

ORIGINAL ARTICLE

Hay fever and hypertension in the US adult population

Chao Li, Ching-Lung Cheung, Tommy T. Cheung, Nithushi R. Samaranayake, and Bernard M. Y. Cheung

Department of Medicine, University of Hong Kong, Queen Mary Hospital, Hong Kong, China

Abstract

Purpose: Hypertension is associated with inflammation. Whether the inflammation caused by allergic diseases such as allergic rhinitis can predispose to hypertension is controversial. Therefore, we studied the association between hay fever and hypertension in the United States National Health and Nutrition Examination Survey (NHANES).

Methods: We analyzed data on 1883 men and 2029 women in NHANES 2005–2006. We included participants aged 20 years or older who had valid data on hay fever and hypertension.

Results: 13.5% of the participants had a previous diagnosis of hay fever and 36.2% of them had hypertension. There were ethnic differences in the prevalence of previous hay fever diagnosis ($p < 0.001$) and hypertension ($p < 0.001$). There was no significant association between previous hay fever diagnosis and hypertension in men in any age group. The association between previous hay fever diagnosis and hypertension in women was significant in those aged 20–39 years (OR = 2.59, 95%CI = 1.26–5.30, $p = 0.013$). The association between previous hay fever diagnosis and hypertension in women aged from 20 to 39 years remained significant after adjustment for age, race and body mass index (OR = 2.74, 95%CI = 1.48–5.06, $p = 0.003$). After further adjustment for physical activity, alcohol consumption and smoking, the association was not attenuated (OR = 2.68, 95%CI = 1.38–5.18, $p = 0.006$). Further adjustment for liver enzymes, C-reactive protein and immunoglobulin E level attenuated the association slightly (OR = 2.72, 95%CI = 1.19–6.22, $p = 0.021$).

Conclusions: In this nationally representative population-based survey, previous hay fever diagnosis is not significantly associated with hypertension in adults overall. There is an association in subgroup of young women aged 20–39. Further work is needed to confirm that this is a true association.

Keywords

Allergic rhinitis, blood pressure, hypertension, NHANES

History

Received 5 February 2013

Revised 24 March 2013

Accepted 2 April 2013

Published online 20 June 2013

Introduction

Allergic rhinitis is common in the general population, especially among adolescents and young adults. It occurs in individuals with a sensitized immune system when an allergen (e.g. pollen, dust or animal hairs) is inhaled and triggers an immunoglobulin E (IgE)-mediated allergic response and inflammation (1).

Low-grade chronic inflammation plays an important role in both the development and the progression of hypertension (2). The mechanism underlying the association between hypertension and chronic inflammation has been studied in the past decades. Recently, epidemiological evidence has demonstrated the association between hypertension and several chronic inflammatory diseases such as periodontitis and inflammatory bowel diseases (3,4). However, studies exploring the association between rhinitis and hypertension have yielded conflicting results (5). Therefore, we studied the

relationship between hay fever, which is a type of allergic rhinitis due to outdoor triggers, and hypertension in the US population.

Methods

Study population

For this analysis, we used data from the United States National Health and Nutrition Examination Survey (NHANES). All participants gave informed consent, and the study protocol was approved by the Centers for Disease Control and Prevention Institutional Review Board. Participants were examined in well-equipped mobile examination centers located around the US. Detailed measurement procedures and protocols are described on the NHANES website (6). Participants were selected using a stratified, multistage probability sampling design from the civilian non-institutionalized US population. In NHANES 2005–2006, there were 4504 participants aged ≥ 20 years who had valid data on hay fever and hypertension. Among them, 3912 participants had valid data on body mass index (BMI), alcohol consumption, smoking status, anti-hypertensive medication status, physical activity and laboratory data, including

Correspondence: Prof Bernard M. Y. Cheung, Department of Medicine, Queen Mary Hospital, 102, Pokfulam Road, Hong Kong, China. Tel: +852-22554347. Fax: +852-28186474. E-mail: mycheung@hku.hk

C-reactive protein (CRP), IgE, white blood cell (WBC) level and liver enzymes. Participants were categorized into age groups of 20–39, 40–59 and ≥ 60 years for analysis.

