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Psychobiology and Treatment of Personality Disorders and Related Prominent Traits

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Abstract

Introduction: This theoretical narrative perspective will focus on pointing out the psychobiological factors which are involved in the genesis and treatment of personality prominent traits and disorders. **Material and method**: After a suitable search, mainly in PubMed and Psyc Info Journal databases, the number of selected quality publications is n= 95. The reduced sample sizes and numerous research designs that are merely exploratory or observational are its main limitations. **Results and discussion**: Regarding the psychopathology of personality neurodevelopment, environmental and genetic factors as well as neurotransmitter impairments are confirmed to be involved. Of interest are the endophenotypic perspective, endocannabinoid system and hypothalamus-regulated endocrine axes, among others. Neuroimmunology predicts important clinical and therapeutic innovations, but they are not a reality yet. It has been suggested that only with psychological interventions of various types is there symptomatic improvement as well as a favourable effect on stress-related epigenetic mechanisms. Psychopharmacotherapy continues to be useful, mainly off-label, with antipsychotics, anticonvulsants and opioid antagonists. **Conclusions**: In the absence of support from convincing qualified evidence, both users and health physicians usually prefer any type of psychological intervention to tackle complex personality clinical dysfunctions, without excluding complementary psychopharmacotherapy.

Keywords: *Psychopharmaceutical, Limit pattern, Mood stabilizer, Personality disorder, Psychotherapy*

Highlights

- Certain personality disorders are inherited, as is the case with ordinary personality and temperament.
- Some dimensions of personality are related to neurotransmitters and other endophenotypic biomarkers.
- The endocannabinoid system appears to be central to emotional homeostasis and cognitive function.
- Peripheral inflammatory processes probably influence neurotransmitter alterations causally.
- Combined treatment with psychotropic drugs and psychotherapy seems to be more effective for personality disorders than both alone.

Introduction

Qualified empirical evidence about the psychobiological or psychosomatic aspects of personality disorders and related traits or patterns, including which treatments are most effective, is insufficient. Such disruptions have a major impact on health resources. Primary care physicians play an important role in initial diagnosis and therapeutic planning [1]. The extensive comorbidity that occurs in the heterogeneous group of these characteristic disorders can be explained by shared genetic and environmental risk factors, and their treatment will have to be personalised to the most relevant issues.

Personality disorders in the *International Classification of Diseases*, eleventh edition (ICD-11

for Mortality and Morbidity Statistics), also in the alternative *Diagnostic and Statistical Manual of Mental Disorders*, Text Revision (DSM-5-TR) or in its complementary *Research Domain Criteria* (RDoC) project, involve pervasive, inflexible and stable styles of thinking and feeling and often disturbances in behaviour and interactions with others. The new ICD-11 is likely to improve the clinical use of these diagnoses and their severity [2].

In summary, the experience of stress is closely linked, among others, to certain personality disorders and to disruptive, dissocial behavioural disorders. The hippocampus, prefrontal cortex, amygdala and hypothalamus are involved, as well as the pituitary and adrenal cortex glands with their secretion of glucocorticoids and androgens [3]. It is well known that the hypothalamic-pituitary-adrenal axis is an important primary neuroendocrine mediator of neural and behavioural responses to stress.

There is thus early evidence that early-onset mental conditions, such as disorders and certain prominent personality traits, may importantly reflect differential aberrant expressions of a small number of biological as well as neuropsychological and social factors [4]. Alterations in personal and interpersonal functioning are at the symptomatic core of such disturbances. This contribution is intended to be useful for a more holistic clinical practice, but does not seek to be exhaustive. It will deal exclusively with personality disorders and related traits, and will focus mainly on the psychobiological or biopsychological factors involved and the relevant treatments in use.

Material and Method

The corresponding ICD-11 nomenclature of the World Health Organisation (WHO), of promising reliability and validity, will be used in preference going forward for simplicity. It introduces a new methodical approach to the diagnosis of personality traits and disorders focusing on functional impairments in everyday life. Finally, the corresponding terms from the DSM-5-TR of the American Psychiatric Association will be used.

