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Anticipating problem alcohol use developmentally from childhood into middle adulthood: What have we learned?

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Abstract

This commentary reviews and comments on six major longitudinal studies from the United States, Great Britain, and Finland, that test predictive models of drinking and problem drinking behavior across a developmental span of one to two generations. The large Ns, in two instances involving population samples, and the broad and study-overlapping variable domains make this collection of studies unique and of special interest vis a vis the issue of cross-study replicability of findings. Significant cross study commonalities are noted, involving the strong cross-study replicability of an undercontrol/externalizing domain as both a childhood and adolescent predictor of problem drinking outcomes in early through middle adulthood, the relative autostability of heavy and problem use of alcohol over intervals of time as long as a generation, the utility of early drinking behavior as an index for later drinking outcomes, the relative parallelism (with some exceptions) of male and female findings, albeit with greater predictability of male over female drinking outcomes and the relatively tighter relational networks of drinking and other behavioral characteristics for males. This impressive group of quasi replications also points the field to address several next step questions, including (a) the need to parse the undercontrol/externalizing domain to identify those subcomponential process characteristics that are causal to heavy and problem drinking outcomes; (b) the need to develop models that will more effectively handle the uneven relationships of negative activity to drinking outcomes, in some instances operating protectively, in other instances operating as risk factors; (c) the need for more carefully articulated, theoretically driven process models that will specify the ordering, developmental saliency, and mediational properties of risk and protective factors as they come on line; and (d) the need for more developmental testing of trait/context interaction models of problem drinking development.

Pervasive drinking by youth in the United States (and in many other countries around the world), and the highly prevalent emergence of alcohol misuse and dependence in late adolescence, are inextricably connected with developmental processes. A developmental perspective is essential, therefore, for fully elucidating the scientific basis of these phenomena and for successfully preventing and treating the causes, problems, and consequences associated with excessive alcohol consumption [1].

The authors in this supplemental issue have more than taken this US National Institute on Alcohol Abuse and Alcoholism (NIAAA) admonition to heart; their work, involving some of the most carefully constructed and longest term longitudinal studies of alcohol involvement now current, has been a significant part of the body of research that led the NIAAA to its present developmental emphasis. The studies reported here extend their earlier work well into adulthood, and the alcohol involvement endpoints they use map onto the dependent variables used by many researchers studying adult clinical populations. Therefore they allow us to ask an essential developmental question: Do childhood predictors account for adulthood variation, even out as far as middle adulthood? If so, are the adolescent/young adult indicators then simply

upstream manifestations of these structures, or are they necessary mediators of what was in place earlier?

There is not perfect cross-study correspondence among variables here. Not all studies were originally designed as substance use studies, and thus while useful for the purposes of this supplemental issue, they vary in measures, time frames, etc. At the same time, there is enough content overlap here to examine which findings replicate across studies. After a brief comment about some of the important attributes of these studies, I examine the degree to which there is across-study correspondence in findings vis a vis four questions:

1. How consistent is the evidence for an externalizing pathway to later alcohol involvement?
2. Is there any consistent evidence for other risk pathways?
3. To what extent are there gender differences in these relationships?
4. What is missing and where do we go from here?

The studies[1]

Four of the six studies were carried out in the United States (the CCLS in New York State, the MSRA in Minnesota, MSALT in southeast Michigan near Detroit, and MTF involving a national sample), one in Great Britain, also a national sample (the NCDS), and one in a single community in Finland (FJYLS). With the exceptions of the MTF which began at age 18 and the MSRA and NCDS which began at birth, the three remaining projects had their first measurement point in the pre-teen school years, and all of the studies had endpoint measurements of drinking outcomes well into adulthood (ranging from age 28 at the low end (MSRA) to age 48 (CCLS). Follow-up intervals between first and last measurement points ranged across the studies from 16 to 40 years, a very long time. The developmental scope of this work is currently unparalleled in the existing longitudinal drinking literature. Variation of population level of consumption would be desirable across the studies because consumption level to some degree drives problem drinking rates [8,9]. If alcohol consumption varies, it would allow some judgment about the robustness of the predictor relationships across varying drinking environments. In fact, such variation does exist; Great Britain has the largest per capita ethanol consumption among the countries and U.S. regions represented (11.8 l/person), and New York residents (i.e., CCLS) the lowest, with a consumption level of 7.4l/person that is approximately 60% of the British rate [10,11].

