

## Using Correlational Evidence to Select Youth for Prevention Programming

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Published online: 8 November 2007  
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**Abstract** In a period of increased accountability and reduced prevention resources, the effective targeting of those limited resources is critical. One way in which limited resources are focused is to identify and provide services to those most at risk for later substance use. Risk status, or propensity, is typically estimated from correlational evidence. Using meta-analytic techniques this paper examines the evidence that 29 of the 35 constructs specified by the CTC risk and protective factor model are related to alcohol, tobacco, or marijuana use. While these factors are generally demonstrated to be predictive of substance use, the strength of relation is modest. Ten factors show a significantly different strength of relation with tobacco than with alcohol and marijuana. Given the correlations observed and the rate of substance use in the population, providing only selective intervention services likely ignores the majority of those who will later use substances. Although selection improves the percentage of those receiving services who are likely to benefit from services, the evidence summarized in this study suggests selective interventions will omit many of those who will likely use substances. Given typical base and selection rates, smaller program effects on universal populations may keep a greater number of youth from becoming alcohol, tobacco, or marijuana involved. *Editors' Strategic Implications:* The data make a strong and provocative argument for primary prevention of youth substance abuse that should be heard by policymakers and service providers involved in strategic planning and appropriate deployment of resources.

**Keywords** Meta-analysis · Youth · Selection · Risk and protective factors · Alcohol · Tobacco · Marijuana · Intervention

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## Introduction

All programs designed to prevent and reduce youth substance use contain at their core a theory explaining why adolescents begin to use substances. Whether these foundational theories are derived from common sense, experience, or scientifically validated evidence (e.g., Durlak 1998; Hawkins et al. 1992; Mrazek and Haggerty 1994; National Institute on Drug Abuse 1997, 2003), the theories all suggest behaviors, attitudes, values, beliefs, experiences or other intra-personal or contextual characteristics that are considered instrumental predictors in the development of adolescent substance use. Once these key factors have been identified and isolated, programs aim the bulk of their energy and resources at reducing not substance use per se, but rather the rate, occurrence, or impact of the predictors believed to lead to substance use (Hansen and McNeal 1996).

Although all substance abuse prevention programs (other than interdiction and incapacitation) work according to this model, few have been as successfully distributed and are as explicit in specifying these risk and protective factors as “Communities that Care” (CTC) developed by Hawkins et al. (1992) and currently distributed through Rainer CTC in the United Kingdom (Rainer 2007) and through Substance Abuse and Mental Health Services Administration’s Prevention Platform in the United States (SAMHSA 2007). The CTC model with its 21 risk and 11 protective factors, has recently been obtained by Substance Abuse and Mental Health Services Administration’s Center for Substance Abuse Prevention (SAMHSA, CSAP) and its risk and protective factors approach to prevention has been adopted by many as a primary model for preventing the development or display of antisocial and drug using behavior.

The general strategy of CTC is to survey youth to develop community profiles that summarize a broad range of the risk and protective factors associated with undesired outcomes for youth. These needs assessment profiles allow communities to rationally allocate resources to those at the highest level of need based on an objective assessment of the rate of these risk and protective factors in a range of communities. By targeting resources to those communities whose members possess the highest levels of risk and lowest levels of protective factors, the CTC model promises an efficient and rationally defensible use of resources. This and other risk and protective factor frameworks have been adopted by federal agencies, by states, and by communities as a central component of prevention needs assessment (Arthur et al. 2002; National Institute on Drug Abuse 2003; Office of Juvenile Justice and Delinquency Prevention 1996; Office of National Drug Control Policy 1999; Substance Abuse and Mental Health Services Administration, Center for Substance Abuse Prevention 2007). Since this program uses risk and protective factors to select communities for prevention programming and because it has been widely implemented, CTC is a particularly attractive program on which to examine some of the assumptions of using risk and protective factors to target individuals and communities for prevention programming.

In this paper we use evidence from 101 independent prospective studies to assess (a) whether the predictors specified as instrumental in the CTC model are, in fact, associated with the development of adolescent alcohol, tobacco, and marijuana

(ATM) use and (b) whether they can reasonably be used to select youth for prevention programming on the basis of their likelihood to use or abuse substances at some future time. Thus we distinguish between the two separate, and somewhat independent, roles to which risk and protective factors can be put in predicting and preventing youth substance use. On the one hand, these factors may be the targeted intermediate or proximal outcome of an intervention. That is, if the risk or protective factor is linked by theory, is capable of being changed, and is correlated with later substance use, then it may be the factor that the prevention program attempts to alter (e.g., improved resistance skills). On the other hand, even if a factor is immutable it may be useful for identifying and selecting individuals or groups into preventive services (e.g., children of alcoholics). In this framing both mutable and immutable factors may be useful for selection, but only those factors capable of change are the appropriate targets of an intervention (Andrews and Bonta 1994).

## Unpacking Evidence for Prevention

### *Identifying Risk and Protective Factors*

Although unpacking these relations is central to development and prevention research, it is not straightforward. As every introductory text in statistics and every instructor in an introductory course in the social sciences will attest, “correlation doesn’t prove causality.” Only through experiments, in which one variable is manipulated and the effect of that manipulation on another variable is observed, can causality be inferred. In applied settings, however, manipulation of many of the antecedent behaviors, characteristics, or experiences that we associate with problem outcomes is typically not possible due to various practical and ethical constraints. We cannot randomly assign youth to school failure, ineffectual parents, or to displaying early substance use. Therefore, we estimate the potential contribution of risk and protective factors to later substance use using relational evidence typically summarized using the correlation coefficient. The presence of a significant correlation allows the ruling-in of potentially meaningful risk and protective factors, but that attribution relies entirely on a plausible theory of connectedness which we term mediation (Baron and Kenny 1986).

Since many prior conditions are highly intercorrelated in real world settings, our assignment of risk and protective factor status to a single variable is confounded by the presence of all the other variables that were not accounted for by our study and statistical design. In the absence of a perfect correlation (i.e.,  $r = -1$  or  $1$ ), any factor identified is neither sufficient nor necessary for producing later substance use. Since these relations are essentially unheard of in substance use prevention, understanding the relationship of antecedent conditions to later substance using behavior is not an exercise in uncovering those necessary and sufficient conditions which cause later substance use, but estimating the probabilistic conditions that are an “*insufficient* but *necessary* part of a condition which is itself *unnecessary* but *sufficient* for the result” (Mackie 1965, p. 245, italics in original). Mackie (1965) considers these INUS conditions as the basis for many causal attributions in real world settings. For

example, Mackie notes that we tend to attribute causality to the light switch when, in fact, a huge power and distribution infrastructure inside and out of the home is required for producing light. Each part of the system is necessary for producing light, no one part is sufficient, and we attribute causality to the part that forms a “difference-in-a-background” in our experience of the system. Substance use is likely the result of many INUS systems and we attribute causality to those antecedent behaviors, characteristics, or experiences that stand out against a background of development as increasing the probable display of later substance use.

In short, predicting substance use is a probabilistic exercise in which behaviors, characteristics, or experiences are statistically linked to the likelihood of later substance use. Since these factors (with the exception of availability) are neither necessary nor (with the exception of forced use, e.g., fetal exposure) sufficient for producing substance use, and because substance use is most likely multiply determined (i.e., there are multiple sufficient conditions capable of producing use), risk and protective factors are so designated because they stand out according to a theory of mediation.

