Self-diagnosis of influenza during a pandemic: a cross-sectional survey

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ABSTRACT

Background: Self-diagnosis of influenza is an important component of pandemic control and management as it may support self-management practices and reduce visits to healthcare facilities, thus helping contain viral spread. However, little is known about the accuracy of self-diagnosis of influenza, particularly during pandemics.

Methods: We used cross-sectional survey data to correlate self-diagnosis of influenza with serological evidence of 2009 pandemic influenza A(H1N1) infection (haemagglutination inhibition titres of ≥1:40) and to determine what symptoms were more likely to be present in accurate self-diagnosis. The sera and risk factor data were collected for the national A(H1N1) seroprevalence survey from November 2009 to March 2010, 3 months after the first pandemic wave in New Zealand (NZ).

Results: The samples consisted of 318 children, 413 adults and 423 healthcare workers. The likelihood of being seropositive was no different in those who believed they had influenza from those who believed they did not have influenza in all groups. Among adults, 23.3% (95% CI 11.9% to 34.7%) of those who reported having had influenza were seropositive for H1N1, but among those reporting no influenza, 21.3% (95% CI 13% to 29.7%) were also seropositive. Those meeting NZ surveillance criteria for influenza infection were more likely to believe they had the flu (surveillance data adult sample OR 27.1, 95% CI 13.6 to 53.6), but these symptom profiles were not associated with a higher likelihood of H1N1 seropositivity (surveillance data adult sample OR 0.93, 95% CI 0.5 to 1.7).

Conclusions: Self-diagnosis does not accurately predict influenza seropositivity. The symptoms promoted by many public health campaigns are linked with self-diagnosis of influenza but not with seropositivity. These findings raise challenges for public health initiatives that depend on accurate self-diagnosis by members of the public and appropriate self-management action.

BACKGROUND

Self-diagnosis is an important component of pandemic control and management. The use of self-diagnosis in an influenza pandemic can prevent some exposures by reducing outpatient visits to primary care clinics. During the 2009 pandemic, following Centers for Disease Control recommendations, patient teaching brochures advised patients to stay at home and avoid contact with other people if they had influenza like illness (ILI), seeking medical assistance only in case of complications or risk factors. While the accuracy of self-diagnosis has been studied for a range of common diseases (eg, uncomplicated urinary tract infections and vaginal yeast infections), it has not been established for influenza.

Although self-diagnosis of influenza is clearly desirable for the purposes of infection containment, it also presents challenges for patients and doctors alike. As the social science literature clearly articulates, diagnosis is central to the practice of medicine and to defining the roles of, and boundaries between, the patient and the professional; however, self-diagnosis blurs these distinctions.
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The diagnosis of influenza by a lay person may be independent of medical contact, using resources such as family, friends or other non-medical sources of information, for example on-line or internet resources (independent self-diagnosis). It may also be supported by a health professional via a helpline without the lay person being seen for a clinical diagnosis (assisted self-diagnosis).

The purpose of this study was to determine whether lay people’s assessment of influenza status is confirmed by serological testing, and whether the presence of particular symptoms assists individuals in the correct identification of influenza. It also aimed to measure the accuracy of self-diagnosis by healthcare workers (HCW). Establishing the current reliability of self-diagnosis will either provide assurances about, or identify shortcomings in, public health strategies to contain the spread of influenza.

METHODS
Population sample
This study was conducted as part of the national Environmental Science and Research (ESR) seroprevalence study in early 2010.11 This study used a purposive, multi-stage random cross-sectional survey of 1147 subjects from selected primary care patient registers from 14 general practitioner (GP) practices. The practices were selected purposively on the basis of observed high, medium and low incidence during the pandemic and on ethnic distribution. Each practice was stratified by age and by ethnic group. Within each stratum, simple random sampling was undertaken, with oversampling in strata for Māori and Pacific respondents to improve the precision of estimates for these groups. A second sample consisted of 540 HCW (369 HCW located in Auckland and Middlemore Hospitals, and 171 from the 14 GP practices in the community study). The HCW sample included medical, nursing and other staff. A simple random sampling procedure was performed to select participants for this sample.11 Sera and risk factor data were collected from November 2009 to March 2010, 3 months after the first pandemic wave in New Zealand (NZ).12 Ethics approval (MEC/09/09/106) was obtained from the Multiregional Ethics Committee of the NZ Ministry of Health. Written informed consent was obtained from all participants.11

We excluded participants born before 1957 because of the higher level of pre-pandemic seropositivity in this group.11 We treated those under age 18 as a separate group because their questionnaires were usually completed by parents (self-diagnosis by proxy) and their health-related behaviours were likely influenced or, for the very young, entirely managed by their parents.

