Heritability of Substance Abuse and Antisocial Behavior: A Study of Monozygotic Twins Reared Apart

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Thirty-two sets of monozygotic twins reared apart since shortly after birth (31 pairs and one set of triplets; median age at separation was 0.2 years) were interviewed separately and blindly using the Diagnostic Interview Schedule for presence of DSM-III Axis I psychiatric disorders and antisocial personality. Because the sample was recruited from a nonclinical population, predictably few subjects met criteria for such disorders. However, items counting toward diagnoses were cumulated into four scores: alcohol-related problems, drug-related problems, childhood antisocial behavior, and adult antisocial behavior. The scores showed within-scale cohesion as measured by Cronbach's coefficient α. The drug scale and both antisocial scales showed significant heritability (p < 0.1), but the alcohol scale had an estimated heritability of zero (albeit with a broad confidence interval). There appeared to be substantial commonalities in the genetic factors responsible for these traits.

Introduction

Numerous studies show that alcoholism, drug abuse, and antisocial behaviors of both childhood and adulthood run in families (e.g., Guze et al. 1967; Cotton 1979). Environmental influences have been generally adduced to explain these facts for decades, but now genetic interpretations are receiving greater scrutiny. The literature goes beyond establishing familiality of such behaviors by use of twin and adoption study designs. There is extensive twin and adoption literature on drinking and criminality. Rather than review and cite study-by-study results, we simply list key findings, as the literature is rather consistent (except for Roe 1945).

Alcohol use and abuse are at least mildly heritable.

Male transmissibility exceeds female.

Younger twin pairs resemble each other more than older pairs.

Frequency of contact in twins correlates with similarity of alcohol use/abuse.

Different patterns of abuse may be differently transmitted.

Criminal behavior is heritable.

Childhood antisocial behavior is seen in adopted away offspring of criminal parents.

There is a consistent but usually weak correlation between alcohol abuse in one relative and criminality in another relative.

Documentation of these findings can be found in excellent reviews such as that of Murray et al. (1983).

Study of Twins Reared Apart

A method combining desirable features of twin and adoption studies is the study of twins reared apart. It avoids potentially problematic assumptions about sources of twin similarity by relying on monozygotic twins reared apart (MZA) from an early age in unrelated homes. Although such twins are very difficult to find, yielding modest-sized samples, they generate more information per pair than does studying MZ and dizygotic (DZ) twins reared together. Four times as many twin pairs are needed to estimate a heritability to a given degree of accuracy using MZ-DZ comparisons than would be needed with the MZA method (Lykken et al., unpublished data). The MZA method is therefore a very useful way to investigate the role of genetic factors in psychiatric disorders.

It would be ideal to study MZA pairs, one of whom had a given disorder, i.e., using the proband method; the data's clinical relevance would be indubitable. However, this is impractical because MZAs are difficult to find. Finding MZA twins with disorders in the 1%–5% prevalence range for most psychiatric disorders would be infeasible. Instead, the study must be of such pairs as can be found, recognizing that subclinical, or at least (possibly) subthreshold manifestations of psychiatric disorders would have to be studied. When appraised in conjunction with other data (family, adoption, and other twin studies), the legitimacy of generalization from mostly normal-range variation to clinical disorder can be evaluated.

Older MZA studies have suffered from having very small samples and studying twins whose average separation age was rather late. Lange (1931) reported one concordant pair of criminal alcoholics, and Shields (1962) reported one pair concordant for alcohol disorders. Newman et al. (1937) apparently did not identify any alcoholic or antisocial twins, and Juel-Nielsen (1964) found just a few alcoholic twins, in their series of MZA twins.

Recently, Finnish (Kaprio et al. 1984) and Swedish (Pedersen et al. 1984) studies of twins reared apart have addressed alcohol use. The Swedish data concern 215 pairs separated before 10 years of age (112 pairs before 18 months) and the Finnish data are on 125 pairs separated by age 11. Finnish probandwise concordance on 30 MZA pairs for heavy drinking was 66.7%. The corresponding intraclass correlation for heavy alcohol consumption among Swedish MZAs was only 0.14 (n = 111–120 older birth cohort twins). The Swedish study intraclass R for total alcohol consumption was 0.71 in the older cohort and 0.42 in a younger cohort of n = 36–59 MZAs, as compared to only about 0.05 in the Finnish data. (Numbers vary by item analyzed). The literature on MZA twins thus conflicts on the degree to which such traits are heritable. These studies do not address criminal and antisocial behaviors or drug abuse. Moreover, the age at separation is late in many of these twin pairs.

