A prospective study of the development of coronary heart disease in a group of 1202 middle-aged men

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SUMMARY A prospective study of the development of coronary heart disease (CHD) in men aged 45–54 was conducted in six group practices in Belfast, Northern Ireland. Of the 1343 men examined once by clinical, electrocardiographic, biochemical, and anthropometric methods, 1202 free from CHD entered the prospective study. All 1202 men were followed for at least five years and the average annual incidence of CHD during this period was estimated as 1.73% (104 men developed). Of the 28 measurements made at initial examination, four—S-T depression, total lifetime tobacco consumption, diastolic blood pressure, and height (significant on classical linear discriminant function analysis)—were investigated in more detail. Using multidimensional contingency table methods, the probability of developing CHD was found to depend, in rank order of importance, on S-T depression, total lifetime tobacco consumption, and diastolic blood pressure. The risk of development was maximal (0.3732, four times the 'random' risk) in men with S-T depression, greater than average total lifetime tobacco consumption, and a diastolic pressure of 100 mm Hg or more.

Prospective epidemiological studies make it possible to measure the influence of personal and environmental factors which precede the emergence of CHD. A number of these studies conducted mainly outside the United Kingdom have now reported, and several so-called 'risk factors' have been identified. The present study, which utilises selected general practices in Belfast, was started in 1964 at a time when few projects of this type were being attempted on a community basis in the United Kingdom. The aim of the study was to identify social and biological characteristics of middle-aged men associated with an increased risk of developing CHD.

Methods

POPULATION STUDIED

This comprised all men born in the 10-year period 1909–18 (that is, those aged 45–54 at the beginning of the study) who were registered in six group practices in Belfast. These practices were chosen because the GPs concerned were prepared to co-operate in the study and a name-age-sex register was available for each practice. Subjects were sent a letter from their GPs explaining the purpose of the study and inviting their co-operation. Non-responders were sent a second letter after a month had passed. Those who still failed to respond

were visited at home by MG before the subject was classed as a refusal, untraced, or unfit for examination. All the subjects were men of working age and the great majority were well and at work. This, and the fact that the examination was concerned with the heart, necessitating attendance at a health centre preferably during working hours, led to some reluctance to attend for examination and to a good many refusals. The examinations started in 1964 after a pilot study and additional practices were included up to 1968. Men born in the period 1909–18 who were examined late in the study were therefore older than those examined at the beginning and some may have died from CHD before being called for examination.

All subjects have now been followed up for at least five years and some for as long as 12 years. In this report we deal with the five-year follow-up and relate the CHD status at five years to the findings at the initial examination. Table 1 shows the derivation of the sample of 1202 men who were finally included in the five-year follow-up study. All those who at the initial examination gave a history of angina of effort¹ or who had had a confirmed myocardial infarction (141 in all) were excluded. All but 46 of the initial examinations and all the follow-ups were carried out by one observer (MG).

Table 1 *Derivation of sample of men born in the 10-year period 1909-18*

Total number of men aged 45-54 on the six practice registers		1943	(100.0)
Exclusions			
Refusals	357	(18.4)	
Untraced	83	(4.3)	
Unfit	74	(3.8)	
Transferred	21	(1.1)	
Incomplete data	65	(3.3)	
	600	(30.9)	
Initial examinations carried out		1343	(69.1)
Subjects with evidence of CHD		141	(7.3)
Present angina	86	(4.4)	
Past angina	4	(0.2)	
History of myocardial infarction	51	(2.6)	
Subjects without evidence of CHD and used in five-year prospective survey		1202	(61.9)
Percentages in brackets			

Table 2 *Distribution by civil state of the men examined compared with all men in Belfast aged 50-59 (1966 census)*

Civil state	Sample		Belfast	
	No.	%	No.	%
Single	85	(6.3)	2 781	(12.7)
Married	1218	(90.7)	18 124	(82.9)
Other	40	(3.0)	954	(4.4)
Total	1343	(100)	21 859	(100)

$\chi^2 = 65.77$ df = 2 P < 0.001

Because the practices were not randomly selected, the question arises of the representativeness of these men in relation to the general population. The distributions of the sample by social class and civil state were examined for this purpose.

Table 2 shows that the sample differed from the population of Belfast with regard to distribution by marital state. The small proportion of single men in the sample may have been due to the greater difficulty of contacting single men of these ages and persuading them to come for a medical examination.