Variables of interest

Hypertension was defined as previous diagnosis by a doctor, with blood pressure $\geq 140/90$ mm Hg or ever use of any anti-hypertensive medications. Definition of previous diagnosis of hypertension was according to the questionnaire “have you/has your patient ever been told by a doctor or other health professional that you/she/he had hypertension, also called high blood pressure?” Participants taking anti-hypertensive medications were determined by the questionnaire “are you/is your patient now taking prescribed medicine for hypertension?” Blood pressure was measured manually by a trained operator using a mercury sphygmomanometer. After resting quietly in a sitting position for 5 minutes, three consecutive blood pressure readings were obtained. Five sizes of cuff fit for appropriate arm were applied in measuring blood pressure. If a blood pressure measurement is interrupted or incomplete, a fourth attempt might be made. All blood pressure measurements (systolic and diastolic) were taken in the Mobile Examination Center.

A previous diagnosis of hay fever was determined by the question in the 2005–2006 survey – “has a doctor or other health professional ever told you/survey participant that you have/she/he has hay fever?”

Race/ethnicity was based on self-report in the questionnaires. Participants were classified as non-Hispanic whites, non-Hispanic blacks, Mexican Americans and others. Adults should wear only underwear, disposable paper gowns and foam slippers when measuring weight. BMI was calculated as the weight in kilograms divided by the square of the height in meters. Never smokers were defined as participants who never smoked or smoked <100 cigarettes in their lives. Current smokers were defined as participants who smoked ≥ 100 cigarettes in their lives and reported current smoking. Former smokers were defined as participants who smoked ≥ 100 cigarettes in their lives, but reported current cessation of smoking. Alcohol drinking was defined as drinking any type of alcohol beverage at least once a week (5 times a month or 53 times a year as an equivalent) in the past 1 year. Participants having vigorous or moderate activity were considered to be physically active.

Plasma CRP was measured in participants using a latex-enhanced nephelometric method. Serum alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transpeptidase (GGT) activities were determined with a Beckman Synchron LX20 analyzer (Beckman Coulter Inc., Fullerton, CA). Samples for ALP and CRP analysis that were not assayed within 48 h were frozen and thawed only once to avoid analyte degradation and abnormal over expression. Serum samples were analyzed for total IgE using the Pharmacia Diagnostics ImmunoCAP 1000 System (Kalamazoo, MI). A more detailed explanation of the study design, interview techniques, measurement procedures and protocol is given on the NHANES website (6). ALT, AST, GGT, ALP, CRP, WBC and IgE were treated as covariates.

Data were analyzed using the complex sampling function of SPSS version 20.0 (IBM Corporation, Armonk, NY). Two-year mobile examination weights were used in all analyses to account for non-response bias and the oversampling of blacks, Mexican Americans and the elderly in NHANES, so that the estimates were nationally representative according to the US Census population. *p* Values were obtained using logistic and multiple regression analysis, where appropriate, using the complex sampling function. Logistic and multiple regressions were used to adjust for covariates. Variables that significantly differed between participants with and without hay fever, or with and without hypertension, were used as covariates in the regression models. Variables with a skewed distribution were log transformed in assessing *p* and all regression analysis.

Results

Table 1 shows the prevalence of previous hay fever diagnosis and hypertension in men and women of different age groups in NHANES 2005–2006. A total of 12.4% of men and 14.4% of women had been told to have hay fever, and 37.0% of men and 35.5% of women had hypertension. All the percentages were calculated after weighting to the US Census population. The prevalence of hypertension increased with age. Men and women aged 40–59 years had a higher rate of previous diagnosis of hay fever than other age groups.

Table 2 shows the characteristics of all participants according to previous hay fever diagnosis. Participants with previous hay fever diagnosis were older ($p=0.039$ after adjusted for gender and race/ethnicity) and more likely to never smoke or smoke previously but not currently, compared to participants without previous hay fever diagnosis ($p=0.052$ after adjusted for age and race/ethnicity). There were significant racial/ethnic differences between participants with and without hay fever ($p<0.001$ after adjusted for gender and age). The GGT level of participants with previous hay fever diagnosis tended to be higher than that of participants without previous hay fever diagnosis ($p=0.057$ after adjusted for age and race/ethnicity). The IgE level of participants with previous hay fever diagnosis was significantly higher than those of participants without previous hay fever diagnosis ($p<0.001$ after adjusted for age and race/ethnicity).

Table 3 shows the characteristics of all participants according to hypertension status. Participants with

Table 1. Prevalence of previous hay fever diagnosis and hypertension.

