

Type 2 diabetes incidence and socio-economic position: a systematic review and meta-analysis

Emilie Agardh,^{1*} Peter Allebeck,¹ Johan Hallqvist,^{2,3} Tahereh Moradi⁴ and Anna Sidorchuk^{1,5}

¹Department of Public Health Sciences, Division of Social Medicine, Karolinska Institutet, Stockholm, Sweden, ²Department of Public Health and Caring Sciences, Uppsala University, Uppsala, Sweden, ³Department of Public Health Sciences, Division of Public Health Epidemiology, Karolinska Institutet, Stockholm, Sweden, ⁴Department of Environmental Medicine, Division of Epidemiology, Karolinska Institutet, Stockholm, Sweden and ⁵St Petersburg State Medical Academy named after II. Mechnikov, Division of Epidemiology, St Petersburg, Russia

*Corresponding author. Department of Public Health Sciences, Division of Social Medicine, Karolinska Institutet, Norrbacka Plan 6, Karolinska Hospital, SE-171 76 Stockholm, Sweden. E-mail: emilie.agardh@ki.se

Accepted 24 January 2011

Background We conducted a systematic review and meta-analysis, the first to our knowledge, summarizing and quantifying the published evidence on associations between type 2 diabetes incidence and socio-economic position (SEP) (measured by educational level, occupation and income) worldwide and when sub-divided into high-, middle- and low-income countries.

Methods Relevant case-control and cohort studies published between 1966 and January 2010 were searched in PubMed and EMBASE using the keywords: diabetes vs educational level, occupation or income. All identified citations were screened by one author, and two authors independently evaluated and extracted data from relevant publications. Risk estimates from individual studies were pooled using random-effects models quantifying the associations.

Results Out of 5120 citations, 23 studies, including 41 measures of association, were found to be relevant. Compared with high educational level, occupation and income, low levels of these determinants were associated with an overall increased risk of type 2 diabetes; [relative risk (RR) = 1.41, 95% confidence interval (CI): 1.28–1.51], (RR = 1.31, 95% CI: 1.09–1.57) and (RR = 1.40, 95% CI: 1.04–1.88), respectively. The increased risks were independent of the income levels of countries, although based on limited data in middle- and low-income countries.

Conclusions The risk of getting type 2 diabetes was associated with low SEP in high-, middle- and low-income countries and overall. The strength of the associations was consistent in high-income countries, whereas there is a strong need for further investigation in middle- and low-income countries.

Keywords Type 2 diabetes incidence, socio-economic position, educational level, occupation, income

Introduction

Diabetes is a health-threatening concomitant disease all over the globe. The world prevalence of diabetes has been estimated to rise from today's 220 million to 300 million by the year 2025,^{1,2} resulting in increasing number of subjects with severe complications in the cardiovascular system, kidneys, eyes and peripheral nerves. As the prevalence rises there is an urge to characterize determinants beyond traditional risk factors, e.g. sedentary behaviour and obesity.

Socio-economic position (SEP) is one example of such a determinant. Although the causal pathways between SEP and disease are not yet fully understood, SEP may contribute to the development of type 2 diabetes through complex processes involving access to health-care services and information, available healthy foods and places to exercise, economic and occupational opportunities as well as individual lifestyle choices.³ In high-income countries, type 2 diabetes is more prevalent in lower socio-economic groups,^{4–10} whereas the opposite has been found in studies from middle- and low-income countries.^{11–15} Sedentary behaviour and obesity are risk factors that are suggested to be responsible for these various scenarios to a large extent.¹⁶ Hence, in high-income countries people in lower SEP groups are more sedentary and obese,^{4,17,18} whereas the reverse has been reported in countries undergoing rapid economic development.^{11–15}

For the prevention of type 2 diabetes, it is crucial to investigate how the disease is patterned by SEP. A first step is to summarize the nature and strength of this association. To avoid problems with reverse causality and unequal survival, use of incidence data is a prerequisite. To our knowledge, no systematic review of type 2 diabetes incidence and SEP has been undertaken previously. We therefore conducted a systematic quantitative review to summarize the nature and strength of the overall association between type 2 diabetes incidence and SEP (measured by educational level, occupation and income) as well as by income level of country.

Methods

Data sources

We followed recommended guidelines on how to conduct systematic reviews and meta-analysis.^{19,20} To identify eligible articles published in English-speaking peer-reviewed journals, we searched PubMed and EMBASE (1966 to 11 January 2010), using the key words 'diabetes vs educational level', 'occupation or income'. The reference lists of included articles and relevant review articles were manually reviewed for potential inclusion of additional studies.

Study selection

We included studies that (i) presented original data using case-control or cohort study design; (ii) provided information on type 2 diabetes incidence as an outcome; (iii) presented risk estimates with confidence intervals (CIs) or sufficient information to calculate these; and (iv) used educational level, occupation or income as individual measures of SEP.

All citations identified by the initial search (5120 articles) were systematically screened and evaluated by one author (E.A.) to exclude publications clearly irrelevant to the inclusion criteria. For publications retrieved for detailed examination (94 articles), two co-authors (E.A. and A.S.) independently filled in standardized forms with predefined inclusion and exclusion criteria. If there was any disagreement, a third co-author (P.A.) independently filled in the form and consensus was reached. In addition, all authors (E.A., P.A., J.H., T.M. and A.S.) participated in a panel discussion regarding additional issues that came up during the detailed examination. At this stage, agreement was made to exclude studies that did not explicitly exclude subjects with prevalent type 2 diabetes at baseline, used less than four cases in the exposed or unexposed group,²¹ used parental- or area-level SEP, measured SEP by means or used subgroups within only one SEP group, for example subgroups within white-collar workers, or military ranking as a measure of SEP. To reduce overlapping, we included original studies used in multiple publications only once, giving preference to studies with largest sample size or most recent publication.

Data extraction

Two authors (E.A. and A.S.) independently extracted the following data from each publication: author, publication year, years of data collection, ages, sex (men, women or combined), country, ethnicity of study population, study design (cohort or case-control), measure of exposure (educational level, occupation or income), type of SEP (own, husband's or household), total number of SEP categories reported in the original studies, type of controls (population-based or hospital-based), duration of follow-up, sample size, risk estimate with CIs, variables controlled for, participation rate and method of type 2 diabetes assessment.

Countries were classified according to geographical area (USA, Europe, Asia/Middle East, Latin America or Africa) and income category according to the World Bank definition (high-income, upper-middle-income or low-income country). We did not identify any studies from lower-middle-income countries. Nested case-control and case-cohort studies were referred to as case-control studies since they all used odds ratios as measure of association. The method of assessing type 2 diabetes was either by self-report, self-report verified (by for example inspection of drug packages, medical records and blood

glucose), blood glucose by screening, medical records or registers. We extracted estimates for men and women separately when possible. If a study reported risk estimates with more than one measure of SEP, each estimate was treated as its own association.

Our main data set consisted of one risk estimate from each study. When a study reported more than one risk estimate we included the most adjusted estimate. Hence, the number of controlled variables could vary from none to more than 10 in this data set. For the purpose of sub-group and sensitivity analysis we also extracted information on both minimally adjusted (crude or adjusted for sex, age and residence) and maximally adjusted (adjusted for well-established outcome-related risk factors) risk estimates from each study when available. Information on the included studies is shown in Table 1.

