# Reduced lung function predicts increased fatality in future cardiac events. A population-based study

Running title: Lung function and case-fatality

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**Abstract**

**Objective** Moderately reduced lung function in apparently healthy subjects has been associated with incidence of coronary events. However, whether lung function is related to the fatality of the future events is unknown. This study explored whether reduced forced vital capacity (FVC) and forced expiratory volumes (FEV, 1s) in initially healthy men is related to the fatality of the future coronary events.

**Design** Prospective cohort study.

**Setting** Population-based study from Malmö, Sweden

**Subjects** 5452 healthy men, 28-61 years of age.

**Main outcome measures** Incidence of first coronary events was monitored over a mean follow-up of 19 years. The fatality of the future events was studied in relation to FEV and FVC.

**Results** 589 men suffered a coronary event during follow-up, 165 of them were fatal during the first day. After risk factors adjustment, low FEV or FVC were associated with incidence of coronary events (fatal or non-fatal) and this relationship was most pronounced for the fatal events. Among men who subsequently had a coronary event, the case-fatality rates were higher in men with low FEV or FVC. Adjusted for risk factors, the odds ratio for death during the first day was 1.00 (reference), 1.63 (95%CI:0.9-3.1), 1.86 (1.0-3.5) and 2.06 (1.1-3.9), respectively, for men with FVC in the $4^{th}$, $3^{rd}$, $2^{nd}$, and lowest quartiles (trend: $p<0.05$). FEV showed similar relationships with the fatality rates.

**Conclusion** Apparently healthy men with moderately reduced lung function have higher fatality in future coronary events, with a higher proportion of CHD deaths and less non-fatal MI.

**249 words.** Key words: Epidemiology, FEV, FVC, cardiovascular disease

## Introduction

Many prospective studies have reported inverse relationships between lung function (forced vital capacity, FVC, or forced expiratory volume, FEV) and incidence of coronary heart disease (CHD) [1-9]. These associations have generally remained significant when other cardiovascular risk factors have been taken into account and the results have been similar in studies of life-long non-smokers [4,5]. The reason for this association is still unclear, despite many years of research.

By studying the relationships between lung function and different CHD end-points, it may be possible to learn more about the relationships between lung function and CHD. Approximately 35% of men with a first acute coronary event die the first day; most of them do not reach hospital [10]. While the pathogenesis of non-fatal MI involves myocardial ischemia and occlusion of the coronary arteries, it is generally agreed that a substantial proportion of the out-of-hospital deaths are due to ventricular arrhythmia rather than coronary thrombosis [11]. Even though many studies have reported relationships between lung function and incidence of CHD, it is unclear whether reduced lung function predicts incidence of fatal events or non-fatal events, or both. Population-based studies have shown that the occurrence of ventricular arrhythmia is higher in men with reduced lung function [2,12], and that the prognosis of ventricular arrhythmia is worse in men with low FEV [2]. This suggests that reduced lung function in apparently healthy subjects could predict coronary events with high case-fatality rates. To the best of our knowledge, this hypothesis has not been explored previously.

In a previous study from the present cohort middle-aged men, it was demonstrated that high blood pressure and high plasma levels of inflammatory proteins was associated with increased case-fatality rates in men who experienced a first coronary event during the mean follow-up of 19 years [13]. The purpose of the present study was to explore whether reduced lung function at baseline predicted coronary events with increased case-fatality rates.

## Methods

Complete birth cohorts from the city of Malmö, Sweden, were invited to a screening examination [13-15]. A total of 22444 men participated (attendance rate: 71%). Information about FEV and FVC was available for 21186 of them. Hypertension and high levels of inflammatory proteins are important determinants of case-fatality after coronary events [13], and the study was therefore restricted to individuals with information on these variables. Determination of 5 plasma proteins was part of the program for 6193 men, randomly selected from birth cohorts examined between 1974 and 1982. After exclusion of 118 men with a history of MI, stroke or cancer, and 623 men with missing information about lung function, 5452 men remained with a mean age of 46.6±3.7 years (range:28-61). The health service authority of Malmö approved and funded the screening program. All participants gave informed consent.

