

DISSERTATION PROPOSAL

Identifying a Subgroup of Adult Children of Alcoholics  
Who Have Signs of Prenatal Alcohol Damage

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Signs of Prenatal Alcohol Damage-Revised (S-PAD-R)  
(Page, 2001)

## CHAPTER I.

### INTRODUCTION

My involvement with prenatal alcohol damage began 18 years ago when I adopted a baby boy in Colombia, but this connection only became clear a few years ago during the time I worked as a school psychologist. In that role I saw many children whose behavior and learning were compromised in ways that traditional teaching and parenting could not touch, in patterns that did not yield to any of the usual explanations. These children appeared to be willfully disobedient and self-defeating, beyond (but usually including) the parameters of Attention Deficit/Hyperactivity Disorder, Learning Disabilities or environmental influences. On closer observation, soft signs of neurological impairment could be detected, such as wide discrepancy between verbal and practical abilities, memory problems, poor handwriting, difficulties with balance, sudden rages, fluctuation from one day to the next in behavior, mood and cognitive ability, and hypo- or hypersensitivity to pain. A disproportionate incidence of physiological abnormalities (though less consistent than the behavioral and cognitive difficulties) was noted as well, including small size, lowered ears, eyes set wide apart, little fingers curved outward, small or malformed teeth, small chin or disproportionately small head. There was also in this group a higher-than-normal tendency toward asthma, allergies and hearing and vision problems. As this collection of attributes emerged as a pattern, the serious difficulties experienced by (and with) my own son began to sort themselves out into a recognizable schema; he was similar to this group in many ways, and what had looked like a puzzling but simple pattern of willful, intractable disobedience and self defeat took on a whole new significance.

I began to explore the issue of Fetal Alcohol Syndrome (FAS) and found there a hypothesis that closely matched the problems these children had in common. Inquiry about the backgrounds of the students I worked with yielded evidence of a very high frequency of exposure to alcohol in utero, and through this particular lens much of the previously baffling behavior began to make sense.

The core disabilities of FAS appear even more sharply in my current position working with the Santa Clara Juvenile Probation Department, Alternative Schools and teen mothers: the disorganized thinking, rages, memory problems, inability to plan, and poor impulse control that these youth demonstrate appear to have landed them in trouble well beyond any malicious intent. Probation staff estimates in-utero exposure to alcohol to be about 85%. From my vantage point as ADHD/LD/FAS consultant to the Juvenile Drug Treatment Court team, it is becoming increasingly clear that a great deal of criminal activity appears to stem from brains that do not work right, that the parents of many of these children have abused substances, especially alcohol, and that the pattern of behavior fits that of FAS.

My interest in FAS has most recently resulted in the development of a diagnostic clinic for Fetal Alcohol Syndrome at Valley Medical Center. This is one of only a few places in the country where brain damage from prenatal alcohol exposure is being diagnosed (especially in the absence of facial abnormalities). Receiving a diagnosis almost always results in a general sense that the mystery of incomprehensible and troubling behavior, sensory and learning problems has finally been solved.

The focus of this study, however, is not the seriously dysfunctional populations described above, but the less visible end of the spectrum of prenatal alcohol damage,

where it crosses over into the population more likely to seek out professional help for perceived character defects: the group of people who identify themselves as Adult Children of Alcoholics (ACAs). Two internships in drug and alcohol treatment centers deepened my understanding of psychodynamic issues around chemically-dependent families, but as I learned more about the sequelae of prenatal alcohol my personal interest took another sideways leap, in addition to interest surrounding my son. I recognized the ghost of these patterns in myself, patterns which had robustly resisted trying hard, psychotherapy, 12-Step groups and abundant reading on the subject of growing up in an alcoholic family. These patterns, a very mild version of FAS, included – in spite of an IQ that was explained 40 years ago to my parents as “off the charts” – a very poor ability to plan, poor impulse control, huge discrepancy between verbal skill and other cognitive abilities, losing track of important aspects of my life (material or otherwise), impaired judgment and great difficulty with follow-through. A diagnosis of Attention Deficit/Hyperactivity Disorder a few years ago was a relief, but still left some odd disabilities unexplained. The arrival of FAS on my mental scene completed the picture, as it had with my son just a couple of years before, and as it continues to do with the troubled youth I work with.

Trying on the hypothesis that a very mild case of brain damage might be behind my troubling and persistent ways of being allowed me to arrange my life accordingly. Neurological impairment was a refreshing way to look at what had been seen (by many, particularly the drinking members of my family) as a simple case of poor character. As I continued to work on my own ACA issues, it became clear to me that in fact many of the classic ACA afflictions can be regrouped into the patterns evinced by people with some

degree of prenatal alcohol damage. As this apparent double vision swam into focus, the unspoken question resolved itself: of COURSE it's the same pattern...it's the same PEOPLE!

The very abundant literature on ACAs describes a set of emotional, cognitive, physiological/medical, behavioral and interpersonal characteristics of people who grew up with alcoholic parents. Few of these characteristics could be seen as assets; untreated, they often result in failures in relationships, unsatisfactory employment or school experience, hospitalizations and trouble with the law. The only positive characteristics noted in a few of the offspring of alcoholics center around what may be seen as hyper-responsibility, bringing its own set of difficulties in relationships later on.

The emerging research on prenatal exposure to alcohol yields a set of patterns that greatly overlaps the ACA characteristics, clustering around Learning Disabilities, aggression, lack of emotional intelligence, Attention Deficit/Hyperactivity Disorder, and a wide variety of medical vulnerabilities. The "secondary disabilities" (Streissguth, Barr, Kogan & Bookstein, 1996) that arise in the absence of early diagnosis and intervention in FAS sharply parallel the interpersonal, legal, academic and physical difficulties of ACAs as well. Although it would seem logical to assume that at least some ACA symptoms may have their origins in fetal alcohol damage, the two fields have largely stayed separate and the link has rarely been postulated.

The ACA field is only one branch of study among many that ignore prenatal alcohol as an explanation for the above-described set of difficulties. Several opposing hypotheses have been floated from various corners of research and theory. The only

agreement among the contenders is that this is indeed a co-occurring set of difficulties, rafted together across time, socio-economic status, gender and ethnicity.

Some theories have congealed by now into conventional wisdom. The field surrounding adoption, for example, attributes the problems in living common in adopted children to the primal narcissistic wound of early separation from the mother. The fact that many adopted children were removed from their birth mothers for conditions resulting from substance abuse does not intrude on this theory in the form of even a hypotheses of brain damage, although the foster and adoption systems are rife with children with the small size, hyperactivity, cognitive problems and even facial characteristics common to FAS. I have heard from clinicians familiar with neurological damage that the common mental health view – that this whole set of problems stems from a narcissistic wound that must be healed – leads to long, frustrating, fruitless relationships with a string of providers, leaving these symptoms of brain damage entirely unrecognized and untouched.

In another example, geneticists will point to overwhelming evidence that this particular raft of problems is inherited, as we see it passing down through generations, evidence that is bolstered by adoption and twin studies. In none of the studies consulted for this project is it acknowledged that the well-known confluence of this constellation with a propensity for substance abuse could masquerade as a genetic, but actually be a teratogenic, legacy. That is, a mother whose own mother was drinking is predisposed (by, for example, cognitive impairment) to the chain of decisions leading to her own alcohol-spiked pregnancy, which may in turn produce a child who (perhaps through poor impulse control) has a lot of unprotected sex while drinking with the mothers of his

future children, which then produces...and so on. It looks like our now-familiar co-occurring characteristics are inherited genetically in this family, and well they may – but to ignore altogether the possibility of alcohol-induced brain damage overlying this genetic contribution is akin to overlooking the elephant in the living room so fabled in chemical dependency lore.

Addiction studies is another field that generally ignores the possible contribution of fetal alcohol damage to its constituents in favor of the genetic and/or environmental explanations addiction. While the risk of alcoholism is undeniably transmitted genetically and environmentally, it also appears to be transmitted through other factors directly affecting the nervous system. One of these factors is outlined in the recent research surrounding dopamine-receptor deficiency (Blum, Cull, Braverman & Comings, 1996), which has been shown to be a powerful factor in the predisposition to addiction. This condition is associated with a particular mutated gene, with little attention to the cause of the mutation, and emphasis focused instead on its heritability. Although it has been demonstrated that exposure to prenatal alcohol can create this mutation in an otherwise normal animal, it is hardly ever asked in human research whether the alcoholic/addict subjects' mothers were drinking when they were pregnant.

If it weren't for the fact that hard-drinking (mother) mice in the laboratory will reliably produce the rodent version of learning disabilities, attentional problems, aggression, physical abnormalities and deficiencies in the dopamine-related pleasure/reward system, the question could remain forever in the mists of conjecture, and maybe should, given the abundance of accepted explanations already in place. But with hard evidence that prenatal alcohol a) causes the above-described damage, b) is predominant

in the populations currently suffering the heaviest burden of this damage, and c) is as yet largely unconsidered as the culprit, it seems important to investigate this factor as one possible neurological substrate over which the shifting layers of undeniable other factors—abandonment, continued parental abuse, possible inherited tendencies, poverty, racism—can then further eat away at this not-very-resilient child, adding to the frightening waves of unreachable, often dangerous children who, in the absence of adequate diagnosis and intervention, elude our best teaching and parenting efforts.

Three years ago I did an informal study to see if there were other Adult Children of Alcoholics besides myself whose issues included intransigent problems with learning, attention, judgment and assorted physiological vulnerabilities. I put an ad in the East Bay Express describing the core disabilities of fetal alcohol damage and inviting anybody who wanted to call me. In the 25 calls I received, 16 of them more or less fit the pattern, and every single one of these people had been working on these issues among others in therapy and/or ACA groups, believing that they would yield to insight, personal growth, working the steps or forgiveness. As one person put it, “I’ve been working in therapy for 15 years, and these are the issues (attentional, learning and judgment difficulties) that haven’t budged. It’s such a relief to think it might not be my fault for not working hard enough, that it might really be brain damage! I can DO something about plugging that up, like if I had epilepsy or something.”

#### A. Goals and Purpose of the Study

From the literature, informal study, personal and professional experience, it appears that there is a subset of ACAs whose problems may be at least exacerbated by brain damage due to prenatal alcohol exposure. It is the purpose of the present study to

examine the experience of ACAs through the neurological lens in order to identify this subset. The goal of this research is to expand the existing literature on Adult Children of Alcoholics, providing more specificity about the etiology of (and thus intervention with) the pattern of difficulties suffered by this population.

### B. The Research Question

The question under investigation in the present study was: Among a group of Adult Children of Alcoholics, is it possible to distinguish a subset whose problems are based in brain damage caused by prenatal alcohol exposure from those whose problems stem primarily from growing up in an alcoholic household?

This study limited the focus of inquiry to Adult Children of Alcoholics who are or have been either in therapy or Twelve-Step groups as this population is the most likely to be a) exposed to prenatal alcohol, b) aware of that fact, and c) interested in discussing the issues involved (see Appendix A, Initial Phone Contact). Six questionnaires (see Appendix B) were filled out by the 30 participants. Four of these questionnaires are existing FAS-related checklists, one is an adaptation of these pre-existing tools, and one is an ACA-related checklist.

### C. Significance of the Study

It has been conservatively estimated that 1 out of 100 people have some degree of brain damage from prenatal alcohol (Sampson, Streissguth, Bookstein, Little, Clarren, DeHaene, Hanson & Graham, 1997), yet the domains of science, education, treatment and personal awareness have woven entire fabrics of understanding and intervention without taking this factor into consideration. This gaping hole in our conceptual map perpetuates the fiction of deliberate, willful disobedience in people whose actions stem

from neurological impairment, resulting in societal responses that misfire, are woefully inadequate and often inhumane.

This study seeks to answer the question of whether, in the particular group of people who identify themselves as Adult Children of Alcoholics, it is possible to separate the brain-based components of their difficulties from the psychodynamic layer, with the intent to more appropriately address problems stemming from each. Using group and individual therapy to illuminate psychological reasons for the “passive-aggressive” dynamics underlying chronic procrastination or losing things, for example, when the real problem is faulty wiring in the memory department, will only result in increased frustration and self-hatred (often on both sides of the couch). When we are able to identify the organic components of ACA behavior and functioning, we will be able to focus in therapy on those layers that are amenable to insight and make accommodations for those that are not, in an elegant echo of the Serenity Prayer which asks for help with accepting “the things we cannot change, the courage to change the things we can, and the wisdom to know the difference.”

The mental health field in general may be similarly alerted by this research to the possibility that fetal alcohol exposure might be at least a contributing factor to client problems, so that conceptualizations may be more accurate and interventions more appropriate, avoiding the enormous waste of time and resources represented by misdirected attempts at psychotherapy in cases where subtle organicity is really the culprit behind many apparently functional difficulties.

Another aim of this study is that the various fields of research focusing on the co-occurring types of problematic behavior outlined above will come to consider the possibility of prenatal alcohol damage as a contributing factor.

The ultimate intent of this research is to raise public awareness of the spectrum of fetal alcohol damage. Health care workers, teachers, the criminal justice system, legislators, as well as the general public must be mobilized to prevent future prenatal alcohol damage and effectively deal with the damage that has already occurred.

## CHAPTER II.

### REVIEW OF THE LITERATURE

Adult Children of Alcoholics (ACAs) have been the subject of intensive research for the last two decades. That this population suffers difficulties as a result of their parents' drinking is generally established. Problems with interpersonal relations, academic and job performance, health, mood and self-concept have been documented in academic as well as popular literature. Exactly what aspect of parental drinking provokes these problems, and exactly which of the offspring of alcoholics suffers them, and what determines the severity, are among the important issues that remain unresolved.

Part of the reason for this lack of resolution is that researchers find different results. Some find pervasive pathology among children of alcoholics in general, some find scattered pathology, and others, finding little consistent pathology, warn of the dangers of overpathologizing what may not be such a big problem after all.

Most of the studies on children of alcoholics (COAs) have been done with children of drinking fathers; alcoholic women are harder to find. It is likely that many of these drinking fathers were accompanied in drink by their spouses, but whether or not the mothers drank is not usually discussed. The problems faced by COAs are therefore generally thought to stem from the dysfunction of families organized around the alcoholic father.

One possible explanation for the inconsistencies in research findings regarding frequency, degree and pattern of pathology in this population might be found in the drinking habits of the mothers of children of alcoholics. Since 1973 it has been increasingly well-documented that when a woman drinks during pregnancy, harm can

result to the fetus. Some of this harm gives rise to a set of problems that overlap the set of problems commonly thought to belong to children of alcoholics in general. Since maternal drinking is rarely accounted for in COA studies, it may be that this is a relatively simple explanation for the inconsistencies: the participants who suffer the most problems in living are the ones whose mothers drank the most during pregnancy.

The literature reviewed for the present study looks at the development of knowledge and theory regarding children of alcoholics, including the various explanations for why some are successful and some are not. Then a similar path is followed regarding Fetal Alcohol Syndrome, particularly the work on secondary disabilities, the outcomes that result from undiagnosed and untreated prenatal alcohol damage. The literature review ends with a summary of the similarities of these two groups and a statement of the aim of this study.

#### A. Adult Children of Alcoholics

This section of the literature review begins with the history of the movement surrounding children of alcoholics (COAs) and adult children of alcoholics (ACAs). The principles and main themes of this movement were first articulated by psychotherapists treating ACAs, and have come to be known as the “popular” view (Seefeldt & Lyon, 1994, p. 1028). Popular ACA literature has generally presented a unified opinion that widespread pathology results from the dynamics of parental alcohol abuse, particularly in the rigidity of family roles and rules. Next, the array of perspectives seen in academic research is demonstrated, ranging from the view that there is little damage to the view that only a small subgroup of ACAs who have suffered from their parents’ drinking, to the view that there is in fact ample evidence for a great deal of harm. Research on the

factors hypothesized to account for such harm is then explored, followed by research describing the differences between successful and unsuccessful ACAs. Fetal Alcohol Syndrome literature is re-introduced in review of an article that demonstrates the similarities between organic damage shown to result from prenatal alcohol exposure and psychodynamic damage thought to originate from inadequate alcoholic parenting.

### 1. History of COA, ACA Movement

Until the late 1970s and early 1980s, research on problems associated with alcohol has focused on the drinking individual: personality traits (Jones, 1968), etiology of the disease (Lisansky, 1960), and methods of treatment (Catanazaro, 1968) were the principle areas of study. Although issues relevant to families affected by alcoholism were addressed in Al-Anon beginning in the early 1970s (Al-Anon Family Group Headquarters, 1984), it wasn't until Margaret Cork wrote her book The Forgotten Children that problems of children of alcoholics began to rise to public attention. Cork's work is seen as the beginning of both the national social movement and the major research efforts surrounding this population (Brown, 1988, p. 13). The inclusion of adults who had grown up with alcoholic parents came to popular awareness when Newsweek (1979) reported on early research by Brown and Black describing adult children of alcoholics as a "new, unrecognized, legitimate population" (Brown, 1988, p. 13). The National Association for Children of Alcoholics was formed in 1983, and the Children of Alcoholics Foundation was started for the purpose of advancing research and awareness.