We also considered HCW separately from lay participants; however, as the sampling methods for this group were different from the main community sample (the geographical area was more restricted in the HCW sample), comparisons between this group and the adult community sample should be made with caution.

Laboratory testing
Blood samples were obtained by phlebotomists in the GP clinics, and serological testing was carried out at the National Influenza Centre at ESR using a haemagglutination inhibition assay in line with the standard protocol provided by the WHO Collaborating Centre in Melbourne. Haemagglutination inhibition titres of $\geq$40 against H1N1 were considered seroprotective as well as seropositive. Laboratory testing methods are fully described elsewhere.11

Questionnaire
A questionnaire was administered by nurses from 14 participating GP clinics at the time of the blood sample collection in order to record information about respondent demographics, whether respondents believed they had contracted influenza in 2009 and their symptoms. Questions were both multiple-choice and open-ended.

Respondents were asked ‘Did you have the flu or influenza over this last winter (June to August)?’, with options being ‘yes’, ‘possibly’, ‘no’ and ‘don’t know’. Those who believed they had had influenza were asked how they knew, choosing either:

1. I could tell on my own or with the help of my family and friends
2. I called the nurse or HealthLine and they helped me to decide, or
3. I saw my doctor or other health professional who told me I did.

Self-diagnosis was defined as including both independent and assisted forms (ie, choosing 1 or 2 above) for those who responded ‘yes’ to the question about having had influenza.

Two additional case definitions of influenza were used based on reported symptoms: ILI defined by the NZ sentinel surveillance definition13 of two or more symptoms from fever, muscle ache and headache (reports of chills are included in this definition, but this information was not collected in this study) and also by the NZ Ministry of Health14 as fever, plus cough or sore throat (reports of chills or sweating are included in this definition, but this information was not collected in the study).

Demographic information included age, gender, self-identified ethnicity and socioeconomic deprivation (using NZDep, a well-validated measure of small-area socioeconomic deprivation based on census-derived characteristics such as income, education and household crowding, and assigned according to domicile address15). Ethnicity classification used the NZ 2006 Census questions, and prioritised ethnicity coding according to Ministry of Health ethnicity data protocols.16 Participants could choose up to nine different symptoms (fever or high temperature; cough; sore
return the questionnaire and were thus excluded from the analysis). This gave a target rate of 76%. For the HCW branch of the study, the minimum sample size was calculated using the same criteria as for the community study. The number of subjects (171 primary HCW and 369 secondary HCW) exceeded the minimum requirement. Of the 1687 subjects with completed questionnaires and serological results across the community and HCW studies, after excluding those respondents born before 1957, 413 responses (unweighted frequency) were considered for the analyses of adult responses. In addition, 318 responses concerning children were considered and 423 HCW responses. This gave a final sample size of 1154 people across the three groups.

The baseline demographic characteristics of the study populations are shown in table 1. These are unweighted frequencies and percentages; all subsequent analyses take the sampling structure into account. The sample was not adequately powered to demonstrate ethnic differences in the findings reported below.

### Accuracy of self-report of influenza

Seropositivity status was compared across the three self-reported influenza status groups (yes, no or possibly had the flu in 2009). Respondents who answered ‘don’t know’ to this question (n=16, 21 and 22 for adult, child and HCW samples, respectively) were excluded from this analysis. As shown in table 2, the likelihood of being seropositive was not significantly different between the three self-reported influenza status groups in any of the three sample groups. For adults in the community sample, point estimates of seropositive status ranged from 21.3% to 25.1% across the three self-report groups; for under 18s in the same sample, seropositive rates were between 40.1% and 45.9%, which was the highest among all three sample groups; and for HCW, the range was between 25.7% and 33.0% seropositive.