### *Baseline examinations*

Subjects were categorized into smokers and non-smokers. Smokers were categorized into consumers of less than 10 cigarettes per day, 10-19 cigarettes and daily consumption of 20 cigarettes or more.

Blood pressure (mmHg) was measured twice in the right arm after a 10-minutes rest. The average of two measurements was used. A sphygmomanometer and a rubber cuff of appropriate size were used. Use of anti-hypertensive medication was assessed in a questionnaire.

Blood samples were taken after an overnight fast. Serum cholesterol and triglyceride concentrations were analyzed with standard methods at the laboratory of the hospital.

Physical activity was assessed in a questionnaire. Men who reported that they mostly were sedentary in spare time were considered to be physically inactive.

Information about occupation was retrieved by data linkage with the national census investigations carried out in 1975, 1980 and 1985 which are total registers of the Swedish population in those years. The subjects were classified according to the census that was closest to the screening examination. Occupation was categorised into manual workers (n=2472), non-manual workers (n=2406) and others (early retired people, unemployed, farmers, enterprisers etc, n=555). Seventeen men with missing data about occupation were included in the latter category.

FVC and FEV were measured using a Spirotron apparatus (Drägerwerk AG, Lübeck, Germany) with the subjects in a standing position without noseclips. Specially trained nurses performed the tests. One acceptable manoeuvre, with respect to the subject's performance and co-operation, was required. The volumes were standardised for age and height using an equation from a reference population of Caucasian non-smokers [16]. Residual FVC and FEV (predicted values subtracted from the observed values) were used [16]. The sample was categorised into quartiles of residual FVC (<-0.69 L, -0.69 to –0.20 L, -0.21 to +0.24 L, and >0.24 L) and residual FEV (<-0.80 L, -0.80 to –0.38 L, -0.38 to +0.01 L, and >0.01 L), in accordance with our previous studies

[9,16-18]. Expressed as percentages of predicted values, the quartiles for FVC correspond to <85, 85-95, 96-105, and >105%, respectively, and the quartiles of FEV correspond to <79, 79-90, 90-100 and >100 %.

*Inflammation-sensitive plasma proteins (ISPs)*

Electroimmuno assay was used to analyze the plasma levels of 5 inflammatory proteins [20]. The samples were analyzed consecutively at the time of screening. In accordance with previous studies, the subjects were categorized according to the number of ISPs in the 4$^{th}$ quartile [13,15]. The 4$^{th}$ quartiles were as follows: fibrinogen >4.0 gram /L, orosomucoid >0.93 g/L, $\alpha$1-antitrypsin >1.42 g/L, haptoglobin >1.76 g/L or ceruloplasmin >0.36 g/L. The reliability in terms of internal consistency was fully adequate for this composite score (Cronbach's alpha=0.65).

*Follow-up, definition of end-points*

At the baseline examination, none of the men had a history of MI according to self-report, the Malmö Myocardial Infarction Register or the Swedish Hospital Discharge register [13,21,22]. All men were followed from the baseline examination until the first coronary event, death or December 31$^{st}$, 1998.

A coronary event was defined as non-fatal MI (ICD-9 code 410) or death from CHD (ICD 410 to 414) in subjects with no prior clinical history of MI. The CHD deaths during the first day include those who died in- or outside hospital during the day of the coronary event.

Of the 165 CHD deaths that occurred during the first day, cause of death was based on autopsy in 128 (78%) cases. Of the remaining 37, cause of death was based on examinations in-hospital before death for 24 cases, on findings from examinations

outside hospital before death for 7 cases and on other sources for 6 cases. Of the 589 coronary events, 398 were non-fatal MI (i.e. survived 28 days).

