Validity of self-reported smoking status: Comparison of patients admitted to hospital with acute coronary syndrome and the general population

Jill P. Pell, Sally J. Haw, Stuart M. Cobbe, David E. Newby, Alastair C. H. Pell, Keith G. Oldroyd, David L. Murdoch, Stuart D. Pringle, Frank G. Dunn, Paul D. MacIntyre, Timothy J. Gilbert, Colin M. Fischbacher, William Borland

Many studies rely on self-reported smoking status. We hypothesized that patients with acute coronary syndrome (ACS), a smoking-related condition, would be more prone to misclassify themselves as ex-smokers, because of pressure to quit. We compared patients admitted with ACS with a general population survey conducted in the same country at a similar time. We determined whether ACS patients who classified themselves as ex-smokers ($n=635$) were more likely to have cotinine levels suggestive of smoking deception than self-reported ex-smokers in the general population ($n=289$). On univariate analysis, the percentage of smoking deceivers was similar among ACS patients and the general population (11% vs. 12%, $p=.530$). Following adjustment for age, sex and exposure to environmental tobacco smoke, ACS patients were significantly more likely to misclassify themselves (adjusted $OR=14.06$, 95% CI 2.13–93.01, $p=.006$). There was an interaction with age whereby the probability of misclassification fell significantly with increasing age in the ACS group (adjusted $OR=0.95$, 95% CI 0.93–0.97, $p<.001$), but not in the general population. Overall, smoking deception was more common among ACS patients than the general population. Studies comparing patients with cardiovascular disease and healthy individuals risk introducing bias if they rely solely on self-reported smoking status. Biochemical confirmation should be undertaken in such studies.

Introduction

Many studies have compared patients with cardiovascular disease and healthy individuals in terms of smoking status. Typically, smoking status is based on self-classification without biochemical confirmation

Jill P. Pell, Stuart M. Cobbe, BHF Glasgow Cardiovascular Research Centre, University of Glasgow, Glasgow; Sally J. Haw, NHS Health Scotland and University of Edinburgh, Edinburgh; David E. Newby, Centre for Cardiovascular Science, University of Edinburgh, Edinburgh; Alastair C. H. Pell, Monklands Hospital, Airdrie; Keith G. Oldroyd, William Borland, Western Infirmary, Glasgow; David L. Murdoch, Southern General Hospital, Glasgow; Stuart D. Pringle, MD, Ninewells Hospital, Dundee; Frank G. Dunn, Cardiac Department, Stobhill Hospital, Glasgow; Paul D. MacIntyre, Royal Alexandra Hospital, Paisley; Timothy J. Gilbert, Hairmyres Hospital, East Kilbride; Colin M. Fischbacher, Information Services Division, NHS National Services, Edinburgh.

Correspondence: Jill Pell, Room C214, BHF Glasgow Cardiovascular Research Centre, University of Glasgow, Glasgow G12 8TA, UK. Tel: +44 (0) 141 330 3473; Fax: +44 (0) 141 330 6955; E-mail: j.pell@clinmed.gla.ac.uk

because this approach is cheap and easy, and does not necessarily require face-to-face contact. Therefore, it is important to confirm not only that this method is valid but also that there is no difference in self-reporting between these groups resulting in bias. The validity of self-reporting is known to be subject to a social desirability bias, whereby smokers under the greatest pressure to quit are more likely to deliberately misclassify themselves as nonsmokers. If patients with a smoking-related condition, such as cardiovascular disease, are more likely to misclassify themselves, studies comparing patients with cardiovascular disease and healthy individuals will inevitably introduce bias.

Previous studies have examined the validity of self-reported smoking status either among healthy individuals or among those with disease. Meta-analysis suggests that self-reports are reasonably accurate in most general population studies, with a pooled sensitivity and specificity of 88% and 89%,

respectively (Partick et al., 1994). The negative predictive value in the general population is around 96% (Newell, Girgis, Sanson-Fisher & Savolainen, 1999). Studies examining patients with newly diagnosed cardiovascular disease, or a previous cardiovascular event, report negative predictive values of 81–96% (Atterbring, Herlitz, Berndt, Karlsson & Hjalmarson, 2001; Jarvis, Tunstall-Pedoe, Feyerabend, Vesey & Saloojee, 1987; Jarvis, Primatesta, Erens, Feyerabend & Bryant, 2003; Wilson, Wallston, King, Smith & Heim, 1993; Woodward & Tunstall-Pedoe, 1992).

