



## MISCONCEPTIONS ABOUT DEPRESSION AND ITS TREATMENT (I)

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*Este trabajo y su segunda parte (Sanz y García-Vera, 2017) analizan la veracidad de diez ideas sobre la depresión y su tratamiento que se defienden en medios de comunicación de amplia difusión en Internet o en algunas guías de práctica clínica y manuales de psicopatología o psiquiatría de prestigio. Estas ideas obstaculizan que los pacientes accedan a un tratamiento adecuado para su depresión y favorecen la medicalización del mismo en perjuicio de los tratamientos psicológicos. En este primer trabajo se contrastan cuatro ideas sobre la naturaleza de la depresión con los resultados de la literatura científica. Una revisión de esta literatura indica que, contrariamente a esas ideas: a) la depresión es considerada actualmente un trastorno mental, no una enfermedad mental; b) la existencia de una causa biológica es simplemente una hipótesis más, no una realidad constatada empíricamente de manera inequívoca; c) los acontecimientos vitales negativos aumentan el riesgo de depresión mientras que la extraversión y el optimismo lo disminuyen, y d) existen tasas de simulación de depresión pequeñas, pero nada despreciables, en contextos clínicos tanto ordinarios como legales o forenses. Los resultados se discuten en el contexto de la fiabilidad de la información sobre la salud en Internet.*

**Palabras clave:** Depresión, Enfermedad mental, Modelo médico, Factores de riesgo, Conocimientos sobre salud, Internet.

*This paper and its second part (Sanz & García-Vera, 2017) analyze the veracity of ten ideas about depression and its treatment that are defended in media widely available on the Internet or in some prestigious clinical practice guidelines or manuals of psychopathology or psychiatry. These ideas hinder patients' access to appropriate treatment for their depression and favor the medicalization of this treatment over the use of psychotherapy. In this first paper, four ideas about the nature of depression are contrasted with the results of the scientific literature. A review of this literature indicates that, contrary to these ideas: a) depression is considered a mental disorder, not a mental illness; b) the existence of a biological cause is simply a hypothesis, not a reality proven empirically in an unequivocal manner; c) negative life events increase the risk of depression whereas extraversion and optimism diminish it, and d) there are small but not insignificant rates of malingered depression in both legal-forensic and ordinary clinical settings. The results of this review are discussed in the context of the reliability of the health information on the Internet.*

**Key words:** Depression, Medical illness, Medical model, Risk factors, Health literacy, Internet.

 In September 8, 2015, in the digital version of the Spanish newspaper *El Mundo* (elmundo.es), an article was published entitled "Misconceptions about depression" that referred to another with the same title from *DMedicina* (dmedicina.com), an Internet portal specialized in health and published by the same multimedia communication group that publishes *El Mundo* (Redacción de *DMedicina* [Editorial team of *DMedicina*], 2015). Currently, this portal is called *CuidatePlus* (<http://www.cuidateplus.com>) and on October 1, 2016 it re-published the same article (Redacción de *CuidatePlus* [Editorial team of *CuidatePlus*], 2016).

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Unfortunately, most of the misconceptions about depression in the article were not as such, that is, they were not wrong in the light of current scientific knowledge. Moreover, both the information and the arguments provided to refute these misconceptions were plagued by erroneous statements and data.

The fact that healthcare information on the Internet is full of errors and inaccuracies and that the quality of websites sharing healthcare information is low is nothing new (e.g., Conesa Fuentes, Aguinaga Ontoso, & Hernández Morante, 2011). However, the errors, inaccuracies and low quality begin to be worrying when the information is published in the digital newspaper that is the second most widely read in Spain and the third most widely read in Spanish in the world (*El País*, 2015), because these errors and inaccuracies could reach millions of people. According to comScore, the official measurer of market audiences in Spain, in September 2015, the month in which the above article was published, almost 13 million people in Spain and more than 10 million in the rest of the world accessed



elmundo.es using personal computers and mobile devices. When some of the statements include declarations (e.g., “psychotherapy cures depression”) that are not only not wrong, but also their being presented as erroneous could have very negative consequences for people with depression and, moreover, they discredit one of the main professional activities of psychologists and produce unfair competition in favor of other healthcare professionals, then those statements require a robust and well-founded response from psychology. If, moreover, some of these statements or some of their variants (e.g., “psychotherapy is effective for mild or moderate depression, but also for severe depression”) are also wrongly considered to be “misconceptions” by a large number of healthcare professionals, including a good number of psychologists, it is worthwhile for these well-founded responses to reach the greatest number of psychologists and other healthcare professionals.