### *Risk Versus Protective Factors*

When the relationship summarized by these indices is thought to be causal and direct, as in a risk factor relationship, the statistic describes the maximum potential change in rates or prevalence of substance use if an effective intervention were to produce changes in the targeted predictor (Hansen and McNeal 1996). If however, a protective factor relationship is summarized, and if that protective factor is not merely a recoded risk factor (e.g., commitment to school versus low school bonding), then the amount of change in substance use that an effective intervention on a protective factor might produce cannot be specified from simple correlational evidence. This is because a protective factor’s relationship with substance use is indirect, “with their effects apparent only by virtue of their interactions with the risk variable” (Rutter 1987, p. 319).

Estimating the causal contribution of protective factors to substance use requires controlling for the presence, absence, or magnitude of the risk factor with which the protective factor interacts. This can be done either through sample selection or through statistical modeling in which the independent contribution of the risk factor to substance use is estimated in the presence of the risk condition. This presents a much greater challenge to the effort to develop a scientific base for prevention. In this scenario, specifying the strength of a protective factor requires specifying its contextual condition(s). Since the strength of the protective relationship changes with the conditions of its application, the underlying population parameter of a protective factor with later substance use cannot be reliably estimated without also specifying the qualifying conditions.

This discussion of the rationale for risk factor research intends to make abundantly clear that correlations alone can never be used to establish causality; they are, instead, a necessary but insufficient part of the evidence that a causal

relationship between two factors may indeed exist. If the correlation is of sufficient magnitude and if it fits a mediating theory of the development of substance use, then we might believe it to be a risk factor. If it is “dynamic” and capable of change, then we may also believe it to be an appropriate target for prevention programming (Andrews and Bonta 1994). If, however, the correlation is modest, but we still believe that the factor is important in the development of substance use, then it is likely a protective factor, whose measured relationship with substance use is moderated by the presence, absence, or level of the risk factor with which it interacts. Because of the conditional nature of protective relations, this paper now turns its focus to the utility of bivariate risk factors for prevention programming.

### *Estimating the Underlying Strength of Relationship*

One of the principal ways risk and protective factors stand out in prevention is according to a measure of the strength of their relationship with later substance use. As might be expected, given the great variability among people and the methods and procedures used to study them, different research studies rarely agree exactly on the underlying correspondence of risk and protective factors with substance use. In some instances these differences are modest. However, when different studies provide vastly disparate estimates of the same relationship or when different studies each point to different risk factors as primary in the development of substance use, a common framework on which to evaluate the evidence must be developed.

Meta-analysis provides such a framework by standardizing the evidence from pertinent studies into a common metric, and then combining the evidence from multiple studies of a given relationship to derive a summary estimate of the potential contribution each risk factor makes to the development of substance use. The social sciences typically use correlations to specify the strength of this potential influence, although several other common statistics are also available for this purpose (e.g., the odds ratio, Relative Improvement Over Chance (RIO), chi-square, and others). These statistics all summarize the amount by which uncertainty about one variable is reduced by knowledge of a second.

Achieving stable, reliable estimates of the correlations between the CTC models’ identified risk factors and later substance use is not a simple matter. A single researcher who documents a relationship between a risk factor and an outcome does so using a single set of questions delivered in a particular manner to a select sample of individuals. When applied to other samples using different questions and data collection methods, the researcher’s results may not be reproducible. One study may find that sensation-seeking is more important than the community availability of substances for predicting individual use, while another may report that ties to delinquent peers is of paramount importance. Still another may declare that family attitudes favorable to substance use lay the foundation from which substance abuse arises. Which finding is to be believed? Or are they all credible?

One way of answering these questions is to statistically combine the evidence from several studies to determine the overall weight of evidence for each claim. The techniques and methods of statistically combining evidence on similar relationships from multiple studies are known collectively as meta-analysis, and the collective findings reported from this approach have several advantages over the findings yielded by any single study. First, because meta-analysis combines the evidence from several studies, researchers are able to examine the generalizability of relationships directly (Cordray 1990). Meta-analysis allows researchers to ask, for example, whether the early onset of problem behaviors and later substance use correlates consistently across multiple samples and methods. Second, because meta-analysis uses several studies to produce each mean estimate of a relationship, the method greatly reduces the likelihood that study findings may be attributed to sampling or methods errors (Hunter et al. 1982; Schmidt 1992). Third, as a method of summarizing evidence from multiple studies, meta-analysis provides a more organized, systematic, and comprehensive approach to literature review than is possible using traditional qualitative research techniques (Cooper 1984). For these reasons, and others, we used meta-analytic techniques to examine the evidence that the predictors specified in the CTC model are associated with the development of youth substance use.

## Method

Since general meta-analytic procedures have been adequately described elsewhere (e.g., Cooper and Hedges 1994; Lipsey and Wilson 2001), only those features specific to the current study will be specified here. The data for this report have been derived from a meta-analytic archive of studies that examined the natural development of individuals over two or more measurement periods. Studies were coded into the database if they contained evidence on the strength of relation between any variable and the likelihood of antisocial, delinquent, or violent behavior or alcohol, tobacco or marijuana use (e.g., Derzon 1996; Derzon and Lipsey 1999a, b; Lipsey and Derzon 1998). This archive was developed primarily through computerized searching of various bibliographic databases (e.g., ERIC, Social SciSearch<sup>®</sup>, PsychINFO<sup>®</sup>, NCJRS, DAI, and MedLine) and by canvassing the bibliographies of retrieved documents and review articles to identify other reports likely to contain eligible data.

Although all documents in the archive ( $N = 3,206$ ) are based on longitudinal prospective studies, only a subset of those documents contained quantitative data on the *direct* relationship of a predictor with current or later substance use (a bivariate correlation or data that could be mathematically transformed to a correlation). When an estimate of the relationship was identified (known in meta-analytic terms as an “effect size”), that estimate was abstracted into a meta-analytic database by trained post-doctoral coders. When necessary, protective factors reported in the literature were renamed and recoded so that all bivariate relations represent risk relationships (i.e., positive effect sizes reflect an increased level of risk and an increased level of substance use). For example, if religiosity was reported as the relationship of ‘high

religiosity' with substance use, then it was recorded as the risk factor 'low religiosity' by changing the direction of the correlation. All bivariate relations reported as protective relations readily fit existing risk relations by changing the sign and the language of the predictor. In addition to coding the effect size, more than 100 items related to the study sample's age and racial, ethnic, and gender composition were entered in the meta-analysis database along with items summarizing the measures, procedures, and methods used to obtain the estimate.

For this analysis of CTC risk factors, we reviewed this larger database to isolate those effect sizes for which the outcome measure was representative of either alcohol, tobacco, or marijuana use, misuse, or abuse, and which had predictor measures that were narrowly construed operationalizations of the risk factors identified by Hawkins and his colleagues in the CTC model (e.g., Hawkins et al. 1992, 1985, 1987). It is important to emphasize that these measures were not originally designed as operationalizations of the CTC risk factors, but were chosen on their face validity. Sorting of the estimates was done by the author, first selecting data on the substance use outcome, then sorting and resorting the verbatim descriptions of the precursor variables into categories representative of the CTC risk and protective factors. It should be noted that this sorting is iterative and performed blind to the effect size estimate.