This article is a structured qualitative, critical review, based on content analysis of a timely, narrative, up-to-date literature review of an issue that is as complex as it is multidimensional. The aim is to synthetically interweave the ideas and arguments that constitute the theoretical and empirical references of the numerous current research studies on the subject, prioritising most of the issues addressed. For this purpose, digital and manual searches were carried out non-systematically until early 2024, with a preference for recent years, mainly in the databases of PubMed, the US National Library of Medicine (National Institute of Health) and the American Psychological Association (*PsycInfo Journal*). The main search terms were *personality disorder*, in combination with *psychotropic medication, neurobiology, neural network, antisocial behavior* and *psychoterapy*. In the following, I have undertaken an expository synthesis so as to remain broad and descriptive, without delving into statistical analysis, covering a huge, discretionally delimited field. It is therefore an updated secondary, qualitative scientific study, with argued or contrasted theoretical components.

With accuracy and objectiveness in accordance with scientific methodology, this contribution's primary objective is to pinpoint the contributions related to clinically significant alterations of disorders and prominent problematic personality traits. The critical review will focus on neurodevelopment, the endocannabinoid system, inflammatory processes, psychic, psychological, verbal or conversational interventions (including psychotherapeutic ones) and psychopharmacology (especially in terms of drug efficacy and tolerability). The set of selected publications of sufficient methodological and empirical quality is n= 95.

The main limitations of the study are the multiple findings based on small sample sizes, observational and only exploratory designs – these three types constitute the majority – as well as the not uncommon risk of poorly controlled bias. These are the main reasons why much research has been discarded from the vast amount of literature published on the subject, and they justify not having conducted more limited systematic reviews, as they were inadequate to the scope of the proposed objectives. With a few exceptions, most of the selected contributions are only suggestive, indicative or of limited value. This is the current state of applied scientific knowledge and the "theoretical framework" or "theoretical development" in this regard, which I summarise below.

Results and Discussion

I. Personality Neurodevelopment and Psychopathology

In the UK, more than two thirds of patients with personality disorders as of 2015 were diagnosed with emotional instability disorder [5]. Such disorders are often considered paradigmatic of severe mental disorders. Genetic and environmental factors, as well as certain neurotransmitters, are most likely involved

in the development of any personality and in the aetiology of its pathologies. Personality is heritable to a considerable extent and a number of genome-wide association studies have shown that temperament is strongly influenced by more than 700 genes that modulate associative conditioning through molecular processes of synaptic plasticity, learning and long-term memory [6,7].

In particular, evidence suggests that "borderline personality disorder" (now described as "personality disorder not otherwise specified" ICD-11) is as heritable as other serious mental disorders [8]. Brain imaging studies have indicated dysregulation in the top-down control of emotions in this pathology [9-11]. Findings already exist providing a neurobiological basis for the different profiles of emotion regulation impairment observed in major depressive disorder ("depressive disorder" in ICD-11) and borderline personality disorder [12]. In contrast, there are insufficient data on which to base a comprehensive neurobiological model of DSM-5-TR obsessive-compulsive personality disorder [13].

Generally speaking, what might be called an individual's "window of tolerance" represents the range of emotional intensity that they will be able to cope with and which will be low when the pattern is unstable. It is assumed that exposure to adverse life experiences or childhood maltreatment is associated with classic borderline personality disorder [14]. The important role of the chemical-dynamic processes of so-called "emotional memory" for affectivity has been proposed. This memory is made up of the "cognitive information" consolidated by the neural networks in which it has been encoded [15]. The importance of investigating associations between personality and cognition, and their relationship to stages of personal psychobiological development, has also been highlighted [16].

