Psychosocial determinants of recovery in depression

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There is a growing body of literature on residual symptoms after apparently successful treatment. The strong prognostic value of subthreshold symptomatology upon remission and the relationship between residual and prodromal symptomatology (the rollback phenomenon) have been outlined. Most residual symptoms also occur in the prodromal phase of depression and may progress to become prodromes of relapse. These findings entail important implications. It is necessary to closely monitor the patient throughout the different phases of illness and to assess the quality and extent of residual symptoms. A more stringent definition of recovery, which is not limited to symptomatic assessment, but includes psychological well-being, seems to be necessary. New therapeutic strategies for improving the level of remission, such as treatment of residual symptoms that progress to become prodromes of relapse and/or increasing psychological well-being, appear to yield more lasting benefits. The sequential model may provide room for innovative treatment approaches, including the use of drugs for specifically addressing residual symptoms. As occurs in other medical disorders (such as diabetes and hypertension), the active role of the patient in achieving recovery (self-therapy homework) should be pursued.

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Clinicians working with depressed patients are often confronted with the unsatisfactory degree of remission that current therapeutic strategies yield, and with the vexing problems of relapse and recurrence.[1] In clinical medicine, the term "recovery" connotes the act of regaining or returning toward a normal or usual state of health. However, there is a lack of consensus regarding the use of this term (which may indicate both a process and a state), as well as of the related word "remission." This latter indicates a temporary abatement of the symptoms of a disease. Such ambiguities reflect on the concepts of relapse (the return of a disease after its apparent cessation) and recurrence (the return of symptoms after a remission).

In an attempt to overcome these flaws, Frank et al[2] proposed a set of definitions which they referred to as longitudinal studies of mood disorders, but may entail more general applicability in psychiatry. Remission (which is differentiated into partial and full remission) is a relatively brief period during which an improvement of sufficient magnitude is observed and the individual no longer meets syndromal criteria for the disorder. Recovery implies a more sustained remission, and raises the possibility that treatment can be discontinued or prolonged with the aim of prevention. Relapse is a return of symptoms satisfying the full syndromal criteria during the period of remission, whereas recurrence

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can occur only during a recovery. The development of these criteria provides helpful ground for decreasing inconsistencies among research reports, yet it does not touch some key issues in the conceptualization of these terms. First, according to these definitions,[2] recovery occurs when the number and severity of symptoms fall below the threshold used for defining onset, and this subthreshold level of symptomatology remains for a specified period of time. However, this state cannot be equated with being asymptomatic, and provides room for a wide range of subclinical conditions. Second, the definition of remission parallels the traditional medical concept of convalescence, a transitional period of reintegration after illness. The trajectory of such a process is thus an important additional dimension which requires a longitudinal consideration of the development of disorders, encompassing the prodromal phase, the fully developed disorder, and residual states. Not only the duration of the acute phase of illness—as is widely acknowledged— may affect the rate of recovery, but also the characteristics of prodromes, the amount of residual symptomatology not alleviated by specific treatments, and the level of premorbid functioning may influence the course of recovery.[3,4] Finally, the distinction between recovery and full remission is made on temporal grounds only. They are not differentiated by whether active treatment is associated, even though recovery implies the possibility that therapy can be discontinued. A recovered depressed patient who is currently drug-free is thus equated to another patient who is receiving long-term, high-dose antidepressant treatment.

The aim of this review is to analyze some issues which would help to define the psychosocial determinants of recovery in depression.

The inadequancies of standard clinical assessment

The staging method, whereby a disorder is characterized according to seriousness, extent, and features, has achieved wide currency in medicine, but is currently neglected in psychiatry.[4,5] The operational definitions of DSM give only a flat, cross-sectional view of the patient's depressive illness, that ignores its longitudinal development, previous episodes, and responses to previous treatments.[4] A prodromal phase can be described in most instances of depression,[6] and only a minority of patients become asymptomatic after successful treatment. Current pathophysiological models of pathogenesis in depression thus neglect intermediate phenomenological steps in the balance between health and disease *(Table I)*.

