The delusional dimension of anorexia nervosa: phenomenological, neurobiological and clinical perspectives

Rosa Behar[1], Marcelo Arancibia[2], María Isabel Gaete[3], Hernán Silva[3], Nicolás Meza-Concha[4]

[1] Department of Psychiatry, School of Medicine, Faculty of Medicine, Universidad de Valparaíso, Valparaíso, Chile.
[2] Biomedical Research Centre (CIB), Universidad de Valparaíso, Valparaíso, Chile. Interdisciplinary Centre for Health Studies (CIESAL), Universidad de Valparaíso, Viña del Mar, Chile. Cochrane Centre, Universidad de Valparaíso, Viña del Mar, Chile.
[3] Department of Psychiatry, School of Medicine, Faculty of Medicine, Universidad de Chile, Santiago, Chile.
[4] School of Medicine, Faculty of Medicine, Universidad de Valparaíso, Viña del Mar, Chile.

Abstract

Background: Delusional characteristics have been largely ignored in patients suffering from anorexia nervosa (AN). **Objectives:** To review the literature on delusional features in AN from phenomenological, neurobiological, and clinical viewpoints. **Methods:** Data were obtained through searches of Medline, PubMed, SciELO and Cochrane Library. **Results:** Distorted beliefs in AN may range from an overvalued idea to an overt delusion, involving affective, personality and/or psychotic disorders. Studies confirm alterations in monoaminergic systems. It has also been seen a decreased integration of visual/proprioceptive information, and alterations in neural networks involved in body processing. It is known that body image distortion may present "delusional proportions" as a consequence of great concern about body. Concomitantly, "embodied defence hypothesis" has been proposed. Restrictive AN exhibits higher levels of delusionality, and a particular delusional type of AN has been suggested, associated with a worse long-term outcome. Low doses of atypical antipsychotics are recommended combined with cognitive techniques. **Discussion:** Delusional thinking in AN is likely a dynamic and dimensional phenomenon that can vary, both in nature and/or severity, whereas high insight levels, before and after refeeding, result in positive outcomes. Neurobiological research on this topic must be encouraged, since clinical and phenomenological approaches are comparatively more frequently reported.

Introduction

Body image and weight concern are the most important features of eating disorders (EDs), particularly in anorexia nervosa (AN), but the knowledge of underlying thoughts, attitudes, behaviours and related neural mechanisms is still scarce[1]. Diagnostic criteria of AN highlight strongly held beliefs related to eating, shape and body weight, but they do not specify the range of its intensity, which sometimes is so severe as near of delusionality[2], which can also be viewed dimensionally, ranging from completely delusional, to having overvalued ideas, and to being with no insight at all[3]. The occurrence of a delusional or "near delusional" reality testing has been largely ignored in anorectic patients, and their belief systems have not been systematically explored[2].

According to Bruch[4], the anorectic patient behaviour is addressed to produce and/or maintain a remarkable weight loss associated to a perceptual overestimation of the subject's body weight or shape, as a consequence of body dissatisfaction. Due to this misperception, patients usually deny the seriousness of weight loss. Nowadays, it is well known that restrictive-type anorectics are more susceptible to exhibit psychotic features. Rigidity, high levels of alexithymia and introversion would be eliciting factors acting as pre-psychotic conditions, while concrete, dichotomous and ruminative thinking style, aided by fasting and emaciation, would make them more prone to dissociative experiences and therefore to psychosis. In this line, depersonalisation and derealisation episodes are often found as dissociative warning signals ready to evolve to a psychotic disclosure. Extreme malnutrition, a highly obsessive state, simultaneous mood disorders, electrolytic and/or hormonal misbalances, substance abuse and iatrogenic effects are possible mechanisms leading to psychosis through hyperactivation of the dopaminergic system, whose most prevalent manifestations are delusions with somatic and paranoid contents and hallucinations[5].

The aim of this article is to conduct a comprehensive review of the literature on delusional features displayed by some patients suffering from AN, emphasizing a holistic view of its psychotic features from phenomenological, neurobiological and clinical dimensions.

Methods

A literature search was carried out using the electronic databases Medline/PubMed, SciELO, Cochrane Library and six specialised handbooks, since 1950 up to 2018, in order to perform a comprehensive literature review about the delusional phenomenon in AN. Keywords included anorexia nervosa, eating disorders, delusions, body image, phenomenology and neurobiology. A total of 452 references were found. After excluding duplicates, title and abstract screening and full-text assessment for eligibility, forty five articles were considered the most relevant because of their contribution to the subject, according to the common agreement of the authors. All of them were critically studied. Considering the design of this review, no ethical approval was required due to the nature of this type of research. The whole process of studies eligibility is detailed in Figure 1.

Results

In this review, we included 3 systematic reviews, 19 research articles, 19 review articles and 4 case reports. We also include 6 specialised handbooks.

