Welcome to the July/August edition of *Neuropsychotherapy in Australia*.

This is our second edition of the new format of the journal. Thank you to the many readers who provided feedback in regards to the Journal. We are encouraged to see a continuing growth in readership (now exceeding 2000).

**APS Neuropsychotherapy Interest Group.**

As a result of growing interest and discussions with peers, some of our colleagues in South Australia have initiated the process to apply to the APS for the establishment of a “Neuropsychotherapy Interest Group”. This will provide an additional important forum for the promotion of Neuropsychotherapy. The focus of this proposed Interest Group will be:

- To promote the application of neuroscience research and theory to the practice of psychotherapy;
- To promote theoretical developments, research and practice related to Neuropsychotherapy;
- To promote research into the outcomes of applying Neuroscientific knowledge to psychotherapy;
- To provide professional development opportunities in the area of neuroscience and its application to psychotherapeutic practice and relevance to the science of behaviour change;
- To provide forums for discussion, information sharing and professional support;
- To facilitate a network within the APS for members with an interest in neuropsychotherapy;
- To promote the field of neuropsychotherapy, through links with other professional groups whose aims are congruent with those of the Interest Group and in accordance with those of the APS;
- To provide informed advice to the APS on issues relating to the specialist area of neuropsychotherapy.

- To promote the theory and practice of Neuroscientific information in a variety of psychotherapeutic settings.

Your support to assist with this process will be greatly appreciated. To support this application you can fill out the APS signature form (last page of the Journal). Please mail the completed form to

**Mediros (Admin)**

PO Box 6460

St Lucia

Qld, 4067

**Neuropsychotherapy workshops**

We are progressing well with our workshop schedule for 2012. Two of the workshop series: *The Brain and Anxiety – Utilizing Neurobiological Information as Psychotherapeutic Tool*; and *The Neuroscience of Depression - New Opportunities for Effective Treatment*, have been completed in all major centres (Sydney, Melbourne, Brisbane, Perth, Adelaide, Canberra and Hobart). The first of the one-day applied skills workshops are nearly completed.

In the coming months the third of our 2-day workshop series will roll out. This workshop, *The Developing Brain and the Neuroscience of Memory and Trauma*, will have a different focus than the previous workshops and focuses on the Neuroscientific markers of brain development as well as the principles of memory, from a cellular level, to the significant implications of these principles when working with clients to change the brain.

From September onwards the second of the very popular 1-day applied skills workshops will also roll out. This workshop focuses on the applications (skills based – case studies) of the Neuroscientific underpinnings of depression and the treatment guidelines. Numbers are limited as these workshops are small group, round table, interactive workshops.

More information about the workshops is included in this Journal.
Childhood Trauma

In this edition we focus on Childhood trauma. The effects of early exposure to trauma can have significant effects on neural development. In this paper we explore some aspects of healthy neural development and demonstrate the effects of violations of basic needs in relation to neural changes. The effects of these violations are considered in terms of neurostructural changes as well as neurochemical shifts. Attention is paid to the implications for psychotherapy – the need for early interventions to prevent the development of strong neural patterns of avoidance (protective neural loops that inhibit open neural sprouting) and strategies for facilitating an effective psychotherapeutic process.

This paper was recently delivered at the Australasian Conference of Child Trauma (July 2012 - Gold Coast).

The developing brain

The neurologist Paul MacLean identified three prominent phases in the development of the human brain (Maclean 1990). Much of this development occurs in the pre-natal phase. However the key role of the interaction between the brain (nature) and its environment (nurture) – genetic expression - has been well documented through significant research findings.

