Sadness vs depression: everyday feelings vs mood disorders and the adaptative value of sadness

Tristeza versus depresión: sentimientos cotidianos versus trastornos del humor y el valor adaptativo de la tristeza

María Inés López-Ibor Alcocer

Académica Correspondiente de la Real Academia de Doctores de España. mariaines@lopezibor.com

An. Real. Acad. Doct. Vol 1, (2016) pp. 103-112.

RESUMEN	ABSTRACT
Este artículo pretende revisar específicamente las diferencias entre tristeza y depresión. No siempre las definiciones y descripciones de los trastornos depresivos distinguen entre trastornos depresivos de estados de ánimo no patológico. Si la sintomatología depresiva aparece como una respuesta a un evento de la vida como el duelo, un diagnóstico de depresión puede no ser claro ya que estas reacciones representan reacciones normales y probablemente adaptación a esas circunstancias de la vida. El reto a enfrentar es distinguir reacciones emocionales normales a eventos cotidianos de un conjunto de enfermedades psiquiátricas caracterizadas por la presencia de tristeza. Algunos estudios recientes de investigación señalan algunos de los correlatos neurobiológicos entre tristeza y depresión. Este trabajo repasa una nueva evidencia de la necesidad de distinguir la tristeza de la depresión clínica, con una visión diferente. La cuestión es no sólo la diversa calidad del episodio de depresión o del depresivo y los síntomas asociados a la misma; sino que también el presente estudio considera el valor adaptativo de la tristeza y sentimientos	This article aims to specifically review the differences between sadness and depression. Definitions and descriptions of depressive disorders do not always distinguish between depressive disorders from non-pathological mood states. If depressive symptomatology appears as a response to a life event such as bereavement, a diagnosis of depression may not be clear since these reactions represent normal and presumably adaptive reactions to such life circumstances. The challenge to face is to distinguish normal emotional reactions to everyday events from a set of psychiatric illnesses characterised by the presence of sadness. Some recent research studies point out some of the neurobiological correlates between sorrow and depression. This paper reviews new evidence for the need to distinguish sadness from clinical depression, supporting a different view. The issue is not only the different quality of depression or depressive episode and the symptoms

relacionados que afecta a casos clínicos específicos. La tristeza tiene algunos aspectos positivos y por lo tanto un valor adaptativo; aumenta la capacidad para resolver problemas relacionados con las pérdidas y daños, la tristeza también nos ayuda a comunicar nuestra vulnerabilidad y a veces necesita, evitar las agresiones en los conflictos jerárquicos y modula las intervenciones que llevan a recuperar vínculos perdidos. La respuesta a las preguntas implicadas en este problema tendrá un impacto en la manera de que la Psiquiatría será concebida, practicada y financiada en un futuro cercano.	associated to it, the present study also considered the adaptive value of sadness and related affects in general in specific clinical cases. Sadness has some positive functions and therefore an adaptive value; it increases the ability to solve challenges related to losses and harms, sadness also helps us to communicate our vulnerability and needs, sometimes avoid aggressions in hierarchical conflicts and modulates interventions that lead to recover from lost bonds. The answer to the questions involved in this problem will have an impact on the way psychiatry will be conceived, practiced and financed in the near future.
Palabras clave: trastorno depresivo mayor, diagnóstico, tristeza, emociones, adaptación.	**Keywords:** major depressive disorder, diagnosis, sadness, emotions, adaptation.

1. INTRODUCTION

There is still a debate on the nature of the symptoms of mental disorders, a confusion about their classification and diagnosis and a preoccupation with the growing inflation of diagnostic categories. And this is probably due because psychiatry does not have a clear definition of mental disorder that covers all situations, and it is difficult to establish a precise distinction between normality and psychopathology (López-Ibor et al, 2013).

Current models for classifying clinical depression are dimensional and therefore out-dated and descriptions of depressive disorders do not always distinguish depressive disorders from non-pathological mood states. If depressive symptomatology appears as a response to a life event such as bereavement, a diagnosis of depression may not be clear since these reactions represent normal and presumably adaptive reactions to such life circumstances.

The challenge to face is the delimitation of normal emotional reactions to everyday events and a set of psychiatric illnesses characterised by the presence of sadness accompanied by other phenomena, which, incidentally, may also be unchained by negative experiences.

DSM 5 (American Psychiatric Association, 2013), and eventually the International Classification of Disorders will follow the same path, putting forward old and important problems of psychiatric classification. DSM 5 proposes that the word "criteria" is substituted by the word "symptoms" to avoid confusion between number of symptoms and number of criteria necessary for diagnosis. This seems reasonable because it underlines the fact that DSM, and by correspondence ICD-10 (International Classification of Mental Disorders, 1992), are not treaties of psychiatry but just instruments to classify diseases and to elaborate statistical documents. Nevertheless, a clear definition of what symptoms are is lacking in both systems.

