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# Short sleep and obesity in a large national cohort of Thai adults

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## ABSTRACT

**Objective:** To investigate the relationship between short sleep and obesity among Thai adults.

**Design:** Both 4-year longitudinal and cross-sectional analyses of a large national cohort.

**Setting:** Thai adults residing nationwide from 2005 to 2009.

**Participants:** Cohort members were enrolled as distance learners at Sukhothai Thammathirat Open University (N=87 134 in 2005 and 60 569 at 2009 follow-up). At 2005 baseline, 95% were between 20 and 49 years of age.

**Measures:** Self-reported sleep duration was categorised as <6, 6, 7, 8 and ≥9 h. For all analyses (2005 and 2009 cross-sectional and 2005–2009 longitudinal), we used multinomial logistic regression models to assess the effect of sleep duration on abnormal body size (underweight, overweight-at-risk, obese). Results were adjusted for an array of relevant covariates.

**Results:** At the last cohort follow-up in 2009, cross-sectional associations linked short sleep (<6 h) and obesity: adjusted ORs (AOR) =1.49, 95% CIs 1.32 to 1.68 for women and AOR=1.36, 95% CI 1.21 to 1.52 for men. The earlier cross-sectional baseline results in 2005 were quite similar. Longitudinal analysis (2005–2009) of 4-year incremental weight gain (5 to <10%, 10 to <20% and 20%+) strongly supported the short sleep–obesity relationship (significant AORs of 1.10, 1.30 and 1.69, respectively).

**Conclusions:** The results are internally consistent (2005 and 2009) and longitudinally confirmatory of a short sleep effect on obesity among Thai adults. Further research is needed to elucidate causal mechanisms underlying the sleep–obesity relationship.

## ARTICLE SUMMARY

### Article focus

- The association between sleep duration and obesity for adults is controversial and has rarely been examined in middle-income countries.
- The evidence for a short sleep–obesity effect in adults is mostly based on cross-sectional data.
- This study examines both cross-sectional and longitudinal evidence linking short sleep and obesity among a large national cohort of Thai adults.

### Key messages

- Our cohort study revealed a consistent and substantial association between short sleep and obesity among Thai adults.
- The ORs connecting short sleep to obesity were substantial and significant: 1.36 for men and 1.49 for women in 2009; corresponding ORs were similar in 2005.
- Longitudinal 2004–2009 cohort data confirmed a significant short sleep–weight gain relationship with a notable dose–response.

### Strengths and limitations of this study

- The strength of this study was the large sample, longitudinal data, involvement of young adults and the opportunity to control for many covariates in the analyses.
- One limitation was the subjective nature of self-reporting sleep duration, a problem noted by many others conducting sleep research.

## INTRODUCTION

Short sleep has increased in prevalence over the last century[1–3] and may now be contributing to the obesity epidemic. A causal association is most accepted for children and adolescents.[4 5] Evidence remains inconclusive for adults.[6–10] But recent research has shown that short sleeping young adults are at increased risk of obesity and of pre-diabetic levels of glycosylated haemoglobin.[11] And many reports suggest potential biological mechanisms linking short sleep and obesity, including obstructive sleep apnoea, insulin resistance, stress, high levels of cortisol and ghrelin, low levels of leptin and adiponectin and disordered thermoregulation.[12–18]

The evidence for a short sleep–obesity effect in adults is mostly based on cross-sectional data.[7–9] However, two large prospective adult cohort studies in the USA report substantive longitudinal weight gain associated with short sleep.[19 20] Other supportive longitudinal evidence of a short sleep–obesity effect in prospective cohort studies was found among adults in Canada,[21] young adults in Switzerland[22] and older adults in Spain.[23] There have been very few longitudinal studies of adults in Asia but one retrospective cohort

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in Japan[24] and another study of serial population panels in Korea[25] both show consistent and significant associations between short sleep and obesity.

We examine short sleep and obesity in our large national cohort study of 87 134 Thai adults. Our report is based on a longitudinal analysis for the 4-year period from 2005 to 2009 and on cross-sectional analyses at baseline (2005) and follow-up (2009). We investigated the association between sleep duration and weight, adjusting for a wide array of potential confounders.

