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Review Article

Impact of smoking cessation and smoking interventions in patients with coronary heart disease

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Introduction

Although it is well known that smoking is strongly associated with coronary heart disease[1,2], many patients continue or resume smoking after being diagnosed with coronary heart disease and even after an important event such as a myocardial infarction, angioplasty or coronary bypass surgery. The evidence that smoking causes cardiovascular disease and new events in patients with coronary heart disease, among other serious disorders such as lung cancer and emphysema, justifies the promotion of smoking cessation. All recommendations on the prevention of coronary heart disease emphasize the importance of smoking cessation in the reduction of the risk of coronary death and non-fatal coronary events[3,4]. The recent EUROASPIRE study on the status of secondary prevention of coronary heart disease in nine European countries has shown, however, that success in smoking cessation among coronary heart disease patients is far from satisfactory[5]. In this era of evidence-based medicine, information from systematic reviews of published studies should guide physicians and other health professionals advising patients in smoking cessation. With this in mind, we have carried out a systematic review of published observational studies on the impact of smoking cessation on the prognosis and on smoking intervention trials in patients with coronary heart disease.

Methods

A Medline search for English language papers from 1966 until the beginning of 1999 was performed by the first author, using the keywords: 'coronary disease' or 'myocardial infarction' or 'angina pectoris' or 'angiography' or 'coronary artery bypass' or 'balloon angioplasty' (about 200 000 hits) and 'smoking (restricted to focus)' or 'smoking cessation (restricted to focus)' (about 25 000 hits), which produced a list of 1191 publications. All these publications were screened, reading titles and, if appropriate, abstracts. All articles about mortality and myocardial (re-)infarction in patients with coronary heart disease and smoking cessation after a cardiac event were selected, supplemented with references regarding occurrence of myocardial infarction and mortality in coronary patients after smoking cessation, which yielded 19 publications. Furthermore, all publications on smoking cessation interventions used as secondary prevention for coronary patients were selected. Again, related references on the effect of smoking cessation interventions in coronary patients were added, resulting in 10 articles.

For each study comparing quitters and continuing smokers, odds ratios and 95% confidence intervals for mortality alone, and the combined outcome of death and non-fatal myocardial infarction, were calculated. Chi-square tests were applied to evaluate statistical significance. Subsequently, sequential meta-analyses were performed and odds ratios with 95% confidence intervals were determined, based on the combined study data beginning with the oldest; subsequent studies were added step by step in chronological order.

Impact of smoking cessation on prognosis

Tables 1 and 2 review 19 observational studies, exploring differences in mortality or (re-)infarction between patients who quit or continue smoking after a coronary event[6–24]. Time of publication ranged from 1975 to 1997 (Table 1). Most studies concerned patients after a (first) myocardial infarction. The average age was

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approximately 56 years. Most studies had an upper age limit ranging from 60 to 67 years, while one study focussed on elderly people with a lower age limit of 55 years[17]. In all studies but one, men were over-represented; in six (early) studies only men were included[6,7,9–11,19], while in one study only women were enrolled[14]. The average number of smokers at baseline was 53%. A significant decrease in smoking can be observed over time, from 71% in the decade 1975–1984 to 33% during 1985–1994. This was in spite of the noted excess of the Japanese study, where smoking prevalence had increased, corresponding with overall smoking rates in Japan[19]. Duration of follow-up ranged from 1–13 years. Definitions of 'smoking' and 'quitting' varied, while some studies did not clearly specify smoking or quitting. To be designated a 'smoker' in studies in which smoking was defined, patients had to have smoked from one to five cigarettes a day during a period which ranged from 'at baseline' to 'the last 2 years'. Smoking cessation at follow-up was always self-reported and varied from not smoking for 3 months to 1 year. Prognosis at follow-up (up to 13 years) was, in all cases, related to smoking status determined at 1 year or earlier after baseline.

