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Relationship between Platelet counts and High Sensitivity C-reactive protein according to Body Mass Index of Korean adolescents

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Abstract

The purpose of this study is to compare the platelet counts (PLT) and high sensitivity CRP(hs-CRP) according to the body mass index (BMI) of Korean adolescents. This study analyzed secondary data of KNHANES V-3, 2015, The Fifth Korea National Health and Nutrition Examination Survey (Korea Center for Disease Control and Prevention). A cross-sectional study was conducted on 488 male and female, who were aged 13-18years. The subjects were categorized into normal BMI 304 subject(68.2%), overweight 56 subject (12.6%) and obese groups 86 subject(19.3%). The subjects were analyzed by questionnaires and blood tests. Data were analyzed using descriptive statistics, Chi-square test, ANOVA, Scheffe's test, Pearson correlation coefficient and multiple regression analysis(SPSS 24.0). The major findings of this study were as follows. First, the obesity of the subjects was found to be 19.3%. Second, PLT was higher in obese group (mean = 307.33) and overweight group (mean = 296.901) than normal group (mean = 277.48). hs-CRP did not differ according to BMI. Third, BMI has a significant positive correlated with PLT and hs-CRP. In addition, PLT appeared to be a factor affecting the BMI of the subjects. We want to provide a basis data for the prevention and management of obesity intervention program for adolescents

Keywords: Adolescents, body mass index, C-reactive protein, Platelet counts

INTRODUCTION

Obesity is a widespread medical problem affecting children and adolescents in many countries around the world[1]. It is estimated that in 2015 approximately 2.3 billion people are overweight and more than 700 million are obese[2]. Also, Korean teenagers' obesity rate increased rapidly[3], especially extreme obesity rate shows a significant increase(over 1%) and this kind of phenomenon correspond with a growing trend of occurrence which is obese related diseases[4]. In many populations, the average BMI has been rising by a few percent per decade, thus fuelling the concern about the effects of increased adiposity on health[5]. It is even more important to prevent childhood obesity, since this unfavorable metabolic, inflammatory state can persist year after year, resulting in serious consequences in adulthood[6].

The obesity is associated with platelet activation and systemic inflammation[7], PLT markers in obese subjects, obesity may be associated with elevated platelet counts in subject with chronic inflammation[8]. Raised BMI is a major risk factor for noncommunicable diseases such as:cardiovascular diseases(mainly heart disease and stroke)[9]. Platelet activation and aggregation are among the main processes, In heart disease, correlations between platelet and white blood cells counts appeared[10]. Higher PLT are associated with adverse clinical outcome in patients with ST-elevation myocardial infarction[11]. Investigation of its association with PLT revealed a statistically significant correlation between BMI with indices of PLT[12]. it has been shown that measurements of central adiposity(e.g. waist circumference, waist-to-hip ratio, height-to-height ratio and abdominal height) are more strongly associated with several diseases or poor health that of the BMI in both genders [13].

The PLT and platelet activation are associated in patients with chronic inflammation, such as patients with essential thrombocytosis[14]. Cytokines, such as Interleukin-6(IL-6), originating from adipose tissue, have a fundamental role in the pathogenesis of atherothrombosis[15]. The association between platelet count and platelet activation has rarely been studied in obese subjects[15], In particular, studies on adolescents are rare. In this study, we investigated the relationship between obesity and PLT in adolescents.

The CRP is a protein produced by the liver and is used as a predictor of infectious, inflammatory and necrotic responses due to rapid increase in acute inflammatory response[16]. Also, CRP is currently recognized not only as an inflammatory marker but also as an epidemiological risk marker of cardiovascular diseases[17]. Obesity is one of the most common and prevalent conditions that promote this low grade inflammatory environment within the body[18]. The CRP is the first cytokine to be elevated in inflammatory conditions such as obesity. A strong relationship between obesity and CRP has been observed in populations. This is because of the inflammatory changes in obese individuals. The CRP in serum decreases significantly after massive weight reduction[19].

