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**PROBLEM BEHAVIOR DEVELOPMENT IN SONS OF ALCOHOLICS:
INDIVIDUAL, FAMILY, AND CONTEXTUAL INFLUENCES**

By

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ABSTRACT

PROBLEM BEHAVIOR DEVELOPMENT IN SONS OF ALCOHOLICS: INDIVIDUAL, FAMILY, AND CONTEXTUAL INFLUENCES

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For children of alcoholics (COAs), externalizing behavior problems may be one of the first steps in a causal process leading to later problems including antisociality and alcohol problems. Guided by risk aggregation theory, the present study examined the impact of multiple factors on the development of child externalizing behavior problems in a sample of young male COAs. Specifically, the associations among lifetime familial alcohol risk structure (parental alcohol problems, antisociality, and low socioeconomic status), current parental stress and psychopathology, family conflict, cohesion, and expressiveness, child risky temperament (i.e., high activity level, high reactivity, and high adaptability), and child externalizing behavior problems were examined. Participants were a community sample of 208 families with 3 to 5 year old biological sons participating in Wave 1 and Wave 2 of the Michigan State University-University of Michigan Longitudinal Study. All families were intact at Wave 1 although by Wave 2 (three years later), 20 families were divorced or separated. Analyses were conducted using hierarchical regressions and structural equation modeling. Results supported the hypothesis that children with risky temperaments living in environments characterized by dense lifetime familial alcohol risk structures, more parental stress and psychopathology, and poor quality family environments are at elevated risk for

externalizing behavior problems. In addition, parental psychopathology increases levels of parental stress and functions to worsen the quality of the family environment. Finally, contrary to what was expected having a child with risky temperament does not worsen the quality of the family environment. Taken together, these findings indicate that children with risky temperaments who are raised in home environments dense with risk are on a developmental trajectory to later problems. This trajectory may begin as early as 3 years of age and if unchallenged across developmental time may lead to later antisociality and alcohol problems.

For my parents and for Patrick

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INTRODUCTION AND LITERATURE REVIEW

Children of alcoholics (COAs) are at heightened risk for a variety of negative outcomes including emotional and behavioral problems in childhood and adolescence and alcoholism in adulthood (Chassin, Pillow, Curran, Molina, & Barrera, 1993; Cotton, 1979; Dawson, 1992; Jansen, Fitzgerald, Ham, & Zucker, 1995; West & Prinz, 1987; Woodside, 1988). However, being the child of an alcoholic does not automatically place one on a trajectory leading to problem drinking or problem behavior. Many COAs function at average or above average levels when compared to nonCOAs (Clair & Genest, 1987; Johnson & Jacob, 1995), suggesting a considerable amount of variability in outcomes for this population (Jacob, 1992). This heterogeneity may be due to contextual variables such as socioeconomic status (SES; Fitzgerald & Zucker, 1995; Velleman & Orford, 1993), parent level variables such as level of parental stress (Moos & Billings, 1982) and presence or absence of psychopathology (Johnson & Jacob, 1995), family climate variables such as degree of conflict, cohesion and expressiveness, and quality of parent-child relationships (Cooper, Pierce, & Tidwell, 1995; Moos & Moos, 1984; Reich, Earls, & Powell, 1988), and also to individual child variables such as temperament (Blackson, Tarter, Martin, & Moss, 1994; Jansen et al., 1995). Thus, to fully understand the etiology of behavior problems in COAs a multifactorial approach must be utilized and the joint contributions of environmental and individual factors must be examined. Developmental Systems Theory (DST) provides an excellent framework for organizing the study of multicausal phenomenon (e.g., Fitzgerald, Davies, Zucker, & Klinger, 1994; Fitzgerald, Zucker, & Yang, 1995; Ford & Lerner, 1992; Tarter,

Moss, & Vanyukov, 1995; Tarter & Vanyukov, 1994; Zucker, 1994; Zucker, Fitzgerald, & Moses, 1995; Zucker & Gombert, 1986), and it is the framework guiding the current study.

Developmental Systems Theory

Developmental systems theory, based on general systems theory (von Bertalanffy, 1968), is concerned with individual continuity and discontinuity and thus, is an appropriate framework for the study of the etiology and maintenance of problem behaviors in COAs. Within DST, individual development is viewed as a process that is dynamic or constantly changing, multicausal and multifactorial. The study of behavior problem development must include the longitudinal examination of the qualities of the individual, the interaction among individuals within the multiple contexts of development, as well as the transactions that occur between developing individuals and the ever-changing contexts in which they develop (Lerner, 1989; 1991; Sameroff, 1975). Individuals can never be considered independent of the context in which they develop not only because the context influences development, but also because the individual influences the context in which development is occurring (Bell, 1968; Ford & Lerner, 1992; Sameroff, 1975; 1995).

Numerous researchers have used DST as the guiding framework in developing a comprehensive view of how COAs develop later problematic outcomes including behavior problems, antisocial behavior, as well as alcoholism (e.g., Fitzgerald et al., 1994; 1995; Tarter & Vanyukov, 1994). For instance, DST provides the guiding framework for the Michigan State University-University of Michigan (MSU-UM) Longitudinal Study. Heuristic models developed by this

longitudinal study incorporate variables from five major domains of biopsychosocial functioning (biological, psychological, peer, familial, and socio-cultural) to identify and map the causal pathways leading to later alcohol and other psychopathological problems in young COAs (Fitzgerald et al., 1995). Although the current study used portions of the data from the MSU-UM Longitudinal Study only the psychological, familial, and socio-cultural aspects of functioning were examined. Furthermore, although DST is the guiding framework of the current study, risk aggregation theory guides the specific hypotheses.

Risk Aggregation and Alcoholism Etiology

Zucker and his colleagues (Zucker, 1987; 1994; Zucker et al., 1995) have combined the general principles of DST with those of risk aggregation theory to explain how and why some individuals develop later alcohol problems while others do not. Risk aggregation theory proposes that maladaptive outcomes are maintained across developmental time in circumstances where a dense set of individual risk factors is continually paired with or nested within high risk environments (Zucker et al., 1995). In other words, the critical variables that guide individuals onto maladaptive pathways are necessarily a combination of both individual and contextual variables. Furthermore, the presence or absence of these same variables across developmental time will determine whether or not the pathway will be maintained. Shifts in individual pathways or trajectories may occur when changes occur in individual-level variables or in the context in which development is occurring. Thus, discontinuities in development may occur when there are drastic changes in risk factors whereas development remains continuous when individual

and contextual risk factors persist across developmental time.

For instance, COAs who have temperaments that are considered difficult by their parents and who live in environments characterized by low SES, parental alcohol problems, antisociality, and chaotic family environments are most likely to be placed on a developmental trajectory to later problems and if maintained possibly to a type of alcoholism that Zucker and his colleagues (1987; 1994) have labeled Antisocial Alcoholism. Antisocial Alcoholism is characterized by the presence of antisociality and is a culmination of the effects of early and sustained child and adult externalizing behavior problems paired with high risk environments. Previous studies have reported a strong association between child externalizing behaviors and later problematic alcohol involvement (see Zucker & Gomberg, 1986 for review). In fact, child externalizing behavior problems are one of the first steps in the hypothesized causal pathway leading to later antisociality and alcohol problems in COAs.

Because of the important antecedent role that child externalizing behavior problems play in later problematic outcomes, the focus of the present study was the prediction of COA externalizing behaviors. Specifically, the purpose of the study was to examine the impact of multiple factors on the development of externalizing behavior problems in young male COAs. Guided by risk aggregation theory, the associations among lifetime familial alcohol risk structure (operationalized by family SES, lifetime parental alcohol problems and antisociality), parental stress and psychopathology, family environment (operationalized by conflict, cohesion, and expressiveness), child temperament, and child externalizing behavior problems were

investigated. Data from the first two waves (three years between each wave) of the MSU-UM Longitudinal Study were used to examine how the multiple level variables interact and how they are associated with problem behavior development.

Lifetime Familial Alcohol Risk Structure

In the present study, lifetime familial alcohol risk structure was conceptualized by family SES, parental lifetime alcohol problems, and parental antisociality. All three of these variables are highly correlated and conceivably represent the cumulative impact of alcohol and alcohol-related problems on parental and family functioning.

Socioeconomic Status

Although SES often only includes measures of family income, parental occupation and education, Sameroff and Seifer (1983) refer to it as an important "summary variable" that may impact child outcome. Socioeconomic status acts as a proxy variable for a range of other variables that may influence child development and are associated with the SES of the family. Thus, children of low SES families can be passively affected by an environment that lacks enriching and stimulating experiences or the same child can be actively affected by an environment that is characterized by a large number of stressful life events and the maltreatment and abuse of the child (Sameroff & Seifer, 1983).

In accordance with Sameroff and Seifer's (1983) argument, a positive association exists between SES and levels of stress experienced by families (McLoyd, 1989). For instance, Adler et al. (1994) reported that higher SES families encounter fewer negative events in their environments than do lower SES families.

In addition, when higher SES families encounter a potentially stressful situation they have access to more resources, such as a wider social network, to help them more effectively cope with the event. Use of appropriate coping strategies results in lower levels of family stress and, thus, protects children from negative outcomes.

Socioeconomic Status and Child Outcomes

Low family SES has also been associated with increased levels of behavior problems in children (e.g., Dodge, Pettit, & Bates, 1994; Dyer Harnish, Dodge, & Valente, 1995), antisocial behavior in adolescence (Moffitt, 1993) and alcoholism in adulthood (Hill, Blow, Young, & Singer, 1994; Fitzgerald & Zucker, 1995; Yang, 1992). Dodge et al. (1994) found that SES rated in kindergarten predicted teacher-rated behavior problems and peer-nominated aggressive behavior in kindergarten, grades 1, 2, and 3. However, various researchers examining the relationship between SES and child outcome have concluded that SES may not directly be related to child outcome, but rather SES impacts other socialization variables, which in turn are directly associated with child outcome (Conger, Ge, Elder, Lorenz, & Simons, 1994; Dodge et al., 1994; Dyer Harnish et al., 1995; Elder & Caspi, 1988; McLoyd, 1989). For instance, although Dodge et al. (1994) reported a direct association between SES and child behavior problems, they also found that socialization variables (e.g., harsh discipline, high rate of violence, low stability in peer group, high rate of family stressful life events) partially mediated (see Baron & Kenny, 1986 for discussion of mediators and moderators) the relationship between SES and child outcome.

Bolger, Patterson, Thompson and Kupersmidt (1995) found that children who

experienced persistent economic hardship had more behavior problems than children who experienced intermittent hardship followed by children who did not experience any economic hardship. Furthermore, the relationship between economic hardship and externalizing behavior problems was partially mediated by maternal involvement. Although maternal involvement did not account for all the variance in child behavior problems it did act as a partial filter on child outcome (Bolger et al., 1995). It is possible that a larger portion of the variance in child behavior problems could have been accounted for if other socialization variables had been included in the analyses (Bolger et al., 1995). For instance, other family environment variables such as mother-child interaction quality (Dyer Harnish et al., 1995), parental mood (Conger et al., 1994), marital conflict and parenting skills (Conger et al., 1992) have been found to partially mediate the relationship between family SES and child outcome.

Economic hardship or low SES is a form of stress that may negatively impact parental socialization variables and produce a disadvantage in socialization for the children (Conger et al., 1992; Dodge et al., 1994). Stress from economic hardship may act to increase parental emotional distress which directly impacts both marital and parental behavior (Conger et al., 1992). Thus, parents under economic pressure may experience more marital conflict, stress, a poorer quality family environment, and poorer parental monitoring and discipline skills that lead to problem behaviors in their children.

Parental Lifetime Alcohol Problems and Antisociality

Since alcohol abuse/ dependence is the most common of all DSM-III disorders (Robins & Regier, 1991), it is not surprising that researchers estimate there are at least 6.6 million COAs under the age of 18, and 22 million who are 18 years of age and older (Russell, Henderson, & Blume, 1985). Although COAs are at heightened risk for developing behavioral disorders (e.g., Dawson, 1992; Fitzgerald et al., 1993; Puttler, Zucker, Fitzgerald, & Bingham, 1996; West & Prinz, 1987; Woodside, 1988), this heightened risk may not be due solely to the parental alcohol abuse/ dependence. Rather, there are other factors that may exacerbate risk (Zucker & Fitzgerald, 1991a). For instance, antisocial personality disorder is the most common comorbidity of alcoholism (Regier et al., 1990), and studies find that alcoholics display antisocial and aggressive behavior in childhood and adolescence (Zucker & Gomberg, 1986). Furthermore, antisociality has been studied extensively as a factor that may differentiate types of alcoholics (Babor et al., 1992; Cloninger, 1987; Zucker, 1987; 1994).

Zucker and his colleagues (Ellis, 1992; Ellis, Bingham, Zucker, & Fitzgerald, 1996; Zucker, 1987; 1994; Zucker, Ellis, & Fitzgerald, 1994; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996) have proposed a typology of alcoholism -- Antisocial Alcoholic (AAL)/Non-Antisocial Alcoholic (NAAL) that is based on familial history of alcoholism, age of onset, severity of alcohol and other drug involvement, and the presence of other psychopathology. When Zucker et al. (1994) tested the typologies on a community sample of 102 alcoholic men participating in the MSU-UM Longitudinal Study they found that AALs and NAALs could be

differentiated on the basis on childhood and adulthood antisociality. Not only did AALs score significantly higher on measures of childhood and adulthood antisocial behavior than did NAALs, they also had an earlier age of onset for drinking problems, more alcohol-related problems, and a longer duration of use. Antisocial Alcoholics reported experiencing more depression and other drug involvement, more divorces and separations, more alcoholism in relatives and a lower attained SES. Therefore, AALs reported more negative life events than did NAALs.

Lifetime Familial Alcohol Risk Structure and COA Outcome

Increased levels of parental alcohol problems and antisociality are associated with lower family SES (Blackson et al., 1994; Fitzgerald & Zucker, 1995; Hill et al., 1994; Smith, 1996) and thus, parents with low SES, alcoholism and antisociality are highly likely to provide their children with environments that are dense with risk.

Parental antisociality is an important factor when considering the impact of parental alcoholism on child outcomes. Antisocial behavior differentiates not only between types of alcoholics but also between groups of alcoholic families (Ellis, 1992; Zucker et al., 1994; 1996) For instance, results from the MSU-UM Longitudinal Study indicate that AAL families have a lower SES than do NAALs who have a lower SES than do non-alcoholic Controls, and that 3-9 year old children of AALs have more behavior problems and more difficult temperaments (Behling, 1996) than do children of NAALs and children of Controls.

Earls, Reich, Jung and Cloninger (1988) examined differences in outcomes of COAs, children of individuals with antisocial personality disorder (ASP) and children of controls. Consistent with previous research, they found that COAs and

children of antisocial individuals (most of whom also were alcoholic) were 2 to 3 times more likely than children of controls to have a behavioral or emotional disorder. However, no differences were found between the rates of emotional and behavioral problems in COAs and children of individuals with ASP. These results are contrary to those reported by Zucker and his colleagues (Ellis, 1992; Zucker et al., 1994; 1996) but could be due to methodological artifacts. For example, the study conducted by Earls et al., (1988) included 50 alcoholic families and only 18 families of individuals with ASP, 12 of whom also had alcoholism. Thus, the small number of ASP families and the unequal sample sizes may have led to bias in the results.

Based on findings that indicate individuals with a history of alcohol problems, antisociality and low SES provide their children with high risk environments, the present study included examination of these variables. Based on risk aggregation theory, it was expected that a dense lifetime familial alcohol risk structure (i.e., lower SES and more alcohol problems and antisociality) would predict higher levels of current parental stress, more parental psychopathology, and a family environment characterized by more conflict, less cohesion and expressiveness.

Parental Stress

Although the study of stress has a long history and cuts across many domains of psychological research (Beckman-Bell, 1981), researchers have not been able to generate a widely accepted, consensual definition of the phenomenon (Lazarus & Folkman, 1984). Yet, despite disagreements surrounding the definition, and use of the term stress, it has been an extremely enduring one (Garmezy, 1983). The

endurance of the concept is attributable to the fact that stress plays an important role in both child and adult outcomes (Compas, 1987; Loukas, 1995).

Stress is a complex rubric rather than a simple variable that is an inevitable part of life (Lazarus & Folkman, 1984). According to Lazarus' (1966) relational, cognitive theory, stress is based on cognitive appraisal, a process by which individuals evaluate to what extent and why a particular transaction or series of transactions between the person and environment is stressful. Perception of events as stressful is idiosyncratic, yet, to some extent individual perceptions are a reflection of actual environmental differences people experience (Lazarus & Folkman, 1984). Stress can be conceptualized as a major change in an individual's life (e.g, the birth of a child, marriage, or the death of a loved one) and is commonly referred to as *life events stress*. Conversely, stress can be caused by ongoing daily transactions with the environment; this is referred to as *daily hassles stress* (e.g., missing the bus, being caught in a traffic jam).

Although Lazarus (1966) argues that stress is based on individual cognitive appraisal, he contends that to some degree, individual differences are a result of actual environmental differences people experience (Lazarus & Folkman, 1984). Thus, the number of daily hassles and life events people report are an index of the stress they are experiencing. The present research evaluates stress in precisely this manner by assessing the number of daily hassles and life events parents are experiencing.

Parental Stress and Child Outcomes

Parent stress, both life events and daily hassles, is positively related to the level of child behavior problems (e.g., Beautrais, Fergusson, & Shannon, 1982; Cohen, Burt, & Bjorck, 1987; Hall & Farel, 1988; Loukas, 1995; Wagner, Compas, & Howell, 1988). For instance, Beautrais et al. (1982) found that mothers who reported a greater number of stressful life events, also reported more behavior problems for their children. Likewise, Hall and Farel (1988) found that levels of both maternal life events and daily hassles stress were related to child behavior problems. Because parental stress plays an important role in the expression of child behavior problems it must be included in a multifactorial examination of problem behavior development in COAs.

Parental Stress and COA Outcomes

Children of alcoholics experience heightened levels of family stress (Loukas, 1995; Moos & Billings, 1982). Alcoholism in and of itself is a form of chronic stress (Clair & Genest, 1987) not only for the alcoholic but for family members as well. Along with the everyday minor events encountered by all families that contribute to stress (Crnic & Greenberg, 1990), the alcoholic family is faced with the added stress of alcoholism. For the alcoholic family "normal" child behaviors such as bickering, whining and minor school problems add to other problems taxing or exceeding coping mechanisms. The build-up of family stress may in turn affect child outcomes (Cohen et al., 1987; Holahan & Moos, 1987).

Muller, Fitzgerald, Sullivan and Zucker (1994) examined the impact of parental stress on child maltreatment in a community sample of alcoholic families

participating in the MSU-UM Longitudinal Study. The results of this study revealed that increased levels of lifetime alcohol problems and more daily hassles stress predicted more child maltreatment for both mothers and fathers. Although the effect of stress on child maltreatment was significant for both parents, the process by which it influenced the outcome differed. For fathers, both stress and social support had independent direct effects upon child maltreatment. However, for mothers, the effects of stress on child maltreatment were moderated by social support.

Chassin, Rogosch and Barrera (1991) examined the influence of family events stress, parental alcoholism, parental antisociality and other psychopathology on the development of behavior problems and substance use in a sample of 10-15 year old adolescent COAs. Compared to the nonalcoholic comparison families, alcoholic families were characterized by more environmental stress, more family disruption, more parental psychopathology, higher levels of adolescent internalizing and externalizing behavior problems, and higher levels of adolescent substance use. Furthermore, current family stress, parental psychopathology, parental antisociality, and family disruption predicted adolescent externalizing behavior problems. Parental alcoholism, however, only predicted adolescent substance use and not adolescent externalizing behavior problems.

One possible explanation offered by Chassin et al. (1991) for the above results is that antisociality and alcohol use are separate phenomena with distinct antecedents and predictors. Another possibility is that parental alcoholism impacts child behavior problems only indirectly through socialization processes (e.g., Patterson, 1982; 1992; Patterson, DeBaryshe, & Ramsey, 1989). That is, parents

with alcohol problems use faulty socialization practices and initiate coercive patterns of interaction with their children that result in increased levels of behavior problems.

Overall, findings from studies examining the relation between family stress and child behavior problems indicate that families with a dense lifetime familial alcohol risk structure create a home environment characterized by increased levels of stress. Based on these findings and within a framework of risk aggregation theory, it was expected that increased levels of parental stress would predict more child externalizing behavior problems.

Parental Psychopathology

Depression frequently co-occurs with alcoholism (Regier et al., 1990). For example, results from one study indicated that the lifetime prevalence of depression in a group of male patients ($n = 928$) seeking treatment for alcoholism was 36% (Penick et al., 1994). Because of the high rate of comorbidity of this factor with alcoholism, parental current depression was examined in the present study.

Parental Psychopathology and Child Outcomes

Parental psychopathology and in particular parental depression, has been studied extensively as a risk factor for child behavior problems. Overall, results of such studies indicate that children of depressed parents are at heightened risk for developing both internalizing and externalizing behavior problems (e.g., see Downey & Coyne for review, 1990). For example, Fendrich, Warner, and Weissman (1990) found that families with a depressed parent (either mother or father) had higher rates of marital discord and divorce, more parent-child discord, and lower family cohesion than families of nondepressed controls. In turn, these variables were associated with

increased levels of conduct problems and depression in the children.

Parental depression has also been associated with increased levels of parental stress and poorer quality family environments. Thus, an association exists between parental stress and depression (Hammen, 1992) as well as between parental depression and marital and parent-child discord (Downey & Coyne, 1990). In a study on parental depression conducted by Billings and Moos (1983) findings indicated that compared to control families, depressed families (one parent was depressed) were characterized by more stressors and family milieus that were more conflictual, less cohesive, and less expressive. Additionally, the children of depressed parents were more likely than the children of controls to have emotional and behavioral problems. Overall, studies examining the influence of depression on nonCOA outcomes indicate that parental depression is associated with other risk factors in the family environment such as increased levels of stress and a poorer quality family environment and that these associations in addition to parental depression predict more child behavioral problems.

Parental Psychopathology and COA Outcomes

Just as co-occurring antisociality is a variable that characterizes one type of alcoholic (e.g., Ellis et al., 1996; Zucker, 1987; 1994) co-occurring depression has been found to characterize another type (e.g., Caplan, 1996; Finn et al., 1997; Zucker, 1987). Co-occurring depression in samples of alcoholics and their families is an important predictor of both adult and child outcomes (e.g., Johnson & Jacob, 1995). Similar to findings in the nonCOA literature, parental psychopathology predicts both internalizing and externalizing behavior problems in samples of COAs

(Chassin et al., 1991; Johnson & Jacob, 1995). Thus, for example, Chassin et al. (1991) found that parental psychopathology was a significant predictor of child externalizing behavior problems. Johnson and Jacob (1995) found that for male children between the ages of 5 and 18 years both maternal depression and family demographic characteristics predicted child internalizing and total behavior problems but not child externalizing problems. Previous studies have also found few differences in child outcomes between sons of depressives and sons of alcoholics. For example, Jacob and Leonard (1986) found that sons of alcoholics and sons of depressives both exhibited significantly higher levels of total behavior problems than sons of controls.

Given the extensive literature on the negative influence of parental depression on COA and nonCOA outcome, it is important that parental psychopathology be included in models predicting COA behavior problems. Based on findings from these literatures and within the framework of risk aggregation theory, it was expected that parental psychopathology would increase levels of parental stress, and function to increase family conflict, and to decrease family expressiveness and cohesion. Finally, it was hypothesized that parental psychopathology would directly predict increased levels of child externalizing behavior problems.

Family Environment

The family environments of alcoholics have been characterized by marital discord, parent-child conflict (Reich et al., 1988), and less happy, cohesive, and stable parent-child relationships (Velleman & Orford, 1993). Alcoholic families also report more divorces and separations than do non-alcoholics (Drake & Vaillant,

1988; Loukas, 1995). These conflictual, non-cohesive, and non-expressive family environments, in turn, have been found to contribute to the development of behavior problems in COAs (e.g., Moos & Billings, 1982; Rubio-Stipec, Bird, Canino, Bravo & Alegria, 1991; Velleman & Orford, 1993).

For instance, Smith (1996) found that for a sample of families participating in the MSU-UM Longitudinal Study, increased levels of alcohol problems and antisocial behavior each predicted poorer quality stimulation of the family environment (operationalized by the Home Observation for the Measurement of the Environment Scale; Caldwell & Bradley, 1984). In addition, Moos and Moos (1984) reported that families of alcoholics had frequent family arguments, less cohesion and expressiveness, and showed less agreement about their family environment and about joint task performance. However, the researchers also reported that cohesion was higher in alcoholic homes when the partners reported more positive and fewer negative events (e.g., family arguments). Thus, although the quality of the environment for some alcoholic families is poor for others the impact of alcoholism is moderated by the number of positive and negative stressful events.

Wilson, Bell and Arredondo (1995) examined family history of alcoholism, family environment, and temperament in a sample of 19-25 year old college students. Family history positive (FHP) participants reported that their families-of-origin were lower on cohesion, intellectual orientation, activity level, organization, and higher on conflict than did family history negative (FHN) subjects. Furthermore, consistent with research presented previously, FHP subjects were higher in the temperamental traits of impulsivity, activity level, and approach-

withdrawal.

Next, Wilson et al. (1995) examined the association between family environment and temperament and found that the pattern of correlations among the family environment subscales and temperament traits differed for the FHP and FHN subjects. The researchers proposed that the pattern of associations indicated that the temperament of FHP subjects was a partial determinant of their perceptions of the family environment. On the other hand, for FHN subjects temperament was not as strong a determinant of family environment perceptions as were actual contextual features.

Family Environment and COA Outcomes

Clair and Genest (1987) examined the impact of the family environment on adult COA outcome and found that adult COAs rated their families-of-origin as having less cohesion, organization and intellectual orientation and more conflict than did a group of adult nonCOAs. Furthermore, a lack of family cohesion, expressiveness, informational support, and emotional support was positively associated with adult maladjustment as defined by depression-proneness and self-esteem.

Rubio-Stipec et al. (1991) examined the influence of the family environment (operationalized by family dysfunction, marital discord, and the number of stressful life events) and parental alcoholism on child behavior problems in a community sample of Puerto Rican families. Alcoholics in this sample reported more family adversity, more stressful life events and marital discord than did non-alcoholics. Furthermore, when parents or psychiatrists were the informants of the child's

behavior only family environment (and not parental alcoholism) was significantly associated with an increased level of child behavior problems. Conversely, when the 4-16 year old children were the informants of their own behavior, both an adverse family environment and parental alcoholism predicted increased levels of child behavior problems. Differences in the results of adult versus child informants may be due to methodological artifacts. As children grow older they spend increasingly less time with parents and more time in contexts outside the home (Maccoby, 1984). Thus, children can report behaviors across a variety of contexts such as the home, school, and in the playground, whereas parental observations are more tied to the home context (Phares, Compas, & Howell, 1989). Nevertheless, quality of the family environment plays an important role in determining child adjustment.

Other researchers have reported that the quality of the family environment is an important predictor of child's emotional functioning (Moos & Billings, 1982) and distress (Yeatman, Bogart, Geer, & Sirridge, 1994). Yeatman and his colleagues (1994) reported that the family environment variables of conflict, cohesion, and expressiveness were stronger predictors of child distress than was parental drinking behavior. Thus, the authors concluded that distress in COAs is more a function of a poor family environment than it is of parental drinking problems. Due to the importance of the quality of the family environment on child outcome, the current study examined the influence of family conflict, cohesion, and expressiveness on child behavior problems. It was expected that a family environment characterized by more conflict, less cohesion and less expressiveness would predict increased levels of child externalizing behavior problems.

Child Temperament

Although there is no single definition of temperament accepted by all researchers, most would agree that temperament is the stylistic component of behavior (it is how individuals perform the behaviors they perform) that pertains to individual differences and has a biological/constitutional basis (Bates, 1989; Lerner, 1993). Temperament is an attribute that is expressed in response to an external stimulus, expectation or demand (Thomas & Chess, 1977) and although temperament is considered relatively stable it is subject to modification by the environment (e.g., Buss & Plomin, 1984; Thomas & Chess 1977; Kagan, 1987; Lerner & Lerner, 1983).

Three Temperament Theories

A number of contemporary researchers have proposed theories of temperament (Buss & Plomin, 1984; Goldsmith & Campos, 1982; Kagan, 1987; Rothbart & Derryberry, 1981). Each of these theories varies not only in the definition of temperament but also in what constitutes temperament. For instance, Thomas and Chess (1977) view temperament as the stylistic component of behavior that is expressed in response to an external stimulus. To Thomas and Chess (1977) the relationship between temperament and the environment is bidirectional; thus, temperament must always be examined within a context. More specifically, the fit or match between the child's physical and behavioral characteristics and the demands of the physical and social environment is paramount in determining child outcomes (Thomas & Chess, 1977). Adaptive or maladaptive child outcomes are not merely the product of temperament, or the influence of the context, but rather

the degree of fit (goodness-of-fit) between child temperament and environmental demands (Thomas & Chess, 1977; Lerner & Lerner, 1983). Adaptive outcomes result from a good fit or match between child characteristics and contextual demands, while maladaptive outcomes are a result of a poor fit or mismatch between child and context.

Thomas and Chess (1977) proposed the following nine attributes of temperament; approach or withdrawal, adaptability, quality of mood, intensity of reaction, rhythmicity, persistence, distractibility, activity level, and threshold of responsiveness. From these nine attributes three clusters of children are identified; the easy child; the difficult child (made up of the first five attributes listed); and the slow-to-warm up child.

Rothbart and Derryberry (1981) have also proposed a theory of temperament. For these researchers temperament is defined as relatively stable, biologically-based individual differences in reactivity and self-regulation. Reactivity is the arousability of motor behavior, affect, autonomic and endocrine response. Self-regulation refers to the processes (e.g., attention, approach, behavioral inhibition) that enhance or inhibit reactivity. Self-regulation processes increasingly modulate and organize reactivity across the lifespan. This theory of temperament is different from others in that it includes individual feelings in addition to overt behaviors. The dimensions of temperament studied by Rothbart and Derryberry (1981) are activity level, smiling, laughing, fear and distress in new situations, fussing and crying in response to limitations, soothability, and duration of orienting.

Buss and Plomin (1984) have proposed a theory of temperament, in which

temperament is a stable set of inherited personality traits that appear during the first year of life. Although temperament is believed to be inherited, it is subject to modification by environmental factors. The three traits that constitute temperament are: emotionality (distress), activity (tempo and vigor), and sociability (preference to be with others). These researchers propose that temperament develops by way of differentiation. For instance, the activity dimension of temperament is composed of tempo and vigor. Early in life tempo and vigor are difficult to separate, however, as an individual develops, the demands for one become greater than the demands for the other and thus differentiation occurs.

Temperament and Child Outcomes

Previous studies have reported an association between child temperamental characteristics and behavior problems in childhood (Behling, 1996; Caspi, Henry, McGee, Moffitt, & Silva, 1995; Jansen et al., 1995; Yang, 1992), criminal activity in childhood and adolescence (Henry, Caspi, Moffitt, & Silva, 1996) as well as antisociality and substance use in adolescence and adulthood (Lerner & Vicary, 1984; Moffitt, 1993; Tarter & Vanyukov, 1994).

Caspi and his colleagues (1995) found that for the participants in the Dunedin Multidisciplinary Health and Development Study, temperament characteristics at 3 and 5 years of age were associated with child behavior problems at ages 9, 11, 13, and 15 years. In particular, the lack of control dimension, composed of elements of emotional lability, restlessness, short attention span, and negativism, was related to externalizing behavior problems in later childhood and adolescence. Undercontrolled children displayed later hyperactivity, attention problems, antisocial behavior, and

conduct disorder (Caspi et al., 1995) and at age 18 were high in danger seeking, impulsivity, aggression, and interpersonal alienation (Caspi & Silva, 1995). Thus, the temperament dimension of lack of control seems to play an important role in the development of later behavior problems. According to Caspi and Silva (1995) lack of control reflects "an inability to modulate impulsive expression, impersistence in problem solving, and a sensitivity to stress that is expressed in affectively charged reactions" (p. 488).

Caspi and his colleagues (1995) offered two speculative explanations for the finding that temperamental undercontrol in early childhood is associated with later behavior problems. The first possibility is that temperamental undercontrol and child behavior problems are different degrees of the same underlying phenomenon. Extreme behavioral styles in early childhood are early manifestations of later behavior problems. Secondly, the researchers proposed that behavior problems are a culmination of poor person-environment interactions. The culmination of poor interactions occur as a consequence of the individual evoking certain responses from the environment, reacting a certain way to that environment, and then actively selecting and creating a type of environment that promotes problem behaviors (Scarr & McCartney, 1983). Although speculative, the latter explanation conforms to a goodness-of-fit view (Lerner & Lerner, 1983; Thomas & Chess, 1977) and corroborates findings indicating that genetically determined individual difference variables elicit responses from parents that place offspring at further risk for antisocial behavior (Ge et al., 1996).

Central to understanding how childhood temperament characteristics act to

predict later problem behaviors is the concept of behavioral regulation (Henry et al., 1996). According to Henry et al. (1996) behavioral regulation is comprised of both social regulation and self-regulation. Social regulation is regulation provided by family environment variables and; thus, is a process of socialization, whereas self-regulation is an individual difference variable and in this case is represented by the lack of control dimension of temperament. Both social regulation and self-regulation interact to influence the development and maintenance of problem behaviors.

Children who are temperamentally undercontrolled display behaviors such as impulsive behavior, temper tantrums, and aggression in early childhood that place them at risk for later problems such as violence, antisociality, and criminal behavior. When this lack of self-regulation is coupled with a lack of social regulation reflected in a chaotic and disorganized family environment this child is placed on a trajectory for later behavior problems, antisociality, and criminality (Henry et al., 1996). This trajectory may include rejection by "normal" peers and alliances with deviant peers who reinforce antisocial behavior and even introduce the individual to other forms of problem behavior (Patterson, 1982; 1992; Patterson et al., 1989). The longer these interactions occur, the fewer chances the individual has to engage in prosocial activities, reinforcing a life-course-persistent antisocial behavior (Moffitt, 1993).

Temperament and COA Outcomes

Numerous researchers have examined the association between parental alcohol problems and child temperament (Blackson, Tarter, Martin, & Moss, 1992; Blackson et al., 1994; Chassin et al., 1993; Ellis et al., 1996; Sher, Walitzer, Wood, & Brent, 1991; Smith, 1996). For instance, Blackson et al. (1994) reported that

compared to a group of control boys, sons of substance abusing parents were more likely to display negative mood, less flexibility and a greater propensity for social withdrawal. Furthermore, Chassin et al. (1993) found that adolescent COAs displayed heightened levels of temperamental emotionality that disposed them to negative affect states, and in turn to substance use.

Sher and his colleagues (1991) examined individual difference variables in a sample of college-aged COAs and found that COAs were more behaviorally undercontrolled (defined by hyperactivity, impulsivity, extraversion, antisociality, and sensation seeking) than were nonCOAs. Furthermore, behavioral undercontrol as well as alcohol expectancies (i.e., expected effects of alcohol) mediated the relationship between family history of alcohol problems and alcohol involvement. Thus, COAs who were more behaviorally undercontrolled and had positive expectancies for reinforcement from alcohol were more likely to use more alcohol than COAs who were not behaviorally undercontrolled and had negative alcohol expectancies.

Tarter, Blackson, Brigham, Moss and Caprara (1995) reported that sons of men with both psychoactive substance use disorder and antisocial personality disorder were more likely to be temperamentally irritable (emotionally and behaviorally dysregulated) than were sons of men with only psychoactive substance use disorder and sons of controls. A positive association was also reported between irritability and alcohol and drug use by sons as a coping response. Alcohol and drugs may act to stabilize behaviorally dysregulated affect (Tarter, Blackson et al., 1995), especially considering that behaviorally disinhibited individuals receive

positive reinforcement effects from alcoholic beverages (Sher & Levenson, 1982). In an effort to explain their findings, Tarter and his colleagues (1995) proposed the possibility of an interaction between behavioral dysregulation and social environment similar to the view proposed by Henry et al. (1996).

Results from the MSU-UM Longitudinal Study have revealed that boys with scores in the clinical range of behavior problems have more difficult temperaments, and have parents who are more antisocial and who have more lifetime alcohol problems than boys not scoring in the clinical range (Behling, 1996; Jansen et al., 1995). Furthermore, a positive association has been reported between parent temperament and child temperament (Smith, 1996), suggesting that parents pass on temperament traits to their children either genetically or through parent-child socialization factors such as the quality of the family environment.

Thus, a series of studies have been conducted to examine the role of temperament in the development of child behavior problems, later antisociality and substance use. And although previous studies have found temperament to be an important predictor of later problems, it must be noted that even within the alcohol literature there is great variability in what aspects of temperament researchers measure. This variability makes the compilation of information difficult and the generalizability of results from one study to the next challenging. Thus, for example, Tarter and his colleagues (Tarter, Blackson et al., 1995) assessed temperamental irritability, Sher et al. (1991) assessed behavioral undercontrol, and the MSU-UM Longitudinal Study assessed difficult temperament (Behling, 1996; Jansen et al., 1995), all in studies focusing on the role of temperament in alcohol

etiology.

Although researchers assess different temperamental attributes their goal, like that of the current study, is to identify risky individual characteristics that when paired with a risky environment produce maladaptive outcomes. Thus, many researchers measure temperament as a proxy for behavioral undercontrol since behavioral undercontrol is hypothesized to place an individual on a trajectory to later problems (Caspi et al., 1995). The current study also attempted to select a group of temperamental dimensions that are theoretically related to later problematic outcomes for COAs and that represent behavioral undercontrol. Because the Dimensions of Temperament Survey (DOTS) (Lerner, Palermo, Spiro, & Nesselroade, 1982) was used to assess temperament, the three dimensions of reactivity, activity level and adaptability/approach-withdrawal (adaptability) were selected to comprise the measure of child risky temperament. Each of these dimensions are theoretically predictive of later problematic outcomes for COAs (see Tarter, 1988 for review) and for this reason were used in the current study.

Although the dimensions of activity level, reactivity, and adaptability were assessed by the DOTS, risky temperament is theoretically different from difficult temperament. Difficult temperament is the construct most researchers assess when using the DOTS and is comprised of high activity level, high reactivity, arrhythmicity, low adaptability, and short attention span. Difficult temperament is relatively stable across time. Results from the MSU-UM Longitudinal Study have indicated more continuity than discontinuity in difficult temperament in a sample of children across 3 years of time (Behling, 1996). Risky temperament, as

conceptualized in the current study, is comprised of high activity level, high reactivity, and high adaptability (not low adaptability as with difficult temperament). This triad of risky temperament dimensions was chosen because it represents one perspective on individual risk factors for later problems (e.g., Tarter, 1988). For example, in a review on individual risk factors, Tarter (1988) noted that alcoholics and pre-alcoholics have a high activity level, are high on emotionality (equivalent of reactivity in the present study) and are gregarious, outgoing, and sociable (equivalent of adaptability in the present study). However, what seems to be sociability at first glance may really be behavioral undercontrol, although more research is necessary before any firm conclusions are drawn (Tarter, 1988).

Given the importance of the combination of individual risk factors and environmental risk factors in producing maladaptive outcomes for COAs, child risky temperament was examined in the current study. Based on findings indicating that COAs are at heightened risk for risky temperament (see Ellis et al., 1996), it was expected that a dense lifetime familial alcohol risk structure would directly predict increased levels of child risky temperament. Furthermore, based on views of children as elicitors of responses from the environment (e.g., Lerner & Lerner, 1983; Scarr & McCartney, 1983; Thomas & Chess, 1977), it was hypothesized that child risky temperament would contribute to worsening the quality of the family environment by increasing conflict and decreasing cohesion and expressiveness among family members. Therefore, it was expected that child risky temperament would both directly and indirectly predict child externalizing behavior problems.

In conclusion, the following study examined the relationships among lifetime

familial alcohol risk structure, parental stress and psychopathology, quality of the family environment (operationalized by conflict, cohesion, and expressiveness), child temperament, and child behavior problems over two waves of time. Based on risk aggregation theory, it was hypothesized that families with a dense lifetime alcohol risk structure would experience more parental stress and psychopathology, more family conflict and less cohesion and expressiveness, and have a child with a risky temperament. Increased levels of parental psychopathology would be directly related to a family environment characterized by conflict, low cohesion and low expressiveness and more parental stress. Risky child temperament would also be associated with a conflictual family environment that is low in cohesion and low in expressiveness (Scarr & McCartney, 1983). Finally, child externalizing behavior problems would be directly predicted by all of the following; higher levels of parental stress and psychopathology, a poorer quality family environment, and child risky temperament.

STATEMENT OF THE PROBLEM

Children of alcoholics are a population at risk for a variety of negative outcomes including externalizing behavior problems. However, the variability in COA outcomes suggests that one variable in isolation cannot fully explain problem behavior development. Rather, a multifactorial, multicausal approach must be utilized and the combined contributions of ecological, social, and individual factors must be examined in the study of the etiology of behavior problems (Fitzgerald et al., 1994; 1995; Tarter, Moss, & Vanyukov, 1995; Tarter & Vanyukov, 1994; Zucker, 1994; Zucker et al., 1995; Zucker & Gombert, 1986).

Although numerous researchers have examined antecedents of behavior problems in COAs, few have examined the simultaneous contribution of multiple level factors to behavior problems in very young COAs. Furthermore, most studies on the etiology of problem behavior development have been cross-sectional and, thus, true developmental progression has not been observed. The present study examined the associations among multiple level factors and problem behavior development in young sons of alcoholics. Two waves of data, with three years between each wave, were used so that change and stability (i.e., developmental progression) in the variables could be observed. At the first wave of data collection, boys were 3-0 to 5-11 years of age and at the second wave they were 6-0 to 8-11 years of age. The young age of the children allows exploration of the early developmental antecedents of problem behaviors and thus, the mapping of the trajectory of problem behaviors for sons of alcoholics.

Specifically, the present study examined the relation among the following

variables and child externalizing behavior problems: The contextual variable of SES; the parent lifetime variables of alcohol problems and antisociality; the parent current variables of stress and psychopathology; the family milieu variables of conflict, cohesion, and expressiveness; and the individual level variable of child temperament. Based on risk aggregation theory, it was expected that a dense lifetime familial alcohol risk structure would predict more parental stress and psychopathology, a worse quality family environment, and child risky temperament. In addition, child risky temperament would contribute to worsening the quality of the environment as would heightened levels of parental psychopathology. Parental psychopathology would also lead directly to more parental stress. Finally, all these associations would simultaneously predict more child externalizing behavior problems (see Figure 1 for hypothetical model).

