BMJ Open Validation of the Framingham general cardiovascular risk score in a multiethnic Asian population: a retrospective cohort study

Yook Chin Chia,¹ Sarah Yu Weng Gray,² Siew Mooi Ching,^{3,4} Hooi Min Lim,¹ Karuthan Chinna⁵

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For numbered affiliations see end of article.

Correspondence to
Dr Yook Chin Chia;
chiayc@um.edu.my

ABSTRACT

Objective: This study aims to examine the validity of the Framingham general cardiovascular disease (CVD) risk chart in a primary care setting.

Design: This is a 10-year retrospective cohort study.

Setting: A primary care clinic in a teaching hospital in Malaysia.

Participants: 967 patients' records were randomly selected from patients who were attending follow-up in the clinic.

Main outcome measures: Baseline demographic data, history of diabetes and smoking, blood pressure (BP), and serum lipids were captured from patient records in 1998. Each patient's Framingham CVD score was computed from these parameters. All atherosclerotic CVD events occurring between 1998 and 2007 were counted.

Results: In 1998, mean age was 57 years with 33.8% men, 6.1% smokers, 43.3% diabetics and 59.7% hypertensive. Median BP was 140/80 mm Hg and total cholesterol 6.0 mmol/L (1.3). The predicted median Framingham general CVD risk score for the study population was 21.5% (IQR 1.2–30.0) while the actual CVD events that occurred in the 10 years was 13.1% (127/967). The median CVD points for men was 30.0, giving them a CVD risk of more than 30%; for women it is 18.5, a CVD risk of 21.5%. Our study found that the Framingham general CVD risk score to have moderate discrimination with an area under the receiver operating characteristic curve (AUC) of 0.63. It also discriminates well for Malay (AUC 0.65, $p=0.01$), Chinese (AUC 0.60, $p=0.03$), and Indians (AUC 0.65, $p=0.001$). There was good calibration with Hosmer-Lemeshow test $\chi^2=3.25$, $p=0.78$.

Conclusions: Taking into account that this cohort of patients were already on treatment, the Framingham General CVD Risk Prediction Score predicts fairly accurately for men and overestimates somewhat for women. In the absence of local risk prediction charts, the Framingham general CVD risk prediction chart is a reasonable alternative for use in a multiethnic group in a primary care setting.

Strengths and limitations of this study

- The strength of our study is that it is done in a primary care setting where the cardiovascular disease (CVD) risk profiles are different from that in secondary care.
- Another strength is that it examines the validity and applicability of the Framingham general CVD risk tool in a multiethnic primary care population.
- Our sample size is large enough to give us a better picture of CVD risk of a multiethnic population in Asia where data about the use and applicability of any CVD risk prediction tool has not extensively been validated to particularly include non-fatal events.
- Our study found the Framingham General CVD risk score to have a good calibration and hence, it can be used by all the three different ethnic groups.
- However, most of our patients were already on treatment and this resulted in a seemingly over-estimation of cardiovascular risk.
- As this is a retrospective study, there will be missing data to some extent. However, we were able to compare the characteristics of those with missing data to those included in the analysis and found no clinical differences between them.

INTRODUCTION

Guidelines on management of cardiovascular (CV) diseases (CVDs) recommend assessing overall CV risk to determine the necessity of pharmacotherapy.^{1–9} To date, many CVD risk assessment tools have devised.¹⁰ The most well studied is the Framingham Coronary Heart Disease (CHD) risk prediction Chart.^{11 12} This tool has been validated in different populations and was found to over-estimate risk particularly in women, the older age group, British men and European

population and certain ethnicities with lower background risk of CVD, for example, the Chinese population.^{1 13–17}

Furthermore, it has been criticised that the Framingham CHD risk score only predicts risk for fatal and non-fatal coronary events and does not take into account strokes as well as patients already receiving anti-hypertensive therapy, which is the case with many individuals.

Hence, the Framingham general CVD risk score tool for primary care was developed in 2008.¹⁸ This newer tool now includes strokes, heart failure and peripheral artery disease, and adjusts for patients already on anti-hypertensive therapy. However, this risk score chart tool has not been widely validated particularly in the communities with different races. Malaysia is a multiethnic country with a population of 30 million. The major ethnic group is the Malay (50.3%) followed by Chinese (21.7%), Indians (6.5%) and others (21.5%).¹⁹

Hence, our aim was to examine the validity of the Framingham general CVD in a multiethnic primary care population.

