Shared Neurological and Psychological Traits of Anorexia Nervosa and Obsessive-Compulsive Personality Disorder: The Implications in Treatment of Anorexia Nervosa

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Eating Disorders (ED) are psychological disorders characterized by harmful or abnormal eating habits in a person. An ED can consume many aspects of a patient’s life, and can even prove to be fatal in many cases. Of these, one particular type of ED – Anorexia Nervosa (AN) is commonly believed to have the highest mortality rate amongst all psychiatric illnesses. AN is characterized by a refusal to maintain normal body weight, intense fear of gaining weight or becoming fat, disturbance in the perception of the body, denial of the seriousness of being underweight, and amenorrhea in those to whom it applies. In addition to these characteristics, AN patients are often perfectionists, and often display signs of obsessive-compulsive behaviors. These obsessive-compulsive behaviors can cause marked distress and interfere with daily personal functioning, as well as healthy social functioning. This aspect of the disorder is very similar to what is experienced by patients suffering from Obsessive-Compulsive Personality Disorder (OCPD), a cluster C personality disorder according to the DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, 2000). OCPD is commonly characterized by symptoms like over-preoccupation with details, compulsive perfectionism, rigid behavior and stubbornness to the point of becoming socially inept. As such, elevated levels of perfectionism are common in both AN and OCPD, but the main difference is that in the OCPD context, these features are ego-dystonic – in conflict with a person’s ideal self or self image. In AN, on the other hand, the perfectionist and compulsive behaviors and thoughts are ego-syntonic and in sync with the person’s self image (Bastiani, Altemus, Pigott & Rubenstein, 1996; Holden, 1990). The presence of a number of similarities between the clinical and etiological features of AN and Obsessive-Compulsive Disorder (OCD), have led to the inclusion of AN as an OCD-spectrum disorder. Generally, inclusion in the OCD-spectrum takes into account the symptom profile, associated features, neurobiology, response to empirically-supported OCD treatments, and etiology of the

1 Cluster C disorders are those currently classified as ‘anxious or fearful disorders’, and also include Avoidant Personality Disorder, and Dependant Personality Disorder.
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disorder in question. It is important to touch upon OCD here to better understand the characteristics of AN, but OCD is not to be confused with OCPD which is an independent disorder.

The focus of this paper is to evaluate and highlight the implications of the shared psychological and neurological traits between AN and OCPD, on the treatment methods in AN. We know the two disorders are similar, and that they are considered to be part of the same spectrum of disorders. However, we wish to investigate the extent to which the similarities between AN and OCPD are relevant in the formulation of treatment methods in AN. Without treatment, an estimated 5 to 20 percent of patients with AN do not survive its complications (National Eating Disorders Association, 2006), and even with treatment, 2 to 3 percent do not survive. This therefore implies that whatever methods are being used to treat AN, they need to be improved. In this paper we will review current research on the different psychological and neurological factors found in AN patients, and discuss directions for future research in this area. We will then combine this with research in the area of OCPD, and find similarities in psychological and neurological factors between the two disorders that may help in creating an integrated, custom tailored treatment plan for different types of AN patients (perhaps sorted on the basis of broader personality type, or pre-morbid psychological factors).

Throughout the paper, we assume that psychological factors as well as neurological factors have a role to play in the causation of AN, although this, like in the case of many psychopathological disorders, is part of a larger unresolved etiological debate.

Current treatment of AN includes both medical and psychological treatment. AN is associated with many serious medical complications, and at times it is necessary to address these problems before embarking on further psychological treatment. Medical treatment usually targets problems such as electrolyte imbalances and dehydration. Once the patient is in relatively less medical danger, AN-targeted treatment proceeds. Weight restoration is
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implemented in the form of nutritional rehabilitation. This is a gradual process because due to long periods of starvation, initially the body is physically resistant to normal amounts of food and may not be able to retain or properly digest it. Also, there is the risk of “re-feeding syndrome” (Ehrlich, Querfeld & Pfeiffer, 2006) occurring. Alongside weight restoration, therapy techniques such as Cognitive-Behavioral Therapy (CBT) or the increasingly recognized Maudsley model of family therapy are used.

CBT is a goal oriented therapy in which certain cognitive or behavioral targets are identified and worked toward. These may include cognitive aspects of AN such as controlling automatic thoughts like “If I eat, I will be fat” and distorted perception of the body wherein the patients see themselves as fat even where they are not. CBT takes a scientific approach to the problem, and emphasizes on the operationalization of general symptoms into more specific, measurable units. While specific goals can be met and the success of treatment can be evaluated objectively through measurement, there may be a general neglect of keeping careful consideration of the patient’s emotional journey through the treatment process.