The delusional spectrum

Distorted beliefs in AN may range from an overvalued idea to an overt delusion, that may imply affective and/or personality components and even a psychotic disorder[6]. In some cases, delusional
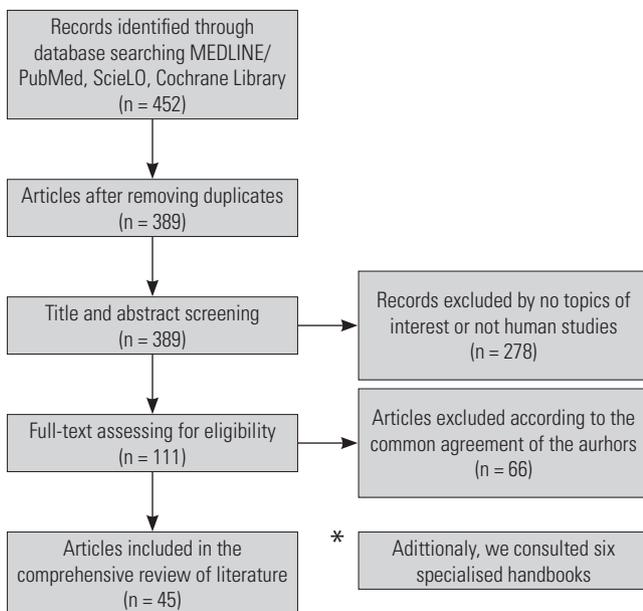
Figure 1. Flowchart of studies included in the review.

conviction occurs with no clear division between delusional and nondelusional thinking. It is often difficult to distinguish between a delusion and an overvalued idea, which is an unreasonable and isolated sustained belief, not as inflexible or as idiosyncratic as a delusion, and more likely to be a passionate religious, fanatical or political conception and typically does not hinder the individual from remaining functional[7]. Jaspers[6] defined a primary delusion as a belief, impossible with regards to its content and contextual implausibility[6], unlikely and often fantastic, with lack of evidence, held with extraordinary deep subjective certainty, incapable of being modified by other experiences, and convincing and/or reasonable counter-arguments. Hypothetically, a disease process in the brain could cause it and therefore it is unsusceptible to psychological inquiry. Nevertheless, the evidence has suggested that sociocultural influences impact the content of delusions rather than on their form, but usually the false belief has a great personal significance and is not always shared by others from a similar socio-cultural-economic group. Secondary delusion can be understandable within the patient's mood circumstances and/or life events, in relation to peer group beliefs or as a long-term development of personality traits, displaying different degrees of insight[8]. It seems to be a key aspect of AN[1], being a dimensional rather than categorical construct, and ranging from a more general insight into having a disorder at all, to a more disorder-specific belief (*e.g.*, body image distortion in AN)[3]. Konstantakopoulos *et al.*[1] have stated that there is a wide range of illness denial rates in EDs, fluctuating from 15% to 80%, probably related to divergent criteria used to categorise patients as delusional or insightful (*e.g.*, clinical judgement, low scores on self-report symptom questionnaires).

Diagnostic criteria

Bruch[4] introduced the concept of "delusional denial of thinness" as a core of the disorder, distinguishing primary AN from an atypical subtype in which patients may not express this "delusion." However, the Fifth Version of the Diagnostical and Statistical Manual of Mental Disorders (DSM-V) does not refer to patients' thoughts about their bodies as delusional, instead using the term "intense" beliefs. This nomenclature reflects that reality testing in anorectics, besides eating-related issues, is generally unimpaired, usually recognising their beliefs as unshared by others[2]. Descriptions of body image disturbances in DSM-IV represent a wide range of intensity of body image beliefs as well[9], whereas the term "intrusive overvalued idea" is

used in the International Classification of Diseases, Tenth Revision[10]. Additionally, DSM-IV-TR criterion C (body image disturbance) for AN is reported as: (I) a "disturbance in the way in which one's body weight or shape is experienced", or (II) "undue influence of body weight and shape", or (III) "denial of the seriousness of the current low body weight"[2].

Body image beliefs in AN according to DSM-IV-TR are "unreasonable and sustained" judgements "maintained with less than delusional intensity"[9]. A specifier meaning delusionality and conveying the broad range of insight from good to absent has already been included in DSM-V for obsessive-compulsive disorder, body dysmorphic disorder, hoarding disorder, and olfactory reference syndrome, but not for AN[1]. Nevertheless, a coherent systematisation of appearance beliefs in AN has been proposed including good or poor insight, or with delusional (psychotic) features, but not in the DSM schema[11].

Neurobiological basis of body image distortion

Evidence that directly associates psychotic symptoms in AN with a neurobiological substratum is limited. On one hand, specialised literature has extrapolated findings from psychotic disorders to AN. On the other, some authors have guessed about possible correlations among other features of the disorder more biologically based with delusional spectrum in AN (*e.g.*, obsessionality, perfectionism). A major issue in research on the neurobiological basis of AN, is the fact that anorectics are food deprived[12]. Hence, neural alterations might correspond to consequences rather than causes in these patients[13] (*e.g.*, appetitive conditioning using food as a reward increases dopamine levels in mesolimbic neurons[14], and also hunger related hormones act on them[15]. Some authors have consistently stated that individuals with AN have anxious, inhibited, and inflexible premorbid traits, probably due to altered monoamine neuronal modulation, or an impaired dorsal caudate function, that predates the onset of the disorder. Some other factors might exacerbate these vulnerabilities during the adolescence. Puberty-related female gonadal steroids might provoke dysregulation of serotoninergic and dopaminergic systems and brain changes might further promote these processes. Increased activity of orbital and dorsolateral prefrontal cortex regions during and after puberty might contribute to excessive worry, perfectionism, and strategising in AN patients. Finally, stress and/or sociocultural pressures may contribute by increasing anxious and obsessional temperament[16].

