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Review

Association of risk factors with type 2 diabetes: A systematic review

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ABSTRACT

Diabetes is the leading cause of severe health complications and one of the top 10 causes of death worldwide. To date, diabetes has no cure, and therefore, it is necessary to take precautionary measures to avoid its occurrence. The main aim of this systematic review is to identify the majority of the risk factors for the incidence/prevalence of type 2 diabetes mellitus on one hand, and to give a critical analysis of the cohort/cross-sectional studies which examine the impact of the association of risk factors on diabetes. Consequently, we provide insights on risk factors whose interactions are major players in developing diabetes. We conclude with recommendations to allied health professionals, individuals and government institutions to support better diagnosis and prognosis of the disease.

Contents

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1. Introduction

Diabetes Mellitus (DM) commonly referred to as diabetes, is a chronic disease that affects how the body turns food into energy [1]. It is one of the top 10 causes of death worldwide causing 4 million deaths in 2017 [2,3]. According to a report by the International Diabetes Federation (IDF) [3], the total number of adults (20–79 years) with diabetes in 2045 will be 629 million from 425 million in 2017 (48% increase). In 2017, diabetes caused at least 727 billion USD in health expenditure, which is 12% of the total spending on adults [3]. According to the National Diabetes Statistics Report [4], 30.3 million (9.4% of the US population) people have diabetes, and 84.1 million (29.06% of the population) have prediabetes. 1 in 2 people (212 million) with diabetes was undiagnosed in 2017 according to IDF [5]. Diabetes if left untreated can cause serious medical issues, such as cardiovascular disease, stroke, chronic kidney disease, foot ulcers, damage to the eyes, and prolonged kidney ailment. To date, there is no permanent cure for diabetes and the patients have to rely on healthy lifestyle and timely medication [6].

There are three main types of diabetes: type 1, type 2, and gestational diabetes (diabetes while pregnant) [1]. Type 1 diabetes mostly occurs in children and adolescents. 1,106,500 children were suffering from type 1 diabetes in 2017 [3]. The symptoms of type 1 diabetes include abnormal thirst and dry mouth, frequent urination, fatigue, constant hunger, sudden weight loss, bed-wetting, and blurred vision. Type 2 diabetes is mostly seen in adults, but it is increasing in children and adolescents due to the rising level of obesity, physical inactivity and unhealthy diet [5]. 372 million adults were at the risk of developing type 2 diabetes in 2019 [3]. In 2017, more than 21 million live births were affected by diabetes during pregnancy [3]. In this paper, we focus on type 2 diabetes due to the alarming numbers.

Type 2 Diabetes is thought to prevail in an individual from an interaction between several lifestyle, medical condition, hereditary, psychosocial and demographic risk factors such as high-level serum uric acid, sleep quality/quantity, smoking, depression, cardiovascular disease, dyslipidemia, hypertension, aging, ethnicity, family history of diabetes, physical inactivity, and obesity [6]. In this paper, we present a systematic review of the literature on the association of these risk factors with the incidence/prevalence of type 2 diabetes. We give insights on the contribution of independent risk factors in the development of type 2 diabetes along with possible solutions towards a preventive approach.

2. Methods

We conduct a systematic literature search using CINAHL, IEEE Xplore, Embase, MEDLINE, PubMed Central, ScienceDirect, Scopus, Springer, and Web of Science databases. Our search criteria does not include a time bound. Its main objective is to retrieve all the studies which examine the association between individual risk factors and the incidence/prevalence of type 2 diabetes. Table A1 shows the search string used for each risk factor. The relevant studies have to meet the following inclusion criteria: 1) published in the English language, 2) prospective cohort or cross-sectional study, 3) type 2 diabetes as a specified risk, 4) one of its risk factors, 5) findings in terms of Odds Ratio (OR), Risk Ratio/Relative Risk (RR), or Hazard Ratio (HR), and the corresponding 95% Confidence Intervals (CIs) for the association between the risk factor and type 2 diabetes. To assess the quality of the studies, we use the National Institutes of Health (NIH) quality assessment tool [7]. The tool consists of 14 questions to evaluate the validity and bias risk of a study. We answered each question by either yes, no, cannot be determined, not applicable, or not reported. The tool then classifies each study as high quality (Good), moderate quality (Fair) and low quality (Poor).

3. Results

Fig. 1 shows the result of our systematic approach that is used to screen the relevant studies. Irrelevant studies that do not meet the inclusion criteria mentioned in the previous section were excluded after screening titles, abstracts and full texts. At last, 106 papers are considered for this review. These papers are divided into ten categories based on the risk factor under study (Fig. 1). Our review reveals that there is no study that examines the association of age or physical inactivity as an independent risk factor with type 2 diabetes. Table A2 shows the quality assessment results for the studies included in this paper. For smoking, cardiovascular disease and hypertension risk factors, the majority of the studies are of high quality. For serum uric acid, sleep quantity/quality, depression, dyslipidemia, ethnicity, family history of diabetes and obesity, the majority of the studies are of moderate quality.

3.1. Serum uric acid

Serum uric acid, a common component of urine generated by the metabolic breakdown of purines, have been associated with insulin resistance and type 2 diabetes [8]. High serum uric acid level in an individual leads to: 1) nitric-oxide mediated vasoconstriction (contraction of blood vessels) leading to impaired glucose uptake in the muscles [9], 2) increase in oxidative stress [10] and 3) increase in inflammation leading to a decrease in adiponectin [11,12]. Consequently, the blood glucose level increases leading to dysfunctional and eventually dead beta-cells [13]. As a result, the individual develops type 2 diabetes. Table 1 shows the characteristics and findings of the work in the literature studying the association between high serum uric acid level and type 2 diabetes.

Perry et al. [14] found that an individual having a uric acid level of more than 411 μmol/l is at 1.5 times more risk of developing type 2 diabetes compared to an individual having uric acid level less than 302 μmol/l. Niskanen et al. [15] also confirmed that change in uric acid levels is associated with a 2 times increase in the risk of incidence type 2 diabetes. Dehghan et al. [16] in their study showed that individuals having uric acid level >370 μmol/l are at high risk of incidence type 2 diabetes (HR 1.68, 95% CI 1.22–2.30) compared to those having uric acid level $\leqslant 267$ µmol/

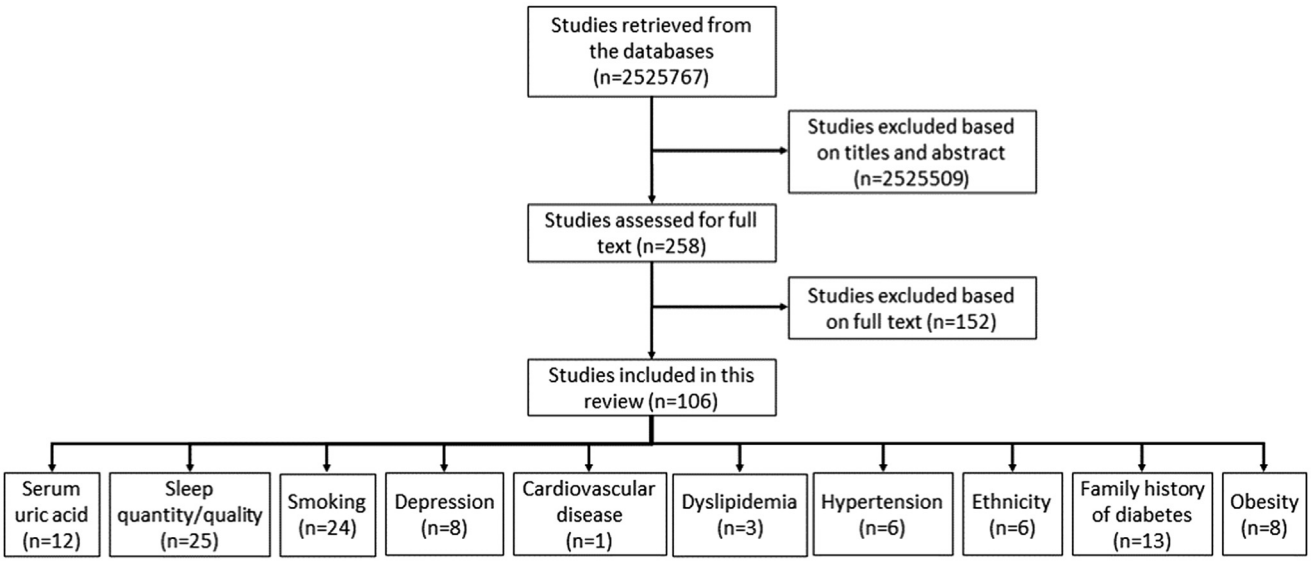

Fig. 1. Flowchart of the selection of relevant studies.

l. The authors concluded that lowering uric acid level can be a novel approach for diabetes prevention. Xu et al. [17] found that the association between high serum uric acid level and diabetes is the same in both men and women (RR 1.131, 95% CI 1.084–1.179). The association (RR 1.17, 95% CI 1.09–1.25) is also examined by Kodama et al. [18]. Nakagawa et al. [19] showed that uric acid is a significant and independent risk factor in predicting hyperinsulinemia. The authors observed that serum uric acid level $\geqslant$5.5 mg/dl is associated with the development of hyperinsulinemia after 6 months (OR 5.47, 90% CI 1.6–1.77) and 12 months (OR 3.4, 90% CI 1.1–10.4). However, the cohort was controlled for gender and age (>60 years). Consequently, it can not be concluded whether uric acid is an independent risk factor or there is an integrated effect of uric acid, gender and age.

Several studies argue that high-level uric acid is not an independent risk factor and it only emphasizes the association between independent risk factors such as age, obesity, hypertension, gender, and dyslipidemia, and type 2 diabetes [20]. Chou et al. show that uric acid has a significant association with type 2 diabetes in old and obese individuals [21]. Another study by Meisinger et al. [22] shows that high-level uric acid is associated with incidence of type 2 diabetes in women only with HR 2.5 per 1 mmol/L increase. Carnethon et al. [23] found that the risk of incidence type 2 diabetes increases (OR 1.3, (1.2–1.4)) with every 1.4 mg/dl increase in uric acid level. However, this is in combination with an increase in waist/hip ratio, smoking and obesity. Chien et al. [24] stated that individuals with a uric acid level of 0.486 mmol/L and having metabolic syndrome have a 3.3 times more risk of incidence type 2 diabetes compared to those with a uric acid level of 0.211 mmol/L and not having metabolic syndrome. Nan et al. [25] examined the impact of ethnicity and gender on the association between uric acid and incidence of type 2 diabetes. The authors found that the high serum uric acid is an independent risk factor for type 2 diabetes in Mauritian Indian men compared to Creole men, and there is a no-to-weak association in women of both ethnicity. Similarly, Choi et al. [26] studied the association between uric acid and type 2 diabetes in men having cardiovascular risk profile. The authors concluded that men with cardiovascular profile having high uric acid level are twice likely to develop type 2 diabetes. The authors also stated that this association between uric acid and diabetes is independent of other risk factors such as obesity, age, family history of diabetes, hypertension, and metabolic syndrome. Kramer et al. [27] analyzed the impact of age and impaired fasting glucose (IFG) on the association and found that high uric acid level can independently predict incidence of type 2 diabetes (OR 1.65, 95% CI 1.25–2.18) in older adults having IFG. Lv et al. [28] found that high serum uric acid level is associated to type 2 diabetes in middle-aged or older people (RR 1.56, 95% CI 1.39–1-76).

In summary, the association between high-level serum uric acid remains obscure. It is debatable whether serum uric acid is an independent risk factor for type 2 diabetes or it only emphasizes the association between other independent risk factors and type 2 diabetes. Some studies reported a positive association between high serum uric acid level and incidence of type 2 diabetes [14–16,19,24], whereas others [25,29] reported no association. On the contrary, some studies reported an inverse association between uric acid and diabetes [30–32]. Furthermore, some studies argue that there is a reverse association, i.e., diabetes leads to high uric acid levels [33,34].

3.2. Sleep quantity/quality

The quality and quantity of sleep are affected by several cultural, social, behavioral, psychological, and environmental factors. The working professionals often experience fatigue, tiredness and daytime napping due to irregular working hours and shifts. Evidence shows that the current average sleep of an individual, i.e., 6.8 h/night, is 1.5 h less than that a century ago [45]. The cause of sleep loss is multi-factorial. For instance 45% of adults report that they sleep fewer hours to get more work done, 43% reported that they watch television or use the Internet, and 22% reported to be suffering from insomnia. The unusual, disturbed and reduced sleep is associated with glucose intolerance [46].

An individual suffering from sleep disorder, known as obstructive sleep apnea (OSA), experiences: 1) deficiency in the amount of oxygen reaching the tissues by total/partial collapse of upper airways while sleeping (hypoxia) and 2) inflammation. Frequent Hypoxia triggers an increase in sympathetic activity [47]. Increased sympathetic activity and inflammation lead to insulin resistance condition [48,49] and eventually to type 2 diabetes. Table 2 shows the characteristics and findings of the work in the literature studying the association between sleep quantity/quality and type 2 diabetes.

