

Community Epidemiology of Risk and Adolescent Substance Use: Practical Questions for Enhancing Prevention

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To promote an effective approach to prevention, the community diagnosis model helps communities systematically assess and prioritize risk factors to guide the selection of preventive interventions. This increasingly widely used model relies primarily on individual-level research that links risk and protective factors to substance use outcomes. I discuss common assumptions in the translation of such research concerning the definition of risk factor elevation; the equivalence, independence, and stability of relations between risk factors and problem behaviors; and community differences in risk factors and risk factor–problem behavior relations. Exploring these assumptions could improve understanding of the relations of risk factors and substance use within and across communities and enhance the efficacy of the community diagnosis model. This approach can also be applied to other areas of public health where individual and community levels of risk and outcomes intersect. (*Am J Public Health*. 2012;102:457–468. doi:10.2105/AJPH.2011.300496)

Although federal and state laws and policies seek to prevent adolescent use of alcohol, tobacco, and illegal recreational drugs, many youths nonetheless experiment with these substances, and some become regular users. Evidence suggests that the earlier exposure occurs, the more likely it is that the individual will become a regular user or abuser later in life.^{1,2} Considerable research has focused on the factors that lead adolescents toward experimentation and substance use.^{3,4} This important body of research is used to guide decisions about prevention programs and policy.

Most research concerns relations of risk factors to problem behaviors at the level of individuals, but decisions regarding prevention policy are frequently made at the community level. A major gap in our understanding, which may affect resource and policy decisions, concerns the distribution of risk factors and relations of risk factors to problem behaviors both within and across communities. To the extent that the distributions of risk factors and problem behaviors, or the relations between them, are not uniformly distributed across the entire population, regardless of community boundaries,

the use of individual-level data to inform community-level intervention planning may be inappropriate.

The potential disjunction of individual-level research and community-level assessment, priority setting, planning, and implementation has not been thoroughly addressed. Relatively little multilevel epidemiological data have been examined to assess risk factor–outcome associations among adolescents both within and across communities. The translation of individual-level findings to community-level policies assumes that correlations detected at the individual and the community level are identical. This assumption belongs to either the ecological or the atomistic fallacy category, depending on whether the group-level dynamics are assumed to correspond to individual-level findings or vice versa.⁵ Moreover, and perhaps more important, the use of individual-level data assumes that the role of risk factors does not vary across communities.

COMMUNITY EPIDEMIOLOGY

Exploration of the community epidemiology of substance use will generate a broader base of

evidence to guide effective community-based decision-making regarding programs and policies to reduce youth substance use. Results of such research may be useful beyond the field of substance use; many of the same individual, family, peer, school, and community risk factors also predict other outcomes, such as violence, dropping out of school, and risky sex.² Moreover, this approach is relevant for other types of public health problems. Adolescent substance use is a revealing example because of the relatively advanced state of prevention related to substance use compared with interventions targeted at other adolescent problems.⁶ However, the epidemiological study of substance use may now lag behind the state of the prevention and intervention tools available to communities.

Terminology

I use the term *risk factors* to refer to both risk and protective factors that increase or lower the probability of substance use. Although *protective factors* can be more narrowly defined as factors that buffer individuals from the effects of risk factors (implying a statistical interaction), I define them more broadly, and the absence of a protective factor can be considered a risk factor. Moreover, *risk factor* refers to a causal influence on a problem behavior, not merely a correlate. Thus, *risk factor* is similar to the concept of an exposure and refers both to external agents and influences and to processes and conditions internal to the individual (e.g., poor self-regulatory capacity or endorsement of certain attitudes) that contribute to unhealthy problem behaviors.

Risk sometimes serves as shorthand for risk factors, often denoting a large range of risk factors. I use *outcome* to denote problem behaviors. *Community* refers to meaningful geographical entities; however, it may also connote nongeographic communities founded on culture, identity, or interaction patterns.

Community-Focused Prevention

A practical reason for examining patterns of community health (defined variably; e.g., school catchment areas, neighborhoods, or counties) is that the community is often the level at which decisions are made regarding the distribution of resources among competing policies and programs. According to the US *National Drug Control Strategy*, “The community is where substance abuse occurs and where prevention must happen.”^{7(p14)} In some cases, the intervention itself occurs at the level of the community—as in the case of environmental interventions or large behavioral health interventions.⁸ Yet, even for interventions directed to individuals (e.g., a nurse’s home visit to a pregnant mother), practicalities usually entail an organized community-level entity (e.g., an agency, hospital, or school) accessing and directing resources. Although funding guidelines and organizations vary according to the degree of local influence in decision-making, no community organization is completely isolated from formal and informal influences of other local stakeholders. Furthermore, intervention selection and resource allocation decisions are often conducted by either counties or states according to assessments of local priorities and needs.

Community coalitions are a popular approach for implementing public initiatives on substance abuse and other problems. More than 5000 antidrug coalitions operate in the United States alone.⁹ Consequently, research has focused on ways that community culture, leadership, institutional infrastructure, and social capital influence the adoption, implementation, effectiveness, and sustainability of community-level interventions.¹⁰ During the past decade, we have evaluated^{11–13} Pennsylvania’s implementation of Communities That Care, a program that attempts to infuse prevention science principles into communities’ approaches to improving adolescent outcomes.¹⁴ This program’s model calls for a prevention board comprising local residents, agency staff, and decision-makers; a community risk and resource assessment; prioritization of elevated risk factors; and selection and implementation of evidence-based prevention programs targeting those risk factors. The model is effective, and trial findings have shown improvements in population-based youth outcomes.^{11,15}

Advances in community prevention such as Communities That Care have led to an emerging best-practice model called community diagnosis, in which communities systematically identify risk factors that are elevated compared with national and state norms and then select preventive interventions targeting those elevated risk factors. The community diagnosis approach is being instantiated in communities through state and federal policies and programs; for example, it forms the basis of the Center for Substance Abuse Prevention’s strategic prevention framework. Community prevention policy initiatives sponsored by the US Departments of Justice and Education, the Office of National Drug Control Policy, and the US Air Force also support the community diagnosis model. Similar models are disseminated internationally.

Research on Community Effects

In the past few decades, increasing interest has focused on the effects of neighborhoods and communities on child and adolescent health, academic performance, delinquency, and substance use.^{16–21} Several important contributions have spurred interest in these directions: for example, Bronfenbrenner provided a theoretical framework highlighting the importance of context for developmental adjustment and maladjustment,^{22,23} and Wilson raised researchers’ interest in neighborhood effects by highlighting the increasing concentration of poverty in urban neighborhoods.²⁴ In 1990, Jencks and Mayer provided a now-classic description of several potential mediating processes responsible for the link between neighborhood poverty and children’s health, such as access to institutional resources and collective socialization.²⁵

Two complementary reviews, in 2000²¹ and 2002,¹⁸ agreed that socioeconomic status differences across neighborhoods were linked to differences in children’s health and problem behaviors and that limited evidence implicated mediating factors such as institutional resources, collective efficacy and social control, neighborly ties and family relationships, and peer influence. Both also underlined the need to link theory to study design, and the availability of multilevel regression models facilitated appropriate analyses of individuals nested within communities.²⁶ Over the past

decade, research has continued to examine the impact of place-based social processes such as social capital and collective efficacy on broad indicators of individual well-being, such as children’s mental health,²⁷ adolescent health,²⁸ and adult mortality.²⁹

Research on community effects on substance use is still at an early stage, and findings have been mixed, ranging from no influence to limited influence to strong influence on substance use.^{30–35} It is unclear to what extent mixed results across studies are attributable to differences across samples, measures, and methodological approaches. Moreover, findings may differ for various substances; for example, one study revealed higher rates of lifetime cigarette and alcohol use among preteens in advantaged than in disadvantaged areas, but lower rates of marijuana use.³⁰

