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Serum cholesterol concentration and coronary heart disease in population with low cholesterol concentrations

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Abstract

***Objective*—To examine the relation between serum cholesterol concentration and mortality (from coronary heart disease and from other causes) below the range of cholesterol values generally seen in Western populations.**

***Design*—Prospective observational study based on 8-13 years of follow up of subjects in a population with low cholesterol concentrations.**

***Setting*—Urban Shanghai, China.**

***Subjects*—9021 Chinese men and women aged 35-64 at baseline.**

***Main outcome measure*—Death from coronary heart disease and other causes.**

***Results*—The average serum cholesterol concentration was 4·2 mmol/l at baseline examination, and only 43 (7%) of the deaths that occurred during 8-13 years of follow up were attributed to coronary heart disease. There was a strongly positive, and apparently independent, relation between serum cholesterol concentration and death from coronary heart disease ($z=3·47$, $p<0·001$), and within the range of usual serum cholesterol concentration studied (3·8-4·7 mmol/l) there was no evidence of any threshold. After appropriate adjustment for the regression dilution bias, a 4 (SD 1)% difference in usual cholesterol concentration was associated with a 21 (SD 6)% (95% confidence interval 9% to 35%) difference in mortality from coronary heart disease. There was no significant relation between serum cholesterol concentration and death from stroke or all types of cancer. The 79 deaths due to liver cancer or other chronic liver disease were inversely related to cholesterol concentration at baseline.**

***Conclusion*—Blood cholesterol concentration was directly related to mortality from coronary heart disease even in those with what was, by Western standards, a "low" cholesterol concentration. There was no good evidence of an adverse effect of cholesterol on other causes of death.**

Introduction

In populations in which the mean serum cholesterol concentration is relatively high (such as those in Europe and North America) prospective observational studies indicate a strong, direct association between serum cholesterol concentration and coronary heart disease.[1-6] Questions remain, however, about the relation at lower concentrations of cholesterol.

Indeed, some have suggested there may even be a "threshold"—sometimes hypothesised to be at, or about, 5·2 mmol/l—below which lower serum cholesterol concentrations are not associated with lower risks of coronary heart disease.[7,8] In view of the possibility in Western populations of lowering the cholesterol concentrations of some individuals into this range by dietary changes (or, in individuals at particularly high risk of coronary heart disease, by drugs) reliable information is needed about the shape of the relation at lower concentrations of cholesterol. But direct investigation of the relation at concentrations below 5·2 mmol/l is difficult in Western populations, in which the proportions of people who really have such cholesterol concentrations after repeated measurements are relatively small. It can, however, be studied directly in populations where most people have low cholesterol concentrations. To help address this question we conducted a prospective study among more than 9000 middle aged adults in urban Shanghai, where the mean cholesterol concentration is much lower than that usually observed in Western populations. This study also provided an opportunity to determine whether in this low range there was any inverse association between blood cholesterol concentration and deaths from various non-coronary causes.

Subjects and methods

The study population comprised two cohorts (A and B) of middle aged factory workers from urban Shanghai. Details of the methods used to recruit subjects and of the baseline survey have been reported.[9] Health screening clinics were set up in 11 factories (three for cohort A and eight for cohort B), to which all staff aged 35-64 years were invited. In cohort A in 1972-3, 1635 men and 1288 women (2923 people, 95% of those invited) were screened,[9] and in cohort B in 1977-8, 4859 men and 1569 women (6428 people, 93% of those invited) were screened. At the time of screening data were recorded on several relevant variables (including systolic and diastolic blood pressure, cigarette smoking, and alcohol consumption) and blood was taken for measurement of serum cholesterol concentration. For the present study 330 subjects were excluded either because their baseline cholesterol measurement was missing (30 in cohort A and 298 in cohort B) or because there was definite evidence of a previous myocardial infarction on the electrocardiogram recorded at baseline (none in cohort A, and two in cohort B). Evidence of left ventricular hypertrophy or of other electrocardiographic abnormalities was not a reason for exclusion.