## 2. Popular Stance: “All...Are Affected”

One of the first people to categorize the problems of ACAs was Janet Woititz (1983), who used her experience as a group therapist. Her Laundry List crystallizing this set of problems has become an official part of the ACA 12-Step folklore, and is as follows:

1. Adult children of alcoholics guess at what normal behavior is.
2. Adult children of alcoholics have difficulty following a project through from beginning to end.
3. Adult children of alcoholics lie when it would be just as easy to tell the truth.
4. Adult children of alcoholics judge themselves without mercy.
5. Adult children of alcoholics have difficulty having fun.
6. Adult children of alcoholics take themselves very seriously.
7. Adult children of alcoholics have difficulty with intimate relationships.
8. Adult children of alcoholics overreact to changes over which they have no control.
9. Adult children of alcoholics constantly seek approval and affirmation.
10. Adult children of alcoholics usually feel that they are different from other people.
11. Adult children of alcoholics are super responsible or super irresponsible.
12. Adult children of alcoholics are extremely loyal, even in the face of evidence that the loyalty is undeserved.
13. Adult children of alcoholics are impulsive. They tend to lock themselves into a course of action without giving serious consideration to alternative behaviors or possible consequences. This impulsivity leads to confusion, self-loathing, and loss of control over their environment. In addition, they spend an excessive amount of energy cleaning up the mess.

Diane Malbin (personal communication, 1996) adds these:

14. Lock themselves into a course of action without giving serious consideration to alternative behaviors or possible consequences.
15. Seek or create tension and crisis and then complain about the results.
16. Avoid conflict or aggravate it; rarely deal with it.
17. Fear rejection and abandonment, yet reject others.
18. Fear failure, but sabotage successes.
19. Fear criticism and judgment, yet criticize and judge others
20. Manage time poorly and can't prioritize.
21. Have difficulty with communication; may demonstrate flat affect, excessive anger, or be people pleasing.
22. May have difficulty concentrating.
23. Engage in black or white, all or nothing thinking.

24. Tend to live life from the viewpoint of a victim, continue this pattern.  
(Victims victimize.)
25. Tend to react rather than respond.
26. Have low self-esteem.
27. Become compulsive.

Another author-therapist in the ACA field, Wayne Kritsberg, breaks down ACA characteristics into four main categories in his The Adult Children of Alcoholics Syndrome (1985): emotional, mental, physical and behavioral. In the emotional category are: fear, anger, hurt, resentment, distrust, loneliness, sadness, shame, guilt and numbness. In the mental category: thinking in absolutes, lack of information, compulsive thinking, indecision, learning disabilities, confusion and hypervigilance. Physical characteristics include: tense shoulders, lower back pain, sexual dysfunction, gastrointestinal disorders, stress-related behaviors and allergies. Behavioral characteristics include: crisis-oriented living, manipulative behavior, intimacy problems, inability to have fun, trying to fit in, compulsive-addictive disorders.

A significant piece of the conventional wisdom in ACA thinking was formalized by Claudia Black (1981) with The Rules of the alcoholic family; these rules have become an integral part of workshops, articles, therapy and conversation focusing on ACA issues. The rules she named were: Don't Feel, Don't Trust, Don't Talk, principles that carried through into adult life from childhood in the alcoholic family, keeping a sense of isolation, numbness and unreality in place. Therapy that addresses the function of these principles in intimate relationships must, many feel, trace these roots down to the early traumatic experience that led to their becoming standard operating procedure in the present (Cermak, 1991; Black, 1991, 1999; Brown, 1991; Kritsberg, 1985).

Sharon Wegscheider-Cruse (1981) described the by-now equally ubiquitous “Hero, Scapegoat, Lost Child and Mascot” roles taken on by children growing up in alcoholic families. These roles were thought to be largely a function of birth order, where the first child, the Hero, took on the responsibilities of the household and often succeeded in school, making everything “look good;” the second, the Scapegoat, acted out to get attention, frequently getting into trouble with school and then the law; and drawing to him- or herself all the negative feelings present in the family; the third, the Lost Child, withdrew, becoming depressed and not noticed in class, no visible wants or needs to bother the rest of this family; and the fourth, the Mascot, became a jokester, with attempts to make the situation go away by chattering and being silly. In this researcher’s professional experience with ACA groups, in most families these roles are not so neatly fixed, especially since most families do not have four children – roles often switch or double up – yet many ACAs report living out a particular role in their families and on into adulthood.

The stance of most of the popular movement surrounding children of alcoholics is that, as Claudia Black stated, “ALL CHILDREN (OF ALCOHOLICS) ARE AFFECTED; ALL NEED HELP” [capitalization in the original] (1981). Many academic researchers have taken the same stance: Pilat and Jones (1984/5), for example, explain the existence of those children who seem to do well: “...they may repress their feelings, and it is only later in their adult relationships that the scars of survival from their childhood may appear. In addition, they are at risk for the development of their own drinking and chemical abuse problems.” M. E. Chafetz, H. T. Blane, and M. J. Hill, in their study of COAs who had been referred to a Child Guidance Clinic, said: “there are

distinct and deleterious social consequences to being the child of an alcoholic parent” (1971, p. 688). In a study of children whose parents were in treatment for alcoholism, the researchers arrived at the same conclusion, but with particular concern stemming from the fact that these troubled children were only identified by chance, and that there must be many other very troubled children of alcoholics who go unidentified and untreated.

F. W. Fine, L. W. Yudin, J. D. Holmes and S. Heinemann have written:

The present study suggests...that the extent of pathology in these children is even greater than previously described. Despite, in some cases, extremely marked behavioral disturbances, none of these children had been referred for help. This might be related to the severe degree of disruption, alienation, and confusion in alcoholic families that allows a child to continue to behave pathologically with no attempt to seek help. (Fine et al., 1976)

### 3. Academic Research is Divided

Growing up in an alcoholic family is not inevitably deleterious, however, according to many other researchers. There is still controversy, with one end of the spectrum arguing that the idea of damage from alcoholic parents is vastly overinflated, with the correlative opinion that pathologizing all children of alcoholics actually causes harm where there wasn't any to begin with (Hunt, 1999; Seefeldt & Lyon, 1994). The middle of the spectrum allows that there do exist certain subgroups of COAs with some limited signs of damage (D'Andrea, 1994; Chassin et al., 1999), and the far end of this spectrum demonstrates accumulated evidence pointing to a wide array of damage stemming from any combination of the effects of having alcoholic parents (Weinberg, 1997).

Seefeldt and Lyon represent the end of the spectrum least likely to agree with the popular literature; in fact, they have taken on Woititz' (1983) “laundry list” and demonstrated (by checklist) that their group of 253 adult children of alcoholics is no more

likely than the average person to conform to this characterization. In another study of people who sought treatment (Seefeldt & Lyon, 1992), the presenting problems were dismissed as “largely revolving around relationships with adult family members...” The “responders indicated that other areas of personal, social, and occupational functioning were relatively unaffected by their parents having been alcoholic.” The authors take up the cause of “these individuals (who) are shepherded into treatment simply because their parents were alcoholic, despite the fact they seem to function well as measured by almost any standard.”

Another study that found little evidence for different outcomes between COAs and non-COAs was done by Marilyn Hunt with students at California State University at San Bernardino (1999). Thirty-one COAs and 120 non-COAs were given the MMPI and the CPI, with nonsignificant results. Such lack of evidence for effects may be more likely when four-year college students are being studied, as the more impaired individuals may have already been screened out by the academic demands of such a setting.

One example of a study that found evidence for a subgroup of ACA-related pathology was done by D’Andrea (1994) at the University of Nevada, using a design similar to Hunt’s. She gave 97 ACAs the CPI, did a cluster analysis, and discovered three distinct subgroups in overall CPI functioning: 44% above the mean, 40% hovering around the mean, and 16% considerably below the mean. Inquiry into the characteristics defining each of these subgroups was beyond the scope of her study.

Laurie Chassin’s group’s study (Chassin, Pitts, DeLucia & Todd, 1999) is another example where a small subgroup is identified among a larger population of adolescent children of alcoholics. She found that the adolescents who used “externalizing”

behaviors were more likely to be those who suffered most from their parents' alcoholism. (This study will be reviewed in detail in the section on Explanatory Factors).

The other extreme of this continuum acknowledging the damaging effects of parental alcoholism is represented by Naimah Z. Weinberg, M.D., a Special Expert in the Division of Epidemiology and Prevention Research at the National Institute on Drug Abuse. Dr. Weinberg's review and synthesis of the scientific literature on cognitive and behavioral deficits associated with parental alcohol use was published in the Journal of the American Academy of Child and Adolescent Psychiatry in September 1997. He compared the research on prenatal exposure to alcohol with the research on children of alcoholics, and found the pathologies studied in each to be strikingly similar to each other. Among his conclusions:

More children may be affected by parental alcohol use than are recognized by clinical, educational, and social institutions. They may, in fact, represent significant subgroups of children with ADHD, learning disabilities, language disorders, and other psychiatric conditions, for whom conventional treatments may be ineffective and may need to be altered. (Further review below.)

Echoing Weinberg's urgent call to understand and address the widespread damage of parental alcohol abuse, Burk and Sher (1988) put it this way: "Without a comprehensive understanding of the pathogenesis of morbid sequelae to parental alcoholism, treatment and prevention programs may provide less-than-optimal (and occasionally harmful) services."

#### 4. Explanatory Factors for Pathology

Enoch Gordis, the Director of the National Institute on Alcohol Abuse and Alcoholism, took a more cautious stance, hinting at a mountain-out-of-a-molehill position

on the part of those who saw widespread dysfunction, and implying that any problems that did exist may have been a result of poor sampling:

Although there is a genetic component to the vulnerability of alcoholism, COA issues are not related primarily to alcoholism itself but to the social and psychological dysfunction that may result from growing up in an alcoholic home...Selection bias means that conclusions based on clinical samples are likely to overestimate the extent of the problems, because only the most troubled come for treatment. (Gordis, 1990)

Certainly family dynamics play a major role in the dysfunction suffered by ACAs. The layers of inadequate, chaotic, sometimes violent parenting, leave scars that can last a lifetime unless worked through in therapy or some other healing medium (Brown, 1999, pp. 6-13). Stephanie Brown, a foremost author in the field of family issues in alcoholism, imputes all of the problems suffered by ACAs to such familial layers, even those directly reflective of organic conditions:

Many of these individuals (ACAs) suffer a variety of problems related to the alcoholism of a parent that was never labeled as such, including school phobia, *learning disabilities*, *attentional disorder*, depression, anxiety, and mood disturbance. (p. 11)

Her explanation for this variety of problems is entirely psychodynamic:

Attachment – early and ongoing – is based on denial of perception which results in denial of affect which together result in developmental arrests or difficulties. The core beliefs and patterns of behavior formed to sustain attachment and denial within the family then structure subsequent development of the self including cognitive, affective and social development. (p. 5)

Brown's work is one of the major sources of theory and practice regarding Adult Children of Alcoholics, laying out the various effects of parental alcoholism with more detail, depth and theory than any other source found in an exhaustive literature review for this study. Yet she is unremarkable among the vast majority of her peers in her

assumption that all problems of ACAs (except for the heritable predisposition to alcoholism itself) have their roots in the postnatal environment.

Brown acknowledges that alcoholism is transmitted from one generation to the next, as established through genetic research using twin, adoption, and half-sibling studies (Schuckitt, Goodwin & Winokur, 1972; Goodwin, 1971, 1979; Goodwin, Schulsinger, Knop, Mednick & Guze, 1977). Goodwin (1979), for example, studied Danish non-family adoptees, matched to two control groups, and demonstrated that children of alcoholics are from four to six times more likely to become alcoholic than people whose parents are not alcoholic. Russell, Henderson and Blume (1985) have identified genetically influenced biological markers related to alcoholism. Nevertheless, Brown insists on “social learning, modeling, and the psychological process of imitation and identification” (1988, p. 15) as the principal components of the familial legacy of alcoholism, giving little attention to possible biological factors.

The environment of the alcoholic family as Brown outlines it (1988, pp. 47-57) is governed by the following themes:

- Control—the false belief in the efficacy of managing the other’s responses
- Chaos—things are or will be out of control
- Inconsistency—between stated values and actual practice, between one set of standards and another, between perception (only certain perceptions permitted) and reality
- Roles—parenting the parent
- Tension—chronic, with projected feelings of hostility
- Shame—in parents, projected onto children

It is Brown’s contention that in order to bring sanity into the lives of individuals affected by these dynamics, a process of recovery is required. If it is a family that is to be in recovery, the family must go through a process similar to that of the individual alcoholic recovering, beginning with an experience of loss of control. (How does it do

that??) This widening of the rips – the loss of control – in the family fabric goes against the view of many family therapists who strengthen the defenses, but in the field of recovery from addictions appears to be necessary for true healing to take place (Brown, 1999, pp. 156-168).

Treatment of alcoholic family dysfunction has been postulated as a possible curative factor for the problems of children of alcoholics by several authors in addition to Brown (Moos & Billings, 1982; Wilson & Orford, 1978). An overview of studies of such treatment was conducted by O'Farrell and Feehan (1999), who concluded that several areas of functioning improved: reduced family stressors; improved marital adjustment; reduced domestic violence and verbal conflict; reduced risk of separation and divorce; improvement in important family processes related to cohesion, conflict and caring; and reduced emotional distress in spouses. The authors note that while these factors have been linked with child mental health and psychosocial functioning in more general child development and psychopathology studies, further research is needed to find out whether such family improvements can lead to similarly positive outcomes for children in alcoholic families.

A wide range of factors in addition to dysfunctional family dynamics have been explored as explanations of the problems suffered by children of alcoholics. Individual parental characteristics such as parental affective disorders (Schuckitt, 1978) were shown to contribute to children's depression or anxiety, and divorce or other undesirable life changes (Moos & Billings, 1982) were related to increased difficulty in school and social relations among children of alcoholics. Zucker, Ellis, Bingham and Fitzgerald (1996) found that parental antisociality greatly affects the risk of future alcoholism in COAs.

Others, taking the more holistic view, acknowledge the layers of problems additively contributing to the difficulties of children of alcoholics (Brown, personal communication, 2000).

##### 5. Difference Between Successful and Unsuccessful ACAs

It would be foolish to try to deny the influences of genetics, family dynamics, or any other logical contributor to the problems faced by ACAs. That this “elephant in the living room” is huge and many-faceted is acknowledged by nearly all. What is disturbing is when the literature purports to explain some differentiating factor between successful and non-successful ACAs, but actually leaves out an essential link in the chain without seeming to notice.

For example, in a study designed to illuminate the differences between children of alcoholics who developed serious problems and those who did not, Emmy Werner (1986) showed that although 41% of the children of alcoholics she worked with in Kauai developed serious coping problems by 18 years of age, 59% went on to enjoy a life similar to non-COAs. She identified these “resilient” children as having a set of characteristics in common: the ability to obtain positive attention from other people, adequate communication skills, average intelligence, a caring attitude, a desire to achieve, and a belief in self-help. She did not question the source of such characteristics or the absence thereof, implying that it was somehow just the luck of the draw. As it happens, these characteristics make up approximately the same set as the ones that are extinguished with prenatal alcohol damage (Streissguth, 1988).

Another example of research that leaves an important – and similar – gap but doesn’t seem to recognize it is the previously mentioned study by Chassin, Pitts, DeLucia

and Todd at Arizona State University in 1999. This longitudinal study used 246 adolescents with at least one biological alcoholic parent, matched with 208 demographically matched adolescents with no alcoholic biological or custodial parents, and measured the relationship between parental alcoholism and offspring depression, anxiety and alcohol/drug use. The study found that the parent's alcoholism is more likely to have a detrimental effect the more the adolescent uses "externalizing" behaviors (from the Child Behavior Checklist) such as physical or verbal aggression, stealing, bullying and the like. The question as to how such behavior got there in the first place is simply missing. This is the flip side of the resiliency research, where the characteristics missing from a prenatally-exposed child's array are precisely those that determine success in life: in the case of the Chassin study, the characteristics typically present in the prenatally-exposed child are exactly those that predict the diagnoses reported

Richard Rose (1998) does tackle the issue of etiology of the externalizing behavior exhibited by children of alcoholics who have a higher risk of future alcoholism themselves. His thesis is that the behavioral precursors of alcoholism can be seen in very young children, and that the field would do well to understand the origin of these behaviors. The behavior he describes is typical of attention deficit disorder (although he never names it such): "novelty-seeking, impulsive, exploratory, excitable, curious, distractible and hyperactive" (pp. 132, 134). In his Developmental Behavior-Genetic perspective, twin studies that demonstrate a higher incidence of this behavior in monozygotic twins seem to be proof enough to Rose that genetics are the principle factor at work, ameliorated by peer and other environmental influences in only one variable: the age of first drinking. Results from his study "...strongly support the heritability of the

risk-relevant behaviors assessed” (Rose et al., 1997a). Rose does not consider the variable that can insert itself between DNA transmission and postnatal environment – the variable of fetal alcohol effects – even though at least some of his 2800 twin-pairs’ mothers are likely to have been drinking during pregnancy, and attention deficit disorder is one umbrella term for the effects of such drinking.