The study of community effects involves, at a minimum, the examination of differences between communities in either levels of risk factors or health-related and psychosocial outcomes. However, the emerging body of work on the effects of communities has only intermittently touched on whether relations between risk factors and outcomes vary across communities. Proceeding on the assumption of universal, generalizable relations across communities may, as Biglan et al. suggest, impede the discovery of important local relations.³⁶ Indeed, a tenet of epidemiology is that whether an exposure (risk factor) leads to disease depends on the distribution of other factors.³⁷ Bronfenbrenner’s influential ecological model focuses on context-driven differences: communities may differ not only in the overall level of risk or outcomes, but also in the relations between risk factors and outcomes.²³ In fact, it would be surprising if individual-level risk factors operated the same way regardless of community context.³⁸

TESTABLE ASSUMPTIONS IN THE COMMUNITY DIAGNOSIS MODEL

Community epidemiology should denote research focusing on the distribution and impact of risk factors within and across geographic communities. Such research examines patterns of risk factors and outcomes with direct pertinence to public health policy decision-making and is distinguished by its

ability to separate individual- from community-level risk.³⁹ It is crucial for such research to question several assumptions embedded in the use of individual-level epidemiology, to inform the community diagnosis approach to formulating community-level prevention policies and decisions. Included among these assumptions are that elevations in risk factors can be judged against average levels of risk factors across communities, that risk factors operate similarly across communities, and that risk factors, and their links to outcomes, are stable across cohorts. I propose turning such assumptions into new research questions. The answers to these questions will provide us with not only a more complete understanding of communities, but also better guidance for the efficient targeting of prevention resources.

Prevention Should Target Elevated Risk Factors

It is often assumed that intervention and resource allocation should be based on the elevation of a community's risk and protective factors, which are determined via comparison of a community's scores to the population average. A basic question for community decision-making regards the threshold at which a risk factor requires attention and resources. Although we can observe the absolute level of a risk factor (e.g., percentage of families below the poverty line in a community), some measures use relative rather than absolute response scales. For example, attitudes toward the risk of drug use or family cohesion are assessed by Likert scales with responses such as agree strongly through disagree strongly or a little versus some versus a lot.

A common approach to understanding levels of risk factors measured with such response scales is to compare a community's average level to the average score across all communities. Decision-makers can then assess which risk factors are more elevated in their community than in other communities. For example, the Communities That Care Youth Survey provides a comparison of a community's average scale scores for risk factors to representative youth populations assessed in other communities via a T score (i.e., a score where the mean = 50 and SD = 10). This comparative approach is an empirically grounded and rational approach for understanding risk and

prioritizing intervention targets. Although this approach is valuable and easily understood, it has some embedded assumptions about assessing risk factors for resource allocation decisions.

The definition of elevated risk factors in this approach entails the assumption that the average score (i.e., T score = 50) represents a normal community—a community that requires no attention to this risk factor. However, most US communities have levels of risk factors (and adolescent substance outcomes) that are unacceptably high,⁷ so assuming that average is normal might be questionable. I have never encountered a community that did not want either to reduce risk factors (e.g., improve family communication, encourage school bonding, reduce intentions to use, change norms) or to reduce underage drinking.

Another issue with this approach is that the comparison of levels of risk factors within a community is based on their respective relative elevations compared with state or national averages. That is, the highest-priority risk factor in a community has a higher T score than other risk factors. Because each risk factor T score is determined by reference to the average of the comparison population, which risk factor is most elevated in a community depends not only on the risk factor scores for that community but also on the average scores for all the risk factors in the comparison population.

Finally, addressing the most highly elevated risk factor with this approach will not necessarily lead to the greatest impact on the outcome. Risk factor X may have a higher T score than risk factor Y, but the greatest possible reduction in alcohol use may not be achieved by reducing X rather than Y. If Y is more predictive of alcohol use than X, a reduction in Y may yield greater benefits than a reduction in X, regardless of their respective elevation in comparison with national averages. For example, if a particular risk factor for underage drinking, such as family conflict, is highly elevated in a community (compared with all other communities), and another risk factor, such as positive adolescent attitudes toward drinking, is not as highly elevated, alcohol attitudes may nonetheless be more predictive of alcohol use than is family conflict. Thus, a community seeking to lower underage

drinking may achieve greater gains by targeting resources toward changing adolescent attitudes rather than toward ameliorating family conflict, regardless of relative T scores.

How then can we gauge the extent to which a risk factor should be targeted? A different approach is to examine this question in the framework of epidemiological statistics regarding the population-attributable fraction.⁴⁰ This framework allows estimation of the proportion of an outcome, such as substance use, that is attributable to a risk factor. This approach changes the question from, Is a risk factor elevated compared to other communities? to How much reduction in substance use can be expected if prevention efforts reduce the level of the risk factor in a community by a certain amount? Such information may be useful to a community planner seeking to apply scarce resources to prevention. (Several other important issues involved in translating data into indices of risk factors, including the use of additive rather than relative measures of effect, are outside the scope of this article.^{37,41})

Of course, additional considerations arise from a community planning perspective. For example, how large an impact would available interventions be expected to have on a risk factor? Such information could be derived from effect sizes in randomized trials of those programs. Cost of implementation is also important to decision-making. Determining the expense of high-quality implementation of a particular prevention program requires examination of the required human capital and infrastructure in a community; such systemic resource and capacity issues are too often ignored.⁴² All of these issues might be considered by a community attempting to maximize the impact of available resources on the problem behavior.

The selection of which risk factor to target depends not only on which risk factor is most elevated, but also on how closely the risk factor is related to the problem behavior, expected program impact and cost, and community capacity. Thus, a planner might want to calculate the expected impact on the problem behavior for approaches targeting several different risk factors before allocating resources. Selection of risk factor target and prevention approach may then be made together.

Comparison of risk factors and outcomes across communities can still yield valuable

information. For example, if a risk factor is relatively low in a community, planners might wonder whether it can be reduced further—and at what cost. In some cases, evidence suggests that prevention programs targeting substance use have a greater effect with higher- than with lower-risk youths.⁴³ Greater impact might therefore be achieved by targeting risk factors that are elevated. In addition, communities are often—and naturally—interested in comparing their levels of risk and protection with other communities.

Differences in Risk Between Communities Are Substantial

Another insufficiently examined assumption is that the distribution of risk factors in the population is largely determined by geography: communities differ substantially by location in elevation of overall risk and in elevation of particular risk domains.

This assumption may seem trivial: of course we expect that communities differ in levels of risk for adolescent substance use. Our subjective perception is that some communities have much higher levels of risk factors and rates of substance use. Still, validating this assumption for both risk factors and problem behaviors, across a wide range of communities, is essential. If most communities do not appreciably differ in risk factors levels, then the cost and effort to assess levels of risk factors in each community may not be necessary.

An obvious question concerns what constitutes a meaningful difference in a risk factor across communities. This question arises regardless of whether a given risk factor is characterized by an absolute measure, such as the number of liquor stores in a community,⁴⁴ or a score on a Likert scale assessing an aspect of community culture or social capital. Numerous methods can characterize differences in community risk: the range of risk levels across communities, the levels of risk factor scores across certain percentages of the distribution (e.g., risk factor scores for communities ranked at the 25th, 50th, and 75th percentiles), and the standard deviation of community-level scores. For example, Ennett et al. reported ranges in lifetime use for sixth graders ranging from 20% to 80% across study schools for alcohol, 0% to 55% for cigarettes, and 0% to 28% for marijuana.³⁰ Although the differences indicated by

these ranges appear striking, a range only provides information about the low and high scores; we still require information about meaningful differences across a substantial number of communities.