*Statistics*

Analysis of variance and Pearson's Chi-square was used to study differences in risk factors between non-fatal MI and CHD deaths. Cox proportional hazards regressions were applied to calculate adjusted relative risks of coronary events. The fit of the proportion hazards model was confirmed by plotting the incidence rates over time in different categories of risk factors. Logistic regression, with fatal CHD (yes vs no) as dependent variable, was used to adjust the relationship between lung function and fatal outcome for potential confounders. The p-values for trends were obtained by modeling the quartiles of lung function as ordinal variables. Blood pressure medication, diabetes, smoking, angina, occupation and physical inactivity were modeled as categorical variables. All other covariates were modeled as continuous variables. Possible interactions were tested by entering interaction terms in the logistic regression model.

## Results

*Lung function and incidence of coronary events*

After adjustments for confounding factors, FEV and FVC were significantly associated with incidence of coronary events (fatal or non-fatal). The relationships with incidence of fatal CHD (death 1$^{st}$ day) were substantially stronger than for non-fatal MI (Table 1).

*Risk factors at baseline in relation to fatality during the first day*

The mean time period between the screening examination and the coronary event was 13.6 years. At the baseline examination, men who subsequently had fatal CHD (1st day) had significantly lower FVC (p=0.02), higher systolic (p=0.05) and diastolic blood pressure (p=0.01) and higher inflammatory proteins (p=0.03) than the non-fatal cases. Age at the coronary event was higher in fatal cases (61.7 vs 60.1 years, p=0.003). In the univariate analysis, FEV tended to be lower in fatal cases (p=0.08) (Table 2). Smoking was not associated with fatal outcome.

*Fatality in future coronary events in relation to lung function at baseline*

Table 3 presents the major cardiovascular risk factors in relation to FEV in men who later suffered an acute coronary event. Age, smoking, diabetes, triglycerides, number of elevated ISPs and occupation level was significantly associated with FEV in this group.

The figure presents the survival rates in relation to FEV quartiles at baseline during the first days after the coronary events. The increased fatality in men with low FEV was apparent already during the first day. Both FVC and FEV were significantly associated with the case-fatality rates after adjustments for risk factors. FEV was significantly associated with case-fatality in the multivariate-adjusted analysis (p=0.04), but not in the age-adjusted analysis (p=0.07) (Table 4).

When the lung volumes were used as continuous variables, both FEV and FVC were significantly associated with case-fatality after full adjustments for risk factors. One standard deviation lower FEV (0.64 L) corresponded to an adjusted odds ratio of 1.23 (CI: 1.01-1.49, p=0.04). One standard deviation lower FVC (0.73 L) corresponded to an adjusted odds ratio of 1.40 (CI: 1.06-1.84).

Interactions terms between lung function (FEV or FVC) and hypertension, inflammatory proteins or age tested possible interaction on case-fatality rates. The interaction terms were non-significant, indicating no interactions.

If case-fatality was defined as death during the first 28 days after the coronary event, both FEV and FVC were significantly associated with case-fatality after adjustments for confounding factors (not shown).

*Separate analyses of smokers and non-smokers*

A total of 210 coronary events (61 fatal) occurred among non-smokers (n=2856) and 379 coronary events (104 fatal) occurred among the smokers (n=2596). After adjustments for confounding factors, FEV was significantly associated with incidence of coronary events both among smokers and non-smokers. FVC was significantly associated with coronary events among non-smokers. In smokers, FVC was significant after adjustments for age, but not after full adjustments for risk factors. Both FEV and FVC were significantly associated with case-fatality rates in non-smokers, after adjustments for confounding factors. In smokers, the case-fatality rates were lowest in men with FEV or FVC in the top quartile. However, this relationship did not reach significance in separate analysis of smokers.

