The Pennsylvania State University

The Graduate School

College of Health and Human Development

MODELING THE SPILLOVER OF FAMILY-BASED PREVENTION: STRENGTHENING CAUSAL INFERENCE OF SOCIAL NETWORK EFFECTS ON ADOLESCENT SMOKING

A Thesis in

Human Development and Family Studies

by

Lawrie Green

Submitted in Partial Fulfillment
of the Requirements
for the Degree of
Master of Science

December 2018

The thesis of Lawrie Green was reviewed and approved* by the following:

Max Crowley
Assistant Professor of Human Development and Family Studies
Thesis Advisor

Greg Fosco
Associate Professor of Human Development and Family Studies
Associate Director of the Edna Bennett Pierce Prevention Research Center

Douglas Teti
Professor of Human Development and Family Studies
Head of Department of Human Development and Family Studies

*Signatures are on file in the Graduate School

ABSTRACT

Despite a substantial evidence-base around preventing tobacco use in adolescence, initiation of smoking among minors remains a serious public health issue. While it is known that parents and the family play an important role in preventing smoking during adolescence, dissemination of family-based substance abuse prevention remains a major barrier. The current study addresses the question of whether the effects of a family-based prevention program can spread beyond the people directly receiving the program and if those effects are sustained across time. By using data from the PROSPER project, in which an evidence-based program was delivered with fidelity under real world conditions, we tested the diffusion of effects through adolescent social networks. We found that having friends at the beginning of middle school who attended a family-based program was associated with lower odds of ever having smoked by the end of high school (OR = .90). Each additional friend represented a 2.45% decrease in the absolute likelihood of smoking by the end of high school.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGEMENTS

The research reported here was supported, in whole or in part, by the Institute of Education Sciences, U.S. Department of Education, through grant R305B090007 to The Pennsylvania State University. It uses data from PROSPER, a project directed by R. L. Spoth and funded by the National Institute on Drug Abuse (RO1-DA013709) and the National Institute on Alcohol Abuse and Alcoholism (AA14702). The opinions expressed are those of the authors and do not represent the views of the granting agencies.

Chapter 1

Introduction

In the United States, nearly 9% of all annual healthcare costs (amounting to $165 billion in 2010) are attributed to smoking related diseases (Xu, Bishop, Kennedy, Simpson, & Pechacek, 2015) and smoking is estimated to result in a 10-year reduction in life expectancy (Jha et al., 2013; Schmitz, Kruse, & Kugler, 2003). Almost 90% of tobacco use begins before age 18 (US Department of Health and Human Services, 2014). As a result preventing adolescent tobacco use before it begins is an important public health issue due to the immense cost to both to individuals and society. Use of tobacco products has detrimental effects on almost every major system of the body with smoking generally impairing lung function, leading to an increased risk of chronic obstructive pulmonary disease and respiratory infections (Fletcher & Peto, 1977; USDHHS, 2014). Secondhand smoke exposure causes more than 41,000 deaths each year in the United States out of the 480,000 deaths that can be attributed to smoking-related causes (USDHHS, 2014).

The Development of Smokers

The developmental process of tobacco use begins in adolescence for the large majority of smokers. Most (almost 90%) adult smokers begin smoking by age 18 (Johnston, O'Malley, Miech, Bachman, & Schulenberg, 2016). The timing of this process is especially concerning because of the changes that nicotine addiction can make to the adolescent brain as it develops (England et al., 2017). Forming a dependence on a highly addictive substance, such as nicotine,

in adolescence can prime the brain to be more prone to addiction later in life (England et al., 2017). While nicotine addiction in adults usually results from heavy daily use over an extended period of time, adolescents can show signs of dependence within weeks of occasional use (DiFranza et al., 2000).

Peer influence is a significant predictor of the initiation and continuation of smoking in adolescence, above and beyond individual and environmental risk factors, such as impulsivity and poor academic achievement (Defoe, Dubas, Somerville, Lugtig, & van Aken, 2016; Forrester, Biglan, Severson, & Smolkowski, 2007). In early adolescence, the perception of smoking among one's peers is a stronger risk factor for initiating smoking than the actual level of smoking in school (Ellickson, Bird, Orlando, Klein, & McCaffrey, 2003). Smoking is often a social activity in adolescence, with adolescents often gaining access to cigarettes through their friends and smoking together in groups (Kirke, 2004). A recent meta-analysis estimated that having at least one friend who smoked resulted in an adolescent having almost twice the odds of both initiating smoking and continued use (Liu, Zhao, Chen, Falk, & Albarracín, 2017). Conversely, smoking cessation also spreads through peer social networks. One study of smoking in large social networks showed that having a friend stop smoking decreases an individual's likelihood of smoking by 36% (Christakis & Fowler, 2008).

The Role of Peer Social Networks in Smoking

When examining the role of peer social networks in adolescent smoking, it is important to disentangle the effects of peer selection (smokers becoming friends with other smokers, nonsmokers becoming friends with nonsmokers) from peer influence (Kirke, 2004). The process of peer selection includes both an individual selecting for friends who are similar and rejecting

peers who are dissimilar (Simons-Morton & Farhat, 2010). Peer networks in adolescence tend to be homogenous in terms of tobacco use, and this selection effect occurs as early as sixth grade (Hall & Valente, 2007). While research is mixed on whether peer selection or influence has stronger effects on adolescent smoking, it is clear that both are important factors (Simons-Morton & Farhat, 2010). Programs to prevent tobacco use in adolescents must take into account peer selection and influence in order to be effective.

Parents can influence adolescent smoking directly, such as by modeling the habit by smoking themselves, or indirectly by encouraging friendships with nonsmoking peers (Simons-Morton & Farhat, 2010). Effective parental monitoring is an important protective factor against adolescent substance use in general, and smoking in particular (Lakon et al., 2015; Steinberg, Fletcher, & Darling, 1994). Prevention programs that involve parents in early adolescence can take advantage of the influence parents have with their children.