Researchers associated with the Minnesota Study of Twins Reared Apart have also

studied psychiatric symptoms using comprehensive psychiatric personal interviews in early separated MZA and DZA twins. This report discusses our data that relate to alcohol and drug use and to antisocial behavior. We began analyzing our MZA psychiatric data with these traits because they show relatively more analyzable variation than do data on other phenotypes such as bipolar illness or psychosis.

Methods

Subjects

Subjects were 65 members of 32 sets of monozygotic sets (twins or triplets) who took part in the Minnesota Study of Twins Reared Apart during 1979–1988. Details of subject recruitment are reported in Bouchard (1984). Zygosity was determined by eight blood group systems, four serum proteins, six blood cell enzymes, fingerprint ridgecount, ponderal index, and cephalic index. Probability of misdetermining zygosity is less than 0.001 (Lykken 1978).

Assessments

Members of a family were assessed during the same week (in all but one pair), undergoing a week-long battery of medical and psychological evaluations. Measures included mental abilities, personality, psychophysiological assessments, anthropometry, photography, and medical and dental histories with additional laboratory tests. Subjects were interviewed for psychiatric problems in the afternoon of the first or second evaluation day, depending on whether they were the (designated by birth order) "A" or "B" twin. Subjects are to be evaluated on repeat occasions approximately 7–10 years apart. Relatively few sets of MZA twins have been reevaluated thus far.

The psychiatric interview was, for initial evaluations of twins (sets 1–42), a nonblind unstructured interview. In the interests of increasing replicability and decreasing possible bias of findings, later-recruited sets (and several earlier-recruited sets seen again for 9-year reevaluations) were interviewed with the Diagnostic Interview Schedule (DIS), 3rd ed. (Robins et al. 1981). This interview is fully structured and designed for use with nonclinical populations. It covers a number of DSM-III diagnoses (American Psychiatric Association 1980), of which only three concern us here: alcohol abuse/dependence, drug abuse/dependence, and antisocial personality disorder. To preserve continuity of diagnoses through the course of the study, DSM-III diagnoses were retained even after DSM-III-R was published (American Psychiatric Association 1987). The DIS interview required from 1 to 2½ hr per subject.

Interviewers

Interviewers were carefully trained by a colleague (Dorothy Hatsukami, Ph.D.) who had herself received DIS training in St. Louis. Interviewers for 14 of the sets were one of the authors (EE), who is a board-certified psychiatrist experienced in structured interviewing, or her research assistant. Latter sets were interviewed by EE or WG, a clinical psychologist experienced with various structured interviews. In no case did a single interviewer see both members of a set.

Ratings were completed blindly. Interviewers knew that they were interviewing a twin,

but were ignorant of their zygosity, as well as any other information about them. (We began recruiting DZA twins just prior to switching from unstructured to DIS interviews, so DIS interviewers did enjoy a true blind.) There was no discussion of evaluations between the interviewers until all ratings were completed, and in no case did discussion alter ratings. Twins were explicitly asked not to discuss the DIS until both had been interviewed.

Scoring

Diagnostic data and symptom count data were examined as relatively few subjects met full criteria for DSM-III disorders in the areas of substance abuse and antisocial behavior (see Results). In order to produce scores that would show more useful variation than did diagnoses, signs and symptoms counting toward DSM-III, RDC (Spitzer et al. 1978), or St. Louis group criteria (Feighner et al. 1972), diagnoses of alcohol abuse/dependence, drug abuse/dependence, the childhood aspects of antisocial personality disorder, and the adult aspects of antisocial personality disorder were amalgamated to produce four quasi-continuous scores. Because the DIS was conducted in the standard manner which requires skipping some items if key previous answers are all or almost all negative, we had to assume unasked questions would have been answered in the negative. This would tend to lower score variability which will, ceteris paribus, lower heritabilities and genetic correlations.

Data Analysis

First, we regressed normal rank scores on gender, age, and year of birth to look for possible gender and age-period-cohort effects. For the latter, we examined the regression (linear and quadratic) on age and on year of birth. Such age period and cohort effects can increase between-set variance in the traits examined. This would yield upward-biased estimates of what the heritability would be in a population balanced on gender and of a common age and birth cohort. Therefore, we corrected for gender, age, and year of birth whenever the total regression of rank score on these factors was significant at the 0.1 level.

