BMJ Open

Income and obesity: what is the direction of the relationship? A systematic review and meta-analysis

Tae Jun Kim, Olaf von dem Knesebeck

To cite: Kim TJ, von dem Knesebeck O. Income and obesity: what is the direction of the relationship? A systematic review and meta-analysis. *BMJ Open* 2018;**8**:e019862. doi:10.1136/bmjopen-2017-019862

► Prepublication history and additional material for this paper are available online. To view these files, please visit the journal online (http://dx.doi.org/10.1136/bmjopen-2017-019862).

Received 2 October 2017
Revised 27 November 2017
Accepted 30 November 2017

Department of Medical Sociology, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Correspondence to
Professor Olaf von dem Knesebeck;
o.knesebeck@uke.de

ABSTRACT

Objective It was repeatedly shown that lower income is associated with higher risks for subsequent obesity. However, the perspective of a potential reverse causality is often neglected, in which obesity is considered a cause for lower income, when obese people drift into lower-income jobs due to labour–market discrimination and public stigmatisation. This review was performed to explore the direction of the relation between income and obesity by specifically assessing the importance of social causation and reverse causality.

Design Systematic review and meta-analysis.

Methods A systematic literature search was conducted in January 2017. The databases Medline, PsycINFO, Sociological Abstracts, International Bibliography of Social Sciences and Sociological Index were screened to identify prospective cohort studies with quantitative data on the relation between income and obesity. Meta-analytic methods were applied using random-effect models, and the quality of studies assessed with the Newcastle-Ottawa Scale.

Results In total, 21 studies were eligible for meta-analysis. All included studies originated from either the USA (n=16), the UK (n=3) or Canada (n=2). From these, 14 studies on causation and 7 studies on reverse causality were found. Meta-analyses revealed that lower income is associated with subsequent obesity (OR 1.27, 95% CI 1.10 to 1.47; risk ratio 1.52, 95% CI 1.08 to 2.13), though the statistical significance vanished once adjusted for publication bias. Studies on reverse causality indicated a more consistent relation between obesity and subsequent income, even after taking publication bias into account (standardised mean difference −0.15, 95% CI −0.30 to 0.01). Sensitivity analyses implied that the association is influenced by obesity measurement, gender, length of observation and study quality.

Conclusions Findings suggest that there is more consistent evidence for reverse causality. Therefore, there is a need to examine reverse causality processes in more detail to understand the relation between income and obesity.

PROSPERO registration number 42016041296.

Strengths and limitations of this study

- This is the first systematic review that gives an overview of causation and reverse causality processes in the link between income and obesity.
- Although only those studies that examined the relation between income and obesity longitudinally were included, the question of the direction of the relationship cannot be fully answered.
- The evidence is restricted to a few countries as all included studies have their origin in the USA, the UK and Canada.

INTRODUCTION

Obesity is a major public health issue. According to a recent trend analysis in 200 countries, age-standardised prevalence of obesity increased from 3.2% to 10.8% between 1975 and 2014 in men, and from 6.4% to 14.9% in women.[1] In this study, like in many others, obesity was defined by a body mass index (BMI) of 30 or higher. Obesity is a major risk factor for all-cause mortality, a number of non-communicable diseases and reduced quality of life.[2–6]

Within social epidemiological research, income (as an indicator of the socioeconomic status; SES) was found to be inversely associated with obesity,[7–9] though this relationship can be interpreted in two directions: (1) the causation hypothesis that explains lower income as a cause for subsequent obesity and (2) the perspective of a reversed causality, in which obesity is not the result, but rather the cause for lower income.

In order to describe why people with lower income are more vulnerable to obesity, the framework of social determinants of health indicates that material conditions confine one's access to (healthy) food and healthcare,[10 11] while also influencing health-related behaviours (ie, dietary behaviours and physical activity),[12] and psychosocial factors that derive from relative deprivation (eg, control over life, insecurity, social isolation, stress).[13–15]

In contrast, one important argument for reverse causality is stigma. Studies suggest that the obese are more likely to be perceived as lazy, unsuccessful, weak-willed

and undisciplined.[16–19] On basis of these negative stereotypes, the obese face various weight penalties in the labour market, which include higher job insecurity, lower chances for a job and general discrimination.[20–23] Furthermore, these stereotypes are also often internalised by those afflicted, which leads to self-stigma, reduced psychological resources as well as mental health problems.[24] By following these two frameworks, there are various pathways in which income relates to obesity and vice versa: with reference to the perspective of causation, income does not only restrict one's access to (healthy) food, but is also linked to higher health literacy which, in turn, is positively related to health-promoting behaviours (ie, healthy nutrition, physical activity).[12 25] Further, lower income is associated with higher levels of psychosocial stressors which include decreased control over life, and higher insecurity, social isolation, stress and mental disorders.[10 13–15] By attempting to integrate the stigma theory into the model of social determinants of health, in our interpretation, the stigmatisation of the obese also correlates with material (ie, less income through weight penalty), behavioural (ie, change in health-promoting behaviour through discrimination), as well as psychosocial factors (ie, self-stigma may inflict lower control over life, social isolation, stress, lower self-esteem) that may, again, lead to a higher risk of obesity.

This work builds on a former review that examined the relative importance of causation and reverse causality in the association between education and overweight/obesity.[25] Though education and income can be conceptualised under the broader term of the SES, specific dimensions of SES should not be regarded as interchangeable in their relation to obesity.[26] First, income rather influences material benefits for health, while education foremost relates to knowledge to gain or retain health. Second, income and education have a different importance across the life course, since educational attainment takes place during childhood and adolescence, while wages, earning and income are generally associated with the occupation in adulthood. For these reasons, income and education have different implications for public health.

This systematic review aims to assess both directions in the link of income and obesity, in order to address the relative importance of social causation processes and reverse causality in explaining the relationship.

METHODS

A systematic review of peer-reviewed studies that addressed the relationship between income and obesity was performed and completed in January 2017. To enhance the reproducibility of our findings, this review was conducted on the basis of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[27] A corresponding checklist is available online (see online supplementary file 1).

