µM oligonucleotide primers *5FU, 3FU,* and *5WK;* 0·1 µM *3M;* 200 µM dNTPs; 1× reaction buffer (43 mM potassium chloride, 8·6 mM Tris-hydrochloric acid (pH 8·3), 2·5 mM magnesium chloride, 0.008% gelatin); and 2 units DNA Taq polymerase (Boehringer Mannheim) overlaid with mineral oil.

The reaction mixture (40 µl) without enzyme was heated to 95°C for five minutes in a thermal cycler (Hybaid) and held at 80°C for the addition of enzyme (2 units of enzyme in 10 µl reaction buffer). Reaction conditions then followed 35 cycles of 94°C for one minute, 60°C for two minutes, and 72°C for two minutes and one cycle of 72°C for 10 minutes. Amplified products were separated in a 3% (3:1 LMP agarose:Nusieve) gel containing ethidium bromide and visualised under ultraviolet light. Three bands potentially resulted from the primer combinations: *5FU-3FU* gave a 459 bp control band; *5WK-3FU* gave a 353 bp band in the presence of the "wild type" *Ile181;* *3M-5FU* gave a 163 bp band in the presence of *Leu181.*

A member of each family segregating *Leu181* was sequenced by the method of Sanger *et al*[17] to ensure accuracy of the polymerase chain reaction and to determine whether *Leu183* was present. The 459 bp *5FU-3FU* band from the above reaction was taken to second round polymerase chain reaction with the following internal primers: *5D* (5'biotinylated)—AAG GACTGGTCAGATGGTAG; *3D*—GGCTTCTAT CTA CCT TGT TTC. A single strand template was prepared with strepavidin labelled magnetic beads (Dynal, Oslo, Norway), and direct solid phase sequencing followed with the sequencing primer *3GS*–TCC TTT GAG TTC TTC CCC A.

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Total cholesterol concentration and mortality at a relatively young age: Do men and women differ?

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Abstract

Objective—To investigate the relation between total cholesterol concentration and mortality from coronary heart disease, cardiovascular diseases, non-cardiovascular causes, and all causes.

Design—Population based cohort study.

Subjects—23 000 men and 26 000 women aged 30-54 years examined between 1974 and 1980.

Main outcome measures—Mortality for the above mentioned end points for fifths of cholesterol distribution, and relative risks estimated by using Cox's proportional hazard (survival) analysis. Adjustment was made for age, smoking, systolic blood pressure, and body mass index.

Results—Mortality from coronary heart disease in men was five times higher than that in women. A strong positive association between total cholesterol concentration and mortality from coronary heart disease and cardiovascular diseases was observed in both men and women. The relative risk for the highest compared with the lowest fifth of the cholesterol distribution was for mortality from coronary heart disease (3·0 (95% confidence interval 1·8 to 5·1) in men and 3·8 (1·1 to 13·1) in women) and for mortality from cardiovascular disease (2·8 (1·8 to 4·2) in men and 2·9 (1·4 to 6·0) in women). No increase of non-cardiovascular mortality at low cholesterol concentration was observed. All cause mortality was significantly higher in the highest compared with the lowest fifth of the cholesterol distribution: relative risk 1·6 (1·3 to 2·0) in men and 1·5 (1·1 to 1·9) in women.

Conclusion—Total cholesterol concentration is a strong predictor of mortality from coronary heart disease, cardiovascular diseases, and all causes in women as well as in men. Low cholesterol concentrations are not associated with increased mortality from non-cardiovascular causes.

Introduction

There is growing awareness that cardiovascular diseases are not only an important public health problem in men but also in women.[1][2] In the Netherlands

about 40% of total mortality is caused by cardiovascular diseases, both in men and women.[3]

Because most research on cardiovascular disease has been carried out on middle aged men longitudinal data for women are still relatively scarce. In the Netherlands a screening project on risk factors for cardiovascular disease has been carried out between 1974 and 1980 in which 50 000 men and women aged 30-54 years were examined.[4] Mortality follow up of this cohort has recently been completed, and to our knowledge this is one of the largest cohorts that provides information on total cholesterol concentration in relation to mortality in women.

