THE END OF THE MEDICAL MODEL?

RECENT FINDINGS IN NEUROSCIENCE REGARDING ANTIDEPRESSANT MEDICATION:

IMPLICATIONS FOR NEUROPSYCHOTHERAPY

NEUROPSYCHOTHERAPY WORKSHOPS 2013
From the Editor

Welcome 2013

I would like to extend best wishes to all our Neuropsychotherapy readers. I trust the year will be filled with down-regulated limbic alertness, controlled cortisol levels, good cortical blood flow, controlled incongruence and many new neural pathways! In other words – lots of joy, fun and a good dose of control.

The year has certainly kicked off with many activities happening on the Neuroscience front. We have received a big jump in our subscription numbers and hope that the editions of our Journal will meet the expectations of our readers.

Antidepressant medication and neuropsychotherapy

Our feature article in this edition focuses on very interesting new research indicating molecular changes facilitated by the use of antidepressant medication. This is linked with more research on the efficacy of antidepressant medication as well as research demonstrating neural changes as result of talking therapies. The implications for Neuropsychotherapy are discussed.

Publication of neuropsychotherapy text

I am happy to report that a book contract has been finalized with W.W. Norton – Publishing House. Norton is probably the largest publisher of Neuroscience books globally. I will be the single author of a book on applied neuroscience for therapists and on many levels this work will be the culmination of the four years of workshops and many years of research on various aspects of Neuropsychotherapy. We hope to finalise this by early 2014.

Interview

I recently did an interview with Richard Hill on aspects of my research and teaching – focusing on applications of Neuropsychotherapy – this interview will be available to view on a very exciting website www.neuropsychotherapist.com

This site brings many scholars in Neuroscience together in a forum to explore ideas and contribute to the field of neuroscience on a variety of levels. Readers are encouraged to visit this site.

Neuropsychotherapy workshops

The Neuropsychotherapy workshops commence soon – due to my teaching and research responsibilities the opportunities are limited to Sydney, Melbourne and Brisbane however the new workshop: The Social Brain and the Neuroscience of Relationships will run in all the normal locations – Sydney, Melbourne, Brisbane, Perth, Canberra, Adelaide and Hobart. More information on the last page of this edition or visit our website www.mediros.com.au

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THE END OF THE MEDICAL MODEL?

Recent findings in neuroscience regarding antidepressant medication – implications for Neuropsychotherapy.

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This paper explores new findings in neuroscience research regarding antidepressant medication as well as the changing paradigm relating to brain functioning. Recent findings in neuroscience challenge the view that antidepressant medication is beneficial and indicates significant areas of risk and even harm that this medication group may produce. Recently the paradigm of understanding the brain as neurochemical system has shifted to a focus on the brain as neural network. Both the new findings in neuroscience regarding antidepressant medication as well as the changing paradigm of understanding neural functioning pose new challenges to mental health provision. The role of enriched environments in general and more specifically talking therapies to facilitate new effective patterns of neural firing is discussed.

Introduction

What drives the human brain? This question has been one of the fundamental debates in modern neuroscience. Since the discovery of neurons and processes (glia) by Waldeyer, Golgi and Cajal in the late nineteenth century, the prevailing notion was that the brain runs on electricity. The brilliant electrophysiologist Julius Bernstein found in 1902 that nerve cells have steady potentials (electrical charges) and that, even at a resting state, there is a difference in voltage between the inside and the outside of the nerve cell. He suggested that the membrane of the cell must have a special channel through which positively charged ions can “leak out” and that this loss of positive charge leaves the inside surface of the nerve cell negatively charged. This discovery was the first indicator of the idea of the brain as an electrical system driven by the concept of “action potentials”. Later research by Nobel laureates Alan Hodgkin and Andrew Huxley confirmed these findings and linked the process to memory systems. Quickly researchers realised that as a consequence of these findings, the way to treat brain conditions was to manipulate the electrical systems – leading to the discovery of electroconvulsive therapy by Ugo Cerletti and Lucio Bini in 1938 (Shorter 2007). This form of treatment became widespread practice and is still used for many psychiatric disorders.

