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CHAPTER 5

Socioeconomic Status and Alcoholism: The Contextual Structure of Developmental Pathways to Addiction

Hiram E. Fitzgerald and Robert A. Zucker

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> *The evils resulting from the abuse of alcohol were never so prevalent as at present, and are now traceable in the diseases of youth, as well as in those of adult existence. Amongst the results of the killing pace at which the race of life is too generally run, from its start to its finish, one of the most serious is, that the period of childhood has become so abridged, in many instances, by the necessity of entering on the struggle for existence before the sufficient development of the moral, mental, and physical powers, that a premature break-down in any of these is no longer exceptional.* (Madden, 1884, p. 358).

The societal consequences of alcohol abuse and alcoholism are very great, and beyond those imaginable even by Madden (1884). In 1994, economic costs related to alcohol abuse and dependence are estimated to exceed 100 billion dollars annually (Rice, Kellman, & Miller, 1991; Parker & Harford, 1992). Human costs of the effects of alcohol abuse and alcoholism are even greater. Of the more than 16 million Americans who meet DSM-III-R diagnostic criteria for alcohol abuse or dependence, approximately 100,000 die annually from alcohol related causes (Grant, Harford, Chou, et al., 1991). Estimates of the number of children of alcoholics (COAs) in the United States range as high as 28 million (Russell, Henderson, & Blume, 1985), with COAs six to ten times more likely to develop drinking problems than

are nonCOAs (Cotton, 1979). Alcohol abuse and alcoholism are linked to a wide variety of health problems including liver cirrhosis, pancreatitis, various neurological, cardiovascular, and endocrine disorders, cancer, disorders involving the reproductive system, and gross as well as subtle teratogenic damage to the fetus (Day, 1992). In addition, there is evidence indicating that alcohol nonspecific factors may be as important as alcohol-specific factors in setting COAs on a developmental pathway leading to maladaptive functioning (Zucker & Fitzgerald, 1991a). COAs are more likely than nonCOAs to live in households characterized by high rates of family violence as well as other forms of antisocial behavior (Martin, 1992). Data from the National Institute of Mental Health (NIMH) Epidemiologic Catchment Area study of the United States adult population indicated that comorbidities, excluding drug disorders, occur in 37 percent of the alcohol abuse/dependent population (Regier et al., 1990). Such comorbidities include antisocial personality disorder, bipolar disorder, schizophrenia, panic disorder, obsessive-compulsive disorder, affective disorder, and anxiety disorder. Clearly, being reared by an alcoholic parent exposes the child to a host of factors that may induce, facilitate, or maintain developmental pathways (Gottlieb, 1991) to substance abuse and related psychopathological behavior.

The Michigan State University–University of Michigan Longitudinal Study

Our purpose in this chapter is to illustrate how socioeconomic status variables, particularly as indexed by family income, may help to structure the developmental pathways to which COAs are exposed. Since our illustrations involve some of the data from the Michigan State University–University of Michigan (MSU–UM) Longitudinal Study (Zucker, Noll, & Fitzgerald, 1986; Zucker & Fitzgerald, 1991b) a brief overview of this ongoing study is in order. Three groups of families are participating: (a) court-recruited alcoholics, (b) community-

recruited alcoholics, and (c) community comparison families. The court-recruited alcoholic families were systematically recruited via a net of administrative arrangements with six local district courts in a four-county area covering all male drunk driving convictions. Men needed to have a blood alcohol concentration (BAC) of 0.15% (i.e., 150 mg/100 ml) or higher at the time of arrest, or a BAC of 0.12% and at least one prior alcohol related driving arrest. Eighty percent of the fathers in the alcoholic sample met a definite alcoholism diagnosis, and the remaining met a probable diagnosis on the basis of the Feighner criteria (Feighner et al., 1972). Subsequent to obtaining each alcoholic family, a matched community control family from the same census tract as the alcoholic family was located in a door-to-door canvass that began one block away from the alcoholic family. To the extent that it was possible, the control family was also selected to match the sibling composition and birth order of the alcoholic family. In the course of our work, 18,989 families were contacted. Of the 509 families with an age-appropriate male child, 475 of these were contacted and 441 agreed to participate; 215 families were ineligible due to ethnicity, socioeconomic status (SES), or parentage (i.e., non-biological); 105 were ineligible due to alcohol/drug involvement, 39 were not contacted due to successful recruitment of a comparison family. Ninety-one of these families were successfully recruited. To restrict ethnic variation that we were not able to oversample because of study locale, all subjects are nonHispanic Caucasian. The same screening procedures were used to ensure that neither the father nor the mother in a control family met either an alcoholism or drug abuse or drug dependence diagnosis. Any community control family whose father met either a probable or definite alcoholism diagnosis, was reclassified as a Community Alcoholic Family. Of the 105 families so classified, 60 were successfully recruited. Of the residual 45, all families had either moved away or separated by the time we attempted to recruit them, so acceptance rates here were actually 100 percent of the families that were available.