Current smokers who deliberately misclassify themselves as nonsmokers are traditionally termed "smoking deceivers." There has been only one previous study that compared deception rates between those with cardiovascular disease and the general public (Woodward & Tunstall-Pedoe, 1992). Woodward and Tunstall-Pedoe (1992) used data from the Scottish Heart Health Study to compare deception rates among 320 male nonsmokers with diagnosed coronary heart disease, 819 with undiagnosed coronary heart disease, and 4,229 with no coronary heart disease. They demonstrated deception rates of 8%, 2%, and 2%, respectively ($p<0.001$). The trend was similar in women. Their results suggested that having a smoking-related condition resulted in an increased likelihood of deceiving. However, they compared only crude deception rates. There was no adjustment for potential confounding because of age differences, and they had no information on exposure to environmental tobacco smoke (ETS). Also, they were unable to distinguish between ex- and never-smokers. Previous studies suggest that smoking deception varies with sex, age, and context. A number of studies have suggested that men are more likely to be smoking deceivers than women (Assaf, Parker, Lapane, McKeeney & Carleton 2002; Bowlin, Morrill, Nafziger, Lewis & Pearson, 1996; Newell et al., 1999). However, this finding is disputed by others (Morabia, Berstein, Curtin & Berode, 2001). In a meta-analysis, Patrick et al. (1994) reported higher deception rates among the young, which they attributed to age differences in social undesirability. The aim of our study was to determine whether patients admitted with acute coronary syndrome (ACS) were more likely to misclassify themselves than the general public, and whether this association was independent of age, sex, and ETS exposure.

Methods

General population

Data from the general population were collected as part of the Health Education Population Survey (HEPS) (NHS Health Scotland, 2004). HEPS is a pan-Scotland survey undertaken periodically since 1996 to monitor health-related knowledge and behavior. We used data from the survey undertaken over 7 months from September 2005. Households were selected from the Postal Address File using rolling, multi-stage, clustered random sampling. Participants were then selected from households using the first birthday method. Interviewers were not involved in the health care of participants. An interviewer-administered questionnaire, on a range of health behaviors, was completed during a face-to-face interview in participants' homes. The smoking module included self-reported smoking status, date of cessation, use of nicotine replacement therapy and exposure to ETS. Samples of unstimulated saliva were collected using salivettes (cotton wool rolls). A salivette was placed in the mouth for 3–5 min without chewing until wet with saliva and then stored in individual containers at 3°C until testing.

ACS patients

Data were collected prospectively on all patients admitted with ACS to nine acute Scottish hospitals over 10 months from May 2005. These hospitals account for 49% of all ACS admissions in Scotland. Interviewers were not involved in the health care of participants. During their hospital admission, participants completed an interviewer-administered questionnaire that included questions on self-reported smoking status, date of cessation, use of nicotine replacement therapy, and exposure to ETS. The wording of these questions was consistent with the HEPS questionnaire. Residual serum from the clinical blood samples taken on admission was used to perform cotinine assays. The samples were centrifuged and stored locally at −20°C before being transported on dry ice to the central laboratory for cotinine assay.

Cotinine assay

In both studies, cotinine was analyzed by ABS Laboratories Ltd in London, United Kingdom, using gas chromatography with a specific nitrogen/phosphorous detector GC-NPD (Feyerabend & Russell, 1990). Cotinine and the internal standard 5-methyl cotinine were extracted using dichloroethane from a 100-μl sample after alkalization using sodium hydroxide. The lower limit of detection was 0.1 ng/ml.

