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Special Feature Article

How Can We Validate Melancholia (Endogenous Depression)?

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Abstract

Whether melancholia (endogenous depression) is a distinct entity or simply a severe case of major depressive disorder (MDD) remains a topic of debate. In this paper, the author discusses how to prove melancholia empirically and the difficulty in doing so. To prove the existence of the disease, it is first necessary to identify the syndrome using factor or cluster analysis. Then, the validity of the diagnosis must be tested by examining the correlation between the syndrome and validators. However, it is difficult to show consistently a correlation between melancholia and validators using the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition criteria for melancholia (DSM-MEL). One of the reasons for this is that four of the eight DSM-MEL items overlap with the criteria for MDD. Another reason is that the symptoms of melancholia have a "distinct quality" in that the patient cannot clearly verbalize, which is difficult to define explicitly. These limitations may be overcome by defining melancholia based on a key characteristic, psychomotor disturbances, or by using a prototypical diagnostic measure. Taking the perspective that psychiatric symptoms do not exist as entities, but rather, are the final result of dialogical co-constructs arising from the clinical relationship, suggests that demonstrating the existence of mental disorders has certain limitations.

Keywords: melancholia, positivism, psychomotor disturbance, validity, distinct quality

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Introduction

Major depressive disorder in DSM-5 2) includes two illnesses in conventional diagnostics: endogenous depression and non-endogenous depression. This concept is referred to as the "binarian" of depressive illness. On the other hand, the concept that major depressive disorder is a single illness is "unitarian". From the unitarian point of view, the two conditions, endogenous and non-endogenous depression, differ only in severity. Here, non-endogenous depression refers to "depression with a reason," which used to be called neurotic or reactive depression.

The binarian view is unpopular today because empirical studies have failed to reproducibly show results in favor of it. Nonetheless, the author stands in defense of the binarian. The reason for this is partly based on the conceptual history of depression 40)61), but in the end, it is simply because it is how he feels in actual clinical practice. If we superimpose the overall impression of an inability to understand the onset of symptoms and characteristics of symptoms, along with the characteristics of endogenous depression emphasized in traditional psychopathology, such as psychomotor retardation 51), vital sadness 51), feelings of having lost feeling 17)51), perplexity 17), and non-reactivity of mood, there seems to be a qualitatively different group of patients not only involving severe cases but also patients with mild depressive pathology who do not require hospitalization.

However, under today's "institutionalized science" 38), such personal experiences are not convincing. They must be empirically verified in a way that is as free from subjective preconceptions as possible. In this paper, the author discusses the methods and difficulties in empirically exploring the existence of endogenous depression, based on the question of whether it is possible to bridge traditional psychopathology, which emphasizes the subjective experiences of medical professionals, and empirical research, which tries to eliminate such subjective experiences as much as possible.

I. Empirically Exploring Existence of the Illness

What kind of efforts are considered "empirical"? According to classical positivism formulated in the mid-19th century, it must be based not on speculation or inference, but on facts based on "observation". Then, hypotheses are derived from them, and objective facts that can be reproduced are established through experimental "verification". Such a procedure is considered the standard method of

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empirical science 37).

If all illnesses existed as objects that could be directly seen and touched, such as pimples and tumors, they would be easy to observe. However, this is not the case with illnesses of the mind. It is impossible to directly observe the true nature of the illness. Furthermore, it has not been determined whether mental disease, like physical disease, has a material lesion. The definition of "illness" varies widely, with some considering physical lesions to be evidence of illness, while others consider them to be patterns of human adaptation to the environment. Others consider subjective suffering to be an illness, while still others consider illness to be something that physicians have a therapeutic interest in 22).

However, since endogenous depression is a disease that requires a biological basis, the discussion here will follow the so-called medical model, in which the presence of physical lesions is evidence of the disease. In other words, the question is whether it is possible to demonstrate the existence of endogenous depression as a "disease entity" as pursued by Kraepelin, E.

A disease entity is a unit of disease that "has the same cause, the same basic psychological form, the same development and course, the same outcome, and the same brain findings" 17). Among these, the cause is unobservable, and there are no known brain findings specific to endogenous depression. Therefore, we must first observe the syndrome, which is a collection of symptoms and signs, and verify the degree to which the syndrome corresponds to the physical cause, in other words, the validity of the diagnosis 41). Specifically, the procedure is as follows: (1) to verify the cohesiveness of the syndromes, and (2) to examine the correlation between the syndromes and validators. As a validator, an index that is independent of the syndrome and not subjective to the evaluator is chosen. Kendler, K.S. 25) divided validators into: (a) antecedent validators, (b) concurrent validators, and (c) predictive validators. Group (a) may include family studies, premorbid characteristics, demographic factors, and precipitating factors; (b) may include laboratory data; and (c) may include diagnostic consistency over time, other follow-up data, and treatment responsiveness. The following section discusses the demonstration of endogenous depression according to this methodology.