MacLean identified the “reptilian brain” as the first stage of brain development. The key areas that can be identified in this phase are the brain stem, pons and cerebellum. These structures are linked with key aspects of the survival mechanisms of an organism – like regulating breathing and heart rate. Whilst development of these structures continues and become more refined, a second phase of brain development emerges - the mammalian brain. Structures situated deep in the brain, and sitting on top of the brainstem and below the corpus callosum, develop at a rapid pace. These structures include the thalamus, hypothalamus, amygdala and hippocampus – a cluster that MacLean called the Limbic System. Research indicates that these structures play a vital role in processing sensory information, developing the implicit memory reaction to stress, short term memory and the first line of emotion regulation. Powerful memory systems are developed in the early stages of
development (especially the first ten months post birth) through these structures. Lastly the cortical areas, the neo cortex, or in MacLean’s terminology the “paleomammalian brain” emerges. These areas form the cognitive, emotional and motor powerhouse of neural functioning. Executive reasoning, emotional control and integration of responses are facilitated in these areas.

This information is vital to understand the impact of violation of basic developmental tasks and needs on the developing brain. This information is even more significant to identify effective strategies to manage/treat victims of childhood trauma (Rossouw 2011a).

Children are born with a fully developed “reptilian” brain. The ability to maintain basic survival function like regulating breathing and heart rate are essential to basic existence. Violation of these abilities may have serious implications to sustain life.

With the survival part of the neural functioning intact, the second “layer” of brain functioning comes into play. At birth, newborns have fully functional limbic structures however the operational ability still needs to develop. Studies with macaque monkeys by Shen and Battersby indicated how high risk genetic factors (two short serotonin transporter gene 5HT-alleles) never expressed in the group that was exposed to enriched safe environments. The genetic risk only expressed in the group exposed to adverse conditions -less safe and poorly enriched environments (Shen & Battersby 2002).

**Violation of basic needs**

From birth, limbic structures (especially the amygdali) of newborns constantly scans the environment for cues of risk, discomfort or danger and need to be down-regulated by fulfilment of basic needs – safe environments: demonstrated through secure attachment and control. Fulfilment of these basic needs down-regulates the need for constant scanning of the environment for danger and allows healthy neural development of the neo cortex. Research clearly indicates that neural growth is enhanced through enriched environments. In order to maximise effective neural growth, sprouting and effective neural pruning, key operational needs must be effectively fulfilled.

These needs are:

- The need for secure attachment
- The need for control
- The need for self-esteem enhancement and self-esteem protection and
- The need for pleasure maximization and distress avoidance.

Safe proximity of the primary carer, normally the mother, provides ongoing down-regulation of the stress response and up-regulation of neural sprouting into the neo cortex. This leads to effective myelination and maturation of neural connections in open firing patterns essential for effective neural development. Implicit and explicit memory systems are facilitated that enhance neural proliferation, plasticity and pruning (Grawe 2007, Rudy 2008). A recent study by Luby and colleagues indicated the link between secure attachment and larger hippocampal volumes in children at school age (Luby et.al. 2012). Van der Kolk proposes a new disorder for the Diagnostic and Statistical manual of Mental Disorders (fifth edition) of a Developmental Trauma Disorder – a disorder to be diagnosed in children with complex trauma histories.

Violation of basic needs – excessive emotional discomfort, pain, bullying, and the absence of essential nutrients - has severe consequences on the developing brain. Early childhood trauma can cause havoc in neural development (Waters et.al. 2000, Rossouw 2012). This happens as a result of up-regulation of the fear
response causing neurochemical and neurostructural changes.

**The stress response**

On the neurochemical level increased activation of the HPA system (hypothalamus-pituitary-adrenal system), facilitates significant increases in the production of CRF (corticotrophin release factor), ACTH (adrenocorticotropic hormone) and adrenalin and corticosteroids (among them cortisol) (Wehrenberg and Prinz 2007). Normal stimulation of the HPA system is important as it acts as the first line defense against external triggers of threat to push the body into hyper-alertness (De Bellis 2003).

