CARDIOVASCULAR DISEASE

Twelve-year changes in vascular risk factors and their associations with mortality in a cohort of 3499 Thais: the Electricity Generating Authority of Thailand Study

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Background	Vascular mortality is increasing in economically developing countries such as Thailand but reliable data about the determinants of these changes are few.
Methods	In 1985, male and female employees of the Electricity Generating Authority of Thailand took part in a cardiovascular risk factor survey. In 1997, a follow-up survey was conducted and causes of death were determined for those subjects known to have died. Changes in levels of vascular risk factors over 12 years, and the associations of baseline risk factors with vascular mortality, were calculated.
Results	The 1985 survey recruited 3499 volunteers (average age 43 years) of whom 23% were female. In 1997, vital status was determined for 3318 (95%) and 2967 (85%) of the study participants were resurveyed. Mean levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP), body mass index, total cholesterol and high density lipoprotein (HDL) cholesterol all increased over the 12-year follow-up period. Over the same time, the prevalence of diabetes also rose but the proportion of current smokers decreased. Vascular diseases were the most frequent cause of death during follow-up (n = 46), were positively associated with baseline age, SBP, DBP, smoking, diabetes, male sex, and total cholesterol, and were negatively associated with HDL cholesterol.
Conclusions	Levels of most vascular risk factors worsened over the 12-year period between 1985 and 1997. The associations between baseline risk factor levels and vascular mortality were consistent with those observed in other populations. Interventions that control vascular risk factors have the potential to avert much premature vascular disease in Thailand.
Keywords	Cohort study, mortality, risk factors, Thailand, vascular diseases

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Worldwide, vascular diseases were the leading causes of death in 1999,[1] when stroke and coronary heart disease were responsible for about 5.1 and 7.1 million deaths, respectively.[1] While there are trends toward improvement in the rates of premature stroke and coronary heart disease in many industrialized countries,[2,3] the converse is true of most developing countries.[4] Since much of the world's population lives in developing countries, the global burden of vascular disease is projected to rise considerably over the next two decades.[4] In large part, this growth is likely to be explained by sociodemographic changes

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and associated deterioration in the levels of vascular risk factors in developing countries.[5,6]

In Thailand, vascular diseases have been the leading cause of death since 1987. In 1998 the five leading causes of death, in order, were heart disease, malignant neoplasms, accidents or poisonings, suicide or homicide or other injury, and hypertension or cerebrovascular disease. In 1998 there were over 54 000 deaths due to vascular causes[7] and between 1985 and 1997 the prevalence of heart disease in Thailand tripled, from 56 per 100 000 population to 168 per 100 000 population.[8] Cross-sectional studies have provided some limited information about levels of vascular risk factors in different population groups within Thailand,[9–15] but changes in risk factor levels with age and the associations of established vascular risk factors with mortality are not well documented. The Electricity Generating Authority of Thailand (EGAT) study was originally designed in 1985 as a cross-sectional study of vascular risk factor levels among employees. In 1997, the same individuals were resurveyed or else information about the cause of death was sought for those known to have died in the interim. The primary aim of these analyses was to describe 12-year changes in vascular risk factors in this cohort of subjects. A secondary aim was to determine the associations between baseline risk factors levels and the risk of vascular death.

Methods

Participants

In 1985, all employees of EGAT, based at the company's head plant in Nonthaburi, and aged 35–54 years, were invited to take part in a survey of vascular risk factors. Of the 7824 individuals that were potentially eligible for inclusion in the study in 1985, 3499 (2702 men and 797 women) volunteered to take part (Figure 1). Volunteers completed a self-administered questionnaire, underwent a physical examination, provided fasting blood samples, and underwent an oral glucose tolerance test. Twelve years later, in 1997, efforts were made to re-contact all living participants by letter, telephone, or personal contact. All identified, and willing, participants were re-surveyed (n = 2967) using procedures similar to those employed at baseline. Information on cause of death was sought for all subjects known to have died. Consent and ethical approval were obtained.

