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**TOWARDS A DIFFERENT CLASSIFICATION OF MOOD AND ANXIETY:
A CRITICAL REVIEW OF THE HISTORY OF
PSYCHOPATHOLOGY CLASSIFICATION AND THE PSYCHOMETRIC
INVESTIGATION OF A NEW SELF-REPORT
MEASURE OF NEGATIVE AFFECTIVITY**

A Thesis in

Psychology

by

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ABSTRACT

This thesis argues for a reconsideration of the current classification of mood and anxiety disorders. It is composed of five sections. The first considers issues related to the classification of psychopathology and, specifically, the ineluctably value-laden nature of nosology and the problem of demarcation. Second, the history of mental disorder classification from antiquity to the present is evaluated with particular reference to the various editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM). Section Three begins by tracing an historical tradition of viewing anxious and depressive symptomatology as intimately intertwined. It is argued that this explicit recognition of interconnectedness dwindled with the publication and ascendancy of the third edition of the DSM, its ethos of “splitting” psychopathology into numerous disorders, and the genesis of the phenomenon of comorbidity for mental disorders. Existing alternatives to the DSM from both Western and non-Western sources are then discussed and evaluated. Fourth, recent research and theory on negative affectivity (NA) are reviewed, and linkages are made between current research and pre-DSM-III conceptions of anxious and depressive symptomatology. Finally, Section Five attempts to synthesize the previous findings and proposes an alternative dimensional system for the classification of negative affectivity. The scope of assessable NA was widened beyond DSM-IV symptoms to include concepts derived from the NA literature, past and present empirical findings, cross-cultural research, and pre-DSM-III theory. A new clinical self-report measure derived from this system, the *Inventory of Negative Affect Symptomatology – Self-Report Version (IONAS-SR)*, was created, and consists of 100 items that can be subdivided into

seven dimensional sets. *IONAS-SR* scores can be visually plotted to yield a symptomatic “profile.” Similarities and differences to existing measures are discussed as well. Initial norms were gathered and the *IONAS-SR* was psychometrically evaluated using a sample of 371 undergraduates. Internal consistency, test-retest reliability, and correlations with criterion measures were all high and discriminant validity was acceptable. The items of the *IONAS-SR* were then submitted to a principle components analysis. A five-factor solution explaining over 40% of the variance resulted. Future directions and theoretical considerations are also discussed.

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PART: I

THE CLASSIFICATION OF MENTAL DISORDERS

Introduction to Classification

Classification

Classification is both a process and the result of a process. With regard to the former, the act of classifying phenomena is a fundamental human conceptual activity. It is a way of forming abstract concepts for the purpose of understanding and delineating our environment, other individuals, and ourselves. Some thinkers, like Immanuel Kant (1996/1781), believe that the act of classification is an inevitable consequence of nativistic capacities towards organization.

Classifications are also a means of viewing the world. Like a theory or paradigm, a classification contains a series of assumptions and inferences regarding what constitutes an individual element in a system and how these individual elements do or do not interrelate (Skinner, 1981, Klein & Riso, 1993). They define, create, and confirm boundaries for the structure of phenomena and, indeed, the territory of our intellectual disciplines themselves (Sartorius, 1990). Bluntly put, classifications focus one's attention on certain phenomena and place other non-relevant phenomena in the periphery.

In the particular case of psychopathology, the focus of this work, classifications hold a primary importance because they embody our ontological and epistemological assumptions about the nature and phenomena of mental disorders and our approach to

understanding them. As embodiments of our assumptions and instantiations of our social intellectual conventions, they reflect, both implicitly and explicitly, our values and our goals.

Though often composed of abstract (sometimes extremely abstract) elements, implicit in most systems of classification/diagnosis is a pragmatic teleology. Namely, they are formalized to meet and serve human needs (these multifarious needs will be made more explicit in the sections that follow). They are functional, or aspire to be, and with regard to psychopathology, intend to ultimately hold a relevance for the “here and now” reality of clinical work.

The purposes/functions/goals of “good” diagnostic classification systems have been discussed in the extant psychological literature. What follows is a list of these functions culled from various sources and orientations. This does not mean to imply that all, or even the majority of these functions have been brought to realization in the dominant systems, but merely serve as exemplars for what “ought” to be included.

Classifications should:

- provide a nomenclature for communication among clinical professionals (Blashfield, 1984)
- provide a basis for scientific research on psychopathology. For, as Cattell (1940) put it, nosology precedes etiology (as cited in Kihlstrom, 2002)
- provide a basis for information retrieval in various books and periodicals (Blashfield, 1984)
- protect the mental health service consumer (McWilliams, 1994)
- allow for administrative and reimbursement purposes (Ustun et. al, 2002)
- provide general descriptive information on a client/patient (Blashfield, 1984)
- provide a summary of all information that is relevant for treatment (OPD Task Force, 2001)
- provide a sense of mastery over the phantasmagoria of psychopathology that would otherwise feel overwhelming
- indicate how a disorder is caused (Brown, 1996)

- provide a basis for prediction, i.e., it should possess prognostic indicators (Blashfield, 1984, McWilliams, 1994, Brown, 1996)
- allow for patient comparisons (Ustun et. al, 2002)
- provide the basic concepts for a theory of psychopathology (Blashfield & Draguns, 1976)
- provide a basis for the education of novices in the field
- create a utility for treatment planning (McWilliams, 1994, Brown, 1996)
- provide both the therapist and client a task to work on before the client trusts the therapist enough to spontaneously open up (McWilliams, 1994)
- provide information about associated features of the disorder (i.e., course, nature of onset, etc., Brown, 1996)
- predict outcome of treatment (Brown, 1996)
- facilitate the collection of statistical information (Ustun et. al, 2002)
- being akin to paradigms, they should be able to attract a sizable number of adherents. They should also be “open-ended” enough that they leave a number of problems available for new practitioners to resolve (Kuhn, 1996).
- assign common names to phenomena (i.e., denomination, Feinstein, 1972)
- be easy to understand by those who deal with commonly encountered disorders (Glatzel, 1990)
- possess stability. Changes should be introduced only when sufficient empirical or theoretical progress has been achieved
- provide definitions of specific subgroups of symptoms or dimensions of behavior that are readily identifiable by independent observers (Brown & Barlow, 2002)

As is hopefully apparent, there are a variety of goals implicit and explicit in classifications. It seems impossible or, to be more (naïvely?) optimistic, exceedingly unlikely that any one unitary system could adequately meet every goal necessary for all flavors of pathology and therapeutic orientation. However, as will be shown in a subsequent section, the values driving these various goals serve as regulative ideals. It will be argued that discussion of these regulative ideals may lead to an improvement of the current nosological situation or, at the very least, may serve to orient ourselves towards what may be at stake in the various alternatives. Before a discussion of values and classification, however, more needs to be said of the classification of psychopathology.

Models of Psychopathology Classification

Of the many possibly ways of classifying psychopathology, five basic models are usually described in the literature (Skinner, 1981). However, these exemplars usually omit an important possibility for classification (*viz.*, the omission of nosology altogether, a thought that will be discussed further below). It is perhaps more useful to consider three higher-order modes of classification in an essential way and then look towards their possible variations and combinations. The first of these modes, and probably the most well known (and commonsensically “intuitive”), is the *categorical* approach. This assumes that assigning clients/patients to a diagnostic category provides a summary of the basic information about the client. Within such an approach, an individual either does or does not have the disorder, and treatment decisions are typically made on a similar all-or-none basis. An alternative to this is the use of dimensions. Instead of utilizing a dichotomous all-or-none approach, *dimensional* models utilize a small number of salient dimensions for the purpose of assessing and visualizing individuals. In this, no sharp categorical disjunctures are assumed, only continua. A third higher-order approach to classification could be called (for lack of a better phrase) *nosological anarchy*¹. In this, classification in the traditional sense is abandoned, usually in favor of a totally individualized approach for each person that is either being researched or treated. A clear recognition of the dangers and limitations contained in the practice of traditional classification is typically present in this approach, though not necessarily so.

The categorical approach contains at least one important subtype. This is the *hierarchical* approach to classification, and it relies on a hierarchical relationship among

individual categories. The current Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) is an example of the hierarchical (categorical) model because specific diagnoses (e.g., dissociative identity disorder) are contained within higher-order categories (e.g., the dissociative disorders) that are nested within the higher-order concept of “mental disorder” itself.

The dimensional approach also possesses an important variant. The *circumplex* model is a subtype in which dimensions (usually two) wrap around each other to form a circular arrangement. An example of this would be Leary’s interpersonal circumplex of affiliation and dominance (Wiggins, 1979). In this model, an individual’s particular pattern of performance on relevant dimensions can be plotted onto a two-dimensional circular space.

The nosological anarchical approach allows for many variations, and these can be divided on the basis of intention and motivation. As these are not often discussed in the literature, a possible example will be described and evaluated in greater detail than the preceding approaches. A *principled nosological anarchy* could be said to arise from a belief that classification too greatly occludes the particularity of an individual. Thus, classification is a potentially dangerous and pointless undertaking that is to be avoided on humanitarian grounds. Individuals working within this approach readily see the dangers of concept reification, stigmatization, and “forcing” a rich clinical presentation into a box too tiny for it to fit and “live”. One could see this as the position of a private practitioner whom only utilizes classifications like the DSM-IV for third-party billing, but attempts to view the individual before him or her in the uniqueness of their therapeutic encounter.

The danger of this viewpoint is that one will necessarily (as it is impossible to come to a situation with no prejudices or prejudgments) have classifications in operation (whether they are subterranean, tacit, or unconscious) that are neither directly observable nor a potential object of focused inquiry on their own terms. Continuing this example, a clinician may be classifying and making theoretical/technical linkages between new patients and patients he or she has formerly worked with or more idiosyncratic theories and beliefs about human health and psychopathology.

Finally, there remains another model possibility, that of a *hybrid* classification (Blashfield, 1984). In this, various combinations of categorical and dimensional models and their various subtypes are utilized for the purpose of formulating novel or better classifications.

Scadding's Ladder

Two likely reasons for the heterogeneity of available models for conceptualizing psychopathology would be the relative nascency of the discipline and the extreme complexity of the subject matter. These two factors in tandem engender a great deal of uncertainty as well as potential openness/flexibility for the field. The current state of psychopathological research has been analyzed with Scadding's "ladder" of taxonomic knowledge in medicine (as discussed in Houts, 2002). Scadding posited that a hierarchy existed in the development of disease constructs in the history of medicine. In the earliest stages, disorders were specified in terms of signs and symptoms. Thus, a syndromal classification was utilized. Next, disorders were classified according to the presence of

structural anomalies. The third level of Scadding's ladder refers to disorder of function or pathophysiology. At the apex of the ladder rests etiological explanations in which failed functions are related to causal agents. It is fairly clear that the majority of psychopathological knowledge lies within the first and lowest (syndromal) level of this hierarchy. Of course, the application of Scadding's ladder to psychopathology assumes that the medical model is the most appropriate overarching conceptual frame with which to view and classify mental disorders, an assumption that has been critiqued by many. Regardless, other factors culled from various sources also lend credence to the idea that our classificatory knowledge may be more limited and uncertain than is typically acknowledged.

Categorical Proliferation as Progress?

Following the well-known progression of the various editions of the DSM (which will be dealt with more explicitly in a later section), what is perhaps most obvious is the sharp increase in the number of available categories for diagnosis. The DSM-IV (APA, 1994) describes almost 400 potential diagnoses, but the meaning of this increase and its suitability remains unclear. Does this categorical proliferation indicate progress? Does it indicate that there have been significant advances in our conceptualizations of mental distress? Several distinct sources of knowledge answer these questions in the negative and, in fact, see such categorical proliferation not only as *not* being indicative of progress, but as being, in fact, retrograde. One example comes from the history and philosophy of science. Carl Hempel, in a well-known paper on scientific taxonomy

(1965), stated that progress occurs when there is a *decrease* in the number of available taxonomic categories. The presence of a large number of categories indicates that such progress has not yet occurred. Presumably, Hempel is referring to such historical phenomena as the discernment of the syphilis/general paresis connection by Richard von Krafft-Ebing. Before syphilis was indicated in the etiology of paresis, many separate categories of paresis existed. With etiological knowledge (i.e., knowledge at the highest level of Scadding's ladder), the number of categories sharply declined, and that which was formerly seen as manifestly variegated became unified when a higher-level of understanding was attained.

Information from another intellectual discipline, that of ethnobiology, provides additional support for the idea that the current DSM and ICD nosologies may in fact be retrograde. Ethnobiology studies how native peoples (who are often preliterate) understand living things in their environment (Flanagan & Blashfield, 2002). These systems of understanding are generally referred to as "folk taxonomies." Flanagan & Blashfield (2002) argue convincingly that these folk taxonomies demonstrate a remarkably close fit to the DSM and ICD classification systems.

Folk Taxonomies and current mental disorder classifications share a number of fundamental similarities. For instance, naming and defining what is termed the "unique beginner" is extremely difficult. In a folk taxonomy, a unique beginner could be the animal "kingdom" whereas in the present context "mental disorder" is the focus. The latter superordinate category has had many attempts at definition, but criticisms of such attempts abound and will likely continue to do so. In this vein, Lilienfeld & Marino

(1995, 1999) have argued that this category of “mental disorder” lacks defining features and possesses “fuzzy” boundaries. The examination of folk taxonomies yields a similar state of affairs. In addition, folk taxonomies and the DSM/ICD systems have between 4 and 6 hierarchically structured ranks. The users of both systems have no names for the ranks. At the center of both systems are sets of categories with terse common names (e.g., panic disorder and generalized anxiety disorder) that are readily recognizable by users of the systems. Further, there exist specific subsets of these generic categories (e.g., social anxiety disorder, generalized type). In addition, both systems possess certain categories (e.g., schizophrenia) whose rank is unclear. Finally, a similarity in the number of available categories exists. In most non-western folk taxonomies, about 500 categories are generally present (Flanagan & Blashfield, 2002). The DSM-IV, as stated above, contains close to 400 diagnostic categories.

Thus, folk taxonomies and current classifications of mental disorder share a number of commonalities. In concert with a number of aspects of *Völker* thought, the methods of folk taxonomies may be a reasonable place to begin a taxonomic inquiry. However, as folk taxonomies also reflect a pre-literate and pre-scientific *Weltanschuaang*, their overt similarities with our current classifications should give one pause. It may very well be (and will be explicitly argued throughout this text) that the time has come to reconceptualize and/or collapse certain groups of mental disorders currently considered to be discrete entities.

Values and Classification

Introduction

Given current thinking in the philosophy of science, it would be difficult to argue for the existence of any epistemologically neutral and value-free domains of intellectual endeavor. This belief, however, is a relatively recent development. For the majority of the past 300 years, the presence of values in scientific endeavors has usually been conceived of as highly suspect and, indeed, both undesirable and “unscientific.” In fact, it has been contended that the Enlightenment can partially be understood as a movement towards the extirpation of values in both the traditional sense (described below) and in the sense of pre-judgments towards viewing the world in a certain manner (e.g., Gadamer, 1975, Bernstein, 1983). In the past 50 years, however, recognition of the ineradicable and ineluctable presence of values in science, and the absence of any inherent limitations to this, has been widely recognized and embraced. As Sadler (2002) stated, thinkers such as Kuhn and Feyerabend (but also Gadamer, Foucault, Kierkegaard, Nietzsche, and the Existentialists) “grabbed science by the genitals (pp. 4)” through their explicit explication of the role of values in science. Thus, values and science are not mutually exclusive, but indeed constitute a reciprocal holism in which values often appear to be the starting point and framing locus that is prior to any empirical investigation or classification.

The classification of psychopathology is certainly no exception to this. All classifications are conventions used to meet human needs and, as such, are value-laden from *any* classification’s conception to the moment of its application. At the most

fundamental level (which is the constitutive act of demarcating “normality” from “mental disorder”) it can be seen that values necessarily enter into play. Though mental disorders possess general public agreement for their level of undesirability, this notion of undesirability itself is value-laden, and necessarily so.

This is not unique to mental disorders, however, but is also contained in the general concept of physical “disease.” Whereas some may argue that the disjuncture between physical health and illness is more “objective”, this has been trenchantly critiqued (e.g., Widiger, 2002), and physical illnesses were found to hold no position of epistemic privilege. Thus, attempts to eliminate the value-laden nature of psychopathology by identifying their biological substrates are doomed to failure, as they do not circumvent the question of value.

What are values?

An understanding of value can arise through a contrast with goals. Whereas goals possess fixed endpoints and the specific path to their attainment is specifiable in advance, values are more open, intrinsic, and flexible in both their outcomes and their potential applications (Martin, Kleindorfer, & Brashers, 1987). Unlike goals, values possess an openness and flexibility and, as one pursues a value, the value itself may change in response to what is learned in the act of pursuit. In the particular context of the present essay, values can usefully be conceived of as regulative ideals. These regulative ideals serve to focus an area of inquiry on certain facets while simultaneously occluding or minimizing others.

In addition to directing action and inquiry, values are also subject to praise or blame in reference to such actions (Sadler, 2002). In a sense, values participate in a “hermeneutic circle” of continuous interplay between the values (conceived of as “wholes”) and the individual actions and thoughts deduced from the values (i.e., individual “parts” which interact with, and receive feedback from, the world). These two levels of value (*viz.*, the general and the specific) continually influence and modify one another in a “to and fro” movement as the part influences the whole and the whole influences the part.

Classification, even of simplistic phenomena/objects, can take on innumerable forms, and what is constitutive of such forms reveals underlying values. In line with psychoanalytic thought, though, the omissions may be more telling than the additions. Regardless, one can easily identify values in diagnostic systems by viewing them as answers to “should” questions (Sadler, 2002). There are numerous “should” questions in any manual such as: Should we have a diagnostic manual?; Where should the line between normal and abnormal be drawn, and should it be drawn at all?; What difficulties or problems should be specifically included and delineated in this manual?; How should this manual be arranged (e.g., categories versus dimensions)?; To what particular individuals should this manual be addressed (e.g., psychologists, psychiatrists, or the lay public)?; and How should we decide which intellectual methods and types of data are worthy of consideration in the particulars of the manual (Sadler, 2002; Sharpless, 2002)? Answers to any of these questions ineluctably rely on values for guidance.

This recognition of the importance of values is doubly important, as it has been convincingly argued (Messer & Woolfolk, 1998) that psychology and psychiatry are contemporary “directive structures.” Directive structures are cognitive frameworks that serve to channel and motivate behaviors through the articulation of values and norms. In an almost Nietzschean analysis, Messer & Woolfolk stated that these directive structures have, at least to some degree, filled the void created by the decline of previously-held belief systems. In other words, our contemporary directive structures serve as a lamppost in a time of twilight. As such, a great deal of responsibility falls upon the hands of those who would modify and formulate such structures.

Value Dichotomies in Psychiatric and Psychological Nosology

In reading the various literatures and authors of psychopathology and classification, one can discern several sets of value dichotomies that appear to be consistently present. While it is likely that these sets overlap considerably and correlate with one another, it is probably more useful to analyze these groups of dyads separately. A ubiquitous dichotomy appears to revolve around the values relating to nosological format and the number of desired categories. For instance, Tyrer (1985, 1989) and Frances et al. (1990, 1991) discussed the differences between “lumpers” and “splitters.” Nosologists who are “lumpers” utilize organizing principles to create higher-level and more encompassing nosological abstractions with few diagnostic categories. In contrast to lumpers, “splitters” prefer simpler descriptions gained from a large number of diagnoses. Thus, a fundamental question (influenced by ones values) relates to how

narrowly or broadly psychopathological units should be conceived. Relatedly, there also seems to be a value dichotomy between coherence and non-redundancy on one hand (lumpers) and discreteness and the presence of symptomatic overlap on the other (splitters). An example of the former would be classification by developmental level (e.g., Kernberg et. al, 1989; McWilliams, 1994) and an example of the latter would be the DSM-IV (1994).

Another value dichotomy appears to focus on whether reliability or validity is preferred. Obviously, both are sought out and desired, but given the complex nature of encompassing human experience, trade-offs necessarily have to occur. The past three editions of the DSM certainly emphasized reliability over validity (OPD Task Force, 2001). The psychiatrist van Praag (1992) charges the current ICD and DSM systems with overemphasizing those symptoms that are well delineated and ascertainable with very little uncertainty (i.e., the symptoms that are reliable). As a result, empirical rigor overshadows phenomenological accuracy or, to situate this dichotomy in a broader context, the objective is emphasized over the subjective. Clinically, this is viewed as unsatisfactory, as little or no guidance for practicing psychotherapy results from such a venture. However, the opposite extreme seems just as inappropriate and, indeed, reactionary. Those clinicians (described in a previous section) who eschew diagnosis entirely due to the perceived uniqueness of each client's psychopathological presentation and the inadequacy of a gross diagnostic category to adequately capture the essence of a fundamental human concern find themselves trapped in a form of solipsism. Easy communication with other therapists is hampered due to the lack of a common language

that rapidly communicates aspects of an individual's functioning in broad and sweeping terms. At its most extreme, such ideological oppositions can border on *ad hominem* attacks. As pithily described by Westen et. al (2002), "Researchers tend to view clinicians as sloppy diagnosticians who do not use structured interviews...[and] clinicians, in contrast, often view researchers as symptomatic bean counters who require precise answers to questions that patients often cannot answer adequately and that may not even be relevant to treatment planning (pp. 225)."

As with other sets of oppositional values, it appears likely that a dialectical solution should occur. Moving back and forth or, for instance, alternately lumping and splitting as new information comes to light, appears to be a reasonable conceptualization of the task of nosology. "Revaluations" of such a sort have occurred previously. The shift from DSM-II to DSM-III marked a shift from a system influenced by lumpers to a system influenced by splitters. The present essay will argue for the necessity of a return to lumping and will present a great deal of evidence in favor of this revaluation. Ultimately, however, there will be a limit on the evidence one can put forth for one's position. For regardless of whatever position in these various dichotomies that one assumes, it will be influenced by extra-scientific and extra-rational foundations. As with other areas of learned and scientific inquiry, "subjective" and aesthetic considerations will play a role, if only a subterranean one. The extent to which one's personal aesthetic sensibilities affect our perception of what particular nosological ordering appears to be "appropriate" or "beautiful," and therefore more attractive, is an intriguing issue. Examples of such conceptual and theoretical decision-making can be found throughout

the history of science (e.g., choosing between paradigms in Kuhn, 1996), but will not be dealt with here.

The Irremovability of Values

In summary, values are inherent to any domain of intellectual inquiry, and nosology is no exception. The various disagreements in the literature on the “best” methods of classification are due, at least in part, to fundamental questions of value. As Sadler (2002) notes, a discussion of values will not necessarily remove conflict and dispute, but an explicit uncovering of them will hopefully alleviate some of the potential for misunderstandings and misconceptions. In fact, such an uncovering may lead to greater understanding. In the Hermeneutic literature (Gadamer, 1975; Bernstein, 1983), a fusion of “horizons,” or what could also be termed paradigms/world views, can occur when formerly “blind” prejudices and prejudgments become “enabling.” When blind, prejudgments (including values) influence an individual without their awareness. They are not truly seen or acknowledged. In this state they are akin to unassailable intellectual dogmas. When able to be seen as prejudgments, a new openness, flexibility, or palpable sense of distance, occurs. This openness allows for the possibility of surveying other vistas. Far from limiting one’s perception or experience, enabling prejudices may provide the ability to access other viewpoints that were formerly not accessible. Such a shift may eventuate in a new and/or richer perspective.

A Question of Boundaries: Essentialist versus Nominalist Ontologies

An important question for any discipline must be the object of study's ontological status. Therefore, brief mention should be made of how the debates of nosology take part, and are embedded within, a fundamental philosophical dispute: the problem of universals. The multifarious values and arguments that come to the fore in the present subject are recapitulations of the long-standing debate between essentialists and nominalists. Essentialism is the metaphysical position that objects or entities have essences, and that categories are based on these entities. In the context of psychopathology, essentialists aspire to "carve nature at its joints" (Meehl, 1995, pp. 268) and identify discrete mental disorders by sifting through the "accidentals" they are presented with. Kraepelin would seem to be an adherent of this view (at least tacitly). However, as Kendall (1989) notes, "the old aphorism that classification is 'the art of carving nature at its joints' loses its force if nature has no joints (pp. 51)." This statement is commensurate with an ideological opponent to essentialism that is termed nominalism. Nominalists believe that there is no underlying reality to entities or objects, but that they are merely words and mental constructions. In psychopathology, nominalists believe that diagnoses are nothing more than constructs that are useful means of organizing information (Flanagan & Blashfield, 2002). Obviously, these two positions are in fundamental opposition with one another, as one is seeking a Ttruth that is valid and unchanging in nature and the other seeks what could be termed a perspectival ttruth that will in all likelihood shift and eventually be replaced. As excellent discussions of the

essentialism vs. nominalism debate can be found in various sources (e.g., Edwards, 1996), it will not be dealt with here.

The Ethics of Classification

“The physician at an insane asylum who is foolish enough to believe that he is eternally right and that his bit of reason is ensured against all injury in this life is in a sense wiser than the demented, but he is also more foolish, and will surely not heal many.”

--Kierkegaard, 1980/1844, pp. 54

As is clear from the previous section, classification is not a value-free or neutral enterprise. Instead, values provide both the genesis and trajectory for classifications. All past and present diagnostic classifications are cultural and intellectual artifacts that arise from all-too-human activities. Not only are they formulated and constructed by fallible humans, but they are also controlled by fallible humans. As the results of classification hold a great deal of import for the diagnosed, diagnoses are certainly capable of causing harm if misused. Principles and notions of right action are involved and, thus, diagnosis falls within the domain of ethics.

As Reich (1999) aptly notes, the underlying theme that is common to all highly criticized psychological and psychiatric acts (e.g., institutionalization, use of medications, behavior modification, psychotherapy, etc.) is diagnosis. A diagnostic interview (for the purpose of assigning a diagnosis) is typically the first step in both research-driven and

purely clinical endeavors. As it is such a foundational act, various consequences will inevitably follow (e.g., acceptance into an study or the development of a treatment plan in therapy). Therefore, one of the clearest examples of the potential for harm in diagnosis is when an individual is assigned an erroneous diagnosis.

There are at least three ways in which misdiagnoses can be made. The first is through an intentional act. This is where a diagnosis is assigned that is not warranted by the information that is available. Misdiagnoses of this type are typically made in order to achieve a desired end. Examples vary in their teleologies and in their level of malevolence. One instance of this could be assigning a diagnosis of adjustment disorder to an individual meeting criteria for major depression due to a discomfort in letting third-party payers know the true extent of a client's illness. A more extreme example would be the government-sanctioned misdiagnosis of political dissidents for the purpose of either limiting freedom (through such means as institutionalization) or reducing their public credibility.

Second, misdiagnoses may occur due to ignorance. Inadequate training or incomplete information about either the client or the corresponding illness may lead to unwarranted diagnoses. An example of this could be a child diagnosed with attention deficit disorder (ADD) when their difficulties concentrating and maintaining focus are more reflective of difficulties with depression or anxiety. Enormously different treatment plans and modalities could result from this failure in differential diagnosis. Examples such as this cast light upon the fact that would-be diagnosticians working within any

classification system must be adequately trained, supervised, and eventually secure licensure if they are to practice independently without supervision.

Finally, non-purposeful misdiagnoses may also occur. In this instance, the diagnostician possesses proper training and also has adequate information about the client and the illness, but provides an incorrect diagnosis because of factors that are extrinsic to the client. While there are multiple examples of this type of misdiagnosis, three will be discussed. These types of misdiagnoses can arise due to limitations in the diagnostic process itself. For instance, utilizing one of the many structured interviews developed for the DSM-IV (e.g., the SCID-I or the ADIS) can be very helpful in some respects, but the limits of these interviews manifest when they are too rigidly adhered to. Subtle (or not so subtle) characterological issues may be omitted from inquiry or not looked at as thoroughly as they should be. Or, as another instance, certain disorders that are either uncommon or not currently “in vogue” may not be assessed at all. An individual can also misdiagnose when he or she is blinded by theory to such an extent that non-paradigmatic material is not noticed. Such theories may be published and widely accepted or can be extremely idiosyncratic. In either case they may lead to misdiagnosis if accepted diagnostic conventions are not adhered to. Related to this, misdiagnoses can also be made when even a well-trained individual utilizes a diagnosis in order to transform that which is chaotic and anomalous into that which is comprehensible and comfortable (Reich, 1999). In order to secure the veneer of understanding (which is really a pseudo-understanding) and alleviate oneself from the burden of anxiety or deep thought, a reification of the diagnostic category occurs and serves to make the chaos abate.

However, regardless of the reasons behind misdiagnosis, whether they are malevolent or ignorantly benign, they all have the capacity to evoke negative consequences for the actual individual who is mislabeled.

Underlying all of the above is a question of power. Diagnosticians hold a power-privileged position and the diagnosed are in a relatively less empowered space. London likens the position of contemporary diagnosticians to that of “a secular priesthood” (as cited in Rosenhan & Seligman, 1995). Though his statement tends towards hyperbole, he does place the issue of power in sharp relief, as have others. In his “archaeology of madness,” Foucault (1987) looked to what may be the most fundamental classification in psychopathology: the particular demarcation made between sanity and insanity during the Enlightenment (i.e., “the age of reason”). Foucault identified a Nietzschean will to power operative in this fundamental demarcation and went on to outline the results of this “labeling.” In summary, such labeling served a repressive function on those who were the recipients of labels. Other examples, such as the diagnosis of “draepetomania” (the uncontrollable urge for African Americans to escape slavery) in the 19th century, clearly reveal a similar trend (Widiger, 2002). For more modern examples of diagnoses being used in a repressive manner, one need only look to the Soviet Union in the 20th century. The “Moscow School” of psychiatry and their extremely broad spectrum-based system of classifying schizophrenia is a popular example. This diagnosis of “schizophrenia” includes a range of psychopathological phenomena from what most Western would term mild neurotic symptoms to floridly active psychosis. This spacious diagnosis led to the labeling and confinement of a number of political dissidents. To make matters worse, as

is sometimes believed in the West, schizophrenia in the Moscow sense is an essentially untreatable disorder, and sufferers from its variegated symptomatology will only continue to decline further and further into madness and have little, if any, hope for convalescence (Chodoff, 1999, Reich, 1999).

Such flagrant power abuses as outlined above are summoning, but they are obviously incomplete and do not tell the whole story. Some, like Thomas Szasz and other members of what has sometimes been termed the “antipsychiatry movement,” believe that power abuses occur in the contemporary West as well. Szasz (1960) believes that the act of diagnosing is a means of preserving the status quo. He argues that psychological symptoms are moral, and not medical, concerns. Psychologists and psychiatrists (naively) believe that they are responding amorally to a physiological disorder and, therefore, they commit the logical error of reducing an “ought” to an “is” (i.e, the “naturalistic fallacy” described by the philosopher G.E. Moore, 1903). A thorough examination of Szasz’s extreme position is beyond the purview of this essay, but critiques are available (e.g., see Miller, 1992, for citations).

In addition, the ethical implications of diagnostic labeling have been called into question by social psychologists. Some of the “classic studies” have been reviewed elsewhere (Blashfield, 1984, Passer & Smith, 2001). Of particular note, however, is the issue of the acceptance of a diagnostic label. What occurs for an individual once they become aware of their new label? Many reactions to labeling are possible, and all may be worthy of discussion in therapy, as they may reveal a great deal about the individual and their internal mental life. For some, labeling may bring a modicum of comfort. A

feeling of relief may be engendered from the knowledge that one is not suffering alone in their “weird,” “shameful,” or painful problems. For others, a label may lead to gaining further understanding of their difficulties or may prompt them to research all that they can about their diagnosis to the point that they themselves become immersed in the available psychological literature. For others a diagnostic description of behavior may become internalized. In other words, a label applied from *without* secures a strong position *within* and, in effect, becomes a component of ones identity. This internalization can obviously occur to a greater or lesser degree and certainly may lead to a multitude of consequences. As one example, Wright (1991, as cited in Passer & Smith, 2001) raises the idea that labeling may in fact lead to a worsening of a client’s condition through negative self-fulfilling prophecies.

In summary, all of the discussions above indicate that diagnosis is not an activity that can afford to be taken lightly. Implications at both the societal and individual level are clearly evident.

Part II

HISTORY OF THE CLASSIFICATION OF MENTAL DISORDERS

“History, like a vast river, propels logs, vegetation, rafts, and debris; it is full of live and dead things, some destined for resurrection; it mingles many waters and holds in solution invisible substances stolen from distant soils.”

