Obsessive compulsive disorder:

Core interventions in the treatment of obsessive compulsive disorder and body dysmorphic disorder

National Clinical Practice Guideline Number ___

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The people with obsessive-compulsive disorder (OCD), body dysmorphic disorder (BDD) and carers who contributed testimonies to the chapter on the experience of OCD and BDD.

1 Introduction

This guideline has been developed to advise on the identification, treatment and management of obsessive-compulsive disorder (OCD) and body dysmorphic disorder (BDD).[1] The guideline recommendations have been developed by a multidisciplinary team of healthcare professionals, people with OCD, a carer and guideline methodologists after careful consideration of the best available evidence. It is intended that the guideline will be useful to clinicians and service commissioners in providing and planning high quality care for those with OCD and BDD while also emphasising the importance of the experience of care for people with OCD, BDD, and carers.

This guideline addresses aspects of service provision, psychological and pharmacological approaches for those with OCD and BDD from the age of 8 upwards. Although the evidence base is rapidly expanding, there are a number of major gaps and future revisions of this guideline will incorporate new scientific evidence as it develops. The guideline makes a number of research recommendations specifically to address these gaps in the evidence base. In the meantime, we hope that the guideline will assist clinicians, people with these disorders and their carers by identifying the merits of particular treatment approaches where the evidence from research and clinical experience exists.

1.1 National guidelines

1.1.1 What are clinical practice guidelines?

Clinical practice guidelines are 'systematically developed statements that assist clinicians and patients in making decisions about appropriate treatment for specific conditions' (Mann, 1996). They are derived from the best available research evidence, using predetermined and systematic methods to identify and evaluate the evidence relating to the specific condition in question. Where evidence is lacking, the guidelines incorporate statements and recommendations based upon the consensus statements developed by the guideline development group.

Clinical guidelines are intended to improve the process and outcomes of health care in a number of different ways. Clinical guidelines can:

[1] On the whole the term obsessive-compulsive disorder (OCD) will be used throughout this guideline as a generic term to cover body dysmorphic disorder (BDD) unless BDD is meant specifically.

- Provide up-to-date evidence-based recommendations for the management of conditions and disorders by healthcare professionals
- Be used as the basis to set standards to assess the practice of healthcare professionals
- Form the basis for education and training of healthcare professionals
- Assist patients and carers in making informed decisions about their treatment and care
- Improve communication between healthcare professionals, patients and carers
- Help identify priority areas for further research.

1.1.2 Uses and limitations of clinical guidelines

Guidelines are not a substitute for professional knowledge and clinical judgment. Guidelines can be limited in their usefulness and applicability by a number of different factors: the availability of high quality research evidence, the quality of the methodology used in the development of the guideline, the generalisability of research findings and the uniqueness of individuals with OCD.

Although the quality of research in OCD and BDD is variable, the methodology used here reflects current international understanding on the appropriate practice for guideline development (AGREE: Appraisal of Guidelines for Research and Evaluation Instrument; www.agreecollaboration.org), ensuring the collection and selection of the best research evidence available, and the systematic generation of treatment recommendations applicable to the majority of people with these disorders and situations. However, there will always be some people and situations for which clinical guideline recommendations are not readily applicable. This guideline does not, therefore, override the individual responsibility of healthcare professionals to make appropriate decisions in the circumstances of the individual, in consultation with the person with OCD and/or carer.

In addition to the clinical evidence, cost-effectiveness information, where available, is taken into account in the generation of statements and recommendations of the clinical guidelines. While national guidelines are concerned with clinical and cost effectiveness, issues of affordability and implementation costs are to be determined by the NHS.

In using guidelines, it is important to remember that the absence of empirical evidence for the effectiveness of a particular intervention is not the same as evidence for ineffectiveness. In addition, of particular relevance in mental health, evidence-based treatments are often delivered within the context of an overall treatment programme including a range of activities, the purpose of which may be to help engage the person with OCD, and to provide an appropriate context for the delivery of specific interventions. It is important to maintain and enhance the service context in which these interventions are delivered; otherwise the specific benefits of effective interventions will be lost. Indeed, the importance of organising care in order to support and encourage a good therapeutic relationship is at times as important as the specific treatments offered.

1.1.3 Why develop national guidelines?

The National Institute for Clinical Excellence (NICE) was established as a Special Health Authority for England and Wales in 1999, with a remit to provide a single source of authoritative and reliable guidance for patients, professionals and the public. NICE guidance aims to improve standards of care, to diminish unacceptable variations in the provision and quality of care across the NHS and to ensure that the health service is patient-centred. All guidance is developed in a transparent and collaborative manner using the best available evidence and involving all relevant stakeholders.

NICE generates guidance in a number of different ways, two of which are relevant here. First, national guidance is produced by the Technology Appraisal Committee to give robust advice about a particular treatment, intervention, procedure or other health technology. Second, NICE commissions the production of national clinical practice guidelines focused upon the overall treatment and management of a specific condition. To enable this latter development, NICE has established seven National Collaborating Centres in conjunction with a range of professional organisations involved in healthcare.

1.1.4 The National Collaborating Centre for Mental Health

This guideline has been commissioned by NICE and developed within the National Collaborating Centre for Mental Health (NCCMH). The NCCMH is a collaboration of the professional organisations involved in the field of mental health, national patient and carer organisations, a number of academic institutions and NICE. The NCCMH is funded by NICE and is led by a partnership between the Royal College of Psychiatrists' research unit (College Research Unit – CRU) and the British Psychological Society's equivalent unit (Centre for Outcomes Research and Effectiveness – CORE). Members of the NCCMH reference group come from the following organisations:

- Royal College of Psychiatrists (RCPsych)
- British Psychological Society (BPS)

- Royal College of Nursing (RCN)
- Social Care Institute of Excellence (SCIE)
- College of Occupational Therapists (COT), now replaced by the Clinical Effectiveness Forum for the Allied Health Professions (CEFAHP)
- Royal College of General Practitioners (RCGP)
- Royal Pharmaceutical Society (RPS)
- Rethink Severe Mental Illness
- Manic Depression Fellowship (MDF)
- Mind
- Centre for Evidence Based Mental Health (CEBMH)
- Centre for the Economics of Mental Health (CEMH)
- Institute of Psychiatry (IoP).

The NCCMH reference group provides advice on a full range of issues relating to the development of guidelines, including the membership of experts, professionals, patients and carers within guideline development groups.

1.1.5 From national guidelines to local protocols

Once a national guideline has been published and disseminated, local healthcare groups will be expected to produce a plan and identify resources for implementation, along with appropriate timetables. Subsequently, a multidisciplinary group involving commissioners of health care, primary care and specialist mental healthcare professionals, patients and carers should undertake the translation of the implementation plan into local protocols taking into account both the recommendations set out in this guideline and the priorities set in the National Service Framework for Mental Health and related documentation. The nature and pace of the local plan will reflect local health care needs and the nature of existing services; full implementation may take a considerable time, especially where substantial training needs are identified.

1.1.6 Auditing the implementation of guidelines

This guideline identifies key areas of clinical practice and service delivery for local and national audit. Although the generation of audit standards is an important and necessary step in the implementation of this guidance, a more broadly based implementation strategy will be developed. Nevertheless, it should be noted that the Healthcare Commission will monitor the extent to which Primary Care Trusts (PCTs), trusts responsible for mental health and social care and Health Authorities have implemented these guidelines.

1.2 The national obsessive-compulsive disorder guideline

1.2.1 Who has developed this guideline?

The Guideline Development Group (GDG) was convened by the NCCMH and supported by funding from NICE. The GDG included people with OCD and a carer, and professionals from psychiatry, clinical psychology, child psychology, nursing, general practice.

Staff from the NCCMH provided leadership and support throughout the process of guideline development, undertaking systematic searches, information retrieval, appraisal and systematic review of the evidence. Members of the GDG received training in the process of guideline development from NCCMH staff and people with OCD received training and support from the NICE Patient Involvement Unit. The NICE Guidelines Technical Adviser provided advice and assistance regarding aspects of the guideline development process.

All GDG members made formal declarations of interest at the outset, which were updated at every GDG meeting. The GDG met a total of 18 times throughout the process of guideline development. The GDG met as a whole, but key topics were led by a national expert in the relevant topic. The GDG was supported by the NCCMH technical team, with additional expert advice from special advisers added where necessary. The group oversaw the production and synthesis of research evidence before presentation. All statements and recommendations in this guideline have been generated and agreed by the whole GDG.

1.2.2 For whom is this guideline intended?

This guideline will be relevant for people with a diagnosis of obsessive-compulsive disorder (OCD) or body dysmorphic disorder (BDD) aged 8 years and over.

The guideline covers the care provided by primary, community, secondary and other healthcare professionals who have direct contact with, and make decisions concerning the care of adults, children and young people with OCD and BDD.

The guideline will also be relevant to the work, but will not cover the practice, of those in:

- occupational health services
- social services
- the independent sector.

The experience of OCD or BDD can affect the whole family and often the community. The guideline recognises the role of both in the treatment and support of people with these conditions.

1.2.3 Specific aims of this guideline

The guideline makes recommendations for the identification, treatment and management of OCD and BDD. Specifically, it aims to:

- Evaluate the role of specific psychological interventions in the treatment and management of OCD and BDD
- Evaluate the physical management and role of specific pharmacological agents in the treatment of OCD and BDD
- Evaluate the role of other biological interventions in the management of OCD and BDD
- Integrate the above to provide best practice advice on the care of individuals with a diagnosis of OCD or BDD throughout the course of the disorder
- Promote the implementation of best clinical practice through the development of recommendations tailored to the requirements of the NHS in England and Wales.

2 Obsessive-compulsive disorder

This guideline is concerned with the identification, treatment and management of obsessive-compulsive disorder as defined in the 10th Edition of the International Classification of Diseases (ICD 10) (World Health Organization, 1992).

2.1 OCD

2.1.1 Symptoms, presentation, and patterns of illness

Obsessive Compulsive Disorder (OCD) is characterised by the presence of either obsessions or compulsions, but commonly both. An obsession is defined as an unwanted intrusive thought, image or urge, which repeatedly enters the person's mind. Obsessions are distressing but are acknowledged as originating in the person's mind, and not imposed by an outside agency. They are usually regarded by the individual as unreasonable or excessive. A minority are regarded as having over-valued ideas (Veale, 2002) and, rarely, delusions. The person usually tries to resist an obsession, but in chronic cases this may be to a very minor degree or not at all. The most common obsessions are listed in Table 1. The percentages refer to the frequency in a survey of 431 individuals with OCD (Foa et al, 1995).

> [**Awaiting copyright permission for Table 1.** Please refer to article for entire table: Foa, E. B., Kozak, M. J., Goodman, W. K. *et al* (1995) DSM-IV Field trial: Obsessive Compulsive Disorder. *American Journal of Psychiatry*, 152, 990-996. The three common obsessions are contamination from dirt, germs, etc., fear of harm, excessive order or symmetry,]

Table 1 – Common obsessions in OCD

Unwanted intrusive thoughts, images or urges are almost universal in the general population and their content is usually indistinguishable from clinical obsessions (Rachman & de Silva, 1978). Examples include having the urge to push someone onto the underground tracks or a thought that the cooker has been left on. According to current psychological models, the difference between a normal intrusive thought and an obsessional thought is the meaning that OCD patients attach to the occurrence and/or content of the intrusions. Individuals with OCD tend to believe that intrusive thoughts and urges are dangerous or immoral and that they are able to prevent harm occurring either to their self or a vulnerable person (Salkovskis et al, 1995).

Compulsions are repetitive behaviours or mental acts that the person feels driven to perform. A compulsion can either be overt and observable by

others, such as checking that a door is locked, or a covert mental act that cannot be observed as in repeating a certain phrase in the mind. Covert compulsions are generally more difficult to resist or monitor than overt ones as they are can be performed anywhere without others knowing and easier to perform. A compulsion is not in itself pleasurable which differentiates it from impulsive acts, such as shopping or skin-picking, that are associated with immediate gratification.

The term "ritual" is synonymous with compulsion but usually refers to behaviours that other people can see. The term "rumination" refers to prolonged thinking that is experienced as uncontrollable around and around the same subject. It includes both intrusive thoughts, often in the form of doubts or questions, and repeated attempts to find an answer. In this way it covers both the obsession (the doubts or question) and the accompanying compulsive thinking which attempt to answer the question. Just as with obsessions, there are many types of compulsions. The most common ones are listed in Table 2, as listed in (Foa et al, 1995).

> [**Awaiting copyright permission for Table 2.** Please refer to article for the entire table: Foa, E. B., Kozak, M. J., Goodman, W. K. *et al* (1995) DSM-IV Field trial: Obsessive Compulsive Disorder. *American Journal of Psychiatry*, 152, 990-996. The four most frequent compulsions are checking, cleaning, repeated acts, and mental compulsions such as special words repeated in a usual pattern.]

Table 2 Common compulsions in OCD

The most frequent presentations are checking and cleaning and are the most easily recognised as on a continuum with everyday behaviour. Repeating compulsions can be of any type of behaviour and may be motivated by a broad range of concerns or fears. In some cases ordering, symmetry and exactness may appear to have a tic-like character to them, but in other cases they are clearly related to perceived threat. Hoarding consists of the acquisition and/or failure to discard objects. In some cases this may be excessive quantities of particular materials that may have some apparent value related to perceived threat or may result from the inability to dispose of materials because of the threat associated with it. However in many cases, hoarding consists of accumulating material that appears to have little or no value. In the former case it is perhaps more likely that other OCD symptoms may be found. In the latter case, there is debate as to the extent to which it may share the key features of OCD (Steketee & Frost, 2003).

Mental compulsions by definition are not observable by others and people may be less likely to be able to describe these cognitive acts. Other terms are also used. "Neutralizing" resembles a mental compulsion but is not identical. Both are usually anxiety reducing. However, neutralising is not necessarily

stereotypic as a compulsive urge but has the aim of "undoing" the perceived harm. By comparison, compulsions are often experienced as involuntary, repetitive and are seldom resisted. The term "safety seeking behaviours" is also used in the literature to refer to any actions in a feared situation that aim to prevent feared catastrophes and reduce harm (Salkovskis, 1985) and will therefore include compulsions and neutralising behaviours. Examples of other safety seeking behaviours include various mental activities such as trying to be sure of the accuracy of one's memory or trying to suppress or distract oneself from unacceptable thoughts which may reduce anxiety in the short term but leads to a paradoxical enhancement of the frequency of the thought in a rebound manner.

The aim of a compulsion or neutralizing behaviour is thus to reduce harm or feel "comfortable" or "just right" and is an additional criterion used for terminating a compulsion. Someone without OCD finishes hand-washing when they can see that their hands are clean. However, someone with OCD and a fear of contamination finishes not only when they can see that their hands are clean but also when they feel "clean", "comfortable" or "just right". Although avoidance behaviour is not part of the definition of OCD, it is an integral part of the disorder and is most commonly seen in fears of contamination. Typically an individual may avoid touching a wide range of objects or activities to prevent the obsession and distress from occurring.

2.1.2 Diagnosis

The diagnostic criteria for the two main international classification systems, ICD-10 and DSM-IV, are virtually identical and must include the presence of either obsessions or compulsions. The patient must acknowledge that the obsessional thoughts, impulses, or images are a product of their mind and are not imposed by outside person or influences. At least one obsession or compulsion must be acknowledged as excessive or unreasonable (although patients holding obsessions with delusional intensity are reported). Furthermore, the obsessions or compulsions must cause marked distress, or significantly interfere with the patient's occupational and/or social functioning, usually by wasting time. The exclusion clause is that the obsessions or compulsions are not best explained by another mental disorder. Insight, that is the ability to recognize the senselessness of the obsessions, has traditionally believed to be a key feature of OCD. However, there is growing recognition that the level of insight is highly variable (Lochner & Stein, 2003). Thus some people with OCD may show stable but low levels of insight, others may show insight when not confronted with the feared situation, but lose this insight when their anxiety is high in situations associated with their obsessive fears. There is some evidence showing that insight into the condition is poor among people with particular forms of the disorder, especially hoarding (Lochner & Stein, 2003).

2.1.3 Physical and social consequences

The severity of OCD differs markedly from one person to another. Individuals may be able to hide their OCD often from their own family. However, OCD may have a major negative impact on social relationships leading to frequent family and marital discord or dissatisfaction, separation or divorce (Koran, 2000) . Most studies have found lower rates of marriage among people with OCD than in the general population. It also interferes with leisure activities (Antony et al, 1998). OCD often interferes with a person's ability to study or work, leading to diminished educational and/or occupational attainment, and unemployment (Koran, 2000; Leon et al, 1995). The social cost, that is the person's inability to fully function within society, has been estimated as $5.9 billion in 1990, or 70.4% of OCD's total economic cost (Dupont et al, 1995). OCD is ranked by the World Health Organization in the Top 10 of the most handicapping illnesses by lost income and decreased quality of life (World Health Organization, 2001).

OCD can severely interfere with daily activities and family life, and family members may report distress (Amir et al, 2000; Magliano et al, 1996). It can be particularly difficult for families when the person with OCD has poor insight into the disorder. In these cases the person will have difficulty recognising that their concerns are excessive, that they may have OCD, or indeed that they may need help. There may also be a financial burden on the family (Chakrabarti et al, 1993). Although people with OCD often succeed in not letting their symptoms interfere with family responsibilities, there is some limited evidence that parental OCD can sometimes affect children (e.g. Black et al, 1998; Black et al, 2003). The mechanisms are not yet known, but in one study, children of a parent with OCD were more likely to have emotional, social, and behavioural problems in comparison to children of parents without OCD, (Black et al, 2003). When children have OCD, parent-child relationships also are changed and there is some evidence that parents and children may behave differently from children with other disorders, particularly around problem-solving and independence (Barrett et al, 2002). Finally, in some rare cases, the symptoms of a parent with OCD may directly impact on the well-being of family members, for example, when concerns about contamination can occasionally lead to extreme hygiene measures that are applied to family members.

2.1.4 Course and prognosis

For some people, the symptom type will remain unchanged, but for others there may be changes over time (Rettew et al, 1992; Skoog & Skoog, 1999). For some, the change may remain within the symptom type, for example different types of checking, especially in the short term (Mataix-Cols et al, 2002b). OCD may follow an acute, episodic or chronic course. In one of the largest follow-up studies, Skoog & Skoog (1999) conducted a 40-year prospective study and reported that approximately 60% of people with OCD

displayed signs of general improvement within 10 years of illness, increasing to 80% by the end of the study. However, only 20% achieved full remission even after almost 50 years of illness; 60% continue to experience significant symptoms; 10% displayed no improvement; and 10% had deteriorated. A fifth of those patients, who had displayed an early, sustained improvement subsequently relapsed, even after 20 years without symptoms. This suggests that early recovery does not eliminate the possibility of very late relapse. Intermittent, episodic disease was more common during the early stage of illness and predicted a more favourable outcome, whereas chronic illness predominated in later years. Worse outcome was predicted by early age of onset, particularly in males, experiencing obsessions and compulsions or magical thinking, poor social adjustment and early chronic course.

2.1.5 Epidemiology of OCD

According to some studies, OCD is the fourth most common mental disorder after depression, alcohol and substance abuse, and social phobia with a lifetime prevalence in community surveys of about 2-3% (Robins et al, 1984). However the instruments used have been criticised and may have over-diagnosed OCD so that the true prevalence may be somewhat lower (Stein et al, 1997b). The mean age of onset is in late adolescence for men and early twenties for women. However, it may take individuals between 10-15 years to seek professional help.

2.1.6 OCD in children and adolescents

In this guideline, OCD and its management is reviewed across all ages, from the youngest age at which the diagnosis might be reliably made (arguably 4-5yrs), through the life-span, into old age. There are two main reasons for this. First, adults with OCD often report that they experienced their first symptoms in childhood (Rasmussen & Eisen, 1994). Second, the disorder is remarkably similar in children, adolescents and adults, and responds to the same treatments. Although there are aspects of the disorder in young people which need special consideration, the main symptoms, clinical understanding of OCD, and key strategies for management have a great deal in common at all ages.

OCD was thought to be uncommon in young people, but reliable population surveys have revealed an OCD prevalence of about 1% in young people (Heyman et al, 2001; Valleni-Basile et al, 1994). It frequently goes undetected and if untreated can not only cause marked psychological distress, but can also disrupt social, educational and emotional development, leading to significant disability. There is some evidence that early diagnosis and intervention improves outcome (Leonard et al, 1993), so the general public as well as health professionals need to be alert to childhood onset of OCD.

2.2 BDD

2.2.1 Symptoms, presentation, and patterns of illness

Body Dysmorphic Disorder (BDD) is characterised by a preoccupation with an imagined defect in one's appearance or, in the case of a slight physical anomaly, the person's concern is markedly excessive. The most common preoccupations concern the skin, hair, nose, eyes, eyelids, mouth, lips, jaw, and chin. However, any part of the body may be involved and the preoccupation is frequently focused on several body parts simultaneously (Phillips et al, 1993). Complaints typically involve perceived or slight flaws on the face, asymmetrical or disproportionate body features, thinning hair, acne, wrinkles, scars, vascular markings, and pallor, or ruddiness of complexion. Sometimes the complaint is extremely vague or amounts to no more than a general perception of ugliness. BDD is characterised by time consuming behaviours such as mirror-gazing, comparing particular features with those of others, excessive camouflaging tactics to hide the defect, skin-picking, and reassurance-seeking. There is usually avoidance of social situations and of intimacy. Alternatively such situations are endured with the use of alcohol, illegal substances or safety-seeking behaviours similar to social phobia.

2.2.2 Diagnosis

According to DSMI-IV, to fulfil diagnostic criteria for Body Dysmorphic Disorder (BDD), the person must also be significantly distressed or handicapped in his or her occupational and social functioning (American Psychiatric Association, 1994). In ICD-10, BDD is not classified as a separate diagnosis and is subsumed under Hypochondriacal Disorder. The beliefs about one's appearance (for example that "my skin is wrinkled and puffy") may be held with poor insight (when it is regarded as an overvalued idea) or no insight (when it is termed delusional). DSM-IV classifies BDD on the strength of such beliefs according to whether there is an additional diagnosis of a Delusional Disorder. In ICD 10, if the beliefs are considered delusional, then a patient would receive an alternative diagnosis of "Other Persistent Delusional Disorder" instead of Hypochondriacal Disorder. There is frequent comorbidity in BDD especially with depression, social phobia and obsessive compulsive disorder (OCD) (Neziroglu et al, 1996; Phillips & Diaz, 1997; Veale et al, 1996a).

Amputee Identity Disorder (AID) is often confused with BDD. It is a term used to describe individuals who desire one or more digits or limbs to be amputated (Furth et al, 2000; Smith & Fisher, 2003). Some patients may hasten amputation (e.g. chainsaw wound) or carry out self-amputation (for example on a railway line). Although such individuals are preoccupied with becoming disabled, they do not believe (as in BDD) that their limbs are defective or wish to alter their limb cosmetically. They feel that one or more

limbs are not part of their "self" (a form of reverse "phantom limb") and that amputation will lead to becoming more able-bodied.

2.2.3 Impairment and disability

People with BDD are significantly handicapped with high rates of unemployment, social isolation, are sometimes housebound, hospitalised and make frequent suicide attempts (Phillips et al, 1993; Phillips et al, 1995; Veale et al, 1996a). People with BDD have a poorer quality of life and in one study reported lower scores on standard scale of quality of life than those reported for other psychiatric outpatients (Phillips, 2000).

2.2.4 Course and prognosis

No systematic research has been done on the course and prognosis in BDD. However, milder symptoms in adolescence may resolve over time but those with moderate to severe symptoms tend to follow a chronic course with increasing comorbidity, unsatisfactory attempts at altering their feature or suicide. For some people, the preoccupation with a particular feature will remain unchanged, but for others there may be changes over time.

2.2.5 Epidemiology of BDD

BDD is rarely included in any catchment area surveys of psychiatric morbidity. The data on the prevalence of BDD in the community is generally lacking. However, one study in primary care in Italy found a prevalence of 0.7% (Faravelli et al, 1997) and one in the community of women age 37-44 found a prevalence of 0.7% (Otto et al, 2001). De Waal *et al* (2004) however, found no cases of BDD in survey of somatoform disorders in 1024 patients. However there was no specific screening for BDD and the study excluded anyone under the age of 25 where the majority of cases of BDD may occur. For example, Bohne *et al* (2002) found the prevalence of BDD in college students (average age 21) from a screening questionnaire to be 5.3%. (Faravelli et al, 1997; Otto et al, 2001) reported the prevalence to be 5% in young adults (Bohne et al, 2002). Surveys of people with BDD attending a psychiatric clinic tend to show an equal sex incidence (Neziroglu & Yaryura-Tobias, 1993a; Phillips et al, 1993; Phillips & Diaz, 1997; Veale et al, 1996a). It is also possible that in the community while more women are affected overall, a greater proportion experience milder symptoms. These surveys also suggest that people with BDD are often single or separated.

2.2.6 BDD in children and adolescents

BDD may also present in children, and may lead to symptoms of school refusal and suicidal plans. Albertini & Phillips (1999) describe a series of 33 children and adolescents with BDD and reported that bodily preoccupations most often focused on the skin (61%) and hair (55%). They all exhibited

similar behaviours to adults with BDD such as camouflaging, comparing their self with others, and mirror-gazing. Social impairment was almost universal and the majority reported impairment in academic functioning due to their BDD. The group displayed significant disturbances with 39% having had psychiatric hospitalisation and 21% reporting suicide ideation or attempted suicide.

2.3 The aetiology and maintenance of OCD

OCD is a very heterogeneous disorder in its manifestations and there have been many attempts to categorise sub-groups based usually on the types of obsessions and compulsions. However, behaviours that may appear similar may be conducted for different reasons when the people conducting them are asked. For example, Rachman (1994) describes several distinct subgroups of people who conduct washing rituals according to their reported motivations to wash. Such heterogeneity makes the study of aetiology difficult. A range of factors have been identified as contributing to the expression of OCD, but any one of the factors involved may not be involved in a specific case. It is more likely that for any given person, a number of contributing factors are involved.

2.3.1 Biological factors

As with most mental health problems, the cause of OCD is not known. There is increasing research evidence for the involvement of biological factors in this disorder, although it is highly responsive to psychological intervention (Stein, 2000; Stein, 2002).

There have been a number of family studies of OCD looking at evidence for genetic patterns. A recent meta-analytic review by Hettema et al (2001) reported that a person with OCD is 4 times more likely to have another family member with OCD than a person who does not have OCD (OR = 4.0 (95% CI=2.2–7.1). Genetic and family studies have shown that OCD appears to be related to tic disorders and Tourette's Syndrome. Among children with OCD, many children have tics as well (reference missing). About 50% of individuals with Tourette's Syndrome also have OCD (reference missing). Some authors have discussed how the recurring themes and stereotyped nature of OCD rituals and intrusive thoughts make them seem like 'tics of the mind' (Rapoport & Inoff-Germain, 2000). However, Pauls et al (1995) sum up findings from their study in the following way :"Some cases are familial and related to tic disorders, some cases are familial and unrelated to tics, and in other cases there appears to be no family history of either obsessive-compulsive disorder or tics" (p. 76) .

Brain imaging studies have consistently demonstrated differing blood flow patterns among people with OCD compared with controls, and that cortical and basal ganglia regions are most strongly implicated (Saxena et al, 1998). However, a recent meta-analysis found that differences between people with OCD and healthy controls were found consistently only in the orbital gyrus and the head of the caudate nucleus (Whiteside et al, 2004). Treatment with either medication or CBT is associated with a reversal of the functional neuroimaging findings to the pattern found in control individuals (Schwartz et al, 1996). The neurochemical correlates of these differences are not known, but the specificity of effectiveness of one class of medication, SSRIs, in the treatment of OCD suggests that serotonin is an important neurotransmitter.

A further recent finding implicating the basal ganglia as a key brain region in OCD, is the discovery that a sub-group of children with OCD may have the disorder triggered by infections (Dale & Heyman, 2002; Swedo et al, 1998). Streptococcal infections, a common cause of sore throat, trigger an immune response, which in some individuals generates antibodies that cross-react with basal ganglia. This mechanism may explain the subgroup of children in whom OCD develops after a streptococcal infection, and worsens with recurrent infections. However, a recent study found no link between subsequent infections and exacerbation of symptoms (Luo et al, 2004).

There is some suggestion that very early onset OCD, probably before puberty, may be a little different to later onset OCD, although of course there are adults with OCD whose illness started when they were young. Some studies suggest that the juvenile-onset form seems more strongly associated with a positive family history for OCD and may be more associated with tics (Geller et al, 1998).

2.3.2 Adverse life events and difficulties

Several studies report major life events in the period preceding the onset of OCD (Gothelf et al, 2004; Khanna et al, 1988b). This does not mean that the events are in themselves causal, but rather that among people who may be biologically or psychologically predisposed to OCD, a life event can be a triggering factor. The type of event is probably less important than how it is experienced and even positive events can, under some circumstances, be associated with the onset of OCD. In the same way that life events may contribute to onset of OCD, there may be an increase in OCD symptoms as stress levels rise in response to ongoing life. Finally, in some cases, the content of the obsessions may reflect the themes of the life events in some people. Once again, this does not mean that the event causes OCD; rather it means that for some people, the specific content of their obsessions can be influenced by things that happen in their lives (Rheaume et al, 1998)

2.3.3 Family factors

There is no doubt that families get caught up in OCD in different ways and to different degrees. There may be greater impact on family life in OCD than in other anxiety disorders (Lochner et al, 2003). As with other mental disorders, there is evidence of the impact of OCD on the family in a number of ways such as worry, the burden of care, and distress at their limited ability to help the person with OCD (Shafran et al, 1995). In OCD there is also evidence that in some cases, family members get involved in rituals, often at significant cost to themselves in terms of effort, time, and upset (Calvocoressi et al, 1995; Calvocoressi et al, 1999). Likewise they may respond to repeated questions and requests for reassurance. The goal of such involvement is often to help the person with OCD or to reduce distress, and indeed it may alleviate distress or help the person function better in the short term. However, it is believed that this type of involvement is ultimately unhelpful. This does not mean that the family member is causing OCD, but rather they are caught up in maintaining the disorder in some ways. Likewise, family tension and disruption can be a source of stress that can contribute to onset or exacerbation of the disorder (Chambless et al, 2001). Although there is speculation that some types of childhood experience may, in association with other factors such as parental overprotectiveness, predispose an individual to OCD, there is no evidence that families play a direct causal role and it is difficult to disentangle changes in parental behaviour in response to a child's OCD from behaviours that may contribute to its development (Barrett et al, 2002; Salkovskis et al, 1999; Turgeon et al, 2002; Vogel et al, 1997).

2.3.4 Socio-cultural factors

Studies from different cultures reveal similar prevalence rates and a surprising consistency in the content and forms of obsessions and compulsions (Horwath & Weissman, 2000). While the exact symptoms of OCD may reflect socio-cultural factors, there is no consistent evidence that any particular factor has any causal role. Thus, socio-cultural factors may shape the expression of OCD (Fontenelle et al, 2004). In this way obsessions and compulsions of a religious nature will reflect the religious views of the individual and perhaps of the society, but are likely to be based on a particularly rigid or extreme set of beliefs or practice that is not widely shared by other members of the community (Raphael et al, 1996; Tek & Ulug, 2001). Preoccupations with contamination may also reflect the society's view of what is clean and what is not, but it is also possible that the individual's concerns are of a highly idiosyncratic nature. Although it is not believed that socio-cultural factors play causal role, they must be taken into account and dealt with sensitively when professionals encounter a person. Without understanding the specific socio-cultural context of the individual, it is almost impossible to determine what is typical culturally sanctioned behaviour and what is excessive due to the influence of OCD. Given socio-cultural differences when talking about such themes as hygiene, sexuality, blasphemy,

etc., great sensitivity must be exercised to enable full disclosure of what may be perceived by the individual as private, embarrassing, or shameful.

2.3.5 Psychological factors

Current psychological models of OCD propose that the way in which people interpret their thoughts is an important maintaining factor (Rachman, 1997; Salkovskis et al, 1995). There is evidence that people with OCD do indeed hold stronger beliefs about the importance of their thoughts and responsibility for harm to others than people without OCD (Frost & Steketee, 2002). Likewise, there is some evidence that people with OCD may hold more perfectionistic beliefs. However, there is as yet little evidence to suggest that these beliefs play a causal role in the aetiology of OCD although there is accumulating evidence that a range of beliefs including responsibility and the over-importance of thoughts may play a maintaining role.

2.3.6 Aetiology and maintenance of BDD

Hypothesised risk factors include genetic factors, temperament and childhood adversity such as teasing or bullying, increased aesthetic sensitivity, a history of dermatological or other physical stigmata (Veale, 2004). However, there is virtually no research that has systematically studied risk factors in BDD with other psychiatric disorders. A cognitive behavioural model has been described for the maintenance of symptoms of BDD (Veale, 2004).

2.4 The treatment and management in the NHS

2.4.1 Children & Young People – OCD

The same types of psychological and drug treatments are generally thought to be effective in both children and adults. However, the numbers of scientific, controlled studies are fewer in children and young people than in adults. Although it may often be reasonable to look at studies done in adults and assume that similar results will be found in children, there are important developmental differences which need considering both for psychological and drug treatments.

2.4.1.1 Pharmacological treatment

Care and thought is needed when using drugs in any illness. The child or young person with OCD and their parents or carers together with the prescriber need to decide whether the potential benefits outweigh any possible risks. Medicines are used in childhood OCD but it is important to bear in mind that the long-term effects of drugs on the immature brain of the child are little understood. Moreover, when prescribing drugs for children with OCD it is essential to use a drug and dose appropriate to the child's age

and size, generally starting with a very low dose and increasing gradually. Drugs most commonly used for children and young people with OCD are the same as those used for adults, including the SSRIs, and clomipramine is also used for young people.

Current published evidence suggests that SSRIs are effective in treating children and young people with OCD. However, with depression SSRIs can cause significant adverse reactions, including increased suicidal thoughts and self-harm, although they may be safer when combined with psychological treatments. The UK regulatory authority has contraindicated all SSRIs in paediatric depressive illness, except fluoxetine. Although the risk associated with the use of SSRIs in children and young people with OCD is unclear, appropriate caution should be observed, especially in the presence of comorbid depression. The only SSRIs licensed for use in children and young people with OCD are fluvoxamine and sertraline.

2.4.1.2 Psychological interventions

The main psychological treatment for OCD is cognitive behavioural therapy (CBT), and the principles of treatment are much the same for children and adults, although account needs to be taken of the developmental changes in cognitive and linguistic abilities and functions that occur with increasing age. CBT is a well-validated psychotherapeutic technique in a number of different settings (National Collaborating Centre for Mental Health, 2005; National Collaborating Centre for Mental Health, 2003; National Collaborating Centre for Mental Health, 2004) developed from experimental psychology principles initially with behavioural strategies. More recent developments have included interventions that target the young person's beliefs and the way they interpret situations. Published protocols for use in children, such as that of March and Mulle (1998) are generally based on "exposure" (facing up to the feared stimulus) and "response-prevention" (resisting the urge to carry out a ritual in these circumstances). Cognitive therapy protocols which tackle underlying beliefs about connections between thoughts and behaviours, are also being evaluated, although these principles form a component of most CBT approaches.

The therapist working with a child or young person should be aware of the impact of OCD on families, and how families can help with treatment. Although some children can be quite secretive and conceal their rituals, many involve an adult in their rituals. Working with children and young people will often require effective liaison with the child's school or other agencies involved in the child's life. Sometimes other forms of psychological work may be used in the overall management of OCD (e.g. family therapy, play therapy etc.), although these are usually offered as an adjunct to CBT. Although this is a little researched area, most practitioners with experience in managing children and young people with OCD find that it is essential to involve families closely in the treatment.

2.4.2 Adults – OCD

2.4.2.1 Pharmacological treatment

The outlook for OCD was dramatically improved by the discovery of effective drug treatments from the early 1980s (Marks et al, 1980; Montgomery, 1980). Intensive pharmacological investigation has consistently demonstrated that OCD responds selectively to drugs that act as potent inhibitors of the synaptic reuptake of serotonin (serotonin reuptake inhibitors, SRIs) (Montgomery et al, 2001; Zohar & Judge, 1996). Currently, this includes the tricyclic drug clomipramine, which stands apart from other tricyclics because of its more potent serotonergic actions, and the more highly selective serotonin reuptake inhibitors (SSRIs) citalopram, fluoxetine, fluvoxamine, paroxetine and sertraline (in alphabetical order). The finding that these drugs are effective even when depression is rigorously excluded in the reference population implies a specific antiobsessional effect. Drugs lacking these properties such as the standard tricyclic antidepressants and monoamine oxidase inhibitors have been found to be ineffective in randomised controlled trials (Ananth et al, 1981; Insel et al, 1983; Jenike et al, 1997; Volavka et al, 1985). Antipsychotics have not been found effective when given on their own, but may have a role as agents of augmentation in cases where the response to SRI is poor or incomplete. The selectivity of the pharmacological response for serotonergic agents distinguishes OCD from depression and other anxiety disorders where a wider range of treatments appear effective, and implicates serotonin in the treatment effect.

Obsessive compulsive disorder responds to drug treatment in a characteristically slow, gradual way and improvements can take many weeks and months to develop. It is usually recommended that patients remain at the lowest effective dose levels for several weeks and are then reassessed before gradually increasing up to the maximum licensed doses according to observed efficacy and tolerability. As with most drug treatments, there is always the possibility of adverse effects with the particular type of effect depending on the drug. In many case the adverse effects decrease over time and people tolerate the drugs well. In other cases switching drugs is indicated as experiencing adverse effects for one does not mean adverse effects on another. Unfortunately there is currently no way of knowing who will respond to which drug, or who will report adverse effects. In this way, it may appear to be "trial and error" as several attempts may be required to find a medication, which on average is of equal efficacy and tolerability as the others, but which has less predictable effects for a given person. Although there is little evidence to inform long-term outcome, the studies that have been performed suggest that SRIs remain effective for as long as they are continued, and continuation protects against relapse. There is no convincing evidence supporting dose-reduction in the longer-term.

2.4.2.2 Psychological treatment

CBT is the most widely used psychological treatment for OCD in adults (Roth & Fonagy, 2004). The main CBT interventions that have been used in the treatment of OCD are exposure and response prevention (ERP) (Foa & Kozak, 1996; Marks, 1997), cognitive therapy (Freeston et al, 1996; van Oppen & Arntz, 1994), and a combination of ERP and cognitive therapy (Kobak et al, 1998; Roth & Fonagy, 2004). ERP and cognitive therapy have different theoretical underpinnings but may be used together in a coherent package. However, it is uncertain whether either treatment is superior to the other, or indeed whether combining these interventions confers any added benefit (Abramowitz, 1997).

A variety of modes of treatment programmes have been developed, including a programme which can be accessed by a touch- telephone system (Baer & Greist, 1997) that can be accessed 24 hours a day, telephone treatment guided by a therapist (Lovell et al, 2000; Taylor et al, 2003), and bibliotherapy (using self-help books) (Fritzler et al, 1997) which may be offered with brief support sessions (Lovell et al, In Press).

Whatever CBT intervention is used the key principles remain the same, and involve first establishing a good therapeutic alliance based on a working partnership between patient and therapist. Then follow a credible and clear rationale, a treatment focus on the here and now, the use of explicit agreed and operationally defined treatment strategies and the use of collaboratively therapeutic strategies between client and therapist (Rachman, 2003; Salkovskis et al, 1999; Steketee, 1993).

Since the first studies showing the efficacy of exposure and response prevention procedures in the 1970s, , many of the studies that have followed have examined the relative efficacy of variations in the mode of delivery of CBT rather than comparison to control conditions. Variants include the length and intensity of treatment, the use of additional components such as cognitive interventions, the value of involving the family in treatment, and employing different formats such as individual or group treatments. In some cases groups are used in the interest of cost effectiveness, in other cases it is believed that particular features of the group therapy experience can enhance the treatment itself.

Variation in therapist time for treating OCD is considerable, ranging from fewer than ten hours over to 50 hours over 10-20 sessions. Consensus guidelines have suggested between 13-20 weekly sessions (March et al, 1997). However, there is little evidence to demonstrate the optimal number of session's required and further research in this area is necessary. Further,

many mental health services in the UK that offer CBT have long waiting lists, precluding easy access to effective interventions (Lovell & Richards, 2000).

Despite the good response rate of CBT with OCD it is important to remember that a significant proportion of people do not respond. Although some work has been completed on predictors of treatment outcome (Keijsers et al, 1994; Lax et al, 1992; Mataix-Cols et al, 2002a), the results remain mixed and further work in this area is necessary. Further work is necessary to determine the optimal interventions for those people who do not initially respond to CBT.

2.4.3 Special considerations in the treatment and management of BDD in the NHS

People with BDD generally feel misunderstood and are secretive about their symptoms because they think they will be viewed as vain or narcissistic. They may indeed be stigmatized by health professionals who view only true disfigurement as worthy of their attention or confuse BDD with body dissatisfaction (Carter, 2001). Therefore when they do present to health professionals, they are more likely to complain of depression or social anxiety as these are less stigmatising, or they may present with drug or alcohol related problems. Insight is often poor and they are more likely to disengage from mental health services and not be followed up vigorously. Sometimes, people with BDD will gain access to mental health services with a diagnosis of hypochondriacal disorder, somatoform disorder or even a psychotic disorder, each likely to lead to inappropriate treatment with anti-psychotic agents or ineffective talking therapies.

2.4.4 The relationship of the evidence base for adults to that of children and adolescents

As there is virtually no evidence base for the treatment of BDD among children and adolescents, general principles of treatment for OCD in these age groups may be relevant as long as the specific issues in point 2.4.3 above are kept in mind.

2.5 Detection, Assessment & Diagnosis

2.5.1 Detection

In the United Kingdom, health care services are organised in such a way that most mental health problems are first detected in primary care by general practitioners, practice nurses, health visitors, mental health and other primary healthcare professionals. In fact access to psychiatrists and psychological therapists almost always requires referral from the GP to secondary or specialist mental health services. In primary care, 40% of patients presenting will have significant mental health problems, whilst in 20- 25% this will be the main reason for attendance (Department of Health, 2001). However the detection rate in general practice, even for relatively "high profile" mental heath problems such as depression, may be no more than 50% (Freeling et al,

1985; Gilbody et al, 2003). Although there are not any data for OCD among adults in the UK, a recent study on other anxiety disorders in primary care in the US concluded that less than one third of patients had received either psychotherapy or pharmacotherapy that met a criterion for quality care (Stein et al, 2004). It is likely that a similar situation exists for OCD. A report from Holland found that two-thirds of patients with OCD referred to a University Medical Centre were receiving no medication, inappropriate medication or ineffective doses of appropriate medication (Denys et al, 2002b). Likewise, half of a consecutive series of young people with OCD referred to specialist centre in the UK had not received evidence-based CBT or pharmacotherapy before referral (Chowdhury et al, 2004).

Why should this be so? Both doctors and patients may perceive psychological problems as having a lower priority than "physical" difficulties, down-playing psychological distress and symptoms by patients and an over-focusing on physical symptoms and findings by doctors working within limited time and other constraints. For patients with OCD who may see their symptoms as stigmatising and potentially shameful, these difficulties may be compounded, limiting the consultation to more "comfortable" physical problems such as skin problems resulting from repeated hand washing but reducing the chances of disclosure of specific psychological symptoms (Eddy & Walbroehl, 1998) Referrals may then be made to physical health settings, such as dermatology, where undiagnosed OCD may be found (Fineberg et al, 2003). In other cases, lack of insight will prevent the person with OCD reporting difficulties to their doctor, although other family members may suspect or recognize some of the difficulties. Even when a family member reports the problems to the doctor, the person with OCD may still be unwilling to discuss the problems because they do not believe that they have problems, do not consider that they wish to receive or require help, or because of the difficulties associated with disclosure.

Despite the increased awareness of mental health in primary care, doctors may not be trained, or may not have the time, to systematically ask the right questions that could lead to rapid detection of OCD. The other health professionals may equally lack the training and experience to consistently detect OCD. Given the difficulties that people may have in disclosure, unless the right questions are asked, detection may be difficult.

When the consultation process is working well, a mutually supportive and trusting relationship between patient and doctor or other health professional will provide a safe context within which the disclosure of difficult information is more likely to occur (Di Blasi et al, 2001). This in turn can lead to more effective collaboration and communication, increasing the chances of early and accurate diagnosis and of timely and effective treatment (Stewart, 1995). Ideally this process will be facilitated by continuity of care where unusual patterns of consultations or requests for treatment such as, for

example, repeated requests for emollients may alert clinicians to the underlying diagnosis .

Even when people are referred to secondary care, there is no guarantee that the referral is for OCD rather than for anxiety, depression or other diagnoses. Depending on the point of entry, a person with OCD may not receive a rapid and accurate diagnosis. For example, in a large community study of OCD among adolescents, although four of the twenty young people with OCD were receiving counselling, in three of the cases, although they had sought help for depression and anxiety, the professional was unaware of the OCD (Valleni-Basile et al, 1994).

2.5.2 Disclosure

The difficulty of initial disclosure by people with OCD both to family and friends and to the medical profession should not be underestimated (Newth & Rachman, 2001). The range of difficulties recognised by service users can include any of the following, (although this list is by no means exhaustive):

For people who do not know they have OCD but have fears of killing or harming people or is constantly washing or checking it can feel almost impossible to start to explain their fears to another person.

- A person who has read about or heard of OCD may be able to say to their GP 'I think I may have OCD' but may fear the response or doubt whether the GP will know anything about the condition.
- Many people with OCD start to have symptoms as children when they may lack the right language to express what they are experiencing.
- Some people with OCD may talk about their symptoms in very general terms to their GP and are often diagnosed with depression or other forms of anxiety
- Many people with shame and embarrassment feel shame or embarrassment at revealing the nature of their obsessions even to supportive family members and friends. They probably feel guilty about the nature of their thoughts and fear that they may carry out their obsessional thoughts.

2.5.3 Stigma and potential consequences of diagnosis

Many patients express relief at being diagnosed as suffering from OCD as this offers an explanation for their symptoms, eliminates "self diagnosis" of other seemingly more serious complaints and opens up the possibility of treatment and help. The patient's family may also be more accepting of a recognised medical condition, and feel they may participate in the patient's therapy. However, the converse is also true due to the stigma that can be attached to mental health issues giving rise to embarrassment, shame, guilt or depression

(Stengler-Wenzke et al, 2004). People may fear stigmatisation at work, and/or concerns about disclosing the condition to new employers, insurers, friends and family.

Once a person knows that they have OCD and that this has been recorded in their medical records they are likely to be concerned about being labelled 'mentally ill' and the consequences of this. The fears recognised by service users might include any of the following (although this list is by no means exhaustive):

- Concern that GPs and medical specialists will assume any illness is then likely to be anxiety related rather than physical.
- Fear of telling their employer with the possibility of losing employment or the impact on promotion.
- Fear of applying for new jobs and whether to disclose this in any medical questionnaire relating to work.
- Questions about whether the information should be disclosed on any other official form, such as insurance, which asks for health information.
- Uncertainty about whether this will affect eligibility to adopt or foster children, to serve on juries or to emigrate.
- Concern about the likely course of the illness and whether they will be on medication for the rest of their life.
- The likely impact in the long term on relationships and the ability to care for children.
- For younger people, the impact on their education.
- Concern about OCD being mistaken for a condition that is associated with criminal behaviour such as that of a psychopath or paedophile.
- Concern about general stigma from friends or family who do not understand the nature of anxiety-related conditions.

2.5.4 Assessment

2.5.4.1 Adult

OCD is a relatively common illness that cuts across the lifespan (Fineberg & Roberts, 2001). One community survey identified a lifetime prevalence of between 2-3% (Robins et al, 1984). Yet the illness is poorly recognised and undertreated (Hollander & Wong, 1998) and although there may be evidence that the time lag between onset of symptoms and correct diagnosis is shortening (Mallery, 1996), individuals with the disorder have been reported to wait on average up to 17 year before correct treatment is initiated (Hollander & Wong, 1998). Given the substantial socioeconomic costs associated with untreated OCD, better recognition and treatment of the disorder has been recognised as a major public health objective (Hollander & Wong, 1998).

Whereas OCD is often underdiagnosed for reasons described above, it is occasionally wrongly diagnosed when people present with repetitive or compulsive behaviours that may resemble compulsions in OCD. These include Tourette's syndrome (Muller et al, 1997), autism and autistic spectrum disorders (McDougle et al, 1995c; McDougle et al, 2000b), Prader-Willi syndrome (Clarke et al, 2002), dementia (Mendez et al, 1997; Rosso et al, 2001; Stein et al, 1997a), and Schizophrenia (Ongur & Goff, In Press). In these cases, although comorbidity between these conditions and OCD remains a possibility, a misdiagnosis of OCD may result in unsuitable or ineffective treatment,

Increased awareness is the key to better recognition and treatment of OCD. Although it can take years before finding a health professional in whom a person with OCD will spontaneously confide, direct enquiry by a sympathetic health practitioner is usually successful (Fineberg et al, 2003). Practitioners in areas known to attract large numbers of patients should be encouraged to look for symptoms. Active enquiry, using brief screening instruments, should be incorporated into every mental state examination . For example, the following questions from the Zohar-Fineberg Obsessive Compulsive Screen (ZF-OCS), may be used: *(1) Do you wash or clean a lot? (1) Do you check things a lot? (3) Is there any thought that keeps bothering you that you'd like to get rid of but can't? (4) Do your daily activities take a long time to finish? (5) Are you concerned about orderliness and symmetry?*.

There has been a lack of suitable OCD screening instruments for detecting OCD in the non-specialist setting. In order to be useful, the instrument needs to be brief and user-friendly as well as able to accurately identify individuals with the disorder without missing too many (sensitive) and also able to screen out individuals with out the disorder without being overinclusive (specific). Positive responses to the screening measure would then be followed up by a more detailed clinical evaluation.

Three instruments have been developed for use in the non-specialist as well as the psychiatric setting. The computerised Symptom Driven Diagnostic System for Primary Care (Weissman et al, 1998) screens for a range of affective and anxiety disorder as well as OCD. It consists of a computerised questionnaire and a diagnostic interview administered by a nurse or clinical assistant who types the answers into a computer to generate a one-page summary of diagnostic information. Unfortunately, comparison of the results of this test with those from a reliable structure clinical interview (Structure Clinical Interview for DSM-IV [SCID-IV] gave poor overall agreement (kappa =0.28) and the test cannot therefore be recommended (Taylor et al, 2002).

The second instrument is a computerised telephone administered version of the Primary Care Evaluation of Mental Disorders (Kobak et al, 1997). It also

assesses a range of affective and anxiety disorders and takes about ten minutes to complete. Individuals dial a telephone number and answer questions that follow an algorithm directed by branching logic. Compared to the SCID-IV the PRIME-MD provided reliable diagnosis reliablilty in diagnosing OCD (kappa = 0.64). The screening instrument is disadvantaged by requiring a specialised computer programme and the system is not widely available in the UK.

The third instrument, the Zohar-Fineberg Obsessive Compulsive Screen (ZF-OCS), was devised by J. Zohar for the International Council on OCD (International Council on OCD 1995). It consists of 5 brief questions designed to be administered by a doctor or a nurse and takes less than one minute to administer (Fineberg & Roberts, 2001). It was validated against the Mini International Neuropsychiatric Interview in a population of UK dermatology outpatients (MINI) (Lecrubier et al, 1997) and found to have good patient acceptability as well as satisfactory sensitivity and specificity (Fineberg et al, 2003). Its psychometric properties are undergoing further evaluation in a range of psychiatric and non-psychiatric settings. In view of its brevity and utility, it can be considered as a possible screening tool for further evaluation.

A variety of self-report questionnaires have been developed for OCD that may be useful for detecting OCD (Taylor, 1995; Taylor et al, 2002). Self-report versions of the Y-BOCS, both paper and computer administered have been developed (Rosenfeld et al, 1992; Steketee et al, 1996) and have equivalent properties to the clinician administered Y-BOCs. It provides both a symptom checklist and a severity score. A short form of the Obsessive Compulsive Inventory has also been developed with good psychometric properties (Foa et al, 2002b), the longer form of which was developed to address some of the issues with other self-report measures. The longer form may be too long for routine detection, but provides a more comprehensive list for general assessment of symptoms (Foa et al, 1998). Another brief measure is the Clark-Beck Obsessive-Compulsive Inventory (Clark & Beck, 2002). These questionnaires may help identify OCD and also provide a measure of severity and an overview of symptoms.

Although there are not extensive data on patients with OCD, a study on patients in FDA drug trials for the anxiety disorders indicated that "suicide risk among patients with anxiety disorders is higher than in the general population by a factor of ten or more" (Khan et al, 2002): there were no significant differences between OCD and the other four anxiety disorders studied. Further, with the high rate of depression in OCD and the risks that may be associated, it is important to investigate depressive symptoms carefully. Measures of depression may prove useful such as the Hamilton Depression Rating Scale, a clinician rated scale, and the Beck Depression Inventory, a self-report measure. The Clinical Outcomes in Routine Evaluation may also be a useful for assessment of a broad range of issues, but

should normally be supplemented by an OCD specific measure such as the mentioned above. Given the potential range of impact that OCD can have on a persons life, measures of quality of life may also be important, especially when assessing for change in the person's overall function.

2.5.5 OCD in specific populations

OCD is frequently complicated by depression which supervenes in over two thirds of cases during their lifetime (Rasmussen & Eisen, 1990a) It is often the depression that encourages the individual to seek help from their primary care physician, and at this point the OCD can be missed if the doctor fails to enquire about the condition. However, surveys have shown that only a small proportion of individuals with OCD actually present to their GP for treatment. For example, a recent survey by De Waal et al (2004) found a point prevalence of 0.5% in a large cohort in general practice. Therefore the pick-up rate for OCD formal screening in primary care is not likely to be high.

Higher numbers of individuals with OCD have been identified by a small number of surveys in a range of hospital (secondary care) settings such as 20% UK dermatology outpatients (Fineberg et al, 2003) and 32% cases presenting to rheumatologists and dermatologists with systemic lupus erythematosis (Slattery et al, 2004). While by no means conclusive, these surveys suggest that screening may be more profitably employed in specific secondary care settings such as those just described.

2.5.6 Children and Young people

In young people as in adults, there can be a long delay in a person suffering from OCD receiving a diagnosis and starting to get treatment. Even in young people, OCD can be causing significant impairment for several years before help is obtained. The reasons for this are not fully understood, but may have some commonalities between children and adults. For example, children with OCD can often feel embarrassed about their symptoms, and try to keep them a secret even from their closest family. Interviews should be undertaken with parents or carers as well as with the young person, making sure that the child has an opportunity to be seen alone. Given that symptoms of OCD may be linked to particular activities or contexts, the impact on school, home and leisure activities need to be investigated.

Pre-school children often have some rituals or repetitive behaviours as part of normal development. These can usually be easily distinguished from OCD as they do not cause distress, take up much time, or stop the child doing other things. In the early stages of OCD, a parent may mistake increased ritualising in their child for ordinary childhood behaviour. Even when it becomes apparent that a young person has a problem with intrusive thoughts and ritualistic behaviour, they and their family and friends may not realise that

this is OCD, and may be reluctant to seek help. Even when help is sought, health professionals may not be aware of the characteristic symptoms of OCD and the diagnosis may not be made, or an incorrect diagnosis made. Sometimes when a diagnosis has been made, young people may be told to 'wait to see if it goes away', or be referred for an inappropriate treatment.

Once the diagnosis of OCD is suspected, it can be helpful to use standardised rating scales to help the young person reveal specific information regarding symptoms, rate severity, and monitor treatment. These might include disorder specific scales such as the Children's Yale Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al, 1997) or the Child Obsessive Compulsive Inventory (Shafran et al, 2003). A general emotional and behavioural symptom checklist, such as the Strength and Difficulties Questionnaire (SDQ; Goodman, 2001) will ensure that comorbid conditions are not missed.

2.5.7 BDD

2.5.7.1 Detection

Detection of BDD is relatively easy to make but is often overlooked. There is a low level of awareness about BDD amongst health practitioners. Few practitioners ask simple questions for the diagnosis. However individuals with BDD are often too ashamed to reveal the true nature of their problem without direct questioning (e.g. Grant et al, 2002). Furthermore, when individuals with BDD do seek help they are more likely to consult a dermatologist or cosmetic surgeon. When BDD patients finally seek help from a family doctor or mental health professional, they are often too ashamed to reveal their main symptoms and present with symptoms of depression, social phobia, or obsessive-compulsive disorder. Individuals may be secretive because they may think they will be viewed as vain or narcissistic.

2.5.7.2 Assessment and diagnosis

First *et al* (1997) suggest the following questions to screen for patients with BDD, "Some people worry a lot about their appearance. Do you worry a lot about the way you look and wish you could think about it less?" In those who answer positively, then one might ask (a) "*What specific concerns do you have about your appearance?* (b) *Do you think about them a lot and is it hard to stop thinking about them? On a typical day, how many hours a day is it on your mind?* (More than a hour a day is considered excessive) (c) *How much does it bother you? What effect does it have on your life? Does it make it hard to do your work or be with friends?"*

Dufresne et al (2001) validated a brief self-report questionnaire to screen for body dysmorphic disorder in cosmetic dermatology settings using a reliable

clinician-administered diagnostic interview for BDD for comparison. The self-report questionnaire had a sensitivity of 100% and a specificity of 93%. The interrater reliability of the defect rating scale was .88. These results suggest this brief questionnaire was an effective screening tool for BDD in a dermatology setting.

