A Longitudinal Causal Relationship among Cardiovascular Risk Factors in the Employees of the Government Savings Bank

PARADEE TEMCHAROEN, Dr.P.H.*,
THAVATCHAI VORAPONGSATHORN, Ph.D.**,
MANDHANA PRADIPASEN, M.D., Dr.P.H.*,
PIYAMITR SRITARA, M.D.***

Abstract

A longitudinal structural causal model was generated to examine the causal relationship among determinants which were composed of four personal factors, stress, four health behaviors, and BMI on five physiological risks for cardiovascular disease: SBP, DBP, FBS, TC and HDL-C in 1,019 bank employees, within a five-year interval. A fourteen-item food frequency questionnaire for assessing eating habits and the Health Opinion Survey for the stress test were included in the self-administered questionnaires. Weight, height and blood pressure were measured and blood samples were collected for blood chemical analysis.

Data analysis by LISREL showed that the determinants in the proposed model explained as much as 96 per cent variation in physiological risks for CVD (R^2 = 0.96, relative chi-square = 1.92, RMSEA = 0.03, GFI = 0.96 and AGFI = 0.95). The findings also indicated that current physiological status was affected by their status of age, education, health behaviors, BMI and physiological status 5 years ago.

Key word : Cardiovascular Disease, Cardiovascular Risk Factors, LISREL, Bank Employee, Longitudinal Study

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* Department of Nutrition,
** Department of Biostatistics, Faculty of Public Health, Mahidol University,
*** Cardiology Unit, Department of Internal Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand.

The mortality rate from cardiovascular disease (CVD) has increased rapidly and become the major leading cause of death in most developing countries including Thailand[1,2]. The increasing large number of premature deaths has led to extensive research into prevention. Epidemiologic researches have sought to discover the factors and lifestyle behaviors associated with development of CVD known as "risk factors". Categories of risk factors were commonly classified in relation to the strategy of intervention into two main groups: modifiable and non-modifiable risk factors[3]. The categories of factors according to mechanism was also proposed[4]. One was the causal risk factor, which had evidence supporting a direct causal role. The others were conditional risk factors consisting of factors associated with an increased risk for CVD and predisposing risk factors. Physiological risk factors categorized as a major causal risk for CVD directly contributing to diseases and death, include hypertension, elevated serum cholesterol or low density lipoprotein cholesterol (LDL-C), low level of high density lipoprotein cholesterol (HDL-C) and diabetes mellitus (DM)[3-5]. These modifiable factors are both independent or combination risks for CVD, and a combination of risk factors increase the risk of CVD[3].

An abundance of previous studies both cohort and retrospective have identified the risk factors for CVD based on several CVD endpoints as diseases or death. These findings may not be appropriate for designing an early prevention intervention program. Therefore, identification of risk factors for CVD based on physiological risk endpoints instead of diseases or death endpoints could help to develop early primary prevention of CVD. In addition, no previous multivariate analyses of causal relationship among categories of risk factors for CVD using a combination of three or more physiological risks for CVD examined have appeared in the published literature. Therefore, this study aimed to generate a longitudinal structural equation modeling (SEM) to examine the causal relationship among determinants of physiological risks for CVD. Also, changes in some determinants and their causal relationship within a five-year interval were investigated. The significant determinants included personal factors (age, gender, education and position), stress, health behaviors (cigarette smoking, alcohol intake, physical activity and eating habits), and body mass index (BMI) in the employees of the Government Savings Bank within a five-year interval, 1995 and 2000. Physiological risks for CVD in this study were composed of blood pressure both systolic blood pressure (SBP) and diastolic blood pressure (DBP), fasting blood sugar (FBS), total cholesterol (TC) and HDL-C. Moreover, advanced statistical technique of a SEM was used to explain both the direct and indirect effects of the determinants on the physiological risks for CVD[6].

MATERIAL AND METHOD

Population and sample

A five-year follow-up study was performed with 1,860 employees of the Government Savings Bank who had had a complete physical check up in 1995 and a repeated examination in the year 2000. Sample selection criteria included; 1) males and females aged 20 years and over; 2) having complete physical examination data for both 1995 and 2000; and 3) having completed self-administered questionnaires composed of all studied variables of both years. Criteria for exclusion included; 1) female participants who were pregnant or had had a child within 6 months; 2) female participants who were taking oral contraceptive pills or estrogen replacement therapy for menopause; 3) participants who were taking medication for treatment of hypertension, diabetes mellitus or hyperlipidemia; 4) participants who had a history of liver disease, renal disease, or disease of the thyroid; and 5) participants who had had an illness that led to severe weight loss within the past 6 months.