This was precisely the objective of this paper and its second part (Sanz & García-Vera, 2017), for which the ideas on depression and its treatment presented by the Editorial team of *DMedicina* (2015) will be reviewed in the light of current scientific literature, as well as some variants of these ideas that appear in some clinical practice guidelines and psychopathology or psychiatry manuals of prestige and wide diffusion (e.g., American Psychiatric Association, 2010; González Pinto, López Peña, & Zorrilla Martínez, 2009; Clinical Practice Guideline Working Group on Adult Depression Management, 2014; Vallejo Ruiloba, 2005; Vallejo & Urretavizcaya, 2015).

Since the article in *DMedicina* presented assertions (e.g.,

“psychotherapy cures depression”), which, for the most part, it classified as false, and since both this paper and its second part (Sanz & García- Vera, 2017) will argue that what is false is precisely to qualify them as false, in order to avoid these double negations that could lead to confusion for the reader, the present work and its second part will directly present the ideas defended by *DMedicina* (e.g., “psychotherapy does not cure depression”) or some clinical practice guidelines or manuals of psychopathology or psychiatry (e.g., “psychotherapy is not effective for severe depression, but only for mild or moderate depression”), and these ideas will be contrasted with the current scientific knowledge.

In this first paper, the ideas about the nature of depression advocated by *DMedicina* (see Table 1) will be checked, while the second paper (Sanz & García-Vera, 2017) covers the ideas related to the treatment of depression which are defended both by this Internet portal and some clinical guidelines and manuals of psychiatry and psychopathology of prestige and wide diffusion.

Since these ideas do not specify the type of population referred to, it will be understood –as is customary in psychopathology when there is no express mention of age– that they all refer to depression in adults and, therefore, all of the data and conclusions of this work and its second part will refer to this population. However, the lack of specification on the type of population to which they refer is already an important limitation of the ideas defended by *DMedicina* and by these clinical guidelines and psychiatry/psychopathology manuals, since, for example, there are important differences in the area of treatment of depression among adults, children and adolescents. In fact, if such ideas are wrong in relation to adult depression, most are even more so in relation to depression in children and adolescents. For example, if the current scientific literature indicates, as it will be seen in the second part of this work, that psychotherapy is just as effective as antidepressant medication for adult depression and that, therefore, the idea is false that psychotherapy is less effective than antidepressant medication for adult depression, the same literature also indicates that psychotherapy is the only treatment that until now has been shown to be efficacious for depression in children and adolescents and that, in this type of population, no pharmacological treatment has had its efficacy fully demonstrated (García-Vera & Sanz, 2016).

**TABLE 1**  
**MISCONCEPTIONS ABOUT DEPRESSION**

Idea	Supposedly correct answer	Answer according to the scientific literature
1. When everything in life is going well, you can get depressed	True	Partially false
2. Depression is a chronic disease that never disappears altogether	False	False*
3. There are no people who fake depression to get out of work	Partially true	False
4. Optimistic and extraverted people get depressed equally as much as those who are not	True	False

*Note.*  
\* Although the supposedly correct answer and the one backed by the scientific literature coincide, the arguments and data offered to support the former (see text) do not coincide with those obtained from the scientific literature and perpetuate misconceptions about depression and its treatment.