Neurotransmitter systems

Research has mainly focused on the serotoninergic system. It is involved in regulatory feeding behaviour such as playing an inhibitory role in eating patterns, regulating meal size and controlling eating rate. Moreover, the system is the cornerstone in several symptoms and behaviours associated with AN, such as obsessional features, anxiety, impulse control, inhibition, attention and mood. Some serotonin deficits reported in AN are: increased 1A and a decreased 2A serotonin receptors binding in ill and weight-recovered patients. Thus, after recovery from AN[17], studies have confirmed persistence in altered serotonin activity, anxiety symptoms, perfectionism and obsessive behaviour as well. Therefore, serotonin dysfunction may be a characteristic independent of the illness state. Some investigations suggest perfectionism as a possible endophenotype in EDs, associated with polymorphisms in the dopamine D4 receptor[18].

As stated, dopamine also plays an important role in behaviours, which are found to be disturbed in AN, such as eating patterns, motivation, reinforcement and reward. Deficits in dopamine function have been reported in AN, specifically, reduced levels of cerebrospinal fluid homovanilic acid and altered dopamine receptor functioning[19]. While there are fewer studies, alterations of dopamine in recovered individuals may persist[20], as has been found in serotoninergic system. It has been suggested that reduced food intake increases the

risk for AN by engaging mesolimbic dopamine neurons (involved in psychotic manifestations), causing an initial reward to dieting. Additionally, functional magnetic resonance studies have confirmed that dopamine neurons are activated in AN, but it is unclear whether this response is due to the disorder or to its resulting nutritional deficit[13]. As with psychotic disorders, the amphetamine model of psychosis would explain some symptoms characteristically observed in AN, namely, image distortions and stereotyped behaviors[21].

Genetic studies

Linkage studies have detected an association between *loci* in chromosome 1p34.2 in the restrictive subtype of AN and serotonin 1D and opioid delta 1 receptor *locus*. Other studies have determined signals in chromosome 2 for obsessionality, chromosome 13 for drive for thinness and chromosome 1 for the two traits[22]. These findings have not been followed up through rigorous replication studies, but they encompass relevant aspects of AN associated to these core features of EDs, involving distorted attitudes, behaviours and thoughts.

Published AN genome-wide association studies have yet to show significant findings for AN. A lack of sufficient sample size likely explains the disappointment of the AN genome-wide research efforts to date, as typically thousands of subjects are needed to yield significant *loci*. Another reason is the complexity of gene interaction and regulatory effects to other genomic factors. For instance, perturbation of the serotonergic and noradrenergic system is likely a result of complex interactions and compensatory mechanisms with other neurobiological systems and brain regions[23].

Structural and functional studies

Relatively little is known about the mechanisms underlying body image distortion in AN. A potential deficit in visual and tactile integration has been proposed. A study compared the size-weight illusion in individuals with AN and healthy controls. The size-weight illusion arises when two objects of equal weight but different sizes are held. Controls should experience a strong and robust illusion that the smaller object feels much heavier than the larger one because of an implicit assumption that weight scales with size. Individuals with AN exhibited a markedly reduced size-weight illusion relative to controls, even though their ability to discriminate weight was unaffected. These findings suggest a decreased integration of visual and proprioceptive information in AN, which may reflect a dysfunction in interactions between sensory integration in the inferior parietal lobule (concerned with body image), affective processing of bodily states in the insula and regulation of appetite through the hypothalamus[24]. Also, there is evidence for different networks in AN and healthy controls during visual processing of human bodies. It has been reported that a left sided effective connectivity in the occipital cortex of women with AN is negatively correlated with body size misjudgment. The altered networks for body processing would explain body size misjudgment, a key feature in AN[25]. Another study has investigated self-other body-shape comparison, concluding that AN patients displayed greater anxiety to the self-other body-shape comparison, and they were less satisfied with their current body shape. The self-other body-shape comparison induced more activation of the right sensorimotor brain regions (i.e., insula, premotor cortex) and less function of the rostral anterior cingulate cortex. These findings may be critical for altered interoceptive awareness to body self-comparison and/or for impaired implicit motivation to thin-idealised body images in AN patients[26]. Concerning body image distortion in AN, it has been considered such feature as a multidimensional construct. Analyses of their components, revealed that: 1) the perceptive component is mainly related to alterations of the precuneus and the inferior parietal lobes; 2) the affective component is mainly linked to changes of prefrontal cortex, insula and amygdala; 3) the cognitive component has been weakly explored and requires further investigations[27].

Phenomenology of delusions in anorexia nervosa

Phenomenological research has distinguished four main domains to describe belief intensity: action (linked with some behaviours), distress (related to negative affects such as depression or anxiety), preoccupation (how often the belief is focused on) and conviction (how strongly the belief is held). The latter, has probably received the greater attention due to its usefulness in distinguishing an overvalued idea from a delusion[28]. For instance, delusions in AN would be dissimilar to those in schizophrenia because AN patients would show higher levels of preoccupation and distress explained by a greater degree of insight[29].

Body image disturbance: delusion or defence mechanism?

