Effectiveness of biomedical risk assessment as an aid for smoking cessation: a systematic review

Raphaël Bize, Bernard Burnand, Yolanda Mueller and Jacques Cornuz

Tob. Control 2007;16;151-156
doi:10.1136/tc.2006.017731

Updated information and services can be found at:
http://tobaccocontrol.bmj.com/cgi/content/full/16/3/151

These include:

References
This article cites 45 articles, 8 of which can be accessed free at:
http://tobaccocontrol.bmj.com/cgi/content/full/16/3/151#BIBL

Rapid responses
You can respond to this article at:
http://tobaccocontrol.bmj.com/cgi/eletter-submit/16/3/151

Email alerting service
Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

Notes

To order reprints of this article go to:
http://www.bmjjournals.com/cgi/reprintform

To subscribe to Tobacco Control go to:
http://www.bmjjournals.com/subscriptions/
Effectiveness of biomedical risk assessment as an aid for smoking cessation: a systematic review

Raphaël Bize, Bernard Burnand, Yolanda Mueller, Jacques Cornuz

Objective: To determine the efficacy of biomedical risk assessment (eg, exhaled carbon monoxide (CO), or genetic susceptibility to lung cancer) as an aid for smoking cessation.


Study selection: Randomised controlled smoking cessation interventions using biomedical tests with at least 6 months follow-up.

Data extraction: Two reviewers independently screened all search results (titles and abstracts) for possible inclusion. Each reviewer then extracted data from the selected studies, and assessed their methodological quality based on the CONSORT (Consolidated Standards of Reporting Trials) statement criteria.

Data synthesis: Of 4049 retrieved references, eight trials were retained for data extraction and analysis.

Three trials isolated the effect of exhaled CO on smoking cessation rates resulting in the following ORs and 95% CIs: 0.73 (0.38 to 1.39), 0.93 (0.62 to 1.41) and 1.18 (0.84 to 1.64). Measurement of exhaled CO and spirometry were used together in three trials, resulting in the following ORs (95% CI): 0.60 (0.25 to 1.46), 2.45 (0.73 to 8.25) and 3.50 (0.88 to 13.92). Spirometry results alone were used in one other trial with an OR (95% CI) of 1.21 (0.60 to 2.42). Ultrasonography of carotid and femoral arteries performed on light smokers gave an OR (95% CI) of 3.15 (1.06 to 9.31).