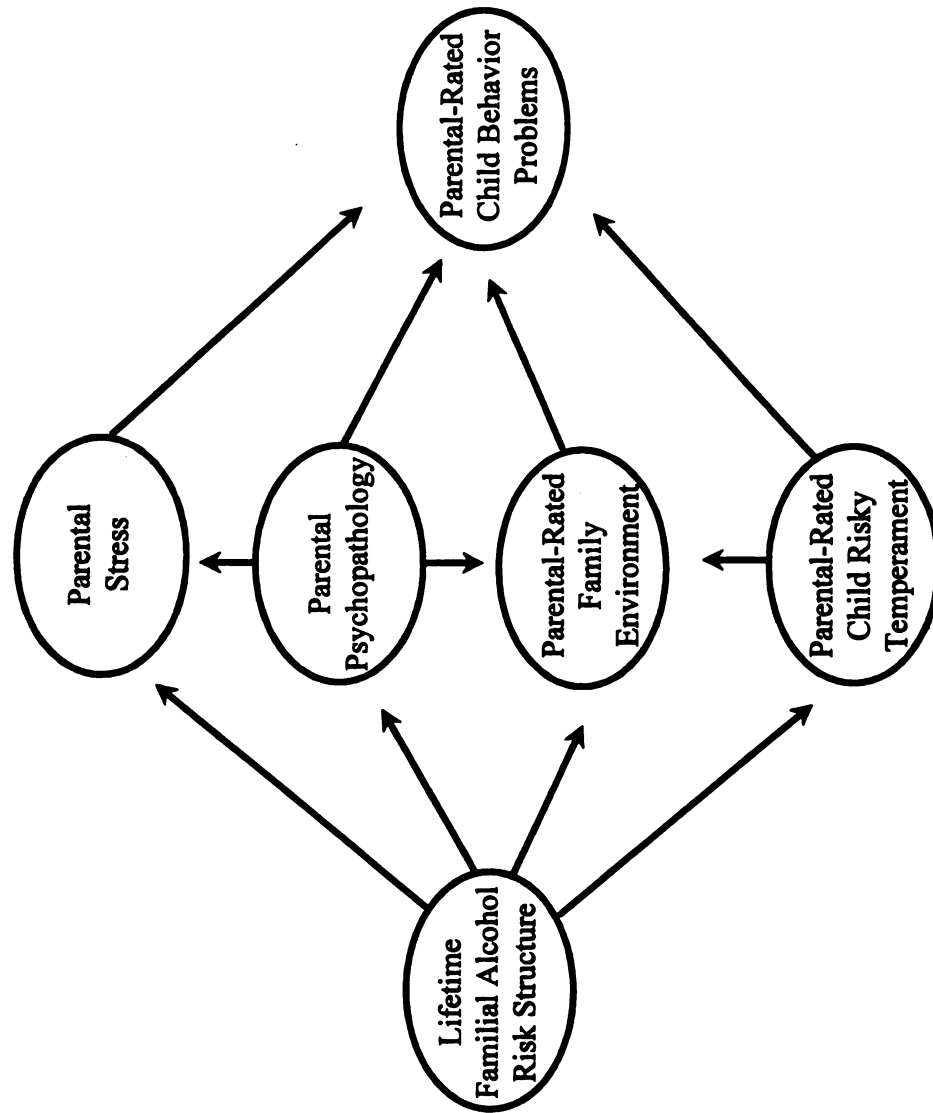


Figure 1. Model representing hypothesized relationships among latent constructs.

HYPOTHESES

The following hypotheses were based on risk aggregation theory:

Hypothesis 1. A more dense lifetime familial alcohol risk structure would directly predict increased levels of parental stress and psychopathology, a poorer quality family environment, and more child risky temperament. In addition, increased levels of parental psychopathology would predict more parental stress and a poorer quality family environment. Child risky temperament would also predict a poorer quality family environment. Lastly, it was expected that increased levels of parental stress and psychopathology, a worse quality family environment, and child risky temperament would simultaneously predict increased levels of child externalizing behavior problems.

Hypothesis 2. High levels of externalizing behavior problems would be maintained across time in families where parental SES remained low, parental alcoholism, alcohol consumption, psychopathology, and stress levels remained high. Elevated levels of child externalizing behavior problems would also be maintained in families where the quality of the family environment remained poor and child risky temperament was maintained across time.

METHOD

Participants

Subjects for the present study were 208 families participating in Waves 1 and 2 of the MSU-UM Longitudinal Study (Zucker & Fitzgerald, 1991b). This ongoing longitudinal project utilizes population-based recruitment strategies to access alcoholic men and their families and a contrast group of families with non-substance abusing parents. One hundred and twenty-five families were alcoholic whereas 83 were non-alcoholic controls. All families were Caucasian. The limited ethnic/ racial composition was dictated by the fact that census data in the area where data collection took place indicated that other ethnic and racial groups would represent less than 10% of the sample. Given the extensive literature demonstrating a substantial relationship between patterns of alcohol involvement and ethnic/ racial status and the fact that effective analyses for such differences could not be undertaken with the proposed study sample size, it was decided to exclude such variation rather than have it contribute to error. Families were invited to participate in a long term study of family health and child development and all received some payment for participation.

At the time of recruitment, all alcoholic fathers were required to have a 3-0 to 5-11 year old son with whom they were living and also to be residing with the child's mother. Mothers' drinking status was assessed, but maternal alcoholism was neither a requirement nor a basis for exclusion. In accordance with study exclusion criteria, no child manifested characteristics sufficient for a diagnosis of fetal alcohol syndrome (FAS). Assessment for potential FAS, conducted by the data collector

responsible for child assessment measures, involved evaluation of the three areas identified by Cooper (1987) as determining a FAS diagnosis (i.e., prenatal/ postnatal growth retardation, central nervous system involvement and characteristic facial dysmorphology). In instances where a symptom pattern suggested a possible diagnosis, the case was staffed by a clinical evaluation team and a consensus diagnosis was reached.

Alcoholic families were recruited by way of father's drinking status.

Alcoholic fathers were identified in one of two ways. The first group ($n = 75$) was recruited from the population of all convicted drunk drivers in a four county area in mid-Michigan. Thereafter, all males meeting the family recruitment criteria involving child age and coupling status who had a blood alcohol concentration (BAC) of 0.15% (150 mg/100 ml) or higher when arrested, or a BAC of 0.12% if a history of prior alcohol-related driving offenses existed, were asked for permission to have their names released for contact by study staff. Seventy-nine percent agreed to have their names released, and of those, 92% agreed to participate. At initial contact, a positive alcoholism diagnosis was established using the short Michigan Alcoholism Screening Test (SMAST; Selzer, 1975); this diagnosis was subsequently verified by way of the NIMH Diagnostic Interview Schedule-Version III (DIS; Robins, Helzer, Croughan & Ratcliff, 1980). All of these men met a "definite" or "probable" criterion for alcoholism using the Feighner Diagnostic Criteria (Feighner, Robins, Winokur, Guze et al., 1972), with 92% making a "definite" diagnosis. Later, DSM-III-R diagnoses were also established. Although this was not a basis for study inclusion, 73% of the alcoholic men met either moderate or severe alcohol

dependence criteria.

The second strategy involved recruiting alcoholic fathers out of the same neighborhoods where drunk driver alcoholic fathers resided. These families ($n = 50$) were accessed during neighborhood canvasses for nonalcoholic (control) families (see below). Thus, they provided an ecologically comparable subset of high risk families drawn out of the same social stratum as the drunk drivers, but where the alcoholism was identified by way of community survey rather than by way of legal difficulty. The alcoholic fathers also met Feighner criteria for probable or definite alcoholism (85% made a definite diagnosis), had children and partners who met the same inclusion criteria as the drunk driving group, but had no drunk driving or drug involved arrest record occurring during the lifetime of the 3 to 5 year old male target child.

In addition to alcoholic families, a group of community control families were recruited via door-to-door community survey techniques. These families ($n = 83$) were recruited out of the same neighborhoods as alcoholic families and were homogenous with them for age of the target child (± 6 months). However, neither parent met Feighner criteria for alcoholism or for other drug abuse/ dependence. In addition, efforts were made to match control families with alcoholic families on the basis of family SES by recruiting controls from the same neighborhood in which the risk family lived. Canvassers initiated a door-to-door search a block away from the alcoholic family, staying within the same census tract, and screened for non-alcoholic families with a child of appropriate age. However, in some cases locating a neighborhood control proved impossible due to high levels of drug and/ or alcohol

abuse among potential control families living in neighborhoods where the alcoholic families resided. In such cases, the recruitment moved to an adjacent neighborhood and in some instances it was necessary to go even more broadly afield in order to locate another sociodemographically comparable community in which to continue the search. Ninety-three percent of families who met eligibility criteria as controls agreed to participate.

Procedure

Data were collected by trained project staff who were blind to family risk status. Because of the large volume of data collected, a number of contacts with the family were necessary. Waves 1 and 2 data collection took place across nine data collection sessions, seven of which took place in the family home and two of which took place on a university campus. At each wave, the visits involved approximately fifteen hours of contact time for each parent and seven hours of time for the target child. Contacts included questionnaire sessions, semi-structured interviews and interactive tasks.

Families were contacted for the first time (Wave 1) when the target children were 3-0 to 5-11 years old. Wave 2 data collection occurred three years after initial contact, when the target children were 6-0 to 8-11 years old.

Instruments

Parent Measures

Lifetime Familial Alcohol Risk Structure

Lifetime familial alcohol risk structure was composed of the following measures: Family occupational prestige; parental antisocial behavior; and parental

lifetime alcohol problem use.

Family occupational prestige. Occupational prestige was assessed at Wave 1 and Wave 2 by the Duncan Socioeconomic Index RDSEI-TSEI2 (Stevens & Featherman, 1981), an occupation-based measure of occupational prestige. This measure was selected as an occupation-based measure of contemporary SES (Mueller & Parcel, 1981). Information regarding work locale, duties, and type of employer was obtained during administration of the Demographic Questionnaire and used to obtain U.S. Census occupation codes. Following coding, a RDSEI score was assigned to each parent and one of three scoring procedures were then used depending on the working status of the parents in each family. If both parents worked, an average occupation score was used to measure family SES. If only one parent was employed, the parent's RDSEI score was used to indicate family SES. A score of 13.0 was assigned to families where both parents were unemployed and it was also applied to housewives even though there are studies indicating that housewives are different and difficult to compare with other professions (Hauser, personal communication, 1993). These scores were reversed scored so that higher scores on this measure reflect a *lower* occupational prestige.

Parental antisociality. The Antisocial Behavior Checklist (ASB; Zucker & Noll, 1980) is a 46-item revision of an earlier antisocial behavior inventory used in the Rutgers Community Study (Zucker & Barron, 1973) that has been modified so that items are salient for both adult and adolescent antisocial activities. The ASB measures the frequency of the parent's participation in a variety of aggressive and antisocial activities both in adolescence and adulthood. The scores for each item

range from Never (0) to Often (3). Higher scores on the ASB reflect more antisocial behavior.

A series of reliability and validity studies on populations ranging from male and female college students to male and female prison inmates has shown that the ASB has adequate test-retest reliability (.91 over 4 weeks) and internal consistency (coefficient alpha = .93) (Zucker & Noll, 1980). The ASB also differentiates among groups with major histories of antisocial behavior (e.g., inmates) versus individuals with minor offenses in district court versus university students (Zucker & Noll, 1980), and between alcoholic and nonalcoholic adult males (Ham, Zucker, & Fitzgerald, 1993). The ASB was completed by both parents at Wave 1 only. Internal consistency of the ASB for the present sample was high (Wave 1 coefficient alpha = .88).

Parental lifetime alcohol problem use. The Lifetime Alcohol Problems Score (LAPS; Zucker, 1991) was the primary lifetime alcohol involvement variable used in the present analyses. The score was designed to assess differences in the extent of drinking problems over the life course, and was derived from information gained from the administration of the Drinking and Drug History Interview (Zucker, Fitzgerald, & Noll, 1990), the DIS (Robins et al., 1980), and the short form of the Michigan Alcoholism Screening Test (SMAST; Selzer, 1971, 1975). The LAPS 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 were reported; and (c) the

life percent component, involving a measure of the interval between the most recent and the earliest drinking problems, corrected for current age. Higher scores on LAPS reflect more problems related to drinking. Scores were standardized separately for males and females within the project sample; e.g., a female score identical to a male score indicates that the female has fewer problems 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 in a wide variety of areas (Zucker, 1991). This lifetime measure of alcohol problems was calculated only at Wave 1 for both mothers and fathers.

Due to the fact that LAPS is a lifetime measure of alcohol problems it was not appropriate for use in analyses requiring both Wave 1 and Wave 2 scores. In the present study, all structural equation models were analyzed using lifetime information pertaining to alcohol problems. However, all "change score" analyses required the utilization of both Wave 1 and Wave 2 measures of the same instrument and thus, a current measure of parental alcoholism was used and is described below.

Parental alcoholism. As described previously, all parents completed the SMAST (Selzer, 1975) and the DIS - Version III (Robins et al., 1980). The SMAST is a well validated screening inventory used to assess alcohol problems. It consists of 13 items that are answered with either a "Yes" or "No" response. The DIS - Version III is a structured interview that allows trained lay interviewers to gather extensive information about physical, alcohol-related, and drug-related symptoms, as well as other areas of mental health. This information can then be

used to make diagnoses by way of popularly used nosological systems. Although the entire DIS was administered to each parent, only the information from the alcohol use section was used in this study.

The Drinking and Drug History Questionnaire (Zucker, Fitzgerald, & Noll, 1990) was also administered to the parents. This instrument gathers information from the subject about his/ her use of alcohol and other drugs. Only information from the alcohol section was used in the present study. This section focuses on the amount of alcohol usage in the past six months. In the Lifetime Version of this instrument (given the first time the family entered into the study), the questionnaire also inquires about the largest amount of alcohol consumed during a 24-hour period at any point in the participant's life. At later time waves, the alcohol section assessed use in the last three years, in addition to the last six months. Finally, the instrument asks the participant whether s/he has experienced various problems as a result of alcohol usage, and if so, the frequency of these problems (during one's lifetime for the Lifetime Version, and during the last one year and last three years for other versions).

Diagnoses regarding mother's and father's alcoholism at Waves 1 and 2 were made by a trained clinician using DSM-IV criteria based on information provided by each parent on the SMAST, and the DIS - Version III, and the Drinking and Drug History. Diagnoses were made over the following periods: lifetime, past three years, and the past year. A positive lifetime diagnosis for alcoholism was made if the individual met at least DSM-IV Alcohol Abuse criteria during their child's life. For purposes of the present study, Wave 1 and Wave 2 information regarding diagnoses

for the past three years was used.

Parental Psychopathology

Parental psychopathology at Wave 1 and Wave 2 was assessed by the short form of the Beck Depression Inventory (BDI; Beck, Rial & Rickels, 1974), an Index of Alcohol Consumption, and an Index of Maximum Alcohol Consumption.

Parental depression. The 13 items of the self-reported short form BDI were rated by each parent on a scale ranging from zero to three with zero reflecting higher functioning and three reflecting lower functioning. This measure focuses on various areas of functioning known to be affected by depression, such as mood, appetite and sleep; considerable evidence supports its reliability and validity (Beck, Steer & Garbin, 1988). Items were summed and higher scores reflect more current depression.

Parental alcohol consumption. Current parental alcohol consumption was assessed via a questionnaire which incorporated items from the American Drinking Practices Survey (Calahan, Cisin, & Crossley, 1969), the 1978 National Institute on Drug Abuse High School Survey (Johnston, Bachman, & O'Malley, 1979), and the V.A. Medical Center Research Questionnaire (Schuckit, 1978). This questionnaire contained information necessary to code the QFV-R, a revision of the Quantity-Frequency-Variability (QFV) Index (Calahan et al., 1969). The QFV was a five-category variable derived by combining the Quantity-Variability (QV) classification with the 11 category frequency classification. The QFV-R uses the basic Calahan and associates scoring system; however, a score that more finely discriminates between different levels of alcohol consumption, and more accurately characterizes

drinking variability at high consumption levels was derived by multiplying the QV class times the approximate number of drinking episodes per year (based on the reported average frequency). This procedure yielded a QFV-R score ranging from 0 (no drinking) to 21,000 (high combined quantity and frequency of drinking). Thus, higher scores reflect more current alcohol consumption

Parental maximum alcohol consumption. Information concerning the largest amount of alcohol consumed during a 24-hour period was assessed by a single item on the Drinking and Drug History Questionnaire (Zucker et al., 1990). The Drinking and Drug History Questionnaire was administered to both parents at both Wave 1 and Wave 2 and is described extensively above. Higher scores on this measure reflect higher levels of maximum alcohol consumption.

Parental Stress

In the present study, parental stress was measured at Wave 1 and Wave 2 by the Daily Hassles and Uplifts Scale (Kanner, Coyne, Schaefer, & Lazarus, 1981) and the Family Crisis List (Patterson, 1982).

Parental daily hassles stress. The Daily Hassles and Uplifts Scale (Kanner et al., 1981) was used at both Wave 1 and Wave 2 to assess the daily stress experienced by parents. This instrument is comprised of two scales: The 117-item hassles scale; and the 135-item uplifts scale. The hassles scale measures daily minor stresses that characterize everyday life, while the uplifts scale measures pleasures that characterize everyday life. Each scale is presumed to be related to the individual's adaptive functioning (Kanner et al., 1981). If endorsed, items for both scales were rated in severity from Somewhat Severe (1) to Very Severe (3). Two

different scores can be derived from these self-ratings: Frequency (number of items endorsed) and intensity (cumulated severity divided by frequency). For the purposes of the present study, only the frequency score from the hassles items was used (higher scores reflect greater levels of daily hassles stress). Test-retest correlations are high for frequency scores (e.g., hassles .79; Kanner et al., 1981).

Parental life events stress. The Family Crisis List was used to assess the extent of parental life events stress (Patterson, 1982). The Family Crisis List is a 70-item self-report questionnaire that is completed by each of the parents at both Wave 1 and Wave 2. This questionnaire was developed at the Oregon Social Learning Center as a measure of family-related stressors (Patterson, 1982), and is divided into seven areas of stress: Family; household and transportation; economics; health; school; social interchange; and legal. A score of one was given to each item that was endorsed. For purposes of the present study only select items pertaining to parental life events stress, and not to daily stress, were used. Eleven items were chosen to represent parental life events stress. These 11 items were selected through a series of factor analyses performed in a previous study (see Loukas, 1995 for results). Examples of the items are; went to apply for welfare or unemployment funds, family member was arrested, got evicted, moved, conflict with ex-spouse, family member appeared in court. The sum of endorsed items reflect the amount of life events stress perceived by each parent.

Family Environment

Conflict, cohesion, and expressiveness. Conflict, cohesion, and expressiveness in the family environment at Wave 1 and Wave 2 were assessed with the Family Environment Scale, Form R (FES; Moos, 1974; Moos & Moos, 1976). The FES is an empirically based taxonomy of family social environments as perceived by family members themselves. It requires fifth or sixth grade reading skills. Form R of the FES consists of a number of scales which describe dimensions of the family climate with which each individual member must cope. The scores on the subscales yield a profile with the family as a central focus; or they may be used to compare the extent of agreement between family members; or they may be used to compare and contrast different family groups. The instrument has been subjected to extensive reliability and validity studies. The instrument provides scores in areas that have previously been significantly implicated in alcoholic and drug abusing families: e.g., Cohesion, Conflict, Expressiveness, Moral-Religious Emphasis, Achievement Orientation. Internal consistency reliability ranges from .64 to .78, and test-reliability ranges from .68 to .86 across the 10 subscales of the FES. For purposes of the present study only maternal and paternal ratings of the Cohesion, Conflict and Expressiveness subscales were used. Higher scores reflect more cohesion, more expressiveness, and less conflict.

Decoupling

Information concerning the intactness of each family was obtained from the Marital Status Index, an instrument developed by the MSU-UM Longitudinal Study. Decoupling information was collected by the family assessor for the first time at the

first session of each Wave and for a second time at the last session of each Wave. Parents indicated their marital status (e.g., married, separated, divorced), as well as their living situation (living with original partner and child, living with child but not with child's biological parent etc.). For the purposes of this study, only the data gathered at the last session of each of the two Waves were used. As indicated by the inclusion criteria, all families were intact at the beginning of Wave 1. Therefore, Wave 2 information was used to complete the post-Wave 1 decoupling variable. The decoupling variable was dichotomous with a score of one indicating that the family was intact (parents were coupled) living with the biological target child. A score of two indicated that the family was not intact (i.e., only one parent was living with the target child by Wave 2, while the other parent was living elsewhere due either to a separation of choice such as a divorce).

Child Measures

Child Risky Temperament

Child temperament was assessed at Wave 1 and Wave 2 by the Dimensions of Temperament Survey (DOTS; Lerner et al., 1982). This survey is a 34-item questionnaire that measures five dimensions of temperament: Activity Level, Attention Span/Distractibility, Adaptability/Approach-Withdrawal, Rhythmicity, and Reactivity. The dimension scores are based on the sums of each item score (1 'true' or 0 'false'). For the purposes of the present study only mother and father ratings of child activity level, adaptability, and reactivity were used. These three dimensions of temperament are theoretically the most related to child externalizing behavior problems (Tarter, 1988) and for each dimension higher scores reflect more activity,

adaptability and reactivity.

Cronbach's alphas obtained from the ratings of preschoolers' mothers and the self-ratings of school-aged children and young adults on the five scales showed moderate to high internal consistency ranging from .31 to .96, with reactivity the only factor that was consistently below .60. In addition, stability coefficients conducted on the various age groups ranged from .60 to .93 (Lerner et al., 1982). Previous studies support the validity of the DOTS, showing that it covaries with the ratings of academic ability, adjustment, and peer relations (Windle & Lerner, 1984).

Child Externalizing Behavior Problems

Child behavior problems was assessed at Wave 1 and Wave 2 with the 118-item Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL is an objective assessment of the target child's social and emotional functioning. This instrument has been normed on children and adolescents 4 to 18 years of age and yields both raw and standardized scores on social competency, two broad-band subscales measuring externalizing and internalizing behavior and eight narrow-band syndrome subscales (withdrawn, somatic complaints, anxious/depressed, social problems, thought problems, attention problems, delinquent, aggressive, and an optional subscale measuring sex problems). Parents rated each of the items on a scale ranging from Not True for my child (0) to Very True for my child (2). All items are summed and higher scores reflect more behavior problems. Internal consistency of the total behavior problems score is good. Achenbach (1991) calculated the coefficient alpha to be .96 and for the MSU-UM Longitudinal Study sample, the coefficient alpha is .93. The CBCL also has good content validity and

distinguishes between clinically-referred and non-referred children (Achenbach, 1991). Wave 1 and Wave 2 parental ratings of the broad-band subscale of raw externalizing behavior problems (aggressive and delinquent narrow bands) were used for the present study.

Missing Data Estimation

The original data set for the present analyses consisted of 211 families (mothers, fathers, and children). Of the 211 families three were missing 21 or more scale scores out of a possible 42; therefore, were not included in the sample for the present analyses. Data imputation was completed for all missing scale scores of the remaining 208 families that comprised the final sample.

Prior to estimating missing scale scores all instruments were examined for missing individual scale items. The number of individual items missing relative to the total number of items within each scale was very small; therefore, rather than deleting all data and estimating at the scale level, the available items were prorated and used as estimates of the scale scores (Rovine & Delaney, 1990).

Scale scores were estimated using two different procedures. A longitudinal data estimation procedure developed by Petersen (1987; see Bingham, 1993; Bingham & Crockett, 1996) was used to estimate the missing scale scores for instruments that were administered at both Wave 1 and Wave 2. For those instruments that were only administered at Wave 1, or for individuals missing both Wave 1 and Wave 2 scores of the same instrument cross-sectional ordinary least squares regression estimation was run on available non-missing data. All data imputation was completed separately for mothers and fathers in each of three risk

groups (see Ellis et al., 1996 and Loukas, 1995 for discussion of these groups; see Appendix A for detailed description of imputation procedures).

Bias in Estimation

Upon completion of data estimation, analyses were conducted in order to ensure that data estimation had not biased the sample. For each latent construct at each of the two waves of analysis, a two-group, stacked model design was tested using LISREL 8 (Jöreskog & Sörbom, 1993). One group consisted of the original data set (with missing data points), while the other group consisted of estimated data combined with the original data set. The first two moments (covariance and means) were estimated and all parameter estimates for both groups were constrained to be invariant.

Analyses were conducted separately for the following latent constructs; parental psychopathology (composed of parental depression, parental maximum drinks in 24 hours, parental alcohol consumption), parental stress (composed of parental daily hassles stress and parental life stress), child risky temperament (composed of activity level, adaptability, and reactivity), and child externalizing behavior problems (composed of delinquency and aggression). Each analysis resulted in a Goodness of Fit Index (GFI) equal to 1.00 and a non-significant Chi-Square, with the exception of the Wave 1 Psychopathology analysis (see Table A1 of Appendix A for fit statistics).

The Wave 1 Psychopathology analysis resulted in a significant Chi-Square and a GFI of .99. These results were likely due to the large number of data points missing for Wave 1 maximum alcohol consumption (92/208 data points or 44% of

the total) due to a change in the instrument during Wave 1 data collection.

Although estimating a large percentage of missing data decreased the fit of the model to the data, the resulting fit was still very good. Taken together, results of all analyses indicated that the data estimation procedure did not significantly alter the structure of the data.

RESULTS

Background Characteristics

A summary of the sample background information is presented in Table 1. For the purposes of sample description and for these analyses only, the sample is divided into AAL, NAAL, and Control risk groups. These groupings are based on paternal antisociality and lifetime alcohol problems scores (see Zucker et al., 1994; 1996). Five one-way Analyses of Variance (ANOVAs) with the dependent variables of maternal age, maternal years of education, paternal age, paternal years of education, and child age were used to examine differences among the risk groups.

Results revealed that only maternal years of education and paternal years of education differed significantly across the three risk groups (see Table 1 for F -tests). Student-Newman-Keuls post-hoc tests revealed that mothers in both the Control and NAAL groups had significantly more years of education than did mothers in the AAL group. There were no differences on years of education between mothers in the Control group and mothers in the NAAL group. For fathers, the post-hoc tests revealed that Control fathers had the most years of education, followed by the NAALs, followed by the AALs.

Structural Equation Modeling

The first hypothesis proposed that for both maternal and paternal data a more dense lifetime familial alcohol risk structure would directly predict more parental stress, more parental psychopathology, a worse quality family environment, and more child risky temperament. Furthermore, parental psychopathology would increase parental stress and along with child risky temperament worsen the quality

Table 1

Age in Years and Total Years of Education of Current Sample at Wave 1

Variable	Risk Group						F
	AAL		NAAL		Control		
	<u>n</u>	<u>M(SD)</u>	<u>n</u>	<u>M(SD)</u>	<u>n</u>	<u>M(SD)</u>	
Mothers							
Age	41	30.4(4.5)	84	31.8(3.6)	83	31.4(3.9)	1.76
Education	41	12.6(1.8)	84	13.6(2.2)	83	13.7(1.8)	3.82* ^{ab}
Fathers							
Age	41	33.9(6.3)	84	33.7(4.8)	83	32.8(4.6)	0.85
Education	41	12.7(1.8)	84	13.9(2.3)	83	14.6(2.1)	11.16** ^{abc}
Children							
Age	41	4.3(0.9)	84	4.2(1.0)	83	4.2(1.0)	0.38

^a AALs < Controls, Student-Newman-Keuls Test^b AALs < NAALs, Student-Newman-Keuls Test^c NAALs < Controls, Student-Newman-Keuls Test

*p<.05. **p<.001.

of the family environment. These variables, in turn, would directly predict more child externalizing behavior problems. This hypothesis was tested using structural equation modeling (SEM).

LISREL 8 (Jöreskog & Sörbom, 1993) was used to obtain the maximum likelihood estimates of the model coefficients and a covariance matrix was analyzed. Initially, all SEMs were conducted separately for mothers and fathers at each of the two waves of data collection. These analyses were followed by stacked two-group models, one at each time point simultaneously analyzing maternal and paternal data. The first set of SEM analyses was conducted with the entire data set (208 mothers and 208 fathers).

Measurement Models

All measurement models consisted of 6 latent variables and 18 indicator variables (see Figure 2 for hypothetical measurement model). The latent constructs were lifetime familial alcohol risk structure, parental stress, parental psychopathology, quality of the family environment, child risky temperament, and child externalizing behavior problems. Familial lifetime alcohol risk structure was composed of family occupational prestige, parental lifetime alcohol problems, and parental antisociality. Parental psychopathology was composed of maximum alcohol consumption, alcohol consumption, and depression. Parental stress was composed of life events stress and two factors created from the daily hassles stress scale. Child risky temperament was composed of three scales that contained a random distribution of the items that constitute activity level, adaptability, and reactivity on the DOTS (Lerner et al., 1982). The latent construct of family environment

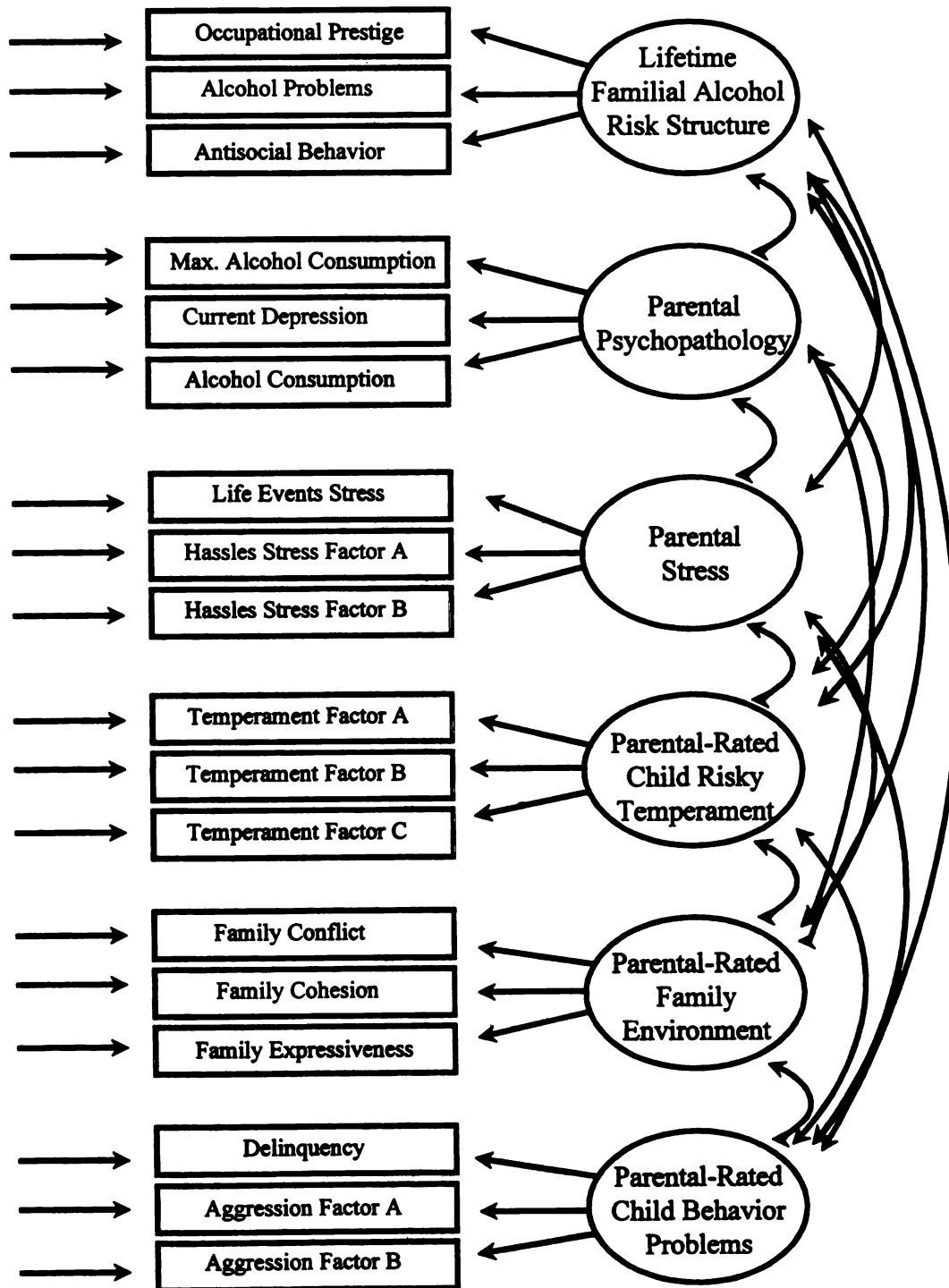


Figure 2. Hypothetical measurement model.

consisted of family conflict, cohesion, and expressiveness and child externalizing behavior problems consisted of child delinquency and two factors created from the child aggression narrow band of the CBCL (Achenbach, 1991).

SEMs and Entire Sample

First, four measurement models were tested for the entire sample; one for each parent at each of the two time periods. Tables 2-5 contain the means, standard deviations, and correlations for variables included in the present analyses. Prior to discussing the results of the measurement models, several values found in Tables 2-5 merit some discussion. The descriptives in each of the four tables reveal that the alcohol consumption variable consistently has a much larger standard deviation (SD; at least two times as large) than mean. This skewness in the scores is due to several outliers who received very high scores on this measure (see Appendix B Figures B1-B4 for a graphical representation of the alcohol consumption variable). Secondly, the family environment variables of family expressiveness, conflict, and cohesion each have a very small SD relative to their means indicating very little variance in the distribution of these variables.

The adequacy of fit for all measurement and structural models was determined by considering the following indices in combination; the Chi-Square statistic, the Goodness of Fit Index (GFI), the Comparative Fit Index (CFI), and the Standardized Root Mean Square Residual (SRMR). In general, a good-fitting model is one that has a non-significant Chi-Square, a GFI and CFI of .90 or larger, and a SRMR of .099 or smaller. The critical t -value for each model was the one-tailed t -test; therefore, paths resulting in values larger than 1.645 were considered

Table 2

Means, Standard Deviations, and Correlations For All Wave 1 Paternal Indicator Variables For Entire Sample (n=208)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.18**	.34***	.23***	.22**	.15*	.19**	.09	.30***
2. Lifetime Alcohol Problems		1.00	.58***	.24***	.53***	.32***	.24***	.15*	.30***
3. Lifetime Antisocial Behavior			1.00	.34***	.43***	.26***	.29***	.23***	.36***
4. Current Depression				1.00	.14*	.13	.55***	.45***	.39***
5. Maximum Alcohol Consumption					1.00	.39***	.14*	.09	.27***
6. Alcohol Consumption						1.00	.12	.07	.20**
7. Hassles Stress Factor A							1.00	.85***	.47***
8. Hassles Stress Factor B								1.00	.26***
9. Life Events Stress									1.00
<u>M</u>	357.63	9.38	16.16	2.45	3.86	2176.45	15.90	14.17	1.13
<u>SD</u>	146.66	2.30	10.08	2.62	2.46	4905.62	12.90	12.92	1.50

*p<.05. **p<.01. ***p<.001.

(table continues)

Table 2 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.09	.01	.08	-.12	-.11	-.20**	.09	.11	.11
2. Lifetime Alcohol Problems	.22**	.01	.13	-.04	-.26***	-.14*	.24***	.31***	.34***
3. Lifetime Antisocial Behavior	.14*	-.02	.11	-.06	-.31***	-.23***	.21**	.28***	.22**
4. Current Depression	.14*	.05	.19**	-.18**	-.37***	-.34***	.20**	.29***	.30***
5. Maximum Alcohol Consumption	.21**	-.02	.07	-.09	-.07	-.10	.21**	.19**	.13
6. Alcohol Consumption	.11	.05	.02	-.08	-.13	-.16*	.24***	.21**	.24***
7. Hassles Stress Factor A	.07	-.02	.10	-.09	-.22**	-.18**	.08	.25***	.24***
8. Hassles Stress Factor B	.04	-.05	.09	.01	-.18*	-.11	.08	.20**	.24***
9. Life Events Stress	.06	.03	.04	-.15*	.22***	.23***	.16*	.09	.03
10. Child Temperament Factor A	1.00	.53***	.52***	.13	-.03	.07	.17*	.26***	.24***
11. Child Temperament Factor B		1.00	.42***	.05	-.00	.07	.22**	.19**	.20**
12. Child Temperament Factor C			1.00	.04	-.00	.05	.13	.23***	.25***
13. Family Expressiveness				1.00	.24***	.40***	-.15*	-.14*	-.12
14. Family Conflict					1.00	.39***	-.31***	-.37***	-.34***
15. Family Cohesion						1.00	-.12	-.09	-.11
16. Child Delinquency							1.00	.56***	.50***
17. Child Aggression Factor A								1.00	.78***
18. Child Aggression Factor B									1.00
M	3.24	3.00	2.50	14.95	14.65	16.47	1.78	5.13	4.71
SD	1.22	1.05	1.16	1.74	2.19	1.63	1.40	2.88	2.89

*p<.05. **p<.01. ***p<.001.

Table 3

Means, Standard Deviations, and Correlations For All Wave 1 Maternal Indicator Variables For Entire Sample (n=208)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.14*	.36***	.10	.15*	.17*	.15*	.07	.24***
2. Lifetime Alcohol Problems		1.00	.56***	.05	.43***	.36***	.25***	.20***	.29***
3. Lifetime Antisocial Behavior			1.00	.21**	.31***	.33***	.29***	.24***	.36***
4. Current Depression				1.00	.05	.01	.39***	.30***	.26***
5. Maximum Alcohol Consumption					1.00	.41***	.07	-.02	.20**
6. Alcohol Consumption						1.00	.05	.05	.25***
7. Hassles Stress Factor A							1.00	.80***	.31***
8. Hassles Stress Factor B								1.00	.23***
9. Life Events Stress									1.00
M	357.63	9.94	10.25	2.71	2.34	454.91	16.98	15.38	1.04
SD	146.66	2.05	6.87	2.87	1.75	1592.91	12.22	11.50	1.28

*p<.05. **p<.01. ***p<.001.

(table continues)

Table 3 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.01	-.14*	.04	-.22***	-.15*	-.14*	.19**	.10	.17*
2. Lifetime Alcohol Problems	-.01	-.06	.01	-.06	-.23***	-.20**	.22**	.19**	.29***
3. Lifetime Antisocial Behavior	.06	-.06	.10	-.08	-.29***	-.27***	.37***	.38***	.46***
4. Current Depression	.06	.05	.11	-.15*	-.28***	-.39***	.18*	.14*	.12
5. Maximum Alcohol Consumption	-.02	-.04	.01	-.02	-.15*	-.07	.04	-.02	.05
6. Alcohol Consumption	-.00	-.13	-.03	-.03	-.14*	-.14*	.13	.12	.16*
7. Hassles Stress Factor A	.05	.07	.09	-.13	-.23***	-.25***	.25***	.21**	.26***
8. Hassles Stress Factor B	.02	.04	.08	-.09	-.22**	-.21**	.25***	.23***	.31***
9. Life Events Stress	.02	-.02	-.03	-.16*	-.26***	-.20**	.24***	.23***	.23***
10. Child Temperament Factor A	1.00	.57***	.50***	.11	.23***	.08	.08	.16*	.21**
11. Child Temperament Factor B		1.00	.36***	.07	.17*	.08	.05	.13	.11
12. Child Temperament Factor C			1.00	.02	.14*	.08	.09	.18**	.25***
13. Family Expressiveness				1.00	.13	.37***	-.07	-.03	-.02
14. Family Conflict					1.00	.41***	-.13	-.17*	-.24***
15. Family Cohesion						1.00	-.20**	-.08	-.13
16. Child Delinquency							1.00	.52***	.47***
17. Child Aggression Factor A								1.00	.75***
18. Child Aggression Factor B									1.00
<u>M</u>	2.97	2.82	2.36	15.46	14.42	16.46	1.85	5.30	5.02
<u>SD</u>	1.28	0.98	1.17	1.70	2.09	1.73	1.61	2.81	2.97

*p<.05. **p<.01. ***p<.001.

Table 4

Means, Standard Deviations, and Correlations For All Wave 2 Paternal Indicator Variables For Entire Sample (n=208)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.18**	.34***	.23***	.20**	.08	.17*	.10	.25***
2. Lifetime Alcohol Problems		1.00	.58***	.25***	.43***	.17*	.17*	.15*	.27***
3. Lifetime Antisocial Behavior			1.00	.19**	.35***	.17*	.23***	.18**	.31***
4. Current Depression				1.00	.16*	.06	.48***	.44***	.24***
5. Maximum Alcohol Consumption					1.00	.32***	.14*	.16*	.21**
6. Alcohol Consumption						1.00	.30***	.32***	.28***
7. Hassles Stress Factor A							1.00	.87***	.25***
8. Hassles Stress Factor B								1.00	.23***
9. Life Events Stress									1.00
<u>M</u>	357.63	9.38	10.25	2.94	3.48	1224.57	14.50	13.03	0.96
<u>SD</u>	146.66	2.30	6.87	3.35	2.38	2929.88	13.51	14.03	1.27

*p<.05. **p<.01. ***p<.001.

(table continues)

Table 4 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.01	.00	.05	-.02	-.14*	-.20**	.14*	.21**	.19**
2. Lifetime Alcohol Problems	.22**	.22***	.20**	.02	-.21**	-.18**	.24***	.24***	.23***
3. Lifetime Antisocial Behavior	.16*	.12	.23***	.05	-.27***	-.26***	.18**	.22***	.25***
4. Current Depression	.15*	.13	.14	-.08	-.19**	-.33***	.16*	.29***	.25***
5. Maximum Alcohol Consumption	.17*	.10	.14*	.01	-.07	-.04	.16*	.08	.11
6. Alcohol Consumption	.15*	.02	.12	.10	-.04	-.00	.13	.10	.17*
7. Hassles Stress Factor A	.17*	.03	.08	.05	-.13	-.19**	.11	.30***	.28***
8. Hassles Stress Factor B	.10	-.04	.01	.02	-.13	-.20**	.08	.21**	.22***
9. Life Events Stress	.11	.11	.11	-.04	-.13	-.21**	.16*	.15*	.13
10. Child Temperament Factor A	1.00	.53***	.48***	.07	-.11	-.02	.19**	.23***	.24***
11. Child Temperament Factor B		1.00	.48***	-.05	-.07	-.09	.14*	.11	.14*
12. Child Temperament Factor C			1.00	.06	-.05	-.01	.17*	.15*	.23***
13. Family Expressiveness				1.00	.21**	.38***	-.05	-.11	-.03
14. Family Conflict					1.00	.43***	-.29***	-.38***	-.37***
15. Family Cohesion						1.00	-.17*	-.26***	-.18**
16. Child Delinquency							1.00	.58***	.53***
17. Child Aggression Factor A								1.00	.80***
18. Child Aggression Factor B									1.00
<u>M</u>	2.91	2.94	2.31	15.11	14.53	16.32	1.72	4.57	4.10
<u>SD</u>	1.31	1.12	1.24	1.64	2.24	1.78	1.67	2.68	2.83

*p<.05. **p<.01. ***p<.001.

Table 5

Means, Standard Deviations, and Correlations For All Wave 2 Maternal Indicator Variables For Entire Sample (n=208)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.14*	.36***	.25***	.11	.16*	.16*	.13	.22***
2. Lifetime Alcohol Problems		1.00	.56***	.11	.41***	.24***	.24***	.18**	.27***
3. Lifetime Antisocial Behavior			1.00	.29***	.35***	.32***	.33***	.24***	.32***
4. Current Depression				1.00	.17*	.21**	.51***	.39***	.17*
5. Maximum Alcohol Consumption					1.00	.36***	.15*	.12	.17*
6. Alcohol Consumption						1.00	.25***	.25***	.24***
7. Hassles Stress Factor A							1.00	.77***	.23**
8. Hassles Stress Factor B								1.00	.24***
9. Life Events Stress									1.00
<u>M</u>	357.63	9.94	10.25	3.33	2.20	604.14	14.74	13.02	1.03
<u>SD</u>	146.66	2.05	6.87	3.64	1.66	2749.09	11.61	10.98	1.34

*p<.05. **p<.01. ***p<.001.

