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The Prevention of Alcohol and Drug Involvement: Forging The future From Past Lessons Learned

La Prevención de la Adicción al Alcohol y Drogas: Forjando el Futuro a partir de las Lecciones Aprendidas en el Pasado

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This paper is focused on prevention science and practice as they apply generally to health and behavior and specifically to problems caused by involvement with alcohol, tobacco, and other drugs (ATOD). The paper which follows, considers issues related to the evaluation of interventions to prevent disease/disorder and promote health.

Focussing on ATOD: The focus of this paper is on *involvement with* alcohol and other drugs (AOD). This terminology is used to refer to the range of problem experiences which arise as a consequence of substance use, abuse, dependence, acquisition and (particularly in the case of drugs) distribution. I discuss these elements below. I use the term of *ATOD* to make explicit that alcohol is a drug and must be recognised as such, especially for children and adolescents. It is essential that health scientists as well as the general public recognise alcohol for what it is, i.e., the second most widely used and potentially lethal of all substances available to people. The most lethal substance is tobacco. Together, alcohol and tobacco, contribute heavily to the major causes of mortality and morbidity in the United States and, I expect, in Chile. Separately or in combination, they represent confirmed risk factors for multiple diseases. Less emphasised but equally substantiated is their association with a variety of emotional and behavioral problems ranging from delinquency to conduct disorder to affective disorders.

Readers should note that I do not use the qualifier *illegal* when speaking about substances. Since my work focuses primarily on youth, *all* of these substances are illegal. Were I able to do so, I would protect youth from involvement with any of these substances for as long as possible. The reasons for doing so are discussed below. It should be clear, however, that tobacco and alcohol represent highly significant threats to the health and welfare of youth. Anything we adults say or do which minimizes that threat should be recognized as such and avoided. It is *our* responsibility to keep youth and these gateway substances as far apart as possible.

At the same time, I am not advocating prohibition against alcohol or tobacco for adults. It is widely known that these substances have been scientifically established for potential for lethal consequences. Used in moderation, however, they can also provide enjoyment, relaxation and, in the case of alcohol, even some health benefits. As noted later, therefore, adult involvement with alcohol and tobacco requires distinct preventive goals. Neither time nor the focus of this meeting allow for a discussion of the legalization of marijuana and other drugs. I will simply state that I remain opposed to legalization for both public health and public policy reasons.

Phases of prevention science: The paper begins by reviewing briefly elements of the history of prevention science in the United States. The intent is not to criticize that work but rather because I firmly believe that if one does not consider history one is likely to repeat it. Hopefully, my words will provide some guidelines about what to avoid as Chilean scientists and health planners cross the challenging minefield of prevention science and practice. In thinking about preventive interventions I have found Cowen's (1980) distinction between generative and executive components of prevention research and Price's (1983) four domains of preventive science most helpful. Elaborating on Cowen's distinction, Price (1983) recognized that, ideally, intervention development proceeds through a series of steps including:

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a) problem analysis (i.e., description, epidemiological and etiological studies);
b) innovation design (i.e., program development and piloting);
c) field trial (i.e., field-based trials of efficacy); and
d) innovation diffusion (i.e., field-based trials of effectiveness).

In its analysis of the state of prevention science (Mrazek & Haggerty, 1994), the National Academy of Science's Institute of Medicine translated Price's phases into the components of the 'preventive intervention research cycle'. The most recent iteration of these steps is the NIMH's (National Institute of Mental Health, 1996) five phases of preventive intervention research. These phases may be perceived as a basic recipe for any prevention program:

a) define the problems of interest and study their extent;
b) study risk and protective processes that influence the (non)development of these problems;
c) develop and assess the efficacy of preventive trials to change the risk and protective factors and thereby influence problem incidence and prevalence;
d) conduct large scale trials of demonstrably effective prevention programs; and
e) facilitate program diffusion and evaluation.

By definition, prevention science requires the true integration of theories and methods from social, behavioral and health sciences. Anthropology, developmental psychology, epidemiology, experimental psychopathology, sociology, clinical medicine and many other disciplines must collaborate for genuine advances to occur. Efforts which focus on ATOD must, of course, incorporate the insights of behavioral and clinical psychopharmacology, clinical psychiatry and psychology and even criminal justice.