--Jacques Barzun, 2002, pp.23-24

Introduction

While an exhaustive review of the history of mental disorder classification would be outside the scope of this essay, a brief survey will be provided for the purpose of orientation and in order to situate the present work in an historical context. This section will primarily deal with the classification of psychopathology as a whole. More specific reflections on the history of conceptions of the mood and anxiety disorders will take place in the subsequent section.

There has always been some understanding of what we would currently term “mental disorder.” However, it is a relatively recent development that these disorders have been classified separately and focused upon in relative isolation from other notions of health and sickness. Previously, as will be seen, mental disorders were often seen in tandem with physical disorders.

Antiquity

As with most topics, the study of mental disorder classification can be traced back to the Greeks. Although poignant descriptions of what we would term mental disorders have been chronicled as far back as Ancient Mesopotamia and Egypt (as cited in Ellenberger, 1970 and Alexander & Selesnick, 1966), the humoral theory of Hippocrates (1952) is a watershed of ancient thought, and seems to be an appropriate starting point.

Hippocrates' humoral theory was derived from earlier philosophical thought on the composition of nature. Several of these "natural philosophies" consisted of quadripartite models that were typically composed of pairs of opposites such as fire/water/air/earth or hot/cold/wet/dry. In his work, *Ancient Medicine* (1952), Hippocrates essentially reinterprets these four forces or elements of nature into four bodily fluids (*viz.*, blood, phlegm, black bile, and yellow bile). However, this reinterpretation was not founded entirely on speculation, but was based on the empirical observation of disease processes. These processes were completely natural, and existed within the individual. And, as Plato (e.g., Phaedrus & Protagoras, 1952) noted, Hippocrates believed that it was impossible to treat any part of the body without taking the entirety of the person into account.

In concert with pythagorean notions of harmony (and the later Stoic harmonic conceptions emphasized by Marcus Aurelius, 1996, and Epictetus, 1996), Hippocrates posited that the body required a delicate balance. In particular, the 4 humours need to be balanced and devoid of significant fluctuations for health to be maintained (Robinson, 1995). Thus, the flu, epilepsy, and depression all arose out of humoral disharmonies.

Classification followed suit, and mental disorders were thought to be the result of deficits and excesses of humours. This, however, was not a purely unidirectional (*viz.* bodily imbalances leading to mental imbalances) matter. As Foucault (1987) aptly noted, reciprocity exists, and passions can instill movement and quantitative fluctuations in the humours themselves.

Ancient Latin authors also provided no dearth of speculation on classification and the nature of mental disorders. The Roman scholar-physician Celsus believed that mental disorders were not associated with particular humoural fluctuations or, as some believed, disturbances of individual body parts, but were caused by a “humoural disorder” of the entire body (pp. 185, Roccatagliata, 1986). He further believed in a tripartite classification of mental disorders (namely phrenitis [a condition seemingly akin to psychosis], melancholy, and mania). Most important of the early Latins, Claudius Galenus unified the psychiatric knowledge gained from the 5th century B.C. to the 2nd century A.D. into a comprehensive system. Galen’s conception of mental disorder had a vitalistic cast, and he believed that the soul was contained in the brain. Thus, disorders of the soul corresponded to brain structure dysfunctions. An Aristotelian Hierarchy existed as well, with the functions of memory, judgment, and imagination at the apex, and “basal” functions such as the appetites at the bottom. Galen also formulated a classification that appears very modern. For example, he discussed such disorders as melancholia (several subtypes), mania, catatonic psychosis, dissociative psychosis, and disturbances in states of consciousness within his encyclopedic framework (Roccatagliata, 1986). Also important during this time, Caelius Aurelianus classified

mental disorders into acute and chronic forms and divided the course of various disorders into precise phases. These various phases corresponded to precise therapeutic and dietary interventions (Roccatagliata, 1986).

The Middle Ages and the Renaissance

Mention must be made of what could be termed an early diagnostic manual. Though not dealing with mental disorders in an explicit way, it dealt with “witches,” a sizable portion of which probably suffered from psychological ailments. Thus, Kramer & Sprenger’s *Malleus Maleficarum* (1968/1486) is important in several respects for the purpose of this essay. Like most manuals, it was put together by experts in the field, described the salient characteristics (of witches), clearly defined how these characteristics were to be discovered, and attempted to be broad enough to be applicable throughout Christendom. In contrast to current manuals, though, it also provided a theory or “etiology” of the witch, and provided clear treatment recommendations. Unfortunately for those diagnosed, however, purgation was typically the treatment of choice.

The Enlightenment

In retrospect, the time period known as the enlightenment can be viewed as very important for the history of psychiatric classification. Foucault (1987), for instance, held that this is where our modern conceptions of “madness” originated. It is also the time when this subject matter began to be studied with methods similar to those used in the physical and biological sciences. As such, the use of empirical observations became

more pronounced than before and began to overshadow rational analysis. In spite of the observation that, “psychiatric nosology outweighed real understanding of the sources of psychological miseries (Alexander & Selesnick, 1966, pp. 108),” the Enlightenment ushered in a meticulous cataloguing and classification of disordered mental phenomena. The majority of these classifications focused on the more aberrant and unusual behaviors, but several psychiatrists concerned themselves with the ubiquity of common “neurotic” phenomena (Jaspers, 1963, Alexander & Selesnick, 1966). Various classifications, the most impressive of which appear to have been constructed by William Cullen and Robert Wyatt, arose and were disseminated during this time. Etiological speculation was as rampant as it is today, with extreme solutions ranging from civilization, as in Rousseau’s (1983) illness-free “noble savage,” to Cullen’s belief that all mental disorders were the result of some form of physiological breakdown (Alexander & Selesnick, 1966).

Interestingly, the Enlightenment appears to be the first time period where “covering laws” of the physical sciences were incorporated into psychopathology (First, Spitzer, & Williams (1989). In 1845 Ernst von Feuchtersleben posited that all psychoses are, at the same time, neuroses, but that not all neuroses are psychoses (Ibid.). Thus, a hierarchy that implicitly and sometimes explicitly remains in the present began to take shape during this time period.

The Enlightenment also produced what was probably the most systematic and sustained treatise on the classification of mental disorders up to that time. This work was penned by the preeminent philosopher, Immanuel Kant. Kant’s *Classification of Mental Disorders* (1964/1798), written towards the end of his life, catalogued what he believed

to be the major categories of mental disorder. He grouped various phenomena as disturbances of mood and disturbances of mind. He believed that the commonality that existed between the various classified disorders was the loss of a “common sense” (*sensus communis*) and the compensatory development of a “unique sense” (*sensus privates*, pp. 19). As such, he appears to have been aware of the conventional and value-laden nature of nosology, an insight that appears to have gone unnoticed until the 20th century writings of Ellis, Jung, and the Existentialists.

Following the Enlightenment, similar themes were developed, and new ones emerged. For example, a variety of degeneracy theories and corresponding classifications of psychopathology arose (e.g., Morel’s and Magnun’s theories, as cited in Pichot 1990). The works of two particular individuals during this period heavily influenced subsequent thinking on psychiatric classification, and they shall be discussed next.

Kraepelin’s and Meyer’s Influence

By far, the two men most influential for modern nosology would be Emil Kraepelin and Adolf Meyer. Kraepelin’s system of classification still retains its classificatory dominance, even today. Thus, it is necessary to reflect on several of his fundamental beliefs and claims. Of primary importance was his belief that mental diseases could be both isolated and classified (1968). This belief was in conflict with his teacher, the great psychiatrist von Gudden, who felt that the task of classifying mental disorders was an impossible one (Blashfield, 1984). Kraepelin was also much more

“psychological” than many of his predecessors, as he advocated careful behavioral analyses in tandem with the search for organic etiologies. However, psychology had its limits. As an example, Kraepelin was very critical of disciplines such as psychoanalysis, and felt that they had no place in the study of mental disorders (Blashfield, 1984). The medical model in its strictest scientific sense was aspired to, not metapsychology.

Kraepelin, at least implicitly, also assumed a platonic perspective on diseases, and this included mental diseases. In this sense, any individual instance of disease was an approximation of an ideal type (Blashfield, 1984). Taking this idea further (and it is unclear that Kraepelin would follow in this direction), diagnostic categories are ontologically valid and timeless entities that are discrete, non-overlapping and, at least potentially, distinguishable on essential grounds. Thus, nature was being “carved at its joints.”

As will be demonstrated in subsequent sections, Kraepelin’s views are still very much alive in contemporary classification. In several respects, the current edition of the DSM is a continuation and elaboration of Kraepelin’s work and reflects the basic structure of his system. Moreover, in viewing the clinical phenomena that he catalogued and classified in his *Lectures on Clinical Psychiatry* (1968, described with such detail as to still be recognizable 100 years after initial publication), he presents a very appealing view of psychopathology, and it is easy to see why the writers of DSM-III and onward owe heavy debts to Kraepelin’s perspectives.

Another important figure of the early 20th century was Adolf Meyer. Meyer’s holistic theory of psychiatry, termed “psychobiology,” differed from Kraepelin’s system

in various ways. Meyer viewed psychological disorders as “reaction sets,” or psychobiological reactions to multi-causal life stressors (Blashfield, 1984). As such, it paid homage to Darwinism, as mental illness was conceived of as an adaptation to a changing environment (Klerman, 1990). Meyer, in contrast to Kraepelin, did not believe that such reaction sets were disease entities with established courses, but viewed illness as individual deviations in adaptation. Thus, Meyer eventually came to the conclusion that classification was a fruitless endeavor and that psychologists and psychiatrists should abandon the attempt to classify individuals as a botanist would sort plants (Blashfield, 1984). As stated in his *Psychobiology* (1957), “One word diagnoses in so plastic and complex a field as human behavior are inadequate (pp. 126).” He felt that diagnoses were unimportant, and what mattered were the particular mechanisms and events that created an understanding of the individual patient (Blashfield, 1984). In his later years, Meyer publicly denounced the labeling of patients that was common in Kraepelinian and Freudian psychology (Burnham, 1977).

Meyer’s psychobiology appealed to many, although Kraepelin’s approach attained ascendancy. Meyer’s pragmatic leanings and emphasis on behavior certainly influenced fledgling behaviorists, and his anti-classificatory stance and focus on adaptation was certainly in concert with various groups of psychoanalysts. However, as will be seen, no synthesis of these two extreme classificatory viewpoints has been achieved or, more likely, no synthesis has achieved mass consensus.

The Evolution of the DSM

Introduction

In the United States the dominant diagnostic manual is the DSM. It has gone through sizable changes and numerous editions since its initial publication in 1952. In the brief overview that will be provided, several salient trends with direct importance for the present topic will become clear.

DSM-I and DSM-II

Until 1933, when the American Psychiatric Association developed a comprehensive psychiatric nomenclature, the diagnosis of psychopathology in America was fairly idiosyncratic (Malik & Beutler, 2002). Although various aggregates of professionals developed diagnostic systems, no nation-wide system existed. Following World War II, however, APA first published DSM-I (1952) for the express purpose of creating uniform diagnostic standards. In order to accomplish this goal, it provided a listing of psychological disorders and a brief description of each one. Compared to the current edition of the DSM, it was a slim volume.

DSM-I and its second edition, DSM-II (1968), were sharply criticized on several fronts. Both were based largely on a (loose) psychoanalytic theory of psychopathology that did not generalize well to a number of clinicians. It provided only three broad categories of mental illness, namely the psychoses, personality disorders, and the psychoneuroses. All three categories, especially the psychoneuroses, were heavily theory-based. Also, the two volumes did not provide enough descriptive detail to be

particularly reliable or valid, and therapists from different orientations demonstrated low inter-rater agreement (Malik & Beutler, 2002, Blashfield, 1984). In addition, both were developed by a small number of psychiatrists and were not empirically based (Kirk & Kutchins, 1992).

An Empirical Turn? DSM-III and Beyond

Given these difficulties and the lack of widespread satisfaction with the various DSMs' nosologies, an effort to massively overhaul the American diagnostic system took place. Several goals for DSM-III (1980) appeared to be tantamount: 1) increase reliability, 2) facilitate communication among professionals, and 3) prompt and emphasize empirical data collection by identifying homogenous groups (Pincus & McQueen, 2002). The goal of this manual was less a fine-tuning of DSM-II and more of a global reconsideration of the diagnostic enterprise itself.

At least 5 major changes from DSM-II to DSM-III can be identified. First, descriptive paragraphs were eliminated in favor of specific diagnostic (symptom-based) criteria. Second, a multiaxial approach (first proposed by Essen-Moller in 1961, as cited in Blashfield, 1984) was utilized, with "major clinical syndromes" such as schizophrenia placed on Axis I. It was hoped that this approach would encourage a broader grasp of client functioning through a parsing out of symptomatology from characterological functioning, general medical conditions, etc. Next, the DSM-III provided expanded descriptive information of the listed disorders, with empirical data included when available. This stemmed, at least in part, from the increased premium placed on

empirical research by the developers of the manual. Fourth, a massive reorganization of the diagnostic categories took place, and the number of available diagnoses increased dramatically (Pincus & McQueen, 2002, Malik & Beutler, 2002). Thus, this edition of the DSM was clearly the child of nosological “splitters.” Hierarchical exclusion rules that eliminated what would later be termed the problem of “comorbidity” were maintained in order to reduce the presence of unnecessary additional diagnoses. And, possibly most important, DSM-III explicitly attempted to be atheoretical. It desired to be based on empirical considerations and a consensus model or, what some have termed the BOGSAT (*viz.*, Bunch Of Guys Sitting Around a Table, Pincus & McQueen, 2002) method.

Due in large part to criticisms of the hierarchical exclusion rules and the multiple controversies encapsulated in articles published with titles such as *DSM-III: Diagnostic Delight or Nosological Nightmare* (Nathan, 1979), a revision of DSM-III took place in 1987 (DSM-III-R). The elimination of such rules allowed for the investigation of disorders that were previously lower on the hierarchy and, thus, were more difficult to identify and study if the DSM-III’s strictures were maintained by investigators. An increase in the number of diagnoses available to users of the manual also took place, but few other substantive changes occurred. It was less a change of diagnostic structure than a refinement of the existing diagnostic scheme.

The publication of DSM-IV (1994) marked a further cultivation of the DSM-III ethos with few substantial changes. In fact, Douchette (2002) feels that the refinements and fine-tunings of the DSM since DSM-III represent a Lakatosian perspective in which

the protective belt around the core beliefs of the system remain intact. Thus, certain fundamental facets of the system are inviolable, whereas others are not. For an instance of the latter, the number of diagnoses from DSM-III-R to DSM-IV again increased significantly. As discussed previously, however, it is questionable whether this indicates that progress has occurred. What had certainly improved was the international nature of the manual, as a shift was also made to facilitate communication with non-Americans. The DSM-IV taskforce worked closely with the developers of the *International Classification of Diseases and Related Health Problems* (ICD-10, 1992) so that the two systems would more closely correspond (Malik & Beutler, 2002). Further, the task force's "BOGSAT" method was supplemented with extensive literature reviews and field trials. A special effort was also undertaken to attain representative samples of minority groups.

Strengths and Weaknesses of the Current DSM Nosology

Strengths

The current DSM possesses a number of strengths and, in a number of ways, is a revolutionary diagnostic instrument. Many of these advantages result from the numerous structural changes that occurred with the genesis of DSM-III. These will be discussed first.

The categorical typology contained in the DSM has a number of advantages. These typologies are familiar to most individuals, as they are the dominant classificatory schemes used in medicine. They are also relatively easy to use, even when thorough

clinical data are absent, and serve to integrate seemingly unconnected or diverse data into coordinated wholes (Jablensky & Kendall, 2002). If it is a good diagnosis, it provides useful information in an economic manner that usually only consists of a few words (Tyrer, 1989).

Related to the categorical model, the DSM's use of codified and operationally defined criteria led to other advantageous consequences. Of prime importance, reliability of the DSM as a system has increased since DSM-II. Though the extent of the increase in reliability has been questioned (please see Malik & Beutler, 2002, for review), DSM-IV appears to be at least as reliable as DSM-III and III-R. The use of operationally defined categories and symptoms led to a clear demarcation between diagnostic and non-diagnostic or, perhaps more appropriately, mental health and mental illness. If an individual meets the minimum threshold of symptoms, the assessor assigns the diagnosis. If the threshold is not reached, a diagnosis is not assigned. The DSM clearly allows for clinical judgment to dictate borderline cases (see, for instance, DSM-IV, 1994, pp. xxiii) but, *ceteris paribus*, the categorical checklist approach rules the day. Thus, even relatively untrained individuals can fairly accurately diagnose when using structured diagnostic interviews.

It has also been stated that the DSM system, starting with the third edition, increased accountability for mental disorder diagnosticians. One cannot arbitrarily label an individual as possessing a mental disorder if this system is utilized. Use of the DSM by third-party payers and other agencies helps to ensure that only consumers who meet criteria receive the diagnostic label and the corresponding reimbursements. Thus, as

Sadler (2002) notes, “what was initially intended as a purely “scientific” enterprise nevertheless brought in a series of relatively novel moral elements to psychiatric practice (pp. 5).”

Three other structural qualities warrant discussion. First, the range of difficulties covered by the current edition of the DSM is impressive. It ranges from the various forms of schizophrenia and mental retardation to caffeine abuse. Second, the hierarchical structure of the DSM possesses the quality of increasing specificity as one moves further down the hierarchy (Blashfield, 1984). Thus, a diagnosis of major depressive disorder, recurrent and moderate, is a subtype of major depressive disorder, which in turn is contained within the mood disorder category, which in turn is a mental disorder. Third, the multiaxial approach, when utilized, provides a good deal of information about the client’s functioning that is over and above that which is garnered from a simple diagnosis. It allows other aspects of the client’s existence to be acknowledged and kept in the foreground.

With the changes begun in DSM-III, a great deal of empirical and theoretical research has resulted. The “splitting” approach allowed for fairly specific symptom clusters to be focused upon and studied. Basic scientific findings resulted from this organization (and, indeed, made the critiques of this system which will soon be discussed possible). Further, the continual modifications in each DSM edition indicated an openness to criticism in the light of new observations. And, while it is unclear to what extent the fundamental tenets of the DSM are or have been able to be criticized, such openness is crucial for any intellectual enterprise (Popper, 1962, Feyerabend, 1975;

1978). Additionally, the attempt to create a diagnostic system containing diagnostic terms that were orientationally neutral was admirable, if a bit naïve. Given the wide-range of conceptualizations of the vicissitudes of mental disorders, it is probably impossible to be non-exclusionary at a linguistic level.

Weaknesses

As with any system that would aspire to widespread application with heterogeneous users, there have been many criticisms levied at the current DSM. A number of these issues relate to questions concerning both the demarcation between normal and abnormal and the demarcation between individual disorders. Due to the monumental nature of the former question and the fact that it has been copiously reviewed in numerous sources, scant attention will be paid to it. Instead, focus will be directed towards the latter.

By its very nature, a classification system implies two or more distinct categories with which to classify phenomena. The number of available categories, the nature of the distinctions, and the evidence accepted for these categorical distinctions are critical variables in both the review and the application of such a system. As will be shown, the DSM has been criticized on all three of these fronts.

In a concise manner, Kendall (1975) summarized the difficulties with demarcation. He states that, “there is little evidence that the boundaries we have drawn between one syndrome and another lie at genuine “points of rarity”... the widespread evidence that different schools of psychiatry draw the boundaries between one syndrome

and another in different places, and the generally low reliability of psychiatric diagnoses all suggest that syndromes merge into one another without natural boundaries in between (pp. 127-128).” While the reliability question may not be as applicable to the recent DSMs, modern reviews have echoed Kendall’s statements. Spitzer (1991) argues that if empirical support were the criteria for inclusion of disorders in the DSM, over 200 individual diagnoses would be omitted. The precise boundaries between disorder categories and constructs are currently unclear and debatable (Clark, Watson, & Reynolds, 1995). And, as a good diagnosis should describe an exclusive and homogeneous population, it seems unlikely that the majority of available diagnoses could make that claim (Tyrer, 1989). Even if this claim could be made in a few cases, this limited nosology would be inadequate to meet administrative and clinical needs (Pincus & McQueen, 2002).

Other and more specific evidence of problems in the demarcation of disorders can be pointed to. For instance, the polythetic approach of the DSM has been criticized due to the presence of inter-disorder symptomatic overlap. One need only look to the symptom overlap between generalized anxiety disorder and major depressive disorder to see this in operation. Over half of the criterion C symptoms (e.g., sleep disturbance, difficulty concentrating, fatigue, irritability) of generalized anxiety disorder are found in the criterion A symptoms of a major depressive episode. Similarly, the intra-disorder variability that results from the polythetic categorical approach can lead to so much heterogeneity between individuals with the same disorder, that fundamental questions can be raised. This is especially the case with Axis II personality disorder diagnoses.

Specifically, obsessive-compulsive personality disorder requires at least 5 of 10 symptoms for a diagnosis. Therefore, it is possible that two individuals with the same diagnosis share no symptoms in common. Another issue raised previously has bearing here as well. The continued increase in the number of diagnoses raises fundamental questions. Does the 300% increase in diagnoses from DSM-I to DSM-IV indicate rapid advances in knowledge about the nature of psychopathology and our ability to recognize formerly unseen distinctions, or are other factors in operation (Houts, 2002)? Controversy over this issue continues (Brown, 1996, Tyrer, 1985). The observation of diagnostic fluctuations between disorders (discussed in depth in subsequent sections) raises additional concerns. Finally, the question of comorbidity (to be discussed in depth in a subsequent section) or, the simultaneous presence of multiple disorders at a single time, may be the single most important problem that the DSM would have to face if it were to claim essential validity.

Given the ubiquitous nature of the problem of demarcation, a number of strategies exist within the DSM to cope with this difficulty. It is unclear to what extent such solutions were formulated with this explicit goal in mind, but what is incontrovertible is the fact that they exist. Comorbidity, along with its role as a “symptom” of the problem of demarcation, can also be utilized as a solution. When hierarchical exclusion roles were lessened in DSM-III-R, comorbidity was utilized in order to capture a clinical reality (*viz.*, the presence of several sets of symptoms) that is not easily encompassed by DSM categories. As is well known (e.g., Barlow, 2002; Costello, 1993, van Praag, 1996), the simultaneous presence of 2 or more disorders is a frequent occurrence. Thus,

the assignment of “multiple diagnoses” serves to flesh out an individual’s symptomatic presentation. Such a move serves to bypass the very question of demarcation, as it posits that several discrete disorders have simultaneously coalesced in one individual. Obviously, as in medical science, disorders can and do co-occur. However, many disorders tend to frequently co-occur (e.g., disorders in general as well as the non-psychotic mood and anxiety disorders) at levels much greater than chance would allow (Boyd et. al, 1984). This suggests that fundamental assumptions of the diagnostic system (namely, the idea of diagnostic discreteness) may be incorrect (Sullivan & Kendler, 1998) and, at the very least, require further exploration.

Two other means for the DSM to cope with the problem of demarcation have also been identified. The first would be the use of “Not Otherwise Specified” (NOS) categories. A large number of individuals do not meet full diagnostic thresholds according to the DSM’s either/or psychometric criteria and particular organization of symptoms. These people are often relegated to an NOS diagnostic category, for example, psychotic disorder not otherwise specified (Malik & Beutler, 2002). Thus, NOS categories serve as a catchall diagnostic option for those individuals experiencing clinically significant distress *sans* meeting full diagnostic criteria.

Another critique related to the demarcation question has been referred to as the “pseudoscience” and “pseudoprecision” of the DSM (Houts, 2002; Pincus & McQueen, 2002). Houts opens his essay with the pronouncement that “after 25 years of following changes in the various editions of the DSMs... there is far more pseudoscience than real science in the modern DSMs (pp. 17, 2002).” Of particular relevance would be the

presence of seemingly arbitrary cut-offs for individual symptoms that are crucial for the assignment of diagnoses (Tyrer, 1989, Pincus & McQueen, 2002). For example, the duration of symptoms required for various diagnoses ranges from several days to several years. However, to many critics these cut-offs appear to have been created spuriously for the purpose of achieving a separate identity for many conditions that would otherwise not achieve such status (Tyrer, 1989; Pincus & McQueen, 2002). Presumably, such seemingly arbitrary temporal distinctions as those found between acute stress disorder (post-traumatic symptoms present for 1 month or less) and post-traumatic stress disorder (post-traumatic symptoms present for longer than 1 month) are what these theorists had in mind.

Obviously, a solution to the problem of demarcation does not seem to be forthcoming. Two extreme and possibly antithetical approaches can be extrapolated from the literature, but there remains the possibility that what are being described are “straw-man” positions. One solution appears to rely on a radically empirical approach. As Joiner & Schmidt (2002) discuss, the DSM has “gone as far as it can go (pp. 107)” because the categories included may not be real, as they were decided by committees, and not by science. Thus, the method of consensus was paramount. If a radical empirical approach was advocated, could it be viable? Given the limited state of basic knowledge in psychopathology, the prospects for this approach seem limited. At the very least, the possibilities for third-party payment would be greatly reduced. Another extreme could be conceived of as developing an all-encompassing and synthetic conception of psychopathology. If this were to be developed, demarcation problems between various

disorders would certainly decrease. However, given the complexity of the task of encapsulating and doing justice to the range of psychopathological functioning, this approach also seems chimerical.

Another important critique of the DSM refers to its influence on thinking and its ability to be influenced by political means. With regard to the former, Malik & Beutler (2002) believe that utilization of the DSM system may have the unintended effect of constraining users to “work within a less-than-optimal system of diagnosis, a situation analogous to that of expending considerable time and resources in the ascent of a single mountain, while neglecting to explore the possibility that even higher peaks may exist nearby (pp. 8).” This is an accurate assessment of the DSM. However, the same charge could be levied at *any* system of classification. All interpretive frameworks run the risk of blinding one to other sources of useful and adaptive information because of the very nature of interpretation. This downside can be avoided if users realize the fallibility of diagnosing. Any blindness arising from inflexible adherence to an interpretive framework is a fault of the user, not a fault of the system. Such a state of affairs would be indicative of cognitive rigidity.

However, other similar charges are more specific to the DSM. Given the categorical nature of the DSM and its grounding in Neo-Kraepelinian assumptions, diagnoses are assigned on an all-or none basis. Either an individual is diagnostic or an individual is not diagnostic. Thus, the DSM has difficulties addressing sub-clinical cases. Not only does this mean that sub-clinical phenomena will in all likelihood receive less research attention than “full” diagnoses, if for no other reason than the fact that external

funding agencies will not allocate funds for these “non-disorders”, but as a result of this prevention research may be hindered. This also means that individuals who are in a significant amount of distress but who are not fully “diagnostic” may not be able to receive third-party benefits (Westen et. al, 2002). For example, approximately 60% of those treated for enduring, maladaptive, and clinically significant patterns of personality cannot be diagnosed on Axis II of the DSM (Westen & Arkowitz-Westen, 1998). Numerous other studies demonstrate similar results with other groups of disorders, such as anxiety (e.g., Zinbarg, Barlow, Liebowitz, & Street, 1994; and Olfson et al., 1996). And while a certain percentage may be able to be assigned NOS diagnoses, a large group of individuals may be ignored or unrecognized if DSM criteria are stringently and dogmatically adhered to.

Related to its categorical framework, the DSM at least implicitly promises all-or-none decisions for treatment. It aims at prescriptiveness – i.e., if an individual has this discrete disorder, it should be combated with this particular palliative (Blashfield, 1984). If this is indeed the case, the development of novel treatment approaches may be hindered. However, given the limited state of the art, this possibility seems fairly unlikely.

A third charge is more presently applicable, though. This would be the fact that the multiaxial nature of the DSM obscures the relationship between Axis I disorders and personality. A lengthy and rich discussion of the interrelationships between clinical symptoms and character patterns can be traced back at least to Freud (and, indeed, to Hippocrates and Galen), and such conceptualizations are not limited exclusively to

psychoanalytic theorists and therapists. With the current separation, it is easy to see the two axes as mutually exclusive when, in fact, evidence would seem to favor a view of their strong interrelatedness. For instance, several studies have shown that the possibility of having an Axis-II diagnosis rises exponentially with the addition of each Axis-I diagnosis (as reviewed in Westen et. al, 2002). Thus, the DSM may serve to limit recognition of the two axes' intimate interconnectedness.

A related issue is the question of what avenues of thought influence the inclusion and exclusion of categories in the DSM. For instance, voting, and not scientific evidence, led to the inclusion of sleep disorders in DSM-III-R. In a review of the literature prior to 1987, sleeping difficulties were not considered to be mental disorders. Therefore, sleeping problems essentially became mental disorders overnight (Houts, 2002). As another example oft spoken of, homosexuality was eventually eliminated as a mental disorder. This exclusion was less based on data, but more on political pressures experienced at the time such as the disruptions of APA meetings by gay rights activists and the powerful rhetoric of Robert Spitzer (for a more complete discussion of this issue, the interested reader is referred to Blashfield, 1984, pp. 32-33). It must be stated explicitly, however, that this is in all likelihood not specific to the DSM. All classifications are, and will always and inevitably be, cultural artifacts that reflect to a greater or lesser degree the values and experiences of the popular *zeitgeist*. The political nature of the DSM may indeed be unavoidable, especially given the fact that so many individuals are involved in each successive edition's construction. Obviously, rhetoric

occasionally eclipses evidence and theory. This may sometimes evince positive consequences, and other times evince negative consequences.

Another set of interesting critiques of the DSM relate to its claim and intention of being atheoretical. This atheoretical claim, however, can be questioned on several fronts. Both philosophy and the history of intellectual ideas have repeatedly demonstrated that those who claim to be unencumbered by theory typically ascribe to unconscious, clandestine, and undesirable theory. Theories shape our perceptions of the world, and taxonomy is no exception. Also, a number of those responsible for the genesis of DSM-III were, as mentioned above, Neo-Kraepelinians, and Kraepelin's strong theoretical beliefs are palpably present through DSM-III onward (for the interested reader, a short synopsis of the characteristics and values of Neo-Kraepelinians can be found in Klerman, 1978). But, as far as the DSM and its creators are concerned, its taxonomy is based on descriptive psychopathology (bereft of etiological speculation) and a consensus strategy (Houts, 2002; Jablensky & Kendell, 2002). What was hoped for in using this an "atheoretical" approach was that clinicians of various orientations could agree on the overt manifestations of clinical disorders without taking recourse to questions of how these disorders originated. This has been criticized on both psychological and philosophical fronts. As stated by Millon in terms that would resonate with Thomas Kuhn, "the belief that one can take positions that are free from theoretical bias is naïve, if not nonsensical [as] it is theory that provides the glue that holds a classification together and gives it both its scientific and its clinical relevance (1991, parentheses mine)." In

concert with this, Doucette questions whether “review committees can simply dismiss their theoretical bias in articulating the criteria for diagnostic categories (2002, pp. 203)?”

In summary, the DSM when considered as a general whole possesses sizable strengths and weaknesses. However, what has yet to be explored is the question of its suitability for the mood and anxiety disorders in specific. This will be one of the principle tasks of the following section.

Part III

THE CLASSIFICATION OF MOOD AND ANXIETY

“The question is not whether it is possible to draw a line between anxiety and depression, but whether this distinction is useful in clinical practice.”

---Kendall, 1975

Anxiety Disorders and Non-Psychotic Mood

Disorders: A History of Overlap

Since the publication of DSM-III (1980), explicit recognition of the overlap between anxious and depressive symptomatology has dwindled. However, a lengthy historical tradition views anxiety and depression as intertwined. Although a definitive text tracing the interconnections between these two phenomena has yet to be written, examples from the available sources will be provided in order to lay an historical foundation for the remainder of this work.

At certain times the ancient world recognized the close interconnection between what more recent scholars would term anxiety and depression. Hippocrates observed two fundamental patterns of their interrelation. First, he recognized their co-occurrence or, what would today be termed “intraepisodic comorbidity”. Second, he recognized the pattern that one disorder, when followed over time, gives way to the other. As stated in his *Epidemics III*, (cited in Akiskal, 1990), “patients with fear... of long standing are

subject to melancholia (pp. 597).” The latter anticipates Alloy et. al’s (1990) “Helplessness-Hopelessness” theory of anxiety and depression described almost 1,500 years later. Similarly, Galen’s humoral theory of melancholia (*viz.*, excess of black bile) appears to have been wider and more encompassing than later conceptions, as it possessed neurasthenic (described later in this section) and anxious symptoms as well as depressive elements. This particular melancholia also anticipates a relatively sophisticated aspect of anxiety that Kierkegaard (1800/1844) would later term (in his characteristic way) its “sympathetic antipathy” and “antipathetic sympathy” or, more clearly, a desire for what one dreads, and a dread for what one ultimately desires.

