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Prevention for Children of Alcoholics and Other High Risk Groups

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1. Introduction

Median age of onset of alcohol use nationally is age 14 and median age of first drunkenness is 17 (Johnston et al., 2003). Thus, it is not surprising that the greatest public concern about drinking among young people begins with a focus on adolescence. It does not necessarily follow that the problems of risk for children of alcoholics[1] (COAs) and other children at high risk for the eventual development of alcohol use disorder (AUD) are the problems of adolescence. In fact, a now substantial body of evidence indicates that the drinking problems and other difficulties of adolescent and young adult COAs are predicted by much earlier markers. Thus the prevention question for this population becomes one of dealing with when the most appropriate time is to begin the intervention (i.e., what age to target) as well as how best to dampen or eliminate risk. Similarly, what is known about the adult disorder can also be informative about what to prevent, and what some of the prevention issues may be, given that this is the parenting generation. This chapter applies a developmental lens to the problem of prevention of risk among these very high risk populations.

1. We use the generic term "alcoholism" as well as the term "alcohol use disorder" interchangeably in this chapter to refer to what is more precisely designated in DSM-IV (American Psychiatric Association, 1994) as alcohol abuse and alcohol dependence. The more differential terminology is used when a more fine grained distinction is called for.

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2. Scope of the Problem

According to National Longitudinal Alcohol Epidemiologic Survey (NLAES) data (Grant, 2000), approximately 9.7 million children age 17 or younger, or 15 percent of the child population in that age range, were living in households with one or more adults classified with an alcohol abuse or dependence diagnosis during the past year (Table 1). Approximately 70 percent of these children were biological, foster, adopted, or step-children. That is, 6.8 million children meet the formal definition of COA, although, as noted below, not all are exposed to the same level of risk. In addition, 12 percent of the 66 million children in this age range were younger siblings of the alcoholic adult, 9 percent were other biological relatives (e.g., cousins, grandchildren) and approximately 6 percent were nonrelatives with or without their own relatives in the household, or were in an unspecified relationship. All of these other children and youth likewise fall under the umbrella of elevated socialization risk, although degree of biologic risk is probably lower.

Table 1. Number and Percentage of Children Living in Households with One or More Adults Who Abused or Were Dependent on Alcohol

	Parent AUD	
	During Past Year No.[a] (%)	During Child's Lifetime No.[a] (%)
Sex		
Male	4.7 (48.4)	14.3 (51.1)
Female	4.9 (50.5)	13.7 (48.9)
Race/ethnicity		
Black	1.1 (11.3)	2.4 (8.6)
Non-Black	8.5 (87.6)	25.7 (91.8)
Age (Years)		
0-2	1.8 (18.6)	5.3 (18.9)
3-5	1.7 (17.5)	5.1 (18.2)
6-8	1.7 (17.5)	5.0 (17.9)
9-11	1.5 (15.5)	4.6 (16.4)
12-14	1.4 (14.4)	4.3 (15.4)
15-17	1.5 (15.5)	4.0 (14.3)
Total Exposed	9.7 (100)	28.0 (100)
Total US Child Population	66 (15)	66 (43)

[a] In millions

Note: Adapted from Grant, B.F. (1997). Estimates of U.S. Children Exposed to Alcohol Abuse and Dependence in the Family. Am. J. Pub. Health, 90: 112-115.

Given that these figures concern *past year* exposure to at least one alcoholic adult, from the perspective of socialization risk they only reflect acute exposure. Other data from the NLAES provide estimates of magnitude of overall child risk pertaining to exposure to an either currently or previously alcoholic adult; the figure is 43 percent of the under-18 population, or slightly less than half of all children (also Table 1). The figure for COAs is only 30 percent, but this is still a literally enormous population of risk. Taken together, these figures speak to the social complexity, and likely risk variability among the families and households in which risk has the potential to unfold. At the same time, they also speak to the enormity of the social problem.

A second point needs to be underscored. COA status is heavily used as a proxy for "alcoholism risk" on the one hand, and socialization risk on the other, but the COA designation more precisely is a proxy for multiple causal inputs, not all of which may be present in the individual case. Thus, being a COA implies elevated genetic risk, on the average, although the alcoholic genetic diatheses may not have been passed on to a particular child. One may be a COA without being undercontrolled, having an attention deficit hyperactivity disorder (ADHD) diagnosis, etc. Moreover, the genetic risk is polygenic, and the alleles conveying risk are not always the dominant ones so that additivity of risk to produce problem outcomes is the rule rather than the exception (Rutter, 1982; Stoltenberg and Burmeister, 2000). Socialization risk involves exposure, but given the heavy divorce rates found in this population, evaluating level of socialization risk is complex, involving quantification not only of how long the exposure has been, but also the developmental period during which the socialization took place. Some developmental periods have the potential to be more vulnerability-producing than others (Fuller *et al.*, 2003). In addition, a substantial amount of marital assortment occurs in alcoholic families (Hall *et al.*, 1983). When assortment is present, risk exposure is multiplied, and COA effects become a function of genetic risk(s), individual parent risk, and the synergistic risk created by marital interaction (Fuller *et al.*, 2003).

Third, COA risk is not simply risk for the development of AUD. Given what is known about the elevated comorbidities found among offspring of alcoholics, this designator is also a marker of elevated risk for behavioral and cognitive deficits. These include attention deficit disorder, behavioral undercontrol/conduct disorder, delinquency, lower IQ, poor school performance, low self esteem, etc. (Noll *et al.*, 1992; Nigg *et al.*, 1998; Poon *et al*, 2000; Sher, 1991; West and Prinz, 1987). Furthermore, the evidence strongly implicates some of these nonalcohol specific characteristics as causal to both problem alcohol use and elevated risk for AUD (Caspi *et al*, 1996; Donovan and Jessor, 1985, Nigg *et al.*, 1998). The converse is also true; the nonalcohol specific characteristics among nonCOA children are markers of elevated risk for alcohol problems, alcoholism, and other drug involvement, hence the title of this chapter and the necessary focus on "other high risk groups" (Biglan *et al.*, 2004; Zucker and Gomberg, 1986).

