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Original Article

Past History and Future Perspectives of the Traditional Japanese Depressive Disease Concept: From Mild Endogenous Depression to Major Depressive Disorder

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Abstract

In the Japanese edition of DSM-5 published in 2014, the translation of major depressive disorder (MDD) was changed from "Dai-utsubyo-sei-shogai" to "Utsubyo". However, in Japanese psychiatric clinics, a unique traditional concept of "utsubyo" that is not related to the DSM was still alive until around 2000. I will refer to this as Nippon Depression, which is the source of the maxim, "Don't encourage people with depressive disease." Many people come to the outpatient clinic with complaints of depression, anxiety, physical discomfort, fatigue, and insomnia related to stress in daily life. Horwitz refers to these as the stress traditions. However, there is a group of people who appear to be in this stress tradition, but have their own medical condition, course, and response to treatment. This group shares characteristics with severe manic-depressive disease that requires admission to a psychiatric hospital. This clinical unit is worthy of differential diagnosis as Nippon Depression. The characteristics of Nippon Depression are as follows. Conscientious and tidy office workers and housewives are affected by changes in their living environment, such as moving to a new house or getting a promotion. The main symptoms are a feeling of gloom and worthlessness, loss of feelings of pleasure and sadness, retardation and inhibition of thoughts (i. e., inability to think clearly and make decisions, fatigue, exhaustion), and a vague feeling of ill health, as well as

insomnia and loss of appetite. These are accompanied by diurnal fluctuations. These symptoms appear to be the stress-reactive depression that we all experience in our daily lives. However, although they appear to be empathic, in reality, emotional interactions are rejected. It is an uncanny experience beyond our imagination. It also takes the course of autonomy. It does not get better even when the person is relieved of stress. However, as a rule, it is mild and can be treated on an outpatient basis, as long as one pays attention to the risk of suicide. Patients get better with rest and antidepressants. Thus, the maxim that people with this condition should not be encouraged. Although it is named Nippon Depression, this type of disease itself has been reported overseas since the late 19th century. Later, in post-World War II West Germany, it became clear that there was a non-negligible number of cases of endogenous depression after the persistent stressful conditions of wartime destruction and postwar confusion. In Switzerland, Kielholz reported a number of exhaustive depressions with an endogenous course beginning with a stress reaction due to occupational exhaustion. He noted that antidepressants are effective for this condition and cautioned that encouragement of the patient is useless and often harmful. The psychopathological research results were first introduced to Japan in 1959. Thereafter, mild endogenous depression was released from its connection with the scars of war defeat and linked to the exhaustion of working businesspeople. Finally, it was termed "Utsubyo" without qualifiers, i. e., Nippon Depression. In the same year, antidepressants became available on the market. Nippon Depression was highlighted and treatment with antidepressants was promoted, and by 1975 it was established as "depression with melancholic personality type". Small psychotherapy and the maxim "Don't encourage people with depressive disease" were included as treatments in textbooks from 1983 onward. However, when serotonin reuptake inhibitors (SSRIs) became available in 1999, the concept of "Utsubyo" was updated. SSRIs were indicated for MDD, not Nippon Depression. The stress tradition was incorporated into "Utsubyo". In 2014, the Japanese term "Utsubyo" was changed to be equivalent to MDD. Here, the Japanese traditional concept of Nippon Depression was considered to be out-of-date. However, we should note that the diagnostic criteria for MDD are not expected to be valid as a biomedical model. It would be more fruitful for future biomedical or therapeutic research on depression-related conditions to use Nippon Depression as a starting point instead of MDD.

Keywords: depression, endogenous depression, exhaustion, melancholia, stress

Introduction

The DSM-5 Japanese edition⁷⁾ published in 2014 changed the translation of major depressive disorder (MDD)⁸⁵⁾ from "大うつ病性障害 (dai-utsu-byo-sei-shogai)" to "うつ病 (utsu-byo)". The exact translation is "うつ病 (DSM-5)/ 大うつ病性障害". However, because of the complexity, "DSM-5" and "major depressive disorder" are omitted except in the headings and in the first occurrence. The term "うつ病," i.e., MDD, is used instead.

On the other hand, until around 2000, the traditional concept of "うつ病"¹³¹⁾, which was not related to DSM, remained alive in Japanese clinical psychiatry. It is "うつ病" without modifiers. No equivalent or interchangeable words exist in other countries. This is a depiction of mild endogenous depression¹³⁸⁾ against the background of Japanese psychiatry in the late 20th century. It is also referred to as "軽症うつ病"³⁰⁾⁴⁹⁾.

In recent years, it has been referred to as "prototypical depression"⁴²⁾, "core group of depression"¹¹⁴⁾¹¹⁵⁾, and "real depression"⁷⁷⁾ *1, and in such cases, to avoid confusion with MDD, it is labeled "内因性うつ病"²⁷⁾⁴²⁾⁷³⁾⁷⁷⁾ and "メランコリー (親和)型うつ病 melancholic type depression"⁹⁹⁾¹¹⁴⁾¹¹⁵⁾. However, these terms often lead to misunderstanding and confusion *2, so I will refer to this

uniquely Japanese traditional "うつ病" as "Nippon Depression" (Nippon no Utsushoku) without brackets⁸⁹⁾⁹⁴⁾⁹⁵⁾.

From the late 19th century to the present, neurologists and general family physicians in outpatient clinics have seen many people complaining of depression, anxiety, physical discomfort, fatigue, and insomnia related to stress in their daily lives. In the past, such people were diagnosed with neurasthenia, nervousness, neurosis, and in recent years, chronic fatigue syndrome, and in Japan, neurosis and dysthymia. The current diagnosis is MDD. Horwitz, A.V.³⁴⁾³⁵⁾ refers to these as the stress tradition.

There is a group of patients who appear to be part of this stress tradition, but who have their own unique symptoms, course, and treatment responsiveness. This group shares characteristics with severe manic-depressive patients who need to be admitted to a psychiatric hospital. This clinical unit is important for differential diagnosis. In short, manic-depressive illness that appears to be stress tradition but is actually a mild manic-depressive illness that does not require hospitalization is Nippon Depression⁹⁴⁾.

Until around 2000, Nippon Depression was characterized as follows⁹³⁾⁹⁴⁾⁹⁴⁾: The main symptom is a depressed mood in which everything is gloomy and boring,

loss of feelings of pleasure and sadness, thought congestion and inability to think clearly and make decisions, fatigue, exhaustion, and vague physical discomforts, as well as insomnia and loss of appetite and diurnal fluctuations are also present, which occurs in orderly and sensible office workers and housewives after a change in living environment, such as moving to a new address or a promotion. These appear to be the stress-responsive depression that we all experience in our daily lives. However, the patient appears to be empathetic, but in reality, emotional interactions are rejected. It is an experience that we cannot imagine. The patient also undergoes a period of autonomy. It does not get better even when the patient is relieved of stress. As a rule, however, the disease is mild and can be treated on an outpatient basis as long as attention is paid to the risk of suicide. Rest and antidepressant medications help the patients to get better. Also, do not encourage these people.