Table 3 shows that among those study subjects who reported having had influenza, the proportion of people who were seropositive was higher among those who reached a decision in conjunction with a health professional than among those who reported reaching a diagnosis on their own (including using a telephone helpline). While this pattern was consistent across all three sample sources, none of these differences were statistically significant (all p > 0.3), which possibly reflects the smaller sample sizes for this analysis.

As shown in table 4, self-reported flu status performed poorly as a screening tool for H1N1 infection, failing to detect the majority of those who were seropositive (adult sensitivity 45.7%). Only about a quarter of those who considered themselves to have had influenza during the preceding winter showed serological evidence of infection (adult PPV 24.1%). Self-reported flu status had higher sensitivity and lower specificity than the Ministry of Health and NZ sentinel surveillance case definitions. Screening performance (sensitivity, specificity, PPV and NPV) was otherwise broadly similar across the three sets of ‘screening’ criteria used. PPV and NPV values across...
all definitions followed the pattern seen for seropositive prevalence (eg, children had the highest PPV, reflecting a higher proportion of seropositive tests).

Seropositive status, symptom profiles and case definitions of influenza
Using Ministry of Health and the NZ sentinel surveillance ILI case definitions, we sought to confirm whether there was an increased likelihood of seropositivity for those who met these case definitions, based on self-report of symptoms. The likelihood of being seropositive was not significantly different between these symptom profile groups for any sample group (see table 5; 95% CIs for all ORs included 1). People who met a case definition had a much greater likelihood of self-reporting having had influenza (table 5, for both definitions).

**DISCUSSION**
**Key findings**
To our knowledge, this is the first published study of the effectiveness of self-diagnosis of influenza compared with laboratory evidence of infection in a broad...
population-based sample during a pandemic. The likelihood of being seropositive was no different in those who believed they had influenza from those who believed they did not have influenza. This finding applied to HCW as well as adults and children. Our study showed that self-diagnosis in a NZ population lacks sensitivity and specificity for diagnosing influenza. The poor sensitivity may lead people with influenza to believe that they are well and therefore to fail to take measures to limit their contribution to influenza spread. The lack of specificity may result in delayed medical treatment when serious treatable illness is present.18

**Strengths and weaknesses of the study**

Limitations of this study include the fact that some of the participants who believed they had the flu and yet were seronegative for H1N1 may have had seasonal influenza or other respiratory pathogens. However, H1N1 was the dominant influenza strain in 2009, accounting for 77.6% of influenza viruses that were sub-typed during the year.19 Further, the fact that seasonal influenza was replaced very quickly by pandemic H1N1 reduces this limitation to some extent. A small proportion of those who were seropositive will have baseline immunity to H1N1 acquired prior to 2009, although testing of stored

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**Table 3** Serological status according to diagnostic approach for people self-reporting having had influenza

| Method of diagnosis | n  | % Seropositive* | 95% CI       | p Value†   |
|---------------------|----|----------------|--------------|------------|
| Adults (18+ years)† (n=108) |    |                |              |            |
| Health professional | 37 | 27.1%          | (7.9 to 46.3) | 0.392      |
| Self-diagnosis      | 61 | 17.7%          | (5.3 to 30.1) |            |
| Children (<18 years)§ (n=86) |    |                |              |            |
| Health professional | 43 | 39.7%          | (18.4 to 61.1) | 0.332      |
| Self-diagnosis      | 34 | 25.2%          | (4.5 to 45.9) |            |
| Healthcare workers¶ (n=94) |    |                |              |            |
| Health professional | 25 | 40.0%          | (21.1 to 61.3) | 0.356      |
| Self-diagnosis      | 67 | 29.9%          | (19.3 to 42.3) |            |

*Weighted percentage.† The p value for healthcare workers is from Pearson’s χ² test; p values for adults and children are from the Rao-Scott χ² test.§ 10 Adults from a community sample were missing information on the pathway of diagnosis.¶ Children were missing information on the pathway of diagnosis.