3. Early Development Origins of Risk among COAS

One of the historically most important findings of the past generation has been the documentation of a link between delinquent and aggressive activity in adolescence and earlier onset of alcohol use, as well as more problematic use (Jessor and Jessor, l977; Kandel, l978; Donovan and Jessor, l985; Ellickson et al., 2003). An extensive body of work has documented how these behaviors emerge from a matrix of personality and temperament influences, attitudes, and parental socialization practices and modeling, that encourage the development of independent and rebellious behavior. (Colder & Chassin, 1999; Tarter et al, l985; Tarter & Vanukov, l994;) This in turn produces more exposure to a deviant peer network, which then drives the emergence of earlier and more problem alcohol use (Blackson & Tarter, l994; Blackson et al, l994; Blackson, 1997; Zucker et al., 1995a).

Until recently, only the adolescent version of these linkages had been established. However, within the past decade three prospective studies beginning in early childhood have shown a direct link between the early child manifestations of these attributes, specifically behavioral undercontrol and aggressiveness, and AUD and other alcohol problem outcomes in adolescence and early adulthood (Caspi et al., 1996; Masse and Tremblay, 1997; Zucker et al., 2000; Mayzer et al., 2001, 2002, 2003). These studies join with two earlier reports of projects beginning in middle childhood (Cloninger et al, 1988; Eron et al, 1987) with similar childhood markers at baseline, and with alcoholism and drunk driving outcomes in adulthood. Three of the studies, the Dunedin Health and Development Study (Caspi et al, 1996), the Columbia County Study (Eron et al, 1987), and the Montreal Longitudinal Study (Masse and Tremblay, 1997) involve general population samples, and two involve COA samples (Cloninger et al., 1988; Zucker et al., 2000; Mayzer et al, 2001, 2002). Table 2 describes the ages at baseline and follow-up, and the baseline behaviors and adolescent/adult outcomes of the study. The level of replication shown across these studies must be taken as definitive evidence that an early childhood behavior-adulthood AUD relationship exists. Combined with the adolescent studies noted above, findings indicate that a continuity pathway exists from very early childhood to an alcoholism outcome in adulthood.

Equally importantly, both the COA studies (Cloninger et al, 1988; Mayzer et al, 2002) and the Dunedin study (Caspi et al., 1996) find a behavioral inhibition/shyness/social fearfulness cluster predicted alcoholism and alcohol problem outcomes in adolescence and early adulthood. These latter characteristics have only sporadically been reported in the adolescent literature (Kaplan, 1975) but they are consistent with the known adult relationship between social phobia and AUD (Kushner et al., 1990), and they also have been reported in some historically earlier prospective studies begun in early childhood. Thus Werner (1986), observed a relationship between a low sociability temperament in infancy and early childhood with the greater likelihood of an alcoholic outcome in early adulthood, and Kellam et al, (1980; 1983) observed a relationship

Table 2. Longitudinal Studies Connecting Early Child Behavior to AUD and Alcohol Problem Outcomes in Adolescence and Adulthood

Study	Early Child Behavior	Baseline Age (Yrs)	Follow-Up Age (Yrs)	Outcome Behavior
General Population Studies				
Dunedin Health & Development Study	Behavioral Undercontrol	3	21	Alcohol Dependence
Dunedin Health & Development Study	Behavioral Inhibition	3	21	More Alcohol Problems
Montreal Longitudinal Study	Low Fearfulness; Hyperactivity	6 and 10	11 to 15	Earlier Drunkenness Onset
Columbia County Study	Aggression	8	30	Driving While Intoxicated
COA Studies				
Michigan Longitudinal Study	Externalizing Behavior	3 to 5	12–14	Early drinking Onset
Michigan Longitudinal Study	Internalizing Behavior	3 to 5	12–14	Early drinking Onset
Swedish Adoption Study-2	High Novelty Seeking; Low Harm Avoid	11	27	Alcoholism
Swedish Adoption Study	High Harm Avoid; Low Novelty Seek	11	27	Alcoholism

Note: See text for study citations.

between shyness/social inhibition in 1st grade and greater alcohol and drug use in adolescence.

It is noteworthy that in all of this work, parallel findings are reported out of both the COA and the general population studies, suggesting that it is the risk factor(s) rather than COA status in particular, that is driving these relationships. At the same time, the socialization environment is virtually uncharacterized in most of the studies. Thus it is not possible to determine the degree to which contextual factors may be moderating or mediating the relationship. Moreover, even in the nonCOA samples, one cannot automatically assume a more benign environment. In fact, in two of the general population studies reviewed above, the Montreal Longitudinal Study (Masse and Tremblay, 1997) and the Woodlawn Study (Kellam et al, 1980), the population sampling was deliberately set to provide a group of families of low socioeconomic status and high social adversity. Thus even in the nonCOA studies, the level of environmental adversity may be sufficiently damaging and sufficiently similar to what exists in alcoholic homes to produce the parallel effects.

In terms of relevance of these findings to prevention activity, one final observation is called for: these studies in toto, are potentially a call to arms for preventionists because they provide easily identifiable targets for preventive

programming at an early age. The etiologic data pertaining to behavioral undercontrol clearly indicate continuity of risk over the course of development, and therefore strongly suggest that change in the risk factor should lead to change in the outcome. Interestingly however, although these findings are dramatic and have now been in the literature for between 8 and 17 years, they remain almost totally neglected in the prevention literature. To my knowledge, only one just published policy book (Biglan et al, 2004), a recent report following families from birth to age 18 (Garnier and Stein, 2002), and a very brief summary in the most recent NIAAA Report to Congress (NIAAA, 2000) begin to address the prevention implications they raise. I will return to this issue at the end of the chapter.

4. Heterogeneity of Risk Pathways

In the previous section, I noted that characterization of environmental adversity has been relatively ignored in most of the long term, early-starting high risk studies. This is a significant omission because of the need to understand the potential for environmental adversity to exacerbate individual risk on the one hand, and for its absence to alleviate individual risk on the other. For the same reason, within the nonCOA population it is important to understand the degree to which environmental adversity, or its absence, makes a difference in producing an adverse outcome. Our group has examined this issue using data from an ongoing longitudinal family study of alcoholic men, their spouses, their initially 3 to 5 year old sons, other siblings, and a suitably matched set of contrast families drawn from the same high risk neighborhoods where the alcoholic families lived, but where neither parent had a lifetime diagnosis for any substance use disorder (Zucker et al, 1996; 2000). Families were followed at 3-year intervals beginning when the target boy was 3 to 5 years of age.

