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REGULAR ARTICLE

Early-Life Adversities and Recalcitrant Smoking in Midlife: An Examination of Gender and Life-Course Pathways

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Abstract

Background Little is known about life-course factors that explain why some individuals continue smoking despite having smoking-related diseases.

Purpose We examined (a) the extent to which early-life adversities are associated with the risk of recalcitrant smoking, (b) psychosocial factors that mediate the association, and (c) gender differences in the associations.

Methods Data were from 4,932 respondents (53% women) who participated in the first and follow-up waves of the Midlife Development in the U.S. National Survey. Early-life adversities include low socioeconomic status (SES), abuse, and family instability. Potential mediators include education, financial strain, purpose in life, mood disorder, family problems/support, and marital status. We used sequential logistic regression models to estimate the effect of early-life adversities on the risk of each of the three stages on the path to recalcitrant smoking (ever-smoking, smoking-related illness, and recalcitrant smoking).

Results For women, low SES (odds ratio [OR] = 1.29; 1.06–1.55) and family instability (OR = 1.73; 1.14–2.62) are associated with an elevated risk of recalcitrant smoking. Education significantly reduces the effect of childhood SES, yet the effect of family instability remains significant even after accounting for life-course mediators. For men, the effect of low SES on recalcitrant smoking is robust (OR = 1.48; 1.10–2.00) even after controlling for potential mediators. There are noteworthy life-course factors that independently affect recalcitrant smoking: for both genders, not living with a partner; for women, education; and for men, family problems.

Conclusions The findings can help shape intervention programs that address the underlying factors of recalcitrant smoking.

Keywords: Adverse childhood experience · Gender · Life course · Smoking · Cardiovascular disease · Cancer

Despite the gradual decline in the prevalence of smoking over the past half century, smoking has persisted as the number one cause of preventable deaths in the USA. Smoking is significantly linked to several cancers, as well as various cardiovascular, metabolic, and pulmonary diseases [1]. Smoking cessation is associated with reduced risk of cardiovascular disease among heavy smokers [2] and reduced mortality risk among individuals with coronary heart disease [3]. Some individuals, however, those we call *recalcitrant smokers*, continue to smoke despite having smoking-related conditions. For example, two-thirds of cancer survivors continue smoking after cancer diagnosis [4] and, after having a myocardial infarction, around half of the smokers continue smoking, even though this is linked to adverse health impacts and low quality of life [5].

Vulnerability to nicotine addiction might be a reason why some individuals with such adverse health conditions continue smoking [6], yet cumulative evidence shows that low socioeconomic position and stressful experiences are well-known predictors of the initiation and cessation of smoking, as well as relapses [7,8]. Such life circumstances that affect smoking habits may originate in early life [9–11]. The association between early-life adversities and recalcitrant smoking might be transmitted through life-course factors, yet we are aware of only one study that has tested such a hypothesis [12]. Some early-life adversities are more strongly and adversely tied to adult health for women than

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men, for example, the link between child abuse and later-life mortality [13]. However, we have little knowledge of whether women and men respond differently to early-life adversities in terms of the likelihood and mechanisms of smoking recalcitrance. To shed light on these issues, we investigate to what degree early-life adversities are associated with the risk of recalcitrant smoking, psychosocial factors that explain the association, and gender differences in any observed associations.

Background

Early-Life Adversities and Smoking

Early-life adversities include a host of negative experiences that may occur in childhood. Low socioeconomic status (SES), family instability, and abuse are among the most commonly occurring early-life adversities that we know of [14–16]. Such early-life experiences affect health and well-being throughout the life course, including the risk of smoking behaviors. For example, growing up in a low-SES family increases the risk of smoking initiation and the likelihood of smoking in adulthood [9]. For women, experiencing physical or sexual abuse in childhood increases the risk of regularly smoking cigarettes by 14 years of age [10] and of smoking in early midlife [11]. Similarly, exposure to family instability, such as growing up in divorced families, increases the risk of smoking and the number of cigarettes smoked for women [17].

Few studies, however, have investigated whether early-life adversities are associated with more harmful smoking behaviors, such as smoking despite having a serious medical condition. Only one study, to our knowledge, has found a significant dose–response association between early-life adversities and smoking among individuals who have smoking-related illnesses and symptoms, including heart disease, chronic lung disease, and diabetes [12]. This study was based on a community sample of adults who completed a standardized medical evaluation at a Health Maintenance Organization, so the findings may not be generalizable to broader populations. Additionally, this prior study focused on a cumulative number of exposures of all observed adversities [12], thus offering no insight into the unique effect of each domain of adversity (e.g., financial difficulties vs. abuse). Moreover, its use of cross-sectional data obviated the possibility of investigating extensive life-course pathways linking early-life adversities to recalcitrant smoking.

Gender Differences in the Effect of Early-Life Adversities

Women might be more vulnerable than men to early-life adversity in terms of later-health outcomes, although the association varies by type of adversity and health outcome. For example, women, but not men, who experienced parental divorce in childhood smoked more [17]. For women, but not men, childhood abuse increases the risk of later-life mortality [13]. An inverse association between childhood SES and later-life body mass index is stronger for women than men [18]. Yet, men, but not women, who grew up in low-SES families are more likely to consume unhealthy foods in midlife [19]. No study has yet examined gender differences in the association between early-life adversities and recalcitrant smoking, yet we have reason to believe that gendered patterns exist.

