HHS Public Access

Author manuscript

Psychol Addict Behav. Author manuscript; available in PMC 2017 December 01.

Published in final edited form as:

Psychol Addict Behav. 2016 December ; 30(8): 876–886. doi:10.1037/adb0000213.

How Is the Effect of Adolescent E-cigarette Use on Smoking Onset Mediated: A Longitudinal Analysis

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Abstract

E-cigarette use by adolescents has been related to onset of cigarette smoking but there is little knowledge about the process(es) through which this occurs. Accordingly, we tested the role of cognitive and social factors for mediating the relation between e-cigarette use and smoking onset. A school-based survey was conducted with a baseline sample of 2,338 students in Hawaii (9th and 10th graders, mean age 14.7 years) who were surveyed in 2013 (Time 1, T1) and followed up 1 year later (Time 2, T2). We assessed e-cigarette use, cigarette smoking, demographic covariates, and four hypothesized mediators: smoking-related expectancies, prototypes, and peer affiliations as well as marijuana use. The primary structural modeling analysis, based on initial never-smokers, used an autoregressive model (entering T2 mediator values adjusted for T1 values) to test for mediational pathways in the relation between e-cigarette use at T1 and cigarette smoking status at T2. Results showed that e-cigarette use was related to all of the mediators and tests of indirect effects indicated that changes in expectancies, affiliations, and marijuana use were significant pathways in the relation between e-cigarette use and smoking onset. A direct effect from e-cigarette use to smoking onset was nonsignificant. Findings were replicated across autoregressive and prospective models. We conclude that the relation between adolescent e-cigarette use and smoking onset is in part attributable to cognitive and social processes that follow from e-cigarette use. Further research is needed to understand the relative role of nicotine and psychosocial factors in smoking onset.

Keywords

e-cigarettes; cigarette smoking; initiation; mediation

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Over the past five years there has been a dramatic increase in the prevalence of e-cigarette use among adolescents (Johnston et al., 2016; Singh et al., 2016) and adolescents now have extensive exposure to marketing of e-cigarettes on television and other venues (Duke et al., 2014; Grana & Ling, 2014). This phenomenon has stimulated a number of studies on the factors associated with adolescent e-cigarette use (Barrington-Trimis et al., 2015; Dutra & Glantz, 2014; Krishnan-Sarin et al., 2014) and the reasons persons give for using e-cigarettes (Kong et al., 2015; Pepper et al., 2014; Pokhrel et al., 2015). A key issue has been the question of whether the availability of e-cigarettes will eliminate smoking of combustible cigarettes (Cobb & Abrams, 2014) or whether e-cigarette use could result in more favorable attitudes about smoking and perhaps increase the likelihood of smoking behavior among previous nonsmokers (Bostean, Trinidad, & McCarthy, 2015; Fairchild et al., 2014; Grana, Benowitz, & Glantz, 2014).

Research with community-based samples of adolescents has begun to address this question. Studies comparing the psychosocial profiles of adolescents who are nonusers of either tobacco product, those who only use e-cigarettes, and those who use both cigarettes and e-cigarettes (i.e., dual users) have indicated that the e-cigarette only users are intermediate in risk status between the nonusers, who are low, and the dual users, who are quite elevated on risk status (Hanewinkel & Isensee, 2015; Kristjansson, Mann, & Sigfusdottir, 2015; Leventhal et al., 2016; Wills et al., 2015). This suggests that e-cigarettes are operating to recruit lower-risk adolescents to substance use. Studies of attitudes toward smoking also have shown that e-cigarette use is related to greater interest in smoking in the future, controlling for several covariates (Bunnell et al., 2015; Coleman et al., 2015; Primack et al., 2015; Wills et al., 2016a). Furthermore, recent longitudinal studies have indicated that among nonsmokers, e-cigarette use is related to an increased likelihood of initiating cigarette smoking 6 months to 1 year later (Barrington-Trimis et al., 2016; Leventhal et al., 2015; Primack et al., 2015; Unger et al., 2016; Wills et al., 2016b).

The fact that e-cigarette use is related to onset of smoking indicates that this is a phenomenon with public health significance. Although longitudinal studies have shown that e-cigarette use is related to smoking onset, at present there is little understanding of the mechanism(s) through which this occurs. Mediation analysis provides a theoretically-based approach to this question because specific mechanisms can be tested and incorrect hypotheses can be disconfirmed (MacKinnon, Fairchild, & Fritz, 2007). In the present research we consider several psychological mechanisms through which e-cigarette use could be linked to smoking onset and conduct a longitudinal mediation analysis to test the role of these mechanisms. Plausible mechanisms, derived from theory and research on adolescent smoking and current findings about e-cigarette use among adolescents, include cognitive/perceptual mechanisms and, perhaps independently, social and problem-behavior mechanisms.

Cognitive-Perceptual Mechanisms

Expectancies have been a major theme in research on adolescent cigarette smoking and other substance use (Patel & Fromme, 2010). Positive expectancies about how substance use can increase self-confidence, reduce boredom, and help regulate negative affect have been linked

to onset of smoking and alcohol use in a variety of populations (Dal Cin et al., 2009; Heinz, Kassel, Bernbaum, & Mermelsten, 2010; Wahl, Turner, Mermelstein, & Flay, 2005). For e-cigarettes, focus-group research and psychometric studies have identified dimensions in reasons for e-cigarette use that are analogous to several dimensions of smoking expectancies (Kong et al., 2015; Pepper et al., 2014). Though some dimensions are unique to e-cigarettes (e.g., evading clean-air restrictions; Pokhrel et al., 2014, 2015), there is considerable similarity. Hence we predicted that using e-cigarettes will encourage more positive expectancies about cigarette smoking and this will be a mediational pathway for smoking onset.