Proportion of variability. Assessing variability across communities is especially complicated when the measure of a risk factor is an individual-level measure, such as parent–child conflict or attitudes toward substance use. In such cases, researchers typically focus on the proportion of overall variability in risk factors or problem behaviors between individuals that is attributable to community membership. The most commonly used statistic is the intraclass correlation (ICC), which measures the proportion of variability in risk factor scores attributable to differences between communities. (The ICC has numerous weaknesses, and alternative measures are available^{37,45,46,47}; the issues discussed here do not depend on use of ICC as the measure of clustering.) Researchers have frequently found statistically significant ICCs for adolescent and adult substance use across schools or communities in the range of 1% to 5%.^{30,48,49} (Ennett and Haws also reviewed school-based ICCs.³¹) These findings indicate that up to 5% of variability in substance use stems from differences between communities. Similar rates of community-level clustering have been reported for individual-level risk and protective factors.⁵⁰

Small proportions of variability in substance use across schools have led some to conclude that school differences are not consequential.³¹ Although the proportions of overall variability attributable to community differences may be small, the denominators in those proportions are large. In other words, youths vary substantially within any community, and thus the proportion of variability caused by community clustering can appear small in contrast.

Statistical significance is not a sufficient guide regarding the importance of a particular level of clustering: statistical significance indicates that the detected degree of clustering at the community level is not the result of chance but not whether such findings are meaningful or important. Even when statistically significant clustering is demonstrated, practical questions remain: How much clustering is meaningful for purposes of prevention policy and planning? How much clustering

justifies the added burden of community-level diagnosis and intervention planning? The answers may require simulation of the relative effects of community-specific targeting of risk factors versus delivering interventions similarly to all communities. Modeling this process would require simulating a range of community risk factors and problem behavior profiles and clustering, formulating a range of decision rules regarding targeting of risk factors and estimating risk factor–targeted intervention effect sizes. Such an approach would demonstrate the benefit, at different points within this matrix of profiles, decision rules, and effect sizes, of community-specific risk factor assessment and targeting. Benefits and costs could then be compared for the population as a whole and for individual communities.

Heterogeneity. The issue of heterogeneity across communities is important because the proportion of community clustering that is found is affected by the heterogeneity of the communities represented in the data set. Van Horn et al. point out that the same absolute level of clustering of individual risk factors within communities yields a higher ICC in a homogeneous set of communities (i.e., communities whose populations have similar levels and distributions of a variable) than in a heterogeneous set of communities.⁵¹ This occurs because ICCs (and similar measures) are constructed as a proportion; as the denominator (overall variance) decreases, the fraction (or proportion) increases. Unfortunately, few data sets with information on risk factors and substance use outcomes are derived from representative community and representative individual sampling.

Most findings on this issue have relied on nonrepresentative samples (typically collected to examine other research questions), likely yielding overestimates of clustering. For example, group-randomized trials may impose eligibility requirements on community participation to ensure equivalence of units, as required by the trial design. Even in research without restrictive eligibility criteria, selection bias in convenience samples can reduce heterogeneity (and selection bias of course can make drawing any inferences from a data set problematic).

This methodological problem suggests an alternative approach: risk factors can be

assessed not within a single community, but within a set of homogeneous communities. If clustering is minimal, then the set of communities can be treated as a single unit. For example, if contiguous communities of north-east Iowa demonstrate little clustering, then they should also demonstrate similar risk factor profiles. Assessment, program selection, and resource allocation decisions can therefore be made for that set of communities as a whole. This approach might also facilitate policies and prevention decisions for sets of communities that are not contiguous but that share certain characteristics (e.g., suburban communities).⁵² However, even if sets of homogeneous communities are created, it remains to be determined whether large enough differences exist between the sets of communities to justify treating the sets differently and whether small enough differences exist within the sets to justify treating the communities within each set similarly.

Defining community. The definition of community can also have a strong impact on assessment of risk factors. Should risk assessment be carried out at the level of a block, census tract, neighborhood, school building catchment, school district, county, or metropolitan area? One of several competing considerations is the nature of the problem being examined: Are there natural communities of individuals created by a setting (e.g., students in a school) in which individuals interact and influence each other regarding the problem? Or is there an institutional context that creates a natural community? For example, a school might be a reasonable community for considering patterns of truancy and dropping out.

A second consideration is that the community should be defined so that it yields substantial clustering of risk factors or problem behavior. In an unpublished analysis of data from the Pennsylvania Youth Survey,⁵³ my colleagues and I found more clustering at the level of school buildings than of the school district for adolescent risk factors and outcomes. In this case, defining the district as the community of interest may limit our ability to target resources and programs where they are needed.

Pragmatic issues should also be considered. Because data are expensive to collect, we frequently must make do with what is available. For example, it is easier to find census data aggregated to the level of school districts

(e.g., from the National Center for Education Statistics) than to the geographic areas covered by individual schools.

Finally, if such data are to inform targeting and allocation of resources, the level of assessment needs to be linked to boundaries defined by institutional decision-making, political will, and organizational implementation capacity. This issue could arise where assessment of a metropolitan region seems to be most scientifically appropriate, but no institutional body is responsible for making decisions affecting the metropolitan area as a whole. Clustering might be most important at a school level, but giving more services to one school over another might be perceived as discriminatory or stigmatizing by local stakeholders. Assessment might be most appropriate at the level of politically and socially defined neighborhoods, but no service organization is able to implement services that conform to these boundaries. In such instances, scientific preference may give way to practical realities.

Risk-Outcome Associations Are Equivalent, Independent, and Linear

Risk and protection factors are often assumed to have roughly equivalent, independent, and linear relations to problem behaviors. Decision-makers are faced with a wide range of identified risk factors linked to adolescent substance use, and it is likely that some risk factors influence substance use more strongly than others. However, few studies have taken a comprehensive approach to assessing the differential association of various risk factors with substance use, leaving decision-makers with little guidance regarding the relative importance of different domains of risk.⁵⁴

In the absence of guidance from research, decision-makers may assume that assessed risk factors are equivalent in their impact on substance use initiation and abuse. Thus, research is needed to understand the relative influence on the emergence of underage drinking of, for example, community factors versus family factors versus peer factors versus individual attitudes. One obstacle that researchers face in determining the relative influence of risk factors on outcomes is the complex way that these factors may intersect and interact.

Independent versus covarying risk. If risk factors in a community are targeted according

to which are most elevated, an implicit assumption is that these factors are independent of one another in their effects on problem behaviors. This assumption of independence requires 2 conditions: that risk factors do not covary to an appreciable extent and that they do not interact in predicting problem behaviors.

The first condition is frequently not present. At the level of individuals, risk factors tend to accumulate.⁵⁵ For example, children raised by single mothers are more likely to live in poverty, and poor children tend to attend poorly performing schools; thus, children in single-parent families are more likely to receive lower-quality education.

Associations between risk factors have several origins; understanding the causal relations among risk factors and their relations to outcomes is important for prioritizing and targeting risk factors. One cause of covariance is that 2 risk factors may be influenced by a third variable. For example, poverty may lead to poor-quality parenting (attributable to increased stress experienced by parents⁵⁶), as well as to youths' unsupervised association with antisocial peers (because parents are working long hours). If negative family relations and deviant peer association are both elevated in a community, but not because one causes the other, targeting both might make sense.