This concept of Nippon Depression began in 1959²⁸⁾, was established in an academic journal in 1975⁴⁵⁾, and was described in a textbook in 1983⁸³⁾. It is the source of the proverb "Do not encourage the depressed person." However, since around 2000, the general habit of referring to all stress traditions as “うつ病” has spread in

Japanese everyday language⁹⁴⁾. This custom has also been adopted in the realm of official academic terminology since 2014⁷⁾⁹⁰⁾. Here, Nippon Depression lost its function as a "prototype of depression," a "core group of depression," and "real depression." This article discusses the beginning and end of this process, as well as the future of the forgotten concept of Nippon Depression.

I. Comparison of MDD and Nippon Depression

1. The Four Types of Depression

“うつ病” has at least four meanings. The classification is based on Hamilton, M.²³⁾²⁴⁾, and the headings A0, A1, A2, and A3 are assigned according to the DSM-5 diagnostic criteria⁷⁾. A1 and A2 of DSM-5 include the words "almost every day of the week." The definition of severity³⁰⁾ differs from that of DSM-5, where "mildest" means no medical intervention is required, "light" means outpatient treatment, and "mild" means inpatient treatment in a psychiatric hospital.

A0. Mildest: depression/sadness without disorder

A1. Mild: depression/sadness with disorder

A2. Mild: loss of pleasure, mood non-reactivity (anhedonia), inability to mourn, vital sadness

A3. Severe: agitation, stupor/catatonia, delusions (guilt, poverty, hypochondria)

A0 and A1 are empathically transferable experiences as an extension of normal psychology, and both occur following a variety of stressful events, including experiences of loss. Mood reactivity is preserved and can be exacerbated or improved by external influences. Thus, psychotherapy can be effective. A1 may be more severe or prolonged than A0, and in clinical practice, it is judged by whether it is accompanied by significant distress or dysfunction (Diagnostic Criterion B for MDD).

Unlike A0 and A1, A2 is a difficult experience to empathize with. The association with stressful events is unclear. Also, mood reactivity is lost and is neither exacerbated nor improved by external influences. Therefore, psychotherapy has limited efficacy. On the other hand, this symptom has attracted attention as a sign that antidepressants may be effective⁵⁹⁾. Even before that, the German theory of endogenous depression had been emphasizing the importance of this character. It does not improve in response to pleasant events, nor does it worsen in response to sad events [Inability-to-mourn (Nichttraurigkeit) in Schulte,

W.]¹¹¹⁾¹¹²⁾. Depression then invades not only the psychological layer but also the physical layer. Specifically, the patient complains of internal restlessness and agitation rooted in the chest, or a feeling of pressure in the chest and stomach [Schneider, K.¹⁰⁶⁾¹⁰⁹⁾, vital sadness (vitale Traurigkeit)]. The patient is aware of the fact that his/her mood is different from normal experience. Therefore, they maintain an awareness of illness¹⁰¹⁾¹⁰⁹⁾¹⁴¹⁾.

A1 and A2 are in conflict in that mood reactivity is "preserved/lost" and are theoretically incompatible. However, not infrequently, patients experiencing A2 complain of A1. This experience, however, is similar to but different from the depression/sadness they experienced when they were healthy. It is depression/sadness as a metaphor²⁴⁾¹¹²⁾. When asked about this matter, the patient usually agree. The diagnostic criterion B1 "Distinct quality of depressed mood" in DSM-5 "Features of Melancholia"⁷⁾¹²⁾ refers to this state of affairs.

A3 is the core image of manic-depressive depression described by Kraepelin, E.⁶¹⁾⁶²⁾. It often interferes with life-sustaining functions and lacks a sense of illness.

2. Two Ds: Difference between "disease" and "disorder"

"Disease" and "disease" are

distinguished here:

D1. disease: Presence or a strong demand for the presence of a physical or medical lesion.

D2. disorder: Clinically significant disturbance, associated with significant distress or impairment.

D1 is based on the biomedical model¹³⁾²⁰⁾¹⁰²⁾¹⁴⁶⁾ represented by Robins, E., Guze, S.B., Winokur, G., and other so-called neo-Kraepelinians⁶⁰⁾.

DSM-III⁴⁾ adopted D2, advocating atheoretical, partly due to political considerations. It was designed to accommodate not only a biomedical model but also a psychobiological⁷⁴⁾ or biopsychosocial model^{16),19) 126) *3)}.

However, neo-Kraepelinians, especially Guze¹³⁾²⁰⁾, equate D1 and D2 and attempt to incorporate DSM into the biomedical model [the disorder-as-disease method¹²⁴⁾ *4].

In fact, DSM-5 loosened the criteria for distress and dysfunction and abolished the multi-axial diagnosis. Here, Cooper, R.¹⁵⁾ admits a bias toward the biomedical model, and after DSM-IV⁶⁾, the term "irrationality" had already been deleted*5.

3. Summary of for types of depression and "disease/disorder" in Nippon Depression and MDD

A2 is essential in Nippon Depression*6

*7. If A2 is not confirmed, then "a distinct quality (of depressed mood)⁷⁾¹²⁾ must be found in A1." If A1 only exhibits the usual quality, it is diagnosed as a depressive reaction (including bereavement reaction), depressive neurosis (including mood modulation disorder), and various other neuroses or depressive personalities*8*9. In other words, it is a stress tradition.

On the other hand, MDD requires either A1 or A2; according to Spitzer, R. L. et al.¹²⁵⁾, MDD includes the depressive form of DSM-II manic-depressive illness, psychotic depressive reactions, and various states of depressive neurosis.

In summary, Nippon Depression is D1, which represents A2, and MDD is D2, which represents A1 or A2.

In severe cases, Nippon Depression and MDD are almost equal. Both have A3 as the primary symptom, and severe cases have both D1 and D2, i.e., they are both diseases and disorders.

II. From the United States after the Civil War to Europe before World War I: Neurasthenia and Mild Endogenous Depression

Mild endogenous depression, which is at the core of the disease type I have named "Nippon Depression," has been reported internationally since the end of the 19th century.

1. Beard, G.M. Neurasthenia: The Beginning of the Stress Tradition

The stress tradition began in the latter half of the 19th century with neurasthenia, which was advocated by Beard⁽⁸⁹⁾, a neurologist in New York in 1869, and quickly spread to European countries such as France, Germany, and the U.K.⁽⁸⁸⁾. In large cities in developed countries, workers were unable to cope with the rapid development of technological civilization and changing lifestyles, as if they were machines that had run out of charge. If blood deficiency is anemia, neurasthenia is the depletion of the vital force of nerves.

Neurasthenia included the early stages of progressive paralysis, for which the pathophysiology was not yet clear, as well as thyroid dysfunction and collagen disease. However, the most common form of neurasthenia was a physical and mental disorder in the absence of any physical medical lesion. To restore the vital force of nerves, DC electrical stimulation therapy using a generator was initially applied. Later, rest cure and psychotherapy became the standard treatment.

2. Lange, C. Periodical depression: Symptoms, Course, and Treatment of Mild Endogenous Depression

In 1886, the Danish neurologist Carl Lange⁽⁶⁵⁾ identified a clinical unit from a diverse set of neurasthenia. This was

periodical depression (periodiske depressionstilstande). It was the first report of a clinical unit that formed the core of Nippon Depression, i.e., mild endogenous depression.