Diffusion in Adolescent Social Networks

One way to understand the spread of behavior through social networks is to consider models from the study of infectious disease (Hill, Rand, Nowak, & Christakis, 2010). In these models, diseases are spread through contact with another person with a given probability. Hill et al. (2010) used this model to study the spread of obesity through social networks and found that the rate of obesity increased by 0.5% per year for each obese social contact. The importance of adolescent peer networks in initiating and maintaining tobacco use suggests that we can extend the infectious disease model to behaviors like smoking (Blok, Van Empelen, Van Lenthe, Richardus, & De Vlas, 2013).

If the spread of smoking through adolescent behaviors can be thought of as being similar to an infectious disease, then prevention programs can work like a vaccine in preventing that spread (Spoth, Guyll, & Shin, 2009). Using techniques from social network analysis, prevention researchers have recently begun to conceptualize how to study the diffusion of effects of prevention programs from participants to nonparticipants (Rulison, Gest, & Osgood, 2015). This approach suggests that prevention programs do not have to reach everyone in a community in order to effect change at a population level, which is an especially important consideration for family-based programs that can have low recruitment rates (Heinrichs, Bertram, Kuschel, & Hahlweg, 2005).

Much research has been done on how to create effective prevention programs targeting tobacco use in adolescence. A Cochrane review of family-based programs found evidence that high quality prevention programs, including the Strengthening Families Program 10-14 (SFP 10-14), can reduce adolescent tobacco use, especially in conjunction with programs offered in schools (Thomas, Baker, Thomas, & Lorenzetti, 2015). SFP 10-14 is a brief, universal prevention program offered to small groups of families that targets protective and risk factors at both the level of the family and the adolescent (Molgaard & Spoth, 2001). For the parents, SFP 10-14 focuses on the relationship between parent and child and improving parental monitoring of adolescent peers and behavior, both significant factors in preventing substance use (Fosco, Stormshak, Dishion, & Winter, 2012; Liddle, 2004). The adolescents in SFP 10-14 receive training on peer refusal skills and improving self-regulation. These protective factors at the family and individual level work together to change how adolescents approach their peer relationships, which suggests that the skills and knowledge learned in these programs can spread through social contact outside the prevention setting.

However, there has been very little research on the potential diffusion of prevention programs beyond their direct recipients. Part of the difficulty in studying these effects is the lack of access researchers have to the social networks of the people targeted in their studies. One study by Rulison and colleagues (2015) tested the diffusion of effects of SFP 10-14 from adolescents who participated in the program to their friends. They found significantly lower odds of having smoked cigarettes in the past month at the beginning of high school among adolescents who had SFP-attending friends compared to those with no SFP-attending friends. This work was a key first step in testing whether diffusion to nonparticipants can happen.

The current study expands on these findings by seeking to address potential selection effects within peer selection through the implementation of propensity models within a Potential Outcomes Framework. While propensity models have not often been used in the context of studying the effects of prevention programs, they have been frequently used in studies of social networks to separate out the effects of diffusion (or social contagion) from peer selection (Aral, Muchnik, & Sundararajan, 2009). For example, one study used a propensity model to estimate the causal effects of a hurricane on social structures in a community – a case in which random assignment was impossible (Phan & Airoldi, 2015). This approach has yet to be applied to understanding the potential spillover of family-based interventions on youth smoking. This method can be used in the context of family-based prevention programs to strengthen causal inference of the impact of exposure to treated families. Further, this work focuses on the key sensitive developmental period surrounding smoking—following youth through the end of high school—where risk of becoming a smoker drops precipitously.

Current Study

The current study aims to assess whether the effects of a family-based prevention program can spread beyond the people directly receiving the program and if those effects are sustained across time—in this case through the sensitive developmental window where most smokers initiate. By using data from the PROSPER project, a longitudinal study in which an evidence-based family intervention was delivered with fidelity under real world conditions, we have the opportunity to test the diffusion of effects through adolescent social networks. Because participants and nonparticipants in SFP 10-14 lived in the same communities and attended the same schools, it is possible that knowledge and skills gained by participants could influence those who did not participate. A conceptual model of what the diffusion of SFP 10-14 might look like is shown in Figure 1. In order to test this model of diffusion, we focused on the outcome of initiation of cigarette use for several reasons. Smoking is legal at age 18 in the areas targeted by the study, unlike alcohol and other drugs, so access to cigarettes is more easily obtained. Cigarettes contain nicotine, which is highly addictive and can cause changes in the developing adolescent brain that make later illicit drug use more likely (Dierker, Braymiller, Rose, Goodwin, & Selya, 2018). Finally, smoking is a behavior particularly susceptible to social network influences (Simons-Morton & Farhat, 2010), which makes it a good target for studying the diffusion effects of a prevention program.

A number of factors, such as socioeconomic status and logistical issues, can influence whether family recruitment and retention in prevention programs (Heinrichs et al., 2005). Some of these factors are also associated with adolescent tobacco use and can therefore confound any potential outcome effects by the program. In addition, adolescent peer selection is a complex process, and peer group membership cannot be randomized. We used a Potential Outcomes

Framework and employed a propensity modeling approach to account for these potential confounding effects on tobacco usage at the end of high school (Austin, 2011). Propensity models have been used extensively to deal with issues of confounding and selection in observational data where randomization was not possible (Rosenbaum & Rubin, 1983; Varvil-Weld, Crowley, Turrisi, Greenberg, & Mallett, 2014). In this study, we used a propensity model to estimate the effects of an adolescent having friends who received SFP 10-14 on their later tobacco use, taking into account the various cofounders that might affect the choice of peer group.