Additional theorizing on the interconnections between anxiety and depression can be found 1,000 years after Hippocrates. For instance, Jonathan Wyer described the intimate relation between melancholia and fears/anxieties in terms similar to Galen (as cited in Foucault, 1987). Further, Thomas Willis described melancholia as “a madness without fever or frenzy, accompanied by fear and sadness (Ibid, pp. 121).” The anxiety that accompanied melancholia led them to be “sad and punctilious (Ibid, pp. 121).” Whytt, along with presenting a tripartite classification of what would later be termed the neuroses, prescribed identical treatments for these constellations of symptoms and aimed for a global “purification” of the patient (Ibid.). Thus, even before Cullen’s 1780 coining of the term “neurosis,” the idea that these constellations of symptoms were similar and, in various ways, overlapped with one another, was not a novel one.

Apart from his neologism, Cullen contributed further to the concept of “neurosis.” He delineated four different types and wrote lengthy clinical descriptions of neurotic

phenomena ranging from what we would now term “panic attacks” to depression or melancholia. Further, he posited that all of the “neurotic illnesses” shared a common etiology. This shared etiology consisted of what he termed a “physiological breakdown” (Alexander & Selesnick, 1966, pp. 111). This breakdown was believed to occur in the nervous system itself and, as noted above, represents one pole of etiological speculation during the Enlightenment.

Given their importance for this essay, it may be useful to briefly discuss the terms “neurosis” and “neurasthenia” and place them within the context of anxious and depressive symptoms. This is an admittedly difficult task, as a sizable amount of the information required to fully understand these diagnostic entities in terms of their nature and their application appears to have been tacit. Thus, in the primary and secondary literatures that the writer reviewed, these terms were not found in a fully articulated and clearly elaborated state. This is not altogether surprising, however, given the many complexities of meaning and experience as well as the general difficulty of the subject matter. This lack of formulation may in fact provide indirect evidence that a broader conception of such symptomatology is preferable to a narrow one.

At a fundamental level, it is unclear whether or not “neurosis” was thought to be a single diagnostic entity or whether it was perceived as a superordinate category. Examples of both interpretations exist in the available literature. It may be that there was a difference of interpretation between a professional’s esoteric knowledge and the public’s exoteric knowledge, with the latter conceiving neurosis as a unitary entity. Or, as another likely possibility, it could be that different groups of professionals utilized the

term in idiosyncratic ways. Prior to the adoption of a “centrally unifying” (at least in principle) text/system like DSM or ICD and prior to the availability of rapid communication over vast distances, it stands to reason that professionals treating mental disorders (and working in relative isolation from other groups of professionals) developed and elaborated upon operational definitions of “neurosis” that may have been very different from what other contemporaneous professionals developed. It is even possible that hospitals working in close geographical proximity developed very different working definitions of “neurosis.” Additional historical analysis is required to clarify this question.

Regardless, the concept of neurosis typically consists of at least four elements. First, neurotics express various symptoms such as anxiety, hypochondriasis, depression, and the various phobias. As is apparent, analysis into the contemporary diagnostic constructs of the anxiety, somatoform, and mood disorders was not present. Second, neurotics experience “neurotic maladjustment” or impairment in their ability to cope with day-to-day problems (Tyrer, 1985). Thus, a level of clinically significant interference is usually present, but this level of impairment does not typically extend to severe maladjustment. Third, the concept of neurosis typically carries etiological implications (Glatzel, 1990). Namely, it was typically thought to be associated with a lack of moral fiber and degenerative attributes or an underlying disturbance of personality/character (as was later made explicit by Franz Alexander, 1930). Implicit in both of these etiologies is an implication that neurosis can be overcome through an exertion of will. Fourth, the concept of neurosis implies no disturbance in gross reality testing (Freud, 1924). This, in

particular, is what distinguishes between neuroses and psychoses. This gross diagnostic distinction, in one form or another, remains present in current systems.

Closely related to neurosis is the concept of neurasthenia. G. M. Beard introduced the concept to the United States in 1869, but it had previously been discussed in 1860 by Bouchut (Hinsie & Campbell, 1970). Until more modern times, neurasthenia was considered to literally be a weakness or exhaustion of the nervous system. It, like neurosis, was thought to be related to degeneracy, and Beard termed it “a disease of American civilization” and the urban exigencies of American life (Glatzel, 1990). Neurasthenia was, and is, characterized by the following: sadness, worry, mental exhaustion, paroxysmal (*viz.*, sudden and intense) anxiety, various unexplained somatic complaints, and what was termed by Janet the “loss of the function of the real” or a general difficulty relating to the real and the present (Foucault, 1987/1954, see also Borkovec & Sharpless, 2004 for a discussion of the temporal dimensions of anxiety). This disorder, though seemingly comprised of diverse symptomatic content, was viewed as a unitary disease entity by 19th century alienists in the U.S. (Gosling, 1987). And whereas Freud eventually separated anxiety neurosis from neurasthenia, he believed that they were both still “actual neuroses”, that their respective symptoms often overlapped, and that they shared a common etiology (Hinsie & Campbell, 1970). Also of importance, neurasthenia was conceptualized as a starting point and vulnerability factor for the subsequent development of the more severe neurotic disorders and constituted “the soil from which they grow (*Tuke’s Dictionary of Psychological Medicine*, 1892, as cited in Hinsie and Campbell, 1970).” Although not currently a DSM or ICD diagnosis, some of

its symptomatic content continued into the DSM's in the form of what was termed "asthenic personality." The DSM-II and ICD-8 descriptions of this disorder (as cited in Andrews et. al, 1978) share remarkable overlap with descriptions of this 19th century nosological entity. In particular, those with asthenic personalities typically demonstrate inflexibility in their existence, an atrophied will, and a lack of enthusiasm. And while it was eventually abandoned as an "in vogue" diagnosis in the West, neurasthenia remains a frequent diagnosis in China, other Eastern countries, and Egypt (Good, 1993). It will be seen in subsequent sections that these "broader" conceptions of neurotic and neurasthenic psychopathology fit well with current data and certain recent conceptions of the mood and anxiety disorders.

Returning to the latter half of the 19th century, other famous physicians and scholars recognized fundamental symptomatic overlaps as described above. Charcot is generally regarded as the first to make a modern systematic study of neurosis or, as he termed it, hysteria (however, it should be noted that Charcot's notion of hysteria was much broader than Freud and Breuer's notions of disorder – or even Janet's – and that it included anxious and depressive elements). Janet followed suit, and his particular contribution was both to group the neuroses according to dynamics and differences in the complexity of psychological functions that the various subgroups could perform and also to recognize the role of forgotten (dissociated) traumatic events in neuroses (Hinsie & Cambell, 1970, Wolpe, 1972). At the risk of gross oversimplification of a subtle theory, Freud saw difficulties in sex, aggression, and meeting the omnipresent exigencies of civilization to be the root cause of neuroses. And, despite his subsequent diagnostic

distinctions, he never ceased recognizing the overlap between anxious and depressive symptoms (e.g., 1962/1895b, 1966/1917).

In the 20th century, such thinking has continued to be recognized. Kraepelin's conception of classification has been discussed previously, but two comments bear particular importance for the present topic. First, as would be expected, Kraepelin split neurosis into separate and distinct categories. However, the very first case study he cites in his *Lectures on Clinical Psychiatry* (1968/1904) contains reference to "apprehensive depression" and, moreover, he provides other instantiations of symptomatic overlap (pp. 6-7). In spite of this recognition, however, Kraepelin can be viewed as one extreme of a debate that, as will be seen, continues on to the present. So, whereas Kraepelin represents a view of anxiety and depression's discreteness, Hans Eysenck posits that they belong to different parts of a similar affective continuum (Alloy et. al, 1990). And, while Eysenck would agree with Kraepelin's emphasis on biology, at least as far as the predisposing factors are involved, he saw neurosis more as a learned behavioral misadaptation (Eysenck, 1967, Glatzel, 1990). Prior to Eysenck, however, Eliot & Patrick Slater proposed that neurosis was a single nosological entity and that the different neurotic groups fade into one another and do not form qualitatively distinct groups (as cited in Tyrer, 1985). Thus, a fluctuating quality was believed to be present in which the manifest symptomatic presentation may differ markedly when cross-sectionally analyzed. This idea of neurotic fluctuation was echoed in Karl Jaspers' *General Psychopathology* (1963) as well.

Moving forward to the birth of the first two editions of the DSM, overlap is also recognized. “Neurosis” was used as a superordinate category in both editions. Etiological factors (i.e., personality factors) were clearly emphasized as well. The first two editions also explicitly recognized the ability of anxiety and agitation to “mask” depressive symptoms. However, all three of these fundamental recognitions of the overlap between anxiety and depression were either eliminated outright, or drastically curtailed with the publication of DSM-III.

The Current Predominant Conception of the Mood and Anxiety Disorders

The publication of DSM-III (1980) was a crucial shift for the contemporary conceptualization of the mood and anxiety disorders. As stated above, it marked the ascendancy of a Neo-Kraepelinian understanding of these disorders. It also complicated (simplified?) the diagnostic process by eliminating the fairly unitary concept of neurosis through splitting it into different classes that contain independent disorders. With DSM-III-R, and its removal of hierarchical exclusion rules, multiple anxious and depressive disorders could now also be diagnosed within a single individual, and the problem (and solution) of comorbidity came to the foray.

As discussed previously, the DSM’s “atheoretical” system eschews etiological speculation in favor of descriptive symptomatology. And, although it is hierarchical in nature (and, thus, is segmented into the mood and anxiety disorder “classes”), no theory is provided to explain either intra-class coherence or the hierarchy itself. In fact, the section on anxiety disorder merely begins with, “The following disorders are contained

within this section...” and commences to list the 12 available permutations of “anxiety disorder” (DSM-IV, 1994, pp. 393). It follows suit with the 10 available mood disorders (Ibid., pp. 317). Thus, no justification for either the disorders or the hierarchy is forthcoming in the text.

Strengths of the DSM

However, there are a number of strengths in the DSM’s classification of mood and anxiety. Firstly, the developers of the system met one of their primary goals, as the reliability of these diagnoses is acceptable. As reviewed by Brown & Barlow (2002, pp. 303), the reliabilities of DSM-IV principal anxiety disorders ranges from .67 (generalized anxiety disorder) to .86 (social phobia). Within this notion of reliability lies another strength. Namely, the diagnostic criteria of the DSM have been standardized for administration in a structured interview format. The two most widely used assessment tools, the Structured Clinical Interview for DSM-IV (SCID) and the more specialized Anxiety Disorders Interview Schedule (ADIS), are both relatively easy to administer and provide reliable diagnoses. They are able to categorically capture a wide range of mood and anxiety disorder phenomena ranging from concentration difficulties to suicidal ideation.

Of prime importance, however, was the splitting of neurosis into the various mood and anxiety disorder categories. Although this premise will be challenged, it allowed for the specific isolation of various symptom clusters. This analysis led to important results. For example, by isolating the symptoms of panic disorder without

agoraphobia from related symptom constellations, a great deal of basic knowledge on the nature of panic was acquired. This basic knowledge, in turn, led to the formulation of extremely effective treatment techniques (e.g., Clark's "cognitive model of panic," 1986, and Craske & Barlow's "panic control treatment", 2000) for this specific symptom constellation. It appears likely that such gains would not have been as forthcoming had the diagnostic entity of panic disorder without agoraphobia not been isolated and pinpointed in this manner.

Weaknesses of the DSM

However, in spite of these strengths, the DSM does possess sizable weaknesses for the conceptualization of mood and anxiety. The primary difficulty (and focus of the remainder of this essay) is that the sizable splitting of the DSM obfuscates the commonalities and overlap between two sets of disorders. Copious evidence will be provided in subsequent sections to demonstrate that the DSM's splitting does not correspond to empirical (or clinical) findings.

Also noteworthy are several other less fundamental critiques. For example, the DSM emphasizes anxious and depressive states over anxious and depressive traits. Thus, the aforementioned idea (implicit in the concept of neurosis) that personality variables predispose individuals to manifest these traits has been lost (Westen et. al, 2002). In addition, the idea that depression has been relegated to categorical status as a "mood disorder" has been criticized by many. Previous to DSM-III, dysphoria was locatable in all of the classes of available diagnoses. With DSM-III and beyond, it became a strictly

Axis-I phenomenon. As Beutler & Malik (2002) state, “It is difficult to justify the conclusion that depression is an independent disorder... instead, it seems to serve as a signal or “marker” of disease, much like fever is a marker for infection (pp. 258).”

However, the most important weakness from a conceptual standpoint is the problem of comorbidity.

Comorbidity between and within the anxiety and mood disorders is rampant (for reviews, please see Costello, 1993 and Barlow, 2002). Multiple diagnoses are the rule rather than the exception (Hunt & Andrews, 1996). The DSM and its Neo-Kraepelinian framework is therefore left in the unenviable position of either positing the aggregation of multiple causative factors in every individual with comorbidity or must recognize that a general factor (or factors) has made the individual vulnerable to such states (Andrews, 1996). The DSM’s position will be analyzed and critiqued further in subsequent sections but, given the importance of comorbidity as a concept and as a problem, an exploration of the term’s history and usage is warranted.

Comorbidity

The actual term “comorbidity was introduced into the medical literature by Feinstein (1970). He defined it as “any additional co-existing ailment... in a patient with a particular index disease (pg. 467).” In his paper, he discussed the deleterious effects of such things as hepatic cirrhosis on rectal cancer and the ability of some diseases to “mask” the symptoms of others. Thus, the concept was not created with psychological disorders *in mente*. However, in spite of the fact that the term was created within the

context of traditional medicine, comorbidity was eventually adopted into the psychiatric and psychological literatures. This concept was used to account for the co-occurrences of diagnoses that were commonly found when hierarchical exclusion rules were reduced (or deliberately ignored) in DSM-III and DSM-III-R. Similarly, the creation and proliferation of structured clinical interviews that coincided with each successive DSM's publication had an impact on the awareness of comorbidity (Andrews, 1996). In line with this, research conducted by Wittchen (1995) demonstrated that that use of these structured interviews yielded three times as many diagnoses as clinicians, left to their own devices, would choose to assign. Before proceeding, however, what is “comorbidity” within the context of psychopathology?

Although Feinstein introduced the term comorbidity into the scientific nomenclature, a number of theorists and researchers have questioned his particular conceptualization of the phenomenon, its utility for psychological explanation, and its exportability from medical illnesses to psychological phenomena. Relevant portions of this ongoing and multifaceted debate will be discussed.

Of obvious importance for the study of comorbidity is a clear and precise definition that is readily agreeable to among theorists. Unfortunately, no real consensus in the field has yet developed, a factor that has caused a great deal of confusion (van Praag, 1996), misunderstanding (Vella et. al., 2000), and sometimes even blind reification (Lilienfeld et al., 1994). This is likely due to the number of philosophical issues that manifest when one attempts to categorize psychological phenomena. In a thorough explication of comorbidity, fundamental ontological and epistemological

questions arise such as: what constitutes a clearly delineated “psychological disorder”; where should the line of demarcation between “normal” and “pathological” be placed; how much of the natural and medical sciences’ classificatory systems is applicable to psychological inquiry; and what constitutes sufficient evidence for inclusion of new disorders into the nosological scheme? Whereas this preliminary list is by no means exhaustive, it provides a view into the complexity of the task and, ultimately, the necessity of relying on one’s values as a guide through such a maze of ultimate uncertainty and confusion.

In the psychological literature comorbidity is typically defined, in line with Feinstein, as the presence of an additional (psychological) disorder in a patient with a particular (psychological) index disorder (Brady, 1998). Although this definition appears to be straightforward and precise, it conceals a number of additional questions. For example, is this comorbidity inter-episode (i.e., lifetime) or intra-episode (Klerman, 1990)? What criteria are most appropriately used for the determination of the “index”, or primary, disorder? Is symptomatic predominance, temporal primacy, or hypothesized causal relation the best choice for this determination (Brown & Barlow, 1992; Klerman, 1990)? Furthermore, can comorbidity occur at the symptom level, or should use of the term be restricted to syndromes and disorders (as “morbid” refers to a state of disease)? Unfortunately, answers in the affirmative to all of these varying positions can be found in the literature. This current state of affairs has led some, like van Praag (1996) and Vella et al. (2000) to argue that the concept of comorbidity conceals more than it clarifies.

It would appear that the resolution of these various debates on comorbidity hinges on whether or not medical and psychiatric classification schemes are indeed commensurable. Many have called psychiatry's incorporation of medical concepts into question on a number of grounds (Lilienfeld, 1994; Andrews, 1996). For instance, knowledge of etiology (or the highest level of Scadding's ladder) is crucial for the determination of a disorder's discreteness. Whereas this is by no means easy to determine in medicine (Feinstein, 1970), it would appear to be even more difficult to apply this criterion to psychological phenomena. As Klein & Riso (1993) noted, no disorder in the DSM has been conclusively demonstrated to be discrete. *Sans* firm etiological evidence, categorization is typically determined according to phenomenology (which is towards the lower end of Scadding's ladder). Thus, though often conceived of as discrete entities, there is no logical reason (and little empirical evidence) for psychological disorders to be viewed as such.

However, as the current state of affairs requires the utilization of the predominant DSM system, the writer would argue that an attempt be made to increase the degree of precision with which the term is used. Comorbidity will therefore be defined in a specific sense (although authors to be cited may have used various permutations of the definitions above), and this definition was used in the writer's own comorbidity study (cited in subsequent sections). It will be defined as the co-occurrence of two or more DSM diagnostic disorders at a particular point in time. Thus, only intra-episode comorbidity will fall under this rubric. And, as the identification of the "principle" disorder holds

importance for research, it is believed that symptomatic predominance is the preferred criterion for most studies.

Additional critiques of the DSM's classification of mood and anxiety will become apparent in the discussion of recent empirical findings and the alternatives to the DSM system. The latter will be discussed first.

Existing Alternatives to the DSM: Theory and Evidence

Alternatives from the Nosologies of Other Cultures

A fundamental question to ask, especially with matters in intellectual vogue, is “why is the study of this important?” or, “why should limited intellectual and temporal resources be directed at this topic rather than others of importance?” In general, there are at least 4 main reasons why the study of other culture's conceptions of psychopathology holds importance. Two derive from intellectual concerns, and two from ethical concerns.

Regarding the former are issues surrounding basic research and the danger of what could be termed “paradigmatic blindness.” It is conceivable that a useful knowledge base of psychopathology could arise through collecting information about culture-specific manifestations of mental distress (and, possibly more fundamentally, determining that culture-specific manifestations of mental distress exist in ways that are alien to other cultures). If it were discovered, for example, that basic human predispositions towards folly or suffering manifest themselves in different ways according to the influence of overarching social “horizons,” it would allow for a better understanding of the pathways that lead from such predispositions and individual human

experiences to the subsequent development of disorder. Moreover, by identifying such symptomatic variability, novel treatment approaches could result.

Second, as stated previously, theories and paradigms allow one to see, but can also obscure that which is not within one's dominant purview. Thus, it is very likely that our prevailing conceptions of psychopathology, as varied and seemingly splintered across departmental and orientational lines as they appear, are conceivably all taking part in a superordinate perspective that may not be shared by non-Caucasians and/or non-occidentals. In looking at the views of those who are outside such a *Weltanschuuang*, and in taking such views seriously, one may be confronted with novel approaches or even the possibility of "extra-paradigmatic" vision. This pluralistic approach has been convincingly argued for within the discipline of science as a whole (Feyerabend, 1975), but the arguments appear equally valid for the specific discipline of psychopathology. As Feyerabend states, "The consistency condition which demands that new hypotheses agree with accepted theories is unreasonable because it preserves the older theory, and not the better theory. Hypotheses contradicting well-confirmed theories give us evidence that cannot be obtained in any other way. Proliferation of theories is beneficial for science, while uniformity impairs its critical power (1975. pp. 24)." Coming from the viewpoint of this particular philosophy of science then, it is important to take seriously, or at least thoughtfully consider, viewpoints seemingly disparate from our own which may partake of fundamental presuppositions that we may not share. A dogmatic reliance on that which is already apparent and comfortable may actually be a hindrance to knowledge

rather than a demonstration of the commonly held (naïve) popular view that science is an ineluctable and systematic march towards the truth and away from error.

In reference to the ethical reasons to study other races and cultures, two seem to hold especial importance. One is the issue of representativeness. It remains an open question how well our current conceptions of psychopathology hold for non-westerners or non-Caucasians. Thus, identifying salient and clinically meaningful differences could conceivably assist in the provision of services to large sections of the population. Closely related to this is the issue of power. As a sizable majority of the developers of the DSM were European and American Caucasians, it is imperative that the majority take the charges of non-representativeness seriously and, at the very least, evaluate the veracity of such claims.

These statements also hold for the study of depression and anxiety. As both anxiety and depression appear to be indigenous aspects of the human condition (Sue, Sue, & Sue, 2000; Guarnaccia, 1997; Horwath & Weissman, 1997; Kirmayer, 1997; and Barlow, 2002), it is useful to review how other countries and cultures classify and conceptualize these phenomena. It is also important to look for their potential differences with dominant perspectives

A comparison of different cultural and ethnic groups within the confines of the DSM and ICD systems reveals interesting information. For some symptom constellations, the similarities are striking. For instance, lifetime panic disorder rates are remarkably consistent across cultures (Horwath & Weissman, 1997). Some disorders, however, seem to display subtle, yet important, differences in symptomatic emphasis.

For example, the inclusion of the “loss of interest/pleasure” question in the criteria for a major depressive episode was motivated by a desire to capture the experience of non-westerners (Hughes, 1985). As another example, African-Americans rarely endorse generalized anxiety disorder (Neil-Barnett & Smith, 1996) but demonstrate higher levels of isolated sleep paralysis than other groups (Bell et. al, 1988). A number of Hispanic individuals experience significant distress but do not meet diagnostic criteria for a listed DSM disorder (Salman et. al, 1997). However, they do meet criteria for “mixed anxiety-depression” (a disorder under investigation, but not presently included in DSM-IV). There is also the interesting example of the Japanese syndrome termed *taijin kyofusho*. Many researchers believe it to be a variant of social phobia (Hofmann & Barlow, 2002). Interestingly, though, instead of being fearful of performing actions that lead to the embarrassment of oneself, the focus of fear in those with *taijin kyofusho* is that they will offend or embarrass *others*. This is a subtle, yet crucial distinction in the disorder’s manifestation that could potentially lead to different treatment foci.

At a more general level, research findings imply that minority groups tend to express their psychological distress somatically, are more comfortable endorsing somatic symptoms, and are more inclined to accept “medicalized” explanations of their illness (Friedman, 1997; Friedman, 1994; Sartorius et. al, 1990). However, in the writer’s review of the literature, it remains unclear whether or not this is due to specific cultural influences or is explainable on the basis of economical and educational variables. More research on this question is clearly needed.

There also exist what appear to be “culturally-bound” syndromes that, while possessing some similarity, manifest in ways that appear alien to Western psychologists and psychiatrists. A vivid example is the Asian disorder *koro*. An individual suffering from *koro* experiences acute anxiety associated with a strong fear that their genitals are retracting and, when completely retracted, that death will ensue (Money & Annecillo, 1987). No diagnostic parallel exists in the West. Also of note is the *Dhat* syndrome of India (Viswanathan, Shah, & Ahad, 1997). An individual suffering from *Dhat* believes that the loss of semen in one’s urine leads to the depletion of both physical and mental energy. Presumably, this bears some similarity with neurasthenia, but manifests in a profoundly different way by focusing so clearly on the genitals and reproductive system and by developing within cultures in which beliefs about sexual potency abound.

Along with analyzing individual “culture-bound” syndromes and disorder variations, analysis of existing classification schemes is also useful.² A number of subtle diagnostic variations exist in European countries. Whereas it appears that most debate in Europe concerns schizophrenia and schizophreniform disorders instead of the “neuroses,” certain variations exist in countries that do not uniformly ascribe to ICD-10. The Finnish classification system, for example, only has one code available for major depression and lacks qualifiers (for multiple episodes, etc., Kuoppasalmi et. al, 1987). As another example, the Danish conception of neurosis, at least at the time of Bech’s (1990) publication, is based on “character” neurosis, and is not conceptualized as what we would term an “Axis I Major Clinical Syndromes.” In the Scandinavian countries as a whole, it has proved difficult to disentangle anxiety and depression (Ibid.).

A number of other systems for classifying and conceptualizing mental disorders exist (for example, those of the Third World as cited in Wig, 1990), but possess less relevance for the present essay. However, the nosologies of Egypt and China warrant some discussion. In the Egyptian Diagnostic Manual of Mental Disorders (DMP), and the Chinese Classification of Mental Disorders (CCMD), the categories of neurosis and neurasthenia are present. Both countries, for economic and practical reasons, emphasize clinical experience and clinical utility in the construction of their systems (Okasha, 1988; Yu-Cun & Changhui, 1988). Thus, both a North African and an Asian system openly espouse the view that neurosis and neurasthenia are useful diagnostic concepts and, at least implicitly, hold the view that anxious and depressive symptoms are intimately interrelated. In China, neurasthenia is an especially useful concept (Good, 1983), and it was retained in the diagnostic system after a pilot study was conducted in 1983 (as cited in Yu-Cun & Changhui, 1988). The current CCMD, in particular, admirably attempts to strike a balance between the predominant ICD system (in order to facilitate communication with non-Chinese professionals) and diagnostic concepts that experience has proven to be idiosyncratically useful.

While there appears to be a great deal of overlap in the manifestation of anxious and depressive symptomatology, enough variability exists between cultures to call into question the utilization of a strictly categorical classification system based exclusively on Western-derived nosological entities. This is because symptom clusters may occur which do not fit neatly into any existing category or syndrome. Such individuals may appear to be subclinical in severity (e.g., the presence of “mixed anxiety-depression” in Hispanics),

even if experiencing distress, and therefore receive no diagnosis. As an alternative to this state of affairs, some have advocated the inclusion of a broad disorder (such as the aforementioned mixed anxiety-depression) encompassing both anxious and depressive symptoms (Guarnaccia, 1997). This would likely improve the current diagnostic situation. However, the writer would advocate, along with Hsia & Barlow (2001) that attempting to categorize culturally bound syndromes and giving each its own “niche” is less than ideal. Looking for similarities across the various syndromes will lead to a greater understanding of psychopathological processes and cultural contributions. Through the remainder of this paper, it will be argued that a broad dimensional model will be beneficial not only for the classification and research of psychopathology, but also in studying other culture’s symptomatology.

Along with looking to other cultures, novel ideas can arise by looking to the ideas of individuals working within the Western psychological and psychiatric traditions. Some have broken markedly with DSM and ICD systems and have fortunately published their alternatives.

van Praag’s Diagnostic Basins

The psychiatrist Hermann van Praag has consistently been a trenchant critic of current nosologies. In particular, he has been no shrinking violet with regard to those aspects of classification that he feels are stagnant, intellectually bankrupt, examples of excessive preoccupation with the obvious, or gross oversimplifications (e.g., the predominant interpretations of comorbidity, as in van Praag, 1996, 1987 & 1992).

Although his writings typically critique the entirety of contemporary classifications, he discusses anxiety and mood disorders in particular. He also bolsters his arguments with original research. This has been particularly evident with depression, a diagnostic entity he feels is in danger of “dissipation” (van Praag, 1995, pp. 269). First, he sees the continuing increase of poorly validated depressive disorder variations as indicative of wishful thinking instead of empirical or phenomenological accuracy. He decries the desubjectivation of diagnosing, and reports data that 28% of clients diagnosed with “major depressive disorder” reject “depression” as accurately depicting their emotional state (van Praag, 1992 & 1995). He also criticizes the rampant avoidance of etiological components (or even speculation about etiological components) that is present in current classification systems. Van Praag echoes the views of others who believe that the relationship between anxious and depressive symptomatology is, in actuality, much closer than a reading of the DSM or ICD would seem to imply. He has remained steadfast on this matter since his early research on 5-HT (van Praag, 1987). As he states, “the overlap between mood, anxiety, and personality disorder psychopathology is so overwhelming that discussion of any depression study should include the issue of whether the observed phenomena relate to depression, to co-occurring anxiety, to personality disorders, or to components of these conditions. Generally, this question is avoided. Avoidance behavior, however, does not promote progress (van Praag, 1998, pp. 771).” Not only does avoidance behavior not promote progress, as van Praag accurately states, but such a standpoint is hypocritical for researchers who in all likelihood continue to pay homage to Mowrer and his explanation of the “neurotic paradox” (1948; 1952).

Throughout van Praag's various writings, one can implicitly and explicitly cull positive methodological recommendations for the classification of mood and anxiety. Of prime importance is what he would term "dissection." Van Praag believes that the elementary units in psychopathology are not syndromal categories, but are psychological dysfunctions (1988, 1995). Thus, our current syndromes should be dissected into their elementary components. Moreover, these elementary components should be phenomenologically precise without overemphasizing elements that are the easiest to assess (van Praag, 1995). Dimensions would be an appropriate classification method, as they do not assume sharp categorical disjunctures and in all likelihood mirror the realities of clinical practice (van Praag, 1990). He feels that dissection along these lines could be the next constitutive step that leads to an increased level of diagnostic sophistication (van Praag, 1988).

Van Praag also advocates the creation of what he terms "diagnostic basins" (1996). In order to form a diagnostic basin, disorders that are currently conceptualized as discrete (such as the mood, anxiety, and somatoform disorders), but which appear to display considerable overlap, would be collapsed into one broad category and studied collectively. If this were combined with the recommendations made in the previous paragraph, what might be gained is an ability to assess whether particular psychopathological variables are associated with individual "disorders" or the diagnostic basin as a whole. Basin linkage would support the "reaction set" model advocated by Meyer, at least in a limited form.

As will be seen, a number of these suggestions have been incorporated into the present work. In addition, they also appear to converge with theoretical and empirical developments. Admittedly, such an approach is a significant departure from the current system, but useful knowledge may result. As van Praag states, “it is hard to deny that in several respects we have been marching ahead in a blind alley. If we fail to acknowledge that, progress will stagnate. If we do retrace our steps... and dare to leave behind old and worn out concepts, there is every reason to believe that... research will bloom (1995, pp. 275).”

Psychoanalytic Structural Theory

Traditionally, psychoanalysts diagnose clients along global dimensions. This trend began with Freud and continues to the present. According to McWilliams (1994), “psychoanalysis has never seriously questioned three of its main convictions: (1) current psychopathological preoccupations reflect infantile precursors; (2) interactions in our earliest years set up the template for how we later assimilate experience... according to categories that were salient in childhood; and (3) identifying a person’s developmental level is a critical part of understanding him or her (pp.40-41).” Developmental level is typically determined according to a client’s defensive style, level of reality testing, and object relations (for additional detail, see McWilliams, 1999 and 1994).

Structural level is broken into three main categories: (1) neurotic; (2) borderline; and (3) psychotic. As is apparent from the prior historical review, the term “neurosis” has been present for quite some time and has been with psychoanalysis since its genesis.

For many theorists, it characterized a wide range of symptomatology and excluded psychotic phenomena such as hallucinatory processes and difficulties with consensual reality. Since the conception of borderline phenomena was developed in the 20th century, however, this term “neurotic” has narrowed. It now refers to individuals with intact reality testing and whole object relations who utilize relatively mature defenses. Thus, neurotic individuals are those who are either “healthy” or possess symptomatology that causes significant emotional suffering but does not seriously impair the individual’s ability to function at fairly high levels (McWilliams, 1994). Those operating at a borderline level have intact abilities to test reality, use relatively primitive defenses, and have a sense of self and others that is poorly integrated. Rapid fluctuations in identity are often present as well. Finally, at the psychotic level, reality testing is impaired, primitive (often “preverbal”) defenses are utilized, and differentiations between self and others are either poor, or there may be a delusional identity present (Kernberg, 1984).

If one were to translate the DSM into structural terms, individuals with a principle diagnosis of a non-psychotic mood or anxiety disorder (*sans* Axis-II pathology) would most likely qualify as neurotically organized. Clearly, this system of classifying clients is not the result of sizable splitting (although diagnosis according to character type may involve splitting tendencies, though of a different sort). It recognizes the overlap between the psychopathology in question and is not as concerned with the overt “surface” manifestations of anxiety or depression. What is important for such psychoanalysts are the underlying personality dynamics that manifest in the individual’s characteristic ways of relating to himself, the world, and other people.

An important strength of this theory is that it provides clear treatment implications for the psychoanalyst. For instance, if a psychoanalyst were treating a neurotically organized client who was not in a crisis situation, techniques such as clarification, confrontation, and interpretation would all be used. A supportive framework in which the therapist engaged in large amounts of self-disclosure would, *a priori*, not be utilized or deemed beneficial. Therapy would be very different if a psychotically organized individual was treated.

