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Depression: Discrete or Continuous?

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Abstract
Elucidating the true structure of depression is necessary if we are to advance our understanding and treatment options. Central to the issue of structure is whether depression represents discrete types or occurs on a continuum. Nature almost universally operates on the basis of continuums, whereas human perception favors discrete categories. This reality might be formalized into a 'continuum principle': natural phenomena tend to occur on a continuum, and any instance of hypothesized discreteness requires unassailable proof. Research evidence for discrete types falls far short of this standard, with most evidence supporting a continuum. However, quantitative variation can yield qualitative differences as an emergent property, fostering the appearance of discreteness. Depression as a continuum is best characterized by duration and severity dimensions, with the latter understood in terms of depressive inhibition. In the absence of some degree of cognitive, emotional, social, and physical inhibition, depression should not be diagnosed. Combining the dimensions of duration and severity provides an optimal way to characterize the quantitative and related qualitative aspects of depression and to describe the overall degree of dysfunction. The presence of other symptom types occurs when anxiety, hypomanic/manic, psychotic, and personality continuums interface with the depression continuum.

Despite over 100 years of research, the structure of depression eludes accurate characterization. One of the key issues is whether depression occurs as discrete types or exists on a continuum [1–5]. Various discrete forms of depression have been identified but have not been proven to represent valid entities [1–7]. Some of the more common types that have been proposed include endogenous/melancholic, major, minor, reactive/neurotic, and dysthymic. By applying conceptual reasoning to existing research results, it is possible to elucidate the nature of depression. Accurate characterization of this mental health condition is crucial due to the enormous suffering resulting from it and less than ideal treatment outcomes.

Discrete or Dimensional?

A prominent theme in depression research has been its characterization. Kraepelin viewed depression as part of a manic-depressive continuum with mania, hypomania, depression, mixed states, and mood temperaments being variants of the same disorder [3]. Diagnostic systems, e.g. the Diagnostic and Statistical Manual (DSM) and the International Classification of Diseases (ICD), on the other hand, have viewed depression and mania as separate entities, with depression further divided into discrete conditions [3]. For example, in DSM-IV-TR, discrete types include 'major depression', 'minor depression', and 'dysthymia', with DSM-5 keeping the first and adding 'persistent depressive disorder' to replace 'dysthymia' [8, 9]. ICD-10 includes 'depressive episodes', 'dysthymia' (under 'persistent mood disorder'), and 'recurrent de-

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pression' [10]. Historically, depression has been separated into more internally based and reactive forms [2–5, 11, 12]. Various terms have been applied to both with the former typically referred to as endogenous, vital, or melancholic and the latter as reactive, neurotic, or dysthymic [5, 7, 11, 12]. Melancholia was identified at the time of Hippocrates and continued through Galenic and medieval times [5]. In 1621, Richard Burton wrote *The Anatomy of Melancholy* [5]. Carl Lange, in 1880, identified a disorder with neurovegetative features that became known as endogenous or vital depression [7].

The concept of reactive depression arose from psychoanalysts led by Freud who theorized that depression results from actual or symbolic losses of a love object [13]. Adolf Meyer advanced the view that all psychiatric illnesses, depression included, occur as psychobiological reactions to stress [5]. The notion that there are at least two discrete forms of depression, endogenous/melancholic and reactive/neurotic, aligns with the disease model maintaining that there are separate conditions with etiological, symptom, course, and treatment differences [5]. In contrast to this binary view of depression is the unitary perspective that it is a single entity with various manifestations [1, 2].

Research strategies typically of a factor analytic nature have been applied over many years to ascertain the structure of depression. Eysenck [14] believed that the unitary/binary debate can be 'translated into factor analytic logic quite simply and clearly', with the unitary view predicting a single large general factor with positive loadings and no interpretable bipolar factor (not to be confused with bipolar disorder). In contrast, the binary position is supported by a bipolar factor involving endogenous and nonendogenous positions [14]. Eysenck [14] believed that the two-factor solution is supported. Parker et al. [1] reviewed major factor analytic studies relevant to the unitary/binary debate. Although interpretation of these studies can be challenging, there appears to be evidence for both positions, with Parker et al. [1] concluding that the strongest support is for an endogenous/melancholic type. According to Parker et al. [1], studies supporting a binary position are mainly describing endogenous and nonendogenous depressions, with the latter a mixture of anxiety, personality, and other nonspecific contributors.

Consistent with the disease model, discrete forms of depression should have different etiologies, symptom profiles, courses, and responses to treatment. Research, however, does not clearly distinguish endogenous/melancholic and reactive/neurotic depression based on these parameters [1–7, 15–22], nor has research supported dysthymia (and its continuation as persistent depressive disorder in DSM-5) and minor depression as distinct forms of depression [23–25]. No clear differences in etiology for the various types of depression proposed have been identified that hold out over repeated studies. Furthermore, one recent study examining levels of brain-derived neurotrophic factor (BDNF), tumor necrosis factor (TNF), adiponectin, and interleukin-6 (IL-6), all hypothesized to play a role in depression, found no differences between reactive depression, major depression (believed to be equivalent to endogenous depression), and bipolar depression [15]. Despite years of attempting to identify a clear symptom picture for endogenous depression, Parker [2] has been unable to clearly distinguish melancholic from nonmelancholic types.

Regarding course, research examining various indicators, such as risk of relapse, likelihood of recurrence, probability of readmission, and suicide, has overall failed to distinguish the two types of depression, with studies yielding varying results [6, 19–22]. For example, Kessing [6] did not find any differences in completed suicide, an important indicator of course. Furthermore, the course and symptom profile of depression often changes over time, fulfilling criteria for various subtypes including major, minor, and dysthymic depression [4, 26–28]. As pertains to treatment, early research by Parker [2] and his group suggested that endogenous/melancholic depression responded better to electroconvulsive therapy and tricyclic antidepressants. However, they later learned that their 'pristine' results were actually an artifact of aging, with older patients responding better to these two treatment modalities regardless of the type of depression [2].

