World Journal of Public Health
2018; 3(4): 111-117
http://www.sciencepublishinggroup.com/j/wjph
doi: 10.11648/j.wjph.20180304.12
ISSN: 2637-6040 (Print); ISSN: 2637-6059 (Online)

Family History and BMI Correlate with Blood Pressure: A Cross-Section Study

An-le Li*, Yue-qin Shao, Hong Yuan, Yi-ying Zhang

Jiading District Center for Disease Control and Prevention, Shanghai, China

Email address:
anle_li@aliyun.com (An-le Li), yueqshao@sohu.com (Yue-qin Shao), hongyuan@sohu.com (Hong Yuan), zyyzyyjd@163.com (Yi-ying Zhang)

*Corresponding author

To cite this article:
An-le Li, Yue-qin Shao, Hong Yuan, Yi-ying Zhang. Family History and BMI Correlate with Blood Pressure: A Cross-Section Study. *World Journal of Public Health.* Vol. 3, No. 4, 2018, pp. 111-117. doi: 10.11648/j.wjph.20180304.12

Received: August 21, 2018; **Accepted**: October 9, 2018; **Published**: November 7, 2018

Abstract: Hypertension is a general disease which is caused by complex polygenic inheritance, environment and multiple risk factors. The specific proportion of genes and the environment, however, was different in the previous published studies. The objective of this study is to identify the correlation between family history of hypertension (FH) and body mass index (BMI) with blood pressure based on cross section data. The correlation and interaction of FH and BMI on blood pressure is measured of 96646 patients with complete data which is selected from the database of screening in hypertension registry and follow-up management system by statistical analysis. 46.52% of patients with high blood pressure had FH, and 43.52% of patients with high blood pressure are overweight or obese. The average onset age of hypertension in cases without hypertension family history is 65.11 years old, and the average onset age in cases with hypertension family history is 60.17 years old. The average onset age of hypertension in cases in normal weight subjects is 64.01 years old, while the average age in overweight and obese subjects are respectively 61.55 and 61.11 years old. The independent effect of FH and BMI on SBP are OR=1.01 (95%CI: 0.98～1.06) and OR=1.26 (95%CI: 1.04～1.54) respectively, and the joint effect is OR=1.43 (95%CI: 1.11～1.83); the independent effect on DBP are OR=1.75 (95%CI: 1.66～1.85) and OR=1.99 (95%CI: 1.89～2.09) respectively, and the joint effect is OR=2.37 (95%CI: 2.25～2.50). The pure attributable proportion of interaction of FH and BMI on SBP is 37.21%, and that on DBP is 27.01%. FH and BMI have positive interaction with blood pressure. FH and BMI have positive correlation with blood pressure. Both of them (FH and BMI) can simultaneously increase the risk of hypertension, and lead to earlier age of onset.

Keywords: Family History, Body Mass Index (BMI), Correlation, Interaction, Blood Pressure

1. Introduction

Hypertension is characterized by chronically elevated blood pressure and affects 20% to 30% of the population worldwide. It is one of the factors that increases the morbidity and mortality of cardiovascular diseases worldwide [1-3]. Lawes had found that 13.5% premature deaths, 54% strokes and 47% ischemic heart disease worldwide are caused by hypertension [4]. Hypertension is a multifactorial disease caused by genetic and environmental factors, The interaction of genes and genes, genes and environment lead to the risk of different populations and individuals suffering from the disease。Unhealthy lifestyle such as obesity and lack of exercise can significantly raise hypertension [5, 6]. The phenomenon of familial aggregation of hypertension showed that in the general population between parents and children, the same prevalence rate of brothers and sisters is as high as 20% to 66%; a plurality of twin studies have estimated the possibility of hereditary blood pressure over 50%, this phenomenon shows that more than half of the blood pressure change can be attributed to the accumulation of genetic effects [7]. It is assumed that blood pressure is controlled by a large number of genes, and each gene has only a relatively weak effect on blood pressure. Therefore, it is difficult to detect genetic variants that affect blood pressure by traditional methods such as candidate gene screening and gene linkage studies, family history is an important marker of genetic factors, it is often used as an alternative indicator to study the relationship between genetic factors and diseases [8-12].

