THE NEUROPSYCHOTHERAPIST

THE NEUROBIOLOGY OF ADDICTION

IMPLICATIONS FOR THERAPEUTIC INTERVENTION

BY

Pieter Rossouw
In the sweltering heat of the summer of 1997, Army Sargent Gail Baker drove to a convenience store in Jasper County, South Carolina, and commenced a 7-hour binge of video poker. When Gail finally returned to her steaming car, she discovered that her 10-day old baby, whom she had left in the car, had not survived her video poker ordeal.

Her husband later argued that he did not hold her responsible for the death of their child because she had an addiction.
Addiction and the brain

The link between addiction and the brain took a huge step forward when researchers James Olds and Peter Milner studied the effects of electrical stimulation in the brain stem of rats. They found that rats respond positively to electrical stimulation. The experimental design was setup in such a way that rats could deliver their own stimulation by pressing a bar. They found the rats could not get enough stimulation and they would continue to press the bar up to 2000 times per hour until some of them passed out from exhaustion. On autopsies of the brains they found that they accidentally implanted the electrodes not in the brain stem but in the medial forebrain bundle – a huge collection of axons that link the hypothalamus with the septum – an area these days known as the nucleus accumbens (NAc) (Olds, Milner 1952; Olds 1958). More recently researchers have clearly demonstrated the role of areas like the nucleus accumbens in the reward process (Robbins, Everitt 1999; Everitt et.al 2000). When the nucleus accumbens is traumatised (physically damaged) rats stop repetitive stimulation of a rewarding drug (such as cocaine).

The nucleus accumbens does not work in isolation; the neural networks and links with other areas of the brain, as well as neurochemical shifts, are crucial in understanding addiction and provide clinicians with important guidelines towards effective treatment strategies. At least two other brain areas need to be explored in relation to addiction; the orbito-frontal cortex and the amygdala. Additionally the key neurochemical Dopa- mine, and its brain function, should also be discussed.

The Nucleus Accumbens

The role of the nucleus accumbens is to determine whether a response is important enough to repeat. It receives signals from the amygdala, hippocampus and medial prefrontal cortex. Some of those areas are also linked with the stress response (like the amygdala that triggers the hypothalamus stress response). Researchers refer to the nucleus accumbens as the brain’s “sensory-motor interface”, the location of the interaction between external stimuli and the coordination of a physical response (Lambert, Kinsley 2011). The implication is that the nucleus accumbens plays a crucial role in initiating and maintaining motivation toward specific stimuli.

As soon as certain variables are associated with the experience of reward, the nucleus accumbens “saves” it to the brain’s long-term memory store, increasing the likelihood of that same action being repeated. This process becomes evident in avoidance behaviour (link with reward when actions are avoided) or gaining rewards (link with reward when actions are repeated).

The avoidance behaviour pattern explains partly why it is difficult to change anxiety patterns like trauma, obsessive compulsive disorder, generalised anxiety, etc. The role of the reward system in avoiding stimuli...
associated with trauma, repetitive actions, constant worrying, etc., plays a significant role in maintaining (unhelpful) default neural firing patterns, resulting in a “resistance” to change.

The gaining rewards pattern, on the other hand, also explains partly why addictive patterns are established as well as why they are difficult to shift. As soon a strong reward pattern takes hold in the nucleus accumbens, an adaptive learning system kicks in that is strongly influenced by the activity in the nucleus accumbens.

The Nucleus Accumbens and Gambling

In the 1900’s two literature professors of Mississippi became addicted to gambling. Their recording of the process illustrates how “bad” gambling was processed as threatening and “good” gambling was processed as rewards. For example, they associated bad luck (bad gambling) with the number of ice cubes in the drink and good luck (good gambling) with certain songs. They ended with an abundance of associations (in neurobiological terms “triggers”) to stimulate the nucleus accumbens to continue the activity (Barthelme & Barthelme 1999). Experiments with rats indicated that when this reward circuitry is well established the rat will endure huge discomfort (severe shocks) to continue the experience of pleasure (the reward in the nucleus accumbens).

“Where a keg of rum in one corner of the room, and were a cannon discharging balls between me and it, I could not refrain from passing before that cannon, in order to get at the rum” (Elster 1999)

**The Orbitofrontal cortex**

The role of the orbitofrontal cortex (OFC) in maintaining patterns of behaviour is well established (Rossouw 2012b). From a neurophysiological perspective the link between the orbitofrontal cortex and the nucleus accumbens is very close. The NAc receives messages from the amygdala and hippocampus and projects signals into the OFC. The OFC also receives messages from the amygdala and the hippocampus. In return it also sends signals back to the NAc. A growing body of evidence is pointing toward the OFC as a specialised extension of the NAc. Where the NAc is the centre of the reward system, the OFC (in conjunction with the hippocampus) applies significance and context to the experience and assist with the maintenance (continuation) of the reward (obsessions, compulsions, addictions).