Chapter 2

Methods

Sample

The participants in this study (N = 5,083, 50% male, 85% White) were middle school students in the 14 treatment communities of the PROSPER (Promoting School-community-university Partnerships to Enhance Resilience) project, a community-randomized trial of an intervention delivery system in two rural Midwestern states (Spoth, Greenberg, Bierman, & Redmond, 2004). There were two cohorts in the study, and they began 6th grade in 2001 and 2002. In order to be considered for this study, communities had to have a school district with between 1300 to 5200 students and at least 15% of students eligible for free or reduced lunch. We included all students in the sample, both participants and nonparticipants in SFP 10-14.

Procedure

Communities in the intervention condition implemented universal substance use prevention programs in their middle schools and offered a family-based program in addition. While the communities were given a choice of family programs to implement, they all opted for SFP 10-14. The family program was offered to students in the spring of sixth grade, and the school program was given in the fall of seventh grade. All students in the intervention communities received the in school universal program. As reported in previous work, 17% of eligible families (N = 803) attended at least one session of the family-based program (Spoth, Clair, Greenberg, Redmond, & Shin, 2007). Students completed a survey at baseline in the fall of 6th grade, before the family program was offered. Follow-up surveys were completed post-

intervention in the spring of 6th grade and the spring of every year following until the end of high school.

Measures

Friends Receiving SFP 10-14. As part of data collection, students nominated up to seven friends in a survey of peer relationships (Rulison, Gest, & Osgood, 2015). These names were compared against the school rosters in each community to find other study participants, and their study IDs were recorded. Names of students that could not be matched or of students who had previously been at the school but were no longer on the rosters were flagged. Each nominated friend from Wave 1 (pre-test) was compared against the list of students who attended at least one session of SFP 10-14 and was marked with their attendance status (0 = no, 1 = yes). The total number of friends was calculated by counting the number of friends who received SFP 10-14 for each student.

Outcomes. Students were asked at each wave whether they had ever smoked cigarettes, and they answered yes (1) or no (0). For this analysis, we used the outcomes at Wave 8. The missing responses at Wave 8 were imputed using the Multivariate Imputation by Chained Equations package in R, which created 20 complete datasets for analysis (van Buren & Groothuis-Oudshoorn, 2011).

Covariates. A number of potential confounders were identified for inclusion in propensity models predicting the number of friends who had received SFP 10-14. The covariates were all measured through self-report at Wave 1. The variables included in the model were age, sex, ethnicity, whether they received free/reduced lunch, personality characteristics, and attitudes toward and expectations of substance use. Some family characteristics (also reported by the

adolescent) were also included in the model, such as parental marital status, family cohesion, and their perception of parenting skills. The full list of covariates is shown in Table 1.

Analytic Approach

We modeled the diffusion effects of a family-based prevention program on adolescent smoking using a four-step analytic approach. First, propensity scores were calculated for the number of friends each student had who attended SFP. Second, each student was weighted based on their propensity score to create groups that were equivalent across confounders. Third, the weights were tested to ensure balance. Finally, the weights were used to estimate the effect of peers receiving SFP on adolescent smoking.

Propensity model. Propensity models are tools to strengthen causal inference when analyzing observational data (Rosenbaum & Rubin, 1983). It is not possible to randomly assign adolescents to peer groups, so a continuous propensity model was used to estimate the probability that a student had a given number of friends who attended SFP, taking into account the values of the measured confounders. The propensity score (π_i) is calculated using the following equation to estimate the probability that an individual received a certain level of the treatment (T_i; in this case the number of friends who received SFP 10-14) given the measured covariates (X_i: Rosenbaum & Rubin, 1983).

$$\pi_i = P(T_i = 1 \mid X_i)$$

Inverse probability weights. Inverse probability weights (IPWs) were calculated from the propensity score to model the inverse probability that a student has a given level of exposure to the treatment (i.e. number of friends who attended SFP) given the potential confounders (Coffman & Zhong, 2012). IPWs are used similarly to survey weights to adjust the sample so

that the distribution of the measured confounders in the sample does not depend on the treatment group (Austin, 2011).

Balance. The IPWs can become unstable or unbalanced for participants who have a low propensity score, so the weights were tested for balance using weighted and unweighted Pearson correlations between the confounders and the exposure variable. The magnitudes of the weighted correlations should be less than 0.2 to be balanced.

Outcome model. We tested the impact of having a number of friends who received SFP at the beginning of middle school on smoking at the end of high school using weighted (with the IPWs) and unweighted logistic regression models with the GLM package in R.

Chapter 3

Results

Descriptive Statistics

The mean number of friends who received SFP 10-14 was 0.66 (*SD* = 0.92, range 0-6). Almost half (44%) of the sample had at least one friend who received SFP 10-14. Table 2 shows the frequency distribution for number of friends who received SFP 10-14. More than half (63%) had ever smoked a cigarette by the end of high school. Among adolescents who had no friends in Wave 1 (sixth grade) who received SFP 10-14, 69% of them had ever smoked a cigarette by the end of Wave 8 (end of high school). The proportion of smokers generally decreased to a low of 40% having tried smoking among those who had six friends who received SFP 10-14. Table 1 provides descriptive statistics on the potential confounders used in the propensity model.

Balance Diagnostics

Twenty-four potential confounders were included in the propensity model to create the inverse probability weights described above. Balance was achieved on all covariates included in the propensity model (used previously in Crowley, Coffman, et al., 2014 and Crowley, Jones, et al., 2014). Specifically, application of inverse probability weighting resulted in the correlation between all potential confounders and the number of friends who received SFP being under .2 as demonstrated in previously published models of family-based programming (Varvil-Weld et al., 2014).

Outcome Analysis

The number of friends an individual has who received a family based prevention program in early middle school was significantly and negatively associated with the odds of an adolescent ever having smoked by the end of high school (OR = .90, 95% CI: 0.84, 0.97). Figure 1 shows the estimated percentage of adolescents who have ever smoked by the end of high school for each potential number of friends who received SFP 10-14. Each additional friend who received SFP represented a 2.45% decrease in the absolute likelihood of smoking by the end of high school.

