# Children of Substance Abusers: Overview of Research Findings

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## Abstract

A relationship between parental substance abuse and subsequent alcohol problems in their children has been documented extensively. Children of alcoholics (COAs) are considered to be at high risk because there is a greater likelihood that they will develop alcoholism compared with a randomly selected child from the same community. COAs and children of other drug-abusing parents are especially vulnerable to the risk for maladaptive behavior because they have combinations of many risk factors present in their lives. The single most potent risk factor is their parent's substance-abusing behavior. This single risk factor can place children of substance abusers at biologic, psychologic, and environmental risk.

Since the turn of the century, many reports have described the deleterious influence of parental alcoholism on their children. A series of studies measured mortality, physiology, and general health in the offspring of alcoholic parents and concluded that when mothers stopped drinking during gestation, their children were healthier. Today, research on COAs can be classified into studies of fetal alcohol syndrome, the transmission of alcoholism, psychobiologic markers of vulnerability, and psychosocial characteristics. Each of these studies hypothesizes that differences between COAs and children of nonalcoholics influence maladaptive behaviors later in life, such as academic failure or alcoholism. This research supports the belief that COAs are at risk for a variety of problems that may include behavioral, psychologic, cognitive, or neuropsychologic deficits.

The vast literature on COAs far outweighs the literature on children of other drug abusers. Relatively little is known about children of heroin addicts, cocaine abusers, or polydrug abusers. Nonetheless, many researchers suggest that the children of addicted parents are at greater risk for later dysfunctional behaviors and that they, too, deserve significant attention to prevent intergenerational transmission of drug abuse. Most research on children of other drug abusers examines fetal exposure to maternal drug abuse.

The overview of the research on children of substance abusers points toward the need for better, longitudinal research in this area. Most studies on COAs or other drug abusers are not longitudinal; they examine behavior at one point in time. Given the studies reviewed in this article, it is unclear whether we see true deficits or developmental delay. Longitudinal studies will allow us to predict when early disorders and behavioral deviations will be transient or when they will be precursors to more severe types of maladaptive behavior. Longitudinal research also will enable us to explain specific childhood outcomes. Differences in outcome could be studied simultaneously to understand whether antecedents discovered for one are specific to it or are general antecedents leading to a broad variety of outcomes.

Addiction to alcohol and other drugs is a serious problem in the United States. Approximately 10% of American adults and 3% of adolescents in the United States are addicted to alcohol or other drugs.[1] As a society, we should be concerned about the rates of alcohol and drug use among our adolescent population. Determining why one adolescent is more vulnerable than another to drug use has been an area of research spanning the past 2 decades. Reviews of the literature on risk factors associated with drug and alcohol abuse in children or adolescents implicate many factors, such as childhood personality, hyperactivity, antisocial traits, stress, and interpersonal risk factors including low academic performance and commitment and associations with substance-using peers.[2–5]

A relationship between parental substance abuse and subsequent alcohol problems in their children has been documented extensively,[6–9] although some have found that parental substance abuse is not directly related to their children's substance-using behavior.[10] Several researchers have found that teenagers are more likely to drink and use drugs if their parents drink and/or use drugs.[11–13] Kandel and associates[13] found that 82% of drinking families raise youth that also drink, and that 72% of families who abstain raise youth who also abstain. Annis[11] found that a same-sex, same-use pattern seems to exist. Mothers and daughters have similar patterns of substance abuse (mostly tranquilizers and painkillers), and fathers and sons share their choice in drugs (usually alcohol and cigarettes). Coombs and Dickson[12] found that the substance abuse behavior of both the mother and the father influenced their children's substance abuse behavior. Mothers and fathers of substance-abusing youth tended to drink and to use other drugs more often and more heavily. Chassin and Barrera[14] explored substance use among adolescents over a 3-year period in 246 adolescent children of alcoholics (COAs) and 208 children of nonalcoholics. They noted important developmental differences in the use of alcohol and drugs among COAs. Older adolescent COAs showed steep escalations in drug use. Younger COAs showed escalations in alcohol and other drug use if their fathers had experienced alcohol-related consequences. If fathers did not experience alcohol-related consequences to their drinking, COAs showed a strong relationship between substance use and self-control reasons for limiting drinking. The research by Chassin and colleagues[14] also has shown other mediating mechanisms involved in adolescent substance use among COAs. She suggests that substance use among adolescent COAs is mediated through stress and negative affect pathways, decreased parental monitoring, and increased temperamental emotionality. These results have been supported partially by other research.[15] Overall, parental alcohol abuse has been determined to be a risk factor for their children's subsequent use.

