

Risk of Hypertension in Overweight Adolescents in Pangkalpinang, Indonesia

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ABSTRACT

Background: Adolescents are the changes of children to be adults. Gender, family history of disease, age, sodium intake and physical activity affect the prevalence of hypertension. Adolescence is the time change from children into adults. Gender, family history of disease, body mass index or BMI according to age, sodium intake and physical activity affect the prevalence of hypertension in adulthood. Hypertension disorders in teenagers most will settle on adulthood. This study aimed to analyze risk factors of hypertension in adolescent aged 15-17 years with over nutritional status.

Subjects and Method: This was an observational analytic study with cross sectional design. This was conducted in Pangkalpinang, Bangka Belitung Indonesia. A total of 120 students in grade X-XI in four high schools were selected by fixed-exposure sampling. BMI measurement used antropometri. Physical activity questionnaire used International Physical Activity Questionnaire. Sodium intake was measured with a food frequency questionnaire. Blood pressure was measured by using a sphygmomanometer. Data analysis was using a multiple linear regression.

Results: Gender ($b= 5.77$; $p= 0.017$) and nutritional status ($b=4.85$; $p= 0.001$) were positively associated with hypertension. Sodium intake ($b= 0.01$; $p= 0.076$), family disease history ($b=-1.73$; $p= 0.481$), and physical activity ($b= -0.01$; $p = 0.592$) were negatively associated with hypertension. Multiple linear analysis showed adjusted $R^2 = 35.6\%$.

Conclusions: Young men have a higher average blood pressure than women of 5.77 mmHg. Any increase of 1 kg/m² equivalent BMI will raise the blood pressure of 4.85 mmHg. Adolescents with normal nutritional status can increase knowledge about hypertension, as well as regulate eating habits according to needs of physical growth and development.

Keywords: hypertension, adolescent, gender, BMI, sodium intake, physical activity.

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BACKGROUND

Adolescence is the second cycle in every individual's life. Growth and development during this period are characterized by physical, psychological and social changes (WHO, 2011b; Soekatri et al., 2011).

Physical changes are characterized by increasing muscle mass, fat tissue and hormonal changes (Andriani and Wirjatmadi, 2012). Psychologically, adolescents experience changes in emotions, thoughts, feelings, social environment, and the responsi-

bilities to face (Istiany and Rusilanti, 2013). Social growth and life patterns in society affect the disease type in adolescents (Soekatri et al., 2011).

WHO (2013) states that global cardiovascular disease accounts for 17 million deaths annually. A total of 9.4 million deaths were due to complications of hypertension and about 45% of deaths from heart disease and 51% of deaths from stroke. Basic Health Study Results of 2013 states that the prevalence of hypertension in the

population aged more than 18 years was 5.9% (from 31.7% in 2007 to 25.8% in 2013). The highest hypertension prevalence in Indonesia is Bangka Belitung Province, which is 30.9% (Indonesia Republic Health Ministry, 2013). The measurement results in seven districts/ municipalities of Bangka Belitung Province at the age more than 15 years state Pangkalpinang has hypertension prevalence 28.5% (Ministry of Health RI, 2013c). The result of blood pressure measurement at high school children in three high schools in Pangkalpinang by health office of Pangkalpinang City in October 2015 resulted 10.28% (Pangkalpinang Health Office, 2015).

Many factors related to adolescent's hypertension. Unhealthy lifestyles include alcohol consumption, smoking, excessive sodium consumption, high levels of stress and lack of physical activity can lead to cardiovascular diseases such as hypertension (Cordentemartinez et al., 2009; Roberta et al., 2015; NkehChungag et al., 2015). When blood pressure is high, it tends to be hypertensive in adulthood and may increase the risk of stroke, heart, kidney, and become a higher risk of morbidity and mortality (Lurbe et al., 2009, Saing, 2005).

Men have a risk of about 2.3 times more increased systolic blood pressure compared with women (Ministry of Health, 2013a). Studies in Hungary and the Middle East show that both systolic and diastolic blood pressure in men is significantly higher than women (Katona et al., 2011).

