SPECIAL SECTION: NORMAL AND ABNORMAL EMOTIONS—THE QUANDARY OF DIAGNOSING AFFECTIVE DISORDER

In Between Ordinary Sadness and Clinical Depression

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Abstract

Since Kraeplin and Kretschmer, the clarification of the limits between ordinary sadness and clinical depression has been a major concern. Much of the controversy has focused on whether and on which bases can be fixed a boundary in the continuum from the experience of sadness to major depressive episode. The new emphasis on the role of clinical judgment introduced by DSM-5 can be regarded as a way to address these issues, though leaving several questions open. After examining the implications of the main topics raised by this still ongoing discussion, we will argue that in a clinical reality both mobility and intensity of emotional states may account for the discontinuity between ordinary sadness and clinical depression.

Keywords

bereavement, depressive personality disorder, major depressive episode, sadness

Introduction

One of the most significant novelties introduced by DSM-5 within the field of depressive disorders is bereavement-related depression. In this way DSM-5 removes DSM-IV exclusion criteria for a major depressive episode that was applied to depressive symptoms lasting less than 2 months following the death of a loved one. As a consequence, bereavement, the intense sadness that follows the death of a loved one, is now regarded as a mental disorder. Indeed, it possesses certain clinical characteristics (e.g., feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss) which make it qualify for a diagnosis of major depression. Therefore, the chief question is on what basis bereavement-triggered and other loss-triggered forms of uncomplicated intense sadness (e.g., no psychotic ideation, no suicidal ideation, no psychomotor retardation, etc.)

should be included or excluded from the diagnosis of major depression. What is at stake here is the continuity and boundary between deep sadness, bereavement, and depression, and the treatment effectiveness.

On the other hand a note included in the DSM-5 criteria for major depressive disorder states that,

[R]esponses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include feelings of intense sadness, rumination about the loss, insomnia, poor appetite and weight loss, which may resemble a depressive episode. (American Psychiatric Association, 2013, p. 134)

And that the decision about whether a major depressive episode (or just a normal response to the loss) is present "inevitably

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Emotion Review Vol. 7, No. 3 (July 2015) 1–7 © The Author(s) 2015 ISSN 1754-0739 DOI: 10.1177/1754073915575405 er.sagepub.com requires the exercise of clinical judgment based on what the clinician knows about the individual in question and the individual's cultural norms for the expression of distress in the context of loss" (Maj, 2013, p. 90).

Hence, two key questions are raised. On one hand a first and more general question concerning the limits between normal reactions to major life events and mental pathology. On the other hand the second question concerns the criteria that should guide the clinician in drawing the line between "normal and abnormal emotions." Indeed, the clinician is left to make the decision on whether the sadness response of the individual is proportional to the triggering situation or not, a decision based on the knowledge that she/he has of the individual, of her/his life story, and of her/ his personal experience.

In the attempt to give an answer and outline our position we will first offer a fresh perspective on the continuity/discontinuity between deep sadness, bereavement, and depression. We will then illustrate the different experiences of sadness, in an attempt to draw the line between typical and atypical sad reactions. Throughout the article we will refer to literature from imaging genetics, behavioral neuroscience, and psychology. Far from being a comprehensive review, we have selected works that we believe help to refine our approach to the subject matter here debated. We will end by placing the debate into the context of the clinical reality, suggesting why and how the personal story is the source allowing one to disentangle between normal sadness and psychopathology.

Sadness, Bereavement, and Depression: Continuity and Discontinuity

The question concerning the continuity and the discontinuity between sadness, bereavement, and depression raises a more profound issue, which concerns the very history of the conceptualization of the disorder within psychiatry and more generally clinical psychology.

Freud (1917) was the first to introduce the relation between bereavement and melancholy, thus making a major contribution to the understanding of the disorder.

Furthermore, in the same century, sadness, depressive temperament, and depression entered contemporary psychiatric discourse in the wake of the German tradition of Kraepelin (1921) and later Kretschmer (1926). Both—albeit with different emphasis—postulated a "depressive temperament" as the basis of the manic-depressive illness. People with this temperament are characterized as being predominantly gloomy, sad, despondent, dejected, shy, inadequate, quiet, conscientious, serious, fatigued, lacking initiative and vitality, etcetera.

Kretschmer, however, thought that having a "depressive temperament" is not enough to incline a person towards melancholy: while such a temperament may predispose the individual towards illness, what proves decisive is the actual experience that a person lives through. That is to say: it is not that the temperament itself is sad, but only that the person "is more easily roused by a sad condition" (1926, pp. 129–130).