**MISCONCEPTIONS ABOUT DEPRESSION**

***When everything in life goes well, you can get depressed***

The arguments made by *DMedicina* to defend the idea that a person can suffer a depression despite everything in their life going well are the following:

The causes of depression (the reason for the appearance of the illness) should not be confused with the triggers (factors that reveal that someone is sick). A person may be “gestating” a depression and trying to



justify the symptoms (for example, overwork would justify apathy and exhaustion). However, in the face of a vital event such as the death of a relative or a job loss, the disease breaks out. Not everyone who suffers from these triggers gets depressed, however, a very large percentage of people who do not experience adverse personal circumstances do. (Redacción de *DMedicina* [Editorial team of *DMedicina*], 2015, para.7)

The Editorial team of *DMedicina* is right to say that when everything in life is good for a person, there is still the possibility that even then this person can be depressed. However, this argument is partially erroneous, since it suggests that the relationship between stressors and depression is weaker than it really is, and the Editorial team also refers to the idea that depression is a disease and therefore it is dependent on a supposed biological alteration and independent of the vital circumstances. In fact, it is false that a very large percentage of people who do not suffer from stressors become depressed. The presence of previous stressors in people that suffer from depression tends to be the norm, rather than the exception. In a review of the scientific literature in this regard, it was concluded emphatically that “stressors are 2.5 times more likely in depressed patients compared to controls, and that in community samples, 80% of depressive cases were preceded by major life events (...) the recent evidence based on sound methods of stress assessment and novel designs strongly suggests that most episodes of major depression are preceded by stressful life events (although most people do not become depressed even if they experience a negative life event)” (Hammen, 2005, p.294-295).

In conclusion, since the existence of negative life events increases the risk of depression, it should be considered that the idea that when everything in life is good for a person, they can be depressed, is FALSE IN PART, since in those conditions one is less likely to suffer from depression.

But beyond the erroneous arguments and data on the relationship between stressors and depression, what underlies the argumentation of the Editorial team of *DMedicina* is the hypothesis, as yet unproven, that depression is a disease, that is, it is caused by some type of structural or functional alteration of the organism and, therefore, it can only be treated with drugs and, consequently, only the physicians can treat it. This hypothesis is shared by many health professionals and there is nothing objectionable about it. However, it is worrying when not only the media but also some clinical practice guidelines, psychiatry and psychopathology manuals, or scientific and professional articles seem to forget their hypothetical nature and consider, without a doubt, that depression is a disease, not a construct or concept that researchers and mental health practitioners use to understand a very complex reality. The construct of depression serves to understand behaviors related to sadness, lack of interest, sleep problems, lack of appetite, ideas about death, etc., which are associated with very high levels of

distress, dysfunction, limitation or incapacity. At present, the causes of these behaviors are not known for sure and, precisely for that reason, such behaviors are included under the more generic term of “mental disorder”, to underline that, accepting their biopsychosocial multicausality, they may have either psychological, sociocultural or biological factors as fundamental causes (García-Vera & Sanz, 2016).

The fact that antidepressant drugs are efficacious in treating depression in adults, at least for severe depressive disorders (Fournier et al., 2010), and the fact that they appear to exert their action by increasing the levels of serotonin, noradrenaline or dopamine in the brain have led to the development of several etiological hypotheses about depressive disorders which assume that these would be the result of a central deficit of these monoamine neurotransmitters (noradrenergic, serotonergic and dopaminergic hypotheses, respectively) and which are collectively referred to as the monoamine hypothesis (Vallejo & Urretavizcaya, 2015). This reduction in the levels of monoamines in the central nervous system would in turn be the result of some type of dysfunction in the processes of synthesis, storage or release of these neurotransmitters, or abnormalities in presynaptic or postsynaptic receptors.

However, the arguments and data supporting the monoamine hypothesis are problematic (Deacon, 2013; Delgado, 2000; France, Lysaker, & Robinson, 2007). Firstly, the response to antidepressants is not in itself a strong proof that a deficit in monoamine neurotransmitters is the cause of depression. As psychiatrists Delgado and Moreno (2000: 5) pointed out: “some have argued that depression may be due to a deficiency of NE [norepinephrine or noradrenaline] or 5-HT [serotonin], because the enhancement of noradrenergic or serotonergic neurotransmission improves the symptoms of depression. However, this is akin to saying that because a rash on one’s arm improves with the use of a steroid cream, the rash must be due to a steroid deficiency.”