The hormones testosterone and oxytocin, as well as the neurotransmitter dopamine, may play a role in modulating behavioural changes induced by social contagion [17,18], which is the most ancestral form of empathy. Empathy is assumed to be a fundamental component of socio-emotional experience. Moreover, the role of the neuropeptide oxytocin in personality neurodevelopment, in the pathophysiology of mental and behavioural disorders, as well as in their treatment, is increasingly being studied.

The limbic system is composed of different subsystems including the amygdala, orbitofrontal cortex, insula, hippocampus and cingulate cortex. It has been proposed that the anterior cingulate cortex, but not the amygdala, is involved in anxiety induced by emotional contagion [19]. This may be because this cortex receives information from the orbitofrontal cortex and connects rewards with actions, thus participating in co-existing emotions [20]. From a psychobiological perspective it is well accepted that personality, individual psychological differences and prominent traits and related disorders originate in the brain. Personality neuroscience studies these dissimilarities using appropriate methods.

As examples of certain personality dimensions linked to neurotransmitters we can point out very schematically while retaining a heuristic approach: a) the cognitive-perceptual relationship with dopamine; b) impulsivity-aggressivity with serotonin or 5-hydroxy-tryptamine; c) affective regulation with noradrenaline or norepinephrine; and d) anxiety modulation with adrenaline or epinephrine [21,22]. The endophenotypic perspective of measurable, potentially heritable traits of individual biochemical, neurophysiological, neuroanatomical and cognitive functions provides the scenarios for the underlying candidate genes that contribute to the corresponding cognitive-behavioural and affective dimensions. It also points the way towards a better understanding of the pathophysiological mechanisms involved.

In recent decades, qualified research has revealed that genetic factors shape the propensity towards aggressive, antisocial and violent behaviour. The best documented gene involved in aggression is monoamine oxidase-A (MAOA gene), which encodes the enzyme that is key to the degradation of serotonin and catecholamines [23,24]. There is ample evidence pointing to the fundamental role of serotonin in the emotional modulation of social responses. Increased serotonin binding to the 5-HT2A receptor has been found in the orbitofrontal cortex of pathologically aggressive individuals [25].

Indeed, clinical dimensions potentially associated with endophenotypic markers have been identified for years [26]. Phenotypic overlap of the association between clinically significant behavioural disturbances and elevated polygenic risk scores for mental disorders has been found in children and adolescents [27]. From a neurotransmitter perspective, anxious personality traits are likely to be regulated by serotonin, noradrenaline, γ-aminobutyric acid and the hypothalamic-pituitary-adrenal axis, in addition to how many neurotransmitters play a common regulatory role. Certain personality traits have also been linked to certain ideological attitudes and types or styles of personal values [28].

Furthermore, the endocrine axes regulated by the hypothalamus (hypothalamic pituitary, gonadal,

thyroid and adrenal glands) are altered in the borderline pattern as a prominent personality trait [29]. There are also data on structural and functional changes of the limbic system in these traits and disorders, especially in the amygdala and hippocampus, including the cortical regions of the limbic system [30,31].

Preliminary results suggest that neurodevelopmental pathology associated with the formation of Heschl's gyri (corresponding to the primary auditory area of the cerebral cortex, located within the superior gyrus of the temporal lobe) may be involved in the early neurobiology of borderline pattern as prominent trait or emotional instability personality disorder of ICD-11 and DSM-5-TR respectively, especially with regard to emotional and behavioural controls [32].

A limited but significant number of studies also seem to indicate an association between chromosomal abnormalities and DSM-5-TR cluster A personality disorders (paranoid, schizoid and schizotypal) [33]. Paranoid and schizoid correspond in ICD-11 to the residual category "not otherwise specified", and schizotypal personality disorder is included in the group of schizophrenias and other primary psychotic disorders.

Altered methylation of recombinant deoxyribonucleic acid (DNA) and the proline-rich membrane anchor 1 (PRIMA1) promoter has been reported to be associated with the pathogenesis of borderline personality disorder [34]. Little is known about the influence of epigenetic mechanisms on personality disorders in general, as well as the critical role of gene-environment interactions and epigenetic changes in their genesis.