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# RISK FACTORS

Children are labeled “at risk” for many different reasons. These “risk factors” are presumed to increase the likelihood of future maladaptation and can be environmental (eg, high or low socioeconomic status), biologic (eg, inheritance of a gene predisposing toward a disease state), or psychologic (eg, low self-esteem). With a rich tradition extending several decades, the high-risk paradigm has been used to study children at risk for a variety of problems related to their parents' depression, psychopathology, or substance use. COAs are considered to be at high risk because there is a greater likelihood that they will develop alcoholism compared with randomly selected children from the same community.[16] It is important to note, however, that research findings that identify risk factors suggest that these factors are associated with increased risk and do not necessarily constitute a causal relationship.

Children of alcoholic and other drug-abusing parents appear to be especially vulnerable to the risk for maladaptive behavior because they have combinations of many risk factors present in their life. The single most potent risk factor is their parent's substance-abusing behavior; this single risk factor can place children of substance abusers at biologic, psychologic, and environmental risk. Evidence suggests that the inheritance of a predisposition to alcoholism is specific and separate from the predisposition toward other types of drug abuse.[17] The evidence for the inheritance of a predisposition to other kinds of substance abuse is less clear, and there is evidence both for and against this notion.

In this review, we examine some of the research both on COAs and on children of other substance abusers. Parental substance abuse and its subsequent effects on their children are great. The Children of Alcoholics Foundation[18] estimates that there are 28.6 million Americans alive today who were raised in homes where one parent was alcoholic. The number of children younger than 18 years currently living with an alcoholic parent are estimated to total 11 to 17.5 million.[9] There are few prevalence estimates about the number of children who live in homes where drug abuse, other than alcoholism, occurs.

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# COAs

## Previous Reviews of the Literature

Since the turn of the century, many reports have described the deleterious influence of parental alcoholism on their children.[19–22] A series of studies published in London in 1910 measured mortality, physiology, and general health in the offspring of alcoholic parents and concluded that when mothers stopped drinking during gestation, their children were healthier.[23–25] Much of the research on COAs has been reviewed extensively by others.[26–40] No two authors have classified the research in quite the same way; it is evident, however, that there are a great many ways in which to approach the field. Overall, the research indicates that there is considerable heterogeneity within the COA population and that differences between COAs and children of nonalcoholics are not always substantial with respect to many of the individual risk factors. This

may be attributable to the subtypes of COAs and substance abusers. For example, many children have differing numbers of risk factors present in their lives (some children live with more risk factors than do others), and the cumulative effects of multiple risk factors are associated with later behavioral outcomes.

We separate research on COAs into studies of 1) the fetal alcohol syndrome (FAS); 2) the transmission of alcoholism; 3) psychobiologic markers of vulnerability; and 4) psychosocial characteristics. These studies hypothesize that differences between COAs and children of nonalcoholics influence maladaptive behaviors in COAs later in life, such as academic failure or alcoholism. This research supports the belief that COAs are at risk for a variety of problems, which may include behavioral, psychologic, cognitive, or neuropsychologic deficits.

## FAS

First described in the medical literature by Jones and Smith,[41] FAS is a cluster of four characteristics found in the offspring of mothers who drank excessively during pregnancy, namely, central nervous system dysfunction, abnormal facial features, behavioral deficits, and growth deficiency. Many studies of infants born to alcoholic mothers report strong relationships between in utero alcohol use and later childhood problems such as minor physical anomalies, hyperactivity, mental retardation, and electroencephalographic (EEG) abnormalities.[41–45] One study of 322 newborn infants showed a frequency of physical abnormalities twice as high among children of mothers who were heavy drinkers as among children of mothers who were not heavy drinkers.[46]

Longitudinal studies of infants exposed to alcohol abuse in utero have shown the lasting effects of their exposure. A large longitudinal study in Seattle, WA, involving 1529 white, middle-class, pregnant women and their offspring revealed that 12% of the infants of mothers who were heavy drinkers exhibited features of altered growth and morphogenesis, compared with only 2% of children of mothers with lower levels of alcohol ingestion.[47–49]

