Linear common-factor analysis in mental disorder validation: Problems and alternatives.

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UMI
LINEAR COMMON-FACTOR ANALYSIS IN MENTAL DISORDER VALIDATION:

PROBLEMS AND ALTERNATIVES

by

Jarkko Jalava

A Thesis
Submitted to the College of Graduate Studies and Research
through the Department of Psychology
in Partial Fulfillment of the Requirements for
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Abstract

This paper provides a critical methodological analysis of current validation and research methods for mental disorders. The purpose of this paper is threefold: First, we will examine the construct validation program and the use of factor analysis in psychiatric validation studies. Second, we will point out some limitations to linear common-factor analysis in this context. This will be done through hypothetical scenarios of mental disorders which do not conform to the linear common-factor model. Third, we will briefly provide some suggestions for dealing with these limitations. These suggestions are intended to alert the researcher to alternative statistical models, such as the unfolding model and the non-linear factor model, to be used in the service of mental disorder classification and validation.
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CHAPTER I

INTRODUCTION

Overview

The enterprise of classifying and validating mental disorders is marked by conceptual and empirical problems. While some of these problems appear inherent to the general practice of classification, some are unique to the subject matter of psychiatry and clinical psychology. The unique problems include, but are not limited to, general uncertainty regarding the causality of psychiatric disorders and the difficulty of choosing the appropriate level or levels of classificatory analysis (i.e., affective, behavioral, cognitive, interpersonal, social, or biological) for each disorder. These and other challenges to psychiatric classification have proven to be of major consequence, resulting in relatively low estimations of reliability and validity for mental disorder categories, and devaluation of the status of psychiatry and psychology as truly scientific activities (e.g., Maxmen & Ward, 1995; Robins & Guze, 1970). In response to the challenges set by the evasive concept of mental health, the field has adopted a set of procedures and assumptions designed to promote the scientific rigor of psychiatric classification. To this effect, a number of statistical methods have been introduced with the assumption that at least some psychiatric conditions are amenable to being validated using statistical analyses of data from patient and control groups.

One widely used statistical method in the validation of mental disorders is factor
analysis. The aim of factor analysis is to reveal $n$ number of correlated groups of clinical features which may be used to indicate the presence of an underlying dimension or dimensions (Blashfield, 1984). Using a metaphor, Cattell (1952, p. 16) describes the premise of factor analysis as follows:

we are like the crew of a ship approaching some strange coast through a fog. It is easy to seize on some arbitrary, transient point of visibility and still easier to convince ourselves that it proves the existence of structures created by our own imaginations on the basis of pretentious hypotheses.

Factor analysis, however, comes to our rescue as a kind of radar to avoid both the trivial and the unreal, for it gives us - however roughly at first - the shape of the real structures hidden in the swirling multiplicity of variables.

When applied to the realm of clinical psychology and psychiatry, the “shape of the real structures hidden in the swirling multiplicity of variables” is understood as underlying dimensions which are related to a matrix of observed, inter-correlated psychiatric symptoms. Sets of observed symptoms resulting from this approach are termed syndromes (e.g. Meehl, 1992).

The present paper is a critical methodological analysis of the use of linear common-factor analysis in mental disorder validation (we will only make general references to the larger issue of classification). Validation, in this paper, is taken to refer to the process of forging a link between a set of observed symptoms and an underlying, true, existing natural process that gives rise to the observed symptoms. The purpose of
this paper is to examine the logical and statistical contours of linear common-factor analysis, and to point out some of its potential limitations in validation studies. This will be accomplished by exploring hypothetical mental disorder scenarios to which models other than the linear common-factor model apply, and which would be misunderstood on the basis of factor analytic results. Through these scenarios we hope to alert the researcher to alternative statistical models to be used in psychiatric validation studies.

The present paper will be divided into two main sections. The first section examines the logic and the use of linear common-factor analysis in psychiatric validation studies. The points made in this section will be exemplified by a DSM-IV category of schizotypal personality disorder (SPD). The second section is critical, pointing out potential sources of error arising from the use of the linear common-factor model. These points will be illustrated by three hypothetical mental disorder scenarios for which the linear common-factor analysis is an inappropriate validation tool. The critiques presented here are purely statistical, and do not involve empirical data.

**Medical and psychiatric classification**

In organic medicine, the process of disease classification proceeds through stages. Initially, diseases may only be defined by description. That is, patients with a similar pattern of symptoms and signs can be said to be suffering from the same disease. This pattern is termed a “syndrome.” When a syndrome is found to be associated constantly with a recognizable anatomical or functional change within the body, it is commonly redefined, and often renamed in terms of the abnormal anatomical or functional unit. That is, the syndrome gives way to the disease entity (of which it is a
manifestation) as the focus of classification. Finally, when the cause of the disease is discovered, the disease is commonly redefined in causal terms. For instance, the term “influenza” refers exclusively to a disease caused by an influenza virus and is to be distinguished from short-term respiratory illnesses with similar manifest symptoms but with different causes. Thus, the general direction of medical classification is towards causation (Scadding, 1988).

In comparison to disease classification in organic medicine, classification of mental disorders is known to be a particularly difficult task. In medicine, as already seen, disease classification is typically a progression from syndromal description to causal definition. Few organic medical disorders in fact remain in the first stage of classification (one example of a syndromally defined medical disorder is migraine) (Scadding, 1988). In the case of mental disorders, on the other hand, structural and functional correlates as well as causes are notably undiscovered. From a causal point view, different schools of psychological and psychiatric thought emphasize different causal mechanisms. From the point of view of disease entities, very little solid medical evidence exists to suggest structural and functional abnormalities associated with mental disorders. Consequently, psychologists and psychiatrists are left with the task of describing and classifying psychiatric symptoms without the help of causal, structural, or functional data. The detection, validation, and hence classification, of psychiatric disease entities must proceed solely via observable symptoms.
Construct validation

Given the above limitations to psychiatric classification, the psychiatric and psychological communities have adopted a set of procedures to extract maximally valid classificatory information from the observable symptoms. To this effect, the method by which psychiatric disorders are commonly validated is through a measurement theory known as construct validation (Cronbach & Meehl, 1955). According to Cronbach and Meehl (1955, p. 290), the aim of construct validation is to reveal "an interlocking system of laws" that relate "observable properties or quantities to each other, theoretical constructs to observables, or different theoretical constructs to one another." In the context of psychiatric classification, "observable properties" are understood as symptoms while "theoretical constructs" are understood as the dimensions underlying them. The alluded to "system of laws," called a nomological network, thus establishes links within and between the observed and unobserved elements. The end result of such a network is to justify explanations of people's behaviors by reference to the constructs that give rise to them (Norris, 1983). As Cronbach (1971, p. 477) states: "a description that refers to a person's internal processes (anxiety, insight) invariably requires construct validation."

For instance, in the construct validation of a clinical scale, the conclusion might be that an underlying mental disorder is responsible for a given profile of scores.

Of special importance in considering construct validation is the assumption of empirical and conceptual similarities. Within the construct validation framework, the degree of empirical association between observed variables is considered to reflect the degree to which the variables have similar conceptual or theoretical meaning. That is,
the more closely two or more variables are associated empirically, the more closely they are considered to be related conceptually or theoretically. Conversely, the closer in meaning one variable, test, or symptom cluster is to another variable, test, symptom cluster etc., the more they are expected to converge empirically. Convergent and discriminant validity of constructs is thus established through empirical associations with other constructs, which, if conceptually or theoretically similar, should correlate highly with the construct, and if conceptually or theoretically dissimilar, should correlate poorly with the construct. These assumptions gain justification from a further assumption that highly correlated observed variables are measurements or indicators of a common "latent" construct (Cronbach & Meehl, 1955).