Operationalized Psychodynamic Diagnostics

In general, psychoanalysis has often been critiqued for its lack of involvement in mainstream clinical science. This has certainly been shifting, however, and a number of psychoanalytically influenced manualized treatment protocols are now available for conducting therapy trials and basic research (e.g., Gillies, 2001, Crits-Christoph & Barber, 1991, and Kernberg et. al, 1989). In spite of this, however, psychoanalysts and psychoanalytically-oriented therapists continue to express extreme dissatisfaction with the DSM classification on a number of grounds, not the least of which being its lack of validity for treatment planning (OPD Task Force, 2001). Thus, a recent and very significant occurrence in the psychoanalytic community has been the formulation of a self-contained diagnostic tool based almost exclusively on psychoanalytic concepts and theories. This new system is termed “Operationalized Psychodynamic Diagnostics.” As is clear from the title, its content has been operationally defined for all of its 5 axes.

The five axes are as follows: (I) Experience of Illness and Prerequisites for Treatment, (II) Interpersonal Relations, (III) Conflicts, (IV) Structure, and (V) Mental and Psychosomatic Disorders. Axis-I measures (on a 0-4 scale) such factors as the extent of disability, presence of secondary benefit, social support, and the client's perception of treatment. Axis-II formalizes the client's characteristic ways of being with others. This is accomplished through collecting data on both the client's subjective experiences and the subjective experiences of the assessor. Axis-III measures the presence of characteristic conflicts such as dependence vs. autonomy and submission vs. control. Axis-IV essentially consists of assigning developmental level through a "structural" interview, and the final Axis documents what DSM-IV diagnostic criteria that the client would meet (for additional information, see OPD Task Force, 2001 and Schneider et. al, 2002).

This diagnostic system possesses a number of strengths. Namely, its operationalized nature allows for precision in describing complex psychodynamic constructs. The OPD system also allows for measuring inter-rater reliability. Such studies have taken place, and reliabilities of 0.62, 0.62, and 0.71 were found for axes II, III, and IV, respectively (Axis I has yet to be tested). In addition, the first 4 axes provide information that is crucial for psychoanalytic treatment planning and case conceptualization. Once a client is situated within the context of the 5 axes, a number of useful treatment deductions result.

While this is a very impressive system that promises a widespread clinical applicability, there are several difficulties. For one, a global acceptance of this system is

highly unlikely at this time due to its overt reliance on psychoanalytic presuppositions. These would in all likelihood be unpalatable to significant portions of the psychological and psychiatric communities. Second, to achieve the reliabilities described above, 60 hours of intensive training was required. Such a time commitment and requirement for OPD training, and the time commitment needed to understand psychoanalytic theory, would likely limit the wide-scale acceptance of this system.

Finally, a weakness of the OPD system is specific to the study of mood and anxiety. As it is intended as a clinically useful and global diagnostic system focused on characteristic defenses, personality, object relations, and conflict, it provides a rich picture of an individual in broad and sweeping terms. At the level of symptomatology, however, it lacks the specific level of analysis required for many purposes. Basic research on individual symptom manifestations, or even clusters of symptom manifestations, would require the inclusion of additional informational resources. A fine-grained analysis of mood and anxiety symptoms and their vicissitudes would be impossible if one were to remain within the confines of OPD. But, as such goals were not a focus of the Task Force, this is not a surprising consequence.

Part IV

TOWARDS A DIFFERENT CLASSIFICATION OF MOOD AND ANXIETY

Several seemingly independent lines of evidence converge and point towards a different classification of the mood and anxiety disorders. A crucial feature of this conception that appears to unify a number of disparate and otherwise anomalous findings is “negative affect” (NA).

Introduction to Negative Affect (NA)

Negative affect can be defined in a number of ways. Closely tied to the concept of neuroticism espoused by Eysenck and others (Watson & Tellegen, 1985; Andrews et al., 1990), NA has been described as a stable and general factor of subjective distress that subsumes a wide range of negative mood states. It is a broad and pervasive predisposition to experience negative emotions that has powerful influences on cognition, sense of security, and world-view (Watson & Clark, 1984; Watson et al., 1988). A sample of adjectival descriptors for an individual with high NA could include tense, nervous, angry, guilty, upset, sad, scornful, self-dissatisfied, worried, and disgusted (Watson & Clark, 1984, 1991b). High NA individuals also tend to react strongly to a multitude of stimuli and experience difficulty getting “back to an even keel” when they become emotionally activated (pg. 9, Eysenck & Eysenck, 1975). In contrast, those with low NA generally display a predilection towards responding slowly, weakly, or mildly to

emotional stimuli and lack the characteristic fluctuations of those with high NA (Jorm, 1989; Eysenck & Eysenck, 1975). Descriptors for low NA individuals could include calm, relaxed, content, secure, and self-satisfied (Watson & Clark, 1984. Clark & Watson, 1991b). Whereas NA may appear to be reactive in nature, it was found to be present even in situations that lacked overt stress. (Watson & Clark, 1984).

Negative Affect can be contrasted with positive affect (PA). The latter is a construct also of sizable importance for latter sections. Although their terminology may imply that they are bipolar opposites, NA and PA are typically considered to be independent and orthogonal. Thus, low NA is not necessarily synonymous with a lack of joy or enthusiasm, (Watson & Clark, 1984). This last point, however, is somewhat controversial and has its dissenters. However, the available evidence would appear to favor the supporters of conceptual independence (for an excellent review of this issue, the interested reader is referred to Tellegen, Watson, & Clark, 1999). In general, PA reflects ones enthusiasm, determination, and the extent to which one experiences a zest for life (Clark et al., 1988; Watson & Clark, 1984). Descriptors for high PA would be active, delighted, proud, enthusiastic, and alert, whereas low PA descriptors would include tired and sluggish (Watson & Clark, 1984; Clark & Watson, 1991a). As will be shown, the concept of NA is the more fundamental of the two constructs, as it appears to be the foundation upon which the mood and anxiety disorders are built. As such, a thorough review of the available evidence documenting NA's existence and its relation to psychopathology is warranted. By way of introduction, three lines of relatively independent research were found to fuse otherwise anomalous findings and provide a

great deal of empirical support for NA and its central role in mood and anxiety disorder symptomatology.

Measurement Evidence

A major impetus towards research that led to the current conceptualization of NA was the unexpected, yet ubiquitous finding that self-report measures of anxiety and depression highly correlate with one another. In many instances, measures of anxiety and depression were found to correlate with each other as strongly, or even higher, than they do among themselves. Replications of this finding using a variety of normal and diagnostic populations are well documented (see Watson et al., 1988 and Watson & Clark, 1984 for reviews). This was found even when using relatively well-known and frequently used measures. For instance, the Beck Depression Inventory correlates more highly with the State-Trait Anxiety Inventory and the Taylor Manifest Anxiety Scale than it does with other measures of depression (Gottlib & Kane, 1989). Some have interpreted these findings as clear demonstrations of the limits of self-report measures. However, a different explanation for this seeming anomaly is that the various anxious and depressive disorders share a fundamental feature.

In their seminal study, Watson & Clark (1984) analyzed the inter-correlations of various tests purported to measure such domains as anxiety, depression, ego strength, neuroticism, and defensiveness. Despite the diversity of nomenclature and the presence of seemingly disparate content, the overall correlations were very high (ranging from 0.44 – 0.88), and many appeared to load onto a single, undifferentiated factor (Ibid.;

Watson et al., 1988). Of the numerous tests analyzed, 12 were identified as relatively “pure” measures of NA. The top 4 measures, in descending order, were the Taylor Manifest Anxiety Scale, Anxiety Scale, Psychasthenia Scale, and Social Desirability Scale. This was an important finding, as the empirical establishment of a novel construct necessitates the availability of reliable and pure means with which to measure it. Through these initial investigations, additional knowledge into the nature of NA was gained.

Evidence accrued from self-report measures to suggest that NA is endogenous and subjective in nature. In this particular context, the term “endogenous” is being used both to indicate that NA has a strong genetic component and also to differentiate it from reactive, situation-based, and temporary phenomena. In reviewing a number of studies in which both “pure” measures of NA and state anxiety measures were both used, Watson & Clark (1984) found that high NA individuals reported more distress across a wide variety of situations, both stressful (e.g., shock, threat of failure) and non-stressful, than those with low NA. Of the 92 available tests of this hypothesis, it was supported 91 times. These findings provide evidence that high NA individuals are not equivalent to individuals with low NA in the absence of stressful stimulation, thus contraindicating a reactive view of NA.

Regarding NA’s subjective nature, NA was conceptualized as relating more directly to mood and self-concept than to overt behaviors. It is marked by interiority, and may not possess outwardly visible manifestations. Thus, it was hypothesized that peer ratings would not correlate as strongly with self-report ratings as is typically found with

more overt, behaviorally-based constructs (Watson & Clark, 1984). It was also hypothesized that the reliability between self and rater reports would converge as subject-rater familiarity increases. Both of these hypotheses were empirically supported (Ibid.).

The stability of self-reported NA is also well documented and impressive. Early stability ratings of 0.64 over 13 years (Ibid.), 0.70 over 13 years (Clark & Watson, 1991), and 0.40 over 30 years (Watson & Clark, 1984) have been reported. More recent measures of rank-order stability are even more impressive. Clark & Watson (1999) report NA stability coefficients of 0.65 over 25 years with higher estimates (generally in the 0.65 to 0.80 range) over intervals of 6 to 12 years.

In summary, due to the fact that NA appears to be a stable and endogenous trait common to both the anxious and depressive disorders (at least in self-report measures), it may provide a link between them or may actually comprise a shared vulnerability factor common to both sets of disorders. In reviewing much of the available data concerning NA, Watson & Clark (1984) arrived at a conclusion similar to that reached in the earlier work of Eysenck. Specifically, it may be that biological factors probably play an important role in producing such stable differences in temperament. However, additional methods of empirical research are needed in order to lend credence to such a claim.

Community and Twin Study Evidence

Data establishing the relationship between NA and the mood and anxiety disorders, both at the symptom and diagnostic levels of analysis, have been found in community and twin studies. At the symptom level, Duncan-Jones (1987) concluded,

after examining the variations in anxious and depressive symptoms in two general population cohorts, that long-term vulnerability factors were vastly more important in the etiology of these disturbances than short-term stressors. In fact, these long-term factors were found to account for 70-75% of the variance associated with the mood and anxiety symptomatology. Of more importance to the present task, Duncan-Jones also concluded that NA, as measured by the Eysenck Neuroticism Scale (one of Clark & Watson's, 1984, top twelve NA measures), accounted for the majority of this effect (Duncan-Jones, 1987; Andrews, 1996). Supporting data have also arisen from research utilizing the Australian Twin Registry (ATR).

The study of twins, both monozygotic and dizygotic is an effective means of ascertaining genetic contributions to psychological functions. While in most cases this cannot definitively ensure genetic primacy (due to such factors as shared environment, similar cultural milieu, chromosomal inhibition, etc.), it is an alternative research tool that has yielded a great amount of data. Jardine et al. (1984) were one set of researchers who took advantage of the ATR's pool of subjects. In their study, they obtained pairwise data from 3,810 pairs of twins (both monozygotic and dizygotic) on anxiety symptoms, depressive symptoms, and the trait of neuroticism (NA, as measured by the Eysenck scale) and then conducted a multivariate genetic analysis. They came to the conclusion that "additive gene effects are more important causes of covariation than environmental factors (pg. 84)." Similarly, they also concluded that the same genetic factors that affect variations in the neuroticism trait also affected the symptoms of anxiety and depression. Others have since reanalyzed and expanded upon the original Jardine et al. (1984) data

and have arrived at similar conclusions. Utilizing uni- and multi-variate techniques Kendler et al. (1986; 1987) concluded that while the symptoms of anxiety and depression loaded on separate factors, genes seemed to influence the overall levels of both sets of symptoms in a largely nonspecific way. Though the affects of the NA traits were not controlled, Kendler et al. did comment that the distinction between symptoms and personality traits may be less clear than is usually thought. Martin et al. (1988) have also analyzed the original Jardine et al. data. They considered the relation between the personality trait of NA and two questions in the data set that focused on panic symptomatology. It was determined that the effects of a general genetic factor shared with the trait of NA were more important than the specific genetic determinants of the two individual panic symptoms. Although these findings are important in linking the trait of NA to the same genes that influence anxious and depressive symptoms, there is no logical reason to conclude that the symptom-based findings are capable of being exported to actually clinical diagnoses. With this in mind, the work of Gavin Andrews will be discussed.

To address the question of whether or not NA holds as much importance for fully diagnostic mood and anxiety disorders as it appears to hold for anxious and depressive symptomatology, Andrews (1990b) studied a quasi-population sample of 446 adult twin pairs from the ATR. The twin pairs were interviewed using the Composite International Diagnostic Interview (CIDI) and were also given the same self report measures as the Jardine et al. (1984) study. When the common measures between the Andrews and Jardine et al. studies were compared, strikingly similar results emerged. In particular,

Andrews' sample showed the same pattern of substantially higher monozygotic correlations for NA and self-report symptom measures than the corresponding dizygotic group evidence. This finding further demonstrates the importance of genetic determinants of NA and symptoms (Andrews, 1990b) and lends support to the sample's representativeness. Moreover, the lifetime prevalence of the assessed mood and anxiety disorders (*viz.*, the "neurotic disorders" of depression, dysthymia, generalized anxiety disorder, panic disorder with and without agoraphobia, social phobia, obsessive-compulsive disorder, and specific phobia) fell within the range of the United States Epidemiological Catchment Area study results (Andrews, 1996), indicating the representativeness of this sample, *vis a vis* diagnostic classification frequencies.

Of the 446 twin pairs studied, 243 individuals met diagnostic criteria at some point in their lives for at least one of the six neurotic disorders. Preliminary analyses revealed that those who met diagnostic criteria evidenced significantly higher levels of NA than those lacking any lifetime diagnoses. Comorbidity was found to be substantial, and the mean number of diagnoses was 1.6 per person. The concordance rate within twin pairs was also assessed. Mathematically, if there were no association between diagnoses, one would expect that 7.4% of the twin pairs would be concordant for any neurotic disorder over the course of their lifetimes (Andrews, 1990b). In fact, 9.9% (44 / 446, 95% confidence interval: 7.4 – 12.9%) of the twin pairs were concordant for any illness. This difference reached significance and indicates that some degree of clustering between (and within) the mood and anxiety disorders exists. Surprisingly, the monozygotic pairs and dizygotic pairs were not significantly different in concordance rates. In the

monozygotic group, 10.2% (19 / 186, 95% confidence interval: 6.4 – 15.1%) were concordant and 9.6% (25 / 260, 95% confidence interval: 6.4 – 13.6%) of the dizygotic group was concordant. This would seem to imply that genetic factors have little influence on the manifestations of the precise anxious or depressive disorders that the twins evidence. Tetrachoric correlations for the risk of comorbidity were then calculated (Andrews, 1996), and a single general factor accounting for 57% of the variance emerged. Next, the diagnostic individuals were ranked by the number of neurotic diagnoses they had experienced, which Andrews took to be a proxy of the general factor; he found a strong association ($P < 0.001$) with the NA measures used (Andrews, 1990b; 1996). This finding lends support to the view that comorbidity may be an index for severity (at least within these disorders), as higher NA levels may result in additional diagnostic criteria being met.

In summary, apart from a strong genetic influence on the general factor of NA, genetics appear to have little role in which particular mood or anxiety disorder is manifested. Related to these twin studies, a common finding in the literature is that approximately half of the variance in NA is attributable to heredity (as in the twin study of Tellegen et. al., 1988). However, it is now believed that this apparent balance represents an overestimation of unshared environments. Clark & Watson (1999) reviewed evidence which suggests that 70% of NA variance is attributable to genetic factors, with only 30% attributable to unshared environments.

Along with community and twin studies, information on NA and its relation to the neurotic disorders can also be gained from studies of clinical populations. Such studies

are obviously important, as disorders seen in the general population may not have the same relationships to etiological factors as do the (generally) more severe disorders seen in clinical settings.

Clinical Evidence

For the purpose of further delineating the results of his twin study, Andrews (1990c) conducted a study utilizing 165 subjects referred to his Anxiety Disorders Clinic for treatment of panic disorder with and without agoraphobia. Those for whom panic disorder with or without agoraphobia was their sole diagnosis were in the minority (26%), and 30% reported the experience of 4 or more mood and/or anxiety disorders over the course of their lifetime. The mean number of diagnoses was 2.9 per patient. As in the twin samples, no particular pattern of disorders was apparent over and above the greater than chance clustering of the “neurotic disorders” (a finding also reported in Sanderson et al., 1990; Maser & Cloninger, 1990a; and Mineka, Watson, & Clark, 1998). When Andrews repeated the analyses using these participants in place of his twin sample, similar results emerged. The first general factor explained the covariation of the other disorders and, as in the twin sample, the residuals did not significantly differ from zero. As before there was a significant association between number of diagnoses and level of NA. In fact, Andrews reported a 0.7 SD unit increase in NA with each additional diagnosis. Similarly, the sample as a whole scored significantly higher (1.55 SD) on NA measures than the general population mean for normals. These findings were also replicated in a later study (Andrews, 1996) and were further supported by a literature

review conducted by Watson & Clark (1984) in which they presented robust evidence taken from numerous studies which demonstrated that individuals suffering from mood and anxiety disorders scored significantly higher on measures of NA than either normals or psychotics. Evidence from another domain of research may help to illuminate the relationship between NA and these disorders. There are many individuals described in the literature who present for treatment of anxious and depressive symptoms and appear to be in substantial distress who do not meet full diagnostic criteria for either a mood or anxiety disorder (Barlow, 1998). In response to this, a DSM-IV field trial for the category of “mixed anxiety-depression” was conducted. It was found that those who presented with such a constellation of symptoms (and who would have formerly been relegated to a “not otherwise specified” category) were at least as common as those who met full diagnostic criteria for one of the mood or anxiety disorders. It was further determined that these individuals experienced significant distress according to clinician’s judgment and Global Assessment of Functioning ratings (Zinbarg et al., 1994). After factor-analyzing symptom presentations on revised Hamilton Anxiety and Depression Scales (and discerning 4 factors: NA, depression, anxiety, and physiological arousal), Zinbarg et al. compared the mixed anxious-depressive group and full diagnostic groups. Those clients in the mixed anxiety-depressive group were able to be significantly differentiated from the full diagnostic group. However, it was concluded that the fundamental component of mixed anxiety-depression was NA. So, whereas mixed anxious-depressive symptoms are, by definition, associated with anxiety and depression, it appeared to be a non-specific association. Data of this type have led to the speculation

that mixed anxiety-depression (like the above-mentioned neurasthenia) may represent a common cognitive and affective state which acts as a priming mechanism for development of the specific features that characterize the common anxiety and mood disorders (Barlow, 1998).

Along with mixed anxiety-depression, generalized anxiety disorder has been conceived of as a disorder that serves as a vulnerability factor for the subsequent development of other anxiety or depressive disorders. Generalized anxiety disorder has been found, like mixed anxiety-depression, to load heavily on the general NA factor as well (Mineka, Watson, & Clark, 1998). Brown et al. (1998) found that the association between generalized anxiety disorder and NA was 0.74. It has also been repeatedly shown to be the most common comorbid condition of other anxiety and mood disorders and, when it is the principle diagnosis, to have a 90.4% lifetime comorbidity rate (as reviewed in Roth & Fonagy, 1996). Findings such as these led Brown, Barlow, & Liebowitz (1994) to the conclusion that GAD is the “fundamental” anxiety disorder. Further, it has been postulated that generalized anxiety disorder and major depressive disorder are genetically indistinguishable, at least in women. Using the technique of bivariate twin analysis with 1033 pairs of female same-sex twins, Kendler et al. (1992a) found that genetic factors were important for both major depression and generalized anxiety disorder and that these genetic factors were completely shared between the two disorders.

When taken together, the data arising from these divergent sources offer the promise of illuminating a number of seemingly anomalous findings. As one example,

within the child literature the reliability and validity of only two categories of psychopathology, *viz.* the internalizing and externalizing disorders, have achieved general consensus. Some support for these broad band categories comes from the degree of overlap between the emotional disorders of anxiety and depression, which make up a large proportion of the internalizing category (Brady & Kendall, 1992; Brady, 1998). Given the role of NA in these disorders and its function as a vulnerability factor to both anxiety and depression, this finding appears more comprehensible than an alternative view that would posit the mood and anxiety disorders' discreteness. This lack of discreteness has been recognized by a number of psychopathology theorists, and the role of NA in their various models will be discussed next.

By way of summary, these three lines of evidence not only instantiate the importance of NA, but they also cast doubt on the notion of the mood and anxiety disorders' ontological discreteness. Several additional lines of evidence directly question the mood and anxiety disorders' discreteness, and are potentially explainable within the confines of the theory of NA.

The Lack of Transmission Specificity of Mood and Anxiety Disorders

Data compiled from seemingly diverse areas of research indicate that the genetic transmission of mood and anxiety disorders is not disorder specific. One of these sources is Andrews' twin study (1990b). In assessing lifetime neurotic disorders in monozygotic and dizygotic twins, Andrews found little evidence of transmission specificity. In fact, of the 243 twins who met criteria for any mood or anxiety disorder, only 1 pair was

concordant for major depression, 4 pairs for dysthymia, 2 pairs for panic disorder with or without agoraphobia, 1 pair for social phobia, and 17 pairs were concordant for generalized anxiety disorder. No pairs were found concordant for obsessive-compulsive disorder. Concordance rates between monozygotic and dizygotic twins did not significantly differ. However, as mentioned previously, twin groups did significantly differ with respect to NA. If different patterns of lifetime comorbidity or vulnerability were associated with different diagnoses, then these should have been evident in the twin samples, but this was not the case. Andrews also reported on the diagnoses of the relatives of this twin sample (1990c). No evidence for the inheritance of specific neurotic disorders over and above the inheritance of a general liability (NA) could be found, though a tendency for transmission of the general class of neurotic disorders was again evident.

Data from the Zurich Cohort Study (Angst et al., 1990) parallel Andrews' findings. In this longitudinal study, 591 subjects were diagnostically interviewed at three separate time periods. Along with tracking diagnostic information over time, data from the proband's parents were also obtained for the purpose of assessing transmission specificity. As the first assessments were conducted in 1979 (a year before DSM-III's publication), diagnoses were grouped into depression and anxiety categories. Upon analysis, it was concluded that there did not appear to be specificity of transmission between the subjects and their parents. Neither the rates of "pure" anxiety nor those of "pure" depression were found to be significantly greater among the parents of subjects with either of these diagnoses. Similarly, rates of anxiety alone or depression alone were

not significantly higher when compared to parents of subjects without either disorder. However, parents of subjects with both anxiety and depression and those with depression only, when compared to those subjects with neither disorder, have a five times greater risk of being diagnostic for both anxiety and depression. Similarly, there is a three-fold risk for parents of subjects with anxiety alone to be diagnostic for both disorders as compared to subjects with neither disorder. These data may lend some additional credence to the conceptualization of comorbidity as a severity dimension (Andrews, 1990c, 1996; Brady, 1998). As NA does appear to be genetically transmittable, at least in part, greater NA levels may predispose one to react adversely to stressful trigger mechanisms (as Andrews, 1996 postulates). Thus, if only mild to moderate levels of NA are transmitted, a stronger stressor may be needed to trigger psychopathology, with an even stronger stressor necessary to trigger symptomatology consistent with multiple diagnoses. This could explain the discrepancies between individual anxiety and depression and the combination (*viz.*, higher NA) of both.

Additional evidence for the lack of specific neurotic disorder transmission can be found in children at risk studies. In seven studies reviewed by Weissman (1990), a total of 350 children of depressed parents, 150 children of anxious parents, and 400 controls were assessed. Six of the studies looked at children of parents with major depression. Diagnoses in children were converted to rates (per 100). The rates of those children diagnosed with major depression ranged from 15-41 ($M = 26.9$). The rates of children diagnosed with any anxiety disorder ranged from 16-44 ($M = 26.9$). This was not a significant difference.

Children of anxious parents were also assessed. However, of the two studies included, they differed on the particular parental anxiety disorder. In Sylvester et al. (1988, as cited in Weissman, 1990), children of parents with panic disorder were assessed. Of these, 48 (per 100) met criteria for major depression and 42 met criteria for any anxiety disorder. This difference was not significant. Similar results were reported in Breslau et al. (1987, as cited in Weissman, 1990). Children of parents with generalized anxiety disorder were assessed. Eleven (per 100) met criteria for major depression, 11 met criteria for generalized anxiety disorder, and 9 met criteria for separation anxiety disorder. Although these data reported by Weissman closely mirror those of Angst and Andrews, they should be interpreted with caution. As Torgersen (1990) reported in a response to Weissman, there were no follow-ups conducted in these studies to ascertain whether anxiety and depression in childhood were the same disorders in adulthood.

However, these accumulated data converge on two main points. First, there appears to be a lack of transmission specificity for developing particular anxious and depressive disorders. Data from all three groups of studies are unanimous on this point. Second a tendency towards transmission of the general class of neurotic disorders appears to be common. Both of these points are consistent with the conception of NA and its proposed relation to the mood and anxiety disorders. Descriptive data on comorbidity will be reviewed next.

Comorbidity

Since the publication of DSM-III-R (1987), evidence documenting the high rates of diagnostic comorbidity has accumulated (Maser & Cloninger, 1990a; Klein & Riso, 1993; Lilienfeld et al., 1994; Vella, 2000). This has been found in DSM disorders in general and between the anxiety and mood disorders in specific. As an example of the former, Boyd et al. (1984) determined that the presence of any DSM-III disorder increased the odds of that individual having almost any other disorder. They also found that the number of individuals with two DSM syndromes was 116.5 times greater than would be expected by chance. Therefore, it appears likely that at least some degree of covariation between the various types of psychological dysfunctions exists.

Of more importance for the present work, however, is the fact that the mood and anxiety disorders have been repeatedly found to cluster together within individuals. In individuals diagnosed with a primary anxiety disorder, between 30 and 80% also meet criteria for at least one additional mood or anxiety disorder (for extensive lists of citations, the interested reader is referred to Roth & Fonagy, 1996; Borkovec, Abel, & Newman, 1995; Brown, 1996; Brown & Barlow, 1992; Sanderson et al., 1990; Maser & Cloninger, 1990). This rather wide range in the rates of comorbidity may be due to differences in sample composition. While not studying the mood and anxiety disorders specifically, Wittchen et al. (1991) found that clinical samples evidenced greater rates of comorbidity than community samples. In a meta-analysis conducted by Clark (1989), it was found that 57% of those diagnosed with major depression also met diagnostic criteria for an anxiety disorder. In the same study, it was found that 67% of those with panic

disorder with agoraphobia also met criteria for major depression. A common finding, reported by Brown (1996) is that panic disorder with agoraphobia and generalized anxiety disorder demonstrate the highest rates of comorbidity across the anxiety disorders. Taken together, these various reports cast doubt on the idea that the mood and anxiety disorders are randomly distributed in the population. In fact, it would appear more likely that there exists a factor (or factors) that is common to both sets of disorders and, indeed, is at least partially responsible for their clustering together at greater than chance levels. As has been argued above, NA is believed to be this common factor.

The Effect of the Presence of Comorbid Conditions on Therapy Outcome

Whereas effective psychosocial treatments have been developed for a number of DSM disorders, relatively few studies have looked at the potential impact of comorbidity on treatment outcome. According to Brown, Antony, & Barlow (1995), this can be attributed to such factors as (a) the infancy of treatments for many DSM disorders, (b) failure to assess comorbid diagnoses, (c) use of comorbid diagnoses as study exclusion criteria, and (d) use of small sample sizes that prevent the examination of the impact of specific patterns of comorbidity. Along with the relative paucity of studies, inconsistent findings further obfuscate clear and distinct knowledge of the impact of comorbidity. For instance, whereas early studies of major depression and the anxiety disorders reported poorer outcome with comorbid personality disorders (Reiche & Vasile, 1993), more recent studies (using mainly cognitive interventions), though indicating higher pre-treatment severity in the comorbid groups, demonstrated little or no negative treatment

outcome (Dreesen et al., 1997; Hardy et al., 1995; Can Velzen et al., 1997). In fact, Steketee (1990) found that clients with dependent personality demonstrated a better initial response to treatment than those without the comorbid personality disorder.

Regarding the impact of Axis I comorbidity on the treatment of other Axis I disorders, similarly equivocal results have been found that parallel those described above. In reviewing the available literature from the 1980's onward (mainly consisting of psychopharmacological treatments), Scheibe & Albus (1996) found that comorbid major depression was generally related to a more unfavorable outcome among other Axis I disorders or a more chronic course of illness. In their own psychopharmacological study, similar results were reported. Congruent findings were also reported by Angst (1996) and O'Rourke (1996). Brown et al. (1996), in a sample of primary care patients treated with either pharmacological or interpersonal interventions, reported that individuals with major depression and comorbid GAD responded to treatment, but took longer than patients with major depression alone. They also found significantly worse outcomes in patients with a combination of major depression and lifetime panic disorder. Findings akin to these prompted Shear & Maser (1994) to include the assessment of comorbidity in their proposed standardized assessment package for panic. However, other studies, mainly those utilizing solely psychosocial interventions, have not reported negative outcomes related to comorbid major depression and other Axis I disorders.

Leibrand et al. (1999), in a treatment outcome study of somatoform disorders, concluded that Axis I (including anxious and depressive disorders) and Axis II comorbidity had little, if any, impact on treatment outcome. However, methodological

flaws (such as the fact that the groups with higher comorbidity rates received significantly more therapist contact time) may have impacted on treatment outcome and imply that the results should be interpreted cautiously.

Brown, Antony, & Barlow's (1995) study of the cognitive behavioral treatment of panic disorder yielded similar results. In particular, those with comorbid conditions evidenced more pre-treatment panic and higher general symptom severity levels than those without comorbid conditions. Those with comorbidity, however, were not significantly less likely to reach the operationally defined measure of high endstate functioning than the non-comorbid group. And whereas sample size limitations precluded individual analyses of all comorbid disorders, those with social phobia were actually more likely to reach high endstate functioning at post-therapy (though the difference only approached significance at the 3-month follow-up) than those without social phobia. Major depression and dysthymia were somewhat associated with lower outcome, but this difference only approached significance at post and was not evident at the 3-month follow-up.

Brown, Antony, & Barlow then assessed the impact of post-treatment comorbidity on long-term outcome. Analyses indicated that the presence of comorbidity at 3-month follow-up was not associated with less favorable long-term (24-month follow-up) outcome than its absence. However, the continued presence of comorbid conditions at the 24-month follow-up was associated with a significantly worse outcome and a lower probability of achieving high endstate functioning.

Childhood anxiety disorder researchers have also begun to study the impact of comorbidity on treatment outcome. Kendall et al. (1997) reported that anxious children with comorbid anxiety disorders and anxious children with comorbid non-anxiety disorders did not evidence significantly different outcomes. In a post-hoc analysis using the Kendall et al. sample mentioned above and additional sample groups of anxious children from other outcome research, Brady (1998) conducted a study similar to Brown, Antony, & Barlow (1995). As in Brown, Antony, & Barlow, all subjects received cognitive-behavioral therapy (CBT). Presence of comorbidity at pre-therapy was associated with greater severity of internalizing symptomatology. However, as in the Brown, Antony, & Barlow study, this was not associated with poorer outcome as measured by remission of primary diagnosis. In fact, 70.6% of those with a comorbid diagnosis at pre-therapy were free of the primary diagnosis at the conclusion of treatment versus 68.4% of those without comorbidity. Also, the continued presence of comorbidity at post-treatment was associated with poorer outcome. This finding was significant with parental, but not child, reports of diagnosis. Moreover, comorbidity at pre-therapy was not associated with poorer maintenance of treatment gains from post-therapy to 12-month follow-up. Taken together, these findings and those below documenting the impact of treatment for a primary disorder on comorbid conditions lend support to the theory that NA is a common underlying factor of both the anxiety and mood disorders.

The Impact of Primary Disorder Treatment on Comorbid Conditions

The extent to which the treatment of a principle disorder reduces or eliminates the presence of non-targeted comorbid diagnoses has obvious relevance for nosology, treatment efficacy, and knowledge of general psychopathology (Brown & Barlow, 1992). In a review of the literature, only 4 studies to date have addressed this topic. One such study is the Brown, Antony, & Barlow (1995) panic disorder outcome study and another is the Sharpless et al. (2006) panic disorder with agoraphobia study. As these particular studies will be addressed more at length in subsequent sections, the other two (Brady, 1998 and Borkovec, Abel, & Newman, 1995) will be reported first.