Arguments in support of the development of interventions to prevent emotional and behavioral disorders echo the longstanding public health maxim that the spread of diseases such as measles, polio, smallpox and AIDS will never be controlled by treatment but only by prevention. Nearly four decades ago, President Kennedy adopted that argument in his call for the development of a 'bold new approach', i.e., a national system of community-based centers to treat and prevent mental illnesses:

"A... more than in any other area, an ounce of prevention is worth more than a pound or cure. For prevention is far more desirable for all concerned. It is far more economical and far more likely to be successful. Prevention will require both selected specific programs directed especially at known causes, and the general strengthening of our fundamental community, social welfare, and educational programs..." (Kennedy, 1963 reported in Bloom 1977, p. 264).

Little recognized is the fact that early in its development, the CMHC movement could have solidly established its capacity to prevent disorder. As early as 1962, the American Public Health Association had identified six categories of mental disorder which, because their etiology was known, were preventable (Bloom, 1984). These categories included diseases caused by: a) poisons (i.e., brain syndromes following exposure to solvents and toxins such as alcohol); b) infections (i.e., rubella); c) genetic disorders (i.e., TAY- SACHS disease); d) nutritional deficiencies (i.e., pellagra); e) injuries (i.e., traumatic head injuries) and f) general systemic disease (i.e., prematurity). At that time, work by Spitz and others had changed how institutionalized infants were cared for to the point that marasmus had been eliminated as an early affective disorder. Guidelines for shifting emphasis from treatment to prevention were described by Caplan in his influential works, *Principles of preventive psychiatry* (1964) and *The theory and practice of mental health consultation* (1970). Riessman (1965; 1967) among others (i.e., Fairweather, Sanders, Maynard, & Cressler, 1969) described innovative ways in which self-help and environmental change, alone or in combination, offered relief which could precede and potentially avoid dysfunction and exacerbation of existing problems. These efforts did not depend on professional resources to operate and, under appropriate circumstances, could have been sustained by, and perhaps sustained, the CMHC movement.

Into the minefield: In retrospect, building on attainable successes, prevention science and practice could have set a national agenda which might have anticipated if not avoided substantially the substance abuse crisis which has plagued the United States since the Vietnam era. At the very least, we would have thought about the problem as a prevention rather than treatment challenge and had at our disposal, methods for conceptualizing, designing and implementing interventions. Instead, Levine and Perkins (1997) concluded that the community mental health movement:

"A... went off in all directions at once, with little coherence and little conceptual clarity. Critics committed to traditional medical model practice looked askance at social activism. Those committed to 'intrapsychic supremacy' (Levine, 1969) - the belief that problems in living result from people's internal

psychological structures, which in turn dictate perceptions, feelings, and actions in everyday situations - viewed the activists as misguided romantics who had foolishly strayed from proper professional roles and activities. Community-oriented critics of traditional practice were equally firm in their convictions but had little to offer by way of alternate conceptualizations (pp. 59-60)."

Even worse for prevention science, its early advocates took Caplan's (1964) call for adoption of the public health model of primary, secondary and tertiary prevention too literally. Caplan (1964) recognized that the mental health disciplines had to move beyond sole reliance on treatment if they were to serve public needs. To organize initial efforts in this direction, Caplan urged mental health to adopt the goals of and the classification system for prevention used in public health at the time (Commission on Chronic Illness, 1957). This system was centered around the epidemiological concept of caseness, i.e., confirmation that the diagnostic criteria defining a syndrome were met. Intervention categories were differentiated within this system in terms of their proximity to the targeted condition's fulfillment of diagnostic criteria, i.e., meeting the symptomatic definition of a syndrome. Preventive interventions were designed to reduce the prevalence of a targeted disease or disorder.