However, risk factors may covary because one risk factor triggers the action of another. For example, low parental engagement, high levels of negativity, and poor supervision may lead a youth to associate with antisocial peers. The association with antisocial peers would then be the proximal risk factor for underage drinking; thus, association with antisocial peers mediates the effects of poor parenting on drinking. When a risk factor mediates the impact of another, several possibilities for intervention may exist: for example, an effort could be made to improve parenting and to allow more competent parenting to deter deviant peer association. However, improving parenting in a community may be more difficult than reducing unsupervised deviant peer association, and thus policymakers might decide to focus only on the easier-to-achieve option. Of course, with sufficient resources, both risk factors could be targeted, with the hope that some impact on drinking will be

achieved through intervening at each stage along the pathway. To the extent that the risk factors are causally related, targeting only a single risk factor may be justified. Where risk factors (moderately) overlap but are not causally related, a stronger case can be made for targeting both.

The covariance among risk factors makes estimation of their distinct influence on outcomes complicated. For example, if deviant peer association mediates the influence of poor parenting on drinking, regression models that incorporate both risk factors will yield a smaller estimate of influence in predicting drinking than will a model that measures only parenting. Usually, it is thought that controlling for several other factors helps elucidate the influence of a variable more precisely. But controlling for a mediating variable (e.g., deviant peer association) will lead to underestimation of a significant part of the total explanatory power of a risk factor (e.g., parenting quality). This underestimation typically occurs for distal factors (e.g., community disorganization) that are mediated by more proximal factors (e.g., peer and family factors).

For planning purposes, it is important to know how closely risk factors covary at the community level. An extreme case arises when risk factors are very strongly correlated across communities: an elevation on one risk factor scale for a community is quite likely to be accompanied by an elevation on another risk factor scale. All risk factors rise and fall together. In this case, communities are distributed along a continuum of overall risk, with few important differences in the profiles of various risk factors across communities. Consequently, it would be most helpful to understand the overall level of risk rather than the particular profile of risk factors for a community. Although this is an unlikely scenario, certain sets of risk factors may be highly correlated. Moreover, a geographically or otherwise defined subset of communities may have strongly linked sets of risk factors. Such linkages across risk factors could simplify assessment and planning.⁵⁷

Independent versus interacting risk. An interaction effect operates when the impact of a risk factor depends on the presence of another risk factor. For example, family risk may be more strongly linked to problem behaviors for

youths who have negative peer relations than for youths with positive peer relations.⁵⁸ Thus, understanding how strongly family risk is associated with the outcome depends on knowing the level of positive peer relations. To the extent that risk factors interact with each other, a focus on elevations among single risk factors may ignore the accentuation or amelioration of risk that occurs when other risk factors also are elevated. Some research indicates that interactive effects of risk are important to consider.⁵⁹

Some have defined protective factors as those conditions and processes that buffer youths from the negative effects of risk factors, thus implying an interaction in which the presence of protection reduces or eliminates the association between risk factors and behavior problems. Some researchers have counseled a greater focus on the promotion of protective factors to enhance youth outcomes.⁶⁰ Little work has comprehensively examined the relative potential benefits of strengthening protection versus reducing risk factors.⁵⁹ To the extent that protective factors buffer the effects of high levels of risk factors, linear models relating levels of risk factors to outcomes will be incomplete guides to prevention. A particular class of risk factor interactions occurs when the effect of a risk factor at one level (e.g., individual attitudes) is conditioned by a factor at another level (e.g., community collective efficacy).

Nonlinear associations. Relations between a risk factor and outcome sometimes occur in a nonlinear pattern for reasons other than interactions among risk factors. For example, a risk factor may not promote substance use until a certain level of risk is reached; conversely, a risk factor may lead to use up to a certain threshold, with further increases in the risk factor not linked to further increases in use. For example, findings indicate a nonlinear relation in which the positive link between associating with antisocial friends and alcohol use no longer holds above a certain threshold of antisocial peer association. That is, among the subgroup of youths who associate most with antisocial friends, no relation of antisocial peer association with levels of alcohol use was detected.⁵⁴ Thus, a threshold existed above which further increases in antisocial peer association no longer predicted increases in levels of alcohol use. Such nonlinear relations may

complicate prevention planning. For example, targeting antisocial peer associations at a population level may lead to reductions in use among much of the population but not among high-risk youths—unless the impact on antisocial peer association is sufficiently strong to reduce their level on this risk factor below the threshold.

Another source of nonlinearity may arise from the variable role and meaning of substance use in adolescence. For example, alcohol experimentation and use become normative in later adolescence, and youths who are more socially competent and popular and who belong to cohesive friendship groups may be more likely than less socially competent youths to use alcohol.^{61,62} Because low social competence is generally a risk factor for problem outcomes, it may be useful to explore whether such nonlinearities exist such that youths with low and high levels of competence have greater risk for drinking than do youths with average social competence.

Associations Are Consistent Across Communities

The majority of research assumes the existence of pathways from risk factors to problem behaviors that are robust across communities.³⁶ However, it is also possible that associations between risk factors and problem behaviors differ across communities. If these associations are not universal, then decision-makers will be challenged to select interventions that target the risk factors that are most influential in their particular context.

Relations between risk factors and outcomes may not be universal for several reasons. Risk factors may be distributed and affect outcomes in idiosyncratic ways that depend on local history and culture. Some dimension of local context, such as rurality or urbanicity, average socioeconomic status, social disorganization, social capital, or prevalence of the problem behavior may create systematic differences in how risk factors operate.⁵⁹ For example, Chuang et al. found that individual socioeconomic status was a weaker predictor of adult alcohol use in neighborhoods with relatively greater social disorganization.⁶³

A study linking risk factors and substance use among preadolescents in 3 communities revealed that levels of risk factors varied across communities and that associations between risk

factors and problem behaviors varied by community.⁶⁴ This study illustrates how a prevention strategy might be formulated differently by examining levels of risk factors or within-community associations of risk factors with outcomes. For example, low school commitment was the only risk factor that was elevated in one community and thus might appear to be a strong target for prevention. However, low school commitment was not correlated with problem behaviors in that community. Thus, promoting school engagement could reduce the level of that risk factor without affecting problem behaviors in that community. The reverse situation is also possible: a risk factor may not be highly elevated in a community, but it may be strongly linked to the outcome in that community.

It is important to empirically examine whether associations between risk factors and outcomes differ significantly across communities. If so, it may be useful to assess not only levels of risk factors, but also the strength of associations between risk factors and outcomes in single communities or sets of similar communities. Research has begun to examine whether contexts such as school and community environments modify the salience of some risk factors.^{65–68} Such examinations assess cross-level interactions between risk factors at a more proximal level (e.g., individual or family characteristics) and a more distal level (e.g., school or community characteristics).^{69,70}

One perspective on cross-level interactions is the buffering hypothesis, which holds that protective factors are likely to be especially salient in high-risk environments.⁷¹ For example, a supportive school environment might offer protection against negative outcomes for adolescents with high peer or family risks.^{68,72} Brook et al. found such a buffering pattern: peer substance use predicted greater adolescent substance use in less well-organized and achievement-oriented schools than in better-organized schools.⁷³

However, cross-level interactions can take the opposite form: protective factors may be less salient in high-risk contexts, possibly because risk factors such as negative peer influence swamp the protective effect of positive individual or family factors. In 2 studies, my colleagues and I found that protective factors (both individual and family) were less

predictive of adolescent substance use in high-risk than in low-risk contexts.^{52,59}

Recent studies have examined the moderating impact of contextual factors on the prediction of a range of other adolescent behaviors. For child conduct disorder, a review of 44 studies that reported significant interactions between family factors and contextual risk found a predominance of evidence for a buffering effect: family protective factors were generally found to be more influential in high-risk contexts.⁷⁴ Yet several studies reported findings inconsistent with the buffering hypothesis, suggesting that protective family factors are less influential in high-risk contexts. A number of studies of other dimensions of adjustment have found that the influence of protective family factors is stronger in less adverse contexts, suggesting a swamping effect for externalizing problems, such as delinquency^{75,76} and youth violence,⁷⁷ as well as internalizing problems, such as anxiety and depressive symptoms.^{78–81}

