Biomedical risk assessment as an aid for smoking cessation (Review)

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[Intervention Review]

Biomedical risk assessment as an aid for smoking cessation

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ABSTRACT

Background

A possible strategy for increasing smoking cessation rates could be to provide smokers who have contact with healthcare systems with feedback on the biomedical or potential future effects of smoking, e.g. measurement of exhaled carbon monoxide (CO), lung function, or genetic susceptibility to lung cancer.

Objectives

To determine the efficacy of biomedical risk assessment provided in addition to various levels of counselling, as a contributing aid to smoking cessation.

Search strategy

We systematically searched the Cochrane Collaboration Tobacco Addiction Group Specialized Register, Cochrane Central Register of Controlled Trials 2008 Issue 4, MEDLINE (1966 to January 2009), and EMBASE (1980 to January 2009). We combined methodological terms with terms related to smoking cessation counselling and biomedical measurements.

Selection criteria

Inclusion criteria were: a randomized controlled trial design; subjects participating in smoking cessation interventions; interventions based on a biomedical test to increase motivation to quit; control groups receiving all other components of intervention; an outcome of smoking cessation rate at least six months after the start of the intervention.

Data collection and analysis

Two assessors independently conducted data extraction on each paper, with disagreements resolved by consensus. Results were expressed as a relative risk (RR) for smoking cessation with 95% confidence intervals (CI). Where appropriate a pooled effect was estimated using a Mantel-Haenszel fixed effect method.

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Main results

We included eleven trials using a variety of biomedical tests. Two pairs of trials had sufficiently similar recruitment, setting and interventions to calculate a pooled effect; there was no evidence that CO measurement in primary care (RR 1.06, 95% CI 0.85 to 1.32) or spirometry in primary care (RR 1.18, 95% CI 0.77 to 1.81) increased cessation rates. We did not pool the other seven trials. One trial in primary care detected a significant benefit of lung age feedback after spirometry (RR 2.12; 95% CI 1.24 to 3.62). One trial that used ultrasonography of carotid and femoral arteries and photographs of plaques detected a benefit (RR 2.77; 95% CI 1.04 to 7.41) but enrolled a population of light smokers. Five trials failed to detect evidence of a significant effect. One of these tested CO feedback alone and CO + genetic susceptibility as two different intervention; none of the three possible comparisons detected significant effects. Three others used a combination of CO and spirometry feedback in different settings, and one tested for a genetic marker.

Authors' conclusions

There is little evidence about the effects of most types of biomedical tests for risk assessment. Spirometry combined with an interpretation of the results in terms of 'lung age' had a significant effect in a single good quality trial. Mixed quality evidence does not support the hypothesis that other types of biomedical risk assessment increase smoking cessation in comparison to standard treatment. Only two pairs of studies were similar enough in term of recruitment, setting, and intervention to allow meta-analysis.

PLAIN LANGUAGE SUMMARY

Does giving people who smoke feedback about the effects of smoking on their body help them to quit

Biomedical risk assessment is the process of giving smokers feedback on the physical effects of smoking by physiological measurements (for example: exhaled carbon monoxide measurement or lung function tests). It was thought to be a possible way of increasing quit rates. In one study, smokers who had their lung function tested and the results explained in terms of their lung age compared to a non smoker of the same age were more likely to quit than people given the same test but without the explanation. Mixed quality evidence does not suggest that other types of biomedical risk assessment increases smoking cessation compared with standard treatments.

BACKGROUND

Smoking is the leading cause of preventable mortality and morbidity in industrialized countries (Phillips 1995; Sargeant 2001; Yach 2000), and increasingly the smoking epidemic is affecting developing countries. This is particularly true for vascular and respiratory diseases, as well as for cancer (DHHS 2004; Doll 2004). Stopping smoking prolongs life and reduces morbidity (DHHS 1990). Despite increasing scientific knowledge about health hazards due to cigarette consumption, there is, in many countries, an increase in prevalence among young people (Gmel 2000; Lewitt 1997). The gap between knowledge and smoking cessation has been attributed, in part, to smokers' underestimation of their personal risks of smoking-related illness (Lerman 1993; Romer 2001). Although many smokers who are successful in quitting do so on their own (Lancaster 2005a; Schwartz 1987), an increasing proportion use specific aids to support a quit attempt.

Evidence is growing on how to help smokers to quit (Kottke 1988; Lancaster 2005b; Stead 2008; Stead 2008a; TUDCPGP 2000; West 2000). Interventions that help include individual or group counselling, pharmacological therapies and possibly some forms of self-help materials. Another possible strategy for increasing quit rates might be to provide feedback on the physical effects of smoking by physiological measurements. We can conceptually distinguish, in this respect, three different types of feedback: the first one explores biomarkers of smoking exposure (cotinine, carbon monoxide (CO)); the second one gives information on smoking-related disease risk (e.g. lung cancer susceptibility according to CYP2D6 genotyping) (Audrain 1997); and the third one depicts smoking-related harm (e.g. atherosclerotic plaque, impaired lung function) (Buist 2002). The rationale for such interventions is to provide personalized motivational feedback to promote risk awareness and to accelerate smoking behaviour change (Curry 1993;

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Miller 1991). Recognition of personal susceptibility to the adverse effects of smoking may be an important step in the pathway to smoking cessation (McClure 2001; Weinberger 1981).

Individual studies have, however, produced conflicting data on the effect of physiological feedback (Petty 1976; Loss 1979; Hepper 1980; Weinberger 1981;Stitzer 1982; Bauman 1983; Li 1984; Lerman 1993; McBride 2000). This review set out to examine systematically data on smoking cessation rates from controlled trials using feedback on the physiological effect of smoking or on the genetic susceptibility to smoking-related diseases.

OBJECTIVES

The main objective was to determine the efficacy of providing smokers with feedback on their exhaled carbon monoxide (CO) measurement, spirometry results, and genetic susceptibility to smoking-related diseases in helping them to quit.

The hypotheses to be examined were:

- Feedback on personal characteristics indicating effects of smoking, or susceptibility to smoking-related illness, increases rates of smoking cessation.

- Multiple types of measurement (e.g. spirometry and exhaled CO measurement used together) are more effective for smoking cessation than single forms of measurement.

METHODS

Criteria for considering studies for this review

Types of studies

Randomized controlled trials. We examined observational studies in the background section of the review, but we did not use them to draw conclusions about effectiveness.

Types of participants

Individuals who smoked and who participated in smoking cessation programmes, or in screening for respiratory disease, or in health check-ups. There were no restriction on whether participants were hospitalised or not, or suffering from co-existent illness.

Types of interventions

Any intervention in which a physical measurement, such as exhaled carbon monoxide (CO) measurement, spirometry or genetic testing, was used as a way to increase motivation to quit. We considered studies in which reporting these measurements was the only component of an intervention, or was tested as an adjunct to another intervention such as counselling, where the control group received all the components except for the reporting of such measurements. We excluded trials in which the effect of biological measurements was confounded by the use of other components in the active intervention.

Types of outcome measures

The main outcome measure was abstinence from smoking, measured at least six months after the start of the intervention. We used the most conservative measure of quitting at the longest follow up. We counted participants lost to follow up as continuing smokers. We excluded studies that measured intermediate outcomes such as withdrawal symptoms, but did not provide data on cessation.

