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Causal Connections Between Anorexia Nervosa and Delusional Beliefs

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Abstract
Numerous studies of the beliefs of people with anorexia nervosa (AN) suggest that a subset of such individuals may experience delusions. We first describe what makes a belief delusional and conclude that such characteristics can be appropriately applied to some beliefs of people with AN. Next, we outline how delusional beliefs may relate to the broader psychopathological process in AN, including: (1) they may be epiphenomenal; (2) they may be an initial partial cause of AN; (3) they may be caused by aspects of AN; or (4) they may be sustaining causes, possibly involved in reciprocal causal relations with aspects of AN. We argue that there is good reason to believe that delusional beliefs of people with AN are not merely epiphenomenal, but rather that they're causally connected to AN. Because of this, empirical studies can be designed to test for the presence of causal relations. We describe how these studies should be designed. The results of such studies have important implications for understanding the experience of individuals with AN and for the treatment of AN. We outline these implications.

1 Introduction

Anorexia nervosa (AN) is a complex condition, involving dramatic alterations in thought, emotion, behavior, and often appearance and the functioning of various bodily systems (e.g., gastrointestinal, reproductive, metabolic). Beliefs play a crucial role in AN – in how it develops, how it's sustained, and how it's treated and managed. Historically, theories accounting for AN have not focused on delusional beliefs. However, recent research suggests that 10–30% of individuals with AN experience delusions (De Young et al. 2022; Mountjoy et al. 2014; Konstantakopoulos et al. 2012; Phillipou et al. 2017). Given the complexity of AN—half of patients do not

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recover following treatment (Wonderlich et al. 2012), 31.4% of individuals with AN recovered at 9 years and 62.8% at 22 years (Eddy et al. 2017), and relapse following treatment is common (Khalsa et al. 2017)—we think exploring causal connections between delusions and AN is important.

Historically, delusions have been associated with psychotic disorders, like schizophrenia, more than with non-psychotic disorders, like AN. Additionally, the unusual or distorted beliefs associated with AN have sometimes been categorized as overvalued ideas, rather than delusional beliefs (Mountjoy et al. 2014, p. 507; Phillipou et al. 2017, p. 563; Veale 2002). But the idea that delusions are present in AN is not a new one. It can be found as early as the 1960s, when Hilde Bruch argued that disturbances in body image of delusional proportions are found in patients with AN (Bruch 1962). As Behar et al. (2018) summarize, "Bruch 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" (2018, p. 17). More recently, Phillipou et al. (2017) summarize a number of findings linking AN and delusional beliefs. And Behar et al. (2018) offers an extensive literature review of a multitude of work connecting AN with delusion.

Additionally, there is a wide body of research showing that delusions are present in many non-psychotic disorders: they can occur in obsessive compulsive disorder (OCD; Kozak and Foa 1994) and major depressive disorder (Maj 2008). They can also be associated with dementia (Cipriani et al. 2013). As Lancellotta and Bortolotti (2019) point out, the delusions associated with OCD and MDD tend to be about matters that are congruent with the *habits* or *practices* of the individual — e.g. believing "my hands are dirty unless I wash them multiple times" is congruent with compulsive washing of hands — or they are congruent with the *moods* of the individual — e.g. believing "I am the only one to blame for my sister's death" is congruent with feelings of deep guilt.[1] This marks a difference between the delusions associated with schizophrenia vs. MDD or OCD. Delusions in schizophrenia often reveal "new and surprising content," whereas, the content of delusions in non-psychotic disorders involves information "already known and familiar" (2019, p. 11). Given this, it makes sense to think that the delusions associated with AN would be more similar to delusions associated with MDD or OCD than schizophrenia. The content of delusions in AN likely involves information that is already known, familiar, and congruent with the person's practices, moods, and other beliefs.

Given these features, some have advocated for describing such beliefs as overvalued ideas, and numerous attempts have made to distinguish overvalued ideas from delusions (e.g., Veale 2002; Mullen and Linscott 2010), though there is a lack of widespread agreement. We address many of the proposed dimensions on which these two concepts are thought to differ in Section 2.2. Focusing on these dimensions opens up new avenues for research that we would otherwise miss if we focused solely on the concept of overvalued ideas.

After some brief clarifications in Section 2, we analyze influential and widely used sources that characterize delusion for clinical research and practice: the *Diagnostic*

[1] See Lancellotta and Bortolotti (2019), pp. 9–11 for discussion.

and Statistical Manual of Mental Disorders (DSM; APA 2022), the Brown Assessment of Beliefs (BABS; Eisen et al. 1998), and the Structured Clinical Interview for DSM Disorders (SCID; First et al. 2015). In Section 3 we outline how delusional beliefs may relate to the broader psychopathological process in AN. We argue that we have good reason to believe that delusional beliefs are not merely epiphenomenal when it comes to AN, but that they're causally connected in important ways. Understanding this causal role can help us better understand many cases of AN, has implications for research, and may contribute to the development of more effective treatments. We end in Section 4 by discussing these implications for research and treatment.

2 How to Distinguish Delusions

To figure out the relations between AN and delusions, we first need a handle on what delusions are. But delusions are difficult to distinguish. It's probably impossible to give a list of necessary and sufficient conditions for them; the heterogeneous nature of delusions defies precise definition.[2] They're "contextually dependent, multiply determined and multidimensional" (Gilleen and David 2005, p. 5). They can have a wide variety of effects, many of which are disruptive and depend on context. They also share a number of features with non-pathological or non-delusional beliefs (Bartolotti 2022). Despite this, the *DSM* offers characterizations of delusions that are extremely clinically influential. Additionally, the BABS and the SCID are widely used to identify delusions in clinical disorders. We find these characterizations to be such a mixed bag that it's worth sorting through them to identify a core feature.

