American Journal of Epidemiology
Vol. 145, No. 10
Printed in U.S.A.

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

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Because the magnitude of tracking coefficients (i.e., stability coefficients and tracking for subjects at risk) greatly depends on the initial age of subjects, the number and spacing of longitudinal measurements, and the length of the total time period, it is difficult to compare tracking coefficients from different studies with each other. Because in the Amsterdam Growth and Health Study both biologic (i.e., lipoproteins, blood pressure, body fatness, and cardiopulmonary fitness) and lifestyle (i.e., dietary intake, daily physical activity, smoking, and alcohol consumption) risk factors for coronary heart disease were measured, this study gives the unique possibility of comparing tracking coefficients of biologic and lifestyle risk factors within one data set. In the Amsterdam Growth and Health Study, six repeated measurements were carried out on 181 subjects over a period from 13 to 27 years of age, beginning in 1977. The results indicated that, over a period of 14 years covering adolescence and young adulthood, both stability coefficients and tracking for subjects at risk for lifestyle risk factors were low (except for smoking), indicating low predictability of early measurements for values later in life. For the biologic risk factors cardiopulmonary fitness and blood pressure, tracking was also low, while for the lipoproteins and body fatness, tracking was much better, indicating good predictability. *Am J Epidemiol* 1997;145:888–98.

alcohol drinking; blood pressure; epidemiologic methods; exercise; longitudinal studies; nutrition; skinfold thickness; smoking

Because in Western society coronary heart disease is one of the most important chronic diseases, one of the main topics of longitudinal epidemiologic studies is the analysis of the longitudinal development of coronary heart disease risk factors. An important aspect of these analyses is the assessment of tracking. Tracking can be defined as 1) the stability of a certain risk factor over time or 2) the predictability of a measurement of a certain risk factor early in life for values of the same risk factor later in life. Because tracking relates to the predictability of values later in life from early measurements, tracking analysis is often related to the early detection of subjects at risk (e.g., to answer the question if it is worthwhile to screen subjects at an early age according to risk factors for chronic diseases). If tracking exists for a certain risk factor, this means that subjects at risk can be identified at a young age, and therefore preventive strategies can start as soon as possible from that age on.

Received for publication June 3, 1996, and accepted for publication January 15, 1997.

Abbreviation: CI, confidence interval.

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In the literature, tracking coefficients were reported for biologic coronary heart disease risk factors such as serum cholesterol, blood pressure, and body fatness (1–4), as well as for lifestyle coronary heart disease risk factors such as dietary intake, physical activity, smoking behavior, and alcohol consumption (5–8). However, the magnitude of the tracking coefficients depends on the initial age of the subjects, the number of repeated measurements, the time period(s) between the measurements, the length of the total time period under consideration, and the methodology used to assess tracking. Therefore, it is difficult to compare tracking coefficients of different biologic and lifestyle coronary heart disease risk factors with each other, when they are calculated in different studies. Because in the Amsterdam Growth and Health Study both biologic and lifestyle coronary heart disease risk factors were measured longitudinally, this study gives the unique possibility of comparing tracking coefficients of both biologic and lifestyle coronary heart disease risk factors with each other within one data set, a comparison that is not confounded by different study designs or different methodology.

Generally, tracking is assessed by calculating the interperiod correlation coefficient between two mea-

surements of the same risk factor in time or by calculating the percentage of subjects who maintain their relative position within a distribution of values at two measurements in time (9). All these "traditional" analyses, however, have two major drawbacks. First, if there are more than two longitudinal measurements, not all the available longitudinal data are used to assess tracking, and second, tracking can only be assessed without taking into account any covariate. Because of these drawbacks, in this paper a new method is applied to calculate a tracking coefficient. In this new method, tracking is assessed with the use of all the available longitudinal data and undercorrection of both time-dependent and time-independent covariates (10, 11).

The purpose of this study is to assess tracking of both biologic and lifestyle coronary heart disease risk factors. The tracking coefficients are calculated over a period of 14 years covering adolescence and young adulthood.

MATERIALS AND METHODS

Study design

The Amsterdam Growth and Health Study is an observational longitudinal study that started in 1977. From one secondary school in Amsterdam, 307 healthy subjects from the first and second forms were selected. During the first 4 years of the study, four consecutive measurements were carried out. The total number of subjects who completed these first 4 years of study was 233. In 1985, the study continued with 200 subjects and, in 1991, a sixth measurement was carried out with 181 subjects. The initial age of the subjects was 13.1 ± 0.8 (standard deviation) years and, at the measurement in 1991, the age of the subjects was 27.1 ± 0.8 years. The population used in the presented analysis consists of the 181 subjects (83 males and 98 females) who remained in the study up to the measurement at the age of 27 years. The total amount of missing observations during the whole period of measurement for these 181 subjects was about 2 percent (12). For the variables of interest in this study, there were no dropout effects between the subjects who did not continue the study up to the age of 27 years and the 181 subjects who remained in the study (13).

