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Disparities in cigarette smoking and the health of marginalized populations in the U.S.: a simulation analysis

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Abstract

Introduction People with low socioeconomic status (SES) or serious psychological distress (SPD) in the U.S. face ongoing and future disparities in tobacco smoking. We sought to estimate how smoking disparities contribute to disparities in life expectancy and aggregate life-years in these marginalized subpopulations.

Methods We used the Simulation of Tobacco and Nicotine Outcomes and Policy (STOP) microsimulation model to project life expectancy as a function of subpopulation (low SES, higher SES, SPD, or non-SPD) and cigarette smoking status. Low SES was defined as having at least one of the following: income below poverty, less than high school education, or Medicaid insurance. Higher SES individuals belonged to none of these categories. SPD was defined as Kessler-6 score ≥ 13; non-SPD was a Kessler-6 score < 13. To project individual life expectancy losses from smoking, we simulated 40-year-olds stratified by gender, subpopulation (by SES or by SPD, with no change), and smoking status (current/never, with no change). To project time to reach 5% cigarette smoking prevalence (U.S.) – reflecting one tobacco "endgame" threshold – in each subpopulation, we simulated the entire subpopulations of people with low SES, higher SES, SPD, and non-SPD, incorporating corresponding distributions of gender, age, and smoking status and accounting for changes in smoking behaviors and secular smoking trends. We then estimated total life-years accumulated under status quo and alternate scenarios in which smoking dynamics in the marginalized subpopulations matched those of their less marginalized counterparts.

Results The model showed that, for individuals with low SES or SPD, smoking is associated with substantial loss of life expectancy (9.8-11.5y). Marginalized subpopulations would reach 5% smoking prevalence 20y (low SES) and 17y (SPD) sooner if smoking trends mirrored their less marginalized counterparts; these differences result in 5.3 million (low SES) and 966,000 (SPD) excess life-years lost over 40y.

Conclusions Differences in cigarette smoking portend substantial ongoing and future disparities in life expectancy and time to reach 5% smoking prevalence. Reducing tobacco-related disparities in the U.S. will require an explicitly equity-focused vision, and the tobacco endgame will only be truly achieved when it includes all groups.

Keywords Tobacco, Health disparities, Simulation modeling

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Introduction

Tobacco smoking causes approximately 480,000 deaths per year in the U.S [1]. Although tobacco continues to harm population health, tobacco control over recent decades has been a major public health and public policy success. Since the first Surgeon General's Report on smoking and health in 1964 and the adoption of numerous tobacco control initiatives, cigarette smoking prevalence among U.S. adults has declined from 42% in 1964 to 11.6% in 2022 [2]. At a population level, these changes saved approximately 157 million life-years from 1964 to 2012 [3]. With this decline in smoking, public health experts are contemplating the path by which cigarette use – and tobacco use more broadly – go from uncommon to rare, or the "tobacco endgame." [4].

As cigarette smoking has declined overall, it has become increasingly concentrated in socially marginalized populations in the U.S [5–8]. Individuals with low socioeconomic status (SES) are the largest of these groups, comprising 9-13% of the U.S. population in 2019 [9–11]. Tobacco companies have long targeted lower-income communities for sales [12, 13]. Of people whose household incomes were less than $35,000, current cigarette use in 2020 was 25.2%, compared to 13.7% among individuals with household incomes of $100,000 or greater [14]. Meanwhile, disparities in smoking are greatest when comparing people with poor mental health to those with average or better mental health. Nicotine may temporarily decrease symptoms associated with serious psychological distress (SPD), but smoking cessation is associated with longer-term improved mental health [15]. Cigarette smoking prevalence among people identified as experiencing SPD (who comprise 3.9% of the 2018 U.S. population) [16] ranges from 31.0 to 40.6%, whereas it ranges from 12.5 to 14.0% among people without SPD [17, 18].

Both relative and absolute disparities are increasing due to the slower rate of decline in cigarette smoking prevalence among marginalized populations [19]. With the approach of the tobacco endgame, an increasingly disproportionate share of morbidity and mortality due to smoking is falling on these marginalized groups in the U.S. Therefore, the three objectives of this study were to (1) quantify the current life expectancy losses associated with cigarette smoking in people with low SES or SPD; (2) project when these subpopulations and their less marginalized counterparts might reach an endgame threshold, defined here as < 5% cigarette smoking prevalence (a population health target set in Healthy People 2030, the U.S. Department of Health and Human Services' decennial report on national health goals [20]); and (3) estimate the future life-year losses due to disparities in reaching this threshold.

Methods

Analytic overview

We used the Simulation of Tobacco and Nicotine Outcomes and Policy (STOP) model, a validated microsimulation of tobacco- and nicotine-related behaviors and clinical outcomes, applied to two different types of analyses [21]. In the first type of analysis (Individual-Level Life Expectancy), we used the STOP model to project the impact on life expectancy of tobacco cigarette smoking (current vs. never) for two pairs of marginalized subpopulations – (1) low SES vs. higher SES and (2) SPD vs. non-SPD, defined below. We compared marginalized subpopulations with their less marginalized counterparts and compared people who currently smoke versus never smoked (with no change in smoking status over time), matched by gender and age. With this analysis, we illustrate an upper bound on the negative effects of tobacco use and SES/SPD status, allowing comparison of the relative impact of each on life expectancy [22].

In the second type of analysis (Population-Level Smoking and Mortality Dynamics), we used the STOP model to simulate cohorts intended to reflect all U.S. adults in each of the four subpopulations (low SES, higher SES, SPD, non-SPD) – accounting for differing distributions of gender, age, and smoking status within each subpopulation and for changes in smoking behaviors over time – to project when each subpopulation would reach a threshold of 5% cigarette smoking prevalence, as well as total life-years accrued over a 40-year period by each subpopulation starting in 2019.

Below, we provide an overview of the model and definitions of the subpopulations. Then, we detail the first type of analysis, followed by a description of key differences in the second type of analysis.

Model overview

STOP is a Monte Carlo microsimulation model in which simulated individuals may transition monthly through various tobacco smoking states, each with associated mortality risks. Each simulation can consist of people homogenous by gender, age, and smoking status, or it can be constituted to resemble real populations with specific distributions of gender, age, and smoking status. Within a simulation, smoking status can vary over time based on monthly probabilities of initiation, cessation, and relapse, or it can remain static by setting these probabilities of smoking status change to zero. We previously validated the STOP model against 1997–2009 National Health Interview Survey (NHIS) data on smoking prevalence and mortality by age and sex; details about the STOP model are described in that work [21]. The model was not separately validated for SES and SPD subpopulations.