Risk factor assessments

Sociodemographic characteristics, current and prior medical conditions, behaviours relating to vascular disease, and prescribed treatments were collected using a self-administered questionnaire. Measures of height and weight were made by examination of the participants dressed in indoor clothing but without shoes. Blood pressure was measured after 5 minutes rest, using a calibrated mercury sphygmomanometer with systolic blood pressure (SBP) and diastolic blood pressure (DBP) recorded as the first and fifth Korotkoff sounds respectively. A single blood pressure measurement was made with participants in the supine position in 1985, whereas in 1997 blood pressure was recorded twice using seated measurements (of which only the first is used in analyses here, for comparability). On each occasion subjects were classified as hypertensive if their blood pressure was ≥140/90 mmHg, or if they were currently taking prescribed blood pressure lowering therapy.[16]

Blood samples were obtained after a 12-hour overnight fast. Serum total cholesterol, high density lipoprotein (HDL)
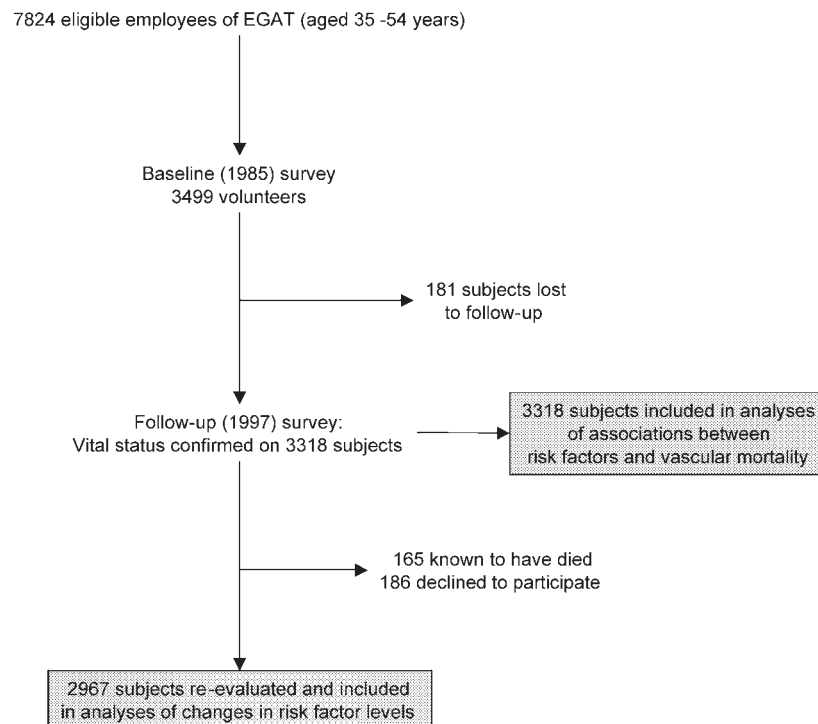

Figure 1 Flow chart indicating participation in baseline (1985) and follow-up (1997) surveys

cholesterol and triglycerides were measured using enzymatic-calorimetric assays (Boehringer Mannheim, Mannheim, Germany). High total cholesterol was defined as a total cholesterol level of ⩾6.2 mmol/l or current use of cholesterol lowering therapy.[17] Blood glucose levels were measured using a glucose oxidase method on capillary blood samples in 1985 (Reflocheck, Boehringer Mannheim, Mannheim, Germany) and on plasma samples in 1997 (Peridochrome, Boehringer Mannheim, Mannheim, Germany). On each occasion, oral glucose tolerance tests were performed by measuring blood glucose levels on fasting samples and on samples drawn 2 hours after the ingestion of a 75-g glucose load. Diabetes was defined on the basis of any of the following (1) a prior clinical diagnosis, (2) a fasting capillary glucose ⩾6.1 mmol/l, (3) a fasting plasma glucose ⩾7.0 mmol/l or (4) a 2-hour capillary or plasma glucose ⩾11.1 mmol/l).[18] Overweight or obesity was defined as body mass index (BMI) ⩾25 kg/m^2.[19]

Causes of death

An independent adjudication committee, of two cardiologists, one neurologist, one gastroenterologist and one internist, determined causes of death based upon all available evidence and using international criteria. Information about the likely cause of death was sought through review of information obtained from interviews with relatives and colleagues, medical notes, medical insurance reimbursement claims, records of hospital admissions, and death certificates. Deaths were classified as being due to one of the following eight broad causes: coronary heart disease (fatal myocardial infarction or sudden unexplained death), stroke (including subarachnoid haemorrhage), other vascular death (e.g. heart failure, valvular heart disease or peripheral arterial disease), respiratory disease (excluding malignancy), gastrointestinal or hepato-biliary disease (excluding malignancy), malignancy (of any kind), injuries (including suicide), or other.