Table 1
Characteristics and findings of the studies examining the association between high level serum uric acid and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[14]	1995	RS	PCS	7577 (2.56%)	100/0	40–59	Britain	12.8	Age, BMI, history of heart disease, physical activity, alcohol intake, smoking status, high blood pressure, HDL cholesterol, and heart rate	Uric acid (μ mol/l)) <302 ⩾412	OR 1.0 1.5 (0.9–2.5)
[21]	1998	RS	PCS	481 (17.6%)	53.02/ 46.98	⩾30	Chinese	3	Age, sex, BMI, WHR, history of hypertension, HDL cholesterol, fasting insulin, and triglycerides	Uric acid (μ mol/l) <420 >420	OR 1.0 2.581 (1.083–6.149)
[22]	2002	MONICA [35]	PCS	6166 (3.45%)	49.5/ 50.5	35–74	Germany	Mean 7.6	Age and BMI	Uric acid (μ mol/l)) increase by 1000	OR 2.05 (1.49–1.29)
[23]	2003	ARIC [36]	PCS	8574 (9.90%)	42.6/ 57.4	45–65	Blacks and Whites (USA)	11	Age, sex, education, baseline insulin concentration, BMI and blood pressure	Uric acid (μ mol/l)) increase by 123.76	OR 1.3 (1.2–1.4)
[19]	2005	RS	PCS	60	75/25	39–80	USA	1	Age, sex, BMI, baseline insulin concentration, and glomerular filtration rate	Uric acid (μ mol/l)) ⩽486 >486 (6 months) >486 (12 months)	OR 1.0 5.47 (1.6–17.7) 3.4 (1.1–10.4)
[15]	2006	FDPS [37]	–	475 (21.68%)	33.68/66.32	40–65	Finland	3.2	Age, sex, and baseline fasting	Uric acid (μ mol/l)) 99–310 311–380 381–622	OR 1.0 1.40 (0.82–2.39) 1.82 (1.07–3.10)
[24]	2008	CSCCS [38]	PCS	2960 (20.37%)	51.7/48.3	35–97	Chinese	Median 9	Age, sex, BMI, alcohol intake, exercise, marital status, educational level, occupation and family history of diabetes	Uric acid (μ mol/l)) 220 280 320 380 460	OR 1.0 1.11 (0.82–1.49) 1.29 (0.96–11.73) 1.40 (1.04–1.90) 1.63 (1.20–2.23)
[16]	2008	Rotterdam [39]	PCS	4536 (10.18%)	NA	⩾55	Netherlands	10.1	Age, sex, BMI, waist circumference, systolic and diastolic blood pressure, and HDL cholesterol	Uric acid (μ mol/l)) ⩽267 260–310 311–370 >370	HR 1.0 1.08 (0.78–1.49) 1.12 (0.81–1.53) 1.68 (1.22–2.30)
[25]	2008	RS	PCS	4259 (16.81%)	45.6/ 54.4	25–74	Indians and Creoles	5	Ethnicity, serum creatinine, alcohol consumption, family history of diabetes and fasting serum insulin	Uric acid (μ mol/l)) Men 363 367 Women 273 287	HR 1.0 1.19 (1.07–1.34) 1.0 1.05 (0.95–1.16)
[26]	2008	MRFIT [40–42]	PCS	11351 (10.70%)	100/0	35–57	Blacks and Whites (USA)	6	Smoking status, BMI, hypertension, physical activity, alcohol consumption, total energy intake, cereal fibre, intake of polyunsaturated, mono saturated and saturated fat, coffee intake, high fasting blood glucose, and low HDL cholesterol	Uric acid (μ mol/l) <333 ⩾464	RR 1.0 1.88 (1.52–2.32)
[27]	2009	RS	PCS	556 (9.89%)	41/ 59	Mean 63.3 ± 8.6	Brazil	13	Age, sex, BMI, diuretic use, and glomerular filtration rate	Uric acid (μ mol/l)) increase by 88.4	OR 1.65 (1.25–2.18)
[30]	2011	NHANES III [43,44]	CSS	14144	47.5/ 52.5	43–51	USA	–	Age, sex, race, educational level, smoking, alcohol consumption, BMI, hypertension, and serum total cholesterol	Uric acid (μ mol/l)) <380 380–460 460–548 >548	OR 1.0 0.54 (0.36–0.80) 0.40 (0.29–0.56) 0.48 (0.35–0.66)

RS-Random Sample, MONICA-Multinational MONItoring of trends and determinants in CArdiovascular disease, ARIC-Atherosclerosis Risk in Communities, FDPS-Finnish Diabetes Prevention Study, CSCCS-Chin Shan Community Cardiovascular study, MRFIT-Multiple Risk Factor Intervention Trial, NHANES-National Health and Nutrition Examination Survey, QFS-Quebec Family Study, M-Men, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

Table 2
Characteristics and findings of the studies examining the association between sleep quantity/quality and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[79]	2003	NHS [80]	PCS	70026 (2.81%)	0/100	40–65	United States	10	Working hours, hypercholesterolemia, hypertension, smoking, snoring, exercise, alcohol, depression, postmenopausal hormone use, BMI, and family history of diabetes	Sleep (Hours)	OR
										⩽5	1.18 (0.96–1.44)
										6	1.10 (0.97–1.25)
										7	1.02 (0.91–1.16)
										8	1.0
										>9	1.29 (1.05–1.59)
[56]	2004	RS [81]	PCS	2265 (1.67%)	100/0	–	Japanese	8	Age, education, occupation, shift work, BMI, leisure time, physical activity, smoking, alcohol consumption and family history of diabetes	Sleep	HR
										DIS (low frequency)	1.0
										DIS (high frequency)	2.98 (1.36–6.53)
										DMS (low frequency)	1.0
										DMS (high frequency)	2.23 (1.08–4.61)
[57]	2004	MPP [82]	PCS	6599 (4.3%)	100/0	Mean 42.6	Swedish and Caucasians	15.2	Age, lifestyle, family history of diabetes, social class, physical activity, BMI, smoking, and alcohol intake	DIS	OR
										No	1.0
										Yes	1.52 (1.05–2.20)
[59]	2005	MONICA [35]	PCS	8269 (2.27%)	50.1/49.9	25–75	Germany	7.5	Age,educational level, parental history of diabetes, smoking, alcohol consumption, hypertension, physical activity, history of angina pectoris, BMI, and dyslipidemia	DIS	OR
										No	1.0
										Yes (M)	1.10 (0.59–2.03)
										Yes (W)	1.42 (0.81–2.50)
										DMS	1.0
											1.60 (1.05–2.45)
										No	1.98 (1.20–3.29)
										Yes (M)	
										No (W)	
[83]	2005	SHHS [84]	CSS	1486	48.6/ 51.4	53–93	United States	–	Age, sex, ethnicity, waist girth, and apnea-hypopnea index	Sleep (Hours)	OR
										⩽5	2.51 (1.57–4.02)
										6	1.66 (1.15–2.39)
										7–8	1.0
										⩾9	1.88 (1.21–2.91)
[52]	2005	RS	PCS	1170 (7.52%)	47/53	45–65	Swedish	12	Age, marital status, living conditions, hypertension, obesity, smoking, alcohol use, snoring and depression	Sleep (Hours)	RR
										7–8	1.0
										⩽5 (M)	2.8 (1.1–7.3)
										⩽5 (W)	1.8 (0.5–6.8)
										⩾9 (W)	2.9 (0.6–15.0)
[50]	2005	RS	PCS	1462 (8.62%)	0/100	38–60	Swedish	32	Age, subscapular skin-fold thickness, serum lipid values, blood pressure, resting heart rate, physical activity, education and socio-economic status	No association between sleep duration and diabetes.	
[85]	2006	MMAS [86]	PCS	1139 (7.90%)	100/0	40–70	Blacks and Whites (USA)	17	Age, hypertension, smoking, self rated health status, waist circumference, education, testosterone, and cortisol	Sleep (Hours)	RR
										⩽5	1.71 (0.81–3.59)
										6	1.95 (1.06–3.58)
										7	1.0
										8	1.40 (0.78–2.54)
										>8	3.03 (1.44–6.37)
[87]	2007	NHANES I [88]	PCS	8992 (4.78%)	37.5/ 62.5	32–86	Whites and Non-whites (USA)	10	Physical activity, depression, alcohol consumption, ethnicity, education, marital status, age, obesity and hypertension	Sleep (Hours)	OR
										⩽5	1.47 (1.03–2.09)
										6	1.08 (0.80–1.47)
										7	1.0
										8	1.09 (0.83–1.43)
										⩾9	1.52 (1.06–2.17)

(*continued on next page*)

Table 2 (*continued*)

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[89]	2007	QFS [90]	CSS	740	43.65/ 56.35	21–64	Europid race	12	Age, marital status, employment status, educational level, annual income, physical activity, alcohol intake, coffee intake, hypertension, heart disease and waist circumference	Sleep (Hours) 5–6 7–8 9–10	OR 2.09 (1.34–2.98) 1.0 1.58 (1.13–2.31)
[58]	2007	HIPOP-OHP [91]	PCS	6509 (3.53%)	78.4/21.6	32–86	Japanese	4.2	Age, sex, BMI, history of smoking, history of hypertension, history of high cholesterol, history of diabetes and physical activity	DIS No Low frequency High frequency	HR 1.0 1.42 (1.05–1.91) 1.61 (1.00–2.58)
[92]	2008	FIN-D2D [93]	CSS	2770	48.2/51.8	45–74	Finland	1	Age, BMI, medication for sleep, antidepressants, smoking, sleep apnea probability, and physical activity	Subjects with ⩽6 and ⩾8 hours of sleep are more likely rightarrow have type 2 diabetes.	
[94]	2009	QFS [90]	PCS	274	42.7/ 57.3	21–64	Europid race	6	Age, smoking habits, employment status, annual household income, shift working history, resting metabolic rate, coffee intake, waist circumference and physical activity	Sleep (Hours) ⩽6 7–8 ⩾9	RR 2.42 (1.49–3.33) 1.0 2.31 (1.41–3.15)
[55]	2009	IRAS [95]	–	900 (16.22%)	43.3/56.7	40–69	Non-Hispanic Whites, Hispanics, and African-Americans	5	Age, sex, glucose tolerance, hypertension, family history of diabetes, smoking, educational level, BMI, insulin sensitivity, and acute insulin response	Sleep (Hours) 8 NHW/Hispanics ⩽7 ⩾9 African-American ⩽7 ⩾9	OR 1.0 2.36 (1.11–5.99) 2.15 (0.50–9.30) 0.63 (0.14–2.90) 0.39 (0.02–7.19)
[96]	2009	RS	CSS	1741	42.6/ 57.4	⩾20	Pennsylvania	-	Age, race, sex, BMI, smoking, alcohol consumption, depression and sleep disordered breathing	Sleep (Hours) ⩽5 5–6 ⩾6	OR 2.95 (1.2–7.0) 2.07 (0.68–6.4) 1.0
[97]	2009	RS	–	515	33/67	40–64	Finland	7	Age, sex, BMI, study center, smoking, alcohol intake, hypertension medication, leisure time physical activity, and 1 year change in body weight	Sleep (Hours) ⩽6.5 7–8.5 9–9.5 ⩾10	HR 1.68 (0.79–3.59) 1.0 2.29 (1.38–3.80) 2.74 (1.67–4.50)
[98]	2010	NIH-AARP [99]	PCS	174344	56.8/43.2	50–71	Whites and non-whites (USA)	8	Age, race, sex, educational level, marital status, smoking, coffee intake, alcohol intake, calorie intake, BMI, and physical activity	Day napping (Hours) 0 <1 ⩾1 Sleep (Hours) ⩽5 5–6 7–8 ⩾9	OR 1.0 1.23 (1.18–1.29) 1.55 (1.45–1.66) 1.46 (1.31–1.63) 1.11 (1.06–1.16) 1.0 1.11 (0.99–1.24)
[100]	2011	RS	CSS	3470 (5.2%)	61.8/ 38.2	⩾25	Taiwan	-	BMI, WHR, family history of diabetes, family history of hypertension, smoking, alcohol consumption and coffee intake	Sleep (Hours) <6 6–8.49 ⩾8.5	OR 1.55 (1.07–2.24) 1.0 2.83 (1.19–6.73)
[51]	2012	EPIC-Potsdam [101]	PCS	23620 (3.6%)	38.63/ 61.37	35–65	Germany	7.8	Age, sex, sleeping disorders, alcohol intake, smoking, walking, cycling, sports, employment status, education, BMI, WHR, hypertension, caffeinated beverages, life satisfaction, health satisfaction, and intake of antidepressants	Sleep (Hours) <6 6-<7 7-<8 8-<9 ⩾9	HR 1.06 (0.80–1.40) 0.94 (0.78–1.14) 1.0 0.92 (0.77–1.10) 1.05 (0.82–1.33)