We used a person-centered approach in examining the interactive nature of family adversity and child risk vulnerability over the interval between 3 and 14 years of age.[2] The adversity index used was one that assessed level of exposure to a highly pathological family environment. A summative family psychopathology measure was created that scaled both currency and severity of AUD as well as the presence/absence of antisocial behavior in each of the parents, then added them together (cf. Wong et al, 1999; Zucker et al, 2003). High family adversity involved having two parents with currently active alcoholism, or one parent with an antisocial alcoholism diagnosis, or both. This index, although established by way of parental psychopathology, is an effective proxy for a number of other pertinent indicators of family adversity, including conflict, violence, economic difficulty, family crises, other psychiatric comorbidity,

2. Findings described in this section are based on data originally reported in Zucker et al, 2003, and the reader is referred to that source for more precise details of measures and analyses.

and trouble with the law (Zucker et al., 1996). In addition, on the basis of national Epidemiologic Catchment Area Study alcoholism comorbidity rates (Helzer et al, 1991) and national familial alcoholism figures (Grant, 2000; Huang et al, 1998), these cutoff criteria would yield a population encompassing slightly less than 1 percent of U.S. households, but approximately 20 percent of alcoholic families (the severest subset).

The child's initial risk status at age 3 to 5 was described by a global socio-behavioral psychopathology measure that was nationally normed. Low risk was defined as being within normal limits on this global index, high risk was defined as being at the 80th percentile or higher on the measure (0.84 SDs above the norm). A two by two grid was created by cross cutting these dimensions. Initially *Resilient children* were defined by having normal to high adaptation (i.e., low "risk" scores) even though they were living in the high family adversity environment. The normal risk under conditions of low family adversity group was labeled *Non-challenged* to emphasize that their behavior was unremarkable, within a family context involving low parent psychopathology that exerted no pressure for deviance, and that was more likely to be nurturant and encouraging. The high risk (high psychopathology) under conditions of high family adversity group was labeled *Vulnerable,* in order to emphasize the continuing exposure to family trouble that took place here. Other evidence from the study shows that these children had been negatively impacted by this exposure (Wong, et al. 1999). Finally, those children with high risk (high psychopathology) under conditions of low family adversity were characterized as *Troubled* in order to emphasize that, even without the familial adversity, they still showed a poor behavioral adaptation. In other words, they showed up as already symptomatic, even with a lack of environmental press.

Figure 1a shows the trajectory of externalizing problems for each of the groups and Figure 1b shows the trajectory of internalizing problems. Overall across-age group differences were significant for both externalizing and internalizing problem trajectories. The non-challenged group sustained the lowest level of externalizing problems over the course of childhood and early adolescence, followed by the resilient group, the troubled group, and the vulnerable group. At all ages, the vulnerable group sustained the highest level of externalizing problems. The figure also shows a consistent pattern of decline in externalizing behavior over childhood, a pattern that is normative for this age range. In addition, there is increasing convergence in level of externalizing difficulties through middle childhood. At the transition to adolescence we again see the normative pattern of a developmental shift, involving increasing externalizing (aggressive/delinquent/impulsive) behavior (cf. Jessor and Jessor, 1977). The individual difference data indicate that whereas the resilient children were not distinguishable from their non-challenged peers as pre-schoolers, they showed a small but reliably higher level of externalizing problems as they grew older. At the same time, they still occupied an intermediate place, having a lower level of these behaviors than did their vulnerable peers. In addition, the divergence of slopes between ages 9 to 11 and 12 to 14 depicts a

significant interaction between child individual differences in initial risk and level of experienced environmental adversity during a period of life when the overall norm is for increasing deviant and impulsive activity. This interactional

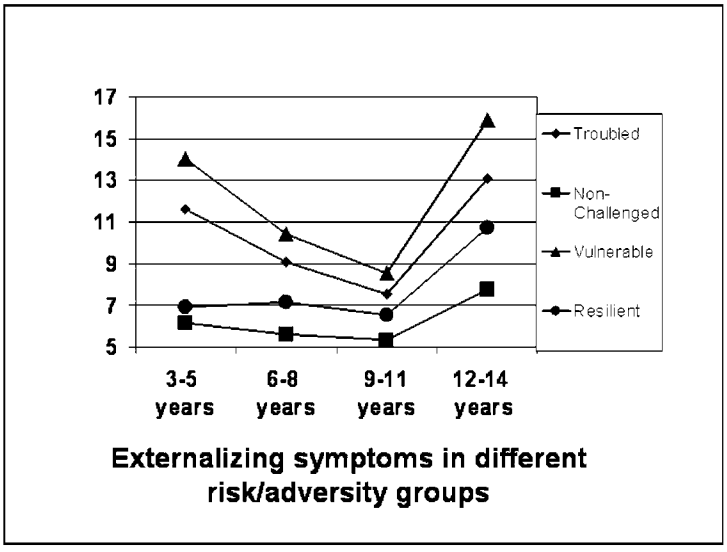
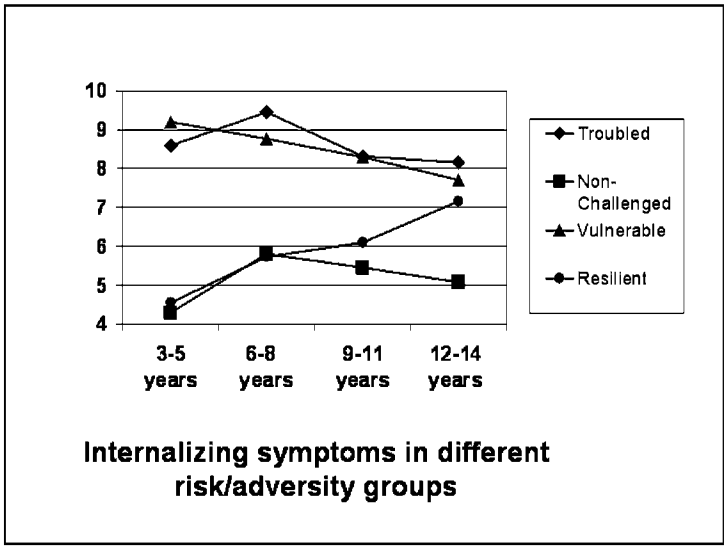


Figure 1a. Externalizing symptoms over time in groups differing on risk and adversity. (*Source:* p. 88 in Zucker, R.A., Wong, M.M., Puttler, L. I. and Fitzgerald, H.E. (2003). Resilience and vulnerability among sons of alcoholics: Relationship to developmental outcomes between early childhood and adolescence. In S. Luthar (Ed.), *Resilience and Vulnerability: Adaptation in the Context of Childhood Adversities.* Cambridge University Press, New York. Reprinted with permission.)

relationship had previously been observed cross-sectionally among these children when they were 3 to 5 years of age (Wong et al., 1999). The trajectory data indicate the pattern is sustained developmentally; they depict continuity over time in group positioning vis a vis level of undercontrolled behavior, and the positioning is sustained across the risk-adversity groups even though level of group differentiation varies, as does absolute level of undercontrolled activity.