Despite the lack of clear distinctions, there is some support, derived from select studies, for the position that melancholic depression is a subtype, based upon several criteria including: more extensive psychomotor impairment consisting of thought and physical slowing, anergia, anhedonia, suicide risk, agitation, fewer anxiety symptoms, higher cortisol levels and failure to suppress them with dexamethasone treatment, and a greater response to electroconvulsive therapy and tricyclic antidepressants combined with a lesser response to selective serotonin reuptake inhibitors [2, 7, 29–32]. So, where does all this leave us with regard to the crucial issue of depression as a discrete or continuous condition? On the one hand, there does not seem to be sufficient evidence for truly discrete types of depression, but on the other hand, there is persistent evidence for distinctions of some type. We have a quandary that is both intriguing and discouraging, the latter response arising from the struggle encountered in characterizing a disease process that has been extensively

researched. A potential resolution to this dilemma resides in a clarification of conceptual issues relevant to discrete and continuous descriptions of disease entities.

One very crucial concept that requires clarification is how well discrete and continuous categories apply to natural phenomena. Nature operates on the basis of continuums, whereas we prefer to see discreteness [33–35]. Even a straightforward single gene trait involving dominant and recessive alleles, such as eye color, which should be fully discrete with brown and blue categories, gravitates to a continuum evidenced by the phenotypes of hazel and green eye color when brown and blue genes mix. Variation in traits is the substance acted upon by natural selection allowing for their evolution [33]. Continuums provide for trait variation, whereas truly discrete entities do not. What this means is that if nature was organized discretely and not continuously, there would be no variation, and hence no evolution [33]. Traits lacking any variation (truly discrete) either persist if selection pressures favor the given characteristics or perish if not favored, an either or scenario. Ample trait variation provided by a continuous organization of forms allows for the most adaptive variant/s to become more represented in succeeding generations, the hallmark of natural selection and evolution.

Discovering that human sexual orientation is organized continuously, Kinsey et al. [34, 35] commented on how, despite our tendency to see discreteness, continuums characterize nature. In *Sexual Behavior in the Human Male* [34] they state, 'The living world is a continuum in each and every one of its aspects', and add in *Sexual Behavior in the Human Female* [35], 'It is a characteristic of the human mind that it tries to dichotomize in its classification of phenomena'. These statements capture how nature, human psychology included, is organized in a continuous fashion, while our perception of discrete categories is an illusion arising from a natural psychological inclination to dichotomize when classifying. Discrete entities are easier to process mentally, accuracy being traded off for simplicity. A formal statement that might be referred to as the 'continuum principle' is warranted, considering our automatic tendency to apply discreteness to what are almost universally continuous variables: natural phenomena tend to occur on a continuum, and any instance of hypothesized discreteness requires unassailable proof. Hence, any researcher or theorist positing discrete forms of depression must provide unambiguous evidence. Clearly, evidence for discrete types of depression does not even come close to achieving this standard.

A second very crucial conceptual issue requiring clarification is that continuous and discrete methods of categorization are ironically not as distinct as is often assumed. This occurrence arises from an important emergent property of quantitative dimensions: quantitative dimensions produce different qualitative states. For example, elevated blood sugar levels can be organized quantitatively: mild elevation involving a prediabetic condition, lacking overt disease manifestation; moderate elevation providing for diabetes resulting in pathological manifestations, such as in the vascular system; severe elevation producing prominent signs and symptoms, such as fluid loss related to osmotic diuresis. Dimensional quantitative variation yields qualitatively different disease states as an emergent property, with different treatment interventions being required. Mild elevation involving prediabetes only requires diet and activity alterations, moderate elevations require formal treatment with medications or insulin, and severe elevations constitute a medical emergency necessitating crisis care. Likewise, with varying quantitative levels of depression, different qualitative 'types' can manifest as an emergent property. Supporting this perspective, Schotte et al. [36] found that nonmelancholic and melancholic depression are organized continuously, with melancholic symptoms emerging as depression severity increases.

Applying clarification to the concepts of how well dimensions or discreteness apply to natural phenomena, and how quantitative differences yield qualitative variation as an emergent property, helps resolve our quandary regarding whether or not depression (or likely for that matter any psychiatric condition) is organized discretely or continuously. It represents a synthesis replacing the polar extremes, and one supporting a dimensional organization while allowing for some qualitative distinctions. Research and treatment approaches then need not be seen as impaired by a dimensional perspective [2]. It implies that adjectives, such as major, minor, dysthymia, endogenous, melancholic, and reactive, cannot be applied to depression. As a continuous entity, depression must be simply identified as 'depression' in any diagnostic system, although it can be understood that the more severe manifestations have a greater probability of producing classic melancholic signs and symptoms. Let us now look at depression as a dimensional entity.

The Dimensional Nature of Depression

Assuming, based on the prior discussion, that depression is organized continuously, the question remains as to how it is structured. Of crucial significance, characteriza-

tion of depression as a continuum must include duration and severity, supported by the intuitive tendency of every experienced clinician to consider these dimensions. Regarding duration, all else being equal, a depression of 2 years is different than a very short-lived episode. DSM-5 somewhat incorporates a time dimension with 'major depression' only necessitating 2 weeks of symptoms and 'dysthymia' and 'persistent depressive disorder' months [8, 9]. However, there is no requirement to note the exact time frame in major classification systems [8, 10]. As pertains to the severity of the depressive state, as we have seen, quantitative dimensions produce qualitatively different states as an emergent property. ICD-10 incorporates a severity dimension distinguishing between 'mild', 'moderate', and 'severe depression' [10]. DSM-IV and DSM-5 also do so to some extent with 'major', 'minor', and 'dysthymia' in DSM-IV, and 'major' and 'persistent depressive disorder' in DSM-5. Hence, applying duration and severity dimensions does not represent a radical departure from current classification systems, but does necessitate the removal of all adjectives leaving only 'depression'.