Table 2 (*continued*)

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[102]	2012	RS	PCS	3570	78.6/ 21.4 (3.4%)	35–55	Japan	4	Age, sex, fasting plasma glucose level, education, working hours, shift work, rate of sedentary work, occupational stress, smoking, alcohol intake and physical exercise	Sleep (Hours) ⩽5 5–6 6–7 7–8	OR 5.37 (1.38–20.91) 1.38 (0.50–3.79) 1.57 (0.64–3.83) 1.0
[53]	2012	NHIS [103]	CSS	29818	53.5/ 46.5	18–85	Blacks and whites (USA)	10	Age, sex, income, hypertension, heart disease, depression and obesity	Sleep (Hours) 6–8 ⩽5 (Blacks) ⩽5 (Whites) ⩾9 (Blacks) ⩾9 (Whites)	OR 1.0 1.66 (1.19–2.30) 1.87 (1.57–2.24) 1.68 (1.21–2.33) 2.33 (1.98–2.73)
[104]	2013	IHHP [105]	CSS	12514	49/ 51	⩾19	–	–	Age, sex, BMI, and waist circumference	Sleep (Hours) ⩽5 6 7–8 ⩾9	OR 1.62 (1.33–1.99) 0.92 (0.75–1.13) 1.0 1.10 (0.83–1.44)
[106]	2013	MC [107]	PCS	47093 (1.85%)	74.4/ 25.6	Mean 34.9	USA	6	Age, sex, BMI, education and race	Sleep (Hours) ⩽5 5 6 7 8 ⩾8	OR 2.04 (1.49–2.8) 1.46 (1.15–1.84) 1.19 (0.99–1.43) 1.0 1.17 (0.95–1.45) 1.30 (0.93–1.81)
[54]	2013	NHIS [103]	CSS	130943 (10.12%)	99.75/ 0.25	Mean 50.6	Blacks and whites (USA)	7	Age, sex, household income, poverty status, education, occupation, employment status, alcohol consumption, smoking, leisure time physical activity, marital status, heart disease, hypertension, and BMI	Sleep (Hours) 7 ⩽6 (Blacks) ⩽6 (Whites) ⩾8 (Blacks) ⩾8 (Whites)	OR 1.0 1.08 (0.95–1.23) 1.16 (1.07–1.25) 1.01 (0.89–1.15) 1.17 (1.09–1.26)
[108]	2013	45 and up [109]	PCS	156902	36/ 64	50–82	Australia	-	Age, sex, education, marital status, residential remoteness, alcohol consumption, smoking status, health insurance status, income, BMI, physical activity and baseline health	Sleep (Hours) 7 ⩽6	HR 1.0 1.29 (1.08–1.53)

DIS-Difficulty Initiating Sleep, DMS-Difficulty Maintaining Sleep, EPIC-European Prospective Investigation into Cancer and Nutrition, FIN D2D-Finnish type 2 Diabetes, HIPOP-OHP-High risk and Population Strategy for Occupational Health Promotion, IHHP-Isfahan Healthy Heart Program, IRAS-Insulin Resistance Atherosclerosis Study, M-Men, MC-Millennium Cohort, MMAS-Massachusetts Male Aging Study, MONICA-Multinational MONItoring of trends and determinants in CArdiovascular disease, MPP-Malmo Preventive Project, NHANES-National Health and Nutrition Examination Survey, NHIS-National Health Interview Survey, NHS-Nurse Health Study, NHW-Non Hispanic Whites, NIH AARP-National Institutes of Health American Association of Retired Persons Diet and Health Study, QFS-Quebec Family Study, RS-Random Sample, SHHS-Sleep Heart Health Study, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

Table 3
Characteristics and findings of the studies examining the association between smoking and type 2 diabetes.

Work	Year	Study	Design	Sample Size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up Duration (Years)	Adjusted Variables	Findings	
[110]	1989	ZS [111]	PCS	841 (6.9%)	100/0	40-59	Dutch	25	Age, subscapular skin-fold, resting heart rate, cigarette use, alcohol intake and energy intake	**Cigarettes/day** 0 20	**HR** 1.0 3.3 (1.4-7.9)
[67]	1993	NHS [80]	PCS	114247 (2.04%)	0/100	30-55	USA	12	Age, BMI, family history of diabetes, menopause, postmenopausal hormone use, oral contraceptive use, alcohol consumption, and physical activity	**Cigarettes/day** 0 1-14 15-24 >25 Ex-smoker	**RR** 1.0 0.90 (0.68-1.19) 1.20 (0.96-1.50) 1.49 (1.19-1.87) 1.17 (1.02-1.35)
[68]	1995	HPFS	PCS	41810 (1.22%)	100/0	40-75	USA	62	Age, BMI, family history of diabetes, alcohol consumption and physical activity	**Cigarettes/day** 0 1-14 15-24 >25 Ex-smoker	**RR** 1.0 1.37 (0.77-2.43) 2.38 (1.57-3.59) 1.94 (1.25-3.03) 1.29 (1.05-1.57)
[112]	1997	RS	PCS	2312 (1.77%)	100/0	-	Japanese	8	-	**Cigarettes/day** 0 1-15 16-25 >26	**HR** 1.0 1.33 (0.40-4.39) 3.59 (1.32-9.76) 2.68 (0.88-8.05)
[73]	1997	SOF [113]	CSS	9435 (7%)	0/100	⩾65	Non-black (USA)	-	Age, resting heart rate, BMI, education level, alcohol intake, energy expenditure, WHR, and postmenopausal hormone use	**Cigarettes/day** 0 ⩽10 >10 Ex-smoker	**OR** 1.0 0.55 (0.30-0.99) 1.21 (0.87-1.71) 0.99 (0.82-1.19)
[114]	1999	OHS	PCS	6250 (7.2%)	100/0	25-60	Japan	16	Age, BMI, alcohol consumption, physical activity, parental history of diabetes, fasting plasma glucose, total cholesterol, and triglycerids	**Cigarettes/day** 0 1-20 21-30 >30	**RR** 1.0 1.40 (1.05-1.86) 1.40 (1.02-1.93) 173 (1.20-2.48)
[69]	2000	PHS [115]	PCS	21068 (3.65%)	100/0	40-84	USA	12.10	Age, BMI, physical activity, history of hypertension, history of high cholesterol, parental history of myocardial infarction, and alcohol consumption	**Cigarettes/day** 0 <20 ⩾20 Ex-smoker	**RR** 1.0 1.5 (1.0-2.2) 1.7 (1.3-2.3) 1.1 (1.0-1.4)
[116]	2001	RS	CSS	3718	19.2/ 80.0	12-88	Chinese	-	Age, BMI, alcohol consumption, and family history of diabetes	**Smoking** No Yes	**OR** 1.0 1.705 (1.106-2.630)
[65]	2001	BRHS [117]	PCS	7124 (4.07%)	100/0	40-59	UK	16.8	Age, BMI, physical activity, alcohol intake, social class, heart disease and antihypertensive treatment	**Smoking** No Yes Pipe/cigar Ex-smoker (15 yrs.) Ex-smoker (10 yrs.)	**RR** 1.0 1.61 (1.05-2.46) 2.15 (1.24-3.70) 1.45 (0.95-2.21) 2.03 (1.22-3.37)
[63]	2001	CPS-I [118]	PCS	709827 (3.6%)	38.8/ 61.2	⩾30	Whites and Blacks (USA)	13	Age, BMI, alcohol consumption, race, amount of exercise, education level, and intakes of fats and carbohydrates	**Cigarettes/day** 0 <20 (M) <20 (W) 20-39 (M) 20-39 (W) ⩾40 (M) ⩾40 (W) Ex-smoker (M) Ex-smoker (W)	**OR** 1.0 1.05 (0.98-1.12) 0.98 (0.93-1.03) 1.19 (1.13-1.26) 1.21 (1.14-1.29) 1.45 (1.34-1.57) 1.74 (1.49-2.03) 1.07 (1.02-1.13) 1.07 (0.99-1.15)
[119]	2001	NHS [80]	PCS	84941 (3.9%)	0/100	30-55	USA	16	Age, family history of diabetes, menopausal status, postmenopausal hormone use, fat intake, and physical activity	**Cigarettes/day** 0 1-14 ⩾15	**OR** 1.0 1.14 (0.85-1.54) 1.40 (1.14-1.71)
[120]	2002	NCDS [121]	-	15396	M/W	-	UK	33	Maternal smoking during pregnancy, sex, mother's age at the time of giving birth, age at which mother left school, family social class at birth, birth weight, own smoking at the age of 16, and BMI at the	**Cigarettes/week Self** 0 <1 1-9 10-19 20-29 ⩾30 **Mother** Non-smoker Medium-smoker Medium to heavy-smoker Heavy-smoker	**OR** 1.0 2.07 (0.25-17.19) 1.92 (0.52-7.10) 2.48 (0.52-11.97) 1.61 (0.20-12.96) 3.62 (1.42-9.24) 1.0 1.01 (0.23-4.53) 3.53 (0.88-14.38) 4.02 (1.14-14.14)

Table 3 (*continued*)

Work	Year	Study	Design	Sample Size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up Duration (Years)	Adjusted Variables	Findings	
									age of 33		
[74]	2004	RIH	CSS	27777	45/ 55	20-69	France	-	Age, BMI, WHR, and alcohol consumption	**Smoking** No Yes (M) Yes (W) Ex-smoker (M) Ex-smoker (W)	**OR** 1.0 1.49 (1.13-1.96) 0.89 (0.54-1.39) 1.31 (1.01-1.70) 1.46 (0.92-2.22)
[122]	2004	NTHS [123]	PCS	38805	46.9/ 53.1	⩾20	Norwegian	11	Age, BMI, and sex	**Cigarettes/day** 0 ⩾20	**RR** 1.0 1.64 (1.12-2.39)
[70]	2005	IRAS [95]	PCS	906 (25%)	43.3/ 56.7	40-69	Non-Hispanic Whites, Hispanics, and African-Americans	5	Age, sex, ethnicity, BMI, WHR, glucose tolerance status, HDL cholesterol level, triglyceride level and hypertension	**Smoking** No Ex-smoker Current-smoker	**OR** 1.0 1.31 (0.82-2.09) 2.66 (1.49-4.77)
[77]	2006	KMIC [124]	PCS	27635	100/0	35-44	Korea	8	Age, baseline fasting serum, glucose, weight change, baseline BMI, family history of diabetes, alcohol consumption, and physical activity	**Cigarettes/day** No <10 10-19 ⩾20 Ex-smoker (⩾8 yrs.) Ex-smoker (7-7.9 yrs.) Ex-smoker (5-6.9 yrs.)	**OR** 1.0 1.23 (1.86-1.77) 1.60 (1.28-2.00) 1.75 (1.35-2.27) 0.95 (0.72-1.25) 1.44 (0.96-2.15) 2.13 (1.51-3.00)
[71]	2009	RS	PCS	-	M/W	40-69	Ansung and Ansan Korean	4	Age, family history of diabetes, rural or urban area, waist, body fat, exercise, alcohol consumption, income, education, WBC, HDL cholesterol, triglyceride, systolic BP, HOMA IR, and HOMA beta	**Cigarettes/day** No <20 Y⩾20 Ex-smoker	**RR** 1.0 2.06 (1.35-3.16) 2.41 (1.48-3.93) 1.60 (1.07-2.39)
[75]	2010	ARIC [36]	PCS	10892 (11.51%)	43.3/ 56.7	45-64	Whites and Non-whites (USA)	9	Race, sex, level of education, BMI, waist circumference, baseline age, physical activity, HDL cholesterol, triglycerides, and systolic BP	**Smoking** No Ex-smoker (9 yrs.) Ex-smoker (6-9 yrs.) Ex-smoker (3-6 yrs.) Ex-smoker (<3 yrs.) Current-smoker	**HR** 1.0 1.16 (0.99-1.36) 1.21 (0.89-1.65) 1.54 (1.10-2.14) 1.80 (1.44-2.25) 1.26 (1.08-1.46)
[66]	2010	KORA S4/F4 [125]	PCS	885	50.4/ 49.6	55-74	Germany	7	Age, sex, parental diabetes, socioeconomic status, alcohol intake, physical activity, intake of meat and sausage, intake of salad and vegetables, intake of whole grain bread, coffee consumption, waist circumference, blood pressure, hypertriglyceridemia, HDL cholesterol, log insulin and log adiponectin	**Smoking** No (passive+active) Passive Passive+prediabetes Active Active+prediabetes	**OR** 1.0 2.5 (1.1-5.6) 4.4 (1.5-13.4) 2.8 (1.3-6.1) 7.8 (2.4-25.7)
[64]	2010	KCPS [126]	PCS	1236443	63.7/ 36.3	30-95	Korea	14	Age, alcohol drinking, BMI, and physical exercise	**Cigarettes/day** No 1-9 (M) 1-9 (W) 10-19 (M) 10-19 (W) ⩾20 (M) ⩾20 (W)	**HR** 1.0 1.30 (1.25-1.32) 1.34 (1.25-1.44) 1.37 (1.34-1.41) 1.26 (1.14-1.38) 1.55 (1.51-1.60) 1.33 (1.15-1.53)
[72]	2011	NHS [80]	PCS	100526 (5.36%)	0/100	41-55	USA	24	Age, BMI, physical activity, husband's education, family history of diabetes, total energy intake, alcohol intake, caffeine, total transa fat, toatl saturated fat, calcium, magnesium and vitamin D	**Cigarettes/day** No Low passive High passive 1-14 15-24 ⩾25 Ex-smoker	**RR** 1.0 1.10 (0.94-1.23) 1.16 (1-1.35) 1.39 (1.17-1.64) 1.68 (1.43-2.01) 1.98 (1.57-2.36) 1.28 (1.12-1.50)
[78]	2012	JPHC [127]	PCS	59834	43.24/ 56.76	Mean 55-57.9	Japanese	5 and 10	Age, BMI, history of hypertension, alcohol intake, family history of diabetes, weight change, study area, and leisure time physical activity	**Smoking** No Current-smoker (M) Current-smoker (W) Ex-smoker (<5 yrs.) (M) Ex-smoker (<5 yrs.) (W)	**OR** 1.0 1.43 (1.16-1.76) 1.42 (1.03-1.94) 1.68 (1.07-2.63) 2.84 (1.53-5.29)