II. Empirical Consideration of Endogenous Depression

1. The Name "Endogenous Depression"

It is important to note the usage of the term "endogenous depression" 32). This term refers to the nature of the cause,

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which is "neither exogenous nor psychogenic," but it is also used as a term of art to describe a symptom pattern. Today, the identification of endogenous depression based on symptom patterns 14)28) is the norm, and even if there are seemingly psychogenic triggers, if the symptom pattern applies, the patient is considered to have endogenous depression. In fact, there are many such cases, and empirical studies have found little relationship between the symptom pattern of endogenous depression and preceding life events 47). Thus, the term "endogenous depression" is no longer an adequate descriptor of its inclusion. This is the reason why the term "melancholia" 43)63) is used instead of "endogenous depression" in modern English-speaking psychiatry 36)56). In this paper, the terms "endogenous depression" and "melancholia" are used interchangeably.

2. Demonstrating the Existence of the Syndrome

The standpoint of empirical research is "observation". In psychiatry, the patient's language, expression, and behavior are the objects of observation. To increase reliability, operative definitions of syndromes and structured interviews are used.

Multivariate analyses, such as factor and cluster analyses, were used to empirically identify symptom patterns, and such studies of depressive states were conducted extensively in Anglo-American psychiatry in the 1960s and 1970s. Kendell, R.E. 23), who reviewed those studies, agreed on the need to divide the two types of depression into those corresponding to endogenous and non-endogenous depression, but they were inconclusive regarding the relationship between the two types: whether they were separate categories or poles of a single dimension.

Nelson, J.C. et al. 36), who extensively reviewed factor analysis, cluster analysis, discriminant analysis, and treatment responsiveness studies, found that "psychomotor disturbances such as retardation and agitation, lack of responsiveness to environmental changes, severe depressive mood, depressive delusions, self-blame, and loss of interest in pleasure" were strongly associated with endogenous depression. A recent review of factor analysis studies 31) also revealed that "psychomotor retardation, non-reactivity of mood" was most relevant to the identification of endogenous depression.

In view of these findings, psychomotor disturbances, non-reactivity of mood, severe depressed mood, depressive delusions, and self-blame may be empirically identified as syndromes. However, how can we verify whether

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this syndrome is an independent category from other depressive syndromes or just a severe case of a depressive syndrome?

3. Category or Dimension?

In the 1970s, Kendell 22)24) emphasized the concept of bimodal distribution to mathematically test whether a natural boundary exists between the two syndromes. The idea is that two independent categories can be proved by taking a distribution with a point of rarity in the center, as shown in the figure. Using this concept, one study 7) was able to prove a point of rarity between endogenous and non-endogenous depression, but was not successful in replicating it 24). This method of proof has many criticisms. For example, it has been pointed out that if the difference between the means of the two groups is small and number of samples is not sufficiently large, the distribution may apparently be normal 27), and that the shape of the peaks can be altered by changing the interval of the scale on the horizontal axis for the same data, making it possible to create an artificial bimodal distribution 10).

Recently, taxometric analysis developed by Meehl, P. E. has attracted attention as a new method to statistically verify whether endogenous depression is a category or dimension 27)34), but at present it is not possible to conclude that endogenous depression is an independent syndrome.

4. Examine Syndrome Validity

To examine the validity of a syndrome, we must test its correlation with a validator that is independent of the syndrome and as free from the evaluator's preconceptions as possible. In the 1980s, Zimmerman, M. et al. 71) identified the following 14 validators: (1) more family history of affective disorders, (2) less family history of alcoholism, (3) less family history of antisocial personality disorder, (4) older age, (5) greater severity of illness, (6) fewer minor suicide attempts, (7) less marital separation or divorce, (8) fewer stressful life events during the year, (9) less personality disorder before illness (10) more social support, (11) fewer cognitive distortions (frequency of perversion or overreaction to neutral events), (12) more abnormal biological findings (e.g., dexamethasone suppression test), (13) better response to physical treatment (antidepressants and electroconvulsive therapy), and (14) lower response to psychotherapy. The diagnostic criteria for endogenous depression used in studies at that time included the Research Diagnostic Criteria (RDC) 55) and the Newcastle Index 7) in addition to DSM-III 1), but in recent years, most studies have used the DSM criteria for melancholia (major

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depressive disorder with melancholic features). The DSM criteria for melancholia were not determined by empirical procedures such as factor analysis 71).

The correlations between DSM criteria for melancholia and validators are not consistent, except for "greater severity of illness" 5)8)33)66). Demographic characteristics reported include more males 8)33), more unemployed 8)9)33), more experience of childhood abuse 5)8), and lower social functioning 8), but there is also a report 66) that when matched for severity, no differences in demographic characteristics were found between melancholia and non-melancholic depression. The association with suicide attempts is also inconsistent 8).