Family history of hypertension has a 3-4 times chance of developing hypertension at an early age compared with those without a family history (Hartono, 2013). If both parents suffer from essential hypertension then 44.8% of children will suffer from hypertension, and if one parent is hypertension then 12.8% of the offspring

will have hypertension (Saing, 2005). Study conducted in adolescents aged 14-17 years in African Americans states that family history with high blood pressure increases the chances of hypertension in adolescents as much as 65% (Covelli, 2007).

Nutritional status plays an important role of hypertension in adolescents can be seen from the body mass index by age (BMI/Age). Study in Lisbon, Portugal, states that the Body Mass Index (BMI) in adolescents with hypertension was found to a result in normal weight 30.4%, obesity 45.2% and obesity 45.5% (Silva et al., 2012). Consuming high sodium foods will also affect blood pressure (Yang et al., 2012). Study conducted by He (2008) in children age 4-18 years stated that there is a significant relationship between salt intake with systolic blood pressure and diastolic.

Increasing physical activity is one way to reduce the risk of hypertension because with physical activity, the energy released will be more so that the energy balance can be achieved and can control the weight (WHO, 2011a). Americans recommend that adolescents begin to increase their physical activity for 1500 minutes/ week or about 3.5 hours/ day, that physical activity at least 15 minutes a day is estimated to decrease 14% of hypertension risk that can cause death (Lauer, 2012).

The study purpose was to determine the factors associated with hypertension incidence in adolescents aged 15-17 years in Pangkalpinang, Bangka Belitung Islands.

SUBJECT AND METHOD

This was an analytic observational with cross sectional design. Study location was in Pangkalpinang, Bangka Belitung in X-XI students in 4 high schools aged 15-17 years. Study subjects were 120 people suffered from adequate nutritional status and more

nutritional status as a case. The sampling technique used a fixed-exposure sampling system. Measurement of nutritional status used anthropometry. Physical activity was measured by an International Physical Activity Questionnaire. Sodium intake was measured by food frequency questionnaire. Blood pressure measurement used Sphygmomanometer. Data analysis used multiple linear regressions. This study had received

approval from the medical study ethic commission Dr. RSUD. Moewardi, Medicine Faculty, SebelasMaretUniversity with number: 233 / III / HREC / 2016.

RESULT

Table 1 showed the frequency distribution Characteristics of the study subjects. The average age of the study subjects was 16 years female of 32.50%.

Table 1. Study subject characteristic frequency distribution

Variable	Gender			
	Men		Women	
	n	%	n	%
Age				
15 years	11	9.17	25	20.83
16 years	35	29.17	39	32.50
17 years	3	2.50	7	5.80
IMT				
Normal	33	27.50	57	47.50
fat	16	13.33	14	11.67
Hypertension Status				
Yes	17	14.17	10	8.33
No	32	26.67	61	50.83
Family illness history				
Hypertension	30	25.00	33	27.50
Stroke	7	5.83	8	6.67
Nothing	12	10.00	30	25.00
Sodium intake				
Sufficient	33	27.50	55	45.83
More	16	13.33	16	13.33
Physical activity				
Medium	34	28.33	53	44.17
High	15	12.50	18	15.00

BMI mean had a normal nutritional status of 47.50%, and no hypertension of 50.83% with female gender. The mean subjects with family history of hypertension were 27.50% and 6.67% of stroke in female gender, and

average sodium intake were 45.83% with adequate sodium intake and physical activity was obtained 44.17% had physical activity with female gender.

Table 2. Frequency distribution of BMI, sodium intake, and physical activity on hypertension

Variable	Category	Hypertension status			
		Normal		Hypertension	
		n	%	n	%
BMI	Normal	84	70.00	6	5.00
	Fat	9	7.50	21	17.50
Sodium intake	Sufficient	76	63.30	12	10.00
	High	17	14.20	15	12.50
Physical activity	Medium	69	57.50	18	15.00
	High	24	20.00	9	7.50

Table 2 showed the average study subjects had normal blood pressure with a normal BMI of 70.00%, had normal blood pressure

with adequate sodium intake of 63.30%, and had normal blood pressure with moderate physical activity of 57.50%.

