



ELSEVIER



Long-term tracking of cardiovascular risk factors among men and women in a large population-based health system

The Vorarlberg Health Monitoring & Promotion Programme

Hanno Ulmer^{a*}, Cecily Kelleher^b, Günter Diem^c, Hans Concini^c

^a*Institute of Biostatistics and Documentation, Leopold Franzens University of Innsbruck, Innsbruck, Austria*

^b*Department of Public Health Medicine and Epidemiology, University College Dublin, Dublin, Ireland*

^c*Agency for Social- and Preventive Medicine, Bregenz, Austria*

Received 14 October 2002; revised 10 January 2003; accepted 5 February 2003

KEYWORDS

Cardiovascular risk factors;
Longitudinal studies;
Body mass index;
Tracking;
Mortality

Aims To document tracking patterns, if any, over time, of classical cardiovascular risk factors in men and women participants in the Vorarlberg Health Monitoring and Promotion Programme (VHM&PP)

Methods and Results 67 413 men and 82 237 women underwent a total of 454 448 standardised examinations in the 15 year period 1985–1999. Measures included were systolic and diastolic blood pressure, height, weight and fasting sample for total cholesterol, triglycerides, gamma-gt and blood glucose. Tracking coefficients were calculated by multivariable regression models using the GEE estimation method. All variables showed evidence of significant tracking over time, whether estimated in 10-year age bands or among individuals categorized as being at high risk using cut-points proposed by international guidelines. Effects were most marked for body mass index (0.87, SE 0.005 in men and 0.89, SE 0.003 in women), and were also associated with increasing age. Women who died during follow-up showed stronger tracking patterns for triglycerides and gamma-gt and weaker effects for blood pressure, but there was no effect on patterns according to survival in men. Tracking coefficients were weaker among initially high-risk individuals.

Conclusions This is the largest study yet of adults to demonstrate significant tracking effects of cardiovascular risk factors over time. The strength of this effect should be considered in assessing effectiveness of risk factor modification programmes. The study is novel too in highlighting more fully differences according to gender and social circumstances and in taking account of the impact on long-term survival.

© 2003 The European Society of Cardiology. Published by Elsevier Science Ltd. All rights reserved.

Introduction

While there has been much research in recent decades on risk factor profiles for predicting cardiovascular disease,¹ there has been considerably less analysis of longitudinal changes in these risk

* Corresponding author. Dr H. Ulmer, Leopold Franzens University of Innsbruck, Department of Biostatistics and Documentation, Schoepfstrasse 41, A-6020 Innsbruck, Austria. Tel.: 0512-507-3201; fax: 0512-507-2711
E-mail address: Hanno.Ulmer@uibk.ac.at (H. Ulmer).

factors in the healthy population in order to predict future values by earlier measurements.² Since risk factors show a greater impact on the development of cardiovascular disease the longer they are present,¹ it is important to know how stable a given measurement is likely to be. Tracking may be defined as the longitudinal stability of a certain risk factor or the predictability of a measurement early in life for values later in life.^{3,4} While it is well understood that risk factor profiles may change over the life-course, (a cohort or secular effect) and that risk profiles of individuals can be altered by concerted modification (as for instance in life-style change or in response to treatment) the tracking concept implies that an individual's rank order of risk stays relatively constant over time, so that for instance a child with high normal weight or blood pressure is more predisposed to having pathological levels requiring treatment later on.⁵ This implies a systematic effect in that individuals at high risk may be predicted to remain so. Tracking weakens, when individuals move from low risk to moderate or high risk, or from high risk to moderate or low risk. From a clinical perspective therefore, tracking patterns may need to be modified or maintained depending on risk status. Segments of the population that are at a higher order of risk should be motivated to lower their risk by all means possible, the rest to maintain their status. The degree of tracking of a certain risk factor is a measure for the attainability of this aim. Whether this phenomenon continues to be important throughout adult life is not well researched. Most conventional risk scores for instance are derived from long-term epidemiological follow-up based on relatively few examination points.

Longitudinal study designs involving repeated measurements on the same subjects over a longer time period are required to perform tracking analyses. Up to now, only a limited number of studies have fulfilled these criteria. Most studies have concerned children rather than adults,⁵⁻⁹ women as a group have been under-researched¹⁰ and no studies to date have been on a scale sufficient to assess the relationship, if any, between tracking pattern and mortality.

In Vorarlberg, the westernmost province of Austria, population-based documentation of cardiovascular risk factors has been performed routinely since 1970 by the Agency for Social- and Preventive Medicine. From the outset, this ongoing extensive risk factor surveillance and treatment referral programme has comprised medical examinations of more than two thirds of the entire population of this province.¹¹⁻¹³ Documentation was considered to

provide the basis for a scientifically justified approach to prevent diseases and to promote the health of the population. The acronym VHM&PP (Vorarlberg Health Monitoring and Promotion Programme) was created to refer to it.

The purpose of the present study was to address the tracking of classical cardiovascular risk factors, body mass index, systemic blood pressure, glucose, serum lipids, as well as gamma-gt in a healthy population of approximately 150 000 persons examined on repeated occasions over a period of 15 years, comprising a total of more than 450 000 examinations. To our knowledge, it is the first such study to examine patterns in both men and women on such a scale over a prolonged time period of follow-up and surveillance.