At the same time, a number of attributes of the work make comparisons difficult. The most obvious is the considerable variation in the quality of the problem alcohol indicator that was available. In only one of the studies (MSRA) was an interviewer administered diagnosis of alcohol use disorder (AUD) (i.e., abuse and dependence) available as well as self-report consumption measures. In three of the studies (MTF, FJYLS, CCLS) relatively good problem/symptom indicators as proxies for AUD were available, as well as measures of consumption. Two of the studies (MSALT and NCDS), both surveys of large samples, unfortunately only had relatively crude consumption measures (ratings of drinking frequency over a six month interval in one case (MSALT), reports of quantity consumed in the past week in the other instance (NCDS). Both of these studies also had single indicator measures of problem use, CAGE items [12] in the NCDS, ratings of number of times drunk in the past six months in MSALT. This range of variation makes it difficult to evaluate whether conflicting findings

[1]To make the text less ponderous, from here on I refer to these studies in acronym form, thus, CCLS = the Columbia County Longitudinal Study [2]; FJYLS = the Finnish Jyväskylä Longitudinal Study [3]; MSALT = the Michigan Study of Adolescent Life Transitions [4]; MSRA = the Minnesota Study of Risk and Adaptation [5]; MTF = the Monitoring the Future Study [6]; and NCDS = the British National Child Development Study [7].

need to be regarded as genuinely different, or result simply from poor measurement of the construct.

Question 1. How consistent is the evidence for an externalizing pathway to later alcohol involvement?

As a number of systematic reviews have pointed out, one of the core predictors of problem alcohol use and alcohol use disorder (AUD) from early childhood onward is behavioral undercontrol [13–16], described in its various forms as externalizing behavior/ symptomatology, aggressiveness, antisocial behavior, delinquency, impulsivity, and sensation seeking. All of these variables share the characteristic of disinhibition, *involving the inability or unwillingness or failure to inhibit behavioral impulses even in the face of negative consequences* [17]. The majority of the studies here that assess this domain continue to report this relationship. Thus, in the CCLS, a strong undercontrol pathway was evident. In their final mediational model, there was no evidence to support the need for gender-specific solutions. Both aggression and behavioral inhibition showed significant autostability from ages 8 to 19 (standardized regression coefficients ~.27) as well as significant (albeit slightly lower ~.15) longitudinal relationships with each other, suggesting the commonality of a broader undercontrol construct. Both age 19 variables also had significant relationships to problem drinking and level of alcohol use consumption at age 30 and a direct path from behavioral inhibition at age 19 to both quantity and problem drinking at age 48 also was present. The age 19 aggression relationships to age 48 outcomes however, were completely mediated by drinking behavior at age 30.

Although the MSRA *N* was not sufficient to model relationships at this level of complexity, Engelund et al. also observed a relationship between a broad-band measure of externalizing at age 9 predicting AUD at age 28 in their male sample, essentially replicating the CCLS findings but in a less fine-grained way [5]. The partial mediation of this relationship by intermediate drinking experience was also suggested by the MSRA finding of heavier alcohol use at age 16 among later-to-be alcoholics. The lack of gender parallelism in the MSRA findings may be attributable to the low power of their female analyses; only 2.5% of females met AUD criteria in adulthood.

This pattern of generally positive undercontrol-to-problem drinking relationships is also found in the FJYLS. Aggressiveness and low self control at age 8 predicted heavy drinking at age 20, and problem drinking at age 27 and age 42 among males but not among females. The undercontrol relationships were substantially stronger in early adolescence: aggressiveness at age 14 for both sexes was predictive of heavy and problem drinking at all ages, as was low self control for problem drink at both ages 27 and 42 among males and females. In addition, low constructiveness, low compliance, high aggressiveness, low self control, and truancy, predicted a variety of problem drinking outcomes among both males and females.

The MTF study also found substantial relationships between undercontrol-domain variation and AUD outcomes. High school theft and property damage were two of the strongest predictors of adult AUD, especially abuse, but risk taking was also positively related, although at a lower level. High risk taking was also systematically related to higher consumption, across all measures and all ages from 22 to 48. Most surprisingly however, at least vis a vis alcohol dependence, high school aggressiveness (the measure actually has strong assaultiveness content) was *negatively* related to dependence.