Figure 1b shows the trajectories for internalizing problems; here also the non-challenged group shows the lowest level of problems, followed by the resilient group. The troubled group was similar to the vulnerable group. The figure also shows important pattern variations. During preschool and up through the early school years, an identical individual difference pattern exists. Non-challenged and resilient children are significantly lower in internalizing symptoms than both the vulnerable and troubled groups, and there are no differences between the resilient and the non-challenged children. The pattern begins to diverge following 2nd-3rd grade, and by early adolescence the non-challenged group is significantly lower than all others, and no differences exist

between any of the other three groups. In other words, at this juncture the resilient children have developed a level of internalizing symptoms that is similar to both the vulnerable and the troubled children. Here also we tested this group by time interaction with a repeated measures analysis of variance. A significant interaction effect of time and adaptation group indicates that the developmental trajectories of internalizing problems varied differently among



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the adaptation groups, with three of the four showing a continuity pattern, and one a discontinuity pattern.

These patterns of trajectory variation in both externalizing and internalizing problems are more than simply patterns of risk variation over time. As already noted in Table 2, they also are proxies for differences in probability of problem drinking, other problem behavior, and also alcohol dependence (Ellickson et al, 2003; Grant and Dawson, 1997; Pederson and Skrondal, 1998). In the Michigan study, Mayzer and colleagues (2000, 2002, 2003) have already confirmed the first step in this chain of effect, by showing that higher levels of early externalizing and internalizing behavior are predictive of both early onset of drinking, as well as higher levels of externalizing and internalizing behavior and delinquent activity in adolescence. On both these grounds, the results indicate that the children identified as vulnerable are at highest risk, the

non-challenged group is of lowest risk, and the resilient group is of intermediary risk, in particular because of the increasing experience of internalizing problems as they move into adolescence.

Finally, it is instructive to remind ourselves who the COAs are in this matrix of individual and contextual risk. They are the youth labeled as Vulnerable and Resilient, the children who were born into, and reared in families with high alcoholism density and high parental antisocial comorbidity. Conversely, the nonCOAs are comprised of the Non-Challenged and the Troubled groups yet they have strikingly different pathways of risk. Given what has already been established about the utility of the externalizing and internalizing behavior measures as proxy indicators of alcohol problems and elevated risk for later AUD, these findings make clear that an understanding of both familial risk and individual risk is essential to an understanding of pathways into problem alcohol use (also see Garnier and Stein, 2002). When individual vulnerability is present early, even a nonchallenging family environment is insufficient to moderate the child's vulnerability. Conversely, from the perspective of risk for externalizing problems, a subset of COAs moves through childhood relatively trouble free, while another subset, showing early risk, is the highest risk subgroup.

This pattern is tempered to a considerable degree for internalizing risk. For one subgroup of young COAs, their early behavior indicates they are relatively free of sadness, anxiety, depression and worry. Exposure to the adversity of an actively alcoholic home, with its attendant strain and conflict (Loukas et al, 2003) leads to a gradual degradation of their affective status, such that by the time adolescence is reached, their level of internal trouble is equivalent to that of their more obviously less fortunate peers.

5. The Timing and Dosing of Prevention Programming: Toward a Hypothetic-Deduction Science of Prevention

A science of timing and dosing for prevention activity does not yet exist. Earlier is perhaps better, but earlier is more expensive, and effects delivered early, if not sustained by boosters, have the potential to decay over time. The variations in externalizing and internalizing trajectories documented above suggest some interesting, and to our knowledge previously undescribed preventive intervention strategies. They also suggest some interesting hypotheses. The trajectory data indicate that the critical timing points for intervention for externalizing and internalizing behaviors may be different. For externalizing problems, despite the variation in level over time, grouping based on early risk and early family adversity holds its order. At the same time, the later elementary school years appear to be the point of greatest subsidence of these risky characteristics. If the hypothesis is that the best way-point to intervene is when the problem behavior is most quiescent, then late elementary school would be the timing point of choice. If the hypothesis is that the point of greatest impact will be when the problem behavior is most active, because there is more to

engage with and potentially change, then an early childhood start would appear to be the timing point of choice. These two alternative strategies need to be pitted against each other and evaluated.

For internalizing problems, for three of the four risk/adversity groups a pattern of continuity exists from early childhood onward, with an essentially flat trajectory over that interval. For the resilient group however, the point of greatest quiescence is either preschool or the earlier elementary school years. "Quiescence" theory does not provide a clear choice about whether it would be more efficacious to begin early, or to begin around the time when higher levels of internalizing problems begin to manifest themselves. One might speculate that an intervention in this content arena that is done too early might have little effect. Conversely, if one's choice is driven by "Activation" theory, then an intervention at the latter part of elementary school, or even at the transition to middle school or junior high might be the most appropriate intervention point. Again, these alternatives should be evaluated.

What about dosing? For vulnerable and troubled children, the long term presence of both sets of risk factors, at the highest levels vis a vis the other groups, points to the need for a multilevel intervention regimen that is based upon a chronic disease model (McLellan et al, 2000). Such programming would involve initial evaluation and dosing, addressed both to the child's difficulties as well as the difficulties of the family in which he/she is grouping up. Periodic check-ups, that provide an opportunity for renewed intervention when called for, would be a part of such a regimen. It would also be expected that such programming be available over a substantial portion of the childhood life course, although not necessarily required at all times. For resilient children, it is not at all clear how long such a developmentally timed intervention would be required. The presence of other coping skills in this group has already been documented (Zucker et al, 2003). These skills (e.g. reading) are suggestive that the intervention would be facilitated by the child's own orthogenic competence, and that the dosing would not need to be as prolonged.

One last point: With the exception of the change in internalizing symptoms among the low child risk/high family adversity resilient children, the mean trajectory patterns are stable, and remain in the same risk rank ordering over time. This is the case as much for the "off-diagonal" groups—where one might anticipate that individual risk-variability and social environmental stress would work at cross purposes, as it is for the "on-diagonal" groups. Although the study design was never set up to evaluate the relative role of environmental and genetic influences, we have elsewhere suggested that the strong auto-stability of risk is consistent with the hypothesis of a substantial genetic contribution to risk (Fuller et al., 2003). Should this ultimately prove to be the case, then it would open the door to considering more physiological and pharmacologic methods for risk reduction.