Search methods for identification of studies

We searched the Cochrane Tobacco Addiction Group Specialized Register for studies that involved any use of a biomedical test as part of an intervention. This includes the results of searches of electronic databases including MEDLINE, EMBASE, PsycINFO and Science Citation Index and abstracts from the Society for Research on Nicotine and Tobacco (SRNT) and World Tobacco or Health conferences. We conducted additional searches of the Central Register of Controlled Trials (CENTRAL), MEDLINE (1966 to January 2009) and EMBASE (1980 to January 2009) for any of the following topic-related keywords in titles, abstracts, or indexing fields: patient education, patient compliance, counselling, persuasive communication, spirometry, respiratory function, bronchospirometry, carbon monoxide, forced expiratory flow rates, obstructive lung diseases, genetic testing, genetic susceptibility, biomarker, feedback. These topic-related terms were combined with the smoking and study design related terms used for the regular tobacco addiction group register searches.

Data collection and analysis

Two reviewers independently pre-screened all search results (abstracts) for possible inclusion or as useful background. They selected studies for full-text assessment if retained by at least one of the reviewers. Two reviewers then independently assessed the selected articles for inclusion, resolving discrepancies by consensus. We recorded reasons for the non-inclusion of studies in the Table of Excluded Studies.

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Each reviewer then extracted and compared the data from the selected studies. This stage included an evaluation of quality. Two independent reviewers assessed each study according to the presence and quality of the randomization process, whether or not trialists and assessors were 'blinded', whether the analysis was appropriate to the study design, and the description of withdrawals and dropouts.
We extracted data, where available, on:

- Country and setting (e.g. primary care, community, hospital outpatient/inpatient).
- Recruitment.
- Method of selection of participants (e.g. willingness to make a quit attempt).
- Definition of smoker used.
- Method of randomization.
- Allocation concealment.
- Smoking and demographic characteristics of participants (e.g. average age, sex, average number of cigarettes smoked a day).
- Description of the experimental and control interventions (provider, length, number of visits, etc).
- Outcomes, including definition of abstinence used, and biochemical validation of cessation.
- Proportion of participants with follow-up data.

Following changes to the Cochrane Tobacco Addiction Group's recommended method of data analysis since this review was prepared, we have changed the way in which we summarize the effects of treatment. We now use the risk ratio rather than the odds ratio for summarizing individual trial outcomes and for estimates of pooled effect. Treatment effects will seem smaller when expressed as risk ratios than when expressed as odds ratios, unless the event rates are very low. For example, if 20 out of 100 participants have quit in the intervention group, and 10 out of 100 in the control group, the risk ratio is 2.0 [(20/100)/(10/100)], whilst the odds ratio is 2.25 [(20/80)/(10/90)]. Whilst there are circumstances in which odds ratios may be preferable, there is a danger that they will be interpreted as if they are risk ratios, making the treatment effect seem larger (Deeks 2008). We estimated a pooled weighted average of risk ratios using a Mantel-Haenszel method, with 95% confidence intervals.

RESULTS

Description of studies

See: Characteristics of included studies; Characteristics of excluded studies.
We identified eleven trials that met the inclusion criteria. One of them (Audrain 1997) tested two interventions. Out of the 12 interventions, three tested the effect of exhaled carbon monoxide (CO) measurements (Jamrozik 1984; Sanders 1989; Audrain 1997), three tested the combination of exhaled CO measurement and spirometry (Walker 1985; Risser 1990; Sippel 1999), three tested the effect of spirometry alone (Segnan 1991; Buffels 2006; Parkes 2008), one tested the effect of undergoing an ultrasonography of carotid and femoral arteries with photographic demonstration of atherosclerotic plaques when present (Bovet 2002), and two tested feedback about genetic susceptibility to lung cancer (Audrain 1997; Ito 2006). In Audrain 1997, the intervention was feedback about genetic susceptibility combined with CO measurement, which could either be compared to a control group of CO measurement alone, or to a control group without biomarker feedback, thereby testing the combination of the two interventions.
The trials were conducted in different settings: eight trials took place in general practice (Jamrozik 1984; Sanders 1989; Segnan 1991; Buffels 2006; Parkes 2008) or outpatient clinics (Sippel 1999; Bovet 2002; Ito 2006), two in a 'smoking clinic' (Walker 1985; Audrain 1997), and one in a health promotion clinic for army veterans (Risser 1990). Four trials took place in the United States (Walker 1985; Risser 1990; Sippel 1999; Audrain 1997), three in the United Kingdom (Jamrozik 1984; Sanders 1989; Parkes 2008), one in Italy (Segnan 1991), one in Belgium (Buffels 2006), one in the Seychelles Islands (Bovet 2002) and one in Japan (Ito 2006).
The mean age of the participants, when given, varied between 35 and 53 years. The proportion of females in the trials varied between 4% and 63%. The mean number of cigarettes smoked per day varied between 11.9 and 29.2. The mean number of cigarettes smoked per day was highest in the trials set in a 'smoking clinic' (35.5 per day in Walker 1985, 22.7 per day in Audrain 1997) or among veterans (Risser 1990). Levels of nicotine addiction as assessed by the Fagerström score (Heatherton 1991) and proportions of patients in the various stages of change according to Prochaska and Di Clemente (Prochaska 1983) were only given in some trials and could not be used to compare the study populations of the different trials.
The therapist delivering the intervention was a physician in four trials (Bovet 2002; Jamrozik 1984; Segnan 1991; Buffels 2006), a nurse in two trials (Risser 1990; Sanders 1989), or a specific study staff member in five trials (Audrain 1997; Sippel 1999; Walker 1985; Ito 2006; Parkes 2008).
We list 26 studies as excluded Characteristics of excluded studies; these included studies where the effect of the biomedical assessment could not be evaluated separately from the rest of the cessation intervention; that had insufficient length of follow-up, or were not true randomized trials.

Risk of bias in included studies

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Only two of the eleven trials reported an adequate procedure for both randomization and allocation concealment (Segnan 1991; Parkes 2008). Five studies did not report the method of randomization, or did not also give enough information to assume that allocation was adequately concealed (Walker 1985; Risser 1990; Audrain 1997; Bovet 2002; Buffels 2006). Three reported inadequate concealment of allocation, with allocation by day or week of attendance (Jamrozik 1984; Sanders 1989), or by odd/even numbered questionnaire at the time of check-in (Sippel 1999). In one of these studies, 120 patients were allocated to the wrong group and were excluded from further analysis (Sanders 1989).

Due to the demonstrative nature of the intervention, participants and interveners could not be blinded to allocation. Only four studies explicitly mentioned that assessors were blinded to allocation at the time of outcome determination (Risser 1990; Sippel 1999; Bovet 2002; Parkes 2008).

Parkes 2008 was powered to detect a 10% difference (eg 5% vs 15%) with 80% power, but did not recruit the intended sample of 600. Ito 2006 was powered to detect a 7.5% difference. One sample size estimate (Sippel 1999), was based on a rather optimistic assumption about the effect of the intervention (a quit rate of 25% in the intervention group versus 10% in the control group).