The first thing to note is that all three of these sources presuppose that delusions are beliefs. We too, conceive of delusions as beliefs. Whenever we use the term 'delusion', we mean 'delusional belief'. This is in keeping with what seems to be the majority of psychologists and philosophers—but there is healthy dissent with this view.[3] For example, delusions do not share some of the primary characteristics of beliefs—delusions are often unresponsive to evidence, and they often fail to guide action.[4] Therefore, one might think they're not beliefs. We're not convinced by this argument.[5] Various candidate alternatives for what a delusion might be—act of imagination, perceptual state, "in-between" mental state, acceptance, etc.—have problems of their own. So, we cast our lot with those who characterize delusions as beliefs. If it turns out that delusions are not beliefs (and instead are imaginings or another mental state), we still have good reasons to think that delusions are causally related to AN.

[2] See Radden (2011), Chap. 2: "Varieties of Clinical Delusion", pp. 17–38, for a nice discussion of delusion features.

[3] For helpful summaries of the debate between doxastic and non-doxastic views of delusion, see Bortolotti (2022), Section 4.2; Bortolotti and Miyazono (2015), pp. 636–638; and Radden (2011), pp. 44–53.

[4] See Bortolotti and Miyazono (2015), pp. 637–639.

[5] The argument rests on thinking of beliefs as essentially reason-responsive and action-guiding, but we think that these are not constitutive features of beliefs, but rather rational standards for belief. Additionally, there is ample reason to think that "regular" beliefs often fail to be reason-responsive and action-guiding. See Bentall (2003) and (2018) for good examples of this, as well as Bortolotti et al. (2017).

2.1 Common Characterizations of Delusion

The *DSM*'s description of delusions has changed across editions. In the *DSM-III* (APA 1980) and *DSM-IV* (APA 1994), a delusion is a "false belief due to incorrect inference about external reality." In the *DSM-5* (APA 2013) and *DSM-5-TR* (APA 2022), delusions are "fixed beliefs that are not amenable to change in light of conflicting evidence." Thus, there has been a shift from a focus on the *veracity* to the *fixedness* of the belief as being central to what makes a belief a delusion. However, the *DSM-5* contains a glossary in the appendix that provides an elaborated definition, stating in part that a delusion is "a false belief based on incorrect inference about external reality that is firmly held despite what almost everyone else believes and despite what constitutes incontrovertible and obvious proof or evidence to the contrary" (p. 819). This description highlights both the veracity and fixedness of the belief.

Complicating matters, there is no glossary in the *DSM-5-TR*, leaving only the in-text description that relies on fixedness. However, it seems clear from the broader context of how delusions are described in psychiatric diagnoses that the content of the belief is considered pertinent in addition to fixedness. For instance, the *DSM* defines types of delusions (e.g., bizarre, grandiose, somatic) that rest on the presumption that the belief is not true, and moreover that it's not based in reality. Similarly, the SCID puts content at the forefront of identifying delusions: delusions are assessed by content-type, and whether a person has a delusion is determined by their responses to content-focused prompts (First et al. 2015). The *DSM-5* glossary provides additional guidance for judging the content of beliefs, stating that "The belief is not ordinarily accepted by other members of the person's culture or subculture (i.e., it is not an article of religious faith). When a false belief involves a value judgment, it is regarded as a delusion only when the judgment is so extreme as to defy credibility" (p. 819).

The *DSM-IV* and *DSM-5* distinguish a delusional belief from an overvalued idea, with the latter being described as "an unreasonable and sustained belief that is maintained with less than delusional intensity (i.e., the person is able to acknowledge the possibility that the belief may not be true)" (APA 2013; p. 826). Providing this distinction introduces another dimension on which potentially delusional beliefs are to be judged – the degree of insight the individual holding the belief has about the belief's relation to the truth. Insight features heavily in the BABS, as well: the BABS introduction states that the entire scale is based on the premise that insight exists on a continuum, ranging from good to poor ("overvalued ideation") to no insight ("delusional thinking").

Lastly, in the section on "Schizophrenia Spectrum and Other Psychotic Disorders," the *DSM-5* states: "The distinction between a delusion and a strongly held idea is sometimes difficult to make and depends in part on the degree of *conviction* with which the belief is held despite clear or reasonable contradictory evidence regarding its veracity" (italics added, APA 2013; p. 87). Thus conviction is another feature put forward to distinguish delusional beliefs, and it is the first assessed dimension on the BABS.

2.2 Distinguishing Delusions

These descriptions about the nature of delusions highlight a number of different features, and it's not clear how to think of each of them. It's also not clear how each feature is related. Below, we sort through these descriptions and argue that there is a central feature at the core of delusional belief, which diagnostic and measurement tools rely on. Identifying this feature helps us better identify delusions in clinical settings.

We see in these descriptions five main features. Delusions are commonly distinguished by (1) content, (2) conviction, (3) insight, (4) irrationality, and (5) fixedness.[6]

2.2.1 Content

Many of the *DSM* characterizations of delusion focus on content. This content is described as false, as unshared by other members of the individual's community, and as defying credibility (*DSM-5*, p. 87 and p. 819). Additionally, the *DSM* and the SCID organize delusions by content (bizarre, persecutory, etc.). This focus might lead us to think that a particular kind of content is crucial to the identification of delusions.

However, content alone is not a good way to distinguish delusions. It can be helpful as a clinical marker insofar as many delusions are wildly implausible or widely unshared. But it's certainly not sufficient, given that there are many false beliefs, implausible beliefs, and unshared beliefs that are not delusional. Content is also not necessary for identifying delusions, given that there are many delusions that *are* plausible (e.g., "someone at work is out to get me", as might be seen in a persecutory delusion). Moreover, a person who persistently believes that she has been abducted by aliens and a person who persistently believes that things will never change both persistently hold a belief, but alien abduction defies credibility while the other belief does not. In summary, while diagnosing delusions on the basis of content can be helpful for flagging certain beliefs as possibly delusional, it only takes us so far, and focusing on content might lead us to miss certain beliefs that are delusional. This means that the content of delusions isn't at the core of what makes a belief delusional.