Within this framework, *primary* prevention interventions are implemented prior to caseness. Successful interventions reduce prevalence (i.e., overall presence of cases of a target disorder in the population) by reducing incidence (i.e., the occurrence of new cases). Potential recipients of such interventions range from the population at large, to asymptomatic subgroups targeted on the basis of epidemiologically-defined risk, to subgroups presenting prodromal signs antecedent to diagnostic status. Taken literally, primary prevention programs could only be applied *before* the onset of the pathogenic process. Given limited agreement on definitions of the onset of emotional, behavioral and substance disorders, the appropriateness of this category for mental health could be and was (i.e., Lamb & Zusman, 1979) challenged. Moreover, far too much time, effort and resources were wasted debating the relative merits and acceptability of primary, early secondary and late secondary interventions (i.e., Albee, 1982, 1986). In fact, traditional public health definitions defined primary prevention as any intervention focused on the pathogenic process to the point of diagnostic criteria. Hence, the debate was moot early interventions of most forms would have served to reduce incidence.

By contrast, *secondary* interventions seek to lower prevalence by reducing the duration of caseness, i.e., through the application of effective treatment. Combinations of sensitive screening procedures which identify cases early and involve them in treatment exemplify this category. Secondary approaches seek both a return to premorbid status and avoidance of subsequent episodes. Finally, *tertiary* interventions reduce the long term disabilities and sequella consequent to caseness. Rehabilitation strategies and support services exemplify tertiary approaches. Insofar as they enable a person with schizophrenia to live independently, tertiary goals are achieved. If recurrence of disabling symptoms is avoided, secondary goals are achieved.

Under many circumstances, primary prevention interventions are preferable. This assumes, of course the availability of necessary etiological information, access to the population at risk and effective intervention strategies. It also assumes that the intervention's effects are, at worse, neutral. This is a particularly important point given that the diseases and disorders of most interest to mental health are relatively rare events, generally effecting between 1 and 6% of the general population (Rouse, 1995). Hence, primary preventive interventions targeted to the general population are unlikely to reduce substantially the prevalence of specific disorders. Given base rates, whatever effects they have can only be detected if evaluation designs involve very large samples. If the intervention has iatrogenic consequences for more than 5% of the population, then the intervention may represent a greater risk than the disorder to be avoided. Truly a case of the cure being worse than the illness. Dismissal of this concern notwithstanding (Albee, 1986), evidence for caution has been reported (i.e., Lorion, 1987; Sameroff & Fiese, 1989) although the frequency of negative consequences appears to be low (Durlak & Wells, 1996).

Secondary and tertiary prevention efforts, by contrast, are targeted only to those meeting diagnostic criteria and hence having confirmed need for the intervention. It would seem reasonable therefore to suggest that our preventive efforts focus on such approaches. As I argued years ago (Lorion, 1983), the decision should be based on science. If we can implement primary prevention efforts which are demonstrably effective, we should do so without question. If, on the other hand, too little is known about the epidemiology and etiology of the outcomes to be avoided, if access to the populations is not available or if evidence of effectiveness is unavailable,

by all means we need to use every resource available to reduce prevalence through the application of early detection and intervention (i.e., secondary prevention). Acceptance of this reality has been invaluable for reducing morbidity and mortality associated with cancer, heart disease and many other physical disorders. As will be discussed later, it has also served those with substance problems well.

Beyond the minefield: For many years, the applicability of the classic public health triad of approaches to emotional and behavioral disorders has been challenged (i.e., Albee, 1982; Lamb & Zusman, 1979; Lorion, 1983). As reflected in the work of Brofenbrenner (1977), Rutter (1989), Sameroff and Fiese (1989) and Sroufe (1997), advances in the developmental sciences provide an emerging appreciation of the etiological complexity of emotional and behavioral problems involving biological, psychological, social and environmental parameters. This complexity obscures determination of the onset of pathogenic processes, the presence of disorder and thereby designation of 'caseness' (Lorion, Price, & Eaton, 1989). The resulting conceptual and methodological changes in understanding etiology add to confusion about the categorization of primary and secondary interventions and how to prove their efficacy.