Body mass index (BMI) is a comprehensive indicator of the outcome of acquired lifestyle, and closely related to the occurrence of hypertension [13-17]. In this study, the residents in Jiading district in Shanghai are studied as the research objects, using large data to analyze the significance of correlation and interaction between hypertension family history and body mass index in the pathogenesis of blood pressure, in order to make a preliminary study of the pathogenesis of blood pressure and providing a reference for future prevention and treatment.

2. Methods

2.1. Study Population

This cross-sectional study was conducted in Jiading district in Shanghai, China. 96646 cases in this study were selected from the database of hypertension registry and follow-up management system in Jiading district in Shanghai. This system is a government project for health of the whole residents, invested by the Shanghai municipal government from 2006 to now, and spent every year for a lot of money; the system is based on the household health records of residents in community, and provides a series of medical services for hypertension, including screening, diagnosis, case report, registration, treatment, regular follow-up and medical evaluation. As the population of this study, they have been selectively collected their family history, blood pressure, height and weight data from the screening database in system.

2.2. Measurement

The physical examination evaluated blood pressure (BP), body weight and height. BP was measured at the right arm supported at the heart level after participants resting for 5 min, using the Omron "Intellisense" HEM-1000 Electronic Blood Pressure Monitor. BP was measured three times, with 30 s between each measurement. The average of three readings was used for analysis [18-19]. Body weight and height was measured without shoes using a standard right-angle device and a fixed measurement tape (to the nearest 0.5 cm).

2.3. Ethics

Ethical approval was granted by Jiading district center for disease control and prevention research ethics committee. All subjects gave verbal and written informed consent to participate in the study, they would like to participate in investigation and answer all the related questions in the questionnaire. For this study did not need to collect biological samples, no injury, only used questionnaires to investigate patients' information, the ethics committee approved this method of consent. The survey was conducted by a full-time investigator using a questionnaire and face-to-face inquiries.

2.4. Definitions

A family history of hypertension was defined as at least one of first-degree relatives (parents and siblings) who have diagnosed or suffered from hypertension. Blood pressure classified as: the Systolic blood pressure (SBP) ≥140 mmHg or and diastolic blood pressure (DBP) ≥90 mmHg was defined as high blood pressure [19]. Body mass index (BMI) = body weight (kg) / height squared (m^2). Body mass index classification standard: BMI< 18.5 is low weight; BMI between 18.5~23.9 is normal weight; BMI between 24~27.9 is overweight; BMI over 28 is obesity [20-22].

2.5. Statistical Processing

All data selected from the database of screening in hypertension registry and follow-up management system was exported to Excel, established initial database, and then checked and processed data. The statistical software (SPSS 16.0 for windows) was used to process the data. The comparison of qualitative data between groups was made by chi square (x^2) test; the comparison of quantitative data between groups was performed by means of variance analysis or t test. Using multivariate Logistic regression model to control the confounding factors, the risk factors of hypertension were analyzed, and the interaction between family history of hypertension and body mass index was analyzed by additive model. When the P value was less than 0.05, the difference was statistically significant. The additive model was used to calculate the additive interaction effect by cross analysis, and the synergistic effect index (S), relative excess risk due to interaction (RERI), attributable proportion due to interaction (AP) and the percentage of the interaction between the pure factor (PAP) calculation formula are as follows:

$$S(AB)=[R(AB)-R(A0B0)] / [R(AB0)- R(A0B0)+ R(A0B)- R(A0B0)]$$

$$RERI(AB)=[R(AB)-R(AB0)-R(A0B)+ R(A0B0]/ R(A0B0$$

$$AP(AB)=[R(AB)-R(AB0)-R(A0B)+ R(A0B0)] / R(AB)$$

$$PAP(AB)=[R(AB)-R(AB0)-R(A0B)+R(A0B0)]/[R(AB)- R(A0B0)]$$

(Note: R(AB) is the risk ratio of A and B factor exposed; R(A0B0) is the risk ratio of A and B factor unexposed; R(AB0) is the risk ratio of A factor exposed but B factor unexposed, R(A0B) is the risk ratio of A factor unexposed and B factor exposed)