2.2.2 Conviction

A second way to distinguish delusions is by the degree of *conviction* with which they are held. Although the *DSM* does not elaborate on the meaning of 'conviction', the BABS does. The questions for assessing conviction involve asking the patient how convinced they are of their belief and how certain they are. So, one way to understand conviction is by the degree to which a person

[6] These aren't the only ways to distinguish delusions. We might note their negative practical impact (impairments to everyday functioning, psychological harm, compromised social relationships, etc.) or their cause (dysfunctional mental states, brain damage, etc.). But delusions can have differing practical effects, both positive and negative, and it's difficult to determine their cause. So, we focus on content, fixity, rationality, conviction, and insight, all of which are featured in the DSM and/or BABS.

is convinced that their belief is true, or the confidence they have in the truth of their belief. If a person is completely convinced of their belief, as opposed to harboring some doubt, then it's more likely that the belief is delusional. For example, consider two individuals presenting with the same (potentially delusional) somatic belief that they cannot digest carbohydrates. Person A says that they are absolutely convinced of this, believing it is true with 100% certainty, while person B says they are quite convinced but rate their certainty at 80%. If we were to rely on conviction to gauge delusionality, person A would be more likely to be holding a delusion than person B. At first glance, this seems to make sense. However, one clear problem with relying solely on conviction to distinguish delusions is brought into relief when we consider beliefs based on math. I believe that $2+2=4$. I believe this with 100% conviction, and I am not particularly open to considering that $2+2=5$, for instance. By solely relying upon my conviction, one might conclude that my belief that $2+2=4$ is delusional, but this is an absurd conclusion that will result in useless classifications of delusions. Similarly, an individual with AN may believe with 100% conviction that eating even small amounts of food will result in nausea and pain, but this may be true if they are experiencing gastroparesis (a condition characterized by substantial delay in the emptying of stomach contents into the duodenum; Mascolo et al. 2017), So, although it might be true that many delusions are held with great conviction, this feature is not at the core of delusion.

2.2.3 Insight

A third approach to distinguish delusions is by how much *insight* the individual holding the belief has regarding the origins of their belief. This is a specific view of insight that is narrower than its more widespread use that often overlaps with other dimensions covered here (e.g., see the use of the insight specifier for obsessive-compulsive disorder, which appears to rely on conviction; APA 2013, p. 237). Here, we use the BABS definition. For example, if one holds the grandiose belief that they are Christ reborn, they may have a variety of ideas about why they think this. If they acknowledge that the reason they hold the belief is a result of their psychiatric condition (e.g., "I believe I am Christ reborn, because I'm experiencing a psychotic disorder."), that would demonstrate insight. Lacking the ability to attribute a delusion to such a cause would indicate a lack of insight. A problem with relying solely on insight is that one could conceivably hold a belief that is demonstrably false with full conviction - it could even be bizarre - but provided they acknowledge their belief results from their psychiatric condition, it would not be a delusion. This seems wrong. Therefore, insight does not appear to be central to delusions.

2.2.4 Irrationality

A fourth way to distinguish delusions is by their irrationality. This irrationality can take many forms. One form is inference-based: for example, the *DSM-5* claims that

delusions are "based on incorrect inferences about external reality."[7] This seems to mean that delusions are unjustified or irrational depending upon what they are based on; they are counted as delusional only if they are based on poor grounds. For example, an individual who experienced weight-based teasing (e.g., was called "fat" by a peer) might develop the belief that they will only be accepted or loved by others if they are thin. While understandable, generalizing from one, or even a set of such experiences, to all social relationships would be irrational.

However, the claim that delusions are based on incorrect inferences is unclear about whether it's the *formation* of the belief that is irrational or whether it's the *maintenance* of the belief that is irrational. We don't think that focusing on the former—irrational belief formation—is a good way to distinguish delusions. Many of our beliefs are formed irrationally (i.e. based on poor grounds in their formation), including beliefs that exhibit cognitive bias and beliefs associated with positive illusions about ourselves. We don't want to count all these beliefs as delusional. Moreover, a person might form a belief on the basis of good reasons, but then maintain the belief in a way that's resistant to new counterevidence; it seems like we should count this as a delusion.

2.2.5 Fixedness

Rather than interpreting the *DSM*'s "incorrect inference" claim in terms of irrational belief *formation*, we might interpret it as irrational belief *maintenance*—that is, a belief is delusional when one continues to hold it in an irrational way. Indeed this idea seems to be present in many other *DSM* descriptions, according to which delusions are beliefs "held despite what constitutes incontrovertible and obvious evidence to the contrary" (APA 2013, p. 819), and "fixed beliefs not amenable to change in light of conflicting evidence" (p. 819). These descriptions speak to a different type of irrationality than forming a belief on the basis of poor grounds. The idea is that delusions are maintained *despite the evidence.* We might consider this feature to be *fixedness*.

However, it's not obvious what is meant by the term, 'fixed', and indeed different sources seem to use it in different ways. The "fixity of ideas" item on the BABS evaluates "how fixed or unshakable the patient's conviction about the belief is" (Eisen et al. 1998; p. 6). But this explanation of fixedness is ambiguous between the notion of strong confidence ('conviction') and the notion of holding the belief no matter what ('unshakeable'). Additionally, when the *DSM*-5 distinguishes delusion from overvalued ideas by appealing to "the degree of conviction with which the belief is held despite clear or reasonable contradictory evidence" (p. 87) this mixes conviction with holding a belief despite counterevidence.

[7] It's not clear what is meant by "external reality". Perhaps it gestures at the fact that the content of many delusions concerns the world external to the agent. This is another way the *DSM* characterizations focus on belief content to identify delusions. But one could have a delusion about internal reality (e.g. one's own mental states), so the "external reality" clause isn't a helpful focus. See also Coltheart et al. for critical discussion of both the focus on inference and the focus on external reality in *DSM* characterizations of delusion (2011, pp. 275–276).