Statistical methods

For analyses of changes in vascular risk factors over time, only those subjects for whom both baseline and follow-up data were available were included. For each survey (baseline and follow-up), sex-specific mean values of risk factors or proportions of the study population with each risk factor were calculated for continuous and categorical measures of exposure, respectively. Differences in the level or prevalence of each risk factor between the two surveys were tested using paired t-tests or McNemar tests.[20] On each occasion, differences between risk factor levels in men and women were examined using t-tests for continuous data and χ^2 tests for categorical data. All P-values were based on two-tailed tests of significance. Secondary analyses compared those people aged 47–54 years at the first survey with those in the same age group in the second survey. This age group was chosen since it represented the only age range that was common to the two surveys. Analyses were similar to the above, except that two-sample tests for independent samples were used.

Associations between risk factors and vascular death (coronary heart disease, stroke or other vascular death) were estimated using data on all subjects for whom data on vital status were available in 1997. Analyses were performed using Cox survival models that included age, sex, smoking, BMI,

diabetes, total cholesterol, HDL cholesterol, and SBP or DBP. The estimates for SBP and DBP and total cholesterol were corrected for measurement error (regression dilution bias) using attenuation factors derived from the Asia Pacific Cohort Studies Collaboration.[21]

All statistical analyses were performed using SPSS software (SPSS Version 10, SPSS Inc., Chicago, IL, USA).

Results

In 1997, vital status was determined on 3318 (95%) of the original cohort of 3499 and 181 (5%) were lost to follow-up (Figure 1). Among those whose vital status was known, 165 were known to have died, 186 declined to participate in the follow-up study, and 2967 were resurveyed. Compared with those who were re-surveyed in 1997, the 532 subjects that were not resurveyed were significantly older, were more likely to have been smokers, and were more likely to have been diabetic at baseline.

Changes in vascular risk factors levels between 1985 and 1997

The mean age of the study population was 42.5 years at baseline (1985) and 54.5 years at follow-up (1997). Mean levels of risk factors and proportions of individuals with abnormal risk factors levels are shown for men and women in Table 1. Over the 12-year study period, for both sexes, mean SBP, mean DBP, mean serum total cholesterol levels, mean serum HDL cholesterol levels, mean triglyceride levels and mean BMI all increased (all $P < 0.001$). Similarly, the proportion of the study population satisfying the criteria for hypertension, high total cholesterol levels, diabetes and obesity each rose markedly, for both men and women (all $P < 0.001$). In contrast, the proportion of current smokers decreased by approximately half in both sexes (both $P < 0.001$).

In 1985, mean levels of SBP and DBP, serum total cholesterol, serum HDL cholesterol, serum triglycerides, fasting capillary blood glucose and BMI were all worse in men than women and men were more likely to be diabetic and to smoke (all $P \le 0.003$). In 1997, mean serum total cholesterol was higher in women than men ($P < 0.001$), there was no longer a sex difference in mean BMI ($P = 0.45$), but otherwise risk factor levels remained worse in men than women (all others, $P < 0.005$).

Table 2 shows the same comparisons as Table 1 but for a common age group, 47–54 years. The changes from 1985 to 1997 are similar to those seen for the cohort comparison in Table 1, except that there is now no evidence of an increase in levels of triglycerides and the magnitude of differences is generally less, particularly where percentages of hypertension and diabetes are concerned.

Associations between risk factors and vascular death

Vital status was established on 3318 participants at the end of the study, and the mean length of follow-up was 12 years. Among the 165 participants who were known to have died, 9 died from unknown causes. Of the remainder, vascular diseases accounted for 46 deaths (28 due to coronary heart disease, 16 due to stroke, and 2 due to other vascular causes). Four male and one female death were sudden deaths. Other leading causes of death were malignancy (43 deaths), injuries (37 deaths), and

Table 1 Differences between vascular risk factor levels in 1985 and 1997 among 2967 Thai men and women

