Temporal Stability of Antisocial Personality Disorder: Blind Follow-up Study at 8 Years

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The study objective was to examine the temporal stability of the antisocial personality disorder (ASPD) diagnosis based on whether specific antisocial symptoms were considered to be related to substance abuse. A total of 407 adults who were initially part of a family study of alcoholism and sociopathy were blindly reassessed an average of 8 years later, using the Home Environment and Lifetime Psychiatric Evaluation Record (HELPER) and basing diagnoses on the clinician's best final estimate using all sources of data. "Narrow" and "broad" ASPD diagnoses were made at both times based on whether individual symptoms were counted toward diagnosis if they occurred in the setting of significant substance abuse. κ values varied from 0.31 to 0.68, with more restrictive methods of diagnosis being less stable. After deriving estimates of sensitivity and specificity of diagnosis, the probability of being a "case" could be assigned based on the reported number of conduct problems occurring before age 15 as a clinical covariate for diagnosis. We conclude that diagnosing ASPD without attempting to attribute the cause of individual symptoms to substance abuse results in substantially greater temporal stability. Using a broader definition, the diagnosis of ASPD is highly sensitive ($P = .97$) and specific ($q = 0.93$). These findings may allow more accurate diagnosis of ASPD in drug-abusing individuals.

ESTIMATES USING DSM-III-R criteria suggest that 5.8% of men and 1.2% of women will merit the diagnosis of antisocial personality disorder (ASPD) on a lifetime basis,[1,2] with approximately half of these individuals reporting ongoing antisocial behavior in the year preceding interview.[3]

This pervasive pattern of disregard for the rights of others, frequently leading to contact with the criminal justice system, and the substantial comorbidity with substance abuse[4,5] is associated with tremendous economic, social, and psychological cost.[6] Given the significant impact of the diagnosis and the current limitations in treatment, it is clear that much remains to be done in elucidating the genetic, neurobiological, and social factors that influence the risk of developing ASPD so that better methods of prevention and treatment can be developed. Accurate, reliable diagnosis is, of course, a cornerstone of such efforts.

One significant element in the validation of any psychodiagnostic construct is temporal stability, i.e., the likelihood that an individual will be rediagnosed with the same condition at some later time. If lifetime psychiatric diagnoses are used (i.e., diagnosis based on ever meeting diagnostic standards regardless of status at interview), most subjects initially unaffected would still be unaffected at reinterview, while all those initially rated as affected should remain so at reinterview. However, two sorts of change in classification are possible. Some subjects initially classified as unaffected would later be classified as affected, representing incident cases. However, any cases initially judged as affected and subsequently rated as unaffected must, by definition, represent erroneous classification.[7,8]

The stability of diagnosis, of course, may be affected by unreliable assessment. In fact, even when using structured interviews to minimize variability in ascertaining putatively objective historical behaviors (e.g., truancy, fighting, and so forth), after adjustment for chance agreement, κ values for short-term diagnostic concordance for ASPD have been found to vary considerably. Obviously, values might vary due to design, e.g., clinician versus structured interview,[9,10] comparison of two different interviews,[11] or short-term agreement between the same structured or clinical interview.[12,13] Base rates of ASPD and most likely patient characteristics are likely to differ, as well, depending on the population from which a study sample is derived, e.g., psychiatric emergency room patients,[14] psychiatric patients,[15,16] chemical dependency patients,[12] criminals,[17] or community samples.[18-20] Possibly

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because of these differences, reported interrater or short-term test-retest agreement ranges from less than 0.40 to 1.00, although most studies report κ values between 0.50 and 0.80.[9,10,12,13,15,16,18,20-26] Longer-term agreement tends to be only slightly lower, in the range of 0.60 to 0.70.[14,17,19,27]