We think that the notion of confidence more closely matches the notion of *conviction* (discussed above) than it does *fixedness*. A person might be very confident in their belief because the evidence overwhelmingly supports the truth of the belief; but if confronted with counterevidence, that same person will lower their confidence in the belief and perhaps even abandon it. For instance, individuals might hold beliefs about the effectiveness of purging for reducing the caloric impact of ingestion with high confidence until a clinician walks them through the available evidence on this topic (e.g., Bo-linn et al. 1983) and explains the physiology for why such methods are not nearly as effective as the individual thought. Although the individual may continue to experience difficulty reducing their use of purging, they may no longer think of the behavior the same way as they did before encountering this information. In this case, the person held their belief with high conviction, but the belief was not fixed. 'Fixedness' seems better reserved as a term for how likely a person is to maintain their belief when confronted with counterevidence.

We propose that fixedness be understood in terms of a belief's ongoing lack of responsiveness to reasons, or in other words, a belief's being insensitive to evidence once it is formed.[8] When an agent is functioning rationally with respect to the maintenance of their belief, they modify the belief in response to changes in evidence; when the rational agent is presented with counterevidence to their belief, they lower their confidence in the belief, and if the counterevidence is strong enough, they abandon it. If the belief is fixed, then the agent doesn't modify their belief in this way. Fixedness is evidentially insensitive belief maintenance.

Fixedness is present in paradigm cases of delusion. It does not seem to be subject to counterexample—we cannot think of a case of delusion that lacks this feature. It's also present in multiple versions of *DSM* characterizations of delusion, and it's present in the BABS' metric: "fixity of ideas". We propose that this way of believing is a constitutive feature of delusions – part of what it is to be a delusion is to be a belief that an agent maintains in a way that is not sensitive to evidence. Thus fixedness is a necessary condition for delusion.[9]

What exactly is it to maintain a belief in a manner that is insensitive to evidence? It's not just a matter of how a person *actually* believes. A person might actually believe that they are a direct descendent of George Washington—perhaps they've always admired him and they just saw him portrayed in *Hamilton* on Broadway. But they never communicate this belief to anyone and never do any research on the matter. If it is true of this person that they would keep believing that they are a direct descendent of George Washington were someone to present sufficient evidence that

[8] It's not necessary to use 'fixedness' to describe this feature. We're not committed to the word. We are committed to the concept we're trying to associate with the word.

[9] Note that thinking of delusion as partly constituted by fixedness doesn't automatically count religious and culturally-shared beliefs as delusional, because while fixedness is a necessary condition for delusion, we have not argued that it's sufficient. In keeping with this, the view also doesn't automatically count belief in conspiracy theories as delusional. Our view of delusion leaves open the possibility that certain of these beliefs may well be delusional, but we think this is a useful result. A good characterization of delusion should not automatically decide the question of whether certain types of beliefs are delusional.

Washington had no children, then this belief is plausibly considered delusional.[10] Whether a belief is delusional is a matter of how a person *would* believe were they to encounter counterevidence, even if they do not actually encounter it. Fixedness is a counterfactual notion. This means that delusions won't always be easy to identify—it will be easier to identify a belief as delusional if the person holding the belief actually encounters counterevidence and maintains the belief in the face of it. It's also the case that lack of responsiveness to reasons comes in degrees, with beliefs being more or less sensitive to evidence. While it's difficult to specify a threshold, if a belief is fixed to a certain high degree then the belief is delusional.

Fixedness is at the core of what makes a belief delusional. Other features commonly put forward to distinguish delusions, like implausible content, irrational basis, strong conviction, or poor insight often accompany fixedness, but they aren't necessary for delusional belief. Moreover all of these features seem related in some way to fixedness—for example, if a delusion is resistant to counterevidence, this helps explain why many delusions have bizarre content. Normally if something is bizarre or extremely implausible, we consider it prima facie reason to think it's false. But a delusion won't be sensitive to this prima facie evidence. Consider *conviction*: perhaps part of the reason why many delusions are held with great conviction (i.e. confidence) is because when holding a belief in a way that's not sensitive to evidence, one's degree of confidence is unconstrained by the evidence, and thus can be as high as one wants.

2.3 Summary

It's difficult to carve out the notion of delusion, but we've noted five main features emphasized to varying degrees in the *DSM* and the BABS. We've argued that the best characterization of delusion helps us clearly identify cases of delusions – even cases that are less common, like delusional beliefs with plausible content. Maintaining a belief in a way that is not responsive to reasons is at the core of delusions. So we ought to focus on this fixedness when trying to identify delusions, and when considering how we might treat disorders, like AN, that are related to them. With this understanding in mind, we turn to the causal relationship between delusions and AN.

3 Causal Relations Between Delusions and Anorexia Nervosa

There are four ways in which delusions may be related to AN. First, delusions may be epiphenomenal to AN. Second, delusions may partly cause the onset of AN. Third, delusions may be caused by aspects of AN. Fourth, delusions might both cause and be caused by aspects of AN. In what follows we discuss both the

[10] One might resist the idea that this belief counts as delusional if one also wants to maintain that all delusional beliefs cause some form of impairment, distress, or harm. But we think it's more useful to count this sort of belief as delusional and address separately whether the belief causes impairment, distress, or harm.

empirical and conceptual evidence for each of these relations. We'll argue that the most likely scenario is that delusions are involved in reciprocal causal relations with AN: they serve as a sustaining cause of AN, and aspects of AN also cause and/or strengthen delusions. Given that delusions have been repeatedly identified in a non-negligible minority of individuals with AN who have no other disorder that can account for them, and given that some causal connections between delusions and AN have potential implications for research and treatment, it is critical to explore how delusions and AN might be connected.