3. Results

3.1. Characteristics of Participants

Among 96646 cases in this study, there are 47596 male (49.25%), 49050 female (50.75%); 422 are under 30 years old

(0.44%), 2889 between 30 to 39 years old (2.99%), 11090 between 40 to 49 years old (11.47%), 27474 between 50 to 59 years old (28.43%), 30002 between 60 to 69 years old (31.04%), 24769 aged 70 and above (25.63%). Among these subjects, There are 1936 low weight (2%); 43232 standard weight (44.73%); 40992 overweight (42.41%); and 10486 obesity (10.85%). 46.52% of subjects with high blood pressure have a family history. 46.42% of subjects with high systolic blood pressure had a family history; 47.86% of subjects with high diastolic blood pressure have a family history. Among those cases with high blood pressure, 1.54% cases are low weight; 34.41% are overweight; 9.11% are obese, and 35.23% are normal weight. The other characteristics of study population see table 1.

Table 1. *Characteristics of study population.*

N	male				female			
	mean	SD	Min	Max	mean	SD	Min	Max
	47596 (49.25%)				49050 (50.75%)			
age(y)	61.95	12.18	28.00	95.00	63.66	12.05	28.00	97.00
SBP(mmHg)	153.47	13.31	100.00	220.00	153.60	13.18	100.00	200.00
DBP(mmHg)	93.31	8.52	60.00	140.00	92.44	8.20	60.00	120.00
Height(cm)	167.16	6.69	147.00	192.00	159.74	6.35	147.00	176.00
weight(Kg)	68.56	11.00	48.00	100.00	61.95	11.07	40.00	96.00
BMI	24.44	2.87	16.17	39.96	24.17	3.09	14.17	39.06

3.2. Association of FH and BMI on BP

Among 96646 cases in this study, the average onset age of cases with family history of hypertension is 60.17±10.52 years old; and the average onset age of cases without family history of hypertension is 65.11±12.98 years old. The difference about two HP groups shows statistically significant (t=64.471, P<0.001). The results suggest that the onset age of hypertension in cases with a hypertension family history is 5 years earlier likely than that of cases without family history. The average onset age of BMI low weight cases is 71.35±12.57 years old; the average onset age of BMI normal cases is 64.01±12.59 years old; the average onset age of overweight cases is 61.55±11.47 years old; and the average onset age of obesity cases is 61.11±11.66 years old. The difference among four groups shows statistically significant (t=31.386, P<0.001). The results remind us, compared with normal weight people, the average onset age of BMI thin cases is 6-7 years likely late, but the average onset age of BMI overweight and obese cases is 3 years earlier likely. The results are shown in table2.

Table 2. *The average onset age of hypertension in family history and BMI groups.*

		N	On set age(y)		t	
			mean	SD		
Family history	No	51690	65.11	12.98	64.471	<0.001
	yes	44956	60.17	10.52		
BMI	Low weight	1936	71.35	12.57	31.386	<0.001
	normal	43232	64.01	12.59		
	overweight	40992	61.55	11.47		
	obese	10486	61.11	11.66		
	total	96646	62.80	12.15		

Among these cases, 44956 (46.52%) have family history of hypertension, the statistical result shows that the difference between family history (FH) and blood pressure is significant, FH and DBP is (x2=11.204, p=0.001); FH and DBP is (x2=357.811, p=0.000)). The difference between body mass index (BMI) and blood pressure is significant, BMI and SBP is (x2=10.608, p=0.001); BMI and DBP is (x2=218.453, p<0.001). The results are shown in table 3.