Urinary cotinine level was used to validate smoking cessation at follow up in three trials (Sanders 1989; Segnan 1991; Parkes 2008). One study used the same validation procedure but only on a subsample (41%) of self-reported ex-smokers and the analysis is based on self-report (Jamrozik 1984). Two studies used expired air carbon monoxide (Risser 1990; Walker 1985). Five studies did not use any biochemical validation (Audrain 1997; Sippel 1999; Bovet 2002; Buffels 2006; Ito 2006).

Methods of recruitment were heterogeneous between studies. Among the five studies conducted in general practice, one recruited patients at their first visit (Jamrozik 1984), another screened outpatients on specific days (Segnan 1991) two screened patients during the recruitment period (Sanders 1989; Buffels 2006) and one invited known recent smokers by letter (Parkes 2008). One study recruited smokers among outpatients in primary care clinics (Sippel 1999) and one outpatients at a cancer centre hospital (Ito 2006). The two studies conducted in 'smoking cessation clinics' recruited smokers by media advertisement (Audrain 1997; Walker 1985). The remaining studies recruited the last consecutive 155 smokers who participated in a health survey (Bovet 2002), and veterans that responded to mailed invitation to attend a health promotion clinic (Risser 1990). Participation rates (i.e. the proportion of those approached who agreed to take part in the trial) were seldom recorded. In two studies (Audrain 1997; Walker 1985), it was not possible to determine the initial allocation of the participants who were subsequently lost to follow up, and the analysis had to be performed per protocol.

All studies included male and female adults who were smokers at the time of inclusion. Only three studies gave a definition of being a smoker at the time of inclusion (Audrain 1997; Bovet 2002; Ito 2006).

Effects of interventions

Results are given as risk ratios (RRs) for smoking cessation up to the longest recorded follow-up time (six to twelve months) between intervention and control groups, with their 95% confidence intervals. A RR greater than 1 favours the intervention group. The trials have been classified by type of intervention. Results of the analyses are reproduced in Figure 1.

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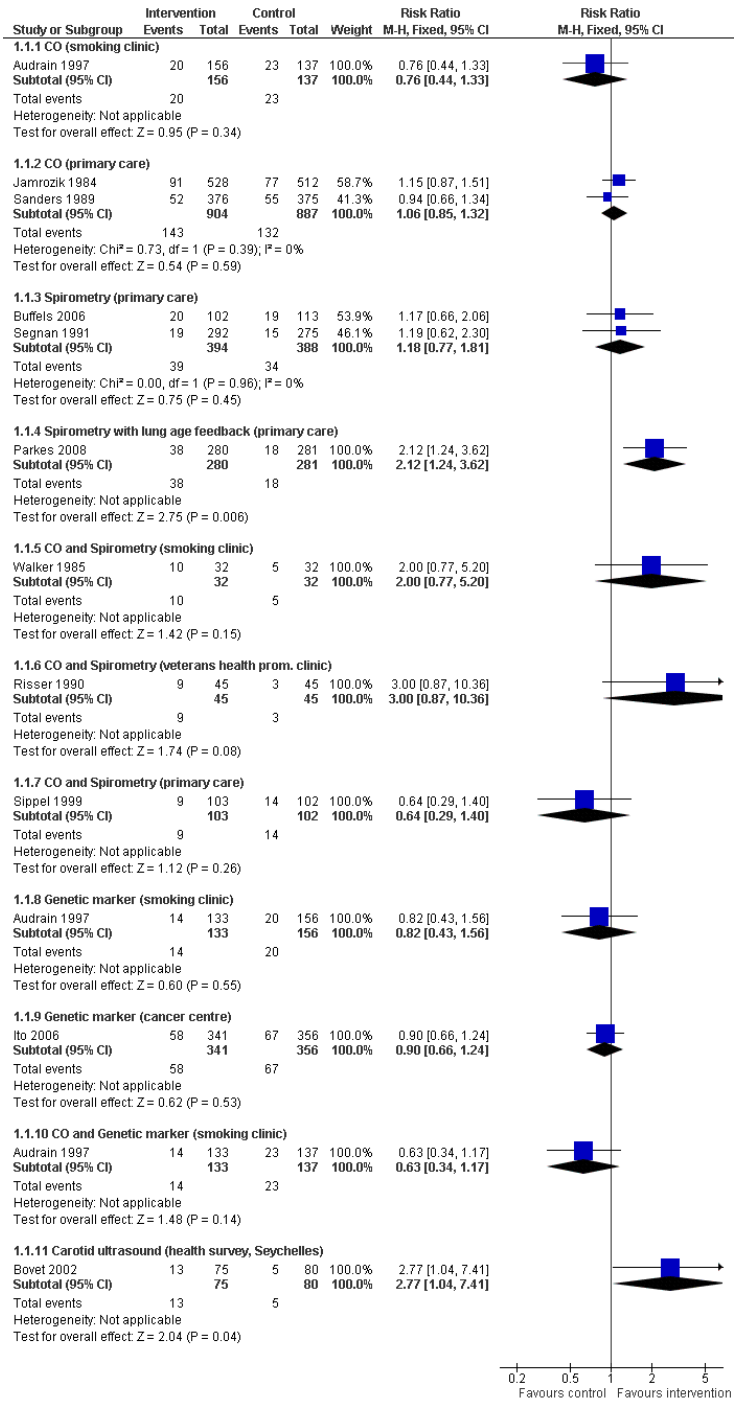
Figure 1. Forest plot of risk ratios for smoking cessation for all intervention comparisons



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Exhaled CO measurement

Three studies isolated the effect of demonstrating levels of exhaled carbon monoxide (CO) on smoking cessation rate (Jamrozik 1984; Sanders 1989; Audrain 1997) with RRs of 1.15 (95% confidence interval (CI) 0.87 to 1.51), 0.94 (95% CI 0.66 to 1.34) and 0.76 (95% CI 0.44 to 1.33), respectively. We pooled only the two studies that took place in a similar primary care setting (Jamrozik 1984; Sanders 1989). There was no evidence of a significant benefit from these pooled studies (Mantel-Haenszel fixed-effect RR 1.06, 95% CI 0.85 to 1.32).

Spirometry

Spirometry results were used in three primary care-based trials. Two had similar small and non significant relative effects (Segnan 1991; Buffels 2006). There was still no evidence of a significant benefit from this type of intervention when the two studies were pooled (RR 1.18; 95% CI 0.77 to 1.81,). The third study (Parkes 2008) did detect a significant effect of giving immediate feedback and explanation of spirometry results in the form of 'lung age' and comparing it to the smoker's chronological age (RR 2.12; 95% CI 1.24 to 3.62). In this study the control group also had spirometry and feedback but the results were given by letter in terms of standard measures of lung function.

Exhaled CO measurement and Spirometry

Exhaled CO measurement and spirometry were used together in three trials (Walker 1985; Risser 1990; Sippel 1999), with RRs of 2.00 (95% CI 0.77 to 5.20), 3.00 (95% CI 0.87 to 10.36) and 0.64 (95% CI 0.29 to 1.40) respectively. We did not pool these studies because of heterogeneous settings that would preclude the drawing of clinically relevant conclusions.

Genetic susceptibility to smoking-related cancer

A test for genetic susceptibility to lung cancer was used in one trial (Audrain 1997) with a RR of 0.82 (95% CI 0.43 to 1.56). Genetic susceptibility to lung and esophageal cancer was used in another trial (Ito 2006) with a RR of 0.90 (95% CI 0.66 to 1.24).