Table 3. Multivariate analysis of hypertension risk factor in adolescent with gender variable, family history of hypertension and stroke, BMI/Age, Na intake, physical activity

Variable	b	95% CI		P
		Upper limit	Lower limit	
Family illness history (hypertension)	-1.73	-6.59	3.13	0.481
Gender (Men)	5.77	1.07	10.48	0.017
IMT (kg/m ²)	4.85	3.03	6.66	<0.001
Sodium intake(mg)	< 0.01	<0.01	< 0.01	0.076
Physical activity (METs)	-0.01	- 0.01	< 0.01	0.592
Constant	100.24	86.84	113.65	<0.001
N	120			
Adj R ²	35.60%			
p	<0.001			

DISCUSSION

The results showed a positive relationship between sex with hypertension risk, men had higher blood pressure than women. Studies in Hungary and Middle East suggest that both systolic and diastolic blood pressure in men is significantly higher than women (Katona et al., 2011).

Men have a risk of about 2.3 times more increased systolic blood pressure compared with women (Indonesia Republic Health Ministry, 2013a). Male adult systolic blood pressure greater than 4 mmHg at age 13-15 years compared to women and age 16-18 years has a blood pressure difference reaching 10 - 14 mmHg higher in men than women (Maranon and Reckelhoff, 2013). One of the differences is caused by hormonal factors such as androgen hormone and testosterone, thought to play a role in blood pressure regulation related to the differences between the genders (Indonesia Republic Health Ministry, 2013a, Maranon and Reckelhoff, 2013).

Androgen hormones are thought to be strong as a mediator of hypertension and cardiovascular disease. Another fact of ET-1

produced by vascular endothelial cells is less in women than men. The presence of androgens in men is known to stimulate ET-1 production. Androgen is thought to play a role in hypertension. In this case, hypertension may be due to the effect of renal reninangiotensin. Testosterone is also known to activate the reninangiotensin system affecting elevated blood pressure (Adhita and Pramuningtyas, 2010).

There is a negative relationship between family history of disease and hypertension risk. This may happen because subject age is still adolescent, the significant risk of hypertension increases by age (Rahajeng and Tuminah, 2009). Study in Europe shows the hypertension prevalence in children and adolescents by 1% to 4% (Kollias, 2011). According to Henuhili et al. (2011) studystates that hypertension genes are dominant, every individual of hypertension is in every generation and offspring who do not inherit hypertension will have hypertensive offspring as well, hypertensive inheritance is not X-linked, which is genes on chromosomes sex, because neither the

father nor the mother can inherit the offspring of both men and women.

According to Mendel's law, if only one of parents suffered from hypertension, then the children may not to suffer from hypertension be 50% (Kalangi et al, 2015). Several studies have suggested that genes can affect blood pressure, among other genes grouped into: genes that encode the renin angiotensin system (Poilmorfisem I / D gen Angiotensi converting enzyme), genes that play a role in renal sodium homeostasis and the genes that regulate steroid metabolism (Zarouk et al., 2012; Tabatabaei et al., 2006; Ehret and Caulfield 2013). Genes that play a role in renal sodium homeostasis are lysine deficient protein kinase 1, amilorid sensitive sodium channel, beta and gamma subunit genes encoding 2 EnaC channel sodium subunits (Toker et al., 2015). The genes affect the Na⁺ -K⁺ pump in the renal tubules thus increasing the sodium and water retention of the kidneys. Increasing sodium reabsorption in the kidney increases plasma volume and extracellular fluid, as well as increasing extracellular volume and causes increasing venous return blood flow to the heart. There was an increase in cardiac output and subsequently increasing arterial pressure (Zarouk et al., 2012; Sayed et al., 2006; Ehret and Caulfield, 2013).