A long line of research about boys' sensitivity to economic hardship leads us to speculate that males' smoking behaviors might be more affected by low childhood SES. Boys are more adversely affected by growing up in economically deprived households, including experiencing less hopefulness, self-esteem, and confidence about their future [20,21]. In response to their increased vulnerability to economic hardship, boys tend to act out with more disruptive behaviors rather than with an emotional response, which may, in turn, increase harsh parental discipline for boys but not girls [22,23]. Due to harsh parenting practices and rejection from parental figures, young boys may be more likely to turn to peer groups that encourage more socially disapproved acts, such as smoking [24]. Such unhealthy behaviors established in early life may continue into later life.

In contrast, family instability and abuse in childhood might have unique consequences for women's smoking. Both family instability and childhood abuse are connected to impaired interpersonal relationships during adulthood [25,26], and the adverse impact might be stronger for women than men [27,28]. Gender differences may stem from highly gendered socialization processes during childhood where the differential emphasis is placed on closeness with others. Sex role theorists contend that girls, but not boys, are socialized to place great emphasis on forming and maintaining close interpersonal ties with others [29,30]. As a result, interpersonal relationships may originate from and cause emotional experiences for girls significantly more than for boys, and this may extend into adulthood [31]. Women are more likely than men to believe that smoking can be used to manage negative emotions and act as a coping tool for previously experienced stressors [32]. Therefore, unstable and abusive relationships in childhood may increase the risk of recalcitrant smoking for women.

Potential Pathways Linking Early-Life Adversities and Recalcitrant Smoking

Cumulative inequality theory suggests that adversities during childhood shape opportunities and risks over the life course, in turn, affecting later-health outcomes [33].

The life-course pathway model [34] suggests that early-life circumstances may influence subsequent material, social, and psychological life-course factors that may contribute substantially to the risk of smoking behavior. There is a well-established association between early-life adversities and decreased psychological well-being, usually in the form of depressive symptoms that extend decades beyond early experiences [35]. Studies based on clinical samples found that, among cancer patients, depressive symptoms are a significant predictor of persistent smoking [36] and smoking relapse [37]. Therefore, *depressive symptoms* could be an important pathway linking early-life adversities to recalcitrant smoking. A prior study, however, found that the mediating role of depression is not large [12].

Other life-course factors may help us understand why experiencing early-life adversities is associated with recalcitrant smoking, for example, *adult SES*. Individuals who experienced early-life adversities are less successful in school and in the labor market [38], and they have more financial difficulties in later life [39]. Compared to individuals with high SES, those with low SES are more likely to smoke and less likely to quit smoking [7]. Some studies have found that, after adjusting for adult SES, the association between childhood SES and adult smoking is attenuated to nonsignificance, suggesting that one's adult SES may be significantly associated with current smoking more so than childhood SES [40,41]. Moreover, childhood adversities can lead to further hardships and stress exposures in later adulthood [42]. Psychological stress, including exposure to stressful life events and chronic and financial strain, is associated with the persistence of and relapse into smoking behaviors [7,43]. A recent study has shown that stressful events impacting family members are also associated with smoking in midlife [8], indicating the important role of *network stress* on an individual's health behaviors.

Another important intervening mechanism is an individual's motivation to quit smoking, which may be bolstered by social support. Studies have found an association between high SES and smoking cessation possibly through desire, intention, or sense of duty to stop smoking [44]. The observed associations may, in part, be attributed to individuals from low SES having lower levels of *purpose in life* [45]—a psychological asset that helps individuals prioritize long-term goals over immediate ones, such as engaging in unhealthy behaviors [46]. Moreover, *social support* best helps individuals stop smoking when the support is consistent and nondirective [47]. Yet, individuals who experienced early-life adversities report lower levels of social support in adulthood, such as not having a spouse/partner and decreased perceptions that one's family is supportive [26,48].

Hypotheses of the Current Study

Based on this extant literature, we hypothesize that early-life adversities (childhood SES, family instability, and abuse) will be statistically significant predictors of the risk of individuals' recalcitrant smoking (*Hypothesis 1*). We expect that some early-life adversities will be more influential for women than men. Specifically, we hypothesize that socioeconomic disadvantage will be more strongly associated with the risk of recalcitrant smoking for men than women (*Hypothesis 2a*), while unstable and abusive relationships in childhood will be more strongly associated with the risk of recalcitrant smoking for women than men (*Hypothesis 2b*). Using selected psychosocial factors from the literature, we then test *Hypothesis 3*: psychosocial factor(s) will mediate the positive association between early-life adversities and recalcitrant smoking. Given that little is known in terms of predictors of recalcitrant smoking, throughout these analyses, we pay close attention to those life-course factors that are significantly associated with recalcitrant smoking, as well as gendered patterns in the associations.