## Transmission of Alcoholism

There are many studies that support a genetic theory of alcoholism transmission, dating from Amark[50] to the present. Goodwin[51,,52] reports a 25% prevalence rate of alcoholism among male relatives of alcoholics, which exceeds the estimated population prevalence for male alcoholics of 3% to 5%. The prevalence of alcoholism among female relatives of alcoholics is 5% to 10%, which also exceeds the estimated population prevalence for female alcoholics (0.1%–1%). Researchers agree that the genetic model of alcoholism is multifactorial. Schuckit and colleagues[53–58] explain that both genetic heterogeneity and environmental influences combine in an unknown manner, placing some people at high risk and others not at risk for developing alcohol abuse or dependence. In high-risk individuals, Schuckit describes a decreased response to alcohol as a genetic risk factor. Tarter[59] discusses temperament traits interacting with environmental contingencies that increase one's risk for alcoholism. Heath and colleagues[60] note the relationship between personality and temperament and cardiac responsivity in high-risk children. Wiers and associates[61] posit two different pathways for COAs. They suggest that the child of a multigenerational, primary alcoholic parent may suffer from an inherited mild

dysfunction of the prefrontal cortex, leading to neuropsychologic and personality characteristics similar to those of the alcoholic parent. The child of a secondary alcoholic parent may be subject to stress and social learning that may lead to negative affectivity and repressive coping style, leading to substance abuse or dependence.

**Studies of Twins**

Several researchers have studied the genetic predisposition to alcoholism in identical and fraternal twins in whom at least one of each twin pair was an alcoholic. Because identical twins share the same genes and fraternal twins do not, a higher level of alcoholism among identical twins would support a heritable basis of alcoholism. Many studies typically demonstrate that the frequency of alcoholism in monozygotic (MZ) twins is higher compared with that in dizygotic (DZ) twins.[62–64] Pickens and colleagues[65] studied both MZ and DZ male and female twin pairs. They noted a significant MZ/DZ concordance in male twins for alcohol abuse, alcohol dependence, and other substance abuse and/or dependence. For the female twin pairs, there was only a MZ/DZ concordance for alcohol dependence.

Findings from other studies of twins indicating high concordance of alcoholism among MZ twins (and thus a genetic basis for alcoholism) are contradicted by the study of 902 male twins in Finland.[66]This study showed no statistically significant differences in alcoholism rates between identical and fraternal twins. It did indicate, however, that the frequency and amount of drinking was significantly similar for identical than for fraternal twins. In a study of 3810 twin pairs from Australia, Heath[60] showed important genetic influences for frequency and quantity dimensions. Findings from other studies of twins are shown in Table 1.

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Table 1.

Summary of Studies

**Adoption Studies**

Examining children born to alcoholic parents adopted at birth and raised by nonalcoholics is a useful method to study genetic and environmental variables associated with alcoholism later in life. Scandinavian adoption studies, especially, provide a convincing picture of the possible genetic influence of alcoholism.[67–73] In one representative study, male adoptees whose biologic fathers were alcoholic were four times as likely to become alcoholic.[71]

Goodwin[52] has been one of the most active investigators of adopted COAs. From his many studies, he[51,,52,67,68,74] concludes that sons of alcoholic biologic parents adopted at birth were four times more likely to become alcoholics than were sons of normal control fathers. Alcohol problems experienced by adopted sons included early onset of heavy drinking, loss of control,

hallucinations, and treatment for drinking. Significant alcoholism was experienced by adopted sons biologically parented by alcoholics.

Cadoret and co-workers[75] studied 197 adult adoptees (95 male and 102 female) of alcoholic biologic parents. They determined that a genetic factor is present for which alcoholism is a marker and that exerts its effect in women as a gene/environment interaction leading to major depression. McGue and colleagues[76]studied 653 adopted families, with one adopted child and other siblings (either biologic children or other adoptees), and found that the relationship between parental problem drinking, family functioning, and adolescent alcohol involvement was moderate and significant among birth offspring, not among adoptive offspring.

Cutrona and associates[77] studied both male and female adoptees and describes that for female adoptees, both early life family conflict and psychopathology in the adoptive family interacted with genetic factors to increase the women's risk for alcohol abuse or dependence. This was not true for the male adoptees studied.