Fortunately, Gordon (1983, 1987) has proposed an alternative. His taxonomy for organizing preventive efforts allows for consideration of contemporary views of pathogenesis and the development of health and disorder and facilitates aspects of evaluative designs. Given the low base rate at which emotional and behavioral disorders occur in the general population, Gordon's system also allows for consideration of the financial costs and iatrogenic potential of preventive interventions. Weighting expected intervention risks with target selection, Gordon (1983) proposed that 'universal', 'selective' and 'indicated' interventions should be designed for and targeted to 'persons' not motivated by current suffering" (p. 108) and neither currently seeking nor in need of treatment:

a) *Universal* interventions are applied generally and combine low-costs per contact with limited likelihood of iatrogenic consequences. Examples include public service announcements advocating seat-belt use, physical exercise or discouraging tobacco use and the consumption of alcohol during pregnancy. The incorporation of information about ATOD in health curricula represents another universal intervention. Perhaps the designator 'universal' may also be applied to the generic nature of the risk factors targeted by such interventions (i.e., exposure to advertising for tobacco or alcohol).

b) *Selective* interventions are targeted to segments of the population for which there is an epidemiologically established risk. Selection may be based on identified links between gender, ethnicity, economic status or family history, for example, and the presence of a substance related disorder in prior generations or siblings. Since targeted subgroups have a higher likelihood of disorder and hence a more focused (and potentially more iatrogenic) intervention may be justified. Groups for children of alcoholics or children placed in foster care because of parental involvement in the sale or use of substances would be a selective approach.

c) *Indicated* interventions target individuals (rather than subgroups) who present risk factors or prodromal signs indicating substantial individual risk for disorder. Examples include programs for adolescents referred to juvenile court for status offenses (Lochman, 1992) as well as court mandated educational programs for those found guilty of driving an automobile under the influence of alcohol or another substance. A program which confronts youth found to possess or use substances would be another such program. However they are designed, indicated interventions are designed to interrupt and hopefully abort the further development of the problem.

A theory of the problem: As discussed in the paper which follows, Gordon's approach to categorizing preventive interventions has important implications for the design and evaluation of these interventions. His proposal distinguishes prevention from treatment and applies concepts of 'risk' and 'vulnerability' to individuals, to situations (i.e., to risky settings) and to individuals *in* situations. It differentiates the conditions to be avoided (i.e., substance abuse or depression) from the individuals in whom they occur. Gordon's position echoes Sameroff's (1977) plea for prevention scientists to avoid equating 'risk' with the actualization of disorder and the presence of risk factors with the absence of competencies and resources. Dra. Bertha Gonzalez reminded me of this during my visit to a health clinic, in a low-income Santiago neighborhood. Since all of her patients were at very high risk, Dra. Gonzalez explained that focusing on their risks provided insufficient information. By understanding their strengths and their resources, however, she could make the distinctions necessary for planning treatment and gauging its effectiveness (personal communication).

The commitment to balance considerations of health *and* pathology, of risk *and* resources and of people

and places fits readily with emerging conceptions of emotional and behavioral development. In the time space available I can merely highlight the primary elements of this work. To prevent a problem one must have a 'theory' of how it develops and spreads through the population. Involvement with substances of any kind results from a developmental process and thus its alteration requires a developmental process. Effective prevention programs depend on understanding such developmental processes. One's theory of the problem reflects one's understanding of how a problem develops. One's theory of the solution reflects one's understanding of necessary change processes.

For decades, the classic 'host-agent-environment' model for understanding the occurrence of disease has provided a theory of the problem and of solutions for infectious diseases and hazardous situations Importantly, this model looked both *within and outside* the individual to understand vulnerability and resistance to diseases. This perspective is central to contemporary models of illness (Lilienfeld & Lilienfeld, 1980). More than six decades ago, Lewin (1935) asserted the importance of contextual factors in his recognition that Behavior is a function of the Person and the Environment. Importantly, Lewin's insight has informed current thinking about how behavior develops and can be changed.

The most important contribution from what is a very substantial body of work on developmental processes to prevention theory and practice is the concept of the "transaction", i.e., the interrelational processes between individual and situational factors in which each reciprocally influences the other. Each component in the transaction influences the successive states of the other. Sameroff's work (i.e., Sameroff & Chandler, 1975; Sameroff & Fiese, 1989) exemplifies this explanation of variation in development (i.e., why all who encounter a risk do not express a disorder). Thus, emotional and behavioral development is the product of an ongoing series of: a) responses by an individual to situational circumstances and demands; b) alterations of those situational circumstances and demands as a function of the individual's responses; and c) responses by an individual to that altered.