Periodical depression was "crammed into the poorly defined little room of so-called neurasthenia." Characteristic symptoms were "a loss of vivacity and joie de vivre due to a sense that everything is rigid" and "no other person, event, or environment can evoke any warmth in the patient, which is their most painful complaint." The loss of pleasure and non-reactivity of mood in A2 are clearly captured here. In addition, fatigue, psychomotor inhibition, loss of interest, difficulty making decisions, physical symptoms (headache, dizziness, back pain, abdominal discomfort, etc.), and autonomic symptoms (weight loss, weakness, cold extremities, abnormal pulse, sleep disturbances, especially early morning awakenings) are also present with diurnal fluctuations.

It has nothing to do with melancholia. Unlike melancholia, periodical depression has a cyclical and repetitive course. The main psychiatric symptom is an unmotivated mood change, and patients do not manifest A3. They do not become hallucinatory or delusional, nor do they require hospitalization. They are not suicidal.

The mainstay of treatment is physical

therapy based on the etiology. Since the cause is uric acid, dietary therapy is used to reduce uric acid intake, and drug therapy (mainly lithium) ^{*10} is used to stimulate uric acid excretion. The effectiveness of psychotherapy is questioned, as in the statement: "It is unfortunate that the patient is treated from a psychological point of view."

Lange, C.'s treatment instructions were also harsh:

"Patients, unwilling to be miserable, try to retreat from company and daily obligations, but they must not be allowed to do so"; "The inactive nervous system needs mental stimulation"; "Therefore, as far as possible, the patient should be continually exposed to strong and changing stimuli." Perhaps the intention here was to highlight the difference from other forms of therapy for neurasthenia.

Rewrite Lange, C.'s statement "No suicide, no rest in treatment" as "High suicide risk, rest in treatment," add an orderly personality, the effects of antidepressants, and the maxim "Do not encourage," and you have Nippon Depression.

3. From Periodical Depression to Mild Manic Depression: Status of Mild Endogenous Depression

Clinical units corresponding to mild endogenous depression were reported by psychiatrists before and after

Lange, C.: Kahlbaum, K.'s *Vecordia dysthymia* (1863)⁴⁰, *cyklische Irresein* (1882)⁴¹, Hecker, E.'s *Cyclothymie* or *Circular Mood Disorder* (*cirkuläre Gemüths-erkrankung*) (1877, 1898)²⁵⁾²⁶⁾, and Ziehen, T.'s *Hypomelancholie* (1896)¹⁴⁸. Unlike Lange, C., psychiatrists saw in this clinical unit the essence of severe melancholia and later manic-depression. The suicide risk is also high.

Kraepelin modified the periodic psychosis (*periodische Irresein*) in the fifth edition of his textbook (1896)⁶¹ and presented manic-depressive psychosis (*manisch-depressive Irresein*) in the sixth edition (1899)⁶². Following this, Wilmanns, K. (1906)¹⁴³ positioned Hecker's cyclothymia as a mild form of manic-depressive illness. Bonhoeffer, K. (1912)¹⁰ called this form endogenous depression. This is a mild form of depression seen in the outpatient setting by general and family physicians, and is often diagnosed as a neurasthenia. Endogenous depression must be differentiated from neurasthenia in order to accurately make a prognosis and be aware of the suicide risk.

III. Britain Between the World Wars: Psychotherapy and Physical Treatment for Mild Endogenous Depression

German medicine emphasizes etiological hypotheses and the nature of

psychopathology in diagnosis. British medicine, on the other hand, emphasizes the diagnosis as an indicator to determine the course of treatment. In contrast to German medicine, which pursues absolute validity, British medicine pursues utility of treatment on a case-by-case basis.

1. Psychotherapy Guidelines and the Sporadic Appearance of "Don't Encourage"

In 1923, Ross, T.A.¹⁰⁴, a British general practitioner, wrote in a textbook on psychotherapy: "The two conditions, the depressive phase of manic-depressive psychosis and neurasthenia, are often intermingled, but the principles of treatment in these diseases are quite different. Therefore, the differential diagnosis between psychosis (the author's note: mild endogenous depression, same hereafter) and neurasthenia is very important." He added: "If we are certain that the patient was voluntarily engaged in excessive work prior to depression, then we have a strong suspicion of psychosis. This overwork must be 'reliable and trustworthy' information from those around him. Too often, the person with a nervous breakdown cites overwork as the cause of his or her collapse, but these stories do not hold up to close scrutiny." This statement holds true

even today.

Ross stated that: "the person with a nervous breakdown should be encouraged to do more than he thinks he can do," and that "on the other hand, it is harmful to ask more of the mentally ill than he feels he can do." This is the beginning of the so-called "don't encourage" or prohibition against encouragement. However, there is no evidence that this textbook was ever referred to in Japan.

2. Guidelines for Electroconvulsive Therapy

Electroconvulsive therapy (ECT) became widespread around 1940¹²³. ECT was effective for a variety of conditions, and was particularly effective for catatonia and severe depression of A3.

In 1944, Cook, L.C.¹⁴ wrote in a review of ECT that: "many of the successful cases of convulsive treatment of psychoneuroses may be reports of essential resolution toward depression masquerading as neurotic anxiety," and Sargant, W. et al.¹⁰⁵ wrote in a textbook of psychiatric somatic treatment that: "The only 'anxiety neurosis' patients who could benefit from the cramps were those who had been mistakenly called neurotic. They were, in fact, suffering from depression foregrounded by anxiety and agitation." In principle, ECT is effective for mild A2 as well as

severe A3. However, it is not effective for neurotic A1. Therefore, if ECT is effective for mild depression, it should be considered as mild endogenous depression (A2), and not neurosis (A1).

3. Reorganization of Differential Diagnosis Based on Treatment Responsiveness

From this point on, a trend toward re-evaluating the diagnosis based on treatment responsiveness emerged⁸⁴. ECT is considered a diagnostic as well as a therapeutic tool in this context. Of course, the roles of therapeutic and diagnostic tools are different. However, the method of diagnostic classification based on treatment responsiveness, rather than etiology or pathogenesis, had a major impact on later psychiatry.

In fact, Watts, C.A.H. (1956)¹³⁷, a general practitioner, based his diagnosis of endogenous depression on the fact that ECT is necessary for cure the illness and that psychotherapy (in a cause-seeking manner) provides no help. However, he was not a psychiatrist and did not use ECT. There were also no antidepressants at that time.

Nevertheless, Watts (1957)¹³⁸ achieved effective treatment results for mild endogenous depression using symptomatic treatment such as barbiturates, amphetamines, and light work based on a good rapport with patients. A diagnosis of mild

endogenous depression is useful even in the absence of specific medications, as it provides an appropriate understanding of the disease and facilitates a prognosis.

IV. German Research on Mild Endogenous Depression: Origin of the Concept of Nippon Depression

The direct origin of the Japanese concept of depression can be traced to studies of mild endogenous depression in West Germany and Switzerland after World War II. The first signs of this trend can already be seen after World War I.

1. Post-World War I - War Aftereffects and the Vitalization of Reactive Depression-

In Germany, interest in neurasthenia waned after the outbreak of World War I. Instead, an urgent problem emerged after the war: people suffering from the psychological aftereffects of the war experience.

