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Glucose-Regulating Effects of H1 Antihistamines: A Comprehensive Systematic Review of Clinical and Preclinical Evidence

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Abstract

Histamine H1-receptor antagonists (H1 antihistamines) are widely used for allergic disorders, and studies have reported mixed effects of these drugs on glucose-related outcomes. This systematic review summarised the glucose-regulating effects reported for H1 antihistamines in human, animal and relevant experimental studies. PubMed was searched on 9 October 2026 (614 records) without filters, and Google Scholar was searched as a supplementary source. After removal of duplicates and unrelated or non-original records, 94 records were screened by title and abstract, and 20 studies were included: six human studies, nine animal studies and five biochemical, cellular or mechanistic studies. Risk of bias was assessed with RoB 2, ROBINS-I and the SYRCLE tool according to study design. Heterogeneity and missing data precluded meta-analysis, so a narrative synthesis was used. In humans, one short study reported higher post-load glucose with cetirizine, a small historical study reported lower fasting glucose with terfenadine, and add-on trials in diabetic kidney disease reported mainly renal benefits, with an HbA1c difference in one levocetirizine trial. Animal studies reported improved glucose tolerance or insulin resistance with cetirizine, levocetirizine and ketotifen in some models, higher fasting glucose with desloratadine, and no clear glucose lowering with bilastine. Fexofenadine inhibited recombinant human DPP-4 in an earlier assay (IC50 4.6 ± 1.0 μM). The available literature does not establish H1 antihistamines as a uniform class of glucose-regulating agents, and improvements in renal or inflammatory markers should not be read as direct glycaemic benefit. Well-designed clinical studies with clearly defined glycaemic outcomes are needed.

Keywords: h1 antihistamines, glucose regulation, diabetes mellitus, systematic review, fexofenadine, dpp-4

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1. Introduction

Diabetes mellitus is a chronic metabolic disorder in which blood glucose stays above the normal range because insulin secretion, insulin action, or both are impaired. Persistent hyperglycaemia can damage the kidneys, eyes, nerves and blood vessels. Many effective glucose-lowering medicines exist, yet long-term management remains difficult, and research continues into the pathways that control glucose and into compounds that might act on them.

One established pathway involves dipeptidyl peptidase-4 (DPP-4), an enzyme that degrades the incretin hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP). Inhibiting DPP-4 prolongs incretin activity and improves glycaemic control in type 2 diabetes (Kasina and Baradhi, 2023).

H1 antihistamines are widely used for allergic rhinitis and urticaria. Second-generation agents such as cetirizine, levocetirizine, loratadine, desloratadine, fexofenadine and bilastine are generally less sedating than first-generation drugs, so exposure in the general population is high. Their purpose is allergy control, not diabetes treatment. Even so, experimental and clinical reports have raised the question of whether some of them change glucose-related outcomes, and the findings point in different directions. A short clinical study reported a significant rise in postprandial glucose with cetirizine (Lal, 2000), whereas other reports describe lower glucose or better insulin sensitivity with other agents. A virtual-screening study identified fexofenadine as a candidate DPP-4 inhibitor and reported inhibition of recombinant human DPP-4 in vitro (Almasri et al., 2019). Clinical trials have also tested fexofenadine and levocetirizine as add-on treatments in diabetic kidney disease (El-Fatatry et al., 2024; Rizk et al., 2025).

The evidence is scattered across human, animal, cellular and biochemical studies that differ in design, drug, dose, duration and outcome. Some studies measure glucose directly, whereas others measure renal or inflammatory markers that are related to diabetes but are not measures of glucose control. Without a systematic approach, the direction and strength of the evidence are easily overstated. This review therefore aimed to identify and summarise the glucose-related outcomes reported for H1 antihistamines, to describe how consistent and reliable the evidence is, and to separate direct glycaemic outcomes from related renal, inflammatory and mechanistic outcomes.

2. Methodology

2.1 Design and reporting

A systematic review with narrative synthesis was carried out and reported following the PRISMA 2020 statement (Page et al., 2021). The review question was: what effects have H1-receptor antagonists been reported to have on glucose regulation and related metabolic outcomes in human, animal and relevant experimental studies, and how consistent and reliable is the available evidence? The review protocol was not registered in a public protocol registry.