Research on schizophrenia spectrum disorders provides a good example of the process of construct validation. Here, extensive discriminant and convergent validity studies have been conducted in order to determine the structure of the underlying schizophrenia spectrum (e.g., Kostaftis & Neale, 1993). It is believed that several disease processes, rather than one, are in fact responsible for schizophrenic and schizophrenia-like symptoms. Construct validation has been used to examine this assumption by forging links between manifest symptoms and underlying processes. This line of investigation has resulted in the construction of several schizophrenia-related disorders, such as schizotypal personality disorder and borderline personality (e.g., Kostaftis & Neale, 1993). The construction and validation of schizotypal personality disorder will be examined later in this paper.
Factor analysis

The establishment of syndromes and construct validation follows relatively strict statistical criteria. Most commonly, such criteria are provided by the method of factor analysis. The following is a brief discussion on the logic and history of factor analysis. Our discussion of factor analysis will be based on the linear common-factor model, since a review of psychiatric and psychological literature shows that it is by far the most commonly used brand of factor analysis in the service of classification and validation of mental disorders.

The method of factor analysis came into being specifically to provide mathematical models for the purpose of explaining psychological theories of human ability and behavior (Blashfield, 1984). Initially, the method was developed for the study of mental abilities. During the early twentieth century, Charles Spearman noted that scores on all ability tests were positively correlated, i.e. a person's good performance on one test increases the probability that he or she will perform well on other tests of mental ability as well. Thus, Spearman postulated a "general factor" \( (g) \) that would explain these correlations. Factor analysis was developed by Spearman (1933) as a test of the hypothesis that such a factor existed and as a tool to provide a definition for \( g \). Since then, factor analysis has been used for a variety of purposes, such as that of classifying diseases by reducing a number of observed symptoms into sets (syndromes).

---

1 Review is based on research conducted by author on psychological and psychiatric literature dealing with classification and validation of mental disorders in the time period of 1991 to present. The research was based on PsycHlit search for keyword "factor analysis." All matches found used the common-factor model.
In this way, symptoms that are correlated with one another, but largely independent of other subsets, are combined into sets, or factors (Blashfield, 1984; Harman, 1967). In a similar manner to the concept of g, factor analysis is then often used to test the hypothesis that a latent component giving rise to the observed symptom correlations exists. As Meehl (1986, p. 221) puts it: "Covariation is the essence of descriptive science and the touchstone of scientific thinking."

Mathematically, the one-dimensional common factor analysis model for n observed variables, \( Y \), is expressed as follows:

\[
Y = \Lambda X + \delta
\]

with \( X \) representing the latent common factor to \( Y \), \( \delta \) the vector of unique factors, or residuals, and \( \Lambda \) the factor loadings. Each observed variable is portrayed as a linear combination of a latent factor common to all of the variables, and a unique factor not shared by the other variables. In addition, common factors are assumed to have zero correlation with the unique factors, and unique factors are assumed to be mutually uncorrelated. The joint consequence is that:

\[
\Sigma = \Lambda \Lambda' + \Psi^2
\]

i.e., the covariance matrix may be decomposed into common factor variance, as represented by outer \( \Lambda \), and unique factor variance as represented by \( \Psi^2 \).

Notable in this description is the assumption of linearity. This assumption is integral for the theory, and has rarely been addressed as a serious constraint (see McDonald, 1962 or 1967a). The linear model is commonly assumed in part because of the desire of scientists to explain phenomena by most parsimonious theories, and in part
because of the apparent complexity commonly associated with the consideration of non-linear models (Harman, 1967).

The "meaning" of the factor (i.e., the nature of the underlying disease entity) is interpreted according to the factor loadings of the variables. This process is called factor interpretation. Typically, those variables with a large loading (for example, in excess of .30) provide the basis for this interpretation (e.g., Tabachnick & Fidell, 1996).

The common heuristic assumption behind the use of factor analysis is either that the latent common factor is an indication of an underlying causal mechanism, or that the variables measure the same trait. Consequently, the common factor \( X \) is taken to serve as an indicator of a characteristic of the subjects (from whom the variable was measured), or a common causal entity, and the unique factor \( \delta \) as an index of what is idiosyncratic to the subject (e.g., McDonald, 1981; Meehl, 1992). Metaphorically, then, the common factor can be seen as a "signal" to be picked up in the face of residual noise with the magnitude of the latter given by \( \psi^2 \).

Through its ability to establish sets of intercorrelations between constructs, factor analysis is an obvious candidate for use in construct validation. With observed symptoms providing the input for factor analysis, the resulting factor solutions are then used as indications that measurements were taken of the same trait or traits (e.g., Blashfield, 1984; Cattell, 1952; Tabachnick & Fidell, 1996; Thurstone, 1949).

Appendix A provides a brief discussion of the philosophical framework by which factor analysis operates in the service of mental disorder validation.
Exemplifying classification: Schizotypal personality disorder

Literature on classification of mental disorders typically describes two distinct taxonomic approaches. One approach is exemplified by the Diagnostic and Statistical Manual of Mental Disorders (DSM) (American Psychiatric Association, 1994) and the World Health Organization’s International Classification of Diseases (ICD) (1992). The DSM and ICD categories have been developed largely through negotiations among panels of experts who select target disorders and the criteria for defining them. These categories are then modified through various field trials (Achenbach & McConaughy, 1997). Generally, these categories undergo numerous validation studies via various multivariate statistical procedures, such as factor analysis.

As opposed to the DSM and ICD schemes, the empirically based approach begins with data on numerous clinical items. Each item is scored on a quantitative scale, and the items are then subjected to multivariate analyses such as factor analysis to identify sets of clinical features that tend to occur together. While empirically derived syndromes do not necessarily reflect diagnostic categories, such as the DSM and ICD varieties, the empirical syndromes provide a starting point for assessment tools, intervention strategies, and outcome evaluations (Achenbach & McConaughy, 1997), and may be used to inform future classification (Frick, Lahey, Loeber, Tannenbaum, Van Horn, Christ, Hart, & Hanson, 1993).

Clearly, the empirically based approach exemplifies the construct validation program as explicated in the present paper more closely than the DSM/ICD approach. Clinical scale construction, for instance, generally follows the empirically based
approach guidelines very closely. Here, the empirical approach is not used directly in the classification of mental disorders, but rather in assessing their prevalence in various populations and in identifying individual cases. In this way, the empirical approach is used, not instead of, but in conjunction with the official classification schemes (Achenbach & McConaughy, 1997).

While the empirical and the DSM/ICD approaches are typically distinguished from each other in principle, the actual process of psychiatric classification relies on both approaches. That is, the end result of classification relies on both expert judgement and multivariate analyses.