The pathophysiological mechanisms linking obesity to elevated levels of CRP are well recognized in obesity, the

accumulation of free fatty acid intermediates activates pro-inflammatory serine kinase cascades[20].

Adipose tissue, the endocrine organ contributes to the development of inflammation process by secreting various pro-inflammatory cytokines and adipokines(i.e. IL-6, TNF-α, leptin, CRP etc). Certain cytokines influence food intake through direct effect of hypothalamus[21]. The previous paper relating obesity with CRP, a great deal of research has been undertaken in order to elucidate the association. There is speculation whether, in the adult population, the increase in CRP is a consequence or if it is directly involved in the pathophysiology of chronic diseases. Since the prevalence of degenerative diseases is low in childhood and adolescent, research into the markers of inflammation in this age group is of great value to understanding these questions[6]. Thus we study to correlate between low grade inflammatory marker CRP in adolescents with different BMI categories. CRP is detected in the acute phase inflammatory reaction, has a measurement limit of 3-8 mg/L, In order to assess the risk of cardiovascular disease by determining chronic inflammation in healthy persons, a much lower limit of 0.2mg /L is required[22]. In this study, we analyzed hs-CRP, which is measured even in the low range of 0-5mg/L. Early treatment of overweight individuals can reduce the incidence of comorbidities in adulthood. Further research is needed in order to demonstrate the association of inflammatory markers related to obesity during childhood and adolescence[6].

The Fifth Korea National Health and Nutrition Examination Survey(KNHANES V-3, 2015) is representative of nationwide survey data. Therefore, it is appropriate sample data to construct domestic reference value. In this study, we tried to suggest a fundamental data of adolescent obesity prevention and management program by checking a correlation of BMI, PLT, CRP, targeting a adolescent.

METHODS

Research Design

This study is a descriptive survey study which attends a secondary analysis of The Six Korea National Health and Nutrition Examination Survey's raw data to check the differences of PLT and hs-CRP depends on Korean adolescents' obesity degree.

Samples and setting

Subject of this study is adolescents(13-18y) and data is based on 'The Sixth Korea National Health and Nutrition Examination Survey (KNHANES Ⅵ-3) 2015' which Ministry of Health and Welfare and Korea Centers for Disease control and prevention conducted.

The sampling framework of the Korea National Health and Nutrition Examination Survey used the most recent Population and Housing Census data available at the time of sample design. This enables the representative sample to be extracted from the target population in the Republic of Korea. In 2015, 192 specimens were extracted, Using a bellwether sampling, we extracted 20 subject family for every enumeration district from the extracted samples.

A total of 7,380 subjects were included in the Sixth Korea National Health and Nutrition Survey. Among them, 13-18 years old were selected as subjects, except those who were under 13 years old, 19 years old and above, and who were missing. The subjects were classified as normal weight in 304(68.2%), overweigh in 56(12.6%) and obese in 86(19.3%).

Measuring methods

BMI

Height and weight were measured after overnight fasting with participants shoeless and wearing a lightweight gown. BMI was calculated as weight(kg) divided by the square of the height(m). Based on this, the BMI weight status cartegories are Normal weight($BMI<23kg/m^2$), Overweight($23<BMI<25kg/m^2$), and Obese ($BMI\geq25kg/m^2$)[23]. The representative method to measure obesity is BMI, and the way to change BMI into clinical obesity category has slight difference following weight distribution of west and east[24], Based on BMI: over 95 percetile or over $25kg/m^2$ obesity is analyzed for all range of subject[25].

