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Childhood and adult exposure to secondhand tobacco smoke and cardiac structure and function: results from Echo-SOL

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ABSTRACT

Objective To describe the relationship of household secondhand smoke (SHS) exposure and cardiac structure and function.

Methods Participants (n=1069; 68 % female; age 45–74 years) without history of tobacco use, coronary artery disease or severe valvular disease were included. Past childhood (starting at age <13 years), adolescent/adult and current exposure to household SHS was assessed. Survey linear regression analyses were used to model the relationship of SHS exposure and echocardiographic measures of cardiac structure and function, adjusting for covariates (age, sex, study site, alcohol use, physical activity and education).

Results SHS exposure in childhood only was associated with reduced E/A velocity ratio (β=−0.06 (SE 0.02), p=0.008). SHS exposure in adolescence/adult only was associated with increased left ventricular ejection fraction (LVEF) (1.2 (0.6), p=0.04), left atrial volume index (1.7 (0.8), p=0.04) and decreased isovolumic relaxation time (−0.003 (0.002), p=0.03). SHS exposure in childhood and adolescence/adult was associated with worse left ventricular global longitudinal strain (LVGLS) (two-chamber) (0.8 (0.4), p= 0.049). Compared with individuals who do not live with a tobacco smoker, individuals who currently live with at least one tobacco smoker had reduced LVEF (−1.4 (0.6), p=0.02), LVGLS (average) (0.9 (0.40), p=0.03), medial E′ velocity (−0.5 (0.2), p=0.01), E/A ratio (−0.09 (0.03), p=0.003) and right ventricular fractional area change (−0.02 (0.01), p=0.01) with increased isovolumic relaxation time (0.006 (0.003), p=0.04).

Conclusions Past and current household exposure to SHS was associated with abnormalities in cardiac systolic and diastolic function. Reducing household SHS exposure may be an opportunity for cardiac dysfunction prevention to reduce the risk of future clinical heart failure.

Key questions

What is already known about this subject?
► Household secondhand tobacco smoke exposure has implications for cardiopulmonary health across generations in a household. The association of secondhand tobacco smoke exposure with adverse changes in cardiac structure and function has not been described.

What does this study add?
► In this observational study of a population-based cohort of US Hispanics/Latinos, self-reported chronic exposure to household secondhand smoke in childhood and adolescence/adulthood was associated with worse left ventricular longitudinal strain. Individuals who reported living with one smoker who smokes in the home had worse left ventricular diastolic and systolic function.

How might this impact on clinical practice?
► Reducing household secondhand tobacco smoke exposure is an opportunity for prevention of cardiac dysfunction to reduce risk of possible future clinical heart failure.

INTRODUCTION

Worldwide, secondhand smoke (SHS) from tobacco accounts for 603 000 deaths, representing 1% of total worldwide mortality, with ischaemic heart disease representing the largest percentage of mortality.[1] While men are more likely to smoke tobacco, female non-smokers and children are more likely to be exposed to SHS compared with male non-smokers.[1] Epidemiological data on the health hazards of SHS exposure resulted in the enactment of public policy that restricted the use of tobacco products in public places including restaurants, bars and hotels.[2–7] Reducing exposure to SHS in public places has reduced the incidence of acute coronary syndromes throughout USA.[8–12] However, while much attention has been directed to reducing acute exposure to tobacco smoke in public places, less attention has been paid to chronic exposure to tobacco smoke in the household.

Hispanics/Latinos in the USA are a heterogeneous population with patterns of tobacco use that vary by national background. While Mexican-Americans tend to have lower prevalence of tobacco use compared with non-Hispanic whites, this trend is not uniform among all Hispanics/Latinos, such as Cubans and Puerto Ricans who report higher tobacco use.[13–15] Overall, 26% of men and 15% of women in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) were current tobacco smokers, while 32% of Puerto Rican women and 21% of Cuban women reported ongoing tobacco use. Additionally, household SHS was reported by 40% HCHS/SOL participants, a finding that has implications for cardiopulmonary health across generations in a household.[14 16] While SHS exposure has been steadily declining in the USA, the decline in SHS exposure among Hispanics/Latinos lags behind the observed decline in non-Hispanic whites.[17] The potential impact of chronic household SHS exposure at various points in the life course on cardiac structure and function has not been well described but has particular relevance for Hispanic/Latino populations who present with heart failure at an earlier age than non-Hispanic whites.[18] The objective of this study was to determine the association of past childhood/adult and current SHS exposure with cardiac structure and function as measured by echocardiography among Hispanics/Latinos.