Secondly, the results of studies that have evaluated monoamine levels in depressed people and compared them with those of normal people have not consistently found lower levels in the former, as would be expected from the monoamine deficiency hypothesis. While some studies have found that some groups of patients with depression (e.g., those with depression with melancholic symptoms) have lower levels of serotonin or noradrenaline than non-depressed people, others have found similar levels in patients with depressive disorders and in non-depressed people (Delgado, 2000; Delgado & Moreno, 2000; Gjerris, 1988). Recently, however, a meta-analysis that compared levels of serotonin transporters in patients with depression and in healthy people (Kambeitz & Howes, 2015), based on the results of in vivo studies with neuroimaging, found reduced levels of serotonin transporters in different areas of the brain (e.g., the striatum, amygdala and brainstem), but not in the hypothalamus or thalamus. Taking into account the results of post-mortem studies, in the same meta-analysis, conversely,



different levels of serotonin transporters were not found in either the brainstem, frontal cortex or hippocampus.

Thirdly, the results of monoamine-lowering studies are also inconsistent. In these studies the levels of monoamines in the central nervous system are temporarily decreased through dietary restrictions or the administration of a substance, and later, when the levels have been recovered, the effects of these changes on the mood are measured (Delgado, 2000). The monoamine hypothesis would predict that the reduction of serotonin, noradrenaline or dopamine levels would produce a decrease in mood in the individuals. Ruhé, Mason and Schene (2007) meta-analytically reviewed the studies on this subject and found a decrease in mood in healthy individuals with a history of major depressive disorder and in non-medicated patients with major depressive disorder in remission, but not in healthy individuals without a history of depression.

In summary, therefore, the results of the studies that have tested the predictions of the monoamine hypothesis, although suggestive, are inconsistent and, in any case, indicate alterations in the levels of monoamines in some groups of people with depression or with risk factors for depression, but not in others. Consequently, such studies indicate that monoamine deficiency by itself is not a sufficient explanation of the causes of depression and that the role of serotonin, noradrenaline and dopamine neurotransmission systems in the origin and maintenance of depression is more complex than initially thought. In fact, in spite of the high number of possible biological abnormalities that have been studied as potential biological markers for the diagnosis of depression (more than 25 according to Vallejo & Urretavizcaya, 2015), including various measures of the activity of monoamines, there is currently no biological test with sufficient sensitivity and specificity to diagnose depressive disorders and consequently neither the DSM-IV-TR (American Psychiatric Association, 2000) nor the DSM-5 (American Psychiatric Association, 2013) nor the ICD-10 (World Health Organization, 1992) include such biological markers among the diagnostic criteria for these disorders.

In conclusion, the concept of illness implies the existence of a structural or functional alteration of the organism that is the cause of a certain health problem, but these biological causes have not yet been found for depression. For this reason DSM-IV-TR, DSM-5 and ICD-10 refer to depression using the term "disorder" (e.g., major depressive disorder, dysthymic disorder, persistent depressive disorder, recurrent depressive disorder, etc.), and not "illness", since the concept of a mental disorder is open to the possibility that a person's problematic behavior is due to a biological alteration, but also to a behavioral or psychological dysfunction and, that these conditions can be defined more correctly as a behavioral or psychological pattern of clinical significance due fundamentally to psychological or sociocultural causes (García-Vera & Sanz, 2016). Of course, to say that there is no solid evidence to indicate the existence of

clear biological causes for depressive disorders does not mean that hypotheses about their biological causes as well as potential biological markers of disease must not be proposed and investigated, as is currently being done profusely. What this simply means is that, accepting the biopsychosocial multicausality of depressive disorders, the question of the fundamental causes of these disorders is unresolved and open to numerous hypotheses of many different kinds (biological, psychological, and sociocultural). In short, at present the idea that depression is a disease is simply a hypothesis and a hypothesis that, in any case, would only cover some types of depressive disorders. In fact, even the question of specifically which of these types of depressive disorders are diseases, would be open to discussion. For this reason, neither the DSM-IV-TR nor DSM-5 nor ICD-10 distinguish between different types of depressive disorders based on their causes, beyond the finding that there may be depressive disorders due to a medical illness or induced by the consumption of a substance (e.g., none of these classifications distinguishes between an endogenous type of major depressive disorder, due to biological causes, and a neurotic type of major depressive disorder, reactive or due to psychosocial causes).

