Causes and risk factors

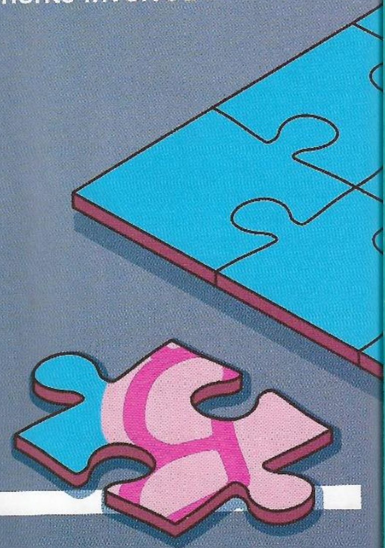
Getting a clear picture of what causes OCD can feel like solving a giant puzzle – with many different segments involved

Words Emma Green

Obsessive-compulsive disorder (OCD) is a chronic mental health condition marked by intrusive thoughts (obsessions) and repetitive behaviours (compulsions). Although the exact cause is not fully understood, most academics think it results from an interplay of biological, psychological, genetic and environmental factors.

A number of risk factors have also been identified that predict an increased likelihood of developing the condition. Risk factors act as vulnerabilities that increase the chances of developing OCD, such as having a family history of the condition, but do not guarantee its onset. Causes, however, refer to something that directly contributed to the condition's emergence and progression, such as a serotonin dysregulation.

By examining each of these components, we can better appreciate the complexity of OCD and why one single hypothesis cannot fully explain what causes the disorder.



Biological theories

Differences in brain structure and function

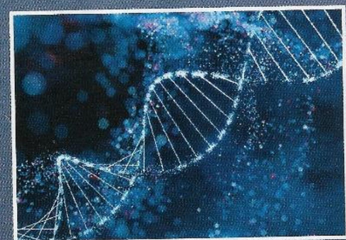
Advances in neuroscience and brain imaging have revealed important insights into the biological underpinnings of the disorder. One of the most compelling areas of study is how brain structure and function seem to differ in those with OCD, compared to those without it.