Biologic coronary heart disease risk factors

Blood cholesterol was determined by taking approximately 10 ml of venous blood from the vena antecubitus with a Vacutainer needle (Becton, Dickinson & Company, East Rutherford, New Jersey). Besides the concentration of total serum cholesterol, which was analyzed according to the methods of Huang et al. (14) and Abell et al. (15), the concentration of high density lipoprotein cholesterol and the ratio of total serum cholesterol to high density lipoprotein cholesterol were also determined. High density lipoprotein cholesterol was analyzed according to the method of Burstein and Samaille (16). External quality control took place with target samples from a World Health Organization reference laboratory (Lipid Standardization Laboratory, Atlanta, Georgia). Blood pressure was measured with an indirect method. A standard pressure cuff was placed around the upper left arm. With a sphygmomanometer, diastolic blood pressure (phase V) and systolic blood pressure were measured twice, and the lower value was recorded. Body fatness was operationalized as the sum of the thickness of four skinfolds (triceps, biceps, subscapular, and suprailiac). Skinfold thickness (expressed in mm) was measured with a Harpenden skinfold caliper (Holtain, United Kingdom; van Rietschoten & Houwens, the Netherlands) (17). Cardiopulmonary fitness was operationalized as maximal oxygen uptake ($\dot{V}_{O2}$-max) relative to body weight and was expressed in ml/(minute · kg$^{2/3}$). $\dot{V}_{O2}$-max was measured during running on a treadmill, with 8-km/hour speed and an increasing slope (18).

Lifestyle coronary heart disease risk factors

Dietary intake was measured by a modification of the cross-check dietary history interview. In this dietary history interview, which was specially tailored for use in the Amsterdam Growth and Health Study (19), all subjects were asked to recall their usual dietary intake by reporting the frequency, amounts, and methods of preparation of the foods consumed. This method was used to assess the usual food intake during the previous month. All consumed food items were transformed into nutrients by the Dutch Food and Nutrition Table (20). In relation to coronary heart disease risk factors, the daily intake of the following nutrients was assessed: 1) the intake of fat (expressed as energy percentage), 2) the ratio between the absolute intakes of polyunsaturated fatty acids and saturated fatty acids, 3) the intake of carbohydrates (expressed as energy percentage), and 4) the intake of cholesterol (expressed in mg/mJ). With the dietary history interview, alcohol consumption (expressed in grams per week) was also measured. Smoking behavior, i.e., the amount of tobacco (in grams) smoked per week, was asked for by a separate questionnaire.

Daily physical activity was measured by a structured interview, which was developed for the Amsterdam Growth and Health Study (21). With this interview, the total time spent on physical activities in relation to school and work and on other activities

(e.g., organized sports activities, unorganized sports activities, other leisure time activities, etc.) was measured. The measured times were combined with the intensity of the different activities to calculate a total weighted activity score (expressed in METs (metabolic unit equivalent to approximately 1 kcal/kg/day) per week). The activity interview covered the period of the 3 months prior to the interview. Both the cross-check dietary history and the daily physical activity interview were shown to be valid measurements of dietary intake and daily physical activity (19, 21).

Other parameters

The magnitude of tracking coefficients depends on the initial age of the subjects at which the study started. Therefore, tracking has to be corrected for initial age. However, in relation to coronary heart disease risk factors, initial biologic age is probably more important than initial calendar age (10, 13). In the Amsterdam Growth and Health Study, biologic age was determined by measuring the skeletal age from radiographs of the left hand, according to the Tanner-Whitehouse 2 method (TW2) (22). Before full maturity is reached, biologic age can be different from calendar or chronologic age. Rapidly maturing subjects have higher biologic ages in comparison with their calendar ages, while slowly maturing subjects have lower biologic ages in comparison with their chronologic ages.

Extensive information about the methods used in the Amsterdam Growth and Health Study is given by Kemper (23) and by Kemper and van Mechelen (24).