As Maj (Maj, 2011) pointed out recently it is not always easy to establish when a depression becomes a mental disorder and he proposed three approaches, the first one is the need to take into account the context in which depression occurs, the second is that there is a qualitative difference between sadness and depression, in depression anhedonia is one of the main diagnostic criteria but it does not appear in sadness and the third one is a pragmatic approach that considers a continuum for sadness to clinical depression .

More recently Wakefield et al (Wakefield et al., 2013) have proposed to take into consideration the recurrence rates of major depression in order to evaluate the validity of diagnosis; uncomplicated symptoms, transient duration, and lack of elevated recurrence suggested that generally it may represent just a non-pathological sadness so to say, not a depressive disorder.

2. SADNESS AND DEPRESSION, FEELING VERSUS DISORDER

The first systematic approach to differentiate (normal, everyday) sadness from depression took place hundred years ago, in continental Europe when it was considered that sadness present in depression had a distinct quality of normal sadness, which can be identified in clinical settings and become the basis for the diagnosis of (morbid) depression. This different quality has been considered as lack of subjective response (blunting, affect anesthesia), as an alteration of vitality (vital sadness) or inhibition (behavioral) associated to the presence of somatic symptoms and to a lack of motivation and anhedonia.

Nevertheless it is worth mentioning that this distinct quality is perceived by observers, in spite of the fact, that patients may have considerable difficulties in verbalizing differences from normal sadness, leading to interfere in the diagnostic process due to semantic factors (Ramos-Brieva & Cordero Villafáfila, 1991).

The concept of vital sadness as a disturbance of the vital feelings was introduced by Kurt Schneider (Schneider, 1959) following Max Scheler's description of four types of feelings:

1. Sensory feelings (*Sinnliche Gefühle*), or feeling-sensations (*Empfindungsgefühle*) concerning specific parts of the body, such as pain, a knot in the stomach due to hunger or cold on the back from fear.
2. Vital feelings (*Lebensgefühle*), or body feelings (*Leibgefühle*) concerning the body experience as a whole such as distress or wellbeing.
3. Psychic feelings (*seelische Gefühle*) or pure feelings of the Ego (*reine Ich Gefühle*), corresponding to the environment and external world. They are reactive to external circumstances such a joyfulness, enjoyment, sadness or despair.
4. Spiritual feelings (*geistige Gefühle*) or feelings of the personality (*Persönlichkeitsgefühle*), which are spontaneous, absolute beyond specific values, such as ecstasy or agony.

In summary, vital sadness is experienced as independent from external circumstances and as a negative subjective experience of the own body, which is felt as weighty, slow, painful, unpleasant or painful while normal (reactive) sadness is directly linked to negative circumstances.

In a similar way López Ibor Sr. (López Ibor, 1950) came to the conclusion that the anxiety which characterises neurotic disorders was a vital and not a psychic or reactive feeling, introducing the concept of “vital anxiety” thus paving the way for the biological treatment of this group of disorders.

The concept of «vital depression» as a disturbance of the vital feelings (K. Schneider, 1959) has recently been considered again. The psychological suppositions of vital feelings are outlined with reference to Scheler's subdivision of emotional life into layers.

3. SADNESS IS AN EMOTION, AND IT HAS AN ADAPTIVE ROLE

Sadness is a universal emotion, the emotion most closely related to depression. It has facial and corporal expressions associated to it that are similar and therefore recognizable in many different cultures (Izard, 1991). Sadness is normally a response to some kind of loss, health, family, money, job but it is also linked to a loss of attachment to a child or to a partner, relative or close friend (Tsuchiya & Adolphs, 2007).

From an evolutionary perspective attachment is adaptive, it begins with the relation to the bond between mother and baby, so important that the loss of this attachment, even briefly, can cause sadness in babies and they actively search for the parent. In the same way attachment is also important for adults and its loss promotes sadness and the search for another partner. Sadness can also result from other losses, for example money, job, or lack of success at work.

From an evolutionary perspective sadness has a clear function which is to motivate the individual to recover from these losses and also to drive us to restore attachment bonds. *The sadness caused by bereavement is the cost of having been attached, and it may also act as a social signal that is a plea for sympathy* as Bowlby (Bowlby, 1981) points out.