(table continues)

Table 5 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational	.03	.05	.08	-.08	-.11	-.12	.25***	.14*	.20**
2. Lifetime Alcohol Problems	.01	.01	.06	.05	-.15*	-.17*	.13	.15*	.20**
3. Lifetime Antisocial Behavior	.14*	.10	.13	.02	-.21**	-.18**	.19**	.27***	.34***
4. Current Depression	.08	.08	.09	-.19**	-.33***	-.29***	.22**	.28***	.34***
5. Maximum Alcohol Consumption	.15*	.06	.17*	.07	-.09	-.06	.23***	.21**	.23***
6. Alcohol Consumption	.04	.04	.05	-.08	-.26***	-.24***	.26***	.26***	.23***
7. Hassles Stress Factor A	.11	.11	.07	-.09	-.34***	-.32***	.15*	.25***	.28***
8. Hassles Stress Factor B	.15*	.07	.06	-.04	-.30***	-.31***	.13	.26***	.25***
9. Life Events Stress	.03	.06	.11	-.14*	-.18*	-.15*	.21**	.20**	.23***
10. Child Temperament Factor A	1.00	.55***	.63***	.04	.08	.10	.10	.18**	.28***
11. Child Temperament Factor B		1.00	.47***	-.07	.02	-.04	.12	.17*	.21**
12. Child Temperament Factor C			1.00	.04	.06	.12	.10	.13	.23***
13. Family Expressiveness				1.00	.07	.24***	-.03	-.11	-.04
14. Family Conflict					1.00	.51***	-.26***	-.37***	-.37***
15. Family Cohesion						1.00	-.22***	-.28***	-.26***
16. Child Delinquency							1.00	.59***	.53***
17. Child Aggression Factor A								1.00	.75***
18. Child Aggression Factor B									1.00
M	2.71	2.56	2.20	15.65	14.54	16.44	1.98	4.60	4.31
SD	1.26	1.09	1.23	1.53	2.14	1.77	1.71	2.76	2.96

*p<.05. **p<.01. ***p<.001.

significant.

Preliminary to the analyses of the stacked two-group structural models, cross-sectional measurement and structural models were analyzed (see Appendix C for results of cross-sectional measurement and structural models). First, four cross-sectional measurement models were analyzed, one at each time period for mothers and fathers separately. Each of the measurement models resulted in a good fit. These measurement models were then used to construct the four cross-sectional structural models, each of which also resulted in a good fit to the data.

Overall, results from the cross-sectional structural models demonstrated that the majority of the hypothesized relationships were verified. However, one finding that was contrary to what was hypothesized, was the positive relationship between child risky temperament and quality of the family environment. That is, the models indicated that the more risky the child's temperament, the better the quality of the family environment. Because this relationship was contrary to what was expected, two exploratory analyses were conducted to test the veracity of the relationship. In the first exploratory analysis, the path from child temperament to family environment was fixed to a value of -1.0 in order to test a negative relationship. This model resulted in values that were improper; that is, values greater than 1.0 in the final completely standardized solution represent estimation errors in LISREL (Bollen, 1989). Secondly, the path from child temperament to family environment was removed and replaced with a path from family environment to child temperament. This path also resulted in a positive but non-significant relationship indicating that a better quality family environment did not significantly predict child

risky temperament. Overall, results of the exploratory analyses indicated that the positive relationship between child risky temperament and family environment was not due to estimation problems.

Stacked Two-Group Models

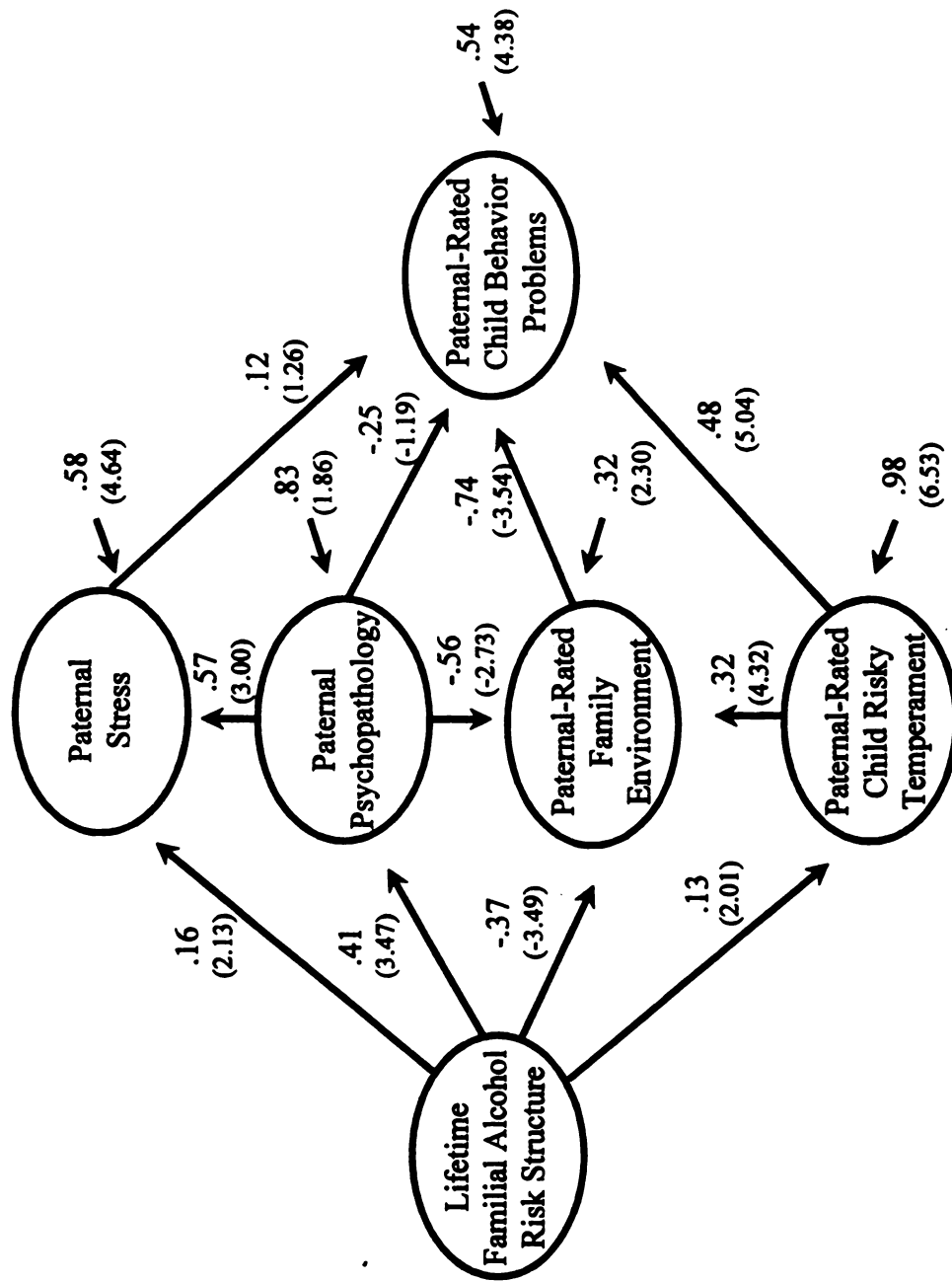
Following the estimation of the cross-sectional models, two (Wave 1 and Wave 2) two-group (maternal data, paternal data) stacked measurement models were analyzed. The best-fitting models for each of the two Waves of data collection, were models in which: (1) the loadings of the indicator variables on the latent constructs were kept invariant across the two groups, (2) the pattern of correlations among the latent constructs was invariant across the two groups but the starting values were free to vary, and (3) the uniquenesses were free to vary across the two groups. Both Wave 1 and Wave 2 stacked models fit the data. For the Wave 1 model, although the Chi-Square was significant [$\chi^2(244, N = 208) = 425.57$, $p < .001$], the GFI was .90, the CFI was .92 and the SRMR was .092. The Wave 2 model also resulted in a good fit; the Chi-Square was significant [$\chi^2(243, N = 208) = 310.41$, $p < .001$], but the GFI was .93, the CFI was .97, and the SRMR was .063 (see Appendix D Figures D1 and D2 for Wave 1 paternal and maternal measurement models and Figures D3 and D4 for Wave 2 paternal and maternal measurement models).

Next, the two-group stacked structural models were estimated. The final models fit the data well and met the following criteria; (1) the loadings of the indicator variables on the latent constructs were kept invariant across the two groups, (2) the pattern of predictive associations among the latent constructs was completely

invariant, (3) the pattern of correlations among the latent constructs was invariant across the two groups although the starting values were free to vary, and (4) the uniquenesses were free to vary across the two groups.

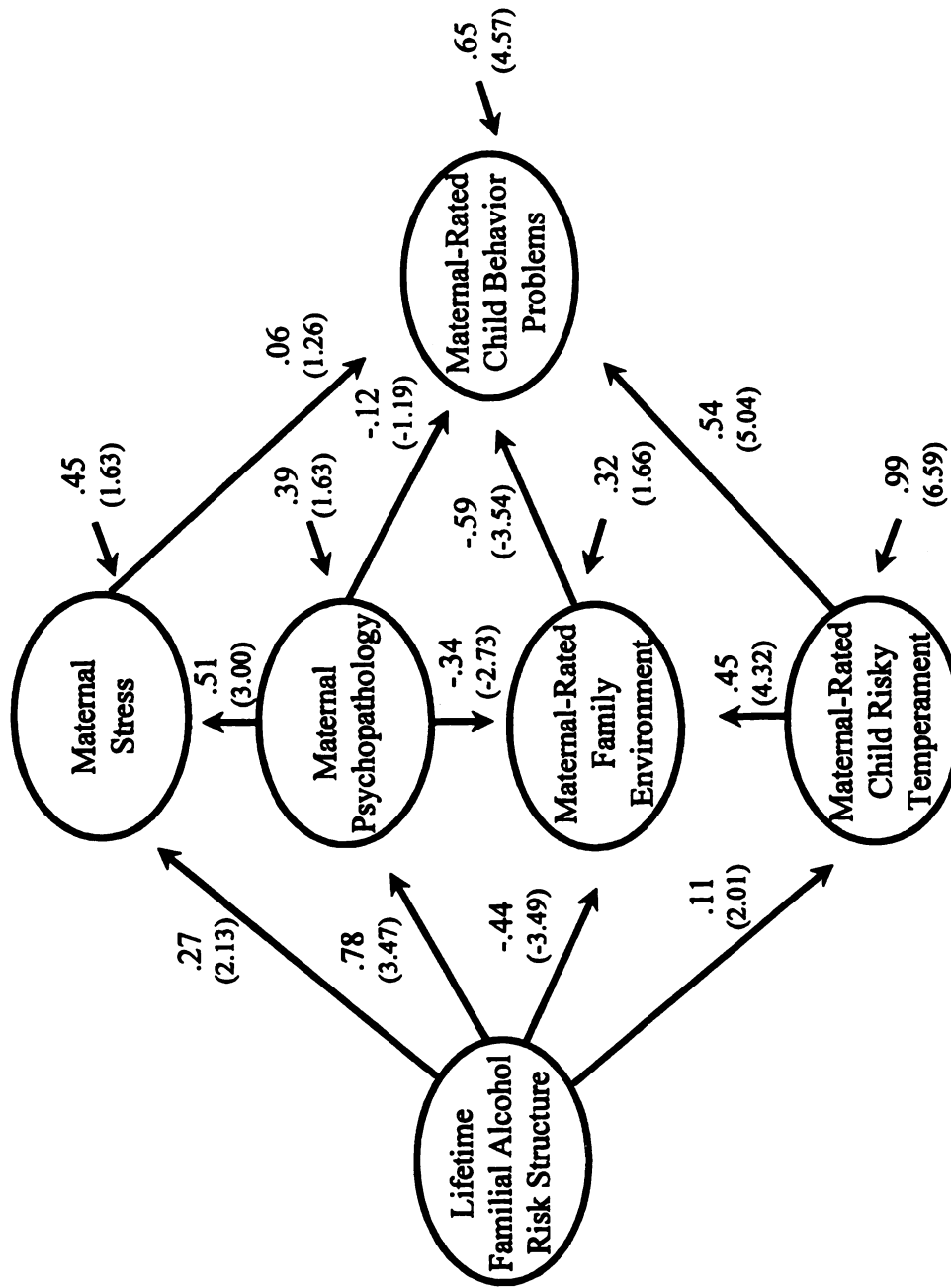
Wave 1 stacked structural model. Results for the Wave 1 two-group stacked structural model partially supported the first hypothesis and also demonstrated that the maternal and paternal models did not differ in structure (see Figure 3 for the within-group completely standardized solution for the paternal data and Figure 4 for the within-group completely standardized solution for the maternal data). The overall model fit was acceptable; although the resulting Chi-Square was significant [$X^2(259, N = 416) = 444.92, p < .001$], the GFI was .90, the CFI was .92, and the SRMR was .090.

Examination of the stacked model revealed that, for both mothers and fathers, a more dense lifetime familial alcohol risk structure directly predicted more Wave 1 parental stress, more parental psychopathology, a worse quality family environment and more child risky temperament. Increased levels of parental psychopathology predicted more parental stress and a worse quality family environment. Better quality of the family environment was predicted by child risky temperament (see Appendix E for detailed examination of the relationship between child risky temperament and family environment), and a poorer quality of the family environment and child risky temperament directly predicted more child externalizing behavior problems. Parental stress and parental psychopathology did not lead to more child externalizing behavior problems, though this was hypothesized.



Note. t-values are in parentheses.

Figure 3. Structural model from Wave 1 two-group stacked analysis: Solution for paternal data for entire sample.



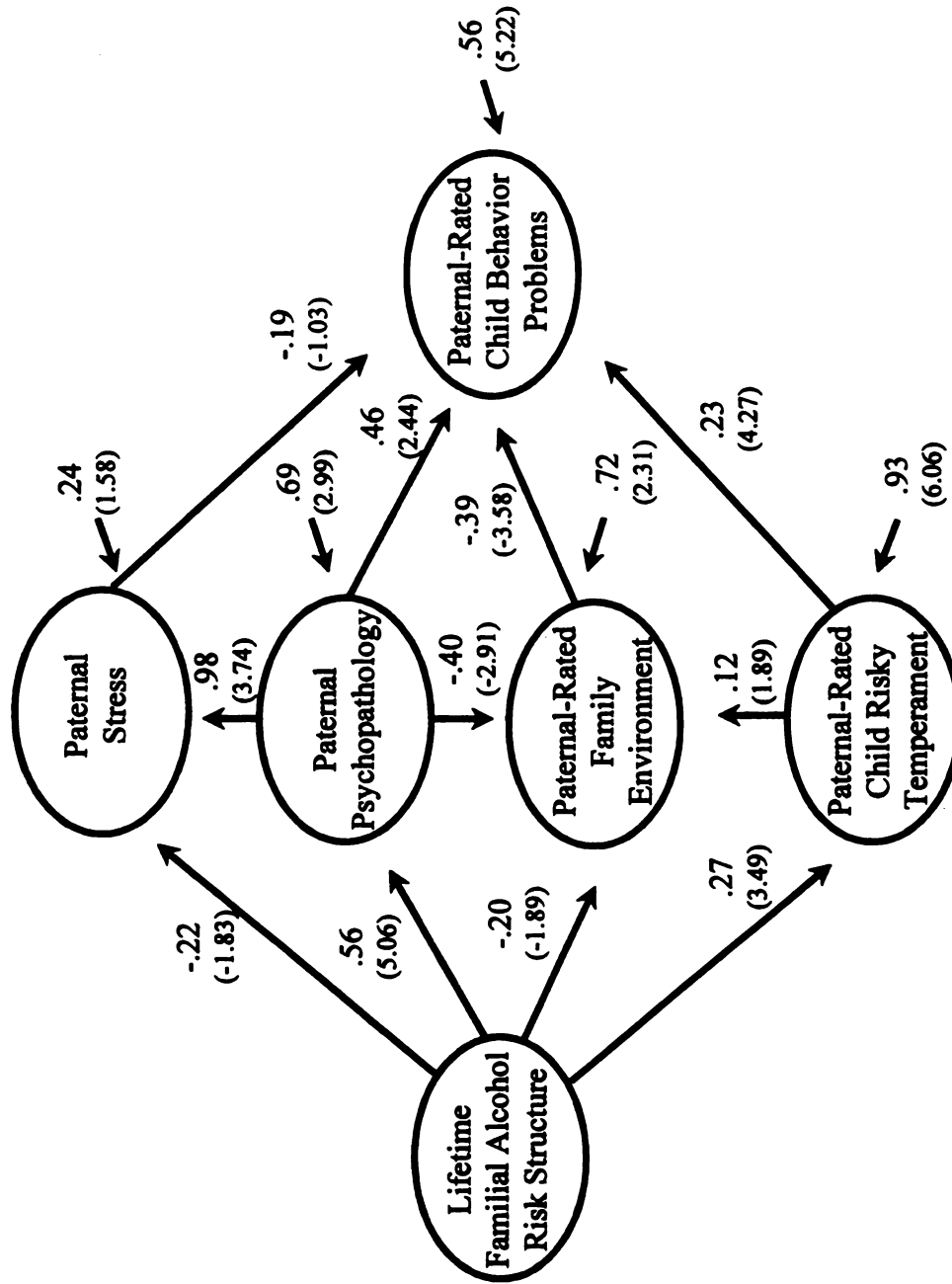
Note. t-values are in parentheses.

Figure 4. Structural model from Wave 1 two-group stacked analysis: Solution for maternal data for entire sample.

Wave 2 stacked structural model. The fit of the Wave 2 two-group stacked model was also acceptable. Although the Chi-Square was significant [$\chi^2(264, N = 416) = 387.88, p < .001$], the GFI was .91, the CFI was .95, and the SRMR was .086 (see Figures 5 and 6 for paternal and maternal within-group completely standardized solutions). Results for the Wave 2 two-group stacked structural model also partially supported the first hypothesis and demonstrated that the maternal and paternal models did not differ in structure.

Results indicated that a more dense lifetime familial alcohol risk structure directly predicted higher levels of Wave 2 parental psychopathology, a worse quality family environment, and child risky temperament. Contrary to what was hypothesized, a more dense lifetime familial alcohol risk structure also predicted lower levels of family stress (likely due to estimation problems). Increased levels of parental psychopathology predicted more parental stress and a poorer quality of the family environment. Child risky temperament predicted a better quality family environment (unexpectedly). A poorer family environment and child risky temperament both predicted increased levels of child externalizing behavior problems, however, increased levels of parental stress did not predict more child externalizing behavior problems.

Following these analyses with the entire sample, a second set of analyses, consisting of data from coupled individuals only (188 mothers and 188 fathers), was conducted. The purpose of these analyses was to examine any differences in patterns of prediction for the entire data set versus the larger subsample of coupled parents only. Prior to re-estimating the SEMs with the coupled individuals only, a



Note. t-values are in parentheses.

Figure 5. Structural model from Wave 2 two-group stacked analysis: Solution for paternal data for entire sample.

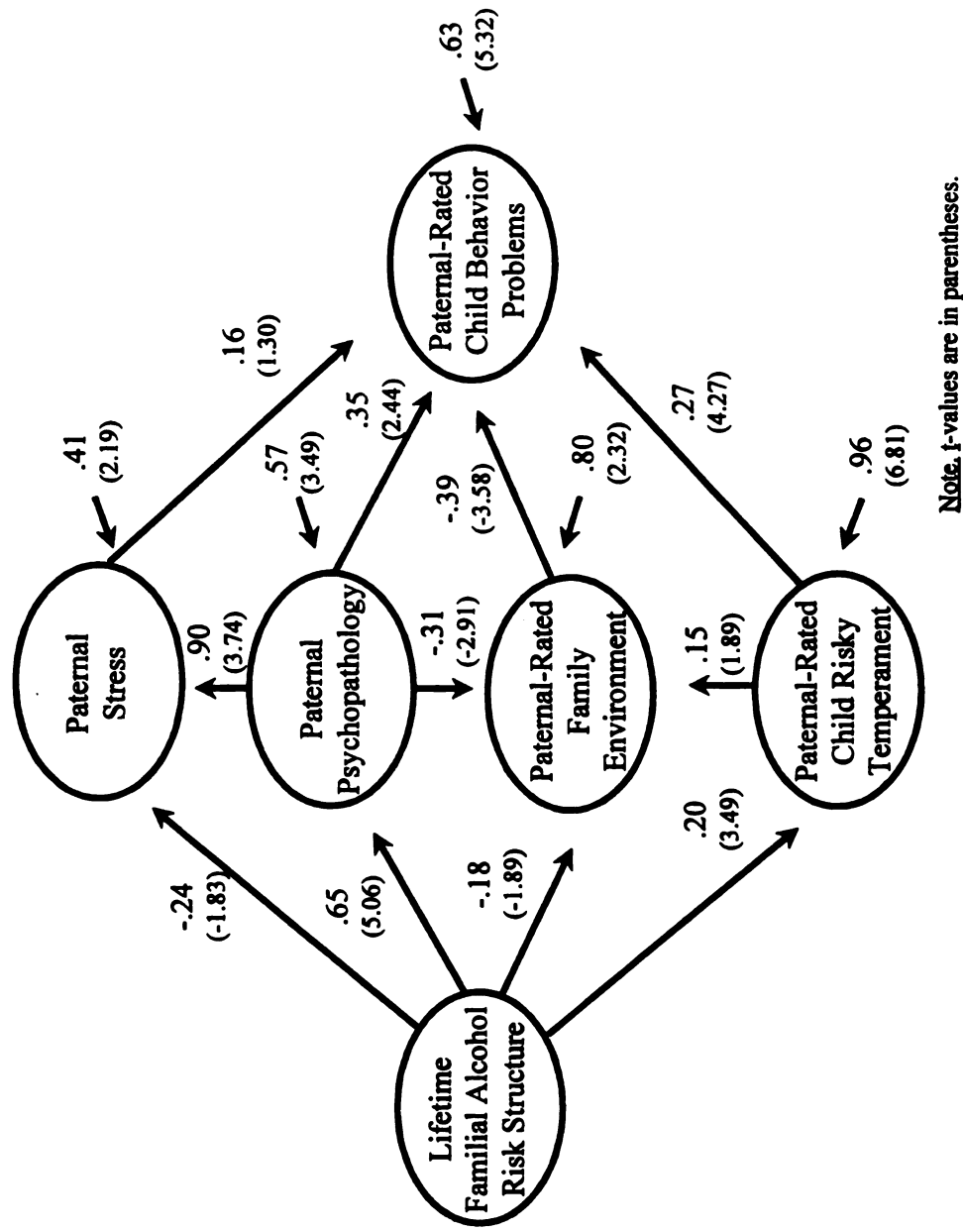


Figure 6. Structural model from Wave 2 two-group stacked analysis: Solution for maternal data for entire sample .

direct discriminant function analysis was conducted to test if coupled individuals differed from decoupled individuals on a number of parent-level predictors. Results revealed that almost all parent-level variables used in the current study discriminated between the coupled and decoupled individuals (see Appendix F for discriminant function analysis results).

Because coupled families are different from decoupled families, all SEMs were re-analyzed with the data from coupled families only (see Appendix G for results). The final models from these analyses partially supported the first hypothesis and revealed very few differences in the patterns of prediction between the analyses conducted on the entire sample and those analyses with the subsample of coupled individuals only. Rather, any differences that did result from including only coupled individuals in the analyses were most likely a result of the truncation of variance that occurred from removing the families with the densest risk structures. That is, compared to the models with the entire sample, the models with coupled individuals only revealed fewer associations among the latent constructs. Additionally, similar to the models with the entire sample, these models also revealed a positive association between child risky temperament and family environment further validating this unexpected finding. Overall, results from these data were analogous to those with the entire sample.

Predicting Change

The second hypothesis proposed that high levels of externalizing behavior problems would be maintained across time in families where parental occupational prestige remained low, parental alcoholism was maintained across time, increased

levels of parental psychopathology and stress were maintained, and where levels of child risky temperament and poor quality family environments were also maintained across time. This hypothesis was tested using two regression analyses (one for maternal data, one for paternal data) with difference or change scores entered into the models.

Change scores were first computed for all variables by subtracting the Wave 2 score on a given measure from the Wave 1 score of that same measure (see Table 6 for change score means and SDs). Next, these difference scores were entered into a 7-stage regression analysis along with the baseline (Wave 1) measure for each difference score. For each regression the Type III Sums of Squares was requested in order to test the unique effect of each independent variable on the outcome. The dependent variable for each of the regression analyses was change in child externalizing behavior problems.

The variables were entered into the seven stages in the following order; (1) the familial alcohol risk variables of Wave 1 occupational prestige, alcoholism status, current depression, maximum alcohol consumption and alcohol consumption along with change in each of these variables were entered simultaneously into the first stage of the model; (2) the child risky temperament variables of adaptability, activity, and reactivity along with their corresponding change scores were entered simultaneously into the second stage of the model; (3) the current parental stress measures of life events stress, daily hassles stress and the difference scores computed from these variables were entered simultaneously into the third stage; (4) the quality of family environment variables of cohesion, conflict, expressiveness

Table 6

Means and Standard Deviations of Variables Used in Change Analyses

Variables	Mother Mean (SD)	Father Mean (SD)
Change in Child Externalizing Behavior Problems	-1.28 (5.9)	-1.23 (5.0)
Change in Parental Alcohol-Related Risk		
Ch Occupational Prestige	-46.44 (173.9)	-14.22 (123.7)
Ch Alcohol Diagnosis	-0.05 (0.7)	-0.39 (1.2)
Ch Depression	0.63 (3.1)	0.50 (3.1)
Ch Alcohol Consumption	149.23 (1864.74)	-951.88 (4319.04)
Ch Max Alcohol Consump.	-0.14 (1.1)	-0.38 (1.7)
Change in Child Risky Temperament		
Ch Activity Level	-0.26 (1.7)	-0.37 (1.4)
Ch Adaptability	0.32 (1.9)	-0.01 (1.7)
Ch Reactivity	-0.61 (1.7)	-0.21 (1.4)
Change in Parental Stress		
Ch Daily Hassles	-4.61 (18.0)	-2.48 (21.9)
Ch Life Events	-0.02 (1.4)	-0.17 (1.4)
Change in Family Environment		
Ch Conflict	0.12 (1.8)	-0.12 (1.9)
Ch Expressiveness	0.19 (1.5)	0.17 (1.7)
Ch Cohesion	-0.01 (1.5)	-0.16 (1.6)

Note. Change scores calculated as Wave 2 variable minus Wave 1 variable.

as well as the difference scores computed from these variables were entered into the fourth stage; (5) the variables entered into the first and second stages were entered simultaneously into the fifth stage; (6) variables entered into the first, second, and third stages were entered simultaneously into the sixth stage; and (7) all variables were entered simultaneously into the seventh and final stage. The order of entry was chosen based on theoretical considerations; it was expected that the familial alcohol risk variables would impact child temperament which would impact parental stress levels and in turn, the quality of the family environment.

Paternal Change Models

Prior to conducting the regression analyses, all predictor variables (with the exception of the baseline alcohol consumption variable) were centered (i.e., the mean was subtracted from the individual items) in order to eliminate multicollinearity. Because the baseline alcohol consumption variable was extremely skewed (see Appendix B for graphical representation) a log base 10 transformation was conducted prior to running the regression analyses. Results from the paternal regression analysis revealed that the second, fourth and seventh models predicted significant portions of variance in child externalizing behavior problems (see Table 7). In the second stage of the analysis both baseline child activity level and change in child activity level predicted change in child externalizing behavior problems. In the fourth stage of the model baseline family cohesion and change in family conflict predicted child externalizing behavior problems. Finally, in the seventh stage of the model, when all variables were entered simultaneously, baseline family cohesion, change in family cohesion, change in family conflict, and change in child activity

Table 7

Paternal Predictors of Change in Child Externalizing Behavior Problems

Variables	1	2	3	4	5	6	7
Paternal Alcohol-Related Risk							
T1 Occupational Prestige	.02				.02	.03	-.01
T1 Alcohol Diagnosis	-.16				-.11	-.12	-.08
T1 Depression	.05				.01	.11	.14
T1 Alcohol Consumption	-.11				-.12	-.09	-.09
T1 Max Alcohol Consump.	.11				.07	.09	.08
Ch Occupational Prestige	-.05				-.05	-.04	-.10
Ch Alcohol Diagnosis	-.22*				-.17	-.17	-.18
Ch Depression	-.07				-.09	-.04	.12
Ch Alcohol Consumption	-.01				-.04	-.02	-.08
Ch Max Alcohol Consump.	.08				.05	.06	.04
Child Risky Temperament							
T1 Activity Level		.21*			.20*	.18*	.15
T1 Adaptability		-.08			-.09	-.08	-.11
T1 Reactivity		.07			.07	.07	.08
Ch Activity Level		.20*			.19*	.19*	.23**
Ch Adaptability		-.01			-.01	-.00	.01
Ch Reactivity		-.07			-.07	-.06	-.03
Paternal Stress							
T1 Daily Hassles			-.17*			-.19	-.12
T1 Life Events			.08			.05	.07
Ch Daily Hassles			-.11			-.07	-.09
Ch Life Events			.08			.06	.05
Family Environment							
T1 Conflict				.05			.14
T1 Expressiveness				-.16			-.13
T1 Cohesion				.18*			.23*
Ch Conflict				.25***			.26**
Ch Expressiveness				-.11			-.12
Ch Cohesion				.15			.18*
R²	.05	.06*	.03	.09**	.05	.12	.21**
df	(10,197)	(6,201)	(4,203)	(6,201)	(16,191)	(20,187)	(26,181)

level predicted change in child externalizing behavior problems (see Appendix H for graphical representation of the significant relationships). These results only partially supported hypothesis 2 and indicated that the maintenance of behavior problems across time was predicted by more conflict, less cohesion and higher child activity levels across time.

Maternal Change Models

Just as for the paternal models, all predictor variables (with the exception of the alcohol consumption variable) were centered prior to conducting the maternal regression analyses, and a log base 10 transformation was conducted on the alcohol consumption variable. Furthermore, the baseline maximum alcohol consumption variable was removed from the analysis of the final model (model 7) because even after it was centered there were problems with multicollinearity. Therefore, the final model was analyzed without the predictor variable of maximum alcohol consumption (see Table 8). Results revealed that none of the models predicted a significant portion of the variance in child externalizing behavior problems. Thus, these results failed to support hypothesis 2.

Table 8

Maternal Predictors of Change in Child Externalizing Behavior Problems

Variables	1	2	3	4	5	6	7
Paternal Alcohol-Related Risk							
T1 Occupational Prestige	.09				.08	.09	.08
T1 Alcohol Diagnosis	.02				.02	.03	-.04
T1 Depression	.04				-.00	-.04	.00
T1 Alcohol Consumption	.07				.05	.10	-.02
T1 Max Alcohol Consump.	-.20				-.18	-.24	
Ch Occupational Prestige	-.05				-.05	-.06	-.07
Ch Alcohol Diagnosis	-.10				-.10	-.07	-.11
Ch Depression	-.02				-.04	-.03	-.02
Ch Alcohol Consumption	.02				.02	.02	.05
Ch Max Alcohol Consump.	-.04				-.05	-.07	.03
Child Risky Temperament							
T1 Activity Level		.12			.10	.13	.10
T1 Adaptability		-.01			-.01	-.01	-.04
T1 Reactivity		.08			.11	.09	.08
Ch Activity Level		.04			.03	.06	.07
Ch Adaptability		-.06			-.05	-.06	-.10
Ch Reactivity		-.08			-.04	-.07	-.09
Paternal Stress							
T1 Daily Hassles			.03			.04	.09
T1 Life Events			-.02			.02	.02
Ch Daily Hassles			-.07			-.05	.01
Ch Life Events			-.14			-.17*	-.14
Family Environment							
T1 Conflict				.11			.10
T1 Expressiveness				-.01			.00
T1 Cohesion				.07			.11
Ch Conflict				.16*			.14
Ch Expressiveness				.08			.07
Ch Cohesion				.10			.14
<u>R²</u>	.07	.03	.03	.06	.09	.12	.14
<u>df</u>	(10,197)	(6,201)	(4,203)	(6,201)	(16,191)	(20,187)	(26,181)

Note. Model 7 calculated without T1 Maximum Alcohol Consumption.

DISCUSSION

Alcoholism is a developmental phenomenon. Although there are different pathways leading to this disorder (Zucker, 1987; 1994), one possible pathway is that which begins early in infancy and childhood with risky temperament and externalizing behavior problems and culminates in adolescence and adulthood with antisociality and alcohol problems (Tarter & Vanyukov, 1994). This process that begins early in developmental time will advance, continue, and evolve only if a dense set of individual risk factors is continually paired with a high risk environment (Zucker et al., 1995). Thus, for instance, an individual risk factor is child risky temperament and high risk environments can be those characterized by one or a more of the following; low family SES, parental alcohol problems, parental antisociality, parental psychopathology, parental stress, and a poor quality family environment.

Numerous researchers have examined the influence of individual and environmental risk factors on COA outcomes (see West & Prinz, 1987 for review), however, fewer have examined the simultaneous contribution of these factors on behavior problem development in COAs. In addition, there has been a paucity of research on the longitudinal relationships among any of these risk variables and COA outcomes. Little prospective research has been conducted regarding change and stability across developmental time in these variables.

The present study, guided by risk aggregation theory, examined the joint contributions of environmental and individual child factors on child externalizing behavior problems in a sample of 3-9 year old male COAs. Furthermore, this study

examined the impact of change in individual and environmental-level risk factors on change in COA externalizing behavior problems.

Predicting Child Externalizing Behavior Problems

The results of the present study demonstrated that risk aggregation theory is an appropriate framework for the study of externalizing behavior problems in COAs. In particular, for both Wave 1 and Wave 2 data and for both the entire sample and coupled parents only, child externalizing behavior problems is an outcome that is predicted by the interplay of sociocultural variables (i.e., SES), lifetime parental-level variables (i.e., lifetime alcohol problems and antisociality), current parental variables (i.e., stress and psychopathology), family climate variables (i.e., quality of the family environment) as well as child individual variables (i.e., child risky temperament).

Cross-Sectional Stacked Analyses and the Entire Sample

Results of the cross-sectional stacked analyses revealed that the pattern of predictions for maternal and paternal data did not differ. Specifically, results of the Wave 1 cross-sectional analyses partially supported the first hypothesis and indicated that for all parents in the present sample, lifetime familial alcohol risk structure predicted parental stress, psychopathology, quality of the family environment, and child risky temperament. In addition, parental psychopathology predicted a poorer quality family environment and increased levels of parental stress. Child risky temperament predicted a better quality (not worse) family environment and increased levels of child externalizing behavior problems were predicted directly only by a poorer quality family environment and child risky temperament but not by parental

stress or psychopathology as was hypothesized.

These results corroborate previous research findings indicating that parents with a dense lifetime familial alcohol risk structure also experience more stress (Cooper, Russell, Skinner, Frone, & Mudar, 1992; Johnson & Pandina, 1993; Loukas, 1995), more psychopathology (Regier et al., 1990; Fitzgerald et al., 1995; Zucker et al., 1994), more family conflict, less cohesion, and less expressiveness (Moos & Moos, 1984; Wilson et al., 1995), and have children with risky temperaments (Chassin et al., 1993; Ellis et al., 1996). In addition, the findings also support research indicating that parental psychopathology is associated with higher levels of parental stress (Hammen, Davila, Brown, Ellicott, & Gitlin, 1992) as well as family environments characterized by more conflict, less cohesion, and less expressiveness (Billings & Moos, 1983). Additionally, results parallel the findings of Tarter and his colleagues (Tarter et al., 1995; Tarter & Vanyukov, 1994) and can be interpreted within risk aggregation theory. A poor quality family environment in combination with child risky temperament may lead directly to increased levels of child externalizing behavior problems. Thus, children who live in homes that are characterized by conflict, low levels of cohesion and expressiveness and who have risky temperaments are at heightened risk for behavior problems and if this combination is maintained across developmental time may also be on a trajectory to later problems.

The first hypothesis was not entirely supported however. Unexpectedly, results revealed that child risky temperament was associated with a better quality family environment. This finding is contrary to what was hypothesized and also is

contrary to arguments that children with risky temperaments are perceived by their parents as difficult thereby evoking interactions that are coercive and conflictual (Lerner & Lerner, 1986).

One possible explanation for the discrepancy between the findings in the current study and those of previous studies is the difference in how temperament is conceptualized. In the present study, child temperament was conceptualized by high activity, high reactivity, and high adaptability. These three dimensions were chosen because they were believed to be theoretically most predictive of later externalizing behaviors for COAs (Tarter, 1988) and thus, were collectively referred to as risky temperament. In fact, results showed that children of parents with a dense lifetime familial alcohol risk structure had risky temperaments, which in turn predicted more child externalizing behavior problems. However, it is plausible that this combination of temperament dimensions does not adversely affect the family environment. That is, children high on activity, reactivity, and adaptability may not be seen as difficult by their parents and thus, the family environment is not more conflictual, less cohesive, and less expressive.

In the present study, the temperament dimension of adaptability was deliberately scored in the opposite direction (i.e., to reflect more adaptability) of how it is traditionally scored. For instance, Thomas and Chess (1977) consider a child low in adaptability (i.e., slow to respond to new situations) to have a difficult temperament, whereas a child high in adaptability is viewed as easy. The difficult child provokes negative reactions from the family environment but the easy child does not. Thus, it follows that children who are temperamentally high in

adaptability live in better quality family environments than children who are temperamentally low in adaptability. Similarly, previous research on COAs has indicated a negative relationship between child sociability (congruent to adaptability) and family conflict (Wilson et al., 1995) suggesting that family conflict is lower in families of children who are highly adaptable.

An alternative explanation for the current finding stems from a "goodness-of-fit" framework (e.g., Lerner & Lerner, 1986). In the present sample, children with risky temperaments were also likely to have parents with risky temperaments; thus, the temperamental styles of children and parents may have been a good fit. In families where parent and child temperament fit is good, children are not viewed as problematic and therefore, the family environment is not adversely affected by the child's temperament (Thomas & Chess, 1977; Lerner, 1983). When the fit is poor, however, children are viewed as problematic or difficult by parents and it is within this situation that the family environment is expected to be conflictual, less cohesive, and less expressive. Thus, the Thomas and Chess (1977) model is a match-mismatch view of fittingness and it would be a mismatch that would be predictive of poor outcome, regardless of the qualitative nature of temperament itself.

Further research is warranted to clearly delineate the relationship between child temperament and family environment. One factor that hampers the delineation of this relationship is the use of different terms as well as different dimensions or aspects of temperament. For instance, some researchers examine difficult temperament (e.g., Jansen et al., 1995), whereas others examine deviations in temperament (e.g., Tarter & Vanuykov, 1994), and still others examine only one

aspect of temperament such as emotionality (e.g., Chassin et al., 1993). These differing methodologies make the comparison of results across studies difficult and the compilation of findings complex. Future studies should be conducted to examine how these differing conceptualizations of individual temperament influence research findings.

Conversely, the relationship between child risky temperament and a better quality family environment may be due to methodological artifacts. The family environment indicator variables of family cohesion, conflict, and expressiveness had very small SDs relative to their means indicating very little variance in these measures across families. It is possible, therefore, that family cohesion, conflict, and expressiveness are not sensitive measures of the different family environments when also considering child temperament. Perhaps other dimensions or measures of family environment are more sensitive to the effects of child risky temperament and thus, additional studies must be conducted in order to conclusively delineate this relationship.

Also contrary to what was hypothesized and to what previous researchers and the present data have reported was the lack of relationship between parental psychopathology and child externalizing behavior problems. Numerous previous studies have indicated that children of depressed parents exhibit increased levels of behavior problems (e.g., Downey & Coyne, 1990; Dyer Harnish et al., 1995). One possible explanation for this contrary finding is that parental psychopathology such as depression is a better predictor of child internalizing problems and not externalizing problems. For instance, Johnson & Jacob (1995) found that in a

sample of male COAs heightened levels of current maternal depression predicted both internalizing and total behavior problems but not externalizing problems.

An alternative explanation for this contrary finding is that parental psychopathology does not have direct and independent effects on child outcome in young COAs but rather asserts its influence on child externalizing behavior problems through other more proximal variables. For instance, in the present study parental psychopathology was associated with child outcome only indirectly through its association with the quality of the family environment. Although contrary to what was expected, this finding is consistent with social interaction theory (Patterson, 1982; 1992; Patterson et al., 1989) and suggests that the major impact of parental psychopathology on child externalizing behavior problems is through the quality of the family environment. Thus, parental psychopathology may act to increase family conflict and to decrease family cohesion and expressiveness and in turn increase behavioral problems in children. However, just as in the general literature on the effects of depression on child outcomes, it is not clear whether parental depression or other family environment variables are more important in predicting child behavior problems (Rutter, 1990); thus, further research is necessary to examine these associations.

The absence of the direct path from parental stress to child outcome was also contrary to what was hypothesized. In previous studies with samples of both COAs and nonCOAs, parental stress levels have been found to predict heightened levels of child behavior problems (e.g., Beautrais et al., 1982; Chassin et al., 1991; Loukas 1995). The present finding may indicate that for children between the ages of 3

and 5 years parental stress is not as important a predictor of child outcome as is the family environment and child temperament. Although parental stress levels are elevated in high risk homes other more immediate stressors may directly impact child outcomes. In other words, in homes characterized by a variety of other problems such as increased conflict, less cohesion and expressiveness, and child risky temperament the presence of parental daily and life events stresses are not perceived as serious or taxing (cf. Lazarus & Folkman, 1984). Yet, to fully evaluate the effects of stress on child outcomes researchers must examine parental social support and coping mechanisms. Even in homes characterized by numerous risk factors the availability of social supports and coping mechanisms moderate the effects of stress on child outcomes (e.g., Muller et al., 1994).

Conversely, this finding may suggest that parental stress is only indirectly related to child outcomes through other more proximal variables such as parental discipline and monitoring. Parents who experience heightened levels of stress may not be able to adequately monitor or discipline their children because they are overburdened or taxed with other problems. Thus, these parents may fail to reinforce appropriate behaviors or fail to punish the children for inappropriate behaviors (Patterson 1982; 1992). These interactions are continually repeated in the home environment and result in the child learning from his parents to exhibit inappropriate behaviors. Although parental monitoring and discipline skills were not included in the present study, future studies should examine more closely the relationship between parental stress, monitoring, discipline skills, and child externalizing behavior problems.

Results of the Wave 2 stacked cross-sectional analyses were virtually identical to those found for the Wave 1 analyses with the exception of two relationships. At Wave 2 a less dense lifetime familial alcohol risk structure predicted more parental stress, a finding that was contrary to what was hypothesized and to what previous researchers have reported. This inverse relationship is likely due to methodological problems associated with SEM analyses and must be further examined in future studies for conclusive statements to be made regarding this relationship.

The second and final difference between the Wave 1 and Wave 2 analyses was that at Wave 2, parental psychopathology was both an indirect (as for Wave 1) as well as a direct predictor of child outcome. This relationship was hypothesized and supports the literature suggesting that parents with psychopathology have children with adjustment problems (Downey & Coyne, 1990; Fendrich et al., 1990). Differences between Wave 1 and Wave 2 results may partly be due to the fact that as children living in high risk environments grow older they become more sensitive to parental characteristics and thus, are more likely to be affected by parental psychopathology.

Cross-Sectional Analyses and Coupled Parents Only

A second set of analyses, identical to the first set, was run on families that remained intact across both Wave 1 and Wave 2 data collection. These analyses were conducted in order to examine the pattern of associations among the variables for families that remained coupled across the two Waves of data collection and to verify that including decoupled families in the Wave 2 analyses did not bias the

results.

The results of the cross-sectional analyses on the coupled families revealed that there were few differences between the analyses conducted on the entire sample and the set of analyses conducted on the coupled individuals only. Overall, the results of the second set of analyses also partially supported the first hypothesis and indicated that risk aggregation theory is an appropriate framework for the study of problem behavior development.

Specifically, the results of the Wave 1 cross-sectional analyses with coupled families were analogous to those reported for the entire sample. However, in the present set of analyses parental stress did predict child outcomes as was hypothesized and therefore, corroborates previous research. This result may be due to the exclusion of some of the highest risk parents from the present sample. That is, previous results from the MSU-UM Longitudinal Study have demonstrated that families with alcoholic and antisocial fathers are the most likely families to be decoupled followed by families with alcoholic and nonantisocial fathers and that nonalcoholic control families are the least likely to be decoupled (Loukas, 1995). Families not included in the present analyses were likely those with the most dense lifetime familial alcohol structures, the highest levels of psychopathology and stress, the poorer family environments and the children with the most risky temperaments (see Zucker et al., 1994; Ellis et al., 1996 for discussion of risk group differences). It is plausible, therefore, that once the lower functioning parents are removed from the sample, the role of parental stress in the development of child outcomes becomes increasingly apparent. Child temperament and the quality of the family environment

no longer play the only role since some of the children with the most risky temperaments living in the lower quality family environments are removed from the analyses.

