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**Using an OCD formulation in the treatment of anorexia nervosa: A useful way to
understand the illness?**

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Running head: OCD formulation in AN

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Abstract

Background: Anorexia nervosa (AN) and obsessive-compulsive disorder (OCD) have been shown to have a number of commonalities, such as genetics, neurobiology, and symptoms. Approaches to treatment of AN have recently been described that take such findings into account, extending interventions recommended for obsessive-compulsive and anxiety disorders to AN. **Aims:** The current paper aims to outline a formulation model of AN in adults, derived from literature on OCD, and introduce this topic as a fruitful area to build on existing treatment techniques, and to prompt further discussion of such techniques. **Methods:** A formulation model is described, followed by a discussion of how this might be applied to AN, using examples from clinical practice. Potential benefits and difficulties are discussed. **Conclusions:** A formulation model is suggested that can easily be adapted to AN, complimenting existing models in eating disorders.

Keywords: formulation, anorexia nervosa, obsessive-compulsive disorder, therapy

1 Introduction

2 Anorexia nervosa (AN) has proven to be an illness with a high mortality rate, often with
3 a chronic outcome (Steinhausen, 2002). Full understanding of this illness for an
4 individual patient, usually reached by a process of psychological formulation (e.g., see
5 Bieling & Kuyken, 2003; Lavender & Schmidt, 2006; Persons & Tompkins, 2007), is
6 important to design and implement a successful treatment (Persons, 2006). This
7 formulation should be unique to the individual, although generic models of pathology
8 (e.g., Fairburn, 2008; Waller *et al.*, 2007; see also Persons, 2006) can help therapists
9 and patients come to a shared understanding of their difficulties, and how they are
10 perpetuated.

11 Features of obsessive-compulsive disorder (OCD) and obsessive-compulsive
12 personality are associated with poorer outcome and greater symptomatology in AN and
13 other eating disorders (Steinhausen, 2002; Zubieta *et al.*, 1995), represent frequent
14 comorbid illnesses with EDs (Swinbourne & Touyz, 2007), and there has also been
15 some genetic and biochemical evidence of an association (e.g., Enoch *et al.*, 1998). As
16 a result of such findings, a number of authors have looked at whether AN is actually a
17 variant of an OC disorder, with some genetic, neurological, and developmental evidence
18 supporting this (e.g., Milos *et al.*, 2002; Serpell *et al.*, 2002; Steinglass *et al.*, 2012).
19 Some authors (e.g., Steinglass *et al.*, 2011; Strober *et al.*, 2007; Waller, 2008) have
20 gone even further, suggesting that eating disorders, including AN, might be included in
21 a wider categorisation of anxiety disorders. The stance of the current article is that AN
22 and OCD are not variants of the same condition, but that some formulation and
23 treatment techniques used in anxiety disorders may be useful when working with
24 individuals with AN. The article will explore whether a formulation model frequently

used in OCD might be applicable to AN, describing a rationale as to why this is suggested and providing a template for its use. The model is targeted at the assessment and formulation of AN in adults, partly as systemic treatment (rather than a CBT model) is advocated in younger patients, and also that the literature referred to in this article primarily concerns adult subjects; less has been written about the overlap of OCD and AN in children, although Hildebrandt, Bacow, Markella, and Loeb (2012) discuss similarities between methods of action in family-based therapy for adolescents and CBT, specifically exposure.

A small body of evidence suggests that treatments used in anxiety disorders might be useful in EDs (e.g., Steinglass *et al.*, 2012), although some techniques reported in ED treatments (e.g., Waller *et al.*, 2007) may achieve change through similar principles. Some authors have written about the process of formulation in EDs, proposing models to understand the illness (e.g., Fairburn, 2008; Lavender & Schmidt, 2006; Waller *et al.*, 2007). Given that AN is a particularly difficult illness to treat, and sufferers often show denial regarding their symptoms (Vitousek, Watson, & Wilson, 1998), a formulation model that references a different condition (i.e., one that might not be seen as so egosyntonic) may offer a ‘back door entry’ to understanding the individual and his or her illness. In light of the literature discussed above, the aims of this article are to suggest how use of an OCD formulation (coming from a CBT approach) can contribute to an understanding of AN. Similarly, the focus on formulation throughout this article is designed to help therapists, particularly those who may be less experienced in the assessment and treatment of eating disorders, by outlining a more familiar model of formulation that might be useful in AN. Although some anecdotal data are presented, there is no formal evaluation of the utility of the model. In concert with the aims and

readership of the journal, elements of the model will be individually expanded on, although this level of detail may not be necessary with every patient.