Table 3 *Distribution by social class of the sample of men examined compared with men of similar age in Northern Ireland in 1971*

Social Class	Sample		Northern Ireland age groups 45-54		55-59	
	No.	%	No.	%	No.	%
I	67	(5.0)	2 681	(3.4)	1 089	(3.1)
II	324	(24.1)	19 624	(25.2)	9 355	(26.4)
III	695	(51.8)	30 813	(39.6)	12 356	(34.8)
IV	176	(13.1)	14 713	(18.9)	7 192	(20.3)
V	81	(6.0)	9 655	(12.4)	5 364	(15.1)
Unclassified	0	(0.0)	400	(0.5)	147	(0.4)
Total	1343	(100.0)	77 886	(100.0)	35 503	(100.1)

Information on social class distribution of men in Belfast was not obtainable. A comparison was therefore made with men in Northern Ireland (Table 3). The Table shows that the study sample differed somewhat from the population of Northern Ireland men of similar age in the distribution by social class. There was a larger proportion of men in Social Classes I and III and a smaller proportion in Social Classes IV and V in the sample. The results with regard to the incidence of CHD or the frequency of the various factors cannot therefore be assumed to be true for the whole male population of Northern Ireland or Belfast.

VARIABLES STUDIED

These included the 15 continuous and 13 categorical measurements listed in Table 4. Total lifetime tobacco consumption was estimated by multiplying daily consumption in grams by duration of smoking and aggregating the amounts for the different periods of the man's smoking life. The systolic and diastolic blood pressures were measured with the subject sitting after 10 minutes rest. The neuroticism and introversion scores were derived from answers to a personality inventory questionnaire.² Weight was measured to the nearest 0.5 kg with the man in socks and trousers only, with pockets empty, and height was measured to the nearest 1.0 cm with the man in socks. The skinfold thickness was measured with the Harpenden caliper³ at six sites: the mid-point of the posterior surface of each upper arm, over the inferior angle of each scapula, and two inches above each anterior superior iliac spine. The mean of the six measurements was used. A non-fasting sample of 10 ml of venous blood was centrifuged at 1700 rpm for one hour to remove particulate lipids⁴ and the serum cholesterol,⁵ total fatty acid,⁶ and alpha beta lipoprotein ratio⁷ were estimated. Haemoglobin concentrations were estimated for all subjects in order to exclude cases of angina pectoris attributable to anaemia. One subject was excluded for that reason. Subjects were classified by social class, socioeconomic group, and economic position

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Table 4 *Factors studied*

Continuous measurements	Categories
1. Age	16. Civil state
2. Total tobacco consumption	17. Social class
3. Current tobacco consumption	18. Socioeconomic group
4. Systolic blood pressure	19. Economic position
5. Diastolic blood pressure	20. Physical activity
6. Neuroticism score	21. Alcohol drinking habit
7. Introversion score	22. Arcus lipoides
8. Weight	23. Q/QS
9. Height	24. T-wave changes
10. Skinfold thickness	25. S-T wave changes
11. Serum cholesterol	26. Current cigarette smoking
12. Serum total fatty acids	27. Mother alive or dead
13. Alpha beta lipoprotein ratio	28. Father alive or dead
14. Age of father at death	
15. Age of mother at death	

according to the Registrar General's *Classification of Occupations*.⁸ The amount of physical activity of each subject was derived from information about the nature of his work, the distance walked or cycled to and from work each day, and the estimated number of leisure hours a week spent on physical activities (see Appendix).

The amount of alcohol consumed was graded as follows: Teetotal always; teetotal now; takes alcohol less often than once a week; once a week or more often but not daily; daily.

Arcus lipoides was graded into: Grade 0 = absent; Grade 1 = incomplete; Grade 2 = complete in both eyes.

An electrocardiographic examination was carried out on each subject. The standard 12 leads were recorded with the subject lying down and after a three-minute stepping exercise at a standardised work rate of 350 kg miles/min.⁹ All the ECGs were read by one cardiologist and coded according to the Minnesota code.^{10 11} Three types of ECG abnormality were analysed in relation to the future development of CHD: Minn I 1-3 (Q/QS changes); Minn IV 1-3 (S-T depression); and Minn V 1-3 (T-wave changes).

