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REVIEW ARTICLE

A call for prevention and early intervention in obsessive-compulsive disorder

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Background: Evidence suggests that many people with obsessive-compulsive disorder (OCD) have subclinical symptoms years before the development of their disorder and that early treatment may reduce its severity.

Aim: To explore prevention and early intervention strategies for OCD.

Methods: A narrative literature review was conducted.

Results: The literature in relation to the prevention of OCD is sparse. Genetic and environmental factors appear to be relevant to the aetiology of OCD, for example, the observation that hoarding symptoms and contamination/cleaning symptoms are more likely to also be present in first-degree relatives. Psychoeducation and the reduction of family accommodation, that is the act of parents, siblings or partners accommodating to the high-risk individual's requests to comply with their compulsions, are promising areas for prevention and early intervention in high-risk groups. Tertiary prevention has also been limited by an inadequate number of trained clinicians to deliver evidence-based treatments.

Conclusions: Much more research is needed in relation to the prevention of OCD. There is limited scope for primary prevention with respect to biological aetiological factors, but there is potential for strategies addressing environmental factors (eg, family factors). The effectiveness of psychoeducation for parents with OCD as a primary prevention strategy for OCD in their children requires scientific evaluation. Improving access to effective treatments for OCD would also improve tertiary prevention.

KEYWORDS

early intervention, learning theory, obsessive-compulsive disorder, prevention

1 | INTRODUCTION

Obsessive-compulsive disorder (OCD) is a disorder characterised by recurrent intrusive and distressing thoughts, images or impulses (obsessions), and repetitive behaviours or mental acts (compulsions). OCD is thought to occur in 2% of the population (Fullana et al., 2010) and is among the top 10 most disabling of all medical conditions (Murray & Lopez, 1996). The course of OCD is often chronic with many people continuing to have symptoms despite treatment (Eisen et al., 1999). Hence, it is important to search for ways in which this distressing and disabling disorder can be prevented.

Prevention of mental disorders requires the identification and eradication of aetiological factors or the enhancement of an individual's resilience (Newton, 2013). Prevention is categorised into 3 levels. Primary prevention involves the prevention of the development of symptoms. This may involve improving an individual's resilience and resistance to OCD symptoms developing. Secondary prevention is the early identification and treatment of individuals who develop obsessive-compulsive symptoms to prevent progression to "full-blown" OCD. Finally, tertiary prevention reduces levels of disability in individuals with OCD.

This review uses a prevention framework to elucidate aetiological factors and opportunities to enhance resistance to OCD symptoms.

2 | METHODS

A systematic search of the databases PubMed (from 1946), EMBASE (from 1966), PsychINFO (from 1806), Google and Google Scholar

through to September 4, 2016 was performed to identify relevant articles. The search used the pre-determined terms "obsessive-compulsive*" OR "obsessions" OR "compulsions" AND "primary prevention" or "secondary prevention" or "tertiary prevention" or "resilience" or "aetiological" or "causative" or "early intervention"; these terms were searched as text word and as exploded medical subject headings where possible. Reference lists of relevant articles were also searched. Papers were included if they referred to prevention or aetiology in relation to OCD. Papers were excluded if they did not mention OCD or if the aetiological mechanism described was not relevant to prevention. No language restrictions were used. Unpublished studies were not searched. The search was conducted solely by author V.B.

3 | RESULTS

There were no papers specifically assessing primary prevention in OCD. The initial search yielded 841 papers, once duplicates were removed, there were 199 papers. Even fewer papers were relevant with most of these papers relating to the 5 major topics presented below.

3.1 | Biological aetiological factors

Despite biological and psychological theories based on behavioural, neuroimaging, and genetic evidence the aetiology of OCD remains speculative. Higher rates of OCD have been found in first-degree relatives of people with OCD (Bhattacharyya, Prasanna, Khanna, Reddy, & Sheshadri, 2005; Grados, 2009; Pauls, n.d.). This rate increases when probands have a comorbid tic disorder, younger age of onset and are male (Chacon et al., n.d.; Leckman et al., n.d.; Mathews et al., n.d.). Hoarding (Lochner et al., 2005; Samuels et al., 2007) and contamination symptoms (Brakoulias et al., 2016) have also been associated with higher rates of similar symptoms in first-degree relatives. Complex genetic associations and twin studies point to gene-environment interactions (Iervolino et al., 2009; Iervolino, Rijsdijk, Cherkas, Fullana, & Mataix-Cols, 2011; van Grootheest, Boomsma, Hettema, & Kendler, 2008). These interactions are, as yet, poorly understood. Hence, genetic testing and therapies do not have a current role in prevention.

OCD has been associated with aberrant cortico-striatal-thalamic-cortical anatomy and physiology. The orbitofrontal cortex in particular has been repeatedly implicated. This same circuitry governs flexible behaviour. There is evidence of inflexible behaviour in adults with OCD, such as excessive avoidance habits after over-learning (Gillan et al., 2014) and impaired outcome prediction (Gillan et al., 2011). Clinical neuropsychological tests have identified impaired response inhibition and set-shifting (Mataix-Cols et al., 1999; Tremblay & Schultz, 2000). Indeed, behavioural inflexibility has been found in people with OCD and their unaffected first-degree relatives—an indication that behavioural inflexibility is an endophenotype (Chamberlain et al., 2008). Such deficits may represent an early risk factor of disease onset and progression—an area of opportunity for indicated primary prevention.