The ecological or atomistic fallacies may produce a finding that a risk factor does not lead to a problem behavior at the individual level within a community but may be associated with the level of problem behaviors across communities. If a community-level association exists—between, for example, low school commitment and substance use—then reducing the level of a risk factor in a community may affect outcomes regardless of whether there is an individual-level association. For example, low school commitment may be particularly low in a community because the school system is disorganized and underperforming, and low levels of teacher morale and quality evoke low levels of school commitment among all students. The overall low levels of school commitment among students may contribute to a general lack of engagement in prosocial activities and a rejection of prosocial norms that diffuse among the entire student population. This local culture may then provide a context in which antisocial norms develop and facilitate increased problem behaviors among the community's youths. However, such a process may unfold without an individual-level link between school commitment and substance use. It is possible, then, that raising school commitment (via improvements in the school itself) for all students would increase bonding to prosocial adults and activities in general,

with a consequent impact on the norms of the peer culture—with the ultimate effect of reducing levels of problem behaviors.

The distinction between the causes of problems in individuals (cases) and the causes of differences in rates of problems across populations (incidence rates) is not new. Rose's classic work on sick individuals versus sick populations discussed this issue 27 years ago.⁸² Causes of cases in a population may not be the same as the causes of incidence rates; Rose believed that genetic variation is more likely associated with cases (i.e., susceptibility of individuals within a population) but that environmental differences are more likely the cause of variability in rates across populations. It remains important to determine whether variation in a risk factor is causally related to variation in the emergence of disorder within or across populations.

One problem in conducting community-level research is the sheer number of communities needed in a single study, with sufficient sampling within communities to estimate within- and between-community effects. Such studies are expensive to mount. Moreover, the absence of random sampling at both community and individual levels may lead to biased estimates. One solution to these barriers of expense and bias is to conduct secondary data analyses with large data sets, combining results across analyses where appropriate to produce findings that are more generalizable. Recently, my colleagues and I reported significant and substantial variability in the influence of individual, peer, family, school, and community risk factors on alcohol use across communities (M. E. F., unpublished data). Moreover, the strength of the influence of risk factors tends to cluster; some communities have generally stronger relations between each domain of risk and alcohol use than do other communities. A next step is to investigate why patterns of use in some communities appear more lawful (i.e., use is a more accurate predictor of risk).

Associations Are Stable Across Time

Finally, the assumption in the community diagnosis model that the strength of associations between risk factors and problem behaviors is stable across age and other cohorts should be questioned. Developmental

researchers have been interested in how the influence of specific risk factors changes as a result of developmental changes in youths' capacities, interests, and time use, as well as in the changing social organization of school and peer relations. For example, family risk factors appear to be more closely related to outcomes for younger than for older adolescents.⁸³ Possibly family conflict in early adolescence leads to heightened risk in other domains, such as negative peer associations, which then transmit the impact of earlier family conflict forward into later adolescence. Thus, targeting family conflict in early adolescence—even if its direct influence on problem behaviors diminishes over time—may be a reasonable strategy. However, we do not know whether developmental changes in salient risk factors are uniform across communities or whether change differs across different kinds of communities.

A second set of questions concerns whether levels and influences of risk factors are stable across cohorts. Implicit in most research designs is the assumption that level of risk in a community is fairly stable, representing a constellation of factors such as the population composition, social structure, and social processes. However, it is also possible that risk profiles of different cohorts vary: a risk factor may be elevated for one cohort and not another. Typically, considerations of historical time relate to large-scale events and trends, such as new laws and regulatory mechanisms, emerging patterns of illicit substance distribution and use, and cultural shifts. Variability might also emerge across local cohorts of youths as a result of local events and trends (e.g., changing demographics, natural disasters). Furthermore, it is possible that seemingly random variability in levels of risk factors and substance use stems from the dynamics of peer networks, norms, and use patterns of particular cohorts. Little empirical work has been done in this area, but one study revealed some degree of variability across grade cohorts.⁸⁴

If the variability across cohorts is limited, then data aggregation across cohorts will yield an improved estimate of the underlying, stable risk factor profile of the community. Aggregation across cohorts will then help smooth out random fluctuations as in a moving average approach to plotting time series

data (e.g., daily stock prices). If, however, the variability across cohorts is large, or change in levels of a risk factor tends to be in a single direction across cohorts (e.g., monotonically increasing over time), then aggregation across cohorts may obscure important information.

It is important to understand the degree of variability in specific risk factors both across development and across cohorts. For example, if a study shows that 6th- and 8th-grade students report low levels of family conflict, but 10th- and 12th-grade students report high levels, aggregating across cohorts would lead community leaders to believe that family conflict is at an average level in the community and does not deserve attention. The elevated risk of substance use attributable to heightened family conflict among youths in the community would not be addressed. Moreover, from these data alone, it would not be possible to differentiate developmental from cohort differences in levels of family conflict in this community. A developmental pattern of increased family conflict in late adolescence would suggest one prevention approach in this community, whereas cohort-level fluctuations in risk factors might suggest another, such as tailoring prevention efforts to specific cohorts.

CONCLUSIONS

My goal is to provoke discussion and research about how to measure and prioritize risk factors and how to incorporate recognition of local context into the prevention research cycle. The need for focused, programmatic, and interdisciplinary research is especially great because of the role that communities play in intervening in public health problems. Children and families are influenced by the culture, norms, and social relations of their communities. In addition, the community—defined variously from neighborhood to school district to county—is often where important political-economic decisions are made about how risk factors are prioritized and resources are applied to public health strategies.

Table 1 lists the 5 assumptions discussed here, some of the issues they raise, and recommendations for how researchers, evaluators, and decision-makers can address these issues. I hope that others will bring other dimensions to this discussion and assist in fleshing out

research and policy directions. For example, gender, ethnic, and cultural differences in the development of and risk for substance use may also contribute to differences in community risk factors and outcome associations. Strategies for understanding these gender and group differences in relation to community-level distributions of risk factors and outcomes, and ways of incorporating gender and group differences into community diagnosis prevention approaches, require careful thought and discussion.

Further Refinement of the Community Diagnosis Model

For several reasons it is important to assess community risk and protective factors in addition to identifying high-priority risk factors to target. Repeated assessment can provide information on whether an intervention is yielding the intended effect. Local assessment and prioritization discussions can also foster the buy-in of local stakeholders to public health initiatives. On the other hand, assessment is costly and burdensome to respondents. Assessment of risk and protective factors can also confer a false sense that the causes—not just risk markers—of local problems are understood, leading to frustration if expected program effects on problem behaviors do not occur.

Examination of common assumptions in community epidemiological research will lead to refinements in community diagnosis models, which should improve decision-making and intervention impact. For example, instead of focusing decision-makers on comparing risk factor levels with other communities, a community diagnosis model may indicate how much impact a reduction in a risk factor would have on a problem behavior. Such estimates of the relative magnitude of risk factor contributions to problem behaviors would clarify the predictive value of risk factors both within and between communities. The development of more sophisticated rubrics for assessing and interpreting risk factor—to—outcome patterns may increase decision-makers' ability to calculate costs and expected return on investment (i.e., reduced problem behaviors) for various intervention strategies targeting different risk factors.