	Male (n = 2252)				Female (n = 715)			
	1985	1997	difference	P	1985	1997	difference	P
Age, years (range)	42.8 (35, 54)	54.8 (47, 66)			41.4 (35, 54)	53.4 (47, 66)		
Body mass index, kg/m²	23.2 (23.0, 23.3)	24.7 (24.5, 24.8)	1.5 (1.4, 1.6)	< 0.001	22.7 (22.5, 23.0)	24.8 (24.5, 25.0)	2.1 (1.9, 2.2)	< 0.001
Systolic blood pressure, mmHg	122 (121.1, 122.4)	139 (138.1, 139.6)	17 (16.4, 18.2)	< 0.001	115 (114.0, 116.2)	127 (125.4, 128.5)	12 (10.3, 13.2)	< 0.001
Diastolic blood pressure, mmHg	76 (75.9, 76.8)	84 (83.0, 84.1)	7 (6.6, 7.8)	< 0.001	71 (69.8, 71.3)	77 (75.6, 77.5)	6 (5.0, 7.0)	< 0.001
Serum total cholesterol, mmol/l	5.80 (5.76, 5.85)	6.13 (6.08, 6.17)	0.33 (0.29, 0.38)	< 0.001	5.65 (5.57, 5.73)	6.34 (6.27, 6.42)	0.69 (0.61, 0.78)	< 0.001
HDL[a] cholesterol, mmol/l	1.18 (1.17, 1.19)	1.33 (1.31, 1.34)	0.15 (0.14, 0.17)	< 0.001	1.36 (1.33, 1.38)	1.48 (1.46, 1.50)	0.12 (0.09, 0.14)	< 0.001
Triglycerides, mmol/l	1.83 (1.78, 1.89)	1.94 (1.89, 2.00)	0.12 (0.06, 0.18)	< 0.001	1.21 (1.16, 1.25)	1.50 (1.44, 1.55)	0.29 (0.23, 0.35)	< 0.001
Fasting glucose,[b] mmol/l	5.01 (4.98, 5.05)	5.42 (5.35, 5.50)	NA	NA	4.68 (4.62, 4.74)	5.04 (4.94, 5.14)	NA	NA
2-hour glucose,[b] mmol/l	6.52 (6.44, 6.60)	6.69 (6.56, 6.82)	NA	NA	6.83 (6.71, 6.95)	6.62 (6.43, 6.80)	NA	NA
Hypertension (%)								
Total	20.7 (19.0, 22.4)	53.3 (51.2, 55.5)	32.6 (30.4, 34.9)	< 0.001	11.3 (9.1, 13.9)	30.7 (27.3, 34.3)	19.3 (15.7, 22.8)	< 0.001
Diagnosed, on therapy	2.4 (1.8, 3.1)	17.3 (15.8, 19.0)	15.0 (13.3, 16.7)	< 0.001	1.5 (0.8, 2.7)	12.7 (10.3, 15.4)	11.1 (8.5, 13.8)	< 0.001
Undiagnosed	18.3 (16.7, 20.0)	36.0 (34.0, 38.1)	17.7 (15.1, 20.3)	< 0.001	9.8 (7.7, 12.2)	18.0 (15.2, 21.1)	8.2 (4.7, 11.8)	< 0.001
High total cholesterol (%)								
Total	34.1 (32.1, 36.1)	51.0 (48.8, 53.1)	17.0 (14.6, 19.5)	< 0.001	26.4 (23.2, 29.8)	58.2 (54.4, 61.9)	31.8 (27.4, 36.1)	< 0.001
Diagnosed, on therapy	0.0 (0.0, 0.2)	13.8 (12.3, 15.3)	13.7 (12.2, 15.2)	< 0.001	0.1 (0.0, 0.8)	14.5 (11.9, 17.3)	14.3 (11.7, 17.0)	< 0.001
Undiagnosed	34.0 (32.1, 36.0)	37.2 (35.1, 39.3)	3.2 (0.3, 6.0)	0.03	26.2 (23.0, 29.6)	43.7 (40.0, 47.5)	17.5 (12.6, 22.4)	< 0.001
Diabetes (%)								
Total	6.1 (5.1, 7.2)	17.5 (15.9, 19.2)	11.3 (9.5, 12.9)	< 0.001	4.3 (3.0, 6.1)	12.4 (10.0, 15.1)	8.0 (5.3, 10.6)	< 0.001
Diagnosed	1.3 (0.9, 1.9)	11.5 (10.2, 13.0)	10.3 (8.8, 11.7)	< 0.001	0.7 (0.2, 1.6)	8.2 (6.3, 10.6)	7.5 (5.4, 9.7)	< 0.001
Undiagnosed	4.8 (3.9, 5.7)	5.9 (4.9, 7.0)	1.2 (−0.2, 2.5)	0.09	3.6 (2.4, 5.3)	4.1 (2.8, 5.9)	0.5 (−1.5, 2.5)	0.64
Overweight or obesity (%)	25.5 (23.7, 27.4)	43.2 (41.1, 45.2)	17.6 (15.6, 19.5)	< 0.001	20.7 (17.8, 23.9)	40.9 (37.3, 44.7)	20.2 (16.8, 23.5)	< 0.001
Proportion of current smokers (%)	53.0 (50.9, 55.1)	28.4 (26.5, 30.3)	−24.6 (22.6, 26.6)	< 0.001	6.2 (4.5, 8.2)	3.6 (2.3, 5.2)	−2.6 (0.9, 4.2)	< 0.001

