Persistent Depressive Disorder (Dysthymia) and Recurrent Unipolar Major Depressive Disorder

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Historical Background/Introduction

The term depression comes from the Latin "*depressio*" and indicates a state of dejection perceived by the subject accompanied by extreme suffering and discomfort. "*Melancholia*" (*melas* means black and *cholé* means bile), the term historically used to describe this condition, was first introduced by Hippocrates in the treatise *On the Nature of Man* [1] based on the humoral theories of Alcmaeon of Croton and then resumed by Galen, who described a melancholic temperament characterized by a black bile excess. This belief was still solid in the sixteenth century, when the French physician Andreas Laurentius related the cause of this pathology to the "coldness and darkness of this humor." In the nineteenth century, Pinel eventually proposed a new theory discontinuing the connection between humor and black bile and describing four new mental disorders, which included Melancholia and Mania [2]. Kraepelin unified all types of affective disorders in the unitary concept of manic-depressive illness, which included "periodic circular insanities," "mania," and "melancholy" [3]. In opposition to this view, Wernicke distinguished five different types of melancholia, going back to taking into consideration the possibility of single episodes of melancholia [4]. Nowadays, the term melancholic represents a subtype of major depressive disorder.

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Clinical Manifestation

Core symptoms that constitute the current definition of depression include depressed mood and anhedonia (reduced ability to experience pleasure from natural rewards) accompanied by neurovegetative symptoms (abnormalities in appetite and sleeps), sexual dysfunction, feelings of worthlessness, alterations in psychomotor skills and in cognitive area, and recurrent thoughts of death.

Epidemiological investigations show how depressive disorder has the highest lifetime prevalence of any psychiatric disorder. Lifetime prevalence for major depressive disorder varies between 5% and 17%. This type of episode can be resolved completely or partially in about two-thirds of cases and is not resolved in about a third. Dysthymia has 3–6% lifetime prevalence and an age of onset at about 20 years. Recurrent brief depressive disorder has an estimated lifetime prevalence of 16% [5].

Patients suffering from depressive disorders frequently describe a feeling of depression, hopelessness, guilt, worthlessness, and inadequacy, sometimes accompanied by a pervasive state of concern, irritability, loss of appetite, weight loss, and insomnia. Typical manifestation of this syndrome is asthenia, agitation or psychomotor slowing (mutism and stupor), and, on the cognitive level, memory gaps, and, with difficulty concentrating, decreased libido. Recurrent ideas of death and suicide are found in the most severe depressions [6]. It is not uncommon for patients to report having lost the ability to feel emotions and hopes for healing; in some cases, these beliefs can assume a delusional nature, especially of a nihilistic type (delusion of guilt, ruin, bodily denial, Cotard delusion) or hypochondriac type. Hallucinations may also be present in the form of accusatory voices or visions of deceased people, accompanied by a strong sense of guilt. If present, both delusions and hallucinations are usually mood-congruent.

Definition and Classification of DSM-5

The DSM-5, introduced in 2013, classifies depressive disorders and bipolar disorders separately, removing the broad category of mood disorders [6].

Among the five symptoms necessary for the diagnosis of a major depressive episode, the DSM-5 signals the depressed mood and the loss of pleasure or interest and then lists a series of other symptoms of the abovementioned areas associated with nuclear symptoms, which must be present for at least 2 weeks.

The DSM-5 includes a new classification of chronic depression introducing persistent depressive disorder (dysthymia), which represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder. This change was caused by the evidence showing the difficulty of differentiating the two diagnoses and their frequent co-occurrence [7]. Persistent depressive disorder is diagnosed when a depressed mood and two or more between a list of six symptoms (poor appetite/overeating, insomnia/hypersomnia, low energy/fatigue, low self-esteem, poor concentration or difficulty making decisions, and feelings of hopelessness) are present for at least 2 years (Table 1). During this period, any symptom-free interval must last no longer than 2 months (Table 2).