Table 3. *The association of family history of hypertension and body mass index on systolic and diastolic blood pressure of study population.*

		SBP		DBP		total
		<140mmHg	≥140mmHg	<90mmHg	≥90mmHg	
Hypertension family history (FH)	no	1578(3.05%)	50112(96.95%)	9597(18.57%)	42093(81.43%)	51690(53.48%)
	yes	1544(3.43%)	43412(96.57%)	6313(14.04%)	38643(85.96%)	44956(46.52%)
	total	3122(3.23%)	93524(96.77%)	15910(16.46%)	80736(83.54%)	96646(100%)
		(x^2=11.204; p=0.001)		(x^2=357.811; p<0.001)		
Body mass index (BMI)	thin	52(2.69%)	1884(97.31%)	400(20.66%)	1536(79.34%)	1936(2.00%)
	normal	1532(3.54%)	41700(96.46%)	7790(18.02%)	35442(81.98%)	43232(44.73%)
	overweight	1494(3.64%)	39498(96.36%)	6373(15.55%)	34619(84.45%)	40992(42.41%)
	obese	351(3.35%)	10135(96.65%)	1360(12.97%)	9126(87.03%)	10486(10.85%)
	total	3429(3.35%)	93217(96.45%)	15923(16.48%)	80723(83.52%)	96646(100%)
		(x^2=10.608; p=0.001)		(x^2=218.453; p<0.001)		

3.3. Effect of FH and BMI on BP

Linear regression used to analyze the correlation FH and BMI to blood pressure, the results shows that the unstandardized coefficients of BMI and SBP is 0.150±0.014, (t=10.463, p<0.001); unstandardized coefficients of FH and SBP is 0.756±0.086, (t=8.806, p<0.001). The unstandardized coefficients of BMI and DBP is 0.242±0.009, (t=26.823, p<0.001); unstandardized coefficients of FH and DBP is 1.350±0.054, (t=25.028, p<0.001).

After confounding factors such as age and gender were controlled by multivariate logistic regression, main effect test results showed that the independent effect of the FH and BMI on SBP and DBP are statistically significant. See Table 4.

Table 4. *The test of independent effect.*

	SBP			DBP		
	Mean Square	**F**	**Sig.**	**Mean Square**	**F**	**Sig.**
Correction model	78021.470	430.020	<0.001	16369.804	225.946	<0.001
intercept	5661338.578	31227.556	<0.001	26183083.383	36139.610	<0.001
BMI	21111.922	116.360	<0.001	9966.447	137.563	<0.001
FH	25330.034	139.608	<0.001	11659.625	160.933	<0.001
BMI*FH	356.056	19.620	0.011	659.687	90.105	<0.001
Error	81.437			72.450		

The independent effect of FH on SBP is OR=1.01 (95%CI:0.98～1.06), and the individual effect on DBP is OR=1.75 (95%CI:1.66～1.85). The OR value of FH on DBP is slightly greater relatively than that on SBP. The independent effect of BMI on SBP is OR=1.26 (95%CI:1.04～1.54), and the individual effect on DBP is OR=1.99 (95%CI:1.89～2.09). The OR value of BMI on DBP was slightly greater relatively than that on SBP. The combined effect of FH and BMI on SBP was OR=1.43(95%CI:1.11～1.83), and the combined effect on DBP was OR=2.37(95%CI:2.25～2.50). The OR value of FH and BMI combined effect on DBP is slightly greater relatively than that on SBP. See table 5. Its shown that FH and BMI positively interact with SBP (SBP [OR (AB) >OR (A) + OR (B) -1], but DBP [OR (AB)<OR (A) + OR (B) -1]).

Table 5. *Analysis of the interaction between family history of hypertension and body mass index to study population.*

Family history	**BMI**	**SBP**		**0R(95%CI)**	**DBP**		**0R(95%CI)**
		-	+		-	+	
-	-	883	25427	1.00	5201	20226	1.00
-	+	853	24385	1.26(1.04～1.54)	2791	21594	1.99(1.89～2.09)
+	-	678	17315	1.01(0.98～1.07)	2216	15099	1.75(1.66～1.85)
+	+	1014	26090	1.43(1.11～1.83)	2552	23538	2.37(2.25～2.50)

(SBP: "-" <140mmHg, "+" ≥140mmHg; DBP: "-" <90mmHg, "+" ≥90mmHg; family history: "-" have not, "+" have; BMI: "-" normal weight, "+" abnormal weight (include thin, overweight and obese))