Although it has been shown in men that the relation between total cholesterol concentration and mortality from coronary heart disease is strong and graded,[5][6] the risk of low concentrations is debatable.[7] There are indications that the benefit with respect to cardiovascular mortality would be offset by an increase in mortality from cancer and other diseases.[6][8] Therefore we also investigated the associations between total cholesterol concentration and non-cardiovascular mortality.

Subjects and methods

Population—The consultation bureau project on cardiovascular diseases was carried out from 1974 to 80 in five towns in the Netherlands: Amsterdam, Doetinchem, Maastricht, Leiden, and Tilburg.[4] The response rate varied from 70 to 80%. The project was aimed primarily at the age group of around 40 years, but in some towns a wider age range was taken. The age range of the population examined was 30 to 54 years, with about 15% aged 30-34 years, 45% aged 35-39 years, 30% aged 40-44 years, and 10% aged 45-54 years.

Examination—Information about smoking habits and cardiovascular complaints and the respondents' history of myocardial infarction, stroke, hypertension, and diabetes mellitus was obtained from questionnaires. Blood pressure, weight, and height were measured. A non-fasting blood sample was taken in which total cholesterol concentration was measured (with an accuracy of 0·1 mmol/l) at the Central clinical chemistry laboratory of the University Hospital Dijkzigt in Rotterdam. This laboratory participated in the standardisation programme of the World Health Organisation (WHO). Total cholesterol concentration was determined according to a direct Liebermann-Burchard method.[9] In more recent projects cholesterol concentration has been determined enzymatically, and cut off points for hypercholesterolaemia according to the Netherlands Cholesterol Consensus are based on enzymatic values.[10] Therefore cholesterol values were converted based on a comparison study carried out in 1981 in which cholesterol was measured with both methods in over 5000 serum samples. The following equation for conversion from Huang to enzymatic values was obtained[11]:

$$\text{total cholesterol}_{\text{enzymatic}} = 1 \cdot 004 \star \text{ total cholesterol}_{\text{Huang}} - 0 \cdot 299 \text{ mmol/l}.$$

Mortality follow up—Mortality follow up was initiated in 1986 and completed in 1993. The administration cards of the project were retrieved from the archives and entered on to a computer. The vital status was checked through the municipal registries. Census date was the date on which the information was obtained from the municipal registry (for the living) or date of death. The minimum length of follow up was therefore six years (person examined in 1980 and checked for vital status in 1986) and the maximum 19 years (person examined in 1974 and checked for vital status in 1993). The total number of person years was over 275 000 in men and over 300 000 in women; mean (SD) duration of follow up was 11·8 (2·3) years for men

and 12·0 (2·2) years for women. If a person had moved to an unknown destination the date on which the person was dropped from the municipal registry was used as census date. For 49 202 of the 50 887 people examined the administration card was still present in the archive. For 49 018 of these mortality follow up was successfully completed, resulting in a loss to follow up of only 3·7%. A total of 1319 people had died. For 1289 subjects the cause of death was obtained from the Central Bureau of Statistics, but for 30 people who died outside the Netherlands such information could not be obtained. Causes of death were coded according to the ninth revision of the *International Classification of Diseases* (ICD-9). For deaths that had occurred before 1 January 1979 (n=89) the eighth revision (ICD-8) was used. For the end points used in the present analyses all codes from ICD-8 remained in the same category when defined according to ICD-9. Cause specific end points used in the analyses were coronary heart disease (ICD 410-414), cardiovascular disease (ICD 390-459), cancer (ICD 140-239), and non-cardiovascular and non-cancer (ICD 0-139, 240-389, 460-999).