ESTIMATION OF SERUM CHOLESTEROL CONCENTRATION

In both cohorts venous blood was drawn after overnight fasting into a 20 ml tube without anticoagulant and taken daily to a central laboratory where the tubes were centrifuged for separation of serum. All blood samples were assayed within two days of collection. In cohort A cholesterol measurements were performed in the lipid laboratory of the Cardiovascular Disease Institute of Shanghai by the method of Zak *et al.*[10] This method produced results that were correct and reasonably reproducible which could be used to divide cohort A into quarters of baseline cholesterol concentration (table I). In cohort B, however, cholesterol measurements were performed in the lipid laboratory of the Shanghai Chest Hospital by the Libermann-Burchard method.[11] This produced results that in absolute terms were too high (see below) but that were also reasonably reproducible and therefore could also be used to divide cohort B into quarters of baseline cholesterol concentration. Lipoprotein fractions were not measured and the samples were not retained.

Internationally standardised quality control comparisons were not available in Shanghai in the 1970s, but in both cohorts substantial efforts were made to ensure the consistency of the cholesterol measurements, and satisfactory intralaboratory quality control was confirmed by blinded remeasurements of selected samples. Interlaboratory quality control, however, with direct comparisons of cholesterol measurements on blood samples exchanged among four laboratories during the follow up study of cohort A, indicated that cholesterol values obtained in the laboratory used for cohort B were an average of about 0·78 mmol/l higher than those obtained in the laboratory used for cohort A and in the other two laboratories involved. Hence the absolute cholesterol values recorded at baseline were 4·2 mmol/l in cohort A and 5·0 mmol/l in cohort B. Internationally validated studies (such as the World Health Organisation MONICA project) on other urban Chinese populations indicate a mean cholesterol concentration of about 4·1 mmol/l for adults in Chinese cities.[12] By the mid-1980s both laboratories had changed their methods of cholesterol measurement to that recommended and standardised by the WHO MONICA project.[12] Subsequent quality control checks indicated good agreement between this new method and the previous method used in the cohort A laboratory, but confirmed that the method used previously in the laboratory of cohort B had been producing results that were about 0·52-0·78 mmol/l too high.[13] The baseline cholesterol measurements in cohort B could still, however, be used to subdivide subjects reliably into quarters of baseline cholesterol concentration for follow up of mortality.

Cholesterol concentrations were remeasured three years after baseline in 2329 (82%) of the subjects in cohort A and 1805 subjects from four factories (68%) in cohort B. Cholesterol was measured by the same method as that used for the baseline sample in that cohort. Comparison of the original and the remeasured serum cholesterol concentrations can help assess the strength of the association between "usual" and baseline cholesterol concentrations. By combining data on this association with data on the association between baseline cholesterol concentrations and the subsequent risk of disease it is possible to estimate the real association between usual cholesterol concentration and the subsequent risk of disease.[14]

FOLLOW UP OF SUBJECTS

Vital state was determined annually through the records of the factory medical room, the workers' union, and the salary department of the factory until 1 January 1987. Only 0·71% (64 people) of the two cohorts was lost to mortality follow up. Causes of death were sought from official death certificates, supplemented by hospital records and by inquiry of physicians and family members or medical staff in each factory. The underlying cause of each death was coded by two trained nosologists in the Shanghai Sanitary and Anti-Epidemic Station, who used the ninth revision of the International Classification of Diseases (ICD).

STATISTICAL ANALYSIS

Baseline cholesterol concentration

To obtain more reliable information about the association between baseline serum cholesterol concentration and cause specific mortality, results from the two cohorts were combined. Two statistical methods were used to analyse the combined data, both of which avoided direct comparison of cholesterol values in one cohort with those in the other.

Firstly the log rank method was used to calculate observed and expected numbers of deaths with respect to four quarters of cholesterol concentration in each separate cohort.[15] For each separate quarter (I, II, III, IV) the observed and expected numbers of deaths in each cohort were then added together to yield an overall observed and an overall expected number for that particular quarter. Overall trends in relative risk (described approximately by the ratio of observed: expected deaths) were tested for significance with the log rank trend test,[15] by using the scores 1-4 for the quarters I-IV of serum cholesterol concentration. This approach avoided any assumptions about comparability of the cohorts or about the exact shape of the relation between cholesterol concentration and disease risks.

To evaluate the independent effect of the baseline cholesterol measurement on disease risks, Cox's proportional hazards model was then used with a linear term for serum cholesterol concentration adjusting simultaneously for other variables (age, sex, diastolic blood pressure, cigarette smoking, and alcohol drinking).[16] For the overall results of the two cohorts combined, the Cox regression analysis was stratified for which cohort (A or B) subjects were in. The estimated log odds ratio corresponding to a 10% difference in cholesterol (0·41 mmol/l, which is about 10% of the mean of the whole sample) and its 95% confidence interval was obtained through multiplying the Cox regression coefficient and its 95% confidence limits by 0·41.