## METHODS AND ANALYSIS

This study is part of an overarching research project entitled *The Thai Health-Risk Transition: a National Cohort Study*, which is examining the ongoing transition from traditional to modern patterns of risk and disease. In 2005, the study recruited 87 134 distance learning adult students enrolled at the Sukhothai Thammathirat Open University and residing nationwide.[26] The 20-page 2005 baseline questionnaire covered socio-demographic characteristics, self-reported height and weight (validated),[27] personal environment, health behaviours, injury and health outcomes.

The Sukhothai Thammathirat Open University cohort is representative of the geodemographic, ethnic composition and income and household assets of the adult Thai population. Based on the results of the 2000 Population and Housing Survey, the median age was 29.2 years for the Thai population and 29.0 years among cohort members, and 51% of the Thai population were women compared with 54% of cohort members.[26 28 29] The follow-up study in 2009 reached 60 569 cohort members (70% response rate) and the age–sex and geographical distribution of respondents remained almost identical to the baseline.[30]

For body mass index (BMI), we used Asian cut-offs in accordance with studies in other Asian populations based on the International Obesity Task Force.[31] At baseline in 2005, 95% of cohort members were aged between 20 and 49 years. Men were twice as likely as women to be overweight (21% vs 9%) and obese (23% vs 10%).[32] Obesity associated with higher incomes for men and lower incomes for women.[33] The distribution of BMI by age and sex did not change much by follow-up in 2009.

Sleep duration was measured directly by asking: "How many hours per day do you sleep (including during the day)," categorised as <6, 6, 7, 8 and ≥9 h. For both 2005 and 2009, we used multinomial logistic regression models to assess the effect of sleep duration on the outcome of abnormal body size (underweight, overweight-at-risk and obese). Thus for short sleepers and normal sleepers, the relative odds for each 'abnormal' weight category versus normal were computed and adjusted for covariates (see below). We also used multinomial adjusted logistic regression to model the longitudinal 4-year incidence of weight gain in three increment categories (see the Results section).

Covariates adjusted in all models included age in years, marital status (married, single and separated/widowed), personal income categories (baht/month), rural–urban geographical residence, self-reported health risk behaviour including smoking (never, current and previous) or drinking (days/week), fruit and vegetable intakes (serves/day), vigorous or moderate physical activity (sessions/week), screen time (hours/day), doctor-diagnosed depression and doctor-diagnosed chronic disorders including type I and type II diabetes, high cholesterol, high blood pressure, heart disease, stroke, cancers (liver, lung, stomach, colon, breast and others), goitre, epilepsy, liver disease, lung disease, arthritis and asthma. These covariates were chosen based on our experience with risk factors of obesity in our cohort[32–34] as well as international literature.[35 36]

We analysed men and women separately as our data show the occurrence of abnormal body size, and the socioeconomic associations vary by sex.[32 33] For data scanning and editing, we used Thai Scandevet, SQL and SPSS software. For analysis, we used SPSS V.16 and Stata V.10. Individuals with missing data (<10%) were excluded from multivariable analyses.

## RESULTS

We present the most recent cross-sectional results (2009) and the longitudinal results for 2005–2009 data. The 2005 cross-sectional data were analysed, but results are not shown because they were very similar to 2009. At the follow-up in 2009, cohort weight results were as follows: 9.5% underweight (BMI <18.5), 48.5% normal (18.5 to <23), 18.4% overweight-at-risk (23 to <25) and 21.7% obese (≥25). Underweight was most common among women aged between 20 and 29 years (23.5%), while overweight-at-risk and obesity were most common among men aged ≥45 years (28.2% and 40.3%).

Among the cohort members responding in 2009, there is a parallel trend between increasing BMI and older age, being married and higher income (table 1). Overweight and obesity increased in frequency with less than four daily serves of fruits and vegetables (especially among men), less than three physical activity sessions per week (especially among women) and >5 h of screen time per day (both for men and women). Increased BMI also was more prevalent among those with doctor-diagnosed chronic conditions.