Whereas 79% of Brady's (1998) childhood anxiety disorder sample met additional diagnostic criteria at pre-therapy, this reduced to 47% at post, a change that reached significance. There was a further (significant) reduction to 20% at follow-up. When the clients were dichotomized into treatment success and treatment failure groups, the success group was found to be significantly more likely to have comorbid disorder reductions than the failure group at both the post-therapy and follow-up assessment periods. Similarly, those children who experienced significant reductions in anxiety were significantly more likely to show total remission of comorbid conditions at post and follow-up. An unexpected reduction in externalizing disorders was also found.

In a post hoc study of generalized anxiety disorder, Borkovec, Abel, & Newman (1995) also assessed reductions of comorbid disorders associated with treatment gains. Although clients meeting criteria for panic disorder or major depression were excluded from the protocol, results similar to those mentioned above were reported. In this study,

78.2% of the sample (n = 55) met diagnostic criteria for an additional mood or anxiety disorder, and there were 70 such comorbid diagnoses. All clients were dichotomized into treatment success (no diagnosable generalized anxiety disorder of any severity) and failure groups. It was found that treatment of generalized anxiety disorder led to significant reductions in comorbidity. In the success group, only 3 of the 35 original diagnoses remained at 12-month follow-up. Surprisingly, significant reductions in comorbidity were also found in the failure group. Only 6 of the 19 diagnoses remained in 5 of the 10 failure clients. This led Borkovec, Abel, & Newman to the conclusion that even some improvement in generalized anxiety disorder processes can significantly affect less severe comorbid conditions. This hypothesis is consistent with finding that generalized anxiety disorder loads highly on NA (Brown et al., 1998; Barlow, 1998) and the view that NA underlies the various mood and anxiety disorders. Whereas dramatic declines from pre-therapy to 12-month follow-up occurred in both groups, the success group experienced significantly greater reductions than the failure group. In a more stringent test that excluded clients who received additional treatment after post, only 1 of 21 diagnoses in 1 of the 15 success clients remained, and 6 of the 17 diagnoses in 4 of the 7 failure clients remained.

Taken together, these two studies indicate that comorbid conditions are not immutable, and can at least be partially eliminated through psychosocial interventions intended to ameliorate a principle diagnosis. The following section will further demonstrate the variability of the neurotic disorders both with and without therapeutic interventions.

The Vicissitudes of the Mood and Anxiety Disorders

In a discussion of whether categories were appropriate for the classification of psychopathology, Jaspers (1923/1963) concluded that categories were suitable for known somatic illnesses and major psychoses, but not for neuroses and characterological disorders. This was because he viewed these disorders as continually fluctuating and shifting into one another. Similarly, Tyrer (1985) studied groups of neurotics over two years and concluded that there were significant temporal variabilities in their anxious and depressive symptoms. Over the past 20 years, additional preliminary evidence in support of this phenomenon has been collected from clinical and cohort studies.

In the Zurich Cohort Study (Angst, 1990) discussed previously, stability of diagnosis in participants with either “pure” anxiety or “pure” depression was tracked over time. Of those with a pure anxiety disorder in 1979, 13% had depression, 36% had both anxiety and depression, 10% had anxiety only, and 41% had no disorder at the 7-year follow-up. Regarding those with pure depression, 28% still had depression only, 19% had anxiety and depression, 14% had anxiety only, and 40% had no disorder at the 7-year follow-up. Fluctuations in diagnosis were also seen only two years after the first assessment. As no report of therapy-seeking behavior was provided, it is difficult to determine the importance of these findings. However, data from therapy outcome research lends corroborative evidence.

Certain mood and anxiety disorders have been known to have fluctuating symptom courses. For example, panic disorder shows marked variability in symptomatic and symptom-free periods over time. In 1995, Brown & Barlow reported on a treated

sample of panic disorder subjects. Whereas 68.3% were panic free at 3-month follow-up and 74.6% were panic free at 24-month follow-up, these percentages obfuscate the fact that 9 of those classified as panic free at 3-months were classified as panickers at 24-months and that 13 panickers at 3-months were panic free at 24-months. Whereas the latter finding is not surprising, the former is somewhat anomalous. This fluctuating quality of symptomatology has also been demonstrated between neurotic disorders.

In a study conducted by Brown, Antony & Barlow (1995), the effect of therapy on comorbid conditions was assessed. Whereas the Brady (1998) and Borkovec, Abel, & Newman (1995) studies demonstrated fairly steady declines in comorbid conditions, Brown, Antony, & Barlow's (1995) study revealed pronounced disorder fluctuations between the assessment periods. Due to sample size, only the mood disorders, social phobia, and the overall levels of comorbidity were analyzed. Whereas 9.4% of the panic disorder sample met diagnostic criteria for a mood disorder at pre-therapy, 5.7% were assigned this diagnosis at 3-month follow-up. At the 24-month follow-up, however, the mood disorder comorbidity rates returned to pre-treatment levels (9.4%). A diagnosis of social phobia was met by 11.3% at pre-therapy, reducing to 1.9% at the 3-month follow-up, and rising to 7.5% at the 24-month follow-up. Whereas this reduction in social phobia approached significance, comorbidity rates did not significantly differ between these three assessment periods.

When overall levels of comorbidity were analyzed, a fluctuating diagnostic course was also revealed. Whereas 39.6% of the clients were assigned at least one additional diagnosis at pre-therapy, this rate was lowered by 17.0% at the 3-month follow-up, a

change that was significant ($p < .01$). However, at the 24-month follow-up the overall rate of comorbidity returned to 30.2%, which did not significantly differ from pre-therapy levels. These cross-sectional analyses may obscure individual variation between clients, however, because comorbid conditions may not remain constant in individuals over time. To address this, Brown, Antony, & Barlow assessed the longitudinal course of the comorbid diagnoses. Of the 28 clients available at the 24-month follow-up who received a comorbid diagnosis at the pre-therapy assessment, 15 continued to meet diagnostic criteria. However, of these 15, 7 were assigned diagnoses that were different from any of the comorbid diagnoses that they received at pre-therapy. Of these 7, 2 met criteria for major depression, 2 for a simple phobia, and one each met criteria for generalized anxiety disorder, social phobia, and a sexual arousal disorder (as will be demonstrated below, these provocative findings have been replicated, and are consistent with a model that espouses underlying commonalities between the mood and anxiety disorders). Similarly, of the 36 clients who evidenced panic disorder only at pre-therapy, 6 were assigned comorbid diagnoses at the 24-month follow-up. These additional diagnoses were for major depression ($n = 2$), generalized anxiety disorder ($n = 2$), anxiety disorder not otherwise specified ($n = 1$), and obsessive-compulsive disorder ($n = 1$).

These longitudinal fluctuations appear to be consistent with a model that espouses underlying commonalities between the mood and anxiety disorders. It seems unlikely that such results would come to light if each of these disorders were, in actuality, etiologically distinct. These findings also cast doubt on the assertion that the structure of comorbidity is stable over time (Maser & Cloninger, 1990b). At least within this clearly

delineated symptomatic groupings (*viz.*, the mood and anxiety disorders), this does not appear to be the case. However, the discrepancy between the Brown, Antony, & Barlow (1995) study and the Borkovec, Abel, & Newman (1995) and the Brady (1998) studies merits some discussion.

A reason why the Borkovec, Abel, & Newman study achieved a more steady and pronounced reduction in comorbidity could be due to generalized anxiety disorder's stronger loading onto the factor of NA. As generalized anxiety disorder was the principle disorder and the focus of treatment, changes in generalized anxiety disorder processes could conceivably tap more directly into NA itself and thus evince a more fundamental change in a client's vulnerability to, and experience of, psychopathology. In regards to the Brady child study, the steady and pronounced reduction in comorbidity could be due to an age-specific effect. It may be that the general factor of NA plays an even more important role in childhood than in adulthood, with a correspondingly decreased emphasis on disorder-specific factors. These hypotheses, however, await further empirical support.

The Potential Modification of NA

Whereas evidence discussed above would indicate that NA has a strong genetic component, this by no means implies immutability. Evidence exists which indicates that NA can be modified through psychosocial interventions in general, and cognitive behavioral techniques in particular. In a 1989 meta-analysis, Jorm reviewed treatments that resulted in NA reductions. He discerned that the only treatments that resulted in

levels of change significantly different from those of placebo were cognitive therapies. However, as the studies analyzed used variations of Ellis's Rational Emotive Therapy and not Becks's Cognitive Therapy, it is unclear whether or not these two therapies resulted in equivalent reductions

In a post-hoc analysis of two outcome studies, Andrews (1996) reported changes in NA that followed cognitive-behavioral therapy (CBT). At a 1-year follow-up of agoraphobic clients treated with CBT, Andrews reported a 1-SD improvement in NA that became evident within the first few months that treatment ceased. In another analysis of 200 panic disorder, panic disorder with agoraphobia, and social phobia clients, Andrews (1996) reported another significant drop in symptomatology and NA following CBT treatment. Moreover, at the 24-month follow-up, the improvement continued to be stable, and a normalization of NA occurred which remained throughout the entire follow-up period.

An Empirical Test of NA in an Outcome Study

Sharpless et al. (2006) attempted to replicate a number of the findings described in the sections above. Specifically, they directly explored the relationships among NA, comorbidity, and therapy outcome in a *post-hoc* study of 74 participants meeting diagnostic criteria for panic disorder with agoraphobia. Participants were treated with cognitive therapy plus graduated exposure, progressive muscle relaxation plus graduated exposure, or graduated exposure alone (original outcome results are published in Michelson et al., 1996). NA was measured by the Taylor Manifest Anxiety Scale (*TMAS*,

Taylor, 1953), which was determined to be the best measure of NA when the original data were collected (Watson & Clark, 1984). Therapy outcome was measured using an operationally defined measure of endstate functioning (Michelson et al., 1996).

As in other samples, comorbidity was extensive, and 64.9% of the sample met criteria for more than one disorder. The total number of additional diagnoses was 90. Generalized anxiety disorder was the most frequent comorbid condition (followed by major depressive disorder and social phobia), and was found in 51% of the sample. As predicted, substantial declines in comorbidity occurred at the follow-up assessments, and the total number of additional diagnoses reduced to 19 at 3-month follow-up, 18 at 6-month follow-up, and 23 at 12-month follow-up. Thus, there appeared to be a trend towards comorbidity increasing at 12-month follow-up. Unfortunately, 24-month follow-up data were not available to determine if a return to pre-therapy comorbidity rates (similar to that found in Brown, Antony, & Barlow, 1995) occurred. Interestingly, of the 3 participants who met high endstate functioning at 12-month follow-up yet still manifested comorbidity, all had generalized anxiety disorder. This may indicate a return to “purer” or more rarified NA as opposed to more focused symptom manifestations. This is also in keeping with generalized anxiety disorder’s hypothesized role as the “basic anxiety disorder” (Brown et al., 1994).

As hypothesized, participants with comorbidity at pre-therapy manifested significantly higher levels of NA. In fact, the continued presence of comorbidity at later assessments was associated with higher NA levels as well. Taken in combination with the significant correlations between number of comorbid diagnoses and NA levels (which

is also a replication of Andrews, 1990b, 1990c), it was argued that the number of additional diagnoses that an individual demonstrates could be considered a proxy for NA level (*viz.*, comorbidity could be considered an index of NA severity). These findings are also consistent with claims made by Andrews (1990c) and Jardine et al. (1984) that symptoms of anxiety and depression are largely dependent on NA.

When looking at therapy outcome, short and long-term response to treatment was relatively unaffected by pre-therapy comorbidity. This is consistent with the findings of Brown, Antony, & Barlow (1995) and Brady (1998). The combination of these 3 studies findings raises questions about our current nosology, however, as it is somewhat counterintuitive that the presence of multiple symptom constellations do not negatively impact on global functioning. If these disorders were, in fact, discrete nosological entities, the comorbid disorders would remain more or less untouched, as they were not a focus of treatment.

Whereas pre-therapy comorbidity was not associated with a poorer outcome, the continued presence of comorbidity at the 6 and 12-month follow-up periods was. This was also found at the post-therapy assessment of Brady (1998) and the 24-month follow-up of Brown Antony, & Barlow (1995). These 3 sets of findings indirectly support the view that NA (as indicated by the presence of comorbidity) and endstate are negatively associated. This hypothesis was directly tested, however, and will be discussed later.

It was also hypothesized that participants who achieved high endstate functioning at follow-up would experience a concomittant remittance of comorbid diagnoses. This was only partially supported, and Chi-Squares only reached significance at the 6-month

follow-up. However, individual ANOVAs demonstrated that those who experienced a complete remittance of comorbid conditions significantly differed in continuous endstate scores from those who did not experience such remittances at 6- and 12-month follow-ups. Therefore, the remittance of additional diagnoses that were not a focus of treatment was associated with a better long-term therapy outcome. Again, it was argued that a plausible explanation for why such a phenomenon occurred is NA's shared role in the mood and anxiety disorders.

Participants meeting high endstate functioning criteria were expected to have lower NA at that assessment than those who did not. This was the case at post-therapy and the 3 follow-up periods. This direct evidence of a relationship between therapy outcome and NA, when taken in combination with the findings associating comorbidity with NA and associating the continued presence of comorbidity with poorer long-term outcome, supports the hypothesized importance NA holds for the mood and anxiety disorders and also their potential remediation. The hypothesis that NA would experience a normalization if high endstate functioning criteria were met was also supported. In fact, the NA means of high endstate participants were very similar to the norms provided in Taylor's original publication (Taylor, 1953). This suggests that NA, although displaying impressive and well-documented stability over time, is not immutable, and can be modified via psychological interventions.

Finally, diagnostic switches similar to those reported in Brown, Antony, & Barlow (1995) occurred. Over 10% of the sample of participants with comorbidity were diagnosed with at least one different disorder at one or more of the follow-ups. Similarly,

7 participants who only met panic disorder with agoraphobia diagnoses at pre-therapy acquired additional diagnoses at one or more of the follow-ups. Such results are interesting for a number of reasons. For instance, if not confounded by random error or demand characteristics, they may imply that comorbidity may not always evidence a stable longitudinal structure. These findings also raise further suspicions that the mood and anxiety disorders may not be discrete entities because, if this were the case, such results would be unlikely to occur.

After evaluating these findings, Sharpless et al. argue that the current DSM system stands in need of remediation in order to better capture the clinical and empirical reality of these phenomena. Specifically, they argue for diagnostic “lumping” within this finely delineated set of mood and anxiety disorders. The current work is an elaboration on that theme. Before proceeding to the particulars of this conceptualization, however, it is important to enumerate how others have utilized NA in various models of psychopathology. Given the importance of NA, various models of psychopathology have been developed which place NA in a pivotal role. These will be described below.

Negative Activation in Various Models of Psychopathology

Andrews, Tyrer, and the General Neurotic Syndrome.

Andrews (1990b; 1990c; 1991; 1996), while not denying the existence of additional etiological contributions to the neurotic disorders, places NA at the apex of his theory of psychopathology. In his conception, the anxiety and non-psychotic mood disorders comprise a “General Neurotic Syndrome” with NA serving as a core

component and chief vulnerability factor for the development of psychopathology. The experience of life adversity, combined with the preexisting vulnerability factor, acts to trigger symptoms (Andrews, 1996). The occurrence of comorbidity is viewed to be a result of this common factor's differing manifestations and the excessive splitting which is inherent in the DSM and ICD. Tyrer, on the other hand, wishes to maintain the basic categories in the DSM and ICD while making a significant addendum.

Peter Tyrer's diagnostic addendum is also termed "the general neurotic syndrome (GNS)," but differs in significant ways from Andrews. For Tyrer, it is a stand-alone disorder that can be added to the existing DSM categories. To meet diagnostic criteria for the GNS, an individual must: (1) meet diagnostic criteria for two or more mood and anxiety disorders (as classified by the DSM or ICD), (2) develop at least one of the diagnoses in the absence of major life stressors, (3) possess either anankastic or passive dependent personality features, and (4) possess a relative with a history of similar symptoms (Tyrer, 1985 and Tyrer et. al, 1992). Thus, the GNS does not eliminate DSM or ICD diagnoses. Instead, it relegates such diagnoses to use in individuals with "pure" instances of the disorders or, as Tyrer elegantly phrases it, "those who do not pass, chameleonlike, through different diagnostic hues depending on the nature of the stresses they encounter (Tyrer, 1989, pp. 687)."

Two-Factor, Three-Factor, and Hierarchical Models.

Other theorists, primarily those associated with Auke Tellegen, have posited two-factor models that retain the importance of NA in the etiology of disorders but include

Positive Affect (PA), a factor seemingly independent of NA that is important for the diagnoses of depression and social phobia (Watson & Tellegen, 1985, Brown et. al, 1998). In a number of factor analyses conducted with varying sets of descriptors and numerous data sets (reviewed in Watson & Tellegen, 1985), two factors comprising NA and PA have consistently emerged. This two-factor structure has also been identified in Japanese, Chinese, Swedish, Croatian, and Gujarati, indicating some degree of generalizability to other cultures. Moreover, relations of NA and PA to the “big five” model have been documented (Watson & Clark, 1992; Clark & Watson, 1999). The relation of the big five neuroticism factor and its facets to NA is strong ($r = 0.83$), and the relation between extraversion and PA is also strong ($r = 0.78$).

In the two-factor model, NA is believed to be common to both mood and anxiety disorders and, as evidence presented earlier indicates, is believed to play an important role in the genesis of both. PA, however, has only been reliably linked with depression. In fact, Clark et al. (1988) reported that the addition of PA increased the ability to predict major depression but not the anxiety disorders (later research conducted by Brown et al., 1998 has discerned some PA involvement with social phobia, but not as much as in major depression). In general, the anxiety disorders are characterized by high NA, but are theoretically unrelated to PA (with the exception of social phobia), and the depressive disorders are characterized by high NA and low PA (Watson & Clark, 1984; Watson et al. 1988; Clark & Watson, 1991a).

Since the initial formulation of the two-factor model, others have expanded the number of general and specific factors. In Clark & Watson’s (1991b) tripartite model,

autonomic arousal (also termed physiological hyperarousal) was proposed as an anxiety disorder-specific factor. Though initially believed to be the anxiety disorder equivalent of PA for depression, subsequent research has relegated autonomic arousal to panic disorder alone as a lower-order factor (Brown, Chorpita, & Barlow, 1998; Zinbarg & Barlow, 1996). This finding remains controversial (for a review, see Mineka et al., 1998).

Recent theorists (Mineka et al., 1998; Brown & Barlow, 2002, Barlow, 1998) have skillfully combined Clark and Watson's tripartite model (1991) with Zinbarg & Barlow's (1996) factor analytically derived hierarchical model of anxiety (a model that lends credence to the DSM-IV's delineation of disorders while acknowledging shared components). In this conceptualization, each individual disorder can be viewed as possessing both common and unique components. All of the mood and anxiety disorders share (and load significantly onto) the factor of NA. PA is another higher-order component specific to major depression and social phobia. Each remaining disorder also contains a unique element. A possible exception to this may be generalized anxiety disorder, which appears to load very highly onto the NA factor alone (Brown, Chorpita, & Barlow, 1998; Mineka et al., 1998).

In conclusion, whereas no consensus has been reached in the field regarding the appropriate model for the conceptualization of the anxiety and mood disorders, a component indigenous to and holding importance for them all would appear to be NA. As described above, NA was incorporated into every model and was held to be a higher order factor that subsumed the various neurotic disorders. Thus, boundaries between the

non-psychotic mood and anxiety disorders are becoming increasingly acknowledged as “more fuzzy” as empirical data accrues to support earlier (pre-DSM-III) conceptions of psychopathology.

Part V

FIRST ATTEMPT AT A SYNTHESIS: A DIMENSIONAL MODEL WITH DIAGNOSTIC PROFILES

“The empirical basis of objective science has nothing 'absolute' about it. Science does not rest on a solid bedrock. The bold structure of its theories rises, as it were, above a swamp. It is like a building erected on piles. The piles are driven down from above into the swamp, but not down into any natural or 'given' base; and if we stop driving the piles deeper, it is not because we have reached firm ground. We simply stop when we are satisfied that the piles are firm enough to carry the structure, at least for the time being.”

--Sir Karl Popper, 2002/1935, pp. 93-94

Introductory Statements

As is apparent from the preceding sections, the DSM-IV's conception of the mood and anxiety disorders stands in need of revision. The current system, based as it is on a Neo-Kraepelinian conception of discrete disorders, has no means available to account for such phenomena as: greater than chance comorbidity, large reductions of comorbid disorders when principal disorders are treated, fairly rapid diagnostic fluctuations, the lack of moderated outcome in individuals initially assessed with multiple disorders, the existence of a common factor (NA) underlying the various mood and

anxiety disorders, and strong associations between this common factor, the number of additional disorders, and therapy outcome. Thus, both theory and empirical data appear to favor an approach that places emphasis on the perceived commonalities between anxious and depressive symptoms as opposed to splitting symptoms into (presumably) separate categories. In addition, the DSM possesses other lacunae that are notable and less than ideal. For example, the DSM *does not*:

- (1) capture subclinical phenomena
- (2) provide a broad and useful description of anxious and depressive psychopathology
- (3) provide a useful measure of treatment outcome
- (4) adequately capture patterns of psychopathology outside of its categories, including culture-bound syndromes and non-traditional symptomatic presentations
- (5) provide prognostic indicators
- (6) provide a very rich clinical picture of clients for use in case conceptualization and treatment planning.

What is proposed³ is an alternative to the current DSM-IV classification/assessment that takes into account the above-mentioned phenomena and encapsulates both recent and pre-DSM-III empirical and clinical findings. In doing so, it is based *explicitly* on theoretical considerations and is also responsive to, and directly influenced by, the current empirical literature. In contrast to the DSM, this assessment/classification attempts to:

- (1) ground the system in a theoretical framework (*viz.*, negative affectivity)
- (2) move away from the categorical pitfalls and reifications in the way mood and anxiety are currently conceptualized (obviously, what is proposed is another category, but one that is “looser” than the alternatives and hopefully better supported by theory and research)
- (3) recognize and capture subclinical phenomena
- (4) provide a thorough assessment of the vicissitudes of negative affect symptomatology
- (5) provide a quantitative and dimensional measure of treatment outcome

- (6) capture fairly individualized patterns of psychopathology that do not necessarily correspond to DSM categories
- (7) assess prognostic indicators that the current literature deems as important,
- (8) provide a clinical picture of an individual's current symptomatology as indicated by their symptomatic "profile" and measure the client's readiness for treatment.

In summary, what is intended is that this system will be a replacement for the DSM-IV's Axis-I diagnostic categories for mood and anxiety. This diagnostic module will provide a thorough assessment of the "basin" of negative affect symptomatology and can be used either alone or in concert with the remainder of the DSM system. Within this framework, there is no presumption that the particular "topographical" manifestations will remain constant over time and, in fact, it is predicted that symptomatic variability will display itself more frequently as individual symptoms are isolated and studied over time.

The Inventory Of Negative Affect Symptomatology (*IONAS*)

Important Decision Points in the Generation of the *IONAS*

Before proceeding to a discussion of the dimensions themselves and, more importantly, the psychometric evaluation of a self-report measure derived from this system, it is appropriate to discuss the various decisions made with regard to the generation of the system itself. In particular, the decision to primarily utilize a dimensional system was made on several grounds. First, dimensions allow for a quantification of each individual symptom. This is important not only because it allows each symptom to be analyzed in isolation, but also because dimensions allow for the assessment of symptomatic change and outcome according to degree, and not higher-

order nosological categories based on presence versus absence of the symptom. Second, dimensions allow for the assessment of sub-clinical symptomatology that would otherwise be ignored in gross categorical systems. This ability to detect sub-clinical phenomena will increase confidence in making the determination of whether or not symptom clusters have truly remitted versus achieved sub-threshold status. As an example of this, it has been found that one of the two most important predictors of relapse in panic disorder with agoraphobia is the continued presence of residual agoraphobia (White & Barlow, 2002). Third, as will be described in greater detail below, the dimensions will result in a symptomatic “profile.” This will aid in the recognition of common (and uncommon) symptomatic distributions and will hopefully foster basic knowledge. Fourth, by analyzing the various anxiety and mood disorders into their constitutive components and grouping them, individual dimensions can be added or eliminated according to the dictates of predominant theory and research findings without accruing the difficulties such modifications cause when modifying “discrete” categorical disorders. The chosen dimensions are not “set in stone” and inflexible, but should be continually tested and refined in order to increasingly and appropriately capture this particular constellation of psychopathology. Indeed, such research may demonstrate that psychopathology itself, being also within the flow of history, changes over time and lacks an absolute basis or foundation. From the standpoint of the author (who would agree with the epigraph from Karl Popper that begins this section), this is an appropriate conceptualization. Finally, as has been reviewed above, dimensional systems make a good deal of sense for psychopathology, as the majority (if not all) of the symptoms of

anxiety and depression are indigenous to human experience. For example, depression, worry, and panic attacks are ubiquitous in the general population and do not appear to represent qualitative disjunctures of experience (e.g., Ruscio & Ruscio, 2002; Ruscio, Borkovec, & Ruscio, 2001).

Another decision point focused on the extent to which the symptomatology assessed in the DSM-IV would be retained. However, in order to avoid some of the weaknesses of the DSM system (e.g., inter-disorder overlap), it was decided that only non-redundant symptoms would be retained. To facilitate this, all of the DSM-IV's individual polythetic criteria for the mood and anxiety disorders were gathered, the many redundancies were eliminated, and the items were converted from dichotomous (*viz.*, present/absent) to numerical scoring (see Appendix A for the reduced set of symptoms). Incorporating the DSM symptoms into the *IONAS* was held to be very important, as they (1) allow for explicit comparisons with the DSM-IV system, (2) facilitate the utilization of already-existing research derived from within the context of that system, and (3) retain diagnostic elements that are already familiar to the field. In addition, the writer believes that these symptoms are not superfluous, but are important components of negative affectivity that are present in a number of patients.

However, the *IONAS* does not limit itself to the DSM symptoms. Other dimensions have been culled from various sources. As will be shown below, these added dimensions have been derived from (1) past and present empirical findings, (2) past and present clinical theory, (3) available cross-cultural psychopathology, (4) psychological speculations from historical antecedents of clinical psychology, and (5) NA theory itself.

All of these sources were included for the purpose of broadening the assessable “net” of NA because it is firmly believed that a great deal of symptomatic heterogeneity exists that is important, yet not captured by the current DSM.

A decision was made to group the many individual dimensions into higher order “dimensional sets.” This was thought to be important, as interpretation of 100 or more individual dimensions can be an overwhelming undertaking. The dimensional sets themselves (described in detail below) were developed on *a priori* grounds using individual dimensions that appeared to hang together in a readily identifiable way. Thus, they are not held to be instances of “nature being carved at its joints” or representations of essential essences, but are intended as useful heuristics to quickly reduce the dimensional data. These dimensional sets, like the individual dimensions themselves, should also change over time and be responsive to new theoretical and empirical developments.

The *IONAS-SR*

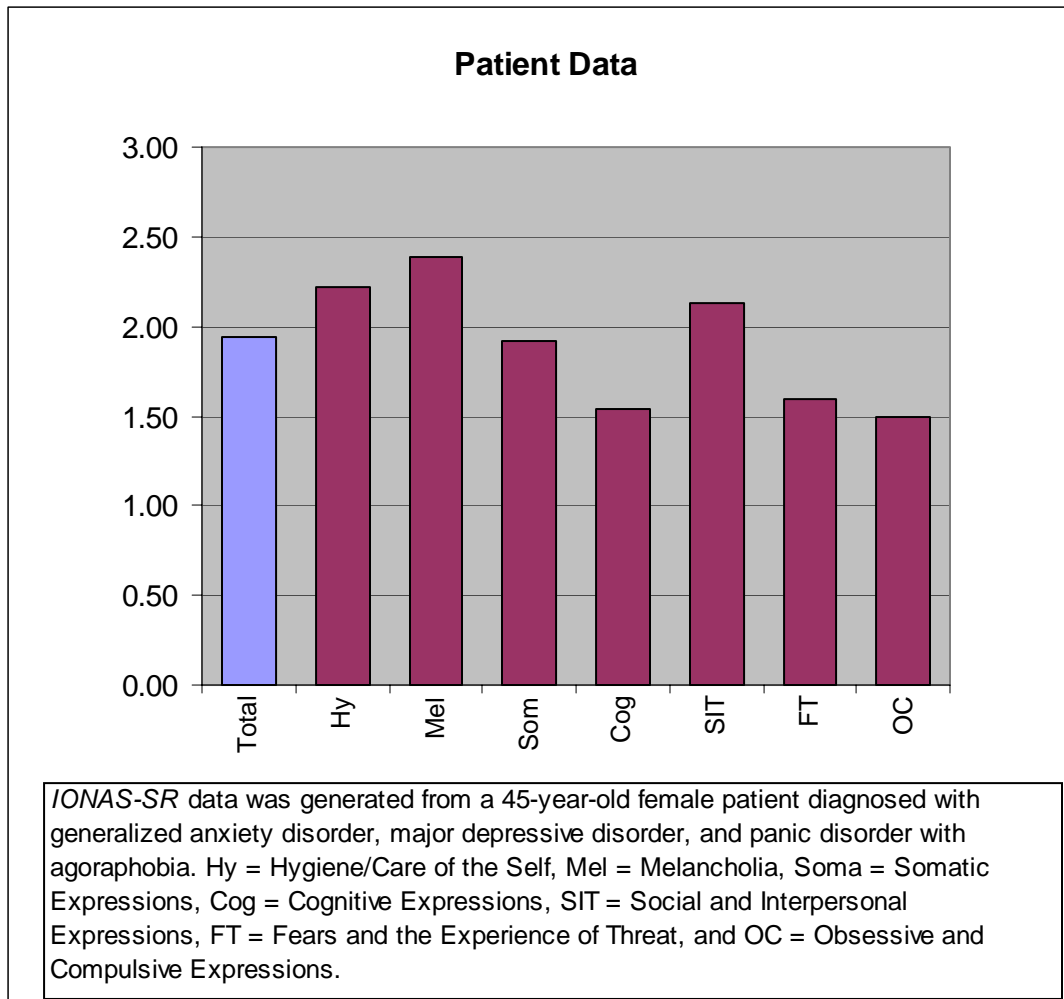
As every program of research must have a starting point, it was decided that the principles and theories discussed above and below would first be systematized and formalized within the context of a relatively brief self-report measure. This measure, the *Inventory of Negative Affect Symptomatology-Self-Report Version (IONAS-SR)* was developed for the express purpose of assessing and adequately capturing the various symptoms associated with both negative affectivity and what are currently conceptualized as the mood and anxiety disorders. Should it prove useful, the next phase of research will

focus on the construction of a much more detailed clinician-administered semi-structured diagnostic interview/treatment outcome package.

The *IONAS-SR* (found in Appendix B along with its scoring procedures) consists of 100 questions rated on 4-point scales. It differs from many of the existing measures in several respects. First, instead of focusing on the presence or absence of a symptom, it directs the individuals completing the measure to rate the impairment/level of distress that each individual symptom causes. Second, in contrast to other measures of negative affectivity (some of which are described below), it is intended to capture a broad topography of negative affectivity. Third, results can be viewed at 3 different levels of abstraction. The first level is derived from aggregating all of the questions for a total score. This yields an overall level of negative affectivity. Second, the 100 individual dimensions can be clustered into the 7 *a priori*, theoretically derived dimensional sets described below. This allows for a more specific, yet relatively broad assessment of symptom elevations. Third, each of the 100 symptoms can be looked at in isolation. When used in the second or third way, the *IONAS-SR* will also allow for a visual “profile” akin to those generated by the *Minnesota Multiphasic Personality Inventory* (please see Figure 1 for the visual representations of the first two levels). This profile, presented in graphical form, allows for the quick discernment of symptomatic elevations as well as patterns of symptoms. This availability of 3 different levels of assessment at varying levels of complexity allows for flexibility in usage and purpose. Thus, the *IONAS-SR* can be utilized as a relatively brief (requiring approximately 10-15 minutes)

Figure 1

Sample *IONAS-SR* Profile of Total and Dimensional Set Scores



screening/outcome instrument or as a more in-depth tool for analyzing and researching very specific manifestations of psychopathology.

The *IONAS-SR* can also be administered with different time periods in order to answer specific research questions. For the purposes of the present study, participants will be asked to answer all questions in relation to the past three months. Whereas the

IONAS-SR will not be directly compared with DSM-IV-derived data in this study, the 3 month time period was thought to be appropriate, as it in effect splits the difference between the shorter 1-month DSM criteria and the longer 6-month to 2-years criteria variously used in DSM-IV for mood and anxiety different. It was also believed that a 3-month durational instruction would focus the individual on fairly recent symptomatic manifestations while simultaneously allowing for some indication that symptoms may be persistent. The derivation of the various dimensions and dimensional sets will be discussed next.