Statistical analysis

Tracking was assessed by a new method, which is explained in the Appendix and which has the advantage that all available longitudinal data were used to calculate the tracking coefficients (10, 11). Tracking was assessed in two ways. The first way was for continuous variables (Appendix, equation A1), which will give information about the stability of a risk factor in time. These tracking coefficients will be referred to as stability coefficients. These stability coefficients, however, will not give an answer to the basic question behind tracking: Is it worthwhile to screen subjects at an early age on coronary heart disease risk factors? To answer this question, the second way of tracking was assessed for subjects "at risk" at the initial measurement in order to maintain "at risk" later in life (Appendix, equations A2 and A3). As a result of these analyses, odds ratios were calculated to indicate this tracking for subjects at risk. Both the stability coefficients and the odds ratios regarding tracking for subjects at risk were calculated with generalized estimating equations (25, 26). To assess tracking for subjects at risk, we divided the subjects at each measurement into risk groups. If possible, this division was based on objective absolute threshold values for risk; if not, the division was based on arbitrary (relative) risk values based on the distribution of values (the 25th percentile). The threshold values used in this study are summarized in table 1 (27–32). The risk value for the sum of four skinfolds was based on the risk value for the percentage of body fat (20 percent for males and 30 percent for females) and is transformed into the sum of four skinfolds by the equations of Durnin and Rahaman (33) and Durnin and Womersley (34). For smoking behavior and alcohol consumption, a slightly different approach was followed. Because the distributions of smoking behavior and alcohol consumption were very skewed, stability coefficients could not be calculated. Moreover, the assessment of tracking for subjects at risk is problematic for these two variables.

TABLE 1. Threshold values for risk for both biologic and lifestyle coronary heart disease risk factors, which were used in the tracking analysis for subjects at risk, Amsterdam Growth and Health Study, 1977

	Biologic risk factors							
	TC* (mmol/liter)	HDL cholesterol* (mmol/liter)	TC/HDL cholesterol ratio	SBP* (mmHg)	DBP* (mmHg)	Sum (mm) of four skinfolds		Cardiopulmonary fitness
						Male	Female	
13–16 years	≥5.2	≤1.1	≥4.0	≥126	≥82	≥36.1	≥58.4	≤P25*
21–27 years	≥6.2	≤0.9	≥5.5	≥140	≥90	≥54.8	≥62.3	≤P25
	Lifestyle risk factors							
	Fat intake (% of energy)	P/S ratio*	Carbohydrate intake (% of energy)	Cholesterol intake (mg/mJ)	Daily physical activity			
13–16 years	≥35	≤0.5	≤50	≥33	≤P25			
21–27 years	≥35	≤0.5	≤50	≥33	≤P25			

* TC, total cholesterol; HDL cholesterol, high density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; P/S ratio, ratio between the intake of polyunsaturated fatty acids and the intake of saturated fatty acids; P25, 25th percentile.

For smoking, this is caused by the fact that the number of subjects who smoked at the initial measurement at the age of 13 years was very low (2.8 percent). The number of subjects who consumed any alcohol was slightly higher (9.4 percent), but the amount of alcohol consumed was little (1.49 ± 1.06 (standard error) g of alcohol per week). Therefore, for both smoking behavior and alcohol consumption, all measurements during the adolescent period (between 13 and 16 years of age) were considered as one. Subjects who had smoked or had consumed alcohol between 13 and 16 years of age were treated as smokers or drinkers. With generalized estimating equations, the odds ratio for smokers and for drinkers between 13 and 16 years was calculated in order to maintain their "at risk" status (i.e., smoker or drinker) at 21 and 27 years of age.

For both the calculation of stability coefficients and the tracking of subjects at risk, different coefficients for males and females were calculated if there was a significant ($p < 0.05$) interaction between the initial value of the risk factor and sex. If the interaction was not significant, the coefficients for males and females were considered to be equal.

All analyses were carried out by the Statistical Package of Interactive Data Analysis (SPIDA) (35).

TABLE 3. Tracking coefficients (stability coefficients) and 95% confidence intervals (CIs) for biologic and lifestyle coronary heart disease risk factors, calculated with generalized estimating equations over a period of 15 years from 1977, Amsterdam Growth and Health Study

	Stability coefficient	95% CI
Biologic risk factors		
Total cholesterol (TC)	0.71	0.65–0.78
High density lipoprotein cholesterol (HDL cholesterol)		
Male	0.51	0.42–0.60
Female	0.65	0.54–0.76
TC/HDL cholesterol ratio	0.71	0.63–0.79
Systolic blood pressure	0.43	0.34–0.53
Diastolic blood pressure	0.34	0.24–0.43
Sum of four skinfolds	0.63	0.56–0.71
Cardiopulmonary fitness	0.31	0.24–0.38
Lifestyle risk factors		
Fat intake	0.42	0.34–0.50
P/S ratio*	0.33	0.21–0.44
Carbohydrate intake	0.37	0.28–0.46
Cholesterol intake	0.34	0.26–0.42
Daily physical activity	0.34	0.19–0.49

* Ratio between the intake of polyunsaturated fatty acids and the intake of saturated fatty acids.