According to Schneider (1920)¹⁰⁶, many soldiers who served in World War II continued to suffer from vital depression i.e. a disturbance of vital feelings (A2), even after the "suffering" that caused their initial depression (A1) disappeared after their discharge from the military. This is called vitalization (Vitalisierung) of Reactive Depression). In this case, the content and theme of the initial suffering are lost. The

semantic continuity, semantic relatedness, and semantic law of the experience are torn away¹⁰⁷⁾¹⁰⁹⁾. In this sense, A1 and A2 are dissimilar experiences and must be distinguished.

According to Johannes Lange (a different person unrelated to Carl Lange mentioned above) (1926)⁶⁶⁾, the personality traits of those who develop A2 mild endogenous depression tend to be obsessive, hard-working, orderly, conscientious etc., rather than the syntonic and cyclothymic personalities typical of those who develop manic-depressive illness (which represents A3). This point of view is consistent with Nippon Depression.

Furthermore, Lange, J. (1928)⁶⁷⁾ proposed the pathogenic mechanism hypothesis whereby an unknown genetic and constitutional vegetative neuro-endocrine instability acts as a preparatory factor for onset of the illness during the vitalization of reactive depression, i.e., the transition from A1 to A2. This hypothesis predates the stress hypothesis of Selye, H.¹¹³⁾ by more than 20 years^{*12)}.

2. Post-World War II - Situational Theory of Depression

In the 1950s, West Germany, like Japan, still bore the scars of World War II. There, it became clear that after the sustained stress of the destruction during the war and chaos of the postwar

period, the number of cases of A2 (rather than A1) endogenous depression, which was mild but chronic in nature, was too large to be ignored³⁰⁾³⁷⁾³⁸⁾.

Furthermore, in West Germany at that time, "one of the great social demands placed on psychiatrists, in terms of liquidation for the wrongs committed by the Nazis, was the issue of psychiatric evaluation for compensation to Nazi persecuted persons."³⁶⁾³⁸⁾ There, psychiatrists were confronted with the undeniable fact that "the principle of 'endogenous' depression, which occurs spontaneously regardless of external events, develops 'reactively' in response to events such as persecution and loss," and were asked to clarify the true nature of this fact⁹¹⁾.

Weitbrecht, H.J.¹³⁹⁾¹⁴⁰⁾ reported a series of clinical units that followed the course of endogenous depression beginning with reactive depression after 1949. The final name was endogenous reactive dysthymia (1952)¹⁰¹⁾¹⁴¹⁾. By 1951, after the war, it was most common among refugees from East Germany who entered West Germany²⁸⁾. It is a mild endogenous depression that starts as reactive depression (A1), but becomes vitalized secondarily, i.e., shifts to vital sadness (A2). It differs from the core (A3) of Zykllothymie (Schneider and Weitbrecht refer to manic-depressive illness as such), but there is undoubtedly a disease (D1), i.e., a

somatomedical lesion. It is not an extension of a normal psychological reaction (A1). The premorbid personality is not syntonetic and the genetic predisposition to manic-depressive illness is rare. He does not switch to manic, and there are no episodes of mania in his medical or family history. There are no primary guilt feelings (Weitbrecht's term for delusions of guilt).

According to Pauleikhoff, B. (1958, 1959)⁹⁶⁾⁹⁷⁾, the course of endogenous depression beginning with reaction is not only from A1 to A2; there are cases in which A2 develops suddenly, skipping A1. This has not been noted since Lange, J.⁶⁶⁾ Pauleikhoff reconsidered the relationship between personality and situation in the onset of illness. When people move, get a promotion, give birth, or their children marry, the external structure of the environment changes, and the order of the individual's being is shaken. In such cases, if the internal structure of the personality cannot be transformed in a resourceful manner, A2 will develop.

Other researchers focused their attention on this area: unrooting depression (Entwurzelningsdepression) (1950)¹¹⁾, burden-reduction depression (Entlastungsdepression) (1951)¹¹⁰⁾, existential depression (existentielle Depression) (1954, 1958)²²⁾¹⁴⁴⁾, and loss depression (Verlustdepression) (1959)⁶⁹⁾.

These were collectively called the situational studies of depression³⁷⁾³⁸⁾.

3. Possibility of Psychotherapy for Mild Endogenous Depression

According to the conventional wisdom of psychiatry at that time, psychotherapy did not cure manic-depressive illness. It was ineffective against depression as well as mania. In 1954, Schneider¹⁰⁸⁾ stated that: "psychotherapy can have at best only a momentary superficial sedative effect on Zykllothymie, and even then only in mild cases" and "if psychotherapy was reported to be effective in Zykllothymie, it was either because the patient did not have Zykllothymie, or because the therapist was coincidentally present at spontaneous remission of the illness."

However, might psychotherapy have a role to play in mild endogenous depression? Schneider's rival Kretschmer, E., and his students responded. Häfner, H. (1954)²²⁾ in his report on existential depression, the onset process of vital depression using Kretschmer's concepts of premorbid personality, value structure, and key experiences. This is, so to speak, a depression version of "sensitive delusions of reference." Winkler, W.T. (1958)¹⁴⁴⁾ argued that non-complex (purely genetic or constitutional) depression can be treated by ECT alone, but for complex (highly situational)

depression such as existential depression, the Häfnerian interpretation creates a bridge of contact with the patient. According to Winkler, psychotherapy for mild endogenous depression is not a causal therapy, but it does provide guidelines for attitudes and postures to approach the patient while providing physical treatment. Physical treatment and psychotherapy are complementary in the treatment of mild endogenous depression.

All that remained was to discover a physical treatment that was simpler than ECT and appropriate for mild outpatient cases.

4. The End of Studies of Situation and Premorbid Personalities of Mild Endogenous Depression

In his book "Melancholy" (1961)¹³⁵⁾, Tellenbach, H., attempted to summarize research on the situation theory of depression. He called the anthropological type that constitutes the pathogenic situation of endogenous depression the melancholy type. The basic characteristic of the melancholy type is love-of-order (Ordnungsliebe). This orderliness is manifested in work, attitude, and conscience as exactness. In interpersonal relationships, patients exist for the sake of others (Sein-für-andere) and respect social common sense and duty. Later, in the context of

Japanese clinical practice, the essential and fundamental characteristic of this melancholy type shifted from love-of-order to "maintenance of amicable relationships with others"⁴⁵⁾, which became established as the premorbid personality of Nippon Depression⁸⁶⁾. It is also called a melancholia affinity-type personality⁴⁵⁾.

After Tellenbach, research on mild endogenous depression and its situation theory in West Germany came to an end⁸⁶⁾. Behind this was the passage of the Federal Compensation Law Termination Act of 1965. Also, the discovery of antidepressants was a major factor. Janzarik, W.³⁹⁾ explains as follows: "The triumph of treatment empiricism, opened up by the new pharmacotherapy, over the previous royal road of research and interpretation of the nature of mental disorders, led to the decline of psychopathology." In other words, once the treatment method had been established, there was no longer any need to question the nature of psychopathology. German essentialism was giving way to Anglo-American pragmatism."