METHODS

Study setting

HCHS/SOL is a population-based study of self-identified Hispanic/Latino men and women (N=16 415) aged 18–74 years.[19 20] HCHS/SOL was designed to assess chronic disease in Hispanic/Latino individuals living in four cities in the USA: Bronx, New York; Chicago, Illinois; Miami, Florida; and San Diego, California. The details of HCHS/SOL sampling and recruitment methods have been previously described.[19] Exclusion criteria included: active-duty military service, not living at the residential address, planning to move from the area within 6 months, unable to complete the study in English or Spanish or physically unable to attend the clinic examination.

Echo-SOL is an ancillary study of HCHS/SOL consisting of 1824 participants recruited through a stratified-sampling process representative of the parent study.[21] Echo-SOL was designed to characterise cardiac structure and function using echocardiography in a representative sample of Hispanic/Latino individuals living in the USA. The baseline Echo-SOL echocardiography examination was performed from 2011 to 2014.[21] Eligibility criteria for Echo-SOL included: age 45 years or older; self-reported Hispanic/Latino of Mexican, Puerto Rican, Cuban, Dominican, Central American or South American background; and enrolment 36 months or fewer from the date of the baseline HCHS/SOL visit. Echo-SOL enrolled on average ~80% of eligible participants. The Institutional Review Board at each study site

provided approval and oversight of all study materials and activities. All Echo-SOL participants gave informed consent.

Phillips IE-33 or Sonos 5500/7500 ultrasound imaging platforms were used in the acquisition of all echocardiographic imaging data. A standard transthoracic echocardiography examination was performed with the participant in the partial left decubitus position. Two-dimensional (2D), spectral, colour flow and tissue Doppler images were acquired in the parasternal long axis, short axis and apical four-chamber and two-chamber long-axis views. Left ventricular (LV) global longitudinal strain (GLS) analysis was performed using the vendor-independent Cardiac Performance Analysis software (TomTec, Hamden, Connecticute, USA) on acquired 2D images. All images were read by a Registered Diagnostic Cardiac Sonographer and over-read by a cardiologist (CJR) with level 3 advanced echocardiography training. Inter-reader and intrareader variability were assessed and determined to have a high degree of interclass correlation for each measurement (0.80–0.99).

Definitions

Chronic SHS exposure was assessed by questionnaire, which was available in both English and Spanish. SHS was characterised in the following manner:

1. *Any chronic exposure to household SHS:* any chronic exposure to household SHS was defined as answering yes to any of the following items: before age 13 years, have you ever lived with a regular smoker who smoked in the home?, or since age 13 years, have you ever lived with a regular smoker who smoked in the home?

2. *Only chronic childhood exposure to household SHS:* chronic childhood exposure to household SHS was defined as answering yes to following question: before age 13 years, have you ever lived with a regular smoker who smoked in the home?; excluding participants who answered yes to the question: since age 13, have you ever lived with a regular smoker who smoked in the home?

3. *Only chronic adolescent/adult exposure to household SHS:* chronic adolescent/adult exposure to household SHS was defined as answering yes to the following question: since age 13 years, have you ever lived with a regular smoker who smoked in the home?; excluding participants who answered yes to the question: before age 13 years, have you ever lived with a regular smoker who smoked in the home?

4. *Chronic childhood and adolescent/adult exposure to household SHS:* chronic childhood and adolescent/adult exposure to household SHS was defined by answering yes to both of the following two questions: (1) before age 13 years, have you ever lived with a regular smoker who smoked in the home, and (2) since age 13 years, have you ever lived with a regular smoker who smoked in the home?