Theories postulate the role of the neurotransmitters serotonin, dopamine and glutamate in the pathophysiology of OCD. People with OCD were found to have elevated levels of glutamate in the cerebrospinal fluid (Chakrabarty, Bhattacharyya, Christopher, & Khanna, 2005). In small studies, agents such as N-acetylcysteine, memantine and riluzole, which act on the glutamatergic system, have shown promise as adjunctive agents in the treatment of OCD (Afshar et al., 2012; Coric et al., 2005; Ghaleiha et al., 2013; Kariuki-Nyuthe, Gomez-Mancilla, & Stein, 2014). The nascent understanding of the role of these neurotransmitters in OCD symptoms is juxtaposed with the risks of medicating people without a disorder. Thus, neurochemical manipulation is not suitable as a primary prevention target. However, serotonergic medications, that is selective serotonin reuptake inhibitors (SSRIs) and clomipramine have long been a mainstay of pharmacological tertiary prevention and are augmented by dopamine blocking agents. SSRI are readily accessible to most patients, but non-specialised practitioners are often less familiar with the longer time OCD symptoms take to improve and for the need for high doses (Chakrabarty et al., 2005).

It has also been hypothesised that some childhood onset OCD may be precipitated by neuroimmunological reactions. The most widely reported being secondary to infection by group A beta-haemolytic streptococci and known as Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcal Infection (PANDAS) (Swedo et al., 1998). In such cases, OCD symptoms have improved with plasma exchange, intravenous immunoglobulin and antibiotic prophylaxis (Kirvan, Swedo, Snider, & Cunningham, 2006; Snider, Lougee, Slattery, Grant, & Swedo, 2005). However, streptococcal infection does not appear to play a role in the majority of cases of OCD, as anti-basal ganglia antibodies were present in 19.8% of adult patients with OCD in 1 study (Nicholson et al., 2012). Without additional risk factors, such as a vulnerability to autoimmune disease, it would not be practical to advocate for widespread antibiotic or immunosuppressant use as an indicated primary prevention strategy.

3.2 | Psychological aetiological factors

Somatic and separation anxiety symptoms in early childhood are predictive of anxiety disorders and OCD, and obsessive concern is predictive of subclinical OC symptoms. Early treatment of these symptoms in children may be important for preventing the course of OCD. Socio-economic disadvantage also appears relevant and social equity measures may have a role in primary prevention (Moreso, Hernández-Martínez, Val, & Sans, 2013).

Psychological theories have attempted to explain OCD symptoms from several perspectives. The most widely used is a behavioural model that proposes that obsessions are learnt fear responses which are reinforced by avoidance and compulsions, which in turn are learnt responses to the anxiety arising from the obsessions (Salkovskis, 1999). However, theories of learnt fear responses in OCD do not explain the consistent OCD symptom domains that occur across cultures. Cognitive models propose that the cognitions that follow obsessions are abnormal and that the appraisal of obsessions leads to anxiety and distress (Salkovskis, 1999). Supporting

cognitive and behavioural aetiological theories is evidence that CBT for OCD changes brain function (Baxter et al., 1992). The strongest evidence base exists for exposure and response prevention therapy (ERP). Unfortunately, it is not always easy to access ERP trained and experienced therapists.

3.3 | Parenting and OCD aetiology

The role of parenting in the aetiology of OCD is unclear. Some evidence indicates that parents of children with OCD are more over-protective, rejecting and less emotionally warm (Alonso et al., 2004; Lennertz et al., 2010; Timpano, Keough, Mahaffey, Schmidt, & Abramowitz, 2010). There have also been reports of parents of children with OCD having a more "authoritarian" parenting style characterised by high demands and high expectations where mistakes tend to be punished harshly and feedback is often negative (Timpano et al., 2010). There have also been some studies (Calvocoressi et al., 1995; Lebowitz, Panza, Su, & Bloch, 2012; Peris et al., 2008) reporting high rates of accommodation (ie, the participation in rituals, avoidance and the giving of reassurance) in the parents of children with OCD. Worse OCD symptoms have been associated with higher levels of accommodation (Stewart et al., 2008); and interventions to reduce accommodation may result in symptom alleviation (Thompson-Hollands, Abramovitch, Tompson, & Barlow, 2015). Hoarding and contamination/cleaning symptoms appear to be more prevalent in first-degree relatives of probands with similar symptoms (Brakoulias et al., 2016). Such observations could support learning theory and behavioural conceptualisations of OCD. Parenting styles are possibly an epiphenomenon, no study has established a temporal relationship. It is also important to note that the findings of these studies may not only apply to parents, but also to other people who live with the person who is suffering from OCD.

