

THE NEUROBIOLOGICAL THERAPIST FEATURE

THE
NEUROBIOLOGICAL ROOTS OF
**Obsessive-Compulsive
Disorder**

THE IMPLICATIONS
FOR TREATMENT

By

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OBSESSIVE-COMPULSIVE DISORDER

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THE COMPULSIVE HAND WASHING OF LADY MACBETH IN SHAKESPEARE'S MACBETH, IS OFTEN REFERRED TO AS AN EXAMPLE OF OCD.

“THOU WOULDST BE GREAT,/ART NOT WITHOUT AMBITION, BUT WITHOUT/THE ILLNESS SHOULD ATTEND IT” . IF AT THIS STAGE SHE NEEDS TO USE A RITUAL OF HAND WASHING TO EASE HER ANXIETY, SHE HIDES IT, THOUGH HER GENTLEWOMAN TELLS HER DOCTOR LATER WHEN HE WITNESSES THE LADY SLEEPWALKING, “IT IS AN ACCUSTOM'D ACTION WITH HER, TO SEEM THUS WASHING HER HANDS. I HAVE KNOWN HER CONTINUE IN THIS A QUARTER OF AN HOUR” .

DSM IV-TR

The Diagnostic and Statistical Manual of Mental Disorders (DSM IV- TR) describes the diagnostic criteria of OCD as:

Either obsessions or compulsions:

Obsessions as defined by (1), (2), (3), and (4):

1. recurrent and persistent thoughts, impulses, or images that are experienced at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress
2. the thoughts, impulses, or images are not simply excessive worries about real-life problems
3. the person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action
4. the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)

Clients often report that they feel driven to perform ritualistic behaviours in an attempt to mitigate anxiety produced by unsettling obsessive thoughts. Are there any neural correlates linked with OCD? Are they linked to genetics, neural structures or neurochemicals? What role does nurture (early life experiences) play in the pathogenesis of OCD? In this brief overview we address some of these questions and explore the implications for therapeutic interventions. Obsessive-compulsive disorder (OCD) is a condition characterized by obsessions (recurring uncomfortable thoughts) and compulsions (ritualistic acts).

If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorder; hair pulling in the presence of Trichotillomania; concern with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges or fantasies in the presence of a Paraphilia; or

guilty ruminations in the presence of Major Depressive Disorder).

The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition. (APA 2000).

Prevalence

OCD affects about 2-3% of the general population and is the fourth most common disorder after phobias, substance abuse and major depression (Bear et. al 2010). The typical age of onset is late adolescence with equal gender distribution.

Genetic basis

There is evidence of a genetic basis for OCD. Concordance rates for monozygotic twins range from 53% to 87% and dizygotic twins from 22% to 47%. (Miguel

ronmental factors are vital to the development of the disorder. This does not rule out the genetic and neurobiological pathogenesis of the disorder but neuroscientists realise more and more that understanding disorders involves understanding a complex interaction of nature and nurture.

Nature vs Nurture

However complex, it is this same interaction that provides a window for treatment from an environmental perspective. The environmental perspective has demonstrated itself as probably the most powerful intervention to treat mental disorders. It simply means that enriched environments (like talking therapies, physical safety, nutrition, exercise, quality sleep) play a vital role in establishing new (more effective) synaptic connections – and thus changing the neural pathways and memory systems that maintain pathological behaviours.

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et.al. 1997). As the concordance rate is less than 100% it means that, although the genetic expression plays a major role in the development of OCD. The envi-

tive) synaptic connections – and thus changing the neural pathways and memory systems that maintain pathological behaviours.



The neuroanatomical circuit linked to OCD

Whilst doing research on neural circuits of epileptic patients in Paris, Jean Talairach stimulated the cingulum (part of the cingulate gyrus – the belt that spans over the corpus callosum) and found that when this area is stimulated patients could not suppress the urge to engage in repetitive behaviours. This area is very close to key limbic structures especially the amygdala (Talairach 1973). More elaborate studies followed. In 1989 Jack Modell and colleagues identified the link with the orbitofrontal cortex (OFC) (Modell et. al. 1989). The OFC is known for its key role in advanced cognitions and associations and more recently linked to the development and maintenance of personality. Research suggests that the frontal lobes and cingulate cortex project neural messages to the striatum (the input centre of the basal

ganglia) and then project inhibitory impulses to the thalamus (the input centre of external stimuli).