Usual cholesterol concentration

The baseline cholesterol concentration, which is based on a single measurement at entry to the study, is subject to random error due partly to the laboratory process and partly to any real but temporary deviation from an individual's long term usual cholesterol concentration.[17-19] Such random errors will result in systematic underestimation of the slope of the real association between usual cholesterol concentration and disease. This is referred to as the "regression dilution" bias as the degree of underestimation is directly related to the extent to which the cholesterol measurements are subject to the phenomenon of regression to the mean.[19] Again, the regression dilution bias can be corrected for and the true association estimated in two main ways.[14]

In the first, the disease rates for categories defined by their baseline cholesterol concentration are plotted not against the mean baseline cholesterol concentration in each category but against an unbiased estimate of the mean usual cholesterol concentration in each category. Cholesterol concentrations were remeasured a few years after the baseline examination in a substantial proportion of the subjects in both cohorts, and these could be used to correct the cholesterol-disease associations. Table I presents the average of cholesterol concentrations at baseline and at three years in cohort A for individuals divided into four categories according to their baseline cholesterol concentration alone (that is, irrespective of the values of any subsequent remeasurements). For the individuals in each category of baseline cholesterol concentration, average values are given for the original baseline cholesterol measurement and for the cholesterol measurements after three years. The means of the baseline cholesterol measurements are seriously biased estimates of the mean usual cholesterol concentration in each category (and the biases are particularly large in the top and bottom categories) whereas the means of the remeasured cholesterol values provide estimates that are substantially less subject to such bias. These data indicate that the differences in mean cholesterol concentration between the top and bottom categories are about twice as great for baseline cholesterol concentration as for cholesterol concentrations remeasured three years later. (Hence when the four relative risks of death from coronary heart disease are appropriately plotted against the estimates of mean usual cholesterol concentration in each of the four categories, the slope of the cholesterol-disease association will be at least twice as steep as when the relative risks are inappropriately plotted against the mean baseline cholesterol values in each category.)

TABLE I—*Mean cholesterol concentrations at baseline and at three years after baseline in cohort A for each of four quarter groups defined only by baseline cholesterol concentration*

	Serum cholesterol at baseline (mmol/l)	No of subjects	Mean cholesterol at baseline (mmol/l)	Mean cholesterol after 3 years (mmol/l)
I	≤3·53	586	3·08	3·83
II	3·54-4·10	588	3·81	4·06
III	4·11-4·62	583	4·34	4·22
IV	≥4·63	635	5·40	4·63
All groups*		2392	4·18	4·19
Range of mean cholesterol			2·32	0·80

*The standard deviations of serum cholesterol concentration were 0·93 mmol/l and 0·91 mmol/l baseline and at three years after baseline, respectively.

In cohort B (by an assay that produced results that were about 0·78 mmol/l too high), the mean cholesterol concentration was 4·97 mmol/l at baseline and 5·31 mmol/l at three years after baseline, while the range of mean cholesterol concentration between the highest and lowest quarters was 2·12 mmol/l at baseline and 0·88 mmol/l at three years after baseline.

The second way of estimating the true association involves a "parametric" correction method. The association between baseline cholesterol measurements and the usual cholesterol concentration suggested by table I is not exactly straight, so the "parametric" correction methods may not be exactly appropriate. It is, however, approximately straight, suggesting that the effects on disease risk of any particular difference in usual cholesterol concentration can be estimated as the effects of a difference at least twice as great as this in baseline cholesterol concentration.

Results

Among the 9021 subjects in the two cohorts, 595 deaths were recorded during the 8-13 years of follow up. Of the deaths, 220 (37%) were ascribed to cardiovascular disease (ICD 390-459), including 43 (7%) from coronary heart disease (ICD 410-414) and 146 (25%) from stroke (ICD 430-438). A further 263 (44%) deaths were attributed to cancer (ICD 140-239), including 63 (11%) from lung cancer (ICD 162), 62 (10%) from stomach cancer (ICD 151), and 51 (9%) from primary liver cancer (ICD 155). Other causes accounted for 112 deaths (19%), 28 (5%) of which were classified as chronic hepatitis or cirrhosis (ICD 571).

