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Relationship of asprosin and diabetes: a meta-analysis

Xiandong Zeng[1], Xin Sun[2], Wei He[3], Jing Xie[2] and Caihong Xin[4*]

Abstract

Background Diabetes characterized by chronic hyperglycemia, has become a serious hazard to human health in the recent decades. Previous research suggests that asprosin may contribute to the development of diabetes by regulating glucose homeostasis, appetite, insulin secretion, and insulin sensitivity. Although some studies have shown that asprosin levels are higher in patients with diabetes than in healthy individuals, the association between asprosin levels and diabetes remains controversial.

Aim This meta-analysis aimed to assess asprosin levels in patients with diabetes and in healthy individuals.

Methods We searched the following electronic databases: Web of Science, ScienceDirect, PubMed, and Willy. The title or abstract uses the following search term: "diabetes" is used in combination with the term "asprosin." The meta-analysis results are presented as standardized mean differences (SMDs) with corresponding 95% confidence intervals (CIs).

Results Fourteen articles were included in this meta-analysis. In our meta-analysis, the asprosin level in patients with diabetes was significantly higher than that in healthy controls (SMD: 0.95, 95% CI [0.66, 1.24]). Moreover, there was a significant difference in the asprosin levels between patients with diabetes without complication and those with complication (SMD: 0.81, 95% CI [0.33, 1.29]).

Conclusions This systematic review is the first to evaluate the relationship between asprosin levels and diabetes. The asprosin levels were significantly higher in patients with diabetes.

Clinical trial number Not applicable.

Keywords Asprosin, Diabetes, Meta-analysis

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Introduction

Diabetes characterized by chronic hyperglycemia, has become a serious hazard to human health in the recent decades. Hyperglycemia causes damage to the basic structure and function of the body's large vessels and micro vessels, leading to heart, brain, kidney, eyes, feet, autonomic neuropathy, and peripheral neuropathy [1]. Therefore, understanding the possible pathogenesis of diabetes and its complications, and conducting early interventions can effectively reduce the decline in quality of life, disability, and death caused by diabetes complications.

Romere et al. discovered a new type of adipose factor in a study of premature aging syndrome in newborns, which is mainly secreted by white fat; hence, it was named asprosin. Asprosin is the C-terminal cleavage product of the precursor fibrillar protein composed of 140 amino acids encoded by exons (exons 65 and 66) of FBN1(fibrillin-1). Although asprosin is mainly produced by white adipose tissue, its target organs of action are distributed throughout the body. In the brain, asprosin receptors are mainly located in the arcuate nucleus of the hypothalamus. Meanwhile, asprosin receptors are mainly located in the liver, pancreas, skeletal muscle, and myocardium [2]. Asprosin has a circadian rhythm and its secretion level is influenced by diet and exercise. It uses the olfactory receptor OLFR734 to regulate liver glucose production and maintain glucose homeostasis [3]. Previous research suggests that asprosin may contribute to the development of diabetes by regulating glucose homeostasis, appetite, insulin secretion, and insulin sensitivity. Asprosin increases blood sugar and insulin levels and is elevated in insulin-resistant humans and mice. Basic studies have revealed that asprosin can act on various organs, such as the liver, brain, skeletal muscles, and pancreas, causing elevated blood sugar, increased food intake, insulin resistance, and pancreatic islet inflammation and dysfunction [4, 5].

Several studies have compared the plasma level of asprosin in diabetes with complications and treatment. Plasma asprosin levels seem to be positively correlated with complications and inversely associated with treatment [6–8, 12–14]. However, association of blood asprosin level with diabetes is unclear. It was found either unchanged [9–11] or elevated [6–8, 12–14] when compared with individuals with diabetes. For this reason, we have undertaken this meta-analysis to confirm whether there was an association between asprosin levels with diabetes.

Methods

Search

We searched the Web of Science, ScienceDirect, PubMed, and Willy electronic databases. The title or abstract uses the following search term: "diabetes" is used in combination with the term "asprosin." We focused our search on the period 1980–2023, and only English was used. The references of the retrieved articles were checked to ensure that no additional eligible studies were included. No unpublished studies were identified to date. This systematic review and meta-analysis was registered in PROSPERO under CRD42023475616. In the supplementary data, you will find a list of all the items that should be reported for systematic reviews and meta-analyses (S1). Due to the aim and search strategy, the information of treatment and complications is limited, it was not possible to confirm or infirm some associations with the asprosin levels.