Figures represent mean values or percentages (95% CI).

[a] High density lipoprotein.

[b] Glucose measures differed between years: capillary blood used in 1985; plasma in 1997. 2-hour glucose was following glucose tolerance test.

Hypertension was classified as BP ≥140/90 mmHg, or current use of prescribed blood pressure lowering therapy.[16] High total cholesterol defined as a total cholesterol level of 6.2 mmol/l or higher or current use of cholesterol lowering therapy.[17] Diabetes defined as: (1) a prior clinical diagnosis, (2) a fasting capillary glucose ≥6.1 mmol/l, (3) a fasting plasma glucose ≥7.0 mmol/l, or (4) a 2-hour capillary or plasma glucose ≥11.1 mmol/l).[18] Overweight or obesity defined as body mass index (BMI) ≥25 kg/m².[19]

Table 2 Differences between vascular risk factor levels in 1985 and 1997 amongst Thai men and women aged 47–54 years old

	Male				Female			
	1985 (n = 594)	1997 (n = 1161)	difference	P	1985 (n = 99)	1997 (n = 445)	difference	P
Age, years (range)	49.5 (47, 54)	50.7 (47, 54)		40.9 (47, 54)	50.6 (47, 54)			40.9 (47, 54)
Body mass index, kg/m²	23.7 (23.4, 23.9)	24.6 (24.4, 24.8)	1.0 (0.7, 1.3)	< 0.001	23.9 (23.3, 24.5)	24.5 (24.2, 24.8)	0.6 (−0.1, 1.3)	0.08
Systolic blood pressure, mmHg	124 (122.3, 125.0)	136 (134.4, 136.7)	12 (10.1, 13.7)	< 0.001	119 (115.7, 122.8)	124 (122.3, 126.0)	5 (0.9, 8.9)	0.02
Diastolic blood pressure, mmHg	78 (77.2, 79.0)	83 (82.1, 83.6)	5 (3.6, 5.9)	< 0.001	73 (71.1, 75.5)	76 (74.5, 76.8)	2 (−0.1, 4.8)	0.06
Serum total cholesterol, mmol/l	5.75 (5.65, 5.85)	6.15 (6.09, 6.21)	0.40 (0.29, 0.52)	< 0.001	6.05 (5.80, 6.30)	6.28 (6.17, 6.38)	0.23 (−0.04, 0.50)	0.10
HDL[a] cholesterol, mmol/l	1.24 (1.21, 1.26)	1.33 (1.31, 1.34)	0.09 (0.06, 0.12)	< 0.001	1.38 (1.31, 1.45)	1.50 (1.47, 1.53)	0.12 (−0.04, 0.19)	0.03
Triglycerides, mmol/l	2.00 (1.88, 2.12)	1.98 (1.90, 2.06)	−0.02 (−0.17, 0.13)	0.80	1.46 (1.32, 1.59)	1.42 (1.36, 1.48)	−0.04 (−0.19, 0.11)	0.60
Fasting glucose,[b] mmol/l	5.14 (5.03, 5.24)	5.24 (5.15, 5.34)	NA	NA	4.88 (4.66, 5.11)	4.86 (4.76, 4.96)	NA	NA
2-hour capillary blood glucose,[b] mmol/l	6.93 (6.73, 7.12)	6.46 (6.30, 6.63)	NA	NA	7.07 (6.66, 7.47)	6.43 (6.20, 6.65)	NA	NA
Hypertension (%)								
Total	27.8 (24.2, 31.6)	46.8 (43.9, 49.9)	19.1 (14.4, 23.7)	< 0.001	19.2 (12.0, 28.3)	26.0 (22.0, 30.5)	6.9 (−1.9, 15.7)	0.15
Diagnosed, on therapy	4.7 (3.2, 6.7)	13.4 (11.5, 15.6)	8.7 (6.1, 11.4)	< 0.001	1.0 (0.0, 5.5)	9.1 (6.5, 12.2)	8.1 (4.7, 11.4)	0.006
Undiagnosed	23.1 (19.7, 26.7)	33.4 (30.6, 36.3)	10.3 (5.9, 14.7)	< 0.001	18.2 (11.1, 27.2)	17.0 (13.5, 20.9)	−1.2 (−9.6, 7.2)	0.77