Statistical methods—SAS statistical programs were used for the analyses.[12] Cholesterol concentration was divided into sex specific fifths and into categories of 0·5 mmol/l width. Cox's proportional hazards (survival) analysis was used to calculated hazard ratios. These hazard ratios provide a good estimate of the more commonly used relative risk and will be referred to as such in this paper. Adjustment for smoking was made based on a dichotomous variable "current smoking: yes or no" whereas age, systolic blood pressure, and body mass index (weight (kg)/height (m)^2) were entered into the model as continuous variables. Trend tests were performed for both linear and non-linear (J shaped or U shaped) associations, but there were no significant differences in fit between linear and non-linear models. In the tables the P values for the linear trend tests are presented. For cause specific analyses subjects with an unknown cause of death were excluded. Exclusion of prevalent cases of angina, stroke, and myocardial infarction (n=1349) did not substantially alter the results, and therefore they were included in the analyses.

Results

The overall mean age was 39 years: the total cholesterol concentration was 5·55 mmol/l in men and 5·19 mmol/l in women. For all cardiovascular risk factors levels were higher in men than in women (table I). In men cardiovascular diseases, cancer, and non-cancer or non-cardiovascular diseases each contributed about a third to total mortality. In women cancer was the predominant cause of death (table II).

Mean total cholesterol concentration per fifth of the

TABLE I—*Characteristics of 49 018 men and women aged 30-54 years at baseline. Figures are means (SD)*

Variable	Men (n=23 389*)	Women (n=25 629*)
Age (years)	39·2 (4·3)	39·4 (4·4)
Total cholesterol (mmol/l)	5·55 (1·10)	5·19 (1·00)
Systolic blood pressure (mmHg)	132·8 (16·2)	126·7 (17·6)
Diastolic blood pressure (mmHg)	81·1 (11·2)	77·8 (11·0)
Body mass index (kg/m^2)	24·9 (3·0)	24·3 (3·7)
Smoking (%)	65·6 (47·5)	47·0 (49·9)

*Maximum number of valid observations.

distribution ranged from 4·18 mmol/l in the lowest to 7·14 mmol/l in the highest fifth in men and from 3·94 mmol/l to 6·69 mmol/l in women (table III).

For coronary heart disease, mortality in men was five times that in women. The highest compared with the lowest fifth showed a threefold higher risk in men

and an almost fourfold higher risk in women after adjustment for age, smoking, blood pressure, and body mass index (table IV). For cardiovascular disease the highest compared with the lowest fifth showed an almost threefold higher risk in both men and women.

In both men and women no associations between

TABLE II—*Main causes of death in 49 018 men and women aged 30-54 years at baseline after on average 12 years of follow up*

Mortality	ICD-9 codes	No (%) of men	No (%) of women
Cardiovascular diseases:	390-459	290 (35·5)	92 (18·4)
Coronary heart disease	410-414	194 (23·7)	38 (7·6)
Cerebrovascular disease	430-439	38 (4·6)	35 (7·0)
Other cardiovascular diseases	390-409, 415-429, 440-459	58 (7·1)	19 (3·8)
Cancer:	140-239	276 (33·7)	292 (58·3)
Digestive	150-159	66 (8·1)	66 (13·2)
Respiratory*	160-169	116 (14·2)	27 (5·4)
Breast	174		95 (19·0)
Other cancers	140-149, 170-173, 175-239	94 (11·5)	104 (20·8)
Non-cardiovascular and non-cancer:	0-139, 240-389, 460-999	232 (28·4)	107 (21·4)
Respiratory diseases	460-519	15 (1·8)	10 (2·0)
Digestive diseases	520-579	32 (3·9)	7 (1·4)
Accidents†	800-999	112 (13·7)	59 (11·8)
Other non-cardiovascular/non-cancer	0-139, 240-389, 580-799	73 (8·9)	31 (6·2)
Unknown		20 (2·4)	10 (2·0)
Total		818	501