The percentage of patients who had quit ranged from 28 to 60%, with an average of 49% (Table 2). This percentage decreased from 50% in the decade 1975–1984 to 42% during 1985–1994. The percentage of deaths and, if investigated, non-fatal myocardial (re-)infarction in patients who continued smoking were compared with those who had stopped smoking after the coronary event. In all publications, smoking cessation reduced mortality at follow-up. In 10 articles a significant difference was found in mortality between the two groups: the odds ratio for dying ranging from 0·13 to 0·72 for quitters compared to those who continued smoking. In the other nine articles, most of which had a small number of patients or a shorter follow-up, a similar trend was reported without reaching statistical significance. Twelve articles assessed the occurrence of death or non-fatal myocardial (re-)infarction in the two groups. In nine of these a significant difference was found, the odds ratio diverging from 0·23 to 0·68. With regards to age, smoking cessation also improved prognosis in elderly coronary patients[10,17].

Multivariate analyses were performed in 11 of the 19 studies. Although the odds ratio increased in most cases, the effect of smoking cessation appeared to be independent. Combining the data of 13 019 smokers, of whom 5776 (44%) had quit smoking after the event, 1010 (17%) of the quitters died albeit at different follow-up intervals, compared to 1838 (25%) in the current smokers: odds ratio 0·62 (95% confidence interval 0·57 to 0·68). Sequential meta-analysis of the 19 studies (OR 0·59, 95% confidence interval 0·53 to 0·66; Fig. 1) shows convincing evidence of the effect of smoking cessation in the late 1970s, while the reduction of 40% mortality was demonstrated by 1986[16]. As regards mortality and non-fatal myocardial infarction, 1310 (23%) events occurred in the quitters as opposed to 2460 (34%) in the smokers (odds ratio 0·57; 95% confidence interval 0·53 to 0·62). The difference in inclusion criteria partly accounts for the large differences in effect between the various studies. Baseline characteristics such as age, gender, cardiac event and period of first survival differed between the studies. Also, different definitions of current smoking and former smoking were used.

Smoking cessation is considered to decrease other end-points as well. Restenosis rate 7 months after angioplasty was higher in continuing smokers than in those who stopped smoking post-PTCA (55% vs 38%, $P=0·03$)[25]. Smokers after coronary bypass surgery have a 3·3 times increased risk for re-CABG after 5 years ($P=0·03$)[23]. Prinzmetal anginal attacks appeared in 62% of the smokers vs 21% in quitters after 3 months ($P<0·05$)[26].

Trials of smoking interventions for coronary patients

Ten studies on smoking cessation interventions in patients with established coronary heart disease were published between 1974 and 1999 (Tables 3 and 4). Five studies were performed on patients who had had a myocardial infarction[27–31], two studies on patients who underwent coronary bypass surgery[32,33], two on patients with coronary heart disease on the angiogram[34,35] and one on patients with a need for cardiac treatment[36] (Table 3). Six studies were performed in the U.S.A., one in Canada and three in Europe. In almost all studies, the smoking cessation programme was part of a multifactorial intervention or rehabilitation programme, which included physical exercise (Table 4). Two studies were not randomized[27,36] and one was not controlled[27]. Smoking rates were biochemically validated in four programmes. All interventions were started in hospital and in most cases carried out by a nurse. Most counselling was individual, although some hospitals also provided additional group therapy. Written material was provided to enforce information and advice given orally. Some interventions provided additional aids, such as (relaxation) cassettes or videotapes and some prescribed nicotine substitution for highly addicted patients. In six of the interventions, follow-up telephone calls were made, varying from one post-discharge call to weekly calls. Length of contact ranged from approximately 1–5 h within 3–6 months, but many interventions were not described precisely. Most studies did not include family members in the intervention.