3.4 | Resilience

Resilience refers to one's ability to positively adapt to adverse circumstances. It entails 3 categories: personal disposition, family support and broader social resources (Garmezy, 1993). Higher resilience has been associated with lower rates of OCD in children (Hjemdal, Vogel, Solem, Hagen, & Stiles, 2011). These studies use cross-sectional comparisons between children with high resilience and children with low resilience. No study has demonstrated that improving resilience levels prevents the development of OCD. Resilience studies are developing clearer definitions and more objective assessment methods (Luthar, Cicchetti, & Becker, 2000).

3.5 | Early intervention

Earlier onset and longer duration before assessment have been shown to predict the likelihood of symptom persistence (Stewart et al., 2004). Although longitudinal studies are required to assess the effectiveness of early intervention, it can be postulated that early intervention provides an opportunity to reduce symptoms and their impact on a child's ability to meet developmental milestones (Freeman et al., 2007).

4 | DISCUSSION

As demonstrated by the limited number of studies that resulted from this literature search, there is a dearth of literature regarding primary prevention of this common and disabling disorder. Hence, there is a need for longitudinal investigations of the role of aetiological factors in the prevention of OCD. There is a genetic component, however, this appears complex (Nicolini, Arnold, Nestadt, Lanzagorta, & Kennedy, 2009) and there appears little hope of a genetic test or therapy in the near future. Environmental factors have arisen from cross-sectional and small case-control studies and centre around the family environment, yet attempts to modulate family factors have focussed on tertiary rather than primary or secondary prevention.

4.1 | Primary prevention

Given the current limitations in our understanding of genetic mechanisms of OCD, our focus should be on modifying environmental factors. Many environmental factors presented relate to the family environment and parenting. OCD begins before the age of 18 years in 80% of people (Goodman, Rasmussen, & Leckman, 1995). Increased mental health literacy could enhance the identification and treatment of OCD symptoms in parents with opportunities for psychoeducation and family interventions. Such interventions could reduce exposure of children of parents with OCD to abnormal fear responses and create a more supportive family environment. Prevention programmes may have the potential to modify parenting styles and reduce family accommodation. Schoolteachers are increasingly aware of other common psychiatric childhood disorders such as attention deficit hyperactivity disorder (ADHD) and oppositional defiance disorder (ODD) and frequently refer cases to mental health services. Autism spectrum disorder organisations provide school diagnostic assessment visits. Programmes to educate teachers on the early detection of subclinical OCD symptoms in students could deliver focussed interventions to children at risk of developing OCD. Strong reactions to everyday sensory events have been associated with high childhood ritualism (Dar, Kahn, & Carmeli, 2012) and obsessive-compulsive symptoms. A retrospective study found that marked ritualistic behaviour was predictive of the development of OCD (Leonard, Goldberger, Rapoport, Cheslow, & Swedo, 1990). Another target population of indicated primary prevention is children of parents with OCD—there is a 10-fold risk of the disorder in such children (Nicolini et al., 2009). Longitudinal research is needed to determine whether education and family interventions can primarily prevent OCD.

4.2 | Secondary prevention (Early intervention)

Early intervention programmes also need to be evaluated using longitudinal studies. Considering that a significant proportion of people with OCD have a chronic and debilitating course (Skoog & Skoog, 1999), it would be important to assess whether treating earlier is associated with symptom reduction and remission. Early intervention programmes for OCD could be delivered analogous to those for psychosis that are now more widely available (Bird et al., 2010). These

services could provide easier access to exposure and ERP and family therapy with an attempt to assess for and address family accommodation to OCD symptoms. Medical expertise in the assertive treatment and monitoring to optimise response with pharmacotherapy, when indicated, would also be of benefit.

4.3 | Tertiary prevention

The time delay between symptom onset and treatment is attributed to factors such as patient secrecy and under-recognition of OCD by the community and clinicians (Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006). Disability associated with OCD is often understated. Improved delivery of specialised services for the treatment of OCD could greatly assist with improving symptoms and function. There is also significant comorbidity associated with OCD with approximately 75% having a co-occurring other anxiety disorder (Brakoulias et al., 2011) and higher rates of ADHD, tic disorders, major depression (Hasler et al., 2007) and suicide (Alonso et al., 2010; Hasler et al., 2007; Kamath, Reddy, & Kandavel, 2007; Maina, Salvi, Tiezzi, Albert, & Bogetto, 2007). Improving access and delivery to established evidence-based treatments for OCD, that is high dose SSRIs and ERP, and to treatment of common OCD comorbidities could also improve tertiary prevention. The wider dissemination of clinical practice guidelines promoting evidence-based treatments would also assist with tertiary prevention.

5 | CONCLUSIONS

Despite the chronic and disabling nature of OCD, there is a glaring lack of knowledge regarding its aetiology and prevention. Aetiological factors for the development of OCD have been broadly divided into genetic and environmental factors. Identified environmental factors such as family accommodation appear to be modifiable. Primary prevention strategies could be assessed by implementing and evaluating programmes and strategies that aim to modify environmental factors that are thought to be associated with OCD. Secondary prevention may involve early intervention programmes analogous to those aiming to prevent the development of psychotic disorders. Tertiary prevention strategies require effective treatments such as high-dose SSRIs and ERP to become more accessible to people with OCD to prevent the long duration of untreated illness that currently exists.

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