Medline and PsycINFO were chosen as the main health-related databases. Moreover, the sociological databases Sociological Abstracts, International Bibliography of Social Sciences and Sociological Index were considered.

For the search, the following equation was used: (adipos* OR obes* OR body-mass-index OR BMI OR 'waist-hip ratio' OR WHR OR 'skinfold thickness') AND ('social status' OR socioeconomic OR socioeconomic OR inequalit* OR income OR earning* OR wage*) AND (longitudinal OR prospective OR 'panel stud*' OR 'cohort stud*').

Inclusion and exclusion criteria

For inclusion, original studies had to be published in a peer-reviewed journal and contain quantitative data on the relation between income and obesity. Further initial restrictions (ie, language, publication years) were not considered.

Population

Studies were deemed eligible for inclusion if participants were part of the general population. Therefore, studies with a focus on specific population groups (ie, patient population) were excluded.

Intervention/exposure

On the one hand, studies were omitted if they did not explicitly focus on income and/or rather referred to global SES measures (eg, neighbourhood SES or SES index).

Control group

Regardless of the study's focus on either causation or reverse causality, a specific control group or non-exposed group (eg, people with higher income or non-obese participants) had to be provided to test the unique influence of an exposure (lower income or obesity).

Outcome

Studies that used overweight as their main outcome were excluded since obesity was found to be more predictive of health-related outcomes.[28 29] Therefore, studies were included if they focused on obesity, regardless of measurement (eg, BMI ≥30, age-specific and sex-specific percentiles, z-scores). For studies testing reverse causality, all types of outcomes associated with one's own income were included (eg, wages, earnings, household income).

Study design

Finally, only studies with a prospective design were considered, since a clear direction of causation or reverse causality can hardly be drawn from cross-sectional investigations.

In case of disagreements on inclusion or exclusion, respective records were discussed by the two reviewers (TJK and Nina Marie Roesler). If a consensus could not be achieved, a third reviewer (OK) was involved until an agreement was found.

Data extraction

Studies were assessed and the following study information retrieved: author(s), study name, country or region,

type of hypothesis, population type, sample size, age at baseline, gender proportion, measurement of income and obesity as well as length of observation, and adjusted covariates. In case multiple ascertainments of income were reported within a single study, the most appropriate measure was chosen. For instance, in studies with a children population, household income better describes the child's financial situation, while measures of parental, paternal and maternal income were the next best alternative measures, respectively. In studies with adults, however, personal wages were the most appropriate measure to characterise one's disadvantage on the labour market, followed by earnings and household income, respectively. TJK extracted the data and performed the meta-analyses.

Data analyses

Since most studies testing the causation hypothesis used ORs and corresponding 95% CIs in their analyses, all statistics were converted to log odds and SEs. In case ORs, log odds or SE were not readily available, effect sizes were estimated on basis of alternative statistics such as t value or P value and sample size. In contrast to the studies on social causation, which mostly referred to a binary outcome (obese vs non-obese), studies on reverse causality rather examined a continuous variable (income), mostly based on unstandardised regression coefficients. In order to provide a better comparability in the meta-analysis, these coefficients were transformed into standardised mean differences (SMD). For the meta-analyses of both hypotheses, effect sizes from fully adjusted models were taken, if available. Random-effect models were employed, and pooled estimates weighted with the restricted maximum likelihood estimator.[30] Cochran's Q test and Higgin's I^2 measure were calculated to evaluate the proportional degree of heterogeneity. Finally, stratified meta-analyses were run to reveal potential moderating effects (ie, study region, population type, measurement of obesity, gender, time lag between baseline and follow-up, and study quality).

To test impeding publication bias, Egger's regression test and the trim-and-fill-method were used.[31 32] The visualisation and calculation of effect sizes, pooled estimates, sensitivity analyses and publication bias were executed with R and the packages 'esc' and 'metafor'.[30 33]

Study quality

In order to assess the quality of non-randomised studies in meta-analyses, we referred to the Newcastle-Ottawa Scale for cohort studies (NOS).[34] The NOS includes a total of nine items across three dimensions (ie, sample selection, comparability of cohorts, the assessment of outcome). However, two of nine criteria could hardly be applied to studies testing the reverse causality hypothesis as they focused on an outcome that was explicitly non-health related. Therefore, the two questions: (1) if the outcome of interest was not present at start of study and (2) if the follow-up duration was long enough for the outcome to occur, were excluded to provide a better precision of the NOS checklist. The application of the NOS checklist was carried out by TJK and OK and discussed in case of divergences.

RESULTS

Literature search

The inclusion and exclusion of studies is shown in figure 1. Through the initial screening of all five databases, 3955 records were found. After removing duplicates,

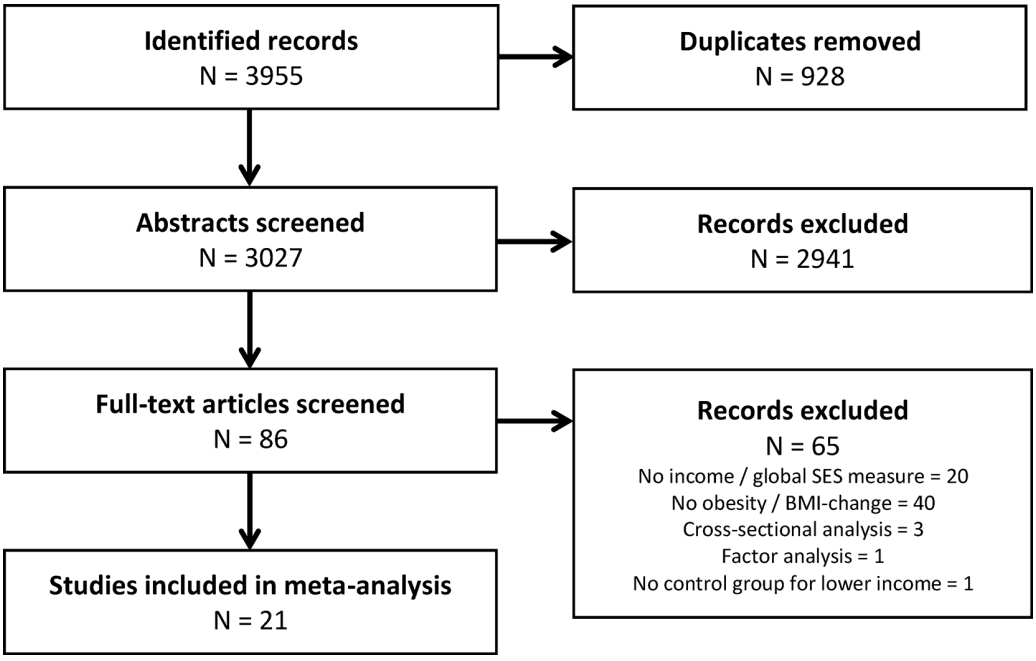

Figure 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow chart of included studies. BMI, body mass index; N, number of records; SES, socioeconomic status.

