Predictors of the Onset of Cigarette Smoking

A Systematic Review of Longitudinal Population-Based Studies in Youth

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Context: The onset of cigarette smoking typically occurs during childhood or early adolescence. Nicotine dependence symptoms can manifest soon after onset, contributing to sustained, long-term smoking. Previous reviews have not clarified the determinants of onset.

Evidence acquisition: In 2015, a systematic review of the literature in PubMed and EMBASE was undertaken to identify peer-reviewed prospective longitudinal studies published between January 1984 and August 2015 that investigated predictors of cigarette smoking onset among youth aged <18 years who had never smoked.

Evidence synthesis: Ninety-eight conceptually different potential predictors were identified in 53 studies. An increased risk of smoking onset was consistently (i.e., in four or more studies) associated with increased age/grade, lower SES, poor academic performance, sensation seeking or rebelliousness, intention to smoke in the future, receptivity to tobacco promotion efforts, susceptibility to smoking, family members' smoking, having friends who smoke, and exposure to films, whereas higher self-esteem and high parental monitoring/supervision of the child appeared to protect against smoking onset. Methodologic weaknesses were identified in numerous studies, including failure to account for attrition or for clustering in samples, and misidentification of potential confounders, which may have led to biased estimates of associations.

Conclusions: Predictors of smoking onset for which there is robust evidence should be considered in the design of interventions to prevent first puff in order to optimize their effectiveness. Future research should seek to define onset clearly as the transition from never use to first use (e.g., first few puffs).
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Context

Legislation, smoking bans, taxation, and public health tobacco control campaigns likely underpin marked declines in smoking prevalence in all age groups over the past decade.[1] However, national surveillance data suggest that the rate of decline in cigarette smoking among adolescents and young adults has slowed considerably.[2] Several recent reviews reinforce that the impact of school-based and other types of targeted prevention programs are often short-term,[1,3] and some studies suggest that such prevention efforts may have unanticipated negative effects.[4,5] These observations may reflect a lack of comprehensive understanding of the factors associated with onset such that tobacco control interventions are not conceptualized optimally.

Acquisition of sustained cigarette smoking is a complex progression, with a major early milestone being the first few puffs, typically occurring during childhood or early adolescence.[1] For many years, it was assumed that smoking acquisition proceeded through predictable

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stages,[6,7] and that nicotine dependence developed only after a period of moderately heavy daily or regular smoking,[8] so that the time window for intervention to prevent long-term smoking was extended. This assumption is perpetuated in the DSM-V criteria for diagnosing tobacco withdrawal, which require daily use of tobacco for at least several weeks.[9] Yet, considerable recent research has demonstrated that nicotine dependence symptoms can manifest soon after onset in some adolescents, often well before daily or even regular smoking,[10–14] and that early onset predicts long-term adult smoking.[15] It is not yet possible for youth themselves or their caretakers to distinguish those who, after first puff, will progress to sustained smoking from those who will maintain control over smoking with the ability to stop if they so desire. Thus, the need to prevent first puff is compelling.

The literature on predictors of smoking onset includes numerous reviews, beginning with the 1994 Surgeon General's Report on Tobacco and Health, which concluded that smoking initiation is largely determined by psychological and social factors, although only "social stimulation" was cited as a consistent element in adolescents' "early and first experiments with smoking."[16] More recently, Mayhew et al.[7] reviewed 11 cross-sectional and 33 prospective studies that examined predictors of five major stages of adolescent smoking —"non-smoking/contemplation or preparation," "tried," "experimenter," "regular," and "established/daily smoker"—the second of which represents initiation. They found that tolerance for antisocial or deviant behavior, being female with smoking parents, and parental approval of smoking were related to "onset," but described the supporting studies as related to the transition from never smoking to experimentation. Because their definition of "tried" included youth who smoked up to two cigarettes in their lifetime and who reported that they had "tried and quit," it is unclear whether these factors predict the point of transition from never smoker to initiator.

Twelve reviews investigated the relationship between smoking onset and single classes of predictors, such as sex differences,[17] genetic polymorphisms,[18–21] smokers in the household,[22] exposure to secondhand smoke,[23] or exposure to tobacco promotion efforts and smoking in films.[24–28] One additional review examined predictors of smoking initiation in young adults,[29] whereas a second examined the links between a number of factors and smoking (not simply onset) in all age groups.[30]

It is difficult to synthesize findings across these reviews. Variation in the definition of smoking onset is a challenge, ranging from "tried only a puff or one or two cigarettes," "smoking a first whole cigarette," "smoking fairly regularly," "smoked at least 100 cigarettes in lifetime," to "age at onset of daily smoking." These definitions in fact represent distinct "milestones" in the smoking onset process that occur at different time points after first puff. For example, smoking a whole cigarette is estimated to occur 2.5 months after first puff, weekly smoking and lifetime total of 100 cigarettes occur at 19 months, and daily smoking at 23 months.[13] It is likely that some or all of these milestones have different sets of predictors.

Beyond the definition issue, with the exception of two Cochrane reviews,[31,32] all systematic reviews to date include cross-sectional studies. Although associations can be detected in cross-sectional designs, they usually preclude establishing a temporal sequence between exposure and smoking onset, a fundamental criterion for causal inference. Numerous longitudinal investigations, though able to address temporal sequence, did not exclude ex-smokers from their baseline sample or analysis so that predictors of onset cannot be identified precisely. Moreover, several reviews included studies with clinic-based samples, the results of which may not be relevant to population-based samples, or they included studies conducted in populations aged 18 years or older so that predictors of early onset are obscured with predictors of later onset. Indeed, in their general review of factors related to smoking, Mak and colleagues[30] lamented the state of the literature, citing "idiosyncratic and fragmentary data" that lacked generalizability beyond the studied populations, and called on researchers to investigate the links between these factors and smoking status prospectively.

This review addresses these issues by focusing on predictors of smoking onset among baseline never smokers in prospective longitudinal population-based studies. Onset was defined as the transition from never smoking (i.e., not even a puff or a few puffs) to any smoking, ranging from the first few puffs to daily smoking.

Evidence Acquisition

Searches were conducted in PubMed and EMBASE using Medical Subject Headings and text keywords *smoking* or *tobacco* and *initiation* or *first puff* or *start* and *longitudinal* or *prospective* or *cohort*, limited to studies in humans aged <18 years, published in English in peer-reviewed journals between January 1, 1984, and August 15, 2015. These searches yielded a total of 4,260 titles after removal of duplicates and conference abstracts. To identify additional titles once the eligibility screening was complete, the "related citations" feature in PubMed was used to search the 50 most relevant related citations of seven randomly selected articles from the titles retained for data abstraction. Figure 1 presents the Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow diagram[33] describing how many titles were retained as eligible.