RESULTS

In table 2, descriptive information is given for all biologic and lifestyle coronary heart disease risk factors as they were measured at the initial measurement in 1977 at the age of 13 years. In table 3, the results of the tracking analysis are given. The stability coefficients of the lipoproteins and the sum of four skinfolds were higher than the ones for the other biologic coronary heart disease risk factors. For high density lipoprotein cholesterol there was a significant interaction between the initial value and sex, so different coefficients were calculated for males and females. For total serum cholesterol and the total serum cholesterol/high density lipoprotein cholesterol ratio, the coefficient was 0.71; for high density lipoprotein

TABLE 2. Mean values of biologic and lifestyle coronary heart disease risk factors at the initial measurement at 13 years of age, Amsterdam Growth and Health Study, 1977

	Biologic risk factors						
	TC* (mmol/liter)	HDL cholesterol* (mmol/liter)	TC/HDL cholesterol ratio	SBP* (mmHg)	DBP* (mmHg)	Sum of four skinfolds (mm)	Cardiopulmonary fitness ($\dot{V}_{O_2}$-max*)
Males	4.5 (0.6)†	1.5 (0.3)	3.1 (0.6)	124.1 (9.2)	74.0 (8.2)	28.4 (10.9)	21.6 (1.8)
Females	4.4 (0.7)	1.4 (0.3)	3.2 (0.7)	124.3 (9.5)	77.5 (7.1)	37.5 (12.8)	18.6 (1.5)
	Lifestyle risk factors						
	Fat intake (% of energy)	P/S ratio*	Carbohydrate intake (% of energy)	Cholesterol intake (mg/mJ)	Daily physical activity (1,000 METs*)		
Males	42.0 (4.4)	0.38 (0.14)	46.0 (4.3)	28.7 (5.9)	4.8 (1.7)		
Females	41.0 (4.7)	0.39 (0.19)	46.1 (4.0)	29.6 (6.2)	3.6 (1.4)		

* TC, total cholesterol; HDL cholesterol, high density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; $\dot{V}_{O_2}$-max, maximum oxygen consumption in ml/(minute · $kg^{2/3}$); P/S ratio, ratio between the intake of polyunsaturated fatty acids and the intake of saturated fatty acids; MET, metabolic unit equivalent to approximately 1 kcal/kg/day.

† Numbers in parentheses, standard deviation.

cholesterol$_{males}$, 0.51; and for high density lipoprotein cholesterol$_{females}$, 0.65. For the sum of four skinfolds, a coefficient of 0.63 was found. The stability coefficients for blood pressure (0.43 for systolic blood pressure and 0.34 for diastolic blood pressure) and for cardiopulmonary fitness (0.31) were more or less of the same magnitude as the coefficients for the lifestyle coronary heart disease risk factors. For the dietary intake variables, the following stability coefficients were found: fat intake, 0.42; carbohydrate intake, 0.37; cholesterol intake, 0.34; and the ratio between the intakes of polyunsaturated and saturated fatty acids, 0.33. For daily physical activity, the stability coefficient was 0.34.

Besides the calculation of the stability coefficients, tracking was assessed for subjects at risk (see table 1). For all biologic and lifestyle risk factors, the number of subjects at risk at the initial measurement in 1977 at the age of 13 years is given in table 4. For most of the biologic risk factors, 10–15 percent of the subjects were at risk at age 13, except for systolic blood pressure (38.5 percent). For the lifestyle parameters (i.e., dietary intake), the percentage of subjects at risk at age 13 was much higher (80–85 percent). Only for cholesterol intake was the percentage of subjects at risk at age 13 somewhat lower (29.6 percent). Because for cardiopulmonary fitness and daily physical activity the subjects were divided into percentile groups, by definition the percentage of subjects "at risk" at age 13 was around 25 percent. The tracking coefficients (odds ratios) for subjects at risk at the initial measurement at age 13 who maintained their risk status over a period of 14 years are also given in table 4. The highest odds ratios were found for the lipoproteins and for the sum of four skinfolds. For the other biologic coronary heart disease risk factors (blood pressure and cardiopulmonary fitness), these tracking coefficients were much lower. For the lifestyle risk factors, the tracking coefficients were even lower. In none of the tracking analysis for subjects at risk was a significant interaction found between the initial value of the risk factor and sex; consequently, all tracking coefficients (odds ratios) are equal for males and females.

For smoking behavior and alcohol consumption, initial subjects at risk (smokers/drinkers) were not defined at the initial measurement at the age of 13 years but over the whole adolescent period between 13 and 16 years of age (table 5). Odds ratios were calculated for these subjects in order to maintain their risk status over the time period from 16 to 27 years of age (including two measurements at 21 and 27 years of age). Table 5 shows the results of these analyses. The odds ratio for initial smokers was 8.3 and for initial drinkers, 2.9.

