

Aristotle, Galileo, and the *DSM* Taxonomy: The Case of Schizophrenia

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With the diagnosis schizophrenia used as an example throughout, the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders's* (American Psychiatric Association, 1994) approach to psychopathologic taxonomy is subjected to critical analysis as representing a fundamentally Aristotelian conception of the phenomena of mental disorders. This approach is contrasted, in the manner suggested by Kurt Lewin's early writing on the subject, with the Galileian mode of thought, emphasizing the dynamic causal matrix in which behavior occurs. Some of the positive implications of an altered view of the problem of taxonomy within the latter perspective are drawn out, and brief suggestions are made as to directions for the future.

It has been almost a century since Emil Kraepelin (1899/1937), in the sixth edition of his celebrated textbook of psychiatry, explicitly identified dementia praecox (or what we now call schizophrenia) as a specific category of mental disorder, discriminable from the undifferentiated mass of mental disorder in general, as well as from certain other discernible types of psychopathology—notably manic-depressive insanity. It would seem timely if not belated, therefore, to ask for some accounting among those professionals assuming stewardship through the ensuing decades over this purportedly most challenging of the mental health problems of humankind. The seriousness of this challenge in the United States and elsewhere appears in fact to be widely underestimated. I begin this essay with a brief, objective examination of this question. How large is the problem, and how well have researchers coped with the severe types of disorder now meriting the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) designation schizophrenia?

Extent of the Problem

Incidence and prevalence estimates for schizophrenia are widely variant (American Psychiatric Association, 1994), largely because of instability over time and place in the criteria used for identifying cases but also probably because of substantial real variations in occurrence of these conditions among differing groups (Stevens & Hallick, 1992). The most accurate data for the United States are undoubtedly those deriving from the venerable National Institute of Mental Health (NIMH) Epidemiologic Catchment Area survey. A searching analysis of these data by Tien and Eaton (1992) suggests an occurrence rate of *DSM*-defined "full" schizophrenia as high as two cases per 1,000 persons per year, an estimate that dwarfs previous, less reliably based ones. The median age for first admission is in the 20s, somewhat earlier for male than for female patients. Behavior that meets criteria for the diagnosis of schizophrenia is, thus, of rather common occurrence. What happens, then, to

this significantly large subgroup of the population who develop behavioral deviances of the schizophreniform variety?

It is important in addressing the question of outcomes with this class of disorder to be clear about the criteria to be used. The mere absence or pharmacologic suppression of the more dramatic signs associated with the diagnosis, often termed *positive symptoms*, have frequently, it would seem, been given undue weight in such assessments. This point is illustrated in some observations offered by Harvard University psychologist Roger Brown (Brown & Herrnstein, 1975). Brown had attended a meeting of a local chapter of Schizophrenics Anonymous to acquaint himself with the clinical phenomenon of schizophrenia. He describes his experience as follows.

The members each seemed to come alone, trailing in and out of the night, with almost no group acknowledgement of the successive arrivals. . . . [A remembered former student of mine] was the group's leader that night, and he began with an optimistic testimony about how things were going with him, designed in part to buck up the others. Some of them also spoke hopefully; others were silent and stared at the floor throughout. I gradually felt hope draining out of the group as they began to talk of their inability to hold jobs, of living on welfare, of finding themselves overwhelmed by simple demands. Nothing bizarre was said or done; there was rather a pervasive sense of inadequacy, of lives in which each day was a dreadful trial. Doughnuts and coffee were served, and then each one, still alone, trailed off into the Cambridge night.