5. *Current SHS exposure*
   a. Participants were asked to specify the number of individuals in the household who currently regularly

smoke tobacco in the home. This variable was parameterised into three categories: 0, 1 and 2+.

b. Participants were asked to specify the number of hours per week that they were in close contact with individuals actively smoking tobacco in any location (ie, house, work and car). This variable was parameterised into three categories: 0, 1 to the median and above the median.

The echocardiographic measurements included in this study included multiple measures of left and right heart structure and function: LV mass indexed to body surface area, LV end-diastolic volume, LV end-systolic volume, LV ejection fraction (LVEF), LV stroke volume, medial and lateral tissue Doppler E′ velocities, mitral inflow E/A ratio, E/E′ ratio, isovolumic relaxation time (IVRT), peak right ventricular/right atrial pressure gradient, tricuspid annular plane systolic excursion, right ventricular fractional area change, left atrial volume index, LV GLS (four-chamber view, two-chamber view and average).

Echo-SOL participant sociodemographic and lifestyle characteristics were obtained by questionnaires conducted during the HCHS/SOL baseline visit. National background was determined by self-report and classified as: Mexican, Puerto Rican, Cuban, Dominican, Central American or South American. Alcohol use and tobacco use were determined by self-report and characterised as current, former or never. Education was described by three categories: less than high school, high school or equivalent or greater than high school. Household income was categorised into four groups: <$20 000, $20 001–$40 000, $40 001–$75 000 or >$75 000. USA-born was defined as born in the 50 United States, excluding US territories. Physical activity was determined using the Global Physical Activity Questionnaire and was classified as low, moderate or high.

Statistical methods

For this cross-sectional analysis, we included only participants without coronary artery disease or severe aortic or mitral valve disease who report no prior or current tobacco smoking. Coronary artery disease was determined by self-report or pathological Q-waves on ECG consistent with prior myocardial infarction. Aortic and mitral valve disease was determined by echocardiography. The baseline characteristics of participants with and without self-reported chronic household exposure to SHS were compared. The corresponding distribution of all baseline sociodemographic and clinical characteristics was summarised for the overall population using means±SEs for continuous variables and proportions for categorical variables. The mean values of the echocardiographic outcome variables in participants with and without SHS exposure were compared using t-tests.

Multivariable linear regression analysis was conducted comparing echocardiographic measures of cardiac structure and function in individuals who reported any childhood or adolescent/adult exposure to SHS to individuals who did not report any exposure to SHS. Linear regression models adjusted for the following potential confounding

Table 1 Population characteristics according to household secondhand tobacco smoke exposure

	Any household secondhand tobacco smoke exposure (N=615)	No household secondhand tobacco smoke exposure (N=454)
Age, mean (SE)	55.5 (0.5)	56.9 (1.0)
Female sex, N (weighted %)	455 (68.4)	331 (67.7)
National background, N (weighted %)		
Dominican	151 (25.4)	78 (17.5)
Puerto Rican	92 (12.2)	71 (22.6)
Mexican	146 (20.3)	140 (23.0)
Cuban	113 (30.4)	51 (21.2)
Central American	57 (5.7)	68 (8.2)
South American	56 (6.0)	45 (7.5)
Low physical activity, N (weighted %)	327 (52.0)	232 (53.5)
Education level, N (weighted %)		
Less than high school	216 (32.2)	165 (30.9)
High school or equivalent	132 (21.7)	92 (15.1)
Greater than high school	266 (46.2)	196 (53.9)
Household income less than $20 000 per year, N (weighted %)	301 (52.8)	205 (46.5)
Not US mainland born, N (weighted %)	575 (92.0)	416 (93.4)

variables: model 1: age and sex; model 2: age, sex, study site, alcohol use, physical activity and years of education. Additionally, linear regression analyses were conducted to compare participants who have no reported chronic household SHS exposure to participants in each of the following categories of SHS exposure: childhood exposure only, adolescent/adult exposure only and both childhood and adolescent/adult exposure. Adjustment for potential confounding variables was conducted as specified above.