Traditionally, endogenous depression was considered to respond well to antidepressants and electroconvulsive therapy 7)26), but recent studies have failed to replicate these results 5)6)33)67)68). It is also difficult to conclude the superiority or inferiority of different types of antidepressants 8)49). The low-level response of patients with endogenous depression to a placebo has attracted attention 6), but this is also not consistent among studies 16). Abnormal findings in the dexamethasone suppression test and sleep EEG, which have attracted attention as biological characteristics, are also found in other psychiatric disorders, and so their specificity is limited 27)49).

In terms of premorbid personality, personality disorders have often been considered a feature of non-endogenous depression in Anglo-American psychiatry 63). However, the premorbid personality that positively characterizes endogenous depression has not been empirically established. In Japan, "immodithymia" and melancholic personality have traditionally been emphasized as indices to differentiate endogenous from non-endogenous depression 58)59)61). However, it must be noted that the personality theories discussed in German and Japanese psychopathology are typological-based understanding and differ in methodology from the Anglo-American trait-based understanding*. As evaluation scales for empirically examining melancholic personality, those created by Kasahara 21), von Zerssen, D. 50)70), and Stanghellini, G. et al. 57) are known. However, no specific association with endogenous depression has been demonstrated 12).

III. Difficulties in Demonstrating Endogenous Depression

Looking at the correlations with validators in this way, it may be difficult to empirically establish endogenous

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depression as independent of other depressive states. Of course, as Kendell 24) stated, significant differences in outcome and demographic data between X and Y patient populations do not immediately prove the existence of X, but even so, the diagnostic validity of endogenous depression is not encouraging. However, the author would like to consider why it is difficult to distinguish endogenous depression from other depressive syndromes in empirical studies from the standpoint of defending the binarian rather than easily giving in to the unitarian perspective.

1. Problems with DSM-5 Criteria for Melancholia

First, there is a problem with the diagnostic criteria. Four out of 8 items in DSM-5 criteria for melancholia overlap with the criteria for major depressive disorder itself. Therefore, there is an opinion that it is difficult to distinguish endogenous from non-endogenous depression as long as these criteria are used 45)49).

2. Distinct Quality of Symptoms

It has been repeatedly pointed out in traditional German psychopathology that the emotions of patients with endogenous depression are dissimilar to those of normal subjects. Tölle, R. 65), for example, states that the experience of patients with endogenous depression "has something that cannot be measured by the categories of normal psychology, and we cannot approach its center. Even the patients themselves have difficulty returning to the state they have overcome after their illness. ... It is an experience foreign and incomprehensible even to the patients themselves".

The DSM-5 criteria for melancholia also include a description of depressed mood of "distinct quality," but it is difficult to explicitly define an experience that patients themselves have trouble verbalizing. DSM notes are exclusionary, stating that "a depressive mood that is described as merely more severe, longer lasting or present without a reason is not considered distinct in quality" (DSM-5), and that they are "different from the kind of feeling experienced following the death of a loved one" (DSM-III). This "distinct quality" means, in essence, that the static understanding described by Jaspers, K. 17) is not possible. Even if a structured interview is used rigorously, it would be difficult for an evaluator other than an experienced clinician to determine whether or not it is of "distinct quality".

3. Holistic and Partial Perspectives

A holistic and intuitive understanding is essential in the evaluation of the

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patient's condition. This includes the emotional movement that occurs when the evaluator is confronted with the patient, the judgment of whether or not the evaluator can "understand" the patient, pathological evaluation based on the image of a typical example (prototype), and the typological understanding of personality, as described earlier 20). As Kasahara 19) stated, "Individual and enumerative merkmal cannot be extracted without the preceding holistic understanding," and so we should not only take into account the sum of partial symptoms in the checklist, but also the holistic and intuitive understanding in our diagnosis 4).

Although endogenous depression is said to be a more homogeneous group than major depressive disorder, this homogeneity is not judged solely on the basis of partial symptom items. If one considers only the partial viewpoint, the mathematical combination of symptom items required to diagnose melancholia in DSM-5 is approximately 340,000, leading to the paradoxical result that it is not more homogeneous than major depressive disorder, but rather 10 times more heterogeneous 11). It may be difficult to empirically distinguish endogenous depression as long as a rating scale for only partial symptoms is used.

IV. What Innovations are Needed?

Given these limitations, what methods can be used to demonstrate endogenous depression? Two attempts are presented.