The genes that have an effect on steroid metabolism are CYP11B2 (aldosterone synthase gene) and NR3C2 (corticoid mineral receptor gene). These genes increase aldosterone production so it will increase sodium retention in kidney. Cardiac output increases and further increases arterial pressure (Zarouk et al., 2012; Sayed et al., 2006; Ehret and Caulfield, 2013). The insertion/ deletion polymorphism of angiotensin-converting enzyme (ACE) is characterized by the appearance or disappearance of the 28bp repeat sequence on

intron 16 and is a gene also thought to play a strong role in the mechanism of hypertension. Genepolymorphism produces 3 genotypes: Homozygous II, heterozygote ID, and homozygous DD. Studies suggest that homozygous individuals with D alleles have higher ACE concentrations than individual heterozygous ID or homozygous II (Kuschnir and Mendonça, 2007). By increasing levels of ACE in the blood and tissues, the levels of Ang II (angiotensin II) also increase. The two main effects of Angiotensin II in increasing arterial pressure are vasoconstriction in various parts of body and decreased excretion of salt and kidney by water. By the appearance of vasoconstriction in various places, there is an increase in perifer total resistance which further increases arterial pressure. Angiotensin II also plays a role in reabsorption of sodium and water from urine. Hypertension mechanism is similar to hypertension mechanism by genes that play a role in renal sodium homeostasis (Zarouk et al., 2012; Sayed et al., 2006; Ehret and Caulfield, 2013). Further study on family history and age of hypertension is needed.

There was no positive relationship between sodium intake and hypertensive risk occurrence. The relationship between sodium intake and hypertension risk may happen because not everyone has an individual's genetic sensitivity affected sodium intake, other possibility happens because individual reactions to the amount of sodium in the body differ depending on individual sensitivity (Kotchen et al., 2006). Long-lasting habits are consumed by high-sodium foods (use of food seasonings, processed foods and preserved foods such as shrimp paste, salted fish, and crackers) (Mahan et al., 2012). The appearance of other nutrients such as potassium intake also affects the sodium response to blood pressure. Potassium is a major cau-

tion in intracellular fluid that has the same function as sodium. High potassium intake can minimize the increase in blood pressure by the presence of excess sodium (Appel, 2011).

There is a study states that the ratio of sodium and potassium in urine is stronger to describe the relationship between blood pressure (Mahan et al., 2012). This study is in line with study conducted by Kautsar et al (2014) states there is no relationship between sodium intake and hypertension risk. While study conducted by Fatta and Sulchan (2012), states there is a significant relationship between sodium intake with hypertension risk, high sodium intake at risk 4536 times to be hypertension. Study He et al., (2008) states that there is a relationship between sodium intake and blood pressure, an increase of 1 g/ day in natrium intake can raise 0.4 mmHg at systolic pressure and 0.6 mmHg diastolic pressures. High sodium intake causes adipocyte cell hypertrophy resulting from lipogenic processes in white fat tissue, if persistent cause constriction of blood vessel channels by fat and result in increasing blood pressure (Fonseca-Alaniz et al, 2008).

High sodium intake can lead to Na ions in food ingredients absorbed in blood vessels, Na ions in the blood will cause the body retain more water to maintain the electrolyte so that the fluid accumulation in body because of sodium binding liquid outside the cells that cannot be removed (Adroque and Madias, 2008; Indonesia Republic Health Ministry, 2013a).

Excessive consumption of sodium over long periods has great potential to increase hypertension (Covelli, 2007). More study is needed connecting sodium intake with potassium.

There was no positive relationship between physical activity and hypertension

risk. This may happen because only a small percentage of students do regular exercise and most of the time is used for routine activities such as housework, studying and watching TV. Sports done by teenagers may still not fully exercise with a good mechanism, meaning that when they do sports, type, time, intensity, and frequency is less precise or too long so it is not as health standards.

According to Sutangi and Winantri (2011), that sports that do not conform to health standards will not provide health effects, isotonic exercise that utilizes foot movements such as roads is better than isometric exercise that utilizes hand gestures such as lifting weights, because the isotonic exercise effects can improve cardiac breathing resistance or suppresses narrowing of the blood vessels while isometric exercise is less favorable in the heart's respiratory system or can increase blood pressure.

In Prasetyo et al. (2015) study, there was no relationship between physical activity and hypertension. Study conducted Sulastrri and Sidhi, (2011) also states that there was no relationship between physical activity with the incidence of hypertension in adolescents. Activities such as exercise can reduce peripheral pressure that will lower blood pressure (hypertension) and can train the heart muscle so it becomes usual when the heart must do a more severe work under certain conditions (Tsioufis et al., 2010).

