Socioeconomic disparities in smoking are partially explained by chronic financial stress: marginal structural model of older US adults

Abtin Parnia [ORCID],[1] Arjumand Siddiqi [ORCID] [1,2]

[1]Division of Epidemiology, Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
[2]Department of Health Behavior, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA

Correspondence to
Abtin Parnia, Division of Epidemiology, Dalla Lana School of Public Health, University of Toronto, Toronto, ON M5T 3M7, Canada;
abtin.parnia@mail.utoronto.ca

ABSTRACT

Background A persistent socioeconomic gradient in smoking has been observed in a variety of populations. While stress is hypothesised to play a mediating role, the extent of this mediation is unclear. We used marginal structural models (MSMs) to estimate the proportion of the effect of socioeconomic status (SES) on smoking, which can be explained by an indicator of stress related to SES, experiences of chronic financial stress.

Methods Using the Health and Retirement Study (waves 7–12, 2004–2014), a survey of older adults in the USA, we analysed a total sample of 15 260 people. A latent variable corresponding to adult SES was created using several indicators of socioeconomic position (wealth, income, education, occupation and labour force status). The main analysis was adjusted for other factors that influence the pathway from adult SES to stress and smoking, including personal coping resources, health-related factors, early-life SES indicators and other demographic variables to estimate the proportion of the effect explained by these pathways.

Results Compared with those in the top SES quartile, those in the bottom quartile were more than four times as likely to be current smokers (rate ratio 4.37, 95% CI 3.35 to 5.68). The estimate for the MSM attenuated the effect size to 3.34 (95% CI 2.47 to 4.52). Chronic financial stress explained 30.4% of the association between adult SES and current smoking (95% CI 13 to 48).

Conclusion While chronic financial stress accounts for part of the socioeconomic gradient in smoking, much remains unexplained.

INTRODUCTION

Socioeconomic disparities in cigarette smoking are persisting despite decades of interventions.[1 2] According to a recent review, smoking specifically contributes the greatest portion of social inequalities in health.[3] Socioeconomic status (SES) affects smoking through multiple mechanisms, but tobacco control interventions have predominately focused on a limited set of determinants.[4] These interventions often target modification of personal orientations to smoking, such as motivation and knowledge[5] or modification of exposures to smoking cues through smoking bans and advertising.[6 7] However, a set of empirical findings suggest that socioeconomic smoking disparities are also a function of socioeconomic differences in the psychological stress and strain of everyday life.[8] Thus, a potential consequence of relying on

traditional smoking interventions is that they will be insufficient for reducing socioeconomic smoking disparities because they neglect the role of stress in producing smoking behaviours.[1 9]

To date, the literature supports the prominent role of stress,[10 11] but the extent of mediation is not known. The complex causal relationships (multiple interconnected mediators and confounders) that describe the association between SES and smoking limit the use of common methods of mediation analysis.[12] For example, the literature suggests that, in addition to stress, factors such as social support and sense of control may be operating as both confounders and mediators of stress.[5] Due to this complexity, one of the assumptions of traditional mediation analysis—no confounding between mediator and outcome associated with exposure—is often unjustifiable.[12] For similar reasons, structural equation modelling (SEM), which has been commonly used to investigate this question,[13–15] carries restrictive assumptions regarding model specification and confounding that may be unjustifiable to study the link between SES and health behaviours with survey data.[16] For instance, in a study, Martinez *et al* used the National Health Interviews Survey to explore several mechanisms between SES and smoking, including social cohesion, sleep disturbance, psychological distress and financial strain.[17] As such, in the presence of confounding in the pathway between mediator and outcome associated with exposure (eg, social cohesion acting as a confounder of financial strain and smoking), or exposure–mediator interaction, the use of traditional mediation models and SEM can result in biassed estimates by inappropriately adjusting for the other pathways.[18] Using other methods of mediation analysis can reduce some of these biases.