What I saw a little of at the meeting of Schizophrenics Anonymous is simply that there is something about schizophrenia that the antipsychotic drugs do not cure or even remit on a long-term basis. (Brown & Herrnstein, 1975, p. 641)

Clinicians having experience with these types of patients will, I suspect, find little quarrel with the accuracy of Brown's observations. In short, what is so devastating about what is called schizophrenia is not flagrant bizarreness—not hearing things other people don't hear or expressing beliefs others find absurd—but rather the kinds of social deficits that Brown so eloquently yet concisely identified. Fenton and McGlashan (1994), in fact, referred to a "deficit syndrome" as representing the ultimate in prediction of poor mental health outcome. By contrast, Romme and Escher (1989) have reported on a well-attended "congress" of chronic hallucinators among the Dutch,

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persons described as leading entirely normal lives while denying any mental health implications in their unusual experiencing. It may also be noted in this connection that it has proven far from easy to specify in what manner "delusional" thinking differs from the "normal" variety (Oltmanns & Maher, 1988).

Certain of the main indicators of schizophrenia, diagnostically speaking, would thus appear to be of less than fundamental significance. And in fact diagnostic criteria for schizophrenia, even those of recent vintage, have a notably unimpressive record in predicting future behavior (e.g., Ciompi, 1984; Endicott, Nee, Cohen, Fleiss, & Simon, 1986; Strakowski, 1994), excepting the deficit syndrome as already noted (Fenton & McGlashan, 1994; McGlashan & Fenton, 1993). Presumably the elevation of *negative symptoms* to the status of a prime criterion for schizophrenia in the fourth edition of the *DSM* (*DSM-IV*; American Psychiatric Association, 1994) was motivated by findings of this sort. The often seemingly inexorable deterioration of communal and agentic (Freud's love and work?) functioning sometimes accompanying the other phenomena associated with the diagnosis appears indeed to be the main basis for the awesomely pessimistic reactions it often continues to inspire among professionals and citizenry alike.

From a prognosis and outcome standpoint, then, it would appear that researchers need mainly to be concerned with what is commonly referred to as the degree of "social recovery" attained by the person in the aftermath of an occurrence of psychosis having schizophreniform features. Accepting that as the criterion, however, entails the rather discouraging conclusion that, overall, the mental health professions—chiefly psychiatry in terms of effort invested over time—have not performed very well; that is, they have failed to achieve an impressive, or some would argue even an acceptable, level of success with this class of disorder. Such a conclusion, moreover, requires no qualification with respect to the availability over the past 40 years of drugs said to have antipsychotic properties. For example, long-term follow-up of patients admitted to Vermont mental hospitals with a diagnosis of schizophrenia indicates no improvement in rates of social recovery since the advent of these drugs (Harding et al., 1987a, 1987b).

Taking an even longer time perspective, Hegarty, Baldessarini, Tohen, Waternaux, and Oepen (1994) have recently published an important and notably rigorous quantitative analysis of worldwide long-term outcomes in schizophrenia from 1895 through 1991—beginning, that is, at about the time Kraepelin differentiated and identified the schizophrenic type of disorder. In the aggregate, the main outcome criteria used in this study involve, again, social recovery. The results are disconcerting in showing a social recovery rate, overall, of 40.2% and a contemporary (from 1986 through 1991) social recovery rate that is slightly lower and not significantly different from that obtained in a lengthy plateau period from 1926 through 1955. Social recovery rates did rise (to about 50%) after 1955, coincident with the introduction and widespread use of the neuroleptic drugs.

Unfortunately, recovery rates began to fall off again after 1975, an effect the investigators attribute—probably at least in part correctly—to progressive return in recent years to more stringent criteria for assigning the diagnosis. In addition, Warner (1994), using a similar research strategy but a more restrictive patient sample, has confirmed the pessimistic outcome data

reported by Hegarty et al. (1994). Indeed, the data analyzed by Warner suggests an even less encouraging role for antipsychotic drugs in achieving significant social gain among patients acquiring the schizophrenia diagnosis.

It requires extraordinary complacency, I submit, to view these results in other than an alarming light. When it is considered that clinicians may be able to restore to some minimal level of effective social functioning somewhat under 50% of the persons admitted to institutions with the diagnosis and that there has been no very substantial improvement in this expectation over a period of 7 decades, it seems to me that the time is long past to stop and examine seriously the possibility that researchers have somehow become derailed in efforts to understand the schizophrenia problem. The remainder of this essay addresses that possibility.

Misdirected Effort?