Statistical analysis

We used relative risks (RRs) as summary estimates throughout the procedure to simplify reporting.²² The included studies used scales ranging from two to five categories, when measuring SEP. To ease comparability between studies, we extracted the lowest vs the highest SEP category from each study, using high SEP as the reference group. If presented in a reverse order, we back-calculated the point estimate and 95% CIs. When articles did not present risk estimates, we calculated them from the raw data presented in the article. The log RRs from the individual studies and corresponding standard error (SE; presented or calculated from the confidence limits) were used to perform the analysis. The pooled estimates were then converted back to RRs and 95% CIs for presentation. We quantified the relation between type 2 diabetes incidence and SEP by using a random-effects model of DerSimonian and Laird,²³ which incorporates both within- and between-study variability, based on the initial assumptions of between-study heterogeneity.

Statistical heterogeneity among studies was evaluated using both the Q -statistic and the I^2 -statistics. The Q -statistic is the test which examines the null hypothesis if differences between the study estimates of RRs is due to chance, by a chi-square test with degrees of freedom equal to the number of studies minus one.²⁴ For Q -statistic, we considered $P < 0.1$ as representative of statistically significant heterogeneity. The I^2 is the proportion of total variance in study estimates explained by heterogeneity between study variation rather than chance.²⁵

We performed a random-effect meta-regression analysis to address the issue of heterogeneity and to identify potential study-level factors contributing to heterogeneity between studies. Study characteristics such as sex, income category of countries, geographical area, minimally or maximally adjusted, publication year, study design, length of follow-up in cohort studies, assessment of cases, number of SEP categories and personal, husband's or household SEP

were used as explanatory variables and the natural logarithm of RR was the dependent variable.^{26–28} A univariate meta-regression was performed for each SEP indicator followed by a multivariate analysis for which a backward stepwise approach was used to select the significant variables to be included.

We then conducted subgroup analyses by stratifying the original data sets by the above mentioned study-level factors. We decided not to run subgroup analyses on type of control (population-based vs hospital-based), since only one study used hospital-based controls.

In addition, we performed leave-one-out influence analysis to assess the stability of the results.²⁹ In this analysis, we estimated the potential influence of one individual study on the overall pooled RR by omitting one study at a time. Finally, we performed sensitivity analysis by repeating our analysis pooling the minimally adjusted estimates that were presented in the original studies.

Publication bias was assessed by funnel plots and by using Egger's regression asymmetry test and the Begg–Mazumdar³⁰ adjusted rank correlation test.³¹ All statistical analyses were performed using STATA version 11 (StataCorp, College Station, TX, USA). P -values < 0.05 were considered to be statistically significant. All statistical tests were two-sided.

Results

Literature search

The article selection procedure is shown in Figure 1. Briefly, after excluding 1422 articles due to overlap between search categories, the initial search identified 5120 publications to be screened. References were excluded after screening abstracts ($n = 2468$) due to: mortality-survival or prevalence data, not reporting original research or because it was not possible to find the article. In addition, 2627 references were excluded after detailed evaluation if type 2 diabetes was not an outcome, or there was lack of socio-economic data and/or no RRs and CIs or enough data to calculate these. Among the 25 references that fulfilled the inclusion criteria, five new references were identified by hand search and seven references were excluded due to overlaps between data sets. A final number of 23 articles were included in the meta-analysis.^{32–54}

Study characteristics

In the 23 included articles, a total of 41 estimates of association were available (Table 2), which involved approximately 21 978 cases. The higher number of risk estimates compared with number of articles was due to the fact that some studies included more than one measure of SEP and some presented risk estimates on men and women separately. For educational level, 23 risk estimates (from 20 studies) were available, for occupation 11 estimates (from 7 studies) and

Table 1 Studies included in the meta-analysis

References	Setting	Country income level	Study period	Ages ^a (years)	Sex	SEP measure	Number with diagnosed type 2 diabetes in study	Assessment of type 2 diabetes cases	RR (95% CI)	Variables controlled for
Kaye <i>et al.</i> ⁴¹	Nested case-control study, IA, USA	High	1985–87	55–69	Females	Educational level	399	Self-report	2.0 (1.51–2.65)	Controlled for: age by adjustment
Haffner <i>et al.</i> ³⁹	Cohort study, San Antonio, TX, USA	High	1979–87	25–64	Combined	Educational level	46	Blood glucose	1.43 (1.24–1.65)	Controlled for: age, BMI and WHR by adjustment
Maskarinec <i>et al.</i> ⁴⁵	Cohort study, Hawaii, CA, USA	High	1993–2007	45–79	Combined	Educational level	11 838	Register	1.96 (1.0–3.84)	Controlled for: age, sex and ethnicity by matching and BMI by adjustment
Ezeamama <i>et al.</i> ³⁶	Cohort study, American Samoa, USA	High	1990–95	≥ 40	Males	Educational level	147	Self-report	2.6 (1.1–3.6)	Controlled for: age, sex, cigarette smoking and unemployment by adjustment
Lidfeldt <i>et al.</i> ⁴⁴	Cohort study, the Nurse's Health Study, USA	High	1992–2002	46–71	Females	Educational level (husbands educational level)	2457	Self-report	1.79 (1.61–2.0)	Controlled for: age by adjustment
Maty <i>et al.</i> ⁴⁶	Cohort study, Alameda county, USA	High	1965–99	58.6	Combined	Educational level	318	Self-report	1.90 (1.43–2.54)	Controlled for: age by adjustment
									1.27 (0.93–1.74)	Controlled for: demographics, behaviours, body composition (BMI, WHR), high blood pressure, depression, health insurance or regular access to medical doctor or clinic by adjustment
					Males	Occupation	143		1.42 (1.01–2.00)	Controlled for: age by adjustment
									1.19 (0.83–1.72)	Controlled for: demographics, behaviours, body composition (BMI, WHR), high blood pressure, depression, health insurance or regular access to medical doctor or clinic by adjustment

(continued)

Table 1 Continued

References	Setting	Country income level	Study period	Ages ^a (years)	Sex	SEP measure	Number with diagnosed type 2 diabetes in study	Assessment of type 2 diabetes cases	RR (95% CI)	Variables controlled for
					Females	Occupation	175		1.55 (0.99–2.43)	Controlled for: age by adjustment
					Combined	Income	318		0.86 (0.53–1.41)	Controlled for: demographics, behaviours, body composition (BMI, WHR), high blood pressure, depression, health insurance or regular access to medical doctor or clinic by adjustment
					Combined	Income	318		0.75 (0.64–0.87)	Controlled for: age by adjustment
					Combined	Income	318		0.90 (0.76–1.06)	Controlled for: demographics, behaviours, body composition (BMI, WHR), high blood pressure, depression, health insurance or regular access to medical doctor or clinic by adjustment
Robbins <i>et al.</i> ⁵²	Cohort study, NHANES, USA	High	1980–92	29–84	Males	Educational level	306	Medical records	1.84 (1.24–2.73)	None ^b
					Females	Educational level	460		3.95 (2.50–6.26)	None ^b
					Males	Income	306		1.74 (1.01–3.00)	None ^b
					Females	Income	460		2.12 (1.32–3.42)	None ^b
					Males	Occupation	306		1.41 (1.02–2.04)	None ^b
					Females	Occupation	460		3.14 (1.97–5.01)	None ^b
					Combined	Educational level	36	Blood glucose	1.46 (0.43–4.93)	None ^b
					Combined	Income	36		1.18 (0.30–4.67)	None ^b
					Combined	Educational level	548	Self-report verified ^c	1.34 (1.16–1.55)	None ^b
Gaillard <i>et al.</i> ³⁷	Case-control study, OH, USA	High	Not reported	44.3	Combined	Educational level	36	Blood glucose	1.46 (0.43–4.93)	None ^b
					Combined	Income	36		1.18 (0.30–4.67)	None ^b
Mehta <i>et al.</i> ⁴⁸	Case-cohort study, ARIC, 4 counties in USA	High	1987–98	44–65	Combined	Educational level	548	Self-report verified ^c	1.34 (1.16–1.55)	None ^b
Burchfiel <i>et al.</i> ³³	Cohort study, Oahu, HI, USA	High	1965–74	45–68	Males	Educational level	394	Self-report	0.95 (0.72–1.26)	None ^b
Kumari <i>et al.</i> ⁴³	Cohort study, Whitehall II, London, UK	High	1985–99	35–55	Males	Occupation	242	Self-report verified ^c	3.13 (2.10–4.70)	Controlled for: age by adjustment
					Males	Occupation	242		1.52 (0.93–2.48)	Controlled for: age, length of follow-up, ethnicity, family history of diabetes, height,