Building on the literature, this study attempts to answer the question of mediation by stress in the SES–smoking association using a marginal structural model (MSM), which is better able to manage these biases as it relaxes assumptions regarding the lack of association between exposure and the confounders of the mediator–outcome relationship.[12 19] In this study, we applied MSM to a longitudinal, nationally representative sample of older adults in the USA to examine how much of the association between SES and smoking is mediated by stress. More specifically, we measured the proportion of the association of SES with smoking, which can be explained by an indicator of stress related to SES, experiences of chronic financial stress.

METHODS

Study populations

The Health and Retirement Study, a biennial, nationally representative longitudinal study of older adults in the USA, was used.[20] Our sample consisted of individuals present in wave 7 (2004: baseline), as psychosocial variables were collected starting in the following wave. The psychosocial questionnaire, containing the key mediating variables of interest, was delivered to half of the sample in wave 8 and the other half in wave 9 following the same pattern of half samples for subsequent waves (ie, wave 10 for the first half sample and wave 11 for the second half sample). We included all participants who were eligible for any of the psychosocial questionnaires (excluding wave 9, as key variables were not included in that year). Based on these specifications, the total sample consisted of 15 260 people who participated in wave 7 and at least one other wave, up to wave 12 (2004–2014). We used all prescribed weighting technique for complex survey design in addition to correction for missing values in the psychosocial questionnaire.[20 21]

Measures

Outcome variable

We examined current smoking (dichotomised yes vs no) as the key outcome variable, measured in all study waves.

Main exposure

The main exposure variable was SES, as conceptualised by Nandi *et al*,[22] which considered both early-life and adult socioeconomic circumstances.[22] These latent factors were created using maximum-likelihood confirmatory factor analysis. The adult SES factor was constructed with the following variables: household wealth and income, education, longest occupation and labour force status at baseline. The early-life SES factor was constructed based on maternal and paternal education, paternal occupation, living in the south of the USA and living in rural areas. The early-life variable was used as a baseline adjustment for the models. The factor scores were then turned into quartiles. To reduce bias due to missing observations related to SES, we used multiple imputation in calculating factor scores based on the other variables in the model. We also explored factor analysis with full-information maximum likelihood estimation as an alternative strategy. Model performances for both methods were similar and are reported in online supplementary appendix A.

Main mediator

Stress was measured using a series of questions related to 'experiences of chronic stress'.[21] Participants were asked to identify problems that were current and lasted 12 months or longer. We used two questions related to SES, general financial strain and housing problems. Both financial strain and housing problems have been linked to smoking in prior studies.[23 24] The responses were dichotomised and recoded as yes if participants answered 'yes' to either of the questions in the measurement wave. As this measure was repeated every second wave, in waves 8–12 (though not available for wave 9), the half sample of participants who were eligible for wave 8 had two additional observations of stress, at waves 10 and 12.

Mediator outcome confounders

Smoking behaviour is influenced by personal coping resources and, in turn, these resources are patterned by SES.[25] In this analysis, we used two common personal coping resources, social support and sense of control.[26]

Several studies have emphasised the role of social support in mediating the relationship between SES and smoking, and between stress and smoking.[17 25 27] Two indices of social support were used to measure the perceived quality of social support, both negative and positive. Each of these measures detailed relationship with spouse, children, other family or friends.[28] The responses were averaged across the relationships, creating two indices of positive and negative social support. The construct of sense of control is a key psychological determinant of SES disparities in health.[29] In this analysis, two measures of control were used, perceived constraints on personal control and perceived sense of mastery, both measured by five items and averaged across the items.

Exposure outcome confounders

Being diagnosed with a condition is likely to be associated with subsequent health behaviours and thus acts as a confounder of the SES–smoking relationship.[30 31] The change in health condition variables was physician diagnosis of high blood pressure, cancer, lung disease, heart problem, stroke, psychiatric condition or diabetes since the last wave of measurement. Self-rated health and drinking frequency were also included due to their close association with smoking and SES.[32 33]