2.2 Eligibility criteria

Eligibility was defined before screening. The population was human participants, animal

models and in-vitro systems. The intervention was an H1 antihistamine (H1-receptor antagonist). The comparator was placebo, no treatment, standard therapy or other control, as reported by each study. The outcomes were measures of glucose metabolism or antidiabetic effect.

Studies were included if they investigated an H1 antihistamine; reported an outcome related to glucose metabolism or antidiabetic effects (blood or fasting glucose, HbA1c, glucose tolerance or oral glucose tolerance test, insulin, insulin resistance, HOMA-IR or HOMA-β, hypoglycaemic effect, or diabetes and metabolic outcomes); were human, animal or in-vitro studies; were relevant to the review question; and gave enough information to determine what was studied.

Studies were excluded if they were unrelated to glucose or metabolic outcomes; concerned H1 antihistamines only for allergy symptoms without a metabolic outcome; were pharmacokinetic studies without a relevant glucose or metabolic outcome; were reviews, editorials or commentaries used as primary evidence; were conference abstracts from which sufficient data could not be obtained; were duplicate publications; or involved a different drug class without an H1 antihistamine intervention. Animal and in-vitro studies were not excluded.

2.3 Information sources and search strategy

PubMed was searched on 9 October 2026 without filters, and the search returned 614 records. The search combined two groups of terms with AND. The first group named H1 antihistamines (the MeSH term Histamine H1 Antagonists, free-text terms for the drug class, and 18 individual drugs from cetirizine to cyproheptadine). The second group named glucose-related outcomes (for example blood glucose, fasting glucose, HbA1c, glucose tolerance, insulin resistance, HOMA-IR, insulin secretion, diabetes and hypoglycaemic). Terms within each group were combined with OR. The full string is given in Appendix 1. Google Scholar was also searched as a supplementary source, but its search terms, date and number of records were not recorded separately, and its results largely overlapped with PubMed.

2.4 Study selection

Thirty duplicate records were removed, and a further 490 records that were unrelated to glucose outcomes or were not original research were removed, which left 94 records. These were screened by title and abstract against the eligibility criteria, and 20 studies were retained. The full texts of 15 of these studies were available and were checked. The remaining five were assessed from abstracts or article previews. No study was excluded at the full-text stage. The author screened all records as first reviewer, and a second reviewer (the supervisor) cross-checked the selection decisions.

2.5 Data extraction and outcomes

For each study, the author, year, evidence type, intervention, population or model, sample size, duration, outcomes and available results were recorded. Details were cross-checked against the available article pages, abstracts and previews. Where means, variability, confidence intervals, sample sizes or p-values were unavailable or conflicted between sources, they were not estimated. Direct glycaemic outcomes were fasting glucose, post-load glucose, glucose

tolerance, HbA1c, and insulin and insulin-resistance indices. Related outcomes were urinary albumin-to-creatinine ratio (UACR), eGFR, renal biomarkers, inflammatory and oxidative-stress markers, antiglycation endpoints, and insulin synthesis. DPP-4 inhibition was treated as a mechanistic outcome and not as proof of clinical glucose lowering.

2.6 Risk of bias

Risk of bias was assessed with a tool matched to each design: RoB 2 for randomised human trials, ROBINS-I for non-randomised human intervention studies, and the SYRCLE tool for animal studies. In-vitro and biochemical studies were appraised with a structured approach, with each domain judged as yes, no, unclear or uncertain. Unclear reporting was treated as uncertainty and not as low risk of bias. Risk of bias was assessed by the first author and the supervisor.

2.7 Synthesis

A narrative synthesis was used, and results were grouped by evidence type (human, animal, and biochemical, cellular or mechanistic) and by outcome domain. No meta-analysis was done, because the studies differed in agent, dose, population or model, comparator, duration and outcome definitions, and because numerical data and variance estimates were missing or unverified for several studies. Pooling would have combined outcomes that cannot be compared and overstated precision.

3. Results

3.1 Study selection

The PubMed search returned 614 records. After 30 duplicates and 490 unrelated or non-original records were removed, 94 records were screened by title and abstract, and 20 studies met the inclusion criteria and were included. Fifteen of the 20 had full texts available and were checked, and none was excluded at the full-text stage. The remaining five were assessed from abstracts or article previews. The selection process is shown in Figure 1.



Figure 1. PRISMA flow diagram of study selection.