(*continued on next page*)

Table 3 (*continued*)

Work	Year	Study	Design	Sample Size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up Duration (Years)	Adjusted Variables	Findings
[76]	2012	RS	PCS	2070 (11.9%)	100/0	40-69	Japan	9.2	Age, blood glucose, fasting, systolic BP, total cholesterol, log-transformed triglycerides, alcohol consumption, exercise, family history of diabetes, BMI, and change in smoking status during follow-up period	**Smoking** No Ex-smoker (>9 yrs.) Ex-smoker (6-9 yrs.) Ex-smoker (3-5 yrs.) Ex-smoker (<3 yrs.) Current-smoker **HR** 1.0 2.22 (1.05-4.69) 0.59 (0.13-2.64) 1.95 (0.62-6.17) 1.91 (0.60-6.06) 2.78 (1.43-5.41)
[128]	2013	WHI [129]	PCS	11838	0/100	50-79	USA	11	Age, ethnicity, education, BMI, waist circumference, alcohol consumption, physical activvity, hypertension and medication for high cholesterol	**Smoking** No Current-smoker (M) Ex-smoker (<3 yrs.) **HR** 1.0 1.28 (1.20-1.36) 1.43 (1.26-1.63)

ZS-Zutphen Study, NHS-Nurse Health Study, NHIS-National Health Interview Survey, HPFS-Health Professionals' Follow-up Study, RS-Random Sample, SOF-Study of Osteoporotic Fractures, OHS-Osaka Health Survey, PHS-Physicians Health Survey, BRHS-British Regional Health Study, CPS-Cancer Prevention Study, NCDS-National Child Development Study, RIH-Regional Institute for Health, NTHS-Nord Trondelag Health Survey, IRAS-Insulin Resistance Atherosclerosis Study, ARIC-Atherosclerosis Risk in Communities, KCPS-Korean Cancer Prevention Study, JPHC-Japan Public Health Center, WHI-Women Health Initiative, KMIC-Korean Medical Insurance Corporation, M-Men, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

The results in the literature show that compared to a reference sleep duration of 7-8 h, an individual having either short sleep duration (<6 h) or long sleep duration (>8 h) is at high risk of developing type 2 diabetes. However, [50,51] concluded that there is no significant association between sleep and incidence of type 2 diabetes. Mallon et al. [52] studied the impact of gender on the association between sleep and diabetes. The authors concluded that short sleep duration increases the risk of incidence diabetes in men, whereas, in women, long sleep duration dominates. The effect of ethnicity on the association is analyzed by [53–55]. Zizi et al. [53] and Jackson et al. [54] showed that the prevalence of type 2 diabetes is more in whites who sleep less than 5 h or more than 8 -9 h compared to blacks. Beihl [55] showed that the association is more in Hispanics/Non-Hispanic Whites compared to that in African-American. Xu et al. examined the association between day-time napping and type 2 diabetes and showed that an individual taking more than 1 h of day-time nap is at 1.5 times more risk to develop diabetes compared to an individual who does not take a nap during the day. In the context of sleep quality, the risk of incidence type 2 diabetes is more in an individual having difficulty initiating sleep (DIS), and the risk increases with increasing DIS frequency [56–58]. Furthermore, the association is more in women having DIS compared to men [59].

In summary, there is a strong association between sleep quantity/quality and the incidence of type 2 diabetes. The association is stronger in women sleeping for more duration and in men with short sleep duration. Moreover, this association is affected by ethnicity.

3.3. Smoking

Smoking leads to more than 8 million deaths per year [60]. This is from both active and passive uses, i.e, non-smokers exposed to smokers. Smokers are 30–40% more likely to develop type 2 diabetes compared to non-smokers [61]. When an individual smokes, the level of nicotine increases in his/her body. This leads to a reduction in muscle glucose intake, developing insulin resistance and leading to type 2 diabetes [62]. The characteristics and findings of table:smokingtable:smoking/passive smoking and the incidence of type 2 diabetes are presented in Table 3.

The results in the literature show that the association between smoking and diabetes increases with an increase in the number of cigarettes smoked/day. Will et al. [63] analyzed the impact of gender on this association and showed that the association between cigarette smoking and type 2 diabetes is more in men compared to women. Similar results are obtained by Jee et al. [64]. Wannamethee et al. [65] revealed that an individual smoking pipe/cigar is 2.15 times more likely to develop type 2 diabetes and an individual smoking cigarette is 1.6 times more likely compared to a non-smoker. Kowall et al. [66] showed that the risk of incidence type 2 diabetes is significantly high in active/passive prediabetic smokers compared to active/passive smokers without prediabetes.

The incidence and prevalence of type 2 diabetes in ex-smokers is examined by [67–72]. and [73] respectively. Results show that ex-smokers are associated with 17–60% increased risk of type 2 diabetes [67,68,70–72]. However, the results obtained by Simon et al. [73] and Manson et al. [69] showed no association between ex-smokers and type 2 diabetes. This discrepancy in the results can be due to the heterogeneous characteristics (sample size, age range, men/women ratio and ethnicity) of the cohorts used in these studies. Beziaud et al. [74] examined gender-based prevalence of type 2 diabetes in ex-smokers and showed that women are at higher risk compared to men. Furthermore, the duration of smoking cessation also impacts the association in ex-smokers [65,75–77]. An individual is at high risk of developing type 2 diabetes during first 5–10 years of smoking cessation. The risk then

Table 4
Characteristics and findings of the studies examining the association between depression and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up Duration (Years)	Adjusted variables	Findings	
[137]	1991	RS	PCS	2380 (1.72%)	100/0	⩾18	Japanese	8	Age	Depression (SDS score)	HR
										20–39	1.0
										40–47	1.07 (0.53–2.13)
										48–80	2.32 (1.06–5.08)
[155]	1996	ECAPS [156]	-	1715 (5.2%)	37.8/ 62.2	⩾18	USA	13	Age, sex, race and BMI	Depression	OR
										No	1.0
										Yes	2.23 (0.90–5.55)
[157]	2003	NHANES I [88]	PCS	6190	45.7/ 54.3	25–74	Whites and Non-whites (USA)	15.6	Age, sex and race	Depression	RR
										No	1.0
										Mild	1.24 (0.91–1.70)
										Major	2.52 (1.73–3.67)
[158]	2004	ARIC [36]	PCS	11615	44.85/ 55.15	48–67	Whites and Non-whites (USA)	6	Age, sex, race, study site, fasting insulin, fasting glucose, HDL cholesterol, BMI, WHR, systolic BP, physical activity, total calorie intake, smoking status, and education	Depression	HR
										No	1.0
										Low	1.12 (0.90–1.39)
										Mild	1.03 (0.81–1.31)
										Major	1.31 (1.04–1.64)
[136]	2004	SWAN [159]	PCS	2662 (3.64%)	0/100	42–52	Caucasian, African-American, Hispanic, Japanese-American and Chinese-American	3	Age, study site, race, education, and medication use	Depressed African-Americans are 2.56 times more likely rightarrow have diabetes.	
[160]	2007	NTHS [123]	PCS	37291	47.2/ 52.8	⩾29	Norwegian	10	Age, sex, education, smoking, physical activity, BMI, WHR, waist circumference, and marital status	Depression	OR
										No	1.0
										Yes	1.40 (1.16–1.69)
[138]	2007	CHS [161]	PCS	4681	40.8/ 59.2	⩾65	USA	8	Age, race, sex, educational level, marital status, physical activity, smoking, alcohol consumption, BMI, and reactive protein level	Depression (CES-D score)	OR
										<8	1.0
										⩾8	1.57 (1.07–2.29)
[139]	2014	RBHCDS	-	971	43/ 57	⩾50	California	8	Age, sex, BMI and exercise	Depression (BDI score)	OR
										<11	1.0
										⩾11	2.50 (1.29–4.87)

RS-Random Sample, SDS-Self rating Depression Scale, ECAPS-Epidemiologic Catchment Area Program Survey, NHANES-National Health and Nutrition Examination Survey, ARIC-Atherosclerosis Risk in Communities, RNH-RegistratieNet Huisarts Praktijken, SWAN-Study of Womens' Health Across the Nation, NTHS-Nord Trondelag Health Study, CHS-Cardiovascular Health Study, CESD-Center for Epidemiological Studies Depression Scale, RBHCDS-Rancho Bernardo Heart and Chronic Disease Study, BDI-Beck Depression Inventory, M-Men, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

decreases with an increase in cessation duration. The association between smoking cessation and the incidence of type 2 diabetes is more in women than men [78].

In summary, both active and passive smoking are strongly associated with the incidence of type 2 diabetes. The association is more in men compared to women. Moreover, the association remains significant in ex-smokers during first the 5–10 years of smoking. After 10 years of smoking cessation, the risk of incidence type 2 diabetes is the same as that in a non-smoker. Women ex-smokers are at a higher risk of developing diabetes compared to men ex-smokers.

3.4. Depression

Depression is a mood disorder that negatively affects the way a person feels, thinks and acts [130]. It can be due to a family history of depression, early childhood trauma, brain structure, medical conditions, drug use or surrounding environment. Depression is associated with multiple health conditions including diabetes [131]. It elevates the sympathetic nervous system activities and hypothalamic–pituitary–adrenal axis activities [132]. Elevated sympathetic nervous system activities lead to an increase in catecholamines and inflammation, and eventually causing insulin resistance [133]. On the other hand, elevated adrenal axis activities lead to an increase in cortisol and eventually blood sugar level [134]. Both insulin resistance and increased blood sugar levels develop type 2 diabetes. The characteristics and findings of the work in the literature examining the association between depression and the incidence of type 2 diabetes are presented in Table 4.

The results show that depression is highly associated with the incidence of type 2 diabetes. In the context of gender, depressed men are at higher risk of incidence type 2 diabetes, whereas depression in women is not associated with type 2 diabetes [135]. Moreover, compared to Caucasian, Hispanic, Japanese-American and Chinese-American, depressed African-Americans are at 2.56 times higher risk of incidence type 2 diabetes [136]. Based on self rating depression scale (SDS) score, an individual having a score of 48–80 is at higher risk of developing diabetes compared to an individual having a score of 20–39 [137]. Similarly, an individual having a score $\geqslant$11 using center for epidemiological studies depression scale (CES-D) or a score $\geqslant$8 using beck depression inventory (BDI) is at higher risk of incidence type 2 diabetes [138,139].

In summary, depression is associated with type 2 diabetes. However, the association is different in men and women. Moreover, the study by Yu et al. [140] show that depression itself is not a risk factor for diabetes, rather the activities related to depression such as physical inactivity, poor diet, and obesity lead to diabetes. In addition, the medical drugs used to treat depression also have an association with the incidence of type 2 diabetes. Consequently, similar to high-level serum uric acid, depression is not an independent risk factor but it emphasizes the impact of other independent risk factors such as gender, ethnicity, physical inactivity, and obesity.