To give a truly comprehensive picture of an event, both duration and severity must be described in relationship to each other. For example, playing your favorite music at low volume for an hour or so is vastly different quantitatively and qualitatively than playing it at the highest volume for a day straight. Duration and severity might be rated low, moderate, and high, with the numbers 1, 2, and 3 assigned, respectively. The product of the assigned values gives a fairly accurate picture of the quantitative and qualitative differences. Your favorite music at low volume for an hour ($1 \times 1 = 1$) numerically is vastly different than the same music at high volume for a day straight ($3 \times 3 = 9$). Likewise, the duration and severity of a given depressive episode can both be rated using this scaling process. For example, a short-lived and low-severity episode can be listed as $1 \times 1 = 1$, and a long-lived and high-severity episode as $3 \times 3 = 9$. Between these two extremes are various scenarios such as shorter-range/high-severity episodes ($1 \times 3 = 3$), moderate-length/moderate-severity episodes ($2 \times 2 = 4$), short-duration/moderate-severity episodes ($1 \times 2 = 2$), moderate-duration/high-severity episodes ($2 \times 3 = 6$), and longer-range/mild-severity episodes ($3 \times 1 = 3$). Given that the same numeric value can describe different states, the ratings assigned for duration and severity should both be recorded. The ratings are for each episode and as such do not indicate recurrent episodes, necessitating that they be recorded separately.

As with varying quantitative music states producing different qualitative experiences, depressions of varying quantitative levels are different qualitatively. For example, a short-duration/mild-severity depression is much different qualitatively than a long-duration/high-severity episode. In the case of the former, there is typically much less overall suffering and disruption to the person's life compared to the latter. The use of three levels – low, moderate, and high (or mild, moderate, and severe) for the severity dimension, and short, moderate, and long for the duration dimension – is recommended, as opposed to scales with more gradients, because the greater the number of levels, the more difficulty is encountered in defining each and assigning the appropriate value. Extensive training is required that is unlikely to occur outside of a research setting. In contrast, most people, and certainly experienced clinicians, have an intuitive sense of what constitutes low, moderate, and high, simplifying rating for the severity dimension, and the three levels on the duration dimension can be defined in time units. Short might be set at less than 3 months, moderate at 3–6 months, and long at greater than 6 months, although research might have to establish the optimal time frame parameters for these three levels.

The validity of a dimensional approach applied to depression severity and duration has rarely been investigated. However, research by Kessing [37] focusing on the ICD-10 mild, moderate, and severe distinctions, supports a three-level rating system for severity: progressing from mild to moderate to severe, the risk of suicide and relapse increased significantly. In addition, greater stability of the diagnosis was found with moderate and severe ratings, highlighting the difficulty often encountered in separating depression from normal states, such as dysphoric mood.

While characterizing the duration dimension is quite straightforward given that it can be assessed in time units, the severity dimension requires more effort. As a starting point, we must consider whether or not the depressive dimension includes other conditions such as mania, anxiety, psychosis, and personality traits. Kraepelin viewed depression and mania as poles on the same dimension [3]. Mixed states involving both depressive and hypomanic/manic symptoms appear to be the norm and not the exception [38–43]. Kraepelin indicated: 'Very often we meet temporarily with states which do not exactly correspond either to manic excitement or to depression, but represent a mixture of morbid symptoms of both forms of manic-depressive insanity' [44].

Considering that hypomanic/manic and depressive symptoms are frequently part of a given episode, it was logical for Kraepelin to place them on the same dimen-

sion [44]. Unfortunately, problems arise from this structuring related to the nature of placement on the same dimension. One such issue consists of extremes of a dimension trading off against each other, with higher levels of depression meaning lower levels of mania and vice versa. Another related problem is that mixed states, represented by the mid-range on the dimension, must consist of less intense mania than when mania is on the extreme pole, and less severe depression than depression at the opposite pole. These statements are without validity because mixed states can involve depression and hypomania/mania of varying levels of intensity [38–43]. It might be suggested that placement on the same dimension only pertains to the presence or absence of manic and depressive symptoms, such that the manic symptoms are maximal at that pole and decline to the depressive pole. However, the dimension we are now considering is severity and not just presence of symptoms.

The same type of problem arises with a severity dimension incorporating anxiety, psychosis, and personality dimensions: polar opposites trade off against each other, and the mid-range involves less intense expressions than the poles. Once again, this arrangement as applied to the severity dimension is without validity. Depression and anxiety states, such as social anxiety, frequently overlap [45]. More severe depressive symptoms do not mean less intense anxiety symptoms and vice versa, nor do mixed states consist of less intense depressive and anxiety states than polar versions. What this logically leaves us with is separate continuums for depression, hypomania/mania, anxiety, psychosis, and personality traits, although these continuums can be present at the same time: depression with hypomania/mania, anxiety, psychosis, and personality problems.

Another critical issue pertaining to the characterization of the severity dimension is what exactly comprises it. If there are different types of depression, then the answer is extremely complicated with separate dimensions for each type. However, as we have seen, evidence does not support there being truly discrete forms [1–7, 15–22]. Ironically, evidence for a unitary dimension of depression severity actually comes from research used to support a discrete type of depression – melancholic/endogenous. Research and reviews of other factor analytic studies by Parker [2] and Parker et al. [1, 12] reveal a depressive factor, referred to by the authors as melancholic/endogenous, and a nonendogenous factor. The latter appears to encompass diverse symptoms including anxiety and personality expressions [1, 2, 12]. This pattern of results is used to support the position that a discrete form of depression – melancholic/endogenous – alone exists, with reactive/neurotic 'depression' only a composite of anxiety and personality symptoms [1, 2, 12]. A psychoanalytically orientated empirical study also supports this conclusion, only finding evidence for one type of depression (endogenous), with all other versions indistinguishable from nondepressed psychiatric states [46]. Given that discrete types of depression are not supported by the bulk of research, and certainly do not provide unassailable proof, the value of these results is evidence for a single dimension of depression severity.