Method

Sample

Data for this study come from the Midlife Development in the United States (MIDUS) study, a national survey designed to assess the role of social, psychological, and behavioral factors in understanding differences in mental and physical health (n = 7,108; 52% women). MIDUS began in 1995–1996 (Wave 1) with noninstitutionalized, English-speaking adults aged 25–74 in the 48 contiguous states [49]. National random digit dialing with oversampling of older people and men was used to select the main sample (n = 3,478) and a sample of twin pairs (n = 1,914). The study also includes a random subsample of siblings of individuals in the main sample (n = 950) and oversamples from five metropolitan areas in the USA (n = 757). MIDUS consists of a two-stage survey: a telephone interview and a self-administered questionnaire. Approximately 89% of the sample completed the two-stage survey at Wave 1 (n = 6,325). Follow-up interviews with MIDUS respondents were completed in 2004–2006 (n = 4,963). The longitudinal retention rate for Wave 2 was 75% after adjusting for mortality. Additional information about sampling, enrollment, and longitudinal retention is documented elsewhere [50]. The present analysis uses data from the 4,932 individuals who participated in both the initial and the follow-up survey (Wave 2). Compared to individuals who died or were

lost to follow-up at Wave 2, those who participated in both waves were more likely to be white, female, married, more highly educated, and to report having better health. This attrition may result in selection bias between Waves 1 and 2.

Measures

Early-life adversities (Wave 1) include socioeconomic disadvantage, family instability, and parental abuse. By maximizing available information in MIDUS, we created the index of low childhood SES (Cronbach's α = .74, mean = 0, standard deviation [*SD*] = 1.00), which is an average of standardized scores of six indicators: mother's and father's education (1 = no school/some grade school to 12 = PhD, MD, or other professional degrees), mother's and father's occupational prestige score as measured by Duncan's Socioeconomic Index [51], welfare status (0 = never on welfare and 1 = ever on welfare), and financial level growing up (1 = a lot better off than the average family to 7 = a lot worse off). Family instability is a binary indicator based on the question, "Did you live with both of your biological parents up until you were 16?" Possible reasons for a negative response ("No") include parental death, separation or divorce, parents not living together, and never knowing a biological parent. For childhood abuse, respondents were presented with a battery of items from the modified version of the Conflict Tactics Inventory [52]. Respondents were asked how often they had endured each of the following forms of abuse, which fall into three lists/domains: List A (made insulting remarks; sulked or refused to talk; stomped away; did something out of spite; made threats; and kicked/smashed something in anger), List B (pushed, grabbed, or shoved; slapped; and object thrown at respondent), and List C (kicked, bit, or hit with a fist; hit with an object [or attempted]; beat up; choked; and burned or scalded). List A includes items related to emotional abuse, while items in Lists B and C represent physical abuse. The response options included 1 = never, 2 = rarely, 3 = sometimes, and 4 = often. The correlation between emotional and physical abuse was .73. By averaging the 15 items (the three domains of abuse with respect to mother, father, brother, sister, and others), we created an index of childhood abuse (α = .86, mean = 1.65, *SD* = .47).

Recalcitrant smoking in midlife (Wave 2)

To identify recalcitrant smokers, we followed the three-stage process described in Fig. 1. We first obtained smoking status through a question at Wave 1 ("Have you ever smoked cigarettes regularly—that is, at least a few cigarettes every day?"). Next, we identified those ever-smokers who developed or had at least one of the following illnesses or symptoms at Wave 2 that may be exacerbated by smoking: ever had heart problems suspected or confirmed by a doctor, ever had a heart attack, ever experienced or been treated for a stroke in the past 12 months, ever experienced or been treated for hypertension in the past 12 months, ever experienced or been treated for asthma, bronchitis, emphysema, or other lung problems in the past 12 months, ever experienced or been treated for diabetes or high blood sugar in the past 12 months, or ever had cancer. Finally, we identified *recalcitrant smokers* as those who have ever had smoking-related illnesses but indicated during the Wave 2 interview that they smoke regularly (*n* = 327). The logic to identify *recalcitrant smokers* is consistent with prior work [12].

Life-course mediators (Wave 1)

Based on prior studies, we included six mediators linking early-life adversities to smoking status in midlife: education, financial strain, mood disorder, recent family problems, purpose in life, and marital or cohabiting status. For *education,* respondents reported their highest grade of school or year of college completed. Response categories ranged from 1 = no school/some grade school to 12 = PhD, MD, or other professional degree. The index of *financial strain* (α = .82, mean = 0, *SD* = 1.00) is an average of standardized scores of four indicators: current financial situation (0 = worst possible through 10 = best possible), control over financial situation (0 = worst possible through 10 = best possible), availability of money to meet basic needs (1 = more than enough through 3 = not enough, reverse coded), and level of difficulty paying bills (1 = very difficult through 4 = not at all difficult). *Mood disorder* is a binary variable that indicates whether a respondent has major depressive disorder or generalized anxiety disorder. Both disorders were assessed through phone interviews that used the screening versions of the World Health Organization's Composite International Diagnostic Interview, Version 10 [53]. Major depressive disorder and generalized anxiety disorder were defined according to criteria specified in the third revised edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R; 1987). Consistent with prior work [8], we created an index of *family problems* using 30 items that capture whether three major family members (spouse/partner, parents, and children) had problems during the past 12 months in any of the following 10 areas: health, alcohol use, substance use, finances, work, school, job, legal issues, marriage, or other relationships. Response options were yes (coded 1) or no (coded 0). We summed the scores from the three family members (index range 0 to 30, mean = 1.03, *SD* = 1.05). A *purpose in life* index was created using a three-item