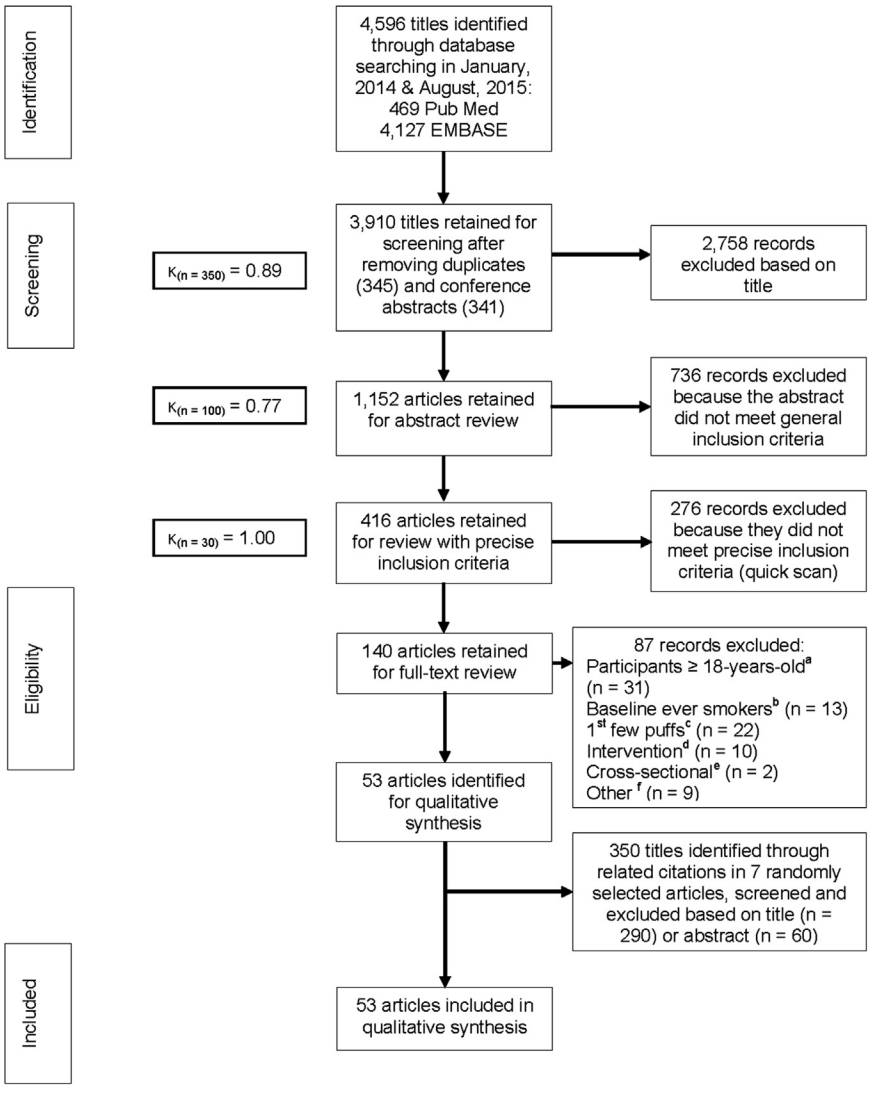

Figure 1. PRISMA flow diagram.

Note: Kappa coefficients are based on random samples of approximately 10% of titles identified at each stage.

[a]Participants >18 years old included in analyses with no sub-group analyses for younger adolescents.

[b]Baseline ever smokers were not clearly excluded from the analyses.

[c]Outcome measure did not capture first few puffs (e.g., smoking a single cigarette, smoking in the past week or month, current smoking, etc.).

[d]Both intervention and control groups were included in analyses with no indication that the intervention did not affect smoking initiation.

[e]Analyses were not entirely longitudinal (i.e., variables measured at the same time point as the outcome were used to predict the outcome).

[f]Other methodologic considerations included lack of measures of statistical significance, outcomes other than smoking initiation, or lack of measures of baseline exposure. More than one reason represents manuscripts that were rejected based on more than one criterion (Appendix 1, available online). Studies rejected for more than one reason (n=3) are counted only under the first category for which they qualify.

The screening process described in Figure 1 involved four steps, each involving two or three reviewers:

1. review of title;
2. review of abstract;
3. review of Methods section; and
4. full-text review.

Inclusion criteria were:

1. longitudinal or prospective study design (i.e., no cross-sectional or retrospective assessments);
2. population-based sample (i.e., not drawn solely from a treatment setting);
3. sample included children or adolescents (i.e., aged <18 years) or, if the sample covered a broader age range, results for youth in the target age range were easily distinguishable through subanalyses;
4. the outcome of interest was smoking onset, defined as the transition from never having smoked, even a few puffs, to having smoked at the next follow-up, regardless of the length of time between follow-up assessments; and
5. the study used a quantitative approach (but was not a meta-analysis).

Studies that used samples drawn from intervention trials were included only if participants in the control group were used in the analysis or, if the intervention was reported to have no statistically significant effect, then data for participants in both the control and treatment groups were included. Articles that reported qualitative data exclusively or comprised a systematic review or a methodologic study were excluded. As the intent was to investigate a discrete outcome (i.e., smoking onset), investigations of smoking trajectories in which onset was often not explicitly identifiable were also excluded. Inter-rater reliability was assessed in random samples of approximately 10% of titles at several steps in the selection process; kappa coefficients are reported in Figure 1. Disagreements between reviewers over eligibility at each step were resolved in team discussions, and final decisions were reached by consensus. Articles rejected at the full-text review are presented in Appendix 1 (available online). Articles rejected at earlier phases are available upon request to the authors. The final search yielded 53 articles for data extraction.[34–86]

Because the review was limited to longitudinal prospective studies not involving interventions, several elements of the Population Intervention Comparison Outcome (PICO) model[87,88] did not apply. Following guidelines suggested by Grimshaw,[89] two reviewers extracted data from each article concerning:

1. *population*: sampling frame (e.g., school-based, household-based), sampling method (e.g., representative sample, convenience sample), sample size for the analyses to identify predictors of onset, participant age(s)/grade(s) at baseline, baseline participation rate and/or attrition (%) if reported, whether there were any exclusions other than restricting the sample to never smokers;
2. *setting and design*: study location, time frame (i.e., calendar year(s) during which the study took place), length of follow-up, number of surveys after the baseline survey (i.e., only those follow-up surveys included in the smoking onset analyses);
3. *statistical analyses*: the main analytic method(s) used, list of variables adjusted for in the final models, threshold of statistical significance different than 0.05, and if sex-specific analysis were conducted; and
4. *results*: whether multivariate results or only univariate results were reported, number and percentage of initiators.

In studies that assessed mediation effects (via structural equation models), only the association related to the direct effect of the predictors on smoking onset were considered. Articles that included the same study population were identified to ensure that results for a single cohort were not presented multiple times.

Potential predictors were sorted according to six broad categories, including sociodemographic factors, personal/psychological factors, smoking-related cognitions, social factors, environmental factors, and other factors. The review was focused on main effects; findings pertaining to interactions were not considered unless studies reported only interactions.

Evidence Synthesis

The 53 articles retained for review pertained to 36 unique cohorts, 25 of which were described in single articles and 11 that were described in 28 articles. Twenty-nine of the 53 studies were conducted in the U.S., nine in the Netherlands, four in Germany, two in mainland China, two in Sweden, two in Canada, and one each in Britain, Denmark, Jordan, Mexico, and Taiwan. Most cohorts (n=28) were selected to represent the population from which they were drawn, whereas the remainder utilized convenience samples. Most cohorts (n=29) were recruited from schools, two were from national and two from statewide household telephone lists, one was identified from statewide birth records, one was drawn from a citywide pool of households, and one included a random sample selected from the newborn discharge lists of two hospitals in a single region of one state. Appendix 2 (available online) describes selected study characteristics of the 53 articles retained for analysis.