Post-hoc Analysis

The sample used in our main outcome analysis contained both participants and nonparticipants in SFP 10-14. It is possible that the diffusion effect we found was driven by those who received SFP themselves – perhaps having friends who also received the program multiplied the effect of the program. To investigate this further, we ran the same outcome model on a sample restricted to nonparticipants only (N = 4,280, 50% male, 84% White). In this model, having friends who received SFP 10-14 was still significantly associated with lower odds of ever smoking (OR = 0.89, 95% CI: 0.83, 0.96).

Chapter 4

Discussion

This work explores the potential spillover benefit of family-based prevention through peer networks. We used a propensity model to examine the diffusion effects of a family prevention program on adolescent lifetime smoking through adolescent peer networks. Based on the previous work in this area by Rulison et al. (2015) looking at smoking at the beginning of high school outside a Potential Outcomes Framework, we expected that having more friends exposed to SFP 10-14 would make it more unlikely that an adolescent ever smoked. Consistent with our hypothesis, the number of friends an adolescent had at the beginning of Grade 6 who received a family-based substance use prevention program was significantly related to lower rates of tobacco use initiation at the end of Grade 12.

Using a propensity model allowed us to better account for peer selection effects that might have biased our outcome model. We included a substantial number of covariates in the propensity model that might have confounded the effects between students who were more likely to have friends who attended a family-based prevention program and students who were less likely to smoke cigarettes. The impact that the diffusion of SFP 10-14 had on reducing the odds of smoking was robust, even when including these factors in the model. Somewhat surprisingly, the diffusion effects were just as strong whether the sample included all adolescents (including those who received SFP 10-14 themselves and those who did not) or was limited to nonparticipants only, as demonstrated in the post-hoc analysis.

The sample in the post-hoc analysis mirrors that in the previous work by Rulison et al. (2015), who only included nonparticipants. As a follow up to that study, we were able to show that the diffusion effects lasted until the end of high school and were robust in the context of a

propensity model to account for peer selection effects. The similarity in findings between the combined sample and the nonparticipant sample suggests that the social network effect seems to be driving the overall population effect of SFP 10-14 on smoking. One possible reason for this is that the lessons taught in SFP 10-14 changed how peers interacted with each other in schools and made the peer context less conducive to trying cigarettes. It is important to note that these effects were found in the context of all youth in the school also receiving a school-based substance abuse prevention program—underscoring the additional value of providing family-based programming in schools.

This study highlights the effectiveness of family prevention programs in changing substance use patterns through social networks. While only 17% of students in this sample attended SFP 10-14 (comparable to recruitment rates for family programs in other studies), the indirect reach through peer networks was much broader. Because SFP 10-14 was offered through middle schools in the PROSPER communities, the families who attended were part of the social networks of the nonparticipating families through their children. Nearly half of the sample had at least one friend in sixth grade who attended SFP 10-14. These findings, strengthened by the application of a potential outcomes framework, indicate that penetration of family programs is likely greater than simply those who directly received the program.

This work is the first to show that these spillover effects persist through the end of the most sensitive developmental period for smoking. This is essential information for informing public health efforts that seek to develop comprehensive initiatives to reduce and ultimately end smoking among adolescents. Further, through the adoption of the Potential Outcomes Framework, this work offers the first demonstration of the use of propensity models to strengthen our ability to draw causal inferences of family-based substance abuse prevention

programs. In particular, this work offers an important analytic strategy for the field and the opportunity to grow our understanding of the unique role family-based interventions can play in community prevention efforts.

Previous work by Rulison et al. (2015) in this area found that unstructured time with friends was a key mediator of diffusion. In other words, parents who attended the family program limited the amount of unsupervised time their children spent with their friends, leading to less substance use. Adolescents who did not attend the program but were friends with those who did likely experienced less unstructured socializing time as well and thus received the benefit of being in a more protective peer context. Family-based prevention programs can help change the attitudes and norms around substance use for both those who participate and their friends, even when a relatively small proportion of families in a community receive them directly.

These findings also suggest that family-based prevention programs are a promising option for schools to offer to their students and families. Even though all students in this sample received a school-based substance use prevention program in middle school, the rate of lifetime smoking at the end of high school was much higher than the national average (63% in this sample vs 46% nationwide in 2013; Arrazola et al., 2014). However, SFP 10-14, delivered in conjunction with the school program, resulted in significantly lower rates of cigarette use for those who had more exposure to it through their friends. However, implementation of these programs should be done thoughtfully, taking into consideration the ways in which peer networks change throughout adolescence. Due to the size of the school districts in the communities in this study, the students likely had continued contact with their peers who received SFP 10-14 throughout middle school and high school. If instead the students had

dispersed into different high schools, it is possible that the effects of SFP 10-14 on peer networks would have weakened due to the lack of reinforcement. In order to leverage the diffusion effects of family-based programs, these programs should be offered strategically based on existing social networks in the community.

Some caution is warranted in generalizing the diffusion effects found in this study of SFP 10-14 to family-based prevention programs as a whole. The factors that likely led to diffusion effects for SFP 10-14, such as the emphasis on parental monitoring and adolescent peer refusal skills, are not necessarily present in every family-based program (Van Ryzin, Roseth, Fosco, Lee, & Chen, 2016). In addition, not every program will be able to capitalize on these peer network effects due to how the program is structured. Some programs, like Parent-Child Interaction Therapy, can be effective at an individual level but are delivered in a one-on-one format that could make it difficult to offer to enough families in the same social networks to see diffusion. When selecting family-based prevention programs for implementation, care should be taken to choose the right programs given the context and needs.

The existence of diffusion effects to nonparticipants within a social network also has implications for how researchers should evaluate these programs in the context of randomized control trials. Researchers need to be mindful of the social networks that the families exist in outside the research study. If the control and treatment groups in a trial are drawn from the same community, it is highly likely that there will be contact between them outside the study, leading to potential contamination. While that might be good news from a prevention perspective, it could lead to reduced effect sizes in the study outcome.