This conception of a depressive temperament which can evolve to a form of mild chronic depression, was incorporated in the classification of depressive disorders with the classification system DSM-III (Akiskal, 2001): this nosological entity was called "dysthymia." Dysthymia was therefore considered as a discontinuity of character and included in Axis I, the DSM's section where mental disorders that are not related to personality disorders are classified. In this respect, it was distinguished from other characterologically based depressions (which did not respond to antidepressant medications) chiefly on the basis of its response to treatment.

DSM-IV later continued to keep dysthymia on Axis I; however, in its Appendix B, it included research criteria for depressive personality disorders (DPD), thus reintroducing the Kretchmerian question of the relation of this construct to both mood disorders and normal character. In the DSM-5 both chronic major depressive disorder and the previous dysthymic disorder are included under the category of persistent depressive disorder.

A series of questions emerge. The most important concerns the relation between a particular nonpathological character type which in the course of one's development engender recurrent ways of feeling sadness—called by several authors "depressionprone style of personality" (Arciero & Bondolfi, 2009; Arciero & Guidano, 2000; Guidano, 1987, 1991)—and the DPD, understood as a pathological condition of one's character. How to account for these two ways of being? How does a particular way of feeling over time become sedimented as a certain tendency to react emotionally that corresponds to the depression-prone style of personality? What relation exists between this "normal" style of personality and the "abnormal" DPD?

Enduring Dispositions

In his essay entitled "Mourning and Melancholia" (1917), Freud touches upon two themes which are crucial in order to understand depressive reactions. The first is the strong similarity between bereavement and melancholy; the second is the hypothesis that this similarity may be due to a loss suffered in childhood, which would thus lie at the basis of the individual's predisposition toward melancholy.

The relation suggested by Freud between early loss and vulnerability to the depressive disorder was the focus of various longitudinal studies made about half a century later (Brown & Harris, 1978; Harris, Brown, & Bifulco, 1986).

The most interesting element which these studies—developed in the context of attachment theory (Bowlby, 1980)—bring to light is the fact that a chronic condition of lack of care (ranging from indifference to detachment, contempt, hostility, and maltreatment) may affect the development of one's personality, to the point of making it more vulnerable to depression.

The repetition of several negative experiences (loss, separation, rejection, etc.) and the sedimentation of the ways of feeling correlated to these circumstances over time as character traits, may thus predispose individuals to develop a depression-prone style of personality or to suffer from DPD (Arciero & Bondolfi, 2009; Arciero & Guidano, 2000; Guidano, 1987, 1991). This process may account for the development of those enduring dispositions that both Kraepelin (1921) and Kretschmer (1926) believed to lie at the basis of the depressive temperament. These tendencies, however, far from being genetically determined, are seen to arise from recurrent experiences that present emotional characteristics similar to those of bereavement—as Freud had already sensed—but which can be more clearly traced back to chronic conditions of rejection and poor social support of various levels of intensity.

The crucial role and the relevance of the actual experience and of individual personal history appear to find confirmation in several genetic studies. One of the more extensively investigated examples of gene–environment interaction, is the influence of the gene that codes for the serotonin transporter (5-HTTLPR) on depressive responses to life stress.

These researches have demonstrated a link between the serotonin transporter (5-HTTLPR, short allele) and the onset of depression following adverse life experiences. However, it is worth noting that such a link played a role only in individuals with histories of child maltreatment (Caspi et al., 2003; Eley et al., 2004; Kaufman et al., 2006; Kaufman et al., 2004; Kendler, 2005). In the absence of similar experiences, not only was the 5-HTTLPR (short allele) not associated with an increased risk of depression, but available positive social support was found to moderate the risk of depression associated with a history of maltreatment and the presence of the short allele of the serotonin transporter (Kaufman et al., 2004).

In other words, the tendency to develop depression and/or a personality profile more vulnerable to depression is not determined solely by genetic heritage but in large part by the sedimentation of more or less favorable life experiences in the individual emotional domain, during child development. As Kretschmer (1926) had already observed, actual experience appears to be codecisive for the development of a tendency toward depression.

Moreover, in line with the developmental systems theory, if the concept of inheritance applies to any resource that is reliably present in successive generations, then certain traits, more or less prone to the development of depression, may be inherited by the next generation if a given niche is again formed that favours the reproduction of this configuration (Oyama, Griffiths, & Gray, 2001).