There is a positive relationship between BMI and the risk of hypertension. Study Fitriana et al., (2013) found that obesity affects the hypertension incidence. Study conducted by Kautsar et al. (2014) shows there is a significant relationship between obesity to the hypertension incidence. Study in Mexico shows that there is a relationship between hypertension with

more nutritional status in adolescents (Flores-Huerta et al., 2009). Overweight increases the incidence of hypertension risk five times higher than normal weight Kautsar et al. (2014). A normal weight gain of 10% results in a 7 mm Hg blood rise (Indonesia Republic Health Ministry, 2013a). According to (Sanchez-Zamoran et al., 2009; Rusilanti and Istiany, 2013), the hypertension risk is 3.6 times greater in adolescents with overweight and 14 times greater than obese teenagers. Obesity will activate cardiac work and may cause long-term cardiac hypertrophy, cardiac output, stroke volume, blood volume and blood pressure will tend to rise otherwise endocrine function is also impaired, pancreatic beta cells will dilate, increased plasma insulin and tolerance glucose also increased so as to facilitate the occurrence of hypertensive disease (Kautsar et al, 2014).

Study results can be concluded that there is a positive relationship between sex and BMI with hypertension risk factors in adolescents aged 15-17 years. Young men have a higher blood pressure level than women of 5.77 mmHg. Any increase of equivalent of 1 kg/m² IMT will increase blood pressure by 4.85 mmHg. Teenagers with more nutritional status can increase knowledge about hypertension, and regulate eating habits according to growth and development needs.

REFERENCES

- Adhita PM, Pramuningtyas R (2010). Perbedaan Angka Kejadian Hipertensi antara Pria dan Wanita Penderita Diabetes Mellitus Berusia ≥ 45 Tahun. *Biomedika*, 2(2): 67–71. Diakses pada tanggal 9 Juni 2016.
- Adroque HJ, Madias NE (2008). Sodium and Potassium in the Pathogenesis of Hypertension. *The New England Journal of Medicine*. 356(19): 1966–1978.
- Andriani M, Wirjatmadi B (2012). Peran Gizi dalam Siklus Kehidupan. Edisi Pertama, Jakarta: Kencana Prenada Media Group.
- Appel LJ (2011). Diet and Blood Pressure Nutrition Diet and Hypertension In: Rous AC, Caballero B, Cousins RJ, Tucker KL, Ziegler TR. *Modern Nutrition And Health Disease*. 11th ed. Philadelphia; Wolters Kluwer. 875-886.
- Cordente MCA, Garcia SP, Sillero QM, Stirling JR (2009). Correlations between the Blood Pressure and Other Health Variables in Spanish Adolescents. *Journal Adolescent Medicine Health*. 21(4): 635–651.
- Covelli MM (2007). Prevalence of Behavioral and Physiological Risk Factors of Hypertension in (African) (American) Adolescents. *Journal Pediatric Nursing*. 33(4): 323–332.
- Dinkes Pangkalpinang (2015). Laporan Hasil pengukuran Tekanan Darah Pada Anak SMU 2015, Pangkalpinang.
- Ehret GB, Caulfield MJ (2013). Genes for blood pressure: An Opportunity to Understand Hypertension. *European Heart Journal*. 34(13): 951–961.
- Fatta LA, Sulchan M (2012). Asupan Tinggi Natrium dan Berat Badan Lahir sebagai Faktor Risiko Kejadian Hipertensi Obesitas pada Remaja Awal. *Journal of Nutrition College*. 1(1): 127–133.
- Fitriana R, Lipoeto NI, Triana V (2013). Faktor Risiko Kejadian Hipertensi pada Remaja di Wilayah Kerja Puskesmas Rawat Inap Sidomulyo Kota Pekanbaru. *Jurnal Kesehatan Masyarakat*. 7(1): 10–15. Diakses pada tanggal 15 Januari 2016.