Empirical Rationale for use of an OCD Model in AN

As detailed above, there is significant literature suggestive of some degree of phenomenological, diagnostic (i.e., comorbidity), and clinical overlap between AN and OC disorders, namely OCD, and a formative body of evidence supporting anxiety-based treatments in AN. There is some emerging evidence supporting the benefit of CBT for AN in adults (e.g., Fairburn *et al.*, 2013), a psychological therapy with documented effectiveness for the treatment of OCD, and there exists good reason to draw parallels between the two disorders.

Steinglass and Walsh (2006) reviewed a number of cognitive, behavioural, and neurobiological commonalities of AN and OCD, drawing parallels between intrusive thoughts, ritualised behaviours, and cognitive inflexibility, for example. The authors note similarities in the neurobiology behind AN and OCD, and build on this in a later article (Steinglass *et al.*, 2011). Steinglass and colleagues (2011) describe a treatment approach, based on the original neurobiological model of Steinglass and Walsh, focused on anxiety. Their model has a strong focus on behavioural fear acquisition, although combines some elements of eating disordered cognitions with avoidance-based behaviours. Citing the high relapse rate of current treatments, Steinglass *et al.* advocate a shift towards the use of behavioural techniques in the treatment of AN to address “both anxiety driven behaviours and irrational beliefs” (p. 140), particularly emphasising exposure and response prevention. They argue that anxiety and obsessional tendencies bring about certain eating behaviours (e.g., food avoidance) that

1 lead to weight loss. This then increases anxiety and obsessionality, thus perpetuating
2 the cycle (Steinglass *et al.*, 2010), and therefore advocate behavioural interventions that
3 promote experiential learning. However, this model does not include early experiences
4 and underlying beliefs, which are similar in anxiety and eating disorders (e.g., Pallister
5 & Waller, 2008; Waller *et al.*, 2000), although it could be argued that the model is
6 easily adapted in this respect.

7 The current article argues that a more complete model of AN would include these,
8 but also the phenomenon in OCD often labelled “catastrophic misinterpretations.”
9 Individuals with AN often report intrusive thoughts which, similar to OCD and other
10 anxiety disorders (such as panic disorder), are misinterpreted. Cognitive behavioural
11 treatment might then look at addressing beliefs and thinking styles related to those
12 intrusions and trying to normalise behavioural responses (see below). Intrusive
13 thoughts are common in the general population, but become obsessional when they are
14 more frequent or intense, or produce greater discomfort (e.g., Rachman & de Silva,
15 1978). Similar to the sense of ‘increased responsibility’ and catastrophic
16 misinterpretations in anxiety (e.g., Salkovskis, 1991; 2007), an individual with AN
17 might escalate an intrusive thought into an obsession. An example of a distortion that
18 may be seen in EDs is provided by Cooper, Todd, and Wells (2009, p. 127); ‘if I get fat,
19 no-one will ever speak to me again’. It may be the case that many individuals
20 demonstrate an aversion to fatness or obesity, or a desire to weigh less (e.g., Whitaker *et*
21 *al.*, 1989), but that this may be misinterpreted in those with ED pathology as having
22 more ruinous consequences, such as extreme social isolation; a trigger might be the
23 initial intrusion, which is subsequently catastrophised.