3.1 Delusions as Epiphenomenal

The first possible relation between delusions and AN is that delusions are epiphenomenal with respect to AN. Delusions may be associated with AN but not related in a direct causal process. For instance, delusions may be partially caused by the neurophysiological effects of starvation and malnutrition that can accompany AN. Upon first consideration, this might seem like a promising way to think of the relation between delusions and AN. When a person is malnourished there are a number of resulting physical effects, such as cortical thickness reductions (Brodrick et al. 2021), that may negatively impact mental functioning (e.g., Rylander et al. 2020; Keeler et al. 2022). We might consider delusions to be one output of such a process.

However, further reflection shows that this is an improbable explanation for the relation between delusions and AN. For one, we don't have any evidence to date that supports the view that the delusions in AN are the result of starvation and/or malnutrition (De Young et al. 2022). If they were, we'd expect that this would reveal itself in various empirical studies of AN. We also don't have any evidence that suggests that starvation and malnutrition in general (even apart from AN) are associated with delusion. Additionally, if delusions were a result of starvation/malnutrition, we might expect them to resolve when starvation/malnutrition were addressed. In a recent study of 50 women with severe or extreme AN assessed at intake and discharge from a specialty medical stabilization unit, delusional intensity did not change in response to medical stabilization and refeeding, although most other measured aspects of psychopathology did change (De Young et al. 2022). These results are consistent with delusions being a more stable feature of AN versus a transient consequence of starvation and/or malnutrition.

One last consideration that undermines the view that delusions are merely a consequence of the starvation and/or malnutrition associated with AN, is that the content of delusions in AN tends to be food and eating-specific. Kambanis et al. (2023) and Steinglass et al. (2007) both noted that the content of potential delusions among individuals hospitalized with AN centered around themes consistent with the disorder, although both studies specifically asked for examples of beliefs relating to eating or body image. If general starvation or malnutrition was causing delusions in AN, then we might not expect that their content would be limited to food and eating in this way. Delusions unrelated to the content of AN would likely be captured by a comorbid psychotic disorder diagnosis. Although this is not unheard of (e.g., Crișan et al. 2022), it is not a comorbidity observed nearly as often as delusions are

observed in AN, suggesting that delusions with content unrelated to AN occurring in individuals with AN is rare.

Finally, another way that delusions may be epiphenomenal to AN is if both AN and delusions share a common cause. Preliminary studies of genetic correlations suggest that schizophrenia and AN may share some genetic risk (e.g., Watson et al. 2019), and some researchers have even considered AN to be similar to psychotic disorders in certain respects (e.g., Poletti et al. 2022), which might imply shared causal processes. Notably, schizophrenia may be present without delusions, so we cannot draw firm conclusions about the relation of delusions and AN from these findings. It is too early in the development of this research area to rule in or out the possibility that AN and delusions are epiphenomenal due to shared cause.

3.2 Delusions as an Initial Partial Cause of AN

Delusions might be a premorbid cause of AN, being part of the process that results in the onset of AN. One reason to think this pertains to the content of many delusions observed in AN, for example, "Eating any food will make me fat." Provided that the individual holding such a delusion has internalized predominant cultural beliefs around fatness, they are likely to experience anxiety and even fear around food, be preoccupied by concerns about food and eating, and engage in restrictive eating behavior and/or compensatory behavior (e.g., fasting, excessive exercise) that consequently results in a body weight that is less than minimally normal. Analogously, an individual with a psychotic disorder who holds the delusion that their neighbor is spying on them is likely to experience anxiety and even fear when near the neighbor, be preoccupied with concerns about the neighbor, their motives, and what they know, and may even engage in extreme behavior aimed at mitigating their fear (e.g., keeping all their curtains closed, avoiding using the telephone, and rarely leaving their home) that results in serious impairment. Because delusions are not responsive to counterevidence, they tend to persist over time, which means they can influence behavior for a long time—long enough to support the development of AN, which tends to unfold over weeks, months, or years (e.g., Ranzenhofer et al. 2022).

If delusions were often (or even sometimes) a partial cause of the onset of AN, then we'd expect some empirical studies to detect them, given their salience to both the individual and those around them. Some evidence supports that individuals with AN experience deficits in flexible cognition, insight, and theory of mind (e.g., Bora and Köse 2016), which are cognitive features that might predispose individuals to delusional thinking. Additionally, psychopathology commonly comorbid with AN, such as depression and anxiety, can interfere with other cognitive skills, such as set-shifting (Roberts et al. 2010), which is the ability to move between different ways of approaching problems and is understood as an indicator of cognitive flexibility. Lack of cognitive flexibility could increase the risk of delusional thinking.

However, there is very little prospective evidence demonstrating that these features precede the onset of AN, and even the temporal ordering of comorbid disorders as risk factors for the onset of AN is not settled (e.g., anxiety disorders; Lloyd

et al. 2019). Further, there is no evidence, and very little theory, that positions delusions as causal or temporally preceding the onset of AN, despite many theories on the development of AN (see Zanella and Lee 2022). Rodgers et al. (2022) tested mood instability as a mediator between psychotic disorder and eating disorders in a large English population-based sample, but their data were cross-sectional and cannot address cause.

Additionally, retrospective research on the timing and sequence of symptoms in the development of AN yields no evidence that delusions cause AN. For instance, Ranzenhofer et al. (2022) found that dieting was the most common initial sign of AN in a group of 71 adolescents with AN interviewed along with their parents; however, cognitive features, including delusions, were not specifically queried. In another study utilizing a similar design, an open-ended question about what parents first noticed also did not result in identifying delusions (Rosello et al. 2022). If delusions were an initial partial cause of AN, we would expect them, or psychotic disorders more broadly, to be identified as risk factors for the development of AN. Given the volume of data that exists and has been examined in patient registries without identifying psychotic disorders as a risk factor for AN, it seems reasonable to conclude that delusions are not a predominant causal pathway to AN. Additionally, a priori, we think the causal pathway is not predominant, because the lack of available evidence for this pathway necessitates that loved ones and care providers systematically overlook the presence of delusions prior to the onset of AN. At the same time, it is critical to acknowledge that premorbid delusions could be overlooked, because their content generally adheres to cultural influences and would be occurring in an apparently otherwise healthy young person.