In accord with study exclusion criteria, no child manifested characteristics of fetal alcohol syndrome (Fitzgerald, Sullivan, Ham, et al., 1993). Specific characteristics are required

for a diagnosis of FAS in three areas: (1) prenatal and/or postnatal growth retardation, (2) central nervous system involvement, and (3) characteristic facial dysmorphology. Although fetal alcohol effects (FAE: Barr, Streissguth, Darby, & Sampson, 1990) may be characteristic of these children, there are doubts as to whether such effects can be reliably documented (see Plant & Plant, 1987), and whether FAE should be used in scientific publications due to the difficulty of operationally defining such effects (Sokol & Clarren, 1989).

Although all Wave 1 data has been collected, not all of it has yet been intensively analyzed. Moreover, at the time of this report, only about 40% of Wave 2 data has been collected. Nevertheless, cross sectional Wave 1 analyses have already provided valuable information about these families. For example, a variety of analyses have documented an overall increased incidence of behavior problems (Fitzgerald, et al., 1993), hyperactivity and attention deficit (Ham, Fitzgerald, & Zucker, 1993), difficult temperament (Jansen, Fitzgerald, Ham, & Zucker, 1993) and impulsivity (Fitzgerald et al., 1993)—among the high risk 3- to 5-year olds than among the comparison children. Among these children's fathers, higher levels of drug involvement are related to higher rates of antisocial behavior, and are inversely related to level of mental health, adaptive functioning, and socioeconomic status (Gonzalez, Zucker, & Fitzgerald, under review). In addition, differences along a dimension of antisociality provide strong evidence for different developmental pathways for two types of alcoholics (Zucker, 1987), which have been labeled antisocial alcoholics (AALs) and nonantisocial alcoholics (NAALs; Zucker, Ellis & Fitzgerald, 1994). Specifically, AALs have an earlier age of onset for first alcohol problems, more alcohol related difficulties, co-occurring psychopathology, and lower achieved socioeconomic status than NAALs (Zucker et al., 1994).

Approximately 45% of wives of alcoholics also make a DSM-III-R diagnosis for alcohol abuse or dependence, and in a number of analyses involving maternal variables—particularly antisocial behavior, lifetime alcohol problems, and depression—maternal variables were more predictive of their son's behavior problems than were paternal variables. For both mothers and

fathers, lifetime alcohol problems predicted child maltreatment (Muller, Fitzgerald, Sullivan, & Zucker, 1994). For fathers, social support and stress contributed independently to child maltreatment, whereas, for mothers social support seems to moderate the effects of stress on child maltreatment. Although father's lifetime alcohol problems predicted child behavior problems, the effect was moderated by a composite family psychopathology index consisting of mothers' alcohol problems, and measures of mother and father antisocial behavior, depression, and drug use (Moses, Gonzalez, Zucker, & Fitzgerald, 1993). Note that in a number of instances, socioeconomic status appears as a significant contributor to findings related either to individual or family functioning.

Socioeconomic Status and Alcohol Problems

At the point of entry into the longitudinal study, high risk families can be characterized as mid-level lower class, as indexed by family income, and as mid-range to lower level blue collar by way of occupation. Recently, Humphreys and Rappaport (1993) have argued that social science research on substance abuse is guided by researchers who accept the premise that the causes of substance abuse are to be found within the individual, not in ecological variables such as poverty or environmental stress. Their scan of PSYCHLIT for the years 1981 through 1992 yielded 170 citations pertaining to personality factors and drug addiction, but only 3 citations for drug addiction and poverty. Irrespective of Humphreys and Rappaport's position, it does not automatically follow that poverty or environmental stress cause substance abuse. That poverty or other ecological factors may contribute etiologically to substance abuse may be sufficient reason for rejecting single cause, internal models of causality, but, alternatively, such causal patterning is not sufficient proof for the exclusive action of external, or victim models. Developmental systems theory provides an bridge between internal and external causal models. From this perspective, it is as legitimate to hypothesize a linkage between personality characteristics and substance abuse, as it is to hypothesize a

linkage between poverty and substance abuse. Moreover, the personality-poverty-substance abuse triad is an even more plausible, multicausal interactive hypothesis to consider.