TABLE 1—Common Assumptions in Epidemiological Research on Adolescent Substance Use and Recommendations to Improve Translation to Interventions

Assumption	Problems	Recommendations
Intervention policy and resource allocation is based on the elevation of community risk factors, determined by comparison of local community scores to population average.	<p>Almost all communities want to reduce problem behaviors.</p> <p>Risk factor elevation determined by national norms may not correspond to risk factor with most potential to affect outcome within a community.</p> <p>Benefit from targeting a risk factor depends in part on magnitude of potential intervention effect from available programs.</p>	<p>Use research approaches that reveal portion of outcome variation attributable to risk factor.</p> <p>When prioritizing risk factor targets, consider effect sizes for available interventions and costs of intervention approaches. This will allow estimates of potential reduction in outcome attributable to reduction in risk factor, cost for achieving X% reduction in problem by targeting Y risk factor with Z intervention, and comparison of costs across targeted risk factors and interventions for similar reductions in problem behaviors.</p>
The distribution of risk in the population is largely determined by geographic location: communities differ from each other in elevation of overall risk and elevation of particular risk domains.	<p>Little is known about how much clustering of risk factors, outcomes, and associations between them exists across communities.</p> <p>Little is known about how much clustering is meaningful for planning decisions.</p>	<p>When possible, researchers should define community in manner that is useful for decision-makers.</p> <p>Assess clustering of risk factors and use across representative sample of communities.</p> <p>If communities do not differ in risk factor and use levels and associations, then no need to assess each one.</p> <p>If set of homogeneous communities identified, then assess a sample across the communities.</p> <p>Conduct simulations to assess impact of community differences on decision-making and intervention effects.</p>
Risk and protection factors show roughly equivalent, independent, and linear relations to problem behaviors.	<p>Little is known about which risk factors are most salient for which outcomes.</p> <p>Risk factors likely are correlated, affect each other, and interact in predicting outcomes.</p>	<p>Assess relative contribution of risk factors to outcomes across development; overlapping, unique, and mediational relations; and nonlinear and interactive effects of risk factors on outcomes.</p> <p>Incorporate such information into rubrics that aid decision-makers in interpreting community risk profile data and prioritizing risk factor targets.</p>
Associations of risk and protective factors with problem behaviors are consistent across communities.	<p>Evidence suggests that context may modulate the impact of risk factors on outcomes.</p> <p>Variation in contribution of risk factors to outcomes across communities may be idiosyncratic.</p>	<p>Research should examine systematic moderators of risk factor–outcome associations.</p> <p>Community assessment should examine not only elevation of risk factors but also association of risk factors with use.</p> <p>Decision-making should incorporate community-specific risk factor–outcome associations or systematic contextual factors in interpretation and planning rubrics.</p>
The strength of associations between risk factors and problem behaviors are stable across ages and across cohorts.	<p>Contribution of risk factors to outcomes may change over development.</p> <p>Community cohort differences in risk factors, outcomes, and associations may be meaningful.</p>	<p>Investigate change in risk factor contribution over development.</p> <p>Investigate extent of cohort differences and whether differences indicate historical trend or random fluctuation. Implement more frequent community assessment if cohort differences are substantial.</p>

Conducting community epidemiological research with sufficient comprehensiveness, rigor, and replication will take time. However, interim steps can be undertaken. First, community diagnosis assessment and interpretation materials can offer guidance, derived from existing research, regarding the relative import of risk factors.⁵⁹ Second, local assessment can be easily adapted to examine within-community associations of risk factors and outcomes and thus provide community-specific information on the salience of local risk factors.

State and federal agencies (e.g., the Substance Abuse and Mental Health Services Administration) can contribute to the next stage of research and refinement of the community diagnosis approach. Federal, state, and some large regional or urban agencies can also promote analysis of existing data to examine distribution of risk factors and outcomes within and across communities and across cohorts (smaller local agencies likely do not have sufficient numbers of community units in their service areas to advance such analyses). Agencies can also multiply the benefit of current state and federal investment in surveillance and evaluation by adding longitudinal tracking of individuals within communities to current repeated, cross-sectional surveys. As just one example, the Centers for Disease Control and Prevention conducts the Youth Risk Behavior Survey every 2 years to monitor adolescent behavioral health. In 2009, it comprised a national school-based survey, 47 state surveys, 6 territory and tribal government surveys, and 23 local surveys. If even a subset of respondents were tracked longitudinally, this ongoing federal investment would yield important insights into the influence of risk factors on individual trajectories within and across communities.

Further Issues With Individual- and Group-Level Factors

Epidemiologists point out that some exposures to risk factors may be the same for all individuals in a group or community, such as disease-carrying mosquitoes or billboard advertisements for tobacco and alcohol products. How would a community epidemiology perspective apply in such case? Some individual variability may still exist in exposures: more mosquitoes may be present in homes near the

village water supply, or some youths may pass more billboards on the way to school. Even if an exposure is similar for all members of a community, individual susceptibility will differ. Research could characterize the distribution of community-level risk factors (e.g., tobacco advertising) across communities and the variability of individual vulnerability to this risk factor. It may be that tobacco advertising raises the risk for smoking among all youths, but more so for youths with low inhibitory control. In this case, the effect of the group-level risk factor is moderated by an individual characteristic.

A community epidemiological analysis could take a further step to examine whether the role of the vulnerability factor differs across communities. That is, low regulatory capacity may be more salient in some communities than in others. In one community, perhaps with a high level of informal social control and collective efficacy, the protective community context (informal control) might buffer individual susceptibility (low inhibitory control) to a community-level risk factor (tobacco advertising).

A final concern is whether the distinction between an individual- and a community-level exposure should dictate how intervention should proceed. In his classic paper on the causes of cases versus incidence rates, Rose drew a parallel to interventions targeting individuals versus populations.⁸² He recognized the need for both types of strategies; however, his discussion may have introduced some confusion. First, he discussed individual-level intervention in terms of what we now call selected or indicated prevention; that is, prevention targeting high-risk individuals who are either at high risk or have developed early signs of the problem (e.g., smoking cessation counseling for youths who have begun smoking). He contrasted such individual-focused strategies with population strategies that would be classified into distinct approaches today: (1) a universal intervention targeting all individuals for behavior change (e.g., helping all youths to resist peer pressure to smoke) and (2) an environmental policy model that attempts to change the context (e.g., enforcing restrictions on sales of legal drugs to minors).

Rose linked the intervention approach targeting high-risk individuals to a focus on causes

of cases, and he linked the population approach to a focus on causes of incidence rates. Rose's recommendation to implement a mix of different intervention strategies is optimal, but we should not draw too strong a parallel between the individual and the population sources of problems and the interventions employed. Both individual and population exposures can be addressed by both individual- and population-level interventions. For example, if poor parental monitoring accounts for variations in substance use within a community, parenting might be improved by administering a family prevention program to all parents, by administering a family program to the highest-risk families, or by promoting workplace policies that provide for flexibility in parental leave and daily schedules.