Subpopulation definitions

Using data from the 2018 and 2019 NHIS (See Additional File 1), we defined low SES as having at least one of the following: income < 100% of the federal poverty level, education level less than high school, or enrollment in Medicaid. We defined SPD as reporting a Kessler-6 score ≥ 13 [23, 24]. The Kessler-6 is a measure designed for population surveys to detect clinically-significant distress associated with serious mental illness [25]. In comparison with low SES and SPD subpopulations, we simulated two less marginalized, complementary subpopulations: one for people who did not meet any of the criteria for low SES (higher SES) and another for people with Kessler-6 score < 13 (non-SPD). We did not separately simulate people with both low SES and SPD because data were sparse for people belonging jointly to both subpopulations.

Analyses

Individual-level life expectancy

In this analysis, we conducted 16 model simulations of 40-year-olds wherein all individuals in each simulated cohort are homogeneous by subpopulation (low SES, higher SES, SPD, or non-SPD), gender (women or men), and smoking status (current or never smoking). For the purpose of defining mortality risks, current smoking was defined as having smoked ≥ 100 cigarettes over a lifetime and currently smoking every day or some days [22]. Never smoking was defined as having smoked < 100 cigarettes over a lifetime. To obtain stable estimates, in each stratification we simulated one million individuals over a modeled lifetime and projected life expectancy assuming no change in their smoking status nor SES/SPD status. We chose to simulate 40-year-olds at model start because tobacco-attributable mortality becomes more evident after age 40y [22]. We report life expectancy as age at time of death for ease of interpretation and comparison with other studies.

Key model input parameters and assumptions are in Table 1. We applied monthly all-cause mortality probabilities stratified by gender, age, smoking status, and subpopulation, deriving them from NHIS data and the linked mortality files. To derive all-cause mortality probabilities, we pooled NHIS data from 2004 to 2014 to obtain a large sample size. The NHIS was redesigned in 2004 [26], and 2014 was the last year prior to the 2015 Public-Use Linked Mortality Files (the latest available files when we performed the analysis) [27]. Given that data suggest that those who quit smoking retain the mortality risk of those who currently smoke for approximately 5 years following cessation, and to limit bias that could be introduced due to changes in smoking status over time, we only considered the mortality for 5 years of follow-up after the NHIS interview [22, 28, 29]. For additional details on the derivation of mortality probabilities, including the derivation of subpopulation-specific values, see Additional File 1.

To assess the uncertainty in our model projections, we performed sensitivity analyses around model input parameters, following guidance on good research practices in modeling [30]. We performed one-way sensitivity analyses and varied: (*i*, *ii*) monthly all-cause mortality probability for people who have never smoked (NS); (*iii*) all-cause mortality hazard ratios for people who currently smoke (CS) compared with NS according to upper and lower bounds of 95% confidence intervals; and (*iv*) all-cause mortality hazard ratios for CS compared with NS according to subpopulation-stratified ratios (Additional File 1).

Population-level smoking and mortality dynamics

In this second analysis, we simulated one million people aged 20y and older in each subpopulation (low SES, higher SES, SPD, or non-SPD), where each subpopulation has a specific distribution of gender, age, and current/former/never smoking status derived from the 2019 NHIS (former smoking was defined as having smoked ≥ 100 cigarettes over a lifetime but not in the last 5 years [22]) [21]. In contrast to the Individual-Level Life Expectancy analysis, we allowed smoking status to change over time (initiation, cessation, relapse) [21]. Simulated subpopulations change each year as individuals die and new 20-year-olds enter the model. Smoking prevalence among entering 20-year-olds declines yearly according to subpopulation-specific secular trends estimated from the NHIS (see Additional File 1). Monthly all-cause mortality probabilities stratified by gender, age, smoking status, and subpopulation were applied as in the base case of the first analysis (see above).

In addition to these "status quo" scenarios, we also estimated "alternate" scenarios for the marginalized subpopulations in which initiation and cessation probabilities, as well as the rate of decline in smoking prevalence for new 20-year-olds entering the simulated subpopulation, match those in the corresponding less marginalized subpopulation. We estimated the time points at which each subpopulation would reach the 5% cigarette smoking prevalence threshold under status quo scenarios and, for marginalized subpopulations, under alternate scenarios. To estimate the total life-years lost among marginalized subpopulations specifically due to disparities in smoking dynamics, we calculated the difference in model-projected life-years accumulated by the low SES and SPD subpopulations under the status quo and alternate scenarios. This difference, derived from the simulated population, was scaled to the U.S. population using weighted data from the NHIS to calculate the total life-years lost

Table 1 Model input parameters

Parameter	Value (Range in sensitivity analysis)				Source
PARAMETERS USED IN BOTH FIRST AND SECOND ANALYSES					
	Low SES	**High SES**	**SPD**	**Non-SPD**	
Examples of all-cause mortality (at age 60y) among people who never smoked, monthly probability ($\times 10^{-4}$)[a]	Women: 9.0 (95% CI 8.4–9.6) Men: 12.7 (95% CI 11.7–13.9)	Women: 4.0 (95% CI 3.7–4.3) Men: 6.5 (95% CI 6.0-7.1)	Women: 10.5 (95% CI 8.6–12.8) Men: 16.3 (95% CI 12.3–21.6)	Women: 5.0 (95% CI 4.8–5.3) Men: 7.4 (95% CI 7.0-7.9)	NHIS[b]
All-cause mortality hazard ratio, people who currently smoke vs. people who never smoked	Women: 2.4 (95% CI 2.3–2.5) Men: 2.1 (95% CI 2.0-2.3)				NHIS[b]
PARAMETERS USED ONLY IN SECOND ANALYSIS					
	Low SES	**Higher SES**	**SPD**	**Non-SPD**	
Women/men, %	55/45	51/49	61/39	51/49	NHIS[c]
Initial age, mean (SD), years	46.1 (18.2)	48.2 (18.4)	46.7 (11.1)	46.8 (11.7)	NHIS[c]
Examples of initial prevalence of never/former/current smoking, by age, %					NHIS[c]
Age 20–24y	Women: 79.8/6.4/13.8 Men: 80.2/9.2/10.6	Women: 89.3/2.8/7.9 Men: 81.6/9.4/9.0	Women: 75.1/6.6/18.2 Men: 53.4/22.7/23.9	Women: 85.7/5.1/9.1 Men: 82.4/6.3/11.3	
Age 40–44y	Women: 65.4/14.9/19.8 Men: 51.0/17.5/31.5	Women: 68.3/20.0/11.7 Men: 56.9/28.3/14.8	Women: 42.7/22.0/35.3 Men: 32.3/26.8/40.9	Women: 71.6/16.4/11.9 Men: 59.9/23.2/16.9	
Age 60–64y	Women: 50.3/17.8/31.9 Men: 44.1/24.0/31.9	Women: 61.4/27.3/11.3 Men: 49.0/35.7/15.3	Women: 47.1/31.6/21.3 Men: 36.0/26.1/37.9	Women: 61.6/26.4/12.0 Men: 53.7/31.1/15.2	
Smoking initiation probability, monthly, by age and gender[d]	0-0.007 (0.75x-1.25x)	0-0.006 (0.75x-1.25x)	0-0.008 (0.75x-1.25x)	0-0.006 (0.75x-1.25x)	NHIS and Jeon [31]
Smoking cessation probability, monthly, by age and gender[d]	0-0.072 (0.75x-2.0x)	0-0.132 (0.75x-2.0x)	0-0.065 (0.75x-2.0x)	0-0.120 (0.75x-2.0x)	NHIS and Jeon [31]
Smoking relapse probability, monthly (t = months since cessation)[e]	$0.62*e^{-0.33*t}$ (0.75x-1.25x)				[54–57]
Annual relative decrease in current smoking prevalence among 20-year-olds entering the cohort, %	-9.0	-12.1	-8.3	-10.5	NHIS[c]
All-cause mortality hazard ratio, people who formerly smoked vs. people who never smoked, by age at smoking cessation					NHIS[b], Thun 2013 [29]
<40y[f]	Women: 1 (assumption) Men: 1 (assumption)				
40–44y	Women: 1.3 Men: 1.1				
45–54y	Women: 1.4 Men: 1.3				
55–64y	Women: 1.7 Men: 1.5				