Table 1 Persistent depressive disorder (dysthymia) diagnostic criteria according to DSM-5

A. Depressed mood for most of the day, for more days than not, as indicated by either subjective account or observation by others, for at least 2 years **Note**: In children and adolescents, mood can be irritable and duration must be at least 1 year
B. Presence, while depressed, of two (or more) of the following: 1. Poor appetite or overeating 2. Insomnia or hypersomnia 3. Low energy or fatigue 4. Low self-esteem 5. Poor concentration or difficulty making decisions 6. Feelings of hopelessness
C. During the 2-year period (1 year for children or adolescents) of the disturbance, the individual has never been without the symptoms in criteria A and B for more than 2 months at a time
D. Criteria for a major depressive disorder may be continuously present for 2 years
E. There has never been a manic episode or a hypomanic episode, and criteria have never been met for cyclothymic disorder
F. The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder
G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse and medication) or another medical condition (e.g., hypothyroidism)
H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning

Table 2 Major depressive disorder diagnostic criteria according to DSM-5

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure **Note:** Do not include symptoms that are clearly attributable to another medical condition 1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, and hopeless) or observation made by others (e.g., appears tearful). (**note**: In children and adolescents, can be an irritable mood) 2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation) 3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**note:** In children, consider failure to make expected weight gain) 4. Insomnia or hypersomnia nearly every day 5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down) 6. Fatigue or loss of energy nearly every day 7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick) 8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others) 9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide
B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
C. The episode is not attributable to the psychological effects of a substance or to another medical condition

Pathophysiology

Several factors are involved in the pathophysiology of depressive disorder such as life events, genetics, neurotransmitters, and neuroendocrine alterations. No single mechanism seems to be able to explain every aspect of this disease. Premorbid personality traits and psychosocial and biological stressors could contribute to the development of a depressive disorder and eventually of a persistent/recurrent form. The depressogenic role of scarce coping resources when facing stressful life events was recently introduced by Tellenbach, who described a personality vulnerable to the development of depression (melancholia) called Typus Melancholicus [8]. The constitutive traits of this personality are orderliness, conscientiousness, hyper/heteronomy, and intolerance to ambiguity, and they represent the nucleus through which the vulnerability to depressive syndrome is expressed. Typus Melancholicus is considered a fundamental construct for understanding the premorbid and intermorbid personality structure liable to the previously defined "endogenous depression." In this regard, it is important to notice that within the current nosographic classification, the "endogenous" and "reactive" opposition was abandoned. In the most recent edition of the Classification of Mental and Behavioural Disorders of the World Health Organization (ICD-10), the adjective "endogenous" was replaced by the term "somatic" and was used to characterize the depressive episode under the symptomatological point of view. Major depressive disorder with Melancholia is a subtype that is interpreted as a clinico-descriptive value, beyond any etiopathogenetic implication.

On the other hand, according to the cognitive model of personality, a depressive personality style is well documented and results to be often undiagnosed or misdiagnosed as a dysthymic disorder. Bowlby named "avoidant" certain attachment behaviors that would lead to a pattern of dysfunctional organization together with early trauma experiences [9]. Children without a secure-base attachment figure are prone to a constant self-referral with a lack of social interactions that emphasize their sense of loneliness. Thus, personal identity will be based on emotional reactions to the experience of loss that return in feelings of desperation, anger, and hopelessness. The depressive personality organization entails a "self-meaning" of loneliness and a particular sensitivity to every loss situation that could happen during life.

In a more complex conception, it is believed that between a stressful event, a personality trait, and a mood disorder there is an interactive—rather than casual—connection, in which a series of genetic, biological, psychological, and environmental factors are involved. Constitutional predisposition and stressing factors are considered complementary based on a psycho-socio-biological model, which allows a unified interpretation of the adaptive process in their dynamic becoming.

Among the identified involved factors, several studies have highlighted how coping styles and the defense mechanisms used by individuals play a fundamental role in the development of depressive symptomatology. More in detail, research has shown that coping styles mainly based on avoidance mechanisms and immature or

primitive defense mechanisms are more correlated with anxiety and depressive disorders. The complex interaction between coping styles and defense mechanisms could provide further elements to better understand how individuals relate to the disease.