In particular, impulsive-antisocial traits seem specifically related to the hypersensitivity of the ventral striatum during reward anticipation [35]. Disrupted reward processing is implicated in the aetiology of disruptive behaviour disorders and callous (lack of guilt and empathy) and unemotional (shallow emotionality) personality traits. These traits represent a notable risk factor that helps to explain the heterogeneity in the emergence of early behavioural problems [36].

Accumulating research suggests that child maltreatment, especially physical or sexual abuse and emotional neglect, is associated with epigenetic modification of genes involved in hypothalamic-pituitary-adrenal functioning, which may cause widespread dysregulation of the psychobiological stress response system. On the contrary, through an orexin-2 receptor-dependent mechanism, activating the pyramidal neurons of that cortex recovers, for example, diminished sociability in women who have been stressed by early separation from their mothers [37].

Regulation of glucocorticoid receptor sensitivity by methylation of the FK506 Binding Protein-5 gene (also FKBP5), which encodes a cytosolic immunophilin stress response regulator protein, has been correlated with improved psychopathology and empathy [38]. This gene is linked to the emergence of post-traumatic stress disorder [39]. There is a growing body of evidence for gene-environment interactions such as, in addition to the one mentioned above, variants in CRHR2, the gene encoding the corticotropin-releasing hormone receptor-2 protein [40].

Psychiatry works to better define the aetiology, pathophysiology and treatment of major mental, behavioural and neurodevelopmental disorders. Many of these disorders involve alterations in large-scale distributed brain networks. For the foreseeable, forthcoming personalised or precision psychiatry, biological signatures are sought that underlie these disorders and allow interventions related to the subtypes pinpointed by individual biomarkers or psychopathological neural correlates [41,42]. Immunopsychiatry, as a subfield of psychoneuroimmunology, in convergence with the rest of the psychiatric discipline (which is eminently psychobiological) promises important clinical innovations [22,30,43,44] which, so far, are more suggestive than real. As outlined above, genetics in mental health is still in its early stages, although it holds great promise for personality and other disorders.

II. Aetiological Role of the Endocannabinoid System

We will focus broadly on the endocannabinoid system, as we will do later on the inflammatory system, as it would be too broad to do so on the dopaminergic, glutamatergic and other systems, all of which would require detailed monographic studies. The involvement of the endocannabinoid system is obvious for metabolic and neuromodulatory regulation, the latter essential for understanding the adjustments of many brain functions. Specifically, the endocannabinoid system appears to be central to emotional homeostasis and cognitive function. There is preliminary evidence that lower amygdala levels of the endocannabinoid enzyme amide hydrolase correlate with higher functional fronto-amygdalar coupling. This seems to point to the role of this

enzyme in regulating brain circuits, which in humans underlie the processing of fear and other emotions [45].

To date, there are preliminary findings suggesting that in antisocial and borderline personality disorders there may be alterations of the endocannabinoid system [46] It seems that the fatty acid amide hydrolase of the prefrontal cerebral cortex would be related to the trait of negative affectivity (or neuroticism), encompassed within the symptoms or signs related to personality characteristics. The general population varies markedly in this trait, which is of profound public health importance. Amide hydrolase is an enzyme linked to mood regulation through modulation of the endocannabinoid lipid mediator anandamide or arachidonoylethanolamide [47].

Cannabinoids and cannabidiol (or cannabis preparations enriched with it) appear to have anxiolytic properties [48]. The close involvement of the type-1 cannabinoid receptor in stress regulation has been demonstrated in rodents. This receptor is a major member of the endocannabinoid system and is coupled to the guanine nucleotide-binding protein (or G-protein). Components of the endocannabinoid system may therefore become promising biomarkers in psychiatry [49].