How though are we to understand the content of the severity dimension? It is proposed that a concept originated by Kraepelin – depressive inhibition – with modifications be used to characterize the structure of the severity dimension. Kraepelin [47] believed that all the phenomenology of manic-depressive illness derives from depressive inhibition and that impediment of volition is the predominate clinical feature of depression: 'All action of the will is extremely difficult … the transformation of impulses of the will into action meets with obstacles that cannot be overcome without difficulty, and often not at all by the patient's own strength'. Current research data indicate that depressive inhibition involves impeded cognitive, emotional, social, and physical behavior. Cognitive consisting of thought slowing and impairments to attention, memory, and learning processes (executive functions); emotional entailing diminished positive emotions, excessive negative emotions, restricted affective range, and emotional information processing limitations; social consisting of reduced social activity and impaired social cognition; physical including tiredness, fatigue, lethargy, sleep problems, appetitive changes, and psychomotor slowing [47–52]. If a person does not experience substantial impairment with regard to thought processes, emotional states providing for hope and motivation, social interactions, or physical behavior, we typically do not diagnose depression. Perhaps, a low mood state or uncomplicated bereavement can be diagnosed, but depression does not exist in the absence of depressive inhibition, supporting the robustness of this concept. Depressive inhibition aligns well with the notion that severe depression involves manifestations such as psychomotor impairment, anergia, and anhedonia [2].

The validity of relying on depressive inhibition to characterize the severity dimension of depression is supported by fundamental motivational parameters, known as the behavioral activation system (BAS) and behavioral inhibition system (BIS). BAS and BIS are very ancient general motivational systems, with the former approach oriented

and based on positive appetitive incentive and the latter regulating sensitivity to threat and nonreward cues and guiding inhibition or avoidance responses [53–56]. BAS and positive affectivity are related concepts, sometimes used interchangeably, as are BIS and negative affectivity [53, 55]. Positive and negative affectivity can be viewed as expressions of BAS and BIS, respectively [55]. BIS is increased in a range of psychiatric conditions including depression, anxiety, schizophrenia, and disordered attachment states [55, 57–59]. BAS, on the other hand, appears more specific to depression with lower levels found in this condition [54, 55]. BIS and BAS typically operate in a fashion that is complementary, even synergistic. For example, increased aversive motivation lowers appetitive motivation [54]. The combination of high BIS and low BAS likely contributes to several symptoms of depression, including loss of energy resulting in fatigue, increased sleep, diminished interest and motivation, impaired concentration, psychomotor impairment consisting of thought and physical slowing, and social withdrawal.

Although the combination of high BIS and low BAS contributes to depressive symptoms, it should not be viewed as being synonymous with depressive inhibition. A prominent difference consists of depressive inhibition being quite resilient to BIS/BAS-relevant stimuli: while a state of high BIS and low BAS can resolve quite rapidly with stimuli favoring high behavioral activation and low behavioral inhibition, as evidenced by the response of seasonal affective disorder (SADS) to bright sunny conditions, depressive inhibition tends to persist [60]. This occurrence suggests that other factors play a major role in depressive inhibition, the contributors including genetic, epigenetic, and neural influences [61–64].

Despite only being one contributor to depressive inhibition, the high behavioral inhibition and low behavioral activation nature of depression aligns well with, and adds support to, the proposition that depressive inhibition can be used to characterize the severity dimension of depression. BIS/BAS scales exist helping facilitate the application of these motivational parameters to depressive inhibition [65, 66]. In addition, measurement strategies can be devised to assess the different components of depressive inhibition (cognitive, emotional, social, and physical) based on low, moderate, and high severity ratings. By averaging the severity ratings for the different components of depressive inhibition, a composite score for depression severity can be arrived at. Treatment interventions tailored to the specific depressive inhibition impairments can be designed and tested. Furthermore, the BIS/BAS scales can be applied to improve behavioral activation treatments [66] oriented to increasing activity and access to reinforcement. The two main versions, behavioral activation and behavioral activation treatment for depression [67–69], are highly effective for depression [70–73] and work just as well for cognitions as cognitive behavioral therapy [68].

Hence, the dimensional nature of depression is captured by the duration × severity model, with severity based upon depressive inhibition in terms of cognitive, emotional, social, and physical behavior. Rating duration and severity on three levels provides a measure of each dimension. The product of these values provides invaluable information pertaining to the quantitative and related qualitative aspects of a depressive episode. Two important additions to this model are, first, how to incorporate depression related to specific circumstances and, second, explaining mixed symptoms.

Depression Related to Specific Circumstances

Depression appears to exist as a continuum, but it can be triggered by diverse sources including genetic vulnerabilities, social stressors, physiological disease processes, and environmental cues relevant to behavioral inhibition and behavioral activation (BIS and BAS, respectively), the latter producing SADS [60]. SADS appears to involve a sensitivity to environmental cues relevant to BIS and BAS, with low light levels and related winter stimuli, such as cold days with few signs of life, increasing behavioral inhibition and reducing behavioral activation, thereby contributing to the onset of depression [60]. It might be opinioned that SADS represents a different subtype of depression, but as with other proposed subtypes, there is too much symptom overlap. For example, with SADS, so-called 'atypical' depressive symptoms involving excessive sleep and carbohydrate cravings occur, but many people with non-SADS depression oversleep and consume high-calorie sweetened foods over healthier alternatives, this occurrence contributing to the problem of excess weight found in those with depression [74]. Furthermore, research, such as by Hansen et al. [75], fails to distinguish SADS as a separate disease entity. Examining a population in Northern Norway living without sun for 2 months, Hansen et al. [75] did not find evidence that depression is higher than in any other population in winter and suggested that 'depression with seasonal pattern' be used to describe depression that recurs in winter [75]. Hence, it appears that the depressive component of SADS does not represent a distinct clinical entity.