As already noted, the empirical approach is used to guide the final DSM/ICD-style classification. This reflects the belief that if mental disorders indeed exist, the two approaches will converge on diagnostic categories. If spectra of mental disorders exist, they will reveal themselves in multivariate analyses particularly as syndromes (e.g., Meehl, 1992). The empirical approach is, therefore, used to validate the DSM and other diagnostic categories. To put it differently, whereas the initial observation of co-occurring symptoms is based on expert judgement, the validity of this observation is evaluated by multivariate analyses, such as factor analysis. In this way, for the most accurate classificatory scheme, there is assumed to be a convergence between a clinical, expert discovery, and a discovery of the statistical kind.

What follows is an example of a psychiatric classification process that illustrates the ideas just presented. The example used here is schizotypal personality disorder (SPD).
The DSM-IV classification of personality disorders provides a good illustration of
the points made above. One quite extensively researched personality disorder is
schizotypal personality disorder (SPD). SPD involves problems in three major areas:
thought, affect, and interpersonal relationships. Thought disturbances include
suspiciousness, odd beliefs, ideas of reference, paranoid ideation, and magical thinking.
Problems in affect include social anxiety, as well as constricted and inappropriate affect.
Schizotypals usually have no close friends or companions aside from first degree
relatives (American Psychiatric Association, 1994). In the context of other DSM-IV
disorders, SPD is reminiscent of an attenuated form of schizophrenia. The inclusion of
SPD in the DSM classification system reflects the thinking that schizophrenia is a group
of disorders with a common genetic origin, but with different phenomenological
manifestations that lie on a continuum of symptom intensity. At the extreme end of this
continuum lies schizophrenia, while schizotypal personality disorder can be located at a
less extreme point (the other end of the continuum being occupied by isolated symptoms)

The classification and validation of SPD as a mental disorder exemplifies the
program of construct validation. SPD is a development from the construct of
schizophrenia. Kraepelin (1919/1971) was the first to note that the relatives of
schizophrenics manifested abnormal patterns of personality, which he called "latent
schizophrenia." These symptoms were further studied by Bleuler (1911/1950), who
established their distribution in the general population. The term "schizotypy" was
coined by Rado (1953) as an abbreviation for "schizophrenic genotype," denoting the
hereditary disposition to schizophrenia. Finally, Meehl (1962) expanded on the notion of schizotypy by defining it as a personality type (i.e. as a cluster of personality traits), and by suggesting a genetic formulation for its transmission (e.g. Kendler, 1985; Kostafitis & Neale, 1993; Torgersen, 1985).

The current nomenclature of schizotypal personality disorder is a result of the work of Spitzer, Endicott, and Gibbon (1979). Previous to Spitzer et al. (1979), the DSM-II (American Psychiatric Association, 1968) contained a category of “borderline schizophrenia,” indicating a condition between neurosis and psychosis, or in other words, a milder form of schizophrenia. However, reviewing the literature on borderline schizophrenia, Perry and Klerman (1978), suggested that the condition might in fact be composed of two or more subtypes. To investigate this claim further, Spitzer et al. (1979) reviewed data of the Danish adoption studies (Kety, Rosenthal, Wender, & Schulsinger, 1968), a large scale interview study of biological relatives of schizophrenic adoptees. Many of the relatives of schizophrenics manifested a cluster of symptoms similar to, but less severe than schizophrenics. This syndrome was termed “borderline schizophrenia.” On the basis of this review, Spitzer et al. (1979) defined two separate personality disorders. These disorders were “schizotypal personality” and “unstable personality,” the latter of which later evolved into “borderline personality disorder” (Kostafitis & Neale, 1993). The symptoms of schizotypal personality were then identified sequentially. First, Spitzer et al. (1979) asked for a list of behaviors believed to be key indicators of borderline schizophrenia from the Danish adoption studies (Kety et al., 1968). Second, they reviewed the indicators and reduced the number of symptoms by
eliminating rare behaviors and combining behaviors that seemed to denote the same clinical concept.

The syndromes of schizotypal personality and unstable personality were then cross-validated with a sample from 4000 members of the American Psychiatric Association who were asked to rate two of their own patients using the two sets of symptoms given in a 22 item questionnaire. The first of these patients was to have been diagnosed by the clinician as borderline personality, borderline personality organization, or borderline schizophrenia, and the second (control group) was to be a moderately to severely disturbed patient, but not with a diagnosis of psychosis or any of the above borderline categories.

Spitzer et al. (1979) received 808 sets of usable questionnaires. These questionnaires were subjected to two factor analyses, the first of which involved the borderline patients only, and the second one, both the borderline and control patients. Both analyses resulted in two factors, with schizotypal items loading highest on one factor, and unstable items loading on the other. The final item sets selected for schizotypal personality were thus established (Spitzer et al., 1979).

diagnosis of SPD requires at least five of nine symptoms listed in the DSM-IV, thus exemplifying a polythetic system of classification.

Since the establishment of the DSM criteria for SPD, a number of investigators have attempted to further validate the disorder through factor analytic studies. A quick review of PSYCH-LIT from 1991 onwards alone revealed more than 20 such published studies. Some of these studies will be discussed shortly.

From the above description it becomes clear that the construction and validation of SPD exemplifies the program of construct validation. The initial pool of symptoms observed by Kraepelin (1919/1971) was originally conceived as a naturally occurring cluster, albeit without statistical proof such as was later to be provided by factor analysis. The resulting symptoms were then refined by Spitzer et al. (1979), at which point rare symptoms were eliminated. The validation of the new syndrome of schizotypal personality was finally accomplished by two factor analyses (Spitzer et al., 1979). Hence, the observed symptoms were factor analyzed to test the hypothesis for an underlying dimension, schizotypal personality disorder. In terms of the construct validation program, a nomological network was established, relating observed symptoms with theorized dimensions. Consequently, the above process can be said to have followed the guidelines provided by Cronbach and Meehl (1955).

As mentioned earlier, there exists a relatively large body of factor analytic validation studies of SPD. Examination of this literature offers further evidence for the belief in the power of factor analysis to indicate latent dimensions. For example, Claridge, McGreery, Mason, Bentall, Boyle, Slade, and Popplewell (1996, p. 104) write:
“A logical part of this development has been the construction of self-rating questionnaires which can act as easily administered screening instruments, used to select... individuals for further examination in genetic, clinical and laboratory studies that bear on the causes, antecedents and mechanisms of schizophrenia.” Or, as Kendler, McGuire, Gruenberg, and Walsh (1995, p. 302) write with regard to their factor analytic results of schizotypal personality disorders:

...these results suggest that further refinement of the personality ‘substrates’ for the major psychoses might hold promise for increasing the power of several key research paradigms in psychiatry. A reliable method of assessing the familial/genetic vulnerability to schizophrenia in a nonpsychotic population could substantially aid research aimed at clarifying the underlying familial/genetic, neurobiologic, developmental, or neuropsychologic substrates of the major psychoses.

Mason (1995, p. 279) writes:

The results of this analysis and the exploratory factor analysis on which it was based have prompted the development of a range of schizotypal scales.... My hope is that this can further the identification of more objective indices of risk in laboratory and genetic studies.... Our emerging understanding of the structure of psychosis proneness promises the possibility of identifying correlates of its various components.