	<i>n</i>	Hay fever	Hypertension
Men	1883	12.4 (1.0)	37.0 (1.4)
20–39	645	7.3 (0.7)	18.5 (2.6)
40–59	605	16.1 (2.1)	38.6 (2.1)
≥ 60	633	14.4 (1.7)	66.7 (2.5)
Women	2029	14.5 (1.0)	35.5 (1.4)
20–39	831	11.6 (2.2)	7.9 (1.2)
40–59	621	17.6 (1.9)	38.5 (3.0)
≥ 60	577	13.4 (1.6)	71.5 (2.1)

Data are expressed as percent (SE).

Table 2. Characteristics of all 3912 participants with and without previous hay fever diagnosis.

Characteristics	Hay fever	No hay fever	<i>p</i> ^a
<i>n</i>	419	3493	—
Age, years	48.9 (1.0)	46.4 (0.8)	0.039
Men, %	44.4 (2.2)	48.8 (0.8)	0.117
BMI, kg/m ²	28.7 (0.5)	28.5 (0.3)	0.534
Race/ethnicity, %			
Non-Hispanic white	84.3 (1.7)	71.7 (2.8)	<0.001
Non-Hispanic black	6.7 (1.5)	11.1 (1.9)	
Mexican American	2.8 (0.7)	8.7 (1.1)	
Others	6.2 (1.2)	8.5 (1.2)	
Smoking, %			
Never	54.8 (3.0)	50.1 (1.5)	0.052
Former	26.9 (3.2)	25.0 (1.1)	
Current	18.3 (2.0)	24.9 (1.3)	
Alcohol drinking, %	36.2 (2.3)	35.0 (1.7)	0.805
Physical activity, %	71.0 (2.9)	67.3 (1.8)	0.267
SBP ^c , mmHg	119.3 (0.7)	119.2 (0.5)	0.354
DBP ^c , mmHg	70.9 (0.9)	69.6 (0.4)	0.107
ALT, U/L	22.95 (21.63–24.34)	22.89 (22.43–23.37)	0.367 ^b
AST, U/L	23.92 (22.82–25.06)	24.14 (23.78–24.49)	0.858 ^b
GGT, U/L	22.24 (20.69–23.91)	20.97 (20.21–21.75)	0.057 ^b
ALP, U/L	65.46 (61.49–69.71)	65.69 (64.37–67.03)	0.924 ^b
CRP, mg/dL	2.10 (1.68–2.62)	1.83 (1.67–2.00)	0.190 ^b
WBC, 1000 cells/uL	7.12 (6.92–7.33)	7.06 (6.93–7.20)	0.739 ^b
IgE, kU/L	57.56 (47.33–70.02)	38.43 (35.43–41.69)	<0.001 ^b

Data are expressed as mean or percent (SE), or geometric mean (95% confidence interval).

SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-reactive protein; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, glucosyltransferase; ALP, alkaline phosphatase; and WBC, white blood cell.

^a*p* was derived from multiple logistic or linear regression models, where appropriate, after adjusting for age (except age-specific estimates), sex (except sex-specific estimates), race/ethnicity (except race/ethnicity-specific estimates) and survey years.

^bData were log-transformed in assessing *p* due to their skewed distribution.

^cA total of 898 participants taking anti-hypertensive medication were excluded from the analysis (*n* = 308 for previous hay fever diagnosis group and *n* = 2706 for no previous hay fever diagnosis group).

Table 3. Characteristics of all 3912 participants with and without hypertension.

Characteristics	Hypertension	No hypertension	<i>p</i> ^a
<i>N</i>	1514	2398	—
Age, years	56.6 (1.3)	41.1 (0.6)	<0.001
Men, %	49.2 (1.4)	47.6 (0.9)	0.036
BMI, kg/m ²	30.4 (0.3)	27.5 (0.3)	<0.001
Race/ethnicity, %			
Non-Hispanic white	75.6 (2.6)	72.1 (2.8)	<0.001
Non-Hispanic black	12.8 (2.2)	9.1 (1.6)	
Mexican American	4.9 (0.9)	9.6 (1.1)	
Others	6.7 (1.1)	9.1 (1.3)	
Smoking, %			
Never	47.8 (2.1)	52.4 (1.4)	0.561
Former	30.8 (1.4)	22.1 (1.3)	
Current	21.4 (1.7)	25.5 (1.6)	
Alcohol drinking, %	34.2 (1.6)	35.7 (2.0)	0.790
Physical activity, %	61.8 (1.6)	71.2 (2.1)	0.271
SBP ^c , mmHg	137.9 (0.8)	114.8 (0.4)	<0.001
DBP ^c , mmHg	76.9 (0.6)	68.1 (0.4)	<0.001
ALT, U/L	23.73 (22.94–24.55)	22.47 (21.88–23.08)	<0.001 ^b
AST, U/L	24.85 (24.27–25.46)	23.71 (23.33–24.10)	0.008 ^b
GGT, U/L	24.99 (23.91–26.10)	19.25 (18.60–19.92)	<0.001 ^b
ALP, U/L	69.84 (67.64–72.13)	63.37 (61.92–64.86)	0.003 ^b
CRP, mg/dL	2.43 (2.19–2.71)	1.60 (1.44–1.78)	<0.001 ^b
WBC, 1000 cells/uL	7.15 (6.98–7.32)	7.03 (6.90–7.16)	<0.001 ^b
IgE, kU/L	43.87 (38.22–50.37)	38.74 (36.0–41.70)	0.010 ^b