2.5.7.3 BDD among those with other psychiatric problems

There is a number of groups of people with psychiatric disorders who are known to be at higher risk of BDD. Grant *et al* (2002) screened 122 consecutive in-patients and 16 (13.1%) were diagnosed as having BDD by a structured interview but none of the participants had been diagnosed as having BDD by their treating physician. Similarly, Zimmermann & Mattia (1998) found that none of 500 referrals to a psychiatric out-patient clinic in the USA had been diagnosed as having BDD through routine unstructured clinical interview, but 3.2% (n=16) of a second group of 500 were diagnosed as having BDD when a structured diagnostic interview was introduced (SCID for DSMIV),. Of 350 out-patients with major depression who entered an anti-depressant treatment study, 23 (6.6%) were diagnosed with current BDD by structured diagnostic interview (Nierenberg et al, 2002). The rate was higher in atypical depression (14.4% compared to 5.1%)

In two surveys of patients attending a clinic for anxiety disorders, the rates of BDD were highest amongst those diagnosed with social phobia (13% in Brawman-Mintzer et al, 1995; 12% in Wilhelm et al, 1997). An additional comorbid diagnosis of social phobia in BDD can only be made when there is a broader fear of negative evaluation by others and not just of one's appearance. The diagnosis of OCD is given only when the obsessions and compulsions are not restricted to concerns about appearance. Sometimes the symptoms overlap – for example a patient may believe that their skin is both ugly and contaminated. A similar situation exists in patients pre-occupied with order and symmetry of which one symptom might be focused on one's hair on another body part being symmetrical and feeling "equal". In a study of 165 patients seeking treatment for an anxiety disorder, 7.7% of OCD patients had a current diagnosis of BDD (Wilhelm et al, 1997).

A common diagnostic dilemma for BDD is that of an eating disorder. BDD and eating disorders share a distorted body image and many other symptoms such as a low self-esteem. DSM-IV states that a diagnosis of BDD should not be used if symptoms are best accounted for by a diagnosis of an eating disorder. If therefore the preoccupation is predominantly focussed on being "too fat" or overweight, it does not meet criteria for BDD. There is a grey area between individuals with disordered eating who do not fulfil the criteria for an eating disorder. True comorbidity of BDD and eating disorder occurs when a patient is preoccupied by imagined defects in their appearance which is unrelated to weight and shape. Grant et al (2002) reported that 16 (39%) of 41 patients with anorexia nervosa were diagnosed with comorbid BDD

unrelated to weight concerns. The most common preoccupations in the study by Grant et al were (in descending order) with the nose, skin, hair, chin, lips and eyes. The patients with anorexia nervosa and BDD had significantly lower overall functioning and higher levels of delusionality than the anorexic patients without BDD, suggesting that the former had a more severe form of illness.

2.5.8 BDD and risk of suicide

There is a significant risk of suicide associated with BDD. According to Phillips & Diaz (1997), 22% of their BDD patients had attempted suicide at some time and Veale *et al* (1996b) reported that in a series of 50 BDD patients in the UK, 24% had attempted suicide. Among individuals with BDD and depression, a sense of hopelessness is more likely to occur when a person with BDD believes that he or she is trapped and has exhausted all their abilities to camouflage or alter their appearance. An individual who has just had cosmetic surgery and realises that all their hopes have been dashed may be at particular risk.

2.5.9 Cosmetic and dermatological procedures and BDD

Several studies have established that people with BDD may also be found in medical settings that deal with cosmetic or dermatological procedures. There have been six surveys of BDD in a cosmetic surgery setting. BDD is relatively common in such settings with a prevalence of between 3-18% (Aouizerate et al, 2003; Castle et al, 2004; Ishigooka et al, 1998; Sarwer et al, 1998; Vindigni et al, 2002). There have been two surveys in a dermatological setting. Phillips et al (2000) found 12% of 367 of dermatological patients in the USA and Uzun *et al* (2003) found 8.8% of 159 dermatological out-patients in Turkey had BDD. The prevalence of BDD depends on the procedures offered at each clinic, the sex ratio, and the diagnostic threshold and instrument used to measure BDD. For example, some procedures like rhinoplasty may attract more people with BDD. There may be difficulties in the diagnosis of BDD in a cosmetic or dermatological setting when someone has a minor physical anomaly and when the concern becomes "markedly excessive". Also aesthetic cosmetic procedures are often concerned with enhancing a "normal" appearance and the diagnosis of BDD centres on the degree of preoccupation, the distress and psychosocial handicap.

There are three retrospective studies of cosmetic surgery in BDD patients attending a psychiatric clinic. Phillips & Diaz (1997) reported in a sample of 188 BDD patients, 131 patients sought and 109 received surgical, dermatological or other medical treatments, however 83% reported an exacerbation of or no change in BDD symptoms. The most common outcome following surgery was no change in overall BDD severity (58%) and no change in the concern with the treated body part (48.3%). More patients worsened in overall BDD severity (24.3%) than improved (17.4%). However, in terms of the treated body part, more patients reported a decrease (34.5%)

than an increase (17.2%) in concern. No data is provided on satisfaction for the procedures.

Veale (2000) reported on 25 BDD patients in the UK after cosmetic surgery, who had had a total of 46 procedures and 76% were dissatisfied postoperatively. Three patients claimed that they were not preoccupied by their appearance prior to the surgery and that their symptoms of BDD developed only after surgery, which they believed, had been done badly. Some operations, such as rhinoplasty appear to be associated with higher degrees of dissatisfaction. Most of the patients in the study had multiple concerns about their appearance and reported that after 50% of the procedures the preoccupation transferred to another area of their body. When patients were dissatisfied with their operation, they often felt guilty or angry with themselves or the surgeon at having made their appearance worse, thus further fuelling their depression and a failure to achieve their ideal.

These studies have limitations. The data is retrospective and there is a selection bias of patients in favour of treatment failures. Mental health practitioners may not see BDD patients who are satisfied with their cosmetic surgery and overcome their symptoms of BDD. Milder cases may also be satisfied with the outcome of surgery and may not immediately return to psychiatric treatment. Further, there is no control group of normal or psychiatric patients who have undergone cosmetic surgery but do not have BDD. However, the majority of individuals who do not have BDD appear satisfied with cosmetic surgery and their self-esteem and other psychological measures tend to improve (Harris & Carr, 2001; Klassen et al, 1996). Cosmetic surgeons usually try to determine whether the patient's expectations of change are realistic. In general, patients will have good psychological outcome if they can clearly describe the problem that concerns them and their desired outcome. The surgeon will then discuss with the patient for the likely result when surgically altering their appearance and outline the consequences and risks. Among people with BDD, expectations for major psychosocial changes (for example getting a better relationship) are often unrealistic.

2.6 Stepped care

Given the complexity of need and healthcare organisation, the way that psychological treatments, particularly CBT, is delivered has become an increasing focus of interest, such as the US model of Stepped Care. Stepped care argues that the least intrusive intervention (e.g. education or self-help) should be used first and only moving to more intense therapy when less intensive treatment has proved to be insufficiently effective (Haaga, 2000)

This guideline suggests that such a model could prove useful if applied to UK settings to encourage access to intensive treatment when severity or risk indicate less intensive treatment would be inappropriate. The Stepped Care Model (Figure 1) provides a model for the most effective but least intrusive treatments appropriate to a person's needs. It assumes monitoring of the course of a person's difficulties and referral to the appropriate level of care. Each step introduces additional interventions; the higher steps normally assume interventions in the previous step have been offered and/or attempted, but there are situations where an individual may be referred to any appropriate level.

Who is responsible for care?	What is the focus?	What do they do?
Step 6: Inpatient care or intensive treatment programmes (CAMHS Tier 4)	OCD or BDD with severe distress or disability, risk to life or severe self-neglect.	Reassess, discuss options, care coordination, SRI, CBT including ERP, or combination of SRI and CBT including ERP, augmentation strategies, consider admission or special living arrangements
Step 5: Multidisciplinary care with expertise in OCD or BDD (CAMHS Tier 3/ 4)	OCD or BDD with significant comorbidity, or more severely impaired functioning and/or treatment resistance, partial response or relapse	Reassess, discuss options: SRI, CBT including ERP, or combination of SRI & CBT including ERP; consider care coordination, augmentation strategies, admission social care. For children and young people: reassess, discuss options: CBT including ERP,/ SSRI/clomipramine/ combined treatment For teenagers consider referral to specialist services outside CAMHS if appropriate
Step 4: Multidisciplinary care in primary or secondary care (CAMHS Tier 2/3)	OCD or BDD with co-morbidity or moderately impaired functioning or poor response to initial treatment	Assess & review, discuss options: For adults: ERP, SSRI, alternative SSRI or clomipramine, consider CBT, combined treatments. For children and young people: CBT including ERP, then consider CBT/ SSRI/clomipramine/ combined treatment
Step 3: GP/primary care team, PCMHW (primary care mental health worker)/ family support team (CAMHS Tier 1 or 2)	Management and initial treatment of OCD or BDD	Assess & review, discuss options. For adults according to impairment: guided self help, computerised ERP, individual or group ERP, SSRI, or consider combined treatments; consider involvement with the family. For children and young people: guided self help, CBT including ERP, involve family and consider involving school
Step 2: GP, practice nurses, school health advisors, general health settings (including hospitals) (CAMHS Tier 1)	Recognition/ Assessment	Detect, educate, discuss treatment options, signpost voluntary support organisations, provide support to individuals, families, work/ schools, or refer to any of the appropriate levels
Step 1: Individuals, public organisations, NHS	Awareness	Provide, seek, share information about OCD or BDD, & its impact on individuals & families

The awareness, recognition and treatment of OCD and BDD is suggested to proceed across six phases, depending upon need and the characteristics of a person's OCD/BDD. The model also provides a framework to organise services to support the public, patients, carers, and healthcare professionals in identifying and accessing the most effective interventions:

- Awareness of OCD or BDD by individuals, public organizations, and the NHS
- Recognition of OCD or BDD in primary care, school health, and general hospital settings
- Management and initial treatment of recognized OCD or BDD in general practice
- Involvement of multidisciplinary care in primary and secondary care for OCD or BDD

- Involvement of multidisciplinary teams with specific expertise in the management of OCD or BDD
- Involvement of inpatient care or intensive treatment programmes for OCD or BDD where there is risk to life, severe self-neglect, severe distress or disability.

At all stages of assessment and treatment, family and carers should be involved as appropriate. This is particularly important in the treatment of children and young people with OCD where it may also be helpful to involve others in their network, for example teachers, school health advisors, educational psychologists, and educational social workers.

2.7 Clinical practice recommendations

2.7.1 Understanding

2.7.1.1 People with OCD or BDD are often ashamed and embarrassed by their condition and may find it very difficult to discuss their symptoms with healthcare professionals, friends or family. Healthcare professionals should help people with OCD/BDD, and their families where appropriate, understand the involuntary nature of the symptoms caused by the disorder, and the shame and distress experienced**. [GPP]**

2.7.1.2 When assessing a person with OCD/BDD, healthcare professionals should sensitively explore the hidden distress and disability commonly associated with OCD and BDD, providing explanation and information wherever necessary. In particular, people with OCD who are distressed by their obsessive thoughts should be informed that such thoughts are occasionally experienced by almost everybody, and when frequent and distressing are a typical feature of obsessive-compulsive disorder. **[GPP]**

2.7.2 Continuity of care

2.7.2.1 OCD and BDD are frequently recurring or chronic conditions which often affect some of the most intimate aspects of a person's life. Healthcare professionals should therefore ensure continuity of care and minimize the need for multiple assessments by different healthcare professionals. **[GPP]**

2.7.3 Religion

2.7.3.1 Where obsessive-compulsive symptoms involve a person's religion, such as religious obsessions and scrupulosity, healthcare professionals should consider seeking the advice and support of an

appropriate religious or community leader to support the therapeutic process. **[GPP]**

2.7.4 Awareness, recognition, and training

Although the more common forms of OCD are likely to be recognised when people report symptoms, less common forms of OCD and many cases of BDD may remain unrecognised, sometimes for many years. Relatively few mental health professionals may have expertise in the recognition, assessment, diagnosis and treatment of the less common forms of OCD and BDD.

2.7.4.1 PCTs, mental healthcare trusts, and children's trusts that provide mental health services should have access to a specialist OCD/BDD multidisciplinary healthcare team that can help increase the skills of mental health practitioners in the assessment and evidenced-based treatment of children and adults with OCD/BDD, provide high quality advice, and, when appropriate, conduct expert assessment and specialist cognitive-behavioural and pharmacological treatment across the lifespan. **[GPP]**

2.7.4.2 Specialist mental healthcare professionals who work with children, young people and adults with OCD/BDD should collaborate with local and national voluntary organisations to increase awareness and understanding, and to improve access to high quality information about OCD and BDD. Such information should also be made available to primary and secondary care professionals, and to professionals from other public services who may come into contact with people of any age with OCD/BDD. **[GPP]**

2.7.4.3 Specialist OCD/BDD teams should work with people with OCD or BDD and carers to provide training in the recognition, basic epidemiology, assessment and treatment of people with OCD and BDD. Such training should be for all mental health workers, and cosmetic surgery and dermatology professionals. **[GPP]**

2.7.5 Recognition and assessment

Given that people with OCD may have difficulty in disclosing their symptoms, people with disorders known to be commonly associated with OCD or BDD should be specifically assessed for these conditions and the possibility of co-morbidity, especially those with depression and anxiety. People with co-morbid depression should be assessed for the risk of suicide.

OCD

2.7.5.1 For children, young people and adults known to be at higher risk of OCD, such as people with symptoms of depression, anxiety, alcohol or substance misuse, BDD or an eating disorder, or for people

attending dermatology clinics, healthcare professionals should routinely consider and explore the possibility of co-morbid OCD. **[C]**

2.7.5.2 In the assessment of people at higher risk of OCD, the following six questions should be asked to identify children, young people or adults with OCD:

- Do you wash or clean a lot?
- Do you check things a lot?
- Is there any thought that keeps bothering you that you'd like to get rid of but can't?
- Do your daily activities take a long time to finish?
- Are you concerned about orderliness and symmetry?
- Do these symptoms interfere with your life? **[C]**

2.7.5.3 If healthcare professionals are uncertain about the risks associated with intrusive sexual, aggressive or death-related thoughts reported by a person with OCD, they should consult mental health professionals with specific expertise in the assessment and management of OCD**. [GPP]**

2.7.5.4 In people who have been diagnosed with OCD, healthcare professionals should assess suicide risk, especially if the person has also been diagnosed with depression. **[GPP]**

2.7.6 BDD

2.7.6.1 For children, young people and adults known to be at higher risk of BDD, such as with symptoms of depression, social phobia, alcohol or substance misuse, OCD or an eating disorder, or for people seeking cosmetic surgery or attending dermatology clinics, healthcare professionals should routinely consider and explore the possibility of co-morbid BDD. **[GPP]**

2.7.6.2 In the assessment of people at higher risk of BDD, the following five questions should be asked to help identify children, young people or adults with BDD:

- Do you worry a lot about the way you look a lot and wish you could think about it less?
- What specific concerns do you have about your appearance?
- On a typical day, how many hours a day is it on your mind? (More than a hour a day is considered excessive)
- What effect does it have on your life?
- Does it make it hard to do your work or be with friends? **[GPP]**

2.7.6.3 In people who have been diagnosed with BDD, healthcare professionals should assess suicide risk especially if the person has also been diagnosed with depression**. [GPP]**

2.7.6.4 Mental healthcare specialists in BDD should work in partnership with cosmetic surgeons and dermatologists to ensure that an agreed screening system is in place to accurately identify people with BDD. **[GPP]**

2.7.6.5 People with suspected BDD attending dermatology departments should be referred for a comprehensive mental health assessment. **[GPP]**

2.7.6.6 People with suspected BDD seeking cosmetic surgery should be assessed by mental health specialists in BDD prior to surgery being offered as the outcome of surgery for these people is unpredictable. Symptoms of BDD may persist; the patient may be dissatisfied with the procedure and continue to seek further treatment. **[GPP]**

3 The experience of people with OCD and carers

This chapter consists of a number of personal testimonies that illustrate the experience of a number of people with OCD and one with BDD and also of those involved as family members and/or carers of people with OCD. The testimonies were chosen to demonstrate something of the range of experience of sufferers and carers and should not be taken as representative. These narratives express the experience of OCD over the lifetime, the effect on family and carers, and the process of obtaining appropriate treatment and from the response to such treatments. The testimonies draw from experiences of OCD and BDD over the last 40 years.

3.1 Personal testimonies from people with OCD and BDD

3.1.1 Daniel

I've just arrived home from work. Tired and tense, I'm convinced my hands are contaminated with some hazardous substance and my primary concern now is to ensure that I don't spread that contamination to anything that I, or others, may subsequently touch. I will wash my hands, but first I will need to put a hand in my pocket to get my door keys, contaminating these, the pocket's other contents, and everything else I touch on my way to the sink. It will be late evening before I will have completed the whole decontamination ritual. Tomorrow I will inadvertently touch another contaminant, and a similarly exhausting process will have to be performed.

That's how it was 40 plus years ago when, in my early twenties, my OCD became firmly established. Fear of contamination was the main manifestation, primarily, I suspect, because my work brought me close to genuinely hazardous materials: taking precautions was the expected norm. However, 'checking' had also become a major preoccupation, not least feeling compelled to ensure that I hadn't been responsible for causing harm to others. Frequently, for example, I would retrace a car journey (often over a very long distance) to make sure I hadn't accidentally hit a pedestrian.

Despite the fact that these compulsions were distressing in themselves and were wasting inordinate amounts of time, I did not seek help. I believed my behaviour to be simply that of a responsible citizen. I struggled on as best I could until, at the age of 27, the distress, exacerbated by an upheaval in my

domestic arrangements, increased to such a level that I was admitted to hospital. At that time (1966) OCD was not widely recognised and I was diagnosed with 'anxiety neurosis'. Hospital provided some respite from both the unsatisfactory domestic/accommodation situation and my OCD triggers, but effective treatment for the disorder was not forthcoming (at one stage a lobotomy was discussed, but fortunately not pursued) and, after 12 weeks, I was discharged.

For the next 15 years or so I ploughed on, always managing to work and support myself, but not having much of a life as the condition, which I had come to regard as unalterable, ebbed and flowed. I did seek treatment from time to time and received prescriptions for medication such as nitrazepam and, later, diazepam. The manifestations of the disorder now included the fear of an extended range of contaminants (my cleaning compulsions demanded the removal of every last molecule of 'dirt', so washing or bathing could take hours). Also, I felt compelled to carry out endless checks to make sure things were safe (doors locked, gas off, and so on) and that no mistakes had been made. The latter could be taken to extraordinary extremes: I would imagine that a decision I had taken as part of my work as an engineer could lead to another engineer somewhere relying on my erroneous decision in his calculations, and so on, and so on, until an aeroplane fell out of the sky. And it would all be my fault, so I felt compelled to reassure myself that all was well. Hours could be spent on such exhausting exercises.

Up until this point I had lived alone and so had been able to indulge my compulsions without anyone really noticing. In the early 1980s things changed: I married, became a father, and got some help. This involved more medication and regular sessions with a psychiatrist who diagnosed/confirmed OCD. I was a private patient and, over a period spanning many years, chose to discontinue and re-start the sessions as I felt necessary. The sessions were useful, although in retrospect I can see their value was limited since, instead of finding the courage to confront my fears, I used the meetings for the comfort and relief that an understanding ear can provide. During this period I was still managing to work, but the OCD was certainly restricting my life. I wouldn't travel on certain bus routes because I believed one of the vehicles used on that route was contaminated. I would cross the road to avoid road sweepers and their contaminated brushes. I wouldn't join in outings because of the risk of getting dirt on my limited range of clean clothing.

But now I was a father, and particularly when we had our second child, matching the children's needs for a normal childhood with my own desires for cleanliness and order was not easy. I coped, after a fashion, but the constant anxiety made my experience of their childhood years pretty joyless.

And then about five years ago things took a turn for the worse. Following a heart attack and a modification to my diet, I became extremely depressed and the OCD got much worse. I became quite desperate. I vividly recall one occasion at that time when, weeping, I tried to explain the distress I felt over a compulsion to wash my hands that I had resisted. 'Can you imagine what it feels like to believe you've poisoned your own children?' I asked.

Once again I sought help. SSRIs were tried, but soon stopped because I couldn't tolerate the side effects. Then I was given two courses of CBT. Each of these consisted of 1-hour sessions every 2 weeks for a total of around 10 weeks. As I recall, the sessions were biased towards the cognitive aspects of CBT, with very little in the way of monitored behavioural tasks. Overall, the sessions were quite helpful in that I was able to discuss my concerns, however, it was a further 9 months of weekly behavioural therapy sessions provided by a support group that really helped me to practise exposure and response prevention (ERP). This technique has brought about significant improvements in my condition.

Now, as I approach the age of 65, I strive to maintain the improvement by means of self-administered ERP. The mood stabilisation effects of medication (carbamazepine) newly prescribed to treat my recently diagnosed epileptic absences also seem to be making a positive contribution. So, although by no means '100%', I am a great deal better. But it has been a long 40 years.

3.1.2 Ruth

I experienced what I now realise were the first symptoms of OCD when I was about 12 years old. I started to feel compelled to cancel out any distressing thought I had, such as failing an exam or the possibility of a family member dying, by repeating whatever I was doing when I had the thought and replacing the 'bad thought' with a 'good thought'. This included getting undressed and dressed again, retracing words and sentences I'd written, re-reading pages of a book or walking back over a stretch or patch of ground. I would carry out these actions repeatedly until I had managed to neutralise the thought. This could take a long time and would also cause embarrassing situations when I would make excuses to go back to places or pretend there was something I had to do.

Nothing happened to trigger the start of this way of thinking. Nothing had changed at home or at school and I didn't have any major physical illnesses. I do remember that the thoughts and rituals began slowly but increased over time. At first I would have two or three thoughts a day that needed to be neutralised, but over a period of months this became dozens of unwelcome thoughts each day.

These symptoms caused me great distress, which I felt unable to express to people although I often used to cry at home and at school. The compelling need to repeat certain actions also wasted time and sometimes stopped me going out with friends or to new places for the fear of leaving a 'bad thought' in a place that I couldn't easily return to. If I was unable to undo these thoughts I would feel anxious and uncomfortable and found it difficult to concentrate on other things. On occasions I would make excuses to return to someone's house or a shop several days or even weeks later in order to try and neutralise a thought I had had on the previous visit. I could sometimes remember leaving 'bad' thoughts behind for many months although. Throughout my teenage years I kept my thoughts and bizarre actions to myself, concealing them from family and friends and hiding away to carry out my compulsions.

Despite this, I managed to work hard at school and go to university at the age of 19. By this time the distressing thoughts were becoming increasingly sinister and even more painful to me – I started to be scared that I might cause an accident, or I would think about murder, or about hurting or molesting children, although I knew I had no desire to carry out any of these things. I was terrified of the type of thoughts I was having and hated myself for having them but the harder I tried to shake them off the stronger they seemed to become. I didn't feel I could confide in my parents or even friends or healthcare workers because I thought the nature of these thoughts was so shocking that I would be considered dangerous and perhaps locked up. At this point I became seriously depressed and suicidal, losing a lot of weight and being constantly on the verge of tears. Over the years I saw a number of GPs who diagnosed depression without exploring the nature of my depression in any way. Antidepressants and some sessions with a counsellor offered some temporary relief. However the counselling sessions didn't touch on the real problems I was experiencing; the counsellor didn't ask me the right questions and I didn't feel I could open up about the type of thoughts going through my head.

Over the next few years the disturbing thoughts continued. There would be bad periods when the thoughts seemed to be with me much of the time and then sometimes for no obvious reason they would fade a little, although they never went away completely. Oddly enough when I was very focused on something like preparing for exams, a time which people often think of as stressful, the thoughts would actually ease off a little.

However, in my early 20s the thoughts were joined by a compelling need to check to ensure that I hadn't caused an accident and constant hand washing to avoid passing on contamination. This included regular episodes of going back over journeys on public transport or walking back down a street to check that I hadn't accidentally brushed past someone and hurt or killed them. My fear of contamination was about the possibility that I might have been in

touch with a poisonous substance. Germs didn't really bother me and I even remember reading an article at that time about a woman having treatment for her obsessive hand washing due to a fear of exposure to germs and I didn't associate it with what I was experiencing.

Eventually OCD was diagnosed by a GP when I was 26 and I was referred to a psychiatrist. Had I been questioned in any way about the nature of my thoughts and anxiety, I feel that OCD would have been diagnosed much earlier. I was desperate to confide in someone but no one ever asked the right questions. A long course of tricyclic antidepressants (clomipramine) and some behavioural therapy helped get my OCD back to a manageable level and relieved the depression.

There was only a very small amount of relief in having an official diagnosis for what I was going through. By this time I honestly thought it was too late to get my life back to normal. In addition, referral to a psychiatrist meant having a medical label that I would have to carry with me for the rest of my life. I was also frightened that I might have to physically confront my fears in some way. The fact that the medication very quickly dampened some of the worst thoughts was a relief. Clomipramine caused unpleasant side effects including severe sweating, constipation and a dry mouth but they were a small price to pay and I believe that having it prescribed at that time saved me from possible suicide.

However, I continued to have relapses over the next ten years and it was only when I received a course of CBT along with a different type of antidepressant (an SSRI) that I felt I made significant steps to dealing with my OCD and learning skills that I could draw on during future relapses. Unlike clomipramine, the SSRIs didn't cause any major side effects and the CBT, although very difficult to do, provided me with a new angle for looking at my obsessions and the skills to stop carrying out the compulsions.

I also tried an OCD support group and I initially enjoyed meeting other people with OCD. After many years of hiding my bizarre rituals and painful thoughts, it seemed quite incredible to meet so many people who had such similar experiences and stories to tell. At the same time I found a tendency amongst some people to want to talk only about how terrible OCD was and after a while I found I didn't gain anything from these groups.

At the age of 41 I still have OCD but I also cope well on a day-to-day basis and have done for a number of years. I hold down a management position at work, am in a long-term relationship and enjoy travelling overseas and socialising with friends—things that didn't seemed possible earlier in my life. I continue to take a maintenance dose of an SSRI (currently fluoxetine). I am aware that if I don't keep on top of my compulsions by not giving in to them then they have a tendency to creep back. I find that having the bad thought

and not trying to neutralise it in any way ultimately helps to make the thoughts seem less important, although I have to live with the initial anxiety. The more I try to neutralise the thoughts the more they start to take over. Sometimes I worry that taking antidepressants on such a long-term basis may be damaging for my health but equally I feel that they do work and help me keep on top of my OCD. I would be equally concerned if I was forced to stop taking them.

In looking back, I feel strongly that had my illness been recognised and correctly treated much earlier then I would have been able to achieve far more in life and would certainly not have wasted so much time carrying out senseless compulsions or avoiding situations because of my obsessive thoughts. I came close to suicide on a number of occasions; found relationships difficult because people would pick up on my sometimes odd behaviour and missed out on many opportunities, including overseas travel, flat sharing with friends, promotion at work and marriage and children, because my obsessions prevented me taking these opportunities. OCD came close to completely ruining my life. I don't know where I would be now if I hadn't received CBT from knowledgeable therapists and been prescribed appropriate antidepressants. I do believe that you need to take a certain element of responsibility for your own recovery by being prepared to try appropriate medication and having a go at CBT if it is offered but it is very difficult to do this without sympathetic professional help and guidance.

3.1.3 Estelle

I am 25 years old and OCD first took over my life almost 10 years ago (although I did not know what it was until about 18 months after it all began). I suppose that when I look back I can see 'signs' of OCD back into my childhood but the full-blown illness was triggered suddenly after the suicide of a boy in my year group at school. I believe it was my attempt to make everything 'certain' and 'safe' again after this horrifying event blew apart my sheltered middle-class life. I began to feel I was being taken over by some force in my brain that I could not control, until eventually my days and nights became ruled by its orders:

'If you don't wash your hands twenty times, you will have to kill yourself…oh no- I don't think you got the soap underneath every fingernail on that nineteenth wash, so it's all negated, you must start the washes again…you must stop yourself committing suicide…no, while you had that thought you didn't wash between those fingers, start again!...but it doesn't make any sense- other people don't do this! But just what if, what IF you don't do it, then you might not be able to stop yourself killing yourself!'

This may sound like a psychotic experience but in fact I could very well see that what I was doing made no rational sense. When eventually I had satisfied

the requirements of the washing ritual I would attempt to get myself out of the bathroom. Walking over the threshold of any doorway was another tortuous experience:

'What did you think about when you stepped over that line? Was it anything related to suicide or death or the boy? You thought about Corn Flakes! But CF were the initials of the boy! Go back and do it again, that connects you to him! Make your mind totally blank!'

In the end I could connect anything I thought of to the awful events, just words sounding slightly similar to 'bad words' was enough, and I had to go back and back and back until my mother came and dragged me away, or I made her say it would be ok, or I fell in a heap from sheer exhaustion. The rituals and thoughts were incessant everywhere I was: getting up from a chair, swallowing food, or writing a word in my schoolbook. If I thought bad thoughts as I did the action, the action had to be repeated. Certain clothes, objects, food, TV programmes, were 'bad' and had to be avoided. I would cry at the end of the day just because I wanted the rituals to leave me alone but they would not, and the consequences seemed so important and so dire that I had to keep doing them to protect myself and others from them.

These experiences, and the fact that it felt as though I no longer had control of my mind, caused me to believe that I had gone insane. I think my parents perhaps thought the same, because they did not want me to see any doctors for fear I would be locked up. They tried to reason with me, to 'rationalise away' the OCD; this did not work and could not work, and so they became more and more involved in my rituals as I asked for constant reassurance that 'bad things' weren't going to happen.

Although I had been to see my GP several times with somatic complaints a bit like glandular fever (unsurprisingly I was exhausted the whole time) she had not probed further to find psychological causes, and I had been too frightened to mention the OCD to her of my own account, because it felt so much like madness. Describing the symptoms to health professionals can be one of the worst things for a person with OCD; the awareness that their thoughts and behaviours are abnormal and bizarre can make them they feel embarrassed and also fearful of being laughed at or worse being told they are mad.

It was a friend's mother, another GP, who mercifully rescued me from this hell, or at least told me that there was a way out of it, when I broke down and told her about it. She had me see a psychiatrist and explained to me about OCD; she even gave me a video that explained it; I was also diagnosed with agoraphobia and social anxiety. I cannot express the relief of being told that I was not 'mad' in any classical sense, and that something could be done to make the uncontrollable thoughts and rituals less uncontrollable. I was firstly given a tricyclic antidepressant [clomipramine] and although I came off that

after 6 years because of side effects such as dyskinesia [involuntary movements], I have since tried the full range of SSRIs and SNRIs, and none has been as effective as clomipramine. It is certainly not a cure all, but for me it was more than I could have hoped for, and allowed me to suppress the OCD enough to get myself through high school final exams and into university with good grades. The psychiatrist tried family therapy with us, which I think is probably essential for any child with severe OCD. One of the most potent perpetuating factors in OCD is family members taking part in rituals or offering reassurance; this may feel like helping but it only reinforces the importance of carrying on this bizarre behaviour in the sufferer's mind. But I was also given fairly ineffective strategies aimed at 'stopping' intrusive thoughts – such as snapping an elastic band on my wrist and saying 'stop' every time one popped up. Unfortunately this just added more focus (and more pain) to the thoughts and became a ritual in itself.

Over the years since then the symptoms have waxed and waned. Each time the OCD hits me again with full force, it seems to be in a different form, and each fixation can last several years. After protecting myself from suicide, the next focus for thoughts and rituals was catching AIDS, which involved much washing and disinfecting and avoiding public places where I believed surfaces harboured the dreaded virus. It made no difference to be told 'you can't catch AIDS from surfaces' – the well-worn 'yes, but I might be the exception' or 'what IF…' or 'better to be safe than sorry' revolved around my mind. My current problem is the fear that I will suddenly lose sight of any sense of morals and enter a state of madness where I will start harming other people in horrible ways. The same types of thoughts still pop into my mind as they did 10 years ago, but these days they have less power over me because of treatment I have received.

Although CBT is always put forward as the treatment of choice for OCD, the initial severity of my illness meant that such a treatment would have been ineffective before medication allowed me to function again to a certain extent. The other therapy that helped me understand better why I might suffer from this disorder was psychodynamic therapy, which I received weekly for 2 years whilst at university. Because OCD is so often accompanied by a range of other anxiety and mood problems, not to mention self-esteem issues, this therapy enabled me for the first time to find some value in myself as a person, and to mitigate the hatred I had long felt for myself partly because of the OCD. I had had problems forming friendships and relationships, but as a result of my therapist's acceptance of me I realised for the first time that I did not have to be a victim of my illnesses and defined by them, and could be liked by other people for the person I am.

Over the last year I have finally had the opportunity to have some CBT in a specialist group for OCD sufferers, but had I not first had the psychodynamic therapy I don't believe I would have thought enough of myself or have had

the confidence to do it. Nor would I have understood that my OCD is my way of trying to have control over uncertainties in life. CBT is very hard work, and requires a lot of thought outside the sessions, but it does equip the sufferer with tools to allow them to realise the difference between thoughts and actions; these two are often fused in the OCD sufferer's mind.

I don't think I will ever be free of all the symptoms of OCD and I may always need medication to make it behave itself, but I think I have been lucky to receive good treatment and have now almost completed a masters degree; it is perfectly possible to lead a satisfying life with OCD but the main problem is to get over the hurdle of explaining the thoughts and rituals to a doctor because of the fear of madness. Voluntary work with sufferers has made me realise too that there is widespread ignorance of the signs and symptoms, not to mention the horrifying nature, of this disorder even amongst GPs. In my own experience, some members of the psychology and psychiatric professions tend to steer clear of patients with OCD as they believe it is a 'difficult' illness to treat.

3.1.4 Jane

Only now do I realise that I actually had all the signs of BDD at an early age. I was always sensitive and self-conscious and felt that I was different from the other girls. My confidence increased slightly when I reached my mid teens and I was able to camouflage my appearance with make up and straighten and control my curly hair. With the arrival of a few boyfriends and my marriage at 18 I felt a little 'more normal' but by this time the obsessive behaviours had also set in. I would spend hours grooming my hair and putting on make up and I would not allow myself or anyone else to see me in my 'natural' state (no make-up and hair left to dry naturally) for fear that they would discover the real ugly and disgusting me. From this time on my whole life revolved around trying to keep up this façade. Having carried out the camouflage rituals I would avoid going out in the rain, swim, anything that could affect my image. I visited countless hairdressers, bought endless amounts of hair and make up products and resulted in cutting my own hair, trying desperately to find a miracle that would make me look acceptable but this never happened. This was to continue until I finally found out that I had BDD at the age of 45 and received the right treatment.

At 21 my marriage broke down and I became severely depressed and the illness took over my life. I was so repulsed and disgusted by my appearance that I thought that no one would ever want me again. My parents took me to the GP who treated me for depression and prescribed Valium. The anxiety and depression became so unbearable that I took an overdose and was then referred to a psychiatrist. The next 7 years of my life were spent in and out of hospital trying countless different types of medication but the symptoms persisted and I took more overdoses. I can't remember now what it was that I

was prescribed but I do remember that at times I felt completely 'spaced out' and unable to function and once experienced hallucinations. I also had the feeling that I was being experimented on with endless different drugs that didn't have any effect. I explained to the doctors that I felt distressed because of the way that I looked but this was dismissed. This increased my feelings of embarrassment and shame and I felt that I was also a 'sinner' for worrying so much about the way that I looked. The illness continued and my life revolved around the level of satisfaction I could achieve with the never-ending cycle of camouflage.

During this time I was unable to work and during one spell in hospital I had a relationship with another patient. This seemed to give me some reassurance that I was not completely undesirable and I went to live with him. This turned out to be a disaster as he had severe mental health problems. I got pregnant and ended up homeless with a 3 month old child.

When I reached 28 I met my second husband and the following years were much better with the distraction of my home, children, and career. However, social activities were still affected because it was difficult to be around others who were attractive and therefore 'adequate', unlike myself.

At 45 things took a serious turn for the worse with the failure of my second marriage and the BDD became severe. I was constantly checking my reflection in the mirror for up to 4 hours at a time. I felt repulsive and hideous and didn't feel that I looked human or deserved to live. I would constantly compare myself with others and look at old photographs, always focusing on what I considered to be my worse features, for several hours at a time. I thought about my ugly appearance every moment and again became suicidal. I couldn't talk to family and friends about my feelings because I was frightened that they would think that I was vain or mad. I couldn't understand why anyone could bear to look at me and not recoil in horror. The rituals around my make-up became worse and I would only remove it to immediately replace it. I styled my hair several times during the day and at times after washing and styling it would have to start all over again. I spent countless amounts of money on cosmetics, hair products, magazines, and salon treatments but the obsessive thoughts got worse. I felt that I could not get on and do anything if I could not get an acceptable image in the mirror and the more that I tried the more hideous I seemed to look and the more distressed I became.

My emotional state also caused physical problems such as irritable bowel syndrome, chest pains, weight loss and muscle pain. I was being treated at this time by a psychiatrist who tried different medication, including anti–psychotics (which didn't help), and referred me to two different psychologists. Most of the sessions were spent going over my past, which was not helpful, and I was discharged because I was making no progress. No one

seemed able to help me. At one stage it was suggested that I put post-it notes in my diary with affirmations like ' I am beautiful' but this had no effect because I didn't believe it – it just made me very angry. The last psychologist that I saw told me, in so many words, that I was a hopeless case because I had received so much help (i.e. from psychiatrists, psychologists, community practice nurses) and was not getting anywhere – this added to my feelings of shame and hopelessness.

I was convinced that unless I could change my appearance I would have to take my own life. Over a 3-year period I spent £20,000 on numerous cosmetic surgery procedures but it made me more ill, this time believing that it was my own fault rather than nature. I had two face lifts, a brow lift, chin tuck, laser treatment, upper and lower eye surgery, human tissue inserted under lip/nasal lines and many injectable treatments to plump out laugh lines. I put myself through hell but it seemed my only option, as I had nowhere else to turn. The feeling as I was going under the anaesthetic was wonderful as I felt that there was the chance that this operation might just work and I would be happy again. In reality this never happened. As the surgery healed the anxiety increased as I still saw the ugliness, made worse now by the guilt and shame of what I had done to myself. Even so, I still felt compelled to have more surgery because 'this time it might work and I have no other chance of life'.

Then one day as I was recovering from my last bout of surgery in 2000 I saw a programme on the TV about a girl with BDD. It seemed incredible that other people felt like me and that there was a name for my condition. I then found a BDD specialist who immediately took me into hospital for treatment. To be able to talk to someone who understood my condition after suffering from BDD for 25 years was overwhelming and the hope that this gave me was such a relief. After 6 months of SSRI medication and CBT I began to feel that I was improving and after a further 12 months of treatment I returned to work and felt back in control of my life. I did a lot of exposure therapy, which was difficult, but in time paid off. I also did a lot of cognitive work and after a while found that I was beginning to think in a more positive way. I eventually found that I was starting to believe that I am a worthwhile person and although I still hate the way that I look I realise that I can lead a normal life. I use the CBT skills on a daily basis to help prevent a relapse and I still take the SSRI.

I believe that if BDD had been recognised earlier and treatment had been available my life would have been happier and more fulfilling. It also had a terrible affect on my family. When I was in my twenties my father often said that I should 'pull myself together' and Mum sometimes remarked that I was being vain. When I was diagnosed it was difficult for them to admit that I had a mental health problem, I think that they may have felt partly to blame, that they had done something wrong as I was growing up, but they were good

and loving parents. So much time and money has been wasted on this illness and up until 4 years ago it has ruled my life. I am grateful to have now received appropriate treatment. It is an ongoing battle but I am finally in control of the BDD.

3.2 The perspective of people with OCD and BDD

As the testimonies demonstrate, OCD often develops slowly with no obvious 'cause', although it may also be triggered or exacerbated by a particular event. It may start with a few intrusive thoughts that lead to rituals and compulsions (for example, needing to check that a door is locked or over-zealous hand washing). In BDD the obsessive thought is related to the person's physical appearance and might mean that the person feels compelled to keep checking his or her appearance in a mirror. Over time these actions or compulsions become more entrenched and part of everyday life and the thoughts that accompany them become more intrusive and obsessive. The range of rituals may also increase; some may fade away completely, but be replaced by new or more complex ones.

The testimonies reveal that the actions or rituals become absolutely necessary to a person with OCD who believes that they serve various purposes. They can protect the person from possible sources of danger and contamination and make him or her feel 'safe'; but they also shield others from the person with OCD, who may consider that his or her actions may be dangerous or contaminating. The actions and rituals can also, at first, impose some kind of order on the world. If the rituals, whether actions or deliberate thought processes, do not take place, the person with OCD will experience overwhelming anxiety and fear. For someone with OCD his or her compulsions may become the only means of neutralising anxiety and preventing harm, although at the same time they can alienate him or her from other people as the obsessions and compulsions become all consuming. For people with BDD, compulsions, such as keeping the face 'masked' with make up or covering up or disguising a perceived physical flaw, are necessary to be able to confront other people.

As shown in the testimonies, OCD, if left untreated, usually become more severe. The symptoms may take on a more 'sinister' and troubling aspect (such as the thought of killing someone), leading the person to feel depressed and suicidal, and can take up a huge amount of time. While the actions are about retaining control, they also become controlling in themselves: people with OCD may feel that they are being 'taken over' by their obsessive thoughts. A person with these conditions may know that the thoughts are not entirely 'rational' but will nevertheless be compelled by them. OCD might also be exacerbated by significant life events, such as starting a new school or university, moving house, marriage breakdown, or health problems.

However other people with OCD may find that periods of stress at school or at work may temporarily take their minds off the obsessive thoughts.

The testimonies also vividly express how OCD can significantly affect a person's daily life and his or her relationships with other people. Although some people can disguise their OCD this comes at a great cost: they might decline social invitations that interfere with their compulsions or completely withdraw from the people closest to them. Other people with OCD might 'involve' other people in their compulsions (see the testimonies from carers below): carers and other family members might feel that they are 'controlled' by a person with OCD to the extent that they subscribe to his or her rituals. This puts considerable pressure on personal relationships. Some people with OCD can find that their lives are severely restricted and may confine themselves to a small part of the house or a single room that is deemed to be 'safe' or 'clean'. With BDD, a person may feel unable to be around other people who are felt to be more attractive.

A person with OCD might be reluctant to seek professional help due to feelings of embarrassment and shame and be worried about talking to someone about what might appear to be 'dangerous' thoughts. It is not uncommon for people with OCD to visit their GPs and not talk specifically about their obsessions and compulsions, but more generally about being anxious or depressed. If a GP diagnoses depression in such instances it is important that the reasons for the depression are explored otherwise diagnosis of OCD may be missed. Diagnosis of OCD may come as a relief for people who have not realised they suffer from a recognised and treatable illness although a diagnosis and a medical 'label' can in themselves bring problems.

Once the condition has been diagnosed, it is essential that the person with OCD works with a healthcare professional who has appropriate training in the treatment of OCD, and with whom the person can build up a relationship of trust and understanding and find the courage to confront their fears and anxiety. Particular types of antidepressants (SRIs) can reduce some of the anxiety and depression associated with OCD and may reduce the obsessions and compulsions to some extent. This can help the person to feel calm enough to benefit from psychological therapy. It is important that specialists listen and sympathise but are also clear, direct and positive about the treatment process. A sense of humour is usually helpful too. The OCD sufferer is often asked to do the things they most fear, therefore support, encouragement and understanding from the therapist, and family if appropriate, are vital. (See section 10. 5 for family and carer support.)

Some people with OCD report finding group therapy sessions particularly helpful because they can learn from seeing how other people react to treatment. In the same context, two therapists can be useful because one

maybe able to reinforce what the other is saying. OCD sufferers are often keen to argue about why their fears may be justified and it is harder to argue against two therapists both offering the same advice.

Once psychological treatment is underway and the sufferer has made some steps towards recovery, the sufferer has to learn to take some responsibility for their treatment, such as taking opportunities to confront on-going obsessions. Home-based tasks or 'homework' will usually be assigned to continue the treatment outside the therapy session. If the person with OCD learns how to tackle their fears and anxieties effectively, this can provide him or her with skills to cope with future relapses. A hierarchy of 'tasks' can help the most difficult seem less daunting once the person has some success tackling more moderate difficulties.

It is also vital that all aspects of OCD and BDD are treated. The nature of the conditions can vary over time and patient may suffer from a number of different types of compulsions. Any one compulsion should not be treated to the exclusion of others, unless the patient is shown clearly how the same principles can be used for different compulsions. It can also be helpful if the patient is treated in the area where the problem exists; this could mean that the contaminated articles from home are brought into therapy sessions or the therapist visits the patient's home.

OCD can be successfully managed, but both sufferers and the medical profession should bear in mind that the condition can last a lifetime and that even sufferers who have responded well to treatment may have periodic relapses. It can be helpful for a patient to know where to return for treatment during a relapse and to be aware of any maintenance treatment that is available. This would help to prevent the patient having to go through the process of waiting for or starting any specialist treatment from scratch.

Some sufferers find support groups beneficial. When meeting others with OCD, people can feel relief that they are no longer isolated. Some people with OCD report finding the most benefit from well managed support groups that focus on dealing with different aspects of the illness. This may include sharing experiences of treatment and useful self-help strategies. This type of group experience can also encourage people to seek or return to CBT. Moreover it can help people to keep up their efforts to confront anxiety provoking situations without resorting to compulsions or, in the case of BDD, camouflaging. Some sufferers say they have found support groups less beneficial when members of the group use the sessions primarily to complain about lack of treatment for OCD or suggest it is impossible to ever recover from OCD.

Although people with OCD might live with the condition all their lives, it is possible to return to fulltime education or work, conduct fulfilling

relationships and regain a sense of equilibrium. The very idea of 'recovery' can in itself improve a person's outlook.

3.3 Summary of the needs of people with OCD and BDD

- Early recognition and diagnosis of OCD and BDD, particularly in people who may be presenting with depression, anxiety or somatic complaints
- Respect and understanding from healthcare professionals
- Awareness and understanding from public sector services including educational establishments, local authorities, police and emergency services
- Healthcare professionals have adequate awareness of the condition and effective treatment for people with OCD and BDD
- Full information about the nature of OCD and treatment options
- Psychological treatment that directly addresses the OCD
- Group therapy sessions and the possibility of working with more than one therapist
- All aspects of the OCD are treated
- Information about what to do in case of relapse
- Information about support groups.

3.4 Personal testimonies from family members/ carers of people with OCD

3.4.1 Sophia's mother

My daughter Sophia was diagnosed with OCD a year ago when she was 16, which is when her behaviour was of particular concern. But in retrospect, it is quite possible to recognise some early signs of OCD and anxiety in Sophia that we did not then identify as a problem. For instance, after the death of the Princess of Wales she and her twin brother Tom both seemed more anxious

about the possibility of something happening to my husband and me. They would both need to be at the door as I left the house and sometimes Sophia would make me look at a piece of paper or take something with me. She had a set of things she said, like 'take care' or 'be safe'. She would often say good-bye many times. Tom did the same to some extent, but not as much, and with him it lessened over time. On another occasion when we were on holiday, Sophia became hysterical when we did not walk in single file along a roadside. The traffic was so incredibly sparse that her concern seemed laughable to us and we gently mocked her. I am sure that there were other signs that we passed over without realising.

Sophia's friendships never seemed easy at school; she was ultra-sensitive, becoming hysterically upset about perceived slights from teachers or friends, and was often reluctant to invite friends home or accept social invitations. She was a perfectionist, not confident in her own abilities and a private person about some of her deeper feelings. In 2003 I noticed that she found revision for exams difficult because she had an odd and laborious routine of writing out her notes ('I have to do it this way', she would say) that would never allow her to fit in all of her revision. This clearly began to cause her worries too, although she passed her exams that summer reasonably well.

However, 2 weeks after starting the autumn term, Sophia took an overdose of paracetamol and aspirin. She told me afterwards that she wasn't trying to kill herself, but that she felt so bad about life in general she wanted someone to notice; she also felt that by taking an overdose and making herself sick she could give some form to her inner pain (and that maybe she could get a day off school!). I took her to A&E where she was treated with great sensitivity by the staff and admitted to the children's ward (she was still 15) to be monitored. Before she was discharged the following day a psychiatrist talked to her, and then interviewed my husband and me. I later realised that this was the cause of a major and continuing trauma for Sophia. She felt that the psychiatrist was hostile, was trying to 'catch her out', or suspected she wanted to 'lock me up because I'm mad'. I don't know if the psychiatrist was in fact insensitive and handled the interview poorly, or if Sophia was paranoid about the experience and reading catastrophe into the scenario, which is something I now recognise she does as part of her OCD. Whatever the truth of the matter, it made her hugely reluctant to accept further counselling, medical or psychiatric advice.

The first thing we did, partly on the advice of the psychiatrist, was to immediately arrange a meeting with Sophia's school counsellor. Sophia was very reluctant but we all attended an hour's session as a family the day after the overdose. The counsellor was, we thought, brilliant, unpatronising in tone, and helpful to us all in untangling the reasons for the overdose. Sophia seemed relieved.

I instinctively felt that the best thing was for Sophia to continue with life in as normal way as possible although we all felt that the world had somehow cracked in two. We naturally felt unbelievably stupid not to have noticed how bad she had been feeling about life. The hospital, acting very promptly, had sent a report to our GP who phoned me the day after the overdose. She told us, as had the counsellor, that 'life as normal' was the best thing. Her call was reassuring, sensitive and useful to us.

We had a follow-up counselling session the week after the overdose. This was with the same counsellor we saw at Sophia's school, who was also a practice counsellor at our surgery. Our GP encouraged us to take up the option of private appointments with her because these were longer than those available on the NHS. Furthermore, she and our GP exchanged information about Sophia's condition, which was really useful.

Despite this support from the healthcare professionals, Sophia started to demonstrate other problems, including panic and anxiety attacks, which we found very frightening and deeply upsetting. Sometimes Sophia managed to get in for only an hour of school. Her sleep was now down to about 3 hours a night because she had nightmares and needed to keep the light on. She worried about chairs stored in a cupboard and had to keep checking them 'to make sure they were ok'. During this very difficult period I was often up with her late into the night so she could talk through what was in her head. It was incredibly exhausting and I saw the counsellor on my own to talk about Sophia's behaviour and to gather some strength to deal with things myself.

Our GP continued to be very helpful by supplying a letter to Sophia's school when her GCSE coursework deadlines could not be met. She saw Sophia regularly and offered her a referral to the Child and Adolescent Mental Health Services clinic. By this time Sophia had passed her 16th birthday and her treatment entered the adult phase. It was no longer up to us as parents to accept or not. Sophia initially rejected the offer of the referral but after a few weeks passed, there came some sort of watershed when she realised she wasn't getting better and certainly wasn't coping. She agreed to the referral. We were then told we were on a waiting list, but given no indication how long we might have to wait. This depressed Sophia hugely, not to mention us. We had felt the lifeline we had been offered had been roughly withdrawn. She feared that the NHS thought she was 'making everything up' and didn't believe her.

During this period Sophia's behaviour was still a great concern and in some ways it worsened. I wrote to our GP with a plea for help. I felt that if Sophia was to have any chance of living a reasonably normal 16-year-old life, attending school and taking her exams, she needed help immediately. I wasn't sure that as a family we would have been able to cope with much more in any case and I told our GP how stressed we all were. I became very

tired, tearful and often short-tempered, and there were times when my husband and I were sick with worry. As I was Sophia's only home confidante, I also had to relay to my husband and to Tom what I felt was going on in her head to help them understand. I think Tom thought that her behaviour was occasionally manipulative and attention seeking.

As a result of my letter, our GP asked for the referral to be expedited. She also obtained permission from a consultant to prescribe citalopram (10 mg per day later increased to 20 mg). The referral (with a community practice nurse (CPN) at a Family Centre) was scheduled to take place within a week of the prescription being made to ensure that any side effects were monitored (there were a few initially—sleeplessness and feeling sick and 'spaced out'). But I was also aware after the first week that a marked improvement had taken place, with significantly reduced levels of anxiety. (A little later this allowed Sophia to attend sessions of CBT with some hope of benefit and also helped her tackle school and exams.)

Our first appointment with the CPN was for the whole family. Subsequent sessions were for Sophia alone, but we also had a further three sessions as a family during 2004 (which Sophia found very stressful). The sessions were, I think, set up mainly to encourage Sophia to talk to us openly about her feelings and anxieties. I think the CPN took the view that if Sophia felt freer to talk, her emotions would be less likely to 'implode' as they had before with the overdose. CBT was recommended and appointments were set up to see a trainee psychologist working with the clinic.

It was while Sophia was receiving treatment at the Family Centre that Sophia was first diagnosed with OCD. The trainee psychologist explained it to Sophia and gave her an information sheet, but this was not an adequate method of explaining OCD to a family member or patient and the effect it can have on the whole family. At the next family session we asked specifically about OCD and for advice and strategies for helping Sophia deal with certain situations we had as a family experienced. But we were not offered much advice. At no point was the subject of 'enabling' (or not 'enabling') raised, or how we might help her when her anxiety compromised us in carrying out our ordinary lives safely or efficiently. The fact that Sophia's tendency to catastrophise comes from her OCD was never made clear. It would have been helpful to be told.

I have learned more about the condition from an OCD online forum than from anything else I have read. It gave me reassurance that there were so many others suffering in the same way. I think we were all a bit frightened by the diagnosis at first. It meant she really did 'have something'. But when we thought about it we saw it was actually positive to have a diagnosis as we knew much more about what we were dealing with.

Sophia had about 10 sessions of CBT in all to help her with some of her checking rituals (we never realised just how many of these she had as she had kept them well hidden). I think Sophia found these beneficial and I am not aware of her checking cupboard doors as often as she did. However, Sophia's obsessive thoughts are very deep and are hard for her to live with. I don't think this has been addressed through the treatment to date. I don't think she even recognises that the thoughts are part of the OCD. She seems to think they are separate.

The CPN has also continued to see Sophia regularly and has been very good about fitting in extra sessions if Sophia has been going through a particularly bad patch, as happened before exams in the summer and also just recently (we are about to move house).

Our understanding is that OCD may not be curable, but must be lived with. We see that if Sophia is in control of her own life she manages better and the fact that she is still subject to the decisions of we, her parents, is a problem. However, we are aware that if it wasn't the house move, she would have found something else to be worried about because her anxiety free-floats. We try to avoid making decisions based on her OCD but at times it's hard. If you know a decision is going to cause an anxiety attack that will make her and us stressed for hours, it's sometimes tempting to take the easy option and not make the decision.

I have found the year immensely wearing and have been unable to work since April because I find it virtually impossible to concentrate. I have become deeply involved with my daughter on a level that may not be good for either of us. However, given that it's clearly dangerous for her not to talk I feel I must be there for her at the moment. I have continued to insist as hard as I can that she continue to see a counsellor. I need her to see someone else because she simply must talk to someone that isn't me.

Tom has also found it hard. He and Sophia often have strident arguments, with Sophia ratcheting up the tension on purpose at times. He then feels he is to blame for causing a scene. He has shown more signs of stress recently, looking sad and withdrawn.

Nevertheless I feel reasonably confident that Sophia has a chance of living a 'normal' life. Although she does not see it that way herself, thinking she is unpopular, incompetent and unattractive, when I look at her with friends she is clearly one of the more mature girls amongst her peers and they look to her for opinions and support. I do worry that her abilities have already been severely hampered by OCD and that her academic results will never reflect her actual ability. But she has had a part-time job that she enjoys and she is clearly valued by her employer.

3.4.2 Peter's mother

My friendly, socially aware, intelligent, considerate 18-year-old son has OCD. The period leading up to Peter's diagnosis 3 years ago was the worst of my life. By the time OCD had escalated into an easily identifiable condition, our family was already exhausted from years of tantrums, which Peter had had since he was 2 years old. OCD was different though, and took us down an enormous slope that we found terrifying.

At its worst, Peter would return from school and then spend 2 hours in the bathroom trying to change out of his uniform. He would check his palms were clean, and then look at the backs of his hands. However, while looking at the backs of his hands the palms may have got dirty, so had to be checked again, then the backs of his hands again, and so on and so on. His hands would be red raw from unnecessary washing. He was unable to meet friends, play the piano, and go in the garden. He was unable to get onto a bus until he'd checked everyone getting on, to see he wasn't being followed. He thought any conversation we had inside a car could be heard by people on the pavement. He believed that his sister and I could get pregnant just by walking into his bedroom. If I got into his bedroom and straightened his bed cover he would think it was contaminated because it had touched a different part of the wall.

Before this, Peter got top marks at school, but this changed over a few months. First his writing changed from being very neat to scrawled and careless. Then he stopped being able to start work, or if he started he would be unable to complete it or would just lose it. He got through his GCSEs on the basis of his earlier work, but 6th form was a problem. Peter is currently re-taking the lower 6th and will complete his A levels next year. While we had once assumed he would go to university, and felt he would love the experience of living away from home, now we know that university at the moment would be pointless, and that it would be positively negligent to suggest he moved out. I feel that his academic and work potential is minimal until he can find a way of overcoming the block that OCD places on his being proactive in making progress on set projects. The more important and interesting the project, the more OCD kicks in to block it. There is great sadness when the person you knew as a child with fantastic potential has to delete a computer file just when he's actually managed to do some work, and walks away from AS levels with one grade 'E' when he has the intelligence and skills to do much better.