High total cholesterol (%)								
Total	32.7 (28.9, 36.7)	49.3 (46.3, 52.4)	16.6 (11.8, 21.5)	< 0.001	38.4 (28.8, 48.7)	51.8 (46.9, 56.6)	13.4 (2.7, 24.1)	0.02
Diagnosed, on therapy	0.2 (0.0, 0.9)	10.6 (8.8, 12.6)	10.4 (8.5, 12.3)	< 0.001	0.0 (0.0, 3.7)	8.2 (5.8, 11.2)	8.2 (5.6, 10.8)	0.003
Undiagnosed	32.5 (28.8, 36.5)	38.8 (35.9, 41.8)	6.2 (1.5, 11.0)	0.01	38.4 (28.8, 48.7)	43.6 (38.8, 48.4)	5.2 (−5.5, 15.8)	0.35
Diabetes (%)								
Total	8.4 (6.3, 11.0)	13.8 (11.8, 16.0)	5.3 (2.3, 7.4)	0.001	9.1 (4.2, 16.6)	9.6 (7.0, 12.9)	0.6 (−5.8, 6.9)	0.87
Diagnosed	1.9 (0.9, 3.3)	8.4 (6.8, 10.2)	6.5 (4.5, 8.5)	< 0.001	1.0 (0.0, 5.5)	5.6 (3.7, 8.3)	4.6 (1.6, 7.6)	0.03
Undiagnosed	6.6 (4.7, 8.9)	5.4 (4.1, 6.9)	−1.2 (−3.6, 1.2)	0.32	8.1 (3.6, 15.3)	4.0 (2.3, 6.3)	−4.1 (−9.8, 1.6)	0.09
Overweight or obesity (%)	30.5 (26.8, 34.3)	42.1 (39.2, 45.0)	11.6 (6.9, 16.3)	< 0.001	33.3 (24.2, 43.5)	38.6 (34.1, 43.4)	5.3 (−5.0, 15.6)	0.33
Proportion of current smokers (%)	50.0 (45.9, 54.1)	31.0 (28.3, 33.8)	−19.0 (−14.2, −23.8)	< 0.001	11.1 (5.7, 19.0)	2.3 (1.1, 4.2)	−8.8 (−2.0, −15.2)	< 0.001

Figures represent mean values or percentages (95% CI) unless indicated.

[a] High density lipoprotein.

[b] Glucose measures differed between years: capillary blood used in 1985; plasma in 1997. 2-hour glucose was following glucose tolerance test.

Hypertension was classified as BP ≥140/90 mmHg, or current use of prescribed blood pressure lowering therapy.[16] High total cholesterol defined as a total cholesterol level of ≥6.2 mmol/l or current use of cholesterol lowering therapy.[17] Diabetes defined as: (1) a prior clinical diagnosis, (2) a fasting capillary glucose ≥6.1 mmol/l, (3) a fasting plasma glucose ≥7.0 mmol/l, or (4) a 2-hour capillary or plasma glucose ≥11.1 mmol/l).[18] Overweight or obesity defined as body mass index (BMI) ≥25 kg/m².[19]

non-malignant gastrointestinal and hepatic disease (22 deaths). Age, SBP, DBP, smoking, and diabetes were all positively and significantly associated with vascular mortality (all $P < 0.05$) (Table 3). High density lipoprotein cholesterol levels were significantly inversely associated with vascular mortality ($P = 0.003$). Non-significant positive associations were also observed for male sex, BMI and total cholesterol (all $P > 0.2$). The CI about all estimates were wide. Correction for regression dilution bias increased the strength of the associations observed for SBP, DBP, and total cholesterol by 93%, 133%, and 61%, respectively.