This anomaly leads us to pose a very basic question, namely what are the core features of the undercontrol domain that are responsible for these positive relationships? For the MTF work, the undercontrol construct encompasses rule breaking as well as risk taking, possibly some thrill/sensation seeking and recklessness (subsumed under the property damage measure) but

not direct assaultiveness. Only analyses at the item level would begin to provide some understanding of that. For the NCDS, "externalizing" includes "destruction of one's own as well as others' belongings", which speaks as much to lack of control of oneself as it does to externalizing vis a vis others. However, "social maladjustment", which includes both hostility (externalizing undercontrol) and restlessness (motoric undercontrol), as well as withdrawal, is not included as part of the same variable/construct. Neither is truancy, which involves another type of social misbehavior/rule breaking, yet this variable is strongly related It is essential to understand the nuances of these connections. It appears that there is a common undercontrol factor which cuts across all of them, but the distinctions that are added by the different manifestations of undercontrol sometimes lead to differences in strength of the pathway. Next-step work needs to build and test competing models of mechanism, that test one conceptually differentiated undercontrol facet against another, which in turn should lead to a more fine grained understanding of the core nature of the undercontrol diathesis. This type of differentiating research is being done in the ADHD [18], and delinquency areas [19,20,21], but none has as yet focused on disaggregation of the undercontrol construct as it relates to the development of alcohol and other drugs of abuse. It needs to take place.

My summary of findings in this area also highlights another point, namely that the pathway of undercontrol to problem alcohol use is not always direct; sometimes the relationship is mediated through earlier drinking, which in turn may have been predicted by earlier undercontrol. This observation has been made in earlier studies [22], and this pathway of effect was present in the CCLS. These is also some suggestion from the findings of both the MSRA and the FJYLS that it is operating, by way of intermediary relationships between externalizing in childhood/adolescence to young adult drinking behavior which in turn then predicted problem use in middle age, but neither study carried out the kind of mediational analyses that would be needed to establish the effect. To the extent that this relationship is present, it also suggests the operation of an epigenetic process, whereby earlier undercontrol leads to earlier/more problematic drinking, which in turn then is both predictive of more externalizing as well as problem drinking/alcoholism in middle adulthood. The underlying mechanism(s) sustaining this mediated process are not evident. They might be peer group effects, they might be neurophysiological effects, whereby the alcohol consumption itself produces more capability for both disinhibition as well as heavier drinking, and they might be both. This is a more microlevel process than can be observed in what are primarily field interviews and surveys but again, it is important to know the answers to these pivotal questions if we are ultimately to identify mechanism.

Question 2: Is there any consistent evidence for other risk pathways?

In addition to the possible mediating role of drinking in the development of an externalizing pathway to problem drinking, the studies point to three other possibilities: (a) A direct drinking pathway to problem alcohol use and alcoholism in middle adulthood; (b) an internalizing pathway, and (c) a competency/maladjustment pathway.

The direct drinking pathway

Does earlier drinking "cause" later drinking and drinking problems, above and beyond the role it plays as a mediator of an externalizing pathway?

In general, the answer is "yes" with some caveats and qualifications. Across study evidence is considerable for autostability of drinking over time, and was observed in MSALT (strong autoregressions for general use and frequency of drunkenness between ages 18–21), in the MSRA finding of alcoholism at age 28 predicted by heavy drinking at age 16, and the strong relationships observed in the CCLS between both quantity of alcohol use and problem drinking at age 30 predicting frequency of use as well as problem drinking at age 48. The MTF analyses

also systematically show these relationships and fill in the picture of process a bit more clearly. Their measurement intervals are shorter in the earlier stages of the study design, and all drinking measures except the abuse/dependence symptomatology indicators were available at all data collection points—thus eliminating measure inconsistency effects. Age 18 drinking predicted *all* drinking outcomes across all ages from 22 to 35. SEM path relationships were strongest for drinking frequency, less so for heavy drinking, and smallest for the prediction of symptomatology, across both genders and all time periods. Put another way, the more isomorphic the predictor was to the dependent variable, the closer the relationship. A clear and graded time dilution effect was also present, with relationships between drinking behavior strongest across the shortest time span (18–22), and decreasing systematically as time span increased. At the same time, since MTF starts at age 18, we cannot know whether the age 18 drinking itself was mediating a developmentally earlier undercontrol trait, or another alcohol nonspecific variable.