Sameroff's model explains the continuing adaptation to events which characterizes the ongoing flow of daily life. It also offers a basis for understanding variations in behavior across situations. In one sense, unyielding settings would constrain behaviors; flexible settings may allow far more variation (with both positive and negative consequences). In the most recent version of the model, Sameroff and Fiese (1989) make explicit that societal (and, presumably, subgroup) expectations regulate definitions of behavioral roles and thereby set contextual limitations on developmental outcomes. Insofar as such expectations and setting demands are fixed, the behavioral responses of those within a setting may be more restricted than assumed or desired. The programmatic importance of this insight is that behavioral outcomes, i.e., substance involvement, can be influenced by changing the individual, the situation and the social regularities which define individuals within situations. As I said earlier, for example, what we as adults and as a society say and do about youth's involvement with alcohol and tobacco has significant impact on their health and welfare. That involvement, in turn, has significant impact on the likelihood of their further involvement with substances and its consequences.

Knowing that both individual and environmental factors influence outcomes is important. Unlike the agent-host-environment model which explains many infectious diseases, few emotional and behavioral conditions (including substance involvement) can be linearly modeled. More typical is Sroufe's (1997) portrayal of emotional and behavioral development as the branches of a tree or the meanderings of its roots. Within this perspective, growth and survival requires maximizing one's access to necessary resources regardless of the impediments encountered in the environment. At any point in time, emotional and behavioral status reflects the cumulative effects of its antecedents. The further back one traces a branch the more completely one can understand what has been and will be encountered.

With such information, the better one can predict the *probability* of the branch's likely direction. Where the branch connects to the trunk is but one element to be considered in predicting its various termini. That beginning is likely to be common across many outcomes (i.e., the principle of multifinality). Equally likely is that termini in close proximity may have begun at different points along the trunk (i.e., the principle of equifinality). Adding to the complexity is that some branches will end close to where they began whereas others continue on toward other terminating points. Presumed antecedents (i.e., alcohol or tobacco use) for some conditions (i.e., substance dependence or addiction) may, for many, represent an endpoint (i.e., substance use is limited, controlled or even ended). In other

cases, however, the process continues toward dependence and addiction.

Looking at substance involvement: Given this complexity, how should we think about substance involvement? If what I have said thus far is accurate, we must acknowledge that common antecedents lead to diverse outcomes; diverse antecedents lead to common outcomes; multiple outcomes occur together. All of this may be perceived as too complex and unresolved to inform the design of preventive interventions (Lamb & Zusman, 1981). Should we wait until the die is cast, the nature of the problem is clear and a demonstrably effective intervention can be selected and applied? Public health practitioners might accept this approach were the costs (health related and financial) of delay minimal, the effectiveness of intervention certain and the secondary consequences of the disorder acceptable. The common cold and the 24 hour flu fit these parameters. Substance involvement, however, does not. While we wait for all or most of the answers, youth are being damaged, families hurt, lives lost. The secondary costs of our failure to effectively reduce substance involvement are too high and too damaging to communities.

Rather than wait, therefore, we should interpret the aforementioned complexity as allowing for, maybe even requiring, non-specific interventions focusing on common etiological factors, be they individual, environmental or transactional. Targeting common risks factors or *simultaneously* pathways which are epidemiologically linked with undesirable outcomes has the potential for impacting multiple outcomes. Simply stated, interventions targeting common antecedents may impact on multiple pathways and thereby serve as a stone which kills (or at least weakens) multiple birds! By aggregating the measured preventive effects across those outcomes sharing common risks, the true impact of such interventions may finally be documented (Lorion, Price, & Eaton, 1989). Applying the principle of multifinality to the design of interventions leads to targeting a limited number of shared risk factors and assessing their impact across alternative pathogenic expressions. Similarly, the principle of equifinality requires that interventions intended to reduce the occurrence of a specific outcome must target the various pathways and risks associated with that outcome. In both instances, basic elements of developmental theory supported by epidemiological findings argue against the likelihood of documenting substantial evidence of preventive outcomes if only a limited number of risks factors are targeted with the intent of impacting a single form of disorder or dysfunction. As noted below, neither prevention theory nor the traditional intervention taxonomy made that point clearly.