In 2009, 15% of the cohort reported short sleep duration (<6 h/day) (table 2) and one-fourth and one-third of cohort members reported sleeping 7 or 8 h daily. Overweight-at-risk was associated with short sleep among women (adjusted ORs (AOR) =1.33, 95% CI 1.18 to 1.51). Obesity had a stronger association with short sleep for women (AOR=1.49, 95% CI 1.32 to 1.68) but was also associated with short sleep for men (AOR=1.36, 95% CI 1.21 to 1.54). Also noted was an association between long sleep duration and obesity for both sexes.

**Table 1** Distribution of socio-demographic attributes, health behaviours and chronic disease by weight outcomes and sex, Thai Cohort Study 2009

| Covariates | Overall, N=60 569 | Weight outcomes by sex* | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Underweight | | Normal | | Overweight | | Obese | |
| | | M | F | M | F | M | F | M | F |
| Age (mean in years) | 35.6 | 33.0 | 30.1 | 35.5 | 33.7 | 38.6 | 36.5 | 39.6 | 36.8 |
| Marital status | | | | | | | | | |
| Married | 55.3 | 38.2 | 33.7 | 54.5 | 49.1 | 67.9 | 60.2 | 70.9 | 55.2 |
| Single | 37.9 | 57.1 | 61.5 | 40.2 | 42.5 | 26.3 | 30.5 | 24.4 | 36.2 |
| Separated/widowed | 6.8 | 4.7 | 4.8 | 5.3 | 8.4 | 5.8 | 9.3 | 4.8 | 8.6 |
| Personal income (baht/month) | | | | | | | | | |
| Up to 3000 | 5.9 | 15.6 | 6.3 | 7.1 | 5.5 | 4.8 | 5.3 | 4.7 | 6.1 |
| 3000–7000 | 12.9 | 18.7 | 18.6 | 11.8 | 15.0 | 8.1 | 14.6 | 7.5 | 14.5 |
| 7000–10 000 | 22.4 | 26.6 | 32.4 | 20.5 | 27.5 | 14.7 | 23.2 | 13.3 | 23.0 |
| 10 000–20 000 | 35.7 | 27.8 | 31.7 | 39.1 | 33.9 | 41.3 | 32.7 | 38.2 | 31.5 |
| >20 000 | 23.1 | 11.3 | 11.0 | 21.5 | 18.1 | 31.1 | 24.3 | 36.4 | 24.9 |
| Geographical residence | | | | | | | | | |
| Rural | 44.0 | 50.8 | 42.9 | 48.5 | 42.7 | 45.2 | 42.8 | 42.6 | 39.9 |
| Urban | 56.0 | 49.2 | 57.0 | 51.6 | 57.3 | 54.8 | 57.2 | 57.4 | 60.1 |
| Smoking | | | | | | | | | |
| Never | 76.8 | 61.4 | 97.1 | 57.4 | 97.0 | 50.7 | 96.5 | 47.2 | 95.2 |
| Former | 14.3 | 17.7 | 2.2 | 23.9 | 2.3 | 31.3 | 2.8 | 33.6 | 3.9 |
| Current | 8.9 | 20.9 | 0.8 | 18.7 | 0.7 | 18.0 | 0.7 | 19.2 | 0.9 |
| Drinking (days/week) | | | | | | | | | |
| 0 | 72.9 | 62.9 | 91.3 | 52.7 | 90.6 | 48.1 | 90.9 | 49.6 | 91.7 |
| 1–3 | 22.5 | 29.8 | 8.3 | 38.6 | 8.8 | 41.7 | 8.5 | 40.1 | 7.5 |
| 4+ | 4.6 | 7.3 | 0.4 | 8.7 | 0.6 | 10.2 | 0.5 | 10.3 | 0.9 |
| Fruit and vegetable intake (serves/day) | | | | | | | | | |
| 0–4 | 41.8 | 48.8 | 39.9 | 44.1 | 38.6 | 43.3 | 38.9 | 46.6 | 41.7 |
| 5+ | 58.2 | 51.1 | 60.2 | 55.9 | 61.4 | 56.7 | 61.1 | 53.4 | 58.3 |
| Physical activity (sessions/week) | | | | | | | | | |
| 0–2 | 19.4 | 18.8 | 22.7 | 13.5 | 21.7 | 14.2 | 23.8 | 18.8 | 25.2 |
| 3+ | 80.6 | 81.2 | 77.3 | 86.5 | 78.4 | 85.9 | 76.2 | 81.2 | 74.8 |
| Screen time (h/day) | | | | | | | | | |
| 0–4 | 82.5 | 81.1 | 80.7 | 82.8 | 83.3 | 83.8 | 82.6 | 81.4 | 80.1 |
| 5+ | 11.5 | 18.9 | 19.3 | 17.2 | 16.7 | 16.2 | 17.4 | 18.6 | 19.9 |
| Ever been diagnosed for chronic conditions† | 30.3 | 20.2 | 20.0 | 26.8 | 23.6 | 36.1 | 31.2 | 48.0 | 36.9 |
| Ever been diagnosed for depression | 1.8 | 2.3 | 2.0 | 1.6 | 1.6 | 1.4 | 1.9 | 2.0 | 2.6 |