(continued)

Table 1 Continued

References	Setting	Country income level	Study period	Ages ^a (years)	Sex	SEP measure	Number with diagnosed type 2 diabetes in study	Assessment of type 2 diabetes cases	RR (95% CI)	Variables controlled for
Bourdel-Marchasson <i>et al.</i> ³²	Cohort study, Gironde and Dordogne, France	High	1988–97	74.9	Combined	Educational level	64	Self-report	0.9 (0.55–1.49)	None ^b
Heidemann <i>et al.</i> ⁴⁰	Nested case-control study, EPIC, Potsdam, Germany	High	1994–2001	35–65	Combined	Educational level	192	Self-report verified ^c	1.67 (1.15–2.43)	Controlled for age and sex by matching ^b
Norberg <i>et al.</i> ⁵⁰	Nested case-control study, Umeå, Sweden	High	1989–2000	40, 50, 60	Males	Educational level	113	Register	2.0 (1.1–3.8)	Controlled for: age, sex and year of survey by matching
									1.4 (0.6–3.1)	Controlled for: age, sex and year of survey by matching and psychological demands and decision latitude by adjustment
					Females	Educational level	78		1.4 (0.7–2.7)	Controlled for: age, sex and year of survey by matching
					Females	Educational level	82	Self-report verified ^c	1.7 (0.6–4.8)	Controlled for: age, sex and year of survey by matching and psychological demands and decision latitude by adjustment
Cabrera <i>et al.</i> ³⁴	Cohort study, Gothenburg, Sweden	High	1968–93	38, 46, 50, 54, 60	Female	Occupation			2.27 (1.43–3.59)	Controlled for: age by adjustment
									1.63 (0.96–2.75)	Controlled for: age, WHR, BMI and number of missing teeth by adjustment
Kouvonen <i>et al.</i> ⁴²	Cohort study, Finland	High	1986–2004	18–65	Males	Educational level	313	Register	1.39 (0.98–1.97)	Controlled for: age by adjustment
Radzeviciene <i>et al.</i> ⁵¹	Case-control study, Kaunas, Lithuania	Middle	2001	35–86	Combined	Educational level	234	Blood glucose	1.78 (1.18–2.68)	Controlled for: age and sex by matching ^b

(continued)

Table 1 Continued

References	Setting	Country income level	Study period	Ages ^a (years)	Sex	SEP measure	Number with diagnosed type 2 diabetes in study	Assessment of type 2 diabetes cases	RR (95% CI)	Variables controlled for
Medalie <i>et al.</i> ⁴⁷	Cohort study, Tel Aviv, Israel	High	1963–68	≥ 40	Males	Educational level	373	Blood glucose	1.66 (1.24–2.21)	None ^b
Nagaya <i>et al.</i> ⁴⁹	Cohort study, Gifu Prefecture, Japan	High	1988–2001	30–49	Males	Occupation	373	Blood glucose	1.17 (0.94–1.45)	None ^b
Wang <i>et al.</i> ⁵⁴	Cohort study, A-Lein Township, Southern Taiwan	High	1997–2003	≥ 40	Combined	Educational level	474	Blood glucose	1.67 (1.32–2.11)	None
Costa <i>et al.</i> ³⁵	Case-control study, Cuiabá city, Brazil	Middle	2005	55	Combined	Educational level	206	Blood glucose	1.25 (0.96–1.63)	Controlled for: gender, overweight, obesity, age, smoking, alcohol consumption and interaction term between anti-HCV + by adjustment.
Gao <i>et al.</i> ³⁸	Cohort study, Mauritius	Middle	1987–98	20–65	Males	Educational level	259	Blood glucose	1.43 (0.82–2.48)	Controlled for: age and sex by matching and BMI, income, race and anti-HCV by adjustment
Swai <i>et al.</i> ⁵³	Case-control study, Dar es Salaam, Tanzania	Low	1981–87	46.5	Combined	Income	206	Blood glucose	1.8 (1.06–3.07)	Controlled for: age and sex by matching and BMI, education, race and anti-HCV by adjustment
					Females	Educational level	252	Blood glucose	1.25 (0.81–1.93)	None ^b
					Males	Educational level	252	Blood glucose	1.82 (1.25–2.65)	None ^b
					Females	Income	252	Blood glucose	1.13 (0.81–1.58)	None ^b
					Males	Income	252	Blood glucose	1.54 (1.02–2.33)	None ^b
					Females	Occupation	259	Blood glucose	1.20 (0.88–1.62)	None ^b
					Males	Occupation	252	Blood glucose	1.75 (0.83–3.70)	None ^b
					Combined	Educational level	1250	Blood glucose	1.27 (0.99–1.61)	None ^b

^aAges at baseline or at diagnosis, for cases only or total, depending on reporting, given as mean or age range.

^bRisk estimate calculated from crude data.

^cSelf-report verified, includes self-reported diabetes which was verified by for example inspection of drug packages, medical records and blood glucose. BMI, body mass index; WHR, waist-hip ratio; FHD, family history of diabetes; HRT, hormone replacement therapy; ECG, electrocardiogram; HCV, hepatitis C virus.