Effective functioning of this neuroanatomical circuit depends on

the appropriate information being transmitted from the thalamus to the frontal cortex. Neural circuits that are overactive and not responding to feedback from the thalamus

tend to engage excessively in particular patterns. These patterns can demonstrate themselves in neural circuits linked with obsessions (thoughts/ images) or compulsions (behaviours) or both.

The OCD loop

The role of the striatum has been mentioned in this loop. The function of the striatum is known to be its involvement in movement – it enables us to carry out basic automatic behaviours (like grooming) without having to focus on the activity. Eventually this enables us to carry out multiple tasks while focussing on higher cognitive or complex activities – (driving a car, listening to the radio, changing gears and talking to the passenger).

OCD is considered to be the result of a neural pattern that is not effectively regulated resulting in ongoing activation of the OFC and striatum (like the sensor of the smoke alarm that sets off too easily). The role of the striatum is considered to be so vital in understanding OCD that some researchers suggested that it should not be classified as an anxiety disorder but a movement disorder however the link with the amygdala and fear response still validates the classification under the anxiety disorders (Lambert & Kinsley 2011; LeDoux 2003).

When the OCD loop is established the “activation” can happen at either end – it can initiate from a sensory input – an actual event activated through the thalamus (“I need to lock the car”; “I need to make sure it is locked and lock it again and again”) or a thought/image (“did I lock the car?”; “I need to go back and check”).

OCD

is not only linked to neural activation patterns in the OFC-striatum-amygdala-thalamus loop. There is also evidence of neurochemical indicators.

The neurochemistry of OCD

OCD is not only linked to neural activation patterns in the OFC-striatum-amygdala-thalamus loop. There is also evidence of neurochemical indicators. A number of neurotransmitters and neuromodulators have been investigated in relation to OCD. On some levels, OCD can be viewed as a “behavioural addiction”. Rats with increased cellular activity in the nucleus accumbens demonstrate increased Dopaminergic activity (linked with pleasure reward but also with addiction) and decreased GABA activity (linked with relaxation and toning down of Dopaminergic action).

Assisting the brain to experience increased GABA activity linked with reward (Dopamine release) is one of the vital goals in addressing OCD. Enhancing Dopamine release through high performance activities (eg. Exercise), increase the adrenalin endorphin levels and does not contribute to down regulate neural activity linked to OCD. This does not mean physical exercise is not beneficial however it seems exercise should not be the therapeutic focus to manage OCD.

Oxytocin is a neuropeptide that is released by the posterior pituitary and is released with breast-feeding and social bonding. This peptide plays a vital role in attachment and safety and forms a vital component in neural development – especially during the first 10 months after birth. Oxytocin seems to enhance the expression of grooming, reproductive and affiliative behaviours.

Oxytocin is also linked with emotional and physical safety. The absence of these factors lead to decrease of Oxytocin production, up regulation of the amygdala activity, increased thalamus scanning of the environment for cues of threat and enhancement of the activation of the OCD loop.

Treatment for OCD

Psychopharmacological treatment was, for many years, the treatment of choice. SSRIs (selective serotonin reuptake inhibitors)

were used with mixed success. Most studies indicate that SSRIs may have an indirect effect on OCD (Dolberg et. al. 1996; Eineberg & Gale 2005) however long term neural changes have not been demonstrated.

In more extreme cases, neurosurgery has been introduced.

- Anterior cingulotomy was originally performed in 1952. In this procedure the anterior portion of the cingulate gyrus is excised. The success rate was around 50% with long term effect of 30% (Jenike et.al 1996).
- Subcaudate tractotomy is a procedure in which lesions are made in the orbito frontal cortex. Clinical improvement was seen in around 50% of patients (Goktepe, Young & Bridges 1975).
- Limbic leucotomy is a procedure of bilateral lesions introduced to the lower medial portion of the orbito-frontal areas and bilateral lesions of the anterior cingulate area. Success rates are as high as 84% with no long term side effects (Perse 1988).
- Deep brain stimulation. This procedure,

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especially used in treatment of Parkinson’s disease consists of a battery operated neurostimulator that is implanted in the subclavicular area allowing for continual stimulation of the brain. Limited information is available regarding the efficacy of this intervention (Denys & Mantione 2009).