Rose names one likely genetic culprit in a sidebar to his article, entitled “Molecular Genetics of Risk-Relevant Behaviors,” saying:

If certain behaviors associated with increased alcoholism risk are heritable, then specific genes must be responsible for eliciting and controlling those behaviors and, by extension, alcoholism risk...One such neurotransmitter is dopamine...involved in eliciting the rewarding effects of alcohol and other drugs (and)...may also underlie novelty-seeking behaviors.

Rose easily assumes heritability as his major premise, without regard for evidence for another pathway from alcoholic parents to impaired dopamine activity in their children. He is joined in his enthusiasm for the dopamine factor by the NACoA Network (Summer 1999), the official publication of the National Association for Children of Alcoholics:

Scientists at Johns Hopkins report that some children of alcoholics have an altered brain chemistry that makes them more vulnerable to addiction. The researchers found that natural opioid activity in the brain is significantly lower in young adults who have a strong family history of chronic drinking. “This single difference in opioid activity may make people more vulnerable to alcoholism for two reasons...it alters the brain’s reward/craving pathway and it also changes the brain’s response to stress. And we know that stress is involved in many kinds of drug-seeking behavior.” The study was reported in the December (1999) issue of the Annals of General Psychiatry.

In a study that tested an alternative hypothesis to the genetic one regarding dopamine, Dr. Laura Lucchi, in the Department of Pharmacology in Milan, Italy, showed that a single dose (comparable to a single alcoholic beverage) of alcohol given to 10 rats

dramatically reduced the levels of striatal dopaminergic receptors in their offspring (Lucchi, 1984). This is clear (and longstanding) evidence that intra-uterine alcohol affects the reward system (the endorphin/dopamine chain) directly, yet such evidence is not included in the connections made between alcoholic parents and vulnerability to stress and substance abuse in the children, even in the connections made between dopamine and psychopathology in the offspring of alcoholics.

It isn't that the effects of prenatal alcohol have been entirely missing from ACA literature. From early days until quite recently, FAS was thought to be a self-contained entity, just one of the many results of maternal drinking. Karen Scheitlin, a nurse practitioner, published a table of "Characteristics of COAs" (1990), with physical, emotional and social categories. FAS is one of 43 characteristics named, surrounded by other characteristics such as hyperactivity, asthma, tics, poor grades, "tendency toward disturbances in personality and emotional upset," and chemical dependency, all of which are known to be characteristic of prenatal alcohol damage (among other causes).

#### 6. Similarities Between Symptoms of FAS and ACAs

The only person in academic or popular literature who seems to have made the connection between the myriad problems of the ACA and prenatal drinking, however, is Naimah Z. Weinberg, briefly mentioned above. In his review of the literature in both fields, Weinberg arrays the effects of prenatal alcohol exposure alongside the effects of having alcoholic parents, and finds a whole web of similarities in the cognitive and behavioral areas.

In prenatally-exposed children, a "pattern of non-verbal learning disabilities, with poor spatial organization and math skills, memory, and problem-solving" was found

(Streissguth et al., 1994). In a “case-controlled study...children identified as learning disabled were more than seven times as likely to show the physical stigmata of prenatal alcohol exposure” (as children not receiving services for learning disabilities) (Marino, Scholl, Karp & Yanoff, 1987). In studies of COAs, deficits were found including “weakness in verbal ability and abstraction/conceptual reasoning in the presence of normal range IQ” (Tarter, Hegedus, Goldstein & Alterman, 1984); as well as “visual-spatial skills, perceptual-motor skills, and auditory-visual attention span” (Pihl, Peterson & Finn, 1990).

Weinberg begins this section: “Studies on PNE (prenatally-exposed) children and on COAs note patterns of difficulties resembling deficits which accompany frontal lobe damage.” “Impaired judgment and poor adaptive behavior...failure to learn from experience, affecting social, moral and academic functioning is often noted (with PNE children)...Impulsivity, impairments in regulation of social behavior, and poor emotional modulation are noted (with COAs).” Weinberg goes on to cite several studies identifying attention deficit disorder in both populations (1997, p. 1182).

Weinberg is unique in his perspective. Even in the literature addressing intervention with children of drug- and alcohol-abusing parents, social service agencies leave out references to the effects of prenatal alcohol. For example, despite its own publication of a wonderful book on FAS (McCreight, 1998) in the same year, the Child Welfare League of America’s tome for social service agencies, Responding to Alcohol and Other Drug Problems in Child Welfare: Weaving Together Practice and Policy does not even mention Fetal Alcohol Syndrome and Effects. In another example, one of the originators of the ACA movement, Claudia Black, has written a book about “dual

dynamics within the chemically dependent home” (1990) with no references to prenatal alcohol effects – clearly a source of many of the diagnoses she mentions (Streissguth, 1988).

In a study quoted in the NACoA Network (Summer 1999), it is flatly and simply stated that: “Children of alcoholics are more likely to have attention-deficit hyperactivity disorders, conduct disorders and anxiety disorders, according to a report in the June issue of the Journal of the American Academy of Child and Adolescent Psychiatry.” The article goes on to discuss treatment implications, focusing on counseling for both the parent and the child, advising further study to determine if such treatment can help reduce the risk of later psychiatric problems. This announcement gives up altogether on etiology, lumping together the clearly organic disorder of ADHD with the often secondary disorders of conduct and anxiety problems, ignoring the huge body of evidence that all three of these disorders appear in abundance in children who were prenatally exposed to alcohol. Ignored as well are the related findings that early diagnosis of FAS/FAE is the single most effective intervention for such disorders, and that counseling in the absence of diagnosis is considered ineffective (Streissguth, 1997).

#### 7. Summary of ACA-Related Literature

This section has reviewed the literature of the popular ACA movement, with its blanket assertions of widespread damage and formulaic conceptualization of how that damage is expressed in daily living. The range of academic research has been outlined, from a near-total denial of damage at all through the postulation of small subgroups to evidence for a great deal of damage. Explanations for this damage were reviewed, with an in-depth look at the family influence, as was research on possible differentiating

factors between successful and unsuccessful children of alcoholics. The destructive potential of prenatal alcohol exposure was revisited in a review of an article summarizing the similarities between problems attributed to children of alcoholics and those attributed to FAS. Related writings that showed particularly striking omissions of the mention of FAS were cited and described.

## B. Fetal Alcohol Syndrome

### 1. History of FAS

This section will review the literature on the effects of prenatal exposure to alcohol, beginning with ancient awareness of these effects, touching on the more recent period of denial that there was harmful potential, and then covering the principal research beginning in the early 1970s when Fetal Alcohol Syndrome (FAS) was first recognized by the scientific world. Research on the primary and secondary disabilities of FAS is described, along with the significant move to expand the definition to include Fetal Alcohol Effects (FAE).

Damage to children from prenatal alcohol has been noted since earliest times: in ancient Carthage, there was a ritual forbidding the drinking of wine by the bridal couple so that defective children would not be conceived; Aristotle stated that “Foolish, drunken and harebrained women most often bring forth children like unto themselves, morose and languid.” In the Bible, Judges 13:7 says: “Behold, thou shalt conceive and bear a son: and now drink no wine or strong drinks.” In Great Britain, having experienced a wave of alcoholic drinking during the “Gin Lane” epidemic, the College of Physicians reported to the British Parliament in 1726 that “Parental drinking is a cause of weak, feeble, and distempered children;” a member of the British House of Commons in 1834 stated that

“Infants born to alcoholic mothers sometimes had a starved, shriveled, and imperfect look.” (Historical citations from Jones & Smith, 1973, p. 15.)

Far from a universally held opinion, however, the idea that drinking could harm the fetus has met with controversy. In 1955, the Yale Center for Alcohol put out a brochure with the statement that the “...old notions about children of drunken parents being born defective can be cast aside, together with the idea that alcohol can directly irritate and injure the sex glands” (as cited in Rosett, 1976). Ashley Montagu echoed this sentiment in 1965 when he said, “It can now be categorically stated, after hundreds of studies covering many years, that no matter how great the amounts of alcohol taken by the mother – or the father for that matter – neither the germ cells nor the development of the child will be affected” (p. 114).

## 2. Recent Understanding

At the present time, there is a vast and growing body of scientific evidence that prenatal alcohol damages the fetus. The current understanding began to unfold in 1968 when Dr. Paul Lemoine and co-workers in Nantes, France described 127 children born to alcoholic mothers. The pattern these children shared included consistent physical anomalies, small size and ceaseless agitation (Lemoine, Harousseau, Borteyru & Menuet, 1968). At the same time, during a study of failure to thrive infants in Seattle, only one common factor could be found: each of the babies had an alcoholic mother (Ulleland, 1972; Ulleland, Wennberg, Igo & Smith, 1970). It was 1973 when worldwide attention came to this birth defect through an article in Lancet which carefully described the constellation of physical features (a consistent pattern of widely-spaced eyes, small palpebral fissure, flat philtrum, small chin, thin upper lip and small overall head

circumference) growth deficiency and intellectual, motor and adaptive impairments of what was then christened “fetal alcohol syndrome” (Jones, Smith, Ulleland & Streissguth, 1973). By 1978, 245 people with FAS had been identified, the “FAS face” widely considered to be uniquely alcohol-related, and prenatal alcohol described as the most frequent known cause of mental retardation (Streissguth, Herman & Smith, 1978).

### 3. Fetal Alcohol Effects

The facial anomalies and small size were essential to the diagnosis of FAS for many years; the three-part definition remaining the same as originally proposed by Jones and Smith (1973): (1) pre- and/or postnatal growth deficiency, (2) a distinct pattern of craniofacial malformations, and (3) central nervous system (CNS) dysfunction.

However, the effects of alcohol can be detected in CNS dysfunction even in the absence of any physical abnormalities, as studies with young rats suggested (Riley, Lochry & Shapiro, 1979). As it became clear that many prenatally-exposed people experienced problems in living and had little or none of the telltale physical phenomena, the diagnostic category of FAS opened up to a wider range of effects, first termed Fetal Alcohol Effects (FAE) in 1978 (Hanson, Streissguth & Smith, 1978), or Possible Fetal Alcohol Effects (PFAE) (Clarren & Smith, 1978). Problems noted with these terms center around the fact that there were no specific criteria for FAE, and it is not officially recognized as a medical diagnosis. Nevertheless, FAE has become the term of choice in the lay and professional populations to describe people with few-to-no physical abnormalities but a wide variety of neurologically-based difficulties stemming from prenatal alcohol exposure.

In 1996, the Institute of Medicine (IOM) proposed five graduated categories of prenatal alcohol damage. A diagnosis of “FAS” requires the full array of facial features along with small size and CNS impairment as listed in the paragraph below, with Categories 1 and 2 of FAS referring to the presence or absence of confirmed maternal alcohol consumption; “partial FAS” (Category 3) includes confirmed exposure to alcohol in utero, some components of the characteristic facial abnormalities and one of the following conditions:

1) growth retardation; 2) evidence of CNS neurodevelopmental abnormalities (including decreased cranial size at birth, structural brain abnormalities, including microcephaly, and neurological hard or soft signs, such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination); or 3) evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher-level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment. (p. 4)

The IOM also suggests two additional diagnostic categories, both of which require documented maternal alcohol ingestion. The first is “alcohol-related birth defects (ARBD)” (Category 4), and refers to physical, congenital anomalies. The other, “alcohol-related neurodevelopmental disorder” includes criteria 2 and 3 from “partial FAS,” listed above. The present study, following the example of Ann Streissguth as well as other major researchers and the vast preponderance of references, will use the terms FAS and FAE, which includes IOM categories 3 and 5, “partial FAS” and “ARND” (Streissguth, 1997). All categories taken together, it is estimated that nearly 1 out of 100 people have some degree of brain damage from prenatal alcohol exposure (Sampson, Streissguth, Bookstein, Little, Clarren, DeHaene, Hanson & Graham, 1997).

#### 4. Secondary Disabilities

The largest, most comprehensive study to date on FAS was published in 1996 by Ann Streissguth in Seattle, with 661 subjects diagnosed by dysmorphologists at the University of Washington. Funded by the Centers for Disease Control, the purpose of this study was to "...build a prevention information base fundamental to the amelioration of secondary disabilities in patients with FAS and FAE" (Streissguth et al., 1996).

The three main goals of the study were: 1) to document the occurrence and range of secondary disabilities associated with FAS and FAE; 2) to determine risk and protective factors associated with these secondary disabilities; and 3) to develop a Fetal Alcohol Behavior Scale to identify patients with probable FAS/FAE.

The study first outlined and measured the primary disabilities of FAS/FAE, the consistent (but not exclusive) array of cognitive, behavioral and emotional difficulties that beset this population. Research with alcohol-exposed laboratory animals is cited, yielding correlations between learning impairments and hippocampal dendritic impairments (Abel, Jacobsen & Sherwin, 1983); olfactory bulb decrease and odor-associative learning (Barron & Riley, 1992); optic nerve hypoplasia with impaired vision (Stromland & Pinazo-Duran, 1994); reduction in cerebellar size with impaired motor development and ability (Meyer, Kotch & Riley, 1990a, b); decreased serotonin synthesis with impairments in instinctive maternal behaviors (Hard et al., 1985); and decreased callosal size with hyperactivity (Zimmerberg & Mickus, 1990).

Next, research regarding neuropsychological impairments in humans using neuropsychological assessment tools and brain imaging techniques is reviewed. Studies cited suggest impairments in the following areas: auditory recall, intrusive errors, spatial

learning, attentional disorders, perseveration and response inhibition. Sensorineural, conductive and central hearing impairments were also found in the studies reviewed, as well as “constructional apraxia,” having to do with reconstructing images from visual memory. Streissguth also cites studies focusing on executive functioning as the center of difficulties for people with FAS/FAE: forming, planning and carrying out goal-directed behaviors.

Because Streissguth and her colleagues did not have the capacity to measure brain structure and functioning on such a large number of subjects, psychological test scores were used “as surrogate measures of ...primary disabilities.” These measures included intelligence tests, achievement tests, tests of adaptive behavioral functioning, and behavioral observations. She found the most widespread primary disability in the area of IQ: the mean IQ for FAS was 79, with a range from 20 to 120; the mean for FAE was 90, with a range from 49 to 142. Other primary disabilities found fell under three broad categories and several specific ones. The three broad categories were attention deficits (80%), memory problems (73%) and hyperactivity (72%). In decreasing order of frequency, the following more specific patterns were identified:

Expresses thoughts that are not sensible	38%
Rocks back and forth, sitting or standing	33%
Is unaware of what is happening in immediate surroundings	27%
Extremely peculiar mannerisms or habits	15%
Exhibits tics	15%
Excessive preoccupation with objects or activities	15%
Self-injurious behavior	15%
Seizure disorders	10%

The construct of secondary disabilities emerged as the researchers in Streissguth’s group (by then known as the Fetal Alcohol and Drug Unit at the University of Washington) did follow-up in several longitudinal studies (Streissguth, Clarren & Jones,

1985; LaDue, Streissguth & Randels, 1992; Streissguth, Aase et al., 1991; Streissguth, Randels & Smith, 1991). Around the age of 12 years, individuals seemed to encounter problems at home and at school beyond what the cognitive impairments alone would have predicted. In 1992, the Centers for Disease Control and Prevention funded a major study (culminating in the research reviewed in this section, published in 1996) to focus on this subject, examining various aspects of problematic development that appeared as people with FAS/FAE matured out of childhood.