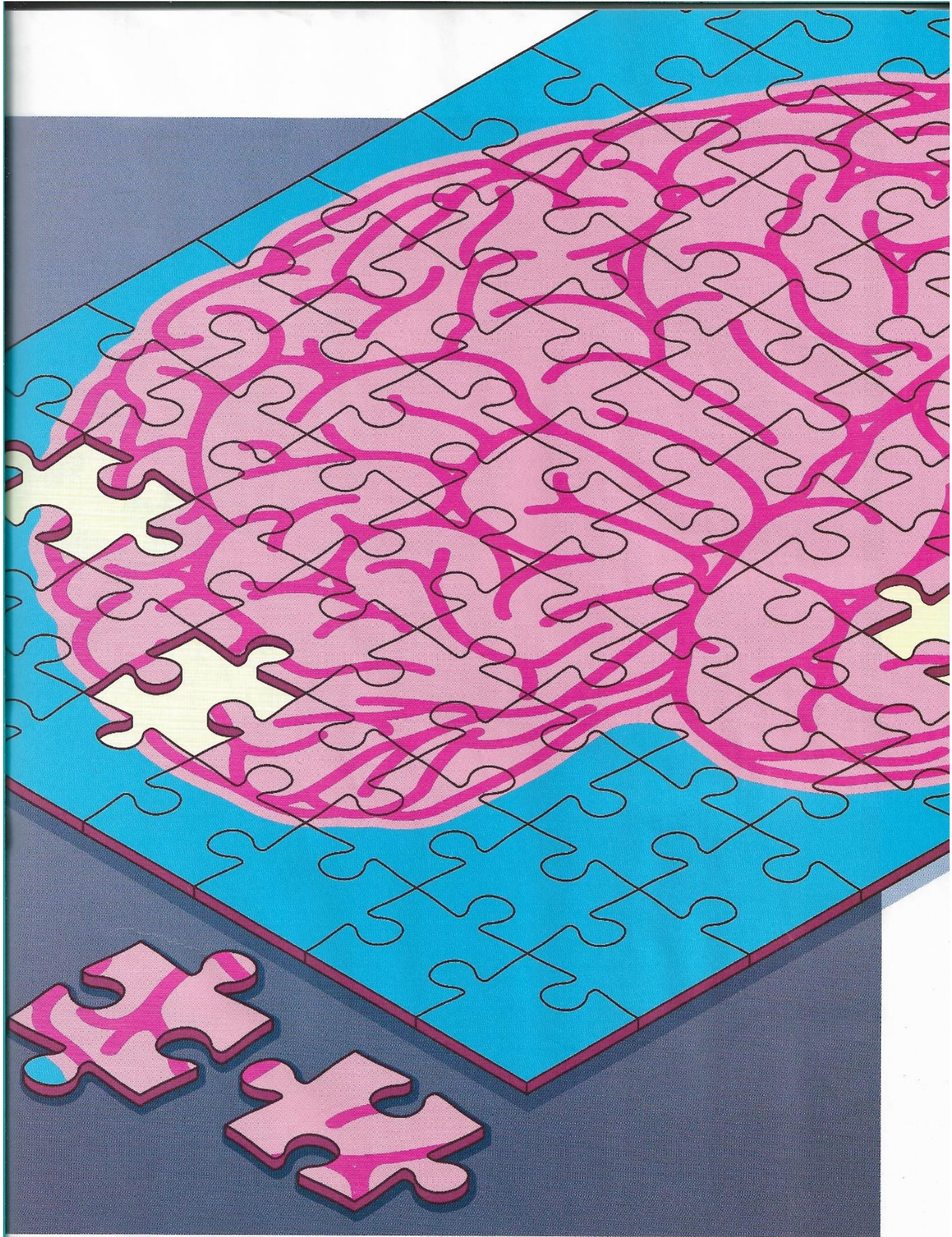
OCD is strongly associated with functional abnormalities in the cortico-striato-thalamo-cortical (CSTC) circuit, a network of interconnected brain regions that work together to control thoughts, emotions and actions.

This network includes:

The orbitofrontal cortex (OFC), which is associated with decision-making, evaluating risk and predicting consequences. In OCD patients, the OFC often shows overactivity, which may explain heightened concerns about danger or contamination, and contribute to intrusive thoughts.

The anterior cingulate cortex (ACC), which is linked with error detection, emotional regulation and impulse control. Overactivation in the ACC may give those with OCD an



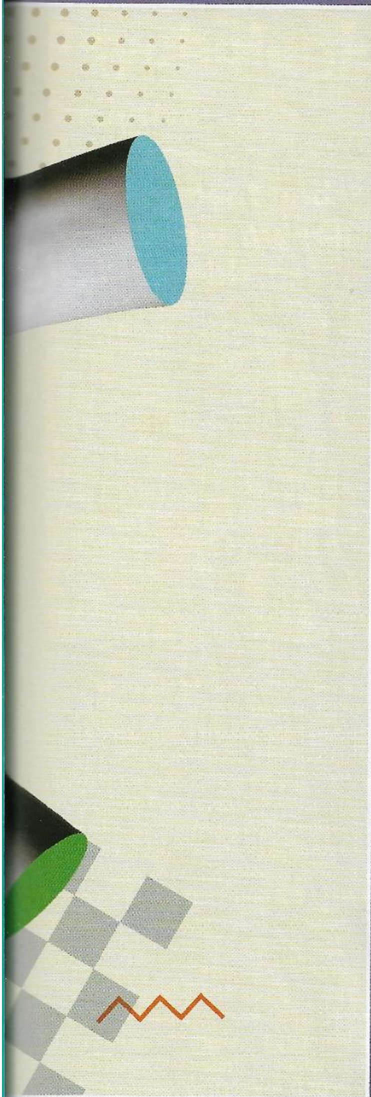


exaggerated sense that something is wrong, driving compulsive checking or correcting behaviours.