Inclusion criteria

Meta-analysis was conducted based on studies meeting the following criteria: (1) sufficient data on asprosin levels in diabetic patients and healthy individuals to performed the meta-analysis, (2) case-controlled design, and (3) language limited to English.

Data extraction and risk of bias

As part of the preliminary screening process, two reviewers (ZXD and XCH) independently used the search strategy and read the titles and abstracts to exclude studies that did not meet the inclusion criteria. To determine whether the studies met the inclusion criteria, the two reviewers methodologically reviewed the full text. If the author's information is incomplete, they can contact and crosscheck the author. If the conclusions of the two evaluators were inconsistent, the differences were resolved through discussion. If the discussion failed to resolve any differences, it was judged and arbitrated by a third researcher (SX). Non-randomised evidence was analysed using the Cochrane Collaboration's Risk Of Bias In Non-randomised Studies of Interventions (ROBINS-I) tool [15, 16], which considers seven domains of bias: two pre-intervention (confounding, selection of participants), one at intervention (classification of intervention) and four post-intervention (deviations from intended interventions, missing data, measurement of outcomes and selection of the reported result). Each domain could be categorized as low, moderate, serious, or critical risk, or no information provided.

Statistical analysis

We assessed heterogeneity among the included studies using the I^2 statistic and presented the data as standardized mean differences (SMDs) and 95% confidence intervals (CIs). Fixed-effects models were used if I^2 was $<50\%$ and heterogeneity among studies was low or moderate; otherwise, random-effects models were used if I^2 was $>50\%$. A sensitivity analysis was performed to evaluate

the stability of the results. Begg's and Egger's tests were used to detect publication bias. $P < 0.05$ was set as the significance level. Data analysis was performed using Stata version 12.0 (College Station, TX).

Results

A total of 270 studies were retrieved from the Web of Science, ScienceDirect, PubMed, and Willy electronic databases. After screening, 14 articles were selected [8–13, 17–24]. A flow diagram of the article selection process is shown in Fig. 1. Table 1 summarizes the characteristics of each study. Supplementary Table S2 shows the results of risk of bias assessment. Overall, all include studies were rated as low or moderate risk in overall methodological quality.

Results of meta-analysis

In total, 14 articles from four countries including 911 diabetes cases and 702 healthy controls during 2018 and 2023 were included. The asprosin level in patients with diabetes was significantly higher than that in healthy controls (SMD: 0.95, 95% CI [0.66, 1.24]; $I^2 = 85.2\%$). Forest plots of asprosin levels in patients with diabetes compared with those in healthy controls are presented in Fig. 2. Four studies included patients with diabetes complication, such as diabetic nephropathy, diabetic peripheral neuropathy and diabetic retinopathy. There was a significant difference in the asprosin levels between patients with diabetes without complication and those with complication (SMD: 0.81, 95% CI [0.33, 1.29]; $I^2 = 74.5\%$). Forest plots of asprosin levels are shown in Fig. 3. The asprosin levels of patients with diabetes was also significantly decreased after treatment (SMD: −0.72, 95% CI [− 1.40, − 0.05]; $I^2 = 86.9\%$).

Sensitivity analysis and publication bias

Each study was subjected to a sensitivity analysis to determine its influence. Sensitivity analysis showed no