### Gender Differences

In the literature, gender differences in risk for substance abuse are shown.[65,,77,78] Hill[79,,80] describes the recent literature on the genetic mediation of alcoholism in women. Moskalenko and co-workers[81] compared female to male alcoholic inpatients and found that women were more likely to have an alcoholic father and/or an alcoholic spouse. Orford and Velleman[82] studied 169 16- to 35-year-old offspring of alcoholics and found that women who had a positive relationship with an alcoholic father were at greater risk for alcohol or other drug use (Table 1).

## Biologic Studies

Biologic mechanisms that differentiate COAs from children of nonalcoholics involve several different physiologic systems.[83] Compared with children of nonalcoholics, COAs differ on EEG findings,[84] event-related potentials (ERPs),[85] and endocrine deviations.[56]Begleiter and colleagues[85] showed that the P3 component of the ERP, an electrophysiologic measure of cognitive processing, is decreased significantly in COAs compared with children of nonalcoholics. This line of research promises to be instrumental in the future identification of biologic markers for alcoholism. However, the research has not yet successfully identified a premorbid biologic pattern to predict those who become alcoholic from those who do not.

Psychobiologic research is still in the early stages of development, and many studies are subject to methodologic limitations. Few findings have been replicated to the extent that conclusions can be regarded as definitive, especially when applied in cultural settings or age groups that are different from those used in the research sample. Sufficient research has been conducted; however, pieces of the puzzle still are missing to suggest a preliminary picture of biologically transmitted vulnerability toward alcoholism in COAs.

Results of these studies indicate that COAs react differently to alcohol or other drugs because of differences in biochemical transmission. Researchers have long hypothesized that COAs may suffer chemical imbalances that make them prone to substance-abusing behaviors. Alcohol and

other drugs may provide increased beneficial and pleasurable effects in COAs that are not experienced by children of nonalcoholics. This could provide stronger reinforcement for continued drinking among COAs. Research has demonstrated that COAs have higher levels of blood acetaldehyde and increased feelings of pleasure and relaxation from alcohol ingestion[54]; increased elation and/or decreased muscle tension in response to alcohol ingestion[86]; decreased feelings of intoxication at the same blood alcohol levels, compared with children of nonalcoholics[87]; and a possible serotinergic deficiency or an exaggerated level of serotonin when ingesting alcohol.[88]

**Temperament Variables**

Tarter[89] and Rowe and Plomin[90] examined temperament in COAs as a possible precursor to subsequent alcohol or other drug abuse. Kumpfer[91] cautions against overgeneralization of this research by suggesting that these temperament traits may only describe a subset of children who have inherited one of the major vulnerability syndromes associated with noncompliant, antisocial, and/or hyperactive behavior. Indeed, the findings of many of these studies may have been affected significantly by sample selection and lack of appropriate controls. Some studies report hyperactivity among children of substance abusers.[92–102] Many of these studies identify significant, but rather weak, linkages between hyperactivity and familial alcoholism. In addition, some support for the genetic transmission of hyperactivity has been established, which confuses causal assumptions.[95] Other studies report: 1) decreased ability in COAs to return to emotional normality after emotional distress[101]; 2) increased aggressive behavior (or decreased social inhibitions) among COAs[96,,97,102,103]; and 3) increased tendency to be "hot tempered,"[68] with decreased emotional control, low frustration tolerance, and increased moodiness and depression in COAs.[104]

**Neurophysiologic Studies**

In reviewing the electrophysiologic research in alcoholism, Porjesz and Begleiter[105] reported that the most consistent finding was the diminished P300 amplitude of the ERP seen in family history-positive men. Results of recent neurophysiologic studies in alcoholism listed in Table 1 are briefly summarized. Polich and colleagues[106] found smaller P300 amplitudes in family history-positive males. Steinhauer and Hill[107] also found decreased P300 amplitude, especially in high-risk, older males. Hill[81] followed high-risk individuals over an 8-year period and demonstrated continued differences in the high-risk individuals when they were compared with a low-risk group. The ERPs of children who were both high-risk and abusing alcohol were different from those of the other nonalcohol-abusing high-risk and low-risk groups. When the ERPs of alcoholic men, their high-risk relatives, and low-risk male controls were compared, no differences were found in the P300 component during auditory tasks. One ERP study looked at boys exposed to opiates in utero[108] and found that the P200 component of the ERP was decreased similarly in these two groups, as opposed to the control group, suggesting an environmental influence on a neurophysiologic process.

