

The Unique Roles of Intrapersonal and Social Factors in Adolescent Smoking Development

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Adolescence is a vulnerable period for the initiation and peak of many harmful risk-taking behaviors such as smoking, which is among the most addictive and deadliest behaviors. Generic metatheories like the theory of triadic influence (TTI) suggest that interrelated risk factors across multiple domains (i.e., intrapersonal and social/environmental) jointly contribute to adolescent smoking behavior. Yet, studies are lacking that investigate risk factors across different domains in the same study, which obscures whether each makes a unique contribution to the increase in smoking throughout adolescence or whether there is overlap across the domains. Hence, to fill this gap using a latent growth approach, the current accelerated longitudinal study investigated the collective contribution of multiple intrapersonal and social risk factors in the development of smoking behavior from ages 12 to 17 in 574 ethnically diverse Dutch adolescents. Results from the latent growth model showed that whereas the contribution of motivational-intrapersonal factors like sensation-seeking was no longer significant in the stringent multivariate model, higher levels of impulsivity (cognitive-intrapersonal) and overt peer pressure (social) at age 12 proved to be robust and unique predictors of linear increases in adolescent smoking up until age 17. Consistent with the TTI, adolescent smoking progression does not occur in isolation and the determinants are wide-ranging as they stem from both intrapersonal and social domains. Thus focusing on such confluence of intrapersonal and social risk factors via prevention programs from as young as age 12 might halt the deadly increase in smoking behavior throughout adolescence.

Keywords: smoking, adolescence, cognitive, motivational, social

Adolescence is marked by significant changes in the intrapersonal and social domains. Concurrently, susceptibility to engage in harmful and addictive risk-taking behaviors increases as well (Steinberg, 2010). A pertinent example of such a risk-taking behavior is *smoking* (e.g., Baker, Brandon, & Chassin, 2004; Park, 2011), as nicotine is often regarded as one of the most addictive substances and it is related to a host of health complications (Centers for Disease Control and Prevention,

2010). Considering that adolescents are particularly susceptible to addiction (Chambers, Taylor, & Potenza, 2003; Paus, Keshavan, & Giedd, 2008), it is imperative that risk factors associated with the increase in smoking in adolescence are identified. Metatheories like the theory of triadic influence (TTI; Flay & Petraitis, 1994; Petraitis, Flay, & Miller, 1995) posit that adolescent smoking does not occur in isolation and the determinants are wide-ranging as they stem from multiple interrelated intrapersonal and nonintrapersonal domains. However, risk factors across different domains are hardly investigated simultaneously within a single study. Hence, via a latent growth design, the current accelerated longitudinal study aims to investigate the unique roles of multiple intrapersonal and social risk factors in predicting the hypothesized increase in smoking among 574 adolescents from 12 to 17 years of age.

TTI

The TTI is a comprehensive theory that integrates risk-factors from multiple domains (intrapersonal, social, and environmental) that are derived from numerous sociological and psychological theories about onset and change of adolescent substance use, such as smoking and alcohol use (Flay, 1999; Flay & Petraitis, 1994; Petraitis et al., 1995). More recently the TTI has been used as a framework in research examining other addictive behaviors like

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gambling, or other risk-taking behaviors like risk-taking in traffic (for an overview, see, e.g., Snyder & Flay, 2012).¹ Intrapersonal factors can include cognitive functions, impulsivity, affective states thrill/sensation seeking that influence self-efficacy and internal motivation to use substances, and via decision making these intrapersonal factors ultimately predict the use of substances (Petraitis et al., 1995). Social factors can include parent and peer influences (e.g., pressure to engage in substance use), which lead adolescents to comply with others and ultimately cause them to decide to engage in substance use (Petraitis et al., 1995). Finally, environmental factors include aspects of adolescents' neighborhood, cultures, general values that influence adolescents' belief and evaluations about the costs and benefits of substance use, which ultimately lead adolescents to decide to use substances (Petraitis et al., 1995). Thus TTI is a broad and complex theory that aims to emphasize the complete puzzle of causation of youth substance use (Flay, Snyder, & Petraitis, 2009).

Similar to ecological models, TTI proposes that intrapersonal factors are embedded within social factors which, are in turn embedded within broader cultural-environmental factors that contribute to attitudes about risk-behaviors (Snyder & Flay, 2012). However, unlike most models, TTI suggests that these three domains have different distances/levels from actual smoking behavior, labeled as ultimate (i.e., underlying), distal (i.e., predisposing), or proximal (i.e., immediate) levels of causation (Flay et al., 2009; Snyder & Flay, 2012). In the current article we investigate intrapersonal (cognitive and motivational factors) and social factors (susceptibility to peer influence and perceived peer pressure) that overlap with the TTI.

Intrapersonal Domain

According to the TTI, intrapersonal influences are hypothesized to affect (a) skills adolescents need to deal with situations when they offered cigarettes or other means of smoking, (b) adolescents' determination/intention whether or not to smoke, and (c) adolescents' smoking self efficacy/behavioral control (Flay, Petraitis, & Hu, 1995). For the current study we investigate four of the many factors related to the intrapersonal stream: impulsivity, inhibitory control, sensation seeking, and reward seeking. Considering that this domain is very broad and consists of wide-ranging personality traits, we further subdivide intrapersonal factors into primarily cognitive-related behaviors (cognitive control: impulsivity and inhibitory control), and primarily motivational behaviors (reward sensitivity: reward seeking and sensation seeking).

Cognitive Factors

Cognitive control is an umbrella term for wide-ranging executive functions (e.g., inhibitory control, impulsivity, working memory). Acquiring cognitive control facilitates the achievement of both short-term and long-term goals via adaptively organizing and coordinating thoughts and actions, especially in response to changing environmental contexts (Crone & Dahl, 2012; Luna, Garver, Urban, Lazar, & Sweeney, 2004). In the current article we investigate inhibitory control and impulsivity aspects of cognitive control. Studies aiming to tap inhibitory control often employ the classic behavioral go/no-go task, which requires participants to inhibit motoric responses (for a review, see Casey & Caudle, 2013;

Geier & Luna, 2009). On the contrary, (*reflection-*)*impulsivity*, which is described as behavior resulting from a lack of forethought, is typically assessed via self-report questionnaires (Dalley, Everitt, & Robbins, 2011). The subjective (self-report) and objective (behavioral) measurements of cognitive control often fail to be related to each other, however, suggesting that there are multiple latent aspects to cognitive control (Buckholz, 2015; Dalley et al., 2011). Accordingly, in the current study we use a questionnaire that taps lack of forethought and a behavioral measure (i.e., go/no-go task) to measure cognitive control. The TTI considers such cognitive-related factors as "ultimate level" influences within the intrapersonal domain (see Petraitis et al., 1995).