Within such a systems framework it is reasonable to ask how variables such as socioeconomic status contribute to the etiology of substance abuse as well as of family functioning. Commenting on the dangers of using "labeled environments" to group proximal variables, Richters and Weintraub (1990) note that one such label, social class, ". . . conveys no information about specific *proximal* experiences to which children within a given level of social class are exposed" (Richters & Weintraub, 1990). To be sure, spousal violence, child abuse, depression, and drunk driving, occur within all socioeconomic classes. Indeed, as Adler et al. (1994) conclude, "There is evidence that the association of SES and health occurs at every level of the SES hierarchy, not simply below the threshold of poverty. Not only do those in poverty have poorer health than those in more favored circumstances, but those at the highest level enjoy better health than do those just below." (p. 15). Nevertheless, there is increasing evidence to suggest that at least one type of alcoholism, antisocial alcoholism (Zucker, Ellis, & Fitzgerald, 1994) is more likely to be linked to or to co-occur with low socioeconomic class than are other types of alcoholism. We have, on a number of occasions, found indices of socioeconomic status to enter into regression equations as mediator or moderator variables in models designed to test direct and indirect effects of parent characteristics on child outcomes. These analyses have involved cross-sectional data from Wave 1 of the longitudinal study.

Socioeconomic class typically is measured by one or a combination of the following factors: occupational status (Stevens & Featherman, 1981), individual or family income, or years of education. A number of investigators have reported that the rate of alcoholism increases as socioeconomic level, education, or income decreases (Calahan, 1974; Helzer, Burnam, & McEvoy, 1991). Helzer et al. (1991) reported correlations of –.80 for men and –.54 for women between income and rates of alcoholism. Mulford (1964) found that male problem drinkers most likely to have related spousal, health, employment, or legal

problems were those most likely to have either the least or the most education, lowest occupational status, and lowest income. Using data from the 1990 census, Hilton and Clark (1991) found a U-shaped function between socioeconomic status and alcohol abuse/dependence. As indicated in Table 1, Midanik and Room's (1992) analysis of data from a representative sample of the adult household population of the 48 conterminous states of the United States, provide support for a U-shaped function when considering an index of average volume based on a mean intake level of two drinks per day (or 60 drinks per month). However, when an index of maximum consumption is used—namely one that evaluates the tendency to drink large amounts per occasions, then a different pattern emerged; drinking five or more drinks per occasion was more likely to be associated with lower income levels and the pattern is closer to linear. A parallel linear function also exists for the group that approximates binge drinking (that is, drinking eight or more drinks per occasion, at least weekly).

In her study of the geographic distribution of families participating in the MSU Longitudinal Study, Pallas (1991) compiled demographic information for each of the 67 census tracts over the five-county region from which our sample was recruited. She then used tract-level data as the unit of analysis to explore the relationship of appearance of alcoholism and a variety of indices of social dysfunction. Consistent with general trends in the literature, Pallas found the highest rates of alcoholic families (number of alcoholic families discovered per 1000 population) occurred near the central part of the largest urban city in the population area; correlations between total rates of appearance of alcoholic families and census tract level of urbanization were .34 for court-recruited alcoholics and .42 for community-recruited alcoholics (both *ps* <.01). Again at the tract level, both individual and family median incomes were inversely related to rates of recruitment of alcoholic families (–.51 and –.44, respectively; p <.001). As predicted, there were positive correlations between rates of alcoholic families and the percentage of families living below the poverty level (.64 for the

TABLE 1
Variation in Level of Alcohol Consumption Among Adults in the Contiguous 48 United States by Income Level During 1990 (Percentages)

	Percent of Adult Population					
	60+ drinks/month		5+ drinks at least once/week		8+ drinks at least once/week	
Income Groups	Men	Women	Men	Women	Men	Women
0–9,000	30	8	19	6	10	4
10,000–19,999	29	3	12	3	4	1
20,000–29,999	22	8	11	1	4	0
30,000–39,999	18	1	7	<.5	5	<.5
40,000–59,000	20	6	6	2	2	1
60,000+	26	8	6	3	3	1

Note: Data in the table were adapted from Midanik and Room (1992) and are in the public domain.

court-recruited alcoholism rate, and .67 for the community-recruited alcoholism rate). Pallas also found that elevated rates of alcoholism at the census tract level corresponded with elevated rates on other indices of family stress (separation, divorce, public assistance families, female heads of households, renter occupied households). Interestingly, there also was a positive correlation between tract level rate of drunk driver convicted families, and the individual father's blood alcohol concentration at the time of his arrest ($r = .30$, $p < .05$).