Current exposure to SHS was assessed in linear regression analyses comparing echocardiographic measures of cardiac structure and function in individuals who currently have no members of the household who smoke tobacco regularly with individuals who report 1 household smoker and/or 2+ household smokers. Additional linear regression analyses were conducted to assess the association between the number of hours per week spent in close contact with smokers in a closed space and echocardiographic measures of cardiac structure and function. Individuals who report 0 hours per week exposed with an active smoker in a closed space were compared with individuals who reported 1 hour to the median number of hours per week and to individuals who reported above the median number of hours per week. Adjustment for potential confounding variables was conducted as specified above. All analyses used sample weights to account for sampling probability and non-response and to make the estimates applicable to the target population based on guidelines suggested by the HCHS/SOL Steering and Data Analysis Committees. Statistical analyses were completed using SAS V.9.3.

Table 2 Association of any childhood or adolescent/adult household secondhand tobacco smoke exposure with echocardiographic measures of cardiac structure and function

	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values
LV mass index, gm/m^2	1.7 (1.9)	0.38	1.4 (1.8)	0.42
LV end-systolic volume, mL	−1.4 (1.0)	0.15	−1.2 (0.8)	0.14
LV end-diastolic volume, mL	−3.6 (2.0)	0.07	−3.2 (1.7)	0.05
LVEF, %	0.3 (0.4)	0.57	0.3 (0.4)	0.51
LV stroke volume, mL	0.2 (1.4)	0.86	0.5 (1.3)	0.71
LV longitudinal strain (four-chamber), %	0.1 (0.3)	0.84	0.02 (0.3)	0.94
LV longitudinal strain (two-chamber), %	0.9 (0.5)	0.07	0.8 (0.4)	0.06
LV longitudinal strain average), %	0.5 (0.3)	0.10	0.4 (0.3)	0.12
Medial E' velocity, cm/s	−0.2 (.1)	0.19	−0.1 (0.1)	0.31
Lateral E' velocity, cm/s	−0.2 (0.3)	0.50	−0.07 (0.2)	0.77
E/E'	0.1 (0.3)	0.67	0.1 (0.3)	0.63
E/A ratio	−0.02 (0.02)	0.43	−0.02 (0.02)	0.41
Isovolumic relaxation time, s	0.001 (0.002)	0.45	0.001 (0.001)	0.34
Peak RA/RV gradient, mm Hg	0.4 (0.6)	0.54	0.5 (0.5)	0.36
TAPSE, cm	−0.04 (0.05)	0.45	−0.02 (0.04)	0.57
RV fractional area change, %	−0.01 (0.01)	0.25	−0.007 (0.009)	0.39
Left atrial volume index, mL/m^2	0.6 (0.5)	0.24	0.7 (0.5)	0.13

Linear regression analyses adjusting for the following variables.
Minimally adjusted: age and sex.
Fully adjusted: age, sex, study site, alcohol use, physical activity and years of education.
E/A, E/A velocity; E/E', E/E'velocity ratio; LV, left ventricular; LVEF, left ventricular ejection fraction; RA, right atrial; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion.

RESULTS

A total of 1069 never smokers (68% women, mean age 56 years) were included in this analysis (table 1). Individuals who reported chronic household SHS exposure were disproportionately of Cuban and Dominican national background. Chronic household SHS exposure was also associated with decreased educational attainment and household income less than $20 000 per year. Individuals who reported any prior chronic childhood or adolescent/adult household SHS exposure did not differ in any echocardiographic measure of cardiac structure and function when compared with individuals who did not report any chronic exposure to household SHS (table 2). However, in assessing childhood and adolescent/adult exposures individually, exposure to household SHS smoke in childhood only was associated with decreased E/A ratio (−0.06 (SE 0.02), p=0.008), after adjusting for covariates (table 3). In the fully adjusted model, exposure to household SHS in adolescent/adulthood only was associated with increased LVEF (1.2% (0.6), p=0.04), increased left atrial volume index (1.8 (0.9), p=0.045) and decreased IVRT (−0.003 s (0.002), p=0.03). Individuals who reported household SHS exposure in both childhood and adolescence/adulthood had worse LV longitudinal strain (two-chamber) compared with individuals who reported no household SHS exposure (0.8% (0.4), p=0.049) after adjustment for covariates.