The secondary disabilities studied included disrupted school experiences, problems with alcohol and other drug abuse, irresponsible parenting, joblessness, homelessness, mental health problems, victimization, trouble with the law, and premature death. This four-year study involved 415 people from 6 to 51 years of age, and used two data collection instruments: the Fetal Alcohol Behavior Scale (FABS) (Streissguth, Bookstein, Barr & Press, 1996) and a life history interview (LHI) with primary caregivers. The FABS was constructed to reflect the behavioral phenotype of typical fetal alcohol behaviors. The LHI, a 70-minute, 37-page comprehensive structured interview, compiled information on “10 major areas of possible long-term functional covariates or consequences characteristic of patients diagnosed with FAS/FAE”:

- 1) household and family environment
- 2) independent living and financial management
- 3) education
- 4) employment
- 5) physical abuse, sexual abuse and domestic violence
- 6) physical, social and sexual development
- 7) behavior management and mental health issues
- 8) alcohol and drug use
- 9) legal status and criminal justice involvement
- 10) companionship and parenting

The LHI looked at these areas of concern in terms of past and present patient status, secondary disabilities, and possible risk and protective factors. Six main secondary disabilities were identified, with the most prevalent being **mental health problems**: 90% of the individuals in the study had experienced some difficulty with mental health, and 80% had received treatment. The most common problem for adults was depression; the most common for children and adolescents was attentional deficits (61%). **Disrupted school experience** had plagued 60% of the adults and adolescents, stemming from attentional problems and repeatedly incomplete schoolwork. Behavior problems in school fell into the categories of not getting along with peers (60%) and being repeatedly disruptive in class (55%-60%). Sixty percent of the adolescents and 14% of the children had had **trouble with the law**, with shoplifting and theft as the most frequent type of crime. Fifty percent of adolescents and adults had been **confined**, either in mental health programs, inpatient drug and alcohol treatment or jail. Forty-nine percent of adolescents and adults and 39% of children had displayed **inappropriate sexual behavior**. Thirty-five percent of adolescents and adults had experienced **drug or alcohol** problems; many patients were abstinent due to “no access to alcohol” or “against the patient’s beliefs.” Of people who were at least 21 years old, two additional secondary disabilities were noted: **Dependent living** characterized 80% of the sample, and 80% had **problems with employment**.

Streissguth et al.’s explanation for these high levels of secondary disabilities found in people with FAS/FAE is that the primary disabilities of permanent organic brain damage are hidden, leading schools, families, the justice system and society at large to expect normal behavior and reasoning. Without a low IQ score, obvious mental illness or

physical signs of birth defect, societal protection is lacking, and blame or punishment is all too often the only response.

This research also examined risk and protective factors associated with secondary disabilities. Risk factors were those that were most associated with elevated rates of secondary disabilities; protective factors with lower rates. Protective factors included:

- 1) living in a stable and nurturing home of good quality
- 2) not having frequent changes of household
- 3) not being a victim of violence
- 4) having received developmental disabilities services
- 5) having been diagnosed before the age of 6
- 6) having a diagnosis of FAS rather than FAE
- 7) having an IQ score below 70

Streissguth's primary conclusion from this study was a strong recommendation that early diagnosis be made available wherever warranted, so that support services could be mobilized, appropriate educational and parenting practices could be implemented, and self-image could be enhanced rather than continually eroded.

This study is one of the most frequently cited in subsequent literature regarding FAS, and the Streissguth team the most prolific of authors. With this comprehensive, in-depth look at the core disabilities of FAS, the secondary disabilities resulting from inappropriate treatment, and the factors that distinguish appropriate from inappropriate response, the field gained immeasurably in understanding this crippling condition. Missing from this study, however, are three elements that may have contributed to the dysfunctional behavior evidenced by the subjects: family dynamics including continuing alcoholism, inherited traits and the possible presence of other drugs in utero. The family dynamic aspect is addressed peripherally by the protective factor of "living in a stable and nurturant home of good quality," but the scope of this study did not allow for a closer

look at the effects of varying parental characteristics. Genetics is untouched in Streissguth's work. The issue of other drugs is partially addressed in the literature review, which includes evidence from studies conducted with laboratory animals whose only intoxicant was alcohol, but it is not stated anywhere in the Streissguth material whether or not the subjects of her study had been exposed to other drugs as well. (While the "crack baby" scare of the 1980s has been shown to be a tremendous overreaction, there does exist some evidence for developmental effects from cocaine and other drugs.) (Chasnoff, 1998)

#### 5. Summary of FAS-Related Literature

Although the harmful effects of in-utero alcohol exposure have been known for thousands of years, only recently has this damage come under scientific scrutiny. This section reviewed the research on the cognitive, behavioral, emotional and physical effects of prenatal drinking, with emphasis on the largest, most-cited study to date. Research estimating the prevalence of these effects at just under 1 person in every 100 people was cited. The research detailing the secondary disabilities that result from undiagnosed FAS/FAE was reviewed in depth.

#### C. Summary of Literature Review

Comparing the two bodies of information regarding Fetal Alcohol Syndrome and Adult Children of Alcoholics calls to mind the image of the blind men and the elephant: same animal, different perspectives of that animal, foolish disagreement – or lack of understanding, anyway, since the two perspectives don't seem to know enough about each other to disagree. It is clear from the FAS literature that issues of learning difficulties, attentional disorders, impaired executive functioning and health problems can

result from prenatal exposure to alcohol. It is equally clear from the Children of Alcoholics literature that there is at least a subgroup of COAs whose problems fall under the same categories, even though we call them externalizing or risk-relevant behaviors, somatization, lack of resiliency characteristics or dopamine deficiency. In some of the COA literature reviewed the problems were described using the same neurologically based nomenclature as in the FAS literature. Only one author connected the dots, laying out the two bodies of literature side-by-side, enumerating the symptoms endorsed by each, demonstrating the enormous parallels between the two. None of the extensive literature on both sides of the question searched for the present study acknowledges the influences of the other: people with FAS are often victims of family situations that are chaotic or in some way woefully inadequate, and children of alcoholics have, some of them, been exposed to alcohol in utero. The resulting layers of influence must be sorted out if we are to have truly useful, accurate understanding and treatment of the problems this overlapping population suffers.

#### D. What the Present Study Aims to Contribute

The present study intends to begin this sorting-out process by addressing the issues of prenatal alcohol that lie submerged in the ACA pattern as outlined in the National Children of Alcoholics Association (NaCoA) literature. It may be that the small subgroup of ACAs with serious difficulties delineated by some researchers is exactly that subgroup of ACAs whose mothers drank during pregnancy. This study will not be conclusive by any means, but it may begin to illuminate the reasons some ACAs have such a hard time while others are resilient, do not somatize, externalize, engage in risk-

relevant behavior, or otherwise fail at life. To uncover the source of these differences rather than simply name them is the ultimate aim of this inquiry.

## CHAPTER III.

### METHODS AND PROCEDURES

#### A. Introduction

The purpose of this research was to discover whether, among the difficulties purported to plague Adult Children of Alcoholics, a pattern of brain damage due to prenatal alcohol exposure could be identified.

Research and anecdotal reports yield evidence for a large number of ACAs whose problems outlive years of immersion in ACA literature, groups or other therapies. It is the hypothesis of this study that some of these recalcitrant difficulties may be misattributed to psychodynamic issues or defenses when in fact they may stem from organic brain damage due to prenatal alcohol exposure.

Identifying the signs of prenatal alcohol damage is not a simple task. There are only a few places in the world where actual diagnosis is even attempted (the clinic at Valley Medical Center in San Jose, California is one), as the pattern can be confounded by other prenatal conditions or events, genetic factors, postnatal environment, illness, endocrine imbalance, head trauma, and even sleep apnea. Nevertheless, screening devices have been created to provide at least a rough cut at such identification.

The problem with the existing screening devices is that by and large they aim for the lower-functioning end of the population: people whose lives are in considerable disarray. Adult children of alcoholics who have steady jobs, relationships, financial independence – even considerable success – may be suffering from the pattern of difficulties associated with prenatal damage, but would not be identified by either the professionals they seek help from or the screening tools that do exist. Their difficulties

would likely either be attributed by the former to the psychodynamic results of alcoholic parenting, or dismissed by the latter, too insignificant to even register.

This project is a basic correlation study: If the frequency of endorsement of the items on the FAS-related questionnaires (see Appendix B) co-varies with the level of alcohol exposure in utero, then a first building block may be present for identification of alcohol-related damage among ACAs. A supporting question is whether, as the level of maternal drinking during pregnancy goes up, the more organically-oriented items in the ACA checklist (the Laundry List, Appendix B) are more frequently endorsed.

The second question has to do with the questionnaires themselves. An instrument has been developed for this study (see S-PAD, Appendix B) because existing instruments to help with diagnosis or screening of FAS (FABS, ARNDD, IPCSI and FAESCEP, Appendix B) are perceived as too crude to catch these signs in higher-functioning people. The four best-known of these instruments have been included along with the newly-created S-PAD in order to test the hypothesis that, using only existing instruments, the population being researched here would not appear to be suffering from neurological damage caused by prenatal alcohol when in fact they are, at a level that the more subtle S-PAD identifies.

The questionnaires also provide demographic data relating to familial patterns of similar neurological difficulties such as learning disabilities or attention deficit disorder. Although it was not possible to guarantee accuracy regarding maternal drinking, participants were asked to assess to the best of their ability, checking with other family members or friends when possible.

## B. Participants, Sample and Recruitment

### 1. The Nature of the Participants

The participants in this study were involved in some type of therapeutic or self-help efforts regarding their status as Adult Children of Alcoholics. They were at least minimally aware of some of the patterns that characterize ACA issues; many of them had been working on these issues in therapy for years. While the vast majority of the people studied in the FAS/FAE field have clinically significant disorders and correspondingly disrupted life experience, the participants in this study ran the full gamut from unemployed to executive director.

### 2. The Nature of the Sample

Anybody whose mother was or is an alcoholic was invited to participate. Demographic variables such as age, education, employment, prior diagnosis of learning disability or attention deficit disorder, family history of such disorders, and socio-economic status were recorded but not controlled for.

### 3. Sample Size

Twenty-nine people participated.

### 4. Recruitment of Participants

Therapists known to the researcher to work with issues surrounding chemical dependency were contacted, and flyers sent (see Recruitment Notice, Appendix A). The researcher announced this study at conferences and talks, and recruited at ACA meetings. Friends and relatives of the researcher were recruited, and as the end of the time permitted drew near, Berkeley Flea Market-goers were recruited by means of a sign with an abbreviated version (see Appendix A) of the recruitment notice.

## C. Data Collection

### 1. Procedure

Respondents were screened over the phone, in person or by email regarding the presence of maternal drinking and experience with ACA self-help or therapy. If they met these criteria and agreed to sign the consent form, personal information was gathered (see Personal Information Form, Appendix A). Questionnaires and the consent forms were given or sent, along with (in the case of contact by mail) a self-addressed, stamped envelope for returning the documents. Participants were asked to sign the consent form before they filled out the questionnaires. Once the questionnaires were received, data analysis began. Participants understood that they had the right to ask questions, explain their answers or otherwise communicate with the researcher at any time. Participants also understood that they could request a copy of the results.

### 2. Instruments Used

There were six instruments used in this study, only one of which (the FABS; see Appendix B) has been through the rigorous test construction process necessary for hard-science research. In neither of the fields addressed here, Adult Children of Alcoholics (ACAs) and Fetal Alcohol Syndrome/Effects (FAS/E), is there yet a single fully accepted instrument that can be used for identification of the condition, although at least one is currently used for screening (ARNDD Checklist, see Appendix B). In the field of ACA work, this may result from the abundance of often-conflicting research results. In the field of FAS/E work, there have been only a few researchers addressing diagnostic issues (Streissguth, Bookstein, Barr, Press & Sampson, 1998; Astley & Clarren, 1999).

The first list of characteristics of Adult Children of Alcoholics was generated in the early 1980s (Woititz, 1983) and remains today the cornerstone of national organizations for both adult and young children of alcoholics. The Laundry List, as it has come to be known, is what callers are referred to when they inquire with the National Council on Alcohol and Drug Dependency or with the National Children of Alcoholics organization. This has long been the gold standard, the defining yardstick, of what it means to be a child, adult or otherwise, of an alcoholic, despite research yielding widely varying results. Issues of mood disorders, pathological behaviors, cognitive impairment, attentional disorders and interpersonal problems have yet to be conclusively pinned to the experience of growing up in an alcoholic home, with many studies showing significance (West & Prinz, 1987; Keperman et al., 1999) and many others failing to show significance (review by Seefeldt & Lyon, 1994).

It is exactly this puzzling variability in findings on Adult Children of Alcoholics that prompted the present study: what is it that differentiates the research subjects who seem to suffer little effect from their alcoholic parents and those who seem to suffer a great deal? With such massive amounts of research conducted, in such respectable quarters, it is unlikely that the variability is an artifact of research practices. It may well be that there is indeed a subset of children of alcoholics lumped in with the general category of ACAs who actually suffer from brain damage from prenatal exposure to alcohol, and that it may be this subgroup who contribute the data in studies showing greater significance.

The Laundry List (see Appendix B) was one of the questionnaires filled out by participants in the present study. It was hypothesized that as the level of alcohol

consumption rises, items 2 and 13 are more likely to be endorsed, as these two items reflect the symptoms of ADHD, the most common neurological result of prenatal exposure to alcohol.

While research regarding Fetal Alcohol Syndrome is gaining substance rapidly, the bulk of this work revolves around the medical aspects. Research on brain structure (Mattson & Riley, 1998), biochemistry (Gabriel, Hofmann, Glavas & Weinberg, 1998), mechanism of action (Voorhees, 1986), physical anomalies (Jacobson & Jacobson, 1997), medications (Hannigan, 1996) and many other medical/physical aspects have been in the literature since the early 1970s. Aspects of research involving laboratory animals abound (Hannigan, Berman & Sajac, 1993; West, Hodges & Black, 1993). Development of a simple diagnostic method to detect the presence of brain damage from prenatal alcohol exposure has not yet occurred, although a great deal of work in this direction has been done by the separate (but collaborative) teams led by Clarren and Streissguth at the University of Washington in Seattle.

The best-known diagnostic system for alcohol-related brain damage is the one developed by Sterling Clarren and Susan Astley in Seattle, requiring a team of experts, extensive review of records, testing, observation and interview with the caregiver (or adult patient) (1999). The diagnostic task is to sort out brain damage from genetic and postnatal/environmental influences, and then to rule out other sources of the brain damage such as traumatic brain injury, birth complications or illness. Charted on a 4 x 4 grid, the chief features of fetal alcohol damage are rated from 1 to 4 according to severity: Facial Features, Growth Retardation, Central Nervous System Damage (“Brain”) and Alcohol Consumption by the mother during pregnancy. Threes and fours

will result in diagnoses of Fetal Alcohol Syndrome, as they include the physical features associated with the full syndrome. If a person looks normal but has distinct neuro-behavioral symptoms of the sort described in detail in Chapters I and II (behavior, attention, cognitive, self-regulation, social skills and communication), he or she will be assigned a diagnosis of Static Encephalopathy or Neurobehavioral Disorder. With confirmed alcohol exposure (ranking 3 or 4), these would be Alcohol-Related. Without confirmed alcohol exposure they would be Alcohol Unknown.

Table 1.

Example of Diagnosis of Fetal Alcohol Damage Using Clarren System

4	Severe	X			
3	Moderate		X	X	
2	Mild/unknown				X
1	Absent				
		<i>Growth</i>	<i>Face</i>	<i>Brain</i>	<i>Alcohol</i>

The above grid demonstrates a diagnosis of 4332, or Sentinel Physical Findings, Static Encephalopathy, Alcohol Unknown. This would be someone who is severely growth-retarded in height and weight, whose facial anomalies show most of the typical FAS features (flat philtrum, thin upper lip, small eye openings set far apart), whose central nervous system shows impairment through some combination of severe cognitive, attentional, neurological, language or self regulation functions, and whose mother's drinking is unconfirmed but suspected.

The diagnostic process outlined above is extremely costly in time, resources and expertise, and can only be found in a few locations around the country. The category on the grid that has to do with central nervous system (third column, "Brain") functioning is the most relevant to the raft of behavioral, attentional, social and learning problems faced

by people with prenatal alcohol damage. These problems are assessed in Clarren's system using history, ADHD criteria, Learning Disability testing, speech evaluation, neuropsychological testing or screening and observation. Finding CNS damage in this way has itself been too unwieldy for wide use, and so a few researchers have developed checklists for purposes of screening and/or streamlining diagnosis. The four best-known of these checklists are addressed in this study, namely Ann Streissguth's Fetal Alcohol Behavior Scale (FABS, 1998), Larry Burd's Alcohol-Related NeuroDevelopmental Disorder Behavioral Checklist (ARNDD, 1999), Susan Doctor's Information Processing Characteristics Screening Instrument (1999), and Deborah Spira and Deb Evensen's "Fetal Alcohol Affected Student Checklist for Educational Programming. (See Appendix B for all scales.)