Table II shows the relation of baseline serum cholesterol concentration with certain other baseline variables for the combined data from the two cohorts. Subjects with higher serum cholesterol concentrations were slightly older and had higher blood pressure (both systolic and diastolic) than those with lower cholesterol concentrations. Smoking was less prevalent and alcohol consumption slightly more prevalent among subjects with higher cholesterol concentrations compared with those with lower concentrations.

ASSOCIATION OF CHOLESTEROL CONCENTRATION WITH CORONARY HEART DISEASE

Table III gives the numbers of deaths from coronary heart disease and various other causes, along with the estimated relative risks in the four quarters of baseline cholesterol concentration. The statistical test results reported are for both the log rank and the Cox regression methods. There was a positive relation

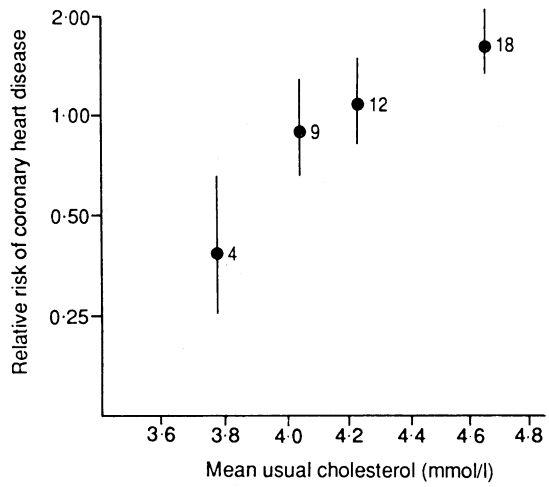
TABLE II—*Mean values of baseline variables for each cholesterol concentration quarter (cohort A and B combined)*

Variables*	Quarters of baseline serum cholesterol† I (lowest) (n=2162)	II (n=2285)	III (n=2405)	IV (highest) (n=2169)
Age (years)	47·7	48·0	48·7	49·3
Male (%)	70·1	67·7	69·5	68·6
Systolic blood pressure (mm Hg)	122·8	124·1	125·2	127·5
Diastolic blood pressure (mm Hg)	77·5	78·4	79·0	81·1
Cigarette smoking (%)	46·0	44·4	43·6	42·1
Alcohol drinking (%)	19·8	19·1	19·4	20·9

*All variables have been adjusted for age. For blood pressure, the weighted mean was computed by using the inverse of the variances as weights in each cohort. For smoking and alcohol drinking, age adjusted means were calculated by direct standardisation to the age distribution of the two cohorts combined.
†The cutoffs between groups I, II, III, and IV were 85%, 99%, and 111% of the cohort A mean, and 89%, 99%, and 111% of the cohort B mean.

between baseline cholesterol concentration and the risk of fatal coronary heart disease. The approximate risk of coronary heart disease death in the quarter with the lowest concentration (I) relative to the whole study population was 0·4, while for the people in the highest quarter (IV) it was 1·6. This trend of increasing risk of death from coronary heart disease with increasing cholesterol concentration was significant ($p<0·01$) in a log rank trend test that did not take other variables into account. In the adjusted Cox regression analysis baseline serum cholesterol concentration was an even more significant predictor of death from coronary heart disease ($p<0·001$) after taking other variables into account. The estimated regression coefficient (0·463) suggests that a 10% difference in baseline serum cholesterol concentration (that is, a difference of 0·41 mmol/l) would be independently associated with a log odds ratio of 0·190 and hence 21% excess risk of death from coronary heart disease (that is, odds ratio 1·21; 95% confidence interval 1·09 to 1·35). The relation between serum cholesterol concentration and coronary heart disease was separately significant in cohort A and in cohort B and was separately significant in men and in women. In cohort A, with 27 deaths from coronary heart disease during 13 years of follow up, the regression coefficient for baseline cholesterol concentration was 0·401 ($z=2·49$, $p<0·05$), while in cohort B, with 16 deaths from coronary heart disease during eight years of follow up, it was 0·560 ($z=2·37$, $p<0·05$). Among men, 32 of whom died of coronary heart disease, the regression coefficient for baseline cholesterol was 0·405 ($z=2·37$, $p<0·05$), while among women, 11 of whom died of coronary heart disease, it was 0·471 ($z=2·09$, $p<0·05$).