Though the database contains 12,667 effect sizes linking risk factors with alcohol, tobacco, or marijuana use, misuse, or abuse, with these restrictions only 2,082 (16.4%) of these effect sizes were eligible for our current analyses. These effect sizes were reported in 190 documents summarizing the findings from 101 independent studies.<sup>1</sup> With 101 studies supplying an average of 20.6 effect sizes per study sample, it was imperative to eliminate statistical dependencies within the data set caused when the same samples contributed multiple effect sizes to the database. These dependencies were eliminated by averaging similar relationships within each study sample prior to synthesizing these weighted mean estimates across different study samples. These procedures reduced the 2,082 dependent effect sizes to 535 independent estimates of 29 of the 35 constructs specified by the CTC risk factor model. As with all averaging procedures presented in this report, these within sample aggregations used standard meta-analytic Z-transformations and inverse variance weighting procedures (Becker and Hedges 1989; Hunter and Schmidt 1990b; Rosenthal 1994). By weighting effect sizes, the meta-analytic technique gives greater credibility to estimates based on larger samples. However, to keep the evidence from largest samples from overwhelming the contribution of smaller ones, samples sizes larger than 699 were recoded to 700 when averaging across the different study samples. See Appendix A for a discussion of the meta-analytic methods used; a listing of the measures summarized and a bibliography of the reports contributing to this synthesis are available from the author upon request.

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<sup>1</sup> In this meta-analysis, the term "study" is used to describe each unique study-sample which is delivered common instruments, in shared settings. The study, in this context, refers to the sample, not the test.

## Results

### Description of the Data Used in this Analysis

Though our eligibility criteria allowed studies from all over the world, nearly three-quarters of the evidence came from studies conducted in the US (see Table 1). In addition, 10 were conducted in Canada, and 6 in Scandinavia. Great Britain, Australia, and New Zealand provided the remaining 10 studies. The samples providing these data were most frequently drawn from the general population (74%), whereas the remainder comprised populations judged at-risk for later substance use or criminal behavior. Most of these samples were predominantly white (62%) or included samples in which no single racial category comprised over 75% of the sample. Approximately 25% of the samples were more than 75% male, while another 4% contained primarily females. Relatively few studies included predominantly low socioeconomic status samples (11%) although it can be observed that these data are not well reported in most study reports. Seventy-five percent of the subjects were born between 1960 and 1980. Most of the predictor variables were measured when the subjects were in their teens and 80% of the substance use data were collected after 14 years of age.

At the meta-analytic level, sample characteristics have not tended to systematically influence effect sizes (for example earlier born samples do not report stronger correspondences than later born samples) the generalizability of these relationships may not be assured based on the available evidence (c.f., Beauvais et al. 1996; Moon et al. 1999). As a form of systematic review, meta-analysis is restricted to the evidence reported. Because it is unusual for studies to report separate analyses for sub-populations, the ability of meta-analysis to detect substantive differences that may exist is compromised. Moreover, even if risk-outcome relations are constant across sample characteristics, the risk and protective factors themselves are likely not equally distributed across subpopulations and this, as we shall see, has implications for prediction.

Although all the data came from longitudinal studies, fully 60% of the effect sizes were based on data collected in the same wave of the study. In other words, although all the data coded for this synthesis came from longitudinal prospective research, in which researchers followed one or more cohorts over time, only 40% of the effect sizes used data in which the risk or protective factor was measured at some point prior to the substance using outcome being measured. No systematic differences in effect size strength were noted between cross-sectional and prospective estimates. Selecting just the subset of prospective effect sizes, nearly one-third claimed no sample attrition between measurement waves, while another 36% showed less than 20% attrition. The bulk of the data for both predictors and outcomes was collected using survey instruments, although nearly one-quarter of the outcome data on substance use was collected in interview settings. Single items from surveys or interviews were used for 40% of predictor and 53% of outcome measures. An additional 36% of the predictor and 26% of the outcome measures were generated using standardized instruments or factors derived empirically from sample data.

**Table 1** Characteristics of the effect sizes used in this meta-analysis: Hawkins risk categories predicting current and later alcohol, tobacco and marijuana use

	<i>N</i> ( <i>p</i> )
<i>General</i>	
Country in which study was conducted <sup>a</sup>	
United States	73 (.72)
Canada	10 (.10)
Great Britain	3 (.03)
Scandinavia	6 (.06)
Other Country	7 (.07)
<i>Subject features</i>	
Population sampled <sup>a</sup>	
General	75 (.74)
At-risk	8 (.08)
Criminal or treatment	7 (.07)
General & at-risk	3 (.03)
General & drug-using	2 (.02)
General & criminal or treatment	4 (.04)
Ethnicity <sup>a</sup>	
Predominantly white	63 (.62)
Predominantly minority	8 (.08)
None dominant	31 (.30)
Gender <sup>a</sup>	
Predominantly male	25 (.25)
Predominantly female	4 (.04)
None dominant	73 (.72)
Estimated socioeconomic status <sup>a</sup>	
Low	11 (.11)
Working	51 (.50)
Middle and above	40 (.39)
Mean age when predictor was measured <sup>b</sup>	
<8 years old	72 (.03)
8–10.99 years old	43 (.02)
11–14.99 years old	584 (.28)
15–18.99 years old	979 (.47)
19–21.99 years old	169 (.08)
22+ years old	243 (.12)
Mean age when outcome was measured <sup>b</sup>	
11–14.99 years old	377 (.18)
15–18.99 years old	1,067 (.51)
19–21.99 years old	196 (.09)
22+ years old	450 (.22)

**Table 1** continued

	<i>N</i> ( <i>p</i> )
<i>Method features</i>	
Mean number of subjects <sup>a</sup>	
0–199	21 (.21)
200–499	28 (.28)
500–1500	36 (.35)
>1500	17 (.17)
Year of birth <sup>a</sup>	
<1950	25 (.25)
1950–1959	27 (.27)
1960–1969	30 (.29)
1970–1979	19 (.19)
Mean year predictor was measured <sup>b</sup>	
<1970	388 (.19)
1970–1979.99	835 (.40)
1980–1984.99	380 (.18)
>1984	487 (.23)
Mean year outcome was measured <sup>b</sup>	
<1970	105 (.05)
1970–1979.99	1034 (.50)
1980–1984.99	413 (.20)
>1984	538 (.26)
Mean interval between measures <sup>b</sup>	
None	1256 (.60)
.01–1.99	196 (.09)
2–4.99	293 (.14)
5–9.99	148 (.07)
10+	189 (.09)
Study attrition for prospective effect sizes <sup>b</sup>	
None	128 (.31)
0.01–9.99%	63 (.15)
10.00–19.99%	88 (.21)
20.00–30.00%	61 (.15)
>30.00%	75 (.18)
<i>Measurement features</i>	
Metric used in reporting <sup>b</sup>	
Correlation	1344 (.64)
2 by 2	317 (.15)
K by J	208 (.10)
<i>F</i> or <i>t</i> -test	22 (.01)
Chi square	15 (.01)
Imputed	47 (.02)

**Table 1** continued

	<i>N</i> ( <i>p</i> )
Source of predictor information	
Subject	1699 (.81)
Other source	391 (.19)
Fit of predictor measure to construct <sup>b</sup>	
Fair	79 (.04)
Good	391 (.19)
Very good	1,601 (.77)
Excellent	19 (.01)
Fit of outcome measure to construct <sup>b</sup>	
Good	26 (.01)
Very good	2,014 (.96)
Excellent	50 (.02)
How predictor data were recorded <sup>b</sup>	
Survey	1,439 (.69)
Interview	400 (.19)
Observation	109 (.05)
Other	142 (.07)
How outcome data were recorded <sup>b</sup>	
Survey	1,410 (.68)
Interview	480 (.23)
Other / Cannot tell	200 (.10)
Reliability indicator of predictor <sup>b</sup>	
Single item	829 (.40)
Multiple items	501 (.24)
Factor score	347 (.17)
Standardized instrument	398 (.19)
Reliability indicator of outcome <sup>b</sup>	
Single item	1,105 (.53)
Multiple items	426 (.20)
Factor score	126 (.06)
Standardized instrument	426 (.20)