Blood measurement

Blood sampling was performed in the morning after fasting for 8 hours or more, and cubital vein was punctured using a vacutainer needle and blood was drawn into a vacuum tube. The PLT measurement is an Hydrodynamic focusing DC detection test method, Cellclean (Sysmex/Japan) for reagent and it is analyzed by the equipment; XN-9000(sysmex/Japan). The hs-CRP measurement is an Immunoturbidimetry test method, Cardiac C-Reactive Protein High Sensitive (Roche/Germany) for reagent and it is analyzed by the equipment; Cobas (Roche/Germany)

Data analysis

Collected data was analyzed by SPSS WIN 24.0 program(SPSS Inc. Chicago, IL, USA). The general characteristic of subjects was analyzed by descriptive statistics. The difference of PLT and hs-CRP depends on subject's obesity degree was analyzed by $\chi 2$-test, ANOVA, Scheffe's test. The relation between subject's BMI, PLT and hs-CRP was analyzed by Pearson correlation coefficient. Factors influencing BMI were multiple regression analysis. When the value of p is under 0.05, it is statistically meaningful.

Ethical consideration

The Korean National Health and Nutrition Examination Survey used in this study is government designated statistics (Approval No. 117002) based on Article 17 of the Statistical Law, and recipient's anonymity and confidentiality were guaranteed by collecting their private information in serial number which is unable to distinguish and we pactionally conducted a 2015 annual survey(48weeks a year).

In this study, according to the raw data open and care regulation of Korea Centers for Disease Control and Prevention, after the researcher got confirmation for using data(June 20, 2017), the data was provided and we used them.

RESULTS

General characteristics of subject

The mean age of the subjects was 15.41 years old, male 54.9%, female 45.1%, middle school 52.7%, high school 47.3%. Subjective health status was very good 14.4%, good 42.2%, average 39.7% and poor 3.6%.

The mean height of the subjects was 165.86cm and the weight was 60.92kg. The mean BMI was 21.90kg/m². Specifically, normal weight 68.2%, overweigh 12.6% and obese was 19.3%. The mean PLT of the subjects was 285.81 Thous/uL and hs-CRP was 0.76mg/L(Table 1).

PLT and hs-CRP differences according to obesity level

The PLT of the subjects was higher in the obese(mean=307.33) and overweight(mean=296.90) than the normal group(mean=277.48)($F=9.052$, $p<.001$).

The hs-CRP of the subjects was not significantly different according to obese (mean=1.12), overweight(mean=0.54) and normal group(mean=0.70)($F=2.335$, $p=.098$) (Table 2).

Table 1: General characteristics of subject(N=488)

Variables	Category	Mean±SD n(%)	Acquired score range
Age(years)		15.41±1.67	13~18
Gender	Male	268(54.9)	
	Female	220(45.1)	
School grade	Middle school	257(52.7)	
	High school	231(47.3)	
Subjective health status	Very good	64(14.4)	
	Good	187(42.2)	
	Average	176(39.7)	
	Poor	16(3.6)	
Height(cm)		165.86±8.21	142~189
Weight(kg)		60.92±13.49	30~134
BMI(kg/m²)		22.04±3.97	13~41
	Normal weight(BMI<23.0)	304(68.2)	
	Overweigh(23<BMI<25)	56(12.6)	
	Obese(BMI≥25)	86(19.3)	
PLT(Thous/uL)		285.81±59.25	53~583
hs-CRP(mg/L)		0.76±1.71	0~20

Table 2: PLT and hs-CRP differences according to obesity level(N=488)

Variables	Category	Normal weigh G¹ (BMI<23.0) 304(68.2%) Mean±SD n(%)	Overweigh G² (23<BMI<25) 56(12.6%) Mean±SD n(%)	Obesity G³ (BMI≥25) 86(19.3%) Mean±SD n(%)	χ2/ F (p value)
Gender	Male	161(53.0)	32(57.1)	53(61.6)	2.138(.343)
	Female	143(47.0)	24(42.9)	33(38.4)	
School grade	Middle school	165(54.3)	29(51.8)	37(43.0)	3.400(.183)
	High school	139(44.7)	27(48.2)	49(57.0)	
Subjective health status	Very good	49(16.2)	9(16.1)	6(7.1)	9.417(.151)
	Good	131(43.4)	22(39.3)	34(40.0)	
	Average	110(36.4)	25(44.6)	41(48.2)	
	Poor	12(4.0)	0(0.0)	4(4.7)	
Height(cm)		165.40±8.31	165.79±8.04	167.52±7.80	2.242(.107)
Weight(kg)		54.65±7.91	65.61±6.54	80.07±13.27	273.066(<.001) 1<2<3
BMI(kg/m²)		19.91±1.93	23.82±0.58	28.41±3.15	559.266(<.001) 1<2<3
PLT(Thous/uL)		277.48±55.87	296.90±63.16	307.33±62.15	9.052(<.001) 1<2,3
hs-CRP(mg/L)		0.70±1871	0.54±0.83	1.12±1.49	2.335(.098)