In relation to adolescent smoking development, as far as we know, there are no empirical studies on whether lower levels of inhibitory control (assessed via objective/behavioral measurements) predict subsequent smoking development from early to late adolescence (but see, e.g., Reynolds, Karraker, Horn, & Richards 2003 for delay discounting in relation to smoking in adolescence). Nevertheless, cross-sectional studies containing late adolescents and emerging adults (17–25 years; mean age = 18.60) have shown that nonsmokers perform worse than smokers on the go/no-go task (although smokers had higher levels of self-reported impulsivity compared to nonsmokers; Dinn, Aycicegi, & Harris, 2004; see also Galvan, Poldrack, Baker, McGlennen, & London, 2011). In seeming opposition, a recent meta-analysis found that lessened inhibitory control as measured by the go/no-go does predict smoking abuse/addiction in adult samples (Smith, Mattick, Jamadar, & Iredale, 2014). Thus, although longitudinal studies spanning early to late adolescence are nonexistent, the summarized mixed results could suggest that there are developmental differences underlying the relationship between inhibitory control and smoking since the predictive power of the go/no-go for smoking appears to differ for late adolescents and emerging adults versus more mature adult samples.

Next, although cross-sectional studies consistently find that self-reported impulsivity is related to smoking (for a review, see Dawe, Gullo, & Loxton, 2004), only a handful of longitudinal studies have investigated this link with adolescent samples (e.g., Audrain-McGovern, Rodriguez, Tercyak, Neuner, & Moss, 2005; Elkins, King, McGue, & Iacono, 2006; Malmberg et al., 2013; Quinn & Harden, 2013). An example of such a longitudinal study used a latent growth design and showed that impulsivity (labeled as self-control in that study) only had an indirect effect (via baseline peer smoking) on the baseline of smoking when adolescents were in the 9th grade (Audrain-McGovern et al., 2005). However, there was no indirect or direct effect of impulsivity on the progression of adolescent smoking from the 9th grade to the 12th grade (Audrain-McGovern et al., 2005; see also Quinn & Harden, 2013). Yet, another longitudinal study showed that impulsivity traits measured at age 17 predicted new onsets of nicotine dependence at age 20 (Elkins et al., 2006). In sum, whereas impulsivity has consistently been shown to concurrently predict

¹ We were initially interested in focusing on smoking, gambling and traffic risk-taking behavior and collected data on these risk behaviors as well. However, we limit the current article to smoking, since this behavior showed sufficient growth between ages 12–17, making it meaningful for us to look into predictors of this progression of adolescent smoking.

adolescent smoking, evidence of this association is mixed in the limited existing longitudinal studies.

Motivational Factors

Adolescents are hypothesized to be hyper-sensitive to rewarding and highly arousing stimuli, and some posit that this is the result of pubertal development (Forbes & Dahl, 2010; Steinberg, 2004). This so-called “reward sensitivity” is conceptualized as a heightened behavioral motivational tendency to seek out rewards (in other words, sensation-seeking and accordingly reward-seeking), and heightened arousal in response to rewards (Galvan et al., 2011). Obviously, one of the reasons that adolescents engage in risk-taking behaviors like smoking is because it can be both (directly) physically and socially rewarding. Reward-seeking adolescents smoke because they anticipate a reward (Baker et al., 2004). Sensation-seeking, however, has been defined more broadly as the pursuit of diverse novel, complex, and intense sensations or experiences and the willingness to take risks to acquire them (Zuckerman, 1979). To tap reward seeking and sensation-seeking we use two subscales of the Behavioral Approach System Scale (BAS) of the classic Behavioral Inhibition & Activation Questionnaire (BIS/BAS; Carver & White, 1994) that describes these behaviors as “affective responses/reactions”, which are part of a “general motivational system that underlie behavior and affect” (Carver & White, 1994). Within the TTI such motivation constructs are considered as intrapersonal “ultimate-level” influences of smoking (Petraitis et al., 1995).

Numerous cross-sectional studies have shown support for a link between sensation-seeking and adolescent smoking (Leeman et al., 2014; Martin et al., 2002; Pokhrel, Sussman, Sun, Kniazar, & Masagutov, 2010). Similarly, one longitudinal study showed that sensation seeking (termed “risk-taking” in that study) at Grade 5 was predictive of levels of smoking 7 years later (Burt, Dinh, Peterson, & Sarason, 2000). However, a longitudinal latent growth study found that although smoking and sensation-seeking were concurrently associated at age 15/16, changes in sensation-seeking were not associated with changes in smoking from ages 15 to 26 years (Quinn & Harden, 2013). Thus whereas there is consistent support for concurrent associations between sensation-seeking and adolescent smoking, evidence for longitudinal associations are inconsistent.

With regard to the more *specific reward seeking*, a cross-sectional study of 14–25 year olds (mean age 16.11) reported that among multiple personality characteristics, the BAS was the best predictor of a composite substance abuse factor that included smoking (Knyazev, Slobodskaya, Kharchenko, & Wilson, 2004). Likewise, a recent cross-sectional study with college students (18–25 years, mean age 19.41) showed that reward seeking was related to a composite score of substance use that included smoking (Richardson, Freedlander, Katz, Dai, & Chen, 2014). In sum, although longitudinal studies are lacking, there is some cross-sectional evidence that reward seeking predicts smoking in adolescence.

Social Domain

According to the TTI, the social domain includes adolescents’ immediate social surroundings, such as the peer context that con-

tribute to the social pressure adolescents experience to engage or not to engage in smoking (Flay et al., 1995). In the current article we focus on peer influence. The TTI proposes that the peer context contributes to adolescent smoking behavior because peers affect (a) adolescents’ subjective perceptions about the normativeness of smoking, (b) with whom adolescents are motivated to conform their behavior to (e.g., deviant peers), and (c) the social pressures adolescents experience to smoke (Flay et al., 1995). Accordingly, in the current study, we investigate two forms of *peer socialization* (see Simons-Morton & Farhat, 2010) that are consistent with the TTI, namely, whether directly perceived peer pressure and susceptibility to peer influence predict adolescent smoking development. Unlike perceived peer pressure, susceptibility to peer influence is when adolescents adopt peer norms whether or not there is direct/perceived pressure from peers to do so. Within the TTI framework, directly perceived peer pressure (labeled as “pressures to use substances”/“beliefs that important others encourage smoking”) is considered as a “proximal” social influence, whereas susceptibility to peer influence (labeled as “strong desire to please peers”) is considered as a “distal” social influence (Petraitis et al., 1995).

A handful of studies have shown that perceived peer pressure is associated with smoking in both early (e.g., Crockett, Raffaelli, & Shen, 2006) and late adolescents (e.g., Santor, Messervey, & Kusumakar, 2000). Although more longitudinal studies on this link are needed, at least one latent growth study demonstrated that peer pressure (labeled as “peer encouragement”), predicted the initial stage and development of smoking from ages 11 to 18 (Duncan, Tildesley, Duncan, & Hops, 1995). As for susceptibility to peer influence, one study showed that a similar “friend compliance” measure longitudinally predicted adolescent smoking (Otten, Bricker, Liu, Comstock, & Peterson, 2011). Thus there is some evidence that both perceived peer pressure and susceptibility to peer influence are prospective predictors of adolescent smoking development.