Follow-up duration ranged from 6 months to 17 years; 20 studies lasted between 1 year and 23 months, 11 between 2 years and 35 months, nine between 3 years and 47 months, six were shorter than 1 year, and seven were longer than 4 years. Only five studies reported results at intermediate follow-up points. All studies used self-report questionnaires to measure exposures and outcome. There was wide variability across studies in the selection, definition, and methods of measuring potential predictors of smoking onset. A total of 98 conceptually different potential predictors were identified, almost all of which were examined in multivariate analyses. Thirty-six studies employed a form of logistic regression as the primary analytic method; five used general linear models or generalized estimating equations, three each used Poisson regression or Cox/survival analysis, two used maximum likelihood log-linear analysis of variance, two used structural equation modeling, and one each used multilevel modeling and cross-tab analyses. Most studies entered covariates simultaneously into the predictive models. Only 12 studies reported on a statistical method for correcting variance attributable to clustering of participants within larger units, such as schools.

Only 30 studies reported the results of attrition analysis comparing participants retained and lost on key characteristics. Missing data were handled in a variety of ways: 11 studies excluded participants (i.e., used a complete case design); seven studies adjusted estimates statistically; five studies imputed missing data, one via hot-deck, three via maximum likelihood, and one via observation carry forward/backward methods. Thirty studies did not report on a method for dealing with missing data.

Four studies reported results for each predictor separately by sex and one reported an interaction by sex for one predictor. Similarly, four studies reported results separately by race/ethnicity.

Table 1 provides a summary of the 73 predictors found to be statistically significantly associated with onset in at least one study, and Appendix 3 (available online) presents a detailed description of the results for all 98 potential predictors investigated in the 53 included studies. In the following paragraphs, the number of studies in the denominator for direction of association represents only studies in which the direction of a statistically significant association was clearly reported. To assess the consistency of the evidence for individual predictors, the direction of the association was examined for predictors that were investigated and found to be significant in a minimum of four studies.

Sociodemographic factors were investigated in 81 analyses, 39 of which reported statistically significant associations with onset. Positive relationships with onset were found for age (10/16 studies) and grade (5/7 studies), whereas an inverse relationship was found between onset and SES (4/6 studies). Two sociodemographic variables that yielded discordant findings were sex and ethnicity. In ten of 29 studies, sex was significantly related to onset; female participants were found to be at greater risk of onset in three, whereas male participants were found to be at greater risk in seven. Finally, six of 13 studies found a statistically significant association between race/ethnicity and onset; whites were more likely to initiate in four studies and non-whites were more likely to initiate in two.

Personal/psychological factors were investigated in 54 analyses, 41 of which reported statistically significant associations with onset. The most frequently examined psychological variables pertained to the personality traits/temperamental characteristics of impulsivity, novelty seeking, sensation seeking, risk taking, or rebelliousness, of which only novelty seeking (examined in one study) was not associated with onset. Rebelliousness (7/7 studies) and sensation seeking (9/9 studies) were statistically significantly predictive of onset, with a positive association reported in all analyses. Inverse relationships were found between onset and academic performance (10/12 studies) and self-esteem (5/5 studies).

Tobacco-related cognitions were examined in 34 analyses, all of which reported a statistically significant relationship with onset in at least one study. Receptivity to tobacco promotions, marketing, and advertising was significantly positively related to onset in nine of ten studies in which it was investigated. Likewise, susceptibility to smoking was significantly positively related to onset in all seven studies in which it was assessed, while intention to smoke in the future was positively related to onset in four of four studies.

Social influences on smoking were investigated in 110 analyses, of which 83 were significant. Smoking by family members and friends were most frequently investigated, accounting for 65 reported analyses. Family smoking was positively related to onset in 29 of 37 studies, with smoking by siblings significantly associated in nine of nine studies. Friends' smoking was positively related to onset in 26 of 28 studies. Parental monitoring/supervision of the child was inversely related to onset in four of five studies.

Environmental influences on smoking were examined in 13 analyses, 11 of which found a statistically significant relationship with onset. In all seven studies in which exposure to smoking in films was investigated, the relationship with onset was positive. Other factors (e.g., asthma, diet, physical activity, neurocognitive functioning, use of waterpipe or other tobacco products, use of non-tobacco drugs) were investigated in 20 analyses and were found to be significantly related to onset in 15; however, none of these factors was investigated in more than three studies.

Discussion

This review examined the evidence on predictors of smoking onset in 53 prospective longitudinal studies published between January 1984 and August 2015. Strengths of this review include restriction to studies in which the sample baseline included never smokers only, which increased the probability that predictors of onset at follow-up were identified. Similarly, by reviewing only prospective studies, confidence in drawing causal inferences was enhanced. The authors deemed that there was evidence for an association between a specific predictor and smoking onset if the finding was statistically significant and the direction of association was common across studies. An increased risk of smoking onset was associated with increased age/grade, lower SES, poor academic performance, sensation seeking or rebelliousness, intention to smoke in the future, receptivity to tobacco promotion efforts, susceptibility to smoking, family members' smoking, having friends who smoke, and exposure to films. By contrast, higher self-esteem and high parental monitoring/supervision of the child appear to protect against smoking onset. Although statistically significant in at least four studies, the direction of the association was discordant across studies for sex and ethnicity. The evidence for all other potential predictors was limited.

It is notable that the studies included in this review originate from diverse disciplines (e.g., psychology, public health, epidemiology) and therefore incorporate different measures and analytic strategies to address similar questions. The findings for the predictors listed above were apparently not sensitive to these diverse

Table 1. Predictors of Smoking Onset in Adolescents From 53 Longitudinal Studies[a]

Predictor	N studies	Statistically significant association	
		n studies	Direction of association
Sociodemographic factors			
Age	16	10	Positive
Grade	7	5	Positive
Sex	29	10	Female < Male: 7
			Male < Female: 3
Race/ethnicity	13	6	Nonwhite > White: 4
			White > Nonwhite: 2
SES	6	4	Inverse
Parent education	6	1	Inverse
Single-parent family	4	3	Positive
Personal/psychological factors			
Academic performance	12	10	Inverse
Attachment to family or community	2	1	Inverse
Attachment to school	1	1	Inverse
Attention-deficit hyperactivity disorder	1	1	Positive
Conduct disorder	1	1	Positive
Depression/depressive disorder	5	3	Positive
Oppositional-defiant disorder	1	1	Positive
Perceived academic performance	3	3	Inverse
Perceived parental control	1	1	Inverse
Personality traits/temperamental characteristics			
Impulsivity	1	1	Positive
Rebelliousness	7	7	Positive
Risk-taking propensity	1	1	Positive
Sensation-seeking	9	9	Positive
Problematic interpersonal relationships in class	1	1	Positive
Self-esteem	5	5	Inverse
Self-regulation	1	1	Inverse
Sociability	1	1	Positive
Stress symptoms	1	1	Positive
Subjective social status in school	1	1	Inverse
Trouble in school	2	1	Positive

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paradigms, thus providing robust evidence that they are indeed associated with smoking onset. The studies also varied to a large extent in terms of which covariates were included in the multivariate models testing similar hypotheses. Again, the similarity in findings despite the variability in methods provides confidence in their robustness.