## Discussion

Many studies have shown that apparently healthy subjects with moderately reduced lung function, as measured by FEV or FVC, have increased risk of CHD [1-9]. However, to the best of our knowledge, no population study has compared the risk of fatal and non-fatal events. The present study shows that the risk of fatal events increases most in men with reduced FEV or FVC. As compared to cases with FEV or

FVC in the top quartile, the odds of dying during the first day was almost doubled among cases with low FEV or FVC. Although this needs to be replicated in other population-based studies, it should be regarded if lung function measurements are considered for risk assessment in primary prevention.

Clinical studies have reported relationships between chronic obstructive pulmonary disease and worse prognosis among patients treated with percutan coronary interventions or coronary by-pass surgery [23,24]. However, most fatal coronary events occur outside hospital, and the inclusion of out-of-hospital death is of vital importance for representative studies of the prognosis after coronary events. In this study, lung function predicted fatal outcome already when the men were healthy, many years before the coronary event. The relationship with fatal outcome is not limited to those with clinical disease.

The reason for the increased fatality rates in men with low lung function is unclear. Size and location of the infarction and ventricular arrhythmia are well-known prognostic markers among those who survive the acute phase. However, it is not possible to study these factors in population-based settings that include out-of-hospital deaths. Population-based studies have shown that ventricular arrhythmia is more common among men with reduced lung function [2,12]. Moreover, in the study 'Men born in 1914', incidence of CHD and death among men with ventricular arrhythmia was significantly higher if lung function was low [2]. Hence, relationships with ventricular arrhythmia could explain why reduced lung function is associated with incidence of fatal CHD.  It is likely that treatment for obstructive symptoms, eg, beta agonists, mainly were used by men with low FEV. Such medication could cause

ventricular arrhythmias. The relationships between FEV and case-fatality was however apparent over the whole range of FEV and not limited to those with poor lung function.

Low lung function is a risk factor for development of hypertension [17,25] and diabetes [18,19]. Low FVC is also associated with raised inflammatory markers [9,26,27]. Hypertension and left ventricular hypertrophy [28-30], diabetes [31] and low-grade inflammation [13] are factors that also have been associated with high case-fatality rates after myocardial infarction. The baseline levels of these risk factors were taken into account in the analysis. However, it is still possible that more men with low lung function developed hypertension and diabetes during the follow-up period. In part, the relationships between lung function and case fatality after future coronary events could be mediated by increased incidences of other risk factors.

We do not know how many received treatment for hypertension during the follow-up period, which is a limitation of the study. Because betablockers should be avoided in subjects with symptoms of pulmonary obstruction, it is possible that men with low FEV or FVC more often received other types of anti-hypertensive medication. This could hypothetically have reduced the survival rates. However, the results were similar after exclusion of men with blood pressure treatment, and there was no significant interaction between hypertension and lung function on the case-fatality rates.

Another limitation is that the laboratory analyses were limited to those that were available in clinical practice at the time of examination. E.g., we have no information about the subfractions of cholesterol or c-reactive protein.

Few population-based studies have investigated the prognosis after MI in relation to the previous exposure to cardiovascular risk factors [13,28-31]. With one exception [28], smoking has not been associated with case-fatality in these studies, and smoking was not associated with fatal outcome in this study. As the relationships persisted after adjustments for smoking, and were most pronounced in non-smokers, it seems unlikely that smoking explains the relationships between lung function and case-fatality.

The health examination program was performed several years before the now commonly used guidelines for standardization of spirometry were published. The equipment and procedure of the lung function test did not meet the current standards. For example, only one acceptable test was required. This can explain why the mean residual volumes among non-smokers were somewhat lower than expected from the normative data (FVC=-0.10 L, FEV=-0.26 L). Despite this limitation, the associations between FEV, FVC and cardiovascular disease were similar to the results reported from other studies. If anything, poor precision of the lung function tests should have reduced the strengths of the associations.