Given this, it is argued that individuals with AN experience intrusive thoughts that are common to the general population, but interpret these thoughts as posing significant harm to themselves, and / or that they hold greater personal responsibility. This inflated responsibility then leads to a pattern of behaviours that have the unintended effect of increasing distress and also increasing the frequency and salience of intrusions (e.g., Salkovskis, 2007). Elements of this model are expanded below but, by way of example, an intrusion in AN (e.g., about eating a certain food and subsequently gaining weight) might trigger responsibility beliefs, which then result in overt efforts to reduce or escape responsibility (e.g., by exercising, or checking for perceived weight gain).

Although some similarity with existing formulations of EDs (e.g., Waller *et al.*, 2007) exists, an OCD model (e.g., Salkovskis, 2007) includes a number of key elements, such as intrusions, responsibility appraisals, catastrophic misinterpretations, and neutralising actions (e.g., thought suppression), that may be applicable to some patients with eating disorders. Indeed, a number of studies (e.g., Coelho *et al.*, 2012; May *et al.*, 2010; Soetens *et al.*, 2006) have reported evidence of such phenomena in eating pathology. The use of an OCD model with certain individuals is proposed to represent a useful account of how different thoughts and behaviours interact and it is argued that specific elements of an OCD formulation (e.g., catastrophic misinterpretations, beliefs about intrusions) may be important in understanding an individual's presenting symptoms. The model also provides a template for constructing a psychological formulation of AN. The current article does not suggest an overhaul in the way AN is conceptualised, but rather that, for some patients, a model based on OCD might offer a 'different way in' to their illness. An OCD model in particular is favoured over other anxiety formulations (e.g., panic disorder, health anxiety disorder) due to a

larger evidence base suggestive of similarities between AN and OCD, as well as the additional flexibility and scope of an OCD model. While some elements (e.g., cognitive bias, safety behaviours) are common to many formulation models of anxiety disorders (and, sometimes, EDs), the OCD model presented here (Salkovskis, 2007) incorporates a wide range of different phenomena (see below), and is argued to present a more complete account of behaviours, cognitions, and so on that might be seen in AN. It is hoped that a useful formulation model is proposed that is accessible to both patients and clinicians who may not have a wealth of experience in EDs.

A CBT model of OCD, carefully applied, can help identify the above elements in treatment, and suggest ways of addressing maintaining mechanisms in AN. One clear advantage of using a purely OCD-type model in joint formulation with a patient with AN, in the author's opinion, is that it can be used as a Socratic, hypothesis-driven way of accessing the impact of thoughts and behaviours. Those with AN often have an egosyntonic view of their behaviours (e.g., Holden, 1990) and it can often be difficult to objectively appraise their function. However, drawing parallels with OCD behaviours (which can often be easily understood by patients) represents a less challenging way of introducing how ED behaviours may function to modulate anxiety, rather than to maintain weight, for example; illustrations from the patient's own experience can also be incorporated.

Clinical Practice

A description of how an OCD model (see Figure 1; Salkovskis, 2007) is used in the formulation of anorexia nervosa now follows, and it is argued to provide a useful and underutilised tool in eating disorders treatment. Although this is not a case series, some

anecdotal data from patients is provided in order to support some of the assertions made.

Insert Figure 1

First, as in many styles of psychotherapy formulation (e.g., Wildes & Marcus, 2011), the patient is introduced to the model. It is suggested that this is done with reference to a fictional, archetypical example of OCD, such as an individual with compulsive hand-washing resulting from intrusive thoughts around contamination. The model is then talked through, helping the patient generate examples of each of the parts in Figure 1. Any questions or confusions are necessarily addressed, and the patient is collaboratively assisted to generalise this formulation to their own illness experiences (e.g., Overholser, 2011; Persons & Tompkins, 2007). A useful, Socratically-driven, question might be something like “Why do you think we have been discussing OCD; what relevance might it have to your eating, for example?”. Often, it is then useful to jointly go through the model, and introduce concepts of an eating disorder specifically relevant to the patient (an example is given in Figure 2). Time can be spent on the individual elements of the model (which are elaborated upon in the current article for the educational purposes), but the main aim is to understand how anxiety processes might function in eating disorders. This process of introducing and discussing the formulation may only require one session of CBT, depending on the level of understanding and comprehension of the patient.