It is conceivable that the construct schizophrenia as it has developed from the time of Kraepelin to the present *DSM-IV*-era represents an altogether commendable rate of progress in precisely identifying the quarry and in gaining some understanding of its nature and some control of its manifestations. The limited success achieved in restoring victims to adequate social functioning would be due, according to this interpretation, to the extremely subtle and refractory nature of the psychopathology involved, by which, in this "decade of the brain," is usually meant some almost never precisely specified defect ("dysfunction" in *DSM* terms) in the biological functioning of the schizophrenic person's brain. As a group, persons with schizophrenia have been demonstrated to harbor a bewildering array of biological brain anomalies. Unfortunately, none has proven to be specific for that diagnosis, and most of them are shared by their nonschizophrenic close biological relatives (see Carson, Butcher, & Mineka, 1996, chap. 12, for a contemporary overview of these findings). The brute fact of the matter is that we still lack any convincing demonstration of what schizophrenia specifically entails at any level of observation beyond the diagnostic criteria themselves.

The substantive reality of some underlying disordered state of affairs that is distinctive, invariant, and isomorphic, with respect to behavior meeting diagnostic criteria for schizophrenia, is thus by no means to be taken as empirically established, despite frequent public assertions suggesting the contrary. Prominent biological researcher and self-styled "neo-Kraepelinian" spokesperson Nancy Andreasen (1984) has recently acknowledged as much in referring to schizophrenia as a "provisional construct" (Andreasen & Carpenter, 1993). My own delight in seeing this acknowledgment in print, however, was diminished somewhat on reflecting that this construct has retained its provisional status for 100 years. Not without considerable justification, *phlogiston* enjoyed a run of comparable duration over most of the 18th century, succumbing finally to a far more generative notion of the nature of combustion. Will schizophrenia have a similar fate? My own guess is that it will have to, if any fundamental progress is to be achieved.

The problems with the schizophrenia construct are not, as it turns out, limited to a variety of mere empirical embarrassments. From a scientific perspective, it is also, as typically used,

a poorly conceived idea, one that on logical grounds alone is unlikely to yield penetrating insights. I attempt to show why this is so in what follows.

DSM and the Aristotelian Mode of Thought

In an earlier critique of the DSM taxonomy (Carson, 1991a), I conjectured that the painfully slow rate of progress in understanding schizophrenia (as well as other presumed entities of disorder) may to a significant extent be the product of a type of intellectual inertia, in which originally inaccurate judgments about the core elements and the boundaries of taxa have simply been permitted to persist unchallenged. Although I continue to find this a likely source of difficulty, further reflection has convinced me that it is merely the inevitable manifestation of a far more basic problem—namely, the thoroughly Aristotelian character of the DSM effort.

As Millon (1991) has aptly noted, “the current state of psychopathologic nosology and diagnosis resembles that of medicine a century ago” (p. 245). It resembles, in other words, a field that has yet to come to grips with the natural and dynamic processes producing the (disordered) phenomena observed. The explanation of the disorder resides in the class or category to which the attendant observations are allocated, as in the lists of symptoms by which 19th-century medical students were taught to identify the various diseases then recognized. Considered in the “world hypothesis” or “root metaphor,” terms originally proposed by Pepper (1942), the approach is a type of *formism*, wherein the ultimate nature of a phenomenon is assumed to reside in the carefully discerned properties it shares with other phenomena (i.e., in its accurate categorization). Thus, for Aristotle and other early Greek philosophers, light objects tended to rise and heavy objects to fall because these properties of locomotion were inherent, respectively, in lightness and heaviness. In like manner, persons with schizophrenia hallucinate, so the implicit proposition affirms, because it is in the nature of schizophrenia for its victims to do so.