Stages	
1	Prodromal phase (anxiety, irritable mood, anhedonia, sleep disorders)
	a. no depressive symptoms
	b. minor depression
2	Major depressive episode
3	Residual phase
	a. no depressive symptoms
	b. dysthymia
4	a. recurrent depression
	b. double depression
5	Chronic major depressive episode (lasting at least 2 years without interruptions)

Table I. Stages of primary unipolar depression.[7]

Staging has the potential to improve the logic and timing of interventions, just as it does in many complex and serious medical disorders.[5] Drug mechanisms which may be operational in the initial phase of treatment may change during long-term treatment and according to the stages of illness.[8] This approach is also in accordance with the sequential model of treatment, which was found to be effective in clinical medicine and psychiatry.[9]

The majority of depressed patients do not qualify for one, but for several, Axis I and Axis II disorders.[10] However, there is comorbidity which wanes upon successful treatment of depression and comorbidity which persists, in syndromal or subsyndromal forms (residual symptoms). Clinical differentiation of such morbidity requires a shift from the current psychometric model (where severity is determined by the number of symptoms and not by intensity or quality) to a clinimetric model,[10-12] which may allow the definition of the progression, extent, and severity of depressive illness.

Measurement

Total absence of psychological symptoms is not a frequent characteristic of the general healthy population.[13] As a result, the determination of recovery depends on the symptom intensity under which recovery is defined, and on the type and characteristics of the measurements we select. In the recovery phase symptoms are typically

milder than those of the full clinical syndrome.[1] The capacity of the assessment instrument to measure small increments or small changes near the normal end of the spectrum becomes important. The ability of a rating or self-rating scale to discriminate between different groups of patients suffering from the same illness (eg, depressed inpatients and outpatients) and to reflect changes in experiments in therapeutics such as drug trials in which the drug effects are small may indicate its degree of sensitivity.[13] This concept is particularly important when treatment effects are small and in the setting of subclinical symptoms.[1] Unfortunately, researchers tend to focus on the psychometric characteristics of validity and reliability and to neglect sensitivity.[10,14,15] They may thus employ inadequately sensitive instruments to establish lack of significant symptomatology.

The Hamilton Depression Scale (HAM-D)[16] is an example of an instrument based on the classical psychometric model. The key flaw of such an instrument is that the same score on the Hamilton Rating Scale for Depression may be the product of few very severe core symptoms (eg, a severely retarded depressed patient) or of several mild accessory symptoms (reflecting perhaps a subject affected by a mild form but with many symptoms and complaining behavior). Correspondingly, the decrease in the final score may be ascribed to the improvement/disappearance of the typical depressive signs (eg, mood, anhedonia, guilt, suicidal ideation, psychic signs, and retardation), which is significant on clinical grounds, or to the alleviation of accessory symptoms (eg, anxiety, appetite, insomnia, sexual interest, and somatic symptoms), which is of limited value. Further, adverse effects of treatments (eg, sleepiness or sedation) may decrease the total score of the rating scale, producing an artificial improvement.[15]

As important is the target of the instruments employed. For instance, in a naive conceptualization, yet the one implicitly endorsed by DSM-III and DSM-IV, well-being and distress may be seen as mutually exclusive (ie, well-being is lack of distress). Yet there is evidence to call such views into question.[17-19] As a result, the appraisal of recovery may rest on purely symptomatic grounds,[1] or may be extended to perceptions (levels of well-being and satisfaction with life), or be expanded to functional capacity (the ability to perform activities of daily life, social and intellectual function, economic status). This latter tridimensional assessment may be subsumed under the rubric of quality of life.[17]

Measurement may also be extended to biological variables, which tend to subside upon clinical recovery and may accompany both prodromal and residual symptomatology and constitutes a psychobiological risk for relapse. Such markers may include abnormalities of the hypothalamic-pituitary-adrenal (HPA) axis,[20,21] impaired lymphocyte glucocorticoid sensitivity,[22] and abnormal sleep electroencephalographic (EEG) patterns.[23-27]

The more sensitive and multidimensional the tools employed, the more arbitrary the nature of the recovery which emerges.