The sample size was determined by two criteria: first, the representative sample size that can be generalized in other groups of population using Daniel's formula[7]. Second, for testing the model, the sample size was estimated by using 20 samples for one parameter[8]. At least 880 samples were needed. There were 1,223 employees who had a repeated physical check up in 2000 and 1,019 samples who met the criteria. Therefore, the researchers decided to include all 1,019 eligible samples.

Research instruments and measures

Instruments used for data collection in 1995 were employed. They included four parts of the self-administered questionnaires; a) general information consisting of details of gender, age, religion, marital status, educational levels, salary, position and family history; b) health information and health behavior including smoking habits, alcohol consumption habits, physical activity composed of work characteristics and exercise habits; c) thirty two-items of the food

frequency questionnaire used in 1995 were reviewed and total item correlation was performed. Fourteen food items were selected forming four food groups, 5 items for high fat and cholesterol, 3 items for high fat and calories, 2 for high sugar and 4 for high sodium. The frequency-of-use response describes frequency as usually eaten more than 3 times per week or three times or less per week. Scoring of 2 points was assigned for food items usually eaten more than 3 times per week and 1 point for food items usually eaten less than 3 times per week. The total score was 14-28 points. The fourth part of the self-administered questionnaire was d) the stress assessment test using the Health Opinion Survey (HOS)[9] consisting of 20 items of questions concerning physical reaction to stress. The choices in the answer to the questions had three scales as "often", "sometimes", "hardly ever or never". Scoring was accomplished by giving points of 3, 2 and 1 for "often", "sometimes" and "hardly ever or never" on the 18 negative statement items. On the other 2 positive statements, 1, 2 and 3 points were assigned for "often", "sometimes" and "hardly ever or never". The total score of HOS was 20-60 points. The reliability of the instruments analyzed by Cronbanch's alpha was acceptably high as 0.80 in 1995 and 0.83 in 2000. Self-administered questionnaires were provided to subjects in advance and were returned to the researcher on the physical examination day. Incomplete questionnaires were sent back to participants to be completed. The anthropometrical measurement data were collected by a Detecto beam balance scale for measuring body weight and Microtoise for measuring body height. Body weight was measured by subjects in the center of the weighing platform. Shoes were removed and minimum clothing worn. Height was measured without shoes in a straight position. A subject stood on the flat floor by the scale with feet parallel, heals, buttocks, shoulders and the back of the head touching the wall. They were measured to an accuracy of 0.1 kilogram for weight and 0.1 centimeter for height. BMI was calculated by dividing body weight in kilograms by the square of height in meters. Blood pressure was measured on the left arm by using a mercury manometer with the subject resting for 5 minutes. A second measurement was taken for participants who had high blood pressure after 30 minutes rest. Venous blood was obtained from each subject in the morning after at least 10 hours of overnight fasting for the determination of FBS level, serum concentration of TC level, and level of HDL-C. Blood analysis was performed as follows: a) serum total cholesterol was determined by the Enzymatic method using Reagent Kits of Human Ltd; b) serum HDL-C was analyzed by precipitating with precipitant, the supernatant obtained was assayed for cholesterol with the Reagent Kits of Human Ltd.; and c) fasting blood sugar was determined by the Enzymatic method using the Reagent Kits of Human Ltd.

All measurement variables used for the formulating model were transformed into a measurement scale. Scores ranged from 1 (masters degree or higher) to 6 (primary school) on educational levels, from 1 (executive administrator) to 5 (service worker) on position, from 1 (no smoking) to 5 (smoked > 20 cigarettes per day) on smoking habits and from 2 (labor work with regular exercise ≥ 3 days/week for ≥ 30 minutes/time) to 8 (sedentary work sitting > 5 hours with no exercise) on physical activity. Regarding difference in unit of measurement, therefore, risk scores of SBP, DBP, FBS, TC and HDL-C were assigned using the scoring risk system proposed by Grundy[4] and Wilson et al[10]. Total score of eating habits and HOS score, age in months, an average amount of alcohol intake in grams per day and actual BMI level were used for data analysis. The higher scores indicating a higher risk for CVD.

Data analysis

Descriptive statistics and cross-tabulations were used to describe studied variables classified by sex. Paired *t*-test was used to compare changes of studied variables within the five-year interval. SEM was conducted to examine the parameters of the hypothesized causal model for the variables[11]. The model was tested through the maximum likelihood (ML) procedure. The analysis program used SPSS, Version 9.0 and LISREL 8.30. Statistical assumptions were tested to assess violation of both univariate and multivariate assumptions using PRELIS 2.30.