Conclusions: Scarcity and limited quality of the current evidence does not support the hypothesis that biomedical risk assessment increases smoking cessation as compared with the standard treatment.
<table>
<thead>
<tr>
<th>Study</th>
<th>Methods</th>
<th>Participants</th>
<th>Interventions</th>
<th>Outcomes</th>
<th>Notes</th>
<th>Allocation concealment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Audrain et al, 1997&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Setting: smoking clinic, USA Design: randomised controlled trial, two intervention and one control groups Recruitment: lay press Selected: advertisement: free smoking-cessation study Randomisation: not detailed</td>
<td>550 smokers (defined as &gt;5 cpd for &gt;1 year) out of 1104 eligible</td>
<td>Intervention 1: exposure biomarker feedback (CO) and 60 min quit-smoking consultation Intervention 2: susceptibility biomarker feedback (CYP2D6), exposure biomarker feedback (CO) and 60 min quit-smoking consultation Control: 60 min Quit-smoking consultation (quit plan, gaining support)</td>
<td>Definition of abstinence: 30-day point prevalence Duration of follow-up: 12 months</td>
<td>Per protocol analysis. Distribution of baseline 550 participants among the three groups not reported</td>
<td>Unclear</td>
</tr>
<tr>
<td>Bovet et al, 2002&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Setting: Seychelles Heart Study II Design: randomised controlled trial Recruitment: age- and sex-stratified sample drawn from general population of Mahé, invited by letter to a cardiovascular risk factor survey Selected: last 155 participants to the Seychelles Heart Study II Randomisation: pre-established random sequences of numbers matched to rank of arrival. Assessors blinded</td>
<td>Mean age 46 years 15% female</td>
<td>Intervention: ultrasonography of carotid and femoral arteries. Smokers with &gt;1 plaque given two photographs of their plaque and explanation along with quit-smoking counselling Control: quit-smoking counselling</td>
<td>Definition of abstinence: 7-day point prevalence Duration of follow-up: 6 months Biochemical validation of non-smokers: none</td>
<td>Two participants lost to follow-up not included in analysis</td>
<td>Unclear</td>
</tr>
<tr>
<td>Jamrozik et al, 1984&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Setting: six general practices, UK Design: randomised controlled trial Recruitment: clinic, first visit Selected: outpatients Randomisation: according to the day of attendance, balanced over 4-weeks Assessors blinded</td>
<td>2110 smoker (defined as a person admitting to smoking cigarettes) out of 6052 screened</td>
<td>Intervention: demonstration of patient exhale CO, verbal advice and booklet Control: verbal advice and booklet</td>
<td>Definition of abstinence: point prevalence without mention of duration Duration of follow-up: 12 months Biochemical validation of non-smokers: urinary cotinine in a sample (41%) of self-reported non-smokers</td>
<td>OR based on unvalidated data Inadequate</td>
<td>Inadequate</td>
</tr>
<tr>
<td>Risser and Belcher 1990&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Setting: US Veterans Administration Demonstration Project. Design: randomised controlled trial Recruitment: veterans attending a health promotion clinic Selected: responding to mailed invitations for health promotion. Same second visit Randomisation: not detailed Assessors blinded</td>
<td>Mean age 53.7 years (55.5 vs 51.7 years) 4% female</td>
<td>Intervention: spirometry, exhaled CO, discussion of pulmonary symptoms and control intervention Control: 50 min educational intervention, review of self-help manual, invitation to a nine-session one-to-one counselling programme</td>
<td>Definition of abstinence: point prevalence without mention of duration Duration of follow-up: 12 months Biochemical validation of non-smokers: exhaled CO&lt;10 ppm</td>
<td>Unclear</td>
<td>Unclear</td>
</tr>
<tr>
<td>Study</td>
<td>Methods</td>
<td>Participants</td>
<td>Interventions</td>
<td>Outcomes</td>
<td>Notes</td>
<td>Allocation concealment</td>
</tr>
<tr>
<td>---------------------</td>
<td>-------------------------------------------------------------------------</td>
<td>---------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Sanders et al, 1989</td>
<td>Setting: 11 UK general practices</td>
<td>751 participants out of 4330 identified smokers (self-defined)</td>
<td>Intervention: exhaled CO measure, discussion of significance and control intervention</td>
<td>Definition of abstinence: point prevalence without mention of duration</td>
<td>Duration of follow-up: 12 months</td>
<td>Inadequate</td>
</tr>
<tr>
<td></td>
<td>Design: randomised controlled trial</td>
<td>Mean age 38.5 years</td>
<td>Control: counselling by practice nurse, written material given and offer of a follow-up appointment</td>
<td>Biochemical validation of non-smokers: urinary cotinine. Cut-off not reported</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recruitment: screening of all outpatients</td>
<td>Other characteristics not mentioned</td>
<td>Therapist: practice nurse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Selected: outpatients and who made appointment for health check</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Randomisation: by day of attendance on a 1:2 basis. Desktop card reminding doctors of right allocation. 120 wrongly allocated patients, excluded from further analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segnan et al, 1991</td>
<td>Setting: 44 general practices, Italy</td>
<td>923 included out of 1009 screened. Smoker definition not given</td>
<td>Intervention: spirometry prescription and control intervention</td>
<td>Definition of abstinence: 7-day point prevalence</td>
<td>Duration of follow-up: 12 months</td>
<td>Adequate</td>
</tr>
<tr>
<td></td>
<td>Design: randomised controlled trial</td>
<td>Age: 20.1% &lt;31 years; 28.0% 31–40 years; 26.8% 41–50 years; 25.0% &gt;50 years</td>
<td>Control: repeated counselling with reinforcement sessions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recruitment: screening of outpatients on specific days. Selected: outpatients</td>
<td>cpd: 16.7% &lt;10 cpd; 55.2% 11–20 cpd; 28.1% &gt;20 cpd</td>
<td>(two other groups not used in our comparison: minimal intervention and repeated counselling and nicotine gum)</td>
<td>Biochemical validation of non-smokers: urinary cotinine &lt;100 ng/mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Randomisation: sequence of random numbers, sealed envelopes</td>
<td>38% female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Therapist: physician</td>
<td>51% reporting symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Therapist: study staff</td>
<td>Mean cpd: 20.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Assessors blinded</td>
<td>Mean age 38.5 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age 31–40 years</td>
<td>Mean pack-years: 28.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Consecutively (time of check-in). Odd-numbered = intervention</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sippel et al, 1999</td>
<td>Setting: two primary care clinics, USA</td>
<td>205 included out of 360 smokers (self-defined)</td>
<td>Intervention: spirometry and exhaled CO and control intervention</td>
<td>Definition of abstinence: sustained from quit date to the time of follow-up</td>
<td>Duration of follow-up: 9 months</td>
<td>Inadequate</td>
</tr>
<tr>
<td></td>
<td>Design: randomised controlled trial with formal estimation of sample size</td>
<td>Mean age 38.5 years</td>
<td>Control: counselling according to transtheoretical model of change, written material and NRT encouraged if prepared to stop</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recruitment: all smokers among outpatients</td>
<td>62.5% female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Selected: outpatients</td>
<td>Mean cpd: 20.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Randomisation: questionnaires numbered consecutively (time of check-in).</td>
<td>Mean cpd 20.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Odd-numbered = intervention</td>
<td>Mean pack-years: 28.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Assessors blinded</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>SoC: 36% in preparation stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walker and Franzini</td>
<td>Setting: stop-smoking clinic, USA</td>
<td>64 out of 141 eligible (smoker, self-defined)</td>
<td>Intervention 1: exhaled CO and spirometry feedback, and taste satiation</td>
<td>Definition of abstinence: “smoking not &gt;1 cigarette in the past 10 days”</td>
<td>Duration of follow-up: 6 months</td>
<td>Unclear</td>
</tr>
<tr>
<td></td>
<td>Therapist: study staff</td>
<td>Mean age: 35.5 years</td>
<td>Intervention 2: exhaled CO and spirometry feedback and focused smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Design: 2x2x2 randomised controlled trial</td>
<td>59% female</td>
<td>Booster sessions for half of each intervention group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recruitment: public service announcement and media advertising</td>
<td>Mean cpd 29.2</td>
<td>Control 1: taste satiation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Selected: those responding to advertising, paying US$45</td>
<td>Mean 3.4 previous quit attempts</td>
<td>Control 2: focused smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Randomisation: not detailed</td>
<td>Therapist: first author</td>
<td>Booster sessions for half of each control group</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CO, carbon monoxide; cpd, cigarettes per day; NRT, nicotine replacement therapy; ppm, parts per million; SoC, stage of change.
with 95% CIs. An OR >1 favours the intervention group. If it seemed appropriate, the results were pooled using a Maentel–Haenszel fixed-effects model.

