

Sulfonylureas

Oral insulin secretagogues that close ATP-sensitive potassium channels on pancreatic beta cells to force insulin release regardless of the current blood glucose level, which is exactly why hypoglycemia - not lactic acidosis or ketoacidosis - is the risk that defines this class.

Online guide: <https://www.medguideglobal.com/pharmacology/endocrine-and-metabolic/sulfonylureas>

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Quick revision

One Liner: Sulfonylureas close pancreatic beta-cell KATP channels to force insulin secretion independent of the ambient glucose level, which is precisely why hypoglycemia - most severe with glyburide, least with glimepiride and glipizide - is the defining risk of the class rather than an incidental side effect.

1. Sulfonylureas bind the sulfonylurea receptor (SUR1) on pancreatic beta-cell ATP-sensitive potassium (KATP) channels, closing them and triggering membrane depolarisation, calcium influx and insulin release, which requires functioning beta cells to work at all. [3][1]
2. Because that insulin release happens regardless of the current glucose level, sulfonylureas can cause hypoglycemia in a way metformin, an SGLT2 inhibitor or a DPP-4 inhibitor cannot on their own. [3]
3. Glyburide carries the highest hypoglycemia risk in the class and is specifically flagged for caution or avoidance in elderly patients and those with renal impairment (eGFR under roughly 60), including a Beers Criteria recommendation against its use in older adults. [3][2]
4. Glipizide and glimepiride are shorter-acting or have properties associated with a lower hypoglycemia incidence than glyburide, and are generally preferred over glyburide in elderly patients when a sulfonylurea is used at all. [3]
5. All three drugs' labels reference the University Group Diabetes Program (UGDP) finding of increased cardiovascular mortality with an older sulfonylurea, tolbutamide, compared with diet alone - a warning the labelling states may extend to the whole oral hypoglycemic drug class. [2]
6. Weight gain is a recognised class effect, distinguishing sulfonylureas from the weight-neutral or weight-reducing profile of metformin, SGLT2 inhibitors and GLP-1 receptor agonists. [3]
7. Glyburide is contraindicated in diabetic ketoacidosis and type 1 diabetes, and its label specifically contraindicates concurrent use with bosentan. [2]
8. Secondary failure - declining glycemic response over years of use as beta-cell function continues to decline - is a recognised limitation of sulfonylurea therapy over the long term. [3]

Overview

1. Sulfonylureas were among the first oral agents developed for type 2 diabetes, and their mechanism is the most direct in this hub: they force the pancreas to release insulin. Specifically, they bind the sulfonylurea receptor (SUR1) subunit of the ATP-sensitive potassium (KATP) channel on pancreatic beta cells. Closing that channel depolarises the cell membrane, opens voltage-gated calcium channels, and the resulting calcium influx triggers exocytosis of stored insulin. Because this pathway depends on functioning beta cells to have insulin to release in the first place, sulfonylureas are ineffective in type 1 diabetes and lose effectiveness as a patient's own beta-cell reserve declines. [3]
2. That mechanism is also the reason hypoglycemia, rather than lactic acidosis or euglycemic ketoacidosis, is the risk that defines this class. Insulin secretion triggered by a sulfonylurea does not pause simply because blood glucose has already fallen to a normal or low level, unlike the body's own physiological insulin response. Hypoglycemia risk is not uniform across the three agents covered here, though: glyburide carries the highest risk and the longest duration of action, and is specifically flagged for caution or avoidance in elderly patients and in renal impairment, while glipizide and glimepiride are generally regarded as safer choices when a sulfonylurea is needed in an older or renally impaired

patient. [3][2]

3. A second class-defining feature is age: sulfonylureas share labelling language, inherited from the University Group Diabetes Program (UGDP) study of the older agent tolbutamide, warning of increased cardiovascular mortality compared with diet alone or diet plus insulin, and stating that this warning may apply to the sulfonylurea class as a whole. That warning long predates the newer classes in this hub and is part of why sulfonylureas, once a default second-line choice after metformin, are now more often positioned behind SGLT2 inhibitors and GLP-1 receptor agonists in patients who have cardiovascular or renal risk that those newer classes specifically address. [2][4]

4. Weight gain and secondary failure round out the practical limitations of this class. Weight gain follows directly from the increase in circulating insulin the drugs are designed to produce, in contrast to the weight-neutral or weight-reducing effect of metformin, SGLT2 inhibitors and GLP-1 receptor agonists. Secondary failure describes the gradual loss of glycemic response over years of therapy as the underlying beta-cell decline of type 2 diabetes continues regardless of the drug being used to stimulate the cells that remain. [3]

Classification and drug examples

Intro: All three agents covered here are second-generation sulfonylureas, more potent at lower doses than the first-generation agents (such as tolbutamide and chlorpropamide) that are now rarely used. They differ mainly in duration of action and hypoglycemia risk rather than in their basic mechanism.