3.5. Cardiovascular disease

Increased heart rate and cardiovascular disease can elevate the blood pressure in the arteries. As a result, the body's glucose uptake decreases leading to insulin resistance condition. Consequently, a person suffering from heart disease is at a higher risk of developing type 2 diabetes. However, this association is still obscure. Few studies argue that a history of cardiovascular disease leads to the incidence of type 2 diabetes [141], while others claim that type 2 diabetes increases the risk of cardiovascular disease [142–144]. Yeung et al. [141] examined the association between family history of coronary heart disease (CHD) and type 2 diabetes (Table 5). The authors concluded that a high family CHD score is associated to the incidence of type 2 diabetes in individuals who have a positive history of family diabetes. For the individuals having a negative family history of diabetes, this association was non-significant. In summary, it is debatable whether cardiovascular disease is a risk factor for type 2 diabetes or not.

3.6. Dyslipidemia

Dyslipidemia refers to an abnormal level of lipids, such as triglycerides and cholesterol. It is characterized by high triglyceride levels, increased low-density lipoproteins (LDL) levels and decreased high-density lipoproteins (HDL) levels [145]. Elevated LDL and lowered HDL levels lead to beta-cell dysfunction inhibiting insulin secretion and consequently type 2 diabetes [146,147]. Table 6 shows the characteristics and findings of the work in the literature studying the association between dyslipidemia and type 2 diabetes.

Dietary fats, that raise the total cholesterol and LDL levels, are considered significant in the development of type 2 diabetes [148]. Substituting saturated fatty acid with polyunsaturated fatty acid and animal fat with vegetable fat can help lower blood cholesterol and eventually type 2 diabetes. This is because both polyunsaturated fatty acid and vegetable fat are inversely related to the risk of incidence type 2 diabetes with RR 0.84 (95% CI 0.71–0.98) and RR 0.78 (95% CI 0.67–0.91) respectively for the highest quintile of intake [148]. Tajima et al. [149] also confirmed the association between high cholesterol diet intake (>273 mg/day) and type 2 diabetes (RR 1.25, 95% CI 1.16–1.36) compared to low cholesterol intake (<185 mg/day).

In order to reduce elevated LDL level, LDL lowering therapy and drugs are suggested. However, these drugs and therapy are found to be associated with a higher risk of type 2 diabetes [150]. Individuals having familial hypercholesterolemia, a genetic disorder that results in high LDL levels, are less likely to have type 2 diabetes

Table 5
Characteristics and findings of the studies examining the association between cardiovascular disease and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[141]	2007	ARIC [36]	PCS	11297 (11.52%)	M/W	45–64	Blacks and Whites (USA)	9	Age, sex, race, smoking, alcohol consumption, educational level, leisure index, BMI, WHR, systolic and diastolic pressure, triglycerides, HDL, glucose, hypertension, WBC count, and fibrinogen	CHD risk score <-0.5 −0.5 to 0.49 $\geqslant$0.5	HR 1.0 1.23 (0.98–1.54) 1.43 (1.03–1.99)

ARIC-Atherosclerosis Risk in Communities, CDH-Coronary Heart Disease, M-Men, W-Women, PCS-Prospective Cohort Study.

Table 6
Characteristics and findings of the studies examining the association between dyslipidemia and type 2 diabetes.

Work	Year	Study	Design	Sample Size(%DM)	%M/W	Age (Years)	Ethnicity	Follow-up Duration (Years)	Adjusted Variables	Findings	
[148]	2001	LWHS [162]	PCS	35988	0/100	55–69	USA	11	Age, total energy, WHR, BMI, physical activity, cigarette smoking, alcohol consumption, education, marital status, residential area and hormone replacement therapy	Median cholesterol intake (mg/day) 185 201 237 281 382	RR 1.0 0.87 (0.74–1.03) 1.07 (0.91–1.25) 1.10 (0.94–1.28) 1.24 (1.07–1.43)
[154]	2015	CCHS [163] and CGPS [164]	PCS	47627	M/W	⩾20	Danish	36	Age, sex, study, BMI, hypertension, smoking, alcohol intake, physical inactivity, postmenopausal status and hormonal replacement in women, lipid lowering therapy, and educational level	HDL cholesterol (m mol/L) 2.5 2 1.5 1	RR 1.0 1.44 (1.08–1.91) 2.72 (2.09–3.54) 5.74 (4.43–7.43)
[152]	2018	REACTION [165]	PCS	4882 (14.42%)	36.5/ 63.5	⩾40	Chinese	3	Age, sex, smoking, alcohol, physical activity, family history of diabetes, BMI, and systolic blood pressure	Non-HDL/ HDL(m mol/ L) 1.4 1.9 2.4 3.1	OR 1.0 1.2 (0.9–1.5) 1.2 (0.9–1.5) 1.4 (1.1–1.8)

LWHS-Lowa Women's Health Study, CCHS-Copenhagen City heart Study, CGPS-Copenhagen General Population Study, MA-Meta Analysis, REACTION-Risk Evaluation of cAncers in Chinese diabeTic Individuals: a lONgitudinal study, RS-Random Sample, M-Men, W-Women, PCS-Prospective Cohort Study.

compared to individuals having high LDL levels due to dietary patterns [151]. Zhang et al. [152] in their analysis found that the ratio of non-HDL and HDL levels is an independent risk factor for incidence diabetes. They show that an individual having a ratio of 3.1 is at 40% increased risk of incidence diabetes (OR 1.4, 95% CI 1.1–1.8) compared to an individual having a ratio of 1.4. Elevated non-HDL and lowered HDL levels are significantly associated with incidence diabetes [153].

On the contrary to studies confirming the association between low-HDL levels and the incidence of type 2 diabetes, Haase et al. [154] in their study concluded that a life-long reduction in HDL levels are not associated with an increased risk of type 2 diabetes. They found that the association is most likely reverse causation, i.e., type 2 diabetes leads to low HDL levels.

3.7. Hypertension

Hypertension, also known as high blood pressure, is a medical condition in which the blood pressure in the arteries is persistently elevated. Hypertension elevates the sympathetic nervous system activity leading to a decrease in the body's glucose uptake. This causes the condition of insulin resistance and eventually type 2 diabetes. Hypertension elevates sympathetic nervous system activities leading to impaired vasodilation of skeletal muscles. Consequently, muscle glucose uptake decreases with the eventual development of type 2 diabetes. Table 7 shows the characteristics and findings of the work in the literature studying the association between hypertension and type 2 diabetes.

Hayashi et al. [166] examined the association between high normal blood pressure (⩾130 and <140 mmHg/⩾85 and <90) and hypertension (⩾140 mmHg/⩾90 mmHg), and the incidence of type 2 diabetes in men. The authors concluded that both high normal blood pressure (RR 1.39, 95%1.14–1.69) and hypertension (RR 1.75, 95% CI 1.43–2.16) are associated with an increased risk of type 2 diabetes. This association is dependent on obesity and hypertension medications. Hypertension medications are considered to increase the risk of diabetes depending on the type of medication [167]. For instance, hypertensive individuals taking thiazide diuretics and angiotensin-converting-enzyme medications are at lower risk of diabetes compared to the hypertensive individuals not taking any medication. However, those taking beta-blockers medication are at 28% higher risk of incidence type 2 diabetes (HR 1.28, 95% CI 1.04–1.57) [167]. The association between hypertension and the incidence of type 2 diabetes is significant in women as well [168]. Women having hypertension are at 2 times increased risk of developing diabetes (HR 2.03, 95% CI 1.77–2.32) compared to women having normal blood pressure (<120/75) [168]. The association is more in overweight and obese women. Irrespective of gender, prehypertension (HR 1.27, 95%CI 1.09–1.48) and hypertension (HR 1.51, 95% CI 1.29–1.76) are associated with increased risk of incidence type 2 diabetes [169]. In the context of ethnicity, whites individuals having hypertension are at higher risk of developing diabetes (HR 1.25, 95% CI 1.03–1.53), but no such association is seen in African American hypertensive individuals (HR 0.92, 95% CI 0.70–1.21) [170].

In summary, hypertension is associated with the development of type 2 diabetes in both men and women. However, the association is ethnicity-dependent. The selection of hypertensive medications should be made properly as the medication impacts the strength of the association. Furthermore, an obese individual with hypertension is at higher risk compared to a non-obese.

3.8. Aging

The number of elderly people (above 60 years) is increasing worldwide. The 900 million global elderly population in 2015 is expected to rise to 2 billion by 2050 [171]. Aging increases the risk of metabolic syndrome and chronic diseases including type 2 diabetes. Aging increases chronic inflammation in an elderly individual leading to insulin resistance [172]. In addition, lipid metabolism disorder due to aging increases the accumulation of body fat leading to elevated free fatty acids concentration in the

Table 7
Characteristics and findings of the studies examining the association between hypertension and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[166]	1999	RS	PCS	7594 (7.9%)	100/0	30–65	Japanese	16	Age, BMI, alcohol consumption, smoking habits, leisure time physical activity, and parental history of diabetes	Blood pressure in mmHg ⩽130/⩽85 130–139/85–89 ⩾139/⩾89	RR 1.0 1.39 (1.14–1.69) 1.76 (1.43–2.16)
[167]	2000	ARIC [36]	PCS	12550	44.39/ 55.61	45–64	Blacks and Whites (USA)	3 and 6	Age, sex, race, BMI, WHR, educational level, smoking status, alcohol consumption, physical activity, systolic and diastolic blood pressure, fasting serum insulin concentration, history of hypercholesterolemia, cardiovuscular diseases, pulmonary dieseases, renal insufficiency, and family history of diabetes	Hypertension medication None ACE inhibitor Beta-blocker Calcium-channel antagonist Thiazide diuretic	HR 1.0 0.98 (0.72–1.34) 1.28 (1.04–1.57) 1.17 (0.83–1.66) 0.91 (0.73–1.13)
[168]	2007	WHS [185]	PCS	38172 (4.38%)	0/100	⩾45	USA	10.2	Age, ethnicity, smoking, BMI, exercise, alcohol consumption, history of hypercholesterolameia, educational level, family history of diabetes, and randomized treatment assignments	Blood pressure in mmHg 120–129/75–84 130–139/85–89 ⩾140/⩾90	HR 1.0 1.45 (1.23–1.71) 2.03 (1.77–2.32)
[170]	2011	ARIC [36], CARDIA [186], and FHS [187]	PCS	10893 (9.45%)	43/57	35–54	African-American and Whites (USA)	Median 8.9	Age, sex, BMI, fasting glucose, DL cholesterol and triglycerids	Blood pressure in mmHg *leq*119/*leq*79 120–139/80–89 ⩾140/⩾90	HR 1.0 1.32 (1.09–1.61) 1.25 (1.03–1.53)
[188]	2012	GPPS [189]	PCS	7494 (12.02%)	100/0	47–55	Swedish	35	Age, BMI, cholesterol level, antihypertensive treatment, smoking, physical activity and occupational class	Blood pressure in mmHg *leq*129 130–159 ⩾160 ⩽84 85–89 ⩾90	HR 1.0 1.43 (1.12–1.84) 1.95 (1.55–2.46) 1.0 1.34 (1.12–1.62) 1.08 (1.06–1.11)
[169]	2015	KGES [190]	PCS	7150 (14.7%)	47.46/ 52.54	40–69	Korean	8	Age, BMI, fasting plasma glucose, total cholesterol, HDL cholesterol, family history of diabetes, education, alcohol consumption and smoking status	Blood pressure in mmHg ⩽120/⩽80 120–139/80–89 (M) 120–139/80–89 (W) ⩾140/⩾90 (M) ⩾140/⩾90 (W)	HR 1.0 1.24 (1.01–1.52) 1.30 (1.03–1.64) 1.65 (1.34–2.05) 1.34 (1.05–1.70)

RS-Random Sample, ARIC-Atherosclerosis Risk in Communities, WHS, Women's Health Study, CARDIA-Coronary Artery Risk Development in Young Adults, FHS-Framingham Heart Study, GPPS-Gothenburg Primary Prevention Study, KGES-Korean Genome and Epidemiology Study, M-Men, W-Women, PCS-Prospective Cohort Study.

blood/plasma and eventually insulin resistance [173]. Consequently, an aged individual is at higher risk of developing type 2 diabetes. However, there is not much work concluding that aging is an independent risk factor for type 2 diabetes. Choi et al. [174] concluded that the risk of diabetes increases with aging only in overweight individuals, and the risk decreases with a moderate level of physical activity. Aging can be considered as triggering the association between independent risk factors and risk of diabetes, but more evidence and studies are required to examine the association between aging as an independent factor and diabetes.

3.9. Ethnicity

Ethnicity is associated with a range of health complications including diabetes because of the heterogeneity in the demographic environmental conditions and lifestyle. It is an independent risk factor which tends to be exacerbated by the social disadvantage and the affluent way of living. Table 8 shows the characteristics and findings of the work in the literature studying the association between ethnicity and type 2 diabetes. Compared to white individuals, type 2 diabetes is more prevalent in Pacific Islanders (OR 3.1, 95% CI 1.4–6.8), followed by Blacks (OR 2.3, 95% CI 2.1–2.6), Native Americans (OR 2.2, 95% CI 1.6–2.9), Hispanics (OR 2.0, 95% CI 1.8–2.3), and Multiracial (OR 1.8, 95% CI 1.5–2.9) [175]. In another study by Shai et al. [176], it was found that compared to whites, Asians (RR 1.94, 95% CI 1.46–2.58), Hispanics (RR 1.70, 95% CI 1.28–2.26), and Blacks (RR 1.36, 95% CI 1.14–1.63) are at higher risk of incidence type 2 diabetes.