As discussed previously, the means of the baseline cholesterol values are biased estimates of the mean usual cholesterol concentrations in each quarter group. The means of the remeasured cholesterol values in cohort A (table I) provide estimates that are substantially less subject to such bias.[14] The data in table I suggest that a 10% difference in baseline cholesterol concentration may correspond to only about a 4 (SD 1)% difference in usual cholesterol concentration. The figure plots the relative risk of coronary heart disease, on a doubling scale, against the estimated mean usual cholesterol concentration in each of the four baseline cholesterol groups. If the slope of the association between the relative risk of coronary heart disease and the mean usual cholesterol concentration, estimated from remeasurements in cohort A, was roughly constant this would imply an approximately log linear relation (that is, one in which the percentage difference in risk associated with a given difference in usual cholesterol concentration is similar at all concentrations of cholesterol). Within the range of serum cholesterol concentration studied in this population (about 3·8-4·7 mmol/l) there was no good evidence of a threshold level of cholesterol concentration below which lower cholesterol concentration was no longer associated with a lower risk of coronary heart disease. Combination of the results in tables I and III suggests that a difference of about 4 (SD 1)% in usual cholesterol concentration might be associated with about a 21 (SD 6)% (95% confidence interval 9% to 35%) difference in the risk of death from coronary heart disease.



Relative risk of death from coronary heart disease by quarters of baseline cholesterol in 9021 Chinese people with 8-13 years' follow up. Combined results from cohort A and B. Estimates of the mean usual cholesterol are from mean values three years after baseline in cohort A. Numbers of deaths from coronary heart disease in each group are shown with vertical lines that represent one standard deviation

TABLE III—*Numbers of deaths from vascular diseases, cancers, and other diseases during 8-13 years of follow up, with adjusted relative risk estimates in the quarter groups of baseline serum cholesterol concentration*

Cause of death	No of deaths (observed/expected deaths†) I (n=2162)	II (n=2285)	III (n=2405)	IV (n=2169)	All subjects (n=9021)	Statistical test for association with cholesterol χ^2 For trend	Regression coefficient‡ (mmol/l)	z Value
Vascular disease:								
Coronary heart disease	4 (0·38)	9 (0·88)	12 (1·07)	18 (1·63)	43 (1·00)	8·35**	0·463	3·47***
Stroke	34 (1·01)	35 (1·00)	34 (0·87)	43 (1·11)	146 (1·00)	0·06	−0·015	−0·17
Other	7 (1·00)	8 (1·13)	9 (1·07)	7 (0·83)	31 (1·00)	0·13	0·023	0·12
All vascular	45 (0·89)	52 (1·00)	55 (0·93)	68 (1·17)	220 (1·00)	1·44	0·097	1·40
Cancer:								
Lung	17 (1·19)	12 (0·79)	12 (0·71)	22 (1·31)	63 (1·00)	0·11	0·054	0·39
Stomach	16 (1·09)	15 (0·99)	15 (0·91)	16 (1·02)	62 (1·00)	0·06	0·008	0·05
Liver	19 (1·59)	11 (0·87)	13 (0·97)	8 (0·61)	51 (1·00)	4·86*	−0·378	−2·18*
Other	22 (1·13)	18 (0·86)	27 (1·16)	20 (0·85)	87 (1·00)	0·28	−0·031	−0·27
All cancer	74 (1·22)	56 (0·88)	67 (0·96)	66 (0·96)	263 (1·00)	1·53	−0·062	−0·89
Other causes:								
Chronic hepatitis and cirrhosis	11 (1·64)	7 (1·03)	7 (0·93)	3 (0·43)	28 (1·00)	4·74*	−0·486	−1·97*
Non-medical	6 (1·55)	6 (1·44)	4 (0·85)	1 (0·23)	17 (1·00)	4·30*	−0·602	−1·94
Other disease	15 (0·95)	23 (1·47)	16 (0·89)	13 (0·74)	67 (1·00)	1·33	−0·127	−0·95
All non-vascular non-cancer deaths	32 (1·22)	36 (1·35)	27 (0·89)	17 (0·59)	112 (1·00)	7·72**	−0·274	−2·45*
All causes	151 (1·10)	144 (1·01)	149 (0·94)	151 (0·97)	595 (1·00)	1·67	−0·027	−0·58

*$p<0·05$, **$p<0·01$, and ***$p<0·001$.
†Ratios of observed number of deaths to expected number of deaths are adjusted for age, sex, and cohort by the log rank method.[15]
‡Estimated in a Cox's proportional hazards model with a linear term for serum cholesterol concentration and covariates corresponding to age (years), sex, diastolic blood pressure (mm Hg), cigarette smoking (no *v* yes), and alcohol drinking (no *v* yes).

