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# Body Mass Index as the Predictor of High Sensitivity C-Reactive Protein: A Risk Marker of Cardiovascular Diseases

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## ABSTRACT

**Introduction:** High sensitivity C-reactive protein (hs-CRP) is an emerging risk marker for cardiovascular diseases (CVD). In Malaysia, CVD has become a major health problem and the risk factors of CVD have also increased among the middle-aged. Thus, this study aimed to determine factors that influence the level of hs-CRP among Malaysian adults aged 30-55 years-old. **Methods:** One-hundred and twenty-two (n=122) adults working at an institution were selected systematically in this cross-sectional study. Body weight, height, hip and waist circumference, systolic and diastolic blood pressure, hs-CRP level, total cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein were measured. Body mass index (BMI), waist-to-hip ratio, and total cholesterol: high-density lipoprotein ratio were calculated. **Results:** The sample consisted of 40.2% male and 59.8% female subjects with a mean age ± SD of 41.93 ± 8.26 years. Pearson's correlation coefficient showed significant positive correlations between log hs-CRP level (mean ± SD = 0.22 ± 0.50 mg/L; 95% CI = 0.13 – 0.31) and age, waist circumference, hip circumference, BMI, systolic blood pressure, total cholesterol, low-density lipoprotein, triglycerides, and total cholesterol: high-density lipoprotein ratio. However, multivariate analysis showed only BMI ($R = 0.489$, $R^2 = 0.239$, adjusted $R^2 = 0.232$, $F = 37.626$, $p < 0.001$) was a predictor of hs-CRP, the risk marker of CVD. Hs-CRP level was greater in overweight (25 – 29.9 kg/m$^2$), and obese (> 30 kg/m$^2$) subjects (ANOVA $p< 0.05$) compared to normal weight subjects. **Conclusion:** BMI is a modifiable risk factor with the change being important for reducing CVD events among adults.

**Key words:** Body mass index, blood pressure, C-reactive protein, lipid profiles nutritional status

## INTRODUCTION

Currently, there are various inflammatory biomarkers that are considered as predictors of cardiovascular risk. C-reactive protein (CRP) is one of the new biomarkers that assists in early detection of the disease. It is an acute phase reactant released from the body in response to acute injury, infection or other inflammatory response (Ledue & Rifai, 2003). CRP is synthesised by the liver and is regulated principally by the cytokine interleukin 6 (IL-6) that is produced in the adipose tissue of healthy humans (Yaqoob & Ferns, 2005). In fact, IL-6 is a powerful inducer of the hepatic acute phase response (Yudkin *et al.*, 2000). On the other hand, the levels of CRP circulated in the body are positively associated with a number

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of classical cardiovascular risk factors such as age, body mass, systolic blood pressure, smoking (Yaqoob & Ferns, 2005), metabolic syndrome, diabetes mellitus, low high-density lipoprotein (HDL), and high triglycerides (TG) levels (Pearson *et al.*, 2003). Elevated concentrations of CRP can predict future risk of acute coronary syndromes in healthy subjects (Yudkin *et al.*, 2000). It is suggested that CRP might also play a major role in atherogenesis, a pathogenesis of cardiovascular diseases (Santos *et al.*, 2005), and as a powerful predictor of first and recurrent cardiovascular events (Pearson *et al.*, 2003). Besides, Pearson *et al.* (2003) also stated that measurement of CRP might be useful in those with a 10-year absolute risk of coronary heart disease (CHD) from 10% to 20%.

A high-sensitivity CRP (hs-CRP) is a CRP that is quantified at low detection limit by using a high sensitivity immunoassay method. A study found that every 10% increase in hs-CRP is associated with 3% odds increase of CVD (Albert & Ridker, 2006). Guidelines by The American Heart Association and Centers for Disease Control and Prevention (AHA/CDC) in 2003 stated that the cutting points for risk determination for hs-CRP are: low = < 1.0 mg/L, average = 1.0 – 3.0 mg/L, and high = > 3.0 mg/L (Pearson *et al.*, 2003).