- Flores HS, Klunder KM, Cruz LR, Santos JI (2009). Increase in Body Mass Index And Waist Circumference is Associated With High Blood Pressure in Children And Adolescents in Mexico City. *Archives of Medical Study*, 40(3): 208–215.
- Fonseca AMH, Takada J, Andreotti S, Campos TBF, Campana AB, Borge SCN, Lima FB (2008). High Sodium Intake Enhances Insulin-Stimulated Glucose Uptake in Rat Epididymal Adipose Tissue. *Journal of Obesity*. 16(6): 1186–92.
- Hartono A (2013). *Dasar-dasar Patofisiologi Penyakit*. Edisi Pertama. Tangerang Selatan: Binarupa Aksara.
- He FJ, Marrero NM, Macgregor GA (2008). Salt and Blood Pressure in Children and Adolescents. *Journal of Human Hypertension*. 22: 4–11.
- Henuhili V, Rahayu T, Nurkhasanah L (2011). Pola Pewarisan Penyakit Hipertensi dalam Keluarga sebagai Sumber Belajar Genetika. 242–247.
- Istiany A, Rusilanti (2013). *Gizi Terapan*. Edisi Pertama. Bandung: PT. Remaja Rosdakarya.
- Katona E, Zrinyi M, Komonyi E, Lengyel S, Paragh G, Zatik J, Fulesdi B, Pall D (2011). Factors Influencing Adolescent Blood Pressure: The Debrecen Hypertension Study. *Kidney and Blood Pressure Study*. 34(3): 188–195. Diakses pada tanggal 12 Desember 2015.
- Kalangi JA, Umboh A, Pateda V (2015). Hubungan Faktor Genetik dengan Tekanan Darah pada Remaja. *Jurnal e-Clinic*. 3: 3–7.
- Kautsar F, Syam A, Salam A (2014). Hubungan Obesitas, Asupan Natrium dan Kalium Dengan Tekanan Darah Pada Mahasiswa Universitas Hasanuddin. 1–9.
- Kementerian Kesehatan Republik Indonesia (2013a). *Pedoman Teknis Peneemuan dan Tatalaksanaan Hipertensi*. Jakarta: Kementerian Kesehatan RI.
- Kementerian Kesehatan Republik Indonesia (2013b). *Riset Kesehatan Dasar (RISKESDAS) 2013*.
- Kementerian Kesehatan Republik Indonesia (2013c). *Risikesdas dalam Angka 2013 Pokok-Pokok Hasil RISKESDAS dalam Angka Provinsi Kepulauan Bangka Belitung Tahun 2013*. Jakarta: Badan Penelitian dan Pengembangan Kesehatan. Diakses pada tanggal 20 November 2016.
- Kollias A (2011). Hypertension in Children and Adolescents. *Pediatric Clinics of North America*. 1(1): 15–19.
- Kotchen TA, Kotchen JM (2006). Nutrition, diet, and Hypertension, In: Shils ME, Shike M, Ross AC, Caballero B, Cousins RJ, Editors, *Modern Nutrition in Health and Disease*. 10th Edition, Philadelphia; Lippincott Williams and Wilkins. 1095-1107.
- Kuschnir MCC, Mendonca GAS (2007). Risk Factors Associated with Arterial Hypertension in Adolescents. *Journal de Pediatria*. 83(4): 335–342.
- Lauer MS (2012). And What About Exercise? Fitness and Risk of Death In “Low-Risk” Adults. *American Heart Association*. 4: 1–4.
- Lurbe E, Cifkova R, Cruickshank JK, Dillon MJ, Ferreira I, Invitti C, Kuznetsova T, Laurent S, Mancina G, Morales OF, Stergiou G, Wuhl E, Zanchetti A (2009). Management of High Blood Pressure in Children and Adolescents: Recommendations of The European Society of Hypertension. *Journal of Hypertension*. 27(9) : 1719–1742.
- Mahan LK, Escott SS, Raymond JL (2012). *Krause's Food and The Nutrition Care*