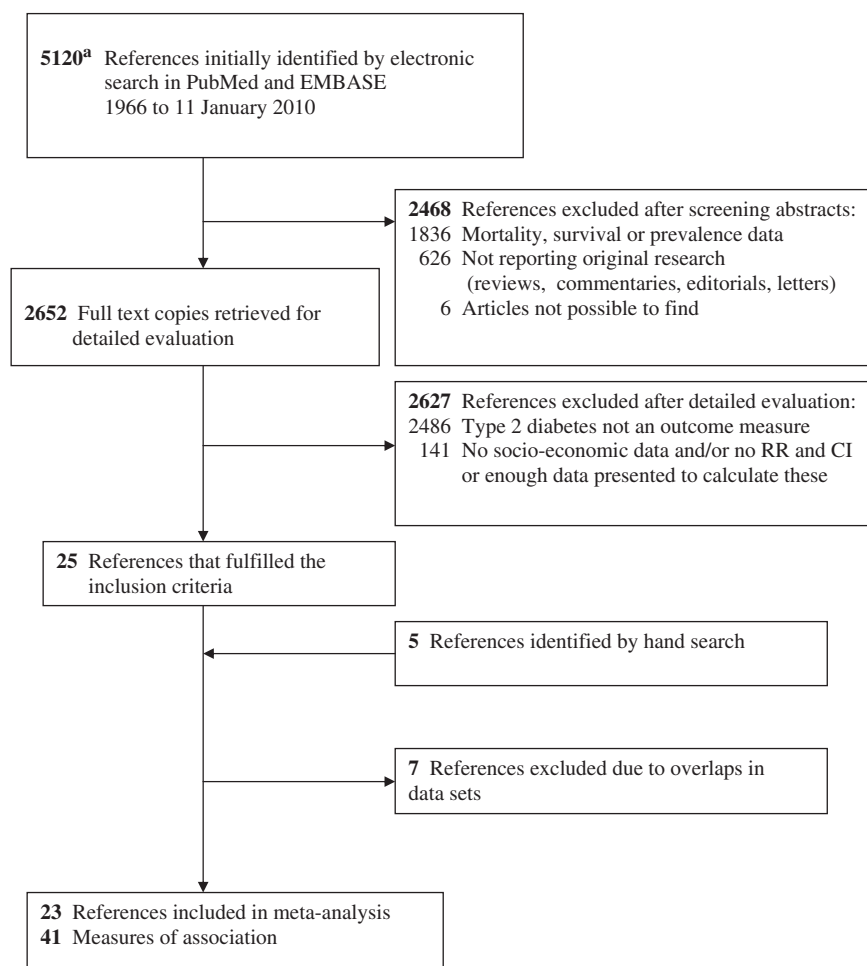


Figure 1 Flow diagram of article selection procedure based on the quality of reports of meta-analyses of randomized controlled trials (QUOROM) statement.¹⁹ ^a6542 publications were identified in the primary electronic search; however, 1422 of these publications were overlaps between search categories and therefore excluded before screening of abstracts

finally for income 7 estimates (from 5 studies) were included. Studies defined low and high educational level differently in different studies. Low educational level ranged from 'no schooling or not having graduated from primary school' to 'no university/academic degree' and high educational level ranged from 'having graduated from primary school and above' to 'university/academic degree'. For occupation, the definitions were not as diverse and categorization of personal income varied according to income level of country.

Fourteen studies presented risk estimates that controlled for confounders other than age and sex, such as for example body mass index and waist-to-hip ratio and ethnicity. The majority of studies (19 studies including 31 estimates) were from high-income countries such as the USA, Great Britain, Sweden, Finland, Japan, Southern Taiwan, Germany and France, whereas three studies (including nine estimates) were from upper-middle-income countries, i.e. Mauritius, Brazil and Lithuania, referred to as

middle-income, and only one was from a low-income country, Tanzania.

In addition, there were 15 estimates of association in men, 12 in women and 14 combined. Thirty estimates derived from cohort studies, whereas 11 came from case-control studies. In seven estimates, diagnosis of type 2 diabetes was assessed through self-report, in seven studies by self-report verified, in 17 studies by blood glucose, in four by register and in six by medical records.

Overall summary of low vs high SEP

In the overall summary, there was an increased risk of type 2 diabetes in the lowest compared with the highest SEP groups, measured by educational level (RR=1.41, 95% CI: 1.28–1.51), occupation (RR=1.31, 95% CI: 1.09–1.57) or income (RR=1.40, 95% CI: 1.04–1.88) (Figure 2). A moderate heterogeneity was observed for all three indicators, i.e. educational level ($P < 0.001$, $I^2 = 65.5\%$), occupation

Table 2 Pooled estimates for lowest vs highest SEP group and incidence of type 2 diabetes, in series of sub-group analysis for studies included in the analysis (including the most adjusted estimate in each study, ranging from crude to multi-adjusted)

Sub-group analysis	Pooled RR (95% CI), P-value for the heterogeneity Q test, I ² statistics (%), number of estimates in included studies (n)					
	n	Educational level	n	Occupation	n	Income
Overall	23	1.41 (1.28–1.51); P < 0.001, I ² = 65.5	11	1.31 (1.09–1.57); P = 0.020, I ² = 52.8	7	1.40 (1.04–1.88); P = 0.002, I ² = 71.9
Sex						
Men	7	1.46 (1.15–1.86); P = 0.020, I ² = 60.0	6	1.19 (1.05–1.36); P = 0.598, I ² = 0.0	2	1.33 (0.88–1.20); P = 0.185, I ² = 43.0
Women	5	1.72 (1.26–2.35); P = 0.001, I ² = 87.4	5	1.52 (0.93–2.49); P = 0.008, I ² = 71.0	2	1.77 (1.29–2.41); P = 0.320, I ² = 0.0
Combined	11	1.28 (1.18–1.38); P < 0.001, I ² = 16.6	0	–	3	1.19 (0.69–2.06); P = 0.050, I ² = 66.6
Income level of countries						
High-income	18	1.45 (1.28–1.63); P < 0.001, I ² = 70.1	9	1.31 (1.05–1.63); P = 0.009, I ² = 60.7	4	1.40 (0.81–2.42); P = 0.002, I ² = 79.9
Middle-income	4	1.59 (1.28–1.97); P = 0.550, I ² = 0.0	2	1.27 (0.96–1.68); P = 0.360, I ² = 0.0	3	1.39 (1.06–1.82); P = 0.274, I ² = 22.7
Low-income	1	1.27 (0.99–1.62)	0	–	0	–
Geographical area						
USA	11	1.41 (1.24–1.62); P < 0.001, I ² = 79.2	4	1.43 (0.91–2.24); P = 0.003, I ² = 78.9	4	1.40 (0.18–2.42); P = 0.002, I ² = 79.9
Europe	6	1.45 (1.20–1.76); P = 0.394, I ² = 3.5	3	1.47 (1.06–2.05); P = 0.646, I ² = 0.0	0	–
Asia/Middle East	2	1.43 (1.08–1.89); P = 0.156, I ² = 50.3	2	1.10 (0.90–1.33); P = 0.294, I ² = 9.2	0	–
Latin America	1	1.43 (1.20–1.76)	0	–	1	1.80 (1.06–3.01)
Africa	2	1.40 (1.15–1.75); P = 0.255, I ² = 26.8	2	1.27 (0.96–1.68); P = 0.360, I ² = 0.0	2	1.29 (0.95–1.74); P = 0.253, I ² = 23.3
Adjustment						
Minimally adjusted	13	1.50 (1.27–1.79); P < 0.001, I ² = 68.7	5	1.51 (1.12–2.03); P = 0.009, I ² = 70.2	5	1.50 (1.17–1.92); P = 0.270, I ² = 22.6
Maximally adjusted	10	1.28 (1.17–1.40); P = 0.086, I ² = 40.7	6	1.14 (0.93–1.39); P = 0.338, I ² = 12.1	2	1.21 (0.62–2.38); P = 0.015, I ² = 83.1
Publication year						
<2000s	6	1.35 (1.13–1.61); P = 0.073, I ² = 50.4	1	1.17 (0.94–1.45)	1	1.18 (0.30–4.66)
≥2000s	17	1.44 (1.29–1.62); P < 0.001, I ² = 68.6	10	1.34 (1.08–1.66); P = 0.015, I ² = 56.1	6	1.41 (1.03–1.91); P = 0.001, I ² = 76.6
Design						
Case-control	9	1.40 (1.24–1.62); P = 0.909, I ² = 0.0	0	–	2	1.70 (1.04–2.80); P = 0.574, I ² = 0.0
Cohort	14	1.42 (1.24–1.62); P < 0.001, I ² = 75.3	11	1.31 (1.09–1.57); P = 0.020, I ² = 52.8	5	1.36 (0.97–1.90); P = 0.001, I ² = 78.0
Length of follow-up						
<10 years	6	1.37 (1.03–1.82); P = 0.006, I ² = 69.6	2	1.10 (0.90–1.33); P = 0.294, I ² = 9.2	0	–
≥10 years	8	1.47 (1.24–1.73); P < 0.001, I ² = 80.4	9	1.41 (1.13–1.76); P = 0.036, I ² = 51.5	5	1.36 (0.97–1.90); P = 0.001, I ² = 78
Assessment of cases						
Self-report	4	1.13 (1.03–1.25); P = 0.386, I ² = 1.20	2	1.06 (0.78–1.44); P = 0.297, I ² = 8.2	1	0.90 (7.62–1.06)
Self-report verified	4	1.47 (1.27–1.72); P = 0.149, I ² = 43.8	3	1.47 (1.06–2.05); P = 0.646, I ² = 0.0	0	–
Blood glucose	9	1.45 (1.28–1.64); P = 0.560, I ² = 0.0	4	1.15 (0.98–1.34); P = 0.456, I ² = 0.0	4	1.36 (1.08–1.71); P = 0.452, I ² = 0.0
Register	4	1.20 (1.15–1.26); P = 0.747, I ² = 0.0	0	–	0	–
Medical records	2	1.67 (1.26–5.65); P = 0.013, I ² = 83.7	2	2.00 (0.98–4.11); P = 0.014, I ² = 83.6	2	1.95 (1.36–2.79); P = 0.592, I ² = 0.0