Table 1 (continued)

Parameter	Value (Range in sensitivity analysis)	Source
65-69y	Women: 1.7 Men: 1.5	
≥70y	Women: 2.2 Men: 2.1	

Abbreviations:

NHIS, National Health Interview Survey. SES, socioeconomic status. SPD, people with serious psychological distress. SD, standard deviation. CVD, cardiovascular disease. CI, confidence interval

Low SES was defined as having income < 100% of the federal poverty level, or having less than a high school education, or being a Medicaid beneficiary. SPD was defined as having a score of 13 or higher on the Kessler-6 scale

In the first analysis, all individuals in each modeled cohort are homogeneous by subpopulation (low SES, higher SES, SPD, or non-SPD), initial age (40 years), gender (women or men), and smoking status (current or never smoking)

In the second analysis, each modeled subpopulation cohort comprises individuals with a specific distribution of age, gender, and current/former/never smoking status, derived from NHIS

[a]Monthly mortality probabilities for 60-year-old people are provided as an example here. The model includes age-specific monthly probabilities for a range of ages

[b]This was derived from NHIS 2004–2014

[c]This was derived from NHIS 2019 (Low SES and Higher SES) and pooled NHIS 2009–2014 (SPD and Non-SPD) data

[d]For the sensitivity analysis around smoking initiation and cessation probabilities, multipliers (e.g., multiply by 1.25) were applied to previously derived (from NHIS 1997–2009 data) initiation and cessation probabilities that were stratified by age and gender but not by subpopulation [21].

[e]This is based on relapse probabilities reported in smoking cessation intervention trials, focusing on placebo arms

[f]We assumed no excess mortality among people who stop smoking before age 40y compared with people who never smoke [22, 29].

due to smoking among people with low SES and among people with SPD in the U.S.

To allow smoking behavior to change dynamically, we applied monthly smoking initiation, cessation, and relapse probabilities in this second analysis (Table 1). Derivations and validation of age- and gender-stratified probabilities from NHIS data are described elsewhere [21]. From these age- and gender-stratified initiation and cessation probabilities, we derived distinct sets of initiation and cessation probabilities for each of the low SES, higher SES, SPD, and non-SPD subpopulations. To do this, we adapted data from Jeon et al., [31] who used NHIS data to estimate smoking initiation and cessation probabilities by birth cohort, age, gender, and family income. Family income brackets were defined as < 100%, 100–199%, 200–299%, 300–399%, and > 400% of the federal poverty level (FPL). Jeon et al. provided us with the 1970 birth cohort datapoints (stratified by gender, age, and family income level) used to create Figs. 1 and 2 of their paper (personal communication). Due to assumed differences in a 1970 birth cohort and our modeled cohort, which was not specific to a birth year but rather to a cross-section of the population, we calculated gender-stratified ratios of smoking initiation and cessation probabilities between less marginalized and marginalized subpopulations. Based on the distribution of income levels within and across subpopulations, we assumed > 400% FPL to represent less marginalized (higher SES and non-SPD) subpopulations and 100–199% FPL to represent marginalized (low SES and SPD) subpopulations. We focused on smoking initiation and cessation probabilities at age 20 and age 45, respectively, because they are approximately representative of age groups initiating and quitting smoking in our simulations [2, 32]. We used weights to reflect the sizes of low SES vs. higher SES subpopulations and SPD vs. non-SPD subpopulations, derived from NHIS data for ages 18–20 and 40–49 (age ranges were used to improve data precision) [33, 34]. The following series of equations were adapted for each age- and gender-specific probability of smoking initiation and cessation for the low SES, higher SES, SPD, and non-SPD subpopulations, with low SES used as an example:

$$wt_{lowSES}pr_{lowSES} + wt_{higherSES}pr_{higherSES} = pr_{all}$$

$$wt_{lowSES}pr_{lowSES} + wt_{higherSES}Rpr_{lowSES} = pr_{all}$$

$$pr_{lowSES}(wt_{lowSES} + wt_{higherSES}R) = pr_{all}$$

$$pr_{lowSES} = \frac{pr_{all}}{(wt_{lowSES} + wt_{higherSES}R)}$$

where pr_x is the age- and gender-specific probability of initiation (or cessation) for *x* subgroup, wt_x is the weight of *x* subpopulation based on its relative size, R is the ratio of initiation (or cessation) probabilities among the less marginalized subgroup compared with the marginalized subgroup, and pr_{all} is the overall probability of initiation or cessation derived from the NHIS (not specific to subpopulation). Because Jeon et al. define cessation as abstinence at two years follow-up, R accounts for subpopulation differences in both quit attempts and relapse. The resulting age- and gender-stratified probabilities for smoking initiation and cessation were entered into the model for each subpopulation.