Perceptions of the typical same-age substance user (i.e., prototypes of users) are another dimension that has been linked to uptake of smoking and other substance use (Gerrard et al., 2008; Gibbons et al., 2015). Although current studies indicate that prototypes of smokers tend to be negative in the general population of adolescents, persons who hold relatively more favorable prototypes are more likely to initiate smoking (Gibbons et al., 2015; Hukkelberg & Dykstra, 2009). Studies of e-cigarettes have indicated that enhancement of social popularity is a salient reason for e-cigarette use (Pokhrel et al., 2015). Thus it can be hypothesized that adolescents who are using e-cigarettes would be inclined to see cigarette smokers as more attractive. This suggests a different mechanism that is not conceptually identical to expectancies about the physical effects of smoking. Accordingly we hypothesized that e-cigarette use would be related to more positive prototypes of smokers and this could be another pathway to smoking onset.

Social and Problem-Behavior Mechanisms

Affiliating with peers who smoke is a reliable risk factor for smoking initiation (Hoffman, Sussman, Unger, & Valente, 2006) and social processes may operate in peer groups so as to encourage smoking behavior (Sussman, Pokhrel, Ashmore, & Brown, 2007). In the case of e-cigarette use, several studies have shown correlations of adolescent e-cigarette use with peers' e-cigarette use (Barrington Trimis et al., 2015; Leventhal et al., 2015), which suggests that an analogous social influence process may be operative. In addition, a substantial proportion of adolescents who use e-cigarettes also smoke cigarettes (Dutra & Glantz, 2014; Wills et al., 2015); hence adolescents who are affiliating with other e-cigarette users are likely to come into contact with adolescents who are smoking cigarettes. These peers may provide cigarettes and encourage smoking behavior. Thus we predicted that increased affiliation with peer smokers would be a mechanism linking e-cigarette use and smoking onset.

Problem behavior theory (Jessor & Jessor, 1977) has been a widely supported perspective on the etiology of adolescent substance use (Jessor, 1998). This approach postulates that adolescents who reject mainstream values will be disposed to adopt several types of deviant behavior. Problem behavior constructs have been shown to be related to adolescent smoking (Chassin et al., 2007; Costa, Jessor, & Turbin, 2007) as well as other substance use (Costa et al., 2005). Marijuana use has generally been discouraged for teenagers by schools and parents; in most states it is an illegal and arguably deviant behavior. Also, as an inhaled substance it seems behaviorally closest to cigarette smoking. Thus we hypothesized that e-

cigarette use would be related to increase in marijuana use and this would be another mechanism linking adolescent e-cigarette use to smoking onset.

Present Research

A sample of adolescents was surveyed on two occasions with a 1-year follow-up interval. (The sample is the same as in Wills et al., 2016b.) For the primary hypothesis test we performed a longitudinal onset analysis, restricting the sample to participants who were never-smokers at Time 1 (T1) and determining variables that predicted cigarette smoking status at Time 2 (T2). Mechanisms hypothesized to link initial e-cigarette use to onset of cigarette smoking were tested with variables previously shown to be temporally antecedent to smoking onset (Hoffman et al., 2006; Patel & Fromme, 2010; Wills, Resko, Ainette, & Mendoza, 2004). Structural equation analysis was conducted with a longitudinal mediation model, regressing T2 values of the hypothesized mediators on their corresponding T1 values and specifying the T2 values of the mediators as predictors of smoking onset. In this conservative analysis, technically termed an autoregressive mediation model (MacKinnon, 2008), prediction of T2 smoking status is based on residual values of the mediators over the 1-year follow-up period (i.e., not accounted for by T1 value); additionally, the model partials any correlation of e-cigarette use with the T1 variables.[1] This is conceptually similar to analysis based on change scores but has several statistical advantages (MacKinnon, 2008, chap. 8). We predicted that T1 e-cigarette use would be linked to T2 smoking onset through change over time in expectancies, prototypes, peer affiliations, and/or marijuana use. The structural model included demographic variables as covariates so that results would be independent of gender, ethnicity, family structure, and parental educational status.

Method

Data were obtained through a school-based study conducted with high school students in Hawaii. Prior research has demonstrated that predictive effects obtained for adolescents in Hawaii are similar to findings obtained elsewhere (Wills et al., 2013, 2015).

Participants and Procedure

The participants were students in six high schools on Oahu, Hawaii. At T1 (2013; N = 2,338), 49% of the participants were 9th graders, 42% were 10th graders, and 9% were 11th graders; age range was 14–16 years (M age 14.7 years, SD = 0.7). Participants were resurveyed approximately 1 year later at T2 (2014; N = 2,239) when M age was 15.8 years (SD = 0.9). The T1 sample was 53% female, 24% were of Asian-American background (Chinese, Japanese, or Korean), 19% were Caucasian, 27% were Filipino-American, 20% were Native Hawaiian or other Pacific Islander, and 10% were of other race/ethnicity. Regarding family structure, 17% of participants lived with a single parent, 12% were in a stepparent family (one or both parents was a stepparent), 60% lived with two biological parents, and 11% were in an extended family structure (two parents plus two or more

[1]The onset model used as the primary test in the present research is a special case of the autoregressive model, in which all participants have the same T1 value for the outcome (i.e., they are nonsmokers) and the analysis tests whether they transition to smoker status at T2.

relatives in the household). The mean for father's education on a 1–6 scale with anchor points grade school and post-college was 4.2 (*SD* = 1.2).