The impediments of Aristotelian thinking to sound scientific progress in psychology were explicated many years ago in a classic work by one of the field’s most seminal thinkers, Kurt Lewin (1935). In that article, in fact, Lewin made direct reference to a continuing problem in the very definition of psychopathology. Noting that Aristotle’s concepts were often heavily infused with value considerations, he went on to suggest that, “like the distinction between earthly and heavenly, the no less valuative distinction between ‘normal’ and ‘pathological’ has for a long time sharply differentiated two fields of psychological fact and thus separated the phenomena which are fundamentally most nearly related” (p. 3). As recently pointed out in important analyses by Gorenstein (1992) and Wakefield (1992a, 1992b), the field of psychopathology very much remains definitionally encumbered by seemingly inextricable issues of value. Even more important, perhaps, is Lewin’s somewhat offhand suggestion that it is a mistake to assume that different psychological principles apply to the normal and the pathological.

Of more immediate relevance to the present argument, however, are Lewin’s observations pertaining to the substitution of dimensional for categorical models as essential to the scientific achievements of the Galileian and post-Galileian eras, and his

insistence that behavioral phenomena must be understood not in terms of statistically average propensities of persons categorized in one or another fashion but rather in terms of an appreciation of the immediate and dynamic causal matrix in which the behavioral events of interest are embedded. The former of these issues, as it relates to distinguishing among various forms of psychopathology, has been the subject of much discussion in recent years, some of it encouragingly by researchers centrally involved in the development of the latest version of the DSM (e.g., Widiger, 1993; Widiger & Frances, 1985). In the end, the recently installed DSM-IV (American Psychiatric Association, 1994), like its predecessors, was organized according to a categorical format. Several formidable problems will have to be overcome (see, e.g., Clark, Watson, & Reynolds, 1995; Frances et al., 1991; Millon, 1990) before we may reasonably look forward to a thoroughgoing dimensionalization of the field, but the strictly scientific benefits of doing so remain a compelling incentive.

Lewin’s latter point, pertaining to causal analysis, is really the heart of his critique of the Aristotelian model and of his argument that scientific progress is ultimately dependent on adoption of the Galileian investigational mode. In his own words,

in the psychological fields most fundamental to the whole behavior of living things the transition seems inevitable to a Galileian view of dynamics, which derives all its vectors not from single isolated objects, but from the mutual relations of the factors in the concrete whole situation, that is, essentially, from the momentary condition of the individual and the structure of the psychological situation. *The dynamics of the processes is always to be derived from the relation of the concrete individual to the concrete situation*, and, so far as internal forces are concerned, from the mutual relations of the various functional systems that make up the individual. (1935, p. 41)

Quite clearly, Lewin is here advocating an abandonment of the *formist* or Aristotelian root metaphor in favor of what Pepper (1942) described as a *contextualist* one, in which every event is a historically unique product of its own causal matrix, and where such contextual matrices may be expected constantly to change. The same “root metaphor” would in contemporary language probably qualify as one or another version of general systems theory (see, e.g., Bronfenbrenner, 1979). The scientific revolution (Kuhn, 1970) spawned by Galileo and other “natural philosophers” of the late Renaissance and beyond is thus one in which events came to be seen not as due to the intrinsic properties of the objects involved in those events but rather as the dynamic outcome of a mix of internal and external influences (forces) immediately determining the observed behavior of those objects. For Galileo, such objects of interest ranged from balls rolling down an inclined plane to the movements of the heavenly bodies of the solar system. Moreover, he demonstrated that the laws governing such motion are potentially discoverable through precise, quantified observation and through experimentation.

For the psychologist Lewin (1935), the objects and events of interest were, respectively, living organisms (chiefly human beings) and the differing behaviors emitted by them in various organismic states and situational settings. It was Lewin’s hope to convince psychologists that, by avoiding Aristotelian think-

ing and adopting the perspective and the methodological approach epitomized in Galileo's work, they might productively transform their science in a manner similar to Galileo's transformation of Aristotelian mechanics. As is well known by historians of psychology, Lewin's message has had a profound impact on the development of American personality and social psychology, which by the late 1960s had become so thoroughly situational in focus as to provoke the question, "Where is the person in personality research?" (Carlson, 1971). The concept of "personality" does not, of course, automatically dictate an Aristotelian mode of thought, but the risk here is a seductive one in that much personality research involves one or another form of categorization of personality "types."