Regarding treatments (which will be specifically addressed in the last section, V), with only indicative results and no official indication, there are current studies suggesting that cannabis-based medicines could be effective in some personality disorders and related traits [50].

III. Aetiological Role of Inflammatory Processes

Closer collaboration between basic and clinical research is needed to apply neuroscientific knowledge to elucidate the mechanisms that produce the symptoms of mental disorders [51]. Because of its potential role in anhedonia, the pathophysiological pathway that receives most attention is the inflammatory pathway with its effects on the brain. Related to anhedonia, it has been observed that conscientious people may be more able to perform tasks that enhance their emotional well-being and be more diligent in engaging in pleasurable activities [52].

The emerging understanding of the mechanisms by which peripheral inflammation can affect brain and behaviour will enable the development of novel pharmacological intervention strategies [53]. Known causality data support the role of inflammatory processes in alterations of neurotransmitters that disrupt specific neurocircuitry and related behaviours. Inflammation-induced neurotransmitter effects manifest as decreased activation of the ventral striatum and reduced functional connectivity in reward circuits involving the striatum with the ventromedial prefrontal cortex [54].

A notable proportion of patients with acute distress from all major diagnostic groups of mental health disorders show evidence of low-grade inflammation, suggesting that inflammation may be relevant to many psychopathologies [55]. Alterations in the immuno-inflammatory and related systems have indeed been implicated in the aetiology, pathophysiology, phenomenology and comorbidity of numerous mental and behavioural disorders. In some recent studies, patients with perinatal mood and anxiety disorders had a unique, distinct signature of plasma proteins that regulate a variety of proinflammatory and neuronal signalling pathways [56].

The degree of awareness or conscientiousness, understood as one of the classic personality dimensions, appears to be inversely related to chronic low-intensity inflammation, as measured by levels of the glycoprotein interleukin-6 [57]. Additionally, the borderline personality pattern appears to be significantly correlated genetically with chronic pain [58]. Overall, however, the data collected so far do not allow us to associate inflammatory imbalance with personality traits [59].

IV. Current Psychological Interventions

This issue would undoubtedly merit a lengthy, dense monographic study. In this overview review we must only state that for personality dysfunctions there is no agreement on the scientific evidence to support psychological treatments as a group. The organisations reporting on this differ in the degree of evidence of therapeutic efficacy and the psychotherapies that would be of choice according to psychopathologies. There is a lack of an international, unified evaluation system in this regard [60]. Thus, we have to assume considerable discrepancies in the published studies, in the different methodologies used and even in the possible disparity in the results obtained after using elaborate statistical tools. Indeed, more than nine out of ten care interventions analysed in numerous recent *Cochrane* reviews are not supported by high-quality evidence [61]. This includes both the use of specific psychotherapies and medication for personality disorders, difficulties and related traits [62,63].

Psychotherapies applied to patients with these

diagnostic categories are usually given for long or very long periods of time, are quite structured [64] and are initiated early [65]. In many people affected by various personality dysfunctions, psychological support, with or without concomitant psychopharmacological treatment, will have to be prolonged, although symptoms usually tend to diminish spontaneously over time. It must be considered that the behavioural pattern characteristic of these disorders and dysfunctions will produce great suffering and heavy burdens for their relatives.

Psychotherapists often overestimate the positive effects of their therapies and overlook their possible ineffectiveness [66,67], as well as the risks they expose patients to by considering them ideally harmless. Little research is available on self-reported outcomes, as well as clinically intermediate and negative outcomes, in addition to economic outcomes [68,69]. For psychotherapy to be more effective and efficient, it is important for mental health professionals to be aware of their frequent emotion of disgust and other negative feelings towards patients with certain personality disorders [70].