3.2 Characteristics of the included studies

The 20 studies comprised 6 human studies, 9 animal studies and 5 biochemical, cellular or mechanistic studies (Table 1). They covered cetirizine, terfenadine, astemizole, fexofenadine, levocetirizine, ketotifen, bilastine, rupatadine, desloratadine, diphenhydramine, azelastine and cyproheptadine. Designs, doses, durations, species and outcomes varied considerably, so human findings are presented separately from animal and cell-based findings. Risk-of-bias judgements are shown separately in Table 2.

Table 1. Characteristics and main findings of the included studies

Study	Model and intervention	Sample; duration	Main glucose-related finding
Human studies			
Lal (2000)	RCT, allergic rhinitis; cetirizine, loratadine, clemastine	36; 7 days	Post-load glucose rose with cetirizine ($p<0.02$)
Chakravarti et al. (1996)	RCT, allergic rhinitis; terfenadine, astemizole	30; 7 days	Fasting glucose fell with terfenadine ($p<0.02$ on day 7)
El-Fatatry et al. (2024)	RCT, diabetic nephropathy; fexofenadine added to ramipril	61 completed; 6 months	UACR fell 16% vs rose 11% in controls ($p<0.001$)
Rizk et al. (2025)	Add-on trial, diabetic kidney disease; levocetirizine	60; 3 months	HbA1c difference between groups ($p=0.007$)
El-Haggar et al. (2015)	Add-on trial, type 2 diabetes; ketotifen with glimepiride	48; 12 weeks	Twice-daily dose improved glycaemic markers (abstract level)
Dolecek (1981)	Diabetic patients with allergy; ketotifen	30; 14 days	Some improvement in glucose tolerance; no values given
Animal studies			
Anvari et al. (2015)	High-fat diet and NOD mice; cetirizine	2 weeks; 21 weeks	Lower glucose AUC in high-fat mice; no protection in NOD mice
Shawky et al. (2014)	Fructose-fed rats; levocetirizine	8 weeks	Lower fasting glucose and HOMA-IR ($p<0.05$)
Anbar et al. (2016)	Streptozotocin rats; levocetirizine	8 weeks	Mild glycaemic improvement; mainly renal effects
Hafez et al. (2020)	Streptozotocin rats; rupatadine	4 weeks	Reduced serum glucose (qualitative)

Gasheva et al. (2019)	Rats; desloratadine	16 weeks	Higher fasting glucose than controls ($p<0.05$)
Verta et al. (2019)	Streptozotocin mice; bilastine	About 14 weeks	No supported glucose lowering
Afzal et al. (2018)	Talc-induced hyperglycaemia, rats; diphenhydramine	21 days	Reduced glucose rise
Elseweidy et al. (2022)	Diabetic hyperlipidaemic rats; azelastine	8 weeks	Reduced glucose and HbA1c
Chen et al. (2017)	High-sugar/high-fat rats; ketotifen	8 weeks	Lower fasting glucose and HOMA-IR ($p<0.05$)
Biochemical, cellular and mechanistic studies			
Almasri et al. (2019)	Enzyme assay and mouse test; fexofenadine	Not applicable	DPP-4 IC50 4.6 ± 1.0 μM; lower glucose AUC in mice
Biedrzycki et al. (2024)	BSA glycation model; 12 antihistamines	In vitro	No consistent antiglycation effect
Hawkins and Fischer (2004)	Insulinoma cells; cyproheptadine	2 hours	Reduced insulin synthesis
Chow et al. (1988)	Rats; cyproheptadine	2 days	Metabolites implicated in insulin loss
Miller et al. (1993)	Insulinoma cells; cyproheptadine	24 hours	Cellular insulin fell to about 30% of control

UACR, urinary albumin-to-creatinine ratio; NOD, non-obese diabetic; AUC, area under the curve; BSA, bovine serum albumin; RCT, randomised controlled trial.

3.3 Human studies

Short studies in allergic rhinitis gave contrasting results. Lal (2000) reported a significant rise in post-load glucose after cetirizine ($p<0.02$), with non-significant changes for loratadine and clemastine. Chakravarti et al. (1996) reported lower fasting glucose with terfenadine, with significant reductions after the first dose ($p<0.05$) and on day 7 ($p<0.02$), but not with astemizole. Both studies were small and short, and both printed glucose units as g%.