Current household secondhand tobacco smoke exposure as determined by the number of smokers who regularly smoke tobacco in the home was associated with worse left and right heart function (table 4). Individuals who report one household smoker had reduced LVEF (−1.4% (0.6), p=0.02), worse LV longitudinal strain (four-chamber and average) (1.1% (0.4), p=0.01% and 0.9% (0.4), p=0.03, respectively), reduced medial E' velocity (−0.5 m/s (0.20), p=0.01), reduced E/A ratio (−0.09 (0.03), p=0.003) and reduced right ventricular fractional area change (−0.02% (0.01), p=0.01). IVRT was increased in individuals who reported one household smoker (0.006 s (0.003), p=0.04). Individuals who reported two or more household smokers (N=31) had reduced E/E' ratio (−1.6 (0.6), p=0.0155). No association between the reported number of hours per week exposed to secondhand tobacco smoke and cardiac structure and function was observed (online supplementary table 1).

DISCUSSION

In a representative sample of Hispanics/Latinos, chronic exposure to SHS was associated with changes in cardiac structure along with systolic and diastolic functions. Chronic SHS exposure in childhood only was associated with worse LV diastolic function, while chronic SHS

Table 3 Univariable and multivariable linear regression analyses of the association of childhood and adolescent/adult chronic household secondhand smoke exposure with echocardiographic measures of cardiac structure and function in The Echocardiographic Study of Latinos (Echo-SOL)