The brain: electrical or chemical system?

The electrical theory of neural activation was questioned by Henry Dale and colleagues who argued that a chemical acetylcholine acts as the transmitter of signals. The implication was clear – the basic operating system of the brain is not electrical but rather chemical. The talented neuroscientists Stephen Kuffler (who fled Europe as result of the rise of Nazism) and Australian John Eccles were strong supporters of the electrical theory. It was Eccles, however, who proved his own theory wrong by demonstrating clearly how the release of acetylcholine gives rise and fully accounts for all phases of action potentials. This work facilitated a paradigm shift in understanding the function of the brain – the birth of the chemical model in understanding and treatment of the human brain – often referred to as the medical model.

Eccles was awarded the Nobel Prize in Physiology or Medicine in 1963 for his ground-breaking work. These discoveries changed the nature of treatment of conditions related to the brain. Thousands of studies focused on chemical interventions to enhance “chemical balance” and neural functioning. In mental health the most profound of these studies was the discovery of the properties of a compound that acts as an inhibitor of serotonin.

The first wave of antidepressant treatment (a loosely used word for management of depression, anxiety obsessive compulsive disorders, eating disorders, chronic pain, snoring, migraines, attention-deficit hyperactivity disorder, substance abuse and even hormone mediated disorders) surfaced when Irving Selikoff and Edward Robitzek began clinical trials on anti-tuberculosis agents – Isoniazid and Ip-
roniazid. The conditions of patients improved dramatically. One of the side effects seemed to be a reduction in depressive symptoms. A psychiatrist, Max Lurie decided to trial the medication and reported that it reduced depressive symptoms in two thirds of his patients. He coined the term “antidepressant” to describe the action (Healy 2001). Imipramine seemed to be a powerful monoamine oxidase inhibitor and was popularized by Nathan Kline, head of Rockland State Hospital as “psychic energizer” (Time 1957). It was marketed as antidepressant with sales growing until it was recalled in 1961 due to reports of lethal hepatotoxicity. Attempts were made to improve the effectiveness of the compound which led to the discovery of “G2355”, later named imipramine. It quickly became the market leader and is still used today.

“Second generation” antidepressants were introduced to the market through work at the company Eli Lilly in the collaboration between Bryan Molloy, Klaus Schmeigel, David Wong and others on an antihistamine diphenhydramine that showed some antidepressant-like properties. Later another Lilly scientist, David Wong worked on derivatives to only inhibit serotonin. In May 1972 Jong-Sir Horng tested a compound that seems to be the most potent inhibitor of serotonin – later called fluoxetine (Prozac). The first article about fluoxetine was published in 1974 (Wong et al 1974; a twenty year follow up study was published by Wong et al 1995). The drug appeared on the Belgian market in 1986. Final drug approval was given in 1987 – within a year sales in the USA alone reached $350 million. Since 1978 a large number of related drugs were introduced to the market with annual sales of USD 11 billion in 2008. Thousands of research papers were published indicating that the primary mode of intervention for people suffering from anxiety/depression is an anti-depressant – a Selective Serotonin Reuptake Inhibitor (SSRI). The medical model of psychiatric care became the preferred mode of delivery as result of outcome based evidence – a chemical intervention as primary mode of treatment.

Evidence based research - questioned

In Zurich, Switzerland, Klaus Grawe and his team of researchers conducted a meta-analysis of 77 studies published between 1980 and 2001 comparing data from different studies accounting for between-study variability (Gallati 2003). There were 29 studies examining pharmacological treatments (5,352 subjects); 24 studies examining psychological therapies (1,297 subjects) and 24 studies comparing pharmacological and psychotherapeutic therapies (2,668 subjects). The treatments lasted an average of 10 weeks and psychotherapies encompassed an average of 13 sessions. For every study, researchers calculated pre- and post-effect sizes for each measure used with participants in both treatment and control conditions.