In people with diabetes, the trials mainly concerned diabetic kidney disease. El-Fatatry et al. (2024) added fexofenadine to ramipril. UACR fell by 16% in the fexofenadine group and rose by 11% in controls ($p<0.001$), and eGFR did not differ significantly between groups. Rizk et al. (2025) added levocetirizine to empagliflozin and valsartan and reported a between-group HbA1c difference ($p=0.007$), together with differences in UACR ($p=0.037$), TNF-α ($p=0.004$)

and cystatin C (p=0.034), but no significant eGFR difference. Exact HbA1c values were not available, and the add-on designs mean the effect of levocetirizine alone cannot be separated. El-Haggar et al. (2015) reported that twice-daily ketotifen added to glimepiride improved glycaemic and inflammatory parameters except adiponectin, whereas once-daily ketotifen changed only inflammatory markers. Dolecek (1981) described some improvement in glucose tolerance with ketotifen in 30 hospitalised diabetic patients, but gave no numerical values.

Overall, the human evidence is limited and mixed, and it does not show a consistent glucose-lowering effect across the class.

3.4 Animal studies

Animal findings varied by antihistamine and by model. Anvari et al. (2015) gave cetirizine (25 mg/kg/day) to male C57BL/6 mice on a high-fat diet for two weeks. Glucose-tolerance AUC was significantly lower in treated mice ($p<0.05$), but insulin sensitivity did not improve. In female non-obese diabetic (NOD) mice, cetirizine from 9 to 30 weeks of age did not prevent diabetes (12 of 20 treated mice and 10 of 20 controls developed diabetes; $p>0.05$).

Levocetirizine (0.5 mg/kg/day) was studied in two rat models. Shawky et al. (2014) used a 60% fructose diet for eight weeks and found significantly lower fasting glucose, glucose-tolerance and insulin-tolerance AUCs and HOMA-IR, and higher HOMA-β ($p<0.05$). Anbar et al. (2016) compared levocetirizine with losartan in streptozotocin-induced diabetic rats over eight weeks and described a mild glycaemic improvement, although most of the report concerned renal and vascular protection.

Other antihistamines gave mixed results. Ketotifen (0.09 mg/kg/day) lowered fasting plasma glucose, fasting insulin, HOMA-IR, lipids, IL-6 and TNF-α and raised HDL and SOD in rats on a high-sugar/high-fat diet, with larger changes at eight weeks than at four (Chen et al., 2017). Rupatadine (3 or 6 mg/kg/day for four weeks) reduced raised serum glucose and markers of nephropathy in streptozotocin-induced diabetic rats (Hafez et al., 2020). Diphenhydramine (1 mg/kg) reduced the rise in blood glucose and several biochemical markers in talc-induced hyperglycaemia in rats (Afzal et al., 2018), and azelastine (4 mg/kg/day for eight weeks) reduced glucose and HbA1c in diabetic hyperlipidaemic rats (Elseweidy et al., 2022). In contrast, rats given desloratadine (50 µg/kg/day) for 16 weeks had higher fasting glucose than controls (110.7 ± 4.7 vs 79.5 ± 2.4 mg/dL; $p<0.05$), together with greater weight gain, altered lipids and more liver fat (Gasheva et al., 2019). Bilastine (1 to 30 mg/kg/day for about 14 weeks) did not produce a statistically supported fall in glucose in diabetic mice, although some renal outcomes improved (Verta et al., 2019).

3.5 Biochemical, cellular and mechanistic studies

Almasri et al. (2019) screened a library of 1,490 drugs against human DPP-4 and identified fexofenadine as a candidate. Fexofenadine inhibited recombinant human DPP-4 with an IC50 of 4.6 ± 1.0 µM, and in mice (34 mg/kg intraperitoneally, 30 minutes before oral glucose) it lowered the glucose curve and AUC compared with vehicle ($p<0.01$). Biedrzycki et al. (2024) tested 12 H1 antihistamines in bovine serum albumin glycation models and docked them against albumin, not DPP-4. Antiglycation activity was not strong or consistent across agents,

although ketotifen reduced advanced glycation end products by 45.4% in the glucose-induced model.