Data are expressed as mean or percent (SE), or geometric mean (95% confidence interval).

^a*p* was derived from multiple logistic or linear regression models, where appropriate, after adjusting for age (except age-specific estimates), sex (except sex-specific estimates), race/ethnicity (except race/ethnicity-specific estimates), and survey years.

^bData were log-transformed in assessing *p* due to their skewed distribution.

^cA total of 898 participants taking anti-hypertensive medication were excluded from the analysis (*n* = 308 for previous hay fever diagnosis group and *n* = 2706 for no previous hay fever diagnosis group).

Table 4. Association of previous hay fever diagnosis with hypertension in men.

	Unadjusted	Model 1	Model 2	Model 3
20–39				
OR	0.88	0.77	0.74	0.73
95%CI	0.29–2.64	0.24–2.43	0.21–2.57	0.23–2.36
p	0.809	0.629	0.616	0.575
40–59				
OR	0.89	0.84	0.87	0.64
95%CI	0.53–1.50	0.50–1.41	0.53–1.43	0.30–1.35
p	0.639	0.477	0.563	0.221
≥60				
OR	1.23	1.50	1.31	1.31
95%CI	0.58–2.62	0.74–3.06	0.61–2.84	0.55–3.12
p	0.570	0.241	0.465	0.514

Model 1: Adjusted for age, race and BMI (kg/m²).
Model 2: Further adjusted for physical activity, alcohol consumption and smoking.
Model 3: Further adjusted for ALT (ln U/L), AST (ln U/L), GGT (ln U/L), ALP (ln U/L), CRP (ln mg/dL), WBC (ln 1000 cells/uL) and IgE (ln kU/L).

Table 5. Association of previous hay fever diagnosis with hypertension in women.

	Unadjusted	Model 1	Model 2	Model 3
20–39				
OR	2.59	2.74	2.68	2.72
95%CI	1.26–5.30	1.48–5.06	1.38–5.18	1.19–6.22
p	0.013	0.003	0.006	0.021
40–59				
OR	0.87	0.97	0.99	0.95
95%CI	0.48–1.58	0.52–1.79	0.53–1.87	0.48–1.87
p	0.634	0.904	0.985	0.868
≥60				
OR	0.68	0.63	0.64	0.59
95%CI	0.35–1.31	0.31–1.25	0.33–1.25	0.31–1.13
p	0.231	0.167	0.178	0.105

Model 1: Adjusted for age, race and BMI (kg/m²).
Model 2: Further adjusted for physical activity, alcohol consumption and smoking.
Model 3: Further adjusted for ALT (ln U/L), AST (ln U/L), GGT (ln U/L), ALP (ln U/L), CRP (ln mg/dL), WBC (ln 1000 cells/uL) and IgE (ln kU/L).

hypertension were older ($p < 0.001$ after adjusted for gender and race/ethnicity) and had a greater BMI ($p < 0.001$ after adjusted for age and race/ethnicity), compared to participants without hypertension. There were significant racial/ethnic differences between participants with and without hypertension ($p < 0.001$ after adjusted for gender and age). Compared with participants without hypertension, participants with hypertension had higher levels of ALT ($p < 0.001$), AST ($p = 0.008$), GGT ($p < 0.001$), ALP ($p = 0.003$), CRP ($p < 0.001$), WBC ($p < 0.001$) and IgE ($p = 0.010$) after adjustment for age and race/ethnicity.