The sampling frame was all students in the target grades with adequate English language ability. The research was approved by the Institutional Review Boards of the University of Hawaii and the Hawaii State Department of Education. Signed parental consent and signed student assent were required at each assessment. The response rates (completed surveys/class size from school lists) were 70% at T1 and 67% at T2. The majority of non-participation was due to parents not returning the consent form to the school (71% of missing cases at both waves). The paper survey took approximately 40 minutes and was administered by trained research staff in school classrooms. Students were instructed by the research staff that data were confidential and they should not write their name on the survey. Students who declined assent were instructed to remain at their desks and do school work or, in some schools, were taken to the school library by a teacher so they could study there. Participants were assigned an arbitrary numerical code to deidentify surveys while linking participants across data collection points.

Measures

Demographics—The demographic variables included gender, family structure ("What adults do you live with right now?" with nine multiple-response alternatives), and parental education ("What is the highest level of education your father/mother has completed?" with six fixed responses from Grade School to Post-College). Ethnicity was assessed with a lead-in item asking "What would you say you are?" followed by 14 multiple-response options including Chinese, Japanese, Korean, Native Hawaiian, Filipino, and Caucasian. Students who checked more than one ethnicity were asked "If you had to choose only one, what would you say?" and this item was used to index primary perceived ethnicity.

E-cigarette and cigarette measures: The measure of e-cigarette use asked: "Which of the following is most true for you about smoking electronic cigarettes (e-cigarettes, Volcanos)? (Check One)."[2] A 0–6 scale had response points "I have never smoked an e-cigarette in my life," "I have smoked e-cigarettes 1–2 times," "I have smoked e-cigarettes 3–4 times," "I usually smoke a few e-cigarettes a year," "I usually smoke a few e-cigarettes a month," "I usually smoke a few e-cigarettes each week," and "I usually smoke e-cigarettes every day." The measure for cigarette use had the stem, "Which of the following is most true for you about smoking cigarettes? (Check One)" and had a similar 0–6 response scale ("I have never smoked cigarettes in my life" to "I usually smoke cigarettes every day").

Mediator Variables

Four variables were hypothesized to mediate the effect of e-cigarette use. The measures had been previously validated as predictors of adolescent substance use (Gerrard et al., 2008; Gibbons et al., 2015; Wills et al., 2011, 2013); we report internal consistency reliabilities (Cronbach alpha) for this sample. Measures were scored so that a higher value indicates more of the attribute named in the variable label.

[2]"Volcano" is the brand name for a popular line of e-cigarettes manufactured in Hawaii.

Smoking expectancies—Items on expectancies about smoking were introduced with the stem, "Here are some things that people have said about smoking cigarettes. Circle a number (from 1 to 5) to show what you think." Sample items were "Smoking makes you more self-confident," "Smoking helps you calm down when you're feeling tense," and "Smoking cheers you up when you're in a bad mood." Responses were on 5-point Likert scales ("Not at All True" to "Very True"). Alphas were .94 and .93 for T1 and T2, respectively.

Prototypes of smokers—Items on perceptions of typical same-age smokers were introduced with the stem, "There are some young people who smoke cigarettes. We want to know what you think about them. Take a moment to think about the type of person your age who smokes. We're not talking about anyone in particular, just the type of person who smokes. Circle a number (from 1 to 5) to show your image of kids who smoke." Responses on a 5-point adjective scale ("Not At All …" to "Very …") were obtained for the items popular, smart, cool, and attractive. On the basis of reliability analyses, popular was dropped. Alphas were .77 and .79 for T1 and T2, respectively.

Peer smoker affiliations—Items on peer affiliation were introduced with the stem, "Here are some simple questions about your friends. Please give the best answer you can for each one. Remember to check only one answer for each question." The item on peer smoking asked, "Do any of your friends smoke cigarettes? [Check One]." Response points were "None of my friends smoke cigarettes," "One of my friends …," "Two of my friends …," "Three of my friends …," and "More than three of my friends smoke cigarettes."

Marijuana use—The measure for marijuana use was introduced with the stem, "Which of the following is most true for you about using marijuana: (Check One)." A 0–6 response scale was analogous to the measure for cigarette smoking, e.g., "I have never had marijuana in my life" to "I usually smoke marijuana every day."

Analysis Methods

Analyses were performed in SAS and Mplus (Muthén & Muthén, 2010). Descriptive statistics were computed for the study variables at the two time points. Missing data rates were generally low for individual variables (1% – 2%) except for parental education. We tested for attrition effects in the longitudinal data and found some evidence of differential attrition (e.g., more attrition among persons with higher rebelliousness), consistent with typical findings in longitudinal studies of adolescents (Wills et al., 2005), but the effect sizes were small (for more detail see Wills et al., 2016b). Structural equation modeling was conducted in Mplus for the subsample of participants who had never smoked cigarettes at T1 (N = 1,984), with the EM algorithm used to include missing data in the analysis (Graham, 2007; Shafer & Graham, 2003). The primary structural model was specified with T1 e-cigarette use and the covariates as exogenous (i.e., not predicted by any prior variables in the model), including all their intercorrelations. The covariates were gender (dichotomous), ethnicity (four binary indices, with Asian-American as the reference group), family structure (three binary indices, with intact family as the reference group), and father's education (6-point scale). Ever-use of e-cigarettes was utilized as the predictor because previous research has shown that any level of e-cigarette use significantly increases the likelihood of smoking