(continued)

Table 2 Continued

		Pooled RR (95% CI), P-value for the heterogeneity Q test, I ² statistics (%), number of estimates in included studies (n)					
Sub-group analysis		n	Educational level	n	Occupation	n	Income
Number of SEP categories							
2	9	1.34 (1.12–1.60); P = 0.047, I ² = 49.1	3	1.01 (0.81–1.26); P < 0.507, I ² = 0.0	2	1.21 (0.62–2.38); P = 0.015, I ² = 83.1	
3	11	1.31 (1.20–1.42); P = 0.065, I ² = 42.7	6	1.26 (1.08–1.47); P < 0.697, I ² = 0.0	2	1.29 (0.95–1.74); P = 0.253, I ² = 23.3	
4	1	1.46 (0.43–4.93)	2	2.00 (0.98–4.11); P < 0.014, I ² = 83.6	1	1.18 (0.30–4.66)	
5	2	2.67 (1.26–5.65); P = 0.013, I ² = 83.7	0	–	2	1.95 (1.36–2.79); P = 0.592, I ² = 0.0	
Type of SEP							
Own	22	1.45 (1.30–1.61); P < 0.001, I ² = 65.8	10	1.29 (1.06–1.56); P = 0.016, I ² = 55.6	3	1.89 (1.33–2.67); P = 0.682, I ² = 0.0	
Husband	1	1.16 (1.04–1.29)	1	1.63 (0.96–2.76)	0	–	
Household	0	–	0	–	4	1.22 (0.89–1.68); P = 0.014, I ² = 71.9	

(P = 0.020, I² = 52.8%) and income (P = 0.002, I² = 71.9%) (Table 2).

Sub-group analysis

In the sub-group analysis by income level of countries, low educational level and low occupation was associated with a 45% and a 31% increased risk of type 2 diabetes in high-income countries, respectively. In addition, low income was associated with an increased risk of disease (RR = 1.40, 95% CI: 0.81–2.42) (Table 2). Also in middle-income countries, the increased risks of type 2 diabetes remained for low educational level, low occupation and low income. Only one study from a low-income country was included (Table 2).

When we performed sub-group analyses for minimally and maximally adjusted estimates, the associations between type 2 diabetes incidence and low educational level were (RR = 1.50, 95% CI: 1.27–1.78) for minimally adjusted and (RR = 1.28, 95% CI: 1.17–1.40) for maximally adjusted. There was also an increased risk in association with low occupation for both minimally adjusted (51%) and maximally adjusted (14%) estimates. Even low income was associated with an increased risk for minimally adjusted (50%) estimates and a tendency for an increased risk of maximally adjusted estimates (RR = 1.21, 95% CI: 0.62–2.38) was observed (Table 2).

When we investigated the relation between type 2 diabetes incidence and SEP by other sub-group analysis such as sex, geographical area, publication year, study design, length of follow-up in cohort studies, assessment of cases, number of SEP categories and type of SEP the increased risk persisted in the majority of analyses (Table 2). Moreover, a low or moderate heterogeneity was present for the majority of sub-group analyses (Table 2).

In random-effect regression analyses for studies using educational level as SEP, both univariate and multivariate analyses indicated a relation between RR of type 2 diabetes and assessment of cases (self-report verified, blood glucose, registers or records combined vs self-reported) with P-values of 0.006 for univariate and 0.007 for multivariate analyses. For studies using occupation as a measure of SEP, only geographical area (Europe, Asia/Middle East, Latin America and Africa combined vs USA) was associated with type 2 diabetes with P-value of 0.04 in the multivariate regression analyses. In the same groups of studies, number of SEP categories (3, 4 and 5 categories combined vs 2 categories) was related to the disease in univariate (P = 0.04), but not in multivariate meta-regression analysis. For studies based on income, assessments of cases were the only variables that revealed an association with outcome in univariate regression analysis (P < 0.001), and the association was no longer apparent in multivariate analysis. No other study-level characteristics for any of the SEP indicators were found in relation to type 2 diabetes.