In six of the 10 studies a significantly higher number of quitters was found in the intervention group (Table 3). In these studies, differences between percentages in quitters in the intervention and the control group varied from 14 to 35%. In two studies no difference was found between the two groups, and in two studies more quitters were found in the intervention group, but this was not significant. Sequential meta-analysis of the

Table 1 Reviewed articles on prognosis of CHD for coronary patients after smoking cessation

Reference	Year	N[1]	Prevalence of smoking[2]	Inclusion	Age (years)	% men	FU (year)	Baseline 'smoker'		Follow-up 'quitter'
								No cigarettes/day	Duration	
Wilhelmsson *et al.*[6]	1975	405	77	1st AMI, discharged alive	<67	100	2 years	≥1	3 months	3 months post-AMI
Mulcahy *et al.*[7]	1977	190	87	1st UAP/AMI, 28 days survival	<60	100	5 years	≥5	6 months	≥6 months post-AMI
Sparrow *et al.*[8]	1978	202	52	1st AMI, discharged alive	mean 60	74	6 years	>1	≥1 year	post-MI
Salonen[9]	1980	535	60	AMI, 6 months survival	<65	100	3 years	?	?	<6 months post-AMI
Aberg *et al.*[10]	1983	983	78	1st AMI	stratified	100	5 year	≥1	<3 months	3 months post-AMI
Daly *et al.*[11]	1983	374	74	1st AMI/UAP, 2 year survival	<60	100	13 year	≥5	<6 months	≥3 months post-AMI
Rønnevik *et al.*[12]	1985	919	53	AMI, timolol-trial	?	79	1 year	continuing	?	1 months post-AMI
Perkins *et al.*[13]	1985	119	60	AMI, 1 month survival	mean 59	76	5 years	1	at baseline	post-AMI
Johansson *et al.*[14]	1985	156	61	1st MI, discharged alive	stratified	0	5 years	≥1	<3 months	3 months post-AMI
Hallstrom *et al.*[15]	1986	310	57	sudden arrest out-hospital	mean 56	80	48 months	?	?	?
Vlietstra *et al.*[16]	1986	4165	34	CAG: ≥1 vessel ≥50% stenosis	≥55	?	5 years	?	at baseline	<1 year before baseline
Hermanson *et al.*[17]	1988	1893	22	1-v ≥50%, no CABG	≥55	78	6 years	?	at baseline	<1 year before baseline
Cavender *et al.*[18]	1992	284	40	CAG ≥70% stenosis	≥65	90	10 years	?	at follow-up	6 months post-AMI
Sato *et al.*[19]	1992	87	86	AMI	>30	100	3 years	?	?	ex-smoker
Gupta *et al.*[20]	1993	225	43	CHD	mean 54	79	6 years	>5	?	since CHD
Herlitz *et al.*[21]	1995	302	37	AMI	median 70	69	5 years	?	?	at follow-up
Greenwood *et al.*[22]	1995	532	44	AMI	?	?	5·5 year	?	?	at follow-up
Voors *et al.*[23]	1996	169	41	CABG, 30 days survival	mean 53	90	15 year	?	at surgery	stopped since surgery
Hasdai *et al.*[24]	1997	1169	22	PTCA	?	?	4·5 year	?	?	?

[1]Number of patients selected for this study.
[2]Percentage of smokers at baseline.

Table 2 Mortality and incidence of coronary events in patients with coronary heart disease who quitted or continued smoking