An additional finding that should be noted from this study is the relatively high rate of tobacco use among the adolescents in this sample. While tobacco use initiation among

adolescents has been declining in the United States over the past few decades due to many significant public health efforts, this sample shows that there are still geographic pockets of the population with high rates of use. Preventing tobacco use is therefore still a pressing public health issue. Smoking has been linked to later drug use, which means these areas might be particularly at risk for substance use (Hanna, Yi, Dufour, & Whitmore, 2001). The use of electronic cigarettes, which still contain nicotine and involve inhaling an aerosol substance with chemicals of questionable safety, has also been rising quickly among adolescents and is a growing public health concern (Jamal et al., 2017).

Family-based prevention programs, like SFP 10-14, should be used as a key component in the public health fight against adolescent tobacco use. These programs, which have been shown to be effective in reducing traditional cigarette use, are also a potential resource in preventing electronic cigarette use. Much research has shown the influence of the family on adolescent development inside the family (Lippold & Jensen, 2017), but this study shows the potential for families to influence adolescents outside the family as well, suggesting an alternative way to reach at-risk youth.

Limitations & Future Directions. The adolescents in this sample were mostly white with married parents and lived in rural communities, so more research is necessary to understand whether these effects may differ in other populations. While we included a number of potential confounders in the model, building off previously published work, there could also be other, unmeasured, confounding variables that affected adolescent peer selection that should be explored. The school districts in this study were also fairly small. It is unclear how these effects would play out in larger districts that had more schools or a more transient population.

Future research should examine these patterns longitudinally to see how a family prevention program offered in sixth grade can affect the peer influence process over time. It is possible that those who did not receive SFP 10-14 (and are therefore more likely to use tobacco or other substances) become isolated socially from those who do. There may be critical transition points, such as the beginning of high school, when booster sessions should be offered for those who might need them. Examining how diffusion of SFP 10-14 affected the peer networks themselves can provide insight into how to target family-based prevention programs to best take advantage of these network effects.

The diffusion effects of SFP 10-14 were seen in the context of offering a family-based program in conjunction with a school-based program. It is unclear to what extent the school-based program reinforced what was taught in the family program, and if that background information being taught to all participants is necessary for the diffusion to occur. The results of this study also suggest that having more than one family in a social network receive a prevention program makes diffusion to nonparticipants more likely. However, many nonparticipants in this sample did not have any friends who received the program. There should be more work done to determine what the goal should be for the percentage of the population reached by a program to have maximum spillover benefit through social networks.

Conclusion

The current study found significant effects of diffusion effects from participants in a family-based prevention program to nonparticipants through their social networks. Using a propensity model with a wide variety of covariates to account for potential peer selection effects, we were able to show that having friends who received SFP 10-14 in sixth grade was linked to

lower odds of ever smoking by the end of high school. A significant percentage of the adolescents in this sample had at least one friend who received SFP 10-14, despite the low participation rate in the program itself, which suggests that the reach of family-based programs is broader than previously thought.

Future research can build on this work by examining the process of diffusion in other contexts, such as larger school districts with more diverse populations. There also should be more research done on how these programs affect the peer selection process longitudinally, which can help inform how these programs can be targeted more effectively. Program developers and implementers should consider the social network context and the potential for diffusion when creating and evaluating family-based programs.

Table 1. Descriptive statistics of potential confounders used in the propensity model.

Variable	Mean (SD)
Demographic information	
Age	11.85 (0.44)
Gender (0 = Female; 1 = Male)	0.5 (0.5)
Received free/reduced lunch (0 = No; 1 = Yes)	0.34 (0.47)
Youth functioning	
Stress management (1 = Never; 5 = Always)	2.89 (0.96)
Assertiveness (1 = Would not; 5 = Would)	4.17 (0.7)
Problem solving (1 = Never; 5 = Always)	3.75 (1.03)
Self-oriented activities (1 = Never; 5 = Always)	1.96 (0.94)
Risky activities (1 = Would not; 5 = Would)	2.8 (1.3)
School adjustment and bonding (1 = Never; 5 = Always)	3.93 (0.62)
Negative attitude toward school (1 = Disagree; 5 = Agree)	1.98 (1.0)
School absences (1 = None; 5 = 16+)	2.77 (0.98)
Youth cognitions about substance use	
Positive attitude toward substance use (1 = Not wrong; 5 = Wrong)	3.76 (0.57)
Substance use norms (1 = No one uses; 5 = All use)	1.83 (0.89)
Positive expectations for substance use (1 = Agree; 5 = Disagree)	4.71 (0.51)
Positive expectations for cigarette use (1 = Agree; 5 = Disagree)	4.65 (0.58)
Positive expectations for alcohol use (1 = Agree; 5 = Disagree)	4.72 (0.52)
Positive expectations for marijuana use (1 = Agree; 5 = Disagree)	4.75 (0.55)
Substance refusal intentions (1 = Not refuse; 5 = Refuse)	4.84 (0.49)
Substance refusal self-efficacy (1 = Not confident; 5 = Confident)	4.38 (1.22)
Family environment	
Parents' marital status (0 = Single parent; 1 = Dual parents)	0.77 (0.42)
Residence with biological parents (1 = Both biological parents)	0.62 (0.48)
Family climate (1 = Negative; 5 = Positive)	3.63 (0.72)
General child management (1 = Negative; 5 = Positive)	3.99 (0.59)

Note: N = 5083. All confounders were measured at Wave 1. Ethnicity was also included. Participants were 85% White, 5% Latino/Hispanic, 3% African-American, 2% Native American, 1% Asian, and 4% Other.

Table 2. Distribution of number of friends who received SFP 10-14.

Number of Friends	Frequency
0	2921
1	1419
2	518
3	172
4	36
5	9
6	8

Note. N = 5083.