Three studies examined cyproheptadine and insulin production. In RINm5F rat insulinoma cells, 10 μM cyproheptadine reduced the proportion of preproinsulin mRNA bound to endoplasmic-reticulum polysomes and altered phosphorylation of eIF2α, eIF4E and 4E-BP1, consistent with reduced translation initiation (Hawkins and Fischer, 2004). Cellular insulin fell progressively to about 30% of control levels after 24 hours, and the desmethylcyproheptadine epoxide metabolite was five times more potent than the parent compound (Miller et al., 1993). In rats, inhibitors of drug metabolism protected against cyproheptadine-induced loss of pancreatic insulin, which supports a role for metabolites (Chow et al., 1988). These are mechanistic findings and not glycaemic outcomes.

3.6 Risk of bias

Risk-of-bias judgements for each study are shown in Table 2. Eight studies were judged to be at low risk of bias, two at low or unclear risk, five with some concerns, three with some concerns or unclear risk, and two at high risk of bias. Several domains could only be judged as unclear because some records were assessed from abstracts or previews. Overall confidence in the evidence is limited: most human studies were small or short, several used add-on designs, and many animal and laboratory studies were preclinical.

Table 2. Risk-of-bias appraisal of the included studies

Study	Evidence type	Tool applied	Overall judgement
Lal (2000)	Human RCT	RoB 2	Low risk of bias
Chakravarti et al. (1996)	Human RCT	RoB 2	Low risk of bias
El-Fatatry et al. (2024)	Human RCT	RoB 2	Some concern
Rizk et al. (2025)	Human add-on trial	ROBINS-I	Some concern
El-Haggar et al. (2015)	Human add-on trial	ROBINS-I	Some concern or unclear
Dolecek (1981)	Human study	ROBINS-I	Some concern or unclear
Anvari et al. (2015)	Mouse model	SYRCLE	Low risk of bias
Shawky et al. (2014)	Rat model	SYRCLE	Low risk of bias
Anbar et al. (2016)	Rat model	SYRCLE	Some concern or unclear

Hafez et al. (2020)	Rat model	SYRCLE	Low risk of bias or unclear
Gasheva et al. (2019)	Rat model	SYRCLE	Low risk of bias
Verta et al. (2019)	Mouse model	SYRCLE	Some concern
Afzal et al. (2018)	Rat model	SYRCLE	Low risk of bias or unclear
Elseweidy et al. (2022)	Rat model	SYRCLE	Low risk of bias
Chen et al. (2017)	Rat model	SYRCLE	Some concern
Chow et al. (1988)	Rat study	SYRCLE	High risk of bias
Almasri et al. (2019)	Enzyme assay and mouse test	Structured appraisal; SYRCLE	Low risk of bias
Biedrzycki et al. (2024)	BSA glycation and docking	Structured appraisal	Low risk of bias
Hawkins and Fischer (2004)	Cell study	Structured appraisal	Some concern
Miller et al. (1993)	Cell study	Structured appraisal	High risk of bias

Low = low risk of bias; RoB 2 = Cochrane risk-of-bias tool for randomised trials; ROBINS-I = risk of bias in non-randomised studies of interventions; SYRCLE = risk-of-bias tool for animal studies.

4. Discussion

4.1 Principal findings

This review found that the reported effects of H1 antihistamines on glucose regulation are inconsistent. Some studies reported lower glucose, better glucose tolerance or less insulin resistance, others found no significant glucose-lowering effect, and some reported higher glucose. The findings depended on the individual drug, the model and the outcome measured, so the metabolic effects of one antihistamine should not be attributed to the whole class.

4.2 Human and animal evidence

The human studies were few, small and heterogeneous. Cetirizine raised post-load glucose in one short study (Lal, 2000), while terfenadine lowered fasting glucose in another (Chakravarti et al., 1996), which suggests that individual agents may act differently, although small samples and short treatment limit these conclusions. The trials in diabetes mainly concerned diabetic kidney disease. The main benefit of fexofenadine was on albuminuria (El-Fatatry et al., 2024), and the HbA1c difference reported with levocetirizine came from an add-on design (Rizk et

al., 2025), so neither result shows an independent glucose-lowering effect. Renal or inflammatory improvement is not direct evidence of glucose lowering.