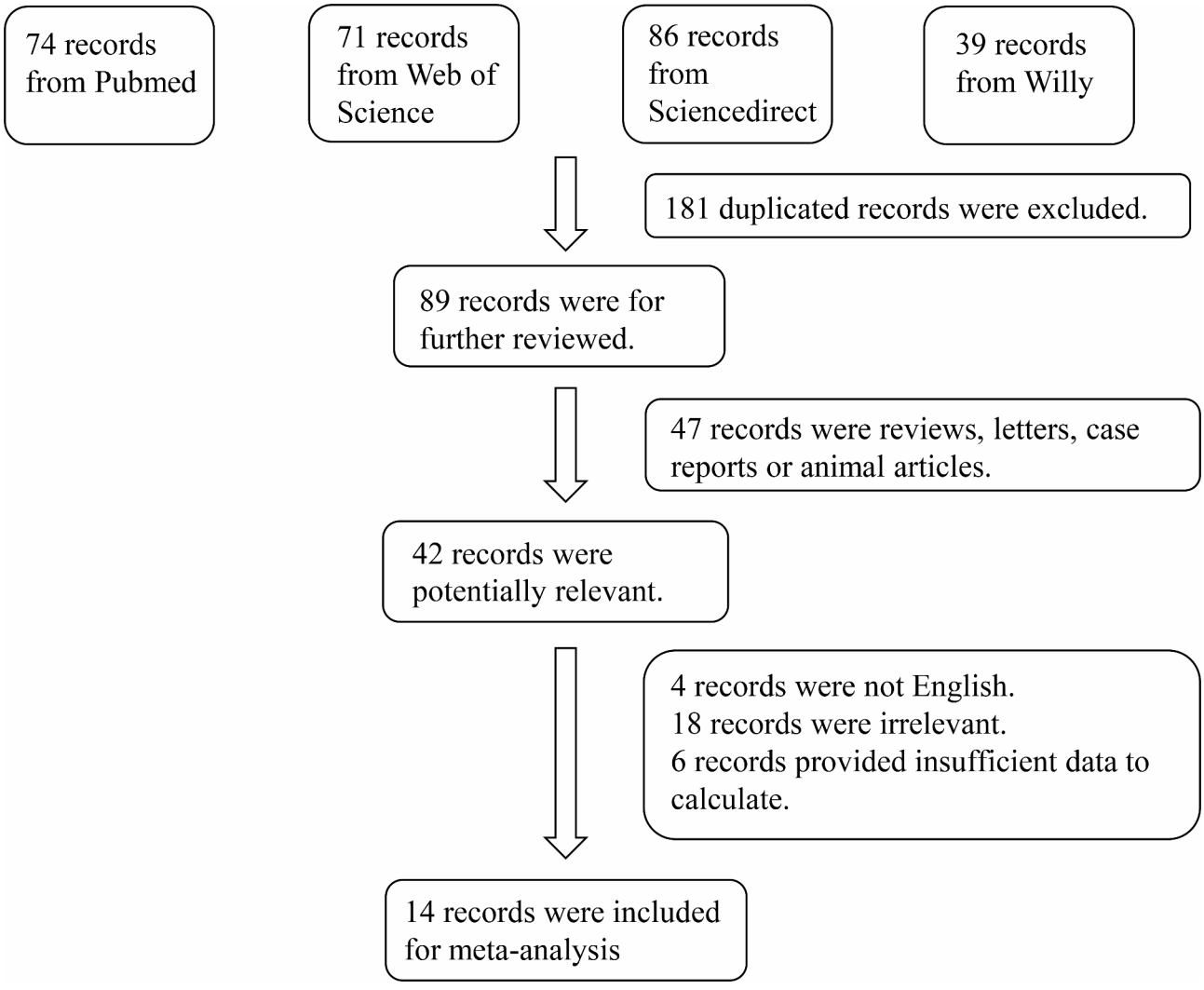

Fig. 1 Flowchart of the detailed procedure for the inclusion or exclusion of selected studies

Table 1 Study characteristics of the published studies included in the meta-analysis