Statistical analyses

Counts, weighted percentages, weighted proportion of current smokers and missing percentage of the variables were calculated. Log-linear regression models were used to examine both segments of the pathway: the path between SES to stress, and stress and other psychosocial variables to smoking. Next, we fitted a series of models using pooled log-linear regressions (with clustered standard errors within each subject for multiple measurements) for the mediation of stress in the pathway between SES-smoking. Model 1 estimates total effect of SES on current smoking; controlling for baseline covariates include early-life SES, race, sex and age. Models 2 and 3 added the mediator and the covariates, respectively. An additional model was also fitted to check for the presence of exposure–mediator interaction (adult SES interaction with stress). Since the mediation between exposure–mediator was not significant at p<0.1, it was not included in the analysis (online supplementary table S2). Model 3 is estimated with equation 1:

$$log[P(Y_{it} = 1 | A_i = a, M_{it} = m, C_i = c, R_{it-1} = r, S_{it} = s)]$$
$$= \beta_0 + \beta_1 A_i + \beta_2 M_{it} + \beta_3 C_i + \beta_4 R_{it-1} + \beta_5 S_{it} \quad (1)$$

In the equation 1, Y represents outcome, current smoking for individual i at time t. Symbol A represents adult SES at the baseline for individual i. The mediator, chronic financial stress, is represented by M at time t, as we hypothesise that the influence of stress on current smoking may be fairly immediate. The baseline covariates are represented by C. The time-varying confounders are health-related factors lagged by a wave (time $t-1$) and coping resources measured at time t, represented by R_{it-1} and S_{it}, respectively. To ensure temporal precedence, health-related factors were lagged. An illustration of these relationships can be found in figure 1.

As discussed, the association of SES with the confounders of the stress–smoking relationship (ie, personal coping resources and health-related factors) invalidates the assumptions of traditional methods of mediation analysis. Thus, equation 1 provides a biased estimate of the controlled direct effect (CDE). Model 4 is the MSM with inverse-probability weights. The details of this method are described in online supplementary appendix A and are reported in the literature.[19 34] Briefly, this method reweights
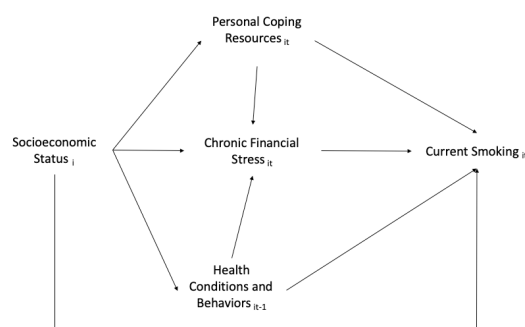
Figure 1 Illustration of the conceptual relationships between the variables in the study and the time of measurement.

the study population to a pseudo-population, based on the likelihood of each participant receiving their observed exposure and mediator values, conditional on the predictors of exposure and mediator (ie, the predicted probabilities of a model for the exposure and a model for the mediator). As a result, a counterfactual comparison is generated to estimate the association in the absence of the effect of mediator. The inverse-probability weights—stabilised as recommended by Cole and Hernan[35]—was constructed for the stress and the adult SES measures to be used in the reweighting required for MSM.[35] Model 4 is estimated using equation 2:

$$log\left[P\left(Y_{it} = 1 \mid A_i = a, M_{it} = m\right)\right] = \beta_0 + \beta_1 A_i + \beta_2 M_{it} \quad (2)$$

In this equation, the CDE is estimated by the value of β_1. Following the MSM, we calculated the proportion of the effect explained by the stress pathway. This measure is calculated by equation 3, described in VanderWeele.[36]

$$Proportion\ Eliminated = \frac{(RR(Total\ Effect) - RR(CDE\ (m)))}{RR(Total\ Effect) - 1} \quad (3)$$

We calculated the proportion eliminated for the difference between each quantile of income and used bootstrapping method with 1500 resampling to calculate the 95th percentile CIs. The analysis was done using R and STATA 14.2.