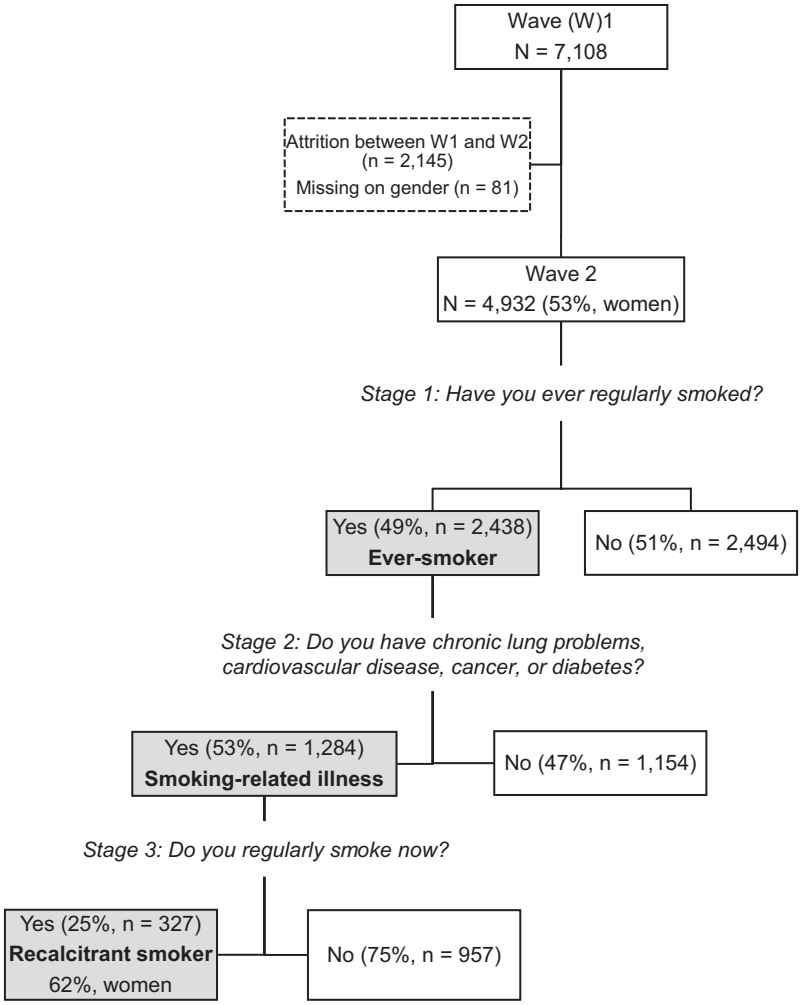

Figure. 1. Sequential process to identify recalcitrant smokers.

version of Ryff's Scale of Psychological Well-Being [46]. On a scale from 1 = strongly disagree to 7 = strongly agree, participants responded to three statements: "I live life one day at a time and do not really think about the future"; "Some people wander aimlessly through life, but I am not one of them"; and "I sometimes feel as if I've done all there is to do in life." Purpose in life was the average of these three items (α = .44, mean = 16.26, *SD* = 3.69). The low reliability of the index is due, in part, to the small number of items. Sensitivity analysis shows that substantial findings are consistent across a 7-item version in Wave 2 (α = .70, mean = 37.31, *SD* = 7.20) vs. a three-item version in Wave 1. Thus, we used the index from Wave 1 to ensure the temporal order between the mediator and outcome.

Married/cohabiting is a binary variable based on a question asking whether the respondent was married or living with someone. We created *family support* using four questions reflecting positive relations with family members: "How much do members of your family really care about you?"; "How much do they understand the way you feel about things?"; "How much can you rely on them for help if you have a serious problem?"; and "How much can you open up to them if you need to talk about your worries?" Possible responses included 1 = not at all, 2 = a little, 3 = some, and 4 = a lot. Given that the average score of the four items was positively skewed, we created a binary indicator of high family support if the average score was equal to 4 (= a lot). We included three demographic confounders from Wave 1, which are associated with smoking status: age as a continuous variable (mean = 46.38, *SD* = 13.00), race (1 = white; 0 = nonwhite), and gender (for gender-stratified models). Racial and gender statuses were created using self-identified racial status and sex (male vs. female).

Analytic Strategies

Descriptive statistics were calculated using two-tailed *t*-tests for continuous indicators and χ^2tests for binary variables. We used sequential logistic regression models

to estimate the effect of early-life adversities on the risk of each of the three irreversible stages toward recalcitrant smoking: one must be a smoker (Stage 1), then develop a smoking-related illness (Stage 2), and then continue smoking despite having such a health condition (Stage 3). As shown in Fig. 1, these three stages consist of the following transitions. The first transition is a choice between being a smoker or not, the second transition is having a smoking-related illness or not among those who smoke, and the third transition is a choice between being a recalcitrant smoker or not among smokers who have a smoking-related illness. At each stage, the model predicts the effects of early-life adversities on the next transition using the sample from the prior transition as

Stage 1: P (being a smoker) $= \text{logit}^{-1}(\beta_{01} + \beta_{11}X)$,
Stage 2: P (having a smoking related illness|being a smoker) $= \text{logit}^{-1}(\beta_{02} + \beta_{12}X)$ and
Stage 3: P (being a recalcitrant smoker|being a smoker & having a smoking related illness) $= \text{logit}\ \beta_{03} + \beta_{13}X$ where βs are regression coefficients, $\text{logit}^{-1}(\cdot) = \frac{\exp(\cdot)}{1+\exp(\cdot)}$, and X represents early-life adversities.