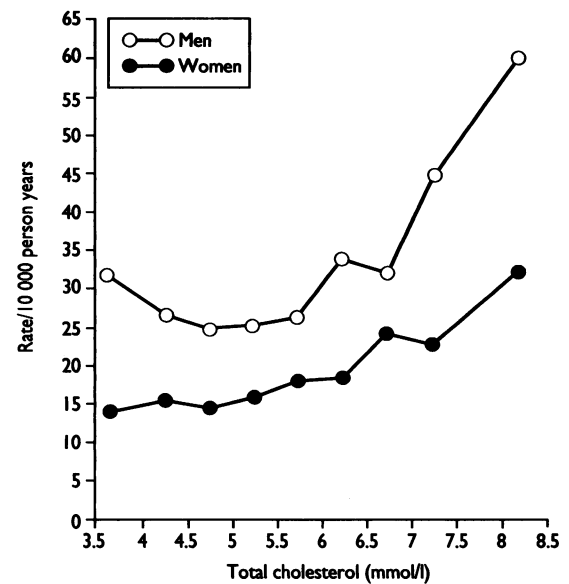*Predominantly lung cancer: 108 men and 26 women.　†Including suicide: 54 men and 30 women.

TABLE III—*Mean (SD) age (years) and cholesterol concentration (mmol/l) by fifth of cholesterol distribution and sex*

	Men				Women			
			Total cholesterol (mmol/l)				Total cholesterol (mmol/l)	
Fifth	No	Age	Range	Mean (SD)	No	Age	Range	Mean (SD)
1	4908	38·1 (4·4)	≤4·6	4·18 (0·40)	5187	38·0 (4·0)	≤4·3	3·94 (0·35)
2	4117	38·9 (4·2)	4·7-5·1	4·92 (0·14)	5166	38·9 (4·0)	4·4-4·8	4·63 (0·14)
3	5409	39·4 (4·3)	5·2-5·7	5·48 (0·17)	5404	39·3 (4·2)	4·9-5·3	5·12 (0·14)
4	4063	39·7 (4·1)	5·8-6·3	6·06 (0·17)	4827	40·0 (4·3)	5·4-5·9	5·65 (0·17)
5	4875	40·1 (4·0)	≥6·4	7·14 (0·76)	4993	40·9 (4·8)	≥6·0	6·69 (0·73)
Total	27 372	39·2 (4·3)	2·1-17·8	5·55 (1·10)	25 577	39·4 (4·4)	1·91-16·3	5·19 (1·00)

Mortality (per 10 000 person years) for all cause mortality according to category of serum total cholesterol concentration in men and women aged 30-54 years after (on average) 12 years of follow up, adjusted for age, smoking, blood pressure, and body mass index

fifths of the cholesterol distribution and mortality from cancer or non-cardiovascular or non-cancer causes were observed (table IV). Also no associations with site specific cancers nor with subgroups of non-cardiovascular or non-cancer causes mentioned in table II were observed (results not shown).

All cause mortality was significantly higher in the highest fifth of the cholesterol distribution by about 60% in men and 50% in women (table IV). To explore the shape of the relation between total cholesterol concentration and all cause mortality in more detail cholesterol concentration was divided into nine categories with 0·5 mmol/l steps (figure). The middle category (5·5-5·99 mmol/l) was used as the reference

TABLE IV—*Relative risks (9·5% confidence interval) for mortality from coronary heart disease, total cardiovascular disease, cancer, non-cardiovascular disease and non-cancer, and all causes according to fifths of distribution of serum total cholesterol concentration in men and women*