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**Table 4** Screening performance of influenza definitions for detecting seropositive status (sensitivity, specificity, PPV and NPV)

| Measure                  | NZ sentinel surveillance ILI definition* | NZ Ministry of Health ILI definition † | Self-reported flu status‡ |
|--------------------------|------------------------------------------|----------------------------------------|---------------------------|
| Points (95% CI)          | Points (95% CI)                          | Points (95% CI)                        |
| Adults (18+ years)§      | (n=413)                                  | (n=413)                                | (n=413)                   |
| Sensitivity             | 37.7 (25.5 to 50.0)                      | 38.0 (25.6 to 50.4)                   | 45.7 (33.0 to 58.3)       |
| Specificity             | 60.5 (53.6 to 67.4)                      | 67.2 (60.6 to 73.8)                   | 58.1 (51.0 to 65.3)       |
| PPV                     | 21.6 (13.6 to 29.6)                      | 25.1 (15.8 to 34.4)                   | 24.1 (16.2 to 31.9)       |
| NPV                     | 77.1 (70.6 to 83.5)                      | 79.0 (73.1 to 84.8)                   | 78.7 (72.0 to 85.3)       |
| Children (<18 years)¶   | (n=318)                                  |                                        |                           |
| Sensitivity             | 32.5 (21.0 to 44.0)                      | 36.0 (24.5 to 47.6)                   | 42.4 (30.0 to 54.9)       |
| Specificity             | 68.4 (58.4 to 78.4)                      | 57.2 (47.3 to 67.1)                   | 52.6 (42.1 to 63.0)       |
| PPV                     | 43.4 (29.1 to 57.7)                      | 38.6 (27.0 to 50.1)                   | 40.9 (29.3 to 52.6)       |
| NPV                     | 57.6 (48.2 to 67.0)                      | 54.5 (44.0 to 65.0)                   | 54.1 (43.0 to 65.3)       |
| Healthcare workers**    | (n=423)                                  |                                        |                           |
| Sensitivity             | 32.5 (23.8 to 41.1)                      | 30.7 (22.2 to 39.2)                   | 48.2 (38.8 to 57.6)       |
| Specificity             | 70.8 (65.7 to 75.9)                      | 76.4 (71.6 to 81.2)                   | 57.7 (51.9 to 63.4)       |
| PPV                     | 29.9 (21.9 to 37.9)                      | 32.7 (23.8 to 41.6)                   | 30.5 (23.6 to 37.3)       |
| NPV                     | 73.5 (68.5 to 78.6)                      | 74.7 (69.8 to 79.5)                   | 74.3 (68.6 to 80.1)       |

*Two or more symptoms from: fever, muscle ache and headache.† Fever, plus cough and/or sore throat.‡ Self-diagnosis, assisted-self-diagnosis or self-diagnosis by proxy.¶ Children: 21 don’t know respondents on self-reported influenza status were excluded from analysis.** Healthcare workers: 22 don’t know respondents on self-reported influenza status were excluded from analysis. ILI, Influenza like illness; NPV, negative predictive value; NZ, New Zealand; PPV, positive predictive value.
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| Table 5 | Association between symptom profiles and self-reported flu status and seropositive status |
|-----------------|-------------------------------------------------|
| **NZ sentinel surveillance definitions*** | **Self-report status** | **Seropositive status** |
| | **OR (95% CI)** | **OR (95% CI)** |
| Adults (18+ years)§ | Reference group | Reference group |
| No or 1 symptom | 27.1 (13.6 to 53.6) | 0.93 (0.5 to 1.7) |
| 2 or 3 symptoms | 21.5 (8.98 to 51.6) | 1.04 (0.52 to 1.09) |
| Children (<18 years)¶ | Reference group | Reference group |
| No or 1 symptom | Reference group | Reference group |
| 2 or 3 symptoms | 18.2 (10.3 to 32.1) | 1.19 (0.75 to 1.9) |
| Healthcare workers** | Reference group | Reference group |
| No or 1 symptom | Reference group | Reference group |
| 2 or 3 symptoms | Reference group | Reference group |