There were several aspects of Peter's care that are worth commenting on. After promising an urgent referral to a child and adolescent psychiatric clinic our GP forgot to send the letter. I found this out 5 weeks later. By this point we were desperate. Peter was moaning and crying with frustration. He sometimes climbed on the roof of our house, and often threatened to jump out of an upstairs window because he saw no point in living. He ran away from

home once and we had to get the police involved. One weekend we went to the out of hours GP who was the first to suggest OCD and the first to give any medication, which was buspirone.

When we eventually met the child and adolescent psychiatrist things started to get better. She was honest, calm and realistic (saying for example that when we went on holiday we would also be taking OCD with us, and that we needed to be practical), and engaged very well with Peter and the family, explaining to us clearly about the contribution we could make in helping Peter resist giving way to his compulsions. She emphasised that, however much we wanted to go along with the compulsions just to get some short-term peace, they would just continue, so in the long run the only way was to be calm but firm in resisting the urge to get involved in fulfilling Peter's compulsions. She was also positive in separating the OCD from the person, and saying that Peter's health came above his exam results (she wrote in advance to the examining board but I don't know if that had any effect).

Peter's psychiatrist also prescribed an SSRI (Seroxat) and described how it was believed to work. Although life is still not completely quiet in Peter's head, Seroxat has helped enormously by completely stopping the tantrums and reducing the effects of the OCD by cutting down the time spent trying to get out of a cycle of compulsions.

Peter also started having sessions of cognitive behavioural therapy. Although he found these sessions mildly interesting, the first person who worked with Peter was not inspiring, and did not involve the other family members. The strong implication was that this was Peter's private work and we shouldn't ask too much. We therefore had no idea how we were supposed to support Peter between visits. We asked to see someone else but the same thing happened. Occasionally Peter mentioned that he was trying to use a strategy he'd been taught, and we were very pleased, but this never lasted very long. The therapist did not appreciate that Peter was simply so exhausted by coping with OCD that he couldn't muster the strength of mind to confront it. I know many people are helped by CBT, but at this stage in Peter's life it was not the right thing for him.

Crucial to our survival as people and as a family is to have hope of some kind, but with OCD we have found that the focus of our hope has kept changing, and generally becoming narrower and lower. We imagine what study Peter could do, then revise it; we imagine what travel he might want to do, then find it's not possible; we suggest employment opportunities, then realise that we're too optimistic. There is a constant need for hope, but a limited number of options to focus on. On bad days, when hope almost disappears I feel that I can't keep living. (I am taking antidepressants—an SSRI.)

Peter's younger sister has also been very badly affected. She was often asked by him to help in the compulsions, and while she really wanted to respond positively, she knew from the psychiatrist that helping in the short term would not be helping in the long term and could entrench the compulsions. Peter's sister found this emotional battle very difficult and at one point she asked our GP for a course of antidepressants, which she has now stopped.

The situation at present is that Peter says he has no hope of winning against OCD, he feels he just has to learn to live with it. We feel however that Seroxat has had a positive impact; I cannot imagine how our family would still be together, or still be functioning at work or school if Peter wasn't taking it (and he would be very unwilling to stop taking it). Because of it the visible signs of OCD are now less obvious to us, allowing Peter the time and energy to take part in activities inside and outside the house. However, Peter is still struggling internally with the compulsions—we need to remain aware of this battle and somehow keep up our mental and emotional energy to support him and ourselves through the bad patches in the future.

3.4.3 Archie's father

Our son Archie was the light of our lives, he was growing normally, he was well behaved, intelligent and someone we were and still are very proud of. But from the age of about 5 or 6, it was clear that Archie was somehow different from other children, although at this time we had never heard of OCD.

From an early age he would ask challenging questions. All children ask 'why', but Archie wanted more detailed, more scientific answers and usually could not be fobbed off with a fairy-tale answer. At school he got by doing the minimum to get a good mark and generally school was easy for him. He had a clear sense of order and things to him would be in 'black or white', or right or wrong, with nothing in between.

When he was slightly older, if he appeared not to like something then there would be no compromise and he would only do things he did not like with great reluctance. It was not just a dislike, but an obsession. For instance, Archie would not like his hands to be dirty or sticky, and he would frequently wash his hands. This developed into a full-blown contamination obsession resulting in ritual hand washing to the point where his skin was in a dreadful state. Art classes therefore posed a 'danger' because he would get paint or clay on his hands. Football and sports were also a problem as this involved getting muddy and dirty. There were also other obsessions as yet unknown to us.

Archie was clearly different and therefore became a target for ridicule and bullying. Pupils would spit at him and as soon as he got home he would strip

off and have a long hot bath. Sadly Archie kept most of this to himself. Both the bullying and OCD affected his schoolwork, but despite this he did obtain some good exam results, if not as good as expected.

It was only when he was in his late teens and when Archie's obsessions had taken over and destroyed his life and ours that we first found out about OCD on a television programme. Had we known about OCD from the outset then maybe we could have done something to help him cope with it. Sadly, despite many trips to our GP both before and after the TV programme Archie was not diagnosed at this time. The only 'advice' we got from the GP was that he would get over it and that it was a teenager thing. All he did was to prescribe some hand cream for Archie's skin problem and then only in hopelessly small quantities. The GP would not even consider referring Archie to someone who might be able to recognise an underlying cause for his obsessions and do something about it. We had taken Archie to the GP on numerous occasions between the ages of 14 and 17 where he had ample opportunity to make a diagnosis or at least try to do something. We foolishly trusted the GP and accepted his advice, something we will regret to our dying days, but it was only after talking to the GP about Archie after he had left home some years later at the age of 22 and had been formally diagnosed (by another GP), that the GP admitted that he had never heard of OCD.

After leaving school Archie started a course in computer science at college, something he excelled in. However, Archie's rituals and contamination fears worsened and often he could not get to college on time for lectures or exams. It took him all day to get out of the house, showering until long after all of the hot water had gone and rituals were completed uninterrupted. Also there were problems leaving the house as this involved touching door handles and so on that were contaminated nor would he go out of the door if there was anyone outside that might see him. In addition there was his constant striving for perfection that meant he was never satisfied with his work so it was never finished or not handed in on time, although the work was of a much higher standard than required. The result was that he never finished the course although he continued to go to the college to unofficially use the facilities and for somewhere to go to get out of the house and to avoid us, his parents, because some of his obsessions and 'bad thoughts' were about us.

It was during the 'unofficial' time at college when Archie was finally diagnosed as having OCD (although we did not know about this at the time). With the help of a friend he went to a new GP and Prozac was prescribed. So at last, years after we had recognised that there was a problem, something could be done, or so we thought.

However, we now entered a very difficult period, which we describe as a 'living bereavement', when we lost our son to OCD. The most destructive and distressing factor was that Archie considered us to be dirty and contaminated.

The effect on our relationship with him was unbearable although still we sought an answer. A major blow came when he left home because of his obsessions about us. He tried to cut all links with us although we realised that it was not what he wanted—it was what his OCD told him he wanted.

Archie made rules about our relationship with him, some of which were that we were not to contact him, go anywhere near where he lived or ask him questions about his condition. The rules were all very one-way and on his terms, but we feared what would happen if we broke the rules. We did not know what was happening to him or how he was coping. Worst of all was the thought that he may commit suicide (something that we now know he was seriously considering). But with no income he was still dependant on us financially, so a very difficult link was maintained under considerable duress via the occasional phone call from him using a disguised voice to protect him from his obsessions surrounding us and, we think, to protect us from his harmful thoughts.

Archie had some very bad experiences, including his flat being flooded during the floods of Easter 1998. Archie has an obsession about faeces and to find his home full of raw sewage must have been unbelievably traumatic. Because of my job at the time I was the person with overall responsibility for the management of rescue, recovery and restoration of the affected area. In Archie's mind I was contaminated and he had to avoid me. He would make the lengthiest, time-consuming detours to miss routes or places my wife or I might use. In the end he left the area altogether and moved to another town. Because of the move and the difficulties the local authority and all local services were experiencing due to the flood, all contact was lost between Archie and the health and social services.

After several months Archie moved back to the town and contact with the social services was initially restored, but they were still overstretched and limited resources could be devoted to him. Because he no longer had contact with the health services he had no medication. He was living in absolute squalor because he would not throw household rubbish away and it accumulated in the flat where it smelled and was a cause for complaint from his neighbours. But worse still because of his obsession about faeces he would defecate into a plastic bag, which would also be kept in the flat (part of his obsession involved reserving the lavatory for urinating, not for disposing of faeces). Some of his neighbours perceived him to be 'abnormal' or 'mad' and ridiculed him; some were aggressive and subjected him to verbal abuse, threats of violence and carried out acts of vandalism such as breaking windows, throwing eggs at his door and putting dirt and dog faeces through his letter box. Because of this Archie would not respond to callers or letters, and tended to sleep during the day. He also incurred considerable debts. He had no income and his OCD dictated a very extravagant lifestyle. For example, underwear could not be washed because he considered it to be

contaminated, so it had to be discarded and new under garments worn every day. He also faced eviction.

These circumstances combined with the chronic situation at social services again resulted in a complete loss of contact with services. We thought at the time, and have since been proved right that he was becoming suicidal. At this point I went to the social services, although this was against his 'rules'. Fortunately they were sympathetic and took action. Archie was allocated a GP and seen by a psychiatrist. His medication was restarted and he was given limited practical help, although no psychological treatment was offered. The assessment was that he was not ready for a therapy such as CBT. That judgement was probably correct at the time. (However, that was in 1998 and I doubt if any further assessment has been carried out.)

There was no significant improvement over the next 4 years but Archie then developed pneumonia and was admitted to hospital and this forced him to allow social workers to make contact with us. This helped enormously with being able to communicate with him and opened a communication channel with his social worker. From then on communication and most importantly his trust in us improved. We found out that his most pressing worry was the substantial debt he had built up. Fortunately we were able to reduce the debt to a manageable level and build the trust between us by 'obeying' his new rules.

This situation however was most frustrating and heartbreaking. We knew that we could do so much to help if only he would allow us to. But if we did this, other than pander to his demands, would it trigger an adverse response leading to his situation deteriorating or even worse? However, we thought it was the only way to proceed and the only way that we could show him that we put his well-being first and to show him that he could trust us to do what was best, or under the restrictions imposed, what we thought would be best for him.

Over time trust did improve and we were able to talk openly and honestly with him about his situation. Gradually we got our son back, that is, he no longer tried to separate and distance himself from us and now we have a good, open parents/son relationship, something we have missed for many years.

There have been other small improvements more due to his own efforts than those of the medical services. He is still on medication and sees a psychiatrist periodically but sessions usually start with the psychiatrist saying that he would like to spend longer with Archie but he is running late so only has a few minutes, which is of no value. There is no suggestion of any further treatment or other sort of action or ways to progress. I have attended the last

two sessions and have suggested various treatments or options be considered but this was met with a rather contemptuous attitude by the psychiatrist.

Archie is 30 now, is still unable to work and has a very restricted life. However, we recently went out together for a meal to celebrate his birthday, something a short while ago we thought would be impossible. We are loving parents, supporting him the best we can, and have been able to improve the quality of his life recently although there is far more to do. We cannot become complacent because if we do we feel that he will become comfortable with his OCD and the restrictions it places on him.

If only Archie had been diagnosed when we first went to the GP. If only the GP was aware of OCD and had referred him to a specialist. If only the GP had done something then maybe Archie's life would have been so much better and we would not have suffered so much distress.

Having a family member with OCD has been a dreadful experience and it continues to have a severe and detrimental effect on our lives and health. We have encountered so much ignorance and lack of awareness amongst professionals and society as a whole that it has depressed us. We have suffered more than can be expressed in a few words, although that is so little compared with the torment sufferers endure—Archie has missed the normal life of a teenager and of a young man due to OCD.

3.4.4 Graham's wife

My husband's OCD shows itself particularly in extreme compulsive hoarding. Graham is 63 years old and in many ways is a wonderful husband—loyal, honest, conscientious and hard working—but he does not really acknowledge the problem either for himself or for the effect it has on me.

We were married in 1975, when Graham was 34 years old. He had been living with his parents in quite a cluttered house, and his mother tended to keep things like yoghurt pots and carrier bags. She was one of the Second World War generation and had the attitude that these things 'might come in useful one day and nothing should be wasted'. Although this was not the way I had been brought up, I did not at that time consider the situation to be particularly abnormal. After we were married I had begun to notice that Graham would also not discard empty packaging, tins and so on, or allow me to get rid of them. Odd bits of wood and other items began to pile up on the landing of our house, causing a fire hazard. He would also keep old newspapers, junk mail, and old letters and cards. By 1982, I was concerned enough about Graham's hoarding tendencies to write a letter to our GP at the time. The matter was never pursued.

When we moved house in 1985 everything came with us 'to be sorted when we get there'. Very little has, but a lot more has been added over the years. I have a constant battle to try and get Graham to dispose of anything. Our parents and an aunt have all died and left complete households for us to clear. Graham will not even let me throw away anything belonging to my own family. According to him, everything should is supposed to be sold and the proceeds shared with other family members, but there are many items that are of no value. We live in a cluttered, chaotic junkyard—several rooms in our very large house are completely inaccessible and have been for many years. He cannot find anything when he needs it and can become angry if he finds that I have moved or disposed of something. Maintenance is often impossible because the problems are inaccessible. Often, he will not trust anybody to do the work. He has to do it himself to ensure that it is up to his standard. He is such a perfectionist that things are achieved at a painfully slow speed, or not at all. If he does start on a job, he will focus on it to the exclusion of everything else, so other things get out of control.

In 1989, our younger son was referred to the Child and Family Clinic because of his difficult behaviour at school. One of the three psychiatrists whom we saw in the course of that year about my son's behaviour told me that Graham was suffering from obsessive-compulsive disorder. There was no further explanation, and nothing was said to him. No further reference was made to it, and there was no suggestion that it should be followed up. I appealed for help from our GP several times over the years, but I was told that nothing could be done unless my husband himself requested help, and Graham didn't think that he needed help at this time and has never acknowledged the need for help. He felt that I was the one who needed help.

During this time I felt that any needs I might have were neither considered nor understood until it was too late. I often felt desperate, helpless and depressed about the situation at home. I was ashamed and embarrassed if anyone had to come into the house, and my social life was also affected. I hated living like this and I broke down completely in 2000. Then all my GP could suggest was for me to take antidepressants, which I refused to do on the grounds that it would not go any way towards solving the underlying problems. Following a visit to an occupational psychologist, I had to ask to be referred to a psychiatrist for myself. I had not been made aware of what services were available, or offered any other help. One psychiatrist whom I saw said that I could not have further treatment as it was my husband who needed help. She said that she would phone my GP that evening, so I made an appointment for a few days later, and wrote to my GP to explain why. I told my husband that if he did not come with me, I would have to leave him. He did come, but the psychiatrist had not contacted the GP. She only realised what was happening because of my letter. That was how my husband was referred to a psychologist.

My husband was then referred to a trainee clinical psychologist in 2002, who diagnosed OCD, and clearly explained the reasons for the diagnosis in understandable terms. Graham was seen at home, which I thought was very helpful, and I was invited to join in with the sessions. The psychologist worked in practical terms, setting targets for each visit. We labelled everything in a certain area with 'Keep (if so, where?)', 'Sort (for a specific reason)' or 'Discard (if so, how and where?)'. She drew up a written contract in which we agreed to work together towards the targets, and which we all signed. The psychologist followed up the progress at each visit. She was very helpful, and Graham had begun to make progress, but when her training placement finished, her supervisor took over. Progress has now more or less ceased. It seems to me that this psychologist has not followed the same practice as his student, has ignored the contract and does not work in the same systematic way on the practical issues. He has not really managed to build any sort of rapport, and seems to have almost given up the attempt. He gives the impression of being extremely frustrated with Graham and spends a lot of time talking to him about 'normal people'. I told him that Graham neither knows nor cares what 'normal people' do, and he acknowledged that that was probably true. He is supposed to be liaising with my psychologist, but I do not believe that this happens with any regularity.

When my own psychiatrist retired, communication broke down regarding my treatment, and it took a letter to my MP to get the matter resolved. I was eventually referred to a psychologist, but she sees me alone at the clinic, and displays very little understanding of the issues involved. She has never met Graham, nor seen the situation at home. She has told me that the situation will not change, and I have to learn to live with it. She has also told me that American 'experts' on hoarding now say that the recognised treatments do not work. I find this thoroughly unhelpful, depressing and untrue. I have learned from my own experience and experiments that it is possible to change the situation gradually; and even the tiniest step is helpful in keeping away my depression.

Because I felt so let down by the NHS, Graham and I are now having private psychological treatment with a GP who is understanding and supportive. We have separate appointments on alternate weeks. It is costing us a fortune, but, from my point of view, is worth every penny—I feel as if I am gaining more control over the situation. It is also helping me to understand my husband's thought processes, so that I can see things from his point of view, and try to respond accordingly. I feel that this has also had an effect on my husband's behaviour.

I am learning to take control of my own emotional states, thus forestalling depressive episodes, and am now more aware of the possibility of learning from experience, and trying new strategies, instead of feeling defeated. This gives me hope for the future.

3.5 Understanding the impact of OCD on family members and carers

3.5.1 The role of family members and carers

'Caring' is the physical act of assisting someone who is ill, but it is also the emotions and feelings that a person has for another person. The degree to which it will be an emotional act will depend on the relationship of the carer to the person with OCD: carers can be close family members or partners, or they can be more distant relatives or friends. Their ability to care may depend on this relationship and their own personal circumstances, including their age (carers can range in age from teenagers to the elderly), financial situation, and health.

The time consuming and disruptive rituals resulting from OCD can affect a patient's whole family and individual relationships and can cause stress, frustration, anxiety, and anger in family members and carers. Many carers and family members often rebuke themselves for not having noticed the symptoms of OCD in their relative or friend before it is established. But it is quite common for people with OCD to hide their rituals and obsessions from the family, partly because of the embarrassment of carrying out apparently bizarre acts. Some people with OCD might deny that there is anything wrong if asked about their rituals or compulsions. In some other families, a family member may realise that something is wrong and seek explanation for it in their local library or on the Internet and in this way 'diagnose' the condition. The family member may encourage the sufferer to seek help, although some sufferers may not accept that there is a problem.

People with OCD may also actively 'involve' the carer/family member in his or her rituals and avoidance behaviour, sometimes surreptitiously. Carers and family members may become involved because of the distress and interference in activities, but compliance with this can 'entrench' the rituals or compulsions still further. Even when they are aware that involvement may be making things worse, carers and family members might feel that submitting to the patient's wishes is the only option. This can be frustrating for carers who feel that they have no other means of 'helping'. Others might go along with the rituals and compulsions in order to 'keep the peace'. The rituals can severely restrict and disrupt the lives of family members who might have to engage in, for instance, decontamination activities, leading to extra work in the home and/or extra expense incurred as a result of the ritual. Some family members can become the focus for the obsessive thoughts, which can be enormously upsetting. Others may try to ignore the condition because they do not understand it or think it will go away, while some may find OCD easier to understand if it is seen as a physical illness instead of a mental illness.

Whatever the circumstances however, family members and carers are inevitably drawn into the illness and the resulting environment. Many will want to help in whatever way they can and be included where appropriate in the treatment process. Indeed, people with OCD are likely to benefit from having a supporter who understands the condition and helps the person confront their fears, and who can continue to offer support after treatment to help maintain any improvements achieved. This might be a family member, but in cases where this is not appropriate a friend may be more suitable (occasionally, some people with OCD may lead such isolated lives that there is no one who can help).

In order to participate in the patient's care and the treatment process, family members and carers should be given full and ongoing information (where confidentiality permits) about how best to support the person with OCD and to cope with the condition (see section 10.5.1). Where appropriate, it may be helpful if they can be part of any decision making process.

Inclusion in the care and treatment process should take into account the carers' circumstances and environment to ensure that an undue burden is not placed on them. It is important to achieve a proper balance between sensitivity to the patient's concerns and avoiding compliance or involvement with the obsessive fears and compulsions. Attention should also be paid to additional problems affecting family members that the patient may not acknowledge.

Carers can feel as lonely and cut off as the person with OCD. This can be exacerbated by the stigma associated with mental health problems, especially where there is an unwillingness to talk about such problems with family members and others. Carers may themselves need support from the professional services including their GP and practice counsellors as a result of stress, anxiety and frustration of living with someone with OCD and providing long-term care. In very severe cases, some carers of patients with long-term OCD, particularly older and lone carers, may need respite care.

3.5.2 Information for carers

Carers and family members should be provided with clear information from healthcare professionals about OCD in a way that they can readily understand and so that they can provide care in the best way.

Carers have requested that the following type of information is made available:

- How best to help people with OCD
- How to deal with the rituals and obsessions of people with OCD

- Recommended treatments for OCD
- Possible side effects of treatment
- The likely prognosis for people with OCD
- How to identify suicidal intent.

3.5.3 Summary of carer needs

- Respect, understanding and sensitivity from all healthcare professionals, public sector services including educational establishments, local authorities, police and emergency services for people with OCD and their families and carers

- Recognition that OCD can be severe and can have a devastating effect on the lives of family members and carers

- Adequate information about the nature, course and treatment of OCD

- Information and advice about how not to become involved in a family member's rituals and compulsions

- Carers and family members may need support and treatment for anxiety and depression

- Information about support groups and voluntary organisations.

3.6 Specific issues for children and families

There is little evidence that life-events *cause* OCD, but in the individual vulnerable to OCD, times of life-stress may be times where symptoms worsen or relapses occur. In childhood this can particularly be around events that affect the family, school transitions, examination times or during difficulties with friendships or other relationships. Children experiencing learning problems, which have perhaps been undetected or where their needs have not been adequately met, may be vulnerable to exacerbations of OCD.

The transition from adolescence to adult life, with increasing independent living demands, can be an especially challenging time, particularly for the anxious individual. Young people with OCD have often been more than usually dependent on their parents, more cautious about exploring new experiences out of the home or with friends, or may have particular symptoms that make aspects of life difficult (for example, sharing a rented house/bathroom).

In the UK, mental health services for young people usually stop at age 16 or 18, and transfer occurs to general adult mental health services. For the young person with OCD, who is at a vulnerable stage of their development, continuity of services at this stage is essential. Child and adolescent mental health services should endeavour to link with the appropriate adult service well before discharge from child services needs to occur, to enable the young person to meet the new team, have joint appointments if necessary and so on.

Whatever the age of the person with OCD, clinicians working with the patient need to give time and attention to family members and carers. The younger the child, the more responsibility and decision making will rest with the adults, but even in young children, the child needs to feel involved in the treatment, able to express preferences and to take charge of aspects of their therapy. However, it is important to remember that young people need to develop autonomy and so for older children and teenagers, the therapist should assess sensitively and collaboratively the degree to which parents need to be involved and negotiating at each stage what information to share. Parents can be invaluable in ensuring therapy is successful, and indeed involvement is essential when they are closely involved in their child's rituals.

Many parents feel guilty about their child having OCD, and therapists need to take active steps to remove any sense of blame the parent might hold. It can be helpful for parents to understand that parents do not cause OCD but can inadvertently become involved in OCD. Most important, they need to understand that that they can be very helpful in the recovery process, and in maintaining good mental health in the future.

Many parents of children with OCD are anxious themselves, perhaps because of their own nature, but also because their child is distressed, has difficulty coping in important life areas, or has changed markedly. The issues raised by parental anxiety needs to be understood by therapists and dealt with sensitively and actively. Treatment for OCD always involves understanding anxiety, and often helping the child confront and deal with anxiety, rather than ritualising or 'running-away'. The anxious parent may find it very challenging to help their child learn to deal with anxiety in this way, and may need help themselves to learn effective strategies.

3.7 Sources of user and carer advice

In addition to professional healthcare services, users and carers may consider the other following sources of information. It is important that users and carers remain aware that the quality of information can be variable, and it may be important to rely on several rather than single sources.

- Books and videos aimed at sufferers, which may include practical advice and guidance on self-help for dealing with different types of obsessions and compulsions. There are also books written by people with OCD and carers of people with OCD.

- National and local support groups and self-help groups.

- National and international charities. There are currently a number of charities supporting those with OCD and other anxiety-related conditions, details of which can be found on the Internet. Some of these have information especially for carers and family members. Membership of such charities may offer newsletters, details of self-help groups and practical advice on getting specialist treatment.

- In the last few years there has been an annual UK conference organised by a group of charities aimed at people with OCD and carers.

3.8 Clinical practice recommendations

3.8.1.1 Treatment and care should take into account the individual needs and preferences of people with OCD or BDD, and they should have the opportunity to make informed decisions about their care and treatment. Where patients do not have the capacity to make decisions, healthcare professionals should follow the Department of Health guidelines – *Reference guide to consent for examination or treatment* (2001) (available from www.dh.gov.uk).

3.8.1.2 Good communication between healthcare professionals and people with OCD or BDD is essential. Provision of information, treatment and care should be tailored to the needs of the individual, culturally appropriate, and provided in a form that is accessible to people who have additional needs, such as learning difficulties, physical or sensory disabilities, or limited competence in speaking or reading English. **[GPP]**

3.8.1.3 Healthcare professionals should inform people with OCD or BDD, family and carers about local self-help and support groups for OCD and/or BDD, and encourage them to participate in such groups where appropriate. This may be particularly helpful as many people with OCD or BDD hide the symptoms of the disorder from others. **[GPP]**

3.8.1.4 Because OCD and BDD often affect not only the person with OCD/BDD but also their family/carers, healthcare professionals should promote a collaborative approach with the person with OCD/BDD and their family/carers, wherever this is appropriate and possible. **[GPP]**

3.8.1.5 In the treatment and care for people with OCD/ BDD, family members should be provided with good information (both verbal and written) about the disorder, its likely causes, its course and treatment. **[GPP]**

3.8.1.6 Assessment and treatment plans for people with OCD or BDD should, where appropriate, involve relevant family members and carers, assess the impact of their behaviours on others, (including and especially dependent children) and the degree to which carers are involved in supporting or carrying out behaviours related to the disorder. **[GPP]**

3.8.1.7 If dependent children are considered to be at risk of emotional, social or mental health problems as a result of the behaviour of parent with OCD and/or the child's involvement in such activity, independent assessment of the child should be requested. **[GPP]**

3.8.1.8 In the treatment of people with OCD or BDD, especially when the disorder is moderate to severe or chronic, an assessment of the carers' social, occupational and mental health needs should be offered. **[GPP]**

4 Methods used to develop this guideline

4.1 Overview

The development of this guideline drew upon methods outlined by NICE (Eccles & Mason, 2001; NICE, 2002b). A team of experts, professionals, people with OCD and a carer, known as the Guideline Development Group (GDG), with support from the NCCMH staff, undertook the development of a patient centred, evidence-based guideline. There are six basic steps in the process of developing a guideline:

- Define the scope, which sets the parameters of the guideline and provides a focus and steer for the development work
- Define clinical questions considered important for practitioners and patients
- Develop criteria for evidence searching and search for evidence
- Design validated protocols for systematic review and apply to evidence recovered by search
- Synthesise and (meta-) analyse data retrieved, guided by the clinical questions, and produce evidence statements
- Answer clinical questions with evidence-based recommendations for clinical practice.

The clinical practice recommendations made by the GDG are therefore derived from the most up-to-date and robust evidence base for the clinical and cost effectiveness of the treatments and services used in the management of OCD. In addition, to ensure a patient and carer focus, the concerns of people with OCD and carers regarding clinical practice have been highlighted and addressed by good practice points and recommendations agreed by the whole GDG. The evidence-based recommendations and good practice points are the core of this guideline.

4.2 The Guideline Development Group

The GDG consisted of professionals in psychiatry, clinical psychology, nursing and general practice; academic experts in psychiatry and psychology; and people with OCD and a carer. The guideline development process was supported by staff from the NCCMH, who undertook the clinical and health economics literature searches, reviewed and presented the evidence to the GDG, managed the process, and contributed to the drafting of the guideline.

4.2.1 Guideline Development Group meetings

Eighteen GDG meetings were held between June 2003 and December 2004. During each day-long GDG meeting, in a plenary session, clinical questions and clinical evidence were reviewed and assessed, statements developed and recommendations formulated. At each meeting, all GDG members declared any potential conflict of interests, and patient and carer concerns were routinely discussed as part of a standing agenda.

4.2.2 Topic leads

The GDG divided its workload along clinically relevant lines to simplify the guideline development process, and individual GDG members took responsibility for advising on guideline work for particular areas of clinical practice (psychological interventions, pharmacological interventions, BDD, children and young people).

4.2.3 People with OCD and carers

Individuals with direct experience of services gave an integral patient and carer focus to the GDG and the guideline. The GDG included two people with OCD and a carer. They contributed as full GDG members to writing the clinical questions, helping to ensure that the evidence addressed their views and preferences, highlighting sensitive issues and terminology associated with OCD, and bringing OCD patient research to the attention of the GDG. In drafting the guideline, they contributed to writing and editing the chapter on the experience of people with OCD and BDD, editing the first draft of the guideline's introduction, and identifying good practice points from the patient and carer perspective.

4.2.4 Special advisers

Special advisers with specific expertise in one or more aspects of treatment and management relevant to the guideline, assisted the GDG, commenting on specific aspects of the developing guideline and making presentations to the GDG. The names of those who agreed to act as special advisers are listed in the introduction.

4.2.5 National and international experts

National and international experts in the area under review were identified through the literature search and through the experience of the GDG members. These experts were contacted to recommend unpublished or soon-to-be published studies in order to ensure up-to-date evidence was included in the development of the guideline. They informed the group about completed trials at the pre-publication stage, systematic reviews in the process of being published, studies relating to the cost-effectiveness of

treatment, and trial data if the GDG could be provided with full access to the complete trial report. Appendix 4 lists researchers who were contacted.

4.3 Clinical questions

Clinical questions, developed from the scope, were used to guide the identification and interrogation of the evidence-base relevant to the topic of the guideline. The questions were initially drafted by the review team and the GDG chair, then refined or developed further by the GDG using informal consensus. The PICO (patient, intervention, comparison and outcome) framework was used to help formulate questions about interventions. This structured approach divides each question into four components: the patients (the population under study); the interventions (what is being done); the comparisons (other main treatment options); and the outcomes (the measures of how effective the interventions have been). Appendix 5 lists the clinical questions.

4.4 Systematic clinical literature review

The aim of the clinical literature review was to systematically identify and synthesise relevant evidence from the literature in order to answer the specific clinical questions developed by the GDG. Thus, clinical practice recommendations are evidence-based, where possible, and if evidence was not available, informal consensus methods were used (see section 3.6.6) and the need for future research was specified.

4.4.1 Methodology

A stepwise, hierarchical approach was taken to locating and presenting evidence to the GDG. The NCCMH developed this process based on advice from the National Guidelines Support and Research Unit (NICE) and after considering recommendations from a range of other sources. These included:

- Centre for Clinical Policy and Practice of the New South Wales Health Department (Australia)
- Clinical Evidence Online
- Cochrane Collaboration
- New Zealand Guideline Group
- NHS Centre for Reviews and Dissemination
- Oxford Centre for Evidence-Based Medicine
- Scottish Intercollegiate Guidelines Network (SIGN)
- United States Agency for Health Research and Quality
- Oxford Systematic Review Development Programme.

4.4.2 The review process

A brief search of the major bibliographic databases for recent systematic reviews and existing guidelines was first conducted to help inform the development of the scope. After the scope was finalised, a more extensive search for systematic reviews was undertaken. At this point, the review team, in conjunction with the GDG, developed an evidence map that detailed all comparisons necessary to answer the clinical questions. The initial approach that was taken in order to locate primary-level studies depended on the type of clinical question and availability of evidence.

After consulting the GDG, the review team decided which questions were likely to have a good evidence base and which questions were likely to have little or no directly relevant evidence. For questions in the latter category, a brief descriptive review was initially undertaken by a member of the GDG (see section 3.6.6). For questions with a good evidence base, the review process depended on the type of clinical question.

4.4.2.1 The search process for questions concerning interventions

For questions related to interventions, the initial evidence base was formed from well-conducted randomised controlled trials (RCTs) that addressed at least one of the clinical questions. Although there are a number of difficulties with the use of RCTs in the evaluation of interventions in mental health, the RCT remains the most important method for establishing treatment efficacy. The initial search for RCTs involved searching the standard mental health bibliographic databases (EMBASE, MEDLINE, PsycINFO, Cochrane Library) for all RCTs potentially relevant to the guideline. If the number of citations generated from this search was large (>5000), question-specific search filters were developed to restrict the search while minimising loss of sensitivity.

After the initial search results were scanned liberally to exclude irrelevant papers, the review team used a purpose built 'study information' database to manage both the included and the excluded studies (eligibility criteria were developed after consultation with the GDG). For questions without good quality evidence (after the initial search), a decision was made by the GDG about whether to: (a) repeat the search using subject-specific databases (e.g. CINAHL, AMED, SIGLE or PILOTS); (b) conduct a new search for lower levels of evidence; or (c) adopt a consensus process (see Section 3.6.6). Future guidelines will be able to update and extend the usable evidence base starting from the evidence collected, synthesised and analysed for this guideline.

Recent high-quality English-language systematic reviews were used primarily as a source of RCTs (see Appendix 11 for quality criteria). However, where existing data-sets were available from appropriate reviews, they were cross-checked for accuracy before use. New RCTs that met the inclusion criteria set

by the GDG were incorporated into the existing reviews and fresh analyses performed. The review process is illustrated in Flowchart 1.

Additional searches were made of the reference lists of all eligible systematic reviews and RCTs, and the list of evidence submitted by stakeholders. Known experts in the field (see Appendix 5), based both on the references identified in early steps and on advice from GDG members, were sent letters requesting systematic reviews or RCTs that were in the process of being published[2]. In addition, the tables of contents of appropriate journals were periodically checked for relevant studies.

4.4.2.2 Unpublished evidence

The GDG used a number of criteria when deciding whether or not to accept unpublished data. First, the evidence must be accompanied by a full trial report or sufficient detail to properly assess the quality of the data. Second, the evidence must be submitted with the understanding that it will be published in the full guideline. For example, the GDG did not accept evidence submitted as *commercial in confidence*. However, the GDG recognised that unpublished evidence submitted by investigators, might later be retracted by those investigators if the inclusion of such data would jeopardise publication of their study.

4.4.2.3 Search filters

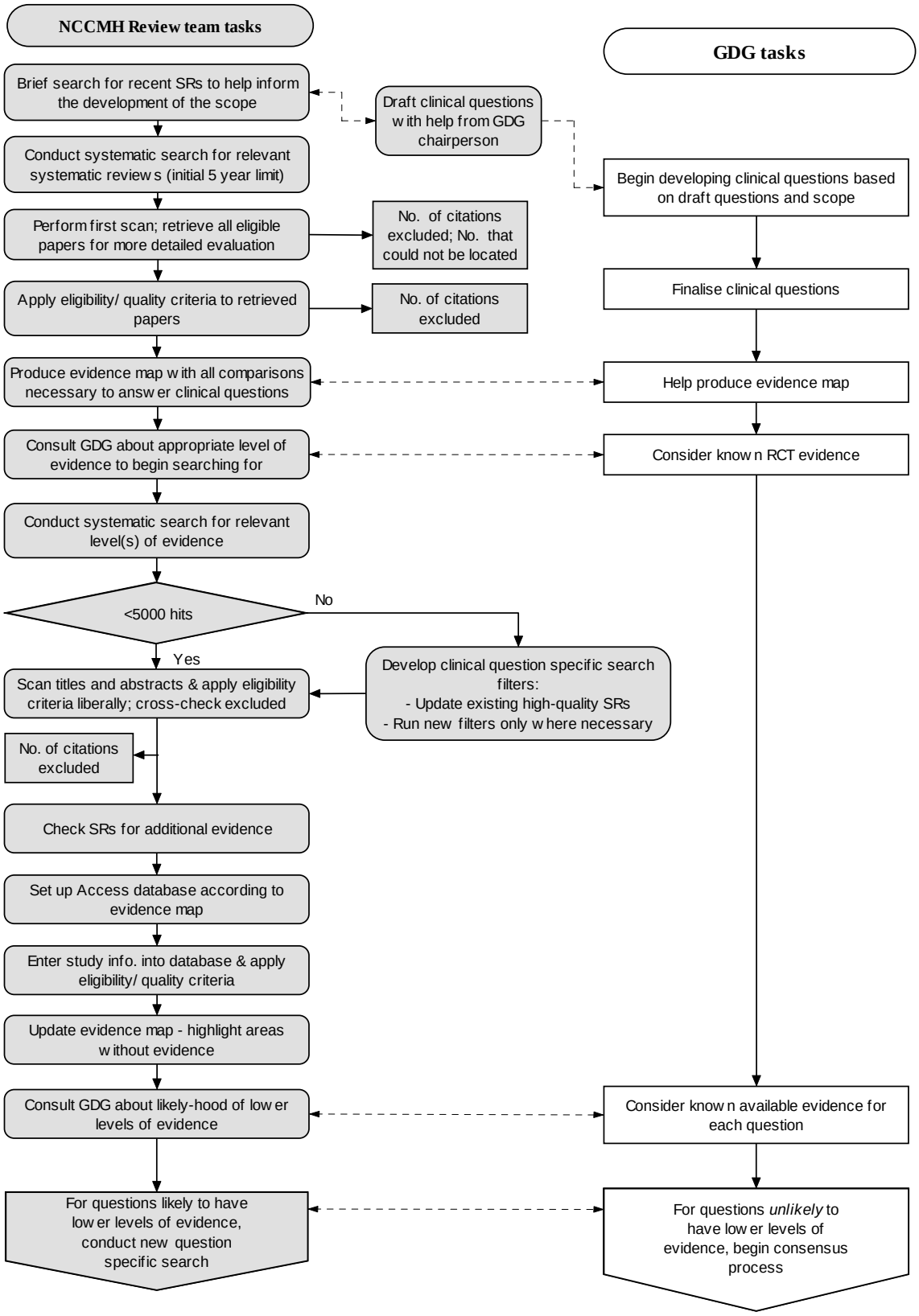
Search filters developed by the review team consisted of a combination of subject heading and free-text phrases. Specific filters were developed for the guideline topic, and where necessary, for each clinical question. In addition, the review team used filters developed for systematic reviews, RCTs and other appropriate research designs (Appendix 6).

4.4.2.4 Study selection

All primary-level studies included after the first scan of citations were acquired in full and re-evaluated for eligibility at the time they were being entered into the study information database. More specific eligibility criteria were developed for each clinical question (see appropriate chapter). All eligible papers were then critically appraised for methodological quality (see Appendix 8). The eligibility of each study was confirmed by at least one member of the group.

[2] Unpublished full trial reports were also accepted where sufficient information was available to judge eligibility and quality.

Flowchart 1: Guideline Review Process



4.4.3 Synthesising the evidence

Where possible, outcome data were extracted directly from all eligible studies, which met the quality criteria, into Review Manager 4.2.7 (Cochrane Collaboration, 2004). Meta-analysis was then used, where appropriate, to synthesise the evidence using Review Manager. If necessary, reanalyses of the data or sensitivity analyses were used to answer clinical questions not addressed in the original studies or reviews. For continuous outcomes, where more than 50% of the total number randomised in a particular study were not accounted for, the data were excluded from the analysis because of the risk of bias. In the case of dichotomous outcomes (except for the outcome of leaving the study early), the effects of high attrition rates were examined with sensitivity analyses.

Evidence tables, generated automatically from the study information database, were used to summarise general information about each study (see Appendix 15). Where meta-analysis was not appropriate and/or possible, the reported results from each primary-level study were also presented in the evidence tables.

Consultation was used to overcome difficulties with coding. Data from studies included in existing systematic reviews were extracted independently by one reviewer directly into Review Manager and cross-checked with the existing data-set. Two independent reviewers extracted data from new studies, and disagreements were resolved with discussion. Where consensus could not be reached, a third reviewer resolved the disagreement. Masked assessment (i.e. blind to the journal from which the article comes, the authors, the institution, and the magnitude of the effect) was not used since it is unclear that doing so reduces bias (Berlin, 2001; Jadad et al, 1996).

4.4.4 Presenting the data to the GDG

Where possible, the GDG were given a graphical presentation of the results as forest plots generated with the Review Manager software. Each forest plot displayed the effect size and confidence interval (CI) for each study as well as the overall summary statistic. The graphs were organised so that the display of data in the area to the left of the 'line of no effect' indicated a 'favourable' outcome for the treatment in question. Dichotomous outcomes were presented as relative risks (RR) with the associated 95% CI (for an example, see Figure 1). A relative risk (or risk ratio) is the ratio of the treatment event rate to the control event rate. An RR of 1 indicates no difference between treatment and control. In Figure 1, the overall RR of 0.73 indicates that the event rate (i.e. non-remission rate) associated with intervention A is about three-quarters of that with the control intervention, or in other words, intervention A reduces non-remission rates by 27%. In addition, the CI

around the RR does not cross the 'line of no effect' indicating that this is a statistically significant effect. The CI shows with 95% certainty the range within which the true treatment effect should lie.

Efficacy outcomes were calculated on an intention-to-treat basis (i.e. a `once-randomised-always-analyse' basis). This assumes that those participants who ceased to engage in the study – from whatever group – had an unfavourable outcome.

Continuous outcomes were analysed as standardised mean differences (SMD) (e.g. see Figure 2).

Figure 1. Example of a forest plot displaying dichotomous data

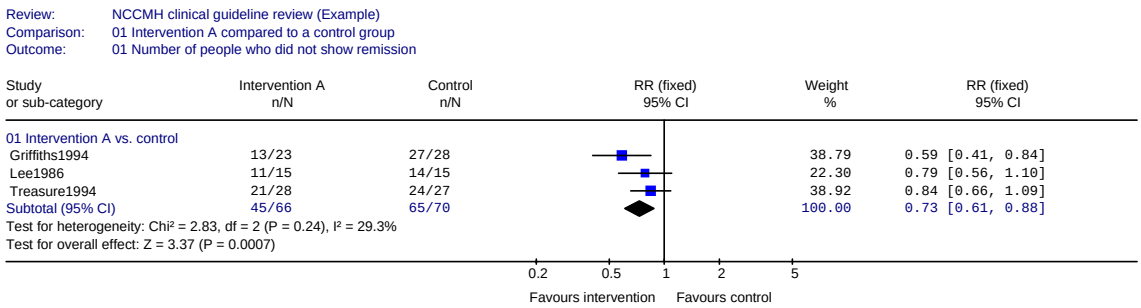
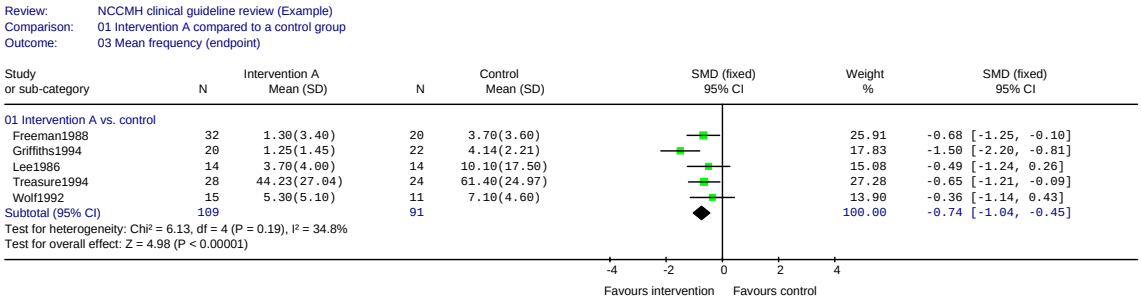


Figure 2. Example of a forest plot displaying continuous data



To check for heterogeneity between studies, both the I^2 test of heterogeneity and the chi-squared test of heterogeneity ($p < .10$), as well as visual inspection of the forest plots were used. The I^2 statistic describes the proportion of total variation in study estimates that is due to heterogeneity (Higgins & Thompson, 2002). An I^2 of less than 30% was taken to indicate mild heterogeneity and a fixed effects model was used to synthesise the results. An

I^2 of more than 50% was taken as notable heterogeneity. In this case, an attempt was made to explain the variation. If studies with heterogeneous results were found to be comparable, a random effects model was used to summarise the results (DerSimonian & Laird, 1986). In the random effects analysis, heterogeneity is accounted for both in the width of CIs and in the estimate of the treatment effect. With decreasing heterogeneity the random effects approach moves asymptotically towards a fixed effects model. An I^2 of 30% to 50% was taken to indicate moderate heterogeneity. In this case, both the chi-squared test of heterogeneity and a visual inspection of the forest plot were used to decide between a fixed and random effects model.

4.4.5 Forming and grading the statements and recommendations

The evidence tables and forest plots formed the basis for developing clinical statements and recommendations.

4.4.5.1 Intervention studies

For intervention studies, all evidence was classified according to an accepted hierarchy. Recommendations were then graded A to C based on the level of associated evidence, as a good practice point (GPP), or noted as coming from a previous NICE guideline or health technology appraisal (see Text box 1).

In order to facilitate consistency in generating and drafting the clinical statements the GDG utilised a statement decision tree (see flowchart 2). The flowchart was designed to assist with, but not replace clinical judgement.

Text Box 1: Hierarchy of evidence and recommendations grading scheme

Level	Type of evidence	Grade	Evidence
I	Evidence obtained from a single randomised controlled trial or a meta-analysis of randomised controlled trials	**A**	At least one randomised controlled trial as part of a body of literature of overall good quality and consistency addressing the specific recommendation (evidence level I) without extrapolation
IIa	Evidence obtained from at least one well-designed controlled study without randomisation	**B**	Well-conducted clinical studies but no randomised clinical trials on the topic of recommendation (evidence levels II or III); or extrapolated from level-I evidence
IIb	Evidence obtained from at least one other well-designed quasi-experimental study		
III	Evidence obtained from well-designed non-experimental descriptive studies, such as comparative studies, correlation studies and case studies		
IV	Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities	**C**	Expert committee reports or opinions and/or clinical experiences of respected authorities (evidence level IV) or extrapolated from level I- or II-evidence. This grading indicates that directly applicable clinical studies of good quality are absent or not readily available
		GPP	Recommended good practice based on the clinical experience of the GDG
NICE	Evidence from NICE guideline or technology appraisal	**NICE**	Evidence from NICE guideline or technology appraisal

Adapted from Eccles, M. & Mason, J. (2001) How to develop cost-conscious guidelines. *Health Technology Assessment* 5, 16; Mann, T. (Mann, 1996) *Clinical Guidelines: Using Clinical Guidelines to Improve Patient Care Within the NHS*. London: Department of Health.

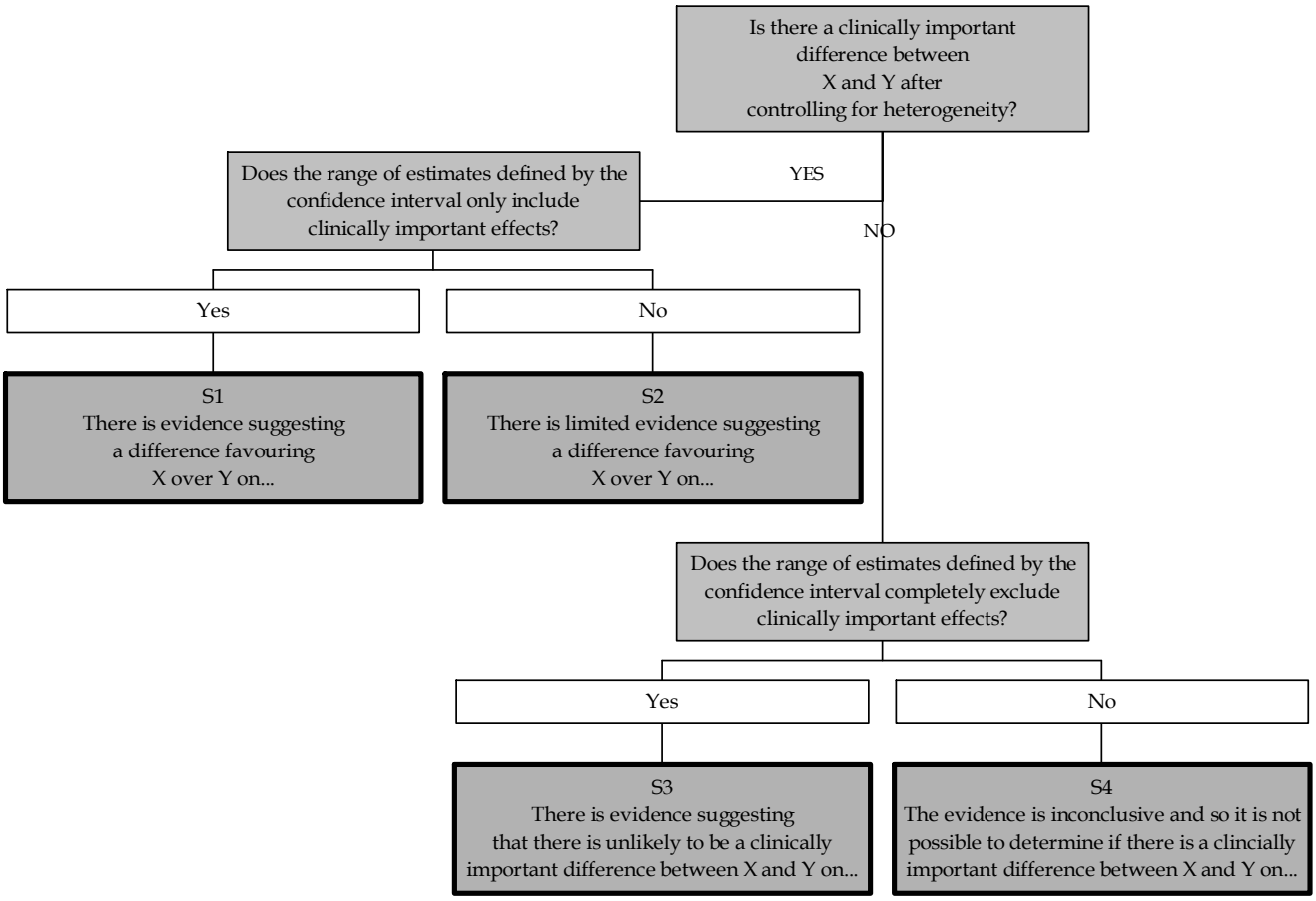
Using the decision tree (flowchart 2), the GDG classified each effect size as clinically important or not (i.e. whether or not the treatment is likely to benefit patients), taking into account several factors including statistical significance, the magnitude and precision of the effect, the trial population and the nature of the outcome.

Where heterogeneity between studies was judged problematic, either a random effects model was used or sub-analyses were conducted to examine the possibility of moderators.

In cases where an effect was judged clinically important, a further consideration was made about the strength of the evidence by examining the range of estimates defined by the CI. For level-I evidence, where the effect size was judged clinically important for the full range of plausible estimates, the result was characterised as *evidence suggesting a difference favouring x over y on…* (S1). For non-level-I evidence or in situations where the CI included clinically unimportant effects, the result was characterised as *limited evidence suggesting a difference favouring x over y on…* (S2). Where an effect size was judged as *not* clinically important and the CI did not include any clinically important effects, the result was characterised as *unlikely to be clinically important…* (S3).

Alternatively, if the range of estimates defined by the CI included clinically important benefits as well as no effect or harmful effects, the result was characterised as *inconclusive* (S4).

Flowchart 2: Guideline Statement Decision Tree



Once all evidence statements relating to a particular clinical question were finalised and agreed by the GDG, the associated recommendations were produced and graded. Grading allowed the GDG to distinguish between the level of evidence and the strength of the associated recommendation. It is possible that a statement of evidence would cover only one part of an area in which a recommendation was to be made or would cover it in a way that would conflict with other evidence. In order to produce more comprehensive recommendations suitable for people in England and Wales, there were times when the GDG had to extrapolate from the available evidence. This led to a weaker level of recommendation (i.e. B, as data were based upon level-I evidence). In addition, it is possible to have methodologically sound (level I) evidence about an area of practice that is of little direct clinical relevance or has such a small effect that it is of little practical importance. In this case, the evidence would attract a lower strength of recommendation (i.e. there would be necessity for extrapolation). It is important to note that the grading of the recommendation is not a reflection of its clinical importance or relevance.

The process also allowed the GDG to moderate recommendations based on factors other than the strength of evidence. Such considerations include the applicability of the evidence to the people in question, economic considerations, values of the development group and society, or the group's awareness of practical issues (Eccles et al, 1998).

4.4.6 Method used to answer a clinical question in the absence of appropriately designed, high-quality research

In the absence of level-I evidence (or a level that is appropriate to the question), or where the GDG decided (on the basis of previous searches or their knowledge of the literature) that there was unlikely to be such evidence, either an informal or formal consensus process was adopted. This process focused on those questions that the GDG considered a priority.

4.4.6.1 Informal consensus

The starting point for this process was that a member of the GDG identified, with help from the systematic reviewer, a narrative review that most directly addressed the clinical question. Where this was not possible, a brief review of the recent literature was initiated.

This existing narrative review or new review was used as a basis for beginning an iterative process to identify lower levels of evidence relevant to the clinical question and to lead to written statements for the guideline. The process involved a number of steps:

1. A description of what is known about the issues concerning the clinical question was written by one of the GDG members

2. Evidence from the existing review or new review was then presented in narrative form to the GDG and further comments were sought about the evidence and its perceived relevance to the clinical question
3. Based on this feedback, additional information was sought and added to the information collected. This may include studies that did not directly address the clinical question but were thought to contain relevant data
4. If, during the course of preparing the report, a significant body of primary-level studies (of appropriate design to answer the question) were identified, a full systematic review was done.
5. Following this, on occasions and as deemed appropriate by the GDG, the report was then sent to appointed experts outside of the group for peer review and comment. The information from this process was then fed back to the GDG for further discussion of the statements
6. Recommendations were then developed and could also be sent for further external peer review
7. After this final stage of comment, the recommendations were again reviewed and agreed upon by the GDG.

4.5 Health economics review strategies

The aim of the health economics review was to contribute to the guideline development process. Data on the economic burden of OCD and evidence of cost-effectiveness of the different treatment options for OCD were collected and assessed to help the decision-making process. See Chapter 9 for the detailed health economic review strategies.

4.6 Stakeholder contributions

Professionals, people with OCD, and companies have contributed to and commented on the guideline at key stages in its development. Stakeholders for this guideline include:

- Patient/carer stakeholders: the national patient and carer organisations that represent people whose care is described in this guideline
- Professional stakeholders: the national organisations that represent healthcare professionals who are providing services to people with OCD
- Commercial stakeholders: the companies that manufacture medicines used in the treatment of OCD
- Primary Care Trusts
- Department of Health and Welsh Assembly Government.

Stakeholders have been involved in the guideline's development at the following points:

- Commenting on the initial scope of the guideline and attending a briefing meeting held by NICE
- Contributing lists of evidence to the GDG
- Commenting on the first and second drafts of the guideline.

4.7 Validation of this guideline

This guideline has been validated through two consultation exercises. The first consultation draft was submitted to the NICE Guidelines Advisory Committee Panel, and circulated to stakeholders and other reviewers nominated by GDG members.

The GDG reviewed comments from stakeholders, the NICE Guidelines Advisory Committee, a number of health authority and trust representatives and a wide range of national and international experts from the first round of consultation. The GDG then responded to all comments and prepared a final consultation draft which was submitted to NICE, circulated to all stakeholders for final comments and posted on the NICE website for public consultation. The final draft was then submitted to the NICE Guidelines Advisory Committee for review prior to publication.

5 Psychological interventions for OCD

5.1 Introduction

Psychological interventions have been described for obsessive-compulsive disorder since the time of Freud. However, despite extensive writing about the disorder, OCD was generally considered to be virtually untreatable for over fifty years. In 1966 Victor Meyer described the successful treatment of two people with OCD by what would now be considered as the forerunner of modern day CBT treatments by changing cognitions and blocking compulsive rituals (Meyer, 1966). Following on from this, staff at the Maudsley Hospital developed behaviour therapy techniques in the early seventies that offered hope for the first time and demonstrated efficacy in a series of small quasi-experimental studies (Marks et al, 1975; Rachman et al, 1971; Rachman et al, 1973). Other researchers in the UK, Europe and North America rapidly experimented with a range of behavioural techniques (Emmelkamp & Kraanen, 1977; Foa & Goldstein, 1978; Rabavilas et al, 1979).

By the early eighties the common elements of several procedures that had been developed at different centres evolved into what is now known as exposure and response prevention (ERP)(see Steketee, 1994 for a review). Given the absence of effective treatments until the seventies, the early studies were so convincing that most researchers explored different ways of delivering the treatment components in trials looking at the differential efficacy of treatment formats rather than conducting randomised controlled trials to establish efficacy against non treatment or attention controls. In fact, with one or two exceptions, most of the controlled trials date from after 1990. With the rise of cognitive therapy in the eighties (Salkovskis, 1985), a variety of cognitive approaches have also been developed, mostly in combination with behavioural techniques (Freeston et al, 1996; Salkovskis, 1999; Salkovskis & Warwick, 1986; van Oppen & Arntz, 1994). While many therapists have continued to offer a variety of psychological approaches, there has been relatively little written about other approaches and even less research.

5.2 Behaviour and cognitive therapies

5.2.1 Introduction

More than 30 years of published research and a large number of authoritative accounts have led to a widely held consensus that behaviour therapy is an effective treatment for OCD. Indeed, the successful treatment of OCD was one of the early success stories for behaviour therapy. The early experimentation with a diverse range of behaviourally based procedures has

evolved into a therapy with a central technique, exposure and response prevention, that can be used in a variety of formats, including book and computer based self-help, group therapy, and individual therapy that ranges from minimal therapist contact or telephone contact through to intensive outpatient and inpatient regimes (Foa & Franklin, 2000; Himle et al, 2003; Lovell et al, 2000; Marks, 1997). Cognitive therapies have emerged more recently with the hope that they would improve the efficacy of behaviour therapy and provide an alternative to those who have difficulty in engaging in exposure and response prevention (Salkovskis & Warwick, 1986; Wilhelm, 2000). Many contemporary treatment approaches combine behavioural and cognitive approaches, but there are proponents of purer forms of both.