Coping skills are the turning point to understand how similar stimuli can elicit different responses even from a neurobiological point of view. Following a stressful life event, many neurotransmitters, hormones, and cytokines work in order to produce a stress response aimed at maintaining homeostasis. The hypothalamic–pituitary–adrenal axis (HPA), in response to stress and modulated by amygdala and prefrontal cortex, cooperate with the nervous and immune system for the fight or flight reaction. Major depression is strongly associated with hyperactivity of the HPA [10–12] and an increased amount of plasma cortisol. Cortisol imbalance is due to the increased CRH production of the hypothalamus, which communicates with the sympathetic system as an acute response determining a release of adrenalin and noradrenalin in response to a perceived dangerous event. An anxiolytic response balancing the fight or flight CRH response is activated by peptides called urocortins. Both CRH and urocortins are regulated by glucocorticoids. Cortisol hyperproduction and CRH overdrive determine an alteration in the balance of monoamines and neuronal atrophy in the amygdala and in several areas of the prefrontal cortex. While an acute stress response activates the monoamines, the chronic stress response determines a reduced activity of serotoninergic, dopaminergic, and noradrenergic neurons.

The "monoamine hypothesis," positing that depression is caused by decreased monoamine function in the brain, derives from the observation made in the mid-twentieth century that the anti-hypertension drugs that would reduce the amount of monoamine could cause depression in a subset of patients. Such theory was supported by the evidence of a "mood elevation" caused by monoamine oxidase inhibitors (MAOIs) and by tricyclic drugs, which increased the availability and excitatory effect of neurotransmitters. Today's antidepressant drugs offer lower rates of side effects, but their efficacy is still linked to the increase in monoamine transmission. Although these drugs produce immediate increases in monoamine transmission, weeks of treatment are required in order to establish mood-enhancing properties. These characteristics led to hypothesis that their efficacy could be linked to secondary neuroplastic changes involving transcriptional and translational changes mediating cellular and molecular plasticity [13].

Studies investigating the genetic contribution to the development of a depressive disorder showed a higher-than-chance incidence of depression among first-degree relatives. Twin studies suggest that genes account for 40–50% of the susceptibility to major depressive disorder in the population [14]. The most accepted hypothesis suggests that different genes interact with the environment increasing the individual's susceptibility [15].

The role of functional polymorphisms in a small set of genes, which could be involved in monoaminergic neurotransmission and in the development and resistance to treatment of depressive disorder, was studied. Most of them are implicated in the synthesis, degradation, or neurotransmission of serotonin (5-HT). Current

research comprises the loci encoding the serotonin transporter SLC6A4 and its methylation [16, 17], the limiting enzyme for dopamine synthesis called tyrosine hydroxylase (TH) [18], the serotonin 2A receptor (5HTR2A), and the tryptophan hydroxylase 1 (TPH1) [19]. Other genes that have been studied are involved in dopamine catabolism (COMT) [20] and the dopamine receptor (DRD4). Regarding dysthymia and recurrent depressive disorder, some studies recently focused on the role of TCF4 gene [21] and circadian clock genes [22] on their etiopathogenesis.

It is noticed that a recent study on large samples focused on 18 genes empirically identified as commonly studied in the last 25 years and do not support previous depression candidate genes findings. It was posited that early hypotheses about depression candidate genes were incorrect and that the large number of associations reported are likely to be false positive [23].

Following works studying the adaptive functions of anxiety, an expanding body of literature has focused on functional explanation of depressive disorder, proposing mood states as the target for an evolutionary analysis. According to these views, depression serves as a method for, de-escalation, energy conservation and for bonding to caregiver. The social risk hypothesis, integrating the over mentioned theories, suggests that depressive phenomena can be considered a defensive psychobiological response to increased risk of exclusion from social contexts vital to dealing with adaptive, socio-reproductive challenges [24].

Assessment

Besides the abovementioned DSM-5 classification, also the International Classification of Diseases (ICD) relies on the presence of a number of key symptoms. Recurrent depressive disorder can be diagnosed when repeated episodes of depression are present without any episode of mania; dysthymia can be diagnosed when a depression of mood is not sufficiently severe or prolonged to diagnose a depressive disorder that lasts at least several years [25]. It is important to notice that a fundamental approach to diagnose a depressive syndrome is the clinical evaluation. Besides the formerly mentioned symptoms, clinical manifestations can be found in signs such as deceleration and reduction in the speech rate, scarce facial expression, frequent crying, and poor care of one's appearance. Activation and anxiety could be present, and affective involvement could be exaggerated or poor. In some cases, somatization is the principal mean of expression of depressive syndrome, especially in elderly patients [26] or some ethnocultural groups [27, 28]. Once depressive syndrome is diagnosed, several tools can be used to assess its severity and to measure the outcomes of the interventions. The Beck Depression Inventory (BDI) [29], the Montgomery–Åsberg Depression Rating Scale (MADRS) [30], and the Hamilton Depression Scale HAM-D [31] are administered by a mental health professional, while the Zung Self-Rating Depression Scale [32] is a self-administered tool. It is also important to provide an in-depth investigation into the suicidal ideation and behavior of patients presenting a depressive syndrome. The