As with BIS/BAS-relevant environmental stimuli, social stressors, such as trauma, and physical disease processes, such as hypothyroidism or cancer, can trigger depression. This process might best be conceptualized as various triggering or eliciting mechanisms inducing depression. Reversal of those inputs can improve or resolve depression. For example, with SADS, reversal of cues for behavioral inhibition/behavioral activation (high BIS and low BAS cues shifting to low BIS and high BAS) can remedy the depressive state. Likewise, with physical illness such as hypothyroidism, reversal of the disease process helps resolve the depressive illness. For example, correcting the thyroid hormone deficiency can improve or remedy depression in this instance. If depressive inhibition becomes too severe, reversal of the input mechanism is often insufficient to reverse it and other means, such as antidepressant medication, are required.

Mixed Symptoms

Depression is commonly mixed with anxiety, hypomanic/manic symptoms, psychosis in some instances, and personality disorder symptoms [28, 44, 45, 47–52]. There are different ways to conceptualize this occurrence. One way, based on the interpretation of factor analytic studies, is that of Watson [76], who views certain types of anxiety (generalized anxiety disorder and posttraumatic stress disorder) and depression (major and dysthymic) as linked on the basis of distress (distress disorders). Meanwhile, panic disorder, agoraphobia, social phobia, and specific phobia are distinct as 'fear' disorders, with bipolar disorders also separate. There are several problems with this organization, a major one being that it fails to consider the different emotional information processing underlying depression and anxiety – depression as amplified sadness, with the root emotion triggered by loss-oriented cognitive activating appraisals, and anxiety as amplified fear, with the root emotion derived from threat- or danger-oriented cognitive activating appraisals [77–85]. Another issue is that depression frequently coexists with 'fear' conditions such as social phobia [45]. There is even the possibility that, in many instances, social anxiety actually represents the primary trigger for depression [86]. Furthermore, panic disorder, agoraphobia, social phobia, and specific phobia can involve equal or greater levels of distress than with generalized anxiety disorder, posttraumatic stress disorder, or depression. For example, a person with a phobia of elevators who has to take them every day at work can be very distressed.

Another way to conceptualize mixed states, consistent with how nature is organized dimensionally, is the separate continuums (proposed earlier) for depression, anxiety, hypomania/mania, psychosis, and personality disorders, interfacing with each other. The linkage of depression and anxiety is understandable based on overlapping emotional information processing: circumstances involving loss resulting in sadness also frequently entail threat or danger triggering fear, such as when being bullied. Depression and anxiety continuums will then naturally interface with one another. Although many researchers prefer to see discrete types of anxiety, a continuum including duration and severity dimensions almost certainly applies. Panic disorder, much like major depression, tends to be of shorter duration and high severity, while generalized anxiety disorder is of longer duration and lesser severity similar to DSM-IV-TR dysthymia [8].

Hypomania and mania occur on a continuum ranging from subsyndromal hypomania to hypomania to subsyndromal mania to mania [60]. Hypomania appears to exist on a one-to-one ratio with depression [87, 88], with mania much less common [89]. One possible way to conceptualize this occurrence is the depression and hypomania/mania dimensions interfacing on the basis of hypomania providing for the most part a defensive compensation for depression, perhaps helping to temporarily override or interrupt depressive inhibition states to restore adaptive functioning in the moment [60]. Mania itself might arise from defective cognitive regulatory control processes allowing compensatory hypomania to progress to dysfunctional mania [60]. A key feature of mania contributing to its dysfunctional nature is psychosis. Psychosis represents the extreme end of a normal continuum [90–92]. When depression is accompanied by psychosis, at least in the absence of bipolar I (depression and mania) or schizoaffective disorder, the depressive state tends to be very severe [5]. Psychosis has been proposed to arise from impaired or damaged cognitive regulatory control processes that normally block psychotic-level cognitive distortions, thought form variants, and sensory perceptual experiences from the conscious and awake state, in order to facilitate reality congruency necessary for adaptive functioning [92]. With severe depression, it is feasible that the relevant cognitive regulatory control processes might be impaired, allowing for the expression of psychosis.

Personality disorder expressions also occur with depression. Disorders of personality are part of a continuum from normal to abnormal [93]. Given the dysfunctional and often self-defeating behavior of those with personality disorders, losses triggering sadness and

depression are to be expected. In addition, with depression, a person's coping capacity often defaults to the lowest level, translating into highly dysfunctional behavior for those with personality disorders, in turn producing further losses and more depression. For these reasons, the depressive and personality disorder continuums often interface with each other. The nonendogenous factor identified by Parker [2] and Parker et al. [1, 12], including diverse symptoms, then appears to represent anxiety, hypomanic/manic, psychotic, and personality disorder continuums interfacing with the depressive continuum.

Depression: Dimensional and Not Discrete

The concept of nature working on the basis of continuums despite our insistence on discreteness strongly suggests that depression is dimensional. Research evidence overwhelmingly supports a dimensional interpretation, although quantitative variation can result in qualitative differences as an emergent property, fostering the appearance of discreteness. It might still be argued that true discreteness applies in certain regards. One possibility is with specific circumstances related to depression. These can be discrete and trigger depression in vulnerable individuals. For example, some people are sensitive to environmental cues for BAS and BIS, such as low light levels, resulting in depression. However, the depression itself is dimensional and is the same as that arising from physiological and psychological stressors.