5.2.2 Current practice

During the rapid development of behaviour therapy in seventies, Professor Isaac Marks established a training programme at the Maudsley Hospital in 1972 to develop behaviour therapy skills among psychiatric nurses. This programme, and others that followed, established a strong core of skilled behaviour nurse therapists working in the NHS (Gournay et al, 2000). The work at the Maudsley and other centres also influenced professional training for psychologists and psychiatrists, among others, and so emerged a strong multi-disciplinary tradition for behaviour therapy in centres throughout the UK. Multi-disciplinary training in cognitive therapy developed in the early nineties and in 2004 there were over twenty post-qualification courses across the UK offering training in cognitive and behavioural therapies (www.babcp.org). Almost all basic professional training in psychiatry, psychology and nursing includes some training in these therapies. Thus, there is a large body of clinicians with knowledge of these approaches, although there are relatively few with specific expertise and experience in the application of cognitive and behavioural therapies to the treatment of OCD.

Therapists with the necessary expertise have traditionally been found in secondary and tertiary care settings. There is an unequal distribution of accredited therapists across the UK (Shapiro et al, 2003) and the picture is likely to be similar with trained but non-accredited therapists. However, there are increasing numbers of clinicians with CBT training in primary care and there are a number of recent training programmes to enable professionals in primary care with little CBT experience to provide assisted self-help to people with anxiety disorders (e.g. Lovell et al, 2003), including OCD.

5.2.3 Interventions included in the review

The following interventions were included:

- Behaviour therapy
- Cognitive therapy
- Cognitive behavioural therapy
- Rational-emotive therapy

5.2.4 Studies considered for review[3]

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of behavioural and cognitive therapies among adults with OCD. Thirty-seven studies were identified, of which 20 did not meet the inclusion criteria of the GDG. The 17 included studies provided efficacy data from 820 participants and tolerability data from 734 participants.

Of the included studies, two compared exposure and response prevention with systematic relaxation or anxiety management (GREIST2002, LINDSAY1997), and two compared cognitive behavioural therapy (CBT) with wait list controls (CORDIOLI2003, FREESTON1997). Four studies compared exposure and response prevention with cognitive therapy (COTTRAUX2001, MCLEAN2001, VANOPPEN1995, WITTAL2004), one study with CBT (VOGEL2004), two with rational-emotive therapy (EMMELKAMP1988, EMMELKAMP1991), and seven with other variants of behaviour therapy (DEARAUJO1995, EMMELKAMP1983, GREIST2002, HISS1994, KENWRIGHT (unpublished report), LOVELL1994, MEHTA1990).

All included studies were between 3 and 44 weeks long (mean length = 12 weeks). Patients were treated in an outpatient setting in eight studies; the setting was unclear in the remaining nine studies. In one study (FREESTON1997), the patients had obsessive symptoms only. Another study (LOVELL1994) was concerned with patients with rituals only. Four studies were conducted in the US, four in the Netherlands, three in the UK, and one each in Australia, Brazil, Canada, France, India and Norway. The average age of the participants was 35 years and the average duration of illness was 12.26 years.

Full details of the studies included in the guideline and the reasons for excluding are given in Appendix 15.

5.2.5 Psychological interventions versus control (systematic relaxation, anxiety management or wait list)

5.2.5.1 Behaviour therapy versus control (systematic relaxation, anxiety management or wait list)

5.2.5.1.1 Clinical evidence statements[4]

[3] Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication in capital letters, except where a study is *in press* or only submitted for publication, then a date is not used).

[4] The full list of all evidence statements generated from meta-analyses (and the associated forest plots) will be available on the CD-ROM that accompanies the guideline..

Efficacy[5]	Included studies
There is evidence suggesting a difference favouring exposure and response prevention over anxiety management control on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 1; N = 18; SMD = -2.89; 95% CI, -4.30 to -1.48). I	LINDSAY1997
There is evidence suggesting a difference favouring clinician-guided exposure and response prevention over systematic relaxation control on reducing obsessive-compulsive symptoms as measured by the self-reported Y-BOCS (K = 1; N = 121; SMD = -1.10; 95% CI, -1.49 to -0.72). I	GREIST2002
There is limited evidence suggesting a difference favouring computer-guided behaviour therapy over systematic relaxation control on reducing obsessive-compulsive symptoms as measured by the self-reported Y-BOCS (K = 1; N = 121; SMD = -0.68; 95% CI, -1.05 to -0.31). I	GREIST2002
There is limited evidence suggesting a difference favouring exposure and response prevention over controls on reducing obsessive-compulsive symptoms as measured by the Padua Inventory (K = 1; N = 18; SMD = -1.28; 95% CI, -2.32 to -0.24). I	LINDSAY1997
There is limited evidence suggesting a difference favouring exposure and response prevention over anxiety management control on reducing the impact of OCD on life and activities as measured on an interference rating scale (K = 1; N = 18; SMD = -3.16; 95% CI, -4.64 to –1.67). I	LINDSAY1997
There is limited evidence suggesting a difference favouring clinician-guided exposure and response prevention over systematic relaxation control on improving functioning as measured by the patient-rated Work and Social Adjustment Scale (K = 1; N = 121; SMD = -0.60; 95% CI, -0.96 to -0.23). I	GREIST2002
There is limited evidence suggesting a difference favouring computer-guided exposure and response prevention over systematic relaxation control on improving functioning as measured by the patient-rated Work and Social Adjustment Scale (K = 1; N = 121; SMD = -0.40; 95% CI, -0.76 to -0.04). I	GREIST2002
There is evidence suggesting a difference favouring clinician-guided exposure and response prevention over systematic relaxation control on the likelihood of treatment response, defined as 'much improved' or 'very much improved' on the Clinical Global Impressions scale (K = 1; N = 125; RR = 0.51; (95% CI, 0.38 to 0.69). I	GREIST2002
There is limited evidence suggesting a difference favouring computer-guided exposure and response prevention over systematic relaxation control on the likelihood of treatment	GREIST2002

[5] In the case of SMD or WMD, negative effect sizes favour the treatment group.

response, defined as 'much improved' or 'very much improved' on the Clinical Global Impressions scale (K = 1; N = 123; RR = 0.73; 95% CI, 0.59 to 0.91). I

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and controls on leaving the study early (K = 2; N = 125; RR = 4.47; 95% CI, 0.51 to 38.92). I

GREIST2002
LINDSAY1997

5.2.5.2 Cognitive behavioural therapy (CBT) versus wait list control

5.2.5.2.1 Clinical evidence statements

Efficacy	Included studies
There is limited evidence suggesting a difference favouring group CBT over wait list control on the likelihood of treatment response, defined as a 35% or greater reduction on the clinician-rated Y-BOCS (K = 1; N = 47; RR = 0.32; 95% CI, 0.17 to 0.59). I	CORDIOLI2003
There is limited evidence suggesting a difference favouring cognitive behavioural therapy over waitlist on reducing obsessive-compulsive symptoms as measured on the clinician-rated Y-BOCS in patients with obsessive symptoms only (K = 1; N = 29; SMD = -1.18; 95% CI, -1.98 to -0.38). I	FREESTON1997
There is evidence suggesting a difference favouring cognitive behavioural group therapy over wait list on reducing obsessive-compulsive symptoms as measured on the clinician-rated Y-BOCS (K = 1; N = 47; SMD = -1.18; 95% CI, -1.81 to -0.56). I	CORDIOLI2003
There is limited evidence suggesting a difference favouring CBT over waitlist control on reducing obsessive-compulsive symptoms as measured by the Padua Inventory (K = 1; N = 29; SMD = -0.83; 95% CI, -1.59 to –0.07). I	FREESTON1997
There is limited evidence suggesting a difference favouring group CBT over waitlist control on improving psychological quality of life as measured by the WHOQOL-BREF psychological subscale (K = 1; N = 47; SMD = -0.59; 95% CI, -1.18 to –0.01). I	CORDIOLI2003
There is limited evidence suggesting a difference favouring cognitive-behavioural group therapy over waitlist control on improving environmental quality of life as measured by the WHOQOL-BREF environmental subscale (K = 1; N = 47; SMD = -1.05; 95% CI, -1.66 to –0.44). I	CORDIOLI2003
There is limited evidence suggesting a difference favouring CBT over waitlist control on reducing anxiety symptoms as measured	

by the Beck Anxiety Inventory in patients with obsessive symptoms only (K = 1; N = 29; SMD = -0.87; 95% CI, -1.64 to – 0.10). I

FREESTON1997

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive behavioural therapy and wait list on the likelihood of leaving the study early (K = 2; N = 76; RR = 0.77; 95% CI, 0.24 to 2.49). I

FREESTON1997
CORDIOLI2003

Exposure to anxiogenic thoughts versus exposure to neutral thoughts

Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on the efficacy of treatment. I

Included studies
LOVELL1994

5.2.6 Psychological interventions versus other psychological interventions

5.2.6.1 Behaviour therapy versus cognitive therapy

5.2.6.1.1 Clinical evidence statements

Efficacy

There is limited evidence suggesting a difference favouring group behaviour therapy over group cognitive therapy on the likelihood of recovering at 12 months follow-up, defined as a reliable change on the clinician-rated Y-BOCS and a clinician-rated Y-BOCS score less than 13 (K = 1; N = 93; RR = 0.74; 95% CI, 0.6 to 0.92). I

Included studies
MCLEAN2004

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of leaving the study early (K = 4; N = 305; RR = 0.97; 95% CI, 0.63 to 1.47). I

COTTRAUX2001
VANOPPEN1995
MCLEAN2004
WHITTAL2004

5.2.6.2 Behaviour therapy versus cognitive behavioural therapy (CBT)

5.2.6.2.1 Clinical evidence statements

Efficacy

There is limited evidence suggesting a difference favouring cognitive behavioural therapy over behaviour therapy on reducing obsessive-compulsive symptoms at 6 months follow-up as measured on the clinician-rated Y-BOCS (K = 1; N = 35; SMD = 0.86; 95% CI, 0.16 to 1.56). I

Included studies
VOGEL2004

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on the likelihood of leaving the study early (K = 1; N = 35; RR = 6.74; 95% CI, 0.94 to 48.29). I

VOGEL2004

5.2.6.3 Behaviour therapy versus rational-emotive therapy

5.2.6.3.1 Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational-emotive therapy on the efficacy of treatment. I

Included studies
EMMELKAMP1988
EMMELKAMP1991

5.2.6.4 Self-exposure versus partner-assisted exposure therapy

5.2.6.4.1 Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on the efficacy of treatment. I

Included studies
EMMELKAMP1983

5.2.6.5 Imaginal plus live exposure/ response prevention versus live exposure/ response prevention

5.2.6.5.1 Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live ERP and live ERP on the efficacy of treatment. I

Included studies
DEARAUJO1995

5.2.6.6 Exposure plus relapse prevention versus exposure plus associative therapy

5.2.6.6.1 Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus relapse prevention and exposure plus associative therapy on the

Included studies
HISS1994

efficacy of treatment. I

5.2.6.7 Audiotaped exposure to anxiogenic thoughts versus audiotaped exposure to neutral thoughts

5.2.6.7.1 Clinical evidence statements

Efficacy	Included studies
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on the efficacy of treatment. I	LOVELL1994

5.2.6.8 Computerised ERP (BTSTEPS) plus scheduled support versus computerised ERP (BTSTEPS) plus requested support

5.2.6.8.1 Clinical evidence statements

Efficacy	Included studies
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus relapse prevention and exposure plus associative therapy on the efficacy of treatment. I	KENWRIGHT2004

Tolerability	Included studies
There is evidence suggesting a clinically significant effect of fewer patients being likely to leave the study early in BTSTEPS plus scheduled support when compared with BTSTEPS plus requested support (K = 1; N = 44; RR = 0.23; 95% CI, 0.08 to 0.7). I	***KENWRIGHT2004***

5.2.6.9 Family-based behaviour therapy versus patient-based behaviour therapy

5.2.6.9.1 Clinical evidence statements

Efficacy	Included studies
There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-Compulsive Inventory (K = 1; N = 30; SMD = -0.89; 95% CI, -1.65 to -0.14). I	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms at 6 months' follow-up as measured by the Maudsley Obsessive-Compulsive Inventory (K = 1; N = 30; SMD = -1.44; 95% CI, -2.25 to -0.62). I	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression as measured by the Zung	MEHTA1990

Self-rating Depression scale (K = 1; N = 30; SMD = -1.38; 95% CI, -2.19 to -0.58). I	
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression at 6 months' follow-up as measured by the Zung Self-rating Depression scale (K = 1; N = 30; SMD = -1.81; 95% CI, -2.67 to -0.94). I	MEHTA1990
There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = -0.91; 95% CI, -1.67 to -0.16). I	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work at 6 months' follow-up as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = -1.34; 95% CI, -2.15 to -0.54). I	MEHTA1990
There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving family adjustment as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = -0.78; 95% CI, -1.52 to -0.03). I	MEHTA1990

5.2.7 Therapist time in psychological interventions

5.2.7.1 Introduction

Although the efficacy of CBT (including ERP) is generally widely accepted, there is great variability in exactly how treatment is delivered. Group and individual treatments for OCD usually show a high degree of overlap in that planning for and carrying out exposure and response prevention exercises between sessions is believed to be one of the most important components. Likewise, ERP may be delivered through guided self-help using books or computer-based package with minimal support from therapists. Indeed, guided self-help may be conceptualised as an alternative delivery format of ERP rather than a distinct form of intervention. Finally, in individual formats, therapist input can vary from extremely brief (e.g. 15 minutes per week) to highly intensive (e.g. 2 hours per day, 5 days per week, for 3 weeks). Consequently, it is difficult to determine what an adequate 'dose' of CBT may be. The aim of this review is thus to examine treatment intensity. Although intensity can be defined in a number of ways such as frequency and duration of sessions, from a resource viewpoint, the therapist time per patient may be considered a proxy. As a result, a course of group CBT, over and beyond any advantages that may arise from the interaction of group members, may be considered a lower intensity treatment if the number of therapist hours per patient is below that of an equivalent course of individual CBT.

5.2.7.2 Current practice

ERP for OCD was developed in the 1970s in specialist units in the UK, often in an inpatient setting (see Marks, 1997), as well as elsewhere in Europe and the US. Since that time psychological treatment for OCD has been delivered mainly in secondary and tertiary care. Despite significant numbers of trained behavioural and cognitive therapists working within the NHS in England and Wales who can provide CBT, there remain difficulties in accessing these therapists in a timely manner and provision varies from one region to another (Shapiro et al, 2003). Consequently, there may be a role for forms of CBT requiring relatively little therapist input that can be delivered by a broader range of healthcare professionals. These professionals may not have been trained extensively in CBT, but if they have been trained to deliver the key components of the therapy in an effective way, access and availability could improve (Lovell & Richards, 2000). Brief CBT-based therapies have become increasingly available in primary care settings over the last 5 years for a variety of disorders and there is evidence of expansion. However, although the essential features of ERP are easy to grasp, its application in OCD can be complicated. Thus, even though low intensity treatments may be effective for some people, there is likely to be a role for the traditional individual CBT for those who have not adequately responded to treatment with lower intensity treatment. Furthermore, given the heterogeneity of clinical presentation found in OCD, there may be a proportion of people with OCD for whom low intensity treatments may not be suitable due to the need to substantially adapt approaches.

5.2.7.3 Method

The aim of this review was to determine whether the number of hours spent by a therapist per client in session predicted the efficacy of psychological interventions in patients with OCD. The review therefore included clinical trials on psychological interventions in patients with OCD. Due to time constraints, the review considered only studies on adult patients with OCD. For inclusion, studies had to report pre- and post-treatment scores of an outcome measure, such as the Y-BOCS, and the number of therapist hours per client. The latter was calculated as [the number of hours per session X total number of sessions] divided by the number of therapists per session. If the mode of treatment delivery was group therapy, then the number of therapists was divided by the number of patients per group.

Based on the distribution of the number of therapist hours per client across the studies, the studies were categorised into high, medium and low intensity groups. Interventions in which the number of therapist hours per client < 10 were classified as low treatment intensity interventions. Interventions in which the number of therapist hours per client ≥ 10 and < 30 were classified

as medium intensity interventions. Interventions with more than 30 therapist hours per client were classified as high intensity interventions.

5.2.7.4 Studies considered

A new systematic search for studies of psychological interventions in adults with OCD was conducted. The review considered studies that included exposure and/or response prevention and/or cognitive therapy as part of the intervention. The search identified 1,107 studies of which 1,037 were excluded as being irrelevant. Of the 69 studies that could potentially be included, 19 studies were excluded because the patients were children with OCD (n = 14) or had BDD (n = 5). Other reasons for exclusion were that the data was not extractable (n = 10), the number of therapist-hours per client could not be extracted (n = 5), the study had less than 5 participants in the treatment group (n = 1), the report was a case study (n =2), the report was a review of the literature (n = 1), the study tested a pharmacological intervention (n = 1), the study was in German (n = 1), .

The number of studies included in the review was 29 (BOUVARD2002, CHAMBLESS1999, CORDIOLI1999, DEARAUJO1995, EMMELKAMP1983, EMMELKAMP1988, EMMELKAMP1991, ENRIGHT1991, FOA1984, FOA1985, FOA2004, FREESTON1995, FRITZLER1997, GREIST2002, HISS1994, HUGHES2004, LINDSAY1997, MCKAY1996, MCLEAN2001, MCLEAN2004, NEZIROGLU2001, OCONNOR1999, ROSQVIST2001, ROTHBAUM2000, TAYLOR2003, VANOPPEN1995, VANNOPPEN1997, VANNOPPEN1998, VOGEL2004).

5.2.7.5 Sub-group analysis

Based on the 10 and 30 cut-off scores of the number of therapist hours per client, 11 interventions from 8 studies were classified as low intensity (CORDIOLI2003, EMMELKAMP1983_COUPLES, EMMELKAMP1983_PATIENT, ENRIGHT1991, FRITZLER1997, HUGHES2004, MCLEAN2001_BT, MCLEAN2001_CT, TAYLOR2003_DELAYED, TAYLOR2003_IMMEDIATE, VANNOPPEN1998), 22 interventions from 13 studies were classified as medium intensity (BOUVARD2002, CHAMBLESS1999, DEARAUJO1995_EXV, DEARAUJO1995_EXI, EMMELKAMP1998_BT, EMMELKAMP1988_CT, EMMELKAMP1991_BT, EMMELKAMP1991_CT, HISS1994_AT, HISS1994_RP, GREIST2002_CLINICIAN BT, LINDSAY1997, OCONNOR1999, ROTHBAUM2000, VANNOPPEN1997_GROUP BT, VANNOPPEN1997_MFBT, VANOPPEN1995_BT, VANOPPEN1995_CT, VOGEL2004_BT, VOGEL2004_CBT, WHITTAL2004_BT, WHITTAL2004_CT) and 10 interventions from 8 studies were classified as high intensity (FOA1984_ERP, FOA1984_EX, FOA1984_RP, FOA1985_EXI, FOA1985_EXV,

FOA2004, FREESTON1997, MCKAY1996, NEZIROGLU2001, ROSQVIST2001). The pre- and post-treatment scores on the study's key measure of efficacy were entered into the Review Manager software, which was used to estimate heterogeneity across all studies and to produce an effect size for each sub-group calculated as the standardized mean difference (SMD) between the pre- and post-treatment scores.

Across all studies, there was statistically significant heterogeneity ($\chi^2 = 174.46$, df = 44, $p < 0.00001$; $I^2 = 74.8\%$).

The effect size for each sub-group was:

Low intensity group: N = 261, SMD = -0.93, 95% CI, -1.11 to -0.75
Medium intensity group: N = 461, SMD = -1.44, 95% CI, -1.59 to -1.29
High intensity group: N = 157, SMD = -1.65, 95% CI, -1.91 to -1.38.

There was significant heterogeneity within the medium intensity sub-group ($\chi^2 = 38.3$, df = 19, $p = 0.005$). Therefore, sensitivity analysis was used to examine the effect of removing outliers. By excluding LINDSAY1997, heterogeneity was reduced ($\chi^2 = 29.9$, df = 18, $p = 0.04$), while the effect size remained similar (N = 452, SMD = -1.42, 95% CI, -1.57 to -1.27).

To examine whether number of therapist hours predicted treatment efficacy, a meta-regression analysis was performed controlling for the year of publication, study design (RCT or non-RCT), and treatment modality (individual or group). The number of therapist hours per client significantly predicted change in efficacy scores following treatment, z = -2.09, $p = 0.04$, after controlling for publication date, $z_{date} = 0.29$, $p = 0.77$, study design, $z_{study\ design} = -1.30$, $p = 0.19$, and treatment modality, $z_{treatment\ modality} = 0.93$, $p = 0.35$. When a sensitivity analysis was conducted by removing outliers (LINDSAY1997), therapist hours still significantly predicted change in efficacy scores, z = -2.24, $p = 0.03$, after controlling for publication date, $z_{date} = 0.24$, $p = 0.81$, study design, $z_{study\ design} = -1.17$, $p = 0.24$, and treatment modality, $z_{treatment\ modality} = 0.82$, $p = 0.41$.

5.2.7.6 Limitations

There are important limitations to this review as (1) therapist time is only a proxy for treatment intensity, (2) therapist time is confounded with treatment format (group vs. individual), (3) these studies were not designed to address this particular question, and (4) although all studies included some degree of ERP, the exact content was unknown. Furthermore, as in almost all studies of CBT, there is generally insufficient control over the degree of patient adherence and particularly of the quantity of work conducted between sessions. Properly designed prospective studies that examine all these factors

are needed. In particular, the severity and complexity of OCD needs to be taken into account.

5.2.8 Clinical summary

As noted in the introduction, despite 35 years of research into the cognitive behavioural treatment of OCD, there is a limited number of randomised controlled trials that compare active treatments with controls. Those that exist indicate that clinician-guided exposure and response prevention is an effective treatment for OCD. One study found that family-based behaviour therapy, including ERP, was superior to individual therapy. This study was conducted in India and these findings may not necessarily apply widely in the UK, although experts would certainly consider involving the family in many cases.

There is also some evidence for computerised exposure and response prevention and some indication that there should be scheduled support as fewer people left the study early when it was delivered with scheduled brief support sessions rather than support on request.

There is as yet little evidence for either cognitive therapy or CBT from randomised control trials against control conditions although the effects observed from head to head trials suggest that effects post-treatment are of a similar order to exposure and response prevention. There is support for CBT (including exposure and response prevention) for obsessive thoughts. Likewise, group CBT has been shown to be effective compared with wait list control.

Currently there is no evidence showing that cognitive therapy is more or less effective than exposure and response prevention alone. One study did find that adding cognitive therapy to exposure and response prevention midway through treatment resulted in a better outcome at six –months' follow-up. However, a second study found that group exposure and response prevention was superior to group cognitive therapy at 12 –months' follow-up. This question may never be answered in a convincing way as it is difficult to deliver two distinct, non overlapping-treatments. Many modern exposure-based treatments do include a high informational content and the strategies used to engage people in ERP can resemble cognitive therapy. Likewise, most current cognitive therapy explicitly seeks behaviour change but is not operating within a habituation paradigm. Although there is as yet no research on those who refuse, fail to engage with, or do not respond to ERP, cognitive therapy may yet have a role to play for these individuals, either as a new modality, or as a means of ultimately engaging them in exposure and response prevention.

The review of therapist hours as an indicator of intensity revealed that the effect sizes (–between pre- and post-treatment) for all three treatment intensity bands were large or greater (>0.8 SMD), Further, patients receiving more therapist hours of cognitive and behavioural intervention per patient were more likely to improve on OCD symptom severity compared with patients receiving fewer therapist hours. This effect was strongest when comparing patients receiving fewer than 10 therapist hours per patient with patients receiving more than 10 therapist hours per patient of psychological intervention. These findings together suggest that there may be benefit in more intensive forms of therapy over less intensive forms of therapy when calculated in terms of therapist hours per client.

There are important implications for stepped care as less intensive therapies clearly have a role to play, particularly in primary care and there may be benefits for some people receiving care in this setting rather than in secondary care or specialist services. There is also clearly a role for more intensive treatment, usually individual therapy, which may be found in increasingly specialist settings, especially for those for whom initial lower intensity interventions have proved inadequate.

5.3 Psychoanalysis

5.3.1 Introduction

Until the 1960s psychoanalysis was widely viewed as the treatment of choice for neurosis and so for all of the anxiety disorders including obsessive-compulsive disorder. Psychoanalysis for OCD was heavily influenced by the work of Freud's 14 papers on the subject, including his classic case history of the 'Rat Man' (Freud, 1909). Freud's conceptualisation of obsessional phenomena focuses on anxiety derived from unresolved Oedipal conflicts resulting in anal-sadistic regression, which the ego fends off through defence mechanisms such as reaction formation, intellectualisation, undoing and isolation (Barth, 1990; Freud, 1909). Although some authors have expanded on, or offered other psychoanalytic formulations of OCD (Esman, 2001; Wells, 1990), the Freudian conceptualisation remains powerful today. As late as 2001 Burgy wrote: 'Attention is focused on the intrapsychological structure and conflicts, so that Freud's theory of an internal dependence on the superego instead of the external dependence on people around continues to prevail in obsessive compulsive neurosis' (Burgy, 2001).

Psychoanalysis focuses on the identification, clarification and alteration of the defence mechanisms that maintain the anxiety (Salzman, 1997). Treatment emphasises the relationship between therapist and patient and involves transference, counter-transference and interpretation (Salzman, 1983). Traditional psychoanalysis involves a highly trained practitioner who provides up to four sessions a week over a period of up to several years

although somewhat less frequent sessions and shorter courses of treatment may be offered.

Psychoanalysis for OCD remains a treatment option in parts of England and Wales where such services are available. However, over the last 3 decades psychoanalytic therapy has become less frequent as a treatment for OCD. This may be because the emergence and widespread acceptance of treatments such as specific pharmacological therapies and CBT for OCD have provided a range of treatment options for which there is an evidence base.

5.3.2 Studies considered for review

No systematic reviews or meta-analyses of the effectiveness of psychoanalysis for OCD were found therefore a narrative review was undertaken. Only papers written in English were considered and a total of 64 papers were reviewed (dating from 1912 to 2002). None of the papers considered for review was an RCT or cohort study and so the evidence reviewed consists of single case reports, a few case series, and theoretical reviews.

5.3.3 Descriptive review

Many of the articles describe single case reports detailing the nature of the analysis in both adults (Boehm, 2002; Cela, 1995; Finell & McDougall, 1985) and children (Fingert Chused, 1999; Karush, 1998). A few articles were found citing case series, though the maximum number of patients in these studies was three (Fingert Chused, 1999; Lang, 1997). Moreover, Lang (1997) used these cases to illustrate unconscious determinants rather than to report outcome. Some authors described group analytic treatment (Schwartz, 1972; Wells, 1990), but these case reports do not report outcome in any systematic way. A number of the single cases provided a description of ongoing analysis with a patient (Boehm, 2002; Deri, 1990; Willick, 1995), but did not provide any measure of outcome.

The single case reports that reported successful outcome (Chatterji, 1963; Parfitt, 1999) did not report clinical process or any measure of outcome. More importantly, the individualised nature of psychoanalytic interventions makes it almost impossible to replicate. Several of the articles and case reports acknowledge the limitations of psychoanalysis for OCD in terms of its ineffectiveness, and argue against its utility in integrating it with other interventions (Fingert Chused, 1999; Gabbard, 2001; Kay, 1996). For example, psychoanalysis has been used to help engage people with OCD to undertake other forms of treatment such as CBT (McCarter, 1997), and in conjunction with pharmacotherapy and behavioural treatments in both adults (Leib, 2001) and children (Gold-Steinberg & Logan, 1999). However, such combination treatments tell us little about the effect of each individual intervention, and

the absence of outcome measures precludes any conclusions regarding treatment efficacy.

Despite generally poor outcomes many of the case reports report intense analytic therapy ranging from one to four sessions per week over periods extending from 6 months to 19 years (Boehm, 2002; Cela, 1995; Juni, 1987). Esman (2001) sums up the evidence base: 'In our series of 21 patients who were collectively the recipients of more than one century of psychodynamic treatments, we had no reason to be …optimistic'.

5.3.4 Clinical summary

There is no evidence of efficacy or effectiveness for psychoanalysis in the treatment of OCD. Given the lack of evidence and the resource required for such intensive treatment, there is doubt as to whether it has a place in mental health services for OCD.

5.4 Other psychological interventions

5.4.1 Introduction

OCD is often a chronic debilitating disorder, impacting on social, personal and professional aspects of the person's life. Not only is there a cost to the self in terms of quality of life, but also to society with unemployment and sickness. The mainstays of treatment for OCD are behaviour therapy comprising exposure and response prevention (ERP), or pharmacotherapy, in particular selective serotonin reuptake inhibitors (SSRIs) (March et al, 1997). Although efficacy and effectiveness have been demonstrated with both these treatment modalities several difficulties limit their utility. Emmelkamp and Foa (1983) cited that 30% of OCD sufferers declined behaviour therapy while Kozak et al (2000) reported during the course of their study that 40% of those who commenced behaviour therapy did not complete treatment. ERP can be unpleasant and distressing to clients and may lead to discontinuation of therapy. When taking into account those who refuse or dropout of treatment and those who do not benefit immediately or relapse, researchers have estimated that those treated successfully with behaviour therapy is around 55% (Stanley & Turner, 1995). In a review of controlled trials with SSRIs, Pallanti et al (2002a) reported that up to 40 to 60% of patients do not have a satisfactory outcome. Adverse side effects are also problematic for some and may lead to discontinuation of treatment or poor compliance. Furthermore relapse is high when pharmacotherapy is discontinued (Kozak et al, 2000).

Although there is a strong evidence base for cognitive and behavioural therapies, people with OCD who are seeking help frequently indicate that they would like to be informed about a range of other treatments, their efficacy and availability. If first-line treatments are unavailable, have been

unsuccessful, or discontinued due to distress or intolerable side effects, it may be useful for clinicians and people with OCD to know about other psychological interventions that could be beneficial.

5.4.2 Current practice

Current practice in the treatment of OCD using other psychological interventions and alternative/complimentary therapies is difficult to determine, as there is a paucity of literature. To date there has been one published RCT on an alternative therapy (yogic meditation) in the treatment of OCD in adults. No RCTs have been published on any of the other psychological interventions that have been used with OCD. Furthermore, no well-designed single case studies have been published on either the other psychological interventions or alternative/complementary therapies. The literature is limited and restricted to clinical case reports of one or more adults with OCD. Subsequently, the numbers of OCD clients treated using other psychological or alternative/complimentary therapies is small, with minimal replication for any given approach.

Although CBT is available in many places in the NHS in England and Wales, people with OCD continue to be offered a range of other psychological treatments. There are a number of factors why this is likely to be the case. First, many psychological therapists are trained in modalities other than CBT and so would tend to offer the therapy in which they are trained. Second, some psychological therapists have a level of knowledge of a range of therapeutic modalities and choose an eclectic position. They would use elements of different therapies based on the way that they understand the person's particular problems and their view of what may be effective elements. Third, referrers to psychological services may choose to refer to particular therapeutic modalities based on their own training and understanding of the person with OCD. Finally, if choice is available, people with OCD may choose particular therapeutic modalities that either conform to their own understanding of their difficulties or because other options may appear too daunting or anxiety provoking.

5.4.3 Interventions included in the review

The following interventions were considered:

- Yogic meditation
- Hypnosis
- Homeopathy
- Marital therapy
- Transactional analysis
- Systemic therapy
- Integrated psychological approach
- Paradoxical intention

- Gestalt therapy
- Counselling
- Morita therapy
- Group cognitive analytic therapy
- Virtual reality therapy.

5.4.4 Studies considered for review

A systematic review of all relevant literature identified one RCT on yogic meditation (Shannahoff-Khalsa et al, 1999) and 26 articles of clinical case reports without comparative treatments that described other psychological and alternative/complementary therapies in the treatment of OCD in adults (Becker & North, 1998; Churchill, 1986; Dormaar, 1987; Erickson, 1973; Fields, 1998; Gomez de Setien, 1982; Gomibuchi et al, 2000; Hafner, 1982; Harvey & Green, 1990; Johnson & Hallenbeck, 1985; Keiley, 2002; Mitzman & Duigan, 1993; Moore & Burrows, 1991; Morphy, 1980; Norland, 1988; Pelton, 1987; Pollard, 2001; Reichenberg & Ullman, 2000; Reichenberg & Ullman, 1999; Reichenberg & Ullman, 1998a; Reichenberg & Ullman, 1998b; Scrignar, 1981; Sheinberg, 1988; Stern, 1973; Walker, 1981; Yoder, 1994). The systematic review did not identify any literature for acupuncture, neuro-linguistic processing, reflexology or aromatherapy in the treatment of OCD in adults.

5.4.5 Randomised controlled trial of yogic meditation

A randomised controlled trial (RCT) of yogic meditation techniques (Shannahoff-Khalsa et al, 1999), compared the efficacy of two meditation protocols in the treatment of OCD. The two protocols were kundalini yoga meditation and relaxation response plus mindfulness meditation. The study was in two phases, the first phase being a 3-month RCT, the second involving the groups merging and the efficacious protocol being employed for a further 12 months. Inclusion and exclusion criteria were determined prior to enrolment and a DSM-III-R diagnosis of OCD was established. The primary outcome measure used was the Y-BOCS. Groups were matched for age, sex and medication status before randomisation of group to therapy. Fourteen adults completed phase one, seven in each group. The authors reported that kundalini yoga meditation was an effective treatment for OCD when compared with baseline measures and also when compared with the alternative protocol. However, these published results should be viewed with caution as subsequent reanalysis with Review Manager (Cochrane Collaboration, 2004) failed to support these assertions apart from a scale measuring a construct labelled Purpose in Life.

5.4.6 Clinical case reports

5.4.6.1 Hypnosis

There were eight published articles on hypnosis; all were case studies reporting on one or more adults (Churchill, 1986; Dormaar, 1987; Erickson, 1973; Harvey & Green, 1990; Johnson & Hallenbeck, 1985; Moore & Burrows, 1991; Scrignar, 1981; Walker, 1981). Three of these case reports were not reviewed further because in two (Erickson, 1973; Walker, 1981) the diagnosis was obsessional personality and the third (Dormaar, 1987) did not include a DSM/ICD diagnosis or equivalent detailed clinical description of OCD. Three of the remaining case reports reported using a multifaceted approach combining hypnosis with one or more other treatment modalities, namely: conjoint family therapy (Churchill, 1986); the behavioural technique of flooding (Scrignar, 1981); relaxation, cognitive and behavioural strategies and pharmacotherapy (Moore & Burrows, 1991). Five of the clinical case reports involved only one client (Churchill, 1986; Harvey & Green, 1990; Johnson & Hallenbeck, 1985; Moore & Burrows, 1991; Walker, 1981), one of which involved the family in treatment (Churchill, 1986). Another article (Scrignar, 1981) reported on the treatment of two people.

Although these case studies generally reported improvement, their validity is severely restricted by the fact that they are uncontrolled case reports and have numerous methodological deficiencies such as a lack of standardised diagnoses, combined treatment modalities, and lack of recognised outcome measures.

5.4.6.2 Homeopathy

Five published articles on homeopathy were identified; three were clinical case reports of one adult (Norland, 1988; Reichenberg & Ullman, 1999; Reichenberg & Ullman, 1998b) and two were clinical case reports of one or more young persons (Reichenberg & Ullman, 2000; Reichenberg & Ullman, 1998a). One of the articles (Reichenberg & Ullman, 1999) also reported on the cases of three young persons. All of the adult case reports reported improvement in symptoms. However, it is difficult to draw meaningful conclusions as the numbers were small and the use of standardised diagnostic or outcome measures was not reported.

5.4.6.3 Marital/ couple therapy

Two articles were identified on marital therapy (Hafner, 1982; Stern, 1973) and one on couple therapy (Keiley, 2002). All were clinical case reports of one or more adults. Hafner (1982) reported on the treatment of five inpatients using a multi-modal treatment programme: behaviour therapy, individual and group psychotherapy, conjoint marital and family therapy, social skills

training and pharmacotherapy. Improvement in symptoms was reported during inpatient stay, however all clients relapsed on returning home. Stern and Marks (1973) reported on the treatment of a single case of obsessive compulsive neurosis with marital discord using contract therapy and Keiley (2002) described the use of affect regulation and attachment focused treatment with one OCD client and their partner. Again a variety of factors such as small numbers, other interventions, and lack of specified outcome measures of OCD symptoms hamper interpretation of these reports.

5.4.6.4 Transactional analysis

Two articles on transactional analysis were identified (Gomez de Setien, 1982; Pelton, 1987); both were primarily a description of the treatment technique. There was insufficient information on diagnosis, assessment and outcome of treatment to allow any conclusions about treatment effects.

5.4.6.5 Other therapies

Other case reports have appeared in the literature for psychological and alternative/complimentary therapeutic strategies, but no single technique has more than one case report. Those identified were systemic therapy (Sheinberg, 1988), an integrated psychological approach (Fields, 1998), paradoxical intention (Yoder, 1994), gestalt therapy (Morphy, 1980), counselling (Pollard, 2001), morita therapy (Gomibuchi et al, 2000) and group cognitive analytical therapy (Mitzman & Duigan, 1993). Many of the difficulties encountered in the above reviews were found in these case reports thereby limiting any conclusions that could be drawn.

5.4.6.6 Virtual reality therapy

Becker and North (1998) described the development of the virtual reality therapy system (VRT-2002) for the treatment of various psychological disorders including OCD through exposure methods. A brief description of one scene for OCD was provided and a second was said to be under development. Evaluation of the effectiveness and efficiency of VRT-2002 in the treatment of OCD has yet to be reported. Further, given the often idiosyncratic nature of concerns in OCD, it may be difficult to develop the required stimuli for more than a few subgroups of patients who have common concerns.

5.4.7 Clinical summary

The literature revealed very few clinical case reports of other psychological therapies in the treatment of OCD in adults. Those that were identified were primarily small in sample size, often including only one client. No clinical case reports used standardised outcome measures, although one report,

(Fields, 1998), used a recognised validated measure at baseline. The largest number of clinical case reports identified for any one intervention was hypnosis: eight articles were identified and five reviewed. Although improvement was reported in these cases, in three cases other concurrent treatment was provided, leading to difficulty attributing causality. No comparative clinical case reports were identified.
There is insufficient evidence to support the use of other psychological therapies, hypnosis, or homeopathy therapies as routine treatments for the core features of OCD. This lack of evidence is in contrast with a much larger evidence base for cognitive and/or behavioural therapies although there are important limitations to the latter. Based on current evidence, ensuring access to adequate cognitive and/or behavioural therapies would appear to provide people with OCD with the best chance of improvement through psychological therapies.

5.5 Psychological interventions for children and young people with OCD

5.5.1 Introduction

The early identification and treatment of children and young people with OCD is essential, especially as symptoms have been found to result in significant distress and functional impairment, leaving the young person with difficulties in pursuing social relationships, education and hobbies (Laidlaw et al, 1999; Leonard et al, 1993). Research has shown that OCD in young people is under-recognised and often goes untreated (Flament et al, 1988), leading to increased risks of morbidity and comorbidity in adulthood (Rasmussen & Eisen, 1990b).

Several interventions have been used in the treatment of children and young people with OCD. These include behavioural interventions such as exposure, response prevention, flooding, extinction, shaping and operant techniques. Cognitive behavioural protocols have been developed that include behavioural techniques, as well as incorporating anxiety management, cognitive restructuring and parental involvement (March et al, 1994). Finally, there have been several uncontrolled studies that have used other therapeutic approaches, almost always in conjunction with behavioural techniques, that include systemic and psychodynamic approaches (Crago, 1995; O'Connor, 1983), individual psychotherapy (Warneke, 1985), insight-orientated therapy (Friedmann & Silvers, 1977; Willmuth, 1988), family therapy (Dalton, 1983; Goodman, 1988), hypnotic induction (Kellerman, 1981), social skills training (Hallam, 1974), and unspecified milieu therapy (Apter et al, 1984).

Management and treatment is often complicated in children and young people with OCD as they frequently have other comorbid problems (Last &

Strauss, 1989). For instance, in a survey of 70 children and young people with OCD, only 26% had OCD as their sole disorder (Swedo et al, 1989). Frequently children and young people also present with secondary anxiety disorders, including generalised anxiety disorder, separation anxiety in younger children, and social anxiety in young people. Furthermore, a major complication of OCD in young people is the development of social avoidance and withdrawal from family and friends. Aggressive behaviour and temper tantrums can also be a management problem, and frequently occur when rituals are interrupted. Therefore the careful assessment and consideration of treating comorbid problems is necessary in clinical practice.

Although OCD in young people is similar to that found in adults, there are various developmental differences that are important to consider in the management and treatment of children and young people. Young children's obsessional thoughts are more likely to be characterised by 'magical' or superstitious thinking (for example, 'if I don't count up to 20 my parents will die'). Treatment needs to take account of the child's developmental stage in order to engage them in a collaborative working relationship. Age-appropriate delivery may also include the use of metaphors in order to explain difficult psychological processes, for example the use of the 'white bear' experiment to explain the persistence of intrusive thoughts (Shafran, 1997).

Researchers have also utilised game formats of exposure and response prevention interventions in order to appeal to younger children (Moritz, 1998). Young children are also more present-orientated than adults and may be less motivated to engage in difficult activities in order to achieve future positive changes.

Furthermore, the child's age-appropriate dependence on his or her family is a key difference between the presentation of children/young people and adults. As outlined in a study by Bolton and colleagues (1983), the family are almost always involved in the young person's rituals, although the nature and extent of involvement often varies. Involvement can range from relatively mild involvement, such as occasionally providing the child with reassurance, to extreme involvement where the parent is highly immersed in all of the child's rituals. The degree to which families can become involved with the child or young person's OCD can lead to difficulties in management and can affect treatment compliance. Frequently, one parent is more involved than the other and the parents may not work together as a team (Bolton et al, 1983; Dalton, 1983). Some families react to their child's presentation of OCD by becoming critical and rejecting, alienating the child and adding to management difficulties. Parental behaviours may inadvertently reinforce and maintain the child's difficulties with OCD, and are often a source of family upset and discomfort. These factors have led to the frequent inclusion

of family members in most of the CBT treatment protocols. Family members are often provided with psychoeducation and may be encouraged to participate in some processes of therapy, such as exposure and response prevention.

5.5.2 Current practice

Current practice for the treatment of young people with OCD varies widely according to professional orientation, training, and availability of resources. Multi-disciplinary teams may offer a range of therapeutic interventions, often combining psychological and pharmacological treatments. CBT protocols for children and young people have been developed that can guide practitioners on the practice of cognitive and behavioural strategies with young people (March & Mulle, 1998). Most young people are treated on an outpatient basis unless the extent of their symptoms, distress and interference in their daily levels of functioning warrants an inpatient admission.

The aim of psychological treatment for children and young people with OCD is to reduce symptoms, distress and interference in daily functioning. A positive outcome would also include improved social, educational and family functioning. Treatment is further aimed at improving the young person's coping skills, and teaching strategies to prevent future relapse.

OCD in children and young people has received relatively little empirical study compared with adult OCD, and many questions currently remain unanswered by the literature. To date there have been only two published randomised controlled trials of the psychological treatment of children and young people with OCD, and no systematic replication studies. Caution is needed when interpreting the results of the published studies as many have significant limitations that reduce the confidence that can be placed in their results. Although most studies report a large percentage of responders to treatment, this is often measured by different criteria across studies, and may not always represent clinically meaningful change. The participants often have a range of comorbid difficulties, may be receiving concurrent pharmacological treatment, or are receiving components of two or more treatment approaches. Furthermore, most studies are of young people, thus making it difficult to generalise to younger children. The methodological weaknesses found in most studies of childhood OCD indicate caution must be exercised in making statements about treatment efficacy.

5.5.3 Interventions included in the review

The contemporary psychological treatment approaches identified by the GDG and included in the review are:

- Behaviour therapy
- Cognitive behavioural therapy
- Cognitive therapy
- Family therapy
- Psychoanalysis, psychoanalytic/psychodynamic/supportive/insight-orientated psychotherapy.

5.5.4 Studies considered for review

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of behavioural and cognitive therapies among children with OCD. The search identified one study (BARRETT2004).

The study compared individual cognitive behavioural family therapy with group cognitive behavioural family therapy and wait list control. The duration of treatment was 14 weeks long, with 3 and 6 months' follow-up. The mean age of the participants was 12 years.

5.5.4.1 Individual cognitive behavioural family therapy versus wait list control

5.5.4.1.1 Clinical evidence statements

Efficacy	Included studies
There is limited evidence suggesting a difference favouring individual CBFT over wait list on reducing obsessive-compulsive symptoms as measured on the clinician-rated CY-BOCS (K = 1; N = 46; SMD = -2.73; 95% CI, -3.55 to -1.91). I	BARRETT2004
There is limited evidence suggesting a difference favouring individual CBFT over wait list on improving family functioning as measured on the MFAD mother's rating scale (K = 1; N = 32; SMD = -0.93; 95% CI, -1.67 to -0.19). I	BARRETT2004

5.5.4.2 Group cognitive behavioural family therapy versus wait list control

5.5.4.2.1 Clinical evidence statements

Efficacy	Included studies
There is limited evidence suggesting a difference favouring group CBFT over wait list on reducing obsessive-compulsive symptoms as measured on the CY-BOCS (K = 1; N = 53; SMD = -2.54; 95% CI, -3.28 to -1.81).	BARRETT2004

I

There is limited evidence suggesting a difference favouring group CBFT over wait list on reducing depressive symptoms as measured on the CDI (K = 1; N = 38; SMD = -0.78; 95% CI, -1.46 to -0.11). I BARRETT2004

There is limited evidence suggesting a difference favouring group CBFT over wait list on improving family functioning as measured on the MFAD mother's rating scale (K = 1; N = 40; SMD = -0.78; 95% CI, -1.45 to -0.11). I BARRETT2004

5.5.4.3 Individual cognitive behavioural family therapy versus group cognitive behavioural family therapy

5.5.4.3.1 Clinical evidence statements

Efficacy	Included studies
There is limited evidence suggesting a difference favouring group CBFT over individual CBFT on reducing anxiety as measured by the MASC (K = 1; N = 42; SMD = 0.66; 95% CI, 0.03 to 1.28). I	BARRETT2004

5.5.5 Clinical summary

The only study to date suggests that cognitive behavioural therapy (including exposure and response prevention) involving the family is effective in reducing OCD symptoms in both individual and group formats. There is some evidence to suggest that these treatments also improve family function. There is also evidence to suggest that group therapy is somewhat more effective than individual therapy in reducing the young person's anxiety.

5.5.6 Descriptive review

Sixty-eight articles were identified that described or investigated the psychological treatment of OCD in one or more children or young people. Of the 68 articles, 53 were direct clinical investigations. This included the following papers:

Fifteen papers –were clinical review articles or chapters of the general psychological treatment of OCD in children and young people (AACP, 1998; Albano & DiBartolo, 1997; Franklin et al, 2003; Geffken et al, 1999; King et al, 1998; King & Scahill, 1999; March & Mulle, 1998; March, 1995; March et al, 2001; March & Mulle, 1996; Piacentini, 1999; Rapoport et al, 1993; Tolin & Franklin, 2002; Wolff & Wolff, 1991; Wolff & Rapoport, 1998).

Nine were open clinical trials involving 10 to 42 children. A protocol driven CBT manual based on the work of March and Mulle (1998) was used for several of the open clinical trials (Barrett et al, 2003), with one using a group format (Thienemann et al, 2001). The protocol incorporated psychoeducation, anxiety management training (AMT), stimulus hierarchies, graded exposure and response prevention (ERP), family participation and cognitive training such as thought stopping, constructive self-talk and cognitive restructuring. One study used a protocol developed by Piacentini and colleagues (2002), which involved exposure and response prevention, behavioural rewards, cognitive restructuring and parental involvement. Two other studies have concentrated upon graded exposure and response prevention with parent sessions (Franklin et al, 1998; Scahill et al, 1996). One open clinical trial used an adolescent group format (Fischer et al, 1998) based on a behavioural protocol developed by Krone and colleagues (1991). Finally, one open clinical trial compared CBT with medication (Wever & Rey, 1997). Methodologically, it is difficult to draw conclusions from these studies as to which components of the treatment package were the most effective ingredients of change. The results of the open trial studies could also be affected by bias as none used assessors that were blind to the treatment conditions.

Three studies were case series of consecutively referred young people, involving six to ten cases, and standardised protocols. The first study (N=10) investigated pharmacological and psychological treatment that included ERP, and cognitive methods for coping with anxiety and challenging appraisals (Williams & Allsopp, 1999). The second study (N=7) investigated individual CBT based on the protocol developed by March & Mulle (1998), and a parallel parent skills training module (Waters et al, 2001). The third study (N=6) investigated cognitive treatment, which included reappraising notions of responsibility (Williams et al, 2002).

Nine were experimental single case designs involving one or more individuals, defined as any report that provides a quantitative baseline assessment plus either assessment across multiple symptom domains (multiple baseline design) or treatments (such as ABAB design) (Detweiler & Albano, 2001; Francis, 1988; Freeston, 2001; Green, 1980; Harris & Wiebe, 1992; Kearney & Silverman, 1990; Knox et al, 1996; March & Mulle, 1998; Moritz, 1998).

Thirty-two papers were clinical case reports of one or more children. These are defined as having insubstantial descriptions of either assessment, treatment, and/or outcome with little or no accompanying quantitative data. None of the studies measured multiple symptom domains (multiple baseline design) or treatments (such as ABAB design). Many of these studies combine two or more treatments, making it difficult to assess which approach was more effective. Two papers described consecutive cases of young people

(eight and fifteen cases respectively) referred for treatment (Apter et al, 1984; Bolton et al, 1983), with one including a long-term follow-up (Bolton et al, 1996). The following described one or more cases (Bolton & Turner, 1984; Clark et al, 1982; Crago, 1995; Dalton, 1983; Desmarais & Lavallee, 1988; Fine, 1973; Franklin et al, 2001; Frare & Lebel, 1996; Friedmann & Silvers, 1977; Goodman, 1988; Hafner et al, 1981; Hallam, 1974; Hand, 1988; Harbin, 1979; Kellerman, 1981; Morelli, 1983; O'Connor, 1983; Ong & Leng, 1979; Owens & Piacentini, 1998; Ownby, 1983; Piacentini et al, 1994; Querioz et al, 1981; Stanley, 1980; Tolin, 2001; Warneke, 1985; Weiner, 1967; Willmuth, 1988; Yamagami, 1978; Zikis, 1983)

5.5.6.1 Evidence for psychological interventions

5.5.6.1.1 What interventions have the best outcome as measured by reduction of symptoms?

Behaviour therapy
Almost all studies have used behavioural therapy interventions, even if they are combined with other psychological therapies. The most commonly used behavioural intervention is graded exposure and response prevention. Studies have also investigated flooding, extinction, operant techniques, modelling, shaping and pacing.

Exposure and response prevention
Exposure and response prevention is the most researched therapeutic intervention in child and adolescent OCD studies, and appears to be the most promising. Out of the 53 studies analysed, more than 30 reported using either exposure or response prevention, or both, indicating that ERP appears to be the treatment of choice. All of the open trials, two of the consecutive case series (Waters et al, 2001; Williams & Allsopp, 1999), and all but two of the single case design studies (Francis, 1988; Franklin et al, 2001) used ERP as part of their intervention packages. The CBT studies that have incorporated ERP have shown a range of outcome from 87% of children rated as improved post-therapy (Bolton et al, 1983) to more modest results of 25% mean reduction in symptoms on the CY-BOCS (Thienemann et al, 2001). Two open clinical trials concentrated upon more of a behavioural invention, focusing on ERP with parental involvement. Franklin and colleagues (1998) reported a mean reduction on the CY-BOCS score of 67% at post-treatment, and 62% at follow-up (Franklin et al, 2003). Scahill and colleagues (1996) reported a mean post-treatment reduction of 61% on the CY-BOCS in a pilot study of behavioural therapy, ERP, and pharmacology, with a reduction of 51% at 3months' follow-up. There has been one open clinical trial of group behaviour therapy, consisting of therapist assisted ERP and behavioural homework, which reported clinically significant improvement post-treatment and at 6 months'

follow-up (Fischer et al, 1998). There have been no open trials that have only used individual ERP. Taken together, the data from these studies is encouraging and points to the efficacy of ERP.

Extinction

Extinction is used to describe the techniques involved when the child or adolescent's OCD behaviours are being maintained by the verbal responses of others in the environment. There are seven case studies that have included extinction in their intervention. It has been mostly used in cases of compulsive questioning and reassurance seeking, where parents have been instructed not to provide reassurance. There has been one single case ABAB design study that used an extinction procedure to treat reassurance seeking behaviour. The study indicated that during the extinction phase reassurance-seeking behaviour remitted, and returned on the non-extinction phase. The results indicated that the child had completely stopped asking for reassurance at one month's follow-up, once extinction was re-employed (Francis, 1988). Similar positive results have been reported in a case report of extinction for reassurance seeking in a 5year-old child (Tolin, 2001). Other case reports have used extinction together with other therapeutic interventions, making it difficult to assess the effectiveness of the extinction component. It has been used as part of family therapy interventions (Dalton, 1983; Fine, 1973; Morelli, 1983), with insight-orientated psychotherapy (Willmuth, 1988), and social skills approaches (Hallam, 1974). The studies have all reported positive treatment gains, but due to the lack of controlled studies and methodological weaknesses the use of extinction in child and adolescent OCD still remains unsubstantiated.

Other behavioural interventions

There are several case reports that have reported positive effects with other behavioural strategies. Three case reports have reported positive effects with modelling, shaping and pacing (Clark et al, 1982; March & Mulle, 1998; Ong & Leng, 1979; Warneke, 1985), mostly in the treatment of obsessional slowness in eating, grooming and washing. Only one case report has used flooding, and this was in conjunction with graded exposure and response prevention (Harris & Wiebe, 1992). A larger number of studies have acknowledged that operant techniques and behavioural rewards may play a positive role indirectly in creating change by helping the child attempt exposure, but again this is always used in combination with other treatment strategies and so is an adjunct to enhance other strategies rather than a strategy in its own right (Bolton et al, 1983; Bolton & Turner, 1984; Dalton, 1983; Fine, 1973; Green, 1980; Ong & Leng, 1979; Owens & Piacentini, 1998; Piacentini et al, 1994; Piacentini et al, 2002; Querioz et al, 1981; Warneke, 1985; Yamagami, 1978)

Cognitive behavioural therapy

Out of the 53 intervention studies, 20 report using a CBT intervention (Benazon et al, 2002; Bolton & Turner, 1984; Detweiler & Albano, 2001; Franklin et al, 2001; Freeston, 2001; Kearney & Silverman, 1990; Kellerman, 1981; March et al, 1994; March & Mulle, 1995; Ownby, 1983; Piacentini et al, 2002; Piacentini et al, 1994; Scahill et al, 1996; Thienemann et al, 2001; Tolin, 2001; Waters et al, 2001; Weiner, 1967; Wever & Rey, 1997; Williams & Allsopp, 1999). The open clinical trials that have used CBT protocols have incorporated sessions that include psychoeducation, anxiety management, cognitive training, behavioural rewards, exposure and response prevention, as well as parental involvement. The cognitive element frequently included training in consecutive self-talk and positive coping strategies (March et al, 1994). The CBT open clinical trials and case series studies have shown a range of outcomes with mean symptom reduction rates ranging from 25% (Thienemann et al, 2001) to 79% (Piacentini et al, 2002). The majority of studies have reported a mean symptom reduction on the CY-BOCS between 45 to 70% at post-treatment and at follow-up (Benazon et al, 2002; Franklin et al, 1998; March et al, 1994; Scahill et al, 1996; Waters et al, 2001; Wever & Rey, 1997). Furthermore results from a small number of single case design studies have also yielded positive results for CBT (Detweiler & Albano, 2001; Freeston, 2001; Kearney & Silverman, 1990; March & Mulle, 1995).

The CBT protocols frequently incorporate anxiety management training (AMT), which often includes progressive muscle relaxation, diaphragmatic breathing, and coping imagery. As this is often presented together with other behavioural strategies including ERP, it is difficult to judge whether AMT is effective or necessary in the treatment of child and adolescent OCD. There have been no studies that have just used AMT to treat OCD. One open trial has examined whether a simplified manualised CBT approach to ERP without AMT is effective (Franklin et al, 1998). The results showed a mean CY-BOCS reduction of 67% at post-treatment, and 62% at follow-up. From these results the authors argue that AMT is not a necessary component of CBT, and may only serve to make ERP work more accessible to younger people (the main active ingredient in treatment being ERP). Some clinicians have gone as far as arguing against the use of AMT as part of a first-line intervention for OCD (Tolin & Franklin, 2002), because intentionally eliciting anxiety through exposure work, whilst also learning strategies to minimise anxiety (through anxiety management), is likely to confuse the young person, and at a theoretical level, may interfere with habituation to anxiety, the putative mechanism for therapeutic change in ERP. They acknowledge that AMT may be a useful adjunct to ERP if children are so anxious at baseline that they are unable to tolerate ERP (Tolin & Franklin, 2002). There is no evidence that AMT alone is effective in the treatment of children and young people with OCD.