Author	Quitters (%) at follow-up	Mortality		Univariate OR (95% CI)	Adjusted OR (95% CI)	Mortality+non fatal MI		Univariate OR (95% CI)	Adjusted OR (95% CI)
		Quitters (%)	Smokers (%)			Quitters (%)	Smokers (%)		
Wilhelmson *et al.*[6]	231 (57)	15 (7)	22 (13)†	0·48 (0·22,1·01)	No	35 (15)	53 (30)*	0·41 (0·24,0·68)	No
Mulcahy *et al.*[7]	89 (47)	13 (15)	23 (23)‖	0·58 (0·25,1·30)	No	?	?		
Sparrow *et al.*[8]	56 (28)	10 (19)	40 (30)‖	0·58 (0·24,1·30)	No	18 (33)	66 (49)‖	0·57 (0·28,1·15)	No
Salonen[9]	221 (41)	26 (12)	60 (20)†	0·56 (0·33,0·95)	0·63†[1]	?	?		
Aberg *et al.*[10]	542 (55)	97 (18)	126 (29)*	0·54 (0·40,0·74)	0·52‡[2]	201 (37)	253 (58)*	0·44 (0·34,0·57)	No
Daly *et al.*[11]	217 (58)	80 (37)	129 (82)*	0·13 (0·07,0·21)	No	?	?		
Rønnevik *et al.*[12]	551 (60)	37 (7)	29 (8)‖	0·84 (0·49,1·45)	No	81 (15)	74 (20)†	0·68 (0·43,0·99)	No
Perkins *et al.*[13]	52 (44)	9 (17)	30 (45)§	0·26 (0·10,0·65)	0·44 (0·20,0·95)†[3]	15 (29)	32 (48)†	0·44 (0·19,1·02)	No
Johansson *et al.*[14]	81 (52)	14 (17)	27 (36)‡	0·37 (0·16,0·83)	0·37	32 (37)	48 (63)§	0·37 (0·18,0·74)	No
Hallstrom *et al.*[15]	91 (29)	34 (37)	104 (47)‖	0·66 (0·39,1·12)	0·51‖[4]	?	?		
Vlietstra *et al.*[16]	1490 (36)	234 (16)	548 (21)*	0·72 (0·61,0·86)	0·57*	340 (23)	850 (32)*	0·63 (0·55,0·74)	No
Hermanson *et al.*[17]	807 (43)	210 (26)	391 (36)*	0·63 (0·51,0·77)	0·59 (0·50,0·71)*	466 (58)	825 (76)*	0·43 (0·35,0·53)	0·67 (0·59,0·83)*
Cavender *et al.*[18]	97 (34)	19 (20)	58 (31)†	0·54 (0·28,1·01)	0·64	?	?		
Sato *et al.*[19]	60 (66)	5 (9)	6 (21)‖	0·34 (0·07,1·51)	No	5 (9)	8 (29)†	0·23 (0·05,0·93)	0·32 (0·10,1·0)†
Gupta *et al.*[20]	173 (77)	56 (32)	24 (46)‖	0·56 (0·28,1·11)	No	?	?		
Herlitz *et al.*[21]	115 (53)	20 (17)	58 (31)‡	0·47 (0·25,0·85)	‖	30 (26)1yr	67 (36)‖	0·63 (0·36,1·08)	No
Greenwood *et al.*[22]	396 (74)	64 (16)	29 (21)‖	0·76 (0·51,1·12)	0·56 (0·33,0·98)	?	?		
Voors *et al.*[23]	72 (43)	26 (36)	37 (39)‖	0·92 (0·46,1·80)	1·11 (0·63,2·0)‖	37 (51)	65 (67)†	0·52 (0·27,1·02)	No
Hasdai *et al.*[24]	435 (37)	41 (9)	97 (13)‖	0·68 (0·45,1·02)	0·69 (0·47,0·98)	50 (11)	119 (16)‖	0·67 (0·46,0·97)	No
Total	5776 (44)	1010 (17)	1838 (25)	0·62 (0·57,0·68)		1310 (23)	2460 (34)	0·57 (0·53,0·62)	

†$P<0·05$; ‡$P<0·01$; §$P<0·005$; *$P<0·001$; ‖=not significant.
[1]Stratification for age, previous MI/AP, heart failure/arrest.
[2]Stratification for age.
[3]Age and gender.
[4]Adjusted for five risk strata (No CHD, ATMI, No Hx, Hx of MI, Hx of CHF).