The causal model development and testing

The hypothesized longitudinal model to explain the causal relationship among personal factors, stress, health behavior, and BMI on physiological risks for CVD is shown in Fig. 1. The subscript number 1 and 2 stand for the years 1995 and 2000 respectively. This hypothesized model was composed of 9 exogenous variables: gender, age (Age1, Age2), education (Ed1, Ed2), position rank (Pt1, Pt2), and HOS (HOS1, HOS2), two endogenous observed variables: BMI1, BMI2 and four endogenous latent vari-

ables: health behaviors (HB1, HB2) and physiological risks for CVD (PRCVD1, PRCVD2). Health behaviors (HB1, HB2) were a combination of 8 observed indicators: eating habits (EH1, EH2), cigarette smoking (Smo1, Smo2), alcohol intake (Alc1, Alc2) and physical activity (PA1, PA2). Physiological risks for CVD (PRCVD1, PRCVD2) were a combination of 10 observed indicators: SBP risk score (SBPrs1, SBPrs2), DBP risk score (DBPrs1, DBPrs2), FBS risk score (FBSrs1, FBSrs2), TC risk score (TCrs1, TCrs2) and HDL-C risk score (HDLrs1, HDLrs2).

General assumptions of SEM were tested. Most studied variables except Smo1, Smo2, Alc1, Alc2, FBSrs1, FBSrs2 and HDLrs2 (skew close to 0 and kurtosis close to 0) met the assumption of normal distribution. All predictive variables had a tolerance value less than 0.10 indicating no multicollinearity. SEM tests two models simultaneously: a measurement model and a structural for the theoretical model. Multiple fit indices (non-significant of chi-square or relative chi-square < 2, RMSEA < 0.05, GFI > 0.9 and AGFI > 0.9) were used to test the model fit[12]. The outputs of testing the measurement model found that the measurement models for HB2 and PRCVD1 did not quite fit the data, which might be due to non-normal distribution of some indicators. However, modification of the measurement indicators was limited because using the same measurement instruments was necessary for a longitudinal study. Therefore, it was decided to include all indicators in the model. The hypothesized causal model was tested, the good fit model which could better explain the causal relationship among studied variables was selected. The model without gender was selected since gender showed a strong direct effect on health behavior ($\beta = 0.76$, $p < 0.001$). This influence led to suppress the influence of health behavior on BMI and PRCVD.

RESULTS

The 1,019 participants were composed of 249 males and 770 females (Table 1). Most of them were in the young-middle aged group, had completed a bachelor degree and worked as officers (level 4-7). The educational level and position were significantly increased after five years. This population group generally had a low level of stress and had not changed within the five-year interval. Cigarette smoking and alcohol intake were commonly found in males and had not significantly changed after five years. Exercise and eating habits had changed signi-

Table 1. Percentage of general characteristics, stress and health behaviors of participants by gender in 1995 and 2000.

Variable	1995			2000		
	Male (n=249)	Female (n=770)	Total (n=1019)	Male (n=249)	Female (n=770)	Total (n=1019)
Personal factors						
Age (year)						
<40	67.9	71.4	70.6	49.0	51.2	50.6
40-49	19.3	21.8	21.2	24.9	33.0	31.0
50-59	12.9	6.8	8.2	24.1	15.2	17.4
≥60	0.0	0.0	0.0	2.0	0.6	1.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	37.1 ± 9.3	36.1 ± 7.7	36.3 ± 8.1	42.2 ± 9.3	41.2 ± 7.7	41.5 ± 8.1
Education						
Elementary	5.2	1.2	2.2	5.2	1.0	2.1
Secondary	5.6	2.2	3.0	3.6	1.7	2.2
High school	15.7	16.2	16.1	13.7	12.1	12.5
Certificate or Diploma	17.3	10.3	12.0	6.0	7.0	6.8
Bachelor degree	51.0	64.8	61.4	61.4	66.9	65.5
≥Master degree	5.2	5.3	5.3	10.0	11.3	11.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = 14.29, p = 0.000			

Table 1. Percentage of general characteristics, stress and health behaviors of participants by gender in 1995 and 2000 (Continue).