Income, Antisociality, and Lifetime Alcohol Problems

Pallas' analysis suggests that at the community context level, indices of status, including family income, operate in a meaningful way to enhance or maintain the vulnerable environments in which children of alcoholics are reared. In addition, at the individual level unit of analysis, such measures, including income, occupational prestige, and education, repeatedly account for 5 to 10% of the variance and/or emerge as significant moderators of outcome variables, in analysis after analysis of data from the first wave of the study.

It seemed reasonable, therefore, to look more closely at the relationship between one of these SES measures, family income, and a number of theoretically important variables (i.e., parental antisocial behavior and parental lifetime alcohol problems) that have been demonstrated to have direct effects on children's ongoing functioning. The Antisocial Behavior Checklist (ASB; Zucker & Noll, 1980) is a 46 item scale that asks the frequency of the respondent's participation in a variety of aggressive and antisocial activities. The total score is derived by summing across all items; a total score of 24 or greater is indicative of antisocial personality disorder based on DSM-III-R criteria (sensitivity = .85; specificity = .83; Zucker et al., under review). The ASB has adequate test-retest reliability of .91 (over 4 weeks) and internal consistency (coefficient alpha = .93) and differentiates among groups with varying histories of antisocial behavior including

felons, alcoholics, misdemeanor offenders, and college students (Ham, Zucker, & Fitzgerald, 1993).

The Lifetime Alcohol Problems Score (LAPS; Zucker, 1991) assesses differences in the extent of drinking problems over the lifespan. It provides a composite score derived from three component subscores: (a) the primacy component, involving the squared inverse of the age at which the respondent reported first drinking enough to get drunk; (b) the variety component, involving the number of areas in which drinking problems are reported; and (c) the life percent component, involving a measure of interval between most recent and earliest drinking problems, corrected for current age. Scores are standardized separately for males and females within the longitudinal study sample. Thus, a female score identical to a male scores indicates fewer problems of the female relative to the male. This measure is unrelated to current drinking consumption in problem drinking samples and has been shown to be a valid indicator of differences in long-term severity of drinking difficulty (Zucker, 1991).

In order to examine the relationship of income level to ASB and LAPS, and to determine if either of these proximal variables would be more strongly connected among families with the lowest incomes, we divided each group at the modal income category for the sample ($18,000). Table 2, therefore, contains means and standard deviations for ASB and LAPS for individuals in families with incomes above and below $18,000. Because at the time of this analysis, available *n*s in the data base were so few for community alcoholic families reporting incomes of less than $18,000, statistical comparisons were only made between the Court Alcoholic and Community Comparison groups.

Analysis of variance on ASB scores revealed significant main effects for RISK [$F(1,360) = 68.31$, $p = .00$] and SEX [$F(1,360) = 36.05$, $p = .00$] as well as a RISK x SEX interaction [$F(1, 360) = 9.93$, $p = .002$]. These findings mirror those obtained in many prior studies involving data from Wave 1 of the longitudinal study. Specifically, ASB scores are higher in alcoholic families than in control families (17.74 vs. 9.28), and are higher in men

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TABLE 2

Means and standard deviations for antisocial behavior (ASB) and lifetime alcohol problems LAPS) for court and community recruited alcoholic and community control families of higher and lower income level: MSU Longitudinal Study