A study by Zimmet et al. [177] showed that type 2 diabetes is 10 times more prevalent in rural Indians compared to rural Melanesians, and 2 times more prevalent in urban Indians compared to urban Melanesians. They also revealed that the prevalence is 5 times more in urban Melanesians compared to rural Melanesians. One of the reason could be that the rural residents have an increased amount of physical activity compared to the urban ones, leading to decreased risk of diabetes [178]. It should thus important to have a moderate amount of physical activity as a therapy for diabetes prevention. Compared to Europeans, type 2 diabetes is 3.8 times more prevalent in Indians, and the prevalence increases to 5 times for 40–64 years old individuals [179]. In another comparison between Asian and non-Asian ethnicity, it is found that the prevalence of type 2 diabetes in Bangladeshis (Asians) is more [180]. Furthermore, the prevalence is high in women (5.75 times) compared to that in men (2.2 times). However, ethnicity can not be considered as an independent risk factor for this association as Bangladeshis had higher smoking rates and a lower ratio of polyunsaturated fatty acids to saturated fatty acids. Consequently, ethnicity, smoking and dyslipidemia all contributed to the risk of incidence type 2 diabetes. Simmons et al. [181] also confirmed in their study that the prevalence is more in Asians compared to Whites. However, in contrast to the results obtained by [180], Simmons et al. [181] found that the prevalence is more in men compared to women. This inconsistency should be examined further.

In summary, ethnicity is associated with the incidence of type 2 diabetes. However, there is no definite explanation of why individuals of a particular ethnicity are at higher risk of type 2 diabetes compared to the others. One possible explanation can be the ethnicity-dependent relation between BMI and body fat. For instance, Asians have around 3–4 kg/m^2 lower BMI compared to Caucasians for a given percentage of body fat [182]. Another reason could be ethnicity-based insulin sensitivity. Studies show that Asians, Blacks and Mexican Americans are less insulin sensitive compared to non-Hispanic Whites [183,184].

3.10. Family history of diabetes

Family history information can serve as a useful tool for prognosis/diagnosis and public health. Family history of diabetes reflects both genetic as well as environmental factors and can lead to better prediction of incidence type 2 diabetes than only genetic factors and environmental factors alone [192]. Table 9 shows the characteristics and findings of the work in the literature studying the association between family history of diabetes and type 2 diabetes.

A study by Tsenkova et al. [193] revealed that a family history of diabetes is strongly associated with incidence diabetes (OR 2.77, 95% CI 2.03–3.78). Another study also shows that parental history of diabetes is an independent risk factor for diabetes (OR 1.73, 95% CI 1.29–2.33) [194]. However, the association becomes weaker in men free of cardiovascular disease (OR 1.63, 95% CI 1.18-.2.24). Moreover, the association is much higher in 45–54 years old men (OR 1.99, 95% CI 1.38–2.89) compared to 55–68 years old men (OR 1.33, 95% CI 0.70–2.52). Furthermore, the prevalence of type 2 diabetes is stronger in men compared to women [195]. This indicates that parental history of diabetes in combination with other risk factors such as aging, gender and cardiovascular diseases, increases the risk of incidence type 2 diabetes.

Rodríguez-Moran et al. [196] showed that a family history of diabetes in first degree of relative (parents, offspring and siblings) is a strong and independent risk factor for the prevalence of impaired fasting glucose (prediabetes) (OR 11.7, 95% 9.5–21.2) in children and adolescents. This is in the absence of obesity. The results reveal that is it important to consider the parental history of diabetes while screening for diabetes children and adolescents. This is because only obesity-based screening could lead to underestimation. Valdez et al. [197] also showed that the family history of diabetes in at least two first-degree relatives or one first-degree and at least two second-degree relatives is significant for prevalence of type 2 diabetes. However, it can not be denied that the presence of a family history of diabetes can make the association between obesity and diabetes stronger [198]. Given a BMI $\geqslant$ 35, an individual with a family history of diabetes is at a higher risk of incidence diabetes (OR 26.7, 95% CI 14.4–49.4) compared to the one without a family history of diabetes (OR 6.1, 95% CI3.4–11.2). Furthermore, ethnicity is also considered an important factor in an obese individual with a family history of diabetes [199,200].

An individual having a family history of diabetes can have an early onset of diabetes compared to the ones without a family history. However, it is hard to conclude that which among the maternal, paternal and both maternal and paternal family history of diabetes is more significant for incidence/prevalence of type 2 diabetes as the results in the literature are inconsistent [195,201–205].

3.11. Obesity

Obesity is a complex health condition that involves an excessive amount of body fat. It is defined by the BMI and further evaluated in terms of fat distribution via the waist-hip ratio. Abdominal fat in the body increases inflammation which decreases insulin sensitivity by disrupting the function of beta-cells. The insulin resistance condition then leads to the prevalence of type 2 diabetes. Table 10 shows the characteristics and findings of the work in the literature studying the association between obesity and type 2 diabetes.

Ishikawa-Takata et al. [206] found that the risk of diabetes increases significantly for an individual having a BMI greater than 29 kg/m^2. The relative risk of diabetes increases up to 38.8 (95% CI 31.9–47.2) for an individual having a BMI greater than 34.9 kg/m^2 [119]. Furthermore, study shows that the association between obesity and incidence diabetes is gender-dependent [207]. For each

Table 8
Characteristics and findings of the studies examining the association between ethnicity and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings
[177]	1983	RS	-	2638	46.81/53.19	⩾20	Melanesians and Indians	-	Age	The prevalence of diabetes in rural Indian men is 7.5 times more than rural Melanesian men, and is 2.93 times more in urban Indian males compared to urban Melanesian men. For women, the prevalence in rural and urban Indians is 12.6 and 1.5 times more compared to rural and urban Melanesians respectively.
[179]	1985	RS	-	61130 (1.87%)	M/W	All age	Asians and Europeans	-	Age	The prevalence of diabetes in Asians was 3.8 times higher than in Europeans. For the patients age between 40–64, the prevalence was at least 5 times higher in Asians.
[180]	1988	RS	-	253	65.6/ 34.4	35–69	Bangladeshi and Non-Asian	-	Age	The prevalence of diabetes in Bangladeshi men and women is 2.2 and 5.75 times compared to Non-Asian men and women respectively.
[181]	1989	RS	-	4020	48.4/ 51.6	20–79	Asian and White	-	Age	The prevalence of diabetes in Asian men and women are 4 and 2 times compared to White men (11.2% vs 2.8%) and women (8.9% vs 4.3%) respectively.
[175]	2003	BRFSS [191]	-	163584	48.6/51.4	⩾30	Asian, Black, Hispanic, Native American, Pacific Islander, White, Other and Multiracial	-	Age, sex and BMI	Ethnicity OR White 1.0 Asian 1.0 (10.7–1.4) Black 2.3 (2.1–2.6) Hispanic 2.0 (1.8–2.3) Native 2.2 (1.6–2.9) American 3.1 (1.4–6.8) Pacific 1.4 (1.0–1.9) Islander 1.9 (1.5–2.9) Other Multiracial
[176]	2006	NHS [80]	PCS	78419 (4.90%)	0/100	30–55	White, Asian, Hispanic, and Black	20	Age, BMI, family history of diabetes, alcohol consumption, physical exercise, and smoking	Ethnicity RR White 1.0 Asian 1.94 (1.46–2.58) Hispanic 1.70(1.28–2.26) Black 1.36(1.14–1.63)

RS-Random Sample, BRFSS-Behavioral Risk Factor Surveillance System, NHS-Nurses' Health Study, PCS-Prospective Cohort Study.

Table 9
Characteristics and findings of the studies examining the association between family history of diabetes and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[204]	1981	RS	-	3177	-	>5	Pima Indians	-	Age and BMI	Family History	OR
										No	1.0
										Mother/father both	2.3
											3.9
[195]	1993	SAHS	-	4914	43/ 57	Mean 42–44.8	Mexicans, Americans and Non-Hispanics	9	Age and ethnicity	Family History	OR (Men)
										No	1.0
										Mother	3.44 (2.32–5.12)
										father	3.49 (2.16–5.64)
										both	3.73 (1.72–8.08)
										Family History	OR (Women)
										No	1.0
										Mother	2.03 (1.47–2.81)
										father	1.35 (0.83–2.19)
										both	2.59 (1.41–4.77)
[200]	1993	MRFIT [40–42]	-	5905	100/0	-	Blacks and Whites (USA)	6	Age	Family History	RR (Black)
										No	1.0
										Mother/father	3.62 (1.55–8.47)
										Family History	RR (White)
										No	1.0
										Mother/father	1.85 (1.38–2.48)
[205]	1994	MA	PCS	11334	M/W	⩾40	Taiwan	-	-	Family History Age at onset	OR
										40–49	1.0
										No	4.41 (1.71–10.13)
										Mother	2.21 (0.25–8.86)
										father	RR
										Family History	1.0 1.57 (0.40–
										Age at onset 50–59	4.41)
										No	2.80 (0.54–9.07)
										Mother	RR
										father	1.0
										Family History	1.22 (0.38–3.05)
										Age at onset ⩾60	0.56 (0.01–3.31)
										No	
										Mother	
										father	
[194]	1995	THHP	-	7210 (12.81%)	100/0	45–68	Japanese-American	6	Age, BMI, subscapular skinfold, triceps ratio, physical activity, glucose, triglycerids, and systolic blood pressure	Family History	OR
										No	1.0
										yes	1.73 (1.29–2.33)
[201]	2000	RS	PCS	1947 (7.34%)	100/0	Mean 49.5–50.3	Norway	22.5	Age, BMI, fasting glucose, fitness and triglycerids	Family History	OR
										No	1.0
										Mother	2.51 (1.55–4.07)
										Father	1.41 (0.657–3.05)
										Both	3.96 (1.22–12.9)

(*continued on next page*)

Table 9 (*continued*)

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[198]	2000	EPIC [101]	CSS	6473	45.54/ 54.46	45–74	USA	22.5	Age and sex	Family History	OR
										No	1.0
										BMI 22.5–24.9	2.0 (1.2–3.1)
										BMI 27.5–29.9	2.5 (1.6–4.0)
										BMI 30–34.9	6.1 (3.4–11.2)
										BMI ⩾35	1.1 (0.2–5.1)
										Yes	2.6 (1.3–5.3)
										BMI ⩽22.4	
										BMI 22.5–24.9	2.8 (1.5–5.3)
										BMI 25–27.4	2.2(1.1–4.6)
										BMI 27.5–29.9	6.4 (3.6–11.3)
										BMI 30–34.9	26.7 (14.4–49.4)
										BMI ⩾35	
[202]	2000	FHS [187]	-	2527	M/W	26–82	African-American and White (USA)	40	Age	Family History	OR
										No	1.0
										Mother	3.4 (2.3–4.9)
										Father	3.5 (2.3–5.2)
										Both	6.1 (2.9–13.0)
[203]	2001	MONICA [35]	CSS	12751	49.6/ 50.4	-	Germany	-	Age and sex	Family History	OR
										No	1.0
										Mother	2.9 (2.3–3.6)
										Father	2.8 (2.1–3.8)
[197]	2007	NHANES [88]	-	16388	49.3/ 50.7	⩾18	USA	6	sex, race/ethnicity, age, BMI, hypertension, and household income	Family History	OR
										Average risk	1.0
										Moderate risk	2.3
										High risk	5.5
[199]	2009	NHANES [88]	CSS	10899	48/ 52	Mean 51.3–61	Blacks, Whites and Hispanics	5	Age and sex	Black	OR
										Average risk, BMI⩽24.9	1.0
										High risk, BMI⩽24.9	20.4 (6.5–64.5)
										High risk, 25⩽BMI⩽29.9	5.2 (2.2–12.3)
										High risk, BMI⩾30	5.0 (2.5–10.3)
										Hispanic	1.0
										Average risk, BMI⩽24.9	14.0 (3.4–58.0)
										High risk, BMI⩽24.9	5.6 (1.8–17.3)
										High risk, 25⩽BMI⩽29.9	8.5 (3.8–19.4)
										High risk, BMI⩾30	
[196]	2011	RS	CSS	3723	49.1/ 50.9	7–15	Mexican	2	Age, sex, and BMI	Family History	OR
										No vYes	1.0
											11.7 (9.5–21.2)
[193]	2016	MIDUS 1 and 2 [212]	-	978	45/ 55	34–84	Black and White (USA)	-	Age, sex, and socioeconomic status	Family History	OR
										No	1.0
										Yes	2.77 (2.03–3.78)

RS-Random Sample, SAHS-San Antonio Heart Study, MRFIT-Multiple Risk Factor Intervention Trial, MA-Meta Analysis, THHP-The Honolulu Heart Program, EPIC-European Prospective Investigation into Cancer, FHS-Framingham Heart Study, MONICA-Multinational MONItoring of trends and determinants in CArdiovascular disease, NHANES-National Health and Nutrition Examination Survey, PD-Prediabetes, IFG-Impaired Fasting Glucose, IGT-Impaired Glucose Tolerance, M-Men, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

Table 10
Characteristics and findings of the studies examining the association between obesity and type 2 diabetes.