Meanwhile in Malaysia, the latest National Health and Morbidity Survey has reported an increase in the prevalence of hypertension. In particular, the prevalence of hypercholesterolemia among the Malay ethnic group had doubled from the previous survey to 38.4%. Besides, the Malay ethnic group also had the second highest prevalence of abdominal obesity in Malaysia. Moreover, it was noteworthy that the prevalence of hypercholesterolemia and overweight was reported higher among semi-government employees followed by obesity, hypertension and abdominal obesity (Ministry of Health, 2006). It is a worrying issue as the statistics showed that Malays and semi-government employees are at risk of developing CVD. Even though studies on CVD are abundant in Malaysia (Ministry of Health, 2006; Rampal *et al.*, 2012), data on risk markers of CVD such as hs-CRP are limited and not well explored. Given this observation as the epitome of our study, this research was conducted to assess the influence of CVD risk factors in terms of nutritional status, blood pressure and lipid profiles on the levels of hs-CRP.

## METHODS

### Study population

Out of the sixteen faculties in Universiti Putra Malaysia, Serdang, four faculties were randomly selected to carry out this study from October 2011 until January 2012. Sample size was calculated by using the formula adapted from Cochran (1977). The formula used was $Z^2_{1-\alpha/2}(p)(1-p) / d^2$ ($Z_{\alpha/2}$= 1.96; p=0.437; d=0.10). The minimum sample size required for this study was 95 subjects. However, an additional 30% of 95 subjects was recruited to avoid missing value or errors. At the end, this cross-sectional study involved 122 academic and non-academic staff aged 30-55 years-old. Subjects were male and female Malay adults. All subjects were systematically selected from each faculty by using a sampling frame from a name list obtained from the registrar's office that had subjects' working position. Subjects who were pregnant and lactating during data collection were excluded from this study. Moreover, those with illness caused by bacterial infections or other inflammatory conditions such as rheumatoid arthritis and injury before or during data collection which might affect CRP results were also excluded. However, subjects with hypertension and hyperlipidemia who were aware of their raised systolic or diastolic or lipid profile readings were included in this study. The study protocol was approved by the Medical Research

Ethics Committee, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia. Permission letters to enter the selected faculties were obtained prior to data collection. An information sheet explaining the purpose of the study was distributed to the respondents. Prior to sample collection, written informed consent was obtained from the respondents.

**Socio-demographic background**

Information regarding age, date of birth, sex, marital status, job category, and educational levels were self-reported.

**Nutritional status assessment and blood pressure measurement**

Nutritional status of the subjects was determined by taking their anthropometric measurements namely weight, height, waist and hip circumferences. Weight was measured to the nearest 0.1kg by using TANITA digital scale model HD-308 (TANITA Corporation, USA) while height was measured to the nearest 0.1cm by using SECA wall stadiometer model 206 (SECA, Germany). Waist and hip circumferences were measured using measuring tape. The OMRON automatic blood pressure monitor model IA2 was used for the measurement of subjects' blood pressure. All the measurements were taken twice, and the average was calculated. Body Mass Index (BMI) and waist-to-hip ratio (WHR) of the subjects were calculated using these formulas: BMI = weight (kg)/ height ($m^2$); WHR = waist circumference (cm)/ hip circumference (cm) (World Health Organisation, 2000).

**Biochemical measurement**

Ten ml of fasting blood sample (10-12 hours) was drawn from subject's vein by a medical personnel. The blood samples were collected in blood collection tubes containing sierogel (plain gel). All the samples were then sent to B.P Clinical Lab Sdn Bhd, Malaysia for the analysis of hs-CRP, total cholesterol (TC), low-density lipoprotein (LDL), HDL, TG, and TC: HDL ratio.