The death rates after re-infarctions are generally higher than after first MIs. All men in this study were without history of MI at the screening examination, according to self-report and local and national registers of hospitalized MIs. All the acute coronary events in this study were thus first clinically apparent events. However, it has been shown that many MIs are unrecognized [32]. As no ECG recordings were performed at the baseline examination, we do not known if some men had had a silent MI.

A validation study from the National Hospital Discharge Register has shown that the diagnosis ‘myocardial infarction’ is false only in 5% the cases [22]. The autopsy rates are very high, particularly for those who died outside hospital, which is a major strength of the study. Even though most of the cases that died during the first day were out-of-hospital deaths, we do not know how many died within the first hour after onset of symptoms, which is a limitation of the study.

Apparently healthy men with moderately reduced lung function have higher fatality in future coronary events, with a higher proportion of CHD deaths and less non-fatal MI. Relationships with cardiac arrhythmia could play a role for the increased cardiovascular mortality in subjects with low lung function. Although this relation needs to be replicated in other population-based studies, it should be regarded when spirometry is considered for risk assessment in primary prevention.

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Table 1. Incidence of non-fatal myocardial infarction and CHD deaths in relation to FEV and FVC in apparently healthy men.

| | **FEV** | | | | |
|---|---|---|---|---|---|
| | **Q4 (high)** | **Q3** | **Q2** | **Q1 (low)** | **P for trend** |
| N of men | 1363 | 1363 | 1363 | 1363 | |
| Coronary events (fatal or non-fatal) (%) | 102 (7.5) | 123 (9.0) | 164 (12) | 200 (14.7) | |
| Age-adjusted RR | 1.00 | 1.25 (0.96-1.6) | 1.69 (1.3-2.2) | 2.13 (1.7-2.7) | <0.001 |
| Risk factor-adjusted RR | 1.00 | 1.07 (0.8-1.4) | 1.29 (1.01-1.7) | 1.44 (1.1-1.9) | 0.001 |
| | | | | | |
| CHD deaths, 1st day (%) | 23 (1.7) | 32 (2.3) | 48 (3.5) | 62 (4.5) | |
| Age-adjusted RR | 1.00 | 1.45 (0.8-2.5) | 2.20 (1.3-3.6) | 2.91 (1.8-4.7) | <0.001 |
| Risk factor-adjusted RR | 1.00 | 1.19 (0.7-2.0) | 1.68 (1.01-2.8) | 1.95 (1.2-3.2) | 0.002 |
| | | | | | |
| Non-fatal myocardial infarction (%) | 76 (5.6) | 87 (6.4) | 105 (7.7) | 130 (9.2) | |
| Age-adjusted RR | 1.00 | 1.18 (0.9-1.6) | 1.45 (1.1-2.0) | 1.86 (1.4-2.5) | <0.001 |
| Risk factor-adjusted RR | 1.00 | 1.01 (0.7-1.4) | 1.08 (0.8-1.5) | 1.24 (0.9-1.7) | 0.11 |
| | **FVC** | | | | |
| | **Q4 (high)** | **Q3** | **Q2** | **Q1 (low)** | **P for trend** |
| N of men | 1363 | 1363 | 1363 | 1363 | <0.001 |
| Coronary events (fatal or non-fatal) (%) | 101 (7.4) | 150 (11) | 149 (10.9) | 189 (13.9) | |
| Age-adjusted RR | 1.00 | 1.49 (1.15-1.9) | 1.54 (1.20-2.0) | 1.95 (1.53-2.5) | <0.001 |
| Risk factor-adjusted RR | 1.00 | 1.31 (1.02-1.7) | 1.24 (0.96-1.61) | 1.38 (1.08-1.8) | 0.03 |
| | | | | | |
| CHD deaths, 1st day (%) | 18 (1.3) | 44 (3.2) | 44 (3.2) | 59 (4.3) | |
| Age-adjusted RR | 1.00 | 2.45 (1.4-4.2) | 2.56 (1.5-4.4) | 3.4 (2.0-5.7) | <0.001 |
| Risk factor-adjusted RR | 1.00 | 2.13 (1.2-3.7) | 2.06 (1.2-3.6) | 2.35 (1.4-4.0) | 0.007 |
| | | | | | |
| Non-fatal myocardial infarction (%) | 80 (5.9) | 97 (7.1) | 100 (7.3) | 121 (8.9) | |
| Age-adjusted RR | 1.00 | 1.22 (0.9-1.6) | 1.31 (1.0-1.8) | 1.58 (1.2-2.1) | 0.001 |
| Risk factor-adjusted RR | 1.00 | 1.07 (0.8-1.4) | 1.04 (0.8-1.4) | 1.12 (0.8-1.5) | 0.50 |