*Weight (body mass index) outcomes: normal = 18.5 to <23; underweight = ≤18.5; overweight-at-risk =23 to <25; obese =≥25; except for age, tabulated numerals represent covariate prevalences (%) within each weight–sex category.
†Doctor-diagnosed chronic conditions include type I and type II diabetes, high cholesterol, high blood pressure, heart disease, stroke, cancers (liver, lung, stomach, colon, breast and others), goitre, epilepsy, liver disease, lung disease, arthritis and asthma.
M, male; F, female.

For longitudinal analysis, we performed multinomial logistic regression of sleep duration on incident weight gain, adjusting for the same covariates as for the cross-sectional analyses. The dependent variable was substantive weight gain between 2005 and 2009, defined by three weight increase categories (5% to <10%, 10% to <20% and 20+%). Each category of weight gain was separately compared with no weight increase (ie, <5%) for calculation of ORs. Progressively larger increments in weight gain show progressively stronger significant associations with short sleep (<6 h), after adjusting for covariates (AOR=1.10, 95% CI 1.02 to 1.20; AOR=1.30, 95% CI 1.18 to 1.44 and AOR=1.69, 95% CI 1.39 to 2.05).

## DISCUSSION

Our large cohort study revealed a consistent and substantial association between short sleep and obesity among Thai adults. Significant covariate AORs for the short sleep–obesity effect were 1.36 for men and 1.49 for women in 2009; corresponding ORs were similar in 2005. Longitudinal 2004–2009 cohort data confirmed a short sleep–weight gain relationship with a notable dose–response.

Cohort evidence from the USA is supportive. The Nurses Health Study analysed sleep and weight in a cohort of women for 16 years and found that those with ≤5 h sleep had more weight gain (additional

**Table 2** Association between sleep duration and weight outcomes, Thai Cohort Study 2009

| Sleep duration | Overall, N=60 569 n (%) | Weight outcomes* Underweight versus normal AOR† (95% CI) | Overweight versus normal AOR† (95% CI) | Obese versus normal AOR† (95% CI) |
|---|---|---|---|---|
| Sleep duration (men) | | | | |
| <6 h | 4070 (14.9) | 1.16 (0.90 to 1.50) | 1.13 (1.00 to 1.28) | **1.36** (1.21 to 1.52) |
| 6 h | 4815 (17.6) | 0.90 (0.71 to 1.16) | 0.98 (0.88 to 1.09) | **1.16** (1.04 to 1.29) |
| 7 h | 5719 (20.9) | Ref | Ref | Ref |
| 8 h | 8664 (31.6) | 1.15 (0.94 to 1.42) | 1.06 (0.97 to 1.17) | 1.08 (0.99 to 1.19) |
| ≥9 h | 4139 (15.1) | 1.08 (0.84 to 1.39) | 1.03 (0.91 to 1.16) | **1.16** (1.03 to 1.30) |
| Sleep duration (women) | | | | |
| <6 h | 4728 (14.3) | 0.97 (0.85 to 1.11) | **1.33** (1.18 to 1.51) | **1.49** (1.32 to 1.68) |
| 6 h | 5120 (15.4) | 0.99 (0.87 to 1.12) | **1.21** (1.07 to 1.37) | **1.28** (1.14 to 1.44) |
| 7 h | 6965 (21.0) | Ref | Ref | Ref |
| 8 h | 11 007 (33.2) | 1.05 (0.95 to 1.16) | **1.13** (1.02 to 1.25) | **1.14** (1.04 to 1.26) |
| ≥9 h | 5342 (16.1) | **1.21**‡ (1.07 to 1.37) | **1.22** (1.07 to 1.39) | **1.36** (1.20 to 1.53) |