Residual symptoms

The notion that the majority of depressed patients experience mild but chronic residual symptoms or recurrence of symptoms after complete remission, which was well delineated in the 1970s,[28] did not receive the attention it deserved in subsequent years. Such a phenomenon was emphasized, in fact, mainly in its etiological role regarding dysthymia. Subsyndromal residual symptoms of major depressive disorder continued to be regarded as minor fluctuations unworthy of clinical attention. However, the literature describing the presence of residual symptoms after completion of drug treatment of major depression and their clinical implications in terms of poor long-term outcome continue to grow.[29-43] Residual subthreshold symptoms were also reported after completion of psychotherapy.[41,44-46]

In 1973 Paykel and associates[47] found social and interpersonal maladjustments in recovered depressed patients compared with controls, despite considerable improvement in social adjustment upon treatment. Submissive dependency and family attachment improved almost completely, whereas two other personal dysfunctions, interpersonal friction and inhibited communication, showed little change and greatest residual impairment.[47] Residual social maladjustment was subsequently reported by other investigators,[38,48-52] and was found to correlate with long-term outcome.[38,48,52-54]

The question has been raised as to whether these interpersonal functioning deficits are trait- or state-dependent.[53,55,56] When monthly ratings of impairment in major life functions and social relationships were obtained during a 10-year follow-up of 371 depressed patients, disability was pervasive and chronic, but disappeared when patients became asymptomatic, confirming the hypothesis that psychosocial disability is state-dependent.

Depressive symptoms at levels of subthreshold depressive symptoms, minor depression/dysthymia, and MDD represent a continuum of depressive symptom severity in unipolar MDD, each level of which is associated with a significant stepwise increment in psychosocial disability.[38] In another investigation[52] in 222 depressed outpatients, an earlier onset of clinical response predicted better overall psychosocial functioning at end point, whereas the number and the severity of residual symptoms predicted poorer overall psychosocial adjustment at end point in responders. Other studies[53,56] suggested that there may be a subgroup of patients with impaired psychosocial and/or early onset of depression with stable interpersonal deficits.

Similarly, dysfunctional attitudes and attributions were found to persist after recovery, despite clinical and cognitive improvement.[57-61] These cognitive patterns were positively correlated with vulnerability to persistent depression or relapse.[58-60,62] These findings were consistent with the fact that vulnerable attitudes such as high neuroticism assessed when the depressed patients are symptomatic predict recovery,[63,64] but that, for the prediction of relapse, cognitive measures when patients are asymptomatic need to be used.[59] Social maladjustment and dysfunctional attitudes may overlap with characterological traits assessed after clinical recovery[65-75] or premorbid personality features.[76,77] Ormel et al[78] studied personality traits such as neuroticism, low self-esteem, and poor coping skills, before, during, and after a major depressive episode, in a 3-wave general population-based investigation. There was no evidence of a negative change from premorbid to postmorbid assessment of personality variables. Postmorbid vulnerability reflected the continuation of premorbid vulnerability. Both were influenced by prodromal and residual symptoms.[78] Ongur et al[79] found that temperamental features were related to patterns of anxiety disorder comorbidity in depressed patients, as was also found to be the case for well-being.[80]

Regardless of the state/trait dichotomy the findings of different studies indicate that there appears to be a residual attributional interpersonal component which is refractory to otherwise successful treatment of depression. Such components may entail considerable predictive value.

Methodological problems in assessment of residual symptoms, however, emerge. There is paucity of psychometric studies addressing the phenomenology of depressed patients after benefiting from treatment. Recovered depressed patients displayed significantly more depression and anxiety than control subjects in one study,[81] but not in another.[82] Differences in the sensitivity of the rating scales which were employed may account for such discrepant results. Using Paykel's[83] Clinical Interview for Depression, only 6 (12.2%) of 49 patients with major depression successfully treated with antidepressant drugs and judged to be fully remitted had no residual symptoms.[84] The majority of residual symptoms were present also in the prodromal phase of illness. The most frequently reported symptoms involved anxiety and irritability. This findings were consistent with previous studies on prodromal symptoms of depression,[85,86] overlapped with results concerned with interpersonal friction,[47] irritability,[77] and anxiety[65] and underwent independent replication. Using a similar methodology, Paykel et al,[34] in fact, found residual symptoms to be present in 32% of 60 patients who remitted from major depression. Previous diagnosis of dysthymia did not predict residual symptoms. Depressed mood, guilt, hopelessness, impaired work and interest, anxiety, and anorexia were identified by the Clinical Interview for Depression.[36] These symptoms tended to persist at 8- to 10-year follow-up.[87] Nierenberg et al[37] found that only 18% of full responders to fluoxetine were free of residual symptoms. Gastò et al[39] reported the same percentage in elderly patients with major depressive disorders. Judd et al[88] found that incomplete recovery from the first lifetime major depressive episode was linked to a chronic course of illness during a 12-year prospective naturalistic follow-up. Angst et al[89] observed that clinical trials overestimate the likelihood of full recovery on a single antidepressant. The usual response rates of 60% to 70% are typically reported when a reduction of 50% or more in the Hamilton Depression Rating Scale occurs. However, using a more conservative score for defining response, only 45% of approximately 900 depressed patients achieved a satisfactory response. Cornwall and Scott[90] reviewed publications relating to a precise definition of partial remission.[1] Partial remission was found to affect at least one third of subjects treated for depression, to increase the risk of further depressive relapse, and to adversely affect social and work performance. In a large, multicenter trial involving 2876 outpatients receiving flexible doses of citalopram, only 28% of subjects were found to have remitted.[91]