First, we must acknowledge that substance involvement is not a problem; it is *many* problems! Before we can design an intervention, we must therefore decide which problem we wish to tackle for each effects a different segment of the population and will likely require a different set of intervention components. Unfortunately, much of the prevention literature appears to equate 'use', 'abuse', 'dependence' and 'addiction'. Some interventions designed to avoid use argue for prohibition and total abstinence from any involvement with the substance. For some of these, the prohibition extends across all substances and all times. For others, the goal is to avoid entirely the use of some substances and to delay the onset of use of others depending on individual (i.e., age) and setting (i.e., driving an automobile) factors.

Elements of an intervention program: Available and emerging scientific findings support the conclusion that the use of any ATOD substance prior to puberty and during early adolescence is to be avoided if at all possible. The physical, psychological, educational and, in some cases, legal consequences of such involvement represent too high a price to pay. Moreover, there is little doubt that ATOD involvement has a sequential or gateway characteristic. Substances such as marijuana, cocaine, heroin and amphetamines are rarely used by individuals who have not already experimented with, and perhaps use(d) regularly alcohol and tobacco. This does not mean that the use of tobacco and alcohol causes the use of the other substances. Rather it means that alcohol and tobacco use represents, generally, a necessary although nor sufficient condition for their use.

We have known about gateway substances for more than 20 years (Kandel, 1975). Yet, few interventions explicitly identify delaying the onset of use as their goal. I am certain that political rather than scientific factors contribute to this decision. Educational interventions to prevent use frequently incorporate scare tactics and messages into their programs. Social psychologists informed us long ago about the limitations of such approaches to changing attitudes and behaviors. Yet, we continue to offer such programs with the stated intent of protecting youth from ever touching alcohol and other drugs. Although I cannot cite evidence, I suspect that those involved in such programs (or receiving such programs) do so

with limited optimism. Moreover, we need to consider the implications of the failure of such programs. Consider, for example, the potential for self-fulfilling prophecies when those receiving such programs had been told that serious involvement with alcohol and other drugs (including, perhaps, an inability to control use) would *inevitably* follow the onset of use. Early educational efforts to control crack use, for example, highlighted the *immediacy* of the substance's addictive quality. Crack users I have spoken with pointed out that they believed they were not vulnerable to addiction since they had been able to control their use of crack for some time after its onset.

An alternative approach for public education prevention programs would be to truthfully discuss the reasons why all substances should be avoided until at least mid-adolescence. Such a program could explain the consequences of early onset in ways that can be confirmed directly by members of the intended audience we wish to reach. Such a program should also include very severe penalties for those who provide ATOD to youth during this especially vulnerable period. The dilemma with this proposal is that it would require careful consideration by health providers, policy makers and citizens of what, if any, levels and forms of ATOD use would be acceptable for pre-adolescents, adolescents, young adults and older adults. It would require that the resulting policies be translated into a comprehensive and consistent program. Public condemnation of driving under the influence of alcohol and other drugs, for example, is rarely accompanied by the mandatory loss of the right to drive. The use of ATOD by high school and college athletes rarely leads to immediate suspension or expulsion. The debate going on now in the United States about controlling the companies which manufacture cigarettes clearly reflects our ambivalence. In spite of clear evidence of an intentional and longstanding program by manufacturers to encourage young people to smoke, there is reluctance to hold the companies accountable. In spite of opposition to alcohol use by minors and by underage college students, little genuine effort is made to enforce that opposition.