*Body mass index (BMI) categories: normal = 18.5 to <23; underweight = ≤18.5; overweight-at-risk = 23 to <25; obese = ≥25. For each 'abnormal' category, multinomial logistic regression compares the outcome odds to the outcome odds of a 'normal' BMI, with the results expressed as an Odd Ratio (OR).
†Adjusted ORs—covariates included age, marital status, personal income, geographical rural–urban residence, physical activity, fruit and vegetable intakes, screen time, smoking, alcohol drinking, doctor-diagnosed depression and chronic health conditions (see the Methods section).
‡Bolded values indicate statistical significance at p<0.05.

1.14 kg) than those with 7 h sleep. Effects were more extreme with less sleep: for a 15 kg weight gain, Hazard Ratios (HRs) were 1.28 and 1.10 for ≤5 and 6 h, respectively.[19] Even more extreme sleep deprivation was studied in a cohort of the US National Health and Nutrition Examination Survey over 9 years; for participants aged 32–49 years, the obesity OR was 3.21, for those with ≤4 h sleep compared with 7 h sleep.[20] A prospective study of young Swiss adults also revealed a short sleep–obesity relationship: the effect was maximum at baseline (OR 7.4 at age 27 years) and attenuated after 13 years (OR 1.1 at age 40 years).[22]

Some longitudinal Asian evidence is also supportive. A national panel study in Korea reported an AOR of 1.25 (95% CI 1.06 to 1.48) for ≤5 h sleep and obesity.[25] For the same exposure–outcome categories, a cohort in Japan showed similar results with an AOR of 1.50 (95% CI 1.10 to 2.10).[24]

The search for mechanisms to link sleep and obesity is ongoing. One obvious pathway for those with short sleep is the increase in time available for eating.[37] Also, fatigue associated with sleep deprivation leads to reduced exercise.[38 39] And as already mentioned, appetite-regulating hormones such as leptin and ghrelin are expected to play a major biological role in linking short sleep and obesity.[19 40] As well, other studies have drawn attention to obstructive sleep apnoea and when that condition occurs, there is a strong relationship to both short sleep and obesity.[14 17]

Our data also showed that some long-sleeping women were underweight, and other reports suggest that this may be related to comorbidity.[41 42] However, our results were adjusted for depression and 17 chronic health conditions, so we do not have an explanation to link long sleep and underweight. Nor do we understand the U-shaped risk pattern whereby both short and long sleep were associated with obesity in our Thai cohort. Such a pattern has been observed in other reports.[8 9 35]

We investigated personal environments and found that short sleep among our Thai cohort was more common for those who lack a fan or an air conditioner at home; at baseline in 2005, this affected 26% of short sleepers and 13% of others. However, we found no link between noisy home environments, hours of work, rural or urban location and short sleep duration. But we did note an occupational travel time effect: in 2005, short sleep was reported by 17%, 14% and 12% of those travelling >1 h, 30 min to 1 h and 30 min to work, respectively.

One limitation of this study was the subjective nature of self-reporting sleep duration, a problem noted by many others conducting sleep research.[9 19] Weight and height of cohort members were also self-reported, but our previous validation study has found these numbers to be reliable.[27] We also noted the relatively higher level of education among cohort members, but it is unclear how this could affect the results.

The strength of this study was the large sample, longitudinal data, national representation, involvement of young adults and the opportunity to control for many covariates in the analyses. We noted similar cross-sectional results for both 2005 and 2009 although on both occasions, cohort members were unaware of any hypothesised relationship between sleep and weight. Even more compelling was the evidence from the 4-year longitudinal analyses yielding similar results.