*Note:* *N* = number of effect sizes. *p* = proportion of effect sizes

<sup>a</sup> Data reflect number and proportion of effect sizes using one aggregated effect size per study sample (*n* = 101)

<sup>b</sup> Data reflect number and proportion of effect sizes prior to aggregation (*n* = 2,090)

It is disappointing that the majority of reported estimates from the longitudinal prospective studies identified for this meta-analysis are cross-sectional. Given the expense and difficulty in conducting these studies, one might expect better use of the predictive data. Mitigating this disappointment, perhaps, is the observation that in these data the strength of relationship was not moderated by timing of measurement. Also disappointing is the reliance on single items to estimate both

predictors and outcomes. Many of the items are drawn from scales, and, in contrast to many of the other sample, study, and report characteristics that did not influence the effect size observed, correlations based on single items were systematically larger than those based on standardized measures.

### Evidence for a Relationship between CTC Predictors and Substance use

The data collected for our larger study of the predictors of alcohol, tobacco, and marijuana use supported analyses of 71 of the 105 possible bivariate relationships between 35 CTC risk factors and three substance use outcomes (see Table 2). All but nine of the estimates are based on more than one study sample, 42% are based on four or more studies, and fully 13 of the estimates are based on 10 or more independent study samples (18.3%). Two of the findings, the correspondence of few rewards for conventional involvement with marijuana and interaction with antisocial peers with alcohol were based on very small samples (45 and 47 respondents respectively), but the majority (77.5%) were based on over 1,000 respondents. Nine out of the top 10 most studied risk and protective factors predicted alcohol use with the top five being: antisocial behavior, academic failure, general social skills, religiosity, and depression.

At the  $P < .05$  level, nearly three-quarters of the mean correlations show significant between estimate heterogeneity as tested by the homogeneity Q-statistic (Hedges and Olken 1985). The Q-statistic tests the assumption that the effect sizes being pooled likely represent a single underlying population parameter. However, many of these study sample sizes are very large and the standard error of each study tends to be small. Because of this, the homogeneity Q may be rejected even when estimates differ only modestly. From a meta-analytic perspective, our confidence in the veracity of an estimate comes from Q, the number of studies, and the number of observations summarized by the mean estimate. Each of these parameters is provided in Table 2. In general, greater confidence in the obtained estimate is judged by the number of studies, the overall sample size, and a presence of a non-significant Q ( $P > .05$ ).

Figure 1 provides an easier forum for comparing and examining the strength and consistency of risk and protective factors for predicting alcohol, tobacco, and marijuana use. Figure 1 shows the mean correlation of 29 risk and protective factors with alcohol, 21 with tobacco use, and another 21 with marijuana use. These mean correlations are sorted according to the comparative strength of their relationships with alcohol use (shown as black circles). Each predictor's relationship with tobacco and marijuana use is shown by white diamonds and light gray triangles respectively. The error bars associated with the synthesized mean of the correlations represent the 95% confidence interval for each relationship. These confidence intervals also show whether the estimate is significant (if the error bars include zero (0) they indicate failure to reject the null hypothesis) and whether estimates differ significantly from each other (if the error bars do not overlap, then the estimates are significantly different at the  $P = .05$  level).

**Table 2** Aggregated effect sizes for Hawkins risk factors predicting current and later alcohol, tobacco, and marijuana use with number of original effect sizes and sample size

Predictor category	Mean ES <sup>a</sup>	95 % Confidence Interval		Q	P	N <sup>b</sup>	O <sup>c</sup>	n <sup>d</sup>
		Lower	Upper					
<b>Low perceived risks of drug use</b>								
Alcohol	.160	.121	.199	34.51	.00	7	33	4,801
Tobacco	.041	.000	.081	128.27	.00	5	27	4,020
Marijuana	.244	.189	.298	8.22	.02	3	7	2,598
<b>Positive attitudes towards substance use</b>								
Alcohol	.326	.302	.351	175.41	.00	19	203	6,663
Tobacco	.421	.388	.454	193.74	.00	5	19	4,067
Marijuana	.287	.244	.329	6.27	.28	6	19	3,298
<b>High religiosity</b>								
Alcohol	.202	.182	.221	277.59	.00	23	80	35,728
Tobacco	.143	.100	.186	25.65	.00	4	8	18,575
Marijuana	.145	.104	.187	11.20	.05	6	18	8,010
<b>Low belief in the moral order</b>								
Alcohol	.205	.162	.247	32.04	.00	6	19	9,050
Tobacco	-.110	-.196	-.023	–	–	1	2	705
Marijuana	.230	.185	.274	102.70	.00	8	18	4,329
<b>High rebelliousness</b>								
Alcohol	.184	.142	.224	16.29	.04	9	20	3,169
Tobacco	.067	.005	.129	2.51	.11	2	3	1,955
Marijuana	.213	.172	.252	40.00	.00	9	32	2,819
<b>Early initiation of problem behavior</b>								
Alcohol	.315	.282	.347	214.36	.00	7	22	5,038
<b>More interaction with antisocial peers</b>								
Alcohol	.066	-.224	.346	–	–	1	2	47
<b>More ties with delinquent peers</b>								
Alcohol	.255	.206	.302	2.17	.71	5	23	1,574
<b>High antisocial behavior</b>								
Alcohol	.252	.237	.267	285.12	.00	49	370	74,441
Tobacco	.244	.210	.277	42.25	.00	11	44	5,486
Marijuana	.199	.177	.221	73.03	.00	22	113	11,519
<b>Low general social skills</b>								
Alcohol	-.007	-.027	.014	174.71	.00	26	63	64,333
Tobacco	-.080	-.129	-.031	5.99	.20	5	15	1,791
Marijuana	.040	.002	.079	41.57	.00	9	85	6,242
<b>High depression</b>								
Alcohol	.093	.068	.117	145.13	.00	22	76	62,572
Tobacco	-.014	-.061	.034	5.23	.16	4	4	7,139
Marijuana	.103	.056	.150	20.10	.00	6	13	7,053