Insert Figure 2

3 **Early Experiences / Critical Incidents**

4 Psychological models of anxiety disorders often refer to critical incidents as activating
5 disease-specific beliefs in vulnerable individuals, and how early experiences also
6 contribute to the formation of specific assumptions and beliefs. This is a fairly
7 ubiquitous viewpoint in CBT, and is extended in the current model to AN. By
8 understanding an individual's history, a greater sense of their belief system can be
9 agreed with the therapist, and the patient helped to understand how their experiences
10 may have contributed to, say, an over-evaluation of weight and shape. Personality
11 factors (e.g., see Serpell *et al.*, 2002; Shafran, 2002), which may also be similar across
12 AN and OCD, are also relevant here. Critical incidents may include the onset of
13 dieting, loss or bereavement, and developmental influences (e.g., puberty) have also
14 been proposed as possible factors (e.g., Stewart, 2005).

16 **Assumptions / General Beliefs**

17 Eating disorders and OCD have been suggested to have a number of beliefs in common,
18 such as perfectionism, importance of thoughts, and control of thoughts (e.g., Lavender
19 *et al.*, 2006; Roncero, Perpiñá, & García-Soriano, 2011). Such beliefs might form a key
20 maintenance mechanism, common to EDs (Roncero *et al.*, 2011), and more specific
21 beliefs might relate to other factors, such as exercise (e.g., Naylor, Mountford, &
22 Brown, 2011). More general beliefs (e.g., schemas) are also shared between anxiety
23 disorders and eating disorders (e.g., Pallister & Waller, 2008), which can be explored
24 using the Salkovskis (2007) model elaborated upon here. Negative self-beliefs in EDs

have been discussed previously (e.g., Cooper *et al.*, 2009), with reference to eating-specific beliefs and more general self-beliefs. Such beliefs might be open to misinterpretations (see below) and attentional bias (Jansen, Nederkoorn, & Mulken, 2005; see Aspen, Darcy, & Lock, 2013, for a review of attentional bias in EDs). Individuals with OCD have been shown to have a number of cognitive biases which increase their level of intrusions, and therefore compulsions (e.g., Spranca, Minsk, & Baron, 1991), part of the large literature regarding attentional bias in anxiety disorders. This is less well understood in EDs (Aspen *et al.*, 2013), and it may be that bias is a relatively automatic (unconscious) process or more deliberate, and, of particular note in AN, may also be affected by nutritional status (e.g., Placanica, Faunce, & Soames Job, 2002; see below). It may be constructive to look at such biases in anorexia nervosa on an individual level, but this is also one area that is not overtly covered in the model of Steinglass *et al.* (2011).

Intrusions

It has been suggested (e.g., Shafran, 2002; Soetens & Braet, 2006) that individuals with EDs, and particularly those who restrict their dietary intake, are likely to experience unwanted (intrusive) thoughts around food, most often ‘bad’ foods (e.g., high calorie, high fat foods). Dieters can become preoccupied with thoughts of food and eating, and may be more likely to use thought suppression than non-dieters (Soetens *et al.*, 2006). Intrusive thoughts are commonly experienced in the general population (e.g., Rachman & de Silva, 1978) and may be an antecedent of eating pathology, as has been suggested in OCD (Salkovskis & Campbell, 1994). However, it is the appraisal of these thoughts (e.g., as personally significant) that often drives pathology, associated distress, and thus

attempts at control; this may be likened to the over-evaluation of weight and shape that is central to EDs (Shafran & Robinson, 2004). As in OCD, individuals with AN may have intrusions of a similar nature, and react in ways that serve to maintain and encourage the disorder. For example, Salkovskis and Wahl (2004) discuss how thoughts only become intrusive “depending on the person’s prior experience and the context in which the intrusions occur,” noting also that “the majority of nonclinical subjects do not regard the occurrence of intrusive thoughts as being of special significance” (p. 141). Similarities might be drawn to eating disorders, with food being of central importance in many people’s lives; however, in nonclinical samples, there is likely to be less emotion and personal responsibility attached to this (e.g., see Rawal, Park, & Williams, 2010).