The conceptual distance between personality typing and psychiatric diagnosis is hazardously small, and hence, the latter field is equally at risk for adopting, inadvertently as it were, the Aristotelian thought mode. As I have already suggested, there is much reason to believe that it has often done so. For example, even the most cursory familiarity with the research literature on schizophrenia underscores the huge gulf existing between present realities and Lewin's epistemologic ideal as represented in the foregoing remarks. To a remarkable extent, in fact, what has been empirically established as reliably true of the psychobiology of "schizophrenia" derives from statistically significant but quantitatively minor mean differences between markedly overlapping criterion (i.e., persons meeting current diagnostic guidelines) and control group distributions.

"Concrete situations" are almost never seriously studied as they may bear on the behavior of persons designated to be schizophrenic, the most notable recent exception being the work on Expressed Emotion; low levels of expressed emotion among family members are associated with delayed relapse after remission from schizophreniform episodes (see, e.g., Hooley, 1985). There is also some now quite dated evidence that poorly understood personality factors in a therapist may importantly determine the (unmedicated) schizophrenic person's response to psychotherapeutic approaches (Betz, 1962; see Carson, 1967, for a review). Hypotheses relating to behavioral dependency on interactions of situations with internal "functional systems" that may be associated with the diagnosis are, in the present climate of lesion seeking, practically unheard of. The dearth of research of this sort seems particularly regrettable in view of its high potential for casting analytical light on the psychology of the schizophrenic experience.

Galileian Developmental Psychopathology

Galileo, being concerned chiefly with the understanding of motion (particularly that of falling bodies) and the demonstration of planetary heliocentricity, appears to have had little interest in historicodevelopmental processes—in the epigenesis, if any, of physical phenomena. Lewin was similarly ahistorical, perhaps even antihistorical, in his approach to psychological problems; by and large, for example, he avoided questions relating to the origins of internal structural and functional variations among behaving persons, although he was by no means loath to make such variations a central aspect of his theorizing. Researchers, therefore, have no direct model on which to base a notion of developmental processes according to the Galileian

mode of thought. If there were one, however, it seems reasonably certain that it would be one having strongly dynamic, interactive, epigenetic features.

By contrast, the deeply entrenched Aristotelian/Kraepelinian mode of thought that has dominated the field of psychiatry over most of its history has encouraged a decidedly static and undynamic view of the origins of psychopathology, perhaps particularly in regard to schizophrenia. A notable example from that history is the etiologic prominence afforded the notion of tainted genes. I think it no exaggeration to suggest that this idea remains a dominant theme in contemporary "biological" psychiatry, despite its evident problems, which include (a) the largely unexamined mysteries of the implicated pathway from tainted genes to the complex phenomena of schizophreniform behavior, (b) the progressive reduction of the "established" pairwise concordance rate for the disorder in monozygotic twins from 86% in the 1940s (Kallman, 1946) to 28% in the 1990s (Torrey, Bower, Taylor, & Gottesman, 1994), (c) the fact that in any event the biological "identity" of monozygotic twins is eliminated shortly after conception (Torrey et al., 1994), and (d) the convincing evidence of widespread genetic-environmental nonindependence (see, e.g., Plomin, Chipuer, & Loehlin, 1990).

These observations do not, of course, imply that genetic or other persistently operative biological factors have no role in the etiology of schizophrenia. The maintenance of any such position would seem to me to require denial of the pertinent empirical evidence on a truly massive scale. They do illustrate, I suggest, the considerable attraction of static and unidimensional etiologic models to modes of thought strongly influenced by an Aristotelian perspective. By contrast, an adequate understanding of the etiology of the schizophreniform disorders will almost certainly require the adoption of an elaborate and multipath model of epigenesis, quite likely (in view of their rampant heterogeneity) one allowing for considerable idiosyncrasy, such as Meehl's (1978, 1989) "bad luck" factor, in the manner in which various risk/protective elements developmentally interact to produce the outcome (Carson & Sanislow, 1993). The solution of this "epigenetic puzzle" (Gottesman & Shields, 1982) will, thus, depend on what is here termed a Galileian approach to the investigative challenge.