V. Switzerland in the Late 1950s: Exhaustive Depression, Imipramine, and "Do not Encourage"

1. Kielholz's exhaustive depression and Weitbrecht's endo-reactive dysthymia

and stress tradition

The setting goes back to the late 1950s in Switzerland. In 1957, Kielholz, P.⁵³⁾ named a depressive condition that began as reactive and exhibited an endogenous course as exhaustive depression (Erschöpfungsdepression). The symptoms and course of depression are very similar to those of endo-reactive dysthymia⁶⁴⁾. Both conditions cause prolonged psychosomatic and autonomic symptoms under prolonged stress and tension, and symptoms become animated. The patient feels chest tightness around the heart. There is no primary guilt feeling. The symptom transitions from A0 to A1 and then to A2, but never to A3. There is no mania. Both forms are mild endogenous depression.

However, the sociocultural aspects are different. The annotations of exhaustive depression describe it as an asthenic form of managerial illness (Managerkrankheit)⁵³⁾. Typical patients with endo-reactive dysthymia were those suffering from postwar scars. In contrast, typical examples of consumptive depression are exhausted and worn-out business people, especially workers who have to use their brains^{*13)}. The disease is precipitated by excessive workload, sudden changes to busy and noisy work, and responsible decisions made under time pressure, especially those that require immediate

solutions in a rushed, tense, and stressful environment, or that require making the right decision in the face of uncertainty and doubt. Under such circumstances, the following phases are observed: (1) the irritable-asthenic prodromal phase, followed by (2) the psychosomatic symptom phase, and finally (3) the depressive phase. This may be a reference to the stress reaction process in Selye's¹¹³⁾ general adaptation syndrome, i.e., (1) the warning phase, (2) the resistance phase, and (3) the exhaustion phase. Exhaustion (Erschöpfung) is one of the key concepts in Selye's theory and stress tradition. Research on mild endogenous depression and its situation studies, which started in and around defeated Germany, met the stress tradition from the victorious U.S. in neutral Switzerland.

2. Discovery of Imipramine

The recommended physical treatment for depression was a combination of insulin shock and 100 to 200 mg of chlorpromazine, with adequate rest⁵³⁾. However, in 1957, the same year as Kielholz's report, Kuhn, R.⁶³⁾ reported a new drug treatment for depression in the same Swiss Medical Weekly. It was G22355, one of the iminodibenzyl derivatives, which later became the antidepressant imipramine. Kuhn's patients were inpatients, and he found

that imipramine was effective not only in treating endogenous depression, but also in treating morbid guilt and poverty ideation. Kuhn's target group was mainly those with A3 severe manic-depressive depression.

How about imipramine for mild endogenous depression with A2 symptoms such as exhaustive depression? In 1958, Kielholz⁵⁴⁾ responded to this question. Again, it was the Swiss Medical Weekly. According to the response, conventional chlorpromazine, reserpine, and meprobamate sedate and relieve anxiety and internal disturbances associated with depression, while stimulant amines (such as amphetamine), some antihistamines, and iproniazid (MAO inhibitor) increase the desire for psychomotor inhibition. However, until then, there were no drugs that were effective for the treatment of vital depression. Since then, imipramine has emerged. The most optimal indications are outpatient exhaustive depression, which is foregrounded by inhibition and vital symptoms, and mild episodic or cyclic melancholia. It is also effective for inpatient cyclothymic melancholy, but is a step behind ECT. In summary, imipramine is effective for all three components of depressive symptoms: (1) vital sadness and anhedonia, (2) thought inhibition, and (3) life of drive

and will. Thus, the efficacy of imipramine for mild endogenous depression is supported.

3. Reappearance of "Do not Encourage"

In 1959, Kielholz⁵⁵⁾ published a pamphlet entitled: "Diagnosis and Treatment of the Depressive States" from Geigy, the company that marketed imipramine. There, he says, "Advice of good intentions from relatives and others, such as, 'Pull yourself together and summon up your courage,' or 'Make your way in life through this difficulty,' can drive the patient to despair (Verzweiflung), leave him at a loss with anxiety, and increase the risk of suicide. We must not forget that." Other harmful responses that can lead to suicide include the admonition to "get a grip, you can do anything if you have the will," and the imposition of fruitless and painful recreation.

Also in West Germany, Schulte¹¹²⁾ in 1962 cautioned against encouraging patients when discussing psychotherapeutic manners for mild endogenous depression. Eight years after Schneider's reminder of the inappropriateness of psychotherapy for Zykllothymia, and in the same German Medical Weekly, Schulte cautioned that: "the patient must be protected from the thoughtless assertion that he can understand 'verstehen' the states of manic-depressive illness on the

emotional scale of a healthy person's sadness-gladness". Therefore, he cautioned: "All false attempts to induce, encourage (ermuntern), or demand too much of the patient must be prohibited."

4. Advances in the Theory of Treatment of Mild Endogenous Depression

Unlike ECT, antidepressant therapy can be easily administered in an outpatient setting, making it ideal for mild endogenous depression. Furthermore, the widespread use of antidepressants has clarified the role and direction of psychotherapy. Psychotherapy does not by itself produce relief. However, understanding the psychopathology of mild endogenous depression itself provides a guideline for psychotherapeutic attitudes and approaches to the patient. The statement that encouraging the patient is not only futile, but often harmful, holds true.

VI. Japan from 1959 to the 1990s: Establishment of the Concept of Depression and Minor Psychotherapy in Japan

1. Japan before Nippon Depression

There were original and pioneering studies in Japan.

One was Shimoda's theory of the immobilithymia and pathogenic process. In 1929, Shimoda identified paranoid personality as a premorbid trait of

manic-depressive illness¹³⁰. This was later called immobilithymia. This was considered a key concept in differentiating neurasthenia from endogenous depression (or depression of upper-middle age in the original work). Even if the chief complaint is the same exhaustion and depression, those with introversion are more likely to have neurasthenia, while those with immobilithymia are more likely to have endogenous depression.

Shimoda (1948, 1950)¹¹⁷⁾¹¹⁸⁾ later discussed the influence of an immobilithymia on the onset of illness. First: (1) overwork (triggering circumstances), followed by (2) a neurasthenic reaction (sleep disturbance, increased fatigability), after which normal persons naturally enter a state of rest due to decreased emotional excitability and loss of desire for activity, while persons with an immobilithymia, due to their constitutionally abnormal emotional excitability, continue their activities, resisting fatigue (3) and become increasingly overworked, and at the peak of this exhaustion, many suddenly develop (4) manic syndrome or depressive syndrome. These theories of premorbid personality and illness status preceded those of Kielholz and Tellenbach.

In 1946, Uchimura¹³⁶⁾ reported on depression after World War II, arguing

that: "The depression I am describing here is one of the manic-depressive illness, ...and this type of illness seems to be increasing markedly, especially with the severe patient's surrounding changes caused by the war and the difficulties in the social environment." This point was made before Weitbrecht in West Germany. This is the first report to use the Japanese Kanji characters for "鬱病 (utsu-byo, depression)" and "鬱病 (utsu-byo, depressive illness)" ("鬱 (utsu)" in the title and text "鬱 (utsu)" (those in the original paper have a difference in spelling). The term "うつ病" did not exist in Japan before the 1946 report.

2. From Mild Endogenous Depression to Nippon Depression

In Japan, the term "neurosis" became popular in both medical diagnosis and everyday language in the late 1950s¹³²⁾. This is the stress tradition mentioned in the introduction. Around that time, differential diagnosis between neuroses and manic-depressive illness/depression began to attract attention⁷⁵⁾⁸⁰⁾⁸¹⁾¹²⁸⁾.