Schilder[30] defined the concept of body image as "the picture of our own body, which we form in our own mind, that is to say, the way in which the body appears to ourselves." This first definition has supported the idea that body image could be more or less "objectively" captured. In agreement with Gaete and Fuchs[31], it is questionable that an objective body image is attainable at all, both for patients and normal individuals[32]. Furthermore, Schilder[30] described the "libidinous aspect" of body image, meaning with it that every mental image or perception has emotional and affective aspects, deeply related to the own personality. Nevertheless, mostly the representational facet of the body image concept has prevailed through the time. Body image distortion in AN was characterised by Bruch[4] as a feature with delusional proportions. Meanwhile, Roa[33] conceptualised AN symptoms as the expression of a "delusion of beauty." Both authors, in different ways, highlight the denial implied in the emaciation state and the strong force to uphold both their nutritional state and their eating behaviours as normal. Bruch[4] described the denial of starvation as the expression of the fear of fatness or of being haunted by the fear of ugliness. In fact, it is this dramatic denial of the illness as a delusional self-concept, including body image, what justifies for Bruch[4] to consider AN as a specific mental disorder[4]. She also distinguished between the external (mirror) and internal image of the body; the former appears to be distorted in anorectic patients and the latter is related to affective, sensorimotor and emotional aspects, which in turn are always participating in the external body image. Roa explains the body image disturbance driven by the "delusion of beauty" and the searching for perfection. What Bruch and Roa[33] describe differently as a fear of ugliness or "delusion of beauty," entrap patients into an extreme attentional concern with their bodies. The puzzling matter is that they do not seem interested in following fashion trends, but to hide their bodies under floating and big clothes, frequently of dark colours, with rather a monotonous and stereotyped style, and without any joyful attention to their images. Both perspectives are thought as two sides of the same coin: patients seeking for perfection as the result of their extreme insecurity and lack of self-esteem that leads to an almost persistent paranoid attitude on avoiding any "mistake," both in daily life and towards their bodies. Thus, the "delusional proportion" of Bruch[4], is understood from the phenomenological perspective as a consequence of great concern: the more fear of fatness, the more delusional degree of body image disturbance. In some cases, it seems that patients are protecting themselves from becoming fat by the irrational idea of being already fat. From this point of view, a delusional thinking is characterised by the lack of logical reasoning and the resistant nature to any argumentation or experience. However, there is no consensus yet whether the beliefs about body image in anorectic patients can be classified as overvalued or obsessive ideas, delusions or a combination of them[34]. Furthermore, Mountjoy *et al.*[34] explore the nature and the intensity of body image beliefs in anorexia nervosa, especially with regard to the so-called overvalued ideas. They propose a *continuum* through normal and delusional thinking, where AN can be placed in between. However, only a minority of anorectic patients have been categorised within compatible ranges for both pathological kinds of thinking. With regard to delusional beliefs, Roa[33] highlights the desperate searching

for individuation, which can be linked with the conceptualisation of a defence mechanism against the threat to self-organisation. This is in agreement with the "embodied defence hypothesis"[31], which refers to the body experience as an object expressed on the extreme attentional concern, leading patients to no longer perceive their bodies as the way for experiencing the world, but becoming the primary attentional focus. This viewpoint states that the emotional bodily experiences represent highly threatening experiences that patients are unable to cope with, due to their vulnerable self-organisation. Therefore, they recover the sense of security and control by retreating their bodies as objects, relieving them of coping with daily life affective and emotional stimuli. Consequently, they feel protected but isolated, replacing their self-others relationship by a self-body as "the other" relationship. So, delusional ideas about their bodies are part of the extreme attentional focus on them as "objects," which necessarily leads to a lack of perspective and to a loss of the emotional bodily resonance[31], a core feature of the subjective emotional bodily experience. The weaknesses that impede patients to cope with affective and emotional stimuli would favour a vicious circle, in which objectification of the body leaves patients without the basic resource to accomplish adaptive responses to daily life events as "somatic markers" of emotions[35]. Thus, they are unable to take emotionally based decisions for adjustment responses, reinforcing the suppression of the bodily resonance of emotions through the body objectification, which at the same time strengthen the deficits of self-organisation as well. It has been suggested that these vulnerabilities are triggered by early attachment experiences, if the child did not experience sensitive responses to its bodily expressions[36-38]. Hence, if there is a conflict with someone affectively relevant, it becomes an extremely unbearable situation for anorectics, suppressing the bodily resonance as a consequence of such an uncomfortable emotion, representing an efficient defence mechanism. This suppression of bodily resonance implies that patients might experience feelings like "nothing is happening with my life, everything is fine; but my only problem is with my body," as object.

Clinical features

There is consistent evidence that restrictive AN exhibits higher levels of delusionality (a less impulsive and less insightful pole), lending support to their significance and major role in the development and the maintenance of EDs, particularly during the early phase of the illness, reflecting a link between cognitive and emotional aspects of body image disturbances in AN[1]. However, ED patients who develop a stable psychosis would preserve psychopathological phenomena of the feeding syndrome[39]. Additionally, emaciation is associated both with serious medical morbidity and firmly held beliefs, which are impressively stereotyped, reporting the most of anorectics as a dominant belief that if they eat, they get fat or change shape in some way immediately after eating[2]. Poor illness insight is a predictor of worse long-term outcome, and vice-versa, decreasing in delusional beliefs or enhancement in disorder awareness constitute an important marker of patients' improvement[1].