Sadness: Between Normality and Psychopathology

As has been pointed out in the previous section, the contribution made by psychoanalysis and attachment theories to the problem of the genesis of a nonpathological character type, the depression-prone style of personality is to be found in the study of those conditions (loss, separation, rejection, etc.) that in the course of one's development engender recurrent ways of feeling. These experiences, which become sedimented over time, incline the individual's sense of personal stability toward a context of reference that is prevalently focused on states of sadness, anger, and anxiety. Hence, the individual's sense of permanence of self is prevalently centered on the hypercognition of these basic emotions that structure his own experience of being stable and with a sense of personal continuity according to the variability of circumstances and his relations to others. Together with Gallagher (2007), we call this mode of experiencing oneself, which is not at the level of narration but already at the prereflexive level (the experience in first person), "to be emotionally situated" (Arciero & Bondolfi, 2009). The endurance of this tendency of being emotionally situated, that is to say what allows an individual to keep a sense of permanence of oneself over time, is further reflected, through a narrative framing, in the ways in which the person shapes his personal identity.

At this point of our discussion, we postulate that the differences between a nonpathological condition of one's character (the depression-prone style of personality), a pathological character type (DPD), and affectively based chronic depression, would appear to hinge on the degree to which this mode of maintaining one's personal stability prevalently focused on states of sadness, anger, and anxiety, is absolute and rigid, and/or on the degree of intensity and mobility of the emotional states that characterize it. In order to explain and to account for the continuity as well as the differences between normality and psychopathology, the analysis of these emotions—and sadness in particular—thus represents the thread to be followed.

Before considering in more detail the experience of sadness, we would like to briefly specify the theoretical framework supporting our reflection on emotions. Inevitably, due to space limitation, the following explanation cannot be exhaustive and do justice to the complexity of this area.

From a cognitive perspective, an emotional state may be considered as constituting the result of the process of evaluating the stimulus situation by a cognitive appraisal. Rather than looking at emotions as internal meanings of an external stimulus situation, in light of the phenomenological tradition (Heidegger, 1962; Petitot, Varela, Pacoud, & Roy, 1999; Zahavi, 2012), we would like to consider experiencing emotion as a way of perceiving oneself in the world. That is to say, experiencing emotion corresponds to this incessant encounter between one's own possibilities and the way the world appears. In this understanding, experiencing an e-motion can be seen like an uninterrupted movement from, as the etymology of the word (ex-movere: *motion from*) suggests.

Coming back to the experience of sadness, let us start from a simple question: why do people become sad? Sadness may be elicited by a number of phenomena, ranging from disappointment to rejection, from separation (even temporary) to the loss of a loved one, or even-to quote Bowlby (1980)-"any trouble or misfortune." Sadness may thus be regarded as a normal and healthy way of feeling that which emerges when an individual faces an adverse and unalterable event. These two characteristics of the event correspond to two distinctive aspects in the experiencing of sadness: on the one hand, the adversity of a circumstance determines unpleasantness and sorrow, which in the case of intense sadness may even take the form of bodily pain. On the other hand, the fact that someone has no possibility to modify an event provokes the emergence of an experience of goal blockage and/or goal loss, which may manifest itself according to various degrees of inactivity. What is the relation between these two components of the subjective, prereflexive, first-person experiencing of sadness: (a) unpleasantness and

sorrow; (b) goal blockage and/or goal loss, in connection with the adversity and the inalterability of an event?

No doubt it is one thing for a person to feel sad because she/ he is forced to shortly leave her/his family on account of her/his job, for instance; it is quite another for her/him to be pervaded by sadness following the death of a dear friend or the end of an important relationship, and another still to feel sad every day because her/his parents have no interest in her/him and take no care of her/him. Yet these three different experiences have something in common. What makes them similar to one another is the impossibility of changing the present state of affairs; only in the first case, however, is sadness a transitory occurrence, as the event that elicited it is a temporary one: sadness here indicates a transient impossibility. In the other two cases sadness endures, and its permanence is connected to the immutability of the events that triggered it.

If e-motioning corresponds to the process of motioning from a given context by generating a renewed range of possible skillful engagements, then sustained sadness would seem to prove that this way of understanding emotions is wrong. If we consider e-moting as an attempt on the part of a person to find a new orientation when faced with changing contexts through the generation of new meaning possibilities (action tendencies), sustained sadness may be seen to signal the impossibility for an individual to apprehend the ongoing situation in any other way. In the experience of the enduring sadness there seems to be no clear action tendency—except inaction, or withdrawal into oneself. Therefore is sustained sadness not even an emotion as Lazarus believes (Lazarus, 1991, p. 251) or is inaction a way of being emotionally situated?