**Biologic Marker Studies**

A few studies have investigated potential biologic markers. These include studies of various neurotransmitters (GABA, metabolites of dopamine), salivary cortisol, cardiovascular responsivity, and motor activity. Eskay and Linnoila[109] reviewed the literature (Table 1). This field of research may be promising; however, the findings have not been replicated or confirmed to any convincing degree.

## Psychosocial Research

Psychosocial studies have examined a wide range of variables associated with psychologic and environmental characteristics of COAs. In this section, we will review the research involving family studies (including family violence), cognition, affect and behavior, medical problems, and physical health.

## Family Studies

Transmission of alcoholism in family members involves many different factors.[30,,110,111] Parental alcoholism disrupts family life and contributes to dysfunction in the offspring, which, in turn, could affect adolescent substance abuse.[40]

One important area of research examines family rituals, ie, dinners, holidays, or vacations.[112,,113] Bennett and colleagues showed that the degree of organization and disruption in the alcoholic family would distinguish the differential well-being of adult COAs.[111,,114,115] Family ritual disruption is significantly associated with differential transmission of alcoholism. Maintaining family rituals during periods of heavy parental drinking results in fewer transmittable cases of alcoholism compared with those families that alter their rituals. Ritual stability in alcoholic families during childhood and adolescence appear to influence later alcoholism. Thus, those families showing more stability also evidence less alcoholism in adult COAs.

Alcoholic families report higher levels of conflict than do nonalcoholic families.[116] Drinking is the primary factor in family disruption. The environment of COAs has been characterized by a lack of parenting, poor home management, and a lack of family communication skills, thereby effectively robbing COAs of modeling or training on parenting skills or family effectiveness.[117]The following family problems have been frequently associated with alcoholic families: increased family conflict; emotional or physical violence; decreased family cohesion; decreased family organization; increased family isolation; increased family stress including work problems, illness, marital strain, and financial problems; and frequent family moves.[28,,29,115118–121]

Substance-abusing parents often lack the ability to provide structure or discipline in family life, but simultaneously expect their children to be competent at a wide variety of tasks earlier than do nonsubstance-abusing parents.[119,,120] Unable to do everything perfectly all the time, children in these families may perceive themselves as failures. Young COAs are negatively effected when the significant caregiver in the family (usually the mother) is heavily involved in alcohol or other drug abuse; the child is still young; the family becomes significantly involved in the abuse problem; the family becomes socially isolated; or there is a lack of an extended family to provide balance and encouragement to the child.[119,,120]

## Family Violence

With respect to the overall negative impact of parental drinking, family violence has been one area that has received considerable attention. According to Sher,[33] although clinical reports often indicate a strong connection between parental alcoholism and family violence, the empirical data give a highly inconsistent picture. Family violence cannot be related conclusively only to parental alcoholism. Studies focusing first on family violence and second on incidence of parental alcoholism (as well as those reflecting on the dynamics of alcoholic families and subsequent assessment of family violence) both have resulted in highly inconsistent rates of reported spousal and child abuse. Mayer and Black[122] report extremely wide-ranging rates (2%–62%) of alcoholism among parents who abuse their children. Sher[33] found that the reported rate of child abuse among alcoholic parents varied between 0% and 92%. In studies of COAs, widespread beliefs on the association between parental alcoholism and family violence may precede any conclusive research.

Although contradictory conclusions emerge from a review of the literature, some studies find significant relationships between living in an alcoholic home and physical child abuse cases.[122,,123] However, Orme and Rimmer[124]found no relationship between child abuse and living in a home where at least one parent was alcoholic. Although data are sparse, a slight relationship exists between acts of incest in alcoholic parents and their children.[125,,126] The typical family model of an incest victim is that of a chronically depressed mother, an alcoholic and violent father, and an elder daughter forced to assume maternal roles in the family.