Misinterpretations

Individuals with EDs have been found to misinterpret the consequences of thoughts particularly in the domains of food, weight, and shape, but also show misinterpretations related to wider beliefs, such as perfectionism or other beliefs about the self (e.g., Shafran, 2002; Vitousek & Ewald, 1993; Williamson, Muller, Reas, & Thaw, 1999). Such misinterpretations of intrusive thoughts are likely to increase the significance and persistence of these thoughts (Shafran, 2002; Shafran & Robinson, 2004) and can also lead to mood changes and safety behaviours (see below). A related concept is that of thought-shape fusion (TSF; Shafran *et al.*, 1999), a cognitive distortion akin to thought-action fusion seen in OCD. Briefly, TSF occurs when thinking about a negative cognition (e.g., eating a high-calorie food) leads to a change in the ‘real world’ (e.g., in behaviour or feelings). TSF may also be strengthened through associative learning

(e.g., thinking about eating a fattening food, and then eating it) and is thought to be a cognitive bias that increases the likelihood of catastrophic misinterpretations and maladaptive coping strategies (Shafran *et al.*, 1999). Such processes might also be accentuated by the relationship with mood changes and beliefs, for example guilt for eating a certain food being (mis)interpreted as ‘evidence’ of personal responsibility and potential negative outcomes, which may also relate to other cognitive biases, such as emotional reasoning. As in OCD, belief in TSF is likely to motivate certain behaviours, which in the case of AN may include dietary restriction, or body checking. Although TSF is also a relatively new area of research in EDs, a few studies have documented an association with ED symptoms and have suggested that it is unique to eating pathology (Coelho *et al.*, 2012). The relationship between such cognitive distortions and misinterpretations in AN may benefit from further research, as has been carried out with OCD (e.g., Abramowitz *et al.*, 2001).

Safety Behaviours and Neutralising Actions

Although included in the lower portion of the model, safety behaviours (e.g., see Pallister & Waller, 2008) can be a particularly useful place to start, explaining the rationale that safety behaviours can actually increase levels of anxiety (e.g., Deacon & Maack, 2008). This might have impacts on recovery as one aim of treatment is to reduce safety behaviours, due to their role as maintaining factors in the illness (Pallister & Waller, 2008). Davis and Kaptein (2006) discuss exercise as one example of a safety behaviour, drawing links to obsessional personality and ritualistic behaviours in AN. Similarly, attempts at thought suppression, designed to eliminate intrusive thoughts, can arise from misinterpretations and increase the persistence of those intrusions. Some

evidence for this in eating pathology has been found and, indeed, thought suppression has been suggested (as in anxiety disorders) to increase the number of subsequent food-related thoughts (e.g., May *et al.*, 2010; Soetens *et al.*, 2006). Other examples of safety behaviours include avoidance of certain foods (e.g., those deemed to contain relatively high levels of fat) and other (possibly related) dietary rules, and mirror or body checking (see Fig. 2). Although there is some similarity between neutralising actions and safety behaviours (Veale, 2007), it may be helpful to elaborate on this with the patient, using Socratic exploration to identify the specific function of relevant thoughts and / or behaviours. A number of different neutralising behaviours seen in an experimental paradigm are described in Radomsky, de Silva, Todd, Treasure, and Murphy (2002) (see also Kostopoulou, Varsou, & Stalikas, 2011). Thus, in the model proposed, there is some evidence for the interactive processes between neutralising actions, intrusions, and misinterpretations. In the therapy session, it would be important to discuss these processes with patients; treatment may involve ‘breaking the cycle’ of problem and attempted solution (Soetens *et al.*, 2006).

Mood

There are a number of suggested links between negative affect and EDs although, in a meta-analytic review, Stice (2002) suggests that the association might be stronger in bulimic disorders. The role of negative mood in eating pathology also needs to be considered on an individual level, particularly as depression and low self-esteem may be antecedents to ED pathology, but also consequences (Waller *et al.*, 2007).