In psychotherapies adapted to the so-called borderline pattern, only the concrete outcome of severity has appeared to have a minimal relevant difference to be considered as a significant clinical improvement due to these interventions [71]. First of all, among borderline patients the attitude or predisposition towards psychotherapies is more favourable than for pharmacotherapies [72]. Among professional psychologists, whether or not they are official health specialists, dialectical behavioural therapy and other specific psychotherapies are often preferred as first-line interventions for borderline disorder, while psychoactive medications are often considered not to improve the primary symptoms [73]. Similarly, there is a tendency to professionally prioritise cognitive behavioural therapy for body dysmorphic disorder, although there is also a lack of sufficient evidence to propose that this is the best psychological intervention [74]. Certainly, there are some indications that after receiving dialectical behavioural psychotherapy, borderline patients show decreased frontal limbic activity in the insula and anterior cingulate cortex [75,76].

Another issue is that the previous ICD-10 term emotionally unstable personality disorder (EUPD) seemed inadequate, stigmatising and too simplistic to reflect the nature, severity and psychopathology of the syndrome [77]. It has therefore become a more residual diagnostic category in ICD-11, although it remains in full force in DSM-5-TR. Generally speaking, there has been a growing recognition of the lack of clinical validity of the different types of personality disorders in ICD-10.

There is very little scientific evidence based on randomised controlled trials to inform favourable therapeutic management of acute crises in people diagnosed with borderline personality pattern [78]. In dissocial personality disorder as described in ICD-10, currently belonging to the group of disruptive and dissocial behavioural disorders, no psychotherapeutic intervention has been reported with convincing evidence that it can induce beneficial behavioural changes [79].

A favourable effect of psychotherapies on epigenetic mechanisms associated with the stress response has been cautiously suggested [80]. Also with reservations, results have been reported that point to possible epigenetic biomarkers predictive of the specific effect of behavioural-dialectical psychotherapy in patients with borderline or emotionally unstable personality disorder (ICD-10) [81]. This psychological therapy promotes the acquisition and use of skills to replace maladaptive behaviours. Some preliminary research has recently suggested that it is the reduced responsiveness of the amygdala to a Pavlovian instrumental transfer task that increases the chances of success of this particular psychotherapy [82].

V. Current Drug Treatments

To be exhaustive, this section, like the previous ones, would have to be dealt with in detail and exclusively. In this review, which provides only a broad, up-to-date perspective, we can highlight that the National Institute for Health and Care Excellence (NICE) established years ago that no psychoactive medication had demonstrated efficacy in the specific treatment or management of EUPD (probably the most researched), which has been confirmed in 2022 by a *Cochrane* review [83]. However, especially in hospital settings, pharmacotherapy is still very common in treating these disorders and pathologies.

The recommendations for off-label drugs in these patients vary widely, with a notable lack of agreement among clinical guidelines. There is considerable recognition that they should be seen as complementary to specific psychotherapy [84]. The so-called second- and third-generation atypical antipsychotics and serotonergic antidepressants can be effective for different core symptoms of borderline or emotionally unstable personality disorder [85,86]. Typically, these medications appear to be effective for, a) personality and related trait disorders; b)

impulse control and disruptive disorders; and c) dissocial behavioural disorders (all named according to ICD-11).

The above includes in particular cognitive-perceptual symptoms, mood, emotional lability, anxiety, irritability, impulse dyscontrol and aggression. Atypical antipsychotics with sedative profiles such as quetiapine and mood-stabilising anticonvulsants such as valproate or zonisamide are usually preferred. Opioid antagonists are also being increasingly used [87,88]. On the scientific horizon there is optimism about the anticipated newer, more effective and better tolerated psychopharmacological agents for most mental, behavioural and neurodevelopmental disorders, including personality disorders.

Thus, leaving aside therapeutic dichotomies, for the psychopharmacological treatment of personality disorders, the classical theoretical-practical models of: a) the biological traits or temperaments underlying the disorder; b) the sub-syndromic of variations of other diagnoses that are major; c) the focus on the major diagnosis and its co-morbidity; and d) the focus on the symptoms that are most prominent can be followed. The personality disorders for which psychopharmacotherapy has so far been relatively more effective have been schizotypal, borderline emotional instability and avoidance disorders.