Genetic susceptibility to lung cancer and exhaled CO measurement

Genetic susceptibility to lung cancer and exhaled CO measurement were used jointly in one trial (Audrain 1997) with a RR of 0.63 (95% CI 0.34 to 1.17) when compared to neither.

Ultrasonography of carotid and femoral arteries +/- photographs of atherosclerotic plaques

Ultrasonography of carotid and femoral arteries was used in one trial (Bovet 2002) with a RR of 2.77 (95% CI 1.04 to 7.41). This study was conducted among light smokers (average 10 to 12 cigarettes a day). Subjects were randomized to undergo ultrasonography (n = 74) or not (n = 79). Those with ultrasonographic demonstration of atherosclerotic plaques (n = 20) were shown photographs of them, whereas the others did not benefit from any further procedure.

DISCUSSION

Due to the scarcity of evidence of sufficient quality, we cannot make definitive statements about the effectiveness of most types of biomedical risk assessment as an aid for smoking cessation. Most studies were small and did not detect significant effects. Only two pairs of studies were similar enough in terms of recruitment, setting, and intervention to allow pooling of data for meta-analysis. There was still no evidence of a significant benefit either from exhaled CO measurement in primary care (Jamrozik 1984; Sanders 1989) or from spirometry in primary care (Segnan 1991; Buffels 2006) when pooling the pairs. Only two studies detected statistically significant intervention effects; neither of these could be pooled. One of these was a trial in primary care (Parkes 2008) that used spirometry and immediate feedback of the results using a graphical display. Intervention participants were told their 'lung age' if this was older than their chronological age. The control group also had spirometry and feedback, but the results were given by letter in terms of standard measures of lung function, with no mention of 'lung age'. All participants were advised to quit and offered a referral to intensive support. Contact time was longer in the intervention group because of the verbal feedback. Because of the way the feedback was given and because there was no non-spirometry control we did not pool it with the two other studies of spirometry in primary care, Giving the spirometry results in term of 'lung age' resulted in an increase in the cessation rate at 12 months from 6.4% to 13.6%. In considering the strength of evidence and generalisability of this trial it should also be noted that the outcome was point prevalence abstinence, and participants were volunteers (Bize 2008).

The other study with a statistically significant positive effect (Bovet 2002) used pictures of ultrasound photographs of atherosclerotic plaques. Its external validity can be questioned, as the sample was made up predominantly of male light smokers (average 10 to 12 cigarettes a day) in a developing country .

In most of the included studies, the biomedical testing component was added to intensive quit-smoking sessions, with counselling lasting up to 60 minutes and completed by written material and reinforcement sessions or follow-up phone 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 characterized as directly leading to a successful quit attempt. Data from those studies do not allow us to verify this hypothesis. However, given the increasing evidence that the transtheoretical model of behaviour change is not a mandatory prerequisite for a smoking cessation intervention to be effective (Riemsma 2003), we do not consider this lack of intermediate data as a major concern.

Biomedical risk assessment was also conceptualised as a part of multi-component interventions (with components other than biomedical risk assessment) by some authors identified by our search strategy (Richmond 1986; Humerfelt 1998; Borrelli 2002;

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Hajek 2002; McBride 2002). Even though two of these studies demonstrated effects significantly favouring intervention versus control groups, they did not isolate the specific effect of biomedical feedback. Nor do the retrieved studies provide us with sufficient data to examine our second research hypothesis, that feedback with different types of measurements is more effective for smoking cessation than feedback of a single measurement.

Demonstration of smokers' child exposure to environmental tobacco smoke by measuring the child's urinary cotinine level was used in another trial (Wakefield 2002) with an OR of 0.15 (95% confidence interval (CI) 0.01 to 2.89). We excluded this study from our analysis because our research hypothesis was that smokers, although adept at estimating the risks of smoking in terms of morbidity and mortality, are prone to underestimate their own risk with regard to smoking (Lerman 1993; Romer 2001). It therefore 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 this 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.

A promising new approach could be for dental practitioners to use a point of care test for salivary nicotine metabolites. A short-term randomised controlled trial (Barnfather 2005) compared smoking cessation after eight weeks for participants who received either counselling with immediate or delayed (at the end of the study) feedback on salivary nicotine level. The odds ratio for smoking cessation favoured those who received immediate feedback: (OR 4.04, 95% CI 1.10-14.53). Trials with longer term follow-up are needed to confirm this effect (Coleman 2005).

Despite broad inclusion criteria regarding the type of participants, we found only scarce data exploring the effect of biomedical risk assessment on hospitalised patients or acutely ill patients. It is possible that such a specific context and the presence of co-existent illnesses could facilitate a modification of risk perception. One study included both patients with and without cancer (Ito 2006). Reported subgroup analyses showed statistically non-significant ORs of 0.54 (95% CI 0.27 to 1.08) for patients with cancer, and of 1.23 (95% CI 0.59 to 2.61) for patients without cancer.

An earlier non-systematic review was conducted on the use of biomarkers in smoking cessation (McClure 2001). The aim of this work was to review the theoretical rationale and 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. The review therefore included non-randomized trials (Loss 1979; Kilburn 1990; Haddow 1991; Scott 1990), trials providing multicomponent interventions that precluded the isolation of the specific effect of biomarker feedback (Bauman 1983; Richmond 1986; Lerman 1997), trials comparing the effect of abnormal test results versus normal test results rather than test versus no test (Li 1984), and trials reporting outcomes other than smoking cessation. One study identified by McClure as 'in press' seems never to have been published (Hoffman 1998), and several attempts to contact the authors failed to provide us with more detailed information. Four studies mentioned by McClure were also retained in our review (Jamrozik 1984; Walker 1985; Risser 1990; Audrain 1997). We identified four more trials for our original review (Sanders 1989; Segnan 1991; Sippel 1999; Bovet 2002). When focusing on efficacy data, McClure 2001 concluded that biomarker feedback may enhance the likelihood of cessation because a trend for increased abstinence was found in three randomized trials (Walker 1985; Risser 1990; Hoffman 1998). The fact that two of these trials (Walker 1985; Risser 1990) are subject to major methodological limitations (small samples, inadequate randomization procedures) and that the report of Hoffman 1998 remains unpublished, calls for great caution in drawing such conclusions.

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. Five included studies provided some insight about smoking cessation rates in subgroups according to test results. Sippel 1999, Buffels 2006 and Parkes 2008 did not find any correlation between smoking cessation and abnormal spirometry results, whereas Bovet 2002 found a statistically non-significant lower smoking cessation rate among participants without plaques at ultrasonography than among participants without ultrasonograpy. Parkes 2008 noted that anecdotally some smokers were motivated by having normal results because it helped them feel it was 'not too late' to quit. Ito 2006 found some evidence that amongst participants without cancer, those told they had increased susceptibility to cancer were more likely to quit. This particular question, and the way to handle and communicate normal results, remain to be answered.

AUTHORS' CONCLUSIONS

Implications for practice

There is little evidence about the effects of biomedical risk assessment. Spirometry combined with an interpretation of the results in terms of 'lung age' may be considered based on a single trial but the evidence is not optimal. Mixed quality evidence does not support the hypothesis that other types of biomedical risk assessment increase smoking cessation in comparison to standard treatment.