Empirical Support for the TTI Framework

One of the primary reasons the TTI was developed was to acknowledge that risk-factors tend to be interrelated, thus multiple risk factors should be simultaneously investigated within a single study (Flay et al., 1995). One of the few longitudinal studies that investigated multiple intrapersonal (e.g., sensation seeking) and social (e.g., peer compliance) factors reported that when tested univariately, although social factors were not significant, sensation seeking measured in Grade 5 was a significant predictor of smoking in Grade 12. However, the effect for sensation-seeking vanished for boys when it was tested in a multivariate model together with “rebelliousness” (Burt et al., 2000), which was a construct that resembled self-regulatory capacities. Another TTI-based study showed that whereas friend compliance and rebelliousness in adolescence predicted smoking progression in young adulthood, thrill seeking (a component of sensation seeking) was not a significant predictor (Otten et al., 2011). Finally, a study using the same sample as Otten et al. (2011) found that scoring high on “friend compliance” contributed a significant probability to the overall probability that an adolescent would try smoking (Transition 1), and transition from the first cigarette to monthly smoking (Transition 2), and from monthly to daily smoking (Transition 3; Bricker et al., 2009). However, thrill seeking was only significant

for Transition 1 and 2, but not for Transition 3 (Bricker et al., 2009). These three studies underscore why risk factors should not be investigated in isolation. This might produce inflated and biased conclusions about their influences. A noteworthy difference between the three summarized studies and the current study is that we additionally investigate risk factors in the cognitive-intrapersonal domain (impulsivity and inhibitory control).

Present Study

In the current study we investigate the developmental pattern of adolescent smoking, and whether TTI-consistent risk-factors in the intrapersonal (cognitive and motivational) and social (peer influence) domains at age 12 concurrently and prospectively predict the variation in the hypothesized growth of adolescent smoking from ages 12 to 17. We hypothesize that linear growth will be detected from ages 12 to 17. The current study adds to the literature by capitalizing on a latent growth design that facilitates the investigation of whether adolescents differ in their initial level and development of smoking (i.e., variance in baseline and progression of smoking), which is neglected in more traditional statistical models. Specifically, we include all predictors in a combined model to ascertain the unique role of each predictor in contributing to the development of smoking in adolescents. In this combined model we additionally account for putative effects of gender and educational track. Finally, an accelerated longitudinal design is used, which provides the advantage of modeling a longer developmental span (i.e., age 12 through 17 years) with the current two cohort sample of 574 adolescents who were either 12–13 years old or 14–15 years old at the beginning of the present 3-year study.

Method

Participants

The sample used in the current article was part of a larger 3-year longitudinal study, in the Netherlands called “The Adolescent Risk-Taking (ART) Project,” which is a research project on adolescent risk-taking in multiple domains that began in 2012. We recruited the participants via schools throughout the Netherlands. In Wave 1, the adolescents ($N = 602$; 46.40% female) were either in the 1st or 3rd year of “preparatory middle-level applied education” (VMBO in Dutch) or “higher general continued education” (HAVO in Dutch). In the first wave, most adolescents (93.2%) reported that they were born in the Netherlands with 61.6% identifying as Dutch, 9.3% as Turkish or Turkish-Dutch, 7.4% as Surinamese or Surinamese-Dutch, and 5.5% as Moroccan or Moroccan-Dutch, and the rest (16.2%) identified with various other ethnicities. In Wave 1 adolescents in the youngest cohort were 12–14 years old and adolescents in the older cohort were 14–17 years old. The number of 16- ($n = 26$) and 17-year-olds ($n = 1$) in the second cohort was very small, so we limited our analyses to the youngest 4 age cohorts. The 12-, 13-, 14-, and 15-year-old cohorts were represented in Wave 1, and the sample sizes were $n = 178, 113, 170,$ and $113,$ respectively, with a total subsample of $N = 574$ for the current study. Via an accelerated longitudinal design procedure, these adjacent cohorts could be linked to form one continuous developmental trajectory spanning ages 12 through 15 during Wave 1. Of this subsample of 574 at

Wave 1, 441 and 349 adolescents took part in Waves 2 and 3, respectively.

Procedure

Participants were recruited from eight high-schools in six different regions in the Netherlands,² the schools were first emailed and then called. We made it a priority to also recruit ethnically diverse schools. Parents received information letters about the research project as well as dissent letters that could be returned to the schools if parents did not want their children to participate in the study. At the beginning of the study, approximately 810 potential students could participate. Of these participants, 9.75% did not have parental permission to participate, the other adolescents who did not participate refused to participate on their own, or were absent during the data-collections due to other conflicts (e.g., illness and thus absent). Adolescents with parental permission who were absent from school in Wave 1, could still partake in future waves, and new adolescents could also join the research after Wave 1.

Data-collection took place at schools, and was led by trained research assistants. Participants could choose to receive a chocolate candy worth 2 euros as a participation prize, or have their name entered in a raffle for a chance to win a 50 euro gift voucher. Data were collected annually for 3 years, with sample sizes across the three waves as 602, 582, and 442, respectively.

Measures

Latent factor models. We constructed latent factors for the variables of interest that consisted of two or more items, since latent factors are a recommended method to reduce measurement error (Kline, 2010). We only used items with sufficient standardized loadings of $> .30$. Thus, it would be redundant to provide information on Cronbach’s alpha’s, and instead, we provide information on the latent factor analysis.

Smoking. Smoking behavior was assessed with the question “Do you smoke tobacco? (cigarette, cigar, shag, [water-]pipe)?”, which was measured on a 6-point scale ranging from 1 (*No, I have never smoked*) to 6 (*Yes, every day*). Adolescents who had never smoked or who have smoked in the past but do not currently smoke were coded as 0 and were included in the analyses (cf. Fuemmeler et al., 2013). Thus we converted the 6-point scale for smoking into a 5-point scale.

Cognitive factors. Impulsivity was assessed with a shortened validated version (Vitaro, Arseneault, & Tremblay, 1997, 1999) of the original Eysenck Impulsiveness Scale (Eysenck & Eysenck, 1978; Eysenck, Easting, & Pearson, 1984) and contained five items that tapped lack of forethought. This questionnaire was translated to Dutch; an example item was, “Do you generally do and say things without stopping to think?” and was measured on a 6-point scale ranging from 1 (*completely disagree*) to 4 (*completely agree*). All items on the impulsivity scale had at least a .30 loading on the factor, indicating adequate factor loadings.