The analysis results from the quantitative interaction showed that, the synergistic effect index (S) of FH and BMI on SBO is 1.59 (95%CI:0.17～2.37); the relative excess risk due to interaction (RERI) of FH and BMI on SBP was 0.16 (95%CI:-0.52 ～ 0.76); The attributable proportion of interaction (AP) of FH and BMI on SBP is 11.19%; and the percentage of the interaction between the pure factor (PAP) is 37.21%. the synergistic effect index(S) of FH and BMI on DBP is 0.78 (95%CI:0.64～0.97); the relative excess risk due to interaction (RERI) of FH and BMI on DBP is -0.37 (95%CI:-0.69 ～ -0.05); The attributable proportion of interaction (AP%) of FH and BMI on DBP is 15.61%, and the percentage of the interaction between the pure factor (PAP) is 27.01%.

4. Discussion

Some studies have shown that hypertension has obvious familial clustering and the family history of hypertension has a heritability of 60%, more than a half of the objects in these studies have family history of hypertension. Compared with patients without family history of hypertension, patients with a family history of hypertension have a lower onset age and higher blood pressure levels, and it indicating that genetic factors can lead to elevated blood pressure levels and advanced onset age [22-23]. This study shows that the average onset age of cases without family history of hypertension is 65.11 years old, the average onset age of cases with family history of hypertension is 60.17 years old, the average onset age of hypertension in cases with family history is likely 5 years earlier than that of cases without family history. Therefore, these results are consistent with those reported in the literature, it was further proved that family history of hypertension is a risk factor for hypertension, family history of hypertension causes the onset age to advance, it's suggested that attention should be paid to the positive family history of hypertension. Due to family history of hypertension may not represent completely the shared genes and there is also the influence of the acquired environment, the influence of family history can explain the interaction between genes and the environment. Our next study will explore the independent role of genes in hypertension.

With the development of national economy, obesity has become one of the most important public health problems in

our country (China). Due to the small relatively size of human bodies in Asia, fat usually tends to accumulate in the abdomen, forming central obesity, and central obesity can lead to chronic non-communicable diseases [24-28]. This study showed that only 35.23% of cases are normal weight, while 43.52% of cases are overweight or obese, and 1.54% of cases are low weight. The average onset age of hypertension in low weight cases is 71.35 years old; the average onset age of hypertension in normal weight cases is 64.01 years old; the average onset age of hypertension in overweight and obese cases are respectively 61.55 and 61.11 years old. This research results remind us that compared with normal weight cases, the average onset age of BMI overweight and obese cases is 3 years earlier likely.

There are two kinds of models for analyzing biological interactions: addition and multiplication [29]. In our opinions, the interaction with additive models is more important for public health, we therefore used the additive model to analyze the interaction between family history of hypertension and body mass index in the pathogenesis of hypertension. The analysis results from the quantitative interaction showed that the combined effect of family history of hypertension and body mass index on systolic blood pressure was OR=1.43, and that on diastolic blood pressure was OR=2.37; the percentage of the interaction between the pure factor of hypertension family history and body mass index on systolic blood pressure was 37.21, and that on diastolic blood pressure was 27.01%. Family history of hypertension and body mass index positively interact with blood pressure, when the two factors co-existed, the risk of hypertension was higher than the risk of two factors alone. In view of the family history of hypertension is not controllable, it is suggested that community residents control their weight by regulating diet and strengthening exercise, to prevent the incidence of hypertension.

This study due to select cases from the database of hypertension registry and follow-up management system, not randomly selected from the population in communities, it is unavoidable some selective bias. And used data of registry, the number of cases with blood pressure <140/90mmHg was relatively less； It may weaken the causal relations, reduced estimation of exposure outcome. However, this study may still have research values and public health significance. The results show the correlation and combined effect of congenital family factors and acquired lifestyle factors. The future case-control studies will further show the independent effect and interaction of congenital genetic factors and acquired lifestyle factors.

5. Conclusions

Hypertension family history and body mass index have positive correlation with blood pressure. Both of them (family history and BMI) exist at the same time, can increase the risk of hypertension, and lead to earlier age of onset.

Abbreviations

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; BP: blood pressure.

S: synergistic effect index; RERI: relative excess risk due to interaction; AP: attributable proportion due to interaction; PAP: the percentage of the interaction between the pure factor.