Name: Second-generation sulfonylureas

Note: The three agents in routine current use; first-generation sulfonylureas such as tolbutamide and chlorpropamide are largely of historical and exam interest, chiefly as the source of the UGDP cardiovascular mortality warning inherited by the whole class.

1. Name: Glipizide

1. Glucotrol

2. Glucotrol XL

1. Route: Oral

1. Note: Shorter-acting than glyburide, generally associated with a lower hypoglycemia incidence and often preferred in elderly patients. [1][3]

2. Name: Glyburide

1. Glibenclamide

2. Glynase

3. DiaBeta

4. Micronase

2. Route: Oral

2. Note: The longest-acting and highest hypoglycemia-risk agent of the three; use in the elderly or in renal impairment is specifically discouraged. [2][3]

3. Name: Glimepiride

Amaryl

3. Route: Oral

3. Note: Once-daily dosing with a hypoglycemia and cardiac-safety profile generally regarded as more favourable than glyburide's. [3] [3]

Mechanism of action

Summary: Pancreatic beta cells sense circulating glucose through its metabolism, which raises the intracellular ATP-to-ADP ratio and closes ATP-sensitive potassium (KATP) channels, a physiological trigger for insulin release. Sulfonylureas bypass the glucose-sensing step entirely by binding the sulfonylurea receptor (SUR1) subunit of the same KATP channel directly, closing it regardless of the current glucose concentration. That is the whole mechanistic story, and it is also why the class cannot work in the absence of a functioning beta-cell population, and why

hypoglycemia is a direct, mechanism-driven risk rather than an off-target effect.

Target: SUR1 subunit of the ATP-sensitive potassium (KATP) channel on pancreatic beta cells

Cidal Static: Not applicable - an insulin-secretagogue effect on the patient's own pancreatic beta cells, not an action against a pathogen

Kill Kinetics: Onset within hours of a dose; effect is present for as long as the drug remains at the receptor and requires a functioning beta-cell population to occur at all

1. Step: 1

1. Title: The sulfonylurea binds SUR1 on the beta-cell KATP channel

1. Detail: Unlike glucose, which closes the channel indirectly through its own metabolism, the sulfonylurea binds the SUR1 regulatory subunit of the channel directly. [3]

2. Step: 2

2. Title: The KATP channel closes

2. Detail: Channel closure occurs independent of the ambient glucose concentration, which is the mechanistic root of the class's hypoglycemia risk. [3]

3. Step: 3

3. Title: The beta-cell membrane depolarises

3. Detail: Closing the potassium channel reduces potassium efflux, depolarising the cell membrane. [3]

4. Step: 4

4. Title: Voltage-gated calcium channels open

4. Detail: Depolarisation opens voltage-gated calcium channels, allowing calcium influx into the beta cell. [3]

5. Step: 5

5. Title: Insulin is secreted by exocytosis

5. Detail: The rise in intracellular calcium triggers exocytosis of insulin-containing secretory granules, releasing insulin into the circulation regardless of whether the patient's glucose is currently high, normal or low. [3][2]

Major clinical uses

1. Drug: Glipizide

1. Organism: Not applicable

1. Indication: Adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus

1. Line: second-line, often after metformin, or in patients who cannot take metformin

1. Note: Extended-release (Glucotrol XL) once-daily formulation available; often preferred over glyburide in older adults when a sulfonylurea is chosen. [1]

2. Drug: Glyburide

2. Organism: Not applicable

2. Indication: As an adjunct to diet and exercise for improving glycemic control in adults who have type 2 diabetes mellitus

2. Line: second-line; generally avoided as first sulfonylurea choice in elderly or renally impaired patients

2. Note: Also used off-label in selected cases of gestational diabetes, though this is not its FDA-labelled indication. [2][3]