Inhibitory control was assessed with the cued go/no-go task (Fillmore, 2003; Fillmore, Rush, & Hays, 2006), which was pro-

² In Wave 2 and 3 we had seven schools participating as one school did not participate after Wave 1 because of organizational changes at the school.

grammed in OpenSesame (Mathôt, Schreij, & Theeuwes, 2012). Participants were instructed to respond to a go target (green rectangle) and withhold a response for a no-go target (blue rectangle). This task manipulates response prepotency as each target is preceded by a go cue (valid cues) or a no-go cue (invalid cues), and the orientation of these cues provided preliminary information about the probability that an actual go or no go target will occur. Vertically and horizontally presented cues signaled go and no-go cues, respectively. Vertically presented cues preceded the go target in 80% of the trials and preceded the no-go target in the remaining 20% of the trials. Horizontally presented cues preceded the no-go target in 80% of the trials and preceded the go target in the remaining 20% of the trials. Thus the cue feature in this task measures the ability to inhibit instigated “prepotent” responses; invalid cues impair response inhibition whereas valid cues facilitate response inhibition (Fillmore & Weafer, 2013). Particularly, for the invalid go cue trial, participants will typically fail to inhibit responses if a go/no go target appears afterward (Fillmore et al., 2006).

Cues were white (i.e., noncolored) rectangles framed in 0.8 mm black outlines, and were presented in the center of a white background on the computer’s monitor. Cues were presented vertically (height = 7.5 cm, width = 2.5 cm) or horizontally (height = 2.5 cm, width = 7.5 cm). The go and no-go targets were colored green and blue rectangles, respectively (Fillmore et al., 2006).

Trials began with a presentation of a fixation point (+) for 800 ms, after which a blank white screen appeared for 500 ms. Hereafter a cue was presented for one of five stimulus onset asynchronies (SOAs; 100, 200, 300, 400 and 500 ms). Finally, a go or no-go target was presented and remained visible until participants either exhibited a response or did not respond after 1,000 ms. At the end of each trial there was an intertrial interval of 700 ms (Fillmore et al., 2006).

The go/no-go task took approximately 10 min to complete and included 250 trials representing all four possible cue-target combinations an equal number of times. Furthermore, for each of the five SOAs, a cue-target combination was presented and each cue-target combination was separated by an equal number of SOAs. The cue-target combinations and SOAs were presented in a random fashion. Per trial, recordings were made for whether participants elicited a response, and the reaction time (RT; ms) for such responses were recorded (for more detailed information, see Fillmore et al., 2006). In the current study the variable of interest was the proportion of failed inhibitions on a NoGo target following a Go cue (in other words, proportion incorrect key presses to no-go target following go cue).

Motivational factors. Reward seeking and sensation seeking were assessed with two subscales of the BAS (Carver & White, 1994) that have been used in past studies to measure these constructs, namely BAS Drive (four items) and BAS Fun Seeking (four items), respectively. We used a Dutch translated version of the BAS that was validated against the psychometric properties of the original BIS/BAS (Yu, Branje, Keijsers, & Meeus, 2011). Answers to the questions were assessed using a 4-point response format that ranged from 1 (*very false for me*) to 4 (*very true for me*).

Reward seeking was measured with the BAS Drive subscale, which measures the behavioral tendency to persistently pursue rewards and desired appetitive goals and reflects the extent to

which (impending) rewards guide subsequent behavior (Beaver et al., 2006; Carver & White, 1994). An example item of BAS Drive is, “I go out of my way to get things I want.” All items on this scale had sufficient factor loadings.

Sensation seeking was measured with the BAS Fun Seeking subscale, which is typically used to measure sensation seeking tendencies (Franken & Muris, 2006; Ko et al., 2008; Zuckerman, 2012), perhaps primarily because of its additional “novelty seeking” aspect that differentiates it from the other subscales of BAS. An example of an item on the BAS fun-seeking scale is “I crave excitement and new sensations.” We excluded one item (i.e., “I will often do things for no other reason than that they might be fun”) on this scale that had a factor loading of less than .30.

Social factors. *Susceptibility to peer influence* was measured with selected items on the Resistance to Peer Influence Scale (RPI; Steinberg & Monahan, 2007), which is a self-report questionnaire that taps the degree to which adolescents are resistant to influence of their peers (Steinberg & Monahan, 2007). The psychometric properties of this scale have been cross-validated in a Dutch sample of adolescents (see Sumter, Bokhorst, Steinberg, & Westenberg, 2009). Eight of the 10 pairs of opposing statements in the RPI were selected to be used in the current study. Participants were instructed to first choose one of the answers per pair that described them, and thereafter decide whether their choice is “really true” or “sort of true.” For example, a pair of two statements was, “Some people would do something that they knew was wrong just to stay on their friends’ good side” versus “other people would not do something they knew was wrong just to stay on their friends’ good side.” A higher score indicates higher resistance to peer pressure. The item

Some people think it’s better to be an individual even if people will be angry at you for going against the crowd vs. Other people think it’s better to go along with the crowd than to make people angry at you.

Had a factor loading lower than .30, and was thus excluded.

Perceived peer pressure was measured with the reliable and validated Peer Pressure Inventory (PPI; Clasen & Brown, 1985), which measures different types of peer pressures adolescents perceived as well as the intensity of the perceived peer pressure. Participants were presented with pairs of opposing statements concerning peer pressure. Per pair, they were instructed to choose the statement that corresponds with their experience, and then indicate to what extent that statement is true for them (i.e., “a little,” “somewhat” or “a lot”). Participants could also choose the option “no pressure” if they did not perceive any pressure from their friends to participate (or not to participate) in a particular behavior. Ten pairs of statements on the PPI that were selected as relevant for the larger longitudinal study on risk-taking, were used in the current study. Four items were related to substance use, two items measured vandalism and stealing, one item was related to school involvement, another item measured peer conformity and one item measured obedience toward parents. An example of a pair of statements is, Pressure to Smoke cigarettes versus Pressure not to smoke cigarettes. Lower scores on the PPI indicated higher levels of perceived peer pressure. Our factor analysis showed that the following three items on the Peer Pressure Scale had very poor factor loadings (i.e., below .30): peer pressure to study/do home-

work, peer pressure to shoplift/steal, and peer pressure to engage in vandalism. Thus we excluded these items.

Statistical Approach

Accelerated latent growth model. Latent growth modeling (LGM) is a comprehensive, powerful and flexible statistical technique for studying parametric development in both individuals and the sample as a whole (Duncan, Tildesley, Duncan, & Hops, 1995). In the current study we apply an accelerated longitudinal (also called cohort sequential) approach to model the hypothesized growth in smoking throughout adolescence using a latent growth model. Accelerated longitudinal designs consist of multiple independent and overlapping age cohorts that are statistically converged into one growth curve. In the current study, we used a multigroup framework for our cohort sequential models. Across groups, equality constraints were imposed on all free parameters (for details, see, e.g., Duncan, Duncan, & Strycker, 2006). As reported earlier, adolescents who were 12 during Wave 1 constituted the “age 12 cohort,” similarly we also had an age group cohort for 13-year-olds, 14-year-olds, and 15-year-olds.