**Statistical analysis**

Data were analysed using IBM SPSS Statistics 20.0 (SPSS Inc., Chicago, IL, USA) and significance value was accepted at $p < 0.05$. Data were tested for normality by using skewness (± 2) and Q-Q Plot. Outliers (determined by using Box-plot) were excluded from the analysis. Differences of mean values for all continuous variables between male and female were assessed by using independent-sample *t*-test (normally distributed data) and Mann-Whitney U-test (not normally distributed data). Since data on hs-CRP were not normally distributed, the data were then log transformed in order to approach normality. Differences of mean log hs-CRP between its risk classifications were also assessed by using an independent-sample t-test and ANOVA. Pearson's correlation coefficient was used to evaluate the correlations between the studied variables and log hs-CRP. Multiple Linear Regression (MLR) analysis was conducted to address the question of which set of these variables would be able to predict the hs-CRP levels. Additional analysis by BMI groups (normal = < 25 kg/ $m^2$, overweight = 25.0 - 29.9 kg/$m^2$, and obese = > 30.0 kg/$m^2$) was also conducted.

## RESULTS

A total of 40.2% male (n = 49) and 59.8% female (n = 73) subjects with a mean age of 41.93 ± 8.26 years participated in this study. The majority of the subjects was non-academic staff, educated up to university or college, and married (Table 1). Male subjects had a significantly larger waist circumference (WC), WHR, high systolic and diastolic blood pressure, high TG levels and TC: HDL ratio than females. However, females had significantly higher HDL levels compared to males. There

**Table 1.** Socio-demographic characteristics of the subjects (n = 122)

| | *n* | *Percentage (%)* | *Mean ± SD* |
|---|---|---|---|
| Age (years) | 122 | | 41.93 ± 8.26 |
| Sex | | | |
| Male | 49 | 40.2 | |
| Female | 73 | 59.8 | |
| Position | | | |
| Academic staff | 37 | 30.3 | |
| Non-academic staff | 85 | 69.7 | |
| Educational level | | | |
| Primary school | 9 | 7.4 | |
| Lower secondary school | 3 | 2.5 | |
| Upper secondary school | 33 | 27.0 | |
| Pre-university / Matriculation / Form 6 | 6 | 4.9 | |
| University / College | 71 | 58.2 | |
| Marital status | | | |
| Single | 10 | 8.2 | |
| Married | 108 | 88.5 | |
| Divorced | 4 | 3.3 | |

**Table 2.** Anthropometric, blood pressure and biochemical characteristics of subjects (n = 122)

| | *Males (n = 49) (Mean ± SD)* | *Females (n = 73) (Mean ± SD)* | *p-value* |
|---|---|---|---|
| WC (cm)[b] | 90.37 ± 12.76 | 78.82 ± 11.83 | <0.001 |
| Hip circumference (cm)[b] | 100.64 ± 6.72 | 102.03 ± 10.18 | 0.366 |
| WHR[b] | 0.90 ± 0 .10 | 0.77 ± 0.07 | <0.001 |
| BMI (kg/m$^2$)[b] | 26.66± 3.46 | 26.91 ± 5.50 | 0.761 |
| Blood pressure (mmHg) | | | |
| Systolic[b] | 132.94 ± 16.33 | 124.51 ± 17.72 | 0.009 |
| Diastolic[b] | 84.06 ± 9.74 | 78.08 ± 10.55 | 0.002 |
| hs-CRP (mg/L)[a] | 2.70 ± 3.39 | 3.63 ± 4.87 | 0.962 |
| TC (mmol/L)[b] | 5.93 ± 1.13 | 5.66 ± 1.00 | 0.166 |
| LDL (mmol/L)[b] | 3.57 ± 0.94 | 3.31 ± 0.76 | 0.099 |
| HDL (mmol/L)[b] | 1.52 ± 0.29 | 1.74 ± 0.32 | <0.001 |
| TG (mmol/L)[b] | 1.80 ± 0.63 | 1.30 ± 0.75 | <0.001 |
| TC:HDL Ratio[b] | 3.92 ± 0.73 | 3.29 ± 0.63 | <0.001 |