THE FOLLOW-UP

After the initial examinations, the subsequent CHD status of the subjects was ascertained in three ways:

- The Registrar General of Northern Ireland was provided with a list of the names and addresses of all the subjects and notified us from time to time of deaths and causes of death of men in our sample.
- Each subject was sent a letter annually inquiring about any pain in the chest and, if present, its relation to exertion.
- MG visited the general practices periodically and examined the records of all the subjects and consulted the GP when the reply to the

letter suggested the development of angina. For subjects who had moved out of the district the names of their new GPs were obtained and inquiries sent to them.

Subjects suspected of having angina pectoris or cardiac ischaemia were referred to a medical outpatient department and diagnosed as such only on the opinion of a consultant physician. Similarly, with the exception of two sudden deaths, subjects were included in the myocardial infarction group only after they had been so diagnosed by a hospital consultant. Of the two sudden deaths ascribed to CHD, one was confirmed at necropsy, and in the case of the other, who died suddenly while on holiday in the Irish Republic, the diagnosis was based on the history.

Results

INCIDENCE OF CHD

Of the 1202 subjects judged to be free of clinical CHD at the initial examination, 27 had died of other diseases by the end of the five-year follow-up period and three men could not be traced. This left 104 men who developed CHD, 'the developers', and 1068 who remained free of it, the 'non-developers' (Table 5). These two groups have been compared in relation to risk factors. Developers were defined as men who developed angina pectoris or cardiac ischaemia or who had a proven myocardial infarction. The 104 developers included 49 who developed angina alone, or cardiac ischaemia (five), and 55 (15 fatal) who developed myocardial infarction with or without preceding or succeeding angina. The morbidity rate in this sample was therefore 1.7% per annum and the mortality rate 0.25% per annum.

Table 5 *CHD status after five years of the 1202 men included in the study*

No evidence of CHD	1068
Developed CHD	104
Myocardial infarction (died of MI:15)	55
Angina (or cardiac ischaemia: five cases) only	49
Died of other causes	27
Untraced	3
Total	1202

COMPARISON OF ANGINAL AND MI CASES

Although it is generally believed that angina pectoris and myocardial infarction are usually different manifestations of the same disease, it is possible that the same risk factors do not apply to both conditions or do not apply equally. It was considered desirable, therefore, to examine the mean values of the continuous variables and the distributions of the

Table 6 Mean initial values of the continuously distributed factors in those who developed angina only and those who developed myocardial infarction with or without angina

Factors	Mean levels		Difference A - B ($\pm$ SE)	t
	A Developed angina n = 49	B Developed myocardial infarction n = 55		
Age (years)	53.51	53.53	-0.02 (0.67)	0.03
Total tobacco consumption (kg)	199.54	199.44	+0.10 (33.33)	0.003
Current daily tobacco consumption (g)	17.33	16.64	+0.69 (2.30)	0.30
Total years of smoking	27.67	28.95	-1.28 (2.67)	0.48
Systolic blood pressure (mm Hg)	138.37	140.73	-2.36 (4.63)	0.51
Diastolic blood pressure (mm Hg)	87.14	86.09	+1.05 (2.63)	0.40
Neuroticism (Heron 1 score)	6.90	5.93	+0.97 (0.66)	1.46
Introversion (Heron 2 score)	5.32	5.58	-0.26 (0.46)	0.57
Weight (kg)	75.57	74.44	+1.13 (2.09)	0.54
Height (cm)	169.67	170.76	-1.09 (1.03)	1.06
Skinfold thickness (mm)	15.59	14.67	+0.92 (1.21)	0.76
Serum cholesterol (mg/100 ml)	227.84	227.20	+0.64 (6.40)	0.10
Serum total fatty acids (mmol/l)	13.89	14.48	-0.59 (0.73)	0.81
Alpha beta lipoprotein ratio	0.71	0.74	-0.03 (0.54)	0.55
Age of father at death (years)	63.38	66.38	-3.02 (2.93)	1.03
Age of mother at death (years)	66.48	68.49	-2.01 (3.29)	0.61

categorical variables in these two groups to see whether there were any significant differences between them.

The mean initial values of the continuous variables are shown in Table 6 for these two groups. None of the mean values differ significantly in the two affected groups.