In conclusion, substantial residual symptomatology appears to characterize depressed patients who successfully responded to pharmacological or psychological

therapies. Anxiety, irritability, and interpersonal friction, in addition to specific depressive symptoms, appear to be common residual symptoms.

The rollback phenomenon and state-trait dichotomy

Detre and Jarecki[92] provided a model for relating prodromal and residual symptomatology, defined as the rollback phenomenon: as the illness remits, it progressively recapitulates (though in a reverse order) many of the stages and symptoms that were seen during the time it developed. According to the rollback model, there is also a temporal relationship between the time of development of a disorder and the duration of the phase of recovery.

> For example, if an illness begins with occasional anxiety attacks that are superseded some weeks later by depressive symptoms which then become progressively more severe until, after several months, the patient develops total insomnia and confusion, the symptoms tend, as the condition improves, to remit in reverse order, the confusion and insomnia diminishing first, and the depressed mood next. After the depression lifts, the patient may again experience anxiety attacks for several weeks, until finally these symptoms, too, disappear."[92]

The rollback phenomenon—or, at least, a strong relationship between prodromal and residual symptomatology—has been substantiated in the treatment of major depression.[84] In one study,[84] almost 70% of the residual symptoms that were found to occur in 40 remitted depressed patients were also present at the prodromal phase of illness. This percentage increased to almost 90% of cases for residual generalized anxiety and irritability. These results achieved independent replication,[93] and are also supported by several lines of evidence. In a prospective study[94] which examined the possibility that episodes of major depression result in lasting personality changes that persist beyond recovery (the scar hypothesis), there was no evidence of negative change from premorbid to postmorbid assessment. These findings were replicated by Ormel et al.[78] Further, a 10-year follow-up study after severe depression[93] suggested that residual symptoms were common and persistent, with considerable fluctuations. This would suggest continuity—whether we rate it in characterological or symptomatological terms—between the prodromal and residual phases. Another line of evidence is based on recognition of specific temporal courses of change during treatment of depression.[96-99] Different types of treatment may affect the temporal course of change in depression,[100] and the use of pattern analysis may differentiate true drug and placebo responses early in treatment.[101] Patients do not suddenly become well, but tend to gradually lose their depressive symptoms over the months following treatment.[102] Stassen and associates[103] found that the time course of improvement among responders to amitriptyline, oxaprotiline, and placebo was independent of the treatment modality, and thus identical in all three groups. Once triggered, the time course of recovery from illness became identical to the spontaneous remissions on placebo. Antidepressant drugs, therefore, may not change the pattern of the natural course of recovery from illness, but simply speed the recovery and change the boundary between "responders" and "nonresponders."[103]

The psychometric distinction between state and trait may also reflect the rollback phenomenon, and may hinder detection of change. If recovery implies the return to premorbid functioning, personality traits are likely to influence its definition. Unfortunately, the state-trait dichotomy and its psychometric counterparts appear to be situated on a continuum with blurred borders which do not permit clearcut differentiation.[1] For instance, certain personality traits may entail enduring, long-term characteristic modes of feeling, thinking, and behaving in the course of depression, whereas antidepressant treatment may be beneficial in the modification of certain personality traits, which are therefore subject to state influences.[68,69] Rafanelli et al[18] introduced the hypothesis that the state/trait characteristics of a specific instrument may be stage-dependent.

The concept of mental health

Ryff and Singer[104] remark that, historically, mental health research is dramatically weighted on the side of psychological dysfunction, and that health is equated with the absence of illness rather than the presence of wellness. They suggest that the absence of well-being creates conditions of vulnerability to possible future adversities, and that the route to recovery lies not exclusively in alleviating the negative, but in engendering the positive. Little is known of the relationship between subclinical symptoms and well-being in the residual phase of affective disorders. In a small investigation,[105] a well-being-enhancing psychotherapeutic strategy (well-being therapy) was

found to be associated with a significant reduction in residual symptoms in patients with affective disorders. The balance between positive and negative affects and its biological counterparts may thus carry considerable weight on the complex regulation underlying the long-term outcome of affective disorders.