The results of the Wave 2 analyses were also analogous to the Wave 2 analyses with the entire sample with two exceptions: For coupled parents only, the results revealed that lifetime familial alcohol risk structure did not predict parental stress or quality of the family environment. These results may be due to methodological artifacts. Once the more psychopathological families were removed from the sample (i.e., decoupled) there was further truncation of variance in the family environment variables and a truncation of variance in the parental stress and lifetime familial alcohol risk structure variables. The truncation of variance may have caused a weakening of the relationships among these variables and a lack of power to detect a real association between the variables.

Change and Child Externalizing Behavior Problems

The second hypothesis stating that high levels of externalizing behavior problems would be maintained across time in families where parental occupational prestige remained low, parental alcoholism was maintained across time, increased levels of parental psychopathology and stress were maintained, and where levels of child risky temperament and poor quality family environments were also maintained across time was only partially supported.

The results for the paternal data partially supported the second hypothesis and indicated that baseline family cohesion, change in family cohesion and change in family conflict predicted change in child externalizing behavior problems. These

results further support the importance of the quality of the family environment on child outcomes. Additionally, results indicated that the child individual variable of change in activity level was an important predictor of child externalizing behavior problems. This result is consistent with research indicating that child temperament is associated with increased levels of child behavior problems (Caspi et al., 1995; Jansen et al., 1995).

The results from the analyses with maternal data failed to support the second hypothesis; none of the models predicted a significant amount of the variance in change in child externalizing behavior problems. These results were unexpected but may be due to the use of change or difference scores to measure continuity or discontinuity. Previous researchers have questioned the reliability and validity of change scores (Burr & Nesselroade, 1990) and, thus, change scores may not be the most appropriate method for measuring change. However, in order to confirm the findings of the current study further research must be conducted on independent samples.

The results from the paternal data have important implications for the later development of COAs. In particular, the findings from the paternal data indicated that children who exhibited more externalizing behavior problems remained high in activity level across time and were raised in family environments characterized by more conflict and less cohesion across time. These findings are consistent with risk aggregation theory and demonstrate that problematic outcomes result from the combination of individual and environmental risk. That is, children with the highest activity levels raised in homes that are conflictual and non-cohesive are placed on a

trajectory to later problems including externalizing behaviors, antisociality, and alcohol problems.

Summary and Implications

Risk aggregation theory is an appropriate framework for the study of the etiology of COA externalizing behavior problems. Within risk aggregation theory, development is viewed as multi-causal, but more specifically behavioral maladjustment is the result of the continuous pairing of high risk environments with individual risk factors.

Thus, implications of the findings from the present study are that parents with alcohol problems, high levels of antisociality and a low SES provide a high risk environment for the development of their children. Given that COAs are more likely than children of nonalcoholics to have risky temperaments, it is likely that the reciprocal interactions among child temperament and the environmental variables will shape a trajectory leading to more behavior problems, antisociality and possibly later alcoholism (Tarter & Vanyukov, 1994). Thus, a child with risky temperament living in an environment dense with risk (i.e., low in SES, high parental antisociality, alcohol problems, psychopathology, stress, and a poor quality family environment) not only lacks self-regulation but also the social regulation (i.e., regulation from the family) that is necessary for adaptive development.

Families dense with risk often lack appropriate family management skills and as such put their child at risk for later problematic outcomes. That is, such parents use inappropriate monitoring and discipline techniques with their children and frequently use coercive behaviors to gain control over chaotic environments (e.g.,

Patterson, 1982; 1992; Patterson et al., 1989). Children repeatedly exposed to such situations learn directly from their parents to use coercive and antisocial behaviors in other situations as well. The longer the child is involved in this environment, the more likely it is for the externalizing behaviors to filter into other environments such as the school (Dishion, Patterson, Stoolmiller, & Skinner, 1991).

Once the child enters the school environment the antisocial behavior may interfere with classroom tasks and with the socialization into a "normal" peer group (Patterson et al., 1989). Children that are rejected by "normal" peers associate with deviant peers who not only reinforce inappropriate behaviors but who may also introduce the child to other antisocial activities (Dishion et al., 1991; Patterson et al., 1989). The child selects an environment that reinforces the inappropriate behavior and further strengthens the trajectory into problematic behavior (Scarr & McCartney, 1989). The early and sustained presence of antisociality may then lead the child on a pathway to more antisocial behavior and alcohol problems in later life (Zucker et al., 1994; 1995). Thus, the importance of the family environment early in life and in particular parental management skills clearly have implications for the future development of children.

Limitations and Future Directions

Although the present study improves upon previous studies on COAs in many ways; by using a community sample, by collecting prospective information from both mothers and fathers, and by following the sample across time, there are still a number of limitations that must be addressed. First, two waves of data were available for the present analyses. However, due to the complexity of the design,

the use of multiple-level factors, and the limited sample size these data could not be examined in one analysis. Including longitudinal autocorrelations in future analyses would allow researchers to infer causation and to observe change and stability across time. Additionally, inclusion of more than two waves of data in analytic designs would allow the examination of individual-level change and stability and would allow researchers to map the multiple trajectories leading to either more problems or to a decrease in problems.

A further limitation of the present study is the reliance on parental self-reports and parental reports on children. This study improves upon past research on child outcomes by providing both maternal and paternal ratings of child behavior problems (Phares & Compas, 1992). However, children interact in many contexts in the absence of their parents outside the home. Thus, parents can only provide a limited assessment of their child's behavioral repertoire. Future studies must include reports from other individuals in the child's environment such as teachers and even the children themselves (Phares et al., 1989).

The present study, unlike most others, examined the simultaneous contribution of multiple-level factors on child outcome, however, it was not able to clearly differentiate the genetic from the environmental influences. Although most researchers view temperament as having a biological/constitutional basis and therefore, make conclusions of genetic influence based on the inclusion of this variable, the fact is that biological or genetic affects can only be accounted for if directly included in research models. The joint examination of biological and psychosocial or environmental variables on COA outcomes will allow a clearer

understanding of the processes involved in the etiology of problematic outcomes.

Finally, although the participants for the present study were recruited from the community and not from a treatment setting, all were Euro-American. The exclusion of ethnic diversity limits the generalizability of these findings to other ethnic groups. Thus, future studies should include ethnically diverse populations so that findings can be compared and contrasted across groups and so that broader generalizations can be made.

Future studies must also include examination of protective factors in addition to risk factors in the development of child externalizing behavior problems. Although COAs are at heightened risk for a variety of later problems the majority of COAs are functioning at levels comparable to nonCOAs (Clair & Genest, 1987). Identification of protective or moderating factors on COA outcome will not only allow a broader perspective on COA development, but may also provide information for strategies regarding prevention and intervention.

APPENDICES

APPENDIX A

Data Estimation

Longitudinal Data Estimation

A longitudinal data estimation procedure (Petersen, 1987) was used to estimate 252 data points (i.e., scale scores) out of a possible 8112 data points (3.1% of the total) for fathers and 210 data points out of a possible 8112 (2.6% of the total) for mothers. This estimation procedure was used only in cases where subjects were missing data at one wave, but not at the other.

This estimation procedure utilized two components; the nomothetic component and the ideographic component. The nomothetic component consisted of the scale score means of each group (i.e., AALs, NAALs, or Controls) at each wave of data collection. The ideographic component is generally the average distance, in units of standard deviation, between the subjects' data points at the waves where data are not missing and the nomothetic component at the wave with missing data (Bingham & Crockett, 1996). However, in the present study, only two waves of data were collected. Thus, the ideographic component consisted of the distance, in standard deviation units, between the subject's scale score at one wave (i.e., the wave with non-missing data points) and the nomothetic component at that wave.

First, the nomothetic component of each variable (calculated using non-missing data) was computed for each wave and each of the six subgroups. Next, an SPSS program was written to compute the missing data. A deviation score was computed for each case at each of the two waves by subtracting each individual's

score from the subgroup mean for each of the two Waves. For example, the deviations from the mean for Variable X were calculated as follows:

$$DX1 = MX1 - X1.$$

$$DX2 = MX2 - X2.$$

DX1 represents the deviation score for the Wave 1 variable, whereas MX1 represents the subgroup mean for variable X at Wave 1, and X1 represents the Wave 1 score for variable X. Since one of the data points was missing, only one of these deviation scores was computed (i.e., either DX1 or DX2).

Therefore, only the one deviation score was used to compute the deviation (across Wave 1 and Wave 2) from the mean of variable X.

For example:

$$DX = DX1.$$

or

$$DX = DX2.$$

This deviation score was then used to compute the estimated data as follows:

$$ED1 = MX1 - DX$$

The missing data point was estimated by subtracting the subgroup mean score of X from the deviation score. If the scale score was not missing, the original score was retained for that instrument (Bingham, 1993).

Cross-sectional Data Estimation

Following the longitudinal data estimation, the entire sample of data (estimated and original) was examined for scale scores that remained missing. Forty-two data points were still missing. These data points were missing because

both Wave 1 and Wave 2 data of the same variable were missing for the same case; therefore, could not be imputed longitudinally. These data points were estimated separately for mothers and fathers in each of the six risk groups using ordinary least squares regression with forced (simultaneous) entry.

Table A1

Chi-Square and Goodness of Fit Values for Wave 1 and Wave 2 Bias Analyses

Model	Chi-Square (df)	Goodness of Fit
Wave 1 Models		
Parental Psychopathology	15.42* (6)	0.99
Parental Stress	0.037 (6)	1.00
Child Temperament	0.037 (15)	1.00
Family Environment	0.010 (6)	1.00
Child Externalizing Behavior	0.040 (3)	1.00
Wave 2 Models		
Parental Psychopathology	0.35 (6)	1.00
Parental Stress	2.22 (6)	1.00
Child Temperament	0.260 (6)	1.00
Family Environment	0.230 (6)	1.00
Child Externalizing Behavior	0.024 (3)	1.00

* $p < .05$.

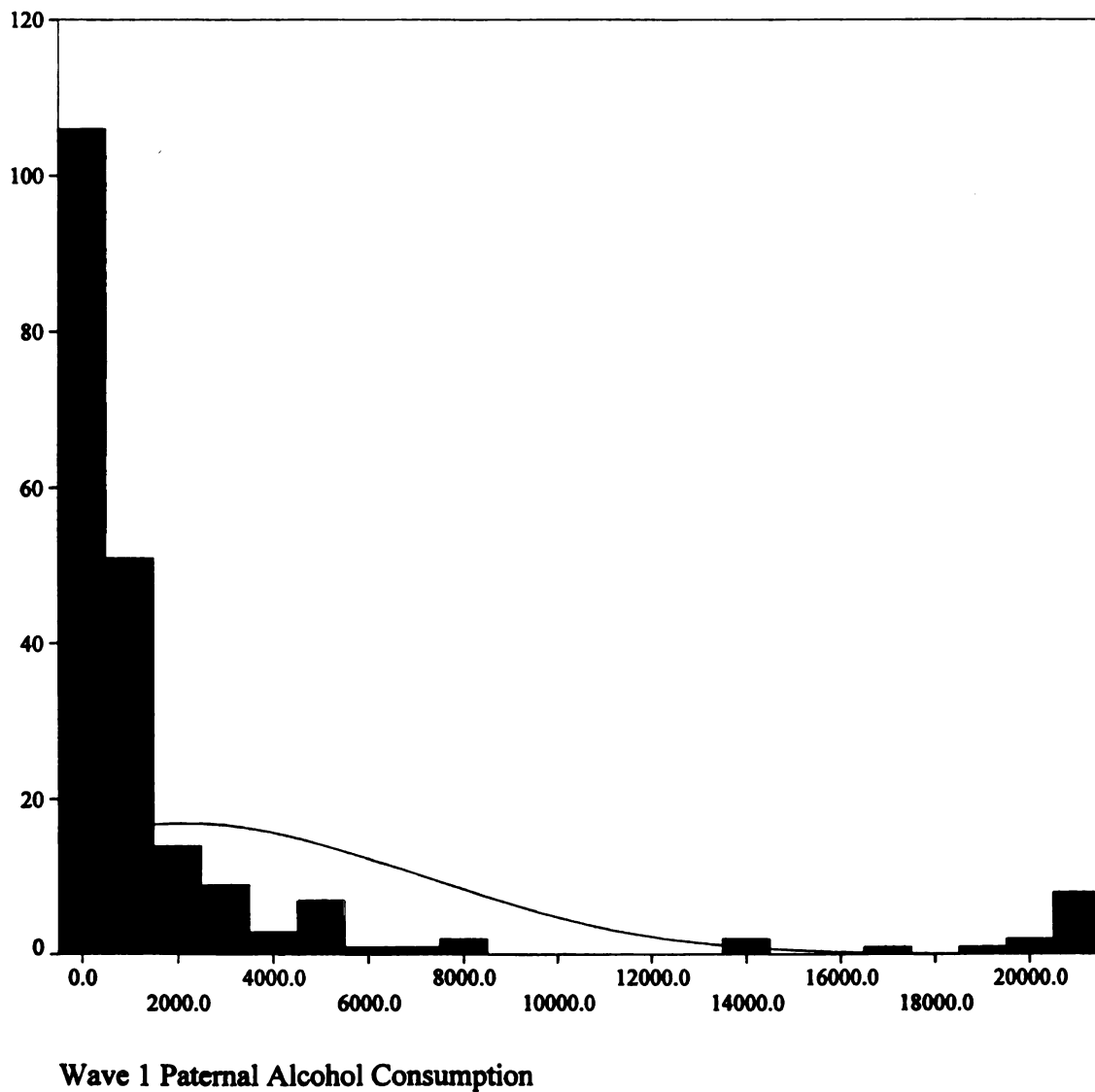
APPENDIX B**Graphical Representation of Alcohol Consumption**

Figure B1. Histogram of Wave 1 paternal alcohol consumption data.

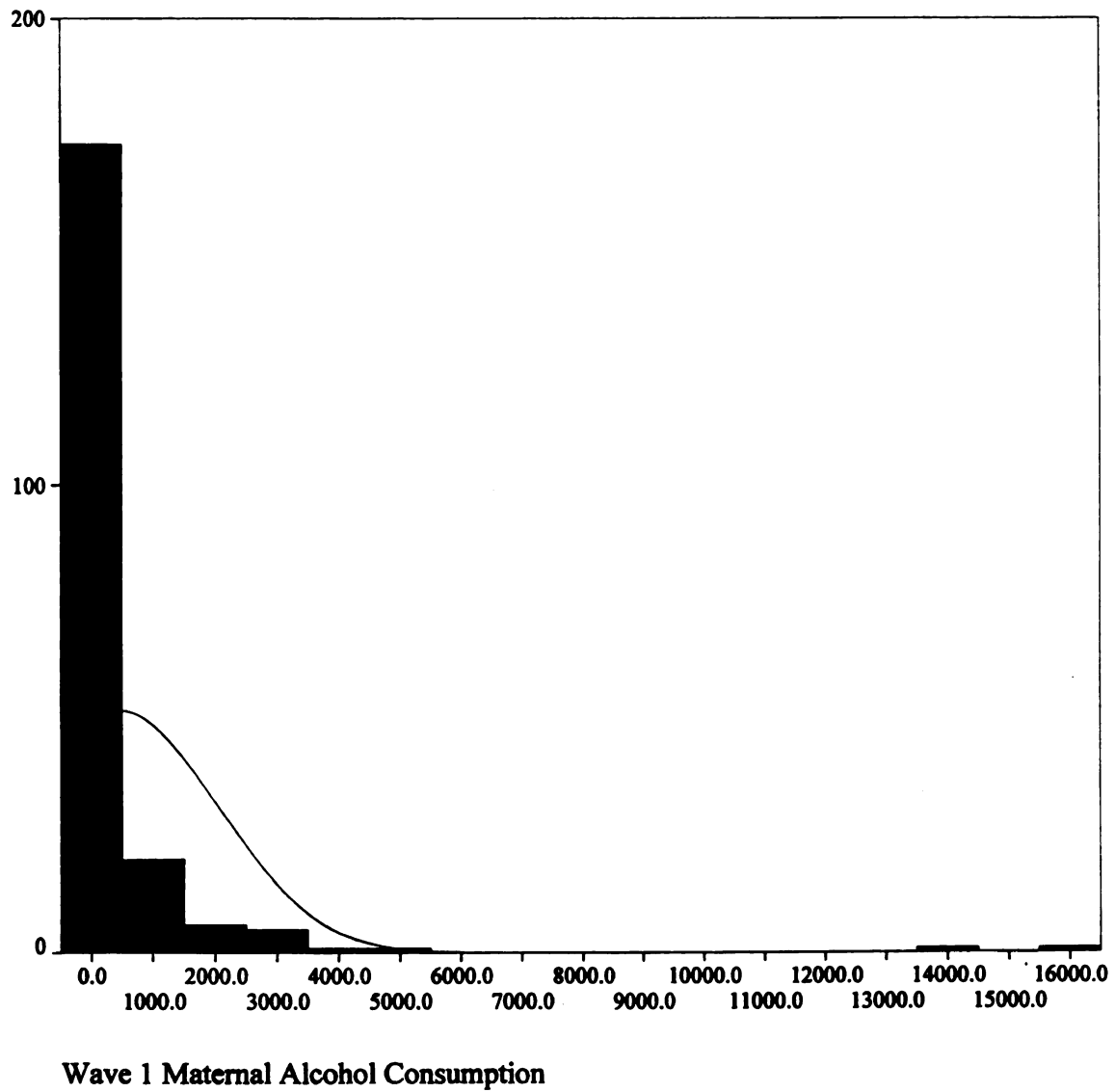


Figure B2. Histogram of Wave 1 maternal alcohol consumption data.

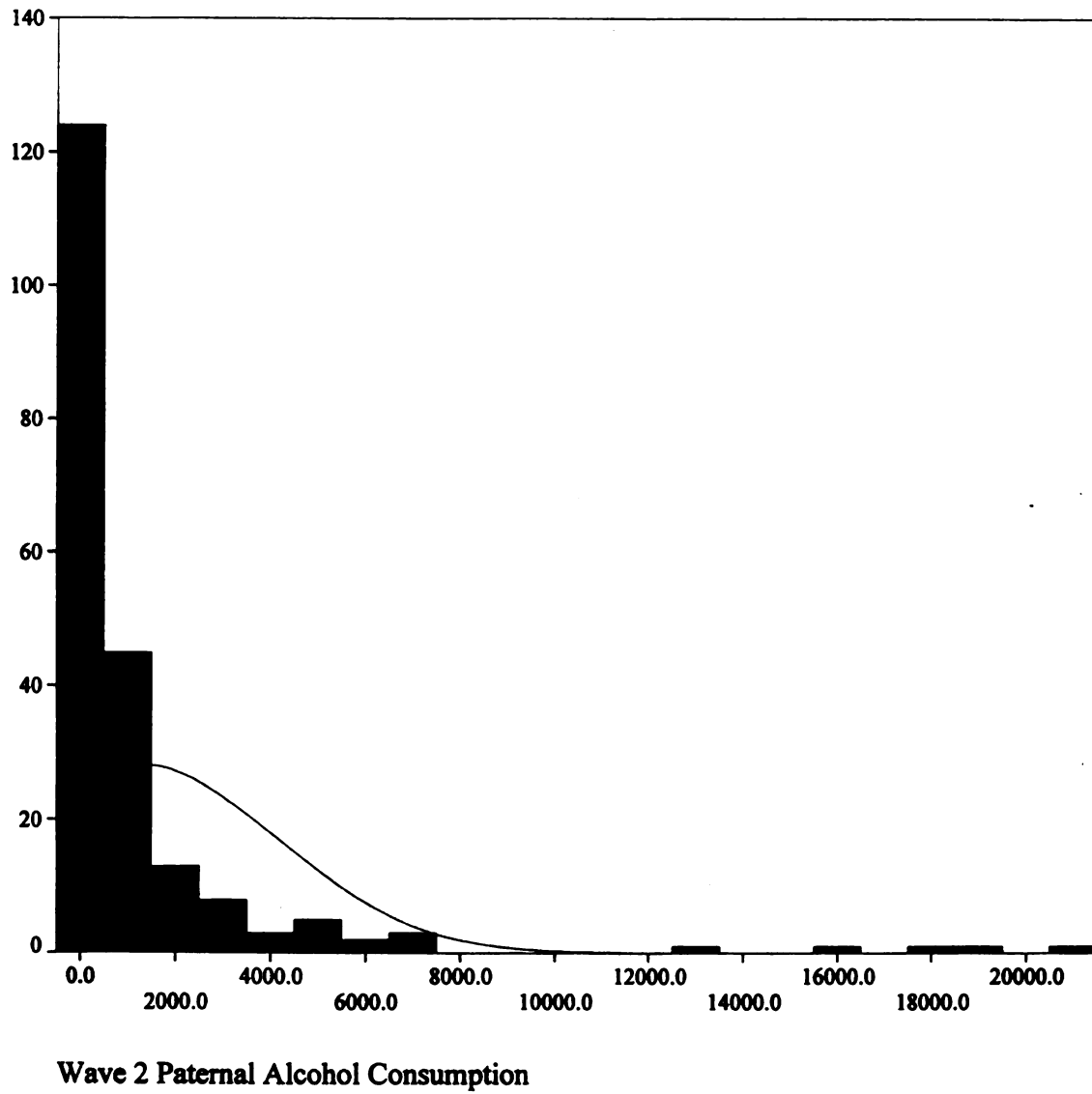


Figure B3. Histogram of Wave 2 paternal alcohol consumption data.

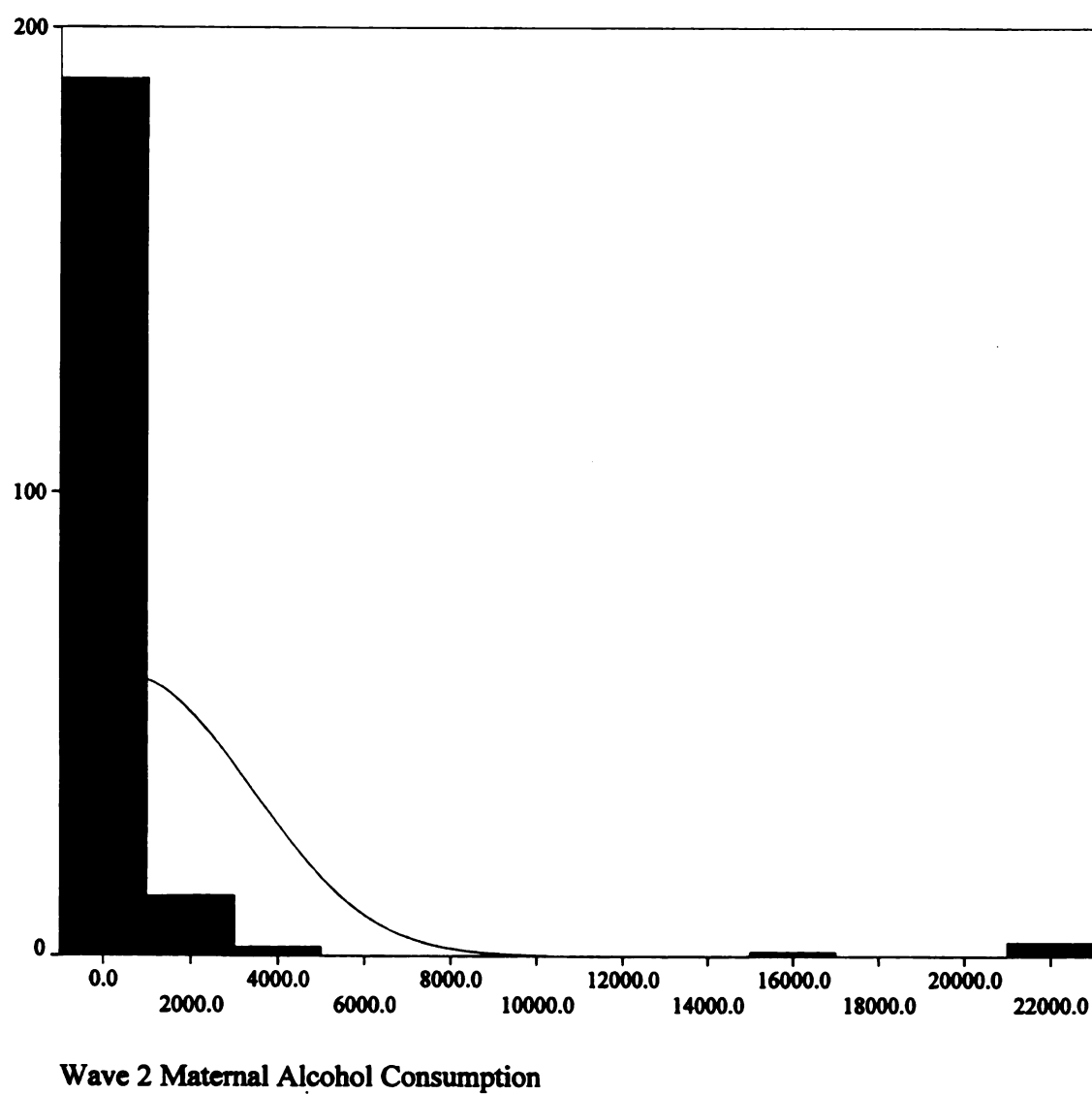


Figure B4. Histogram of Wave 2 maternal alcohol consumption data.

APPENDIX C

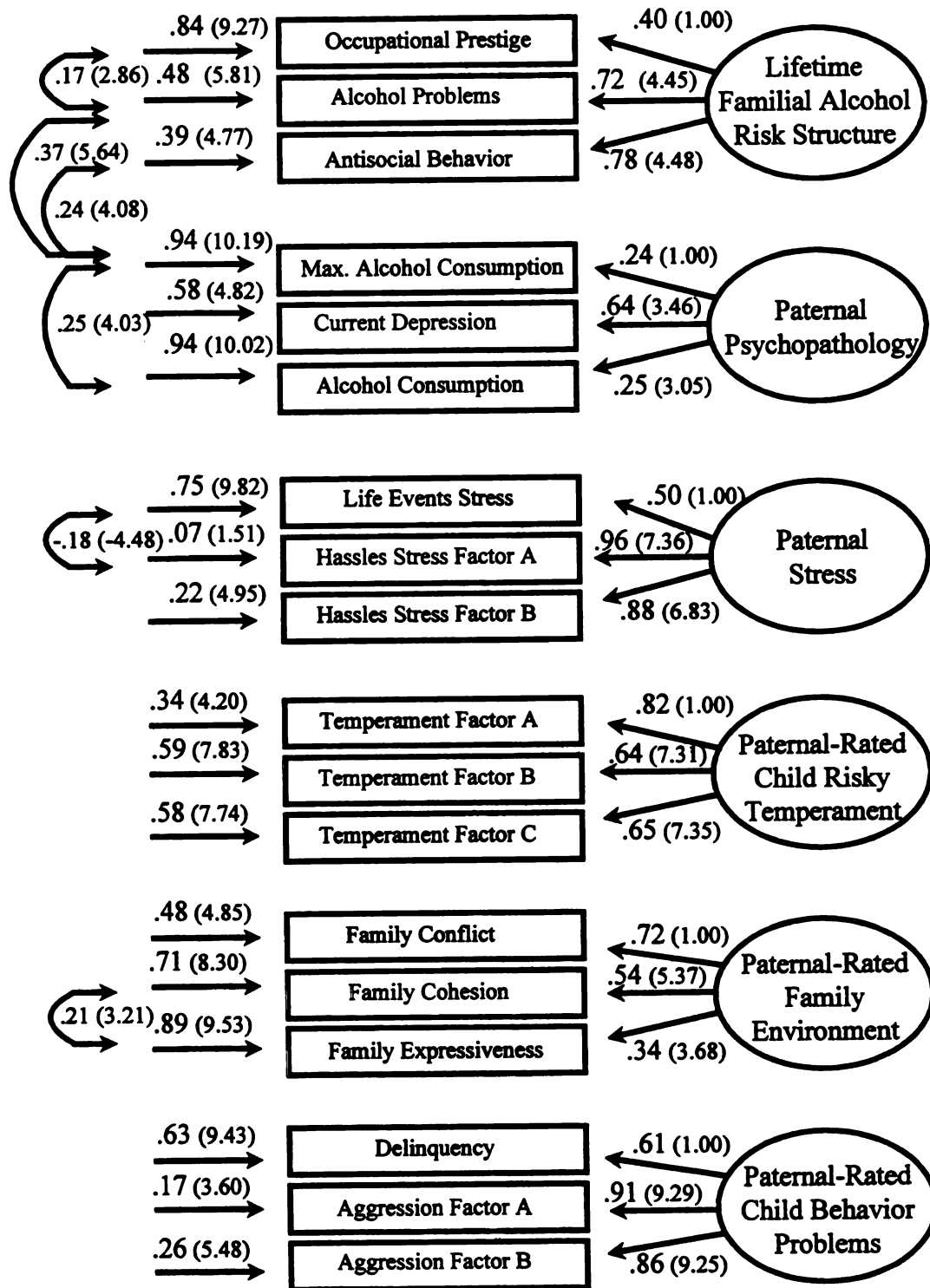
Cross-Sectional Measurement and Structural Models - Entire Sample

Measurement Models - Entire Sample

Each of the measurement models fit the data well. Although the Wave 1 paternal measurement model resulted in a significant Chi-Square [$X^2(114, N = 208) = 173.54, p < .001$], the GFI (.92) and CFI (.95) were large and the SRMR was small (.066) (see Figure C1 for measurement model and Table C1 for correlations among latent constructs). The Wave 1 maternal measurement model also resulted in a significant Chi-Square [$X^2(116, N = 208) = 164.01, p < .001$], a GFI of .92, a CFI of .96, and a SRMR of .069 (Figure C2 for measurement model and Table C2 for correlations among latent constructs). The Wave 2 paternal measurement model had a significant Chi-Square [$X^2(116, N = 208) = 161.36, p < .001$], a GFI of .92, a CFI of .96, and a SRMR of .067 (Figure C3 for measurement model and Table C3 for correlations among latent constructs). Lastly, the Wave 2 maternal measurement model resulted in a significant Chi-Square [$X^2(118, N = 208) = 154.59, p < .05$], a GFI of .93, a CFI of .97, and a SRMR of .061 (see Figure C4 for measurement model and Table C4 for correlations among latent constructs).

Wave 1 Paternal Structural Model

Fit indices for the Wave 1 paternal structural model indicated the model fit the data; although the Chi-Square was significant [$X^2(116, N = 208) = 168.98, p < .001$], the GFI and CFI were large and the SRMR was small; .92, .96, and .071



Note. t-values are in parentheses and all latent constructs are correlated.

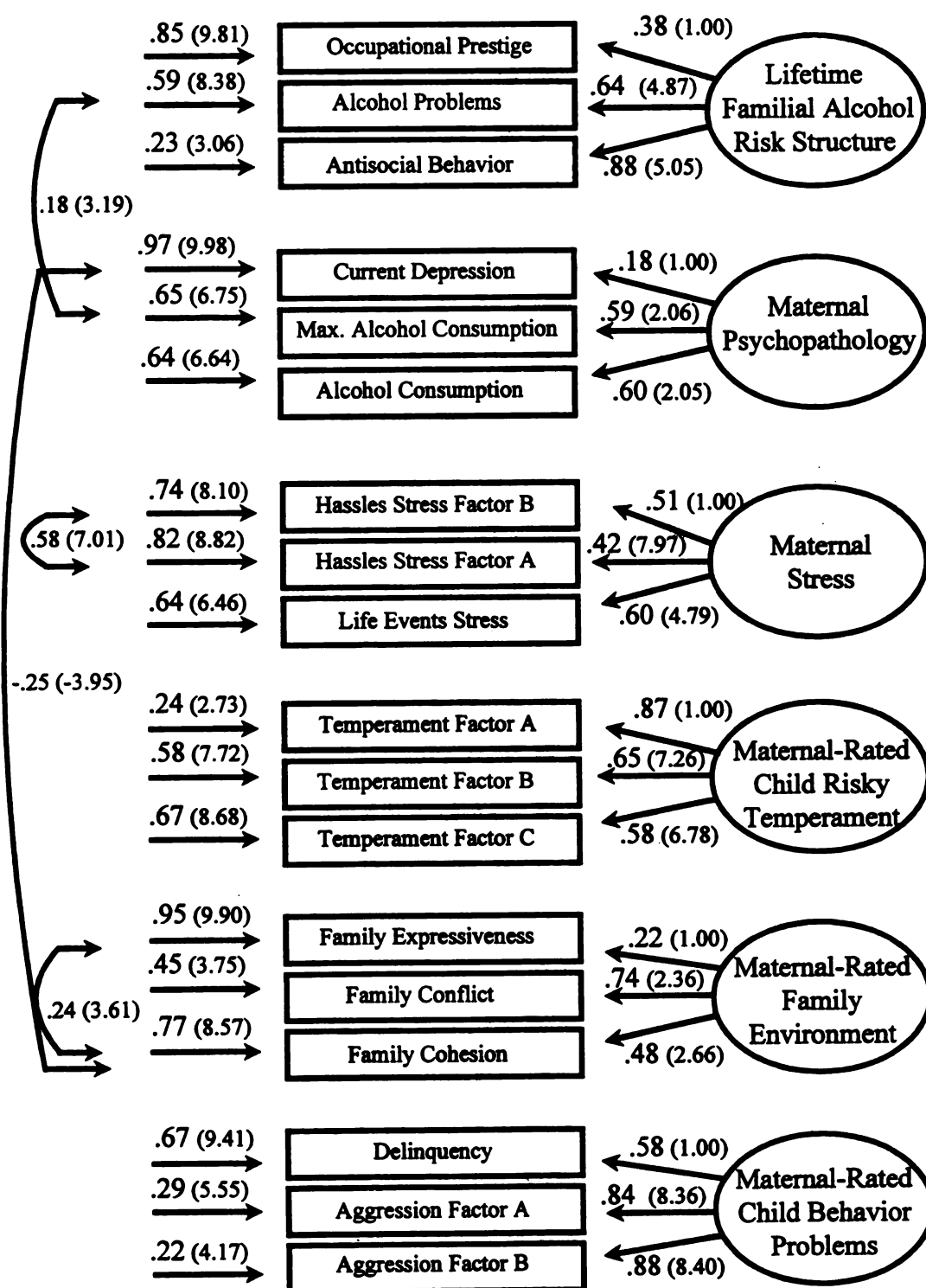
Figure C1. Measurement model for Wave 1 paternal variables for entire sample.

Table C1

Correlations Among Wave 1 Latent Constructs for Fathers Only (n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.50)	.70 (2.55)	.39 (3.18)	.18 (1.96)	-.53 (-3.56)	.41 (3.36)
2. Psychopathology		1.00 (1.63)	.84 (3.05)	.31 (2.11)	-.83 (-3.10)	.56 (2.86)
3. Stress			1.00 (3.65)	.08 (0.92)	-.32 (-3.06)	.27 (3.03)
4. Child Risky Temperament				1.00 (5.64)	.03 (0.33)	.36 (3.78)
5. Family Environment					1.00 (4.25)	-.48 (-4.23)
6. Child Behavior Problems						1.00 (4.61)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

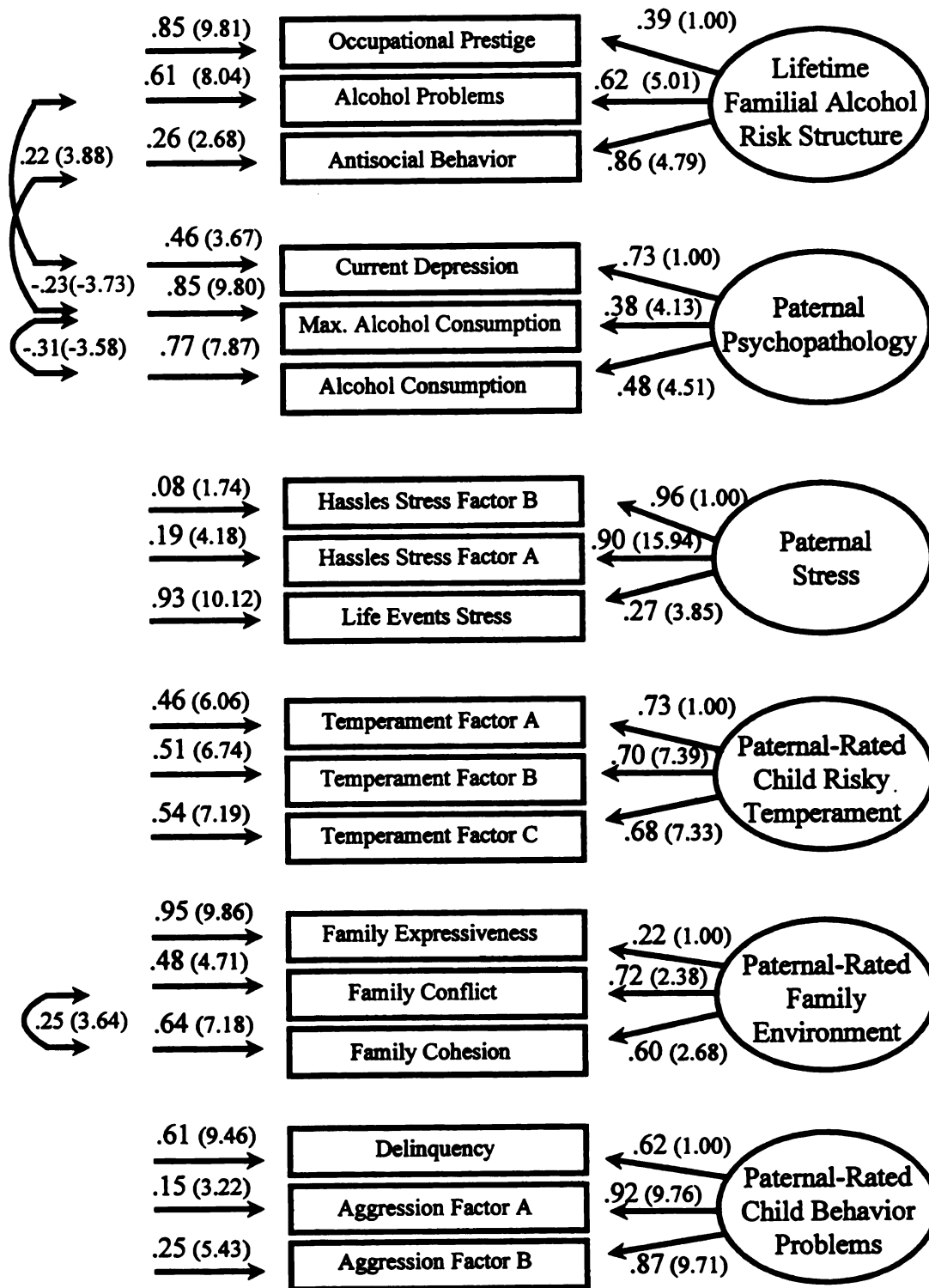
Figure C2. Measurement model for Wave 1 maternal variables for entire sample.

Table C2

Correlations Among Wave 1 Latent Constructs for Mothers Only (n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.63)	.66 (1.90)	.70 (3.50)	.04 (0.48)	-.49 (-2.04)	.55 (3.71)
2. Psychopathology		1.00 (1.07)	.53 (1.82)	-.03 (-0.31)	-.40 (-1.42)	.20 (1.44)
3. Stress			1.00 (3.01)	.07 (0.58)	-.60 (-2.09)	.51 (3.59)
4. Child Risky Temperament				1.00 (5.96)	.33 (1.99)	.26 (2.90)
5. Family Environment					1.00 (1.26)	-.30 (-1.90)
6. Child Behavior Problems						1.00 (4.26)

Note. Numbers in parentheses represent *t*-values.



Note. t-values are in parentheses and all latent constructs are correlated.

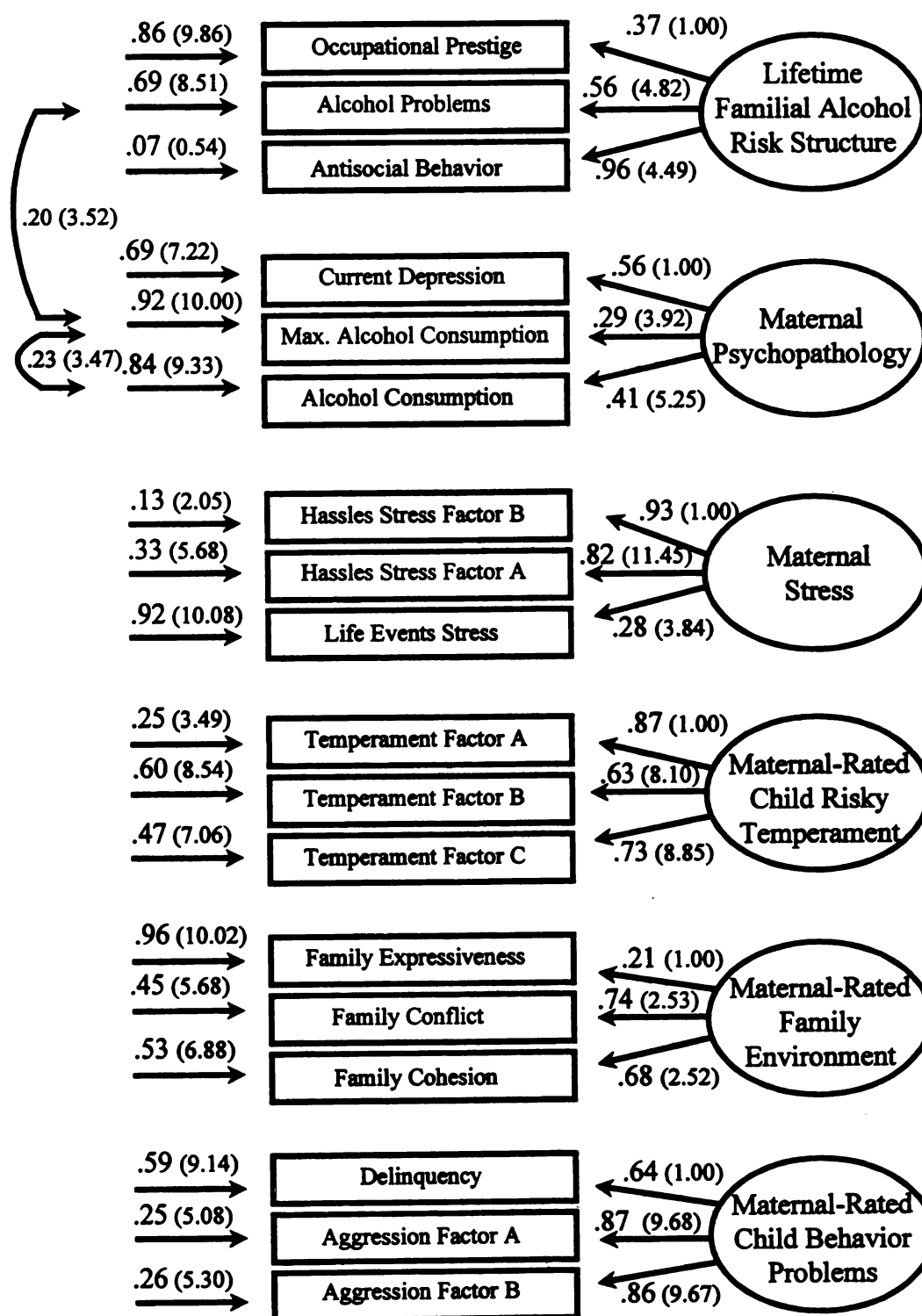
Figure C3. Measurement model for Wave 2 paternal variables for entire sample.