**Table 2** continued

Predictor category	Mean ES <sup>a</sup>	95 % Confidence Interval		Q	P	N <sup>b</sup>	O <sup>c</sup>	n <sup>d</sup>
		Lower	Upper					
<i>High sensation seeking</i>								
Alcohol	.328	.290	.365	93.18	.00	9	36	2,448
Tobacco	.198	.112	.281	–	–	1	2	710
<i>High impulsiveness</i>								
Alcohol	.243	.190	.295	2.54	.47	4	13	1,424
Marijuana	.151	.080	.221	1.98	.58	4	16	756
<i>Family constructs</i>								
<i>Poor family management</i>								
Alcohol	.066	.030	.102	96.15	.00	14	43	4,726
Tobacco	.344	.211	.465	5.84	.02	2	2	190
Marijuana	.082	.025	.139	11.34	.01	4	12	3,392
<i>Poor discipline</i>								
Alcohol	.060	.007	.113	21.59	.00	6	30	2,851
Tobacco	.161	.020	.300	0.12	.73	2	2	190
Marijuana	.019	–.042	.080	6.09	.05	3	4	2,897
<i>Low attachment</i>								
Alcohol	.131	.103	.158	70.04	.00	20	81	10,111
Tobacco	.112	.055	.168	12.03	.02	5	12	1,191
Marijuana	.133	.096	.169	7.60	.27	7	15	7,711
<i>Few opportunities for positive family involvement</i>								
Alcohol	.223	.146	.298	9.63	.05	5	28	611
Tobacco	.001	–.100	.102	3.02	.08	2	4	380
<i>Few rewards for conventional involvement</i>								
Alcohol	.068	–.006	.141	3.59	.31	4	28	710
Marijuana	.061	–.250	.361	–	–	1	2	45
<i>Family conflict</i>								
Alcohol	.063	.020	.105	14.15	.08	9	17	2,624
Tobacco	.150	.038	.258	–	–	1	1	304
Marijuana	.153	.069	.234	2.00	.16	2	3	920
<i>Family history of antisocial behavior</i>								
Alcohol	.086	.041	.131	20.07	.00	7	17	2,444
Tobacco	.159	.016	.295	0.06	.81	2	4	190
Marijuana	.329	.216	.433	0.21	.65	2	3	264
<i>Family attitudes favorable to substance use</i>								
Alcohol	.064	.030	.098	19.94	.01	9	52	7,291
Tobacco	.260	.221	.297	50.78	.00	7	19	3,593
Marijuana	.016	–.047	.078	1.44	.70	4	20	2,011
<i>School constructs</i>								
<i>Academic failure</i>								
Alcohol	.176	.160	.192	1,570.41	.00	39	87	40,589

**Table 2** continued

Predictor category	Mean ES <sup>a</sup>	95 % Confidence Interval		Q	P	N <sup>b</sup>	O <sup>c</sup>	n <sup>d</sup>
		Lower	Upper					
Tobacco	.117	.086	.147	33.09	.00	10	24	32,510
Marijuana	.076	.046	.105	53.42	.00	12	20	16,653
Low commitment to school								
Alcohol	.108	.084	.131	72.04	.00	20	56	13,609
Tobacco	.194	.139	.248	29.26	.00	3	4	2,146
Marijuana	.23	.19	.270	44.11	.00	9	20	3,655
Few opportunities for positive school involvement								
Alcohol	.031	-.020	.081	26.46	.00	4	6	2,788
Tobacco	-.039	-.126	.049	–	–	1	1	1,415
<i>Community constructs</i>								
Community and societal norms that condone substance use								
Alcohol	.236	.170	.301	0.05	.98	3	7	1,662
High level of transitions and mobility								
Alcohol	.050	-.010	.111	8.60	.01	3	6	2,104
Tobacco	.035	-.032	.101	6.18	.05	3	5	1,936
Marijuana	.060	-.028	.147	–	–	1	1	875
Community disorganization								
Alcohol	.159	.098	.219	0.69	.41	2	3	2,163
Marijuana	-.076	-.163	.012	–	–	1	2	1,533
Perceived availability of alcohol, tobacco, illicit drugs, and guns								
Alcohol	.230	.170	.288	29.50	.00	2	20	23,848
Tobacco	.489	.440	.535	10.29	.00	2	4	1,571
Marijuana	.443	.397	.487	86.95	.00	3	11	2,666
Few opportunities for conventional involvement								
Alcohol	.365	.215	.498	–	–	1	1	146
Total						535	2,082 <sup>e</sup>	174,934

<sup>a</sup> Weighted mean of the Winsorized aggregated effect sizes in each predictor category. All factors were coded or recoded to represent risk factors

<sup>b</sup> Number of aggregated effect sizes

<sup>c</sup> Number of original effect sizes that went into aggregated effect sizes for each factor category

<sup>d</sup> Total sample size represented by all aggregate effect sizes in each factor category

<sup>e</sup> Eight effect sizes were deemed ineligible dropped from further analysis

As can be seen in the mean estimates and confidence intervals presented in Fig. 1, there is considerable variation in the ability of CTC risk factors to predict later substance use. In fact, two risk factors (general social skills and belief in the moral order) show significant negative relationships with current and later tobacco use. In other words, stronger social skills and a stronger belief in the moral order were associated with *increased* tobacco use. Fifteen of the 71 relationships and four of the risk factors (opportunities for positive school involvement, high levels of transitions and mobility, interaction with antisocial peers, and few rewards for

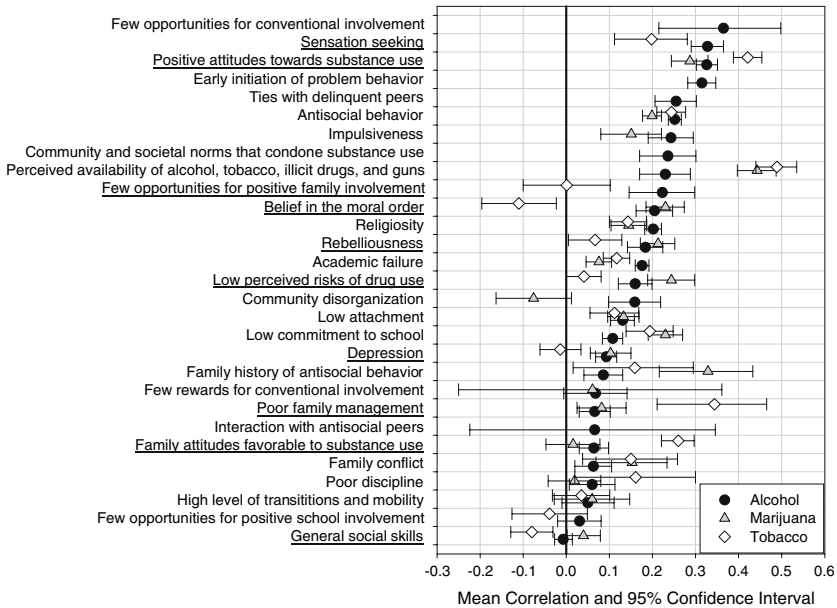
conventional involvement) were not significantly different from zero, indicating that it is unlikely that they contribute to substance use. Nevertheless, most of the risk factors specified by the CTC model showed positive and significant relationships with the substance use outcomes, and several of them were of sufficient magnitude to be considered useful and appropriate targets for prevention programming.

That different risk factors manifest variations in predictive strength is to be expected. More interesting from an epidemiological perspective, perhaps, is the variation in mean effect size for many of the predictors according to the specific outcome predicted. For example, perceived availability of licit and illicit drugs and guns is a very strong predictor of tobacco and marijuana use, but is only modestly associated with alcohol use. Similarly, positive attitudes toward substance use, poor family management, and family attitudes favorable toward substance use are each significantly stronger predictors of tobacco use than they are of either alcohol or marijuana use. In fact, fully 10 of the predictors were significantly stronger (or weaker) in predicting tobacco use than in predicting either alcohol or marijuana use. Availability of licit and illicit drugs and guns and low commitment to school, in contrast, showed a significantly weaker relationship with alcohol than with tobacco or marijuana. Community disorganization was, however, a much stronger predictor of alcohol use than marijuana use. Taken together, these findings suggest a similar developmental path for alcohol and marijuana use, while tobacco use appears to follow a different development path.