ASSOCIATION WITH STROKE

The mortality from stroke was high in this Chinese population (25% of total deaths). A positive and highly significant association was seen between blood

pressure (both systolic and diastolic) and subsequent death from stroke (data not shown), but there was no apparent association between serum cholesterol concentration and death from stroke, even among subjects with a raised blood pressure (that is, diastolic blood pressure ≥90 mm Hg) at baseline.[20]

ASSOCIATION WITH NON-VASCULAR DISEASES

Overall, no significant association was evident between baseline cholesterol concentration and total mortality from cancer. After subdivision with respect to cancer site there was still no suggestion of any association with cholesterol concentration for lung or stomach cancer, but there was an inverse trend for liver cancer. The approximate relative risk of liver cancer in the lowest quarter of cholesterol concentration was 1·6, while for the top quarter it was 0·6. This inverse association between cholesterol concentration and death from liver cancer was significant ($p<0·05$) even after exclusion of those subjects who died in the first three years after the baseline examination. For death from liver cancer four or more years after the baseline survey the Cox regression coefficient was $-0·556$ ($z=2·67$, $p<0·01$), and the baseline serum cholesterol concentration among the 37 people who died was 0·39 mmol/l lower than that among survivors.

In view of the inverse association observed between liver cancer and cholesterol concentration, deaths from chronic hepatitis and cirrhosis were also examined separately. The approximate relative risk of death from chronic liver disease other than cancer in the lowest quarter of cholesterol concentration was again 1·6, while for quarters II, III, and IV it was 1·0, 0·9, and 0·4 respectively. As was the case for liver cancer, this inverse association of cholesterol concentration with other chronic liver diseases was significant ($p<0·05$). When deaths from liver cancer and other liver disease were combined the inverse relation with cholesterol concentration became more clearly significant ($p<0·01$). The inverse association with non-medical causes of death was marginally significant when other variables were not taken into account, but became non-significant after adjustment. Due chiefly to the inverse relation with death from chronic liver disease, there was a significant inverse relation between serum cholesterol concentration and all deaths from non-vascular, non-cancer causes ($p<0·05$).

Discussion

DEATH FROM CORONARY HEART DISEASE

The role of high blood cholesterol concentration as a major risk factor for coronary heart disease has been well established by several epidemiological studies,[1-6] but these were mostly conducted in Western populations that are at high risk of the disease. Nevertheless, results from comparisons between different populations indicate that, throughout a wide range, the lower the mean cholesterol concentration the lower the risk of coronary heart disease.[21-23] In the seven countries study the median plasma cholesterol concentrations in the 16 study cohorts ranged from about 4·1 mmol/l to about 6·5 mmol/l.[21] Comparing these 16 cohorts with each other, there was a strong positive correlation between the median plasma cholesterol concentration and the 10 year mortality from coronary heart disease, with a large proportion of the between-population variation in mortality from this disease explained by differences in plasma cholesterol concentrations. Evidence from the Ni-Hon-San study also suggested that the observed international differences in coronary heart disease risk could not be attributed chiefly to genetic differences.[23] In that study of Japanese populations living in three different environments it was found that the differences in the mean cholesterol concentrations were a major determinant of the differences in death rates from coronary heart disease.

By contrast, a few of the prospective studies conducted within populations in which the mean cholesterol concentrations were high have failed to show a relation between cholesterol and mortality from coronary heart disease in the lower range of serum cholesterol concentration, where coronary heart disease is less common.[1,8] The results of these within population studies are sometimes interpreted as showing a threshold, perhaps at about 5·2 mmol/l, below which a lower cholesterol concentration is not associated with a lower risk of coronary heart disease.[7] The real relation at lower concentrations of cholesterol is, however, difficult to assess reliably in Western populations. This is partly because the risk of death from coronary heart disease is low among people who have low cholesterol concentration, so the lower end of the cholesterol-coronary heart disease relation is particularly affected by random fluctuations, and partly because few people in Western populations really have low concentrations after repeated cholesterol measurements. Thus even in the large prospective studies that have been conducted in Western populations (such as the multiple risk factor infarction trial, which included nearly 360 000 people[6]) the amount of information on subjects who really had low cholesterol concentrations is limited.