Table C3

Correlations Among Wave 2 Latent Constructs for Fathers Only (n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.66)	.62 (3.89)	.30 (2.94)	.29 (2.68)	-.50 (-2.06)	.35 (3.10)
2. Psychopathology		1.00 (3.66)	.66 (6.36)	.26 (2.75)	-.36 (-2.02)	.35 (3.66)
3. Stress			1.00 (8.49)	.12 (1.38)	-.25 (-1.84)	.31 (3.71)
4. Child Risky Temperament				1.00 (5.05)	-.13 (-1.15)	.29 (3.12)
5. Family Environment					1.00 (1.25)	-.53 (-2.24)
6. Child Behavior Problems						1.00 (4.78)

Note. Numbers in parentheses represent *t*-values.



Note. t-values are in parentheses and all latent constructs are correlated.

Figure C4. Measurement model for Wave 2 maternal variables for entire sample.

Table C4

Correlations Among Wave 2 Latent Constructs for Mothers Only (n=208)

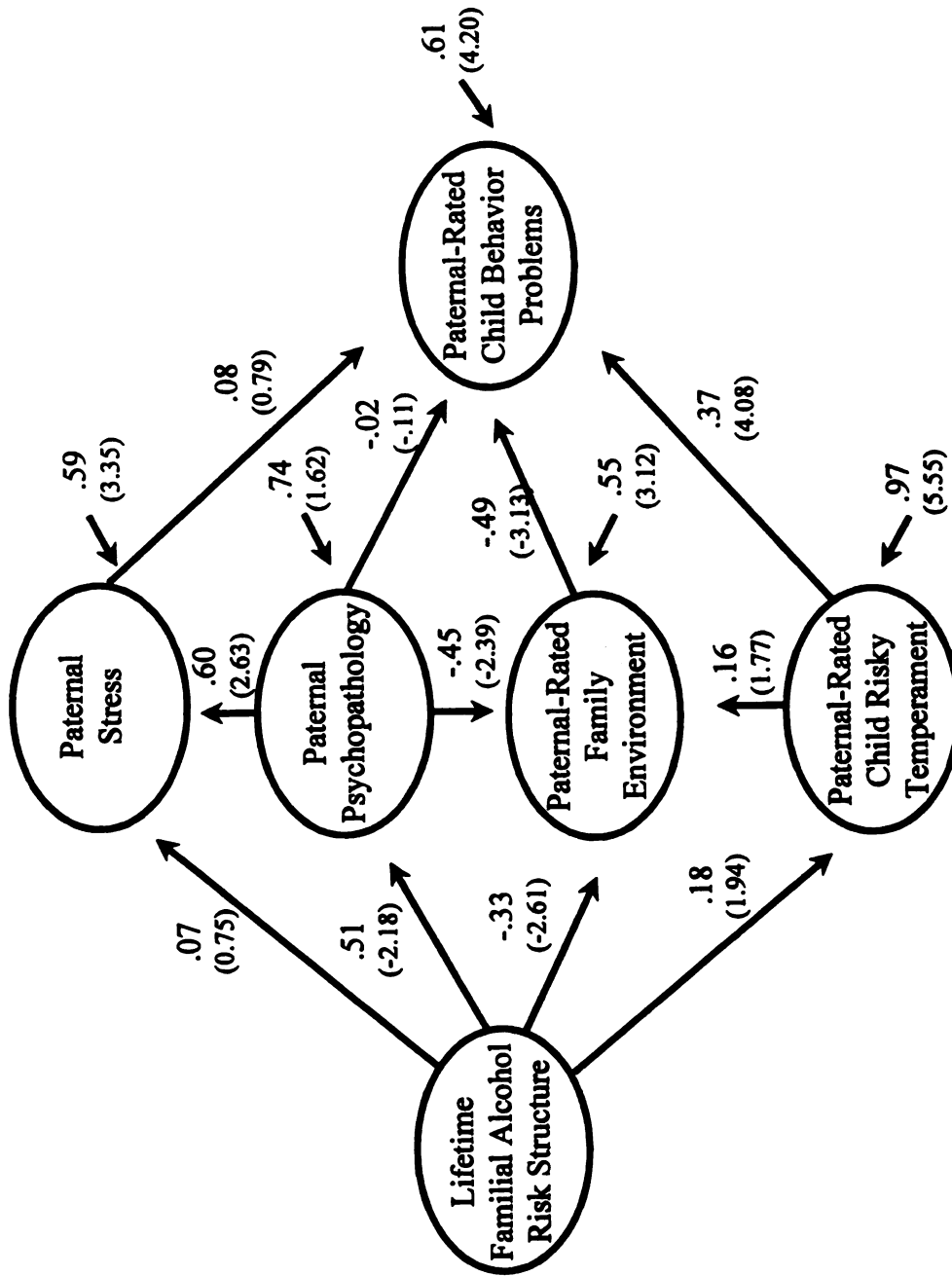
Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.56)	.68 (3.47)	.37 (3.24)	.16 (1.87)	-.29 (-1.86)	.36 (3.09)
2. Psychopathology		1.00 (3.18)	.85 (6.04)	.22 (1.79)	-.77 (-2.33)	.69 (4.72)
3. Stress			1.00 (7.58)	.15 (1.83)	-.51 (-2.34)	.33 (3.81)
4. Child Risky Temperament				1.00 (6.44)	.13 (1.20)	.29 (3.30)
5. Family Environment					1.00 (1.29)	-.52 (-2.31)
6. Child Behavior Problems						1.00 (4.89)

Note. Numbers in parentheses represent t-values.

respectively (see Figure C5 for structural model). Results of the model demonstrated that a more dense lifetime familial alcohol risk structure directly predicted more paternal psychopathology, a worse quality family environment, and heightened levels of child risky temperament, but not higher levels of paternal stress. Increased levels of paternal psychopathology directly predicted more paternal stress and also a worse quality family environment. Child risky temperament and a poorer quality family environment predicted increased levels of child externalizing behavior problems. The hypothesized paths from increased levels of paternal stress to more child behavior problems and from increased levels of paternal psychopathology to more behavior problems were not significant. Lastly, the hypothesized path from child risky temperament to quality of the family environment was significant but the direction of relationship was opposite to what was expected.

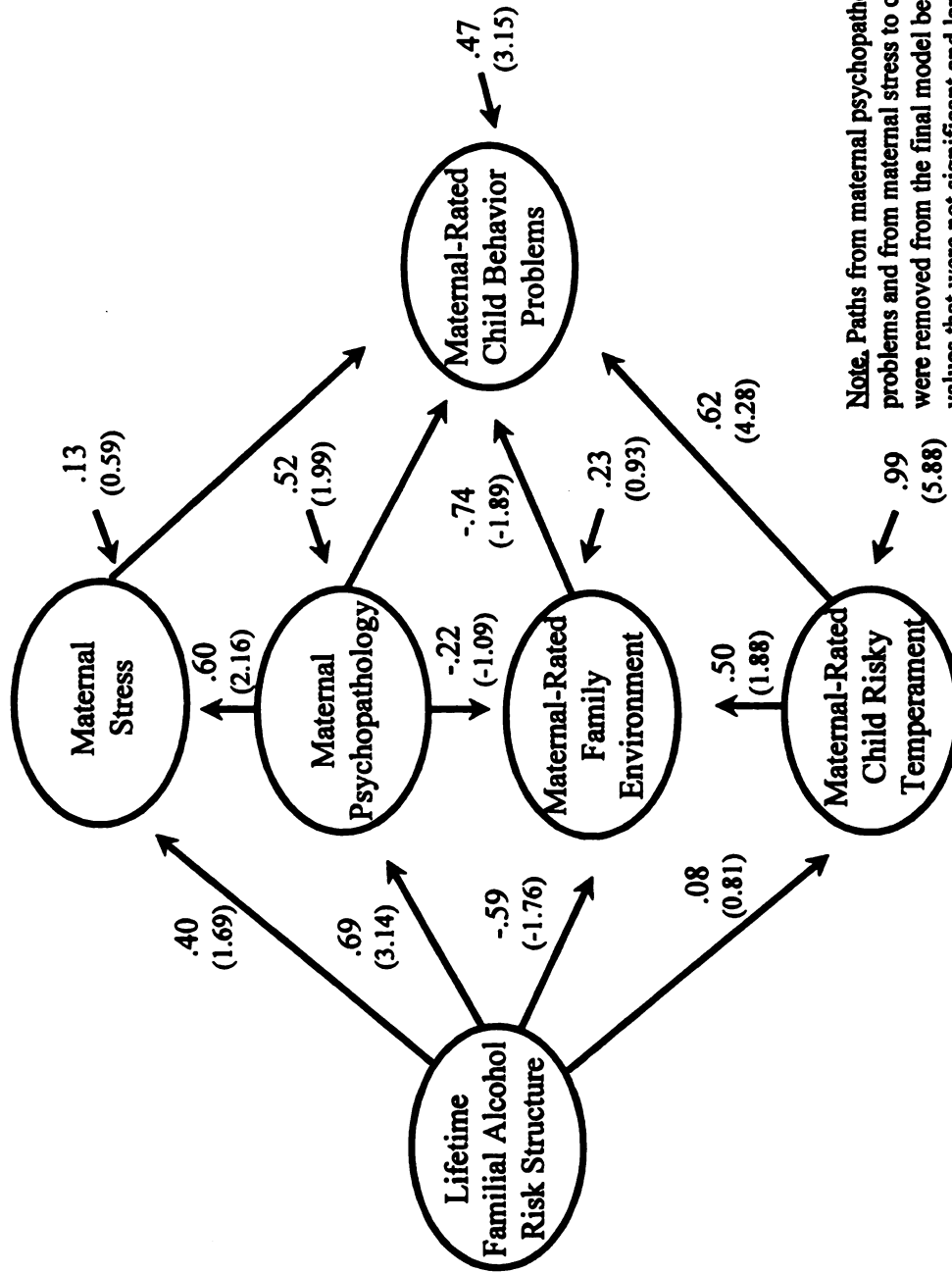
Wave 1 Maternal Structural Model

In order to interpret the results of the Wave 1 maternal structural model, two hypothesized paths were removed prior to estimating the final model. The paths leading from maternal stress to child behavior problems and from maternal psychopathology to behavior problems were consistently larger than 1.0 in the final completely standardized solution. However, because these large values were not significant the paths were removed and only the remaining paths were estimated. The fit of the final structural model was acceptable. The Chi-Square was significant [$X^2(123, N = 208) = 150.53, p < .05$] and the SRMR was larger than expected (.12), however, the GFI was .92, and the CFI was .99 (see Figure C6 for structural model). This model indicated that at Wave 1 a more dense lifetime familial alcohol risk



Note. t-values are in parentheses.

Figure C5. Structural model representing relationships among all Wave 1 paternal latent constructs for entire sample.



Note. Paths from maternal psychopathology to child behavior problems and from maternal stress to child behavior problems were removed from the final model because they resulted in values that were not significant and larger than 1 and t-values are in parentheses.

Figure C6. Structural model representing relationships among all Wave 1 maternal latent constructs for entire sample.

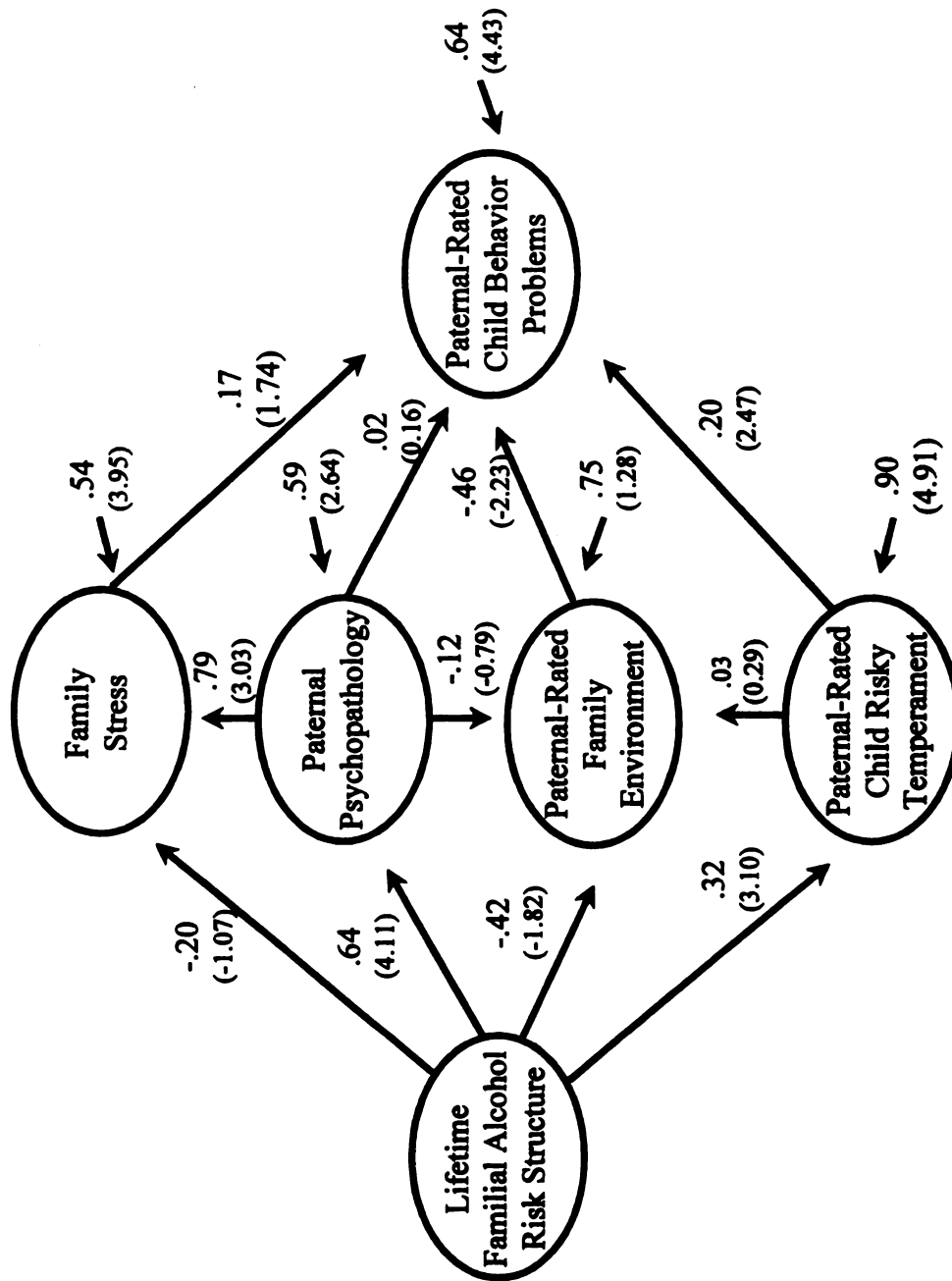
structure predicted higher levels of maternal psychopathology, higher levels of maternal stress, and a worse quality family environment but not higher levels of child risky temperament. Lower levels of maternal psychopathology were predictive of less family stress but not a better quality family environment. Child risky temperament was directly related to a better quality family environment and more child risky temperament and a poorer quality family environment increased levels of child externalizing behavior problems.

Wave 2 Paternal Structural Model

The Wave 2 paternal structural model fit the data well; although the Chi-Square was significant [$X^2(120, N = 208) = 162.99, p < .01$], the GFI was .92, the CFI was .96, and the SRMR was .068 (see Figure C7 for structural model). Results indicated that, as hypothesized, a more dense familial alcohol risk structure predicted higher levels of paternal psychopathology, a worse quality family environment, and more child risky temperament (but not more paternal stress). Paternal psychopathology predicted higher levels of paternal stress, however, neither paternal psychopathology nor child risky temperament predicted the quality of the family environment. Finally, more paternal stress, child risky temperament, and a worse quality family environment predicted child externalizing behavior problems but increased levels of paternal psychopathology did not.

Wave 2 Maternal Structural Model

The Wave 2 maternal structural model resulted in a good fit. The Chi-Square was significant [$X^2(122, N = 208) = 157.91, p < .05$], but the GFI and CFI were large and the SRMR was small; .92, .97, and .064 respectively (see Figure C8 for



Note. t-values are in parentheses.

Figure C7. Structural model representing relationships among all Wave 2 paternal latent constructs for entire sample.

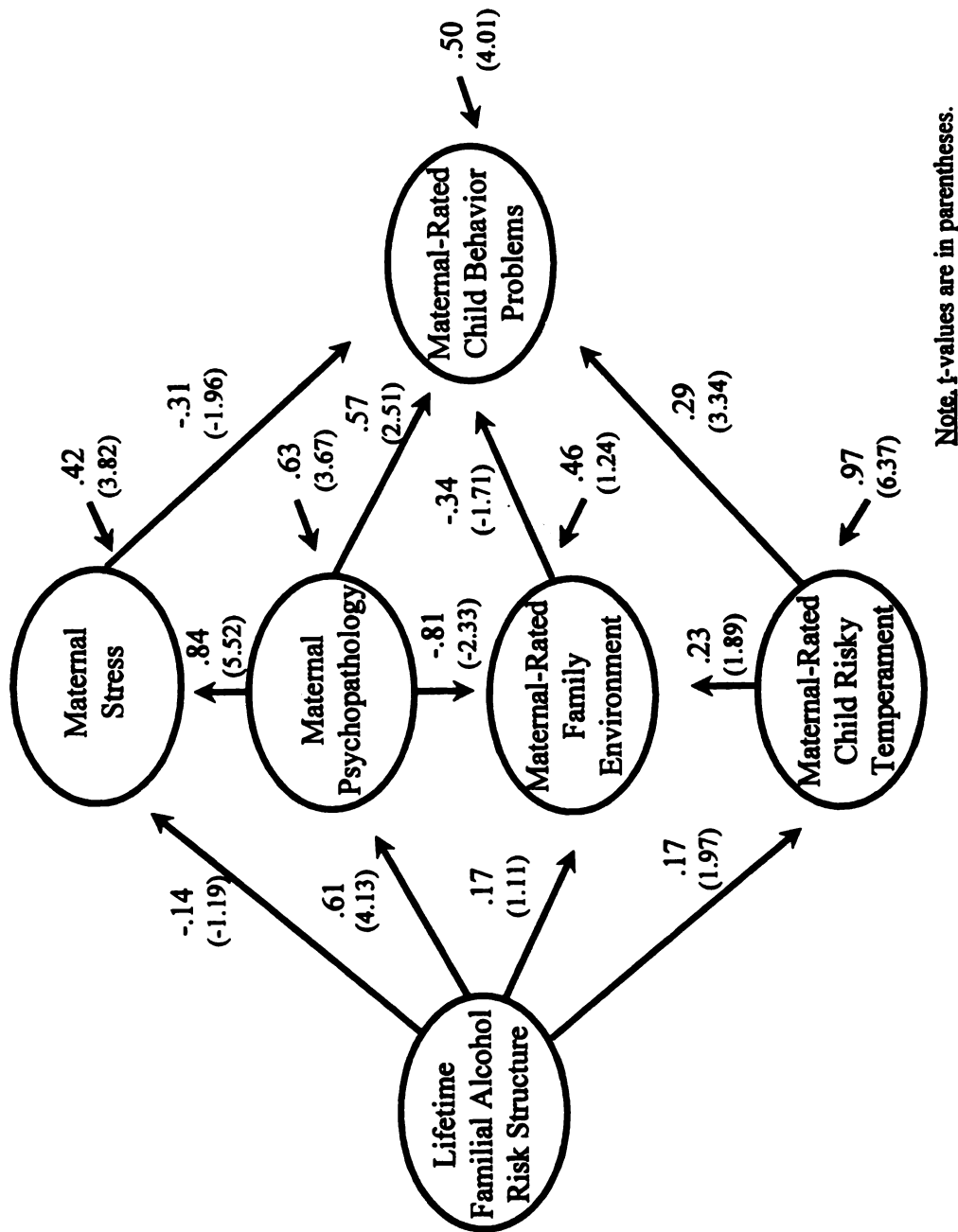
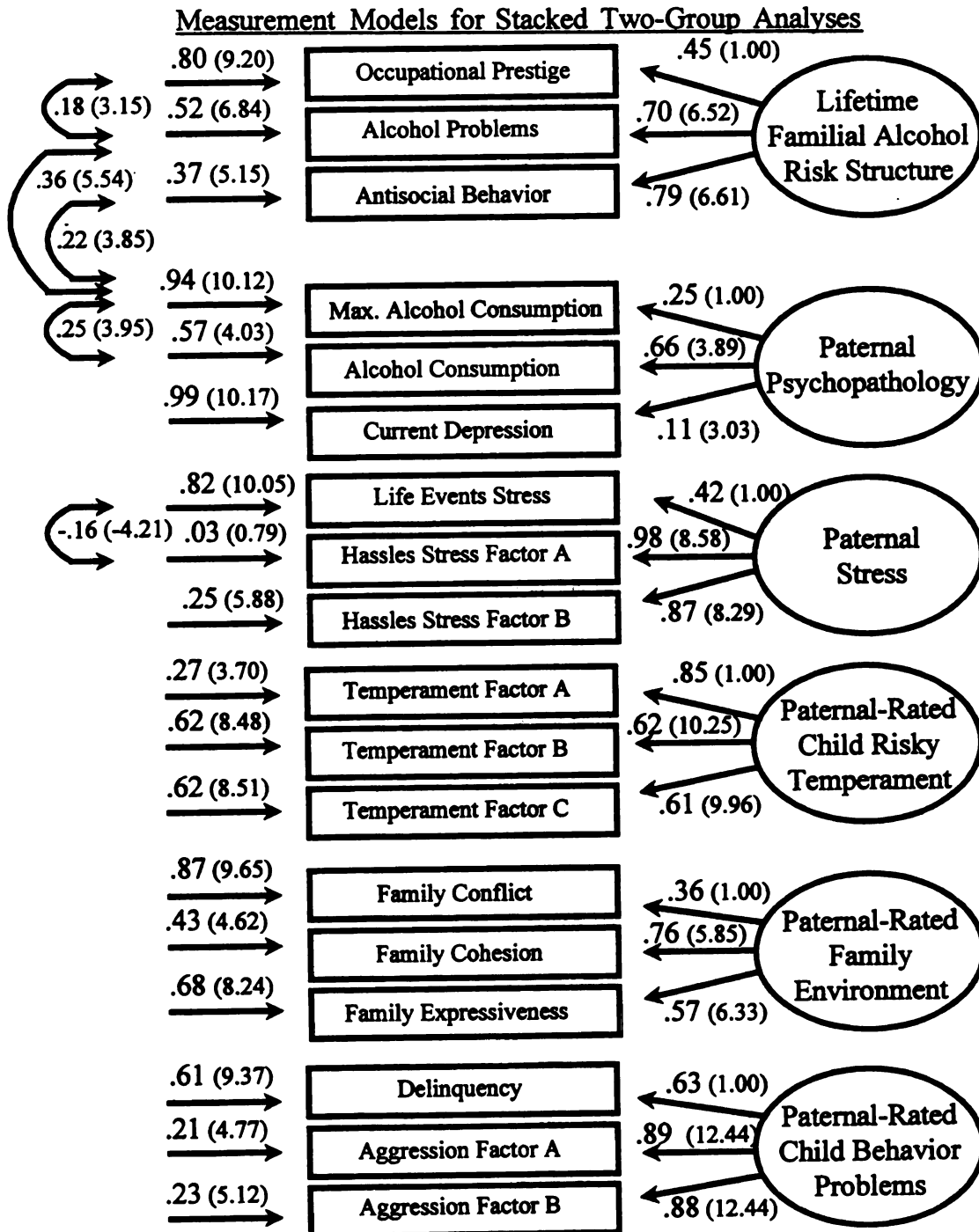


Figure C8. Structural model representing relationships among all Wave 2 maternal latent constructs for entire sample.

structural model). Results revealed that a more dense familial lifetime alcohol risk structure only predicted higher levels of maternal psychopathology and more child risky temperament but not more maternal stress and a worse quality family environment. More maternal psychopathology directly predicted more maternal stress and a worse quality family environment. More child externalizing problems was predicted by more maternal psychopathology, a poorer quality family environment, and child risky temperament. Contrary to what was expected, child risky temperament predicted a better quality family environment. Finally, also contrary to what was expected, higher levels of maternal stress resulted in less child externalizing behavior problems. This finding does not occur in any other model and is likely due to estimation problems: LISREL does not constrain estimates of variances to be positive and occasionally estimates deviate from acceptable values (Hayduk, 1987).

APPENDIX D



Note. t-values are in parentheses and all latent constructs are correlated.

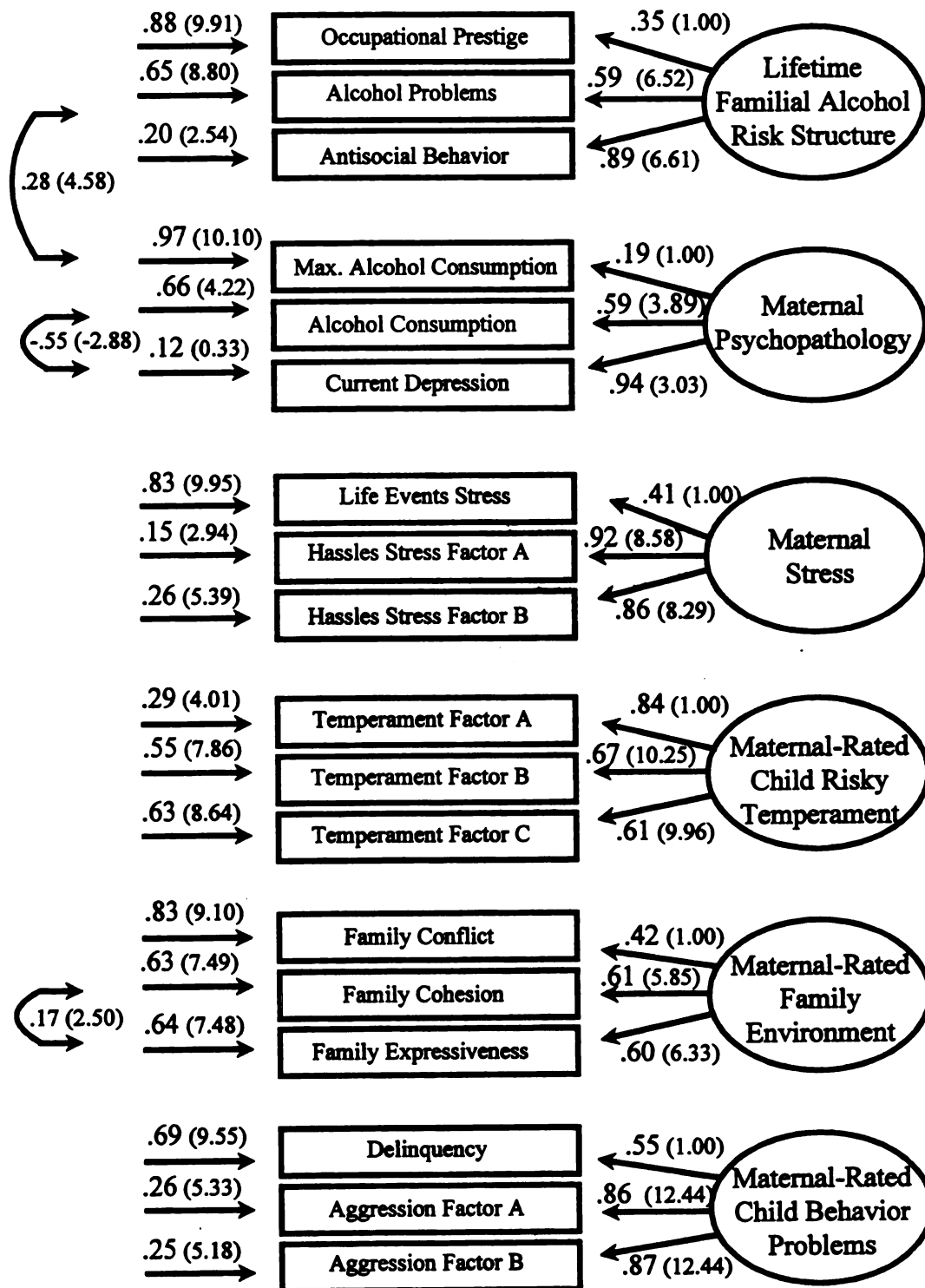
Figure D1. Measurement model for Wave 1 two-group stacked analysis: Solution for paternal data for entire sample.

Table D1

Correlations Among Wave 1 Latent Constructs for Stacked Models - Fathers Only**(n=208)**

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.57)	.68 (2.87)	.40 (3.71)	.19 (2.14)	-.39 (-3.14)	.42 (3.99)
2. Psychopathology		1.00 (1.73)	.83 (3.35)	.30 (2.18)	-.70 (-2.96)	.53 (3.04)
3. Stress			1.00 (4.12)	.08 (0.97)	-.24 (-2.46)	.27 (3.17)
4. Child Risky Temperament				1.00 (6.60)	.13 (1.32)	.35 (3.93)
5. Family Environment					1.00 (3.14)	-.26 (-2.54)
6. Child Behavior Problems						1.00 (5.65)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

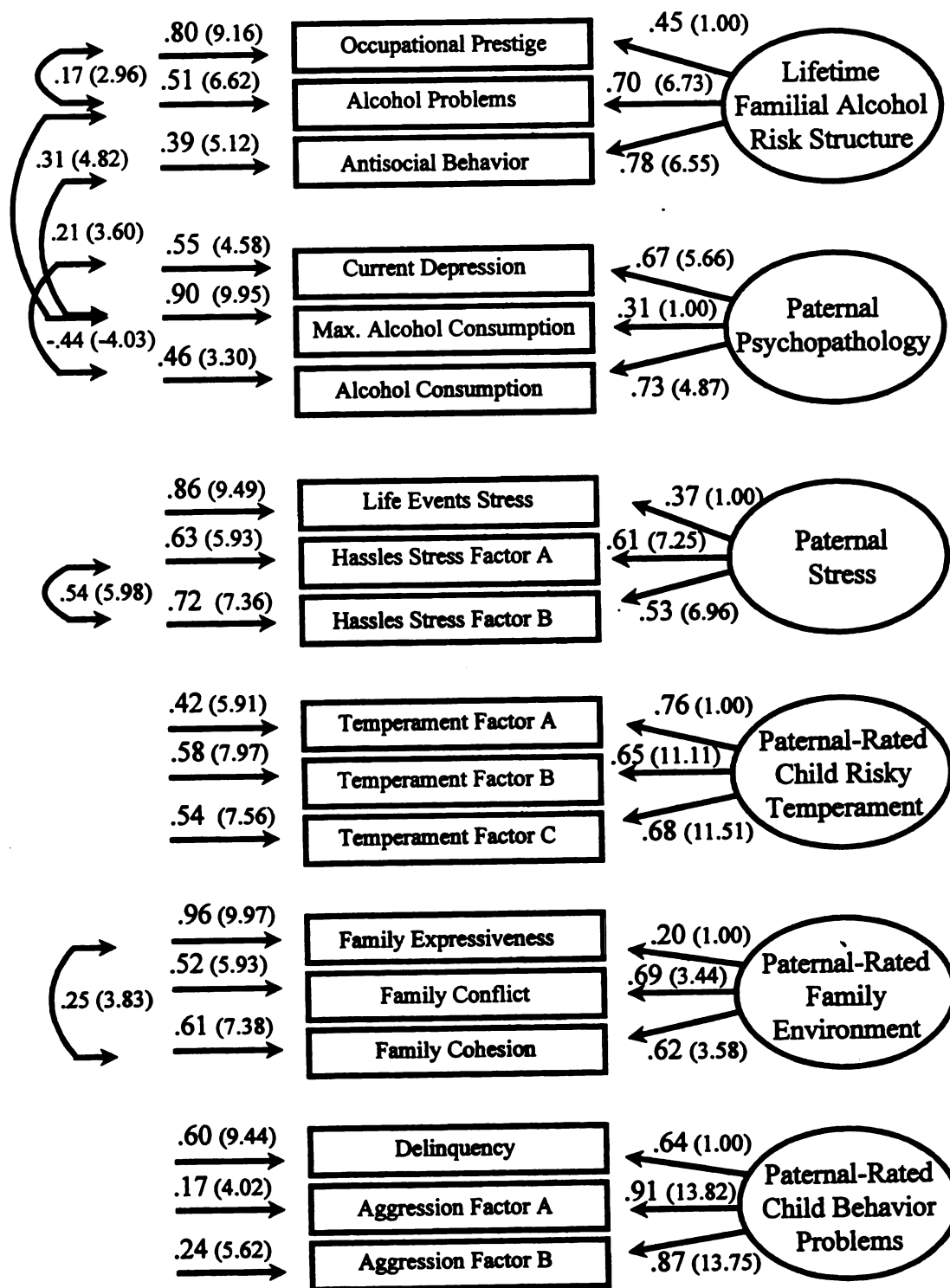
Figure D2. Measurement model for Wave 1 two-group stacked analysis: Solution for maternal data for entire sample.

Table D2

Correlations Among Wave 1 Latent Constructs for Stacked Models - Mothers Only
(n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.33)	.35 (2.59)	.37 (3.44)	.04 (0.52)	-.50 (-3.43)	.55 (4.36)
2. Psychopathology		1.00 (2.21)	.34 (2.67)	.04 (0.57)	-.51 (-2.72)	.21 (2.22)
3. Stress			1.00 (4.00)	.08 (0.94)	-.43 (-3.37)	.34 (3.60)
4. Child Risky Temperament				1.00 (6.56)	.31 (2.69)	.26 (2.98)
5. Family Environment					1.00 (3.00)	-.29 (-2.59)
6. Child Behavior Problems						1.00 (5.54)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

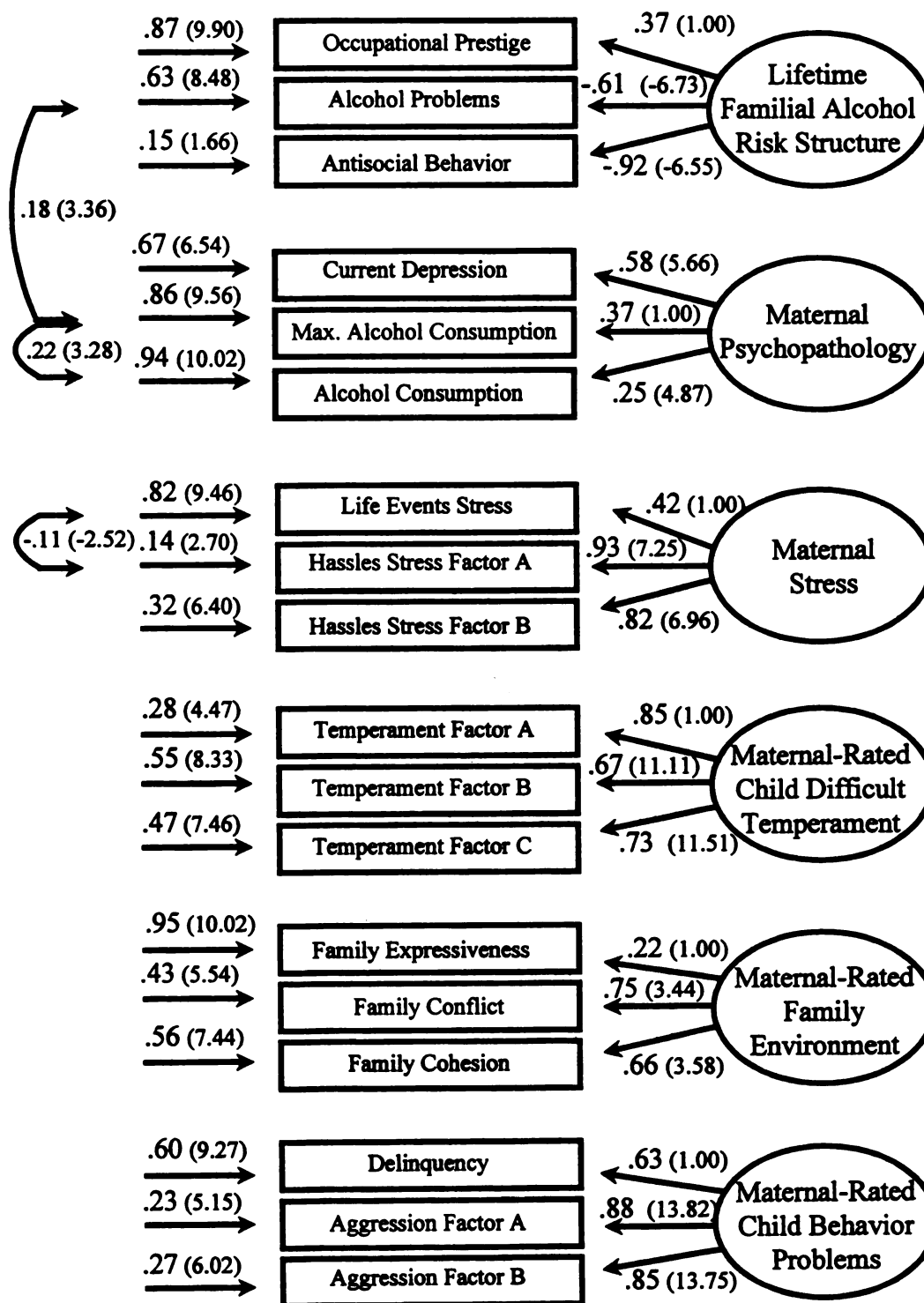
Figure D3. Measurement model for Wave 2 two-group stacked analysis: Solution for paternal data for entire sample.

Table D3

Correlations Among Wave 2 Latent Constructs for Stacked Models - Fathers Only(n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.60)	.39 (3.15)	.60 (3.74)	.29 (2.95)	-.49 (-2.67)	.37 (3.72)
2. Psychopathology		1.00 (3.20)	.93 (4.08)	.25 (2.59)	-.29 (-2.16)	.32 (3.26)
3. Stress			1.00 (2.75)	.27 (2.11)	-.46 (-2.32)	.50 (3.73)
4. Child Risky Temperament				1.00 (6.27)	-.13 (-1.21)	.30 (3.35)
5. Family Environment					1.00 (1.69)	-.53 (-2.88)
6. Child Behavior Problems						1.00 (6.03)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

Figure D4. Measurement model for Wave 2 two-group stacked analysis: Solution for maternal data for entire sample.

Table D4

Correlations Among Wave 2 Latent Constructs for Stacked Models - Mothers Only(n=208)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.41)	.70 (4.25)	.42 (3.60)	.16 (1.85)	-.31 (-2.32)	.38 (3.66)
2. Psychopathology		1.00 (3.18)	.83 (4.88)	.26 (-2.11)	-.70 (-2.92)	.67 (4.96)
3. Stress			1.00 (3.52)	.15 (1.82)	-.51 (-2.81)	.35 (3.63)
4. Child Risky Temperament				1.00 (6.91)	.12 (1.21)	.29 (3.35)
5. Family Environment					1.00 (1.77)	-.53 (-2.97)
6. Child Behavior Problems						1.00 (6.02)

Note. Numbers in parentheses represent t-values.

APPENDIX E**Family Environment and Child Risky Temperament**

To further examine the unexpected positive relationship between child risky temperament and a better quality family environment a series of exploratory correlational analyses were conducted. Three indexes were computed at each time period. A family environment index was created by first standardizing the three subscales of more cohesion, more expressiveness, and less conflict and then summing them. Next, the three dimensions of high activity level, high reactivity, and high adaptability were standardized and also summed to form the risky temperament index. Finally, the third index was composed of the summed standardized dimensions of only high activity level and high reactivity (but not adaptability). This final index was composed of the two dimensions of temperament that have received the most support from the literature (see Tarter, 1988).

Once the indices were created four correlations were computed. The first correlation was between the index for Wave 1 child risky temperament and the index for Wave 1 family environment ($r = .09, p < .05$). Just as in the SEMs this association was positive and significant suggesting that the better the quality of the family environment the riskier the child temperament (see Figure E1 for graphical representation). Next, a correlation was conducted between the Wave 1 family environment index and the Wave 1 index composed of activity level and reactivity and resulted in a negative but not significant relationship ($r = -.005$; see Figure E2 for graphical representation). These analyses were repeated for the Wave 2 data; the

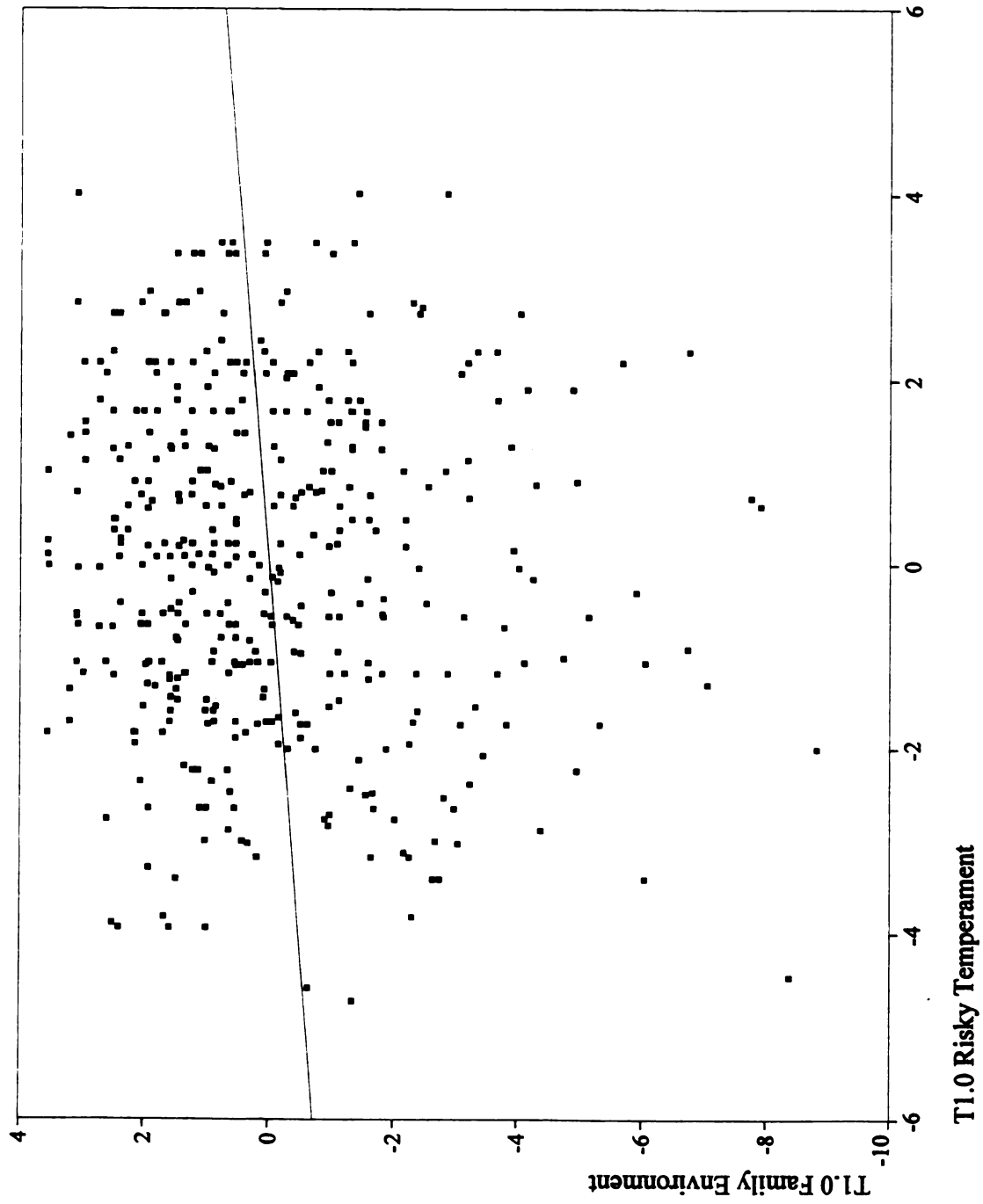


Figure E1. Relationship between Wave 1 family environment index and Wave 1 child risky temperament index.