The FJYLS data are able to address this question directly because of the range of their variable network, the developmental breadth of the study, beginning at 8 and extending into the ‘40s, and because of an explicit focus in the analyses on the relative predictive power of drinking variation vis a vis other influences. Pitkänen et al were also interested in examining the developmental hypothesis that drinking variation would become more predictively salient in anticipating adulthood drinking outcomes, while individual and contextual/familial influences would be more salient in earlier life [3]. They anticipated autostability of effects, and also predicted that the more proximal drinking relationships would be stronger than the more distal ones. The data did not fall exactly as anticipated. Drinking in early adolescence (age 14) predicted drinking behavior in early middle age, and it also had independent power as a predictor when familial and behavioral precursors were controlled; i.e., it was more than just a mediator of precursive risk factors. The study also found strong autostability effects, and generally continuity was highest across adjacent measurement points. However, in contrast to the MTF findings, they did not find the same predictability and ordering over longer spans of time. That is, early adolescent drinking was more highly predictive of drinking frequency at age 42 than was heavy drinking at age 20. Why might this be, and how might we account for the differences? One possibility is that individual differences may sometimes be obscured, or superseded, by age specific developmental and contextual effects which tend to dissolve once the developmental epoch has been traversed. Such effects have been observed by O’Malley and Johnston, whose analyses of national MTF longitudinal data show that the age 18–22 period is one of greatest changes in heavy drinking--involving a move into heavier use, followed by a gradual drop off [23]. This developmental interval is also a period that is subject to significant contextually created divergence in the drinking trajectories of those who attend college and those who do not. The differentiation occurs at time of college entry, and reaches its greatest separation right around age 20. Following college, the pathways re-join, indicating an age/development-specific perturbation [23]. On both these grounds, drinking assessments at age 20 are likely to be more unstable than those occurring a bit earlier, or a bit later. In contrast, the MTF first drinking data point at age 18 avoids this peak instability since it occurs at the beginning of the adolescent-to-adulthood transition and the college entry transition. Their second data point occurs toward the end of this period of instability, thus to a greater degree minimizing the developmental perturbation effects.

In any case, to the extent that alcohol consumption and problem alcohol use have some trait-like characteristics, what might account for such autostability? These studies cannot provide an answer to the question because the range of variables at hand is not suitable to address it. On the basis of other work it is likely that intimate peer group networks, including both same sex and romantic partners play a strong role [24]. Other likely contributors to this effect are genetic variation, showing itself by way of individual differences in disinhibition between heavier, at risk drinkers and lighter ones [25], and also the physiological processes that

determine pharmacodynamic and pharmacokinetic effects, which show themselves via greater or lesser sensitivity to alcohol consumption [26], and greater or lesser differences in speed of metabolism and its concomitant effects [27].

Is there consistent evidence for an internalizing pathway?

Despite strong longer term longitudinal evidence for early internalizing/negative affect symptomatology systematically predicting alcoholism and problem drinking outcomes in adulthood (see [16] for a review), the evidence from shorter term prospective studies is not as consistent. The present studies, although not covering short time spans, yield decidedly inconsistent results. Thus, the NCDS found remarkably inconsistent and puzzling results, with internalizing behavior at age 11 sometimes predicting greater alcohol involvement at later ages, sometimes lower, and almost always with strong sex differences. Even here, the relationships were not more systematic among females. In the MTF study the SEM analyses showed a relationship to *lower* heavy consumption at ages 22, 26, and 35. The FJYLS observed a positive relationship between anxiety at age 8 and heavy drinking at age 20, but only for males, and not at other ages. The MSRA found no relationships between teacher reports at age 9 and any of their drinking outcome measures.

Unfortunately, the sparse measurement network for the internalizing domain, the uneven measurement across different ages, and the differences in the drinking dependent variable across studies make it impossible to come to any conclusion other than that if an internalizing pathway exists, it is probably not a linear one, and may involve interactions with age, gender, life stress, parental drinking, or even parental internalizing problems. Probing such relationships will require considerably more fine-grained developmental analysis than was able to be carried out here [28]. Further, a number of the long term longitudinal studies reviewed by this author [16] that found positive relationships had baseline assessments earlier than the ones found here, and predicted more often to an alcohol problem/AUD criterion as opposed to a consumption one. Finally, as Peck et al. so clearly demonstrate, some relationships are only capable of being articulated via pattern centered analyses which require profiling of predictor variables and probing for homogeneity of relationships within clusters of people who share a common profile [4].