Derivation of the Dimensions and Dimensional Sets of the *IONAS* and *IONAS-SR*.

The proposed system contains 8 higher order sets of dimensions. Most of these sets will be intuitively familiar, and those that are not will be explained further below. All of these dimensional sets will be assessed in the (not yet developed) interview version of the *IONAS*, but Dimensional Set VIII will not be assessed in the *IONAS-SR*. This is primarily due to the fact that a number of these items are not conducive to self-report rating (e.g., the presence of secondary gain from illness) and that they were primarily included for the purpose of making the *IONAS* a "stand-alone" assessment module that retains clinical relevance. Be that as it may, these 8 dimensional sets include:

- (1) *Hygiene / Care of the Self*
- (2) *Melancholia*
- (3) *Somatic Expressions of NA*
- (4) *Cognitive Expressions of NA*
- (5) *Social and Interpersonal Expressions of NA*
- (6) *Fears / The Experience of Threat*
- (7) *Obsessive and Compulsive Expressions of NA*
- (8) *Personal Factors / Readiness for Treatment*

The individual items that compose each dimensional set of the *IONAS-SR* can be found in Appendix C. As discussed above, a number of these items were derived from DSM-IV symptoms. Both be discussed in greater detail below.

Before proceeding, however, a brief note on the inclusiveness of the *IONAS* is warranted. As will be noticed upon a perusal of Appendix C, the majority of post-traumatic stress disorder (PTSD) symptomatology was omitted. This was deliberate, and is in keeping with the sizable NA literature generated by Zinbarg & Barlow (1996) and Andrews (1996). In all of the extant models of NA psychopathology, PTSD is not included, and a decision was made to omit PTSD, acute stress disorder (as well as the somatoform disorders) from the current formulation of the *IONAS* until further data accrues. It may be, as some have speculated (e.g., van der Kolk et. al, 1996), that PTSD fits better within a dissociative spectrum of disorders.

With regard to the more novel dimensions, the inclusion of prognostic indicators of psychopathology was given a high priority. This has been a notable omission in the DSM. Furthermore, whereas there are only a limited number of empirically supported prognostic indicators available, every effort has been made to incorporate them into the present system. As a number of these prognostic indicators are located within the *Personal Factors/Readiness for Treatment* dimensional set, they are not assessed in the *IONAS-SR* and will not be discussed in the present work. Those prognostic indicators located within the *IONAS-SR* will be listed below.

- A majority of studies (reviewed in Newman et. al, in press) report that less symptomatic distress and severity predicts a better prognosis.

- Avoidance behavior (measured in individual dimensions as well as in a supplemental *Avoidance* dimensional set described in Appendix C) has been found in many studies to be a predictor of poor outcome (reviewed in Scheibe & Albus, 1996).
- The combination of anxious and depressive symptoms indicates a poorer prognosis than anxiety alone (Wittchen, Essau, & Krieg, 1991).
- Trait Anxiety (i.e., NA) has been found to be a significant predictor of outcome in certain types of phobias (Muris, Meyer, & Merckebach, 1998)
- Low social functioning (assessed in the *Social and Interpersonal Expressions* dimensional set) has been found to be a negative predictor of obsessive-compulsive disorder (Skoog & Skoog, 1999 Barlow cite)
- Those individuals with hoarding and symmetry/ordering concerns have been found to respond less well to behavioral and pharmacological interventions (see Steketee & Barlow, 2002). Therefore, symptoms have been sorted into hoarding/symmetry and non-hoarding/non-symmetry dimensions.
- The degree of behavioral avoidance in obsessive-compulsive disorder (as measured in the supplemental *Avoidance* dimensional set) is predictive of treatment outcome (reviewed in Newman et. al, in press).
- Symptom severity in disorders such as generalized anxiety disorder is a major predictor of long-term functioning (Angst & Vollrath, 1991). Therefore, the degree of distress and/or clinical impairment is assessed for each symptom.
- Individuals with generalized social anxiety symptomatology (assessed in the *Social and Interpersonal Expressions* dimensional set) typically remain more impaired after treatment (as cited in Newman et. al, in press).

In addition to these prognostic indicators, many other items were included in the dimensional sets for the purpose of increasing the breadth and comprehensiveness of the assessment of negative affectivity. Those directly relevant to the *IONAS-SR* will be discussed below.

The Seven Main Dimensional Sets.

The *Hygiene/Care of the Self* set includes a number of items that measure the extent to which typically human (and also animal) functions are impaired or preserved. Those most relevant to negative affectivity were included. Some of these functions can be found in the various DSM-IV categories, but others have been added where appropriate. Most fundamentally important to care of the self, this dimensional set assesses the presence of suicidal thoughts and the desire for non-being. Other dimensions assess disturbances in appetite, weight, and sleep. Sexual disturbances such as weak or strong libido, sexual performance difficulties, the avoidance of intercourse, and problems reaching orgasm were incorporated as well. These sexual disturbances have been found to be important considerations in the anxiety literature (e.g., Barlow, 2002) and are presumably affected by, or can lead to, negative affectivity. Three types of lassitude are assessed (*viz.*, physical, cognitive, and emotional) in order to serve as general markers of organismic well-being. Analysis of lassitude into three different manifestations was deemed important from a basic knowledge standpoint, as general “fatigue” or “energy loss” lacks specificity. Disturbances of volition are also assessed, as both productivity and the ability to meet important obligations presumably serve as markers of clinical impairment. Closely related to volition, the ability to concentrate/focus on a task was also included.

The *Melancholia* set closely mirrors the DSM-IV’s symptomatology for depression and dysthymia, but contains additional features. Characteristic symptoms such as depressed mood, irritability, crying, and anhedonia are all assessed, as are the symptoms

of guilt, worthlessness, hopelessness, and self-criticality. Subjective feelings of dissatisfaction with one's life and pessimism towards the future are included as well. Several symptoms address the temporal nature of melancholia and negative affectivity. A focus on the past and past failures (Borkovec & Sharpless, 2004), as well as the presence of intrusive thoughts about such failures are all assessed.

The intention of the *Somatic Expressions* set is to capture the various bodily expressions of NA. This includes both symptoms that are often thought of as paroxysmal (e.g., fast pulse or becoming dizzy when anxious) as well as those usually conceptualized as more chronic and perseverative (e.g., muscle tension and subjective feelings of restlessness) are assessed. As can be seen with these examples, this set possesses a high degree of overlap with DSM-IV symptoms. It does, however, contain several novel components. Specifically, it directly assesses what is thought to be a core component of NA (*viz.*, taking a long time for one's body to return back to an even keel when aroused and upset). In addition, it includes several reactions to experiencing anxiety or embarrassment such as flushing, having shaky/sweaty hands, and feeling the need to urinate. Symptoms such as headaches and teeth grinding (bruxism) at night are also included due to the fact that they are often associated with negative affectivity.

The *Cognitive Expressions* set derives its components from DSM-IV criteria (especially generalized anxiety disorder) as well as general NA theory and western philosophical sources. With regard to the DSM, worry, the excessive nature of worry, and the uncontrollability of worry are all prime components of this set. Also included are the ideas that worry may be used defensively in order to divert focus from more

important concerns (i.e., worry as avoidance) as well as worrying that bad events in the past will be repeated. Adrian Wells' (1994a) concept of "meta-worry" is assessed as well, as the worry process itself can become the object of apprehensive thought. The future directedness of worry and other expressions of NA were also thought to warrant inclusion, as were difficulties remaining "in the present moment" (both reviewed in Borkovec & Sharpless, 2004). A core cognitive feature of NA (*viz.*, difficulties with thoughts slowing down and returning to normal when upset) was added to serve as a cognitive parallel to the similarly worded *Somatic Expression* item. Assessing preoccupation with existential issues was also deemed important. Though not often discussed in the mainstream nosological literatures, anxiety over death, preoccupation with non-being, and questions of meaning are recurring human themes that hold important positions in both existential and psychoanalytic approaches. While certainly endemic to the human condition, an excessive focus on these issues could be problematic and interfere with an individual's life. It was included in this particular dimension due to the self-reflective nature of such concerns. The concept of tergiversation (*viz.*, the avoidance of straightforward action through excessive reflection and deliberation) artfully described by the Danish philosopher Kierkegaard (1846/1962), and sometimes termed "excessive reflection" in the psychological literature, was included. The more general subjective feeling of "stuckness" in life was included as well. These two items and another assessing general difficulty making decisions were added for their clinical relevance as well as for the purpose of adding additional breadth.

The *Social and Interpersonal Expressions* dimensional set contains a broad range of items focused on symptomatology related to interactions with others. It is the largest dimensional set of the *IONAS* for several reasons. First, when the DSM-IV categorical system is used, social anxiety disorder (also known as “social phobia”) has been found to be the most common type of anxiety disorder and is also the third most common mental disorder in the population (reviewed in Hofmann & Barlow, 2002). This obviously implies that such difficulties are not uncommon. Second, the ubiquity of social situations means that impairment in this area can have far-reaching implications for an individual’s life. Third, the complex nature of human interaction, along with the wealth of possible situations and dynamics, makes this a particularly wide (and difficult to operationalize) category. Whereas it is impossible to adequately (and briefly) represent the range of social and interpersonal phenomena with regard to NA, a decision was made to assess the broad sweep of an individual’s social difficulties as well as the generalizability of these difficulties. Items were derived from the DSM-IV, the Anxiety Disorders Interview Schedule (ADIS), and other sources. As verbal communication with others is fundamental to functioning, the presence of anxious apprehension and avoidance over the “range” of conversation (i.e., starting, continuing, and stopping a conversation) is separately assessed. Similarly, the *IONAS* separately assesses the degree of anxious apprehension and avoidance over a range of social situations (formal and informal speaking, everyday activities, situations that demand assertiveness, etc.). The apprehension one experiences in the company of groups of people is separately analyzed and assessed. Groups include friends, family, peers (coworkers/classmates), and

“authority figures.” One of the hallmarks of the DSM-IV’s social anxiety disorder is the fear of embarrassment in public. This is included as well as the fear that one will embarrass others (a manifestation of social anxiety common in Asian cultures and termed *taijin kyofusho* in Japan and Korea). Finally, 2 items focus on what could be called “impression management” or the use of a “persona” and rate the degree of anxious apprehension and avoidance individuals have in letting others come to know them on a personal level.

A number of the symptoms and phenomena included in the DSM-IV categories of specific phobia, panic disorder, and agoraphobia can be found in the dimensional set titled *Fears and the Experience of Threat*. For this set, various situations and objects covered by DSM-IV (e.g., animals, flying, situations in which escape might be difficult or embarrassing) are rated separately for the extent of fear and the degree of avoidance. Given that blood injury-injection phobic responses are associated with both a different physiological response (“vasovagal syncope,” or the process through which heart rate and blood pressure suddenly drop as described by Lewis, 1932) and different treatment recommendations, their respective degree of fear and avoidance are endorsed separately. Finally, the phenomenon of panic is assessed along with two of its most frequent indicators (feeling that one is going crazy and feeling like one is going to die).

The seventh main dimensional set of the *IONAS* assesses *Obsessive and Compulsive Expressions* of NA. The major DSM-IV symptoms are represented. With regard to phenomena typically labeled as “obsessive”, the *IONAS* assesses the presence of repetitive thoughts that are often the object of attempted suppression/avoidance as well

as repetitive thoughts that, although often present, do not make sense. An addition item inquires into Salkovskis' concept of "thought-action fusion," or the belief that thinking something is as bad as doing it. This concept has recently been related to NA in general, and is not specific to obsessive-compulsive disorder (Abramowitz et. al, 2003). Also included in this set are various types of compulsive actions. These items are assessed according to the degree of distress that would be generated if one were unable to perform such acts. These compulsive acts include: compulsions to count, preoccupations with symmetry, the compulsion to "hoard" objects, washing compulsions, and difficulties deviating from established routines.

Supplemental Sets.

Given the number of items and the breadth of NA phenomena covered in the *IONAS*, the partitioning of questions into dimensional sets is extremely flexible and very conducive to answering specific research and clinical questions. Many supplemental dimensional sets are derivable than the seven listed above, and some of these are discussed briefly below. Specific item content can be found in Appendix C.

A rather large (i.e., 18 question) dimensional set can be derived from the *IONAS-SR* that consists of totaling the various items assessing *Avoidance* behaviors. It essentially yields a measure of the generalization of avoidance. Other, smaller dimensional sets can be derived to measure such units of interest as generalized lassitude, presence of suicidal ideation, degree of NA reactivity, generalization of social fears, and

generalization of social avoidance. Others are certainly possible, but will not be discussed at this time.

The Present Study

Although the content of the *IONAS-SR* was derived from a number of sources (some of which have been empirically established) and the items possess face validity, the measure's descriptive statistics and psychometric properties have not been established. The study proposed here is a preliminary investigation for describing descriptive statistics and psychometric properties for the *IONAS-SR*. The following hypotheses were tested.

Hypotheses.

1. Descriptive statistics of the *IONAS-SR* will be gathered. Given that a non-clinical student sample will be utilized, it is predicted that the sample distribution will cluster towards the lower end of the range (i.e., a mean of less than 100). As is typical with other measures of mood and anxiety, it is predicted that males will score lower than females.
2. The test-retest reliability of the *IONAS-SR* will be assessed using the *IONAS-SR* at time one and the *IONAS-SR* a week later. Although theory would predict that symptomatic fluctuations occur over time, reliability over this short interval is expected to be high. In addition, test-retest correlations and reductions in score from the initial testing to retesting (i.e., the psychometric attenuation effect, as reviewed in Edelbrock et. al, 1985 and Jensen et al., 1993) will be assessed.

3. The internal consistency of the *IONAS-SR* and its dimensions will be assessed utilizing Cronbach's alpha. An overall alpha of at least 0.80 will be considered acceptable.
4. The validity of the *IONAS-SR* as a measure of negative affectivity will be tested using four criterion measures. These will be the negative affect dimension of the *Multidimensional Anxiety Questionnaire (MAQ)*, Reynolds, 1999), the *Positive and Negative Affect Scale (PANAS)*, Watson, Clark & Tellegen, 1988), the *Beck Depression Inventory, Second Edition (BDI-II)*, Beck 1996), and the *State-Trait Anxiety Inventory – Trait Version (STAI-T)*, Spielberger et al., 1983). It is predicted that the *IONAS-SR*'s total score will positively correlate highly with the total scores of the *MAQ*, the NA scale of the *PANAS*, and the *STAI-T*. The *IONAS-SR* as a whole will correlate slightly less with the *BDI-II*, but will still demonstrate a strong relationship due to the presence of NA in the latter's item content (Watson & Clark, 1984). However, it is predicted that the *Melancholia* dimension of the *IONAS-SR* will correlate more strongly with the total scores of the *BDI-II* and will negatively correlate to a higher degree with the positive affect scale of the *PANAS*.
5. The discriminant validity of the *IONAS-SR* will be tested by correlating the *IONAS-SR* and its dimensions to a self-report measure in a content domain conceived of as unrelated to NA (e.g., aggression). Correlations no higher than 0.30 will be expected.

6. Finally, the simple structure of the *IONAS-SR* will be assessed using an exploratory principal component analysis with varimax rotation.

Method

Participants

A total of 371 participants took part in this study. All were undergraduate students derived from the Pennsylvania State University psychology subject pool and voluntarily took part in this research as part of their course credit. Participants signed up over the internet. Of these 371, 77 participants volunteered for the test-retest portion of the study and were included on a first-come, first-serve basis. For this portion of the study, an equal numbers of slots were open for men and women.

Regarding the sample as a whole, 117 men (31.7%) and 252 women (68.3%) participated (note: two individuals did not include gender data). Participants had a mean age of 19.03 years ($SD = 1.23$, range = 18-28), estimated GPA of 3.26 ($SD = .44$, Range = 1.00 – 4.00), and an estimated semester standing of 2.63. Males did not significantly differ from females on any of these variables. As for ethnicity, there were 305 Caucasians (82.2%), 33 Asians/Pacific Islanders (8.9%), 14 African-Americans (3.8%), 10 Latino/Hispanics (2.7%), 5 who described themselves as “Other” (1.3%), and 4 interracial participants (1.1%).

The test-retest sub-sample ($n = 77$) consisted of 39 men (50.6%) and 38 women (49.4%). This group did not differ from the test-only group with regard to age or semester standing, but did display a significantly lower GPA ($M = 3.15$, $SD = .56$) than the test only group ($M = 3.30$, $SD = .40$), $F(1,367) = 6.857$, $p < .01$. With regard to ethnicity, there were 64 Caucasians (83.1%), 5 Asians/Pacific Islanders (6.5%), 4 Latinos/Hispanics (5.2%), 3 African-Americans (3.9%), and 1 other (1.3%).

Procedures

All participants were treated in accordance with the ethical standards of the APA. After signing the informed consent forms, the entire sample was administered the *IONAS-SR*, the *MAQ-NA*, the *BDI-II*, the *PANAS*, the *STAI-T*, and the *AQ*. They were tested in quiet classroom settings in groups of no more than 50 participants. Following the initial session, those who took part in the test-retest reliability sub-sample were scheduled for a second session held exactly one week later. At this time individuals were administered the *IONAS-SR* only.

Measures

IONAS-SR

The *IONAS-SR* (see Appendix B) is a self-report measure of negative affect symptomatology measured on a 0-3 scale. It contains 100 items that are further subdivided into 7 dimensional sets measuring *Hygiene/Care of the Self*, *Melancholia*, *Bodily Expressions of NA*, *Cognitive Expressions of NA*, *Social and Interpersonal Expressions of NA*, *Fears and the Experience of Threat*, and *Obsessive and Compulsive Expressions of NA*. A total score can be derived, as can individual scores for each dimensional set. Several supplemental scales are also derivable, and samples of these are listed in Appendix C.

MAQ-NA

The negative affect dimension of the *Multi-Dimensional Anxiety Questionnaire* (MAQ) consists of 9 items that assess negative affect symptomatology. Clinical norms are available, and it has been widely utilized. The *MAQ* has been demonstrated to be reliable and valid (Reynolds, 1999).

PANAS

The *PANAS* is a widely used 20-item self-report measure in which half of the items assess NA, and the other half assesses positive affect. The two factors have been found to correlate very highly with other commonly used measures of clinical symptomatology (Watson, Clark, & Tellegen, 1988; Watson & Clark, 1999). The *PANAS*'s items provide reliable, precise, and largely uncorrelated measures of NA and PA, regardless of the subject population studied (Ibid.).

BDI-II

The *Beck Depression Inventory – Second Edition (BDI-II)* is a 21-item self-report measure of depressive symptomatology developed to assess the existence and severity of DSM-IV (1994) symptoms of depression. Items are rated on a 4-point scale (0-4). It is probably the most widely used self-report measure of depression in the field of clinical psychology, and clinical norms are available. It has been shown to be reliable and valid, and has been extensively studied (Beck, 1996).

STAI-T

The *State-Trait Anxiety Inventory – Trait Version (STAI-T)* is a widely used measure of the relatively persistent and stable individual differences in anxiety proneness. It has been shown to be reliable and valid (Spielberger et al., 1983) and correlates highly with other instruments that measure NA (e.g., $r = .80$ with the *Taylor Manifest Anxiety Scale*, citation from website). The *STAI-S* (State Version) was omitted.

AQ

The *Aggression Questionnaire (AQ)* (Buss & Perry, 1992) is a 29-item self-report instrument that measures trait aggression. It is scored on a 1-5 scale, and two items are reverse scored. Along with a total score, the measure can be further analyzed into four subscales measuring physical aggression, verbal aggression, anger, and hostility. Several studies have supported the psychometric soundness of this measure (as reviewed in Tremblay & Ewart, 2005), and it is probably the most widely used self-report measure of aggression.

Results

Descriptive Statistics

Table 1 presents means and standard deviations for both the raw score of items (sums) for each construct (i.e., the total and dimensional set scores) as well as the average item scores (i.e., the sum divided by the number of items). A wide range of raw scores was evident (6 to 236), and skewness in the distribution was positive (*skewness* = 0.78). *Melancholia* evidenced the highest average score, whereas the lowest average score was found in *Fears and the Experience of Threat*. Table 1 also presents means, standard deviations, and *F*-test values for gender on each construct. Males and females were found to significantly differ, with females scoring higher than males on the total score and all but two dimensional sets (*viz.*, *Social and Interpersonal Expressions* and *Obsessive and Compulsive Expressions*). (Given this difference, analyses were also conducted separately on each gender, and these results can be found in relevant appendices.) Although a one-way ANOVA using ethnicity as the grouping variable was significant ($F(5, 365) = 8.25, p < .001$), post-hoc tests were not conducted due to small sample sizes among ethnic minority groups (see Appendix D for means and standard deviations by group for the total score as well as the dimensional sets).

Table 1

IONAS-SR and its Dimensional Sets' Descriptive Statistics: Means, Standard Deviations, F-Values, Number of Items, and Average Scores for the Total Sample and by Gender

| | Gender | M | SD | N | Items | AS | SD |
|-------------------------------|--------|-------|-------|--------|-------|------|------|
| Total | Male | 68.85 | 38.49 | 117 | 100 | 0.69 | 0.38 |
| | Female | 78.10 | 41.63 | 252 | 100 | 0.78 | 0.42 |
| | Total | 74.96 | 40.82 | 371 | 100 | 0.75 | 0.41 |
| $F(1, 368) = 4.129, p < .05$ | | | | | | | |
| Hy | Male | 12.27 | 6.06 | 117 | 14 | 0.88 | 0.43 |
| | Female | 13.85 | 6.44 | 252 | 14 | 0.99 | 0.46 |
| | Total | 13.30 | 6.36 | 371 | 14 | 0.95 | 0.45 |
| $F(1, 367) = 4.941, p < .03$ | | | | | | | |
| Mel | Male | 11.61 | 6.92 | 117.00 | 13 | 0.89 | 0.53 |
| | Female | 13.27 | 7.57 | 252 | 13 | 1.02 | 0.58 |
| | Total | 12.70 | 7.41 | 371 | 13 | 0.98 | 0.57 |
| $F(1, 367) = 4.082, p < .05$ | | | | | | | |
| Soma | Male | 7.22 | 5.32 | 117 | 13 | 0.56 | 0.41 |
| | Female | 9.37 | 6.25 | 252 | 13 | 0.72 | 0.48 |
| | Total | 8.67 | 6.04 | 371 | 13 | 0.67 | 0.46 |
| $F(1, 367) = 10.368, p < .01$ | | | | | | | |
| Cog | Male | 10.97 | 7.00 | 117 | 13 | 0.84 | 0.54 |
| | Female | 13.19 | 7.51 | 252 | 13 | 1.01 | 0.58 |
| | Total | 12.44 | 7.41 | 371 | 13 | 0.96 | 0.57 |
| $F(1, 367) = 7.290, p < .01$ | | | | | | | |
| SIP | Male | 16.80 | 11.64 | 117 | 24 | 0.70 | 0.49 |
| | Female | 16.53 | 12.60 | 252 | 24 | 0.69 | 0.52 |
| | Total | 16.57 | 12.28 | 371 | 24 | 0.69 | 0.51 |
| $F(1, 367) = .037, p > .80$ | | | | | | | |
| FT | Male | 5.98 | 4.85 | 117 | 15 | 0.40 | 0.32 |
| | Female | 7.83 | 6.18 | 252 | 15 | 0.52 | 0.41 |
| | Total | 7.23 | 5.84 | 371 | 15 | 0.48 | 0.39 |
| $F(1, 367) = 8.115, p < .01$ | | | | | | | |
| OC | Male | 4.01 | 3.46 | 117 | 8 | 0.50 | 0.43 |
| | Female | 4.06 | 3.66 | 252 | 8 | 0.51 | 0.46 |
| | Total | 4.04 | 3.57 | 371 | 8 | 0.51 | 0.45 |
| $F(1, 367) = .016, p > .89$ | | | | | | | |

Notes: AS = Average Score, Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, and OC = Obsessive and Compulsive Expressions.

Reliability

Test-Retest Reliability

Test-retest reliability was acceptable for the tested sub-sample as well as for men and women. Means of summed items, standard deviations, and test-retest correlations for the total *IONAS-SR* and the seven dimensional sets can be found in Table 2 (gender data can be found in Appendix E). Of the seven dimensional sets, the highest test-retest reliability was found for *Melancholia*, and the lowest was found for the *Obsessive and Compulsive Expressions* set.

Attenuation from the initial testing to the retest was assessed using paired-sample t-tests, and mean change scores can be found in Table 2. Mean change for the total score was significant, $t(74) = 7.69, p < .001$, and indicated that retest scores were significantly lower at re-testing. Mean change for each dimensional set was also significant (all p 's < .001), with t 's ranging from 3.65 to 7.27. This pattern was also found when data were separately analyzed by gender. Males evidenced significant score reductions from test to retest, $t(36) = 4.87, p < .001$, as did females, $t(37) = 6.14, p < .001$, but men and women did not significantly differ on mean change ($p > .96$) when a t-test was conducted using gender as a grouping variable. Similar results were found when individual dimensional sets were analyzed according to gender, and relevant statistics can be found in Appendix E.

Table 2

Test-Retest Reliability and Mean Change Scores for the *IONAS-SR* and its Dimensions

| Measure | r | Test M | Test SD | Retest M | Retest SD | MC |
|--------------|-----|--------|---------|----------|-----------|--------------------------|
| Total | .87 | 74.96 | 40.82 | 53.84 | 35.69 | 15.99 |
| | | | | | | $t(74) = 7.69, p < .001$ |
| Hy | .82 | 13.30 | 6.36 | 9.71 | 5.49 | 2.81 |
| | | | | | | $t(74) = 7.27, p < .001$ |
| Mel | .89 | 12.70 | 7.41 | 10.37 | 7.78 | 1.73 |
| | | | | | | $t(74) = 4.17, p < .001$ |
| Soma | .71 | 8.67 | 6.04 | 5.17 | 4.61 | 1.79 |
| | | | | | | $t(74) = 4.25, p < .001$ |
| Cog | .83 | 12.44 | 7.41 | 9.40 | 7.28 | 2.21 |
| | | | | | | $t(74) = 4.56, p < .001$ |
| SIP | .88 | 16.57 | 12.28 | 11.88 | 10.15 | 4.81 |
| | | | | | | $t(74) = 8.05, p < .001$ |
| FT | .76 | 7.23 | 5.84 | 4.68 | 4.07 | 1.55 |
| | | | | | | $t(74) = 4.10, p < .001$ |
| OC | .61 | 4.04 | 3.57 | 2.65 | 2.65 | 1.05 |
| | | | | | | $t(74) = 3.65, p < .001$ |

Notes: MC = Mean Change, Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, OC = Obsessive and Compulsive Expressions, and MC = Mean Change.

All correlations significant with p 's < .001

Internal Consistency

Table 3 displays Cronbach's alphas for the total *IONAS-SR* and for each dimension (N = 371). As predicted, the *IONAS-SR* was found to be highly consistent. On individual dimensions, only the *Obsessive and Compulsive Expressions* set was low, whereas the remaining alphas ranged from .80 (*Fears and the Experience of Threat*) to 0.94 (*Social and Interpersonal Expressions*).

Table 3

Internal Consistency: Cronbach Alphas for the *IONAS-SR* and its Dimensional Sets

| | Alpha |
|-----------------|--------------|
| IONAS-SR | 0.97 |
| Hy | 0.83 |
| Mel | 0.89 |
| Soma | 0.82 |
| Cog | 0.88 |
| SIP | 0.94 |
| FT | 0.80 |
| OC | 0.73 |

Notes: Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, and OC = Obsessive and Compulsive Expressions.

Validity

Criterion Validity

Criterion validity for the *IONAS-SR* was assessed using 4 measures (*MAQ-NA*, *PANAS*, *BDI-II*, and *STAI-T*), and correlational results for the total and dimensional set scores can be found in Table 4. The *IONAS-SR* was most strongly associated with the *STAI-T*, followed closely by the *BDI-II*, *MAQ-NA*, and the *PANAS-NA*. All of these correlations were also significant within each gender (Appendix F).

Hypothesis 3 was partially supported. Contrary to prediction, the correlation between the *BDI-II* and *Melancholia* was not significantly higher than the *BDI-II*'s

Table 4

IONAS-SR and Dimensional Sets' Correlations with Criterion Validity Measures

| | BDI | | MAQ | | PANAS-NA | | PANAS_PA | | STAI | |
|--------------|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> |
| Total | .75** | 364 | .72** | 371 | .69** | 371 | -.26** | 371 | .78** | 371 |
| Hy | .67** | 364 | .65** | 371 | .58** | 371 | -.23** | 371 | .64** | 371 |
| Mel | .75** | 364 | .75** | 371 | .72** | 371 | -.34** | 371 | .81** | 371 |
| Soma | .61** | 364 | .62** | 371 | .52** | 371 | -.11** | 371 | .56** | 371 |
| Cog | .69** | 364 | .73** | 371 | .67** | 371 | -.27** | 371 | .76** | 371 |
| SIP | .56** | 364 | .47** | 371 | .51** | 371 | -.23** | 371 | .62** | 371 |
| FT | .52** | 364 | .48** | 371 | .51** | 371 | -.10 | 371 | .52** | 371 |
| OC | .58** | 364 | .54** | 371 | .52** | 371 | -.11* | 371 | .56** | 371 |

Notes: *Hy* = Hygiene/Care of the Self, *Mel* = Melancholia, *Soma* = Somatic Expressions, *Cog* = Cognitive Expressions, *SIP* = Social and Interpersonal Expressions, *FT* = Fears and the Experience of Threat, and *OC* = Obsessive and Compulsive Expressions.

** Correlation is significant at .001 level

* Correlation is significant at .05 level

correlation with the entire *IONAS-SR*. However, *Melancholia* negatively correlated with the positive affect factor of the *PANAS* more strongly than the entire *IONAS-SR* and the other dimensions for both the total sample and females. However, as shown in Appendix F, males did not display a significant relationship.

With regard to the dimensional sets' relation to the criterion measures, all were significant with the exception of the correlation between the positive affect factor of the *PANAS* and the *Fears and the Experience of Threat* dimensional set ($p > .06$). The five highest correlations (in descending order) were the *STAI-T* with *Melancholia* and *Cognitive Expressions*, the *BDI-II* with *Melancholia*, and the *MAQ* with *Melancholia* and *Cognitive Expressions*.

Discriminant Validity

The discriminant validity of the *IONAS-SR* was tested through assessing its relationship with the *AQ*. Correlations between the *IONAS-SR* (and its dimensional sets) and the *AQ* (and its dimensions) can be found in Table 5 (with analyses by gender found in Appendix G). The correlation between *IONAS-SR* total scores and the *AQ* was significant and somewhat higher than anticipated. Supplementary analyses explored the *IONAS-SR*'s correlations with the *AQ*'s 4 subscales of *physical aggression*, *verbal aggression*, *anger*, and *hostility*. Three of the four (*physical aggression*, *anger*, and *hostility*) were significantly correlated with the *IONAS-SR*, but the correlation for *verbal aggression* was not. A partial correlation between the *IONAS-SR* and the *AQ* (controlling

for *physical aggression, anger, and verbal aggression*) yielded an R^2 of .27 and indicated that the *hostility* subscale was responsible for the majority of the shared variance.

Table 5

IONAS-SR and Dimensional Sets' Correlations with Discriminant Validity Measure

| | AQ | | AQ-PAg | | AQ-VAg | | AQ-Ang | | AQ-Hos | |
|--------------|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> |
| Total | .37** | 371 | .19** | 371 | .07 | 371 | .28** | 371 | .56** | 371 |
| Hy | .34** | 371 | .22** | 371 | .12* | 371 | .25** | 371 | .45** | 371 |
| Mel | .41** | 371 | .22** | 371 | .12* | 371 | .32** | 371 | .57** | 371 |
| Soma | .30** | 371 | .18* | 371 | .12* | 371 | .23** | 371 | .40** | 371 |
| Cog | .35** | 371 | .14* | 371 | .09 | 371 | .29** | 371 | .53** | 371 |
| SIP | .24** | 371 | .10 | 371 | -.04 | 371 | .17* | 371 | .47** | 371 |
| FT | .25** | 371 | .13* | 371 | .03 | 371 | .21* | 371 | .37** | 371 |
| OC | .24** | 371 | .15** | 371 | .05** | 371 | .15* | 371 | .37** | 371 |

Notes: *Hy* = Hygiene/Care of the Self, *Mel* = Melancholia, *Soma* = Somatic Expressions, *Cog* = Cognitive Expressions, *SIP* = Social and Interpersonal Expressions, *FT* = Fears and the Experience of Threat, *OC* = Obsessive and Compulsive Expressions, *AQ-PAg* = Physical Aggression, *AQ-VAg* = Verbal Aggression, *AQ-Ang* = Anger, and *AQ-Hos* = Hostility.