MATERIALS AND METHODS

Setting

This is a retrospective cohort study. We computed the Framingham general CVD risk score retrospectively in a randomly selected cohort of patients registered with an urban primary care clinic. This selected sample consisted of adult patients aged 30 years and older, who were free of any cardiovascular events at baseline and who had complete data needed to compute their overall CV risk. The study was conducted in an outpatient clinic of an academic teaching hospital.

Participants

There were 1536 patients in our original cohort. We excluded 563 patients as they did not have all the variables needed to calculate the Framingham general CVD risk score. Another six patients were excluded as we could not ascertain their CVD status by the end of 2007. Thus, a total of 967 patients (62.9%) were eligible for analysis (figure 1). Of these, 912 (94.3%) patients completed 10 years follow-up and only 55 (5.7%) patients

defaulted follow-up. Of these, 26 (2.7%) had died with 19 (73%) deaths from non-CVD causes.

Inclusion criteria

All adult patients 30 years and older, without any cardiovascular events and with documented blood pressure (BP) readings whether they were on or not on treatment, total cholesterol, high-density lipoprotein (HDL) cholesterol, smoking status, and presence or absence of diabetes mellitus (DM) were eligible for this study. These parameters were needed to compute each individual's general CVD risk level.

Design

This sample was randomly selected using a computer-generated number based on the patient's unique registration number with the clinic. All patient records were in paper form.

Data collection

We extracted the patients' sociodemographic data and clinical information based on the first entry in 1998 from their records manually, according to a predetermined proforma (clinical report form). Use of hypoglycaemic and lipid-lowering agents were also captured. Using age, SBP (systolic BP; treated or not treated) total cholesterol, HDL cholesterol levels, smoking and diabetes status, we computed each individual's general CVD risk score.

CVD events, namely, fatal and non-fatal CHD, fatal and non-fatal strokes, heart failure and peripheral artery disease were also captured from the records. For those who did not complete their subsequent 10-year follow-up at our clinic (n=24), we traced and examined the case records from the main hospital to determine their CVD outcome. For those patients who did not complete their follow-up in our clinic, we contacted them individually (n=31) to ascertain their CVD status and for any events fatal or non-fatal. Each CVD event was counted as one event in individuals who had more than one event during the 10-year follow-up. We further categorised the 10-year CVD risk as low (<10% 10-year CVD risk), medium (10–20% CVD risk), or high >20% CVD risk.²⁰

Blood pressure (BP) was measured by our attending doctor using a mercury sphygmomanometer as part of the daily routine clinical practice. Diagnosis of hypertension was made in accordance with standard recommendations.⁷ Target control BP is defined as BP<140/90 mm Hg for patients without DM and <130/80 mm Hg for patients with DM. DM is defined as documented by the attending physician or the use of hypoglycaemic agents or both. Smokers were defined if they were still smoking. Non-smokers were those who never smoked or currently not smoking regardless of when they had stopped smoking. Total cholesterol, low-density lipoprotein (LDL) and HDL cholesterol were also collected. All blood tests were performed in our teaching hospital's

Figure 1 Flow chart of patients included in the analysis.

laboratory, which is certified by the Royal College of Pathologists of Australasia standards.

Statistical analysis

All statistical analysis was carried out using the Statistical Package for Social Sciences (SPSS V.21). Continuous data are described with mean and SD if the distribution is normal. When the data is a skewed distribution, median and IQR (25–75th centile) are used. Categorical data are reported as proportions (percentage). The χ^2 test (χ^2) or Fisher exact tests were used for the categorical or dichotomous variables.

Discrimination

Discrimination is defined as the ability of the risk prediction model in distinguishing patients who suffered from a CVD event from those who do not use an overall c index. The c index is analogous to the area under the receiver operating characteristic curve (AUC). Discrimination is defined as good when c index is closer to 1, whereas a value of 0.5 implies that the risk score tool is no better than chance.²¹ A value of 0.75 is considered as good discrimination. Values between 0.51 and 0.74 are considered moderate and ≤ 0.5 as poor.²²

Calibration

Calibration was used to assess if the observed 10-year CVD events differed significantly from the predicted CVD events.⁸ The calibration of the Framingham general CVD risk score was determined using Hosmer-Lemeshow test.²³ A χ^2 value of greater than 20 or a p value of less than 0.05 indicates poor calibration.