The integrity of discrete models might be upheld by other psychological variables, with one of the most promising being human sexual orientation. Discrete homosexual and heterosexual identities have commonly been ascribed [94], although bisexuality detracts somewhat from the possibility of full discreteness. Prior to industrialization (and also in some modern South Pacific cultures), sexual orientation involved homoerotic and heteroerotic behaviors, as opposed to homosexual and heterosexual identities [95–97]. With industrialization, there has been a shift to sexual orientation identities, often framed in discrete terms [94]. As shown by Kinsey et al. [34, 35], these sexual orientation identities are actually dimensional – homosexuality and heterosexuality on opposite poles of a single continuum. As it turns out, conceptual and practical difficulties with this arrangement support sexual orientation as being even more dimensional than Kinsey et al. [34, 35] realized: on a single dimension, bisexuality must be placed in the middle, meaning that bisexuals are less homosexual than 'full' homosexuals and less heterosexual than 'full' heterosexuals, a completely false scenario given that many bisexuals have equal or stronger motivations than homosexuals and heterosexuals [98, 99]. Even more difficulty is encountered in placing asexuals, the fourth sexual orientation category. If placed on the scale, they must be positioned with bisexuals in the middle, a completely ludicrous scenario. The option chosen by Kinsey et al. [34, 35] was to place them off the scale, an arrangement that weakens the applicability of the scale and model. Logically, what we are left with is two dimensions – homoerotic and heteroerotic – with asexuals to the zero or low end, homosexuals having a strong homoerotic motivation and low heteroerotic motivation, heterosexuals the reverse motivational profile, and bisexuals substantial motivation on both dimensions [98–100]. Hence, human sexual orientation is actually highly dimensional, supporting the principle that human psychology gravitates to continuums.

Based on the existing evidence, it appears that nature (and certainly psychological variables) has a strong tendency to be organized dimensionally. The depression continuum is best characterized by duration and severity dimensions, with the latter understood in terms of depressive inhibition. In the absence of some degree of cognitive, emotional, social, and physical inhibition, depression should not be diagnosed. Combining the dimensions of duration and severity provides an optimal way to characterize the quantitative and related qualitative aspects of depression and describe the overall degree of dysfunction. The mixing of depression with other symptom types appears to involve anxiety, hypomanic/manic, psychotic, and personality disorder continuums interfacing with the depression continuum. Moving ahead, empirical and theoretical research efforts are best directed towards a more refined analysis of the dimensional structure of depression and the development of specific strategies for remedying the various components of depressive inhibition.

References

1 Parker G, Hall W, Boyce P, Hadzi-Pavlovic D, Mitchell P, Wilhelm K: Depression sub-typing: unitary, binary, or arbitrary? Aust NZ J Psychiatry 1991;25:63–76.
2 Parker G: A case for reprising and redefining melancholia. Can J Psychiatry 2013;58:183–189.
3 Benazzi F: Various forms of depression. Dial Clin Neurosci 2006;8:151–161.
4 Kessing LB: Epidemiology of subtypes of depression. Acta Psychiatr Scand 2007; 115(suppl 433):85–89.
5 Paykel ES: Basic concepts of depression. Dial Clin Neurosci 2008;10:279–289.