3027 titles and abstracts were screened for eligibility. Hereafter, another 2941 records were excluded. The full texts of the remaining 86 records were then screened for eligibility, from which 65 were dismissed. A detailed summary of reasons of exclusion is accessible online (see online supplementary file 2). Finally, 21 articles met all predefined inclusion criteria and were considered for meta-analysis. In an additional screening of the references of included studies, no further eligible records were found. Overall, 14 studies addressed the social causation and 7 the reverse causality hypothesis (see table 1 for an overview of the included studies). Information about the quality of the studies according to the NOS checklist is available online (see online supplementary file 3).

Studies testing the causation hypothesis

In 10 of the studies investigating the causation hypothesis, ORs were calculated, while in four studies risk ratios (RRs) were documented. Pooled estimates indicate the likelihood or risk for subsequent obesity among people with a low income compared with those having a high income (figure 2). Overall, results reveal a higher chance (OR 1.27, 95% CI 1.10 to 1.47) and an increased risk (RR 1.52, 95% CI 1.08 to 2.13) for obesity among low-income groups. Across studies referring to ORs, 4 of the 10 studies revealed statistically significant effects. In terms of RRs, two out of four were significant. None of the studies analysing causation indicated a positive relationship. For the 10 studies with ORs, a statistically significant publication bias was detected (see online supplementary file 4), Egger's regression test: z=5.0846, P<0.0001). After the imputation of studies to correct for publication bias, the OR decreased considerably and became statistically insignificant (adjusted OR 1.10, 95% CI 0.90 to 1.34). And though no publication bias could be detected for the four studies testing the causation hypothesis with RRs, an imputation of studies to adjust for potential publication bias yielded a decreased and statistically insignificant effect size for this meta-analysis (adjusted RR 1.16, 95% CI 0.73 to 1.82) (see online supplementary file 5).

Studies testing reverse causality

In seven studies that analysed the reverse causality hypothesis (figure 3), the pooled estimate (SMD), which expresses the size of the effect in each study relative to the variability observed, was −0.15 (95% CI −0.30 to 0.001), implying that people with obesity had a significantly lower income, when compared with the non-obese. This effect was statistically significant in five studies, while one study found a positive effect,[35] and one study revealed a relationship that was statistically insignificant.[36] Through the test for funnel plot asymmetry, no publication bias was detected. The effect sizes did not change after the imputation to adjust for publication bias (see online supplementary file 6).

Sensitivity analyses

In order to reveal potential moderating effects, stratified meta-analysis were performed (table 2). Sensitivity analyses showed that the majority of included studies were conducted in the USA (causation: 71%; reverse causality: 85%), whereas the only other study countries were the UK (causation: 21%) and Canada (causation: 7%; reverse causality 14%). Furthermore, the stratification for population revealed that causation mostly relied on children populations (79%), while studies on reverse causality exclusively focused on adults. The results for both region and population, however, remained fairly inconsistent, and did not reveal a clear trend.

According to studies on the causation hypothesis, higher effect sizes were evident when the height and weight of participants was actually measured instead of using a self-report (OR 1.48, 95% CI 1.04 to 2.10; RR 1.73, 95% CI 1.46 to 2.06), when the observation period exceeded 10 years (OR 1.59, 95% CI 1.11 to 2.27), and when the study quality was assessed as high (OR 1.40, 95% CI 1.06 to 1.83; RR 1.88, 95% CI 0.95 to 3.74). Subgroup analyses for gender could not be performed for studies analysing causation since gender-specific results were not documented.

In terms of the reverse causality hypothesis, the subgroup analysis of gender showed that the relation between obesity and subsequent income was more pronounced among women (SMD −0.16, 95% CI −0.30 to 0.02) than men (SMD −0.07, 95% CI −0.16 to 0.01). Similar to the studies on causation, it was shown that a longer observation period (>10 years) was associated with an effect size increase (SMD −0.52, 95% CI −0.62 to 0.41). The same pattern was found in the rating of higher study quality (SMD −0.52, 95% CI −0.62 to 0.41).

Heterogeneity between studies

With reference to figures 2 and 3, degrees of heterogeneity were relatively high in both meta-analyses that tested the causation hypothesis (I^2=89.9% and 83.1%) and studies that referred to reverse causality between income and obesity (I^2=98.5%). This furthermore indicates that the observed variance between studies is more likely to occur due to heterogeneity than chance alone (figures 1 and 2). High degrees of heterogeneity were also observed within most subgroup analyses (table 2).

DISCUSSION

Main findings

The results of this review revealed statistically significant effects of income on obesity (social causation) as well as of obesity on income (reverse causality). Therefore, individuals exposed to lower income are more likely to develop obesity, and the obese have lower wages when compared with their non-obese counterparts. However, after adjustments for publication bias, only the reverse causality hypothesis remained significant, whereas the meta-analytical association between lower income and subsequent