Streissguth's FABS is the most researched of the four, studied in at least five different research projects in the Fetal Alcohol and Drug Unit in Seattle (Streissguth et al., 1998). This 36-item scale began with caregivers' comments describing the behavior of their charges, transcribed by Streissguth and her colleagues, then eliminating items for lower frequency or item-to-scale correlations. Five studies, using a reference sample of 472 patients diagnosed with FAS or FAE (by Clarren), demonstrate high item-to-scale reliability (Cronbach's alpha = .91) and good test-retest reliability ( $r = .69$ ) over an average interval of 5 years. The FABS identifies many of the subjects with known or presumed prenatal alcohol exposure in detection studies using both prison and general samples. FABS scores also predict dependent living among adult patients with FAS/FAE. Within the sample used, the FABS is uncorrelated with IQ, sex, age, race, and diagnosis (FAS vs. FAE).

It should be noted that most of the people in Streissguth's studies had fairly serious problems in living. The original 472 who made up the reference sample had come to clinical attention in the first place, referred for diagnosis by physicians, therapists and school personnel because of a level of dysfunction that suggested brain damage. Another group contributing data were 81 men in a special unit in the Washington State Prison System for developmentally disabled, emotionally disturbed (nonpsychotic) inmates who either thought their mothers had drinking problems or not. Of the men who did ( $n = 13$ ), three scored relatively high on the FABS. The final group, for the Normative Study, was the group without identified serious difficulties: 186 parents in a waiting room at the University of Washington Medical Center. Thirteen of these parents identified mothers with drinking problems; seven of the children so identified scored high on the FABS; the others, including children identified with fathers with drinking problems, scored very low, indicating that the problems the children manifested were not the "children of alcoholic" issues, but related directly to maternal drinking. Whether there was prenatal drinking was unknown. Out of the 498 subjects who were identified in Streissguth's studies as having been affected by their mothers' drinking, only 13 – from the medical center waiting room – were (presumably) living normal lives. This skew toward the dysfunctional results in a behavior scale that would rather completely exclude people whose lives fall within normal limits of functionality.

Although the FABS is the most researched and widely-known of the available instruments, the present study used it as one of the comparison questionnaires. The extreme difficulties in living experienced by both the reference group and subsequent samples result in a picture of this disorder that presumes broken lives, with visible

manifestations of organic damage. The perspective of this researcher follows Streissguth's own dose-response theory (Streissguth, Bookstein & Barr, 1996) that states that depending on the severity of the drinking, the pattern of cognitive and behavioral damage caused by prenatal alcohol can range from the most severe (mental retardation, dependent living, chronic and inevitable failures in work and relationships) to the most subtle (attentional problems, learning disabilities, poor judgment, social difficulties). As detailed above, Streissguth's work has been with people on the more severe end of difficulty; the FABS reflects this level of severity.

The participants of the present study were people who identify as Adult Children of Alcoholics, having sought information and/or assistance regarding the issues pertaining to growing up in an alcohol-affected household. Keeping jobs and relationships, staying out of jail and generally getting along in society is the norm for this population. Nevertheless, as everyone in this group had mothers who drank at some level during pregnancy, it is the assumption of this researcher that there would be symptoms of prenatal alcohol damage; indeed, as noted above, two items on the standard ACA checklist (Laundry List) may be reflective of such damage. These symptoms, however, would be of a more subtle, higher-functioning variety than the items on the FABS would identify, by virtue of the difference in socioeconomic functioning between the two populations measured. This overshooting of the FABS items can be illustrated by item # 2 from the section on Personal Manners: "Messy: paperwork is smudgy and ruffled; makes more of a mess eating than others the same age; unconcerned about personal cleanliness (for example, hands, face and clothes are often dirty)." Where messiness may indeed be an issue for our ACA self-help-group members, they are unlikely to leave their

hands, faces or clothes dirty, and certainly not often. This item would not be endorsed, giving a false impression of nonrelevance. Many of the items on the FABS have been adjusted upward to create the instrument in this study, reflecting a more sophisticated and generally well-functioning manifestation of the same core disability.

The next most-studied of the tools is the Alcohol-Related NeuroDevelopmental Disorders (ARNDD, Appendix A) Screen by Larry Burd, Ph.D. This is a self-published screen along with several other tools by Dr. Burd, being used in a large Centers for Disease Control study. This is a 34-item checklist, with 20 checked items or more resulting in a positive screen, and was used in the present study in a manner similar to the FABS: it was one of the background tools, used for comparison. Like the FABS, the ARNDD screen overshoots for the population of conscious, growth-seeking Adult Children of Alcoholics. Each individual item may indeed reflect a true difficulty for ACAs, but as expressed in this screen take a too-broad approach. One example: “Can’t follow routine – needs reminders to get dressed, brush teeth, etc.” As in the FABS example, while the underlying disability may exist, expressed perhaps as a need to keep post-its scattered around the house in order to maintain a schedule, or an experience of despair when the Day-Runner is lost, the present population does not need a reminder to get dressed. Again, many of the items here have been adapted upward for the instrument in the present study.

Another instrument used for comparison was the “Information Processing Characteristics Screening Inventory” (Appendix A developed by Susan Doctor for her dissertation, also still in progress). Ms. Doctor is a consultant for Superior Court in Reno, Nevada on Fetal Alcohol Syndrome. Her task is to help identify FAS/FAE in

minors and adults in the criminal justice system, and then devise interventions to assist them in maintaining a crime-free life. Her years of training, observation and research have gone into this 43-item instrument, including a validity scale of seven items. So once again the scale is a 36-item checklist, and once again it is aimed at a less sophisticated, lower-functioning group than the present study is looking at. One example: “I sometimes can’t get much done for days, weeks and months.” While procrastination is common with people in general, it is more pronounced with people with Attention Deficit Hyperactivity Disorder, some symptoms of which are considered core disabilities of FAS/FAE. Nevertheless, returning to the participants in the present study, if one doesn’t get much done for months at a time then one’s main preoccupation might be where the next meal is coming from rather than exploring the finer points of interpersonal difficulties resulting from an alcoholic upbringing. As with the other instruments, the items on Ms. Doctor’s screen have been included in the present study’s questionnaire, in a more sophisticated, higher-functioning form.

The final of the five comparison instruments is the “Fetal Alcohol Affected Student Checklist for Educational Programming” (see Appendix B), developed by Deborah Spira, Ph.D., and Debbie Evensen, M.S. for use in their consultation practice in Alaska working with mostly Native American students damaged by exposure to very heavy doses of prenatal alcohol. This 38-item checklist is meant to apply to young school children, but the instructions to participants in the present study were to recall whether any of these difficulties pertained to them in early childhood. This checklist is based on organic difficulties that would probably result in placement in a Special Day Class or other non-mainstream educational situation. It was not expected that most of the

current research participants would endorse more than one or two of these items, but it was included as a most-serious-case measure: it was possible that those ACAs who had experienced the most problems as measured by the other instruments would also endorse several of these items. Examples from this checklist are: “Frequently unaware of surroundings and may be oblivious to very dangerous situations;” “Severe temper tantrums.”

The questionnaire developed for the present study, Signs of Prenatal Alcohol Damage (S-PAD) (see Appendix B) has 51 items, drawn from the pre-existing instruments as well as from research on Fetal Alcohol Syndrome. As noted above, the items similar to those on other checklists have been adjusted to reflect a more subtle version of the same core disability. The purpose of the S-PAD in general was to detect a pattern of fetal alcohol damage in relatively high functioning people; for this study the S-PAD was used to look for this pattern among people self-identified as Adult Children of Alcoholics whose organicity might not show up on the other scales.

Table 2 is an accounting of the roots of the items on the S-PAD. That is, each of the S-PAD’s items (with a few exceptions discussed below) is an adaptation of some combination of items from the FABS, the ARNDD and the IPCSI. For example, the S-PAD’s first item, “Do you lose track of material items frequently (keys, paperwork, jewelry)?” is drawn from item # 5 in the Personal Manners section of the FABS: “Tends to lose or misplace things a lot,” items # 2 and # 4 from the ARNDD: “Poor attention” and “Disorganized,” and items # 7, # 32 and # 40 from the IPCSI: “I think there is something wrong with my memory,” “I forget things a lot,” and “I lose things all the time.” The adaptation in this case makes the distinction between the general, pervasive

habit of losing things (clothing, money, identification) that could seriously interfere with the ability to live normally, and the less serious but still very annoying losses that result from the memory impairments in the higher-functioning person with fetal alcohol damage.

Table 2.

(Note: The letters under the FABS column represent the various subtitles of that instrument.)

S-PAD Items' Roots in Other Scale Items

S-PAD	FABS	ARNDD	IPCSI
1.	PM5	2, 4	7, 32, 40
2.	PM 5, 1	2, 4, 17	7,14, 40
3.	A/WP 3	4, 19, 22	10, 16
4.	A/WP 1	3, 5	18
5.	A/WP 3	4, 5	8, 16, 29, 39
6.	PM 2, 5	5, 22	10, 40
7.		3	14
8.	SSI 4	3, 7, 10	13
9.-12.*			
13.	A/WP 2	19	10
14.	A/WP 2	19	10
15.			41
16.	PM 1	29	
17.	PM 1, MS 1 & 2	29	
18.	BoP 1		
19.	BoP 4		
20.	MS 2		
21.	PM 2		
22.	CS 2, 5	14	24
23.	CS 7		
24.**			
25.	CS 5		
26.	A/WP 2, SSI 3		
27.	SSI 9	7, 10	
28.		12	21, 22
29.	SSI 6		
30.	CS 3, 7	12	
31.	SSI 3, 4, 5	8	
32.- 40.***			

41.	E 1, 2		
42.	E 2		
43.****			
44.		24	
45.	A/WP 2	19	
46.		26	
47.		27	
48.			43
49.		20	42
50.	BP 5	28	14
51.		32	

\*Items # 9-12 address the cognitive difficulties outlined in both the Institute of Medicine's (1996) report and Streissguth et al.'s (1996) work on secondary disabilities.

\*\*Item # 24 refers to lying, a characteristic much more discussed in caregiver and professional circles than academic. Often lumped in with stealing and cheating, lying is attributed variously to short memory, poor sequencing ability, panic under pressure, inflexibility of thinking or lack of empathy. It is reported by parents (on faslink, an online support group of about 75 families), a foster agency (Families First, personal communication, 1999), and consultants (Diane Malbin, personal communication, September 2000, and John Lyle, personal communication, 1999) to be an extremely common characteristic of fetal alcohol damage, and among the most destructive.

\*\*\*Items # 32-40 refer to typical medical problems and physical abnormalities associated with damage from prenatal alcohol consumption (Clarren, 1995; Institute of Medicine, 1996).

\*\*\*\*Item # 43: Depression is a very common secondary disability accompanying fetal alcohol damage, suffered by 90% of the 472 subjects in the Seattle study (Streissguth et al., 1996).

### 3. Data Analysis

Analysis of variance tests of significance were conducted on the questionnaires to seek patterns of main effect of prenatal alcohol exposure on item endorsement; that is, whether items on scales reflecting possible organic damage were more frequently endorsed by respondents who were in fact more exposed. Two items on the Laundry List are analyzed in addition to the FAS/E scales.

The second major question was whether the S-PAD more reliably identified prenatal alcohol damage in higher-functioning respondents than the other scales. An ANOVA test of significance was conducted on the respondents' level of functioning compared to the scores on the six scales.

#### D. Reliability and Validity

All of the instruments used in the study were examined for reliability. (See Table 1.) The analyses revealed that the scales ranged from a Cronbach's Alpha of .6103, on the Woititz Laundry List, to an Alpha of .9370 on the Information Processing Characteristics Screening Instrument. When the Signs of Prenatal Alcohol Damage (S-PAD) questionnaire was analyzed, a number of items were identified as contributing to a reduction in the reliability of the scale. When the S-PAD was revised, these items were removed from the total scale measurement and the Alpha increased. The items that were removed included: item 18 "Do tags on clothes bother you;" item 22 "Do you like to talk, perhaps more than the average person;" and items 32 through 40. Items 32 through 40 were all of the Physical/Medical items and asked questions about physical characteristics related to Fetal Alcohol Syndrome (e.g. "Do you have asthma, allergies, ear infections, birth defects, thin upper lip?" etc.) In general, physical manifestations are more likely when the level of prenatal exposure to alcohol is severe. It may be that the other aspects of the S-PAD are measuring more subtle effects. In the results section, both the original 51-item S-PAD and the 39-item Revised S-PAD are used and discussed.

Table 3.  
Reliability of Scaled Instruments for Diagnosing Brain Damage  
in Children of Alcoholics

Instrument	Alpha
Laundry List (Woititz, 1983)	.6103
The Fetal Alcohol Behavior Scale (Streissguth et al., 1997)	.8944
Fetal Alcohol Affected Student Checklist for Educational Programming (Spira & Evensen)	.8403
Information Processing Characteristics Screening Instrument (Doctor, 1999)	.9370
The ARNDD Behavioral Checklist (Burd, 1999)	.8673
Signs of Prenatal Alcohol Damage (Page, 2000)	.7615
Signs of Prenatal Alcohol Damage, Revised (Page, 2001)	.8720

Only one of the instruments used for this study has been published with formal statistical analysis (FABS, Streissguth et al., 1997). Neither of the fields under focus, Adult Children of Alcoholics and Fetal Alcohol Syndrome, has produced a state-of-the-art, reliable, valid diagnostic instrument. Streissguth has come the closest, but her FABS, like the other three used for comparison in this study, is calibrated toward the more dysfunctional of the populations affected by prenatal alcohol exposure and, it assumed, is likely to miss the higher-functioning end of this population who still suffer from the core disabilities, but at a level that allows them to carry on a reasonably orderly life.

The instrument created for this study is called: “Signs of Prenatal Alcohol Damage,” or S-PAD. Thirty-seven of the 51 items are taken from the existing tools and address the core disabilities therein, but have been adapted to a level that reflects the

sophistication and functionality of the population under focus in this study: Adult Children of Alcoholics who have sought therapy or self-help for the kinds of interpersonal and intrapsychic difficulties commonly attributed to growing up in an alcoholic home. All the items on the S-PAD are rooted as well in the research on the patterns of damage that result from prenatal exposure to alcohol.

The next chapter deals with results, and will have.....5—with anecdotal and demo.....three parts as discussed above in Data Analysis. The first section presents findings regarding the relationship of the prenatal maternal drinking to signs of organic damage using ANOVA tests of significance comparing level of maternal consumption to scores on the six scales. The second section presents the results of the ANOVA test of significance regarding the S-PAD's ability to identify difficulties that the other scales miss. The third section presents ANOVA results comparing the relationship between maternal prenatal drinking and the two (possibly brain-based) items on the ACA checklist, the Laundry List (see Appendix B).

The final chapter addresses the patterns that emerge from the above comparisons. Limitations of the present study are addressed, along with discussion of relationships between existing research and the results from the present study, with implications for clinical application and further research.

## CHAPTER IV.

### RESULTS

#### A. Introduction

Data was collected on 29 participants. It consisted of demographic information and six questionnaires: one Adult Children of Alcoholics checklist and five instruments regarding Fetal Alcohol Syndrome or Effects. ANOVA tests for significance were done on the effects of 1) level of prenatal alcohol exposure, 2) level of life functioning, and 3) absence or presence of prenatal binge drinking, all on the group mean scores of the questionnaires. In addition, an ANOVA test was done on the effect of level of alcohol exposure on two items in the ACA scale.

##### 1. Demographics

There were 29 participants, 3 of whom did not complete all the questionnaires, so some of the totals do not equal 100%. All participants were self-identified Adult Children of (maternal) Alcoholics who had sought help at some point in the past for ACA issues. There were 22 women and 7 men, with 5 people in their 20s, 3 in their 30s, 6 in their 40s, 9 in their 50s and 2 people in their 60s. Education ranged as follows: 2 people had not finished high school, 3 people had finished high school, 7 people had some college and 7 had graduated, and 7 people had more than 17 years all together. There were 2 adoptees and 2 foster children. Eight people answered the question regarding level of chaos in the family; all noted scores between 7 and 10 on a scale of 1-10. (All but one of these people were in the high-functioning category explained below.) The question regarding a history of Attention Deficit/Hyperactivity Disorder and Learning

Disabilities in parents or grandparents yielded 14 no's, 4 question marks and 11 left blank.

## 2. Levels of Prenatal Alcohol Exposure

Three levels of prenatal exposure to alcohol were assigned. There were 8 people at Level 1, with reported exposure to the lightest doses of alcohol, ranging from a few glasses of wine a week at the most to two drinks a day at most. Level 3, with 7 people, was assigned to people whose mothers consumed over 14 drinks a week. Level 2, with 13 people, comprised those whose consumption fell in between.