Author	Publication Year	Study Period	Ethnicity	Region	Number (*n*)		Level of Asprosin		Details		Sample
					Case	Control	Case	Control	Case	Control	
Wang Y	2018	–	Asian	China	51	52	73.25 ± 91.69 ng/mL	16.22 ± 9.27 ng/mL	M 27, F 24; 53.73 ± 10.06 years; T2DM	M 17, F 35; 54.73 ± 11.90 years	Plasma
Zhang L	2019	March to June 2017	Asian	China	84	86	3.52 ± 4.2 ng/mL	1.77 ± 1.64 ng/mL	M 52, F 32; 49.93 ± 10.99 years; newly diagnosed T2DM	M 49, F 37; 47.60 ± 7.95 years	Serum
Zhang H	2020	April to December 2019	Asian	China	42	30	6.23 ± 0.87 ng/mL	5.08 ± 1.31 ng/mL	M 23, F 19; 55.98 ± 8.59 years; T2DM; duration of DM: 10.0 (6.0–13.0) years	M 14, F 16; 54.30 ± 4.92 years	Serum
Zhong L	2020	–	Asian	China	40	40	1.35 ± 0.68 ng/mL	0.5 ± 0.84 ng/mL	All females; 34.18 ± 3.24 years; GDM; gestational age: 270.45 ± 8.81 days	All females; 34.2 ± 3.31 years; gestational age: 273.63 ± 6.51 days	Plasma
Zhang X	2020	May to December 2018	Asian	China	60	60	6.2 ± 1.13 ng/mL	5.22 ± 1.26 ng/mL	M 32, F 28; 56.40 ± 7.49 years; T2DM	M 34, F 26; 54.62 ± 5.97 years	Serum
Naiemian S	2020	August to December 2018	Asian	Iran	97	97	4.18 ± 3.26 ng/mL	3.5 ± 1.37 ng/mL	M 50, F 47; 54 ± 7 years; newly diagnosed T2DM	M 50, F 47; 52 ± 10 years	Serum
Goodarzi G	2021	January 2019 to Jan 2020	Asian	Iran	54	55	6.7 ± 1.35 nmol/L	4.81 ± 1.09 nmol/L	M 32, F 22; 61.83 ± 7.78 years; T2DM	M 36, F 19; 58.71 ± 7.93 years	Serum
Gozel N	2021	–	Caucasian	Turkey	30	30	49.52 ± 41.98 ng/mL	25.16 ± 14.49 ng/mL	M 17, F 13; 54.63 ± 10.8 years; newly diagnosed T2DM; treated with metformin	M 16, F 14; 30.10 ± 8.8 years	Plasma
You M	2022	June to October 2021	Asian	China	33	30	173 ± 40.7 ng/mL	158.5 ± 10.25 ng/mL	M 18, F 15; 60.85 ± 13.91 years; T2DM; duration of DM: 8 (3–14) years	M 16, F 14; 60.63 ± 8.08 years	Serum
Dai C	2022	February 2021 to January 2022	Asian	China	90	66	7.38 ± 1.08 ng/mL	5.87 ± 0.71 ng/mL	M 48, F 42; 54.91 ± 8.24 years; T2DM, treated with liraglutide	M 35, F 31; 52.15 ± 7.97 years	Serum
Atli H	2022	May to August 2021	Caucasian	Turkey	21	21	28.22 ± 15.41 ng/mL	25.49 ± 12.37 ng/mL	M 9, F 12; 56.05 ± 10.54 years; T2DM	M 15, F 6; 42.70 ± 7.55 years	Serum
Timurkaan M	2022	–	Caucasian	Turkey	60	60	47.7 ± 26.5 ng/mL	35.7 ± 12.7 ng/mL	M 30, F 30; 50.3 ± 5.76 years; newly diagnosed T2DM	M 29, F 31; 47.6 ± 7.64 years	Plasma
Boz IB	2023	October 2019 to November 2020	Caucasian	Turkey	30	33	42.8 ± 7.63 ng/mL	20.5 ± 8.0 ng/mL	All females; 33.6 ± 5.6 years; GDM; gestational age: 27.1 ± 3.1 weeks	All females; 27.6 ± 4.6 years; gestational age: 25.6 ± 6.2 weeks	Serum
Nedeva IS	2023	February 2020 to August 2021	Caucasian	Bulgaria	38	42	1.09 ± 0.51 ng/mL	0.71 ± 0.28 ng/mL	54.32 ± 10.97 years; newly diagnosed DM	47.71 ± 12.91 years	Serum

T2DM: Type 2 diabetes mellitus; DM: diabetes mellitus; GDM: gestational diabetes mellitus