Cognitive therapy

There has been little research that has specifically investigated cognitive therapy in children and young people with OCD. As cognitive therapy strategies are often combined with other treatment approaches in CBT protocols, it is difficult to judge how effective the cognitive components are, compared with ERP, in effecting change. Cognitive restructuring is aimed at challenging the child's thought processes by questioning the reality of his or her obsessions and the necessity of the compulsive behaviours, and it has been used in two studies that reported positive results (Kearney & Silverman, 1990; March & Mulle, 1995). Finally, several uncontrolled case studies specify teaching children positive self-statements (e.g. 'I'm not afraid of germs, I can do this') to repeat during exposure (Willmuth, 1988; Zikis, 1983). Only one study has focused on utilizing a more cognitive approach to treat a consecutive case series of six young people with OCD. The treatment protocol focused upon normalizing intrusive thoughts, reappraising notions of responsibility, helping the young person re-evaluate the basis of their fears, and conducting behavioural experiments (Williams et al, 2002). The results showed good clinical outcomes for the cases, with cognitive changes in responsibility appraisals being associated with clinical improvement. One single case study has used an alternating design of response prevention and cognitive therapy, with the cognitive element including identifying obsessional thinking, examining more realistic probabilities, and conducting behavioural experiments (Kearney & Silverman, 1990). The results indicated that the total average improvement in symptoms for both procedures was similar, and that the combination of the two treatments was found to be effective in eliminating OCD. Research into cognitive therapy with children and young people is still in its infancy, therefore the specific efficacy of cognitive therapy for the treatment of OCD in children and young people has yet to be proven.

Strategies for obsessive thoughts

In some of the open clinical trials, treatment protocols include other thought stopping and satiation. These interventions have also been used in various case reports. Thought stopping is intended to interrupt the occurrence of obsessive thoughts, and positive treatment effects have been reported by the few studies that have specifically implemented thought stopping in the treatment of OCD (Frare & Lebel, 1996; Friedmann & Silvers, 1977; Kellerman, 1981; Ownby, 1983), although three of these studies are more than 20 years old. Finally, satiation is produced by repeating obsessional thoughts, or replaying an audiotape of obsessions. Five investigations of OCD have reported positive effects when satiation techniques have been incorporated (Friedmann & Silvers, 1977; Green, 1980; Kellerman, 1981; O'Connor, 1983; Taylor, 1985). These approaches would now be considered a variant of exposure that may or may not have been accompanied with response prevention.

Family therapy

There have been nine case reports that have incorporated family therapy into their treatment protocols with one or more children and young people. These have mostly described strategies aimed at altering the family system, and increasing communication and emotional expression within the young person's family. There have been two reports that have included procedures designed to alter family dynamics directly (Bolton et al, 1983; Dalton, 1983). However most of the family therapy studies also include either acknowledged or unacknowledged behavioural components, including exposure, extinction and operant reinforcement (Dalton, 1983; Fine, 1973; Hafner et al, 1981; Harbin, 1979; O'Connor, 1983). Two studies adopted systemic family therapy approaches where OCD was represented as a metaphor for family dysfunction (Dalton, 1983; O'Connor, 1983). These investigations encouraged ERP as a 'paradoxical intervention', making it impossible to determine how or whether the family intervention added to conventional, if implicit, cognitive behavioural approaches. Only two case reports focused more specifically on strategic therapy (Goodman, 1988) and marital and family therapy (Hand, 1988) specifically. One other case report describes positive gains for a child with OCD by using a cognitive intervention aimed at decreasing angry cognitions in the mother (Morelli, 1983). Although the results of these reports all outline improvements in symptoms, they have methodological flaws, therefore the specific efficacy of these approaches has yet to be proven.

Psychoanalysis, psychoanalytic/psychodynamic/supportive/individual psychotherapy

There are seven reports of different forms of individual psychotherapy for children and young people with OCD that appear in the recent literature (Apter et al, 1984; Bolton et al, 1983; Crago, 1995; Friedmann & Silvers, 1977; O'Connor, 1983; Warneke, 1985; Willmuth, 1988). All of the studies used a theoretically eclectic combination of treatment approaches. Three of these reported unspecified 'milieu therapy' as an additional feature (Apter et al, 1984; Bolton et al, 1983; Friedmann & Silvers, 1977). Several included individual, parent work, and group activities, frequently with behavioural interventions, or narrative approaches (Crago, 1995). The studies all report symptom reduction, but this is unsubstantiated as none uses standardised measures of outcome. To date the specific efficacy of these approaches for the treatment of OCD in children and young people has yet to be proven.

5.5.6.1.2 Are there developmental differences in the treatments most likely to achieve improvements in the identified outcomes for children (aged 8 –11 years) and young people (12 – 18 years)?

Most of the intervention studies have concentrated upon the adolescent age group (12 –18 years). There have only been 13 studies describing children with one or more children aged 11 years and under (Desmarais & Lavallee, 1988; Fine, 1973; Francis, 1988; Frare & Lebel, 1996; Goodman, 1988; Knox et al, 1996; March, 1995; Moritz, 1998; O'Connor, 1983; Querioz et al, 1981; Stanley, 1980; Tolin, 2001; Waters et al, 2001). These have highlighted the usefulness of CBT protocols, ERP, and extinction with younger children. Several open clinical trials have used a range of ages, from 7-17 years, but analyses have not been conducted to ascertain whether there is a difference in treatment outcome dependent upon age. One study involved five children under the age of 11 years, and seven young people over the age of 11 years, and reported that 11out of 12 did not meet criteria for OCD post-therapy (Barrett et al, 2003), indicating that age did not appear to affect outcome. One study used a single subject cross-over design with four children aged 6-11 years. The researcher used a manualised game program to developmentally present psychoeducation and behavioural interventions. The results showed that OCD symptom severity decreased during treatment (Moritz, 1998). Preliminary evidence therefore suggests that CBT protocols may be equally accessible to younger children, but further research is needed.

5.5.6.1.3 What should the duration and intensity of the specified treatment be?

Most of the treatment trials have used weekly sessions, with CBT treatment protocols ranging from 12 to 22 sessions. There has been only one study that has compared intensive CBT sessions (18 sessions over 1 month) with weekly CBT sessions (16 sessions over 4 months). Children with daily sessions did not show superior outcomes to those with weekly sessions (Franklin et al, 1998). However, methodological limitations, including lack of random assignment, limits the confidence that can be placed in this finding. It is likely that the more severely affected children received daily ERP, whereas less severely ill children were given weekly treatment. One participant was written up as a case report, and the authors reported markedly reduced OCD symptoms after two evaluation sessions and 11 daily sessions (Franklin et al, 2001), with the child falling in the sub-clinical range at post-therapy. A further study investigating a combined behavioural and pharmacological protocol offered on an intensive basis (10 daily sessions of CBT) showed a 68% remission rate and a 60% decrease in symptoms at 4 weeks (Wever & Rey, 1997). There are currently insufficient comparative studies to draw conclusions about the effectiveness of more intense treatment approaches.

5.5.6.1.4 What is the most effective format for treating children and young people with OCD?

Very few studies in the literature have taken a purely individual format of treatment. The large majority of studies combine individual therapy with some family sessions aimed at parent skills training. All of the open case trials and CBT protocols include parent sessions in order to provide psychoeducation, build problem-solving skills, teach strategies to reduce family involvement in the OCD, and encourage family support. All report positive effects by involving parents in therapy. Parents are often involved by assisting in the between-session exposure sessions and with ensuring treatment adherence. Only one study to date has attempted to empirically investigate the role of involving parents in CBT protocols. Knox and colleagues (1996; 1997) used a staggered baseline design to assess whether the addition of active parental participation to ERP would improve the effectiveness of the treatment. The results indicate that children reported less distress associated with their rituals (decreased SUDS ratings) when their parents were involved in therapy and were taught to ignore their compulsions (Knox et al, 1996). One single case design study (Francis, 1988), and one case report (Tolin, 2001), found that extinction, practiced by the parents, was effective in decreasing compulsive reassurance seeking.

One RCT investigated group formats of CBT and showed no difference between individual and group formats (Barrett et al, 2003). Two open clinical trials investigated group formats of CBT treatment with young people (Fischer et al, 1998; Thienemann et al, 2001). The first study was an open trial of behavioural group therapy with 15 young people, concentrating on ERP. Fischer and colleagues reported that all participants showed significant improvements on the CY-BOCS at post-treatment and at 6 months' follow-up. The second study investigated a CBT group that incorporated ERP with cognitive therapy with 18 children. Thienemann and colleagues reported a mean CY-BOCS reduction of 25%.

Preliminary results indicate that group formats of treatment may be an effective format of treatment, but both studies also incorporated parent sessions. From the other studies it is difficult to judge the treatment effects of group format of treatment, compared with the content of treatment, but involving parents in the child's therapy seems to be the treatment of choice.

5.5.7 Clinical summary

- Research evidence from one RCT, open clinical trials, case series, single case studies and case reports all point to the efficacy of CBT, which incorporates ERP, in the treatment of OCD in children and young people.

- In terms of format of treatment, there is evidence to suggest that involving parents in the treatment of their children, especially in CBT protocols incorporating ERP, is linked to good outcome.
- Limited research indicates that there is no difference between intensive and weekly sessions, but further research is needed to substantiate these findings.
- Although cognitive therapy may have some utility, to date the lack of outcome studies makes it difficult to draw definite conclusions about its effectiveness.
- Anxiety management training is often included in CBT protocols, but there is little evidence to point to its direct treatment effect for OCD for young people.
- For compulsive questioning and reassurance seeking, some studies suggest that extinction may be beneficial, but its use for other forms of OCD remains unsubstantiated.
- There is no evidence for the use of modelling, shaping and pacing in the treatment of OCD.
- Many studies include the use of operant rewards as an adjunct to therapy, alongside ERP interventions to increase the child's motivation in therapy.
- As little research exists investigating family therapy approaches with childhood OCD, its efficacy as yet remains unproven.
- There is no evidence to suggest that psychotherapy approaches (psychodynamic, insight-oriented) are effective in the treatment of OCD.
- In terms of developmental factors, lack of research makes it difficult to ascertain whether there are differences in treatment outcomes for children under the age of 11 years, compared with young people aged over 11 years of age. However, the incorporation of younger children in several open clinical trials indicates that CBT protocols appear to be equally accessible when adapted to younger children.

5.6 Psychological interventions for people with BDD

5.6.1 Introduction

BDD involves the belief or conviction that a physical feature is imperfect, preoccupation with this defect, and attempts to conceal, modify, or check the status of the feature. These cognitive and behavioural manifestations resemble, at least superficially, those found in other disorders as do the social anxiety and avoidance that are commonly associated with BDD. Consequently, as for other disorders with these types of features, a variety of psychological approaches have been attempted or developed with the aim of alleviating the distress and reducing the impairment experienced by people with BDD.

5.6.2 Current practice

There are no surveys on what psychological interventions are used for BDD in the UK or what proportion of patients with BDD receives a psychological treatment. Many individuals with BDD have difficulty accepting a psychological or pharmacological intervention and prefer to either avoid or camouflage their appearance. Alternatively they seek a cosmetic or dermatological procedure. Few mental health practitioners have clinical experience in treating many patients with BDD.

Current practice is not underpinned by a strong evidence base. There are few studies upon which to base clinical decisions and doubts about the generalisability of research findings to people encountered in practice who may refuse to participate in therapy.

5.6.3 Interventions included in the review

The following interventions were included:

- Cognitive behavioural therapy
- Behaviour therapy
- Cognitive therapy

5.6.4 Studies considered for the review

The review team conducted a new systematic search for studies that examined psychological interventions in BDD. Two RCTs were identified.

Rosen et al (1995) conducted an RCT of group cognitive behavioural therapy in 54 participants with BDD. Results indicated that 81.5% of the 27 patients were clinically improved after treatment. Treatment involved a small group format for an 8-week period. Therapy sessions consisted of education about causation and treatment of BDD, constructing a hierarchy of distressing aspects of their appearance, homework assignments involving exposure to anxiety provoking situations and preventing body checking behaviours, as well as keeping a body image diary. The participants in this study were different than those described in other centres, as they were less severely handicapped, they were all female and the most common preoccupation was their weight and shape. However they did not have a diagnosable eating disorder.

Veale et al (1996b) conducted an RCT of cognitive behavioural therapy in 19 participants who were predominantly female but more severely handicapped

than those in the Rosen and colleagues study. There was a 50% reduction in symptoms on the main outcome measure (Y-BOCS, modified for BDD). The emphasis in the therapy was helping the individual to have a good psychological understanding of the factors that maintained the symptoms, behavioural experiments to test out an alternative theory, exposure to situations avoided and dropping of excessive safety behaviours and rituals.

5.6.5 Descriptive review

Older RCTs and case series on body image therapy were excluded from the meta-analysis or narrative review as these were for body dissatisfaction and not body dysmorphic disorder or dysmorphophobia (Butters & Cash, 1987; Rosen et al, 1989).

Neziroglu & Yaryura-Tobias (1993b) report on the use of exposure and response prevention and cognitive therapy in five individuals with BDD and OCD. Participants were not on any medication and received either weekly or daily 90-minute sessions for 4 to 12 weeks. One individual dropped out and the other four showed significant improvement on observer rated measures. Results suggest that intensive sessions, more than once a week, seem to provide the greatest gains.

McKay and colleagues (1997) evaluated a maintenance follow-up program for individuals with BDD after cognitive behavioural therapy. Individuals were contacted bi-weekly for assessment with all measures for a total of 6 months. All subjects were assessed at the follow-up and all had remained symptom free. Patients in the maintenance group, however, had continued to improve on measures of anxiety and depression and showed significantly lower levels of anxiety and depression at follow-up. McKay (1999) followed up these patients 2 years later and noted treatment gains were maintained.

Wilhelm and colleagues (1999) evaluated cognitive behavioural group therapy in BDD. It led to significant improvement in both BDD and depressive symptoms. Participants received weekly 90-minute group CBT including psychoeducation, self-monitoring, cognitive restructuring, exposure and response prevention, and scheduling of pleasant events and achievement oriented activities.

Geremia and Neziroglu (2001) investigated the role of cognitive restructuring. Four individuals with BDD were treated in a single-subject multiple baseline design in which each patient served as his/her own control. Treatment consisted of 7 weeks of 75-minute sessions twice a week for cognitive treatment followed by 3 weeks of follow-up data gathering. Results indicated that cognitive therapy resulted in statistically significant reductions in BDD symptoms, depression and anxiety in three out of the four patients. Minimal

improvement was seen with overvalued ideation. In this study, no behavioural assignments were given but the authors suggest that this may enhance treatment efficacy.

There are also several older case reports or case series on the successful use of behaviour therapy by Munjack (1978), Solyom and colleagues (1985), Campisi (1995), Watts (1990), Marks and Mishan (1988), and Gomez and Marks (1994). Some of the cases in the latter two were also being treated with medication. There also case reports of cognitive behavioural therapy by Schmidt & Harrington (1995) and Neziroglu and colleagues (1996) and descriptions of the addition of reverse role-play to behaviour therapy by Newell & Shrubb (1994) and Cromarty & Marks (1995). Vitiello & DeLeon (1990) report one unsuccessful case after many years of psychoanalysis and then behaviour therapy with medication. Eye movement desensitization and reprocessing (EMDR), a form of CBT, resulted in improvement in six out of seven cases (Brown et al, 1997). There is one case report describing the use of psychodynamic psychotherapy (Bloch & Glue, 1988). A review and summary of the literature in cognitive behavioural treatments for BDD is provided by Neziroglu & Khemlani-Patel (2002).

Children and young people with BDD

There are no RCTs of any psychological interventions in children and young people with BDD. There is one successful case report of behaviour therapy (Braddock, 1982); one successful case report of behaviour therapy combined with doxepine (Sobanski & Schmidt, 2000); one of multiple treatment modalities (psychodynamic therapy, cognitive behavioural therapy, family therapy and medication) by Horowitz and colleagues (2002) and one case report of psychodynamic therapy (Philippopulis, 1979).

5.6.6 Clinical summary

There is some evidence from two RCTs and several case series on the benefit of cognitive behavioural therapy in adults with BDD. Little is known about the optimum frequency, type or duration of the therapy or the rate of relapse in the long term. One case report and expert opinion suggest that optimal therapy may need more intensive sessions (for example more than once a week especially in the early stages). The average duration of outpatient therapy may need to be slightly longer than other disorders (for example 20 to 25 sessions). Some evidence exists on the benefit of group cognitive behavioural therapy but this has not been compared with individual CBT or a combination of the two. There is virtually no evidence on psychological interventions in young people with BDD.

5.7 Clinical practice recommendations

5.7.1 Interventions for people with OCD or BDD

The treatments for OCD and BDD that are effective should be offered at all levels of the health care system. The difference in the treatments at the higher levels will reflect increasing experience and expertise in the implementation of a limited range of therapeutic options. For many people, initial treatment may be best provided in primary care settings. However, people with more impaired functioning, higher levels of co-morbidity, or poor response to initial treatment will require care from teams with greater levels of expertise and experience in the management of OCD or BDD.

Initial treatment options

Irrespective of level of care, the following recommendations should be taken into account when selecting initial treatments for people with OCD or BDD. The specific recommendations as to how to provide these treatments follow in the subsequent sections.

Regulatory authorities have identified possible risks associated with the use of SSRIs in depression especially among children, young people and young adults. The risks in other disorders including OCD/BDD are currently uncertain. Consequently, the recommendations about the use of SSRIs for people with OCD or BDD have taken account of the position of regulatory authorities.

Adults

In the current regulatory context, offer adults with milder impairments low intensity cognitive behaviour therapy (CBT) first, reserving higher intensity CBT and specific drug treatments for those with greater impairment. The intensity of psychological treatment has been defined as the hours of therapist input per patient. By this definition most group treatments meet the definition of a low intensity treatment (less than 10 hours therapist input per patient), although each patient may be receiving a much greater number of hours of therapy. Professionals offering psychological treatments should have received appropriate training for the intervention they are offering.

5.7.1.1 In the initial treatment of adults with OCD or BDD, low intensity psychological treatments including exposure and response prevention (ERP) (up to 10 therapist hours per patient) may be offered if the degree of functional impairment is mild and/or the patient expresses a preference for a low intensity approach. Low intensity treatments include:

- Computer guided ERP with brief scheduled contacts with a trained support worker

- Brief individual CBT (including ERP) using structured self-help materials
- Brief individual CBT (including ERP) by telephone
- Group CBT (including ERP). **[C]**

Children and young people
In the current regulatory context regarding prescribing SSRIs, offer children and young people with OCD or BDD psychological treatments first.

5.7.1.2 For children and young people with OCD with mild functional impairment, guided self-help may be considered in conjunction with support and information for the family. **[C]**

5.7.1.3 For children and young people with OCD with moderate to severe functional impairment, and for those with OCD with mild functional impairment for whom guided self help has been ineffective or refused, CBT (including ERP) involving the family and adapted to suit the developmental age of the child, should be offered as the treatment of choice. Group or individual formats should be offered depending upon the preference of the child or young person and their family/carers. **[B]**

5.7.1.4 All children and young people with BDD should be offered CBT (including ERP) involving the family and adapted to the developmental age of the child as first-line treatment**. [C]**

5.7.1.5 If the child or young person's OCD or BDD is not responding to treatment as a result of other co-existing factors such as the presence of co-morbid conditions, learning disorders, persisting psychosocial risk factors such as family discord, or the presence of parental mental ill-health, additional or alternative interventions for these aspects should be considered. The child or young person will still require evidence-based treatments for his or her OCD. **[C]**

How to use psychological interventions for adults

Cognitive behavioural treatments involving exposure and response prevention are effective treatments for OCD and BDD. The format and delivery of such therapy should take into account specific features of problems experienced by the person with OCD or BDD and the interventions should be adapted accordingly.

5.7.1.6 For people with obsessive thoughts who do not have overt compulsions, CBT including exposure to obsessive thoughts and

response prevention of mental rituals and neutralizing strategies, should be considered. **[B]**

5.7.1.7 For people with OCD living with family/carers, involving a family member or carer as a co-therapist in exposure and response prevention should be considered where this is appropriate and acceptable to the person with OCD and the family member or carer **[B]**

5.7.1.8 For people with more severe OCD who are house bound, unable or reluctant to attend a clinic, or have significant problems with hoarding a period of home based treatment may be offered. **[C]**

5.7.1.9 For people with more severe OCD who are housebound and unable to undertake home treatment because of the nature of their symptoms (e.g. contamination concerns or hoarding that prevents therapists' access to the person's home), a period of CBT by telephone may be considered. **[C]**

5.7.1.10 For people with OCD who refuse, or do not engage with, treatments that include ERP, individual cognitive therapy specifically adapted for OCD may be considered. **[C]**

5.7.1.11 When family members or carers of people with OCD or BDD have become involved in compulsive behaviours, avoidance or reassurance seeking, treatment plans will need to help them reduce their involvement in these behaviours in a sensitive and supportive manner. **[GPP]**

5.7.1.12 People with OCD or BDD with significant functional impairment may need access to appropriate support for travel and transport to allow them to attend for their treatment. **[GPP]**

5.7.1.13 For adults with OCD, cognitive therapy adapted for OCD may be considered as an addition to exposure and response prevention to enhance long-term symptom-reduction**. [C]**

5.7.1.14 For adults with BDD, group or individual CBT should be offered based on treatment protocols that address the specific features of BDD; the decision to use group or individual formats should be jointly decided by the individual with BDD and the healthcare professional. **[GPP]**

5.7.1.15 Towards the end of treatment, healthcare professionals should inform people with OCD or BDD about how the principles learned can be applied to the same or other symptoms if they occur in the future. **[GPP]**

5.7.1.16 Psychoanalysis, transactional analysis, hypnosis, marital/couple therapy and therapies other than cognitive and/or behavioural therapies should not routinely be offered as specific treatments for people with OCD**. [C]**

How to use psychological interventions for children and young people

Psychological treatments for children and young people should be collaborative and engage the family. Always consider the wider context and the other professionals involved with the child. Rewards to encourage the child can be helpful. When working with young people, the recommendations on the use of psychological interventions for adults may also be considered when appropriate.

5.7.1.17 In the cognitive-behavioural treatment of children and young people with OCD or BDD, particular attention should be given to:

- Developing and maintaining a good therapeutic alliance with the child/young person as well as their family
- Maintaining optimism in both child and family
- Collaboratively identifying initial and subsequent treatment targets with the child or young person
- Actively engaging the family in planning treatment and in the treatment process, especially in ERP where, if appropriate, they may be asked to assist the child or young person
- Encouraging the use of ERP if new or different symptoms re-emerge after successful treatment
- Liaising with other professionals involved in the child/young person's life, including teachers, social workers and other health professionals, especially when compulsive activity interferes with the ordinary functioning of the child/young person. **[GPP]**

5.7.1.18 In the psychological treatment of children and young people with OCD or BDD, addition and/or inclusion of behavioural or operant rewards in order to enhance the child's motivation and reinforce desired behaviour changes, should be considered. **[C]**

6 Pharmacological interventions for OCD

6.1 Introduction

The outlook for OCD was dramatically improved by the discovery of effective drug treatments in the early 1980s and 1990s, which revolutionised research into the neurobiology of the disorder (Marks et al, 1980; Montgomery, 1980). Intensive pharmacological investigation, in the form of large, multicentre randomised double-blinded placebo-controlled trials has consistently demonstrated that OCD responds selectively to drugs that act as potent inhibitors of the synaptic reuptake of serotonin (serotonin reuptake inhibitors, SRIs) (Montgomery et al, 2001; Zohar & Judge, 1996). Currently, this includes the tricyclic drug clomipramine, which stands apart from other tricyclics because of its more potent serotonergic actions, and the more highly selective serotonin reuptake inhibitors (SSRIs) citalopram, fluoxetine, fluvoxamine, paroxetine and sertraline (in alphabetical order). The finding that these drugs are effective even when depression is rigorously excluded in the reference population implies a specific antiobsessional effect. Drugs lacking these properties such as the standard tricyclic antidepressants amitriptyline, nortriptyline and desipramine and the monoamine oxidase inhibitors clorgyline and phenelzine have been found to be ineffective in randomised controlled trials (Ananth et al, 1981; Insel et al, 1983; Jenike et al, 1997; Volavka et al, 1985). Studies looking at benzodiazepines and lithium have also not produced positive findings (Hollander et al, 2003c; McDougle et al, 1991). Antipsychotics have not been found effective when given on their own, but may have a role as agents of augmentation in cases where the response to SRI is poor or incomplete.

The selectivity of the pharmacological response for serotonergic agents distinguishes OCD from depression and other anxiety disorders where a wider range of treatments appear effective, and implicates serotonin in the treatment effect.

6.2 Current practice

The Expert Consensus Panel for OCD (March et al, 1997) consisted of 85 worldwide experts and their guidelines present specific judgements on a comprehensive range of issues relating to pharmacological and psychological treatments. Although the guidelines did not distinguish between clomipramine and SSRIs, improved tolerability of the latter was acknowledged. Combined CBT and medication was preferred by experts in terms of speed, efficacy, durability, tolerability and acceptability, and was thought the best approach for most patients. More recently a smaller group of

members of the World Council on Anxiety met to agree recommendations for long-term treatment. They emphasised the importance of continuing treatment long-term from the outset and recommended 1-2 years continuation in treatment-responsive individuals (Greist et al, 2003).

Obsessive compulsive disorder responds to drug treatment in a characteristically slow, gradual way and improvements can take many weeks and months to develop. Patients often need to be encouraged to persevere in the early stages when progress can seem frustratingly slow. Dose titration is usually recommended, with patients remaining at the lowest effective dose levels for several weeks and reassessed before gradually increasing up to the maximum licensed doses according to observed efficacy and tolerability.

There remains a paucity of data to inform on long-term outcome, but the studies that have been performed suggest that the SRIs remain effective for as long as they are continued, and continuation protects against relapse. There is no convincing evidence supporting dose-reduction in the longer-term, and the adage 'the dose that gets you well keeps you well' probably applies.

6.3 Selective serotonin reuptake inhibitors (SSRIs)

6.3.1 Treatments included

The following treatments were included:

- Citalopram
- Fluoxetine
- Fluvoxamine
- Paroxetine
- Sertraline

All the above compounds with the exception of citalopram have Marketing Authorisation for the treatment of OCD in the UK.

6.3.2 Studies considered[6]

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of SSRIs among adults with OCD. Fifty-five studies were identified, of which 20 did not meet the inclusion criteria of the GDG. The 33 published trials plus one unpublished

[6] Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication, except where a study is *in press* or only submitted for publication, then a date is not used).

trial (BURNHAM1993) provided efficacy data from 4102 participants and tolerability data from 4907 participants.

Of the included studies, 18 compared SSRIs with placebo (BEASLEY1992, BURNHAM1993, CHOUINARD 1990, GOODMAN1989, GOODMAN1996, GREIST1995A, HOLLANDER2003B, HOLLANDER2003D, JENIKE1990A, JENIKE1990B, JENIKE1997, KAMIJIMA2004, KRONIG1999, MALLYA1992, MONTGOMERY1993, MONTGOMERY2001, PERSE1987, ZOHAR1996A), with two of these studies (BEASLEY1992, MONTGOMERY1993) featuring an extension phase involving participants classified as responders on completion of the acute phase of treatment.

Six studies examined the effects of different doses of SSRI (BEASLEY1992, BOGETTO2002, GREIST1995A, HOLLANDER2003D, MONTGOMERY1993, MONTGOMERY2001), with two of these studies (BEASLEY1992, MONTGOMERY1993) featuring an extension phase involving participants classified as responders on completion of the acute phase of treatment.

Two studies compared SSRIs with other SSRIs (BERGERON2002, MUNDO1997A), 10 with clomipramine (ASKIN1999, BISSERBE1997, BURNHAM1993, FREEMAN1994, KORAN1996A, LOPEZ-IBOR1996, MILANFRANCHI1997, MUNDO2001, SMERALDI1992, ZOHAR1996A) and four with other drugs including desipramine (GOODMAN1990A, HOEHN-SARIC2000), phenelzine (JENIKE1997) and venlafaxine (DENYS2003A).

All 32 acute phase studies (ASKIN1999, BEASLEY1992, BERGERON2002, BISSERBE1997, BOGETTO2002, BURNHAM1993, CHOUINARD1990, DENYS2003A, FREEMAN1994, GOODMAN1996, GOODMAN1989, GOODMAN1990A, GREIST1995A, HOEHNSARIC2000, HOLLANDER2003B, HOLLANDER2003D, JENIKE1990A, JENIKE1990B, JENIKE1997, KAMIJIMA2004, KORAN1996A, KRONIG1999, LOPEZIBOR1996, MALLYA1992, MILANFRANCHI1997, MONTGOMERY1993, MONTGOMERY2001, MUNDO1997A, MUNDO2001, PERSE1987, SMERALDI1992, ZOHAR1996A) were between 8 and 16 weeks long (mean length = 10.96 weeks). Where the treatment duration was greater than 16 weeks (BERGERON2002), efficacy data was extracted at the 12-week time-point. Participants were classified as outpatient in 18 studies, inpatient in one study and unclear in 13 studies. The average age of the patients in the acute phase studies was 36 years. The average duration of illness in 20 studies was 13.72 years. Although ten studies included patients with comorbid depression, the remaining two-thirds of the studies excluded individuals with comorbid depression, so the trials were testing the effect specifically for obsessive-compulsive symptoms. Ten studies were multi-centre trials, of which three were conducted in the US and six in Europe. Fourteen other

studies were conducted in the US, three in Italy, one each in Austria, Canada, Japan, the Netherlands and the UK.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.3.3 SSRIs versus placebo

6.3.3.1 Clinical evidence statements[7]

Efficacy[8]	*Included studies*
There is evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25%+ or 35%+ reduction on the clinician-rated Y-BOCS and/or CGI "much improved" or "very much improved" (K = 10; N = 2588; RR = 0.77; 95% C.I., 0.73 to 0.82). I	BEASLEY1992 BURNHAM1993 GREIST1995A HOLLANDER2003B HOLLANDER2003D KAMIJIMA2004 MALLYA1992 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A
There is limited evidence suggesting a difference favouring fluvoxamine over placebo on the likelihood of remission, defined as a score of 8 or less on the clinician-rated Y-BOCS (K = 1; N = 253; RR = 0.90; 95% C.I., 0.82 to 0.98). I	HOLLANDER2003B
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 12; N = 1629; SMD = -0.42; 95% C.I., -0.53 to -0.31). I	BEASLEY1992 BURNHAM1993 GOODMAN1989 GOODMAN1996 HOLLANDER2003B JENIKE1990A JENIKE1990B JENIKE1997 KAMIJIMA2004 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale (K = 3; N = 608; SMD = -0.28; 95% C.I., -0.44 to -0.11). I	MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A
There is limited evidence suggesting a difference favouring	ZOHAR1996A

[7] The full list of all evidence statements generated from meta-analyses (and the associated forest plots) will be available on the CD-ROM that accompanies the guideline.
[8] In the case of SMDs, negative effect sizes favour the treatment group.

paroxetine over placebo on reducing the severity of illness as measured by the CGI severity of illness subscale (K = 1; N = 293; SMD = -0.36; 95% C.I., -0.61 to -0.06). I	
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of family life as measured by the Sheehan Disability Scale family subscale (K = 1; N = 203; SMD = -0.33; 95% C.I., -0.61 to -0.06). I	MONTGOMERY2001
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of social life as measured by the Sheehan Disability Scale social subscale (K = 1; N = 203; SMD = -0.33; 95% C.I., -0.61 to -0.05). I	MONTGOMERY2001
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of work as measured by the Sheehan Disability Scale work subscale (K = 1; N = 203; SMD = -0.35; 95% C.I., -0.63 to -0.08). I	MONTGOMERY2001
There is limited evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25%+ or 35%+ reduction on the clinician-rated Y-BOCS and/or CGI "much improved" or "very much improved" in patients with comorbid depression (K = 3; N = 763; SMD = -0.42; 95% C.I., -0.53 to -0.31). I	BEASLEY1992 KAMIJIMA2004 MONTGOMERY1993
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS in patients with comorbid depression (K = 4; N = 489; SMD = -0.58; 95% C.I., -0.76 to -0.4). I	BEASLEY1992 GOODMAN1989 KAMIJIMA2004 MONTGOMERY1993

Tolerability

There is limited evidence suggesting that SSRIs when compared to placebo increase the risk of reporting adverse effects (K = 10, N = 1786, RR = 1.16; 95% C.I., 1.1 to 1.23). I	BURNHAM1993 GOODMAN1996 GREIST1995A JENIKE1990A JENIKE1990B KAMIJIMA2004 KRONIG1999 MALLYA1992 MONTGOMERY2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and placebo on the likelihood of leaving the study early (K = 16; N = 2623; RR = 0.98; 95% C.I., 0.85 to 1.13). I	BURNHAM1993 CHOUINARD1990 GOODMAN1989 GOODMAN1996 GREIST1995A HOLLANDER2003B HOLLANDER2003D JENIKE1990A JENIKE1990B JENIKE1997

	KRONIG1999 MALLYA1992 MONTOGOMERY1993 MONTGOMERY2001 PERSE1987 ZOHAR1996A
There is evidence suggesting that SSRIs when compared to placebo increase the risk of leaving the study early due to adverse effects (K = 13; N = 3009; RR = 2.15; 95% C.I., 1.62 to 2.86). I	BEASLEY1992 BURNHAM1993 CHOUINARD1990 GOODMAN1989 GOODMAN1996 GREIST1995A HOLLANDER2003B HOLLANDER2003D KAMIJIMA2004 KRONIG1999 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A

6.3.4 Different doses of SSRIs

6.3.4.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring 20 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over placebo on treatment response (K = 4; N = 666; RR = 0.82; 95% CI, 0.75 to 0.91). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 40 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over placebo on treatment response (K = 4; N = 661; RR = 0.79; 95% CI, 0.71 to 0.87). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting a difference favouring 60 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over placebo on treatment response (K = 4; N = 666; RR = 0.71; 95% CI, 0.64 to 0.8). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting there is unlikely to be a clinically important difference between 40 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) and 20 mg of a SSRI on treatment response (K = 4; N = 655; RR = 0.96; 95% CI, 0.85 to 1.08). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 60 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over 20 mg of a SSRI on treatment response (K = 4; N = 660; RR = 0.87; 95% CI, 0.76 to 0.98). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting there is unlikely to be a clinically important difference between 60 mg of a SSRI (citalopram/	BEASLEY1992 HOLLANDER2003D

fluoxetine/ paroxetine) and 40 mg of a SSRI on treatment response (K = 4; N = 655; RR = 0.90; 95% CI, 0.8 to 1.03). I	MONTGOMERY1993 MONTGOMERY2001

Tolerability

There is evidence suggesting that 20 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) when compared to placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 666; RR = 3.69; 95% CI, 2.03 to 6.7). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting that 40 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) when compared to placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 661; RR = 2.22; 95% CI, 1.17 to 4.22). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting that 60 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) when compared to placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 666; RR = 2.67; 95% CI, 1.44 to 4.98). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 40 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over 20 mg of a SSRI on leaving the study early because of adverse events (K = 4; N = 655; RR = 0.60; 95% CI, 0.39 to 0.92). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 60 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) over 20 mg of a SSRI on leaving the study early because of adverse events (K = 4; N = 660; RR = 0.72; 95% CI, 0.48 to 1.07). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60 mg of a SSRI (citalopram/ fluoxetine/ paroxetine) and 40 mg of a SSRI on leaving the study early because of adverse events (K = 4; N = 655; RR = 1.21; 95% CI, 0.75 to 1.93). I	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001

6.3.5 SSRIs versus other SSRIs

6.3.5.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring sertraline over fluoxetine on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS at 12 weeks (K = 1; N = 148; SMD = 0.39; 95% C.I., 0.07 to 0.72). I	BERGERON2002
There is limited evidence suggesting a difference favouring fluvoxamine over citalopram on reducing obsessive-compulsive symptoms as measured by the NIMH-OC (K = 1; N = 21; SMD = -1.22; 95% C.I., -2.17 to -0.27). I	MUNDO1997A

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between different SSRIs on the tolerability of treatment. I	BERGERON2002 MUNDO1997A

6.3.6 SSRIs versus clomipramine

6.3.6.1 Clinical evidence statements

Efficacy	*Included studies*
There is evidence suggesting there that is unlikely to be a clinically important difference between SSRIs and clomipramine on treatment response (OCD) (K = 8; N = 1019; RR = 1.02; 95% C.I., 0.89 to 1.17). I	BISSERBE1997 BURNHAM1993 LOPEZ-IBOR1996 KORAN1996A MILANFRANCHI1997 MUNDO2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (Y-BOCS) (K = 7; N = 739; SMD = 0.14; 95% CI, -0.01 to 0.29). I	LOPEZ-IBOR1996 KORAN1996A MILANFRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (NIMH-OC) (K = 3; N = 666; SMD = 0.08; 95% C.I., -0.08 to 0.23). I	BURNHAM1993 MUNDO2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (Y-BOCS) in patients with comorbid depression (K = 3; N = 192; SMD = 0.22; 95% CI, -0.06 to 0.51). I	LOPEZ-IBOR1996 MUNDO2001 SMERALDI1992
Tolerability	
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on adverse effects (K = 7; N = 1037; RR = 0.95; 95% C.I., 0.89 to 1). I	ASKIN1999 BISSERBE1997 BURNHAM FREEMAN1994 KORAN1996A MUNDO2001 ZOHAR1996A
There is limited evidence suggesting that clomipramine when compared to SSRIs increases the risk of leaving the study early (K = 10; N = 1139; RR = 0.72; 95% C.I., 0.59 to 0.88). I	ASKIN1999 BISSERBE1997 BURNHAM1993 FREEMAN1994 KORAN1996A LOPEZ-IBOR1996 MILANFRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A
There is limited evidence suggesting that clomipramine when	BISSERBE1997

compared to SSRIs increase the risk of leaving the study early due to adverse effects (K = 8; N = 1095; RR = 0.62; 95% C.I., 0.46 to 0.84). I	BURNHAM1993 FREEMAN1994 KORAN1996A LOPEZ-IBOR1996 MILANFRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A

6.3.7 SSRIs versus placebo or clomipramine: continuation treatment

6.3.7.1 Descriptive review

Five trials examined the continuation of treatment in patients with OCD (ANSSEAU1995, LOPEZ-IBOR1996, MONTGOMERY1993, TOLLEFSON1994A, GREIST1995A).

In MONTGOMERY1993, treatment responders (N = 173) continued their blinded treatment for an additional 16 weeks (after an 8-week, double-blind, placebo-controlled trial of three fixed doses of fluoxetine; Total N = 217).

Response was maintained during the extension phase. The mean improvement of Y-BOCS scores was fluoxetine 20mg (-1.3, N=18); fluoxetine 40mg (-1.6, N=20); fluoxetine 60mg (-1.7, N=23); placebo (-1.8, N=12). However, there was no significant difference between fluoxetine and placebo on mean improvement on the Y-BOCS.

ANNSEAU1995 was an unpublished 30-week extension phase trial of a 12-week acute-phase RCT (ZOHAR1996A) and it compared the maintenance of efficacy in patients who had responded to paroxetine, clomipramine or placebo. Patients (N=83) continued on the drug they received during the acute phase.

Ninety percent of patients in the paroxetine group had maintained a response to treatment (defined as a reduction of at least 25% in the total Y-BOCS scores), compared with 89.5% of patients in the clomipramine group and 75% of patients in the placebo group. However, there was no significant difference between the continuation of paroxetine and placebo (N = 63, SMD = -0.51, 95% C.I., -1.15 to 0.12) or between the continuation of paroxetine and clomipramine (N = 71, SMD = -0.01, 95% C.I., -0.53 to 0.5) on reducing obsessive-compulsive symptoms as measured by the Y-BOCS. There was also no significant difference between treatment groups on the likelihood of reporting adverse events, paroxetine versus placebo (82% v 66%, N = 63, RR = 1.24, 95% C.I., 0.81 to 1.88), paroxetine versus clomipramine (82% v 75%, N = 71, RR = 1.1, 95% C.I., 0.83 to 1.46).

In TOLLEFSON1994A, treatment responders (N = 76) continued their blinded treatment for an additional 6 months after a 13-week, double-blind, placebo-controlled trial of three fixed doses of fluoxetine (BEASLEY1992; N = 355).

Sixty-seven percent of placebo-treated patients improved on the Y-BOCS at the end of the responder extension phase, as did 69.6% fluoxetine 20 mg-treated patients, 76.2% fluoxetine 40 mg-treated patients, and 76.9% fluoxetine 60 mg-treated patients. Combining the fluoxetine-treated groups, 74.3% patients experienced improvement beyond that seen during the 13-week acute phase. In terms of the number of patients who achieved ≥25% improvement in their Y-BOCS score, there was no difference between any dose of fluoxetine and placebo and between different doses of fluoxetine, 40mg v 20mg (RR = 0.94, 95% C.I., 0.57 to 1.54), 60mg v 20mg (RR = 0.7, 95% C.I., 0.4 to 1.21) and 60mg v 40mg (0.74, 95% C.I., 0.41 to 1.32).

In GREIST1995A, responders to a 12-week randomised trial of one of three fixed doses of sertraline (50, 100 or 200mg) or placebo were continued on their treatment. Responders to the acute phase, defined as "marked" or "moderate" improvement on the Clinical Global Impressions Efficacy index were offered an additional 40 weeks of double-blind treatment at their assigned doses. Three hundred and twenty-five patients entered the acute phase, of which 125 patients were classified as responders. One hundred and eighteen patients entered the continuation phase.

Overall, the pooled sertraline group exhibited greater improvement on measures of efficacy over the 48-week treatment period, Y-BOCS mean change scores (S.D.s): sertraline -5.7 (7.4) versus placebo –2.8 (5.8), F (1,289) = 7.06, p = 0.001. When changes from week 12 to endpoint were examined on efficacy, no differences were seen among any of the individual treatment groups or between the pooled sertraline group and placebo. Patients in the sertraline groups were more likely to report adverse events compared to patients in the placebo group, sertraline 50mg 94%, sertraline 100mg 91%, sertraline 200mg 96%, placebo 79%. Two patients in the sertraline group and none in the placebo group attempted suicide.

LOPEZ-IBOR1996 continued patients for 12 weeks on the same double-blind fluoxetine versus clomipramine treatment received during an 8-week acute phase. Responders (fluoxetine, n = 11; clomipramine, n = 13) received a lower dose of the drug (20mg fluoxetine and 100mg clomipramine), while non-responders (fluoxetine, n = 14; clomipramine, n = 8) received a higher dose of the same acute-phase drug (60mg fluoxetine and 200mg clomipramine).

No formal statistical analysis took place due to the small size of the treatment arms. However, among responders to fluoxetine the mean endpoint Y-BOCS score was lower (8.8 ± 5.79) compared to baseline (9.6 ± 5.35), whereas among

responders to clomipramine the mean endpoint score was higher (13.7 ± 11.79) compared to baseline (11.4 ± 6.13). Among non-responders, there was a decrease in the mean Y-BOCS score from baseline in patients receiving fluoxetine (mean baseline score 26.7 ± 6.38, mean endpoint score 24 ± 7.54), and similarly in patients receiving clomipramine (mean baseline score 21.5 ± 8.62, mean endpoint score 15 ± 9.32).

6.3.8 SSRIs versus placebo or clomipramine: relapse prevention

6.3.8.1 Descriptive review

Six trials were included in the review that examined relapse prevention in patients with OCD (ANSSEAU1995, BAILER1995, HOLLANDER2003, KORAN2002, RAVIZZA1996A, ROMANO2001). RAVIZZA1996A was an open-label trial and therefore excluded from the review.

In BAILER1995, following a 6-month acute phase RCT of paroxetine versus placebo (BURNHAM1993) and a 6-month open label phase with flexible dosing of paroxetine (N=154), non-responders (N=44) were randomised to a 6-month double-blind relapse prevention phase of fixed dose paroxetine versus placebo.

For the percentage of patients with partial relapses (defined as a Y-BOCS score increase from the acute phase baseline score and an increase of one or more points on the CGI Severity of Illness subscale from the last open label score), the treatment effect was not significant (p = 0.22), although the percentage of partial relapses in the placebo group (63.6%) was greater than the percentage of partial relapses in the paroxetine group (42.1%). There was a significant difference favouring paroxetine over placebo on the Y-BOCS (N=20, SMD = -1.17; 95% C.I., -2.15 to –0.19).

In HOLLANDER2003, treatment responders (N = 105) were randomised to 6-month double-blind, fixed dose, parallel paroxetine/placebo treatment (after completing both a 12-week RCT of paroxetine/placebo [Total N = 348] and 6 months of open-label paroxetine treatment [Total N = 263].

The results indicated that more of the patients randomised to receive placebo (N=52) experienced a relapse than those who continued to receive paroxetine (N=53) when the criteria included an increase of one point or more on the CGI severity scale (38% v 60%; RR = 0.63, 95% CI, 0.42 to 0.96). With a stricter criterion of a return to baseline of patients' Y-BOCS scores, the effect was stronger, but the difference was not statistically significant (9% v 23%; RR = 0.41, 95% CI, 0.15 to 1.08). The mean time it took for patients continuing with paroxetine to relapse was 62.9 days compared with 28.5 days for those who relapsed after switching to placebo. Statistically significant differences

favouring paroxetine over placebo on mean Y-BOCS scores were found as early as week 2 (F = 5.25, df = 1,68, p = 0.02) and up until 5 months. However, no difference was found between the groups at the end of 6 months. Significantly more patients receiving placebo left the study early because of adverse events compared with those receiving paroxetine (38% v 5%, N=105, RR = 0.15; 95% CI, 0.05 to 0.47).

In ANNSEAU1995, following an acute phase RCT of paroxetine versus clomipramine versus placebo (ZOHAR1996A) and an 8-week maintenance phase, patients were re-randomised within group to the drug or placebo (paroxetine/paroxetine versus paroxetine/placebo, clomipramine/clomipramine versus clomipramine/placebo, and placebo).

There was no significant difference between paroxetine and placebo on the rate of partial relapse (defined as an increase on the Y-BOCS from the baseline score or an increase in CGI severity by one or more points from the last observation), paroxetine 1/10 (10%) vs. placebo 2/8 (25%). Based on a 25% response criterion on the Y-BOCS, the rate of response was lower in paroxetine (18.2%) compared to placebo (33.3%), but this was not statistically significant (p = 0.45). There was also no significant difference between paroxetine and placebo on the likelihood of reporting adverse effects, paroxetine 57% vs. sertraline 50%.

In ROMANO2001, treatment responders (N = 71) were randomised to 52 weeks double-blind, fixed dose fluoxetine or placebo treatment (after completing a 20-week single-blind treatment of fluoxetine [Total N = 130]). People who continued to receive fluoxetine had a lower estimated 1-year relapse rate compared to those randomised to placebo, though this difference was not statistically different (20.6% v 31.9%, p = 0.14). Among patients who responded to acute phase treatment with fluoxetine 60mg/day, a subset of those who continued to receive fluoxetine, the 1-year estimated rate of relapse was lower compared to patients randomised to placebo (17.5% v 38%, p = 0.04). This estimate was not significantly different for people who responded to acute phase treatment with fluoxetine 20 or 40mg/day and who continued to receive fluoxetine for a year compared to patients randomised to placebo (28.6% v 21.3%, p = 0.79). Change on the Y-BOCS from randomisation to endpoint was not statistically different between fluoxetine and placebo (p = 0.54).

In KORAN2002, treatment responders (N = 223) were randomised to 28 weeks double-blind sertraline or placebo treatment (after completing a 52-week single-blind treatment of sertraline [Total N initially enrolled = 649]). Patients assigned to sertraline continued on the same dose of sertraline as during the 52-week phase, while patients assigned to placebo were blindly discontinued from sertraline. Mean Y-BOCS scores increased in both groups

(mean change at end-point, –1.3 and –3.1 in sertraline and placebo groups respectively). However, patients continuing with sertraline had a lower mean score at endpoint than those receiving placebo (N = 221; SMD = -0.37; 95% CI, -0.63 to –0.10). When a strict criteria for relapse (increase in Y-BOCS >=5, Y-BOCS score>=20 and CGI-I >=1 continuing for a month) was applied, there was no difference between sertraline and placebo (3.6% v 5.2%, N = 223, RR = 0.7, 95% C.I., 0.2 to 2.4). However, more patients receiving placebo experienced a relapse or insufficient clinical response compared with sertraline (RR = 0.39; 95% CI, 0.20 to 0.76) and more patients experienced an acute exacerbation of OCD (RR = 0.34; 95% CI, 0.19 to 0.60).

6.3.9 Clinical summary

There is evidence from ten studies involving 2,588 patients for the efficacy of SSRIs (citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) for adults with OCD from placebo controlled trials. The tolerability data indicate that they are generally well tolerated as the likelihood of leaving the study is unlikely to be greater for active treatment than for placebo. However there is limited evidence for more adverse events reported in the active arms and a greater chance of leaving the study because of adverse effects indicating that some people will experience adverse effects. In addition, there is some evidence that higher doses may be more efficacious than lower doses for citalopram, fluoxetine and paroxetine and may be associated with relatively fewer premature withdrawals associated with adverse effects. This may be due to the greater response at higher doses encouraging people to stay in the study whether they experience adverse effects or not. There is yet very little evidence suggesting that any one SSRI is more effective than another.

There is evidence from eight studies involving 1,019 patients that there is unlikely to be any clinically important differences between SSRIs and clomipramine either for efficacy or for adverse effects, but there is a greater likelihood of people discontinuing clomipramine based on those leaving the studies early and leaving early due to adverse effects. Thus, although SSRIs and clomipramine are both efficacious treatments (see also 4.6 below, SSRIs may be better tolerated. The continuation studies have investigated long-term treatment with SRIs for up to 48 weeks. The results suggest that patients who have responded to acute-phase treatment continue to respond in the longer term. The relapse prevention studies have used survival analysis to investigate patients for up to 12 months. The results suggest that SSRI treatment continued over the longer term protects against relapse of OCD.

6.4 Clomipramine

6.4.1 Introduction

Clomipramine was the first pharmacotherapeutic agent found to have efficacy in OCD (Marks et al, 1980; Montgomery, 1980). The drug shares the pharmacological properties of the tricyclic group of antidepressants from which it is derived, but can be distinguished from other tricyclics by its potent effects at inhibiting the synaptic reuptake of the neurotransmitter serotonin. However, its effects are not selectively mediated by serotonin mechanisms. As a tricyclic, clomipramine is associated with the adverse effects and toxicity in overdose that typifies this group of drugs. For this reason it is usually considered second-line after SSRIs for patients who have failed to respond to SSRIs or who are unable to tolerate them.

The following treatments were included:

- oral clomipramine
- intravenous clomipramine.

6.4.2 Studies considered

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of clomipramine. Sixty-four studies were identified, of which 36 did not meet the inclusion criteria of the GDG. The 27 included studies provided efficacy data from 2121 participants and tolerability data from 2208 participants.

Of the included studies, seven compared clomipramine with placebo (BURNHAM1993, CCSG1991, KATZ1990, MONTGOMERY1990, STEIN1992, THOREN1980A, ZOHAR1996A), five with other tricyclic anti-depressants (ANANTH1981, KHANNA1988, THOREN1980A, VOLAVKA1985, ZOHAR1987A), ten with SSRIs[9] (ASKIN1999, BISSERBE1997, BURNHAM1993, FREEMAN1994, KORAN1996A, LOPEZ-IBOR1996, MILANFRANCHI1997, MUNDO2001, SMERALDI1992, ZOHAR1996A), and five with other drugs (ALBERT2002, HEWLETT1992, INSEL1983B, PATO1991, VALLEJO1992). Other comparisons included intravenous clomipramine versus placebo (FALLON1998) and oral clomipramine (KORAN1997).

All included acute phase studies (N=25) were between 2 and 16 weeks long (mean length = 9.13 weeks). Patients were classified as outpatient in 11 studies, inpatient in 3 studies and mixed in 3 studies. Eight studies did not

[9] The results of the analysis of these studies are considered in the earlier section on SSRIs vs. Clomipramine

report patient setting. The average age of the patients was 33.65 years. The average duration of illness based on 10 studies was 10.94 years. Six studies were multi-centre studies, of which two were conducted in the US, two in Europe, one in France and Spain and the other in France and Belgium. Nine other studies were conducted in the US, three in Italy, three in the UK and one each in Austria, Canada, India and Sweden.

Two cross-over trials comparing clomipramine with SSRIs (KHANNA1988, ZOHAR1987A) are summarised in narrative form, as it was not possible to extract data at the point of cross-over. The other two cross-over trials, INSEL1983B and HEWLETT1992, are considered narratively in the sections on MAOIs and Anxiolytics respectively.
Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.4.3 Clomipramine versus placebo

6.4.3.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring clomipramine over placebo on the likelihood of treatment response, defined as a 25% or greater reduction on the clinician-rated Y-BOCS, or as "much improved/very much improved" on the Clinical Global Impressions scale (K = 3; N = 401; RR = 0.81; 95% CI, 0.68 to 0.96). I	BURNHAM1993 STEIN1992 ZOHAR1996A
There is evidence suggesting a difference favouring clomipramine over placebo on the likelihood of remission, defined as a score of 1 to 6 on the NIMH-OC scale (K = 1; N = 520; RR = 0.54; 95% CI, 0.48 to 0.61). I	CCSG1991
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 3; N = 694; random effects SMD = -1.04; 95% CI, -1.72 to -0.36). I	CCSG1991 (1) CCSG1991 (2) ZOHAR1996A
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the CPRS or 6-item OCD scale (K = 2; N = 30; SMD = -1.12; 95% CI, -1.92 to -0.32). I	MONTGOMERY1990 THOREN1980A
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC scale (K = 4; N = 847; random effects SMD = -0.87; 95% CI, -1.37 to -0.38). I	BURNHAM1993 CCSG1991 (1) CCSG1991 (2) ZOHAR1996A
There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing the severity of illness as measured by the Clinical Global Impressions severity of illness subscale (K = 1; N = 193; SMD = -0.32; 95% CI, -0.60 to –0.03). I	ZOHAR1996A

There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing psychological distress as measured by the Symptom Checklist-90 (K = 1; N = 151; SMD = -0.32; 95% CI, -0.64 to 0.00). I	ZOHAR1996A

Tolerability

There is evidence suggesting that clomipramine when compared to placebo increases the risk of reporting adverse effects (K = 3; N = 877; random effects RR = 1.25; 95% CI, 0.96 to 1.62). I	BURNHAM1993 CCSG1991 ZOHAR1996A
There is evidence suggesting that clomipramine when compared to placebo increases the risk of leaving the study early due to adverse effects (K = 2; N = 357; RR = 2.35; 95% CI, 1.31 to 4.22). I	BURNHAM1993 ZOHAR1996A

6.4.4 Clomipramine versus other TCAs

6.4.4.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessional Neurotic Scale (K = 1; N = 16; SMD = -1.17; 95% CI, -2.26 to –0.09). I	VOLAVKA1985
There is limited evidence suggesting a difference favouring clomipramine over imipramine on improving global efficacy as measured by the Global Evaluation of Efficacy (K = 1; N = 16; SMD = -1.05; 95% CI, -2.12 to 0.02). I	VOLAVKA1985
There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing depression as measured by the Hamilton Rating Scale for Depression (K = 1; N = 16; SMD = -1.04; 95% CI, -2.11 to 0.02). I	VOLAVKA1985

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and imipramine on leaving the study early because of adverse effects Depression (K = 1; N = 23; RR = 1.09; 95% CI, 0.18 to 6.48). I.	VOLAVKA1985

6.4.4.2 Results summary from additional cross-over trials

In KHANNA1988, patients received double-blind clomipramine or nortriptyline in a randomised fashion for 6 weeks and then crossed over to the other drug after a 4-week drug-free interval. Out of the 18 patients who entered the trial, eight completed the full cross-over sequence.

There was no significant difference between pre- and post-trial scores on the Leyton Obsessional Inventory and the Maudsley Obsessive-Compulsive Inventory. There was also no difference between clomipramine and nortriptyline on obsessive-compulsive symptoms.

In ZOHAR1987A, after a 2-4 week placebo-phase, patients were randomly assigned to clomipramine or desipramine. After a 4-week placebo interval, patients were crossed-over to the other drug. The duration of each active treatment was 6 weeks. Fourteen patients entered the trial, of which 10 patients completed the entire cross-over sequence.

The reduction in obsessive-compulsive symptoms as measured by the Comprehensive Psychiatric Rating Scale – Obsessive-Compulsive subscale (CPRS-OC) and the NIMH Global Obsessive-Compulsive scale was greater in Clomipramine than Desipramine (CPRS-OC-5: F1,8 = 8.03, p = 0.02; NIMH Global-OC: F1,8 = 7, p = 0.03). Using the Comprehensive Psychiatric Rating Scale – Obsessive-Compulsive subscale, the mean (± SD) improvement during the 6 weeks for clomipramine was 28.4% ± 20.1% compared to 4.2% ± 11.4% for desipramine.