Implications for research

There is room for improvement in the methodological quality of studies aimed at evaluating the efficacy of biomedical risk assessment as an aid to smoking cessation. The methodological areas

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that can be improved are an adequate sample size, the concealment of allocation following the randomization procedure, the use of a consensual definition of a smoker, the use of a consensual definition of abstinence, the systematic use of biochemical validation of smoking abstinence, and the systematic inclusion of all those lost to follow up in the denominator of each group (on an intention-to-treat basis).

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* *Indicates the major publication for the study*

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CHARACTERISTICS OF STUDIES

Characteristics of included studies *[ordered by study ID]*

Audrain 1997

Methods	Setting: 'smoking clinic', USA Recruitment: lay press Selected: advertisement: 'free smoking-cessation study'
Participants	550 smokers (defined as >=5 cpd for >=1 year) out of 1104 eligible; mean age 44yrs, 62.8% female, 83.9% white, Mean cpd: 22.7 SoC: Preparation stage: 37.5%, Mean Fagerström score: 5,4 Therapist: trained health educator.
Interventions	Intervention 1: EBF group: Exposure Biomarker (CO) Feedback + 10 min motivational counselling before QSC. Intervention 2: SBF group: Susceptibility Biomarker (CYP2D6) Feedback + 10 min motivational intervention + EBF + QSC. Control: QSC group: 60 min Quit-Smoking Consultation (quit plan, gaining support).
Outcomes	Definition of abstinence: 30-days abstinence Duration of follow up: 12 months. Biochemical validation of non-smokers: none
Notes	

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Unclear	Randomized between 2 interventions & control, method not described
Allocation concealment?	Unclear	No details given
Blinding? All outcomes	Unclear	No details given
Incomplete outcome data addressed? All outcomes	Yes	Per protocol analysis, 33% lost to F-UP. Distribution of baseline 550 participants among the 3 groups not reported, 'There were no significant differences between treatment groups with respect to loss to follow-up'.

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Bovet 2002

Methods	Setting: Seychelles Heart Study II 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.
Participants	155 smokers (defined as >=1 cpd during previous week); mean age 46 yrs, 15% female, Mean cpd: 11.9 Therapist: physician
Interventions	Intervention: Ultrasonography of carotid and femoral arteries. Smokers with >= 1 plaque given 2 photographs of their plaque + explanation. + quit-smoking counselling. Control: Quit-smoking counselling
Outcomes	Definition of abstinence: 7-days abstinence Duration of follow up: 6 months. Biochemical validation of non-smokers: none (assessor blinded)
Notes	

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Yes	Pre-established random sequences of numbers matched to rank of arrival
Allocation concealment?	Unclear	Unclear that allocation was fixed
Blinding? All outcomes	Yes	Follow-up assessors blind to allocation group
Incomplete outcome data addressed? All outcomes	Yes	Two participants lost to follow up, reincluded as smokers for analysis

Buffels 2006

Methods	Setting: 14 general practices, Belgium Recruitment: screening of all attending patients above 15 years of age Selected: smokers in the motivation or action stage
Participants	215 randomized out of 1206 screened smokers. 182 analysed. Smoker definition not given. Mean age: 47.0, 47.3% female, mean pack-years: 24.6 Therapist: physicians
Interventions	Intervention: Spirometry + control intervention Control: Minimal intervention based on the 5A model: ask, advise, assess, assist, arrange + Quit date + NRT or bupropion + F-UP contact

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Buffels 2006 (*Continued*)

Outcomes	Definition of abstinence: continuous abstinence from quit date Duration of follow up: 12 months (24 month data not used in analysis because of increasing loss to follow-up) Biochemical validation of non-smokers: none at the 12 months F-UP. Very partial at 24m
Notes	New for 2009 update. Additional data provided by author

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Yes	Randomization by coin toss
Allocation concealment?	Unclear	No statement that random sequence generated by coin toss was concealed at time of enrollment
Blinding? All outcomes	Unclear	Unclear
Incomplete outcome data addressed? All outcomes	Yes	13 (13%) I, 20 (18%) C lost before 6m, all losses included as smokers.

Ito 2006

Methods	Setting: First-visit at Aichi Cancer Center Hospital (ACCH), Japan Recruitment: screening of first-visit outpatients (whether consecutive or not is unknown)
Participants	697 included out of 859 eligible smokers. Smoker defined as smoking at least one cigarette on the previous day. Mean age 46.5, 40.5% female, mean cpd: 22.2, pre-contemplator/ contemplator: 70% Therapist: trained interviewer
Interventions	Intervention: Information at baseline on the effect of L-myc polymorphism on modulating the risk of cancer due to smoking (5-10 min). Sent genotype report at 3m, with same information about effect of polymorphism (65% got genotype information) Control: just followed-up for the smoking status
Outcomes	Definition of abstinence: point prevalence at 9 months (continuous abstinence, not smoking at both the 3- and the 9-month f-ups also reported but genotype only provided after 3m f-up Duration of follow-up: 9 months Biochemical validation of non-smokers: none (Attempt made but none agreed to return for CO measurement)
Notes	New for 2009 update. Not given genotype until 3m. Most participants in both groups were already quit at 3m, Small proportion of new quitters after 3m

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Ito 2006 (*Continued*)

Risk of bias		
Item	**Authors' judgement**	**Description**
Adequate sequence generation?	No	Pseudo-random allocation by week of attendance
Allocation concealment?	No	Allocation known at time of enrollment so potential for selection bias
Blinding? All outcomes	Unclear	Outcomes assessed by postal questionnaire
Incomplete outcome data addressed? All outcomes	Yes	Complete data for 52.9% (369), did not differ significantly between groups

Jamrozik 1984

Methods	Setting: 6 general practices, United Kingdom Recruitment: clinic, first visit Selected: outpatients
Participants	2110 smoker (defined as a person admitting to smoking cigarettes) out of 6052 screened. 1040 in relevant arms 61% female, No detailed patient characteristics given. Significant difference of social classes between groups. Therapist: physician
Interventions	Intervention: demonstration of patient exhaled CO + verbal advice + booklet. Control: verbal advice + booklet.
Outcomes	Definition of abstinence: point prevalence Duration of follow up: 12 months. Biochemical validation of non-smokers: urinary cotinine in a sample (41%) of self-reported non-smokers.
Notes	Outcome based on unvalidated data. Between 24 & 40% may have misrepresented smoking status but no evidence of differential misreporting between groups

Risk of bias		
Item	**Authors' judgement**	**Description**
Adequate sequence generation?	No	Randomized according to day of attendance, balanced over 4 weeks.