Methods

Study population

Men, 67 413 (44.9%) and women, 82 237 (55.1%), a total of 149 650 individuals, participated in the VHM&PP between 1985 and 1999. During this period, men underwent 191 629 (42.2%) and women 262 819 examinations (57.8%); a total of 454 448 in all. Informed consent to store and process the data was obtained from all participants at each examination time-point and ethical approval was obtained. The participants' age range was between 19 and 96 years with a mean age of 42 years (standard deviation=15 years) for both men and women. Socio-demographic details are given in [Table 1](#). More women than men were examined, men were more likely to be married than women, there was a representative range across social class groups and the vast majority were of Austrian origin, though migrant workers were also included. A total of 5373 persons died in the course of follow-up and cause of death was linked in the database.

Measurements

The VHM&PP participants underwent unequal number of repeated examinations, ranging from one up to 14 visits. The examinations were taken at arbitrary time-points during a fifteen-year period from 1985 to 1999. They were performed in a standardized way by trained general practitioners and internists and included a physical examination and the recording of socio-demographic information. The methodology has been described previously.¹¹ The physical examination included fasting blood sample and measurements of height, weight and blood pressure. Systolic and diastolic

Table 1 Sociodemographic characteristics of the participants, VHM&PP 1985 to 1999

	Men		Women	
	n	%	n	%
Baseline age groups ^a				
20–24 years	7893	11.7	12 561	15.3
25–34 years	18 619	27.6	20 601	25.1
35–44 years	14 503	21.5	16 037	19.5
45–54 years	12 563	18.6	14 037	17.1
55–64 years	8610	12.8	10 370	12.6
65 years and older	5225	7.8	8631	10.5
Total	67 413	45.0	82 237	55.0
Marital status ^b				
Single	12 092	18.5	12 887	15.8
Married	48 232	73.7	52 511	64.4
Divorced	3521	5.4	9648	11.8
Widowed	1602	2.4	6444	7.9
Work status ^c				
White collar	33 883	52.3	42 976	54.2
Blue collar	23 899	36.9	30 034	37.9
Self employed	7056	10.9	6275	7.9
Austrian origin				
Austrian	60 932	90.4	75 823	92.2
Mortality	2942	4.4	2451	3.0
Number of examinations				
Only one examination	25 808	38.3	27 539	33.5
Two examinations	13 812	20.5	15 901	19.9
Three examinations	8859	13.1	10 955	13.2
Four and more examinations	18 934	28.1	27 842	33.4
Interval to second examination				
Within 1, 5 years	8292	12.3	12 693	15.4
Between 1, 5 and 3 years	17 364	25.8	22 648	27.5
Later	15 949	23.7	19 357	23.5
Only one examination	25 808	38.3	27 539	33.5

^aFour missing values.

^b2717 (1.8%) missing values.

^cIncluding pensioners, housewives were classified according to their husband's job, 5531 (3.7%) missing values.

blood pressure were measured with a mercury sphygmomanometer in the sitting position. Total cholesterol, triglycerides, gamma-gt and blood glucose were determined enzymatically by two central laboratories. The two laboratories underwent a standardized internal and external quality procedure. Between 1985 and 1988 glucose measurement was done in a non-fasting manner. Only fasting glucose measurements after 1989 were used for statistical analysis.

Tracking and its assessment

Tracking refers to the tendency of individuals to maintain their rank or position within a group over time. Longitudinal data for at least two points in time are necessary, and correlations between the measurements are used to estimate tracking. A measure of the strength of a correlation is provided by the coefficient of correlation, in this case called

a tracking coefficient. Values of a tracking coefficient range between -1 and 1 , however since negative values indicate an inverse relationship, which is unlikely between repeated measurements, only values from 0 to 1 are of importance. Since correlations are influenced by factors such as age at the first observation, measurement variability and the time span between measurements, tracking coefficients should be estimated through a statistical model that is able to adjust for these factors. Multivariate adjusted tracking coefficients are reported in table 5, 6 and 7. The following is a suggested guide to the interpretation of tracking coefficients: <0.30 low correlation, 0.30 to 0.60 moderate, 0.60 to 0.90 moderately high, >0.9 high correlation. Low correlation means that intra-individual changes of risk factor values from the initial to subsequent examinations are very likely. Positive and negative changes count the same way. A tracking coefficient is just a pure measure for longitudinal changeability. That does not inform about the direction of individual changes. To evaluate additionally directions of change, tracking can be further assessed by focusing on maintaining a certain characteristic over time.

Statistical analysis

Prevalence of selected high risk factor categories (obesity, hypertension, hypercholesterolaemia, hypertriglyceridaemia, hyperglycaemia and elevated gamma-gt) were calculated for the baseline examinations and followed for their persistence at subsequent examinations. This categorization, based on cut-points proposed by international guidelines,¹ has been reported previously.¹¹ Multivariable logistic regression analyses were performed to study the effect of socio-demographic indicators for remaining in a high risk factor class for the participants who were at high risk at the initial examination and who had at least two examinations. Variables for these regression models were selected according to their bivariate association with the dependent variable evaluated through Chi-square and Mann–Whitney U tests.