Table 3: Correlation between BMI, PLT and CRP(N=488)

Variables	BMI	PLT	hs-CRP
	r(p value)		
BMI	1		
PLT	.203(<.001)	1	
hs-CRP	.126(.012)	.042(.408)	1

Correlation between BMI, PLT and hs-CRP

There was a significant positive correlation between the subject's BMI and PLT (r=0.203, p<.001), hs-CRP(r=0.126, p=.012) (Table 3).

Factors affecting BMI in the subjects

In order to confirm the relative influence of factors influencing BMI, we conducted a hierarchical regression analysis (Table 4) after controlling the gender, school grade,

Table 4: Factors affecting BMI in the subjects(N=488)

	Model I		Model II	
	β	t (p value)	β	t (p value)
Gender	-.175	-5.132(<.001)	-.183	-5.019(<.001)
School grade	.057	1.669(.096)	.079	2.177(.030)
Subjective health status	.703	20.578(<.001)	.684	18.529(<.001)
PLT			.111	3.011(.003)
hs-CRP			-.009	-.247(.805)
R^2	.684		.712	
Adj R^2	.468		.507	
F (p)	193.383(<.001)		99.396(<.001)	

and subjective health status that could affect the subject's BMI. In the model I, the control variable was used. In model II, the model I was used as the independent variable PLT and hs-CRP.

Model I indicates the degree of influence of the control variables on the subject's BMI. The regression model fit was statistically significant at 193.383 (p<.001). Gender(β=-.175, p<.001) and subjective health status (β=.703, p<.001) were found to affect the BMI of the subjects.

The regression model fit of model II was 99.396(p<.001), and PLT(β=.111, p=.003) was added as a variable that affected the subject's BMI. The explanatory power was improved by 3.9% over the model I.

DISCUSSION

The study was to investigate the relationship between PLT and hs-CRP according to BMI in Korean adolescents.

In this study, overweight and obese were found to be 12.6% and 19.3%, One out of five patients was obese. According to the statistics issued by the world health organization[26], obesity has more than doubled in the world in the last three decades, and based on relates reports issued in 2014, from among 109 billion overweight adults over the age 18,600 million were obese. For children is not much different in that in 2013 around 42 million children under 5 were identified and reported as overweight or obese. Also, adolescent obesity needs prevention and care because it mostly proceed to adult obesity[27], Supported the results of this study.

Definition of obesity is excessive fatness and excessive accumulation of fat is connected with obesity related complication occurrence[28]. Childhood obesity should be taken seriously because it is a risk factor for adulthood disease and thus affects morbidity and mortality in later life[29].

In this study, BMI and PLT were correlated, and PLT was higher in obese and overweight than normal weight. In addition, PLT appeared to be a factor affecting the BMI of the subjects. The PLT is associated with obesity[7] and PLT increased with BMI in both genders[8], The relationship between central and general adiposity as an inflammation factor, and higher count of platelets count in both genders[12].

The obesity is associated with low-grade subclinical and smoldering inflammation [15]. The study demonstrate an association between obesity and platelet counts in subject probably due to higher body fat mass[8], Similar results were obtained in this study.