- Process, 13th edition, Philadelphia. 900-918.
- Maranon R, Reckelhoff JF (2013). Sex and Gender Differences in Control of Blood Pressure. *Clinical science National Institutes of Health*, 125(7): 311-318.
- Nkeh CBN, Sekokotla AM, Sewani RC, Namugowa A, Iputo JE (2015). Prevalence of Hypertension and Prehypertension in 13-17 Year Old Adolescents Living in Mthatha - South Africa: a Cross-Sectional Study. *Central European Journal of Public Health*. 23(1): 59-64.
- Prasetyo DA, Wijayanti AC, Werdani EK (2015). Faktor-Faktor yang Berhubungan dengan Kejadian Hipertensi pada Usia Dewasa Muda di Wilayah Puskesmas Sibela Surakarta.(1).
- Rahajeng E, Tuminah S (2009). Prevalensi Hipertensi dan Determinannya di Indonesia. *Maj Kedokteran Indonesia*. 59(12): 580-587.
- Roberta G, Holanda I, Vieira EES, Nunes RB, Vilarouca AR(2015). Prevalence of Arterial Hypertension and Risk Factors in Adolescents. *Acta Paul Enferm*. 28(1): 81-87.
- Rusilanti, Istiany A (2013). *Gizi Terapan*. Edisi Pertama. Bandung: PT. Remaja Rosdakarya.
- Saing JH (2005). Hipertensi pada Remaja. *Sari Pediatri*. 6(4): 159-165.
- Sanchez ZLM, Salazar ME, Anaya-Ocampo R, Lazcano PE (2009). Body Mass Index Associated with Elevated Blood Pressure in Mexican School-Aged Adolescents. *Preventive Medicine*. 48(6): 543-548.
- Sayed TFA, Oostra BA, Isaacs A, Van DCM, Witteman JCM (2006). ACE Polymorphisms. *Circulation Study*. 98(9): 1123-1133.
- Silva D, Matos A, Magalhaes T, Martins V, Ricardo LA (2012). Prevalence of Hypertension in Portuguese Adolescents in Lisbon, Portugal. *Revista Portuguesa de Cardiologia (English Edition)*. 31(12): 789-794.
- Soekatri M, Almatsier S, Soetardjo S(2011). *Gizi Seimbang dalam Daur Kehidupan*. Jakarta: Gramedia Pustaka Utama.
- Sulastri D, Sidhi(2011). Faktor Risiko Hipertensi pada Siswa SMU Adabiah di Kota Padang. *Majalah Kedokteran Andalas*. 35(2): 149-158.
- Sutangi H, Winantri (2011). Faktor yang Berhubungan dengan Kejadian Hipertensi pada Wanita Lansia di POSBINDU Desa Sukaurip Kecamatan Balongan Indramayu. *e-jurnal Kesehatan Masyarakat Universitas Wiralodra Indramayu*, pp.1-8. Available at: ISSN 1693-7945.
- Toker RT, Yildirim A, Demir T, Ucar B, Kilic Z(2015). Circadian Blood Pressure Rhythm in Normotensive Offspring of Hypertensive parents. *Cardiology Journal*, 22(2): 172-178.
- Tsioufis C, Kyvelou S, Tsiachris D, Tolis P, Hararis G, Koufakis N, Psaltopoulou T, Panagiotakos D, Kokkinos P, Stefanadis C(2010). Relation between Physical Activity and Blood Pressure Levels in Young Greek Adolescents : The Leontio Lyceum Study. 21(1): 63-68.
- WHO (2013). *A Global Brief on Hypertension Silent Killer*, Global Public Health Crisis World Health Day 2013, 1211. Geneva, Switzerland.
- WHO (2011). *Strengthening the Health Sector Response to Adolescent Health and Development*. 805-813. Diakses pada tanggal 12 November 2015.
- Yang Q, Zhang Z, Kuklina EV, Fang J, Ayala C, Hong Y, Loustalot F, Dai S, Gunn JP, Tian N, Cogswell ME, and Merritt

R (2012). Sodium Intake and Blood Pressure Among US Children and Adolescents. *Pediatrics*. 130(4): 611–619.

Zarouk WA, Hussein IR, Esmail NN, Raslan HM, Reheim HAA, Moguib O,

Emara NA (2012). Association of Angiotensin Converting Enzyme Gene (I/D) Polymorphism with Hypertension and Type 2 Diabetes. *Bratisl Lek Listy*, 113(1): 14–18.