## Cognition

Lowered academic functioning in COAs has been reported by several researchers.[53,,97,127] Some data, however, do not agree with these findings.[128] COAs, partially because of the lack of parental supervision, are typically characterized as having both social and academic problems at school. Kumpfer and deMarsh[119] report that these children frequently are absent or tardy and poorly clothed and fed, and receive less help from parents with their schoolwork. Lowered levels of intellectual functioning in COAs have been reported by some researchers,[34,,92,129,130] but not by others.[131–133]

Many researchers offer different explanations for the inconsistencies found in the literature. Tarter and co-workers[134]suggested recently that an anterior cerebral dysfunction was responsible for the observed cognitive deficits in COAs, implicating a possible biologic basis for the observed cognitive differences in them. After examining perceptions of cognitive competence and actual cognitive performance, Johnson and Rolf[135] suggested that the observed negative perceptions of cognitive competence in COAs may effect the motivation to perform at an optimal level. Werner's[136] research shows that cognitive deficits may not characterize COAs as a group. Her longitudinal study on the island of Kauai compared a subgroup of COAs with problems (eg, repeated or serious delinquencies, mental health problems requiring treatment) to COAs without problems. She showed that COAs with problems scored lower on verbal and quantitative cognitive measures. Werner suggested that only a subgroup of COAs were at risk for cognitive deficits.

## Affect and Behavior

Research shows that COAs have more adjustment problems in home, health, social, and emotional domains,[137] but these problems do not always meet clinical diagnostic levels. In a study conducted by Nylander and Rydelius,[138] COAs raised in low socioeconomic environments were compared with children raised in high socioeconomic environments. Both groups were found to be more inclined that children of nonalcoholic biologic fathers to develop social maladjustment problems and addictions later in life. Furthermore, children from lower-class families showed no significantly increased inclination for addiction compared with the group of children from higher socioeconomic status groups, thus indicating that it was parental alcoholism, rather than socioeconomic status, that contributed to the child's behavioral problems.

Earls and colleagues[139] reported recently on the frequency of psychopathology in COAs. Results from extensive structured interviews with 75 children 6 through 17 years of age showed that these children were diagnosed more frequently with a behavioral disorder, an attention-deficit disorder with hyperactivity, an oppositional disorder, or a conduct disorder. An earlier study corroborates this finding showing that COAs present more frequently with behavioral problems similar to those behaviors associated with these psychiatric disorders.[140] Mutzell[141] has also published similar findings. Others, however, do not support this contention.[59] These authors demonstrated that parents diagnosed with substance abuse do not necessarily impart maladjustments in physical or mental health to their children.

Wolin and associates[114] provide convincing data showing that children from intact (eg, 2-parent) alcoholic families function less successfully on aggregate measures of emotional and behavioral functioning than do children from intact nonalcoholic families. They compared a homogeneous sample of 64 COAs to 80 children of nonalcoholics on an extensive psychosocial battery that included measures of self-concept, behavior problems, and psychiatric symptomatology. COAs scored significantly lower on 6 of the 13 measures of behavioral and emotional functioning.

Other research illustrative of the findings of behavioral problems in COAs shows:

- Lack of awareness of the perceived impression of one's behavior on others, lack of insight into personal relations, and lack of empathy for other persons[142];
- Decreased social adequacy and interpersonal adaptability[34,,96];
- Increased levels of anxiety and depression, low self-esteem, and lack of control over the environment.[34,,96,116,131,133,135143–146] All researchers but Tarter and colleagues[133] found a positive relationship between parental alcoholism and impaired emotional development in children;
- More diagnostic disorders among COAs that reach clinical levels[147];
- Higher rates of oppositional and conduct disorders, but not of attention deficit disorders[148];
- A tendency to engage in more delinquent behavior, compared with controls.[10] These findings are not consistent; Hill and Muka[149] and Hill and Hruska[150] found no differences between the two groups.

## Medical Problems and Physical Health

Recent research has examined the medical and physical health problems in children of substance-abusing parents. Woodside and associates [151] found that COAs spent more days in the hospital, incurred greater hospital charges, and were more susceptible to specific illnesses such as mental illness, substance abuse, injuries, and poisonings. These problems, however, do not always differentiate COAs from normal controls. For example, Dobkin et al[152] found that COAs were not sicker than were children of nonalcoholics. There were subgroup differences that showed that daughters of alcoholics and sons of nonalcoholics living in nonintact families were more likely to have used psychologic services, similar to sons of alcoholics in intact families.