I am not suggesting that we oppress our youth or become a police state. I am merely pointing out that the effectiveness of any preventive intervention based on information or attitude change is likely to be seriously compromised by inconsistencies between what we as a society say about ATOD use for youth and what we do (Resnik, Gardner, Lorion, & Marcus, 1990). The implementation of policies at the national level as well as at the community and even home levels are important parts of the real information used by youth as they decide whether or not to begin, continue, increase or end substance involvement. Evidence of the value of consistent information is best provided by Project Star (Pentz, Dwyer, Mackinnon, Flay, Hansen, Wang, & Johnson, 1989). This program takes place at the community level and involves the family, the school the neighborhood and the community in presenting a single message to youth about the importance of avoiding use. To aid in this effort, the program provides information about the consequences, physical, emotional, educational, etc. of gateway drug use. Supporting these messages are school-based programs to assist youth to develop peer-resistance skills (i.e., to say no to opportunities to use drugs) and other programs to enhance youth's self-esteem. Recreational elements provide substance-free alternatives for having fun and relating to peers. Compared to comparable communities without this program, Project Star settings report substantial reductions in substance which appear to hold during the critical adolescent years.

In a series of studies, my research team first examined the prevalence of ATOD use among children ranging in age from 9 to 18. Our work began as an epidemiological study to establish the base rate at which ATOD use began early. We found that by age 10, approximately 20% of children had experimented with some form of substance, especially alcohol and tobacco. We were surprised to find that girls were as likely as boys to become involved with substances of most concern such as inhalants and crack/cocaine. In a second study, we discovered that children had learned about the dangers of ATOD use in their health course and expected negative reactions from parents, teachers and peers for ATOD but not for becoming involved in tangential roles in the sale and distribution of drugs. Our initial preventive effort was to modify the curriculum to clarify the dangers of such involvement.

We also learned (as had many other researchers) that young children had interpreted public discussions about ATOD, the contents of their courses on alcohol and drugs, and the comments of others as evidence that ATOD use was far more prevalent than reported. Generally, their estimates were 2-3 times as high. Thus, if 3-4 children reported ATOD use in a classroom of 30 children, those in the classroom might predict that 9-10 were using. This seemed to us to

suggest an artificial source of social pressure (i.e., the sense that I'm one of the few who hasn't tried...). We changed the curriculum further by adding information about levels of use in that classroom, that school and that grade level. Now those who were using became different than their peers.

Our studies also revealed that factors associated with early use included elements of what we referred to as developmental asynchrony, i.e., the experience of being confronted with demands for which one was not developmentally prepared. Simply stated, the child was placed in a situation before he/she was ready. Examples included assuming parental duties in a single parent family (i.e., a young girl had to raise younger siblings while mother worked many hours outside the home or a young boy had to raise money to help support the family); experiencing puberty earlier (for girls) or later (for boys) than one's peers; and living in a violent community. These same studies showed us that a protective factor against such experiences was the presence of a concerned parent or caregiver who took steps to match the child's developmental status and demands. Simply stated, these parents and caregivers established and enforced rules about curfews, dress styles, avoiding certain people and places, etc. We added to local interventions a component for parents which said little things can make a difference. Early findings suggested parents liked these hints and felt empowered by them.

We also developed a program in two high-risk schools in which Home-School Liaisons were trained to create a supportive bridge between children and teachers and teachers and mothers (Flores-Fah, Lorion, & Jakob, 1997). The Liaisons were from the schools' neighborhoods and assisted the children in acquiring the academic and interpersonal skills necessary to successfully move on to middle school. Our findings after five years revealed that the Liaisons made an important difference in how well the children were doing but also in how comfortable parents and teachers felt about discussing their concerns with each other. Our sense was that the program assisted the two to become a team in support of the children's needs. By the program's end, we had evidence of very low ATOD involvement in our program schools. As importantly, the school's Principals found ways to support the continuation of the program after our research grant was completed.

Felner and his colleagues describe a school-based program to assist youth with the transition from elementary to middle school and high school (Felner & Adan, 1988). Essentially, the program reflects an appreciation of the importance of the peer group and peer support during early and mid-adolescence. Rather than having the students randomly move across classes, they are organized into fixed groups which remain together throughout the school day. Each group has a specific guidance counselor assigned to it throughout its enrollment in the school. As a group, the students discuss substance use, reasons for non-use and their shared commitment to assist each other to avoid use. Early findings from this program reveal both substantial academic gains and impressive reductions in reported ATOD use. This model certainly merits further replication and long-term study.