In many Asian populations, however, the situation is different: most of the adults really have cholesterol concentrations that by Western standards are low, so differences between the effects of different low concentrations can be compared directly. Thus in a prospective study of 16 711 Japanese subjects with a mean baseline serum cholesterol concentration of 4·3 mmol/l there was a significant positive relation between risk of coronary heart disease and baseline cholesterol.[24] Our study in Shanghai, with a mean baseline serum cholesterol concentration of about 4·2 mmol/l confirms this positive relation, and within the range of usual cholesterol concentrations studied (3·8-4·7 mmol/l) there was no evidence of any apparent threshold below which a lower cholesterol concentration was not associated with a lower risk of coronary heart disease.

In our study population a 10% difference in baseline serum cholesterol concentration was associated with about a 21% difference in the risk of death from coronary heart disease. This difference in risk is similar to that reported for prospective studies in Western populations in which the mean cholesterol concentrations were high.[6] In all such studies, however, the reported relation is only of baseline cholesterol measurement with coronary heart disease. This will, because of the "regression dilution bias,"[14] tend to underestimate substantially the strength of the relation between usual cholesterol concentration and coronary heart disease. Remeasurements of cholesterol in a sample of the population in our study allowed approximate correction for this bias, and it was then estimated that a 4 (SD 1)% difference in usual cholesterol concentration was associated with a 21 (SD 6)% (95% confidence interval 9% to 35%) difference in the risk of coronary heart disease. Even if the strength of this relation was due partly to chance, it does illustrate the statistically inevitable prediction that the true relation between serum cholesterol concentration and coronary heart disease must be substantially stronger than is suggested by standard analyses of only baseline cholesterol concentration.

DEATH FROM STROKE

It has been suggested by some investigators that low cholesterol concentrations may cause haemorrhagic stroke[25] and that this might be of particular import-

ance in countries such as China and Japan, where mortality from haemorrhagic stroke is high and cholesterol concentrations are low. In our study, which included about as many subjects with stroke as the multiple risk factor infarction trial follow up study,[25] no association was seen between cholesterol concentration and death from stroke, even among subjects with diastolic blood pressure ≥90 mm Hg.[20] The aetiology of the fatal strokes is not reliably known, but other studies in China have shown that more than half of all fatal strokes are due to haemorrhage.[26,27] So, although the present study cannot address the question of whether haemorrhagic stroke rates are slightly increased in subjects with low cholesterol concentrations, it does indicate that no strong association is likely to exist. Furthermore, our study provides direct evidence that even within a population in which haemorrhagic stroke rates are high, low cholesterol concentration is not associated with any appreciable increase in total mortality from stroke.

DEATH FROM CANCER

Before prospective survey data can be used to address the question of whether cholesterol affects mortality from cancer it is necessary to allow for the fact that established cancer can reduce cholesterol concentrations[28-30] and for the theoretical possibility that life threatening chronic diseases of any other systems (such as the liver) that importantly affect the uptake, transport, or synthesis of cholesterol might do likewise. For cancer, the results from other prospective studies have been inconsistent. Some indicate an inverse relation of serum cholesterol concentration with risk of cancer,[28-35] others show no association,[36,37] and some even indicate a positive relation.[38] In several of the studies in which an inverse association has been observed there is evidence that this is largely or wholly secondary to the metabolic consequences of cancer present at the time of entry to the study (a phenomenon referred to as the "preclinical cancer" effect[28-30]), although some other studies have not found this.[32-35] Our study, in which the mean cholesterol concentration is unusually low, might be particularly informative about any possible inverse relation between serum cholesterol concentration and risk of cancer.

Overall, there was no significant association between cholesterol concentration and total mortality from cancer, but there did seem to be an inverse relation between serum cholesterol concentration and liver cancer. This persisted even after exclusion of those subjects who died within the first three years after baseline examination, indicating that the inverse relation was not chiefly attributable to some short term preclinical effect of liver cancer reducing cholesterol concentration. An inverse association was also seen between serum cholesterol concentration and death from chronic non-malignant liver disease. In China 10-15% of the population have lifelong persistent hepatitis B virus infection of the liver, which accounts for most of the liver cancer and for many other deaths from chronic liver disease.[39,40] In many cases infection with hepatitis B virus starts in early childhood, and if chronic infection of the liver with hepatitis B virus lowers serum cholesterol concentrations long term this could explain the inverse association between cholesterol concentration and deaths from liver disease in this study. Further studies of cholesterol concentration in people with and without chronic hepatitis B virus infection are needed to test this hypothesis.