Table 1 Description of included studies

Author, study	Country, region	Direction	Population	Sample size	Age at baseline	Gender (male %)	Income measure	Obesity measure	Follow-up duration	Covariates in multivariate analysis
Brophy *et al*[40] MCS	UK (national)	Causation	Children	17 561	5 (mean)	NA	Income	95th BMI-percentile	4 years	Ethnic group, birth weight, enjoyment of physical activity, sedentary behaviour (watching TV), indoor activities, early introduction of solid food, smoking near child, mothers prepregnancy weight, education.
Chaffee *et al*[41] NLSY79	USA (national)	Causation	Women	4780	40 (mean)	0	Household income	BMI ≥30	31 years	Birth outside the USA, urban residence as a child, and residence in the South as a child, maternal variables (age, marital status, smoking during pregnancy, educational attainment, pregnancy BMI, previous excessive/inadequate gestational weight gain).
Chia[42] NLSY79	USA (national)	Causation	Children	3958	8.6 (mean)	51.3	Family income	95th BMI-percentile	6 years	Mother's characteristics (education, armed forces qualification test, age at birth of child, health limitations, migration status, marital status, overweight/obesity, living with both parents at age 14), child's characteristics (age, gender, region of residence, birth weight, firstborn status, race, breast feeding), household size.
Demment *et al*[43] BMHP1	USA (New York state)	Causation	Children	595	2 (mean)	53.0	Family income	BMI z-scores	16 years 2 years	Mother's age at time of delivery, multiparty, maternal overweight/obesity, child's characteristics (birth weight, sex, ADHD medication use, asthma medication use, antidepressant medication use, puberty status, early life rapid weight gain).

Continued

Table 1 Continued

Author, study	Country, region	Direction	Population	Sample size	Age at baseline	Gender (male %)	Income measure	Obesity measure	Follow-up duration	Covariates in multivariate analysis
Goisis *et al*[44] MCS	UK (national)	Causation	Children	11 965	5 (mean)	50.8	Family income	95th BMI-percentile	8 years	Mother smoking during pregnancy, length of breast feeding, maternal BMI, early introduction to solid foods, child's gender, physical activity (frequency of sport, active playing with parent, use of a playground, use of a bike), sedentary behaviour (watching TV, PC use), bedtime, fruit portion per day, skipping breakfast, sweet drinks consumption.
Hoyt *et al*[45] CYGNET	USA (national)	Causation	Girls	174	8–10 (range)	0	Household income	95th BMI-percentile	4 year	Race/ethnicity, baseline BMI, puberty status, year of outcome measure, no of street segments household size, education (of financial provider), neighbourhood SES, food and service retail scale.
Jo[46] ECLS-K	USA (national)	Causation	Children	9287	5.9 (mean)	0.51	Family income	95th BMI-percentile	9 years	Grade level, race, gender, household size, mother's age, father's age, school lunch, school fixed effects.
Kakinami *et al*[47] QLSCD	Canada (Québec)	Causation	Children	698	9.2 (mean)	45.6	Household income	85th BMI-percentile	12 years	Child's birth weight and sex, mother's education and migration status.
Kim and Leigh[48] PSID	USA (national)	Causation	Adults	6312	41.9 (mean)	0.85	Log hourly wage	BMI ≥30	4 years	Age, sex, race, marital status, education, health insurance, smoking, region of residence, survey year.
Lee *et al*[49] Add health	USA (national)	Causation	Adolescents	9730	12–19 (range)	49.2	Poverty status	BMI ≥30	7 years	Age, low parental education, family structure, trouble paying bills, neighbourhood poverty, parental monitoring (watching TV, eating dinner, low-parent-child interaction, no curfew, full-time working mother), physical activity, skipping breakfast, inadequate sleep, race/ethnicity, parent obesity status.

Continued

Table 1 Continued

Author, study	Country, region	Direction	Population	Sample size	Age at baseline	Gender (male %)	Income measure	Obesity measure	Follow-up duration	Covariates in multivariate analysis
Lee *et al*[50] SECCYD	USA (national)	Causation	Children, adolescents	1150	3–15 (range)	50.7	Family income	95th BMI-percentile	15 years	Age, poverty status lagged, sex, race/ethnicity, birth weight, maternal variables: age, education, figure rating scale score, marital status lagged.
Pearce *et al*[51] NCMP, MCS	UK (national)	Causation	Children	2 620 422	3–7 (range)	51.2	Household income	95th BMI-percentile	4 years	Maternal education, area deprivation, maternal social class.
Salsberry and Reagan[52] NLSY79	USA (national)	Causation	Young women	3707	14–21 (range)	0	Income	BMI ≥30	33 years	Age, parental education, own education.
Strauss and Knight[53] NLSY	USA (national)	Causation	Children	2913	0–8 (range)	56.0	Family income	95th BMI-percentile	6 years	Maternal BMI, initial weight-for-height z-score, gender, race, maternal education, marital status, cognitive score, emotional score.
Amis *et al*[54] Add health	USA (national)	Reverse causality	Adolescents	11 308	16 (mean)	47.2	Annual income	95th BMI percentile	13 years	Age, sex, race, no of siblings, mother's education, mother works, father works, closeness to mother, closeness to father, school skipped, grade repeated, attention problem, watching TV (hours), playing sports, playing computer games, hanging out with friends, type of school, neighbourhood environment, mental health, general health, smoking, alcohol use, drug use, ever had sex.
Baum and Ford[55] NLSY	USA (national)	Reverse causality	Young adults	51 500 (PY)	28–31 (range)	51.7	Log real wage	BMI ≥30	17 years	Race, age, education, marital status, no of children, human capital accumulation, area of residence, local unemployment rate, industry working in, AFQT score (Armed Forces Qualifying Test), migration status, speaking foreign language, mother's education, father's education, siblings, rotter test score (efficacy), attitudes about family roles, health limitations, At age 14: lived with both parents, received magazines, received newspaper, library card, area of residence, mother worked.