Variable	1995			2000		
	Male (n=249)	Female (n=770)	Total (n=1019)	Male (n=249)	Female (n=770)	Total (n=1019)
Position						
Executive administrator	1.6	0.5	0.8	6.8	2.1	3.2
Administrator	18.1	10.3	12.2	20.1	19.4	19.5
Officers						
Level 4-7	37.3	65.3	58.5	52.2	69.0	64.9
Level 1-3	31.7	22.1	24.4	12.0	7.9	8.9
Service worker	11.2	1.8	4.2	8.8	1.7	3.4
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = -20.38, p = 0.000			
Stress (HOS score)						
<40	95.6	94.0	94.0	97.2	90.7	93.0
≥40	4.4	6.0	6.0	2.8	9.3	7.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	30.1 ± 5.1	31.3 ± 5.1	31.0 ± 5.1	29.6 ± 4.7	31.1 ± 5.4	30.8 ± 5.3
			t = 1.50, p = 0.133			
Health behaviors						
Smoking						
Did not smoke	56.2	98.7	88.3	55.4	98.4	87.9
Used to smoke	18.5	0.8	5.1	24.5	1.0	6.8
Smoked (cigarettes per day)						
<10	19.7	0.5	5.2	14.9	0.5	4.0
10-19	5.2	0.0	1.3	4.8	0.0	1.2
≥20	0.4	0.0	0.1	0.4	0.0	0.1
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = 1.21, p = 0.225			
Drinking alcohol						
No	59.4	96.0	87.1	57.8	94.5	85.6
Yes (g/d)						
<15	16.4	3.4	6.7	11.6	4.5	8.1
15-29	10.8	0.5	3.1	9.7	0.8	2.7
≥30	13.3	0.1	3.1	20.9	0.1	3.5
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = 1.12, p = 0.26			
Physical activity score (2-8)						
2-4	12.0	2.6	4.9	17.2	5.8	8.7
5-8	88.0	97.4	95.1	92.8	94.2	91.3
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = 5.51, p = <0.001			
Eating habits score (14 - 28)						
<21	76.3	75.7	73.4	96.0	98.0	98.2
≥21	23.7	24.3	26.6	4.0	2.0	1.8
Total	100.0	100.0	100.0	100.0	100.0	100.0
			t = 42.42, p = 0.001			

ficantly for the better after five years. Table 2 shows that BMI ≥ 25.0 kg/m^2 had increased from 15.7 per cent in 1995 to 24.0 per cent in 2000. There was an increase in hypertension as defined by SBP > 140 mmHg and/or DBP > 90 mmHg from 16.8 per cent in 1995 to 38.7 per cent in 2000. There were also increases in all blood chemistries within the five-year interval especially total cholesterol > 200 mg/dl which had rapidly increased from 34.3 per cent to 61.7 per cent.

Results from the SEM tests (Fig. 1) showed that health behaviors were mainly explained by smoking habits and alcohol intake. Eating habits and physical activity were weak indicators of measurement model for health behavior among this population group. HB1 was determined only by education. The positive direct effect of education (Ed1) on health behaviors (HB1) (β = 0.22) revealed that participants who completed a higher educational level had lower health behavior risks for CVD than participants who completed a lower educational level. The opposite direction of Ed2 on HB2 (β = -0.03) could be explained that an increase in educational level within five years may not correlate with improvement of their health behavior. Moreover, HB2 had a strong indirect effect of their previous education (Ed1) (Table 3). This result clearly showed that an increase in formal education after five years did not lead to an increase in better health behavior. Even though there was a marked improvement in eating and exercise behavior, the 0.99 correlation between HB1 and HB2 signifies that the improvement occurred only among those who already had better behavior 5 years ago. In addition, the negative factor loading of physical activity in HB1 and HB2 means that participants who had higher physical activity usually had poorer eating habits, more smoking and drinking of alcohol.

The findings showed that SBP and DBP were strong indicators of measurement for PRCVD1. Five years later, FBS, TC and HDL-C became more important indicators of measurement for PRCVD (Fig. 1) whereas SBP and DBP remained high. The 0.92 correlation between PRCVD1 and PRCVD2 (Table 3) illustrated that more than 80 per cent of PRCVD2 was determined by a person's health status 5 years ago, the rest was by HB, BMI, Ed and age at the initial exam. BMI2 was the only current factor significantly determining about 2.6 per cent of PRCVD2 (β = 0.16).

Table 2. Percentage of BMI, BP and lipid profiles of participants by gender in 1995 and 2000.