| Ministry of Health (MoH) definition† | **OR (95% CI)** | **OR (95% CI)** |
| Adults (18+ years)§ | Reference group | Reference group |
| Met MoH definition | 11.5 (6.1 to 21.8) | 1.3 (0.7 to 2.3) |
| Did not meet definition | Reference group | Reference group |
| Children (<18 years)¶ | Reference group | Reference group |
| Met MoH definition | 9.5 (4.5 to 20) | 0.8 (0.4 to 1.4) |
| Did not meet definition | Reference group | Reference group |
| Healthcare workers** | Reference group | Reference group |
| Met MoH definition | 13.3 (7.4 to 23.9) | 1.46 (0.9 to 2.4) |
| Did not meet definition | Reference group | Reference group |

ORs and 95% CI derived from independent logistic regression models.
*Two or more symptoms from: fever, muscle ache and headache.
†Fever, plus cough and/or sore throat.
‡Self-diagnosis, assisted-self-diagnosis or self-diagnosis by proxy.
§Adults: 16 ‘don’t know’ respondents on self-reported influenza status were excluded from analysis.
¶Children: 21 ‘don’t know’ respondents on self-reported influenza status were excluded from analysis.
**Healthcare workers: 22 ‘don’t know’ respondents on self-reported influenza status and five respondents missing immunological status were excluded from analysis.

Sera shows that the level of such infection is low, ranging from 6.5% to 7.5% in the 20–59-year-old population. Further, this survey was based on symptom recall rather than symptom reports at the time of presentation. Symptoms reported retrospectively may well not match the actual symptoms experienced during the illness. However, the pandemic was an unusual event of some concern to the individual and recall bias tends to be minimal in such situations. Furthermore, there is some validity in focusing on recalled symptoms, because these may reflect the participants’ enduring perceptions of influenza, which may guide their behaviour in relation to future episodes of ILI. The higher likelihood of positive serology in those adults who consulted a health professional may be related to greater severity of their disease which this study does not capture. Also, it is likely that a higher proportion of people than usual may have consulted a healthcare provider due to the high media attention given to ‘swine-flu’. The findings of this study might not be generalisable to other influenza viruses causing seasonal and pandemic disease.

**Strengths and weaknesses in relation to other studies**

Other studies have attempted to understand how lay people report ILI, but have not obtained medical or laboratory confirmation of the diagnosis as ours did. In excess of nine H1N1 seroprevalence studies have been carried out following the pandemic. Almost all used unlinked specimens and so were not able to question participants about their symptom history. Two studies in selected military populations collected symptom data. One prospective study of Singaporean military personnel tracked symptomatic illness during the pandemic and found that less than a third of those who were seropositive reported symptoms. A small cross-sectional study reported seroconversion following an H1N1 outbreak in a Finnish military garrison, and found that sensitivity for seropositivity was 50% on the basis of self-reported upper respiratory tract infection symptoms (ie, half of those with serological evidence of infection reported a history of upper respiratory tract infection symptoms). This is comparable with the sensitivities for the current dataset, which were 45.7% and 48.2% for the community adults and the HCW adults, respectively. Participants in this NZ seroprevalence survey were more likely to believe they had been infected if they had symptoms commonly advertised by public health campaigns as being linked with the flu. However, these symptom profiles were not significantly associated with seropositivity. This finding is consistent with a recent systematic review of symptoms in volunteer challenge studies, where nearly one in three participants demonstrated no clinical symptoms of influenza despite laboratory confirmed infection. The authors of that study questioned whether naturally acquired influenza might produce more marked symptoms. Our study would appear to show that this is not the case, at least for pandemic H1N1 influenza.
Implications for clinicians and policymakers

These study findings raise important questions for pandemic control policies. On the positive side, they show that the NZ public has absorbed a fairly coherent ILI case definition that includes the symptoms traditionally linked with influenza. Unfortunately, we have demonstrated that this generic picture of ILI is a poor predictor of influenza infection. The classic symptoms of influenza are non-specific and accompany other infections commonly seen during the influenza season. A systematic review comparing influenza symptoms to independent criterion standards for influenza highlighted that epidemiological data (for example, reports of regional influenza patterns) were probably more useful than clinical indicators for predicting whether an individual had influenza. In addition, daily temperature measurement plus reporting of respiratory symptoms resulted in reduced transmission of H1N1 virus. It is also useful to note that HCW perform no better than non-professionals: the PPV of an ILI diagnosis by a HCW was 30.1%. Interestingly, this value is similar to the PPV of clinical diagnosis by a GP for patients presenting to sentinel sites over the same period in 2009 (31.3% based on 624 viruses from 1993 swabs received). These findings reinforce public health advice during the pandemic that patients should seek medical care on the basis of disease severity rather than for the purpose of diagnosis.