For this second analysis, we varied the following parameters in one-way sensitivity analyses (*v*): monthly

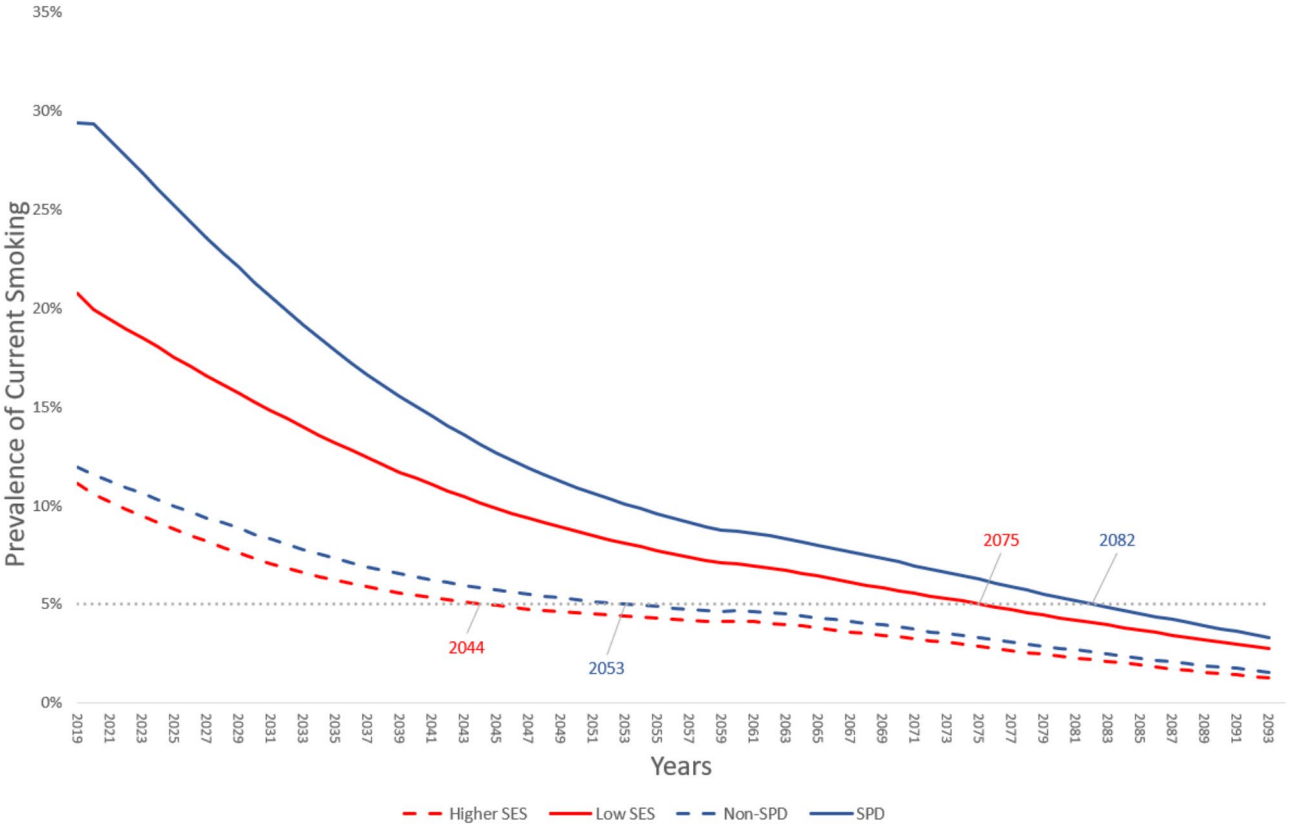

Fig. 1 Projected current cigarette smoking prevalence for marginalized and less marginalized groups in the U.S. – status quo scenario
SES, socioeconomic status; SPD, serious psychological distress

probabilities of smoking initiation, cessation, and relapse. In a multi-way sensitivity analysis (*vi*), we simultaneously varied the monthly probabilities of smoking initiation, cessation, and relapse to maximally increase or decrease smoking prevalence. For these sensitivity analyses, we used life expectancy as an outcome for comparison purposes. Also, for simplicity, we did not stratify initiation, cessation, or relapse probabilities by subpopulation because the sensitivity to these input changes would vary similarly across groups. Lastly, we performed a sensitivity analysis (*vii*) in which smoking initiation and cessation probabilities are varied between less marginalized and marginalized subpopulations using relative risks calculated from Jeon et al., [31] rather than based on ratios and population-level weighting (see Additional File 1).

Results

Individual-level life expectancy

In the first type of analysis, where we simulated 40-year-old people, smoking throughout adulthood (compared with never smoking) is associated with a greater reduction in life expectancy than is low SES (compared with higher SES) or SPD (compared with non-SPD) experienced throughout adulthood. For example, women with low SES who currently smoke have life expectancy (reported as age at death) of 70.3 years (Table 2). Their life expectancy loss associated with smoking, compared with the life expectancy of women with low SES who never smoke, is 10.1 years; their life expectancy loss associated with low SES, compared with the life expectancy of women with higher SES who currently smoke, is 7.6 years (Table 2). Similar trends are seen among men with low SES who currently smoke, women with SPD who currently smoke, and men with SPD who currently smoke – the life expectancy loss associated with smoking is greater than that associated with low SES or SPD. People with low SES and people with SPD have lower life expectancy than people with higher SES and people without SPD, respectively (Table 2).

Population-level smoking and mortality dynamics: smoking prevalence threshold

According to our second type of analysis, given recent smoking prevalence and trends (status quo), the higher SES subpopulation would reach a threshold of 5% smoking prevalence in 2044 while the low SES subpopulation would reach that threshold 31 years later (Fig. 1). People without SPD would reach the 5% smoking prevalence threshold in 2053 while those with SPD would reach that threshold 29 years later.