Variable	1995			2000		
	Male (n=249)	Female (n=770)	Total (n=1019)	Male (n=249)	Female (n=770)	Total (n=1019)
BMI (kg/m^2)						
<18.5	3.6	11.5	9.6	0.8	6.5	5.1
18.5-24.9	75.9	74.4	74.8	63.9	73.1	70.9
25.0-29.9	18.9	13.0	14.4	31.3	17.1	20.6
≥30.0	1.6	1.2	1.3	4.0	3.2	3.4
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	23.1 ± 2.8	21.6 ± 3.0	22.0 ± 3.1	24.2 ± 2.9	22.5 ± 3.4	22.9 ± 3.3
			t = -20.60, p = 0.000			
SBP (mmHg)						
<120	39.4	76.1	67.1	18.1	48.6	41.1
120-129	37.3	16.4	21.5	27.3	22.1	23.4
130-139	18.1	5.3	8.4	27.3	17.1	19.6
140-159	5.2	1.8	2.6	23.7	9.9	13.2
160-179	0.0	0.4	0.3	2.8	1.9	2.2
≥180	0.0	0.0	0.0	0.8	0.3	0.5
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	119.2 ± 11.1	109.5 ± 12.0	111.8 ± 12.5	130.4 ± 15.0	120.6 ± 15.3	123.0 ± 15.8
			t = -24.80, p = 0.000			

Table 2. Percentage of BMI, BP and lipid profiles of participants by gender in 1995 and 2000 (Continue).

Variable	1995			2000		
	Male (n=249)	Female (n=770)	Total (n=1019)	Male (n=249)	Female (n=770)	Total (n=1019)
DBP (mmHg)						
<80	35.7	69.4	61.1	36.5	64.2	57.4
80-84	34.5	23.0	25.8	28.1	20.4	22.3
85-89	6.0	1.4	2.6	11.2	6.1	7.4
90-99	22.5	5.7	9.8	20.9	7.4	10.7
100-109	1.2	0.1	0.4	2.0	1.8	1.9
≥110	0.0	0.4	0.3	1.2	0.1	0.4
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	79.6 ± 8.9	72.6 ± 8.7	74.3 ± 9.3	80.9 ± 11.0	74.5 ± 10.3	76.0 ± 10.9
			t = -5.30, p = 0.000			
FBS (mg/dl)						
<110	95.6	99.0	98.1	92.8	96.8	95.7
110-126	3.6	0.9	1.6	3.6	2.1	2.5
≥ 126	0.8	0.1	0.3	3.6	1.2	1.8
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	88.7 ± 11.0	85.5 ± 10.1	86.3 ± 10.4	95.5 ± 13.1	91.1 ± 11.7	92.2 ± 12.2
			t = -15.62, p = 0.000			
Total cholesterol (mg/dl)						
<160	19.7	25.1	23.7	4.8	10.9	9.4
160-199	38.6	43.1	42.0	22.5	30.9	28.9
200-239	30.9	27.0	28.0	40.6	33.5	35.2
240-279	8.4	4.2	5.2	21.3	17.0	18.1
≥280	2.4	0.7	1.1	10.8	7.7	8.4
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	192.0 ± 40.7	184.1 ± 33.8	186.0 ± 35.7	224.5 ± 40.5	213.3 ± 43.3	216.0 ± 42.9
			t = -24.14, p = 0.000			
HDL-C (mg/dl)						
≥60	27.3	36.2	34.1	63.9	66.2	65.6
50-59	18.1	23.4	22.1	30.5	31.3	31.1
45-49	11.2	10.1	10.4	2.4	2.3	2.4
35-44	27.3	20.8	22.4	3.2	0.1	0.9
<35	16.1	9.5	11.1	0.0	0.0	0.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
$\bar{X} \pm SD$	51.8 ± 19.4	57.2 ± 20.3	55.9 ± 20.2	61.1 ± 7.2	62.2 ± 6.7	61.9 ± 6.9
			t = -9.19, p = 0.000			

Fig. 1 reveals that PRCVD1 was determined mainly by HB1 (β = 0.50), then BMI1 (β = 0.25), Age1 (β = 0.14) and HOS1 (β = -0.12) whereas PRCVD2 was determined mainly by PRCVD1 (β = 0.92) and slightly by BMI2. These findings could conclude that PRCVD2 was a direct affect of PRCVD1 and BMI2 but was an indirect effect of HB1 (β = 0.47), BMI1 (β = 0.37), Age1 (β = 0.26), ED1 (β = 0.10) and HOS1 (β = -0.11) (Table 3). Overall, the longitudinal SEM composed of the selected determinants could explain the change of variance of PRCVD2 as high as 96 per cent (R^2 = 0.96).