(Wills et al., 2016a,b). The four hypothesized mediators were specified as endogenous (i.e., could be predicted by prior variables in the model), with residual covariances of their error terms. Smoking status at T2 was the criterion variable; onset was defined as ever smoked because previous research and current analyses have shown that even experimental use of cigarettes significantly increases risk for subsequent regular and dependent smoking (Chassin et al., 1990; Dierker & Mermelstein, 2010; Sargent et al., 2016). The model was initially specified with all paths from the exogenous variables to the mediators, all four paths from the mediators to the criterion variable, and a direct effect from e-cigarette use to smoking onset. Nonsignificant paths ($p > .05$) were then trimmed from the model and modification indices were examined for other direct effects, but none were added in the primary model. For a sensitivity analysis we performed a parallel analysis for the total sample with metric scores for T1 e-cigarette use and T2 smoking and with Time 1 smoking score included as a covariate. In addition, to verify temporal relations of variables in this sample we analyzed prospective models with only the T1 values of the mediators included as predictors of T2 smoking. The two models with a dichotomous criterion variable were estimated with the WLSMV estimator (weighted least squares with robust estimates of standard errors and mean-and variance-adjusted chi-square statistic). The two models with a continuous criterion variable were estimated with MLR (maximum likelihood with robust estimates of standard errors). Mediation was tested in Mplus using the delta method as this was comparable across models.

Results

Descriptive Statistics

For the total T1 sample, the prevalence for ever-use of e-cigarettes was 31% at T1 and was 38% at T2. The prevalence for ever-use of cigarettes was 15% at T1 and was 21% at T2. Of the participants who had only used e-cigarettes at T1, 78% were still only using e-cigarettes at T2 and 22% had initiated cigarette smoking (98% as dual users). Of the participants who were dual users at T1 (e-cigarettes + cigarettes), 89% were still dual users at T2, 5% were only using e-cigarettes, and 6% were only using cigarettes. In the analytic subsample of never smokers at T1 ($N = 1,984$), the raw onset rate was 5% for adolescents who had never used either tobacco product and was 20% for adolescents who had used e-cigarettes. Because the former group was considerably larger, the overall 1-year smoking onset rate was 8%.

Frequency distributions indicated that in the subsample of never-smokers at T1, 60% had no friends who smoked, 25% had 1–3 friends who smoked, and 15% had more than three friends who smoked. Corresponding proportions at T2 were 57%, 28%, and 15%. Analyzed with a 7-point scale, the skewness values were 1.19 and 1.13 for T1 and T2, respectively. Data on T1 marijuana use indicated 90% of the subsample had never used marijuana, 7% had used it 1–5 times, 2% used marijuana yearly or monthly, and 1% used it weekly or daily. Corresponding proportions at T2 were 82%, 10%, 6%, and 2%. For the 7-point marijuana scale, skewness values were 4.27 and 2.84 for T1 and T2, respectively.

Descriptive statistics for smoker prototypes and smoking expectancies in the analytic subsample of never-smokers at T1 are presented in Table 1. Distributions for the variables

generally had only moderate skewness. In terms of absolute level, the data did not reflect very positive prototypes or expectancies. However it should be recalled that these are data for nonsmokers, and nonsmokers would be expected to hold less positive prototype perceptions than smokers. We tested this in the present data (for the total sample) and found that compared to T1 never-smokers, T1 current smokers in fact held more positive prototypes of smokers ($t = 5.62$, $p < .0001$) and more positive expectancies about smoking ($t = 11.40$, $p < .0001$).

Correlations of the study variables from a confirmatory model for the analytic sample of never-smokers, analyzed in Mplus, are presented in Table 2.[3] E-cigarette use had a significant zero-order correlation at T1 with all the hypothesized mediators (smoker prototypes, smoking expectancies, peer smoking, and marijuana use). Correlations with demographics were generally minimal but some correlations with marijuana use and peer smoking can be noted. The mediator variables showed moderate stability over time; the T1-T2 correlations were .56, .62, .52, and .50, respectively. Each of the hypothesized mediator variables showed a significant correlation with T2 smoking onset, with these correlations being higher for the T2 values than for the T1 values.

Structural Modeling Analysis

The primary structural model was analyzed in Mplus for the analytic sample of T1 never-smokers with smoking onset specified as a binary variable. Weighted-least-squares (WLSMV) was the method used to estimate the model. The final model, presented in Figure 1A, had chi-square (39 *df, N* = 1,984) = 77.32, Comparative Fit Index (CFI) = .98, and Root Mean Square Error of Approximation (RMSEA) = .022, all parameters indicating good fit of the model to the data. Covariances of exogenous variables, included in the model but excluded from the figure for graphical simplicity, are presented in Table 2. Residual correlations of the endogenous variables, presented in Table 3, indicated that prototypes of smokers were moderately correlated with smoking expectancies and peer smoker affiliations, but other residual correlations were lower.

Results for the structural model (Figure 1A) indicated significant paths ($p \leq .05$) from T1 e-cigarette use to the hypothesized mediators. Paths from T1-T2 change in the mediators to T2 smoking onset were significant ($p \leq .01$) in all cases.[4] With the indirect pathways included in the model, the direct effect from T1 e-cigarette use to T2 smoking onset was not significant ($p = .11$). Prior variables in the model (including demographics and T1 values) accounted for 30% to 51% of the variance in the T2 mediators. Together the variables in the model accounted for 34% of the variance in smoking onset.

Tests of the indirect effects for the primary structural model are presented in Table 4. Significant indirect effects from e-cigarette use to smoking onset were found though more

[3]Family structure indices were included in the structural model but had no significant effects so for presentational simplicity, data for family structure are not included in the correlation table or the figure. Because of skewness, marijuana use was analyzed with a log transform but all other variables were analyzed in their original metric.