Sensitivity analyses

Sensitivity analysis was conducted to explore the various assumptions on mediating and moderating relationships, measurement error and unmeasured confounding. We explored the role of the coping resources variables, as the personal coping variables can act as mediators or moderators of the relationship between stress and smoking. By including interaction terms for the coping resources with SES and with stress, the role of these factors in moderating the SES–smoking and SES–stress relationships was tested. Furthermore, social support and sense of control have mediating roles in association between SES and smoking.[29 37] Hence, models were created to examine the role of each mediators independently as well as the three mediators combined together.

The effect of missing observations of the inference of the study was tested by including a missing category for stress. Lastly, as this inference is based on an observational study, there is a likelihood of unmeasured confounding that can affect the results. To quantify the likely impact of this problem, we have calculated E values, as proposed by VanderWeele and Ding,[38] to measure the

minimum strength of association for a confounder that can shift the estimate to null effect.[38]

RESULTS

Table 1 details the distribution of the variables in the study. To capture the entire sample, the first measurement for time-varying variables is used. Online supplementary tables S1.1–S1.4 are the observations in the sample by each wave. The weighted prevalence of smoking in the overall sample was 13.47% (95% CI 12.5% to 14.5%). This prevalence is lower than 18.1% (95% CI 17.4% to 18.7%), the national current smoking prevalence in 2004 (as the sample is weighted to 2004 population) for US adults above 45 years old.[39] The missing observations in most variables is below 5%, with the exception of psychosocial variables, where missingness is closer to 18%.

Tables 2 and 3 describe the associations between SES and psychosocial variables, and these variables and smoking, respectively. As expected, compared with study participants in the top quartile of adult SES (most advantageous) category, those in the bottom quartile of SES (least advantageous) had 2.29 times (95% CI 2.09 to 2.50) higher likelihood of having experienced chronic financial stress. Moreover, in table 3, the likelihood of being a current smoker is 36% (95% CI 22% to 52%) higher in those who experienced chronic financial stress compared with those who did not.

Table 4 details the results of the mediation models. Model 1 estimates that those in the top quartile of adulthood SES compared with the bottom quartile of adulthood SES have 4.37 (95% CI 3.35 to 5.68) times higher likelihood of being current smokers, with decreasing magnitude for other quartiles. Models 2 and 3 demonstrate an attenuation of the effect size with the addition of stress and other covariates in the model. In model 3, the effect of experience of chronic financial stress is 1.13 (95% CI 1.01 to 1.26), which is attenuated compared with model 2. The CDE for the comparison between the top quartile of adulthood SES and the bottom quartile is 3.34 (95% CI 2.47 to 4.52). Overall, for various categories of adulthood SES, around one-third of the association between adulthood SES and current smoking was explained by the chronic financial stress pathway.

Results of sensitivity analysis indicates lack of moderation by personal coping resources. The exploration of multiple mediation pathways shows that the combined use of all psychosocial variables results in the largest attenuation of the effect. However, when exploring pathways independently, the largest estimate is for the financial chronic stress mediation pathway. The results of the sensitivity analysis are reported in online supplementary tables S4–S7.

To account for the likelihood of unmeasured confounders, we calculated E-values for our total effect measurements and measurements of CDE. The full results are provided in online supplementary table S8. The calculations suggest that the minimum strength of an unmeasured confounder for the comparison of the bottom to top quartiles of adulthood SES to be null is 6.1 (4.4 to 8.5 for the 95% CI of the estimate).

DISCUSSION

This study examined the mediation of chronic financial stress in the association between SES and smoking. By addressing major biases produced in previous studies, which used more traditional mediation analysis techniques, this study builds on the literature that explains how health behaviours are shaped.