A particular phenomenon known as "anorectic voice" has been described. It is often experienced as a second or third person commentary about actions related to eating, weight and shape. It is possible to distinguish it from auditory hallucinations because they are recognised as internally generated. At the early disease stages they would be supportive, but then they become controller and hostile. The importance of the understanding of this phenomenon has been pointed out in cognitive models of psychosis, which stress the relevance of the power and nature of the voice[40].

Differential diagnosis

Psychotic disorders

A close relationship between a previous psychotic structure and AN symptomatology has been observed, with weight loss contributing to exacerbate both delusions and hallucinations[39]. Psychotic episodes are found in 10% to 15% in ED patients. Most of them are transient, although 1% to 3% will obtain a schizophrenia diagnosis[5].

Classification of anorectic patients according to the degree of delusionality of body image beliefs could facilitate further research on the role of insight deficits. In this sense, Konstantakopoulos et al.[1] have recognised a psychotic form of AN with poor insight correlated with restrictive eating and early beginning of the symptomatology.

Schizophrenia

The prevalence of diagnosable schizophrenia in patients with EDs is estimated as below 10%. Males would have a higher risk than females, particularly for the hebephrenic type[41]. Those who suffer from restrictive AN share neurocognitive deficits with schizophrenics: diminishing in shifting attention, overestimation of detail and reduced central coherence (i.e., a difficulty to understand the general context)[5]. Although they could display marked body image disturbances, their content does not seem to be specifically related to weight and appearance[4]. Thus, the diagnosis of schizophrenia should preferably be made and is only valid after re-nutrition has been achieved[39]. It is important to take into account the possibility of developing ED behaviours induced by antipsychotic medications as well[42].

Affective and schizoaffective disorders

Psychotic symptoms in EDs also co-occur in patients with co-morbid schizoaffective and/or bipolar disorder, although the latter display greater levels of comorbidity for full and partial EDs. Indeed, eating disturbances in childhood often precede affective psychosis in adulthood, while patients who develop psychotic symptoms after the onset of AN may show schizothymic personality traits[43]. On the other hand, anorectics seem to show higher preoccupation and distress levels for their beliefs in comparison to patients with schizophrenia/schizoaffective disorder[34].

Furthermore, Cotard syndrome comprises a delusion in which the subject believes he is dead or does not exist; a denial of self-existence within a severe melancholic depression. It may also be observed in schizophrenia or bipolar disorder, which could be related to temporo-parietal lesions of the non-dominant brain hemisphere[44].

Obsessive-compulsive disorder

Perfectionism has been considered a transdiagnostic process within the obsessive-compulsive spectrum and AN[45]. Higher levels of it have been correlated with poorer degrees of insight and delusional symptomatology. Obsessive-compulsive patients feel compelled to carry out rituals to compulsively relieve the associated upheaval as a consequence of their intrusive thoughts associated to several contents (e.g., fears of contamination, a need for symmetry or orderliness, fears of harming others by mistake, cleaning, checking, thinking "neutralising" thoughts, etc.) with an understandable and coherent link between both, in contrast to AN where perfectionism, obsessive thoughts and compulsive rituals are focused mainly on body image, weight gaining, some kind of foods and feeding, versus schizophreniform psychosis, where a primary core delusional belief emerges characterised by an incoherent behaviour with no related rituals[1].

Body dysmorphic disorder

Delusionality of appearance beliefs is present in individuals with AN, but is less pronounced than in body dysmorphic disorder. In AN, delusionality is related to shape concerns and drive for thinness, whereas delusionality in dysmorphophobia would be associated to the severity of symptoms[3,46]. In this line, Grant et al. found that 39%

of anorectic patients exhibited a comorbid body dysmorphic disorder, displaying significantly lower global functioning and higher levels of delusionality[47].

Risk factors

Overvalued ideas appear to be frequent in both anorexia and bulimia nervosa. Nevertheless, Konstantakopoulos *et al.*[1] observed that only anorectics (28.8%) had delusional body image beliefs associated with restrictive behaviours, early onset of the condition and body dissatisfaction, but these were not related to the duration of illness (insight would remain stable through time)[1]. Neither age nor educational level were found to affect the degree of delusionality, which would constitute a independent phenomenon of weight loss or eating behaviour, a different psychopathological component of AN, perhaps contributing to therapeutic resistance and illness chronicity[1]. These findings point out that a delusional type of AN represent the end of a *continuum* in patients with EDs[1].

Treatment

Irrational eating-related thinking may contribute to avoidance of therapy, difficulties to be engaged in treatment and high rates of dropout and relapse[1]. Mental rigidity, body distortion and magical thinking play an essential role in resistance to therapies, in particular as seen in chronic patients with complex developmental histories[39,42].

Delusional thinking in AN, mainly related to body image distortion and drive for thinness to which pursues changes in beliefs and/or cognitive patterns, seems to be underlying the emphasis that cognitive techniques have received along the development of diverse EDs treatments[31].

Pharmacotherapy

An important aspect to consider is that a group of patients exhibit a good symptomatic response with antipsychotics. This would support that a subgroup in AN requires distinct pharmacological treatment, with special attention to structural deficits for long-term maintenance.