RR: relative risks adjusted for age at baseline or risk factors (age, smoking, tobacco consumption, systolic blood pressure, blood pressure medication, BMI, cholesterol, triglycerides, diabetes, physical inactivity, angina, cough, number of elevated ISPs, respiratory infection, occupation level).

Table 2. Risk factors at screening in relation to fatality during the first day in men who 13±6 years later experienced an acute coronary event.

| | **Non-fatal MI (n=398)** | **Fatal CHD (1st day) (n=165)** | **P** |
|---|---|---|---|
| FVC (%) | 92±16 | 88±17 | 0.02 |
| residual FVC (L) | -0.36±0.72 | -0.52±0.74 | 0.02 |
| FEV (%) | 86±17 | 83±18 | 0.08 |
| residual FEV (L) | -0.52±0.63 | -0.63±0.64 | 0.08 |
| | | | |
| Age at screening (y) | 47.5±3.8 | 48.0±3.6 | 0.18 |
| Age at coronary event (y) | 60.0±6.6 | 61.7±5.8 | 0.003 |
| Smokers (%) | 66 | 63 | 0.49 |
| Diabetes (%) | 7.3 | 8.5 | 0.63 |
| Cholesterol (mmol/L) | 6.0±1.0 | 6.1±1.2 | 0.29 |
| Triglycerides (mmol/L) | 1.9±1.1 | 1.9±1.3 | 0.72 |
| Systolic blood pressure (mmHg) | 133±18 | 137±18 | 0.05 |
| Diastolic blood pressure (mmHg) | 89±11 | 92±11 | 0.01 |
| Blood pressure drug (%) | 6.0 | 5.5 | 0.79 |
| BMI ($kg/m^2$) | 25.5±3.5 | 26.1±4.0 | 0.11 |
| Angina (%) | 2.5 | 2.4 | 0.95 |
| Physical inactivity (%) | 56 | 60 | 0.39 |
| Non-manual workers (%) | 37 | 33 | 0.33 |
| **ISPs in the top quartile** | | | |
| None (%) | 27 | 22 | |
| One (%) | 25 | 21 | |
| Two (%) | 18 | 18 | Trend: |
| Three or more (%) | 30 | 39 | 0.028 |

Table 3. Cardiovascular risk factors in relation to FEV among 589 men who subsequently suffered an acute coronary event.