Cotinine was assayed in saliva in HEPS and serum in ACS patients. In a random sample of 605 members of the general population, Jarvis et al. demonstrated that saliva cotinine levels were 25% higher than in serum/plasma, and this ratio applied both at the low

levels attributable to ETS and across the range of active smoking values (Jarvis et al., 2003). Therefore we applied cut-offs of 12 ng/ml for serum and 15 ng/ml for saliva as our objective measure of current versus nonsmokers (Jarvis et al., 1987).

Definitions

All participating hospitals routinely measured troponin levels on all patients admitted with suspected ACS. Therefore, ACS was defined as a patient admitted as an emergency with cardiac chest pain, in whom the troponin level was raised in the absence of a noncardiac cause such as renal failure, thromboembolic disease, myocarditis or coronary revascularization. This definition was chosen because it was unambiguous and could be applied easily and consistently across all patients in all hospitals. Nonsmokers were defined as either ex- or neversmokers. Smoking deceivers were defined as people who classified themselves as nonsmokers but whose cotinine level was above the cut-off (12 ng/ml for serum and 15 ng/ml for saliva). Cotinine levels do not fall to those of a nonsmoker until around 4 days of abstinence from smoking (Gilbert, 1993). Therefore, we excluded from our study ex-smokers who had quit smoking less than 1 month prior to providing the sample for cotinine assay.

Statistical methods

We used two-sided tests throughout so as not to prejudge the direction of any difference. In testing for differences in summary statistics, we used Mantel–Haenszel tests, chi-square tests for trend, and Mann–Whitney U tests for binary, ordinal, and continuous data, respectively. We defined a dichotomous dependent variable on the basis of whether the cotinine level exceeded the cut-off value or not, and then used multivariate logistic regression analysis to identify the factors associated with this outcome. We tested for interactions between disease status and the other covariates. We report actual p values to three decimal places.

Results

Of the 1,735 patients admitted to hospital with ACS, 666 were excluded because they were current smokers, and 8 because they were taking nicotine replacement therapy. Of the remaining 1,061, 635 (60%) classified themselves as ex-smokers and 426 (40%) as never-smokers. The HEPS survey provided questionnaire and cotinine data on 1,061 members of the general public. Of these, 309 were excluded because they were current smokers and 6 because they were taking nicotine replacement therapy. Of

the remaining 746, 289 (39%) classified themselves as ex-smokers and 457 (61%) as never-smokers.

Smoking deceivers were more likely to classify themselves as ex-smokers then never-smokers. Among self-reported nonsmokers, 82% of those above the cotinine cut-off classified themselves as ex-smokers, compared with only 49% below the cut-off ($p<.001$). Overall, only 22 (3%) self-reported never-smokers had cotinine levels above the cut-off (smoking deceivers) and there was no significant difference between patients admitted with ACS and the general population (2% vs. 3%, $p=.500$). There was no significant association between disease status and likelihood of deception on either univariate ($OR=1.34$, 95% CI 0.57–3.18, $p=.501$) or multivariate ($OR=1.90$, 95% CI 0.62–5.84, $p=.262$) logistic regression analysis.

Among self-reported ex-smokers, 103 (11%) had a cotinine level above the threshold (smoking deceivers), with no significant difference between ACS patients and the general population on univariate analysis (11% vs. 12%, $p=.530$). However, the characteristics of self-reported ex-smokers differed between ACS patients and the general population. In comparison with the general population, patients admitted with ACS tended to be older (median age 73 vs. 54 years, MWU $p<.001$) and were more likely to be male (66% vs. 45%, χ^2 test $p<.001$). Among ex-smokers as a whole, 50% of the reported total duration of ETS was related to exposure in the subject's own home. However, there was no statistically significant difference between ACS patients and the general public in terms of the number of household members who smoked or reported hours of ETS exposure. Following adjustment for demographic differences and exposure to ETS, self-reported ex-smokers admitted with ACS were significantly more likely overall to have a cotinine level above the cut-off level than the general population (adjusted $OR=14.06$, 95% CI 2.13–93.01, $p=.006$) (Table 1).