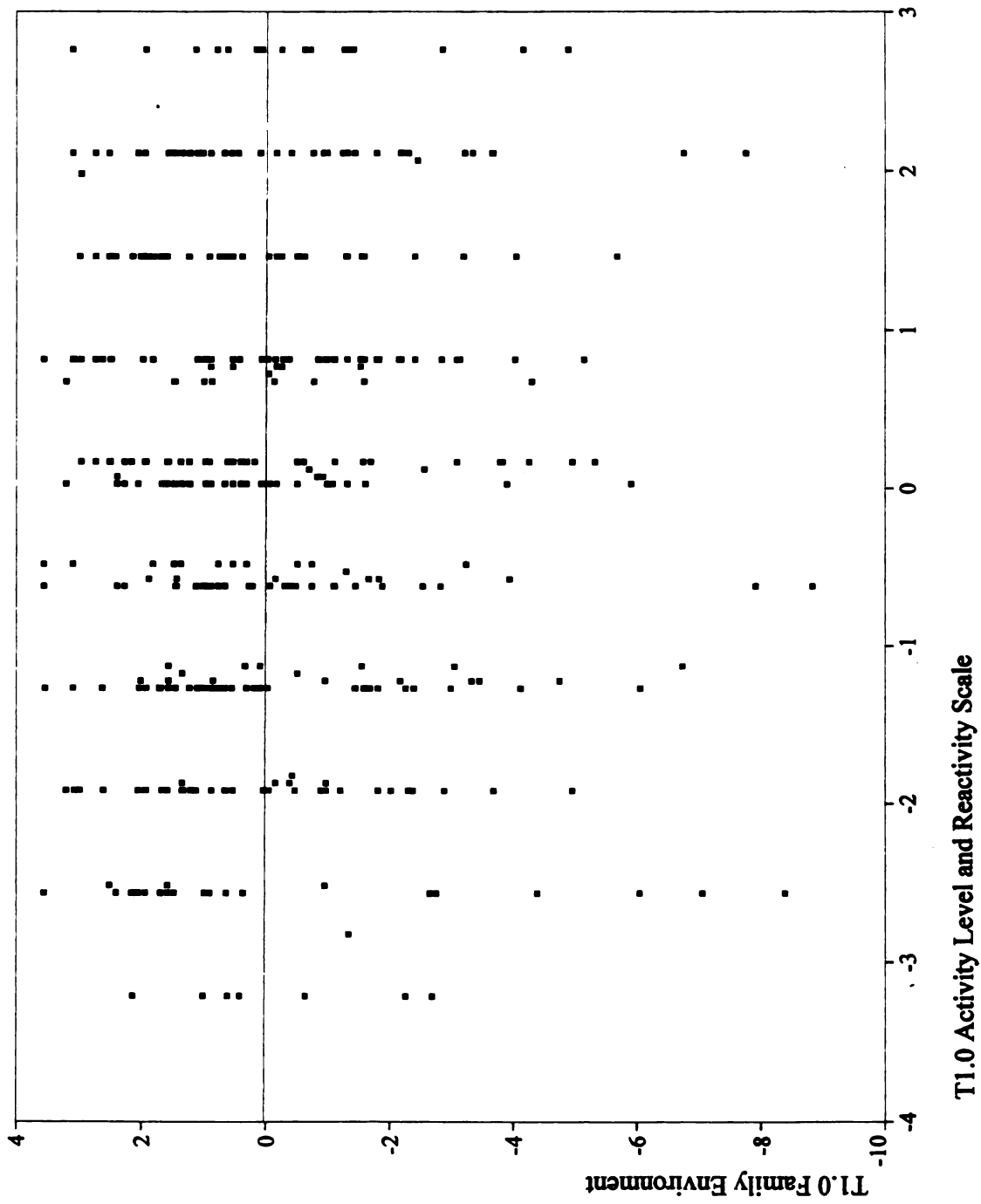


Figure E2. Relationship between Wave 1 family environment index and Wave 1 activity level and reactivity index.

relationship between Wave 2 family environment and Wave 2 risky temperament (see Figure E3 for graphical representation) was negative but non-significant ($r = -.02$) and the association between Wave 2 family environment and Wave 2 activity level and reactivity (see Figure E4 for graphical representation) was negative but significant ($r = -.13$, $p < .001$).

These results indicate that when the adaptability dimension of temperament is removed from the risky temperament index, the association between family environment and activity level and reactivity becomes increasingly negative. In other words, adaptability is most likely responsible for the positive relationship between child risky temperament and a better quality family environment.

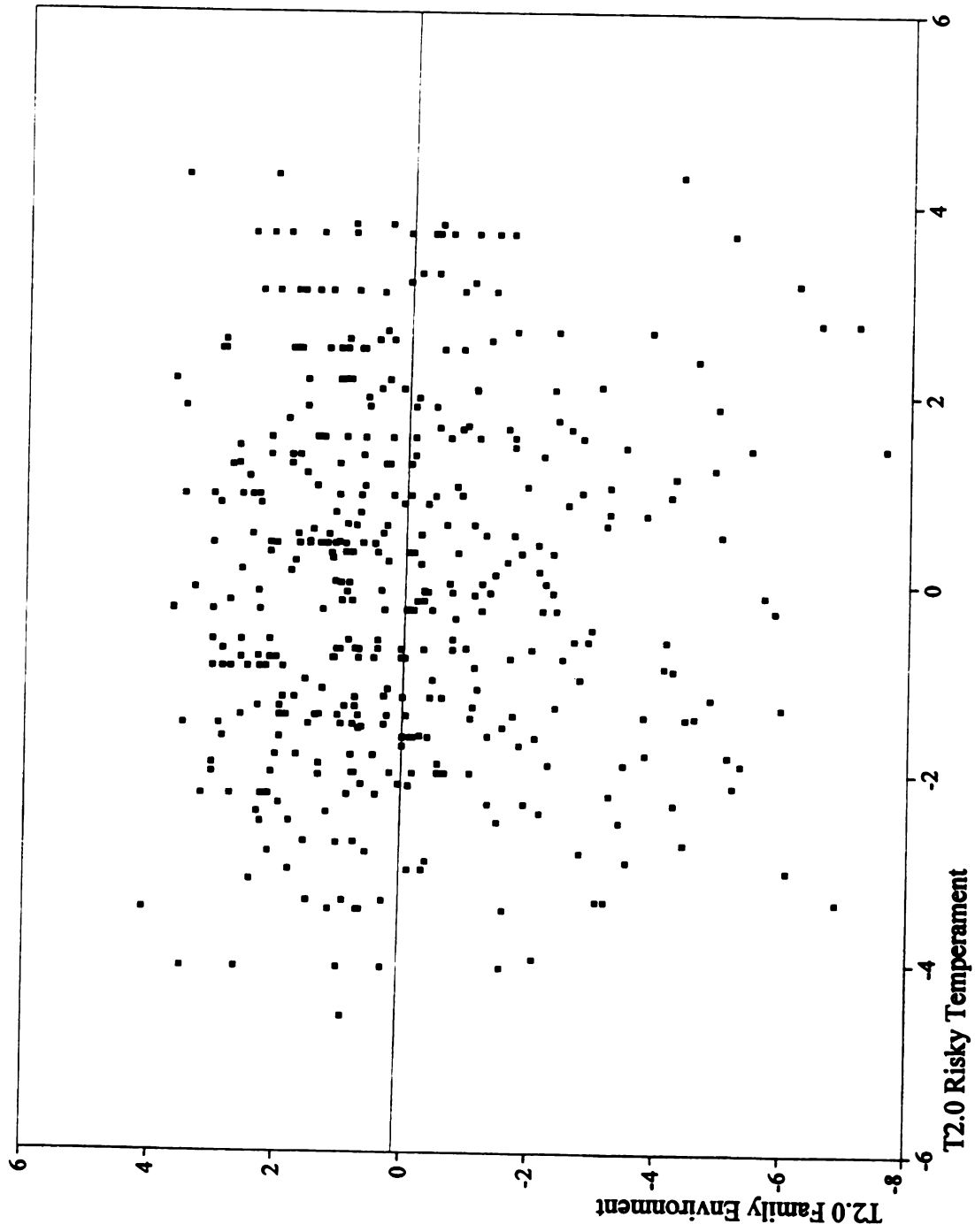


Figure E3. Relationship between Wave 2 family environment index and Wave 2 child risky temperament index.

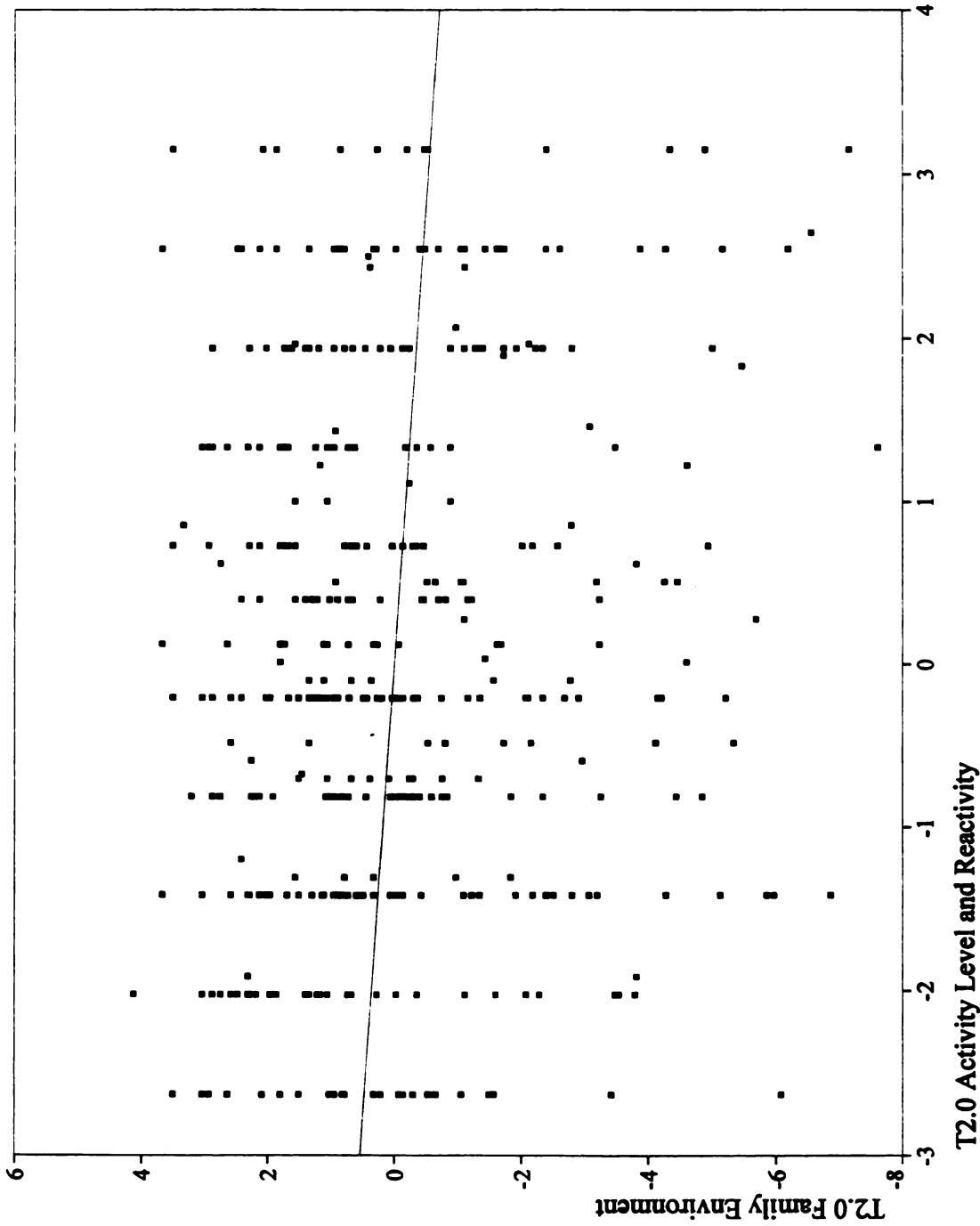


Figure E4. Relationship between Wave 2 family environment index and Wave 2 activity level and reactivity index.

APPENDIX F**Discriminators of Decoupled and Coupled Status**

A direct discriminant function analysis was performed to determine prediction of membership in two groups (coupled $n = 188$ and decoupled $n = 20$) from 16 parental-level predictor variables. The 16 parental variables were maternal and paternal lifetime alcohol problems, maternal and paternal antisocial behavior, maternal and paternal depression, maternal and paternal alcohol diagnosis, maternal and paternal alcohol consumption, maternal and paternal maximum alcohol consumption, maternal and paternal daily hassles stress, and maternal and paternal life events stress.

One discriminant function was calculated and revealed a strong association between the groups and predictors [$X^2(16, N = 208) = 39.57, p < .001$]. The criteria for discrimination among the predictors was having a discriminant coefficient of at least .30 and a significant F -test statistic. Results revealed that all variables with the exception of paternal lifetime alcohol problems, paternal daily hassles stress, and maternal maximum alcohol consumption significantly discriminated between coupled and decoupled individuals. Overall, 92% of individuals were classified correctly using this procedure compared to 82% that would be classified by chance alone. Results indicated that a variety of parental predictors can significantly discriminate between coupled and decoupled individuals.

APPENDIX G**SEMs and Coupled Parents Only**

At Wave 1 all 208 families in the present sample were intact, however, by Wave 2 a small number of families (20) were decoupled (separated or divorced). To examine the associations among the variables for coupled families only and to examine if there differences in the patterns of association for the entire sample versus the subsample of coupled families, all SEMs were re-analyzed with the coupled individuals only

Cross-Sectional Measurement Models

Four cross-sectional measurement models were tested first; one for each parent at each of the two time periods. Tables G1-G4 contain the means, standard deviations, and correlations for variables included in the present analyses. Just as for the descriptive statistics provided for the entire sample, the alcohol consumption variable for coupled parents only showed a larger SD relative to its mean (see Figures G1-G4 for graphical representation). The SDs of the family environment variables of expressiveness, conflict, and cohesion were very small relative to the means indicating very little variability in the distribution of these variables.

The fit indices of each of the four measurement models indicated that all models fit the data well. The Wave 1 paternal model resulted in a significant Chi-Square [$X^2(113, N = 188) = 172.40, p < .001$], a GFI of .91, a CFI of .95, and a SRMR was .064 (see Figure G5 for measurement model and Table G5 for correlations among latent constructs). The Wave 1 maternal model also had a

Table G1

Means, Standard Deviations, and Correlations For All Wave 1 Paternal Variables for Coupled Fathers Only (n=188)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.15*	.34***	.19**	.19**	.09	.16*	.08	.29***
2. Lifetime Alcohol Problems		1.00	.57***	.21**	.52***	.32***	.24***	.16*	.29***
3. Lifetime Antisocial Behavior			1.00	.29***	.39***	.27***	.30***	.24***	.35***
4. Current Depression				1.00	.06	.07	.55***	.47***	.39***
5. Maximum Alcohol Consumption					1.00	.36***	.11	.07	.22**
6. Alcohol Consumption						1.00	.06	.05	.10
7. Hassles Stress Factor A							1.00	.86***	.45***
8. Hassles Stress Factor B								1.00	.28***
9. Life Events Stress									1.00
M	361.78	9.28	15.60	2.24	3.69	1921.45	15.24	13.93	1.00
SD	146.03	2.33	9.72	2.45	2.41	4567.61	12.82	13.19	1.37

*p<.05. **p<.01. ***p<.001.

(table continues)

Table G1 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.06	.02	.07	-.14*	-.09	-.18*	.10	.09	.13
2. Lifetime Alcohol Problems	.22*	.01	.15*	-.04	-.27***	-.09	.27***	.09	.20**
3. Lifetime Antisocial Behavior	.09	-.05	.10	-.05	-.31***	-.15*	.22**	.18*	.19**
4. Current Depression	.09	.00	.16*	-.13	-.30***	-.26***	.21**	.14	.19**
5. Maximum Alcohol Consumption	.21**	-.03	.09	-.10	-.04	-.04	.21**	.09	.14
6. Alcohol Consumption	.06	.04	.01	-.14	-.14	-.17*	.23**	.08	.11
7. Hassles Stress Factor A	.05	-.04	.10	-.07	-.20**	-.16*	.08	.07	.13
8. Hassles Stress Factor B	.03	-.08	.08	.01	-.18*	-.11	.07	.07	.13
9. Life Events Stress	.02	.01	.04	-.15*	-.20**	-.21**	.17*	.09	.09
10. Child Temperament Factor A	1.00	.52***	.55***	.12	-.01	.12	.16*	.20**	.16*
11. Child Temperament Factor B		1.00	.45***	.06	.01	.08	.23**	.17*	.08
12. Child Temperament Factor C			1.00	.09	.04	.09	.12	.17*	.14
13. Family Expressiveness				1.00	.19**	.37***	-.16*	.04	-.08
14. Family Conflict					1.00	.34***	-.31***	-.07	-.16*
15. Family Cohesion						1.00	-.13	.02	-.10
16. Child Delinquency							1.00	.17*	.18*
17. Child Aggression Factor A								1.00	.75***
18. Child Aggression Factor B									1.00
<u>M</u>	3.21	2.96	2.52	14.98	14.70	16.57	1.77	5.21	4.90
<u>SD</u>	1.20	1.04	1.15	1.74	2.17	1.55	1.40	2.76	2.90

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table G2

Means, Standard Deviations, and Correlations For All Wave 1 Maternal Variables for Coupled Mothers Only (n=188)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	-.11	-.34***	-.15*	-.13	-.14	-.14	-.04	-.26***
2. Lifetime Alcohol Problems		1.00	.53***	.08	.46***	.38***	.22**	.12	.27***
3. Lifetime Antisocial Behavior			1.00	.26***	.33***	.35***	.30***	.20**	.36***
4. Current Depression				1.00	.06	.06	.37***	.30***	.23**
5. Maximum Alcohol Consumption					1.00	.47***	.03	-.04	.18*
6. Alcohol Consumption						1.00	.02	.00	.23**
7. Hassles Stress Factor A							1.00	.80***	.29***
8. Hassles Stress Factor B								1.00	.22**
9. Life Events Stress									1.00
M	361.78	9.84	9.70	2.54	2.27	380.27	16.18	14.75	0.91
SD	146.03	1.96	6.48	2.76	1.74	1217.46	11.97	11.39	1.16

*p<.05. **p<.01. ***p<.001.

(table continues)

Table G2 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.02	.14	.00	.24***	.23**	.15*	-.21**	-.09	-.13
2. Lifetime Alcohol Problems	-.01	-.06	.03	-.08	-.20**	-.18*	.18*	.17*	.26***
3. Lifetime Antisocial Behavior	.09	-.03	.12	-.09	-.29***	-.26***	.38***	.38***	.45***
4. Current Depression	.04	-.00	.08	-.11	-.26***	-.37***	.19*	.20**	.17*
5. Maximum Alcohol Consumption	-.01	-.04	.02	-.05	-.14	-.07	.06	-.01	.08
6. Alcohol Consumption	-.01	-.13	-.04	-.10	-.19**	-.21**	.10	.16*	.20**
7. Hassles Stress Factor A	.09	.07	.09	-.12	-.20**	-.25***	.26***	.24***	.30***
8. Hassles Stress Factor B	.03	.04	.07	-.09	-.18*	-.19**	.23**	.25***	.31***
9. Life Events Stress	.03	-.02	-.01	-.13	-.21**	-.14	.25***	.26***	.26***
10. Child Temperament Factor A	1.00	.57***	.51***	.11	.23**	.09	.08	.20**	.24***
11. Child Temperament Factor B		1.00	.37***	.08	.20**	.12	.02	.16*	.14
12. Child Temperament Factor C			1.00	.04	.12	.09	.12	.23***	.25***
13. Family Expressiveness				1.00	.13	.34***	-.10	-.06	-.06
14. Family Conflict					1.00	.39***	-.08	-.19**	-.28***
15. Family Cohesion						1.00	-.23**	-.13	-.19**
16. Child Delinquency							1.00	.50***	.47***
17. Child Aggression Factor A								1.00	.75***
18. Child Aggression Factor B									1.00
<u>M</u>	3.00	2.82	2.35	15.48	14.53	16.52	1.80	5.21	4.90
<u>SD</u>	1.29	0.99	1.17	1.71	2.03	1.68	1.61	2.76	2.90

*p<.05. **p<.01. ***p<.001.

Table G3

Means, Standard Deviations, and Correlations For All Wave 2 Paternal Variables for Coupled Fathers Only (n=188)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.15*	.34***	.18*	.12	.01	.10	.03	.19**
2. Lifetime Alcohol Problems		1.00	.57***	.25***	.40***	.19**	.17*	.15*	.28***
3. Lifetime Antisocial Behavior			1.00	.20**	.30***	.21**	.28***	.22**	.33***
4. Current Depression				1.00	.10	-.07	.47***	.45***	.15*
5. Maximum Alcohol Consumption					1.00	.29***	.06	.07	.08
6. Alcohol Consumption						1.00	.04	.04	.20**
7. Hassles Stress Factor A							1.00	.84***	.18*
8. Hassles Stress Factor B								1.00	.13
9. Life Events Stress									1.00
M	361.78	9.28	15.60	2.72	3.31	956.72	13.61	12.37	0.78
SD	146.03	2.33	9.72	3.26	2.31	2168.75	12.21	11.93	1.00

*p<.05. **p<.01. ***p<.001.

(table continues)

Table G3 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.04	.03	.03	-.03	-.13	-.18*	.13	.19**	.18*
2. Lifetime Alcohol Problems	.22**	.24***	.20**	.02	-.22**	-.14*	.24***	.24***	.22**
3. Lifetime Antisocial Behavior	.14	.09	.18*	.07	-.28***	-.20**	.22**	.25***	.27***
4. Current Depression	.13	.12	.13	-.13	-.24***	-.33***	.16*	.28***	.23**
5. Maximum Alcohol Consumption	.14	.10	.11	.01	-.04	.02	.16*	.07	.08
6. Alcohol Consumption	.16*	.03	.16*	.07	-.03	-.02	.10	.07	.12
7. Hassles Stress Factor A	.16*	.03	.10	.00	-.21**	-.24***	.12	.33***	.27***
8. Hassles Stress Factor B	.10	-.06	.02	-.05	-.22**	-.27***	.08	.24***	.19**
9. Life Events Stress	.13	.11	.09	-.03	-.17*	-.19**	.21**	.15*	.14
10. Child Temperament Factor A	1.00	.53***	.48***	.07	-.09	.01	.19**	.23**	.22**
11. Child Temperament Factor B		1.00	.47***	-.02	-.07	-.08	.17*	.10	.13
12. Child Temperament Factor C			1.00	.08	-.04	.02	.20**	.17*	.24***
13. Family Expressiveness				1.00	.18*	.35***	-.08	-.14*	-.07
14. Family Conflict					1.00	.43***	-.29***	-.40***	-.38***
15. Family Cohesion						1.00	-.20**	-.29***	-.20**
16. Child Delinquency							1.00	.57***	.53***
17. Child Aggression Factor A								1.00	.81***
18. Child Aggression Factor B									1.00
M	2.88	2.93	2.28	15.13	14.51	16.40	1.73	4.53	4.05
SD	1.33	1.11	1.25	1.62	2.23	1.72	1.70	2.73	2.85

*p<.05. **p<.01. ***p<.001.

Table G4

Means, Standard Deviations, and Correlations For All Wave 2 Maternal Indicator Variables for Coupled Mothers Only (n=188)

Indicator Variable	1	2	3	4	5	6	7	8	9
1. Family Occupational Prestige	1.00	.11	.34***	.27***	.06	.13	.19*	.12	.19**
2. Lifetime Alcohol Problems		1.00	.53***	.16*	.45***	.23***	.22**	.13	.17*
3. Lifetime Antisocial Behavior			1.00	.37***	.35***	.35***	.38***	.25***	.29***
4. Current Depression				1.00	.18*	.31***	.53***	.39***	.12
5. Maximum Alcohol Consumption					1.00	.35***	.15*	.10	.11
6. Alcohol Consumption						1.00	.25***	.24***	.25***
7. Hassles Stress Factor A							1.00	.76***	.21**
8. Hassles Stress Factor B								1.00	.19**
9. Life Events Stress									1.00
M	361.78	9.84	9.70	3.19	2.11	518.56	14.40	12.52	0.84
SD	146.03	1.96	6.48	3.58	1.63	2456.85	11.60	10.93	1.10

*p<.05. **p<.01. ***p<.001.

(table continues)

Table G4 (cont'd)

Indicator Variable	10	11	12	13	14	15	16	17	18
1. Family Occupational Prestige	.10	.10	.01	-.09	-.15*	-.14	.24***	.12	.15*
2. Lifetime Alcohol Problems	-.04	-.02	.01	.01	-.12	-.19**	.11	.12	.18*
3. Lifetime Antisocial Behavior	.09	.08	.06	-.01	-.20**	-.23***	.23***	.29***	.33***
4. Current Depression	.07	.03	.05	-.14	-.36***	-.29***	.19**	.24***	.31***
5. Maximum Alcohol Consumption	.11	.08	.14	.05	-.11	-.09	.24***	.22**	.21**
6. Alcohol Consumption	.01	.07	.08	-.14*	-.26***	-.29***	.14	.24***	.24***
7. Hassles Stress Factor A	.11	.09	.06	-.10	-.33***	-.32***	.13	.23***	.28***
8. Hassles Stress Factor B	.14*	.06	.03	-.04	-.29***	-.27***	.11	.23***	.23**
9. Life Events Stress	.02	-.00	.05	-.10	-.17*	-.14	.14	.11	.15*
10. Child Temperament Factor A	1.00	.56***	.62***	-.00	.06	.08	.11	.19**	.25***
11. Child Temperament Factor B		1.00	.45***	-.06	.01	-.04	.11	.16*	.18*
12. Child Temperament Factor C			1.00	.03	.04	.12	.11	.10	.16*
13. Family Expressiveness				1.00	.07	.21**	.09	-.09	-.03
14. Family Conflict					1.00	.52***	-.27***	-.37***	-.39***
15. Family Cohesion						1.00	-.24***	-.29***	-.29***
16. Child Delinquency							1.00	.56***	.53***
17. Child Aggression A								1.00	.75***
18. Child Aggression B									1.00
<u>M</u>	2.69	2.54	2.15	15.68	14.61	16.47	1.92	4.43	4.13
<u>SD</u>	1.25	1.10	1.21	1.53	2.15	1.78	1.59	2.71	2.91

* $p < .05$. ** $p < .01$. *** $p < .001$.

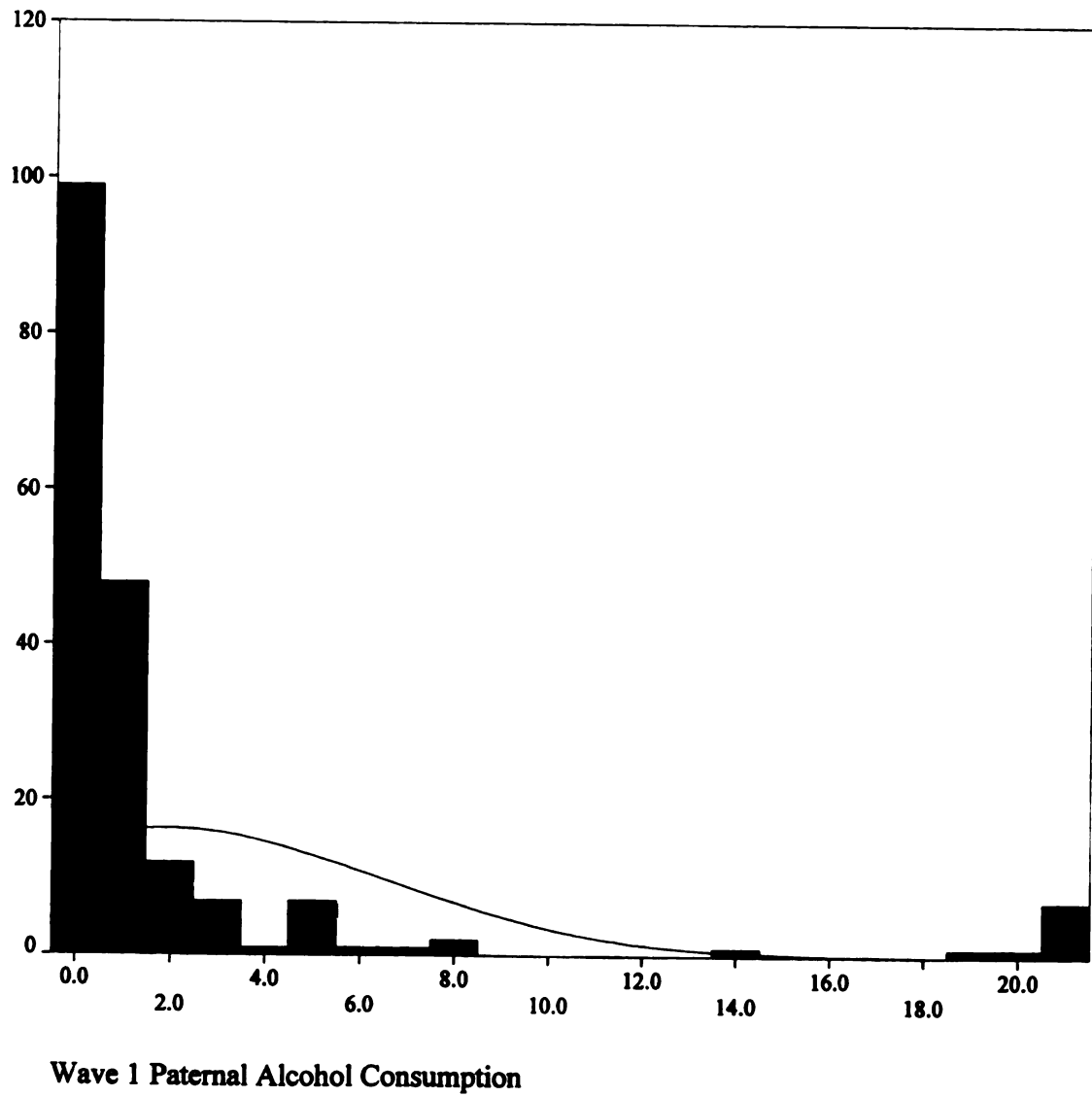
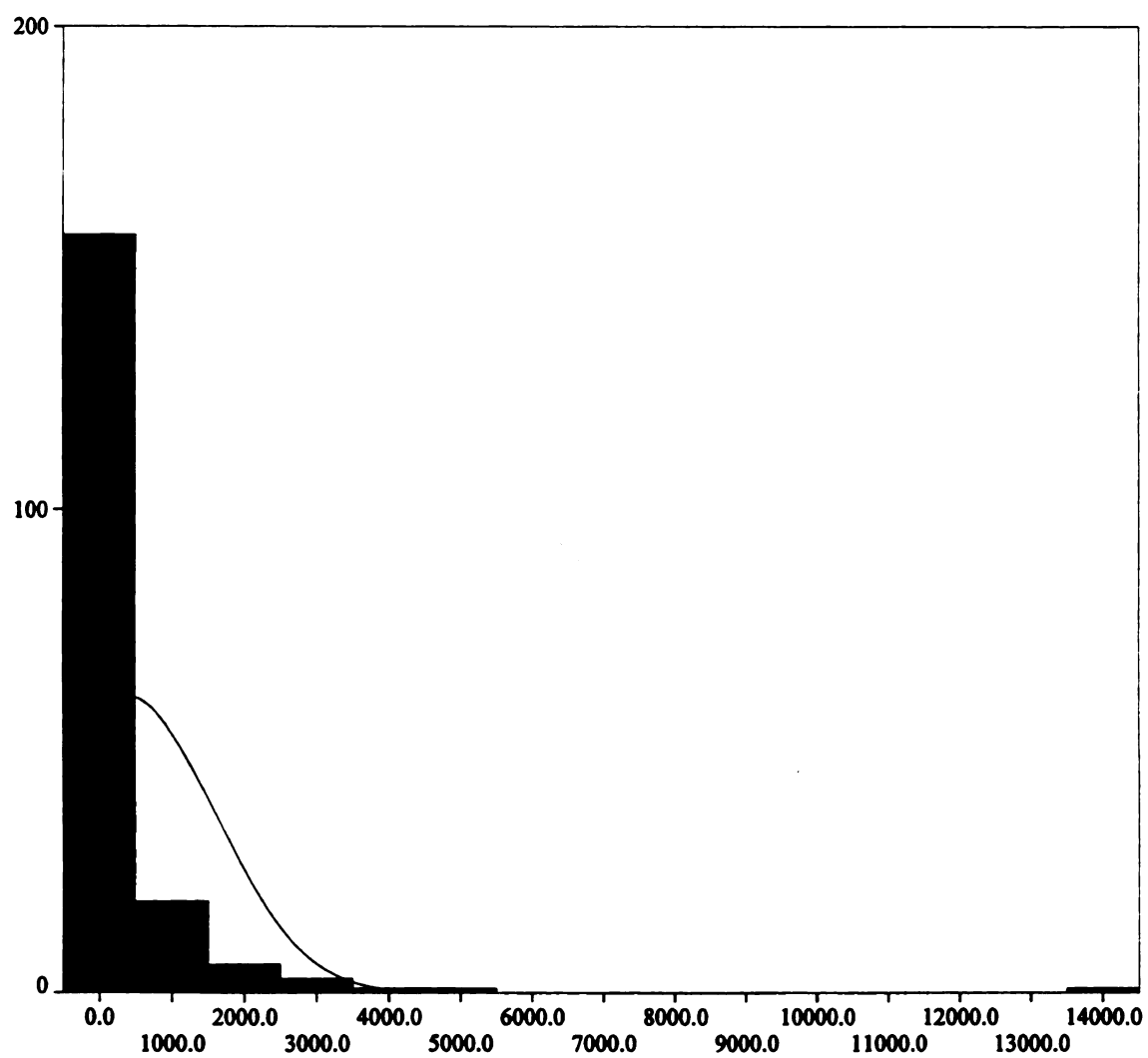


Figure G1. Histogram of Wave 1 paternal alcohol consumption data: Coupled fathers only.



Wave 1 Maternal Alcohol Consumption

Figure G2. Histogram of Wave 1 maternal alcohol consumption data: Coupled mothers only.

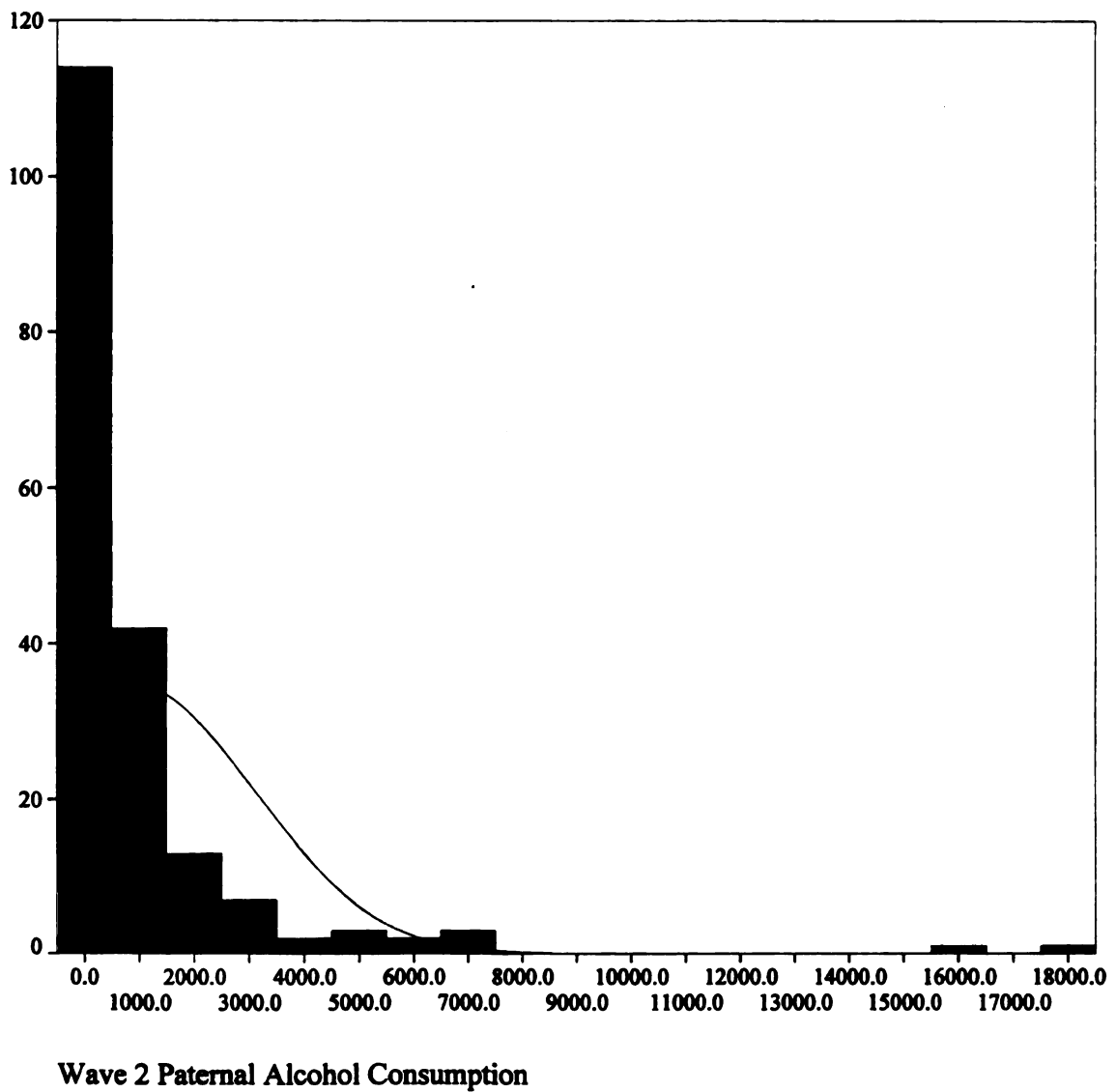


Figure G3. Histogram of Wave 2 paternal alcohol consumption data: Coupled fathers only.

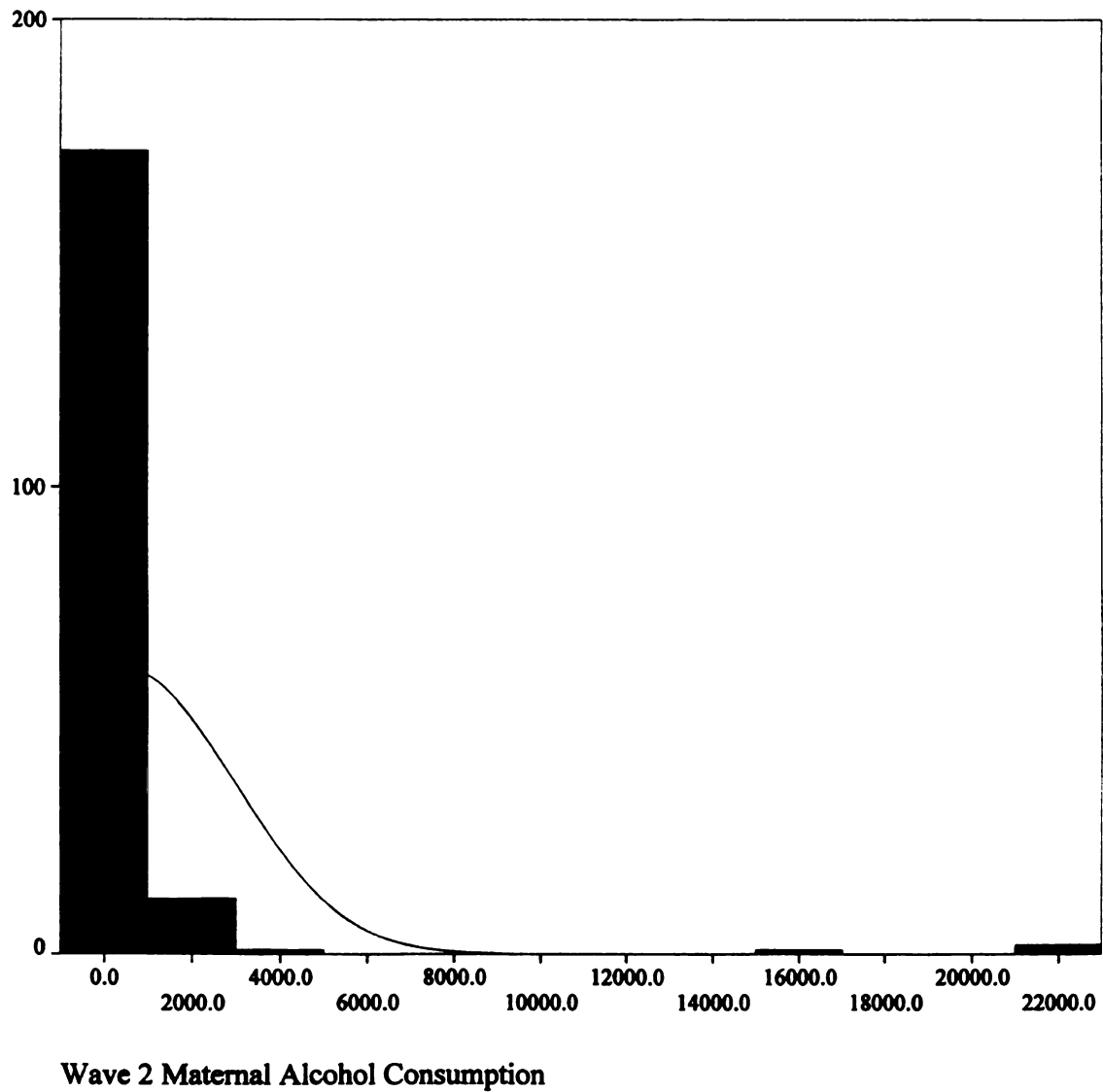
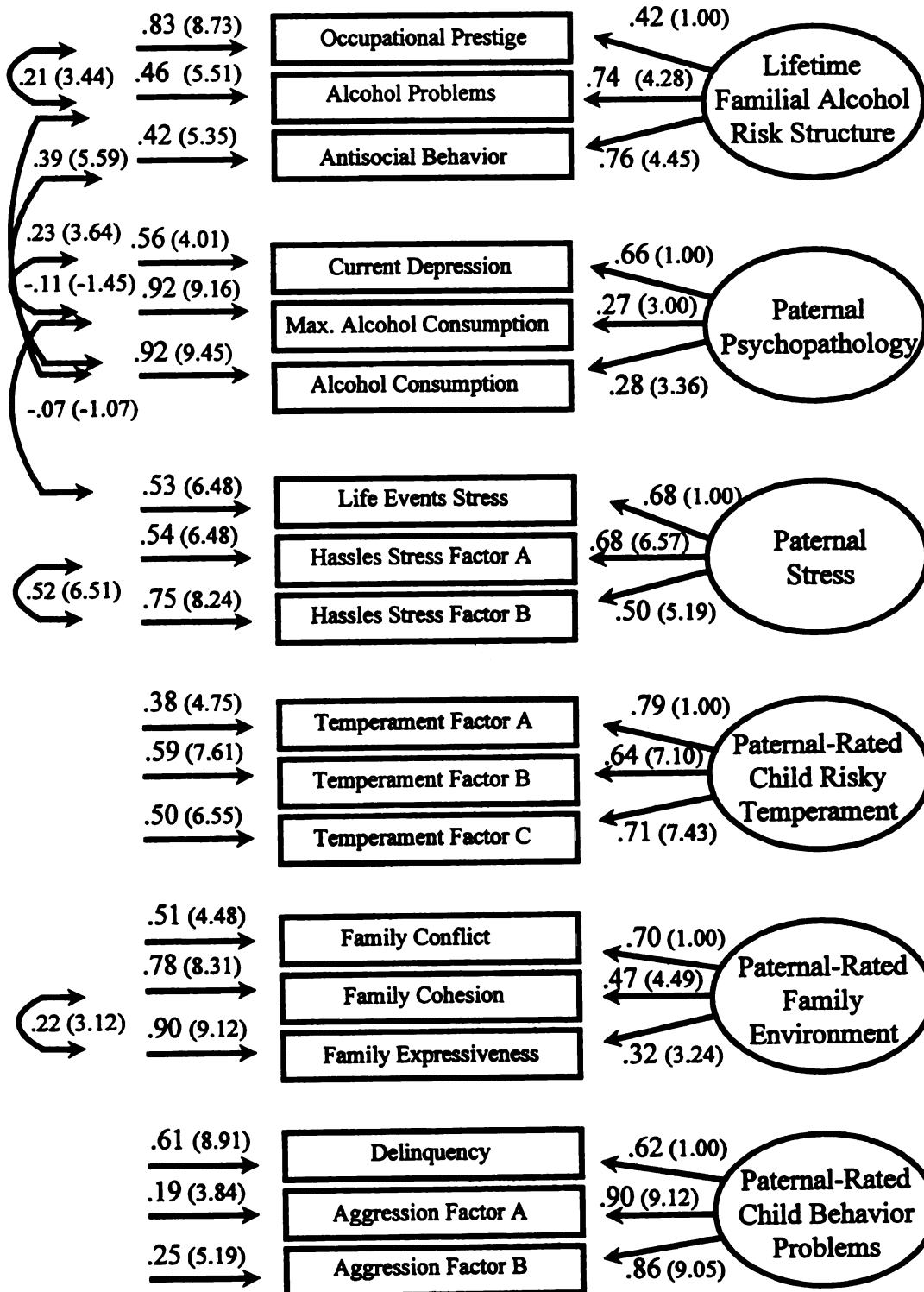


Figure G4. Histogram of Wave 2 maternal alcohol consumption data: Coupled mothers only.