**Dopamine**

The quest for understanding addiction has not only a neuro-structural explanation but also a neuro-chemical one. There are key neuromodulators that play a role in addiction like dopamine, serotonin and acetylcholine. Of these modulators, dopamine (DA) seems to play the most significant role in addiction (Caine et al. 2007).

With a technique called microdialysis, researchers have found that addictive behaviours lead to increased levels of Dopamine in the NAC (Chiara et al. 2004). Most substances of abuse (like cocaine) increase DA availability in the synapse by inhibiting the dopamine transporter. But there is another
twist to understanding addiction. Imaging studies show that the psychological high, experienced by people with an addiction, is also associated with a gradual reduction in the number of actual DA receptors in the NAc and OFC. The implication is that addicts gradually desensitise to the previous “high”, and as result they need to increase both the dose and the frequency to counter the loss of DA receptors (the addictive pattern).

Stress and Addiction

Does stress increase the risk of addiction? Research indicates that the stress response kicks in when the hypothalamus is triggered from the amygdala in relation to perceived threat. This activates the release of a number of stress hormones in the hypothalamus-pituitary-adrenal axis: corticotropin-releasing hormone (CRF), adrenocorticotropic hormone (ACTH), adrenalin and cortisol. As soon as the stress is effectively dealt with, cortisol signals the hypothalamus to discontinue the stress signals and the stress response discontinues. This leads to a sense of control which activates the release of dopamine (reward).

When a person is exposed to stress, the brain seeks to resolve the stress, and the end result of successful stress management is control and release of DA. The “shortcut” route (pathology) is to find immediate DA release to experience the positive outcome (the stress is over). Unhelpful stress management is the shortcut rout to activate DA release as quickly as possible, and so emerges an addictive pattern in the presence of stress (Al Absi 2007). We can thus say:

- People faced with stress are more prone to addiction or
- Stress increases the risk of addiction or
- Addiction increases stress but that leads to intensifying the addiction.

Therapeutic interventions for addictions

In light of the neurobiological aspects of addiction briefly discussed, the question arises: what are key indicators for therapeutic interventions?
The bottom-up approach

The neurobiological basis of addictions highlight the neuro-chemical and neuro-structural changes associated with addictions. These changes cause a fundamental shift in brain functioning, suggesting that we should first address neurophysiology – the bottom up approach (Rossouw 2012a). A person with a significant cocaine or heroin addiction does not have the neural activation capacity to effectively stop taking the drug or activate cognitive capabilities to understand and address the addiction, let alone the ability to down regulate the stress response when it is suggested that the drug should be discontinued.

Does the same apply to people with behavioural addictions (gambling, pathological spending etc.)? The answer to this question is probably: “no” and “yes”. “No” as there is no substance abuse that intensifies the chemical and neurological imbalance however “yes” as the “behavioural” addictions are just as deeply rooted in neurochemical changes and patterns. On this level there are significant similarities between all forms of addiction.

1. Detoxification

With substance abuse addiction the first step is detoxification. The intake of chemicals that sustain the addiction need to be addressed first. The detoxification program needs to be planned depending on the substance that is associated with the addiction. In more serious cases hospitalization with opiate agonists, like methadone or naltrexone, would be considered. This can be a slow process as the addictive nature of the medication used in the detoxification process (methadone, naltrexone, benzodiazepines) also need to be tapered and “detoxed”.

2. Safety

The up regulation of stress (a sense of not being in control) enhances the stress response and increases the risk of the default pattern – the addictive behaviour. Physical and emotional safety is paramount in the treatment process. Being in a safe environment, establishing a strong supportive therapeutic rapport is essential in down regulating the amygdala – HPA system and facilitates the ability for the brain to establish new neural pathways.

3. Stress and environmental cues

As discussed above, stress and addiction are closely linked. The person struggling with an addiction have often established a network of “stressors” that trigger the need for Dopamine as a release, and addictive behaviours giving brief gratification. A clear understanding of both the social and psychological patterns and systems, can assist the person with an addiction to take control, make choices, and activate new patterns. These initiatives activate new neural patterns and eventually a sense of reward (effective DA release associated with functional neural patterns - gradually diminishing default patterns).

4. Cognitive Control

As the brain becomes more readily able to activate cortical blood flow to the frontal cortical regions, cognitive interventions become more effective. Understanding the role of cognitions, the effect of cognitive therapies to take control, and facilitating neuro-chemical release as a result of effective thinking and behavioural patterns form the powerful link to long term neural facilitation towards healing and gradually decreases the risk of utilizing the default patterns to gratification (pathological route to DA release).
Once these patterns are well established the default patterns in the OFC loses its stronghold, due to the lack of activity in the default firing, which in turns lead to less myelination (glial activity). The old pattern becomes structurally weaker (less craving and less risk of relapse).

References:

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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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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Go to www.mediros.com.au for information on Australian Neuropsychotherapy Workshops
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
*Applied strategies for treatment*

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