The influence of mood on both cognitive bias and thought suppression has been highlighted, for example, in that low mood may lead to more negative interpretation of

thoughts (Shafran & Robinson, 2004) or influence information processing and interpretation in EDs (Aspen *et al.*, 2013). Furthermore, Altman and Shankman (2009) discuss how compensatory behaviours (e.g., purging in EDs, or handwashing in OCD) function to reduce negative affect, as well as anxiety, and thus may establish a mood-improving cycle. Existing models of EDs (e.g., Fairburn, 2008; Waller *et al.*, 2007) also comment that negative affect may be alleviated (albeit temporarily) by ED behaviours, but that low mood might also be responsible for changes in ED behaviour. Thus, low mood (as seen in the Figures) may form a complex part of an individual's formulation which both drives, and is driven by, symptoms of an eating disorder.

Initial Impressions

Based on the author's experience of using such a model in psychological treatment (which is not in 100% of cases seen in clinical practice), individuals with AN report positive engagement with the model, and have generally felt it to be helpful and informative. Patients appear to understand the links between intrusions, catastrophic misinterpretation, and safety behaviours, and are able to assimilate these elements into an overall formulation. The key to this technique is therefore drawing parallels to their eating disorder. One patient, for example, remarked even before concluding the model: "So, is my laxative use like a safety behaviour, then?". Another patient (see Figure 2) felt that the model was very clear, practical, and easy to follow. She particularly highlighted the processes (i.e., "the arrows") in the model as useful in understanding her illness, and felt more informed by understanding the relationships between different symptoms. She mentioned catastrophic misinterpretations (giving the example thought of "if I have a bit of cake, I will put on a lot of weight"), and how she would typically

1 cope with this through compulsive exercise. She reflected that exploring this
2 relationship in therapy helped her draw links between the thoughts and behaviours, and
3 also linked this with emotions (e.g., “If you feel stressed, it gets worse”). The
4 frequency of her compulsive exercise behaviour since decreased in treatment. Further
5 study of patient views on the use of such a model would add confidence to the
6 suggestions provided here.

7 With some individuals, it may be beneficial to focus on certain elements of the
8 model. For example, one patient was particularly interested in how she placed
9 significant emphasis on calorie-counting, and how perception of ‘minor’ differences in
10 the calorie content of food was (catastrophically) interpreted as having a definite impact
11 on weight. Steinglass *et al.* (2011) cite many pertinent examples of these elements,
12 such as fear (of food, ‘fatness’, weight, and so on) as akin to feared stimuli in anxiety
13 disorders. As in OCD, such thoughts are then acted on through ritualised and rigid
14 behaviours (see Pallister & Waller, 2008; Steinglass & Walsh, 2006), which serves to
15 maintain (or increase) both the thoughts and associated anxiety. Neutralising and safety
16 behaviours may thus increase other elements of pathology (see Figures), such as
17 increasing intrusions via thought suppression or by maintaining illness-related beliefs
18 through avoidance of perceive harm or exposure, preventing disconfirmation of these
19 beliefs. As Waller (2008) notes: “as with all such safety behaviours, the immediate
20 effect is anxiety reduction, but the longer-term effect is maintenance and elaboration of
21 the anxiety” (p. 169). Pallister and Waller (2008) provide useful examples of seeing
22 safety behaviours in eating disorders, whilst also pointing out some of the difficulties in
23 decreasing the use of these in patients. Although such illustrations can likely be
24 described to patients without reference to the complete formulation model outlined here,

1 it is argued that the model provides a thorough understanding of how safety behaviours
2 function, and what beliefs might underlie them. Thus, it is hoped that such a
3 formulation can help patients to see the impact of their safety behaviours, and appreciate
4 some of the cognitive factors that might maintain their pathology. Further study will be
5 required to identify techniques based on these suggestions (e.g., Steinglass *et al.*, 2012)
6 and to look at the therapeutic effectiveness of such techniques. Furthermore, as in
7 anxiety disorders (e.g., Sloan & Telch, 2002), safety behaviours might maintain fear,
8 and also negatively affect treatment outcome. Continued exposure to anxiety-provoking
9 triggers can provide an individual also with “evidence about the link between anxious
10 stimuli and catastrophic outcomes,” and developing new memories and associations
11 (Hildebrandt *et al.*, 2012, p. e9).