Continued

Table 1 Continued

Author, study	Country, region	Direction	Population	Sample size	Age at baseline	Gender (male %)	Income measure	Obesity measure	Follow-up duration	Covariates in multivariate analysis
Cawley and Danziger[56] WES	USA (national)	Reverse causality	Women	874	18–54 (range)	0	Earnings	BMI ≥30	6 years	No of children the respondent cares for, the no of children between the ages of 0 and 2 that the respondent cares for, indicator variables for no job market skills, low job market skills, less than a high school education, more than a high school education, one of the respondent's children has a physical or mental health problem, respondent is currently cohabitating with a husband or boyfriend, never married, age, wave 3, wave 4, respondent has a conviction for other than a traffic offence, and respondent has a learning disability.
Conley *et al*[57] PSID	USA (national)	Reverse causality	Adults	3340	46–49 (range)	46.5	Log wages	BMI ≥30	18 years	Educational attainment, labour market experience, age of youngest child and age.
Han *et al*[36] NLSY79	USA (national)	Reverse causality	Adolescents	1974	16–20 (range)	54.1	Hourly wage	BMI ≥30	12 years	Age, race, marital status, time from latest pregnancy to the interview, education of the parents, AFQT score, self-esteem, years of employment, participated in on-the-job training, area of residence, unemployment rate in the residential unit, no of private businesses at state level, average income by state, consumer price index, education, occupation, occupation requiring social interaction.
Larose *et al*[35] NPHS	Canada (national)	Reverse causality	Adults	3993	40.2 (mean)	50.71	Hourly wage rate	BMI ≥30	6 years	Age, presence of small children in the household, migration status, area of residence, marital status, non-wage/spouse income, home ownership, education, smoking behaviour, drinking behaviour.

Continued

Table 1 Continued

Author, study	Country, region	Direction	Population	Sample size	Age at baseline	Gender (male %)	Income measure	Obesity measure	Follow-up duration	Covariates in multivariate analysis
Mason[37] NLSY97	USA (national)	Reverse causality	Young adults	2427	12–17 (range)	50.72	Income	BMI ≥30	9 years	Education, parental status, work experience, occupation, race, socioeconomic background (1997), household income, mother's education, father's education), health limitations, (Armed Services Vocational Aptitude Battery).

Add Health, National Longitudinal Study of Adolescent to Adult Health; ADHD, Attention Deficit Hyperactivity Disorder; BMHP1, Bassett Mothers Health Project; BMI, body mass index; Cygnet Study, Cohort Study of Young Girls Nutrition, Environment and Transitions; ECLS-K, Early Childhood Longitudinal Study Kindergarten; MCS, Millennium Cohort Study; NA, not available; NCMP, National Child Measurement Programme; NLSY (97), US National Longitudinal Survey of Youth (1997); NPHS, Canadian National Population Health Survey; PSID, Panel Study of Income Dynamics; PY, person-years; QLSCD, Québec Longitudinal Study of Child Development; SECCYD, Study of Early Child Care and Youth Development; SES, socioeconomic status; WES, Women's Employment Study.

risk of obesity vanished and became inconclusive. These findings indicate that studies testing the social causation hypothesis are more likely to remain unpublished if they contain negative results. In order to explain why this especially applies to studies testing the causation hypothesis, we assume that it is difficult to publish negative results, since the relation between income and the risk of subsequent obesity has been well established in social epidemiological and public health research. In contrast, evidence for reverse causality is relatively scarce for the relation between obesity and income, which may explain the higher chances to get negative results published in this field. Though the overall effect size for the social causation perspective became statistically insignificant after the imputation of studies, it is still noteworthy that there were some studies that found statistically significant associations, even after adjusting for a range of covariates (table 1).

According to the sensitivity analyses, it was shown that reverse causality was more pronounced among women than among men. To explain these differences, Mason, for instance, suggests that obese women are confronted with disadvantages that derive from the stigmatisation of fatness, and additionally face higher expectations to perform their gender properly.[37] According the cultivation theory of the social sciences, there is a stronger idealisation of thin women, which may help to explain why there is a stricter weight penalty for women than for men.[38] With regards to the ascertainment of obesity in studies, sensitivity analyses revealed that effects of social causation were stronger when height and weight of respondents were actually measured rather than based on self-reports. Similar to results from nutritional studies, where a gender-specific social desirability bias was evident in self-reports of dietary intake,[39] a comparable bias in height and weight reports can be suspected for our results. Therefore, as actual measurements can be considered as less biased, if compared with self-reports, it can be assumed that the overall effect of income on obesity is underestimated when self-reported measures are used. All studies investigating reverse causality were based on self-reported measures. As effect sizes were generally stronger in studies with higher quality scores (if compared with studies with a medium or low rating, regardless of causation or reverse causality), the overall effects can be expected to be somewhat stronger than indicated through the meta-analytic results.

Limitations

Some methodological issues should be considered when interpreting the findings of this meta-analysis. First, the risk for missing out other relevant articles remains. Second, all included studies have their origin in western societies, most in the USA. Therefore, the evidence is restricted to a few countries, leaving out the possibility that the relationship between income and obesity plays out differently in other regions of the world. Third, and though only studies were included that examined the relation between income and