3. Drug: Glimepiride

3. Organism: Not applicable

3. Indication: Adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus, alone or in combination with metformin or insulin

3. Line: second-line; once-daily dosing supports adherence

3. Note: Labelled with a lower cardiac-tissue affinity than some older sulfonylureas, discussed in some literature as a reason to favour it over glyburide. [3]

4. Drug: All three as a class

- 4. Organism: Not applicable
- 4. Indication: Type 2 diabetes mellitus not adequately controlled by diet, exercise and, where tolerated, metformin
- 4. Line: typically added after metformin, or used earlier when metformin is contraindicated or not tolerated
- 4. Note: Increasingly positioned behind SGLT2 inhibitors and GLP-1 receptor agonists in patients with cardiovascular, renal or obesity-related risk. [4]

Pharmacokinetics

- 1. Drug: Glipizide
 - 1. Route: Oral, immediate-release or extended-release
 - 1. Absorption: Well absorbed orally; food delays absorption of the immediate-release formulation somewhat
 - 1. Csf Penetration: Not clinically relevant to this drug's mechanism
 - 1. Metabolism: Extensive hepatic metabolism to largely inactive metabolites
 - 1. Elimination: Primarily renal, as inactive metabolites
 - 1. Dose Adjust: Hepatic impairment can prolong action and raise hypoglycemia risk; conservative starting doses are used in elderly and hepatically impaired patients [1]
 - 2. Drug: Glyburide
 - 2. Route: Oral
 - 2. Absorption: Well absorbed orally
 - 2. Csf Penetration: Not clinically relevant to this drug's mechanism
 - 2. Metabolism: Hepatic, to metabolites that retain some hypoglycemic activity, unlike glipizide's largely inactive metabolites
 - 2. Elimination: Renal and biliary/faecal, roughly evenly split
 - 2. Dose Adjust: Renal or hepatic insufficiency raises drug levels and impairs counter-regulatory glucose production, compounding hypoglycemia risk; caution or avoidance is advised in the elderly and in renal impairment [2]
 - 3. Drug: Glimepiride
 - 3. Route: Oral
 - 3. Absorption: Completely absorbed orally
 - 3. Csf Penetration: Not clinically relevant to this drug's mechanism
 - 3. Metabolism: Hepatic, via CYP2C9, to metabolites with reduced but not absent hypoglycemic activity
 - 3. Elimination: Renal and biliary, in roughly comparable proportions
 - 3. Dose Adjust: Dose titration begins conservatively (1 mg) and is adjusted more cautiously in renal impairment; CYP2C9 status can influence individual exposure [3]
1. This page gives no dose regimens by design. Starting dose, titration and maximum dose depend on the specific agent, renal and hepatic function, and concomitant therapy, and belong in a current prescribing reference used by the treating clinician.
2. The key pharmacokinetic distinction driving relative hypoglycemia risk across this class is duration of action and whether hepatic metabolism produces active or inactive metabolites - glyburide's active metabolites and longer effective duration are why it carries the highest risk, while glipizide's largely inactive metabolites contribute to its comparatively shorter, more predictable effect. [1][2]
3. All three depend on adequate hepatic function to be metabolised and adequate renal function to clear the parent drug or its metabolites, so combined hepatic and renal impairment compounds hypoglycemia risk more than either alone. [2][3]

Adverse effects

- 1. Effect: Weight gain
 - 1. Detail: A recognised class effect resulting from the sustained increase in circulating insulin that sulfonylureas are designed to produce. [3]

2. Effect: Mild gastrointestinal upset

2. Detail: Nausea and mild gastrointestinal discomfort are reported, though at a lower rate and severity than with metformin. [1]

3. Effect: Skin reactions

3. Detail: Rash and mild hypersensitivity reactions are reported across the class, generally mild and self-limited. [2]

1. Effect: Hypoglycemia

1. Detail: The defining risk of the class: insulin release triggered by a sulfonylurea does not stop when blood glucose is already normal or low. Risk is highest with glyburide and is amplified by renal or hepatic impairment, advanced age, missed or delayed meals, alcohol use, and concurrent use of other glucose-lowering agents.