Gallati and colleagues found that the pre-post effect sizes after an average of 10 weeks of treatment were indeed very high, compared to the pre-post effect sizes normally found in psychotherapy. Whereas the average pre-post effect size across various forms of psychotherapy and types of disorders has been estimated at 1.21 (Gallati 2003), this value in pharmacological treatment is on average 2.11. The SSRI’s Venlafaxine (Zoloft) and Paroxetine (Aropax) even resulted in effect sizes of more than 3!T

The team found the effect size of psychological therapies alone or in combination with drug therapies was on average 1.76 – much lower than the effect size of drug therapy alone (2.11). The average effect sizes for various psychotherapeutic treatment modalities are clearly lower than those observed for pharmacological treatment (Gallati 2003).

The above findings seem to suggest three conclusions: anxiety and depression, compared to other disorders can be treated particularly effectively.

• Medication is the first line of treatment.
• Psychotherapy is very effective in depression sufferers even though it is not as effective as antidepressant medication.
• There are no marked differences in the effectiveness of various psychotherapy modalities according to this meta-analysis.

These are exactly the messages that are conveyed at some congresses accompanied by huge advertising campaigns by the pharmaceutical industry. The implication seems to be clear – if a patient is suffering from depression and/or anxiety – the first primary mode of treatment is drug therapy. The real state of affairs however, looks completely different.

Research demonstrates that depression and anxiety are conditions that are quite stable over long periods of time (unless treated). However longitudinal studies show that these disorders tend to show fluctuations, or signs of improvement from time to time (before dropping back the significant
level of disorder symptomatology). If a study is conducted over a 10 week program the likelihood of spontaneous symptom reduction/brief remission is high. Studies that examined the effect size of symptom reduction in control groups found an effect size as high as 1.59 without any intervention! Of course this does not mean the depression or anxiety has shifted into permanent remission. The same pattern applies to positive outcomes demonstrated in other interventions. If one subtracts the effect size of medications (2.11) from this spontaneous effect size (1.59), the net effect size actually is only 0.52.

Grawe and his team went one step further. They applied a measure that was developed for the assessment of medication effects for depressed patients (the Montgomery-Asberg Depression rating Scale – MADRS) on these studies and found the effect size across all drugs was estimated to be 1.88. On the face of it, it looks quite impressive. The same scale, however, found the effect size of the control group (no interventions) to be 1.82. This yields a net effect of 0.06! This is a tiny improvement – considering the significant side effect profile of many drugs. This, figure of 0.06, according to Grawe, is the true average effect size of pharmacological interventions (Grawe 2004, Grawe 2007).

Grawe and his team also demonstrated a further problematic issue with the drug trials – these studies did not factor in the dropout rate. Dropouts indicate treatment failures. For every person that discontinues treatment (mostly due to negative side effects) the outcomes for the smaller group that finish the trials, improves. The greater the attrition, the higher the effect size for the smaller number that finish the trial. The dropout rate for drug therapies was on average 25%, whereas this figure was on average 13% - almost 50% less for studies focusing on psychotherapies. This drastically skews the effect size of the medication group and significantly favours the effect size of this group. If this fact is accounted for, the effect size of the psychotherapy group is actually higher than the medication group.

Lastly, there is no research on the long term effect size of drugs to treat anxiety or depression whereas there are many studies that examine the effect size of psychotherapy to treat depression and anxiety over longer time periods. Recent advances in neuroimaging (fMRI, PET and SPECT scans) demonstrate the structural and functional changes that happen in the brain by means of talking therapies. Grawe and his team highlighted the flaws surrounding the notion that “research” indicates that the primary mode of intervention for significant anxiety and mood disorders is a chemical intervention, as many variables were not taken into account in the studies (Grawe 2004). This conclusion is clearly supported in the study by Lloyd Linford and John Arden and (2009) where they indicate that the era of the “pax medica” (the end of pharmacological preferences directing the intervention process as result of recent neuroresearch that pointed in a new direction) (Hollon 2011; Kumari 2006; Potoski and Heimberg 2010).