Tracking coefficients for the different risk factors were calculated according to the procedure outlined by Twisk et al.¹⁴ by multivariable regression models using the GEE estimation method.¹⁵

$$Y_{it} = \beta_0 + \beta_1 Y_{it1} + \beta_2 t + \beta_3 \text{Age}_i + \beta_4 \text{Smoking}_i + \varepsilon_{it}$$

where Y_{it} is the z-transformed risk factor variable for measurements of individual i from t_2 to t_m (for t_1 the first and t_m the last examination). Age_i means the age at the individual's initial

Table 2 Means and standard deviations of baseline risk factor values by sex and age-groups, VHM&PP 1985 to 1999

	Men			Women		
	20–44 years	45–64 years	65 years and older	20–44 years	45–64 years	65 years and older
Body mass index (kg/m ²) ^a	24.6 (3.4) ^b	26.3 (3.5)	25.8 (3.5)	22.9 (4.1)	26.2 (4.6)	26.1 (4.4)
Systolic blood pressure (mmHg)	126.9 (15.4)	138.3 (19.8)	148.6 (21.1)	119.1 (15.5)	138.3 (21.2)	153.1 (21.6)
Diastolic blood pressure (mmHg)	79.8 (10.2)	85.0 (11.3)	84.4 (10.6)	76.1 (9.9)	84.2 (11.3)	85.4 (10.9)
Total cholesterol (mg/dl)	208.8 (45.8)	236.1 (45.8)	232.5 (45.5)	198.0 (37.5)	239.1 (45.8)	250.9 (47.3)
Triglycerides (mg/dl)	144.9 (104.0)	171.0 (115.5)	151.0 (94.1)	100.0 (55.1)	128.8 (78.1)	150.9 (85.6)
Glucose (mg/dl) ^c	83.8 (17.7)	94.6 (31.0)	98.5 (35.7)	82.0 (15.4)	91.7 (26.2)	98.6 (34.0)
Gamma gt (mg/dl)	21.6 (29.2)	30.3 (42.1)	26.0 (34.8)	11.25 (15.0)	17.0 (23.6)	18.5 (22.7)
	%			%		
Regular smoking	30.2	23.7	14.7	24.7	11.5	4.2

^aExcluding 2100 (1.4%) missing values within body mass index and blood pressure and 4500 (3.1%) missing values within cholesterol, triglyceride, glucose and gamma-gt (values missing partly due to missing consent of the participants).

^bMeans (standard deviations).

^cExcluding measurements before introduction of fasting glucose measurement.

examination, Smoking_i means whether the individual is a current smoker at the first examination. ε_{it} is the measurement error for individual i at examination t . The regression coefficient β_1 represents in its standardized form the tracking coefficient. It is standardized from -1 to 1 by using subgroup-specific z -transformed variables¹⁶ and can be interpreted similarly to a simple correlation coefficient. Statistical tests of subgroup differences (gender, mortality) between tracking coefficients were performed by using the test statistic $(\beta_{1\text{subgroup1}} - \beta_{1\text{subgroup2}}) / \sqrt{(\text{se}(\beta_{1\text{subgroup1}})^2 + \text{se}(\beta_{1\text{subgroup2}})^2)}$, where $\text{se}(\beta_1)$ is the robust standard error¹⁷ of the tracking coefficient. To take account of possible false-positive significant results through multiple comparisons a hierarchical procedure for sub-groups comparisons was chosen; only in case of an overall significant effect of for instance, mortality, differences between subgroups such as causes of deaths were evaluated.

p -values smaller than 0.05 were considered to indicate statistical significance. Statistical analysis was performed using Stata¹⁸ and SPSS statistical software.¹⁹

Results

Baseline risk factor values for individuals' initial examinations are reported in Table 2, separately for men and women. These show the expected sex differences, with women in general having significantly lower values than men. However the pattern according to age differed considerably between men and women. In men only systolic blood pressure and glucose level continued to rise among those 65 years and older, mean values of all the other variables tended to be lower in the older

compared with the middle-aged group. In women body mass index and diastolic blood pressure levels stabilised, but all other variables were highest in the oldest group. Values for gamma-gt show a wide range at all ages, reflected in the standard deviation. Smoking rates declined steeply with age in the case of both men and women.

Table 3 shows for selected risk factor categories whether individuals who were 'at risk' at the initial examination maintained that risk status at the subsequent examination. The effect was most marked in the case of obesity; 79.5% of the men and 83.2% of the women with an initial body mass index greater than 30 kg/m^2 were still in the same category at the next examination. The pattern was less consistent for the other risk factors. Severe hypertension for instance persisted in only 46.2% of the male and 49% of the female participants; however it must be noted that treatment may well have been initiated in the interim, though such information is not recorded in the database.