Ethics Approval and Consent to Participate

Ethical approval was granted by Jiading district center for disease control and prevention research ethics committee. All subjects gave verbal and written informed consent to participate in the study, they would like to participate in investigation and answer all the related questions in the questionnaire. The survey was conducted by a full-time investigator using a questionnaire and face-to-face inquiries.

Availability of Data and Material

The datasets supporting the conclusions of this article are available via the hypertension registry and follow-up management system in Jiading district in Shanghai, contactable at http://219.233.222.99:8090/netchss/.

Competing Interests

The authors declare that they have no competing interests.

Funding

This study was funded by Jiading district health and family planning commission research project in Shanghai (N0:2016-KY-18)

Authors' Contributions

An-le Li is responsible for the design and implementation of the project, support of project funds, analysis of data and materials, and writing of manuscript; Yue-qin Shao seeks administrative support for projects and assists in the implementation of investigations; Hong Yuan participates in the project and assists in the investigation; Yi-ying Zhang participates in project investigation and quality control. All authors contributed to subsequent drafts and approved the final manuscript, and approved it for publication.

Acknowledgements

Heartfelt thanks the hard work of all doctors, nurses and public health workers in 13 community health service centers in Jiading district in Shanghai! and thank for the advice of the experts!

References

[1] Delles C, Mcbride MW, Graham D, Padmanabhan S, Dominiczak AF. Genetics of hypertension: from experimental animals to humans [J]. Bio Chim Bio physacta. 2010;1802(12):1299-1308.

[2] Whitworth JA. World Health Organization, International Society of Hypertension Writing Group. 2003 World Health Organization (WHO) / International Society of Hypertension(ISH) statement on management of hypertension [J]. J Hypert, 2003, 21(11):1983—1992

[3] Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data J 1. Lancet, 2005, 365(9455):217—223.

[4] Lawes CM, Vander Hoorn S, Rodgers A. Global burden of blood-pressure-related disease [J]. Lancet. 2008;371(9623):1513-1538.

[5] Ko-Ko-Zaw, Tint-Swe-Latt, Phyu-Phyu-Aung, Thein-Gi-Thwin, Tin-Khine-Myint. Prevalence of hypertension and its associated factors in the adult population in Yangon Division, Myanmar [J]. Asia Pac J Public Health, 2011, 23(4):496—506.

[6] Center for chronic non communicable diseases control and prevention of China Center for Disease Control and Prevention. Surveillance report of chronic diseases and their risk factors in China (2010)[M].{H} Beijing: Military Medical Science Press 2012.

[7] Levy D, Ehret GB, Rice K, Verwoert GC, Launer LJ. Genome-wide association study of blood pressure and hypertension [J]. Nat Genet.2009.41(6):677—687.

[8] Newton—Cheh C, Johnson T, Gateva V, Tobin MD, Bochud M. Genome-wide association study identifies eight loci associated with blood pressure [J]. Nat Genet, 2009, 4 1(6):666—676.

[9] Kato N, Takeuchi F, Tabara Y, Kelly TN, Go MJ. Meta—analysis of genome-wide association studies identifies common variants associated with blood pressure variation in east Asians [J]. Nat Genet, 2011, 43(6):53 1-538.

[10] International Consortium for Blood Pressure Genome-Wide Association Studies, Ehret GB, Munroe PB. et a1. Genetic variants in novel pathways influence blood pressure and cardiovascular disease risk [J]. Nature, 2011, 478(7367):103-109.

[11] Zhang Y, Shen J, He X. rare variant at the KYNU gene is associated with kynureninase activity and essential hypertension in the Han Chinese population [J]. Circ Cardiovasc Genet. 2011. 4(6):687—694.

[12] Rafiq S, Anand S, Roberts R. Genome-wide association studies of hypertension: have they been fruitful? [J]. Cardiovasc Transl Res. 2010;3(3):189-196.

[13] Tadic M, Cuspidi C, Vukomanovic V, Kocijancic V, Celic V, Stanisavljevic D. The association between obesity, blood pressure variability and right ventricular function and mechanics in hypertensive patients [J]. J AM Soc Echo cardiogr. 2016, 29(8): 802 -811.