The basal ganglia (particularly the striatum and caudate nucleus), which play a role in motor control and habit formation, and help to filter out irrelevant information. In OCD, dysfunctional striatal activity may allow intrusive thoughts to pass through, fuelling compulsions.

The thalamus, which acts as a relay station for sensory and motor information, influencing cognitive and emotional processes. When overactive, the thalamus can reinforce the constant looping of obsessions and compulsions.

Hyperactivity within the CSTC circuit makes it difficult for the brain to suppress intrusive thoughts or to disengage from compulsive behaviours. Neuroimaging has also shown that people with OCD often have subtle structural differences compared to those without the disorder. Increased grey matter volume within the subcortical brain



regions (including the basal ganglia) may reflect an overuse of obsessive thought processing, whereas disruptions in white matter pathways connecting the frontal, striatal and thalamic regions suggest inefficient communication between the areas that control thought and behaviour. Some studies have found thalamic enlargement in those with OCD, which suggests overactivity in repetitive and compulsive thought-action loops.

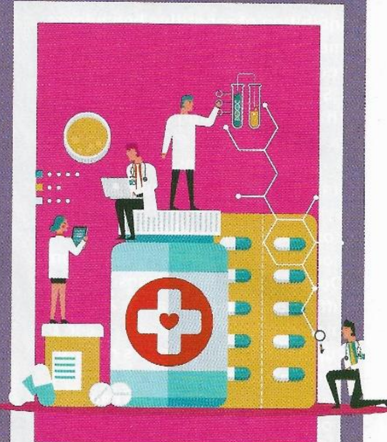
Other studies have shown that in some cases, a reduced hippocampal volume can contribute to difficulties with memory and a compulsive need to repeat checking behaviours. These structural differences may help to explain why the OCD brain is wired to become stuck in repetitive loops of thought and action.

Although brain scans can give some indication of who might have OCD, they can't be used to diagnose the condition. "There are significant overlaps and inconsistencies in imaging findings," says Dr Katie Barge, a chartered psychologist specialising in child and adult mental health. "Neuroimaging cannot capture personal history, symptom patterns or cognitive-behavioural context, which are all crucial for an accurate OCD diagnosis. They also can't accurately measure another potential biological cause of OCD: a neurochemical imbalance."

Disruption of key neurotransmitter systems

Another critical component in our understanding of the OCD puzzle are neurotransmitters – the brain's chemical messengers. These tiny molecules transmit signals that allow neurons to communicate with one another, helping to regulate thought, emotion and behaviour. It is believed that a dysregulation in several important neurotransmitters may play a central role in the development of the condition.

The prominent hypothesis is that a serotonin deficiency causes OCD. Serotonin is a neurotransmitter that helps to regulate mood, emotions, sleep, appetite and digestion. It is often linked to feelings of wellbeing and happiness, and imbalances in serotonin levels are associated with disorders like depression and anxiety. Serotonin is also responsible for the



Implications for pharmacological treatments

The first-line treatment for OCD are medications that enhance serotonin signalling, usually selective serotonin reuptake inhibitors (SSRIs). Dysregulation in serotonin transmission is strongly associated with OCD, which is supported by the general effectiveness of SSRIs in treating the disorder – they do this by blocking the neurotransmitter's reabsorption, hence increasing its availability in the brain for longer and reducing OCD symptoms. It would also explain why relapse is much more frequent after withdrawal in those with OCD compared to those who take SSRIs for other conditions, such as depression, because serotonin (or a lack of it) appears to be a core feature of the disorder.

Not everyone with OCD, however, seems to respond well to SSRIs, suggesting that serotonin is only part of the picture. In some cases, dopamine-modulating medications such as antipsychotics are used as an add-on treatment when SSRIs are not enough. Medications that regulate glutamate are also being studied as possible new therapies for treatment-resistant OCD.