The distributions of the categorical variables in the angina and myocardial infarction groups were also examined and were shown not to differ significantly except for the socioeconomic group. The difference in this case was due to the fact that the group described as 'employers and managers' were more

likely to develop myocardial infarction than angina pectoris alone when compared with the whole group. With this single exception, our findings therefore supported the accepted view that angina of effort and myocardial infarction are manifestations of the same disease process and that any risk factors which are identified will apply to both. In all subsequent Tables the two groups are therefore amalgamated.

COMPARISON OF CONTINUOUS FACTORS IN DEVELOPERS AND NON-DEVELOPERS

Table 7 shows the mean values of the continuous factors at the initial examination in those who

Table 7 Mean initial values of continuously distributed factors in those who developed CHD and those who did not

Factors	Mean levels		Difference A - B ($\pm$ SE)	t
	A Developed CHD n = 104	B Did not develop CHD n = 1068		
Age (years)	53.52	53.16	+ 0.36 (0.36)	1.00
Total tobacco consumption	199.48	156.77	+42.71 (14.48)	2.95*
Current daily tobacco consumption (g)	16.96	14.19	+ 2.77 (1.19)	2.32*
Total years of smoking	28.35	24.60	+ 3.75 (1.46)	2.56*
Systolic blood pressure (mm Hg)	139.62	135.14	+ 4.48 (2.19)	2.04*
Diastolic blood pressure (mm Hg)	86.59	83.39	+ 3.20 (1.23)	2.60*
Neuroticism (Heron 1 score)	6.39	5.86	+ 0.53 (0.36)	1.49
Introversion (Heron 2 score)	5.46	5.60	- 0.14 (0.26)	0.54
Weight (kg)	74.97	74.24	+ 0.73 (1.11)	0.66
Height (cm)	170.25	171.71	- 1.46 (0.66)	2.21*
Skinfold thickness (mm)	15.11	14.12	+ 0.99 (0.57)	1.75
Serum cholesterol (mg/100 ml)	227.50	220.93	+ 6.57 (3.17)	2.07*
Serum total fatty acids (mmol/l)	14.20	13.67	+ 0.53 (0.30)	1.79
Alpha beta lipoprotein ratio	0.724	0.772	- 0.048 (0.03)	1.80
Age of father at death (years)**	64.91	66.53	- 1.62 (1.62)	1.00
Age of mother at death (years)***	67.52	67.23	+ 0.29 (1.71)	0.17

* Significant where $t = 2$ $P = 0.05$, $t = 3$ $P = 0.01$.

** 1015 deceased fathers.

*** 851 deceased mothers.

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developed the disease and in those who did not. The following were significantly greater in the developers:

- Total tobacco consumption.
- Current tobacco consumption.
- Duration in years of smoking.
- Systolic blood pressure.
- Diastolic blood pressure.
- Serum cholesterol.

The mean height of the developers was significantly less than that of the non-developers.

No significant relationship was found between the risk of developing CHD and (mean) age, neuroticism score, introversion score, weight, skinfold thickness, serum total fatty acids, alpha beta lipoprotein ratio, and age of either mother or father at death. The lack of relationship to age may be explained by the narrow age band (10 years) of the subjects.

COMPARISON OF CATEGORICAL FACTORS IN DEVELOPERS AND NON-DEVELOPERS

Economic position

Table 8 shows that those who were working at the time of the initial examination were significantly less likely to develop CHD than the group of men who

were unemployed or had been off work due to non-CHD sickness for more than six months or who had retired prematurely.

ECG findings

Table 9 summarises the ECG findings. Of the 74 men who had Q/QS changes at the initial examination 11 (14.9%) developed CHD, whereas of the 1098 men without Q/QS changes only 93 (8.5%) developed CHD. This difference did not, however, reach the conventional level of statistical significance. Those who had T-wave changes or S-T depression at the initial examination were significantly more likely to develop CHD in the next five years than those who did not have these abnormalities.