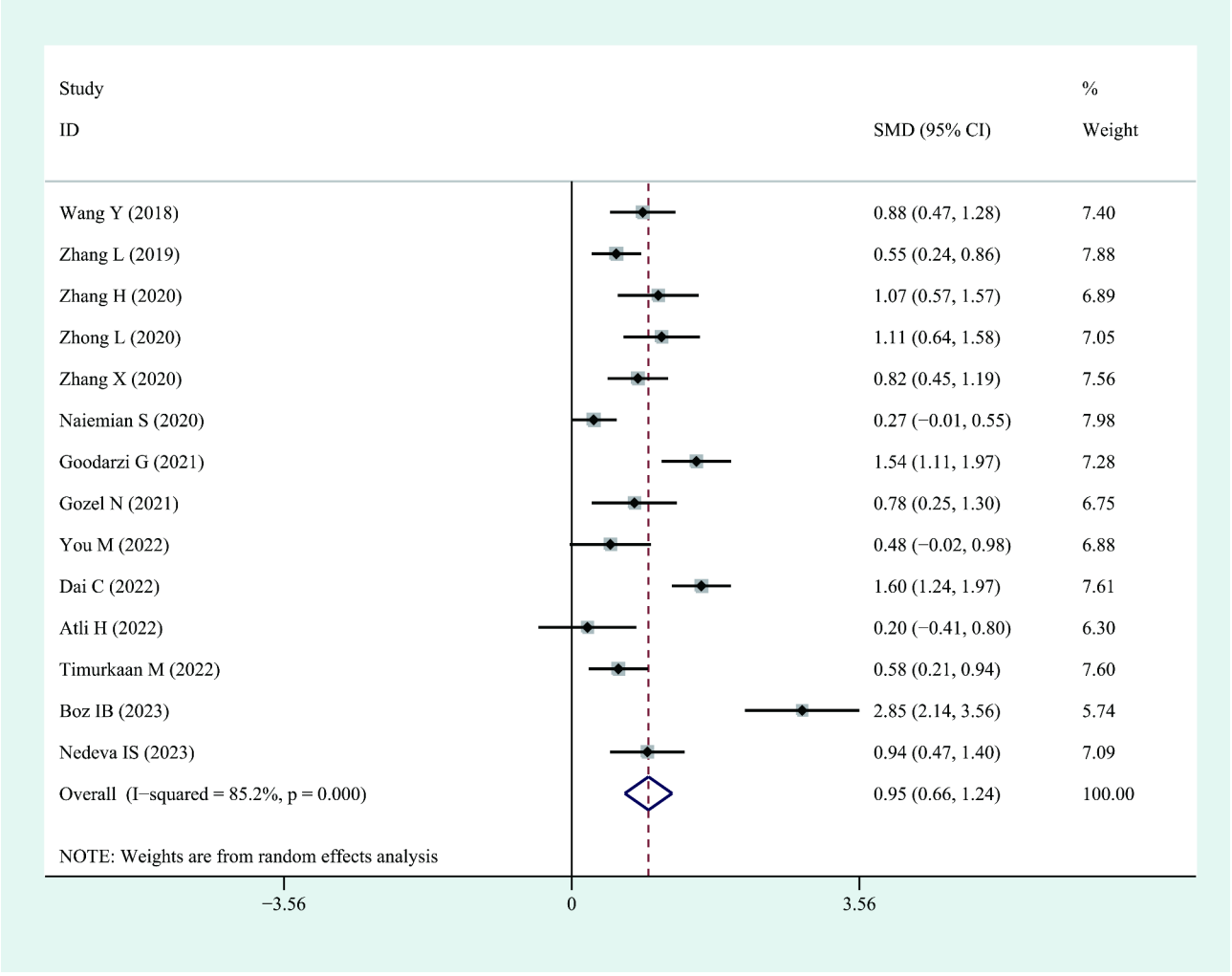

Fig. 2 Forest plots and funnel plots of asprosin level in patients with diabetes compared to healthy individuals. Diamond represents the pooled SMDs at 95% CI. SMD, standardized mean difference; CI, confidence interval

significant differences from our previous estimates, indicating that a single study had a marginal impact on the overall estimate (Figs. 4 and 5). Accordingly, the meta-analysis yields stable results. A thorough and comprehensive search of the databases was conducted. Begg's and Egger's tests were conducted to identify whether publication bias was present in the reviewed studies. The results ($P > 0.05$) indicated that there was no publication bias.

Discussion

This systematic review is the first to evaluate levels of asprosin in patients with diabetes and healthy controls. Although some studies have shown higher levels of asprosin in patients with diabetes than in healthy individuals, the relationship between asprosin levels and diabetes remains controversial. Fourteen independent studies were included in this meta-analysis. We conclude that levels of asprosin are significantly higher in patients with diabetes than in healthy controls.