The obesity and IL-6 levels were found to be related, a large proportion of IL-6 in the circulation originates from the adipose tissue that in turn may contribute to atherogenesis and thrombosis, by promoting inflammation. One of the proposed mechanisms for IL-6 contribution to atherogenesis and thrombosis is its effect on platelets, fibrinogen concentrations, and coagulation[15]. IL-6, in addition to other interleukins, plays a crucial role in the proliferation of megakaryocyte progenitors and acts synergistically with thrombopoietin and stem cell factor in stimulating megakaryocytopoiesis[30], Supported the results of this study.

On the other hand, The association between obesity related diseases has been documented in previous studies, increase in tertiles of abdominal height would significantly be associated with an increase in the mean number of platelets in female, but no association was observed in male[31]. The number of platelets increased with BMI in both genders. But, only in female, the number of platelets was significantly high in obese and overweight subjects[12]. The study, the female had higher PLT than the male, The mechanisms of sex-related differences in the PLT remain unknown. However, female begin to have a higher PLT than male only after around 14years of age, supporting the hypothesis that puberty exerts an effect[32]. The study suggest that separate reference intervals for male and female are required for analyzing their PLT, As such, there are some limitations to the present study[7].

In this study, we investigated the difference of platelets according to obesity level. Therefore, there is a limit to understanding differences according to gender. In future studies, it is necessary to identify the difference between the

obesity level and other platelet counts by gender and to ascertain the reason.

The elevated PLT found in obese subject are secondary to the presence of a chronic inflammation as evident by elevated high sensitivity-CRP levels[8].

In this study, there was a positive correlation between BMI and hs-CRP, however there was no difference in hs-CRP between obese, overweight, and normal weight. The Previous research identified an increased CRP in obese versus lean subjects, BMI showed significantly positive correlation with serum CRP[33]. Significant correlations between both BMI and hs-CRP concentration were noted, BMI as an indicator of adipose tissue accumulation are significantly related to CRP concentration, Also, The obesity group exhibited greater hs-CRP concentrations than the control group[34].

The study carried out research with children(9-11 years old) to evaluate CRP concentrations and their relationship with adiposity and cardiovascular risk factors, There was a relationship between CRP concentration and ponderal index[35]. The study published their analysis of the data from 5,305 individuals aged 6 to 18 years, finding that 90% of them had CRP concentrations <2.1mg/L. The percentage of participants with CRP > 2.1 mg/L increased to the extent that the BMI increased[36], Previous studies supported the results of this study in which there was a correlation between BMI and hs-CRP.

In a study comparing 100 obese children with 50 not obese children, compared to the control group, the obese children had higher levels of CRP[37]. The study comparing a group of overweight children and adolescents with a group without overweight, hs-CRP concentrations were higher in the group with overweight or obesity[6]. The study assessed CRP concentration in 3,512 children and adolescents (8-16 years), They observed a greater prevalence of elevated CRP results(>2.2 mg/L) among overweight or obese children and adolescents, when compared with those whose BMI was below the 85th percentile. Among those children and adolescents whose BMI was above the 85th percentile, 20.6% of the males and 18.7% of the females exhibited elevated CRP values[38], Unlike the usual studies in which hs-CRP levels were significantly different according to normal, overweight, and obesity groups, In this study, there was no difference in hs-CRP concentration. For this reason, In obese people, with BMI above 30kg/m², inflammation is considerably increased[39]. The same adipocytokines, released from adipose tissue, also influence leukocyte count and interfere with their function[40]. In this study, it is thought that BMI of 25 or more is the standard of obesity, which is different from previous studies. Therefore, it is necessary to confirm the difference of hs-CRP by standardizing the standard of obesity, and it is also necessary to confirm whether there is a difference in the effect of BMI on hs-CRP according to race.

There is some sort of association between the increased number of immune cells and obesity as a result of a chronic inflammatory condition which is created by an increase in the production of cytokines by adipose tissue[41]. In the obese group, with increasing BMI, CRP concentration rise four folds, Simultaneous increase in CRP levels in the presence of excessive fat content of the body, This might be caused due to inflammatory conditions prevailing in the presence of increased adipose tissue[34].