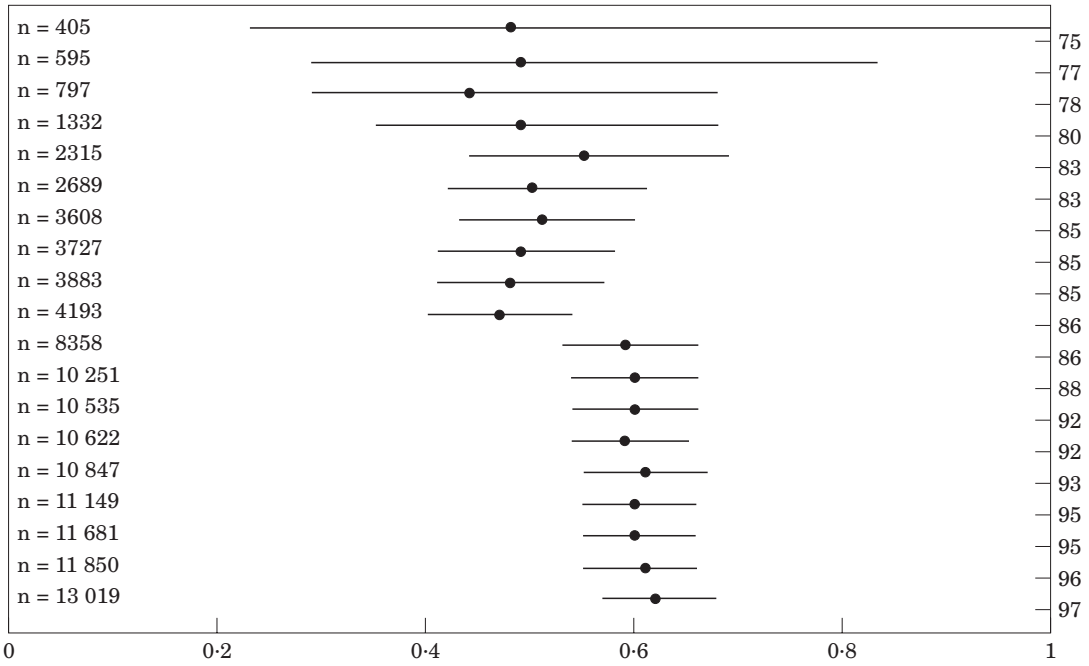

Figure 1 **Cumulative odds ratios on mortality of quitters compared to smokers of the nineteen studies.**

10 intervention studies resulted in a total population of 1685 smokers; with 61% quitters in the intervention group and 42% in the control group (OR=0·50, 95% CI 0·41 to 0·61, Fig. 2), thus 19% more quitters were observed if an intervention was offered. It is difficult to discern characteristics of the successful but very diverse studies, apart from the fact that none of the less effective studies had myocardial infarction as an inclusion criterion.

In three articles multifactorial risks were studied and smokers formed only a small part of the total study group, therefore a significant difference between the intervention group and the control group could not be expected. Interventions which had smoking cessation intervention as part of a larger programme described the intervention less extensively. This raises the question as to whether the interventions were less elaborate than programmes aimed only at smoking cessation. Differences between the diverging results can be explained by varying circumstances. Inclusion criteria, demographic characteristics, intervention, intervenor and smoking definitions differed widely, which makes comparisons among them unreliable. Moreover, because up to now only a few studies have been performed on smoking cessation interventions for coronary patients, further investigations are needed.

Discussion

Patients who continue smoking at follow-up have a worse prognosis compared to those who stop smoking after a myocardial infarction. Mortality declines by an average of 35%, and mortality or non-fatal myocardial (re-)infarction by 36% in those who have stopped smoking. The relative risk of mortality following a coronary event for quitters compared to permanent smokers ranged from 0·13 to 0·72, while the relative risk of myocardial infarction ranged from 0·23 to 0·68. Publication bias, however, should be taken into account. Short-term effects can be explained by the withdrawal of nicotine, which increases heart rate and blood pressure and thus causes an increase in myocardial oxygen demand[37], and carbon monoxide, which, by carboxyhaemoglobin formation, decreases the oxygen-carrying capacity of the blood[38]. Long-term effects could, in part, be explained by a positive effect on the lipid profile[39], but exact mechanisms are still not clear.