At the last stage presented, we created a series of nested models to investigate the life-course factors that explain why individuals exposed to early-life adversities become recalcitrant smokers. Given that we have multiple exposures to early-life adversities and mediators, we proceeded as follows. First, we estimated the effect of each adversity and life-course factor on recalcitrant smoking individually (baseline models). We, then, added all three adversities together to investigate the additive effects of each adversity on recalcitrant smoking (Model 1). If the additive effect of an exposure was significant, we examined the mediation effect of the exposure via life-course factors. In the following models, only the life-course factors found to be significant in the baseline models were added to Model 1 as potential mediators one at a time. The final model includes all exposures and mediators in order to examine the mediation effects via all combined life-course factors and to identify life-course factors, which have a significant effect net of other covariates.

Coefficients from nested nonlinear probability models are not comparable because of unobserved heterogeneity—that is, the variation in the dependent variable that is caused by variables that are not observed [54]. Therefore, to ensure the comparability across nested models in logistic regression, we used the Karlson–Holm–Breen (KHB) method [55] and, then, computed the percentage of the mediation effect relative to the total effect via each/all mediators. The KHB method adjusts coefficients by separating the variation that is caused by omitted variables. To yield estimates comparable across predictors, all continuous predictors were standardized at the mean with the *SD* equal to 1. The analysis was stratified by gender, and gender differences in direct effects of Early-life adversities and indirect pathways were tested by pooling data from both genders and testing gender interaction terms.

The sequential model, which we have used for a primary method, is a conventional model for addressing this type of dependent variable with multiple stages. However, this model cannot address possible selection biases due to excluding nonsmokers (Stage 2) and nonsmokers and healthy smokers (Stage 3). To handle the possible selection biases, we used the Heckman model [56] as a sensitivity analysis. Given that the Heckman model only addresses two stages, we confined our sample to smokers and replicated the analysis of Stage 3 (i.e., being a recalcitrant smoker). The results from the Heckman model, which handles possible selection biases, are not substantially different from the results from sequential models (see Supplementary Table 1A).

All control variables and mediators have 1%–2% of data missing on average. We handled missing data for these confounders using standard practices of multiple imputation [57]. To adjust for the possible selection bias between Waves 1 and 2 in terms of sociodemographic characteristics, we weighted the remaining sample by the inverse of the probability of dropping out (due to death or being lost to follow-up) given characteristics, such as race, gender, marital status, and education. Detailed procedures are provided elsewhere [58,59]. Robust standard errors (*SE*s) were used to correct for intrafamily correlation given that multiple individuals are from the same family (e.g., twins and siblings). As a result, all analyses employed (a) multiple imputation, (b) a poststratification weight, and (c) robust *SE*s due to clustering. All analyses were implemented using Stata 15.0 [60], except gender differences in indirect pathways, which were analyzed with Mplus 8.0 [61].

Results

Baseline Characteristics by Smoking Status

As displayed in Fig. 1, 49% of respondents who participated in Wave 2 were identified as *ever-smokers*. Around half of them reported that they had at least one disease or symptom that might be exacerbated by smoking. Among respondents who had a smoking-related illness, one-fourth continued smoking despite having such an adverse health condition. The prevalence of recalcitrant smoking was around 7% in the MIDUS sample (327/4,932). The likelihood of being recalcitrant smokers was slightly higher for women (8%) than men (5%).

Table 1 displays the results from bivariate analyses that tested whether all variables used in the analyses varied by the stages toward recalcitrant smoking. In the comparison between nonsmokers and ever-smokers (Stage 1), ever-smokers were more likely than nonsmokers to have experienced low SES, abuse, and family instability in childhood. Ever-smokers tended to be male and older compared to nonsmokers. Among respondents in Stage 2, those who had a smoking-related illness were older and more likely to have experienced low childhood SES than those who did not have a smoking-related illness. Among respondents with a smoking-related illness (Stage 3), individuals who continued smoking experienced more adversities in early life than those who quit smoking. They also showed lower levels of resources that tend to inhibit smoking—such as education, purpose in life, family support, and living with someone—and higher levels of risk factors of smoking, including financial strain, mood disorder, and family problems. Recalcitrant smokers, however, were more likely to be younger and female than those who quit smoking after having a smoking-related illness.

Early-Life Adversities and Being an Ever-Smoker

Table 2 shows the odds ratios (ORs) and confidence intervals (CIs) of the sequential response model at Stages 1 (being an ever-smoker) and 2 (having a smoking-related illness). We estimated the effect of each adversity in baseline models and, then, estimated the effects of all three adversities in the additive model. Results from logistic regression models in the association between early-life adversities and ever-smoking are shown in Table 2 (left). In baseline models, we found that all three adversities were significantly related to an elevated risk of ever-smoking for both genders. When all adversities were considered simultaneously, childhood abuse (OR = 1.39, CI = [1.27–1.52]) and family instability (OR = 1.72, CI = [1.40–2.11]) were significantly associated with ever-smoking for women, while all three adversities—low SES (OR = 1.24, CI = [1.12–1.37]), childhood abuse (OR = 1.22, CI = [1.10–1.35]), and family instability (OR = 1.70, CI = [1.33–2.18])—remained statistically significant for men.

