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Effect of Dietary Sodium and Potassium Intake on Left Ventricular Diastolic Function and Mass in Adults ≤40 Years of Age (From the Strong Heart Study)

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Abstract

The aim of this study was to investigate whether intake of dietary sodium or potassium is related to changes in left ventricular (LV) diastolic functioning and LV mass index in young individuals with normal or elevated blood pressure. We prospectively analyzed echocardiographic data in 1,065 young adults (18–39 years) enrolled in the Strong Heart Family Study who were free from cardiovascular disease at baseline: 501 (47%) participants were normotensive and 564 (53%) were pre-hypertensive or hypertensive. Dietary sodium and potassium intake was ascertained by using a Block food-frequency questionnaire at baseline. Cardiac geometry and functioning was assessed at baseline and 4 years later. Marginal models were used to assess the associations of average intakes of sodium and potassium with echocardiographic measures. Participants with pre-hypertension or hypertension were older, had higher body mass index and reported higher intakes of sodium than normotensive individuals at baseline. In prospective analyses, potassium intake was found to be negatively related to mitral E-velocity (p=0.029) in normotensive individuals whereas sodium/potassium ratio was positively associated with atrial filling fraction (p=0.017). In pre-hypertensive

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Disclosures

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or hypertensive participants sodium consumption was positively associated with atrial filling fraction (p=0.034) and an increase in sodium/potassium ratio was related to higher LV mass index (p=0.046). In conclusion, an increase in dietary sodium/potassium ratio was related to an accentuation of atrial-phase LV diastolic filling in normotensive young individuals while in pre-hypertensive or hypertensive individuals it was associated with higher LV mass index.

Keywords

Sodium; Potassium; Sodium/Potassium Ratio; Diastole; Left ventricular mass; Echocardiography

Sodium restriction and potassium supplementation are recommended dietary measures to prevent cardiovascular disease.[1] Previous studies have linked dietary sodium intake with changes in cardiac structure, notably increased left ventricular (LV) mass.[2,3] Already prior to cardiac remodeling, measures of cardiac functioning, deteriorate.[4–6] In otherwise healthy patients at high risk or at an early stage for hypertensive disease, subtle changes in cardiac diastolic functioning can be detected by echocardiography as a consequence of dietary sodium loading which may indicate an early stage of LV remodeling.[7] To this point surprisingly little is known about the relation between dietary sodium and potassium intake and LV structure and function in young individuals in large population-based samples as comprehensive prospective analyses are largely missing.[3] Early identification of echocardiographic alterations in LV function in young populations could help to screen for those individuals at high risk for LV structural changes. The aim of this study was to investigate whether dietary sodium or potassium intake is related to changes in LV diastolic function and LV mass in young individuals with normal or elevated blood pressure (BP).

Methods

The Strong Heart Study (SHS) is a longitudinal population-based survey of cardiovascular risk factors and disease in American Indians from 13 communities in Arizona, Oklahoma, and South and North Dakota that was initiated in 1988. The SHS design and methods have been described previously.[8] In brief, the Strong Heart Family Study (SHFS) was conducted between 2001 and 2003 (SHS exam IV) with a follow-up visit in 2007–2009 (SHS exam V). It enrolled 1468 men and 2197 women from 96 large families of SHS participants. All participants of the SHFS received extensive examinations including a transthoracic echocardiogram at both visits.[9] For this analysis, we included only SHFS participants aged 14-to 39-years. Participants with a history of any cardiovascular disease (i.e. myocardial infarction, angina pectoris, heart failure, coronary bypass surgery, angioplasty, carotid endarterectomy, valve replacement and significant valve disease [aortic or mitral stenosis or more than mild regurgitation] or history of stroke at SHS exam IV were excluded. Individuals with missing or incomplete dietary information or with extreme calorie intake (i.e., <500 kcal or > 3500 kcal per day) were also excluded from this analysis. Our final study population consisted of 1,065 study participants. Participants were followed-up for an average of 4 years. The institutional review boards (Cornell University, MedStar Health, and University of Oklahoma), Indian Health Service IRB (Phoenix, Oklahoma and Aberdeen) and each participating tribe approved the study. Written informed consent was obtained