Disease and fifth of distribution	Men			Women		
	Crude mortality per 10 000 person years	Relative risk*	Relative risk† (95% confidence interval)	Crude mortality per 10 000 person years	Relative risk*	Relative risk† (95% confidence interval)
Coronary heart disease:						
1	2·77	1·0	1·0	0·48	1·0	1·0
2	3·30	1·14	0·93 (0·47 to 1·84)	0·64	1·34	1·16 (0·26 to 5·21)
3	3·77	1·29	0·94 (0·50 to 1·75)	0·62	1·28	1·00 (0·22 to 4·48)
4	9·84	3·37	2·29 (1·31 to 4·01)	1·20	2·50	1·65 (0·42 to 6·45)
5	15·64	5·32	3·03 (1·79 to 5·14)	3·32	6·86	3·80 (1·10 to 13·10)
P value for linear trend		<0·001	<0·001		<0·001	0·004
Cardiovascular diseases:						
1	4·85	1·0	1·0	1·45	1·0	1·0
2	5·57	1·12	0·93 (0·55 to 1·58)	2·09	1·45	1·30 (0·56 to 3·05)
3	6·91	1·39	1·05 (0·65 to 1·68)	2·15	1·50	1·23 (0·53 to 2·85)
4	12·35	2·48	1·76 (1·13 to 2·76)	2·75	1·91	1·38 (0·61 to 3·16)
5	22·77	4·53	2·77 (1·84 to 4·18)	6·65	4·60	2·85 (1·36 to 5·99)
P value for linear trend		<0·001	<0·001		<0·001	0·001
Cancer:						
1	8·83	1·0	1·0	8·05	1·0	1·0
2	10·52	1·20	1·14 (0·78 to 1·69)	8·68	1·06	1·00 (0·68 to 1·47)
3	7·86	0·89	0·82 (0·52 to 1·22)	7·38	0·91	0·82 (0·55 to 1·21)
4	10·89	1·24	1·11 (0·75 to 1·65)	11·86	1·45	1·24 (0·86 to 1·79)
5	12·51	1·41	1·24 (0·86 to 1·80)	11·63	1·42	1·11 (0·77 to 1·62)
P value for linear trend		0·069	0·293		0·011	0·275
Non-cardiovascular and non-cancer:						
1	7·62	1·0	1·0	2·74	1·0	1·0
2	6·39	0·84	0·80 (0·50 to 1·26)	2·73	1·01	0·96 (0·49 to 1·88)
3	8·01	1·08	0·98 (0·65 to 1·47)	3·54	1·30	1·21 (0·64 to 2·27)
4	9·42	1·25	1·09 (0·72 to 1·67)	2·92	1·08	0·95 (0·48 to 1·88)
5	10·43	1·38	1·15 (0·72 to 1·72)	5·48	2·02	1·67 (0·91 to 3·06)
P value for linear trend		0·029	0·228		0·018	0·102
All causes:						
1	21·30	1·0	1·0	12·24	1·0	1·0
2	22·48	1·06	0·95 (0·74 to 1·23)	13·50	1·10	1·01 (0·75 to 1·37)
3	22·94	1·08	0·90 (0·71 to 1·15)	13·07	1·06	0·93 (0·69 to 1·27)
4	32·66	1·53	1·24 (0·98 to 1·56)	17·53	1·42	1·17 (0·87 to 1·57)
5	45·71	2·13	1·60 (1·29 to 1·99)	23·77	1·92	1·46 (1·10 to 1·94)
P value for linear trend		<0·001	0<0·001		<0·001	0·003

*Unadjusted.　†Adjusted for age, smoking, systolic blood pressure, and body mass index.

TABLE V—*All cause mortality according to cholesterol categories of 0·5 mmol/l width in men and women: mean total cholesterol, number of subjects, adjusted mortality, adjusted relative risks (95% confidence interval) per category; 5·5 to 5·99 mmol/l is used as reference category*

Cholesterol category	Range (mmol/l)	Men			Women		
		No	Rate*	Relative risk† (95% confidence interval)	No	Rate*	Relative risk† (95% confidence interval)
1	<4·0	1269	31·14	1·18 (0·81 to 1·71)	2311	14·09	0·78 (0·52 to 1·17)
2	4·0-4·49	2288	26·39	1·00 (0·73 to 1·6)	3812	15·54	0·86 (0·62 to 1·20)
3	4·5-4·9	3812	24·81	0·94 (0·73 to 1·23)	5388	14·64	0·81 (0·60 to 1·10)
4	5·0-5·49	4380	25·33	0·96 (0·75 to 1·23)	5174	15·90	0·88 (0·65 to 1·18)
5	5·5-5·99	4216	26·39	1·00	3899	18·07	1·00
6	6·0-6·49	3061	34·00	1·29 (1·01 to 1·65)	2473	18·61	1·03 (0·73 to 1·44)
7	6·5-6·99	2054	32·20	1·22 (0·93 to 1·61)	1327	24·39	1·35 (0·94 to 1·95)
8	7·0-7·49	1133	44·34	1·68 (1·25 to 2·25)	632	23·13	1·28 (0·78 to 2·08)
9	≥7·5	1159	60·43	2·29 (1·76 to 2·97)	561	32·35	1·79 (1·16 to 2·77)
Total		23 372	29·69		25 577	16·25	