That numerous diverse studies consistently identified these factors as predictors of smoking onset may have implications for programs and policy. At a minimum, tobacco control practitioners and policymakers should reflect on the relevance of addressing these factors in the conceptualization and design of interventions targeted to preventing cigarette smoking onset. Failure to do so could lead to interventions that are fundamentally flawed because they do not address the full range of factors known to be associated with smoking onset. Indeed, the mitigated results of evaluations of even the most carefully conceptualized and well-funded tobacco control interventions[1–5] may reflect a lack of comprehensive action that address relevant risk factors at the individual and environmental levels. It will be important to distinguish modifiable risk factors (e.g., exposure to family and friends' smoking, exposure to films) from those that are not modifiable (e.g., age or sex) but may be helpful in terms of targeting intervention. For example, although the risk of onset appears to rise as age increases during adolescence, 90% of adult smokers began smoking prior to age 18 years and 99% before age 26 years.[90] Recent efforts to raise the legal age to purchase tobacco in the U.S. from 18 to 21 years[91–94] reflect an evidence-based targeted public health approach. Future studies will also need to address whether the predictors of onset at younger ages differ from those when onset occurs at a later age.

This review underscored several limitations in this literature. Almost

Table 1. Predictors of Smoking Onset in Adolescents From 53 Longitudinal Studies[a] *(continued)*

Predictor	N studies	Statistically significant association	
		n studies	Direction of association
Smoking-related cognitions			
Curiosity about smoking	1	1	Positive
Feeling like one really needs a cigarette	1	1	Positive
Intention to smoke in the future	4	4	Positive
Perceived accessibility of cigarettes	2	2	Positive
Perceived prevalence of peer smoking	1	1	Positive
Perceived parents'/friends' smoking norm	1	1	Positive
Perceived similarity between self vs. smokers	1	1	Positive
Positive attitude toward smoking	2	2	Positive
Positive outcome expectations about smoking	2	2	Positive
Receptivity to tobacco advertising, marketing or promotion, or warnings about smoking			
Marketing	10	9	Positive
Warnings	1	1	Inverse
Self-efficacy in resisting smoking	2	2	Inverse
Susceptibility to smoking	7	7	Positive
Social factors			
Access to cigarettes	2	1	Positive
Familial smoking			
Parents' smoking	23	16	Positive
Siblings' smoking	9	9	Positive
Household smokers	5	4	Positive
Friends' smoking	28	26	Positive
Household smoking ban	1	1	Positive
Maternal responsiveness	2	2	Inverse
Parental engagement or connectedness	2	2	Inverse
Parental communication about smoking			
General/permissive	2	1	Positive
Risks	1	1	Inverse
Parental knowledge of child's and friends' smoking	1	1	Inverse

(continued on next page)

all the studies included in this review defined smoking onset as the transition from never having smoked (even a few puffs) to any level of cigarette consumption. Only two studies[46,66] assessed participants at 3-month intervals over a span of years, which increased the likelihood of capturing the first few puffs compared with studies that assessed participants less frequently. Inclusion of smokers who have achieved different milestones in the natural history of smoking acquisition calls into question whether the identified predictors are indeed predicting onset or are predicting a combination of milestones from onset to maintenance or progression. In short, true predictors of smoking onset (i.e., the transition from never smoking to the first few puffs or first one or two cigarettes) have yet to be identified. This highlights the need for prospective studies of large, representative samples that are followed over the time span during which most youth initiate smoking (e.g., from age 11–12 years to 17–18 years) with sufficiently frequent assessments (e.g., every few months) to capture the moment at which each youth reports having puffed for the first time.

One strength in the existing literature is that 80% of the cohorts were representative of the populations from which they were drawn. Nevertheless, ethnicity is not systematically addressed because most studies were conducted in the U.S. and included primarily Caucasians. Additionally, only four included studies[40,64,82,83] were conducted in non-Western countries (i.e., Taiwan, Jordan, and China), so the extent to which predictors of onset might differ across countries and cultures cannot be synthesized meaningfully. Also, the literature generally points to sex differences in tobacco use,[95] but no single-sex studies were found (although four studies reported sex-specific results) and few studies tested for sex interactions. Therefore,

Table 1. Predictors of Smoking Onset in Adolescents From 53 Longitudinal Studies[a] *(continued)*

Predictor	N studies	Statistically significant association	
		n studies	Direction of association
Parental monitoring/supervision of child	5	4	Inverse
Parental smoking norm (disapproval)	6	2	Inverse
Parenting style (poor parenting)	2	1	Positive
Peer antismoking norm	1	1	Inverse
Peer use of tobacco or other substances	3	1	Positive
Perceived parental reactions to child's smoking			
Accepting (e.g., reward or laissez-faire)	2	1	Positive
Rejecting (e.g., punishment or anger)	2	1	Inverse
Quality of parent–child communication	1	1	Inverse
Secondhand smoke exposure at home	1	1	Positive
In-class factors related to smoking			
Antismoking curricula	1	1	Inverse
Class members smoking	1	1	Positive
Schoolwide factors related to smoking (e.g., antismoking policy/activities, school tolerance of smoking, smoking cessation offered, teachers or school staff smoke)	2	1	Positive
In-class factors unrelated to smoking (problematic interpersonal relationships)	1	1	Positive
Sociometric status (controversial, neglected, rejected) of adolescent	1	1	Positive
Unsupervised after school	1	1	Positive
Environmental factors			
Allowed to watch age-restricted movies	1	1	Positive
Exposure to tobacco advertising	2	2	Positive
Favorite film star smokes on screen	1	1	Positive
Exposure to smoking in films/movies	7	7	Positive

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differences in predictors of smoking onset by sex are largely unexplored. Finally, few studies included children under age 10 years, so little is known about predictors of smoking at this age.

Methodologic weaknesses were also apparent in the included studies. In studies in which participants were clustered within larger units (e.g., schools), appropriate variance corrections were often not used, thereby yielding artificially narrow CIs. Only 12 studies reported a method to correct for clustering. Lack of information on the handling of missing data is also a weakness. As listwise deletion of cases with missing data on any variable is the default in most statistical programs, one might assume that this was the technique employed in the 30 studies that did not report on a method. Although listwise deletion is relatively robust in both linear and logistic regression,[96] it reduces the effective sample size, particularly in multivariate analyses where cases will be deleted for missing data on any of the predictors or covariates in a model. Few studies used data imputation to manage missing data or assessed (or corrected for) possible non-differential attrition over time. The measurement of possible confounders was not carefully operationalized in some studies, and among studies that investigated multiple potential predictors, the causal pathways through which the predictors are operating might not have been carefully conceptualized. This may have resulted in including variables on the causal pathway between the exposure of interest and onset, thereby potentially over-fitting models and obscuring a possible association. Studies that used structural equation models tended to be somewhat more thoughtful about possible causal pathways. The authors have found the directed acyclic graph approach helpful in identifying and adjusting for confounding.[97–99]

Table 1. Predictors of Smoking Onset in Adolescents From 53 Longitudinal Studies[a] *(continued)*

Predictor	N studies	Statistically significant association	
		n studies	Direction of association
Other factors			
Asthma diagnosis or adherence to medication	3	2	Inverse
Asthma coping by hiding condition	1	1	Positive
Compromised neurocognitive functioning	1	1	Positive
Family member job loss	1	1	Positive
Few extracurricular activities	1	1	Positive
Know someone with a smoking-related disease	1	1	Positive
Physical activities in past week	1	1	Positive
Poor diet	1	1	Positive
Shopping frequency	1	1	Positive
TV in bedroom	1	1	Positive
Youth uses non-tobacco drugs	2	2	Positive
Youth uses other tobacco products	2	2	Positive

[a]All predictors found to be statistically significantly related to smoking onset in at least one study are included in this table. Appendix 3 (available online) presents detailed results on all individual indicators of the 98 conceptually different potential predictors investigated in the 53 included studies.