Around 1960, new psychopathology and psychopharmacology were introduced to Japan from West Germany and Switzerland. This was a time when the despondency of a defeated nation was being replaced by the uplift of a period of rapid economic

growth. The mild endogenous depression was released from its association with the scars of defeat, and became linked with the exhaustion and weariness of working people. This was the beginning of Japan's own traditional "うつ病" or Nippon Depression.

Hirasawa, who had studied under Weitbrecht in the late 1950s, returned to Japan and introduced the most advanced research on mild endogenous depression to Japan. The term "うつ病 (utsu-byo, depression)" in hiragana became popularized from his review article: "On the present stage (1945-1958) in research of depression from clinical psychiatry", published in the April issue of *Clinical Psychiatry* in 1959^{28)*14)}. Coincidentally, imipramine was launched in Japan in May 1959. Since then, the concept of depression has been established as a depressive condition for which tricyclic antidepressants (TCAs) such as imipramine are effective. The concept of Nippon Depression was clarified by pairing it with TCAs. Furthermore, at the same time that Hirasawa²⁹⁾ re-evaluated Shimoda's theory of immobilithymia, Tellenbach published his theory of melancholy¹³⁵⁾. The concept of Nippon Depression was refined by adding premorbid personality theory and illness onset situation theory to the symptoms, course, and treatment responsiveness³⁰⁾.

In 1975, Kasahara and Kimura's clinical classification of depression⁴⁵⁾ downgraded severe depressive states of manic-depressive illness, which had long been considered the classic and core form of depression since Kraepelin, to Type II, and mild endogenous depression of "melancholic personality type depression" or "personality (reactive) type depression," which was still new at that time, was placed in Type I. This was the establishment of the Japanese concept of depression. Here, the core of depression in clinical psychiatry shifted from A3 (severe depression) to A2 (mild depression).

3. Establishment of "Do not encourage" or Contraindication to Encouragement

In 1978, Kasahara⁴⁶⁾ presented the "Seven Principles for the Treatment of Depression in the Acute Phase,"¹⁵⁾ which were widely used in Japanese psychiatric practice as the "Seven Articles of Oral Communication for Depression,"⁴⁷⁾ or "Small Psychotherapy." In addition, in 1982, Shinfuku¹²²⁾ admonished family members and colleagues not to encourage a depressed person "even out of the goodness of their heart."

Based on this, in the second revised edition of "Contemporary Clinical Psychiatry," published in 1983,⁸³⁾ Okuma added, along with Kasahara's Seven Articles of Oral Communication:

"Do not encourage the patient by saying, 'Pull yourself together and cheer up,' because this will increase the patient's sense of a guilty conscience and despair." Suwa further stated in the newly revised 31st edition of "The Latest Psychiatry," published in 1984¹²⁹⁾ that: "mere cheering up, however, must be avoided, as it can catch up with the patient and cause him to become depressed." Suwa wrote in "The Latest Psychiatry," Newly Revised, 31st Edition, published in 1984¹²⁹⁾, that: "mere encouraging, however, must be avoided, for it will only drive the patient on and intensify his suffering."¹⁶⁾

Here, unique concept of Nippon Depression was completed. It consists of A2 mild endogenous depression that is often mistaken for a stress tradition, an autonomous and recurrent course, a melancholic personality that is orderly and consciousness, and a treatment plan that includes mental and physical rest, TCA such as imipramine as an antidepressant, small psychotherapy, and characteristics such as "do not encourage"⁸⁷⁾⁸⁸⁾⁹³⁾⁹⁴⁾.

VII. Japan Since the 1990s: From Nippon Depression to Depression (DSM-5)

1. From Academic Terms to Everyday Language

In the 1980s, the Japanese concept of depression was a code used only among

clinical psychiatrists. It was not until around 1984 to 2000 that the term "うつ病／うつ" began to be used routinely in newspapers and weekly magazines. The main topics of discussion were the 1984 workers' accident compensation certification for suicides caused by "reactive depression" (the first recognition as a work-related accident in Japan due to a mental disorder) and the dispute over suicides caused by overwork that occurred in 1991 until its conclusion in 2000⁵⁸⁾⁷⁰⁾. Furthermore, in 1998, the number of annual suicides in Japan exceeded 30,000 for the first time, and social interest in "うつ病" increased.

In parallel with this, Japanese psychiatric journals began to use the terms "大うつ病" and "大うつ病性障害," which correspond to MDD, instead of "うつ病" around 1994. This was also the year DSM-IV was published. At this time, related conditions that could not be encompassed by the Nippon Depression category began to attract attention as a new form of depression. A pioneering example is 逃避型抑うつ escape-type depression (Hirose, 1977)³¹⁾. Since the 1990s, changes over time in the illness and premorbid personality of Nippon Depression, or new depression-related pathologies have been reported one after another. These include 現代型うつ病 modern-type depression (Matsunami et al., 1991)⁷¹⁾, 未熟型うつ病 immature-type depression (Abe,

1995)¹⁾, and 職場結合性うつ病 workplace-connective depression (Kato, 2002)^{51)*17)}.

2. From Nippon Depression to MDD: Expansion of So-called "うつ病"

In 1999, fluvoxamine was launched. It was the first selective serotonin reuptake inhibitor (SSRI) approved in Japan. When antidepressants became new, the concept of "うつ病" was also updated; SSRIs were indicated for MDD (A1+A2), but not for Nippon Depression (A2). Thus, a new group, the vast stress tradition group (A1), was added to the post-SSRI "うつ病" group. This was the first stage of the expansion of depression.

In addition, direct-to-consumer (DTC) advertising of drugs was permitted at this time; disease awareness of MDD and packaged advertising of SSRIs were delivered directly to the consumer.

Most people have an idea of the stress tradition: "They call it depression," "I heard there's a good drug that works well," "That's good news." Thus, SSRIs have combined the long-standing "craving for chemical solutions to life's problems," i.e. drugs³³⁾, with "うつ病". The core of the new "うつ病" moved from traditional Nippon Depression (A2) to a stress tradition (A1).

Thus, "うつ病" became part of the vocabulary of everyday speech. The total number of patients with "うつ病"

and "躁うつ病" and the market size of antidepressants in Japan began to rise steadily. Around the year 2000 marked the watershed moment between Nippon Depression and MDD. In 2002, the Japanese Journal of Psychiatric Treatment published a special issue: "Has 'うつ病' Changed: Evaluation and Classification"¹²⁷⁾, in which the pros and cons of the MDD concept were questioned. The later proposed dysthymia-affinity depression (Tarumi, 2005)¹³³⁾ is MDD, but it is not Nippon Depression. However, it was presented as a subtype of "うつ病" rather than "non-うつ病," and was widely accepted in Japanese clinical practice. The substance of "うつ病" had already shifted from Nippon Depression to MDD.

Soon, MDD even swallowed the normal stress reactions (A0) of non-disabling daily life. Most of the nine items in Criterion A for the diagnosis of MDD are nonspecific and can be noted in healthy individuals. Therefore, in order to distinguish between normal and disability, it is necessary to judge Criterion B: "Clinically significant distress or dysfunction" instead of Criterion A. This is a value judgment, not a factual judgment. Thus, false-positives where a healthy person is judged to have a disability, increase in number. This is where the abuse of diagnosis and antidepressants comes in¹⁸⁾³²⁾⁹⁵⁾; from A1+A2 to A0+A1+A2.