Variance components for between- and within-family variation on these ranks scores (as outlined above) were computed using maximum-likelihood estimators. These variance components in turn yielded heritabilities by the simple formula $h^2 = r_{MZ}$ where r_{MZ} is the sample intraclass correlation, in turn equal to $\hat{\sigma}_B^2/(\hat{\sigma}_B^2 + \hat{\sigma}_W^2)$, the ratio of between-set to total variation. Standard errors of heritabilities were obtained via Fisher's z_r transformation (Lykken et al. unpublished data). Heritabilities were tested for whether they differed significantly from zero at the 0.1 level, because with 32 sets, power is still rather low even with the MZA design.

Genetic correlations and their standard errors were computed according to the method of Falconer (1981, Equations 19.3 and 19.4). All computations were made with version 6.03 of the Statistical Analysis System (1988), procedures RANK, VARCOMP, and CORR.

Summary scores showed marked skewing, as might be expected in a nonclinical population. Many subjects massed at or near the zero end of the scales. In order to obtain scores that had an approximate normal distribution for further statistical analyses (dependent on distributional assumptions), power transformations were explored to reduce

Table 1. Demographic Characteristics of Subjects

Characteristic	Mean or %	SD
Female	68.7%	
Age (range 16–68)	43.0	13.7
Age separated (range 0–4.5)	0.60	1.86
Age reunited (range 0–64)	34.0	16.2

skewness. Such transformations, however, proved inadequate so that Blom (1958) normal rank scores were used instead. These scores have the property that when normally distributed variables are transformed monotonically and Blom scores are computed from them, the original normally distributed scores will be recovered. In any case, the Blom scores will appear to be normally distributed in the sample, which is important in deriving correct variance components (and correct standard errors) in the analyses. Very similar heritabilities are found if the raw scores are analyzed instead of the Blom scores (but the variance component standard errors are not trustworthy).

Results

To date, one set of triplets and 31 pairs of twins have been interviewed with the DIS. Table 1 summarizes sample characteristics. As usually occurs in twin studies, most subjects are female. Ages ranged from 16 to 68 years with a median age of 43. Many subjects are still within the period of risk for first developing alcohol or drug problems; this is also true, but less so, for adult antisocial symptoms as studies suggest that such behaviors are most common in early adulthood. Age at separation is quite early and separations are long. Those scored zero for age at reunion are a few pairs reared in separate adoptive homes but with intermittent childhood contact.

Diagnostic Concordance

Twelve twins met criteria for alcohol abuse and/or dependence (18%), 9 for drug abuse and/or dependence (14%), and 7 for antisocial personality disorder (11%). With regard to alcohol disorders, the 12 cases were distributed in 10 pairs, for a probandwise concordance rate of 33%. The drug disorder cases were in seven pairs for a concordance rate of 36%, and the antisocial cases were in six pairs for a concordance rate of 29%. Probandwise concordance estimates the probability of having an affected co-twin, given that one is an affected twin.

These concordance rates do not provide very impressive evidence of a genetic role in the genesis of these disorders. Four competing explanations must, however, be considered. First, genes may not be very important causes of these disorders, at least not in this nonclinical sample. Second, genes might be important, but they might act through gene-environment interactions, so that genes play "permissive" rather than "mandating" roles in determining the presence of clinical disorders. Third, interviewer unreliability of diagnoses may have attenuated concordance. Fourth, the use of diagnoses as the variables analyzed may render a misleadingly low estimate of heritability for the following reason: 2 cases might have highly similarly drinking behavior and drinking problems, yet 1 may barely meet criteria whereas the other barely fails to do so. This is especially likely to

be a problem in a nonclinical sample in which those who meet criteria are unlikely to qualify with many criteria to spare.

The first possibility is a negative assertion and so can never be conclusively proven. For reasons of statistical power, the second possibility is one we will defer examining until additional twins are studied; we have systematically collected data on the rearing environments of the twins. The third and fourth possibilities can be addressed by analyzing symptom counts, aggregated as stated in the Methods section. We predicted that studying continuously distributed symptoms scores would yield higher heritabilities than clinical diagnoses because symptom scores should be more reliable than diagnoses, and because twins with similar behaviors would be counted as concordant even if they happened to fall on opposite sides of a clinical threshold. If heritability were still low in such analyses, we would be more decisively inclined to infer that genetic factors exhibit minimal influence in this sample.