Groups	Family Income < $18,000			Family Income > $18,000		
	M	*SD*	*N*	*M*	*SD*	*N*
ASB						
Court Alcoholics	21.89	12.11	62	15.79	9.90	132
Men	26.19	13.05	31	21.08	10.34	65
Women	17.58	9.47	31	10.66	6.04	67
Community						
Alcoholics	14.33	9.20	6	12.00	8.17	92
Men	20.00	9.00	3	14.95	7.76	44
Women	8.67	5.86	3	9.29	5.38	48
Controls	11.69	6.52	26	8.88	5.71	148
Men	13.31	7.16	13	10.23	6.25	74
Women	10.08	5.38	13	7.53	4.80	74
LAPS						
Court Alcoholics	11.04	2.22	63	10.40	2.00	127
Men	11.38	1.92	32	10.53	1.75	65
Women	10.69	2.47	31	10.26	2.24	62
Community						
Alcoholics	10.33	2.46	4	9.61	1.38	32
Men	9.26	2.87	2	9.52	.98	16
Women	11.40	2.31	2	9.70	1.72	16
Controls	8.80	1.60	20	8.08	1.68	107
Men	8.41	1.64	10	7.16	1.61	53
Women	9.20	1.54	10	8.99	1.21	54

Note: Family income is based on father's report. Note also that all court and community alcoholic men meet Feighner alcoholism diagnostic criteria as do some of the wives in the court alcoholic group.

than in women (16.71 vs. 10.37), although low income high risk women score higher than control men in both the low and high income groups (see Table 2). There also was a significant main effect for INCOME [F(1,360) =18.06, p = .000]; low income families had higher ASB scores than high income families. Moreover, there were no interactions involving income, indicating that it contributed independently to differences involving antisociality.

ANOVA of the lifetime alcohol problems index (LAPS) revealed main effects for RISK [F(1,360) = 73.09, p = .000] and INCOME [F(1,360) = 6.59, p = .01]. Individuals in alcoholic families had higher LAPS scores than did individuals in the community comparison families (10.61 vs. 8.20); whereas individuals in low income families had higher LAPS scores than individuals in high income families (10.40 vs. 9.45). Thus, income also contributes independently to LAPS. At the individual level, these analyses parallel findings from Pallas' study, as well as the extant literature linking income and other indices of socioeconomic status to alcohol abuse and dependence. Specifically, they suggest that investigators should consider entering SES indicators in their models as potential mediators or moderators of the relationships between parent and child variables. Of course, this presupposes that investigators view etiology as a dynamic process that changes in relation to changes in the stochastic relationships among system components external to the individual over the life course, as well as in relation to variation of internal factors.

Developmental Systems Theory and Models of Addiction

In recent years investigators have turned increasingly to developmental systems models of addiction in an effort to understand the complex biopsychosocial organizational dynamics that create pathways to and from alcohol abuse and dependence (Fitzgerald, Davies, Zucker, & Klinger, 1994; Zucker, 1994). Developmental systems theory views adaptive

functioning as a set of dynamic organizational processes that involve change and transformation, and that views behavior as embedded within contexts. Thus, substance abuse is conceptualized as a life-span problem with origins that to some degree are manifestations of the social structure, well beyond the confines of the individual (e.g., living in a drinking culture, being of lower social status), that begin to play themselves out even at conception (Boyd, Howard, & Zucker, 1994). Returning to late 19th century London:

> *Dr. Barlow (London) . . . confessed it was quite a revelation to him to hear about little children of tender years coming out of public houses in a state of actual intoxication. But he had seen the evil results among the London poor of giving small doses of spirit; especially gin, to little babies, even at the breast, on account of flatulence. This was a very common habit. He had also found it to be customary to give to quite young children, among some of the poorer classes, a daily quantum of beer. He was quite certain that physicians should pay attention to this habit of giving small doses of alcohol to children over long periods.* (Madden, 1884, p. 359)

Clearly the behavioral outcome of substance misuse is also impacted by influencing structures operating at the individual organism, as well as the suborganismic levels (e.g., biological variation). The dilemma of modelling this multilevel structure is that there is reason to posit both across structure influences (i.e., interactions), as well as within structure semi-independence. Thus, social class variation may impact early developmental processes both by providing a climate of poverty, within which jobs are hard to come by, psychosocial stress is higher, and impulsivity and action and heavy drinking are highly valued for their escape and masking functions as well as by way of its impact upon child rearing processes, spousal abuse, and family instability. Similarly, alcohol specific variation may impact individual development by way of societal level structures regulating alcohol availability, costs, consequences of use, by way of its likely sustaining influences upon poverty and the adversity of life circumstances (cf. Brenner, 1973), by way of its impact upon family structures including spousal violence, and by way of its impact upon individual social achievement. If one

is frequently drunk, job stability is impossible to sustain and downward occupational drift is one outcome.