*Notes:* [a]Mann-Whitney U-test; [b]Independent sample t-test
WC: waist circumference; WHR: waist-to-hip ratio; BMI: body mass index; hs-CRP: high-sensitivity C-reactive protein; TC: total cholesterol; LDL: low-density lipoprotein; HDL: high-density lipoprotein; TG: triglycerides

were no significant differences for both males and females in the measurement of hip circumference, BMI, TC levels, LDL levels, and hs-CRP levels. Female subjects had higher mean hs-CRP levels than males (Table 2). Since there was no difference in hs-CRP levels between genders, the data for both males and females were combined for further analysis.

The level of log hs-CRP was significantly different among subjects who were older, obese, having larger WC, and high ratio of waist-to-hip. There was a tendency for subjects who had high blood pressure and high lipid profiles to have higher log hs-CRP concentration (Table 3).

There were also significant positive correlations between log hs-CRP levels

**Table 3.** Log hs-CRP level based on age, nutritional status (WC, WHR, BMI), blood pressure, and lipid profiles (TC, LDL, HDL, TG, TC:HDL ratio)

| | *Log hs-CRP* | | |
|---|---|---|---|
| | *n* | *Mean ± SD* | *p-value* |
| Age (years) [a] | | | |
| 30 - 37 | 48 | 0.106 ± 0.507 | 0.036 |
| 38 – 45 | 20 | 0.156 ± 0.436 | |
| 46 - 55 | 54 | 0.354 ± 0.503 | |
| WC [b] | | | |
| Low risk | 63 | 0.085 ± 0.499 | 0.001 |
| At risk | 59 | 0.373 ± 0.470 | |
| WHR [c] | | | |
| Low risk | 111 | 0.188 ± 0.485 | 0.011 |
| At risk | 11 | 0.592 ± 0.572 | |
| BMI [a, d] | | | |
| Normal (< 25 kg/m$^2$) | 45 | 0.027 ± 0.497 | <0.001 |
| Overweight (25 – 29.9 kg/m$^2$) | 49 | 0.211 ± 0.447 | |
| Obese (> 30 kg/m$^2$) | 28 | 0.565 ± 0.444 | |
| Systolic blood pressure | | | |
| Normal (< 120 mmHg) | 44 | 0.119 ± 0.484 | 0.082 |
| Above normal (≥ 120 mmHg) | 78 | 0.284 ± 0.509 | |
| Diastolic blood pressure | | | |
| Normal (< 80 mmHg) | 58 | 0.153 ± 0.519 | 0.139 |
| Above normal (≥ 80 mmHg) | 64 | 0.289 ± 0.486 | |
| TC | | | |
| Desirable (< 5.2 mmol/L) | 37 | 0.175 ± 0.450 | 0.481 |
| Above desirable (≥ 5.2 mmol/L) | 85 | 0.246 ± 0.528 | |
| LDL | | | |
| Optimal (< 2.6 mmol/L) | 21 | 0.213 ± 0.459 | 0.915 |
| Above optimal (≥ 2.6 mmol/L) | 101 | 0.226 ± 0.516 | |
| HDL | | | |
| Average (1.0 – 1.5 mmol/L) | 51 | 0.296 ± 0.434 | 0.186 |
| High (> 1.5 mmol/L) | 71 | 0.173 ± 0.547 | |
| TG | | | |
| Normal (< 1.7 mmol/L) | 79 | 0.161 ± 0.533 | 0.061 |
| Above normal (≥ 1.7 mmol/L) | 43 | 0.340 ± 0.429 | |
| TC:HDL ratio | | | |
| Low risk (≤ 4.5) | 109 | 0.216 ± 0.514 | 0.609 |
| At risk (> 4.5) | 13 | 0.292 ± 0.431 | |