** Correlation is significant at .001 level

* Correlation is significant at .05 level

Factorial Validity

The 100 items of the *IONAS-SR* were submitted to a principle components analysis (PCA) with a varimax rotation using Statistical Package for the Social Sciences (SPSS) version 11.0.0. Perusal of the correlation matrix indicated that the vast majority of inter-item correlations were significant. Singularity did not appear to be present, however, as no correlations rose above .75. The Kaiser-Meyer-Olkin measure of sampling adequacy was .92 (“superb”, Hutcheson & Sofroniou, 1999) and indicated that

the *IONAS-SR* yielded compact, distinct, and reliable factors. Bartlett's Test of sphericity was significant ($\chi^2(4950, N = 371) = 21042.74, p < .001$) and rejected the null hypothesis that the correlation matrix was an identity matrix.

Table 6

Total and Cumulative Variance Percentages of the *IONAS-SR*'s Factors

| Component | Initial Eigenvalues | | Extraction Sums of Squared Loadings. | | Rotation Sums of Squared Loadings | |
|-----------|---------------------|--------|--------------------------------------|--------|-----------------------------------|--------|
| | % of Var. | Cum. % | % of Var. | Cum. % | % of Var. | Cum. % |
| 1 | 26.31 | 26.31 | 26.31 | 26.31 | 12.58 | 12.58 |
| 2 | 5.28 | 31.59 | 5.28 | 31.59 | 12.44 | 25.02 |
| 3 | 3.56 | 35.15 | 3.56 | 35.15 | 5.54 | 30.56 |
| 4 | 2.58 | 37.73 | 2.58 | 37.73 | 5.47 | 36.03 |
| 5 | 2.39 | 40.12 | 2.39 | 40.12 | 4.08 | 40.12 |
| 6 | 2.12 | 42.23 | | | | |
| 7 | 2.01 | 44.24 | | | | |
| 8 | 1.79 | 46.04 | | | | |
| 9 | 1.66 | 47.70 | | | | |
| 10 | 1.62 | 49.32 | | | | |
| 11 | 1.54 | 50.86 | | | | |
| 12 | 1.50 | 52.36 | | | | |
| 13 | 1.43 | 53.79 | | | | |
| 14 | 1.37 | 55.17 | | | | |
| 15 | 1.30 | 56.46 | | | | |
| 16 | 1.29 | 57.75 | | | | |
| 17 | 1.28 | 59.04 | | | | |
| 18 | 1.22 | 60.25 | | | | |
| 19 | 1.20 | 61.45 | | | | |
| 20 | 1.15 | 62.60 | | | | |
| 21 | 1.10 | 63.70 | | | | |
| 22 | 1.07 | 64.77 | | | | |
| 23 | 1.02 | 65.80 | | | | |
| 24 | 1.02 | 66.81 | | | | |

Note: % of Variance = Total Variance due to the fact that the *IONAS-SR* contains 100 items.

Twenty-four factors with Kaiser's eigenvalues above 1 emerged, and variance for the individual factors can be found in Table 6. Cattell's scree test indicated a 5-factor solution, and PCAs were conducted for 4, 5, and 6-factor solutions. The 5-factor solution was ultimately retained⁴, and variance for the rotated solution and the individual factors can also be found in Table 6.

Qualitative analysis and interpretation of the items comprising each factor yielded (in descending order of variance) (1) *NA/Neurasthenia*, (2) *Social and Interpersonal Anxiety*, (3) *Suicide/Life Dissatisfaction*, (4) *Sexual Difficulty and Compulsive Behavior*, and (5) *Phobic Fear and Avoidance*. The individual *IONAS-SR* items that make up these factors, as well as their respective loadings, can be found in Table 7. Items were included if their loadings were above .35. In the event of an item loading on two factors, the item was relegated exclusively to its highest loading factor.

The *NA/Neurasthenia* factor contains a number of "classic" NA items (e.g., worry, depression, guilt, reactivity and difficulty returning to an even keel) as well as items measuring lassitude, paroxysmal anxiety, somatic complaints, and difficulties remaining "in the present moment". Thus, it was named for its close correspondence to the (currently anachronistic) diagnosis of neurasthenia (see pg. 52 of the present work for a brief symptom list). The *Social and Interpersonal Anxiety* factor contained 88% of the items from the corresponding *IONAS-SR* dimensional set (*Social and Interpersonal Expressions*) as well as items measuring agoraphobic symptoms, tergiversation, and difficulty making decisions. The *Suicide/Life Dissatisfaction* factor primarily consists of items from the *Melancholia* dimensional set as well as items from two other sets (*viz.*,

Table 7

Factor Loadings for Individual *IONAS-SR* Items

| | | Factor | | | | |
|---|----------------------------------|----------|----------|----------|----------|----------|
| | Factor 1: NA/Neurasthenia | 1 | 2 | 3 | 4 | 5 |
| 97 "I've felt emotionally fatigued" | | .72 | | | | |
| 45 "My worry has been excessive" | | .69 | | | | |
| 28 "When I've been upset, it's taken a long time for my thoughts to slow down and get back to normal" | | .68 | | | | |
| 44 "When I've been upset, it's taken a long time for my body to relax and get back to normal" | | .65 | | | | |
| 2 "I've felt mentally fatigued" | | .64 | | | | |
| 78 "It's been difficult to start worrying once I've started" | | .64 | | | | |
| 29 "I've felt restless, agitated, keyed up, or on edge" | | .62 | | | | |
| 6 "I've worried" | | .62 | | | | |
| 36 "I've felt depressed or down" | | .60 | | .46 | | |
| 42 "I felt like I was going crazy" | | .56 | | | | |
| 26 "I've been critical of myself" | | .54 | | | | |
| 16 "I've had repetitive thoughts that I've tried to suppress, ignore, or avoid" | | .54 | | | | |
| 22 "I've cried" | | .54 | | | | |
| 17 "I've felt irritable" | | .54 | | | | |
| 27 "I've had aches and pains" | | .53 | | | | |
| 18 "I've slept either too much or too little" | | .53 | | | | |
| 60 "I've had muscle tension" | | .50 | | | .39 | |
| 86 "I've felt stuck in my life" | | .50 | | .45 | | |
| 68 "I've worried about worrying" | | .50 | | | | |
| 10 "I've had problems concentrating or staying focused" | | .49 | | | | |
| 38 "I've had headaches" | | .48 | | | | |
| 80 "I've worried that I will repeat bad things that have happened to me in the past" | | .48 | | | | |
| 8 "I've felt physically fatigued" | | .48 | | | | |
| 87 "Thoughts about past failures entered my mind when I didn't want them to" | | .47 | | | | |
| 3 "I've thought about past failures or bad things that happened in my past" | | .47 | | | | |
| 89 "I've had panic attacks" | | .46 | | | | |
| 11 "I've had difficulty getting important things done" | | .45 | | | | |
| 41 "When anxious, I've felt light headed, dizzy, or faint" | | .44 | | | .38 | |
| 74 "I've felt pessimistic" | | .40 | | .37 | | |
| 71 "It's been difficult for me to stay in the present moment" | | .40 | | | | |

| | | | | | |
|--|-----|---------------|----------|----------|----------|
| 50 "I've worried about minor events to keep from thinking about more important concerns" | .39 | | | | |
| 24 "I've felt guilty" | .38 | | | | |
| 15 "I've flushed or had hot flashes when I was anxious or embarrassed" | .38 | | | | |
| 20 "I've thought more about the past than the present or future" | .38 | | | | |
| 21 "I've lost interest or pleasure in things that I used to enjoy" | .37 | | | | .36 |
| 92 "I've been bothered by thoughts that keep coming back to me but don't make sense" | .35 | | | | |
| | | Factor | | | |
| | | 1 | 2 | 3 | 4 |
| | | | | | 5 |
| Factor 2: Social and Interpersonal Anxiety | | | | | |
| 84 "I've been anxious about informally speaking or performing" | | | .78 | | |
| 58 "I've avoided speaking or performing in informal situations" | | | .76 | | |
| 75 "I've avoided speaking or performing in public" | | | .71 | | |
| 65 "I've been fearful that I would embarrass myself in social situations" | | | .70 | | |
| 57 "I've been anxious keeping conversation going" | | | .69 | | |
| 12 "I've been anxious starting conversations with people" | | | .68 | | |
| 85 "I've been fearful of crowds" | | | .67 | | |
| 94 "I've avoided starting conversations with people" | | | .66 | | |
| 47 "I've avoided social situations that are unclear or ambiguous" | | | .63 | | |
| 96 "I've avoided keeping conversations going" | | | .62 | | |
| 77 "I've been anxious when I've had to assert myself" | | | .62 | | |
| 30 "I've been anxious in social situations that are unclear or ambiguous" | | | .62 | | |
| 56 "I've avoided situations in which leaving might be difficult or embarrassing" | | | .60 | | |
| 48 "I've been anxious about public speaking or performances" | | | .59 | | |
| 66 "I've been fearful that I would offend other people in social situations" | | | .55 | | |
| 37 "I've been anxious when trying to stop conversations with people" | | | .53 | | .36 |
| 53 "I've avoided asserting myself" | | | .53 | | |
| 72 "I've been anxious around authority figures" | | | .53 | | |
| 34 "I've been afraid of situations in which leaving might be difficult or embarrassing: | | | .50 | | |
| 4 "I've avoided being in crowds" | | | .49 | | |
| 23 "I've been anxious about letting people know the real me" | | | .47 | | |
| 5 "People watching me do everyday activities makes me anxious" | | | .45 | | |
| 61 "I've avoided stopping conversation with people" | | | .44 | | |
| 9 "I've been anxious around my friends" | | | .41 | | |
| 52 "I've had difficulty making decisions" | .36 | | .40 | | |
| 40 "I've avoided letting people get to know the real me" | | | .38 | | .42 |
| 54 "I've thought about choices so much that I end up not taking action" | | | .38 | | |
| 79 "I've avoided doing everyday activities in front of other people" | | | .37 | | |

| | Factor | | | | |
|--|---------------|----------|----------|----------|----------|
| | 1 | 2 | 3 | 4 | 5 |
| Factor 3: Suicide/Life Dissatisfaction | | | | | |
| 64 "I've felt that I would be better off dead" | | | .77 | | |
| 93 "I've had thoughts about suicide" | | | .73 | | |
| 39 "I've felt worthless" | .43 | | .64 | | |
| 90 "I felt like I was going to die" | | | .61 | | |
| 76 "I've felt hopeless" | .49 | | .55 | | |
| 14 "I've felt dissatisfied with my life" | .41 | | .53 | | |
| 40 "I've avoided letting people get to know the real me" | | .38 | .42 | | |
| Factor 4: Sexual Difficulty and Compulsive Behavior | | | | | |
| 99 "I've had problems with my sexual performance" | | | | .51 | |
| 33 "I've had breathing difficulties" | | | | .50 | |
| 25 "My sex drive/level of interest in sex has been either too strong or too weak" | | | | .50 | |
| 46 "I've had problems reaching sexual satisfaction" | | | | .49 | |
| 59 "I've had heart difficulties" | | | | .49 | |
| 98 "I felt as if I had to wash my body or a part of my body. If I didn't, I would feel anxious" | | | | .48 | |
| 95 "I've felt as if I had to hoard objects. If I didn't, I would feel anxious" | | | | .44 | |
| 67 "I've been anxious around my family members" | | | | .41 | |
| 1 "My appetite has been either too strong or too weak" | .37 | | | .41 | |
| 91 "I've gone out of my way to avoid sex" | | | | .39 | |
| 62 "I felt as if I had to make sure that objects are straight, or line up perfectly. If they didn't, I would feel anxious" | | | | .35 | |
| Factor 5 Phobic Fear and Avoidance | | | | | |
| 83 "I've avoided blood, injuries, injections, or the sight of them" | | | | | .75 |
| 55 "I've been fearful of blood, injuries, or injections" | | | | | .68 |
| 82 "I've avoided certain animals" | | | | | .68 |
| 31 "I've been fearful of certain animals" | | | | | .59 |
| 73 "I've avoided certain situations" | | | | | .54 |
| 7 "I've been fearful of being outside of my home alone" | | | | | .42 |
| 13 "I've been fearful of certain situations" | | | | | .42 |
| 88 "I've avoided being outside of my home alone" | | | | | .42 |

Note: Only factor loadings above .35 were included in this Table. If an IONAS-SR item did not have a factor loading, it was not included.

Fears and the Experience of Threat and Social and Interpersonal Expressions). Factor 4, *Sexual Difficulty and Compulsive Behavior*, is the least well-defined factor, and contains items from the *Hygiene/Care of the Self*, *Somatic Expressions*, and *Obsessive and*

Compulsive Expressions sets. Finally, the *Phobic Fear and Avoidance* factor is comprised of 53% of the items from the *Fears and the Experience of Threat* dimensional set.

Discussion

Hypothesis 1: Descriptive Statistics and Gender Differences

As predicted, means for the *IONAS-SR* and its seven dimensional sets fell below an average score of 1.00 (i.e., mild impairment or distress). This is noteworthy for at least two reasons. First, the available NA literature would expect negative affect symptoms to be present in a normal population. Evidence indicates that psychopathology in this particular domain is a matter of degree, and not kind (see Part V above). Second, the skew of the distribution implies a rather high ceiling level. Therefore, the *IONAS-SR*'s range can accommodate scores up to five and a half standard deviations beyond the full sample mean (*viz.*, in the direction of higher negative affectivity). This may prove useful when clinically assessing individuals with extreme levels of negative affect symptomatology.

As predicted, males scored lower than females on the total *IONAS-SR* score and the majority of dimensional sets. This is in keeping with the majority of self-report measures of depressive and anxious symptomatology. It is unclear why genders did not differ on the Social and Interpersonal and the Obsessive and Compulsive dimensional sets. However, the diagnoses of social phobia and obsessive-compulsive disorder are equally present in clinical samples of men and women, whereas the other anxiety and mood disorders show a higher rate of female prevalence (APA, 1994).

Hypothesis 2: Test-Retest Reliability

As predicted, test-retest reliability for the *IONAS-SR* was acceptably high and comparable to other instruments that measure similar clinical constructs. An attenuation effect was present (see Edelbrock, Crnic, & Bohnert, 1999 for a discussion of the phenomenon), and retest scores averaged 15.99 points lower than those of the initial testing. Reasons for this (ubiquitous psychometric) phenomenon are unclear, but may be due to artifacts of the self-report assessment method itself, demand characteristics, or other surreptitious factors.

Hypothesis 3: Internal Consistency

The *IONAS-SR* displayed high internal consistency when considered in its entirety and in all of the dimensional sets except *Obsessive and Compulsive Expressions* (.73).

Hypothesis 4: Criterion Validity

As predicted, the *IONAS-SR* was strongly associated with the NA criterion measures, and correlations ranged from .72 (*MAQ*) to .78 (*STAI-T*). These correlations, while high, do not imply redundancy of the assessed constructs and, as discussed in the section outlining the derivation of the *IONAS-SR*, unique item content is present.

The hypothesis that the *BDI-II* would associate more strongly with *Melancholia* than the total *IONAS-SR* was not supported. Reasons for this are unclear. Whereas the *BDI-II* is heavily NA-loaded and correlates strongly with other anxiety measures (sometimes more strongly than with other measures of depression as in Watson & Clark,

1984), it was expected that the overlap in item content with *Melancholia* would lead to a higher correlation.

Hypothesis 5: Discriminant Validity

Although considerably lower than correlations with the NA criterion measures, the correlation with the *AQ* was somewhat higher than expected and indicates that additional tests of the *IONAS-SR*'s discriminant validity are warranted. As supplementary analyses determined, the *IONAS-SR* was most strongly associated with the *Hostility* and *Anger* dimensions of the *AQ*. It is conceivable that the expressions of anger and scorn contained within this instrument may explain the *AQ*'s overlap with a measure of NA. Both of these emotional states have been conceived of as being contained within the construct of NA (Watson & Clark, 1984).

Hypothesis 6: Factorial Validity

A 5-Factor solution explained 40.12% of the *IONAS-SR*'s variance and also yielded interesting factors. The close resemblance of factor 1 (*NA/Neurasthenia*) to the 19th century diagnostic entity of neurasthenia was unexpected, and may imply that additional clinical and research attention should be devoted to symptoms such as lassitude (all 3 types), avolition, and difficulties remaining in the present moment as well as the more common DSM-IV symptoms (e.g., anhedonia, restlessness, panic attacks). Of particular note is the fact that every DSM-IV generalized anxiety disorder symptom and all but four symptoms (weight loss/appetitive disturbance, psychomotor retardation,

indecisiveness, and recurrent thoughts of death) of major depression and dysthymia were contained in this factor. This is entirely consistent with what the NA literature would predict. It is also noteworthy that both of the items that measure NA reactivity (viz., items 28 and 44) are contained in this factor, and are strongly loaded, lending additional credence to its title.

Items in the *Social and Interpersonal* factor closely mirrored the content of the *Social and Interpersonal Expressions* dimensional set. Some content, however, was different. Items measuring DSM-IV agoraphobia symptoms loaded onto this factor. This is certainly interpretable, as many of the core features of agoraphobia (e.g. fear of crowds, requiring a “safe person” to travel with) contain strong interpersonal elements. The inclusion of items assessing tergiversation and difficulty making decisions, however, is more difficult to explain. It may be that the potentially evaluative nature of deciding to take action (as well as actually *initiating* a course of action) opens up the possibility for negative critique and embarrassment. Also related to the notion of interpersonal embarrassment, it is important to note that the one item assessing *taijin kyofusho* (fear of offending others in social situations) loaded highly onto this factor.

The *Suicide/Life Dissatisfaction* factor shares a strong resemblance with a supplemental dimensional set measuring *Suicide Risks* that can be found in Appendix C. With the exception of the last item on this factor (avoiding letting others know “the real me”), all of the items seem to be easily interpretable and cohesive. It may be that this remaining item is a sequela of the others, as profound feelings of worthlessness and

inadequacy would understandably preclude an individual from wishing to be transparent to another.

The *Sexual Difficulty and Compulsive Behavior* factor is less clearly interpretable than the others. A 7-factor solution dissected this factor into three smaller, yet more unitary factors measuring obsessions/compulsions, somatic expressions of anxiety, and sexual disturbances. However, this solution left too few items for usable factor scales, and was abandoned. Reasons as to why the sexual disturbance items linked with compulsions are unclear, but psychoanalytic theory would certainly posit their close linkage in the form of the latter typically defending against the former.

Summary

By way of summary, the *IONAS-SR* demonstrated high reliability, internal consistency, and criterion validity, and displayed acceptable discriminant validity. The 5-factor structure provided a relatively good fit to the data and several factors paralleled the *a priori* dimensional sets. However, it is clear that a great deal of variance remains that is unaccounted for by the factors, so the entirety of the *IONAS-SR* will likely be retained in order to capture the wide swath of negative affectivity's expressions. One or more of the factors, however, may be useful in constructing a shortened form of the *IONAS-SR*.

The *IONAS-SR* was constructed to differ from existing measures in several respects. Namely, it focuses on clinical impairment for each symptom, intends to capture the broad topography of negative affectivity, and its scores can be viewed at 3 levels of abstraction (*viz.*, total NA score, dimensional set scores, and individual symptom scores).

Thus, it is flexible, comprehensive, and inclusive enough to be utilized in various research and applied settings and may be a useful addition to the current self-report armamentarium.

General Discussion

The present work attempted to provide a theoretical and historical critique of psychopathology classification. Those figures and theories most directly related to the intellectual history of concepts such as depression and anxiety were the primary foci, and this survey ranged from Hippocrates to van Praag. In reviewing this material, it became clear that the majority of issues that present-day nosologists must face (e.g., where and how to demarcate, the role of values in classification, goals for classification) have been present for quite some time. It is believed that the decisions others have committed to when struggling with analogous problems can be instructive in both negative and positive ways, and some of their approaches were subsequently adopted.

The ubiquitous and recurrent linkage between symptoms of depression and anxiety occupied the majority of this work. It was argued that contemporary psychopathology became increasingly alienated from this intimate linkage when DSM-III and its splitting ethos assumed hegemony. This splitting, however, facilitated and ushered in a period of intense basic research into clinical psychopathology and its associated features, and a wealth of empirical findings were generated. A number of these findings (as reviewed in Part IV) did not appear to fit within a (naïvely) atheoretical and categorical framework. However, a body of empirical research (*viz.*, NA) was

available and, when the seemingly anomalous findings were viewed from within its confines, they became less anomalous and, in fact, less unexpected. Various approaches have been taken to incorporate NA into contemporary classifications, and these attempts span from emphasis on the shared features (e.g., Andrews, 1996) to emphasis on the specific features while also recognizing their commonality (Barlow, 1998).

Taken together, it is clear that the current classification of mood and anxiety stands in need of revision. Empirical data and theoretical work would appear to favor a system that assumes the linkage between depression and anxiety over one that assumes, even implicitly, their ontological discreteness. The proposed alternative relies on a purely dimensional system. It was generated from a combination of pre-DSM knowledge of psychopathology as well as the innumerable empirical developments that arose since psychopathology began to be studied scientifically. Many of the dimensions were derived from the various DSM's, but they are utilized in a very different way, and no higher order classification is assumed at this point. At the present state of the research, the writer would argue in favor of maintaining these various symptoms within a single "diagnostic basin" until a preponderance of evidence suggests the need for additional splitting. What might be gained is the ability to research particular symptoms and their interrelations, and not just syndromes and their comorbidity. In the course of using measures such as the *IONAS-SR*, certain expressions of psychopathology may be discovered to link with the "basin" alone, whereas others may come to be associated with individual "disorders" that demonstrate a distinct point of rarity with NA. Regardless, a great deal of knowledge may result without the dangers of categorical reification.

Limitations of the Study

Several factors limit the generalizability of the *IONAS-SR* data. First, the sample consisted entirely of college students, most of whom were freshman and under the age of twenty. Second, structured diagnostic information was not available, so it is unclear to what extent diagnosable psychopathology was present in the sample. Third, whereas non-Caucasians comprised 18% of the sample, small sample sizes of discrete ethnic groups precluded more specific analyses. Fourth, as with all self-report studies, self-ratings of impairment may not be consistent with trained clinician ratings.

Future Directions

With regard to the historical and theoretical component of this work, several areas warrant further attention. First, it would be useful to have a better historical understanding of how the term “neurosis” was actually used and applied in treatment settings. Utilizing 18th and 19th century archives at institutions/hospitals may allow for the discernment of “operational definitions” of this term through cataloguing how it was actually utilized in clinical settings. Second, tracing the various developments of the concept of neurosis within the psychoanalytic literature may lead to novel connections with the mainstream empirical literature and would certainly provide interesting theoretical speculation. This may particularly be the case with regard to the fluctuating quality of negative affectivity, as psychoanalysis has a rich tradition of recognizing symptom substitution. Third, more theoretical work needs to be done assessing potential links between the somatoform disorders and NA. Historically, somatic expressions of

distress were discussed long before Freud and his early and mature works on hysteria, and it is conceivable that this is a neglected area of the NA literature. Certainly, the somatoform disorders themselves are paid scant attention compared to anxious and depressive syndromes.

With regard to the *IONAS-SR*, many research possibilities exist. First and foremost, however, norms from a large clinical sample should be gathered in order to determine which scores and/or items best differentiate between clinical and non-clinical populations. When this task is sufficiently completed, utilizing the *IONAS-SR* as part of a therapy outcome package would be a natural choice. The ability to represent NA at three levels of abstraction may engender a more sophisticated assessment of therapy outcome. Instead of focusing on remission vs. non-remission as a criterion, researchers could isolate which particular symptoms remit, in response to what types of treatment, and could determine when in the course of treatment that this occurs. Knowledge of this kind may lead to the identification of new prognostic indicators and greater understanding of therapy change processes. Related to this, a longitudinal study of an untreated sample of people suffering from negative affect symptoms may also provide insight into how often fluctuations occur. Several possibilities exist. It may be that only certain symptoms are reliably likely to fluctuate, or it may be the case that it is in the nature of NA to be fluid and inconstant over time.

Another future direction would be the expansion of the *IONAS-SR* into a self-contained and semi-structured diagnostic interview. However, unlike other structured interviews such as the *SCID* or the *ADIS*, the *IONAS* interview would result in a

diagnostic profile of the topography of negative affectivity as opposed to a categorical diagnosis of a mood or anxiety disorder.

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Endnotes

¹This label is not meant in a pejorative sense, but in the sense that there is an absence of traditional orders of classification. There may be a number of justifiable reasons to adhere to this belief and practice. However, it may possess sizable dangers (some of which will be described below), and may ultimately engender a feeling of clinical solipsism in which communication with others is hindered.

² It is important to keep in mind that some of the classification differences that will be discussed in this section may no longer hold as much prominence as when they were originally discussed and published. However, regardless of whether or not they represent “cutting edge” thinking in these countries (which unfortunately seems to consist of approximations of the DSM and ICD), relevant findings will be presented for their unique viewpoints and contributions.

³A tension between two incompatible positions exists in this document and bears brief mention. First, there exists a desire to formulate useful solutions to difficulties in the classification of a certain “diagnostic basin” of psychopathology (i.e., the vicissitudes of negative affectivity), and thus, identify a “point of rarity” between this basin and others. Simultaneous with this, however, is the recognition, born out by historical and philosophical research, that not only have all attempts to formulate final immutable solutions failed, but that a miasma of negative consequences can follow such endeavors. So, while the author has presented strong arguments in favor of the proposed system, there is no presumption that this system should either be preemptive or an instance of

“carving nature at its joints”. At most, this work may serve as a particular perspective that clinicians and researchers in psychopathology may find useful for the time being.

⁴The 5-factor solution was chosen over the 4-factor solution due to the fact that the latter appeared to remove items from the former’s first factor and placed them in the third factor, muddying the more unique interpretation of each. With regard to the 6-factor solution, it created an interesting and relatively unitary factor closely mirroring the *a priori* dimensional set of *Somatic Expressions of NA*. However, this solution was not retained for two reasons. First, it removed some of the richness of dimension 1 by collapsing some of its content into factor 3. Second, the 6-factor solution yielded a factor that contained only 5 items, 3 of which strongly correlated with other remaining factors.

Appendix A

Outline of Non-Redundant DSM Symptoms of Mood and Anxiety

| <u>SYMPTOMS</u> | <u>DISORDER OVERLAP</u> |
|---|--------------------------------|
| Clinically-significant distress | MD, PD, AG, SpP, SP, OCD, GAD |
| Recognition of Excessiveness/Unreasonableness | AG, SpP, SP, OCD |
| Depressed Mood | MD |
| Irritable Mood | MD, GAD |
| Anhedonia | MD |
| Weight Fluctuation | MD |
| Sleep Disturbance | MD, GAD |
| Agitation/Retardation/Restlessness | MD, GAD |
| Fatigue/Energy Loss | MD, GAD |
| Guilt/ Worthlessness | MD |
| Decreased Concentration | MD, GAD |
| Death Thoughts | MD |
| Panic Attacks | PD, SpP, SP |
| Concern for Future Sx's | PD, GAD |
| Worry over Implications | PD, GAD, MD |
| Change in Behavior | PD |
| Anx over escape difficulties | AG |
| Avoidance of Situations | AG, SpP, SP |
| Social Anx/Evaluation | SP |
| Generalization of Fear | SP, SpP |
| Obsessions | OCD |
| Suppression of Thoughts | OCD |
| Compulsions | OCD |
| Acts to Prevent Distress | OCD, GAD |
| Uncontrollable Worry | GAD |
| Muscle Tension | GAD |

Key to Abbreviations

MD = Major Depression/Dysthymia
GAD = Generalized Anxiety Disorder
OCD = Obsessive-Compulsive Disorder
SP = Social Phobia
SpP = Specific Phobia
PD = Panic Disorder
AG = Agoraphobia

Appendix B

The *IONAS-SR* and Scoring Instructions***IONAS – SR***

INSTRUCTIONS: Please check the item on the right hand side that best represents the extent to which the items on the left are troubling and/or interfere with your life. For all questions, choose the answer that best represents how these things have affected you in the *past 3 months*. Please answer all items.