1. Action: Elderly and renally impaired patients are generally started at the lowest effective dose, or a shorter-acting agent such as glipizide or glimepiride is chosen over glyburide; patients are counselled on recognising and treating hypoglycemia symptoms promptly. [3][2]

2. Effect: Increased cardiovascular mortality (UGDP-derived warning)

2. Detail: Labelling for oral sulfonylureas references the University Group Diabetes Program finding that tolbutamide was associated with cardiovascular mortality roughly two and a half times that of diet-alone treatment, a warning the label states may apply to the entire oral hypoglycemic drug class.

2. Action: This finding is disclosed as part of the risk discussion the treating clinician has with the patient, particularly when choosing among sulfonylureas versus other glucose-lowering classes with a more favourable modern cardiovascular evidence base. [2]

3. Effect: Hemolytic anemia in glucose-6-phosphate dehydrogenase (G6PD) deficiency

3. Detail: Sulfonylureas, as a class, have been associated with hemolytic anemia in patients with G6PD deficiency.

3. Action: A non-sulfonylurea alternative is considered in patients with known G6PD deficiency. [1]

1. Drug: Glyburide

1. Effect: Contraindicated in diabetic ketoacidosis, type 1 diabetes, and with concurrent bosentan use; carries the highest hypoglycemia risk of the three and is specifically discouraged in elderly and renally impaired patients. [2]

2. Drug: Glipizide

2. Effect: Largely inactive hepatic metabolites give it a more predictable, shorter duration of hypoglycemic effect than glyburide. [1]

3. Drug: Glimepiride

3. Effect: Once-daily dosing and a hypoglycemia and cardiac-safety profile generally regarded in the literature as more favourable than glyburide's, though it is still capable of causing significant hypoglycemia. [3]

Contraindications, precautions and interactions

1. Glyburide: diabetic ketoacidosis, with or without coma; type 1 diabetes mellitus; concurrent use of bosentan; known hypersensitivity to glyburide. [2]

2. Glipizide and glimepiride: known hypersensitivity to the drug or, given the sulfonylurea structure, a documented severe sulfonamide hypersensitivity reaction is a caution some clinicians extend to this class, though the evidence for true cross-reactivity is limited. [1][3]

1. Elderly, debilitated or malnourished patients, and those with adrenal or pituitary insufficiency, are particularly susceptible to the hypoglycemic action of sulfonylureas. [2]

2. Renal or hepatic insufficiency increases hypoglycemia risk by raising drug or active metabolite levels and impairing the body's own counter-regulatory glucose production. [2][3]

3. Glyburide specifically is discouraged in patients 65 years and older and in renal impairment (eGFR under roughly 60), per prescribing caution reflected in geriatric prescribing guidance. [3]

4. G6PD deficiency raises the risk of sulfonylurea-associated hemolytic anemia. [1]

1. With: Other glucose-lowering agents, including insulin

1. Effect: Additive hypoglycemia risk with any concurrently used insulin secretagogue or insulin itself. [3]

2. With: Alcohol
2. Effect: Can potentiate hypoglycemia and, less commonly, precipitate a disulfiram-like reaction with some sulfonylureas. [3]
3. With: Bosentan (glyburide specifically)
3. Effect: Concurrent use is contraindicated with glyburide because of an increased risk of hepatotoxicity when the two are combined. [2]
4. With: Drugs that displace sulfonylureas from plasma protein binding or inhibit their hepatic metabolism (for example, certain sulfonamide antibiotics, fibrates, and CYP2C9 inhibitors for glimepiride)
4. Effect: Can raise free drug levels and increase hypoglycemia risk. [3]

Comparison tables

Title: Same mechanism, different hypoglycemia risk

Caption: All three drugs close the same beta-cell KATP channel; what differs is duration of action, metabolite activity, and the resulting hypoglycemia risk in vulnerable patients.