[4]The models were replicated as a multilevel analysis with school included as a clustering variable. Findings for models with school included were generally quite similar to those without this procedure except that standard errors were slightly larger in the former models; but none of the conclusions of the present analyses were changed.

favorable expectancies about smoking ($p = .03$), increased affiliation with peer smokers ($p = .01$), and increase in marijuana use over time ($p < .0001$). The indirect effect through prototypes of smokers was only marginally significant (p = .07); although the path from prototype change to smoking onset was significant, the path from e-cigarette use to favorability of prototypes was smaller in magnitude than the other paths.

In this structural model, the demographic variables had only a few effects to the mediators. Males had more friends who smoked ($\beta = .05$, $p < .05$) and adolescents from families with higher education had fewer friends who smoked ($\beta = -.11$, $p < .001$). Compared to Asian-American adolescents, Caucasian adolescents had more favorable prototypes of smokers ($\beta = .10$, $p < .001$) and engaged in more marijuana use ($\beta = .09$, $p < .001$).

For the sensitivity analysis we fit a conceptually similar structural model that had the same specifications but was based on all participants (including T1 smokers), utilized metric scores for T1 e-cigarette use and T2 smoking, and included a path from T1 smoking score to T2 smoking score. This model examined the T1-T2 residual smoking score as a function of residuals for the mediators, thus testing for change in smoking over time controlling for initial level. This model (Figure 1B) had excellent fit, with chi-square (56 *df, N* = 2,772) of 92.58, CFI = .99, and RMSEA = .014. Results from this analysis were quite similar to those for the dichotomous model. The only differences were that the path from prototype change to T2 smoking score was smaller in magnitude ($\beta = .04$, $p = .05$) and the direct effect was almost zero ($\beta = 0.01$, p = .78). Results for tests of indirect effects were parallel to those for the other model, with the path through prototypes marginally significant (p = .06) whereas paths for the other mediators were all significant ($p \leq .001$).

In the autoregressive models there might conceivably be temporal ambiguity in the relation between the mediators and smoking onset; if the smoking onset occurred well before T2, then smoking behavior could have influenced one or more of the mediators (e.g., affiliation with peer smokers). To address this issue we analyzed prospective models, in which only T1 values of the mediators were included and smoking at T2 was the criterion (Figures 2A and 2B). The prospective model with a binary criterion variable (i.e., onset model), based on initial nonsmokers and estimated with WLSMV (Figure 2A), had chi-square (32, N = 1,984) = 40.26, CFI = .99, and RMSEA = .011, all indices representing good fit. Paths from T1 expectancies and marijuana use to T2 smoking onset were significant ($p \leq .01$) as were their indirect effects ($p \leq .01$). Paths from T1 prototypes and peer affiliations to smoking onset were nonsignificant. In this model, a significant direct effect was indicated from e-cigarette use to smoking onset ($p < .0001$) because variance accounted for by change in the mediators was removed from this model. This model (Figure 2A) accounted for 18% of the variance in smoking onset compared to 34% for the autoregressive model (Figure 1A), so modeling T1-T2 mediator change made a substantial incremental contribution for predicting smoking onset.

The prospective model with continuous scores, presented in Figure 2B, also had good fit, with chi-square (40 df, N = 2,772) = 84.69, CFI = .99, and RMSEA = .018. Results were similar to those for the model in Fig. 1B. Paths from e-cigarette use to the T1 mediators were significant in all cases, and paths from the T1 mediators to the T2 smoking score were

significant ($p < .05$) excepting the path for prototypes. (In this model there were paths from T1 smoking score to the T1 mediators; these were modeled as covariances in the autoregressive models.) Tests of indirect effects were parallel to results for the autoregressive model, with significant indirect effects ($p \leq .04$) through smoking expectancies, peer affiliations, and marijuana use and a nonsignificant indirect effect through prototypes. Thus in three of four cases the T1 mediators were significant predictors of smoking onset by T2 and these were strictly temporally antecedent to the outcome.

Discussion

This research was conducted to understand how e-cigarette use is related to onset of cigarette smoking. We followed a sample of adolescents over a 1-year period and studied four factors that were hypothesized to be involved in mediating the effect of e-cigarette use for smoking onset. The analytic model was a conservative one, testing how e-cigarette use was related to residual values for hypothesized mediators controlling for initial level, and how this change in mediators was related to smoking onset. Consistent with our predictions, the results showed e-cigarette use was significantly related to both cognitive and social variables that are risk factors for smoking. Tests of mediation effects demonstrated that changes in expectancies, peer affiliations, and marijuana use constituted significant pathways for the relation between e-cigarette use and smoking onset, and the findings on mediation pathways were replicated across two different types of structural models. It should be noted that these are independent pathways because the intercorrelations among the mediator constructs (Table 3) are partialled for the proximal effects in the models.

Cognitive Processes as Mechanisms

We hypothesized that cognitive factors could mediate the effect of e-cigarette use and the results provided some confirmation for this postulate, showing that increases in favorable expectancies about smoking represented a significant pathway from e-cigarette use to smoking onset. The sensory effects of e-cigarette use through the process of inhaling and exhaling and through the taste of components in the e-cigarette liquid may be involved in stimulating positive expectancies about smoking tobacco cigarettes. Whether relaxation and stress reduction are involved in this process is not clear but these have been noted as salient reasons for e-cigarette use in studies with slightly older samples (Pokhrel et al., 2014, 2015).

Results for the indirect pathway involving prototypes of smokers were marginal. While the path from prototypes to smoking onset was substantial, the path from e-cigarette use to prototypes of smokers was smaller in magnitude. While this could have been affected by statistical issues (prototypes of smokers were substantially correlated with the other mediators) it may also derive from perceptions of smoking. There is a secular trend toward more negative perceptions of smoking (Johnston et al., 2014, Chap. 8) so adolescent e-cigarette users may have identities that are somewhat distinct from their perceptions of cigarette smokers (Hertel & Mermelstein, 2012). Hence any effect of e-cigarette use on perceptual variables might not generalize strongly to cigarette smoking. This question could be clarified through further research on prototypes of e-cigarette users and the role of social-

cognitive variables in e-cigarette use (Shadel & Cervone, 2011; Wills, Sussman, & McGurk, 2015).