In parallel fashion, the complexity of these processes is likewise illustrated at the biobehavioral end of the biopsychosocial continuum. For example, substantial evidence links difficult temperament to behavior problems in childhood and antisocial personality and alcohol abuse in adulthood (Tarter, 1988; Tarter, Moss, & Vanyukov, in press). Characteristics of difficult temperament include withdrawal from novel stimulation, low adaptability, high response intensity, negative mood, low distractibility, and have been identified early in infancy. The consensual view is that individual differences in temperament are heavily regulated by genetic mechanisms, but that environmental factors can moderate its expression. Young children with difficult temperaments who are reared in chaotic, antisocial, substance-abusing households are more likely to be on a developmental pathway that will reflect continuity for psychopathology from childhood to adulthood. Conversely, young children with difficult temperaments who are reared in stable, loving, and non-substance abusing households are likely to be on a developmental pathway that will reflect discontinuity for psychopathology. A relevant question, of course, is whether the diathesis for such biobehavioral organismic characteristics as difficult temperament interact with SES, such that the risk for difficult temperament increases as socioeconomic level decreases.

Elsewhere we proposed a multifactorial systems approach to the study of alcohol (Fitzgerald et al., 1994) that is theoretically and methodologically linked to developmental systems theory (Ford & Lerner, 1992; Lerner, 1991), dynamical systems modeling (Levine & Fitzgerald, 1992), and probabilistic contextualism (Zucker, 1987; 1994). Five levels of analysis related to the structure and function of the alcoholic family system are posited. The first level requires identification and description of the presenting state characteristics of individual members of the family, including genetic differences that may trigger different behavioral propensities or sensitivities. For example, one may want to assess parental antisociality given the strong link between antisocial behavior and alcohol abuse/dependence

(Zucker, 1994). The second level requires analysis of the structural and functional connections of subunits; that is, assessment of spousal, parent-child, and sibling relationships. For example, one may be interested in assessing how an increase in the rate of parental prosocial behavior will affect the rate of children's externalizing behavior (Maguin, Zucker, & Fitzgerald, 1994). The critical proximal influences on the developing child are to be found at these first two levels of organization.

Level three focuses on the analysis of the properties that emerge when system components couple and generate a specific dynamic structure. Functionally, this means assessing family traditions, values, beliefs, resources (including financial resources), and cohesiveness. It is from this level and the next that more distal variables, such as those that traditionally comprise socioeconomic status, may exert their mediating or moderating influences on proximal variables. The fourth level incorporates analysis of adjunctive systems and their direct and indirect effects on the family unit and/or individual members of the family. This level includes evaluation of cultural standards related to drinking, the availability of alcohol and other drugs, the economic status of the neighborhood, and the social-historical events (cohort effects) that contribute to cultural values. Finally, level five requires analysis of predictive models of individual, family, and ecosystem stability and change over time. Note that this level demands analysis of continuity as well as the bifurcations that disorganize systems and impel reorganization and change. At the most elementary level, this scheme provides a convenient way to categorize findings across diverse methodologies. At a more sophisticated level, however, it provides an organizing framework from which one can generate and test alternative etiologic models, or developmental pathways, to addiction (e.g., Zucker et al., 1994). Of course, to this point, most of our discussion has been based on assumptions of linearity. It is possible that the disorganization associated with alcohol abuse/dependence may better be represented by chaotic models; that is, by nonlinear dynamics rather than linear dynamics (Ehlers, 1992), further complicating the search for the critical determinants of alcoholism and alcohol related problem behavior.

Summary

Evidence that children reared in alcoholic environments are exposed to heightened levels of parental antisocial behavior, alcohol related problems, and other types of psychopathology seems incontrovertible. The data we presented here suggest that socioeconomic status variables may play a mediating or moderating role in regulating the effects of proximal influences on child outcome. However, we know little about where SES variables enter into the person-environment equation. While it is critical that investigators continue to identify the causal factors that lead to alcohol abuse and dependence, it is equally critical to identify the factors that exacerbate or buffer proximal causes. Perhaps we will find that substance abuse is one of the invisible threads woven through the fabric of poverty that, when identified, may help to unravel it.

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Round Table Commentary

Barnard. I found your paper to be very interesting. I guess the bottom line is that anything in and of itself is not the genesis of future functioning, rather that a complex of things (problems) are.