DISCUSSION

Physiological risks for CVD include high blood pressure, elevated serum cholesterol or LDL-C, low HDL-C and high plasma glucose are recognized as major or causal risk factors prior to CVD endpoints

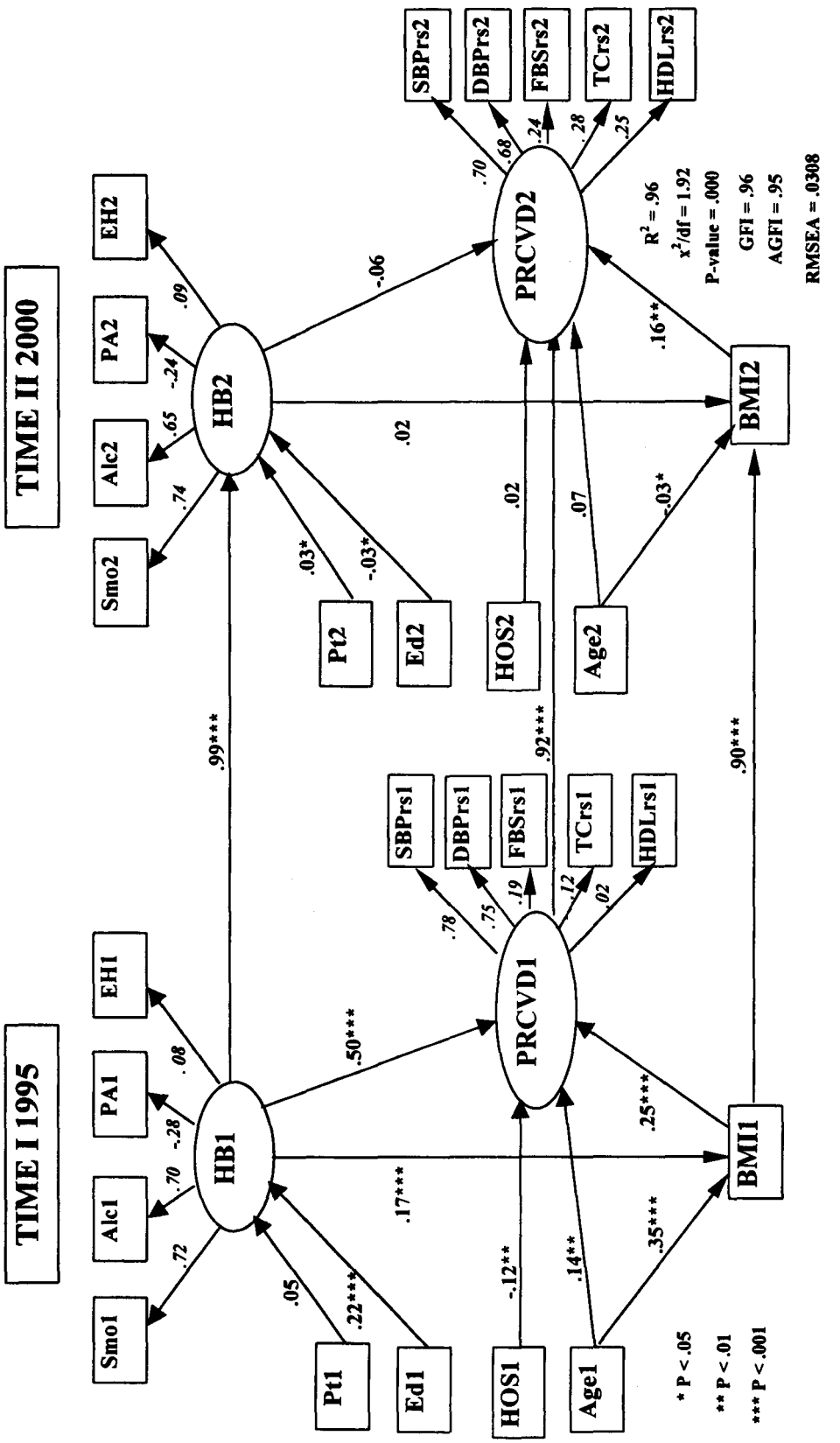

Fig. 1. A longitudinal causal model of cardiovascular risk factors in the employees of the Government Savings Bank with a five-year interval.

Table 3. Effect of direct, indirect and total effect of independent variables on dependent variables of longitudinal causal model.

Independent variable	Dependent variable																	
	HB1			BMI1			PRCVD1			HB2			BMI2			PRCVD2		
	DE	IE	TE	DE	IE	TE	DE	IE	TE	DE	IE	TE	DE	IE	TE	DE	IE	TE
Age1				0.35***		0.35***	0.14**	0.09***	0.23***					0.32***	0.32***		0.26***	0.26***
Age2													-0.03*		-0.03*	0.07	-0.01	0.08
Ed1	0.22***		0.22***		0.04**	0.04**		0.12***	0.12***		0.22***	0.22***		0.04**	0.04**		0.10***	0.10***
Ed2										-0.03*		-0.03*		0.00	0.00		0.00	0.00
Pt1	0.05		0.05		0.01	0.01		0.03	0.03		0.05	0.05		0.01	0.01		0.02	0.02
Pt2										0.03*		0.03*		0.00	0.00		0.00	0.00
HOS1							-0.12**		-0.12**								-0.11**	-0.11**
HOS2																0.02		0.02
HB1				0.17***		0.17***	0.50***	0.04**	0.54***	0.99***		0.99***		0.17***	0.17***		0.47***	0.47***
HB2													0.02		0.02	-0.06	0.00	-0.06
BMI1							0.25***		0.25***				0.90***		0.90***		0.37***	0.37***
BMI2																0.16**		0.16**
PRCVD1																0.92***		0.92***
R^2		0.06			0.16			0.43			0.98			0.79			0.96	