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|---|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 1. My appetite has been either too strong or too weak. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. I've felt mentally fatigued. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. I've thought about past failures or bad things that happened in my past. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 4. I've avoided being in crowds. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 5. People watching me do everyday activities (eating, writing) made me anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 6. I've worried. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 7. I've been fearful being outside of my home alone. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 8. I've felt physically fatigued. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 9. I've been anxious around my friends. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 10. I've had problems concentrating or staying focused. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 11. I've had difficulty getting important things done. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 12. I've been anxious starting conversations with people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 13. I've been fearful of certain situations (heights, driving, flying, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|---|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 14. I've felt dissatisfied with my life. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 15. I've flushed or had hot flashes when I was anxious or embarrassed. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 16. I've had repetitive thoughts that I've tried to suppress, ignore, or avoid. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 17. I've felt irritable. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 18. I've slept either too much or too little. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 19. I felt as if I had to count (numbers, letters, or things around me). If I didn't, I would feel anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 20. I've thought more about the past than the present or future. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 21. I've lost interest or pleasure in things that I used to enjoy. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 22. I've cried. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 23. I've been anxious about letting people know "the real me." | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 24. I've felt guilty. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 25. My sex drive/level of interest in sex (libido) has been either too strong or too weak | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 26. I've been critical of myself. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 27. I've had aches and pains. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 28. When I've been upset, it's taken a long time for my thoughts to slow down and get "back to normal." | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 29. I've felt restless, agitated, "keyed up," or on edge. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 30. I've been anxious in social situations that are unclear or ambiguous. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 31. I've been fearful of certain animals (spiders, snakes, etc.) | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 32. I've had sweaty, shaky, or trembling hands. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|---|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 33. I've had breathing difficulties (shortness of breath, feeling pressure in my chest, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 34. I've been afraid of situations in which leaving might be difficult or embarrassing. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 35. My weight has increased or decreased | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 36. I've felt depressed or down. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 37. I've been anxious when trying to stop conversations with people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 38. I've had headaches. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 39. I've felt worthless. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 40. I've avoided letting people get to know "the real me." | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 41. When anxious, I've felt lightheaded, dizzy, or faint. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 42. I felt like I was going crazy. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 43. I felt the need to urinate in anxious situations. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 44. When I've been upset, it's taken a long time for my body to relax and get "back to normal." | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 45. My worrying has been excessive. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 46. I've had problems reaching sexual satisfaction (orgasm). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 47. I've avoided social situations that are unclear or ambiguous. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 48. I've been anxious about public (formal) speaking or performances. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 49. I've spent more time thinking about the future than the past or the present. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 50. I've worried about minor events to keep from thinking about more important concerns. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|---|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 51. I've been preoccupied with "existential" issues (issues of death and mortality, the meaning of life, whether or not there is an afterlife, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 52. I've had difficulty making decisions. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 53. I've avoided asserting myself. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 54. I've thought about choices so much that I end up not taking action. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 55. I've been fearful of blood, injuries, or injections. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 56. I've avoided situations in which leaving might be difficult or embarrassing. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 57. I've been anxious keeping conversations going. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 58. I've avoided speaking or performing in informal situations (telling a story at a party, being the center of attention in a group, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 59. I've had heart (cardiac) difficulties (fast pulse, palpitations, chest pain, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 60. I've had muscle tension. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 61. I've avoided stopping conversations with people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 62. I felt as if I had to make sure that objects are straight, or line up perfectly (symmetrical). If they didn't, I would feel anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 63. I've grinded my teeth at night. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 64. I've felt that I would be better off dead. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 65. I've been fearful that I would embarrass myself in social situations. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 66. I've been fearful that I would offend other people in social situations. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|--|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 67. I've been anxious around my family members. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 68. I've worried about worrying. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 69. I've been anxious around my peers (classmates, co-workers, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 70. I've had gastro-intestinal difficulties (nausea, constipation, diarrhea, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 71. It's been difficult for me to stay "in the present moment." | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 72. I've been anxious around "authority figures" (bosses, professors, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 73. I've avoided certain situations (heights, driving, flying, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 74. I've felt pessimistic. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 75. I've avoided (formal) speaking or performing in public. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 76. I've felt hopeless. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 77. I've been anxious when I've had to assert myself. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 78. It's been difficult to stop worrying once I've started. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 79. I've avoided doing everyday activities (eating, writing a check, etc.) in front of other people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 80. I've worried that I will repeat bad things that have happened to me in the past. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 81. I believed that thinking certain thoughts (sexual, aggressive, etc.) was as bad as actually performing the acts. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 82. I've avoided certain animals (spiders, snakes, etc.) | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 83. I've avoided blood, injuries, or injections, or the sight of them. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Over the past 3 months _____. This has caused distress or impairment in one of more important areas of my life (social, romantic, family, academic, employment, my health, etc.):

| | Rarely or Not at All | Sometimes or Mildly | Often or Moderately | Almost Always or Severely |
|---|-------------------------------|---------------------------|---------------------------|------------------------------------|
| 84. I've been anxious about informally speaking or performing (telling a story at a party, being the center of attention in a group, etc.). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 85. I've been fearful of crowds | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 86. I've felt "stuck" in my life. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 87. Thoughts about past failures entered my mind when I didn't want them to. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 88. I've avoided being outside of my home alone. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 89. I've had "panic attacks" (a sudden rush of intense anxiety or fear with a lot of strange sensations). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 90. I felt like I was going to die. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 91. I've gone out of my way to avoid sex (sexual intercourse). | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 92. I've been bothered by thoughts that keep coming back to me but don't make sense. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 93. I've had thoughts about suicide. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 94. I've avoided starting conversations with people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 95. I felt as if I had to "hoard" (collect) objects. If I didn't, I would feel anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 96. I've avoided keeping conversations going. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 97. I've felt emotionally fatigued. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 98. I felt as if I had to wash my body or a part of my body. If I didn't, I would feel anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 99. I've had problems with my sexual performance. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 100. I felt as if I had to stick with my routine. If I didn't, I would feel anxious. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

IONAS – SR Scoring Information

General Information

| <i>Dimensional Set</i> | | <i>Questions</i> | <i>Total (0-3)</i> |
|------------------------|--|------------------|--------------------|
| I | (Hy) Hygiene/Care of the self | 14 | 42 |
| II | (Mel) Melancholia | 13 | 39 |
| III | (Soma) Somatic Expressions of NA | 13 | 39 |
| IV | (Cog) Cognitive Expressions of NA | 13 | 39 |
| V | (SIT) Social and Interpersonal Expressions | 24 | 72 |
| VI | (FT) Fears and the Experience of Threat | 15 | 45 |
| VII | (OC) Obsessive and Compulsive Expressions | 8 | 24 |
| Total NA | | 100 | 300 |

SCORING PROCEDURE

| | |
|------|--|
| I. | 1, 2, 8, 10, 11, 18, 25, 35, 46, 64, 91, 93, 97, 99 |
| II. | 3, 14, 17, 20, 21, 22, 24, 26, 36, 39, 74, 76, 87 |
| III. | 15, 27, 29, 32, 33, 38, 41, 43, 44, 59, 60, 63, 70 |
| IV. | 6, 28, 45, 49, 50, 51, 52, 54, 68, 71, 78, 80, 86 |
| V. | 5, 9, 12, 23, 30, 37, 40, 47, 48, 53, 57, 58, 61, 65, 66, 67, 69, 72, 75, 77, 79, 84, 94, 96 |
| VI. | 4, 7, 13, 31, 34, 42, 55, 56, 73, 82, 83, 85, 88, 89, 90 |
| VII. | 16, 19, 62, 81, 92, 95, 98, 100 |

Other Possible Supplemental *IONAS-SR* Scores

| | |
|---------------|---|
| Avoidance | 4, 40, 47, 50, 53, 54, 56, 58, 61, 73, 75, 79, 82, 83, 88, 91, 94, 96 |
| Lassitude | 2, 8, 97 |
| NA Reactivity | 28, 44 |
| Suicide Risk | 39, 64, 76, 93 |

Appendix C

IONAS-SR Sets of Symptoms

I. Hygiene/Care of the Self (Hy)

1. My appetite has been either too strong or too weak.
2. I've felt mentally fatigued.
8. I've felt physically fatigued.
10. I've had problems concentrating or staying focused.
11. I've had difficulty getting important things done.
18. I've slept either too much or too little.
25. My sex drive/level of interest in sex (libido) has been either too strong or too weak.
35. My weight has increased or decreased.
46. I've had problems reaching sexual satisfaction (orgasm).
64. I've felt that I would be better off dead.
91. I've gone out of my way to avoid sex (sexual intercourse).
93. I've had thoughts about suicide.
97. I've felt emotionally fatigued.
99. I've had problems with my sexual performance.

II. Melancholia (Mel)

3. I've thought about past failures or bad things that happened in my past.
14. I've felt dissatisfied with my life.
17. I've felt irritable.
20. I've thought more about the past than the present or future.
21. I've lost interest or pleasure in things that I used to enjoy.
22. I've cried.
24. I've felt guilty.
26. I've been critical of myself.
36. I've felt depressed or down.
39. I've felt worthless.
74. I've felt pessimistic.
76. I've felt hopeless.
87. Thoughts about past failures entered my mind when I didn't want them to.

III. Somatic Expressions (Soma)

- 15. I've flushed or had hot flashes when I was anxious or embarrassed.
- 27. I've had aches and pains.
- 29. I've felt restless, agitated, "keyed up," or on edge.
- 32. I've had sweaty, shaky, or trembling hands.
- 33. I've had breathing difficulties (shortness of breath, feeling pressure in my chest, etc.).
- 38. I've had headaches.
- 41. When anxious, I've felt lightheaded, dizzy, or faint.
- 43. I felt the need to urinate in anxious situations.
- 44. When I've been upset, it's taken a long time for my body to relax and get "back to normal."
- 59. I've had heart (cardiac) difficulties (fast pulse, palpitations, chest pain, etc.).
- 60. I've had muscle tension.
- 63. I've grinded my teeth at night.
- 70. I've had gastro-intestinal difficulties (nausea, constipation, diarrhea, etc.).

IV. Cognitive Expressions (Cog)

- 6. I've worried.
- 28. When I've been upset, it's taken a long time for my thoughts to slow down and get "back to normal."
- 45. My worrying has been excessive.
- 49. I've spent more time thinking about the future than the past or the present.
- 50. I've worried about minor events to keep from thinking about more important concerns.
- 51. I've been preoccupied with "existential" issues (issues of death and mortality, the meaning of life, whether or not there is an afterlife, etc.).
- 52. I've had difficulty making decisions.
- 54. I've thought about choices so much that I end up not taking action.
- 68. I've worried about worrying.
- 71. It's been difficult for me to stay "in the present moment."
- 78. It's been difficult to stop worrying once I've started.
- 80. I've worried that I will repeat bad things that have happened to me in the past.
- 86. I've felt "stuck" in my life.

V. Social and Interpersonal Expressions (SIT)

5. People watching me do everyday activities (eating, writing) made me anxious.
9. I've been anxious around my friends.
12. I've been anxious starting conversations with people.
23. I've been anxious about letting people know "the real me."
30. I've been anxious in social situations that are unclear or ambiguous.
37. I've been anxious when trying to stop conversations with people.
40. I've avoided letting people get to know "the real me."
47. I've avoided social situations that are unclear or ambiguous.
48. I've been anxious about public (formal) speaking or performances.
53. I've avoided asserting myself.
57. I've been anxious keeping conversations going.
58. I've avoided speaking or performing in informal situations (telling a story at a party, being the center of attention in a group, etc.).
61. I've avoided stopping conversations with people.
65. I've been fearful that I would embarrass myself in social situations.
66. I've been fearful that I would offend other people in social situations.
67. I've been anxious around my family members.
69. I've been anxious around my peers (classmates, co-workers, etc.).
72. I've been anxious around "authority figures" (bosses, professors, etc.).
75. I've avoided (formal) speaking or performing in public.
77. I've been anxious when I've had to assert myself.
79. I've avoided doing everyday activities (eating, writing a check, etc.) in front of other people.
84. I've been anxious about informally speaking or performing (telling a story at a party, being the center of attention in a group, etc.).
94. I've avoided starting conversations with people.
96. I've avoided keeping conversations going.

VI. *Fears and the Experience of Threat (FT)*

- 4. I've avoided being in crowds.
- 7. I've been fearful being outside of my home alone.
- 13. I've been fearful of certain situations (heights, driving, flying, etc.).
- 31. I've been fearful of certain animals (spiders, snakes, etc.).
- 34. I've been afraid of situations in which leaving might be difficult or embarrassing.
- 42. I felt like I was going crazy.
- 55. I've been fearful of blood, injuries, or injections.
- 56. I've avoided situations in which leaving might be difficult or embarrassing.
- 73. I've avoided certain situations (heights, driving, flying, etc.).
- 82. I've avoided certain animals (spiders, snakes, etc.).
- 83. I've avoided blood, injuries, or injections, or the sight of them.
- 85. I've been fearful of crowds.
- 88. I've avoided being outside of my home alone.
- 89. I've had "panic attacks" (a sudden rush of intense anxiety or fear with a lot of strange sensations).
- 90. I felt like I was going to die.

VII. *Obsessive and Compulsive Expressions (OC)*

- 16. I've had repetitive thoughts that I've tried to suppress, ignore, or avoid.
- 19. I felt as if I had to count (numbers, letters, or things around me). If I didn't, I would feel anxious.
- 62. I felt as if I had to make sure that objects are straight, or line up perfectly (symmetrical). If they didn't, I would feel anxious.
- 81. I believed that thinking certain thoughts (sexual, aggressive, etc.) was as bad as actually performing the acts.
- 92. I've been bothered by thoughts that keep coming back to me but don't make sense.
- 95. I felt as if I had to "hoard" (collect) objects. If I didn't, I would feel anxious.
- 98. I felt as if I had to wash my body or a part of my body. If I didn't, I would feel anxious.
- 100. I felt as if I had to stick with my routine. If I didn't, I would feel anxious.

SUPPLEMENTAL DIMENSIONAL SETS

Avoidance

- 4. I've avoided being in crowds.
- 40. I've avoided letting people get to know "the real me."
- 47. I've avoided social situations that are unclear or ambiguous.
- 50. I've worried about minor events to keep from thinking about more important concerns.
- 53. I've avoided asserting myself.
- 54. I've thought about choices so much that I end up not taking action.
- 56. I've avoided situations in which leaving might be difficult or embarrassing.
- 58. I've avoided speaking or performing in informal situations (telling a story at a party, being the center of attention in a group, etc.).
- 61. I've avoided stopping conversations with people.
- 73. I've avoided certain situations (heights, driving, flying, etc.).
- 75. I've avoided (formal) speaking or performing in public.
- 79. I've avoided doing everyday activities (eating, writing a check, etc.) in front of other people.
- 82. I've avoided certain animals (spiders, snakes, etc.).
- 83. I've avoided blood, injuries, or injections, or the sight of them.
- 88. I've avoided being outside of my home alone.
- 91. I've gone out of my way to avoid sex (sexual intercourse).
- 94. I've avoided starting conversations with people.
- 96. I've avoided keeping conversations going.

NA Reactivity

- 28. When I've been upset, it's taken a long time for my thoughts to slow down and get "back to normal."
- 44. When I've been upset, it's taken a long time for my body to relax and get "back to normal."

Lassitude

- 2. I've felt mentally fatigued.
- 8. I've felt physically fatigued.
- 97. I've felt emotionally fatigued.

Suicide Risks

- 39. I've felt worthless.
- 64. I've felt that I would be better off dead.
- 76. I've felt hopeless.
- 93. I've had thoughts about suicide.

Appendix D

Means, Standard Deviations, and Average Scores for the
IONAS-SR and its Dimensions by Ethnicity

| | | M | SD | N | AS | SD | | | | | | | |
|-------------------|------------------|------|------|-----|-----|-----------------|------------------|------------------|------|------|-----|-----|-----|
| | | M | SD | N | AS | SD | M | SD | N | AS | SD | | |
| Tot al | | 69.9 | 37.0 | 30 | 0.7 | 0.3 | | | | | | | |
| | White | 3 | 3 | 6 | 0 | 7 | Hy | 12.7 | | 30 | 0.9 | 0.4 | |
| | African- Am | 87.7 | 46.4 | | 0.8 | 0.4 | | White | 3 | 6.03 | 5 | 1 | 3 |
| | | 9 | 0 | 14 | 8 | 6 | | African- Am | 15.9 | | | 1.1 | 0.4 |
| | | 94.3 | 47.4 | | 0.9 | 0.4 | | | 3 | 6.88 | 14 | 4 | 9 |
| | Asian/PI | 0 | 4 | 33 | 4 | 7 | | Asian/PI | 14.3 | | | 1.0 | 0.4 |
| | Latino/Hi sp. | 105. | 52.0 | | 1.0 | 0.5 | | Latino/Hi sp. | 9 | 6.88 | 33 | 3 | 9 |
| | Interraci al | 157. | 54.1 | | 1.5 | 0.5 | | Interraci al | 18.2 | | | 1.3 | 0.6 |
| Other | 99.2 | 32.6 | | 0.9 | 0.3 | Other | | 0 | 8.73 | 10 | 0 | 2 | |
| | 0 | 3 | 5 | 9 | 3 | | 22.5 | | | 1.6 | 0.3 | | |
| | | | | | | | 0 | 5.20 | 4 | 1 | 7 | | |
| | | | | | | | 16.6 | | | 1.1 | 0.4 | | |
| | | | | | | | 0 | 6.35 | 5 | 9 | 5 | | |
| Mel | | 11.8 | | 30 | 0.9 | 0.5 | So ma | | | 30 | 0.6 | 0.4 | |
| | White | 7 | 6.92 | 5 | 1 | 3 | | White | 8.19 | 5.44 | 5 | 3 | 2 |
| | African- Am | 15.1 | | | 1.1 | 0.6 | | African- Am | 11.0 | | | 0.8 | 0.7 |
| | | 4 | 8.47 | 14 | 6 | 5 | | | 7 | 9.34 | 14 | 5 | 2 |
| | | 15.4 | | | 1.1 | 0.6 | | Asian/PI | 9.55 | 7.06 | 33 | 3 | 4 |
| | Asian/PI | 8 | 7.99 | 33 | 9 | 1 | | Latino/Hi sp. | 13.8 | | | 1.0 | 0.7 |
| | Latino/Hi sp. | 17.6 | | | 1.3 | 0.6 | | Interraci al | 0 | 9.78 | 10 | 6 | 5 |
| | Interraci al | 29.2 | | | 2.2 | 0.1 | | Other | 14.0 | | | 1.0 | 0.7 |
| | 5 | 2.22 | 4 | 5 | 7 | | 0 | 9.13 | 4 | 8 | 0 | | |
| | 15.4 | | | 1.1 | 0.6 | | 11.0 | | | 0.8 | 0.3 | | |
| | 0 | 7.83 | 5 | 8 | 0 | | 0 | 4.06 | 5 | 5 | 1 | | |
| Co g | | 11.5 | | 30 | 0.8 | 0.5 | SIP | | | 30 | 0.6 | 0.4 | |
| | White | 7 | 6.73 | 5 | 9 | 2 | | White | 15.2 | 11.3 | 5 | 3 | 7 |
| | African- Am | 14.1 | | | 1.0 | 0.6 | | African- Am | 18.5 | 10.2 | | 0.7 | 0.4 |
| | | 4 | 8.21 | 14 | 9 | 3 | | | 0 | 9 | 14 | 7 | 3 |
| | | 15.3 | | | 1.1 | 0.6 | | Asian/PI | 23.6 | 13.6 | | 0.9 | 0.5 |
| | Asian/PI | 0 | 8.73 | 33 | 8 | 7 | | Latino/Hi sp. | 7 | 5 | 33 | 9 | 7 |
| | Latino/Hi sp. | 18.9 | | | 1.4 | 0.7 | | Interraci al | 19.2 | 13.8 | | 0.8 | 0.5 |
| | Interraci al | 28.0 | | | 2.1 | 0.7 | | Other | 0 | 2 | 10 | 0 | 8 |
| | 0 | 9.70 | 4 | 5 | 5 | | 40.7 | 24.1 | | 1.7 | 1.0 | | |
| | 16.8 | | | 1.2 | 0.3 | | 5 | 4 | 4 | 0 | 1 | | |
| | 0 | 4.21 | 5 | 9 | 2 | | 22.8 | 14.7 | | 0.9 | 0.6 | | |
| | | | | | | | 0 | 2 | 5 | 5 | 1 | | |
| FT | | | | 30 | 0.4 | 0.3 | OC | | | 30 | 0.4 | 0.4 | |
| | White | 6.54 | 5.11 | 5 | 4 | 4 | | White | 3.69 | 3.35 | 5 | 6 | 2 |
| | African- Am | | | | 0.5 | 0.4 | | African- Am | 4.36 | 2.98 | 14 | 5 | 7 |
| | | 8.64 | 6.56 | 14 | 8 | 4 | | | | | | 0.6 | 0.5 |
| | | 10.5 | | | 0.7 | 0.5 | | Asian/PI | 5.36 | 4.17 | 33 | 7 | 2 |
| | Asian/PI | 5 | 8.46 | 33 | 0 | 6 | | Latino/Hi sp. | 11.7 | | | 0.7 | 0.4 |
| Latino/Hi sp. | 11.7 | | | 0.7 | 0.5 | Interraci al | 0 | 8.71 | 10 | 8 | 8 | | |
| Interraci al | 13.0 | 7.87 | 4 | 0.8 | 0.5 | | 10.2 | 4.99 | 4 | 1.2 | 0.6 | | |

| | | | | | | | | | | | |
|-------|------|------|---|-----|-----|-------|------|------|---|-----|-----|
| al | 0 | | | 7 | 2 | al | 5 | | | 8 | 2 |
| | 10.4 | | | 0.6 | 0.2 | | | | | 0.7 | 0.4 |
| Other | 0 | 2.97 | 5 | 9 | 0 | Other | 6.20 | 3.57 | 5 | 8 | 5 |

Notes: AS = Average Score, Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, and OC = Obsessive and Compulsive Expressions.

Appendix E

IONAS-SR Test-Retest Means, Standard Deviations, and Mean Change Scores by Gender

| Group | | r | Test M | Test SD | Retest M | Retest SD | MC |
|--------------|---|-----|--------|------------------------|----------|-----------|-------|
| Total | M | .86 | 68.85 | 38.49 | 51.35 | 36.38 | 16.08 |
| | F | .89 | 78.10 | 41.64 | 56.26 | 35.33 | 15.89 |
| | | | | t(36) = 4.87, p < .001 | | | |
| | | | | t(37) = 6.14, p < .001 | | | |
| Hy | M | .8 | 12.27 | 6.06 | 8.73 | 4.70 | 3.19 |
| | F | .83 | 13.85 | 6.44 | 10.66 | 6.07 | 2.45 |
| | | | | t(36) = 6.12, p < .001 | | | |
| | | | | t(37) = 4.28, p < .001 | | | |
| Mel | M | .9 | 11.61 | 6.92 | 9.81 | 8.17 | 1.27 |
| | F | .87 | 13.27 | 7.57 | 10.92 | 7.45 | 2.18 |
| | | | | t(36) = 2.18, p < .05 | | | |
| | | | | t(37) = 3.69, p < .002 | | | |
| Soma | M | .61 | 7.22 | 5.32 | 4.78 | 4.83 | 1.78 |
| | F | .82 | 9.37 | 6.25 | 5.55 | 4.41 | 1.79 |
| | | | | t(36) = 2.44, p < .03 | | | |
| | | | | t(37) = 4.07, p < .001 | | | |
| Cog | M | .83 | 10.97 | 7.00 | 9.14 | 7.57 | 2.11 |
| | F | .83 | 13.19 | 7.51 | 9.66 | 7.09 | 2.32 |
| | | | | t(36) = 2.89, p < .01 | | | |
| | | | | t(37) = 3.56, p < .002 | | | |
| SIP | M | .88 | 16.79 | 11.64 | 11.95 | 10.77 | 4.92 |

| | | | | | | | |
|---|---|-----|-------|------------------------|-------|------|------|
| | F | .88 | 16.53 | 12.60 | 11.82 | 9.66 | 4.71 |
| | | | | t(36) = 5.42, p < .001 | | | |
| | | | | t(37) = 5.94, p < .001 | | | |
| FT | M | .69 | 5.98 | 4.90 | 4.05 | 3.78 | 1.86 |
| | F | .83 | 7.83 | 6.18 | 5.29 | 4.30 | 1.24 |
| | | | | t(36) = 3.06, p < .005 | | | |
| | | | | t(37) = 2.74, p < .01 | | | |
| OC | M | .70 | 4.01 | 3.46 | 2.95 | 2.70 | 0.89 |
| | F | .50 | 4.06 | 3.66 | 2.37 | 2.60 | 1.24 |
| | | | | t(36) = 2.30, p < .05 | | | |
| | | | | t(37) = 2.81, p < .01 | | | |
| <p><i>Notes: MC = Mean Change, Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, OC = Obsessive and Compulsive Expressions.</i></p> <p><i>All correlations significant with p's < .001</i></p> | | | | | | | |

Appendix F

Criterion Validity of The IONAS-SR and Dimensional Sets by Gender

| | | PANAS- | | | | | | | | | |
|--------------|---|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| | | BDI | | MAQ | | NA | | PANAS_PA | | STAI | |
| | | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> |
| Total | M | .73** | 116 | .70** | 117 | .63** | 117 | -.10 | 117 | .74** | 117 |
| | F | .75** | 246 | .72** | 252 | .72** | 252 | -.30** | 252 | .79** | 252 |
| Hy | M | .71** | 116 | .65** | 117 | .52** | 117 | -.09 | 117 | .60** | 117 |
| | F | .65** | 246 | .64** | 252 | .61** | 252 | -.27** | 252 | .65** | 252 |
| Mel | M | .72** | 116 | .73** | 117 | .70** | 117 | -.13 | 117 | .77** | 117 |
| | F | .77** | 246 | .75** | 252 | .73** | 252 | -.41** | 252 | .83** | 252 |
| Soma | M | .62** | 116 | .52** | 117 | .39** | 117 | -.00 | 117 | .42** | 117 |
| | F | .61** | 246 | .64** | 252 | .57** | 252 | -.14* | 252 | .60** | 252 |
| Cog | M | .64** | 116 | .67** | 117 | .58** | 117 | -.12 | 117 | .68** | 117 |
| | F | .71** | 246 | .75** | 252 | .72** | 252 | -.31** | 252 | .79** | 252 |
| SIP | M | .54** | 116 | .53** | 117 | .51** | 117 | -.13 | 117 | .67** | 117 |
| | F | .57** | 246 | .46** | 252 | .52** | 252 | -.26** | 252 | .62** | 252 |
| FT | M | .59** | 116 | .54** | 117 | .52** | 117 | -.00 | 117 | .57** | 117 |
| | F | .49** | 246 | .45** | 252 | .51** | 252 | -.11 | 252 | .49** | 252 |
| OC | M | .59** | 116 | .53** | 117 | .47** | 117 | -.06 | 117 | .54** | 117 |
| | F | .58** | 246 | .56** | 252 | .55** | 252 | -.13* | 252 | .57** | 252 |

Notes: Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, and OC = Obsessive and Compulsive Expressions.

** Correlation is significant at .001 level

* Correlation is significant at .05 level

Appendix G

Discriminant Validity of the *IONAS-SR* and Dimensional Sets by Gender

| | | AQ | | AQ-Pag | | AQ-VAg | | AQ-Ang | | AQ-Hos | |
|--------------|---|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| | | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> | <i>r</i> | <i>n</i> |
| Total | M | .39** | 117 | .17** | 117 | .18 | 117 | .29** | 117 | .59** | 117 |
| | F | .39** | 252 | .27** | 252 | .04 | 252 | .29** | 252 | .54** | 252 |
| Hy | M | .34** | 117 | .23** | 117 | .13 | 117 | .23* | 117 | .44** | 117 |
| | F | .38** | 252 | .30** | 252 | .12* | 252 | .28** | 252 | .45** | 252 |
| Mel | M | .47** | 117 | .21** | 117 | .27* | 117 | .41** | 117 | .61** | 117 |
| | F | .41** | 252 | .30** | 252 | .07 | 252 | .30** | 252 | .55** | 252 |
| Soma | M | .30* | 117 | .16** | 117 | .19* | 117 | .23* | 117 | .37** | 117 |
| | F | .36** | 252 | .30** | 252 | .11 | 252 | .27* | 252 | .41** | 252 |
| Cog | M | .36** | 117 | .11** | 117 | .18* | 117 | .29** | 117 | .57** | 117 |
| | F | .38** | 252 | .25** | 252 | .06 | 252 | .32** | 252 | .51** | 252 |
| SIP | M | .24** | 117 | .04** | 117 | .02 | 117 | .17 | 117 | .50** | 117 |
| | F | .25** | 252 | .13* | 252 | -.07 | 252 | .17** | 252 | .46** | 252 |
| FT | M | .32** | 117 | .14** | 117 | .16 | 117 | .24** | 117 | .48** | 117 |
| | F | .26** | 252 | .21** | 252 | .00 | 252 | .23 | 252 | .33** | 252 |
| OC | M | .35** | 117 | .21** | 117 | .25** | 117 | .20* | 117 | .46** | 117 |
| | F | .20** | 252 | .15* | 252 | -.03 | 252 | .13* | 252 | .33** | 252 |

Notes: Hy = Hygiene/Care of the Self, Mel = Melancholia, Soma = Somatic Expressions, Cog = Cognitive Expressions, SIP = Social and Interpersonal Expressions, FT = Fears and the Experience of Threat, and OC = Obsessive and Compulsive Expressions, AQ-PAg = Physical Aggression, AQ-Vag = Verbal Aggression, AQ-Ang = Anger, and AQ-Hos = Hostility.

*** Correlation is significant at .001 level*

** Correlation is significant at .05 level*

Appendix H

Rotated Factor Components of the *IONAS-SR* 5-Factor Solution

| <i>IONAS-SR</i> Item | Component | | | | |
|---|-----------|-----|-----|-----|-----|
| | 1 | 2 | 3 | 4 | 5 |
| 1 "My appetite has been either too strong or too weak" | .37 | | | .41 | |
| 2 "I've felt mentally fatigued" | .64 | | | | |
| 3 "I've thought about past failures or bad things that happened in my past" | .47 | | | | |
| 4 "I've avoided being in crowds" | | .49 | | | |
| 5 "People watching me do everyday activities makes me anxious" | | .45 | | | |
| 6 "I've worried" | .62 | | | | |
| 7 "I've been fearful of being outside of my home alone" | | | | | .42 |
| 8 "I've felt physically fatigued" | .48 | | | | |
| 9 "I've been anxious around my friends" | | .41 | | | |
| 10 "I've had problems concentrating or staying focused" | .49 | | | | |
| 11 "I've had difficulty getting important things done" | .45 | | | | |
| 12 "I've been anxious starting conversations with people" | | .68 | | | |
| 13 "I've been fearful of certain situations" | | | | | .42 |
| 14 "I've felt dissatisfied with my life" | .41 | | .53 | | |
| 15 "I've flushed or had hot flashes when I was anxious or embarrassed" | .38 | | | | |
| 16 "I've had repetitive thoughts that I've tried to suppress, ignore, or avoid" | .54 | | | | |
| 17 "I've felt irritable" | .54 | | | | |
| 18 "I've slept either too much or too little" | .53 | | | | |
| 20 "I've thought more about the past than the present or future" | .38 | | | | |
| 21 "I've lost interest or pleasure in things that I used to enjoy" | .37 | | | .36 | |
| 22 "I've cried" | .54 | | | | |
| 23 "I've been anxious about letting people know the real me" | | .47 | | | |
| 24 "I've felt guilty" | .38 | | | | |
| 25 "My sex drive/level of interest in sex has been either too strong or too weak" | | | | .50 | |
| 26 "I've been critical of myself" | .54 | | | | |
| 27 "I've had aches and pains" | .53 | | | | |
| 28 "When I've been upset, it's taken a long time for my thoughts to slow down and get back to normal" | .68 | | | | |
| 29 "I've felt restless, agitated, keyed up, or on edge" | .62 | | | | |
| 30 "I've been anxious in social situations that are unclear or ambiguous" | | .62 | | | |
| 31 "I've been fearful of certain animals" | | | | | .59 |
| 33 "I've had breathing difficulties" | | | | .50 | |

| | | | | |
|----|---|-----|-----|-----|
| 34 | "I've been afraid of situations in which leaving might be difficult or embarrassing: | .50 | | |
| 36 | "I've felt depressed or down" | .60 | .46 | |
| 37 | "I've been anxious when trying to stop conversations with people" | .53 | | .36 |
| 38 | "I've had headaches" | .48 | | |
| 39 | "I've felt worthless" | .43 | .64 | |
| 40 | "I've avoided letting people get to know the real me" | .38 | .42 | |
| 41 | "When anxious, I've felt light headed, dizzy, or faint" | .44 | | .38 |
| 42 | "I felt like I was going crazy" | .56 | | |
| 44 | "When I've been upset, it's taken a long time for my body to relax and get back to normal" | .65 | | |
| 45 | "My worry has been excessive" | .69 | | |
| 46 | "I've had problems reaching sexual satisfaction" | | | .49 |
| 47 | "I've avoided social situations that are unclear or ambiguous" | .63 | | |
| 48 | "I've been anxious about public speaking or performances" | .59 | | |
| 50 | "I've worried about minor events to keep from thinking about more important concerns" | .39 | | |
| 52 | "I've had difficulty making decisions" | .36 | .40 | |
| 53 | "I've avoided asserting myself" | | .53 | |
| 54 | "I've thought about choices so much that I end up not taking action" | .38 | | |
| 55 | "I've been fearful of blood, injuries, or injections" | | | .68 |
| 56 | "I've avoided situations in which leaving might be difficult or embarrassing" | .60 | | |
| 57 | "I've been anxious keeping conversation going" | .69 | | |
| 58 | "I've avoided speaking or performing in informal situations" | .76 | | |
| 59 | "I've had heart difficulties" | | | .49 |
| 60 | "I've had muscle tension" | .50 | | .39 |
| 61 | "I've avoided stopping conversation with people" | .44 | | |
| 62 | "I felt as if I had to make sure that objects are straight, or line up perfectly. If they didn't, I would feel anxious" | | | .35 |
| 64 | "I've felt that I would be better off dead" | | .77 | |
| 65 | "I've been fearful that I would embarrass myself in social situations" | .70 | | |
| 66 | "I've been fearful that I would offend other people in social situations" | .55 | | |
| 67 | "I've been anxious around my family members" | | | .41 |
| 68 | "I've worried about worrying" | .50 | | |
| 69 | "I've been anxious around my peers" | .37 | .56 | |
| 71 | "It's been difficult for me to stay in the present moment" | .40 | | |
| 72 | "I've been anxious around authority figures" | .53 | | |
| 73 | "I've avoided certain situations" | | | .54 |

| | | | | |
|----|--|-----|-----|-----|
| 74 | "I've felt pessimistic" | .40 | .37 | |
| 75 | "I've avoided speaking or performing in public" | | .71 | |
| 76 | "I've felt hopeless" | .49 | .55 | |
| 77 | "I've been anxious when I've had to assert myself" | | .62 | |
| 78 | "It's been difficult to start worrying once I've started" | .64 | | |
| 79 | "I've avoided doing everyday activities in front of other people" | | .37 | |
| 80 | "I've worried that I will repeat bad things that have happened to me in the past" | .48 | | |
| 82 | "I've avoided certain animals" | | | .68 |
| 83 | "I've avoided blood, injuries, injections, or the sight of them" | | | .75 |
| 84 | "I've been anxious about informally speaking or performing" | | .78 | |
| 85 | "I've been fearful of crowds" | | .67 | |
| 86 | "I've felt stuck in my life" | .50 | .45 | |
| 87 | "Thoughts about past failures entered my mind when I didn't want them to" | .47 | | |
| 88 | "I've avoided being outside of my home alone" | | | .42 |
| 89 | "I've had panic attacks" | .46 | | |
| 90 | "I felt like I was going to die" | | .61 | |
| 91 | "I've gone out of my way to avoid sex" | | | .39 |
| 92 | "I've been bothered by thoughts that keep coming back to me but don't make sense" | .35 | | |
| 93 | "I've had thoughts about suicide" | | .73 | |
| 94 | "I've avoided starting conversations with people" | | .66 | |
| 95 | "I've felt as if I had to hoard objects. If I didn't, I would feel anxious" | | | .44 |
| 96 | "I've avoided keeping conversations going" | | .62 | |
| 97 | "I've felt emotionally fatigued" | .72 | | |
| 98 | "I felt as if I had to wash my body or a part of my body. If I didn't, I would feel anxious" | | | .48 |
| 99 | "I've had problems with my sexual performance" | | | .51 |

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