Many articles support smoking cessation in patients with coronary heart disease. Simple smoking cessation advice from a physician alone resulted in 3% of quitters without relapse within 1 year. Additional support in the form of letters, visits and information folders resulted in a mean efficacy of 5%[40]. Interventions in special groups, such as healthy patients with a high-risk of coronary heart disease, are effective[41]. In this review, differences of up to 35% with on average almost 20% more quitters, were found among those who participated in a smoking cessation programme, compared to those who received the usual care. Publication bias could explain the high odds ratios found in the first three studies and the gap in the studies in the 1980s. Smoking cessation seems to be most effective in myocardial infarction patients, since this is often a (first) serious warning and great emphasis is put on the risk factors. Patients, on the other hand, who undergo coronary intervention are often believed to be cured and therefore the need for smoking cessation could be underestimated by both the physician and

Table 3 Description of smoking cessation intervention studies in coronary patients

Reference	Year	Country	Population	N[1]	% smoking	Follow-up	'Non-smoker'	I/C (N)[2]	Quitters (%) (intervention)	Quitters (%) (control)	I-C (%)[3]	*P*
Burt *et al.*[27]	1974	England	AMI, male	223	78	1–3 years	CO<10, thioc<110[4]	125/98	63	28	35	<0·001
Pozen *et al.*[28]	1977	USA	AMI, <=70 years	102	?	6 months	self-reported	55/47	39[5]	21	18	<0·05
Barr Taylor *et al.*[29]	1990	USA	MI, ≤70 years	173	46	1 year	self-reported	86/87	71	45	26	<0·001
Ockene *et al.*[34]	1992	USA	post-CAG	267	26	6 months	no puff ≥1 week	135/132	62	51	11	=0·06
Engblom *et al.*[6][32]	1992	Finland	post-CABG	45	20	1 year	self-reported	25/20	44	20	24	<0·01
Rigotti *et al.*[33]	1994	USA	CABG	87	23	5·5 years	cotinine <20 ng/ml	44/43	44	44	0	ns
DeBusk *et al.*[30]	1994	USA	AMI, <=70 years	585	43	1 year	cotinine ≤10 ng/ml	293/292	70	53	17	<0·001
Haskell *et al.*[35]	1994	USA	CAG: CHD	34	11	4 years	CO, thiocyanate	12/22	0	0	0	ns
Carlsson *et al.*[31]	1997	Sweden	AMI, >50 years	67	40	1 year	self-reported	32/35	50	29	21	<0·05
Johnson *et al.*[36]	1999	USA	Cardiac treatment	102	?	6 months	self-reported	50/52	46	31	15	ns
Total				1685				857/828	61	42	19	<0·0001

[1]Number of patients forming the trial population.
[2]Number of patients in Intervention group (I) and Control group (C).
[3]Difference of number of patients in Intervention group (I) and Control group (C).
[4]Carbonmonoxide <10, Thiocyanate <110 μmol . l^{-1}.
[5]High-risk group (low-risk not mentioned).
[6]Subset of smokers in a multifactorial intervention.

Table 4 Smoking cessation interventions in coronary patients

Reference	Multifactorial	Intervention	Duration[1]	Intervenor	Indiv/group	Written info	Additional	Setting	Phone	Family
Burt *et al.*[27]	yes	Information, reinforced advice	?,?	nurse+cardiol.	indiv	info+advice	no	hosp.+fu clinic	no	yes
Pozen *et al.*[28]	yes	In-hospital talks	> ± 5 h, ?	nurse	both	literature	no	hosp+visits/phone	1/week	yes
Barr Taylor *et al.*[29]	no	In-hospital and telephone contacts	?, 6 m	nurse	indiv	manual	tapes (NRT)	hosp+outpatient clinic (if smoking)	1/w-1/m	no
Ockene *et al.*[34]	no	In-+out-hospital and telephone	1·5 h, 3–4 m	health educator	indiv (both)	if no counselling	no	hosp+outpatient clinic	4	no
Engblom *et al.*[2][32]	yes	Pre- and post-CABG rehabilitation	?, 10 weeks	multidisciplinary	both	no (?)	no	hosp/rehabilitation	no	no
Rigotti *et al.*[33]	no	3 sessions in-hospital	1 h, ?	nurse	indiv	manual	video	hosp+1 post-discharge telephone	1	yes
DeBusk *et al.*[30]	yes	Risk-factor and rehabilitation	2 h, 6 m	nurse	indiv	manual, tailored	video,tape,(NRT)	hosp (+1 visit if relapsed)	1/m	no
Haskell *et al.*[35]	yes	Rehabilitation programme	?, ?	psychologist	indiv	goal+instruction	no	out-patient clinic	no	no
Carlsson *et al.*[31]	yes	Education at outpatient clinic	1·5 h, 3 m	nurse (cardiol)	both	yes	no?	2r prevention unit	no	no
Johnson *et al.*[36]	no	Info and increasing self-efficacy	?, 3 m	nurse	group (?)	manual	video	2 in-hospital sessions	6/3 m	no