6. Current Prevention Strategies

In 1994 the Institute of Medicine proposed a revised set of definitions of prevention programming. Refining the earlier distinction between Primary, Secondary, and Tertiary Prevention, new categories of activity were again proposed at three levels. *Universal prevention activity* targets entire populations, and involves working with a group that has not been identified on the basis of individual risk. *Selective intervention* targets individuals or subgroups whose risk is known to be higher than the population at large, but where the disorder or problem has not yet manifested itself. *Indicated preventive intervention* targets individuals who have already shown prodromal signs or symptoms, but who do not yet meet diagnostic criteria (Institute of Medicine, 1994).

Given the evidence just reviewed that indicates risky behaviors prodromal to AUD can be identified in COA and other high risk populations at a very early age, one might conclude that the data are sufficient to require a level of programming that is at the least at the Selective level, and perhaps even the Indicated level for most COA and other designated high risk populations. Moreover, the sheer magnitude of the at risk population, involve 15 percent of children under 17, suggests that preventive programming targeted at COAs should be regarded as a major public health effort. Interestingly, it is not.

In the literature review conducted for this report, I was able to identify only two selective programs focusing on COAs, that have been (or are in the process of being) subjected to the rigorous evaluation of the randomized clinical trial. The first, carried out by the author and colleagues in the late '80s and early '90s (Maguin et al, 1994; Nye et al., 1999; Zucker and Noll, 1987) used a population based recruitment protocol to recruit families with active alcoholism in the father at the time of first contact. The project used a manualized 10 month parent training and marital problem solving protocol modeled after Patterson et al.'s (1975) social learning therapy. The child focus was reduction in conduct problems and development of prosocial behaviors, a focus that was theoretically selected as a precursive pathway to later alcoholism risk. At end of treatment and 6 month follow-up, as predicted, positive changes in child behavior and parenting style occurred. Unfortunately, later evaluation of potential drinking offsets were not conducted. The second program, a joint U.S./Canadian program still in progress, is being conducted by Nochajski, DeWit, and colleagues at the Research Institute on Addictions in Buffalo and the Centre for Addiction and Mental Health in London, Ontario. The intervention makes use of Kumpfer's (1998) Strengthening Families Program (SFP) and enrolls families with an alcohol abusing parent and their school aged children. The intervention involves a 14 session group therapy program that combines parent training with family communication skills training and child social skills training. Results to date show that as compared to a minimal attention control group, SFP produced significant improvements in child externalizing behavior problems (Maguin, et al., 2003) and family functioning (Safyer, et al., 2003). A longer term delay of onset of alcohol use, as well as

reduction in alcohol problems among the children is eventually anticipated, but the study has not been running long enough to determine whether this effect occurs. Nonetheless, the robustness of externalizing problems as an early proxy for alcohol problem outcomes is such that the anticipated outcome is a highly plausible one.

One other selective early intervention program, while not specifically alcohol focused, also has shown long term alcohol prevention effects; it is David Olds' Nurse Visitation Program. The program initially involved home visits with high risk, high poverty, primarily teen mothers during pregnancy; reassessments were at 2 years, and again at a 15 year follow-up. At the 15 year follow-up, the visited group of mothers reported fewer days drinking, and fewer cigarettes smoked per day in the prior six months than did their no treatment control group (Olds et al., 1998).

The programs just described involve families whose parents are from clinical or quasi-clinical populations, where level of risk for problem alcohol outcomes among offspring is substantially elevated. However, several hundred universal programs focusing on delay of substance use, or alternatively, delay or reductions in delinquent behavior, have been carried out, and a number of those have demonstrated specific alcohol related prevention effects (Biglan et al, 2004). Programs have rarely been simply child focused, but rather have chosen interventions to address *systems interacting with the child* (parent behavior, family interactions and relationships*), systems interacting around the child* (teacher training and curriculum development, parent training, working with the courts and legal system), *systems acting at the community level* (community action programs, changes in the rigorousness of enforcement of alcohol and cigarette access, and *systems addressing policy* (establishment of drug courts, zero tolerance drivers' license programs, changes in pricing, etc.). Although there is a plethora of such programming, relatively few protocols have been subjected to rigorous process and outcome evaluation. The reader is referred to a recent comprehensive review of this spectrum of offerings by Biglan and colleagues (2004) for detailed descriptions and evaluations of the most rigorous of these programs. Following, we briefly describe four of the most comprehensive, that have been rigorously developed and carefully evaluated:

(1) A truncated (7 session) version of the Strengthening Families Program has been used as a universal prevention program involving initially 6th grade (ca. age 12) children from rural elementary schools in Iowa. The program is of special interest because of its promise to ultimately impact new cases of AUD as program participants grow into adulthood. (The study also evaluated another program, Preparing for the Drug Free Years (PDFY), a 5 session family competency program based on the Social Development Model (Hawkins et al, 1999), but since PDFY effects were always weaker than the SFP arm of the study, only findings from the SFP protocol are discussed here.) Remarkably strong improvements both in parenting skills and in family relations (Kumpfer, Alvarado, Tait, Turner and Alder, 2002) were demonstrated both 4 years out and 6 years out from the intervention (Spoth, Redmond and

Chin, 2001; Spoth, 2003). More importantly from the standpoint of this chapter, the program successfully delayed onset of alcohol use as well as dampened increases in level of consumption over time (Guyll et al, in press; Spoth, 2003), and also slowed the rate of initiation into tobacco, marijuana, and other illicit drug use. Benefit-cost calculations relating to projected rates of AUD prevented indicated a return of $9.60 per dollar invested, and net benefits per family of $5,923 (Spoth et al, 2002).

Other preventive intervention programs, all universal in focus, have been able to demonstrate impact upon early drinking behavior during adolescence, although none have evaluated their ultimate impact on a later AUD outcome. Several are noteworthy for their comprehensive focus on individual, school and community, their relatively early initial contact with the child, and/ or their impressive impact on alcohol related behaviors.