This is the second stage of the expansion of "うつ病".

3. To うつ病(DSM-5)-Nippon Depression Loses its Function as a "Core Group of うつ病"

The Japanese version of DSM-5 published in 2014 changed the translation of MDD from "大うつ病性障害 (dai-utsu-byo-sei-shogai)" to "うつ病 (DSM-5) (utsu-byo, DSM-5)/大うつ病性障害 (dai-utsu-byo-sei-shogai)" in light of this trend⁷⁾⁹⁰⁾. In effect, MDD is "うつ病".

The Japan Depression Society also changed the target diagnosis in its treatment guidelines from "大うつ病性障害 (dai-utsu-byo-sei-shogai)" in the first edition in 2012 (published in 2013)⁷⁸⁾ to "うつ病 (utsu-byo)" in the second edition in 2016 (published in 2017)⁷⁹⁾. In the preface, it is clearly stated that "the term 'うつ病(utsu-byo)' in the guidelines is synonymous with 'うつ病 (DSM-5) (utsu-byo, DSM-5)/大うつ病性障害 (dai-utsu-byo-sei-shogai),' which is a translation of 'major depressive disorder'."

Thus, the equivalence of "うつ病" and MDD was approved by the academic community. Here, Nippon Depression lost its function as a "prototype of うつ病"⁴²⁾ "core group of うつ病"¹¹⁴⁾¹¹⁵⁾, and "real うつ病"⁷⁷⁾. Until then, psychogenic depression⁵⁶⁾⁶⁶⁾⁶⁷⁾⁸²⁾, reactive depression⁶⁶⁾⁶⁷⁾⁸²⁾⁸³⁾, neurotic

depression⁸²⁾⁸³⁾¹¹⁹⁾¹²⁰⁾¹²¹⁾, and conflict-responsive depression⁴⁵⁾ were referred to as various types of "うつ病" but were required to be differentiated or excluded from Nippon Depression because the symptoms and course of the disease are heterogeneous and the treatment strategies are different. ^{*18} However, these conditions and Nippon Depression have now been combined under a single category, MDD, or new "うつ病".

VIII. Problems of MDD and Future of Nippon Depression

The diagnostic criteria for MDD originated at the Psychobiology of the Depressive Illness Conference held at the National Institute of Mental Health in the United States in 1969⁵²⁾¹⁰³⁾. Currently, these diagnostic criteria are used in biomedical models, biopsychosocial models, epidemiological studies, and all other contexts.

The diagnostic hierarchies of Nippon Depression and MDD are different when compared in the biomedical model. Nippon Depression is a disease with a single set of symptoms, course, and prognosis, although the etiology and pathogenesis are unknown¹²⁰⁾. MDD, on the other hand, is a syndrome that arises from various origins, including stress traditions and Nippon Depression⁹⁵⁾¹⁴⁵⁾, a final common pathway of depression, so to speak¹⁴⁵⁾.

The psychobiology underlying the American psychiatric tradition is as follows. It is true that the etiology of MDD may vary depending on such factors as heredity, constitution, upbringing, personality, stress, etc. However, even if the causes are different, the symptoms are the same, MDD. Conversely, no matter how the symptoms are considered, classification based on the cause cannot be achieved. In order to classify MDD, we must grasp the multiple dimensions of family history, constitution, chemical messengers, cerebral blood flow, life history, personality, lifestyle, social environment, etc., in line with the biopsychosocial model⁶⁸⁾¹⁴⁵⁾. Treatment should also flexibly combine pharmacotherapy, psychotherapy, and rehabilitation according to these multiple dimensions.

In reality, however, MDD was treated as if it were a single disease, and antidepressants were administered in a uniform manner. The psychosocial dimension was often ignored, and MDD was treated as if it occupied a biomedical model¹⁸⁾³²⁾⁹⁵⁾. This trend became widespread with the spread of SSRIs in the U.S. after 1988 and in Japan after 1999. Fortunately, this trend is now being controlled.

Future research is expected to improve the accuracy of diagnosing MDD and develop supplementary diagnostic tests,

such as biological markers, to help in the selection of treatment methods. However, biological markers cannot be used to distinguish between normal and disability. Criterion B, "clinically significant distress or dysfunction," is a value judgment based on psychosocial dimensions and cannot be reduced to biology. In addition, the causes and pathogenic mechanisms of the underlying causes of MDD, such as mild endogenous depression, depressive neurosis, and depressive personality, are unknown. Therefore, the targets that biological markers measure are not defined. Their validity is limited⁹²⁾.

The validity of MDD concept in a biomedical model is not promising; it should be reconsidered in the context of the original biopsychosocial model. It is a genealogy of stress traditions that originated in neurasthenia. In the late 19th century, several physical illnesses were isolated from it, including a mild form of manic-depressive illness that still exists today. For this group, the biomedical model, as well as for the physical illnesses, must be prioritized. This is mild endogenous depression. Future biomedical research and treatment of depression-related conditions will be more fruitful using mild endogenous depression concept, rather than MDD concept, as a starting point.

Nippon Depression is a comprehensive

view of the multiple dimensions of "premorbid personality - situational theory of illness - illness picture - response to treatment - course of illness"⁴⁵⁾, with mild endogenous depression as its core. It is based on a solid biomedical model and takes psychosocial aspects into consideration. The melancholic personality and guilty consciences tendencies of this illness have long been said to be decreasing or changing since around 1990²⁷⁾. This problem is significant for social and cultural considerations^{*19)}.

Through this process, we may be able to extract universal characteristics of mild endogenous depression. For example, the "maintenance of amicable relationships with others" and loyalty to the organization to which one belongs, which Kasahara considered essential and fundamental characteristics of the melancholy affinity type personality, have disappeared in recent years. However, Matsunami⁷¹⁾⁷²⁾ pointed out the obsessive-compulsiveness and orderliness that patients show in their private sphere (e.g., hobbies) outside of the workplace in modern depression. Kasahara⁵⁰⁾ also recently described a patient who "used to be called a lazy person in the world, but under 'certain conditions,' he or she can be very orderly." These may all be variants of the manifestation of orderliness that is a basic characteristic of the melancholy

type. If we take this approach, we may be able to open the door to biomedical research, such as biological rhythm research, with a breakthrough in the Japanese theory of depression.

Conclusion

There is no real description of Nippon Depression in medical papers or textbooks. It is found in the personal accounts of those who have suffered from it. Manabu Senzaki 9-dan, a Shogi player, describes his own experience of depression as follows: "There was a black coal tar like substance that was hardened in my mind, shutting out not only joy, anger, sorrow, and pleasure, but also all dark emotions such as regret and jealousy. In a nutshell, the symptoms of real depression are non-reactivity: no response to pleasure or sadness. In the beginning, it may be just a state of depression, like sadness or pain, but in real depression, all sensitivities disappear"¹¹⁶). He clearly describes not only the painful experience of mood non-reactivity and inability to mourn (A2) in Nippon Depression, but also the difference from normal sadness (A1) in MDD.