Sustained sadness may be elicited by various other kinds of loss besides bereavement, for instance: marital dissolution or conjugal infidelity, parental rejection, unexpected loss of social status, failure to attain important goals, unexpected job loss, and chronic conditions of rejection. In these circumstances, two characteristics play a fundamental role: the first is the fact that the condition lost was fundamental for the individual's experiencing of being in the world, of being situated with a sense of personal continuity; the second, that it is precisely the unalterable nature of the condition (of loss or rejection) in which one finds oneself that makes motioning from it impossible. Intricately connected to inaction is the tendency to focus attention inwardly. As an emotion, sadness calls forth a specific action tendency (Frijda, 1986). Hence the paradoxical nature of sadness: an emotion that binds us to the world while at the same time distancing us from it. Several theories and a variety of studies have been developed to account for this inward focus of sadness.

According to the attachment theory (Bowlby, 1980), when a child is exposed to recurrent situations of lack of care with significant others, such situations tend to elicit basic emotions such as sadness, anger, and anxiety. Over time, the repetition of these interactions induce the child to structure emotional patterns, as well as personality traits, focused prevalently on these basic emotions.

Parallel to this process, the child focuses his attention on the bodily states which have given rise to the emotional states. Precocious and recurrent activation of these basic emotions, in relation to such difficult and painful interactions, guides the child's perception of personal stability according to a frame of reference that employs a predominantly body-centered focus. This allows the child to regulate his relationship, both with others and in accordance with the variability of the situation through bringing internal states into focus, thus privileging a body-bounded sense of self. Recurrent emotional states are gradually integrated in the course of development in the form of complex character traits, perceptions, cognitions—connected to emotions, actions, and expressive communications—but also as habits, norms, and values (Dougherty, Abe, & Izard, 1996; Izard, Libero, Putnam, & Haynes, 1993; Magai & McFadden, 1995; Malatesta, 1990).

According to other researchers, the tendency to focus attention inwardly is related to a unique depressive attributional style for one's loss of control over outcomes (Abramson, Seligman, & Teasdale, 1978); others have suggested that it is linked to a depressive self-focusing style characterized by the fact that selffocus sets off a self-evaluative process in which one's current and desired states are compared (Pyszczynski & Greenberg, 1987; Pyszczynski, Hamilton, Herring, & Greenberg, 1989). Others still maintain that low intensity moods are a determinant of attentional focus (Cunningham, 1988; Sedikides, 1992).

None of these theories, however, has ever dwelled upon another fundamental aspect of sadness: the experience of suffering. How then does this experience of suffering take the form of bodily pain? It is precisely this experience that polarizes the individual's attention, anchoring the depression-prone style of personality to a body-centered reference system (Arciero & Bondolfi, 2009; Mazzola et al., 2014). Indeed, individuals presenting these psychological predispositions tend to be more viscerally aware, more sensitive in the detection of changes in bodily states occurring during emotions and feelings. We showed this in a recent fMRI study, where such an individual variability in emotion processing was demonstrated (Mazzola et al., 2010).

In fact the experience of pain consists of two components: pain sensation, whose neural substrate is found in the somatosensory cortex and the posterior insula, and pain affect, which is associated with the dorsal area of the anterior cingulate cortex (dACC). The sensory processing of pain provides information about ongoing tissue damage, whereas the feeling of unpleasantness signals the perceived aversive state and "motivates behaviour to terminate, reduce, or escape exposure to the source of the noxious stimulation" (MacDonald & Leary, 2005, p. 204). Not only do different languages employ the same expressions used for physical pain (hurt, injured, harmed, broken bones, etc.) to describe painful experiences such as rejection or the loss of a loved one (social pain), but patients with depression often present unexplained physical pain (Bair, Robinson, Katon, & Kroenke, 2003; Simon, VonKorff, Piccinelli, Fullerton, & Ormel, 1999; Trivedi, 2004; Tylee & Ghandi, 2005). The hypothesis that the brain areas recruited for physical pain may be similar to those recruited for social pain was tested in an fMRI study that investigated the neural substratum of social exclusion (Eisenberger, Lieberman, & Williams, 2003). Subjects scanned in a situation in which they were prevented from participating in a game by other players showed increased activity in the dACC (Areas 24 and 32). The degree of activation of the dACC strongly correlated with self-reports of social distress felt during the exclusion episode. This study provided evidence that the experiences of social and physical pain share a common neural substratum: the dACC.