RESULTS

A total of 967 of 1536 patient records from 1998 (baseline) were entered into our analysis.

Table 1 shows the demographic data of our cohort. Our study population was made up of three main ethnic groups, namely, the Chinese (45.4%), Malays (23.3%) and Indians (30.2%). The mean age was 56.9 (9.9) years and two-third (66.2%) were females. At baseline in 1998, over half (59.7%) had hypertension, less than half (43.3%) had diabetes and less than 10% (6.1%) were current smokers, 80.9% (n=339) of the patients with DM were receiving treatment with either oral hypoglycaemic agents and/or insulin. The mean SBP was 140.7 $\pm$ 18.6 mm Hg and more than half (59.7%) of them were on antihypertensive agents. However, only 9.8% of the population (n=95) received statin therapy in 1998. We also compared the baseline characteristics for those who were entered into our analysis to the patients who were excluded. Basically, there were no significant differences between their baseline characteristics except for the smoking status and statin use.

Table 1 also demonstrates the change in CVD risk factors between baseline and at the end of 10 years. In spite of the increase in the prevalence of diabetes from 43.3% to 46.3%, glycated haemoglobin (HbA1c) control actually improved from a mean of 7.8% to 7.5%. The mean SBP was reduced by 5.8 mm Hg, from 140.7 to 134.9 mm Hg. The control rate for BP improved from 41.3% to 55.6%. There was an increase in the use of renin-angiotensin system (RAS) blockers from 7.3% to 48.1%. The number of patients receiving statin increased significantly from 9.8% to 62.9% in 2007. There was also improvement in the lipid profile of patients at the end of

Table 1 Comparison of CV risk factors between study population and excluded patients as well as changes in CV risk factors in 1998 and 2007

	Study population (n=973)	Excluded population (n=563)	p Value	2007
Age, year (mean)	56.9 $\pm$ 9.9	55.8 $\pm$ 10.4	0.06	66.9 $\pm$ 9.9
Sex, female (n, %)	640 (66.2)	362 (64.3)	0.45	640 (66.2)
Ethnicity (n, %)			0.16	
Malay	225 (23.3)	155 (27.5)		225 (23.3)
Chinese	438 (45.4)	221 (39.3)		438 (45.4)
Indian	291 (30.2)	180 (32.0)		291 (30.2)
Others	13 (1.2)	7 (1.2)		13 (1.2)
DM (n, %)	419 (43.3)	260 (46.3)	0.28	556 (57.5)
HbA1c, % (mean)	7.8 $\pm$ 1.9	7.9 $\pm$ 1.9	0.28	7.5 $\pm$ 1.7
DM controlled with HbA1c $\leq$ 6.5% (n, %)	113 (11.6)	64 (11.4)	0.89	168 (17.4)
Systolic BP, mm Hg (mean)	140.7 $\pm$ 18.6	139.0 $\pm$ 19.7	0.22	134.9 $\pm$ 16.5
Use of antihypertensive agents (n, %)	577 (59.7)	308 (54.7)	0.09	791 (81.8)
RAS blocker use (n, %)	71 (7.3)	27 (4.8)	0.05	465 (48.1)
Total cholesterol, mmol/L (mean)	6.1 $\pm$ 1.1	5.8 $\pm$ 1.1	0.06	4.9 $\pm$ 1.0
HDL cholesterol, mmol/L (mean)	1.2 $\pm$ 0.4	1.4 $\pm$ 0.2	0.49	1.3 $\pm$ 0.3
LDL cholesterol, mmol/L (mean)				3.0 $\pm$ 0.8
Statin use in 1998 (n, %)	95 (9.8)	32 (5.7)	0.005	608 (62.9)
Smoker (n, %)	59 (6.1)	47 (8.3)	<0.001	59 (6.1)

BP, blood pressure; CV, cardiovascular; DM, diabetes mellitus; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RAS, renin-angiotensin system.

10 years compared with baseline. Mean total cholesterol was reduced from 6.1 to 4.9 mmol/L, while the mean HDL increased from 1.2 to 1.3 mmol/L.