Tables 4 and 5 show the logistic regression analysis before and after adjusting for covariates. There was no significant association between previous hay fever diagnosis and hypertension in men in any model in any age group before or after adjustment. The association between previous hay fever diagnosis and hypertension in women was also

insignificant except those aged 20–39 years (OR = 2.59, 95%CI = 1.26–5.30, $p = 0.013$). The association between previous hay fever diagnosis and hypertension in women aged form 20 to 39 years remained significant after adjustment for age, race and BMI (OR = 2.74, 95%CI = 1.48–5.06, $p = 0.003$). Further adjustment for physical activity, alcohol consumption and smoking did not attenuate the association (OR = 2.68, 95%CI = 1.38–5.18, $p = 0.006$). After adjustment for liver enzymes, WBC and IgE, the association was slightly attenuated but still significant (OR = 2.72, 95%CI = 1.19–6.22, $p = 0.021$).

Discussion

The main finding in our analysis of NHANES data was that a previous diagnosis of hay fever was associated with hypertension in women aged 20–39 years. This association remained significant after adjustment for covariates.

An association between rhinitis and increased systolic blood pressure was found in a study of 330 men aged 28–56 years in the European Community Respiratory Health Survey (ECRHS) (7). However, this conclusion was challenged by another group analyzing the MONICA/KORA study database whose rhinitis assessment was according to ECRHS screening questionnaire (8). Besides, a study of Japanese adolescents showed that diastolic blood pressure was higher in participants without rhinitis than in those with rhinitis (9).

While findings of previous studies are controversial, there are some possible mechanisms linking rhinitis with hypertension. Inflammation occurs in both allergic rhinitis and hypertension. In patients with both hypertension and allergic rhinitis, treatment of allergic rhinitis can improve blood pressure control (10). In hypertensive patients, the serum levels of inflammatory marker like CRP, ALT, AST, GGT and ALP are increased. Serum CRP level also predicts the future development of hypertension (11,12). In our study, serum levels of CRP, ALT, AST, GGT and ALP were associated with hypertension but not related to hay fever though inflammation occurs in allergic rhinitis as well. Perhaps, elevated CRP level in individuals with hay fever is not sensitive enough to reflect the systemic inflammation in allergic rhinitis. Therefore, inflammatory mechanism behind the possible link between hypertension and allergic rhinitis still has to be well hypothesized and tested.

Although no strong evidence that allergic rhinitis is a significant cause of hypertension has been found in this clinical study, there is a theoretical basis for this association and several mechanisms can be envisaged. A defect in the Na⁺/K⁺ pump is one of the possible elements for linking allergic rhinitis with hypertension. Na⁺/K⁺ pump exports sodium from the cell and imports potassium into the cell. A defect in the Na⁺/K⁺ pump leads to intracellular sodium accumulation. Previous studies suggested an accumulation of sodium in the platelets, leukocytes and the peripheral blood mononuclear cells in individuals with allergic rhinitis (13,14). Elevated cytosolic sodium level also promotes calcium influx, thereby raising intracellular Ca²⁺ levels (15,16). Increased intracellular calcium is required for the contraction of vascular smooth muscle cell that is highly sensitive to

extracellular calcium influx. If Na⁺/K⁺ pump is defective in vascular smooth muscle, these processes could possibly further lead to elevation in blood pressure. It was proposed that Na⁺,K⁺-ATPase inhibitor of unknown molecular structure may partly regulate the mechanical activity of cardiovascular muscle and participate in the genesis of certain forms of hypertension (17). Therefore, a defect in Na⁺/K⁺ pump activity was suggested to be associated with both rhinitis and hypertension. Furthermore, hypertension in allergic individuals may be controlled by a medication that targets Na⁺/K⁺ pump (14), which also supported that Na⁺/K⁺ pump participated in the possible association between rhinitis and hypertension.

Another reason to account for the association between rhinitis and high blood pressure is that patients with hay fever have inflamed nasal passages leading to nasal obstruction, snoring, hypopnea and apnea during sleep (18,19). Sleep apnea is a well-known risk factor for hypertension and cardiovascular diseases by inducing hypoxemia, hypercapnia, a decrease in serum pH and an increase in catecholamine levels. A study in Italy showed that rhinitis and snoring might indirectly increase the risk of hypertension in post-menopausal women (20).

Our study had several limitations. First of all, previous diagnosis of hay fever was determined by questionnaire in NHANES database. The diagnosis was not checked with the participants' medical records. Second, this is a cross-sectional analysis. Thus, we focus on exploring whether there is an association, but these findings are inherently limited in the ability to eliminate causal relationships between hay fever and hypertension. Finally, we divided the population into three age subgroups due to differences in the prevalence of allergic rhinitis and hypertension in different age groups. Dividing the population into different age groups was a necessary process but reduced the sample size.