m=months ; cardiol=cardiologist; indiv=individual; NRT=nicotine replacement therapy; hosp=hospital; w=week.
[1]Number of contact hours, period of intervention.
[2]Subset of smokers in a multifactorial intervention.

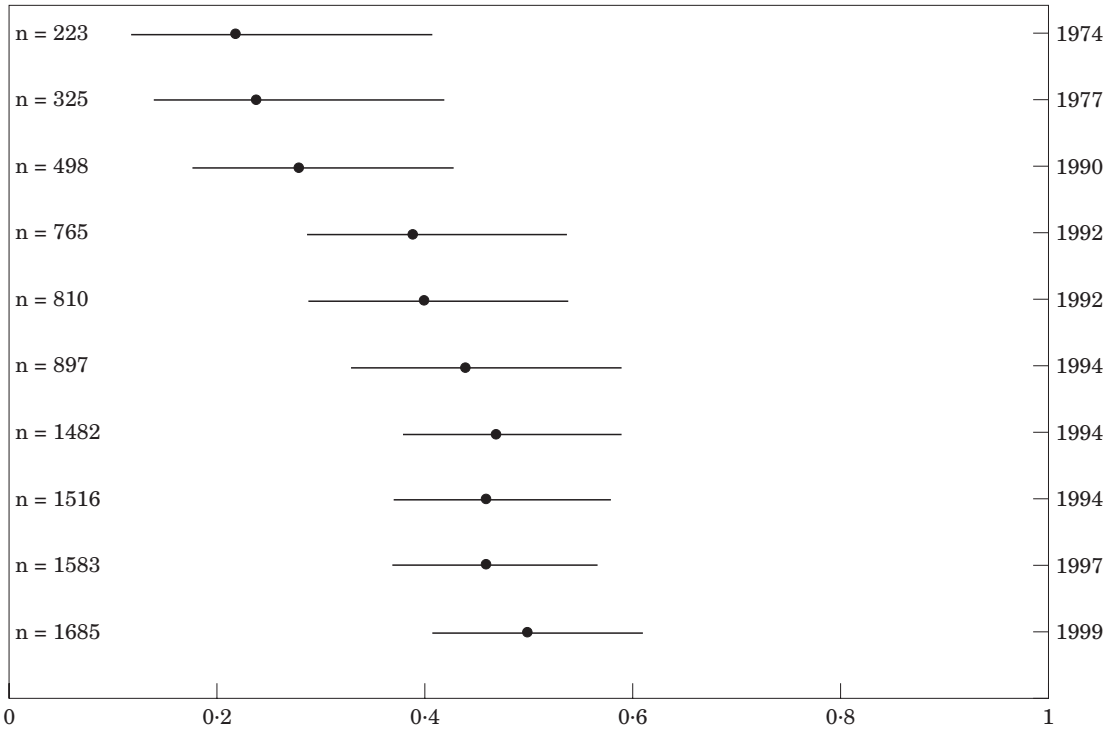

Figure 2 **Cumulative odds ratios on smoking of control patients compared to those who received a smoking cessation intervention.**

the patient. Many different approaches and tools for smoking cessation interventions are applied. To date, the most effective approach in specific categories of smokers has not been investigated. No particular intervention was shown to be more effective than any other, but reinforcement is important. In general, smoking cessation interventions, with a high number of contacts and prolonged duration, are the most successful. Furthermore, a multidisciplinary approach with face-to-face contact are determinants of success, as are the number of intervention modalities[42]. The exact content of the interventions are often not described in detail. The risk for clinical complications in coronary patients who continue smoking is obviously high, and can enhance motivation to stop smoking. So far, only general smoking cessation programmes have been used in coronary patients, and have been shown to be successful.