*Notes:* [a]Obtained from ANOVA test
[b]WC defined as, low risk, for male ≤ 90 cm; for female ≤ 80 cm; at risk, for male > 90 cm; for female > 80 cm (WHO, 2000)
[c]WHR defined as, low risk, for male ≤ 1.0; for female ≤ 0.85; At risk, for male > 1.0; for female > 0.85 (WHO, 2000)
[d]Classification based on Rawson *et al.* (2003)
WC: waist circumference; WHR: waist-to-hip ratio; BMI: body mass index; hs-CRP: high-sensitivity C-reactive protein; TC: total cholesterol; LDL: low-density lipoprotein; HDL: high-density lipoprotein; TG: triglycerides

(Mean ± SD = 0.22 ± 0.50 mg/L; 95% CI = 0.13 – 0.31; min – max = - 1.00 – 1.39) with age, WC, hip circumference, systolic and diastolic blood pressure, BMI, TC levels, LDL levels, TG levels, and TC:HDL ratio of the subjects (Table 4). This suggests that log hs-CRP correlated with all tested variables except WHR and HDL levels. After the

**Table 4.** Correlation between log hs-CRP with age, waist circumference (WC), hip circumference, waist-hip ratio (WHR), body mass index (BMI), blood pressure, and lipid profiles

| | *Log hs-CRP* | |
|---|---|---|
| | *r-value* | *p-value* |
| Age | 0.223 | 0.014 |
| WC | 0.374 | <0.001 |
| Hip circumference | 0.488 | <0.001 |
| WHR | 0.143 | 0.115 |
| BMI | 0.489 | <0.001 |
| Systolic blood pressure | 0.199 | 0.028 |
| Diastolic blood pressure | 0.227 | 0.012 |
| TC | 0.180 | 0.048 |
| LDL | 0.192 | 0.034 |
| HDL | -0.146 | 0.109 |
| TG | 0.236 | 0.009 |
| TC:HDL ratio | 0.261 | 0.004 |

*Notes:* WC: waist circumference; WHR: waist-to-hip ratio; BMI: body mass index; hs-CRP: high-sensitivity C-reactive protein; TC: total cholesterol; LDL: low-density lipoprotein; HDL: high-density lipoprotein; TG: triglycerides

assumptions of normality, linearity, multicollinearity and homocedascity had been met, the MLR analysis showed that BMI was the only significant predictor to influence log hs-CRP levels ($R = 0.489$, $R^2 = 0.239$, adjusted $R^2 = 0.232$, $F = 37.626$, $p < 0.001$). Besides, by BMI groups (< 25 kg/m$^2$, 25 – 29.9 kg/m$^2$, > 30 kg/m$^2$), post-hoc analysis (Tukey HSD) showed that log hs-CRP levels appeared to be significantly greater in overweight (Mean ± SD = 0.21 ± 0.45 mg/L) and obese subjects (Mean ± SD = 0.56 ± 0.44 mg/L) (ANOVA $p < 0.05$). In overweight and obese subjects, further analysis found that the majority were above desirable levels of TC (76.6%), above optimal level of LDL (84.4%) and average level of HDL (50.6%) while only some of them had above normal level of TG (46.8%) and at risk of TC: HDL ratio (15.6%). Also, in this study, among females, the majority (82.9%) of subjects who were overweight and obese had above optimal level of LDL.

## DISCUSSION

In the regression model, a study by Festa *et al.* (2001) found that BMI only explained 0.4% of the variability in CRP levels. Nevertheless, our finding showed that BMI accounted for about 24% of the variability ($R^2 = 0.239$) in log hs-CRP. The main reason might be due to the difference in number of predictors found in the regression model; this study only showed BMI as the main predictor while a previous study by Festa *et al.* (2001) found other predictors to have a share in the outcome such as waist circumference. In this study, other risk factors were also found to be significantly correlated with log hs-CRP; however, when all of these risk factors were combined with BMI, BMI become the one predictor that could predict log hs-CRP level. This finding provides an interesting insight on the correlation between BMI and the levels of CRP. In fact, in female subjects, a study has found two factors, namely TG