6.4.5 Intravenous clomipramine + placebo pills vs. oral clomipramine + placebo IV

6.4.5.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on the likelihood of treatment response, defined as a 25% or greater reduction on the clinician-rated Y-BOCS from baseline (K = 1; N = 15; RR = 0.16; 95% CI, 0.03 to 1.02). I	KORAN1997
There is limited evidence suggesting a difference favouring intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 1; N = 15; SMD = -1.26; 95% CI, -2.40 to -0.12). I	KORAN1997
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on the tolerability of treatment.	KORAN1997

6.4.6 Clomipramine versus placebo or SSRIs: continuation or discontinuation of treatment

There were 3 trials that examined the continuation or discontinuation of clomipramine. They were KATZ1990, LOPEZ-IBOR1996, RAVIZZA1996A. RAVIZZA1996A was an open-label trial and therefore excluded from the review.

In KATZ1990, outpatients received an initial 10-week double-blind treatment of clomipramine or placebo. Patients who responded to the 10-week acute phase, response being defined as response to treatment on at least three occasions during the 10 weeks as judged by the treating physician, entered a double-blind 1-year extension. Two hundred and eighty-six patients entered the study, of which 124 patients with a baseline Hamilton Depression Scale score ≤ 17 and responding to the acute phase treatment entered the 1-year extension.

Patients from both groups showed improvement over the course of the extension phase compared to baseline as measured by the NIMH Global Obsessive-Compulsive scale, clomipramine –4.7 (S.D. 2.7) and placebo –1.7 (S.D. 2.5). The improvement was greater in clomipramine compared to placebo ($p < 0.001$). Ninety-eight percent of the clomipramine patients and 65% of the placebo patients reported one or more adverse events. Twenty-five patients taking clomipramine discontinued the extension trial because of medical problems, whereas no placebo patients discontinued the study because of medical problems. Twelve clomipramine patients had serious adverse events. However, the authors suggest that the small sample size, small entry cohort and discontinuation patterns argue against any general or long-term efficacy of placebo, and also against any conclusive interpretations of the data for this highly selected group.

LOPEZ-IBOR1996 continued patients for 12 weeks on the same double-blind fluoxetine versus clomipramine treatment received during an 8-week acute phase. Responders (fluoxetine, n = 11; clomipramine, n = 13) received a lower dose of the drug (20mg fluoxetine and 100mg clomipramine), while non-responders (fluoxetine, n = 14; clomipramine, n = 8) received a higher dose of the same acute-phase drug (60mg fluoxetine and 200mg clomipramine).

No formal statistical analysis took place due to the small size of the treatment arms. However, among responders to fluoxetine the mean endpoint Y-BOCS score was lower (8.8 ± 5.79) compared to baseline (9.6 ± 5.35), whereas among responders to clomipramine the mean endpoint score was higher (13.7 ± 11.79) compared to baseline (11.4 ± 6.13). Among non-responders, there was a decrease on the mean Y-BOCS score from baseline in patients receiving fluoxetine (mean baseline score 26.7 ± 6.38, mean endpoint score 24 ± 7.54),

and similarly in patients receiving clomipramine (mean baseline score 21.5 ± 8.62, mean endpoint score 15 ± 9.32).

6.4.7 Clinical summary

The results of these studies show that clomipramine is effective in the acute treatment of OCD. There have been no 'dose-finding' studies for clomipramine so we cannot judge which is the most effective dose for OCD. There is evidence from one study that intravenous clomipramine may be more effective than oral in partially responsive individuals . The evidence supporting the ongoing efficacy in continuation studies is limited by the shortage of studies in this area.

6.5 Other tricyclic antidepressants

6.5.1 Introduction

Unlike SSRIs and clomipramine, other antidepressants that have been investigated in OCD so far, for example, tricyclic antidepressants (apart from clomipramine), tricyclic related antidepressants, serotonin and noradrenaline re-uptake inhibitors (SNRIs) and monoamine oxidase inhibitors (MAOIs) have not been shown to have convincing antiobsessional efficacy (see below).

6.5.2 Treatments included

The following treatments were included:

- Amitriptyline
- Desipramine
- Imipramine
- Nortriptyline.

6.5.3 Studies considered

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of TCAs other than clomipramine. Twelve studies were identified, of which five did not meet the inclusion criteria of the GDG. The seven included studies provided efficacy data from 258 participants and tolerability data from 259 participants.

Of the included studies, five compared TCAs with clomipramine (ANANTH1981, KHANNA1988, THOREN1980A, VOLAVKA1985, ZOHAR1987A) and two with SSRIs (GOODMAN1990A, HOEHN-SARIC2000). One study featured an additional placebo comparison (THOREN1980A).

All included studies were between 5 and 28 weeks long (mean length = 12.4 weeks). Patients were classified as outpatient in three studies, inpatient in one study and mixed in one study. One study did not report patient setting. The mean age of the patients was 36.6 years and the mean duration of illness in two studies was 18 years. Four studies were conducted in the US and one each in Canada, India and Sweden.

The results of two cross-over studies (KHANNA1988, ZOHAR1987A) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.5.4 Tricyclic antidepressants versus placebo

6.5.4.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on the efficacy of treatment in adults with OCD (K = 1, N = 16).	THOREN1980A

6.5.4.2 Results summary from additional cross-over trials

In KHANNA1988, patients received double-blind clomipramine or nortriptyline in a randomised fashion for 6 weeks and then crossed-over to the other drug after a 4-week drug-free interval. Out of the 18 patients who entered the trial, 8 completed the full cross-over sequence.

There was no significant difference between pre- and post-trial scores on the Leyton Obsessional Inventory and the Maudsley Obsessive-Compulsive Inventory. There was also no difference between clomipramine and nortriptyline on obsessive-compulsive symptoms.

In ZOHAR1987A, after a 2-4 week placebo-phase, patients were randomly assigned to clomipramine or desipramine. After a 4-week placebo interval, patients were crossed-over to the other drug. The duration of each active treatment was 6 weeks. Fourteen patients entered the trial, of which 10 patients completed the entire cross-over sequence.

The reduction in obsessive-compulsive symptoms as measured by the Comprehensive Psychiatric Rating Scale – Obsessive-Compulsive subscale

(CPRS-OC) and the NIMH Global Obsessive-Compulsive scale was greater in Clomipramine than Desipramine (CPRS-OC-5: F1,8 = 8.03, p = 0.02; NIMH Global-OC: F1,8 = 7, p = 0.03). Using the Comprehensive Psychiatric Rating Scale – Obsessive-Compulsive subscale, the mean (± SD) improvement during the 6 weeks for clomipramine was 28.4% ± 20.1% compared to 4.2% ± 11.4% for desipramine.

6.5.5 Clinical summary

The results of these studies do not support the efficacy of tricyclic antidepressants (apart from clomipramine) for OCD.

6.6 Tricyclic related antidepressants

6.6.1 Treatments included

The following treatments were included:

- Trazodone

6.6.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of tricyclic related antidepressants. Two studies were identified, one of which did not meet the inclusion criteria of the GDG (ANTONELLI1973). The included study (PIGOTT1992) compared trazodone with placebo in a 10-week outpatient trial that provided efficacy data from 17 participants. Participants were begun on a fixed oral dosage regimen of 50mg/day and were gradually increased, as tolerated, to a maximum of 300mg/day.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.6.3 Descriptive review

In PIGOTT1992, patients received 10 weeks of either trazodone or placebo in a randomised fashion. There were no treatment differences between trazodone and placebo in patients completing the 10-week trial. Mean maximum decreases from baseline on the Y-BOCS was 2.7 ± 1.87, t = -1.44, p = 0.18 in patients receiving trazodone and –2.83 ± 1.19, t = -2.37, p = 0.06 in patients receiving placebo.

6.6.4 Clinical summary

There is no evidence supporting the efficacy of tricyclic related drugs in OCD.

6.7 Serotonin and noradrenaline reuptake inhibitors (SNRIs)

6.7.1 Treatments included

The following treatments were included:

- Venlafaxine.

6.7.2 Studies considered

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of SNRIs. Five studies were identified, of which three did not meet the inclusion criteria of the GDG (DENYS2002, TENNEY2003, YARYURATOBIAS1996). The two included studies provided efficacy data from 218 participants and tolerability data from 223 participants, and compared venlafaxine with clomipramine (ALBERT2002) and paroxetine (DENYS2003A).

Both studies were 12 weeks long. Patients were classified as outpatient in both studies. The duration of OCD was 5.15 and 15 years in ALBERT2002 and DENYS2003A respectively. ALBERT2002 was conducted in Italy and DENYS2003A was conducted in the Netherlands.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.7.3 Venlafaxine versus other antidepressants

6.7.3.1 Clinical evidence statements

Efficacy	*Included studies*
There is evidence suggesting there is unlikely to be a clinically important difference between venlafaxine and other SRIs on treatment response (OCD) (K = 2; N = 223; RR = 1.13; 95% CI, 0.91 to 1.40). I	ALBERT2002 DENYS2003A
There is evidence suggesting there is unlikely to be a clinically important difference between venlafaxine and other SRIs on OCD symptoms (K = 2; N = 218; SMD = 0.11; 95% CI, -0.16 to 0.38). I	ALBERT2002 DENYS2003A
Tolerability	
There is limited evidence suggesting a difference favouring venlafaxine over clomipramine on the likelihood of reporting adverse effects in adults with OCD (K = 1; N = 73; RR = 0.67, 95% CI, 0.49 to 0.92). I	ALBERT2002

6.7.4 Clinical summary

There is no placebo-controlled evidence to support the efficacy of SNRIs in OCD. Although two studies showed similar levels of improvement on venlafaxine that have been found with drugs known to have shown efficacy

in previous OCD, the absence of a placebo for comparison in these studies prevents us from concluding that in these studies the drug was actually effective. However, the results are suggestive of efficacy and point to further systematic investigation of SNRIs as a potential treatment for OCD.

6.8 Monoamine oxidase inhibitors (MAOIs)

6.8.1 Treatments included

The following treatments were included:

- Clorgyline
- Phenelzine.

6.8.2 Studies considered

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of MAOIs. Five studies were identified, of which two did not meet the inclusion criteria of the GDG (INSEL1982, ZAHN1984). The three included studies provided efficacy data from 67 participants and tolerability data from 94 participants.

One study compared phenelzine with placebo (JENIKE1997) and all three studies compared MAOIs with other drugs, including fluoxetine (JENIKE1997) and clomipramine (INSEL1983B, VALLEJO1992).

The included studies were between 6 and 12 weeks long (mean length = 9.3 weeks). Patients were classified as outpatient in two studies and mixed in one study. The average age of the patients was 33 years. The duration of illness was 6.7 and 11 years in INSEL1983B and VALLEJO1992 respectively. Two studies were conducted in the US and one in the UK.

The results of one cross-over study (INSEL1983B) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.8.3 MAOIs versus placebo

6.8.3.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on the efficacy of treatment in adults with OCD.	JENIKE1997

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on the likelihood of leaving the study early (K = 1; N = 41; RR = 1.05; 95% CI, 0.24 to 4.61) I.	JENIKE1997

6.8.4 MAOIs versus other drugs

6.8.4.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring phenelzine over clomipramine on reducing anxiety as measured by the Hamilton Anxiety Scale (K = 1; N = 26; SMD = -0.88; 95% CI, -1.69 to –0.07). I	VALLEJO1992

Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between MAOIs and other drugs on the tolerability of treatment.	JENIKE1997 VALLEJO1992

6.8.4.2 Results summary from additional cross-over trials

In INSEL1993B, 24 patients with OCD were randomised to placebo-clorgyline-placebo or placebo-clomipramine-placebo sequences and then crossed over to the other treatment over a 6-month period. The duration of each active treatment was 6 weeks. Ten patients completed the entire cross-over sequence.

Neither clorgyline nor clomipramine were effective in reducing symptom severity as measured by the Leyton Interference score, though clomipramine was effective in ameliorating scores on 13 of 19 measures while clorgyline showed no improvement on all 19 measures. When the 10 patients who completed the entire cross-over sequence were compared, improvement with clomipramine surpassed that seen with clorgyline on the Comprehensive Psychiatric Rating Scale – Obsessive-Compulsive modified subscale, the Global Obsessive-Compulsive scale and the Obsessive-Compulsive rating scale. Only one patient displayed more improvement with clorgyline than with clomipramine on obsessive-compulsive symptoms. Side effects were prevalent on placebo, clorgyline and clomipramine.

6.8.5 Clinical summary

There is no convincing evidence supporting the efficacy of MAOIs in OCD.

6.9 Anxiolytics

6.9.1 Introduction

OCD is classified in some diagnostic systems, such as DSM-IV, together with the anxiety disorders. However, anxiolytics (excluding SSRIs) are not considered effective for the treatment of the core symptoms of OCD. The dependence producing effects of benzodiazepines argue against their use as long-term treatments.

6.9.2 Treatments included

The following treatments were included:

- Buspirone
- Clonazepam.

6.9.3 Studies considered

The review team conducted a new systematic search of electronic databases for RCTs that assessed the efficacy and tolerability of anxiolytics. Seven studies were identified, of which four did not meet the inclusion criteria of the GDG (KIM1997, LIN1979, ORVIN1967, WAXMAN1977). The three included studies provided efficacy data from 70 participants and tolerability data from 47 participants.

One study compared clonazepam with placebo (HOLLANDER2003C) and two with other drugs (HEWLETT1992, PATO1991).

The included studies were between 6 and 26 weeks long. Patients were classified as outpatient in one study. The average age of the patients was 35 years. All three studies were conducted in the US.

The results of one cross-over study (HEWLETT1992) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

6.9.4 Anxiolytics versus placebo

6.9.4.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the efficacy of treatment.	HOLLANDER2003C

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the tolerability of treatment. HOLLANDER2003C

6.9.5 Anxiolytics versus other drugs

6.9.5.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the efficacy of treatment in adults with OCD.	PATO1991

Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the tolerability of treatment in adults with OCD.	PATO1991

6.9.5.2 Results summary from additional cross-over trial

In HEWLETT1992, 28 patients were initially randomly assigned to clomipramine, clonazepam, clonidine or diphenhydramine as control and then crossed-over to each of the other treatments. The study took place over 26 months, with 6 weeks in each treatment. Sixteen patients completed the entire cross-over sequence. Eight patients discontinued because of adverse side effects.

Clonazepam and clomipramine produced significantly lower Y-BOCS scores than did diphenhydramine and clonidine. In turn, Y-BOCS scores in diphenhydramine were significantly lower than baseline, whereas there was no difference between ratings for clonidine treatment and baseline. While the response rate for diphenhydramine and clonidine was 27% and 20% respectively, the response rates for clonazepam and clomipramine was higher at 48% and 54% respectively. Patients who responded to clonazepam tended to respond to clomipramine, the cross-response rate being 73% and patients who responded to clomipramine tended to respond to clonazepam, cross-response rate 80%.

6.9.6 Clinical summary

Although there is some evidence suggesting potential efficacy for clonazepam, the dependence-producing effects known to occur with benzodiazepines argues against the routine use of the drug for OCD.

6.10SSRIs/ clomipramine versus non-SRIs

6.10.1 Introduction

There is a body of opinion that SSRIs and clomipramine are superior to non-SRIs. This is based on the evidence that SRIs have been found to be effective compared to placebo, whereas non-SRIS have not. Thus prescribers may well be more likely to choose an SRI for treating OCD Head-to-head trials comparing SRIs with non-SRIs may influence the confidence with which treatment preferences are made.

6.10.2 Treatments included

The following treatments were included:

- SSRIs
- Clomipramine
- TCAs
- MAOIs
- Anxiolytics

Venlafaxine was excluded because it has the properties of both SRIs and TCAs.

6.10.3 Studies considered

Trials which performed head-to-head comparisons of a SSRI or clomipramine with a non-SRI were considered. Six studies were included, GOODMAN1990A, HOEHNSARIC2000, JENIKE1997, PATO1991, VALLEJO1992, VOLAVKA1985. The studies provided efficacy data on 278 patients and tolerability data on 343 patients. The duration of treatment ranged between 6 and 12 weeks. Patients were classified as outpatient in 4 studies, patient setting was not reported in 2 studies. The average age of the patients was 35 years. Five studies were conducted in the US and one study was conducted in the UK. Two studies included patients with comorbid depression.

6.10.3.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring SSRIs/Clomipramine over non-SRIs on reducing obsessive-compulsive symptoms, as measured by the clinician-rated Y-BOCS in adults with OCD (K =4; N = 258; SMD = -0.3; 95% C.I., -0.54 to –0.05). I	GOODMAN1990A HOEHNSARIC2000 JENIKE1997 PATO1991

Tolerability

There is limited evidence suggesting a difference favouring SSRIs/Clomipramine over non-SRIs on the likelihood of leaving the study early due to adverse events in adults with OCD (K = 5; N = 279; RR = 0.51; 95% C.I., 0.28 to 0.95). I	GOODMAN1990A HOEHNSARIC2000 PATO1991 VALLEJO1992 VOLAVKA1985

6.10.4 Clinical summary

There is some evidence that SRIs, not considering venlafaxine, are better than non-SRIs such as TCAs, MAOIs and anxiolytics on efficacy and tolerability, but this is limited by the small number of studies in this comparison. This evidence is supported by the fact that SRIs are better than placebo, whereas TCAs are not better than placebo.

6.11 Other pharmacological interventions

6.11.1 Introduction

In the search for alternative treatment for OCD, a wide range of other pharmacological treatments have been investigated. However, each has been subjected to only very limited study and the results have not supported further systematic exploration. This section considered alternative treatments in patients who were not yet considered as treatment-refractory.

6.11.2 Treatments included

The following treatments were included:

- Inositol
- Pindolol
- Tryptophan
- Gabapentin
- Triptans
- Anti-testosterone
- St. John's Wort
- Kava kava
- Ginko biloba
- Amphetamine

- Oxytocin
- Clonidine
- Practolol
- Ondansteron
- Ritanserin
- Anti-androgen
- Cyproterone.

6.11.3 Studies considered

A systematic search was conducted for the above reports on the above treatments in patients with OCD or Body Dysmorphic Disorder. The search yielded 704 records, of which 2 studies were RCTs and 9 studies were non-RCTs.

Full details of the search strategy are given in Appendix 6.

6.11.4 Inositol versus placebo

6.11.4.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between inositol and placebo on the efficacy of treatment.	FUX1996

6.11.5 Oxytocin versus placebo

6.11.5.1 Clinical evidence statements

Tolerability	*Included studies*
There is limited evidence suggesting a difference favouring placebo over oxytocin on the likelihood of reporting adverse effects in adults with OCD (K = 1; N = 12; RR = 6.00; 95% C.I., 1.00 to 35.91). I	DENBOER1992

6.11.6 Descriptive review

The quality of the evidence was poor, since most studies were case reports (Ansseau et al, 1987; Casas et al, 1986; Eriksson, 2000; Feldman et al, 1988; Knesevich, 1982) or had very small sample sizes (Hewlett et al, 2003; Salzberg & Swedo, 1992; Taylor & Kobak, 2000; Yaryura Tobias & Bhagavan, 1977).

Ansseau et al (1987) reported the treatment of a patient with OCD with intranasal oxytocin or placebo in a double-blind cross-over manner, each randomised period lasting 4 weeks. While no significant changes in obsessive-compulsive symptoms were present during the placebo period, intranasal oxytocin induced a clear improvement on the Comprehensive Psychopathology Rating Scale (CPRS-OC). However, the patient also complained of gross memory disturbances during oxytocin therapy and increased psychotic symptoms. In contrast, Salzberg & Swedo (1992) failed to find any discernible effects of oxytocin or vasopressin on obsessive-compulsive symptoms in 3 patients randomised in a blinded fashion to intranasal oxytocin, intranasal vasopressin or normal saline.

Casas et al (1986) studied the anti-obsessional effect of antiandrogen, Cyproterone actate (CPA). In patients with OCD, during the 10 days of treatment there was a notable improvement in obsessive symptoms and a decrease in compulsive rituals. After the third month of treatment, there was a gradual reappearance of obsessive symptoms, in particular anxiety and the need to perform compulsive rituals. Feldman et al (1988) failed to find improvement in obsessive-compulsive symptoms after treating a patient with OCD with CPA for five months. Eriksson (2000) studied the effect of a long-acting gonadotropin-releasing hormone analogue, triptorelin. After 4 months of treatment, the patient experienced fewer obsessive-compulsive symptoms and further improvement after 3 more months of treatment.

Hewlett et al (2003) studied the anti-obsessional properties of 5-HT3 receptor antagonist, Ondansetron, in 8 patients with OCD. Over the course of the trial, 8 patients experienced an average of 28% reduction on the Y-BOCS from baseline, while 3 patients achieved a clinically significant response (≥ 35% reduction in Y-BOCS score). Moreover, at 2 weeks follow-up of the 6 completers, there was a 45% worsening of symptoms associated with discontinuing treatment.

Taylor et al (2000) conducted a 12-week open-label trial of St. John's Wort in 12 patients. Treatment consisted of a fixed dose of 450mg of 0.3% hypericin twice daily. At the end of treatment, five out of 12 patients were rated as "much improved" or "very much improved" on the Clinical Global Impression of Improvement scale. A significant improvement similar to that found in clinical trials was found on the Y-BOCS, with a mean change of 7.4 points.

Knesevich (1982) reported improvement on compulsions following a 2-week trial with clonidine (0.1mg t.i.d.) in a patient with OCD. Increasing the dose to 0.1mg q.i.d. caused ritualistic behaviour to return, but the symptoms diminished after dosage was brought back to 0.1mg t.i.d.

Yaryura-Tobais & Bhagavan (1977) reported improvement among seven patients with OCD after treatment with L-tryptophan (3 to 9 g daily) for 1 month. The patients' conditions were stabilized after 6 months to 1 year of treatment.

6.11.7 Clinical summary

In conclusion, the paucity of clinical evidence on other pharmacological treatments for OCD limits our confidence in the efficacy of these alternative pharmacological treatments.

6.12 Treatment strategies for patients showing an incomplete response to SRIs

6.12.1 Introduction

Although most individuals with OCD experience substantial improvements on first-line treatment with SRI drugs, for many the treatment response is not complete. In about 30% cases residual symptoms remain in spite of prolonged treatment. The clinical management of 'incomplete responders' is an area that has not yet been thoroughly investigated, although there is much interest in the area and treatment-studies indicating promising strategies are already entering the scientific literature.

Research into the area of treatment-resistant OCD has been bedevilled by the lack of universally accepted definitions of treatment-response and treatment-failure. Many of the published studies have used different criteria, making it rather difficult to draw generalised conclusions from them. For example, some studies included only extremely refractory cases who had failed to respond to successive treatments with more than one SRI, whereas others included those who had made a partial response to treatment with a single SRI drug. Pallanti et al (2002a) recently proposed criteria based on expert consensus opinion. Specifically, they argued an improvement of ≥35% in baseline Y-BOCS score, or 'much' or 'very much improved' on the CGI-I, represented a meaningful clinical response, while 'remission' required a total Y-BOCS score of less than 16. Those showing between 25-35% improvement in Y-BOCS scores were considered partial responders. Relapse was defined as a 25% worsening in Y-BOCS (or a CGI score of 6), after a period of remission, and the term 'treatment-refractory' was reserved for those who do not

respond to 'all available treatments'. Levels of non-response, according to the numbers of failed treatments, were also defined. It remains to be seen whether these criteria will be universally accepted by the scientific community.

This section reviews research on the pharmacological treatment of individuals failing to completely respond to SRI medication. First there will be a description of current practice, including predictors of treatment failure and definitions of incomplete response. Then the drugs and the studies will be considered, followed by a narrative review of the studies and a clinical summary encapsulating practice points and areas for further research.

6.12.2 Current practice

In contrast to the large amount of data for first-line treatments for OCD, the evidence base for the treatment of individuals who have failed to respond adequately to first-line treatments is rather slim. Indeed, we do not understand clearly which clinical factors might predict a better or worse outcome after pharmacological treatment; few pharmacological studies provide information on response status at outcome and the literature on pharmacological response predictors is sparse and inconsistent. The factor analysis by Mataix-Cols et al (1999) suggested that adults with compulsive rituals, early-onset age, longer duration, chronic course, comorbid tics and personality disorders (especially schizotypal), responded poorly to clomipramine and SSRIs. Additional analyses of large databases for clomipramine and fluoxetine reported better responses for previously SRI-naïve individuals, and poorer responses for those with sub-clinical depression. Those with an earlier age of onset responded poorly to clomipramine, but not to fluoxetine (Ackerman & Greenland, 2002). The more recent analysis of data from a large trial of citalopram also reported that patients with a longer duration of illness or previous SSRI treatment, as well as greater illness severity, were less likely to respond well to drug-treatment (Stein et al, 2001). One small study identified better responses in females (Mundo et al, 1999) and children with comorbid ADHD, tic disorder and oppositional defiant disorder showed a less favourable response (Geller et al, 2003).

OCD responds to treatment gradually, and gains accrue with the passage of time. Some patients respond more slowly than others, and it is important not to anticipate treatment-failure prematurely. There is no agreed consensus on what constitutes an adequate period of treatment, but published expert consensus guidelines suggest at least 12 weeks (Greist et al, 2003; March et al, 1997) and possibly longer may be required in some cases.

In the UK, a number of strategies are currently employed for individuals failing to respond adequately to first-line treatment with SRI drugs.

Sometimes the individual is treated for longer on the original drug to see if a delayed response occurs. Alternatively the individual may be switched to an alternative SRI, or the SRI continued with the introduction of a drug of either the same class (i.e. two SRIs given together) or a drug of another class added in combination (see below). Sometimes the dose of SRI is increased beyond usual recommended daily-dosage limits, or the mode of delivery is changed e.g. to intravenous as opposed to oral delivery. Sometimes the individual is switched to a drug of another class used as a monotherapy. These strategies are usually managed under the guidance of a consultant psychiatrist or her team. The evidence for these strategies will be reviewed below. The evidence for those involving only SRIs will be reviewed first, and those involving drugs of other classes will be reviewed next.

6.12.3 Descriptive review of strategies involving SRIs

The studies considered in this section were based on a literature review on the pharmacotherapy of OCD (Fineberg & Gale, 2004).

6.12.3.1 Switching SRI

Given the limitations of data supporting alternative strategies, and the acceptability of switching from one SSRI to another, this remains the preferred option for many clinicians. Sometimes, however, it is appropriate to persist for longer with a particular SRI even in patients who show little sign of improvement, since a delayed response may occur after 6 months or more. A panel of international OCD experts made recommendations on switching based on their clinical experience at that time (Expert Consensus Guideline, March et al, 1997). They suggest changing the SRI after 8-12 weeks on the maximal dose if the clinical effect was incomplete. They proposed a 40% chance of responding to a second SRI, and a lesser response to a third and suggested switching to clomipramine after 2-3 failed trials on SSRIs. Since that time, a limited evidence-base supporting switching has accrued, but the data remains sparse and inconclusive. In a placebo-controlled sertraline trial, one third of patients benefited from a switch to a second SSRI (Koran et al, 2002). Denys et al (2004) took 43 non-responders to 12 weeks treatment with either paroxetine or venlafaxine and switched them under double blind conditions to the alternate treatment, whereupon 18 (43%) showed a clinical response. A single-blind study in 29 cases of SRI-resistant OCD showed encouraging results for venlafaxine (37.5-375mg) as a monotherapy (Hollander et al, 2003b).

These results hint that patients who have failed to respond to an SSRI may benefit from switching agent, but fail to control for the effect of prolongation of treatment per se. RCTs comparing continuation of the original drug with switching are required to properly evaluate the role of switching in OCD.

6.12.3.2 Increasing dose

Results from uncontrolled case studies suggest that, for some patients, increasing SSRI doses above formulary limits may procure a better effect (Bejerot & Bodlund, 1998; Byerly et al, 1996). Although doses of clomipramine up to 300mg have been systematically investigated and found to be acceptable, the risk of seizures and cardiotoxicity associated with this drug suggest that doses exceeding this should be generally avoided, and if attempted ECG and clomipramine plasma-level monitoringshould be considered.

6.12.3.3 Changing mode of drug-delivery

Changing mode of administration may be considered though this is not practical in many cases. Intravenous clomipramine has been shown to be more effective than placebo in a single double-blind study investigating refractory OCD, with 6 out of 29 patients randomised to clomipramine classed as responders after 14 daily infusions, compared to none receiving placebo infusions (Fallon et al, 1998). A positive open study showing benefit for 21 days intravenous citalopram has also been reported (Pallanti et al, 2002b) but requires confirmation using a controlled study design.

Intravenous clomipramine vs intravenous placebo (for treatment resistant patients)

6.12.3.3.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring intravenous clomipramine over intravenous placebo on the likelihood of treatment response, defined as "much improved" or "very much improved" on the Clinical Global Impressions scale (K = 1, N = 54; RR = 0.79; 95% CI, 0.66 to 0.96). I	FALLON1998
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on the likelihood of leaving the study early (K = 1; N = 54; RR = 0.43; 95% CI, 0.04 to 4.48). I	FALLON1998

6.12.3.4 Combining two SRIs

If a patient fails to respond to successive SRI trials, augmented with CBT, the Expert Consensus Guideline (March et al, 1997) recommended adding another agent to the SRI. The evidence is acknowledged to be limited, based on the results of small RCTs and open case-series.

Combining clomipramine with an SSRI has been proposed for adults or children unresponsive to, or intolerant of SRI monotherapy. This strategy should be approached with caution since the pharmacokinetic interactions on the hepatic cytochrome P 450 isoenzymes may lead to a build-up of clomipramine that could be dangerous, and ECG and clomipramine plasma-level monitoring is advisable. Positive results have been reported from small, uncontrolled case series (Szegedi et al, 1996), although the fluoxetine–clomipramine combination resulted in ECG changes in some cases. Pallanti et al (1999) compared 9 treatment-refractory patients given citalopram plus clomipramine with 7 given citalopram alone, in a randomised open-label trial. They reported a significantly larger improvement in Y-BOCS ratings for those given the combination, all of whom experienced decreases ≥ 35% from baseline. This combination is advantageous in not altering the metabolism of clomipramine, and was well tolerated. No controlled studies of the co-administration of different SSRIs have been published.

6.12.4 Augmentation strategies with drugs of other classes

The following drugs, given in order to improve clinical outcome in individuals failing to completely respond to SRI monotherapy, were included:

- Buspirone (added to fluvoxamine)
- Desipramine (added to SSRI)
- Haloperidol (added to fluvoxamine)
- Inositol (added to SRI)
- Lithium (added to fluvoxamine)
- Nortriptyline(added to clomipramine)
- Olanzapine (added to fluoxetine)
- Pindolol (added to paroxetine)
- Quetiapine (added to SRI)
- Risperidone (added to SRI)
- Venlafaxine.

6.12.4.1 Studies considered

The review team conducted a new systematic search to identify studies that used augmentation strategies in the treatment of OCD. The search identified 46 studies that were of interest (Atmaca et al, 2002; Barr et al, 1997; Bejerot &

Bodlund, 1998; Blier & Bergeron, 1996; Bogetto et al, 2000; Byerly et al, 1996; Crocq, 2002; D'Amico et al, 2003; Dannon et al, 2000; Denys et al, 2002a; Denys et al, 2004; Francobandiera, 2001; Fux et al, 1999; Grady et al, 1993; Hollander et al, 2003a; Koran et al, 2000; Koran et al, 2002; Maina et al, 2003; McDougle et al, 1990; McDougle et al, 1993; McDougle et al, 1995b; McDougle et al, 2000a; McDougle et al, 1991; McDougle et al, 1994; McDougle & Walsh, 2001; Metin et al, 2003; Mohr et al, 2002; Mundo et al, 1998; Noorbala et al, 1998; Pallanti et al, 1999; Pigott et al, 1992b; Pigott et al, 1991; Pigott et al, 1992a; Sevincok & Topuz, 2003; Shapira et al, 2004; Weiss et al, 1999).

6.12.4.2 Combining SRIs and drugs exerting antidepressant or anxiolytic properties

Controlled studies have found no evidence for the efficacy of lithium augmentation in OCD (McDougle et al, 1991; Pigott et al, 1991). Similarly, three double-blind placebo controlled studies have demonstrated that combining buspirone with an SRI is not helpful (Grady et al, 1993; McDougle et al, 1993; Pigott et al, 1992a). Clonazepam is a benzodiazepine with putative effects on serotonin neurotransmission. As a monotherapy it fails to impact on the core symptoms of OCD (Hollander et al, 2003c) though it may help with associated anxiety. Pigott et al (1992b) reported limited efficacy for clonazepam given together with fluoxetine or clomipramine in a placebo-controlled study. Pindolol is a beta-blocker which also acts as an antagonist at presynaptic 5HT1A autoreceptors. Dannon et al (2000) demonstrated efficacy for pindolol when combined with paroxetine in a double-blind placebo-controlled study of 14 treatment resistant cases, but another study combining it with fluvoxamine did not (Mundo et al, 1998). Blier and Bergeron (1996) found a beneficial effect for pindolol only when l- tryptophan was openly added to the combination.

The limitations of adding drugs acting on serotonin led investigators to re-examine the role of noradrenergic agents for OCD. While not evidently effective as a monotherapy, nortryptyline (50mg) administered in combination with 150mg clomipramine was found to be more effective than clomipramine plus placebo in a small 8 week study looking at 30 non-refractory individuals (Noorbala et al, 1998). However, Barr et al (1997) investigated the addition of another noradrenergic antidepressant desipramine to 20 patients who had failed SSRI monotherapy, in a double-blind placebo-controlled study, and found no added benefit.

6.12.4.3 Combining SRIs and drugs with antipsychotic properties

No positive studies of antipsychotic monotherapy in OCD meet today's standards, and OCD is not recognised to respond to these drugs individually. McDougle et al (1990) reported a benefit from adding open-label pimozide (6.5mg) in 17 patients unresponsive to fluvoxamine. Patients with comorbid chronic tics or schizotypal disorder were most responsive. A subsequent

double-blind placebo-controlled study by the same author demonstrated a significant Y-BOCS improvement for low-dose haloperidol (6.2mg) added to fluvoxamine. Eleven of 17 patients receiving the active drug achieved 'responder' status by as early as 4 weeks, compared to none on placebo. Again, a preferential response was seen in patients with comorbid tics (McDougle et al, 1994). Antipsychotics such as haloperidol and sulpiride are first-line treatments for the Tourette Syndrome, so this finding supports a theoretical link between these disorders. This combination increases the side-effect burden, including extra-pyramidal effects. It is therefore recommended to start treatment with very low doses, and increase cautiously subject to tolerability (e.g. 0.25-0.5mg haloperidol, titrated slowly to 2-4mg: (McDougle & Walsh, 2001).

Newer second-generation antipsychotics that modulate serotonin and dopamine neurotransmission also offer promise and have lower risks of side effects. Positive reports from open case-series were confirmed by McDougle et al (2000a) in the first reported double-blind placebo-controlled study showing efficacy for risperidone augmentation in 36 patients unresponsive to 12 weeks on an SRI. Risperidone (2.2mg) was superior to placebo in reducing Y-BOCS scores as well as anxiety and depression, was well-tolerated and there was no difference between those with and without comorbid tics or schizotypy. A smaller double-blind study by Hollander et al (2003a) examined patients failing to respond to at least 2 trials of SRIs. Four of 16 patients randomised to risperidone (0.5-3mg) turned out to be responders, defined as a CGI-I of 1 or 2 and Y-BOCS decrease of > 25% at the 8-week end-point, compared with none of the 6 patients randomised to placebo. However, the results, which were limited by the small sample size, did not reach statistical significance.

Quetiapine has also been the subject of recent controlled investigation. There have been contradictory results from open studies (Denys et al, 2002a; Mohr et al, 2002; Sevincok & Topuz, 2003). However, a placebo-controlled single-blind study looking at 27 cases of SRI-resistant OCD showed a significant advantage from adding quetiapine in doses up to 200mg daily to ongoing SRI (Atmaca et al, 2002). Moreover, the recent double-blind placebo controlled study by Denys et al (2004) showed evidence of efficacy for 8 weeks quetiapine (<300mg) augmentation in 20 patients who had failed to respond to at least 2 SRIs, showing a mean decrease of 31% on baseline Y-BOCS, compared to 20 placebo-treated controls who showed only 6% improvement. Eight out of twenty (40%) patients responded to the quetiapine therapy, compared to two out of twenty (10%) on placebo.

Encouraging results from a small number of open-label studies of olanzapine (Bogetto et al, 2000; Crocq, 2002; D'Amico et al, 2003; Francobandiera, 2001; Koran et al, 2000; Weiss et al, 1999) were not, however, supported by the double-blind placebo controlled study by Shapira et al (2004), which

investigated 44 partial or non-responders to 8 weeks fluoxetine. Both groups improved over the 6-week study period, with no additional advantage for the olanzapine-treated patients compared with extending the duration of fluoxetine monotherapy. Differences in entry criteria between studies may partly explain differences in the results.

There has been a positive open-label study of amisulpride (Metin et al, 2003).

Augmentation with clozapine has not been systematically investigated. The results for clozapine monotherapy in OCD have not been encouraging (McDougle et al, 1995a). Some authors have reported emergent obsessions during treatment with atypical antipsychotics, which may be related to their mixed receptor antagonist properties.

Altogether, these results favour the use of second generation antipsychotics such as risperidone and quetiapine as the first-line strategy for augmentation in resistant OCD. It remains uncertain as to how long patients need to remain on augmented treatment. A small retrospective study by Maina et al (2003) showed that the vast majority of patients who had responded to the addition of an antipsychotic to their SRI, subsequently relapsed when the antipsychotic was withdrawn.

6.12.4.4 Other strategies for refractory OCD

Inositol (18g/day) is an experimental compound that acts through intracellular messenger systems. It was thought to have mild anti-obsessional efficacy but results from a placebo-controlled augmentation study by Fux et al (1999) refute this. Sumatriptan is a 5HT1D agonist used to treat migraine. A small open case series suggested improvement over 4 weeks treatment but, in a double-blind placebo-controlled study of 10 patients, 5 day treatment was associated with a worsening of OCD (Koran et al, 2001).

6.12.5 Clinical summary

Treatment-resistant OCD is now receiving systematic evaluation. The first step to managing resistant OCD is to check adherence to the original treatment . In the case of resistance, augmentation with second generation antipsychotic agents appears a promising strategy for which there is some supportive evidence from controlled clinical trials. Other techniques for resistant cases such as increasing the dose above SPC limits, changing to another SSRI or clomipramine, combining SRIs or changing the mode of drug-delivery are under evaluation. Important questions requiring further investigation include identification of clinically relevant predictors relating to treatment–response and relapse, the clarification of optimal duration of treatment and the evaluation of anti-obsessional treatment in comorbid disorders such as schizophrenia with OCD. Agreed definitions for response, relapse, resistance and refractoriness will improve research in this area.

6.13 Pharmacological interventions for children and young people with OCD

6.13.1 Introduction

OCD frequently begins in childhood or adolescence, its prevalence in under 18s being of the order of 1% (Heyman et al, 2001; Valleni-Basile et al, 1994). As well as causing significant distress to young people, it can be highly disruptive to educational and social development, and if chronic, lead to adult OCD and other mental health problems (Leonard et al, 1993). There is need for long term follow-up studies, to establish whether early recognition and assertive treatment of OCD in young people results in improved adult adaptation, and decreased rates of OCD in adult life. Clinical impressions are that this is the case, so there are two major reasons to treat child and adolescent OCD effectively; alleviation of a serious mental illness in young people, and prevention of chronic mental illness and secondary disabilities in adults.

Symptoms of OCD are similar in children and adults, but developmental stage affects the way they present, and the way they are described by the sufferer. It is very common for children and adolescents to involve family members in their rituals. Treatment of young people needs to involve their family/carers, and also often needs liaison with school or college. The effective treatments in adults also seem to be effective in children, so it is important that the condition is recognised as early as possible, with access to the specifically effective treatments (Heyman, 1997; Rapoport & Inoff-Germain, 2000).

6.13.2 Current practice

Childhood and adolescent OCD is often undiagnosed for long periods, or is misdiagnosed by practitioners unfamiliar with the characteristic symptoms (Chowdhury et al, 2004). Because the disorder has specific and effective treatments, it is desirable for children's OCD to be detected and treated as soon as possible. Equally, it is important not to confuse a normal stage of child development, which is characterised by some repetitive behaviours, with OCD. This normally occurs between age 3 and 6 years, and is self-limiting and rarely time-consuming or distressing.

Diagnosis of OCD can be assisted by the use of standardised instruments, such as the Children's Yale Brown Obsessive Compulsive Schedule (Scahill et al, 1997). Once the diagnosis of OCD has been made in a young person, it is important that they receive the correct treatment, and that therapists are not distracted by focussing on additional problems which might detract from treating the OCD. Children with OCD often have additional anxiety

disorders, school or separation problems, and mood difficulties, but these are often secondary to the OCD, and resolve with treatment for the primary disorder. If they do not, it is important to target comorbidities with an appropriate intervention.

Attention needs to be given to educating the child and their family about OCD, how anxiety operates to maintain symptoms, and the range of treatment choices. With all treatments, including drug treatments, parents and young people need to be fully appraised of the benefits and risks as we currently understand them. At present (2004) in the UK two SSRIs are licensed for use in children and adolescents. As with many drugs used in paediatrics, a specialist may recommend a medication for the treatment of OCD in young people which is unlicensed, but for which there is clinical trial evidence of safety and efficacy. Explanations should be given to the family about why an unlicensed preparation is suggested.

6.13.3 Treatments included

The following treatments were included:

- SSRIs (fluoxetine, fluvoxamine, sertraline)
- Clomipramine
- Desipramine.

6.13.4 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy of pharmacological interventions among children and young people with OCD. Eighteen published trials were identified, of which four did not meet the inclusion criteria of the GDG. One unpublished trial (CARPERNTER1999) was identified. The 15 included studies provided efficacy data from 1034 participants and tolerability data from 1068 participants.

Of the included studies, eight compared SSRIs with placebo (CARPENTER1999, GELLER2004, GELLER2001, LIEBOWITZ2002, MARCH1998, POTS2004, RIDDLE1992, RIDDLE2001), four compared clomipramine with placebo (DEVEAUGHGEISS1992, MARCH1990, FLAMENT1985, RAPOPORT1980), three compared clomipramine with desipramine (LEONARD1989A, LEONARD1991A, RAPOPORT1980) and one study compared continuation of clomipramine treatment with desipramine substitution (LEONARD1991A).

All included acute-phase randomised studies were between 8 and 20 weeks long (mean length = 12 weeks). Patients were classified as outpatient in 7

studies, mixed in 3 studies and unclear in four studies. The average age of the patients was 13.2 years. The average duration of illness based on 7 studies was 3.6 years. All studies were conducted in the US, including three studies which were multi-centre studies and one study which was conducted in the US and Canada.

Additional data on suicidal behaviour/ ideation was obtained from a review made public by the FDA (Hammad, 2004). The review came about after the FDA commissioned a re-classification of the original patient-level data by experts in suicidality at Columbia University (Iyasu, 2004). The FDA concluded that this blinded classification process identified and corrected many misclassification errors, providing more accurate risk estimates (Hammad, 2004). Four studies were included in the review (GELLER2004, GELLER2001, MARCH1998, RIDDLE2001) providing data on 616 participants.

The results of three cross-over studies (FLAMENT1985, LEONARD1989A, RAPOPORT1980) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

6.13.5 SSRIs versus placebo

6.13.5.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25%+ 40%+ reduction on the Children's Y-BOCS (K = 3; N = 430; RR = 0.70; 95% C.I., 0.59 to 0.83). I	GELLER2001 GELLER2004 RIDDLE2001
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured on the Children's Y-BOCS (K = 7; N = 718; SMD = -0.43; 95% C.I., -0.58 to -0.28). I	GELLER2001 GELLER2004 LIEBOWITZ2002 MARCH1998 POTS2004 RIDDLE1992 RIDDLE2001
Tolerability	
It is possible that SSRIs when compared to placebo increase the risk of suicidal behaviour/ ideation (K = 4; N = 616; RR = 1.81; 95% C.I., 0.46 to 7.13). I[10]	GELLER2001 GELLER2004 MARCH1998 RIDDLE2001

[10] Given the seriousness of the outcome, the risk was considered to be clinically important despite the wide confidence interval

There is evidence suggesting that SSRIs when compared to placebo increase the risk of leaving the study early due to adverse effects (K = 6; N = 732; RR = 2.74; 95% C.I., 1.46 to 5.14). I	GELLER2001 GELLER2004 LIEBOWITZ2002 MARCH1998 POTS2004 RIDDLE1992 RIDDLE2001

6.13.6 Clomipramine versus placebo

6.13.6.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the Children's Y-BOCS (K = 1; N = 16; SMD = -0.94; 95% CI, -1.99 to 0.11). I	MARCH1990

Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the tolerability of treatment.	DEVEAUGHGEISS1992 MARCH1990

6.13.6.2 Results summary from additional cross-over trials

In FLAMENT1985, following a one-week evaluation period patients were randomised to 5 weeks of clomipramine or placebo and then crossed-over to 5 weeks of the other treatment. Twenty-three patients entered the drug study and 19 completed the entire cross-over sequence.

Compared with placebo, clomipramine was significantly more effective in relieving obsessional symptoms, as measured on LOI-CV symptom (t = 2.19, p = 0.04), resistance (t = 2.12, p = 0.05) and interference scores (t = 2.24, p = 0.04), the OCR scale (t = 3.05, p 0.007), and the NIMH-OC scale (t = 2.83, p = 0.02). Side-effects scores increased markedly during the first week with clomipramine and by week 5, they were significantly higher than with placebo (t = -2.52, p = 0.02). One patient experienced a grand mal seizure during clomipramine treatment.

In RAPOPORT1980, patients were randomised to 3-5 week consecutive periods of treatment with clomipramine, desipramine or placebo and then crossed-over to each of the other 2 treatments over 16 weeks. Nine patients entered the study and eight completed the entire cross-over sequence.

At the end of treatment, symptom severity as measured by the Leyton Interference and Resistance subscales was comparable across clomipramine, desipramine and placebo. Side effects did not differ between treatment periods.

6.13.7 Clomipramine versus desipramine substitution

6.13.7.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring clomipramine continuation over desipramine substitution on the likelihood of relapse as defined by the Physician's Relapse Scale (K = 1; N = 20; RR = 4.89; 95% CI, 1.37 to 17.49). I	LEONARD1991A
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine continuation and desipramine substitution on the likelihood of leaving the study early (K = 1; N = 21; RR = 3.27; 95% CI, 0.15 to 72.23). I	LEONARD1991A

6.13.7.2 Results summary from additional cross-over trials

In LEONARD1989A, following a 2-week single-blind placebo phase patients were randomised to 5-week consecutive periods of treatment with clomipramine or desipramine and then crossed-over to the other treatment. Forty-nine patients participated in the trial.

At the end of treatment, symptom severity was decreased to a greater extent by clomipramine than desipramine based on the NIMH Obsessive-Compulsive scale (F = 16.62, p = 0.002), Ward OCD Rating scale (F = 21.16, p = 0.00001) and CPRS Obsessive-Compulsive subscale (F = 10.34, p = 0.003). There was a significant effect of order of drug, whereby the rate of relapse was greater in the clomipramine-to-desipramine cross-over sequence than the desipramine-to-clomipramine cross-over sequence.

In RAPOPORT1980, patients were randomised to 3-5 week consecutive periods of treatment with clomipramine, desipramine or placebo and then crossed-over to each of the other 2 treatments over 16 weeks. Nine patients entered the study and eight completed the entire cross-over sequence.

At the end of treatment, symptom severity as measured by the Leyton Interference and Resistance subscales was comparable across clomipramine,

desipramine and placebo. Side effects did not differ between treatment periods.

6.13.8 SSRI versus placebo: continuation/discontinuation

6.13.8.1 Descriptive review

Two studies examined the continuation of SSRIs in children, CARPENTER1999 and LIEBOWITZ2002.

CARPENTER1999 was an unpublished 32-week two-phase trial of paroxetine. In the first phase, patients received open-label paroxetine for 16 weeks. In the second phase, patients who responded to the first phase were either continued with paroxetine at the final daily dose achieved during the first phase or discontinued from paroxetine onto placebo in a double-blind randomised fashion. The duration of the second phase was 16 weeks. Three hundred and thirty-five patients participated in the phase I trial, of which 194 entered the second phase.

The results indicated that the rate of relapse, defined as (a) an increase in CGI Global Improvement score by 1 point for 2 consecutive visits, (b) an increase in CGI Global Improvement score by 2 or more points at any single visit, and (c) a CGI Global Improvement score of 5 or more points at any time, was not significantly different between patients continuing with paroxetine (34.7%) and patients discontinuing from paroxetine (43.9%). Comparing the response rates between the two groups based on a 25% or greater reduction of CY-BOCS from baseline favoured paroxetine continuation over discontinuation (25% vs. 13.3%, RR = 0.86, 95% C.I., 0.75 to 0.99). Symptom severity increased in both groups during the continuation phase as measured by the CY-BOCS, though this increase was lower in the paroxetine continuation group than the discontinuation group (SMD = -3.3, 95% C.I., -5.77 to –0.83).

In LIEBOWITZ2002, patients who responded to an 8-week acute phase of fluoxetine or placebo were entered into an 8-week maintenance phase. Response was defined as "much improved" or "very much improved" on the Clinical Global Impression – Improvement scale. Patients continued to receive the same drug as during the acute phase and were maintained on the final dose achieved during the acute phase.

Of the 43 patients who entered the acute phase, 18 entered the maintenance phase. At the end of the 8-week maintenance phase, fluoxetine patients had lower CY-BOCS scores than did placebo patients, mean (SD) was 5.64 (5.41) and 11.43 (11.12) respectively, F (1,14) = 9.22, p = 0.009. None of the fluoxetine patients relapsed, while one placebo patient relapsed. There were significantly more fluoxetine than placebo responders (57% vs. 27%, Chi2 =

3.94, p = 0.05). Twenty-seven percent of the fluoxetine patients reported at least one adverse event, compared to none among the placebo patients.

6.13.9 Clinical summary

There is evidence supporting the treatment of OCD in children and adolescents with SSRIs. The literature is not extensive, but it is consistent in showing beneficial effects in terms of symptom remission and improvements in global functioning. There are always concerns about the use of drugs in young people, particularly medications which act on the developing central nervous system. These potential risks have to be weighed against the known risks of untreated OCD on emotional, educational and social development, and the impact of chronic OCD on adult adaptation. More research is needed in children and adolescents, in the in the acute phase, in long term follow-up, and on educational and cognitive progress.

Although the evidence-base is small for psychological treatment, clinical consensus recommends the use of psychological treatment as a first-line in young people with OCD. However, in severe or chronic cases, where cognitive behaviour therapy has been ineffective or is unavailable, or where the patient chooses medication, this is an effective treatment option, either alone or ideally with, cognitive behaviour therapy.

There is consensus opinion that for children and young people with OCD psychological treatment (CBT) should be the first-line treatment. Clinical trial evidence, although not extensive, suggests that CBT and SSRI/SRI treatment have similar efficacy, but drug treatments are assumed to have more side-effects. At the time of writing this guideline, there is concern about the use of SSRIs in children and young people with major depression, because of limited evidence of antidepressant efficacy, and evidence of adverse events, particularly suicidal thought and behaviours. In contrast, in children and young people with OCD clinical trials have shown strong evidence for efficacy and no evidence of increased suicidal thought and behaviours.

OCD in adults and in children/young people has similar symptoms, course and treatment responsiveness. The good practice points emphasised above for adults receiving treatment with SSRI medication all apply to children, and some additional ones are given below.

6.14Pharmacological interventions for people with BDD

6.14.1 Introduction

This topic is of interest because of the lack of experience by psychiatrists in treating BDD and the paucity of evidence concerning pharmacotherapy in this area

6.14.2 Current practice

There are no surveys of how BDD is currently treated or managed in the UK. Our clinical impression is that that many patients are treated with anti-psychotic drugs after an influential case series of pimozide in delusional disorder (Riding & Munro, 1975). The popularity of pimozide has since dwindled probably because of concerns about its toxicity and having to perform an ECG prior to its administration. This does not seem to have stopped prescribing of anti-psychotic drugs for BDD.

Current practice is not underpinned by a strong evidence base .e. g. there are few studies upon which to base clinical decisions, and there are concerns about the generalisability of patient samples.

6.14.3 Treatments included

The following treatments were included:

- Fluoxetine
- Clomipramine
- Desipramine
- Pimozide.

6.14.4 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy of pharmacotherapy in BDD.

Two trials met the eligibility criteria set by the GDG. Phillips et al (2002) entered 74 participants with BDD or its delusional variant into the study. Sixty seven were randomised to either fluoxetine or a placebo for 12 weeks. The range of the dose of fluoxetine was between 40 and 80mg a day. Fluoxetine was significantly more effective than placebo on the YBOCS modified for BDD at week 8 (N = 77; SMD = -0.6; 95% C.I., -1.09 to –1.11) and continuing at weeks 10 and 12. The baseline for the fluoxetine group was 31.5 (S.D. 5.6) reducing to 21.0 (S.D. 9.8) at 12 weeks, which represented a 33% reduction on the main outcome measure. The rate of response, defined as a 30% or greater decrease from baseline on the Y-BOCS, was greater in fluoxetine than placebo at 8 weeks (N = 77, RR = 0.58; 95% C.I., 0.39 to 0.85). Delusional patients were as likely as those of non-delusional patients to respond to fluoxetine, and no delusional patients responded to the placebo. The effect was independent of comorbid diagnoses of OCD or depression.

There has been one double-blind randomised crossover trial comparing clomipramine with desipramine (Hollander et al, 1999). Forty participants with BDD or its delusional variant were entered and 29 were entered in a 2-

week, single-blind run-in, followed by 8 weeks of either clomipramine or 8 weeks of desipramine which was then crossed over. Clomipramine is a potent serotonergic reuptake inhibitor and a tricyclic anti-depressant. Desipramine is a potent noradrenergic reuptake inhibitor and another tricyclic. Both drugs are equally effective in the treatment of depression. Both clomipramine and desipramine effected reduced severity of obsessive-compulsive symptoms from baseline as measured by the BDD-YBOCS scores, baseline BDD-YBOCS was 25.4 ± 7.2, BDD-YBOCS following clomipramine treatment was 16.2 ± 8.5 and following desipramine treatment was 20.7 ± 7.7. The reduction in symptom severity was significantly greater following clomipramine treatment than following desipramine treatment, ($F1,21 = 11.03$, $p = 0.003$). Response rates were 65% for clomipramine and 35% for desipramine, based on 25% improvement on the BDD-YBOCS. Treatment efficacy was not influenced by comorbidity of OCD, depression or social phobia.

There are limitations to this trial, including a lack of a placebo arm, a maintenance phase after the crossover and potential carry over effects, which are inherent in crossover designs. There is some evidence that the response may have been greater if a higher dose of clomipramine (mean 138mg/day) was used and for a longer duration (at least 12 to 16 weeks).

6.14.5 SSRI Augmentation

Phillips (2005) has conducted a randomised controlled trial of pimozide augmentation of fluoxetine. Twenty eight people with BDD or its delusional variant who had failed to respond to fluoxetine participated in an 8-week double-blind study of up to 10mg pimozide or placebo augmentation of fluoxetine. Pimozide was no more effective than placebo. 18.2% of subjects responded to pimozide and 17.6% to placebo. There was no significant effect of baseline delusionality on endpoint BDD severity. Delusionality did not decrease significantly more with pimozide than placebo. Possible explanations of the lack of efficacy include the study's low power and the modest mean pimozide dose. In OCD, augmentation of an SSRI with pimozide and haloperidol has found higher response rates in patients with a tic disorder or schizotypal personality disorder. No BDD subject in this study had either additional diagnosis.

6.14.6 Descriptive review

6.14.6.1 Anti-depressants

Serotonin reuptake inhibitors (SRIs) other than fluoxetine or clomipramine may be of benefit both theoretically and from the evidence of four case series. There are similar modest benefits from 4 open label trials. There are two open

label case series of fluvoxamine (Perugi et al, 1996; Phillips et al, 1998), one of citalopram (Phillips & Najjar, 2003) and one case series of clomipramine (Hollander et al, 1989).

Phillips et al (1998) entered 30 participants with BDD who received fluvoxamine over 16 weeks. The average dose was 238.8mg and the range was 50 to 300mg. The YBOCS modified for BDD decreased from 31.1 (S.D 5.4) at baseline to 16.9 (S.D. 11.8) at 16 weeks. This represents a 45% reduction on the main outcome measure. Sixty-three per cent of participants responded based on a criterion of a 30% decrease or more on the YBOCS modified for BDD. Fluvoxamine was as effective in participants with an additional diagnosis of delusional disorders as without.

Perugi et al (1996) entered 15 participants with BDD in an open label trial. The duration was for 10 weeks. The average dose was 208mg and the range of dose was 100 to 300mg. They did not use the modified YBOCS as an outcome measure but reported a 60% reduction over 12 weeks on symptom scores and 10 out of the 15 participants responding on the Clinical Global Impression scale.

Phillips & Najjar (2003) entered 15 participants with BDD or its delusional variant in an open label study of citalopram over 12 weeks. The average dose was 51.3 mg and the range was 10 to 60 mg. The YBOCS modified for BDD decreased from 30.7 (S.D 4.9) at baseline to 15.3 (S.D. 10.6) at 12 weeks. This represents a 50% reduction on the main outcome measure. 73.3% of participants responded defined as 30% decrease or more on the YBOCS modified for BDD. Citalopram was as effective in participants with an additional diagnosis of delusional disorders as without.

Hollander et al (1989) report on a case series of 5 participants with BDD who all responded to either clomipramine or fluoxetine. Four of the five patients had failed to respond previously to drugs that had some serotonergic action including tricyclics, trazodone and lithium.