In a survey on factors identified by depressed outpatients as important in determining remission, the most frequently judged as such were the presence of features of positive mental health, such as optimism and self-confidence, a return to one's usual, normal self, and a return to the usual level of functioning.[106] In 1958 Marie Jahoda[107] outlined some tentative criteria for positive mental health, encompassing attitudes toward the self, growth, integration, autonomy, perception of reality, and environmental mastery. Such criteria were refined and expanded in Carol Ryff's multidimensional model,[108] which encompasses six dimensions: mastery of the environment, personal growth, purpose and meaning of life, autonomy, self-acceptance, and positive relationships. This theoretical model of psychological well-being was then applied in a variety of clinical settings.[109] Ryff's psychological dimensions[108] may be instrumental in assessing both the process and the definition of recovery *(Table II)*.

The neglect of self-therapy

An increasing body of evidence links the progression of several medical disorders to specific lifestyle behaviors.[110] Half of the deaths that take place in the US can be attributed to "largely preventable behaviors and exposures," such as tobacco smoking, obesity, and physical inactivity.[111] Similarly recovered depressed patients continue to show social and interpersonal maladjustments and dysfunctional attitudes which have serious consequences in terms of vulnerability to persistent depression or relapse. Unfortunately, psychiatrists tend to view treatment and prevention of relapse of depression purely in pharmacological terms, and they overemphasize the need for providing maintenance therapies, without paying attention to lifestyle and problems related to tolerance.[8,112]

Frank and Frank[113] have clarified how "certain types of therapy rely primarily on the healer's ability to mobilize healing forces in the sufferer by psychological means. These forms of treatment may be generically termed psychotherapy."

Cognitive behavioral therapy may be seen as guided self-therapy which aims at developing the patient's control over his or her own problems or behaviours.[114] Homework assignments (whether consisting of self-observation or

Dimensions	Optimal level
Environmental mastery	A: Has a sense of mastery and competence in managing the environment
	B: Makes effective use of surrounding opportunities
	C: Is able to create or choose contexts suitable to personal needs and values
Personal growth	A: Has a feeling of continued development
	B: Has sense of realizing own potential
	C: Sees improvement in self and behavior over time
Purpose in life	A: Has goals in life and a sense of direction
	B: Feels there is meaning to present and past life
	C: Holds beliefs that give life purpose
Autonomy	A: Is self-determining and independent
	B: Is able to resist social pressures
	C: Evaluates self by personal standards
Self-acceptance	A: Has a positive attitude toward self
	B: Accepts his or her good and bad qualities
	C: Feels positive about his past life
Positive relations with others	A: Has warm and trusting relationships with others
	B: Is capable of strong empathy, affection, and intimacy
	C: Understands give and take of human relationships

Table II. Modification of the 6 dimensions of psychological well-being according to Ryff's model.[108]
Note: At least A or B or C should be present to satisfy criteria for each dimension.

performing specific tasks) are given and reviewed by the therapist.

The patient's contribution to obtaining recovery has been traditionally outlined in anxiety disorders,[115,116] with particular reference to self-exposure. More recently, a number of psychological strategies have been developed for prevention of relapse in depressive disorders. They include cognitive restructuring and increase in of coping skills,[117-120] promotion of psychological well-being,[105,117] mindfulness meditation,[121] lifestyle modification.[117] The optimal application of these therapies has taken place within the sequential model of therapy.[9]

The sequential model

There is increasing literature on the bleak long-term outcome of depression as to relapse and recurrence.[122-129] This unsatisfactory outcome seems to be associated with the presence of substantial residual symptomatology, which are probably the most consistent predictors of relapse. In a large cohort study, asymptomatic recoverers relapsed in 157 weeks, compared with residual recoverers who relapsed in about 28 weeks.[35] At the same time, there is growing awareness of the fact that current forms of treatment seem to be insufficient for many patients, both in adult[91,130] and adolescent[131] depression. Increasing the level of remission thus appears to play a key role for yelding optimal treatment outcome.