We estimated a hybrid model that included a factor model for our predictors, with latent variables that were used as predictors for the variance in the slope. Specifically, we used a four-step procedure for the LGM analyses. In the first step we investigated an unconditional growth curve, without predictors (cf. Duncan et al., 1995). In a second step, we entered the control variables (i.e., gender and educational track) in the model, by regressing the intercept and slope on these control variables. In a third step, we investigated whether the independent variables individually predicted the growth parameters, that is, we regressed the intercept and slope on the level of the independent variables at age 12. This resulted in six models, namely one model per independent variable. In the fourth step, we included all predictors in one combined model, along with the potential covariates gender and educational track.

In subsequent analyses hereafter, we performed a stepwise backward elimination procedure based on p values of the predictors in order to come to a final combined model (Duncan et al., 2006). In other words, per step, we deleted the predictors with the highest p values until a model with only significant predictors remained. Consistent with this format, each predictor is treated as if it were added last to the model (Duncan et al., 2006). This procedure is recommended as it takes multicollinearity between variables into account and it avoids the deletion of relevant predictors (Duncan et al., 2006).

Furthermore, a robust maximum likelihood estimator was used for all models (Satorra & Bentler, 1994) to account for non-normality and to ensure that incomplete data could be included in the analyses. Any item-missing or wave-missing data were dealt with using the Full Information Maximum Likelihood algorithm in Mplus 7.1 (Muthén & Muthén, 2012).

Results

Preliminary Analyses

Gender was entered in the models as a dichotomous variable (boy = 0; 1 = girl), and educational track was also dichotomized

(VMBO/lower educational track = 0; HAVO/higher educational track = 1). Next, we excluded unreliable smoking data for 8.89% of the subsample who gave inconsistent answers about their smoking history (i.e., participants who indicated that they had no experience with smoking, who in previous years indicated that they did have experience with smoking).

We also ran some bias checks to determine whether persons who dropped out the study (i.e., 15% of participants) after Wave 1 were different from persons who remained in the study, with respect to gender, educational track and smoking levels. We controlled for age when examining smoking. There were no significant results for gender. However, for educational track and smoking, there were some significant differences. Students who only participated in the first wave did not differ from the other students with respect to educational track, however, they were heavier smokers, $F(1, 519) = 10.52, p < .01$. Compared to Wave 1, students who did not participate at the second wave were from a higher educational track, $\chi^2(1) = 5.24, p = .02$, and were heavier smokers, $F(1, 519) = 5.57, p = .02$; whereas students who did not participate at the third wave were more likely to be from the lower educational tracks, $\chi^2(1) = 19.33, p < .01$, but they did not differ in their smoking levels. The results with respect to educational track are to be expected at Wave 2, considering that one school that had students attending higher educational tracks dropped out of the study (as mentioned earlier). The results with respect to educational track are also to be expected at Wave 3, because by that time some participants of the lower educational tracks had already finished high school, and thus did not take part in the school data-collections. Overall, these findings suggest that the sample of students who dropped out of the study were more likely to be heavier smokers.³

Table 1 illustrates the correlations among the individual items at baseline (age 12). Only inhibitory control was significantly correlated with smoking at age 12.

Main Analyses

Fit indices for the unconditional model (Step 1), $\chi^2(32) = 42.74; p = .10$; Tucker-Lewis index (TLI) = .95 and root mean square error of approximation (RMSEA) = .05, suggest that an accelerated longitudinal design is suitable for our data. Furthermore they suggest that the growth in smoking can be described adequately with a linear model. The intercept was not significantly different from 0 ($b_0 = .04; p = .27$), indicating that at age 12, nearly all the adolescents did not currently smoke (93.6%). However, at age 17 a total of 29.40% adolescents reported that they currently smoke. It should be noted that the lack of variance in the intercept produced estimation problems. Thus we constrained the variance of the intercept to 0, which solved these problems (see the above-mentioned model fit indices). Next, the slope was significant ($b_1 = .20; p < .01$) and there was significant variance in the slope (variance (b_1) = .15; $p < .01$). These results indicate that the

³ However, the readers should bear in mind that participants who did not participate in one wave were still allowed to participate in subsequent waves, and a cohort-sequential design which results in some overlapping age cohorts was used. Thus it is not straightforward to conclude to what extent these results can be interpreted and to what extent they are meaningful for our SEM models.

Table 1
Bivariate Concurrent Correlations for the Individual Items at Age 12

Items	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	
1. sm	—																												
2. i1	.03	—																											
3. i2	.09	.50 [†]	—																										
4. i3	.08	.58 [†]	.56 [†]	—																									
5. i4	.13	.60 [†]	.39 [†]	.55 [†]	—																								
6. i5	.13	.52 [†]	.41 [†]	.48 [†]	.54 [†]	—																							
7. rs1	-.03	-.03	.05	-.16*	-.14	-.10	—																						
8. rs2	.08	.16*	.11	.14	.10	.00	.21 [†]	—																					
9. rs3	.07	-.02	-.11	-.02	.14	.04	.14	.22 [†]	—																				
10. rs4	-.02	.04	.07	-.04	-.05	-.07	.42 [†]	.22 [†]	.18*	—																			
11. ss1	.07	.11	.17*	.18*	.13	.15*	-.11	.31 [†]	.05	.12	—																		
12. ss2	-.01	.08	.05	.02	-.04	-.05	-.18*	.14	.27 [†]	.32 [†]	.14	—																	
13. ss3	.05	.13	.24 [†]	.13	-.02	.07	-.08	.04	.12	.14	.14	.11	—																
14. ps1	.10	-.08	-.14	-.16*	-.07	-.04	.18*	-.19*	.18*	.05	-.28 [†]	-.04	-.09	—															
15. ps2	-.02	-.28 [†]	-.18*	-.30 [†]	-.23 [†]	-.26 [†]	.23 [†]	-.10	.13	.23 [†]	-.31 [†]	-.01	-.02	.36 [†]	—														
16. ps3	-.08	-.23 [†]	-.20*	-.26 [†]	-.19*	-.22 [†]	.13	-.21 [†]	.05	.01	-.29 [†]	-.06	-.07	.29 [†]	.38 [†]	—													
17. ps4	.00	-.24 [†]	-.04	-.18*	-.18*	-.18*	.11	-.15	.04	.11	-.18*	.02	-.01	.28 [†]	.20*	.45 [†]	—												
18. ps5	-.10	-.05	-.17*	-.11	-.12	-.12	.04	-.08	-.01	.06	-.06	-.02	-.07	.05	.18*	.28 [†]	.31 [†]	—											
19. ps6	-.10	-.30 [†]	-.24 [†]	-.23 [†]	-.34 [†]	-.25 [†]	.06	-.07	-.02	.10	-.17*	-.10	-.15	.27 [†]	.35 [†]	.39 [†]	.30 [†]	.33 [†]	—										
20. ps7	-.15	-.28 [†]	-.31 [†]	-.29 [†]	-.27 [†]	-.35 [†]	.20*	-.05	.03	.05	-.34 [†]	.08	-.12	.19*	.30 [†]	.44 [†]	.32 [†]	.30 [†]	.41 [†]	—									
21. pp1	-.02	-.08	-.07	-.08	-.11	-.18*	-.03	-.03	-.06	.00	.03	.00	.12	-.05	.00	.14	.02	.19*	.06	.15	—								
22. pp2	-.06	-.14	-.12	-.11	-.17*	-.21 [†]	.04	-.04	-.06	.11	-.06	.02	.08	-.02	.14	.23 [†]	.11	.26 [†]	.22 [†]	.22 [†]	.75 [†]	—							
23. pp3	-.03	-.03	.01	-.01	-.05	-.07	-.07	-.22 [†]	-.05	-.10	-.15	-.05	.04	.13	.15	.21*	.10	.13	.10	.08	.35 [†]	.40 [†]	—						
24. pp4	-.13	-.11	-.12	-.10	-.17*	-.17*	.01	-.04	-.09	.06	.00	-.04	.02	-.04	.05	.14	.11	.23 [†]	.19*	.19*	.73 [†]	.85 [†]	.41 [†]	—					
25. pp5	-.02	-.08	-.08	-.08	-.05	-.20*	.02	.03	-.02	.11	.05	.04	.06	-.06	.03	.14	.05	.22 [†]	.10	.19*	.73 [†]	.81 [†]	.40 [†]	.86 [†]	—				
26. pp6	-.04	-.14	-.04	-.07	-.08	-.15	-.02	-.05	.05	.16*	-.05	-.01	.05	.01	.19*	.22 [†]	.18*	.06	.08	.13	.33 [†]	.44 [†]	.31 [†]	.46 [†]	.45 [†]	—			
27. pp7	-.06	-.10	-.10	-.07	-.07	-.09	.11	.03	.16	.00	-.04	.08	-.01	.15	.04	.25 [†]	.18*	.14	.09	.27 [†]	.25 [†]	.29 [†]	.25 [†]	.28 [†]	.27 [†]	.25 [†]	—		
28. ic	.25*	.06	.13	-.04	.10	.16	.09	.06	.12	.11	.01	.16	-.14	.00	-.03	-.19	.02	-.25*	-.04	-.09	-.18	-.27*	-.03	-.17	-.13	.09	-.16	—	