1. Key: drug
1. Label: Drug
1. Is Row Header: true
2. Key: duration
2. Label: Relative duration of action
3. Key: hypo_risk
3. Label: Hypoglycemia risk
4. Key: elderly_renal
4. Label: Elderly / renal impairment
1. Drug: Glipizide
1. Duration: Shorter-acting, largely inactive metabolites
1. Hypo Risk: Lower than glyburide
1. Elderly Renal: Generally preferred sulfonylurea choice when one is needed [1][3]
2. Drug: Glyburide
2. Duration: Longer-acting, metabolites retain some activity
2. Hypo Risk: Highest in the class
2. Elderly Renal: Use discouraged; a recognised Beers Criteria concern in older adults [2][3]
3. Drug: Glimepiride
3. Duration: Once-daily, intermediate
3. Hypo Risk: Lower than glyburide, higher than none
3. Elderly Renal: Used cautiously with conservative titration [3]

High-yield exam pearls

1. Pearl: Sulfonylureas force insulin release independent of glucose level - that single fact is the mechanistic root of the class's hypoglycemia risk.
1. Why: Every hypoglycemia question in this class traces back to the KATP-channel mechanism bypassing the body's own glucose-sensing step. [3]
2. Pearl: Glyburide is the sulfonylurea to avoid in the elderly and in renal impairment; glipizide and glimepiride are the safer choices when the class is needed at all.
2. Why: This is one of the most consistently tested drug-selection points across geriatric and renal-safe prescribing questions. [3]
3. Pearl: Sulfonylureas require functioning pancreatic beta cells to work.
3. Why: This is why they are ineffective in type 1 diabetes and why their effect erodes over years of type 2 diabetes as

beta-cell reserve continues to decline (secondary failure). [3]

4. Pearl: The cardiovascular mortality warning on sulfonylurea labels traces back to the UGDP study of tolbutamide, an older first-generation agent, not to trial data specific to glipizide, glyburide or glimepiride themselves.

4. Why: It is a class-wide regulatory extension similar in structure to the JAK-inhibitor boxed warning pattern - one historical study's finding extended by caution across a pharmacologically related class. [2]

5. Pearl: Weight gain, not weight neutrality, is the sulfonylurea norm.

5. Why: It is the class effect most often used to contrast sulfonylureas against metformin, SGLT2 inhibitors and GLP-1 receptor agonists on an exam comparison question. [3]

Common exam traps

1. Trap: Assuming all sulfonylureas carry an equal hypoglycemia risk.

1. Correction: Glyburide's longer duration of action and active metabolites give it a materially higher hypoglycemia risk than glipizide or glimepiride, which is why it is specifically discouraged in the elderly and in renal impairment. [2][3]

2. Trap: Believing sulfonylureas can be used in type 1 diabetes because they are oral glucose-lowering agents.

2. Correction: Their mechanism depends on functioning pancreatic beta cells releasing endogenous insulin; type 1 diabetes involves beta-cell destruction, so sulfonylureas are not effective and are contraindicated (as reflected explicitly in glyburide's own label). [2]

3. Trap: Treating sulfa allergy as an absolute, well-evidenced contraindication to every sulfonylurea.

3. Correction: Sulfonylureas share a sulfonamide-derived structure, but the evidence for clinically significant cross-reactivity with antibiotic sulfonamide allergy is limited; this is a caution to weigh individually rather than a hard, uniformly evidenced contraindication. [3]

4. Trap: Assuming secondary failure means the drug itself has stopped working due to a resistance mechanism.

4. Correction: Secondary failure reflects the ongoing decline in the patient's own beta-cell function that underlies type 2 diabetes progression, not a pharmacological resistance mechanism in the drug-receptor interaction. [3]

Self-test questions

1. Question: What is the immediate molecular target of a sulfonylurea on the pancreatic beta cell?

1. The insulin receptor
2. The SUR1 subunit of the ATP-sensitive potassium (KATP) channel
3. GLUT2 glucose transporter
4. AMP-activated protein kinase

1. Explanation: Sulfonylureas bind SUR1, the regulatory subunit of the beta-cell KATP channel, closing it directly rather than through glucose metabolism. [3]

2. Question: Why can sulfonylureas cause hypoglycemia in a way metformin cannot?

1. Sulfonylureas are renally toxic
2. Sulfonylureas trigger insulin release independent of the current blood glucose level, while metformin does not stimulate insulin secretion at all
3. Metformin blocks insulin receptors
4. Sulfonylureas increase glucagon secretion

2. Explanation: Because sulfonylureas close the KATP channel directly rather than in response to glucose-driven ATP changes, insulin is released even when glucose is already normal or low - a mechanism metformin does not share. [3]

3. Question: Which sulfonylurea carries the highest hypoglycemia risk and is specifically discouraged in elderly patients?