Interestingly, frequencies of reported activation of brain areas during induced sadness across 22 relevant studies show that the ACC (Areas 24, 25, 32) is the second most prominent region recruited (after the basal ganglia; Freed & Mann, 2007). This brain region, along with others, is also activated in cases of grief (Gündel, O'Connor, Littrell, Fort, & Lane, 2003), separation from loved ones (Najib, Loberbaum, Kose, Bohning, & George, 2004), depression (Davidson, Pizzagalli, Nitschke, & Putnam, 2002), distress vocalizations emitted by young mammals when separated from caregivers (Lorberbaum et al., 1999; Lorberbaum et al., 2002), and the maternal response of rodents to the vocalizations of their pups (Eisenberger & Lieberman, 2004; Murphy, MacLean, & Hamilton, 1981). Hence, a range of evidence suggests that physical-pain distress and social-pain distress share overlapping substrates (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). From an evolutionary perspective the function of social pain would thus appear to be similar to that of physical pain. In particular, just as physical pain sensations focus the individual's attention on physical injury, motivating him to perform a series of actions aimed at mitigating his pain.

As we have argued, inaction and inward individual's attentional focus are two aspects that are closely and mutually integrated in the experiencing of sadness, particularly in the case of conditions such as bereavement or chronic rejection, which are characterized by sustained sadness. In these cases it is even more evident that the individual's inability to change-by generating actions-the suffering situation in which he finds himself forces him to disengage from the situation by producing a change of experiential focus: almost a need to take care of himself and his own pain. This may be an acute need, as in the case of bereavement or unwanted separation (when it extends in various ways over time), or a need related to structural conditions such as the lack of adequate parental care. In both cases-albeit in different ways-being emotionally situated is perceived by focusing on inner states connected to the unalterable situations. Only in the case of sustained rejection, however, does the person's way of feeling incline her/his sense of personal stability over time toward a reference context focused on inner states: for as the studies conducted by Harris et al. (1986) have shown, an early loss is not enough to predispose individuals toward melancholy.

The fundamental characteristic of this style of personality (and of DPD) is the individual's anchoring to a predominantly body-centered reference system that allows him to focus on inward signals in order to face the multiplicity of situations and his relation with others. This would explain why, for example, in order to prevent the activation of attachment behaviors which would probably not be met with comfort support, infants exhibit active prevention of contact with caregiver (Aisnworth, 1985; Bowlby, 1980). The individual, that is, maintains his own sense of permanence of self by avoiding situations and actions that may engender painful feelings, and by paying stable attention to his own inward states, the management of which monopolizes his cognitive resources. Because he/she has the feeling that reality is precarious, corresponding to a form of sensitivity ready to grasp the most evasive and defective aspects of the human condition. It focuses the subject's cognitive resources on the search for consistent and solid inner realities: for enduring characteristics which may allow him to face the perceived evanescence and fatuousness, or absurdity and pointlessness, of existence. And according to the more or less skillful use of his/her cognitive resources, the individual will be able to cope in a more or less adaptive way, as we will explain shortly in the next section.

Since Abramson et al. (1978), numerous studies have emphasized the aforementioned process, while stressing only its cognitive aspects (treated apart from the emotional states that determine them): aspects that have been explained as a dispositional tendency to make internal attributions. The strong reflexive engagement of the individual may actually be said to be completely subordinated to emotional activation, as the cognitive domain is here made to do what sadness cannot do, namely: engender a renewed range of possible skillful engagements in order to apprehend the ongoing situation in any other way. Sadness, by contrast, binds the person to the situation, deactivation of the motor system being but one clear sign of this.

It is worth noting that for this style of personality, the strong inward anchoring is not only body-centered but concerns also their cognitive engagement (supported by the experience of suffering). The cognitive resources constitute the only means to try to change the aversive emotional states through which he/she feels situated by generating new meaning possibilities. Sadness therefore plays a double and complementary role.

It is precisely this crucial point that allows us to distinguish the "normal" depression-prone style of personality from DPD: the possibility and capacity on the part of the individual of cognitively disengaging from the situation that triggers sadness. It is by grasping these affective states, not as destructive emotions but as a source of meaning, that the individual can generate new possibilities of engagement in her/his relation to the world and others.

Modalities through which cognition may represent an adaptive way or, conversely, the origin of a pathological condition are discussed in the following section.

Personal Stories and Clinical Judgment: A Quest for a New Methodology

According to the points argued in the previous section, the most evident difference between a depression-prone style of personality and DPD is found in the role played by cognition. In the first case, cognition serves as an instrument which allows the individual to disengage from situations that trigger destructive emotions; in the second case, it represents a way of stabilizing and—in pathological forms—amplifying a negative perception of one's being in the world (ruminations).