The role of cortisol is, among others to deactivate the hypothalamus to discontinue the stress activation. Ongoing exposure to perceived threat over activates the HPA system leading to increased production of stress hormones. Overproduction of cortisol can lead to a condition called hypercortisolemia – a destructive process that results in the death of glia and neurons, and a related decrease in the volume of neural areas (atrophy). Areas that tend to be affected by this process are the left prefrontal cortex, orbitofrontal cortex, anterior cingulate, and sections of the limbic system – especially the hippocampus. Excessive cortisol secretion and chronically elevated cortisol levels can lead to a host of related metabolic disturbances and an increased risk for developing a variety of chronic conditions (Carrion et.al. 2002).

These conditions include:

- Elevated cholesterol and triglyceride levels - heart disease
- Elevated blood pressure - heart disease
- Alterations in brain neurochemistry (involving dopamine and serotonin) - depression/anxiety
- Physical atrophy (shrinkage) of brain cells – key neural areas
- Insulin resistance and elevated blood-sugar levels - diabetes
- Accelerated bone resorption (breakdown) - osteoporosis
- Reduced levels of testosterone and estrogen - suppressed libido (reduced sex drive)
- Suppression of immune-cell number and activity - frequent colds/flu/infection
- Reduced synthesis of brain neurotransmitters - memory/concentration problems, neural sprouting.

Increased cortisol levels are typically found in traumatized children. For example higher levels of cortisol were found in sexually abused girls (ages 6-15 years) within 6 months of disclosure as compared to non-abused socio-demographically matched controls (Putnam et al 1991). Similarly Gunnar and colleagues (2001) showed elevated cortisol in 6-12 year old children raised in Romanian orphanages for more than 8 months as compared to adopted children. Elevated levels of cortisol have been described in maltreated children with depression (Carrion et. al. 2002).

**Neurostructural changes**

Pituitary volumes of maltreated subjects were significantly larger among maltreated subjects as compared to a normal population (Thomas & De Bellis 2004). This indicates excessive CRF production – linked with survival stress, destabilizing limbic activation and neural
development. Studies by Baker and colleagues show that sexually abused girls manifested a disregulatory disorder of the HPA system associated with hypo-responsiveness of the pituitary to CRH. It seems that hyper secretion led to an adoptive down regulation of CRF receptors which is similar to the mechanisms of adult PTSD (Van der Kolk 2004).

It is hypothesized that in posttraumatic stress disorder (PTSD) the HPA system has become maladaptive causing long term negative consequences. Triggers of stress (internal or external) are over-amplified resulting in over-activation of the HPA system. Due to this over-activation and the long term detrimental effect of stress hormones on neural structures, the ability to down regulate continues to reduce over time (Soderlund et al. 2011). This process is closely linked to hippocampal volume loss, ineffective synaptogenesis (neural sprouting) and the formation of neural loops (MacMaster & Kusumakar 2004). In adults, PTSD is closely associated with smaller hippocampi or hippocampal volume loss/atrophy (Sapolski 2000). In children, however, trauma is not associated with hippocampal volume loss (Carrion et al. 2001; De Bellis et al. 2002). This is an important neurophysiological difference between adult and child PTSD.

The hippocampus is generally seen as the powerhouse for neural plasticity and neurogenesis (due to the production of the protein brain derived neurotrophic factor (BDNF). It seems that this very fact that safeguards the hippocampus from atrophy (for the child at least). However continuing decrease of this function increases the hippocampal vulnerability and lowers the resilience to manage ongoing cortisol release. Eventually atrophy sets in. The introduction of chemicals (high fat diets, high refined sugar intake and ETOH – alcohol) decreases BDNF production and neural plasticity and enhances neural rigidity and so these are also contributing factors to increase detrimental neurobiological indicators as result of trauma (Rudy 2008).

The developing brain shows robust resilience to trauma however intensity and duration of exposure to distress are key role players to inhibit effective neural maturation (Sapolski 2000), myelination (Dunlop et al. 1997), production of BDNF proteins (Rudy 2008), neural pruning (Lander 1988), synaptogenesis (Rudy 2008) and neurogenesis (Sporns 2011). Myelinated areas of the brain are particularly susceptible to the effects of early exposure to significant levels of stress hormones. Since the early study of Teicher and colleagues (1990), fMRI studies confirmed the inhibition of development of the corpus callosum linked with early childhood trauma. Non-effective development of the corpus callosum has a significant effect on the development of the effective neural connections that regulate mood and cognitions (Sporns 2011, De Bellis et al. 2005). One of the consequences of this process eventually is the inability to cognitively down regulate distress – the result of implicit memory loops.