[14] Tadic M, Cuspidi C, Pencic B, Majstorovic A, Sljivic A. The interaction between blood pressure variability, obesity, and left ventricular mechanics: findings from the hypertensive population [J]. J Hypertens. 2016, 34(4): 772 -780.

[15] Manimunda S. P., Sugunan A. P., Benegal V., Balakrishna N., Rao M. V., Pesala K. S. Association of hypertension with risk factors & hypertension related behaviour among the aboriginal Nicobarese tribe living in Car Nicobar Island, India. Indian Journal of Medical Research. 2011;133(3):287–293.

[16] Kotchen T. A. Obesity-related hypertension: epidemiology, pathophysiology, and clinical management. American Journal of Hypertension. 2010;23(11):1170–1178.

[17] Modesti P. A, Agostoni P, Agyemang C, Basu, S, Benetos, A, Cappuccio, F. Cardiovascular risk assessment in low-resource settings: a consensus document of the European Society of Hypertension Working Group on Hypertension and Cardiovascular Risk in Low Resource Settings. Journal of Hypertension. 2014;32(5):951–960.

[18] Wang Z, Zhang L, Chen Z, Miao Y, Gao S, Deng Y, et al. China Hypertension Survey G: survey on prevalence of hypertension in China: background, aim, method and design. International journal of cardiology 2014;174:721–3.

[19] Revision Committee of Guidelines for the Prevention and Treatment of Hypertension in China. Guidelines for the prevention and treatment of hypertension in China 2010 [J]. Chin J Cardio Med. 2011, 39(7):579-616.

[20] Joint Data Aggregation Analysis Collaboration Group of Working Group on Obesity in China of International Life Science Institute Focal Point in China. An introduction to the recommendation opinions on the classification of the adult body mass index in China [J]. Chin J Prev Med, 200l, 35(5):349—350.

[21] International Life Science Society China office, China Obesity Task Force, joint data analysis and collaboration group. A brief introduction to the recommendation of Chinese adult body mass index classification [J]. Chin J Prev Med.2001, 35(5):349—350.

[22] Liu M, He Y, Jiang B. Association between family history and hypertension among Chinese elderly [J]. Medicine, 2015, 94(48): e2226.

[23] Liu Dong-xia, Hua Qi, Guo Jin-cheng, Liu Rong-kun. Investigation and analysis of the characteristics and risk factors of hypertensive patients with family history and without family history [J]. Journal of cardiovascular and cerebrovascular diseases, 2006, 25(2):7l-74.

[24] Macumber IR, Weiss NS, Halbach SM, Hanevold CD, Flynn JT. The association of pediatric obesity with nocturnal non-dipping on 24-hour ambulatory blood pressure monitoring [J]. Am J Hypertens, 2016, 29 (5): 647-652.

[25] Olack B, Wabwire-Mangen F, Smeeth L, JM Montgomery, N Kiwanuka. Risk factors of hypertension among adults aged 35-64 years living in an urban slum Nairobi, Kenya [J]. BMC Public Health, 2015, 15(1):1.

[26] JOshi MD, Ayah R, Njau EK, Wanjiru R, Kayima JK, Njeru EK. Prevalence of hypertension and associated cardiovascular risk factors in an urban slum in Nairobi, Kenya: A population-based survey [J]. BMC Public Health, 2014, 14(1):1.

[27] Aune D, Sen A, Norat T, Janszky I, Romundstad P, Tonstad S, Vatten LJ. Body Mass Index, Abdominal Fatness, and Heart Failure Incidence and Mortality: A Systematic Review and Dose-Response Meta-Analysis of Prospective Studies [J]. Circulation, 2016, 133(7): 639-649.

[28] Wang Wen-juan, Wang Kean, Li Tian-lin, H Xiang, L Ma. The practical value and its suggested value of body mass index, waist circumference and waist hip ratio in predicting hypertension and hyperglycemia [J]. Chinese Journal of Epidemiology, 2002, 23(1):16—19.

[29] Zhao Zhong-tang editor. Epidemiology research methods and Applications (Second Edition) [M]. Science Press, medical publishing branch, August 2005.