In sum, it's possible that delusions are an initial partial cause of AN. This causal relationship seems reasonable conceptually. But the relationship is not supported by available evidence. To be fair, it's not contradicted either. However, we think that if this were the predominant causal relation between delusions and AN, we would see some supporting evidence given extant studies. So, the fact that we don't, provides us some reason to think that it's *not* the predominant causal pathway.

3.3 Delusions as a Causal Effect of AN

A third way in which delusions might be causally related to AN is that the psychopathology of AN may directly cause delusions. For example, an individual with AN may observe themself restricting food intake, refusing others' efforts to help them, and exercising excessively, and feel compelled to explain their behavior in a way that limits their distress. Although a delusion such as "I cannot eat because my body cannot process food" is inconsistent with the evidence for an outsider, it may well be the glue that holds together an internally consistent set of thoughts, emotions, and behavior for the individual with AN. Thus, delusions might play a regulatory role in the cognitive life of someone with AN.

Recent philosophical research on the role of delusions in mental disorders suggests that delusions can play an adaptive role in the mental lives of those who hold

them.[11] For example, there is evidence that delusions in schizophrenia can relieve anxiety, at least in the short-term, and can enhance meaningfulness and contribute to an agent's sense of coherence (Bortolotti 2016). McKay and Dennett (2009) argue that some delusions might function metaphorically as a "doxastic shear pin." Shear pins in machinery are metal pins designed to break under stress and thus allow the machinery to continue functioning, albeit imperfectly; shear pins can save a snowblower, for example, from complete (and expensive!) breakdown. Similarly, delusions might be viewed as a cognitive "breakage" that would "ordinarily be rejected as ungrounded, but that would facilitate the negotiation of overwhelming circumstances" (2009, p. 501). Among other things, the positive role that delusions can play makes delusional belief more intelligible. This is not to say that delusions should be viewed as rational simpliciter or that they make a positive contribution to our mental lives *on the whole*, but rather that they make *some* positive contributions.

For example, Lancellotta and Bortolotti suggest that delusions in OCD and MDD can serve to preserve a person's sense of rational agency; they help an agent cohere their beliefs with their behavior and feelings. Delusions can be seen as "reducing the conflict caused by a clash in the person's emotions, beliefs, and behaviors and as restoring some levels of intrapsychic coherence" (2019, p. 12). This is a helpful framework for understanding the role that delusions can play in AN. The delusion provides a belief that makes sense of otherwise irrational or unintelligible *actions* (e.g. severely restrictive eating) as well as *emotions* (e.g. fear of even one bite of food).

Additionally, Lancellotta and Bortolotti (2019) argue that delusions can function as a temporary coping mechanism in response to difficulty or trauma. Applied to AN, a delusion might help a person manage various fears, like the fear of becoming fat or the fear of losing control. For example, if someone is afraid of becoming fat, believing that the only way to not be fat is to not eat helps manage that fear. The person can focus on not eating, rather than focusing on the paralyzing fear or on the difficulty of getting rid of the fear. In this way delusions can be emotion regulatory in AN. If a particularly damaging emotion can be avoided, then a certain cognitive peace is achieved, at least in the short term. The damaging emotion may range from moderate discomfort to absolute terror. Imagine a person with extreme AN whose appearance is startling, evidencing starvation not commonly witnessed in modern, wealthy nations, with medical complications requiring acute inpatient management. In the absence of a belief that rationalizes the behaviors that have caused and are perpetuating their condition, such an individual is likely to experience a compounding severe emotional state (e.g. intense fear) and corresponding intense cognitions (e.g., believing they are going crazy).

In addition to shielding a person from fear, a delusion might also shield a person from other difficulties, like the difficulty with change. Someone with AN who is undergoing treatment is asked to make many difficult changes: changes in perspective, values, goals, life vision, sense of self, sense of the future, definition of success,

[11] See Bortolotti (2015) and (2016); Lancellotta and Bortolotti (2019); Bortolotti and Miyazono (2016); and Bortolotti et al. (2017).

and so forth. Delusion might preserve the ability of an individual with AN to maintain what seems to be working for them. Indeed, many individuals with AN do not seek treatment on their own and are brought to treatment by concerned loved ones (Guarda 2008). This fits with the view that the behaviors of many individuals with AN are *egosyntonic*—they are compatible with the individuals' own values and beliefs, even if they're incompatible with the beliefs of others, like friends and family (Guarda 2008).

All of this reasoning supports the notion that AN can cause delusions. However, like the possibility that delusions precede the onset of AN, the possibility that delusions follow the onset of AN is beset by a lack of clear evidence. It is insufficient to identify delusions among individuals with AN and conclude that the delusions followed the onset of AN. One must further demonstrate that the delusions were absent prior to the onset of AN. To our knowledge no studies have investigated this. We think that the idea that AN causes delusions is very plausible. Moreover, a priori, this is a more plausible causal pathway than delusions causing AN, given the deteriorating course that AN can follow (Treasure et al. 2015). It's likely that more severe features (e.g., delusions) follow rather than precede less severe features (e.g., dieting).

3.4 Delusions as Reciprocally Causal with AN

As noted, it is plausible that individuals adopt delusions in a process partially motivated by maintaining a sense of coherence about their experience with AN and/or partially motivated by emotion regulation. Once these beliefs are adopted and held at delusional intensity, they may worsen AN psychopathology. For instance, an individual with the belief that eating anything with dietary fats will make them fat immediately would understandably avoid eating dietary fats, if they wish to avoid becoming fat. In this way, delusions may be reciprocally causal with AN, worsening the psychopathology in a positive feedback cycle, even amplifying over time, as in a dynamical causal process (e.g., Salvi et al. 2021).