Socio-economic indicators as well as lifestyle factors such as smoking showed significant associations with the stability of a risk factor, evaluated by logistic regression analyses to assess predictors for remaining at high risk (Table 4). While the odds are modest, because of the huge sample size, they are statistically significant. Women for instance were more likely to persist in the obesity category, but less likely to keep elevated triglycerides, glucose or gamma-gt. Blue collar and self-employed workers were also more likely to maintain an adverse risk factor profile. In general, risk factors were more likely to persist in older individuals. This was most pronounced in the case of elevated glucose. The odds of having a glucose level greater

Table 3 Elevated risk factors by sex for individuals with at least two examinations, VHM&PP 1985 to 1999

	Men				Women			
	Elevated at 1st examination		Still elevated at 2nd examination		Elevated at 1st examination		Still elevated at 2nd examination	
	n	%	n ^a	%	n	%	n ^a	%
Body mass index >30 kg/m ²	3616	8.8	2827/3556	79.5	5595	10.3	4582/5506	83.2
Blood pressure >160/95 mmHg	4754	11.5	2157/4670	46.2	5779	10.6	2794/5703	49.0
Total cholesterol >250 mg/dl	10341	25.4	6311/10152	62.2	11958	22.6	7730/11698	66.1
Triglycerides >200 mg/dl	8894	22.0	5018/8714	57.6	4542	8.6	2220/4463	49.7
Glucose >100 mg/dl ^b	1992	15.4	894/1915	46.7	1872	12.2	757/1800	42.1
Gamma-gt >28 mg/dl	9280	22.8	6368/9097	70.0	3591	6.8	1980/3517	56.3

^aProportion of individuals keeping the high risk factor level from the first examination at the second examination, excluding individuals with missing values.

^bExcluding non fasting glucose.

than 100 mg/dl increased on average by 4% per life-year gained. Elevated gamma-gt was more likely to persist in smoking individuals rather than in non-smokers. Remaining with an elevated gamma-gt greater than 28 mg/dl was also significantly more frequent in participants who died later in the observation period, but otherwise the effect of subsequent death was not consistent.

In addition to these results contrasting first and second visits, which might be influenced by either a regression effect to the mean or the clinical effect of intervention, the calculated tracking coefficients making use of all measurements over repeated visits showed clearly that there are considerable differences regarding the stability of risk factor variables. As may be seen in Table 5, there are statistically significant relationships between baseline and subsequent measures in the case of all variables, irrespective of the age of original examination. In the case of both men and women, relationships were strongest with body mass index, followed by gamma-gt and total cholesterol levels. Body mass index showed tracking-coefficient values greater than 0.8 in all age- and sex-groups. In general relationships tended to be stronger from the age of 35 years and upwards than for the youngest cohort group. Men and women differed most notably in the case of gamma-gt. In this parameter, men showed tracking coefficients between 0.70 and 0.72, much higher than the coefficients in women (between 0.49 and 0.68). Similarly, as with the results reported in Table 3, women showed a higher degree of tracking for blood pressure, body mass index and cholesterol.

Tracking coefficients are categorized according to whether individuals died or not during follow-up in Table 6. Mortality did not have any statistically significant effects on tracking coefficients of cardiovascular risk factors in men, but it did signifi-

cantly influence degree of tracking of systolic blood pressure, triglyceride, glucose and gamma-gt in women. Tracking coefficients were lower for systolic blood pressure (0.52 vs 0.58) and higher for triglycerides (0.68 vs 0.62), glucose (0.66 vs 0.60) and gamma-gt (0.73 vs 0.65) in women who died in the course of the study. It may be seen however that the magnitude of the difference in coefficients between survivors and those who died is not great.

Significant effects of mortality in women could also be seen when calculating tracking coefficients for individuals with elevated risk factors at their initial examinations only (Table 7). What is chiefly notable here, by contrast with the general tracking patterns seen in Table 5, is that the tracking coefficients are not as strong, whether for survivors or not, in high risk individuals as they are for the group as a whole. There continue to be differences in pattern between women and men. Women who died during follow-up tracked significantly better for high cholesterol (greater than 250 mg/dl), high triglyceride (greater than 200 mg/dl) and high gamma-gt (greater than 28 mg/dl). In all three variables, tracking was significantly higher in women who died during follow-up.

Disease-specific patterns were also evaluated. Deaths from cardiovascular disease revealed the highest tracking coefficients among women: 0.47 (0.05) for cholesterol, 0.47 (0.07) for triglycerides and 0.46 (0.1) for gamma-gt. For deaths through stroke the coefficients were quite similar: 0.47 (0.08) for cholesterol, 0.36 (0.1) for triglycerides and 0.46 (0.1) for gamma-gt. Cancer as cause of death showed lower tracking with 0.42 (0.06) for cholesterol, 0.30 (0.09) for triglyceride and 0.21 (0.14) for gamma-gt. There were no significant disease-specific effects of mortality in men.

Table 4 Factors for remaining in the high risk class at the second examination, calculated by multiple logistic regression analyses, VHM&PP 1985 to 1999