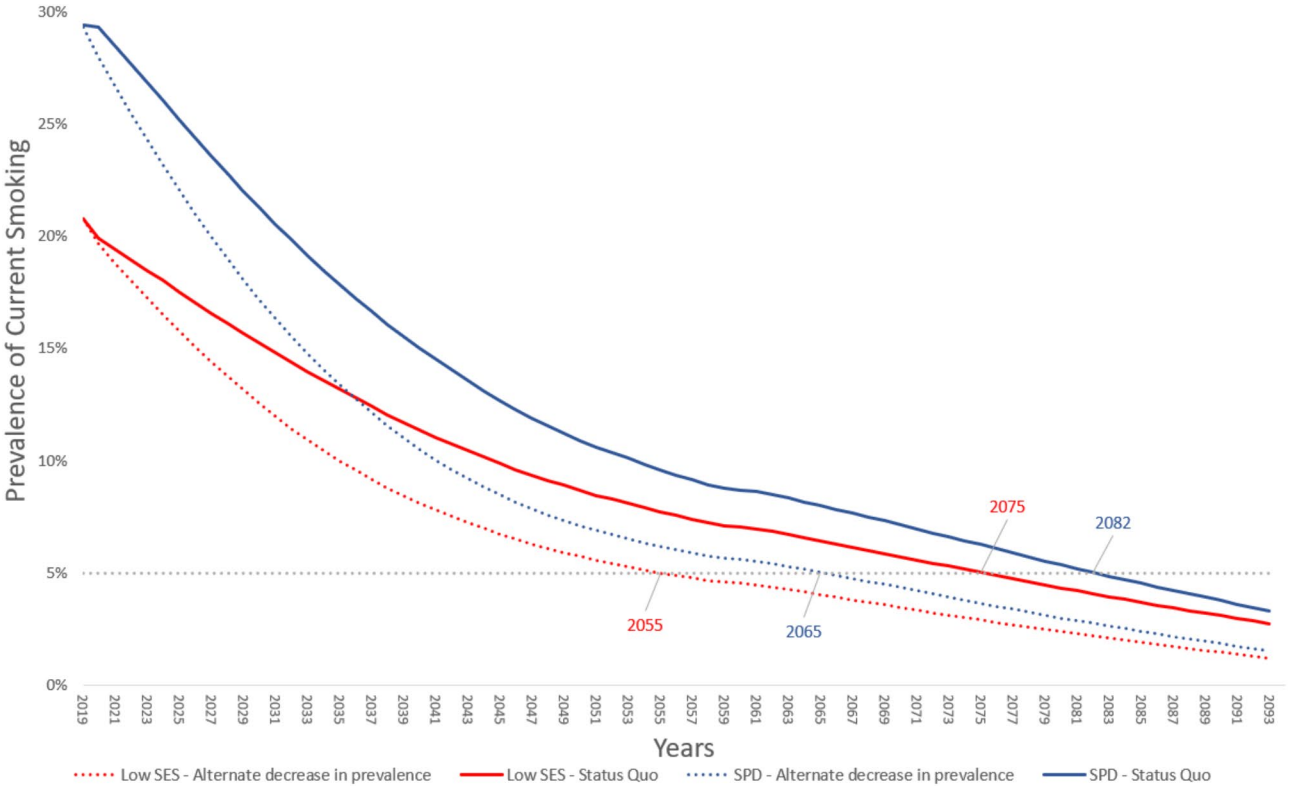

Fig. 2 Projected current cigarette smoking prevalence for marginalized groups in the U.S. in the status quo and alternate scenarios
SES, socioeconomic status; SPD, serious psychological distress. Predicted prevalences in the alternate scenarios are adjusted for the age and gender distributions of each subpopulation

Table 2 Model-projected life expectancy for 40-year-old people by gender, smoking status, and subpopulation

	Women			Men		
	Current smoking, LE, years	**Never smoking, LE, years**	***LE loss associated with lifelong smoking, years***	**Current smoking, LE, years**	**Never smoking, LE, years**	***LE loss associated with lifelong smoking, years***
Low SES	70.3	80.4	*10.1*	67.7	76.3	*8.6*
Higher SES	77.9	86.4	*8.5*	74.1	81.7	*7.6*
LE loss associated with low SES, years	*7.6*	*6.0*		*6.4*	*5.4*	
SPD	68.6	78.3	*9.7*	65.0	73.3	*8.3*
Non-SPD	75.9	84.7	*8.8*	73.0	80.8	*7.8*
LE loss associated with SPD, years	*7.3*	*6.4*		*8.0*	*7.5*	

Abbreviations: LE, life expectancy. SES, socioeconomic status. SPD, serious psychological distress

The individuals in each model simulation are homogenous by initial age (40 years), subpopulation, gender, and smoking status. In these simulations, we assumed no changes in smoking status over time, such that "current smoking" means continued smoking until death and "never smoking" means never initiating smoking. Similarly, SES and SPD were assumed unchanged from age 40 years through death

Low SES was defined as having income < 100% of the federal poverty level, or having less than a high school education, or being a Medicaid beneficiary. SPD was defined as having a score of 13 or higher on the Kessler-6 scale

Figure 2 includes the alternate scenarios, where smoking initiation and cessation probabilities, as well as the downward trends in smoking prevalence among new 20-year-olds in the low SES and SPD subpopulations, are similar to those in the higher SES and non-SPD subpopulations, respectively. Among people with low SES, the threshold of 5% smoking prevalence is reached 20 years sooner in the alternate scenario than in the status quo scenario; among people with SPD, the threshold is reached 17 years sooner in the alternate scenario.

Population-level smoking and mortality dynamics: aggregate Life-years lost

The second type of analysis also indicates that the low SES population, comprising 61.4 million people, would accrue 5.3 million (0.2%) fewer life-years over 40 years

under the status quo scenario than they would in the alternate scenario. The SPD population, comprising 8.0 million people, would accrue 966,000 (0.3%) fewer life-years over 40 years under the status quo scenario than they would in the alternate scenario.

Uncertainty analyses

We tested the sensitivity of our findings to variations in input parameters. For our Individual-Level Life Expectancy analyses, we found the following: (*i*) setting the mortality probabilities for NS to their upper and lower confidence bounds changes life expectancy estimates by approximately 1 year or less (≤1.4%) in all subpopulations except for SPD, in whom life expectancy estimates change by up to 2.6y (3.3%) for women and 3.5y (4.8%) for men (Additional File 1, Supplementary Table 1); (*ii*) when mortality probabilities for NS increase or decrease by 10%, life expectancy changes by ≤1.2y (≤1.6%) compared with the base case in all subpopulations (Additional File 1, Supplementary Table 2); (*iii*) when mortality multipliers for CS (hazard ratios vs. NS) are set at their upper and lower confidence bounds, life expectancy estimates vary by ≤0.7y (≤1.1%) (Additional File 1, Supplementary Table 3); and (*iv*) when subpopulation-stratified mortality multipliers for CS (hazard ratios vs. NS) are applied, life expectancy estimates vary by ≤3.1y (≤4.2%) (Additional File 1, Supplementary Table 4).

For our second type of analysis (Population-Level Smoking and Mortality Dynamics), we found that (*v*) increasing or decreasing the smoking initiation, cessation, or relapse rates by 25% changes life expectancies by ≤0.3y (Additional File 1, Supplementary Table 5). In a multi-way sensitivity analysis (*vi*) in which initiation, cessation, and relapse are simultaneously varied by 25% in a manner to either maximize or minimize the prevalence of CS, life expectancies change by ≤0.6y (Additional File 1, Supplementary Table 6). Note that for simplicity, these two sensitivity analyses used models where initiation, cessation, and relapse probabilities were not stratified by subpopulation, but the results would be similar if they had been. In the sensitivity analysis utilizing relative risks of smoking initiation and cessation without weighting by subpopulation size (*vii*), the difference in time needed to reach 5% smoking prevalence increases by 8 years between low SES and higher SES subpopulations and 11 years between SPD and non-SPD subpopulations (Additional File 1, Supplementary Fig. 1).