Columbia Suicide Severity Rating Scale (C-SSRS) [33] represents a fundamental tool for its exploration.

Treatments

In 2009, the National Institute for Health and Care Excellence published guidelines for the recognition and management of depression in adults, updated in 2018. The proposed stepped-care model provides a sequence of interventions starting from the most effective and less intrusive one (assessment, support, psychoeducation, active monitoring, and referral for further assessment and interventions) to evolve, when necessary, into more targeted interventions. Step 3 and step 4 are dedicated to mild-to-severe forms of depression resistant to treatment for which proposed therapies start from medication, high-intensity psychological interventions, combined treatment, and collaborative care for step 3 with the addition of ECT, crisis service, and multiprofessional and inpatient care in addition for step 4.

In 2016, the Canadian Network for Mood and Anxiety Treatment published an update of the evidence-based clinical guidelines for the treatment of depressive disorder published in 2009 [34]. The first of the six sections is dedicated to psychological treatments, which are especially indicated in moderately severe and low-risk cases, depending on patient preferences, and availability of treatment, with the exclusion of psychotic depression. CANMAT guidelines report studies that showed the comparison in the efficacy of specific models of psychological treatments and propose a list of recommendations. The efficacy in comparison with control groups—and not to other psychological treatments—is expressed by the first- to third-line treatments.

Cognitive-behavioral therapy (CBT), interpersonal therapy (IPT), and behavioral activation (BA) are first-line acute treatments, while CBT together with mindfulness-based cognitive therapy (MBCT) are first-line maintenance treatments. Either CBT or IPT combined with SSRIs or TCAs shows more effectiveness compared to psychological treatment alone and psychological treatment with a placebo or with antidepressants alone [35, 36] (Table 3).

Starting from the mid-twentieth century, pharmacotherapy for depressive symptoms has been founded on the enhancement of monoaminergic neurotransmission, but newer antidepressant agents target different brain systems such as melatonin, NMDA receptors, or GABA. Available antidepressants act on postsynaptic and presynaptic receptors and neurotransmitter transporters, and they may be classified according to many aspects, notably based on their principal pharmacological action as in the proposed Table 4. Pharmacodynamics and pharmacokinetics of different classes go beyond the scope of this paper.

The effectiveness of continuation and maintenance treatments for persistent depressive disorder was recently investigated. Ten studies were described comparing pharmacological (SSRIs, MOIs, SNDRIs, TCAs), placebo, and psychological therapies in seven different combinations. Five studies showed to favor continuation

Table 3 Recommendation for psychological treatment for acute and maintenance treatments of major depressive disorder. Partly adapted from CANMAT guidelines

	Acute treatment	Maintenance treatment (relapse prevention)
Cognitive-behavioral therapy (CBT)	First line	First line
Interpersonal therapy (IPT)	First line	Second line
Behavioral activation (BA)	First line	Second line
Mindfulness-based cognitive therapy	Second line	First line

Table 4 Principal pharmacological actions

Class	Drugs
TCAs	Amitriptyline, clomipramine, doxepin, imipramine, nortriptyline, trimipramine
MAOIs	Tranylcypromine, moclobemide, phenelzine
SSRIs	Citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline
SNRIs	Desvenlafaxine, duloxetine, levomilnacipran, milnacipran, venlafaxine
NDRIs	Bupropion
SMSs	Vortioxetine
Others	Mirtazapine, trazodone, agomelatine, tianeptine, sulpiride/amisulpride, mianserin

and maintenance pharmacotherapy as an effective treatment to prevent relapse and recurrence in persistent depressive disorder compared to placebo. Nevertheless, this primary outcome did not reach significance when only studies with a low risk of bias were included. An appropriate duration of assumption was not drawn emphasizing the need for further studies. Concerning psychotherapy, it was assumed that maintained active psychotherapy has a positive effect on depression outcomes and on relapse or recurrence rate of depression. No statistically significant differences were found when comparing combined psychological and pharmacological continuation and maintenance therapy with therapies alone [37]. It is important to notice that most antidepressants cause sexual dysfunctions [38].