and BMI, to be the contributing factors that can influence hs-CRP and these variable explained 30% of the variability (Arena *et al.*, 2006). This may suggest that the risk of CVD among females was higher compared to males. As known, an increase in BMI is associated with increased prevalence of hypertension, diabetes, dyslipidemia and metabolic syndrome which can influence the level of hs-CRP (Nguyen *et al.*, 2008). Thus, hs-CRP levels can be lowered by undertaking some lifestyle modifications such as indulging in increased physical activity and weight loss programs, a similar intervention technique suggested to overweight and obese subjects.

In this study, at similar BMI, results have shown that male subjects had large WC and WHR while females had larger hip circumference. Females may have 'gynoid' or pear-like shape while males may tend to develop 'android' or apple-like shape due to different sex hormones. Estrogen in females may cause fat to be stored around the hips, butt and thighs (also called lower-body fat distribution) while testosterone in males may cause fat to be accumulated around the central abdomen (upper-body fat distribution) (Byrd-Bredbenner *et al.*, 2009). However, of great concern is the accumulation of fat at the abdominal region as studies have shown the strong influence of adiposity on the CRP circulation in the body (Festa *et al.*, 2001; Chandorkar, Vaidya & Patel, 2011). Moreover, the use of BMI, WHR and WC as the indicators of adiposity provide invaluable insight as several studies have shown strong correlations between CRP and BMI (Arena *et al.*, 2006; Yaqoob & Ferns, 2005), WHR and WC (Lin *et al.*, 2010) in females than males. For instance, Huffman *et al.* (2010) found stronger associations between BMI and WC with the level of hs-CRP among subjects with and without obesity. Of great significance is the link between BMI, WC and obesity and the development of cardiovascular diseases (Huffman *et al.*, 2010). Further analysis from this study revealed that log hs-CRP was higher among overweight and obese subjects. Thus, this study had shown that an increase in BMI and being overweight and obese can influence the level of this inflammatory marker and can be a significant predictor of cardiovascular diseases in both males and females.

BMI might play an important role in the development of chronic diseases. This is because changes in BMI can disrupt normal biological process in the human body. Increased BMI has been associated with adverse changes in serum lipids including HDL, TC, and TG in both the male and female (Wilsgaard & Arnesan, 2004). Changes in serum lipid profile in turn will affect the inflammatory marker as a previous study has shown significant correlations between CRP with TC, TG, and LDL levels (Santos *et al.*, 2005). Indeed, it can be explained as abnormal serum lipids might induce and accelerate the inflammatory process. Again, this study finding has shown that increasing BMI is closely related with an abnormal lipid profile level. It is shown that subjects who have a slightly high BMI level might also have poor control of their lipid profile which then can affect the CRP level.

As mentioned previously, males and females have different sites of fat deposition because of different sex hormones. The differences in sex hormones might also affect the metabolism of lipids and the inflammatory response. For example, a few studies have shown that females may have a high risk of developing cardiovascular events when their hs-CRP level is higher than LDL. Moreover, the chance of surviving a cardiovascular event becomes slim as these markers continue to rise (Ridker *et al.*, 2002). In fact, both LDL and CRP can have a significant impact on human health as high levels of LDL and CRP can increase the risk of chronic disease such as coronary heart disease and stroke.