In summary, all but 16 of the 71 relations tested using risk and protective factors specified by the CTC model were in the expected direction and predictive of current and later alcohol, tobacco, or marijuana use. However, the evidence for these relations is thinner and more variable than might be expected given the nearly ubiquitous acknowledgment of the importance of these factors in the development of substance use. Due primarily to limitations of the database from which we were drawing estimates, we were unable to document the role of six of the 35 risk and protective factors identified by the CTC model. These missing items were primarily community factors that are rarely examined in studies using multi-wave panel designs. More notable perhaps, are the observations that nine of the estimates are based on evidence from a single trial with another ten based on two trials. Two of the estimates had fewer than 50 persons providing evidence; another seven estimates are based on evidence from fewer than 500. Finally, it is worth observing that although it is clear that the majority of factors specified by the CTC model indeed are significant and positive predictors of substance use, the overall strength of many of these mean correlations is somewhat underwhelming.

### Using Correlations to Select Individuals

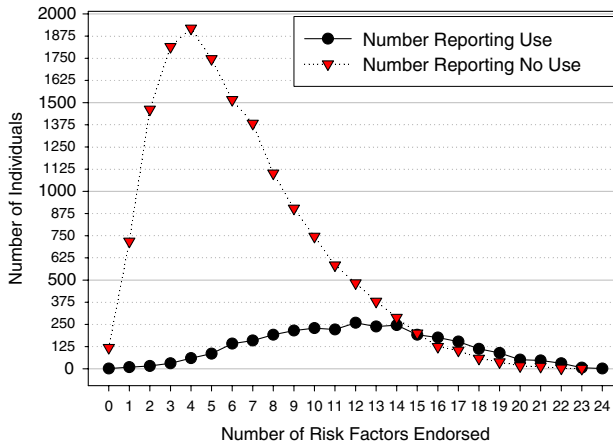
“Significant” and “positive,” however, are statistical terms that tell us little about how to use risk factors to identify individuals as “at risk” and thus potentially eligible for selective intervention. The relationships displayed in Fig. 1 show the maximum potential causal contribution of each predictor to each outcome. These values direct our attention to those categories effective intervention might hope to



**Fig. 1** Synthesized mean correlations of 29 CTC risk factors with substance use sorted by strength of relationship with alcohol use with underlines indicating which predictors differed significantly for tobacco use

influence, and through that influence reduce substance use. Fundamentally different in purpose is the task of identifying that subset of individuals at risk for substance use. Substance use is probabilistic because not all those at risk will use substances and not all those not at risk will avoid use. For example, using data on 12- to 17-year-old youths from the 1999 National Household Survey of Drug Abuse (the most current year such a diversity of risk and protective factor data were collected), we identified 24 risk factor variables associated with substance use (e.g., approval of drug use and antisocial or delinquent behaviors; U.S. Dept. of Health and Human Services 1999). For continuous-variable factors and factors with at least three response categories, we standardized the factors by converting them to z-scores. Respondents who scored in the upper 33% of a given risk factor (or lower 33%, depending on the direction of the item) were considered “at-risk” on that particular factor. For dichotomous-variable factors, we considered a positive response to be “at-risk.” We then calculated the total number of “at-risk” variables each respondent endorsed (i.e., one person may be at-risk on three factors while another person may be at-risk on 10 factors).

We then dichotomized the sample on 30-day alcohol use and plotted the cumulative probability of having a particular number of risk factors given 30-day alcohol use and non-use for males and females (see Fig. 2). This graph illustrates the distribution of risk among users and non-users of alcohol. The average number of at-risk identifications for youth who reported using alcohol during the past 30-days was 12.0, while the average for non-users was 6.3. The non-user



**Fig. 2** Number of youth respondents using and not using alcohol at different levels of risk, data from the NHSDA, 1999

distribution is noticeably skewed with most non-using respondents being positively identified on five or fewer risk factors (out of the 24 measured).

Though there are many interesting lessons and inferences to be drawn from Fig. 2 (and it can be noted that this distribution remained fundamentally similar across gender and race for both tobacco and marijuana use and avoidance), the key point is this: it isn't until respondents are positively identified as at-risk on more than 14 risk factors that the number of users exceeds the number of non-users. Until that point, any selection would include more likely non-users of alcohol than it would users. It might also be observed that making 14 risk factors the cut-off point for eligibility to intervention would omit 71% of reported users from beneficial intervention.

Therefore, because use is probabilistic and our prediction imperfect, when selecting specific individuals or groups on whom to intervene, we should focus on the extent to which the selection process identifies all who might benefit from intervention and omits those for whom the intervention is unnecessary. If the intervention itself is costly, either to those identified or to those providing the intervention we might also be interested in estimating the proportion of those identified for intervention who actually later use substances. On the other hand, if the intervention is beneficial, we might also be concerned with the proportion of those not identified who might have benefited if offered intervention.

These eligibility and selection issues bear on the conditional relationships between the selection variable and actual substance use. Because selection and substance use involve binary classification, in which risk status is dichotomized so that individuals may be placed either above or below some criterion value of eligibility for intervention, it involves different considerations than those germane to assessing the overall strength of the predictor-use relationship. Correlation coefficients assess an entire relationship between variables, not the distinct contingent relationships between the dichotomies that are or can be defined by risk, protective, and substance using variables (Derzon and Lipsey 1999b).

		OUTCOME		
		STATUS		
		Outcome Present	Outcome Absent	
PREDICTOR	Predictor Present	(A) True Positives	(B) False Positives	(A + B) At Risk
	Predictor Absent	(C) False Negatives	(D) True Negatives	(C + D) Not At Risk
		(A + C) Abuse	(B + D) No Abuse	(A + B + C + D) Total

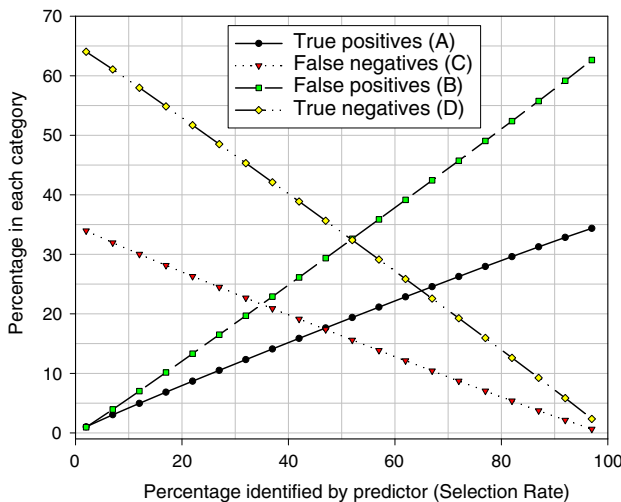
Fig. 3 The 2 × 2 or foursquare contingency table

When predictors and outcomes are each dichotomized, the data necessary to describe those contingent relationships can be fully presented in a 2 × 2 contingency table (see Fig. 3). The cells of this table and their interrelations provide the information necessary for estimating the utility of each risk factor for selecting people into a given intervention. Although the contingency table may be new to some, the essential ideas behind it are familiar. As an example let us imagine we are selecting truant students into intervention to improve school performance as measured by grade point average (GPA). In this example, we dichotomize the predictor into students who are truant at least once, and those who are not. We further dichotomize the outcome GPA to “C” and above as acceptable while “below C” averages are unacceptable. In this example, the proportion of students who are identified as truant is known as the “selection rate.” The proportion of students who are failing is called the “base rate.” Students who are truant and failing are considered “true positives” while truants who are passing are “false positives.” Students who are not truant but who are still failing are “false negatives” while not-truant passing students are “true negatives.” Applied to a population of students, each student fits into one and only one of the cells in Fig. 3. Once all students have been placed in the 2 × 2 table, we can begin to estimate the potential error associated with using truancy as a predictor of school failure.