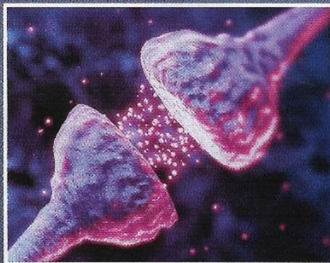
inhibition of repetitive behaviours, memory and impulse control, and reduced serotonin signalling in the CSTC circuit is believed to underlie the obsessions and compulsions seen in OCD.

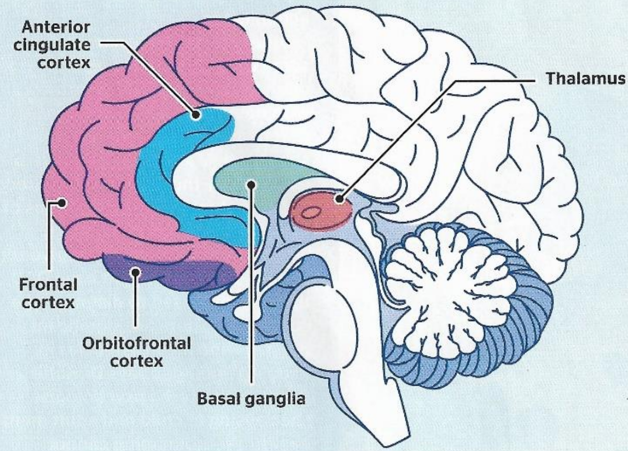
Other neurotransmitters play important roles in shaping the symptoms and severity of the condition, particularly regarding compulsivity and habit formation. Dopamine is a neurotransmitter affiliated with motivation and pleasure, and plays a key role in the functioning of the brain's reward system. Overactivity of dopamine pathways in the striatum can cause compulsions to become strongly reinforced, making them harder to resist.

Another neurotransmitter, glutamate, is the brain's main excitatory messenger, firing off neurons and communicating information to different networks. New research, including a recent study conducted by scientists at the University of Cambridge, has discovered that elevated levels of glutamate found in the CSTC circuit can fuel repetitive loops of obsessions and compulsions.

Furthermore, it has been found that gamma-aminobutyric acid (GABA), the brain's primary inhibitory neurotransmitter responsible for balancing the excitatory effects of glutamate, is reduced in the anterior cingulate cortex, which can make it even harder for the brain to switch off intrusive thoughts.

Genetic research has identified several candidate genes associated with OCD, many of which influence serotonin, dopamine and glutamate pathways. Variants of the SERT gene, for example, can disrupt serotonin balance, which contributes to the development of OCD symptoms. This then leads to another emerging player in the debate about OCD causes: genetics.





The CSTC circuit

The feedback loop between the cortex, basal ganglia, thalamus and then back to the cortex seems to be in overdrive in those with OCD.

Hereditary factors

"OCD is heritable," says Dr Barge. "Those with relatives such as a parent or a sibling with OCD are at elevated risk."

Family studies have indicated that up to 20% of those diagnosed with OCD also have an immediate family member who has it too. Twin studies also provide strong evidence of genetic influence, with higher concordance rates among identical twins compared to fraternal twins. A 2010 review[1] of twin studies examining OCD found that 68% of identical twins and 31% of non-identical twins shared the condition.

"A familial history of anxiety disorders, depression, hoarding and checking behaviours appears to cluster in families," says Dr Barge. Studies have shown a heritability rate of up to 50% between OCD and similar disorders of compulsivity, such as tic disorders like Tourette syndrome, body dysmorphic disorder, trichotillomania (hair pulling) and excoriation (skin picking). This genetic link with OCD is also seen with other family members who were diagnosed with anxiety or mood disorders like depression.

This could be because some first-degree relatives, despite being asymptomatic, share the same brain differences as their close relatives with OCD. Interestingly, several neuroimaging studies have also shown that relatives without the condition can have the same dysconnectivity between parts of the CSTC circuit, which is a biomarker for OCD.

However, a genetic predisposition alone does not determine OCD. Instead, it provides a vulnerability that may be triggered or exacerbated by environmental factors.