NON-MEDICAL CAUSES OF DEATH

Studies of the relation of cholesterol concentration with death from non-medical causes (that is, accidents or violence) are sparse and the results have not been consistent. A recent review of six trials of cholesterol lowering purported to show an excess of such deaths in the group allocated cholesterol lowering treatment.[41] But these analyses were based on only a small subset of the relevant trials (and, even among these, follow up data for a substantial number of randomised patients were excluded so that appropriately unbiased "intention to treat" analyses could not be performed), and they were not supported by a more complete and more systematic overview of all of the available data (published or not) from all randomised trials of cholesterol lowering by diet or drugs in which no such excess was seen.[42] Similarly, the large prospective observational studies do not provide any consistent evidence that low cholesterol concentration is associated with any material increase in the risk of accidental or violent deaths.[43] In our study there was a marginally significant inverse association between deaths attributed to non-medical causes and serum cholesterol concentration. This group of deaths from non-medical causes included accidents (five deaths), external injury (four), suicide (five), and other causes (three). The number of such deaths was small, the association became non-significant after taking other variables into account, no single cause was predominant, other studies do not support it, and there is no biologically plausible explanation, so it seems that this marginally significant inverse association may largely or wholly represent a chance finding.

IMPLICATIONS

Cholesterol concentrations and rates of coronary heart disease vary widely between populations. The question of what constitutes a desirable blood cholesterol concentration is an important practical issue. Comparison with populations in which cholesterol concentrations are low and coronary heart disease is rare suggests that few people in Western populations have a biologically normal (as opposed to population average normal) cholesterol concentration. Our results in a Chinese population suggest that cholesterol may be an important cause of coronary heart disease not only in individuals with a high mean cholesterol concentration but also in those with what is, by Western standards, a normal or low cholesterol concentration. This implies that even for the majority of individuals in an urban Chinese population with serum cholesterol concentrations of about 4 mmol/l, a lower cholesterol concentration should eventually confer a lower risk of coronary heart disease, and that this would be still more so for most people in Western populations. On the other hand, our study provides no direct evidence that at the low end of the cholesterol range now seen in Western populations, lower cholesterol concentration would cause any material increase in deaths from other causes. The existence in rural China of adult populations in which the mean serum cholesterol concentrations are below 3 mmol/l and in which the rates of coronary heart disease are even lower than in Shanghai,[44] provides an interesting context for speculation about what might eventually be achievable in the West if practicable methods could be devised to reduce cholesterol concentrations on a population basis.

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The Clerkenwell scheme: assessing efficacy and cost of a psychiatric liaison service to a magistrates' court

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Abstract

***Objective*—To determine the efficacy of psychiatric liaison schemes to magistrates' courts in shortening the period that mentally ill accused people spend in custody between arrest, the provision of psychiatric reports, and admission to hospital under the Mental Health Act 1983 and to establish the direct costs of setting up such schemes.**

***Design*—A nine month prospective study of court referrals and concurrent analysis of prison records.**

***Setting*—An inner London magistrates' court (Clerkenwell) and a large remand prison (Brixton).**

***Patients*—Consecutive series of 80 remand prisoners receiving psychiatric assessment through a liaison scheme; 50 remand prisoners placed on hospital orders by magistrates' courts after being remanded to prison for reports; 364 psychiatric prisoners undergoing second opinion assessments at a remand prison; 520 offenders in a remand prison placed on hospital orders.**

***Main outcome measures*—Comparison of lengths of time spent in custody for different stages of the assessment and disposal process.**

***Results*—For the 50 remand prisoners assessed in prison the mean time from arrest to appearance in court with a psychiatric report was 33·7 days and from arrest to admission to hospital 50·8 days. For those examined in court under the liaison scheme the equivalent figures were 5·4 days ($t=12·63$, $p<0·0001$) and 8·7 days ($t=13·04$, $p<0·0001$). The number of hospital orders made at the court increased fourfold after the liaison scheme began. The additional direct costs of the scheme were negligible.**

***Conclusion*—Psychiatric liaison services to magistrates' courts can greatly reduce the length of time that offenders with mental disorders spend in custody. Such schemes may increase recognition of offenders suitable for admission to hospital. A scheme could be established in some areas within existing service provision.**

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Introduction

People who are accused of criminal offences and thought to be mentally disordered may spend considerable periods in custody on remand so that psychiatric reports can be prepared by catchment area services. Yet conditions in remand prisons are in general not suitable for the care of mentally disordered people.[1,2] Treatment in such settings is problematical in that the