There was a significant interaction between ACS admission and age (Table 1, Figure 1). Among self-reported ex-smokers admitted with ACS, the

Table 1. Multiple logistic regression analysis of the determinants of a cotinine level above cut-off among self-reported ex-smokers.

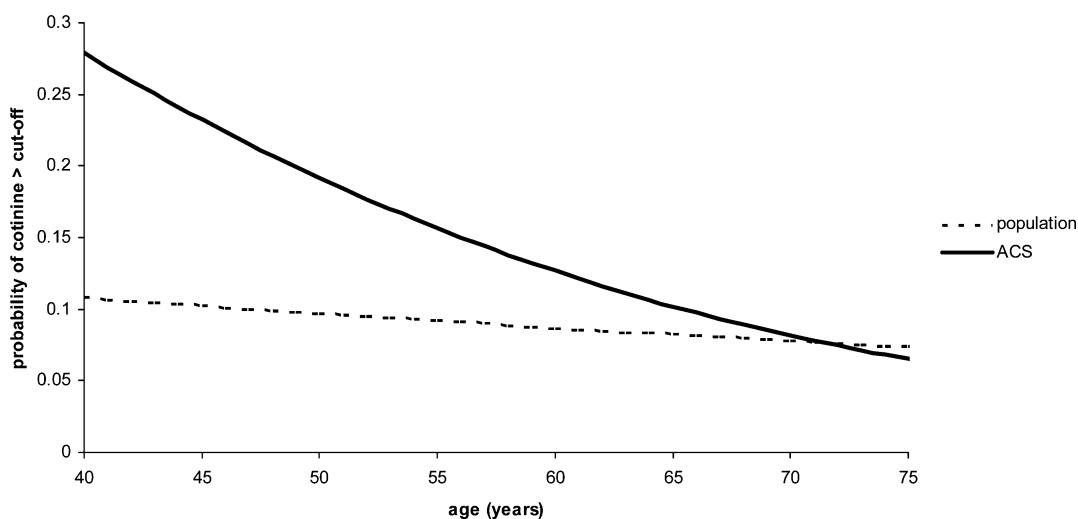|	OR	95% CI	p value
Disease status			
General population	1.00	—	—
Acute coronary syndrome	14.06	2.13–93.01	.006
Sex			
Male	1.00		
Female	1.03	0.65–1.61	.909
Age (years)	0.99	0.97–1.01	.320
Number of smokers in household	1.76	1.24–2.51	.002
Weekly exposure to ETS (hr)	1.01	1.00–1.02	.159
Age × disease status	0.96	0.93–0.99	.021

Figure 1. Probability of self-reported ex-smokers having a cotinine above the cut-off by age. Example is for a man with no current smokers in the same household and total ETS exposure per week equivalent to the median (1 hr).

likelihood of cotinine levels being above the cut-off fell significantly with increasing age (adjusted $OR=0.95$, 95% CI 0.93–0.97, $p<.001$). By contrast, the decline in likelihood of deception with increasing age was nonsignificant in the general population. As a result, the difference in risk of deception between ACS patients and the general population was most pronounced among younger patients and reduced with increasing age (Figure 1).

Discussion

Our results suggested that deception was more likely to take the form of misclassification as an ex-smoker than never-smoker. This is not surprising, since smoking history is more difficult to conceal than current smoking habit. Overall, smoking deception was more common among ACS patients than the general population. An observed difference in the deception rates among patients with coronary heart disease may be artifact or a real finding. We believe that our findings are unlikely to be artifactual. Cotinine is the major metabolite of nicotine, and is the analyte of choice for measuring exposure to cigarette smoke (both active and passive) and for discriminating between smokers and nonsmokers (Dhar, 2004). It has a sensitivity of 96%–97% and a specificity of 99%–100% (Jarvis et al., 1987). It is specific to tobacco, is directly proportional to the quantity of nicotine absorbed, and the presence of other compounds does not interfere with estimation of the marker. It has an appropriate half-life (18–20 hr), has relatively constant levels during the day, is amenable to estimation in a number of body fluids (blood, saliva, and urine), with concentrations highly correlated between all three fluids (Jarvis et al., 1987), and is available at concentrations that can be