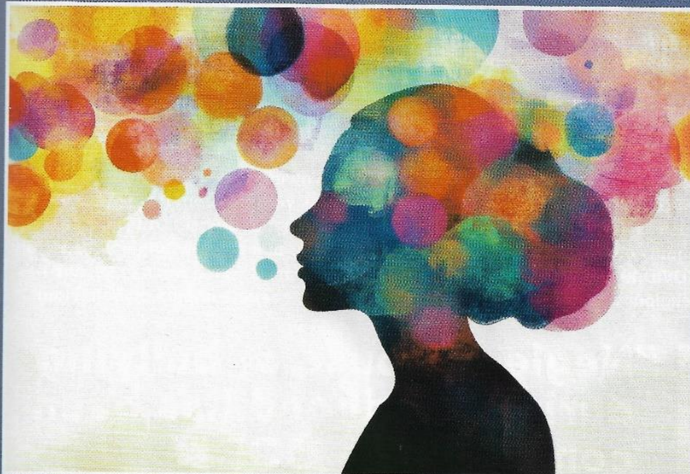
Psychological theories

Cognitive distortions, temperament and co-existing mental health problems

Everyone has intrusive thoughts from time to time, but people with OCD interpret them as dangerous or reflective of character, leading them to engage in compulsions to neutralise the perceived threat.

Cognitive theorists believe that certain thought patterns play a crucial role in the development and maintenance of OCD. These include an inflated responsibility to prevent harm, placing an overvalue on thoughts and a need to control them, overestimating threat and thought-action fusion (believing that having a thought is morally equivalent to acting on it). "Personality traits such as perfectionism, a high sensitivity to uncertainty, high harm avoidance and an excessive need for control are commonly implicated in OCD vulnerability," says Dr Barge.

It is clear to see why people who are highly conscientious, are overly responsible or have a meticulous nature are more likely to develop OCD than those who do not share these traits. People with excessively high standards of themselves or others are more prone to obsess over mistakes and generate compulsions aimed at correcting perceived errors. Those who find it difficult to tolerate ambiguity or doubt may engage in compulsive behaviours to achieve a feeling of certainty. Someone who is methodical may be more drawn to rigid thinking and strict rituals. Research has demonstrated a link between some of the Big Five personality traits and OCD; high scores in neuroticism and low scores in extraversion and agreeableness have also been associated with the disorder. None of these traits is inherently problematic, but coupled with other vulnerabilities, they can tip the balance towards developing OCD. Additionally, individuals with certain psychiatric conditions, such as anxiety and depression, are at higher risk of having OCD too. The presence of these additional disorders may perhaps reflect a shared underlying susceptibility in brain regions and neurotransmitter systems.



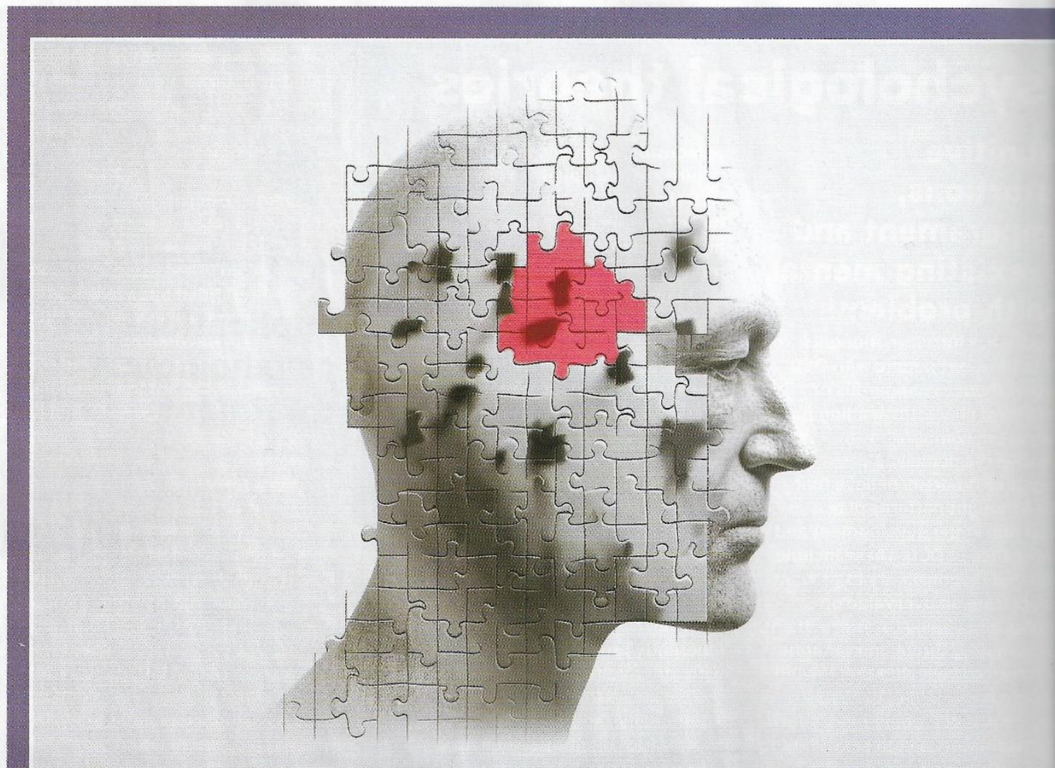
Implications for psychological treatment