	Body mass index >30 kg/m ² n=5362 ^a Odds Ratio (95% CI), Sig. ^b	Blood pressure >160/95 mmHg n=10113 Odds Ratio (95% CI), Sig.	Total cholesterol >250 mg/dl n=21271 Odds Ratio (95% CI), Sig.	Triglycerides >200 mg/dl n=12885 Odds Ratio (95% CI), Sig.	Glucose >100 mg/dl n=3584 Odds Ratio (95% CI), Sig.	Gamma-gt >28 mg/dl n=12355 Odds Ratio (95% CI), Sig.
Age (per year)	0.99 (0.99–1.01), p=0.1	1.02 (1.01–1.03), p<0.001	1.02 (1.01–1.03), p<0.001	1.01 (1.01–1.02), p<0.001	1.04 (1.03–1.05), p<0.001	1.02 (1.01–1.03), p=0.001
Female vs male gender	1.28 (1.15–1.43), p<0.001	1.01 (0.92–1.09), p=0.92	1.07 (1.01–1.14), p=0.03	0.69 (0.64–0.75), p<0.001	0.77 (0.67–0.88), p<0.001	0.53 (0.49–0.58), p<0.001
Blue vs white collar	1.11 (0.99–1.24), p=0.07	1.04 (0.95–1.13), p=0.41	1.04 (0.98–1.11), p=0.18	1.09 (1.01–1.18), p=0.02	1.29 (1.12–1.49), p<0.001	1.13 (1.04–1.23), p=0.004
Self empl. vs white collar	1.21 (0.97–1.50), p=0.09	1.08 (0.93–1.26), p=0.32	1.00 (0.90–1.11), p=0.92	1.08 (0.95–1.22), p=0.26	0.92 (0.70–1.19), p=0.51	1.02 (0.89–1.17), p=0.764
Non- vs Austrian origin	Not in the final model	Not in the final model	0.75 (0.60–0.93), p=0.01	Not in the final model	Not in the final model	0.69 (0.53–0.89), p=0.004
Smoker vs non-smoker	Not in the final model	0.91 (0.81–1.01), p=0.08	Not in the final model	1.25 (1.15–1.35), p<0.001	1.10 (0.93–1.31), p=0.28	1.16 (1.06–1.26), p=0.001
Months from baseline ^c	Not in the final model	Not in the final model	1.01 (1.00–1.02), p=0.09	0.98 (0.97–0.99), p=0.001	1.00 (0.99–1.01), p=0.44	0.98 (0.97–0.99), p<0.001
Year of baseline exam ^c	Not in the final model	Not in the final model	1.00 (0.99–1.01), p=0.88	Not in the final model	1.09 (1.05–1.13), p<0.001	0.98 (0.97–0.99), p<0.001
Death	0.85 (0.67–1.08), p=0.188	Not in the final model	Not in the final model	Not in the final model	1.18 (0.69–2.02), p=0.54	1.31 (1.10–1.58), p=0.003

^aOnly participants who were 'at risk' at the first examination and who had a second examination qualified for these analyses.

^bCalculated by multiple logistic regression analyses with 'remaining at risk (yes/no)' as dependent variable. An Odds Ratio greater than 1 expresses a higher chance to remain in the high risk factor class.

^cThe results of the logistic regression analyses were adjusted for the time between the initial and the second examination as well as for the year of the initial examination, as appropriate.

Table 5 Tracking coefficients for cardiovascular risk factors and gamma-gt, by age at baseline and sex, VHM&PP 1985 to 1999

Men n=36760 ^a Baseline age-groups	Systolic bp.	Diastolic bp.	Body mass index	Cholesterol	Triglycerides	Glucose ^b	Gamma-gt
25–34 years	0.39 (0.01) ^c	0.35 (0.01)	0.85 (0.02)	0.66 (0.01)	0.56 (0.01)	0.37 (0.03)	0.70 (0.01)
35–44 years	0.41 (0.01)	0.36 (0.01)	0.88 (0.01)	0.65 (0.01)	0.58 (0.01)	0.51 (0.03)	0.72 (0.01)
45–54 years	0.46 (0.01)	0.39 (0.01)	0.88 (0.01)	0.63 (0.01)	0.59 (0.01)	0.63 (0.03)	0.72 (0.01)
55–64 years	0.47 (0.01)	0.37 (0.01)	0.87 (0.01)	0.63 (0.01)	0.61 (0.01)	0.64 (0.04)	0.72 (0.01)
65 years and older	0.46 (0.02)	0.37 (0.02)	0.89 (0.01)	0.66 (0.02)	0.60 (0.01)	0.67 (0.04)	0.71 (0.02)
25 years and older	0.52 (0.004)	0.39 (0.004)	0.87 (0.005)	0.66 (0.005)	0.62 (0.004)	0.60 (0.02)	0.72 (0.005)
Women n=46638							
	Systolic bp.	Diastolic bp.	Body mass index	Cholesterol	Triglycerides	Glucose	Gamma-gt
25–34 years	0.36 (0.01)	0.32 (0.01)	0.85 (0.01)	0.61 (0.01)	0.47 (0.01)	0.35 (0.02)	0.49 (0.01)
35–44 years	0.42 (0.01)	0.37 (0.01)	0.88 (0.01)	0.62 (0.01)	0.55 (0.01)	0.47 (0.02)	0.61 (0.01)
45–54 years	0.46 (0.01)	0.39 (0.01)	0.89 (0.01)	0.63 (0.01)	0.59 (0.01)	0.58 (0.03)	0.65 (0.01)
55–64 years	0.45 (0.01)	0.37 (0.01)	0.89 (0.01)	0.61 (0.01)	0.63 (0.01)	0.68 (0.04)	0.68 (0.01)
65 years and older	0.42 (0.01)	0.36 (0.01)	0.88 (0.01)	0.66 (0.01)	0.63 (0.01)	0.72 (0.03)	0.68 (0.01)
25 years and older	0.59 (0.004)	0.43 (0.004)	0.89 (0.003)	0.69 (0.004)	0.63 (0.004)	0.60 (0.015)	0.65 (0.005)

^aOnly participants with at least two examinations qualified for this analyses.

^bCalculated for measurements after 1989 (introduction of fasting glucose measurements).