Limitations

As is the case with any review, potential publication bias may have favored articles with statistically significant findings. Studies not published in English were excluded, so the findings may be most relevant to English-speaking countries and contexts. Third, although data quality was controlled for to a certain extent by including only longitudinal studies that followed individuals who were never smokers at baseline, study quality may have varied. The authors opted not to formally assess the quality of other study characteristics (e.g., selection criteria, data collection methods, validity of measures) based on an extensive examination of available tools for quality assessment, which focus primarily on intervention studies and typically score studies based on an algorithm.[100,101] Use of such tools has been called into question, as the same study can be assigned very different quality scores depending on the tool used.[102] Instead, the relevant characteristics of each study are presented (Appendix 2, available online) and readers themselves may use this information to assess their quality. Finally, evidence for an association between a specific predictor and smoking onset was based on whether the finding was statistically significant and if the direction of association was common across studies. This review did not compare the strength of the associations observed across studies because this would require meta-analysis. The detailed data presented in Appendix 3 (available online) offer others the opportunity to conduct such analyses.

Conclusions

Increased age/grade, lower SES, poor academic performance, higher sensation seeking/risk taking/rebelliousness, susceptibility to smoking, intention to smoke in the future, smoking among family members and friends, and exposure to smoking in films were associated with an increased risk of smoking onset among youth. Future longitudinal research should define smoking onset as the transition from never having tried cigarettes to having puffed or smoked one or two cigarettes. Frequent monitoring over a long time span should be incorporated, attrition and clustering should be accounted for, and potential confounders should be thoughtfully selected. Effectiveness of interventions might be increased by addressing the modifiable risk factors identified in this review. Although consideration of age, grade, and SES might help target interventions, the value of targeting by sex or ethnicity remains an open question.

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References

1. U.S. DHHS. *Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General.* Atlanta, GA: DHHS,

CDC, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2012.
2. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. *Monitoring the Future National Survey Results on Drug Use, 1975-2014: Volume I, Secondary School Students*. Ann Arbor: Institute for Social Research, The University of Michigan, 2015.
3. Thomas RE, McLellan J, Perera R. School-based programmes for preventing smoking. *Cochrane Database Syst Rev*. 2013;4:CD001293. http://dx.doi.org/10.1002/14651858.CD001293.pub3.
4. Renaud L, O'Loughlin J, Dery V. The St-Louis du Parc Heart Health Project: a critical analysis of the reverse effects on smoking. *Tob Control*. 2003;12(3):302–309. http://dx.doi.org/10.1136/tc.12.3.302.
5. Kairouz S, O'Loughlin J, Laguë J. Adverse effects of a social contract smoking prevention program among children in Québec, Canada. *Tob Control*. 2009;18(6):474–478. http://dx.doi.org/10.1136/tc.2009.030445.
6. Pallonen UE, Prochaska JO, Velicer WF, Prokhorov AV, Smith NF. Stages of acquisition and cessation for adolescent smoking: an empirical integration. *Addict Behav*. 1998;23(3):303–324. http://dx.doi.org/10.1016/S0306-4603(97)00074-9.
7. Mayhew KP, Flay BR, Mott JA. Stages in the development of adolescent smoking. *Drug Alcohol Depend*. 2000;59(suppl 1):S61–S81. http://dx.doi.org/10.1016/S0376-8716(99)00165-9.
8. Leventhal H, Cleary PD. The smoking problem: a review of the research and theory in behavioral risk modification. *Psycholl Bull*. 1980;88(2):370–405. http://dx.doi.org/10.1037/0033-2909.88.2.370.
9. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed., Washington, DC: American Psychiatric Association; 2015.
10. DiFranza JR, Rigotti NA, McNeill AD, et al. Initial symptoms of nicotine dependence in adolescents. *Tob Control*. 2000;9(3):313–319. http://dx.doi.org/10.1136/tc.9.3.313.
11. DiFranza JR, Savageau JA, Fletcher KE, et al. Symptoms of tobacco dependence after brief intermittent use: the Development and Assexxment of Nicotine Dependence in Youth study II. *Arch Pediatr Adolesc Med*. 2007;161(7):704–710. http://dx.doi.org/10.1001/archpedi.161.7.704.
12. O'Loughlin J, DiFranza JR, Tyndale RF, et al. Nicotine-dependence symptoms are associated with smoking frequency in adolescents. *Am J Prev Med*. 2003;25(3):219–225. http://dx.doi.org/10.1016/S0749-3797(03)00198-3.
13. Gervais A, O'Loughlin J, Meshefedjian G, Bancej C, Tremblay M. Milestones in the natural course of onset of cigarette use among adolescents. *Can Med Assoc J*. 2006;175(3):255–261. http://dx.doi.org/10.1503/cmaj.051235.