easily quantified. The same laboratory was used to assay cotinine in both studies, and used the gold standard method of gas chromatography with a specific nitrogen/phosphorous detector GC-NPD (Feyerabend & Russell, 1990). We excluded from our study participants on nicotine replacement therapy, snuff or chewing tobacco, and those who had quit smoking within the previous month. Cotinine was assayed in saliva in HEPS and in serum for ACS patients. Ideally, cotinine would have been assayed using the same medium in both studies. However, Jarvis et al. (2003) demonstrated that applying a ratio of 1:1.25 for serum/plasma:saliva produces a good correlation across the whole range of cotinine levels, including both ETS and active smoking. Furthermore, in our study, very few participants had cotinine values around the cut-off values. Across both the general population and ACS patients combined, only two participants had a cotinine value in the range 10–20 ng/ml. Therefore, our results are not sensitive to the cut-off values applied.

Forewarning participants that smoking status will be confirmed biochemically may result in modification of smoking behavior to avoid detection. However, Jarvis et al. (1987) demonstrated that 97% of current smokers smoked on the day of their test. Therefore, deliberate avoidance of detection would require 2–4 days of atypical abstinence. Among ACS patients, cotinine was assayed on residual clinical samples obtained on admission and prior to recruitment to the study. In HEPS, the salivette was used on the same day that information was recorded on self-reported smoking status. Therefore, neither study afforded the opportunity to modify behavior to appear consistent with self-reported status. Deception rates vary according to whether information is collected by face-to-face

interview or telephone interview and whether the interviewer in involved in the participant's clinical management (Galobardes et al., 1998; Luepker, Pallonen, Murray, Pirie, 1989). However, both studies employed face-to-face interviews with research staff not involved in the care of participants. Both studies recruited participants over a similar period, and both completed recruitment prior to the Scottish ban on smoking in public places, which may have impacted on social desirability.

Etzel (1990) suggested that there may be difficulty using cotinine to distinguish between very light active smoking and regular passive smoking. However, Lee (1988) reported that even heavy exposure to passive smoke rarely results in plasma cotinine levels above 10ng/ml. We were able to demonstrate that there were no differences between ex-smokers admitted with ACS and ex-smokers in the general population in terms of household and overall exposure to ETS and we adjusted for ETS exposure when comparing deception rate among ACS patients and the general population. We included age in our model and demonstrated a significant interaction between age and deception. Metabolism of cotinine is significantly slower in black smokers than white smokers. However, well over 99% of the Scottish population is white so we did not need to adjust for race.

A real difference among those with cardiovascular disease is plausible because of a social acceptability bias (Partick et al., 1994). Studies have demonstrated a significantly higher deception rate among people attending a smoking cessation programme and among others advised to quit smoking (Murray, Connett, Lauger & Voelker 1993; Ohlin, Lundh & Westling, 1976; Sillett, Wilson, Malcolm & Ball, 1978). People with a smoking-related disease are under greater pressure to quit. They may feel guilty that their condition is self-inflicted and that failure to quit demonstrates a lack of gratitude to health care workers. Alternatively, they may be concerned that treatment will be withheld if they fail to comply with smoking advice.

In general, self-reported smoking status is a satisfactory tool for large-scale population studies where biochemical confirmation is not feasible or is too expensive. However, when comparing patients with a smoking-related condition and healthy individuals, we need to be aware that the former are under greater pressure to quit smoking and therefore are more likely to deliberately misclassify themselves as nonsmokers. Some investigators have employed a ''bogus pipeline'' approach, in which study participants are advised incorrectly that their smoking status will be confirmed by either corroboration or biochemical tests. This may be helpful in reducing deception, or may simply result in short-term modification of smoking behavior. Ideally, biochemical confirmation should be used in all participants in studies comparing people with and without a smoking-related condition. If this is not feasible, biochemical validation should, at least, be undertaken in a sub-group to either confirm that the results are free from bias, or to quantify and correct for bias where it exists.

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