Note. t-values are in parentheses and all latent constructs are correlated.

Figure G5. Measurement model for Wave 1 paternal variables for coupled fathers only.

Table G5

Correlations Among Wave 1 Latent Constructs for Coupled Fathers Only (n=188)

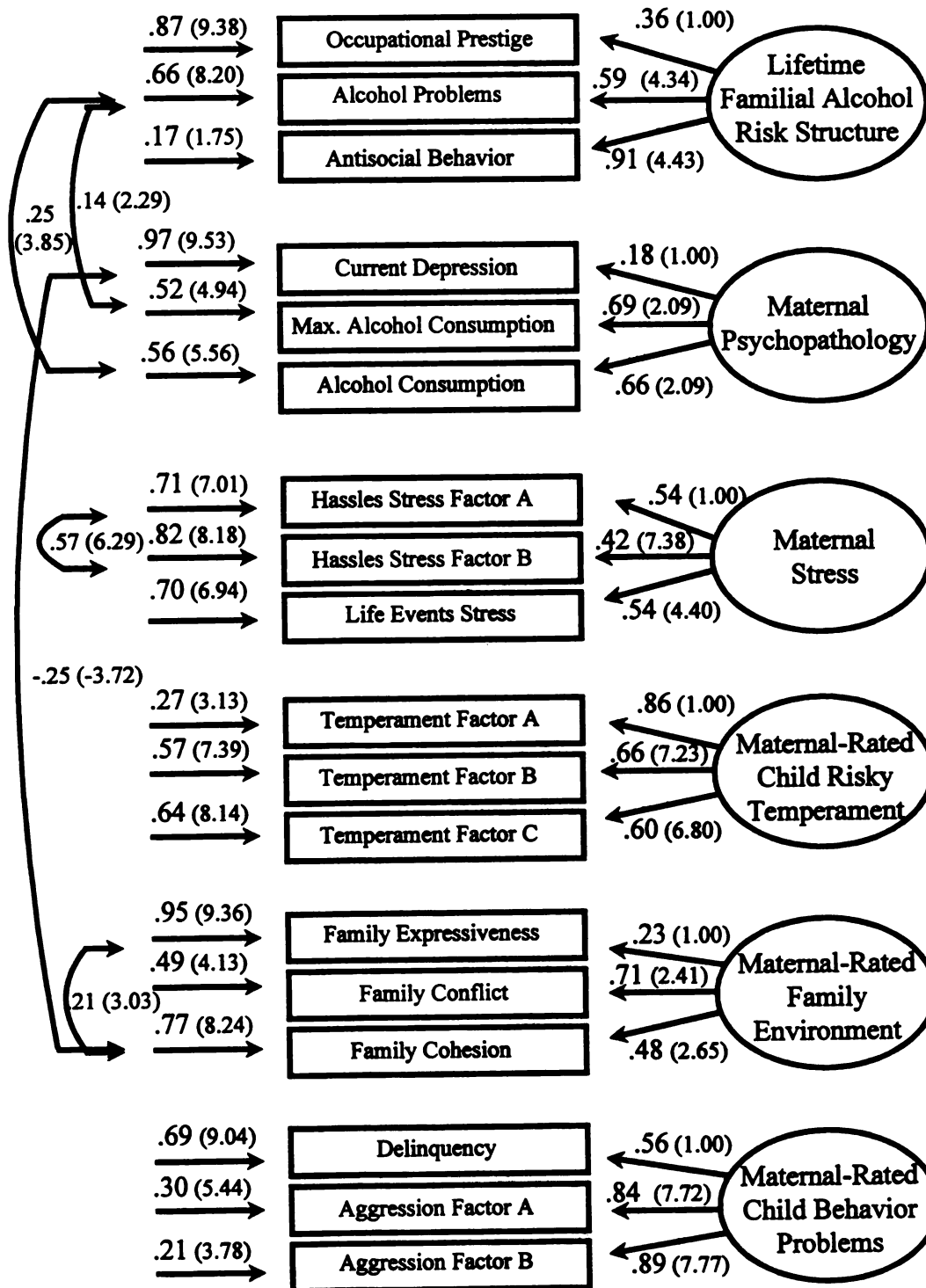
Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.46)	.65 (3.59)	.61 (3.64)	.16 (1.69)	-.52 (-3.41)	.43 (3.31)
2. Psychopathology		1.00 (2.86)	.97 (5.65)	.22 (1.83)	-.68 (-4.44)	.53 (4.13)
3. Stress			1.00 (4.38)	.07 (0.71)	-.45 (-3.43)	.27 (2.59)
4. Child Risky Temperament				1.00 (5.28)	.10 (0.87)	.36 (3.55)
5. Family Environment					1.00 (3.68)	-.54 (-4.28)
6. Child Behavior Problems						1.00 (4.51)

Note. Numbers in parentheses represent *t*-values.

significant Chi-Square [$X^2(115, N = 188) = 149.94, p < .05$], a large GFI (.92) and CFI (.96), and a small SRMR (.072) (see Figure G6 for measurement model and Table G6 for correlations among latent constructs). The Wave 2 paternal model resulted in a significant Chi-Square [$X^2(117, N = 188) = 172.66, p < .001$], a GFI of .91, a CFI of .95, and a SRMR was .072 (see Figure G7 for measurement model and Table G7 for correlations among latent constructs). The final measurement model, Wave 2 maternal model, resulted in a non-significant Chi-Square [$X^2(118, N = 188) = 126.94$], a GFI of .93, a CFI of .99, and a SRMR was .054 (see Figure G8 for measurement model and Table G8 for correlations among latent constructs). These analyses were followed by the estimation of four structural models, one for each parent at each time period.

Wave 1 Paternal Structural Model for Coupled Fathers Only

Fit indices for the Wave 1 paternal structural model demonstrated the model fit the data well; although the Chi-Square was significant [$X^2(118, N = 188) = 179.81, p < .001$], the GFI was .91, the CFI was .95, and the SRMR was .076 (see Figure G9 for structural model). Results from this model demonstrated that a more dense lifetime familial alcohol risk structure predicted higher levels of paternal psychopathology and a worse quality family environment, but not higher levels of paternal stress or child risky temperament. Paternal psychopathology predicted both increased levels of paternal stress and a poorer quality family environment, however, child risky temperament did not predict quality of the family environment. Finally, child externalizing behavior problems was predicted only by a poorer quality family environment and child risky temperament but not by higher levels of paternal stress



Note. t-values are in parentheses and all latent constructs are correlated.

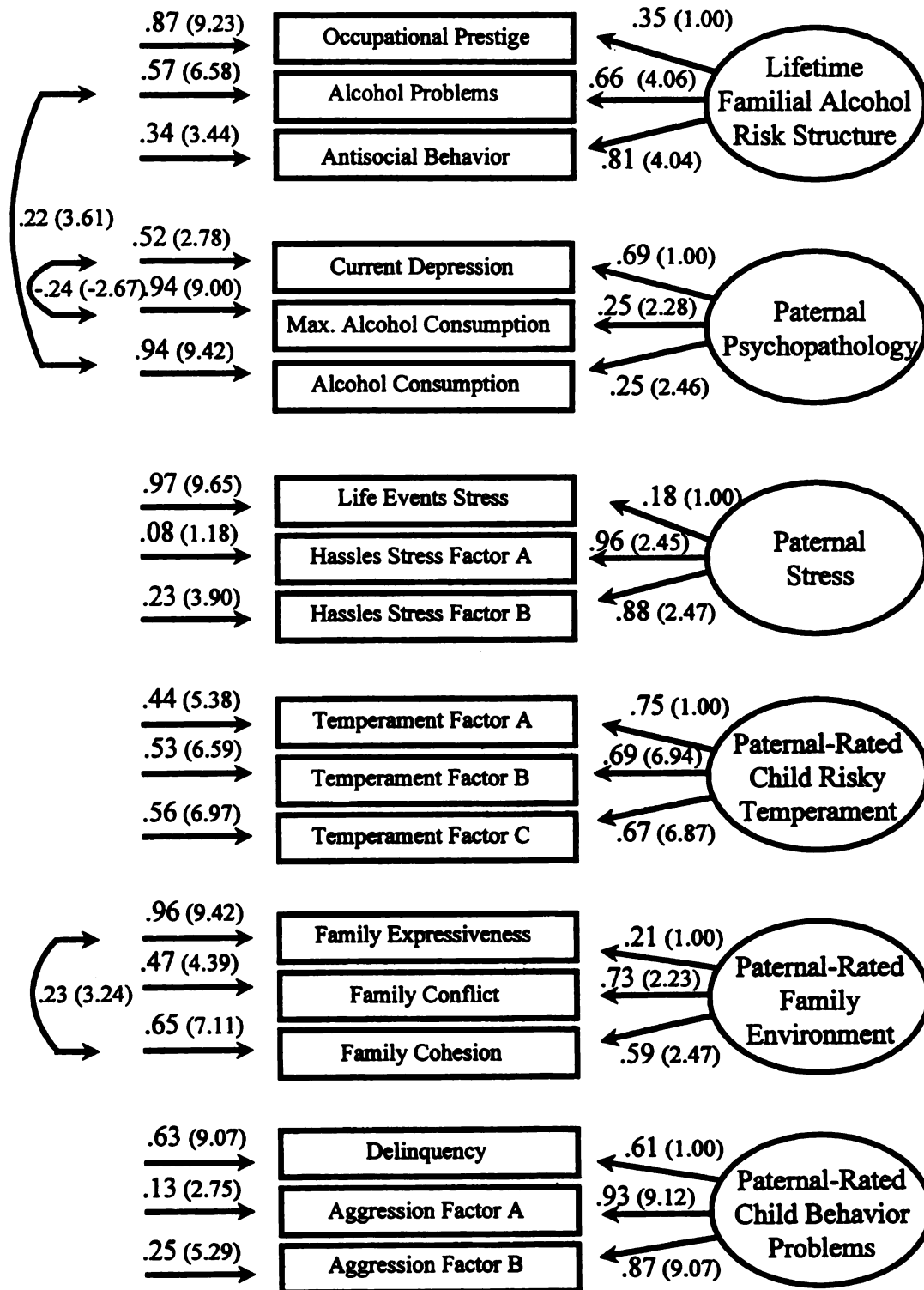
Figure G6. Measurement model for Wave 1 maternal variables for coupled mothers only.

Table G6

Correlations Among Wave 1 Latent Constructs for Coupled Mothers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.37)	.57 (1.84)	.68 (3.24)	.08 (0.86)	-.48 (-2.00)	.54 (3.34)
2. Psychopathology		1.00 (1.08)	.34 (1.61)	-.02 (-0.20)	-.39 (-1.44)	.24 (1.59)
3. Stress			1.00 (2.90)	.12 (0.99)	-.53 (-2.03)	.57 (3.72)
4. Child Risky Temperament				1.00 (5.74)	.36 (2.01)	.30 (3.12)
5. Family Environment					1.00 (1.28)	-.37 (-2.02)
6. Child Behavior Problems						1.00 (3.92)

Note. Numbers in parentheses represent *t*-values.



Note. t-values are in parentheses and all latent constructs are correlated.

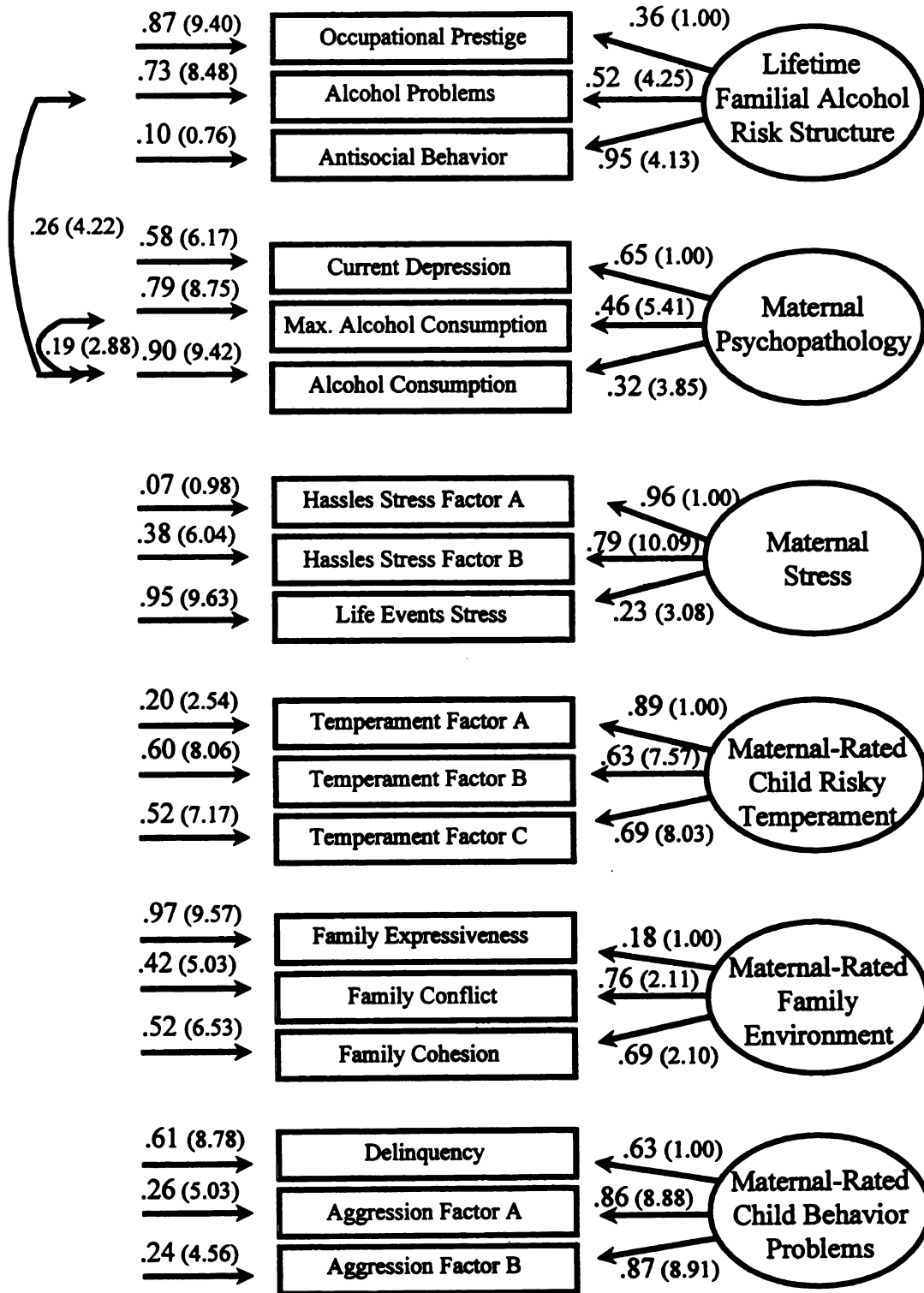
Figure G7. Measurement model for Wave 2 paternal variables for coupled fathers only.

Table G7

Correlations Among Wave 2 Latent Constructs for Coupled Fathers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.18)	.55 (3.16)	.34 (1.87)	.28 (2.36)	-.46 (-1.86)	.38 (2.88)
2. Psychopathology		1.00 (2.40)	.64 (2.25)	.32 (2.77)	-.50 (-2.00)	.41 (3.57)
3. Stress			1.00 (1.23)	.13 (1.27)	-.36 (-1.54)	.33 (2.06)
4. Child Risky Temperament				1.00 (4.86)	-.10 (-0.90)	.29 (2.97)
5. Family Environment					1.00 (1.16)	-.56 (-2.10)
6. Child Behavior Problems						1.00 (4.44)

Note. Numbers in parentheses represent *t*-values.



Note. t-values are in parentheses and all latent constructs are correlated.

Figure G8. Measurement model for Wave 2 maternal variables for coupled mothers only.

Table G8

Correlations Among Wave 2 Latent Constructs for Coupled Mothers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (2.31)	.68 (3.38)	.42 (3.21)	.09 (1.05)	-.32 (-1.67)	.38 (2.93)
2. Psychopathology		1.00 (3.82)	.75 (5.95)	.14 (1.23)	-.70 (-1.99)	.54 (4.14)
3. Stress			1.00 (7.31)	.13 (1.57)	-.47 (-1.97)	.30 (3.39)
4. Child Risky Temperament				1.00 (6.22)	.09 (0.87)	.27 (2.98)
5. Family Environment					1.00 (1.07)	-.54 (-1.97)
6. Child Behavior Problems						1.00 (4.49)

Note. Numbers in parentheses represent *t*-values.

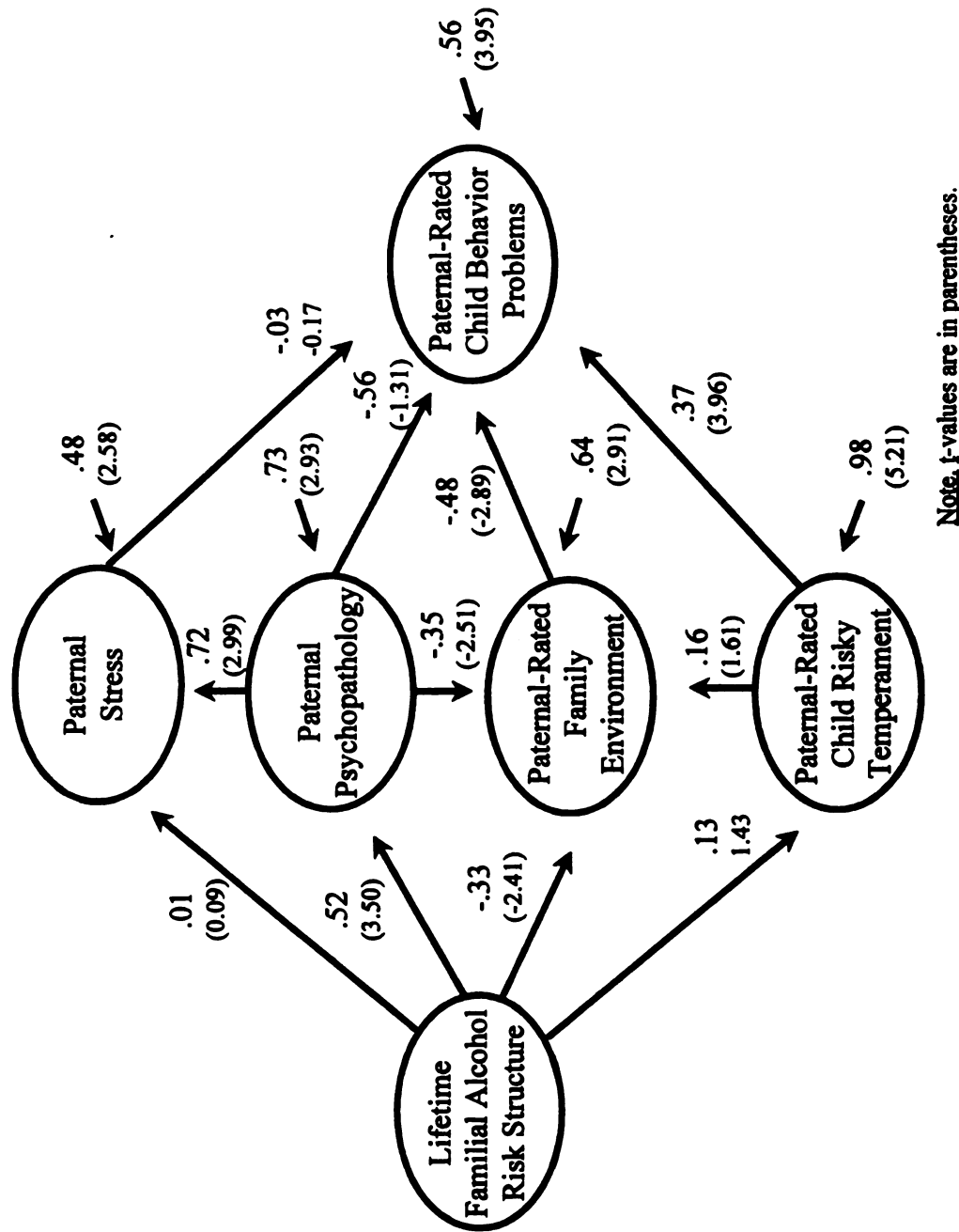


Figure G9. Structural model representing relationships among all Wave 1 paternal latent variables for coupled fathers only.

or paternal psychopathology.

Wave 1 Maternal Structural Model for Coupled Mothers Only

Considered together, the fit indices of the Wave 1 maternal structural model indicated the model fit was adequate; although the Chi-Square was significant [$X^2(122, N = 188) = 150.55, p < .001$], the GFI was .91, the CFI was .99, and the SRMR was .12 (see Figure G10 for structural model). This model revealed that a more dense lifetime familial alcohol risk structure directly predicted higher levels of maternal stress and psychopathology but not a worse quality family environment or more child risky temperament. Increased levels of maternal psychopathology only predicted a worse quality family environment (and not maternal stress) and child risky temperament predicted a better quality family environment. Finally, high levels of maternal stress and child risky temperament both predicted more child externalizing behavior problems. Unexpectedly, neither maternal psychopathology nor quality of the family environment predicted child externalizing behavior problems.

Wave 2 Paternal Structural Model for Coupled Fathers Only

The Wave 2 paternal structural model fit the data well; although the Chi-Square was significant [$X^2(121, N = 188) = 175.68, p < .001$], the GFI and CFI were .91 and .95 respectively, and the SRMR was .073 (see Figure G11 for structural model). Results showed that a more dense lifetime familial alcohol risk structure directly predicted higher levels of paternal psychopathology and child risky temperament but not higher levels of paternal stress or a worse quality family environment. As expected, increased levels of paternal psychopathology predicted

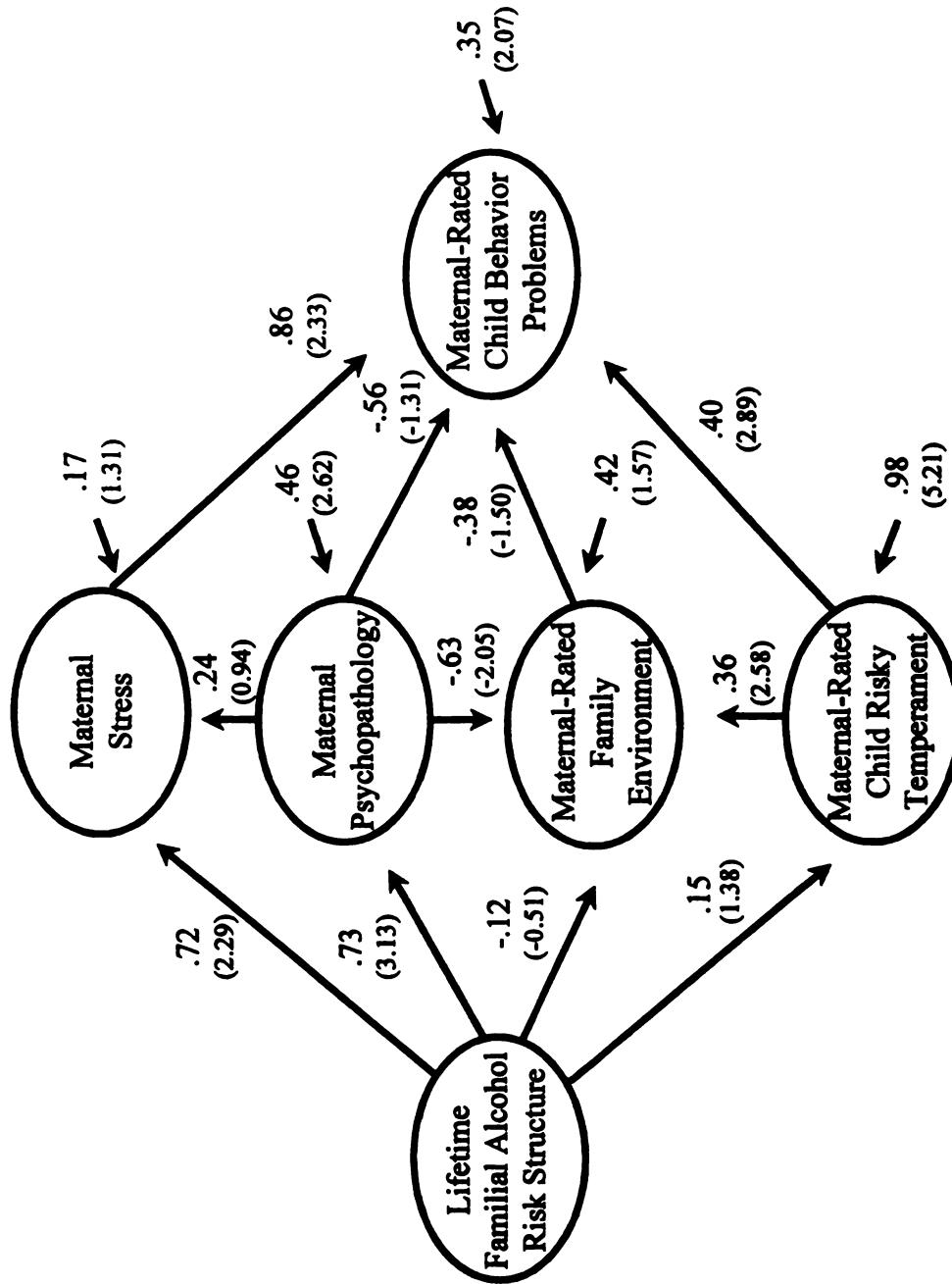
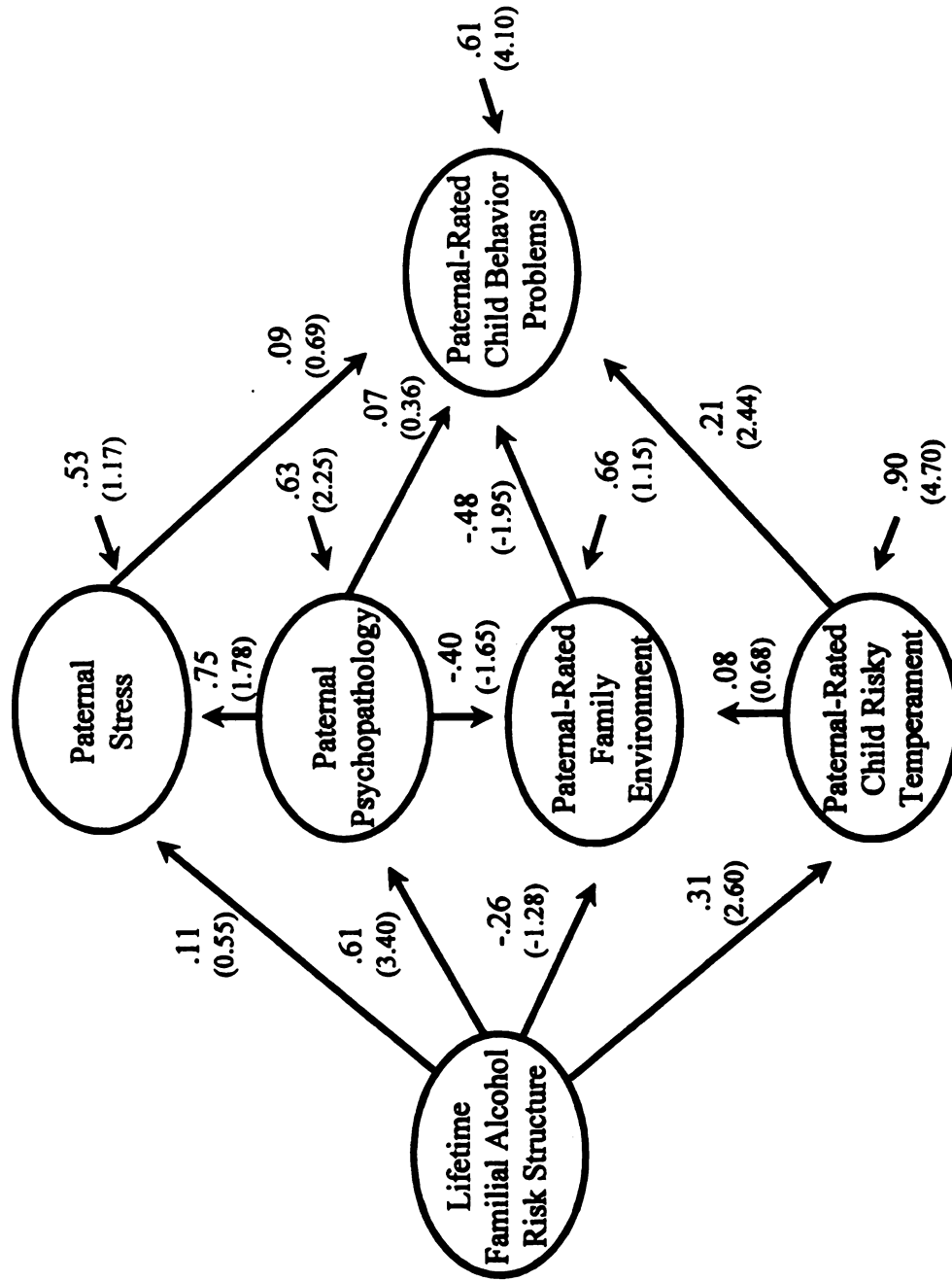


Figure G10. Structural model representing relationships among all Wave 1 maternal latent variables for coupled mothers only.



Note. t-values are in parentheses.

Figure G11. Structural model representing relationships among all Wave 2 paternal latent variables for coupled fathers only.

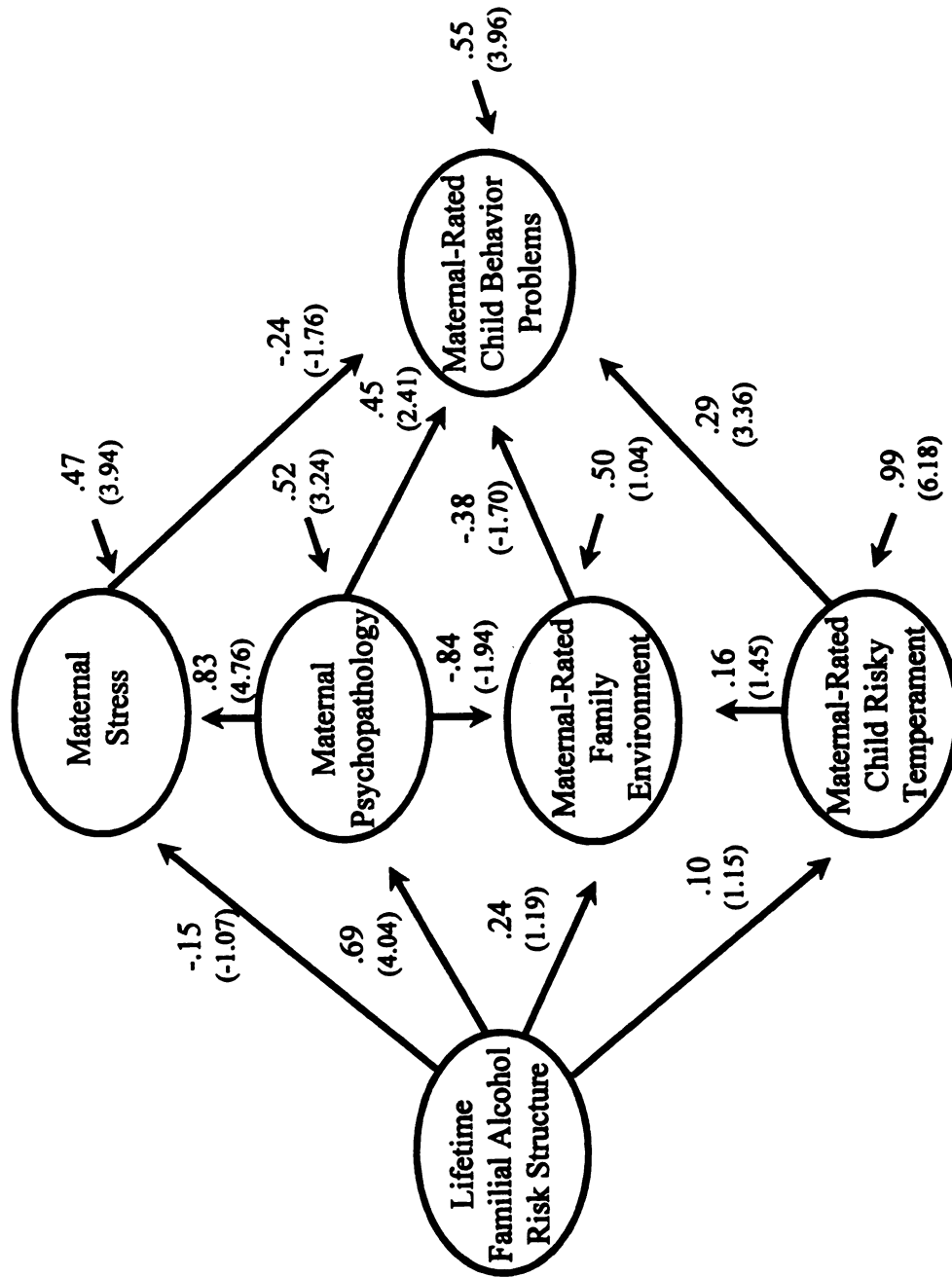
more paternal stress and a poorer quality family environment, however, child risky temperament did not predict a poorer quality family environment. Lastly, only a poorer quality family environment and child risky temperament directly predicted higher levels of child externalizing behavior problems (paternal stress and psychopathology did not).

Wave 2 Maternal Structural Model for Coupled Mothers Only

The fit of the Wave 2 maternal structural model was very good. The Chi-Square was non-significant [$\chi^2(122, N = 188) = 129.61, p < .001$], the GFI and CFI were .93 and .99 respectively and the SRMR was .056 (see Figure G12 for structural model). Results indicated that a more dense lifetime familial alcohol risk structure only predicted higher levels of maternal psychopathology. Contrary to what was hypothesized, a more dense lifetime familial alcohol risk structure did not predict higher levels of family stress, child risky temperament or a worse quality family environment. Higher levels of maternal psychopathology predicted more maternal stress and a poorer quality family environment. Child risky temperament did not predict quality of the family environment. Finally, child externalizing behavior problems was predicted by higher levels of maternal stress and psychopathology in addition to a worse quality family environment and child risky temperament.

Two-Group Stacked Models and Coupled Parents Only

Following the estimation of the cross-sectional models, two (Wave 1 and Wave 2) two-group (maternal data, paternal data) stacked measurement models were estimated. The Wave 1 stacked measurement model fit the data well when; (1) the loadings of the indicator variables on the latent constructs were kept invariant across

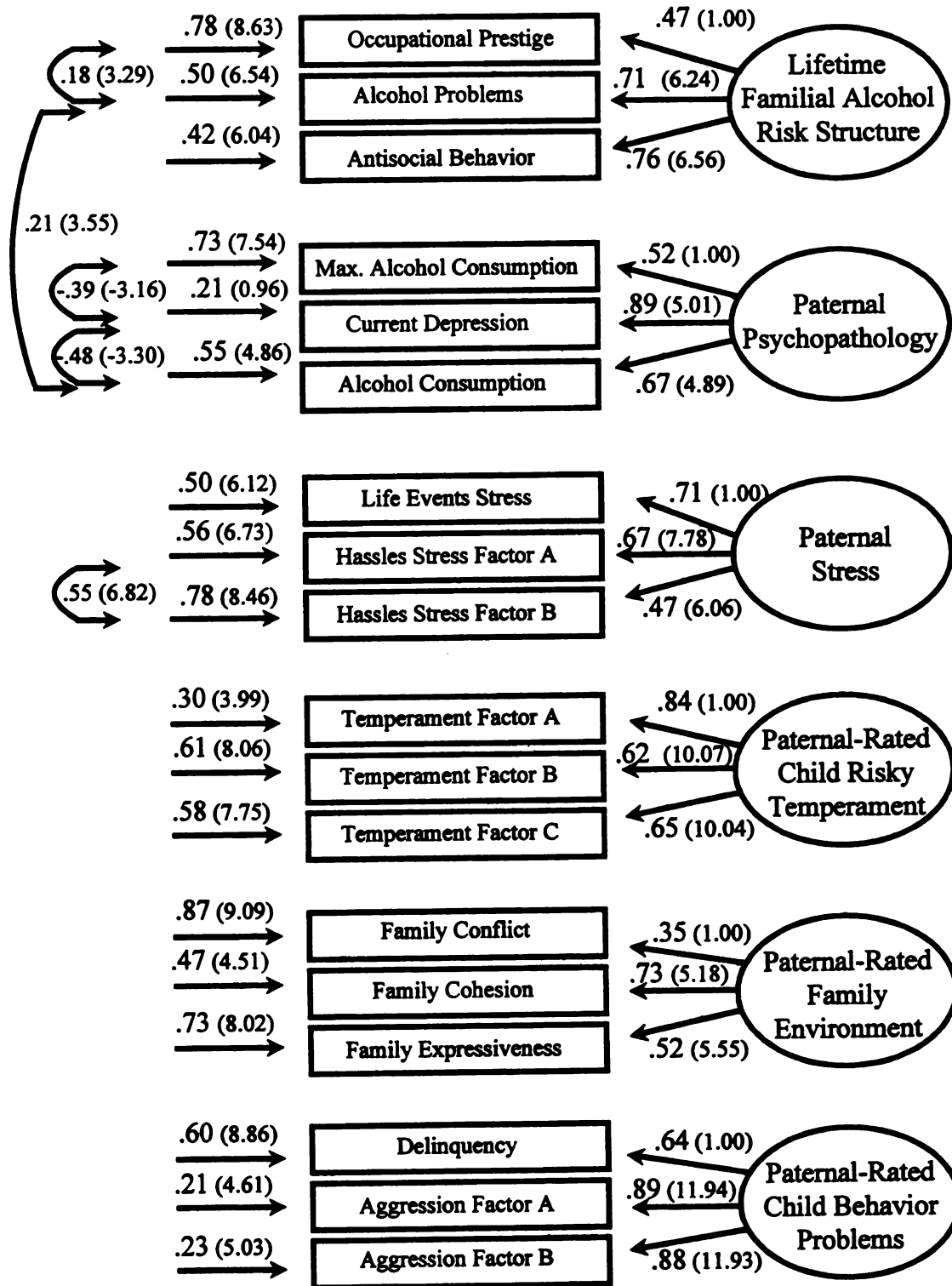


Note. t-values are in parentheses.

Figure G12. Structural model representing relationships among all Wave 2 maternal latent variables for coupled mothers only.

the two groups, (2) the pattern of correlations among the latent constructs was invariant across the two groups but the starting values were free to vary, and (3) the uniquenesses were free to vary across the two groups. This model resulted in a significant Chi-Square [$X^2(242, N = 376) = 354.60, p < .001$], the GFI was .90, the CFI was .95, and the SRMR was .079 (see G13 for paternal measurement model and Table G9 for correlations among latent constructs and Figure G14 for maternal model and Table G10 for correlations among latent constructs). The Wave 2 stacked measurement model also fit the model well when; (1) the loadings of the indicator variables on the latent constructs were kept invariant across the two groups, (2) the pattern of correlations among the latent constructs and the start values were invariant across the two groups, and (3) the uniquenesses were free to vary across the two groups. The Wave 2 stacked measurement model resulted in a significant Chi-Square [$X^2(265, N = 376) = 314.05, p < .001$], a GFI of .91, a CFI of .98, and a SRMR of .078 (see Figure G15 and Table G11 and Figure G16 and Table G12).

The two-group stacked structural models were analyzed next. The models fit the data well when the following restrictions were placed on the model parameters; (1) the loadings of the indicator variables on the latent constructs were kept invariant across the two groups, (2) the pattern of predictive associations among the latent constructs was completely invariant across the two groups, (3) the pattern of correlations among the latent constructs was invariant across the two groups although the starting values were free to vary, and (4) the uniquenesses were free to vary across the two groups.



Note. t-values are in parentheses and all latent constructs are correlated.

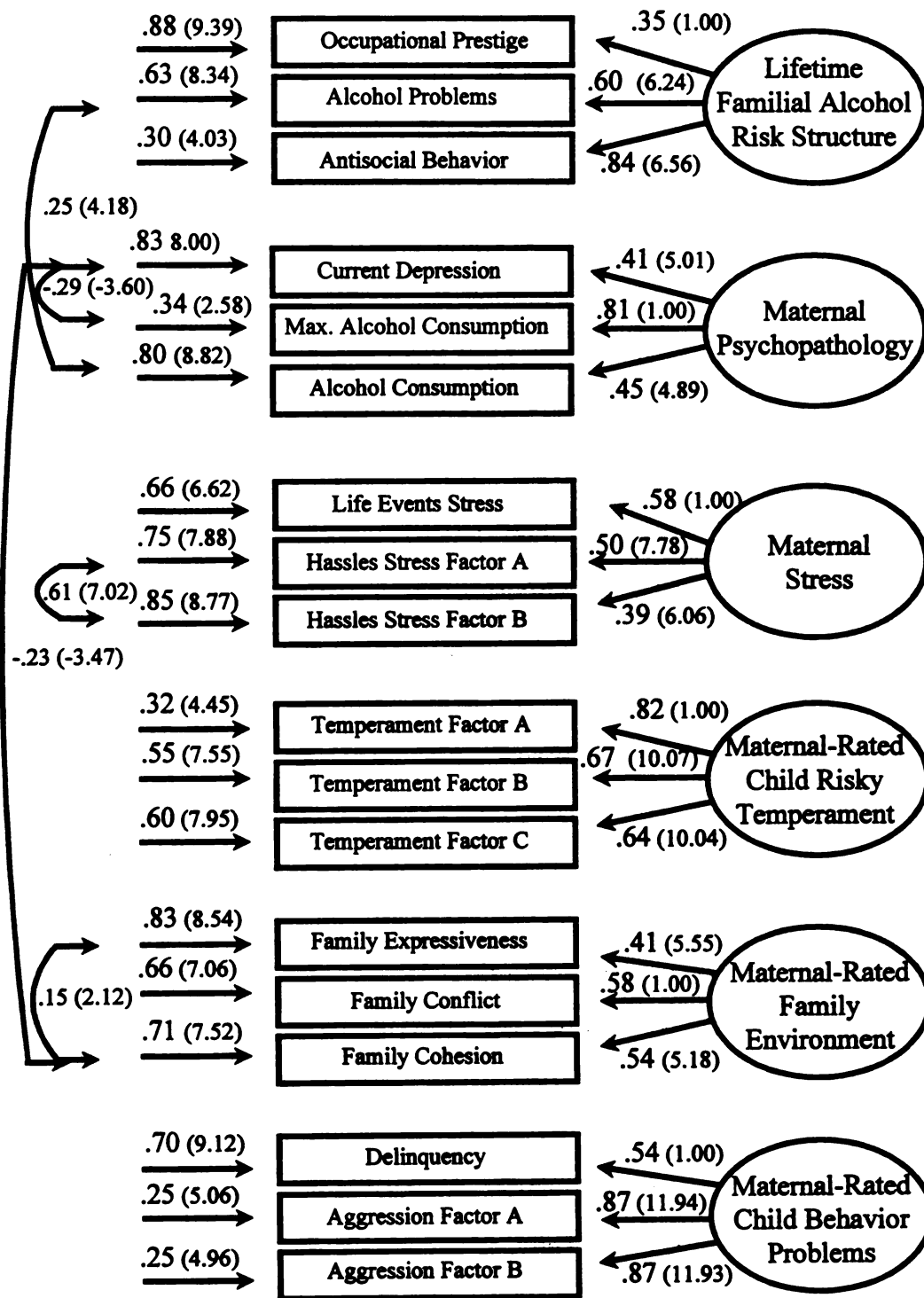
Figure G13. Measurement model for Wave 1 two-group stacked analysis: Solution for paternal data for coupled fathers only.