Work	Year	Study	Design	Sample size (%DM)	%M/W	Age (Years)	Ethnicity	Follow-up duration (Years)	Adjusted variables	Findings	
[206]	2002	-	PCS	4737	100/0	45–64	Japanese	4	Age, smoking status, alcohol intake, family history, and baseline value of fasting blood glucose.	BMI (kg/m^2) ⩽18.49 ⩽29 ⩽30 and <35	RR 1.0 5.16 (1.92–13.80) 5.25 (1.96–14.04)
[213]	2007	BWHS [214]	-	49766 (4.96%)	0/100	21–69	African-American (USA)	8	Age, physical activity, family history of diabetes, cigarette smoking, years of education, and time period of data collection	BMI (kg/m^2) <23 ⩾45	IRR 1.0 23 (17–31)
[119]	2001	-	PCS	84941 (3.88%)	0/100	30–55	-	16	Age (in five-year categories), time (eight periods), presence or absence of a family history of diabetes, menopausal status, and use or nonuse of postmenopausal hormone therapy	BMI (kg/m^2) <23 23–24.9 25–29.9 30–34.9 ⩾35	RR 1.0 2.67 (2.13–3.34) 7.59 (6.27–9.19) 20.1 (16.6–24.4) 38.8 (31.9–47.2)
[207]	2006	27 cohorts	PCS + CSS	154989 (0.20%)	54/ 46	Mean 51	-	Mean 8	Age, sex, cohort, and smoking habit	5.3 cmEach 2 kg/m^2 lower BMI is associated with a 23% (15–30%) lower risk of total DM in men and 27% (23–31%) lower risk in women. In the Asian cohort, each 2 kg/m^2 lower BMI was associated with a 37% (26–46%) lower risk and in Australasian cohorts the same reduction in BMI was associated with 25% (21–29%) lower risk.	
[208]	2006	RS	-	827 (7.86%)	-	-	Japanese	10	Age, sex, total cholesterol, systolic pressure, smoking and overall obesity	WC (cm) ⩾85 (M) ⩾90 (W)	RR 2.07 (1.03–4.16)
[209]	2006	TLGS	PCS	4479 (3.70%)	41.34/ 58.66	>3	Tehran	3.6 (mean)	Age, smoking, family history of diabetes, HTN, TG, HDL and other anthropometric variables	5.3 cmCentral obesity is defined as WC ⩾102 cm in men and WC ⩾88 cm in women. The central obese individuals ⩾60 years old are at higher risk of incidence type 2 diabetes (OR 3.8, 95% CI 1.8–7.7).	
[210]	2009	RS	-	5071	37.80/ 62.2	⩾40	Chinese	-	Educational level, age group, smoking and alcohol drinking	WC (cm) <90(M) ⩾90(M) <80(W) ⩾80(W)	OR 1.0 2.308 (1.473–3.615) 1.0 2.875(1.987–4.160)
[211]	2001	MAHES	-	835	39.16/ 60.84	60–92	Hispanics and Non-Hispanics	-	Age, physical activity and smoking	WC (cm) >102(M)(H) >102(M)(NH) >88(W)(H) >88(W)(NH)	OR 2.1(1.2–3.9) 0.9 (0.3–3.1) 1.6 (1.0–2.8) 15.1(1.9–117.6)

SWHS-Shanghai Women's Health Study, BWHS- Black Women's Health Study, RS-Random Sample, WC-Waist Circumference, TLGS-Tehran Lipid and Glucose Study, MAHES-Massachusetts Hispanic Elderly Study, H-Hispanics, NH-Non Hispanics, M-Men, W-Women, PCS-Prospective Cohort Study, CSS-Cross-Sectional Study.

2 kg/m^2 lower BMI, men are at 23% (15–30%) lower risk of diabetes, whereas women are at 27% (23–32%) lower risk. Further, the association between obesity and diabetes is also dependent on ethnicity [207]. For each 2 kg/m^2 lower BMI, Asians are at 37% (26–46%) lower risk of diabetes, whereas Australians are at 25% (21–29%) lower risk.

Ohnishi et al. [208] found that compared to overall obesity, central obesity is highly associated with the risk of type 2 diabetes (RR 2.07, 95% CI 1.03–4.16). This association is more in elderly people ($\geqslant$60 years) (OR 3.8, 95% CI 1.8–7.7) [209]. The association between central obesity and the incidence of type 2 diabetes is found significant in both men and women. However, centrally obese women are at higher risk (OR 2.875, 95% CI 1.987–4.160) compared to centrally obese men (OR 2.308, 95% CI 1.473–3.615) [210]. The prevalence of type 2 diabetes in obese individual is ethnicity dependent [211]. Non-Hispanics centrally obese women are at higher risk of developing type 2 diabetes (OR 15.1, 95% CI 1.9–117.6) compared to centrally obese Hispanic women (OR 1.6, 95% CI 1.0–2.8). The centrally Hispanic men are also at risk of developing type 2 diabetes (OR 2.1, 95% CI 1.2–3.9). No such association is found in centrally obese Non-Hispanic men. However, all these studies examining the association between central obesity and the incidence of type 2 diabetes consider different definitions of central obesity. For instance, [208] defines central obesity as waist circumference (WC) $\geqslant$85 cm in men and $\geqslant$90 cm in women, whereas [211] defines it as WC$>$102 cm in men and $>$88 cm in women. Consequently, it is difficult to conclude the association between central obesity and the incidence of type 2 diabetes.

In summary, although obesity is a significant predictor, the association between obesity and diabetes is a factor of gender and ethnicity. Women with high BMI are at greater risk of diabetes compared to men. Moreover, the association is stronger in Asians compared to Australians. The association between central obesity is also found to be significant for the prevalence of type 2 diabetes. This association is the strongest in Non-Hispanics women. However, more studies are required to examine the association between central obesity and type 2 diabetes following one standard criterion defining central obesity.

3.12. Physical inactivity

An individual is considered physically inactive if he/she does not get the recommended 30–60 min of exercise three to four times a week. Physical inactivity decreases insulin sensitivity with progressive loss of beta-cells. This leads to impaired glucose tolerance and eventually type 2 diabetes. However, no work examines the association between physical inactivity as an independent factor and the prevalence of diabetes. One of the reasons that physical inactivity leads to type 2 diabetes can be that physical inactivity can cause obesity which in turn is a significant risk factor for type 2 diabetes.

4. Conclusion

Diabetes is a global crisis that is primarily driven by rapid urbanization, changing lifestyles, and uneven dietary patterns [215,216]. It is crucial to predict the prevalence of diabetes in an individual to reduce the risk of diabetes development and save lives. Diabetes is thought to prevail due to several risk factors such as high-level serum uric acid, sleep quality/quantity, smoking, depression, cardiovascular disease, dyslipidemia, hypertension, aging, ethnicity, family history of diabetes, physical inactivity, and obesity. Studies in the literature have examined the association between each of these risk factors and the risk of developing type 2 diabetes. In this review, we provide an analysis of the studies in the literature to deduce inferences on the relationship between the risk factors and incidence/prevalence of type 2 diabetes.

In conclusion, it can be observed that sleep quantity/quality, smoking, dyslipidemia, hypertension, ethnicity, family history of diabetes, obesity and physical inactivity are strongly associated with the development of type 2 diabetes. Both sleep quantity and quality are found to be strongly associated with the development of type 2 diabetes. The association is stronger in women sleeping for more hours and in men sleeping for fewer hours. However, the sleeping quantity and quality data in these studies are self-reported by the participants, and therefore, prone to errors. More studies are required that use measurement techniques for data collection to validate the association between sleep quantity/quality and type 2 diabetes. Smoking is also found to be a significant risk factor for type 2 diabetes. Both active and passive smokers are at higher risk of developing type 2 diabetes. Moreover, the risk for developing type 2 diabetes remains high in ex-smokers for the first 5–10 years of smoking cessation. Dyslipidemia is associated with the development of type 2 diabetes. Increased non-HDL and decreased HDL levels are strongly associated with type 2 diabetes. However, in the majority of these studies, the incidence or prevalence of type 2 diabetes is self-reported. Consequently, further studies are needed to validate this association between dyslipidemia and type 2 diabetes using standardized measurement techniques, such as A1C test [217]. Hypertension is a significant risk factor for type 2 diabetes and this is further elevated in obese individuals. Ethnicity strongly associates with the development of type 2 diabetes. This could be due to the fact that insulin sensitivity varies among individuals of different ethnicity. Family history of diabetes in first degree of relatives is strongly associated with the development of type 2 diabetes. In addition, family history of diabetes also signifies the association between obesity and type 2 diabetes. Obesity is found to a significant risk factor for incidence of type 2 diabetes and the association is stronger in women compared to men.

The association between serum uric acid and type 2 diabetes remains obscure. It can not be concluded that serum uric acid is an independent risk factor for type 2 diabetes or it only elevates the association between other independent risk factors such as obesity, hypertension, and dyslipidemia, and type 2 diabetes. Moreover, our analysis shows that there might be no association between serum uric acid and the development of type 2 diabetes, but rather there might be a reverse association, i.e., diabetes leads to elevated serum uric acid level. Similarly, based on the evidence in the literature, aging can not be considered as an independent risk factor for type 2 diabetes. Aging only emphasizes the association between obesity and type 2 diabetes. Depression as well is not found to an independent risk factor contributing to the development of type 2 diabetes. Rather, the activities related to depression such as physical inactivity, poor diet, and obesity leads to diabetes. There is no sufficient evidence to conclude the association between cardiovascular disease and type 2 diabetes. It is debatable whether cardiovascular disease leads to the development of type 2 diabetes. Consequently, more studies are required to study the direct association between these risk factors, i.e., serum uric acid, aging, depression, and cardiovascular disease, and incidence of type 2 diabetes.

Based on this study, we devise recommendations to different stakeholders leading to better patient care. In particular, we provide recommendations for allied healthcare professionals, individuals, and government institutions as follows:

- **Allied healthcare professionals:**The hypertensive medications and the LDL lowering therapy and drugs should be carefully prescribed as they are associated with increased risk of type 2 diabetes. In addition, overweight and obese adults should be screened for diabetes.
- **Individuals:**A healthy lifestyle, which involves intake of polyunsaturated fatty acids and vegetable fats, regular exercise, a healthy diet and proper sleep, is crucial. Individuals should avoid both active and passive smoking.
- **Government:**Physical activity in the nation should be promoted for a healthy nation. Law policies should be implemented to restrict public smoking as passive smoking significantly increases the risk of type 2 diabetes. For instance, designated smoking areas can be established to eliminate the risk of developing passive smokers. It would be beneficial to have periodic surveys that include the demographic and lifestyle features of the citizens and the surveys' results can be then used to develop a nation-wide diabetes prevention plan, in coordination with the allied health professionals.

CRediT authorship contribution statement

Leila Ismail: Conceptualization, Methodology, Investigation, Writing - original draft, Writing - review & editing. **Huned Materwala:** Investigation, Writing - original draft. **Juma Al Kaabi:** Validation, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A

Table A1
Search string used to retrieve the studies on the association between risk factor and type 2 diabetes.

Risk factor	Search string
Serum uric acid	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("uric acid" OR uric-acid OR hyperuricemia OR "serum uric acid" OR gout) AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Sleep quantity/quality	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("sleep hour" OR "sleeping hour" OR "hours of sleep" OR "sleep duration" OR "sleep time" OR "sleep length" OR "sleep period" OR "sleeping time" OR "sleep span" OR nap OR napping OR "daytime sleep" OR vsleep quality" OR "sleep disturbance" OR "sleep apnea" OR insomnia OR "sleep deprivation") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Smoking	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND (smoking OR "smoking cessation" OR cigarette OR "cigarette smoking" OR "passive smoking" OR "secondhand tobacco smoke") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Depression	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("depressive disorder" OR depression OR "dysthymic disorders") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Cardiovascular disease	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("cardiovascular disease" OR stroke OR "heart disease") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
dyslipidemia	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND (cholesterol OR "cholesterol intake" OR "cholesterol consumption" OR diet* OR fat OR "density lipoprotein" OR density-lipoprotein OR dyslipidemia) AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Hypertension	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("high blood pressure" OR "blood pressure" OR hypertensi* OR "Hypertension-*") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Aging	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND (age OR aging OR old OR elderly) AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Ethnicity	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND (ethnicity OR race OR *rac* OR community) AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Family history of diabetes	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("family history" OR "parental history" OR "parental diabetes" OR "parental transmission" OR paternal OR maternal) AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Physical inactivity	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("physical inactivity") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")
Obesity	(risk OR "risk factor" OR etiology OR association OR development OR progression OR incidence) AND ("body mass index" OR BMI OR "body fat distribution" OR "over weight" OR overweight OR obesity OR "weight change" OR "weight gain" OR "central obesity") AND (diabetes OR "diabetes mellitus" OR "type 2 diabetes" OR "type II diabetes" OR "non-insulin dependent diabetes" OR "non insulin dependent diabetes" OR "noninsulun dependent diabetes")

Table A2
Quality assessment of the included studies according to the Quality assessment tool for observational cohort and cross-sectional studies.