Table 1 Descriptive statistics of the variables, N=15 260

Variables	Level	Count	Weighted %	Weighted %, smokers	95% CI
Sex	Male	6268	45.26	15.04	13.82 to 16.26
	Female	8992	54.74	12.04	10.97 to 13.12
Race	White	12 327	83.8	12.57	11.42 to 13.72
	Black	2136	10.67	17.96	15.78 to 20.14
	Other	796	5.53	17.09	12.93 to 21.24
Early-life SES	<25th	3471	20.11	15.67	14.11 to 17.23
	25th–50th	3638	21.95	13.61	11.58 to 15.64
	50th–75th	3961	26.63	13.64	12.23 to 15.06
	<75th	4190	31.31	11.58	10.05 to 13.11
Adulthood SES	<25th	3353	19.6	19.18	17.49 to 20.86
	25th–50th	3704	22.99	16.08	14.17 to 17.99
	50th–5th	3978	25.79	13.43	11.61 to 15.25
	<75th	4225	31.63	7.84	6.71 to 8.98
Age	<55	2691	21.7	20.15	18.19 to 22.27
	55–64	4748	38.55	16.17	14.62 to 17.84
	65–75	4930	23.52	9.451	8.317 to 10.72
	75+	2891	16.23	3.87	3.048 to 4.902
Self-rated health	1. Excellent	1485	10.74	6.93	5.32 to 8.54
	2. Very good	4448	30.35	10.32	8.83 to 11.81
	3. Good	4795	30.59	13.66	12.57 to 14.74
	4. Fair	3245	20.11	18.29	16.12 to 20.47
	5. Poor	1267	8.12	20.3	17.47 to 23.12
	Missing	20	0.1		
Change in conditions					
Diabetes	0=no	14 806	96.96	13.5	12.44 to 14.56
	1=yes	417	2.81	10.69	6.77 to 14.60
	Missing	37	0.23		
Cancer	0=no	14 858	97.57	13.44	12.40 to 14.47
	1=yes	360	2.2	11.3	7.27 to 15.32
	Missing	42	0.24		
Lung disease	0=no	14 973	98.25	13.14	12.11 to 14.17
	1=yes	253	1.59	30.47	23.69 to 37.25
	Missing	34	0.16		
Heart disease	0=no	14 691	96.39	13.48	12.43 to 14.52
	1=yes	544	3.45	11.02	7.58 to 14.46
	Missing	25	0.16		
Stroke	0=no	14 977	98.36	13.43	12.43 to 14.43
	1=yes	266	1.56	11.31	6.83 to 15.79
	Missing	17	0.08		
Psychiatric condition	0=no	14 891	97.61	13.33	12.34 to 14.33
	1=yes	337	2.21	16.24	10.55 to 21.92
	Missing	32	0.17		
Number of drinks in a day	0 or none	10 218	64.23	12.56	11.44 to 13.68
	1 or 2	3982	27.32	9.67	8.52 to 10.81
	3 or 4	749	5.79	28.61	23.90 to 33.33
	5 or more	272	2.34	38.12	31.27 to 44.98
	Missing	39	0.31		
Chronic stress	0=no	7412	46.87	10.02	9.16 to 10.95
	1=yes	4888	33.48	16.72	15.03 to 18.56
	Missing	2960	19.65	16.14	14.34 to 18.12
Social support, positive	Below 50th	6412	43.21	14.58	13.10 to 16.06
	Above 50th	6318	39.65	10.78	9.62 to 11.95

Table 1 Continued

Variables	Level	Count	Weighted %	Weighted %, smokers	95% CI
	Missing	2530	17.14	16.47	14.44 to 18.50
Social support, negative	Below 50th	6799	42.97	11.07	9.88 to 12.26
	Above 50th	5937	39.92	14.6	13.19 to 16.01
	Missing	2524	17.11	16.44	14.42 to 18.47
Sense of control, mastery	Below 50th	7169	46.43	13.17	11.74 to 14.59
	Above 50th	5385	35.31	12.33	11.12 to 13.54
	Missing	2706	18.26	16.05	14.13 to 17.97
Sense of control, constraints	Below 50th	7005	46.27	10.98	9.84 to 12.11
	Above 50th	5536	35.41	15.23	13.63 to 16.83
	Missing	2719	18.31	15.98	14.07 to 17.89

Based on a nationally representative sample of older adults in the USA, our study estimates that one-third of the socio-economic disparity in current smoking is explained by underlying differences in the experience of chronic financial stress. Notably, our results also suggest that the majority of the association between adult SES and current smoking remains unexplained by our model, which included most of the variables that have been implicated in prior literature.