Discussion

Using a simulation model of cigarette smoking, we projected substantial ongoing and future disparities in smoking-associated health outcomes between U.S. marginalized groups at high risk for tobacco smoking and their less marginalized counterparts. At an individual level, we find that smoking is associated with a major loss in life expectancy for people with low SES and people with SPD, even greater than the loss in life expectancy associated with low SES or SPD themselves. At a population level, given current trends a 5% cigarette smoking prevalence threshold will be reached much later in marginalized compared to less marginalized subpopulations. Together, these findings show that the delay in reaching this threshold will result in millions of life-years lost for marginalized subpopulations compared to an alternate scenario with no disparity in smoking dynamics due to SPD or low SES.

Our results are generally robust to changes in model assumptions and statistical uncertainty. Because of wider NHIS-derived confidence intervals for never smokers' mortality rates among people with SPD, their life expectancy estimates varied more than those of other subpopulations in sensitivity analysis. When we simultaneously varied initiation, cessation, and relapse probabilities by 25% in directions to maximally increase or decrease smoking prevalence, life expectancy across the scenarios for people with SPD vary by up to 0.6y, whereas life expectancy vary by up to 0.4y in the other three subpopulations. For SPD and non-SPD subpopulations, our estimates of the individual-level life expectancy effects of tobacco use are similar to previously published estimates, differing by no more than 9% [35]. Similar comparisons for the SES subpopulations are not available.

Our projections may be considered alongside recent estimates of future smoking prevalence and life-years lost due to smoking by SPD status. Xi et al. estimated that through 2100, smoking prevalence among individuals with SPD will fall no lower than 10.7% absent changes to the status quo [36]. However, they assume for modeling simplicity that youth smoking initiation will remain constant, whereas we assume it will continue to decline (as reflected in smoking prevalence among 20-year-olds entering the simulated cohort each year) as it has for many years [37]. Under a "maximum potential reduction in premature mortality" scenario (i.e., all smoking initiation and current smoking ceases in 2024), those authors find 3.1 million life-years would be saved through 2100. Our alternate modeling scenario focuses on a different question, estimating the potential benefits of improving marginalized populations' smoking dynamics (initiation, cessation) to the point where they equal those of less marginalized groups, and finds comparable impact with 966,000 life-years projected to be saved over 40 years for people with SPD compared to people without SPD. Again, similar estimates by SES are not available. Other studies report population-level life-years lost due to smoking or gained from tobacco control interventions in the U.S. general population. Holford et al. estimated the impact of U.S. tobacco control efforts over the nearly

50 years following the first Surgeon General's report on smoking in 1964, finding that tobacco control efforts over that period resulted in 157 million life-years saved [3]. A more narrowly focused study by Tam et al. estimated the potential impact of cigarette package graphic warning labels on population health through 2100, finding that such a policy change would likely result in 7.9 million life-years gained [38]. Although focused on policy changes rather than sociodemographic differences in smoking dynamics, these two studies support the face validity of our estimates of the impact of SES- and SPD-associated tobacco use. Additionally, those studies show that policy changes can prevent adverse population health outcomes of the sort we estimate.

Despite the U.S. Department of Health and Human Services' goal of achieving 5% cigarette smoking prevalence by 2030, there are few reports projecting whether the U.S. is on a trajectory to achieve that target. Since 1999, the prevalence of current cigarette smoking in the U.S. has declined by approximately 0.5% points per year [39, 40]. If this trend continues, the smoking threshold of 5% would be reached in approximately 2034. However, such a rough calculation does not adjust for evolving population demographics, including trends that may result in "hardening," the term for what would happen if tobacco use concentrates increasingly among those who have the most difficulty quitting, a particular concern among marginalized groups [41].

As the public health community looks toward the tobacco endgame, our results demonstrate that there is not a single tobacco endgame, but many tobacco endgames for different subpopulations affected by smoking. We focused on individuals with low SES and with SPD because they are, respectively, the largest subpopulation with significantly elevated smoking prevalence and a subpopulation with very high smoking prevalence [9–11, 16–18, 42]. Despite the SPD subpopulation being only 13% the size of the low SES subpopulation, the projected life-years lost associated with smoking in the SPD group is 18% the estimate for the low SES group. While these subpopulations have important overlap which we do not assess here due to data limitations, it is clear that the harm of smoking is particularly acute in the subpopulation with SPD and potentially even more so in those with both low SES and SPD. We focused on cigarette smoking given data availability, but reaching a comprehensive tobacco endgame will require a reduction in the use of other tobacco products, as well.

Narrowing the disparity in tobacco use between marginalized and less marginalized populations will require significant, concerted, and often targeted effort. *Healthy People 2030* lays out several policy goals to reduce the harms of tobacco use, including advocacy for increasing tobacco taxes and wider prohibitions of smoking in multiunit housing [43]. Disparities may also be reduced through improved logistical and financial access to tobacco treatment. Generous Medicaid coverage for smoking cessation treatments has been shown to reduce smoking prevalence and heart disease among beneficiaries with low SES who smoke [40, 44]. Adverse social determinants of health are common among those with low SES and those with SPD; [45] studies have shown that addressing social determinants of health as part of a tobacco treatment initiative can improve cessation [46–49]. Further, state tobacco quitlines that tailor their messaging to callers with mental health conditions have shown promise [50].

Our estimates of the impact of tobacco-related health disparities are based on a microsimulation model that has been validated relative to overall historical patterns of tobacco use in the US general adult population [21]. However, it is uncertain if these historical trends will continue or if they match those of subpopulations defined by SES or mental health. Data about smoking initiation, cessation, relapse, and mortality probabilities came from slightly different NHIS periods due to data availability and the need to aggregate some data across years to improve precision. We consider low SES and SPD to be stable characteristics in our modeling populations, although these are non-permanent states for many individuals. Despite this being cited as a limitation in other modeling analyses as well, we sought to minimize the impact of this limitation on mortality risk estimates by generating these estimates using mortality data only within five years of the low SES or SPD status determination [35, 36]. Our analyses focus on cigarette smoking. To the extent that reductions in cigarette smoking prevalence are offset by other combusted tobacco use – particularly if this occurs disproportionately in the marginalized groups – we will have underestimated the outcomes disparities. Finally, although our smoking-associated mortality hazard ratios were adjusted for several factors as in a prior study, there remains the possibility of unmeasured confounding of the relationship between smoking and mortality [29]. Nonetheless, the association between smoking and mortality has been extensively studied and documented [22, 29, 51–53].