Figure 1. Conceptual Model of Peer Network Connections to SFP 10-14 Recipients

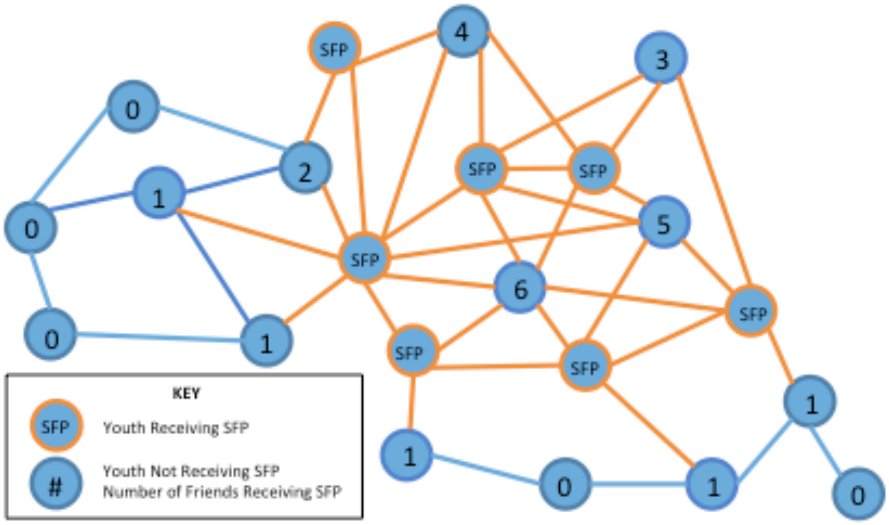
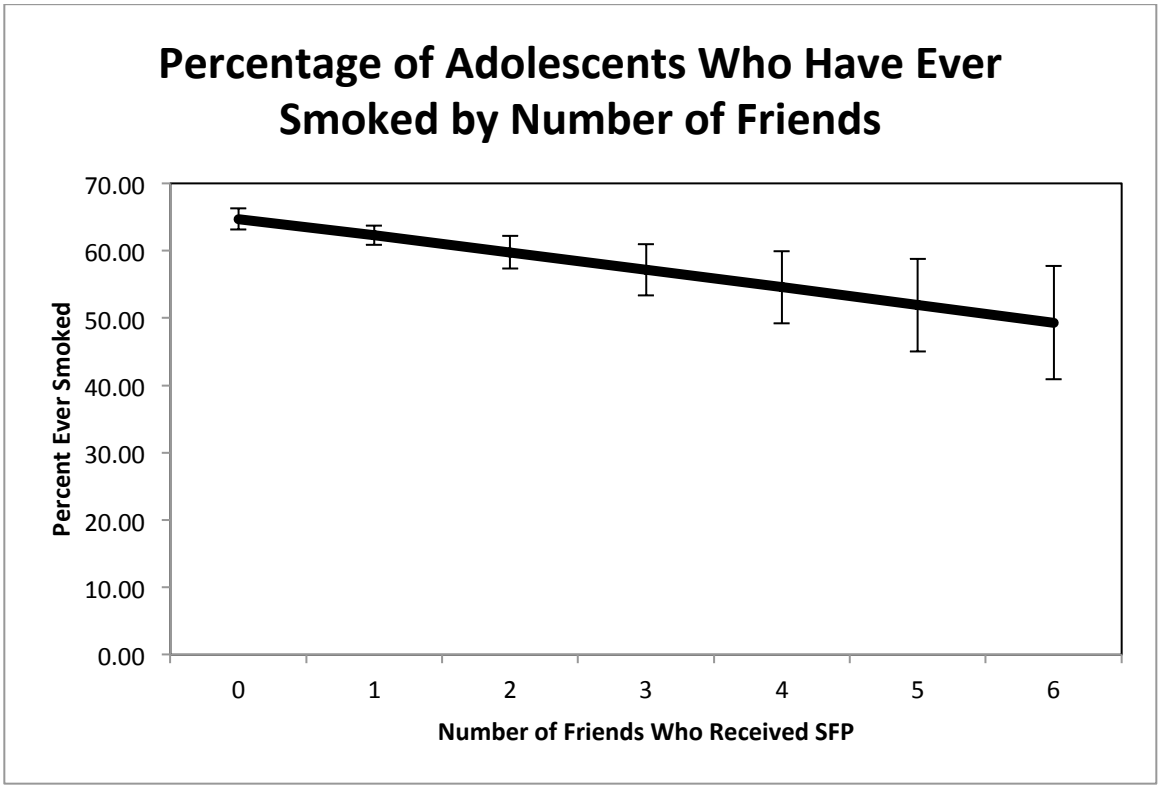


Figure 2. Estimated impact of the number of friends an adolescent has who received SFP 10-14 in sixth grade on lifetime smoking in twelfth grade. Error bars indicate 95% confidence intervals.



References

Aral, S., Muchnik, L., & Sundararajan, A. (2009). Distinguishing influence-based contagion from homophily-driven diffusion in dynamic networks. *Proceedings of the National Academy of Sciences*, *106*(51), 21544–21549. http://doi.org/10.1073/pnas.0908800106

Arrazola, R. A., Neff, L. J., Kennedy, S. M., Holder-Hayes, E., Jones, C. D., & Centers for Disease Control and Prevention (CDC). (2014). Tobacco use among middle and high school students--United States, 2013. *MMWR. Morbidity and Mortality Weekly Report*, *63*(45), 1021–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/25393220

Austin, P. C. (2011). An introduction to propensity score methods for reducing the effects of confounding in observational studies. *Multivariate Behavioral Research*, *46*(3), 399–424. http://doi.org/10.1080/00273171.2011.568786

Blok, D. J., Van Empelen, P., Van Lenthe, F. J., Richardus, J. H., & De Vlas, S. J. (2013). Unhealthy behaviour is contagious: an invitation to exploit models for infectious diseases. *Epidemiology and Infection*, *141*(03), 667–669. http://doi.org/10.1017/S0950268812000891