## 3. Levels of Functioning

Levels of functioning were also assessed on a 3-point scale. The lowest level, Level 1, consisted of people who were living at a subsistence level, had a hard time keeping jobs, or had more than the usual amount of chaos in their lives. An example is a 65-year-old woman who has never worked, lives on a couch with relatives, and relies on medications to control her bipolar mood disorder. In spite of the difficulties inherent in her life, however, she continued to seek self-improvement, joining a 12-Step group and reading self-help literature. She also enjoyed good relationships with friends and family, going on frequent outings like concerts or picnics. There were 5 people at this level, including one hairdresser, one who was chronically unemployed, one flea market salesman, one disabled person and one long-term student.

The 5 participants who were categorized as Level 1 shared three aspects: life problems, some internal and external resources and some form of voluntary self-improvement. The extent of the life problems shared by these 5 individuals generally fell into the passive category – not enough money, stability or other resources – rather than

the active category of law-breakers, drug or alcohol addicts, mental patients, expellees-from-school also included in Ann Streissguth's Secondary Disabilities study (1988). It is assumed that the criterion of having sought help for ACA issues may have screened out people with this more serious level of dysfunction.

Level 2, with 8 people, included those who either seemed not to be living up to their capacity, as determined by comparing education with employment, or to be living with considerable difficulty. A representative example is the 35-year-old housewife who had studied to be a marketing consultant and began a career in this work, to find that the stress of managing a job and a family was overwhelming. She quit in her early 20s, wishes she could go back to work, but doesn't think she could do it. Others were college graduates with work as a temporary phone operator, a waitress and a secretary. Three in this group reported living "on the edge," "just keeping my head above water," and "worried all the time."

Level 3 consists of people who run agencies, teach, counsel, write – people who might seem to be manifesting their talents and gifts with little interference from their heritage as Adult Children of Alcoholics. Nevertheless, from comments gleaned from the "Other Information Volunteered" section of the Personal Information Form (Appendix A), it is apparent that even in this group there is a common theme of frustration with at least a few aspects of malfunctioning that have resisted attempts at traditional remediation.

## B. Results of Data Analysis

### 1. Difficulties Related to Prenatal Alcohol Exposure

The raw means and standard deviations for each of the scales at each level of exposure are presented in Table 4, below. An ANOVA test of significance on the effect of the level of prenatal exposure was conducted.

Table 4.

Raw Score Means and Standard Deviations of Diagnostic Scales  
by Level of Prenatal Exposure to Alcohol

Exposure	Level 1 (N=8)		Level 2 (N=13)		Level 3 (N=7)		Sig
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	
Instrument							
Laundry List	8.8	3.0	9.8	2.1	10.4	2.3	*
FABS	6.9	5.6	14.5	7.2	14.6	6.0	*
FAA Student Checklist	3.8	2.3	6.5	7.0	8.9	5.7	ns
IPC Screening Instrument	5.7	1.7	13.8	9.3	16.0	8.1	*
ARNDD Behavioral Checklist	4.14	1.2	9.8	6.8	11.1	5.1	ns
S-PAD	16.5	5.7	20.8	9.6	27.7	5.5	*
S-PAD, Revised	13.6	6.1	18.1	9.3	23.0	4.4	ns

\*  $p < .1$

As Table 4 indicates, each of the instruments distinguished between the levels of prenatal exposure to alcohol. As exposure increased, so did the mean ratings on all of the scales.

In order to make direct comparisons between the diagnostic instruments, the raw scores were adjusted so that all scales had a range of 0-1. The adjusted score means are presented below, in Table 5.

Table 5.  
Adjusted Score Means and Standard Deviations of Diagnostic Scales  
by Level of Prenatal Exposure to Alcohol

Exposure	Level 1 (N=8)		Level 2 (N=13)		Level 3 (N=7)		Sig
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	
Laundry List	.67	.22	.76	.16	.80	.18	*
FABS	.19	.15	.40	.20	.40	.17	*
FAA Student Checklist	.10	.06	.18	.19	.25	.16	ns
IPC Screening Instrument	.16	.04	.38	.26	.44	.23	*
ARNDD Behavioral Checklist	.12	.03	.29	.20	.33	.15	ns
S-PAD	.32	.11	.41	.19	.54	.11	*
S-PAD, Revised	.35	.16	.46	.24	.59	.11	ns

\*  $p < .1$

Adjusting the raw score means reveals that, other than the Laundry List, the revised S-PAD is the most sensitive instrument, with the highest mean at each level of prenatal exposure to alcohol.

Past research has suggested that maternal binge drinking during pregnancy has a particularly strong effect on the incidence of FAS/FAE. In order to assess if participants whose mothers were binge drinkers could be distinguished from the others through the use of the diagnostic instruments, information about maternal binge behavior was entered into the analysis. In Table 6, the average instrument ratings of participants with mothers who binged were compared to those with mothers who did not binge, using the adjusted scores. An ANOVA test of significance on the effect of the binge-drinking variable was conducted.

Table 6.  
Adjusted Score Means and Standard Deviations of Diagnostic Scales  
by Prenatal Exposure to Maternal Binge Drinking

Exposure	No Binges (N=19)		Binge Drinking (N=10)		Sig
	Mean	Std. Dev.	Mean	Std. Dev.	
Laundry List	.72	.19	.75	.23	.640 ns
FABS	.34	.18	.36	.23	.806 ns
FAA Student Checklist	.16	.17	.21	.16	.531 ns
IPC Screening Instrument	.31	.21	.41	.27	.309 ns
ARNDD Behavioral Checklist	.24	.16	.29	.19	.395 ns
S-PAD	.40	.15	.47	.19	.357 ns
S-PAD, Revised	.42	.18	.54	.23	.115 ns

\*  $p < .1$

None of the instruments were able to significantly distinguish between respondents who were and those who were not exposed to maternal binge drinking, although the revised version of the S-PAD approached a significant difference that might be achieved if the sample size was larger. In further analysis, there was also no significant correlation between the maternal binge drinking variable and the level of functioning measure ( $r = -.109$ ,  $p = .574$ ).

## 2. Analysis of Laundry List Items Related to Prenatal Exposure

Two of the items on the Laundry List may be indicators that reveal prenatal exposure: item 2, “Adult children of alcoholics have difficulty following a project through from beginning to end,” and item 13, “Adult children of alcoholics are impulsive. They tend to lock themselves into a course of action without giving serious consideration to alternative behaviors or possible consequences. This impulsivity leads to confusion, self-loathing, and loss of control over their environment. In addition, they spend an excessive amount of energy cleaning up the mess.”

When the responses to the above items based on the level of exposure to prenatal alcohol were analyzed, very different patterns appeared. The two items are compared in Table 7, below.

As the table indicates, item 2 is significantly related to exposure, with all of the people in the group with the highest exposure endorsing the item. Item 13 did not have a pattern of responses that was related to prenatal exposure. Regardless of exposure level, the response rate was about the same.

Table 7.

Adjusted Score Means and Standard Deviations of Laundry List Items  
by Level of Prenatal Exposure to Alcohol

Exposure	Level 1 (N=8)		Level 2 (N=13)		Level 3 (N=7)		Sig
Laundry List Item	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	
Item 2	.38	.52	.77	.44	1.0	.00	**
Item 13	.75	.46	.69	.48	.76	.44	ns

\*\* p <.05; \* p <.1

Using the S-PAD to Identify Prenatal Alcohol Exposure

The sensitivity of the various instruments in identifying the effects of prenatal alcohol exposure for the various levels of functioning is examined in Table 8.

Table 8.

Adjusted Score Means and Standard Deviations of Diagnostic Scales  
by Level of Functioning

Functioning	Level 1		Level 2		Level 3		Sig
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	
Laundry List	.68	.26	.73	.17	.70	.20	ns
FABS	.51	.18	.42	.17	.22	.15	**
FAA Student Checklist	.31	.17	.23	.21	.10	.08	*
IPC Screening Instrument	.56	.30	.42	.21	.21	.09	**
ARNDD Behavioral Checklist	.44	.12	.36	.21	.14	.05	**
S-PAD	.53	.12	.48	.15	.30	.13	**
S-PAD, Revised	.60	.16	.54	.17	.32	.16	**

\*\* p <.05; \* p <.1

All of the instruments, with the exception of the Laundry List, have scores that decrease as the level of functioning increases. One of the original goals of the S-PAD development was to produce a screening device that would facilitate the identification of people who were affected by alcohol exposure before birth, even though they could be considered quite high-functioning. Of all the instruments in the study, the revised S-PAD is the one with the highest mean score at all levels of functioning. Overall, the instrument appears to be the most sensitive.

### C. Summary of the Results

Scores on the seven scales indicate that there are indeed signs of prenatal alcohol damage in the group of Adult Children of Alcoholics who participated in this study. These signs were found at all the levels of functioning and all levels of exposure. As the level of exposure rises, so do the scores on all scales, including the ACA checklist in general, and on one ACA item in particular. The S-PAD is the most sensitive instrument

of the six FAS/E scales, better identifying participants at all levels of functioning than any of the others.

## CHAPTER V.

### DISCUSSION AND IMPLICATIONS OF THE FINDINGS

#### A. Study Question

This study looks into the relationship between prenatal alcohol exposure and the difficulties experienced by Adult Children of Alcoholics. It was the intent was to see if, within the constellation of academic, interpersonal and practical problems that are said to plague ACAs, a pattern of organicity could be detected and correlated with the level of prenatal alcohol exposure.

It was also hypothesized that the instrument developed for this study, the S-PAD, could spot the more subtle manifestations of organic damage found in the higher-functioning population represented by self-improvement-seeking ACAs at a higher level of sensitivity than existing tools.

The findings of this study suggest that there may indeed be a pattern of organically-based difficulties to be found in many ACAs, alongside – even embedded within – the well-documented detritus of perceptions and behavior learned in the alcoholic household. The findings also show that this pattern of organicity intensifies with higher levels of exposure to prenatal alcohol. The third primary finding is that the S-PAD is more sensitive at all levels of this population to signs of organic damage.

All participants except one endorsed over half of the items on the Laundry List, reflecting a consistent identification with such traditionally-defined ACA characteristics as troubles with judgment, inability to have fun, difficulty with intimate relationships, need for approval, sense of being different, hyperloyalty and impulsivity. This clearly shows that the participants identified themselves as ACAs and it was assumed that most

of them would endorse a majority of these items, independently of the level of prenatal exposure. However, the scores on the Laundry List rose as the levels of prenatal alcohol exposure went up. The lightest-exposed group endorsed an average of 67% of the 13 items and the heaviest-exposed group an average of 80%. The fact that the Laundry List scores rise with the level of exposure could be attributed to at least two possible factors: first, the general level of chaos and dysfunction in the household could co-vary with the level of drinking during pregnancy. The second possibility is that some of the items on the Laundry List actually do reflect organic damage, and would then be endorsed more frequently with heavier exposure.

The two items on the Laundry List most likely to reflect organicity are items # 2 and # 13. These items are related to the most to one of the most common diagnoses found in people diagnosed with FAS/E: Attention Deficit Hyperactivity Disorder. It was therefore hypothesized that the frequency of endorsement of these two items would increase as at the higher levels of prenatal alcohol exposure.

This turned out to be true for item # 2. One third of the least exposed, two thirds of the moderately exposed, and all of the most exposed people endorsed item # 2. This item, embedded as it is in a list of purportedly psychodynamic results of growing up in the alcoholic family, can thus be proposed as a red flag for organicity.

Item # 13, on the other hand, was not supported by the data as an indicator of organicity. This item was equally highly endorsed by the least exposed and the heaviest exposed. It may be that this particular constellation of ADHD-type behaviors is extremely common in the dysfunctional household; it may also be that these behaviors

are among the first to be affected by prenatal alcohol consumption, therefore being endorsed at all levels of exposure.

The scores on the Laundry List established that most participants in this study identified with characteristics thought to reflect ACA dynamics. The dramatic correlation of item # 2 with level of prenatal alcohol exposure suggests that signs of organicity can be detected within the traditional ACA paradigm, and the co-varying rise in overall Laundry List scores and level of exposure point to the possibility that several of the other items may reflect organicity as well.

The six other scales (this includes the revised S-PAD as well as the original) used for this study each take a slightly different approach to screening for FAS/E, and each one shows a different amount of increase in mean scores as prenatal exposure goes up. On the whole, however, it is clear that signs of prenatal alcohol exposure are seen at each level, even the least exposed. According to half the scales, there are detectable differences at each level of exposure, and according to the other half, the main difference in scores is between light exposure and moderate-to-heavy exposure.

The FABS, ARNDD and IPC follow a similar pattern, finding signs of prenatal alcohol damage at all levels, with a big split separating the low-exposure/high-functioning group from the moderately-to heavily-impacted groups. The ARNDD and IPC results were almost identical. Two conclusions can be drawn from this: first, that there are signs of prenatal alcohol damage showing up at all levels of exposure and functioning, including the least affected; second, that these scales are consistently more sensitive with the more affected participants.

The fact that signs of organic damage can be found even at the level of functioning occupied by teachers, marketing managers and executive directors is underscored by the last scale, the FAA. Meant for preschoolers already identified as having brain damage from prenatal alcohol exposure, this scale contains items that one would not expect any of the people mentioned above to endorse, yet the average number of endorsed items on this scale is 3.8 for this group.

Reported binge drinking was analyzed to see if, independent of the level of ongoing drinking, high blood alcohol levels were related to the various scale scores. All the group means in the no-binge group were lower than the means in the binge group, suggesting that there was some effect, although the S-PAD was the only scale that came close to significance. It may be that the information itself was inaccurate: with the moderate and heavier drinking of several decades ago, weekend parties were such a normal part of the pattern even during pregnancy that binges, commonly calculated at five drinks or more on one occasion, did not stand out enough to be recalled or reported.

Addressing the question regarding the sensitivity of the S-PAD versus the other instruments, it was found that both versions of the S-PAD had the highest percentage of item endorsement at all levels of both prenatal exposure and functioning, with scores increasing roughly equally between levels. It therefore appears that the S-PAD could be considered to be the most sensitive of the instruments used in this study. Since these instruments are currently the best recognized in the field, the S-PAD may be the most sensitive in the field to date, particularly among a population that is relatively free from legal, interpersonal, financial, mental or other entanglements.

As explained and illustrated in Chapter III, the S-PAD was constructed with its roots in the disabilities noted in the literature as well as the other scales, but the phrasing of the items was adjusted to include description of behavior considered more acceptable to a higher functioning group than the groups traditionally screened for FAS/E. An example from the S-PAD is: “Do you spill or drop things more than others do,” compared to the FABS: “...unconcerned about personal cleanliness...hands, face and clothes are often dirty.” This difference between simple reflection of visual-motor impairment in the S-PAD and description of specific outcomes in the FABS may be an illustration of the reason for the higher endorsement rate of the S-PAD. The impairment will show up at all levels with varying manifestations, but it is only necessary to name the impairment – stopping short of describing the particular manifestation will give it a broader utility. In addition, the FABS’ attribution of a socially unacceptable attitude (“unconcerned”) clouds the issue, and would certainly be a deterrent to endorsement among people who do indeed care about personal cleanliness but might have a harder time maintaining it.

Six of the items removed from the S-PAD to improve internal consistency were those related to physical and medical characteristics associated with Fetal Alcohol Syndrome. The facial features, size and heart irregularities (#s 35-40), are by and large limited to people who have been so severely impacted by prenatal alcohol exposure that they are not able to function independently and so would not be endorsed by the current population. The other items in this section, regarding asthma (# 32), allergies (# 33) and ear infections (# 34) were endorsed randomly. These maladies might be common enough

in the general population to also affect many people without prenatal alcohol exposure. In any case, they didn't contribute any useful information to this study.

Another item removed was # 11, "Was 'Not working up to capacity' a frequent entry in your report card?" This item was also endorsed in no particular pattern. Although such underachievement is a consistent feature of prenatal alcohol damage, it has been widely noted with ACAs in general. Depression, the above-mentioned dissociation, family chaos, anxiety or other concomitants of the alcoholic household can certainly cause a person to operate at less than optimal levels.

The final item removed was # 18, having to do with sensitivity to tags on clothing. Sensitivity to tactile stimuli is thought to be as common a feature of prenatal alcohol damage as the cognitive or attentional features and it was expected that this item would be endorsed at the same frequency as most of the other items. However, endorsement of this item was also random, suggesting that, among this group of participants, sensory integration was not one of the features that co-varied with levels of prenatal alcohol exposure. It is unclear why that would be so, but one possible (if far-reaching) explanation might be that ACAs, being well-practiced at "dissociation and compartmentalization of feelings" (Cermak, 1991, p. 38), just do not allow the more subtle intrusions on physical well-being into consciousness.