The Maudsley method, on the other hand, focuses on weight gain and healthy functioning of the body (and consequently, mind) as the first priority, and tries to implement this through focus on reward systems for healthy eating and so on. The Maudsley method also excludes speculations on what caused AN, and whose fault it was, and in this regard it is perhaps less stressful for the patient. However, due to the high responsibility and role of supportive parents and family, this method may not be appropriate for adults, or in all family situations.

Drugs are also administered for AN, including two main types: Antidepressants and Atypical Antipsychotics. Antidepressants are drugs of a class of selective serotonin re-uptake inhibitors (SSRI), which focus, as the name suggests, on the activity of the neurotransmitter Serotonin. It is thought that general depression exacerbates the negative body image feelings and obsession with body weight seen in AN. Hence this is one line of treatment used, in
which the use of antidepressants is hoped to also alleviate specific AN symptoms. The second type of drug administered is the newly recognized class of Atypical Antipsychotic drugs. These focus on the activity of the neurotransmitter Dopamine, and are thought to have fewer side effects than the traditional class of antipsychotic drugs popularly administered in more severe disorders like schizophrenia and so on.

In spite of these commonly used treatments, which have a number of advantages and positive aspects, the prognosis for AN remains relatively poor. Currently, only an estimated 60% of AN patients completely recover. About 30% have some persistent symptoms after 5 years and 20% continue to have chronic persistent symptoms after 5 years. Part of the reason AN is especially hard to treat, is because the behavioral characteristics associated with it are ego-syntonic, as mentioned before. In practical terms this means that although AN patients are fully aware of their obsessive-compulsive, perfectionist behavior, and about their constant preoccupation with fatness, thinness, and food, they do not see this as abnormal in any way, or conflicting with what they want to do or feel. They also deny the importance of having normal body weight compared to their height and weight and are thus overall very resistant to treatment or intervention, perhaps making it less effective than in ego-dystonic disorders in which patients are responsive to help and to change.

*Distinguishing between the obsessions and compulsions in AN, OCD and OCPD*

OCPD, one of the most frequently diagnosed personality disorders across community (Ekselius, Tillfors, Furmark & Fredrikson, 2001) and clinical (Stuart, Pfohl, Battaglia, Bellodi, Grove & Cadoret, 1998) samples, is defined as “a chronic maladaptive pattern of excessive perfectionism, preoccupation with orderliness and detail, and need for control over one’s environment that leads to significant distress or impairment, particularly in areas of interpersonal functioning” (DSM-IV-TR; American Psychiatric Association (APA), 2000). Diagnosis is made upon the confirmation of at least four of the following eight criteria:
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preoccupation with details, perfectionism, excessive devotion to work, hyper-morality, inability to discard worn or useless items, inability to delegate tasks, miserliness, and rigidity. In essence, OCPD is seen as a pervasive pattern of inflexibility which causes distress to the person, and often leads to difficulties in personal relationships, with others feeling the pressure of dealing with someone who is ‘high maintenance’.

Due to the rigidity, perfectionism and inflexibility displayed by AN patients, relationships between EDs and obsessive-compulsive symptoms have long been recognized. Palmer & Jones (1939) were first to describe AN as a manifestation of OCD. However, a core issue in the debate about EDs/OCD relationships concerns the types of symptoms which qualify for individuals with EDs to be diagnosed with OCD. For OCD-like symptoms in AN, it is necessary to note that if they are food-related (i.e. Cutting up of food, particular exercise regime etc.) then it is not classified as an OCD, but simply OCD-like symptoms/traits within the ED. However, if a patient with AN has additional non food-related obsessions and compulsions, he/she is said to have an ED which is comorbid with OCD. Furthermore, unlike the obsessions and compulsions present in OCD, repetitive thoughts and behaviours with regards to food and weight in ED patients are not necessarily seen as intrusive (Mazure, Halmi, Sunday & Romano, 1994) or senseless (Halmi et al., 2002).

Co-occurrence of OCPD in patients with AN

Sub-threshold Personality Disorders (PD) are often present in ED patients. For example, Borderline Personality Disorder (BPD) traits such as impulsiveness are frequently observed in Bulimia Nervosa (BN) patients, while AN patients tend to present with the perfectionist trait typical of OCPD. Anorectic individuals are often described as being rigid, inflexible, stubborn and obstinate; terms that are similar to the ones with which we describe OCPD characteristics. In spite of these common notions, Halmi (2004) points out that AN patients rarely seem to meet the diagnostic criteria for OCPD. In one study the rate was found to be
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only 6% in restricting type anorectics. The same study also found however that 18% of the AN patients were diagnosed with sub-threshold OCPD, even though they fell short of a complete OCPD diagnosis (Braun, Sunday & Halmi, 1994). It is important to bear in mind however that current research does not indicate a causal relationship between PD and ED, merely co-occurrence of PD when other mental disorders like ED are present.