The above quotes highlight the assumption that factor analytic sets of symptoms are related to underlying processes. Should a known psychiatric disorder reveal stable
and theoretically coherent patterns (sets) of symptoms, it would, according to the quoted authors, be reasonable to assume that the number of sets indicates the number of possible sub-diseases previously assumed to be a single disease, schizophrenia. Thus, by researching the structure of observed symptoms investigators hope to be simultaneously studying the latent dimensions as well. To wit, by “structure of psychosis proneness” Mason (1995, p.279) refers to the factor structure of SPD, while “identifying correlates of its various components” can be understood to refer to the various hypothesized dimensions or causes of schizophrenia and schizophrenia spectrum disorders. In other words, the factor structure of SPD and other spectrum disorders may tell us the number of diseases (and corresponding causes) encompassed by the construct of schizophrenia.

As described earlier in this paper, the basis of disease classification typically progresses from syndromal description to structural and functional abnormalities to, finally, causality. Hence, once a disease entity in question has been established through a method such as factor analysis, causal research may begin. Schizotypal personality disorder exemplifies this notion.

Even though personality disorders are often considered less likely than Axis I disorders to have a single, uniform cause, SPD has been researched extensively. A review of relevant literature reveals several lines of investigation into the causes of SPD. Genetically, diagnosis of SPD has been linked with schizophrenia (e.g. Kendler, McGuire, Gruenberg, O’Hare, Spellman, & Walsh, 1993; Torgensen, Onstad, Skre, Edvardsen, & Kringlen, 1993). Also, brain morphological abnormalities have been noted in association with SPD (e.g., Schulsinger et al., 1984). Finally, SPD has been linked
with abnormal neurotransmitter (dopamine) concentrations (e.g., Siever et al., 1991; Siever et al. 1993).
CHAPTER II
THREE SCENARIOS

Overview

The discussion provided so far in this paper should give the reader an indication of the use, the prevalence and the logic of factor analysis and construct validation in the service of mental disorder validation. The remainder of the paper will address some particular problems associated with these procedures. Even though many aspects of the current psychiatric classification system (American Psychiatric Association, 1994) have been criticized, the critiques presented here are largely unique to the present paper. For critiques of mental disorder classification from a different angle, consult for example Szasz (1960).

The purpose of this section is to critically evaluate the assumptions of construct validation and the linear common-factor analysis in the service of construct validation. However, before we begin, we wish to make two qualifications to our critique.

First, we do not posit that the outlined assumptions of factor analysis and construct validation are patently false. Some (organic) disease processes do give rise to sets of symptoms that are compatible with the linear common-factor analytic solution. Existence of a latent common factor describing a matrix of correlations among observed symptoms would in such cases be a good indication of the presence of an underlying process of one sort or another. Our critique, therefore, does not apply to all uses of factor analytic techniques in psychiatric research. Our sole intention is to point out the fact that the use of factor analysis and construct validation may lead to erroneous conclusions.
about the world. We will attempt to detail examples of such instances. The exact implications of these critiques to psychiatric classification and research are, therefore, relatively subtle, yet, I hope, useful in clarifying the source of some longstanding problems in classification, and in suggesting new directions for research. These implications will be discussed at the end of the paper.

Secondly, factor analytic methods can be used to answer different questions. Most importantly, factor analysis can be used as a) a criterion, b) a tool for data reduction, or c) a tool of discovery and validation. When these methods are used as a criterion, no apparent problems arise. Should one wish to build a model of mental illness, for instance, the use of factor analysis would be a useful tool in determining whether a certain phenomenon expresses itself in a manner similar to that depicted by the model. Here, one would apply factor analysis to see if the data are in keeping with one's theory. In this capacity, factor analysis is a valid tool. Also, factor analysis can be effectively used to reduce data. For instance, should a researcher wish to cluster psychiatric symptoms together for nothing but pragmatic reasons (e.g., to facilitate communication), factor analysis would be an applicable tool. In this scenario, the researcher does not claim to be answering more profound questions about what underlies such sets. Our critique, therefore, is directed towards the use of factor analytic techniques as a tool of discovery and validation.

The main critical question concerning factor analysis and the classification of psychiatric disorders asked in this paper is the following: Is it possible that there exist mental disorders that are not appropriately validated by the linear common-factor
analysis? That is, what are the limitations to the linear common-factor model, and what are alternative models? As McDonald (1986) explains, while the popular linear common-factor model is known to have its problems, the risks involved in its application are seen as acceptable. Also, as McDonald (1986) points out, the additional work involved in postulating alternative models is sometimes seen as not worth the effort, especially since software to implement factor analyses is widely available while software to implement other models (some of which are considered in this paper) is not. Consequently, alternatives to the linear common-factor model are rarely considered, even where factor analysis is clearly an inappropriate tool (Van Schuur & Kiers, 1994).

In considering this point, the question to be asked is: If an underlying disorder process of some kind exists, why should it manifest itself in a way that is amenable to a factor analytic solution? Or more generally, why should a disease entity manifest itself in one particular statistical manner rather than any other? For, logically, there is no necessity for disease entities to manifest themselves in any one manner; indeed, there are an infinite number of ways in which this may happen. The most commonly given answer in support for factor analysis is the fact that a great number of well documented diseases in fact do produce sets of symptoms in a way that factor analysis can discover them. Admittedly this is true of many known disorders, but surely this argument does not exclude the possibility that diseases (known or unknown) with factor analytically incompatible indicators exist. Consequently, factor analysis is naturally the right method for discovering disorders in the case of many diseases where indicators cluster in a factor analytic manner. However, in the cases where this does not happen, factor analysis is not
the right method for validating disease entities. Let us first examine a case of a factor analytically compatible symptom/disease relation.

Consider a population whose members are scored with respect to a disorder D (e.g., on a continuum from "not very ill" to "very ill"). The disorder has indicators $I_j$. The expression of the $I_j$ are linear functions of the level of D. Furthermore, in any group of people who are equally sick with D, they will differ only randomly with respect to the expression of the $I_j$. Hence, the $I_j$ will be uncorrelated within any of these conditional populations. Together, these two conditions imply the well-known factor analytic covariance structure. For instance, in the case of chicken pox, as the degree of illness increases, the expression of certain symptoms (e.g., itchiness and spots) also increases. For those who are very sick, almost all will have a large number of spots and itchiness, and hence these two indicators will be uncorrelated, and similarly for any other sub-population. Such disease manifestation is evident in a normal linear factor analytic solution, and correct assumptions about the number of disorders could logically be made using this method.

The above illustration exemplifies a case in which a linear relationship between the indicators, $I_j$, and the disease, D, exists. As shown earlier, the logic of factor analysis relies on the existence of a linear relationship between the disease and its indicators (symptoms). That is, the higher the level of disease, the higher the probability of manifesting each individual symptom.

This assumption may, however, be violated. We will next discuss in practical terms instances of such violations. These will, consequently, be instances in which the
use of linear common-factor analysis for validation is incorrect. The following scenarios outline such violations by way of examples of plausible disease/indicator relationships that factor analysis is unable to reveal. That is, if a disorder conforming to the models described here existed, factor analysis would not produce a correct picture of them. Statistical models and procedures better suited to describe these scenarios will be briefly examined at the end of the paper.