In 2016, Romere et al. found that overexpression of FBN1 in the mouse liver or direct subcutaneous injection of asprosin can lead to elevated blood sugar levels [2]. Asprosin may form a classic negative feedback mechanism based on blood sugar levels. During fasting, the body mainly relies on the release of liver glycogen to maintain blood sugar stability. It has been reported that asprosin is primarily transported to the liver. After hatching mouse primary liver cells with recombinant asprosin, the PKA activity of the liver cells increased. The effects of asprosin on liver glucose production and PKA activity can be blocked by the G protein competitive antagonists sulamin and cAMP competitive antagonists, indicating that asprosin may increase liver glucose release through the G protein cAMP-PKA pathway. In 2017, Duerrschmid constructed an FBN1 gene mutant mouse model using CRISPR/Cas9. Compared to wild-type mice, mutant mice showed a decrease in asprosin levels, food

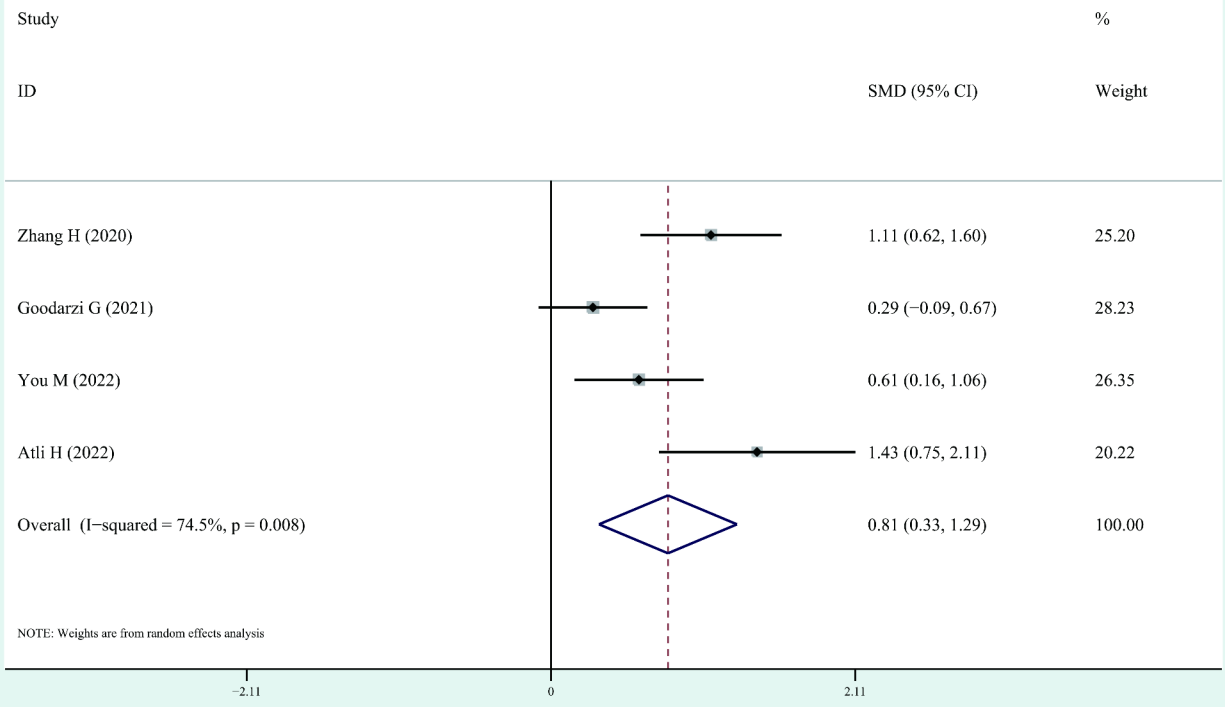

Fig. 3 Forest plots and funnel plots of asprosin level in patients with diabetes without complication and those with complication. Diamond represents the pooled SMDs at 95% CI. SMD, standardized mean difference; CI, confidence interval