| | **Quartile of FEV** | | | | |
|---|---|---|---|---|---|
| | **Q4 (high)** | **Q3** | **Q2** | **Q1 (low)** | **P (trend)** |
| N of men | 102 | 123 | 164 | 200 | |
| Age (y) | 48.2±3.7 | 47.6±3.5 | 47.5±3.5 | 47.7±3.8 | <0.001 |
| Age at cardiac event (y) | 61.2±6.4 | 60.4±6.2 | 60.5±6.3 | 60.3±6.6 | <0.001 |
| Smokers n (%) | 46 (45) | 78 (63) | 114 (70 ) | 141 (70) | <0.001 |
| Diabetes n (%) | 4 (3.9) | 9 (7.3) | 13 (7.9) | 28 (14) | 0.003 |
| Cholesterol (mmol/L) | 6.0±1.1 | 6.0±0.9 | 6.1±1.1 | 6.1±1.2 | 0.19 |
| Triglycerides (mmol/L) | 1.8±1.1 | 1.6±0.7 | 1.8±1.0 | 2.2±1.5 | <0.01 |
| Systolic BP (mmHg) | 136±19 | 134±19 | 134±17 | 134±18 | 0.48 |
| Diastolic BP (mmHg) | 92±11 | 89±12 | 90±11 | 90±11 | 0.38 |
| Blood pressure drug n (%) | 9 (8.8) | 4 (3.3) | 12 (7.3) | 11 (5.5) | 0.61 |
| BMI ($kg/m^2$) | 25.6±3.6 | 25.3±2.8 | 25.6±3.5 | 26.1±4.2 | 0.08 |
| Angina n (%) | 3 (2.9) | 2 (1.6) | 4 (2.4) | 7 (3.5) | 0.57 |
| Physical inactivity n (%) | 62 (61) | 59 (48) | 101 (62) | 113 (57) | 0.87 |
| $\geq$2 ISPs in top quartile, n (%) | 45 (44) | 58 (47) | 75 (46) | 117 (59) | 0.01 |
| Non-manual workers, n (%) | 44 (44) | 56 (46) | 58 (36) | 57 (29) | <0.01 |

BP blood pressure
ISP inflammation sensitive proteins

**Table 4.** Proportions of fatal CHD among 589 men with first acute coronary events, in relation to FEV or FVC 13±6 years earlier

| | **Quartile of FEV** | | | | |
|---|---|---|---|---|---|
| | **Q4 (high)** | **Q3** | **Q2** | **Q1 (low)** | **P (trend)** |
| N of men | 102 | 123 | 164 | 200 | |
| Age (y) | 48.2±3.7 | 47.6±3.5 | 47.5±3.5 | 47.7±3.8 | <0.001 |
| Age at cardiac event (y) | 61.2±6.4 | 60.4±6.2 | 60.5±6.3 | 60.3±6.6 | <0.001 |
| Fatal 1st day (%) | 22 | 26 | 29 | 31 | |
| Adjusted OR* | 1.00 | 1.26 (0.7-2.3) | 1.48 (0.8-2.6) | 1.63 (0.92-2.8) | 0.07 |
| Adjusted OR** | 1.00 | 1.33 (0.7-2.5) | 1.62 (0.9-3.0) | 1.80 (0.99-3.3) | 0.04 |
| | | | | | |
| | | | | | |
| | **Quartile of FVC** | | | | |
| | **Q4 (high)** | **Q3** | **Q2** | **Q1 (low)** | **P (trend)** |
| N of men | 101 | 150 | 149 | 189 | |
| Age (y) | 48.0±3.5 | 47.6±4.2 | 47.4±2.8 | 47.8±4.1 | 0.71 |
| Age at cardiac event (y) | 60.6±6.2 | 61.3±6.2 | 60.5±6.1 | 59.9±6.8 | 0.14 |
| Fatal 1st day (%) | 18 | 29 | 30 | 31 | |
| Adjusted OR* | 1.00 | 1.89 (1.0-3.5) | 1.98 (1.1-3.7) | 2.18 (1.2-4.0) | 0.02 |
| Adjusted OR** | 1.00 | 1.63 (0.9-3.1) | 1.86 (0.97-3.5) | 2.06 (1.1-3.9) | 0.03 |
| | | | | | |

*adjusted for age at coronary event and year of coronary event
** +smoking, tobacco consumption, systolic blood pressure, blood pressure medication, BMI, cholesterol, triglycerides, diabetes, physical inactivity, angina, cough, respiratory infection, occupation level, number of elevated ISPs.

Figure 1. Survival during the first days after the coronary events in 589 men, in relation to quartiles of FEV.