**Scenario 1: Unfolding model**

Consider a set of individuals, $P_i$, and a set of indicators, $I_j$ of a disorder $D$. The individuals may be positioned on a continuum from "not very ill" to "very ill" with respect to $D$. Furthermore, the indicators $I_j$ may be positioned on the same continuum. Their positioning reflects the level of $D$ at which they are most likely to be expressed. Hence, if the value of $I_2$ (the second indicator) is 5, then $I_2$ will be expressed with higher probability by those individuals at point 5 on the continuum. Specifically, the probability that individual $P_i$ will express symptom $I_p$, say $P_{jp}$, is a function of the distance from $P_i$ to $I_j$ along the continuum:

$$P_{jp} = f(|P_i - I_j|)$$

This implies that at each level of illness there will be a characteristic indicator/symptom (or possibly a group of symptoms, as each individual symptom may be replaced by a distinct cluster of symptoms). As a person gets sicker and sicker with $D$, he or she moves through a number of indicators/symptoms in a standard order (i.e., according to the scale values of $I_j$). Symptoms expressed at lower levels of illness are no longer expressed at
higher levels of illness. Thus, even though persons 1 and 2 are experiencing the same illness, D, the symptoms they manifest (all indicators of D) are very different.

The above type of manifestation has been termed the *unfolding model*. The difference between the standard factor analysis model and the unfolding model is that the former locates only people and not the indicators along the latent dimension, D, as the unfolding model does (Coombs & Kao, 1960; Van Schuur & Kiers, 1994). When applied to diseases, people's positions in the unfolding model reflect their optimal point along the disease dimension, whereas in factor analysis their positions (i.e., factor scores) reflect the severity of their symptoms.

It should be noted, that the referenced authors apply the unfolding model to attitude data as measured in terms of preference, that is, data is accrued from individuals' ranking of variables, such as political positions or traits as referring to oneself (e.g. Coombs, 1964). The clinical application of this model is original to the present paper. We will next attempt to give a brief explication of the model in this application.

As shown in scenario 1, the unfolding model posits a single latent dimension on which both the disease indicators and the individuals can be placed. Each individual has only one scale position with regard to the indicators, and each indicator has only one scale position with regard to the individuals. The closer a given individual to a given indicator, the more likely he or she is to manifest it. For example, consider a hypothetical mental disorder with indicators A, B, and C. Individuals can be rank-ordered with reference to A, B, and C. In other words, individuals manifest A, B, and C with varying intensity depending on their location on disease continuum D. Each
individual thus presents an order of severity (or likelihood). This means that six individual orderings for the disorder can exist: ABC, ACB, BAC, BCA, CAB, and CBA. An individual with a BAC order, for example, manifests indicator B more severely than indicator A, and A more severely than indicator C. A given individual's ordering data is referred to as that individual's I scale, and the point of individual $i$ on the disease continuum $D$ is known as that individual's ideal point.

The set of I scales from all individuals constitutes the data in unfolding analysis. Unidimensional unfolding analysis attempts to "unfold" the I scales onto a single D scale by placing the indicators and individuals in locations on D consistent with the observed I scales. For each individual, this process can be conceptualized as a hinge placed on the D scale at the individual's ideal point. The D scale is then folded at the ideal point, reproducing an ordering of indicators which, if the model is correct, will correspond to the individual's I scale. The observed data are said to fit a unidimensional unfolding model if all I scales can be "unfolded" onto a common D scale.

To illustrate the unfolding model, consider syphilis. The cause of syphilis is a treponema pallidum spirochete. The disease manifests itself in three separate stages: In the primary stage, which occurs roughly three weeks after contact with an infected individual, a single, firm ulcerated lesion appears at the site of contact. Without treatment, the lesion heals in 2-3 weeks. In the secondary stage, a rash appears. Again, without treatment, the rash disappears, and the disease organisms withdraw into the body, and remain dormant for varying lengths of time. In the tertiary state, a number of different manifestations may occur. The most common manifestations are
cardiovascular disease and central nervous system disease manifested as paralysis, insanity, and blindness (Kent & Hart, 1993).

There is no clear example of a mental disorder exemplifying the unfolding model in the current DSM scheme (this is the very reason for considering the model here). For the purpose of illustration, however, let us consider a hypothetical scenario. Here, it is possible to imagine a mental disorder with compensatory symptom manifestation, that is, each symptom (or some symptom) arises as a compensation for preceding symptoms. Possible functional relationships may, for example, be of the form whereby one symptom arises in order to alleviate the perceived suffering associated with another symptom. Many unusual behaviors may in fact arise to mask and replace underlying anxiety or depression. Such behaviors may, then, in turn be replaced by unusual mentation or by further, but different behavior. When such new behaviors and thoughts successfully replace the underlying feelings as the sole clinical manifestation, an unfolding model may be applicable.

If scores on a set of indicators similar to the syphilis, or the hypothetical example above, were produced on the basis of a disease model, they would certainly not fit a unidimensional linear factor model (even though only one disease, syphilis, is in play). Factor analysis would, in fact, lead to the conclusion that there exist at least two disease entities to be discovered. The reason for this is clear; the linear factor model implies that as the level of D increases, so too does the probability of manifesting every symptom or indicator. The model implies a linear increasing indicator/disease regression instead of the inverted U-shape regressions that characterize the relationship between syphilis and
its indicators.

To illustrate the way in which the unfolding model behaves when factor analyzed, the following hypothetical scenario was constructed: six indicators of a hypothetical mental disorder were entered on a line in random order and at random distances from each other. The line, then, represented a single disorder, D (see Figure 1). On the same line were placed ten individuals in random order and at random distances from each other. The distance along the line between a given individual and given indicator represents the probability of the individual manifesting the symptom. In other words, the shorter the distance between an individual and an indicator, the more likely he or she is to manifest it. This probability can be understood for example as a score on a relevant clinical Likert scale.

The line was converted into a $X \times Y$ matrix of measured distances of individuals from the indicators. A correlation matrix of indicators was computed, and the resulting matrix was factor analyzed. Table 1 shows the matrix of measured distances between each individual and each indicator. Table 2 shows the correlation matrix among the indicators. The matrix was subjected to a linear factor analysis, and two factors emerged [eigenvalues 3.897 and 2.017]. Factor loadings and total variance explained are given in Table 3.

When these results are interpreted from the point of view of construct validation, it is clear that there exists a potential for misinterpretation. Drawing from the logic of factor analysis, a researcher may well conclude that the two factors suggest the presence
Figure 1

Scenario 1: Unfolded D Scale

\[
\begin{array}{cccccccccc}
I_2 & I_4 & I_1 & I_6 & I_5 & I_3 \\
-5 & -4 & -3 & -2 & -1 & 0 & 1 & 2 & 3 & 4 & 5
\end{array}
\]