Ed = Education, Pt = Position, HOS = Health opinion survey, HB = Health behaviors, BMI = Body mass index, PRCVD = Physiological risks for CVD,
DE = Direct effect, IE = Indirect effect, TE = Total effect, 1 = 1995, 2 = 2000, * Significant at p<0.05, ** Significant at p<0.01, *** Significant at p<0.001

as diseases or death(4). Obesity is PRCVD categorized as a predisposing factor(4), because obesity itself is not a direct cause of CVD, but obesity contributes to an increase in blood pressure level(13,14), dyslipoproteinemia(13,15,16) and diabetes mellitus (13,17,18). In addition, it was reported that a combination of risk factors increases the risk of CVD(5). Therefore, this study included all major physiological risk markers for CVD in measurement for PRCVD. Moreover, these five physiological markers for CVD were also selected based on their common use for a general physical examination. Among these physiological risks for CVD, SBP and DBP were reported as one of the strong determinants(10), which was confirmed by this study. An increase in the prevalence of overweight, obesity, high blood pressure and disorder of blood chemistry profiles despite an improvement in eating and exercise behaviors may be the result of age changes on BMI(19), blood pressure(20) and the metabolism of glucose and cholesterol when one gets older (4,21). Eating habits and physical activity were weak indicators of measurement for HB, which might be due to the need to use the same measuring instruments in the longitudinal study. Therefore, improvement of either content validity or reliability of measurement instruments including physical activity and eating habits were limited in this study. More comprehensive assessment of physical activity including activity during leisure time and analysis of physical activity in terms of energy expenditure was recommended(22). In addition, more comprehensive food items associated with condition risk for CVD should include both quantity and quality for further study. Moreover, health behavior strongly determined by gender, smoking and alcohol intake were mainly found in males (Table 1). Therefore, a separate model was suggested to better explain the causal relationship among more specific determinant variables for gender difference.

This study demonstrated that BMI was a stronger determinant of PRCVD than age both for actual BMI status in 1995 ($\beta = 0.25$) and increasing BMI with a five-year interval ($\beta = 0.16$). Change et al (18) found that current BMI, early obesity, absolute weight gain throughout adulthood are important independent risk factors for diabetes. Therefore, maintaining a healthy body weight program is a strong recommendation for this population group starting at a younger age and should be maintained throughout adulthood. The result also confirmed that male had higher PRCVD than females(10), whereas,

male bank employees were more overweight than females of the same age. The problem was opposite to the finding in the general population[23]. This might be explained that most female bank employees work closely with the public, therefore, they are more concerned with their figure than male employees.

Diabetes mellitus, hyperlipidemia and hypertension are physiological risk factors associated with heredity[24,25]. People with a family history of these diseases have a higher risk of being affected. A family history is an unchangeable variable, therefore, it was not included in the analysis. Stress is one factor correlated to an increase in blood pressure level[26]. The assessment of stress using the Health Opinion Survey (HOS) which is generally accepted as a high reliability test was used in this study. A reliability test using Cronbanch's coefficient alpha was high both in 1995 ($\alpha = 0.80$)[27] and in 2000 ($\alpha = 0.83$). This result confirmed that the test is of high reliability. The negative direct effect of HOS on PRCVD instead of a positive effect (Fig. 1) was confirmed by a previous report[27]. The analysis of model with gender (unpublished data) found that samples who had higher stress had a higher health behavior risk for CVD than those who had less stress ($\beta = 0.12$, $p < 0.001$), but had no effect on PRCVD. This illustrated that stress may have a direct association with other factors more than PRCVD.