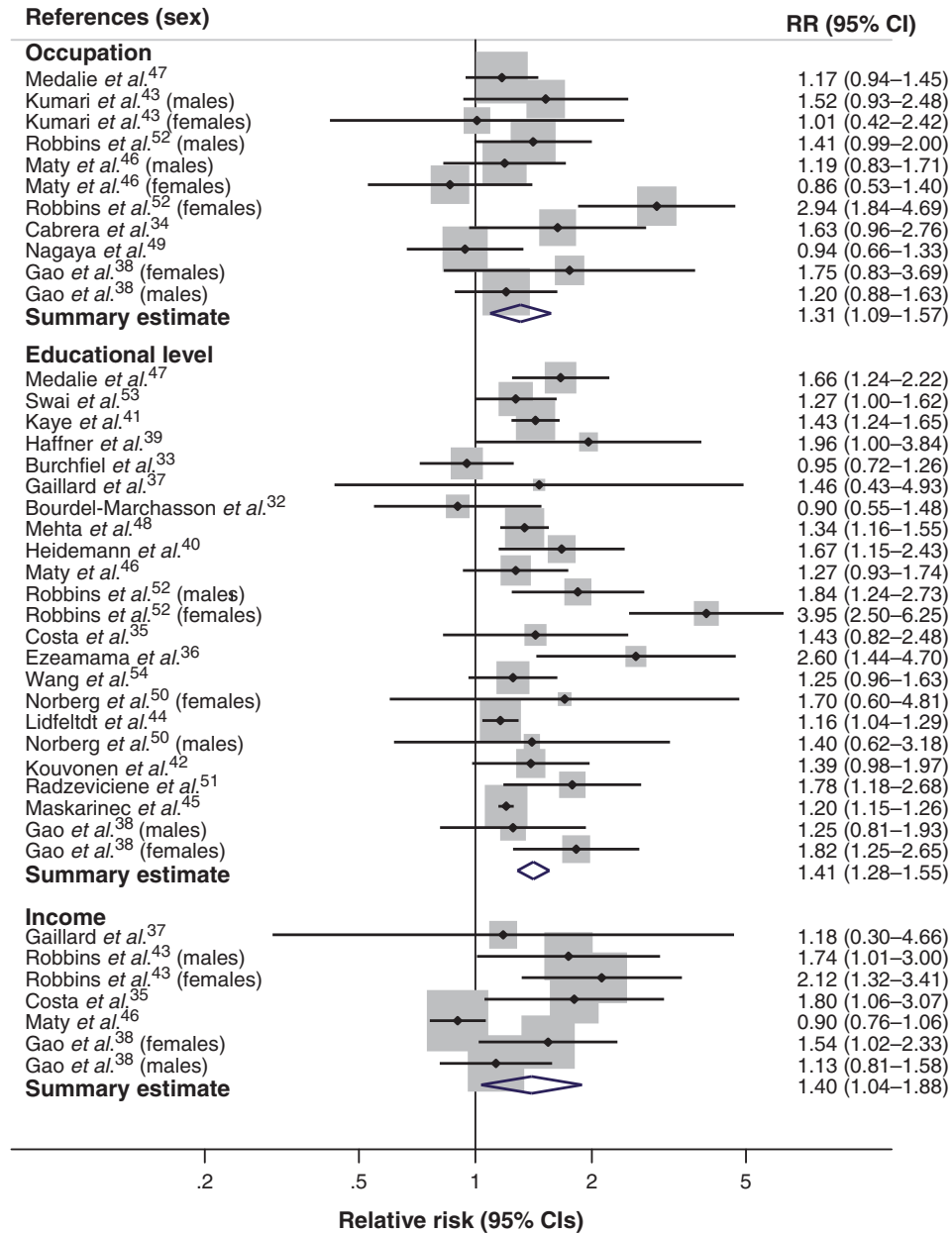


Figure 2 Relative risks and 95% CIs of type 2 diabetes for the lowest vs highest level of educational level, occupation and income

Publication bias, influence and sensitivity analysis

Publication bias was observed when pooling studies on educational level (Egger’s test for publication bias; $P=0.005$) and for income ($P=0.034$). However, publication bias was not obvious for occupation ($P=0.373$). The influence analysis showed that no individual study significantly altered the overall estimates based on the results from educational level and occupation. For income, however, two studies,^{38,52} including four estimates, could influence the pooled results if omitted. However, even if decreasing the estimate to some extent (2–6%), the

increased risk of type 2 diabetes still persisted (data not shown).

In the sensitivity analyses, 36 risk estimates (19 for educational level, 11 for occupation and 6 for income) from 18 studies originally reporting minimally adjusted RRs and 95% CIs were included. The results were in line with pooled estimates found in meta-analysis (data not shown).

Discussion

The results of our study suggest an overall increased risk of type 2 diabetes in low socio-economic groups,

whether measured by educational level (41%), occupation (31%) or income (40%). The socioeconomic differential was consistent in high-income countries. Although increased risks were observed with lower SEP also in middle- and low-income countries, data from these economies were very limited.

Type 2 diabetes has been reported to be more prevalent among those with high SEP in middle- and low-income countries.^{11–15} Our systematic review and meta-analysis of incident cases of type 2 diabetes showed a reverse scenario. It is possible that including prevalence data on type 2 diabetes and SEP from these countries would have changed the picture. On the other hand, even if few incidence studies were identified, the results should not be neglected, although further investigation is strongly needed to clarify this issue. We found for example no publication from either China or India, countries that have been going through rapid economic growth during the previous decade, together with steadily increasing numbers of diagnosed patients.^{14,16,55–57}

In our meta-analysis, we did not combine educational level, occupation and income. It was apparent that educational level was the most commonly used measure of SEP, and also most consistently associated with increased risk of disease. Although it is evident that education in general leads to occupations that influence level of income, it has been argued that these SEP measures cannot be used interchangeably as they represent different causal processes and pathways.⁵⁸ Education has been described as capturing the transition from parental SEP to adult SEP, reflecting factors such as material and intellectual resources of family origin. The skills and knowledge attained through education may affect the receptiveness to health information and appropriate communication with health-care services.⁵⁹

The mechanisms through which low SEP could relate to type 2 diabetes are not clear. In most of the included studies, unhealthy characteristics could not fully explain socio-economic differences in type 2 diabetes incidence, indicating that other factors may be involved. For example, few of the included studies adjusted for psycho-social stress factors.^{43,50} A lower socio-economic status is related to higher stress levels⁶⁰ and long-term stress affects the entire neuroendocrine system involving endocrine perturbations which in turn may lead to type 2 diabetes.^{61,62} It should be mentioned, however, that explaining causes and underlying mechanisms is beyond the scope of this study due to the observational nature of studies included in our meta-analysis.

Socio-economic inequalities in type 2 diabetes incidence were more pronounced in women than men. This is in line with previous cross-sectional findings,⁶ and a possible explanation could be that women in lower SEP groups are obese, physical inactive and experience psycho-social stress to a higher extent than men in these groups.^{10,63} It has been suggested that

genetic susceptibility may be responsible for differences of type 2 diabetes in certain ethnic groups.^{64,65} However, it has also been argued that a continuous focus on ethnicity as a primary determinant may divert effort from interventions improving social circumstances.⁶⁶ We did not perform any separate analyses of ethnicity since there were too few studies investigating this more than as a confounding factor.

There are limitations of this study. There may be potential bias related to reverse causality. If those with type 2 diabetes in the selected studies had lower SEP due to their disease, the effects of the association may be overestimated. However, we believe that this scenario is less likely since formal education is normally completed in young adulthood, and we assume that many had started to work before their diabetes diagnosis. Moreover, one of the strengths of this study is that our quantitative assessment was based on incident cases. Still, although prevalent cases were excluded at baseline, income tends to fall when someone gets chronically ill.⁵⁸

Educational level, occupation and income may have been defined and classified differently across studies, due to different meanings for different birth cohorts and different geographical settings.⁵⁹ For example, educational level and income may vary significantly between countries due to differences in country economies and educational systems, while occupation may be organized differently with regard to social standing,⁵⁹ physical and psychological work environment⁵⁸ and also due to a changing structure and composition of workforce.⁵⁹ In addition, in many studies SEP was not the exposure of interest and hence poorly explained. This may introduce difficulties when combining data and making international comparisons. However, by dividing the SEP groups into two extreme categories, high and low, we assume that we have captured the sense of SEP, irrespective of time and place.

The different ways of controlling for confounding factors between studies may have influenced the results. To address the possible effect of this phenomenon, we performed a series of sub-group analyses of minimally and maximally adjusted estimates as well as sensitivity analysis. Although the increased risk persisted, the effect was diluted when studies adjusted for outcome-related risk factors were pooled. This raises the question whether the included variables should be considered as confounders or intermediates in the causal pathway between type 2 diabetes incidence and SEP. However, a possible adjustment for intermediates rather than confounders would lead to underestimation of the pooled RRs.

Type 2 diabetes is a chronic insidious disease that develops gradually⁶⁷ and many people are undiagnosed.⁶⁸ Since health-care services have been found to be less accessible to people with low SEP,³ it is also possible that they are not diagnosed to the same extent as those with high SEP. Exclusion of cases at

baseline based on self-report could mean that more cases with low SEP were diagnosed during follow-up and hence the results may be overestimated. Moreover, different studies used different ways of diagnosing type 2 diabetes. Lack of uniformity in outcome definition may contribute to the heterogeneity that was indicated in the meta-regression analysis.

Publication bias is a significant concern to the validity in meta-analysis,⁶⁹ and can lead to overestimation of the risk estimates. In addition, it was shown that there was evidence of publication bias for both educational level and income. Furthermore, our meta-analysis was restricted to peer-reviewed publications written in English, which may result in lack of data from certain countries. The way this may influence the results is however difficult to predict.

Recall bias may be a problem in case-control studies. However, it has been found that recall of SEP is reliable regardless of SEP group⁷⁰ and hence this should not influence the findings.