(2) Project Northland (Perry et al, 1996) is currently the only specifically alcohol focused program addressing the distal domains as well as the child's micro-social environmental domains, as part of a unified effort to delay onset of use as well as reduce problems once drinking has begun. Results have included significantly lower prevalence of alcohol use after three years of intervention, with strongest effects among those who were nonusers at baseline. The magnitude of effects in this program was small, but because of the low initial base rates of drinking at younger ages, the comparative effects were substantial. Looking only at students who were nondrinkers at the 6th grade baseline, 15.3 % in the treatment sites at 8th grade follow-up had past month alcohol use, while 21.2 percent had use in the control sites. The protocol also showed effects in reducing marijuana use (3.1 vs. 6.2 percent) and cigarette use (15.5 vs. 24.6 percent). All of these effects were confined to the baseline nondrinker group. No significant changes were found among those who had already begun drinking. This work was not able to parse out the reasons for these differential effects on initial nonusers vs. users, but youth who are already using at 6th grade are very much an early onset group, given that median age of onset of first use is 14. Given also what is known about the impulsivity, heavier drinking of parents, and conflicted family backgrounds of early onset users (Ellickson et al, 2003; Mayzer et al, 2001, 2002, 2003) it is likely that the social micronetworks within which the early onset drinkers moved would have insulated them to a greater degree from program effects. Effects decayed after the intervention was no longer active (Perry et al, 1998, reported in Wagenaar, 2000).

(3) In another long term and very comprehensive universal prevention program, the Seattle Social Development Project (Hawkins et al, 1992) targeted a high risk community sample in a program that involved individual, school/teacher, and family interventions. The program emphasized the creation and maintenance of strong family and school bonds, and also had a component focused on cognitive and social skills training in the early school years and refusal and life skills training in late elementary school. One subset of youngsters received all levels of the program (the Full Intervention Group), a second Late Intervention Group received only the later programming, and a

third subset was a No Intervention Control. Long term follow up at age 18 showed a number of differences for the full intervention group on school attachment and achievement, no differences on lifetime prevalence of alcohol use, cigarettes or other drug use, but reduced past year heavy alcohol use at this point. In other words, the problem level of alcohol use was impacted downstream but overall use was not (Hawkins et al, 1999). The late-dosing-only group did not show this effect.

(4) Another universal program, with its point of entree and raison d'etre being the reduction of bullying and related problems was implemented by Olweus (1989) in a national program conducted in Norway in the mid'80s. The evaluation for this work utilized a quasi-experimental design, and a subset of students initially in grades 4 to 7 in a large number of elementary and middle schools in Bergen, Norway in 1984–1985 to conduct the evaluation. This extraordinarily comprehensive program incorporated a number of levels: first a questionnaire to increase awareness as well as gauge severity, then feedback to the schools and discussion, then setting up structures to monitor level of the problem and effectiveness of solutions at the school and classroom levels, conduct of classroom discussions, as well as individual discussion with perpetrators victims, and their parents, etc. The program produced significant reductions in antisocial/delinquent activity, as well as drunkenness as far out as two years from baseline. This is but one of a host of examples in the universal focus literature where a focus on the undercontrolled aspects of behavior also has an effect on drinking.

The Olweus program involved a large number of individuals and groups in a multi-tiered framework of interventions involving interwoven, "across level" relationships. Moreover, the rule structure legislating this prevention activity was at the political, community leader, as well as the educational policy levels, given that the program was community wide (in actuality, the entire project, not all of which was subject to formal evaluation, was the country of Norway), and the educational system had agreed to modify itself, by conducting all day conferences, changing monitoring practices for the bullying behavior in and outside of class, setting up coordinating committees, etc.

One final note: Population generalizability for those treated (i.e., those participating in a program) against those eligible is not well documented in existing studies. However, the available data across studies indicates that the selectivity of who is being treated vis a vis who is high risk is a potentially significant problem given that programs routinely have very high initial nonparticipation rates. Thus, the Iowa program reported a 51% completion rate of the baseline assessment among all 6th graders in the schools they recruited from (Spoth et al, 2001), the New York/ Ontario program reports a 70% treatment entry rate for those who completed the baseline assessment (rate of involvement in the baseline assessment vis a vis the population of those eligible was not available) , and participation rates more generally in indicated as well as universal programs have hovered around the 60 to 70 % range (Tremblay et al, 1995; Dishion et al, 2002) . Much like the Project Northland program issue of

only having impact on initial nondrinkers, this is a critical policy concern if one's interest is in addressing risk among the most disadvantaged. It is such families, with high rates of family disorganization and lack of child involvement that are the ones most likely to not initially engage (Ary et al, 1999; Chilcoat et al, 1996). Some researchers have attempted to demonstrate that the problem is a minor one (e.g., Spoth, 2003) because the limited contrast data comparing participants to nonparticipators suggests minimal differences. However, the problem is a difficult one to address because nonparticipators do not provide the same level of descriptive data as do the enrollees. The jury must still remain out on this issue.

7. Unresolved Issues and Next Steps

Two questions persist as meta-issues in this review. We address each in turn, with some observations about how the issues might be resolved.

(1) Why are so few selective programs focused on the COA population, given what is known about short and long term risk?

There are two issues here: The problem of preventing AUD in a nation where 32 percent of its men and 15 percent of its women will at some point in their lives make the diagnosis (Kessler et al, 1997) is a problem that initially requires placing a diagnostic label on the activity. In so doing, the potential to produce shame and stigmatization is a much larger one than would be the case if prevalence were confined to only 5 percent of the population. Grouping the problem with the abuse of other drugs, behavior that is more clearly regarded as negative, and including it as part of a larger category rather than giving it its own name effectively diffuses the issue of what is actively being prevented. This is at least part of the difficulty.

The second issue is that from a public health standpoint, the prevention of instances of abuse, single events, rather than the prevention of diagnoses (changing the behavior of individuals) , is a more effective strategy because the total of problem events created by persons without AUD is greater than the total of those with it. Thus, in terms of solving health problem issues at the community level, it is more cost effective to work at preventing the single events, which moves the discourse away from AUD.

(2) Why have not the clearly replicated findings of the predictability of AUD risk entered the mainstream of early identification and prevention programming? The findings remain largely unknown to alcohol researchers, they have not been disseminated to health educators, and family practice and pediatric physicians are also largely unacquainted with them.

Undoubtedly part of the explanation of why it has been difficult to make this work visible is the same as what has just been described; it is the problem that labeling creates shame and stigma. There is also another, more practical reason. It is extraordinarily difficult to face a painful experience and not be able to remediate it. Knowing that a youngster is at high risk for later AUD without

having any way to address the problem is to create a considerable amount of pain and despair in the observer. This is a part of the dilemma that any health professional or educator must grapple with in attempting to assimilate this new knowledge. If programming can be created that provides some effective plan of action, it is reasonable to expect that this resistance to understanding will dissipate.