Once the higher dropout rate and the inferior long-term effectiveness of pharmacological treatments are taken into consideration, one arrives at the conclusion that psychological therapy is more likely the treatment of choice in the management of depression and that medications should be used only when combined with psychological therapy and in cases in which psychotherapy alone has not led to the desired success (Haby et. al. 2006). This view is shared by Steven Hollon from Vanderbilt University. In a recent study he found CBT to be more beneficial with better long term outcomes than ADM (antidepressant medication). He shows how the efficacy of ADM's has been overstated, and he also refers to the grossly skewed publication bias (Hollon 2011).

Publication bias

Hollon refers to the study by the University of Oregon in 2008 that demonstrated publication bias towards superiority of drugs over psychotherapy
and or placebos (Turner et al 2008). In this study Erik Turner and colleagues obtained reviews from the Food and Drug Administration (FDA) of 12 antidepressant agents involving 12,564 patients. They conducted a systematic literature searches to identify matching publications and also compared effect size derived from the published reports with those derived from the entire FDA data set. The found that, according to published literature, 94% of the trials conducted were positive. Meta-analysis of the FDA data set indicated only 32% positive effect. Among the 74 FDA-registered antidepressant studies, the team found that 23 trials (31%) had not been published. Among the 38 of 74 studies (51%) that the FDA deemed to be positive, almost all of them (37) were published. The remaining 36 studies (49%) were deemed to be either negative (24 studies) or questionable (12). Of these 36 studies, 22 were not published, 11 were published as positive, and 3 were published as negative. The implication is clear: studies indicating negative/adverse outcomes were not published. Turner and colleagues concluded that the publication bias may have adverse consequences for researchers, study participants, health care professionals and patients (Turner et al 2008).

Another example of bias reporting is the widely reported study - the “Sequenced Treatment Alternatives to Relieve Depression (STAR*D)”, and is often referred to as evidence that antidepressants are effective long-term to reduce depressive symptoms. (Insel and Wang 2009). The STAR*D did not include the placebo control group and also did not report the 93% relapse/dropout rate. This demonstrates a massive outcome bias and if factored in, demonstrates no benefit to long term antidepressant use in comparison to placebo (Piggott, Leventhal, Alter and Boren 2010).

Evidence from molecular neuroscience questioning the benefits of antidepressants

A research paper with a bold title questioning the benefits of antidepressants was published in 2012 in Frontiers of Psychology (Andrews et al 2012). The Latin title of the paper “Primum non nocure” (first, do no harm) refers to the essence of the Oath of Hippocrates (although the original Hippocratic words are: “to abstain from doing harm” (Greek: ἐπὶ δηλήσει δὲ καὶ ἀδικίᾳ ἐξρήσειν). The rest of the title of the paper is clear “an evolutionary analysis of whether antidepressants do more harm than good”.

In this paper, the authors state that current medical model of antidepressant medication as first line treatment modality needs to be re-evaluated against current neuro-molecular evidence. The processes of regulating serotonin from its ancient basis in plants and fungi to adaptive processes evolving emotion regulation, neural growth and neural death (apoptosis), attention and reproduction are explored. One of the key principles of molecular science is that disruptions of evolved adaptations will degrade biological functioning. The key role of serotonin in adaptation processes has been clearly established and accepted in neuroscience (Kandel 1976, Kandel 2001, Kandel 2005, Kandel et al 2013). Disruption of the role of serotonin may have adverse health effects. Inhibition of neurobiological actions (serotonin reuptake) causes morphological changes to neural structure resulting in higher risk of apoptosis (neural death). This means relapse rates will increase with prolonged intake of serotonin inhibition.