^cTracking coefficients (standard errors), estimated as standardized regression coefficients of the baseline measurement through GEE models. Adjusted for age, smoking, body-mass-index and days from baseline, as appropriate. Triglycerides, glucose and gamma-gt were transformed logarithmically.

Table 6 Tracking coefficients in men and women 25 years and older, by survivorship, VHM&PP 1985 to 1999

	Men		Women	
	Survived n=35189 ^a	Died during follow-up n=1571	Survived n=45277	Died during follow-up n=1361
Body mass index	0.87 (0.005) ^b	0.88 (0.04)	0.89 (0.003)	0.88 (0.03)
Systolic blood pressure	0.51 (0.004)	0.52 (0.02)	0.58 (0.004)	0.52 (0.02) ^c
Diastolic blood pressure	0.39 (0.005)	0.41 (0.02)	0.43 (0.004)	0.41 (0.03)
Total cholesterol	0.66 (0.005)	0.66 (0.02)	0.70 (0.004)	0.70 (0.02)
Triglycerides	0.62 (0.004)	0.62 (0.02)	0.62 (0.004)	0.68 (0.02) ^c
Glucose	0.60 (0.02)	0.60 (0.09)	0.60 (0.015)	0.66 (0.068)
Gamma-gt	0.72 (0.005)	0.74 (0.02)	0.65 (0.005)	0.73 (0.02) ^c

^aParticipants with at least two measurements excluding missing values.

^bTracking coefficients (standard errors), estimated as standardized regression coefficients of the baseline measurement through GEE models. Adjusted for age, smoking, body-mass-index and days from baseline, as appropriate. Triglycerides, glucose and gamma-gt were transformed logarithmically. Models were calculated separately for the four subgroups.

^cDifferences are statistically significant ($p < 0.05$) for systolic blood pressure, tryglyceride and gamma-gt in women.

Discussion

Confirmation of tracking patterns

This analysis confirms the significant tendency for risk factors in adults to track over time, utilising the method described previously by Twisk et al.⁴ and employed also in the analysis of the Tromso study.² The strength of this particular analysis is the scale of the database available, which allows for a more precise estimate of this effect than was possible in previous studies, particularly among women where the question has been less well studied to date. It is

clear that there is a strong consistent effect over time, particularly for body mass index. Most studies of tracking have mainly concerned children.^{5–9} Only a few studies have to date reported tracking coefficients in adults. The results of the comparable, though smaller-scale Tromso Study, were based on the analysis of non fasting blood samples whereas the present results are solely based on fasting blood samples. Triglycerides showed considerably lower tracking coefficients (between 0.3 and 0.49 in men and 0.33 and 0.51 in women) in comparison to the present study. However, with the exception of

Table 7 Tracking coefficients for men and women with elevated risk factors at initial examination, by survivorship, VHM&PP 1985 to 1999

	Men		Women	
	Survived	Died during follow-up	Survived	Died during follow-up
Body mass index >30 kg/m ²	0.58 (0.01) ^a n=3124 ^b	0.62 (0.04) n=205	0.55 (0.01) n=4779	0.56 (0.06) n=225
Blood pressure >160/95 mmHg				
Systolic blood pressure	0.33 (0.01)	0.29 (0.04)	0.28 (0.01)	0.23 (0.05)
Diastolic blood pressure	0.26 (0.01) n=4112	0.30 (0.05) n=382	0.28 (0.01) n=5151	0.33 (0.04) n=421
Total cholesterol >250 mg/dl	0.31 (0.01) n=8812	0.33 (0.04) n=516	0.31 (0.01) n=9984	0.38 (0.03) ^c n=574
Triglycerides >200 mg/dl	0.27 (0.01) n=7524	0.24 (0.05) n=375	0.26 (0.01) n=3820	0.40 (0.05) ^c n=285
Glucose >100 mg/dl ^c	0.45 (0.02) n=2121	0.38 (0.12) n=70	0.53 (0.02) n=2116	0.64 (0.09) n=42
Gamma-gt >28 mg/dl	0.37 (0.01) n=7487	0.43 (0.04) n=427	0.18 (0.02) n=2905	0.35 (0.07) ^c n=181

^aTracking coefficients (standard errors), estimated as standardized regression coefficients of the baseline measurement through GEE models. Adjusted for age, smoking, body-mass-index and days from baseline, as appropriate. Triglycerides, glucose and gamma-gt were transformed logarithmically. Models were calculated separately for the four subgroups.

^bParticipants with elevated risk factor at initial visit and with at least two measurements, excluding missing values.

^cDifferences regarding mortality are statistically significant ($p < 0.05$) only in women, for cholesterol, triglyceride and gamma-gt.

triglycerides, as might be expected for this reason, the order of tracking was the same, body mass index showed the highest degree of tracking, followed by cholesterol levels and blood pressure. The findings for cholesterol from the Framingham Study²⁰ are also in agreement with the present results, indicating tracking coefficients for total cholesterol of 0.69 in men and 0.61 in women. The Dormont High School Study (mean baseline age 34 years) reported coefficients of 0.38, 0.44, and 0.88 for systolic blood pressure, diastolic blood pressure and body mass index in men and 0.54, 0.54 and 0.81 in women.²¹ We may reasonably therefore conclude that tracking occurs in adults as well as children, most especially in relation to body mass index. While conventional risk prediction scores may not necessarily take this explicitly into account, it should be considered by clinicians in assessing the feasibility of a given individual making successful efforts to modify risk.