This could be attributed to certain mechanisms associated to the pathogenesis of obesity, such as low grade inflammation[42]. The study yielded high serum CRP levels in obese individuals. The study suggest that obesity may cause activation of certain inflammatory mechanisms and increased cytokine secretion from adipose tissue, which in turn increases the hepatic secretion of CRP[19].

In this study, it is meaningful to confirm the difference between PLT and hs-CRP according to BMI of Korean adolescent. The results of this study are intended to provide basic data for prevention and management of obesity in adolescent.

CONCLUSION

In this study, the BMI of Korean adolescents showed a significant correlation with PLT and hs-CRP. The PLT is an influencing factor of BMI, and PLT of the subjects was higher in the obese, overweight than the normal weight. The hs-CRP of the subjects was not significantly different according to obese, overweight, and normal weight. In conclusion, PLT and CRP intervention should be included in adolescent obesity prevention and management program.

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REFERENCES

[1] Mirmiran, P., Sherafat-Kazemzadeh, R., Jalali-Farahani, S., and Azizi, F., 2010, “Childhood obesity in the Middle-East: a review,” East Mediterr Health J., 16(9), pp. 1009–17.

[2] Wellen, K. E., and Hotamisligil, G. S., 2003, “Obesity-induced inflammatory changes in adipose tissue,” The Journal of clinical investigation., 112(12), pp.1785–1788.

[3] Park, Y.S., Lee, D. H., Choi, J.M., Kang, Y.J., and Kim CH., 2004,“Trend of obesity in school age children in seoul over the past 23 years,” Korean J Pediatr., 47(3), pp.247-257.

[4] Chung, S., 2010,"Management of severe obesity in children and adolescents," J Korean Soc Pediatr Endocrinol., 15(2), pp.85-92.

[5] WHO Global NCD Infobase Team, 2005, "Surveillance of chronic disease risk factors: country level data and comparable estimates," Geneva,World Health Organization.

[6] Brasil, A. R., Norton, R. C., Rossetti, M. B., Leão, E., and Mendes, R. P., 2007, "C-reactive protein as an indicator of low intensity inflammation in children and adolescents with and without obesity," J Pediatr (Rio J), 83(5), pp.477-480.

[7] Riyahi, N., Tohit, E. R. M., Thambiah, S. C., and Ibrahim, Z., 2017, "Platelet-related cytokines among normal body mass index, overweight, and obese Malaysians" Asia Pacific Journal of Clinical Nutrition, pp.1-14. doi: 10.6133/apjcn.032017.01

[8] Samocha-Bonet, D., Justo, D., Rogowski, O., Saar, N., Abu-Abeid,S., Shenkerman,G., Shapira, I., Berliner, S., and Tomer, A., 2008, "Platelet Counts and Platelet Activation Markers in Obese Subjects," Hindawi Publishing Corporation Mediators of Inflammation, Article ID 834153, pp.1-6. doi:10.1155/2008/834153

[9] Bastien, M., Poirier, P., Lemieux, I., Despres, J.P., 2014, "Overview of epidemiology and contribution of obesity to cardiovascular disease," Prog Cardiovasc Dis, 56(4), pp.369–81. doi: 10.1016/j.pcad.2013.10.016.

[10] Coban, E., Ozdogan, M., Yazicioglu, G., and Akcit, F., 2005, "The mean platelet volume in patients with obesity," Int J Clin Pract, 59(8), pp.981–2.

[11] Hung, Q. L., Ajay, J. K., Sabina, A. M., Jacki, B., Christopher, P. C., Eugene, B., and Michael, C. G., 2006, "Association of platelet counts on presentation of clinical outcomes in ST elevation myocardial infarction," American Journal of Cardiology, 98(1), pp.1–5.