The authors argue that, contrary to the widely held belief that antidepressants promote production of brain derived neurotrophic factor (BDNF) and as such, neurogenesis, the method to detect this, 5-bromo-2'-deoxyuridine (BrdU) that identifies DNA synthesis – interprets the synthesis as indication of neurogenesis. However the researchers point out that DNA synthesis often occurs during the process of apoptosis (neural death) and is most likely part of the cycle-related cell death (Herrup et al 2004). More recent sophisticated studies found no evidence that antidepressants triggered neural growth (Kobayashi 2010). Kobayashi did find that fluoxetine caused mature neurons to take on immature functional characteristics. Constant serotonergic input is needed to maintain the mature state of neurons. The implication is that long term inhibition of serotonin uptake may lead to much greater risk of relapse when inhibition discontinues (discontinuation of medication). This leads to a vicious cycle where the neural maturation will be compromised when medication continues and even more compromised when it discontinues (double negative – the loose–loose effect). Apart from apoptosis, the
The brain as a network

Research by Kandel (Kandel 2006; Kandel et al 2013), Sporns (2011), Davidson & Begely (2012), Schore (2012), Rossouw (2011b; 2012) and many others has demonstrated that the notion of the brain as a network of connections has progressed to understanding the brain as a network of connections. Freud suggested that the space between neurons is the essence of the subconsciousness (Freud 1895). Researchers now agree that this notion, albeit simplified, is correct on a molecular level. Neural connection is the essence of memory. However memory is more than the connection of a single axon with a single dendrite. It is the sequence – the network that forms the basis of neural functioning. Although these connections are genetically programmed, the interaction with the environment facilitates unique expressions of genetic predispositions, resulting in the formation of emotions, cognitions, the self and the mind. These networks are in constant process of adjustment to its environment and thus environment plays a significant role in how these networks activate, change direction and facilitate new pathways of firing – memories which change our emotions, cognitions and ultimately what defines the “self”. For example, trauma results in the formation of neural patterns of protection and avoidance; whereas positive experiences result in patterns of approach and growth.

Chemical interventions can facilitate changes in neural firing but cannot facilitate the production of new neural firing patterns. Research with rodents, primates and humans clearly demonstrates that antidepressant interventions can only happen through two processes – (1) negative – (through trauma) resulting in closed, fear based, protective, neural patterns and (2) positive patterns – (through enriched environments – facilitation of a safe environment – the availability of environmental safety), resulting in down-regulation of stress, attachment and control. These enriched environments include safe social interaction – a key function of healthy neural proliferation which is facilitated through talking therapies.

Implications for neuropsychotherapy

The implications of this paradigm shift in understanding the brain are significant. Interventions to facilitate effective neural pathways should be the preferred (first line) intervention strategy. Antidepressant medication interventions cannot facilitate wiring of new neural connections. It can, at best, facilitate inhibition of Serotonin reuptake in default neural pathways. This reuptake inhibition may be beneficial but there are also risks involved. When default neural patterns are activated in terms of Serotonin reuptake inhibition, increased activity in those patterns occurs. In mental health terms it means that unhelpful patterns of perception, emotions, cognitions and behaviors are enhanced – hence the increase in risk. The implication is clear – unhelpful patterns of neural activation need to be addressed through enriched environments to facilitate effective neural activation that is geared towards functional outcomes. These enriched environments include variables like down regulated stress, controllable incongruence, safety, quality sleep, exercise and good nutrition. Talking therapies have been shown to be highly effective to facilitate change in neural firing and neural structure (Furmark 2002; Goldapple and Siegal 2004).
A therapist providing talking therapies facilitates a number of neural changes. First, the therapeutic environment facilitates safety. Safety is essential for anyone in distress as it down regulates the stress response system. Distress activates the Hypothalamus-pituitary-adrenal system (HPA-axis) as result of chemical changes (release of corticotrophin releasing factor, adrenocorticotropic hormone, adrenalin and cortisol) which is the result of activation of the fear response triggered from the amygdala and basal ganglia. When these patterns are activated on regular basis, the patterns of firing become well-established and strengthened with supportive communication systems (glia) that leads to a default neural activation when a trigger is received. Down regulation of these patterns is essential as a starting point to develop new patterns of neural activation. Administration of medication without this down regulation will not facilitate new neural activation and will only provide brief symptom relief with increased risk of relapse (as demonstrated by Andrews et al 2012).