DISCUSSION

To assess the stability of certain variables in time or to assess the predictability of early measurements of a certain variable for values of the same variable later in life, we found the calculation of tracking coefficients to be an important topic in longitudinal epidemiologic studies. However, most "traditional" methods of calculating tracking coefficients are limited (9). In this

TABLE 4. Number of subjects at risk at the initial measurement at 13 years of age and tracking coefficients for biologic and lifestyle coronary heart disease risk factors, calculated with generalized estimating equations over a period of 15 years from 1977, Amsterdam Growth and Health Study

	No. of subjects at risk	OR*	95% CI*
Biologic risk factors			
Total cholesterol (TC)	23 (12.7)†	10.4	5.0–21.7
High density lipoprotein cholesterol (HDL cholesterol)	23 (12.7)	14.1	7.2–27.5
TC/HDL cholesterol ratio	20 (11.0)	22.9	10.6–49.4
Systolic blood pressure	69 (38.5)	4.0	2.5–6.5
Diastolic blood pressure	26 (14.5)	4.8	2.5–9.4
Sum of four skinfolds	13 (15.9)	17.7	9.2–34.1
Cardiopulmonary fitness	44 (24.6)	4.4	2.6–7.4
Lifestyle risk factors			
Fat intake	158 (88.3)	2.6	1.1–6.0
P/S ratio*	147 (82.1)	1.9	1.1–3.3
Carbohydrate intake	140 (78.2)	3.4	1.9–6.1
Cholesterol intake	53 (29.6)	2.3	1.5–3.6
Daily physical activity	46 (25.8)	3.6	2.4–5.4

* OR, odds ratio; CI, confidence interval; P/S ratio, ratio between the intake of polyunsaturated fatty acids and the intake of saturated fatty acids.
† Numbers in parentheses, percentage.

TABLE 5. Number of subjects at risk at the age of 13–16 years and tracking coefficients for smoking behavior and alcohol consumption, Amsterdam Growth and Health Study, 1977–1980

	No. of subjects at risk	OR*	95% CI*
Smoking behavior	48 (26.5)†	8.3	4.3–16.1
Alcohol consumption	119 (65.7)	2.9	1.6–5.0

* OR, odds ratio; CI, confidence interval.
† Numbers in parentheses, percentage.

paper, tracking coefficients were assessed by using a new method, which overcomes most of the limitations of earlier "traditional" methods (10, 11).

One of the major problems in tracking analysis is the interpretation of the results. Generally, conclusions about the tracking phenomenon are based on the significance of either the stability coefficients or the tracking coefficient for subjects at risk (9). Looking only at the significance of coefficients, however, does not say anything about the strength of the relation and does not give an answer to the basic question behind tracking: Is it worthwhile to screen subjects at an early age for coronary heart disease risk factors? In other words, the magnitude of the stability coefficient or the magnitude of the odds ratio for subjects at risk gives the most important information about the tracking phenomenon. Some authors define certain threshold values above which a particular variable is supposed to track (9). However, this strict rule does not take into account that the magnitude of the tracking coefficient greatly depends on the initial age of the subjects and on the length of the time period. In evaluating tracking coefficients, we found that the magnitude of the point estimate, the width of the 95 percent confidence interval of the coefficient (which gives information about the reliability of the estimate), and the age period over which the coefficient is calculated must be considered. The unique feature of the present study is that tracking coefficients of both biologic and lifestyle coronary heart disease risk factors were calculated over the same time period, and with the same method, which gives the possibility of comparing the tracking coefficients for different coronary heart disease risk factors with each other.

If the conclusion about whether or not tracking exists depends on the significance of the stability coefficients, then in our studies all coronary heart disease risk factors seem to track. However, if the magnitude of the stability coefficients is taken into account, then the results show that the coefficients for the lipoproteins and body fatness were much higher than the coefficients for blood pressure, cardiopulmonary fitness, and all the lifestyle coronary heart disease risk factors.

Then the question arises of whether or not it is worthwhile to screen subjects at an early age for subjects at risk for coronary heart disease. To give an answer to that question, we calculated odds ratios for subjects at risk for all biologic and lifestyle coronary heart disease risk factors. In the present study, risk groups were predominantly defined according to objective risk values, while in many other studies, risk groups were arbitrarily defined, based on the distribution of values (high risk quartiles, quintiles, or deciles). The latter should be avoided, because the results of the tracking analysis greatly depend on the arbitrary choice for the division of subgroups (9). Defining risk groups according to the distribution of values is suitable only in situations where no objective risk values are available. There remains the question of whether it is worthwhile to screen subjects at an early age.

Suppose tracking is calculated for subjects in a particular risk quartile in a longitudinal study with two measurements in time. When 50 percent of the subjects in the high risk quartile at the initial measurement maintain their position in the high risk quartile at the follow-up measurement, the initial measurement (i.e., "the potential screening test") had a predictive value of 50 percent. In this particular situation, an odds ratio of 5.0 would be found for subjects in the high risk quartile at the initial measurement in order to maintain their position in the high risk quartile at the follow-up measurement. If this is considered the other way around, an odds ratio of 5.0 calculated for risk quartiles translates to a predictive value of the initial measurement of 50 percent. This holds only for situations in which 25 percent of the subjects (risk quartiles) are identified as the risk group. When a lower percentage of the subjects is identified as the risk group (which is the case in most of the coronary heart disease risk factors presented in this study), an odds ratio of 5.0 translates to even lower predictive values. In our study, odds ratios cannot be transformed into predictive values of a potential screening test because we used all available longitudinal data in the analysis. However, it is obvious that odds ratios less than 5.0 indicate that the predictive value of a potential screening test is rather low. So, for the tracking of subjects at risk, the odds ratios for only the lipoproteins and body fatness are high.