12 It is not yet known for which patients (if not all of those with AN) this model may
13 – or may not – be useful, and future work may help to elucidate this. Initial suggestions
14 from the current author are that those with a chronic presentation, or those who report
15 high egosyntonicity and / or limited insight into their illness may benefit from this
16 approach, which argues that a non-confrontational, Socratic approach is more likely to
17 foster a positive therapeutic relationship and open the possibility for subsequent
18 behavioural change. However, as noted above, AN is frequently an egosyntonic illness,
19 and the model may be helpful for many patients regardless of level of insight. It may
20 also be helpful in individuals who have both AN and OCD that are functionally linked,
21 as some ED treatment manuals suggest simultaneous treatment in such cases (see
22 Waller *et al.*, 2007, p. 250). A comprehensive formulation approach may help sufferers
23 appreciate the impacts of their behaviours, and Waller *et al.* (2007) describe how good
24 formulation is essentially a set of hypotheses that helps both patient and clinician gain

insight into the illness. It can help select appropriate interventions, but it may also be necessary to include elements of more complex formulation models, and the model presented here may be useful as a starting point to understanding the illness, or as an addition to an existing model. It should also be emphasised that what is argued here is an approach to understanding AN using a formulation that has elements of existing models (e.g., Waller *et al.*, 2007) but one that elaborates on key factors that are likely to maintain AN. AN and OCD are different psychiatric disorders, and, despite significant similarities, there are different traits that distinguish the two illnesses (e.g., Steinglass & Walsh, 2006; Sunday, Halmi, & Einhorn, 1995) and considering the two as distinct entities has a number of clinical advantages (Holden, 1990).

A Note on Low Weight and Dietary Restriction

The model here has been aimed at adults with AN, a disorder for which low body weight is of central importance. The exact contribution of weight loss needs to be understood, therefore, particularly as it has been suggested that anxiety predates weight loss in many cases, and often persists after restoration of normal weight (Strober *et al.*, 2007). It may be the case that starvation and / or weight loss are responsible for all, or many, of the OCD-type symptoms seen in AN, i.e., that obsessions and compulsions (particularly around food) can arise from malnourishment and food restriction (see Serpell *et al.*, 2002).

As Davis and Kaptein (2006) note, “It is quite probable that some of the psychological and behavioural symptoms of AN are either caused – or at least exacerbated – by malnutrition” (p. 210; also Steinglass & Walsh, 2006). However, there is also some evidence that OC beliefs in EDs are present regardless of the level of

emaciation (Roncero *et al.*, 2011) although further study is needed in this area. In the context of the formulation model suggested here, the influence of low weight and dietary restriction may be evident in different parts of the model, which will need to be explored on an individual level. For example, dietary restriction may operate as a safety behaviour (to prevent the feared outcome, e.g., uncontrolled weight gain) or as a precipitating event. It might also increase attentional bias (Placanica *et al.*, 2002), have a reciprocal relationship with food-related anxiety (Steinglass *et al.*, 2011) and, as Waller *et al.* (2007) remark, might bring about low mood. The effects of starvation have also been suggested to intensify the trait of cognitive inflexibility (Friederich & Herzog, 2011; Tchanturia & Hambrook, 2010), and thus normalising eating patterns and restoring weight should form part of treatment goals for such individuals.

Although it as yet appears that weight restoration is only a part of long-term recovery, the importance of addressing low weight and dietary restriction is central in AN, and should be considered in a psychological formulation of an individual sufferer; this may be prioritised for treatment in light of the seriousness or risk of such behaviours. Treatment framed around the formulation will then, therefore, consider how best to address symptoms such as weight loss and restriction, and it may be that the interventions vary according to the hypothesised role of these symptoms and behaviours within a given individual.