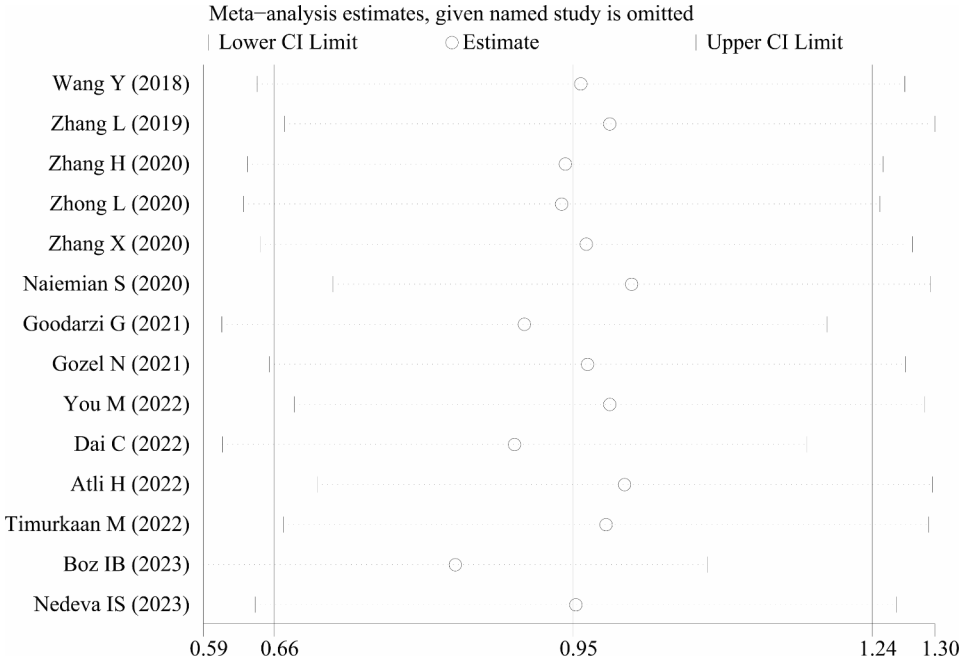

Fig. 4 The sensitivity analysis results of asprosin level in patients with diabetes compared to healthy individuals

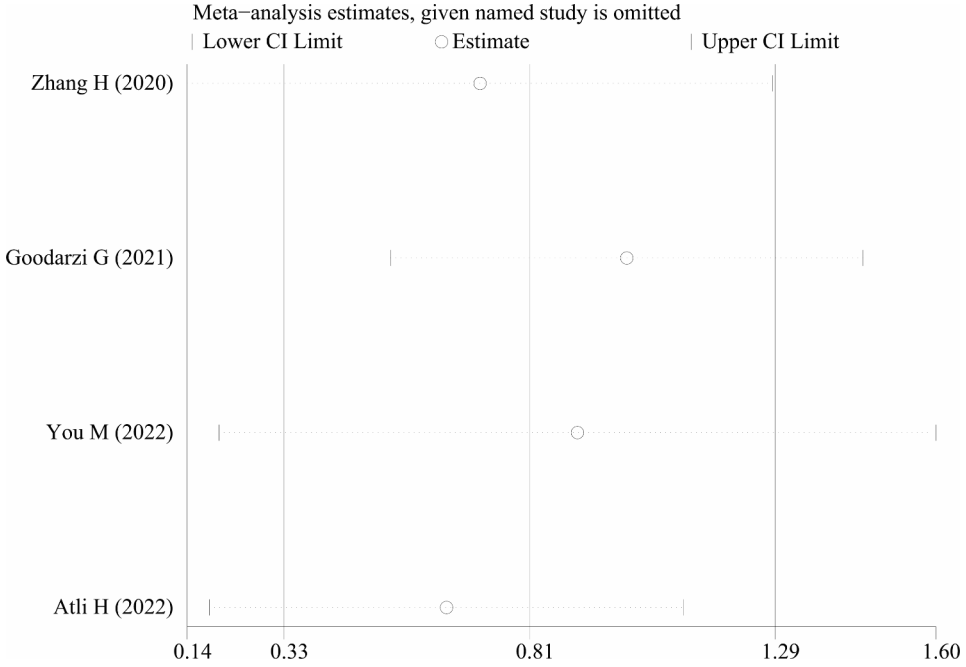

Fig. 5 The sensitivity analysis results of asprosin level in patients with diabetes without complication and those with complication

intake, and weight loss, suggesting that asprosin may have a regulatory effect on food intake [25].