1. Focus on Psychomotor Disturbances

One of the core features of endogenous depression is psychomotor disturbances (PMD), such as retardation and agitation 36)43)48)54)66). Recently, PMD was shown to be associated with a favorable response to electroconvulsive therapy 45)69). Since PMD is also almost the only quantitatively evaluable feature of the symptoms of endogenous depression, it has the potential to be a reasonable starting point for empirical studies 54). Parker, G. et al. developed the CORE scale 43)60) to evaluate PMD as a behavioral characteristic rather than a subjective complaint of the patient, and argued that many patients with endogenous depression diagnosed by conventional diagnostic criteria can be defined only by scores on the CORE scale 42). This suggests that the primary symptoms of endogenous depression are related to PMD 13). The authors 62) conducted a multivariate analysis of subjective symptoms of endogenous depression correlated with CORE scores in 106 patients with major depressive disorder, and found that the five symptomatological characteristics of: (1) feelings of having lost feeling, (2)

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depressive delusions, (3) perplexity, (4) indecisiveness, and (5) no aggression against others, correlated more favorably with CORE scores than the DSM-5 melancholia criteria.

2. Quantify Prototype Diagnosis

Another measure is an attempt to quantify the holistic and intuitive understanding: Parker et al. developed not only the CORE scale but also a rating scale to quantify the prototype diagnosis (Sydney Melancholia Prototype Index: SMPI) 44)46). The SMPI lists 12 characteristics of endogenous and non-endogenous depression, and after selecting the items that apply to each, the clinician is asked to rate the overall picture on a 5-point scale, indicating which type the patient is more likely to have. The classification of depression developed by Kasahara, Y. and Kimura, B. and popularized in Japan 18) was also a prototype diagnosis, and such an assessment method that deals with holistic understanding may also be useful.

Conclusion

The difficulty of empirically studying mental disorders is not limited to endogenous depression. The logical positivism that influenced the establishment of DSM-III 3)15)52) was based on the reductionism that both psychology and physics can be explained under a unified science. It is the assertion that "sociology can be reduced to psychology, psychology to biology, biology to chemistry, and chemistry to physics" 37). However, given the fact that mental symptoms are not objects like pimples or tumors, but "dialogical co-constructions" that are created by the evaluator's interpretation of the patient's language and behavior 4)30), it is obvious that the "demonstrability" of psychiatry is different from that of physics and chemistry, as well as from that of somatic medicine. However, this does not mean that empirical research in psychiatry is impossible. Rather than basing our research on naive reductionism, we need to define "demonstrability" at a level appropriate to each academic system 37).

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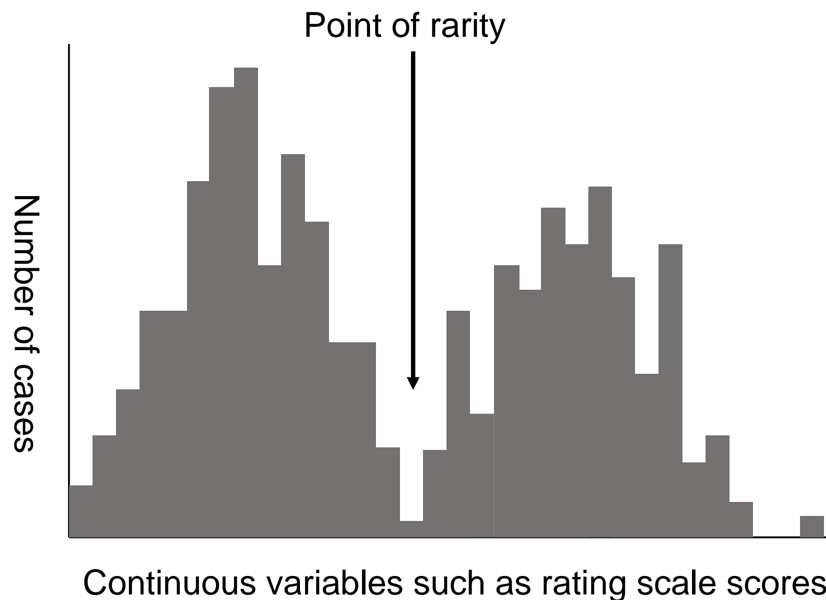

Figure: Example of bimodal distribution

Notes

*There are two theories of personality research: type and trait theory 35). Type theory is an intuitive and holistic approach to understanding the degree of similarity to typical types, and thus it is easy to grasp the image of "this is the kind of person". On the other hand, it has the disadvantage that it is impossible to classify cases that fall in the middle of each type: Kretschmer, E.'s cyclothymia 29), Shimoda's immodithymia 53), and Tellenbach, H.'s melancholic type 64) are examples of typologies. On the other hand, trait theory is a method to describe an individual's personality by the combination of each trait, considering the consistently appearing behavioral characteristics, or "traits," as units of personality structure. It is easy to verify empirically because it allows quantification and comparison among individuals, but it is difficult to gain an intuitive overall picture because the profile is fragmented. The mainstream of contemporary personality research is the trait theory.

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