Fitzgerald & Zucker. Well, of course, but the scientific question is, what is the complex? In other words, systems have boundaries, even open systems, and we know relatively little about the way in which boundaries are constructed or how they are affected by exogenous and endogenous variables. For example, family systems theorists have described some alcoholic families as essentially closed systems with very rigid boundaries, boundaries that isolate the family and fuel internal processes that act to maintain alcoholism and other aspects of family pathology.

García-Coll. Several times you refer to the possibility of poverty being either a mediator or moderator between parent and child variables. Since mediators and moderators are so different conceptually can you elaborate which of these two concepts fits your multifactorial systems approach better ?

Fitzgerald & Zucker. This is an intriguing question and one that we wrestle with every time we test another model. At this point it seems that SES variables may be best conceptualized as moderators, if by moderator we mean variables that affect the duration or strength of the relationship between a predictor and an outcome variable. If SES was a mediator we would expect it to account for the relationship between predictors and outcomes. So, for example, for males there is increasing evidence to support

a strong relationship between antisociality and alcoholism. Does SES account for this relationship (mediate), or does it affect the duration or intensity of the relationship. If antisocial behavior is a trait, we suppose that one might look for mediational effects. On the other hand, if antisocial behavior is a state, then it may be that SES enters the equation more as a moderator than a mediator. What is becoming clear is that the days of treating SES as something to control rather than something to explain are rapidly going. It is one of psychology's litanies, often recited with some disdain, that, "SES is a marker variable." Well, it may be, but it sure is one remarkably powerful marker!

García-Coll. The role of genetics in your systems approach is still confusing to me. How do you operationalize and assess the first level of analysis, including what you call "genetic differences that may trigger different behavioral propensities or sensitivities?" Are you implying that parental antisociality has a genetic component?

Fitzgerald & Zucker. Genetics enters into a systems perspective in much the same way that Environment enters; that is, there are sources of variance that can be tied to biological variables just as there are sources of variance attributable to psychological and social variables as well. Arnold Sameroff has described the relationships among genotypes, phenotypes and what he refers to as environtypes, quite eloquently. If one views the nature of nature systemically, then one must build all potential sources of variance into one's model at least at some level of analysis. Yes, it may be the case that antisociality has a genetic component, but we are not sure what such a statement means. One interesting line of work involves the study of amplitude differences in the P300 event-related brain potential. Although to date this work cannot support a strong genetic component for alcoholism, there is good evidence that P3 amplitude differences can be linked to individuals with an antisocial personality that is co-active with alcoholism. Methodologically there are a number of well known techniques for studying biological aspects of the biopsychosocial triad: pedigree studies, twin studies, biological marker studies via analysis of blood samples, MRI and CAT scans, and

electrophysiological studies such as those that identified the P300 event-related brain potential.

Barnard. Do you foresee a time when childrearing by antisocial alcoholic parents will be discouraged? That is, that the children will be eligible for, or in foster care in order to reduce exposure to the affected parent(s).

Fitzgerald & Zucker. Remember some years ago Harriet Rheingold published a paper in the *American Psychologist* that discussed a variety of issues related to the "rights" of adults to bear children? She suggested that it may be beneficial to require adults to demonstrate parental competence before they were allowed to bear children. She cited licensing individuals to drive as an analogy. In a way we have made some progress, although not to the extent advocated by Rheingold. No, there is nothing on the horizon to suggest that such interventions are in the offing, despite the fact that alcoholism in general is one of the major health issues in contemporary society. Problems of substance abuse comprise the third largest health problem in the United States, right after cancer and heart disease. If we consider only mental health issues, substance abuse is right at the top; it is worse than problems of depression, anxiety, and delinquency. And alcoholism is the drug related to the biggest problems. It involves more people in traffic accidents, is closely linked to crime, delinquency, family violence, physical and sexual abuse, and leads to more deaths per year than any other drug. Just as we often excuse SES as a variable by controlling for it, so too we excuse gender by failing to note that all drugs of abuse are abused more by males than by females. Evidence that males are more aggressive than females is overwhelming. So, the combination of antisociality, alcoholism, and poverty in males, especially, is potentially explosive. Regardless of how powerfully we can predict risk at the population level, we are considerably less credible with respect to predicting individual outcome. For example, even though the sons of alcoholic fathers are four to eight times at greater risk for alcoholism than are sons of non-alcoholic fathers, nearly 70% of the sons of alcoholics do not become alcoholic! It seems we are stuck by the very