As we have already emphasized, these different roles played by cognition are not primary ones; rather, they depend on the emotional structure of cognition. That is, the inability to disengage is connected to the pervasiveness (across situations) and recurrence (across time) of the painful feelings elicited in delicate moments in one's life, and hence to the difficulty of articulating these feelings. The particular emotive structure that underlies the construct we have termed DPD—and the cognitive configuration related to it—must thus be traced back to a repeated exposure to negative events (i.e., chronic conditions of emotional, but also physical and sexual, maltreatment; Harkness & Lumley, 2008).

The kind of stability deriving from the previous structure is centered not only on negative emotions, but also on a way of bearing these emotions which is characterized by the development of personal themes of derogation, unacceptableness, unworthiness, defectiveness, unamiability, and so on. In this way sadness that increases the individual's attentional focus more inwardly drives the cognitive abilities.

The factors of vulnerability to depression, therefore, must be sought not only in the characteristics of cognition (Abela & Hankin, 2008; Abramson, Metalsky, & Alloy, 1989; Abramson et al., 1978; Beck, 1967; Bondolfi, 2004).

For example, for a depression-prone style of personality, the nonpathological condition of one's character, a given event may cause sadness and this sadness—by taking hold of cognition comes to be articulated in terms of one's own defectiveness. This is due to the characteristics of sadness itself, which—as we have seen—forces this individual to take care of himself in order to disengage from the situation that caused it. By doing this, he/she thus can achieve a certain degree of internal stability under distressing circumstances that he/she is facing.

On the other hand, for those who are vulnerable to depression (DPD), this concern with oneself—with one's perceived unworthiness, for instance—rather than enabling a disengagement from the event through the obtention of an internal stability, as in the previous example, amplifies the sadness from which it originates thus initiating a downward spiral that may lead to depression. From this perspective, vulnerability to the disorder would thus appear to be linked to a personality structure that, by its very way of maintaining stability, predisposes or not the individual to the development of depression following the occurrence of negative events. This more vulnerable personality may be identified with the depressive temperament, which, as Kretschmer has pointedly observed, "is more easily roused by sad conditions" (Kretschmer, 1926, pp. 129–130).

On the other hand, regardless of the structure of the personality, for each human being a range of conditions that lead to the loss of the personal stability can trigger a depressive reaction.

How then can the clinician decide on whether the sadness response of the individual is proportional to the triggering situation or not? Individual experience takes a meaning by dint of the fact that it is found within a configuring framework, the personal story. The ongoing experience is placed within a referential context by the story that it helps to articulate and develop, both in the case where the event falls within the range of expectations that derive "naturally" from the story, and in the case where it takes "by surprise" the predictions made regarding the development of the story, thereby generating new expectations. In this respect, each individual story may lead to a state of depression in conjunction with life circumstances capable of initiating a downward spiral: a vicious circle of cognition and sadness. Therefore it is evident that operational criteria cannot have priority over clinical judgment, but it is equally clear that to decide that the response to a loss is "normal" on the basis of what the clinician knows of the individual and her/his cultural background requires a method. A method allowing the clinician to investigate the uniqueness of the personal story of her/his patients. This may be the most important challenge issued from DSM-5 new emphasis on clinical judgment, and it can be particularly relevant in the case of depression.

Conclusion

Misclassification of normality as psychiatric disorder remains a challenging task. Going beyond comments on the diagnostic and construct validity of DSM-5, the note quoted before refers to the inevitable exercise of clinical judgment. Consequently, it makes one focus more on the individual in question, which implies that understanding the context of symptoms is crucial in order to evaluate disorder. Therefore, one of our aims was to suggest food for thought in rethinking deep sadness, bereavement, and depression. Therefore, we introduced a perspective which takes into account the experience of feeling "sad." Indeed, in the case of deep sadness, bereavement, and depression one cannot tell whether there is a disorder or normal functioning without reference to the individual's social affective ongoing context and personal story.

Declaration of Conflicting Interests

None declared.