Nicotine substitution has proved to be effective as an aid to stop smoking[43]. However, clinicians are cautious about prescribing nicotine replacement for cardiac patients, because of adverse cardiovascular effects, particularly if patients continue smoking[44]. In a randomized, placebo-controlled study of transdermal nicotine in 156 patients with coronary heart disease, no difference in adverse affects was demonstrated between the nicotine replacement therapy group and the control group[45]. This was confirmed in a study of 584 outpatients with a diagnosis of cardiovascular disease[46].

Sociodemographic predictors of smoking cessation were shown in several studies: older age, male, higher educational level, recent hospitalization and in women the number of cigarettes and marital status[47–49]. For patients, the following clinical predictors for smoking cessation were found: a history of coronary heart disease, a long hospital (CCU) stay as well as high creatine phosphokinase elevation[47,50,51]. An intervention, such as PTCA or CABG and the presence of other coronary risk factors predicted continued smoking, whereas having unstable angina was associated with quitting smoking[48,52].

In addition, the psychological aspects of behaviour change need to be taken into account when offering cardiac patients a smoking cessation intervention. The continuing smoker has a less negative attitude to smoking and tends to be more of a worrier than the ex-smoker[47]. In a study of 164 post-myocardial patients, the relationship between personality characteristics and smoking behaviour modification was investigated. Persistent smokers appeared to have higher levels of anxiety and depression than quitters 5 months after the myocardial infarction. This applied especially to elderly smoking patients, who were also characterized by a low level of somatisation[53]. A history of a major depressive disorder is often found in smokers, while more smokers are individuals with a history of a major depressive disorder. Smokers with a major depressive disorder were less successful in their attempts to stop smoking, since smoking cessation for these patients can result in depressive symptoms[54]. Several studies found a high incidence of psychiatric disorders in cardiac patients, in particular depressive symptoms[55]. Only a

minority of depressive cardiac patients received antidepressive treatment[56], presumably because of inadequate diagnosis and reluctance by cardiologists to prescribe antidepressant drugs because of cardiovascular side-effects[57,58]. Recently, the positive effect of bupropion on smoking cessation was published, which could be of additional help in the future[59]. Smoking cessation interventions should be adjusted in the light of psychiatric disorders such as depressive symptoms, and also social, financial, and other possible individual influences should be taken into account, to enhance the effect.

Clinical implications

It is generally accepted that it is the physician's task to repeatedly draw the patient's attention to his/her unhealthy behaviour. In the new recommendations of the European Task Force on coronary prevention, it is stated that patients should be encouraged *and* supported to stop smoking[3]. However, making patients stop smoking is a difficult task. It is important to identify at what stage patients are to be given appropriate support. Prochaska *et al.* divided smokers' intentions to change in five continuous stages: pre-contemplation (do not want to stop), contemplation (consider stopping), preparation (make preparations to stop), action (attempt to stop) and maintenance (sustain non-smoking). With a few simple questions a patient's stage of change can be assessed. Matching cessation interventions to the stage of change improves its success[60]. Unfortunately, it is very difficult to offer help to smokers who do not wish to stop smoking. Information about the hazards of smoking and evidence of the effects of smoking cessation, as offered in this review, could persuade them to consider quitting. Subsequently, smoking intervention can be offered to enlarge motivation, so that preparations can be made to set a date to stop smoking. In addition to regular smoking cessation counselling, written material and possibly nicotine substitution could be offered to those heavily addicted. Individual support should also be considered, especially in patients with psychological or other personal problems. Social support seems to enhance the chance of success[61], so involvement by the family could be profitable. Research on how to help cardiac patients who continue to smoke is scarce. Studies performed so far are promising, but intervention programmes need further elaboration to assess which intervention is most effective for whom.

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