Social and Problem-Behavior Processes as Mechanisms

Social factors are likely to be an important part of initiation processes and the results showed that an increase in affiliation with peers who smoked represented a significant pathway from e-cigarette use to smoking onset. We suspect that this occurs in part because peer groups that contain e-cigarette users also contain some cigarette smokers, who can provide cigarettes and model smoking-related attitudes and behavior. It is also possible that teens who have started smoking are interested in situations where e-cigarettes are present. Studying the interface between contexts of use for the two substances (Hoffman et al., 2006; Pokhrel et al., 2015; Simons-Morton & Farhat, 2010) may be a useful direction for further research.

Problem behavior theory postulates an attitudinal basis for the correlation among involvement in different deviant behaviors. Although similarity in physiological responses to inhaled substances could be part of the mechanism for smoking onset, we think that attitudinal variables may play a significant role. As a lower-risk adolescent adopts e-cigarette use, he/she would be susceptible to viewing more deviant behaviors in a positive light and this would encourage initiation of cigarette smoking and possibly other substance use. The present findings could reflect a process in which lower-risk adolescents are attracted to e-cigarettes because they are perceived as less risky and less socially sanctioned (Hanewinkel & Isensee, 2015; Wills et al., 2015); however, once a teen has initiated then attitudinal changes may influence movement to substances such as marijuana. Because attitudes towards marijuana are themselves changing (Johnson et al., 2016), further research is warranted to study the effect of e-cigarette use on constructs from problem behavior theory (e.g., tolerance for deviance) and their possible role in use of other substances.

Context of Onset

The general picture emergent from this research is that adolescent e-cigarette use can involve cognitive, social, and possibly physiological components (Barrington-Trimis et al., 2015; Grana et al., 2014; Leventhal et al., 2016; Wills et al., 2015) and various psychosocial processes operate in concert to encourage movement toward use of tobacco. It is also possible that physiological effects of nicotine (contained in e-cigarettes) are part of an addictive process that contributes to smoking onset (Primack et al., 2015) and the adolescent brain is particularly sensitive to nicotine (England et al., 2015; Yuan et al., 2015). In the present models a direct effect from e-cigarette use to smoking onset, which in principle could represent a nicotine effect, did not reach statistical significance; however, such effects should be tested in other studies with various designs.

The autoregressive models have the advantage of testing for change in mediators over time, but carry a possible temporal ambiguity if mediator change were to overlap with smoking onset. To deal with this issue we analyzed two prospective models, which had only T1 values for the mediators. Results showed that T1 values for three of the mediators were significant predictors of smoking onset. This demonstrates clearly that higher levels of these variables were temporally prior to smoking onset. The prospective models produced an

additional finding: A comparison of results showed that modeling change in the mediators accounted for more of the variance in smoking onset (34% for the autoregressive model compared with 18% for the prospective model). Levels of the mediators can and do change over time, and addressing this in the analyses resulted in much better prediction of smoking onset.

It should be noted that some aspects of this research represent possible limitations. The measure of e-cigarette use was a relatively simple one but the manufacturing field is continually evolving, with different types of products now on the market. Further research should give attention to assessing specifically the type of product used and the social context in which use occurs. Our longitudinal study assessed participants at one period in adolescence, and while onset effects have been found at several ages (Primack et al., 2015; Unger et al., 2016), research is needed to study effects of e-cigarette use at other developmental periods. The present study did not include a measure of nicotine content in the devices used by the participants, and further research with detailed measures of nicotine intake and dependence symptoms is needed to obtain information about the relative role of psychosocial factors and nicotine-linked processes for onset of cigarette smoking.

Implications

This research demonstrated that several psychosocial processes are implicated in e-cigarette use and smoking onset. We think this has implications for regulation of e-cigarettes and for educational programs designed to decrease the appeal of these products to adolescents. In particular, educational programs could emphasize how e-cigarette use sets in motion subtle effects on attitudes and affiliations that place an adolescent at risk for cigarette smoking. How aware persons are of these effects is not known but effects of e-cigarette marketing on implicit attitudes have been demonstrated (Pokhrel et al., 2016) and this is a topic of interest for preventive intervention research.

Acknowledgments

This work was supported by grants R01 CA153154 and P30 CA071789-16S2 from the National Cancer Institute.

We thank the Hawaii Department of Education for their support, the parents and students for their cooperation, and Rebecca Knight for supervising the data collection.