Delusions are associated with more severe eating disorder psychopathology in AN (e.g., Kambanis et al. 2023), and notably, delusional intensity predicted worse fear of fatness and restrictive eating 3.6 weeks later in an inpatient AN sample (De Young et al. 2022). These findings are consistent with, but do not directly support, a reciprocally causal process. However, one must ask why the delusions observed in AN almost uniformly involve eating disorder-related content (Kambanis et al. 2023; Steinglass et al. 2007). One could explain similar behavior by believing that food is poisonous, yet delusions of this type have been only rarely documented in AN (e.g., Pruccoli et al. 2021). We suggest that the delusions that form following the onset of AN are consistent in content with premorbid, non-delusional beliefs, that themselves may be related to the risk for AN (e.g., beliefs about eating and thinness that motivate dieting behavior; Simmons et al. 2002).

There are likely many partial causes of AN (Zanella and Lee 2022), none of which wholly account for the disorder in any specific case. Premorbid non-delusional beliefs may be one such partial cause. For example, one might believe (non-delusionally) that dietary fats make them gain weight. This may lead the individual to restrict their

dietary intake of fats. This restriction, together with a host of other causal influences (e.g., genetic, hormonal, sociocultural, etc.), may result in AN. Once AN has onset, the individual may experience a heightened sense of needing to explain their behavior. They may do this by increasing their conviction and resistance to counter-evidence for the belief that dietary fats make them gain weight, perhaps even by modifying the belief to be more extreme (e.g., "Any ingestion of dietary fats will make me gain 5 lbs. immediately"). Holding this, now delusional, belief makes continued restriction of intake predictable. Notably, a very similar process is also possible wherein the premorbid non-delusional belief is not a partial cause of AN but is nevertheless the material out of which the delusional belief is made once AN has developed.

3.5 Summary

We've canvassed how delusions might relate to AN. First, it's unlikely that delusions are epiphenomenal to AN: we lack the empirical support for the view that the starvation and/or malnutrition associated with AN causes delusions, although research on common causes of both delusions and AN is nascent. Second, it's unlikely that delusions are part of what causes the onset of AN, because there are no clear mechanisms explaining why a person would develop delusional beliefs about eating and food-oriented content apart from AN; we also lack both prospective and retrospective evidence that delusions partially cause the onset of AN, despite relevant extant studies. Third, we argued that it's plausible to think that the psychopathology of AN causes delusions: delusions might play a role in preserving a person's sense of rational coherence, and in helping an individual regulate their emotions. We lack evidence for this, but it's also not been tested. Lastly, we outlined plausible ways that delusions might be caused by the psychopathology of AN, and in turn, cause the psychopathology to worsen and/or help sustain it over time. This seems like the most likely causal relation between delusions and AN, especially given that premorbid non-delusional beliefs related to food, weight, and exercise might explain the development of delusions in conjunction with the symptoms of AN.

4 Implications for Research and Treatment

The definitions of delusions and explanations of relations between delusions and AN described here have numerous implications for research and treatment. We first provide possible research directions that, if tested by gathering relevant data, would expand our understanding of the convergence of these phenomena. Second, we discuss the implications of the possible conclusions resulting from this research in terms of treatment.

4.1 Proposed Research Directions

If holding a belief despite counterevidence is the core of delusions, we can design assessments of that specific construct. Presently we do not have measures of this

construct. The BABS contains one item (i.e., fixity of ideas) that is intended to quantify this construct, and it does so on a four-point, ordered categorical scale. Together with other BABS items, this item contributes to assessing the dimension of delusionality, as operationalized by the BABS. However, alone, it is unlikely to be an adequate assessment of holding a belief despite counterevidence for a simple reason - measurement error. Assessments that perform well utilize multiple indicators of the same construct to offset errors inherent in any single indicator. Using multiple indicators has the additional advantage of approximating a dimension when ratings are aggregated. Thus, the BABS fixity of ideas item is a good place to start for designing an assessment of holding beliefs despite counterevidence, but work is needed to develop and validate an assessment for this specific construct.

One idea would be to use a breakpoint assessment, sequentially providing counterevidence with increasing weight. Breakpoints are described in the behavioral economic literature as a way to quantify the relative reinforcing value of multiple reinforcers and are postulated as indicating elasticity in response tendencies (Bickel et al. 2000). Elasticity can be thought of as the opposite of fixedness, reflecting the extent to which individuals demonstrate behavior change in response to changing contingencies. To assess holding beliefs despite counterevidence, items could ask whether individuals would reconsider their beliefs given evidence of increasing evidential weight. Those who maintain that they would not reconsider through to the end of such an exercise may be judged to be delusional in their beliefs. Otherwise, the point at which individuals reconsider (i.e., the breakpoint) is an estimate of the degree to which they hold their belief despite counterevidence. This type of assessment is generally brief and repeatable, which is critical to its utility for tracking change.

A second research direction regards possible shared causes of the co-occurrence of delusions and AN. Such research on comorbidities would require assessing *delusions specifically* and not solely as a feature of psychotic disorders. For example, Watson et al.'s (2019) study linking schizophrenia to AN by shared genetic risk does not help here, because delusions do not always occur with schizophrenia, as mentioned in Section 3.1. Delusions are not a required feature of a number of diagnoses but may be present in several. This means that, at the diagnostic level, comorbidities cannot be fully informative about whether AN is accompanied by delusions. Thus, research on shared causes at the diagnostic level will not likely be sufficiently granular to unveil causes of the relation between delusions and AN.

A third direction concerns the necessity of using longitudinal research to establish temporal precedence in the ordering of delusions and AN when they co-occur. This requires prolonged commitment of resources to broad-based risk factor research on AN. We are aware of how unlikely it is that a funded, longitudinal research agenda is established for this low base rate phenomenon (i.e., delusions plus AN), especially in the US, given the underfunding of research on eating disorders despite their substantial social and economic burden (Streatfeild et al. 2021). However, many longitudinal projects on other psychiatric conditions and risk factors already exist and will continue to be funded. Creative and collaborative researchers should work to include the measurement of eating disorders, known eating disorder risk factors, and delusions in such projects to elucidate the ordering of the onset of these phenomena.