6 Kessing LV: Endogenous, reactive and neurotic depression – diagnostic stability and long-term outcome. Psychopathology 2004; 37:124–130.
7 Healy D: Melancholia: past and present. Can J Psychiatry 2013;58:190–194.
8 First MB, Frances A, Pincus HA: DSM-IV-TR: Handbook of Differential Diagnosis. Washington, American Psychiatric Publishing, 2002.
9 American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition: DSM-5. Washington, American Psychiatric Publishing, 2013.
10 World Health Organization: The ICD-10 Classification of Mental and Behavioral Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva, World Health Organization, 1992.
11 Kiloh LG, Andrews G, Neilson M, Bianchi GN: The relationship of the syndromes called endogenous and neurotic depression. Br J Psychiatry 1972;121:183–196.
12 Parker G, Blignault I, Manicavasagar V: Neurotic depression: delineation of symptom profiles and their relation to outcome. Br J Psychiatry 1988;152:15–23.
13 Shorter E: A History of Psychiatry. Toronto, John Wiley & Sons, 1997.
14 Eysenck HJ: The classification of depressive illness. Br J Psychiatry 1970;117:241–250.
15 Su S, Sun M, Wen M, Lin C, Chen Y, Hung Y: Brain-derived neurotrophic factor, adiponectin, and proinflammatory markers in various subtypes of depression in young men. Int J Psychiatry Med 2011;42:211–226.
16 Welch GW, Ellis PM: FACTOREP: a new tool to explore the dimensions of depression. J Affect Disord 1991;21:101–108.
17 Bech P: Rating scales for mood disorders: applicability, consistency, and construct validity. Acta Psychiatr Scand Suppl 1988;345:45–55.
18 Bronisch T, Klerman GL: The current status of neurotic depression as a diagnostic category. Psychiatr Dev 1988;6:245–275.
19 O'Leary D: The endogenous subtype and naturalistic course in depression. J Affect Disord 1996;41:117–123.
20 Ramana R, Paykel ES, Cooper Z, Hayhurst H, Saxty M, Surtees PG: Remission and relapse in major depression: a two-year prospective follow-up study. Psychol Med 1995;25:1161–1170.
21 Surtees PG, Barkley C: Future imperfect: the long-term outcome of depression. Br J Psychiatry 1994;164:327–341.
22 Brodaty H, Luscombe G, Peisah C, Anstey K, Andrews G: A 25-year longitudinal, comparison study of the outcome of depression. Psychol Med 2001;31:1347–1359.
23 Rhebergen D, Graham R: The re-labelling of dysthymic disorder to persistent depressive disorder in DSM-5: old wine in new bottles? Curr Opin Psychiatry 2014;27:27–31.
24 Rhebergen D, Graham R, Hadzi-Pavlovic D, Stek M, Friend P, Barrett M, Parker G: The categorisation of dysthymic disorder: can its constituents be meaningfully apportioned? J Affect Disord 2012;143:179–186.
25 Newman SC, Bland RC: A population-based family study of minor depression. Depress Anxiety 2009;26:389–392.
26 Angst J, Sellaro R, Merikangas KR: Depressive spectrum diagnoses. Compr Psychiatry 2000; 41(suppl):39–47.
27 Angst J, Merikangas KR: The depressive spectrum: diagnostic classification and course. J Affect Disord 1997;45:31–39.
28 Judd LL, Akiskal HS, Maser JD: A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. Arch Gen Psychiatry 1998;55:694–700.
29 Harris M, Farquhar F, Healy D, LeNoury J, Baker D, Whitaker C, Linden S, Green P, Roberts AP: The incidence and prevalence of admissions for melancholia in two cohorts (1875–1924 and 1995–2005). J Affect Disord 2011;134:45–51.
30 Harris M, Farquhar F, Healy D, LeNoury J, Linden S, Hughes JA, Roberts AP: The morbidity and mortality linked to melancholia: two cohorts compared, 1875–1924 and 1995–2005. Hist Psychiatry 2013;24:3–14.
31 Parker G, Fletcher K, Barrett M, Synnott H, Breakspear M, Rees AM, Hadzi-Pavlovic D: Inching toward Bethlehem: mapping melancholia. J Affect Disord 2010;123:291–298.
32 Parker G, McCraw S, Blanch B, Hadzi-Pavlovic D, Synnott H, Rees AM: Discriminating melancholic and non-melancholic depression by prototypic clinical features. J Affect Disord 2013;144:199–207.
33 Darwin C: The Origin of Species. New York, Signet Classic, 1858/1958.
34 Kinsey AC, Pomeroy WB, Martin CE: Sexual Behavior in the Human Male. Philadelphia, WB Saunders, 1948.
35 Kinsey AC, Pomeroy WB, Martin CE, Gebhard PH: Sexual Behavior in the Human Female. Philadelphia, WB Saunders, 1953.
36 Schotte CK, Maes M, Cluydts R, Cosyns P: Cluster analytic validation of the DSM melancholic depression. The threshold model: integration of quantitative and qualitative distinctions between unipolar depressive subtypes. Psychiatry Res 1997;71:181–195.
37 Kessing LV: Severity of depressive episodes according to ICD-10: prediction of risk of relapse and suicide. Br J Psychiatry 2004;184: 153–156.
38 Marneros A: Origin and development of concepts of bipolar mixed states. J Affect Disord 2001;67:229–240.
39 Carlson GA, Goodwin FK: The stages of mania: a longitudinal analysis of the manic episode. Arch Gen Psychiatry 1973;28:221–228.
40 Cassidy F, Murry E, Forest K, Carroll BJ: Signs and symptoms of mania in pure and mixed episodes. J Affect Disord 1998;50:187–201.
41 Akiskal HS, Hantouche EG, Bourgeois ML, Azorin JM, Sechter D, Allilaire JF, Lancrenon S, Fraud JP, Liliane CD: Gender, temperament, and the clinical picture in dysphoric mixed mania: findings from a French national study (EPIMAN). J Affect Disord 1998;50: 175–186.
42 Benazzi F, Akiskal HS: Delineating bipolar II mixed states in the Ravenna-San Diego collaborative study: the relative prevalence and diagnostic significance of hypomanic features during major depressive episodes. J Affect Disord 2001;67:115–122.
43 Bauer MS, Whybrow PC, Gyulai L, Gonnel J, Yeh HS: Testing definitions of dysphoric mania and hypomania: prevalence, clinical characteristics and inter-episode stability. J Affect Disord 1994;32:201–211.
44 Freeman MP, McElroy SL: Clinical picture and etiologic models of mixed states. Psychiatr Clin North Am 1999;22:535–546.
45 Fava M, Rankin MA, Wright EC: Anxiety disorders in major depression. Compr Psychiatry 2000;41:97–102.
46 Cochrane N: A contribution from the field of psychodynamics to the controversy over reactive and endogenous depression. Br J Med Psychol 1977;50:87–94.
47 Himmelhoch JM: Social anxiety, hypomania and the bipolar spectrum: data, theory and clinical issues. J Affect Disord 1998;50:203–213.
48 Benazzi F: Factor structure of recalled DSM-IV hypomanic symptoms of bipolar disorder. Compr Psychiatry 2004;45:441–446.
49 Benazzi F: Is overactivity the core feature of hypomania in bipolar II disorder? Psychopathology 2007;40:54–60.
50 Bilder RM, Goldman RS, Robinson D, Reiter G, Bell L, Bates JA: Neuropsychology of first-episode schizophrenia: initial characterization and clinical correlates. Am J Psychiatry 2000;157:549–559.
51 Elvevag B, Goldberg TE: Cognitive impairment in schizophrenia is the core of the disorder. Crit Rev Neurobiol 2000;14:1–21.
52 Fiszdon JM, Richardson R, Greig T, Bell MD: A comparison of basic and social cognition between schizophrenia and schizoaffective disorder. Schizophr Res 2007;91:117–121.
53 Gray JA: Perspectives on anxiety and impulsivity: a commentary. J Res Pers 1987;21:493–509.
54 Fowles DC: Psychophysiology and psychopathology: a motivational approach. Psychophysiology 1988;25:373–391.
55 Kasch KL, Rottenberg J, Arnow BA, Gotlib IH: Behavioral activation and inhibition systems and the severity and course of depression. J Abnorm Psychol 2002;111:589–597.
56 Johnson SL, Sandrow D, Meyer B, Winters R, Miller I, Solomon D, Keitner G: Increases in manic symptoms after life events involving goal attainment. J Abnorm Psychol 2000;109: 721–727.
57 Campbell-Sills L, Liverant GI, Brown TA: Psychometric evaluation of the behavioral inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. Psychol Assess 2004;16: 244–254.