Note. sm = smoking; i = impulsivity; rs = reward seeking; ss = sensation seeking; ps = susceptibility to peer influence; pp = peer pressure; ic = inhibitory control. The number behind the labels of the items denotes the item number on the scale.
[†] $p < .01$. * $p < .05$.

mean level of smoking significantly increased each year with .20 units further on our 5-point scale for smoking. Thus at age 17, adolescents progressed on average 1 unit further on our 5-point scale. The significant variance indicates that adolescents differ in their rate of increase of smoking. In the second model that included the control variables, we found that educational track (but not gender) predicted faster increases in smoking. Specifically, adolescents with a lower educational track at age 12, showed faster increases in smoking behavior over time.

The separate models per predictor (i.e., step 3) showed that higher levels of impulsivity, sensation seeking, perceived peer pressure, and susceptibility to peer influence at age 12 predicted faster increases in smoking behavior. Results in the final combined model (see Table 2), after the backward deletion procedure, showed that higher levels of impulsivity ($\beta = .26$), greater perceived peer pressure ($\beta = -.21$), and lower educational track ($\beta = -.16$) were significant predictors of the increase in smoking from age 12 to 17.⁴ Thus, unlike in the univariate model, sensation-seeking and susceptibility to peer influence were no longer significant in the multivariate model. This final combined model had a good fit to the data, $\chi^2(506) = 619.87$; $p < .01$; TLI = .96 and RMSEA = .04.

Discussion

The present accelerated longitudinal study was designed to investigate the developmental pattern of smoking behavior from ages 12 to 17 in 574 ethnically diverse Dutch adolescents. We used a metatheory, the TTI, to investigate whether relevant risk factors at age 12 that were derived from intrapersonal (cognitive and motivational) and social domains (peer influence) would explain the hypothesized variance in the increase in smoking development throughout adolescence. Our results showed that the intercept (baseline of smoking) was not significant, and no variance was detected, indicating that nearly all participants (i.e., 93.6%) were nonsmokers at age 12 whereas 29.40% of adolescents smoked at age 17. Although slightly higher, these prevalence statistics are quite comparable to population statistics in the Netherlands (Statistics-Netherlands, 2015). Furthermore, we found significant variance in the slope showing that adolescents differ in the rate of increase in their smoking development. As for the TTI-based risk-factors, higher levels of self-reported impulsivity and perceived peer pressure at age 12 predicted faster increases in adolescent smoking behavior development when investigated alone and simultaneously in the same model. Interestingly, whereas sensation seeking (intrapersonal domain) was significant in a univariate model, its contribution became nonsignificant in the mul-

tivariate model wherein other intrapersonal and social predictors were simultaneously estimated. Finally, as for putative covariates, lower educational track at age 12 predicted faster increases in smoking behavior from age 12 to 17. Below we further discuss these findings and their implications in relation to the TTI and in light of findings from previous studies.

Intrapersonal Factors

We found that self-reported impulsivity was a significant predictor of the increase in adolescent smoking. Although this is in line with the TTI and with cross-sectional studies that show that impulsivity and adolescent smoking are related, the small number of longitudinal studies that have investigated this link showed mixed findings (e.g., Audrain-McGovern et al., 2005; Elkins et al., 2006; Malmberg et al., 2013; Quinn & Harden, 2013). However, the present study was distinct in that it measured impulsivity during early adolescence and treated it as a prospective longitudinal predictor of the growth in smoking throughout adolescence. Although replications are needed, the current study provides evidence showing that impulsivity during early adolescence serves as a robust predictor of increases in smoking behavior up until the age of 17.

We did not find a significant link between our behavioral measure of inhibitory control (i.e., cued go/no-go task) and smoking development. These opposing findings support the notion that has been put forward that although impulsivity and inhibitory control are both indices of cognitive control, they tap into subtle different abilities (Dalley et al., 2011). This assertion also has implications for the TTI, which suggests that cognitive-related factors (ultimate level) predict adolescent health risk behaviors, because our results suggest that some cognitive-related behaviors might be more relevant than others for predicting adolescent smoking progression. As far as we know, there are currently no studies that have investigated whether an experimental measure of inhibitory control prospectively predicts smoking development in adolescents, so our results are not directly comparable to the existing literature, and thus await to be replicated. At least for late adolescents and emerging adults, inhibitory control measured via behavioral tasks also did not predict smoking (Dinn et al., 2004; Galvan et al., 2011).