References

- Abela, J. R. Z., & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents: A developmental psychopathology perspective. In J. R. Z. Abela & L. Hankin Benjamin (Eds.), *Handbook of depression in children and adolescents* (pp. 35–78). New York, NY: Guilford.
- Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, 96, 358–372.
- Abramson, L. Y., Seligman, M. E., & Teasdale, J. D. (1978). Learned helplessness in humans: critique and reformulation. *Journal of Abnormal Psychology*, 87, 49–74.
- Ainsworth, M. D. S. (1985). Patterns of infant-mother attachment: Antecedents and effects on development. *Bulletin of New York Academy of Medicine*, 61, 771–791.
- Akiskal, H. S. (2001). Dysthymia and cyclothymia in psychiatric practice a century after Kraepelin. *Journal of Affective Disorders*, 62, 17–31.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.
- Arciero, G., & Bondolfi, G. (2009). Selfhood, identity and personality. London, UK: Wiley-Blackwell.
- Arciero, G., & Guidano, V. F. (2000). Experience, explanation and the quest for coherence. In R. A. Neymeyer & J. D. Raskin (Eds.), *Constructions* of disorder (pp. 91–118). Washington, DC: American Psychological Association.
- Bair, M. J., Robinson, R. L., Katon, W., & Kroenke, K. (2003). Depression and pain comorbidity: A literature review. *Archives of Internal Medicine*, 163, 2433–2445.

- Beck, A. T. (1967). Depression: Causes and treatment. Philadelphia, PA: University of Pennsylvania Press.
- Bondolfi, G. (2004). Traitement intégré de la dépression: De la résistance à la prévention de la rechute. Darmstadt, Germany: Steinkopff-Verlag.
- Bowlby, J. (1980). Attachment and loss Vol. 3: Loss: Sadness and depression. New York, NY: Basic, Books.
- Brown, W., & Harris, T. (1978). Social origins of depression. A study of psychiatric disorder in women. London, UK: Tavistock.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., ... Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301, 386–389.
- Cunningham, M. R. (1988). What do you do when you're happy or blue? Mood, expectancies, and behavioral interest. *Motivation and Emotion*, *12*, 309–331.
- Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. (2002). Depression: Perspectives from affective neuroscience. *Annual Review* of Psychology, 53, 545–574.
- Dougherty, M. L., Abe, A. J., & Izard, E. C. (1996). Differential emotions theory and emotional development in adulthood and later life. In C. Magai & S. H. McFadden (Eds.), *Handbook of emotion, adult development, and aging* (pp. 27–38). San Diego, CA: Academic Press.
- Eisenberger, N. I., Jarcho, J. M., Lieberman, M. D., & Naliboff, B. D. (2006). An experimental study of shared sensitivity to physical pain and social rejection. *Pain*, 126, 132–138.
- Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain. *Trends in Cognitive Sciences*, 8, 294–300.
- Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, 302, 290–292.
- Eley, T. C., Sugden, K., Corsico, A., Gregory, A. M., Sham, P., McGuffin, P. ... Craig, I. W. (2004). Gene–environment interaction analysis of serotonin system markers with adolescent depression. *Molecular Psychiatry*, 9, 908–915.
- Freed, P. J., & Mann, J. J. (2007). Sadness and loss: Toward a neurobiopsychosocial model. *The American Journal of Psychiatry*, 164, 28–34.
- Freud, S. (1917). Mourning and melancholia. Complete psychological works (Vol. 14). London, UK: Hogarth Press.
- Frijda, N. H. (1986). *The emotions*. New York, NY: Cambridge University Press.
- Gallagher, S. (2007). Phenomenological and experimental research on embodied experience. In T. Ziemke, J. Zlatev, R. Frank, & R. Dirven (Eds.), *Body, language and mind* (Vol. 1, pp. 241–263). Berlin, Germany: Mouton de Gruyter.
- Guidano, V. F. (1987). Complexity of the self. New York, NY: Guilford.
- Guidano, V. F. (1991). The self in process: Toward a post-rationalist cognitive therapy. New York, NY: Guilford.
- Gündel, H., O'Connor, M. F., Littrell, L., Fort, C., & Lane, R. D. (2003). Functional neuroanatomy of grief: An fMRI study. *The American Journal of Psychiatry*, 160, 1946–1953.
- Harkness, K. L., & Lumley, M. N. (2008). Child abuse and neglect and the development of depression in children and adolescents. In R. Z. J. Abela & L. Hankin Benjamin (Eds.), *Handbook of depression in children and adolescents* (pp. 466–488). New York, NY: Guilford.
- Harris, T., Brown, W., & Bifulco, A. (1986). Loss of parent in childhood and adult psychiatric disorder: The role of lack of adequate parental care. *Psychological Medicine*, 16, 641–659.
- Heidegger, M. (1962). Being and time. New York, NY: Blackwell.
- Izard, C. E., Libero, D. Z., Putnam, P., & Haynes, O. M. (1993). Stability of emotion experiences and their relation to traits of personality. *Journal* of Personality and Social Psychology, 64, 847–860.
- Kaufman, J., Yang, B. Z., Douglas-Palumberi, H., Grasso, D., Lipschitz, D., Houshyar, S., ... Gelernter J. (2006). Brain-derived neurotrophic factor–5-HTTLPR gene interactions and environmental modifiers of depression in children. *Biological Psychiatry*, 59, 673–680.
- Kaufman, J., Yang, B. Z., Douglas-Palumberi, H., Houshyar, S., Lipschitz, D., Krystal, J. H., & Gelernter, J. (2004). Social supports and serotonin transporter gene moderate depression in maltreated children.