Table 2 compares CVD risk factors at baseline and events rate at the end of 10 years in males and females. Generally, males had higher CV risk profiles than females. They were older and more of them were smokers; they had poorer control of BP and lipid profiles, but had a slighter better glucose profile. The CHD events were twice more common in males (13.5%) than in females (6.4%). Similarly, strokes were higher in males (6.1%) than in females (3.1%). In total, six CVD events were fatal in males and only one fatal in females.

Table 3 shows the risk categories of Framingham general CVD risk score and the CVD events that occurred. The median Framingham general CVD risk score for the study population was 21.1% (IQR 1.2–30.0%) with a 95% CI of 19.9% to 21.1%. The actual number of CVD events that occurred in the 10 years was 127 (13.1%), while the predicted number was 204 (21.1%). In males, their median risk score was 30.0% (95% CI 24.9% to 26.4%), while the events that occurred were 62 (19%). In females, their median risk score was 18.5% (95% CI 24.9% to 26.4%) while the events that occurred were 65 (10.2%).

Table 3 Comparison of risk categories of Framingham general cardiovascular disease (CVD) risk score and CVD events in a 10-year interval (1998–2007)

Framingham general CVD risk score in 1998, %	Total, N	Predicted CVD events (n, %)	Observed CVD event (n, %)	
<i>All adults</i>				
Median score: 21.1%	967	204 (21.1)	127 (13.1)	<0.001
<10%	148	9 (6.3)	9 (6.1)	
10–20%	292	40 (13.7)	25 (8.6)	
>20%	527	158 (30.0)	93 (17.6)	
<i>Male</i>				
Median score: 30.0%	327	98 (30.0)	62 (19.0)	0.003
<10%	12	1 (6.3)	1 (8.3)	
10–20%	56	9 (15.4)	2 (3.6)	
>20%	259	74 (28.7)	59 (22.8)	
<i>Female</i>				
Median score: 18.5%	640	118 (18.5)	65 (10.2)	0.098
<10%	136	9 (6.3)	8 (5.9)	
10–20%	236	34 (14.4)	23 (9.7)	
>20%	268	72 (27.0)	34 (12.7)	

Table 2 Comparison CV risk factors in 1998 and events at the end of 10 years according to gender

	Male (N=327)	Female (N=640)	p Value
Age, year (mean)	57.7 (10.1)	56.5 (9.7)	0.07
Hypertension	217 (66.4)	360 (56.3)	0.002
Systolic BP, mm Hg (mean)	142.3 (17.6)	139.9 (19.1)	0.07
Diastolic BP, mm Hg (mean)	87.6 (10.4)	83.3 (9.5)	<0.001
BP control rate (N, %)	22 (25.0)	66 (75.0)	<0.001
Total cholesterol, mmol/L (mean)	5.8 (1.1)	6.2 (1.1)	<0.001
HDL cholesterol, mmol/L (mean)	1.1 (0.4)	1.3 (0.3)	<0.001
Triglyceride, mmol/L (mean)	1.9 (1.0)	1.6 (0.9)	<0.001
LDL cholesterol, mmol/L (mean)	3.4±0.9	3.8±1.1	0.04
LDL cholesterol <2.6%, mmol/L (N, %)	29 (46.8)	33 (53.2)	0.03
Smoking (N, %)	51 (15.6)	8 (1.3)	<0.001
Diabetes	143 (43.7)	276 (43.1)	0.89
HbA1c % (mean)	7.5 (1.6)	7.9 (1.9)	0.03
HbA1c %≤6.5% (N, %)	40 (12.2)	71 (11.1)	0.59
Body mass index, kg/m ² (mean)	26.6 (4.3)	26.7 (4.9)	0.73
Events N (%)			
	Male (N=327) n (%)	Female (N=640) n (%)	
Coronary heart disease, N=85 (8.8%)	44 (13.5)	41 (6.4)	<0.001
Fatal myocardial infarction	2 (0.6)	1 (0.2)	0.23
Non-fatal myocardial infarction	3 (0.9)	4 (0.6)	0.61
New angina	39 (11.9)	36 (5.6)	0.001
Heart failure, N=7 (0.7%)	1 (0.3)	6 (0.9)	0.27
Cerebral vascular event, N=41 (4.2%)	20 (6.1)	20 (3.1)	0.03
Fatal stroke	4 (1.2)	0 (0.0)	0.005
Non-fatal stroke	17 (5.2)	20 (3.1)	0.11
Peripheral vascular disease N=0 (0%)	0 (0.0)	0 (0.0)	–