Christakis, N., & Fowler, J. H. (2008). The collective dynamics of smoking in a large social network. *The New England Journal of Medicine*, *358*(21), 2249–58. http://doi.org/10.1056/NEJMsa0706154

Coffman, D. L., & Zhong, W. (2012). Assessing mediation using marginal structural models in the presence of confounding and moderation. *Psychological Methods*, *17*(4), 642–664. http://doi.org/10.1037/a0029311

Crowley, D. M., Coffman, D. L., Feinberg, M. E., Greenberg, M. T., & Spoth, R. L. (2014). Evaluating the Impact of Implementation Factors on Family-Based Prevention

Programming: Methods for Strengthening Causal Inference. *Prevention Science*, *15*(2), 246–255. http://doi.org/10.1007/s11121-012-0352-8

Crowley, D. M., Jones, D. E., Coffman, D. L., & Greenberg, M. T. (2014). Can we build an efficient response to the prescription drug abuse epidemic? Assessing the cost effectiveness of universal prevention in the PROSPER trial. *Preventive Medicine*, *62*, 71–77. http://doi.org/10.1016/j.ypmed.2014.01.029

Defoe, I. N., Dubas, J. S., Somerville, L. H., Lugtig, P., & van Aken, M. A. G. (2016). The unique roles of intrapersonal and social factors in adolescent smoking development. *Developmental Psychology*, *52*(12), 2044–2056. http://doi.org/10.1037/dev0000198

Dierker, L., Braymiller, J., Rose, J., Goodwin, R., & Selya, A. (2018). Nicotine dependence predicts cannabis use disorder symptoms among adolescents and young adults. *Drug and Alcohol Dependence*, *187*, 212–220. http://doi.org/10.1016/j.drugalcdep.2018.02.037

DiFranza, J. R., Rigotti, N. A., McNeill, A. D., Ockene, J. K., Savageau, J. A., St Cyr, D., & Coleman, M. (2000). Initial symptoms of nicotine dependence in adolescents. *Tobacco Control*, *9*(3), 313–319. http://doi.org/10.1136/tc.9.3.313

Ellickson, P. L., Bird, C. E., Orlando, M., Klein, D. J., & McCaffrey, D. F. (2003). Social context and adolescent health behavior: does school-level smoking prevalence affect students' subsequent smoking behavior? *Journal of Health and Social Behavior*, *44*(4), 525–535. http://doi.org/10.2307/1519797

England, L. J., Aagaard, K., Bloch, M., Conway, K., Cosgrove, K., Grana, R., … Wakschlag, L. (2017). Developmental toxicity of nicotine: A transdisciplinary synthesis and implications for emerging tobacco products. *Neuroscience & Biobehavioral Reviews*, *72*, 176–189. http://doi.org/10.1016/j.neubiorev.2016.11.013

Fletcher, C., & Peto, R. (1977). The natural history of chronic airflow obstruction. *British Medical Journal*, *1*(6077), 1645–8. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/871704

Forrester, K., Biglan, A., Severson, H., & Smolkowski, K. (2007). Predictors of smoking onset over two years. *Nicotine and Tobacco Research*, *9*(12), 1259–1267. http://doi.org/10.1080/14622200701705357

Fosco, G. M., Stormshak, E. A., Dishion, T. J., & Winter, C. E. (2012). Family Relationships and Parental Monitoring During Middle School as Predictors of Early Adolescent Problem Behavior. *Journal of Clinical Child & Adolescent Psychology*, *41*(2), 202–213. http://doi.org/10.1080/15374416.2012.651989

Hall, J. A., & Valente, T. W. (2007). Adolescent smoking networks: The effects of influence and selection on future smoking. *Addictive Behaviors*, *32*(12), 3054–3059. http://doi.org/10.1016/j.addbeh.2007.04.008

Hanna, E. Z., Yi, H., Dufour, M. C., & Whitmore, C. C. (2001). The relationship of early-onset regular smoking to alcohol use, depression, illicit drug use, and other risky behaviors during early adolescence: Results from the youth supplement to the Third National Health and Nutrition Examination Survey. *Journal of Substance Abuse*, *13*(3), 265–282. http://doi.org/10.1016/S0899-3289(01)00077-3

Heinrichs, N., Bertram, H., Kuschel, A., & Hahlweg, K. (2005). Parent recruitment and retention in a universal prevention program for child behavior and emotional problems: Barriers to research and program participation. *Prevention Science*, *6*(4), 275–286. http://doi.org/10.1007/s11121-005-0006-1

Hill, A. L., Rand, D. G., Nowak, M. A., & Christakis, N. A. (2010). Infectious disease modeling

of social contagion in networks. *PLoS Computational Biology*, *6*(11). http://doi.org/10.1371/journal.pcbi.1000968

Jamal, A., Gentzke, A., Hu, S. S., Cullen, K. A., Apelberg, B. J., Homa, D. M., & King, B. A. (2017). Tobacco Use Among Middle and High School Students - United States, 2011-2016. *MMWR. Morbidity and Mortality Weekly Report*, *66*(23), 597–603. http://doi.org/10.15585/mmwr.mm6623a1

Jha, P., Ramasundarahettige, C., Landsman, V., Rostron, B., Thun, M., Anderson, R. N., … Peto, R. (2013). 21st-Century Hazards of Smoking and Benefits of Cessation in the United States. *New England Journal of Medicine*, *368*(4), 341–350. http://doi.org/10.1056/NEJMsa1211128

Johnston, L. D., O'Malley, P. M., Miech, R. A., Bachman, J. G., & Schulenberg, J. E. (2016). *Monitoring the Future national survey results on drug use, 1975-2015: Overview, key findings on adolescent drug use.* Ann Arbor: Institute for Social Research, The University of Michigan.