Exposure and response prevention (ERP) therapy is the gold-standard treatment for OCD. A type of cognitive behavioural therapy (CBT), it works by gradually exposing individuals to situations, thoughts or images that trigger their obsessions, while helping them resist the urge to perform compulsive behaviours in response. By repeatedly facing feared triggers without engaging in rituals, the brain learns that the anxiety naturally decreases over time and that the feared outcomes are unlikely to occur. This process helps to break the cycle of obsessions and compulsions, reducing OCD symptoms and improving daily functioning.

ERP therapy also helps to rewire the brain structures involved in OCD. By addressing cognitive distortions and maladaptive learning patterns, it has been shown to retrain neural circuits, decreasing hyperactivity in the OFC and indirectly influencing neurotransmitter activity. Areas of the brain involved in self-control and decision-making, like the prefrontal cortex, are also strengthened.

Other possible therapies that can be useful in the treatment of OCD are acceptance and commitment therapy (ACT), as well as those that address any potential trauma that might have contributed to the onset of symptoms, such as trauma-focused cognitive behavioural therapy.

Stress management, mindfulness practices, exercise, good sleep hygiene and establishing support systems can also help to reduce environmental triggers.



External stressors, trauma and adverse childhood experiences

Traumatic life events such as an illness, accident or experiencing loss can sometimes act as triggers, or worsen existing OCD symptoms. Major life changes, such as starting a new job or having a child, can act as a catalyst. Exposure to chronic stress heightens the brain's threat-detection system, leading to increased obsessions and compulsions.

Exposure to neglect, abuse or bullying during childhood has also been connected to the occurrence of OCD symptoms in later life. Some studies suggest that harsh, critical or overly controlling parenting styles can contribute to OCD vulnerability, although research is mixed. This could be due to children using obsessions and compulsions as a coping mechanism for dealing with the anxiety they felt growing up under such circumstances.

Another potential environmental cause of OCD is learning from and modelling a caregiver's behaviour. Children who grow up observing obsessive or ritualistic behaviours may internalise similar coping methods or believe them to be normal. Often, the themes of obsessions reflect the period and culture that someone grew up in, such as having heightened concerns about contracting HIV/AIDs or COVID-19, or following extreme religious practices.

A rare but serious risk factor for OCD in children is infection-related autoimmunity. Some children can develop sudden-onset OCD followin a streptococcal infection. This condition – known as paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS) – is thought to occur when the immune system mistakenly attacks brain tissue, disrupting neural circui involved in OCD. This subtype can look very different from other forms of childhood-onset OCD, as it can occur at a much younger age, usuall appears within a week or so, and has a very severe impact on behaviour.

