia events and pattern of use. *J Gerontol A Biol Sci Med Sci*. 2015;70(2):247–254.
47. Kimura N, Okuda M, Inui K. Metformin transport by renal basolateral organic cation transporter hoct2. *Pharm Res*. 2005;22(2):255–259.
48. Dawson D, Conlon C. Case study metformin associated lactic acidosis could orlistat be relevant diabetes care. 2003;26(8):2471–2472.

Citation: Sheneni VD, Shaibu IE. Drug interactions and drug-food interactions in patients receiving diabetes mellitus treatment. *Endocrinol Metab Int J.* 2023;11(2):23–28. DOI: 10.15406/emij.2023.11.00327