	Childhood only (N=215)				Adolescent/adult only (N=86)				Childhood and adolescent/adult (N=314)			
	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values
LV mass index, g/m²	1.1 (2.0)	0.58	0.7 (2.0)	0.73	−1.0 (2.3)	0.65	−0.8 (2.3)	0.73	1.7 (1.8)	0.35	1.5 (1.7)	0.38
LV end-systolic volume, mL	−1.1 (0.9)	0.19	−1.3 (0.8)	0.12	−2.0 (1.1)	0.07	−1.6 (1.1)	0.16	−0.4 (0.9)	0.67	−0.2 (0.8)	0.81
LV end-diastolic volume, mL	−3.5 (1.8)	0.053	−3.5 (1.8)	0.059	−3.4 (2.4)	0.15	−2.8 (2.3)	0.23	−1.3 (2.0)	0.51	−0.9 (1.7)	0.59
LVEF, %	−0.3 (0.5)	0.64	0.008 (0.5)	0.99	1.4 (0.6)	0.02	1.2 (0.6)	0.04	−0.5 (0.5)	0.91	−0.06 (0.4)	0.89
LV stroke volume, mL	−1.3 (1.5)	0.41	−0.6 (1.5)	0.7	−1.8 (2.1)	0.38	−2.3 (2.0)	0.25	1.4 (1.7)	0.42	1.5 (1.5)	0.35
LV longitudinal strain (four-chamber), %	0.6 (0.4)	0.09	0.5 (0.4)	0.15	−0.8 (0.6)	0.19	−0.7 (0.6)	0.25	−0.2 (0.4)	0.97	−0.06 (0.3)	0.84
LV longitudinal strain (two chamber), %	0.5 (0.4)	0.25	0.3 (0.4)	0.38	−0.08 (0.7)	0.90	−0.3 (0.7)	0.73	0.8 (0.4)	0.06	0.8 (0.4)	**0.049**
LV longitudinal strain (average), %	0.6 (0.3)	0.09	0.5 (0.3)	0.16	−0.4 (0.6)	0.5	−0.4 (0.6)	0.50	0.5 (0.3)	0.15	0.4 (0.3)	0.18
Medial E' velocity, cm/s	−0.2 (0.2)	0.24	−0.08 (0.2)	0.58	−0.03 (0.2)	0.91	0.0003 (0.2)	0.999	−0.1 (0.2)	0.48	−0.1 (0.1)	0.43
Lateral E' velocity, cm/s	−0.2 (0.3)	0.45	−0.1 (0.3)	0.65	0.3 (0.4)	0.43	0.5 (0.4)	0.21	−0.2 (0.2)	0.43	−0.2 (0.2)	0.48
E/E'	−0.2 (0.3)	0.38	−0.3 (0.3)	0.29	0.07 (0.4)	0.86	−0.009 (0.4)	0.98	0.3 (0.3)	0.26	0.4 (0.3)	0.14
E/A ratio	−0.07 (0.02)	**0.002**	−0.06 (0.02)	**0.008**	0.01 (0.04)	0.70	0.01 (0.04)	0.75	0.02 (0.02)	0.45	0.02 (0.02)	0.47
Isovolumic relaxation time, s	0.002 (0.001)	0.08	0.002 (0.001)	0.15	−0.003 (0.002)	0.06	−0.003 (0.002)	**0.03**	0.001 (0.002)	0.56	0.001 (0.001)	0.34
Peak RA/RV gradient, mm Hg	0.4 (0.7)	0.57	0.5 (0.7)	0.46	0.02 (0.8)	0.98	0.3 (0.8)	0.69	0.2 (0.6)	0.78	0.2 (0.6)	0.7
TAPSE, cm	−0.04 (0.04)	0.37	−0.03 (0.04)	0.55	0.05 (0.08)	0.49	0.04 (0.08)	0.57	−0.04 (0.05)	0.43	−0.03 (0.05)	0.53
RV fractional area change, %	−0.02 (0.01)	0.09	−0.02 (0.01)	0.095	−0.01 (0.03)	0.68	−0.004 (0.03)	0.88	−0.001 (0.01)	0.93	0.003 (0.009)	0.70
Left atrial volume index, mL/m²	−0.7 (0.6)	0.23	−0.4 (0.6)	0.48	1.8 (0.9)	**0.045**	1.7 (0.8)	**0.04**	0.5 (0.7)	0.41	0.5 (0.6)	0.40

Linear regression analyses adjusting for the following variables.
Minimally adjusted: age and sex.
Fully adjusted: age, sex, study site, alcohol use, physical activity and years of education.
Bold denotes p value <0.05.
Reference: participants who report no childhood or adolescent/adult chronic household exposure to secondhand tobacco smoke.
LV, left ventricular; LVEF, left ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion.

Table 4 Association of current number of household smokers that regularly smoke in the home and echocardiographic measures of cardiac structure and function in Echo-SOL