Conclusions

In summary, we found that, while tobacco use continues to decline, existing disparities in cigarette smoking portend substantial ongoing disparities in health outcomes, including life expectancy, well past the point at which less marginalized subpopulations have reached a surrogate of the tobacco endgame goal. Effective strategies to reduce disparities in tobacco use exist, but success will depend on multipronged efforts in the policy and clinical spaces that prioritize marginalized subpopulations.

Ultimately, reducing tobacco-related disparities in the U.S. will require an explicitly equity-focused vision and the tobacco endgame will only be truly achieved when it includes all groups.

Supplementary Information

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Supplementary Material 1

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Author contributions
DEL conceived the study, co-led the project, and drafted the manuscript. SSL, YQ, FMS, NMM, and NKA obtained data and executed the analyses. SLG obtained data and assisted with data visualization. JEB and EPH contributed expertise on study populations. KPR developed the simulation model, co-led the project, and drafted the manuscript. All authors revised, read, and approved the manuscript.

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Data availability
Data inputs were derived or obtained from publicly available data or published sources and are detailed with citations in the manuscript and supplement.

Declarations

Ethics approval and consent to participate
This simulation model-based research was approved by the Mass General Brigham Human Research Committee (Protocol #2019P001772). This study used publicly available data.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. U.S. Department of Health and Human Services. The Health Consequences of Smoking -- 50 Years of Progress: A Report of the Surgeon General. Rockville, MD; 2014.
2. VanFrank B, Malarcher A, Cornelius ME, Schecter A, Jamal A, Tynan M. Adult smoking Cessation - United States, 2022. MMWR Morb Mortal Wkly Rep. 2024;73(29):633–41.
3. Holford TR, Meza R, Warner KE, Meernik C, Jeon J, Moolgavkar SH, et al. Tobacco control and the reduction in smoking-related premature deaths in the united States, 1964–2012. JAMA. 2014;311(2):164–71.
4. Bhatnagar A, Whitsel LP, Blaha MJ, Huffman MD, Krishan-Sarin S, Maa J, et al. New and emerging tobacco products and the nicotine endgame: the role of robust regulation and comprehensive tobacco control and prevention: A presidential advisory from the American heart association. Circulation. 2019;139(19):e937–58.
5. Compton WM, Einstein EB. Tobacco use in High-Risk populations. JAMA Netw Open. 2020;3(3):e200474.
6. Cornelius ME, Wang TW, Jamal A, Loretan CG, Neff LJ. Tobacco product use among adults—United States, 2019. MMWR Morb Mortal Wkly Rep. 2020;69(46):1736.
7. Hitchman SC, Fong GT, Zanna MP, Thrasher JF, Chung-Hall J, Siahpush M. Socioeconomic status and smokers' number of smoking friends: findings from the international tobacco control (ITC) four country survey. Drug Alcohol Depend. 2014;143:158–66.
8. National Cancer Institute Tobacco Control Monograph 22. A socioecological approach to addressing tobacco-related health disparities. NIH publication no. 17-CA-8035A. Bethesda, MD: US Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 2017.
9. U.S. Census Bureau. Educational Attainment in the United States: 2019. 2020. Available from: https://www.census.gov/data/tables/2019/demo/educational-attainment/cps-detailed-tables.html
10. Medicaid, Payment CHIP, and Access Commission. Changes in MedicaidCHIP Enrollment. MACPAC; 2019. November 2019.
11. Semega J, Kollar M, Shrider EA, Creamer J. Income and poverty in the United States: 2019 (Report P60–270). US Census Bureau, Current Population. 2020.
12. The Truth Initiative. Tobacco is a social justice issue: Low-income communities. 2017. Available from: https://truthinitiative.org/research-resources/targeted-communities/tobacco-social-justice-issue-low-income-communities
13. The Truth Initiative. Why are 72% of smokers from lower-income communities? 2018. Available from: https://truthinitiative.org/research-resources/targeted-communities/why-are-72-smokers-lower-income-communities
14. Cornelius ME, Loretan CG, Jamal A, Davis Lynn BC, Mayer M, Alcantara IC, et al. Tobacco product use among Adults - United States, 2021. MMWR Morb Mortal Wkly Rep. 2023;72(18):475–83.
15. National Institute on Drug Abuse. Do people with mental illness and substance use disorders use tobacco more often? 2023. Available from: https://nida.nih.gov/publications/research-reports/tobacco-nicotine-e-cigarettes/do-people-mental-illness-substance-use-disorders-use-tobacco-more-often
16. McGinty EE, Presskreischer R, Han H, Barry CL. Psychological distress and loneliness reported by US adults in 2018 and April 2020. JAMA. 2020;324(1):93–4.
17. Jamal A, King BA, Neff LJ, Whitmill J, Babb SD, Graffunder CM. Current cigarette smoking among adults—United States, 2005–2015. Morbidity and mortality weekly report. 2016;65(44):1205–11.
18. Wang TW, Asman K, Gentzke AS, Cullen KA, Holder-Hayes E, Reyes-Guzman C, et al. Tobacco product use among Adults - United States, 2017. MMWR Morb Mortal Wkly Rep. 2018;67(44):1225–32.
19. Dickerson F, Schroeder J, Katsafanas E, Khushalani S, Origoni AE, Savage C, et al. Cigarette smoking by patients with serious mental illness, 1999–2016: an increasing disparity. Psychiatr Serv. 2018;69(2):147–53.
20. U.S. Department of Health Human Services. Reduce current cigarette smoking in adults—TU-02. Available from: https://health.gov/healthypeople/objec