Table G9

Correlations Among Wave 1 Latent Constructs for Stacked Models - CoupledFathers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.46)	.52 (3.82)	.62 (4.36)	.19 (1.95)	-.34 (-2.65)	.44 (3.88)
2. Psychopathology		1.00 (3.25)	.58 (4.25)	.15 (1.93)	-.36 (-3.00)	.34 (3.55)
3. Stress			1.00 (4.82)	.07 (0.68)	-.40 (-2.93)	.25 (2.51)
4. Child Risky Temperament				1.00 (6.30)	.19 (1.76)	.35 (3.66)
5. Family Environment					1.00 (2.84)	-.32 (-2.76)
6. Child Behavior Problems						1.00 (5.42)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

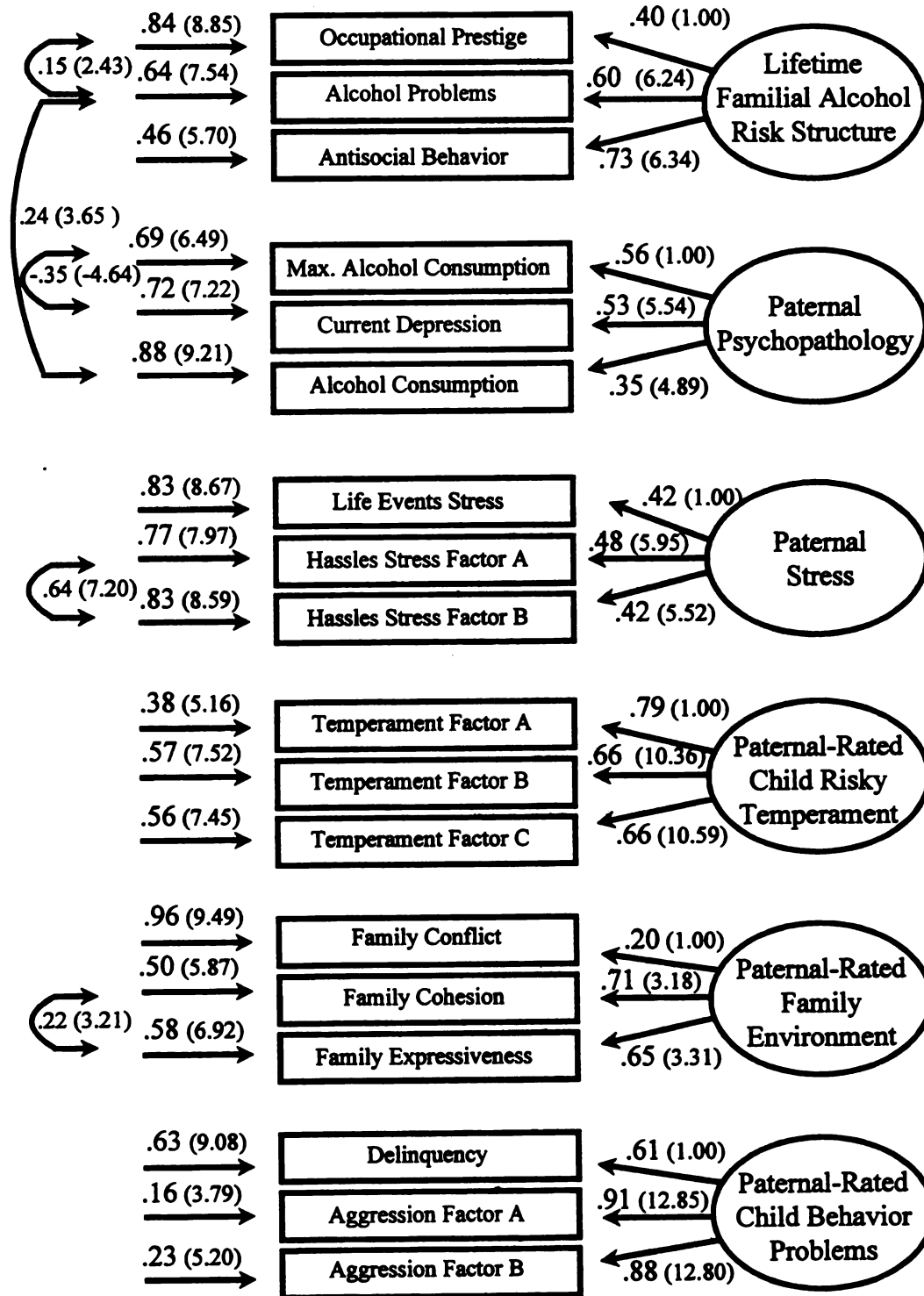
Figure G14. Measurement model for Wave 1 two-group stacked analysis: Solution for maternal data for coupled mothers only.

Table G10

Correlations Among Wave 1 Latent Constructs for Stacked Models - CoupledMothers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.20)	.60 (3.65)	.74 (4.20)	.06 (0.67)	-.57 (-3.35)	.56 (4.20)
2. Psychopathology		1.00 (2.75)	.51 (3.36)	-.04 (-0.46)	-.49 (-3.04)	.30 (2.90)
3. Stress			1.00 (3.53)	.11 (0.88)	-.58 (-3.22)	.56 (4.25)
4. Child Risky Temperament				1.00 (6.22)	.37 (2.80)	.31 (3.27)
5. Family Environment					1.00 (2.71)	-.37 (-2.90)
6. Child Behavior Problems						1.00 (5.28)

Note. Numbers in parentheses represent t-values.



Note. t-values are in parentheses and all latent constructs are correlated.

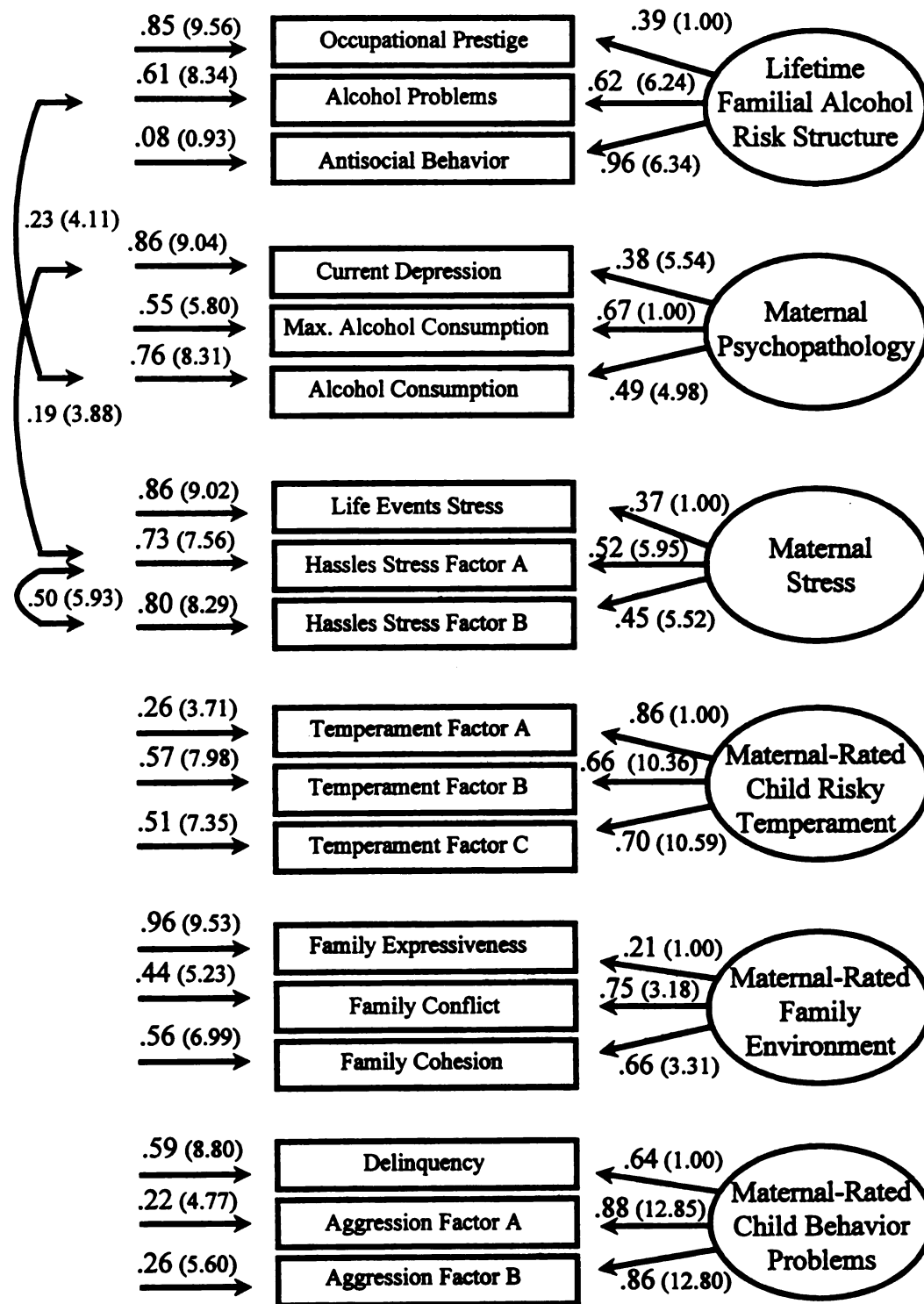
Figure G15. Measurement model for Wave 2 two-group stacked analysis: Solution for paternal data for coupled fathers only.

Table G11

Correlations Among Wave 2 Latent Constructs for Stacked Models - CoupledFathers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.47)	.59 (4.40)	.78 (4.44)	.18 (2.55)	-.38 (-2.59)	.39 (4.36)
2. Psychopathology		1.00 (3.47)	.85 (4.69)	.24 (2.96)	-.47 (-2.71)	.42 (4.54)
3. Stress			1.00 (3.00)	.26 (2.55)	-.71 (-2.79)	.53 (4.46)
4. Child Risky Temperament				1.00 (7.88)	.01 (0.09)	.28 (4.23)
5. Family Environment					1.00 (1.64)	-.55 (-2.97)
6. Child Behavior Problems						1.00 (6.37)

Note. Numbers in parentheses represent *t*-values.



Note. t-values are in parentheses and all latent constructs are correlated.

Figure G16. Measurement model for Wave 2 two-group stacked analysis: Solution for maternal data for coupled mothers only.

Table G12

Correlations Among Wave 2 Latent Constructs for Stacked Models - CoupledMothers Only (n=188)

Latent Constructs	1	2	3	4	5	6
1. Familial Alcohol Risk Structure	1.00 (3.47)	.59 (4.40)	.78 (4.44)	.18 (2.55)	-.38 (-2.59)	.39 (4.36)
2. Psychopathology		1.00 (3.47)	.85 (4.69)	.24 (2.96)	-.47 (-2.71)	.42 (4.54)
3. Stress			1.00 (3.00)	.26 (2.55)	-.71 (-2.79)	.53 (4.46)
4. Child Risky Temperament				1.00 (7.88)	.01 (0.09)	.28 (4.23)
5. Family Environment					1.00 (1.64)	-.55 (-2.97)
6. Child Behavior Problems						1.00 (6.37)

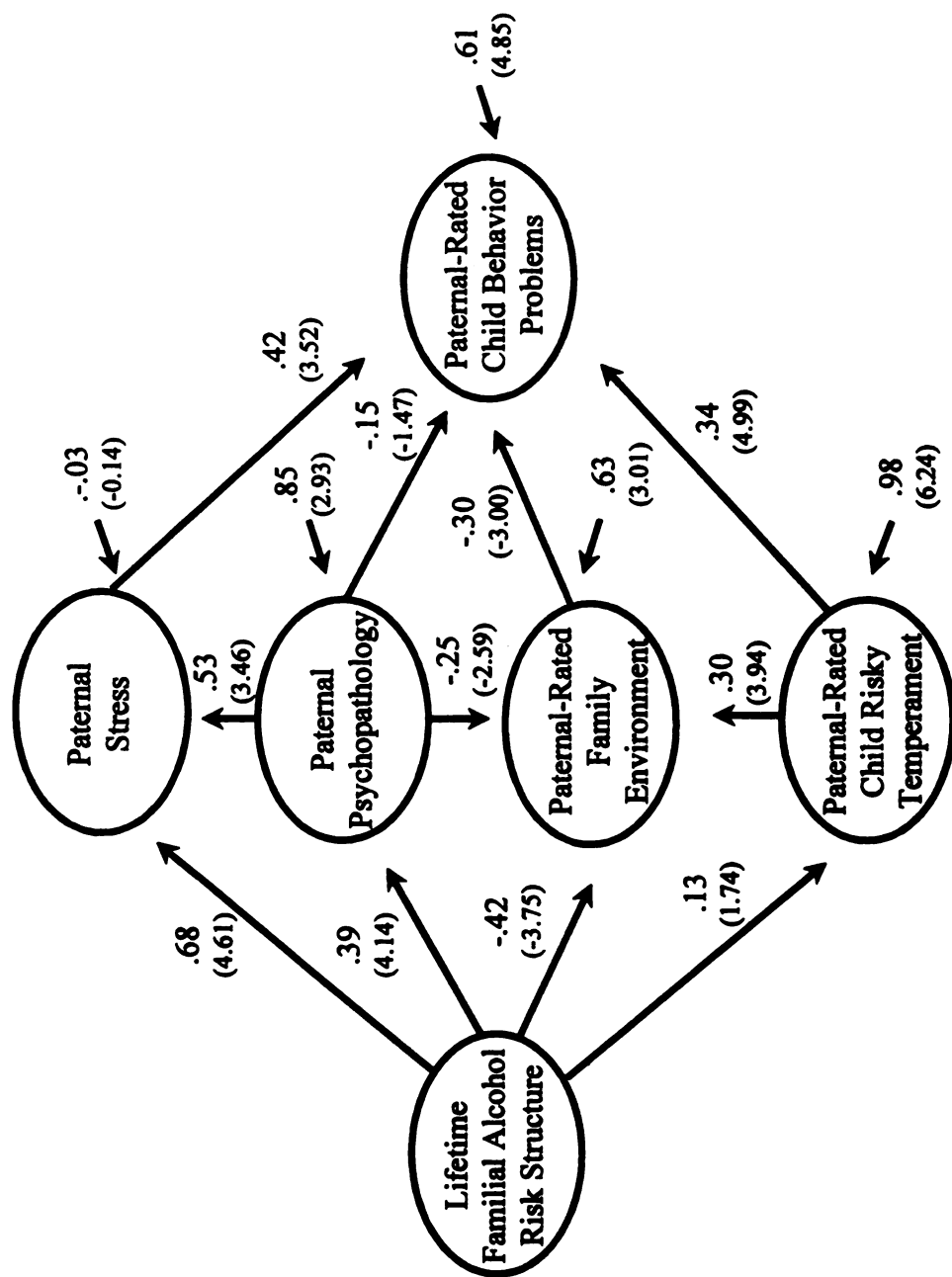
Note. Numbers in parentheses represent *t*-values.

Wave 1 stacked structural model for coupled parents only. This model fit the data; although the Chi-Square was significant [$X^2(259, N = 376) = 364.23, p < .001$], the GFI was .90, the CFI was .95, and the SRMR was .079 (see Figures G17 and G18 for within-group completely standardized solutions). Results for the Wave 1 two-group stacked structural model partially supported the first hypothesis and demonstrated that the maternal and paternal models did not differ in structure.

As was hypothesized, the results revealed that a more dense lifetime familial alcohol risk structure directly predicted higher levels of parental stress, parental psychopathology, a worse quality family environment, and more child risky temperament. Increased levels of parental psychopathology predicted more parental stress and a worse quality family environment and unexpectedly child risky temperament predicted a better quality of the family environment. Lastly, more child externalizing behavior problems was predicted by more parental stress, a poorer quality family environment, and more child risky temperament but not more parental psychopathology.

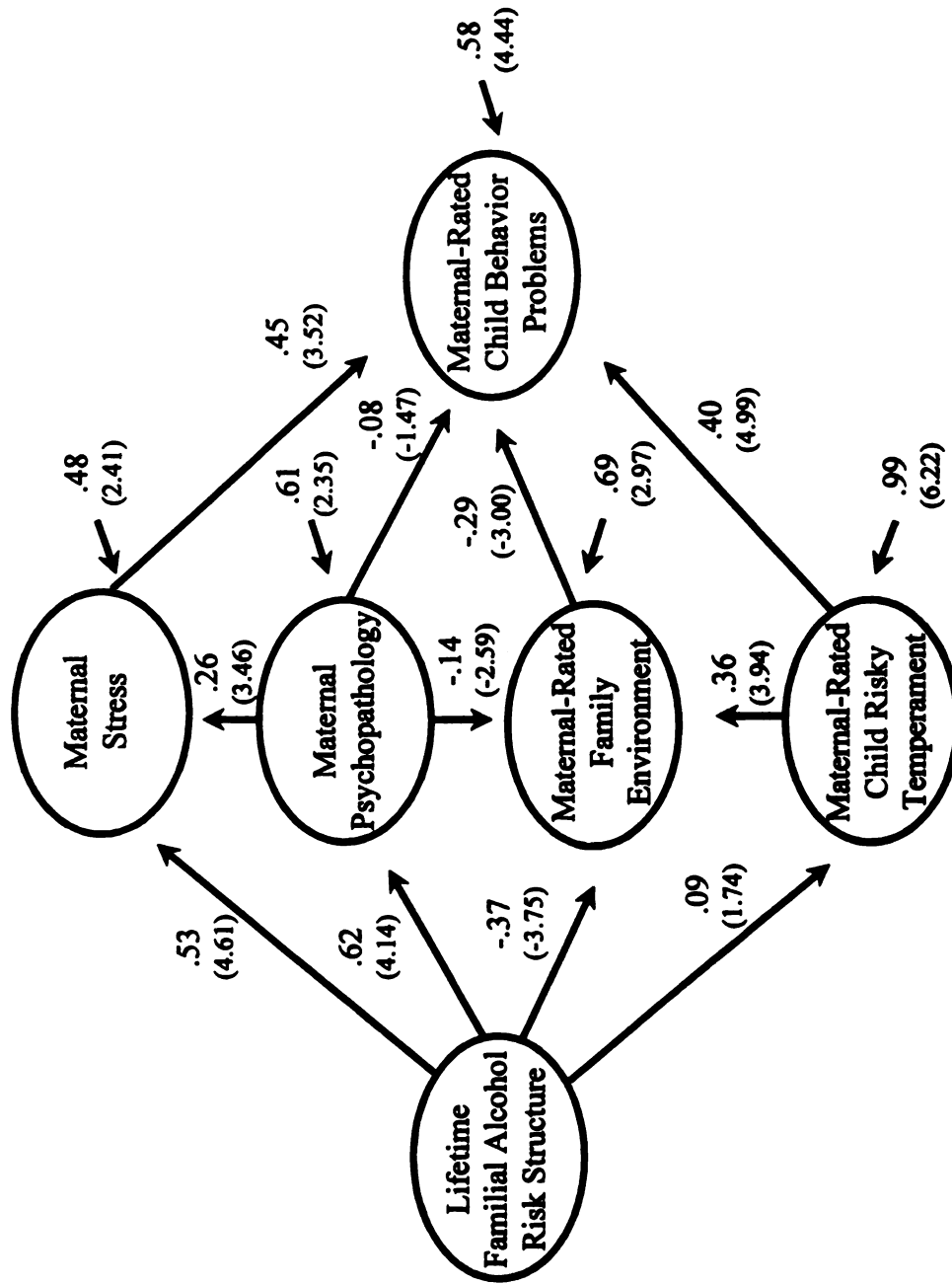
Wave 2 stacked structural model for coupled parents only. This model resulted in a good fit; the Chi-Square was significant [$X^2(263, N = 376) = 341.19, p < .001$], however, the GFI was .90, the CFI was .96, and the SRMR was .082 (see Figures G19 and G20 for within-group completely standardized solutions). Results for the Wave 2 two-group stacked structural model also partially supported the second hypothesis and revealed that the maternal and paternal models did not differ in structure.

As hypothesized, a more dense lifetime familial alcohol risk structure directly



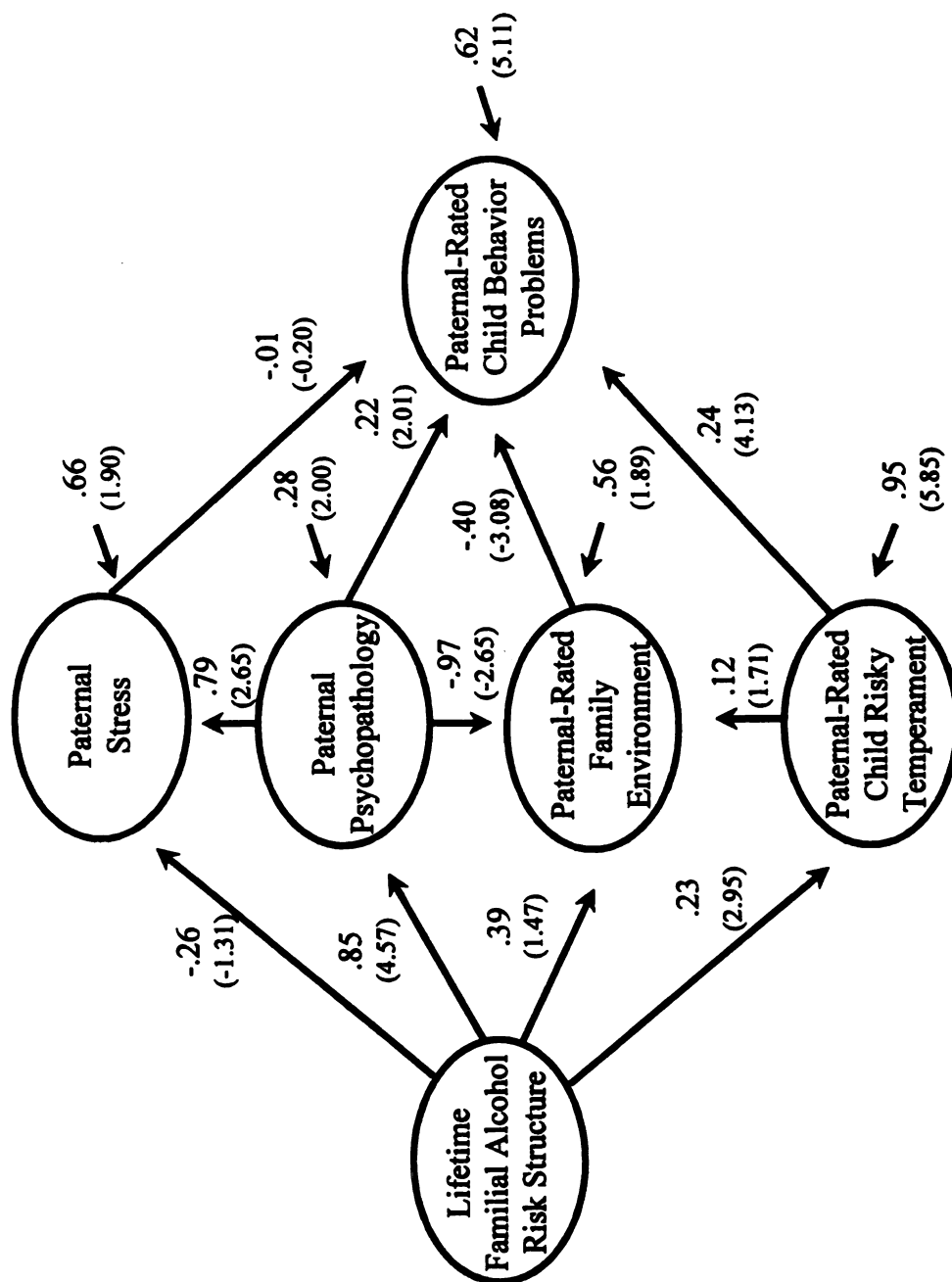
Note. t-values are in parentheses.

Figure G17. Structural model from Wave 1 two-group stacked analysis: Solution for paternal data for coupled fathers only.



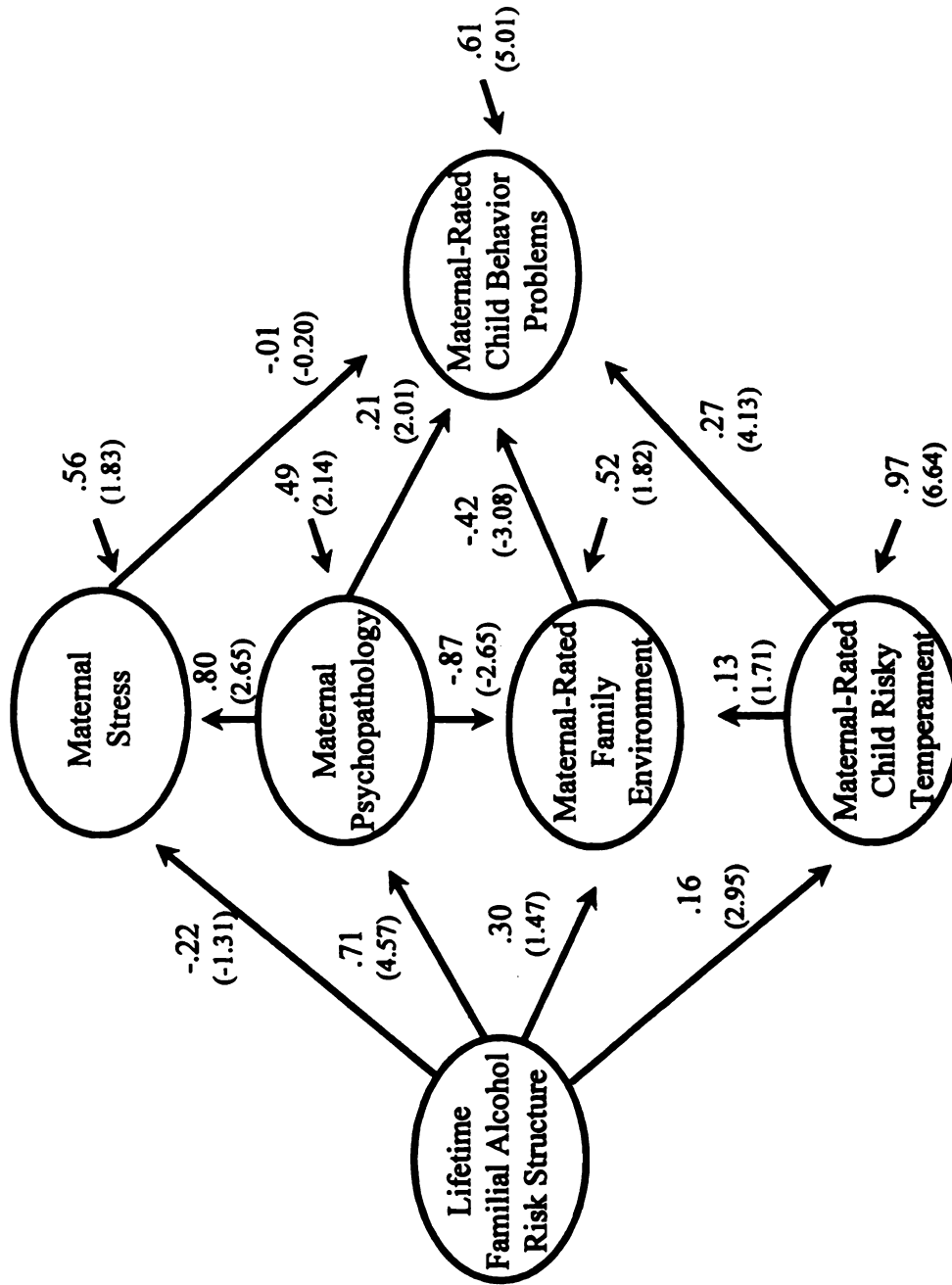
Note. t-values are in parentheses.

Figure G18. Structural model from Wave 1 two-group stacked analysis: Solution for maternal data for coupled mothers only.



Note. t-values are in parentheses.

Figure G12. Structural model from Wave 2 two-group stacked analysis: Solution for paternal data for coupled fathers only.



Note. t-values are in parentheses.

Figure G20. Structural model from Wave 2 two-group stacked analysis: Solution for maternal data for coupled mothers only.

predicted higher levels of parental psychopathology and more child risky temperament but not more parental stress or a worse quality family environment. Increased levels of parental psychopathology predicted more family stress as well as a worse quality family environment. Contrary to what was expected, child risky temperament directly predicted a better quality family environment. Finally, child externalizing behavior problems was predicted by increased levels of parental psychopathology, more child risky temperament and a poor quality family environment but not more parental stress.

APPENDIX H

Interpreting Change Models

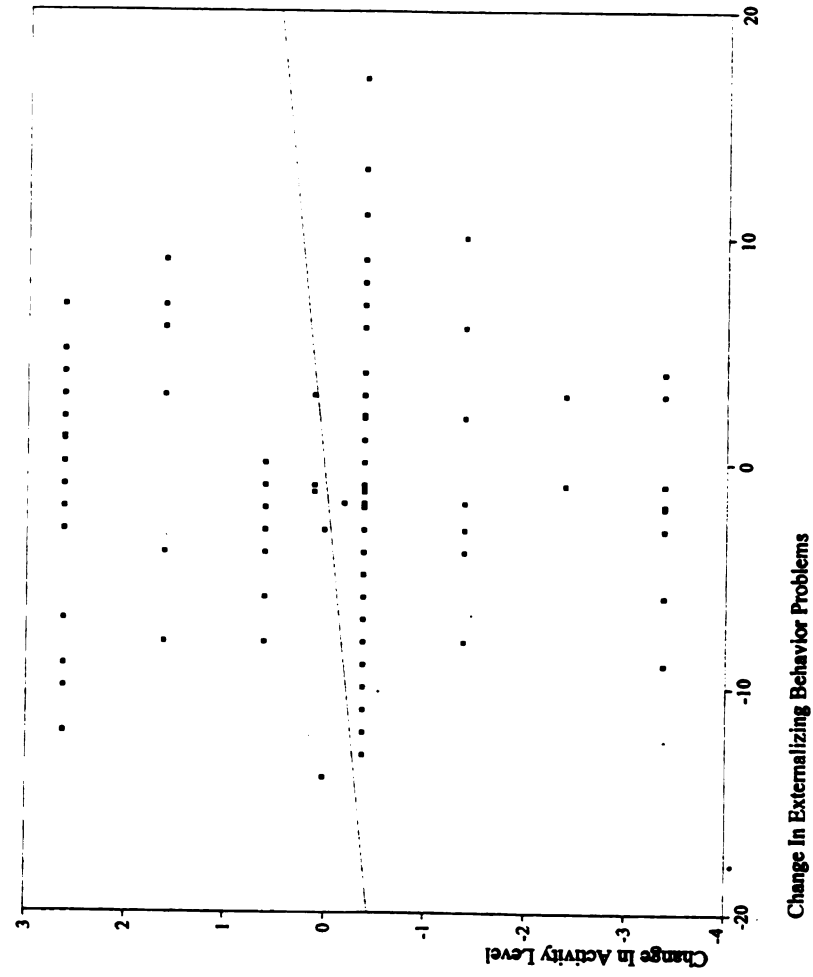
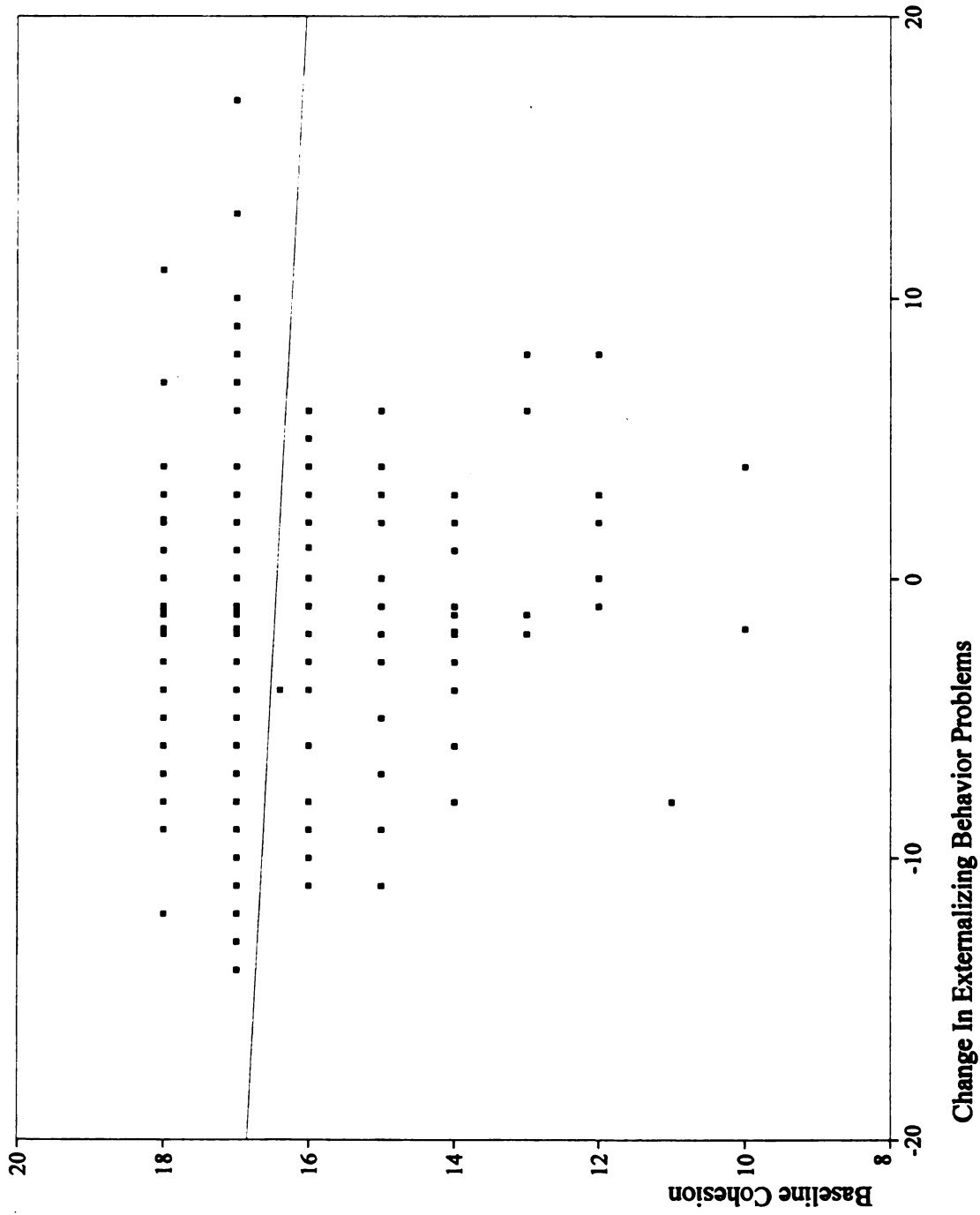


Figure H1. Relationship between paternal-rated change in child activity level and change in externalizing behavior problems.



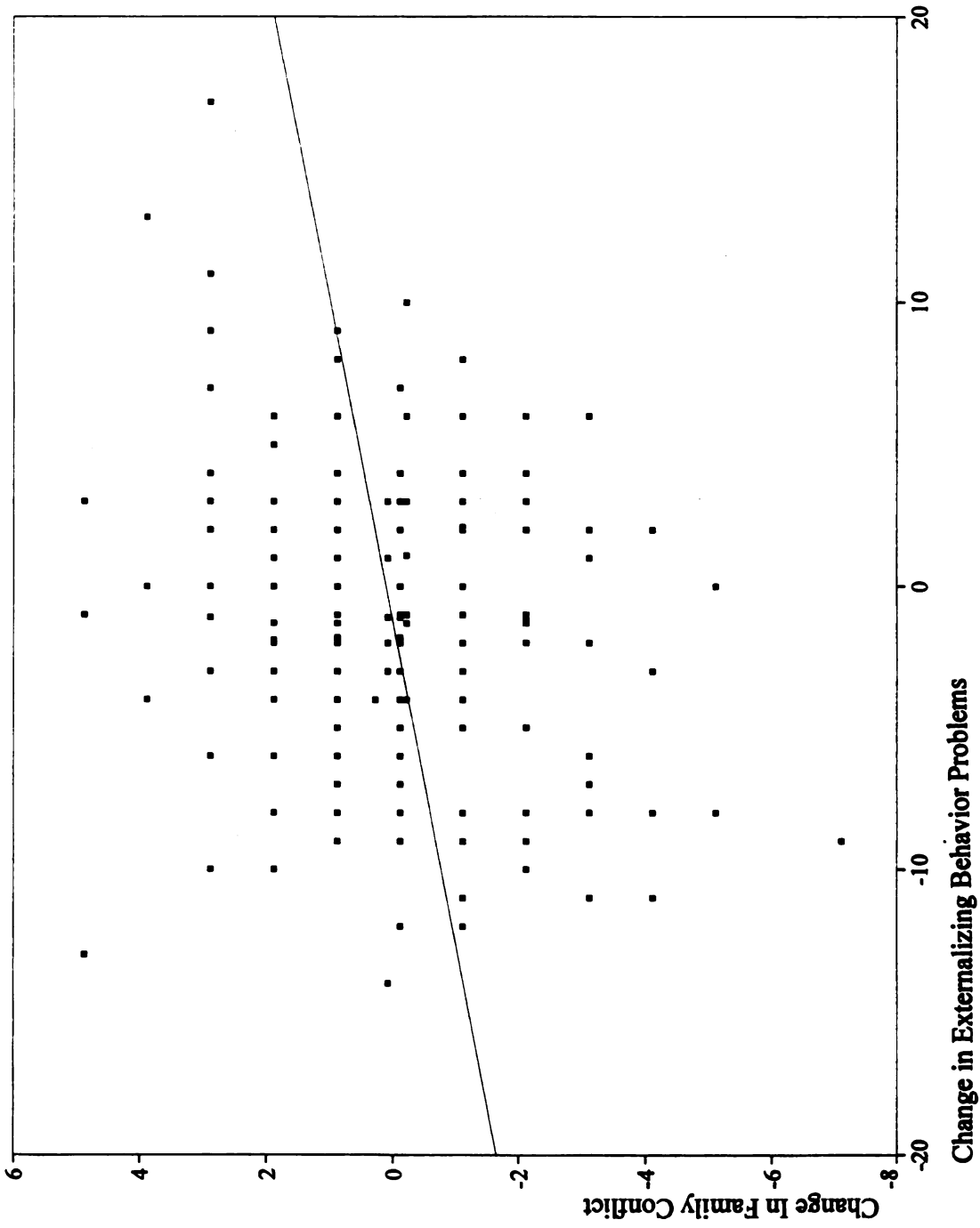


Figure H3. Relationship between paternal-rated change in family conflict and change in externalizing behavior problems.

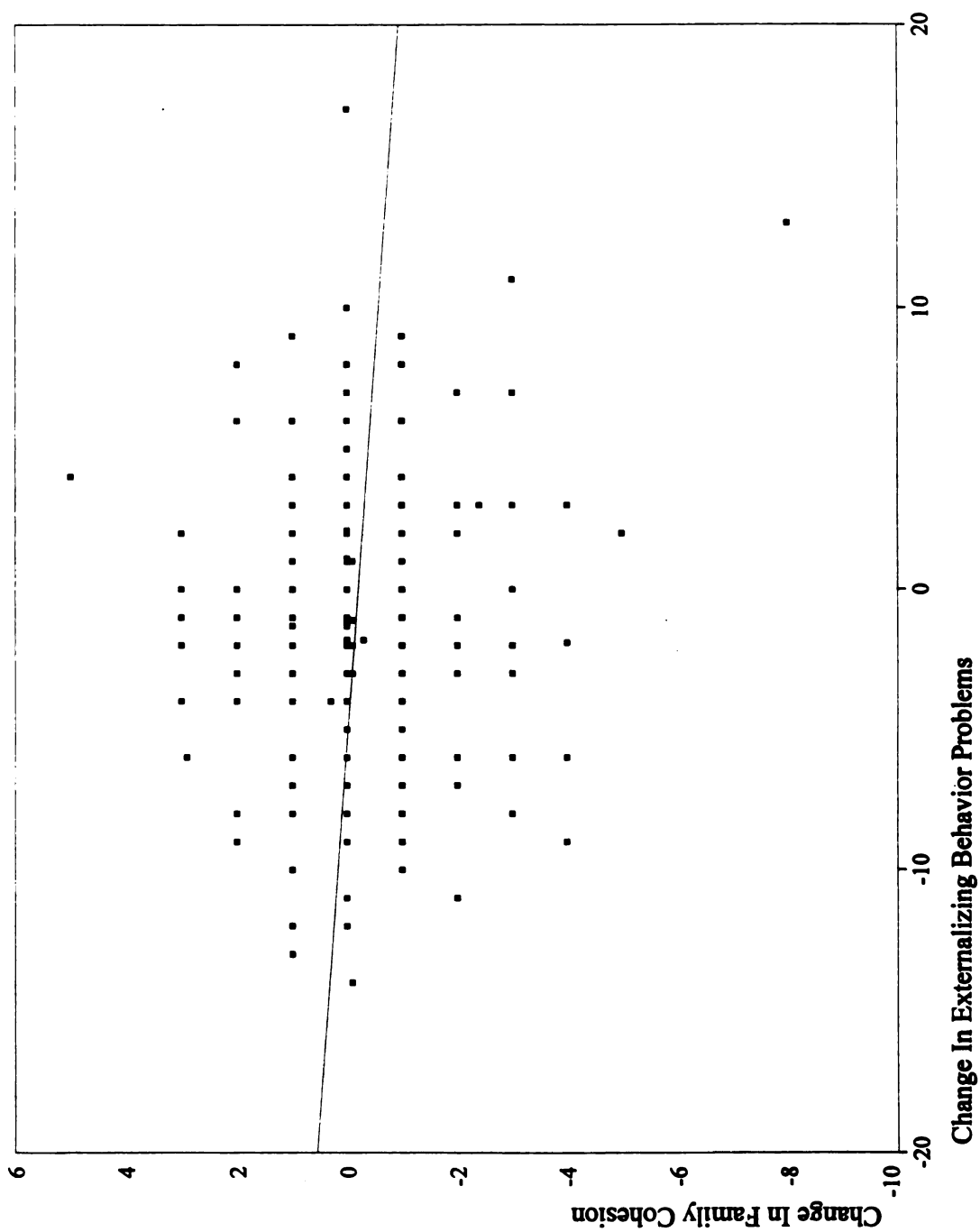


Figure H4. Relationship between paternal-rated change in family cohesion and change in externalizing behavior problems.

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LIST OF REFERENCES

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