from all participants at enrollment. BP status was assessed by the average of two blood-pressure readings at baseline examination. Pre-Hypertension was defined as systolic blood pressure (SBP) between 120–139 mmHg and diastolic blood pressure (DBP) between 80–89 mmHg with no hypertension treatment. Hypertension was defined as SBP≥140 or DBP≥90, or taking hypertension medication.[10] Diabetes was diagnosed if fasting plasma glucose ≥ 126 mg/dL or if the participant was on diabetes medications.[11] Body mass index was calculated as body weight divided by height squared (kg/m^2). An interviewer-administered Block 119-item food frequency questionnaire (FFQ) was applied to all participants at baseline.[12,13] To compute measures of average daily sodium and potassium intake, the frequency response for each food on the FFQ was multiplied by the nutrient content of the documented portion size of the food, then summed for all foods.[12,13] Echocardiographic measures of cardiac geometry and function were collected in all participants by expert sonographers and reviewed offline by a highly-experienced investigator. Cardiac geometry was assessed by the following parameters: LV internal diameter was measured at end diastole and adjusted for height while left atrial diameter was measured in end-systole. LV mass was calculated by a necropsy-validated formula and was normalized to height in meters$^{2.7}$ (LV mass index).[14] Left ventricular systolic function was assessed by calculating ejection fraction as the ratio of stroke volume/end diastolic volume.[15] Stroke volume was calculated as the difference between end diastolic and end systolic volumes by the z-derived method and was normalized to height $^{2.04}$.[16] LV diastolic function was evaluated by Doppler interrogation. Transmitral early (E) and late (A) LV filling velocity were measured at the annular level and used to compute the early peak rapid filling velocity to peak atrial filling velocity ratio (E/A ratio). Similarly, deceleration time (DT) of early diastolic LV filling, and the atrial filling fraction were calculated. Isovolumic relaxation time (IVRT), a raw index of active LV relaxation, was measured between aortic valve closure and mitral valve opening.[4,17,18] The baseline characteristics and echocardiographic measures by hypertension status at baseline (SHS-exam IV) are compared using t-test, logistic or marginal models.[19] The marginal models were also used to assess the association of average intakes of sodium and potassium as well as sodium/potassium ratio in SHS exam IV with echocardiographic measures in SHS exam V separately for normotensive and pre-hypertension/hypertension groups. Models were adjusted for age, sex, field center, relatedness among family members, and the respective baseline echocardiographic measures. Similar to previous analyses from the Strong Heart Family Study the impact of relatedness among family members was considered by using standard kinship coefficients (i.e., 0.25 for parent/offspring, 0.25 for full siblings, 0.125 for half siblings, and 0 for no consanguinity).[18–20] All p-values were 2-tailed. A p-value <0.05 was considered significant. Data were analyzed with SAS 9.3 (SAS Corp, Cary, NC).

Results

Characteristics of study participants by BP status at baseline (SHS exam IV) are presented in Table 1. Participants with pre-/hypertension were significantly older, predominantly female, more likely to be diabetic, had higher body mass index, and consumed higher intakes of sodium. Echocardiographic measures of pre-/hypertensive and normotensive individuals at baseline and the follow-up exams are presented in Table 2. Left atrium diameter, LV