Tracking for smoking behavior and alcohol consumption cannot be compared directly with the other coronary heart disease risk factors, because a different approach was used. However, the results reveal that tracking from adolescence into young adulthood exists for smoking behavior but not for alcohol consumption.

It is difficult to compare tracking coefficients calculated in different studies with each other, not only because the time period under consideration differs among studies but also because tracking coefficients are calculated with different methods. In other longitudinal studies, stability coefficients were usually calculated as Pearson's correlation coefficients, while tracking for subjects at risk was usually estimated by the percentage of subjects from a certain group at the initial measurement who maintained their position in that risk group at a follow-up measurement.

There are a few longitudinal studies in which tracking for coronary heart disease risk factors was assessed over more or less the same age period over which the Amsterdam Growth and Health Study was carried out. In the Bogalusa Heart Study (1, 36–40), tracking was assessed for most of the biologic coronary heart disease risk factors. For both stability coefficients as the tracking for subjects at risk, the general impression is comparable with the results from the Amsterdam Growth and Health Study. For total serum cholesterol, higher coefficients were found than for systolic blood pressure and diastolic blood pressure, while the coefficients for systolic blood pressure were higher than the ones for diastolic blood pressure. For body fatness (operationalized as triceps skinfold thickness), the stability coefficient was of the same magnitude as the total serum cholesterol coefficient. In addition, in the Muscatine Study (3, 41–43), tracking was assessed for lipoproteins, blood pressure, and body fatness. The latter, however, was operationalized as body mass index instead of skinfold thickness. Nevertheless, comparable results were found. In the Fels longitudinal study (44–46), a different approach was used to calculate tracking coefficients for the lipoproteins and for blood pressure. With this approach, all available longitudinal data were used to assess tracking. Moreover, they calculated stability coefficients for both total cholesterol and high density lipoprotein cholesterol. These latter coefficients were lower than the ones reported for total serum cholesterol. With this approach also, the other results were comparable with the results of the Amsterdam Growth and Health Study. With regard to tracking analysis for cardiopulmonary fitness, limited results are available. Beunen et al. (5) calculated tracking coefficients for cardiopulmonary fitness (operationalized by a step test) with subjects with an initial age of 13 years over a period of 18 years. Based on a tracking coefficient of 0.26, they concluded moderate tracking for cardiopulmonary fitness. In the Cardiovascular Risk in Young Finns Study (4, 7), tracking coefficients were calculated for not only biologic coronary heart disease risk factors but also daily physical activity. Over a period of 6 years, with subjects initially aged 12 years, a stability coefficient of 0.18 was calculated for physical activity. With subjects initially aged 15 years, the coefficient of 0.27 was a bit higher. Kelder et al. (8) also analyzed tracking of daily physical activity in adolescents. They did not calculate a coefficient, but they compared different quintiles in a profile analysis over a period of 6 years. They concluded that tracking exists for daily physical activity, because a significant trend was found. In the same study, a significant trend for food choice behavior was also found. Accordingly, tracking of real nutrient intake information only is available in younger (47, 48) or older age groups (49–51). In general, it seems that tracking of nutrient intake becomes better as the initial age of the subjects increases.

Regarding tracking of smoking behavior, Flay et al. (52) calculated the risk for smoking adolescents versus nonsmoking adolescents whose smoking status remained unchanged 7 years later. They found an odds ratio of 7.1 (95 percent confidence interval (CI) 2.2–22.4). Oygard et al. (53) found comparable results over a period of 10 years, while Stanton et al. (54) calculated a lower risk (odds ratio = 4.2, 95 percent CI 2.5–7.0) for smokers with an initial age of 13 years over a period of 2 years. In young adults, these risks seem to be much higher (55). With data from the National Longitudinal Survey, Harford (6) calculated the risk for drinkers with an initial age of 17–24 years still drinking 6 years later. In a population with more than 10,000 subjects, an odds ratio of 5.5 (95 percent CI 5.0–6.1) was found. For older subjects (51, 56), relatively high stability coefficients of around 0.70 were found for alcohol consumption.