Second, talking therapies also provide controlled incongruence - a process whereby change is facilitated in such a way that safety is maintained whilst a sense of disequilibrium is facilitated. This is vital to enable change however this must be achieved in a carefully controlled way so as not to activate the default distress response – the basis of resistance. If change is facilitated too fast, the stress signal is activated and default patterns of pathology are facilitated.

The process is not a quick fix. Therapeutic interventions need to continue for a while to facilitate the wiring of new neural patterns. If this process is discontinued too quickly the new patterns of neural activation are not well-established and the risk of relapse to former default neural patterns increases.

Without psychotherapeutic intervention (activation of new neural patterns) a mere introduction of antidepressants are potentially harmful. Understanding the brain from a network theory (in comparison to the older chemical theory) means that the first line intervention is always linked with the facilitation of enriched environments – safety and structured talking (therapeutic interventions).

The essential need is to assist with the formation of effective neural networks (Kandel 1998, Kumari 2006, LeDoux 2005). Although much more research is needed to demonstrate the effective formation of specific new neural pathways, the focus on chemical interventions as first line intervention is unsustainable. Unless the presentation of a disorder is very severe, first line chemical intervention compromises (and even violates – Andrews et al 2012, Rossouw 2002, Rossouw 2010, Rossouw 2011) the basic principles of molecular neuroscience in particular and mental wellness in general.

The new paradigm of understanding neural activation and brain activity indicates the need for enriched environments as a primary intervention mode to facilitate effective neural pathways.

**Literature**


Deciphering the link between the human brain and behaviour has always been one of the most intriguing—and often challenging—aspects of scientific endeavour. The sequencing of the human genome, and advances in molecular biology, have illuminated the pathogenesis of many neurological diseases and have propelled our knowledge of how the brain controls behaviour.

To grasp the wider implications of these developments and gain a fundamental understanding of this dynamic, fast-moving field, Principles of Neuroscience stands alone as the most authoritative and indispensable resource of its kind.

In this classic text, prominent researchers in the field expertly survey the entire spectrum of neural science, giving an up-to-date, unparalleled view of the discipline for anyone who studies brain and mind. Here, in one remarkable volume, is the current state of neural science knowledge—ranging from molecules and cells, to anatomic structures and systems, to the senses and cognitive functions—all supported by more than 900 precise, full-colour illustrations. In addition to clarifying complex topics, the book also benefits from a cohesive organization, beginning with an insightful overview of the interrelationships between the brain, nervous system, genes, and behaviour. Principles of Neural Science then proceeds with an in-depth examination of the molecular and cellular biology of nerve cells, synaptic transmission, and the neural basis of cognition. The remaining sections illuminate how cells, molecules, and systems give us sight, hearing, touch, movement, thought, learning, memories, and emotions.

The new fifth edition of Principles of Neural Science is thoroughly updated to reflect the tremendous amount of research, and the very latest clinical perspectives, that have significantly transformed the field within the last decade. Ultimately, Principles of Neural Science affirms that all behaviour is an expression of neural activity, and that the future of clinical neurology and psychiatry hinges on the progress of neural science. Far exceeding the scope and scholarship of similar texts, this unmatched guide offers a commanding, scientifically rigorous perspective on the molecular mechanisms of neural function and disease—one that you'll continually rely on to advance your comprehension of brain, mind, and behaviour.