diameter, and LV mass index were significantly higher in prehypertensives/hypertensives. Among parameters of cardiac functioning, prehypertensives/hypertensives had a significantly higher heart rate and stroke work but lower systolic output reflected by lower ejection fraction. In diastole, mitral E velocity was similar, whereas mitral A velocity was significantly higher resulting in a lower E to A ratio in rehypertensives/hypertensives. Last, DT and IVRT were significantly longer, and atrial filling fraction was higher in prehypertensives/hypertensives. All changes of those cardiac geometry and functioning examinations in Table 2 were significant except heart rate and DT in both prehypertensives/hypertensives and normotensives, A-velocity in normotensives and stroke work in prehypertensives/hypertensives. Associations of changes in cardiac geometry are presented in Table 3 and Table 4. In normotensive participants (Table 3), sodium or potassium intake was not associated with changes in left atrium diameter, LV diameter, or LV mass. Among parameters of cardiac diastolic functioning, potassium intake at baseline was found to be negatively related to change in mitral E-velocity ($p = 0.0294$, $p = 0.0231$ after further adjustment for baseline diabetes status), whereas sodium/potassium ratio was positively associated with change in the atrial filling fraction ($p = 0.0171$, $p = 0.018$ after further adjustment for baseline diabetes status). Sodium or potassium intake did not alter LV systolic functioning or other LV diastolic parameters such as DT, IVRT, or E to A ratio. In prehypertensives/hypertensives (Table 4), sodium/potassium ratio was found to be positively associated with LV mass index ($p = 0.0459$, $p = 0.0486$ after further adjustment for baseline diabetes status). Atrial filling fraction was observed to be positively related to sodium consumption ($p = 0.0338$, $p = 0.0335$ after further adjustment for baseline diabetes status), whereas other cardiac functioning parameters were not associated with either sodium or potassium intake.

Discussion

In this prospective community based study of young American Indians, an increase in sodium/potassium ratio was related to an increase in atrial filling fraction in normotensive individuals, whereas in pre-hypertensive or hypertensive individuals it was associated with an increase in LV mass.

Thus far, cross-sectional data on the impact of dietary sodium intake on LV diastolic filling in young and healthy individuals indicate that in normotensives and in individuals at early stage of hypertension high sodium intake is associated with impaired LV diastolic filling.[7] The largest prospective analyses including 1,042 participants to examine an association between sodium and potassium intake and changes in cardiac structure among young individuals were undertaken using data from the CARDIA Study.[3] There the baseline higher sodium to potassium excretion ratio was found to be significantly associated with the higher prospective (5 years later) left ventricular mass among healthy young adults. However, this association was found to be contributed by the baseline left ventricular mass since it did not persist once the baseline left ventricular mass was adjusted in the model.[3] Further structural and functional parameters were unfortunately not presented in this cohort study. Our data do not support changes in cardiac structure in normotensive individuals as a consequence of sodium or potassium intake. However, we observed slight changes and modifications in diastolic parameters associated with dietary sodium and potassium intakes. Mitral E-velocity

was inversely related to potassium intake whereas sodium/potassium ratio was positively associated with atrial filling fraction. These changes may be explained by hemodynamic responses to sodium loading. High sodium intake in normotensive individuals produces a rise in cardiac output by increasing LV preload which influences LV diastolic filling. Thus, a dietary pattern rich in sodium increases plasma volume as compared to a potassium rich diet and thereby atrial filling is increased. Despite diastolic changes, cardiac structure remained unchanged in normotensive individuals suggesting that the undamaged myocardium of normotensive individuals is still capable of tolerating increased preload for a certain period without increases in LV or left atrial diameters. Eventually, cardiac geometry will adopt. In fact, in pre-hypertensive or hypertensive participants, we found an increase in sodium/potassium ratio to be positively associated with LV mass after adjusting for baseline LV mass. Our findings therefore emphasize the view that an unhealthy dietary pattern characterized by excess sodium intake deteriorates the existing negative effects of BP elevations on LV mass.[21] Our analyses in young individuals complement previous studies linking excess sodium intake with early cardiac diastolic functional changes and eventual structural changes on a population level.[2] Strengths of our analysis include the sample size of our study population, a prospective design with long follow-up and standardized assessment of echocardiographic measures. In contrast, our study may lack generalizability as our cohort was limited to American Indians. Sodium and potassium intakes were determined at baseline using food frequency questionnaires that may not represent actual sodium and potassium intakes.[22]