Solomon (Solomon, 2003, 2004, 2006a, 2006b, 2000) emphasizes the personal and ethical character of our emotions. Emotions are not something that happen to us, nor are they an irrational part of individuals but emotions are judgements and strategies that we build about the world and help us to live in it. Not only sadness but there are other feelings as fear, anger, love, guilt, jealousy, compassion that are essential to our values, to live happy, healthy, and well. Solomon points out that if we are able to recognize these feelings, we can make our emotional live more coherent with our values and be more "true to our feelings" and cultivate emotional integrity.

First of all we have to understand what normal emotions are for and then we can consider whether an emotion is abnormal or not. Emotions evolve because they adjust the body to deal with situations that have occurred again and again over millions of years. Therefore emotions are useful if they appear in the situation they evolved for, otherwise we have to consider them as abnormal. None of the emotions are good nor bad and even negative emotions such as anxiety and sadness, are as useful as positive emotions are (Nesse, 1990, 1999).

An emotion tends to have a clear focus (i.e., its cause is self-evident), while mood tends to be more unfocused and diffused. Mood is an affective state that involves feelings about our general expectations of a future experience of pleasure or pain, or of positive or negative affect in the future (Batson, Shaw & Oleson, 1992).

Study of animal behaviour from an evolutionary perspective is currently being applied to human emotions, it is considered that sadness has some adaptive functions; first of all it help us to develop behaviours to recover from a loss of

bonds, second it increases the ability to confront challenges, avoid dangers, losses, harms, and third it is the way of communicating a need for help.

4. DEPRESSION AS A MOOD DISORDER

Depression diagnosis is based on the presence of several of the symptoms that have to last at least over two weeks: depressed mood most of the day; gain or loss of weight; too little or too much sleep; fatigue; thoughts of death or suicide; inability to concentrate; and feelings of guilt or worthlessness. But diagnosis based on these criteria may not distinguish low mood or sadness from a genuine clinical condition. This has been emphasised by Horwitz and Wakefield (2007).

Recently, Angst et al. (2014) have reviewed the validity and clinical relevance of the length of depressive syndromes, defined by the presence of 5 or more of 9 diagnostic symptoms (DSM-IV). The present study found that depressive syndromes lasting under 2 weeks to be highly prevalent, and those lasting 4+ days to have equal validity (family history, age of onset, course) and treatment rates to episodes of 2-4 weeks, suggesting that the criteria of length of duration of symptoms should be reviewed.

Depression does vary along a continuum from low mood or sadness to clinical depression. Feeling low or feeling sad is not different from what could be called a low level of depression. By contrast, severe or clinical depression is very hard to describe; everything is perceived as being negative, patients believe they will never recover, and sometimes physical symptoms and suicidal thoughts appear.

Depression is a disorder of emotion regulation and is sustained by negative affect. The prevalence of negative affect seems to be disproportionate; 15% of the US population has had an episode of severe depression. Many other have *bad days* when they are worried, sad or angry and are not able to function. Most attempts to understand this state is often based on the assumption that there is something wrong with these suffering people (Gotlib & Joormann, 2010).

But a diagnosis of depression requires the presence of either sustained negative affect or loss of pleasure. Depression not only changes the way we feel, it also changes how we perceive ourselves and the world around us. Negative views of the self, the world, and the future, as well as recurrent and uncontrollable negative thoughts that often revolve around the self, are debilitating symptoms of depression. Biases in cognitive processes such as attention and memory may not only be correlates of depressive episodes; they may also play a critical role in increasing individuals' vulnerability for the first onset and recurrence of depression. Depression is characterized by increased elaboration of negative information, by difficulties disengaging from negative material, and by deficits in cognitive control when processing negative information.

Cognitive theories of depression posit that people's thoughts, inferences, attitudes, and interpretations, and the way in which they attend to and recall events, can increase their risk for the development and recurrence of depressive

episodes. Most cognitive theories propose vulnerability-stress hypotheses that posit that the onset of this disorder is due to the interaction of a psychological vulnerability and a precipitating stressor (Doesschate et al., 2010)

According to evolutionary-biological principles some people are more vulnerable than others to the negative consequences of some stressful life events or adversity (Conradi, Ormel & deJonge, 2010; Belsky & Pluess, 2009).

The authors of the present study believe that he "Coping with Depression" course (CWD) is one of the best research projects that takes into account psychoeducational interventions for the treatment and prevention of depression, and it is widely spread and used in clinical routine practice in several countries. A meta-analysis of the up to then published 25 randomized controlled trials gave the following results: the 6 studies aimed at the prevention of new cases of major depression resulted in a reduced risk of suffering from major depression in 38% (incidence rate ratio was 0.62); the 18 studies examining the CWD as a treatment of depression found a mean effect size (Cohen's d) of 0.28. The CWD is a structured cognitive-behavioral intervention that can be easily adapted to different populations and has contributed considerably to improve prevention and treatment and recovery from depression (Cuijpers et al., 2009).