Other studies that have explored the role of financial stress in various populations show strong effect on smoking as well. Most studies have not included similar mediation analysis and therefore are not directly comparable. Some studies have stratified their samples by income and education or have only sampled low SES population. For instance, Guillaumier et al[40] demonstrate twice the odds of experiencing financial strain in low SES smokers compared with low SES non-smokers.[40] In a study by Grafova,[23] financial strain was investigated and found a higher likelihood of being smoker for the lowest income quartile compared with the highest (for men 17.66% vs 3.28% and for women 5.1% vs 3.6%).[23] Lastly, Shaw et al also found interaction effects between financial strain and education, in the direction of higher smoking likelihood in low SES groups with financial strain.[41] Based on these studies, our findings are in line with the strong effect of chronic financial stress in explaining smoking disparities by SES. Our results also converged with those of Martinez et al,[17] who used SEM to explore a similar question on the pathways that mediate SES effects on smoking.[17] Martinez et al[17] found that the four pathways (including financial strain) they examined accounted for one-third of the effect of SES on current smoking, while the remaining two-thirds of the effect of SES remains unexplained by the pathways studied.[17] When they specifically examined mediation by the financial strain pathway, they found the indirect effect of this pathway to be 26% of the total effect of SES on smoking. Both studies confirm the major role of financial strain in the mediation of SES and smoking association as well as the large unexplained effect in the differences in smoking rates.

Table 2 Association between adulthood SES, personal coping resources and financial chronic stress

	Chronic financial stress	
	Rate ratio	
Adulthood SES		
<25th quartile (most disadvantageous)	2.29***	1.88***
	(2.09 to 2.50)	(1.72 to 2.06)
25th–50th	1.91***	1.69***
	(1.76 to 2.07)	(1.57 to 1.83)
50th–75th	1.48***	1.39***
	(1.37 to 1.59)	(1.29 to 1.49)
>75th quartile (least disadvantageous) (ref)	1	1
Sense of Control Scale		
Mastery		0.94***
		(0.92 to 0.95)
Constraints		1.13***
		(1.11 to 1.15)
Social Support Scale		
Positive		0.91***
		(0.88 to 0.95)
Negative		1.30***
		(1.25 to 1.36)

Models are adjusted for baseline covariates: age, sex, early-life SES and race.
Rate ratio; 95% CI in parentheses.
Person-wave=19 918; N=11 778.
*P<0.05, **P<0.01, ***P<0.001.
ref, reference; SES, socioeconomic status.

Table 3 The association between psychosocial factors and current smoking

	Likelihood of current smoking			
	Rate ratio			
Chronic financial stress				
No (ref)	1			
Yes	1.36***			1.28***
	(1.22 to 1.52)			(1.14 to 1.44)
Sense of Control Scale				
Mastery		1.02		1.03
		(0.97 to 1.07)		(0.98 to 1.09)
Constraints		1.16***		1.14***
		(1.11 to 1.21)		(1.09 to 1.19)
Social Support Scale				
Positive			0.87*	0.93
			(0.78 to 0.98)	(0.83 to 1.04)
Negative			1.01	0.91
			(0.89 to 1.15)	(0.80 to 1.04)

Person-wave=19 918; N=11 778.
*P<0.05, **P<0.01, ***P<0.001.
ref, reference.

Table 4 Rate ratio of smoking and mediation by chronic financial stress (N=11 778/person-wave=19 918)