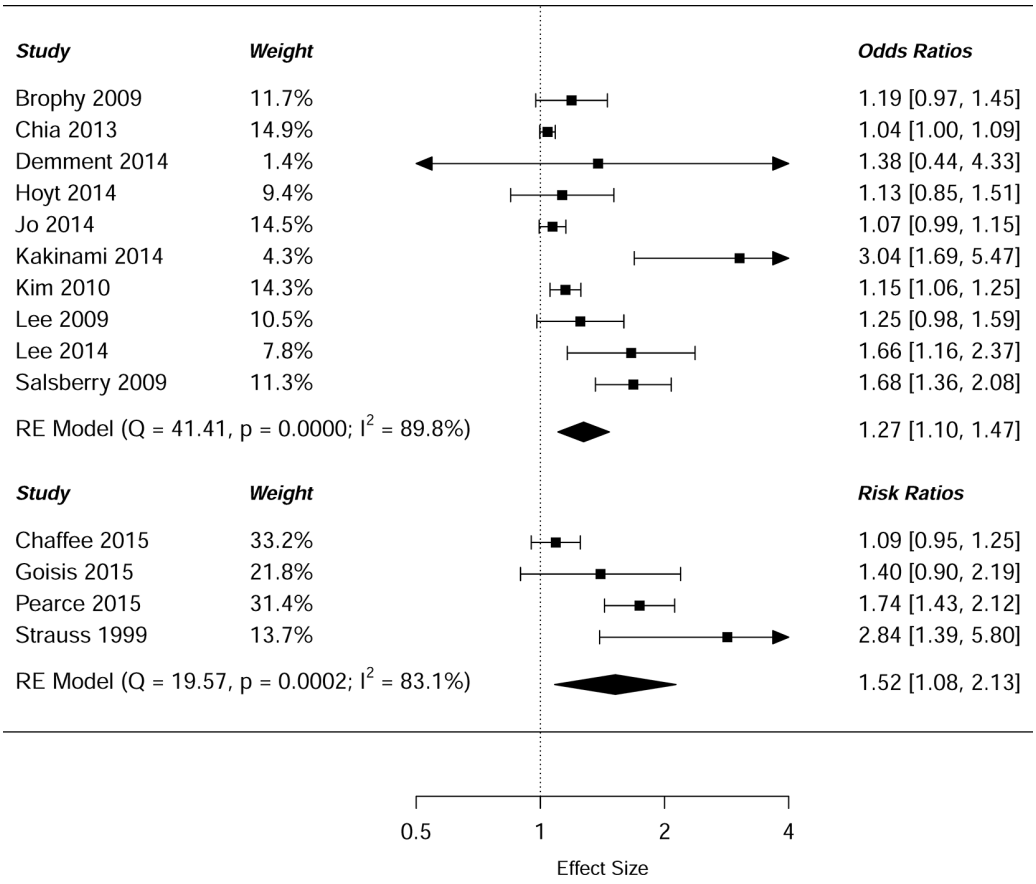

Figure 2 Pooled estimates of studies testing the causation hypothesis. RE, random effects.

obesity longitudinally (thus enabling to carve out the direction of the respective influence), the question of causality cannot be fully answered due to two main reasons. On the one hand, the methodological issue remains that (cohort) studies are not able to adjust for transitions between the individual income status as well as the obesity status that take place between the baseline and the follow-up survey. Thus, the results of longitudinal observational studies

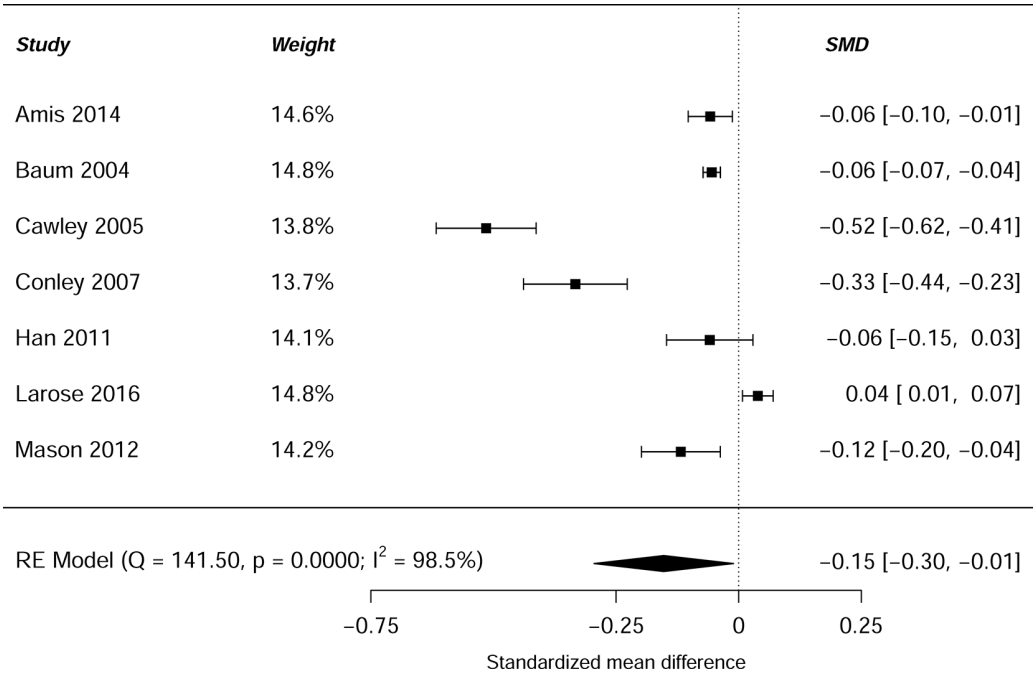

Figure 3 Pooled estimates of studies testing the reverse causality hypothesis. RE, random effect; SMD, Standardised mean difference.

Table 2 Sensitivity analyses

	Social causation hypothesis				Reverse causality hypothesis	
	n	OR (CI), I^2	n	RR (CI), I^2	n	SMD (CI), I^2
Overall	10	1.27 (1.10 to 1.47), 90%	4	1.52 (1.08 to 2.13), 83%	7	−0.15 (−0.30 to 0.01), 98%
Study region						
USA	8	1.22 (1.06 to 1.40), 88%	2	1.65 (0.65 to 4.17), 85%	6	−0.19 (−0.34 to 0.03), 97%
UK	1	1.19 (0.97 to 1.45), NA	2	1.68 (1.40 to 2.01), 0%	–	–
Canada	1	3.04 (1.69 to 5.47), NA	–	–	1	0.04 (0.01 to 0.07), NA
Population						
Children	8	1.33 (1.08 to 1.64), 93%	3	1.73 (1.46 to 2.06), 0%	–	–
Adolescents	1	1.25 (0.98 to 1.59), NA	1	1.09 (0.95 to 1.25), NA	–	–
Adults	1	1.15 (1.06 to 1.25), NA	–	–	7	−0.15 (−0.30 to 0.01), 98%
Obesity						
Self-report	5	1.20 (1.03 to 1.41), 88%	1	1.09 (0.95 to 1.25), NA	7	−0.15 (−0.30 to 0.01), 98%
Measured	5	1.48 (1.04 to 2.10), 84%	3	1.73 (1.46 to 2.06), 0%	–	–
Gender						
Male	–	–	–	–	6	−0.07 (−0.16 to 0.01), 90%
Female	–	–	–	–	7	−0.16 (−0.30 to 0.02), 93%
Observation						
<5 years	3	1.15 (1.07 to 1.25), 0%	1	1.74 (1.43 to 2.12), NA	1	−0.06 (−0.10 to 0.01), NA
5–10 years	2	1.11 (0.98 to 1.25), 30%	2	1.88 (0.95 to 3.74), 63%	5	−0.10 (−0.22 to 0.02), 97%
>10 years	5	1.59 (1.11 to 2.27), 88%	1	1.09 (0.95 to 1.25), NA	1	−0.52 (−0.62 to 0.41), NA
Study quality						
Low	3	1.09 (1.00 to 1.18), 57%	1	1.09 (0.95 to 1.25), NA	1	−0.06 (−0.10 to 0.01), NA
Medium	5	1.47 (1.04 to 2.08), 90%	1	1.74 (1.43 to 2.12), NA	5	−0.10 (−0.22 to 0.02), 97%
High	2	1.40 (1.06 to 1.83), 40%	2	1.88 (0.95 to 3.74), 63%	1	−0.52 (−0.62 to 0.41), NA

n, number of studies; NA, not available; RR, risk ratios; SMD, standardised mean difference.