The essential point here is that when the relationship of a risk factor with an outcome is less than perfect, that is, described by a correlation between -1.0 and 1.0, it is likely that some of the people positively identified by a predictor will not use substances, while some percentage not identified will. Using the observed correlation and the marginal base and selection rates we can use algebra to solve a simultaneous equation to estimate these false positive and false negative error rates directly. These estimated values can then be graphically presented and their precision as a basis for case selection assessed.

To apply this logic to the correlations identified by the CTC model and to estimate what the categories identified by that model might mean for identifying youth at risk for developing substance use, we turned to the Monitoring the Future (MTF) study. MTF is an annual survey of 8th, 10th, and 12th grade students that uses a representative sample of the nation’s high schools and is generally acknowledged to provide the best estimate of youth substance use in the United States. According to the 2006 survey, 36.5% of the nation’s seniors reported using marijuana in the past year, 21.6% said they had smoked cigarettes in the past 30 days, and 30% had drunk five or more drinks in a row on at least one occasion in the prior 30 days (Johnston et al. 2006). To examine the implications of using the CTC risk factors as tools for identifying individuals at risk for the outcomes listed above, we assumed an average outcome base rate of 35% and solved a series of simultaneous equations based on the .05 and .35 correlations that characterized the lower and upper effect sizes from our data. We used the values generated by this process to build Figs. 4 and 5. In these figures the proportion of a hypothetical population that would be expected to in each cell a  $2 \times 2$  table is shown as a function of a changing selection rate, a given correlation, and a fixed base rate. Since these proportions are read vertically, at any given level of selection the proportions total 100% and the points on each line represent the proportion of individuals in each cell of the table at a specific selection rate.

What Fig. 4 shows is that given a 35% substance use rate and a correlation of .05, our ability to accurately predict who is likely to use substances is far from perfect. For example, assume 20 out of 100 people are selected as at risk by a predictor (the “X” axis). Of those 20, only eight are true positives and are in fact likely to use

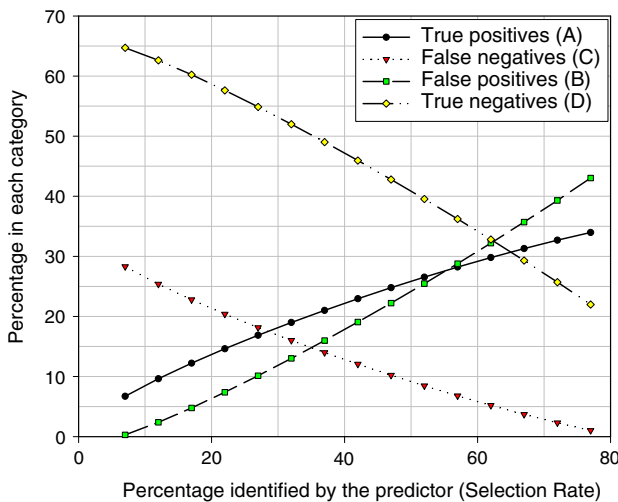


**Fig. 4** Selection accuracy given an underlying correlation of .05 and 35% of the population exhibiting the outcome behavior

substances (Fig. 4). Twelve of the 20 (60%) are not likely to misuse substances even when we assume the predictor is significant and positive. Furthermore, given the .05 correlation and the 35% substance use rate, at every level of selection in this figure the majority of those identified as at risk are not, probabilistically speaking, likely to use substances (i.e., they are false positives).

Noteworthy also from the standpoint of selective prevention is the observation that given a .05 correlation, selecting 20% of the population into intervention omits an additional 27% of the population who are likely to use substances and who might possibly have benefited from intervention (false negatives). These are individuals who were not identified as at risk by a predictor but who use, or are likely to use, substances. Put in another way, under the conditions specified in this example, for every person accurately identified as at risk for the substance use outcomes specified above, three persons equally likely to engage in substance use and equally likely to benefit from intervention will not be identified as such. Only when 47% of the population is identified by a predictor does the percentage of false negatives equal the percentage of true positives.

In contrast, if a risk factor correlates at the .35 level and 20% of the population is again identified as at risk, then nearly 70% are of those so identified are in fact likely to engage in the substance using outcomes described above. This represents considerable improvement over the 40% true positive identification rate when the correlation between predictor and substance use was .05. In contrast with the data shown in Fig. 4, over half of those identified in Fig. 5 for selective prevention are, in fact, likely to use substances (until selection includes at least 55% of the population). Moreover, fewer than half of those missed by selection are likely to use substances once selection identifies 27% or more of the population.



**Fig. 5** Selection accuracy given an underlying correlation of .35 and 35% of the population exhibiting the outcome behavior

## Conclusion

The evidence from longitudinal research suggests that the risk factors identified by the CTC model are, on the whole, likely to predict substance use. However, the degree of association with use is generally modest. Nearly one-third of the mean correlations are between zero and .10 and another 50% are between .10 and .30. Two mean correlations were significant and negatively associated with the substance use, suggesting a relationship with use that is the opposite of that expected from theory. Fourteen correlations were not significantly different from zero, suggesting that these items may be unrelated to substance use. Exceptions to these general findings are the constructs “Availability of Licit and Illicit Drugs or Guns,” which strongly predicts marijuana and tobacco use ( $r = .44$  and  $.49$  respectively), “Positive Attitudes Toward Substance Use,” which strongly predicts tobacco, alcohol, and, to a lesser degree, marijuana use ( $r = .42$ ,  $.33$ , and  $.29$  respectively), and “Early Initiation of Problem Behavior,” which predicts alcohol use ( $r = .32$ ).

Of perhaps even greater import for prevention science is the evidence that out of the 27 risk and protective factors examined, fully 10 showed significantly different strengths of association with tobacco use when compared with alcohol or marijuana use. One implication of this finding is that it may be unrealistic to expect an intervention on one of these 10 factors to be universally effective in reducing different forms of substance use. A slightly broader implication is that tobacco use may have a significantly different etiology than alcohol or marijuana use, an implication that is supported by the vastly different settings, occasions, and rituals surrounding the use of these substances. Tobacco use is ubiquitous and occurs in public settings. Although some smoking behavior is ritualized, much is mere habit. Alcohol and marijuana use, on the other hand enjoy significant privacy, occurring mainly in specific settings, and often accompanied by significant ritual. Whatever the locus of the difference, these findings suggest that reducing tobacco use may require targeting different factors than those targeted by programs designed to reduce marijuana or alcohol use.

Given the modest strength of relationship of many of these risk and protective factors, their observed distribution in a normal youth population, and the error terms associated with selection given the necessary relations imposed by the base and selection rates, it is clear that prevention must maintain a strong commitment to universal intervention on the basis of mere probability. Selection, given the strength of relation observed in many of these risk and protective factors, is unlikely to provide definitive identification of all those who might benefit from prevention programming while it includes some non-trivial percentage of those unlikely to engage in later substance use. This is not to suggest that there isn't a role for selective intervention,<sup>2</sup> but these findings strongly suggest that risk-focused intervention cannot be the only prevention service provided.

Our findings from the National Household Survey of Drug Use suggest that atheoretical totaling of risk factors may have limited utility for improving selection.

<sup>2</sup> In fact, because selection increases the percentage of those likely to exhibit an outcome, purposive selection prior to randomization is an excellent strategy for increasing research sensitivity to detect intervention effects.