First generation antipsychotics have been considered, but given their side effects, poorer efficacy in changing eating behaviour and body image distortion, their usage is not recommended. Low doses of haloperidol have been found to be effective in the AN restricting subtype[48]. On the other hand, atypical antipsychotics can diminish anxiety, agitation, aggressiveness and obsessive thoughts linked to food control and body image distortion[49], by modulating both serotoninergic and dopaminergic systems[39]. Administration at the minimal effective dosage has been suggested[5]. Some agents like olanzapine and quetiapine can be a desirable choice due to their orexigen and sedative effects[5], in decreasing inner tensions and phobia regarding food intake, related to paranoid ideation on body image and/or weight gain. Particularly, olanzapine, recommended from 2.5 to 15 mg daily[50], might be considered in patients with long-term history of AN, several hospitalisations, poor insight, rejection of therapy, delusional thinking and risk of dropout with life-threatening consequences, by improving the compliance to treatment[39] with refeeding and weight gain as well as postdischarge weight maintenance. In Table 1 the main findings of the review are summarised.

Discussion

Dimensionally, delusional spectrum in AN goes from an overvalued idea to a categorical psychotic picture, although in many cases, clinical differences between nondelusional and delusional states are not so clear. Consequently, evidence shows that illness denial is so variable, depending on diverse degrees of individual self-awareness. Certainly, emaciation and somatic disturbances due to malnourishment

Table 1. Main findings summary

Perspective	Findings
Clinical perspective	– Restrictive AN exhibits higher levels of delusionality. – Alexithymia and introversion would facilitate pre-psychotic conditions. – Concrete, dichotomous and ruminative thinking style would foster dissociative experiences and therefore psychotic manifestations. – Poor illness insight is a predictor of worse long-term outcome. – The "anorectic voice" has been described as a second/third person commentary about actions related to eating behaviours, recognised as internally generated. – The presence of overvalued ideas has been proposed as a risk factor in delusional phenomena. – The main differential diagnoses in delusional AN are psychotic disorders, schizophrenia, affective and schizoaffective disorders, obsessive-compulsive disorder and body dysmorphic disorder. – The treatment of delusional aspects in AN considers cognitive techniques, pharmacotherapy (haloperidol, quetiapine and particularly olanzapine), among others.
Neurobiological perspective	– Altered monoamine neuronal modulation (*e.g.*, altered metabolism of serotonin and dopamine receptors). – Impaired dorsal caudate, amygdala and precuneus functioning. – Increased orbital and dorsolateral prefrontal cortex activity. – Dysfunction in sensory integration of inferior parietal lobule, insula and hypothalamus. – Decreased cingulate cortex activity.
Phenomenological perspective	– In AN, body image distortion may present "delusional proportions" as a consequence of great concern about body. – The phenomenon of "denial" is deeply implied in emaciation state. – "Delusion of beauty": extreme attentional concern to the body, seeking for perfection. – Searching for individuation: conceptualisation of a defence mechanism against the threat to self-organisation. – "Embodied defence hypothesis": emotional bodily experiences represent threatening experiences that patients are unable to cope with; therefore, the sense of security and control is recovered by retreating the body as an object.

contribute to a lack of reality judgement. Additionally, some pre-morbid psychological traits have been identified (*e.g.*, dichotomous and ruminative thinking, alexithymia, rigidity and perfectionism), which interact with the Western sociocultural, historical and economic context, that emphasises the drive for thinness to achieve an ideal feminine slim body shape. Nonetheless, some authors state that sometimes delusionality is independent of weight loss or eating pathology and therefore a manifestation of a different AN subtype, related to treatment resistance and a chronic illness course[1]. In this sense, classification of AN patients based on the level of delusionality beliefs could facilitate further research on the role of insight deficits in these disorders[1], which was not included in the DSM-V as a specifier[9]. Hence, a systematic assessment of the irrational thinking in AN would contribute by identifying cognitive processes underlying the clinical picture[2]. Besides, some researchers have verified that if self-perception is altered, it is often mildly, unsatisfactory to explain the tremendous delusional denial of reality seen in severe cases of AN. Thus, Gardner and Bokenkamp propose that perceptual distortion is

due mainly to comorbid affective disorders usually observed during AN evolution[51]. Conversely, recent studies[13] have documented that alterations in dopamine system found in AN patients, are more likely to be physiological responses to starvation than signs of mental pathology. Nevertheless, as stated above, neurobiological aspects of psychotic features in AN have been scarcely explored, and due to the permanent malnourishment state of these patients, it is difficult to distinguish causal and consequent mechanisms,

McElroy *et al.*[45] stated that, although EDs show differences from obsessive-compulsive disorder they also display some similarities (obsessive thinking and/or compulsive behaviours, course, comorbidity, family patterns, biological abnormalities and treatment responses) which allow to include them in the obsessive-compulsive spectrum among other disorders. Moreover, they hypothesised that some differences may be partly explained by variation along a dimension of compulsivity *versus* impulsivity, and, because most of these conditions appear to be related to mood disorders, the obsessive-compulsive spectrum may belong to the larger family of affective spectrum.