If residual symptoms are the rule after completion of drug or psychotherapeutic treatment and their presence has been correlated with poor outcome, residual symptoms upon recovery may progress to become prodromal symptoms of relapse and treatment directed toward residual symptoms may yield long-term benefits.[1] Treatments which are administered in a sequential order (psychotherapy after pharmacotherapy, psychotherapy followed by pharmacotherapy, one drug following another, and one psychotherapeutic treatment following another) may be more successful in increasing the spectrum of therapy and in yielding disappearance of residual symptomatology.[9] There is a substantial body of evidence supporting the use of cognitive behavioral therapy after successful pharmacotherapy for decreasing the likelihood of relapse during follow-up.[84,117,118,132-138] In two studies[132,133] follow-up was up to 6 years.

The rationale of this approach was to spend cognitive behavioral treatment resources when they are most likely to make a unique and separate contribution to patient well-being and to achieve a more pervasive recovery. Weissman and associates[139] showed a significant effect of interpersonal psychotherapy on social adjustment symptoms of depressive patients, whereas there was no effect on the patient's social adjustment for amitriptyline and there were no drug-psychotherapy interactions. Since social adjustment is a major part of residual symptomatology in depression, as described previously, the findings of this study may now be reinterpreted according to a sequential, stage-oriented model,[4] where different therapeutic strategies can be applied to different stages of illness.

There has been little research on other forms of sequential treatment in depression.[9] It has been suggested that the most effective drugs in treating acute depression may not be the most suitable for postacute or continuation treatment.[140] During a 6-year follow-up of a randomized trial comparing the sequential use of pharmacotherapy and cognitive behavioral treatment versus clinical management in patient with recurrent depression,[134] no antidepressant drugs were used unless a relapse ensued. Patients were then treated with the same antidepressant drug that had been used in the previous episode. Clonazepam was added to the treatment regimen and continued when the antidepressant drug was stopped. The mean survival time after introduction of clonazepam was significantly longer than the one before the first relapse. Menza et al[141] have postulated the sequential use of antidepressants and drugs which may specifically improve fatigue, sexual dysfunction, anxiety, and sleep disturbances.

On the contrary, the effect sizes favoring combined treatment have been generally rather modest.[142,143] One study that specifically addressed the effect of combined treatment (imipramine plus interpersonal psychotherapy) versus each treatment alone or no active treatment (upon the levels of residual symptoms) revealed no significant differences among the four strategies.[41] Patients in the combined treatment group, however, had fewer symptom peaks during the maintenance phase.

The definition of recovery

A basic problem in the criteria developed by Frank and associates[2] is the lack of criteria for judging a patient to be asymptomatic. The fact that a patient no longer meets syndromal criteria is insufficient and the number and quality of minimal symptoms allowed are not specified. Not all symptoms are equally important.[14,15] For instance,

persistence of depressed mood is different from lack of concentration in an improved depressed patient. Often different treatments are generally compared on the rate of response they may yield, instead of the amount of residual symptomatology they may leave. Unfortunately, currently used scales for assessing treatment outcome, such as the Hamilton Depression Rating Scale, are inadequate for assessing the wide spectrum of residual symptomatology.[6] Further, the concept of recovery should involve psychological well-being.[1] Finally, Frank and associates[2] emphasized the connection between the declaration of recovery and the possibility that treatment can be discontinued or prolonged only for preventive purposes. The symptomatic state of patients who are drug-free could be equated, in this case, to that of patients receiving continuation therapy. As a result, the criteria for recovery[2] seem to need a multidimensional redefinition, which reflects the clinician's orientation and prognosis, aside from a symptomatic assessment. Fava and colleagues[1] have recently suggested a new set of criteria for defining recovery that encompass psychological well-being *(Table III)*.

Commonly, the concept of recovery reflects that of "improvement" which refers to the clinical distance along which the current state of the patient is compared with the pretreatment position. In this sense, recovery can be expressed either as a categorical variable (present/absent) or as a comparative category (nonrecovered, slightly recovered, moderately recovered, greatly recovered). Both expressions require arbitrary cutoff points related to the amount of improvement. A depressed patient who, when asked how he or she feels after 3 weeks of treatment replies "just fine" (instead of "better"), uses a self-monadic component. The amount of change induced by treatment, however, may make him/her overlook the distance from an intended goal, such as the pre-episode state. The physician may collude with the patient in this illusion of wellness, since he/she may be gratified more by the amount of improvement induced in the patient, than by the current distance from an intended goal.[1] Clinicians may choose recovery as a target that is negotiated between the doctor and the patient. The doctor can insist that the target be reasonable (eg, not asking to be better than before the illness). Nevertheless, the idea of successful recovery may differ from one patient to the next and should not be constrained too much by the doctor's ideas. We should accept the possibility that a treatment may determine abatement of symptoms in some patients, leave a substantial residual symptomatology in others, yield an unsatisfactory response in others, and provide no benefit, or even cause harm, in a few. The type of residual symptomatology varies widely from patient to patient and needs to be assessed individually.[8]