Looking at the literature, one is surprised by the fact that, although the magnitude of the tracking coefficients differs markedly among the different risk factors, almost all studies conclude that tracking exists for both biologic and lifestyle coronary heart disease risk factors. In addition, though it is difficult to decide whether or not tracking exists for a certain variable, according to the presented results of the Amsterdam Growth and Health Study and according to a more realistic interpretation of the results of other studies, tracking seems to exist only for the lipoproteins and for body fatness. For the other biologic coronary heart disease risk factors and all lifestyle coronary heart disease risk factors (except for smoking behavior), both the stability coefficients and tracking coefficients for subjects at risk are too low to conclude that tracking exists for these variables. Another point of interest is the fact that the presented tracking analysis focuses on the longitudinal development of coronary heart disease risk factors but not on the actual risk of getting coronary heart disease. However, before a decision

can be made as to whether it is worthwhile to screen subjects at an early age for a particular risk factor, the causal role of that risk factor in the development of coronary heart disease has to be established. In fact, this should not be limited to coronary heart disease, but the general health consequences of a particular risk factor in a certain time frame should be considered. A combination of both "risks" (the risk calculated by tracking analysis and the general health consequences) has to be taken into account to decide whether or not screening at early ages should be carried out. Furthermore, ethical and economical aspects should be taken into account before deciding whether or not it is worthwhile to screen subjects at an early age for a particular risk factor or, in other words, whether or not it is worthwhile to initiate preventive interventions for that particular risk factor. Based on all this, smoking behavior and body fatness seem to be the prime candidates for preventive strategies at an early age. Screening and intervention for the lipoproteins (the other coronary heart disease risk factors with reasonable tracking) seem to be less obvious candidates (57).

The relatively low tracking coefficients for the lifestyle coronary heart disease risk factors can also be interpreted in another way. In preventive medicine, a lot of attention is paid to moving toward a healthy lifestyle at an early age. The results of the tracking analysis, however, reveal that the adolescent lifestyle (except for smoking) is hardly predictive of the adult lifestyle. This can have important implications for preventive intervention strategies to improve the lifestyle in younger age groups. It is questionable whether or not possible changes in lifestyle due to intervention programs during adolescence endure over time.

It can be concluded that, over a period of 14 years from adolescence to young adulthood, both stability coefficients and tracking for subjects at risk for lifestyle coronary heart disease risk factors were low, which indicates low stability over the time period under consideration and low predictability of early measurements for values later in life. Only smoking behavior tracking for subjects at risk was rather high. For the biologic risk factors cardiopulmonary fitness and blood pressure, tracking was also low, but for the lipoproteins and body fatness, tracking was much better, indicating relatively high stability during adolescence and young adulthood and relatively good predictability of measurements early in life. Although it is questionable whether or not it is worthwhile to screen subjects at an early age for these coronary heart disease risk factors, smoking behavior and body fatness seem to be the major candidates for possible early interventions.

ACKNOWLEDGMENTS

This study was supported by the Dutch Heart Foundation (grants 76051–79051 and 90–312), the Dutch Prevention Fund (grants 28–189[a], 28–1106, and 28–1106–1), the Dutch Ministry of Well Being and Public Health (grant 90–170), and the Dairy Foundation of Nutrition and Health.

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(Appendix follows)

APPENDIX

To calculate the stability coefficients, the following statistical model, which is comparable to a simple regression model, was used:

$$Y_{it} = \beta_0 + \beta_1 Y_{it_1} + \beta_2 t + \sum_{j=1}^{J} \beta_{3j} X_{itj} + \sum_{k=1}^{K} \beta_{4k} Z_{ik} + \varepsilon_{it}, \quad \text{(A1)}$$

where Y_{it} = observations of individual i from t_2 to t_m (where m = the number of measurements); β_0 = intercept; Y_{it_1} = initial (first) observation of individual i at t_1; β_1 = standardized regression coefficient used as tracking coefficient; t = time; β_2 = regression coefficient of time; X_{itj} = time-dependent covariate j of individual i; β_{3j} = regression coefficient of time-dependent covariate j; J = number of time-dependent covariates; Z_{ik} = time-independent covariate k of individual i; β_{4k} = regression coefficient of time-independent covariate k; K = number of time-independent covariates; and ε_{it} = measurement error of individual i.