Early-Life Adversities and Having a Smoking-Related Illness

Early-life adversities continued to be significant predictors in the second stage, that is, having a smoking-related illness (right in Table 2). However, only some early-life adversities appeared statistically significant in the second stage. Specifically, the results from additive

Table 1. Descriptive statistics of sample by sequential processes of recalcitrant smoking

	Stage 1: Ever regularly smoked? (n = 4,932)		Stage 2: Have smoking- related illness? (n = 2,438)		Stage 3: Regularly smoke now? (n = 1,284)	
	No (51%)	Yes (49%)	No (47%)	Yes (53%)	No (75%)	Yes (25%)
Early-life adversities						
Low childhood SES	−0.16	0.05***	−0.07	0.16***	0.11	0.32**
Childhood abuse	−0.14	0.12***	0.15	0.10	0.05	0.24**
Family instability, %	16%	24%***	24%	24%	21%	33%***
Controls						
Age	45.29	47.65***	42.67	52.14***	54.06	46.55***
White, %	92%	94%**	94%	94%	94%	93%
Female, %	57%	49%***	51%	48%	44%	62%***
Midlife mediators						
Education					−0.04	−0.37***
Financial strain					−0.13	0.23***
Mood disorder, %					14%	28%***
Purpose in life					−0.01	−0.23**
Family problems					0.12	0.46***
High family support, %					30%	22%**
Married/cohabiting, %					77%	66%***

Two-tailed t-test for mean difference in continuous indicators and chi-square test for percentage difference in binary variables.

SES socioeconomic status.

*p < .05, **p < .01, ***p < .001.

Table 2. Odds ratios (ORs) and confidence levels (CIs) of the sequential response model of smoking stages

	Ever regularly smoked (Stage 1)				Smoking-related illness (Stage 2)			
	Women (n = 2,630)		Men (n = 2,302)		Women (n = 1,203)		Men (n = 1,235)	
	Baseline models OR (95% CI)	Additive model OR (95% CI)	Baseline models OR (95% CI)	Additive model OR (95% CI)	Baseline models OR (95% CI)	Additive model OR (95% CI)	Baseline models OR (95% CI)	Additive model OR (95% CI)
Early-life adversities								
Low childhood SES	1.10* (1.01, 1.20)	1.01 (0.93, 1.11)	1.3*** (1.20, 1.45)	1.24*** (1.12, 1.37)	1.08 (0.96, 1.22)	1.05 (0.93, 1.18)	1.25** (1.09, 1.42)	1.22** (1.06, 1.40)
Childhood abuse	1.40*** (1.28, 1.53)	1.39*** (1.27, 1.52)	1.26*** (1.15, 1.39)	1.22*** (1.10, 1.35)	1.16* (1.03, 1.31)	1.15* (1.02, 1.30)	1.14 (0.99, 1.31)	1.10 (0.95, 1.26)
Family instability	1.74*** (1.42, 2.14)	1.72*** (1.40, 2.11)	1.88*** (1.48, 2.40)	1.70*** (1.33, 2.18)	1.31 (0.99, 1.75)	1.28 (0.96, 1.70)	1.14 (0.83, 1.56)	1.04 (0.75, 1.44)

All models control for age and race/ethnicity.

SES socioeconomic status.

*p < .05, **p < .01, ***p < .001.

models show that childhood abuse was associated with elevated odds of having a smoking-related illness for women (OR = 1.15, CI = [1.02–1.30]), while low SES was associated with elevated odds for having a smoking-related illness for men (OR = 1.22, CI = [1.06–1.40]).

Early-Life Adversities, Life-Course Mediators, and Being a Recalcitrant Smoker

For the third stage, we investigated early-life adversities and psychosocial factors that help us understand why some individuals with a smoking-related illness continue to smoke while others quit. For women, results from baseline models in Table 3 show that the risk of recalcitrant smoking is higher for women who grew up in low SES (OR = 1.32, CI = [1.10–1.58]) and unstable families (OR = 1.83, CI = [1.23–2.73]). The unique effects of these two early-life adversities remained statistically significant even after all early-life adversities were estimated simultaneously in Model 1. There are five psychosocial factors that are significantly related to elevated risk of recalcitrant smoking for women at baseline: higher levels of education (OR = 0.57, CI = [0.46–0.71]), higher levels of purpose in life (OR = 0.79, CI = [0.68–0.94]), living with a partner (OR = 0.57, CI = [0.39–0.83]), more financial strain (OR = 1.19, CI = [1.01–1.40]), and mood disorder (OR = 1.65, CI = [1.08–2.50]). In mediation analyses, we found that education has a significant indirect effect on the association between childhood SES and recalcitrant smoking for women by accounting for around 79% of the association (p < .001). After controlling for education, the unique effect of childhood SES was no longer statistically significant (Model 2). Regarding the unique effect of family instability on recalcitrant smoking, there are no life-course factors that show significant indirect effects (Models 2–6).

For men in Table 4, low SES in early life (OR = 1.48, CI = [1.15–1.90]) is associated with an increased risk of recalcitrant smoking in the baseline model. Yet, neither childhood abuse nor family instability had a significant effect on recalcitrant smoking. The results from baseline models showed that the risk of recalcitrant smoking for men is inversely associated with purpose in life (OR = 0.77, CI = [0.63–0.94]), high family support (OR = .53, CI = [0.31–0.94]), and living with a partner (OR = 0.51, CI = [0.31–0.86]), yet the risk is positively associated with family problems (OR = 1.27, CI = [1.05–1.54]). In the mediation analysis, we found that no life-course factors have significant indirect effects on the association between low childhood SES and recalcitrant smoking.