Nippon Depression is a concept that accepts the sincere narratives of these patients and reinterprets them in the context of psychiatry's body of knowledge. Psychiatrist Tsunetaka Tanaka¹³⁴, who experienced

endogenous depression himself, complains that he is not satisfied with DSM's lumping together of a wide variety of conditions into a single major depressive disorder. For the patients concerned, MDD is not a substitute for Nippon Depression. The concept of Nippon Depression needs to be re-evaluated before it is completely forgotten.

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Notes

1 However, it was not called a prototype, core, or genuine from the beginning. After World War II, this type of illness attracted attention as an "atypical"³⁰⁾⁹⁸⁾ condition on the border between reactive (grief reaction) and endogenous (severe manic-depressive illness)⁹¹⁾. It was not until the efficacy of antidepressants was established that it came to be regarded as a typical and core form of the illness.

2 The term "endogenous" often has a connotation of severity. The term "melancholic type" is unclear as to whether it refers to the premorbid personality (Tellenbach's melancholic type) or pathological picture (DSM-5's melancholia features).⁹⁹⁾ Moreover, melancholia is mainly seen in severe cases⁶⁾⁷⁾.

3 Unlike the neo-Kraepelinians⁶⁰⁾¹⁰²⁾¹⁴⁶⁾, DSM-III⁴⁾ does not require a delineation between mental disorder and normality, nor between mental disorder and other mental disorders. Furthermore, they established a multiaxial diagnosis and required the recording of psychosocial dimensions¹⁴²⁾. These are considerations for the biopsychosocial model¹⁵⁾¹⁷⁾.

4 Guze²¹⁾ even asserts that "all psychological processes are brain processes, so they are all included in the medical model."

5 Incidentally, since DSM-IV, the Japanese version has translated "mental disorder" as "精神疾患 (seishin-shikkann)"⁶⁾, which means D1=D2. This is consistent with the direction of DSM-5.

6 The notation for this pathotype has been confusing. Initially, the term "鬱病 (ustu-byou)"¹²⁸⁾ "うつ病 (ustu-byou)"⁸⁰⁾⁸¹⁾¹²⁰⁾ was used without qualifiers to compare it with neurosis. Later, however, when compared within the manic-depressive illness category, it was also described as "反応(性)うつ病 (hannou'sei'-ustu-byou)"⁸²⁾⁸³⁾ "抑うつ神経症 (yoku-utsu-shinkeishou)"⁴⁴⁾ and "神経症性うつ病(メランコリー型) (shinkeishou-sei-uetsu-byou (melancholic type)"⁴⁴⁾, among others. Here, "neurotic" and "neuroticism" mean "mild illness." According to Kielholz⁵⁶⁾, this is not endogenous depression, but a form of psychogenic depression. It was not until the introduction of Tellenbach's monograph¹³⁵⁾ that this form of depression came to be described as "endogenous depression," as it has in recent years.

7 The article²⁸⁾, which opened the door to this concept, begins by introducing Schneider's vital depression.

8 The diagnostic criteria for persistent depressive disorder (dysthymia), which is equivalent to depressive neurosis in DSM-III⁴⁾ and III-R⁵⁾, include A1 but not A2. The same is true for adjustment disorder with depressed mood.

9 It is true that some neurotic depressions, such as Kasahara-Kimura Type III "葛藤反応型うつ病 (kattou-hannou-gata-utsu-byou, conflict-response depression),"⁴⁵⁾ are only A1, an extension of normal psychopathology, even though they are called "うつ病 (ustu-byou)." However, Kasahara et al.⁴⁵⁾ suggest that "the indefinite term '神経症うつ病 (shinkei-sei-utsu-byou, neurotic depression)' should be abandoned." For Kimura⁵⁷⁾, neurotic depression is not うつ病 (ustu-byou); it is a non-depressive depressive symptom of non-depressive illness. Okuma⁸²⁾⁸³⁾ states that "神経症性うつ病 (shinnkei-sei-utsu-byou, neurotic depression) is not manic depressive illness in the narrow sense of the term, but belongs to the category of neurosis," and Shinfuku et al.¹²¹⁾ state that "神経症性うつ病 (neurotic depression) is a state of depression, but it is essentially a neurosis. The classification of DSM(-I)²⁾ and DSM(-II)³⁾ is similar regarding this point. "神経症性"うつ病" (neurotic depression,)

as a name of a disease may be a mistranslation of "神経症性"抑うつ" (neurotic depression) as a state.

10 Lithium administration for depression is indeed pioneering. However, there is no historical or theoretical continuity between lithium therapy for depression and lithium therapy for bipolar disorder today. At that time, lithium therapy for gout was in vogue, as was the proposal by Lange, C.

11 Some of the work of Kahlbaum and Hecker preceded that of Lange, C., but only names and theories were given, and specific cases were not described.

12 In his 1926 paper, Lange, J. distinguished between "reactive melancholie," in which A1 is followed by A2, and "psychologically provoked melancholia," in which A2 develops directly from an event, without A1⁶⁶). In 1928, these were grouped together under "reactive depression (melancholia)."

13 In addition to managers, politicians, lawyers, doctors, and housewives who are overwhelmed by numerous worries are also listed. Physical workers are scarce. The reason is that physical labor and regular working hours act as a deterrent against mental exhaustion⁵⁵). This nuance, which includes a painful but slightly proud description of a modern disease that strikes a relatively high social status and importance group, is very similar to the discourse surrounding the nervous breakdown of the late 19th century.

14 The term "鬱病 (utsu-byou)" in Kanji can be found in Uchimura¹³⁶) and Suwa¹²⁸), and "うつ病 (ustu-byou)" in hiragana in a 1956 academic publication¹⁰⁰) and in a book for the general public in 1958⁸⁰). However, the origin of Nippon Depression is the review by Hirasawa.

15 This prototype had already been published in 1970⁴³).

16 By the way, Kasahara himself wrote about the treatment of endogenous depression in the textbook "Essential Psychiatry" published in 1984⁴⁸). There are seven articles by oral communication, but there is no mention of contraindications to encouragement.

17 Matsunami⁷¹⁾⁷²⁾ clearly states: "Contemporary manifestations of endogenous depression". However, there is some controversy regarding whether these symptoms fit into the category of endogenous depression⁷⁶⁾.

18 In fact, Shinfuku¹¹⁹⁾ suggested that since "none of the physical treatments such as pharmacotherapy and ECT are very effective," psychotherapy "focuses on improving personality, encouragement, sports, recreation etc., to Alleviate the mood (and so on)", and was proposing a policy that conflicted with the therapy of Nippon Depression.

19 In 1962, Hirasawa²⁹⁾ discussed the characteristics of the immobilithymia found in outpatients with mild endogenous depression in Kyoto. The main characteristic of the immobilithymia is not enthusiasm but orderliness. This difference was considered to be caused by the difference in severity of the target cases. In the same year, Yoshinaga¹⁴⁷⁾ investigated the chronological changes in the personality signs of immobilithymia at Kyushu University Hospital, where Shimoda used to work. While "Enthusiasm," "Responsibility," and "Frankness" decreased in frequency with the end of the war, "Orderliness" and "Thoroughness" showed no significant difference. Yoshinaga postulated socio-cultural influences as the reason for this. The melancholic personality may have been like a snapshot of a moment in a dynamic structure that changes over time. It is ironic that the concept of the melancholic-type itself includes temporality.