Discussion

In this cohort of workers employed by the EGAT, the levels of most established risk factors for vascular disease deteriorated during the 12-year period from 1985 to 1997. The observed differences reflect both the increase in the mean age of the study participants and other population-wide secular changes in social and behavioural determinants of risk factor levels, such as increased consumption of dietary fat and decreased physical exercise.[5,6,22,23] Only for triglycerides was there evidence that changes were due only to ageing. The associations of the baseline risk factor levels with vascular death were similar to those observed in other populations[24,25] and vascular diseases were the leading cause of mortality during follow up. The predominance of vascular causes of death in this cohort is consistent with the documented rise in the rates of vascular disease in Thailand,[8] for which this study suggests deterioration in classical cardiovascular risk factors as an important cause. The rise in vascular disease in Thailand is comparable to that observed in many other developing countries in which chronic and non-communicable conditions are replacing nutritional deficiencies, maternal and perinatal conditions, and infectious diseases as the principal determinants of the national disease burden.[4,5]

The average rise in blood pressure (16 mmHg [SBP]/7 mmHg [DBP]) and the increase in the proportion of the population with hypertension (from 18% to 48%) were greater than would be expected for ageing alone.[26,27] While minor methodological differences between the procedures used to measure blood

Table 3 Hazard ratios (95% CI) for the association of risk factors with vascular death among 3318 Thais followed for an average of 12 years

	Unadjusted	Adjusted[a]
Age (10 years)	3.7 (2.1, 6.5)	2.7 (1.5, 4.8)
Sex (male/female)	6.7 (1.6, 27.7)	2.6 (0.6, 11.1)
Body mass index (5 kg/m^2)	1.6 (1.1, 2.4)	1.0 (0.6, 1.6)
Systolic blood pressure (10 mmHg)[b]	1.7 (1.3, 2.2)	1.3 (1.0, 1.8)
Diastolic blood pressure (5 mmHg)[b]	1.7 (1.4, 2.2)	1.5 (1.1, 1.9)
Total cholesterol (1.0 mmol/l)[b]	1.1 (0.8, 1.7)	1.0 (0.7, 1.6)
HDL[c] cholesterol (0.2 mmol/l)	0.6 (0.5, 0.8)	0.7 (0.6, 0.9)
Diabetes[d] (yes/no)	5.3 (2.7, 10.2)	3.3 (1.6, 6.6)
Current smokers (yes/no)	2.8 (1.5, 5.2)	2.2 (1.1, 4.1)

[a] Adjusted for every other variable in the Table (except that diastolic and systolic blood pressure were not adjusted for each other).

[b] Corrected for regression dilution bias using factors derived from the Asia Pacific Cohort Studies Collaboration.[21]

[c] High density lipoprotein.

[d] Prior clinical diagnosis of diabetes and/or one or more of the following: (1) fasting capillary glucose ≥6.1 mmol/l, (2) fasting plasma glucose ≥7.0 mmol/l, (3) 2-hour capillary, or (4) plasma glucose ≥11.1 mmol/l following an oral glucose tolerance test.[18]

pressure at the baseline and follow-up survey may have contributed to the observed differences, these are unlikely to account for all the changes observed. Studies that have compared supine and seated blood pressure measurements in the same subjects demonstrate that SBP is unaffected by posture while DBP is approximately 5 mmHg higher when measured with subjects in the seated position.[28,29] Thus, while the observed increase in DBP might be explained by the altered posture during measurement, this difference in technique is unlikely to have accounted for the rise in SBP. Deteriorating dietary and behavioural practices of the study population most likely also contributed importantly to the rises in blood pressure observed.[23] Similar levels of changes were observed for undiagnosed and treated hypertension.