## CONCLUSIONS

Our large adult cohort in Thailand shows a consistent relationship between short sleep duration and obesity. This is one of the few sleep–obesity studies in a middle-income country, one of the first in Southeast Asia and one of only a few longitudinal studies investigating this topic worldwide. The epidemiological evidence available now points to a consistent, substantial and presumably causal association for adults but mechanisms should be further explored.

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**Competing interests** None.

**Patient consent** Obtained.

**Ethics approval** Ethics approval was obtained from Sukhothai Thammathirat Open University Research and Development Institute (protocol 0522/10) and The Australian National University Human Research Ethics Committee (protocol 2004344 and 2009570). Informed written consent was obtained from all participants.

**Contributors** VY and CB conceptualised and designed the sleep study. AS and SS devised and directed the cohort study. VY analysed and drafted the manuscript, with input by AS. All authors approved the final manuscript submission.

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**Data sharing statement** Additional unpublished data are available upon request, please contact Principal Investigators Professor Adrian C Sleigh (adrian.sleigh@anu.edu.au) or Associate Professor Sam-ang Seubsman (sam-ang.seu@stou.ac.th).

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**STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cohort studies***

| Section/Topic | Item # | Recommendation | Reported on page # |
|---|---|---|---|
| **Title and abstract** | 1 | (*a*) Indicate the study's design with a commonly used term in the title or the abstract | Page 1 |
| | | (*b*) Provide in the abstract an informative and balanced summary of what was done and what was found | Page 1 |
| **Introduction** | | | |
| Background/rationale | 2 | Explain the scientific background and rationale for the investigation being reported | Page 3 |
| Objectives | 3 | State specific objectives, including any prespecified hypotheses | Page 3 |
| **Methods** | | | |
| Study design | 4 | Present key elements of study design early in the paper | Page 3 |
| Setting | 5 | Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection | Page 4 |
| Participants | 6 | (*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up | Page 4 |
| | | (*b*) For matched studies, give matching criteria and number of exposed and unexposed | n/a |
| Variables | 7 | Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable | Page 4 |
| Data sources/ measurement | 8* | For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group | Page 4 |
| Bias | 9 | Describe any efforts to address potential sources of bias | Page 4 |
| Study size | 10 | Explain how the study size was arrived at | Page 4 |
| Quantitative variables | 11 | Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why | Page 5 |
| Statistical methods | 12 | (*a*) Describe all statistical methods, including those used to control for confounding | Page 5 |
| | | (*b*) Describe any methods used to examine subgroups and interactions | Page 4 |
| | | (*c*) Explain how missing data were addressed | Page 5 |
| | | (*d*) If applicable, explain how loss to follow-up was addressed | Page 4 |
| | | (*e*) Describe any sensitivity analyses | Page 5 |
| **Results** | | | |

| | | | |
|---|---|---|---|
| Participants | 13* | (a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed | Page 4 |
| | | (b) Give reasons for non-participation at each stage | Page 5 |
| | | (c) Consider use of a flow diagram | Page 5 |
| Descriptive data | 14* | (a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders | Page 5 |
| | | (b) Indicate number of participants with missing data for each variable of interest | Page 5 |
| | | (c) Summarise follow-up time (eg, average and total amount) | Page 4 |
| Outcome data | 15* | Report numbers of outcome events or summary measures over time | Pages 4-5 |
| Main results | 16 | (*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included | Page 5 |
| | | (*b*) Report category boundaries when continuous variables were categorized | n/a |
| | | (*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period | Page 5-6 |
| Other analyses | 17 | Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses | |
| **Discussion** | | | Page 6 |
| Key results | 18 | Summarise key results with reference to study objectives | Page 6 |
| **Limitations** | | | |
| Interpretation | 20 | Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence | Page 7-8 |
| Generalisability | 21 | Discuss the generalisability (external validity) of the study results | Page 8 |
| **Other information** | | | |
| Funding | 22 | Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based | Page 9 |

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

**Note:** An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.