A fourth direction involves testing for reciprocally causal relations between delusion and AN. From a research design perspective, this is the most challenging. To examine reciprocal cause, we would ideally manipulate delusions to examine effects on AN *and* manipulate AN to examine effects on delusions. However, even if we could, we would not induce delusions nor would we induce AN. This means that the only available manipulations would be treatments to which individuals comorbid with AN and delusions would have to be randomized. If delusions and AN are reciprocally causal, intervening on delusions should have some ameliorative effect on AN, and intervening on AN should improve delusions. One difficulty with this approach is that we do not know the strength of these potential causal relations, which makes study design difficult. Relative to other partial maintaining causes of AN, delusions might be a small influence present in ~20% of inpatient cases. Therefore, detecting a statistically reliable effect on AN by intervening on delusions will be difficult, unless that effect is large. Additionally, inactive control conditions would be unacceptable, given the severity of this comorbid presentation. A realistic approach, though limited in its ability to inform on the causal relations between delusions and AN, is to add an intervention component for delusions to current best practices for treatment. If the addition improves treatment outcomes, we might infer that delusions were a partial maintaining cause of AN. These interventions may target the delusion directly or the cognitive biases that make an individual vulnerable to delusions (e.g., McKenna et al. 2014). Similarly, we could measure delusions over the course of treatment for AN to track the extent to which delusions improve with the successful treatment of AN despite not being directly targeted. Surprisingly, this has not yet been done.

An alternative approach that might inform on reciprocal causality is network analyses (McNally 2021), which test for bidirectional causal relations among several variables hypothesized to be interconnected within a causal network. Network theory posits that disorders *are* their various signs and symptoms, and the relations between them, rather than some latent pathology that manifests as independent signs and symptoms. An advantage of this approach is that it does not rely on randomization and manipulation. Consequently, causal conclusions are tenuous. Given that delusions appear to be stable during the initial medical stabilization and refeeding of AN (De Young et al. 2022), repeated assessments of the variables in a network analysis would not need to occur especially frequently. Once per month during the course of treatment might be adequate for testing whether and how the relations represented in the network have changed. Network analyses are being increasingly employed to study the psychopathology of AN and tested for their utility in predicting response to treatment, but to date they have not included many variables of interest, including delusions (Monteleone and Cascino 2021).

4.2 Potential Treatment Implications

Conceptualizing delusions as beliefs that are not responsive to counterevidence has a few compelling implications for the treatment of AN, if delusions are identified as existing in a reciprocal causal relation with AN. First, approaches rooted in cognitive therapy that are commonly included in cognitive-behavioral therapy (CBT) protocols aim to have

individuals examine the evidence supporting their thoughts, engage in active disputation of their thoughts, and ultimately alter their thoughts in ways that support adaptive functioning. Given our conceptualization of delusions, such approaches seem doomed to fail when applied to delusions. Further, the content of many delusions in AN involves vague constructs (e.g., fat, unhealthy), which present additional challenges for determining what counterevidence is relevant. Therefore, we consider alternatives.

Cognitive therapy adapted for delusions conceptualizes delusions as culturally unacceptable interpretations of intrusions into awareness driven by faulty self and social knowledge and maintained by cognitive and behavioral responses (Morrison 2001). Although this approach has demonstrated large effects on delusions in schizophrenia (Grant et al. 2012), to date, it has not been trialed in AN.

Cognitive remediation therapy (CRT) has been suggested as a possible remedy for cognitive deficits, including set-shifting and central coherence, for a host of psychiatric disorders (Trapp et al. 2022) and has been applied to AN (e.g., Herbrich-Bowe et al. 2022). This treatment, often proposed as adjunctive to other treatments, uses computerized tasks that can be delivered as challenging games, increasing retention while strengthening cognitive skills that could undermine delusions. Unfortunately, evidence of the effectiveness of this approach on key outcomes in AN is mixed (Hagan et al. 2020). CRT has not been tested as a targeted intervention for individuals with AN experiencing delusions; however, the idea of only providing the intervention to individuals with significant cognitive impairment is suggested for improving outcomes and obtaining more consistent results with CRT (Trapp et al. 2022).

Acceptance based approaches might be promising. For instance, both Emotion Acceptance Behavior Therapy (Wildes et al. 2014) and Acceptance and Commitment Therapy (Parling et al. 2016) involve acknowledging thoughts and feelings but acting in accordance with one's goals and values despite them. The focus is on making change despite cognitions and emotions rather than on trying to change them. Acceptance based approaches have demonstrated positive effects when applied to individuals with schizophrenia (e.g., El Ashry et al. 2021), but are not superior to cognitive behavioral therapy for AN (Linardon et al. 2017). But again, these approaches have not been tested specifically in individuals with AN experiencing delusions.

Finally, although preliminary and mixed, recent findings on the utility of atypical antipsychotic medications for AN (Muratore and Attia 2021) suggest that testing these medications among individuals with AN experiencing delusions may be worthwhile. Critically, whatever treatment approach is chosen to address this vexing presentation, is it crucial that researchers monitor changes in delusions, identify resistance to counterevidence as a treatment relevant variable to track, and test these measurements as outcome predictors.

5 Conclusion

We've sorted through common descriptions of delusion in the *DSM* and the BABS, and we argued that the most important feature to look for in a belief to determine whether it's delusional is whether the belief is fixed—that is, whether it's maintained

in a way that is insensitive to evidence. We argued against the likelihood that delusions are epiphenomenal with respect to AN. Instead we think they're causally related: it's possible, but not likely, that delusions partially cause the onset of AN; it's plausible that AN causes delusion; and it's even more plausible that AN causes delusions that worsen or sustain AN. Given that 10–30% of people with AN have delusions, and given the low rate of treatment success and high rate of relapse, it's important to design research to test for the causal relations between delusion and AN. We also outlined several implications for treatment, given our conception of delusions and given the plausibility of the causal relation between delusions and AN, that may lead to improvements in treatment outcome.

Declarations

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