5. MAKING SENSE OF SADNESS

Although there still is considerable controversy regarding where the frontiers of normal sadness and clinical depression are, not enough research studies that have tried to integrate insights from the spiritual, philosophical, and neurobiological literature have been carried out. Pies (Pies, 2008) has proposed that such a synthesis is possible as recent brain imaging studies are also beginning to reveal the neurobiological correlates of sorrow and depression that will help to understand correlations in specific alterations in "neurocircuitry" with their phenomenological expressions . The high recurrence rate in depression suggests that there are specific factors that increase people’s risk for developing repeated episodes of this disorder such as cognitive functioning and cognitive biases in the processing of emotional information.

Most affective disorders are pathological states (and not adaptive ones), due to dysfunction of an innate precipitating mechanism (IPM) that regulates energy and activity levels according to intensity and duration of light (namely IPM-A). This IPM-A is responsible for vegetative, endocrine and behavioural responses that are present in humans and more ancient phylogenetic animals. More recently in the phylogeny, other mechanisms (IPM-AA) have coupled to this IPM-A. In the human being, the precipitating factors of IPM-AA are predominately social. IPM-AA adds new responses (such as mood) to the older responses of IPM-A (McLoughlin, 2002).

6. CONCLUSIONS

The main argument of this study is not that depressed mood and other manifestations of mood (affective) disorders have an adaptive value, as considered by some authors (Montañés & de Lucas Taracena, 2006), on the contrary, they are the expression of an adaptive reaction (everyday life sadness), and this perspective can help to differentiate between "normal" and "morbid" mood states. From an evolutionary perspective of psychopathology, emotions are the response system that allows us to adapt to the environment and that increases the ability to cope with threats and opportunities. Sadness and low levels of depression are adaptive since they lead the individual to try and help to recover from any kind of losses. The problem arises when a severe depression arises, it is not anymore adaptive, but can be thought of as sadness having become malignant (Wolpert, 2008).

The ability to experience normal low mood may be useful in certain situations. If some situations are recurrent in the course of evolution, it could be speculated that natural selection may have shaped subtypes of low mood that are parallel to the subtypes of anxiety that protect against different kinds of danger (Keller & Nesse, 2006).

It has to be taken into account that most classification systems are not a treaty of psychopathology, able to define what illnesses are. Main diagnostic criteria used to define what a clinical depression is are depressive mood and sadness, therefore the challenge is to define not only what mood is but also to define what depressive mood is, as well as to define what sadness is and to establish a relationship between depressive mood and sadness and of both of them with the rest of diagnostic criteria. This is even more important for the less severe cases in which the difference between normal sadness and depression may not be clear. Research difficulties come from the fact that feelings are everything what in psychic life is not prone to be objectified.

Therefore the present study considers that the question of normal and pathological sadness has to be answered at two levels: 1) what concerns a particular person visiting a psychiatrist and 2) what are the public health consequences of setting limits (López-Ibor, 2010).

BIBLIOGRAPHY

1. Angst, J., Hengarther, M.P., Addajic-Gross, V., Roessler, W. (2014). Is Two Weeks Optimum Duration Criteria for Major Depression. *Actas Esp Psiquiat*. Jan –Feb 42 (1), 18-27.
2. Ayton, A. (2000). Implications of Evolutionary Theory for Psychiatry. *British Journal Psychiatry:* 177: 370.
3. Batson, C.D., Shaw, L. L. & Oleson, K.C. (1992). Differentiating Affect, Mood and Emotion: Toward Functionally-Based Conceptual Distinctions. *Emotion*. Newbury Park, CA: Sage.
4. Belsky, J. & Pluess, M. (2009). Beyond Diathesis Stress: Differential Susceptibility to Environmental Influences. *Psychol Bull*. Nov;135 (6):885-908.