**Indicators for interventions**

What are the lessons that we learn from neurobiology research in terms of childhood trauma?

- **In terms of prevention:**

The developing brain is robust and resilient but is also highly susceptible to violations of basic needs. These violations effect the neural development and direction of neural connections and eventually brain structure. Gene expressions happen as a result of ongoing interactions with the environment. Risk factors need to be clearly identified and addressed. Down regulation of limbic alertness (emotional, physical and cognitive) safety is paramount for effective neural sprouting. The concept of controllable congruence - as opposed to
incontrollable incongruence (trauma), should be the guiding principle (Grawe 2007).

**In terms of intervention:**

Although there are many theoretical models for childhood interventions, neurobiological evidence show that the overlap between intervention models is much bigger than the differences between models. The key principles for effective interventions point towards early intervention, limbic down regulation, neurochemical balance and enriched environments to facilitate effective change.

The brain is a dynamic, plastic entity that continues to grow and change. It has an amazing ability to compensate for areas negatively affected and to heal itself. This “healing” can be functional or dysfunctional. Proper facilitation of enriched environments facilitates effective neural connection, and activation leading to safe situations of controllable incongruence and deconstruction of emerging unhelpful default neural patterns. Introduction of early interventions decreases the risk of powerful default neural patterns and can facilitate lasting changes in a shorter time with less risk of returning to default neural patterns (relapse).

Limbic down-regulation is paramount. The establishment of a secure therapeutic relationship down-regulates the limbic alertness and stress response, and enhances neural activation to frontal cortical structures. This is essential to facilitate neural change. Without a safe, supportive therapeutic relationship any changes that are facilitated will be solely cosmetic.

Neurochemical balance is needed to enhance neural change. Chemicals introduced to the system play a significant role in helpful or unhelpful neural activity. High intake of caffeine, alcohol, tobacco, sweeteners, and high fat diets affect production of key neurotransmitters, elevate blood sugar levels, inhibit neural plasticity and neurogenesis. Healthy intake of chemicals enhances therapeutic interventions.

Sleep and exercise play a vital role in mental health and even more so in recovery. Without effective hippocampal discharge (quality sleep) and regular cortisol burning (exercise), the brain is at risk of strengthening unhelpful neural patterns rather than developing open neural activation patterns as well as becoming more rigid due to the negative effect on synaptic connections and neural strength (Lambert & Kinsley 2011).

**Healing trauma**

The good news is that neurochemical and more importantly neurostructural changes due to trauma are not necessarily permanent. Many neuro-imaging studies indicate effective neural changes due to psychotherapeutic interventions. Nobel laureate in Medicine, Eric Kandel refers to the effectiveness of talking therapies, to facilitate neurostructural change as the remarkable scientific revolution that is transforming the way mental health services are provided (Kandel 1998, 2006, Davidson & Begley 2012). This remarkable scientific revolution is indeed happening, and clear neurobiological evidence demonstrates the power of talking interventions to facilitate effective changes in the brains of traumatized children (Rossouw 2011b, Siegel 2010, Siegel & Branson 2011, and Murphy & Mathews 2010).

**Literature**


“What lies behind us and what lies ahead of us are tiny matters compared to what lives within us.”
— Henry David Thoreau
BOOK REVIEW

POCKET GUIDE to INTERPERSONAL NEUROBIOLOGY
an integrative handbook of the mind
Norton & Company. NY.

This new publication from the editor of the Norton Series on Interpersonal Neurobiology, Interpersonal Neurobiology; an integrative handbook of the mind by Dan Siegel (2012) focus on the following issues: What is the mind? What makes a healthy mind? How do we become aware and come to know about life? And perhaps most importantly, what are the connections among the mind, brain, and relationships?