Potential study limitations

There are some limitations to the VHM&PP to be mentioned, primarily due to the mechanism of recruiting the participants into the study. Although the sample is very large and covers a substantial part of the whole population of the region and hence is likely to be representative, it is still a self-selected sample. There were differences regarding the participation of women and men. Participation of women was higher and more equally

distributed across the age-span, whereas participation of men was less frequent in the youngest and oldest age groups. In addition, there are varying numbers of examinations at different time-points taken by the participants. There were of course individuals with elevated risk factors at their initial examinations who did not return for a second visit, whose outcome is therefore, not considered here, since our primary focus was on the impact of tracking so that only those who returned for further follow-up have available data.

Another limitation concerns medication of the individuals at risk. Since the recording of medication data was not available, no analyses on the influences of treatment on tracking could be performed. We do know, however, from the standard protocol employed by the physicians that frequency of recall is influenced by level of risk factor detected since this is a well-established primary care treatment programme. We may therefore, infer that those with elevated risk factors were likely to have received treatment, which may explain why levels of blood pressure and glucose were less likely to track for instance and also why the high-risk individuals were generally less consistent trackers. Persons who were treated for hypertension and for hypercholesteraemia were not excluded from the analyses. This should not be treated as a drawback at a clinical practice level however, since it offers a useful reflection on real life situations for practitioners. It should be

noted in this context that in the Tromso study, where treatment status was known, there was no appreciable effect on the tracking patterns.²

Clinical implications for risk factor modification

The contribution of tracking has important implications for intervention programmes. It can be brought to the attention of individuals as part of risk factor modification counselling that a tendency identified even when relatively young is likely to persist, particularly in the case of overweight and obesity. Given the epidemic proportions of obesity worldwide²² it is important to be aware that individuals will have inherent difficulty in altering their relative rank order in respect of body weight, a well-established clinical fact in obesity clinics which is underlined systematically here. This suggests that public health strategies that shift the population average in the appropriate direction from the earliest age might in the long-term be more successful than individual-level strategies that target high risk individuals after clinically significant overweight has been established.²³ Intervention is most likely to be effective the younger the age of the individuals, based on these findings.

Our data are also interesting in assessing the influence of socio-demographic factors on this phenomenon as it is now well established that social variations have an important influence on patterns of heart disease.²² We demonstrate for instance that those in less affluent groups are more likely to sustain high risk status, in keeping with what we know about the likely differential effectiveness of health promotion interventions according to social class.²³ The results of this study further suggest that particular attention needs to be paid to those who continue to smoke, for the same reason. Since there can be no physiological basis for these differences it does suggest scope for successful modification in risk if sufficient support is provided.

The differences identified between men and women are seen to be of increasing clinical importance, given the general ageing profile of western populations. It has long been established that in the post-menopausal period, women's risk factor profiles for cardiovascular disease deteriorate, though the reasons for this are controversial.^{24,25} What is interesting in the present study is the degree to which the patterns among the older men and women diverge, though both share the expected rise in systolic blood pressure and glucose levels. Moreover, the tracking patterns differ among men and women too, suggesting that consider-

ation of this fact should be taken account of in likely strategies for risk factor treatment and intervention.

The significance for long-term prognosis is more difficult to interpret. Are people who track more likely to die than those who do not and are high-risk individuals who track at more or less risk than those who do not? Comparable previous studies also did not address a possible survivor effect. In most of these studies participants were too young and the sample sizes were too small to address this issue. To our knowledge, no previous study has reported the association between tracking and mortality in adults. From the findings in this study there seems little effect on mortality of tracking per se, in men, but some effect in women, though the magnitude of the effect is small. Since individuals are tracking at all levels of risk one might not expect much effect on mortality per se from tracking and this seems to be the case here. However that fact that in women there is a more marked tracking effect for triglyceride and gamma-gt levels in relation to mortality and that this is more pronounced among high risk women for total cholesterol as well, suggests that prolonged high risk exposure is important. Given that we know risk factor levels tended to dip in older men but continued to rise in older women, the implications are that risk factor modification might well be beneficial in the latter group, though this has been controversial in the past.¹

In conclusion, this very large scale population based clinical intervention programme affords a detailed examination of risk factor profiles over long-term follow up, confirms the importance of risk factor tracking as a phenomenon in adults, highlights differences between men and women that merit further study and affords an opportunity to examine for the first time the effect on all cause and disease specific mortality.

VHM&PP Working Group

Dr Hans-Peter Bischof, Dr Elmar Bechter (Government of Vorarlberg, Austria).

Dr Leopold Bischof, Dr Josef Bachmann, Karl Huber, Dr Klaus Zitt, Dr Paul Gmeiner, Dr Johann Brändle (Agency for Social- and Preventive Medicine, Bregenz, Austria).

Prof. Larry Brant (National Institute on Aging, Baltimore, USA), Prof. Michael Kunze, Prof. Anita Rieder (Institute of Social Medicine, University of Vienna, Austria), Prof. Rudolf Bruppacher (Institute of Social- and Preventive Medicine, University of Basel, Switzerland).