1. Glipizide
2. Glimepiride
3. Glyburide
4. All three carry identical risk

3. Explanation: Glyburide's longer duration of action and active metabolites give it the highest hypoglycemia risk of the three, and it is specifically flagged for avoidance in elderly and renally impaired patients. [2][3]

4. Question: The UGDP-derived cardiovascular mortality warning on sulfonylurea labels originated from a trial of which drug?

1. Glyburide
2. Tolbutamide
3. Glimepiride
4. Metformin

4. Explanation: The University Group Diabetes Program studied tolbutamide, an older first-generation sulfonylurea, and its finding of increased cardiovascular mortality versus diet alone is now referenced on labels across the class. [2]

5. Question: Why are sulfonylureas ineffective in type 1 diabetes?

1. Type 1 diabetes patients metabolise the drug too quickly
2. Sulfonylureas require functioning pancreatic beta cells to release insulin, and these are destroyed in type 1 diabetes
3. Sulfonylureas are too weak for type 1 diabetes glucose levels
4. They cause paradoxical hyperglycemia in type 1 diabetes

5. Explanation: The KATP-channel mechanism only produces an effect if there is stored insulin in functioning beta cells to release, which is not the case once beta cells are destroyed in type 1 diabetes. [2]

6. Question: Which adverse effect distinguishes the sulfonylurea class from metformin, SGLT2 inhibitors and GLP-1 receptor agonists?

1. Lactic acidosis
2. Weight gain
3. Euglycemic ketoacidosis
4. Genital mycotic infection

6. Explanation: Weight gain is a recognised sulfonylurea class effect tied to the sustained rise in circulating insulin, in contrast to the weight-neutral or weight-reducing profile of the other classes listed. [3]

Frequently asked questions

1. Question: Why does glyburide get singled out as riskier than the other sulfonylureas?

1. Answer: Glyburide has a longer duration of action and its hepatic metabolites retain some hypoglycemic activity of their own, unlike glipizide's largely inactive metabolites. Combined with reduced renal clearance in older or renally impaired patients, this gives glyburide a materially higher hypoglycemia risk, which is why prescribing guidance and the Beers Criteria specifically discourage its use in the elderly. [2][3]

2. Question: Can someone with a penicillin or sulfa drug allergy safely take a sulfonylurea?

2. Answer: Sulfonylureas share a sulfonamide-derived chemical structure, and some clinicians use caution in patients with a severe sulfonamide antibiotic allergy, but the evidence for genuine cross-reactivity between antibiotic sulfonamides and sulfonylureas is limited. This is a judgement the treating clinician makes based on the severity and nature of the prior reaction, not an automatic, uniformly evidenced contraindication. [3]

3. Question: Why are sulfonylureas used less often now than in the past?

3. Answer: Newer classes - SGLT2 inhibitors and GLP-1 receptor agonists - offer cardiovascular and renal benefits beyond glycemic control and do not carry the same hypoglycemia or weight-gain profile. Current guidance increasingly favours adding those classes early in patients with relevant risk factors, positioning sulfonylureas further down the treatment sequence than they occupied a decade ago, though they remain a low-cost, effective option in appropriate patients. [4]

4. Question: What is secondary sulfonylurea failure?

4. Answer: It describes the gradual decline in a sulfonylurea's glycemic effect over months to years of use, reflecting the ongoing loss of pancreatic beta-cell function that characterises type 2 diabetes itself rather than any change in how the drug works at the receptor level. It typically prompts addition of, or switch to, another glucose-lowering class rather

than escalating the sulfonylurea dose indefinitely. [3]

References

- [1] GLUCOTROL XL (glipizide) - prescribing information. DailyMed, U.S. National Library of Medicine .
<https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=eed99b60-d043-4249-9b2a-f05e46fb588d>
- [2] GLYBURIDE tablet - prescribing information. DailyMed, U.S. National Library of Medicine .
<https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=33418bba-c5ea-4c0f-9d28-31dc2724c6c3>
- [3] Sulfonylureas. StatPearls Publishing / NCBI Bookshelf 2023. <https://www.ncbi.nlm.nih.gov/books/NBK513225/>
- [4] 9. Pharmacologic Approaches to Glycemic Treatment: Standards of Care in Diabetes-2026. American Diabetes Association, Diabetes Care 2026.
https://diabetesjournals.org/care/article/49/Supplement_1/S183/163934/9-Pharmacologic-Approaches-to-Glycemic-Treatment

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