What are some of the barriers, and how might they be resolved? In an era of managed care, escalating health costs, and carve out medical plans that provide little reimbursement for behavioral health, it is utopian to believe that any new long term identification and treatment program would be embraced by the health care system. This is especially so for a condition such as alcoholism, which is realistically regarded as a chronic and recurring disorder (McLellan et al., 2000). In contrast, identification (and treatment) have more likelihood of being sustained if they are piggybacked onto an already existing and compensated program. There are currently a number of venues where such a plan would be feasible: regular check up time in a managed care pediatric or family medicine program would be one readily accessible access point for screening, and possibly also for brief intervention programming if it were not too costly. Another point of access would be screening at pediatric emergency medicine facilities. Impulsive sensation seeking and aggressiveness are both markers of high AUD risk. They also are more likely to get the youngster into the Emergency Department. A third would involve family contact and brief family screening for all adults who come in for outpatient alcoholism or other drug treatment. A child and family focused brief intervention package would be simple to implement once agency staff were accepting of such a new, extended family model of treatment. Furthermore, the health care context is one that routinely expects repeat checkups and follow through. This would permit a program of booster sessions, on an as needed bases. There are undoubtedly other natural settings where such a spin off assessment and brief treatment could be carried out.

8. Epilogue

In a 1997 review and critique of prevention efforts for substance abuse programs, the eminent developmental psychologist and initiator of Head Start, Edward Zigler, and his colleague Nancy Hall observed the following:

> "Thirty years of research findings indicate that the most promising intervention/prevention efforts are likely to be those that are truly ecological in nature—programs that target children within the context of families (e.g., two generation programs such as Head Start. . . . and that address children and families within the context of their communities. . . .
>
> Myriad attempts to inoculate children against later substance abuse. . . . have sprung up in direct response to current policy mandates. If these ini-

> tiatives are to make inroads in the nation's battle against drug abuse, however, the next wave of such programs must reflect greater understanding of the knowledge base with respect to the development and socialization of young children, the onset of delinquent behavior, and the importance of implementing and applying both process and outcome evaluations" (Hall & Zigler, 1997, p. 141).

These observations seem are as true today as they were in 1997. Contextually based interventions still show the greatest promise, and a number of them have been evaluated and shown to be efficacious. But the field is still in its infancy in conceptually and practically addressing the problems of COAs and other high risk populations before they become manifest. Alcohol is the nation's most common drug of abuse, but those children who have the greatest potential to abuse it still remain a hidden and untended population. The technology and the knowledge base now exists to remedy that situation.

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IV

Treatment

Cherry Lowman, *Section Editor*

The purpose of the treatment section is to highlight several emerging trends in treatment research on adolescent alcohol use disorders. In 1997, the NIAAA initiated a program of adolescent treatment research. Since then, 20 clinical projects have been funded, the majority of which are clinical trials. Fifteen of these are behavioral projects and three are pharmacotherapy projects. These are the first controlled, manualized, and randomized studies to specifically assess the efficacy of interventions for the treatment of alcohol use disorders in adolescents. The objective of this initial wave of studies is to design and test innovative developmentally tailored interventions that provide evidence-based knowledge to improve treatment outcomes in adolescents.

Results for most of these projects will be forthcoming over the next few years, and will yield a broad perspective on the potential efficacy of family-based, cognitive behavioral, brief motivational, and guided self-change interventions in a range of settings and subgroups of adolescents, including homeless and runaway youth, high school students, juvenile justice-involved youth, and minority youth. In the meantime, new emphases are beginning to emerge in adolescent treatment research related to what research questions are important to pursue next. A research approach is emerging which unifies developmental and transdisciplinary perspectives on the etiology, development, and course of substance abuse disorders in order to better understand alcohol effects in youth, and ultimately to use this knowledge to design more effective interventions for youth.[1,2,3]

In most adolescent alcohol research treatment studies, developmental criteria have been limited to age and grade as indicators of position along the developmental continuum. There is now a nascent trend to adopt more devel-

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opmentally specific models and methods from developmental psychology, developmental psychopathology, and developmental neuropsychology as a means to improve design and outcomes of adolescent treatment interventions. The translational approach to research in the health sciences represents a major paradigm shift in the way research is conducted, one supported by the U.S. National Institutes of Health. The aim of this approach is to solve major public health problems by bringing together scientists from relevant disciplines in the basic, clinical, and social sciences to develop transdisciplinary, integrated theoretical models and interventions based upon them that can resolve the target problem.[4,5,6,7] The authors who have contributed chapters to the treatment section provide both direct and indirect empirical evidence of this emerging research approach.[8,9,10,11,12]

Brown and colleagues[8] examine the complex interactions across and changes over time in four major domains of functioning during adolescent development. These include biological (puberty, neurological development), socioemotional (family influence, emotionality, intimate relations), cognitive (executive functioning, spatial operations, and attention), and behavioral (self-regulation and risk management) domains. Adolescent long-term risk pathways (i.e., trajectories) for alcohol use appear to be influenced by these factors, particularly developmental dysregulation and family- and experience-based psychopathologies. The authors distinguish three pathways of risk for underage alcohol use and disorders—normative risk, personality/temperament risk, and psychopathological risk—and illustrate each of these with empirical data. They also discuss the long-term as well as acute health consequences of adolescent alcohol use and how these along with developmental stage need to be taken into consideration in the design of treatment outcome studies. Guidelines to development of substance abuse interventions for adolescents are provided, and a number of evidence-based adolescent treatments are reviewed. In addition, the authors recommend that developmental and environmental specificity be assessed by including variables which represent environmental constraints on alcohol consumption, developmental milestones and transitions (and delays in these), age-normed neurocognitive functioning, family functioning, and job performance.

The authors conclude with a summary of alcohol treatment outcome evaluations from the perspective of the four domains of development considered in this chapter. They note, for example, that different domains of functioning post treatment have been observed to improve at different rates and therefore, to adequately assess treatment effectiveness, evaluation needs to be timed such that all salient improvements are assessed. Evaluation also needs to take into account the reciprocal influences between positive change in one domain and positive changes in other interdependent domains. This discussion of outcome evaluation from a longitudinal, developmental perspective should be invaluable not only to those planning future research in this area but also to inform clinicians, educators, and parents about the nature, interdepend-

ence, and sources of change in long-term adolescent developmental pathways related to alcohol and drug disorder treatment outcomes.