Psychotherapy

A variety of psychotherapeutic interventions were used over the course of the last

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100 years. While the psychoanalytical approach focussed on the meaning of the obsessions and compulsions the behavioural approaches were more directly focused on treating the actions. The Exposure and Response Prevention (ERP) proved to be the first empirically validated successful treatment (Kobak et.al 1998). Patients were instructed to expose themselves to feared stimuli and encouraged to stay in contact with the stimuli and not to avoid the stimuli. Due to this approach (facing the fear head on) a significant number of clients tend to drop out during the program (Marks 1997).

Cognitive therapy focuses on the cognitive aspect rather the behavioural aspect of the OCD loop. The key is on developing an "anxiety profile" and "facing the fear" (Clark & Beck 2012) and assisting the client to establish a sense of cognitive control.

Neuropsychotherapy

The challenge with managing OCD is the prevalence of the OCD loop in terms of neural activity and the neuromodulators (the key role players being Oxytocin, Serotonin, GABA and Dopamine). Both these systems play a vital role in maintaining a level of congruence and control (albeit dysfunctional). As the OCD loop links closely with primitive systems in the brain evolution, cognitive interventions may not be that successful unless these systems are taken into account.

This means that concepts like "facing the fear" may have a counter productive result when the client is not provided with a warm safe and secure environment. Well established therapeutic rapport and empathetic understanding is vital for down regulation of limbic activity, ensuring a decrease of stress hormones that excite the OCD loop.

A safe, trusting relationship also encour-

ages oxytocin and GABA reactions resulting in more effective cortical blood flow to the frontal cortical regions and up regulating effective cognitive patterns (an important reason why cognitive therapy is at least preferred to behavioural therapy).

A neuropsychotherapeutic approach takes into consideration the "fact" that the OCD loop is well established and neuromodulators and stress hormones feed the OCD pattern. As a result the therapeutic alliance need to be established and new neural connections introduced.

This means learning to relax (feeling good - GABA linked with Dopamine) needs to be introduced to the neural pathways and then a hierarchy of control (slow exposure to the OCD loop) developed whilst keeping the stress response down regulated. This is a balance of neural excitability (changing synaptic connections towards new neural pathways) and synaptic inhibition (less activation of default – unhelpful patterns)



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(Grawe 2007).

This process is enhanced with cognitive activities such as challenging unhelpful thoughts and images. The clinician needs to be mindful of demonstrations of distress and assist the client to identify this as: "my OCD loop is kicking in – what do I need to do to shift the neurons in my brain to activate the new pattern rather than the old pattern".

Eventually the goal of Neuropsychotherapy for OCD is to shift the synaptic strength of the OCD loop toward a new, well established neural pathway that links more effectively with the left prefrontal cortical region to problem solve more effectively and integrate high levels of controllable congruence in all situations.

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Dr Pieter Rossouw

specialises in Neuropsychotherapy in Australia and is an expert in anxiety and mood disorders. He has published five scientific books and over twenty scientific articles. He has been involved in research in extensive clinical trials and presented research papers at thirty international conferences worldwide. He is on the Advisory Board of The Neuropsychotherapist.

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Australian Neuropsychotherapy Workshops

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Pieter is the Director of the Master of Counselling Program at the School of Psychology and the School of Social Work and Human Services at The University of Queensland, Australia. His research and teaching focuses on Neuropsychotherapy. Pieter is also the Director of Mediros – a company that provides training in Neurobiology and Neuropsychotherapy.

Pieter has established a distinguished career as Clinical Psychologist, Lecturer, Clinical Consultant and Supervisor. He has been in Private Practice for the past 25 years. Pieter holds Honours Degrees in Philosophy and Psychology, a Master Degree in Clinical Psychology and a PhD. Pieter is a member of the Australian Psychological Society and the APS College of Clinical Psychologists. He provides Mental Health training for GP's and is accredited at the Royal Australian College of General Practitioners. In this role he developed and facilitated a Clinical Audit for General Practitioners (30 PD point activity) with over 600 GP's Nationwide involved in the training.