should be regarded as a mere tendency, and must be interpreted with caution. On the other hand, there is reason to believe that processes of social causation are not simply replaced by reverse causality during the life course, but rather coexist in one's biography. As noted above, it can be assumed that causation processes and reverse causality rather augment than neutralise each other, so that the link between income and obesity is likely to follow a bidirectional relationship. Fourth, a further limitation of studies testing the causation hypothesis is the heterogeneity of the low-income control groups. In this regard, the reference groups varied substantially, which limits the comparability between different income measures. In terms of studies testing the reverse causality, comparability was not as problematic as all studies referred to a non-obese reference that was defined by a BMI lower than 30. Fifth, and even though the subgroup analyses revealed a few factors that may moderate the relationship between income and obesity, results for the reverse causality hypothesis were mostly based on the same subgroups. Finally, the capacity of Egger's regression test to detect publication bias depends on the number of included studies.[32] Therefore, there is a clear lack of statistical power in Egger's regression test of studies that used RRs (n=4), which has to be viewed as a further limitation of this study. In addition, the trim-and-fill method of these studies led to a substantially decreased and statistically insignificant effect size (adjusted RR 1.10, 95% CI 0.90 to 1.34), further implying the inconsistencies of results testing the causation hypothesis.

Future research

By taking these limitations into account, future studies should aim at investigating the relation between income and obesity outside of western societies. An extended view on the association in other countries could aid in detecting cultural influences that frame the magnitude of both causation processes and reverse causality between income and obesity. Moreover, and in order to clarify potential bidirectional effects between income and obesity, future research should investigate the interaction between causation processes and reverse causality in a single cohort over the life course. Finally, future studies could focus on detecting other factors that may influence the relation between income and obesity for both hypotheses.

CONCLUSIONS

This review was performed to give an overview of causation processes in the link between income and obesity, while also investigating a reverse causality between these two variables. Meta-analyses revealed significant links between lower income and the risk of obesity as well as obesity and subsequent income (reverse causality hypothesis). However, after adjusting for publication bias, the relation between lower income and the risk of subsequent obesity vanished, indicating a higher likelihood of unpublished studies due to negative findings. In contrast, results from studies testing the reverse causality perspective remained consistent even after adjusting for potential publication bias. Therefore, a stronger consideration of potential reverse causality is needed to address income-related inequalities in obesity.

Acknowledgements The authors would like to thank Nina Marie Roesler for helping with the literature search and the screening.

Contributors TJK and OvdK developed the research question. TJK then conducted the literature search, screened all found records and extracted the relevant data. TJK performed the meta-analysis and wrote the first draft of the manuscript. OvdK revised the manuscript. Both authors read and approved the final version of the manuscript.

Funding This study is part of the joint research project 'Nutrition, Health and Modern Society: Germany and the USA' and is funded by the Volkswagen Foundation.

Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement We retrieved all data for the meta-analyses from already published material. Therefore, the data are available in the respective articles.