5. Bowlby, J. (1981). *Attachment and Loss, Loss: Sadness and Depression.* Vol. 3. London: Hogarth Press; Harmondsworth, Penguin.
6. Conradi, H.J., Ormel, J., deJonge, P. (2010). Presence of Individual (Residual Symptoms during Depressive Episodes and Periods of Remission: A 3-year Prospective Study. *Psychol Med.* Oct 8: 1-1.
7. Cuijpers, P., Muñoz, R.F., Clarke, G.N., Lewinsohn, P.M. (2009). Psychoeducational Treatment and Prevention of Depression: The "Coping with Depression" Course Thirty Years Later. *Clin Psychol Rev*. Jul: 29 (5):449-58.
8. Doesschate, M.C, Bocting, C.L, Koeter, M.W., Schene, A.H. (2010): DELTA Study Group. Prediction of Recurrence in Recurrent Depression: A 5.5-year Prospective Study. *J Clin Psychiatry*, Aug 71 (8): 984-91.
9. DSM-5. Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (2013). *American Psychiatric Association.*
10. Gotlib Ian, H. & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annu Rev Clin Psychol*. April 27; 6: 285–31.
11. Horwitz, A.V. & Wakefield, J.C. (2007). *The Loss of Sadness: How Psychiatry Transformed Normal Sorrow into Depressive Disorder.* Oxford: Oxford University Press.
12. ICD 10. *International Classification of Mental Disorders* (1992). WHO, Geneva, Switzerland.
13. Izard, C.E. (1991). *The Psychology of Emotions.* London: Plenum.
14. Keller, M.C. & Nesse, R.M. (2006). The Evolutionary Significance of Depressive Symptoms: Different Adverse Situations lead to Different Depressive Symptom Patterns. *J Pers Soc Psychol*. Aug; 91(2):316-30.
15. Lopez Ibor, J.J. (1965). Basic Anxiety as the Core of Neurosis. *Acta Psychiatr Scand,* 41(3):329-32.
16. López-Ibor, J.J. (2010). The Limits of Depression: Normal Sadness or Mental Disorder. *Actas Esp Psychiatr.* 38 (Suppl 3, 3-4).
17. López-Ibor, J.J. & López-Ibor, M.I. (2013). Paving the Way for New Research Strategies in Mental Disorders. First Part: The Recurring Crisis of Psychiatry. *Actas Esp Psi*. Jan–Feb: 41(11), 33-43.
18. López-Ibor, J.J. & López-Ibor, M.I. (2013). Paving the Way for New Research Strategies in Mental Disorders. Second Part: The light at the End of the Tunnel. *Actas Esp Psi*. Mar-Apr 41(2) 67-75.
19. Maj, M. (2011). When does a Depression become a Mental Disorder? *British J Psychiatry.* 119, 85-86.
20. McLoughlin, G. (2002). Is Depression Normal in Human Beings? A Critique of the Evolutionary Perspective. *Int J Ment Health Nurs*. Sep;11(3):170-3.
21. Montañés, F. & de Lucas Taracena, M.T. (2006). Evolutionary Aspects of Affective Disorders, Critical Review and Proposal of a New Model. *Actas Esp Psiquiatr*. Jul-Aug: 34(4):264-76.
22. Nesse, R.M. (1990). Evolutionary Explanations of Emotions. *Human Nature:* 1:261–289

23. Nesse, R.M. (1999). Proximate and Evolutionary Studies of Anxiety, Stress and Depression: Synergy at the Interface. *Neurosci Bio Behav Rev*.: 23:895–903.
24. Pies, R. (2008). The Anatomy of Sorrow: A Spiritual, Phenomenological and Neurological Perspective. *Philos Ethics Humanit Med*. Jun (17)3:1.
25. Ramos Brieva, J.A & Cordero Villafáfila, A. (1991). The Distinctive Quality of Depressed Mood. VIII. A Review of the Concept and a Proposal. *Actas Luso Esp Neurol Psiquiatr Cienc Afines*. Jan-Feb: 19(1):31-46.
26. Schneider, K. (1959). *Clinical Psychopathology*. New York: Grune and Stratton.
27. Solomon, R.C. (2003). What Is An Emotion?. *Classic and Contemporary Readings*. Oxford.
28. Solomon, R.C. (2004). *Thinking about Feelings: Contemporary Philosophers on Emotions (*Oxford, 2004).
29. Solomon, R.C. (2006a). *Dark Feelings, Grim Thoughts: Experience and Reflection in Camus and Sartre*. Oxford.
30. Solomon, R.C. (2006b). True to Our Feelings: What Our Emotions Are Really Telling Us. Oxford.
31. Tsuchiya, N. & Adolphs, R. (2007). Emotions and Consciousness. *Trends Cogn Sci*. April 11 (4) 158-167.
32. Wakefiel J.C. & Schmitz, M.F. (2013). When does a Depression become a Disorder? Using recurrence Rates to evaluate the Validity of proposed changes in Mayor Depression Thresholds. *World Psychiatry* 12:1 44-52.
33. Wolpert, L. (2008). Depression in an Evolutionary Context. *Philos Ethics Humanit Med*. (3): 8.