FEATURES

- The cornerstone reference in the field of neuroscience that explains how the nerves, brain, and mind function
- Clear emphasis on how behaviour can be examined through the electrical activity of both individual neurons and systems of nerve cells
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- Detailed discussions of cognitive and behavioural functions, and an expanded review of cognitive processes
- A focus on the increasing importance of computational neural science, which enhances our ability to record the brain’s electrical activity and study cognitive processes more directly
- Chapter-opening Key Concepts provide a convenient, study-enhancing introduction to the material covered in each chapter
- Selected Readings and full reference citations at the close of each chapter facilitate further study and research
- Helpful appendices highlight basic circuit theory; the neurological examination of the patient; circulation of the brain; the blood-brain barrier, choroid plexus, and cerebrospinal fluid; neural networks; and theoretical approaches to neuroscience
Neuropsychotherapy is an exciting science, not least because of the recognition that significant changes occur for clients in neural firing and structure as a result of talking therapies. Neuropsychotherapy is the “language” used in the interaction between the clinician and the client to guide the client in the process of restructuring the brain towards higher levels of functioning and well-being. It uses information from neurosciences to assist clients suffering from a wide range of biological, psychological and social challenges to apply strategies to down regulate unhelpful neural stress responses and up regulate neural activation towards neural change. Understanding the neurophysiology of these disorders and activation patterns of neural pathways as well as discussing practical applications, assist clinicians greatly to apply more effective strategies to treat depression, anxiety and trauma.

ABOUT THE PRESENTER
Pieter J. Rossouw  MClinPsych;PhD;MAPs;CClin.

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. 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 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. Pieter was a Professor in Clinical Psychology at Universities in Canada, Holland and South Africa where he also spearheaded a Psycho-Therapeutic Assistance Program to support people being exposed to trauma.

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 and the Australasian Cognitive Neuroscience Society.

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Royal Melbourne Hospital, Grattan Street, Parkville

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Portside Centre, Portside Centre, Level 5, 207 Kent Street, Sydney,

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- **Brisbane**: 27 & 28 June 2013, RBW Hospital, Herston Rd, Herston, Brisbane

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- **Brisbane**: 12 & 13 Sept 2013, RBW Hospital, Herston Rd, Herston, Brisbane
- **Melbourne**: 20 & 21 Sept 2013, Royal Melbourne Hospital, Grattan Street, Parkville
- **Sydney**: 3 & 4 October 2013, Portside Centre, Level 5, 207 Kent Street, Sydney

**The Social Brain and the Neuroscience of Relationships**
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- **Canberra**: 18 & 19 Oct 2013, Calvary Private Hospital, Mary Potter Cct, Bruce, ACT
- **Adelaide**: 25 & 26 Oct 2013, Hackney Hotel, 96 Hackney Road, North Adelaide
- **Perth**: 1 & 2 Nov 2013, St Catherine’s Coll, UWA, 2 Park Rd, Nedlands, Perth
- **Melbourne**: 15 & 16 Nov 2013, Royal Melbourne Hospital, Grattan Street, Parkville
- **Brisbane**: 21 & 22 Nov 2013, RBW Hospital, Herston Rd, Herston, Brisbane
- **Sydney**: 28 & 29 Nov 2013, Portside Centre, Level 5, 207 Kent Street, Sydney
- **Hobart**: 25th & 26 Nov 2013, Grand Mercure Hadley’s Hotel, 34 Murray St, Hobart

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- **Brisbane**: 04 December 2013, RBW Hospital, Herston Rd, Herston, Brisbane
- **Melbourne**: 07 December 2013, Royal Melbourne Hospital, Grattan Street, Parkville
- **Sydney**: 13 December 2013, Portside Centre, Level 5, 207 Kent Street, Sydney

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**Signature:** _______________________________________________

**Amount:** ____________________________

**Cheque**

**Bank Transfer – we will email you the invoice & Mediros bank details**

**Email to:** andie@mediros.com.au

**Fax:** 07 3294 3220

**Mail:** Mediros (Admin), PO Box 6460, St Lucia, Qld, 4067

**Mediros Phone Number:** 07 3217 7266