Alternatively, the unsystematic nature of these findings may suggest that an internalizing pathway, or even some multi-class characterization involving internalizing symptomatology simply does not exist. At the moment, there is no way to resolve the differences between the null or protective effect findings observed here and those from other work reporting a pathognomic relationship. If in fact such a relationship exists, it is likely that the form of the relationship will be a complex, interactive one.

Is there consistent evidence for a (social) competence pathway, or alternatively, a (social) incompetence pathway?

This is not a common hypothesis among alcohol researchers, but the roles of social competence, intelligence, and social attainment in social functioning are mainstream issues for developmental researchers. Given the developmental and social psychologist professional identities of most of the contributors to this special issue, it is therefore not surprising that the issue gets probed here. Results across the studies are varied, and illustrate the complexity of the matrix surrounding drinking behavior.

Drinking can be either a benefit or a scourge, and findings from these studies illustrate both, involving variables that are in many ways quite similar to each other yet predict in opposite ways. These paradoxical findings are not new. Almost a generation ago, Shedler and Block made the observation that drug involvement was not always a negative outcome [29].

Adolescents who were socially successful were also heavier marijuana users, but not the heaviest. The point they made at the time, and more recently observed by others [30], is that a certain level of drug involvement is part of the fabric of social relationships and social success, and to be a nonuser is an index of a less successful adaptation with peers.[2] These findings continue in varying degrees to appear in the studies in this issue. They appear in the NCDS, wherein early academic ability was systematically related to consumption at a wide variety of ages ranging from 16 to 42. Both academic ability and SES/drinking relationships were slightly stronger for females, and/or applicable throughout a greater interval in adolescence and early adulthood.

In the NCDS however, early academic ability was not just related to consumption, but also to middle adulthood problem alcohol use. A parallel set of findings was observed when the social advantage indicator was family socioeconomic status. The FJYLS also found high parental SES related to one index of adult consumption —frequency, but the obverse for problem use: low family SES predicts more problem drinking in middle adulthood. Problem use was also predicted by poor school success, problems in social behavior, and maladjustment in school, among both sexes. The CCLS also observed relationships between early social position and drinking outcomes. Popularity in childhood had a low order positive relationship to consumption at both ages 30 and 48. A relationship to problem use only showed up in middle adulthood (age 48), but not at age 30. The MSALT study also observed that early positive social functioning indicators (social ability at age 12, and peer involvement at age 18) both predicted regular use at age 28, but difficulty making friends was related to *heavy* age 28 use. In contrast, the relationship between academic ability and achievement in two of the population samples (MTF and FJYLS) shows up as protective against heavy alcohol use. In MTF, higher high school grades were negatively associated with heavy drinking at all ages, for both genders. Those who planned on attending college at age 18 drank *more* at age 22, but *less* at age 35. There were no sex differences.

How is one to make sense of this complexity? The positive relationship between social adaptation, social skill, and drinking appears to be a general one. The issue is, when does it also lead to problem use? These studies suggests that problem use is more likely to appear under conditions of greater stress and social disadvantage (FJYLS), with individuals of lesser social competence (MSALT, CCLS), and among those of greater social mobility (the MSRA). These findings also point to another competency pathway that is less about social function and more about intellectual/academic skill. To the extent that school performance is positively related to compliance and negatively related to social involvement (grades are mildly negatively correlated with “evenings out” in MTF, and zero-order to negatively related to difficulty making friends and social ability in MSALT), these relationships should be protective against heavy use.

Taken together, these findings highlight a point that is often forgotten about alcohol use. Those who consume alcohol are potentially a servant to two masters. One master draws them closer to others, encourages affiliation and celebration. Here the relational activity is the master, and the drinking is carried out to enhance the activity. The other master is the drug itself. The drug is the focus, and use is sought out in order to alleviate negative feelings and reduce stress. As it does so, it also leads down a road toward increasing interpersonal difficulty, decreasing performance capability, undercontrol, craving, abuse, and dependence.