[12] Leila, J., and Asghar, S., 2017, "Association Between Obesity, White Blood Cell and Platelet Count," Zahedan J Res Med Sci, 9(2), e4955.

[13] Meisinger, C., Doring, A., Thorand, B., Heier, M., and Lowel, H., 2006, "Body fat distribution and risk of type 2 diabetes in the general population: are there differences between men and women? The MONICA/KORA Augsburg cohort study," Am J Clin Nutr, 84(3), pp.483–9.

[14] Michiels, J.J., Berneman, Z., Schroyens, W., Finazzi,G., Budde, U., and van Vliet, H. H., 2006, "The paradox of platelet activation and impaired function: platelet-von Willebrand factor interactions, and the etiology of thrombotic and hemorrhagic manifestations in essential thrombocythemia and polycythemia vera," Seminars in Thrombosis and Hemostasis, 32(6), pp.589–604.

[15] Yudkin, J. S., Kumari,M., Humphries, S. E., and Mohamed-Ali, V., 2000, "Inflammation, obesity, stress and coronary heart disease: is interleukin-6 the link?" Atherosclerosis, 148(2), pp.209–214.

[16] Ledue, T.B., and Rifai, N., 2003, "Preanalytic and analytic sources of variations in C-reactive protein measurement: implications for cardiovascular disease risk assessment," Clin Chem, 49(1), pp.1258-71.

[17] Sekitani, Y., Hayashida, N., Kadota, K., Yamasaki, H., Abiru, N., Nakazato, M., Maeda, T., Ozono, Y., and Takamura, N., 2010, "White blood cell count and cardiovascular biomarkers of atherosclerosis," Biomarkers, 15(2),pp.454-460

[18] Shiwaku, K., Anuurad, E., Enkhmaa, B., Kitajima, K., and Yamane, Y., 2004, "Appropriate BMI for Asian populations," Lancet, 363(1), pp.1077.

[19] Choi, J., Joseph, L.,Pilote, L.,2013,"Obesity and C-reactive protein in various populations:a systematic review and meta-analysis,"Obesity Rev. 14(3), pp.232-44.

[20] Rocha, V.Z., 2009,"Libby P.Obesity, inflammation and athero sclerosis," Nature Rev Cardiol, 6(1), pp.399-409.

[21] Kershaw, E. E., and Flier, J. S., 2004, "Adipose tissue as an endocrine organ," J Clin Endocrinol Metab, 89(6),pp.2548-56.

[22] Dominici, R., Luraschi, P., and Franzini, C., 2004, "Measurement of C-reactive protein: Two high sensitivity methods compared." J Clin Lab Anal, 18(3), pp.280-4.

[23] KorMedi (2009, November 20), Retrieved June 20, 2012, Available at: http://www.kormedi.com/care/center/Default.aspx?category=041010&idx=188

[24] Koo, H. J., Lee, S. M., Lee, S. P., and Han, E. N., 2014,"Association of body mass index with asthma, allergy rhinitis, and atopic dermatitis among adolescents in Incheon, South Korea," Allergy Asthma Respiratory Disease, 2(4),pp.243-50.

[25] Korea Youth Risk Behavior Web-Based Survey, 2014,"2014 Korea Youth Risk Behavior Web-Based Survey," Available at: http://yhs.cdc.go.kr/data/news.asp?board_name=tbl_data_board&code=repor

[26] World Health Organization, 2008, "2008-2013 action plans for the global strategy for the prevention and control of non-communicable diseases 2008," Available from: http://www.who.int/nmh/publications/9789241597418/en/8T.

[27] Park, H. W., and Chung, S., 2013, "Body Composition and Obesity in Korean Adolescents and its Impact on

Diabetes Mellitus," Korean J Obes, 22(3), pp.137-44. http://dx.doi.org/10.7570/kjo.2013.22.3.137

[28] Aronne, L. J., and Segal, K.R., 2002,"Adiposity and fat distribution outcome measures: assessment and clinical implications," bes Res, 10(Suppl), pp.14-21.