The risk of developing CHD with particular combinations of these three ECG abnormalities rose from 79 (7.83%) in the 1009 men with none of these ECG changes at the initial examination to three (50%) of the six men who had all three. A hierarchical log-linear analysis of Table 9 revealed that the probability of developing CHD depended mainly on the presence of S-T depression. The expected numbers on this model are shown in brackets. There are no statistically significant departures between

Table 8 *Distribution of developers and non-developers by economic position*

CHD status after five years	Economic position				Total	
	1	2	3	4		
	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)
Developers	94 (8.4)	5 (16.7)	4 (26.7)	1 (12.5)	104	(8.9)
Non-developers	1024 (91.6)	25 (83.3)	11 (73.3)	7 (87.5)	1067	(91.1)
Total	1118 (100)	30 (100)	15 (100)	8 (100)	1171	(100)

$$\chi^2 = 6.28 \quad df = 3 \quad 0.02 > P > 0.01$$

1. Employed.
2. Unemployed at time of interview.
3. Off sick for >6 months (not CHD).
4. Retired.
- 2, 3, and 4 grouped together for χ^2 .

Table 9 *Distribution of developers and non-developers by three ECG abnormalities*

ECG abnormality			Non-developers	Developers	A %	B %
Q/QS	T-wave	S-T depression				
-	-	-	930 (925.52)*	79 (83.48)	7.83	7.83
+	-	-	49 (50.45)	6 (4.55)	10.91	12.31
-	+	-	44 (45.86)	6 (4.14)	12.00	
-	-	+	21 (19.27)	4 (5.73)	16.00	
+	+	-	8 (9.17)	2 (0.83)	20.00	22.22
+	-	+	3 (2.31)	0 (0.69)	0.00	
-	+	+	10 (10.79)	4 (3.21)	28.57	
+	+	+	3 (4.63)	3 (1.38)	50.00	50.00
All subjects			1068	104	8.87	

- + = present A = % developed
 - = absent B = grouped % developed
 * Expected numbers in brackets (see text).

observation and expectation ($\chi^2 = 7.53$, $df = 6$, $P > 0.05$). It should be emphasised that the majority of the men who developed CHD (79 out of 104) showed no ECG abnormalities at the initial examination.

Other categorical factors

No relationship was found between the development of CHD and the individual distributions of the following categorical variables: social class, socioeconomic group, physical activity, frequency of alcohol consumption, presence or absence of arcus lipoides, or whether the subject's father or mother was alive or dead.

MULTIVARIATE ANALYSIS

With relatively few developed cases of CHD the hierarchical classification of the data in the measured factors is impracticable. Therefore, to reduce the number of variables for multivariate study on objective and reproducible criteria, a stepwise forward linear discriminant analysis was carried out on 13 continuous and 10 categorical variables (nos. 1 to 13 and 16 to 25, Table 4). Before the analysis the categorical variables were given an appropriate design matrix parameterisation. The resulting discriminant function, here formally equivalent to a correlated main effects model, contained only four variables: S-T depression (partial $F = 11.24$); total tobacco consumption (partial $F = 10.45$); diastolic blood pressure (partial $F = 5.71$); and height (partial $F = 4.13$). Using these four variables, the linear discriminant explained only 16.51% of the total sums of squares available for partitioning and on this criterion must be considered an inadequate predictive tool. Furthermore, it is known that possible interactions between the variables are ignored by this technique.

In order to investigate further the probability of developing CHD in the five-year period, the simultaneous distribution of the data in the four factors selected by the discriminant function analysis

was subjected to hierarchical log-linear analysis. Before analysis the three continuous variables were categorised as: diastolic blood pressure <80 , 80 to 99.99, 100+; height ≤ 171 cm (mean height for sample), 172+ cm; total tobacco consumption ≤ 439 (mean grams lifetime consumption/365), 440+. The severity of this categorisation merely reflects the small number of developers available for analysis.

The hypothesis that the probability of developing CHD is independent of the four factors studied was easily rejected ($\chi^2 = 52.16$, $df = 23$, $P < 0.05$). The model containing all possible two factor interaction terms between CHD and the other factors was found to fit the data satisfactorily ($\chi^2 = 24.65$, $df = 18$, $P > 0.05$) although further investigation revealed the height term to be redundant. Accordingly, Table 10 shows the conditional probabilities of developing CHD in the next five years derived from the reduced model which excludes the height by CHD interaction term.