Phillips (1996b) conducted a retrospective case review of 130 patients who had 316 treatment trials of which 42% of 65 SRI trials had led to an improvement on the Clinical Global Impression scale, compared to 30% of 23 trials with a monoamine oxidase inhibitor and 15% of 48 trials with a non-SRI tricyclic drug.

No continuation, maintenance or discontinuation studies of an SRI have been reported. Expert opinion and clinical experience suggest that, like OCD, there may be small further gains with an SRI after 12-16 weeks treatment. Furthermore, like OCD, there is a high rate of relapse on discontinuation on a SRI (Phillips et al, 2001).

Lastly there are case reports on the benefit of sertraline (El-Khatib & Dickey, 1995) and two cases with intra-venous clomipramine (Pallanti & Koran, 1996). There are two case reports of the benefit of an MAOI, tranlcypromine (Jenike, 1984) and one with a combination of phenelzine, trimipramine and perphenazine (Phillips, 1991).

6.14.6.2 Anti-psychotic drugs as a monotherapy

Anti-psychotic drugs are frequently prescribed for BDD after a case series describing the benefit of pimozide in individuals with delusional disorder (Riding & Munro, 1975). However the case series included cases of delusions of infestation, delusions of body odour and dysmorphic delusions.

Phillips et al (1996b) report a retrospective survey of medication trials. She reported that only 3% of 83 trials of an anti-psychotic were of any benefit in BDD. Grant (2001) has described one case report of olanzapine for BDD without delusional disorder. The individual however fulfilled diagnostic criteria for BDD, alcohol dependence and bipolar II disorder. At the end of 3 weeks, she reported no preoccupation with her appearance and no longer met criteria for BDD.

6.14.6.3 SSRI switching or augmentation studies

In individuals with BDD who have failed to respond to an SSRI, or who have a partial response to an SSRI, then switching to another SSRI or augmentation with another drug has been tried.

Phillips et al (2001) report that in those subjects who failed to respond to an adequate SRI trial, 42.9% (n=6) responded to at least one subsequent SRI trial and 43.5% (n=10) of subsequent SRI trials received by these subjects were effective.

Phillips et al (2001) report on an open label series of buspirone (extended from Phillips, 1996a) in a chart review of patients who failed to respond to an SRI alone or have had only a partial response. In 12 participants, buspirone was added after an adequate does of an SRI, 33.3% of trials was successful. The mean dose was 56.5mg (range 30-80mg daily) and was as effective in delusional as non-delusional cases.

6.14.6.4 Adolescents with BDD

Albertini & Phillips (1999) report on 33 children and adolescents with BDD of whom 10 out 19 (53%) treated with an SRI improved on the Clinical Global Impression scale. No non-SRI medications in 8 trials were effective.

There is one case report on clomipramine in an adolescent with BDD with delusional disorder (Sondheimer, 1988) and one case report of doxepine and behaviour therapy in an adolescent with BDD (Sobanski & Schmidt, 2000).

6.14.7 Clinical summary

The only placebo controlled RCT in BDD suggest benefit from fluoxetine or clomipramine in BDD. There are also several case series of other SRIs that support this finding.

No evidence exists on the optimal dose of a SRI in BDD but expert opinion is that SRIs in BDD may have a dose response relationship similar to OCD and that the maximum tolerated dose should be tried.

No evidence exists on the optimal duration of trial of an SRI but expert opinion suggests that at least 12 weeks is required. A SRI is however associated with a high rate of relapse on discontinuation (similar to the treatment of OCD) although this has not been formally evaluated.

There is no evidence for the efficacy of an anti-psychotic in BDD as a monotherapy or as augmentation strategy with an SRI. An anti-psychotic may still be useful for the symptomatic treatment of individuals with BDD who are highly agitated.

There is no evidence for the benefit of MAOIs, non-SRI tricyclics, or atypical SRIs as a monotherapy for BDD. There is no evidence for the benefit or ECT or psychosurgery in BDD.

No studies have yet compared a SRI with CBT or a combination of the two treatments. Expert opinion suggests that the combination of CBT with an SRI is helpful in moderate to severe BDD.

Limited evidence suggests that SSRIs can be effective in children and adolescents with BDD with a similar response to adults with BDD and adolescents with OCD. However no evidence exists for the safety of SSRIs in children and adolescents with BDD.

6.15 Clinical practice recommendations

6.15.1 Initial treatment options

Children and young people

6.15.1.1 If psychological treatment is declined by children or young people with OCD and their families, or they are unable to engage in treatment, an SSRI may be cautiously considered with specific arrangements for careful monitoring of adverse events**. [B]**

6.15.2 How to use pharmacological interventions for adults

Current published evidence suggests that SSRIs are effective in treating people with OCD and BDD, although evidence for the latter is limited and less certain. However, SSRIs may increase the risk of suicidal ideas and self harm in people with depression and in younger people . It is currently unclear whether there is an increased risk for people with OCD or BDD. Regulatory authorities recommend caution in their use until evidence for differential safety has been demonstrated. Appropriate monitoring is therefore needed, especially when initiating treatment and around dose changes. Patients should also be warned about, and monitored for, relapse and discontinuation/ withdrawal symptoms when stopping or reducing SSRIs.

Starting the treatment

6.15.2.1 Common concerns about taking medication for OCD/BDD should be addressed. Patients should be advised, both verbally and with written material, that:

- Craving and tolerance do not occur [C]
- There is a risk of discontinuation/withdrawal symptoms on stopping, missing doses, or reducing the dose of the drug [C]
- There is a range of potential side effects, including worsening anxiety, suicidal thinking and self-harm, which needs to be carefully monitored, especially in the first few weeks of treatment [C]
- There is commonly a delay in the onset of effect of up to 12 weeks, although depressive symptoms improve more quickly [C]
- Taking medication should not be seen as a weakness. [GPP]

Monitoring risk

6.15.2.2 People with OCD or BDD started on SSRIs who are not considered to be at increased risk of suicide or self harm should be monitored closely and seen at an appropriate and regular basis agreed by the patient and the healthcare professional, and this should be recorded in the notes. **[GPP]**

6.15.2.3 Because of the potential increased risk of suicidal thoughts and self harm associated with the early stages of SSRI treatment,

people with OCD or BDD who are younger adults, are depressed, or are considered to present an increased suicide risk, should be carefully and frequently monitored by healthcare professionals. Where appropriate, other carers **-** as agreed by the patient and the healthcare professional **-** may also contribute to the monitoring until the risk is no longer considered significant. This should be recorded in the notes. **[C]**

6.15.2.4 For people with OCD or BDD at high risk of suicide, a limited quantity of medication should be prescribed**. [C]**

6.15.2.5 When a person with OCD or BDD, especially when combined with depression, is assessed to be at a high risk of suicide, the use of additional support such as more frequent direct contacts with primary care staff or telephone contacts should be considered, especially during the first weeks of treatment**. [C]**

6.15.2.6 For people with OCD or BDD, particularly in the initial stages of SSRI treatment, healthcare professionals should actively seek out signs of akathisia or restlessness, suicidal ideation, and increased anxiety and agitation. They should also advise patients to seek help promptly if these are at all distressing. **[C]**

6.15.2.7 People with OCD or BDD should be monitored around the time of dose changes for any new symptoms or worsening of their condition. **[C]**

Choice of drug treatment

Selective Serotonin Reuptake Inhibitors (SSRIs)

6.15.2.8 For adults with OCD, the initial pharmacological treatment should be one of the following SSRIs: citalopram, fluoxetine, fluvoxamine, paroxetine or sertraline. **[A]**

6.15.2.9 For adults with BDD the initial pharmacological treatment should be fluoxetine as there is more evidence for its effectiveness in BDD than other SSRIs. **[B]**

6.15.2.10 When prescribing an SSRI for OCD, consideration should be given to using a product in a generic form. Fluoxetine and citalopram, for example, would be reasonable choices because they are generally associated with fewer discontinuation/withdrawal symptoms. However, fluoxetine is associated with a higher risk for drug interactions. **[C]**

6.15.2.11 In the event that a person with OCD or BDD develops marked and/or prolonged akathisia, restlessness or agitation while taking an SSRI, the use of the drug should be reviewed. If the patient prefers, the drug should be changed to a different SSRI. **[C]**

6.15.2.12 Healthcare professionals should be aware of the increased risk of drug interactions when prescribing an SSRI to people with OCD who are taking other medications. **[GPP]**

6.15.2.13 When OCD or BDD symptoms fail to respond to a full course of treatment with an SSRI, healthcare professionals should check that the patient has taken the drug regularly and in the prescribed dose and that there is no interference from alcohol or substance use. **[GPP]**

6.15.2.14 If the response to a standard dose of an SSRI for a person with OCD or BDD is inadequate and there are no significant side effects after 4 to 6 weeks, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. **[B]**

6.15.2.15 For people with OCD or BDD, the rate at which the dose of an SSRI should be increased should take into account therapeutic response, adverse effects, and patient preference. Patients should be warned about, and monitored for, the emergence of side effects during dose increases **[GPP]**

6.15.2.16 If treatment for OCD or BDD with an SSRIs is effective, it should be continued for at least 12 months to prevent relapse and allow for further improvements. **[B]**

6.15.2.17 When a person with OCD or BDD has taken an SSRI for 1 year after remission, healthcare professionals should review the need for continued treatment with the patient. This review should include consideration of the severity and duration of the initial illness, number of previous episodes, presence of residual symptoms, and concurrent psychosocial difficulties. **[GPP]**

6.15.2.18 If treatment for OCD or BDD with an SSRI is continued for an extended period beyond 12 months, the need for continuation should be reviewed at regular intervals, agreed between the patient and the prescriber, and written in the notes. **[GPP]**

6.15.2.19 For people with OCD or BDD, to minimize discontinuation/withdrawal reactions when reducing or stopping SSRIs, the dose should be tapered gradually over several weeks according to the person's need. The rate of reduction should take into account starting dose, drug half-life and particular profiles of adverse effects. **[C]**

6.15.2.20 Healthcare professionals should encourage people with OCD or BDD who are discontinuing SSRI treatment to seek advice if they experience significant discontinuation/withdrawal symptoms. **[C]**

Other drugs

With the exception of clomipramine, other antidepressants should not normally be used in the treatment of OCD or BDD. Most other drugs have limited or no use in this context.

6.15.2.21 Tricyclic antidepressants other than clomipramine should not normally be used for treating OCD**. [B]**

6.15.2.22 Tricyclic related antidepressants should not normally be used for treating OCD. **[C]**

6.15.2.23 SNRIs, including venlafaxine, should not normally be used for treating OCD. **[B]**

6.15.2.24 MAOIs should not normally be used for treating OCD. **[B]**

6.15.2.25 Anxiolytics should not normally be used for treating OCD , except cautiously for short periods to counter the early activation of SSRIs. **[B]**

6.15.2.26 Antipsychotics as a monotherapy should not normally be used for treating OCD . **[B]**

6.15.2.27 Antipsychotics as a monotherapy should not normally be used for treating BDD or its delusional variant. **[C]**

6.15.3 How to use clomipramine for adults

Clomipramine can be offered as a second line drug for OCD/BDD. Always do an ECG and check blood pressure before starting treatment if there is significant risk of cardiovascular disease. Dose changes should be gradual.

6.15.3.1 For people with OCD or BDD who are at a significant risk of suicide, healthcare professionals should only prescribe small amounts of clomipramine at a time and monitor the patient regularly until the risk of suicide has subsided because of the toxicity of clomipramine in overdose. [GPP]

6.15.3.2 An ECG should be carried out and blood pressure measurement taken before prescribing clomipramine for a patient with OCD or BDD at significant risk of cardiovascular disease. [C]

6.15.3.3 For people with OCD or BDD, if the response to the standard dose of clomipramine is inadequate, and there are no significant side effects, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. [C]

6.15.3.4 For people with OCD or BDD, treatment with clomipramine should be continued for at least 12 months if the treatment appears to be effective and because there may be further improvement. [B]

6.15.3.5 For people with OCD or BDD, when discontinuing clomipramine, doses should be reduced gradually in order to minimise potential discontinuation/withdrawal symptoms. [C]

6.15.4 How to use pharmacological treatments in children and young people

In adults with OCD treated by medication, there is clinical trial evidence that supports practice on the onset of therapeutic response, dose needed, rate of increase of dose, duration of treatment, likelihood of relapse on discontinuation. Trials of these aspects have not been done in children and/or young people, but the following good practice for prescribing SSRIs or clomipramine is based on adult trials and clinical experience.

How to use SSRIs in children and young people

6.15.4.1 When antidepressant medication is prescribed to children and young people with OCD or BDD, it should be in combination with concurrent CBT. If children and young people are unable to engage with concurrent CBT, specific arrangements must be made for careful monitoring of adverse events. [C]

6.15.4.2 Children and young people with OCD or BDD started on SSRIs should be carefully and frequently monitored and seen at an appropriate and regular basis agreed by the patient, his or her family

and the healthcare professional, and this should be recorded in the notes. [GPP]

6.15.4.3 If an SSRI is to be prescribed to children and young people with OCD or BDD, it should only be following assessment and diagnosis by a psychiatrist who should also be involved in decisions about dose changes and discontinuation. [GPP]

6.15.4.4 When an SSRI is prescribed to children and young people with OCD, it should be a licensed medication, sertraline or fluvoxamine, except in cases with significant co-morbid depression, when fluoxetine should be used (because of current regulatory requirements). [A]

6.15.4.5 When an SSRI is prescribed for children and young people with BDD it should be fluoxetine. [C]

6.15.4.6 For children and young people with OCD or BDD who also have significant depression, the NICE recommendations for the treatment of childhood depression should be followed (('Depression in children: identification and management of depression in children and young people in primary care and specialist services', publication expected August 2005), and caution should be observed with specific monitoring for suicidal thoughts or behaviours. [GPP]

6.15.4.7 Children and young people with OCD or BDD started on SSRIs should be informed about the rationale for the drug treatment, the delay in onset of effect (up to 12 weeks), the time course of treatment, the possible side effects, and the need to take the medication as prescribed. Discussion of these issues should be supplemented by written information appropriate to the child/young person's and family's needs. [GPP]

6.15.4.8 The starting dose of medication for children and young people with OCD or BDD should be low, especially in younger children. A half or quarter dose of the normal starting dose may be considered for the first week. [C]

6.15.4.9 If a lower dose of medication for children and young people with OCD or BDD is ineffective, the dose should be increaseduntil a therapeutic response is obtained with careful and close monitoring for adverse events. The rate of increase should be gradual and should take into account the delay in therapeutic response (ie up to 12 weeks) and the age of the patient. Maximum recommended doses for children and young people should not be exceeded. [C]

6.15.4.10 Children and young people prescribed SSRIs should be carefully and closely monitored for agitation, disinhibition, hostility, suicidal ideation and self-harm by the prescribing doctor and the professional delivering the psychological intervention. Children and young people and their families should be informed that if there is any sign of new symptoms of these kinds, they should make urgent contact with the prescribing doctor. [GPP]

6.15.4.11 Where children or young people with OCD or BDD respond to treatment with an SSRI, medication should be continued for at least 6 months post remission (symptoms that are not clinically significant and full functioning for at least 12 weeks). [C]

How to use clomipramine in young people

6.15.4.12 Young people with OCD or BDD and their parents should be advised about possible side-effects of clomipramine, including toxicity in overdose. [C]

6.15.4.13 Before starting to treat a young person with OCD or BDD with clomipramine, an ECG and blood pressure monitoring should be carried out to exclude cardiac conduction. [C]

6.15.4.14 If the response of the young person with OCD or BDD to the standard dose of clomipramine is inadequate, and there are no significant side effects, a gradual increase in dose may be cautiously considered. [C]

6.15.4.15 Treatment of a young person with OCD or BDD with clomipramine should be continued for at least 6 months if the treatment appears to be effective and because there may be further improvement. [B]

Stopping or reducing SSRIs and clomipramine in children and young people

6.15.4.16 In children and young people with OCD or BDD, an attempt should be made to withdraw medication if remission has been achieved (i.e. symptoms are no longer clinically significant and the child or young person is fully functioning) and maintained for 6 months, and if this is their wish. Patients and their family should be warned that relapse and/or discontinuation/withdrawal symptoms may occur, and to contact their medical practitioner should symptoms of discontinuation/withdrawal arise. [C]

6.15.4.17 For children and young people with OCD or BDD, to minimize discontinuation/withdrawal reactions on reducing or

stopping SSRIs, the dose should be tapered gradually over several weeks according to the individual's need. The rate of reduction should take into account starting dose, drug half-life and particular profiles of adverse effects.. [C]

6.15.4.18 Children or young people with OCD or BDD should continue with psychological treatment throughout the period of drug withdrawal because this may reduce the risk of relapse. [C]

Other drugs

6.15.4.19 Tricyclic antidepressants other than clomipramine should not be used to treat OCD or BDD in children and young people. [C]

6.15.4.20 Other antidepressants (MAOIs, SNRIs) should not be used to treat OCD or BDD in children and young people. [C]

6.15.4.21 Antipsychotics should not be used alone in the routine treatment of OCD or BDD in children or young people, but may be cautiously considered as an augmentation strategy. [C]

7 Combined interventions and intensive interventions for OCD

7.1 Introduction

There have been many advances in the treatment of OCD over the past 35 years, with the development of effective pharmacological and psychological treatments for a disorder that was previously considered extremely refractory to treatment (Black, 1974). Despite the overall success of these treatments, the situation is far from ideal (Foa et al., 2000). The acute efficacy of SRI-based pharmacotherapy is still moderate both in terms of the proportion of people who respond and their average response; a significant proportion of people report adverse effects, and many relapse on discontinuing medication. Likewise, the acute efficacy of exposure and response prevention-based treatments is also moderate both in terms of the proportion of people who respond and their average response. The long term maintenance of gains is unknown, and a significant proportion refuse treatment or fail to complete. Given the moderate efficacy of both treatment types, their differential and combined effects become a question of obvious interest. From the point of view of the person with OCD, the ability to make informed choices depends on information on the relative efficacies, possible adverse effects, durability of effects and availability of treatments and their combinations. From the point of view of health care providers, knowledge of efficacy and effectiveness is required in order to make decisions about which resources to provide.

7.2 Psychological versus pharmacological interventions

7.2.1 Introduction

There have been at least five English language meta-analyses addressing the comparison but some of these have contrasted arms from different studies, in some cases using different measures, in order to address this question (Abramowitz, 1997; Christensen et al, 1987; Cox et al, 1993; Kobak et al, 1998; Van Balkom et al, 1994). These reviews have not reached any consistent conclusions. Although there has been a keen debate between proponents of psychological and biological approaches to understanding OCD and its treatment, as yet there is no unified theory that can readily accommodate the key elements of both approaches of OCD. However, a broad biopsychosocial framework is accepted by many, at least for the treatment of OCD. Beyond its academic interest, the question of differential efficacy has important implications for people with OCD, their families and carers, and health care providers.

7.2.2 Current practice

With the advent of SSRIs that are generally better tolerated than clomipramine and better known by medical practitioners given their use for a range of disorders, pharmacotherapy has become more widely available. People are often offered pharmacotherapy in primary care, whether or not they are referred on to secondary or tertiary care. Despite the fact that many professionals are trained each year in cognitive-behaviour therapies, there is an increasing demand on therapists as CBT becomes indicated for a greater range of disorders and waiting lists for psychological services are common (Lovell & Richards, 2000). Although there is increasing provision of mental health resources in primary care, there are relatively few professionals who have currently received training to provide the focused and structured treatments that are required for OCD. Consequently, many people do not have ready access to CBT of any type. Although some people are at least eventually able to choose when they obtain access to both, in a proportion of cases the treatment received may be determined more by referral patterns and availability of resource rather than by informed choice.

7.2.3 Studies considered[11]

The review team conducted a new systematic search for RCTs that assessed the efficacy of alternative (psychological versus pharmacological) or combination interventions among adults and children and young people with OCD. Six studies were identified, of which two studies (HEMBREE2003, OCONNOR1999) did not meet inclusion criteria. Of the four included studies, two studies were on adults with OCD (FOA2005, MARKS1980), while two studies were on children and young people with OCD (DEHAAN1998, POTS2004). The studies on adults with OCD provided efficacy data on 104 patients and tolerability data on 84 participants (FOA2005). The studies on children and young people with OCD provided efficacy and tolerability data on 79 participants (DEHAAN1998, POTS2004).

The studies involving adults compared exposure and response prevention with clomipramine (FOA2005) and exposure and response prevention and placebo with relaxation and clomipramine (MARKS1980). The studies involving children compared CBT with clomipramine (DEHAAN1998) and CBT with sertraline (POTS2004).

The included studies were 12 weeks long, except MARKS1980, which was 10 weeks long though data from this study was extracted at the 7-week time-point when all patients receiving relaxation were switched to exposure. In the

[11] Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication, except where a study is *in press* or only submitted for publication, then a date is not used).

adults studies, the average age of the participants was 35 years and the mean duration of illness was 14 years. The adults studies did not report the mean final dosage.

In the studies on children and young people, participants were classified as outpatient. Participants received a maximum dosage of the drug of 200mg per day and a mean final dosage of the drug of 196 mg per day. The average age of the participants ranged from 12-18 years across studies. Comorbid conditions included anxiety disorder, eating disorder and tic disorder.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

7.2.4 Behaviour therapy versus clomipramine

7.2.4.1 Clinical evidence statements[12]

Efficacy[13]	*Included studies*
There is limited evidence suggesting a difference favouring exposure and response prevention over clomipramine on reducing obsessive-compulsive symptoms as measured on the Y-BOCS or the Compulsive Checklist (K = 2; N = 68; SMD = -0.67; 95% CI, -1.16 to -0.17). I	FOA2005 MARKS1980
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure and response prevention and clomipramine on the likelihood of leaving the study early (K = 1; N = 84; RR = 1.02; 95% CI, 0.62 to 1.67). I	FOA2005

7.2.5 CBT versus clomipramine (children and young people)

7.2.5.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and clomipramine on the efficacy of treatment.	DEHAAN1998

[12] The full list of all evidence statements generated from meta-analyses (and the associated forest plots) will be available on the CD-ROM that accompanies the guideline. Where a meta-analysis was not possible (or not appropriate), a summary of the results from each study used to generate a statement can be found in Appendix 15.

[13] In the case of SMD or WMD, negative effect sizes favour the treatment group.

Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and clomipramine on the likelihood of leaving the study early (K = 1; N = 23; RR = 2.36; 95% CI, 0.11 to 52.41). I	DEHAAN1998

7.2.6 CBT versus Sertraline (children and young people)

7.2.6.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and sertraline on the efficacy of treatment.	POTS2004

Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and sertraline on the likelihood of leaving the study early (K = 1; N = 56; RR = 1.5; 95% CI, 0.27 to 8.3). I	POTS2004

7.2.7 Clinical summary

Based on the two head-to-head comparisons for adults with OCD reviewed here, there is some evidence for greater efficacy of exposure and response prevention over clomipramine. The results on tolerability were inconclusive. There are no comparisons for SSRIs. These results would argue for ERP being offered to all adults with OCD, although ultimately it will depend on patient preference.

The evidence for children and young people is inconclusive for both efficacy and tolerability based on the two studies reported here. Given concerns about the safety of SSRIs and clomipramine for children and young people, CBT should be offered as initial treatment. When CBT is not available or when the young person and their family prefer, SRIs may be considered.

7.3 Combination interventions

7.3.1 Introduction

Despite numerous studies of both CBT and SRIs, there are relatively few that have investigated the combination of both. This is somewhat surprising given that both are only partially effective, many people relapse on discontinuing SRIs, and some people cannot tolerate CBT, especially the exposure and response prevention component because of anxiety. Consequently it is important to investigate whether the combination can increase efficacy and overcome some of the limitations of each treatment.

7.3.2 Current practice

The Expert Consensus Panel for OCD (March et al, 1997) compiled guidelines with recommendations on a comprehensive range of issues relating to pharmacological and psychological treatments. They concluded that CBT should be the first line treatment for mild OCD for adults and young people and for children (regardless of severity). Combined SRI and CBT should be the first line treatment for moderate to severe OCD for both adults and young people, but not children. However, it is not clear at present what the optimal timing should be for introducing each treatment (Foa et al, 2002a).

It is likely that in the UK many people with OCD do indeed receive combined treatment, especially for moderate to severe OCD, but due to availability of CBT this may be offered sequentially and may happen in a relatively unplanned manner rather than by explicit decision and careful planning. In some cases there may be little coordination between the various professionals involved in care who may be in different services with different levels of experience and knowledge of the treatment of OCD, particularly of the other treatment modality. In other cases, especially in integrated multidisciplinary teams who can provide both treatments, the combined treatment is more likely to be explicitly planned and better coordinated.

7.3.3 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of combination interventions among adults and children with OCD. Eighteen studies were identified, of which eleven did not meet the eligibility criteria of the GDG. Five studies had no extractable data (KASVIKIS1988A, MARKS1988, MAWSON1982, OSULLIVAN1991, PETER2000), three studies were based on analyses from other studies (COTTRAUX1993, KASVIKIS1998, LAX1992), one study included patients with phobic neurosis (AMIN1997), one study was not an RCT (HEMBREE2003) and in another study patients were not properly randomised to treatment groups (OCONNOR1999). The seven included studies (COTTRAUX1990, FOA2005, HOHAGEN1998, MARKS1980, NEZIROGLU2000, POTS2004, VANBALKOM1980) provided efficacy data on 470 participants and tolerability data on 469 participants.

All five included studies on adults featured behaviour therapy (BT) and serotonin reuptake inhibitor (SRI) combinations (COTTRAUX1990, HOHAGEN1998, MARKS1980, FOA2005, VANBALKOM1998). One study also featured a cognitive therapy (CT) and fluvoxamine combination (VANBALKOM1998). Of the two studies on children and young people, one study (NEZIROGLU2000) featured a behaviour therapy and fluvoxamine combination intervention, while the other study (POTS2004) featured a CBT and sertraline combination intervention.

Six studies were between 8 weeks and 24 weeks long (mean length = 13 weeks), while one study was one year long (NEZIROGLU2000). In four studies participants were classified as outpatient, inpatient in one study, mixed in one study and unclear in one study. In the adult studies, the mean age of the participants was 35 years. The duration of illness based on 4 studies on adults was 13 years. The average age of the participants in the studies on children and young people was 13 years. Three studies were conducted in the US, one each in the UK, France, Germany and the Netherlands. Participants receiving fluvoxamine received up to 300mg per day and participants receiving clomipramine received a mean final dose of 180 mg per day. Participants in the children study received up to 200mg of the drug per day.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 15.

7.3.4 BT + SRIs versus BT

7.3.4.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring multimodal behaviour therapy + fluvoxamine over multimodal behaviour therapy + placebo on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS (K = 1; N = 49; RR = 0.31; 95% CI, 0.10 to 1.00). I	HOHAGEN1998
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (K = 3; N = 126; SMD = -0.37; 95% CI, -0.72 to -0.01). I	HOHAGEN1998 FOA2005 VANBALKOM1998
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing compulsive symptoms as measured by the Compulsive Activity Checklist (K = 2; N = 51; SMD = -0.55; 95% CI, -1.12 to 0.01). I	COTTRAUX1990 MARKS1980
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing the time spent performing rituals (K = 2; N = 51; SMD = -0.81; 95% CI, -1.38 to –0.23). I	COTTRAUX1990 MARKS1980
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing depressive symptoms (K = 4; N = 137; SMD = -0.73; 95% CI, -1.08 to –0.38). I	COTTRAUX1990 HOHAGEN1998 MARKS1980 VANBALKOM1998
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + SRI	COTTRAUX1990 FOA2005

combinations and BT on the tolerability of treatment. VANBALKOM1998

7.3.5 BT + clomipramine versus clomipramine

7.3.5.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring exposure and response prevention and clomipramine over clomipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (K = 1; N = 45; SMD = -0.63; 95% CI, -1.23 to –0.03). I	FOA2005
There is limited evidence suggesting a difference favouring exposure and response prevention plus clomipramine over clomipramine on the likelihood of response, defined as "much improved" or "very much improved" on the Clinical Global Improvement scale (K = 1; N = 80; RR = 0.53; 95% CI, 0.33 to 0.87). I	FOA2005
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure and response prevention + clomipramine and clomipramine on the tolerability of treatment.	FOA2005

7.3.6 Exposure and response prevention (ERP) + fluvoxamine versus Anti-ERP + fluvoxamine

7.3.6.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the efficacy of treatment.	COTTRAUX1990
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the tolerability of treatment.	COTTRAUX1990

7.3.7 CT+ fluvoxamine versus CT

7.3.7.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CT + fluvoxamine and CT on the efficacy of treatment.	VANBALKOM1998
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CT + fluvoxamine and CT on the likelihood of leaving the study early (K = 1; N = 49; RR = 1.74; 95% CI, 0.75 to 4.03). I	VANBALKOM1998

7.3.8 BT + fluvoxamine versus CT + fluvoxamine

7.3.8.1 Clinical evidence statements

Efficacy	*Included studies*
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + fluvoxamine and CT + fluvoxamine on the efficacy of treatment.	VANBALKOM1998
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + fluvoxamine and CT + fluvoxamine on the likelihood of leaving the study early (K = 1; N = 52; RR = 0.86; 95% CI, 0.43 to 1.70). I	VANBALKOM1998

7.3.9 BT + fluvoxamine versus fluvoxamine (children and young people)

7.3.9.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring BT + fluvoxamine over fluvoxamine on reducing obsessive-compulsive symptoms as measured by the Children's Y-BOCS 52 weeks after beginning the treatment (K = 1; N = 10; SMD = -1.50; 95% CI, -3.00 to 0.00). I	NEZIROGLU2000

7.3.10 CBT + Sertraline v Sertraline (children and young people)

7.3.10.1 Clinical evidence statements

Efficacy	*Included studies*
There is limited evidence suggesting a difference favouring CBT + sertraline over Sertraline on the likelihood of relapse, defined as	POTS2004

a score of less than or equal to 10 on the Children's Y-BOCS (K = 1; N = 56; RR = 0.59; 95% CI, 0.38 to 0.92). I	
There is limited evidence suggesting a difference favouring CBT + sertraline over Sertraline on reducing the severity of obsessive-compulsive symptoms as measured by the Children's Y-BOCS (K = 1; N = 56; SMD = -0.59; 95% CI, -1.13 to -0.05). I	POTS2004
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and Sertraline on the likelihood of leaving the study early (K = 1; N = 56; RR = 1; 95% CI, 0.22 to 4.54). I	POTS2004

7.3.11 CBT + Sertraline v CBT (children and young people)

7.3.11.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the efficacy of treatment.	POTS2004
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the likelihood of leaving the study early (K = 1; N = 56; RR = 1; 95% CI, 0.22 to 4.54). I	POTS2004

7.3.12 Clinical summary

The evidence from five studies suggests that there is greater improvement for OCD symptoms from combined SRIs and exposure and response prevention when compared to exposure and response prevention alone. There is also evidence from a single study that the combination may be better than SRI alone (clomipramine in this case). These results suggest that adults with OCD should be offered the possibility of combined treatment. However, the evidence to date is from simultaneous combined treatment and we do not know whether this is the best way of using the two treatments together.

For children and young people, one study found greater improvement in OCD symptoms with the combination of sertraline and CBT over sertaline alone but not over CBT alone. For children and young people, one small study found some superiority for the combination of CBT and fluvoxamine over fluvoxamine alone after 52 weeks. These results suggest for children and young people, especially given the concerns about safety of SRIs in young people, CBT should be offered first, but that combined treatment may also be offered.

7.3.13 Combinations of an SRI and CBT in BDD

No RCTs have been conducted that compare an SRI or any other medication with CBT or a combination of the two. There are a few case reports of combination treatments highlighted in the psychological interventions but they do not assist in guiding clinical practice.

7.4 Clinical practice recommendations

7.4.1 Initial treatment options

Adults

7.4.1.1 For adults with OCD with mild functional impairment who are unable to engage in low intensity CBT or for whom low intensity treatment has proved to be inadequate, healthcare professionals should offer the choice of either a course of an SSRI or more intensive CBT (of more than 10 therapist hours per patient) that includes ERP because these treatments appear to be comparably efficacious. [C]

7.4.1.2 For adults with OCD with moderate functional impairment, healthcare professionals should offer the choice of either a course of an SSRI or more intensive CBT (of more than 10 therapist hours per patient) that includes ERP because these treatments appear to be comparably efficacious. [B]

7.4.1.3 Adults with BDD with mild to moderate functional impairment should be offered a course of CBT (including ERP that addresses key features of BDD such as checking, comparing, avoidance and preoccupation) in individual or group formats or an SSRI, depending upon the patient's preference. [B]

7.4.1.4 Adults with OCD or BDD with severe functional impairment, or for whom psychological or pharmacological treatments have proved ineffective, should be offered combined treatment with an SSRI and CBT. [C]

7.4.2 Poor response to treatment

Poor response to initial treatment for adults

If initial treatment does not result in a clinically significant improvement in both symptoms and functioning, other treatment options should be considered. When additional treatment options also fail to produce an adequate response, multidisciplinary teams with specific expertise in OCD/BDD should become involved.

7.4.2.1 If a person with OCD/BDD has not responded adequately to treatment with an SSRI (within 12 weeks) or CBT (of more than 10 therapist hours per patient), he or she should be offered combined treatment with CBT (including ERP) in addition to an SSRI. [C]

7.4.2.2 If a person with OCD/BDD has not responded adequately after 12 weeks of combined treatment with CBT (including ERP) and an SSRI, or has not responded to an SSRI alone nor engaged with CBT, he or she should be offered either a different SSRI or clomipramine. [C]

7.4.2.3 Clomipramine should be considered in the treatment of OCD or BDD after an adequate trial of at least one SSRI has been ineffective or poorly tolerated; if the patient prefers clomipramine; or has had a previous good response to it. [C]

7.4.2.4 If an adult with OCD or BDD has not responded to a full trial of at least one SSRI, a full trial of combined treatment with CBT (including ERP) and a full trial of clomipramine, they should be referred to multidisciplinary teams with specific expertise in the treatment of OCD/BDD for assessment and further treatment planning. [GPP]

7.4.2.5 The assessment of people with OCD and BDD referred to multidisciplinary teams with specific expertise in OCD/BDD should include a comprehensive assessment of their symptom profile, previous pharmacological and psychological treatment history, adherence to prescribed medication, history of side effects, co-morbid conditions such as depression, suicide risk, psychosocial stressors, relationship with carers and personality factors. [GPP]

7.4.2.6 For adults with OCD who have not responded to a full trial of at least one SSRI, a full trial of combined treatment with CBT (including ERP) and a full trial of clomipramine, the following treatment options should also be considered (note: there is no evidence of the optimal sequence of the options listed below):

- Additional CBT or CT [C]
- Adding an antipsychotic to an SSRI or clomipramine [C]
- Combining clomipramine and citalopram. [C]

7.4.2.7 For adults with BDD who have not responded to a full trial of at least one SSRI, a full trial of combined treatment with CBT (including ERP) nor a full trial of clomipramine, the following

treatment options should also be considered (note: there is no evidence of the optimal sequence of the options listed below):

- Additional CBT or CT [GPP]
- Adding buspirone to an SSRI [C]
- Combining clomipramine and citalopram. [C]

7.4.2.8 Treatments such as combined antidepressants and antipsychotic augmentation should not be routinely initiated in primary care. [GPP]

Poor response to initial treatment in children and young people

If CBT involving the family has not produced an adequate response in terms of a clinically significant reduction in symptoms and increase in functioning within 12 sessions, then review and consider further options according to the age of the child as described below.

Current published evidence suggests that SSRIs are effective in treating children and young people with OCD. The only SSRIs licensed for use in children and young people with OCD are fluvoxamine and sertraline. However, with depression SSRIs can cause significant adverse reactions, including increased suicidal thoughts and self-harm, although they may be safer when combined with psychological treatments. The UK regulatory authority has contraindicated all SSRIs in paediatric depressive illness, except fluoxetine. Although the risk associated with the use of SSRIs in children and young people with OCD is unclear, appropriate caution should be observed, especially in the presence of comorbid depression.

7.4.2.9 If a child or young person with OCD or BDD has not shown an adequate response to a full trial of CBT involving the family within 12 sessions, a multidisciplinary review should be carried out. [GPP]

7.4.3 Intensive treatment services and inpatient services for people with OCD or BDD

OCD and BDD can usually be treated managed in the community and in primary care. However, people with severe and/or chronic problems that have not responded adequately to treatment should be referred to multidisciplinary teams with specialist expertise in the treatment of OCD/BDD. Occasionally inpatient treatment may be needed for children, young people or adults who are at particular risk or whose ability to function is severely impaired. Special support may be needed, especially for young

adults with impaired autonomy and personal functioning as a result of severe OCD with onset in childhood or adolescence.

7.4.3.1 People with severe, chronic, treatment-refractory OCD/BDD should have access to specialist treatment services staffed by multiprofessional teams of healthcare professionals with expertise in the management of the disorders. [C]

7.4.3.2 Inpatient services, with specific expertise in OCD/BDD, are appropriate for a small proportion of people with OCD or BDD, and may be considered when:

- there is risk to life
- there is severe self-neglect
- there is extreme distress or impairment
- a person has not responded to adequate trials of pharmacological/psychological/combined treatments over long periods of time in other settings
- a person has additional diagnoses, such as severe depression, anorexia nervosa or schizophrenia, that make out-patient treatment more complex
- a person has a reversal of normal night/day patterns that make attendance at any day-time therapy impossible
- the compulsions and avoidance behaviour are so severe or habitual that they cannot undertake normal activities of daily living. [GPP]

7.4.3.3 A small minority of adults with long-standing and disabling obsessive-compulsive symptoms that interfere with daily living and have prevented them from developing a normal level of autonomy may, in addition to treatment, need suitable accommodation in a supportive environment that will enable them to develop life skills for independent living. [GPP]

7.4.3.4 Children and young people with severe OCD or BDD with high levels of distress and/or impaired functioning and who have not responded to adequate treatment in outpatient settings, or those with significant self-neglect or risk of suicide should be offered assessment for intensive inpatient treatment in units where specialist treatment for children or young people with OCD or BDD is available. [GPP]

7.4.4 Discharge after recovery

After full recovery, children, young people and adults with OCD or BDD should be followed up for a year. After discharge, those re-referred should be seen quickly and should not be placed on a routine waiting list.

7.4.4.1 When a child, young person or adult with OCD or BDD is in remission (symptoms that are not clinically significant and full functioning for 12 weeks), they should be reviewed regularly for 12 months by a mental healthcare professional. The exact frequency of contact should be agreed between the professional and service user and/ or carer and recorded in the notes. At the end of this period if recovery is maintained the person can be discharged to primary care. [C]

7.4.4.2 Children, young people or adults who have been successfully treated, discharged but re-referred after a first episode of OCD or BDD should be seen as soon as possible rather than placed on a routine waiting list. [GPP]

7.4.4.3 Following multidisciplinary review, if a child (6 to 11 years) with OCD or BDD with moderate to severe functional impairment does not respond adequately to CBT involving the family, the addition of an SSRI to ongoing psychological treatment may be cautiously considered. [B][14]

7.4.4.4 Following multidisciplinary review, if a young person (12 to 18 years) with OCD or BDD with moderate to severe functional impairment does not respond adequately to CBT involving the family, an SSRI should be offered in addition to continuing psychological treatment. [B][14]

7.4.4.5 If treatment with an SSRI, in combination with CBT involving the family, for a young person (12 to 18 years) with OCD or BDD is unsuccessful or is not tolerated because of side effects, cautious consideration may be given to the use of another SSRI or clomipramine, especially if the young person has had a positive response to these alternatives in the past. **[B][14]**

[14] For BDD the recommendation is grade C.

8 Other medical interventions for OCD

8.1 Introduction

A minority of people suffering from OCD remain refractory to all standard pharmacological and psychological treatments. It is largely, but not exclusively, this group that have been considered for treatment with other medical interventions. The medical interventions reviewed for OCD were electroconvulsive therapy (ECT), transcranial magnetic stimulation, ablative neurosurgical procedures, and two non-ablative procedures, namely, deep brain stimulation, and vagus nerve stimulation. It should be noted that because of the nature of these interventions, many of which may involve invasive and ablative procedures, a high proportion of patients who have undergone these procedures would have met strict referral criteria, especially in the more recent studies. These criteria will include previous and often repeated trials of pharmacotherapy and psychological therapies such as behaviour therapy. Consequently, the studies reviewed below are based on a limited group of patients and so represent a select sample with particular characteristics. Most studies are necessarily small in nature and reflect particular practices conducted at specific sites, and often by a small group of individuals (see also Freeman et al, 2000, for a further discussion of these and related issues).

In the case of invasive procedures, evaluation of the evidence is further complicated by the difficulties in performing controlled trials, particularly randomised control trials with credible sham procedures. Overall there were insufficient data to complete a systematic review and hence a narrative review was undertaken. It should be noted that the review addresses OCD symptoms only in the context of refractory OCD rather than other potential co-morbid disorders. This decision, although perhaps arbitrarily limiting access to some potentially useful data on mixed samples, follows the strategy used elsewhere in the OCD Guideline.

8.2 Electroconvulsive therapy

8.2.1 Introduction

Historically, ECT has occasionally been used for the treatment of intractable OCD. Although there are relatively few reports specifically for OCD, severe and intractable OCD is often associated with severe depression for which ECT may be indicated. It is entirely possible that significant numbers of people with OCD have received ECT, although the primary indication for the treatment would be the severe depression. Some protocols for neurosurgery

for intractable OCD suggest trials of ECT before considering ablative procedures (e.g. Matthews & Eljamel, 2003). Consequently, it is important to consider the evidence base for ECT for OCD.

8.2.2 Current practice

People with severe OCD may occasionally receive ECT and it has been recommended by the Expert Consensus Guideline for OCD for treatment refractory patients who may also be depressed only if they have not responded to three or more trials of SRIs nor to CBT (March et al, 1997). There are no current recommendations for ECT for OCD in the UK. The practice of ECT for other conditions, namely depressive illness, schizophrenia, mania, and catatonia, is discussed in detail in NICE technology Appraisal No. 59.

8.2.3 Studies considered

A total of nine papers were found specifically addressing ECT for OCD from 1973-2003: one descriptive paper, five single case reports (two of which are letters), one letter describing three cases, one open trial, and one retrospective review of 32 cases treated over a 20 year period.

8.2.4 Descriptive review

There are four case reports describing a successful outcome for ECT as a treatment for OCD (Casey & Davis, 1994; Husain et al, 1993; Mellman & Gorman, 1984; Thomas & Kellner, 2003). The case reports are generally of poor quality and lack methodological rigour. A further case report describes the onset of mania following the use of ECT with OCD and treatment was discontinued (Chung et al, 2001). Three cases of successful outcome following ECT for OCD are described in a letter (Beale et al, 1995) although the absence of outcome measures precludes any firm conclusions regarding outcome.

Khanna et al (1988a) conducted an open trial with 9 subjects, all of whom met DSM-III criteria for OCD (APA, 1980). Measures of OCD symptoms were administered at pre-treatment, during and post-ECT. Monthly follow-up assessments were conducted for 6 months. The authors reported that all subjects returned to pre-trial state within 6 months. The largest study (Maletzky et al, 1994) is a retrospective review of 32 patients with OCD (19 of whom were described as non depressed). All subjects had previously received trials of CBT and pharmacotherapy with little or no effect. Subjects were evaluated on the MOCI (Maudsley Obsessive Compulsive Inventory) and two depression scales at pre- and post-treatment and at 6- and 12-month follow-ups. The results indicated that the non-depressed group improved on measures of OCD symptoms at 12-month follow-up, but depression scores deteriorated substantially. Overall, 18 of 32 (56%) maintained some

improvement at 12-month follow-up. These results have to be treated with extreme caution as all of the subjects also received active treatment such as medication during the 1-year follow-up so any improvement cannot be attributed solely to ECT.

8.2.4.1 Electroconvulsive therapy in BDD

There are eight published case reports of ECT, six of which were unsuccessful (Phillips, 1991) and one case report noting it to be successful (Carroll, 1994). Phillips (1996b) notes that in her retrospective chart review none of the eight ECT trials were successful.

8.2.5 Clinical summary

There is a paucity of work undertaken regarding the efficacy of ECT and OCD. Most of the literature comprises single case reports without standardised measures, with one open trial and one retrospective review. Given the serious methodological weaknesses and the lack of convincing evidence for sustained improvement in the above studies, it is concluded that there is insufficient evidence to consider the use of ECT for OCD, especially given potential associated risks with ECT (NICE, 2003).

8.3 Transcranial magnetic stimulation

8.3.1 Introduction

Transcranial magnetic stimulation (TMS) involves the use of a pulsed magnetic field to induce changes in function in cortical structures. It was developed in 1985 (Barker et al, 1985) to investigate cerebral cortical activity and more recently has been used therapeutically for some mental disorders, namely, depression and OCD. In OCD this treatment aims to modify prefrontal cortical activity in order to influence obsessive –compulsive symptoms.

8.3.2 Current practice

Although TMS is not currently available for OCD in the UK, TMS has been used in two trials for depression at the Maudsley Hospital in London.

8.3.3 Studies considered

Three studies, one open trial and two randomised trials were identified. In addition, a recent systematic review (Martin et al, 2003) identified the three papers and concluded that there was insufficient evidence from randomised controlled trials to determine efficacy of this technique.

8.3.4 Descriptive review

The interest in TMS for OCD arose from an open trial of 12 subjects who received repetitive transcranial magnetic stimulation (Greenberg et al, 1997). Each patient received TMS to right or left lateral prefrontal areas or to a control (midoccipital) region. There was a short-lived significant increase in mood and temporary reduction in OCD symptoms with right prefrontal stimulation. Two patients reported headache and one reported visual distortion. The authors concluded that TMS might be a useful probe for studying cortical mechanisms in OCD.

Others have explored the therapeutic potential of TMS. One double blind placebo controlled study has been undertaken for OCD (Alonso et al, 2001) where patients were randomly allocated to either repetitive transcranial magnetic stimulation (rTMS) or to sham rTMS. Ten patients were allocated to the experimental arm and eight to the sham arm. All but five were receiving medication. There were no significant differences post intervention between the two conditions on either OCD (Y-BOCS) or depression (Hamilton) symptoms. One patient reported mild headache. Finally, Sachdev et al (2001) randomised 12 patients with resistant OCD to either right or left prefrontal rTMS. There were no significant differences between right and left rTMS. A significant linear trend from pre-treatment to post-treatment was reported for the two groups combined, indicating improvement in obsessive-compulsive symptoms. However, there was no sham arm so the treatment effect is uncontrolled. An examination of individual response indicated that four of the 12 participants showed a 40% or greater symptom reduction at post intervention, though one had relapsed at 1-month follow-up. Three patients reported headaches.

8.3.5 Clinical summary

The evidence for transcranial magnetic stimulations as a treatment for OCD is as yet inconclusive. Its possible interest lies in the fact that it is a non-invasive procedure.

8.4 Neurosurgery

8.4.1 Introduction and current practice

Neurosurgery for mental disorders has changed significantly since its initial introduction in the 1930s (see Freeman et al, 2000, for a historical overview) and although there remains interest worldwide, the number of centres in the UK offering neurosurgery has decreased over the last ten years. Freeman and colleagues (2000) suggest there is greater specificity as procedures have evolved, but it is less clear whether surgical innovation is due to theoretical advances or to pragmatic considerations. Neurosurgery has been recommended by the Expert Consensus Guideline for treatment refractory OCD in the case of non-response to three or more trials of SRIs (including

clomipramine) and to CBT as an "infrequently needed, but sometimes life saving intervention" (March et al, 1997). Although the number of neurosurgical interventions conducted in the UK for OCD has decreased in recent times (see Freeman et al, 2000), there are currently two centres in the United Kingdom where patients with OCD from England and Wales may be referred for assessment for possible neurosurgical intervention (Matthews & Eljamel, 2003). These centres are in Cardiff and Dundee.

It is important to review the evidence for ablative neurosurgery for OCD given that earlier reviews have reported promising results. For example, Kiloh et al (1988) reported that among 478 patients from 24 studies between 1961 and 1980, 58% showed marked improvement. Over half the operations in this review were non-stereotactically guided. A review of 12 studies from 1961 to 1988 by Waziri (1990) reported that 67% of the 300 patients fell into the "Symptom free" or "Minor Symptoms" categories; all but three studies used stereotactically guided procedures. Finally, Freeman et al (2000) reviewed five studies involving 198 patients, and found an identical result of 67% into these categories.

Although a variety of different procedures are used, all involve the ablation or disconnection, of ventral and medial prefrontal cortical areas. Four main ablative procedures have been used in OCD:

- Anterior capsulotomy
- Anterior cingulotomy
- Subcaudate tractotomy
- Limbic leucotomy

In addition, there are two non-ablative procedures:

- Vagus nerve stimulation
- Deep brain stimulation

8.4.2 Studies considered

Forty-nine studies were identified describing neurosurgery for OCD (including ablative and non-ablative procedures). No randomised control trials were found that compared ablative neurosurgical procedures with a placebo or credible treatment control. One double blind RCT and one systematic review were found for the more recent non-ablative procedures.

8.4.3 Ablative procedures

8.4.3.1 Subcaudate tractotomy

This procedure was developed by Geoffrey Knight in the UK (1969). Although it was used extensively for affective disorders for several years, , the

operation is no longer performed. Radioactive 90-Yttrium rods were inserted into the target area, a region called the substantia innominata, found below the head of the caudate nucleus. Much of the literature on this technique describes the treatment of depression and there are limited reports on its use for OCD (Bartlett & Bridges, 1977; Cosyns et al, 1994; Goktepe et al, 1975; Hodgkiss et al, 1995; Strom-Olsen & Carlisle, 1971). In an early study (Strom-Olsen & Carlisle, 1971), 20 patients with OCD received this procedure; 10 improved, but four relapsed during the follow-up period. In a further study (Goktepe et al, 1975), 50% of 18 patients were reported to have shown significant improvement or better. These early studies report global ratings only rather than specific measures of OCD symptoms. Although this intervention as described above is no longer practiced as such, lesions in the same area of the brain are part of the limbic leucotomy described below.

8.4.3.2 Anterior capsulotomy

Two main procedures for making lesions in the anterior capsule have been described, namely, radiofrequency thermo-capsulotomy and radiosurgical gamma knife capsulotomy (Rasmussen et al, 2000). There are a number of earlier reports between 1961 and 1982 but these used global rather than OCD-specific scales to evaluate thermo-capsulotomy (see Freeman et al, 2000 for a review).

One prospective series from the Karolinska Hospital in Stockholm, Sweden, has been reported on extensively (Lippitz et al, 1997; Lippitz et al, 1999; Mindus et al, 1994; Mindus et al, 1999; Nyman et al, 2001). Twenty-four patients from 1979-1990 underwent thermocapsulotomy and 19 were included in a study on personality characteristics (Mindus et al, 1999). Patients were assessed pre-intervention and a mean of eight years later. Five of the patients received a second intervention during the follow-up period. The authors reported that five patients were unchanged or deteriorated on the CPRS-OC, six were less than 50% improved and eight were more than 50% improved. On 15 self-report scales designed to measure personality (Karolinska Scales of Personality), there was a decrease at eight-year follow-up (average) in anxiety proneness dimensions, but no evidence of changes for other aspects of personality, except one person who had haemorrhaged showed increases in psychopathic traits.

The patients who had received thermocapsulotomy between 1978 and 1999 (n = 21) were compared at follow-up to a group who had been assessed but had not yet received neurosurgery (n = 8) (Nyman et al, 2001). No significant differences were found on a battery of neuropsychological tests; this is unsurprising given the small size of the study. However, the authors conclude "patients with OCD by capsulotomy generally perform in the lower

region of the normal range or show mild impairment on standardized tests 2 to 15 years after the operation" (page 94).

Lippitz et al (1999) reported again on this series but also included those who had received the gamma knife intervention. They conducted a retrospective study to define potential common topographic denominators among the lesions from the patients reported earlier (Mindus et al, 1999) who had been successfully treated with gamma knife capsulotomy in (n=10). The 19 patients receiving thermocapsulotomy reported in this study are the same as those reported in the Mindus et al (1999) study. Magnetic Resonance Imaging indicated the location and size of the lesion. Clinical outcome was reported, mostly using the CPRS-OC although later patients may have been rated on the Y-BOCS. Results are presented as percentage improvement on psychiatric rating scales and showed that 9/19 of the patients who received thermocapsulotomy and 7/10 who received gamma knife capsulotomy showed at least a 50% improvement on either CPRS or Y-BOCS.

Christensen et al (2002) reports on 2 cases of severe OCD in 'younger' (18 yrs) and 'older' (64 yrs) patients and who received capsulotomy. They reported a successful outcome based on a reduction in OCD symptoms on the Y-BOCS. Oliver at al (2003) reported on a series of 15 patients who underwent thermo-capsulotomy. Three patients received a second capsulotomy. The authors report that 46% reported a 50% or greater reduction on the Y-BOCS. Two patients reported transient adverse effects (one case each of hallucinations and seizure) and one "postoperative bifrontal swelling with permanent behaviour impairment". Finally, a review by Greenberg et al (2003) reported preliminary results of an unpublished study of 15 patients who had undergone gamma knife capsulotomy. The authors reported that single bilateral lesions in the anterior capsule were ineffective. However, following placement of a second set of bilateral lesions, four of the 15 were judged as showing a 35% decrease on the Y-BOCS and at least a 15-point improvement on the Global Assessment Scale at 5-year follow-up. They also reported a second study of 16 patients who received two pairs of bilateral lesions. At 3-year follow-up, 10 met the same improvement criteria described in the latter study. These data have not yet been subject to peer review and should be treated cautiously.

In summary, anterior capsulotomy has been reported to be effective based mainly on retrospective trials using global ratings and, more recently, on prospective case series or open trials using OCD specific measures. Although the data are reported in increasing detail on a broader range of measures, conclusions are ultimately limited by the design and the small number of patients involved in the more recent series. The serious persistent adverse

effects (1/19 in the Swedish series and 1/15 in the Oliver study) would suggest caution.

8.4.3.3 Anterior cingulotomy

In an early study, Fodstad et al (1982) randomised two patients to either stereotactic anterior capsulotomy or cingulotomy. Follow-up to 12 to 24 months indicated that all four remained improved or much at the last follow-up. However, those who had received cingulotomy were judged as lesser response compared to those who had received capsulotomy.

A case series of five patients underwent what is labelled a "modified leucotomy" but describe lesions to the cingulated gyrus and so would correspond to cingulotomy (Tippin & Henn, 1982). The authors reported improvement on a five-point clinician-rated global outcome measure, with four out of five classed as marked improvement or symptom free at follow-ups at 1 to 6.6 yrs. No specific measures of OCD were used.

There are two series, one retrospective and one prospective, from Massachusetts General Hospital in Boston that have each been described in several reports. The retrospective series described in most detail by Jenike et al (1991) followed-up 35 patients with OCD who had received cingulotomies from 1962 until the late eighties. It was determined that 33 would have met criteria for OCD according to DSM-III-R; 26 of these also met criteria for depression. Of the 33, 23 had received additional interventions, second cingulotomies in 16 cases, second and third cingulotomies in four cases, and other interventions in six cases. In this early series, only three patients had received behaviour therapy and six had received clomipramine pre-operatively. Adverse effects included seizures (3/33), decreased memory (1/33), suicide (4/33) and death from myocardial infarction six weeks post-operatively (1/33 with previous history of cardiac problems). Based on retrospective ratings of pre-operative symptoms of 14 patients who were interviewed, 8 (58%) showed moderate to marked improvement. However, in at least two cases, improvement was attributed to additional treatment. Overall, the authors conclude that 9/29 surviving patients showed significant improvement that could be attributed to psychosurgery.

Dougherty (2002) reported on a prospective study of 44 patients with treatment refractory OCD who received cingulotomy from 1989 onwards. Data from some of these patients had previously been reported elsewhere (Baer et al, 1995; Spangler et al, 1996). All patients had previously failed treatment regimes of both medication and behaviour therapy. Of the 44 patients operated during the study period, 17 patients had received two cingulotomies and one had undergone a third. Clinical outcome measures,

including the Y-BOCS, were administered pre- and post-intervention (mean of 6.7 months), at the first post-surgical procedure (mean of 7 months) after the second cingulotomy, and at a mean of 32 months follow-up from the first cingulotomy. Response rate was defined as 35% improvement on the Y-BOCS and a Clinical Global Improvement (CGI) score of less than or equal to 2. Patients needed to attribute their improvement to the cingulotomy. At first follow-up, 5/44 patients met the response criteria and 14/44 at final follow-up. Nine patients (9/44) reported adverse effects post operatively: memory (2/44), apathy (1/44), urinary disturbance (3/44), seizure (1/44), postoperative oedema with resulting hydrocephalus (1/44). One patient, who showed improvement in OCD symptoms, committed suicide 6 years after the operation. In all but two cases (1 each of urinary incontinence and seizure), the adverse effects resolved.

Finally, 14 patients with refractory OCD underwent cingulotomy in a prospective study in Korea (Kim et al, 2003). All patients referred for the trial had received behaviour therapy and medication. Patients were assessed using the Y-BOCS, Clinical Global Impression Scale (CGI) and the Hamilton Rating Scales for Depression and Anxiety. The response criteria were defined as at least 35% improvement on the Y-BOCS and a CGI score of 1 or 2 (improved or very much improved). Four patients met this criterion at 6-month follow-up and six at 12-month follow-up. Mean improvement rate on Y-BOCS was 36%. Two patients reported transient headache, three gained weight and 1 lost weight but normalised subsequently. There was no evidence of cognitive dysfunction on a range of measures.