In the final model, which includes all early-life adversities and psychosocial factors, we found that the effect of family instability (OR = 1.55, CI = [1.01–2.39]) remained significant and that there were two life-course factors—lower

education (OR = 0.58, CI = [0.45–0.74]) and not living with a partner (OR = 0.51, CI = [0.34–0.77])—which significantly differentiated women with a smoking-related illness who quit smoking versus those who continued smoking despite having such a health problem. For men, the effect of childhood SES (OR = 1.48, CI = [1.10–2.00]) remained significant in the final model. Having more family problems (OR = 1.28, CI = [1.04–1.58]) and not living with a partner (OR = 0.54, CI = [0.32–0.91]) were independent factors that explained why male smokers continued smoking despite having a smoking-related illness.

In terms of the direct effect of early-life adversities in the final models, we found that gender differences were not statistically different. The indirect pathway from childhood SES to recalcitrant smoking through adult education was stronger for women than men ($p < .01$) even after accounting for all other potential mediators.

Discussion

Our study yielded three major findings. We found that individuals who experienced childhood adversities are more likely to be recalcitrant smokers in midlife. There are gendered patterns in the type of early-life adversities that uniquely shape the likelihood of being recalcitrant smokers. Based on prior research on childhood development [20], we expected that males' smoking behaviors would be more strongly affected by socioeconomic position in childhood. Indeed, we found an inverse association between childhood SES and the probability of being recalcitrant smokers, particularly for men. Theories of gender socialization and sex roles [29] further guide us that abusive and insecure relationships in early life might harm women more than men. We found that family instability is significantly associated with the increased risk of recalcitrant smoking for women but not for men. Prior research on substance abuse calls for more interventions that target those with childhood trauma [62]. Interventions that target women while addressing both substance abuse and complex trauma produce more favorable outcomes when compared to programs that only address substance abuse [63,64]. In a similar vein, smoking-cessation interventions for recalcitrant smokers who experienced early-life adversities might be more effective if the program provides coping skills for managing early-life trauma. More research is needed regarding whether gender-specific interventions would improve outcomes.

Guided by cumulative inequality theory [33] and the life-course framework [34], we hypothesized that there

Table 3. Sequential logit estimates (95% confidence intervals) predicting recalcitrant smoking (Stage 3) for women ($n = 620$)

	Baseline	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Early-life adversities								
Low childhood SES	1.32** (1.10, 1.58)	1.29** (1.06, 1.55)	1.06 (0.86, 1.31)	1.27* (1.05, 1.54)	1.30** (1.07, 1.58)	1.25* (1.03, 1.52)	1.31** (1.08, 1.60)	1.09 (0.87, 1.35)
Childhood abuse	1.04 (0.89, 1.23)	1.00 (0.84, 1.19)	0.98 (0.83, 1.17)	0.98 (0.82, 1.17)	0.97 (0.81, 1.16)	0.98 (0.83, 1.17)	0.98 (0.82, 1.16)	0.92 (0.77, 1.11)
Family instability	1.83** (1.23, 2.73)	1.73** (1.14, 2.62)	1.77** (1.17, 2.68)	1.70* (1.12, 2.57)	1.63* (1.07, 2.47)	1.71* (1.13, 2.60)	1.68* (1.11, 2.54)	1.55* (1.01, 2.39)
Midlife mediators								
Education	0.57*** (0.46, 0.71)		0.59*** (0.47, 0.76)					0.58*** (0.45, 0.74)
Financial strain	1.19* (1.01, 1.40)			1.15 (0.97, 1.37)				0.96 (0.80, 1.17)
Mood disorder	1.65* (1.08, 2.50)				1.58* (1.01, 2.47)			1.39 (0.85, 2.25)
Purpose in life	0.79** (0.68, 0.94)					0.83* (0.70, 0.98)		0.94 (0.78, 1.13)
Family problems	1.12 (0.97, 1.31)							1.16 (0.98, 1.38)
High family support	0.87 (0.58, 1.31)							0.96 (0.62, 1.49)
Married/cohabiting	0.57** (0.39, 0.83)						0.56** (0.39, 0.83)	0.51** (0.34, 0.77)

All models control for age and race/ethnicity.

SES socioeconomic status.

*$p < .05$, **$p < .01$, ***$p < .001$.

are psychosocial factors that explain why some individuals continue to smoke despite having a smoking-related illness. We found that education in adulthood substantially explains why women who grew up in low-SES families are more likely to be recalcitrant smokers. However, even after accounting for all potential mediators, the direct effect of some early-life adversities remains significant. In particular, family instability is a robust predictor for women, while childhood SES is a robust predictor for men. For men, the mediating role of education in the association between childhood SES and recalcitrant smoking is negligible. None of the potential mediators substantially explain why women who grew up in unstable families tend to be recalcitrant smokers in later life. We interpret our findings in line with the biological embedding model, that is, early-life adversity induces significant developmental changes in children, modifying the maturation and responsiveness of physiological systems and developmental changes [65]. Early-life adversities may alter brain areas that govern executive functioning and reward systems [66], which may result in fostering impulsiveness and unhealthy coping behaviors, such as smoking [67]. Moreover, we cannot exclude the possibility that data limitations may affect the significance and strength of mediators given that all mediators were measured in midlife. Although we carefully selected the mediators based on the literature, we acknowledge the possibility that these mediators may not be unique to the early-life adversity. There may be other potential mediators (e.g., living in a disadvantaged neighborhood) that future researchers should consider.