Wonderlich et al., (1990) and Strober, Freeman, Lampert & Diamond (2007) found a significantly higher prevalence of generalized anxiety and OCPD (among other anxiety related disorders), in relatives of AN probands2, than in relatives of never-ill probands. The authors concluded that perhaps one factor that makes a person vulnerable to AN, is the intra-familial transmission of a tendency towards anxiety in one form or the other. They also suggested future research into the neuro-circuitry of fear and anxiety as a framework in which to interpret pre-morbid symptoms of anxiety in EDs. If a “propensity for anxiety” (Strober et al., 2007) is what is transmitted through genes, then anti-anxiety medication would probably be more effective than antidepressants, in the treatment of AN. Furthermore these results suggest that anxiety could be one of the main factors in AN, and not merely incidental to the disorder. In that case, more focus on anti-anxiety techniques being taught as part of therapy and counseling could prove effective as a method of treatment.

Although we cannot determine for sure the causal factors of PD based on evidence available so far, we do know that PD patients benefit from a variety of interventions, and what these interventions are. At present, the most successful strategy has been for PD patients to remain in long-term contact with one or more healthcare professionals, and seek emergency back-up measures in case of crises. However, needless to say, this plan is difficult to maintain due to the persistent, long-lasting nature of PDs, due to which long-term commitments are required.

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2 “Proband” here, refers to the human subject being tested
on the parts of the healthcare provider as well as patient. Moreover, even with regular patient-
doctor contact, pronounced traits never completely disappear or get replaced.

Alternative strategies include the use of short-term, intermittent, and long-term psychotherapy. Cognitive therapy and behavior modification techniques or drug treatments alongside social support from the immediate community can also be helpful.

The treatment process for patients with AN follows a similar trajectory as that of those with PDs. Long term commitments are required for treatment to be effective and to avoid relapse of behavioral symptoms, and all too often an AN patient will drop out of treatment before fully recovering.

The common and pervasive problems that PD and AN patients experience, including cognitive and perceptual organization, anxiety, and inhibition, warrants the need for more research with regards to applying PD treatment methods to AN treatment. Additionally, as a majority of patients with clinically significant PDs show a varied mixture of antisocial, borderline, histrionic and narcissistic PDs, this could also be the case in co-occurring sub-threshold PDs in AN patient. For this reason there is a need to determine the personality type of the AN patient, and using this information to design a customized, more personally effective treatment plan.

Neurological studies in AN

The neurotransmitter serotonin (5-HT) is thought to mediate feelings of satiety and hunger (Leibowitz, 1990) and thus theoretically, 5-HT disturbances are thought to contribute to the dysregulation of appetite. Anxious, obsessional behaviours and extremes of impulse control are also linked to these disturbances. One way to test for 5-HT functioning is to measure the concentration of the neurotransmitter in the cerebrospinal fluid (CSF). Underweight AN patients have been found to have a significantly reduced concentration of 5-hydroxyindoleacetic acid (5-HIAA) in the CSF, compared to healthy control women (Kaye et
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al., 1984). Interestingly, follow up studies of the same patients after their weight was restored revealed higher levels of 5-HIAA than normal controls. In a future study, it was found that this significantly higher level of 5-HIAA persists in AN patients up to 6 months of weight being restored to normal (Kaye et al., 1991). Although these results are interesting in pointing towards the involvement of 5-HIAA in AN, the reliability of these studies is brought into question by Gerner et al. (1984), who found no differences in CSF levels of 5-HIAA between patients with anorexia and normal control subjects.

Kaye (2007) also studied the possible role of neuropeptides in AN. It was found that brain 5-HT neurotransmission appears to be affected by the choice of diet. Tryptophan (TRP) is an essential amino acid only available in the diet and participates in the chemical reaction that produces 5-HT. Therefore the proportion of carbohydrate and protein consumed can affect brain 5-HT release and appetite regulation. What this is essentially means is that even though patients with AN may show a reduction in 5-HIAA as seen in Kaye (2007), this may be (a) caused by a typical sort of diet seen in AN, and (b) lead to further loss of appetite, or, on the other extreme – binge eating. Binge eating can also be associated with lack of impulse control, and binge eating behaviours cause a lot of stress and anxiety in AN patients, as the strict control usually sought in one’s diet is suddenly lost. The “hard work” of managing ones weight and food intake carefully, may be wasted due to this dysregulation of appetite based in 5-HIAA, leading to even more obsessional behaviour, or eating/weight related anxiety.