In sum, these studies would suggest that cingulotomy has shown some modest effects with up to a third of patients responding. The two prospective studies are stronger designs than those found for some other interventions but remain limited by the lack of control groups. There were relatively few persistent adverse effects but the suicide rate in the retrospective study (Jenike et al, 1991) would indicate caution.

8.4.3.4 Limbic leucotomy

Limbic leucotomy, a multi-target procedure, was developed in the UK by Kelly in the 1970's and consists of lesions corresponding to both cingulotomy and subcaudate tractotomy. An early study reported a high response rate with OCD (Mitchell-Heggs et al, 1976) but was methodologically flawed and the results have been disputed (Chiocca & Matuza, 1990).

A number of reports describe an Australian series of 26 patients operated on from 1972 until 1989, most of whom (n = 17) received both cingulate and orbitomedial lesions similar to limbic leucotomy while six received cingulate

lesions and four received orbitomedial lesions only (Cumming et al, 1995; Hay et al, 1993; Sachdev & Hay, 1995; Sachdev, 1996). The first six patients received the orbitomedial lesions through open neurosurgery; the remaining all received stereotactic interventions. Although this series is more difficult to interpret because of the mixed interventions, it is worth considering because of the extensive reports on cognitive and personality function. At follow-up of 10 years (mean), five (19%) were considered much improved or recovered, five were moderately improved, six were mildly improved, six showed no improvement, four were worse (Hay et al, 1993). Of the four who were rated worse, three were dead to suicide and one because of marked personality change. In addition to transient post-operative adverse effects in three patients, one suffered from post-operative haemorrhage, delirium, periods of psychosis and permanent personality change. An additional three were considered to have shown personality change and two suffered from recurrent seizures. In all, 6/26 suffered serious permanent adverse effects. In a report on self- and informant-rated personality change in a sub-group of 16, the majority reported little change in personality across 34 items (Sachdev & Hay, 1995).

Finally, 17 of this series were compared to a control group of patients with long term OCD on a battery of neuropsychological tests. There was no evidence of impaired IQ or memory function compared to the OCD controls, but there was evidence of impaired performance in the operated group on executive function (Cumming et al, 1995). Interestingly, an MRI study on 14 of these patients revealed accurately placed orbitomedial lesions (10/10), accurately placed cingulated lesions in only 10 of 13, and inadvertent lesions to the anterior capsule in 3 cases (Sachdev, 1996).

A more recent case series has been reported with 21 patients with major depressive disorder (MDD) or OCD who underwent stereotactic limbic leucotomy between 1993-1999 (Montoya et al, 2002). All had failed to respond to pharmacotherapy and over three quarters had received ECT.

In this study, there were 15 patients for whom the primary indication for surgery was OCD, although three of those whose primary indication was for MDD also had OCD. Eight of those in the OCD group also received a diagnosis of MDD. For a significant proportion of the participants (76%), limbic leucotomy was the second (following unsuccessful bilateral anterior cingulotomy, n =5) or third neurosurgical procedure (following enlargement of earlier cingulotomy lesions). It is unclear what percentage of patients with OCD had undergone a second or third operation as the authors have reported for the whole sample only.

Two patients, one with MDD and one with OCD, both with previous history of suicidal attempts, committed suicide during the follow-up period. Transient somnolence (6/21), apathy (5/21), and fever (2/21) were reported. Post-operative and persistent headache were reported by one patient. Four patients reported postoperative seizures that were transient in all but one. Five patients (5/21) reported bladder incontinence that was persistent in three (3/21). Finally, five patients (24%) reported short-term memory problems that were persistent for two people (10%).

Outcome measures used were the Y-BOCS and Clinician Global Improvement Scale (CGI). Response was determined in a similar way to other neurosurgical studies of OCD, namely, an improvement score of 1 or 2 on the CGI (very much or much improved) and a 35% decrease in Y-BOCS score. Of the 15 patients with OCD, only nine had pre- and post-operative ratings on the Y-BOCS and only eight had both Y-BOCS and CGI ratings. Of these eight patients, only two (25%) met the double criteria for response. When based on one or other of the measures the authors report a 36% (Y-BOCS) to 62% (self-rated CGI) response. The authors also report that for the entire cohort (N=21), the response rate was better for those undergoing limbic leucotomy where it was the second or third procedure. In sum, this case series describes a group of chronically ill patients who had received multiple previous non-neurosurgical treatments and indeed 76% had also received one or more previous cingulotomies. The findings are limited by the partial data collected and the difficulty in separating out some of the findings for the OCD patients alone.

Given the shortcomings in the designs, the lack of robust evidence of efficacy and the high rate of persistent adverse events reported in both series, the evidence for this type of multi-target procedure is not promising.

8.4.3.5 Neurosurgery in BDD

Phillips (2002) notes one published case report and 2 personal communications describing benefit in 3 individuals with BDD (modified leucotomy in one, capsulotomy in one, and bilateral anterior cingulotomy and subcaudate tractotomy in one) and no benefit in 2 individuals (who received anterior capsulotomy).

8.4.3.6 Clinical summary

Although there are reports of modest improvement for three of the procedures (capsulotomy, cingulotomy and limbic leucotomy), there are also sufficient reports of both transient and persistent adverse effects to cause concern. The more recent prospective series all report on criteria for entry to these studies that include previous trials of pharmacological treatments and

exposure and response prevention, supporting the contention that at least in the last 15 years, these are generally treatments of last resort. Given the relative rarity of these interventions, studies are generally small and conducted over long periods. Importantly, none have control conditions, although it is unlikely that credible sham procedures could be ethical for ablative procedures, nor are they likely to be possible as patients are unlikely to accept randomisation for treatments of last resort.

Some studies have used comparison groups to compare neuropsychological function and personality function in people with OCD that have received neurosurgery to those who have not. While these studies are attempting to answer important questions about effects of neurosurgery, the small sample size and the fact that they are essentially testing null hypotheses means that the most that can be concluded is the absence of large effects, but nothing can be said about whether smaller but potentially clinically significant differences may be present. Although there have been significant improvements in measuring obsessive-compulsive symptoms, there are still few prospective studies investigating neuropsychological changes and those measuring personality changes are using measures that are not widely known. In summary, the evidence is inconclusive at best.

8.4.4 Non-ablative procedures

Concerns regarding the irreversibility and possible long term adverse effects of ablative neurosurgical procedures have led to the investigation of a number of non-destructive neurophysiological interventions. Tissue damage could still occur during the intervention or through repeated stimulation, but the intervention does not seek to produce lesions.

8.4.4.1 Vagus nerve stimulation

Vagus nerve stimulation (VNS) was developed for the control of epilepsy that cannot be managed by normal medical treatment. It involves the electrical stimulation of the vagus nerve within the neck by an electrode connected to a programmable stimulator. VNS is available in the UK. Early reports on the use of VNS in the treatment of depression are said to be encouraging (George, 2000) and although it has been discussed as a treatment for OCD, there is no evidence as yet for its efficacy and it will not be considered further in this review.

8.4.4.2 Deep brain stimulation

Electrical deep brain stimulation (DBS) is a relatively new technique and developed as a treatment for OCD through collaboration between Belgian and Swedish researchers. The effects of electrical stimulation of the brain have

been previously investigated during stereotactic surgery for OCD before permanent lesions were made. For example, Laitinen and Singounas (1988) report on the effects of stimulation in a series of 20 patients undergoing neurosurgery under local anaesthetic. However, the therapeutic use differs in that no lesions are made and electrodes are implanted within brain structures, which are then stimulated via an external electrical source. The efficacy and safety of DBS for the treatment of neurological conditions such as Parkinson's disease are well established. It has not yet been used for OCD in the UK although it is used for Parkinson's disease.

Essentially, DBS uses high frequency pulses that have a blocking effect of the targeted area and mimics the effect of tissue lesioning without destroying them (Tass et al, 2003). In an initial report, Nuttin et al (1999) described on 4 patients who were treated with DBS; three were reported to have improved although little detail was provided. Since then, a series of six patients has been reported in several publications that address a range of variables as well as issues about placement of electrodes and stimulation parameters (Gabriels et al, 2003; Nuttin et al, 2003a; Nuttin et al, 2003b). In this series, quadripolar electrodes stereotactically implanted in both anterior limbs of the internal capsules in six patients with OCD, all of whom had been deemed to have severe OCD by a selection committee. Four of the patients were randomly crossed over from continuous stimulation to stimulation off. Two were not crossed over (one received a capsulotomy and one was still in the post-operative screening phase).

The authors report improvement in the "stimulator on" condition compared to "stimulator off", but these data should be interpreted with caution due to the small size of the study. Of the four patients, one did not respond and 3 responded (improvement of >35% on the Y-BOCS). A number of side effects were reported including transient hypomanic states, swelling of the face, awareness of the leads, fatigue, and weight loss/gain (Nuttin et al, 2003b). There were also technical difficulties due to broken electrical contacts and fracture of an electrode, but the main technical issue was short battery life necessitating replacement of the stimulators every five to twelve months. There is one additional independent report of DBS (Anderson & Ahmed, 2003) that reported a positive outcome in a case of severe OCD.

8.4.4.3 Clinical summary

DBS is a very recent procedure. The studies so far are too small to reach any conclusions about efficacy. The side effects reported so far suggest extreme vigilance especially as it is not yet established whether DBS is completely reversible. The technical issues around battery life would seem to present a significant limitation at the moment. The non-destructive nature of the intervention means that well designed controlled trials would be possible.

8.4.5 Issues about neurosurgery for OCD

All of the recent series report selection criteria and despite agreement on general principles such as severity, disability and non-response to previous treatment, there is a degree of variability. There is also wide variability in terms of the range of assessments used although all recent studies have used the Y-BOCS. Consequently, combining or comparing data from different centres is almost impossible and centres with few operations do not contribute at all to these series. As several authors have pointed out, international agreement on these issues would allow the field to advance.

The main issues to be agreed, both in routine practice and research, are selection criteria that reflect severity and chronicity of OCD, the definition of adequate previous treatment, a standardised assessment protocol pre- and post-operatively, and agreements and protocols about post-operative care.

Matthews and Eljamel (2003) have outlined a series of criteria both for inclusion and for determining adequacy of previous treatment. These criteria are broadly in line with, and generally exceed those reported by authors in Sweden, the US and Korea. However, as Bejerot (2003) points out, "Yesterday's intractable OCD patient may well be treatable today and intractability may depend on the ambition of the prescriber" (p. 242). Likewise, for psychological treatments, an adequate trial cannot be defined by number, duration and frequency of sessions alone. Engagement in therapy is believed to be essential to outcome and therapeutic strategies must be implemented. Unwillingness or inability to engage actively in therapy is a feature of some people with severe OCD.

Sometimes several unsuccessful attempts to engage in therapy precede a successful attempt, perhaps with a therapist of equal skill who has a different style, or who is using the same basic techniques in a different manner. Consequently, guidelines such as those by Matthews and Eljamel (2003) must remain guidelines. It is essential that multi-disciplinary teams with specific expertise in the management and treatment of OCD be involved in treatment and assessment before concluding that a patient has not responded to adequate treatment. In addition, given that non-ablative methods are now being developed, it may be that non-ablative methods should be considered first.

Assessment should include standardised measures of obsessive-compulsive symptoms, depression and anxiety, psychosocial functioning including quality of life, personality, cognitive function, and possible adverse effects. Ratings should be sought from independent assessors, the patient, and family members or friends. If it is decided to proceed with an intervention, protocols for post-operative care should be agreed that allow provision for pharmacological and psychological care.

8.4.6 Clinical summary

The evidence is generally inconclusive for all of the medical interventions considered. Besides a variety of design issues, evidence of efficacy is scarce and at best response rates are modest. Neurosurgical techniques all have the potential for adverse effects and most studies report some persistent adverse effects. However it is important to note that especially in the more recent studies, many of the patients, though not all, have severe OCD with high Y-BOCS scores, poor functioning, and significant levels of comorbidity, including, in many cases, severe depression. In addition, most have previously received evidence-based treatments and selection committees have approved surgery. For such patients refractory to other treatment approaches, the modest response reported may be clinically important.

At present, there is no compelling evidence comparing different neurosurgical procedures. It may be that by using MRI scanning pre-operatively, more accurate and perhaps smaller lesions can be placed, perhaps reducing the risks of some types of adverse effects. However, several studies have suggested multiple lesions may be needed for both capsulotomy and cingulotomy. Small samples have prevented identification of predictors of response to any of the treatments. The newer non-ablative and non-invasive interventions may yet prove to be of value. Overall, evidence is either inconclusive or insufficient and so prevents recommendations being made for any of the procedures. However, it is likely that neurosurgical methods will continue to be considered for a small number of patients who have failed to respond to adequate treatment.

8.5 Medical interventions in children with OCD due to PANDAS

8.5.1 Introduction

There is accumulating research evidence that OCD may arise following infection with particular subtypes of streptococcal bacteria, Group A beta haemolytic streptococcus (GAS). This hypothesis and the subsequent studies, were stimulated by the longstanding observation that patients with Sydenham's chorea (SC) had high rates of obsessive compulsive symptoms. SC is the neuropsychiatric manifestation of rheumatic fever, a disorder now known to be triggered by GAS. A sub-group of children with OCD were identified who had developed their condition following GAS infection, but did not meet criteria for SC. These children were given the acronym PANDAS (paediatric autoimmune disorders associated with streptococcal infection). The most striking feature of this sub-group of OCD is that the onset is very rapid, following streptococcal infection, and remits fully. Relapses occur with recurrent infection, giving these children an unusual fluctuating course to their OCD episodes.

The proposed mechanism of this disorder is one of 'molecular mimicry'. Antibodies generated by the body as part of the immune response against streptococcal infection, cross react with binding sites in the basal ganglia, a brain region thought to be important in OCD and related movement disorders. This autoimmune reaction only occurs in susceptible individuals, perhaps those with a genetic predisposition.

This proposed aetiology has suggested possibilities for novel prevention/treatment options in this subgroup. Could prevention of recurrent streptococcal infections prevent recurrences of OCD symptoms? Prophylactic antibiotics are given to individuals with rheumatic heart disease; might a similar approach also be effective in a post-streptococcal neuropsychiatric condition? A second approach that has been explored is to modify the immune response which is thought to be the direct pathogenic mechanism. Antibody production could be inhibited by immunosupression, or antibodies removed by 'mopping-up' with another binding protein, or removing them with plasmaphoresis.

There have been limited trials of these approaches, and these are summarised below.

8.5.2 Current practice

PANDAS is difficult to diagnose, and a parental report of a sore throat preceding OCD, or a fluctuating history of OCD symptoms, is not adequate to make a diagnosis. The original definition of PANDAS described a homogenous group of children for the purpose of research into mechanisms, phenomenology and treatment. To diagnose PANDAS according to the criteria of the original investigators, streptococcal infection must be demonstrated in conjunction with at least two episodes of OCD symptoms, as well as demonstrating absence of infection and reduced antibodies during neuropsychiatric remission.

The clinical importance of a post-streptococcal subtype of OCD remains controversial. We do not know how common this sub-type of OCD might be, as there are no epidemiological studies in the general population. Children attending specialist psychiatric/OCD clinics may have higher than average evidence of previous streptococcal infections. However, even in the sub-group of children who are clearly thought to have PANDAS, there is no strong evidence to support immunomodulation or antibiotic prophylaxis currently, although these interventions remain the subject of active research. Similarly, there is no evidence to suggest that the post-streptococcal forms of OCD respond differently to medication or psychological treatments, as there have been no direct comparative studies of different OCD-subtypes.

Currently, there is no straightforward diagnostic system for determining whether an OCD patient meets PANDAS criteria, other than longitudinal follow-up, combined with repeat throat swabs and blood antibody tests. Even if there is a strong suggestion that they do fall into the post-streptococcal group, there is too little evidence currently to be recommending novel treatments, other than as part of a research program or clinical trial. In addition, children falling into this group should be given full trials of the conventional OCD treatments known to be effective, i.e. cognitive behaviour therapy and SSRI medication.

8.5.3 Studies considered

Two double-blind randomised controlled trials were identified that tested whether a medical intervention targeting GAS infection would be effective in reducing OCD symptoms (Garvey et al, 1999; Perlmutter et al, 1999).

8.5.4 Descriptive review

Garvey et al (1999) tested whether penicillin prophylaxis would reduce neuropsychiatric exacerbation in children with PANDAS by preventing streptococcal infections. The study was an 8-month double-blind cross-over trial, with 4 months in penicillin and 4 months in placebo. Children with a DSM-III or IV diagnosis for tic disorder and/or OCD, a history of a sudden onset of symptoms or an episodic course with abrupt symptom exacerbations interspersed with periods of partial or complete remission, and who showed evidence of an association between streptococcal infection and the onset or exacerbation of symptoms were included in the study. Participants received a standard prophylactic dose of 250 mg twice daily penicillin V. Thirty-seven children entered the study, of whom 35% had both a primary diagnosis of OCD and tics and 35% had tics and subclinical OCD. Overall, there was no difference between the two groups on OCD and tic symptoms as measured by the Y-BOCS and the Yale Global Tics Severity Scale. There was also no difference between groups in the incidence of streptococcal infections, though fewer infections occurred in the penicillin than placebo phase. There was thus a failure to even achieve an adequate level of prophylaxis.

Perlmutter et al (1999) tested whether plasma exchange and intravenous immunoglobulin (IVIG) would be better than placebo in decreasing neuropsychiatric symptoms in children with PANDAS. Children meeting similar inclusion and exclusion criteria to those in the Garvey et al study were recruited to the study. Children were randomly assigned to plasma exchange, IVIG, or placebo. The plasma exchange procedure was done over 10-12 days, while the IVIG and placebo procedures were done over 2 days. Treatment outcome was assessed at 1 month and 1 year after start of therapy. Thirty children entered the study of which 63.33% had a primary diagnosis of OCD and 33.33% had a primary diagnosis of tic disorder. At one-month

follow-up, there was a significant improvement in symptom severity from baseline as measured by the Y-BOCS in the plasma exchange and IVIG groups. In turn, the plasma exchange group appeared to have greater improvement in OCD symptoms than the IVIG group, though this was not statistically significant. This improvement in OCD symptoms remained in the 17 children in the plasma exchange and IVIG groups who were followed-up at 1 year.

8.5.5 Clinical summary

Describing PANDAS as an autoimmune disorder rather than as a streptococcal infection has more benefit in terms of therapeutic gain. Treatments that target the autoimmune feature of PANDAS, such as plasma exchange and intravenous immunoglobulin, seem to improve OCD symptoms. Penicillin prophylaxis which aims to reduce streptococcal infection, however, does not seem to be effective in preventing OCD symptom exacerbations. Further research is needed to determine which children with OCD will benefit from immunomodulatory therapies.

8.6 Clinical practice recommendations

Neurosurgery is not recommended as a treatment for OCD, although it is recognized that some people may wish to consider this option when all else has failed.

8.6.1.1 If neurosurgery were to be considered for severe refractory OCD among adults, the following should be taken into consideration:

- Existing criteria (e.g. Matthews and Elmajel, 2003) should be used to guide decisions about suitability
- Multidisciplinary teams with a high degree of expertise in the pharmacological and psychological treatment of OCD should have been recently involved in the patient's care; all pharmacological options should have been considered and every attempt should have been made to engage the individual in CBT and CT, including very intensive and/or inpatient treatments
- Standardized assessment protocols should be used at pre- and post-operation and follow-up in order to audit the interventions. These assessment protocols should include standardized measures of symptoms, quality of life, and personality as well as comprehensive neuropsychological tests.
- Post-operative care should be carefully considered, including pharmacological and psychological therapies. [GPP]

9 Use of health service resources

9.1 Methods of economic evaluation

Methods of economic evaluation command a fairly high level of consensus and are reported in Drummond and colleagues (1997). However, where economic evaluations have been undertaken for anxiety disorders generally (see Issakidis et al, 2004 for example), costing data for direct and indirect costs attributable to OCD are scarce[15]. Part of the issue is that OCD is often a chronic, fluctuating condition, although for some it make take a relapsing and remitting course, where the results of treatment may be modest. As OCD often starts in and continues beyond childhood and adolescence and may increase in severity over time, its economic burden accrues in the total direct and indirect costs that accumulate with time. In attempting to quantify this burden, few studies have met rigorous criteria for health economic appraisal.

9.2 Use of health service resources

Using the human capital approach to health economic appraisal, DuPont and Colleagues (1995) estimated the direct and indirect costs of OCD to be $8,400 million in 1990 USD prices, 6% of their estimated $147,800 million cost of all mental illness in the United States. The same study estimated the indirect cost component of OCD, due to lost productivity, which amounted to $6,200 million, or 74% of the total estimated cost cited above (*Ibid*.).

9.3 Primary care drug therapy vs. secondary care CBT and CCBT

A number of medications are routinely administered in a primary care setting for the treatment of OCD. Traditionally, the most expensive of these has been the SSRIs. However, now that most SSRIs are comparably priced, it is less important to perform cost-effectiveness evaluations between alternative drugs in comparison to the need to perform these evaluations with alternative treatment modalities such as drug therapy vs. CBT vs. computerised cognitive behavioural therapy (CCBT) and the monotherapies vs. combined drug with

[15] The database searches for general health economic evidence for OCD resulted in a total of 41 references. Of these, nine were identified as potentially relevant. Secondary searches for relevant pharmacoeconomic papers resulted in a further eight references, of which, three were initially considered relevant to accepted criteria for health economic appraisal (as reported by Drummond et al, 1997). A further four potentially eligible references were found by handsearching. Full texts of all potentially eligible studies (including those where relevance/eligibility was not clear from the abstract) were obtained, a total of 16 papers. At this stage inclusion was not limited to papers only from the UK.

cognitive therapy. To accomplish this, one needs to know both the cost and effectiveness of each treatment.
A one-year course of the SSRI paroxetine (20mg tablet in generic form) would cost an estimated £289, including direct and indirect GP prescribing and follow-up costs,[16] which is +/- £10 in comparison to other generic drugs.

The efficacies of drug and clinician-guided CBT are identical, with an estimated 50% of patients recovering in the first 12 months of treatment (GDG Expert Opinions 2004). The efficacy of combined drug and clinician-guided CBT is estimated to be slightly higher than these therapies alone, with 60% recovering in this timeframe; 10% more than in the case of the component monotherapies. No assumptions have been made for this analysis about ongoing treatment beyond one year for any of the options, although continuation of SSRIs is often recommended.

In comparison to drugs, treatment costs are known to be higher with clinician-guided CBT because this therapy is labour intensive and requires specialist knowledge for optimal delivery. Therefore, 16 one-hour sessions of CBT delivered by a clinical psychologist will cost an estimated £1,056[17], or £767 more than the above SSRI treatment scenario. This assumes a slightly more conservative delivery regimen than the one reported in Greist and Colleagues (2002).

By contrast, the efficacy of CCBT is estimated to be 30%, or half the efficacy of the combined therapies (e.g. 60%). The cost of administering CCBT is currently estimated to be quite similar to the cost of clinician-guided CBT. A cost of £1,096 per patient receiving a full course of treatment was calculated based upon an estimated OCD prevalence of 2% and presenting prevalence of 0.05% (De Waal et al, 2004); a baseline cost of £1,000 per client for the treatment package; and a counsellor on duty to administer assisted self-help at a rate of three hours per client, costing an additional £32 per hour (de Waal et al., 2004; Netten and Curtis, 2003). In brief, there are variations in both cost and efficacy between alternative therapies and their combinations. To illustrate these differences, the cost of treatment is compared to alternative recovery rates in the following table:

[16] £180 for the direct drug costs (West Midlands Medicines Information Service, 2003) plus £31 for first prescribing session (Netten & Curtis, 2003), plus 3 x £26 for clinical consultations (Ibid), including indirect and qualification costs = £289.
[17] £66 (per hour of client contact, Netten & Curtis, 2003) * 16 = £1,056.

9.4 Table 1: Relative cost-effectiveness of therapies

Per Patient per year	Drug Therapy [18] (generic SSRI, 12-month course)	Clinician-guided CBT	CCBT (BT STEPS)	Combined Drug Therapy and Clinician-guided CBT	References
Rate of recovery (%)	**50**	**50**	**30**	**60**	**Drugs[19], CBT[20], CCBT[21,22], Combined[23]**
Cost (£) per patient *treated*	**289**	**1,056**	**1,096**	**1,345**	**Drugs[24], CBT[25], CCBT[26], Combined[27]**
Cost per treatment-related recovery [28] (£)	**578**	**2,112**	**3,653**	**2,242**	**Standard methodology of health economic evaluation cf. Drummond and Colleagues (1997)**

Table 1 illustrates the rates of recovery and costs of treatment per year. The differences in cost, effectiveness and cost-effectiveness ratios are presented between therapies. The cost per treatment-related recovery reflects the

Footnotes to Table 1:
[18] 12-month course of the SSRI paroxetine, calculations as cited previously.
[19] Expert opinions (GDG)
[20] Expert opinions (GDG)
[21] Greist *et al* (2002)
[22] De Waal *et al.* (2004)
[23] Expert opinions (GDG)
[24] West Midlands Medicines Information Service (2003)
[25] Netten & Curtis (2003)
[26] Expert opinions (GDG) adapted from de Waal et al (2004): Calculated at £1,000 per patient baseline plus 3 hrs of counsellor time, with oncosts and training included, over 12 weeks at £32 per hour (Netten & Curtis, 2003) = £96+£1,000 = £1,096.
[27] Sum of drug therapy and clinician-guided CBT
[28] Calculated thus: (cost per patient treated)/(improvement in effectiveness) * 100 = cost to achieve at least 1 treatment-related recovery.

number of treatments needed to achieve a single recovery, according to the type of therapy or combined therapies. The rates of recovery for drug therapy, clinician-guided CBT, BT STEPS/CCBT and combined drug and CBT therapy are: 50%, 50%, 30% and 60%, respectively. CBT is comparably priced with CCBT; however, each of these therapies is more than threefold the cost of SSRI therapy. The combined drug and CBT therapy is slightly higher in cost compared to the CBT and CCBT monotherapies.

The cost-effectiveness, meaning the cost per treatment-related recovery, is £578 in the case of SSRI therapy, £2,112 in the case of clinician-guided CBT, £3,653 in the case of BT STEPS/CCBT, and £2,242 in the case of combined SSRI therapy and clinician-guided CBT. Therefore, based on the initial assumptions of cost and effectiveness, the SSRI therapy is six-times more cost-effective than CCBT, and four-times more cost-effective than either clinician-guided CB T or combined SSRI and CBT therapies.

It is evident, moreover, that CBT alone or combined with SSRI therapy have comparable cost-effectiveness ratios of around £2,000 per treatment-related recovery. The cost of BT STEPS/CCBT is higher, and this may be reflective of the comparatively lower efficacy rate for and/or the low take-up rate by patients.

The cost-effectiveness estimation for CCBT is not so sensitive to changes in the hours of counsellor care in comparison to the baseline cost of treatment and overall lower effectiveness in relation to the other monotherapies. Whereas 0.5-2% of the background population may have OCD, only 25% of sufferers actually present to a GP and fewer still continue through the full course of treatment when such is made available. However, it is important to note that all of these cost-effectiveness ratios are well within the generally acceptable ranges as cited by NICE, for they are all less than £5,000 per treatment-related recovery. Since relative cost-effectiveness ratios may change with time, more studies are needed to address the long-term trends of treatments and their costs.

9.5 Treatment vs. non-treatment

In summary, the additional cost of administering cost-intensive treatments needs to be weighed against the reduction of work-related absences that may be much more costly than any of the treatments. For instance, a survey of an OCD consumer advocacy group estimated that, on average, a person with OCD loses fully 3 years of wages over their lifetime (Hollander & Wong, 1995). If an OCD sufferer incurs losses of £483.04 for every week they are absent (Income data services, 2004), this would amount to a total of £75,354 due to unemployment over this 3-year period, not to mention lost opportunities for career advancement and the cost to families and carers over

their respective working lifetimes. If a single year of unemployment were saved, it would fund a number of treatments. Hence the following contention:

Even assuming only 20% of the patients treated reach full recovery following one year of CBT, then from a societal perspective, the prevention of a person's work absences in one year alone could potentially fund the most cost-intensive treatments (CCBT) for 7 patients. Prevention of a person's one-year work absences could fund combined CBT and drug therapies for 11 patients, therapist-led CBT for 25 patients, or one year of SSRI treatment for 87 patients. A recovery rate of 20% is two to three-times lower than actual estimations so extrapolations based on this assumption are highly conservative, reflecting the pessimistic rather than nominal or optimistic scenarios.

There are more than direct costs to hospitals and carers, also. Since work absences for family and carers are prevented over these individuals' employable lifetimes, savings should increase indirectly and exponentially. Sensitivity analysis shows that prevention of work absences far outweighs any treatment costs, even when +/- 10% fluctuations are introduced to component cost and efficacy estimations. Therefore, the estimations are believed to be robust.

It has been the impression that CCBT, particularly the internet-based treatments, may cost a very nominal amount and yet prove as effective as therapist-led CBT or usual GP care, if delivered to suitable individuals (NICE, 2002a). There are currently a number of types of CCBT treatments for anxiety and depression, each with varying costs. However, it has been assumed that for OCD, CCBT is not at present as efficacious as therapist-guided CBT, and half as efficacious as combined SSRI and CBT. CCBT is the most cost-intensive (e.g. least cost-effective) option for treating OCD, because of its high current cost and proportionately lower efficacy in comparison to the other therapies. More evidence is needed to assess the components that drive the cost-effectiveness ratio of CCBT to be more than six-times the cost of drug therapies. It is recognised that for select groups of patients CCBT may be cost-effective, yet only to the point that healthcare providers are willing to pay an amount which is higher than the cost of alternative therapies. As the delivery structure of CCBT changes, so will the cost-effectiveness ratio.

The economic evidence clearly shows that even if only a small fraction of the number of treated patients fully recover, the amount of money saved in preventing their work-related absences will offset any potential deficit in delivering comprehensive treatments, such as drug therapy, CBT and CCBT. Lost work time of people with OCD and carers is the more important costing

component and this should be minimised through whatever pharmacological or psychological treatments are necessary.[29]

9.6 Non-healthcare burden

OCD presents a considerable economic burden to the individual, family, health services, and society as a whole. The total cost accrued as a result of OCD-related illness is difficult to measure, because it extends beyond the primary, secondary, and tertiary care settings. Often people with OCD accrue costs attributed to work-related absences, and beyond the immediate family. These latter costs may arise from the affected individuals as well as from family and friends who care for them. It is therefore the long-term, societal cost that is ultimately the more important variable.

9.7 Conclusions and future recommendations

There is a need for additional efficacy data and reliable cost estimates of treatments that can be administered before people with OCD accrue long terms costs. In particular, follow-up studies are required that report on continued or repeat courses of the same treatment, or the addition of alternative treatments that may be required to keep people who have responded to initial treatment well in the longer term. Studies should report direct treatment costs alongside indirect costs that accrue from OCD and refine these estimates in relation to the number of years lost compared to life expectation, as well as the number of years of healthy life lost compared to severity and duration of the condition, with provisions for uncertainty (Murray & Lopez, 1997). The sum of disability-adjusted life years (DALYs) would yield a more comprehensive estimation of the economic burden of OCD for the UK population.

Since OCD ranks tenth in the World Bank and World Health Organisation's leading causes of disability (World Health Organization, 2001), its overall costs are important. The economic burden related to OCD comprises not only direct medical costs but also costs due to premature death, unemployment and reduced productivity if the condition is left untreated. By treating OCD as soon as it is identified and through cost efficient means, it is likely to be more cost-effective than treating a more severe presentation downstream when patients may experience a worsening of symptoms. The long-term costs associated with OCD strengthen the impression that early and effective

[29] The above estimates are for illustrative purposes only. It is at present unclear how many OCD sufferers will be cured by each of these treatments. When certain medications or treatments are withdrawn or replaced, it is essential that the alternative costs and effectiveness for each possibility be estimated. Such estimates will continuously need to be refined and rigorous cost-effectiveness analyses, undertaken.

interventions would be cost-effective. Efficient service utilisation based upon rigorous health economic evaluations of OCD would reduce the social and economic burden of this condition, which would ensure optimal healthcare is delivered within the constraints of the national budget.

ABBREVIATIONS

A&E	Accident and emergency
AGREE	Appraisal of Guidelines Research and Evaluation
APA	American Psychiatric Association
BAI	Beck Anxiety Inventory
BDD	Body Dysmorphic Disorder
BDI	Beck Depression Inventory
BNF	British National Formulary
BPS	British Psychological Society
BT	Behaviour Therapy
CAMHS	Child and adolescent mental health services
CAT	Cognitive analytic therapy
CBT	Cognitive behaviour therapy
CEBMH	Centre for Evidence Based Mental Health
CEFAHP	Clinical Effectiveness Forum for the Allied Health Professions
CEMH	Centre for Economics in Mental Health
CI	Confidence interval
CMR	Crude mortality rate
CORE	Centre for Outcomes Research and Effectiveness
COT	College of Occupational Therapists
CPRS	Comprehensive Psychiatric Rating Scale
CRU	College Research Unit, Royal College of Psychiatrists
CT	Cognitive Therapy
CY-BOCS	Children's Yale-Brown Obsessive-Compulsive Scale
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, Fourth edition
ECT	Electro-Convulsive Therapy
EEG	Electroencephalography
EMDR	Eye movement desensitisation and reprocessing
ERP	Exposure with response prevention
GDG	Guideline development group
GP	General practitioner
GPP	Good practice point
GSH	Guided self help
HADS	Hamilton Anxiety and Depression Scales
ICD10	International Classification of Diseases, 10th Edition
IoP	Institute of Psychiatry
IPT	Interpersonal psychotherapy
LOI	Leyton Obsessive Inventory
MAOI	Monoamine-oxidase inhibitors
MOCI	Maudsley Obsessive-Compulsive Inventory
n	Number of participants
N	Number of studies
NCCMH	National Collaborating Centre for Mental Health
NHS	National Health Service

NICAPS	National Inpatient Child and Adolescent Psychiatry Study
NICE	National Institute for Clinical Excellence
NNT	Number needed to treat
OA	OCD Action
OCD	Obsessive Compulsive Disorder
OCI	Obsessive-Compulsive Inventory
OR	Odds ratio
PANDAS	Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcus
PCTs	Primary Care Trusts
PSH	Pure self help
QOL	Quality of life
RCGP	Royal College of General Practitioners
RCN	Royal College of Nursing
RCPsych	Royal College of Psychiatrists
RCT	Randomised controlled trials
RET	Rational Emotive Therapy
RPS	Royal Pharmaceutical Society
RR	Relative risk, risk ratio
SCID	Structured Clinical Interview for DSM-IV
SCIE	Social Care Institute of Excellence
SMD	Standard mean difference
SMR	Standardised Mortality Rate
SNRI	Serotonin and noradrenalin reuptake inhibitors
SRI	Serotonin reuptake inhibitor
SSRI	Selective serotonin reuptake inhibitors
STAI	State-Trait Anxiety Inventory
TS	Tourette's Syndrome
Y-BOCS	Yale-Brown Obsessive-Compulsive Scale
Y-BOCS mod BDD	Yale-Brown Obsessive-Compulsive Scale modified for BDD
WHO	World Health Organization

Glossary of Terms

Ablative neurosurgery: Surgery in which parts of the brain are disconnected from one another.

Adherence: The behaviour of taking medicine according to treatment dosage and schedule as intended by the prescriber. In this guideline, the term 'adherence' is used in preference to 'compliance', but is not synonymous with 'concordance', which has a number of meanings.

Adverse event: any untoward medical occurrence in a patient who was given a pharmaceutical product, and which does not necessarily have a causal relationship with this treatment.

Anxiety management training (AMT): A psychological approach aimed at teaching people a set of skills to help them manage their own anxiety and stress. These include:

- Relaxation training: teaching techniques for relaxing major muscle groups in a way hat decreases anxiety.
- Breathing retraining: teaching techniques of slow, abdominal breathing to avoid hyperventilation and the unpleasant physical sensations that accompany it.
- Positive thinking and self-talk: positive statements (e.g. "I did it before and I can do it again") are written on cards and rehearsed so that they can be used to replace the negative thoughts that often occur during stressful experiences).
- Assertiveness training: teaching the person how to express wishes, opinions, and emotions appropriately and without alienating other.

Anxiolytic medication: A drug used to alleviate anxiety states.

Anti-exposure: A comparator or control (placebo) treatment used in a few studies. It involves asking the patient to avoid anxiety by avoiding contact with anything that led to compulsive behaviour, thereby leading to a reduction in discomfort and urge to ritualise.

Antipsychotic medication: This group of medicines all act on a brain chemical, dopamine. Their main use is in psychotic illness, but their dopamine blocking properties can help, when used together with other medicines, in some people with OCD, especially those who do not respond to standard treatments

Augmentation: The addition of more than one potentially effective treatment together with the aim of enhancing the benefits.

Avoidance: People with OCD and BDD may be unable to engage in particular behaviours, go to specific places or interact with certain people related to their main fears and preoccupations. Even if they wished to do these things, they

may find this impossible to do so because of the distress that would arise or through fear of unacceptably dangerous consequences. In OCD, people tend to avoid situations and objects as they can potentially trigger obessional thoughts and compulsions. The individual sees such situations as risky and knows that they lead to a high level of anxiety and tension. For example, people with obsessions about germs and cleaning compulsions usually strive to avoid objects and situations that they believe to be contaminating. Therapeutic approaches such as BT and CBT seek to help people to overcome avoidance**.**

Behaviour Therapy (BT): is a psychological therapy and is an umbrella term for a range of interventions including exposure and response prevention and behavioural activation (see below). Behaviour therapy, also called behaviour modification or behavioural psychotherapy refers to the use of learning theory in the treatment of psychological disorders. It is based on the belief that psychological problems are caused by faulty learning rather than a medical disease. BT aims primarily to help people to manage/change unhelpful behaviours. For example, in OCD, behaviour therapy often involves confronting feared situations (exposure) and refraining from performing rituals (response prevention). For OCD and BDD, BT is often synonymous with Exposure and Response Prevention (see below).

Body Dysmorphic Disorder: A preoccupation with an "imagined" defect in one's appearance or where there is a slight physical anomaly, the person's concern is markedly excessive. To fulfil diagnostic criteria in DSM-IV,the person must be either significantly distressed or handicapped in their occupational or social functioning. The older term, "dysmorphophobia" was first introduced by an Italian psychiatrist, Morselli. in 1886 although it is now falling into disuse probably because ICD-10 has discarded it and subsumed it under that of Hypochondriacal disorder..

Case Series: A study of the treatment of a number of people which is normally evaluated with standardised instruments at different times such as before treatment, after treatment and at follow-up some time after treatment. Unlike controlled trials or cohort studies, there is usually no control or comparison group. Although useful in early studies of new treatments, they are not considered to be a rigorous test of a treatment,

Case Study: A detailed description of the treatment of a single individual. Such studies may have an important role in the development of new treatments, but do not generally allow strong conclusions to be made about effectiveness.

Cognitive Analytic Therapy (CAT): A form of psychotherapy that is based on a combination of ideas from psychodynamic theory, with techniques from cognitive therapy. Therapy involves looking at the individual's patterns of relating to others, as well as their behaviours and problems, which is based on a model called the Procedural Sequence Model.

Cognitive Behaviour Therapy (CBT): In the treatment of OCD and Body Dysmorphic Disorder, CBT generally combines elements of BT such as Exposure and Response prevention and elements of CT such as techniques to change beliefs about the things that they find distressing. For people with OCD this often involves reducing catastrophic thinking and the exaggerated sense of responsibility often seen in people with OCD. All CBT programs for OCD include an element of psychoeducation about obsessions and compulsions and a rationale for the interventions.

Cognitive Therapy (CT): Is a psychological therapy and is an umbrella term that covers a range of interventions that focus on altering/modifying unhelpful thoughts and behaviour. Typically it helps people to modify unhelpful negative cognitions (that is, interpretations, thoughts and beliefs) that lead to disturbing emotions, unhelpful behaviours and impaired functioning. For OCD, this may be about taking excessive responsibility or giving too much importance to obsessive thoughts. For BDD, this may be about altering extreme self-focussed attention on a distorted body image and the meaning that is attached to the body image . People may be encouraged to test out the new ways of thinking through behavioural experiments, but CT does not usually rely on repeated exposure as in ERP.

Cohort study (also known as follow-up, incidence, longitudinal, or prospective study): An observational study in which a defined group of people (the cohort) is followed over time and outcomes are compared in subsets of the cohort who were exposed or not exposed, or exposed at different levels, to an intervention, or other factor of interest. Cohorts can be assembled in the present and followed into the future (a 'concurrent cohort study'), or identified from past records and followed forward from that time up to the present (a 'historical cohort study'). Because random allocation is not used, matching or statistical adjustment must be used to ensure that the comparison groups are as similar as possible.

Comorbidity: More than one diagnosis/disorder occurring in the same person

Compulsions: Compulsions, sometimes known as rituals, are behaviours that people feel pressured to do to reduce anxiety, guilt and distress, or to prevent harm from occurring. Compulsions are often repeated, conducted according to strict rules, and time consuming. Although the goal may be to reduce anxiety, performing them can also lead to distress and frustration. The pressure to engage in these behaviours can prevent people from doing other things that they wish to do and cause significant impact on their lives and the lives of those around them. Compulsions can take almost any form but common forms include washing and cleaning, checking, hoarding, ordering and arranging, and repeated questions. Many compulsions are overt, that is, they could be observed by others, for example hand washing rituals. However, other compulsions are covert, that is, they could not be seen by

others because they are of a mental nature, for example, mentally repeating sentences. Although many people may try to resist these behaviours, they may find themselves unable to do so either because of the distress caused by resisting them or because they believe that the consequences of not performing the compulsion are unacceptably dangerous.

Confidence interval: The range within which the 'true' values (e.g., size of effect of an intervention) are expected to lie with a given degree of certainty (e.g., 95% or 99%). Confidence intervals represent the probability of random errors, but not systematic errors – or bias.

Controlled trial: An experiment in which investigators allocate eligible people into groups to receive or not to receive one or more interventions that are being compared.

Cost-effectiveness analysis: A type of full economic evaluation that compares competing alternatives of which the costs and consequences vary. The outcomes are measured in the same non-monetary (natural) unit. It expresses the result in the form of an incremental (or average or marginal) cost-effectiveness ratio.

Costing study: This is the simplest economic study, measuring only the costs of given interventions. It does not provide answers to efficiency questions.

Cost-of-illness/economic burden studies: An economic analysis of the total costs incurred by a society due to a specific disease.

Costs (direct): The costs of all the goods, services and other resources that are consumed in the provision of a health intervention. They can be medical or non-medical.

Costs (indirect): The lost productivity suffered by the national economy as a result of an employee's absence from the workplace through illness, decreased efficiency or premature death.

Counselling and Supportive Psychotherapy: A range of counselling methods are used in practice, including supportive, psychodynamic, and cognitive-behavioural counselling. The most widely practiced form of counselling is supportive counselling/psychotherapy. This is defined as a way of relating and responding to another person, so that the person is helped to explore their thoughts, feelings and behaviour; to reach clearer self-understanding; and then is helped to find and use their strengths so that they cope more effectively with their lives by making appropriate decisions, or by taking relevant action. Essentially then, counselling is a purposeful relationship in which one person helps another to help him- or herself. In most cases it does not attempt to directly change the key features of OCD or BDD but can address a range of issues that may affect the individual.

Crossover Study Design: The administration of two or more experimental therapies one after the other in a specified or random order to the same group

of patients.

Double blind: A trial in which neither the participants nor the investigators (outcomes assessors) are aware of which intervention the participants are given. The purpose of blinding the participants (recipients and providers of care) is to prevent performance bias. The purpose of blinding the investigators (outcome assessors) is to protect against detection bias.

Drop out: A term no longer used to indicate leaving a study before its completion (the term 'leaving the study early' is now preferred).

Economic evaluation: Technique developed to assess both costs and consequences of alternative health strategies and to provide a decision-making framework.

Effectiveness: The extent to which a specific intervention, when used under ordinary circumstances, does what it is intended to do. Clinical trials that assess effectiveness are sometimes called management trials.

Efficacy: The extent to which an intervention produces a beneficial result under ideal conditions. Clinical trials that assess efficacy are sometimes called explanatory trials and are restricted to participants who fully co-operate. The randomised controlled trial is the accepted 'gold standard' for evaluating the efficacy of an intervention.

Exposure and Response Prevention (ERP): The person is encouraged to confront the feared object, situation, or thought that provokes anxiety (Exposure) and resist engaging in the compulsive or other behaviour that would rapidly reduce anxiety (Response Prevention) until the anxiety gradually reduces of its own accord. For example, people with obsessions about contamination are encouraged to stay in contact with the 'germy' object (e.g. handling money) until their anxiety decreases (habituates). Thus, through repeated exposure, the person is said to habituate so that the object, situation or thought no longer provokes anxiety and the urge to engage in the compulsive behaviour is no longer present. The ERP programme is usually conducted in a progressive way, starting with objects, situations, or thoughts that produce relatively low levels of distress. Exposure can be conducted in a variety of ways including, ***in vivo***, that is direct confrontation with the feared object or situation, and **in imagination**, where the person repeatedly imagines the feared object, thought, or situation. Taped scenarios can be used for exposure, especially when the cause of distress is particular obsessive thoughts. The technique can be used within structured therapy with the therapist present or with instructions from the therapist who is not present. It can also be used with support from a family member or other members of self-help groups, or alone as part of a pure self-help programme.

Eye Movement Desensitisation and Reprocessing (EMDR): Originally developed for the treatment of trauma, patients are instructed to focus on a trauma-related image and its accompanying feelings, sensations and

thoughts, while visually tracking the therapist's fingers as they move back and forth in front of the patient's eyes. After each set of approximately 20 eye movements, patients are instructed let go of the memory and to discuss their reactions. This process is repeated, and includes focusing on different memories that come up in connection with the trauma. Once distress is reduced, patients are instructed to focus on the target image while rehearsing a positive thought connected to the image.

Family Therapy: A form of psychotherapy that is based on the assumption that psychological problems are the product of, or result in, abnormalities in communication and interaction between family members. All family members are therefore seen together in order to discuss and change the ways that they relate together. It aims to help family members understand patterns of communication in their family, and to develop more functional patterns of organising and interacting within the family. There are various forms of family therapy used in the treatment of OCD, including Systemic, Strategic and Cognitive-behavioural family therapy:

- Systemic Family therapy tends to focus on the meaning of OCD symptoms within the family unit. The therapy tends to see OCD symptoms as a sign that the family unit is stressed, leading to difficult, unspoken emotions between family members. A systemic treatment might involve the therapists exploring 'OCD stories' within a family, thus changing the way that the family members have co-created the meaning of OCD. This is aimed at improving relationships within the family and in turn, changing the meaning of the individuals symptoms to allow for changes in their behaviour.
- Strategic Family Therapy tends to focus on power issues within the family, which in turn may well impact on both family members understanding and response to OCD, and the OCD symptoms themselves. The rationale is that a more flexible, creative family structure may reduce stress, and challenge the 'power' of the OCD symptoms.
- Cognitive-Behavioural Family Therapy as a treatment for OCD is based on the recognition that families of people with OCD become involved in trying to help manage the distress and the interference caused by the compulsions. The OCD thus disrupts family relationships. The focus of the treatment is to help the family understand how their well-intentioned involvement can inadvertently maintain the disorder and then help them withdraw from the compulsions. In some cases, especially with children, family members may act as co-therapists to help with exposure and response prevention. Although the aim is to improve family relationships, the focus is more on reducing a particular individual's obsessive-compulsive symptoms in which other family members have become involved.

Forest plot: A graphical display of results from individual studies on a

common scale, allowing visual comparison of trial results and examination of the degree of heterogeneity between studies.

Good Practice Point (GPP): Recommended good practise based on the clinical experience of the Guideline Development Group.

Group Therapy: A way of providing psychotherapy that usually involves one or more health professionals and a group of patients. The patients are encouraged to understand their own and one another's difficulties with the goal of making changes. Most individual psychological therapies have also been provided in group format: e.g. Group cognitive behaviour therapy or group behaviour therapy

Guided self-help (GSH): A self-help programme for OCD in which a health professional provides support, guidance and encouragement but does not take on the role of a formal therapist. Such programmes may use a self-help manual and the health professional may work face-to-face, by telephone or by computer.

Guideline recommendation: A systematically developed statement that is derived from the best available research evidence, using predetermined and systematic methods to identify and evaluate evidence relating to the specific condition in question.

Heterogeneity: This occurs when there is more variation between the study results (in a systematic review) than would be expected to occur by chance alone.

Hypnotherapy: This involves giving the patient instructions (e.g., "focus on your right arm and on the sensation that it is getting lighter and lighter") to induce a state of highly focused attention, a reduced awareness of peripheral stimuli and a heightened responsiveness to social cues (suggestibility).

Intention to treat analysis: A method of analysis for randomised trials in which all patients randomly assigned to one of the treatments are analysed together, regardless of whether or not they completed or received that treatment.

Interpersonal psychotherapy (IPT): A specific form of focal psychotherapy that is designed to help patients identify and address current interpersonal problems. It was originally developed for the treatment of depression (Klerman et al, 1984) but has been adapted for the treatment of other disorders such as eating disorders. In IPT there is no emphasis on directly modifying target symptoms; rather, it is expected that they will change as interpersonal functioning improves.

Licensing (now known as marketing authorisation): A process in which the doses, indications, cautions, contraindications, and side effects of a drug are authorised for use by regulatory authorities. The decision to apply for a license for a patient condition depends on many factors. Drugs may be used

outside their licensed indications.

Meta-analysis: The use of statistical techniques in a systematic review to integrate the results of the included studies. Also used to refer to systematic reviews that use meta-analysis.

Mindfulness: A meditation based approach to treatment that has been developed in particular for relapse prevention. Mindfulness has been defined as "paying attention in a particular way: on purpose, in the present moment and non-judgmentally". Mindfulness- based cognitive therapy aims to help patients make a shift in their relationship to thoughts, feelings and sensations, learning to perceive them as "events in the mind" rather than as "self" or necessarily true.

Monoamine-oxidase inhibitors (MAOI). a group of drugs that act by inhibiting the enzyme monoamine oxidase

Number of people leaving the study early: For the purposes of the guideline, the number of people leaving the study early due to any reason is taken as a proxy for treatment acceptability, whereas the number of people leaving the study early due to adverse events is taken as a proxy for treatment tolerability. An exception to this is when the comparison group is a wait-list control, in which case this assumption is not made.

Odds ratio: This is a measure of the relative benefit of the experimental treatment that can be obtained by dividing the experimental odds by the control odds.

Obsessions: Sometimes known as obsessive thoughts or ruminations, obsessions are unwanted and recurrent thoughts, doubts, or images that intrude into one's mind despite attempts to resist or control them. They may be fleeting thoughts, or they may stick in one's mind for long periods of time despite attempts to dislodge them. They are upsetting and may cause anxiety, guilt and shame. They usually lead to compulsions and/or avoidance (see above) as people try to remove or control the thoughts, deal with the situations that the thoughts refer to, or reduce the distress. The thoughts may be about almost any content, but common themes include dirt and contamination and their effects on self or others, harm that one may cause to others or failure to prevent harm from occurring, personally unacceptable blasphemous, immoral, or sexual thoughts, or excessive preoccupation with moral, religious or existential questions.

PANDAS (Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus) refers to research studies which show that in some individuals OCD appears to be triggered by Streptococcal infection

Patient: The terms 'patient' or 'person with OCD/BDD', etc.', are used in this guideline to identify the person presently or formerly with the condition

and/or receiving services in the present or past. The term "sufferer" is sometimes used by self help groups.

Psychoanalysis: A school of psychology and a method of treating mental health problems that is based upon the teachings of Sigmund Freud (1856-1939). There are many different psychoanalytic theories of OCD. Obsessions and compulsions are seen as symptoms of some deeper problem in the person's unconscious mind. The compulsive acts and obsessional thoughts are seen as defensive reactions which suppress the real hidden anxieties.

Psychodynamic Psychotherapy: Focuses on understanding the meaning of the target symptoms such as obsessions in the context of the individual's personality, attitudes and early experiences. The emphasis lies on resolving the unconscious conflicts that are thought to underlie the symptoms. Treatment strategies include exploratory insight-oriented, supportive or directive activity, working with transference, but with the therapist using a less strict technique than that used in psychoanalysis.

Psychoeducation: Educating people with OCD/BDD and their families about the symptoms of OCD/BDD, possible origins of the condition, its evolution over time, and the various treatments available. It also includes education about the symptoms and treatment of any comorbid disorders such as depression.

Quality of Life (QOL): Used in some treatment studies to show improvement in a person's condition beyond reduction in symptoms, measures of QOL can be defined broadly and include satisfaction, especially within important areas of one's life, the level of functioning in different areas, and the objective circumstances in which one lives. In many studies, however, QOL is defined narrowly as the level of functioning or degree of handicap which is one important aspect but limited as a marker of quality.

Randomisation: Method used to generate a random allocation sequence, such as using tables of random numbers or computer-generated random sequences. The method of randomisation should be distinguished from concealment of allocation, because if the latter is inadequate selection bias may occur despite the use of randomisation. For instance, a list of random numbers may be used to randomise participants, but if the list were open to the individuals responsible for recruiting and allocating participants, those individuals could influence the allocation process, either knowingly or unknowingly.

Randomised controlled trial (RCT): Also termed 'randomised clinical trial', this is an experiment in which investigators randomly allocate eligible people into groups to receive or not to receive one or more interventions that are being compared. The results are assessed by comparing outcomes from the different groups. Through randomisation, the groups should be similar in all aspects apart from the treatment they receive during the study.

Relative risk: Also known as risk ratio; the ratio of risk in the intervention group to the risk in the control group. The risk (proportion, probability or rate) is the ratio of people with an event in a group to the total in the group. A relative risk (RR) of 1 indicates no difference between comparison groups. For undesirable outcomes, an RR that is less than 1 indicates that the intervention was effective in reducing the risk of that outcome.

Relaxation: Relaxation therapy is aimed at teaching a patient to reduce the uncomfortable physical sensations that anxiety produces. Although there is a range of relaxation techniques, the most common one aims to help people relax by systematically tensing and relaxing various muscle groups. This can be used by itself to help people cope with stressful situations or as a part of desensitisation to specific fears. It is also a part of Anxiety Management Training. In some trials of psychological treatments, relaxation has been used as a comparator condition.

Rituals: See *compulsions*

Ruminations: see *obsessions*

Serotonin reuptake inhibitors (SRIs): A group of drugs that act by inhibiting the neuronal reuptake of the neurotransmitter serotonin.

Selective serotonin reuptake inhibitors (SSRIs): Medicines that more selectively inhibit the reuptake of the neurotransmitter serotonin into the presynaptic neurone

Self-help: This involves following a self-help programme for OCD, either on one's own (pure self-help), with the support of a group (self help groups), or with support, guidance and encouragement from a mental health professional (guided self-help), or Self-help programmes can use books, computerised materials, audio and videotapes. Effective self-help programs aim to improve self-management skills in addition to providing knowledge and information.

Self-help groups: Many people with health problems find it helpful to meet others with similar difficulties, for support, advice and social contact. These are usually run by people with OCD, for people with OCD (and sometimes their carers/families). Some groups also have professional support for some of their activities such as providing information. Throughout the UK there is an informal network of support groups for people with OCD, which are self-organised and run. The national charity for people with OCD and their carers, OCD Action attempts to maintain a directory of these.

Sensitivity analysis: Sensitivity analysis is a technique used in economic analysis or decision-making to allow for uncertainty by testing whether plausible changes in the values of the main variables affect the results of the analysis.

Stepped-care model: A sequence of treatment options aiming to provide the most appropriate and cost effective interventions according to both patient

need and locally available resources. When appropriate, simpler and less expensive interventions will be offered first, moving to more complex and intensive interventions if the patient has not benefited.

Sufferer: See patient

Tics: A tic is an involuntary, rapid, recurrent movement or sound. Examples of simple motor tics include eye-blinking and neck jerking. Simple vocal tics include throat clearing, barking noises, sniffing. Complex tics include jumping, or saying whole words. There is some suggestion that tics seem to be genetically related to OCD in that they can run in families; some people with OCD also have tics.

Tourette's Syndrome (TS or Gilles de la Tourette Syndome). A chronic form of tic disorder where both vocal and motor tics have been present for a year or more. Some studies suggest that about half of people with Tourette's also have OCD although the opposite has not been shown.

Transcranial magnetic stimulation (TMS): involves the use of a pulsed magnetic field to induce changes in function in cortical structures. It was developed in 1985 (Barker et al, 1985) to investigate the cerebral cortical activity and more recently has been used therapeutically for some mental disorders, namely, depression and OCD. In OCD this treatment aims to modify prefrontal cortical activity in order to influence obsessive –compulsive symptoms.

Treatment resistance: A relative failure to respond adequately to a treatment.

Uncontrolled Trial: A treatment trial where no attempt is made to compare the investigated treatment with a matched comparator, either active or neutral placebo.

Vagus nerve stimulation (VNS): developed for the control of epilepsy that cannot be managed by normal medical treatment. It involves the electrical stimulation of the vagus nerve within the neck by an electrode connected to a programmable stimulator.

Weighted mean difference: A method of meta-analysis used to combine measures on continuous scales (such as the Y-BOCS), where the mean, standard deviation and sample size in each group are known. The weight given to each study (e.g., how much influence each study has on the overall results of the meta-analysis) is determined by the precision of its estimate of effect and, in the statistical software used by the NCCMH, is equal to the inverse of the variance. This method assumes that all of the trials have measured the outcome on the same scale.

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