A possible explanation for our null finding is that cognitive control might only be an issue for adults who suffer from nicotine dependence or addiction as was the case in a meta-analysis that showed that inhibitory control measured via the go/no-go task was significantly lower in adult smokers versus nonsmokers (i.e., Smith et al., 2014). These results might be similar for adolescent samples with nicotine dependence/addiction versus nonsmoking adolescents, however this is just a speculation as we did not assess nicotine dependence/addiction in the current study. Perhaps in samples with persons with nicotine dependence/addiction, there might be more variability in inhibitory control, depending on the severity of nicotine dependence/addiction. Relatively little variability in our sample might also be due to the simplicity of the task (i.e., most participants made only a few errors), however we did not encounter any modeling issues concerning low variability. Moreover, the performance (i.e., the mean) on the inhibitory

Table 2

Final Model: Unique and Robust Predictors of Adolescent Smoking Development

Predictor	<i>B</i>	<i>SE</i>	β	<i>p</i>
Impulsivity	.12	.03	.26	<.01
Perceived peer pressure	-.06	.02	-.21	<.01
Educational track	-.13	.04	-.16	<.01

Note. The *p* value for reward-seeking was .051. *B* = .16; *SE* = .08; β = .15.

⁴ Reward-seeking had a *p*-value of .051 in the combined model.

control task used in the current study was very similar to a study that employed the same task (labeled as the “Cued reaction time task” in that study) in a sample of adolescents who were 11 years on average (see [Derefinko et al., 2008](#)). Considered together, measurement error is not likely to be the cause of the current null finding. Instead, perhaps our sample size was not large enough to detect very small effects, however our sample size was larger than the sample size in similar studies with adult samples that did find that performance on the go/no-go task predict adult smoking behavior. Considered together, it also seems unlikely that a lack of power is the cause of our null finding.

As for the predictions of the TTI, although the TTI suggests that cognitive processes in general are risk factors for adolescent substance use, the current study suggests that inhibitory control is not relevant for adolescent smoking development, although it might be relevant for predicting the use of other substances in adolescents. Equally possible is that perhaps inhibitory control is more predictive of the onset of smoking, but not for the development of adolescent smoking, which we cannot conclude for sure with our data as there was negligible variability at baseline (age 12). Nevertheless, inhibitory control and smoking were significantly correlated at age 12 (see [Table 1](#)).

The current results underscore that when assessing cognitive control, diverse methods should be employed, as cognitive control is a heterogeneous construct, that involves wide ranging cognitive abilities that might not be strongly correlated with each other⁵ ([Dalley et al., 2011](#)), and that might have different effects on smoking as evident from the current study. Finally, our results also suggest that future studies on adolescent smoking development should focus more on the impulsivity (lack of forethought) aspect of cognitive control rather than the inhibitory aspect of cognitive control.

Motivational Factors

None of our motivational predictors proved to be unique and robust predictors of smoking progression in our combined model. This current longitudinal finding contradicts past cross-sectional studies that showed that particularly sensation-seeking in adolescents (e.g., [Leeman et al., 2014](#); [Martin et al., 2002](#); [Pokhrel et al., 2010](#)), and reward seeking in mid- (e.g., [Knyazev et al., 2004](#)) and late- adolescents ([Richardson et al., 2014](#)) are concurrently related to adolescent smoking. On the one hand, we do note that if a statistical significance level of $< .05$ is used, then in a larger sample reward seeking would have perhaps reached statistical significance as it had a p value of $.051$ in our sample. On the other hand, considering that the p values for the significant predictors in the combined model were all $p < .01$, it is clear that the other predictors are more likely to be relevant than reward seeking in the prediction of smoking development in the combined model. Also, taking into account that we ran multiple models, it is then more appropriate to use a p value of $.01$ as the criterion for statistical significance. Thus all things considered we conclude that motivational predictors such as reward seeking and sensation seeking are less relevant for adolescent smoking development compared to other cognitive-intrapersonal and social predictors.

Also interesting to note is that in our univariate model, sensation-seeking was a significant predictor of increases in smoking. However, this link did not hold up in our stringent multivariate model that also

accounted for other motivational, cognitive and social predictors. Importantly, however, the current finding does concur with other longitudinal studies that have investigated sensation seeking simultaneously with peer factors (see [Chun, 2015](#); [Otten et al., 2011](#)). In addition, it is worth considering that at least one study that also accounted for peer factors showed that although scoring high on thrill seeking (component of sensation seeking) did not contribute a significant probability to the overall probability that an adolescent would transition from monthly to daily smoking, scoring high on thrill seeking did predict smoking onset and the transition to monthly smoking ([Bricker et al., 2009](#)). This finding, in combination with the current finding could imply that some risk factors outlined by the TTI might be more relevant for smoking onset, or the beginning phases of smoking, but not for smoking escalation (or smoking dependence/addiction).

Finally, the finding that social factors like peer pressure were found to be more predictive of adolescent smoking compared to motivational factors like sensation-seeking is in line with the TTI, because the TTI describes sensation seeking as a “ultimate” level influence, whereas peer pressure is described as a “proximal” level influence ([Flay et al., 2009](#); [Snyder & Flay, 2012](#)). Below we further describe the importance of social factors in adolescent smoking development.

Social Factors

Finally, consistent with the TTI, we investigated peer influences on adolescent smoking. Our results show that the effect of perceived peer pressure is above and beyond the significant links we found for the other risk factors on the development of adolescent smoking. Considered together, perceived peer pressure is not only a concurrent predictor of adolescent smoking (e.g., [Crockett et al., 2006](#); [Santor et al., 2000](#)), but it is also a unique longitudinal predictor of smoking progression throughout adolescence, which mirrors the latent growth findings of [Duncan et al. \(1995\)](#).

Next, although susceptibility to peer influence was a significant predictor when tested individually, it became nonsignificant when tested in a multivariate model, whereas perceived peer pressure remained significant. Thus, susceptibility to peer influence is perhaps already an underlying component of perceived peer pressure,⁶ making it redundant to account for both of these peer pressure forms in the same model. This interpretation is in line with TTI, as although both susceptibility to peer influence and peer pressure are identified by TTI as risk-factors of the social domain, the former is described as a “distal/predisposing” influence, whereas the former is described as a “proximal/direct” influence of adolescent smoking. That is, although all levels of influence (ultimate, distal, proximal) influence behavior, the influence of proximal risk factors are more direct ([Snyder & Flay, 2012](#)). Other studies that have investigated susceptibility to peer influence (i.e., [Chun, 2015](#); [Otten et al., 2011](#)) did not simultaneously consider the more direct/proximal perceived peer pressure. Thus, our finding that, after accounting for perceived peer pressure, susceptibility to peer influence becomes less relevant for adolescent smoking develop-

⁵ In the current study, inhibitory control and the impulsivity items were not significantly correlated at age 12 ([Table 1](#)).

⁶ The correlation between the factors for perceived peer pressure and susceptibility to peer influence was $r = .24$.

ment suggests that it would be worthwhile for future studies to include both measures to further investigate why and when this suppression might occur.