In conclusion, this first systematic review and meta-analysis suggest an association between type 2 diabetes incidence and low SEP. The strength of the association measured by educational level and occupation is consistent in high-income countries. Although the risk among those with low SEP is

increased also in middle- and low-income countries, available data are limited. It should be noted that we carefully searched for and picked up all eligible published raw data, also in studies not directly investigating the association between type 2 diabetes incidence and SEP. Against the background of an epidemic increase of type 2 diabetes, and for possible targeting of prevention, it is important to pattern the disease in different groups of society. Future well-designed research is therefore necessary and greatly needed to characterize the relationship between type 2 diabetes incidence and SEP also in middle- and low-income countries.

Funding

Research grants from the Swedish Council for Working Life and Social Research (FAS 2007-0606 and 2006-0230).

Conflict of interest: The study founder had no role in the design, collection, analysis or interpretation of the data or in writing or decision to submit the article.

KEY MESSAGES

- Type 2 diabetes incidence is associated with low SEP whether measured by educational level, occupation or income, worldwide and when sub-divided into high-, middle- and low-income countries.
- The associations were consistent in high-income countries and although an increased risk was apparent also in middle- and low-income countries, available data from these countries were limited.
- There is a pressing need for well-designed research characterizing the relationship between SEP and type 2 diabetes incidence in middle- and low-income countries.

References

- 1 Amos AF, McCarty DJ, Zimmet P. The rising global burden of diabetes and its complications: estimates and projections to the year 2010. *Diabet Med* 1997; **14**(Suppl. 5):S1–85.
- 2 King H, Aubert RE, Herman WH. Global burden of diabetes, 1995–2025: prevalence, numerical estimates, and projections. *Diabetes Care* 1998; **21**:1414–31.
- 3 Brown AF, Ettner SL, Piette J *et al.* Socioeconomic position and health among persons with diabetes mellitus: a conceptual framework and review of the literature. *Epidemiol Rev* 2004; **26**:63–77.
- 4 Agardh EE, Ahlbom A, Andersson T *et al.* Explanations of socioeconomic differences in excess risk of type 2 diabetes in Swedish men and women. *Diabetes Care* 2004; **27**: 716–21.
- 5 Connolly V, Unwin N, Sherriff P, Bilous R, Kelly W. Diabetes prevalence and socioeconomic status: a population based study showing increased prevalence of type 2 diabetes mellitus in deprived areas. *J Epidemiol Community Health* 2000; **54**:173–77.
- 6 Espelt A, Borrell C, Roskam AJ *et al.* Socioeconomic inequalities in diabetes mellitus across Europe at the beginning of the 21st century. *Diabetologia* 2008; **51**:1971–79.
- 7 Evans JM, Newton RW, Ruta DA, MacDonald TM, Morris AD. Socio-economic status, obesity and prevalence of Type 1 and Type 2 diabetes mellitus. *Diabet Med* 2000; **17**:478–80.
- 8 Meadows P. Variation of diabetes mellitus prevalence in general practice and its relation to deprivation. *Diabet Med* 1995; **12**:696–700.
- 9 Robbins JM, Vaccarino V, Zhang H, Kasl SV. Socioeconomic status and type 2 diabetes in African American and non-Hispanic white women and men: evidence from the Third National Health and Nutrition Examination Survey. *Am J Public Health* 2001; **91**:76–83.
- 10 Tang M, Chen Y, Krewski D. Gender-related differences in the association between socioeconomic status and self-reported diabetes. *Int J Epidemiol* 2003; **32**:381–85.
- 11 abu Sayeed M, Ali L, Hussain MZ, Rumi MA, Banu A, Azad Khan AK. Effect of socioeconomic risk factors on the difference in prevalence of diabetes between rural and

- urban populations in Bangladesh. *Diabetes Care* 1997;**20**: 551–55.
- ¹² Ali O, Tan TT, Sakinah O, Khalid BA, Wu LL, Ng ML. Prevalence of NIDDM and impaired glucose tolerance in aborigines and Malays in Malaysia and their relationship to sociodemographic, health, and nutritional factors. *Diabetes Care* 1993;**16**:68–75.
- ¹³ Illangasekera U, Rambodagalla S, Tennakoon S. Temporal trends in the prevalence of diabetes mellitus in a rural community in Sri Lanka. *J R Soc Promot Health* 2004;**124**: 92–94.
- ¹⁴ Ramachandran A, Snehalatha C, Kapur A *et al.* High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetologia* 2001; **44**:1094–101.
- ¹⁵ Xu F, Yin XM, Zhang M, Leslie E, Ware R, Owen N. Family average income and diagnosed Type 2 diabetes in urban and rural residents in regional mainland China. *Diabet Med* 2006;**23**:1239–46.
- ¹⁶ Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. *Nature* 2001;**414**: 782–87.
- ¹⁷ Feinstein JS. The relationship between socioeconomic status and health: a review of the literature. *Milbank Q* 1993;**71**:279–322.
- ¹⁸ Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. *JAMA* 1998;**279**: 1703–08.
- ¹⁹ Moher D, Cook DJ, Eastwood S, Olkin I, Rennie D, Stroup DF. Improving the quality of reports of meta-analyses of randomised controlled trials: the QUOROM statement. Quality of Reporting of Meta-analyses. *Lancet* 1999;**354**:1896–900.
- ²⁰ Stroup DF, Berlin JA, Morton SC *et al.* Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. *JAMA* 2000;**283**: 2008–12.
- ²¹ Rothman K, Greenland S, Lash T (eds). *Modern Epidemiology*. 3rd edn. Philadelphia: Lippincott Williams & Wilkins, 2008.
- ²² Greenland S. Quantitative methods in the review of epidemiologic literature. *Epidemiol Rev* 1987;**9**:1–30.
- ²³ DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials* 1986;**7**:177–88.
- ²⁴ Cochran W. The combination of estimates from different experiments. *Biometrics* 1954;**9**:101–29.
- ²⁵ Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ* 2003; **327**:557–60.
- ²⁶ Baker WL, White CM, Cappelleri JC, Kluger J, Coleman CI. Understanding heterogeneity in meta-analysis: the role of meta-regression. *Int J Clin Pract* 2009;**63**:1426–34.
- ²⁷ Thompson SG, Sharp SJ. Explaining heterogeneity in meta-analysis: a comparison of methods. *Stat Med* 1999; **18**:2693–708.
- ²⁸ van Houwelingen HC, Arends LR, Stijnen T. Advanced methods in meta-analysis: multivariate approach and meta-regression. *Stat Med* 2002;**21**:589–624.
- ²⁹ Tobias A. Assessing the influence of a single study in meta-analysis. *Stata Technical Bulletin* 1999;**47**:15–17.
- ³⁰ Begg CB, Mazumdar M. Operating characteristics of a rank correlation test for publication bias. *Biometrics* 1994;**50**:1088–101.
- ³¹ Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 1997;**315**:629–34.
- ³² Bourdel-Marchasson I, Dubroca B, Letenneur L *et al.* Incidence and predictors of drug-treated diabetes in elderly French subjects. The PAQUID Epidemiological Survey. *Diabet Med* 2000;**17**:675–81.
- ³³ Burchfiel CM, Curb JD, Rodriguez BL *et al.* Incidence and predictors of diabetes in Japanese-American men. The Honolulu Heart Program. *Ann Epidemiol* 1995;**5**:33–43.
- ³⁴ Cabrera C, Hakeberg M, Ahlqvist M *et al.* Can the relation between tooth loss and chronic disease be explained by socio-economic status? A 24-year follow-up from the population study of women in Gothenburg, Sweden. *Eur J Epidemiol* 2005;**20**:229–36.
- ³⁵ Costa LM, Mussi AD, Brianeze MR, Souto FJ. Hepatitis C as a risk factor for diabetes type 2: lack of evidence in a hospital in central-west Brazil. *Braz J Infect Dis* 2008;**12**: 24–26.
- ³⁶ Ezeamama AE, Viali S, Tuitele J, McGarvey ST. The influence of socioeconomic factors on cardiovascular disease risk factors in the context of economic development in the Samoan archipelago. *Soc Sci Med* 2006;**63**:2533–45.
- ³⁷ Gaillard TR, Schuster DP, Bossetti BM, Green PA, Osei K. Do sociodemographics and economic status predict risks for type II diabetes in African Americans? *Diabetes Educ* 1997;**23**:294–300.
- ³⁸ Gao WG, Qiao Q, Pitkaniemi J *et al.* Risk prediction models for the development of diabetes in Mauritian Indians. *Diabet Med* 2009;**26**:996–1002.
- ³⁹ Haffner SM, Hazuda HP, Mitchell BD, Patterson JK, Stern MP. Increased incidence of type II diabetes mellitus in Mexican Americans. *Diabetes Care* 1991;**14**:102–08.
- ⁴⁰ Heidemann C, Hoffmann K, Spranger J *et al.* A dietary pattern protective against type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition (EPIC)—Potsdam Study cohort. *Diabetologia* 2005;**48**: 1126–34.
- ⁴¹ Kaye SA, Folsom AR, Sprafka JM, Prineas RJ, Wallace RB. Increased incidence of diabetes mellitus in relation to abdominal adiposity in older women. *J Clin Epidemiol* 1991;**44**:329–34.
- ⁴² Kouvonen AM, Vaananen A, Woods SA, Heponiemi T, Koskinen A, Toppinen-Tanner S. Sense of coherence and diabetes: a prospective occupational cohort study. *BMC Public Health* 2008;**8**:46.
- ⁴³ Kumari M, Head J, Marmot M. Prospective study of social and other risk factors for incidence of type 2 diabetes in the Whitehall II study. *Arch Intern Med* 2004;**164**: 1873–80.
- ⁴⁴ Lidfeldt J, Li TY, Hu FB, Manson JE, Kawachi I. A prospective study of childhood and adult socioeconomic status and incidence of type 2 diabetes in women. *Am J Epidemiol* 2007;**165**:882–89.
- ⁴⁵ Maskarinec G, Erber E, Grandinetti A *et al.* Diabetes incidence based on linkages with health plans: the multi-ethnic cohort. *Diabetes* 2009;**58**:1732–38.