It was also found that when AN patients ate food, or were exposed to food, they had activated temporal regions, and show signs of increased anxiety. This suggests that amygdale activation is related with anxiety provocation. If the amygdale is the storehouse of the emotional aspects and values of our experiences, then this finding confirms what one might have predicted considering how deeply affected AN patients are by food and eating, and how prominent thoughts and emotions related to food are in their lives.
The findings from Kaye (2007) indicate that individuals with AN have 5-HT dysregulation within the brain regions that make up the limbic circuits. It appears that these alterations tend to be present in the ill state and persist after recovery. However, these inferences are speculative. It is important to keep in mind that no receptor works in isolation in the brain. These findings might reflect the function within limbic circuits, but the specific cause of AN remains debatable. Kaye (2007) therefore hypothesizes that a trait-related disturbance of 5-HT occurs before the onset of AN, and contributes to the pre-morbid symptoms of anxiety and inhibition often seen in AN patients, through the dysregulation of emotion and reward pathways.

Apart from serotonin, the catecholamine group of neurotransmitters (norepinephrine, epinephrine, dopamine amongst others) have also been implicated in appetite and behaviour disturbances in both human and animal studies (Morley & Blundell, 1988). The concentration of Homovanillic Acid (HVA), a major metabolite of Dopamine in humans, can be measured in the CSF to give an indication of Dopamine activity. When testing AN patients, reduced CSF-HVA concentrations (Kaye et al., 1984) were found, compared to normal control women. In order to control for the casual effects of pathological eating, all the patients with AN were tested after 6 months of normal eating and normal or almost normal body weight. Post-hoc testing also showed that CSF-HVA was significantly lower in the restricting group of anorectics, than in the bulimic group.

Dopamine has been associated with motivation levels and feelings of reward (Salamone, 1996; Blum et al., 1995), pleasure (Lambert et al., 2003) and novelty seeking behaviour (Sohlber & Strober, 1994) and AN restrictors show reductions in both these areas, and “seem to find little in life rewarding apart from losing weight” (Kaye et al., 1999). This study suggests that disturbed dopamine activity in anorectic individuals could indeed play a role in AN, and particularly, in the restricting subtype. This is not to say that dopamine irregularities cause AN
necessarily, but the authors suggested that it may make a person more vulnerable or prone to restricting type AN, judging from particular qualities like anhedonia, lack of motivations and perceived rewards, as well as lack of novelty seeking behaviour – which all link to irregular dopamine levels. It is also possible that dopamine levels are severely affected in the face of malnutrition, starvation, and other pathological features of AN, and that the dysregulation in the neurotransmitter takes more than 6 months to normalise. More research is needed in the area to come to a conclusive answer about the cause and effect relationship here.

In an earlier study done on the genetic basis of the dopamine receptor gene, it was found that there is no significant association between AN and the gene. (Bruins-slot et al., 1998) This reminds us that the difference in neurotransmitter activity may be caused by environmental factors, or due to co-morbidity with some other disorder/pathology, and not a genetic difference in the individuals at the time of birth.

Similarities between OCPD and AN

In restricting type AN we find that patients diet obsessively, and are strictly bound by their own rules to restrict the amount of food they eat. In OCPD, we find a similar type of behaviour whereby a person is obsessive about their morality, perfectionism, and so on. Ironically, in both cases, what starts out as giving the patient a sense of control and a comfort level and satisfaction with things going exactly as planned, often ends up in many aspects of the patient’s life spiralling out of control, including workplace or academic success, reputation, and personal relationships. In “The Golden Cage”, Bruch wrote about self-starvation in AN as being a struggle for autonomy and control. Psychoanalytical theorists explain the obsession in OCPD in terms of inner conflicts and a fundamental need for security and control (Lang, 2007), and have pointed out that a need for control is the most significant clinical aspect of the personality (Mallinger, 1984). The similarities are numerous between the psychological characteristics of the two disorders, and as mentioned before, we
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actually see co-occurrence of OCPD or sub-threshold OCPD in AN patients, or at the family level.