Medication prescription in personality disorders is generally directed at ameliorating affective and impulsive symptom clusters. Relevant psychotropic medicines will be useful, especially, and even if outside the official indications, to reduce discomfort during acute crises [89,90]. In routine clinical care settings, psychotropic drugs are frequently prescribed in patients with these disorders, which very often results in polypharmacy. There is a positive association between the number of drugs used and the effectiveness of the respective hospital care programmes, which seems to contradict the iatrogenic effect of simultaneous and allegedly excessive use of multiple drugs, which is often suggested to be the case for these patients [91].

Specifically in Spain, EU, in the last twenty years the psychopharmacological treatment of outpatients with borderline emotional instability disorder has undergone significant changes, with a decrease in the use of benzodiazepines and an increase in the use of atypical antipsychotics [86,92]. On the other hand, and by way of comparison, there is also very low- or low-quality evidence that new generations of antidepressants can be effective in adults in treating the body-related symptoms that are characteristic of certain disorders. Among the latter are particularly body distress disorder and psychogenic or somatoform pruritus disorder [93], both of which are excluded *per se* as part of mental, behavioural and neurodevelopmental disorders.

To date, studies have shown, with the usual methodological limitations, little or no effect size of pharmacological treatments on brain activity and connectivity in patients with borderline personality disorder during the processing of emotional tasks [94]. As non-drug psychobiological treatments, modulation of brain functions with non-invasive brain stimulation interventions, such as repetitive transcranial magnetic and transcranial direct current, could have a favourable role in the treatment of borderline pattern personality disorders or difficulties [95].

Conclusions

Formerly referred to in ICD-10 as borderline emotionally unstable personality disorder (EUPD), which today remains in cluster B of the DSM-5-TR, is quite prevalent, devastating and heterogeneous, and is a real clinical challenge. Note that it is now typified in ICD-11 as a “borderline pattern”, considered an additional specifier of prominent personality traits or patterns. This characteristic may be either isolated or in addition to a specific personality disorder, which must necessarily be classified as mild, moderate, severe, or of unspecified severity.

There is a problematic lack of valid animal models for the limit pattern. Complex integrative research prototypes are needed to properly understand the psychobiology of various personality traits, patterns and disorders. It may have to be assumed that categorical diagnoses will remain rather inconsistent on this issue in the future. This concerns problems associated with personal interactions, which are part of the factors influencing health status and contact with health services, but are not in themselves considered mental pathologies. In a complementary way, it will be useful to emphasise the dimensions of basic personality processes that are protective for mental health.

It is well established that many brain deficits characteristic of personality disorders and related traits appear not to be influenced by medication. However, atypical antipsychotics and serotonergic antidepressants can be effective for different core symptoms of emotional instability in a borderline personality pattern. In general and for almost all mental disorders, including personality disorders, as well as for prominent problematic personality traits or patterns, combined treatment with psychotropic

drugs and psychotherapy seems to be more effective than exclusive or single psychotherapy or pharmacotherapy.

Environmental, social, physical, affective-emotional and ideational imbalances are the norm in these prominent traits and disorders. It will usually be important that the interaction with the patient by psychiatrists and clinical psychologists is attentive, real and empathic. But, just as for depressive disorders the current main cost-effective therapeutic option is integrated psychopharmacological treatments, there is no quality evidence to be able to claim the same for prominent negative personality traits and disorders.

Although without good quality scientific evidence, there is often a preference among both users and mental health professionals for any psychotherapy as the main maintenance intervention for these dysfunctions, without excluding psychopharmacotherapy as an adjuvant. What is certain is that psychotherapy is still an implicit part of standard psychiatric treatment, which will place a different emphasis on the medication prescribed or the psychological intervention practised. In any case, combined study and action in the corresponding biological, personal, social and psychological domains of each patient by the same or several health specialists, when competent and trained, will be important.

Declarations

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