RESULTS
We identified 22 trials for possible inclusion out of 4049 references. Eleven studies were excluded because the effect of biomedical risk assessment could not be isolated,20–30 one because smoking cessation was not considered as an outcome,31 one because the biomedical risk assessment was not carried out on the smoker himself but on his or her children32 and one because the full-text article could not be found.33 One of the excluded trials34 generated two reports.35 36

We therefore analysed data from eight trials (table 1). One of them3 tested two interventions (CO measurement and the combination of the latter with feedback about genetic susceptibility), giving rise to three possible comparisons of effectiveness. Three trials tested the effect of exhaled CO measurements alone,35 36 37 three trials tested the combination of exhaled CO measurement and spirometry,37–39 one trial tested the effect of CO and feedback about genetic susceptibility,1 one trial tested spirometry alone,40 one trial tested the effect of undergoing an ultrasonography of carotid and femoral arteries with photographic demonstration of atherosclerotic plaques when present41 and one trial tested feedback about genetic susceptibility to lung cancer.3 The mean number of cigarettes smoked per day varied between 11.9 and 29.2 and was highest in the trials set in a “smoking clinic”.42

Only one of the eight trials reported an adequate randomisation procedure.40 Only three studies explicitly mentioned that assessors were blinded to allocation at the time of outcome determination.37 38 41 Only one study proposed a formal estimation of sample size before recruitment.43 Biochemical validation of smoking cessation was adequately used in four studies.36 37 39 40 Participation rates (ie, the proportion of those approached who agreed to take part in the trial) were seldom recorded. In two studies,35 36 it was not possible to determine the initial allocation of the participants who were subsequently lost to follow-up, and analysis had to be performed per protocol.

Figure 1 shows the ORs and 95% CIs from the two trials using exhaled CO in a primary care setting as a way to motivate smokers to quit.35 36 These two studies were similar enough in terms of recruitment, intervention and setting to allow the pooling of data. \( \chi^2 \) test did not show evidence for significant heterogeneity. There was no evidence of a significant benefit from these pooled studies (Mantel–Haenszel fixed-effect OR 1.07, 95% CI 0.83 to 1.39).