Apart from general character descriptions, there seem to be a number of specific cognitive similarities between AN and OCPD. A study on set shifting in anorexia nervosa (Tchanturia, Morris, Anderluh, Collier, Nikolaou & Treasure, 2004) examined set shifting before and after weight gain, in full recovery and relationship to childhood and adult OCPD traits. Inflexibility or difficulty adapting to change, as well as the preoccupation with details are one type of trait used to classify personality type. (Strober, 1980) The typical individual with OCPD shows these traits and it reflects also in their cognitive processing of tasks. The Set Shifting task requires the patient to shift between mental representations or response sets, and performance on them indicates mental flexibility. The study found impairments in an AN group, as compared to healthy controls. Furthermore, no difference was seen in the mental flexibility level of the AN group, and a separate group of AN-recovered individuals. In addition, no improvement was found in the set shifting task performance of the AN group in a follow up study after weight restoration and recovery. These results suggest that mental inflexibility as a trait is independent of the level of nutrition and illness in AN patients, and that the OCPD trait of mental inflexibility exists even before the onset of AN, perhaps even contributing to the black and white views of “fat” and “thin” seen in AN. An alternative theory accounting for poor flexibility in AN, is that the onset of anorexia nervosa in adolescence may interrupt the process of pruning and maturation within the frontal lobe. (Hutenlocher, 1979), and neurological studies of OCPD patients can further confirm or disconfirm this sort of biological basis for mental inflexibility.

Other traits in AN have also been studied in detail. A longitudinal study on AN cognition, the Wechsler Adult Intelligence Scale - Revised (WAIS-R) was administered to a group of individuals 10 years after onset of the disorder. It was found that the AN group as a whole
showed poor results on the ‘object assembly’ subtest of the WAIS-R, indicating weak central coherence with a tendency to focus on details at the expense of configural information (Gillberg et al., 2007). This cognitive pattern may be one of the bases for AN individuals’ obsession with details, a trait long established in OCPD.

Additional research on personality traits shows that a high level of perfectionism (a trait used in classification and diagnosis of OCPD) is a risk factor for AN (Karwautz et al., 2001). In fact perfectionism and preoccupation with orderliness have been found to persist after recovery (Matsunaga et al., 2000; Strinivasagam et al., 1995). The perfectionism trait is found to exist in increased frequency amongst first degree relatives of people with AN (Lilenfeld et al., 1998).

The above studies have shown that traits common to individuals with OCPD are clearly present in individuals with AN, in both individuals who have recovered, as well as those still underweight or in treatment. This implies that these traits such as rigidity and perfectionism (seen in OCPD) might be what predisposes a person to AN, and whether he/she has recovered is unimportant. This suggests that many characteristics of anorectic individuals take the form of ingrained personality features as in the case of OCPD, and hence prove harder to treat than if the psychopathology is found only after onset of AN.

However, with regards to studies done with so called “recovered” anorectics, it is possible that not all the individuals in the recovered-group have psychologically recovered. Weight restoration is usually taken as an indicator for recovery but this is by no means complete in accounting for psychological factors like depression, over critical body image, lack of self worth or negative and obsessive thoughts found in AN. Consequently it is also not indicative of normal neurotransmitter activity in the brain, and thus the real status of the recovered patients should be questioned. Arguably, the state of recovery of each individual is on a
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different place on the recovery spectrum and this has to be kept in mind and groups for statistical analysis needs to be formed accordingly.

Another similarity in the psychological characteristics of AN and OCPD, is that patients with both illnesses deny having anything wrong with them and often decline help and treatment at first. This is largely due to the fact that the obsessions and compulsions in both OCPD and AN are seemingly “goal-oriented”. An anorexic obsesses about food and weight and exercises compulsively with a goal in mind: weight-loss. A person with OCPD may be obsessive about his/her work and compulsively re-do or rectify things with a goal in mind: to bring the piece of work to perfection no matter what it takes.

Status of current treatments in AN

In order to constructively think about the future of AN treatment, we must first understand what works, what does not, and why. Anorexia Nervosa is commonly a problem with adolescents, which explains the recent developments of family based therapy for such individuals. Amongst all other forms of treatment for this disorder, family therapy is the most extensively researched (Fairburn, 2005; Vitousek & Gray, 2005). The best studied approach, however, is the specific form of family therapy known as the Maudsley model (Dare & Eisler, 1997; Lock & le Grange, 2005), mentioned earlier. Applied to adolescent patients, intervention involves around 10-20 family sessions spaced over the time period of 6-12 months. The recommended ‘conjoint’ format specifies that all family members be seen together, and that parents take active control of their anorectic child’s eating and weight in the first phase of the treatment. A published manual also outlines the entire treatment procedures in detail (Lock, le Grange, Agras & Dare, 2001).

As a means of preventing post-hospitalization weight loss in anorexic patients of different sub-groups, the Maudsley model was first tested in a study done by Russel, Szmukler, Dare, and Eisler (1987). Results displayed that in the subset of younger patients with a more recent
onset of the illness, conjoint family therapy yielded an impressive recovery rate of 90% being symptom-free at 5 years, and was said to be ‘far more effective than a dynamically oriented individual approach’ (Eisler et al., 1997; Russel et al., 1987). However, neither treatment appeared beneficial in patients who were either at an older age at onset or had a longer history of the illness.