The S-PAD, then, was found to detect signs of prenatal alcohol damage at all levels at a higher rate, and therefore with more sensitivity, than the other instruments. The revised version had better internal consistency than the original, and came close to detecting effects from binge drinking. It appears that this instrument could be used effectively with a high-functioning population such as help-seeking Adult Children of

Alcoholics where the other instruments would be likely to overlook the subtle pattern of prenatal alcohol effects in this group.

## B. Discussion of Findings in Relation to the Literature

### 1. Adult Children of Alcoholics

Enoch Gordis, the Director of the National Institute on Alcohol Abuse and Alcoholism, declared that ACA issues are related to the “social and psychological dysfunction that may result from growing up in an alcoholic home” (1990, p. 4). This stance is not unique to Dr. Gordis; most of the thinking in the ACA field conforms to this philosophy. Stephanie Brown enumerates the problems common to ACAs as including “learning disabilities, attentional disorder, depression, anxiety, and mood disturbance” (1999, p. 11). She has attributed such difficulties to family development: “...patterns formed...within the family then structure...cognitive, affective and social development” (p. 5). The remedy for such maladaptive development is psychotherapy, where, she says, the family must go through a process similar to that of the individual in recovery (pp. 156-158).

The findings of the present study suggest otherwise, as does a great deal of literature in fields that focus on neurological conditions. The first two ACA issues highlighted above by Brown, for example, are well-documented neurobehavioral problems, rooted firmly in the brain and remedied rarely if at all. Learning disabilities and attentional problems can be found at the heart of many problems in school, interpersonal relations, finances, even legal problems.

They are also at the heart of the disabilities that occur to people who are exposed to alcohol in utero, and certainly appear in the data collected for this study. Even at the

highest level of functioning, participants endorsed items reflecting brain-based difficulties of the sort that are commonly categorized as learning disabilities and attention deficit/hyperactivity disorder; as the level of prenatal alcohol exposure rises, so do these difficulties.

The results of this study, therefore, contradict traditional ACA thinking regarding both the assumed etiology and the normal intervention for at least this subsection of ACA problems: it does not appear that these problems come from family dysfunction, but from neurological damage created by exposure to alcohol before birth.

An area of controversy within the ACA field noted in Chapter II is reflected in the wide range of conclusions about whether ACA troubles really exist or not. Many of the studies that find little evidence for pathology have been done on college students. The study by Hunt (1999) for example, gave students the MMPI and the CPI, and found nonsignificant results, leading her to conclude that the fabled ACA problems were an exaggeration. It should be noted that neither of these tests asks much about manifestations of learning disabilities or attention deficit disorder. It is also noteworthy that this population is of the higher-functioning academic variety, and any LD/ADHD symptoms are being managed sufficiently at this point if they exist at all in the population studied. It is hypothesized that these nonproblematic ACAs may be those who were not exposed to prenatal alcohol.

Other studies certainly found pathology among ACAs, as articulated by Naimah Weinberg (1997): “More children may be affected by parental alcohol use than are recognized...(and) represent significant subgroups of children with ADHD, learning disabilities, language disorders and other psychiatric conditions...”

Findings of this study suggest that there is indeed a subset of ACAs who suffer brain-based problems – the subset whose mothers exposed them to alcohol in utero. It is postulated that the split in the research between ACAs who seem to have no problems and ACAs who seem to have a great many problems may be divided along the lines of prenatal exposure. Since most of the studies are done on paternal drinkers, the maternal contribution in general is missing, and in particular the contribution of maternal drinking during pregnancy.

## 2. Secondary Disabilities of FAS

Ann Streissguth's work on secondary disabilities (Streissguth et al., 1996) shows that, without proper identification and intervention, people who have clearly been damaged by prenatal alcohol exposure are very likely to have significant problems in academic, legal, financial, interpersonal and mental arenas. The results of this study confirm her finding and expand it to a relatively higher-functioning population; even in this group of voluntary self-improvers, the pattern is clear: as exposure to prenatal alcohol goes up, level of functioning goes down. Participants' comments frequently sounded the theme that if they had only known it wasn't their fault (that they didn't do well in school, kept forgetting things, couldn't read social cues, etc.), and that if somebody could have helped them work around these difficulties, things would have been different. This is exactly the point of the present study.

## C. The Research Questions and the Scales

The two research questions were: 1) whether prenatal alcohol damage could be found in this group and then correlated with increasing exposure, and 2) whether the S-PAD could spot prenatal alcohol damage in higher-functioning ACAs where existing

scales might not. The scales used in this study are examined to see how the present results match up to expectations from the literature. (The S-PADs and the Laundry List are addressed in detail in the Study Question section above.)

The Fetal Alcohol-Affected Student Checklist for Educational Programming: This scale is used with preschool children who have already been identified as probably alcohol-affected, for the purpose of aiming interventions appropriately for each child. The items on this list reflect very serious difficulties: “learns a simple task but forgets quickly,” “developmental delay was identified at 30 months of age,” “problems in gross motor control” are representative. The highest-functioning group endorsed an average of about three and a half items, or 10 %; the lowest-functioning group endorsed an average of nine items or 31%. That any of these items was endorsed by anyone in this relatively high-functioning group was surprising, as the prognosis for someone with these features would not be particularly good: brain damage is assumed. That fully 10% were endorsed by the group containing teachers and executives was extremely surprising. The results of this scale suggest that even the highest of the high-functioning people in this study had at least some isolated signs of brain damage in early childhood, and that those whose mothers drank more heavily had more.

The Fetal Alcohol Behavior Scale: The FABS, like the other screening tools used here, names the more dysfunctional manifestations of impairment; the results of this study show that while heavy and moderate exposure produced high scores on the FABS, the low-exposed, high-functioning group scored much lower, although they did endorse some of the items (all of which are related to prenatal alcohol damage). This would make sense, as the people on whom the FABS was normed have a diagnosis of either Fetal

Alcohol Syndrome or Fetal Alcohol Effects – known in other diagnostic systems as Static Encephalopathy – in either case: clear, recognizable, measurable brain damage. The FABS was not normed on or for people who may have damage to a lesser degree; the present comparison would tend to confirm this distinction and point toward continued use of the FABS with heavier-exposed, more-troubled groups of people.

The Alcohol Related Neurodevelopmental Disabilities Behavioral Checklist (ARNDD): the scores on this scale also separate the low-exposure/high-functioning people from those who were moderately to heavily exposed and functioning moderately to poorly. The ARNDD, like the others, is a blunt instrument: “Always talking,” “Too loud,” “Can’t remember from one day to the next,” “Can’t follow routine – needs reminders to get dressed, brush teeth, etc.” It was expected that high-functioning people who may be a little extra talkative, have some trouble knowing when they are, be a bit loud, have memory problems or tend to get sidetracked would not endorse such items and so would remain unidentified as having organic difficulties related to prenatal alcohol. Again it was surprising that although they were few, some signs of prenatal alcohol damage are noted even the highest-functioning end of this group.

The Information Processing Checklist Screening Instrument (IPC): scores on this scale follows a similar pattern to the ARNDD and the FABS, with heavy endorsement of the items by people in the two lower-functioning, more-exposed groups. This scale was designed for use in a criminal justice population (Doctor, personal communication, 2001); if it were used in a population such as the highest-functioning of the ACAs in this study, the results would suggest a near-absence of effect from prenatal exposure to alcohol, although again some signs are noted.

#### D. Implications for Clinical Service with ACAs

The implications of the findings of this study are simple and broad: in working with Adult Children of Alcoholics who have problems in living or who do not seem to be living up to their capacity, it is important to inquire about exposure to alcohol in utero. Then the sorting process can begin: which of the problems are organic and should be treated as such, and which are primarily psychodynamic and can be addressed through psychotherapeutic intervention. Organic problems such as difficulty reading social cues, for example, can be addressed through direct teaching and practice or other guided activities. Memory problems (which can masquerade as a symptom of passive aggression) can be addressed through a combination of self-advocacy (“If I don’t call you back it’s not that I don’t care; I just have a bad memory – give me a call”) and assistive technology, a fancy term for datebooks, clocks, reminders, signs and post-its all over the house. Poor impulse control can be managed by a pledge to oneself that important decisions be run past someone else; if impulse control is beyond managing, then medication can be considered. These are a few of the many conditions that have organic roots but may appear to be psychodynamic in nature. Treated as brain dysfunction rather than “issues,” they can be handled with a minimum of fuss, leaving time, energy and resources available for the serious work of untangling the true psychological sequelae of family dysfunction.

#### E. Limitations of the Study

Information regarding maternal drinking is often speculative in nature; this study was no exception. Although participants appeared to give the question a sincere, thoughtful answer, it is almost always difficult to know the exact drinking of someone

out of our immediate awareness, at a time before we were born. This is a limitation of all (non-prospective) FAS/E studies as it is with all studies in general that rely on retrospective information about somebody else.

Selection of participants was a general area of limitation for this study and comprises several specific aspects. First, it was much more difficult than expected to find people who satisfied the criteria. Many people whose fathers were the alcoholics of the family volunteered, having overlooked the maternal criterion. Many of the people who did satisfy the criteria of maternal drinking and were self-identified ACAs really had no idea whether or how much their mothers drank during pregnancy. Access to ACA functions was limited by 12-Step traditions, and most of the therapists contacted preferred not to offer participation to their clients. As a result, the size of the group was limited and unfortunately devoid of mothers who did not drink at all during pregnancy.

Self-selected, this group is probably not representative of the entire spectrum of Adult Children of Alcoholics. These participants are more likely to be outspoken, and possibly more interested in highlighting their problems.

Another limitation of this study is that some of the scales were constructed to be self-report; others were designed to be answered by someone else (although they have all been used in self-report situations except the one for preschool children). This may have contributed to the uncomplimentary aspect to some of the items, putting off respondents who might be described by someone else as fitting the items but who denied these characteristics in themselves.

It was intended that information on paternal and grandpaternal symptoms of learning disabilities and attentional problems would be documented in the demographic

portion of the questionnaire. As it turned out, nobody claimed any ancestral LD/ADHD – this is probably inaccurate, and may have resulted from the way the question was phrased: brief clinical terms were used rather than a more descriptive listing of symptoms.

Since none of the instruments used in this study has been normed on high-functioning populations (and only one has been normed at all), one should be cautious about generalizing the present results to the population as a whole until further research confirms the findings.

#### F. Further Analysis

Further studies similar to the present one would be more substantial, with a control group of people whose mothers were alcoholics, but who were not exposed to alcohol in utero. Without such a control, inferences about whether these scales really measure prenatal alcohol damage must be tentative. To this researcher's knowledge, none of the scales has been tested on people known to have had no prenatal alcohol exposure.

In addition, a thorough history of the presence of learning disabilities and attention deficit/hyperactivity disorder in parents and grandparents would be necessary in order to assert that brain dysfunction associated with prenatal alcohol exposure is not confounded with dysfunction from genetic sources. By the same token, other sources of brain dysfunction such as high fevers or traumatic brain injury should be thoroughly checked as well.

It would also be helpful to do item analysis of all relevant scales, especially the Laundry List, to see exactly which characteristics increase in frequency with increase in

exposure. Without this level of analysis gaps remain in our understanding of the pattern; we just know that there is one: that overall the scores increase with increasing alcohol exposure. Another analysis that would round out our understanding would include a more formal look at the relationship between level of functioning and level of alcohol exposure; informal analysis suggests that there is a clear inverse correlation, but this suggestion must remain tentative until further analysis can be done.

The S-PAD was found to be the most sensitive of the instruments used here to detect signs of prenatal alcohol damage. Determining the exact statistical significance of the S-PAD's sensitivity in comparison to the other instruments would take it out of the "It's a little better" category.

Going beyond the immediate category of improving upon and substantiating the findings in the present study opens the door to an array of possibilities so wide that a generality must suffice: further study into difficulties suffered by Adult Children of Alcoholics would benefit from a question regarding maternal drinking while pregnant, and the resulting information correlated with type and severity of symptoms.

The overarching generality is that any study which examines causality of problems of "at risk" people should at least consider prenatal alcohol as one possible source.

#### G. Concluding Remarks

The impetus for this study was to see if what seemed to be a pattern in myself and a few ACA friends and associates could be more widespread: a subtle manifestation of perceptual, attentional and cognitive features similar to the more deleterious version found in people diagnosed with fetal alcohol syndrome or effects. Adult Children of

Alcoholics have long been the subject of controversy regarding the veracity of claims that they suffer serious ill-effects from parental drinking; in any case, group or individual psychotherapy, 12-Step and self-help work, positive affirmations or other cognitive approaches have been the treatment most often recommended. Of course it has been gratifying personally to see my hunch moved a step closer to confirmation; more important is that the huge number of us who were affected in utero may be able to more directly and effectively address the issues that plague us – separating out the organic from the psychodynamic. Researchers looking into ACA-related patterns will perhaps be more likely to recognize the disparate findings (problems vs. no problems) as a reflection of the difference between brain damage and no brain damage. It would be ideal if studies on this population would reliably inquire into prenatal alcohol exposure, and take that into account when cataloguing the difficulties or lack thereof experienced by participants.

The underlying motive for all my work surrounding fetal alcohol damage, and most specifically this dissertation, has been to help lift the shame that inevitably arises when expectations are not met and the only imaginable explanation is that the person doesn't really want to meet those expectations, isn't trying hard enough, must want to drive the other crazy, has a fear of success, or is deliberately, willfully disobedient. Or – perhaps the worst – just doesn't care enough. These oppressive misattributions, so often internalized, later erupt into a whole array of maladaptive behaviors and perceptions ranging from depression to sociopathy, with substance abuse, nihilistic behavior and miscellaneous miseries sprinkled in along the way. These disabilities, secondary to the primary one of brain damage, are preventable. All we have to do is consider the possibility that the unmet expectations of self or other are a result of organic impairment,

not deliberate or unconscious opposition; then and only then can we move ahead to effective intervention.

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## VII. APPENDICES

## APPENDIX A

(Recruitment Notice)

**ADULT CHILDREN OF FEMALE ALCOHOLICS:  
HAVE YOU HAD  
PROBLEMS WITH INTIMACY OR EXTERNAL SUCCESS,  
NOW OR IN THE PAST?**

As a student of psychology and addiction-related issues, I am interested in the sources of difficulty besetting children of alcoholics, particularly children of female alcoholics. I am especially looking to see if there is a recognizable pattern of problems related to prenatal alcohol exposure.

If you are reasonably sure you know whether and how much your mother drank during pregnancy, you identify as an ACA and would like to participate in this research for a doctoral dissertation, please contact me at 650-365-0490, or email [kpage99999@aol.com](mailto:kpage99999@aol.com).

(Abbreviated Recruitment Notice Used on Sign at the Berkeley Flea Market)

ADULT CHILD OF (MOM) ALCOHOLIC?  
HELP ME GET MY DISSERTATION DONE!!

ANSWER SOME QUESTIONS;  
ABOUT HALF AN HOUR

THANK YOU!!!!!!

Informed Consent Statement

I, \_\_\_\_\_ hereby willingly consent to participate in the research project, Signs of Prenatal Alcohol Damage Among Adult Children of Alcoholics, to be conducted by Kathryn Page, M.S., under the direction of Margaret Barbee, Ph.D., faculty member at the Center for Psychological Studies.

This research will seek to identify characteristics of fetal alcohol effects in people who consider themselves to be Adult Children of Alcoholics.

I understand the procedure to be as follows:

I will fill out six questionnaires plus a Personal Information Form, and will return these to the researcher. If I have any questions I understand the researcher is available by telephone.

One of the questionnaires concerns Adult Children of Alcoholics; the other five are related to fetal alcohol damage. I understand this will take approximately half an hour to an hour to complete.

I am aware that there is potential discomfort involved in the study, arising from realizations or memories triggered by this material. If this should happen, I will be able to contact the researcher who will make provisions for me to receive professional help free of charge for a reasonable and limited time.

I understand no obligation is involved and I am free to withdraw from the study at any time. I also understand that this study may be published and that my personal identity will be protected unless I give my written consent. Otherwise, no names or identifying information will be used in any oral or written materials. I am aware that I may be quoted, in the interest of the research, when what I say would not reveal my personal identity.

I understand that I have the option to receive feedback from the results of the study.