Table 1

Scenario 1: Measured distances between individuals and indicators

<table>
<thead>
<tr>
<th>Individual</th>
<th>Indicator 1</th>
<th>Indicator 2</th>
<th>Indicator 3</th>
<th>Indicator 4</th>
<th>Indicator 5</th>
<th>Indicator 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.00</td>
<td>1.00</td>
<td>10.00</td>
<td>2.00</td>
<td>8.00</td>
<td>6.50</td>
</tr>
<tr>
<td>2</td>
<td>6.00</td>
<td>10.00</td>
<td>1.00</td>
<td>9.00</td>
<td>3.00</td>
<td>4.50</td>
</tr>
<tr>
<td>3</td>
<td>2.50</td>
<td>1.50</td>
<td>7.50</td>
<td>.50</td>
<td>5.50</td>
<td>4.00</td>
</tr>
<tr>
<td>4</td>
<td>3.50</td>
<td>7.50</td>
<td>1.50</td>
<td>6.50</td>
<td>.50</td>
<td>2.00</td>
</tr>
<tr>
<td>5</td>
<td>1.00</td>
<td>3.00</td>
<td>6.00</td>
<td>2.00</td>
<td>4.00</td>
<td>2.50</td>
</tr>
<tr>
<td>6</td>
<td>.50</td>
<td>4.50</td>
<td>4.50</td>
<td>3.50</td>
<td>2.50</td>
<td>1.00</td>
</tr>
<tr>
<td>7</td>
<td>4.50</td>
<td>8.50</td>
<td>.50</td>
<td>7.50</td>
<td>1.50</td>
<td>3.00</td>
</tr>
<tr>
<td>8</td>
<td>2.00</td>
<td>6.00</td>
<td>3.00</td>
<td>5.00</td>
<td>1.00</td>
<td>.50</td>
</tr>
<tr>
<td>9</td>
<td>5.50</td>
<td>9.50</td>
<td>.50</td>
<td>8.50</td>
<td>2.50</td>
<td>4.00</td>
</tr>
<tr>
<td>10</td>
<td>2.00</td>
<td>2.00</td>
<td>7.00</td>
<td>1.00</td>
<td>5.00</td>
<td>3.50</td>
</tr>
</tbody>
</table>

*Note:* The unit of measurement is given in Figure 1
### Table 2

**Scenario 1: Correlation matrix of indicators**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>--</td>
<td>.553</td>
<td>-.369</td>
<td>.657</td>
<td>.059</td>
<td>.672</td>
</tr>
<tr>
<td>2</td>
<td>--</td>
<td>--</td>
<td>-.969</td>
<td>.984</td>
<td>-.753</td>
<td>-.194</td>
</tr>
<tr>
<td>3</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>-.918</td>
<td>.875</td>
<td>.389</td>
</tr>
<tr>
<td>4</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>-.667</td>
<td>-.076</td>
</tr>
<tr>
<td>5</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.763</td>
</tr>
<tr>
<td>6</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>
### Table 3

**a) Scenario 1: Factor loadings**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Factor 1</th>
<th>Factor 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.425</td>
<td>.874</td>
</tr>
<tr>
<td>2</td>
<td>.984</td>
<td>.161</td>
</tr>
<tr>
<td>3</td>
<td>-.988</td>
<td>.0528</td>
</tr>
<tr>
<td>4</td>
<td>.950</td>
<td>.284</td>
</tr>
<tr>
<td>5</td>
<td>-.853</td>
<td>.490</td>
</tr>
<tr>
<td>6</td>
<td>-.351</td>
<td>.937</td>
</tr>
</tbody>
</table>

**b) Scenario 1: Total variance explained**

<table>
<thead>
<tr>
<th></th>
<th>Eigenvalue</th>
<th>Percentage of variance</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>3.897</td>
<td>65.0</td>
<td>65.0</td>
</tr>
<tr>
<td>Factor 2</td>
<td>2.017</td>
<td>33.6</td>
<td>98.6</td>
</tr>
</tbody>
</table>
of two separate disorders (latent dimensions). In this case, the two disorders can be seen as responsible for their respective clusters of symptoms. Here, symptoms 2, 3, 4, and 5 could be considered to indicate one disorder, and symptoms 1 and 6 another (with symptoms 1, 5, and 6 loading significantly on each). While this interpretation is in keeping with the construct validation program, it is clearly not a correct representation of the actual disease process. The correct representation would, in fact, be that only one disease process, with an unfolding manifestation, is in play. The unfolding model, then, can be used to exemplify a clinical scenario whereby the linear common-factor model is an inappropriate tool of validation.

A further note on the factor analytic results of scenario 1 should be made: it is evident that indicator location along the unfolding scale can be inferred to an extent by examining the factor loading matrix. Particularly, note that adjacent indicators along the unfolding scale load highly on the same factor (2 and 4, 1 and 6, 5 and 3).

**Scenario 2: Non-linear model**

Consider a standard factor analytic model, except that the indicator/disease regressions are merely monotone increasing. That is, as the severity of D increases, so too does the extremity of expression of each of the symptoms. The functional form of these regressions are left unspecified. While it is possible to postulate a scenario whereby a given causal agent or agents give rise to a disease entity D, and D is linearly related to each symptom, it is also possible to postulate a scenario whereby D is either non-linearly (but similarly) related to each indicator, or a scenario whereby D is
differently related to each indicator. Certainly, there is no necessity for each
disease/indicator regression to be linear (see Figure 2). This model is a unidimensional
non-linear (monotone) factor analysis model.

Consider also a situation where the disease/indicator relationship is neither linear
nor monotone increasing. Various U-shaped regressions, for example, can be considered
here.

Fundamentally, the above scenario means that at the covariance structure level it
is not possible to distinguish between a disease/indicator relationship that conforms to a
one-dimensional monotone factor analysis model (or a U-shaped model) and an s>1
dimensional linear factor analysis model. If symptoms are generated in this way, and the
linear factor analysis model is applied, the investigators would conclude that there
existed a number of disorders linked linearly to a set of indicators.

As a clinical example, consider for instance suicidality as an indicator of
underlying depression. Here, suicidality (or the actual likelihood of committing suicide)
is not linearly linked to depression. In fact, suicide risk is higher at lower levels of
depression than at higher levels, the risk being highest after the depressive episode begins
to lift.
Figure 2

Scenario 2: Examples of possible functional regressions of indicators

\[ \text{x axis = disease} \]
\[ \text{y axis = severity of symptom} \]
To illustrate the above numerically, a scenario was constructed whereby a single disorder, D, was manifested by seven indicators\(^2\). Each indicator was differently related to D. Here, a normally distributed variable \(x\), called factor, with mean of 0, and standard deviation of 1 was generated. \(X\) was then transformed according to seven different equations. Each equation (factor) was also given a residual (\(e_1, e_2, e_3\), etc.) that was independent of the factor. The seven variables were thus linear combinations of the equations plus a residual.

The constructed regressions of each indicator are listed in Table 4. From Table 4, it is obvious that regressions 3 and 6 are linear, regression 1 is U-shaped, regressions 5 and 7 are inverted U-shaped, and regressions 2 and 4 are S-shaped. A total of 464 cases were randomly generated and the sample correlation matrix was subjected to a linear factor analysis. Two factors emerged, with eigenvalues of [3.965 and 2.925]. Factor loadings and total variance explained are given in Table 5.