	1 Household smoker (N=123)				2+ Household smokers (N=31)			
	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values	Minimally adjusted β (SE)	P values	Fully adjusted β (SE)	P values
LV mass index, gm/m2	5.3 (3.1)	0.09	4.6 (3.2)	0.15	−3.5 (5.1)	0.50	−3.0 (4.7)	0.52
LV end-systolic volume, mL	1.6 (1.3)	0.23	1.7 (1.3)	0.18	3.2 (2.2)	0.14	3.3 (2.1)	0.12
LV end-diastolic volume, mL	0.5 (2.8)	0.87	1.4 (2.7)	0.59	3.7 (4.4)	0.41	4.1 (4.4)	0.35
LVEF, %	−1.8 (0.6)	**0.003**	−1.4 (0.6)	**0.02**	−1.8 (1.0)	0.06	−1.7 (1.0)	0.07
LV stroke volume, mL	0.5 (2.2)	0.82	2.0 (2.2)	0.37	−1.4 (3.8)	0.72	−0.7 (3.7)	0.84
LV longitudinal strain (four-chamber), %	1.4 (0.4)	**0.002**	1.1 (0.4)	**0.01**	−1.3 (1.2)	0.27	−1.5 (1.3)	0.26
LV longitudinal strain (two-chamber), %	0.9 (0.5)	0.08	0.6 (0.5)	0.20	−1.7 (1.5)	0.27	−1.5 (1.3)	0.26
LV longitudinal strain (average), %	1.2 (0.4)	**0.006**	0.9 (0.4)	**0.03**	−1.5 (1.3)	0.25	−1.5 (1.2)	0.23
Medial E' velocity, cm/s	−0.7 (0.2)	**0.0004**	−0.5 (0.2)	**0.01**	0.7 (0.4)	0.11	0.9 (0.5)	0.0599
Lateral E' velocity, cm/s	−0.7 (0.4)	0.06	−0.5 (0.4)	0.18	0.5 (0.8)	0.51	0.8 (0.8)	0.36
E/E'	0.006 (0.4)	0.99	−0.05 (0.4)	0.88	−1.4 (0.6)	**0.026**	−1.6 (0.6)	**0.016**
E/A ratio	−0.1 (0.03)	**0.005**	−0.09 (0.03)	**0.003**	0.02 (0.06)	0.74	0.03 (0.06)	0.68
Isovolumic relaxation time, s	0.007 (0.003)	**0.02**	0.006 (0.003)	**0.04**	−0.002 (0.007)	0.76	−0.002 (0.006)	0.69
Peak RA/RV gradient, mm Hg	−0.6 (0.9)	0.54	−0.21 (0.92)	0.82	−2.1 (2.0)	0.31	−2.1 (2.1)	0.32
TAPSE, cm	−0.04 (0.06)	0.5	−0.01 (0.06)	0.85	−0.02 (0.2)	0.92	−0.03 (0.1)	0.84
RV fractional area change, %	−0.03 (0.01)	**0.002**	−0.02 (0.01)	**0.01**	0.01 (0.03)	0.71	0.006 (0.03)	0.83
Left atrial volume index, mL/m^2	−1.5 (0.8)	0.06	−0.9 (0.8)	0.24	−1.9 (1.4)	0.19	−1.7 (1.2)	0.16

Linear regression analyses adjusting for the following variables.
Minimally adjusted: age and sex.
Fully adjusted: age, sex, study site, alcohol use, physical activity and years of education.
Bold denotes p value <0.05.
Reference: participants who currently do not live with a tobacco smoker who regularly smokes in the home.
LV, left ventricular; LVEF, left ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion.

exposure in adolescence/adulthood only was associated with increased left atrial size and shorter IVRT signalling reduced LV compliance causing a rapid increase in LV intracardiac pressures with early cessation of LV filling. Chronic SHS exposure in adolescence/adulthood was also paradoxically increased LVEF possibly as a means of compensation to maintain cardiac output in the setting of abnormal diastolic function. Chronic SHS exposure in both childhood and adolescence/adulthood was associated with decreased myocardial deformation consistent with a pattern seen in early heart failure with preserved ejection fraction (HFpEF) where there is abnormal diastolic function, normal systolic function but abnormal myocardial deformation mechanics.[22] Current SHS exposure from living with a tobacco smoker was associated with decreased LV systolic function as measured by ejection fraction and global longitudinal strain.

To our knowledge, this study represents one of the first studies to examine the relationship of chronic SHS exposure with cardiac structure and function. The results of this study contribute to the emerging body of evidence regarding the adverse effects of exposure to tobacco smoke on cardiac structure and function, which has primarily been studied in active current smokers. Within Echo-SOL, increased pack-years of tobacco smoking was associated with decreased systolic and diastolic function and increased cigarettes

smoked per day were associated with increased LV mass.[23] A study of the Coronary Artery Risk Development in Young Adults (CARDIA) cohort found that tobacco smoking was associated with a trend towards increased LV mass in all groups except for African-American men and increased LV stress in women; however, the echocardiographic variables in this study were limited to linear measurements, and 2D quantitation was not performed.[24] In Multi-Ethnic Study of Atherosclerosis (MESA), a dose–response relationship between cigarette consumption measured in pack-years and regional LV dysfunction was noted.[25] Furthermore, a controlled experiment of the acute response to active tobacco smoking demonstrated immediate impairment in multiple measures of diastolic function.[26] However, the data from our study support that notion that even passive SHS is associated with alterations LV systolic function and diastolic function.