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Jamrozik 1984 (*Continued*)

Allocation concealment?	No	Allocation known at enrollment, possibility of selection bias
Blinding? All outcomes	Unclear	No details given
Incomplete outcome data addressed? All outcomes	Yes	28% lost to f-up across all 4 arms, treated as smokers. No information on differential loss

Parkes 2008

Methods	Setting: Primary care (5 general practices, UK) Recruitment: By letter to registered patients Selection: not selected by motivation
Participants	561 current smokers (not defined) aged >35, 54% female, average age 53, av. cpd 17, 29% pre-contemplative, 32% contemplative, 17% preparation, 21% action Therapists: Spirometry
Interventions	All participants had lung function assessed by spirometry before randomisation.Strongly encouraged to give up and given written contact details of NHS smoking cessation services. If evidence of asthma or restrictive lung disease advised to see GP. Told they would be retested at 12m. 1. Immediate verbal feedback of lung age with explanation. Described as normal if lung age less than or equal to chronological age. 2. Letter giving test results without lung age
Outcomes	Definition of abstinence: unspecified Duration of follow up: 12 months. Biochemical validation of non-smokers: exhaled CO, cotinine <14.2ng/ml
Notes	New for 2009 update

Risk of bias

Item	**Authors' judgement**	**Description**
Adequate sequence generation?	Yes	"A clerk (who then took no further part in the study) prepared 600 sequentially numbered opaque sealed envelopes, each containing a card with allocation group determined by computer generated random number (odd = intervention)"
Allocation concealment?	Yes	"If the participant met the inclusion criteria and gave consent, he or she was entered into the study and underwent base-

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Parkes 2008 (*Continued*)

		line spirometry. The next numbered envelope in the series was then opened to determine allocation group"
Blinding? All outcomes	Yes	Follow-up assessors blind to allocation group
Incomplete outcome data addressed? All outcomes	Yes	11% lost to f-up in each arm, included as smokers

Risser 1990

Methods	Setting: US Veterans Administration Demonstration Project Recruitment: veterans attending a health promotion clinic. Selected: responding to mailed invitations for health promotion. Some second visit
Participants	90 smokers (not defined); mean age=53.7 years (55.5 vs 51.7), 4% female, Mean cpd: 23.5, Mean pack-year: 60.4 Initial cessation intent 51% vs 44%. Therapist: nurse-practitioner
Interventions	Intervention: spirometry, exhaled CO, discussion of pulmonary symptoms + control intervention. Control: 50 min educational intervention, review of self-help manual, invitation to a nine-session one-to-one counselling programme.
Outcomes	Definition of abstinence: point prevalence Duration of follow up: 12 months. Biochemical validation of non-smokers: exhaled CO<=10 ppm.
Notes	

Risk of bias

Item	**Authors' judgement**	**Description**
Adequate sequence generation?	Unclear	Randomized, method not stated
Allocation concealment?	Unclear	No details given
Blinding? All outcomes	Yes	Follow-up assessors blind to allocation group
Incomplete outcome data addressed? All outcomes	Yes	13 (29%) I, 6 (13%) C lost to f-up at 12m, included as smokers.

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Sanders 1989

Methods	Setting: 11 United Kingdom general practices Recruitment: screening of all outpatients. Selected: outpatients + made appointment for health check.
Participants	751 participants out of 4330 identified smokers (self-defined) Mean age 38.5 years. Other characteristics not mentionned. Therapist: practice nurse
Interventions	Intervention: exhaled CO measure + discussion of significance + control intervention Control: Counselling by practice nurse + written material given + offer of a follow-up appointment.
Outcomes	Definition of abstinence: point prevalence. Duration of follow up: 12 months. Biochemical validation of non-smokers: urinary cotinine for sample, cut-off not reported. Outcomes used are self-report
Notes	

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Unclear	Randomized by day of attendance on a 1:2 basis. Desktop card reminding doctors of right allocation. 120 wrongly allocated patients, excluded from further analysis. Second step randomized health check attenders for the CO intervention, method not described
Allocation concealment?	No	First stage randomization had potential for selection bias; only attenders eligible for second stage, no information on concealment at this stage.
Blinding? All outcomes	Unclear	No details given
Incomplete outcome data addressed? All outcomes	Yes	Non responders included as smokers

Segnan 1991

Methods	Setting: 44 general practices, Italy. Recruitment: screening of outpatients on specific days. Selected: outpatients.

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Segnan 1991 (*Continued*)

Participants	923 included out of 1009 screened. smoker definition not given. Age: 20.1% <31yrs; 28.0% 31-40yrs; 26.8% 41-50yrs; 25.0% >50yrs. 38% female, cpd: 16.7% <=10 cpd; 55.2% 11-20 cpd; 28.1% >20 cpd. 51% reporting symptoms. Therapist: physician
Interventions	Intervention: Spirometry prescription + control intervention. Control: Repeated counselling with reinforcement sessions. (2 other groups not used in our comparison: minimal intervention and repeated counselling + nicotine gum).
Outcomes	Definition of abstinence: 7 days abstinence Duration of follow up: 12 months. Biochemical validation of non-smokers: urinary cotinine < 100 ng/mg.
Notes	In the intervention group, 124 participants out of 292 reported having a spirometry test.

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Yes	Randomized; 'blocked treatment allocation was based on a sequence of random numbers'
Allocation concealment?	Yes	'Closed, numbered envelopes ...'. 'The envelopes were indistinguishable from the outside ...'. 'The research staff checked physicians' com-pliance with the procedure for assignment by comparing envelope numbers and dates of recruitment.'
Blinding? All outcomes	Unclear	No details given
Incomplete outcome data addressed? All outcomes	Yes	13% lost to f-up at 12 months, included as smokers

Sippel 1999

Methods	Setting: 2 primary care clinics, USA Recruitment: all smokers among outpatients. Selected: outpatients
Participants	205 included out of 360 smokers (self-defined); mean age 38.5 yrs, 62.5% female, mean cpd: 20.0, mean pack-years: 28.9. SoC: 36% in preparation stage. Therapist: study staff

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Sippel 1999 (*Continued*)

Interventions	Intervention: Spirometry and exhaled CO + control intervention Control: counselling according to transtheoretical model stage + written material + NRT encouraged if prepared to stop.
Outcomes	Definition of abstinence: sustained quitting rate Duration of follow up: 9 months Biochemical validation of non-smokers: none
Notes	

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	No	Pseudo-randomized; questionnaires numbered consecutively and subjects enrolled in chronologic order based on time of check-in). Odd-numbered=intervention
Allocation concealment?	No	Not concealed, potential selection bias, although nurses performing patient check-in were blinded to the questionnaire numbers and 'As 4 to 6 nurses conducted patient check-in independently and simultaneously at each clinic, it is unlikely that any given patient would be preferentially enrolled into either study arm'.
Blinding? All outcomes	Yes	Follow-up assessors blind to allocation group
Incomplete outcome data addressed? All outcomes	Yes	18.6% I and 12.6% C lost to f-up, included as smokers

Walker 1985

Methods	Setting: 'stop-smoking clinic', USA Recruitment: public service anouncement + media advertising Selected: those responding to advertising, paying US$45
Participants	64 out of 141 eligible. (smoker self-defined) Mean age: 35.5yrs, 59% female, Mean cpd: 29.2, mean 3.4 previous quit attempts Therapist: first author
Interventions	Intervention: exhaled CO and spirometry feedback + Taste Satiation (TS) (in 50%) or Focused smoking (FS) (in 50%) and booster sessions for half of each subgroup. Control: 50% TS sessions or 50% FS sessions, booster sessions for half of each subgroup.

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Walker 1985 (*Continued*)

Outcomes	Definition of abstinence: 10 days abstinence Duration of follow up: 6 months. Biochemical validation of non-smokers: exhaled CO <8ppm
Notes	

Risk of bias

Item	Authors' judgement	Description
Adequate sequence generation?	Unclear	Randomized in 2x2x2 factorial design, method not described
Allocation concealment?	Unclear	No details given
Blinding? All outcomes	Unclear	No details given
Incomplete outcome data addressed? All outcomes	No	Only participants reached at 6m included in analysis. 'Five subjects dropped out of treatment and 3 additional subjects were randomly eliminated to facilitate data analysis'.