Work	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Quality
High-level serum uric acid															
[14]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes*	NR	NR	Yes	Yes	Good
[21]	Yes	Yes	NR	Yes	No	CD	No	No	NR	No	CD	NR	No	Yes	Poor
[22]	Yes	Yes	Yes	CD	No	Yes	Yes	CD	Yes	Yes	No	NR	No	Yes	Fair
[23]	No	Yes	NR	Yes	No	Yes	Yes	CD	Yes	Yes	Yes	NR	CD	Yes	Fair
[19]	No	No	NR	NR	No	Yes	CD	No	NR	Yes	NR	NR	NR	No	Poor
[15]	No	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	NR	NR	Yes	Good
[24]	No	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	NR	NR	Yes	Good
[16]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NR	Yes	NR	NR	NR	Yes	Fair
[25]	Yes	Yes	NR	No	No	Yes	Yes	No	Yes	Yes	Yes	NR	NR	Yes	Fair
[26]	Yes	Yes	NR	Yes	No	Yes	Yes	No	Yes	Yes	Yes	NR	Yes	Yes	Fair
[27]	Yes	No	NR	CD	No	Yes	Yes	No	Yes	Yes	Yes	NR	NR	Yes	Fair
[30]	No	Yes	NR	Yes	No	No	No	Yes	Yes	NR	Yes	NR	NR	Yes	Fair
Sleep quantity/quality															
[79]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	Yes	Yes	NR	NR	Yes	Fair
[56]	No	No	NR	Yes	No	Yes	Yes	NA	No	Yes	Yes	NR	Yes	Yes	Fair
[57]	Yes	Yes	NR	Yes	No	Yes	Yes	NA	No	No	No	NR	NR	Yes	Fair
[59]	Yes	Yes	Yes	Yes	No	Yes	Yes	NA	No	No	Yes	NR	NR	Yes	Good
[83]	No	Yes	NR	Yes	No	No	No	Yes	No	NR	Yes	NR	NR	Yes	Fair
[52]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	No	No	No	NR	Yes	Yes	Fair
[50]	Yes	Yes	Yes	Yes	No	Yes	Yes	No	No	Yes	Yes	NR	No	Yes	Fair
[85]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	Yes	NR	NR	No	Yes	Fair
[87]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	Yes	No	NR	Yes	Yes	Fair
[89]	Yes	Yes	NR	Yes	No	No	No	Yes	No	No	Yes	NR	NR	Yes	Fair
[58]	Yes	Yes	No	Yes	No	Yes	Yes	Yes	No	CD	Yes	NR	NR	Yes	Fair
[92]	Yes	Yes	NR	Yes	No	No	No	Yes	No	NR	Yes	NR	NR	Yes	Fair
[94]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	NR	Yes	NR	NR	Yes	Fair
[55]	Yes	Yes	No	Yes	No	Yes	Yes	Yes	No	No	Yes	NR	Yes	Yes	Fair
[96]	CD	Yes	Yes	No	No	No	No	Yes	No	NR	Yes	NR	CD	Yes	Fair
[97]	Yes	No	No	Yes	No	Yes	Yes	Yes	No	NR	Yes	NR	NR	Yes	Fair
[98]	Yes	Yes	CD	Yes	No	Yes	Yes	Yes	No	No	No	NR	NR	Yes	Fair
[100]	Yes	Yes	NR	CD	No	No	No	Yes	No	NR	Yes	NR	NR	Yes	Fair
[51]	No	Yes	CD	Yes	No	Yes	No	Yes	No	Yes	No	NR	Yes	Yes	Fair
[102]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	NR	No	NR	Yes	Yes	Fair
[53]	Yes	Yes	Yes	No	No	No	No	Yes	No	NR	No	NR	NR	Yes	Fair
[104]	Yes	Yes	NR	CD	No	No	No	Yes	No	NR	Yes	NR	NR	Yes	Fair
[106]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	NR	No	Yes	Good
[54]	Yes	Yes	Yes	No	No	No	No	Yes	No	Yes	No	NR	NR	Yes	Fair
[108]	Yes	Yes	No	Yes	No	Yes	NR	No	No	NR	No	NR	NR	Yes	Poor
Smoking															
[110]	No	Yes	NR	Yes	No	Yes	Yes	No	NA	Yes	No	NR	NR	Yes	Fair
[67]	No	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	Yes	NR	Yes	Good
[68]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	No	Yes	Yes	Good
[112]	No	No	Yes	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[73]	CD	Yes	NR	Yes	No	No	No	Yes	NA	NR	No	NR	NR	Yes	Poor
[114]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[69]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	No	Yes	NR	Yes	Good
[116]	No	Yes	NR	Yes	No	No	No	No	NA	NR	Yes	NR	NR	Yes	Poor
[65]	Yes	Yes	NR	Yes	No	Yes	Yes	No	NA	Yes	Yes	NR	Yes	Yes	Fair
[63]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	NR	CD	NR	NR	Yes	Fair
[119]	No	Yes	No	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[120]	No	CD	NR	CD	No	Yes	Yes	Yes	NA	Yes	NR	NR	NR	Yes	Fair
[74]	Yes	Yes	Yes	CD	No	No	No	No	NA	No	Yes	NR	NR	Yes	Fair
[122]	Yes	Yes	Yes	Yes	No	Yes	Yes	No	NA	No	Yes	No	No	Yes	Good
[70]	Yes	Yes	No	Yes	No	Yes	Yes	No	NA	No	Yes	NR	NR	Yes	Fair
[77]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	CD	Yes	Good
[71]	Yes	CD	Yes	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	NR	Yes	Good
[75]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	NR	Yes	Good
[66]	No	Yes	Yes	Yes	No	Yes	Yes	No	NA	No	Yes	NR	No	Yes	Good
[64]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	CD	NR	NR	Yes	Fair
[72]	Yes	No	NR	CD	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[78]	Yes	Yes	Yes	Yes	No	Yes	Yes	No	NA	Yes	Yes	NR	Yes	Yes	Good
[76]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[128]	No	Yes	NR	Yes	No	Yes	Yes	No	NA	NR	No	NR	NR	Yes	Fair
Depression															
[137]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	No	Yes	NR	Yes	Yes	Good
[155]	No	Yes	NR	Yes	No	Yes	Yes	No	Yes	No	No	NR	NR	Yes	Fair
[157]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	NR	No	NR	NR	Yes	Fair
[158]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	NR	Yes	NR	NR	Yes	Fair
[136]	Yes	Yes	NR	Yes	No	Yes	CD	No	Yes	Yes	Yes	NR	CD	Yes	Fair
[160]	Yes	Yes	Yes	Yes	No	Yes	Yes	No	Yes	Yes	Yes	NR	NR	Yes	Good
[138]	No	Yes	NR	Yes	No	Yes	Yes	No	Yes	No	CD	NR	NR	Yes	Fair
[139]	No	Yes	NR	Yes	No	Yes	Yes	No	Yes	No	Yes	NR	NR	Yes	Fair

Table A2 (*continued*)

Work	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Quality
Cardiovascular disease															
[141]	No	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	NR	NR	Yes	Good
Dyslipidemia															
[148]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	CD	CD	No	NR	No	Yes	Fair
[154]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	NR	NR	NR	NR	Yes	Yes	Fair
[152]	No	Yes	NR	Yes	No	Yes	CD	Yes	Yes	No	Yes	NR	Yes	Yes	Fair
Hypertension															
[166]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	NR	Yes	NR	Yes	Yes	Good
[167]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	No	Yes	NR	NR	Yes	Good
[168]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	No	No	NR	CD	Yes	Fair
[170]	Yes	Yes	NR	No	No	Yes	Yes	Yes	Yes	No	Yes	NR	NR	Yes	Good
[188]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	No	Yes	NR	CD	Yes	Good
[169]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	No	Yes	NR	NR	Yes	Good
Ethnicity															
[177]	No	Yes	Yes	No	No	No	No	NA	NA	NR	Yes	NR	NR	Yes	Fair
[179]	No	Yes	Yes	No	No	No	No	NA	NA	NR	NR	NR	NR	Yes	Poor
[180]	No	Yes	Yes	No	No	No	No	NA	NA	No	Yes	NR	NR	Yes	Fair
[181]	Yes	Yes	Yes	CD	No	No	No	NA	NA	No	Yes	NR	NR	Yes	Fair
[175]	Yes	Yes	Yes	Yes	No	No	No	NA	NA	No	No	NR	NR	Yes	Poor
[176]	Yes	Yes	NR	Yes	No	Yes	Yes	NA	NA	Yes	Yes	NR	NR	Yes	Good
Family history of diabetes															
[204]	No	No	NR	CD	No	Yes	NR	No	Yes	NA	Yes	NR	NR	Yes	Fair
[195]	Yes	Yes	Yes	Yes	No	No	No	Yes	No	NA	Yes	NR	NR	Yes	Fair
[200]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	No	NA	Yes	NR	NR	Yes	Fair
[205]	No	Yes	NR	No	No	Yes	CD	Yes	NR	NA	NR	NR	NR	No	Poor
[194]	No	Yes	NR	Yes	No	Yes	Yes	No	NR	NA	Yes	NR	NR	Yes	Fair
[201]	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	CD	NA	Yes	NR	Yes	Yes	Good
[198]	Yes	Yes	NR	Yes	No	No	No	No	No	NA	Yes	NR	NR	Yes	Poor
[202]	No	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	NA	Yes	NR	NR	Yes	Good
[203]	Yes	Yes	Yes	No	No	No	No	Yes	No	NA	No	NR	NR	Yes	Fair
[197]	Yes	Yes	NR	Yes	No	No	No	Yes	No	NA	Yes	NR	NR	Yes	Fair
[199]	Yes	Yes	No	No	No	No	No	Yes	Yes	NA	Yes	NR	NR	Yes	Fair
[196]	Yes	Yes	Yes	Yes	No	No	No	No	Yes	NA	Yes	NR	NR	Yes	Fair
[193]	Yes	Yes	Yes	Yes	No	CD	NR	No	No	NA	Yes	NR	NR	Yes	Fair
Obesity															
[206]	Yes	Yes	NR	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	NR	NR	Yes	Good
[213]	Yes	Yes	NR	Yes	No	Yes	Yes	No	Yes	Yes	Yes	NR	Yes	Yes	Fair
[119]	No	Yes	No	Yes	No	Yes	Yes	Yes	NA	Yes	Yes	NR	Yes	Yes	Good
[207]	No	No	NR	CD	No	Yes	Yes	Yes	NR	NR	Yes	NR	NR	Yes	Fair
[208]	No	Yes	NR	Yes	No	Yes	Yes	No	Yes	NR	Yes	NR	NR	Yes	Fair
[209]	Yes	Yes	Yes	Yes	No	Yes	Yes	No	Yes	NR	Yes	NR	No	Yes	Good
[210]	Yes	Yes	Yes	Yes	No	CD	CD	No	Yes	NR	Yes	NR	NR	Yes	Fair
[211]	Yes	Yes	NR	Yes	No	Yes	CD	No	Yes	NR	Yes	NR	NR	Yes	Fair

Q1. Was the research question or objective in this paper clearly stated?.
Q2. Was the study population clearly specified and defined?.
Q3. Was the participation rate of eligible persons at least 50%?
Q4. Were all the subjects selected or recruited from the same or similar populations (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?.
Q5. Was a sample size justification, power description, or variance and effect estimates provided?.
Q6. For the analyses in this paper, were the exposure(s) of interest measured prior to the outcome(s) being measured?.
Q7. Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?.
Q8. For exposures that can vary in amount or level, did the study examine different levels of the exposure as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)?.
Q9. Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?.
Q10. Was the exposure(s) assessed more than once over time?.
Q11. Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?.
Q12. Were the outcome assessors blinded to the exposure status of participants?.
Q13. Was loss to follow-up after baseline 20% or less?.
Q14. Were key potential confounding variables measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)?.
CD-Cannot be Determined; NA-Not Applicable; NR-Not Reported.

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