The mechanisms by which tobacco smoke exposure impacts myocardial function are not well described, although exposure to SHS has been associated with increased serum homocysteine and fibrinogen levels, which are associated with increased inflammation and thrombosis.[27] Experimental and clinical studies have shown the direct toxic effects of cigarette smoke on the myocardium, including abnormalities in energy metabolism, lipotoxicity and oxidative stress.[28 29] Additionally, animal models

suggest that chronic exposure to tobacco smoke increases blood pressure and oxidative stress and causes endothelial dysfunction.[30] Of note, in our study, current exposure to a household smoker was associated with more profound decreases in systolic and diastolic function than any other measure of chronic SHS smoke exposure. Yet, in the absence of current exposure to SHS, chronic SHS exposure in childhood was still associated with decrease diastolic function, thus highlighting the potential long-term impact of remote SHS exposure on cardiac function. In utero exposure to maternal and paternal smoking has been found to increase the risk of decreased cardiac function in children.[31–34] In our study, it is difficult to determine whether participants that report childhood exposure to SHS were also exposed in utero and thus separate the cardiac effects of in utero exposure from childhood SHS exposure. However, these findings are hypothesis generating and highlight the potential impact of childhood environmental exposures on future cardiac health in adulthood.

The observed relationship between SHS exposure and cardiac function has implications for public health strategies for primordial prevention of heart failure, particularly in Hispanics/Latinos. During 2011–2012, about 58 million non-smokers in the USA were exposed to SHS; with two out of every five children—including 7 out of every 10 black children—being exposed to SHS regularly.[17] Racial and ethnic differences in LV mass, diastolic function, systolic function and myocardial deformation are not completely explained by cardiometabolic risk factors and comorbidities.[35] Within Echo-SOL, the average values of LV size and mass are different than what has been reported in non-Hispanic white cohorts of healthy participants.[36] Additionally, LV diastolic dysfunction is more highly prevalent in Hispanics/Latinos than in cohorts of non-Hispanic whites.[37] Similar differences have been observed in reference values for global longitudinal strain in healthy African-Americans when compared with healthy whites in the CARDIA study.[38] The degree to which intervenable environmental exposures, such as household exposure to SHS, might contribute to racial/ethnic differences in cardiac function, and thus future heart failure risk, is an important consideration.[39]

This study has several limitations. First, SHS exposure was assessed by self-report and thus is subject to recall bias. Second, considerable heterogeneity in SHS exposure intensity for each measure of SHS likely exists. Serum cotinine levels were not measured to quantitatively assess current SHS exposure and assess for dose response. Third, the cross-sectional design of this observational study limits our ability to determine causality. We acknowledge that we did not account for multiple testing. However, most of our findings are at the p value less than the 0.01 level. SHS is conceptualised as a form of indoor air pollution,[40] placing hypertension, obesity and diabetes[41] on the causal pathway of our cardiac outcomes and thus justifying their exlusion fromour statistical models as confounders. Finally, the sample size of Echo-SOL is modest, limiting statistical power and type II error. Future studies of SHS and cardiac function with a larger sample and a longitudinal design are needed to better assess cardiac function and clinical heart failure outcomes.

CONCLUSION

In Hispanics/Latinos, chronic SHS smoke exposure in childhood was associated with decreased LV diastolic function. Living with a tobacco smoker who smokes in the home was associated with abnormal diastolic function, abnormal systolic function and abnormal myocardial deformation. While it is known that subclinical LV dysfunction increases risk for clinical heart failure,[42] the relationship between chronic SHS exposure and incident heart failure warrants further investigation in future studies. Reducing childhood and adulthood exposure to SHS in the home is a potential opportunity for primordial prevention of heart failure, particularly among Hispanics/Latinos.

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