Although understanding the community epidemiology of substance use provides information about the role of risk factors in promoting problem behaviors within and across communities, it does not limit the diverse types of strategies that may be employed to address those factors. Community epidemiology should be used to understand the distribution of risk factors and influences on substance use and in that way sharpen our ability to prioritize risk factors and target interventions for maximal impact within and across communities—not to constrain the types of intervention approaches considered. ■

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References

- Kandel D, Simcha-Fagan O, Davies M. Risk factors for delinquency and illicit drug use from adolescence to young adulthood. *J Drug Issues*. 1986;16(1):67–90.
- Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. *Psychol Bull*. 1992;112(1):64–105.
- Allen M, Donohue WA, Griffin A, Ryan D, Turner MMM. Comparing the influence of parents and peers on the choice to use drugs. *Crim Justice Behav*. 2003;30(2):163–186.
- Ryan SM, Jorm AF, Lubman DI. Parenting factors associated with reduced adolescent alcohol use: a systematic review of longitudinal studies. *Aust N Z J Psychiatry*. 2010;44(9):774–783.
- Diez-Roux AV. Bringing context back into epidemiology: variables and fallacies in multilevel analysis. *Am J Public Health*. 1998;88(2):216–222.
- Institute of Medicine. *Preventing Mental Disorders Among Young People: Progress and Possibilities*. Washington, DC: National Academies Press; 2009.
- National Drug Control Strategy. Washington, DC: Office of National Drug Control Policy; 2010.
- Prinz RJ, Sanders MR. Adopting a population-level approach to parenting and family support interventions. *Clin Psychol Rev*. 2007;27(6):739–749.
- Community Anti-Drug Coalitions of America. Available at: <http://www.cadca.org/about-us>. Accessed August 14, 2011.
- Feinberg ME, Chilenski SM, Greenberg MT, Spoth RL, Redmond C. Community and team member factors that influence the operations phase of local prevention teams: the PROSPER project. *Prev Sci*. 2007;8(3):214–226.
- Feinberg ME, Jones D, Greenberg MT, Osgood DW, Bontempo D. Effects of the Communities That Care model in Pennsylvania on change in adolescent risk and problem behaviors. *Prev Sci*. 2010;11(2):163–171.
- Feinberg ME, Bontempo DE, Greenberg MT. Predictors and level of sustainability of community prevention coalitions. *Am J Prev Med*. 2008;34(6):495–501.
- Feinberg ME, Gomez BJ, Puddy RW, Greenberg MT. Evaluation and community prevention coalitions: validation of an integrated Web-based/technical assistance consultant model. *Health Educ Behav*. 2008;35(1):9–21.
- Hawkins JD, Catalano RF Jr. *Communities That Care: Action for Drug Abuse Prevention*. San Francisco, CA: Jossey-Bass; 1992.
- Hawkins JD, Brown EC, Oesterle S, Arthur MW, Abbott RD, Catalano RF. Early effects of Communities That Care on targeted risks and initiation of delinquent behavior and substance use. *J Adolesc Health*. 2008;43(1):15–22.
- Lee REC. C. Neighborhood context and youth cardiovascular health behaviors. *Am J Public Health*. 2002;92(3):428–436.
- Sampson RJ, Squires GD, Zhou M. *How Neighborhoods Matter: The Value of Investing at the Local Level*. Washington, DC: American Sociological Association; 2002.
- Sampson RJ, Morenoff JD, Gannon-Rowley T. Assessing “neighborhood effects”: social processes and new directions in research. *Annu Rev Sociol*. 2002;28:443–478.
- Brooks-Gunn J, Duncan G, Aber JL. *Neighborhood Poverty I: Context and Consequences for Children*. New York, NY: Russell Sage; 1997.
- Hogan DP, Kitagawa EM. The impact of social status, family structure, and neighborhood on the fertility of Black adolescents. *Am J Sociol*. 1985;90(4):825–855.
- Leventhal T, Brooks-Gunn J. The neighborhoods they live in: the effects of neighborhood residence on child and adolescent outcomes. *Psychol Bull*. 2000;126(2):309–337.
- Bronfenbrenner U. Contexts of child rearing: problems and prospects. *Am Psychol*. 1979;34(10):844–850.
- Bronfenbrenner U. Ecology of the family as a context for human development: research perspectives. *Dev Psychol*. 1986;22(6):723–742.
- Wilson WJ. *The Truly Disadvantaged: The Inner City, the Underclass, and Public Policy*. Chicago, IL: University of Chicago Press; 1987.
- Jencks C, Mayer S. The social consequences of growing up in a poor neighborhood. In: Lynn LE, McGeary MFH, eds. *Inner-City Poverty in the United States*. Washington, DC: National Academies Press; 1990:111–186.
- Diez-Roux AV. Multilevel analysis in public health research. *Annu Rev Public Health*. 2000;21:171–192.
- Xue Y, Leventhal T, Brooks-Gunn J, Earls FJ. Neighborhood residence and mental health problems of 5- to 11-year-olds. *Arch Gen Psychiatry*. 2005;62(5):554–563.
- Boyce WF, Davies D, Gallepe O, Shelley D. Adolescent risk taking, neighborhood social capital, and health. *J Adolesc Health*. 2008;43(3):246–252.
- Lochner KA, Kawachi I, Brennan RT, Buka SL. Social capital and neighborhood mortality rates in Chicago. *Soc Sci Med*. 2003;56(8):1797–1805.
- Ennett ST, Flewelling RL, Lindrooth RC, Norton EC. School and neighborhood characteristics associated with school rates of alcohol, cigarette, and marijuana use. *J Health Soc Behav*. 1997;38(1):55–71.
- Ennett ST, Haws S. The school context of adolescent substance use. In: Scheier LM, ed. *Handbook of Drug Use Etiology: Theory, Methods, and Empirical Findings*. Washington, DC: American Psychological Association; 2010:443–459.
- Novak SP, Reardon SF, Raudenbush SW, Buka SL. Retail tobacco outlet density and youth cigarette smoking: a propensity-modeling approach. *Am J Public Health*. 2006;96(4):670–676.
- Newcomb MD, Felix-Ortiz M. Multiple protective and risk factors for drug use and abuse: cross-sectional and prospective findings. *J Pers Soc Psychol*. 1992;63(2):280–296.
- Andreas JB, Watson M. Etiology of adolescent substance use: neighborhood effects on initiation. *Eur Psychiatry*. 2010;25(Suppl 1):427.
- Allison KW, Crawford I, Leone PE, Trickett E, Perez-Febles A, Burton LM. Adolescent substance use: preliminary examinations of school and neighborhood context. *Am J Community Psychol*. 1999;27(2):111–141.
- Biglan A, Ary D, Wagenaar AC. The value of interrupted time-series experiments for community intervention research. *Prev Sci*. 2000;1(1):31–49.
- Rothman KJ, Greenland S, Lash TL. *Modern Epidemiology*. 3rd ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2008.
- Pickett KE, Pearl M. Multilevel analyses of neighbourhood socioeconomic context and health outcomes: a critical review. *J Epidemiol Community Health*. 2001;55(2):111–122.
- Scott KG, Mason CA, Chapman DA. The use of epidemiological methodology as a means of influencing public policy. *Child Dev*. 1999;70(5):1263–1272.
- Davis CH, MacKinnon DP, Schultz A, Sandler I. Cumulative risk and population attributable fraction in prevention. *J Clin Child Adolesc Psychol*. 2003;32(2):228–235.
- Poole C. On the origin of risk relativism. *Epidemiology*. 2010;21(1):3–9.
- Crowley DM, Jones DE, Greenberg MT, Feinberg ME, Spoth RL. Resource consumption of a dissemination model for prevention programs: the PROSPER Partnership Model. *J Adolesc Health*. In press.
- Spoth R, Redmond C, Clair S, Shin C, Greenberg M, Feinberg M. Preventing substance misuse through community-university partnerships and evidence-based interventions: PROSPER outcomes 4 1/2 years past baseline. *Am J Prev Med*. 2011;40(4):440–447.
- Chilenski SM, Greenberg MT, Feinberg ME. The community substance use environment: the development and predictive ability of a multi-method and multiple-reporter measure. *J Community Appl Soc Psychol*. 2010;20(1):57–71.
- Merlo J, Chaix B, Ohlsson H, et al. A brief conceptual tutorial of multilevel analysis in social epidemiology: using measures of clustering in multilevel logistic regression to investigate contextual phenomena. *J Epidemiol Community Health*. 2006;60(4):290–297.
- Müller R, Büttner P. A critical discussion of intraclass correlation coefficients. *Stat Med*. 1994;13(23–24):2465–2476.
- Murray DM, Rooney BL, Hannan PJ, et al. Intraclass correlation among common measures of adolescent smoking: estimates, correlates, and applications in smoking prevention studies. *Am J Epidemiol*. 1994;140(11):1038–1050.
- Dielman TE. *School-Based Research on the Prevention of Adolescent Alcohol Use and Misuse: Methodological Issues and Advances*. Hillsdale, NJ: Lawrence Erlbaum Associates; 1994.
- Murray DM, Alfano CM, Zbikowski SM, Padgett LS, Robinson LA, Klesges R. Intraclass correlation among measures related to cigarette use by adolescents: estimates from an urban and largely African American cohort. *Addict Behav*. 2002;27(4):509–527.
- Siddiqui O, Hedeker D, Flay BR, Hu FB. Intraclass correlation estimates in a school-based smoking prevention study. Outcome and mediating variables, by sex and ethnicity. *Am J Epidemiol*. 1996;144(4):425–433.
- Van Horn ML, Hawkins JD, Arthur MW, Catalano RF. Assessing community effects on adolescent substance use and delinquency. *J Community Psychol*. 2007;35(8):925–946.
- Cleveland MJ, Collins LM, Lanza ST, Greenberg MT, Feinberg ME. Does individual risk moderate the effect of contextual-level protective factors? A latent class analysis of substance. *J Prev Interv Community*. 2010;38(3):213–228.