When these results are interpreted from the point of view of construct validation, there is again potential for misinterpretation. The two factors may be taken to suggest the presence of two disorders with indicators 2, 3, 4, and 6 being measurements of one underlying disorder, and indicators 1, 5, and 7 being measurements of another disorder. The interpretation might run something like this: disease process 1 gives rise to elevation in symptoms 2, 3, and 6, and a near-absence of symptom 4, while disease process 2 gives

\(^2\) The number of possible scenarios is infinite. Each regression can be defined differently, and each indicator can have an infinite number of functions with a given regression. The scenario here, hence, is not meant as an empirical disease model, but as a demonstration of an instance in which linear common-factor analysis is an incorrect tool of validation.
Table 4

**Scenario 2: Constructed regressions of indicators**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$w=0.17x +1.115[(x^2-1)/\sqrt{2}]+0.73e_1$</td>
</tr>
<tr>
<td>2</td>
<td>$d=[\exp(2.34x)/1+\exp(2.34x)]+0.123e_2$</td>
</tr>
<tr>
<td>3</td>
<td>$y=0.995x+0.099e_3$</td>
</tr>
<tr>
<td>4</td>
<td>$z=-1.087[\exp(2.34x)/1+\exp(2.34x)]-0.276e_4$</td>
</tr>
<tr>
<td>5</td>
<td>$a=0.013x-1.128[(x^2-1)/\sqrt{2}]+0.114e_5$</td>
</tr>
<tr>
<td>6</td>
<td>$b=0.894x+0.447e_6$</td>
</tr>
<tr>
<td>7</td>
<td>$c=-1.108[(x^2-1)/\sqrt{2}]+0.76[\exp(2.34x)/1+\exp(2.34x)]+0.218e_7$</td>
</tr>
</tbody>
</table>
Table 5

a) **Scenario 2: Factor loadings**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Factor 1</th>
<th>Factor 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indicator 1</td>
<td>0.259</td>
<td>-0.966</td>
</tr>
<tr>
<td>Indicator 2</td>
<td>0.982</td>
<td>0.087</td>
</tr>
<tr>
<td>Indicator 3</td>
<td>0.987</td>
<td>0.043</td>
</tr>
<tr>
<td>Indicator 4</td>
<td>-0.984</td>
<td>-0.085</td>
</tr>
<tr>
<td>Indicator 5</td>
<td>-0.106</td>
<td>0.993</td>
</tr>
<tr>
<td>Indicator 6</td>
<td>0.984</td>
<td>0.041</td>
</tr>
<tr>
<td>Indicator 7</td>
<td>0.103</td>
<td>0.994</td>
</tr>
</tbody>
</table>

b) **Scenario 2: Total variance explained**

<table>
<thead>
<tr>
<th></th>
<th>Eigenvalue</th>
<th>Percentage of variance</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>3.965</td>
<td>56.6</td>
<td>56.6</td>
</tr>
<tr>
<td>Factor 2</td>
<td>2.925</td>
<td>41.8</td>
<td>98.4</td>
</tr>
</tbody>
</table>
rise to elevation in symptoms 5 and 7, and a near-absence of symptom 1. The non-linear model may, thus, be taken as an example of a mental disorder about which wrong conclusions would be made when validation is attempted using the linear common-factor model.

Scenario 3: Guttman’s idealized scale

Consider a threshold model where indicators can be scored dichotomously (0 or 1). That is, individuals along continuum D either do or do not manifest a given indicator. Before point \( \theta \) along the continuum D, a given indicator is manifested at 0% probability, and after \( \theta \), the indicator is manifested at 100% probability. Indicators are placed along D sequentially. As an individual gets sicker and sicker with the D, he or she moves through a number of indicators/symptoms in a standard order. Consequently, all symptoms at lower levels of D are also present at higher levels of illness. This model is known as Guttman’s idealized scale, or a deterministic model, as an individual’s probability of endorsing an item, or exhibiting a symptom, is either 0% or 100% with no intermediate values (Guttman, 1941; Waller, Tellegen, MacDonald, & Lykken, 1996). Figure 3 shows a Guttman’s idealized scale with a numerical illustration of six indicators and ten individuals.

As a clinical example of this type of manifestation, consider a progressive viral infection which gives rise to psychological and neurologic symptoms in a standard order. Here, a client may lose given cognitive functions (such as various memory deficits) as a result of an infection, which in turn may give rise to psychiatric symptoms (e.g.,
Scenario 3: Guttman’s Idealized Scale with numerical illustration.

x axis = Disease

y axis = Probability of manifesting symptom
depression, anxiety, etc.) at points of disease process where these symptoms become apparent to him or her.

The assessment of dimensionality when done through factor analysis is shown to yield an incorrect representation of the data when applied to Guttman's idealized scale (e.g. Zwick, 1986). While only one disorder, D, is clearly in play, a linear factor analysis of Guttman's idealized scale results in two or more factors, lending support for the conclusion that more than one disorder is involved.  

To illustrate the above, a numerical representation of Guttman's scale was constructed. Ten individuals on six indicators were entered on a matrix. Manifestation of an indicator was scored as 0 or 1 (yes, no). The resulting correlation matrix among the indicators was subjected to a linear factor analysis.

Table 6 shows the matrix of individual scored on each indicator. Table 7 shows the correlation matrix among the indicators (note, the presence of Guttman scalable items should be a warning sign for researchers from the outset not to apply factor analysis to the data). The matrix was factor analyzed, and two factors with eigenvalues larger than 1 emerged [eigenvalues 3.333 and 1.144]. Factor loadings and total variance explained are given in Table 8.

When these results are interpreted from the point of view of construct validation, the two emerging factors may lead to a conclusion that two disorders are responsible for the data. Here, symptoms 1 to 6 may be taken to indicate one disorder, with symptoms 2

---

3 Guttman's idealized scale has its application in test theory, where the extra factor was initially, and wrongly, considered to be a difficulty factor (McDonald & Ahlawat, 1974).
Table 6

Scenario 3: Matrix of scores on indicators 1 to 6

<table>
<thead>
<tr>
<th>Individual</th>
<th>Indicator 1</th>
<th>Indicator 2</th>
<th>Indicator 3</th>
<th>Indicator 4</th>
<th>Indicator 5</th>
<th>Indicator 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>2</td>
<td>1.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
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### Table 7

**Scenario 3: Correlation matrix of indicators 1 to 6**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<tr>
<td>1</td>
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<td>.272</td>
<td>.218</td>
<td>.111</td>
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<td>.802</td>
<td>.535</td>
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Table 8

a) Scenario 3: Factor loadings

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Factor 1</th>
<th>Factor 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>-.282</td>
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<tr>
<td>2</td>
<td>.821</td>
<td>-.508</td>
</tr>
<tr>
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<td>.829</td>
<td>-.244</td>
</tr>
<tr>
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<td>.829</td>
<td>.244</td>
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<tr>
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<td>.821</td>
<td>.508</td>
</tr>
<tr>
<td>6</td>
<td>.428</td>
<td>.282</td>
</tr>
</tbody>
</table>

b) Scenario 3: Total variance explained

<table>
<thead>
<tr>
<th></th>
<th>Eigenvalue</th>
<th>Percentage of variance</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>3.333</td>
<td>55.6</td>
<td>55.6</td>
</tr>
<tr>
<td>Factor 2</td>
<td>1.144</td>
<td>19.1</td>
<td>74.6</td>
</tr>
</tbody>
</table>
and 5 indicating a potential second disorder. However, since the second factor does not have very strong factor loadings, the researcher may conclude, that in fact only one "true" disease process exists. This would naturally constitute a misrepresentation as well, since Guttman-scalable data does not conform to the factor analytic solution. A factor-analytic interpretation of the data would clearly miss the dichotomous, sequential nature of the symptom manifestation. Hence, in the case of a Guttman-scalable scenario, an attempt to validate the disorder by way of linear common-factor analysis would yield wrong conclusions.
CHAPTER III

CONCLUSION

In summary, this paper first examined the enterprise of medical and psychiatric classification. Here, it was noted that while disease classification in medicine typically proceeds from symptom classification to classification of functional and structural, and finally, causal elements, psychiatric classification was characterized as relying on syndromal description alone. Validation of mental disorders was then examined within the framework of construct validation and factor analysis. The use of these methods in psychiatric and psychological research was exemplified by the construction and validation of schizotypal personality disorder. The rest of the paper examined the possibility that mental disorders may give rise to symptom manifestations that are not compatible with the linear common-factor model. Factor analysis and construct validation, with their psychometric assumptions, were thus shown to be theoretical positions with the potential of mischaracterizing the nature of mental disorders.