Animal studies reported both favourable and unfavourable signals. Cetirizine improved glucose tolerance in high-fat-fed mice but not insulin sensitivity or diabetes outcome in NOD mice, which shows that better glucose tolerance does not always accompany better insulin sensitivity. Levocetirizine and ketotifen gave favourable results in selected rodent models, whereas desloratadine was linked to higher fasting glucose and bilastine did not clearly lower glucose. Differences in species, methods of inducing metabolic disturbance, doses and durations make comparison difficult, and animal findings cannot be assumed to apply to humans.

4.3 Possible mechanisms

Several mechanisms may be relevant. Changes in insulin sensitivity were reported for levocetirizine and cetirizine in diet-induced models, and several studies reported better inflammatory and oxidative-stress markers, but these studies do not show that reduced inflammation caused the change in glucose. The cyproheptadine studies indicate that some antihistamine-related compounds can reduce insulin synthesis under experimental conditions, and these effects should not be generalised to other H1 antihistamines or to therapeutic doses. Fexofenadine inhibited recombinant human DPP-4 in an earlier study (Almasri et al., 2019), which gives a basis for further investigation. Enzyme inhibition in vitro does not by itself show that fexofenadine is effective as a glucose-lowering treatment in people, and no included study tested this directly in humans.

4.4 Implications

The evidence is not sufficient to recommend H1 antihistamines for glucose control or to replace established diabetes treatment. Their possible role in glucose regulation should be considered investigational. Future clinical studies should assess clearly defined outcomes, including fasting glucose, HbA1c and insulin sensitivity, record adverse effects, and keep direct glycaemic outcomes separate from renal and inflammatory outcomes. Comparative studies of different antihistamines under standardised conditions would show whether effects are drug-specific, and the DPP-4 finding for fexofenadine should be tested in further enzyme and in-vivo experiments.

4.5 Strengths and limitations

A strength of this review is that it combined human, animal and mechanistic evidence and kept direct glycaemic outcomes separate from related outcomes. Several limitations should be noted. The review protocol was not registered, so the methods could not be checked against a pre-specified plan. Only the PubMed search was documented in a reproducible way, and the Google Scholar search was not recorded, so relevant studies may have been missed. Five of the 20 studies were assessed from abstracts or previews, which limited data extraction and the risk-of-bias judgements, and exact numerical values were not available for several studies. The studies differed too much in design, drug, dose, duration and outcome for a meta-analysis, and publication bias could not be assessed. Several favourable findings came from animal or

laboratory experiments, and some clinical studies tested antihistamines alongside established treatments, so the contribution of the antihistamine could not be isolated.

5. Conclusion

The current evidence does not establish H1 antihistamines as effective treatments for diabetes. Their reported effects on glucose regulation differ between drugs, models and settings, and the human evidence is small, short and mixed, with several studies measuring renal or inflammatory outcomes instead of glucose. The review is significant because it separates direct glycaemic outcomes from related outcomes and shows how easily the evidence can be overstated. Future research should include well-designed clinical trials with clearly defined glycaemic outcomes, comparisons of individual antihistamines under standard conditions, and enzyme and in-vivo tests of the DPP-4 finding for fexofenadine. Until such evidence exists, any role of these drugs in glucose regulation should be regarded as investigational.

Declarations

Ethics approval: Not applicable. This review used published literature and did not recruit participants or carry out experiments.

Funding: None.

Conflicts of interest: None declared.

Data availability: The data extracted from the included studies are in Table 1 and Table 2, and the search string is in the Appendix.

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Appendix. PubMed search string (9 October 2026, 614 records)

(

"Histamine H1 Antagonists"[Mesh]

OR "histamine H1 antagonist*"

OR "H1 antihistamine*"

OR "H1 antagonist*"

OR cetirizine OR levocetirizine OR loratadine OR desloratadine

OR fexofenadine OR bilastine OR rupatadine OR ebastine

OR mizolastine OR olopatadine OR terfenadine OR astemizole

OR ketotifen OR clemastine OR hydroxyzine OR diphenhydramine

OR chlorpheniramine OR cyproheptadine

)

AND

(

"blood glucose" OR "blood sugar" OR "fasting glucose"

OR "fasting blood glucose" OR "fasting plasma glucose"

OR HbA1c OR "glycated hemoglobin" OR "glycosylated hemoglobin"

OR "glucose tolerance" OR OGTT OR "oral glucose tolerance test"

OR "insulin resistance" OR HOMA-IR OR "insulin secretion"

OR diabetes OR diabetic OR hypoglycemic

)