Proceedings of the National Academy of Sciences of the United States of America, 101, 17316–17321.

- Kendler, K. S. (2005). "A gene for. . .": The nature of gene action in psychiatric disorders. *The American Journal of Psychiatry*, 162, 1243–1252.
- Kraepelin, E. (1921). Manic depressive insanity and paranoia. Edinburgh, UK: Livingstone.
- Kretschmer, E. (1926). *Hysteria*. New York, NY: Nervous and Mental Disease.
- Lazarus, R. S. (1991). Emotion and adaptation. Oxford, UK: Oxford University Press.
- Lorberbaum, J. P., Newman, J. D., Dubno, J. R., Horwitz, A. R., Nahas, Z., Teneback, C. C., ... George, M. S. (1999). Feasibility of using fMRI to study mothers responding to infant cries. *Depression and Anxiety*, 10, 99–104.
- Lorberbaum, J. P., Newman, J. D., Horwitz, A. R., Dubno, J. R., Lydiard, R. B., Hamner, M. B., ... Georges, M. S. (2002). A potential role for thalamocingulate circuitry in human maternal behavior. *Biological Psychology*, *51*, 431–445.
- MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, 131, 202–223.
- Magai, C., & McFadden, S. H. (1995). The role of the emotion in social and personality research: History, theory and research. New York, NY: Plenum Press.
- Maj, M. (2013). "Clinical judgment" and the DSM-5 diagnosis of major depression. World Psychiatry, 12, 89–91.
- Malatesta, C. Z. (1990). The role of emotions in the development and the organization of personality. *Nebraska Symposium on Motivation*, 36, 1–56.
- Mazzola, V., Arciero, G., Latorre, V., Petito, A., Gentili, N., Fazio, L., ... Bondolfi, G. (2010). Affective response to loved one's pain: Insula activity as a function of individual differences. *PLoS ONE*, 5(12), e15268. doi:10.1371/journal.pone.0015268
- Mazzola, V., Marano, G., Biganzoli, E. M., Boracchi, P., Lanciano, T., Arciero, G., & Bondolfi, G. (2014). The In-Out Dispositional Affective Style Questionnaire (IN-OUT DASQ): An exploratory factorial analysis. *Frontiers in Psychology*, 5, 1–12.
- Murphy, M. R., MacLean, P. D., & Hamilton, S. C. (1981). Species-typical behavior of hamsters deprived from birth of the neocortex. *Science*, 213, 459–461.
- Najib, A., Lorberbaum, J. P., Kose, S., Bohning, D. E., & George, M. S. (2004). Regional brain activity in women grieving a romantic relationship. *The American Journal of Psychiatry*, 161, 2245–2256.
- Oyama, S., Griffiths, P. E., & Gray, R. D. (2001). Cycles of contingency: Developmental systems and evolution. Cambridge, MA: MIT Press.
- Petitot, J., Varela, F., Pacoud, B., & Roy, J. (1999). Naturalizing phenomenology. Stanford, CA: University Press.
- Pyszczynski, T., & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self- focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin*, 102, 122–138.
- Pyszczynski, T., Hamilton, J. C., Herring, F. H., & Greenberg, J. (1989). Depression, self-focused attention, and the negative memory bias. *Journal of Personality and Social Psychology*, 57, 351–357.
- Sedikides, C. (1992). Mood as a determinant of attentional focus. Cognition & Emotion, 6, 129–148.
- Simon, C. E., VonKorff, M., Piccinelli, M., Fullerton, C., & Ormel, J. (1999). An international study of the relation between somatic symptoms and depression. *The New England Journal of Medicine*, 341, 1329–1335.
- Trivedi, M. H. (2004). The link between depression and physical symptoms. *The Primary Care Companion to the Journal of Clinical Psychiatry*, 6(Suppl. 1), 12–16.
- Tylee, A. P., & Ghandi, P. (2005). The importance of somatic symptoms in depression in primary care. *The Primary Care Companion to the Journal of Clinical Psychiatry*, 7, 167–176.
- Zahavi, D. (2012). *The Oxford handbook of contemporary phenomenology*. Oxford, UK: Oxford University Press.