14. O'Loughlin J, Gervais A, Dugas E, Meshefedjian G. Milestones in the process of cessation among novice adolescent smokers. *Am J Public Health*. 2009;99(3):499–504. http://dx.doi.org/10.2105/AJPH.2007.128629.
15. Chassin L, Presson CC, Sherman SJ, Edwards DA. The natural history of cigarette smoking: predicting young-adult smoking outcomes from adolescent smoking patterns. *Health Psychol*. 1990;9(6):701–716. http://dx.doi.org/10.1037/0278-6133.9.6.701.
16. CDC. *Preventing Tobacco Use Among Young People. A Report of the Surgeon General*. Washington, DC: U.S. Government Printing Office; 1994.
17. Okoli C, Greaves L, Fagyas V. Sex differences in smoking initiation among children and adolescents. *Public Health*. 2013;127(1):3–10. http://dx.doi.org/10.1016/j.puhe.2012.09.015.
18. Ohmoto M, Hirakoshi M, Mitsumoto Y. Effects of moderating factors including serotonin transporter polymorphisms on smoking behavior: a systematic review and meta-analysis update. *Nicotine Tob Res*. 2013;15(2):572–582. http://dx.doi.org/10.1093/ntr/nts149.
19. Ohmoto M, Sakaishi K, Hama A, Morita A, Nomura M, Mitsumoto Y. Association between dopamine receptor 2 TaqIA polymorphisms and smoking behavior with an influence of ethnicity: a systematic review and meta-analysis update. *Nicotine Tob Res*. 2013;15(3):633–642. http://dx.doi.org/10.1093/ntr/nts196.
20. Verhagen M, Kleinjan M, Engels RC. A systematic review of the A118G (Asn40Asp) variant of OPRM1 in relation to smoking initiation, nicotine dependence and smoking cessation. *Pharmacogenomics*. 2012;13(8):917–933. http://dx.doi.org/10.2217/pgs.12.76.
21. Munafò M, Clark T, Johnstone E, Murphy M, Walton R. The genetic basis for smoking behavior: a systematic review and meta-analysis. *Nicotine Tob Res*. 2004;6(4):583–597. http://dx.doi.org/10.1080/14622200410001734030.
22. Leonardi-Bee J, Jere ML, Britton J. Exposure to parental and sibling smoking and the risk of smoking uptake in childhood and adolescence: a systematic review and meta-analysis. *Thorax*. 2011;66(10):847–855. http://dx.doi.org/10.1136/thx.2010.153379.
23. Okoli CTC, Kodet J. A systematic review of secondhand tobacco smoke exposure and smoking behaviors: smoking status, susceptibility, initiation, dependence, and cessation. *Addict Behav*. 2015;47:22–32. http://dx.doi.org/10.1016/j.addbeh.2015.03.018.
24. Nunez-Smith M, Wolf E, Huang HM, et al. Media exposure and tobacco, illicit drugs, and alcohol use among children and adolescents: a systematic review. *Subst Abus*. 2010;31(3):174–192. http://dx.doi.org/10.1080/08897077.2010.495648.
25. DiFranza JR, Wellman RJ, Sargent JD, Weitzman M, Hipple BJ, Winickoff JP. Tobacco promotion and the initiation of tobacco use: assessing the evidence for causality. *Pediatrics*. 2006;117(6):e1237–e1248. http://dx.doi.org/10.1542/peds.2005-1817.
26. Wellman RJ, Sugarman DB, DiFranza JR, Winickoff JP. The extent to which tobacco marketing and tobacco use in films contribute to children's use of tobacco: a meta-analysis. *Arch Pediatr Adolesc Med*. 2006;160(12):1285–1296. http://dx.doi.org/10.1001/archpedi.160.12.1285.
27. Paynter J, Edwards R. The impact of tobacco promotion at the point of sale: a systematic review. *Nicotine Tob Res*. 2009;11(1):25–35. http://dx.doi.org/10.1093/ntr/ntn002.
28. Charlesworth A, Glantz SA. Smoking in the movies increases adolescent smoking: a review. *Pediatrics*. 2005;116(6):1516–1528. http://dx.doi.org/10.1542/peds.2005-0141.
29. Freedman KS, Nelson NM, Feldman LL. Smoking initiation among young adults in the United States and Canada, 1998-2010: a systematic review. *Prev Chronic Dis*. 2012;9:110037. http://dx.doi.org/10.5888/pcd9.110037.
30. Mak K-K, Ho S-Y, Day JR. A review of life-course familial and lifestyle factors of smoking initiation and cessation. *Curr Cardiovasc Risk Rep*. 2013;7(6):457–463. http://dx.doi.org/10.1007/s12170-013-0341-x.
31. Lovato C, Linn G, Stead LF, Best A. Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. *Cochrane Database Syst Rev*. 2003;4:CD003439. http://dx.doi.org/10.1002/14651858.CD003439.
32. Lovato C, Watts A, Stead LF. Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. *Cochrane Database Syst Rev*. 2011(10):CD003439. http://dx.doi.org/10.1002/14651858.CD003439.pub2.
33. PRISMA. Transparent reporting of systematic reviews and meta-analyses. www.prisma-statement.org. Accessed August 15, 2015.
34. Albers AB, Biener L, Siegel M, Cheng DM, Rigotti N. Household smoking bans and adolescent antismoking attitudes and smoking initiation: findings from a longitudinal study of a Massachusetts youth cohort. *Am J Public Health*. 2008;98(10):1886–1893. http://dx.doi.org/10.2105/AJPH.2007.129320.
35. Aloise-Young PA, Hennigan KM, Graham JW. Role of the self-image and smoker stereotype in smoking onset during early adolescence: a longitudinal study. *Health Psychol*. 1996;15(6):494–497. http://dx.doi.org/10.1037/0278-6133.15.6.494.
36. Aloise-Young PA, Kaeppner CJ. Sociometric status as a predictor of onset and progression in adolescent cigarette smoking. *Nicotine Tob Res*. 2005;7(2):199–206. http://dx.doi.org/10.1080/14622200500055277.