Winters and Kahnhorst provide an overview of assessment issues in adolescent substance abuse research from a developmental perspective.[9] They discuss the importance of early assessment of alcohol and other drug (AOD) use in order to distinguish normative from problematic use. They also discuss barriers to early assessment, indicators of progression in use (e.g., age of onset, regular use, polydrug use), and issues related to valid diagnosis of alcohol abuse and dependence in adolescents whose use patterns and consequences of use often vary from those of adults. Also emphasized is the importance of identifying comorbid psychiatric disorders, which may contribute to AOD relapse as may emotional dysregulation which can occur during this developmental stage.

The emerging human developmental and translational research trends are augmented by increasing emphasis on the importance of evaluating and treating psychiatric comorbidity and polydrug use associated with alcohol and drug use disorders. Longitudinal developmental research has shown that severe adolescent alcohol disorders have been, in the majority of cases, chronologically preceded by psychiatric and other disorders or symptomology and are often associated with multiple concurrent substance use disorders.[2,3,13,14] It has become increasingly clear that effective treatment for adolescents with advanced alcohol use disorders will require a multifaceted and possibly transdisciplinary treatment approach.

The chapter contributed by Cornelius[10] and colleagues reviews state-of-the-art approaches to treating comorbid adolescents with an emphasis on medications, knowledge gaps, and future research needs. Prescription of medications for substance use comorbidities has been increasing over the past ten years despite an absence of evidence-based knowledge on their safety, side effects, and efficacy in this population. To address this important medical issue, the authors provide a useful review (see also Dawes and Johnson)[15] of potential pharmacological approaches to treating concurrent alcohol use and other psychiatric disorders including major depression, bipolar disorder, anxiety disorders, conduct disorder, and attention deficit hyperactivity disorder. The authors stress that this area of research is in its infancy and needs to begin with the basics, including conduct of safety and sequencing studies followed by double-blind, placebo-controlled pharmacotherapy trials to establish long term efficacy and optimal combinations of pharmacotherapies and behavioral therapies in comorbid youth.

Another emerging emphasis involves efforts to deconstruct complex treatment processes in order to better understand and evaluate the mechanisms of positive change associated with particular components. Even brief interventions are sufficiently complex that their mechanisms of action are not yet fully understood.[16] Once achieved, this knowledge can be utilized to customize, combine, and sequence treatment components such that they meet the

specific needs of youth as identified through both developmental and environmental assessments.

Kaminer and Slesnick[11] discuss the varied and complex nature of cognitive behavioral therapies, interventions based on classical and operant conditioning models, and social learning models. This has resulted in the creation of distinctive cognitive behavioral treatments (CBT)—integrated multicomponent strategies which focus on unique aspects of substance abuse. Among the active ingredients of CBT identified to date in adolescent studies are training in coping skills, problem solving skills, identification of high-risk situations, and role playing. The authors acknowledge that establishing the effectiveness of cognitive behavioral therapies is challenged by lack of comparability across clinical trials. Despite the analytic challenge, they note significantly more rapid overall response of subjects to CBT as a whole in early weeks of a clinical trial as compared with other credible psychotherapies.

The different types of family therapy discussed by Kaminer and Slesnick reveal similar issues in comparing treatment results across family intervention clinical trials. CBT and family therapies not only lack a standard battery of outcome assessment instruments, they both comprise complexes of interventions, particularly the ecologically focused multisystemic and multidimensional family therapies, which include community components in the treatment as well. Clearly needed is treatment process research that has as its aims (1) the parsing of specific treatment components and evaluation of the processes that underly their independent effects on treatment outcomes and (2) discrimination of unspecified treatment effects (e.g., assessment effects in the placebo group) and evaluation of their overall contributions to treatment outcome.

Godley and White[12] provide in their chapter a comprehensive overview of youth substance abuse treatment service systems and report current data on the number and distribution of adolescents receiving treatment for alcohol and drug use in both public and private programs. Included in their discussion is a summary of the current status of existing adolescent evidence-based substance abuse treatments. The authors also discuss the need for aftercare services to maintain treatment gains during recovery.

The need for post-treatment continuing care introduces the final emerging research trend to be discussed in this introduction to the treatment section—the extension of the chronic model of alcohol use disorders to a subset of adolescent substance abusers. For most adolescent drinkers, alcohol-related problems are likely to be transient and to resolve with maturation. But for those adolescents most likely to be seen in substance abuse treatment settings, alcohol-related problems can be chronic in nature. The chronic model is based on the recognition that recovery from addiction to substance use may be a long and complex biopsychosocial process during which some adolescents in recovery may need further intervention to achieve long-term sobriety.[17,18] The authors report that nearly 75% of adolescents treated for marijuana abuse/dependence in clinical trials conducted in five outpatient settings

reported having experienced multiple treatment episodes, either before or after the current treatment episode.

Godley and White discuss the need for post-treatment interventions to address the longer-term recovery process in which recovery and relapse to alcohol use and related problems are "precariously balanced." Among the stabilizing post-treatment interventions which the authors recommend are formalized programs of continuing care such as those that include proactive linkages to youth-specific recovery groups. The authors also provide evidence for the effectiveness of assertive continuing care services that give responsibility for maintaining contact with aftercare services (e.g., monitoring, support, recovery education, re-intervention) to the treatment professional (for example, by telephone or home visits) rather than to the client.[19] In addition, Godley and White point to the importance of developing environmental interventions to reduce adolescents' risks of relapse, often attributable to peer or familial influences.

In sum, a number of new emphases and trends characterize emerging research related to improving the effectiveness of treatments for youth with alcohol use disorders. This emerging research includes the adoption and integration of human developmental and transdisciplinary research perspectives and methodologies.[8] Adoption of a transdisciplinary human developmental framework in epidemiologic and natural history studies can be expected to yield salient and specific knowledge on the origins and causes of alcohol abuse and dependence in youth, and on variations in the nature of associated biopsychosocial problems in this subgroup. To achieve these results, it will be critical to develop core batteries of instruments tailored to a developmental perspective.[8,9] Adoption of this approach in research to develop more effective prevention and treatment interventions should improve ability to match treatments to developmental subtypes of adolescents. Another emerging research area focuses on testing the effectiveness of pharmacotherapies in subtypes of youth characterized by comorbid alcohol use and psychiatric disorders.[10] Yet another emerging research target is to identify mechanisms of positive change in complex interventions in order to better guide improvement in treatment effects through customization, combination, and sequencing of treatment components.[11] The final emerging area discussed here is development of post-treatment interventions designed to maintain treatment gains during the recovery phase by providing continuing care monitoring and services.[12] Overall, research findings arising from these new directions in youth treatment research could provide even more developmentally sensitive and specific interventions with associated gains in both short- and long-term treatment outcomes.

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