Before relocating to Australia, Pieter was a Professor in Clinical Psychology for 11 years. He was a guest lecturer at Universities in Canada, Holland and South Africa where he also spearheaded a Psycho-Therapeutic Assistance Program to support people being exposed to trauma. In Sydney he worked as Senior Clinical Psychologist at the Northern Beaches Adolescent Service – Department of Health, he was the Clinical Director of the St John of God Psychiatric Hospitals – both Richmond and Burwood Hospitals as well as worked in Private Practice. He provided clinical supervision to many Masters and PhD students as Clinical Associate of the Universities of Sydney, New South Wales, Western Sydney, Macquarie, Wollongong and Newcastle. Currently he is involved in full time research in the fields of neurobiology and neuropsychotherapy as well as clinical training for clinicians, psychologists and general practitioners.

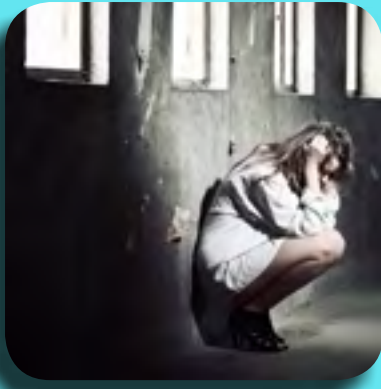
Pieter specialises in neuropsychotherapy and is an expert in anxiety and mood disorders. He has published 5 Scientific Books and 20 scientific articles. He has been involved in research in extensive clinical trials and presented research papers at 30 International Conferences worldwide.

He is a member of the Global Association for Interpersonal Neurobiology Studies, the International Society for Traumatic Stress Studies, the International Association for Family Therapy and the Professional Association for Drug and Alcohol Workers. He also facilitates a Global Neuropsychotherapeutic Interest Group through its e-Journal (Neuropsychotherapy in Australia) and local specialist research and discussion groups. Currently it has over 2500 active members – comprising of clinicians and academics in the field of neuroscience.



Pieter developed three 2-day APS, specialist endorsed workshops – The Brain and Anxiety: Utilizing Neurobiological Information as Psychotherapeutic Tool; The Neuroscience of Depression: New Opportunities for Effective Treatment; and The Developing Brain and the Neuroscience of Memory and Trauma. He also runs neuropsychotherapy applied skills classes.

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The Developing Brain and the Neuroscience of Memory & Trauma

Implications for effective skills based interventions

The psychological and neurobiological effects of trauma have significant implications for well being. Theoretical and treatment modalities for trauma have been the focus of study for many researchers. Recent discoveries in neurobiology have changed the landscape of theory and treatment of Psychological Trauma. These discoveries assisted with our understanding of neural processes, memory and neural communication. Clarity about these concepts assists clinicians towards more effective interventions with clients suffering from the aftermath of trauma.



The Brain and Anxiety

Utilizing neurobiological information as a psychotherapeutic tool

Anxiety is a prevalent problem among Australians. Over one quarter of adults suffer from anxiety in any given year. The last decade of brain research made possible by fascinating advances in brain imaging and neurobiological data has moved the understanding of anxiety disorders into a new dimension. Although we can successfully treat clients without knowing the full implication of research, we can be more effective with more people in less time if we have a grasp of the neurobiology and why and how our treatment methods change brain function



The Neuroscience of Depression

New opportunities for effective treatment

Depression is a common disorder without geographic, educational, socioeconomic, or racial boundaries. Recent advances in neuroscience provided new dimensions to the understanding and treatment of depression. Discoveries in the association of depression with neural plasticity and neurogenesis as well as insight in the role of talking therapies to change neural functioning as well as neural structure opened fascinating new perspectives and treatment options.



Neuropsychotherapy for Anxiety Neuropsychotherapy for Depression

Applied strategies for treatment

These workshops look at recent advances and research into anxiety and depression and the implications for therapeutic interventions.