[29] Weiss, R., and Caprio, S., 2005, "The metabolic consequences of childhood obesity,"Best Pract Res Clin Endocrinol Metab, 19(3),pp.405-19.

[30] Lazzari,L., Henschler,R., Lecchi,L., Rebulla, P., Mertelsmann, R., and G. Sirchia, 2000, "Interleukin-6 and interleukin-11 act synergistically with thrombopoietin and stem cell factor to modulate ex vivo expansion of human CD41+ and CD61+ megakaryocytic cells," Haematologica, 85(1), pp.25–30.

[31] Charles, L.E., Fekedulegn, D., McCall, T., Burchfiel, C.M., Andrew, M.E., and Violanti, J. M., 2007, "Obesity, white blood cell counts, and platelet counts among police officers," Obesity(Silver Spring), 15(11), pp.2846–54.

[32] Biino, G., Santimone, I., Minelli, C., Sorice, R., Frongia, B., Traglia, M., et al., 2013, "Age and sex-related variations in platelet count in Italy: a proposal of reference ranges based on 40987 subjects' data," PLoS One, 8(1),e54289.

[33] Alam, F., Memon, A.S., and Fatima, S.S., 2015, "Increased Body Mass Index may lead to Hyperferritinemia Irrespective of Body Iron Stores," Pak J Med Sci, 31(6), pp.1521-1526.

[34] Płaczkowska, S., Pawlik-Sobecka, L., Kokot, I., Sowiński, D., Wrzosek, M., and Piwowar, A., 2014, "Associations between basic indicators of inflammation and metabolic disturbances," Postepy Hig Med Dosw(online), 68(1), pp.1374-82.

[35] Cook, D.G., Mendall, M.A., Whincup, P.H., Carey, I.M., Ballam, L., Morris, J. E., et al., 2000,"C-reactive protein concentration in children: relationship to adiposity and other cardiovascular risk factors," Atherosclerosis, 149(1), pp.139-50.

[36] Ford, E.S., Galuska, D.A., Gillespie, C., Will, J.C., Giles, W.H., Dietz, W.H.. 2001, "C-reactive protein and mass index in children: findings from the Third National Health and Nutrition Examination Survey, 1988-1994," J Pediatr, 138, pp.486-92.

[37] Iannuzzi, A., Licenziati, M. R., Acampora, C., Salvatore, V., DeMarco, D., Mayer, M.C., et al., 2004,"Preclinical changes in the mechanical properties of abdominal aorta in obese children," Metabolism, 53(1),pp.1243-6.

[38] Visser, M., Bouter, L.M., McQuillan, G.M., Wener, M.H., Harris, T.B., 2001, "Lowgrade systemic inflammation in overweight children," Pediatrics, 107, E13.

[39] Farhangi, M.A., Keshavarz, S. A., Eshraghian, M., Ostadrahimi, A., Saboor-Yaraghi, A. A., 2013, "White blood cell count in women: relation to inflammatory biomarkers, haematological profiles, visceral adiposity, and other cardiovascular risk factors," J. Health Popul, Nutr, 31(2), pp.58-64

[40] Van Greevenbroek, M. M., Schalkwijk, C. G., and Stehouwer, C. D., 2013, "Obsity-associated low-grade inflammation in type 2 diabetes mellitus: causes and consequences," Neth. J. Med., 71(4), pp.174-87.

[41] Womack, J., Tien, P.C., Feldman, J., Shin,J.H., Fennie, K., Anastos, K., Cohen, M. H., Bacon, M. C., and Minkoff, H., 2007, "Obesity and immune cell counts in women. Metabolism," 56(7), pp.998–1004. doi: 10.1016/j.metabol.2007.03.008.

[42] Lecube, A., Hernandez, C., Pelegri, D., and Simo, R., 2008, "Factors accounting for high ferritin levels in obesity," Int J Obesity, 32(11). pp.1665-1669.