58 Mineka S, Watson D, Clark LA: Comorbidity of anxiety and unipolar mood disorders. Ann Rev Psychol 1998;49:377–412.
59 Scholten MR, van Honk J, Aleman A, Kahn RS: Behavioral inhibition system (BIS) and the behavioral activation system (BAS) and schizophrenia: relationship with psychopathology and physiology. J Psychiatr Res 2006; 40:638–645.
60 Bowins BE: Hypomania: a depressive inhibition override defense mechanism. J Affect Disord 2008;109:221–232.
61 Azorin JM, Kaladjian A, Fakra E, DaFonseca D, Adida M, Maurel M, Richieri R, Bottai T, Pringuey D: Gene-environment interactions in affective disorders. Encephale 2010; 36(suppl 6):162–172.
62 Du X, Pang TY, Hannan AJ: A tale of two maladies? Pathogenesis of depression with and without the Huntington's disease gene mutation. Front Neurol 2013;4:81.
63 Treadway MT, Pizzagalli DA: Imagining the pathophysiology of major depressive disorder – from localist to circuit-based analysis. Biol Mood Anxiety Disord 2014;4:5.
64 Pandya M, Altinay M, Malone DA, Anand A: Where in the brain is depression? Curr Psychiatry Rep 2012;14:634–642.
65 Carver CL, White TL: Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. J Pers Soc Psychol 1994;67: 319–333.
66 Bowins BE: Augmenting behavioural activation treatment with the behavioural activation and inhibition scales. Behav Cogn Psychother 2012;40:233–237.
67 Hopko DR, Lejuez CW, Ruggiero KJ, Eifert GH: Contemporary behavioral activation treatments for depression: procedures, principles, and progress. Clin Psychol Rev 2003; 23:699–717.
68 Jacobson NS, Dobson KS, Truax PA, Addis ME, Koerner K, Gollan JK: A component analysis of cognitive-behavioral treatment for depression. J Consult Clin Psychol 1996;64: 295–304.
69 Lejuez CW, Hopko DR, Hopko SD: A brief behavioral activation treatment for depression. Behav Modif 2001;25:255–286.
70 Kanter JW, Puspitasari AJ, Santos MM, Nagy GA: Behavioural activation: history, evidence, and promise. Br J Psychiatry 2012;200:361–363.
71 Ly KH, Truschel A, Jarl L, Magnusson S, Windahl T, Johansson R, Carlbring P, Andersson G: Behavioural activation versus mindfulness-based guided self-help treatment administered through a smartphone application: a randomised controlled trial. BMJ 2013;4: e003440.
72 McEvoy P, Law A, Bates R, Hylton K, Mansell W: Using behavioural activation in the treatment of depression: a control theory perspective. J Psychiatr Ment Health Nurs 2013;20: 890–895.
73 Chartier S, Provencher MD: Behavioural activation for depression: efficacy, effectiveness and dissemination. J Affect Disord 2013;145: 292–299.
74 Davidson K, Kaplan B: Nutrient intakes are correlated with overall psychiatric functioning in adults with mood disorders. Can J Psychiatry 2012;57:85–92.
75 Hansen V, Skre I, Lund E: What is this thing called 'SAD'? A critique of the concept of seasonal affective disorder. Epidemiol Psychiatr Soc 2008;17:120–127.
76 Watson D: Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM-V. J Abnorm Psychol 2005;114:522–536.
77 Bowins BE: Psychological defense mechanisms: a new perspective. Am J Psychoanal 2004;64:1–26.
78 Bowins BE: How psychiatric treatments can enhance psychological defense mechanisms. Am J Psychoanal 2006;66:173–194.
79 Finlay-Jones R, Brown G: Types of stressful life event and the onset of anxiety and depressive disorders. Psychol Med 1981;11:803–815.
80 Shrout P, Link B, Dohrenwend B, Skodol A, Stueve A, Mirotznik J: Characterizing life events as risk factors for depression: the role of fateful loss events. J Abnorm Psychol 1989; 98:460–467.
81 Eley T, Stevenson J: Specific life events and chronic experiences differentially associated with depression and anxiety in young twins. J Abnorm Child Psychol 2000;28:383–394.
82 Beck A: Cognitive therapy: a 30-year retrospective. Am Psychol 1991;46:368–375.
83 Lazarus R: Cognition and emotion in motivation. Am Psychol 1991;46:352–367.
84 Lazarus R: On the primacy of cognition. Am Psychol 1984;39:124–129.
85 Clore GL, Ortony A: Cognition in emotion: always, sometimes, or never?; in Lane RD, Nadel L (eds): Cognitive Neuroscience of Emotion. New York, Oxford University Press, 2000, pp 24–61.
86 Schneier FR, Blanco C, Antia SX, Liebowitz MR: The social anxiety spectrum. Psychiatr Clin North Am 2002;25:757–774.
87 Akiskal HS: The prevalent clinical spectrum of bipolar disorders: beyond DSM-IV. J Clin Psychopharmacol 1996;16:4S–15S.
88 Akiskal HS: Searching for behavioral indicators of bipolar II in patients presenting with major depressive episodes: the 'red sign,' the 'rule of three' and other biographic signs of temperamental extravagance, activation and hypomania. J Affect Disord 2005;84:279–290.
89 Weissman MM, Myers JK: Affective disorders in a US urban community. Arch Gen Psychiatry 1978;35:1304–1311.
90 Chapman LJ, Chapman JP: Scales for rating psychotic and psychotic-like experiences as continua. Schizophren Bull 1980;6:476–489.
91 Strauss JS: Hallucinations and delusions as points on continua function. Arch Gen Psychiatry 1969;21:581–586.
92 Bowins BE: A cognitive regulatory control model of schizophrenia. Brain Res Bull 2011; 85:36–41.
93 Bernstein DP, Iscan C, Maser J: Opinions of personality disorder experts regarding the DSM-IV personality disorders classification. J Personal Disord 2007;21:536–551.
94 Weeks J: Sexuality and Its Discontents. London, Routledge and Kegan Paul, 1985.
95 Thorp J: The social construction of homosexuality. Phoenix 1992;46:54–65.
96 Herdt GH: Cross-cultural forms of homosexuality and the concept 'gay'. Psychiatr Ann 1988;18:37–39.
97 Bhugra D, McMullen I, Popelyuk D: Paraphilias across cultures: contexts and controversies. J Sex Res 2010;47:242–262.
98 Storms MD: Theories of sexual orientation. J Pers Soc Psychol 1980;38:783–792.
99 Shively MG, De Cecco JP: Components of sexual identity. J Homosex 1977;3:41–48.
100 Muscarella F: The homoerotic behavior that never evolved. J Homosex 1999;37:1–18.