- ⁴⁶ Maty SC, Everson-Rose SA, Haan MN, Raghunathan TE, Kaplan GA. Education, income, occupation, and the 34-year incidence (1965–99) of Type 2 diabetes in the Alameda County Study. *Int J Epidemiol* 2005;**34**:1274–81.
- ⁴⁷ Medalie JH, Papier C, Herman JB *et al*. Diabetes mellitus among 10,000 adult men. I. Five-year incidence and associated variables. *Isr J Med Sci* 1974;**10**:681–97.
- ⁴⁸ Mehta SH, Brancati FL, Strathdee SA *et al*. Hepatitis C virus infection and incident type 2 diabetes. *Hepatology* 2003;**38**:50–56.
- ⁴⁹ Nagaya T, Yoshida H, Takahashi H, Kawai M. Policemen and firefighters have increased risk for type-2 diabetes mellitus probably due to their large body mass index: a follow-up study in Japanese men. *Am J Ind Med* 2006;**49**:30–35.
- ⁵⁰ Norberg M, Stenlund H, Lindahl B, Andersson C, Eriksson JW, Weinehall L. Work stress and low emotional support is associated with increased risk of future type 2 diabetes in women. *Diabetes Res Clin Pract* 2007;**76**:368–77.
- ⁵¹ Radzeviciene L, Ostrauskas R. Smoking habits and the risk of type 2 diabetes: a case-control study. *Diabetes Metab* 2009;**35**:192–97.
- ⁵² Robbins JM, Vaccarino V, Zhang H, Kasl SV. Socioeconomic status and diagnosed diabetes incidence. *Diabetes Res Clin Pract* 2005;**68**:230–36.
- ⁵³ Swai AB, Lutale J, McLarty DG. Diabetes in tropical Africa: a prospective study, 1981–7. I. Characteristics of newly presenting patients in Dar es Salaam, Tanzania, 1981–7. *BMJ* 1990;**300**:1103–06.
- ⁵⁴ Wang CS, Wang ST, Yao WJ, Chang TT, Chou P. Hepatitis C virus infection and the development of type 2 diabetes in a community-based longitudinal study. *Am J Epidemiol* 2007;**166**:196–203.
- ⁵⁵ Pan XR, Yang WY, Li GW, Liu J. Prevalence of diabetes and its risk factors in China, 1994. National Diabetes Prevention and Control Cooperative Group. *Diabetes Care* 1997;**20**:1664–69.
- ⁵⁶ Ramachandran A, Ma RC, Snehalatha C. Diabetes in Asia. *Lancet* 2010;**375**:408–18.
- ⁵⁷ Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M. Rising prevalence of NIDDM in an urban population in India. *Diabetologia* 1997;**40**:232–37.
- ⁵⁸ Geyer S, Hemstrom O, Peter R, Vagero D. Education, income, and occupational class cannot be used interchangeably in social epidemiology. Empirical evidence against a common practice. *J Epidemiol Community Health* 2006;**60**:804–10.
- ⁵⁹ Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 1). *J Epidemiol Community Health* 2006;**60**:7–12.
- ⁶⁰ Meyer IH, Schwartz S, Frost DM. Social patterning of stress and coping: does disadvantaged social statuses confer more stress and fewer coping resources? *Soc Sci Med* 2008;**67**:368–79.
- ⁶¹ Bjorntorp P, Holm G, Rosmond R. Hypothalamic arousal, insulin resistance and Type 2 diabetes mellitus. *Diabet Med* 1999;**16**:373–83.
- ⁶² Rosmond R, Bjorntorp P. The hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. *J Intern Med* 2000;**247**:188–97.
- ⁶³ Loucks EB, Rehkopf DH, Thurston RC, Kawachi I. Socioeconomic disparities in metabolic syndrome differ by gender: evidence from NHANES III. *Ann Epidemiol* 2007;**17**:19–26.
- ⁶⁴ Dowse G, Zimmet P. The thrifty genotype in non-insulin dependent diabetes. *BMJ* 1993;**306**:532–33.
- ⁶⁵ Neel JV. Diabetes mellitus: a ‘thrifty’ genotype rendered detrimental by ‘progress’? *Am J Hum Genet* 1962;**14**:353–62.
- ⁶⁶ Link CL, McKinlay JB. Disparities in the prevalence of diabetes: is it race/ethnicity or socioeconomic status? Results from the Boston Area Community Health (BACH) survey. *Ethn Dis* 2009;**19**:288–92.
- ⁶⁷ Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 2003;**26**(Suppl. 1):S5–20.
- ⁶⁸ Harris MI. Undiagnosed NIDDM: clinical and public health issues. *Diabetes Care* 1993;**16**:642–52.
- ⁶⁹ Zwahlen M. Meta-analysis in medical research: potentials and limitations. *Urol Oncol* 2008;**26**:320–29.
- ⁷⁰ Krieger N, Okamoto A, Selby JV. Adult female twins’ recall of childhood social class and father’s education: a validation study for public health research. *Am J Epidemiol* 1998;**147**:704–08.