Figure 2 shows the individual ORs and 95% CIs from all the included interventions. Three studies isolated the effect of exhaled CO measurement on smoking cessation rate35 36 with
DISCUSSION

Owing to the scarcity of evidence of sufficient quality, we could make no definitive statements about the effectiveness of biomedical risk assessment as an aid for smoking cessation. Existing evidence of lower quality does not, however, support the hypothesis that biomedical risk assessment increases smoking cessation as compared with the standard treatment.

Only two studies were similar enough in terms of recruitment, setting and intervention to allow pooling of data and meta-analysis. Their combined results further tended towards the null hypothesis. The external validity of the only study with a statistically significant positive OR can be questioned as the sample was made up predominantly of male light smokers (average 10–12 cigarettes a day).

Other studies identified by our search strategy did not isolate the specific effect of biomedical feedback. Two of these studies demonstrated an OR significantly favouring the intervention group rather than the control group. Demonstration of smokers’ child’s exposure to environmental tobacco smoke by measuring the child’s urinary cotinine level was used in another trial with an OR (95% CI) of 0.15 (0.01 to 2.89). We excluded this study from our analysis, because, it seemed to us that providing biomarker feedback about someone else’s health (even one’s own children) would act differently and may not contribute to counteracting the hypothesised personal optimistic bias.

Smoking cessation was, moreover, documented as a secondary outcome in this study, as the primary outcome was a smoking ban in the home. In any event, this trial did not show a positive effect; the study had low power to detect an effect and its quality was limited. One study identified by McClure as “in press” seems never to have been published, and several attempts to contact the authors failed to provide us with more detailed information.

An earlier non-systematic review was conducted on the use of biomarkers in smoking cessation. The aim of this work was to review the theoretical rationale and the empirical evidence regarding this practice. Focus was, therefore, not specifically directed at the assessment of the efficacy of biomarker feedback as a way to increase smoking cessation. Therefore, the review included non-randomised trials, comparing the effect of abnormal test results versus normal test results rather than test versus no tests, and trials reporting outcomes other than smoking cessation. Four studies mentioned by McClure were also retained in our review. We identified four more trials for our review. When focusing on efficacy data, McClure concluded that biomarkers feedback may enhance the likelihood of cessation, because a trend for increased abstinence was found in three randomised trials. The fact that two of these trials are subject to major methodological limitations (small samples, inadequate randomisation procedures), and that the report of Hoffman remains unpublished, calls for great caution in drawing such conclusions.

In most of the studies included in the current review, the biomedical testing component was added to intensive quit-smoking sessions, with counselling lasting up to 60 min and completed by written material and reinforcement sessions or follow-up telephone calls. The incremental effect of biomedical risk assessment might have been diluted by the high intensity of the standard care used. It is also possible that the changes in motivational stages induced by biomedical risk assessment are too subtle to be characterised as directly leading to a successful quit attempt. Another possible explanation for the absence of effectiveness of biomedical risk assessment provided in addition to counselling could be the potentially counterproductive effect of communicating normal results to smokers. Only two included studies provided some insight about smoking cessation rates according to test results. Sippel et al did not find any correlation between smoking cessation and abnormal spirometry results, whereas Bovet et al found a non-significant lower smoking cessation rate among participants without plaques at ultrasonography compared with participants who did not undergo ultrasonography. Similarly, whether the presence of smoking-related symptoms may modify the effect of biomedical feedback is unknown. These particular questions, and the way to communicate normal test results should be explored in future trials.

ACKNOWLEDGEMENTS

We thank Olivier Terraz of the Institute for Social and Preventive Medicine, University of Lausanne, for his assistance in retrieving and selecting references identified by our search strategy, and Alvine Bissery of the same institution for her statistical expertise. We also thank Jon Britton and Jonathan Foulds for their helpful suggestions on the protocol, and Andy McEwen and Lion Shahab for constructive comments on the earlier drafts of this review.

AUTHORS’ AFFILIATIONS

Raphaël Bize, Jacques Cornuz, Department of Ambulatory Care and Community Medicine, University of Lausanne, Lausanne, Switzerland

www.tobaccocontrol.bmj.com
not necessarily shared by The Cochrane Collaboration.

The results of a Cochrane Review can be interpreted differently, depending on people’s perspectives and circumstances. Please consider the conclusions presented carefully. They are the opinions of review authors, and are not necessarily shared by The Cochrane Collaboration.

REFERENCES