#### ***Depression is a chronic disease that never disappears altogether***

According to *DMedicina*, the content of this statement is FALSE, since:

Chronic diseases accompany the patient from the moment they contract them until the end of their life. With current pharmacological treatments, specialists can eliminate all symptoms of depression in almost 90 percent of cases. If it is correctly treated, depression remits, although it is true that symptoms may return on other occasions (Editorial team of *DMedicina*, 2015, para. 9).

The Editorial team of *DMedicina* is correct in saying that it is FALSE that depression can never disappear altogether, but, once again, the argument used to justify their answer is plagued with errors. As discussed in the previous section, today depression cannot be considered an illness, but must be considered a mental disorder. On the other hand, it is not true that the rate of efficacy of the drugs is 90%. I wish it were true. Using as an efficacy criterion a reduction at post-treatment of at least 50% of the symptoms present at pre-treatment measured by the Hamilton Rating Scale for Depression (HRSD) or a post-treatment score on this scale ranging between 6 and 12, in the meta-analytic review by Steffens, Krishnan and Helms (1997), response rates of 48% and 48.6% were found for, respectively, selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants, results that coincide with those of more recent meta-analytic reviews. For example, using as a criterion of posttreatment efficacy a reduction in the HRSD score of at least 50%, Magni et al. (2013) found a response rate of 52.9% for



fluoxetine, the first of the SSRIs introduced on the market, which soon became the most prescribed antidepressant in many countries under the trade name Prozac, and a response rate of 53.7% for all other antidepressants. Leucht, Huhn and Leucht (2012) found a response rate of 56.6% for amitriptyline, a tricyclic antidepressant in use since 1961 and still among the most popular in some European countries. Omori et al. (2010) found a response rate of 52.5% for fluvoxamine, one of the oldest and most prescribed SSRIs in many countries, and 52.8% for all other antidepressants. Based on the data presented by Cipriani, Santilli et al. (2009, Figure 3), a response rate of 62% can be calculated for escitalopram, one of the latest SSRIs introduced on the market, and 57.7% for the rest of the SSRIs. Finally, based on the data of Cipriani et al. (2010, Figures 3 and 10), response rates of 53.7% and 64.7% can be estimated for sertraline, another of the latest SSRIs introduced on the market, and 53.4% for tricyclic antidepressants and 59.6% for the rest of the SSRIs. Thus, in summary, the efficacy rates of antidepressant drugs at post-treatment range from 50% to 60%, and even for the drugs considered by experts today to be the most efficacious – escitalopram and sertraline (Cipriani, Furukawa et al., 2009) –, they do not exceed 65%. In this sense, in a recent meta-analysis by Johnsen and Friborg (2015) on the results of 43 studies, it was shown that, at the end of cognitive-behavioral therapy, 57% of patients could be considered to have recovered from their depression. Therefore, the efficacy of psychotherapy –at least for treatments that have solid empirical support, such as cognitive-behavioral therapy– is no different from that of antidepressant medication (Sanz & García-Vera, 2017).

#### ***There are no people who pretend to be depressed to get leave from work***

The idea that, apart from the exceptions, there are no people who pretend to be depressed in order to get leave from work is defended by *DMedicina* on the basis of the argument that, “although in some anecdotal cases physicians can be deceived, it is generally very difficult to simulate depression” (*DMedicina*, 2015, para. 15).

The statement, as it is formulated, i.e., with that level of generality, is FALSE, since, with all the limitations and cautions of the research on the prevalence of malingering (Santamaría Fernández, 2014), there is no doubt that, at least in the medical-legal field, there are a not inconsiderable number of people who simulate depression. For example, Mittenberg, Patton, Canyock and Condit (2002) estimated, based on a study carried out in the USA with 131 neuropsychologists, that the simulation rates for diseases, mental disorders and health problems could range from 8% in general medical or psychiatry consultations up to 30% in contexts of disability or work compensation claims, and among the cases of simulation in litigation or indemnification contexts, almost 15% pretended to have a depressive disorder. In the same vein, a study carried out in Spain with 161 physicians, the majority of insurance companies in charge of

dealing with work leave cases indicated that their perception is that 50% of the patients who come for leave due to depression could be malingering (Santamaría, Capilla Ramírez & González Ordi, 2013).