Analysis of Summary Scores

Table 2 shows the psychometric characteristics of our four scales, whose names are abbreviated alcohol, drug, child, and adult in all tables. Skewing is obvious from the quartiles and is the reason for the rescaling described under Data Analysis. It is also clear that the scales are relatively cohesive, i.e., that the covariation of items within scales is relatively high. The phenotypic correlations between rank score transformed scales are substantial, indicating that common genes, common environmental factors, or both influence pairs of these traits. Table 2 also shows item-total correlations; the item names are given in the Appendix.

When we examined the scales for gender, age, and year-of-birth effects, we averaged family members together producing one observation per family. If this is not done, the natural correlation between twins can badly bias significance levels. We used one-tailed tests for the gender effect because we expected men to show higher scores on each scale. We used two-tailed tests for effects of age and year of birth. We found that the genders differed significantly on alcohol problems and childhood and adult antisocial behavior ($t(30) = 3.82$, $p < 0.001$; $t(30) = 2.36$, $p < 0.03$; and $t(30) = 2.33$, $p < 0.03$, respectively) in the expected directions. The linear + quadratic year-of-birth combined regression was significant for drug problems ($F(2,29) = 4.21$, $p < 0.03$), with more recently born subjects showing more drug problems, an effect most pronounced among the youngest subjects. As all twins were interviewed over the span of the last several years, the year-of-birth effect is very highly confounded with the age effect, but it shows a slightly stronger association with risk than does age. Hence, alcohol problems and childhood and adult antisocial behavior were gender corrected and drug problems year-of-birth corrected by linear + quadratic regression.

Given the presence of triplets in the sample, the usual analysis of variance (ANOVA) procedures are known to be nonoptimal for computing variance components and heritabilities. The optimality of ANOVA variance component estimators is only proven for balanced data, e.g., only twins in the sample and no twin uninterviewed. For this reason we used maximum likelihood (ML) estimators instead. Table 3 gives the estimated variance components with asymptotic standard errors (SE) as well as the estimated heritabilities, which are just intraclass correlations. Alcohol problems show little between-family variability and hence small heritability. The other scores show appreciable between-family variation; all their heritabilities are significant at the 0.1 level.

Table 2. Descriptive Statistics for Summary Scores

| Statistics | Summary score | | | |
	Alcohol	Drug	Child	Adult
Mean (SD)	1.75(3.51)	0.98(2.09)	2.41(1.82)	1.22(1.51)
Quartiles	0,0,1	0,0,1	1,2,3	0,1,2
Coefficient α	0.91	0.89	0.71	0.62
Item-total correlations[a]				
1	0.72	0.61	0.24	0.30
2	0.76	0.76	0.64	0.42
3	0.48	0.83	0.18	0.49
4	0.74	0.64	0.20	0.45
5	0.68	0.54	0.44	0.38
6	0.72	0.64	—	0.48
7	0.62	0.44	0.01	0.13
8	0.48	0.50	0.40	0.26
9	0.41	0.74	0.48	0.15
10	0.12	0.64	0.23	−0.03
11	0.56	0.80	0.65	0.42
12	0.38		0.44	0.20
13	0.46			
14	0.68			
15	—[b]			
16	—			
17	—			
18	0.71			
19	0.76			
20	0.65			
21	0.76			
22	0.64			
23	—			
24	0.64			

[a]See Appendix for item names.
[b]Denotes item with no sample variation.

Table 4 gives phenotypic correlations between scores (above diagonal), heritabilities estimated by ANOVA methods (rather than ML; on diagonal) for consistency with the genetic correlations (below diagonal). The phenotypic correlations are ordinary Person rs between scores. The genetic correlations combined with corresponding environmental

Table 3. Maximum Likelihood Variance Components for Rank Scores

| Component | Summary score | | | |
	Alcohol	Drug	Child	Adult
Between (SE)	0.060 (0.093)	0.22 (0.096)	0.30 (0.14)	0.20 (0.13)
Within (SE)	0.47 (0.12)	0.27 (0.068)	0.43 (0.11)	0.54 (0.13)
Heritability	0.11	0.45[a]	0.41[a]	0.28[a]
90% Confidence Interval	0,0.39	0.18,0.65	0.14,0.62	0,0.52

[a]$p < 0.10$.