tives-and-data/browse-objectives/tobacco-use/reduce-current-cigarette-smoking-adults-tu-02
21. Reddy KP, Bulteel AJB, Levy DE, Torola P, Hyle EP, Hou T, et al. Novel microsimulation model of tobacco use behaviours and outcomes: calibration and validation in a US population. BMJ Open. 2020;10(5):e032579.
22. Jha P, Ramasundarahettige C, Landsman V, Rostron B, Thun M, Anderson RN, et al. 21st-century hazards of smoking and benefits of cessation in the united States. N Engl J Med. 2013;368(4):341–50.
23. Kessler RC, Andrews G, Colpe LJ, Hiripi E, Mroczek DK, Normand SL, et al. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol Med. 2002;32(6):959–76.
24. Prochaska JJ, Sung HY, Max W, Shi Y, Ong M. Validity study of the K6 scale as a measure of moderate mental distress based on mental health treatment need and utilization. Int J Methods Psychiatr Res. 2012;21(2):88–97.
25. Kessler RC, National Comorbidity Survey K. and K6. Available from: https://www.hcp.med.harvard.edu/ncs/k6_scales.php
26. National Center for Health Statistics. National Health Interview Survey 2004, Data Release 2004. Available from: https://archive.cdc.gov/www_cdc_gov/nchs/nhis/nhis_2004_data_release.htm
27. National Center for Health Statistics. National Health Interview Survey 2014 Data Release. 2014.
28. Reddy KP, Parker RA, Losina E, Baggett TP, Paltiel AD, Rigotti NA, et al. Impact of cigarette smoking and smoking cessation on life expectancy among people with HIV: A US-Based modeling study. J Infect Dis. 2016;214(11):1672–81.
29. Thun MJ, Carter BD, Feskanich D, Freedman ND, Prentice R, Lopez AD, et al. 50-year trends in smoking-related mortality in the united States. N Engl J Med. 2013;368(4):351–64.
30. Briggs AH, Weinstein MC, Fenwick EA, Karnon J, Sculpher MJ, Paltiel AD, et al. Model parameter Estimation and uncertainty: a report of the ISPOR-SMDM modeling good research practices task Force–6. Value Health. 2012;15(6):835–42.
31. Jeon J, Cao P, Fleischer NL, Levy DT, Holford TR, Meza R, et al. Birth Cohort–Specific smoking patterns by family income in the U.S. Am J Prev Med. 2023;64(4 Suppl 1):S32–41.
32. Ali FRM, Agaku IT, Sharapova SR, Reimels EA, Homa DM. Onset of regular smoking before age 21 and subsequent nicotine dependence and cessation behavior among US adult smokers. Prev Chronic Dis. 2020;17:E06.
33. National Center for Health Statistics. National Health Interview Survey. 2018. Available from: https://archive.cdc.gov/www_cdc_gov/nchs/nhis/nhis_2018_data_release.htm
34. National Center for Health Statistics. National Health Interview Survey. 2019. Available from: https://www.cdc.gov/nchs/nhis/documentation/2019-nhis.html
35. Tam J, Warner KE, Meza R. Smoking and the reduced life expectancy of individuals with serious mental illness. Am J Prev Med. 2016;51(6):958–66.
36. Xi Q, Meza R, Leventhal A, Tam J. Modeling cigarette smoking disparities between people with and without serious psychological distress in the US, 1997–2100. Prev Med. 2023;166:107385.
37. Jamal A, Park-Lee E, Birdsey J, West A, Cornelius M, Cooper MR, et al. Tobacco product use among middle and high school Students - National youth tobacco survey, united States, 2024. MMWR Morb Mortal Wkly Rep. 2024;73(41):917–24.
38. Tam J, Jeon J, Thrasher JF, Hammond D, Holford TR, Levy DT, et al. Estimated prevalence of smoking and smoking-Attributable mortality associated with graphic health warnings on cigarette packages in the US from 2022 to 2100. JAMA Health Forum. 2021;2(9):e212852.
39. Centers for Disease Control and Prevention. Cigarette smoking among adults–United States, 1999. MMWR Morb Mortal Wkly Rep. 2001;50(40):869–73.
40. Centers for Disease Control and Prevention, TOBACCO PRODUCT USE AMONG ADULTS—United States. 2022; 2022 National Health Interview Survey (NHIS) Highlights 2022. Available from: https://www.cdc.gov/tobacco/media/pdfs/2024/09/cdc-osh-ncis-data-report-508.pdf
41. Hughes JR. An update on hardening: A qualitative review. Nicotine Tob Res. 2020;22(6):867–71.
42. Cornelius ME, Loretan CG, Wang TW, Jamal A, Homa DM. Tobacco product use among Adults - United States, 2020. MMWR Morb Mortal Wkly Rep. 2022;71(11):397–405.
43. U. S. Department of Health and Human Services. Healthy People 2030 Topics & Objectives: Tobacco Use 2020. Available from: https://health.gov/healthypeople/objectives-and-data/browse-objectives/tobacco-use
44. Land T, Warner D, Paskowsky M, Cammaerts A, Wetherell L, Kaufmann R, et al. Medicaid coverage for tobacco dependence treatments in Massachusetts and associated decreases in smoking prevalence. PLoS ONE. 2010;5(3):e9770.
45. Blumenthal DS. Barriers to the provision of smoking cessation services reported by clinicians in underserved communities. J Am Board Family Med. 2007;20(3):272–9.
46. Curry SJ, Ludman EJ, Graham E, Stout J, Grothaus L, Lozano P. Pediatric-Based smoking cessation intervention for Low-Income women: A randomized trial. Arch Pediatr Adolesc Med. 2003;157(3):295–302.
47. Evins AE, Cather C, Maravic MC, Reyering S, Pachas GN, Thorndike AN, et al. A pragmatic Cluster-Randomized trial of provider education and community health worker support for tobacco cessation. Psychiatr Serv. 2023;74(4):365–73.
48. Haas JS, Linder JA, Park ER, Gonzalez I, Rigotti NA, Klinger EV, et al. Proactive tobacco cessation outreach to smokers of low socioeconomic status: a randomized clinical trial. JAMA Intern Med. 2015;175(2):218–26.
49. Okuyemi KS, James AS, Mayo MS, Nollen N, Catley D, Choi WS, et al. Pathways to health: A cluster randomized trial of nicotine gum and motivational interviewing for smoking cessation in Low-Income housing. Health Educ Behav. 2007;34(1):43–54.
50. Morris CD, Lukowski AV, Vargas-Belcher RA, Ylioja TE, Nash CM, Bailey LA. Quitline programs tailored for mental health: initial outcomes and feasibility. Am J Prev Med. 2021;60(3):S163–71.
51. Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British Doctors. BMJ. 2004;328(7455):1519.
52. Pirie K, Peto R, Reeves GK, Green J, Beral V, Million Women Study C. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. Lancet. 2013;381(9861):133–41.
53. Sakata R, McGale P, Grant EJ, Ozasa K, Peto R, Darby SC. Impact of smoking on mortality and life expectancy in Japanese smokers: a prospective cohort study. BMJ. 2012;345:e7093.
54. Brendryen H, Kraft P. Happy ending: a randomized controlled trial of a digital multi-media smoking cessation intervention. Addiction. 2008;103(3):478–84. discussion 85–6.
55. Bullen C, Howe C, Laugesen M, McRobbie H, Parag V, Williman J, et al. Electronic cigarettes for smoking cessation: a randomised controlled trial. Lancet. 2013;382(9905):1629–37.
56. Hughes JR, Keely J, Naud S. Shape of the relapse curve and long-term abstinence among untreated smokers. Addiction. 2004;99(1):29–38.
57. Tonstad S, Tonnesen P, Hajek P, Williams KE, Billing CB, Reeves KR, et al. Effect of maintenance therapy with varenicline on smoking cessation: a randomized controlled trial. JAMA. 2006;296(1):64–71.

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