However, if what is meant by the assertion in this heading is that the vast majority of people attending primary care, psychiatry or psychology clinics who complain of depression are not simulating this disorder, the simulation rate of 8% provided by Mittenberg et al. (2002) for this context would adequately justify this.

Regarding the argument that physicians cannot be deceived, except in very rare cases, and that it is very difficult to simulate a depression, it is important to note that although there is not much research on this subject, and even less specifically about the simulation of depression, studies do seem to indicate the existence of a bias among clinicians to believe that simulation is evident to a well-trained professional when, in fact, the data indicate that “the ‘clinical eye’ is inaccurate in the correct determination of simulation, which can give rise to a high number of both false positives and false negatives” and, furthermore, “the practitioners’ confidence in their ‘clinical eye’ has not proved to be a good predictor of this capacity for accuracy” (González Ordi, Santamaría-Fernández, & Capilla Ramírez, 2013, p.10; see also Santamaría Fernández, 2014).

In conclusion, given the not inconsiderable simulation rates in both ordinary clinical contexts and legal or forensic ones and given the fallibility of clinical judgment, it should be considered that the idea that there are no people who fake depression to get out of work is FALSE.

#### ***Optimistic and extraverted people get depressed as much as those who are not***

The *DMedicina* article argues that optimistic and extraverted people get depressed equally as much or even more so than those who are not optimistic or extraverted, affirming that “precisely the personality profile that is most extraverted and euphoric is that which has a greater affective load and, therefore, more risk of suffering a depression” (*DMedicina*, 2015, para.17).

It is true that optimistic and extraverted people can also get depressed, but the scientific literature unequivocally points out that their risk is lower than that of pessimistic and introverted persons or that of people with other personality traits and, therefore, the idea that optimistic and extraverted people get depressed as much or more than people who are not optimistic or extraverted is FALSE and the argument that *DMedicina* offers to defend this idea is also incorrect. For example, in their meta-analysis, Kotov, Gamez, Schmidt and Watson (2010) demonstrated that, in comparison with the control groups (e.g., groups of people representative of the general population), patients with unipolar depression and dysthymia showed significantly lower levels of extraversion, with large differences in terms of effect size (Cohen’s  $d = -0.92$  for unipolar depression



and Cohen's  $d = -1.47$  for dysthymia), while patients with major depressive disorder also had lower levels of extraversion, although the difference was only moderate ( $d = -0.62$ ) and it was not statistically significant. In contrast, all groups of patients with unipolar depression, dysthymia, and major depressive disorder showed significantly higher levels of neuroticism, all with large differences compared to control groups (between  $d = 1.33$  for major depression and  $d = 1.93$  for dysthymia).

Regarding optimism, a meta-analysis on its main measure as a personality trait, the Life Orientation Test, found a negative, significant and almost large ( $r = -0.46$ ) relationship between optimism and depression measured by the Beck Depression Inventory (Andersson, 1996). In the same vein, several studies have found that optimism is a protective factor for the future onset of depressive symptoms (Giltay, Zitman & Kromhout, 2006; Vickers & Vogeltanz, 2000). Furthermore, in a study with monozygotic twin pairs in which one of them had suffered from major depressive disorder at some time in their life, while the other had not, it was found that, after controlling for a good number of possible third variables, the existence of a low level of optimism was one of the characteristics that best distinguished the twins with major depressive disorder from the twins without the disorder (Kendler & Gardner, 2001).

In summary, the scientific literature demonstrates that extraversion and optimism are two personality factors that are negatively related to depression and, therefore, the idea that optimistic and extraverted people become depressed equally as much as or more than people who are neither optimistic nor extraverted is FALSE, since optimistic and extraverted people are less likely to suffer from depression.