Table 4. Phenotypic Correlations, Heritabilities, and Genetic Correlations

Score	Alcohol	Drug	Child	Adult
Alcohol	0.13 ± 0.030	0.258	0.27	0.28
Drug	0.78 ± 0.027	0.46 ± 0.019	0.48	0.62
Child	0.54 ± 0.053	0.87 ± 0.0080	0.42 ± 0.21	0.47
Adult	0.75 ± 0.044	0.53 ± 0.031	0.62 ± 0.030	0.29 ± 0.026

± Standard error.

correlations account for the phenotypic correlations. Such genetic correlations have sometimes been interpreted as if they directly indicated the extent of overlap in sets of genes responsible for two traits. Carey (1988) has recently shown how low genetic correlations can occur even with high overlap. However, the converse is not true; that is, when high genetic correlations are found, it can be inferred that two traits share common genetic causes. The figures in Table 4 suggest that all the pairs of scores analyzed here do, in fact, share some causal genes in common. In particular, the Table shows that the genes contributing to drinking behavior and drug use overlap strongly.

It may seem paradoxical to use a high genetic correlation (e.g., 0.778 for alcohol and drug problems) to explain a much smaller phenotypic correlation (0.258). However, the actual relationship between genetic correlations, environmental correlations, and phenotypic correlations is such that when a genetic correlation between two traits is high, but at least one trait's heritability is low, then phenotypic correlations may be low (absent compensating environmental correlations). This population of twins suggests that alcohol abuse is under such a modest degree of genetic control that the phenotypic correlation is low even in the face of substantial genetic correlation. The other genetic correlations are also substantial and may point to a common core set of genes for all these reported behaviors.

Of note, Tables 3 and 4 show the power of the MZA design. The standard errors in Table 3 would be four times larger if the traditional MZ-DZ design were used with the same total number of twin pairs.

Discussion

Some limitations of the present study should be noted. First, some twins are young and so some pairs will turn out to be only pseudodiscordant. Second, the temporal stability of the interview data have not been assessed, so that some discordance could be due to unreliable measures. Third, the sample is modest, even though the MZA design multiplies statistical power. Fourth, more complex genetic models, which might lead to a more accurate theory of the genesis of substance abuse and antisocial behavior, have not been fitted or tested. In particular, environmental influences and gene-environment interaction implicated in the genesis of alcohol problems (Cadoret et al. 1985; Cloninger et al. 1981) have not been addressed.

Collection of further data is ongoing so that all these questions can be answered with maximal precision. The sample size will greatly expand: we are reinterviewing twin pairs originally seen in nonblind interviews (blindly with the DIS), and over 100 reared-apart MZ and DZ pairs have been identified. Structured interviews are to be repeated on twins

at 7- to 9-year follow-ups after initial evaluation to look for incident cases of clinical disorders and to assess temporal stability of previously documented problems. Dizygotic twins are being recruited as well (we have currently interviewed 38 pairs). This new body of data will provide independent tests of model fit, enhance statistical power, and also allow for the fitting of more sophisticated models. Environmental indices, such as parental drinking behavior and psychopathology, are also being gathered on all twins, these will allow for the estimation of gene-environment interaction and correlation as well as some measure of statistical control over correlated twin rearing environments.

The objection might be raised that a sample of twins recruited for study is liable to selection biases. Healthier-than-average twins could be more likely to participate, lowering our yield of diagnostically positive twins. However, our rates are as high as or higher than those in the general population [Epidemiological Catchment Area (ECA) data; Robins et al. 1984]—11%, 5%, and 2% for definite alcohol abuse or dependence, probable drug abuse or dependence, and definite antisocial personality, respectively (weighted combination of ECA gender-specific rates to match our gender ratio). Hence, we think that we probably do not recruit a "supernormal" sample with respect to the traits studied in this report. The high rate of antisocial personality is puzzling, and we have no explanation for it at present; our sample is still small enough that sampling variation could be the cause.

Despite these limitations, the present data demonstrate that antisocial behavior, defined much more broadly than just commission of criminal acts, is heritable. Problems related to drug abuse are likewise heritable. Our data conflict somewhat with the literature on alcohol problems, with our composite measure showing lower heritability. Due to the broad confidence interval on this quantity, we cannot say that we do not replicate the results of others.