53. Pennsylvania Commission on Crime and Delinquency. Pennsylvania Youth Survey. Available at: http://www.portal.state.pa.us/portal/server.pt/community/pennsylvania_youth_survey/5396. Accessed November 12, 2011.
54. Jones DE, Feinberg ME, Cleveland MJ, Rhoades BL. A multi-domain approach to understanding risk for underage drinking. *Am J Public Health*. In press.
55. Feinberg ME, Ridenour TA, Greenberg MT. Aggregating indices of risk and protection for adolescent behavior problems: the Communities That Care Youth Survey. *J Adolesc Health*. 2007;40(6):516–513.
56. Conger RD, Ge X, Elder GH Jr, Lorenz FO, Simons RL. Economic stress, coercive family process, and developmental problems of adolescents. *Child Dev*. 1994; 65(2 Spec No):541–561.
57. Feinberg ME, Ridenour TA, Greenberg MT. Aggregating indices of risk and protection for adolescent behavior problems: the Communities That Care Youth Survey. *J Adolesc Health*. 2007;40(6):506–513.
58. Cleveland MJ, Feinberg ME, Greenberg MT. Protective families in high- and low-risk environments: implications for adolescent substance use. *J Youth Adolesc*. 2010;39(2):114–126.
59. Pollard JA, Hawkins JD, Arthur MW. Risk and protection: are both necessary to understand diverse behavioral outcomes in adolescence? *Soc Work Res*. 1999;23(3):145–158.
60. Theokas C, Almerigi JB, Lerner RM, et al. Conceptualizing and modeling individual and ecological asset components of thriving in early adolescence. *J Early Adolesc*. 2005;25(1):113–143.
61. Moody J, Brynildsen WD, Osgood DW, Feinberg ME, Gest S. Popularity trajectories and substance use in early adolescence. *Soc Networks*. 2011;33(2): 101–112.
62. Kreager DA, Rulison K, Moody J. Delinquency and the structure of adolescent peer groups. *Criminology*. 2011;49(1):95–127.
63. Chuang YC, Li YS, Wu YH, Chao HJ. A multilevel analysis of neighborhood and individual effects on individual smoking and drinking in Taiwan. *BMC Public Health*. 2007;7:151.
64. Ridenour T, Feinberg ME. Using correlational analyses to improve prevention strategies based on survey data from youth. *Eval Program Plann*. 2007; 30(1):36–44.
65. Ennett ST, Foshee VA, Bauman KE, et al. The social ecology of adolescent alcohol misuse. *Child Dev*. 2008; 79(6):1777–1791.
66. Foshee VA, Ennett ST, Bauman KE, et al. A test of biosocial models of adolescent cigarette and alcohol involvement. *J Early Adolesc*. 2009;27(1):4–39.
67. Chuang YC, Ennett ST, Bauman KE, Foshee VA. Relationships of adolescents' perceptions of parental and peer behaviors with cigarette and alcohol use in different neighborhood contexts. *J Youth Adolesc*. 2009;38(10): 1388–1398.
68. Beyers JM, Loeber R, Wikström PO, Stouthamer-Loeber M. What predicts adolescent violence in better-off neighborhoods? *J Abnorm Child Psychol*. 2001;29(5): 369–381.
69. Kumar R, O'Malley PM, Johnston LD, Schulenberg JE, Bachman JG. Effects of school-level norms on student substance use. *Prev Sci*. 2002;3(2):105–124.
70. Swaim RC. Individual and school level effects of perceived harm, perceived availability and community size on marijuana use among 12th-grade students: a random effects model. *Prev Sci*. 2003;4(2):89–98.
71. Luthar SS, Cicchetti D, Becker B. The construct of resilience: a critical evaluation and guidelines for future work. *Child Dev*. 2000;71(3):543–562.
72. Plybon LE, Klierer W. Neighborhood types and externalizing behavior in urban school-age children: tests of direct, mediated, and moderated effects. *J Child Fam Stud*. 2001;10(4):419–437.
73. Brook JS, Nomura C, Cohen P. A network of influences on adolescent drug involvement: neighborhood, school, peer, and family. *Genet Soc Gen Psychol Monogr*. 1989;115(1):123–145.
74. Schonberg MA, Shaw DS. Do the predictors of child conduct problems vary by high- and low-levels of socioeconomic and neighborhood risk? *Clin Child Fam Psychol Rev*. 2007;10(2):101–136.
75. Butler S, Fearon P, Atkinson L, Parker K. Testing an interactive model of symptom severity in conduct disordered youth. *Crim Justice Behav*. 2007;34(6):721–738.
76. Simons RL, Simons LG, Burt CH, Brody GH, Cutrona C. Collective efficacy, authoritative parenting and delinquency: a longitudinal test of a model integrating community- and family-level processes. *Criminology*. 2005;43(4):989–1029.
77. Knoester C, Haynie DL. Community context, social integration into family, and youth violence. *J Marriage Fam*. 2005;67(3):767–780.
78. Hammack PL, Richards MH, Luo Z, Edlynn ES, Roy K. Social support factors as moderators of community violence exposure among inner-city African American young adolescents. *J Clin Child Adolesc Psychol*. 2004; 33(3):450–462.
79. Wickrama KAS, Bryant CM. Community context of social resources and adolescent mental health. *J Marriage Fam*. 2003;65(4):850–866.
80. Wickrama KAS, Noh S, Bryant CM. Racial differences in adolescent distress: differential effects of the family and community for Blacks and Whites. *J Community Psychol*. 2005;33(3):261–282.
81. Wight RG, Botticello AL, Aneshensel CS. Socioeconomic context, social support, and adolescent mental health: a multilevel investigation. *J Youth Adolesc*. 2006; 35(1):109–120.
82. Rose G. Sick individuals and sick populations. *Int J Epidemiol*. 1985;14(1):32–38.
83. Cleveland MJ, Feinberg ME, Bontempo DE, Greenberg MT. The role of risk and protective factors in substance use across adolescence. *J Adolesc Health*. 2008;43(2): 157–164.
84. Fagan AA, Van Horn ML, Hawkins JD, Arthur M. Using community and family risk and protective factors for community-based prevention planning. *J Community Psychol*. 2007;35(4):535–555.

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