Conclusions

Although recurrent unipolar major depressive disorder and persistent depressive disorder represent two manifestations of chronic forms of depression, these disorders stand out in intrinsic fundamental differences regarding their clinical presentation and etiopathogenesis. While the former shares some aspects with unipolar but also with bipolar disorder in which etiopathogenesis is more easily reported to

neurobiological causes than to life events, the latter represents a personality trait characterizing a life path. As described in 1963, a depressive personality determines a feeling of a "little of the normal joy of living" in people "inclined to be lonely and solemn, to be gloomy, submissive, pessimistic, and self-deprecatory (…) prone to express regrets and feelings of inadequacy and hopelessness" [39]. Such a description of depressive personality is consistent with more recent studies showing how being anxious, pessimistic, and shy could be related to future depressive symptoms, while being responsible, purposeful, and resourceful could be a marker of executive functions that protect a person from depression [40].

Sexuality and Quality of Life

While there is statistical evidence that sexuality is practiced from adolescence [41], it seems likely that sexually active adolescents often use birth control inconsistently, have multiple sexual partners, and use alcohol or drugs at the time of sex.

These behaviors are often associated with mood disorders and depression. Approximately 14% of adolescents have experienced at least one episode of major depression, resulting in a serious impairment of emotional stability: at home, at school, or at work, in intimate relationships or in social life [42]. All the more so since the percentage has risen considerably due to the COVID 19 pandemic and the resulting emotional fragility.

The scientific literature associates the phenomena of adolescent depression with family events experienced at an early age [43].

For example, early parental separation (before age 5) can prompt multi-partner sexual behavior in adolescence, while early father absence anticipates higher rates of sexual activity among 16-year-old female adolescents [44].

The timing of parental relationship instability may also predict increased psychological distress among children and adolescents [45].

When an individual's parents separate in early childhood, they tend to have poorer well-being as pre-adolescents than those whose parents separated later in childhood [46].

Depressive symptoms associated with the experience of parental separation during childhood may also worsen over time as individuals reach adolescence and early adulthood.

Accordingly, studies have shown *early emotional instability, sexual behavior, and depression in adolescents.*

A study conducted by Kelly L. Donahue BA et al., [47] assessed 585 children (52% male; 81% Caucasian) and their families. Assessments were conducted annually, and those who moved after the initial assessment were followed up by mail or telephone. At age 24, 83% of the original sample ($N = 484$) continued to participate in the assessments.

The study started from the hypothesis that parental relationship instability before the age of 5 years would be associated with a higher probability of reporting early

sexuality (SP) at the age of 16 years and a higher probability of experiencing a major depressive episode (MDE) between the ages of 13 and 18 years.

The following were assessed: (a) less parental awareness of activities, (b) parents with little interaction with children, (c) exposure to numerous transitions in the parental relationship (change of city, job, new marriage, etc.) during development, or (d) fewer available caregivers (grandparents and friends).

Methods of the Survey

At the age of 12, using a 3-point scale, adolescents rated how much their parents knew about their friends, shopping, after-school activities, and leisure time [48]. At age 13, interviewers asked primary caregivers (94% mothers, 3% fathers, and 3% others) a series of 35 questions about their adolescents' homework, school experiences, entertainment choices, and peer relationships. The interviewers rated how knowledgeable the parent was in each of the four domains, using a five-point scale.

At the age of 12, adolescents reported on their pubertal development in terms of height growth, body hair growth, and skin changes; facial hair growth and deepening of the voice (boys); and breast growth and menstruation (girls).

Results: At the age of 16 years, more than one-third of participants reported SP during the previous year. The prevalence of sexual activity at this age is in line with rates found in larger population-based samples [49, 50]. Between the ages of 13 and 18, 16% of participants had at least one MDE, consistent with estimates from recent epidemiological studies.