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# CHILDREN OF OTHER DRUG-ABUSING PARENTS

The literature on COAs far outweighs the literature on children of other drug abusers. Relatively little is known about children of heroin addicts, cocaine abusers, or polydrug abusers.[153,154] Many researchers, nonetheless, suggest that the children of addicted parents are at greater risk for later dysfunctional behaviors and that they, too, deserve significant attention to prevent intergenerational transmission of drug abuse later in life.[155–160] Children of substance-abusing parents are at great risk for behavioral problems and physiologic damage when exposed in utero to their mother's drug addiction. Some of these problems may last well through maturation. We currently lack the necessary longitudinal data allowing any firm conclusions about the long-term effects of parental substance abuse. Even if children are not exposed to chemicals in utero, they are at greater risk for childhood behavioral problems if their parents are involved in the drug culture. Most research on children of other drug abusers examines fetal exposure to maternal drug abuse. The following section reviews the published literature on this topic. We have categorized this section into family studies/heritability, fetal exposure, and psychosocial risk factors.

## Family Studies/Heritability

Family history variables are considered one of the leading risk factors contributing to substance-abuse behavior.[91]Croughan[163] summarized the brief literature on family studies of drug abuse by concluding that family factors play a major role in substance use and abuse habits. Parents' and adolescents' use of illicit substances is strongly correlated. Adolescents who use drugs are more likely to have one or more parents who also use drugs.[8,11162–165] Parental attitudes about their children's drug-taking behaviors may be as important as actual drug abuse among the parents.[166] If adolescents perceive their parents are permissive about drug use, then they will be more likely to use drugs themselves.

As with studies on alcoholism, researchers now suggest that genetics may play a role in drug use and abuse.[72] Two recent studies dispute this. Kosten et al[78] studied opioid addicts and found gender differences in drug versus alcohol transmission. Women required genetic loading to become alcohol-dependent. They also found that the transmission of drug use compared with the transmission of alcohol was specific for women and not for men. In another study, Smith and Frawley[167] found increased rates of alcohol abuse/dependence in relatives of nonalcohol-abusing,

cocaine-dependent patients, suggesting a more general genetic inheritance for addiction, rather than for abuse/dependence, of a specific substance.

A molecular genetic study was reported in the literature. Noble et al[168] studied the allelic prevalence of the D2 dopamine receptor (DRD2) gene in cocaine-dependent male subjects. They found a significantly higher prevalence of both the A1 and B1 alleles of the DRD2 gene in these subjects, compared with community samples and with nonsubstance-abusing subjects. They postulate that perhaps a gene, located in the q22–a23 region of chromosome 11, confers susceptibility to cocaine dependence.

### Fetal Exposure

Because most drugs cross the placenta, pregnant addicts risk passive drug dependency in their fetus.[169] Fifty-eight percent[10 000] of the 17 000 heroin-addicted women entering National Institute on Drug Abuse-funded drug treatment programs have children living with them.[170] Prenatal drug withdrawal, caused by a pregnant woman's withdrawal, can inhibit fetal oxygen consumption, resulting in hypoxia or death. Postnatal drug withdrawal is characterized by the neonatal abstinence syndrome that includes hyperirritability, tremors, gastrointestinal dysfunction, respiratory distress, and amorphous autonomic system problems. Infants of heroin addicts or methadone-maintained (MM) mothers exhibit more tension, activity, and poorer coordination than their age-matched peers.[171–175] Cocaine abuse during pregnancy is a significant predictor of low birth weight and gestational age.[176]

Infants of drug addicts also are at risk for a variety of other problems. Child abuse or neglect is a significant concern for these infants.[175] Infants of drug-addicted women also are at risk for HIV infection.

## Psychosocial Risk Factors

The scarcity of research on school children of heroin-addicted parents is discussed in a literature review by Hayford and associates.[177] This review includes only 11 studies, 10 of which are about infants. The few clinical reports available describe psychologic and social problems for the children of addicted parents.[156,,159] Bauman and Levine[178] compared preschool children of MM mothers to children of nondrug-addicted mothers. On an extensive battery that included tests of intelligence and personality, they showed that children of MM mothers were more impulsive, immature, and irresponsible. Furthermore, children of MM mothers performed more poorly on intelligence tests. Sowder and Burt[179] also report decreased IQ scores among 3- to 7-year-old children of MM mothers.