Acknowledgements

Professor Kelleher, Dr Diem and Dr Ulmer are participants in the European Union funded Health Risk Monitoring Programme (EHRM). The planning of this paper was undertaken during an advisory visit on Ireland's National Health and Lifestyle Survey, SLAN to Centre for Health Promotion Studies, National University of Ireland, Galway by Dr Hanno Ulmer in March 2002. We are grateful to Professor Rudolf Bruppacher, Institute of Social- and Preventive Medicine, University of Basel and Professor Andrew Murphy, Department of General Practice, National University of Galway for their support with helpful comments.

References

1. Task Force Report. Prevention of coronary heart disease in clinical practice. *Eur Heart J* 1998;**19**:1434–503.
2. Wilsgaard T, Jacobsen BK, Schirmer H et al. Tracking of cardiovascular risk factors. The Tromso Study, 1979–1995. *Am J Epid* 2001;**154**:418–26.
3. Twisk JWR, Kemper HCG, Mellenbergh GJ. Mathematical and analytical aspects of tracking. *Epid Rev* 1994;**16**:165–83.
4. Twisk JWR, Kemper HCG, van Mechelen W et al. Tracking of risk factors for coronary heart disease over a 14-year period: a comparison between lifestyle and biologic risk factors with data from the Amsterdam growth and health study. *Am J Epid* 1997;**145**:888–98.
5. Kemper HC, Snel J, Verschuur R et al. Tracking of health and risk indicators of cardiovascular diseases from teenager to adult: Amsterdam Growth and Health Study. *Prev Med* 1990;**19**:642–55.
6. Webber LS, Srinivasan SR, Wattigney WA et al. Tracking of serum lipids and lipoproteins from childhood to adulthood. The Bogalusa Heart Study. *Am J Epid* 1991;**133**:884–99.
7. Porkka KV, Viikari JS, Taimela S et al. Tracking and predictiveness of serum lipid and lipoprotein measurements in childhood: a 12-year follow-up. The Cardiovascular Risk in Young Finns study. *Am J Epid* 1994;**140**:1096–110.
8. Tan F, Okamoto M, Suyama A et al. Tracking of cardiovascular risk factors and a cohort study on hyperlipidemia in rural schoolchildren in Japan. *J Epidemiol* 2000;**10**:255–61.
9. Porkka KV, Viikari JS. Tracking of serum lipids in children; association with the absolute lipid level – the cardiovascular risk in young Finns study. *J Clin Epidemiol* 1995;**48**:221–8.
10. Swan GE, Carmelli D, Larue A. Systolic blood pressure tracking over 25 to 30 years and cognitive performance in older adults. *Stroke* 1998;**29**:2334–40.
11. Ulmer H, Bachmann J, Huber K et al. Follow-up studies of routine health screening in Vorarlberg 1986–1994. *Wien Klin Wochenschr* 1997;**109**:160–4.
12. Ulmer H, Diem G, Bischof HP et al. Recent trends and sociodemographic distribution of cardiovascular risk factors: results from two population surveys in the Austrian WHO CINDI demonstration area. *Wien Klin Wochenschr* 2001;**113**:573–9.
13. Ulmer H, Bischof HP, Bachmann J et al. 12-years of public screening for cardiovascular risk factors in Vorarlberg/Austria. *Eur Heart J*. 1998;**19**(Suppl.):572.
14. Twisk JW, Kemper HCG, Mellenbergh DJ et al. Factors influencing tracking of cholesterol and high density lipoprotein: the Amsterdam Growth and Health Study. *Prev Med* 1996;**25**:355–64.
15. Zeger SL, Liang KY. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics* 1986;**42**:121–30.
16. Wang Y, Wang X. Re: Tracking of cardiovascular risk factors: the Tromso Study, 1979–1995. *Am J Ep* 2002;**155**:1144–5.
17. White H. A heteroskedasticity–consistent covariance matrix estimator and a direct test for heteroskedasticity. *Econometrica* 1980;**48**:817–30.
18. Stata Corporation. Stata Statistical Software: Release 7 Special Edition, 2001. College Station, TX.
19. SPSS Inc. SPSS for Windows: Version 11, 2001. Chicago, MN.
20. Castelli WP, Garrison RJ, Wilson PW et al. Incidence of coronary heart disease and lipoprotein cholesterol levels. The Framingham Study. *JAMA* 1986;**256**:2835–8.
21. Yong LC, Kuller LH. Tracking of blood pressure from adolescence to middle age: the Dormont High School Study. *Prev Med* 1994;**23**:418–26.
22. Kelleher C. Editorial: Evolution of cardio-vascular risk factors–light at the end of the tunnel? *Wien Klin Wochenschr* 2001;**113**:552–7.
23. Kelleher C. Evaluating Health Promotion in four key settings. In: Davies JK, MacDonald G, editors. Quality, Evidence and Effectiveness in Health Promotion. London: Routledge; 1998, p. 47–67.
24. Meade TW, Dyer S, Howarth DJ et al. Antithrombin-III and procoagulant activity –sex differences and effects of the menopause. *Br J Haematol* 1990;**74**:77–81.
25. Lawlor DA, Ebrahim S, Davey-Smith G. Sex matters: secular and geographical trends in sex differences in coronary heart disease mortality. *BMJ* 2001;**323**:541–5.