Send me a summary of the results. Yes \_\_\_\_\_ No \_\_\_\_\_

Date \_\_\_\_\_ Signature \_\_\_\_\_

## PERSONAL INFORMATION FORM

(To be completed by the researcher)

Date \_\_\_\_\_

Name \_\_\_\_\_ Phone \_\_\_\_\_

Address \_\_\_\_\_

Email \_\_\_\_\_

Mother drank? \_\_\_\_\_

Any idea of pattern: frequency, amount, "heavy drinker,"  
etc. \_\_\_\_\_

Source of information \_\_\_\_\_

Education: highest grade completed  
\_\_\_\_\_

Employment: occupation \_\_\_\_\_

how long \_\_\_\_\_

Head trauma \_\_\_\_\_

Serious illness, high fevers \_\_\_\_\_

Parents or grandparents have learning disability? ADHD?

Who \_\_\_\_\_ Which dx \_\_\_\_\_

Adopted? \_\_\_\_\_ (info about mom would then be about the birth mom)

On a scale of 1-10, how chaotic was family life? \_\_\_\_\_

Other information volunteered \_\_\_\_\_  
\_\_\_\_\_

## APPENDIX B

LAUNDRY LIST (Woititz, 1983)

1. Adult children of alcoholics guess at what normal behavior is.
2. Adult children of alcoholics have difficulty following a project through from beginning to end.
3. Adult children of alcoholics lie when it would be just as easy to tell the truth.
4. Adult children of alcoholics judge themselves without mercy.
5. Adult children of alcoholics have difficulty having fun.
6. Adult children of alcoholics take themselves very seriously.
7. Adult children of alcoholics have difficulty with intimate relationships.
8. Adult children of alcoholics overreact to changes over which they have no control.
9. Adult children of alcoholics constantly seek approval and affirmation.
10. Adult children of alcoholics usually feel that they are different from other people.
11. Adult children of alcoholics are super responsible or super irresponsible.
11. Adult children of alcoholics are extremely loyal, even in the face of evidence that the loyalty is undeserved.
12. Adult children of alcoholics are impulsive. They tend to lock themselves into a course of action without giving serious consideration to alternative behaviors or possible consequences. This impulsivity leads to confusion, self-loathing, and loss of control over their environment. In addition, they spend an excessive amount of energy cleaning up the mess.

THE FETAL ALCOHOL BEHAVIOR SCALE (Streissguth et al., 1997)

Instructions: Please circle “yes”, “no”, or “don’t know” for each of the items below.

Communication and Speech

1. yes no don't know Loud, deep or unusual sounding voice.
2. yes no don't know Talks too much and too fast.
3. yes no don't know Interrupts; talks with poor timing in terms of the listener.
4. yes no don't know Unusual conversational topics; dwells on one or two particular subjects or speaks about unrealistic or unusual topics.
5. yes no don't know Likes to talk; the talking seems more important than the context.
6. yes no don't know Repeats certain words or phrases often.
7. yes no don't know Makes “off the wall” comments; sometimes says things that seem completely out of context.
8. yes no don't know Talks a lot but says little; is chatty but with shallow content.

Personal Manner

1. yes no don't know Klutzy: tasks often unintentionally end up in a mess; tends to upset or spill things more than normal.
2. yes no don't know Messy: paper work is smudgy and rumped; makes more of a mess eating than others the same age; unconcerned about personal cleanliness (for example, hands, face and clothes are often dirty).
3. yes no don't know Touches things and people frequently; seems to need to touch or be touched more than others.
4. yes no don't know Loves to be the center of attention; draws attention to self.
5. yes no don't know Tends to lose or misplace things a lot.

Emotions

1. yes no don't know Has rapid mood swings; can be happy one moment and mad or upset the next, with mood swings triggered by seemingly small things.
2. yes no don't know Overreacts to situations; emotional reactions are often stronger than you would expect.

Motor Skills and Activities

1. yes no don't know Has difficulty performing precise tasks or difficulty learning precise tasks.
2. yes no don't know Finds team sports like soccer or football difficult, or has had trouble playing on a team.

Academic/Work Performance

1. yes no don't know Has poor attention span.
2. yes no don't know Tries hard and wants to please, but the end result is often disappointing.
3. yes no don't know Has trouble completing tasks.

Social Skills and Interactions

1. yes no don't know Overly friendly with strangers.
2. yes no don't know Often demands attention or monopolizes a conversation.
3. yes no don't know Establishes superficial friendships easily but has no close friends.
4. yes no don't know Seems unaware of the consequences of his/her behavior, particularly the social consequences.
5. yes no don't know Seems unaware of or ignores "good manners", for example may pass gas or burp.
6. yes no don't know Can't take a hint; needs strong, clear commands because the fine points escape him/her.
7. yes no don't know Is physically loving and demonstrative; enjoys bodily contact more than most people his/her age; sometimes touches peers more than they prefer.
8. yes no don't know Gets overstimulated in social situations, especially in a crowded room or when strangers are present.
9. yes no don't know Shows poor judgment in whom he/she trusts.
10. yes no don't know Inappropriate interactions at home, for example with brothers or sisters, parent, family pets.
11. yes no don't know Inappropriate interactions outside the home, such as at school with teachers or other students, in the neighborhood.

Bodily or Physiologic Functions

1. yes no don't know Seems very sensitive to loud noises (for example, startles easily; does not tune out repetitive noises; seems bothered by certain sounds).
2. yes no don't know Fidgety; can't sit still.
3. yes no don't know Has had sleeping problems (such as unpredictable sleep/wake patterns; difficulty going to sleep at night, waking very early in the morning; irregular naps).
4. yes no don't know Has problems with personal hygiene; for example, forgets to bathe, wash hands, brush teeth.
5. yes no don't know Has had problems with sexual functioning, such as inappropriate masturbation; inappropriate touching of others; other unusual sexual activity.

THE ARNDD BEHAVIORAL CHECKLIST (Burd, 1999)

Check all that apply.

1. \_\_Hyperactive
2. \_\_Poor attention
3. \_\_Impulsive
4. \_\_Disorganized
5. \_\_Seems unaware of consequences of actions
6. \_\_No fear
7. \_\_Would leave with a stranger
8. \_\_Poor social skills
9. \_\_Few friends
10. \_\_Will talk or interact with anyone
11. \_\_Easily manipulated and set up by others
12. \_\_Socially inept (inappropriate speech or touching)
13. \_\_Difficulty staying on topic during conversation
14. \_\_Always talking
15. \_\_Cocktail speech—little content
16. \_\_Too loud
17. \_\_Can't remember from one day to the next
18. \_\_Below average IQ
19. \_\_Poor school performance
20. \_\_Suspended or expelled from school
21. \_\_Poor sleeper
22. \_\_Can't follow routine—needs reminders to get dressed, brush teeth, etc.
23. \_\_Temper tantrums
24. \_\_Extreme mood swings
25. \_\_Requires constant supervision
26. \_\_Been in trouble with the law
27. \_\_Inpatient treatment for mental health or substance abuse or in jail for a crime
28. \_\_Inappropriate sexual behavior
29. \_\_Poor motor skills
30. \_\_Has or needs glasses
31. \_\_Had foster care or was adopted
32. \_\_Medication for behavior—ever
33. \_\_Mother used alcohol during pregnancy
34. \_\_Mother used alcohol in last five months of this pregnancy

INFORMATION PROCESSING CHARACTERISTICS SCREENING INSTRUMENT

(Doctor, 1999)

Please put a check by the statements that apply to you.

(after 6 demographic questions)

7.  I think there is something wrong with my memory.
8.  Sometimes I do nothing when I should be working.
9.  I am often confused.
10.  My mind jumps around—I can't keep track of my thoughts.
11.  I have never been punished.
12.  It's hard for me to sit still.
13.  I do things that are dangerous just for fun.
14.  I would like to be able to control myself better than I do.
15.  I have trouble making myself slow down.
16.  I sometimes can't get much done for days, weeks and months.
17.  I'm no good—I feel hopeless.
18.  I do things without thinking about what might happen.
19.  I always tell the truth.
20.  When I am upset it is hard to calm down.
21.  I think that strangers look at me and don't like me.
22.  I don't have many friends.
23.  I usually go along with the crowd doing what others think I should.
24.  People tell me I talk too much.
25.  When I have things to do I take them very seriously.
26.  I don't know how to change my situation.
27.  I sometimes make mistakes.
28.  I am sad a lot.
29.  I don't know how to plan for my future.
30.  I don't know how to look for a job.
31.  I frequently feel startled.
32.  I forget things a lot.
33.  I don't know how to do things that most people know how to do.
34.  Sometimes other people are to blame for my troubles.
35.  I have problems knowing what time it is.
36.  I like it when my day has a regular routine. I don't like a lot of change.
37.  I always do everything I am asked to do.
38.  I often get in fights with other people.
39.  Sometimes I can do things and sometimes I can't do the same things.
40.  I lose things all the time.
41.  When somebody gives me directions I have a hard time following them.
42.  I have trouble keeping a job.
43.  I have a lot of trouble managing my money.

FETAL ALCOHOL AFFECTED STUDENT CHECKLIST FOR EDUCATIONAL  
PROGRAMMING (Spira & Evensen, date not available)

- ( ) learns a simple task but forgets quickly
- ( ) hurts others by biting, kicking, etc.
- ( ) has not developed any friendships
- ( ) seems not to hear so a hearing loss is suspected
- ( ) frequently does not attend to social/environmental stimuli
- ( ) does not follow simple commands that are given once
- ( ) does not use toys appropriately
- ( ) strong reactions to changes in routine or environment
- ( ) does a lot of lunging and darting about
- ( ) not responsive to other people's facial expressions/feeling
- ( ) has special abilities in one area of development which seems to rule out mental retardation
- ( ) does not follow simple commands involving prepositions
- ( ) severe temper tantrums or frequent minor tantrums
- ( ) hurts others by biting, kicking, etc.
- ( ) does not imitate other children at play
- ( ) does not wait for needs to be met, wants things immediately
- ( ) difficulties with toilet training
- ( ) often frightened or very anxious
- ( ) looks through people
- ( ) frequently unaware of surroundings and may be oblivious to dangerous situations
- ( ) is very destructive
- ( ) a developmental delay was identified at or about 30 months of age
- ( ) stares into space for long periods of time
- ( ) as a baby was irritable with weak sucking reflex
- ( ) as a baby had feeding difficulties
- ( ) as a baby experienced difficulty establishing regular sleeping patterns
- ( ) not afraid of strangers
- ( ) short for age
- ( ) small head
- ( ) strong need for bodily contact (patting, touching, etc.)
- ( ) problems in fine motor control
- ( ) problems in gross motor control
- ( ) trouble with sequencing (counting, etc.)
- ( ) difficulty controlling impulses
- ( ) difficulty understanding abstract concepts
- ( ) difficulty seeing sameness in daily living situations and in making generalizations

## SIGNS OF PRENATAL ALCOHOL DAMAGE (S-PAD)

Instructions: These questions are asking about your behaviors in comparison to your peers; occasionally you will be reminded of this explicitly, but keep it in mind throughout. Circle the numbers of the items that are true for you.

### Attention/Memory

1. Do you lose track of material items frequently (keys, paperwork, jewelry)?
2. Do you lose track of nonmaterial things (parking place, important dates, even sometimes relationships)?
3. Do you have trouble finishing tasks and projects, even the ones you're interested in?
4. Is it difficult for you to concretely imagine a future farther than a few days (or hours) ahead?
5. Is it hard to set and achieve goals?
6. Are you more disorganized on the whole than your peers?
7. Is it hard (or impossible) for you to put off eating, buying or doing something that you know you will regret later?
8. Do you tend to take more risks than your friends?

### Cognitive

9. Has math been much harder for you than other subjects?
10. Was your school performance significantly better in the early grades?
11. Was "Not working up to capacity" a frequent entry in your report cards?\*
12. Are you considered stubborn, sticking to a particular position in spite of lessons or logic to the contrary?
13. Were you ever diagnosed with a learning disability?
14. If you weren't diagnosed with a learning disability, have you ever thought you might have one?

### Visual-Spatial/ Sensory Integration

15. Do you tend to get lost a lot?
16. Do you spill or drop things more than others do?
17. Do you bump into or trip over things more than others do?
18. Do tags on clothes bother you?\*
19. Are you often unaware of how you feel (cold, hot, tired, thirsty, bored)?
20. Does your balance seem to be worse than other people's?

### Speech/ Communication

22. Do you like to talk, perhaps more than the average person?
23. Do you sometimes blurt things out, in marked contrast to your normal good manners?
24. Do you find yourself lying or stretching the truth when it would be just as easy not to?
25. Have you noticed a considerable difference between your abilities to "talk the talk" and "walk the walk" (more than most people)?
26. Do you get jobs more easily than you keep them?

Social Skills/ Relationships

27. Would you say you are gullible?
28. Does social interaction often feel strange, awkward or unnatural?
29. Do you find it hard to read cues—to take a hint, or to understand without words what someone is feeling?
30. Is it more difficult than you would expect to feel empathy for others?
31. Have you had to deliberately learn how to get along in a social sense?

Physical/Medical\*

32. Do you have asthma?
33. Do you have allergies?
34. Did you have frequent or severe ear infections?
35. Have you had heart irregularities?
36. Do you have any physical/birth defects that you know of?
37. Are you smaller than your parents?
38. Do you have small eyes, set far apart?
39. Do you have a very thin upper lip?
40. Is the little furrow between your nose and your upper lip more flat than most people's?

Mood

41. Can little things really set you off?
42. Do you take a long time to calm yourself back down?
43. Have you experienced significant depression?
44. Do you have mood swings beyond normal ups and downs?

Secondary Disabilities

45. Did you have trouble in school? (More than your intellectual peers)
46. Have you ever been in trouble with the law?
47. Have you ever abused substances?
48. Has it been difficult (or did it take you a long time) to establish financial independence?
49. Have you ever been fired from a job?
50. Have your sexual experiences been more numerous, risky or indiscriminate than you wish?
51. Have you taken medication for mood or other mental health problems?

\*The S-PAD-Revised is the same as the above, with these items (11, 18 and 32-40) removed. The S-PAD-R follows.

SIGNS OF PRENATAL ALCOHOL DAMAGE-REVISED (S-PAD-R) (Page, 2001)

Instructions: These questions are asking about your behaviors in comparison to your peers; occasionally you will be reminded of this explicitly, but keep it in mind throughout. Circle the numbers of the items that are true for you.

Attention/Memory

1. Do you lose track of material items frequently (keys, paperwork, jewelry)?
2. Do you lose track of nonmaterial things (parking place, important dates, even sometimes relationships)?
3. Do you have trouble finishing tasks and projects, even the ones you're interested in?
4. Is it difficult for you to concretely imagine a future farther than a few days (or hours) ahead?
5. Is it hard to set and achieve goals?
6. Are you more disorganized on the whole than your peers?
7. Is it hard (or impossible) for you to put off eating, buying or doing something that you know you will regret later?
8. Do you tend to take more risks than your friends?

Cognitive

9. Has math been much harder for you than other subjects?
10. Was your school performance significantly better in the early grades?
11. Are you considered stubborn, sticking to a particular position in spite of lessons or logic to the contrary?
12. Were you ever diagnosed with a learning disability?
13. If you weren't diagnosed with a learning disability, have you ever thought you might have one?

Visual-Spatial/ Sensory Integration

14. Do you tend to get lost a lot?
15. Do you spill or drop things more than others do?
16. Do you bump into or trip over things more than others do?
17. Are you often unaware of how you feel (cold, hot, tired, thirsty, bored)?
18. Does your balance seem to be worse than other people's?

Speech/ Communication

19. Do you like to talk, perhaps more than the average person?
20. Do you sometimes blurt things out, in marked contrast to your normal good manners?
21. Do you find yourself lying or stretching the truth when it would be just as easy not to?
22. Have you noticed a considerable difference between your abilities to "talk the talk" and "walk the walk"(more than most people)?
23. Do you get jobs more easily than you keep them?

Social Skills/ Relationships

24. Would you say you are gullible?
25. Does social interaction often feel strange, awkward or unnatural?
26. Do you find it hard to read cues—to take a hint, or to understand without words what someone is feeling?
27. Is it more difficult than you would expect to feel empathy for others?
28. Have you had to deliberately learn how to get along in a social sense?

Mood

29. Can little things really set you off?
30. Do you take a long time to calm yourself back down?
31. Have you experienced significant depression?
32. Do you have mood swings beyond normal ups and downs?

Secondary Disabilities

33. Did you have trouble in school? (More than your intellectual peers)
34. Have you ever been in trouble with the law?
35. Have you ever abused substances?
36. Has it been difficult (or did it take you a long time) to establish financial independence?
37. Have you ever been fired from a job?
38. Have your sexual experiences been more numerous, risky or indiscriminate than you wish?
39. Have you taken medication for mood or other mental health problems?