In sum, a combination of intrapersonal (particularly cognitive) and social risk-factors robustly and uniquely predicted the variance in increases in adolescent smoking. Specifically, higher levels of impulsivity (cognitive-intrapersonal) and perceived peer pressure (social) at age 12 predicted faster increases in smoking behavior from ages 12 to 17 above and beyond their individual effects. Moreover, more motivational risk factors such as sensation-seeking appeared to be no longer significant for increases in smoking behavior when cognitive and social factors were taken into account. In other words, the current results show that when investigating effects of sensation-seeking on adolescent smoking, significant cognitive and social factors like impulsivity and perceived peer pressure should be accounted for as they might override the predictive power of sensation-seeking when tested alone. This is one of the primary reasons the TTI was developed, namely to acknowledge that risk-factor tend to be interrelated (Flay et al., 1995). More specifically, for the current study, this suppression of motivational factors as unique and robust predictors might be because cognitive and social factors already contain a motivational component. Perhaps motivational (affective) factors such as sensation-seeking can be seen more as underlying components of the cognitive domain (Duncan & Barrett, 2007), as well as underlying components of the social (peer) domain (Pfeifer & Blakemore, 2012). Along these lines, when cognitive and social risk factors are taken into account, an additional pure motivational component becomes redundant, and as our results suggest, this might be particularly true for increases in smoking throughout adolescence.

Finally, the effect sizes were moderate for (cognitive-intrapersonal) impulsivity and perceived (social) peer pressure whereas the effect size for educational track was small. This implies that the significant cognitive and social risk factors are of equal importance for the increases in smoking throughout adolescence, but that educational track is a relatively weaker predictor. Nevertheless, the mechanism by which educational track is linked to adolescent smoking increases warrants further investigation.

Strengths, Limitations and Future Directions

The current accelerated longitudinal study has provided new insights into the combined roles of interrelated but unique risk factors of adolescent smoking development. Furthermore, capitalizing on a stringent latent growth design via structural equation modeling, we were able to ascertain if variance exists in the baseline as well as the progression of smoking throughout adolescence. Finally, we (a) used self-report as well as behavioral measures, (b) investigated intrapersonal as well as social predictors, (c) accounted for potential covariates such as gender and educational track, and (d) used latent factors, which accounts for measurement error. However, despite overcoming several methodological challenges of past studies, there are also limitations inherent in the current study that should be noted.

Readers should consider that during the data-collection for this Dutch sample of adolescents, on January 1, 2014, a law was implemented in the Netherlands that prohibits the sale of tobacco to individuals who are younger than 18 years old. In prior years,

youth were allowed to purchase tobacco from the age of 16. As we mentioned in the introduction, such environmental and legal-related factors also influence the use of substances in adolescents. In our sample, 72.9% of 16-year-olds reported they were non-smokers before the law changed, whereas 82.4% of the 16-year-olds reported they were nonsmokers after the law changed. Thus there was a decline in 16-year-olds who smoked after the new law was implemented. Whether this change exerted significant influence on our findings cannot be known. Nevertheless, the fact that significant growth was observed in the model in spite of these societal shifts speaks to the robustness of the predictive model of smoking observed in the present study.

In addition, bias checks showed that persons who completely dropped out of the study after Wave 1 had higher smoking levels than persons who did not drop out altogether. However, persons who dropped out of the study after Wave 1 was only 15% of the sample, and similar to what we mentioned above, our model was still robust enough to show significant increases in smoking despite the relatively high smoking levels of the participants who dropped out. Nonetheless, the results might not be generalizable to early heavy smokers since these students were more likely to drop out of the study. Future investigations would have to start at younger ages in order to examine whether the same factors predict smoking onset and development for these adolescents.

A methodological limitation is that we used only one item to measure smoking behavior, from which we can only conclude that adolescents smoke more often as they get older, but not that they increase in the amount they smoke. In the future, researchers could consider using more items, in particular quantity items (e.g., "how many cigarettes do you smoke per week?"), because they could provide more information about the escalation of smoking, or even about nicotine addiction during adolescence.

Next, adolescents in the lower educational track showed faster increases in smoking. This raises the question as to whether there are other intrapersonal or social factors that are linked to educational track that might be the source of this link. This is an interesting and important avenue to explore in future research on adolescent smoking development. It is also important to point out that although educational track is a significant predictor of adolescent smoking progression, importantly our findings further imply that the significant cognitive-intrapersonal and social predictors are above and beyond any confounding effects of educational track.

Finally, it is also important to consider that the effect sizes were small to medium ($\beta = .16-.26$) in magnitude, but that predictors with even small effect sizes can be meaningful, particularly when dealing with health-related issues.

Conclusion

The current study suggests that cognitive-intrapersonal and social factors like impulsivity and perceived peer pressure are both of equal importance, as they uniquely contribute to adolescent smoking development when investigated simultaneously. Moreover, accounting for such cognitive-intrapersonal and social factors suppress the contribution of motivational-intrapersonal factors like sensation-seeking in predicting increases in smoking throughout adolescence. These findings underscore why it is essential to investigate the contribution of interrelated risk factors simultane-

ously, a strong assertion of the TTI. Had we not done so, the seemingly importance of sensation-seeking when tested alone might have led to inaccurate conclusions about its predictive power. Finally, these findings could have practical implications for the contents of prevention programs on adolescent smoking development. Most noteworthy is that the current study additionally pinpoints which confluence of risk factors are relevant for early prevention programs, and that tackling this confluence of factors, from as young as age 12, might halt the deadly increase in smoking behavior throughout adolescence.

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Call for Nominations

The Publications and Communications (P&C) Board of the American Psychological Association has opened nominations for the editorships of *Clinician's Research Digest: Adult Populations* and *Child and Adolescent Populations*; *Journal of Experimental Psychology: Learning, Memory, and Cognition*; *Professional Psychology: Research and Practice*; *Psychology and Aging*; and *Psychology, Public Policy, and Law* for the years 2019 to 2024. Thomas Joiner, PhD; Robert L. Greene, PhD; Ronald T. Brown, PhD; Ulrich Mayr, PhD; and Michael E. Lamb, PhD, respectively, are the incumbent editors.

Candidates should be members of APA and should be available to start receiving manuscripts in early 2018 to prepare for issues published in 2019. Please note that the P&C Board encourages participation by members of underrepresented groups in the publication process and would particularly welcome such nominees. Self-nominations are also encouraged.

Search chairs have been appointed as follows:

- *Clinician's Research Digest: Adult Populations* and *Child and Adolescent Populations*, Chair: Pamela Reid, PhD
- *Journal of Experimental Psychology: Learning, Memory, and Cognition*, Chair: Stephen Rao, PhD
- *Professional Psychology: Research and Practice*, Chair: Kate Hays, PhD
- *Psychology and Aging*, Chair: Pamela Reid, PhD
- *Psychology, Public Policy, and Law*, Chair: David Dunning, PhD

Candidates should be nominated by accessing APA's EditorQuest site on the Web. Using your browser, go to <http://editorquest.apa.org>. On the Home menu on the left, find "Guests/Supporters." Next, click on the link "Submit a Nomination," enter your nominee's information, and click "Submit."

Prepared statements of one page or less in support of a nominee can also be submitted by e-mail to Sarah Wiederkehr, P&C Board Editor Search Liaison, at swiederkehr@apa.org.

Deadline for accepting nominations is Monday, January 9, 2017, after which phase one vetting will begin.