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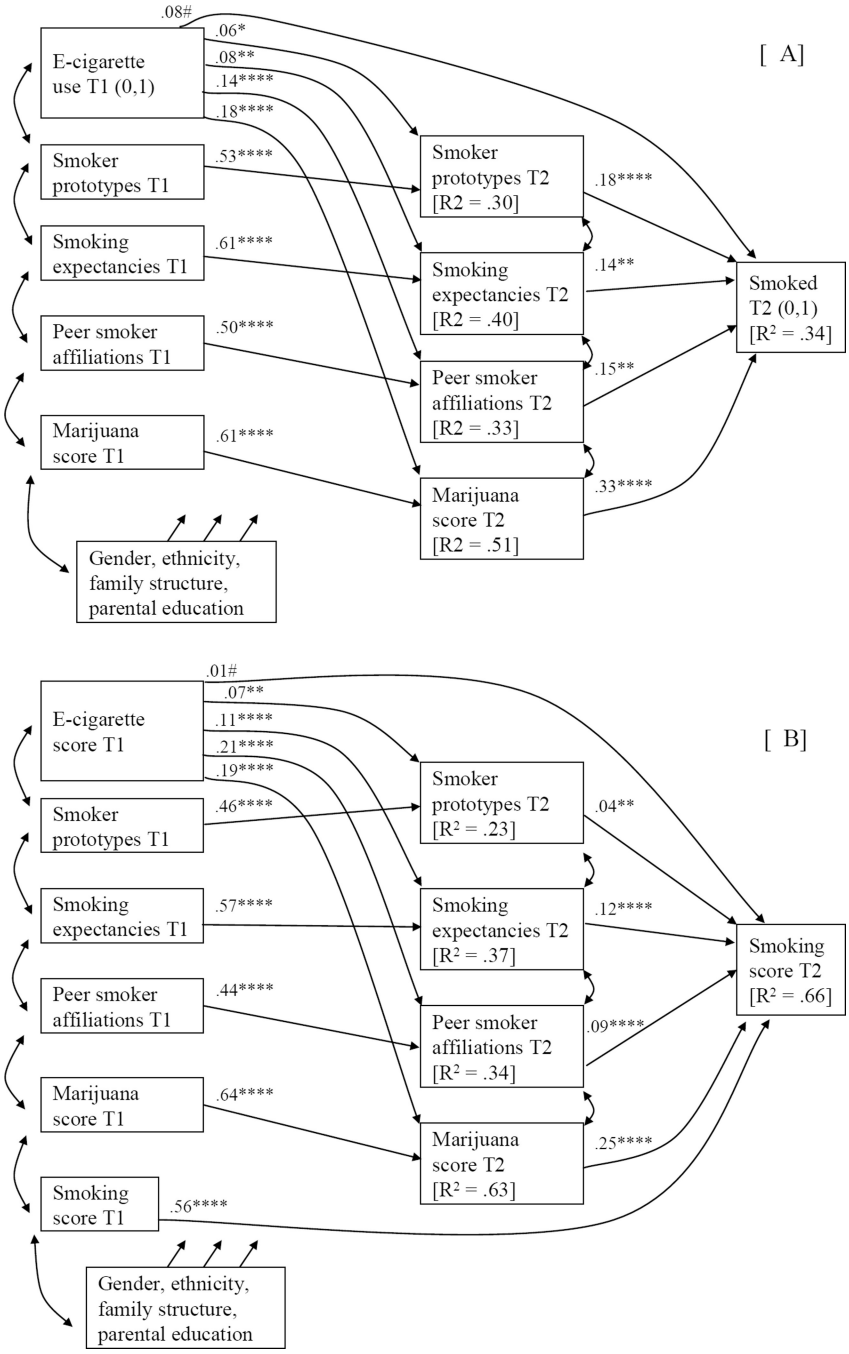

Figure 1.

Dichotomous and continuous structural models for e-cigarette use and smoking onset. Values in figures are standardized coefficients. Straight single-headed arrows are path coefficients; curved double-headed arrows are covariances. All covariances were included in the model but some covariances were excluded from the figure for graphical simplicity. Values in circles at top of figure are R^2s, the variance accounted for in a given construct by all variables to the left of it in the model. * $p < .05$; ** $p < .01$; *** $p < .001$; **** $p < .0001$; # indicates ns.

A: Autoregressive model for smoking onset (T1 and T2 values for mediators), dichotomous e-cigarette predictor and smoking criterion. Data are for onset sample, **excluding** initial smokers (N = 1,984).

B: Autoregressive model for smoking onset (T1 and T2 values for mediators), continuous e-cigarette predictor and smoking criterion. Data are for total sample, **including** initial smokers (N = 2,772).