Limitations

The current paper aims to introduce a model of formulation and offers no systematic assessment of its efficacy or acceptability. Furthermore, numerous reviews of the ‘overlap’ between OCD and AN have been expertly written (e.g., Altman & Shankman,

2009; Pallister & Waller, 2008; Serpell *et al.*, 2002; Steinglass *et al.*, 2011), and the current paper does not offer a comprehensive overview of such issues. Thus, empirical studies may be needed in order to more fully examine how useful the proposed model is in clinical settings. The various similarities noted above between AN and OCD suggest that an OCD-specific formulation might be the most helpful, as it includes a number of elements thought to be similar across the two disorders. However, alternative models of anxiety may be just as helpful in understanding AN, as might current models of EDs, although it is also argued here that basic CBT principles (e.g., avoidance, exposure to feared stimuli, links with mood) can be included in the above formulation, whilst also including elements more in common with OCD; in this way, the model is seen as flexible but also comprehensive.

In order for treatment to work well, the principles of CBT must be adhered to, and the formulation presented here is no exception. Formulation of the complex cases that are so often seen in AN requires preparation and mindful planning on the part of the therapist. Some of the techniques, while seemingly benign, might provoke misinterpretation on the part of the patient, and may in fact be counterproductive to the aims of treatment. Novice therapists are advised to be judicious in the application of an OCD formulation model to AN and addressing elements such as safety behaviours. Thus, therapists practising with this model should feel confident and competent in working with such individuals, and seek appropriate supervision where necessary.

Conclusions

The current article adds to existing opinion regarding the advantages of conceptualising eating disorders as similar in many ways to anxiety disorders, such as obsessive-

compulsive disorder. It builds on existing reports (e.g., Steinglass *et al.*, 2011; 2012) by providing clinical techniques to approach formulation in anorexia nervosa, a notoriously difficult-to-treat illness with frequent anxiety symptoms and high relapse rates (Steinglass *et al.*, 2011; Steinhausen, 2002).

The formulation proposed in the current study seems acceptable to patients, and, from the author's experience, seems to help both patient and therapist understand eating behaviours in a number of ways that have not been fully explored or combined in existing models. Case series and controlled studies will be required to test the efficacy and utility of this approach.

Rather than being seen as alternative to existing models (e.g., Pallister & Waller, 2008; Steinglass *et al.*, 2011), the ideas advanced in the current article might be seen as a compliment. It is hoped that the ideas suggested will help therapists, particularly those who feel they have a limited understanding of anorexia nervosa (e.g., see Kaplan & Garfinkel, 1999; Yager, 1992). Similarly, the extent to which these ideas are used might also depend on patient factors. For example, a patient's level of engagement, or perhaps insight into their illness, might suggest how direct to be in formulating their ED behaviours; patients who present with high levels of denial (e.g., Vitousek *et al.*, 1998) might be helped to understand their condition by drawing parallels to OCD.

Summary

There is some degree of conceptual, genetic, symptom, and phenomenological overlap between anxiety disorders, such as OCD, and anorexia nervosa (AN). The article describes a popular formulation model of OCD which can be used by practitioners to understand some aspects of AN. This is seen as a compliment to existing formulation

models in eating disorders, and it is hoped that it can be used as a tool to both guide an intervention and also to help patients become more aware of some of the maintaining factors in their illness. Further empirical evaluation of the model is required.

Recommended follow-up reading

Pallister E, Waller G (2008). Anxiety in the eating disorders: Understanding the overlap. *Clinical Psychology Review* **28**, 366-386.

Steinglass J, Sysko R, Glasofer D, Albano AM, Simpson HB, Walsh BT (2011). Rationale for the application of Exposure and Response Prevention to the treatment of anorexia nervosa. *International Journal of Eating Disorders* **44**, 134-141.

Learning objectives

1. To understand the use of an OCD formulation in AN
2. To apply a model of anxiety disorders to an eating disorder (AN)
3. To gain insight into some of the potential maintaining mechanisms of AN
4. To reflect on the similarities between two distinct psychiatric disorders

Declaration of interests

None

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1 Figure 1: Cognitive model of OCD. As presented in Salkovskis (2007, p. 229).

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1 Figure 2: Cognitive model of OCD, applied to anorexia nervosa

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