"Neglect, abuse or bullying during childhood has been connected to OCD"

The diathesis–stress model

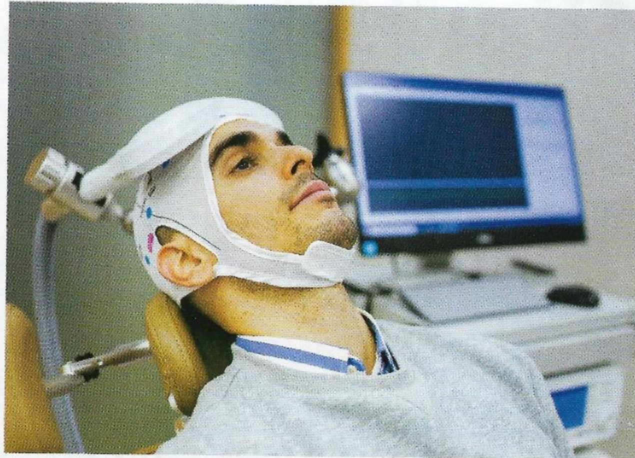
So, what is the most important piece to this puzzle? According to Dr Barge, there's no single factor that stands out. "The best explanation is viewing OCD as a biopsychosocial model with a neurodevelopmental-genetic core, interacting with psychological and environmental factors," says Dr Barge. "It is the interaction among these domains that best explains OCD vulnerability."

Most experts agree that OCD results from an interaction between predisposition (genetics, brain chemistry, personality) and environmental triggers (stress, trauma, infections). This is often referred to as the diathesis-stress model. In this framework, genetic and biological vulnerabilities as well as temperament (diathesis) create the foundation for OCD, while stressful life events activate or worsen symptoms. This model explains why some people with genetic vulnerabilities never develop OCD and why others without a family history can go on to develop the disorder after a traumatic event.

What implications does this have, then, for treatment? "Understanding potential contributing factors can help tailor treatments, guide prognosis and potentially improve outcomes by addressing root vulnerabilities," says Dr Barge.

Understanding the causes and risk factors of OCD is not purely an academic exercise. Because OCD typically develops in young people and can be highly impairing, by identifying individuals at higher risk, clinicians and families can monitor for early signs of OCD and intervene sooner. Knowledge of risk factors allows for personalised treatment, perhaps through targeted stress-management strategies, resilience training and psychoeducation. Promoting societal education around the fact that OCD is influenced by genetics, brain function and life experiences will help to combat misconceptions. This will hopefully reduce stigma and shame surrounding the condition.

OCD is a complex psychiatric condition. Although we don't need to pinpoint its cause in every single case, progress in neuroscience, genetics and psychology has helped to greatly improve our collective understanding of it. Most crucially, this knowledge has fuelled the development of more effective treatments, offering hope to millions who struggle with OCD worldwide.



Implications for neuromodulation treatments

While medication, therapy and lifestyle interventions are the typical pathway for treating OCD, in some cases, they may not be enough. For individuals with treatment-resistant OCD, neurostimulation techniques such as transcranial magnetic stimulation (TMS) and deep brain stimulation (DBS) can offer advanced treatment options by directly targeting brain circuits involved in OCD. Both options are typically used when traditional treatments have failed, and should be pursued under the care of specialists.

TMS is a non-invasive procedure that uses magnetic fields (via an electromagnetic coil placed against the scalp) to stimulate specific areas of the brain, particularly the orbitofrontal cortex, which is often overactive in OCD. Repeated sessions of TMS can help reduce obsessive thoughts and compulsive behaviours by modulating this brain activity. TMS is typically offered in outpatient settings to those with moderate to severe OCD who haven't responded to medication or therapy.

DBS is a surgical procedure used in only the most severe, treatment-refractory cases of OCD. It involves implanting electrodes deep in the brain, usually in the basal ganglia area or regions connected to the CSTC circuit. These electrodes deliver controlled electrical impulses to help regulate abnormal brain activity associated with OCD. It is usually only reserved for patients with chronic, disabling OCD who have not improved with multiple medication attempts and behavioural therapies.

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[1] Nestadt et al. (2010).