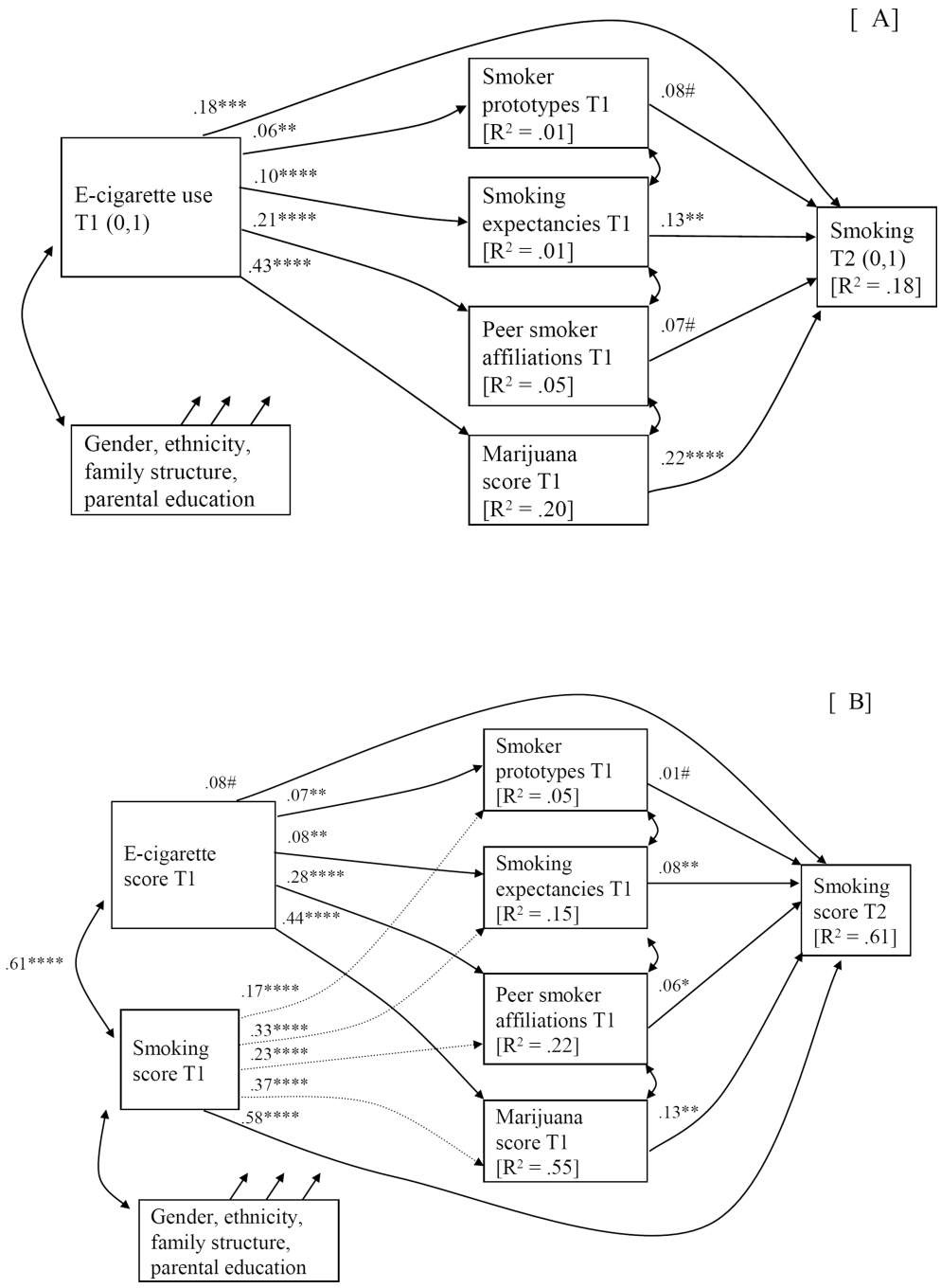

Figure 2.

A: Prospective model for smoking onset (only T1 values for mediators), dichotomous e-cigarette predictor and smoking criterion. Data are for onset sample, **excluding** initial smokers (N = 1,984).

B: Prospective model for smoking onset (only T1 values for mediators), continuous e-cigarette predictor and smoking criterion. Data are for total sample, **including** initial smokers (N = 2,772).

Table 1

Descriptive Statistics for Mediator Variables

Variable	Range	M	SD	Skew
Smoker prototype T1	3–15	6.26	2.71	0.48
Smoker prototype T2	3–15	6.11	2.73	0.51
Smoking expect. T1	6–30	9.16	5.27	1.73
Smoking expect. T2	6–30	9.83	5.55	1.40

Note: Data for T1 never-smokers. Expect. = expectancies. N = 1,984 approximately.

Table 2

Correlations of Model Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. E-cigarette use T1	.[1]															
2. Gender	.05	.[2]														
3. Native Hawaiian	.13	−.03	.[3]													
4. Filipino	.06	.00	−.30	.[4]												
5. Caucasian	−.03	.05	−.23	−.30	.[5]											
6. Other ethnicity	.03	.00	−.15	−.19	−.15	.[6]										
7. Parental education	−.13	.00	−.21	−.13	.09	.01	.[7]									
8. Smoker prototype T1	.07	−.05	.01	.02	−.02	.04	−.06	.[8]								
9. Smoking expect. T1	.11	.02	−.01	.00	.01	−.03	.00	.20	.[9]							
10. Friends smoke T1	.19	.05	.06	−.04	.01	.02	−.06	.25	.17	.[10]						
11. Marijuana use T1	.41	.01	.12	−.08	.06	.06	−.05	.09	.09	.17	.[11]					
12. Smoker prototype T2	.06	.01	−.04	.03	.08	.02	−.07	.48	.18	.23	.15	.[12]				
13. Smoking expect. T2	.11	.03	−.08	.03	.03	.02	.01	.20	.59	.10	.16	.27	.[13]			
14. Friends smoke T2	.24	.09	.06	−.02	.02	.04	−.15	.20	.10	.48	.22	.33	.18	.[14]		
15. Marijuana use T2	.41	−.01	.05	−.08	.13	.05	−.13	.11	.14	.22	.64	.22	.18	.30	.[15]	
16. Smoke T2	.29	−.02	.13	−.02	.12	.05	−.12	.15	.19	.19	.32	.36	.27	.35	.46	.[16]

Note. Data for never-smokers at T1, $N = 1{,}984$. Expect. = expectancies. Approximate significance levels are: $r > |.06|$, $p < .01$; $r > |.08|$, $p < .001$; $r > |.10|$, $p < .0001$.

Table 3

Residual Correlations of Endogenous Variables

	1.	2.	3.	4.
1. Prototypes	.xx			
2. Expectancies	.28	.xx		
3. Peer affiliations	.33	.16	.xx	
4. Marijuana use	.25	.16	.24	.xx

Note: Tabled entries are standardized values, i.e., correlations.

Table 4

Critical Ratios for Tests of Indirect Effects for Smoking Onset in Structural Modeling Analysis

Pathway	Indirect effect (*b'*)	*SE*	Critical ratio	*p*
E-cig use => Prototypes => Onset	0.029	0.016	1.80	.07
E-cig use => Expectancies => Onset	0.028	0.013	2.14	.03
E-cig use => Affiliations => Onset	0.054	0.020	2.72	<.01
E-cig use => Marijuana => Onset	0.143	0.027	5.28	< .0001

Note: E-cig = e-cigarette. Values are unstandardized effects.