37. Bauman KE, Carver K, Gleiter K. Trends in parent and friend influence during adolescence: the case of adolescent cigarette smoking. *Addict Behav*. 2001;26(3):349–361. http://dx.doi.org/10.1016/S0306-4603(00)00110-6.
38. Bidstrup PE, Frederiksen K, Siersma V, et al. Social-cognitive and school factors in initiation of smoking among adolescents: a prospective cohort study. *Cancer Epidemiol Biomarkers Prev*. 2009;18(2):384–392. http://dx.doi.org/10.1158/1055-9965.EPI-08-0584.
39. Bohnert KM, Rios-Bedoya CF, Breslau N. Parental monitoring at age 11 and smoking initiation up to age 17 among blacks and whites: a prospective investigation. *Nicotine Tob Res*. 2009;11(12):1474–1478. http://dx.doi.org/10.1093/ntr/ntp160.
40. Chang HY, Wu WC, Wu CC, Cheng JY, Hurng BS, Yen LL. The incidence of experimental smoking in school children: an 8-year follow-up of the child and adolescent behaviors in long-term evolution (CABLE) study. *BMC Public Health*. 2011;11:844. http://dx.doi.org/10.1186/1471-2458-11-844.
41. Dalton MA, Sargent JD, Beach ML, et al. Effect of viewing smoking in movies on adolescent smoking initiation: a cohort study. *Lancet*. 2003;362(9380):281–285. http://dx.doi.org/10.1016/S0140-6736(03)13970-0.
42. D'Amico EJ, McCarthy DM. Escalation and initiation of younger adolescents' substance use: the impact of perceived peer use. *J Adolesc Health*. 2006;39(4):481–487. http://dx.doi.org/10.1016/j.jadohealth.2006.02.010.
43. den Exter Blokland EA, Hale WW 3rd, Meeus W, Engels RC. Parental anti-smoking socialization. associations between parental anti-smoking socialization practices and early adolescent smoking initiation. *Eur Addict Res*. 2006;12(1):25–32. http://dx.doi.org/10.1159/000088580.
44. den Exter Blokland EA, Hale WW 3rd, Meeus W, Engels RC. Parental support and control and early adolescent smoking: a longitudinal study. *Subst Use Misuse*. 2007;42(14):2223–2232. http://dx.doi.org/10.1080/10826080701690664.
45. Distefan JM, Pierce JP, Gilpin EA. Do favorite movie stars influence adolescent smoking initiation? *Am J Public Health*. 2004;94(7):1239–1244. http://dx.doi.org/10.2105/AJPH.94.7.1239.
46. Doubeni CA, Li W, Fouayzi H, Difranza JR. Perceived accessibility as a predictor of youth smoking. *Ann Fam Med*. 2008;6(4):323–330. http://dx.doi.org/10.1370/afm.841.
47. Engels RC, Vitaro F, Blokland ED, de Kemp R, Scholte RH. Influence and selection processes in friendships and adolescent smoking behaviour: the role of parental smoking. *J Adolesc*. 2004;27(5):531–544. http://dx.doi.org/10.1016/j.adolescence.2004.06.006.
48. Galanti MR, Rosendahl I, Post A, Gilljam H. Early gender differences in adolescent tobacco use—the experience of a Swedish cohort. *Scand J Public Health*. 2001;29(4):314–317. http://dx.doi.org/10.1177/14034948010290040401.
49. Gilpin EA, Lee L, Pierce JP. How have smoking risk factors changed with recent declines in California adolescent smoking? *Addiction*. 2005;100(1):117–125. http://dx.doi.org/10.1111/j.1360-0443.2005.00938.x.
50. Hanewinkel R, Isensee B, Sargent JD, Morgenstern M. Cigarette advertising and teen smoking initiation. *Pediatrics*. 2011;127(2):e271–e278. http://dx.doi.org/10.1542/peds.2010-2934.
51. Hanewinkel R, Morgenstern M, Tanski SE, Sargent JD. Longitudinal study of parental movie restriction on teen smoking and drinking in Germany. *Addiction*. 2008;103(10):1722–1730. http://dx.doi.org/10.1111/j.1360-0443.2008.02308.x.
52. Hanewinkel R, Sargent JD. Exposure to smoking in internationally distributed American movies and youth smoking in Germany: a cross-cultural cohort study. *Pediatrics*. 2008;121(1):e108–e117. http://dx.doi.org/10.1542/peds.2007-1201.
53. Harakeh Z, de Sonneville L, van den Eijnden RJ, et al. The association between neurocognitive functioning and smoking in adolescence: the TRAILS study. *Neuropsychology*. 2012;26(5):541–550. http://dx.doi.org/10.1037/a0029217.
54. Harakeh Z, Scholte RH, Vermulst AA, de Vries H, Engels RC. Parental factors and adolescents' smoking behavior: an extension of the theory of planned behavior. *Prev Med*. 2004;39(5):951–961. http://dx.doi.org/10.1016/j.ypmed.2004.03.036.
55. Henriksen L, Schleicher NC, Feighery EC, Fortmann SP. A longitudinal study of exposure to retail cigarette advertising and smoking initiation. *Pediatrics*. 2010;126(2):232–238. http://dx.doi.org/10.1542/peds.2009-3021.
56. Huver RM, Engels RC, de Vries H. Are anti-smoking parenting practices related to adolescent smoking cognitions and behavior? *Health Educ Res*. 2006;21(1):66–77. http://dx.doi.org/10.1093/her/cyh045.
57. Jackson C. Cognitive susceptibility to smoking and initiation of smoking during childhood: a longitudinal study. *Prev Med*. 1998;27(1):129–134. http://dx.doi.org/10.1006/pmed.1997.0255.
58. Jackson C, Brown JD, L'Engle KL. R-rated movies, bedroom televisions, and initiation of smoking by white and black adolescents. *Arch Pediatr Adolesc Med*. 2007;161(3):260–268. http://dx.doi.org/10.1001/archpedi.161.3.260.
59. Jackson C, Henriksen L, Dickinson D, Messer L, Robertson SB. A longitudinal study predicting patterns of cigarette smoking in late childhood. *Health Educ Behav*. 1998;25(4):436–447. http://dx.doi.org/10.1177/109019819802500403.
60. Killen JD, Robinson TN, Haydel KF, et al. Prospective study of risk factors for the initiation of cigarette smoking. *J Consul Clin Psychol*. 1997;65(6):1011–1016. http://dx.doi.org/10.1037/0022-006X.65.6.1011.
61. King SM, Iacono WG, McGue M. Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction*. 2004;99(12):1548–1559. http://dx.doi.org/10.1111/j.1360-0443.2004.00893.x.
62. Lee DJ, Trapido E, Rodriguez R. Self-reported school difficulties and tobacco use among fourth- to seventh-grade students. *J Sch Health*. 2002;72(9):368–373. http://dx.doi.org/10.1111/j.1746-1561.2002.tb03561.x.
63. Mahabee-Gittens EM, Xiao Y, Gordon JS, Khoury JC. The dynamic role of parental influences in preventing adolescent smoking initiation. *Addict Behav*. 2013;38(4):1905–1911. http://dx.doi.org/10.1016/j.addbeh.2013.01.002.
64. McKelvey K, Attonito J, Madhivanan P, Yi Q, Mzayek F, Maziak W. Determinants of cigarette smoking initiation in Jordanian schoolchildren: longitudinal analysis. *Nicotine Tob Res*. 2015;17(5):552–558. http://dx.doi.org/10.1093/ntr/ntu165.
65. Milton B, Cook PA, Dugdill L, Porcellato L, Springett J, Woods SE. Why do primary school children smoke? A longitudinal analysis of predictors of smoking uptake during pre-adolescence. *Public Health*. 2004;118(4):247–255. http://dx.doi.org/10.1016/j.puhe.2003.10.006.
66. O'Loughlin J, Karp I, Koulis T, Paradis G, DiFranza J. Determinants of first puff and daily cigarette smoking in adolescents. *Am J Epidemiol*. 2009;170(5):585–597. http://dx.doi.org/10.1093/aje/kwp179.
67. O'Loughlin J, Paradis G, Renaud L, Sanchez Gomez L. One-year predictors of smoking initiation and of continued smoking among elementary schoolchildren in multiethnic, low-income, inner-city neighbourhoods. *Tob Control*. 1998;7(3):268–275. http://dx.doi.org/10.1136/tc.7.3.268.
68. Pierce JP, Distefan JM, Jackson C, White MM, Gilpin EA. Does tobacco marketing undermine the influence of recommended parenting in discouraging adolescents from smoking? *Am J Prev Med*. 2002;23(2):73–81. http://dx.doi.org/10.1016/S0749-3797(02)00459-2.
69. Pierce JP, Distefan JM, Kaplan RM, Gilpin EA. The role of curiosity in smoking initiation. *Addict Behav*. 2005;30(4):685–696. http://dx.doi.org/10.1016/j.addbeh.2004.08.014.
70. Rosendahl KI, Galanti MR, Gilljam H, Bremberg S, Ahlbom A. School and class environments are differently linked to future