In conclusion, in our 2-year database study, previous diagnosis of hay fever was not associated with hypertension in adults overall. There is an association in subgroup of women aged 20–39 years before or after adjustment but not in other groups. Studies of large sample size with more comprehensive information on risk factors are needed to elucidate the complex relationship.

Declaration of interest

The authors declare no conflict of interest.

B. M. Y. Cheung received support from the University of Hong Kong Seed Funding Programme for Basic Research.

References

1. Ricca V, Landi M, Ferrero P, et al. Minimal persistent inflammation is also present in patients with seasonal allergic rhinitis. *J Allergy Clin Immunol* 2000;105:54–7.
2. Savoia C, Schiffrin EL. Vascular inflammation in hypertension and diabetes: molecular mechanisms and therapeutic interventions. *Clin Sci* 2007;112:375–84.
3. Tsioufis C, Kasiakogias A, Thomopoulos C, Stefanadis C. Periodontitis and blood pressure: the concept of dental hypertension. *Atherosclerosis* 2011;219:1–9.
4. Leal-Valdivieso C, Naves JE, Manosa M, et al. Portal hypertension in patients with inflammatory bowel disease. *Gastroenterol Hepatol* 2010;33:297–302.
5. Chester AC. Evidence is lacking for link between rhinitis and hypertension. *Am J Respir Crit Care Med* 2004;169:428.
6. Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS): National Health and Nutrition Examination Survey questionnaire, examination protocol and laboratory protocol. Hyattsville (MD): United States Department of Health and Human Services, Centers for Disease Control and Prevention, 1999–2002. Available from: <http://www.cdc.gov/nchs/nhanes.htm> [last accessed 30 Nov 2011].
7. Kony S, Zureik M, Neukirch C, et al. Rhinitis is associated with increased systolic blood pressure in men: a population-based study. *Am J Respir Crit Care Med* 2003;167:538–43.
8. Heinrich J, Doring A. Blood pressure and rhinitis in adults: results of the MONICA/KORA-study. *J Hypertens* 2004;22:889–92.
9. Saito I, Mori M, Shibata H, et al. Relation between blood pressure and rhinitis in a Japanese adolescent population. *Hypertens Res* 2003;26:961–3.
10. Magen E, Yosefy C, Viskoper RJ, Mishal J. Treatment of allergic rhinitis can improve blood pressure control. *J Hum Hypertens* 2006;20:888–93.
11. Sesso HD, Buring JE, Rifai N, et al. C-reactive protein and the risk of developing hypertension. *JAMA* 2003;290:2945–51.
12. Cheung BM, Ong KL, Tso AW, et al. C-reactive protein as a predictor of hypertension in the Hong Kong Cardiovascular Risk Factor Prevalence Study (CRISPS) cohort. *J Hum Hypertens* 2012;26:108–16.
13. Hirota SA, Janssen LJ. Sodium and asthma: something borrowed, something new? *Am J Physiol Lung Cell Mol Physiol* 2007;293:L1369–73.
14. Skoner DP, Gentile D, Evans RW. A circulating inhibitor of the platelet Na⁺,K⁺ adenosine triphosphatase (ATPase) enzyme in allergy. *J Allergy Clin Immunol* 1991;87:476–82.
15. Blaustein MP, Lederer WJ. Sodium/calcium exchange: its physiological implications. *Physiol Rev* 1999;79:763–854.
16. Sweeney G, Klip A. Mechanisms and consequences of Na⁺,K⁺-pump regulation by insulin and leptin. *Cell Mol Biol (Noisy-le-grand)* 2001;47:363–72.
17. Haddy FJ. Potassium, Na⁺-K⁺ pump inhibitor and low-renin hypertension. *Clin Invest Med* 1987;10:547–54.
18. Storms W. Allergic rhinitis-induced nasal congestion: its impact on sleep quality. *Prim Care Respir J* 2008;17:7–18.
19. Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep-disordered breathing. The University of Wisconsin Sleep and Respiratory Research Group. *J Allergy Clin Immunol* 1997;99:S757–62.
20. Corbo GM, Forastiere F, Agabiti N, et al. Rhinitis and snoring as risk factors for hypertension in post-menopausal women. *Respir Med* 2006;100:1368–73.