BP, blood pressure; CV, cardiovascular; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 4 Comparison of risk categories of Framingham general CVD risk score and medications used in a 10-year interval (1998–2007)

Sex	Framingham: CVD risk category in 1998 (%)	Anti-HPT agents (N, %)		Antilipid agents (N, %)		Hypoglycaemic agents (N, %)	
		1998	2007	1998	2007	1998	2007
Male (N=327)	Low risk score <10	4 (33)	8 (80)	1 (8)	4 (40)	6 (50)	5 (50)
	Medium risk score 10–20	29 (52)	43 (78)	5 (9)	27 (49)	19 (34)	23 (43)
	High risk Score >20	184 (71)	224 (94)	19 (7)	145 (60)	143 (55)	168 (70)
Female (N=640)	Low risk score <10	45 (33)	85 (66)	17 (13)	78 (60)	59 (44)	57 (44)
	Medium risk score 10–20	120 (51)	193 (85)	29 (12)	158 (69)	112 (48)	135 (59)
	High risk score >20	195 (73)	238 (96)	24 (9)	196 (79)	160 (60)	187 (75)

CVD, cardiovascular disease; HPT, hypertensive.

The number of events occurring in the low-risk group, in men and women, were consistent with the number predicted. In the medium risk group, the risk score tool overpredicted risk in men and women, but more so for men. In the high-risk group of men, the prediction of CVD events is consistent with the actual events that occurred. However, in females with high risk, the risk score tool overpredicted the CVD risk.

Table 4 shows the risk categories of Framingham general CVD risk score and medications used in a 10-year interval (1998–2007). Overall, the use of medications for CVDs was highest in the high-risk group in men and women at baseline. Nearly three-quarters of men and women in the high-risk group were already receiving antihypertensive at baseline. Medication use increased across all risk categories in men and women over 10 years; the greatest increase in use was the use of antihypertensive in females with medium risk (increment of 34%), and use of antilipid agents by females in high-risk groups (increment of 70%).

The AUC for Framingham general CVD risk score was 0.63, showing moderate discrimination as seen in figure 2. The calibration for Framingham general CVD risk score was good as the Hosmer-Lemeshow test result was $\chi^2=3.25$, $p=0.78$.

Table 5 shows the comparison between Framingham general CVD risk score and CVD events according to ethnicity. The Framingham general CVD risk score discriminates moderately well for Malay race (AUC 0.65, $p=0.01$), Chinese (AUC 0.60, $p=0.03$) and Indians (AUC 0.65, $p=0.001$). Males (AUC 0.63, $p=0.002$) seem to have a better discrimination index than females (AUC 0.58, $p=0.04$).

DISCUSSION

Our study shows the Framingham general cardiovascular risk score tool to have moderate discrimination and good calibration in an Asian population. These findings are not surprising as our study cohort are actually patients from a clinic and not subjects in the community. Hence, not unexpectedly, the overall CVD risk profile of our cohort is already high before CVD events have occurred,

with the majority of them having underlying diabetes and/or hypertension. Therefore, a clustering of patients with higher risk and fewer patients with lower risk explain our finding of only moderate discrimination. Perhaps this risk prediction model may give a better discrimination in a general population of subjects, with a wider range of CV risk. Similarly, the overall CVD risk as predicted by the Framingham general CVD risk score tool for our cohort of primary care patients, was high in males and medium in females. This is again not a surprising finding as males in our cohort generally had higher CVD risk profiles (poorer BP and LDL<2.6%). Consequently, they also had more CVD events and higher mortality rates. Thus, the Framingham CVD risk tool helps to identify appropriately patients at high risk where more needs to be done to reduce their risk. A recent study validated the Framingham cardiovascular risk score in Tehran,

Figure 2 Receiver operating characteristic (ROC) and area under the curve for Framingham general cardiovascular disease risk score.