Recovery from a major depressive episode is defined to occur when:

- the patient remains in full remission despite discontinuation of treatment (whether pharmacological or psychotherapeutic)
- if subclinical or subsyndromal symptoms are present, these are judged to be likely to improve spontaneously over time or not to affect the course of the illness. Residual symptoms which occurred also in the prodromal phase of illness are unlikely to be devoid of clinical implications
- the patient reports psychological well-being in at least one of the six areas described in Ryff's model.[108]

Table III. Definition of recovery.[1]

Conclusions

The literature surveyed in this paper suggests that standard treatment of depression, even in specialized settings, seems to yield modest and temporary benefits and to leave a large amount of residual symptomatology, which appears to be one of the the strongest predictors of unfavorable outcome. Increasing the level of remission thus appears to play a key role in yielding an optimal treatment outcome. It is hoped that more stringent criteria for recovery and endorsement of a longitudinal appraisal of affective disturbances may result in therapeutic efforts yielding more lasting relief. ❑

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Determinantes psicosociales en la recuperación de la depresión

Existe una literatura creciente acerca de los síntomas residuales después del tratamiento aparentemente exitoso. Se ha resumido el valor pronóstico potente de la sintomatología subumbral en la remisión, y la relación entre la sintomatología residual y prodrómica (el fenómeno de la reducción). La mayor parte de los síntomas residuales ya aparecen en la fase prodrómica de la depresión y pueden progresar hasta constituirse en pródromos de la recaída. Estos hallazgos suponen importantes consecuencias. Es necesario monitorear estrechamente al paciente a través de las diferentes fases de la enfermedad y evaluar la calidad y la magnitud de los síntomas residuales. Parece necesario contar con una definición más rigurosa de recuperación, la cual no esté limitada a la evaluación sintomática, sino que incluya también el bienestar psicológico. Las nuevas estrategias terapéuticas para mejorar el nivel de remisión, como el tratamiento de los síntomas residuales que progresan hasta constituirse en pródromos de la recaída y/o el aumento del bienestar psicológico, parecen dar paso a beneficios más duraderos. El modelo secuencial puede proporcionar espacio para aproximaciones terapéuticas innovadoras, incluyendo el uso de fármacos orientados específicamente a los síntomas residuales. Como ocurre en otros trastornos médicos (como diabetes e hipertensión), se debe conseguir un papel activo del paciente para alcanzar la recuperación (ejercicios de autoterapia).

Déterminants psychosociaux de la guérison dans la dépression

La littérature fait de plus en plus état de symptômes résiduels après un traitement antidépresseur apparemment efficace. La valeur pronostique élevée de la symptomatologie infraclinique sur la la rémission et le lien entre la symptomatologie résiduelle et prodromique (phénomène de régression inversée ou roll back) ont été décrits. La plupart des symptômes résiduels surviennent également durant la phase prodromique de la dépression et peuvent évoluer en prodromes de rechute. Ces résultats ont des implications importantes : le patient doit être surveillé attentivement au cours des différentes phases de la maladie, afin d'évaluer la nature et l'étendue des symptômes résiduels. Il semble nécessaire de donner une définition plus rigoureuse de la guérison, qui ne se limite pas à l'évaluation symptomatique mais inclut le bien-être psychologique. De nouvelles stratégies thérapeutiques pourraient apporter des bénéfices plus durables : améliorer le niveau de guérison en traitant les symptômes résiduels qui évoluent en prodromes de rechute et/ou en augmentant le bien-être psychologique. Le modèle thérapeutique séquentiel offre un espace pour les traitements innovants comme les médicaments spécifiques des symptômes résiduels. Comme c'est le cas dans d'autres pathologies (diabète ou hypertension), le patient doit poursuivre un rôle actif pour guérir (exercices d'autotraitement à domicile).

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