Kirke, D. M. (2004). Chain reactions in adolescents' cigarette, alcohol and drug use: Similarity through peer influence or the patterning of ties in peer networks? *Social Networks*, *26*(1), 3–28. http://doi.org/10.1016/j.socnet.2003.12.001

Lakon, C. M., Wang, C., Butts, C. T., Jose, R., Timberlake, D. S., & Hipp, J. R. (2015). A Dynamic Model of Adolescent Friendship Networks, Parental Influences, and Smoking. *Journal of Youth and Adolescence*, *44*(9), 1767–1786. http://doi.org/10.1007/s10964-014-0187-7

Liddle, H. A. (2004). Family-based therapies for adolescent alcohol and drug use: research contributions and future research needs. *Addiction*, *99 Suppl 2*, 76–92.

http://doi.org/10.1111/j.1360-0443.2004.00856.x

Lippold, M. A., & Jensen, T. M. (2017). Harnessing the strength of families to prevent social problems and promote adolescent well-being. *Children and Youth Services Review*, *79*(December 2016), 432–441. http://doi.org/10.1016/j.childyouth.2017.06.066

Liu, J., Zhao, S., Chen, X., Falk, E., & Albarracín, D. (2017). The influence of peer behavior as a function of social and cultural closeness: A meta-analysis of normative influence on adolescent smoking initiation and continuation. *Psychological Bulletin*, *143*(10), 1082–1115. http://doi.org/10.1037/bul0000113

Molgaard, V., & Spoth, R. (2001). The Strengthening Families Program for Young Adolescents: Overview and Outcomes. *Residential Treatment for Children & Youth*, *18*(3), 15–29. http://doi.org/10.1300/J007v18n03_03

Phan, T. Q., & Airoldi, E. M. (2015). A natural experiment of social network formation and dynamics. *Proceedings of the National Academy of Sciences*, *112*(21), 6595–6600. http://doi.org/10.1073/pnas.1404770112

Rosenbaum, P. R., & Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. *Biometrika*, *70*(1), 41–55. http://doi.org/10.2307/2335942

Rulison, K. L., Feinberg, M., Gest, S. D., & Osgood, D. W. (2015). Diffusion of Intervention Effects: The Impact of a Family-Based Substance Use Prevention Program on Friends of Participants. *Journal of Adolescent Health*, *57*(4), 433–440. http://doi.org/10.1016/j.jadohealth.2015.06.007

Rulison, K. L., Gest, S. D., & Osgood, D. W. (2015). Adolescent Peer Networks and the Potential for the Diffusion of Intervention Effects. *Prevention Science*, *16*(1), 133–144.

http://doi.org/10.1007/s11121-014-0465-3

Schmitz, N., Kruse, J., & Kugler, J. (2003). Disabilities, Quality of Life, and Mental Disorders Associated With Smoking and Nicotine Dependence. *American Journal of Psychiatry*, *160*(9), 1670–1676. http://doi.org/10.1176/appi.ajp.160.9.1670

Simons-Morton, B. G., & Farhat, T. (2010). Recent findings on peer group influences on adolescent smoking. *Journal of Primary Prevention*, *31*(4), 191–208. http://doi.org/10.1007/s10935-010-0220-x

Spoth, R., Clair, S., Greenberg, M., Redmond, C., & Shin, C. (2007). Toward dissemination of evidence-based family interventions: Maintenance of community-based partnership recruitment results and associated factors. *Journal of Family Psychology*, *21*(2), 137–146. http://doi.org/10.1037/0893-3200.21.2.137

Spoth, R., Greenberg, M., Bierman, K., & Redmond, C. (2004). PROSPER community-university partnership model for public education systems: Capacity-building for evidence-based, competence-building prevention. *Prevention Science*, *5*(1), 31–39. http://doi.org/10.1023/B:PREV.0000013979.52796.8b

Spoth, R., Guyll, M., & Shin, C. (2009). Universal Intervention as a Protective Shield Against Exposure to Substance Use: Long-Term Outcomes and Public Health Significance. *American Journal of Public Health*, *99*(11), 2026–2033. http://doi.org/10.2105/AJPH.2007.133298

Steinberg, L., Fletcher, A., & Darling, N. (1994). Parental monitoring and peer influences on adolescent substance use. *Pediatrics*, *93*(6), 1060–1064. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8197008

Thomas, R. E., Baker, P. R., Thomas, B. C., & Lorenzetti, D. L. (2015). Family-based

programmes for preventing smoking by children and adolescents. *Cochrane Database of Systematic Reviews*. http://doi.org/10.1002/14651858.CD004493.pub3

US Department of Health and Human Services. (2014). The health consequences of smoking—50 years of progress: a report of the Surgeon General.

van Buren, S., & Groothuis-Oudshoorn, K. (2011). mice: Multivariate Imputation by Chained Equations in R. *Journal of Statistical Software*, *45*(3), 1–67.

Van Ryzin, M. J., Roseth, C. J., Fosco, G. M., Lee, Y., & Chen, I.-C. (2016). A component-centered meta-analysis of family-based prevention programs for adolescent substance use. *Clinical Psychology Review*, *45*, 72–80. http://doi.org/http://dx.doi.org/10.1016/j.cpr.2016.03.007

Varvil-Weld, L., Crowley, D. M., Turrisi, R., Greenberg, M. T., & Mallett, K. A. (2014). Hurting, Helping, or Neutral? The Effects of Parental Permissiveness Toward Adolescent Drinking on College Student Alcohol Use and Problems. *Prevention Science*, *15*(5), 716–724. http://doi.org/10.1007/s11121-013-0430-6

Xu, X., Bishop, E. E., Kennedy, S. M., Simpson, S. A., & Pechacek, T. F. (2015). Annual Healthcare Spending Attributable to Cigarette Smoking. *American Journal of Preventive Medicine*, *48*(3), 326–333. http://doi.org/10.1016/j.amepre.2014.10.012