A longitudinal model of five years follow-up in the same samples using SEM illustrated that the selected determinants could explain the 43 per cent ($R^2 = 0.43$) variation in PRCVD in 1995 (Table 3). These determinants could explain as high as 96 per cent of variation of PRCVD in the longitudinal model. The result illustrated that in the longitudinal model previous status such as health behavior, BMI and PRCVD in 1995 became significant determinants of PRCVD in 2000, therefore, the present health status was influenced by previous health behavior and status. These effects could not be observed in a cross-sectional model. This result illustrated the superiority of a longitudinal model over a cross-sectional one. In SEM analysis, measurement errors which are commonly found during the process of research were controlled. Moreover, the indirect effect of determinants on the dependent variable were compatible. These are the two advantages of using LISREL over the linear multiple regression analysis.

In conclusion, the findings of this study illustrated that the current PRCVD has a strong effect of previous health behavior and PRCVD. Therefore, it should be recommended that a health promotion program to prevent cardiovascular disease should start early in life to reduce the occurrence of CVD. Moreover, it is also suggested that a regular physical check up might lead to an increase in health concern and improvement of some behaviors among employees. However, it is not enough to improve or maintain their health status. Strengthening of a health promotion policy applying these findings should be emphasized. In addition, these findings could also be generalized either for other population groups who have similar characteristics or for the general population promoting good health to prevent CVD.

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ความสัมพันธ์เชิงเหตุและผลระยะยาวระหว่างปัจจัยเสี่ยงของโรคหัวใจและหลอดเลือดในพนักงานธนาคารออมสิน

ภารดี เต็มเจริญ, ส.ด., มันทนา ประทีปะเสน, พ.บ., ส.ด.*,*
*ธวัชชัย วรพงศธร, ปร.ด.**, ปิยะมิตร ศรีธรา, พบ.****

รูปแบบความสัมพันธ์เชิงเหตุและผลระยะยาวสร้างขึ้นเพื่ออธิบายความสัมพันธ์ระหว่างตัวทำนายซึ่ง ประกอบด้วย ปัจจัยส่วนบุคคล 4 ปัจจัย ความเครียด พฤติกรรมสุขภาพ 4 ด้าน และดัชนีมวลกาย ต่อการผันแปรของปัจจัยเสี่ยงทางสรีรวิทยา ต่อการเกิดโรคหัวใจและหลอดเลือด 5 ชนิด (ความดันโลหิตซีสโตลิคและไดแอสโตลิค ระดับน้ำตาลในพลาสมา โคเลสเตอรอลรวม และไลโปโปรตีนชนิดความหนาแน่นต่ำ) ในพนักงานธนาคารออมสิน 1,019 คน ในช่วงเวลาห่างกัน 5 ปี การประเมินนิสัยบริโภคใช้แบบสอบถามความถี่อาหารบริโภค 14 ข้อ และวัดความเครียดด้วย The Health Opinion Survey (HOS) ชั่งน้ำหนักและวัดความสูง คำนวณดัชนีมวลกาย วัดความดันโลหิต และเก็บตัวอย่างเลือดเพื่อวิเคราะห์ค่าชีวเคมี

ผลการวิเคราะห์ข้อมูลด้วย LISREL พบว่าตัวทำนายที่กำหนดในรูปแบบที่สร้างสามารถทำนายการผันแปรของปัจจัยเสี่ยงทางสรีรวิทยาต่อการเกิดโรคหัวใจและหลอดเลือดได้สูง (R^2 = 0.96, relative chi-square = 1.92, RMSEA = 0.03, GFI = 0.96 and AGFI = 0.95) และการศึกษายังพบว่า ภาวะของปัจจัยเสี่ยงทางสรีรวิทยาต่อการเกิดโรคหัวใจและหลอดเลือดในปัจจุบัน ได้รับอิทธิพลจากสภาวะเมื่อ 5 ปีก่อนทางด้านอายุ การศึกษา พฤติกรรมสุขภาพ ดัชนีมวลกาย และปัจจัยเสี่ยงทางสรีรวิทยาของกลุ่มตัวอย่าง

คำสำคัญ : โรคหัวใจและหลอดเลือด, ปัจจัยเสี่ยงโรคหัวใจและหลอดเลือด, โมเดลลิสเรล, พนักงานธนาคารออมสิน, การศึกษาระยะยาว

ภารดี เต็มเจริญ, มันทนา ประทีปะเสน,
ธวัชชัย วรพงศธร, ปิยะมิตร ศรีธรา
จดหมายเหตุทางแพทย์ ฯ 2545; 85: 863–874

* ภาควิชาโภชนวิทยา,
** ภาควิชาชีวสถิติ, คณะสาธารณสุขศาสตร์ มหาวิทยาลัยมหิดล,
*** หน่วยโรคหัวใจ, ภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ โรงพยาบาลรามาธิบดี, มหาวิทยาลัยมหิดล, กรุงเทพ ฯ 10400