Models	1	2	3	4	Proportion explained
Adult SES					
<25th quartile (most disadvantageous)	4.366***	4.103***	3.178***	3.343***	0.304***
	(3.354 to 5.683)	(3.150 to 5.343)	(2.428 to 4.160)	(2.472 to 4.520)	(0.132 to 0.476)
25th–50th	3.091***	2.944***	2.529***	2.324***	0.367***
	(2.443 to 3.911)	(2.328 to 3.723)	(1.991 to 3.213)	(1.736 to 3.109)	(0.204 to 0.529)
50th–75th	2.115***	2.055***	1.856***	1.796***	0.286*
	(1.705 to 2.623)	(1.658 to 2.548)	(1.503 to 2.293)	(1.360 to 2.373)	(0.051 to 0.520)
>75th quartile (least disadvantageous) (ref)	1	1	1	1	
Chronic stress					
No		1	1	1	
Yes		1.188**	1.126*	1.247**	
		(1.064 to 1.327)	(1.007 to 1.258)	(1.079 to 1.440)	
Sense of control					
Mastery			1.048		
			(0.997 to 1.102)		
Constraints			1.075**		
			(1.026 to 1.127)		
Social support					
Positive			0.886		
			(0.781 to 1.004)		
Negative			0.946		
			(0.851 to 1.052)		

Rate ratio; 95% CI in parentheses.
Model 1 is adjusted for baseline variables, namely, age, sex, race, early-life SES.
Model 2 is adjusted for baseline variables and chronic financial stress.
Model 3 is model 2 with additional adjustments for baseline variables and time-varying confounders.
Model 4 is a marginal structural model estimating the controlled direct effect using inverse probability weighting.
*P<0.05, **P<0.01, ***P<0.001.
ref, reference; SES, socioeconomic status.

There are several limitations to this study. As the population for this study is older adults in the USA, there is potential for selection bias that under-represents smokers and those of low SES who experience earlier mortality.[20] For instance, the proportion of deaths in the follow-up from 2004 to 2014 is twice as much in the smokers in the low SES category compared with smokers in the high SES category (weighted % of deaths among smokers, <25th percentile SES= %41.18, >75th percentile SES=%12.45). This type of selection bias can result in underestimation of the coefficients, so our study is likely a conservative estimate of the SES gradient in smoking. The large percentage of missing values in the coping resources and stress variables may also have had an impact on our results, as weighted proportion of missing from these variables is between 15% and 20% and may not be missing at random. However, our analysis investigating the impact of missing values in the mediator variable produced similar results, by using a missing category in the measurement of chronic stress (online supplementary table B3 in appendix B), suggesting that the effect size observed in this analysis is likely unaffected by the missingness in the main mediation variable. Furthermore, the application of the non-response adjustment factor to the survey weights reduces some of the potential biases.[21]

The strength of this study is in the use of MSM with inverse probability weighting for mediation analysis. As this method does not have the strict assumptions of SEM, and the potential bias resulted from the use of traditional mediation methods due to the association between exposure and the confounders of the mediator–outcome relationship.[12 18] The findings of our study suggest that chronic financial stress plays a large role in mediating the association between SES and smoking. It also suggests that SES shapes our behaviours through other means not associated with chronic financial stress.

The basis of the variables selected in this study is described in detail in online supplementary appendix A. Briefly, the conceptual framework of Harwood et al[25] includes most of the variables included in this study.[25] Hiscock et al[2] discuss the possibility of other mechanism underlying SES disparities in smoking.[2] These factors include health literacy, access to cessation resources, peer influence or tobacco company marketing. These pathways were unaccounted for in this study due to limitations of the survey used, so it is possible that they accounted for the remaining (unexplained) effects of adult SES on smoking yielded by our analyses. However, other studies that have explored the role of health literacy pathway or factors associated with cessation resources suggest these factors may only explain a small amount of the association.[2 42] Future research can further explore the large unexplained portion of the link between SES and smoking.

Original research

What is already known on this subject

► Cigarette smoking rates are much higher in those with disadvantageous socioeconomic conditions. Stress likely contributes to these differences, but the extent of this contribution is not clear. There have been studies on this topic, but they have either not used robust methods or omitted important factors.

What this study adds

► By using robust statistical methods and accounting for variables related to stress, this study estimated that approximately one-third of socioeconomic disparities in smoking for older adults can be explained by the experience of financial stress. Knowing how much financial stress influences the disparities in smoking rates can help public health interventions assess their priorities for reducing health inequities.

ORCID iDs
Abtin Parnia http://orcid.org/0000-0003-4321-3260
Arjumand Siddiqi http://orcid.org/0000-0002-2458-9011

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