Previous literature established the heritability of these kinds of problems and symptoms; our data generally confirm it using the stringent MZA design. Of perhaps even greater relevance is the demonstration of relatively high genetic correlations between these kinds of problems. This implies that in certain kinds of studies (e.g., pedigree analyses) it may at times be advisable to count individuals with apparently different symptoms as affected by the same underlying disease. The degree of genetic influence on alcohol problems is so low in our data that a high genetic correlation between alcohol and other problems is consistent with a modest phenotypic correlation.

The significant genetic correlation between adult and childhood antisocial behaviors demonstrates a common thread between them, one that is not due simply to common environmental causes. Therefore, the St. Louis group, RDC, and DSM-III convention of requiring childhood and adult manifestations of antisocial behavior is genetically supported.

One possibility deserves investigation in clinically ill twin or twin-family populations, namely, that the common genetic factors between disorders seen here are polygenic risk-modifying gene systems. They could, for example, represent sensation-seeking or impulsivity genes. There could be, in higher-risk populations, specific genes segregating which conduce to antisocial behavior, alcoholism, or drug dependence. Some of these genes could have a strong effect. In a nonclinical sample like the present one, such genes will be absent or at least very hard to detect. Therefore, our data do not refute the hypothesis that there are specific genes for specific substance dependencies or for antisocial personality without substance dependence.

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Appendix

Alcohol 1 = had to have a drink to function
Alcohol 2 = couldn't stop drinking
Alcohol 3 = tried to control drinking
Alcohol 4 = binges/benders
Alcohol 5 = heavy drinking—20/day once or 7/day for a week
Alcohol 6 = blackouts
Alcohol 7 = had illness, kept on drinking
Alcohol 8 = fights while drinking
Alcohol 9 = job/school problems
Alcohol 10 = lost job/kicked out of school
Alcohol 11 = drunken driving
Alcohol 12 = arrested
Alcohol 13 = family objected
Alcohol 14 = shakes
Alcohol 15 = seizures
Alcohol 16 = DTs
Alcohol 17 = hallucinations
Alcohol 18 = thought self drank too much
Alcohol 19 = saw doctor
Alcohol 20 = professional objected
Alcohol 21 = drink before breakfast
Alcohol 22 = neglected responsibilities while drunk
Alcohol 23 = 2+ benders
Alcohol 24 = liver, stomach trouble, neuropathy, pancreatitis, or memory problem

Drug 1 = took drug
Drug 2 = took drug 5+ times
Drug 3 = daily use >2 weeks
Drug 4 = needed drug
Drug 5 = couldn't cut down
Drug 6 = tolerance
Drug 7 = withdrawal
Drug 8 = fits, overdose, cough, infection, etc.
Drug 9 = family, friends, job, or school trouble
Drug 10 = psychological problems
Drug 11 = saw MD/professional/treated self/often interfered with life

Child 1 = hooky 5+ days in 2 years (except last year in school)
Child 2 = expelled/suspended from school

Child 3   = juvenile arrest/juvenile court
Child 4   = 2+ runaways
Child 5   = lying
Child 6   = sexual intercourse before 15
Child 7   = drunk/stoned 2+ times before 15
Child 8   = stealing
Child 9   = vandalism
Child 10 = repeated grade, or underachiever
Child 11 = in trouble for school misbehavior
Child 12 = fighting

Adult 1   = 3+ jobs/5 years, or 6+ months unemployed, or 3+ day/mo absenteeism, or quit 3+ jobs
Adult 2   = neighbor kept child, or accused of neglect, or left children alone, or ran out of food money
Adult 3   = arrested 2+ times, or felony conviction, or prostitution, or pimping, or fencing, or running numbers, or dealing drugs
Adult 4   = divorced or separated 2+ times (spouse or common-law spouse), or sex with 10 people/year, or walked out on spouse
Adult 5   = instigated 2+ fights with spouse, or beat child, or 2+ fights with others
Adult 6   = sued for bad debt 2+ times
Adult 7   = wanderlust, or no fixed address for 1 month
Adult 8   = used alias and lied often
Adult 9   = DWI or drunk driving
Adult 10 = 3+ extramarital affairs
Adult 11 = used weapon in fight
Adult 12 = fired from 2+ jobs