smoking among preadolescents. *Prev Med*. 2002;34(6):649–654. http://dx.doi.org/10.1006/pmed.2002.1033.
71. Sargent JD, Beach ML, Dalton MA, et al. Effect of parental R-rated movie restriction on adolescent smoking initiation: a prospective study. *Pediatrics*. 2004;114(1):149–156. http://dx.doi.org/10.1542/peds.114.1.149.
72. Sargent JD, Hanewinkel R. Comparing the effects of entertainment media and tobacco marketing on youth smoking in Germany. *Addiction*. 2009;104(5):815–823. http://dx.doi.org/10.1111/j.1360-0443.2009.02542.x.
73. Spelman AR, Spitz MR, Kelder SH, et al. Cognitive susceptibility to smoking: two paths to experimenting among Mexican origin youth. *Cancer Epidemiol Biomarkers Prev*. 2009;18(12):3459–3467. http://dx.doi.org/10.1158/1055-9965.EPI-09-0765.
74. Tanski SE, Stoolmiller M, Dal Cin S, Worth K, Gibson J, Sargent JD. Movie character smoking and adolescent smoking: who matters more, good guys or bad guys? *Pediatrics*. 2009;124(1):135–143. http://dx.doi.org/10.1542/peds.2007-3420.
75. Thrasher JF, Sargent JD, Huang L, Arillo-Santillan E, Dorantes-Alonso A, Perez-Hernandez R. Does film smoking promote youth smoking in middle-income countries? A longitudinal study among Mexican adolescents. *Cancer Epidemiol Biomarkers Prev*. 2009;18(12):3444–3450. http://dx.doi.org/10.1158/1055-9965.EPI-09-0883.
76. Titus-Ernstoff L, Dalton MA, Adachi-Mejia AM, Longacre MR, Beach ML. Longitudinal study of viewing smoking in movies and initiation of smoking by children. *Pediatrics*. 2008;121(1):15–21. http://dx.doi.org/10.1542/peds.2007-0051.
77. Unger JB, Hamilton JE, Sussman S. A family member's job loss as a risk factor for smoking among adolescents. *Health Psychol*. 2004;23(3):308–313. http://dx.doi.org/10.1037/0278-6133.23.3.308.
78. Urberg KA, Degirmencioglu SM, Pilgrim C. Close friend and group influence on adolescent cigarette smoking and alcohol use. *Dev Psychol*. 1997;33(5):834–844. http://dx.doi.org/10.1037/0012-1649.33.5.834.
79. Van De Ven MO, Engels RC, Kerstjens HA, Van den Eijnden RJ. Bidirectionality in the relationship between asthma and smoking in adolescents: a population-based cohort study. *J Adolesc Health*. 2007;41(5):444–454. http://dx.doi.org/10.1016/j.jadohealth.2007.05.015.
80. Van De Ven MO, Engels RC, Otten R, Van Den Eijnden RJ. A longitudinal test of the theory of planned behavior predicting smoking onset among asthmatic and non-asthmatic adolescents. *J Behav Med*. 2007;30(5):435–445. http://dx.doi.org/10.1007/s10865-007-9119-2.
81. Van De Ven MO, Engels RC, Sawyer SM. Asthma-specific predictors of smoking onset in adolescents with asthma: a longitudinal study. *J Pediatr Psychol*. 2009;34(2):118–128. http://dx.doi.org/10.1093/jpepsy/jsn063.
82. Wang MP, Ho SY, Lam TH. Parental smoking, exposure to second-hand smoke at home, and smoking initiation among young children. *Nicotine Tob Res*. 2011;13(9):827–832. http://dx.doi.org/10.1093/ntr/ntr083.
83. Wang MP, Ho SY, Lo WS, Lam TH. Overestimation of peer smoking prevalence predicts smoking initiation among primary school students in Hong Kong. *J Adolesc Health*. 2011;48(4):418–420. http://dx.doi.org/10.1016/j.jadohealth.2010.07.017.
84. Wilkinson AV, Shete S, Vasudevan V, Prokhorov AV, Bondy ML, Spitz MR. Influence of subjective social status on the relationship between positive outcome expectations and experimentation with cigarettes. *J Adolesc Health*. 2009;44(4):342–348. http://dx.doi.org/10.1016/j.jadohealth.2008.08.003.
85. Woodruff SI, Candelaria JI, Laniado-Laborin R, Sallis JF, Villasenor A. Availability of cigarettes as a risk factor for trial smoking in adolescents. *Am J Health Behav*. 2003;27(1):84–88. http://dx.doi.org/10.5993/AJHB.27.1.9.
86. Woodruff SI, Laniado-Laborin R, Candelaria JI, Villasenor A, Sallis JF. Parental prompts as risk factors for adolescent trial smoking: results of a prospective cohort study. *Addict Behav*. 2004;29(9):1869–1873. http://dx.doi.org/10.1016/j.addbeh.2004.03.031.
87. Counsell C. Formulating questions and locating primary studies for inclusion in systematic reviews. *Ann Intern Med*. 1997;127(5):380–387. http://dx.doi.org/10.7326/0003-4819-127-5-199709010-00008.
88. O'Connor D, Green S, Higgins JPT. Defining the review question and developing criteria for including studies. In: Higgins JPT, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions*. Chichester: Wiley-Blackwell, 2008:83–94. http://dx.doi.org/10.1002/9780470712184.ch5.
89. Grimshaw J. A knowledge synthesis chapter. www.cihr-irsc.gc.ca/e/41382.html. Accessed August 20, 2015.
90. U.S. DHHS. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: U.S. DHHS, CDC, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health; 2014.
91. Winickoff JP, Gottlieb M, Mello MM. Tobacco 21: an idea whose time has come. *N Engl J Med*. 2014;370(4):295–297. http://dx.doi.org/10.1056/NEJMp1314626.
92. Winickoff JP, McMillen R, Tanski S, et al. Public support for raising the age of sale for tobacco to 21 in the United States. *Tob Control*. 2016;25(3):284–288. http://dx.doi.org/10.1136/tobaccocontrol-2014-052126.
93. Winickoff JP, Hartman L, Chen ML, et al. Retail impact of raising tobacco sales age to 21 years. *Am J Public Health*. 2014;104(11):e18–e21. http://dx.doi.org/10.2105/AJPH.2014.302174.
94. IOM. *Public health implications of raising the minimum age of legal access to tobacco products*. Washington, DC: The National Academies Press; 2015. http://books.nap.edu/openbook.php?record_id=18997&page=R1 Accessed August 13, 2015.
95. Global Youth Tobacco Survey Collaborating Group. Differences in worldwide tobacco use by gender: findings from the Global Youth Tobacco Survey. *J Sch Health*. 2003;73(6):207–215. http://dx.doi.org/10.1111/j.1746-1561.2003.tb06562.x.
96. Allison PD. *Missing Data*. Thousand Oaks, CA: Sage; 2002.
97. Shrier I, Platt RW. Reducing bias through directed acyclic graphs. *BMC Medl Res Methodol*. 2008;8:70. http://dx.doi.org/10.1186/1471-2288-8-70.
98. Textor J, Hardt J, Knüppel S. DAGitty: a graphical tool for analyzing causal diagrams. *Epidemiology*. 2011;5(22):745. http://dx.doi.org/10.1097/EDE.0b013e318225c2be.
99. Textor J. DAGitty: a browser-based environment for creating causal models. www.dagitty.net.
100. Viswanathan M, Berkman ND, Dryden DM, Hartling L. Assessing risk of bias and confounding in observational studies of interventions or exposures: further development of the RTI Item Bank. Methods research report. (Prepared by RTI–UNC Evidence-based Practice Center under Contract No. 290-2007-10056-I). AHRQ Publication No. 13-EHC106-EF. Rockville, MD: Agency for Healthcare Research and Quality; August 2013. www.effectivehealthcare.ahrq.gov/reports/final.cfm.
101. Katikederiddi SV, Egan M, Petticrew M. How do systematic reviews incorporate risk of bias assessments into the synthesis of evidence? A methodological study. *J Epidemiol Community Health*. 2015;69(2):189–195. http://dx.doi.org/10.1136/jech-2014-204711.
102. Sanderson S, Tatt IT, Higgins JPT. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: a systematic review and annotated bibliography. *Int J Epidemiol*. 2007;36(3):666–676. http://dx.doi.org/10.1093/ije/dym018.

Appendix

Supplementary data

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