[2]This reference applies to involvement with peers who are part of the dominant, drinking subculture found in all Atlantic basin countries. It obviously does not apply to peers from those subcultures where abstinence is the dominant adaptation.

Question 3: To what extent are gender differences present, and how are they to be understood?

The fact that all these studies show level differences between females and males on all of the drinking variables is to be expected. These relationships have been observed as far back as epidemiologic data on alcohol use and problems have been collected [31], and they continue to remain true world-wide [10], albeit in decreasing magnitude in post-industrial societies where gender role equalization has started to take place [32]. However, the degree to which relationships among variables is parallel across gender is another matter entirely.

Three of the six studies observed either non-existent (CCLS) or small to minimal (MTF, FJYLS) sex differences in predictive relationships; for two others, the relationships varied depending upon type and developmental stage of the predictor variable and the outcome variable (MSRA; NCDS), and one observed substantial sex differences in pattern of the predictive relationship (MSALT). At the same time, across all studies, associations predicting drinking outcomes tended to be somewhat stronger among males than females. This was especially evident in the FJYLS and MTF, but was also observable in MSALT. That is, a tighter connection appears to exist between developmental antecedents and drinking outcomes among males than females. The pattern is also more evident in relationships involving the prediction of heavy drinking and abuse/dependence than it is in the prediction of use.

What might account for such across study constancy in magnitude differences of alcohol involvement, yet also account for across study variation in the detection of gender differences? To put this issue in context, the preponderance of evidence points toward a conclusion that sex differences are relatively small, if present at all. Thus, it appears that the most parsimonious, and most preponderant causal model for the development of drinking and alcohol problems is one involving traits (and mechanisms) that are largely non-gender specific. When such differences appear, they point to the fact that developmental course, and the social environment surrounding development at particular life stages, is different for the two genders. This is not an earthshaking observation to a developmental psychologist but it is one that is frequently ignored—at one's peril—by substance abuse researchers. I illustrate the issue with one example from the MSRA. The Minnesota study found that among males, *lower* academic achievement at age 12 predicted an increased likelihood of becoming a heavy drinker in the mid-20s. In contrast, *higher* academic achievement at age 12 predicted heavy drinking at age 23 among females. In probing this anomaly, the authors discovered that more academically inclined women were more likely to be attending college in their early 20s, and hence were involved in a heavier drinking environment. The same pattern was not true among men, wherein a continuity pathway applied. Although the authors did not report intervening data here, I would surmise that poorer school performance leads thereafter to a lower likelihood of both academic and occupational success, and the greater likelihood of a heavier drinking career. In other words, poorer earlier school performance is associated with involvement in a problem behavior matrix [33], which in turn predicts poorer school involvement and later heavier drinking. The more general point is that continuity in one domain may lead to apparent discontinuity in another. When the relationship is probed more closely, the linkage is evident.

Why might relationships be stronger among males than females? There are two plausible explanations, one biological, the other socio-environmental, but both consistent with such results. The biological explanation is that male drinking, and especially heavy drinking, is more under genetic control, and therefore less susceptible to environmental effects than the female pattern. Genetic influences, including those related to risky trait variation as well as alcohol pharmacodynamics, would provide a scaffolding of greater determinism across the lifespan than would environmental effects. It is noteworthy that this speculation is also consistent with the weight of evidence on sex differences in heritability of alcoholism, which suggests somewhat greater heritability, and a lower magnitude of both shared and nonshared

environmental effects among males [34]. And as noted by Merline et al. [6], the observation that parental drinking was generally a stronger predictor of offspring drinking among males (MTF) is also consistent with this hypothesis. The socio-environmental explanation is that, despite the increasing convergence of patterns of alcohol use across the sexes, there is still more heterogeneity in attitudes regarding the acceptability of heavy use among women. In some subcultures heavy use is increasingly expected (e.g., college women), in other subcultures, particularly those with a strong religious value emphasis, drinking to excess, and for some, even drinking at all is still taboo for women. This greater range in cultural acceptance of drinking among women would lead to more variability in relationships between distal antecedents and drinking outcomes of adulthood.