CO: Carbon Monoxide
cpd: cigarettes per day
NRT: Nicotine Replacement Therapy
OPD: Outpatient Department
ppm: parts per million
SoC: stage of change

Characteristics of excluded studies *[ordered by study ID]*

Anthonisen 1994	Effect of spirometry cannot be isolated. Spirometry performed in all patients. Randomization to 3 groups: smoking intervention (12-session programme) + bronchodilatator, smoking intervention + placebo, no intervention.
Barnfather 2005	Only 8 weeks follow-up. Intervention was immediate feedback from a point of care test for salivary nicotine metabolites
Borrelli 2002	Effect of CO cannot be isolated. No results available. Intervention: Precaution Adoption Model: CO feedback + environmental tobacco smoke to the child feedback+ motivational counselling. Control: Behavioral Action Model: self-efficacy enhancing counselling

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(*Continued*)

Borrelli 2005	Effect of CO cannot be isolated. Intervention: Motivational interviewing and CO feedback Control: Standard care
Cope 2003	Not a true randomized trial
Cox 2003	No control group. All participants received chest computed tomography scan screening for lung carcinoma.
Frank 1999	Full text not available. Intervention: urinary cotinine feedback to prenatal patients.
Hajek 2001	Effect of CO cannot be isolated. Intervention: counselling, written materials, CO Control: standard anti-smoking leaflets
Hajek 2002	Effect of CO cannot isolated. Intervention: CO feedback, booklet, quiz, 'buddy', declaration of commitment to give up, sticker in patient's notes. Control: verbal advice + different booklet
Humerfelt 1998	Effect of spirometry cannot be isolated. Intervention: spirometry + written counselling + pamphlet Control: spirometry
Kanner 1996	Effect of spirometry cannot be isolated. Spirometry performed in all patients. Randomization to 3 groups: smoking intervention (12-session program) + bronchodilatator, smoking intervention + placebo, no intervention.
Kanner 1999	Effect of spirometry cannot be isolated. Spirometry performed in all patients. Randomization to 3 groups: smoking intervention (12-session program) + bronchodilatator, smoking intervention + placebo, no intervention.
Lipkus 2007	Outcomes were perceived smoking-related health risks, worries and desire to quit, not smoking cessation
McBride 2002	Effect of genetic biomarker feedback cannot be isolated. Intervention: feedback on GSTM1 + 4 phone calls + control intervention. Control: self-help manual +/- NRT. No phone calls.
McIntosh 1994	Smoking cessation is not considered as an outcome.
Nuesslein 2006	Only 6 weeks follow-up. Intervention: Notification of urine cotinine levels + control Control: Written advice to quit from a paediatrician
Richmond 1986	Effect of biomarker feedback cannot be isolated. Spirometry, blood CO, urinary cotinine in both groups. Intervention: four visits and discussion of manual..

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Sanderson 2005	Short term study of effect of genetic testing for lung cancer susceptibility. Follow-up at 10 weeks. Main outcomes motivation to quit, perceived risk, depression and anxiety.
Shahab 2007	Pilot study of visual personalized biomarker feedback with follow up at 4 weeks. Main outcomes perception of susceptibility, engagement in cessation behaviours and intentions to stop.
Shoptaw 2002	Effect of CO feedback cannot be isolated. CO feedback given to all groups. 2x2 design: 1. relapse prevention counselling + NRT 2. contingency management: vouchers given for validated abstinence + NRT Control: NRT
Stratelis 2006	Not a true randomized trial, no control without biomedical risk assessment. Smokers with normal lung function at baseline received different frequencies of spirometry (annually or in three years).
Taylor 2007	No control group. Study of impact of screening on smoking cessation and readiness to stop smoking amongst participants in trials of lung screening
Terazawa 2001	Effect of biomarker feedback (CO and urinary cotinine) cannot be isolated, combined with 1 counselling session and 4 follow-up calls.
Townsend 2005	No control group. Longitudinal study of smoking behaviour in people receiving lung cancer screening.
Wakefield 2002	Biomarker feedback given on the subject's child health, not on his own health. The motivational component here differs from the approach in the other included studies.
Wilt 2007	Review

CO: carbone monoxyde
NRT: Nicotine Replacement Therapy

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DATA AND ANALYSES

Comparison 1. All interventions

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Smoking cessation	11		Risk Ratio (M-H, Fixed, 95% CI)	Subtotals only
1.1 CO (smoking clinic)	1	293	Risk Ratio (M-H, Fixed, 95% CI)	0.76 [0.44, 1.33]
1.2 CO (primary care)	2	1791	Risk Ratio (M-H, Fixed, 95% CI)	1.06 [0.85, 1.32]
1.3 Spirometry (primary care)	2	782	Risk Ratio (M-H, Fixed, 95% CI)	1.18 [0.77, 1.81]
1.4 Spirometry with lung age feedback (primary care)	1	561	Risk Ratio (M-H, Fixed, 95% CI)	2.12 [1.24, 3.62]
1.5 CO and Spirometry (smoking clinic)	1	64	Risk Ratio (M-H, Fixed, 95% CI)	2.0 [0.77, 5.20]
1.6 CO and Spirometry (veterans health prom. clinic)	1	90	Risk Ratio (M-H, Fixed, 95% CI)	3.0 [0.87, 10.36]
1.7 CO and Spirometry (primary care)	1	205	Risk Ratio (M-H, Fixed, 95% CI)	0.64 [0.29, 1.40]
1.8 Genetic marker (smoking clinic)	1	289	Risk Ratio (M-H, Fixed, 95% CI)	0.82 [0.43, 1.56]
1.9 Genetic marker (cancer centre)	1	697	Risk Ratio (M-H, Fixed, 95% CI)	0.90 [0.66, 1.24]
1.10 CO and Genetic marker (smoking clinic)	1	270	Risk Ratio (M-H, Fixed, 95% CI)	0.63 [0.34, 1.17]
1.11 Carotid ultrasound (health survey, Seychelles)	1	155	Risk Ratio (M-H, Fixed, 95% CI)	2.77 [1.04, 7.41]