Two conclusions of this study are supported by subsequent research. First, the higher-than-expected rate of recovery in adolescents with anorexia following family therapy has also been apparent in clinical series (le Grange, Binford & Loeb, 2005) and randomized controlled trials (e.g., Eisler et al., 2000; le Grange, Eisler, Dare & Russell, 1992; Lock, Agras, Bryson & Kraemer, 2005). In both controlled trials and naturalistic catchment-area studies, outcomes for young adolescents are much more encouraging than the aggregate 50% recovery rate cited for all patients with anorexia nervosa (e.g., Nilsson & Hagglof, 2005; Steinhausen, 2002). Such favorable results however, may merely reflect the characteristics of the samples to which the approach has been delivered to (Fairburn, 2005; Vitousek & Gray, 2005).

Second, in line with the Russell et al. (1987) study, symptom duration is a strong predictor of the outcome to family therapy. In a trial of family therapy administered to adolescents with a relatively recent onset, patients who attained a positive outcome had been symptomatic for just 8 months prior to the start of treatment, as opposed to 16 months for those who attained intermediate or poor outcomes (Eisler et al., 2000).

However, the third notable finding of the Russell et al. (1987) study (that the Maudsley model was much more effective than individual treatment for adolescent patients) has little support, and according to Fairburn (2005) there is little basis at present for the widespread belief that family therapy is particularly efficacious for adolescents with the illness.

Although the Maudsley model seems to yield some good results, a limitation is that it applies largely to patients of a younger age. Use of the Maudsley model in an adult sample with an
average duration of 6 years produced minimal clinical improvements in the majority of patients (Dare, Eisler, Russell, Treasure & Dodge, 2001). In addition, according to the National Institute for Clinical Excellence (NICE, 2004) in the UK which has conducted comprehensive and rigorous evaluations of the available evidence on the treatment of eating disorders, family interventions should be offered to younger patients—although not necessarily in place of individual therapy.

Although few clinicians would disagree to the inclusion of parents in the treatment of adolescents, optimal means of doing so have yet to be discovered. It is not clear why the published manual strongly recommends the conjoint model despite findings that the ‘separate’ model (where the anorectic individual and her parents attend different sessions) produced a favorable trend (Eisler et al., 2000; le Grange et al., 1992). Two randomized controlled trials have compared the conjoint format to a “separated” version and in both trials, the “separated” version was more preferred.

In addition, le Grange (2005) reviewed three studies on family-based treatment for AN. The first study compared outpatient family-based therapy and individual supportive therapy following inpatient weight restoration, the second compared the Maudsley conjoint (CFT) and separated family therapy (SFT), and the third sought to compare behavioral systems family therapy (BSFT) based on Maudsley treatment to Ego-Oriented Individual Treatment (EOIT). Results were favorable towards Maudsley family-based treatment, with a significantly better outcome in the first study, inpatient treatment not required at all in one of the studies (Le Grange, 1992) and a significantly rapid treatment response to BSFT in the third study. Weaknesses and limitations, however, include the fact that FBT is perhaps disadvantageous for families with high levels of hostility or criticism towards the patient, as well as the fact that the very engagement of family therapy is difficult.
Overall, the results for Maudsley based family therapy are encouraging, however perhaps only to the younger demographic of AN patients. More research needs to be done in the area of patients at an older age at onset of AN, as well as a longer history of the illness. Also, the efficacy of such a collaborative form of therapy depends very much on the dynamics of the family, implying that there is no clear-cut form of family therapy.

*The use of antidepressants – Fluoxetine*

Apart from family based therapies, studies have been done to evaluate and better understand the scope of drug-based therapy for AN. Our earlier discussion has established that dysregulated serotonin (5-HT) function may be implicated in the causes of AN. Hence in theory, the use of antidepressants should be helpful in treating AN. Earlier generations of antidepressants (Lacey & Crisp, 1980) had insignificant effect on weight gain, possibly due to the non-specific action of these drugs. Selective Serotonin Reuptake Inhibitors (SSRIs) work on specific serotonin receptors. Hence, we now need to confirm the efficacy of SSRIs – specifically fluoxetine – as a treatment method in AN.