4. Next steps in articulating a model of the development of drinking behavior

Taking a step back from this work, there are several questions that need to be posed:

a) How well do these studies anticipate drinking and problem alcohol use 15 to 30 years ahead of time?—If one solely depends upon prediction from early childhood to mid adulthood, this group of studies clearly indicates that relationships are there, albeit of quite low order. Estimates of amount of variance accounted for are in the 2 to 5 percent range in predicting consumption and problem endpoints based on early childhood indicators alone, and generally with problem endpoints being more strongly predicted than consumption endpoints (FJYLS). However, they increase to a very robust 20 to 50 percent when all predictors at all intermediate ages are included in the model. This pattern of relationships is also evident in the MSRA and the NCDS, albeit with a much lower ceiling of variance accounted for in the British study. The MTF analyses do not provide these comparisons, although their R^2 of 10 to 20 percent from late adolescence to the mid ‘30s, also systematically show a drop-off in strength of prediction for the more distal predictions.

The fact that direct pathways of effect are evident from early childhood is thus again affirmed by this work (cf. [16]), but these studies also provide concrete evidence of epigenetic progression, wherein earlier relationships are mediated by intermediate functioning, albeit often of epigenetically parallel content. Thus, the CCLS shows that aggression and behavioral inhibition at age 8 are mediated by these characteristics at age 19, and they in turn are partially mediated by drinking behavior at age 30 which in turn predicts age 48 frequency and problem symptomatology. These traits assess opposite ends of the undercontrol domain, and their age 8 level also contributes to age 19 functioning of the obverse trait. To put this another way, developmental continuity is sustained by interactions in the intervening period that maintain the behavioral consistency. Results from the Minnesota and Finnish studies are generally in accord with this explanation.

b) What developmental issues have not yet been addressed?—Despite the overall elegance of this work, there are still two issues that have remained relatively ignored. One is lack of attention to heterogeneity in developmental course of the problem drinking phenotype; the other is lack of focus on processes related to reduction of problem consumption and offset of diagnosis (or symptomatology).

b1. Heterogeneity of course: Both recent and not so recent studies of the development of alcoholism and its precursive drinking and symptomatic representations provide considerable reason to postulate that the phenotype is a heterogeneous one for both developmental course as well as outcome. This observation has been made at the diagnostic level [16,35,36,37], at the symptomatic level [38], and at the consumption (binge drinking) level [39,40]. Yet despite this considerable body of evidence, only one study, the MSALT, analyzed their data to reveal such variation. While low N studies are less likely to uncover such variation, the evidence for

such variability—when it is examined—is the rule rather than the exception, and we are now at a point where the statistical software is widely enough available to carry out growth mixture modeling on most longitudinal multipoint data sets [41,42].

B2. Longitudinal analysis that incorporates models of offset/desistance as well as continuity and growth of risk: The low order predictability of long term drinking outcomes into middle age and the importance of mediators in determining developmental continuity are but two examples that illustrate the potential for discontinuity between earlier risk and later nonproblem outcomes. Despite the simplicity of this observation, the models utilized here have only the most rudimentary conceptualizations of this process. One important part of developmental theory is to characterize turning points, where the potential for discontinuity as well as continuity exists [43]. Such conceptualizations characterize when the possibility for offset or downturn in a trajectory may take place (e.g., at puberty, when leaving home, following college, at marriage, at time of parenthood, etc.). Such turning points mark an interval where the potential for change is higher. At the same time, the field is lacking a vocabulary of variables, both individual and environmental, that operate at such turning points, which would explicitly lead to such downturns in drinking behavior, and which also might relate to the rate of change of such variation. Given the success that these studies indicate is possible in predicting developmental emergence, it is time now to turn more explicitly to the flip side of that process.

Epilogue: In the focus on detail that this commentary requires, it is easy to lose track of how momentous the accomplishments are that are being presented here. The span of work ranges from a minimum of 16 years to a maximum of 40, and three of the six studies have been going on for more than 30 years. All of the studies have relatively low attrition rates and one, the FJYLS, has an amazing 94 percent retention rate over a 42 year interval. The dedication that such work requires, the attention to detail, and the investigator skill and fortitude that is necessary to sustain support for such work over so long a time, is not easily understood by those who choose to do cross-sectional and short term studies Each of these studies is, in its own right, an extraordinary scientific resource, and those of us who have the privilege of mining these treasures need to pay tribute to the career-long commitments that have made such activity possible.

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