Delusional thinking in AN, seems undeniable, but it is likely a dynamic and dimensional phenomenon that can vary both in nature and in severity. This means that not all patients show a delusional thinking, and in some good insight levels are preserved. However, the presence of delusional thinking probably means an outcome index that must be taken into account from a therapeutic point of view. Thus, this feature can be understood as a part of a broader phenomenon described by Gaete and Fuchs[31] and named "disturbance of the emotional bodily experience," where the embodied defence mechanism is involved. Hence, cognitive or perceptual approaching techniques for body image disturbance may disregard the severe difficulties of patients related to the bodily resonance of emotions, that permit adaptive responses to daily life, which can explain the high risk of relapse and complexity in modifying cognitive patterns and "perceptual distortions." It seems relevant to develop new psychotherapeutic techniques, which offer patients a safe way of recovering their living bodies as subjects.

Delusional features in AN have been reported from multidimensional perspectives along the history of psychiatry (i.e., phenomenological, neurobiological, psychodynamic clinical). However, current versions of mental diseases guidelines often do not include them. It is therefore of utmost importance to encourage neurobiological research on this topic, since clinical and phenomenological approaches are more developed and have contributed to a comprehensive understanding of the delusional dimension of AN. Insight assessment before and after refeeding, as a marker of the nature and severity of the disorder, should be systematically performed in order to provide an optimal treatment option.

References

1. Konstantakopoulos G, Varsou E, Dikeos D, Ioannidi N, Gonidakis F, Papadimitriou G, et al. Delusionality of body image beliefs in eating disorders. Psychiatry Res. 2012;200(2-3):482-8.
2. Steinglass JE, Eisen JL, Attia E, Mayer L, Walsh BT. Is anorexia nervosa a delusional disorder? An assessment of eating beliefs in anorexia nervosa. J Psychiatr Pract. 2007;13(2):65-71.
3. Hartmann AS, Thomas JJ, Wilson AC, Wilhelm S. Insight impairment in body image disorders: delusionality and overvalued ideas in anorexia nervosa versus body dysmorphic disorder. Psychiatry Res. 2013;210(3):1129-35.
4. Bruch H. Perceptual and conceptual disturbances in anorexia nervosa. Psychosom Med. 1962;24:187-94.
5. Sarró S. Transient psychosis in anorexia nervosa: review and case report. Eat Weight Disord. 2009;14(2-3):e139-43.
6. Spitzer M. On defining delusions. Compr Psychiatry. 1990;31(5):377-97.
7. Veale D. Over-valued ideas: a conceptual analysis. Behav Res Ther. 2002;40(4):383-400.
8. Kendler KS, Glazer WM, Morgenstern H. Dimensions of delusional experience. Am J Psychiatry. 1983;140(4):466-9.
9. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013.
10. World Health Organization. The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines. Geneva: WHO; 1992.
11. van der Zwaard R, de Leeuw AS, van Dael F, Knook LM. [...with psychotic features? An investigation into the delusional nature of convictions in patients with non-psychotic disorders]. Tijdschr Psychiatr. 2006;48(6):461-6.
12. Bang L, Treasure J, Rø Ø, Joos A. Advancing our understanding of the neurobiology of anorexia nervosa: translation into treatment. J Eat Disord. 2017;5:38.
13. Södersten P, Bergh C, Leon M, Zandian M. Dopamine and anorexia nervosa. Neurosci Biobehav Rev. 2016;60:26-30.
14. Sunsay C, Rebec GV. Extinction and reinstatement of phasic dopamine signals in the nucleus accumbens core during Pavlovian conditioning. Behav Neurosci. 2014;128(5):579-87.
15. Cone JJ, Roitman JD, Roitman MF. Ghrelin regulates phasic dopamine and nucleus accumbens signaling evoked by food-predictive stimuli. J Neurochem. 2015;133(6):844-56.
16. Kaye WH, Wierenga CE, Bailer UF, Simmons AN, Bischoff-Grethe A. Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa. Trends Neurosci. 2013;36(2):110-20.
17. Rikani AA, Choudhry Z, Choudhry AM, Ikram H, Asghar MW, Kajal D, et al. A critique of the literature on etiology of eating disorders. Ann Neurosci. 2013;20(4):157-61.
18. Bachner-Melman R, Lerer E, Zohar AH, Kremer I, Elizur Y, Nemanov L, et al. Anorexia nervosa, perfectionism, and dopamine D4 receptor (DRD4). Am J Med Genet B Neuropsychiatr Genet. 2007;144B(6):748-56.
19. Phillipou A, Rossell SL, Castle DJ. The neurobiology of anorexia nervosa: a systematic review. Aust N Z J Psychiatry. 2014;48(2):128-52.
20. Barbarich NC, Kaye WH, Jimerson D. Neurotransmitter and imaging studies in anorexia nervosa: new targets for treatment. Curr Drug Targets CNS Neurol Disord. 2003;2(1):61-72.
21. Kontis D, Theochari E. Dopamine in anorexia nervosa: a systematic review. Behav Pharmacol. 2012;23(5-6):496-515.
22. Yilmaz Z, Hardaway JA1, Bulik CM. Genetics and Epigenetics of Eating Disorders. Adv Genomics Genet. 2015;5:131-50.
23. Shih PA, Woodside DB. Contemporary views on the genetics of anorexia nervosa. Eur Neuropsychopharmacol. 2016;26(4):663-73.
24. Case LK, Wilson RC, Ramachandran VS. Diminished size-weight illusion in anorexia nervosa: evidence for visuo-proprioceptive integration deficit. Exp Brain Res. 2012;217(1):79-87.
25. Suchan B, Bauser DS, Busch M, Schulte D, Grönemeyer D, Herpertz S, et al. Reduced connectivity between the left fusiform body area and the extrastriate body area in anorexia nervosa is associated with body image distortion. Behav Brain Res. 2013;241:80-5.
26. Friederich HC, Brooks S, Uher R, Campbell IC, Giampietro V, Brammer M, et al. Neural correlates of body dissatisfaction in anorexia nervosa. Neuropsychologia. 2010;48(10):2878-85.
27. Gaudio S, Quattrocchi CC. Neural basis of a multidimensional model of body image distortion in anorexia nervosa. Neurosci Biobehav Rev. 2012;36(8):1839-47.
28. Combs DR, Adams SD, Michael CO, Penn DL, Basso MR, Gouvier WD. The conviction of delusional beliefs scale: reliability and validity. Schizophr Res. 2006;86(1-3):80-8.
29. Phillipou A, Mountjoy RL, Rossell SL. Overvalued ideas or delusions in anorexia nervosa? Aust N Z J Psychiatry. 2017;51(6):563-4.
30. Schilder P. The image and appearance of the human body. New York: International Universities Press; 1950.
31. Gaete MI, Fuchs T. From body image to emotional bodily experience in eating disorders. J Phenomenol Psychol. 2016;47(1):17-40.
32. Jansen A, Smeets T, Martijn C, Nederkoorn C. I see what you see: the lack of a self-serving body-image bias in eating disorders. Br J Clin Psychol. 2006;45(Pt 1):123-35.
33. Roa A. Anorexia Nerviosa o Delirio de Belleza. Rev Psiquiatr Clín. 1978;15:27-54.
34. Mountjoy RL, Farhall JF, Rossell SL. A phenomenological investigation of overvalued ideas and delusions in clinical and subclinical anorexia nervosa. Psychiatry Res. 2014;220(1-2):507-12.
35. Damasio AR. Descartes' error: emotion, reason, and the human brain. London: Picador; 1995. 463p.