The identification of individuals most at risk of pathological outcomes, such as those experiencing parental separation or divorce prematurely, could help sexologists and psychiatrists to build a personalized treatment model for each adolescent experiencing depression and deviant sexual behavior [51].

And the Adults?

Depression in adults can lead to hypersexuality, compulsive sexual behavior (often also indicative of bipolar disorder), psychosocial distress, relationship, and work problems [52].

Some articles in the literature have investigated the relationship between hypersexuality and trauma in veterans, highlighting various forms of sexual compulsivity or problematic sexuality associated with traumatic life experiences [53].

In this regard, it has been hypothesized that hypersexuality may arise more generally from PTSD symptom (abuse, traumatic death of a parent, abandonment, violence, etc.).

This would explain how in the case of depression, adult hypersexual behavior is acted out in order to cope with internal suffering caused by trauma in the past and related to psychopathological symptoms.

There Is More

Drawing on the pandemic period, some studies have assessed the interaction between depression, sexuality, and alcohol abuse.

Specifically, Ellesse-Roselee Akré et al. collected, from May 21 to July 15, 2020, 3245 adults living in the five major metropolitan areas of the United States (Atlanta, Georgia; Chicago, Illinois; New Orleans, Louisiana; New York, New York; and Los Angeles, California) [54].

Participants were categorized as straight cisgender or LGBTQ+ (i.e., lesbian, gay, bisexual and transgender people, and men who have sex with men and women who have sex with women who do not identify as lesbian, gay, bisexual, or transgender).

The age groups examined were 18–26, 27–49, 50–64, and ≥65 years. They were subdivided by gender (male and female), race/ethnicity (African American/Black, Asian, Hispanic/Latino, White), educational level (below high school, high school diploma, some college or associate or technical degree, and bachelor's degree or higher), household income in relation to the federal poverty level (FPL; according to the US Department of Health and Human Services) relationship status (married or partnered; in a romantic relationship; widowed; and single, divorced, or separated); health insurance (uninsured, private, public, and other); and city of residence (Atlanta, Chicago, Los Angeles, New Orleans, and New York City).

The results showed more frequent psychological distress and alcohol abuse in times of pandemic among LGBTQ+ and heterosexual cisgender people. Heterosexuals reported hypoactive craving and increased eating compulsivity, frustration, anhedonia, memory and sleep disturbances, and mild depression.

Megan E Patrik et al. [55] confirmed in a study from spring 2019 to autumn 2020 that the young adults studied (1244) reported stress, economic difficulties, mood disorders, autoerotic compulsivity, and drug use to cope with the COVID-19 pandemic.

In particular, 15.7% of the sample reported marijuana use, 8.9% reported increased use of some type of substance, and 8.2% reported increased use of alcohol. About 1% reported smoking more cigarettes, using prescribed and/or non-prescribed medication for mood disorders. [56, 57].

Staying with the pandemic theme, studies [58] have also shown a lowering of mood and sexual frequency with a significant drop in female sexual function index (FSFI) scores in adult pregnant women.

We refer to hypoactive desire, disturbance of arousal, lubrication, orgasm, satisfaction, and pain, with negative effects on the quality of life of affected women.

Data show a reduction of up to 64% in interest in sexual activity after childbirth. The return to sexual activity occurs on average 4 months after childbirth, varying from 1 month to 2 years.

Therefore, the importance of assessing women's sexual health in one of the countries most affected by COVID-19 has become apparent, especially during a vulnerable period with regard to sexual dysfunction, such as puerperium [59].

It appears that sexual dysfunction in the third trimester of pregnancy is linked to the fear of hurting the fetus, triggering mechanisms of low self-esteem, mood disorders, and relationship problems [60, 61].

Conclusion

Depression, sexuality, and quality of life remain a triad that must be investigated with the utmost care and by means of a multi-specialist criterion: psychiatric and psychosexological. The complementary anamnesis would allow the patient to learn the causes of his or her behavior and to associate the pharmacological therapy with a process of re-evaluation of himself or herself and of his or her weaknesses. Any hyposexual or hypersexual behavior would take on more relevance in the individual's life, limiting the use of alcohol and substances.

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