Whether combining risk factors on a theory-driven basis would yield greater predictive ability depends, of course, on the inter-correlations among the items. If highly inter-correlated, they are functionally redundant for predictive purposes and their predictive ability combined is no greater than that of the best of the variables alone. However, if two or more risk variables are individually predictive but not highly inter-correlated, their combined predictive strength will be greater than that of any one alone.

Unfortunately, as prediction accuracy goes up in this probabilistic world of prevention, so does the percentage of false negative identifications. That is, increasing predictive accuracy increases the percentage of those who are positively identified who are likely to later use substances. But in making the eligibility criteria more stringent, decreasing the selection rate, fewer are eligible for prevention services and a larger percentage of the population is not exposed to potentially beneficial prevention services. Even with a fairly strong correspondence of  $r = .35$ , it was not until selection reached about 35% that the population percentage of true positives exceeded the population percentage of false negatives. It can be noted that this probabilistic effect is equally profound if the outcome base rate is reduced by attempting to predict the smaller percentage of those who abuse alcohol or other drugs.

Improving predictive accuracy is only one half of the prevention equation. In the extreme, improving prediction accuracy may make success irrelevant. Given the overall rates of risk in the population and the strength of relationship of these factors with substance use outcomes, it may well be that small effect on a large population may improve the health and outcomes of more persons than a large effect on a carefully articulated and much smaller at-risk sample. But for any given population this is an empirical question. Correlational strength is not a sufficient guide to reasoned selection. Unpacking the relationships nested in the correlation through use of  $2 \times 2$  contingency tables and the selection and outcome curves moves prevention science closer to a rational model of selection and application, and provides evidence necessary to anticipate the potential impact of effective prevention in real world settings. Correlational evidence alone cannot do that.

**Acknowledgments** Thanks and appreciation goes to Drs. Ali Habibi, Evangeline Danesco, Doug Mains, and Valerie Malabonga for their unflagging attention to detail and tireless assistance in coding the primary studies. This work was supported by a grant from the National Institute on Drug Abuse (DA09981) and by the Substance Abuse and Mental Health Services Administration, Center for Substance Abuse Prevention's National Center for the Advancement of Prevention. The opinions expressed in this study are those of the author and do not necessarily reflect the opinions or policies of the National Institute on Drug Abuse or the Substance Abuse and Mental Health Services Administration (SAMHSA), its staff, or employees.

## Appendix A: Meta-Analysis Procedures Applied in this Study

### Criteria for Selecting Studies

1. The study used a panel design with at least two waves of measures on the same persons and contained sufficient information to allow a reasonable inference of the age of the sample at each time of measurement.

2. The study reported quantitative results for the relationship of at least one predictor variable with some concurrent or later ('outcome') measure of alcohol, tobacco, or marijuana use.
3. The study was conducted in a western, economically developed culture, and the findings were reported in English.
4. Studies evaluating the effectiveness of interventions were excluded. Also excluded were studies in which samples were selected on the basis of subjects' status on the outcome behavior and then contrasted on various predictors, even when those antecedent predictors were prospectively measured.

### Effect Sizes

The primary effect size index used for this meta-analysis was the product-moment correlation coefficient between a predictor variable and an outcome variable representing marijuana use by the same subject sample. Many studies reported the relationships of interest directly as correlation coefficients; others reported statistical results in a form that could be converted to correlation coefficients, most frequently as  $2 \times 2$  cross-tabulations. It is often appropriate in meta-analysis to adjust some or all of the individual effect sizes in a distribution prior to analysis. These adjustments may transform effect sizes into more convenient forms, correct known biases, recode outliers, and adjust for various artifacts. The adjustments that applied to the effect sizes in the present meta-analysis were as follows.

### *Dichotomized Variables*

Each relevant statistical relationship was first converted to a correlation coefficient if it was not already in that form. This was accomplished using standard formulas such as those found in Becker and Hedges (1989), Hunter and Schmidt (1990a), and Rosenthal (1991). In cases where correlation coefficients were derived from dichotomized interval or ratio data, the resulting phi coefficients are known to be attenuated (Hunter and Schmidt 1990a, b). To correct this downward bias, the 147 effect sizes that were coded from dichotomized predictor variables whose underlying distributions were believed to be normally distributed were statistically adjusted to estimate the correlation that would be obtained if the predictor had not been dichotomized. Given that the underlying distribution of marijuana use is not normally distributed, effect sizes were not adjusted for dichotomization of the outcome. The formula used to adjust the 147 effect sizes for predictor dichotomization was as follows:

$$\hat{r} = \frac{r}{\phi(z)} \sqrt{PQ}$$

Where  $\hat{r}$  = Adjusted correlation,  $r$  = Coded correlation with dichotomous predictor variable,  $P$  = Predictor variable base rate (marginal probability),  $Q = 1 - P$ ,  $\phi(z) = y$

ordinate of the normal distribution at the point along  $z$  that corresponds to a cumulative  $P$  equal to the predictor baserate.

### *Data Reduction Procedures*

To avoid statistical dependencies, multiple effect sizes coded for this synthesis were aggregated within each unique study-sample prior to averaging across study-samples. Achieving this reduction required several steps, beginning with an aggregation procedure that averaged each unique combination of predictor factor with age at time of predictor measurement, age at time of outcome measurement and study-sample. This reduced the dataset from 2,090 effect sizes to 931 sample size weighted aggregated effect sizes. Next, these 931 effect sizes were examined to determine if there were any remaining dependencies from studies contributing data from both the study population and from breakouts within that study population (e.g., separate estimates for males and females). When multiple estimates were identified, those mean estimates based on the most observed data were retained. Finally, we removed the dependencies caused by having multiple study-sample effect sizes from different periods of measurement. This final aggregation reduced the original 931 effect sizes to 535 independent effect sizes, with each study-sample contributing no more than a single aggregated effect size to each outcome by predictor relationship.

### *Z-transformation and Weighting*

Following the advice of Becker and Hedges (1989) and Rosenthal (1994), the aggregated correlations were transformed prior to analysis using Fisher's  $Z_r$  transformation. The  $Z$ -transformation takes the following form:

$$Z_r = .5 \log_e \left[ \frac{1+r}{1-r} \right]$$

Where;  $Z_r$  = the Fisher's  $Z_r$  transformed value,  $r$  = the correlation coefficient,  $\log_e$  = the natural logarithm.

The  $Z$ -transformed correlation can be converted back to a correlation via the inverse of the  $Z_r$ -transformation:

$$r = \frac{(e^{2z} - 1)}{(e^{2z} + 1)}$$

Where;  $r$  = the correlation coefficient,  $Z_r$  = the  $Z$ -transformed correlation,  $e$  = the base of the natural logarithm (approximately 2.71828).

All computations and analyses used the  $Z$ -transformed aggregated effect sizes and, additionally, weighted those effect sizes to reflect the greater reliability of effect sizes based on larger samples than those with smaller samples. However, the effect sizes were computed from samples ranging from 5 to 49,458 subjects. To

keep studies with exceptionally large sample sizes from dominating the weighting procedure, sample size weights exceeding  $n = 699$  were recoded to 700.

The sample size weights applied to the  $Z$ -transformed aggregated effect sizes were calculated simply as  $w_i = (n_i - 3)$ , where  $w_i$  is the weight that is multiplied times each effect size and  $n_i$  is the sample size upon which that effect size is based (Becker and Hedges 1989; Hunter and Schmidt 1990b).

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