Another contributor to the temporal instability of diagnosis may be psychiatric comorbidity, i.e., co-occurrence within the individual of two or more psychiatric disorders. In the case of ASPD, psychoactive substance use disorders (PSUDs) are particularly prominent: the presence of ASPD has been shown to increase the odds for a comorbid PSUD by a factor of 21,[28] and a substantial proportion of those in treatment for PSUDs meet diagnostic criteria for ASPD.[4,5] The phenomenologic and nosologic overlap between ASPD and PSUDs is particularly problematic, because some behaviors may meet diagnostic criteria for more than one disorder. For example, under DSM-IV, repeated instances of driving while intoxicated might be considered both reckless behavior, consistent with ASPD, as well as a symptom of alcohol abuse. More generally, individuals who repeatedly commit antisocial acts when under the influence of psychoactive substances in order to obtain them or to obtain money to purchase them may confront the clinician with a diagnostic dilemma, with the possibility of counting the symptom toward either or both diagnoses. Without rules to govern such diagnostic choices, instability in test-retest situations is inevitable.

Two recent studies illustrate this point. Carroll et al.,[25] using data from 399 cocaine abusers, contrasted three diagnostic systems with differing emphasis on "core" sociopathic traits such as a lack of remorse and the requirement of separating antisocial behavior from substance abuse. They found markedly different lifetime population prevalences, ranging from 7% of the sample using very restrictive criteria (including both childhood conduct disorder and adult antisocial behavior independent of substance abuse) to 53% of the sample using DSM-III-R criteria. They found that the most restrictive criteria were actually less stable ($\kappa = 0.14$ for Research Diagnostic Criteria [RDC] items; $\kappa = 0.32$ for restrictive RDC criteria at 1 year) than the more inclusive DSM-III-R criteria ($\kappa = 0.55$), and further found that ". . . kappas for individual RDC criteria, which required judgments regarding the relationship of ASP symptoms to substance abuse, were lower than simply rating whether or not the behavior had occurred."[25]

Using a comparable approach, Dinwiddie and Reich[29] compared "narrow" and "broad" definitions of ASPD in subjects derived from a family study of alcoholism. Comparable to the approach taken by the RDC, the narrow ASPD definition required that antisocial symptoms not be counted toward the diagnosis of ASPD if they occurred in the setting of drug or alcohol misuse, while for the broad definition, symptoms were counted toward the diagnosis regardless of the interviewer's judgment as to the relationship to substance use. It was found that although the population prevalence of the disorder was substantially changed (from 8.5% of men under the narrow definition to 37% under the broad definition), no differences between the groups were found for age at onset or type of antisocial activity or psychiatric comorbidity, with the exception of drug- or alcohol-related diagnoses. A family history of psychiatric illness also failed to differentiate the groups. Thus, whether antisocial symptoms were judged by the interviewer to be related to substance use did not affect other measures of validity of diagnosis.

Thus, there is evidence that requiring a judgment as to the relationship of substance misuse to antisocial behavior decreases interrater diagnostic agreement, thus potentially weakening the validity of current constructs. The present study addresses a related point: Does attribution of antisocial symptoms in the setting of coexistent substance abuse also affect the temporal stability of diagnosis?

METHOD

Subjects

The St. Louis Family Study of Alcoholism was begun in 1978 by T. Reich and C.R. Cloninger, and has been described in more detail elsewhere.[30] At the time of original interview (1978 to 1983), 503 proband subjects were ascertained based on treatment for alcoholism (alcoholic group) or referral from the local office of probation and parole (felon group), or were controls derived from the medical and surgical services of Washington University Medical Center. Some 1,640 adult relatives, primarily spouses or first-degree biological relatives, were also interviewed at that time. Written informed consent was obtained from all interview participants; in addition, permission was obtained from the probands to contact family members and solicit their participation.

As part of a follow-up study involving pedigree extension for linkage studies of alcoholism, certain families (mostly from the alcoholic sample) were selected for reinterview some 3 to 16 years after the original study. From that subsample, 407 subjects