*Mortality per 10 000 person years adjusted for age, smoking, blood pressure, and body mass index.
†Relative risk adjusted for age, smoking, systolic blood pressure, and body mass index.

category. A total cholesterol concentration of 7·5 mmol/l or more gave a relative risk of 2·3 (95% confidence interval 1·8 to 3·0) in men and a relative risk of 1·8 (1·2 to 2·8) in women (table V). Risk increased continuously with increasing cholesterol concentration in women. In men the lowest all cause mortality was observed in the category 4·5-4·99 mmol/l. The relative risk for a total cholesterol concentration below 4·0 mmol/l compared with a concentration of 5·5-5·99 mmol/l in men was 1·18 (0·81 to 1·71).

Discussion

During the follow up period only 38 women died of coronary heart disease compared with 194 men, showing that in middle age coronary heart disease is already a prominent cause of death in men but not yet in women. The association between total cholesterol concentration and mortality from coronary heart disease was comparable in men and women: risk was higher above the fourth quintiles, although in women risk above the fifth quintile only was significant. The relative risk for the highest compared with the lowest fifth was 3·0 in men and 3·8 in women. A slightly higher relative risk for women compared with men has been observed in other studies as well.[1][13] In all studies, however, absolute risk was lower in women than in men.

In the present study a single measurement of total cholesterol concentration was obtained, leading to underestimation of the relative risk, a phenomenon often referred to as regression dilution bias.[14] Law et al estimated the magnitude of the dilution factor to be 1·4.[15] When we apply this dilution factor to the relative risk for the highest fifth in the present study the observed ("diluted") relative risk of 3·03 for men would correspond to an undiluted relative risk of 4·7 (3·03[1·4]), and the relative risk for the highest fifth in women would increase from 3·8 to 6·5.

The relative risks for coronary heart disease in women in the present study were high compared with those reported in other studies, even after adjustment for age, smoking, blood pressure, and body mass index.[1][13] This might be due to the fact that the respondents were relatively young at the time of examination: three quarters were aged 35-45 years. Several studies have shown that the relative risk is higher the younger the age at examination.[16-18] Law et al estimated that after 13 years of follow up a 0·6 mmol/l decrease in total cholesterol concentration was associated with a 31% decrease in mortality from coronary heart disease in men aged 35-44 at baseline, whereas in men aged 55-64 this decrease was only 12%.[15]

The Dutch cholesterol consensus applies the same cut off point for hypercholesterolaemia (total choles-

terol ≥6·5 mmol/l) to men and women.[10] Because concentrations of high density lipoprotein cholesterol are on average 0·25 mmol/l higher in women than in men,[19] this cut off point is in fact more strict for women. In the present study the risk of coronary heart disease in women with cholesterol concentrations above 5·9 mmol/l was almost four times that in women with concentrations below 4·3 mmol/l. Therefore, the consensus criteria seem justified. As absolute risk is much lower in women compared with men, however, the decision to treat hypercholesterolaemia in women has to take the absolute risk (and therefore also other risk factors for coronary heart disease besides total cholesterol) into account.

Recently, concern has been raised about an increased risk of mortality at the lower end of the cholesterol distribution.[7] Several studies have reported an increased risk for non-cardiovascular mortality at a low cholesterol concentration or even inverse associations over the entire cholesterol range.[8][20-25] In many studies these associations remained after exclusion of the first few years of follow up, contradicting the hypothesis that the associations were due to pre-existing diseases at baseline.[8][20][23-25] In most studies in which these observations were found study subjects were middle aged men. It has been suggested that in "healthy" cohorts consisting of working people[27] or relatively young people,[27] these associations are not present. In the present study of relatively young people no association was observed between total cholesterol concentration and mortality from cancer and non-cardiovascular or non-cancer causes. Also for site specific cancers (digestive, respiratory, and breast) and for subgroups of non-cardiovascular or non-cancer mortality (respiratory diseases, digestive diseases, and accidents) no association with total cholesterol concentration was observed. Our data thus support the healthy cohort hypothesis.