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Table 1

Characteristics of study participants at baseline

Variable	Pre-HTN or HTN (n=564)		Normal Blood Pressure (n=501)		
	Mean	STD	Mean	STD	*p*-value**
Age (years)	29.29	6.51	27.4	6.79	<0.0001
Women (%)	53.19%		21.96%		<0.0001
Systolic Blood Pressure (mmHg)*	126	11	108	7	<0.0001
Diastolic Blood Pressure (mmHg)*	82	9	69	7	<0.0001
Diabetes Mellitus (FPG) (%)	16.37%		5.41%		<0.0001
Body Mass Index (kg/m^2)	34.58	8.12	30.87	8.27	<0.0001
Sodium Intake (g/d)	2717	1158	2554	1099	0.0192
Potassium Intake (g/d)	2500	1043	2442	1070	0.3675
Sodium/Potassium Ratio	1.11	0.27	1.09	0.31	0.3140
Total Energy Intake (kcal/d)	2088	765	2003	749	0.0686
Field Center					
Arizona	38.48%		32.73%		
Oklahoma	34.40%		29.34%		
South Dakota	27.13%		37.92%		

HTN, hypertension; pre-HTN, pre-hypertension; STD, standard deviation.

* From those without on hypertension medications.

** p-value from testing difference of means/rates between Pre-HTN/HTN and Normal blood pressure groups.

Table 2

Echocardiographic measures of study participants at the baseline and follow-up exams

	Baseline exam (Phase IV)					Follow-up exam (Phase V)					
	Pre-HTN or HTN		Normal Blood Pressure			Pre-HTN or HTN			Normal Blood Pressure		
Variable	**Mean**	**STD**	**Mean**	**STD**	***p****	**Mean (%)**	**STD**	***p****	**Mean (%)**	**STD**	***p****
Cardiac Geometry											
Left atrium diameter (cm)	3.74	0.44	3.48	0.43	<0.0001	3.85	0.47	<0.0001	3.61	0.48	<0.0001
Left ventricular diameter (cm) at diastole/height(m)	3.17	0.23	3.12	0.21	0.0047	3.27	0.25	<0.0001	3.21	0.24	<0.0001
LVmass index	78.60	14.23	71.54	13.46	<0.0001	79.67	15.84	0.0345	69.97	12.81	0.0007
Cardiac Functioning											
Heart Rate (beats/min)	69	12	65	10	<0.0001	69	12	0.5264	65	15	0.9677
EF (%)	59.65	4.96	60.59	4.94	0.0008	58.06	4.68	<0.0001	59.19	4.13	<0.0001
Stroke work (ml)	149.34	34.32	122.18	24.20	<0.0001	150.11	38.90	0.4984	126.72	30.65	0.0017
E-velocity (cm/s)	89.53	17.02	90.46	15.14	0.2116	83.32	17.36	<0.0001	83.20	14.98	<0.0001
A-velocity (cm/s)	59.36	15.39	54.81	13.43	<0.0001	61.42	16.31	0.0072	56.23	13.45	0.1437
E to A ratio	1.59	0.44	1.74	0.50	<0.0001	1.42	0.39	<0.0001	1.54	0.39	<0.0001
Deceleration time (ms)	172.43	36.11	166.07	37.15	0.0082	173.73	43.14	0.6330	165.60	41.09	0.8530
IVRT (ms)	78.80	10.44	76.01	10.53	0.0005	74.65	10.88	<0.0001	73.92	10.45	0.0006
Atrial filling fraction	0.27	0.08	0.24	0.07	<0.0001	0.35	0.34	<0.0001	0.33	0.29	<0.0001

HTN, hypertension; pre-HTN, pre-hypertension; STD, standard deviation.

*p-value from testing difference of means between Pre-HTN/HTN and Normal blood pressure groups.