One should also not lose sight of the possibility, indeed likelihood, that the "course" of schizophreniform processes after onset of disorder is not determined, once and for all, by static and prior properties of the affected organism. A far more reasonable assumption is that the schizophrenic person and his or her disorder continue to respond to impinging environmental events. Considered from this perspective, the notion of a "deficit syndrome" in which a deteriorated state is the necessary and unalterable outcome must be regarded as an extremely pernicious idea—one that contains the seeds of its own fulfillment. For example, there is evidence in the longitudinal data gathered by Warner (1994), cited earlier, that such improvement as had occurred in outcomes in schizophrenia after World War II was mainly due to alterations in the psychosocial environments of mental hospitals, rather than to the advent of new drugs.

Researchers are actually beginning to see some reemergence here and there of the sort of dynamic perspective, largely abandoned in psychiatry by the 1970s that I suggest is sorely needed.

For example, there is now a wealth of evidence that many of the persons acquiring the diagnosis of schizophrenia as young adults began life with subtly compromised nervous systems, producing various neurological "soft signs" and minimally impairing neuromotor abnormalities. The causal factors involved in these anomalies appear to be quite varied, and the anomalies themselves are not uncommon in the histories of many persons who do not become psychiatric casualties (Carson et al., 1996). Depending, one may surmise, on the reactions of the affected child's social environment, these anomalies, which may be fairly obtrusive (see, e.g., Grimes & Walker, 1994; Walker, Grimes, Davis, & Smith, 1993; Walker, Savoie, & Davis, 1994), might (or might not) have detrimental effects on the youngster's personality and social development. Such detrimental effects, where they occur, might (or might not) in turn enhance the risk for a schizophreniform outcome, probably again depending on a host of other largely unknown impinging influences (see Berenbaum & Fujita, 1994; Zborowski & Garske, 1993, for other variants of this general approach).

The foregoing scenario obviously contains a large component of speculation. It is offered here not as a formal hypothesis relating to the etiology of schizophrenia but rather as an example—in fact, a probably oversimplified one—of the kind of extended and complicated pathway that appears to be involved in many if not most developmental patterns eventuating in a schizophreniform outcome. The tunnel vision encouraged by an Aristotelian world view and so endemic to this field of inquiry (Carson, 1991b) is, in my judgment, a very serious obstacle to the scientific progress of which, as earlier shown, researchers of schizophrenia are in rather desperate need.

Fixing the *DSM*

It is easy to underestimate the impact of the *DSM* and its fundamentally Aristotelian character on both the direction of psychopathologic research and the methodologies used. Two somewhat related issues seem paramount here: (a) In the present era, it is virtually axiomatic that research participants be selected according to the prevailing *DSM* diagnostic criteria, thus ensuring the continuing prominence in the literature of the field and in the cognitive schemas of its investigators of the category thus delineated, whether or not the category created or the taxonomic system of which it is a component represents maximally productive ways of organizing the phenomena addressed; and (b) investigative effort is powerfully but subtly propelled toward making the category, and the noncriterial characteristics of persons who fit it, the focus of investigation—in emulation, as it were, of Aristotle—in contradistinction to a focus on careful functional analysis relating to the origins and maintenance of the problematic behaviors enacted by individuals. For example, Paul and Lentz (1977) have demonstrated in impressive fashion the powerful effects that an alteration of reinforcement contingencies can have on the behavior of people with schizophrenia, in this case even after many years of continuous hospitalization.

In my own judgment, it cannot be seriously doubted that a strategy of directly addressing specific dysfunctional behaviors as difficulties to be eradicated and replaced with more effective techniques of social survival would be maximally effective in

terms of clinical outcomes in schizophrenia. Instead, the mental health professions have uncritically adopted the Aristotelian notion that these problematic behaviors are the manifestation of a generalized and still wholly mysterious intrinsic property existing within persons whose behavior meets certain classificatory criteria—criteria, moreover, that lack obvious internal coherence or even great stability over time (Carson & Sanislow, 1993). From this perspective, it is less than surprising that we can demonstrate little overall success in coping with the challenge "schizophrenia" presents.