Further research

Given the importance of self-diagnosis to containment and mitigation measures, further investigations around the low accuracy of self-diagnosis would be useful. Priorities for such research could include more in-depth qualitative investigation of patient reports of influenza, prospective exploration of patient self-diagnosis at the time of respiratory infection, and variations in self-diagnosis by ethnicity, socioeconomic status and age, particularly given the differential distribution of respiratory illness across these groups (our sample was not sufficiently large to enable these analyses). The presenting symptoms of influenza may vary depending on the type of influenza responsible. In Singapore, H1N1 and seasonal influenza had different symptom profiles, with fever and runny nose being more common among seasonal influenza cases and the prevalence of specific symptoms among H1N1 cases also varied between studies, so further exploration is warranted.

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Correction notice

The “To cite: ...” information and running footer in this article have been updated with the correct volume number (volume 1).

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Competing interests

None.

Ethics approval

This study was conducted with the approval of the Multiregional Ethics Committee of the NZ Ministry of Health (MEC/09/09/106).

Contributors

AJ devised and led the study, guarantees the report and is the corresponding author. MGB refined the design. JS carried out the data analysis. AJ, JS and MGB prepared the manuscript. QSH and DB managed the seroprevalence study. All authors contributed to writing and revising the report.

Provenance and peer review

Not commissioned; externally peer reviewed.

Data sharing statement

No additional data available.

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of cross-sectional 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 | 1                  |
|                        |        | (b) Provide in the abstract an informative and balanced summary of what was done and what was found | See manuscript management system generated abstract |
| Introduction           | 2      | Explain the scientific background and rationale for the investigation being reported | 1                  |
| Background/rationale   | 3      | State specific objectives, including any prespecified hypotheses                | 1                  |
| Methods                | 4      | Present key elements of study design early in the paper                        | 2                  |
| Study design           | 5      | Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection | 2                  |
| Setting                | 6      | (a) Give the eligibility criteria, and the sources and methods of selection of participants | 2                  |
| Participants           | 7      | Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable | 3                  |
| Variables              | 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 | 3-4                |
| Data sources/measurement| 9      | Describe any efforts to address potential sources of bias                      | 4                  |
| Bias                   | 10     | Explain how the study size was arrived at                                      | 3                  |
| Study size             | 11     | Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why | 4-5                |
| Statistical methods    | 12     | (a) Describe all statistical methods, including those used to control for confounding | 4-5                |
|                        |        | (b) Describe any methods used to examine subgroups and interactions            | 4-5                |
|                        |        | (c) Explain how missing data were addressed                                    | tables             |
|                        |        | (d) If applicable, describe analytical methods taking account of sampling strategy |                    |
| **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 |  |
| (b) Give reasons for non-participation at each stage |  |
| (c) Consider use of a flow diagram | No |
| Descriptive data | 14* |
| (a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders | 6 and table 1 |
| (b) Indicate number of participants with missing data for each variable of interest | See tables |
| Outcome data |  |
| Main results | 15* |
| (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 | yes |
| (b) Report category boundaries when continuous variables were categorized | 5 |
| (c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period | n/a |
| Other analyses | 17 |
| Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses | 6-8 |

| **Discussion** |  |
|---|---|
| Key results | 18 |
| Summarise key results with reference to study objectives |  |
| Limitations | 19 |
| Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias | 8,9 |
| Interpretation | 20 |
| Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence | 8 |
| Generalisability | 21 |
| Discuss the generalisability (external validity) of the study results | 8,9 |

| **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 | See Scholar One |

*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.