**p-value from testing measure difference between follow-up and baseline exams.

Table 3

Changes of cardiac geometry and cardiac functioning measures after adjusting for age, gender, center, family relatedness and respective baseline echocardiographic measures in normotensive participants

Variable	Sodium			Potassium			Sodium/Potassium Ratio		
	Estimated Coeff.	StdErr	P	Estimated Coeff.	StdErr	P	Estimated Coeff.	StdErr	P
Cardiac Geometry									
Left atrium diameter (cm)	−0.000015	0.000012	0.1996	−0.000022	0.000012	0.0693	0.0215	0.0421	0.6104
Left ventricular diameter (cm) at diastole/height(m)	−0.000006	0.000007	0.3484	−0.000002	0.000007	0.7535	−0.0197	0.0242	0.4156
Left ventricular mass index	−0.000103	0.000376	0.7835	0.000189	0.000379	0.6178	−1.8375	1.3430	0.1720
Cardiac Functioning									
Heart Rate (beats/min)	−0.000225	0.000660	0.7332	−0.000035	0.000670	0.9587	−0.6536	2.3437	0.7805
Ejection Fraction (%)	−0.000072	0.000152	0.6348	−0.000180	0.000155	0.2462	0.6311	0.5451	0.2476
Stroke work (ml)	−0.000702	0.001186	0.5543	−0.000833	0.001207	0.4904	−2.0224	4.3474	0.6420
E-velocity (cm/s)	−0.000687	0.000619	0.2677	−0.001363	0.000624	**0.0294**	2.4487	2.2276	0.2723
A-velocity (cm/s)	0.000259	0.000526	0.6226	0.000148	0.000532	0.7815	0.1488	1.9013	0.9376
E to A ratio	−0.000018	0.000016	0.2513	−0.000026	0.000016	0.1055	0.0141	0.0571	0.8054
Deceleration time (ms)	−0.000715	0.001839	0.6977	−0.000443	0.001863	0.8119	−5.5109	6.6320	0.4065
Isovolumic relaxation time (ms)	0.000126	0.000467	0.7877	−0.000221	0.000475	0.6425	1.3776	1.6658	0.4087
Atrial filling fraction	0.000006	0.000013	0.6216	−0.000010	0.000013	0.4408	0.1147	0.0479	**0.0171**

Coeff., coefficient; P, p-value; StdErr, standard error.

Table 4

Changes of cardiac geometry and cardiac functioning measures after adjusting for age, gender, center, family and respective baseline echocardiographic measures in pre-hypertensive/hypertensive participants

Variable	Sodium			Potassium			Sodium/Potassium Ratio		
	Estimated Coeff.	StdErr	P	Estimated Coeff.	StdErr	P	Estimated Coeff.	StdErr	P
Cardiac Geometry									
Left atrium diameter (cm)	0.000006	0.000011	0.5799	0.000004	0.000012	0.7654	−0.0093	0.0481	0.8475
Left ventricular diameter (cm) at diastole/height(m)	0.000003	0.000006	0.6025	−0.000002	0.000007	0.8093	0.0345	0.0275	0.2098
Left ventricular mass index	0.000365	0.000431	0.3983	−0.000204	0.000470	0.6642	3.6782	1.8370	**0.0459**
Cardiac Functioning									
Heart Rate (beats/min)	0.000512	0.000435	0.2398	0.000432	0.000471	0.3592	0.0993	1.8570	0.9574
Ejection Fraction (%)	−0.000041	0.000155	0.7915	0.000009	0.000169	0.9557	−0.1008	0.6641	0.8795
Stroke work (ml)	0.000266	0.001301	0.8379	0.001277	0.001423	0.3700	−5.3119	5.5745	0.3412
E-velocity (cm/s)	0.001178	0.000605	0.0520	0.000394	0.000659	0.5503	4.7186	2.5557	0.0655
A-velocity (cm/s)	0.000747	0.000568	0.1893	0.000640	0.000618	0.3008	0.3644	2.4079	0.8798
E to A ratio	0.000005	0.000013	0.6986	−0.000001	0.000015	0.9247	0.0547	0.0570	0.3374
Deceleration time (ms)	−0.000900	0.001669	0.5898	−0.000468	0.001815	0.7965	−3.8228	7.2230	0.5969
Isovolumic relaxation time (ms)	−0.000168	0.000442	0.7040	−0.000030	0.000480	0.9497	−1.0752	1.8916	0.5700
Atrial filling fraction	0.000030	0.000014	**0.0338**	0.000024	0.000015	0.1237	0.0681	0.0615	0.2685

Coeff., coefficient; P, p-value; StdErr, standard error.