The maximum risk of development (0.3732) occurs in men with S-T depression and above average total tobacco consumption and a diastolic blood pressure of 100+. For these men the risk of developing CHD is approximately four times greater than that noted in the study population as a whole, in which the undifferentiated, or random, risk was 8.87%. The minimum risk occurred among men with no S-T depression whose total tobacco consumption was average, or less than average, and whose diastolic pressures were less than 80. In general, the probability of development increased with the presence of S-T depression, greater than average total tobacco consumption, and rising diastolic pressure. From Table 10 it is apparent that S-T depression was the major determinant: with one exception (average or less than average total tobacco consumption and diastolic pressure <80), the conditional probabilities of development were higher—on average 2.5 times greater on a corresponding cell basis—when this ECG abnormality was present. Formal testing, based on

Table 10 Estimated* conditional probabilities of development of CHD by S-T depression, total tobacco consumption, height, and diastolic blood pressure

Diastolic blood pressure (mm Hg)	S-T (+)				S-T (-)			
	Smoking** (+)		Smoking (-)		Smoking (+)		Smoking (-)	
	Ht (+)	Ht (-)	Ht (+)	Ht (-)	Ht (+)	Ht (-)	Ht (+)	Ht (-)
<80	0.2349	0.2349	0.1282	0.1282	0.0872	0.0872	0.0438	0.0438
80-99.99	0.3012	0.3012	0.1711	0.1711	0.1182	0.1182	0.0604	0.0604
100+	0.3732	0.3732	0.2219	0.2219	0.1563	0.1563	0.0815	0.0815

+ = present, or greater than average level.

- = absent, or less than or equal to average level.

* = on the basis of a log-linear model (see text).

** = total tobacco consumption.

the degradation of the χ^2 goodness-of-fit statistic, when the two factor interaction terms in the reduced model were systematically deleted, confirmed the prominence of S-T depression, and ranked total tobacco consumption before diastolic blood pressure in order of importance.

Discussion

The incidence of CHD in this study was on average 1.7% per annum, a figure similar to that of 1.5% per annum found in the Framingham study¹² for men aged 45–62.

Like several other investigations,¹³ this one has shown that the following characteristics, considered singly, are significantly and positively associated with the risk of developing CHD: tobacco consumption, systolic and diastolic blood pressure, serum cholesterol, and certain ECG abnormalities. We also found that height was significantly and negatively associated with this risk. In general, these risk factors seldom exert mutually independent effects and using a combination of multivariate methods the probability of a CHD-free man developing the disease within five years was found to depend on only three characteristics measured at initial examination. These were S-T depression in the ECG, total lifetime tobacco consumption, and diastolic blood pressure. While the chance of development was maximal (0.3732) when these three factors assumed their uppermost values, the absolute risk of developing CHD was still small, over the period of observation. Care must be exercised when interpreting the findings of longitudinal studies. The results are conditional on the particular population studied and on the length of the follow-up period and, conceivably, different results may obtain later in this study as more CHD-free men develop the condition. Also, in this study measurements made at a single examination are related to the future development of CHD; consequently there is no evaluation of subsequent risk factor modification. It is probable that some men who would initially have satisfied the entry criteria for the prospective study had developed the condition by the time they were contacted and were therefore excluded. The actual incidence of the condition may therefore be higher than the figure reported. The relatively small numbers developing CHD precluded the simultaneous investigation of more than four risk factors by hierarchical methods, and while the adopted analytical compromise appears reasonable it cannot be considered exhaustive.

It would seem that with current knowledge the majority of cases of CHD are unpredictable. However, it could be argued that, since prediction is

often based on only one initial examination, more frequent follow-up examinations for selected characteristics, together with longer periods of observation, would make possible the compilation of more accurate prognostic indices. It could also be argued that the rather poor prediction which has been found so far indicates that we have identified only some of the characteristics associated with the development of CHD, and that there must be other more significant but as yet undiscovered factors which are concerned in its aetiology.

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Appendix

Estimate of physical activity of each subject

The men were ranked 1–3 for occupational activity using the classification (1, 3, and 5) prepared by Dr. R. Murray and Dr. D. Turner for use in the National Necropsy Survey.¹⁴ The classification is in terms of the physical activity habitually and typically involved in the given occupation.

Light work = 1
Active work = 2
Heavy work = 3

The men were ranked 1–3 for leisure activity from the sum of the leisure hours spent in physical

activities each week and the hours spent walking or cycling to and from work each week. Three miles walking or nine miles cycling were taken as equivalent to one hour of physical activity.

Under five hours leisure activity a week	= 1
Five to 9 hours inclusive leisure activity a week	= 2
Ten hours or more leisure activity a week	= 3

The two ranks were summed to give a total activity score.

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