It is a reasonable prediction that institutionalized psychiatry would strongly resist abandonment of a categorical taxonomic model of the present *DSM* variety. The disorders as so arrayed bear a strong analogy to classical medical diseases, and the re-medicalization of psychiatry in recent years has been quite successful in furthering certain peripheral aims of that profession, not all of them (as in all professions, including other mental health professions) necessarily favorable to scientific advance. There may be, however, a curious irony in this position.

The distinguished nuclear physicist J. Robert Oppenheimer, of World War II Manhattan Project fame, many years ago gave an address to the American Psychological Association (Oppenheimer, 1956), in which he warned psychologists not to pattern the development of their discipline after a physics that was not there anymore. He was speaking, of course, of the paradigmatic revolution involving the overthrow (absorption?) of classical physics by quantum mechanics. Whether psychologists have heeded this advice is debatable, but a variant of Oppenheimer's warning may well apply to the present situation of psychiatry: Do not pattern the development of your discipline after a medicine that is not there anymore. My nonpsychiatric medical friends, some of whom are at the cutting edge of their specialties, tell me that they no longer treat diseases; they treat patients, or more specifically, pathophysiologic and pathoanatomic systems that interfere with essential or desired functioning. On the assumption that my friends are correct and that medicine is increasingly Galileian in its conception of individual illness, psychiatry's reliance on the disease metaphor may yet prove to have been ill advised.

Politico-economic considerations aside, there appears to be ample reason from both scientific and clinical perspectives for a profound and serious reconsideration of what characteristics a good taxonomic model for mental disorders should have, as the case of schizophrenia illustrates. In my own view, there is nothing more important to the future promotion of rapid and genuine advance in the understanding of psychopathology than finding a more productive solution to the taxonomic conundrum. The challenge, I acknowledge, is a formidable one, but like many others (e.g., Clark et al., 1995), I am persuaded to begin.

As my friend Allen Frances, Chair of the *DSM-IV* Task Group, has reminded me directly (personal communication, May 10, 1994), it is much easier to be a critic in this area than it is to suggest compelling and pragmatically realistic solutions. Without question, he is correct in that surmise, and I readily admit that the many hundreds of hours I have spent mulling over this agonizing problem have produced chiefly personal frustration and enhanced humility. I do have two somewhat related suggestions, or perhaps they would be better considered predictions, about the likely form of a satisfactory solution, nei-

ther of them either new or originating with me. They are as follows.

1. I think it extremely unlikely that the apparently seamless character of human behavior, including abnormal human behavior, will ever satisfactorily yield to a categorical system of classification (Carson, 1996). Fortunately, there also appears to be no substantive need for such a system in the area of psychopathology, inasmuch as researchers would probably do as well or better by focusing on particular behavioral problems deemed to be in need of therapeutic intervention. Of course, there would sometimes be value-based controversy in rendering such judgments, but such issues already confront researchers and clinicians and, as earlier noted, may be inescapable for any currently conceivable definition of psychopathology.

2. Advances in scientific understanding have historically been, to a striking degree, dependent on increasingly precise quantification with respect to the phenomena of interest. I see no reason why it should prove to be otherwise for the science of psychopathology. On the contrary, although I do not underestimate the difficulties to be overcome, I see no acceptable long-term alternatives to (a) establishing what are the critical underlying dimensions in behavioral pathology and (b) proceeding to develop reliable and valid means of measuring persons with respect to them. The common complaint that such a cumbersome procedure applied to individual patients would unduly complicate the lives of busy clinicians is, it seems to me, without notable merit when considered in relation to the high stakes involved. Misdirection and triviality at the level of assessing, diagnosing, and conceptualizing the problems patients present will inevitably produce both compromised treatment options and, over the longer term, compromised scientific advance.

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