Studies of school children of addicted parents are compromised by the possibility of fetal exposure to heroin. Distinguishing environmental from genetic effects is difficult when the child may have been contaminated in utero by the mother's substance abuse. Wilson and colleagues[180,,181] reported behavioral disturbances in heroin-exposed children 12 to 24 months of age. Sardemann and colleagues[182] found delayed language development in heroin-exposed children 24 to 32 months of age. Learning problems and behavioral disturbances in 33 children of addicted parents also have been reported.[159]

Questions about differences in personality, psychosocial competence, and affect in children of addicted parents remain unanswered. Wilson and colleagues[180] compared four groups of children on a comprehensive psychologic assessment battery. These four groups, each with 77 children between 3 and 6 years of age, were 1) exposed to heroin in utero; 2) not exposed to heroin in utero, but their mothers were involved in the drug culture (either through marriage to an addict or through substance abuse subsequent to the birth of the child); 3) a high-risk comparison group (birth complications attributable to medical problems); or 4) a socioeconomic comparison group. The extensive assessment battery included perinatal measures, a physical examination, social and environmental information, parent's reports, psychometric measures (primarily measures of intelligence), sensorimotor tests, and behavioral measures. Not surprisingly, the heroin-exposed group scored lower than all other groups on physical, intellectual, sensorimotor, and behavioral measures. The children whose mothers were actively involved in the drug culture scored slightly higher than did the heroin-exposed group, but significantly lower than the two comparison groups.

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# CONCLUSIONS

This overview of the research on children of substance abusers points toward the need for better, longitudinal research in this area. Most studies on COAs or other drug abusers are not longitudinal; they examine behavior at one point in time. Given the studies reviewed in this article, it is unclear whether we see true deficits, or developmental delay. Longitudinal studies will allow us to predict when early disorders and behavioral deviations will be transient or when they will be precursors to more severe types of maladaptive behavior. Longitudinal research also will enable us to explain specific childhood outcomes. Differences in outcome could be studied simultaneously to understand whether antecedents discovered for one are specific to it or are general antecedents leading to a broad variety of outcomes.

In 1974, Anthony[183] suggested the possibility that there were different groups of children of substance abusers and that all children of substance abusers could not be considered a single, unitary entity. Similar experiences affect children differently because of individual differences in factors such as temperament, intelligence, and environmental resources. Therefore, every summary of children of substance abusers should take into account that there is most probably no single profile of children of substance abusers.

Most importantly, however, there actually may be subgroups of children of substance abusers who, despite all odds, do enjoy good health from birth; experience a positive environment at home; and develop rather normally into socialized, competent, and self-confident individuals. Certain individuals may be more competent in adapting to stressful living environments than are others. This is what many have referred to as the resilient individual. Such a child is able to compensate for and cope with the various negative biologic or environmental influences in his/her life. Certain individuals may be able to manipulate their environment by choosing roles and goals in life that stabilize their developmental process and bring them the positive reinforcement they need to develop a positive self-image and eventually a relatively healthy life. Other individuals may be able to master the environment and to conceptualize the environment

in such a way as to choose positive behaviors in life that compensate for whatever problems are present. Garmezy[184,185] posits that resilient characteristics include effectiveness in play behavior, work behavior, and love relationships; self-esteem; self-discipline; and the ability to think abstractly. Some evidence from Miller and Jang[186] bears witness to this. In their 20-year study of children from lower-class multiproblem urban families, they found that parents' alcoholism was related to increased problems during childhood and an increased probability that the child of an alcoholic would develop drinking problems later in life. The greater the degree of parental alcoholism, the greater the negative influence on the children of the family. However, they also found that although parental alcoholism might contribute to problems for their child in later adulthood, predicting intergenerational transmission of alcoholism is impossible. Thus, Anthony's[183] proposal that children of substance abusers actually may be a complex group of individuals that cannot be described by single, unitary profiles of personality or behavior may prove to be the rule, rather than the exception.

COA =
: children of alcoholics •
FAS =
: fetal alcohol syndrome •
EEG =
: electroencephalography •
MZ =
: monozygotic •
DZ =
: dizygotic •
ERP =
: event-related potential •
DRD2 =
: D2 dopamine receptor (gene) •
MM =
: methadone-maintained •
FHP =
: Family History Positive •
FHN =
: Family History Negative •
HVA =
: homovanillic acid •
DBH =
: dopamine-β-hydroxylase

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