From psychologists to linguists, neuroscientists to philosophers, people have explored the nature of mental life, yet no interdisciplinary framework has existed for wisely answering these fundamental questions or even offering a definition of what the mind is.

In this book Siegel bridges domains of knowledge to offer a book that reveals the way the mind works via a format that reflects the brain’s natural mode of learning (flip the Pocket Guide open to any page and you will find an “entry point” that guides you to explore, in your own way, the web of integrated knowledge).

Walking us through the intricate foundations of interpersonal neurobiology, Siegel allows us to see the personal and professional applications of this exciting new approach to developing a healthy mind, an integrated brain, and empathic relationships.

Daniel J. Siegel, MD, is a graduate of Harvard Medical School, Clinical Professor at UCLA School of Medicine, Co-Director of the UCLA Mindful Awareness Research Centre, and Executive Director of the Mindsight Institute. He is an internationally-acclaimed writer and speaker, and founding editor of the Norton Series on Interpersonal Neurobiology.

--- Aristotle

There is a foolish corner in the brain of the wisest man.
Two Day Workshops

The Brain and Anxiety – Utilizing Neurobiological Information as Psychotherapeutic Tool
APS Endorsed 12 CPD hours (CCLIN, CCOUN, CCOM)
- 28,29 April 2012 – Sydney
- 20,21 April – Melbourne
- 1,2 June – Brisbane

The Neuroscience of Depression – New Opportunities for Effective Treatment
APS Endorsed 12 CPD hours (CCLIN, CCOUN, CCOM)
- 11,12 May 2012 – Perth
- 18,19 May 2012 – Canberra
- 25,26 May 2012 – Adelaide
- 15,16 June 2012 – Melbourne
- 22,23 June 2012 – Sydney
- 6,7 July 2012 – Brisbane
- 13,14 July 2012 – Hobart

The Developing Brain and the Neuroscience of Memory and Trauma. Implications for Effective Skills Based Interventions.
APS Endorsed 12 CPD hours (CCLIN, CCOUN, CCOM)
- 7,8 September 2012 – Melbourne
- 14,15 September 2012 – Brisbane
- 21,22 September 2012 – Perth
- 12,13 October 2012 – Adelaide
- 26,27 October 2012 – Canberra
- 2,3 November 2012 – Sydney
- 9,10 November 2012 – Hobart

One-Day Skills workshops

Focused Neuropsychotherapy – Applied strategies for the treatment of Anxiety – skills based training
(1 day round table class – case demonstrations, discussions and interactive learning) (limited spaces)
APS Endorsed—6 CPD Hours (CCLIN, CCOUN, CCOM)
- 24 March 2012 – Brisbane
- 14 April 2012 – Adelaide
- 2 May 2012 – Melbourne
- 30 June 2012 – Perth
- 21 July 2012 – Sydney
- 1 September 2012 – Canberra

Focused Neuropsychotherapy – Applied Strategies for the treatment of Depression – skills based training
(1 day round table class – case demonstrations, discussions and interactive learning) (limited spaces) 6 Learning hours
- 18 August 2012 - Adelaide
- 27 September 2012 - Sydney
- 5 October 2012 – Hobart
- 17 November 2012 – Melbourne
- 1 December 2012 – Canberra
- 8 December 2012 – Brisbane
- 15 December 2012 – Perth
Signature Form
In support of the formation of a new Interest Group

I ________________________________ (full name)
Of ________________________________ (home or work address)
____________________________________
____________________________________
am interested in the proposal to set up an APS interest group on
_______________________________ and Psychology

Signed: ________________________________

My contact details are______________________ (phone)
______________________________ (email)
______________________________ (APS Member No)

Please post the signed original copy of this form to:

APS Member Groups Administrator
PO Box 38
Flinders Lane
Melbourne VIC 8009