There were also methodological differences in the techniques used for assay of glucose levels in 1985 (capillary blood) and 1997 (venous blood) and it is therefore not possible to reliably compare the mean glucose levels recorded on the two occasions. However, recent World Health Organization criteria for the diagnosis of diabetes provide definitions based on both capillary and plasma samples,[18] and it is therefore possible to compare the prevalence of diabetes on the two occasions. The observed, almost threefold, rise in the prevalence of diabetes over the 12-year follow-up period is greater than the differences in the prevalence of diabetes observed in cross-sectional studies in other populations,[30,31] or amongst the 47–54 year olds at the two time points in this study. Again, therefore, the changes observed in the EGAT participants most likely comprise both the effects of the increased mean age of the cohort and other changes such as a greatly increased prevalence of overweight and obesity. Changes in sex-specific diabetes prevalence were almost completely due to diagnosed diabetes; undiagnosed diabetes hardly altered. This is partially explained by the effect of being in the study, i.e. as a result of diagnosis in 1985.

The proportion of smokers among the EGAT participants declined sharply during the 12 years of follow-up. Similar trends in smoking habits have been observed in other cross-sectional surveys conducted in Thailand, although the magnitude of the decrease observed in EGAT is greater than that observed in national surveys.[32–34] This may reflect a particularly good uptake of a national anti-smoking campaign by this well educated and affluent cohort.[34] The observed decrease in smoking is all the more remarkable given recent evidence from Europe that, despite extensive public health campaigns, the prevalence of smoking has declined little over the last few years, even among those with established coronary heart disease.[35]

The risk factor levels and the changes recorded among the participants of the EGAT study are likely to be precise and reliable. However, the employees of EGAT are largely middle-class, well educated, urban-dwelling individuals receiving an above average level of income and medical care and as such, are not representative of the overall Thai population.[36] Further, classic sources of bias in epidemiological studies are sure to have played a role: the 'healthy worker effect'[20] (employment of relatively healthy people); non-response (the response rate in the first survey was only 45%—women had higher response rates than men and the older had higher response rates than the younger); and loss to follow-up (which probably led to underestimation of the effects of smoking and diabetes). Accordingly, the absolute levels of risk factors recorded in these surveys,

and the absolute changes in risk factor levels over time, are unlikely to be generalizable to the Thai nation as a whole. It is likely, nonetheless, that the observed changes do give an indication of the most likely trends in risk factor levels in Thailand and that these data might usefully contribute to the development of national strategies for cardiovascular disease prevention.

In addition to clear data about risk factor levels, this study also provides the first ever information about the effects of vascular risk factors on the occurrence of major vascular events in a Thai population. The total number of fatal events recorded in the EGAT participants during the 12-year follow-up period was small and, consequently, evidence about the determinants of vascular death is rather imprecise. However, the associations observed are consistent with those seen in other much larger studies conducted in Eastern and Western populations,[24,25] and confirm the likely importance of established vascular risk factors for the Thai population.

In summary, the changes in the levels of vascular risk factors observed in the EGAT study are consistent with the increase in mortality from vascular disease observed in Thailand over the last few decades.[8] While trends in smoking were favourable, the deterioration in most other major vascular risk factors was substantial, and for blood pressure in particular, the increases appear to exceed those expected from ageing of the population alone. The implementation of interventions that improve levels of vascular risk factors have great potential to prevent much premature vascular disease in Thailand over the next few decades.

Acknowledgements

KEY MESSAGES

- Non-communicable diseases are now the leading cause of death in many developing countries.
- Over the period 1985–1997 levels of all major vascular risk factors, except smoking, worsened in this occupational study population.
- Associations between risk factors and vascular death was much as would be expected from studies in Western populations.
- Implementation of strategies to reduce vascular risk factors should be a priority in Thailand.

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Commentary: Cardiovascular implications of the epidemiological transition for the developing world: Thailand as a case in point

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Cardiovascular disease (CVD) is currently the leading cause of death and disability in developed nations, and is increasing

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rapidly in the developing world.[1] If demographic trends continue, it is estimated that 90% of the global CVD burden will occur in low and middle-income countries by the year 2025. The rapid increase in CVD rates in developing regions is occurring at a time when infectious and nutritional deficiency diseases are in decline, a phenomenon that has been termed 'the epidemiologic transition'.[2] East Asia, in particular, is expected to suffer