In one double-blind, placebo-controlled study, Attia, Haiman, Walsh & Flater (1998) set out to determine if fluoxetine was associated with greater weight gain and improved psychological functioning in AN patients, when combined with a structured inpatient program. Their results showed that fluoxetine was well tolerated, probably due to the relatively few side effects that the drug is associated with. Patients in both groups showed statistically significant improvement on virtually all measures of anorectic behaviour, depression, body shape perception, and eating attitudes. However, there was no significant difference overall in this improvement between the placebo and treatment groups. In the placebo and treatment groups with co-morbid depression, again, no significant difference in improvement was seen between the groups.
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While the use of double-blinds and placebo controls added strength to this study, it must be noted that the sample size was small, at 31 patients. Also, it was not established if the dose of fluoxetine used was adequate. The efficacy of fluoxetine as the only form of treatment was also not ascertained as the medication was provided in addition to the inpatient program.

In a later stronger study, a similar double-blind, placebo-controlled design was used to determine first, whether fluoxetine would improve outcome and reduce relapse after weight restoration in restricting-type AN outpatients and second, whether fluoxetine was useful because of its effects on core eating disorder symptoms, obsessionality, or depression (Kaye et al., 2001). This was the first controlled study done to determine the efficacy of fluoxetine as the sole form of treatment. Previous studies were either single-blind or open studies.

It was found that only subjects on fluoxetine had significant increases in weight and reduction in core eating disorder symptoms, obsessive thoughts, and depressed and anxious mood, suggesting that fluoxetine is an effective form of treatment after all. However, this success should not be taken for granted. Some subjects were started on the medication trial during the re-feeding phase and before complete weight restoration. The subjects are therefore not statistically equal to begin with. There was also a small sample size of 35, with a 30% dropout rate from the study. Furthermore, no standardized psychological treatment was administered during the trial. There was also a high refusal rate in invited participants, lowering the generalizability to the AN population, and perhaps restricting the results to a motivated subset of anorectics.

One of the largest controlled medication trials conducted to date in AN, was done by Walsh, Kaplan, Attia et al., (2006), and included 93 patients as part of the trial. The objective of the study was to determine, in a large sample, the ability of fluoxetine to promote recovery and prolong time-to-relapse among patients with Anorexia Nervosa following weight restoration.
Participants were randomly assigned to receive fluoxetine or placebo and were treated up to a year as outpatients in double-blind fashion. All patients also received individual CBT.

The main outcome measures of the study were 1) Time-to-relapse; and 2) The proportion of patients successfully completing a year of treatment. The results showed no significant difference between fluoxetine and placebo in time-to-relapse and therefore, the study failed to demonstrate any benefit from fluoxetine in the treatment of patients with anorexia nervosa following weight restoration. It was suggested by the authors that “therapeutic efforts would be better devoted to psychological and behavioural interventions for which there is some, albeit modest, evidence of efficacy”.

It appears then, that the prescription of antidepressants has questionable effectiveness in the treatment of AN. In non weight-restored patients, this could be attributed to the possibility that malnutrition may neutralize the therapeutic effects of SSRIs. Malnutrition can induce changes in the 5-HT1A receptor and extracellular 5-HT concentrations, decreasing the efficacy of SSRI usage in AN patients. Antidepressants are thus not a complete cure for AN in themselves though they may help alleviate certain symptoms.

Physicians may also wish to reconsider the prescription of fluoxetine based on its potential for abuse. Fluoxetine is known to cause a loss of appetite as a side effect and AN patients may abuse this side effect to lose more weight. Such a case has indeed been recorded in the past (Wilcox, 1987). Even if the drug is not abused, it is possible that the recovery rate may be hampered by the appetite-suppressing effects of fluoxetine.

Fluoxetine therefore does not appear to be a merited form of treatment in itself as of current research into the drug.

*The use of Atypical Antipsychotics - Olanzapine*

As mentioned before, another line of treatments is to use atypical antipsychotic drugs, which target the dopamine levels in the brain. To recapitulate an earlier discussion, dopamine
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dysregulation is currently considered a possible problem area in AN. The dopaminergic system modulates the motivation to feed (Volkow, Wang, Fowler et al., 2002), underpinned by restraint and emotionality linked to the dorsal stratum (Volkow, Wang, Maynard et al., 2003). Clinical findings have implicated the dopaminergic system in patients with restricting-type AN (Barry & Klavans, 1976; Kaye, Frank & McConaha, 1999).

Olanzapine is an atypical antipsychotic that encompasses serotonergic and dopaminergic receptor affinities. The following studies have sought to investigate the effectiveness of antipsychotics, more specifically olanzapine, in the treatment of AN.

The first study (Bosanac, Norman, Burrows & Beumont, 2004) consisted of four trials that were either open label or single-blinded trials. The aim of these trials were to review the evolving utility and efficacy of atypical antipsychotic medications in the management of AN. The first trial found a significant decline in eating disorder symptoms, a significant decline in positive and negative symptoms, and a significant decline in depressive symptoms. Reduced anxiety before and during meals, reduced difficulty in eating, and reduced frequency of obsessive thoughts about body image and fear about being fat, were found in the second trial.