In 2019, Lee et al. found asprosin promoted the release of tumor necrosis factor and human monocyte chemotactic protein-1, thereby weakening glucose-stimulated insulin secretion and islet cell activity [26]. These effects were reversed by the application of siRNAs to inhibit asprosin expression. In addition, the treatment of mouse insulinoma cells and human primary pancreatic islet cells with recombinant asprosin exacerbated inflammation, cellular dysfunction, and apoptosis. Asprosin induces the phosphorylation of toll-like receptor 4 (TLR4) and c-Jun amino-terminal kinase (JNK). When the expression of TLR4 and JNK was inhibited, the effect of asprosin on inflammation and cellular functions was weakened. Li et al. found that the OLFR734 receptor had the highest correlation with gluconeogenesis [27]. Although it belongs to the olfactory receptor family, it is also highly expressed in liver tissue. Li used the CRISPR-Cas9 technology to construct OLFR734 gene knockout mice. Compared with wild-type mice, the levels of gluconeogenesis-related genes and gluconeogenesis in liver cells were significantly reduced, indicating that OLFR734 promotes gluconeogenesis. In OLFR734 gene knockout mice, insulin levels decreased, liver fat accumulation decreased, and insulin sensitivity increased. The authors further demonstrated that asprosin activates OLFR734 coupled with cAMP signaling to promote liver glucose production and that the lack of OLFR734 receptors significantly reduces glucose production in fasting and high-fat diets.

Skeletal muscle is the main organ for glucose uptake; therefore, insulin resistance in the skeletal muscle is an important mechanism of diabetes. In 2019, Jung et al. studied the effects of asprosin on insulin resistance in skeletal muscles [28]. It was found that asprosin promoted insulin resistance in human C1C12 myocardial cells and mouse skeletal muscle cells, and the expression of ER stress and inflammatory markers increased, indicating that asprosin may promote a decrease in insulin sensitivity in skeletal muscle through ER stress and inflammation. The researcher further discovered treatment of asprosin augmented protein kinase C-δ (PKCδ) phosphorylation and nuclear translocation, and the mRNA expression level of sarcoplasmic reticulum Ca^{2+}ATPase 2b is inhibited in human C1C12 myocardial cells and mouse skeletal muscle cells. Application of siRNA knockdown to reduce PKC-δ in C1C12 myocardial cells. Subsequently, the effects of asprosin were significantly weakened. In summary, asprosin may induces ER stress and inflammation, leading to a decrease in insulin sensitivity in skeletal muscles through PKC-δ pathway.

In the present study, we evaluated asprosin levels in patients with diabetes and healthy individuals, while this association is likely confounded. Firstly, we included different types of diabetes, and duration of diabetes varied among the studies. Secondly, different samples of patients were used in these studies. Thirdly, most of the included studies were from China and Turkey, resulting in a lack of data from other populations. Additionally, we found that the concentration of asprosin in the blood of patients with diabetes decreased significantly after treatment,

suggesting that asprosin may become a therapeutic target for diabetes in the future. However, due to limited inclusion in the study, a strong conclusion could not be drawn. It is important to interpret the results of this meta-analysis cautiously because all these factors could have affected them; therefore further research is necessary.

Conclusion

This systematic review indicates that the asprosin levels are significantly higher in patients with diabetes. However, further investigations are required for confirming this elevation in large cohort studies and therefore, for highlighting asprosin as therapeutic target.

Abbreviations

SMD	Standardized mean difference
CI	Confidence intervals
TLR4	Toll-like receptor 4
JNK	C-Jun amino-terminal kinase
PKCδ	Protein kinase C-δ

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12902-025-01843-1.

Supplementary Material 1: **Table S1**: Preferred reporting items for systematic review and meta-analyses (PRISMA) checklist

Supplementary Material 2: **Table S2**: Risk of bias assessment by Risk Of Bias In Non-randomised Studies of Interventions (ROBINS-I) tool.

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Author contributions

XCH designed the study. SX searched databases and collected the data. XJ and ZXD assessed the quality of the study. HW performed the analysis. SX wrote the manuscript. ZXD revised the manuscript. All authors contributed to this systematic review and meta-analysis.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not Applicable.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Disclosure statement

The authors have nothing to disclose.

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