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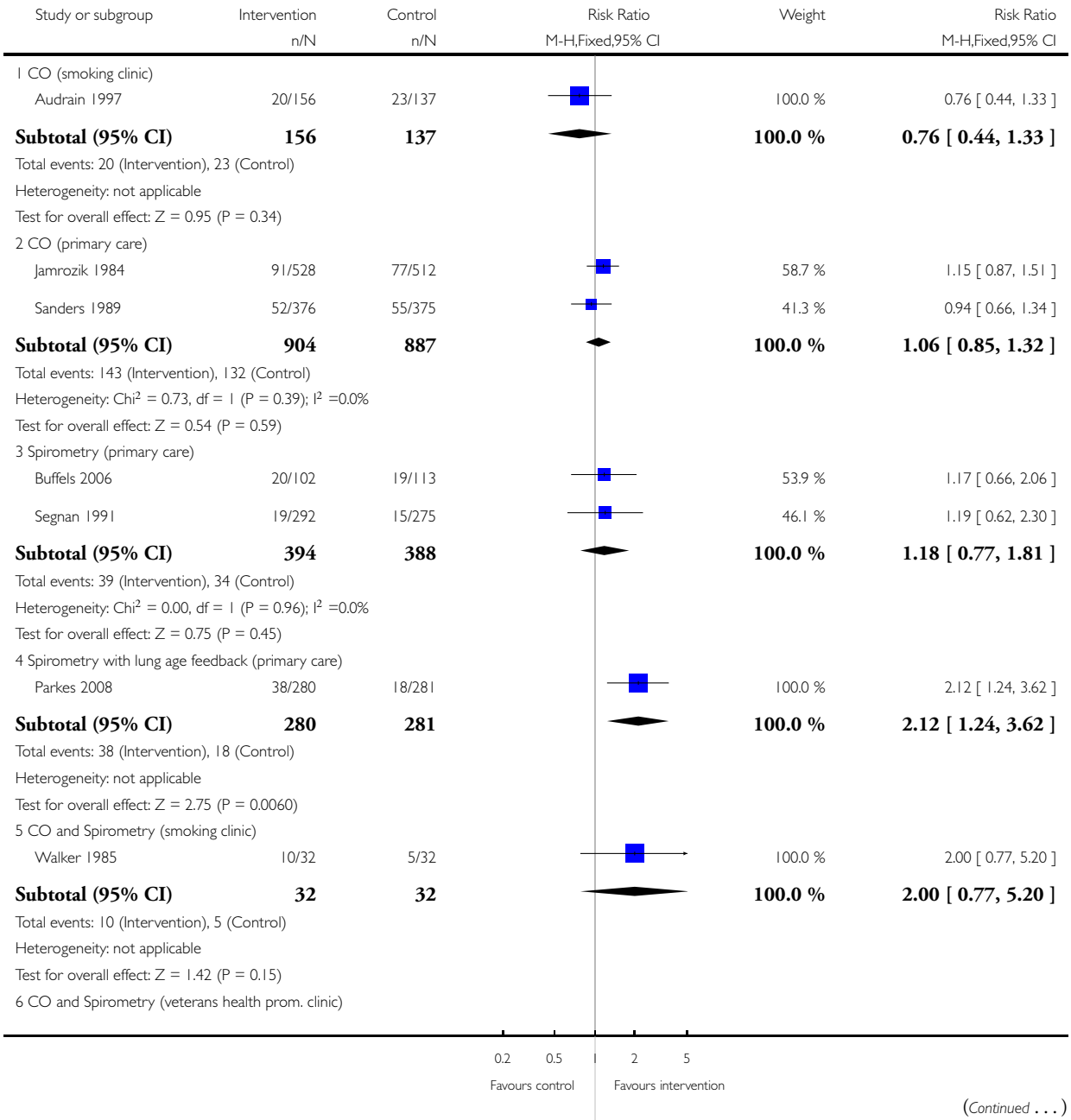
Analysis 1.1. Comparison 1 All interventions , Outcome 1 Smoking cessation.

Review: Biomedical risk assessment as an aid for smoking cessation

Comparison: 1 All interventions

Outcome: 1 Smoking cessation



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Study or subgroup	Intervention n/N	Control n/N	Risk Ratio M-H,Fixed,95% CI	Weight	Risk Ratio M-H,Fixed,95% CI
Risser 1990	9/45	3/45		100.0 %	3.00 [0.87, 10.36]
Subtotal (95% CI)	**45**	**45**		**100.0 %**	**3.00 [0.87, 10.36]**
Total events: 9 (Intervention), 3 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 1.74 (P = 0.082)					
7 CO and Spirometry (primary care)					
Sippel 1999	9/103	14/102		100.0 %	0.64 [0.29, 1.40]
Subtotal (95% CI)	**103**	**102**		**100.0 %**	**0.64 [0.29, 1.40]**
Total events: 9 (Intervention), 14 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 1.12 (P = 0.26)					
8 Genetic marker (smoking clinic)					
Audrain 1997	14/133	20/156		100.0 %	0.82 [0.43, 1.56]
Subtotal (95% CI)	**133**	**156**		**100.0 %**	**0.82 [0.43, 1.56]**
Total events: 14 (Intervention), 20 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 0.60 (P = 0.55)					
9 Genetic marker (cancer centre)					
Ito 2006	58/341	67/356		100.0 %	0.90 [0.66, 1.24]
Subtotal (95% CI)	**341**	**356**		**100.0 %**	**0.90 [0.66, 1.24]**
Total events: 58 (Intervention), 67 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 0.62 (P = 0.53)					
10 CO and Genetic marker (smoking clinic)					
Audrain 1997	14/133	23/137		100.0 %	0.63 [0.34, 1.17]
Subtotal (95% CI)	**133**	**137**		**100.0 %**	**0.63 [0.34, 1.17]**
Total events: 14 (Intervention), 23 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 1.48 (P = 0.14)					
11 Carotid ultrasound (health survey, Seychelles)					
Bovet 2002	13/75	5/80		100.0 %	2.77 [1.04, 7.41]
Subtotal (95% CI)	**75**	**80**		**100.0 %**	**2.77 [1.04, 7.41]**
Total events: 13 (Intervention), 5 (Control)					
Heterogeneity: not applicable					
Test for overall effect: Z = 2.04 (P = 0.042)					

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WHAT'S NEW

Last assessed as up-to-date: 26 January 2009.

28 January 2009	New citation required and conclusions have changed	One new study shows a significant effect.
27 January 2009	New search has been performed	Three new studies identified in update for Issue 2, 2009

HISTORY

Protocol first published: Issue 2, 2004

Review first published: Issue 4, 2005

21 April 2008	Amended	Converted to new review format.
11 August 2005	New citation required and conclusions have changed	Substantive amendment

CONTRIBUTIONS OF AUTHORS

RB : coordinating the review, conception and design of the review, data collection, developing search strategy and undertaking searches, screening search results, organising retrieval of papers, screening papers for inclusion, quality appraisal of papers, data abstraction, data management, data entry into RevMan, data analysis and interpretation, writing the review, writing to authors of papers for additional information, providing a clinical perspective.

YM: screening search results, organising retrieval of papers, screening papers for inclusion, quality appraisal of papers, data abstraction, data management, data entry into RevMan, data analysis and interpretation, writing the review, writing to authors of papers for additional information.

JC: conception and design of the review, developing search strategy, screening search results, screening papers for inclusion, quality appraisal of papers, interpretation of data, clinical and methodological perspective, general advice on the review.

BB: conception and design of the review, developing search strategy, screening papers for inclusion, quality appraisal of papers, interpretation of data, methodological perspective, general advice on the review, securing funding.

DECLARATIONS OF INTEREST

One of the authors (JC) was the co-author of one of the studies included in the review.

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External sources

- No sources of support supplied

INDEX TERMS

Medical Subject Headings (MeSH)

Biofeedback, Psychology [*methods]; Breath Tests; Carbon Monoxide [analysis]; Genetic Predisposition to Disease; Randomized Controlled Trials as Topic; Risk Assessment; Smoking [*adverse effects; metabolism]; Smoking Cessation [methods; *psychology; statistics & numerical data]; Spirometry

MeSH check words

Humans

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