Interventions based in individual schools, be they peer-resistance programs, DARE or skill-building programs appear to have generally positive effects. The consistency of these effects across settings and over time, however, remain in question. The most critical variable appears to be the consistency of implementation and the commitment of the providers of these programs. Given substantial evidence that settings which are involved in program development tend to have the best effects, it seems reasonable to identify effective components, develop the means to deliver them and encourage settings to organize them in the most setting-appropriate manner. Potentially, the process of this planning, organizing, testing and implementing may itself be an important element of effective prevention.

Targeted programs: What I have described thus far are generally universal interventions directed to all youth in a community or school. As I noted before, however, programs to avoid the onset of use must be understood as different in purpose from those targeted to youth (or adults) who have already begun to use. Some might argue that the goal must be the same, i.e., to eliminate all use regardless of its nature or the age of the user. I appreciate that position and, at times, support it. But then I am confronted with several realities. First, I must determine which is more important to me, the elimination of use or the elimination of the problems which result from or correlate with use? Am I willing to accept those consequences and remain focused on the elimination of use? We all know some of the problems. In the short term, the use of alcohol and other drugs is associated with death and injury resulting from automobile and other forms of accidents. ATOD use is associated with adolescent

suicide, homicide, pregnancy, delinquency, and date rape.

Programs such as SADD (Students Against Drunk Driving) target the problem of drinking and driving by establishing ways to disconnect these two risk factors. Programs which offer alcohol-free alternatives to prom and graduation parties, programs to establish agreement among parents concerning matters such as curfews, adult presence at parties, access to alcohol, all seem to reduce the incidence of problems associated with alcohol consumption. The problem with such programs is not that they do not work! The problem is that they represent acceptance of the reality of youth's ATOD use. The problem is that they convey the message that ATOD use, at least insofar as alcohol is concerned, is acceptable *if it is responsible.*

Are we as a society, as parents, ready to say that? Epidemiological data indicate that only a small portion of those who try alcohol and other drugs during adolescence will become dependent or addicted. Far more will suffer injury and death from the misuse of those substances. Can we design programs which will control the problems but not the use of alcohol? If so, what other drugs will be acceptable if used responsibly? What of that small but most important segment of the youth population who are physiologically or emotionally vulnerable to substances and highly likely to become dependent or addicted? Can we take chances with their lives in order to protect those whose risk is not the substance but its associated problems? These questions should give you insight into why I oppose legalization. They should also make evident the need for a considered discussion of what, to date, have been the unasked questions and the unspoken agenda.

If the reduction of substance-related problems represent examples of selective interventions, what of indicated interventions. Two come to mind. The first targets youth found to be involved with substances and mandates their participation in educational and, at times, group interventions. The second focuses on children with a family history of alcohol or other drug abuse and attempts to enhance their appreciation of their heightened risk for physiological problems with substances. Neither of these approaches has been carefully studied and thus we know relatively little about their effectiveness.

Next steps: In thinking about how to end these comments, I tried to find some really good news to share with you. I hoped to provide the 'magic bullet', i.e., to tell you that if you do the following, your ATOD problems will be controlled and our children will grow up happy and alive. Unfortunately, I could not find such an intervention. Then it struck me that one must exist. Recall the epidemiological finding I just mentioned, i.e., a substantial number of youth leave high school having taken a 'bite of the apple'. Nearly 3 out of 4 will have consumed alcohol; nearly one in two will have tried another substance. Yet, a decade later, most will have discontinued use other than under socially accepted conditions. The vast majority who try substances other than alcohol, discontinue such use. Some say they don't like how it makes them feel. Some say they fear its capacity to capture them. Many say they just lose interest. Why not look upon the discrepancy between the proportion who experiment and those who continue as both evidence of some success and as a potential source of information about what we need to say and do to accelerate that insight. Perhaps those who have tried and discontinued on their own as well as those who have never tried, in spite of opportunities to do so, have information we need to tap if we are to develop the next generation of ATOD prevention programs.

The only other recommendation I can make is to encourage families, neighborhoods, schools and communities to accept this as their problem and not wait for professionals to solve it. Ultimately, it will be solved by individuals making a difficult decision alone or with the advice and support of family and friends in the context of what others say and do about ATOD use.

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