## CONCLUSIONS

The objective of this paper and its second part (Sanz & García-Vera, 2017) was to analyze, in light of the current scientific literature, the veracity of ten ideas about depression and its treatment that are defended in the widely disseminated media on the Internet and even in some prestigious clinical practice guidelines and psychopathology and psychiatry manuals. Of the four ideas on the nature of depression analyzed in this paper, three are considered by these media to be true or partially true and the remainder are considered to be false. However, according to the current scientific literature, the three true or partially true ideas are actually false or partially false, and the remainder are actually false, but for reasons other than those argued in the media (see Table 1). In summary, contrary to the ideas defended by these media: a) depression is considered a mental disorder, not a mental illness; b) the existence of a biological cause is simply another hypothesis, not a reality that has been empirically verified unequivocally; c) negative life events increase the risk of depression while extraversion and optimism diminish it, and d) there are small, but not insignificant, rates of depression simulation in both ordinary and legal or forensic clinical contexts.

The problem with these divergences between what the scientific literature says about depression and what the ideas and arguments of some media say about it is that the latter present depression as a disease, when this conceptualization is currently only a hypothesis. In doing so, these media promote, with no scientific basis, the medicalization of the treatment of depressive disorders to the detriment of the application of psychotherapy, even when the latter, or at least cognitive-behavioral therapy, has a better efficacy profile than that of antidepressant medication, as demonstrated in the second part of this work (Sanz & García-Vera, 2017). In fact, several studies have shown that an explanation of depression in terms of disease, such as in terms of the existence of a neurochemical imbalance, encourages people to be more pessimistic about their possible recovery and about the efficacy of non-biological treatments (Deacon & Baird, 2009).

The present paper and its second part confirm again that not all health information found on the Internet is reliable, even when it comes from a site specialized in health information (Conesa Fuentes et al., 2011; see, however, Mayer, Leis & Sanz, 2009, for a more positive assessment of quality in some specific healthcare areas). In fact, *DMedicina* and its current version, *CuidatePlus*, are health portals that subscribe to the principles of the *Health on Net* code of conduct (HON; <http://www.hon.ch>). HON is an NGO founded in 1995 and accredited by the Economic and Social Council of the United Nations. Its code of conduct, HONcode, offers a consensual standard to protect citizens from misinformation about health and is the most internationally widespread quality seal (Mayer et al., 2009). This code of conduct supposedly guarantees the authority of the content (i.e. the news and advice published on the portal are provided by medical professionals) and its justification (i.e., the treatments, products and services mentioned in the portal are backed by scientific and referenced information). This is more worrying since it ultimately refers to the reliability of these medical professionals and this scientific and referenced information (e.g., clinical practice guidelines, psychiatry or psychopathology manuals), which, in some cases and as will be seen in the second part of this paper, also perpetuate ideas that do not correspond to current knowledge about depression and its treatment.

However, despite these limitations, this work and its second part do not intend to recommend that mental health information should not be consulted on the Internet, but that it should be done critically. To do this, some advice, in the form of a question, based on that offered by the National Institute of Aging of the USA (National Institute of Aging, 2014), may be of great help to both patients and health professionals:

- ✓ Can you easily check who sponsors the website?
- ✓ Is the sponsor of the website an official institution (e.g., a ministry or health council), a professional association, a medical school or psychology faculty, or is it related to one of these entities?



- ✓ Can you distinguish the sponsor's mission or goal from the website?
- ✓ Can you identify who works for the sponsor and who is the author of the information presented on the website? Is there information included for contacting them? Who reviews the information? Is there an editorial board that reviews the information included on the website?
- ✓ Can you determine when the information was written?
- ✓ Are statements made that are too good to be true? Do they promise quick and miracle cures? Do these cures extend to all kinds of disorders and diseases?

Moreover, the authors trust that by publishing this work and its second part in a journal with open access on the Internet and sponsored by the Spanish Psychological Association, the correct answers, within the limits of current scientific knowledge, to the ideas that are presented in Tables 1 of this article and its second part will be able to reach both patients and health professionals, and, moreover, will serve as a reliable counterpoint to the truly erroneous ideas about depression and its treatment that proliferate on the Internet.

#### CONFLICT OF INTERESTS

There is no conflict of interest

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