In the trial that compared olanzapine and chlorpromazine, there was significantly greater reduction in ruminative thinking on mental activities subscale of Padua Inventory (which evaluates a person’s tendency to worry and doubt (obsessions) and perform behaviors intended to ward off those doubts (compulsions), in four main areas: (a) contamination, (b) checking, (c) impaired control over mental activities, and (d) worries about losing control over one’s behaviors) for the olanzapine group. There was also a clinically but not statistically significant decrease in global and subscale score of EDI-2 (Eating Disorder Inventory; specifically drive for thinness, body dissatisfaction, and bulimia). A last trial that compared the use of olanzapine in a test group and control group found no significant difference in weight gain.
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Olanzapine does appear to have an overall benefit as reflected in the results of this study, and indeed in each trial. However, we must keep in mind some shortcomings of this study. These were all open label and single-blinded trials which may have led to inherent selection and observer bias. Reliance on non-validated, non-standardized, self-report instruments is also a weakness. Furthermore, no replication studies were done, there were no controls for motivation or readiness to recover, and there was no fixed criteria to define outcome or remission.

A second double-blinded study (Brambilla, Garcia, Fassino et al., 2007) was done to investigate the psychobiological effects of olanzapine therapy in AN. They found a significant increase in Body Mass Index (BMI) in both groups. However, as found in Bosanac et al. (2004), there was no significant difference in the increase between the two groups. The obsessive-compulsive symptoms were also significantly reduced, which again confirmed the findings in Bosanac et al. (2004).

Another, more recent double-blind, placebo-controlled study done on the use of olanzapine therapy identified two main research aims: (1) To evaluate effect of olanzapine on weight gain in patients with AN, and (2) To evaluate the anti-obsessive, anti-compulsive, anti-anxiety, and antidepressant properties of olanzapine (Bissada, Tasca, Barber & Bradwejn, 2008). Adherence to the treatment was established in all the patients through the use of urine tests that were collected and analyzed at an independent laboratory. It was found that no serious adverse side effects were experienced. There were significantly greater increases in BMI for the olanzapine group, which contradicts the findings in Bosanac et al. (2004) and Brambilla et al. (2007). There was also no significant difference in decrease in depression and no significant difference in decrease in anxiety found in this study, which again contradicts the findings of Bosanac et al. (2004). The significantly greater decrease in obsessions in the Olanzapine group does, however, support what was found in both the prior studies.
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It is worth noting that this was the largest study done so far and the findings were robust, with two different analytical methods used (hierarchical linear modelling, and survival analysis). However, the generalizability of these findings to the AN population is reduced because more than half the patients originally approached were not willing to participate in the study. This is similar to the fluoxetine studies, where it was discussed that motivation may be a confounding factor that affected the validity of the results.

One must also consider the other possible impacts of olanzapine therapy. The effect of weight gain is exactly what anorectic patients wish to avoid, thus it is possible that a marked level of distress may be experienced by AN patients. Other possible cardiac, gastrointestinal and neurological side effects caused by malnutrition, as well as the low body mass of AN patients, also has to be factored in before olanzapine therapy is considered.

*Implications of OCPD research on AN treatment*

The precise causes of AN have yet to be found, and it appears unlikely that an exclusive precipitating factor will be established in the near future. At best, it is only possible to note that anorexia nervosa, or eating disorders in general, are the result of a complex interaction between biological, psychological and social influences.

Research in these areas has definitely come a long way. The hypothesized involvement of serotonergic and dopaminergic dysregulation has led to the use of SSRIs and atypical antipsychotics respectively, in the treatment of AN. This, as we have evaluated earlier, has proved to have limited success.

Having recognized the need for treatment on the psychological front, CBT and FBT are currently the more popularly used forms of treatment. The Maudsley model has proved to be particularly efficacious when applied to younger anorectics with adequate familial support. But for the older anorectics, those who have a longer history of illness, and those with less than ideal family support, the prognosis remains relatively poor.
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Having identified a number of shared personality and neurological traits in AN and OCPD, we feel that this is a viable option that can be looked into when dealing with the abovementioned subset of anorectics. Interpersonal therapy has been the treatment of choice when dealing with PD patients. Individuals with AN tend to withdraw from previous friendships and other peer-relationships as they become increasingly entrenched in the disorder. Relationships with the family also deteriorate. The dysfunctional interpersonal relationships suggest that therapy tailored to the individual’s personality-type could possibly prove to be of use. This of course, would imply the need for more research to be done in the area of management of the different personality types.
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