REFERENCES

1. Di Cesare M, Bentham J, Stevens GA, *et al*. Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19·2 million participants. *Lancet* 2016;387:1377–96.
2. Flegal KM, Kit BK, Orpana H, *et al*. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA* 2013;309:71–82.
3. Lenz M, Richter T, Mühlhauser I. The morbidity and mortality associated with overweight and obesity in adulthood: a systematic review. *Dtsch Arztebl Int* 2009;106:641–8.
4. Peeters A, Barendregt JJ, Willekens F, *et al*. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* 2003;138:24–32.
5. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. *Biol Psychiatry* 2003;54:330–7.
6. Sturm R, Wells KB. Does obesity contribute as much to morbidity as poverty or smoking? *Public Health* 2001;115:229–35.
7. Dinsa GD, Goryakin Y, Fumagalli E, *et al*. Obesity and socioeconomic status in developing countries: a systematic review. *Obes Rev* 2012;13:1067–79.
8. McLaren L. Socioeconomic status and obesity. *Epidemiol Rev* 2007;29:29–48.
9. Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 1989;105:260–75.
10. Marmot M. The influence of income on health: views of an epidemiologist. *Health Aff* 2002;21:31–46.
11. Reidpath DD, Burns C, Garrard J, *et al*. An ecological study of the relationship between social and environmental determinants of obesity. *Health Place* 2002;8:141–5.
12. Laaksonen M, Prättälä R, Helasoja V, *et al*. Income and health behaviours. Evidence from monitoring surveys among Finnish adults. *J Epidemiol Community Health* 2003;57:711–7.
13. Marmot M. Social determinants of health inequalities. *Lancet* 2005;365:1099–104.
14. Marmot M, Wilkinson RG. Psychosocial and material pathways in the relation between income and health: a response to Lynch et al. *BMJ* 2001;322:1233–6.
15. Moore CJ, Cunningham SA. Social position, psychological stress, and obesity: a systematic review. *J Acad Nutr Diet* 2012;112:518–26.
16. Puhl RM, Heuer CA. Obesity stigma: important considerations for public health. *Am J Public Health* 2010;100:1019–28.
17. Roehling M. Weight-based discrimination in employment: psychological and legal aspects. *Pers Psychol* 1999;52:969–1016.
18. Puhl RM, Brownell KD. Psychosocial origins of obesity stigma: toward changing a powerful and pervasive bias. *Obes Rev* 2003;4:213–27.
19. Puhl RM, Heuer CA. The stigma of obesity: a review and update. *Obesity* 2009;17:941–64.
20. Caliendo M, Gehrsitz M. Obesity and the labor market: a fresh look at the weight penalty. *Econ Hum Biol* 2016;23:209–25.
21. Muenster E, Rueger H, Ochsmann E, *et al*. Association between overweight, obesity and self-perceived job insecurity in German employees. *BMC Public Health* 2011;11:162.
22. Giel KE, Zipfel S, Alizadeh M, *et al*. Stigmatization of obese individuals by human resource professionals: an experimental study. *BMC Public Health* 2012;12:525.
23. Daniels J. Weight and weight concerns: are they associated with reported depressive symptoms in adolescents? *J Pediatr Health Care* 2005;19:33–41.
24. Nowrouzi B, *et al*. Weight Bias in the workplace: a literature review. *Occup Med Health Aff* 2015;3:206.
25. Kim TJ, Roesler NM, von dem Knesebeck O. Causation or selection – examining the relation between education and overweight/obesity in prospective observational studies: a meta-analysis. *Obes Rev* 2017;18:660–72.
26. Geyer S, Hemström O, Peter R, *et al*. 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.
27. Moher D, Liberati A, Tetzlaff J, *et al*. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med* 2009;6:e1000097.
28. Swallen KC, Reither EN, Haas SA, *et al*. Overweight, obesity, and health-related quality of life among adolescents: the National Longitudinal Study of Adolescent Health. *Pediatrics* 2005;115:340–7.
29. Afzal S, Tybjaerg-Hansen A, Jensen GB, *et al*. Change in body mass index associated with lowest mortality in Denmark, 1976–2013. *JAMA* 1989;2016:315.
30. Viechtbauer W. Conducting meta-analyses in R with the metafor package. *J Stat Softw* 2010;36:1–48.
31. Duval S, Tweedie R. Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics* 2000;56:455–63.
32. Egger M, Davey Smith G, Schneider M, *et al*. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 1997;315:629–34.
33. Lüdecke D. Esc: effect size computation for meta-analysis. https://cran.r-project.org/package=esc (accessed Mar 2017).
34. Wells G, Shea B, O'Connell D, *et al*. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp (accessed Mar 2017).
35. Larose SL, Kpelitse KA, Campbell MK, *et al*. Does obesity influence labour market outcomes among working-age adults? Evidence from Canadian longitudinal data. *Econ Hum Biol* 2016;20:26–41.
36. Han E, Norton EC, Powell LM. Direct and indirect effects of body weight on adult wages. *Econ Hum Biol* 2011;9:381–92.
37. Mason K. The Unequal Weight of Discrimination: Gender, body Size, and income inequality. *Soc Probl* 2012;59:411–35.
38. Tyrrell J, Jones SE, Beaumont R, *et al*. Height, body mass index, and socioeconomic status: mendelian randomisation study in UK Biobank. *BMJ* 2016;352:i582.

39. Hebert JR, Ma Y, Clemow L, *et al*. Gender differences in social desirability and social approval bias in dietary self-report. *Am J Epidemiol* 1997;146:1046–55.
40. Brophy S, Cooksey R, Gravenor MB, *et al*. Risk factors for childhood obesity at age 5: analysis of the millennium cohort study. *BMC Public Health* 2009;9:467.
41. Chaffee BW, Abrams B, Cohen AK, *et al*. Socioeconomic disadvantage in childhood as a predictor of excessive gestational weight gain and obesity in midlife adulthood. *Emerg Themes Epidemiol* 2015;12:4.
42. Chia YF. Dollars and pounds: the impact of family income on childhood weight. *Appl Econ* 2013;45:1931–41.
43. Demment MM, Haas JD, Olson CM. Changes in family income status and the development of overweight and obesity from 2 to 15 years: a longitudinal study. *BMC Public Health* 2014;14:417.
44. Goisis A, Sacker A, Kelly Y. Why are poorer children at higher risk of obesity and overweight? A UK cohort study. *Eur J Public Health* 2016;26:7–13.
45. Hoyt LT, Kushi LH, Leung CW, *et al*. Neighborhood influences on girls' obesity risk across the transition to adolescence. *Pediatrics* 2014;134:942–9.
46. Jo Y. What money can buy: family income and childhood obesity. *Econ Hum Biol* 2014;15:1–12.
47. Kakinami L, Séguin L, Lambert M, *et al*. Poverty's latent effect on adiposity during childhood: evidence from a Québec birth cohort. *J Epidemiol Community Health* 2014;68:239–45.
48. Kim D, Leigh JP. Estimating the effects of wages on obesity. *J Occup Environ Med* 2010;52:495–500.
49. Lee H, Harris KM, Gordon-Larsen P. Life course perspectives on the links between poverty and obesity during the transition to young adulthood. *Popul Res Policy Rev* 2009;28:505–32.
50. Lee H, Andrew M, Gebremariam A, *et al*. Longitudinal associations between poverty and obesity from birth through adolescence. *Am J Public Health* 2014;104:e70–6.
51. Pearce A, Rougeaux E, Law C. Disadvantaged children at greater relative risk of thinness (as well as obesity): a secondary data analysis of the England National Child Measurement Programme and the UK Millennium Cohort Study. *Int J Equity Health* 2015;14:61.
52. Salsberry PJ, Reagan PB. Comparing the influence of childhood and adult economic status on midlife obesity in Mexican American, white, and African American women. *Public Health Nurs* 2009;26:14–22.
53. Strauss RS, Knight J. Influence of the home environment on the development of obesity in children. *Pediatrics* 1999;103:e85.
54. Amis JM, Hussey A, Okunade AA. Adolescent obesity, educational attainment and adult earnings. *Appl Econ Lett* 2014;21:945–50.
55. Baum CL, Ford WF. The wage effects of obesity: a longitudinal study. *Health Econ* 2004;13:885–99.
56. Cawley J, Danziger S. Morbid obesity and the transition from welfare to work. *J Policy Anal Manage* 2005;24:727–43.
57. Conley D, Glauber R. Gender, body mass, and socioeconomic status: new evidence from the PSID. *Adv Health Econ Health Serv Res* 2007;17:253–75.