Thus, instead of having a high level of hs-CRP alone, the combination of this marker with a high level of LDL can hasten disease progression. However, the risk may not only increase among females but also slowly increase among the males since this study found that the majority of the male subjects had above optimal level of LDL. Nissen *et al.* (2005) noted that a reduction in both LDL and CRP is important to alleviate the progression rate of atherosclerosis. In our study, we also found that both males and females had different levels of TG and TC: HDL ratio. The difference may be due to the lifestyle practice of subjects. The correlation of TG and this inflammatory marker has been described in previous studies (Piché *et al.*, 2005; Khera *et al.*, 2005). They found a significant correlation between CRP and TG in both male and female subjects. Besides, another study found the levels of CRP and TC: HDL ratio to be the strongest independent factor for the development and incidence of coronary diseases (Koenig *et al.*, 2004). This may suggest that an increase in TG and TC: HDL ratio might promote vascular inflammation which may then cause blocking in the blood vessel. The risk of getting CHD due to increases in TG and TC: HDL ratios can be high among male subjects as the majority of males who were overweight and obese had above normal TG level and a high percentage of at risk TC: HDL ratios compared to the females in this study. For both the male and female genders, BMI still showed direct or indirect contribution to the development of chronic disease by increasing other potential risk factors. Thus, this makes the BMI the most suitable predictor for CVD risk marker.

Age was found to be significantly correlated with log hs-CRP in this study. Aging has become a significant risk factor in men at age 45 years and older and at 55 years and older among women for CHD (National Institutes of Health, 2001). Research has shown that when men and women grow older, the risk of CHD rises and might reflect the steady progression of atherosclerosis (Ford, Giles & Dietz, 2002). CRP is one of the acute phase reactants that might play a direct role in the development of atherogenesis (Pearson *et al.*, 2003). The level of CRP has been shown to increase with age and its measurement might be more useful in the middle-aged (Kritchevsky, Cesari & Pahor, 2005).

Furthermore, systolic and diastolic blood pressures were also found to be correlated with log hs-CRP in this study and our finding is supported by a previous study (Prospective Studies Collaboration, 2002). Increased blood pressure may promote vascular inflammation by modulation of mechanical stimuli from pulsatile blood flow. At the vessel wall, these multiple stimuli might induce the production of inflammatory markers which may then promote atherogenesis (Shafi Dar *et al.*, 2010). In contrast, Santos *et al.* (2005) found that blood pressure alone may not influence circulation of CRP levels. However, when blood pressure is combined with the presence of central obesity, the levels of CRP were increased about two-times (Santos *et al.*, 2005). In another analysis, we found that the majority of subjects who were at risk of CVD based on waist circumference had above normal systolic (81.4%; $\chi^2 < 0.001$) and diastolic (67.8%; $\chi^2 = 0.001$) blood pressures. Besides, the majority of subjects who were overweight and obese also had systolic (74.0%) and diastolic (59.7%) blood pressure levels above normal. This finding shows that an increase in BMI and central adiposity could be the main factors to increase blood pressure which then might influence the inflammatory markers. In addition, a cohort study has shown that, after 11years of follow up among middle-aged men, those with hs-CRP ≥ 3.0 mg/L were 2.8 times more likely to develop hypertension (Niskanen *et al.*, 2010). This suggests that the circulation of CRP can be an independent or dependent factor in the progression of cardiovascular disease.

The main limitation of this study was the sample size. This small sample size had resulted in only BMI being found to be the significant predictor for log hs-CRP level; also no significant difference of log hs-CRP was found between males and females. However, this study had used biochemical data by performing a blood test which is believed to give more valuable results. It is recommended that a larger sample size be used in future studies. In addition, this study only focused on some factors that contributed to the risk markers. Other factors such as metabolic syndrome have also been found to be associated with the risk marker but were not investigated in this study. The presence of metabolic syndrome is believed to influence the increasing level of hs-CRP. It is recommended that other contributing factors be studied as an extension of this study.

## CONCLUSION

This study suggests that age, body fat distribution (WC, hip circumference, BMI), blood pressure, and lipid profiles (TC, TG, LDL, TC:HDL ratio) are associated with circulating levels of CRP. However, among these factors, only BMI was found to be a significant predictor of hs-CRP concentration. Thus, in Malaysia, CVD events amongst adults can be reduced by promoting a healthy level of BMI and changing the risk factors associated with hs-CRP.

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