In men and women all cause mortality was increased by about 60% and 50%, respectively, in the highest compared with the lowest fifth of the cholesterol distribution. To examine the possibility of an increase in all cause mortality at the lower end of the cholesterol distribution in more detail smaller categories than fifths were used. In women all cause mortality increased continuously with increasing cholesterol concentration. In men all cause mortality was lowest at a cholesterol concentration of 4·5-4·9 mmol/l, but the difference with mortality at cholesterol concentrations below 4·5 mmol/l was not significant. In the multiple risk factor intervention trial all cause mortality at cholesterol concentrations below 4·1 mmol/l was increased in men.[8] In a recent meta-analysis all cause mortality was increased at low cholesterol concentrations in men but not in women.[7] In the national

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Key messages

● Total cholesterol concentration in women was as strongly related to mortality from coronary heart disease as in men

● Although relative risks for mortality from coronary heart disease were similar in men and women, the absolute risk of death in women was about one fifth that in men

● No significant excess mortality from non-cardiovascular causes was observed at low cholesterol concentrations

● Total mortality in the highest fifth of the cholesterol distribution was 60% higher in men and 50% higher in women compared with the lowest fifth

health and nutrition examination survey (NHANES I) epidemiological follow up study all cause mortality at low cholesterol concentrations was increased in both men and women, but this increase was significant only in those aged over 60 at baseline.[27] Our results support the hypothesis that the relation between total cholesterol concentration and mortality is dependent on age at baseline.

The present study shows that total cholesterol concentration is a strong risk factor for coronary heart disease and all cause mortality in men as well as in women. No significant increase in all cause mortality at low concentrations was observed. Our results add to the evidence that increased non-cardiovascular mortality at low cholesterol concentrations is limited to subjects who are middle aged or older at baseline.

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Waiting list dynamics and the impact of earmarked funding

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Abstract

Objective—To determine how changes in the number of admissions from waiting lists and changes in the number of additions to the lists are related to list size and waiting times, in the context of local waiting list initiatives.

Design—Review of national and Körner statistics.

Setting—England (1987-94) and districts of the former Oxford region (1987-91).

Main outcome measures—Correlation of quarterly changes in the number of admissions from waiting lists in England with changes in total list size, numbers of patients waiting one to two, or over two years, and number of additions to the lists; examination of changes in waiting list statistics for individual district specialties in one region in relation to funding for waiting list initiatives.

Results—Nationally, changes in the number of admissions to hospital from lists closely correlated with changes in the number of additions to lists ($r = 0.84$; $P < 0.01$). After adjusting for changes in the number of additions to lists, changes in the number of admissions correlated inversely with changes in list size ($r = -0.62$; $P < 0.001$). Decreases in the number of patients waiting from one to two years were significantly associated with increases in the number of admissions ($r = -0.52$; $P < 0.01$); locally, only six of 44 waiting list initiatives were followed by an increase in admissions and a fall in list size, although a further 11 were followed by a fall in list size without a corresponding increase in admissions.

Conclusions—An increase in admissions improved waiting times but did not reduce list size because additions to the list tended to increase at the same time. The appropriateness of waiting list initiatives as a method of funding elective surgery should be reviewed.

Introduction

The number of people on hospital waiting lists, and the length of time that they wait, are used extensively as performance indicators in the NHS.[1] Although there are still over a million people on waiting lists in England, the number waiting over a year has decreased steadily since 1990. Policy initiatives to reduce waiting times include the patient's charter,[2] earmarked funds of about £30m a year nationally from 1987 to 1993, and the funding of 100 new consultant posts in 1990 specifically to reduce waiting times.