There are noteworthy life-course factors that independently explain why middle-aged individuals continue to smoke in the face of a smoking-related illness. For both genders, the risk of recalcitrant smoking is low for those who live with a partner. Our findings are in line with the literature on the positive aspects of marriage/cohabitation on smoking behaviors [68]. We also found gender-specific life-course factors. In line with research on the health benefits of education [69], education plays a role in recalcitrant smoking for women. Although it is established that smoking prevalence is higher among those with lower education [70], our findings expand prior work by indicating that education predicts a more harmful form of smoking, that is, smoking despite having serious medical conditions, particularly for women. We also found that the risk of recalcitrant smoking was higher when men have family members who struggle with their own health, substance use, finances, work, or marriage. These findings are in line

Table 4. Sequential logit estimates (95% confidence intervals) predicting recalcitrant smoking (Stage 3) for men (n = 664)

	Baseline	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Early-life adversities							
Low childhood SES	1.48** (1.15, 1.90)	1.52** (1.16, 1.99)	1.49** (1.14, 1.95)	1.52** (1.16, 1.99)	1.52** (1.17, 1.99)	1.50** (1.14, 1.96)	1.48* (1.10, 2.00)
Childhood abuse	1.01 (0.81, 1.26)	0.95 (0.76, 1.19)	0.93 (0.75, 1.16)	0.93 (0.74, 1.17)	0.93 (0.74, 1.17)	0.96 (0.77, 1.21)	0.90 (0.72, 1.14)
Family instability	1.06 (0.64, 1.78)	0.87 (0.50, 1.50)	0.85 (0.50, 1.47)	0.86 (0.49, 1.48)	0.86 (0.50, 1.48)	0.89 (0.51, 1.53)	0.83 (0.48, 1.43)
Midlife mediators							
Education	0.88 (0.70, 1.11)						1.00 (0.77, 1.31)
Financial strain	1.21 (0.97, 1.51)						1.02 (0.79, 1.30)
Mood disorder	1.51 (0.85, 2.67)						1.17 (0.64, 2.16)
Purpose in life	0.77** (0.63, 0.94)		0.79* (0.65, 0.96)				0.83 (0.67, 1.04)
Family problems	1.27* (1.05, 1.54)			1.28* (1.05, 1.56)			1.28* (1.04, 1.58)
High family support	0.53* (0.31, 0.94)				0.53* (0.30, 0.93)		0.62 (0.34, 1.12)
Married/cohabiting	0.51* (0.31, 0.86)					0.53* (0.32, 0.90)	0.54* (0.32, 0.91)

All models control for age and race/ethnicity.

SES socioeconomic status.

*p < .05, **p < .01, ***p < .001.

with the principles of "linked lives" [20] and stress crossover [71]. That is, family members who suffer from their own problems may provide less support and more strain, which may inhibit men's smoking cessation. Like prior work [36,37], mood disorders matter for women's recalcitrant smoking, but the effect is not significant when an extensive set of mediators is taken into account.

Several methodological limitations should be acknowledged. First, given that MIDUS lacks information on when respondents developed smoking-related conditions, we cannot establish the temporal order between the initiation of smoking and the development of a smoking-related illness. However, around 90% of ever-smokers in MIDUS began regularly smoking before age 25 and most smoking-related conditions are likely to develop in later life. Thus, we assumed that, in the vast majority of cases, respondents would have begun smoking long before they developed a smoking-related illness. Second, indicators of early-life adversities are vulnerable to recall bias, and some indicators of early-life adversities may not fully capture difficulties that individuals encounter in early life. For example, family instability is measured with a single indicator (whether a respondent had lived with both biological parents until age 16), so the diverse causes (e.g., parental incarceration, death, and divorce), duration, or severity of the adversity were not measured.

Moreover, the measure of childhood abuse only includes emotional and physical abuse but not sexual abuse, which is more common among women [72]. Given the significant association between sexual abuse and smoking, particularly for women [11], this would lead to conservative estimates of the effect of childhood abuse. Third, we have created indexes—for example, family problems—by summing a wide array of indicators across different family members. Our findings, thus, do not provide more specific information (spouse vs. children) relevant to smoking habits. Finally, although we have included potential variables that are carefully selected from the literature, we cannot rule out the possibility that there still remains confounding by unmeasured variables, a common limitation in observational research.

In conclusion, using a longitudinal study of middle-aged U.S. adults, we observed that disadvantaged environments and stressful experiences in early life might increase the risk of recalcitrant smoking. Moreover, the nature of early-life experiences may affect girls versus boys differently in terms of their risk of such a harmful smoking behavior. Future research should continue to examine how and why early-life adversities and potential mediators operate differently for men and women. To reduce the prevalence of such an intractable smoking habit, our results call for interventions for recalcitrant smokers who were disadvantaged in early life.

Supplementary Material

Supplementary material is available at *Annals of Behavioral Medicine* online.

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Compliance With Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards The authors declare that they have no conflict of interest.

Authors' Contributions C.L. led the conceptual and analytic design, analyzed the data, and drafted and revised the article. L.H. helped to formulate the initial idea, conducted preliminary analyses, drafted parts of the manuscript and contributed to the revision process. S.P. designed, conducted and drafted parts of the method section and contributed to the revision process.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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