36. Atwood GE, Stolorow RD. Structures of subjectivity: explorations in psychoanalytic phenomenology and contextualism. 2nd ed. New York: Routledge; 2014. 174p.

37. Bruch H. Eating disorders: obesity, anorexia nervosa, and the person within. New York: Basic Books; 1973.

38. Lichtenberg JD. Motivational systems and model scenes with special references to bodily experience. Psychoanal Inq. 2001;21(3):430-47.

39. Delsedime N, Nicotra B, Giovannone MC, Marech L, Barosio M, Marzola E, et al. Psychotic symptoms in a woman with severe anorexia nervosa: psychotic symptoms in Anorexia Nervosa. Eat Weight Disord. 2013;18(1):95-8.

40. Pugh M, Waller G. The anorexic voice and severity of eating pathology in anorexia nervosa. Int J Eat Disord. 2016;49(6):622-5.

41. Striegel-Moore RH, Garvin V, Dohm FA, Rosenheck RA. Psychiatric comorbidity of eating disorders in men: a national study of hospitalized veterans. Int J Eat Disord. 1999;25(4):399-404.

42. Bou Khalil R, Hachem D, Richa S. Eating disorders and schizophrenia in male patients: a review. Eat Weight Disord. 2011;16(3):e150-6.

43. Miotto P, Pollini B, Restaneo A, Favaretto G, Sisti D, Rocchi MB, et al. Symptoms of psychosis in anorexia and bulimia nervosa. Psychiatry Res. 2010;175(3):237-43.

44. Gardner-Thorpe C, Pearn J. The Cotard syndrome. Report of two patients: with a review of the extended spectrum of 'délire des négations'. Eur J Neurol. 2004;11(8):563-6.

45. McElroy SL, Phillips KA, Keck PE Jr. Obsessive compulsive spectrum disorder. J Clin Psychiatry. 1994;55 Suppl:33-51.

46. Behar R, Arancibia M, Heitzer C, Meza N. Trastorno dismórfico corporal: aspectos clínicos, dimensiones nosológicas y controversias con la anorexia nerviosa. Rev Med Chil. 2016;144(5):626-33.

47. Grant JE, Kim SW, Eckert ED. Body dysmorphic disorder in patients with anorexia nervosa: prevalence, clinical features, and delusionality of body image. Int J Eat Disord. 2002;32(3):291-300.

48. Cassano GB, Miniati M, Pini S, Rotondo A, Banti S, Borri C, et al. Six-month open trial of haloperidol as an adjunctive treatment for anorexia nervosa: a preliminary report. Int J Eat Disord. 2003;33(2):172-7.

49. McKnight RF, Park RJ. Atypical antipsychotics and anorexia nervosa: a review. Eur Eat Disord Rev. 2010;18(1):10-21.

50. Dunican KC, DelDotto D. The role of olanzapine in the treatment of anorexia nervosa. Ann Pharmacother. 2007;41(1):111-5.

51. Gardner RM, Bokenkamp ED. The role of sensory and nonsensory factors in body size estimations of eating disorder subjects. J Clin Psychol. 1996;52(1):3-15.