To calculate a tracking coefficient for a certain risk factor, one must regress the value of the initial measurement at t_1 (Y_{it_1}) on the entire longitudinal development of that risk factor from t_2 till t_6. The relations between the initial value at t_1 and the values from t_2 till t_6 are tested simultaneously, leading to one standardized regression coefficient (β_1), which can be interpreted as a longitudinal correlation coefficient, i.e., the stability coefficient (10). Although this stability coefficient can range between -1 and 1, assuming the correlations between the repeated observations to be positive, this stability coefficient takes values between 0 and 1. One of the biggest advantages of this model is the fact that all longitudinal data are used to calculate the stability coefficient. Besides that, another advantage of the method is that it can be used in longitudinal studies in which the points of measurements are not equally spaced. A third advantage is the possibility to correct for both time-dependent (X_{itj}) and time-independent covariates (Z_{ik}). These covariates can be continuous as well as discrete. The most important time-dependent covariate is time itself. The most important time-independent covariates to account for are sex and initial (biologic) age. By estimating the parameters of the models, one encounters the problem that the repeated observations of the same individual are not independent of each other. Therefore, a statistical technique is used in which these intrasubject correlations are taken into account. This approach is called generalized estimating equations and is developed by Zeger and Liang (25). The advantage of generalized estimating equations is that the parameters of the model are estimated, adjusting for these within-subject correlations, which were treated as nuisance parameters. This is done by assuming a priori a certain correlation structure for the repeated measurements of the dependent variable (Y_{it}). The correlation structure can be chosen from a set of available structures: 1) independent (all correlations are zero), 2) exchangeable (all correlations are the same), and 3) m-dependent (correlations k occasions apart are the same for $k = 1, \ldots, m$, whereas correlations more than m occasions apart are zero). One of the features of generalized estimating equations is that the method is robust in the estimation of the parameters with respect to the choice of a certain correlation structure. This means that, irrespective of the chosen correlation structure, the point estimates of the model parameters are almost the same. The choice for a certain correlation structure has to depend on the correlation structure of the actual data. According to the data of the Amsterdam Growth and Health Study, an independent correlation structure seemed to be less suitable, because the interperiod correlation coefficients for the coronary heart disease risk factors were far from zero. An exchangeable correlation structure was also not suitable. Because the Amsterdam Growth and Health Study was designed with irregularly spaced time periods, the correlation coefficients calculated between measurements of the same risk factor one period apart are not equal. Based on the data, therefore, a stationary 5-dependent correlation structure was chosen to be the most appropriate.

Another advantage of using generalized estimating equations to calculate the tracking coefficients is that the method can be used not only for the analysis of continuous variables but also for the analysis of discrete (dichotomous) variables (25, 26), or in other words, to calculate tracking for subjects at risk. To assess tracking for subjects at risk, we used the following statistical model, which is comparable to a simple logistic model:

$$\Pr(Y_{it} = 1) = \frac{1}{1 + \exp^{-(\beta_0 + \beta_1 Y_{it_1} + \beta_2 t + \sum_{j=1}^{J} \beta_{3j} X_{itj} + \sum_{k=1}^{K} \beta_{4k} Z_{ik} + \epsilon_{it})}}, \quad \text{(A2)}$$

or after a logit transformation:

$$\text{logit}[\Pr(Y_{it} = 1)] = \beta_0 + \beta_1 Y_{it_1} + \beta_2 t + \sum_{j=1}^{J} \beta_{3j} X_{itj} + \sum_{k=1}^{K} \beta_{4k} Z_{ik} + \epsilon_{it}, \quad \text{(A3)}$$

where $\Pr(Y_{it} = 1)$ = the probability that the observations at t_2 to t_m of subject i equal 1 (where m = the number of measurements, and 1 means that subject i belongs to the defined risk group); Y_{it_1} = the initial (first) observation of subject i at t_1; β_0 = intercept; β_1 = regression coefficient; t = time; β_2 = regression coefficient of time; X_{itj} = time-dependent covariate j of subject i; β_{3j} = regression coefficient of time-dependent covariate j; J = the number of time-dependent covariates; Z_{ik} = the time-independent covariate k of subject i; β_{4k} = regression coefficient of time-dependent covariate k; K = the number of time-independent covariates; and ε_{it} = measurement error of subject i at time t.

In this model, the probability of belonging to a risk group from t_2 till t_6 depends first on the initial membership of the risk group at t_1 (Y_{it_1}). Furthermore, the model has the same advantages as has been mentioned for the model used to calculate the stability coefficients (equation A1). The coefficient of interest is β_1, because this coefficient reflects the relation between belonging to a risk group at t_1 and the development of that particular risk group from t_2 till t_6, which is in fact the definition of tracking. As in simple logistic regression, this coefficient (β_1) can be transformed into an odds ratio (e^{β_1}), which gives the magnitude of the risk of a subject belonging to a risk group at t_1, regarding the development of the subject's risk status from t_2 till t_6, relative to the risk of a subject not belonging to that risk group at t_1 (11, 26). As mentioned before, the parameters of the above statistical model were also estimated with generalized estimating equations. In designs where $m = 2$, the calculated odds ratios with the generalized estimating equations approach are exactly the same as they were calculated with multiple logistic regression. The only difference between the longitudinal generalized estimating equations approach and multiple logistic regression is that, with generalized estimated equations all available longitudinal data were used to calculate the risk, i.e., to calculate the magnitude of the tracking and that, with multiple logistic regression, only two points in time can be used. As mentioned before, the choice for a certain correlation structure has to depend on the correlations of the actual data. Because it is impossible to calculate these correlations for the dichotomous variables, the same correlation structure was chosen that has been used for the calculation of the stability coefficients, i.e., a stationary 5-dependent structure.