The critiques in this paper are not considered to warrant complete abandonment of factor analysis and construct validation. Rather, the purpose of the paper is to alert the researcher to some potential problems in the prevalent scheme for classifying mental disorders. Given the present critique, the most serious problem associated with the construct validation using linear common-factor analysis appears to be the possibility of serious misinterpretation and of overlooking disorders that do not manifest themselves in accordance with the linear factor model. In the light of the critiques presented in this paper, let us next consider some suggestion for psychiatric and psychological research.
1. We suggest analyses of existing and future data be conducted with statistical techniques equipped to screen for the presence of disease/indicator relationships alternative to the linear common-factor model. As was shown, the linear common-factor model may lead to misinterpretation of data that does not fit into the standard conception of the relationship between indicators and diseases/disorders. To this end, this paper has examined occasions in which mistaken assumptions may arise when the linear model is applied. What follows, then, are brief suggestions promoting data analyses and research to avoid such pitfalls.

In the case of the unfolding model, the most important research tool is the awareness that such models may exist. If the researcher is knowledgeable about the type of model he or she is dealing with, its detection can be fairly simple. The detection must, of course, first be done at the level of clinical observation. Here, the sequential nature of symptoms must first be established. After this, the presence of an unfolding model may be confirmed statistically. For example, Davison (1977) offers a few diagnostics for the detection of an unfolding model. Firstly, if data conform to a unidimensional unfolding model, the correlation matrix of the stimuli, when ordered according to their position along the unfolding dimension, forms a pattern whereby the correlations are highest between adjacent stimuli and decrease monotonically from the diagonal downward and to the left. This may be done in order to compare the efficacy of an unfolding analysis to represent the data as compared to other types of analyses. Table 9 exemplifies this for scenario 2 for a case in which an unfolding manifestation of a particular order (indicator 2, 4, 1, 6, 5, and 3) is suspected.
Secondly, in the case of a unidimensional unfolding model, the sign of the partial correlation of two stimuli with a third depends on the relative position of the three stimuli. A partial correlation \( r_{AB.C} \) is positive if \( C \) is located to the left or right of \( A \) and \( B \), but it is negative if \( C \) is positioned between \( A \) and \( B \). Ross and Cliff (1964) suggest double centering (i.e. rendering the means of both rows of individuals and columns of stimuli 0.0) the data matrix, and establishing and inspecting its rank.

In the case of nonlinear disease/indicator relationships, a valuable, but seldom used tool of quadratic factor analysis (McDonald, 1967a) can be used. The linear common-factor model holds the assumption of a linear conditional expectation of symptom manifestation given the common factor. As shown by McDonald (1962; 1967a; 1986), this expectation can be liberated. Quadratic (nonlinear) factor analysis represents a factor model which assumes that the observed variables (symptoms) are nonlinear functions of a common factor. In this model, disease indicators are treated as a function of an individual’s position along the latent continuum and the conditional nonlinear expectation of the indicators given the common factor. The regressions do not need to be monotone. Quadratic factor analysis can be applied with relative ease through PROTEAN (McDonald, 1967b) or NOHARM (Normal Ogive Harmonic Analysis Robust Method) (Fraser & McDonald, 1988) computer programs, which screen for possible nonlinear models. For a technical discussion of this model and the appropriate software, consult McDonald (1967b) and Fraser and McDonald (1988) respectively.

2. We suggest that construct validation should not be made a criterion for a mental disorder classification scheme. That is, the validity of a diagnostic category should not
Table 9

Scenario 2: correlation matrix of indicators according to position along unfolding dimension

<table>
<thead>
<tr>
<th>Indicator</th>
<th>2</th>
<th>4</th>
<th>1</th>
<th>6</th>
<th>5</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>.984</td>
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<td></td>
<td></td>
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<td></td>
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<tr>
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<td>.553</td>
<td>.657</td>
<td>--</td>
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<td>.672</td>
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<tr>
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<td>.763</td>
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<td>-.369</td>
<td>.389</td>
<td>.875</td>
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</table>
be considered rest on construct validation findings alone. Particularly, it is important to note that while some symptoms may not be properly embedded in the nomological network, they may still be indicators of an underlying process or processes. That is, underlying disease processes must by no necessity give rise to a set of symptoms in a particular statistical manner; it is equally possible that single symptom, or many symptoms exhibited in a non-factor analytic manner may be linked to a single structural or functional abnormality or causal source.

3. We suggest that efforts be made to construct new models of possible types of disease/indicator relationships, into which existing and future data can be fitted. Data on theoretically and intuitively unlikely indicator combinations should be examined in this way. Various theories of psychopathology may be useful in constructing psychiatric classification systems. However, understanding the importance of using various theories of data as well, such as those discussed in this paper, may be helpful in discovering previously unconsidered mental disorders.
References


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Appendix A

Empirical realism and factor analysis

The use of factor analysis in the service of the mental disorder validation is an offshoot of an ontological theory known as empirical realism. The main tenet of the empirical realist view is that there is no fundamental difference between what are commonly understood as theoretical terms that refer to unobservable entities and entities that are more commonly understood as observable and physical. The realist aim of science from this perspective is the production of ontological (existential) statements about unobserved entities (Harre, 1970). Applied to psychiatric research, this view holds that the observed sets of correlations between symptoms justify ontological claims about unobserved entities, namely causality (Norris, 1983). Rozeboom (1984, p. 212-213) describes the position as follows:

This is the thesis that although theoretical terms get their meanings from the data-language contexts in which they are used, what they semantically designate are causal features of natural reality generally concealed from perception but knowable through their data consequences. [emphasis original]

A straightforward example of Rozeboom's "data consequences" is a factor analytic solution which points to a number of causal entities.

The empirical realist position, like many other theoretical positions, is commonly expressed through metaphors. The main metaphor here is that of objects. Mulaik (1996, p. 589-590) describes this metaphor, and its implications for factor analysis and the
concept of factor indeterminacy as follows:

I regard the common factor model as inspired by the object metaphor. Thus I like to think of the value of a latent variable as like an attribute of an object, with the value of each indicator variable like a different point of view of the object's attribute, displaying both invariant properties of the latent attribute as well as idiosyncratic properties, represented by the unique factors.... Being "objective" the latent variable is not dependent on a particular set of variables, but "exists" in its own right independent of the knower as a measure of a property of an independently existing set of objects.

Mulaik's presentation emphasizes all the important tenets of the empirical realist position as applied to modern scientific investigation. First, observed covariations are to be taken to indicate the presence of unobserved entities. Second, these unobserved entities yield a causal effect on observable variables. Third, unobserved entities exist independently of the organisms observing and categorizing them. Finally, empirical investigations shed light on meanings of concepts (i.e., concepts begin as undetermined, but become less so as more information is gathered about the objects that denote them).
VITA AUCTORIS

Jarkko Jalava was born in 1970 in Mantsala, Finland. He completed a B.A. in English at Dalhousie University in 1994, and a B.A. in Psychology at Simon Fraser University in 1996. Currently, he is enrolled in the PhD program in Adult Clinical Psychology at the University of Windsor.