Table 5 Comparison Framingham general CVD risk score and CVD events according to ethnicity and gender

	Framingham general CVD risk score (%)	CVD event		AUC (95% CI)	p Value
		Observed N (%)	Predicted N (%)		
Overall (N=967)	21.1	127 (13.1)	204 (21.1)	0.63 (0.58 to 0.68)	<0.001
Malay (N=225)	18.5	26 (11.6)	42 (18.5)	0.65 (0.53 to 0.77)	0.014
Chinese (N=438)	23.2	45 (10.3)	102 (23.2)	0.60 (0.52 to 0.68)	0.027
Indian (N=291)	21.5	54 (18.6)	63 (21.5)	0.65 (0.57 to 0.73)	0.001
Male (N=327)	30.0	62 (19.0)	98 (30.0)	0.63 (0.56 to 0.70)	0.002
Female (N=640)	18.5	65 (10.2)	118 (18.5)	0.58 (0.51 to 0.65)	0.04

AUC, area under the receiver operating characteristic curve; CVD, cardiovascular disease.

a Middle Eastern population, and found it also works very well.²⁴

In spite of the Framingham risk score seemingly over-predicting CVD risk to some extent, we feel that it is appropriate for use in our cohort. This is because the prediction is accurate for both men and women with low risk and for men with high risk as the predicted and the observed risk score belong to the same category of risk, that is, when the predicted risk is high, the observed risk is also high. In fact, one could argue that the tool underestimates risk as the majority of high-risk patients were already receiving one or another medication for the CVD; hence, their risk is expected to be reduced.

Our study found in general that this new risk tool over-estimated risk in women. This is consistent with findings in the other studies which used other risk tools.^{1 13–17} While this appears as an overestimation, this may not be the case. It could be because of the increase in use of antihypertensive agents which would have lowered their risk. For example, a great increase in the use of anti-hypertensive agents was seen over the 10 years of follow-up.²⁵ Similarly, while use of antihypertensive increased across all risk categories, the greatest increase was seen in both men and women with medium risk. Furthermore, at the end of 10 years, the achieved mean BP was lower and the control rate of BP higher in the medium risk-group compared to the high-risk group. The greater use of medication and better control of BP could have led to a greater reduction in CV events and hence, this lead to what appears to be like an overestimation of risk.

Our present study has several strengths and few limitations. First, the strength of our study is that it is done in the primary care setting where the CVD risk profiles is different from that in secondary care. Another strength of this study is that it examines the validity and applicability of the Framingham general CVD risk tool in a multiethnic primary care population. Second, our sample size is large enough to give us a better picture of CVD risk of a multiethnic population in Asia, where data about the use and applicability of any CVD risk prediction tool is lacking. Our study found that it did have a good fit and good calibration to be used by all the

three different ethnic groups. Although it seems to be apparently overestimating risk, it must be remembered that this could be due to the treatment effect. Use of such a tool is of particular importance in the light of limited resources and the rapidly increasing CVD burden in the region as it can help identify those most at risk; these need to be treated in order to reduce their risk.

A limitation of our study is that the use of CV risk prediction tools are meant to be used to estimate overall CV risk in order to decide on treatment. Most of our patients were already on treatment and that would result in an overestimation of risk in most situations. However, based on current clinical practice, it would be unethical to do a study where patients at risk are not treated in order to derive a risk tool without treatment being on board. As this is a retrospective study, there will be missing data to some extent. However, we were able to compare the characteristics of those with missing data (and hence excluded from the analysis) to those included in the analysis and found no major clinical differences between them, suggesting there was no substantial bias.

CONCLUSIONS

The Framingham general CVD risk prediction tool is valid for use in primary care population. In the absence of local risk prediction tools, the Framingham CVD risk tool can be used as a surrogate to stratify risk and hence determine the indication for pharmacotherapy. Furthermore this Framingham general CVD risk prediction tool which is meant for use in primary care provides a good fit for use in a multiethnic population in Asia.

Author affiliations

¹Department of Primary Care Medicine, Faculty of Medicine, University of Malaya Primary Care Research Group (UMPCRG), University of Malaya, Kuala Lumpur, Malaysia

²Department of Urology, Barnet Hospital, Wellhouse Lane Barnet, Hertfordshire, UK

³Department of Family Medicine, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Malaysia

⁴Department of Gerontology, Universiti Putra Malaysia, Serdang, Malaysia

⁵Department of Social and Preventive Medicine, University of Malaya, Kuala Lumpur, Malaysia

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